Case Resolution

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Hydroxyapatite Crystal Deposition Disease

ABSTRACT
The disease is described, along with its imaging findings as observed with each diagnostic modality.
Keywords: Knee; calcifications; pain.
Level of Evidence: IV

Enfermedad por depósito de cristales de hidroxiapatita

RESUMEN
Se desarrolla la enfermedad y cómo se visualiza las imágenes, en cada caso, según el estudio.
Palabras clave: Rodilla; calcificaciones; dolor.
Nivel de Evidencia: IV

DIAGNOSIS

Hydroxyapatite crystal deposition disease.

DISCUSSION

Crystal deposition diseases comprise a group of conditions in which the accumulation of crystals within the joint space or periarticular tissues leads to acute or chronic inflammatory manifestations. From a practical standpoint, they can be classified into three main categories: those due to monosodium urate deposition (gout), those related to calcium pyrophosphate deposition, and those secondary to basic calcium phosphate deposition.[1-3]

Hydroxyapatite deposition disease belongs to the latter group. In the literature, it is referred to by various terms that partly reflect the anatomical structure involved and partly the clinical presentation: hydroxyapatite deposition disease, hydroxyapatite crystal deposition disease, basic calcium phosphate deposition, calcific tendinitis, calcific periarthritis, calcific bursitis, and, in intra-articular locations or more destructive forms, hydroxyapatite arthropathy. Within this spectrum, the so-called Milwaukee syndrome represents an advanced form of arthropathy associated with basic calcium phosphate crystals, most commonly described in the shoulder, although knee involvement has been reported in some series.[1-5]

Unlike calcium pyrophosphate deposition disease, which typically presents as chondrocalcinosis with linear or lamellar calcifications in cartilage and fibrocartilage, and urate deposition, which tends to produce bone erosions and tophi, hydroxyapatite deposition disease is characterized by amorphous, periarticular calcifications most commonly located in tendons, bursae, and the joint capsule, and less frequently in ligamentous structures. This distinction is important in imaging assessment, as it helps guide the differential diagnosis based on the pattern of distribution and the morphology of the calcifications.[1-4,6]

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From a pathophysiological perspective, three phases are described: precalcific, calcific, and postcalcific, a classification originally established for calcific tendinitis of the shoulder and applicable, by extension, to other periarticular locations. The calcific phase, in turn, includes formative, resting, and resorptive stages. The resorptive stage usually corresponds to the period of greatest clinical expression, as the deposit may fragment or liquefy and trigger a marked inflammatory response in the surrounding tissues. This evolutionary behavior explains the variability of imaging findings and the fact that the same lesion may appear as a well-defined calcification at one time and as a poorly defined lesion with perilesional edema at another.[1,3,6]

Although the shoulder is the most common site of involvement, the knee is also a recognized location. In this joint, the quadriceps tendon, patellar tendon, periarticular bursae, capsule, and ligamentous structures may be affected. Involvement of the medial collateral ligament is uncommon but of particular interest, as it may mimic enthesopathy, post-traumatic sequelae, or avulsion injuries.[1-4,6]

On radiographs, the condition typically appears as an amorphous, rounded, or oval calcification with variable density. During resting phases, it tends to be more homogeneous and well defined, whereas in symptomatic or resorptive phases, it may appear faint, cloud-like, or poorly defined. Radiography therefore remains the most useful initial modality for detecting calcifications and guiding diagnostic suspicion.[1,3,4,6]

Computed tomography allows more precise confirmation of the calcific nature of the deposit, accurate assessment of its size and location, and evaluation of possible cortical erosions or extension into adjacent tissues. It is also particularly useful in the differential diagnosis with mature ossification processes, such as Pellegrini-Stieda lesion, in which imaging typically shows trabecular or cortical organization, unlike the amorphous appearance characteristic of hydroxyapatite deposits.[1,3,6]

Ultrasound demonstrates the deposits as hyperechoic foci, with or without posterior acoustic shadowing, and allows real-time assessment of the inflammatory changes in adjacent soft tissues. In more active phases, Doppler imaging may show associated hyperemia. In addition, ultrasound has therapeutic value, as it enables image-guided procedures such as aspiration or lavage in selected cases.[1,3,6]

Magnetic resonance imaging is not the most sensitive modality for detecting calcifications but is highly useful for assessing the inflammatory context. Deposits typically appear as foci of signal void or hypointensity on all sequences, associated with perilesional edema, bursitis, or reactive synovitis. MRI may pose diagnostic challenges when interpreted in isolation, as the inflammatory changes may predominate over visualization of the deposit and mimic traumatic, infectious, or even neoplastic conditions. For this reason, correlation with radiography, CT, or ultrasound is essential.[1,3,6]

In the cases presented, the findings were located along the medial aspect of the knee, anterior to the femoral insertion of the medial collateral ligament, with calcific morphology and associated perilesional inflammatory changes. The anatomical distribution, the appearance of the deposits, and the adjacent soft-tissue reaction constitute a pattern consistent with hydroxyapatite deposition in the medial capsuloligamentous region. In one case, ultrasound confirmed the calcific nature of the finding, whereas in the other, radiography demonstrated faint calcifications in the same location, supporting this interpretation.[6]

In the differential diagnosis of medial knee calcifications around the insertion area, the following entities should be considered: enthesopathy, remote avulsion injury, post-traumatic ossification (Pellegrini-Stieda lesion), medial bursitis, and other crystal-induced arthropathies, particularly calcium pyrophosphate deposition disease. Recognition of the morphological pattern of the deposits and their multimodality correlation helps avoid misinterpretation and guides appropriate diagnostic and therapeutic management.[1-3,6]

From a therapeutic standpoint, this is generally a self-limiting condition initially managed conservatively with relative rest, physical therapy, and nonsteroidal anti-inflammatory drugs. In cases of persistent symptoms, interventional options may be considered, such as extracorporeal shock wave therapy and image-guided percutaneous aspiration or lavage. Arthroscopic or open surgery is reserved for severe or refractory cases. In this context, imaging plays a role not only in diagnosis but also in the planning and guidance of therapeutic procedures.[1,3]

CONCLUSIONS

These two cases illustrate a rare and likely underdiagnosed cause of medial knee pain: perinsertional calcific deposits of the medial capsuloligamentous complex, which, in their symptomatic phase, may be associated with marked inflammatory changes and mimic trauma, infection, or other arthropathies.

The diagnostic approach should be based on multimodality imaging correlation. Radiography is essential for identifying calcifications, even when subtle. Computed tomography confirms the deposit, precisely defines its location and extent, and helps differentiate calcification from ossification. Ultrasound confirms its calcific nature, may provide information about the stage of the process, and enables image-guided therapy. Finally, magnetic resonance imaging delineates the extent of edema and reactive changes, although it requires correlation with calcium-sensitive modalities to avoid misinterpretation.

In the medial knee, including this entity in the differential diagnosis when evaluating enthesopathy or Pellegrini-Stieda lesion helps prevent overdiagnosis of chronic ligament injury or post-traumatic sequelae and reduces unnecessary additional studies or interventions.

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