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Painful Os Peroneum Syndrome Secondary to Hydroxyapatite Deposition Disease

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Abstract

Painful os peroneum syndrome (POPS) is one of the uncommon causes of lateral foot pain in orthopaedic practice. POPS encompasses a spectrum of pathologies such as an acute or a chronic fracture, peroneus longus tenosynovitis, and attrition or partial rupture of the peroneus longus tendon. Herein, we reported the first case of POPS secondary to hydroxyapatite deposition disease in a 32-year-old female patient. The clinical presentation, the role of radiological interventions, particularly ultrasound-guided barbotage in its diagnosis and effective management, is highlighted.

Keywords
- foot pain
- calcification
- hydroxyapatites
- radiology
- ultrasound
- barbotage

Introduction

Painful os peroneum syndrome (POPS) is a condition characterized by lateral foot pain associated with the os peroneum, a sesamoid bone embedded in the peroneus longus tendon. It can result from acute or repetitive trauma, tendon pathology, or biomechanical abnormalities. Hydroxyapatite deposition disease (HADD), involving the deposition of calcium hydroxyapatite crystals in periarticular soft tissues, is another condition that can lead to significant pain and inflammation. The coexistence of POPS and HADD is extremely rare and, to our knowledge, has not been previously reported in the literature. This case report described a unique presentation of POPS secondary to HADD, highlighting the diagnostic challenges and innovative treatment strategies employed. The use of ultrasound in detecting calcific deposits, which were not identified by X-ray or magnetic resonance imaging (MRI), played a pivotal role in the accurate diagnosis and successful management of this condition. This case report aims to contribute to the understanding of POPS and HADD, emphasizing the importance of comprehensive imaging and minimally invasive treatment options. By sharing this case, we hope to enhance the diagnostic and therapeutic approaches for similar presentations in clinical practice.

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Case Report

A 32-year-old woman came to the orthopaedic clinic with a 3-month history of painful left lateral foot. She described the pain as severe and sharp, aggravated by movement, and significantly impacting her daily activities, although it did not disturb her sleep. She had no history of trauma, diabetes, or previous foot surgeries. On physical examination, mild tenderness was noted in the lateral foot without cuboid tenderness or limitation of movement.

Initial radiographs revealed the os peroneum and subtle calcification anterior to the os peroneum (►**Fig. 1**). MRI was performed to evaluate this further, which showed mild edema in the os peroneum and surrounding soft tissue but no calcification (►**Fig. 2**). Ultrasound was subsequently performed, which revealed normal appearance of the peroneal tendons and os peroneum, with minimal hyperemia and a small calcific deposit distal to the os peroneum, consistent with HADD (►**Fig. 3**). All blood investigations, in particular serum uric acid and serum calcium, were normal.

A diagnosis of POPS secondary to HADD was made, which has not been previously reported in the literature. The patient underwent an ultrasound-guided barbotage using a lateral approach. Dry needling of the calcific deposit was performed, followed by an injection of 20-mg triamcinolone acetonide and 0.2% ropivacaine around the os peroneum (►**Video 1**). No immediate complications occurred, and the patient experienced significant symptomatic relief.

Video 1

Video showing dry needling of the calcification. Online content including video sequences viewable at: https://www.thieme-connect.com/products/ejournals/html/10.1055/s-0044-1789190.

Discussion

It is well understood that calcifications, which result from calcium hydroxyapatite crystals being deposited at the insertion sites of tendons and ligaments, can be painful and debilitating, particularly if left undiagnosed.[1] Ankle retinacula thickening and scarring have been observed in asymptomatic football players; the likely mechanism involves recurrent submaximal stress on normal tissue that causes long-term local tissue inflammation and scar tissue formation.[2–4] A PubMed database literature search revealed no reported cases of POPS from HADD. Although HADD frequently affects the supraspinatus tendon in the rotator cuff, a wide range of other periarticular soft tissues may also be involved. Tendons, tendon sheaths, bursae, ligaments, periarticular connective soft tissues, and infrequently the joints and retinacula[1] can all have calcific deposits. The supraspinatus tendon of the rotator cuff is most frequently affected. Other less frequently involved tendons include the rectus femoris tendon.[5] Intramuscular involvement of HADD has also been reported.[6]

According to Uhthoff and Loehr, HADD can manifest in tendons in three different phases: precalcific, calcific, and postcalcific.[7] The calcific stage is subdivided into the formative, resting, and resorptive stages.[7] Fibrocartilaginous metaplasia, which is most likely caused by a drop in oxygen tension, causes calcification during the precalcific phase, which gives rise to the formative stage. Cellular response surrounding the calcific deposits and enhanced vascularity during the resorptive phase may cause the deposits to expand and become less well defined. This stage of the disease is the most painful and symptomatic[8] since it may also cause the calcific deposits to migrate to nearby tissues. Formation of granulation tissue and tendon remodeling are noted in the postcalcific phase. Depending on the percentage of calcium in the deposits, Bianchi and Becciolini[8] identified three different types of calcifications that are visible in the disease: type 1 (rich in calcium) corresponds to the formative and resting phases and appears as hyperechoic foci with

Fig. 1 (A) Dorsoplantar and (B) lateral radiographs of the foot demonstrating the os peroneum (*blue arrow*) and subtle calcification (*yellow arrow*). cu, cuboid.

