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Calcific Enthesitis of Lateral Patellofemoral Ligament: A Rare Cause of Anterolateral Knee Pain

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Abstract

Hydroxyapatite deposition disease is a common musculoskeletal pathology that often affects rotator cuff tendons in the shoulder joint. Here, we present the first documented case of acute calcific enthesitis involving the lateral patellar retinaculum, specifically the lateral patellofemoral ligament (LPFL). A 35-year-old male presented with insidious onset atraumatic pain persisting for 3 months on the lateral aspect of the knee, intensifying over the past week. Clinical examination revealed focal tenderness in the anterolateral region of the knee, with no signs of patellar instability. Magnetic resonance imaging was performed, which revealed thickening and calcification within the LPFL and perilesional edema, further confirmed on computed tomography and ultrasonography. Patient was managed successfully with barbotage with complete resolution of symptoms. This case highlights the importance of considering calcification of LPFL in the differential diagnosis of anterolateral knee pain and underscores the importance of different imaging modalities for prompt diagnosis.

Keywords
- periarthritis
- HADD
- knee
- USG
- patellar retinaculum
- MRI
- calcific enthesitis

Introduction

Hydroxyapatite deposition disease (HADD) is characterized by the deposition of calcium hydroxyapatite crystals within and around connective tissues, usually in a periarticular location.[1] Crystals may deposit in tendons, ligaments, joint capsules, bursae, or soft tissues. Although HADD typically involves the shoulder joint, it is also described in other areas like the wrist, hand, foot, elbow, hip, knee, and spine.[1,2] However, calcific enthesitis affecting ligaments in the anterolateral knee are rare. Some documented cases include calcification in the lateral collateral ligament and the iliotibial tract in the lateral aspect of the knee.[3] Notably, there have been no reports describing calcification of the lateral patellofemoral ligament (LPFL) or the lateral patellar retinaculum itself and this is the first reported case.

Relevant Anatomy

The lateral patellar retinaculum of the knee, comprising a network of fascial layers and ligaments, plays a pivotal role in stabilizing the knee joint's extensor mechanism, particularly the patella. Various authors have described different iterations of the lateral retinaculum and its anatomy over time. The superficial lateral retinacular layer is formed by the merging of fibers originating from the iliotibial band, quadriceps aponeurosis, and the tendon of the vastus lateralis

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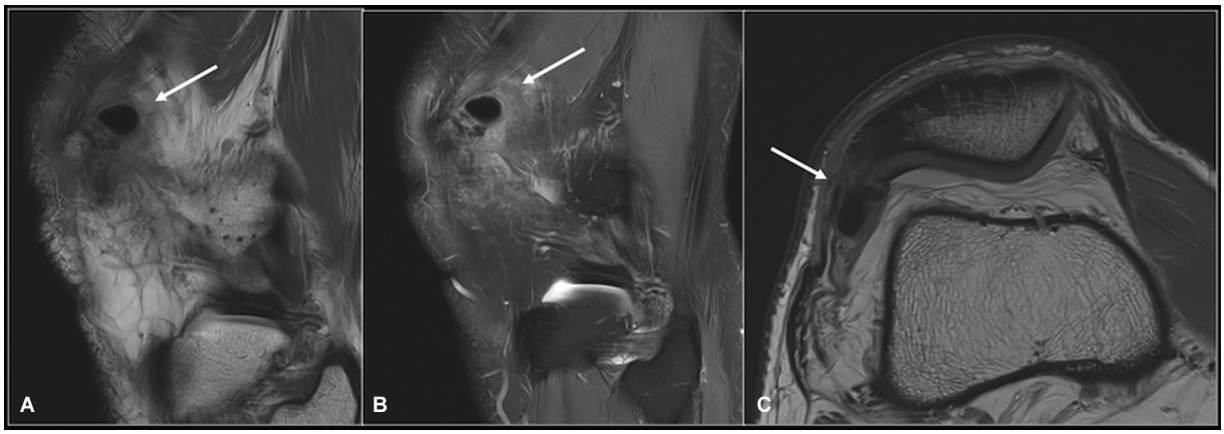

