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ORTHOPAEDICS
Perspective

The Weil Osteotomy for Correction of Fixed Claw-Toe Deformity and Metatarsalgia

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Abstract

Aim: The aim of our study is to evaluate the efficacy of the Weil osteotomy to correct fixed claw lesser toe deformity with dorsal subluxation or dislocation of the MTP joint and overloading metatarsalgia.

Material: 120 Weil osteotomies in 60 feet of 45 patients have retrospectively been studied with a minimum follow-up of 12 months (mean f.u. 17 months). Thirty-five patients were women and ten men. For the evaluation of the result we used the AOFAS grading scale. Antero-posterior and lateral weight bearing radiographs were taken preoperatively and at the time of follow-up. Forty patients had a correction of hallux valgus deformity although 5 of the lesser toe only. All patients received a PIP fusion for the correction of the claw deformity.

Results: The subjective result was at their expectation for 39 patients and beyond their expectation for 5. One patient was dissatisfied. The AOFAS score improved from 45 preoperatively to 89 postoperatively. Thirty-nine patients did not complain of metatrsalgia although for five patients metatarsalgia improved and for 1 remained at preoperative level. Shoe wearing improved in all 42 patients although for 3 this was below their expectations.

Conclusion: The Weil osteotomy combined with PIP fusion is capable of correcting stiff claw lesser toe deformities with dorsal subluxation or dislocation of the MTP joint and significantly improve metatarsalgia.

Keywords: *Ankle; Arthroscopy; impingement; foot; sports injury; pain*

Introduction

Ankle pain is a common musculoskeletal problem, affecting more than 10% of the total Greek population. In a case series examining arthroscopic management of anterior ankle impingement (AAI), 90% of patients with AAI were found to have moderate to severe limitation in activities of daily living due to pain. AAI is defined by anterior ankle pain and painful terminal dorsiflexion on physical examination. Weight-bearing radiographs typically show so-called kissing exostoses on the anterior distal tibia and dorsal talus. The tibiotalar joint space is preserved; this feature distinguishes AAI from early ankle osteoarthrosis. Both nonsurgical and surgical management have proved to be successful. Surgically, AAI has traditionally been treated with open debridement.

Arthroscopic minimally invasive techniques have become more popular in recent decades, however. In 1949, Sir Thomas McMurray penned his final publication, titled "Footballer's Ankle." [1]. In this classic article, McMurray described a chronic ankle injury found in professional soccer players who reported point tenderness over the anterior ankle joint and pain with passive dorsiflexion. Radiographs showed ankle osteophytes. Intraoperatively, exostoses were found deep to the joint capsule in the setting of a healthy articular surface. This finding was in stark contrast to the eburnated articular surface seen in osteoarthritis. McMurray treated six professional footballers with open decompression, all of whom were able to return to professional athletics.

The 1980s saw renewed interest in AAI. Since then, research has focused on the etiology and management of AAI [2].

Anatomy

The ankle is an imperfect and highly constrained hinge joint composed of three articulations: the tibiotalar, fibulotalar, and tibiofibular joints. The distal tibia and fibula form the ankle mortise, with the trochlea of the talus acting as the keystone. This trochlea has been likened to a truncated cone with its apex oriented medially. From above, the talus appears wedge-shaped, narrowing posteriorly. This shape contributes to ankle containment and stability. Motion at the ankle is multiplanar, with an obliquely oriented axis of rotation that runs through the tips of the malleoli. This axis of rotation results in downward and posterior inclination in the sagittal plane and posteromedial inclination in the transverse plane [3].

The end result is a combined rolling movement of flexion-extension associated with a horizontal sliding rotation and coronal plane abduction adduction. The three articulations of the ankle are stabilized by a balance of static and dynamic structures. The ligaments that impart stability are divided into three groups: syndesmotic, lateral, and medial. At the level of the tibial plafond lie the syndesmosis and its encompassing ligaments, which maintain the relationship of the distal fibula and tibia. These are the anteriorinferior tibiofibular ligament (AITFL), posterior-inferior tibiofibular ligament, transverse tibiofibular ligament, and interosseous tibiofibular ligament. This strong ligament complex, in combination with the bony architecture, prevents proximal migration of the talus between the tibia and fibula and contributes transverse stability to motion of the tibiofibular joint.

