

Ankle Impingement Syndromes

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KEYWORDS

- Ankle impingement • MR imaging • Ultrasound
- Athletic injury

The ankle impingement syndromes are defined as pathologic conditions causing painful restriction of movement at the tibiotalar joint caused by osseous or soft tissue overgrowth or by the presence of accessory ossification centers. First described by Morris¹ in 1943 and then by McMurray² in 1950, who termed the condition “footballer’s ankle,” ankle impingement is now an established cause of ongoing ankle dysfunction, often following seemingly trivial trauma. Originally studied in athletes, impingement is now also acknowledged as a cause of persisting symptoms in the general population. Ankle sprains are common, and although in most no significant sequelae develop, 15% to 20% of sports injuries result in continuing symptoms caused by a range of chronic and subacute pathologies including osteochondral defects, tendon injuries, mechanical instability, and impingement.³

Classification of the ankle impingement syndromes is anatomic according to their relationship to the tibiotalar joint. Each anatomic site may have similar injury etiology, but presents with differing clinical signs and symptoms and imaging findings.

ANTERIOR IMPINGEMENT SYNDROME

Anterior impingement is a relatively common cause of chronic ankle pain seen particularly in such athletes as ballet dancers and soccer players.^{4–6} Symptoms are generally progressive and are caused by impingement of hypertrophied soft tissue and bony spurs within the anterior ankle joint.

Anatomy and Pathophysiology

Characteristically, there is formation of bony prominences at the anterior rim of the tibial plafond and at the opposing aspect of the talus. Originally, these bony spurs were thought to represent a response to capsular traction during plantar flexion.^{1,2} Anatomic studies have demonstrated, however, that these anterior tibiotalar spurs are formed within the anterior ankle joint capsule (**Figs. 1** and **2**), rather than at the site of capsular insertion, making repetitive capsular traction an unlikely etiology.^{4,7}

These bony spurs are thought to develop in response to repetitive trauma to the anterior articular cartilage rim either from repeated dorsiflexion when there is impaction between the anterior tibia and talus,^{4,7} or from external direct trauma to the anterior ankle joint as is seen during ball strike in soccer players. Evaluation of ankle biomechanics during ball strike has revealed that spurs typically form at the location of ball impact, suggesting that the spurs develop in response to the direct trauma rather than capsular traction.⁶ Microfractures of trabecular bone and periosteal hemorrhage resulting from the repetitive trauma heal to form new bone that develops into the characteristic bony spurs. In addition to injury to the bony articular rim, trauma to the anterior chondral margin of the ankle joint may lead to reparative formation of fibrous tissue and subsequent ossification.⁸

In addition to the osseous element of anterior impingement there is an important associated soft tissue component.⁷ Bony spurs have been noted to be present in both athletes and

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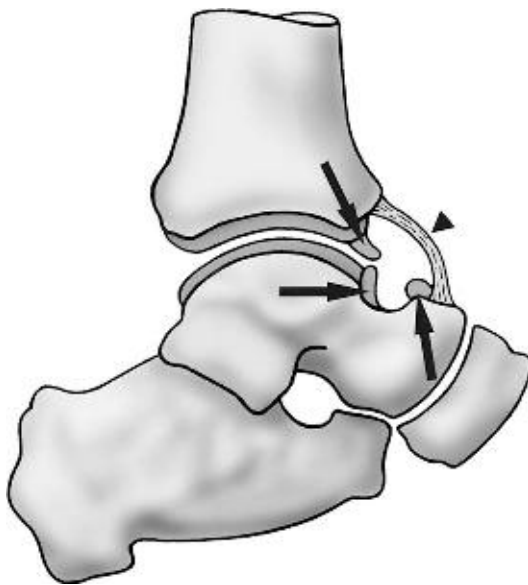


Fig. 1. Diagrammatic representation of the ankle shows the most common sites for anterior tibiotalar spur development (arrows) within the joint capsule (arrowhead).

nonathletes who have no symptoms of anterior ankle impingement.^{4,7,9} In addition, postexcision recurrence of the bony spurs is not accompanied by recurrence of symptoms indicating the



Fig. 2. Lateral radiograph shows anterior tibiotalar spurs (arrows) in a soccer player with clinical anterior impingement.

significance of the associated soft tissue abnormality in precipitating the clinical syndrome.^{8,10}

Clinical Features

Typically, anterior impingement syndrome presents with anterior ankle pain exacerbated by dorsiflexion. Clinical examination may reveal soft tissue swelling over the anterior aspect of the ankle joint with reduced range of dorsiflexion. Movement limitation can sometimes be overcome by excessive ankle pronation, but this additional abnormality in ankle biomechanics may have further consequences.⁵ In some instances the anterior bony spurs may be palpable.

Imaging

The diagnosis of anterior impingement is primarily a clinical one. Imaging can exclude other causes of ankle pain, such as osteochondral defects or fracture, and conventional radiography can confirm the presence of anterior tibiotalar spurs, although anteromedial spurs may be more difficult to identify. Combining a lateral radiograph with an oblique anteromedial impingement radiograph has been shown to increase the sensitivity of plain radiography, but there is a concomitant decrease in specificity.¹¹ Although there is a grading system for anterior impingement based on clinical findings and the size of spurs on conventional radiography,¹² these features have not been shown to correlate well with outcome. The absence of preoperative degenerative changes within the ankle joint is a more consistent indicator of likely treatment success. Several studies have demonstrated that patients with associated ankle joint degenerative changes are significantly more likely to have poor long-term outcome following surgical treatment.^{8,10} Further imaging is often unnecessary but, particularly if coexistent ankle pathology is suspected, MR imaging may be useful.¹³ As in anterolateral impingement demonstration of extensive bone marrow edema is uncommon, but the extent of synovitis and joint capsule thickening may be evident (Fig. 3).¹⁴

Treatment

Conservative management including limitation of symptom-provoking activity combined with physiotherapy is successful in most patients. Particularly in ballet dancers this should be performed in conjunction with correction of technique to correct overpronation where appropriate.

Surgical intervention is generally reserved for those cases where conservative therapies have failed to provide adequate relief. Long-term follow-up studies have shown open and arthroscopic



Fig. 3. Sagittal T2-weighted fat-suppressed MR image shows anterior tibial and talar spurs (arrows), anterior effusion, and irregular capsular thickening (arrowhead).

excision of the bony spurs, hypertrophic synovium, and scar tissue to be effective treatments for anterior impingement providing there is no evidence of preoperative joint space narrowing.^{8,10,15,16} With either technique a return to full activity is expected, even in elite athletes.

