REVIEW ARTICLE

Clinical and roentgenographic aspects of pseudogout: a study of 50 cases and a review

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Pseudogout, defined as recurrent acute arthritis due to intrasynovial deposition of calcium pyrophosphate dihydrate crystals, is a relatively common arthritic disorder of the elderly. The clinical and roentgenographic aspects of 50 cases of pseudogout in hospitalized patients are reviewed in this paper. Oligoarticular and polyarticular episodes were observed in half of these patients. Antecedent problems included infection, trauma, surgery and vascular events. Consistent with previous reports, most patients had roentgenographic evidence of chondrocalcinosis. A third had asymptomatic capsular or periarticular calcific deposits or both, and a third had pyrophosphate arthropathy, a progressive, destructive, accelerated form of osteoarthritis. An attack of pseudogout may offer a clue to the presence of an unsuspected metabolic disease, such as primary hyperparathyroidism or idiopathic hemochromatosis.

La pseudo-goutte, définie comme étant une arthrite aiguë récidivante due au dépôt intrasynovial de cristaux de pyrophosphate de calcium dihydraté, est une affection arthritique relativement fréquente chez les gens âgés. On étudie ici les aspects cliniques et radiologiques de 50 cas de pseudo-goutte chez des patients hospitalisés. Des atteintes oligoarticulaires ou polyarticulaires ont été observées chez la moitié de ces patients. On a relevé des antécédents d'infection, de traumatisme, de chirurgie et de maladies vasculaires. En commun avec les rapports précédents, la plupart des patients montrait des signes radiologiques de chondrocalcinose. Un tiers avaient des dépôts capsulaires ou périarticulaires de calcium mais étaient asymptomatiques, et un tiers souffraient d'arthropathie à pyrophosphate, une forme d'arthrose destructrice et d'évolution accélérée. Une crise de pseudo-goutte peut être l'indice d'une maladie métabolique insoupçonnée telle que l'hyperparathyroïdie primaire ou l'hémochromatose idiopathique.

Calcium pyrophosphate dihydrate crystal deposition disease is a relatively common arthritic disorder that occurs most often in elderly patients. It may manifest clinically as attacks of acute arthritis associated with the intra-articular deposition of calcium pyrophosphate dihydrate crystals (pseudogout syndrome).[1,2] Roentgenographic features include articular chondrocalcinosis (calcification of hyaline or fibrous articular cartilage or both),[1,3-11] capsular or periarticular calcific deposits or both,[7,10-14] and pyrophosphate arthropathy, a progressive, destructive, accelerated form of osteoarthritis with distinctive roentgenographic findings.[7,9-11,15-26] Although most cases are idiopathic, there are rare familial forms,[27-29] and the condition has been observed in coincidence with other metabolic disorders.[30] This report describes 50 hospitalized patients with acute pseudogout seen in the 7-year period 1972 through 1978.

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Methods

All patients included in the study had acute pseudogout, as defined by the demonstration of intra-articular rhomboid, rod-shaped or needle-shaped crystals of calcium pyrophosphate dihydrate.[31] The criteria for identifying the crystals were quite strict, requiring that they show both a triclinic form and a weakly positive birefringence when studied by compensated polarized light microscopy. Forty-seven patients met the diagnostic criteria for definite pseudogout.[32] The other three had no calcinosis and fell into the "probable" category. The clinical data obtained from all 50 patients included their age and sex, any history of previous attacks, and information on the pattern of their articular symptoms, factors that precipitated these symptoms, associated metabolic diseases and the extent of clinical osteoarthritis. Laboratory investigations included: complete blood count; measurement of the erythrocyte sedimentation rate (by the Westergren method); determination of the serum urea nitrogen, calcium, phosphorus, alkaline phosphatase, albumin, uric acid

and iron concentrations and the serum iron-binding capacity; tests for rheumatoid factor (by latex fixation) and fluorescent antinuclear antibody; and synovial fluid analysis. The synovial fluid leukocyte count was determined using saline as a diluent. An "arthritis survey", including roentgenograms of hands, wrists, elbows, shoulders, hips, pelvis, knees, feet and spine, was obtained in 40 patients. The remaining 10 patients had limited roentgenographic studies. The knee roentgenograms were graded for the degree of osteoarthritis by the criteria of the Empire Rheumatism Council.[33]

