CALCIFICATION OF ARTICULAR CARTILAGE

Report of a Case

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A few cases have been described of patients with recurrent multiple arthritis, which has been variously diagnosed as gout or rheumatoid arthritis, and which has been found to have a curious calcification apparently affecting the articular cartilage. Edwards and Davis (1953) described a man of forty-five who had a history of three years of intermittent pain in the knees, right ankle and right shoulder, and who attended with a swollen, painful right knee which had troubled him for two weeks. The joint was warm and had an effusion into it but it retained a full range of movement. No other joint was affected at the time. The knee became normal again in four weeks simply with rest in bed. Four months later there was another slight attack. Radiographs showed changes like those we are to describe in our case, and which were found in the knees, shoulders, the left carpus and the spine between the bodies of C.6-7 and T.10-11. Radiological appearances were unchanged five months later. Edwards and Davis considered that the unusual feature of their case was calcification of hyaline articular cartilage. They pointed out that many joints so affected had been symptomless and thought that the presenting symptoms may have been due to early osteoarthritis. There was no evidence of a generalised disturbance of calcium metabolism.

Harmon (1944) described two cases. The first was suggestive of degenerative arthritis in a man of seventy-five who had had "rheumatism all over" for thirty years but especially intermittent pain and swelling of the left knee. Radiologically there was calcification following the contours of the articular cortex of both femoral condyles in each knee. The knees also showed osteoarthritis. The patient was treated with rest, extension of the legs and aspiration of the knees, with relief. Later the patient died with an enlarged prostate, urinary infection and auricular fibrillation. At necropsy sections of the semilunar cartilages showed only "extremely light and sporadic" yellowish discoloration indicating small scattered foci of calcification. These did not cast shadows when the semilunar cartilages were radiographed. Articular cartilage was missing from the central portions of the medial tibial and femoral condyles, these defects being due, it was thought, to advanced degenerative changes. Sections of the cartilage peripheral to the bare bone showed, grossly and microscopically, an advanced state of diffuse calcification.

The second case was of a man of fifty-five thought to have gout. The history was of four days of pain in the right knee with an effusion into the joint, followed by a similar affection of the left knee. The patient's temperature ranged between 100 and 102 degrees Fahrenheit. The total white cell count was 9,800 per cubic millimetre and erythrocyte sedimentation rate was 21 millimetres in the first hour (Cutler-tube method). The serum calcium was 10-4 milligrams per cent, the serum phosphorus 2-5 milligrams per cent, the urea nitrogen 19 milligrams per cent, and the serum uric acid on three separate occasions 6-7 milligrams, 7-6 milligrams, and 10 milligrams per cent respectively. Radiologically there was no evidence of gout, but the knees and wrists showed calcification following the contours of the articular surfaces of the joints. Radiographs two years later showed a slight increase of the calcification. The patient was treated with rest in bed, colchicine, bandages to the knees, and traction on the right leg. A slightly cloudy yellow liquid was aspirated from the right knee and contained a moderate number of polymorphonuclear neutrophil cells and was sterile. It was pointed out that no specific treatment was given to the two patients, except of the acute symptoms.
Harmon claimed that there was no account in the literature of this kind of dense calcification in articular cartilage.

Wolke (1935) examined radiographs of 2,569 knees and found calcification of the semilunar cartilages in eight cases; and five of these eight had other joints affected, including calcification of some intervertebral discs and the articular cartilages of various joints. This was remarked as something that had not been observed before. So far as could be judged there had been no symptoms. In four cases the changes were known to have been unaltered in from one to nine years.

Israelski (1931) reported a case of diffuse calcification of the semilunar cartilages in both knees, there being smaller patches of calcification outside the semilunar cartilages and a narrow band of calcification lying parallel to the articular surfaces of the femurs. The large arteries around the joints were calcified. The patient was a man of sixty, with a history of diabetes mellitus for three years. He complained of pains and stiffness in the knees, examination of which revealed only coarse crepitation on movement. He had no treatment and no change was observed in the joints during the year.

