Radiology

Imaging of CPPD Disease (Calcium Pyrophosphate Dihydrate Crystal Deposition) Simulating Osteoarthritis of the Knee: A Case Report

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Abstract

Case Report

Objective: The purpose of this case report is to describe a case where calcium pyrophosphate dehydrate crystal deposition disease (CPPD) simulated osteoarthritis of the knee. *Clinical Features:* A 76-year-old woman had a 10 days history of severe right knee pain accompanied by mild swelling and fever, the onset was sudden and did not involve a history of trauma or previous injury to the right knee. Inspection and palpation revealed pain along the medial joint line and marked difficulty ambulating. Diagnostic imaging was performed and showed degenerative changes with diffuse calcification of the fibrocartilage and hyaline articular cartilage within the knee joint and the medial collateral ligament consistent with the appearance of CPPD crystal deposition. Intervention and Outcome: the patient was treated as pseudogout disease and was prescribed colchicine and non steroidal anti-inflammatory drug (NSAID); Eight days later, at follow-up, the knee 's pain and immobilization symptomatology reduced 50% and 90% after 4 weeks. *Conclusion:* Although knee osteoarthritis is much more common than knee CPPD, it is important to consider both diagnoses in elderly patients who present with unilateral knee pain. Diagnosis should be based on clinical presentation, history, and radiographic or histological means to ensure accuracy and proper diagnosis.

Keywords: Osteoarthritis; Chondrocalcinosis; Calcium pyrophosphate; Musculoskeletal diseases, IRM, CT.

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INTRODUCTION

The calcium pyrophosphate deposition disease (CPPD) is a crystal arthropathy and may present with various clinical manifestations such as asymptomatic CPPD, osteoarthritis with CPPD, acute CPPD crystal arthritis (formerly pseudogout), and chronic CPPD crystal inflammatory arthritis [1]. Aging, trauma, and osteoarthritis are known as major risk factors for CPPD. However, if CPPD develops in individuals younger than 45 years, metabolic diseases such as hyperparathyroidism, hemochromatosis, and hypomagnesemia should be considered [1, 2]. Acute CPP arthritis can occur as monoarticular or oligoarticular and usually involves large peripheral joints such as the knees, wrists, and ankles [1, 2]. CPPD is characterized by the sudden onset of severe pain, swelling, and periarticular erythema, and systemic symptoms such as fever, chills, and weakness may occur. Such inflammatory attacks can be triggered by medical or surgical interventions, and the differential diagnosis between septic arthritis and acute CPPD may not always be easy [3].

Radiology tests (Radiographics, CT and MRI) plays à huge role in evaluation of calcium pyrophosphate dehydrate crystal deposition disease (CPPD). The hallmark finding is chondrocalcinosis of hyaline or fibrocartilage [1]. CPPD is the third most common inflammatory arthritis, and it tends to affect areas not typically involved by degenerative jointdisease [2]. Chondrocalcinosis is depicted as radiopaque calcifications that are linear, punctate, or granular in shape and are seen in the cartilage within affected joint spaces [3]. In the knee, meniscal involvement is seen as coarse granular calcifications, whereas hyaline cartilage involvement appears more as linear densities that parallel the articular surfaces [5]. Chondrocalcinosis is often asymptomatic and occurs most commonly in women older than 80 years [3]. It predominantly affects large joints including the knees, wrists, and hips [6, 7]. It most commonly affects the knee, where cartilage space narrowing and osteophytes may involve all 3 compartments.

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CLINICAL CASE

A 76-year-old woman was previously diagnosed with diabetes mellitus and hypertension presented to urgences with a 10 days history of severe right knee pain and mild swelling.

The onset of symptoms occurred suddenly with no known history of recent trauma or previous injury to her right knee. The pain was described as a constant, deep, achy pain that became sharp upon walking. The pain did not refer into the lower extremity. The laboratory results were as follows: creatinine: 0.75 mg/dL, calcium: 8.4 mg/dL, albumin: 3.91 g/dL, white blood cell count: 7.50 $10^3/\mu$ L, hemoglobin: 10.90 g/dL, hematocrit: 34.2%, platelet: 460 $10^3/\mu$ L, C-reactive protein (CRP): 172 mg/L, erythrocyte sedimentation rate (ESR): 122 mm/h, rheumatoid factor (RF): negative.

