Magnetic resonance imaging of ankle impingement syndrome: iconographic essay*

Síndrome do impacto do tornozelo na ressonância magnética: ensaio iconográfico

Claudio Marcio Amaral de Oliveira Lima[1], Erica Barreiros Ribeiro[2], Elisa Pompeu Dias Coutinho[3], Evandro Miguelote Vianna[4], Rômulo Côrtes Domingues[4], Antonio Carlos Coutinho Júnior[3]

Abstract **Ankle impingement syndrome is a painful condition resulting from friction of joint tissues that is both cause and effect of an altered joint biomechanics. The leading causes of such condition are post-traumatic lesions, particularly the ligamentous ones, resulting in chronic ankle pain. From an anatomic and clinical point of view, these syndromes may be classified as anterolateral, anterior, anteromedial, posteromedial, and posterior. Magnetic resonance imaging is an excellent diagnostic method for demonstrating bone and soft tissue abnormalities resulting from different types of ankle impingement, providing useful data to confirm the diagnosis as well as to rule out other possible causes of joint pain. The present essay is aimed at illustrating the main magnetic resonance imaging findings in ankle impingement syndrome.**

Keywords: Magnetic resonance imaging; Syndrome; Impingement; Ankle.

Resumo **A síndrome do impacto do tornozelo é uma condição dolorosa causada por atrito de tecidos articulares, que é tanto causa quanto consequência de uma biomecânica alterada desta articulação. A sua principal causa são as lesões pós-traumáticas, principalmente lesões ligamentares, resultando em dor crônica no tornozelo. Do ponto de vista anatômico e clínico, estas síndromes são classificadas em: ântero-lateral, anterior, ântero-medial, póstero-medial e posterior. A ressonância magnética é um ótimo método diagnóstico para demonstrar as alterações ósseas e as partes moles dos vários tipos de impacto do tornozelo, fornecendo dados que auxiliam não só na comprovação desse diagnóstico, como na diferenciação com outras causas de dor articular. Os autores objetivam ilustrar os principais achados de ressonância magnética na síndrome do impacto do tornozelo.**

Unitermos: Imagem por ressonância magnética; Síndrome; Impacto; Tornozelo.

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INTRODUCTION

Ankle impingement syndrome is a painful condition resulting from friction of joint tissues that is both cause and effect of an altered joint biomechanics. Main causes include post-traumatic lesions, particularly ligament lesions, resulting in chronic ankle pain. From the clinical and anatomical point of view, such syndrome is classified into anterolateral, anterior, anteromedial, posteromedial and posterior[1–3].

The present study is aimed at illustrating, as an iconographic essay, the main magnetic resonance imaging (MRI) findings of ankle impingement syndrome.

MRI PROTOCOLS

The patients are previously instructed to not utilize any type of substance (ointments, gel or cream) on the ankle, and before being submitted to MRI, they are interviewed by the clinical team on the presence, intensity and localization of the pain, type of trauma and respective dynamics (inversion, eversion, hyperextension or flexion, if either direct or indirect, etc.) previous history of surgery, endocrinopathy or rheumatologic diseases, either in association or not, and type of physical activity, if any.

The sequences utilized in the protocol for study of the ankle, both with a 1.5 T unit (Magnetom Avanto; Siemens Medical Systems, Erlangen, Germany) and with a 3 T unit (Magnetom Trio; Siemens Medical Systems; Erlangen, Germany) are respectively: sagittal, T1-weighted (Avanto – FOV: 170; slice thickness: 4/25; matrix: 512 × 282; NEX: 1; acquisition time: 1.55 min; TR: 400; TE: 20. Trio – FOV: 160; thickness: 3/0.6; matrix: 384 × 307; NEX: 2; acquisition time: 1.16 min; TR: 641; TE: 11) and axial (Avanto – FOV: 160; thickness: 3/30; matrix: 384 × 214; NEX: 1; acquisition time: 1.21 min; TR: 552; TE: 24. Trio – FOV: 160; thickness: 3/0.3; matrix: 384 × 307; NEX: 2; acquisition time: 1.45

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1. MD, Radiologist, Centro de Diagnóstico por Imagem Fátima Digittal, Nova Iguaçu, RJ, Brazil.