ANTEROLATERAL IMPINGEMENT

Anterolateral impingement is a relatively uncommon cause of anterolateral ankle pain caused by entrapment of hypertrophied soft tissues within the anterolateral recess of the ankle.

Anatomy and Pathophysiology

The anterolateral recess is triangular in cross-section and is limited posteriorly by the anterolateral tibia and talus, anteromedial fibula, and anteriorly by the joint capsule, the anterior inferior tibiofibular, anterior talofibular, and calcaneofibular ligaments (**Fig. 4**).¹⁷ Classically anterolateral impingement syndrome is described in young athletic patients who have experienced a relatively minor injury to the anterolateral ankle ligaments or the joint capsule, either as a single inversion injury or because of repetitive plantar flexion and inversion.^{18,19} The initial sprain may be minor and not recalled. Despite no initial apparent sequelae, recurrent subclinical instability and associated microtrauma may cause intra-articular and soft tissue hemorrhage, localized reactive synovial hyperplasia, and scarring. Compression of the abnormal anterolateral soft tissue during



Fig. 4. Illustration of anterolateral anatomy of the ankle. Anterolateral recess (thick arrow) sited between anterior tibiofibular ligament (arrowheads) and anterior talofibular ligament (thin arrow). Note calcaneofibular ligament (curved arrow).

eversion or dorsiflexion causes symptoms of impingement. In advanced cases the soft tissue can become molded within the anterolateral recess to form a reactive hyalinized connective tissue mass termed the “meniscoid lesion” by Wolin and colleagues.²⁰

Anterolateral impingement has also been described in a subset of individuals with a distal or accessory fascicle of the anterior inferior tibiofibular ligament.^{17,21} This ligament is multifascicular and may be present as two or three separate bands extending from the anterior aspect of the lateral malleolus to the anterolateral tubercle of the tibia. An accessory fascicle has been identified in 21% to 97% of patients as a band oriented in parallel to the main ligament but separated from it by a fibrofatty septum.²² The apparent disparity in incidence is likely to be caused by lack of consensus as to the definition of a separate fascicle but its presence is thought to represent a normal anatomic variant. Contact between the anterolateral corner of the talus and the anteroinferior tibiofibular ligament may be normal in some cases, but if the anterolateral ankle is lax this allows increased pressure because of anterior extrusion of the talar dome in dorsiflexion. This potentially initiates inflammatory changes within the ligament with resultant hypertrophy and injury, predisposing it to impingement. Chondral injury of the anterolateral corner of the talus caused by abrasion from the impinging anteroinferior tibiofibular ligament can be identified at arthroscopy and on imaging.^{17,21}

Clinical Features

Clinical assessment is reasonably accurate in the diagnosis of anterolateral impingement.²³ Anterolateral ankle pain with tenderness and swelling are suggestive of the diagnosis but can be present in other entities, such as peroneal tendon pathology, sinus tarsi syndrome, stress fractures, chronic ankle instability, and intra-articular loose bodies, many of which may also develop subsequent to ankle trauma.

Several clinical tests have been described but symptoms exacerbated by single leg squatting and ankle eversion or dorsiflexion have been shown retrospectively to correlate best with positive findings of impingement at arthroscopy.²⁴ Molloy and colleagues²⁵ have described a clinical sign elicited by impinging abnormal hypertrophic synovium within the tibiotalar joint causing pain. They report a 94.8% sensitivity and 88% specificity in a prospective study of this lateral impingement test. The subset of individuals with impingement secondary to an accessory anteroinferior tibiofibular ligament may describe a popping sensation or an audible pop on ankle dorsiflexion and eversion.

Imaging

A study of ultrasound assessment in patients with clinical anterolateral impingement identified a nodular, mixed echogenic, synovitic mass within the anterolateral recess of 100% of eight patients (Fig. 5A).²⁶ The mass could be extruded anteriorly with manual compression and in the study group measured greater than 1 cm diameter. Power Doppler interrogation was found to be unhelpful. Importantly, findings were not dependent on the presence of an ankle joint effusion, unlike MR imaging.²⁷ Ultrasound also identified bony spurs and injury to the anterior talofibular ligament (Fig. 5B). Similar findings were seen in two of the control group, however, where anterolateral masses were identified in patients without accompanying symptoms.²⁶ A similar study of 14 sportsmen, however, concluded that ultrasound evaluation was nonspecific with changes demonstrated within the anterior talofibular ligaments of all subjects.²⁸

The predominating soft tissue changes and lack of a bony component to anterolateral impingement makes plain radiography and conventional CT assessment unhelpful. A study of CT arthrography showed nodular fraying or thickening, seen best on coronal images that correlated well with soft tissue impingement at arthroscopy.²⁹

The use of conventional MR imaging in anterolateral impingement remains controversial. Some

authors have found it to be beneficial, but several studies have found conventional MR imaging to be unhelpful.^{30–33} Thickening of the anterior talofibular ligament and lateral gutter fullness is suggestive of the diagnosis and is most reliably demonstrated on T1 axial imaging (Fig. 5; Fig. 6).^{18,34} Confirmation of axial findings using a sagittal T1 sequence has been suggested, because displacement of the normal fat anterior to the fibula by synovitis or scar tissue can be evaluated (Fig. 5D).³⁴ Most groups, however, believe that conventional MR imaging is only reliably diagnostic in the presence of an ankle effusion.^{14,27}

MR arthrography has proved to be an accurate method of evaluation of the anterolateral recess. Positive findings of anterolateral scarring and synovitis can be identified as capsular adherence to the fibula and tibia, presumably because of adhesion formation (Fig. 6C). As in ultrasound assessment, clinical correlation is essential because these features can also be seen without symptoms of anterolateral impingement. MR arthrography also demonstrates additional pathology, such as abnormalities of the anterior talofibular ligament, chondral damage, and bony spurs.²³

Management

As in other impingement syndromes, initial treatment is conservative with immobilization, physiotherapy, and nonsteroidal anti-inflammatory medication, reserving surgery for resistant cases.^{24,35} Dry needling with injection of steroid and local anesthetic can be performed under ultrasound guidance allowing a return to previous levels of activity even in elite athletes, but this technique has not been evaluated in the literature. Arthroscopic resection of hypertrophic synovium and scar tissue gives symptomatic and functional improvement and allows resection of a distal fascicle of the anterior talofibular ligament when that is the underlying etiology (Fig. 6B).