A Microscopic analysis of joint aspirate with polarization-optical finding rhombus-shaped and rodshaped crystals of calcium pyrophosphate dihydrate (CPPD) crystals.



Figure 1

A CT was performed finding Multiple osteolytic subchondral lesions with marginal sclerosis without rupture of the cortex opposite, in certain sites of some partitions, Arthrotic changes made of marked articular pinching at the level of the internal condyle, subchondral geodes, and some osteophytes visible at the level of the lateral condyle; Joint effusion of moderate abundance and Intra-articular linear calcifications (Figure 1).

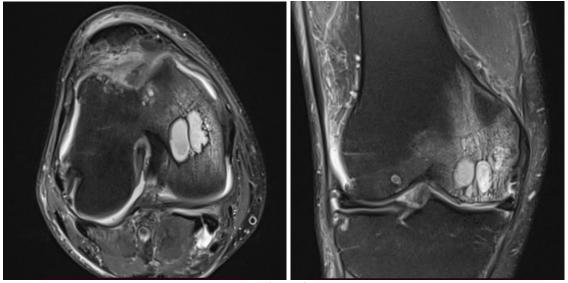


Figure 2

MRI finding some giant subchondral cysts associated to multiples osteolytic lesions surrounded by

marginal sclerosis at the level of the internal femoral condyle in low T1, high T2 DP, enhanced intensely in

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level of their fleshy portions after injection of Gadolinium with irregular contours; associated with nearby bone edema, Nodular synovial thickening enhanced after Gadolinium injection, no sign of cortical rupture or infiltration of soft tissues (Figure 2).

DISCUSSION

Symptoms of OA and severe pain caused by acute CPPD crystal arthritis are very similar in nature. Both OA and CPPD are common articular diseases especially in elderly people [5, 9]. Although chondrocalcinosis is most commonly due to CPPD crystals, it is not exclusive of this disease and could appear as an incidental finding or coexist with structural changes that resemble OA [15]. Common findings of OA on radiographs include nonuniform joint space narrowing, osteophyte formation, subchondral cyst formation, and subchondral sclerosis. CPPD is characterized by a radiographic finding of calcification within the articular cartilage (Chondrocalcinosis) and periarticular soft tissues and may involve a number of variants.

Although OA and CPPD are unique disorders, crystal deposition can also be present in patients with OA, and it has been suggested that destruction of the articular cartilage caused by the crystals may predispose a patient to developing secondary OA [7]. In addition, it has also been hypothesized that OA could favor local development of CPPD deposits in the articular cartilage due to metabolic changes in the joint matrix [5]. Although radiographic evidence of synovial calcification can be an effective tool in the diagnosis of CPPD, the absence of calcification does not rule out the diagnosis [15]. Joint aspiration and histologic evaluation of crystals in the synovial fluid by light microscopy, compensated polarized light microscopy, or phase contrast light microscopy are considered the criterion standard in the diagnosis of CPPD [2]. Although joint aspiration with crystal identification is the criterion standard, this test is rarely ever performed in a clinical setting because the exact crystal composition is unlikely to alter either the management or the prognosis.

Chondrocalcinosis is indeed one of the most common joint disorders, affecting up to 5% of the human population, with the prevalence rising because of the aging population [15]. Although OA is much more prevalent than CPPD, it is important to consider both diagnoses in elderly patients who present with unilateral knee pain. By knowing one's anatomy and through observing the changes that the disease has produced in the anatomy, it is possible to identify the pathologic process(es) that produced those changes. Therefore, diagnosis should be based on clinical presentation, history, and radiographic or histological means to ensure accuracy and proper diagnosis.

CONCLUSION

Knee OA is more common than knee CPPD. However, it is important to consider both diagnoses in elderly patients who present with unilateral knee pain. In this particular case, the patient was diagnosed with CPPD through, microscopic examination oriented by knee imaging. She was treated acutely and her pain and knee limitations decreased by approximately 80% after 4 weeks.

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