2. MD, Radiologist, Unit of Magnetic Resonance Imaging – Hospital São José, Criciúma, SC, Brazil.

3. MDs, Radiologists, Clínicas de Diagnóstico Por Imagem (CDPI)/Multi-Imagem, Rio de Janeiro, RJ, and Centro de Diagnóstico por Imagem Fátima Digittal, Nova Iguaçu, RJ, Brazil.

4. MDs, Radiologists, Clínicas de Diagnóstico Por Imagem (CDPI)/Multi-Imagem, Rio de Janeiro, RJ, Brazil.

Mailing address: Dr. Antonio Carlos Coutinho Júnior. CDPI – Clínica de Diagnóstico Por Imagem. Avenida das Américas, 4666, sala 325, Barra da Tijuca. Rio de Janeiro, RJ, Brazil, 22649-900. E-mail: cmaol@br.inter.net / cmaolima@gmail.com

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min; TR: 500; TE: 11), STIR sequences or proton density with fat suppression at sagittal plane (Avanto – FOV: 170; thickness: 4/25; matrix: 320 × 208; NEX: 1; acquisition time: 2.04 min; TR: 3000; TE: 33. Trio – FOV: 160; thickness: 3/0.6; matrix: 320 × 256; NEX: 2; acquisition time: 1.53 min; TR: 2500; TE: 44), sequences at proton density, coronal plane (Avanto – FOV: 170; thickness: 3/30; matrix: 320 × 146; NEX: 1; acquisition time: 2.29 min; TR: 4810; TE: 33. Trio – FOV: 160; thickness: 3/0.6; matrix: 320 × 256; NEX: 2; acquisition time: 1.45 min; TR: 2690; TE: 42) and axial plane (Avanto – FOV: 160; thickness: 3/30; matrix: 320 × 179; NEX: 1; acquisition time: 1.36 min; TR: 2600; TE: 25. Trio – FOV: 160; thickness: 3/0.3; matrix: 320 × 256; NEX: 2; acquisition time: 2.01 min; TR: 3100; TE: 42).

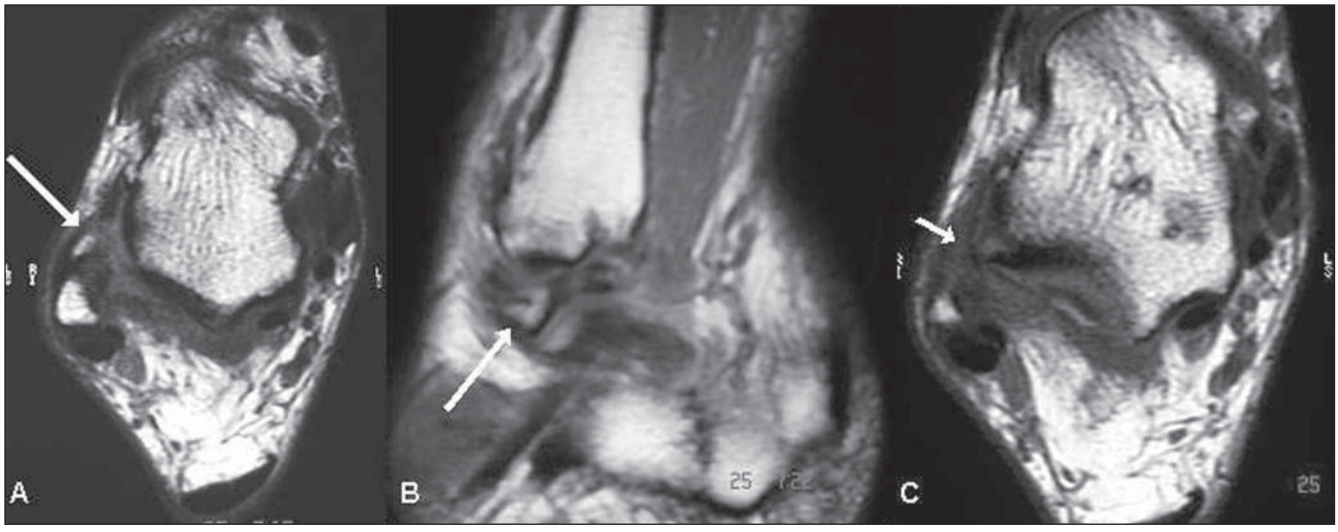

Figure 1. Axial (**A,C**), and sagittal (**B**) T1-weighted images of a patient presenting repetition torsion, complaining of pain in the anterolateral region of the ankle, demonstrating poorly individualized anterior talofibular ligament with an small avulsioned bone fragment at the fibular insertion (long arrows). Additionally, isointense tissue thickening is observed in the region of the anterolateral joint recess (short arrow).

