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Case presentations

Presented by the Department of Medicine, Bristol Royal Infirmary, Bristol BS2 8HU

CASE 1

An 89 year old woman with diuretic-induced tophaceous gout. Points of note were:

(1) The patient had been receiving diuretics for many years, but had no impairment of renal function.

(2) Radiographs showed typical gouty erosions in the hands, but she had never had an acute attack of gout (Fig. 1).

(3) Large tophi were seen, mainly on the hands, and in particular over the distal interphalangeal joints. Some of these tophi had discharged (Fig. 2).

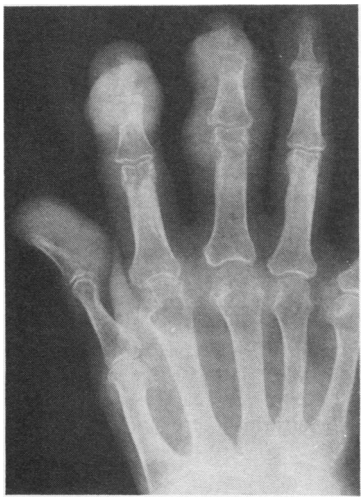

Fig. 1 *Radiograph of hand (case No 1) showing gouty tophi of the soft tissues and typical bone erosions of gout. The patient had never had an attack of acute gout.*

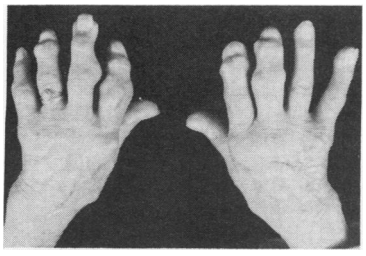

Fig. 2 *Hands of case No 1. Note the large gouty tophi and the distribution over the interphalangeal joints.*

Discussion points included:

(1) The role of diuretics in the genesis of gout in the elderly.

(2) The contributions of aging of connective tissue and osteoarthritic changes around the distal interphalangeal joints in dictating the distribution of crystal deposits in the elderly.

(3) The striking lack of inflammation around the tophi and the absence of acute attacks.

(4) The aetiology of the bony erosions in gout in the apparent absence of inflammation.

This patient was thought to be similar to others described recently with gouty tophi without gouty arthritis (see Scott, p. 16).

The possible factors relating to distribution, including connective tissue changes, temperature, and osteoarthritis had been discussed by Dr Calvert (see Fiddis *et al.*, p. 12). Professor Dixon suggested the term 'impostumous gout' to describe such cases.

CASE 2

A 31 year old woman suffered a knee injury when aged 16, and first presented with an acute monoarthritis of that knee 12 years later. She has since had five well-characterised attacks of pseudogout in that knee. Points noted included:

(1) Cruciate instability of the left knee only.

(2) Radiological chondrocalcinosis limited to the left knee.

(3) The lack of any metabolic abnormality or family history of arthritis.

(4) The good preservation of cartilage thickness in the joint.

Discussion centred around the concept of secondary calcific chondrocalcinosis occurring in localised areas of previous joint damage in the absence of any systemic disorder. Although this association has not been widely recognised, similar cases have been reported (see Doherty, p. 38).

CASE 3

A 75 year old ex-ballet dancer with generalised hypermobility and CRST syndrome. She developed a self limiting attack of severe low back pain and recurrent attacks of arthritis affecting the wrists and metacarpophalangeal joints.

Points noted included:

(1) The typical hands of CRST syndrome with associated reversible ulnar deviation of the fingers and a Z thumb deformity.

(2) Radiological chondrocalcinosis of the knees, symphysis pubis, and triangular ligaments of wrists (Fig. 3).

(3) The presence of calcification around the annulus fibrosis of several disc spaces in the lumbar spine.

Discussion points included:

(1) The possibility that the low back pain could have been related to the

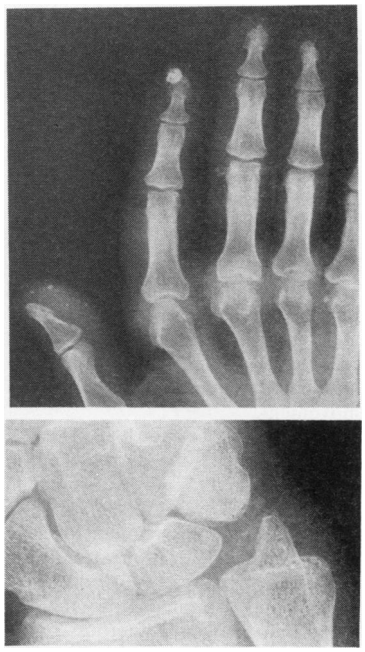

Fig. 3 *Radiographs of the hand (case No 3) showing* (a) *close up of the fingers to show the typical soft tissue calcification and resorption of the terminal phalanges seen in the CRST syndrome, and* (b) *close up of the wrist of the same hand to show the associated chondrocalcinosis of the triangular ligament.*

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presence of crystal deposits in the spine, as has been suggested previously (see Doherty, p. 38).