ANTEROMEDIAL IMPINGEMENT

An uncommon cause of chronic ankle pain, anteromedial impingement rarely occurs as an isolated finding.^{36,37}

Anatomy and Pathophysiology

First described in a case report of an anteromedial meniscoid lesion similar to that seen in anterolateral impingement, the initial proposed mechanism of injury was eversion of the ankle joint with resultant traction of the anterior tibiotalar ligament.³⁸ Subsequent surgical and radiologic series, although

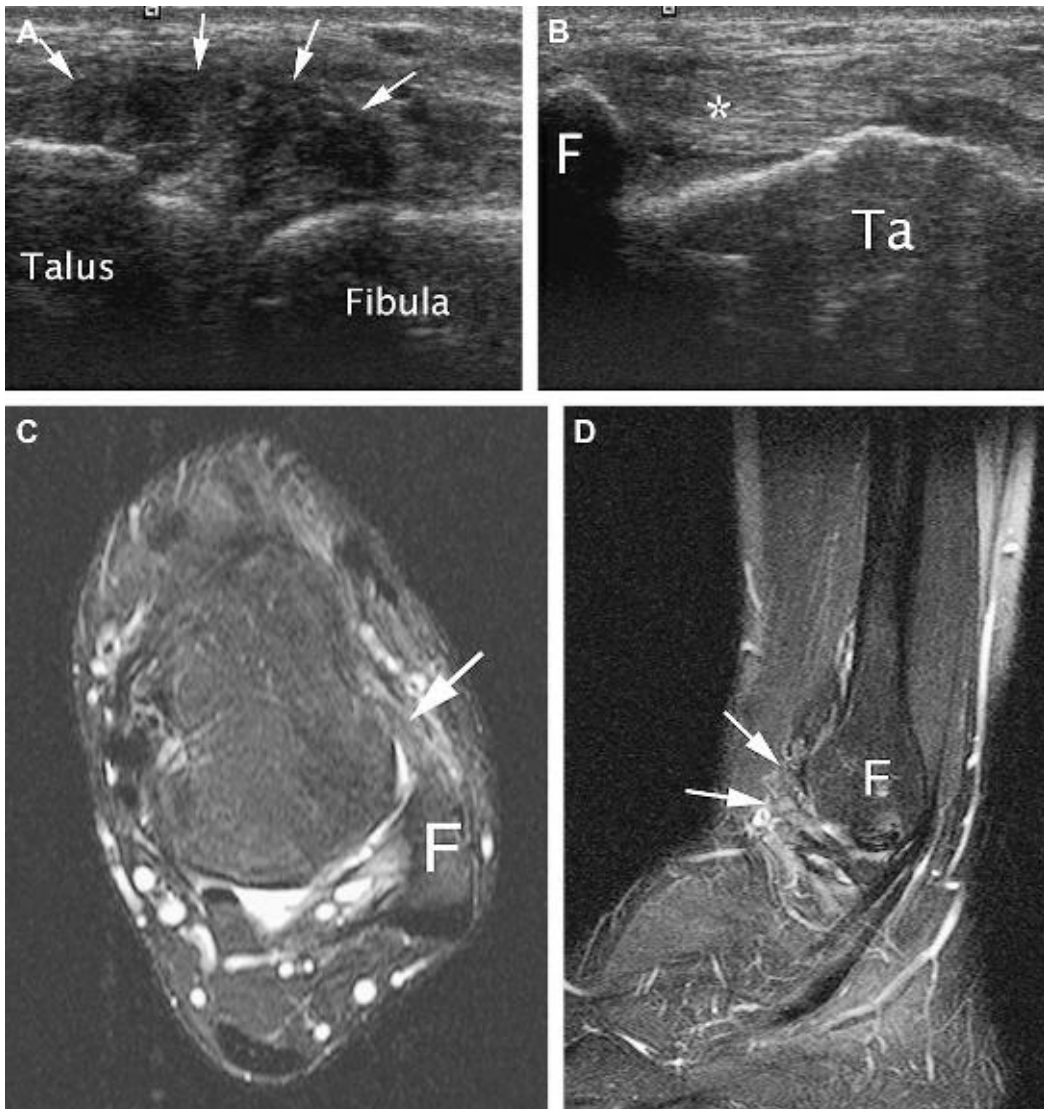


Fig. 5. Patients with clinical anterolateral impingement. Axial ultrasounds show (A) hypoechoic nodule (arrows) in anterolateral recess and (B) thickening of anterior talofibular ligament (*) F, fibula; Ta, talus axial (C) and sagittal (D) T2-weighted fat-suppressed MR images show anterolateral synovitis (arrows) anterior to the fibula (F).

not large, have suggested that anteromedial impingement instead represents a rare complication of inversion trauma at the tibiotalar joint, perhaps with a rotational component.^{36,37} As in anterolateral impingement it is thought that subsequent microtrauma and healing following the original inversion injury initiates synovial, ligamentous, and capsular thickening within the anteromedial compartment that can become compressed during dorsiflexion and inversion.

Clinical Features

Patients describe focal anteromedial tenderness and pain that is exacerbated by dorsiflexion and

inversion, clinical examination shows restriction of these movements. There may be associated soft tissue thickening.

Imaging

Although in some patients bony spurs have been shown to be a feature of anteromedial impingement,³⁶ they are not the dominant feature and so plain radiography and conventional CT assessment are usually not of use.

There have been no imaging studies assessing the use of ultrasound or conventional MR imaging in anteromedial impingement. In the largest clinical series 2 of 11 patients underwent MR imaging that

