Chondrocalcinosis a Differential in Young Patient: A Case Report

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Abstract

Calcium Pyrophosphate Dihydrate (CPPD) crystal deposition is the hallmark of chondrocalcinosis (CC), a disorder that usually affects the elderly and manifests as inflammatory arthritis. It is indeed rare among younger patients but warrants investigation to find the underlying causes. This case report showcases a 37-year-old woman who had a history of recurrent, self-limiting knee pain for four years and right hip pain that lasted for a year. A clinical examination showed widespread tenderness and swelling in the knees. Elevated erythrocyte sedimentation rates and distinctive punctate and linear calcifications seen on radiological examination of the right hip joint and both knees were part of the diagnostic evaluation. Synovial fluid analysis revealed rhomboid or rod- shaped CPPD crystals under polarized light microscopy, confirming the final diagnosis. The initial screening for metabolic causes were negative. Conservative methods of management included physiotherapy and analgesics for pain relief. This case emphasizes the significance of taking into account CC in younger patients who exhibit radiographic evidence of calcifications and persistent joint pain, even in cases where typical metabolic risk factors are not conclusive. Appropriate patient care depends on a precise diagnosis made via synovial fluid analysis.

Keywords: Calcium pyrophosphate deposition disease, chondrocalcinosis, crystal-induced arthropathy, young adult, synovial fluid analysis.

Introduction

Calcium Pyrophosphate Dihydrate (CPPD) crystal deposition disease, or chondrocalcinosis (CC), is a common type of crystal-induced arthropathy that primarily affects the knee and is characterized by the deposition of CPPD crystals in articular and periarticular tissues [1]. Acute or chronic inflammatory arthritis, pseudo-gout attacks, or asymptomatic radiographic CC are common presentations, especially in older people [1-3]. Even though CPPD is more common in older people —some studies show that 30% to 50% of patients are over 85—and its prevalence rises dramatically with age [2, 4], its symptoms can frequently resemble those of other arthropathies, making diagnosis more difficult [5]. Because familial forms and certain metabolic conditions can result in premature severe disease, the presence of CPPD in younger patients (under 60 years of age) is relatively uncommon and often warrants investigation into underlying metabolic or genetic factors [2, 5, 6]. This case study highlights the diagnostic issues and treatment of CPPD in an atypical age group with a chronic, self-limiting presentation by presenting an unusual manifestation of CC in a 37-year-old female patient.

Case Report

A female of age 37 years presented with chief complaint of chronic bilateral knee pain with right hip pain and swelling of one year of duration. She gave a history of recurrent knee pain since past 4 years and was managing with analgesics. This pain was self-limiting in nature and resolves within two to three weeks. There was no family history of similar complaints.

On examination, patient had generalised knee swelling and knee tenderness with full range of motion of knee but painful terminally. Radiological assessment revealed calcification of menisci in both knees and calcification of articular cartilage of right hip joint (Fig. 1). Suspicion of CC was made, although there were negative findings on screening for metabolic causes in order to confirm the diagnosis, further investigations were done which showed elevated erythrocyte sedimentation rate at 35 mm/hr and a uric acid level of 6.8 mg/dL, with all other routine laboratory tests falling within normal limits.

Radiological evaluation played a crucial role in the diagnostic assessment. Plain radiographs of the both knee were performed to identify characteristic features that is punctate or linear calcifications within the articular cartilage which is a hallmark sign of CC. To confirm the diagnosis, synovial fluid analysis was done which showed rhomboid or rod shaped calcium pyrophosphate dihydrate crystals exhibiting weak birefringence under polarized light microscopy. Based on clinical presentation, radiological findings and identification of CPPD crystals in synovial fluid analysis, a definitive diagnosis of CC was confirmed. Patient was managed with conservatively aiming reduction in pain using analgesics and physiotherapy and reducing uric acid.

Discussion

This case report details a 37-year-old woman who has had recurrent knee pain for four years and has been experiencing chronic, self-

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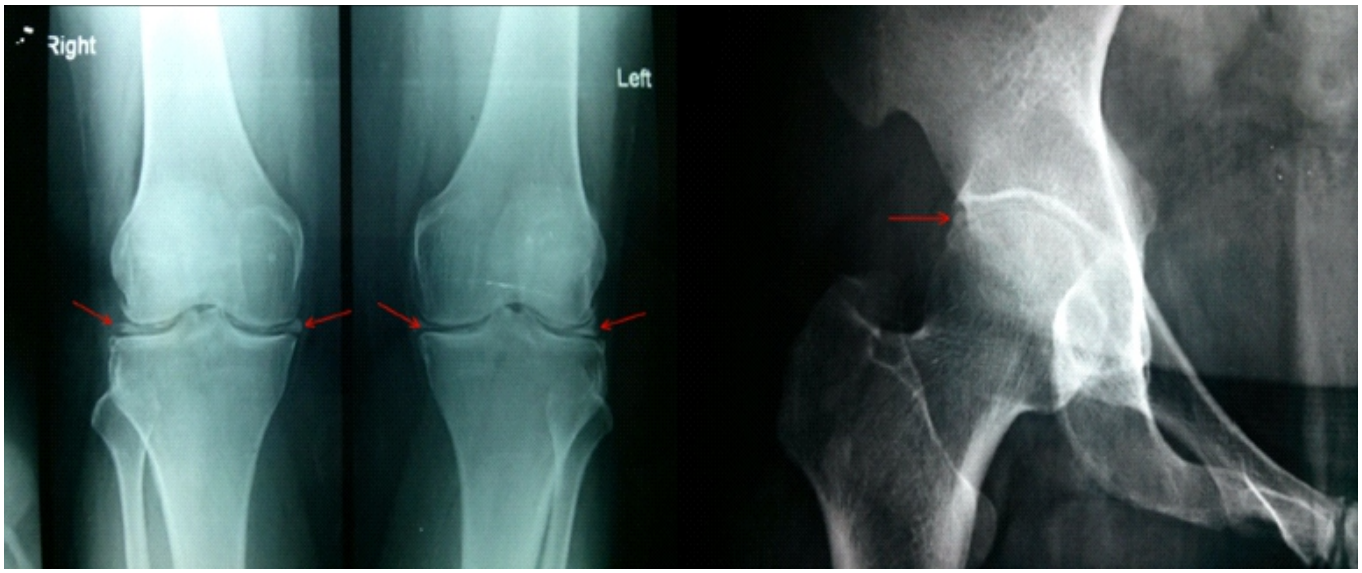