**CASE REPORT**

The present account is of a coloured Jamaican woman of about thirty-one years of age. There was no family history of arthritis. Married for twelve years, she had had an abortion at three months, two years after marriage, and denied further pregnancies. She denied having had venereal disease, and said that her general health had been good. She said that she had nausea before her menstrual periods and before attacks of joint pains (which were unrelated to menstruation). Four years before our seeing her she had weighed 150 lb. and had lost some 30 lb. since.

The history of her arthropathy is compiled from notes taken at the Kingston Public Hospital, which she first attended in 1952, and from her own account to us at the University College Hospital, Jamaica, which she first attended in 1954. The presenting symptom was always of joint pain and she was first troubled in 1945 when she was between twenty-four and twenty-seven years (there are discrepancies in her declared age at various times). Between 1945 and 1952 she had four similar attacks of joint pain and the first three were treated with injections given by her own doctor and each attack had gone entirely within a few weeks. The fourth attack occasioned her first admission to hospital in January 1952. At that time she had been unwell for two weeks, having had a sudden onset of fever and "tonsillitis" followed, a few days later, by joint pain and loss of appetite. On examination she had a temperature of 101 degrees Fahrenheit, a pulse of 116 per minute, and a respiration rate of 24 per minute. There was a trace of protein in the urine. The right tonsil was inflamed and the liver and spleen were enlarged and tender. The knees were swollen and inflamed, the tibiae tender, and the ankles and feet were swollen. The next day the right wrist was swollen and tender. The temperature was of the order of 101 degrees Fahrenheit, then 99 degrees Fahrenheit for two days and thereafter she was afebrile until her discharge from hospital three weeks later. A diagnosis of rheumatoid arthritis was made and she received intramuscular injections of Myocrisin, four in all, beginning with 0.01 gramme and rising by 0.01 gramme with each dose. She had also had an injection of procaine penicillin, 600,000 units, and sodium bicarbonate, sodium salicylate and ferrous sulphate by mouth. Two weeks after her discharge she was readmitted to hospital with a history of fever but with no sore throat. The pains in her joints had returned, moving from the left ankle to the left hip, the right ankle, right wrist, right shoulder, elbow, knee, and a sacral joint. She was given a similar course of Myocrisin and discharged three weeks later.

She was next seen by her own doctor two years later, when she had an acute attack of joint pain and fever which she treated with an injection of one million units of penicillin and with salicylates by mouth. She was improved by the time she was admitted to the Kingston
Public Hospital again three days later, and gave a history of three weeks of fever and fleeting pains in various joints. At the onset the pain had been in the left heel. There had been swelling and tenderness of the joints which had subsided, leaving the joints "stiff." Examination showed that she had a temperature of 100.6 degrees Fahrenheit (which subsided over the next three days), swelling and tenderness of the right ankle, but merely stiffness of the right wrist and knee. For the first time, apparently, the patient had had pain in a small joint and for two days before admission had pain in the metacarpo-phalangeal joint of the right index finger. There was no deformity of the hands. She was discharged after twelve days' treatment in hospital with rest, penicillin and salicylates. Five and a half months later she first came to the University College Hospital saying that for ten days she had had pains in the neck, hips, knees, ankles and left hand, in that order. She was found to have a temperature of 100 degrees Fahrenheit and the first and second metacarpo-phalangeal joints of the left hand were swollen, tender, slightly reddened and hot. She was advised to rest in bed at home and given aspirin, 30 grains, to be taken four times daily. A week later there was considerable improvement. Her temperature was normal. There was still some swelling of the first and second metacarpo-phalangeal joints of the left hand and some swelling on the back of the left carpus. Pain slightly limited movement of the left wrist although the wrist joint itself did not appear to be involved. Just over three weeks from the start of the attack she had recovered. Apart from the joint condition, physical examination showed the patient to be normal.

