MRI imaging of impingement syndromes of the ankle.

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Learning objectives

- - To review the anatomy and physiopathology of this entity.

- - To describe the clinical features, radiological findings, specially MRI, and the therapeutic management of the impingement syndromes of the ankle.

Background

Impingement syndromes on the ankle are entities characterized by pain and/or articular movement limitation, caused by bone or tissue friction in the joint. They were described for the first time in 1943 by Morris and later Mc Murray in 1950 named it as football player’s ankle. Nowadays they are a cause of ankle dysfunction frequently secondary to an apparently trivial traumatism. Ankle’s sprain (strain) are frequent though most of the cases don’t develop sequels, up to 15-20% have clinical persistence of the symptoms.

Post-traumatic synovitis, intra-articular fibrous and scar tissue or bone prominences are the main causes of impingement of the ankle.

The classification of these syndromes is anatomical based on their relationship with the talofibular joint; so they are classified in anterolateral, anterior, anteromedial, posteromedial, and posterior. In spite of that the damaging etiology can be similar in all of them, each location has a different symptom and clinical signs and specific findings in imaging studies.

Findings and procedure details

- **Anterolateral impingement (ALI)**

  a) **Anatomy:**

  The anterolateral channel is a triangular shaped space limited posteromedial by the tibia, lateral by the fibula, the anterior talofibular ligament (ATFL) and the ankle’s capsular, its anterior margin and caudal by the calcaneofibular ligament This space can have a small amount of fluid in normal conditions.

  b) **Pathogenesis:**
Soft tissue impingement in most occasions happen as a complication of a plantar flexion-inversion lesion that breaks the ATLL and the capsule. Secondarily to the presence of hemarthrosis and intra-articular fibrine, a post-traumatic can occur that impacts on the talar dome during dorsal flexion causing pain and movement restriction. With the passing of time, this synovitis can organize and develop fibrosis, that makes a meniscoid lesion inside the anterolateral recess.

Post-traumatic synovitis and meniscoid lesions can also be secondary to a damage in the syndesmotic element of the anteroinferior tibiofibular ligament (ATFL), because of the existence of an accessory element that can predispose to impingement or because of hyper-mobility of the ATFL with anterior displacement of the talus that can cause ALI, secondary to the ligament supporting the talar dome. Though more infrequent ganglion cysts can happen originating from the anterolateral recess of the articular capsule and can produce ALI symptoms.

The anterolateral bone impingement of the ankle is secondary to bone prominences that originate from the anterior margin of the tibial platform, lateral to the midline and inferior to the anterior insertion of the capsule. These spurs are a frequent finding in athletes and secondary to repeated impacts.

c) Clinical features:

Patients have pain with pronation supination with dorsal flexion of the foot.

d) Radiological findings:

In the anterolateral impingement of soft tissues, MRI can show a post-traumatic synovitis of the AL recess that we can see as an intermediate signal focus in protonic density weighted images (PD) with or without fat suppression (FS) in T2 weighted images. When this synovitis becomes fibrosis, it converges and progressively diminishes its signal intensity. The meniscoid viewed in MRI studies consists of a fibrous band with triangular shape of low signal intensity in T2 weighted axial images Fig. 2 on page 12.

We can also see capsular synovial ganglions that we can identify as rounded or multilobular lesions with similar signal intensity to fluid in MR studies, and if they are small they can be unobserved and we can mistake them with articular fluid or pericapsular vessels Fig. 3 on page 14.
In the bone impingement we can identify bone proliferations that originate from the anterior margin of the tibial platform. It is important to mention the size, its extension and the presence of complications as fractures and free ossicles in our report Fig. 4 on page 14, Fig. 5 on page 15 y Fig. 6 on page 15, that must be evaluated with a CT scan. We can also evaluate with MRI the chondral delamination of the tibial platform, the bone subchondral edema, and the superficial chondral (railway) lesion of the talar dome.

- **Anterior impingement (AI)**

The anterior impingement refers to the damage of the anterior and central aspects of the ankle.

**a) Pathogenesis:**

The AI is related to bone spurs in the tibial platform, typical of athletes that suffer a repeated forced dorsal flexion (dancers and football players) or repeated microtrauma. Less frequently they originate in the inferior recess of the talar neck Fig. 7 on page 16. In any of these cases, they develop as a response to trauma in the anterior articular cartilage with repeated dorsal flexion and impacts between the anterior tibiotalar portion or a direct lateral trauma in the anterior part of the ankle. The trabecular microfractures and periosteal hemorrhage that originates with the repeated trauma stimulates the formation of reactive bone with the development of bone proliferation of the anterior margin of the joint.