Figure 1: Showing calcification of menisci in both knees and calcification of articular cartilage of right hip joint

limiting bilateral knee pain and right hip pain for a year. The patient's relatively young age makes this presentation noteworthy because CPPD disease usually affects people over 60, and its prevalence rises with age [1, 2, 4]. Since a sizable portion of confirmed cases occur in patients under the age of 55, some research indicates that the prevalence of CPPD at a young age may actually be underestimated [7]. Her symptoms are consistent with chronic CPPD arthropathy because they are self-limiting and persistent, and she also has widespread knee pain and swelling [1].

In this instance, the diagnostic procedure made good use of both laboratory and radiological results. Only an elevated erythrocyte sedimentation rate of 35 mm/hr and a uric acid level of 6.8 mg/dL were found in the initial investigations; all other investigations were within normal limits. Although hyperuricemia is not a direct diagnostic marker for CC itself, it can coexist with CPPD [1]. Nonetheless, punctate or linear calcifications in the articular cartilage of the right hip joint and both knees are radiological indicative of CC [1, 2]. For the purpose of identifying these calcifications, radiographic imaging is essential, especially in locations such as the symphysis pubis, the menisci of the knees, and the triangular cartilage of the wrist [1, 2]. Diffuse calcification, or cartilage icing, and CC are frequently observed in CPPD and can help distinguish it from other conditions such as gout [8]. Additionally, CPPD can be detected with high sensitivity using ultrasound imaging, which reveals hyperechoic deposits in a variety of joint structures [9].

Finding CPPD crystals in the synovial fluid is necessary for a conclusive diagnosis of CPPD crystal deposition disease. The presence of rhomboid or rod-shaped crystals with weak positive birefringence under polarized light microscopy, which is regarded as the gold standard for diagnosis, was confirmed in this patient by synovial fluid analysis [1, 7]. Screening for secondary causes of CPPD, such as hypothyroidism, primary hyperparathyroidism, hereditary hemochromatosis, hypomagnesemia, and hypophosphatasia, is typically advised given the patient's age [1–3, 5, 6, 10]. Even though the initial workup for metabolic causes was found to be negative, it is crucial to keep an eye out for these conditions in younger people with CPPD because they can be linked to the disease at any age [7]. There are also familial forms of CPPD, which should be investigated, particularly in patients under 60 [2].

In this instance, conservative pain management techniques included physiotherapy and analgesics. Although there isn't a specific treatment to get rid of the CPPD crystals or stop or slow their deposition [11, 12], symptom relief and inflammation reduction are usually the main goals of management [2, 12]. This includes systemic glucocorticoids for acute attacks, colchicine (which can also be used prophylactically to prevent recurrent flares), and non-steroidal anti-inflammatory medications [2, 12]. Although there is little information on their effectiveness, NSAIDs and/or colchicine, hydroxychloroquine, low-dose glucocorticoids, and methotrexate may be used for chronic calcium pyrophosphate inflammatory arthritis, as this patient has [12]. Adjunctive non-pharmacologic treatments include arthrocentesis, heat, ice, and rest [13].

This case emphasizes how crucial it is to take into account CPPD as a differential diagnosis in younger patients who exhibit radiographic evidence of calcifications and chronic joint pain, even in the absence of typical metabolic causes at first. In order to guide appropriate management and avoid needless or ineffective treatments, early and accurate diagnosis through synovial fluid analysis is essential [14].

Conclusion

This case study emphasizes that CPPD crystal deposition disease should be taken into consideration as a differential diagnosis in younger people experiencing chronic joint pain, even in the absence of clear underlying metabolic disorders, by highlighting an unusual presentation of CC in a 37-year-old female. The identification of CPPD crystals in synovial fluid analysis and the distinctive radiographic findings of cartilage calcification are crucial for the final diagnosis. The patient's symptoms were successfully reduced by conservative treatment using analgesics and physical therapy. This case highlights the need for a comprehensive diagnostic strategy for crystal arthropathies in a wide range of age groups.

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Declaration of patient consent: The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his/her consent for his/her images and other clinical information to be reported in the Journal. The patient understands that his/her name and initials will not be published, and due efforts will be made to conceal his/her identity, but anonymity cannot be guaranteed.

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