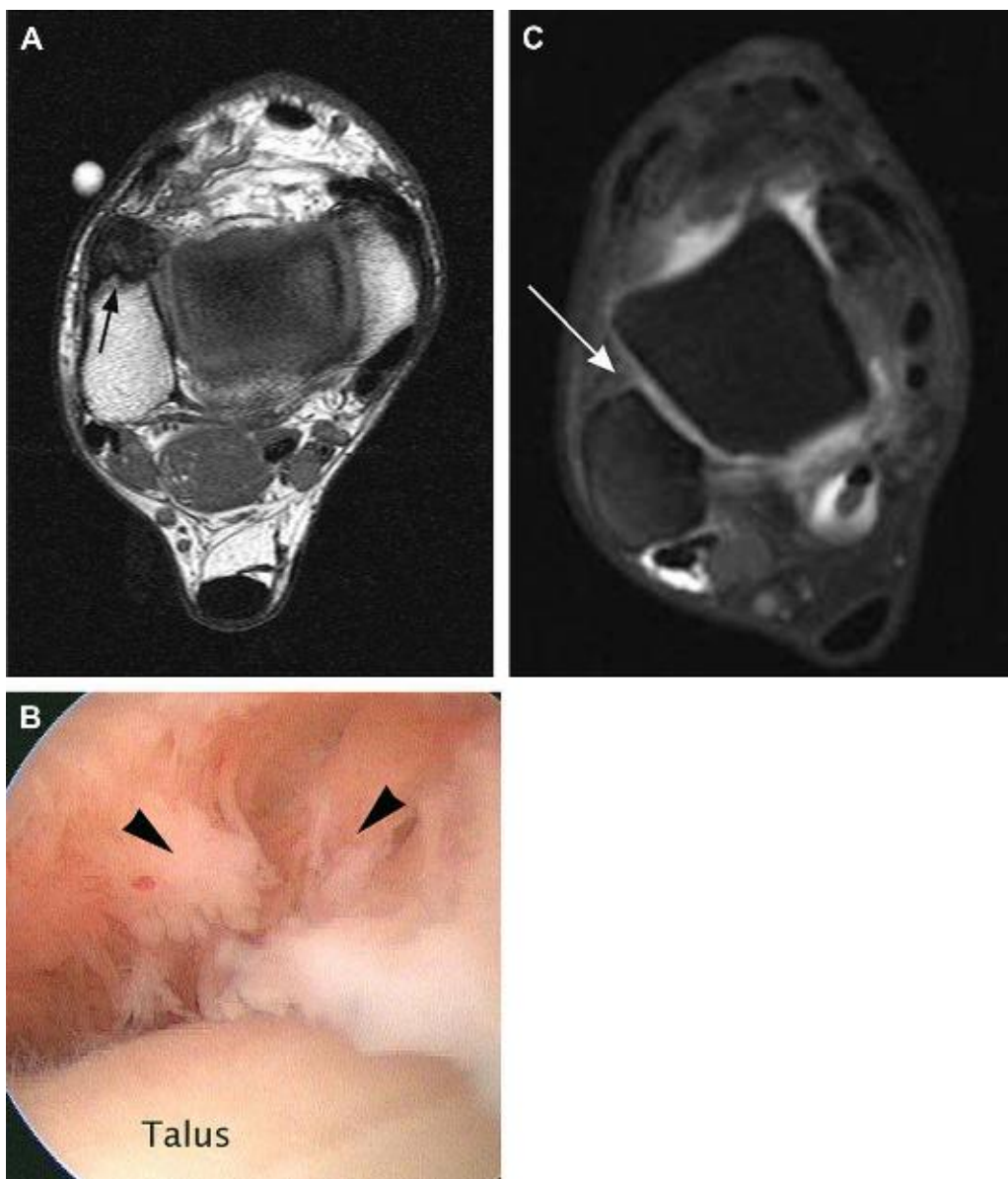


Fig. 6. Patients with clinical anterolateral impingement. (A) Axial T1-weighted MR image shows irregular soft tissue thickening (*arrow*) in anterolateral recess. (B) Corresponding arthroscopic image shows synovitis and nodularity (*arrowheads*) within anterolateral recess. (Courtesy of Steve Bollen, MD, Bradford, UK.) (C) Axial T1-weighted fat-suppressed MR arthrogram image shows capsular adherence with no recess of fluid anterior to the fibula (*arrow*).

was thought to be inconclusive. Perhaps unsurprisingly given the proposed mechanism of injury, five cases were found at surgery to have lateral ligamentous injury.³⁶ A prospective evaluation of MR arthrography in two patients with clinical anteromedial impingement demonstrated irregular soft tissue thickening anterior to the anterior tibiotalar ligament in both patients, which was shown to represent synovitis at subsequent arthroscopy (**Fig. 7**). One patient also had an anteromedial joint

capsule tear diagnosed at MR imaging arthrography and confirmed surgically.³⁷ In some patients, bony spurs have been shown to be a feature of anteromedial impingement and have been described on imaging and at surgery.

Management

There are currently no cases describing nonsurgical treatment of anteromedial impingement in the

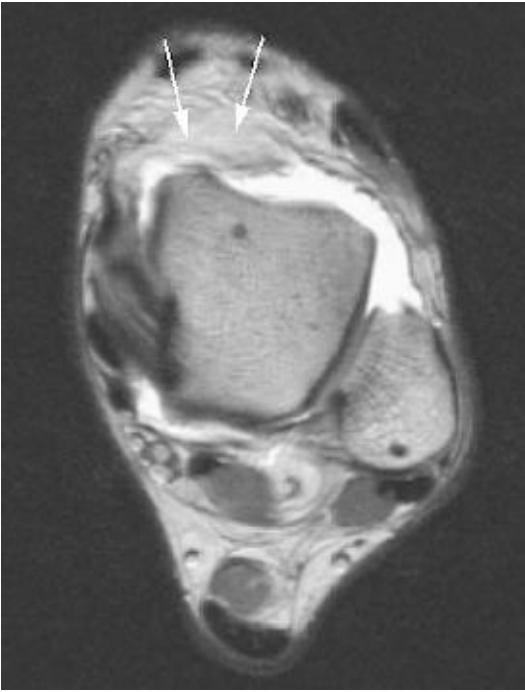


Fig. 7. Axial T1-weighted MR arthrogram image from patient with anteromedial impingement shows irregular soft tissue thickening within anteromedial ankle joint (arrows).

literature. Surgical excision of abnormal synovial tissue and bony spurs gives symptomatic relief and functional improvement.^{35,36,38}

POSTEROMEDIAL IMPINGEMENT

Until recently posteromedial impingement was one of the least described ankle impingement syndromes and accordingly its etiology was less well understood. Although there are few reported series they have identified relatively characteristic imaging findings.

Anatomy and Pathophysiology

Surgical and radiologic studies have identified posteromedial impingement following severe inversion injury at the ankle joint.^{39,40} Injury of the anterior talofibular ligament during inversion allows compression of posteromedial structures including the posteromedial joint capsule, posterior tibiotalar ligament, and posteromedial flexor tendons between the medial wall of the talus and the medial malleolus (**Fig. 8**). In some cases inadequate healing of posteromedial soft tissues forms thickened and disorganized fibrous tissue within the posteromedial ankle joint, which impinges

