Osteoarthritis and articular chondrocalcinosis in the elderly

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SUMMARY One hundred consecutive admissions to an acute geriatric unit were examined for clinical and radiographic evidence of osteoarthritis (OA) and articular chondrocalcinosis (ACC). Thirty-four patients had ACC. This was age related, the prevalence rising from 15% in patients aged 65–74 years to 44% in patients over 84 years. The commonly involved joints were the knee (25%), pubic symphysis (15%), and wrist (9%). No other aetiological factors predisposing to ACC were found. Of the 25 patients with ACC in the knee 7 had no symptoms or signs and no radiographic evidence of OA at that site. However, the combination of ACC and radiographic OA was characterised by an increase in clinical joint disease. Features of inflammation (joint swelling and joint line tenderness) involving the knee, wrist, and elbow were particularly common in ACC. It is concluded that ACC is common in the elderly and is associated with an increased incidence of joint disease.

The relationship between intra-articular deposition of calcium pyrophosphate dihydrate crystals and arthritis was first described in 1962 by McCarty *et al.*¹ The original description was of a monoarticular acute arthritis, and because of similarities with urate arthropathy the condition was termed pseudogout. Recently it has been suggested that pyrophosphate deposition can be associated with a number of differing patterns of joint disease, on occasions mimicking both osteoarthritis and rheumatoid arthritis.²

The radiographic appearance of linear calcification of joint cartilage as described by Zitnan and Sitaj³ is one sign of pyrophosphate crystal deposition. Such calcification (chondrocalcinosis) is common in the elderly, but its significance is unknown. It is often found in asymptomatic joints, suggesting that articular chondrocalcinosis (ACC) can be a function of aging and not a marker of joint disease.

The present study was designed to assess the prevalence of radiographic ACC in the elderly and to relate such calcification to clinical and radiographic evidence of joint disease.

Patients and methods

The study was approved by the local ethical com-

mittee. One hundred and twenty consecutive admissions to an acute geriatric unit were studied, the age, sex, and reason for admission noted, and verbal consent obtained before inclusion in the study.

Clinical details. A general examination of the musculoskeletal system was followed by a detailed examination of the hands, wrists, and knees. The findings were tabulated by a single observer and a simple grading system was employed: joint pain, swelling, and crepitus were graded 0–none, 1–mild, 2–moderate, and 3–severe. Joint warmth and effusion were scored as present or absent. Joint deformity at the knee was defined as valgus or varus deformity of more than 10° or a fixed deformity of more than 5°; a restriction of arc movement at the wrist to below 60° was also considered abnormal.

Radiographic details. The radiographs were taken on either a GEC D8/150 with Dynamax tube (focal spot size 1.0 mm) or a GEC Linotome with a Dynamax tube (focal spot sizes 0.6 mm or 1.3 mm). Anteroposterior and lateral views of both knees were taken on Ilford Rapid R film, average exposure 10 mAs at 57–60 kV. An anteroposterior view of the pelvis was taken on Kodak X-O-Mat RP-X RPI with an average exposure of 36 mAs at 72 kV and an AP view of both hands and wrists on Ilfex 90 film, average exposure 30 mAs at 60 kV.

The radiographs were assessed independently by the radiologist (G.E.), who had no knowledge of the

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Fig. 1 Prevalence of articular chondrocalcinosis.

clinical data, and by the clinicians. Agreement on grading, after the initial analysis, was obtained in 85% of the cases. The remaining 15% were then subjected to a combined assessment to obtain a final agreed grade. The presence of linear shadows of calcific density in joint cartilage (ACC) was recorded as unilateral, bilateral, linear, stippled, dense, or hazy, and graded 1–4 (1–visible with magnifying glass, 2–faint but visible with naked eye, 3–moderate, 4–dense). Radiographic evidence of osteoarthritis was graded mild, moderate, or severe in accordance with the criteria laid down by Kellgren and Lawrence.⁴ Other investigations included full blood count, blood calcium, phosphate, alkaline phosphatase, serum creatinine, and thyroid function tests.