We consider that these spurs irritate the anterior capsule with synovitis that causes the symptomatology.

Though these bone prominences predispose to AI, they are frequently asymptomatic.

They can also happen after a sprain: the fibrin in the anterior recess of the ankle can fibrose and form bands that extent to the lateral and medial margins and impact in the talar dome during dorsal flexion.

**b) Clinical features:**

Patients with AI typically present with pain in the anterior aspect of the ankle with subjective sensation of blockade with dorsal flexion.

**c) Radiological findings:**
In plain x-rays and CT scan we can evaluate the bone spurs and tibiotalar articular space, the presence of ossicles and also the degenerative changes in the joint.

MRI is useful to show the edema of the bone spur when there is a fracture, the synovitis of the anterior recess, the thickening and capsular and pericapsular edema. Fig. 8 on page 17, Fig. 9 on page 18 y Fig. 10 on page 19. It is important to rule out if there are degenerative changes in the ankle joint, because those are an indicator of the success probability of the surgical treatment.

- **Anteromedial impingement (AMI)**

  a) **Anatomy:**

  The anteromedial recess is posteriorly limited by the medial malleolus, the anteromedial margin of the talar dome and the neck laterally, and the superficial anteromedial margin of the capsule.

  b) **Pathogenesis:**

  Soft tissues implication can cause AMI in the absence of bone spurs. The soft tissues AMI is caused by a lesion in the flexion inversion of the sole of the foot, with a component of impaction in the medial rotation. There is a bruising of the deltoid ligament and microtrabecular lesion of the medial malleolus and in the medial margin of the talar neck/body (kissing contusion) Fig. 11 on page 20. The anteromedial capsule and the ATTL can also originate AMI.

  Bone spurs are also one of the main causes of AMI, and they can originate from the dorsomedial margin of the talar neck, anteromedial in the tibial platform or from anterior margin of the medial malleolus. It is believed that the origin of these anterior spurs are repeated low energy traumas because of impaction. They can also be caused by the presence of post-traumatic ossicles secondary to the avulsion of the ATTL or anteromedial capsule, dystrophic ossification of ligamentous lesions or bone spurs fractures; this ossicle can injure the deep fibers of the deltoid ligament or the medial capsular margin.

  c) **Clinical features:**

  Patients have a chronic pain in the anteromedial margin of the ankle that increases with dorsal flexion.
d) Radiological findings:

The bone proliferations can be seen in CT and MRI studies; in the last case it can also evaluate the presence of bone edema in the spur and the synovitis or capsular thickening and pericapsular edema of the anteromedial recess Fig. 12 on page 22.

With MRI we can also evaluate the presence of edema and the thickening of the ATTL in the sagittal and coronal planes. The axial and sagittal images show the presence of synovitis and fibrous bands in the anteromedial recess. MRI is specially useful to rule out entities that can clinically mimic AMI (kissing contusion, osteochondral lesions or lesion of the anterior margin of the flexor retinaculum) Fig. 13 on page 22.

• Posteromedial Impingement (PMI)

a) Anatomy:

Posteromedial recess is anteriorly limited by the posterior edge of the medial malleolus and posteriorly by the PTTL the posteromedial margin of the superficial capsule. The posteromedial edge of the talar body/dome and the talar processus form the deep margin. In normal conditions, it is identified in axial images as a small recess that can include a small amount of fluid, deep to the space between flexor digitorum longus (FDL) and the flexor pollicis longus (FPL).

b) Pathogenesis:

The PMI is caused as a result of a plantar flexion inversion injure and intern rotation impaction with contusion of the posterior/deep fibers of the PTTL and the posteromedial margin of the capsule, secondary to the compression of the talus and medial malleolus Fig. 14 on page 23.

The edema and immature scar formation that affect the deep fibers of the deltoid ligament, can evolve to fibrosis and thickening of them that can protrude in the PM recess. There can also be an excessive post-traumatic synovitis with thickening and displacement of the posteromedial capsule that can overhang between the posterior and intern edge of the talar body/dome and the posterior margin of the medial malleolus. An avulsion fracture that affects the posteromedial talar process in the PTTL insertion with formation of fibrous and scarring tissue can predispose to PMI.

c) Clinical features:
Patients have pain between the posteromedial edge of the talar body/dome and the posterior margin of the medial malleolus. In physical examination, pain in the posteromedial region with inversion of the ankle in plantar flexion helps to differentiate PMI from posterior tibial dysfunction.

d) Radiological findings:

In MRI we can see in the subacute phase: signal intensity alteration of the deep fibers of the deltoid ligament secondary to edema with loss of their normal striate appearance and synovitis projection and scar tissue within the intern recess posteriorly with significant thickening of the posteromedial margin of the capsule and effacement of the normal space of the PM recess between FDL and FHL tendons Fig. 15 on page 24. There can also be kissing bone contusions that affect the medial malleolus and the medial margin of the talar body. The simultaneous injure of flexor retinaculum can cause partial scarring that surrounds the posterior tibial tendon PTT between the retinaculum and LTAP.