Six months later pains returned to the right thumb and wrist, then to the left knee and to several finger joints. The pains were severe enough to keep her awake. She was admitted

**TABLE I**

<table>
<thead>
<tr>
<th>Date</th>
<th>Haemoglobin (grammes per cent)</th>
<th>Red blood cells (million)</th>
<th>Packed cell volume (per cent)</th>
<th>White blood cells</th>
<th>Erythrocyte sedimentation rate</th>
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<td>30.1.52</td>
<td>9.6</td>
<td>3.7</td>
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<td>14,300</td>
<td>-</td>
</tr>
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<td>6.3.52</td>
<td>11.3</td>
<td>3.85</td>
<td>-</td>
<td>8,200</td>
<td>-</td>
</tr>
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<td>26.1.54</td>
<td>10.7</td>
<td>4.24</td>
<td>-</td>
<td>12,350</td>
<td>-</td>
</tr>
<tr>
<td>21.7.54</td>
<td>-</td>
<td>4.24</td>
<td>-</td>
<td>-</td>
<td>62 (Westergren)</td>
</tr>
<tr>
<td>29.11.54</td>
<td>12.5</td>
<td>4.42</td>
<td>42</td>
<td>8,450</td>
<td>28 (Wintrobe)</td>
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<tr>
<td>16.2.55</td>
<td>14.9</td>
<td>4.15</td>
<td>41</td>
<td>10,250</td>
<td>40 (Wintrobe)</td>
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<tr>
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<td>13.0</td>
<td>-</td>
<td>38</td>
<td>7,200</td>
<td>-</td>
</tr>
</tbody>
</table>

**TABLE II**

**Biochemical Examinations of Blood**
(All figures are in milligrams per 100 millilitres)

<table>
<thead>
<tr>
<th>Date</th>
<th>Calcium</th>
<th>Uric acid</th>
<th>Urea</th>
<th>Phosphorus</th>
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<tr>
<td>1.2.54</td>
<td>14</td>
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<td>-</td>
</tr>
<tr>
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<td>5.0</td>
<td>54</td>
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</tr>
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<td>3.3</td>
<td>-</td>
<td>6.0</td>
</tr>
</tbody>
</table>
to hospital with mild pyrexia but no other abnormal findings apart from the joints. The most severely affected of these was the right wrist. She would permit no movement of the wrist and there was redness of the skin over the joint. There was considerable pitting oedema about the wrist and on the back of the hand. The fingers of the right hand ached and were held in moderate flexion and the soft tissues of the fingers were slightly swollen. The right elbow was painful, swollen, and held at a right angle. Though she complained of pain in the
knees, they were normal on examination. Treatment was confined to rest in bed and a few doses of salicylates were given. The condition subsided abruptly and a week after the onset the joints were normal on examination.

FIG. 4
Right and left hips, and interpubic joint.

FIG. 5
Right and left knees.

A gynaecological examination showed that she had thickened Bartholin’s glands and had undoubtedly had gonorrhoea but there was no evidence of serious residual pathology in the pelvis.
Fig. 6
Lateral radiographs of knees.

Fig. 7
Detail radiograph of knee.
When last seen a month later she was in good health and was free from symptoms in the joints.

**Investigations**—In 1952 a trace of protein had been found in the urine with pus cells in the centrifuged deposit and staphylococcus albus on culture. While she was under our observation no significant urinary abnormality was detected.

The Kahn test for syphilis, the V.D.R.L. test, the Kolmer-Wassermann test, the lymphogranuloma venereum complement-fixation test, and the gonococcal complement-fixation test were all negative. The red blood cells did not show any characteristics of sickle-cell disease.

Two chest radiographs and electrocardiographs were normal.