Results

One hundred and twenty consecutive admissions were studied over a 10-week period. Complete data were obtained on 100 patients. There were 3 general categories of admission; 32% were referrals from other specialties for rehabilitation, mainly from strokes (14 patients) and limb fractures (13 patients); 29% were referred by their general practitioners because of the patients' failure to cope with living in the community (19 for psychiatric reasons and 17 with chronic 'physical' disability). The remainder (49%) were admitted for investigation and treatment of 'general' medical conditions such as heart failure (8 patients), anaemia (7 patients), and weight loss (7 patients). Ages ranged from 65 to 97 years with a mean of 79.4 years (SD 6.67). There were 31 males and 69 females (male:female ratio of 1:2.2).

Thirty-four patients had ACC. There was a clear

relationship to increase in age; the prevalence rose from 15% (3 out of 20) in patients aged between 65 and 74 years, to 36% (20 out of 55) between 75 and 84 years, and to 44% (11 out of 25) in patients above 84 years (Fig. 1).

Most of the subjects with ACC (25 out of 34) had involvement of the knee joint. Fibrocartilage was more commonly affected than hyaline; the lateral fibrocartilage being most frequently involved (18 out of 25).

Fifteen patients had ACC at the pubic symphysis. Ten had involvement at the wrist, all had triangular ligament ACC, half had bilateral involvement. Involvement at more than one site was common: 15 subjects had ACC at the knee and pubic symphysis, while 9 out of the 10 subjects with ACC at the wrist also had knee involvement. Six subjects had involvement of knee, wrist, and pubic symphysis (Table 1). The character of the chondrocalcinosis varied with the site of involvement. Articular hyaline cartilage calcification was generally linear and that of the menisci hazy or dense. A faint opacity in the hyaline cartilage at the medial half of the lateral femoral condyle was in some cases the only sign of calcification. Anteroposterior radiography of both knees gave the best single view for detecting the presence of ACC.

General metabolic disorders that have been implicated in pyrophosphate crystal deposition^{5,6} were not apparent, and aging alone appeared to be the main aetiological factor. The small number of patients who had diabetes mellitus (4) or thyroid disease (10) was

Table 1 Distribution of articular chondrocalcinosis

Site	Numbers	Unilateral	Bilateral
Hands			
IP joint	1	0	1
MCP joint	4	2	2
Intracarpal joint	3	1	2
Total	8	5	3
Wrists			
Triangular	10	4	6
Radiocarpal	6	3	3
Total	10	5	5
Knees			
Hyaline:			
Femur	14	6	8
Tibia	5	2	3
Fibrocartilage:			
Medial	14	4	10
Lateral	18	6	12
Total	25	9	16
Symphysis	15	—	—
Hips			
Hyaline	3	1	2
Glenoid	2	0	2
Total	5	1	4

Table 2 Articular chondrocalcinosis: relationship to clinical and laboratory data

	Chondro. group	No chondro. group
Total in each group	34	66
Mean age	81.4 (± 6.2)	79.0 (± 6.9)
Male to female ratio	7:27 (1:3.8)	21:25 (1:2.5)
Medical history of:		
Diabetes mellitus	2	2
Thyrototoxicosis	2	3
Myxoedema	2	3
Osteomalacia	1	4
Drug history of:		
Steroids	1	1
Anti-inflammatory drugs	9	19
Diuretics	14	24
Laboratory data: (Mean values and SD)		
Hgb	12.1 (± 1.8)	12.4 (± 2.0)
Blood viscosity	1.77 (± 0.17)	1.73 (± 0.4)
Blood:		
Calcium	2.30 (± 0.21)	2.31 (± 0.15)
Alkaline phosphatase	111.0 (± 32)	111.0 (± 41)
Phosphate	1.3 (± 0.20)	1.08 (± 0.24)
Creatinine	106.0 (± 46)	93.0 (± 38)
Thyroid function	117.0 (± 30)	124.0 (± 36)

not sufficient to allow comment on any possible relationship between these conditions and ACC.^{7,8} Laboratory measurements in patients with and without ACC are shown in Table 2; there were no significant differences.