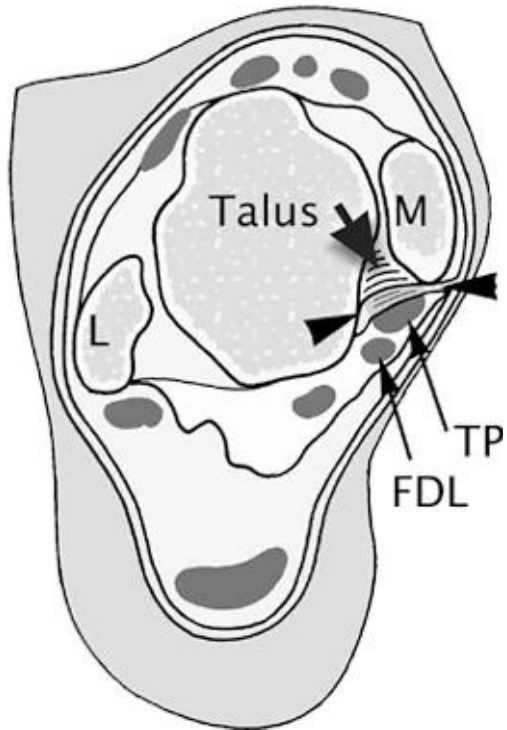


Fig. 8. Axial illustration shows structures involved in posteromedial impingement (see text). M, medial malleolus; TP, tibialis posterior tendon; FDL, flexor digitorum longus tendon; L, lateral malleolus. Arrowheads denote posterior fibers of tibiotalar ligament; short arrow indicates deep fibers of tibiotalar ligament.

between the posterior aspect of the medial malleolus and the medial talus.³⁹

Clinical Features

Following the initial injury lateral symptoms predominate; as these settle there is insidious onset of posteromedial and medial activity-related pain, typically after 4 to 6 weeks.³⁹ There is focal posteromedial tenderness on examination and pain can be elicited by posteromedial palpation during plantar flexion and inversion. Similar symptoms may also be found in early tibialis posterior dysfunction, although patients have no history of trauma. Distinction between posteromedial impingement and tibialis posterior dysfunction is clinical; in the latter patients describe progressive pes planus and are restricted in their ability to stand on tiptoe with loss of normal heel inversion during this maneuver.

Imaging

Because soft tissue abnormality is the basis of posteromedial impingement, plain radiography

and CT are usually unhelpful but very rarely accessory medial talar tubercle ossification can contribute to soft tissue thickening (**Fig. 9**).

A single study identified increased radionuclide uptake in the region of the posteromedial ankle on isotope bone scanning; however, bone scanning was unable to define further associated injuries subsequently identified and treated at arthroscopy.⁴¹

Ultrasound and MR imaging have been jointly assessed in radiologic studies of patients with clinically diagnosed posteromedial impingement. Messiou and colleagues⁴⁰ imaged nine elite athletes 4 to 6 weeks following injury using both modalities as did Koulouris and colleagues⁴¹ who assessed 25 patients greater than 1 year following initial trauma. Despite small numbers of subjects, these studies revealed relatively consistent findings on MR imaging and on ultrasound. MR imaging demonstrated loss of the normal fat striations within the posterior tibiotalar ligament, posteromedial synovitis, and abnormal signal and thickening within the posteromedial joint capsule that in some cases displaced or surrounded the adjacent tendons (**Fig. 9B**; **Fig. 10A**).^{40,41} Posteromedial capsulitis, however, was also identified in the control group of one study. These patients clinically had posterolateral impingement and no symptoms or

signs of posteromedial impingement.⁴⁰ A total of 100% of patients in both studies were shown to have posteromedial abnormalities on ultrasound, either thickening of the posteromedial capsule or posteromedial synovial hypertrophy and, as on MR imaging, displacement or entrapment of adjacent tendons was also elicited (**Fig. 10B**).^{40,41} It is possible that the apparent differences in scarring around the tendons between the two studies are related to differences in timing of imaging following the initial trauma and reflect the natural history and development of posteromedial impingement syndrome.

Management

In addition to identifying posteromedial impingement, MR imaging helps to exclude or confirm other pathologies particularly within the anterolateral ankle.^{40,41} Clinical correlation is essential, because imaging findings do not always reflect symptomatic impingement.⁴⁰ Ultrasound-guided dry needling of the capsular abnormality with injection of steroid and local anesthetic, in most cases, allows a return to previous levels of activity even in elite athletes (**Fig. 10**).⁴⁰ Successful outcome has also been demonstrated following surgical resection of abnormal posteromedial soft

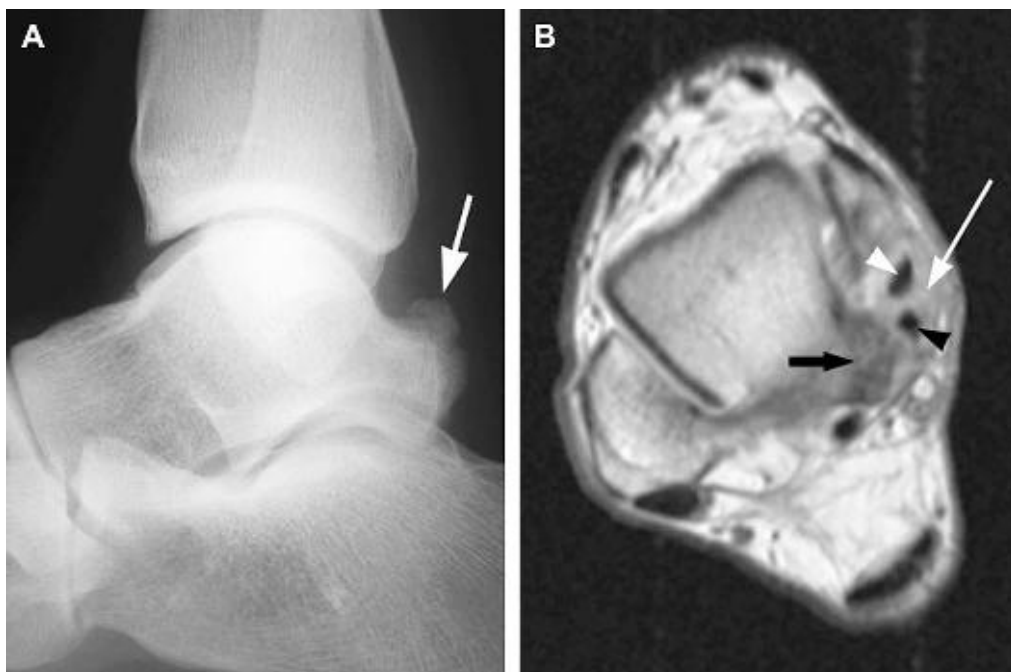


Fig. 9. Rugby player with posteromedial impingement. (A) Lateral radiograph shows accessory medial talar tubercle (arrow). (B) Axial proton-density weighted MR image shows accessory medial talar tubercle (black arrow). Posteromedial soft tissue thickening (white arrow) encases tendons of tibialis posterior (white arrowhead) and flexor digitorum longus (black arrowhead).