An avulsion fracture could be difficult to identify in MRI studies because of the signal intensity that can be similar to adjacent edematous scar tissue.

- **Posterior impingement (PI)**

Posterior impingement has been clasically described in ballet ballerinas that develop an echinus position. Other athletes as javelin throwers, football players, cricket players, gymnasts and artistic skaters, can also suffer this disease.

It is known with different names: trigonus syndrome, talar compression syndrome, and posterior blockade of the ankle.

a) **Anatomy:**

The anatomy of the posterior ankle is a key factor in the impingement syndrome. The secondary ossification nucleus is formed in the posterolateral aspect of the talus between 8 and 13 years of age and fusion with the rest of the talus in the first year after its formation. In 7% of the population there is a failure of this fusion, becoming the bone trigonum (os trigonum). Other anomalies that can predispose to PI is a posterolateral prominent talar process (Stieda process), prominence of the posterior tibial malleolus and a protuberant posterior calcaneus process.

The soft tissue lesion in the posterior aspect of the ankle can also cause PI: fibulotalar, intermalleolar and posteroinferior tibiofibular ligament, synovial sheath of the flexor pollicis and the posterior synovial recess of the tibiotalar and subtalar joints.
b) Pathogenesis:

The PI can be the consequence of an acute traumatic lesion in plantar hyper-flexion or it can be secondary to low grade repeated trauma associated to plantar hyper-flexion as it happens ballet dancers or football players. The posterior talus and the soft tissues that surround it compress between the tibial platform and the posteroinferior margin of the calcaneus during plantar flexion in which is known as the nutcracker phenomenon Fig. 16 on page 26.

The anatomical posterior talus bone variations can predispose individually to PI. This variations include the os trigonum, the posterolateral process of Stieda and a prominence of the superior calcaneus tuberosity Fig. 17 on page 26; it is important to outline that the size of the os trigonum and the prominence of the the posterolateral process are not related to the severity of the PI.

Instability of the chondral synchondrosis between the os trigonum and the talar body can happen as a consequence of repeated microtrauma or chronic inflammation, and is potentially a cause of pain.

Fractures of the talar posterolateral process (Sheperd fracture) or the absence of consolidation of them, can also be potential causes of PI symptoms Fig. 17 on page 26.

Bone proliferations depending on the tibial platform margin secondary to a previous injure of the syndesmosis, and posteroinferior exostosis that originate directly in the posterolateral talar process, can contribute also to posterior bone impingement of the ankle Fig. 17 on page 26.

Free bodies located in the ankle’s posterior recess or in the subtalar, and a synovitis affecting these points, can cause PI symptoms Fig. 17 on page 26.

There are entities that cause soft tissue injuries that can predispose individually to PI, as scarring of PTF, intermalleolar, posteroinferior tibiofibular ligaments and hypermobility of the lateral ligamentous complex of the ankle.

The PTF ligament can develop mucoid degeneration, with or without ganglion cyst formation. Ganglion cysts form typically adjacent to the fibular ligamentous insertion and decompress posteriorly. Both entities can also cause PI.
A distal displaced break of the CFL, with projection of the free ligament in the posterior recess or subtalar joint is too, though infrequently, a PI cause.

b.1) Pathogenesis of the flexor hallucis longus tenosynovitis.

In ballerinas the repeated overload of the FHL and the posterior tenosynovitis can be secondary to the "en pointe" position, foot pronation and external rotation of the hips the repeated irritation and retinaculum thickening, that forms the ceiling of the fibrous tunnel of the FHL, can cause a reduction of this tunnel and difficulty in the sliding of the tendon within its sheath. This entity is known as stenosing tenosynovitis and clinically presents as a FHL tendon dysfunction. Typically, is a stenosis that affects a small segment (5 mm). The low musculotendinous junction of the FHL and an accessory flexor muscle (intern calcaneofibular that goes inside the FHL sheath and inserts in the medial aspect of the calcaneus) can also cause PI or tendinitis of the FHL symptoms.

c) Clinical features:

PI typically causes pain and swelling in the posterolateral margin of the ankle, excruciating with plantar flexion. The differential diagnosis from the clinical point of view, includes retrocalcaneal bursitis or achilleal or fibular tendinous implication.

