Case report

Acute synovitis with intra-articular apatite deposits in an osteoarthritic metacarpophalangeal joint

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SUMMARY A patient was shown to have acute arthritis in a metacarpophalangeal joint, with local calcification indicated by *x*-rays. Surgical and pathological examinations showed strictly intra-articular apatite crystal deposits and an erosive osteoarthritis. These crystal deposits could account for the synovial inflammation; they are thought to be related to bone fragments embedded in the synovium. The predisposing role of previous local injections of corticosteroids is debatable.

During the past decade hydroxyapatite (HA) crystals have been considered as possible initiating factors of acute inflammatory osteoarthritis.¹⁻³ The present case provided an opportunity to study this problem.

tion of mobility in the cervical spine, the rest of the articular examination was normal.

The erythrocyte sedimentation rate (Westergren)

Case report

CLINICAL DATA

Since 1973 a right-handed upholsterer, aged 53, had suffered from recurrent pain in the metacarpophalangeal (MCP) region of the right index finger. The only predisposing factor was that of minor occupational traumas. In 1980 the frequency of the painful episodes increased, and over a period of three years the joint was injected 10 times on its dorsoulnar aspect with 2 to 3 mg of triamcinolone hexacetonide. In March 1983, two weeks after the last injection and without any previous trauma, the patient woke in the early morning with acute pain in the proximal part of the right index finger. For the first time local redness was reported but without fever. When the finger was examined next day, a red, warm, and fluctuating swelling of the MCP joint was noted (Fig. 1). There was no fever, skin erosion, fistula, sclerodactyly, or axillary adenopathy. Heberden's and Bouchard's nodes were present on both hands. Apart from a slight limita-

Fig. 1 Photographs of both hands (March 1983) showing fusiform swelling of the proximal phalanx of the right index finger.

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was 3 mm/1st h. Blood count, serum fasting glucose, creatinine, uric acid, calcium, phosphorus, alkaline phosphatase, and thyroid function tests were all normal. The rheumatoid factor was negative. During the following 10 days the joint was aspirated on three occasions; 1–2 ml of white milky fluid was drawn off. Diclofenac (100 mg per day) was given, but the pain and swelling persisted.

On 8 April 1983 surgical synovectomy of the dorsal part of the MCP joint and total arthroplasty with Swanson's prosthesis were successfully performed. Follow up examination in June 1984 showed the index finger to be free of pain, with no limitation of movement.

It should be added that the patient had a previous history of other articular complaints. In 1962 and 1972 he underwent an operation for osteoarthritis of both first metatarsophalangeal joints. In 1980 he suffered from bilateral shoulder peri-arthritis. Since November 1983 paraesthesia in the first three fingers of the right hand was reported. Carpal tunnel syndrome was diagnosed on clinical grounds, and a surgical decompression of the median nerve was performed in January 1984. On pathological examination synovial tendon sheath and carpal volar ligament were normal, with no deposits of calcium.

RADIOLOGICAL DATA

X-rays taken during the period of acute flare-up revealed joint space narrowing of the second metacarpophalangeal joint with dystrophic remodelling of the metacarpal head, and calcified deposits (Fig. 2b and c). Arthrography showed a dilated and villous synovial cavity, in contrast to normal appearance⁴ (Fig. 2d). X-rays taken seven years previously had shown only mild narrowing of the joint space (Fig. 2a). In addition moderate signs of osteoarthritis in distal interphalangeal joints were noted and juxta-articular calcifications of the right supraspinatus tendon near its trochanteric insertion, and of the medial ligament of the left knee near its origin. No signs of chondrocalcinosis were seen in the knee, shoulder, or wrist joints.

PATHOLOGICAL DATA

Synovial fluid contained about 100 000 leucocytes/mm³ (100 × 10⁹/l) (98% polymorphonuclear) but no birefringent crystals; it was sterile. Calcium clumps stained with alizarin red S⁵ were shown by scanning electron microscopy to consist of microspheroids (8–10 µm in diameter) with a molar phosphorus to calcium ratio of 0.44 found by energy dispersive analysis. (Standard HA has a ratio of 0.43.) The x-ray diffraction pattern of the spheroids showed interplanar d-spacings characteristic of HA crystals, as compared with the American Society for Testing

and Materials Card 9–432; the width of the diffraction lines indicated crystals whose sizes were similar to those of bone mineral and tissue dystrophic calcifications. No transmission electron microscopic examination was performed in searching for signs of crystal phagocytosis. Total hydroxyproline in the fluid used for washing the joint (determined after hydrolysis by the Kivilico method) was 666.6 µmol/l (87.4 mg/l).