CLASSIFICATION

Anterolateral ankle impingement

Anterolateral ankle impingement is a common cause of chronic lateral pain in the ankle produced by hypertrophic synovial tissue entrapment in the anterolateral gutter of the ankle. It is estimated that approximately 3% of ankle sprains lead to anterolateral impingement[1–4].

The etiology is related to small repeated inversion traumas, initially with no significant mechanical instability. Microtraumas continuity results in inflammation, synovial hypertrophy and fibrosis, causing pain and mechanical instability. In the most advanced forms, an organized meniscal lesion develops with hypertrophic synovial tissue and fibrosis[1,4], as described by Wolin et al.[5].

The anterior talofibular ligament and the tibiofibular ligament were implied as causes for such impingement[4]. The presence of anterior or anterolateral bone impingement caused by osteophytes may exacerbate this condition. The accessory fascicle of the anterior tibiofibular ligament (Bassett's ligament)[6], that is a common anatomic variation, may present hypertrophy after repeated trauma, resulting in anterolateral impingement, particularly if other supporting structures of the region are involved[1,4].

At MRI, the presence of soft tissue mass or fibrotic band in the anterolateral ankle gutter, different from the anterior talofibular ligament, suggests the diagnosis[1,4] (Figures 1 and 2). Conventional MRI accu-

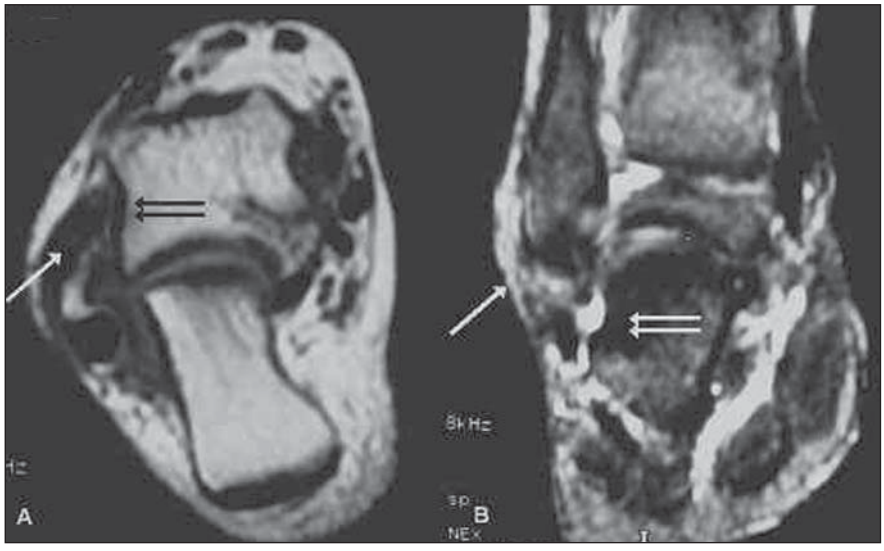

Figure 2. T1-weighted axial (**A**) and coronal, proton density with fat suppression (**B**) demonstrating isointense tissue in the anterolateral gutter (arrows) and minimal corresponding sclerosis on the lateral talar surface (double arrows) characterizing the impingement.

racy still remains controversial, and many authors advocate the utilization of MRI only in the presence of a significant joint effusion. Magnetic resonance arthrography represents an excellent tool for diagnostic elucidation before arthroscopy, and a relevant finding is the absence of fluid in the recess between the soft tissues of the anterolateral region and the anterior fibular surface, because of the presence of adhesions and cicatricial tissue between the fibula and the joint capsule, preventing the fluid to enter the recess[1,4].