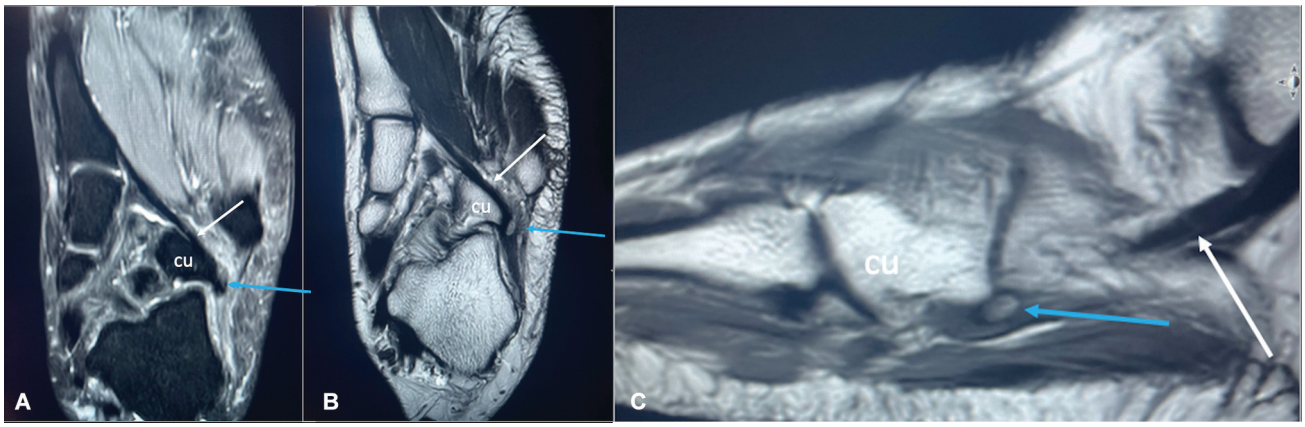

Fig. 2 Axial (**A**) PDFS and (**B**) PD, and (**c**) sagittal PD showing the peroneus longus tendon (*white arrow*) and os peroneum (*blue arrow*) with edema. cu, cuboid; PD, proton density; PDFS, proton density fat saturated.

