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Stieda process versus Haglund's deformity: An uncommon presentation of posterior ankle impingement

Case Report

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Abstract: Posterior ankle impingement syndrome represents an important cause of chronic ankle pain and has been extensively described in the orthopaedic and radiology literature. A rare case of this painful hindfoot disorder that resulted from two concurrent developmental anomalies is presented herein.

Keywords: *MR imaging • Posterior ankle impingement • Stieda process • Haglund's deformity*

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1. Introduction

Ankle impingement syndromes can be an important cause of chronic ankle pain, which can negatively influence the performance of professional athletes [1]. Biomechanically they are caused by friction between joint tissues and they are characterized by limitation of full range of ankle motion due to osseous and soft tissue abnormalities [2]. Although impingement syndromes were initially studied in elite athletes, they are nowadays considered a cause of persisting ankle pain in the general population [3]. They are classified from an anatomic and clinical point of view into anterolateral, anterior, anteromedial, posteromedial, and posterior impingement syndromes [4]. We report a case of a 27-year-old football player with a rare posterior impingement syndrome and describe the MR imaging findings.

2. Case Report

A 27-year-old male football player presented at the orthopaedic department with a two month's history of progressive posterior ankle pain. Physical examination revealed mild swelling and tenderness on palpation along the posterolateral ankle, between the Achilles and peroneal tendons, as well as severe restriction in the range of motion during plantar or dorsal flexion. The plain X-ray (performed elsewhere, not shown) showed a hypertrophic and prominent posterolateral talar recess opposite a bony overgrowth of the posterosuperior calcaneal surface area.

MR imaging was performed to further clarify the clinical and X-ray findings. T1-w images in the sagittal plane depicted the hypertrophic and prominent talar recess opposite the bony overgrowth of the calcaneus (Figure 1). Proton density with fat suppression (Figure 2) and T2 gradient echo images (Figure 3) confirmed the presence of marrow edema in the opposing bony protuberances. A small effusion of the tibiotalar joint (Figure 2) and soft tissue edematous and fibrotic changes in the periarticular fat (Figure 4a to c) were also depicted. The patient's history of sports activity (football), the clinical features, physical examination and imaging findings were indicative for the diagnosis of posterior ankle impingement syndrome due to a Stieda talar process impinging upon a Haglund's deformity of the calcaneus.

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Figure 1. Sagittal T1-w MR image, showing the marrow edema in opposing bony protuberances (arrows). Note Haglund's deformity in calcaneus (open arrow).

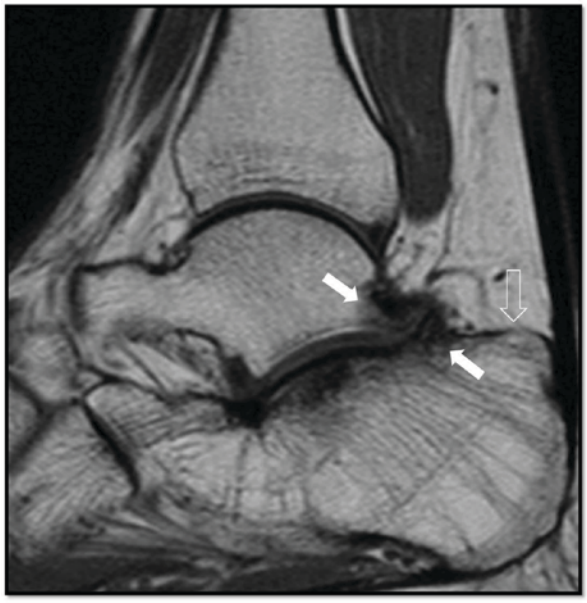
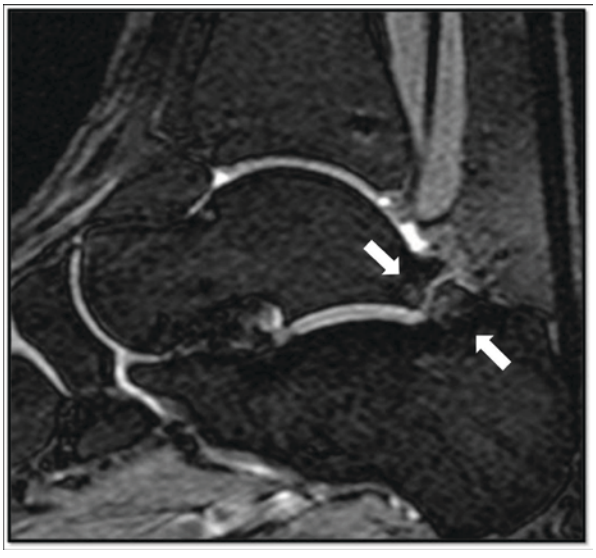


Figure 2. Sagittal fat suppressed proton density MR image, showing the bone marrow edema in Stieda process and in Haglund's deformity (area circled in yellow).

Figure 3. Sagittal gradient echo (MEDIC) MR image, showing the impinging bony structures as well as the bone marrow edema (arrows).

