Review Article

Anterior Ankle Impingement: Diagnosis and Treatment

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Abstract

Anterior ankle impingement is a common clinical condition characterized by chronic anterior ankle pain that is exacerbated on dorsiflexion. Additional symptoms include instability; limited ankle motion; and pain with squatting, sprinting, stair climbing, and hill climbing. Diagnosis is typically confirmed with plain radiographs. Nonsurgical management includes physical therapy, strengthening exercises, activity modification, bracing, and anti-inflammatory medication. Although arthroscopic treatment is sufficient in some patients, most require an open approach to address related pathology. We advocate aggressive range of motion as well as weight bearing postoperatively. Further study is needed to confirm current understanding of anterior ankle impingement and to better define treatment options and prevention strategies.

Ankle pain is a common musculoskeletal problem, affecting more than 15% of the total US population.[1] In a case series examining arthroscopic management of anterior ankle impingement (AAI), 91% of patients with AAI were found to have moderate to severe limitation in activities of daily living due to pain.[2]

AAI is defined by anterior ankle pain and painful terminal dorsiflexion on physical examination.[3] 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 débridement. 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."[4] 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[4] treated six professional footballers with open decompression, all of whom were able to return to professional athletics.

O'Donoghue[5] took issue with the term footballer's ankle because he thought that this process was not confined to athletes but could also be seen in people who climb ladders or squat frequently. In 1957, he coined the term "impingement exostoses" to describe this nonarthritic anterior ankle pain syndrome coupled with exostoses on the dorsal talar neck and distal anterior tibia.

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The 1980s saw renewed interest in AAI.[6-8] Since then, research has focused on the etiology and management of AAI.

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.[9] From above, the talus appears wedge-shaped, narrowing posteriorly. This shape contributes to ankle containment and stability. Motion at the ankle is multiplanar,[10] 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.[9] The end result is a combined rolling movement of flexion-extension associated with a horizontal sliding rotation and coronal plane abduction-adduction.[11]

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 anterior-inferior 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.[12] 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 (ie, plantar calcaneonavicular). These ligaments resist posterior and lateral translation as well as valgus tilt of the talus.[12] 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[4] proposed a "pull" etiology on the bone spurs and thought that AAI initiated with traction injuries to the anterior capsule. O'Donoghue[5] disagreed, believing repetitive impact from forced dorsiflexion to be the cause of bone spur formation.

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.[13,14] Tol and van Dijk,[13] 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.[15-18] In a study of professional soccer players, Massada[17] found that 42 of 88 patients had radiographic evidence of tibiotalar exostoses. 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.[18]

Soft-tissue structures can cause AAI without actual bony impingement, however.[13,14] Berberian et al[14] 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. Neither were the spurs found to overlap in the cadaver study by Tol and van Dijk.[13] Soft-tissue inflammation and cartilage lesions may cause pain.[3,13,14,18-22] A triangular-shaped area of soft tissue consisting of synovium, collagen, blood vessels, and adipose is located in the anterior

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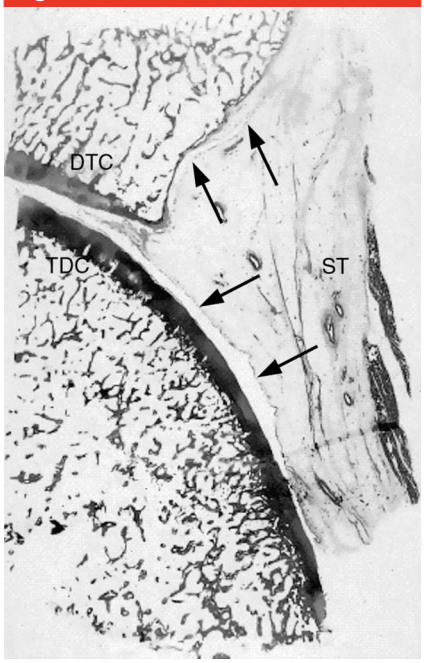
Figure 1