Fig. 1 Sagittal proton density (PD) (**A**), proton density fat-saturated (PDFS) (**B**), and PD axial (**C**) showing calcification within the lateral patellofemoral ligament (LPFL) (arrow).

obliquus. Deeper capsular structures, such as the LPFL and the lateral patellomeniscal ligament, connect the patella to the lateral femoral epicondyle and the lateral meniscus, respectively, bolstering overall knee stability. The LPFL attaches to the proximal half of the lateral patella and typically inserts just anterior and distal to the lateral femoral epicondyle.[4,5]

Case Report

A 35-year-old male presented with a history of insidious onset pain over the lateral aspect of the knee persisting for 3 months, particularly aggravated by walking. The pain intensified over the past week, with recent localization to the anterolateral region. No history of injury or trauma preceded the symptoms. Clinical examination revealed focal tenderness at the anterolateral aspect of the knee, corresponding to the lateral patellofemoral retinaculum, without signs of patellar instability. The knee exhibited a full range of motion, and there were no soft tissue swelling or erythema. The patient had no significant medical history or comorbidities. Magnetic resonance imaging (MRI) was performed to evaluate this further, which revealed thickening of the intact LPFL and a 12-mm homogeneous low-signal lesion within the ligament on all sequences, with perilesional edema observed on fluid-sensitive sequences (►**Fig. 1**). No osseous edema of the patella or femoral condyle was noted. Subsequent computed tomography (CT) confirmed soft tissue calcifications in the LPFL (►**Fig. 2**). Ultrasonography (USG) revealed distinct calcifications with posterior acoustic shadowing and irregularly bordered calcifications with faint posterior acoustic shadowing, along with mild edema in adjacent soft tissues. Color Doppler study revealed neovascularization surrounding the calcific foci within the ligament (►**Fig. 3**). The appearance of calcification on MRI, USG, and CT resembled that seen in acute calcific enthesitis, leading to a diagnosis of HADD. Patient's condition was successfully managed symptomatically with barbotage (►**Fig. 4**, ►**Videos 1** and **2**). Barbotage was performed using a 16-gauge needle aspirating the calcification using multiple passages into the focus of calcification following which 40 mg of Depo-Medrone was injected. He had complete resolution of symptoms at 6 weeks' follow-up.

Video 1

Video showing barbotage of calcification of lateral patellofemoral ligament. Online content including video sequences viewable at: https://www.thieme-connect.com/products/ejournals/html/10.1055/s-0044-1793807.

Video 2

Video showing syringe post barbotage with calcifications. Online content including video sequences viewable at: https://www.thieme-connect.com/products/ejournals/html/10.1055/s-0044-1793807.

Discussion

Acute calcific periarthritis is a self-limiting, monoarticular, periarticular process of dystrophic mineral deposition and adjacent inflammation. It is commonly seen in middle age with female preponderance.[1,2] Patients typically experience atraumatic pain initially localized to a single joint, although it may involve multiple joints over time. Clinical manifestations include acute pain accompanied by local tenderness and swelling at the affected joint, along with some limitation in movement and occasionally mild fever.[3,6,7]

Soft tissue musculoskeletal calcifications are not uncommonly seen on radiographs. In the case of HADD, these deposits appear as distinct periarticular densities, localized to the symptomatic site, and lacking a definable cortex. However, they may go unnoticed due to bony superimposition or misinterpretation as accessory ossicles or avulsion fractures.[8,9]