The lateral ligaments consist of the anterior talofibular ligament, the calcaneofibular ligament, and the posterior talofibular ligament. This ligamentous complex is the primary restraint to anterior translation of the talus. The medial ligaments are the superficial deltoid, deep deltoid, and spring (i.e., plantar calcaneonavicular). These ligaments resist posterior and lateral translation as well as valgus tilt of the talus. Together, these ligaments play a crucial role in guiding stable joint motion. Damage to these structures will affect the coupled motion of the intact ankle joint. Further, ligamentous instability may coexist and play a role in the etiology of AAI. Thus, a thorough understanding of the original geometry of the ankle complex is crucial to restoration of function.

Pathophysiology

The exact cause of AAI remains unclear. McMurray proposed a "pull" etiology on the bone spurs and thought that AAI initiated with traction injuries to the anterior capsule [4]. O'Donoghue disagreed, believing repetitive impact from forced dorsiflexion to be the cause of bone spur formation [5]. McMurray's hypothesis of a traction etiology leading to exostosis formation is unlikely because the exostoses are found deep to the joint capsule rather than within the substance of the capsule. Van Dijk, for example, noted that the exostoses on the tibia and talus tend to be intra-articular and not attached to any capsular structures that would impart traction. Activities that produce repetitive trauma seem to be more correlated to the development of AAI [6].

Dance is another activity in which the ankle may be subjected to repetitive abnormal motions. The plié, consisting of forced dorsiflexion with locked external rotation, has been described as the most common movement contributing to AAI. Soft-tissue structures can cause AAI without actual bony impingement, however Berberian., *et al*. performed CT scans on 9 patients (10 ankles) with AAI and found that the talar spur lies medial to the midline, whereas the tibial spur lies lateral to the midline; typically, the spurs do not overlap [7].

A triangular shaped area of soft tissue consisting of synovium, collagen, blood vessels, and adipose is located in the anterior joint space between the talus and tibia; in a normal ankle, these tissues are compressed between the talus and tibia on dorsiflexion. In patients with AAI syndrome, pain may be secondary to impingement of this soft tissue. Coincident chondral and osteochondral lesions may be found in patients with anterior ankle pain. Rasmussen., *et al.* [3] performed 105 ankle arthroscopies in patients with painful dorsiflexion and found 20 chondral lesions and 16 loose bodies. In a series of ankle arthroscopies for exostoses resection, Moon., *et al*. [8] found that 80.7% of patients with distal tibial exostoses also had a corresponding cartilage lesion on the talar dome. Ankle instability may also contribute to AAI syndrome. Between 13% and 35% of patients continue to have ankle pain after a lateral ankle stabilization procedure. This may be due to unaddressed intra articular pathology [9-11].

Patient Evaluation

Physical Examination

Despite controversy regarding the underlying pathophysiology of AAI, its clinical presentation is consistent and straightforward. In the early stages of the disorder, anterior ankle pain is elicited with long periods of exercise and is relieved with rest. As AAI becomes chronic, additional symptoms may include instability; limited ankle motion; and pain with squatting, sprinting, stair climbing, and hill climbing. Normal gait may be unaffected. Physical examination typically elicits tenderness over the anterior ankle joint and pain with forced dorsiflexion. Ankle swelling maybe present, as well. Patients also should be evaluated for other etiologies or concurrent ankle pathology. The physical examination should include inspection of the ankle for swelling, erythema, and alignment. An anterior drawer test should be performed to evaluate for lateral ankle instability. The Silfverskiold test should be performed to evaluate for isolated gastrocnemius contracture. This test measures ankle dorsiflexion with the knee in extension and in 90° of flexion. The test is considered positive when ankle dorsiflexion is greater with the knee in flexion than in extension [12-15].