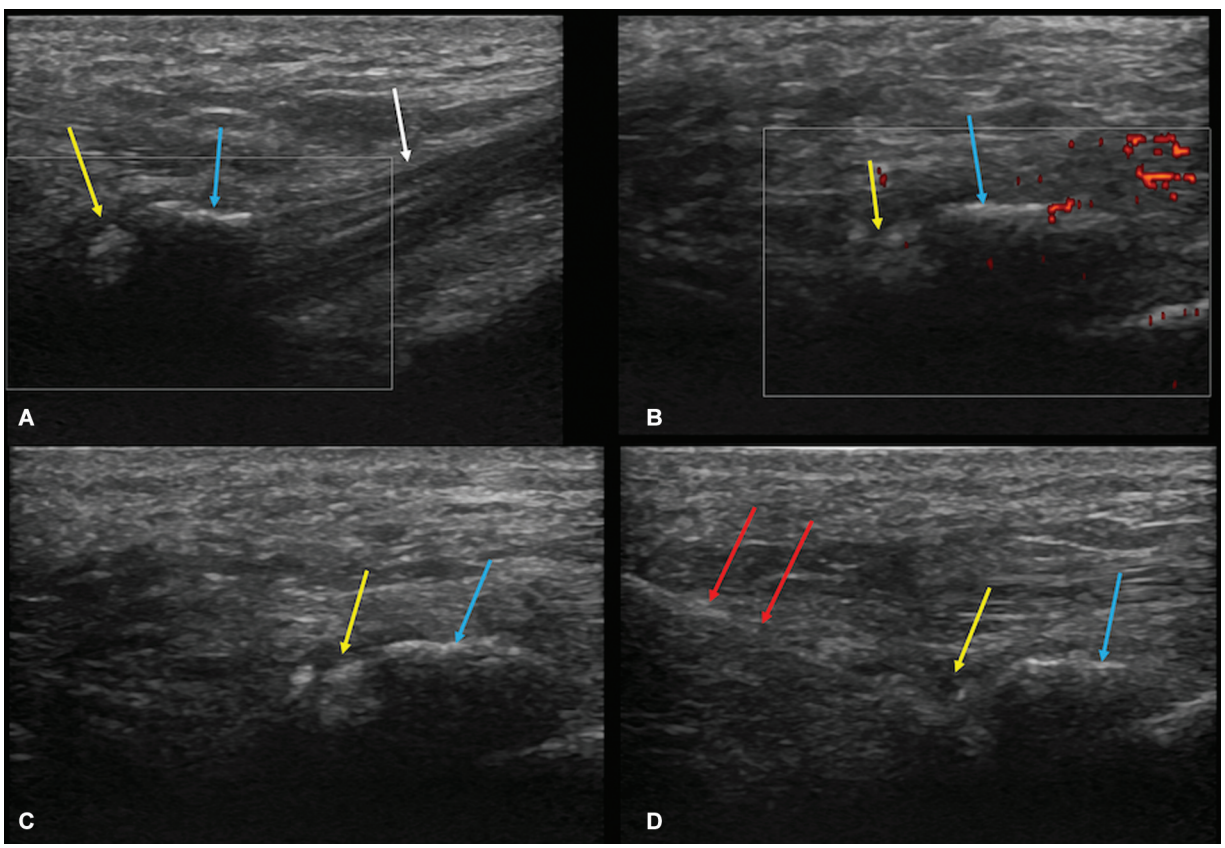

Fig. 3 (**A**) Longitudinal and (**Bb, C**) short axis (power Doppler) ultrasound images showing peroneus longus tendon (*white arrow*), os peroneum (*blue arrow*), and calcification anterior to the os peroneum (*yellow arrow*). (**D**) Ultrasound-guided barbotage and dry needling showing needle (*red arrow*) with the tip in calcification.

acoustic shadowing on ultrasound; type 2 (hyperechoic) calcifications only show faint acoustic shadowing; and type 3 (isoechoic to tendons) calcification appears ill defined and lacks acoustic shadowing. Patients with acute symptoms are more likely to have type 2 and 3 calcifications, as they typically correspond to the resorptive phase. Although calcific tendinopathy is the pathogenesis described by Uhthoff and Loehr[7] and the calcification types described by Bianchi and Becciolini,[8] similar parallels can be drawn to patients with calcific periarthritis as both the conditions result from deposition of hydroxyapatite crystals.

Ultrasound-guided barbotage is an established method for treating painful calcific tendinopathy and rotator cuff periarthritis. It has been demonstrated that this method works well for treating rotator cuff calcific tendinopathy.[9] We employed the same technique in our patient. Barbotage involves the needling and lavage technique. A needle is used under ultrasound guidance to fragment the hydroxyapatite crystals in the calcific deposits

and then flushed with normal saline.[10] The aspirated mixture of fragmented calcium hydroxyapatite and the saline appear as a milky white mixture. To ensure that the fractured calcifications can be adequately aspirated, a rather large gauge needle (usually at least an 18-gauge needle) is employed.[11] Both single- and double-needle methods can be used for the process; the latter uses separate needles for aspiration and fragmentation/saline flush. Since corticosteroids have anti-inflammatory qualities that can aid in analgesia and enhance function, they are frequently injected into the affected area after the treatment.[11,12]

In addition to the advantages of directly lavaging the deposits, excellent results in calcific tendinopathy have been reported following partial removal of calcific deposits as there is sufficient disruption and localized bleeding to allow for spontaneous resorption of the remaining calcium or its dispersion. Better results have been shown when there is a demonstrable decrease in size and echogenicity of the calcium postprocedure,[13] as was the case with our patient where the calcifications exhibit faint acoustic shadowing. To the best of our knowledge, there are no reports in the literature about POPS from HADD and usage of ultrasonography (USG) barbotage in the treatment of this entity. Barbotage has been successfully used to treat HADD in rare sites other than the rotator cuff, such as the spring ligament.[14] Given the shared pathophysiology with carpal tunnel, a similar approach can be used with successful outcomes as demonstrated in this case. Although USG-guided barbotage is generally a safe technique, one should be aware of the possible risks involved. The difficulties could stem from corticosteroid injection that often accompanies the procedure, or they could be due to barbotage itself.

Barbotage-related complications include bleeding, infection, tendon rupture, injury to the neurovascular structures, and brief inflammatory aggravation.[15] These hazards can be mitigated by carrying out the procedure in an aseptic environment, ensuring sufficient needle visualization under ultrasound guidance, and taking safety measures to avoid important structures.

Conclusion

Overall, this case report highlighted the need for a high index of suspicion about unusual causes of lateral foot pain and confirms the versatility of USG as a therapeutic modality in patient management.

Patient Consent

Informed consent of the patient was obtained for inclusion in the study.

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None.

Conflict of Interest

None declared.

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