(2) The association between the CRST syndrome and the chondrocalcinosis. It was felt that the fingertip calcification was typical of CRST syndrome but that the chondrocalcinosis may be a chance association, as a high percentage of 75 year olds have age-related chondrocalcinosis.

(3) The influence of her generalised hypermobility on the crystal deposition. The question of a primary collagen abnormality or of hypermobility induced by her dancing was discussed, and the question of hypermobility in relation to generalised chondrocalcinosis was brought up. Factors such as aging, hypermobility, and connective tissue changes in the pathogenesis of chondrocalcinosis had been discussed previously (see Mitrovic, p. 19 and Doherty, p. 38).

CASE 4

A 43 year old man with acromegaly presented with a stiff, painful back and painful swollen knees. He had undergone hypophysectomy and radiotherapy to the pituitary fossa in the previous year.

Points noted included:

(1) Persistence of gross features of acromegaly.

(2) A mild kyphoscoliosis and restriction of spinal mobility.

(3) Large effusions, pronounced crepitus, and bony swelling of the knees, with retention of a full range of movement.

(4) Radiological features characteristic of those described in acromegalic arthropathy were noted in the spine and knees.

(5) He was also noted to have periarticular calcification around the left knee.

(6) Fluid aspirated from the left knee had shown numerous round globules staining positively with Alazarin red (see Schumacher, p. 54).

Discussion points included:

(1) The possible role of growth hormone in predisposing to apatite deposition in the cartilage.

(2) The origin of the apatite in the synovial fluid. The *x*-ray films show good preservation of cartilage, suggesting that the particles would have to come from new deposits in the superficial areas of the cartilage, or synovium and capsule, rather than from the bone. The possible disruption of particles from preformed deposits in the soft tissues during the process of aspiration was noted.

(3) The nature of the arthropathy. The knee disease was thought to be typical of acromegalic arthropathy, and associated apatite deposits in synovial fluid have been described previously (see Schumacher, p. 54). The spinal disease was thought to be less typical in view of the pronounced restriction of movement, but it was noted that this was responding well to physiotherapy.

CASE 5

A 71 year old woman with longstanding generalised nodal osteoarthritis presented with acute inflammation around the distal interphalangeal joint of the left middle finger. The lesion ulcerated, discharging fluid containing numerous apatite crystals ('impostumous apatite deposition disease' Fig. 4).

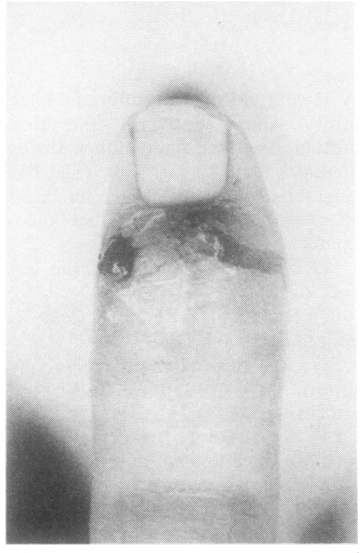

Fig. 4 *Distal interphalangeal joint of the left middle finger (case No 5), showing the inflammation and discharge from the finger after flare up of the 'Heberden's node'. The discharge contained masses of calcium containing particles thought to be apatite, and the patient had evidence of calcific periarthritis elsewhere.*

Points noted included:

(1) The clinical and radiological evidence of gross osteoarthritis of the interphalangeal joints.

(2) Radiological evidence of calcific periarthritis is the left ring finger.

(3) Numerous para-articular cysts around the distal interphalangeal joints typical of those seen in generalised osteoarthritis.

Discussion points included:

(1) The mode of formation of para-articular cysts adjacent to the distal interphalangeal joints in osteoarthritis. It was felt that these might be generated in a similar way as other joint cysts—that is, with an initial communication with the joint, and a valvular mechanism pushing hyaluronate into the cyst.

(2) The presence of apatite in cysts and in distal interphalangeal joints in osteoarthritis was discussed. Various contributors noted that they had never been able to identify apatite in the hyaluronate cysts, but that it had on occasion been found in joint fluid aspirated directly from the distal interphalangeal joint in Heberden's nodes.

(3) It was agreed that no good data were available on the relationship between calcific periarthritis and osteoarthritis but that the patient had obviously had a typical attack of acute calcific periarthritis in addition to having generalised nodal osteoarthritis (see Faure, p. 49).