Results

Clinical findings (Table I and Fig. 1)

Episodes of pseudogout involved a single joint in 25 patients, two joints in 16 and three or more joints in 9. The knee and wrist joints were the most commonly affected. The arthritis was characterized by a sudden onset of pain and swelling, associated with tenderness and variable degrees of warmth, redness and effusion. It was the first attack of pseudogout for 20 patients; the remaining 30 had had one or more previous attacks. In 35 patients the attacks occurred during the course of another illness for which the patient was already hospitalized. Antecedent events included an infection in 12 patients, acute myocardial infarction in 7, congestive cardiac failure in 4, nonoperative trauma in 4, a surgical procedure in 3, acute alcoholic intoxication in 2, and single cases of cerebral infarction, subarachnoid hemorrhage and pernicious anemia. The remaining 15 patients were in hospital because of the acute arthritis, which was often polyarticular. In three of these patients pseudogout was misdiagnosed as pyogenic arthritis because of a high synovial leukocyte count.

The pseudogout episodes responded well to joint aspiration and therapy with phenylbutazone, indomethacin or corticosteroids injected into the joint. Colchicine was tried in two patients, but the response was poor. With treatment the attacks lasted from 2 to 15 (mean 5.3) days.

Laboratory findings

A synovial fluid leukocyte count was available for 30 patients (Table II). The number of leukocytes varied with the duration of the patient's symptoms before joint aspiration: samples of fluid examined

Table I—Age and sex of 50 patients with pseudogout

Age (yr)	No. of patients	Sex	
		Female	Male
50–59	2	1	1
60–69	9	5	4
70–79	20	13	7
80–89	18	12	6
90+	1	–	1

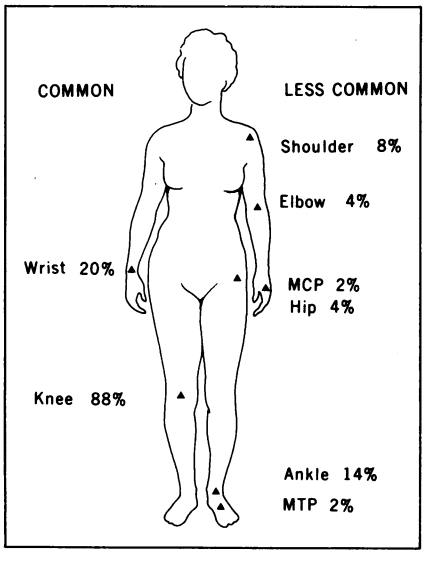

FIG. 1—Pattern of joint involvement in 50 patients with pseudogout. MCP = metacarpophalangeal, MTP = metatarsophalangeal.

Table II—Number of leukocytes in specimens of synovial fluid from 30 patients with pseudogout

No. of leukocytes/mm³	No. of samples
< 200	1
200–2000	5
2001–50 000	21
> 50 000	3