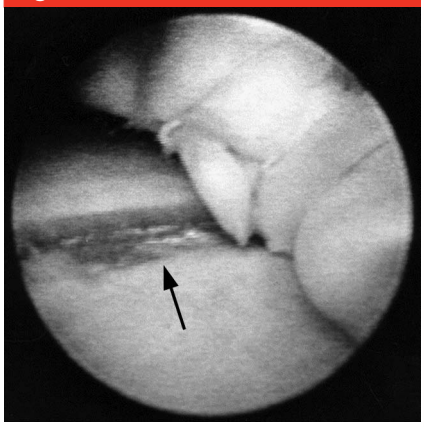
Hematoxylin-eosin stain of the tibiotalar joint demonstrating a triangular-shaped area of soft tissue bordered by synovium (arrows). Part of the soft-tissue component projects into the joint space. DTC = distal tibia cartilage, ST = synovial tissue, TDC = talar dome cartilage. (Reproduced with permission from Tol JL, van Dijk CN: Etiology of the anterior ankle impingement syndrome: A descriptive anatomical study. *Foot Ankle Int* 2004;25[6]:382-386.)

joint space between the talus and tibia; in a normal ankle, these tissues are compressed between the talus and tibia on 15° of dorsiflexion.[13] In patients with AAI syndrome, pain may be secondary to impingement of this soft tissue[3,18,20] (Figure 1).

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[21] found that 80.7% of patients with distal tibial exostoses also had a corresponding cartilage lesion on the talar dome. Kim et al[22] described the so-called tram track lesion, a carved-out cartilage lesion in the talar dome that corresponds to the tibial osteophyte (Figure 2).

Ankle instability may also contribute to AAI syndrome.[6,23] Between 13% and 35% of patients continue to have ankle pain after a lateral ankle stabilization procedure.[24] This may be due to unaddressed intra-articular pathology. Between 14% and 26.4% of patients undergoing a modified Broström procedure for chronic lateral ankle instability were found to have coexisting anterior tibial osteophytes during concomitant ankle arthroscopy.[25,26] Bassett et al[6] reported no osseous exostoses in their series of patients with anterior ankle pain. All seven patients in that study reported that they had sustained a plantar flexion–inversion injury in the past with subsequent chronic ankle pain. At the time of surgery, the authors found that a distal portion of the AITFL was impinging on the anterolateral talus. In a subsequent cadaver dissection, 10 of 11 specimens had an accessory AITFL ligament that contacted the talus at a mean of 12° of dorsiflexion. The authors postulated that an incompetent lateral ligament leads to pressure and friction between the accessory AITFL and the talus, resulting in anterior ankle pain.[6]

Figure 2



Arthroscopic photograph demonstrating distal tibial exostoses and a tram track lesion in the talar dome cartilage (arrow). (Reproduced with permission from Kim SH, Ha KI, Ahn JH: Tram track lesion of the talar dome. *Arthroscopy* 1999;15[2]:203-206.)

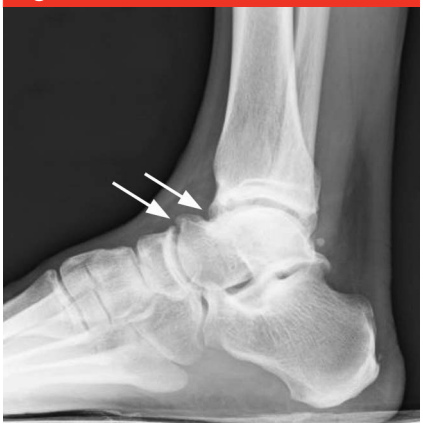
Patient Evaluation

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.[2] As AAI becomes chronic, additional symptoms may include instability; limited ankle motion; and pain with squatting, sprinting, stair climbing, and hill climbing.[2,7,14,27] Normal gait may be unaffected.[7]

