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CASE REPORT

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Monoarticular pseudogout of the hip presenting as septic arthritis: a case report

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Calcium pyrophosphate dihydrate (CPPD) disease is the second most common crystal-induced form of arthropathy, frequently seen in the knee, shoulder, wrist, elbow, and ankle. The acute form of the disorder is referred as pseudogout, which can cause a severe joint inflammation. We present a case of monoarticular pseudogout of the hip joint whose symptoms mimicked septic arthritis. The definitive diagnosis was only confirmed after the microscopic analysis of joint aspirate.

Key words: Hip; pseudogout; septic arthritis.

Calcium pyrophosphate dihydrate (CPPD) disease is the second most common crystal-induced form of arthropathy. CPPD disease usually presents as polyarthritis, most commonly involving the knee, shoulder, wrist, elbow, and ankle joint. The small joints of the hands and feet may also be affected in rare cases. The heterogenous presentation of CPPD disorders has led to its classification into seven subgroups (Table 1).[1] Pseudogout, the acute presentation of the disorder, occurs in one fourth of patients. Pseudogout can present as pseudo-rheumatoid arthritis, pseudo-osteoarthritis or pseudo-neurotrophic arthritis.[1] Recently, isolated pseudogout of the hip has been reported to mimic longstanding intermittent hip pain.[2]

We report a case of pseudogout in the hip joint presenting as septic arthritis. To our knowledge, this is the first case of pseudogout of the hip mimicking septic arthritis.[3]

Case report

An 89-year-old man presented with acute onset, severe hip pain, the inability to bear weight and fever. He had a history of fall three weeks before the onset of pain. No previous history of similar involvement of the hip joint was noted. He suffered from osteoarthritis of both knees, which had never flared up acutely. There was no family history of gout or pseudogout. He had a significant medical history that included ischemic heart disease, chronic renal failure, hypothyroidism, abdominal aortic aneurysm and diverticular disease. He was taking levothyroxine, aspirin, diltiazem, lansoprazole, amiodarone and simvastatin.

On physical examination, the right hip area was warm and exquisitely tender even on superficial palpation. Any attempt to move the hip was extremely painful. At presentation, a full blood count showed a normal white cell count and Acute-phase reactant protein (CRP) of 290 which rose to 315 with an erythrocyte sedimentation rate (ESR) of 40. Renal func-

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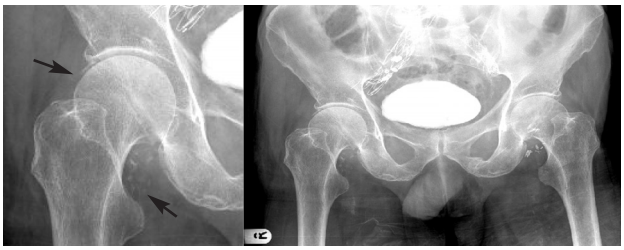

Fig. 1. Plain radiograph showing calcification within the hip capsule (**arrows**).

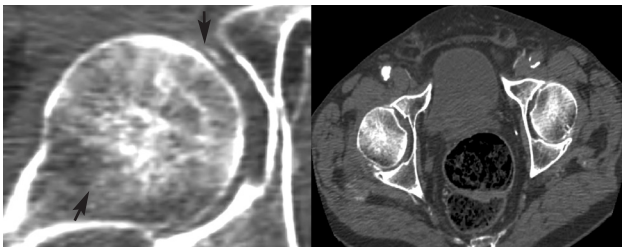

Fig. 2. CT images showing evidence of chondrocalcinosis (**arrows**).

tion was minimally deranged. Plain radiographs showed specks of calcification within the right hip capsule (Fig. 1). CT scan of the hip joint confirmed calcification within the joint space and revealed a collection around the joint (Fig. 2). MRI scan confirmed the presence of a hip joint effusion on proton density images with inflammatory changes around the joint and ipsilateral trochanteric bursitis (Fig. 3). A fluoroscope-guided hip aspiration under local anaesthesia (without steroid injection) of the affected hip joint yielded 2 ml of thick, yellow-coloured fluid.[4] Microscopy of the aspirate did not show any organism, but scanty polymorphs and lymphocytes. Further analysis of the sample under polarised light microscopy revealed birefringent crystals with optical characteristics of calcium pyrophosphate crystals.