In the other hand, FHL tenosynovitis is characterized by pain and swelling of the posteromedial aspect of the ankle, pain in the first finger with active or passive movement, mobility limitation and sensibility under the course of the FHL. The DD includes deltoid ligament injury, posterior tibial synovitis, posteromedial tarsal coalition, talar osteochondral lesion, plantar fascitis and tarsal tunnel syndrome.

d) Radiological findings:

In plain X-rays we can identify the os trigonum or a Stieda process. It can also show cystic changes or sclerotic phenomena in the synchondrosis. The os trigonum should be differentiated from a fracture of the posterolateral talar process; CT scan can be useful to establish this difference and also to evaluate the changes of the joint facets of the synchondrosis.

With MRI studies we can evaluate the alteration of the signal intensity of the os trigonum: there can be bone edema in active PI, but sclerosis is less frequent. In the same way, with MRI we can study more correctly the synchondrosis implication, an increase of signal intensity in T2 or proton density-weighted (PD) sequences with fat suppression.
usually indicates a certain degree of overload of the joint and frequently is associated with marginal bone edema. The presence of fluid indicates synchondrosis irritability.

Other findings suggesting PI, though unspecific, are bone edema of the synchondrosis margin; ankle’s posterior recess synovitis and the posterior subtalar joint and pericapsular edema Fig. 18 on page 27. The presence of sclerosis and cysts in the synchondrosis margins indicate chronic stress of the joint.

Sagittal images are used to evaluate the posterolateral process of the talus. It is considered prominent if it goes posterior to the curvature arch of the talar dome in the sagittal plane. If there is a fracture of this process it is identified bone edema and a low intensity line in T1 images corresponding to the fracture.

Though the PTF ligament breaks infrequently, if there is one, mixoid changes develop with increase of the signal intensity in all the pulse sequences and thickening. The posterior intermalleolar ligament is identified in coronal and axial images; in the PI we can see its thickening and increase of the signal intensity with well delimited margins in PD or PD with fat suppression sequences, and adjacent synovial thickening as well.

The presence of fluid in the posterior recess is unspecific. The synovial thickening and the edema can be present in PI. An infrequent cause is the broken LPC displacement, with protrusion of the ligamentous end in the posterior recess of the subtalar joint.

The FHL tendon implication can mimic or associate with PI. MRI has a limited sensibility for the diagnosis of the tendinous FHL. There can be fluid in the in the tendinous sheath of asymptomatic patients and this findings, by itself, is not pathological. The distension of the tendinous sheath of the FHL in the absence of joint effusion in the ankle or subtalar joint, suggests tenosynovitis, but is still unspecific Fig. 19 on page 28. The MRI findings of tenosynovitis are tenosynovial thickening, retinaculum thickening for this tendon at the fibrous tunnel, tendinous thickening and signal intensity alteration within the tendon with edema in the adjacent fat.

The radiological report mainly must be focused to identify the cause that causes the symptoms of impingement and to define the bone or soft tissues disease to see if it´s possible a precutaneous treatment with steroids or debridement/surgical excision. It is important to try to define the presence of anatomical variants that predispose to impingement.
In the other hand, we should take into account the existence of associated complications (presence of osteochondral lesions above all) and to evaluate the rest of the joint because in some cases it will be decisive in the therapeutic success as we have seen.

**Therapeutic management.**

The initial treatment is conservative with immobilization, physiotherapy and antiinflammatory drugs. Surgery should be reserved for these cases resistant to other measures.

Percutaneous injection of steroids (with or without ultrasound guidance) allow us occasionally a return to normal activity of the patient, including elite athletes, however, there are no scientific papers that support this technique.

Arthroscopy with hypertrophic synovitis and fibrous and scar tissue resection and also the ligamentous fascicles or spurs when that is the underlying etiology. In AI many papers have demonstrated that open arthroscopic excision of the bone prominences or soft tissue is effective if there is no degenerative disease of the articular space previous to surgery.

**Images for this section:**
Fig. 1: Anatomy of the anteromedial recess of the ankle. The anterior recess (thick arrow) is located between the anterior tibiofibular ligament (arrowhead) and the anterior talofibular ligament (thin arrow). The curved arrow signals the calcaneofibular ligament.
Fig. 2: Fig. A and B Axial T2 sequence in a patient with a severe ligamentous lesion that had chronic instability of the ankle, ALI secondary to an important thickening and fibrosis of the TFAL (white arrow) that partially obliterates the anterolateral recess that had a small amount of fluid, deltoid ligament thickening (red arrow). Fig C and D: Axial T2 sequence. Inferior fascicle thickening of the TTFIL (yellow arrow) because of a syndemotic lesion.
with a underlying triangular shaped band (meniscoid lesion). Note the decreased signal intensity with the evolution to fibrosis.

