Ankle impingement syndromes are painful conditions caused by the friction of joint tissues, which is both the cause and the effect of altered joint biomechanics. The leading causes of impingement lesions are posttraumatic ankle injuries, usually ankle sprains, resulting in chronic ankle pain [1].

From anatomic and clinical viewpoints, these syndromes are classified as anterolateral, anterior, anteromedial, posteromedial, and posterior [1, 2].

Careful analyses of patient history and signs and symptoms at physical examination can suggest a specific diagnosis in most patients. MR imaging and MR arthrography are the most useful imaging methods for detecting the osseous and soft-tissue abnormalities present in these syndromes and for ruling out other potential causes of chronic ankle pain [1–3].

Treatment of all impinging lesions is the same regardless of the cause. The initial treatment is conservative, but when this fails,arthroscopic examination is indicated to identify and resect the impinging lesion [1, 2].

The purpose of this article is to describe the clinical, MR imaging, and MR arthrography features of ankle impingement syndromes.

**Anterolateral Impingement Syndrome**

Anterolateral impingement of the ankle is a relatively uncommon cause of chronic lateral ankle pain produced by entrapment of abnormal soft tissue in the anterolateral gutter of the ankle [2–4] (Fig. 1). Anterolateral impingement is thought to occur subsequent to relatively minor inversion injuries of the ankle. It is estimated that approximately 3% of ankle sprains may lead to anterolateral impingement [1]. Such trauma may result in tearing of the anterolateral soft tissues and ligaments without substantial associated mechanical instability. Repeated microtrauma can result in hypertrophied synovial tissue and fibrosis in the anterolateral gutter of the ankle (Fig. 2), causing pain and mechanical impingement [2–4]. In advanced cases, mechanical impingement may mold the tissue into a hyalinized meniscoid lesion, which was originally described by Wolin et al. [5].

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**Fig. 1.—Diagram shows site and extent of anterolateral impingement lesion (arrow).**
Other contributing factors are thought to include hypertrophy of an accessory fascicle of the anterior tibiofibular ligament and osseous osteophytes [2–4]. The accessory fascicle of the anterior tibiofibular ligament is a common variant (Fig. 3) that was first described by Bassett et al. [6]. This ligament may hypertrophy after repeated trauma, resulting in anterolateral impingement (Fig. 4), particularly when other anterolateral supporting structures are compromised [2–4, 6].

The clinical diagnosis of anterolateral impingement can be established on the basis of the combined presence of anterolateral ankle tenderness, swelling, and pain exacerbated by single leg squatting, ankle eversion, or dorsiflexion. However, the clinical diagnosis of anterolateral impingement is one of exclusion [1–4].

The MR findings of an abnormal soft-tissue mass or fibrous band in the anterolateral ankle gutter, distinct from the anterior talofibular ligament, suggest the diagnosis of anterolateral impingement [2, 3]. Controversies exist about the accuracy of MR imaging in the diagnosis of anterolateral impingement. Most authors believe that assessment of the anterolateral recess with conventional MR imaging is accurate only when a substantial joint effusion is present [1–4]. MR arthrography has been proven to be an accurate technique for assessing the presence of soft-tissue scarring in the anterolateral recess of the ankle and elucidating its extent in patients with anterolateral impingement before arthroscopy [2, 3] (Fig. 2). Another MR arthrography finding of anterolateral impingement is the absence of a recess of fluid between the anterolateral soft tissues and the anterior surface of the fibula. This absence may be due to the presence of adhesions and scar tissue that prevent fluid entering the normal recess between the fibula and the joint capsule [2, 3].

**Anterior Impingement Syndrome**

Anterior impingement is a relatively common cause of chronic pain in the ankle, espe-
MR Imaging of Ankle Impingement

Fig. 4.—26-year-old man with anterolateral impingement syndrome. A, Axial T1-weighted spin-echo MR image of right ankle shows nodular fibrous thickening of accessory fascicle of anterior tibiofibular ligament in superior aspect of anterolateral gutter (arrow). B, Arthroscopic image confirms diagnosis of anterolateral impingement syndrome caused by hypertrophy of accessory fascicle of anterior tibiofibular ligament (asterisk). L = lateral malleolus, T = talus.

