Crystal Deposition Disease

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Synopsis

Several crystals (urate, CPPD and hydroxyapatite) can deposit in tissues of the musculoskeletal system in asymptomatic individuals. I in some cases, those deposits can be associated with diseases such as Gout, Pseudogout and Calcific Tendinitis. MRI examination can be very useful in the diagnosis of such entities, differential diagnosis and help treatment decisions.

Several crystals can deposit in tissues of the muscle skeletal system in asymptomatic individuals, and in some cases, be associated with diseases. There are various imaging features for common crystal-related deposition entities including monosodium urate, calcium pyrophosphate dihydrate as well as hydroxyapatite. Magnetic resonance imaging (MRI) examination features associated with crystal deposition diseases include: a) location (hyaline cartilage and fibrocartilage, tendons, ligaments, bone, synovial membrane), b) morphology (size, soft-tissue signal characteristics), c) surrounding tissue signal alteration, d) arthritic changes (bone, synovial membrane), leading to a deferential diagnosis among those crystal-related deposition entities and other diseases. Monosodium urate monohydrate crystals or uric acid deposits into tissues can cause a disease named Gout. It happens when hyperurecimia is above 6 to 7 mg/dL. Gout is the most common inflammatory arthritis in men over the age of 30 years. Radiographic findings of gout do not occur until the disease has been present for at least 6 or more years. In acute phase MRI findings of the joint effusion and synovial thickening are nonspecific. Gouty tophi are mostly of low- or intermediate-signal intensity on T1W images with variable gadolinium enhancement. The imaging findings of tophi on T2W images are variable. Calcification, fibrosis, or hemosiderin can occur within the tophi. With dual source computed tomography, the uric acid deposits can be differentiated with high accuracy and high imaging contrast. Calcium pyrophosphate dihydrate (CPPD) crystal deposit can be found with a high frequency in older people in the hyaline cartilage, fibrocartilage, tendons and ligaments. Calcification in the spinal ligaments specifically around the odontoid process is common in this entity. Focal calcifications forming conglomerates around joints like tophaceous gout may be also seen. When CPPD crystal deposits become symptomatic it receives the name of Pseudogout, most commonly occurring in knee and wrist. It is often seen in women >80 years old (``disease of octogenarians´´). The shoulder is a non-weight-bearing joint and degenerative changes and narrowing and loss of articular cartilage seen in this joint with the absence of prior injury raises the possibility of CPPD. CPPD deposit may cause arthritis with severe inflammation or may present as destructive joint disease, which may mimic neuropathic joint. The appearance of scapholunate advanced collapse (SLAC) in the wrist is an example of destructive features of pseudogout. Intervertebral disk CPPD crystal deposits can be found in asymptomatic individual but in some more rare cases can cause destructive disc disease. Calcium hydroxyapatite (HA) deposits also can be found in asymptomatic individuals, are often periarticular or intratendinous. Although CPPD is the most common cause of radiographic signs in chondrocalcinosis, both CPPD and calcium hydroxyapatite can be present. On MR imaging, HA deposits are of low signal. Moreover, there is often overlying inflammation and edema, which the radiologist may misinterpret as tenosynovitis or joint synovitis with infection or injury. At high calcium concentrations (above 30% to 40%) susceptibility effects and decreases in proton density dominate, leading to signal intensity loss. However, T1 shortening effects resulting in hyperintensity on T1-weighted images are also present. They have been attributed to surface interaction of protons with calcified tissue. At lower concentrations of calcium, T1 shortening effects dominate, resulting in isointensity or even hyperintensity. Gradient echo sequences best show these calcific foci. The deposition of HA may mimic an acute inflammation. For those patients that are symptomatic, trauma or acutely increased use of the joint seem to be initiating factors. Calcific tendinosis is most commonly seen in the shoulder in the distal supraspinatus tendon insertion at the greater tuberosity of the humerus. Less common locations may include tendons of infraspinatus, subscapularis, and deltoid, wrist, elbow, gluteus maximus, knee, and neck. Rarely erosion of bone adjacent to calcification at the insertion site of the tendons can be visualized.

