CALCIFICATION IN ARTICULAR CARTILAGE


From Queen Mary’s Hospital, Roehampton, London

That calcification in articular cartilage is uncommon is shown by the few references in the literature to this condition, and also by the fact that it is not described in modern text-books of radiology. This case is reported in order to put on record the association of this condition with a parathyroid tumour.

CASE REPORT

A man of fifty-seven was admitted to Queen Mary’s Hospital, Roehampton, in December 1957 complaining of general listlessness, effort dyspnoea and angina of effort of some fourteen months’ duration.

Previously he had had attacks of left-sided renal colic, which started in 1928 and which necessitated a left nephro-lithotomy in 1944 and a left nephrectomy in 1948. Thereafter he remained moderately well until the development of the recent symptoms, with the exception of an occasional small stone in the urine and a few episodes of urinary infection.

The patient had slight—but recently increasing—deafness; he had had nocturia twice nightly for many years but no polyuria or polydipsia. He had no history of joint pain, swelling or stiffness, nor of any bone pain.

The family history revealed that the father had died, aged sixty-three, of cancer, and that one aunt and one cousin had each had a kidney removed. There was no family history of gout.

Examination revealed a man with grey hair, a “biscuit coloured” appearance and pallor of the mucous membranes. He had a slight perceptive deafness. There was no calcification in the cornea and the fundi were normal. The tongue showed a slight degree of atrophic glossitis; no lumps were felt in the neck. The heart and lungs were normal and the blood pressure was 145/90 millimetres of mercury. The abdomen had no palpable masses and showed only a featureless scar in the left loin. The bones were not tender or deformed, nor was there any arthritis. The central nervous system showed no abnormality.

INVESTIGATIONS AND TREATMENT

Examination of the blood revealed fairly severe pernicious anaemia. The haemoglobin was 7.7 grammes (52 per cent) with a red blood cell count of 2,100,000 per cubic millimetre. The packed cell volume was 21 per cent and the mean cell haemoglobin was 37μμ grammes, with the mean cell volume 100 μμ and a mean cell haemoglobin content of 37 per cent. A fractional test meal showed a histamine-fast achlorhydria. The urine contained many pus cells and staphylococcus pyogenes was grown on culture. An intravenous pyelograph suggested a deformity of the upper calyx of the right kidney associated with a calculus.

The urinary infection was successfully treated with tetracycline and the anaemia responded well to treatment with vitamin B12, a maximum reticulocyte response of 30 per cent ensuing with the gradual expected rise in the haemoglobin; oral iron supplements were necessary in the later stages to bring the haemoglobin up to 100 per cent.

Soon after the reticulocyte crisis, he developed, for the first time in his life, an acute attack of gout in the tarsus of the left foot which cleared up on colchicine. The serum uric acid was 7.2 milligrams per cent.

In view of the history of recurrent nephrolithiasis, serial investigations of the serum calcium, phosphate and alkaline phosphatase were done (Table I).
Other tests revealed the twenty-four hour urinary calcium output to be 730 milligrams, and the calcium balance—on a fixed intake of 120 milligrams of calcium daily—showed an output of 490, 380 and 400 milligrams respectively on three successive days. The plasma calcium fractions showed: ionised calcium 7·33 milligrams per cent (the upper limit of normal being 6·2); complexed calcium 0·19 milligrams per cent; protein-bound calcium 3·98 milligrams per cent; the total calcium was 11·5 milligrams per cent, with a plasma albumin and globulin of 4·4 and 2·8 milligrams per cent respectively.

The cortisone test, in which 150 milligrams were given daily for ten days, did not produce any fall in the level of serum calcium, thus confirming the diagnosis of hyperparathyroidism.

The renal function test revealed no albuminuria after the urinary infection had been treated and the specific gravity ranged from 1·015 to 1·004. The blood urea was 39 milligrams per cent. Radiographs showed no evidence of bone disease, but several joints showed calcification in the articular cartilage or joint capsules (Figs. 1 to 3).

### TABLE I

<table>
<thead>
<tr>
<th>Date</th>
<th>Serum calcium (milligrams per cent)</th>
<th>Serum inorganic phosphate (milligrams per cent)</th>
<th>Serum alkaline phosphatase (King units)</th>
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<tr>
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<td>11·9</td>
<td>2·8</td>
<td>6·8</td>
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<tr>
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<td>13·6</td>
<td>2·8</td>
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<td>2·5</td>
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<td>28/1/58</td>
<td>13·0</td>
<td>2·1</td>
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</tr>
<tr>
<td>15/2/58</td>
<td>14·4</td>
<td>2·5</td>
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</table>

The patient was transferred to University College Hospital where, in March 1958, Mr D. R. Davies carried out an exploration of the parathyroids, finding three normal glands, from which material was taken for biopsy, and one tumour—weighing 850 milligrams and composed mostly of chief cells—which was removed from the lower pole of the right lobe of the thyroid.