Fig. 5.—Drawing shows abnormal conditions that characterize anterior ankle impingement including chondral fraying, anterior tibial and talar osteophytes (arrows), synovitis in anterior capsular recess (asterisk), reduction of joint space, and osteochondral loose bodies (arrowhead).

Cially in athletes subjected to repeated stress in ankle dorsiflexion, which is typical in soccer players [1, 2]. This condition involves a beaklike prominence at the anterior rim of the tibial plafond, usually associated with a corresponding area over the opposed margin of the talus proximal to the talar neck, well within the anterior ankle joint capsule (Fig. 5). These osteophytes can impinge on each other, especially with ankle dorsiflexion, and soft tissues can become entrapped. Anterior impingement syndrome may actually limit motion [1, 2].

The cause and origin of anterior impingement are uncertain, and many factors are probably involved. It has been suggested that forced dorsiflexion results in repeated microtraumas on the tibia and talus, leading to microfractures of trabecular bone or periosteal hemorrhage that then heals with the formation of new bone. Another mechanism suggested in the etiology of these lesions is forced plantar flexion trauma that causes capsular avulsion injury [1, 2].

Radiographs most often show anterior osteophytes, and lateral stress radiographs obtained in maximum dorsiflexion may show physical impingement of the osteophytes [1, 2].

MR imaging is useful in assessing the degree of cartilage damage and in detecting bone marrow edema and synovitis in the anterior capsular recess [2] (Fig. 6).

Anteromedial Impingement

Anteromedial impingement is an uncommon cause of chronic ankle pain that can be a result of a meniscoid lesion, which is represented by a soft-tissue thickening anterior to...
the tibiotalar ligaments [2, 7]. The anteromedial meniscoid lesion can appear isolated or arising from a partially torn deltoid ligament. Another reported cause of anteromedial impingement is a thickened anterior tibiotalar ligament [1, 2, 7]. This thickened ligament or a meniscoid lesion impinges on the anteromedial corner of the talus during dorsiflexion of the ankle, resulting in osteophyte formation, a chondral lesion, or both [1, 2, 7] (Fig. 7).

Anteromedial impingement is rarely an isolated condition but is most commonly associated with an inversion mechanism of injury with lateral and medial ligamentous injury [1, 2, 7].

Conventional MR imaging has not yet been proven useful in detecting medial impingement syndromes. MR arthrography is the imaging method of choice, clearly defining the medial meniscoid lesion (Fig. 8), the thickened anterior tibiotalar ligament, and any chondral or osteochondral associated lesions [2, 7].

**Posteromedial Impingement**

Posteromedial impingement is an uncommon cause of posteromedial ankle pain after a severe ankle inversion injury in which the deep posterior fibers of the medial deltoid ligament become crushed between the medial wall of the talus and the medial malleolus [8]. Initially, posteromedial symptoms do not predominate compared with the symptoms of the lateral ligament disruption, and they usually resolve without specific treatment. However, inadequate healing of the contused deep posterior deltoid ligament fibers may lead to chronic inflammation and hypertrophic fibrosis and metaplasia. In such cases, this disorganized fibrotic scar tissue may impinge between the medial wall of the...
talus and the posterior margin of the medial malleolus [8] (Fig. 9).

MR imaging can show the lesion, thickened soft tissues, and evidence of bone marrow edema of both the medial talus and medial malleolus [8] (Fig. 10).

**Posterior Impingement**

Posterior ankle impingement syndrome refers to a group of abnormal entities that result from repetitive or acute forced plantar flexion of the foot [9]. Different names have been given to posterior ankle impingement syndrome, including the os trigonum syndrome, talar compression syndrome, and posterior block of the ankle. The mechanisms of injury have been likened to a nut in a nutcracker because the posterior talus and surrounding soft tissues are compressed between the tibia and the calcaneus during plantar flexion of the foot [2, 9] (Fig. 11). This syndrome has been extensively described in classical ballet dancers, but it also has been recognized in individuals who are active in sports [2, 9].