Anterior ankle impingement

Anterior ankle impingement is well established and relatively common cause of chronic ankle pain, and may affect any athlete who is subjected to stress resulting from repeated dorsiflexion, especially soccer players and ballet dancers[1,3].

It is widely recognized that dorsiflexion or supination injuries can cause damages to the anterior and medial edges of the joint cartilage, and it is believed that repair of such lesion with cicatricial tissue and proliferative fibrosis leads to the development of osteophytes and joint capsule avulsion. Also, another theory suggests that forced dorsiflexion results in repeated microtraumas on the tibia and talus, with consequential microfractures of the trabecular bone/periosteal hemorrhage, stimulating the formation of a new bone tissue. Anterior tibial and talar osteophytes impinge on each other, particularly at dorsiflexion, determining an impingement on the interposed soft tissues[1,3] (Figure 3).

Remarkably in soccer players, at the moment of the kick, the Ball causes a direct trauma on the anterior tibiotalar joint, since at this moment the joint cartilage is covered only by the subcutaneous fat[1].

A study has demonstrated that a significant percentage of professional athletes (45–59%) presented osteophytes at conventional radiography, with no symptom of previous impingement, which leads to the conclusion that the association of cicatrization and synovial thickening is more significant than simply the presence of osteophytes to produce the clinical syndrome[1,3].

Conventional radiography demonstrates osteophytes and the conventional MRI allows the evaluation of soft tissues, visualization of tibiotalar osteophytes positioning in relation to the capsule, the synovitis in the anterior capsular recess, and the presence, although rare, of bone marrow edema. Synovial thickening usually presents hypointense signal on T1-weighted sequences, and low to intermediate signal intensity on T2-weighted sequences, more clearly outlined in the presence of joint effusion[1,3].

Anteromedial ankle impingement

Anteromedial ankle impingement is a clinical entity increasingly recognized in the orthopedic literature as an uncommon cause of chronic ankle pain[1].

The exact mechanism of the anteromedial ankle impingement is still to be understood, however it is believed that it may be a result, initially, of a forced supination with a rotational component, leading to an injury of the anteromedial joint capsule. The repeated anterolateral impingement and subsequent microtrauma produce synovitis and joint capsule thickening. Additionally, bone and joint cartilage lesion may result in osteophytes formation, besides joint and synovial capsule thickening on the anteromedial surface. The result is a meniscal lesion represented by thickening of soft tissues anterior to the tibiotalar ligaments that may appear isolatedly or be secondary to a partial injury of the deltoid ligament[1,3]. Another cause of impingement is thickening of the anterior tibiotalar ligament. Such thickening or the meniscal lesion determine impingement on the region corresponding to the anteromedial corner during ankle dorsiflexion, resulting in development of osteophytes, chondral lesion or both, commonly associated with an inversion mechanism with injury of lateral or medial ligament[1,3].

Magnetic resonance imaging demonstrates the meniscal mass, chondral or osteochondral lesions, ligament thickening, besides osteophytes[1,3] (Figure 4), and conventional radiography demonstrates osteophytes, but, considering the uncommon nature of this condition, no extensive study reporting such imaging findings is available in the literature[4].

Posteromedial ankle impingement

Posteromedial ankle impingement is a common cause of pain after a severe ankle inversion injury in which the deep posterior fibers of the deltoid ligament remain compressed between the medial wall of the talus and the medial malleolus[1,3]. Initially, posteromedial symptoms are not noticeable as compared with the symptoms of lateral ligament rupture, and spontaneous resolution is achieved without a specific treatment. However, the inappropriate healing of the posterior deep fibers of the deltoid