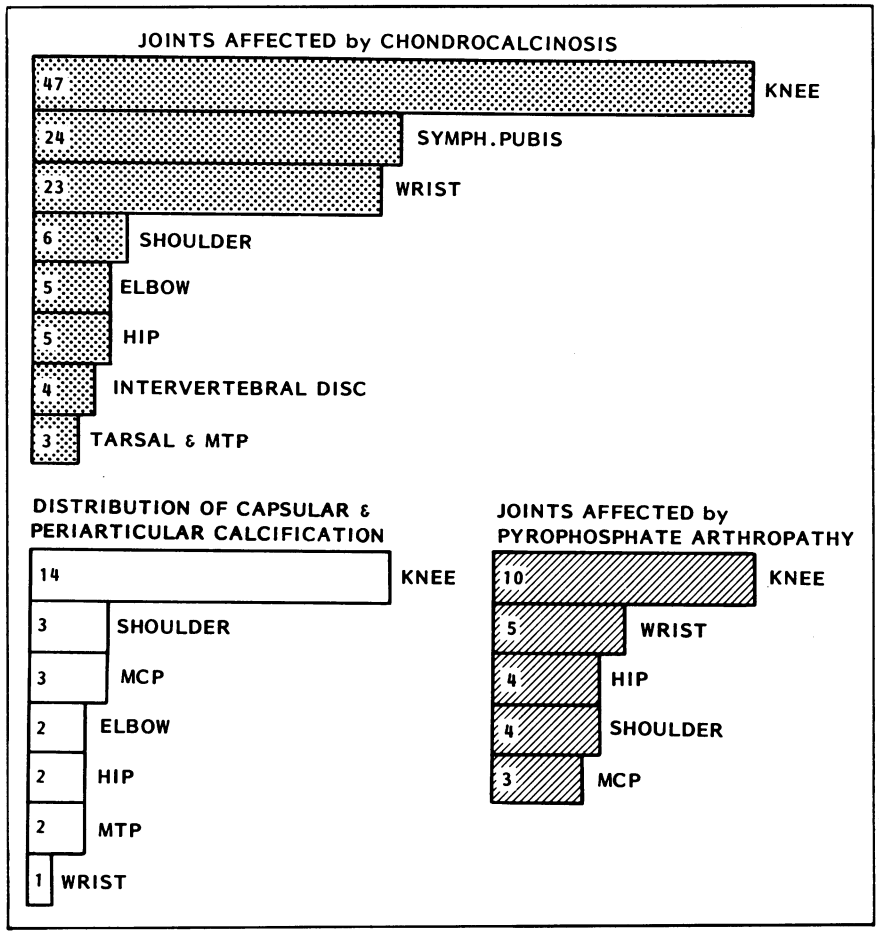

FIG. 2—Specific radiologic findings in 50 patients with pseudogout.

within 2 to 4 days of the onset of symptoms yielded higher counts than those studied a week or more later. Between 38% and 100% (mean 88.6%) of the leukocytes were neutrophils. Calcium pyrophosphate dihydrate crystals were seen both intra- and extracellularly. During acute episodes the erythrocyte sedimentation rate rose moderately in 44 patients. The results of rheumatoid factor tests were negative in 40 patients, weakly positive (titre less than 1:320) in 5 and unavailable in 5. Nine patients had diabetes mellitus, four had asymptomatic hyperuricemia, three had primary hyperparathyroidism and one had idiopathic hemochromatosis. In two of the three patients with primary hyperparathyroidism acute pseudogout was the first clue to the underlying metabolic disorder. The third patient had an attack of pseudogout 4 days after excision of a parathyroid adenoma.

Roentgenographic findings

Chondrocalcinosis: The roentgenograms of 47 patients showed bilateral, fairly symmetric, fine-to-dense calcification of articular fibrocartilage, most frequently in the menisci of the knees (Figs. 2 and 3). Calcification of hyaline articular cartilage was observed less frequently. It appeared as either a continuous or a disrupted band of density following the contour of the underlying bone (Fig. 3), most frequently in the knee, elbow, shoulder and hip. In three patients who had acute pseudogout with intra-articular calcium pyrophosphate dihydrate crystals, extensive roentgenographic skeletal surveys failed to reveal chondrocalcinosis.

Capsular and periarticular calcification: This type of calcification was observed in 17 patients, including 1 with primary hyperparathyroidism (Figs. 2, 4 and 5). The knee was the most commonly affected joint. The deposits were frequently capsular and showed either a fine linear or a dense globular pattern. Synovial, tendinous, ligamentous

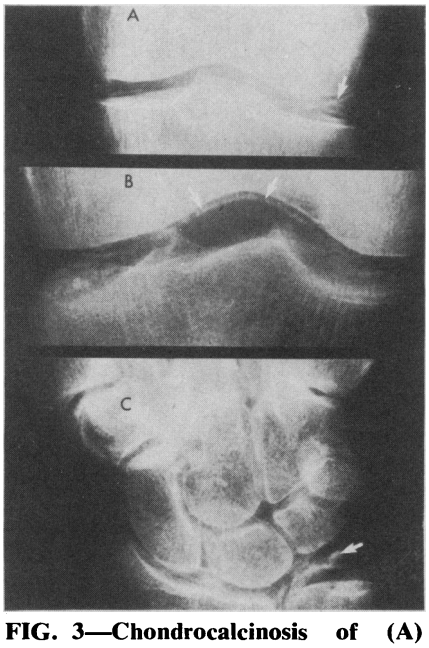

FIG. 3—Chondrocalcinosis of (A) meniscus of knee, (B) hyaline articular cartilage of knee and (C) triangular fibrocartilage of wrist.