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Figures

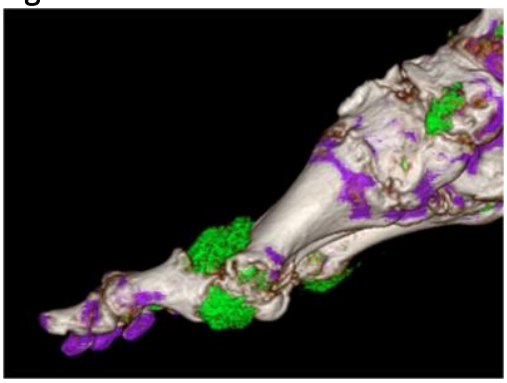

Figure 1. Computed tomography with dual source technology demonstrating urate crystal deposit (green color) at MTP joint in a patient with Gout. MRI of this patient only demonstrated signs of nonspecific synovitis.

Figure 2. Sagittal thin (3mm) slice of a cadaveric specimen demonstrating CPPD crystal deposits in meniscus and hyaline cartilage, chondrocalcinosis (white dots). Image from UCSD research laboratory VA health care San Diego.

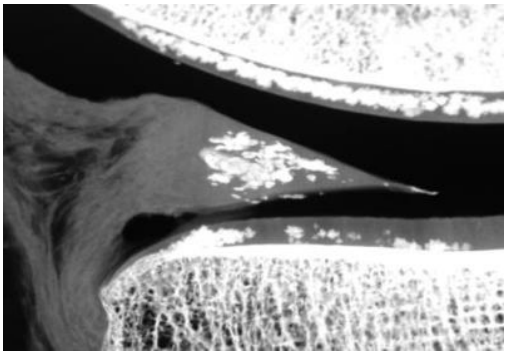

Figure 3. Sagittal slice Faxitron (High Definition special radiographic examination) of the cadaveric specimen with chondrocalcinosis (CPPD).Image from UCSD research laboratory VA health care San Diego.

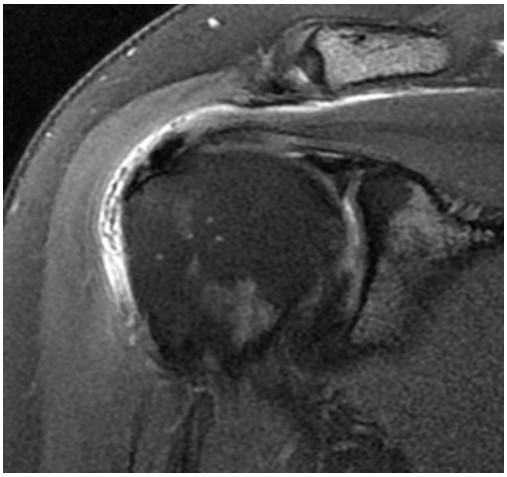

Figure 4. Calcific tendinitis of supraspinatus tendon caused by hydroxyapatite crystal deposits within the tendon with migration of some crystals (black dots) to subacromial inflamed bursa. Coronal T2-weighted fat suppressed MR image.

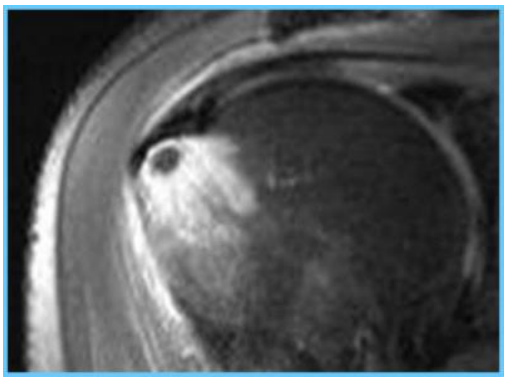

Figure 5. Hydroxyapatite crystal deposit migrated to humeral greater tuberosity (black low signal dot) with great adjacent bone marrow edema. Some crystal deposits still within the supraspinatus tendon (low signal intensity). Coronal-T2 weighted fat suppressed MR image.