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Calcific enthesopathy of the superior extensor retinaculum – an unusual cause of medial ankle pain

Ali Shah[1], Karthikeyan P. Iyengar[2], Ganesh Hegde[1], James Ramos[3], Rajesh Botchu[1]

[1] *Department of Radiology, The Royal Orthopaedic Hospital, Birmingham, United Kingdom*
[2] *Department of Orthopaedics, Southport and Ormskirk Hospital NHS Trust, Southport, United Kingdom*
[3] *Department of Orthopaedics, University Hospitals of Birmingham, Birmingham, United Kingdom*

Corresponding author: Rajesh Botchu, Radiology, The Royal Orthopaedic Hospital, Department of Musculoskeletal Radiology, B31 2AP, Birmingham, United Kingdom; e-mail: drbrajesh@yahoo.com

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enthesopathy; ankle pain; superior extensor retinaculum; ankle ligament calcification; barbotage

Abstract

Aim of the study: Ankle pain can present a clinical dilemma to the foot and ankle surgeons, with a multitude of entities to which the symptoms could potentially be attributed. Enthesopathy around the ankle joint could be due to overuse, injury, inflammation or infection. Calcific ligamentous enthesopathy around the ankle is a well-recognised condition with a spectrum of causes. **Case description:** To our knowledge, a clinically symptomatic presentation of calcific enthesopathy specifically affecting the entheses of the superior extensor retinaculum has not been described in the literature. We report the first case of symptomatic calcific enthesopathy of the superior extensor retinaculum in a healthy young female, and highlight the role of radiological interventions in its diagnosis. The condition was managed successfully by ultrasound-guided barbotage. **Conclusions:** Calcific enthesopathy of the attachment of the superior extensor retinaculum is a rare condition that should be considered in the differential diagnosis of patients with medial ankle pain.

Introduction

The region around the ankle joint is an intricate area stabilised by static osseous and dynamic structures. It consists of ligament complexes, traversing tendons, and important neurovascular structures. Consequently, pathologies arising from all these structures can pose a diagnostic dilemma for a clinician in patients presenting with ankle pain. A thorough understanding of the anatomy and pathological entities pertaining to the area is key to reaching a definitive diagnosis and, consequently, ensuring appropriate patient management. The movement of the ankle is dependent upon the contraction of the flexor, extensor, and peroneal groups of muscles[(1)].

The retinacula of the ankle are regions of localised thickening of the investing deep fascia. They hold the coursing tendons of the leg and foot at the ankle to ensure efficient functioning and prevent bowstringing. These consist of the extensor retinaculum (superior and inferior), the peroneal retinaculum, and the flexor retinaculum[(2)].

The extensor retinaculum is derived from the superficial crural aponeurosis of the leg and can be divided into the transverse rectangular superior and the more complex X- or Y-shaped inferior extensor retinaculum[(1,3,4)].

The superior (proximal) extensor retinaculum (SER) attaches at the anterior tibial crest and the medial malleolus medially and the anterior border of the fibula and the lateral malleolus laterally. This is approximately 3 cm proximal to the tibiotalar joint[(5)]. Its deep relations include the following extensor tendons (from medial to lateral): tibialis anterior, extensor hallucis longus, extensor digitorum longus, the dorsalis pedis vessels, the deep peroneal nerve, and the peroneus tertius. The tibialis anterior tendon may