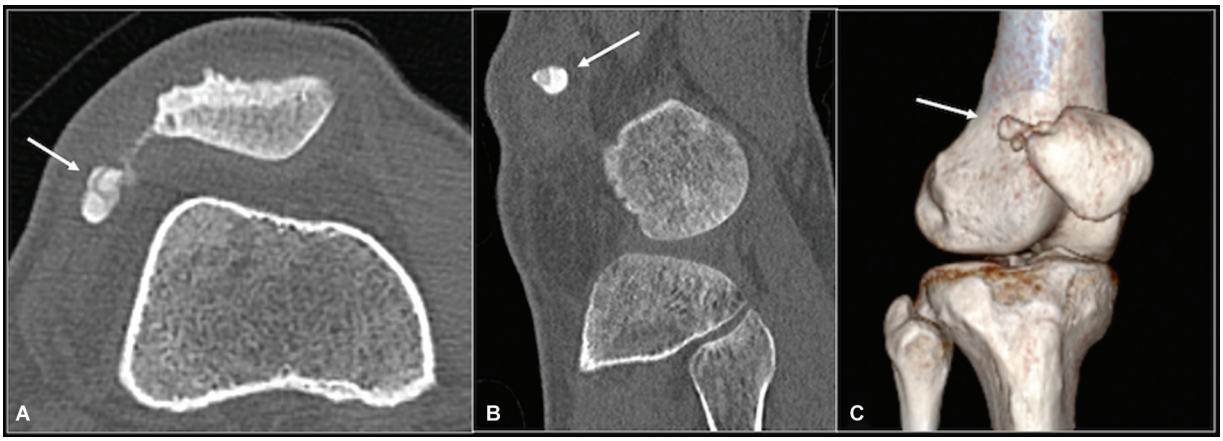

Fig. 2 Computed tomography (CT) axial (**A**), coronal (**B**) bone windows, and reformats (**C**) showing calcification within the lateral patellofemoral ligament (LPFL) (arrow).

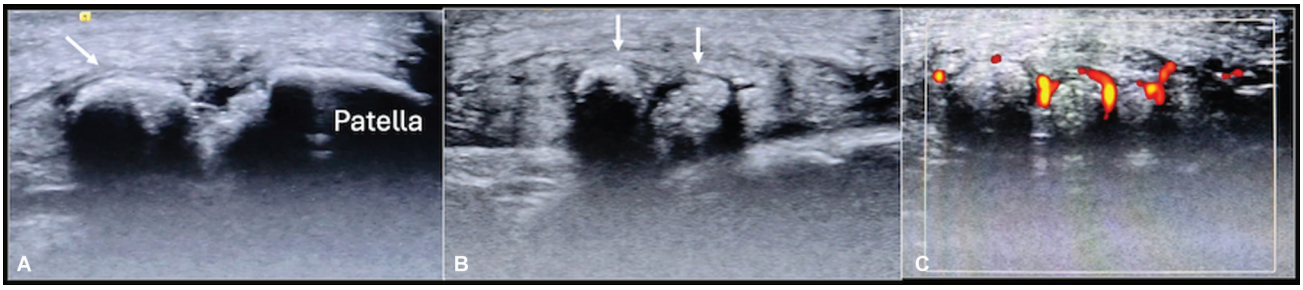

Fig. 3 Short axis (**A, B**) ultrasound of knee shows multiple foci of calcification (arrow) in relation to the lateral patellofemoral ligament (LPFL) with mild increased signal on Doppler (**C**).

Fig. 4 Image of syringe showing calcification postbarbotage.