During surgery through a dorsolunar approach no periarticular calcium deposits were seen. Intra-articular injection of methylene blue stain showed no link with periarticular tissues. The fibrous capsule adhered to the underlying thickened synovial membrane. Articular surfaces were devoid of cartilage and were eburnated. Rare, punctate, white deposits were visible on synovial surfaces, and these were analysed by x-ray diffraction with a Guinier's camera (Ni filtered Cu Kα radiation, 40 kV, 24 mA). Patterns were characteristic of HA crystals; the width of the diffraction lines indicated crystals whose sizes were similar to those of bone mineral and tissue dystrophic calcifications.

The histological examination was performed on material fixed in 10% neutral formalin, embedded in paraffin, and stained by haematoxylin-eosin, by van Gieson-elastin, by Prussian blue for iron, and by von Kossa's method for phosphate. The fibrous capsule was flanked by a hyperplastic synovium with superficial necrotic areas containing dusty calcareous material and some deeply embedded small calcified debris, and haemosiderin deposits were present among fibroblasts and histiocytes in the subintimal region. A thrombotic vein and rare deep foci of lymphocytes were observed but no circumscribed dystrophic calcinosis, synovial chondromatosis, obvious bone fragments, calcium pyrophosphate dihydrate (CPPD), or sodium urate crystal deposits were found.

The metacarpal head (resected during surgery at 1 cm from the articular surface and decalcified in formalin-formic acid) showed signs of osteoarthritic remodelling with only marginal cartilaginous remnants and no calcareous deposits. Under the eburnated surfaces the bone remodelling was associated with marrow changes, lymphoplasmocytic infiltrates, and small areas of basophilic necrosis, possibly containing dusty calcium deposits.

Transmission electron microscopy was performed on samples taken during the operation and immediately fixed for two hours in 2.5% glutaraldehyde buffered with piperazine diethane sulphonate (PIPES) (0.2 M, pH 7), then washed with PIPES, postfixed in 1% osmium tetroxide, washed again (in 0.1 M cacodylate buffer, pH 7.4), and embedded in a resin Epon B. Ultrathin sections were prepared

Fig. 2 X-ray of the second and third metacarpophalangeal joints of the right hand. (a) AP view (28 November 1976). (b) AP view (13 March 1983). (c) Oblique view (18 March 1983). (d) Arthrogram - oblique view (22 March 1983).

and examined with a Jeol C $\times$ 100 electron microscope. Specimens from the eburnated articular surface showed collagen fibres and apatite crystals, indicating calcified cartilage or bone tissue. Synovial specimens showed only areas of apatite crystal

deposits devoid of collagen fibres (as checked on sections decalcified by phosphotungstic acid) adjacent to normal mesenchymal cells. The crystal deposits were partly grouped in spheroid clumps.

Discussion

The findings of this case imply that a severe inflammatory arthropathy may be caused by intra-articular HA deposits. Analyses of synovial fluid and surgical specimens failed to show CPPD crystals, the calcium salt generally considered responsible for acute microcrystalline arthritis. The inflammatory properties of HA crystals, both in intra- and juxta-articular location, have been well demonstrated.^{2 6 7}

In this case initial x-rays did not show periarticular calcifications of the second MCP joint (Fig. 2a). It is interesting to note that a circular area of opacity, probably a juxta-articular HA deposit, was found on the radial aspect of the third MCP joint; it was asymptomatic and its shape remained unchanged in the latest x-ray (Fig. 2a and b).

Intra-articular HA deposits might come from the basal calcified plate of articular cartilage or from subchondral bone of abraded articular surfaces, or from both. This origin is suggested in our case by the discovery of fine calcified debris embedded within the synovium, though HA deposits observed by electron microscopy were not associated with any collagen network, which one would expect with broken down cartilage or bone. This discrepancy might be explained by the crystallisation of calcium phosphate released during the dissolution of pre-existing HA crystals in bone debris. Such a process would be comparable to the mineral redistribution which has been reported in the growing skeleton.⁸

The importance of the osteoarticular erosions in our case is shown by the high level of synovial hydroxyproline compared with reported normal values in plasma (1.3–1.8 mg/l) and with values in the synovial fluid of osteoarthritic shoulders.⁶ Erosive osteoarthritis with abrasion of exposed and eburnated bone, which is associated with HA crystal deposits, has already been observed.^{1 2 9–11}

The origin of this severe osteoarthritis is unclear. A predisposition is indicated by the presence of Heberden's nodes. Occupational microtraumas could also have a predisposing role, as could the depot formed by repeated injections of corticosteroids (whose crystal length is about 10 µm). Development of joint calcifications by corticosteroids has been suggested in rheumatoid arthritis.^{12 13}

HA crystal deposits have been found only in intra-articular locations in this case. Generally they are found in extra-articular sites as dystrophic calcifica-

tions at the insertion of tendons, or ligaments. The distinction between these two kinds of HA deposition disease which are different in nature could be useful in assessing possible risks of local corticosteroid therapy.

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