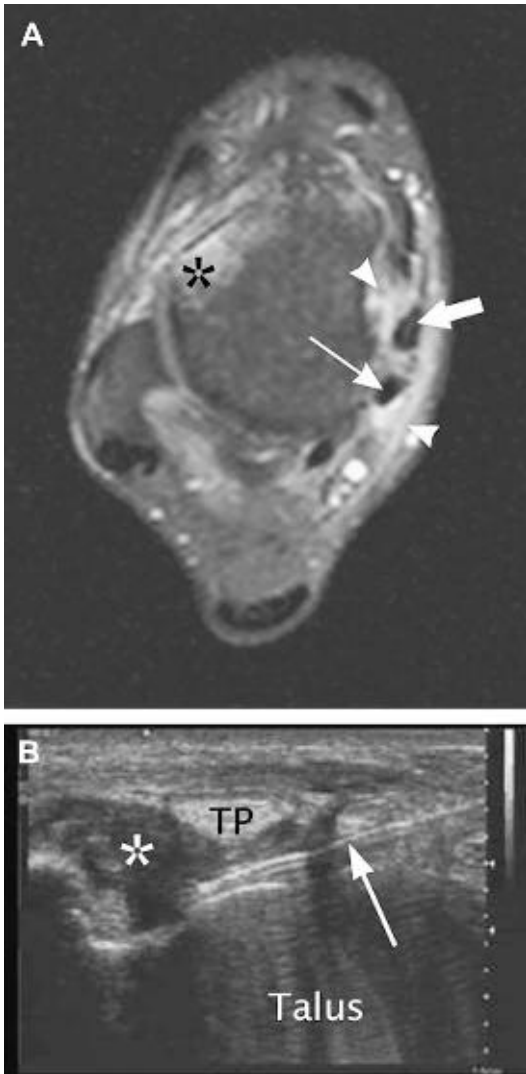


Fig. 10. Soccer player with clinical posteromedial impingement. (A) Axial T1 fat-suppressed image following IV gadolinium shows posteromedial synovitis (arrowheads) displacing tibialis posterior (thick arrow) and flexor digitorum longus (thin arrow) tendons. Note resolving anterolateral talar injury (*). (B) Axial ultrasound image of posteromedial recess during guided injection (arrow) shows displacement of tibialis posterior (TP) by hypoechoic synovitis (*).

tissue without ligamentous repair,^{39,41} but should be reserved for cases resistant to ultrasound-guided therapy.

POSTERIOR IMPINGEMENT

Posterior impingement syndrome encompasses a group of pathologies that are characterized by posterior ankle pain in plantar flexion. Symptoms result from compression of the talus and soft

tissues between the posterior tibia and calcaneum. Other terms, such as “os trigonum syndrome,” “talar compression syndrome,” and “posterior block,” have also been used to describe the same collection of signs and symptoms. Posterior ankle pain is a common symptom and may be caused by a range of soft tissue and osseous abnormalities. Overall, pathology of the Achilles tendon is the most common cause of posterior ankle pain but similar symptoms are also experienced with flexor hallucis longus tendonopathy, osteochondritis dissecans, retrocalcaneal bursitis, and tarsal tunnel syndrome.

Anatomy and Pathophysiology

Pathology related to the os-trigonum-talar process is the most common cause of posterior impingement and has been extensively studied. The posterior talar process extends posteromedial to the tibiotalar joint and has two projections. The smaller medial process is separated from the larger lateral tubercle by a shallow groove that contains the flexor hallucis longus tendon. Ossification of the talar body is evident from 6 months of fetal development. The posterior talus ossifies from a secondary ossification center, mineralization starts between 7 and 13 years, and generally fusion with the talar body occurs within 12 months to form the lateral tubercle. In some individuals the lateral tubercle is particularly elongated and is termed the “Steida process.” Failure of fusion between the talar body and the lateral tubercle occurs in 14% to 25% of the normal population⁴² and is bilateral in 1.4%. The ossicle formed is known as the “os trigonum” and articulates with the main body of the talus at a cartilaginous synchondrosis (Fig. 11). Although the os trigonum appears round or oval on radiographs it is, as its name suggests, triangular in shape having three articular surfaces.⁴² The anterior articular surface articulates with the talar body, the posterior facet gives attachment for the posterior talofibular and posterior talocalcaneal ligaments,⁴³ and the inferior aspect may articulate with the superior surface of the calcaneum.

Although the presence or absence of a Steida process or os trigonum is important in posterior impingement, the articular surface of the posterior tibia and the calcaneal tuberosity are also involved in the impingement mechanism. The posterior tibial articular surface, or posterior malleolus, may have a more or less downward sloping configuration and in some individuals the posterior process of the calcaneum may be prominent.

Soft tissue impingement may involve the posterior capsule, the posterior talofibular, and posterior



Fig. 11. Lateral radiograph shows os trigonum (arrow) separated from talus by cartilaginous synchondrosis (arrowheads).

intermalleolar and tibiofibular ligaments, any of which can become compressed between the posterior tibial plafond and the superior aspect of the calcaneum. The flexor hallucis longus tendon runs between the medial and lateral talar tubercles where it is held in a fibro-osseous tunnel by the overlying extensor retinaculum. Injury to the tendon as part of posterior impingement is typically, but not exclusively, seen in ballet dancers and can result in tenosynovitis; in extreme cases the tendon can become tethered.^{44,45}

The posterior intermalleolar ligament is an anatomic variant of the posterior ligaments of the ankle. The ligament extends obliquely from the posterior margin of the medial malleolus to the superior margin of the fibular malleolar fossa between the inferior transverse ligament and the posterior talofibular ligament (**Fig. 12A**). Cadaveric studies suggest it is present in 56% of individuals, although it was identified in only 19% of MR images of asymptomatic patients (**Fig. 12B**).⁴⁶ During its course the posterior intermalleolar ligament can herniate into the posterior ankle joint and bucket handle tears and entrapment of the ligament have been described as a cause of posterior impingement in ballet dancers.⁴⁷

Whatever the underlying anatomy, the typical etiology for the development of posterior ankle

impingement is chronic repetitive stress in plantar flexion. This is commonly encountered in ballet dancers because of the forced plantar flexion position required en pointe, and in this group posterior impingement has been extensively described.^{43–45,47,48} Other athletes, such as soccer players and downhill runners, are also prone to posterior impingement because of stresses placed on the ankle joint during sporting activity. Soccer players experience forced plantar flexion during ball strike; the impact of the ball on the anterior ankle is a cause of anterior impingement but the compression of posterior structures also means this group of athletes is also at risk of posterior impingement.⁴⁹ Although posterior impingement is generally diagnosed because of repetitive stresses on the posterior ankle, the syndrome is also encountered following acute trauma. Forced plantar flexion can cause fracture of the lateral talar tubercle or separation of the cartilaginous synchondrosis if an os trigonum is present. Soccer players are prone to inversion injury with the ankle in a neutral position that places strain on the calcaneofibular and posterior talofibular ligaments and can result in posterior impingement subacutely.⁴⁹