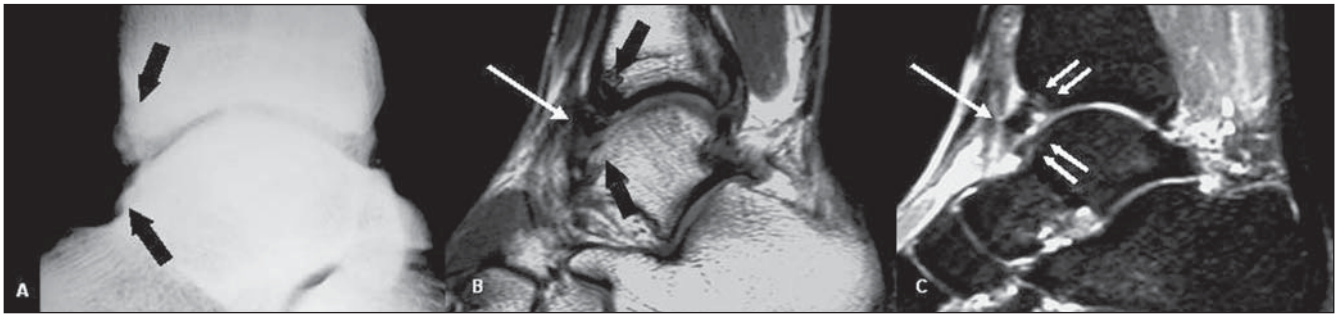

Figure 3. Lateral ankle radiography (**A**), sagittal T1-weighted (**B**), and sagittal proton density with fat suppression (**C**) images demonstrating anterior tibial and talar osteophytes (black arrows) with subtle bone marrow edema (double white arrows), besides anterior capsular thickening (long white arrow) characterizing anterior ankle impingement.

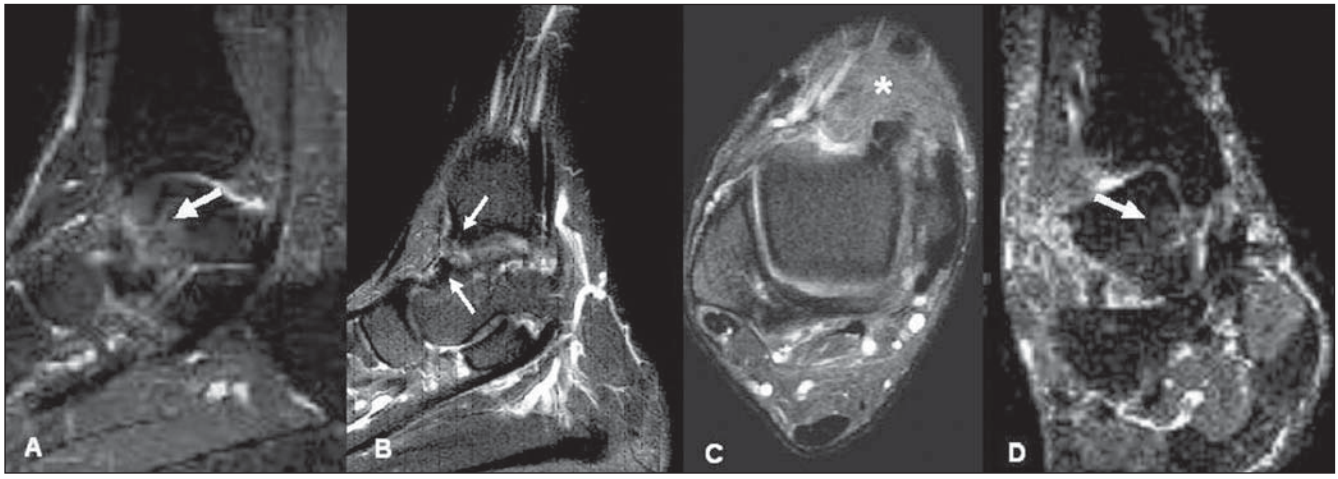

Figure 4. Sagittal (**A,B**), axial (**C**), and coronal (**D**) fat-suppressed proton density-weighted images demonstrate tibial and talar osteophytes (thin arrows), isointense (meniscal) tissue thickening in the anteromedial region of the ankle (asterisk) and also a minimal irregularity on the medial talar surface, with subtle corresponding bone marrow edema (thick arrows), characterizing anteromedial ankle impingement.

ligament leads to chronic inflammation, hypertrophic fibrosis and metaplasia.

In many cases, such disorganized fibrotic tissue may lead to impingement between the medial wall of the talus and the posterior rim of the medial malleolus[1,3].

Magnetic resonance imaging demonstrates the soft tissues thickening and the bone marrow edema of the medial talus and malleolus[1,3] (Figure 5).