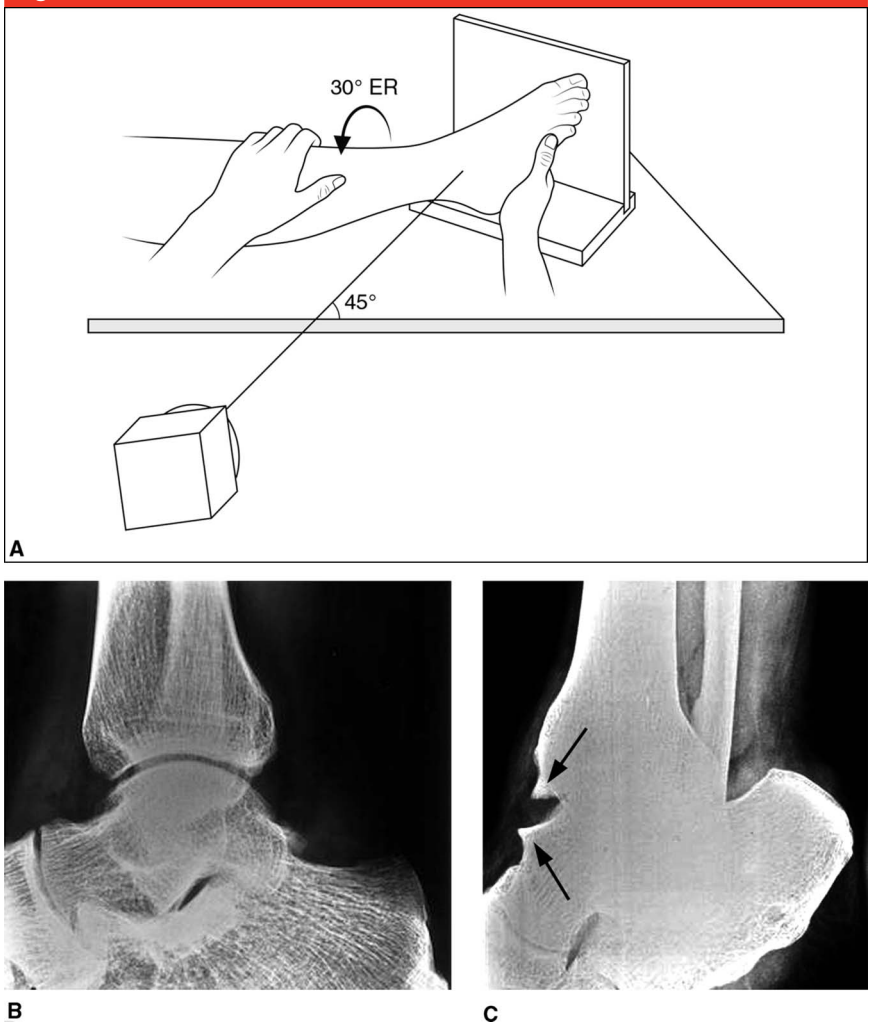
Physical examination typically elicits tenderness over the anterior ankle joint and pain with forced dorsiflexion[2] (ie, anterior impingement test[28]). Ankle swelling may be present, as well.[28] Patients also should be evaluated for other etiologies or concurrent ankle pathology. The physical examination should include inspection of the ankle for swelling, erythema, and alignment. Gait analysis may reveal asymmetry and malalignment. An anterior drawer test should be performed to evaluate for lateral ankle instability. The Silfverskiöld 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.

Figure 3



Lateral weight-bearing radiograph of a patient with anterior ankle impingement demonstrating preservation of the ankle joint space with anterior tibial and dorsal talar exostoses (arrows). (Courtesy of John S. Reach, MD, New Haven, CT.)