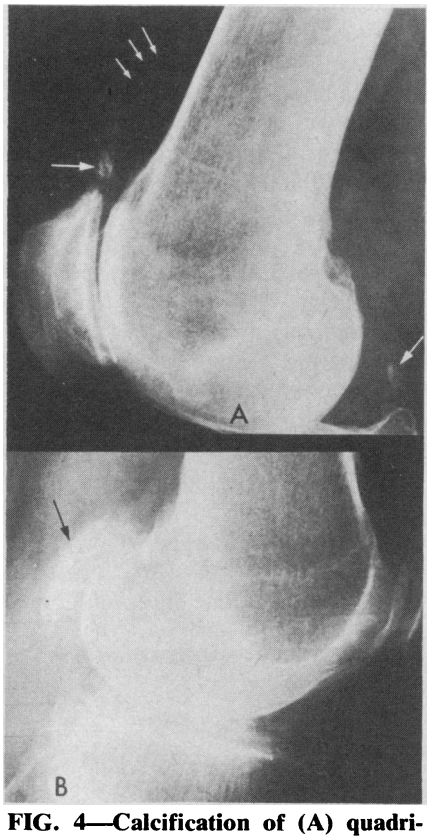

FIG. 4—Calcification of (A) quadriceps tendon, suprapatellar bursa and posterior capsule (arrows) and (B) posterior capsule (arrow) of knee.

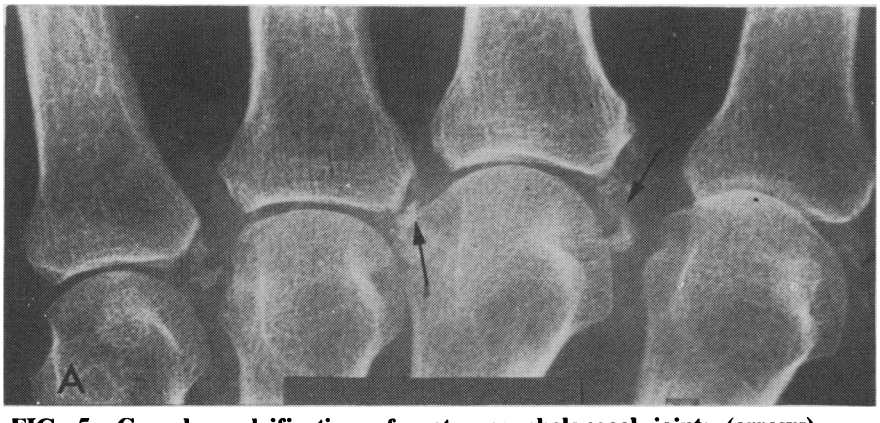

FIG. 5—Capsular calcification of metacarpophalangeal joints (arrows).

