CHONDROCALCINOSIS ARTICLARIS (Pseudogout) A Study of 72 Patients

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Chondrocalcinosis articularis was first described as a clinical disease entity about 20 years ago but the existence of this relatively common disease may not be that well known to general physicians and specialists outside the field of rheumatology. Like gout it is a crystal induced disease. In clinical practice in this country, one diagnoses one new case of symptomatic chondrocalcinosis for every two cases of gout. Gout is caused by deposition of monosodium urate crystals, primarily in and around the joints, while chondrocalcinosis is due to deposition of calcium pyrophosphate dihydrate (CPPD) crystals in articular cartilage.

The disease is also known as "pseudogout" because it often clinically simulates gout. It is also called "CPPD crystal deposition disease," and "pyrophosphate arthropathy."

The clinical hallmark of the disorder and the finding that most readily suggests the diagnosis is the calcification of articular cartilage and menisci, visible on x-rays of the involved joints.

There can be following consequences of CPPD crystal deposition in articular cartilage:

a) The first possibility is that the person may remain asymptomatic. Approximately 5% of elderly people have knee joint CPPD crystal deposition. How many of them have symptoms? There is no clear answer to this question as yet, but it is likely that many, or even most, individuals with chondrocalcinosis remain asymptomatic.

b) The second possibility is that the patient may get an acute attack of arthritis mimicking gout and is called "pseudogout."

c) The third possibility is that the patient may get chrome arthritis with or without superadded acute attacks of pseudogout.

Chondrocalcinosis is a great mimic, often superficially resembling gout, osteoarthritis, rheumatoid arthritis, traumatic arthritis and neuropathic joints.

In the present paper I would like to report the results of a study of 72 symptomatic patients with chondrocalcinosis articularis collected over a period of seven years at Cleveland Metropolitan General Hospital. The disease was almost twice as frequent in females as in males. The mean age at the diagnosis was 67 years, the youngest patient was 45 years old and the oldest was 84 years old.

Two patients had no x-ray evidence of chondrocalcinosis but CPPD crystals were identified in their synovial fluid samples. The remaining seventy patients had x-ray evidence of chondrocalcinosis. Synovial fluid was studied in 39 of these patients and CPPD crystals were identified in 36 of them.

Clinical features: Knee was the most frequently involved joint. Other joints that were frequently involved were wrists, hips, shoulders, elbows, 2nd and 3rd metacarpophalangeal joints and ankles.

Acute arthritis (i.e. "pseudogout") usually involving one joint, was seen in 43 patients (60%). Knees and wrists were the most frequently involved joints. Twenty of these 43 patients had underlying chronic arthritis resembling osteoarthritis.

Twenty-six patients presented with chronic arthritis resembling osteoarthritis without superadded acute attacks of "pseudogout." In these patient's knees, wrists, hips, ankles, shoulders, elbows and metacarpophalangeal joints were commonly involved.

Three patients had chronic arthritis which mimicked rheumatoid arthritis.

Synovial fluid analysis yielded CPPD crystals in 38 of 41 patients. Mean white cell count of the

synovial fluid was 20,720/cmm, mostly polymorphonuclear cells. The cell count ranged from as low as 200 to as high as 96,800 cells/cmm. Synovial fluid mucin clot was fair to good, protein and glucose contents were within normal limits and cultures for microorganisms were sterile.

On radiological examination, joint chondrocalcinosis was present in 70 of 72 patients and was most commonly seen in knees. The other joints commonly showing chondrocalcinosis were wrists, pubic symphysis, hips, elbows, shoulder, metacarpophalangeal joints, ankles and rarely even intervertebral discs.

Radiological changes in patients with chondrocalcinosis resembled mostly those seen in patients with primary osteoarthritis (unassociated with any underlying associated disease), but there are certain differenting points:

1. Involvement of joints (e.g. wrists, shoulders and metacarpophalangeal joints) which are rarely affected by OA.

2. More prominent and more numerous subchondral cyst formation.

3. Joint degeneration more severe and more progressive, with subchondral bony collapse and fragmentation.

4. Isolated narrowing of patello-femoral joint space in knees and radiocarpal joint space in wrists more common.

Associated diseases: Three patients had associated hyperparathyroidism and one had hemochromatosis. Although one finds these associated diseases in only a minority of patients with chondrocalcinosis, it is important to exclude them in every patient. Chondrocalcinosis can be the first clinical manifestation of these associated diseases, as was seen in this patient with hemochromatosis who had no other clinical manifestation of hemochromatosis, e.g. hepatic dysfunction, diabetes or bronzed skin.

Three patients had associated gout. Both the urate and the CPPD crystals were identified in synovial fluids of these patients. Two patients had associated reheumatoid arthritis. Some of the knee joint symptoms in these two patients were due to CPPD crystal deposition because such crystals were identified in the synovial fluid aspirated from these swollen knees.

Treatment: Acute or subacute attacks of pseudogout are readily recognized by identification of CPPD crystals in synovial fluid leukocytes by compensated polarized light microscopy. Such attacks usually respond to aspiration of the swollen joints. Sometimes intra-articular injection of hydrocortisone may be needed to treat most severe attacks. When needle aspiration of the swollen joint is not feasible or when many joints are involved, short course of treatment with aspirin or preferably other non-steroidal anti-inflammatory drugs like indomethacin and butazolidine is quite effective. Effect of colchicine in pseudogout is not as predictable as in gout. For chronic arthritis, long term treatment with nonsteroidal anti-inflammatory drugs is needed. Occasionally, arthroplasty is needed for severely damaged joints.