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Case Report

Pseudogout in a Young Patient

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Abstract: Calcium pyrophosphate dihydrate (CPPD) crystal deposition disease is conventionally classified into cases that are hereditary, idiopathic (sporadic) or associated with other disorders. In the idiopathic form, the disease usually occurs in middle-aged or elderly patients. An earlier age of disease onset is observed in the hereditary form and the form associated with other disorders. Therefore, the occurrence of CPPD crystal deposition disease in a young patient merits thorough investigation for an underlying cause such as haemochromatosis, hyperparathyroidism, Wilson's disease, hypophosphatasia or hypomagnesaemia and requires a family study to investigate a possible hereditary cause. We report a case of a young female patient who presented with pseudogout at the age of 24 years; no associated diseases or familial occurrence were found despite a follow-up of more than 12 years.

Keywords: Calcium pyrophosphate crystal deposition disease; Pseudogout

Introduction

Calcium pyrophosphate dihydrate (CPPD) crystal deposition disease is predominantly a disease of the elderly [1]. The occurrence of this disease in young patients requires investigation for an underlying metabolic disease or for possible familial occurrence.

We present the case of a young patient who developed pseudogout at the age of 24 years. No metabolic diseases were found during a 12-year follow-up and there was no obvious familial occurrence.

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Case Report

This 36-year-old female patient has had recurrent arthritis for the past 12 years. She was aged 24 years when she first developed pain and swelling of the right knee, which subsided after 4 days after taking ibuprofen. Over the next 2 years, she had several similar attacks affecting both knees, which lasted for 4–7 days with a good response to ibuprofen.

At the age of 26 years, she was referred to the rheumatology clinic with an acute attack of arthritis involving the right knee. A radiograph of the knee revealed chondrocalcinosis bilaterally (Fig. 1). Aspiration of the synovial fluid showed a white cell count of 11 000/μl, with 88% neutrophils and 12% lymphocytes.

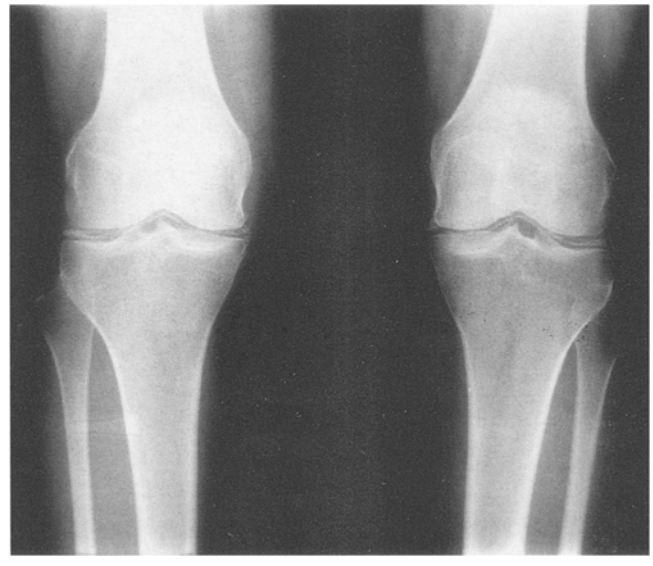

Fig. 1. Anteroposterior radiographs of both knees showing calcification of the articular cartilage typical of chondrocalcinosis.