Figure 4



A, Illustration of patient and equipment positioning to obtain the oblique anteromedial impingement view. The beam is tilted 45° craniocaudally from the standard lateral position, with the foot externally rotated (ER) 30°. The foot is maximally plantarflexed, and the beam is centered just anterior to the lateral malleolus. The film is exposed 50% of the normal value for a standard lateral ankle radiograph. **B,** Lateral radiograph of a patient with anterior ankle pain. No significant tibiotalar exostoses are appreciated on this view. **C,** Oblique anteromedial impingement radiograph of the same patient demonstrating tibiotalar exostoses (arrows). (Panel A adapted with permission from and panels B and C reproduced with permission from van Dijk CN, Wessel RN, Tol JL, Maas M: Oblique radiograph for the detection of bone spurs in anterior ankle impingement. *Skeletal Radiol* 2002;31[4]:214-221.)

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.[7] Unlike the presentation in ankle osteoarthritis, the joint space is preserved on weight-bearing lateral ankle radiographs[19,28] (Figure 3).

Anteromedial exostoses may not be visible on lateral radiographs due to the presence of superimposed structures. van Dijk et al[29] developed a technique using an oblique radiographic view to detect anteromedial tibiotalar osteophytes that might otherwise be missed on standard lateral radiographs. They attached a barium-clay osteophyte to the anteromedial tibial rim of 10 cadaver specimens. The osteophyte was found to be most prominent on an oblique view with the beam aimed 45° craniocaudal and with the leg in 30° of external rotation (Figure 4). Smaller anteromedial osteophytes that were not detectable on standard lateral radiographs were visible on this oblique anteromedial impingement view.

A prospective clinical study confirmed the clinical utility of the oblique anteromedial impingement view in 60 patients with anterior ankle pain who subsequently underwent arthroscopy.[30] Standard lateral radiographs detected 40% of tibial osteophytes and 32% of talar osteophytes. When both lateral and oblique anteromedial impingement radiographs were used, 85% of tibial and 73% of talar osteophytes were visualized.

Advanced imaging is not always necessary, but it may be helpful. CT

detects the presence of exostoses,[14] and MRI is useful in evaluating for soft-tissue impingement as well as chondral or subchondral injuries.[31] Huh et al[20] showed that MRI has 92% sensitivity and 64% specificity for detecting synovitis and 77% sensitivity and 97% specificity for detecting soft-tissue impingement.

In our practice, we have found static and dynamic ultrasonography to be very helpful in diagnosing AAI. The use of handheld ultrasound is complementary to static 3-T magnetic resonance images, for which we use a dedicated foot and ankle coil, in differentiating bone marrow edema from AAI as well as other coincident pathology, such as peroneal tendon injury, osteochondral lesion, and loose body.

Management

Nonsurgical

Robust outcome studies investigating the optimal treatment of AAI are lacking. There is neither consensus nor validation of traditional nonsurgical treatment modalities. Physical therapy (especially lateral ankle stability protocols), shock-absorbing shoes, steroid injection, NSAIDs,[28,32] use of a heel-lift orthosis to prevent dorsiflexion, and activity restriction[33] have all been advocated and seem to be reasonable nonsurgical options for patients with AAI.

In our practice, we attempt to optimize ankle stability with proprioception and peroneal strengthening. Most of our patients also try activity modification, brace treatment, oral or topical NSAIDs, and a series of diagnostic and potentially therapeutic image-guided corticosteroid injections.

Surgical

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,[19] and soft-tissue inflammation and impingement may contribute significantly to the pain.[3,18,20] There are no studies describing débridement of anterior ankle soft tissue without removal of coincident bony exostoses. We recommend removal of all bony and soft-tissue structures that may potentially result in impingement.

Prior to 1988, published reports advocated open arthrotomy to remove ankle spurs.[4,5,8] However, in his 1988 article, Hawkins[7] argued that arthroscopic treatment allows the visualization of structures in the joint with less risk to surrounding structures and results in reduced postoperative pain. He described a technique in which a burr is used to shape the anterior tibia and dorsal talus to their original contour to avoid impingement of the joint space and in which the anterior ankle synovial tissue is débrided. Although arthroscopic resection is an option in some cases, many patients with AAI also have attenuated lateral ankle ligaments. These cases require an open lateral extensile approach through which the ankle exostoses may be rapidly removed.