Posterior ankle impingement

Posterior ankle impingement corresponds to a set of abnormalities resulting from forced repeated or acute plantar flexion. This syndrome is also called *os trigonum* syndrome, talar compression syndrome or posterior block of the ankle[1,3,7], and has been extensively described in professional soccer players as well as in classical ballet dancers[1,3]. Such type of impingement develops as a result of significant acute injury, such as avulsion of the posterior talofibular ligament, talar fracture or accessory ossicles injury, but, in most of cases the syndrome onset is insidious in predisposed athletes. It is believed that repeated forced foot flexion results in chronic bone and soft tissue lesions. The injury mechanism is similar to a "nutcracker effect" where the posterior portion of the talus and adjacent soft tissues are compressed between the tibia and the calcaneus during the feet plantar flexion[1,3,7].

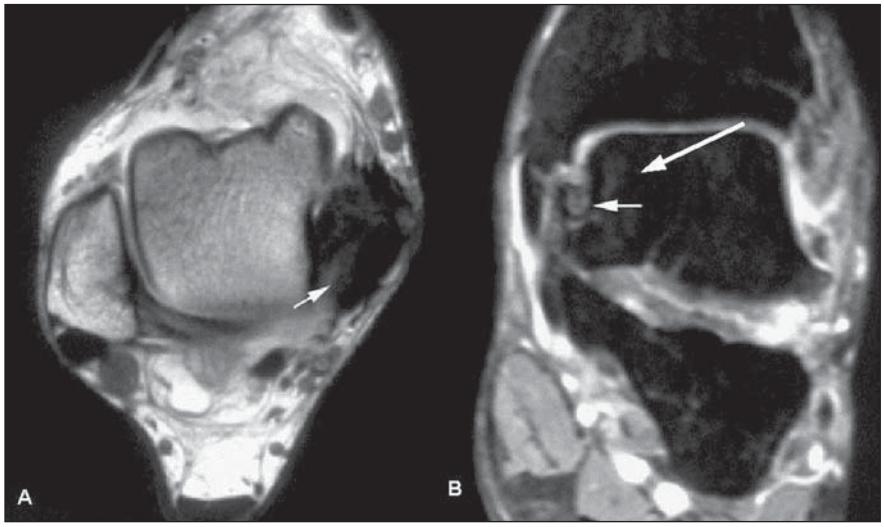

Figure 5. Axial T1-weighted (**A**) and coronal, fat-suppressed proton-density weighted (**B**) images demonstrating isointense tissue in the posteromedial region of the ankle (short arrows) and irregularity on the posteromedial talar surface, with subtle corresponding bone marrow edema (long arrow), characterizing posteromedial ankle impingement.

The anatomy of the posterior region of the ankle is a key factor in the development of this type of impingement whose causes are divided into osseous and soft tissue causes. The most frequent cause is osseous in nature and is represented by the *os trigonum* that is an accessory ossicle of the posterior talar tubercle that may remain unfused in up to 7% of the general population, or by the Stiedas's process that is a prominent lateral talar tubercle. Among the causes originating in soft tissues, synovitis involving the flexor hallucis longus tendon sheath, the posterior intermalleolar ligament and the posterior synovial recess of the tibiotalar and subtalar joints[1,3,7].

Such type of impingement may present as soft tissues inflammation in the posterior ankle, accessory ossicles injury or both. Bone lesions include fracture, fragmentation and pseudoarthrosis of accessory ossicles or lateral talar tubercle. Soft tissue lesions include posterior and subtalar synovitis, as well as tenosynovitis of the flexor hallucis longus[1].

The hallucis longus flexor runs deeply in the gutter between the medial and lateral talar tubercles, and stenosing tenosynovitis may develop as it is involved in the posterior ankle impingement[1].