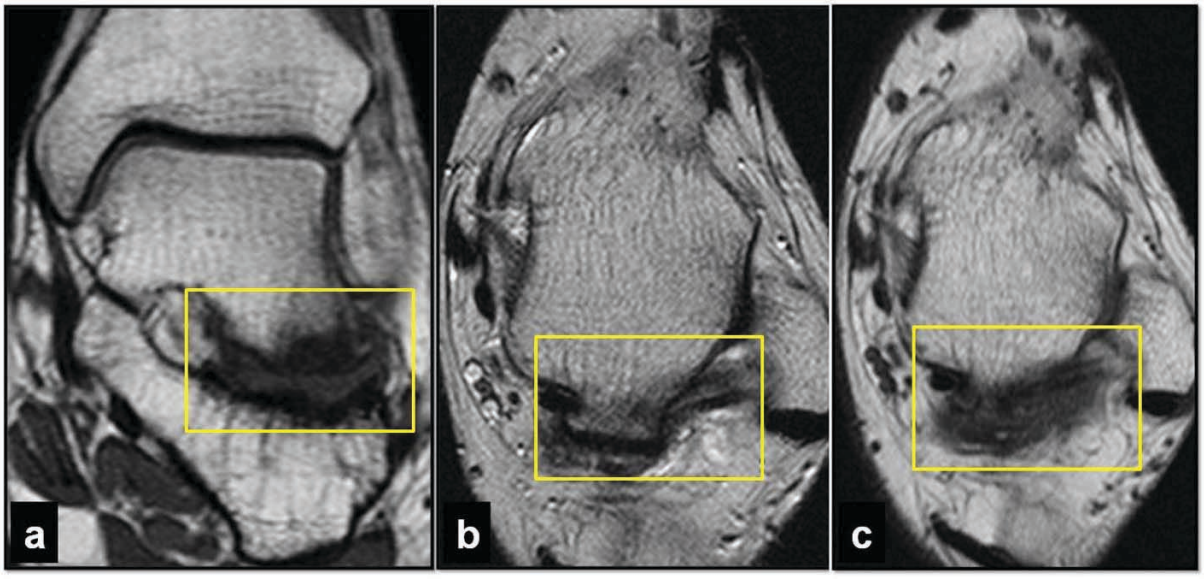


3. Discussion

Posterior ankle impingement syndromes include a variety of abnormalities that lead to compression of the soft tissues between the talus, the calcaneus and the posterior tibia on plantar flexion of the ankle [1-6]. It has been described in literature with different names such as os trigonum syndrome, talar compression syndrome, and posterior block of the ankle [1-6]. In our case the predisposing factors for impingement was the coexisting presence of a Stieda talar process and a Haglund's deformity and to the best of our knowledge, it represents an extremely rare report of such a painful hindfoot syndrome.

During skeletal development, a secondary ossification center forms at the posterolateral aspect of the talus between the ages of 7 and 13 years and normally fuses within 1 year [3-5]. In the case of bony overgrowth, a large lateral talar process is formed, which is called a Stieda process. If there is failure of fusion, an os trigonum is formed, which articulates with the talus through a synchondrosis [3-6]. Haglund's deformity is caused by bony overgrowth of the posterior calcaneus and is considered a developmental anatomic variant [7-9]. This retrocalcaneal exostosis has been implicated in a variety of painful entities, most commonly Achilles tendinosis and retrocalcaneal bursitis [7-9]. In our case, however, the concomitant presence of opposing calcaneal and talar developmental variants resulted in impingement syndrome. The soft tissues that may be injured in posterior ankle impingement syndrome include the posterior articular capsule, the posterior talofibular, intermalleolar, and tibiofibular ligaments and the flexor hallucis longus tendon [6-9]. Biomechanically, repetitive forced plantar flexion of the foot results in chronic injury to the aforementioned posterior elements. Ballet dancers, athletes in jumping sports and professional football players, like in our case, are at increased risk

Figure 4. Coronal T1-w (a), axial T2-w (b) and axial T1-w (c) MR images, showing the Stieda process of the talus along with marrow edema and surrounding edematous and fibrotic changes in the surrounding fat (areas in rectangles).



of developing this syndrome due to prolonged and continuous plantar flexion [7-9]. The patient's history and physical examination can suggest the correct diagnosis. Conventional radiographs and CT may show anatomic variants such as the lateral talar (Stieda) process, os trigonum or Haglund's deformity, but further imaging is required to confirm if these findings are the source of symptoms. MR imaging and MR arthrography are the best imaging modalities for identifying soft-tissue abnormalities and joint pathology. Although MRI is not the method of choice to diagnose a bone deformity, it is considered the imaging modality of choice to diagnose impingement syndromes [4-6]. The initial treatment of posterior ankle impingement syndrome is conservative by means of physiotherapy. If this should fail, arthroscopic or open debridement results in symptomatic relief and in improvement of ankle function along with restoration of range of motion [7-10].

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