The anatomy of the posterior aspect of the ankle is a key factor in the occurrence of posterior ankle impingement syndrome. The more common causes are osseous in nature, such as the os trigonum (an accessory ossicle of the lateral tubercle of the talus that may persist unfused into adulthood in 7% of individuals), an elongated lateral tubercle of the talus termed “Stieda’s process,” a downward sloping poste-
rior lip of the tibia, the prominent posterior process of the calcaneus, and loose bodies [2, 9] (Fig. 12). Soft-tissue causes of impingement encompass synovitis of the flexor hallucis longus tendon sheath, the posterior synovial recess of the subtalar and tibiotalar joints, and the posterior intermalleolar ligament.

Posterior ankle impingement syndrome may manifest as inflammation of the soft tissues of the posterior ankle, an osseous injury, or both (Fig. 13). The osseous injuries include fracture, fragmentation, and pseudoarthrosis of the os trigonum or lateral talar tubercle. As such, posterior ankle and subtalar synovitis as well as flexor hallucis longus tenosynovitis are soft-tissue changes associated with posterior ankle impingement syndrome [2, 9].

MR imaging is useful in establishing the diagnosis of posterior ankle impingement syndrome. This modality shows abnormal signal intensity in the lateral talar tubercle, the os trigonum, or both, consistent with bone marrow edema that is believed to be the result of bone impaction and thus represents bone contusions or occult fractures [2, 9] (Figs. 14 and 15). MR imaging also depicts inflammatory changes in the soft tissues of the posterior ankle—namely, the posterior synovial recess of the subtalar and tibiotalar joints and the flexor hallucis longus tendon sheath (Fig. 14). The combined presence of bone marrow edema and posterior ankle synovitis may suggest the diagnosis of posterior ankle impingement [2, 9].

Detection of an abnormal posterior intermalleolar ligament on MR imaging requires a thickened posterior intermalleolar ligament that can readily be separated from the surrounding posterior talofibular ligament and the transverse inferior tibiotalar ligament [10] (Figs. 16 and 17).

**Conclusion**

In the appropriate clinical settings, MR imaging and MR arthrography are useful
MR Imaging of Ankle Impingement

Fig. 11.—Diagram shows nutcracker phenomenon of posterior ankle impingement.

Fig. 12.—Diagrams show osseous anatomic structures involved in posterior impingement: 1 = Stieda’s process (arrow), 2 = os trigonum (arrow), 3 = fractured lateral tubercle of talus (arrow), 4 = prominent down slope in posterior tibial articular surface (arrowhead), 5 = calcified inflammatory tissue (arrowhead), and 6 = prominent superior surface of calcaneal tuberosity (arrowheads).

Fig. 13.—22-year-old male basketball player with posterior impingement. A and B, Axial (A) and sagittal (B) fat-suppressed proton density-weighted MR images of right ankle show abnormal high signal intensity in posterior aspect of talus and in os trigonum (arrows). Note joint effusion in posterior synovial recess of tibiotalar and subtalar joints. (Fig. 13 continues on next page)
Fig. 13. (continued)—22-year-old male basketball player with posterior impingement. C, Arthroscopic image shows removal of os trigonum (asterisk). T = talus, C = calcaneus.

Fig. 14.—42-year-old man with posterior impingement. Sagittal fat-suppressed proton density-weighted MR image of right ankle shows abnormal high signal intensity in os trigonum and posterior aspect of talus with associated tenosynovitis of flexor hallucis longus (asterisks).

Fig. 15.—34-year-old man with posterior impingement. A, Sagittal T1-weighted MR image of left ankle shows prominent lateral tubercle of talus (Stieda’s process) with low signal intensity (arrow). B, Sagittal fat-suppressed proton density-weighted MR image shows bone marrow high signal intensity in Stieda’s process (arrow) and in dorsal aspect of calcaneus (arrowheads). Associated inflammation in adjacent soft tissues is present.
techniques for assessing the soft-tissue and osseous disorders present in the impingement syndromes of the ankle and for detecting other potential causes of ankle pain.

References