Clinical Features

The predominant symptom is progressive posterior ankle pain. Patients may also complain of mild posterior swelling. Tenderness can be elicited on palpation of the posterolateral ankle between the Achilles and peroneal tendons.^{48,49} On examination, pain is reproduced by plantar flexion or by dorsiflexion of the great toe. Associated tethering of the flexor hallucis longus tendon within the fibro-osseous tunnel behind the talus causes restricted plantar and dorsiflexion movement of the great toe itself.⁴⁸

Imaging

Plain radiography may demonstrate a Steida process or an os trigonum (see **Fig. 11**) that may or may not be the source of symptoms; however, it is not always possible to differentiate between a fractured lateral tubercle and an os trigonum even using more complex imaging.^{48,50} Lateral radiographs taken in plantar flexion may reveal impingement of the lateral tubercle–os trigonum between the posterior tibia and the calcaneal tuberosity, although the size of the os trigonum is not a good predictor of symptoms.⁵¹ Improved bony detail may be displayed using CT that more accurately demonstrates fractures of the lateral tubercle or separation at the cartilaginous synchondrosis.^{50,52} More recently ultrasound has been used to interrogate the ankle in soccer players with

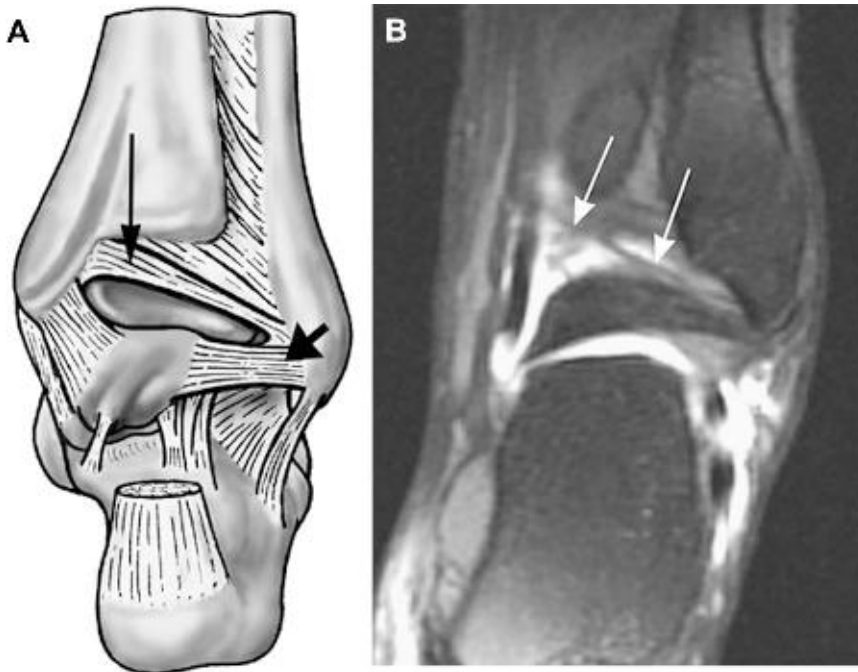


Fig. 12. (A) Illustration of posterior ankle shows posterior intermalleolar (*long arrow*) and posterior talofibular ligaments (*short arrow*). (B) Coronal T2-weighted MR image shows normal posterior intermalleolar ligament (*arrows*).

posterior impingement. This revealed nodular, hypoechoic thickening of the posterolateral joint capsule in 100% of 10 patients (**Fig. 13**).⁴⁹

Posterolateral capsule thickening and synovitis within the posterior ankle is seen on MR imaging, which may also demonstrate osseous abnormalities, such as bone marrow edema, fragmentation of the lateral tubercle–os trigonum, and the presence of a pseudoarthrosis (**Figs. 14–16**).⁵⁰ Fluid within the flexor hallucis tendon sheath can also be identified (**Fig. 14B**);⁵⁰ however, care must be taken in interpretation of this finding because in 20% of normal individuals the tendon sheath communicates with the ankle joint and, not infrequently, a sizeable volume of fluid is seen in asymptomatic individuals.⁵³ A differential quantity of fluid above and below the level of the posterior talus has been suggested as the most likely finding consistent with entrapment of the flexor hallucis tendon.⁵¹ Conventional MR imaging may also detect thickening of the posterior intermalleolar ligament, but this requires separate identification of the adjacent posterior talofibular and inferior tibiofibular ligaments and this is not always possible.⁵⁴

Management

Initial management of posterior impingement is conservative. Confirmation of the os trigonum

synchondrosis as the site of pain can be achieved by injection of local anesthetic under fluoroscopic guidance if there is clinical uncertainty as to the diagnosis (**Fig. 17**).⁵⁵ Ultrasound-guided dry needling combined with local anesthetic and corticosteroid has been shown to be successful in the treatment of posterior impingement syndrome in soccer players, particularly in the absence of an os trigonum (see **Fig. 13**).⁴⁹ Surgical excision of

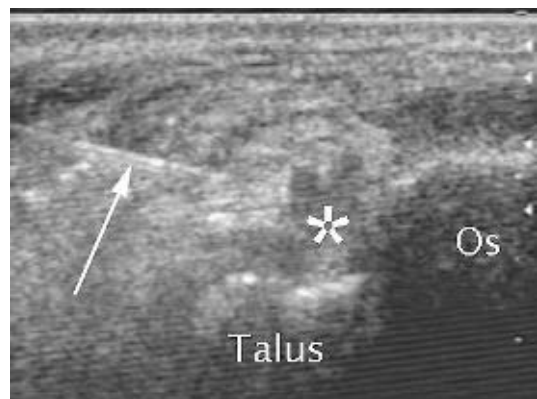


Fig. 13. Nonsurgical injection treatment of soccer player with clinical posterior impingement. Axial ultrasound shows nodular hypoechoic synovitis (*), os trigonum (Os), talus, and needle placement (*arrow*).