Authors' Preferred Method of Surgical Management

In our practice, most patients have already tried several months of physical therapy, medications, and non–image-guided injections before they come to our practice. Such patients are carefully examined and imaged to confirm the diagnosis of AAI. We also assess for commonly associated pathologies, including lateral ankle instability, peroneal pathology, and osteochondral lesions. After ruling out instability and confirming intra-articular symptomatology with the use of an image-guided injection of lidocaine and corticosteroid, arthroscopic débridement is offered.

For optimal visualization, we routinely employ a leg holder at the level of the thigh, a noninvasive ankle distraction device, and a tourniquet placed about the thigh. With the patient in this position, stress examinations for instability and gastrocnemius contracture can be performed and any findings acted upon accordingly. Standard anterior lateral and medial portals are used to gain access to the joint. The plantar flexion fourth ray and the transillumination techniques are used to avoid the superficial peroneal nerve and the saphenous nerve and vein. Direct palpation and, occasionally, ultrasound guidance help to define these and other surrounding soft-tissue structures.

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 débride this material to enhance visualization. Given the relatively small fluid volume and flow, we do not recommend the use of thermal devices in the ankle joint. 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. Intraoperative fluoroscopy, dynamic ultrasonography, and direct visualization are routinely used to confirm adequate removal of the bone spurs.

If the impinging bone and hypertrophic soft tissue cannot be adequately débrided 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 débridement of such pathology. 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. Given the frequency of coexisting lateral ligament and peroneal pathology, we advise strongly against the use of nonextensile incisions.

In the setting of equinus contracture, a Silfverskiöld test is performed to determine whether the gastrocnemius, soleus, or both contribute to the soft-tissue deformity. We prefer a proximal or midsection Strayer procedure if the gastrocnemius is deemed to be the major contributor. If the soleus and gastrocnemius both contribute to the contracture, a triple hemisection of the Achilles tendon is performed.

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,[34,35] whereas others prefer non–weight bearing in a splint for 5 days followed by progressive weight bearing.[36] Active and passive range of motion are begun soon after surgery, as are physical therapy (for strengthening) and proprioceptive exercises.[35,36]

Our rehabilitation protocol for isolated AAI is designed to balance the need for relative immobility to allow wound and skin healing with the critical need for physiologic motion to prevent scarring and neuromuscular atrophy. Thus, we advocate aggressive range of motion and weight bearing. Physical therapy may be necessary for some patients. Many patients with AAI have coincident lateral ankle instability, however, and often an osteochondral lesion. Their postoperative rehabilitation is limited by recovery from the treatment of these coincident pathologies.

Outcomes

In a series on arthroscopic débridement, Ogilvie-Harris et al[37] reported average 9° improvement in ankle dorsiflexion, with 15 of 17 patients returning to sporting activities with a substantial improvement in pain. Another retrospective study showed complete resolution of anterior impingement symptoms in 12 of 13 patients at a mean of 15 months after arthroscopic removal of osteophytes and soft-tissue débridement.[34] Baums et al[35] performed a prospective study in which 25 of 26 patients returned to full athletic participation at a mean follow-up of 24 months after arthroscopic synovectomy with removal of exostoses. Coull et al[38] reported on 27 patients with AAI who were treated with an open procedure. Seventy-nine percent of patients were able to return to sports at a competitive level, and 92% were satisfied with their outcome.

There are no randomized controlled trials comparing open versus arthroscopic treatment of AAI. In a nonrandomized trial, Scranton and McDermott[39] compared 22 open procedures and 21 arthroscopic procedures. Although there was no difference in surgical time between the two groups, the arthroscopic group reported quicker return to subjective full recovery (ie, return to unlimited athletic activity).

Summary

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 débridement 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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Evidence-based Medicine: Levels of evidence are described in the table of contents. In this article, references 21, 25, 26, and 34 are level IV studies.

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