Most subjects had some clinical evidence of joint disease. The hands were involved in 90% of subjects, with swelling and/or deformity of the carpometacarpal joint the commonest abnormality (68%). 62% had abnormal knee joints; 31% had valgus, varus, or flexion deformity, and 20% had joint tenderness. 37% had abnormal elbow joints, the majority with fixed flexion abnormalities. 29% had abnormalities at the wrist, mainly restriction of movement. Sixteen patients had pain and loss of movement at the shoulder.

The prevalence of clinical disease of the large joints was greater in patients with ACC than in those without (Table 3).

The relationship between the clinical assessment of joint disease and radiological grading of OA at the knee is shown in Table 4. OA changes were seen in 48% of all knees (62% of patients with ACC and 41% of those without). It was also noted that 6 out of 8 patients with ACC and mild OA had clinical abnormalities, compared with 4 out of 15 patients with mild OA and no ACC.

The nature of the relationship between joint disease and ACC was further illustrated in the 9 subjects

Table 3 Articular chondrocalcinosis and clinical joint disease

	Subjects with ACC (n=34)				Subjects without ACC (n=66)			
	Presence of joint disease				Presence of joint disease			
	Pain	Swelling	Loss of movement	No. with joint abnormality	Pain	Swelling	Loss of movement	No. with joint abnormality
Wrist:								
R	3	7	9	15 (57%)*	6	7	15	14 (25%)*
L	2	4	4		6	6	11	
Elbow:								
R	7	8	12	18 (53%)**	2	3	13	19 (36%)**
L	3	2	7		3	4	15	
Shoulder:								
R	5	3	6	8 (25%)	5	1	4	8 (12%)
L	4	4	5		2	2	2	
Knee:								
R	14	18	9	23 (67%)	16	27	22	39 (59%)
L	10	16	7		17	26	21	
Hip:								
R	2	Excluded	23	26 (83%)	6	Excluded	42	42 (75%)
L	2	Excluded	26		4	Excluded	40	

Joint abnormalities due to fracture excluded.

*p=0.006. **p=0.012.

Statistical method: The χ^2 test was used to evaluate the probability that subjects with ACC and joint disease were from the same population as those with joint disease and no ACC (difference is significant with p values ≤ 0.05).

Table 4 Clinical joint disease, radiological OA and ACC at the knee

		Joint tenderness (Grade 2-3)		Joint swelling (Grade 2-3)		Flex. deformity	
		With ACC	No ACC	With ACC	No ACC	With ACC	No ACC
Mild radiographic OA Number with ACC—8 without ACC—15	R	4 (50%)	2 (13%)	4 (50%)	3 (20%)	2 (25%)	2 (13%)
	L	2 (25%)	1 (7%)	2 (50%)	2 (13%)	4 (50%)	5 (33%)
Moderate and severe radiographic OA Number with ACC—4 without ACC—11	R	1 (25%)	3 (27%)	3 (75%)	5 (45%)	2 (50%)	7 (64%)
	L	1 (25%)	3 (27%)	2 (50%)	6 (55%)	2 (50%)	6 (55%)
No radiographic OA Number with ACC—13 without ACC—13	R	2 (15%)	4 (10%)	0	5 (12%)	1 (8%)	6 (15%)
	L	2 (15%)	2 (5%)	0	3 (8%)	3 (23%)	5 (13%)

Nine patients with ACC in joints other than the knee not included. One patient with seropositive rheumatoid arthritis not included.

who had unilateral ACC at the knee. Six had clinical and radiographic evidence of joint disease, 4 had bilateral disease with abnormalities most marked on the side with ACC, one patient had joint disease on the side opposite to the site of ACC, while the remaining patient had disease at the site of calcification with a normal joint the other side.

Discussion

Radiographically detectable intra-articular calcification was first described by Pearson and Davin in 1921.⁹ However, its relationship to arthritis remained unrecognised until the work of Zitnan and Sitaj in 1958.¹⁰ They described an arthritis associated with punctate and linear radiodensities in hyaline and fibrocartilage, and termed such abnormalities articular chondrocalcinosis.