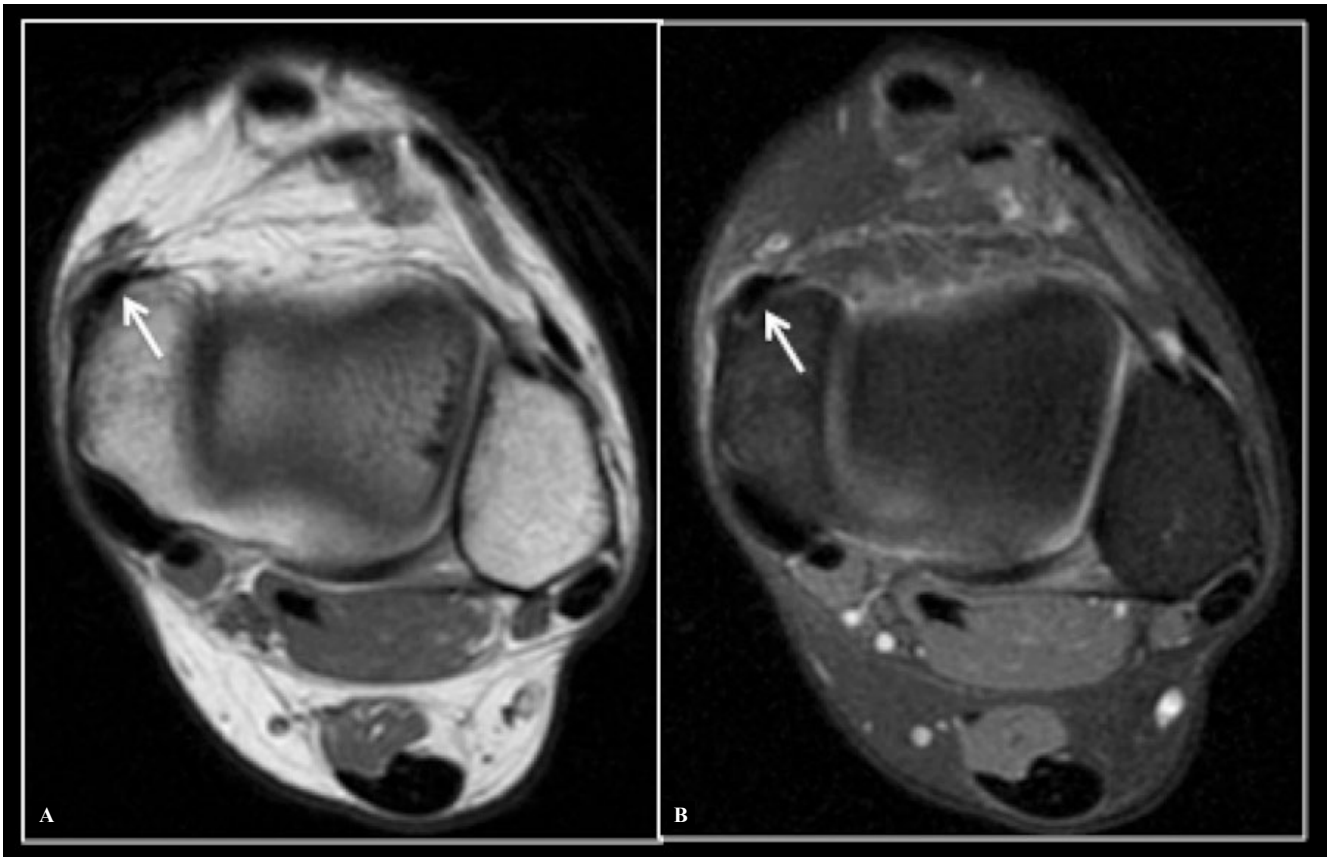

Fig. 1. *Axial PD (***A***) and PDFS (***B***) showing calcification with mild oedema in relation to the tibial attachment of the superior extensor retinaculum*

run in a separate tunnel formed from the superficial and deep fibres in 25–29% of cases[4,1].

Ankle pathologies presenting as ankle pain involve a spectrum of acute and/or chronic conditions. Ankle injuries involving soft tissue ligament and/or osseous elements are well described in the literature.

Symptomatic presentations of ligament calcifications have been reported at the lateral collateral[6] and medial collateral ligaments of the knee[7], the ulnar collateral ligament of the elbow[8] and the spine[9,10].

To date, calcifications of the SER itself have not been described in the literature. We present a rare case of calcification of the SER in a 51-year-old female that was managed successfully by ultrasound-guided (USS) barbotage.

Case report

A 51-year-old , fit and otherwise healthy, with no co-morbidities, presented with an insidious-onset ankle pain localised to the anterior and medial aspects of the left ankle joint, persisting for over a year.

This was associated with the development of a firm swelling over the medial aspect of the ankle. The swelling itself did not prevent the patient from walking or activities of daily living. However, it was painful on pressure, and she was concerned enough to seek medical advice. There was no history of specific injury or trauma that preceded the onset of symptoms.

On clinical examination, there was no erythema or redness around the left ankle joint. There was moderate focal tenderness at the anteromedial aspect of the tibia, corresponding with the attachment of the SER. The ankle revealed a full range of motion, with no features of instability. The patient was able to stand on tiptoes and perform a single heel rise test.

Radiographs of the left ankle revealed no osseous abnormality. She underwent a magnetic resonance imaging (MRI) scan of the ankle, which demonstrated a 10 mm hypointense homogenous lesion on T1-weighted sequences, representing calcification at the attachment of the SER at the medial malleolus (Fig. 1) and the medial attachment of the SER. There was low signal on fluid-sensitive sequences, with mild perilesional oedema. No osseous oedema of the medial malleolus was noted. The tibiotalar joint and other joints were unremarkable. The tibialis posterior, the flexor digitorum longus, the flexor hallucis longus, and the peroneal and anterior tendon complexes were intact. The medial and lateral ligament complexes, the plantar fascia and the Achilles tendon, as well as the sinus tarsi, were normal.