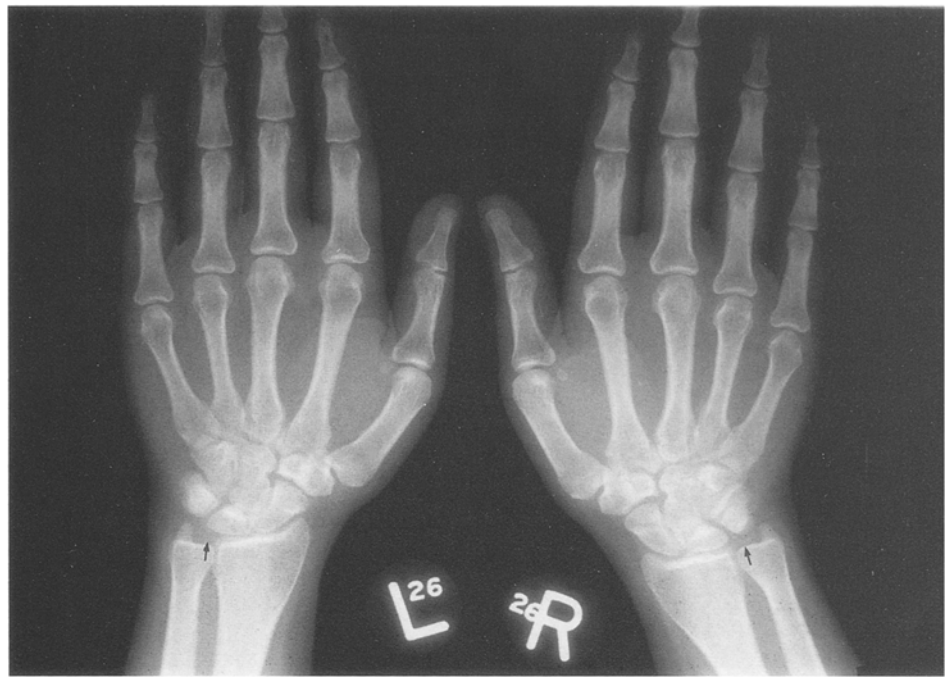

Fig. 2. Radiographs of both hands showing chondrocalcinosis in both radiocarpal joints.

Intracellular calcium pyrophosphate crystals were seen under a polarised microscope. A radiograph of both wrists revealed chondrocalcinosis of the triangular cartilage (Fig. 2).

A detailed family history was obtained, but did not show a similar history for her mother, father, four brothers, three sisters and cousins. Three of the siblings were older and four were younger than the patient.

Laboratory investigations revealed a white cell count of 12 000/µl, haemoglobin (Hb) 12.6 g/dl, calcium 9.6 mg/dl, alkaline phosphates 80 U/l (normal range (N): 36–92 U/l), magnesium 1.7 mg/dl, serum iron 50 µg/dl (N: 6–160), iron binding capacity 430 µg/dl (N: 250–460) and glucose 102 mg/dl.

Over the next few years, she continued to develop intermittent arthritis which involved both knees, both wrists and both metatarsophalangeal joints. Two of the episodes occurred while the patient was pregnant. On several occasions the synovial fluid was aspirated and consistently it revealed intracellular calcium pyrophosphate crystals.

At the age of 32 years, the investigations were repeated and revealed a calcium of 10.1 mg/dl, phosphorus 3.2 mg/dl, iron 42 µg/dl, iron binding capacity 450 µg/dl, ferritin 6 µg/ml, Hb 10.6 g/dl, cholesterol 202 mg/dl, triglycerides 175 mg/dl, magnesium 1.9 µg/dl and glucose 99 mg/dl. Alkaline phosphates, serum glutamic oxaloacetic and pyruvic transaminases (SGOT, SGPT) and thyroid-stimulating hormone (TSH) were within normal limits. A radiograph of the pelvis showed chondrocalcinosis of the pubis symphysis and both hip joints (Fig. 3).

Treatment with colchicine 1 mg daily resulted in a reduction of the frequency of the attacks, but the patient was not compliant in taking the drugs because of mild diarrhoea. The acute attacks were treated either by local steroid injection or by non-steroidal anti-inflammatory drugs with a good response.

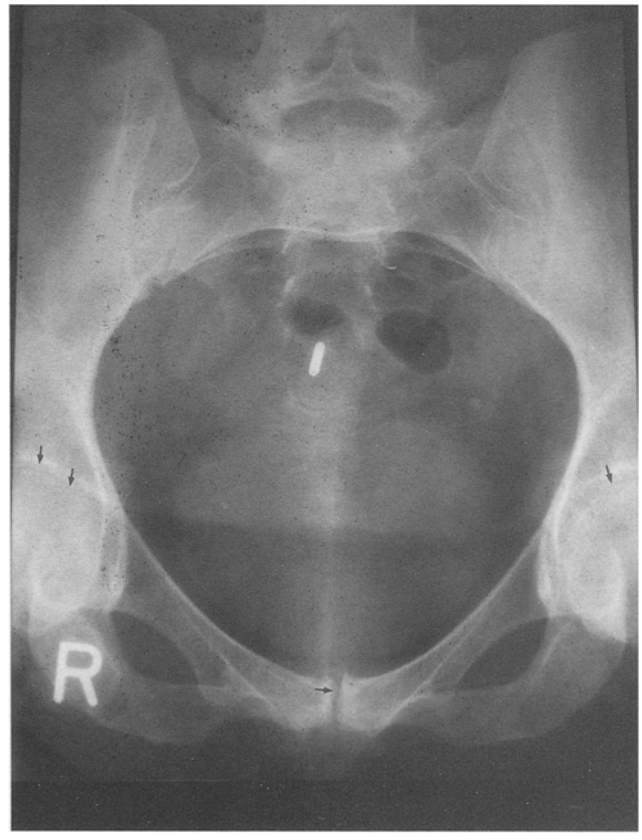

Fig. 3. Radiograph of the pelvis showing chondrocalcinosis of the pubis symphysis and both hip joints.