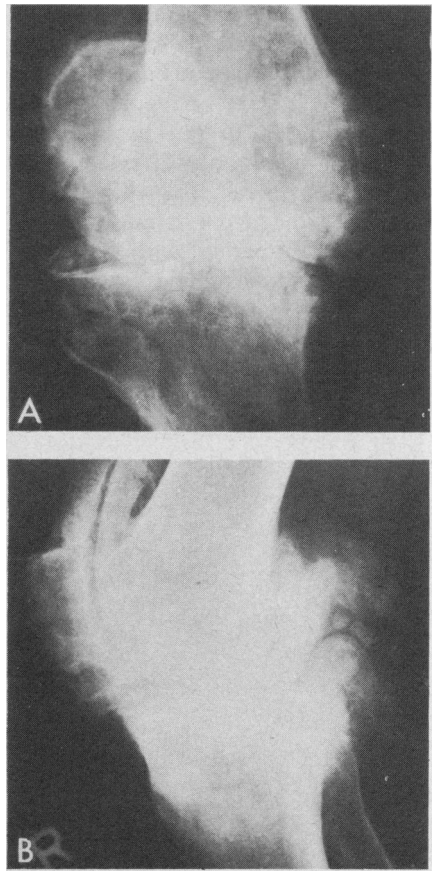

FIG. 6—Posteroanterior (A) and lateral views (B) of knee, showing pyrophosphate arthropathy, with loss of articular cartilage, subchondral sclerosis and bone collapse, osteophytes, subluxation and extensive synovial calcification simulating synovial chondromatosis.

and bursal calcifications were less common. One patient had extensive synovial calcification, with diffuse amorphous intra-articular densities of one knee (Fig. 6). Serial roentgenography in 5 of the 17 patients showed that the calcific deposits remained unchanged over 2 years.

Osteoarthritis and pyrophosphate arthropathy: The roentgenograms of 45 of the 50 patients showed bilateral, fairly symmetric osteoarthritis of the knee that ranged from grade 1 or 2 (28 patients) to grade 3 or 4 (17 patients). Of these patients 23 had Heberden's nodes and generalized osteoarthritis of the "primary" type.[34] In addition, 16 of these 45 patients (10 women and 6 men, including the patient with idiopathic hemochromatosis) had a destructive form of osteoarthritis that suggested pyrophosphate arthropathy.[11] Their roentgenograms were characterized by large subchondral bony cysts, collapse and fragmentation of subchondral bone, intra-articular osseous bodies and, frequently, large irregular osteophytes (Figs. 6 to 8). The knee was the most commonly affected joint, followed by wrist, then hip, shoulder and metacarpophalangeal joints (Fig. 2). The patient with idiopathic hemochromatosis had pyrophosphate arthropathy of the metacarpophalangeal and left hip joints. None of the remaining 15 patients had an underlying metabolic disease, neurologic abnormality or inflammatory arthritis, and none had received repeated intra-articular corticosteroid injections.

Discussion

The association of arthritis, chondrocalcinosis and the intra-articular deposition of calcium pyrophosphate dihydrate crystals is now well established. The spectrum of articular manifestations ranges from acute attacks of inflammatory arthritis ("pseudogout" or type "A" arthritis according to McCarty's classification[32]) to calcification of fibrous or hyaline cartilage or both (chondrocalcinosis), capsular and periarticular calcifications, and a progressive, destructive type of osteoarthritis designated pyrophosphate arthropathy[11] (corresponding to McCarty's type "C" pseudo-osteoarthritis and type "F" pseudo-neuropathic joints).

This review describes the joint abnormalities in 50 hospitalized patients presenting with acute pseudogout. The disease commonly affects elderly patients;[1,4-8,11,16,24,35,36] 39 of our patients were aged 70 years or older, and only 2 were less than 60 (Table I). The preponderance of women in this series may be due to the higher proportion of female patients in the hospital population rather than to a higher incidence of pseudogout in women. Others[1,37,38] have confirmed our observation that pseudogout episodes are often triggered by surgical and nonsurgical trauma, acute infections, and cardiac and cerebrovascular events. In half of our patients more than one joint was affected, and attacks were often associated with fever, tachycardia and, occasionally, leukocytosis. Polyarticular attacks were described in other studies[1,6,8,24,37] and seem to occur with greater frequency in pseudogout than in gout. Our experience indicates that phenylbutazone, indomethacin and the intra-articular administration of corticosteroids are equally effective in controlling attacks of acute pseudogout. No prophylactic treatment has been proven effective, although temporary improvement may follow reduction of the number of crystals by joint lavage.[39]