The progression of HADD is a temporal phenomenon, and the imaging appearance of the calcific deposits is contingent upon the calcification stage.[2,7,8,10] In the initial silent or formative phase, calcium hydroxyapatite is entirely contained within the tendons, ligaments, or synovium with minimal patient symptoms. The calcium hydroxyapatite appears as thin and poorly defined deposits in early phases, gradually becoming denser and homogenous, resulting in well-defined round-to-ovoid soft tissue calcific deposits on conventional radiographs, well-defined hyperechoic structures with posterior acoustic shadowing.[3,10] In the mechanical phase, patients experience acute painful episodes due to the increasing size of liquefying calcific deposits and due to secondary rupture of liquefied calcific deposits into and around the adjacent bursa. In this phase, the calcific deposits will appear ill-defined with a comet tail-like appearance and may mimic a periosteal reaction. On USG, they appear dense, hyperechoic with indistinct borders and faint acoustic shadow. The final phase, adhesive periarthritis, represents an advanced stage characterized by overall weakness, pain, and restricted range of motion. Patients in the adhesive periarthritis phase exhibit adhesive bursitis, calcific apatite deposits within the tendon, ligament, and intraosseous, and dumbbell-like calcium deposits with secondary bony erosions.

USG has been shown to be an excellent imaging modality for the evaluation of calcific periarthritis. USG can be used for both diagnosis and treatment of HADD.[7,10] It can also illustrate adjacent inflammation and is less time-consuming and

effective for accessible joints. It can show calcifications inside most of the appendicular tendons, ligaments, and bursae. Fluffy, ill-defined, inhomogeneous calcifications are associated with the acute symptomatic phase, while well-defined, homogeneous calcifications tend to present in patients with chronic pain or no symptoms. Increased flow on color Doppler showed a strong association with the acute symptomatic phase.[9,10] In our case, both types of calcifications have been noted on USG, with increased signal on Doppler and perilesional edema suggesting acute phase.

On MRI, fluid signal intensity around calcifications suggests active inflammation during the acute phase. The edema may be capsular or pericapsular, bursal, ligamentous/periligamentous, or peritendinous.[2] Calcifications appear hypointense on all pulse sequences and exhibit blooming artifacts on MRI. However, small calcifications may be overlooked on MRI, highlighting the significance of CT. MRI is the sole technique capable of assessing bone edema and aids in identifying and localizing calcific deposits and surrounding edema, as well as bursitis.[2,3]

CT is superior to USG in visualizing deeper soft tissue calcifications or the deeper surface of a calcification. Additionally, it surpasses radiographs when dealing with superposition of adjacent bones to accurately localize the calcification.[8,9]

Differentiating HADD from other soft tissue calcifications is vital as it changes the course of management. Differentials to be considered are septic arthritis and crystalline arthropathies. HADD clinically may mimic septic arthritis, with the clinical presentation of localized pain and reduced joint mobility. However, one factor that may be helpful in differentiation is a mildly elevated or even normal level of white blood cells in HADD. Crystalline arthropathies pose another diagnostic dilemma. On imaging, gouty tophi are typically less dense and demonstrate more soft tissue density than HADD calcifications. Moreover, gout is usually associated with elevated urate levels and joint destruction. Calcium pyrophosphate deposition disease involves articular cartilage, leading to joint destruction[9,10]

Management strategies for HADD may include local anesthetic or corticosteroid injection and oral nonsteroidal anti-inflammatory drugs. These measures aim to provide symptomatic relief and reduce the disease's clinical course. One treatment modality that can be used to eliminate the calcium deposit and avoid surgery is ultrasound-guided barbotage, which is done as an outpatient procedure. Barbotage refers to the percutaneous intervention leading to the fragmentation and aspiration of calcific material. Barbotage is effective in the short term and in the long term in periarthritis treatment, with results similar to or better than those published for other techniques like shockwave therapy and are not worse than those for surgery.[11,12] It is minimally invasive, widely available, and allows patients to return to work quickly.[13]

Conclusion

This case represents the first documented instance of calcific enthesitis of LPFL. It underscores the importance for radiologists and clinicians to consider acute calcific enthesitis of LPFL when evaluating anterolateral knee pain in patients without trauma. It emphasizes the significance of recognizing its appearance on different imaging modalities and distinguishing between acute and chronic phases, as this differentiation can influence the management approach.

Patient's Consent

Informed consent obtained from patient.

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None.

Conflict of Interest

None declared.

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