Imaging

The diagnosis of AAI is usually made clinically and confirmed with plain radiographs. Patients often have exostoses on the distal anterior tibia and dorsal talar neck [16]. Unlike the presentation in ankle osteoarthritis, the joint space is preserved on weight-bearing lateral ankle radiographs. Anteromedial exostoses may not be visible on lateral radiographs due to the presence of superimposed structures. CT detects the presence of exostoses, and MRI is useful in evaluating for soft-tissue impingement as well as chondral or subchondral injuries. Huh., *et al*. [17] showed that MRI has 92% sensitivity and 64% specificity for detecting synovitis and 77% sensitivity and 97% specificity for detecting soft-tissue impingement.

Management

Conservative

Physical therapy (especially lateral ankle stability protocols), shock-absorbing shoes, steroid injection, NSAIDs, use of a heel-lift orthosis to prevent dorsiflexion, and activity restriction have all been advocated and seem to be reasonable nonsurgical options for patients with AAI [18].

Surgical Management

Surgical intervention is considered in patients with persistent symptoms. Surgical goals include removing the exostoses and débriding the soft tissue in the anterior ankle joint. Patients with tibial, talar, and/or fibular exostoses may be asymptomatic and soft-tissue inflammation and impingement may contribute significantly to the pain. A complete diagnostic arthroscopy should be performed to ensure a thorough examination for intra-articular pathology. Hypertrophic synovium is often encountered, and a shaver is used to debride this material to enhance visualization. The anterior, medial, lateral, and posterior compartments are thoroughly débrided. Osteochondral lesions are microfractured, nonviable cartilage flaps trimmed, and loose bodies removed. No studies describe how much bone should be removed from the anterior distal tibia or talar neck. We routinely use a 3.5-mm arthroscopic burr to contour the anterior tibia until it is flush with the anterior border of the medial malleolus. Bossing or exostoses of the talar neck are débrided so that there is no tibial talar contact at maximal dorsiflexion. If the impinging bone and hypertrophic soft tissue cannot be adequately debrided using the standard anteromedial and anterolateral portals, accessory portals may be created to access the ankle joint. Extending either the lateral or the medial portal to create an open arthrotomy allows rapid and complete debridement of such pathology [19,20]. In cases of lateral ankle instability, tibial, fibular, talar, and soft-tissue impingement can be easily removed through the standard extensile lateral arthrotomy, after which ligament reconstruction is performed [21-23].

Rehabilitation

Despite the critical importance of postoperative treatment, no studies specifically address the rehabilitation protocol after removal of exostoses or soft-tissue impingement. Some surgeons allow weight bearing as tolerated following surgery, whereas others prefer non-weight bearing in a splint for 5 days followed by progressive weight bearing. Active and passive range of motion is begun soon after surgery, as are physical therapy (for strengthening) and proprioceptive exercises and aggressive range of motion and weight bearing [24-27].

Outcomes

In a series on arthroscopic debridement, of 12 patients (10 athletes) we had average 8° improvement in ankle dorsiflexion, with 11 of 12 patients returning to sporting activities with a substantial improvement in pain at a mean of 3 months after surgery. Full athletic participation at a mean follow-up of 9 months after arthroscopic synovectomy with removal of exostoses and 92% were satisfied with their outcome. There were no complications in all series and there was no need for open arthrotomy of the joint for further debridement [28-30].

Conclusion

AAI is a common condition characterized by chronic anterior ankle pain that is exacerbated by dorsiflexion. In the acute phase, pain is elicited following long periods of activity and relieved with rest. As the condition becomes chronic, patients report limited ankle motion and diminished exercise tolerance. They also report anterior ankle tenderness and pain with ankle dorsiflexion. Typically, radiographs demonstrate exostoses off the anterior distal tibia and dorsal talar neck. When nonsurgical measures are unsuccessful, surgical debridement of the offending soft tissues and exostoses has shown great success in returning patients to their previous levels of activity. Surgeons should carefully assess and simultaneously treat these patients for other foot and ankle pathology.

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