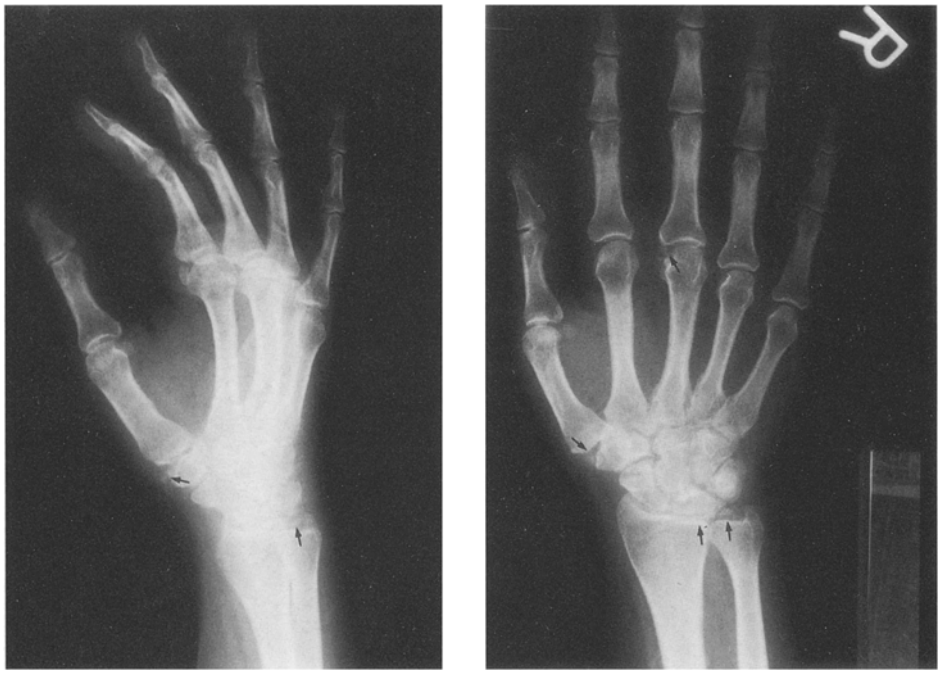

Fig. 4. Recent radiographs of both hands showing denser chondrocalcinosis of the triangular fibrocartilage of the wrist, third metacarphophalangeal and first carpometacarpal joints (arrows) and a narrowed radiocarpal joint space (compared with radiograph in Fig. 2, which was done at 26 years of age).

An examination of the two elder brothers of the patient was normal and a radiograph of their knees revealed no chondrocalcinosis.

Investigations were repeated at the age of 35 years and revealed normal levels for Hb, calcium, phosphorus, cholesterol, triglycerides, magnesium, iron binding capacity, alkaline phosphates, glucose, BUN, creatinine, ferritin, T4 and TSH. A radiograph of the left foot showed chondrocalcinosis of the first metatarsophalangeal joints and of the tarsal joints.

In between the attacks the patient has had no symptoms except in a recent attack in her right wrist, which lasted for almost 6 weeks with residual pain and mild limitation of the range of movement of the joint. A radiograph of the wrists showed denser and wider chondrocalcinosis (Fig. 4) compared with a previous radiograph 9 years earlier (Fig. 2). The radiocarpal joint was narrowed, indicating the development of osteoarthritic changes in the wrist.

Discussion

There is a striking association of chondrocalcinosis with ageing [1]. The prevalence of radiographic chondrocalcinosis is rare under the age of 50 years, but rises from 10–13% in those aged 65–75 years to 30–60% in those over 83 years of age [1]. Familial chondrocalcinosis has been reported in several countries and is characterised by either an early onset (third to fourth decade) with variable severity of arthropathy or a late onset (sixth to seventh decade) with oligoarticular chondrocalcinosis [2].

The secondary form of chondrocalcinosis and pyrophosphate arthropathy may be associated with a younger age of presentation and a tendency to florid polyarticular chondrocalcinosis [2]. Though numerous metabolic associations have been suggested [3], many reflect a chance occurrence of common age-related conditions [3]. The strongest evidence of an association is related to hyperparathyroidism and haemochromatosis [4]. In a few reported cases, premature chondrocalcinosis is associated with hypophosphatasia, hypomagnesaemia and Wilson's disease [5].

Our patient presented at the age of 24 years with recurrent knee arthritis and at 26 years of age she was found to have chondrocalcinosis. It is difficult to speculate how long the patient had had chondrocalcinosis before she became symptomatic at 24 years of age. Investigations over a 12-year period did not reveal any abnormalities in the tests mentioned above. Furthermore, there was a negative family history and neither of the two members of the family who were examined had any chondrocalcinosis of the knees. Furthermore, to our knowledge, no familial chondrocalcinosis has been reported in the Arab countries. We followed-up the patient for almost 12 years, which is probably long enough to uncover any metabolic diseases or familial predisposition.

We believe that although rare but idiopathic, CPPD disease may occur at a young age.

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