Table I gives the results of studies on the blood cells. The first three readings indicate an anaemia. On the three occasions that the white blood cell count was above 10,000 per cubic millimetre the count was made within two days of the patient's being admitted to hospital in an acute attack and while she still had fever. The readings for the white blood cells below 10,000 per cubic millimetre were all taken when the patient was well or past the acute phase. Differential white blood cell counts were normal or, with the raised counts, had an increase in the polymorphonuclear neutrophil cells. The two higher sedimentation rate readings occurred during acute phases. The figure of 28 was obtained when the patient was well.

Table II gives the results of some investigations of the blood chemistry. The serum calcium was once raised and this was at a time when the patient was having treatment in hospital but when the acute attack had passed off. The blood urea and the blood uric acid were once raised on an occasion when the patient was, in fact, without symptoms.

**Radiological findings**—Calcification was first seen over the surfaces of the joints of the hands and over the humeral heads (seen in a routine chest film). All the large joints of the upper and lower limbs were then found to be similarly affected (Figs. 1 to 8). The calcification was seen as a thin linear deposit on the articular surface and separated from the articular bone cortex by a narrow translucent zone. The contour of the articular bone cortex was not eroded or deformed and there was no change in the "joint-space" distance between the
adjacent bony surfaces. The calcified surfaces were clearly seen to be in contact with each other at many sites, suggesting that the calcium lay in or on the superficial layers of the hyaline cartilage of the articular surfaces. In the hands and feet the deposition was less extensive in the peripheral joints but was fairly extensive over the carpal and tarsal bones. Calcium was present over the whole articular surfaces of the shoulders, elbows, hips and knees. Bone density was normal with no evidence of any demineralisation.

Calcification was also visible in the fibrocartilage discs of the symphysis pubis, knee, and ulnar-carpal joints. It could not be seen in the intervertebral discs or articular facets of the spine, nor in the sacro-iliac joints though, had it been in the latter in only a thin layer, the density of the overlying soft tissues might have obscured it. The calcification did not alter over the period of observation of eight months. Comparison of recent radiographs with those taken a year previously at the Kingston Public Hospital showed no change.

Conclusion—Calcification was found in the fibrocartilage discs and on the hyaline articular cartilage surfaces of many joints, associated with intermittent fever, joint pain and swelling, but with otherwise normal physical findings and blood studies. No perceptible radiological changes developed during or after acute attacks of arthropathy.

DISCUSSION
Calcification occurring in the fibrocartilage discs alone is relatively common, but is not associated with pain or with calcification of the hyaline cartilage surfaces. Calcification in the joint synovial membranes has been described in association with hyperparathyroidism by Keynes and Taylor (1933). The distribution of calcification is not the same in our case, nor is there evidence of disturbed calcium metabolism.

The adjective “asymptomatic” used by Edwards and Davis (1953) for this condition seems inadvisable as the pain was severe enough in our case to keep the patient awake, and to cause her to seek medical aid and to be admitted to hospital. It is certainly remarkable, with such unchanging radiological appearances, that the symptoms should sometimes be confined to so few joints at a time and that the acute phases should be so transitory.

The cause of this condition is unknown, but it is suggested that the calcium deposition is a result of repeated attacks of a mild, acute arthritis of unknown etiology causing calcium deposition in the fibrocartilage articular discs and calcium to be laid around or over the superficial layers of hyaline cartilage as Edwards and Davis surmised. Otherwise the joints are remarkably normal and the patient is well after the acute phase has subsided, suggesting that the articular surface cannot be roughened or its function disturbed in spite of the persistent layer of calcium.

SUMMARY
1. Idiopathic calcification of articular cartilages is described in a Jamaican woman of thirty-one years who had intermittent joint pains for ten years and who had evidence of past gonococcal infection. She was otherwise normal.
2. The etiology of the condition is unknown.
3. Previous literature is reviewed.

Our thanks are due to Professor E. K. Cruickshank in whose ward the case was studied, and to the Kingston Public Hospital, Jamaica, for kindly lending us their notes and radiographs of the patient.

REFERENCES

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