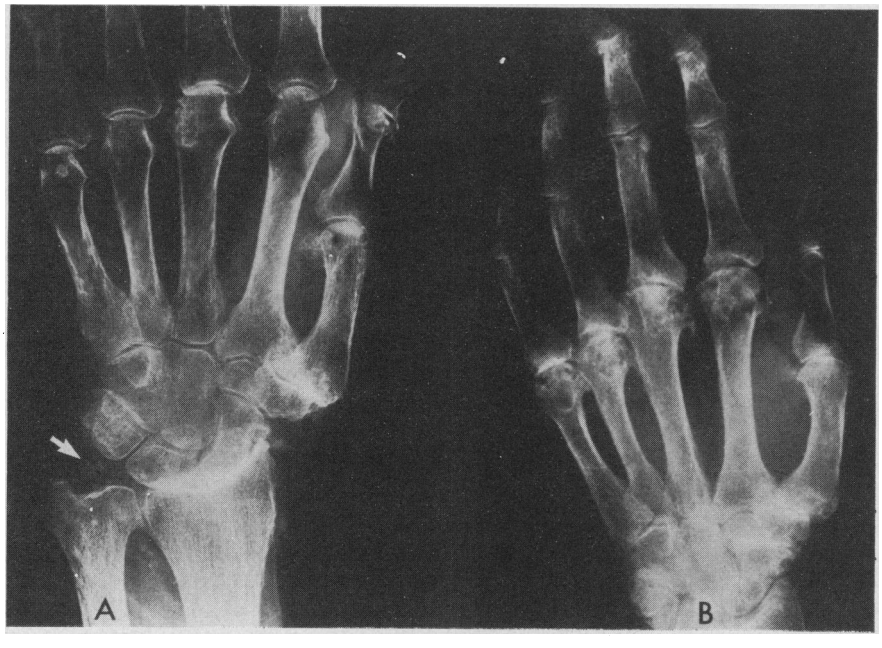

FIG. 7—Pyrophosphate arthropathy of (A) radiocarpal joint, with narrowing of joint space, calcification of triangular fibrocartilage and subchondral sclerosis with cysts, and of (B) metacarpophalangeal joints, with narrowing of interosseous space, sclerosis, subchondral cysts and hook-like osteophytes.

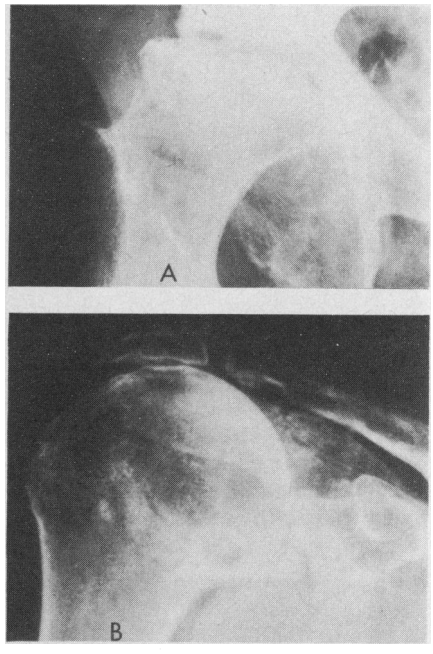

FIG. 8—Pyrophosphate arthropathy of (A) hip, with joint space narrowing, sclerosis, subchondral lucencies and gross destruction of the femoral head mimicking avascular necrosis, and of (B) shoulder, with calcifications in supraspinatus tendon, narrowing of glenohumeral joint space, sclerosis, subchondral lucencies, upward migration of humerus and pseudoarticulation between humeral head and acromion, indicating rotator cuff rupture.

An association between pseudogout attacks and both primary hyperparathyroidism and idiopathic hemochromatosis has been well documented.[30,32,40-42] Among our 50 patients we found three cases of primary hyperparathyroidism (a frequency of 6%, which corresponds with that reported in other series[4,30,32]) and one case of idiopathic hemochromatosis. It is therefore important to screen patients with pseudogout for unsuspected metabolic disorders.