The patient was treated conservatively with opioid analgesics. Due to the coexisting chronic renal failure and duodenal ulcer, NSAIDs could not be used. However, within three days of analgesic treatment, pain began to ease. CRP came down to 111 within one week and the patient could begin mobilization. He underwent rehabilitation and was eventually discharged on the seventeenth day.

Discussion

Onset of pseudogout occurs typically in the elderly at an average age of 72 years.[1] Symptomatic CPPD, however, has been reported to be more common in females.[5] It is known that the probability of CPPD increases with age.[6] There are valid associations with hypophosphatasia, hypomagnesaemia, and hyperparathyroidism, but the association with hypothyroidism is controversial.[7] Clearly, an abnormality of pyrophosphate and calcium metabolism is likely to be the precipitating factor. As in our case, impaired renal function with increasing age might be the underlying mechanism.

Table 1. Classification of CPPD disorders.

Group A	Pseudogout
Group B	Pseudorheumatoid
Group C and D	Pseudoosteoarthritis
Group E	Lanthanic/asymptomatic
Group F	Pseudoneurotrophic
Others	Tophaceous, spinal CPPD deposition, crowned dens syndrome, spinal stenosis

Pathogenesis of CPPD involves deposition of uricase-resistant CPPD crystals in cartilage and synovial fluid. Trauma may precipitate increased dissolution of the crystals in synovial fluid, which may be responsible for the acute symptoms. Deposition of crystals occur both in fibrocartilage and hyaline cartilage. The precipitating metabolic cause is uncertain. It is also unclear whether aging cartilage favours the deposition of crystals.[8] The presence of numerous polymorphs suggests an acute presentation. CRP is elevated and the joint may be swollen and acutely painful, mimicking septic arthritis.

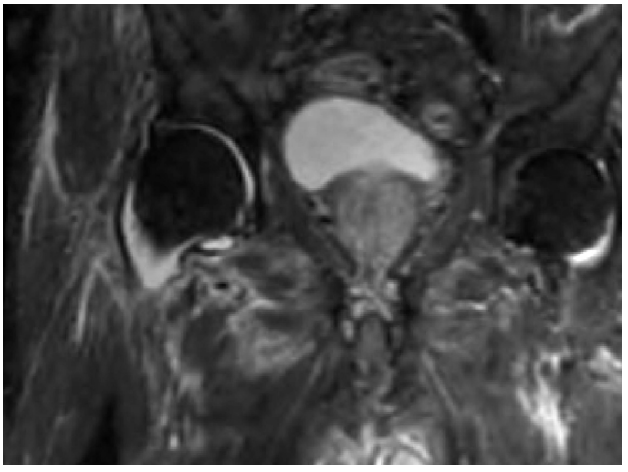

Fig. 3. MRI showing joint effusion and inflammation.

The presence of chondrocalcinosis on radiographs may be associated with CPPD disorders. The knee and wrist are the common sites of chondrocalcinosis. Monoarticular chondrocalcinosis of the hip has not previously been reported. Other characteristics in the advanced stage of the disease include joint space narrowing and subchondral cyst formation.

In the acute presentation of local tenderness with painful limitation of all hip joint movements in elderly patients, septic arthritis should be excluded first. However, patients should not be rushed to the theatre solely on clinical suspicion of septic arthritis. Aspiration of the hip joint should be attempted under image guidance to obtain microbiological confirmation of the diagnosis. In our patient, we managed to avoid surgery, and while NSAIDs could not be used for treatment, symptomatic relief was obtained using opioid analgesics. The clinical condition gradually improved and the patient was eventually discharged. In the previously reported case of pseudogout of the hip, diagnosis was eventually made with hip arthroscopy. This reinforces our belief that aspiration of the hip should be attempted before any surgical intervention is undertaken in these cases.

Conflicts of Interest: No conflicts declared.

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