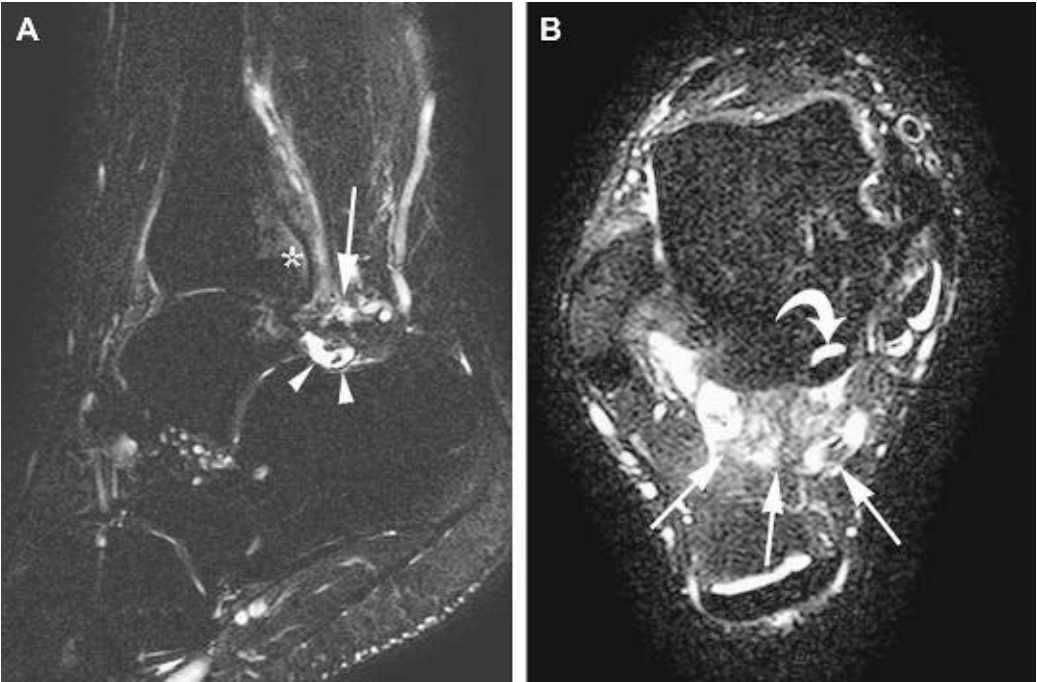


Fig. 14. Ballet dancer with clinical posterior impingement. (A) Sagittal T2-weighted fat-suppressed MR image with the ankle in dorsiflexion to simulate en pointe position shows bone marrow edema in posterior tibia (*), joint effusion (arrowheads), posterior recess nodularity, and thickening (arrow). (B) Axial T2-weighted fat-suppressed MR image shows marked nodular synovitis (arrows) and minor fluid around flexor hallucis longus tendon (curved arrow).

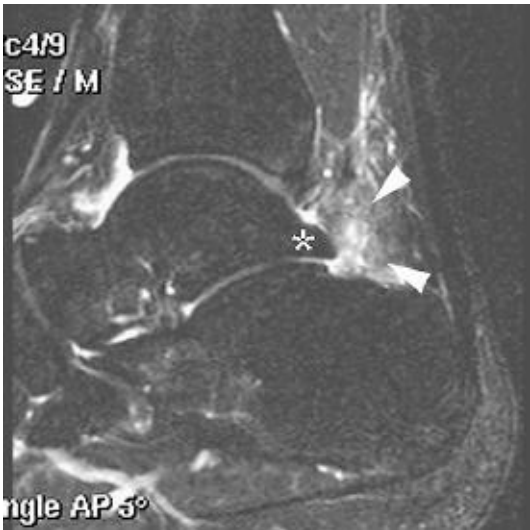


Fig. 15. Soccer player with posterior impingement. Sagittal T2-weighted fat-suppressed MR image shows steida process (*) and posterior capsular synovitis (arrowheads).



Fig. 16. Soccer player with posterior impingement. Sagittal T1-weighted fat-suppressed MR image following IV gadolinium shows os trigonum (*), cartilaginous synchondrosis (arrowhead), and posterior capsule enhancement (arrows).

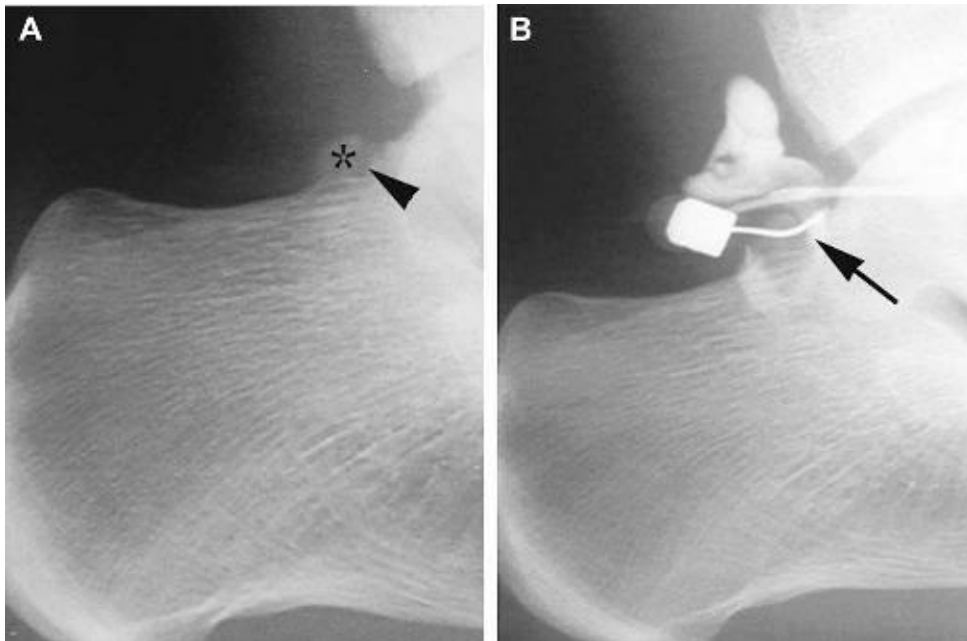


Fig. 17. Fluoroscopic-guided injection into os trigonum synchondrosis. (A) Os trigonum (*) and synchondrosis (arrowhead). (B) Contrast within synchondrosis (arrow).

osseous elements and any soft tissue component has a good prognosis in patients who fail to respond satisfactorily to conservative measures and can be combined with release of the flexor hallucis tendon if involved.^{43,48}

SUMMARY

The ankle impingement syndromes are an established cause of ankle dysfunction within the general population and within the athletic community. In many cases the diagnosis is clinical, although imaging has a significant role to play particularly in the exclusion of alternative or concomitant pathology or when the diagnosis is in doubt. For most patients conservative management or nonsurgical intervention allows resumption of their previous level of activity, even in elite athletes. Surgical treatment for more resistant cases has a low complication rate and a high level of success.

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