Conventional Radiology in Crystal Arthritis

Gout, Calcium Pyrophosphate Deposition, and Basic Calcium Phosphate Crystals

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KEYWORDS

• Gout • Calcium pyrophosphate • CPPD • Crystal deposition • Crystal arthropathy • Basic calcium phosphate • Hydroxyapatite

KEY POINTS

- Crystal deposition diseases are a common finding and their radiographic features should be known to help the referring physician in the differential.
- Classic features of gout are tophi, large para-articular erosions contrasting with articular-space sparing, and bone hyperostosis without bone rarefaction.
- CPPD arthropathy involves joints usually spared by osteoarthritis, with severe joint narrowing, marked subchondral osteosclerosis, with no or few osteophytes.
- Basic calcium phosphate deposits are usually amorphous and frequently asymptomatic.
- Their density and shape vary when resorbing or migrating into an adjacent structure during an acute and painful flare.

This article reviews the radiographic aspects of the 3 main crystal deposition diseases: monosodium urate (gout), calcium pyrophosphate (CPPD), and basic calcium phosphate (BCP).

GOUT

Introduction

Gout is a crystal-induced deposition disease caused by saturation and precipitation of monosodium urate (MSU) crystals. This common condition affects up to 1% of the general population. Its prevalence is increasing in both developed[1,2] and developing[3,4] countries, mostly because of the evolution of lifestyle and particularly eating habits.[5]

Men are affected 4 to 10 times more often than women,[6] gout being the most frequent cause of inflammatory arthritis in men older than 60 years.[7] In cases of early onset, secondary causes must be considered, because their diagnosis can change further patient care.

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Several factors are required for MSU crystals to form. The main underlying factor is hyperuricemia, which is a consequence of either excessive production of uric acid or a lack of its clearance.

Excessive production can be either exogenous, due to excessive intake of purines (eg, meat, seafood, alcohol),[6,8] or endogenous, especially in case of massive cell lysis (eg, chemotherapy or myeloproliferative disorders). Insufficient uric acid clearance can result from chronic kidney disease, drug interactions, or genetic predispositions. Familial diseases that directly affect enzymes involved in purine metabolism are less frequent, such as Lesch-Nyhan disease.

Several local factors can play a crucial role in MSU crystal formation, such as trauma or repetitive microtrauma, arthritis, infection, lack of tissue perfusion, lower blood pH, or lower tissue temperature.[9]

Clinically, gout is characterized by recurrent episodes of arthritis involving one or multiple joints. In the absence of care, the disease can evolve into chronic tophaceous gout. Ultimately, patients can suffer from visceral consequences, such as gouty nephropathy. However, hyperuricemia remains asymptomatic in 90% of patients.

Acute flares are either due to oversaturation of MSU crystals in synovial fluid or to their relapse from hyaline cartilage into the joint, triggering inflammation pathways and resulting in the production of cytokines such as tumor necrosis factor-α and interleukin (IL)-1β.[10] Secondary recruitment of neutrophils worsen local inflammation.[11]

Acute gouty arthritis is typically of sudden onset, involving one or few joints (less frequently polyarticular), with a preference for inferior limbs (in 85% of cases[12]), particularly for the first metatarsophalangeal space. Clinical and biological inflammatory patterns are frequently noted,[13] sometimes mimicking an infectious process, also leading to difficult differential diagnoses with inflammatory diseases, such as rheumatoid arthritis. The synovial fluid analysis would display an inflammatory fluid containing 2000 to 5000 white blood cells per mm^3 (sometimes more) and typical MSU crystals, elongated and birefringent under polarized light, but no germs on Gram staining.

These acute flares usually resolve spontaneously in 5 to 10 days, followed by an asymptomatic period lasting from months to years before recurring.[14] Chronic tophaceous gout is characterized by the presence of *tophi*, which are MSU deposits in either hypodermic soft tissues, articular and para-articular spaces, tendons, or bursae. It tends to occur years after the initial episode of gout and in the absence of adequate treatment (5 years for 30% of patients).[12] *Tophi* are related to the duration and levels of hyperuricemia, and tend to diminish under treatment.

Imaging Findings

In the event of an acute arthritis (if the patient has not yet reached the chronic tophaceous gout stage), plain radiographs are most of the time normal or display a nonspecific joint effusion and/or periarticular soft tissue edema (**Fig. 1**).

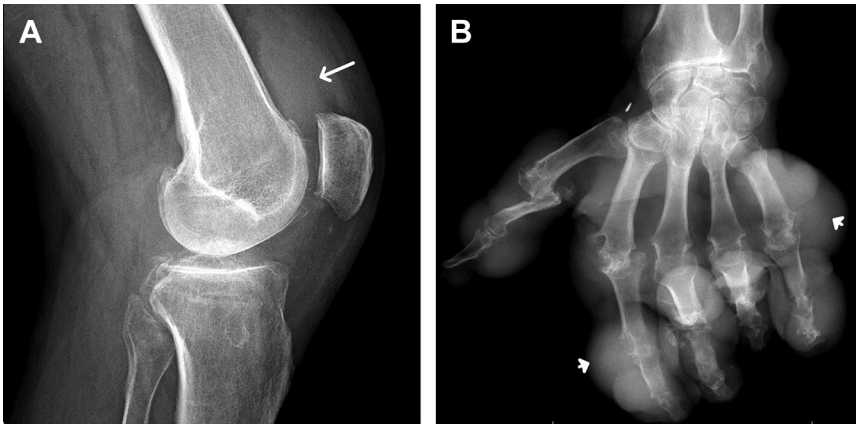

Fig. 1. (*A*) Acute knee arthritis with nonspecific intra-articular effusion (*arrow*); joint aspiration showed MSU crystals. (*B*) Severe chronic tophaceous gout with large subcutaneous *tophi* (*arrowheads*) and polyarticular involvement of the hand.