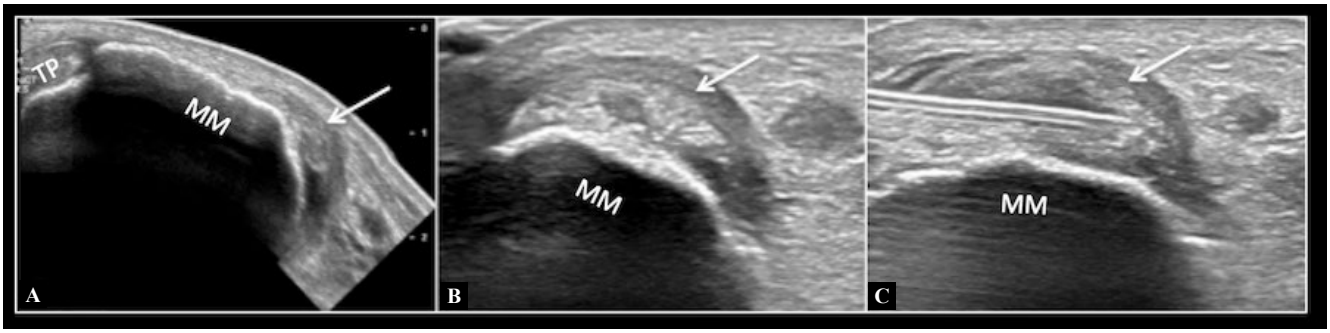

Fig. 2. *Panoramic ultrasound of the medial malleolus (MM) (***A***) and axial ultrasound of the lesion (***B***) showing calcification in relation to the superior extensor retinaculum treated with barbotage (***C***). TP (tibialis posterior)*

USS evaluation demonstrated a hemispherical mixed echoic lesion with central foci of calcification at the site of the medial attachment of the superior extensor retinaculum. The central hyperechoic foci represented calcification with peripheral hypoechoic focus corresponding to thickened SER. This corresponded to the abnormal lesion noted on the MRI sequences. A diagnosis of SER calcific enthesopathy was made and the patient was treated with USS-guided barbotage with a 16G white needle (Fig. 2) after obtaining informed consent. A significant improvement in symptoms was shown at the six-month clinical follow-up appointment.

Discussion

The pathogenesis of calcification of ligaments and tendons is often unclear. Rotator cuff calcific tendinopathy is a very common condition and hence calcific tendinopathy is thought to be a cell-mediated disease in which tenocytes transform into chondrocytes and induce calcification within the tendons[5]. However, it can also be argued that it is a degenerative process involving tendon fibres which undergo necrotic changes progressing to calcification. On the other hand, this hypothesis can be challenged by the fact that some calcification can resolve spontaneously with restoration of the gross tendon morphology[5].

Nevertheless, it is well understood that calcification occurring due to the deposition of calcium hydroxyapatite crystals at the attachment sites of tendons and ligaments can cause pain and disability, especially if unrecognised.

Calcific tendinitis is rare in the foot and ankle. Only a few case reports have been published in the literature to address medial ankle and foot pain from calcific tendinitis of the posterior tibialis tendon at the navicular insertion[11].

Symptomatic ligament calcification is even more uncommon in this anatomical location. Our literature search through the PubMed database revealed no reported cases of calcification/mineralisation of the SER. Thickening and scarring of the ankle retinacula have been described in asymptomatic football players, with the probable mechanism involving repeated sub-maximal stress on normal tissue inducing local tissue inflammation and scar tissue formation in the long term[1,12,13].

Ding *et al*.[14] reported a retrospective review of seven cases of traumatic avulsion of the SER with subperiosteal haematoma as a hypoechoic lenticular structure responsible for the elevation of the hyperechoic periosteum at the fibular insertion of the SER. The SER itself was thickened and hypoechoic, without mineralisation. Our patient was a healthy middle-aged female and even though she had a known pathology, it was present at an unusual site. We hypothesise that that calcification of the SER could be idiopathic or post-traumatic due missed ankle sprain.

Our case is unique in that not only it is the first case of calcification affecting the medial attachment of the SER retinaculum, but also USS-guided barbotage allowed successful management of the patient's symptoms.

This case report highlights the need for a high index of suspicion about unusual causes of medial ankle pain and confirms the versatility of USS as a therapeutic modality in patient management.

Conclusion

Calcific enthesopathy of the medial attachment of the superior extensor retinaculum is a rare condition and should be considered in the differential diagnosis of patients presenting with medial ankle pain.

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Conflict of interest

The authors do not report any financial or personal connections with other persons or organizations which might negatively affect the contents of this publication and/or claim authorship rights to this publication.

Informed consent

The procedure was undertaken following appropriate informed consent process.

Author contributions

Original concept of study: AS, KI, JR, RB. Writing of manuscript: AS, KI, GH, RB. Analysis and interpretation of data: KI, RB. Final acceptation of manuscript: AS, RB. Collection, recording and/or compilation of data: AS, JR, RB. Critical review of manuscript: AS, KI, GH, RB.

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