**Fig. 3:** Axial T2 (a) and sagittal FST2 (B) sequences that show an anterior ganglion of the ATFL (white arrow) with a probable intraarticular origin (red arrow).
**Fig. 4:** Arthroscopic MRI correlation Fig A and B that show a spur depending form the lateral margin of the talus (asterisk) and an accessory component of the ATFL (arrow) Fig B Axial T2 sequence where we can see the bone prominence (white arrow). Fig C T2 fat suppression (FS) sequence in which we can identify the accessory ligament (yellow arrowhead).

**Fig. 5:** Arthroscopy of the previous patient. In Fig A and B we identify the accessory ligamentous component of the ATFL (red arrow) and the talar bone proliferation (asterisk). Figs C and D are images after the resection of both.
Fig. 6: Axial T2 (A) and sagittal T1(B) sequences in which we identify a lesion of the ATFL (red arrow) and the presence of an ossicle in the anterolateral recess (yellow arrow) probably secondary to a ligamentous avulsion. The asterisk signals a slight distension of the posterior recess and the white arrow a small amount of fluid in the FHL tendon sheath.
Fig. 7: Most frequent location of tibiotalar spurs (arrow) with the articular capsule (arrowhead).
**Fig. 8:** Arthroscopic/MRI correlation. Dorsal talar spur (asterisk) with associated synovitis. In FST2 sagittal sequences we can identify bone proliferation in the anterior tibial margin (kissing lesion).
Fig. 9: Arthroscopic/MRI correlation. Dorsal talar spur (asterisk) with associated synovitis. In FST2 sagittal sequences we can identify bone proliferation in the anterior tibial margin (kissing lesion).
Fig. 10: Arthroscopic image previous to the calcaneal spur resection (Fig A asterisk) and after excision (Fig B arrow).
**Fig. 11:** Anteromedial impingement of the ankle. Lesion with thickening and meniscoid lesion of the anterior tibiotalar ligament (arrowhead) and osteochondral lesion or osteophyte in the anteromedial margin of the joint surface of the talus (arrow).

![Anteromedial impingement of the ankle](image)

**Fig. 12:** AMI caused by a spur in the dorsomedial margin of the talar neck (arrow).
Fig. 13: Coronal FSDP sequence that show deltoid ligament lesion with thickening, increase of signal intensity and loss of the normal ligamentous striation (white arrow), Bone contusion (kissing bone contusion) in the medial malleolus and medial margin of the talus (red arrow).
Fig. 14: On the left: Representation of the posteromedial impingement location. On the right axial anatomy and structures involved in the posteromedial impingement. M (Medial malleolus); TP (posterior tibial tendon); FDL (flexor digitorum longus); PTTL; L (lateral malleolus), arrowheads point to the posterior fibers of the tibiotalar ligament and the thick arrow the deep fibers.
Fig. 15: Posteromedial impingement. Axial PD sequence that show a loss of the normal striation of the tibiotalar ligament (arrow) with inflammatory soft tissue thickening that displaces the posterior tibial tendon (asterisk).
Fig. 16: Nutcracker phenomenon in the posterior impingement of the ankle.
Fig. 17: Posterior bone impingement of the ankle A) Stieda process; B) Os trigonum; C) Lateral fracture of the talar tuberosity; D) Prominence of the tibial platform; E) Calcified inflammatory tissue; F) Prominence of the superior margin of the calcaneal tuberosity.
Fig. 18: MRI study of the previous patient. In sagittal images corresponding to T1 (A) and FST2 (B) we can see the os trigonum that show slight alteration of its signal intensity (asterisk) with degenerative phenomena and bone edema of the subtalar joint (red arrows).
**Fig. 19:** Axial T2 sequence of the same patient. In A we can see os trigonum and in B we can identify slight amount of fluid within the flexor hallucis longus tendon sheath (yellow arrow).
Conclusion

Impingement syndromes of the ankle are an infrequent cause of dysfunction and chronic articular pain. Though in most of the cases the diagnosis is clinical, imaging studies play an important role in those dubious cases and above all in the exclusion of concomitant pathology.

It is important to know and take into account this entity in the evaluation of the painful and unstable ankle and to understand its physio-pathology, a main column to look for and recognize their radiological findings, occasionally very subtle.

The radiological report must be oriented to the evaluation and classification of those findings (soft tissue and bone impingement) and to the detection of complications or associated lesions with the therapeutic management in mind.

Personal information

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