After operation, as expected, the serum calcium fell to 7·6 milligrams per cent and the urine calcium excretion was 24 milligrams in twenty-four hours; there were mild signs of latent tetany and fairly severe mental depression. At the same time, acute pain developed in the right knee and foot with pyrexia; this appeared to be typical of a severe attack of gout precipitated by the operation, and the serum uric acid was 6·0 milligrams per cent; this responded to colchicine and the hypocalcaemic manifestations soon passed off. Two weeks after operation the serum calcium was 8·7 milligrams per cent, the ionised calcium being 5·28 milligrams per cent, and he was discharged from University College Hospital two days later.

He was readmitted to Queen Mary's Hospital, Roehampton, in April 1958 complaining of acute pain and stiffness of the neck in which all movements were severely restricted by pain and there was some fullness to be felt on the right side. Shortly afterwards he developed a further attack of gout in the tarsus of the right foot—the serum uric acid being 5·4 milligrams per cent—and again treatment with colchicine gave dramatic relief of the pain in the neck, as well as in the foot, indicating that the episode in the neck was a gouty manifestation.

When last seen as an out-patient in June 1958 he was very well and had no complaints. His only treatment was a regular dose of vitamin B12, and the blood count was normal. In April 1958 the serum calcium was 10·2 milligrams per cent, the inorganic phosphate was 2·5 milligrams per cent and two months later they were 11 milligrams per cent and 3·2 milligrams per cent respectively. Further radiographs of the joints have as yet shown no regression of the calcification of the articular cartilages and joint capsules.
DISCUSSION

Israelski (1931) in an article on semilunar cartilage calcification described a patient who, besides showing calcification in these cartilages, also showed a narrow band of calcification parallel to the articular surface of the femur, which was presumed to represent an incrustation of calcification on the surface of the articular cartilage. The patient was a diabetic whose complaint was of pain and stiffness in the knees.

FIG. 1
Radiographs of the shoulders showing calcification in the articular cartilage of the head of the humerus.

FIG. 2
Radiograph showing calcification in the cartilage of the symphysis pubis.
Wolke (1935) examined 2,569 radiographs of knees, and in eight found calcification in the semilunar cartilages. Five had unilateral or bilateral calcification in the articular cartilage of one or more joints, which included the knee, hip, acromio-clavicular, shoulder, elbow and radio-carpal joints and the pubic symphysis. Wolke discussed the etiology of this calcification. In his opinion it occurred in subjects with a constitutional disposition to cartilage changes, as a result of a nutritional deficiency owing to changes conditioned by advancing age in the vessels of the joints.

Harmon (1944) described two patients in whom there was calcification in the articular cartilage of the knee, and also, in one, calcification in the radio-carpal joint. One of the patients, a man aged seventy-five, complained of "rheumatism all over for thirty years." He had prostatic enlargement and urinary infection. The other, a man aged fifty-five, had pain, swelling and limitation of movement of the right knee. He was febrile and was found to have, on one occasion, a serum uric acid of 10-0 milligrams per cent. The serum calcium was 10-4 milligrams per cent and the serum phosphorus 2-5 milligrams per cent. The calcification was described as degenerative.

Edwards and Davis (1953) described a case designated "Primary asymptomatic calcification of articular cartilage."

Bunjé and Cole (1956) noted calcification in the articular cartilages of a woman who first complained of joint pains in 1945, and who had, on one occasion, a serum calcium of 14-0 milligrams per cent. They suggested that "calcium deposition is a result of repeated attacks of a mild acute arthritis of unknown aetiology, causing calcium deposition in the fibrocartilage articular discs and calcium to be laid around or over the superficial layers of hyaline cartilage."

Marziani (1953) described a man with calcified articular cartilage who had suffered from joint pains at twenty and at twenty-six years of age. The serum calcium was 11-0 milligrams per cent.
Losada, Cox, Rodriguez, Ronban and Silva described in 1957 a patient with calcification of articular cartilage of the shoulder, knees, wrists, ankles, hips, elbows, a metacarpo-phalangeal joint and a metatarso-phalangeal joint. There was also aortic insufficiency, considered to be rheumatic in origin. Laboratory investigations included serum calcium estimation which was 12.0 milligrams per cent and a calcium excretion estimation which was 160 milligrams in twenty-four hours.

Radiographs of the patient described here show changes in the articular cartilage which appear identical with those previously published.

The finding of a parathyroid tumour suggests that all patients with a similar calcification should be investigated carefully for hyperparathyroidism. Of the cases described by others, we think that those of Bunjé and Cole and of Losada and his fellow workers in particular may possibly have had a parathyroid adenoma as the underlying basis of the generalised articular calcification.

Calcified articular cartilage may be found, as in this patient, without associated joint symptoms.

SUMMARY
A case of calcification of articular cartilage in association with a parathyroid tumour is described. Previously reported cases of articular calcification are briefly discussed, and it is recommended that patients with articular calcification of undetermined cause should be investigated for hyperparathyroidism.

REFERENCES