In 1962 McCarty, Kohn, and Faires¹ described the pseudogout syndrome, that is, arthritis, articular chondrocalcinosis, and the presence of calcium pyrophosphate dihydrate crystals in synovial fluid. More recently it has been shown that calcium crystals other than pyrophosphates can cause ACC, but linear deposits in hyaline cartilage and meniscal calcification are usually due to deposits of calcium pyrophosphate dihydrate.¹¹ In view of the characteristic radiographic pattern seen in most of our subjects we can assume that their ACC was related to pyrophosphate, although for ethical reasons we were unable to obtain crystallographic data on the calcified tissues.

Information relating ACC to clinical and radiographic evidence of joint disease in the elderly is sparse.

Bocher *et al.*¹² in 1965 studied the radiographs of

450 patients. Most of these were knee radiographs from files of local hospitals and little clinical data were available. However, they did include 100 patients randomly selected from a local Jewish home for the elderly. Ten patients had ACC, and 8 of these had significant clinical and radiographic evidence of joint disease. Ellman and Levin in 1975¹³ studied 58 residents of a Jewish home for the elderly, average age 83, and they used Kodak type M film to achieve maximum sensitivity. The overall prevalence of ACC was 27.6%. Clinical examination was performed on all subjects and there appeared to be an increase in varus deformity at the knee, and in pain and deformity at the wrist in patients with ACC. Pritchard and Jessop in 1977,¹⁴ as part of their study on chondrocalcinosis in hyperparathyroid patients, looked at the knee x-rays of 100 elderly patients and found 11 with ACC. All patients had calcification in all 4 menisci, and there was little radiological evidence of joint disease; a clinical examination was not performed.

Ellman and colleagues have recently published 2 more articles on the prevalence of chondrocalcinosis in the elderly and its relationship to joint disease.^{15 16} They found a high incidence of radiological chondrocalcinosis, related to age, and more narrowing of the joint space on nonweight bearing films in those with ACC than those without. However, as in the present study, chondrocalcinosis without significant joint disease was also seen.

In the present study of 100 consecutive patients admitted to a geriatric unit 34 had articular chondrocalcinosis. This is a surprisingly high figure but is similar to the findings of Ellman and Levin. The prevalence of ACC increased in a linear fashion with aging. This not only confirms the importance of aging in the aetiology of ACC, but also stresses the importance of expressing such data in terms of decades

rather than in 'the elderly'. Radiographic techniques and materials adopted for use in this study were selected to achieve maximum sensitivity in the detection of calcium pyrophosphate deposition. Nevertheless, it is likely that radiography can detect only the more obvious ACC and that the 34% may be an underestimation of the true prevalence.

None of our subjects had hypercalcaemia, only 4 had diabetes mellitus, 3 had myxoedema, and 2 were thyrotoxic. There was no obvious relationship between ACC and diabetes mellitus or thyroid disease, and apart from aging no other aetiological factors emerged.

The relationship between ACC and joint disease in the elderly population studied was not simple. Overall there was an undoubted increase in clinical joint disease in patients with ACC, and yet some patients with florid ACC had normal joints, and conversely others with obvious clinical and radiological evidence of joint disease had no ACC.

There was also a lack of correlation between clinical and radiological grading of joint disease at the knee. This anomaly was apparent in 2 groups of subjects. The first was a group of 5 with ACC but no evidence of radiological OA; they had considerable (grade 1-11) joint tenderness, swelling, and flexion deformity. The second was a group of 8 subjects with mild OA and ACC. In this group joint tenderness and swelling and the number with flexion deformity was much greater than expected, either from the appearance of the knee radiographs or in comparison with patients who had mild OA but no ACC.

Patients with OA and ACC often presented with involvement of the elbow, shoulder, wrist, and knee; flexion deformity at the elbow seemed particularly characteristic.

The distribution of ACC and associated arthritis seen in our elderly patients was in many cases similar to that seen in chronic pyrophosphate arthropathy, though the progressive and severe joint destruction usually seen in the latter was not so apparent.

The part played by intra-articular crystal deposition in the aetiology of osteoarthritis^{17 18} is still unclear. The result of our study suggests that traditional 'chicken and egg' concept is an oversimplification. We would postulate that intra-articular crystal deposition is in most cases secondary to age-related changes in cartilage. Osteoarthritis can develop

independently, is related to aging, but is also dependent on other factors. However, in the presence of OA, intra-articular crystal deposition acts as an additional stimulus to the inflammatory response.

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