In accordance with other published studies,[1,3-11] articular chondrocalcinosis was the most common and conspicuous roentgenographic feature. It is noteworthy that three of our patients had no identifiable local or distant chondrocalcinosis. Such cases are rare; in some of these patients roentgenographic calcification developed subsequently.[5,7,8,18,22,43]

A third of our patients had capsular calcification and, less commonly, synovial, tendinous, ligamentous and bursal calcifications. Extensive synovial calcification simulating synovial chondromatosis was noted in one patient (Fig. 6) and in two previous cases.[44] The deposits are often bilateral and asymptomatic,[7,10-15,43] though in rare cases they may cause local inflammation with pain and swelling.[7,13] Limited pathological studies of these calcific deposits have shown that they contain masses of calcium pyrophosphate dihydrate crystals.[13,45] In a few cases similar deposits have been found in remote extra-articular sites.[41,46] Although pathological and serial roentgenographic studies over extended periods were not done in this study it is our impression, and that of others,[43] that these extra-articular deposits tend to appear later in the course of the disease, particularly in patients with advanced pyrophosphate arthropathy.[7] The periarticular calcific deposits of hydroxyapatite-associated calcific periarthritis[47] can be distinguished from those of calcium pyrophosphate dihydrate crystal deposition disease by their usually transient nature and homogeneous pattern, and by the absence of articular chondrocalcinosis.

In this study pseudogout was strongly associated with degenerative joint disease, especially of the knee, a frequent finding in almost all published reports.[4,7,8,11,16,18,24,30,36] As well, a third of our patients had an accelerated, destructive form of osteoarthritis, designated pyrophosphate arthropathy.[11] The frequency of this type of arthritis among patients with pseudogout in other studies has varied from 13.5% to 77.5%.[11,25,26] The arthropathy has an unusual distribution, with a predilection for knee, hip, radiocarpal, metacarpophalangeal and glenohumeral joints and, less commonly, elbow, talocalcaneal and ankle joints.[7,11,25,48] Other distinctive features include large subchondral cysts; collapse and fragmentation of subchondral bone with formation of osseous bodies; large, irregular and often exuberant osteophytes; and, at a later stage, gross destruction of the joint. Many of these features have now been included in the revised diagnostic criteria for calcium pyrophosphate dihydrate crystal deposition disease.[32] In our patients pyrophosphate arthropathy of the knee was characterized by predominant involvement of the patellofemoral compartment; collapse of the tibial plateau and occasionally of the femoral condyle; osseous bodies; chondrocalcinosis; instability and deformity; and, at a later stage, disorganization of the joint (Fig. 6). Wrist arthropathy was associated with narrowing of the radiocarpal joint space, chondrocalcinosis and subchondral bony sclerosis with large cysts (Fig. 7). Metacarpophalangeal joint abnormalities consisted of narrowing of the interosseous space, sclerosis, subchondral radiolucencies and fragmentation of the metacarpal heads with large hook-like osteophytes that produced a squared-off appearance (Fig. 7).[10] Two of the four patients with hip arthropathy had partial dissolution of the femoral head mimicking avascular necrosis (Fig. 8A). Shoulder arthropathy was characterized by narrowing of the glenohumeral joint space, large subchondral cysts, variable osteophytosis and upward migration of the humeral head (Fig. 8B).

Conclusion

Pseudogout is a relatively common arthritic disorder in the elderly. It rarely occurs with roentgenographically identifiable articular chondrocalcinosis. Capsular and periarticular calcifications are fairly frequent and approximately a third of patients may have pyrophosphate arthropathy.

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This list is an acknowledgement of books received. It does not preclude review at a later date.

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CARDIOVASCULAR PATHOPHYSIOLOGY. Tjeerd Van Der Werf. 296 pp. Illust. Oxford University Press, Toronto, 1980. $20.50, paperbound. ISBN 0-19-261229-8

CLINICAL PHARMACOKINETICS. Concepts and Applications. Malcolm Rowland and Thomas N. Tozer. 331 pp. Illust. Lea & Febiger, Philadelphia, 1980. $35.50 (Can.). ISBN 0-8121-0681-4

DIAGNOSIS AND MANAGEMENT OF SEXUALLY TRANSMITTED DISEASES. A Manual for Health Workers. Published by authority of the Minister of National Health and Welfare, Health Programs Branch. 72 pp. Illust. Health and Welfare Canada, Ottawa, 1978. $2.25, paperbound. ISBN 0-660-01757-1; catalogue number H31-16-1978E

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