MRI demonstrates findings such as *os trigonum*, prominent posterior talar process

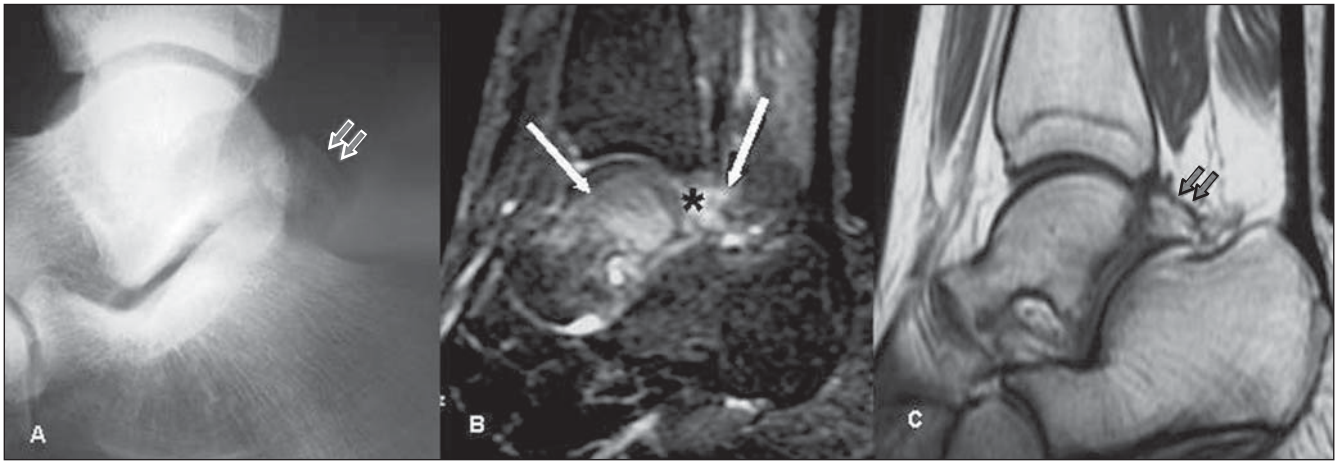

Figure 6. Lateral ankle radiography (**A**), sagittal STIR MRI sequence (**B**), and T1-weighted (**C**) images demonstrating the presence of *os trigonum* (double arrows) with bone marrow edema. Additionally, bone marrow and soft tissues edema is observed in the posterior portion of the talus (long arrows), between the posterior rim of the tibial epiphysis (double arrows) and the *os trigonum* (asterisk).

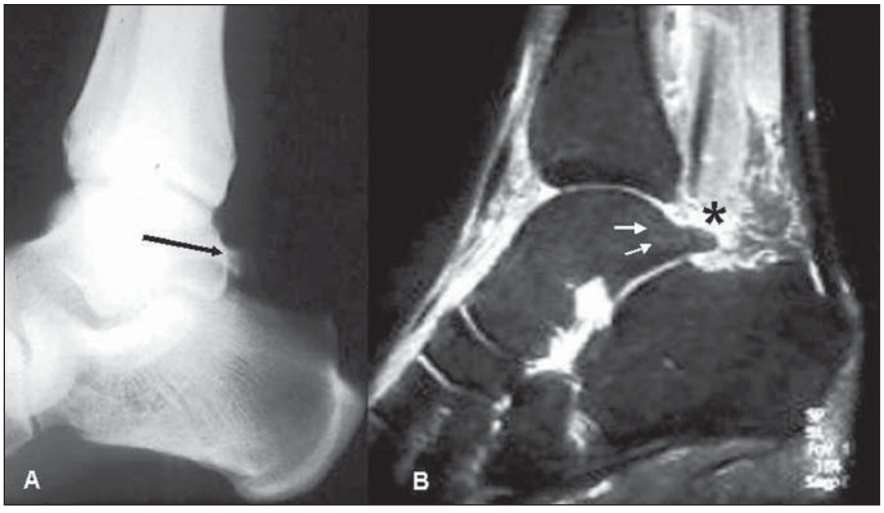

Figure 7. Lateral ankle radiography (**A**) and sagittal MRI fat-suppressed proton-density-weighted (**B**) images demonstrate prominent posterior talar process, Stieda's process (long arrow) with subtle bone marrow edema (short arrows). Additionally, soft-tissue edema is observed between the posterior rim of the tibial epiphysis and the posterior talar process (asterisk).

(Stieda) in association with bone marrow edema, as well as edema or synovitis in the posterior joint recess and adjacent soft tissues[1,3,7] (Figures 6 and 7).

Finally, MRI is an excellent method for demonstrating bone and soft tissues abnormalities in the different types of ankle impingement syndrome, providing useful information not only for confirming the diagnosis but also to differentiate from other causes of joint pain.

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