CASE 6

A 73 year old woman with a four year history of a destructive arthropathy, principally affecting the shoulders, knees, and midtarsal joints. Trauma precipitated the inflammation of the right shoulder at the start of the illness. Over the next two years this joint rapidly became grossly disorganised, and an arthroplasty was carried out. She subsequently developed pain, effusions, and radiological evidence of progressive destructive changes, chiefly affecting the left shoulder, knees (left worse than right), and midtarsal joints.

Points noted were:

(1) Radiological evidence of calcific periarthritis preceding the destructive changes in the shoulder.

(2) Loss of the rotator cuff apparatus both clinically and

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radiologically, with upward subluxation of the head of the humerus in the shoulder remaining on the left (see Fig. 11 in Watt, p. 73).

(3) Gross instability of the left knee, with a marked valgus deformity.

(4) The large, cool effusions in all the affected joints. These had been aspirated and shown to contain a few mononuclear cells but no polymorphs, and large numbers of Alazarin red positive particles.

(5) The radiological evidence of destructive changes, including collapse of the medial tibial plateau of the left knee. Pyrophosphate as well as apatite crystals had been aspirated from this joint but not from the shoudler.

Discussion points included:

(1) The case was thought to be in some ways similar to those described as 'Milwaukee shoulder' (see Schumacher p. 54 and Watt, p. 73). However, it was obvious from this and other reported cases that the site of involvement was not specific, and that other calcium phosphate crystals might be associated with these types of cases. The term 'apatite-associated large joint lysis' was suggested by the presenters for cases of this sort.

(2) The possible mechanism of the destructive changes was also discussed. Some participants felt that a primary disorder of subchondral bone could not be ruled out, and that crystals could be a marker of disease, rather than a cause of these extraordinary changes.

CASE 7

A 65 year old woman with an 18 year history of calcinosis cutis, chiefly affecting the right index finger. She had presented with SBE in 1980, resulting from infection round discharging areas of calcification in the fingers.

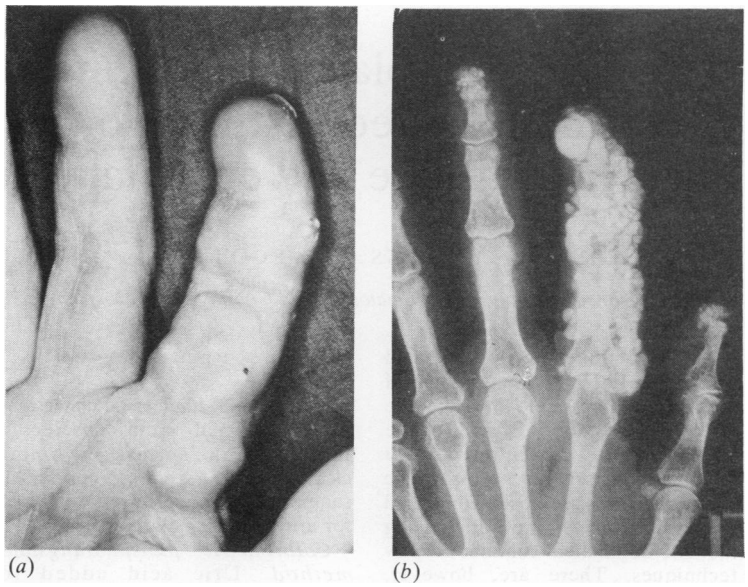

Fig. 5 *Clinical picture and radiograph of the right index finger (case No. 7).* (a) *Clinical photograph showing extensive nodular swelling of the finger.* (b) *Radiograph showing the masses of non-trabeculated calcific bodies in the soft tissues, with normal bones and joints. Carbonated apatite was identified in the material extracted from these deposits.*

The points noted included:

(1) The gross calcium deposits in the finger with characteristic radiological features of rounded non-trabeculated deposits (Fig. 5).

(2) Material often discharged, analysis of a large amount by infrared spectrophotometry showed apatite with some carbonate.

(3) There was no clinical evidence of any associated connected tissue disorder or of any metabolic abnormality which might help explain the deposition.

(4) Radiological screening had shown an associated area of calcification in the spine, and on direct questioning, the patient had admitted to back pain isolated to that area.

Discussion highlighted a considerable interest in such a gross example of para-articular and soft tissue crystal deposition.

(1) The lack of inflammation around the lesion, except at times of discharge through small ulcerated skin areas was also noted and discussed.

(2) Although thought to be of considerable interest, this case highlighted the lack of understanding available to explain the formation and effect of huge crystal deposits such as these.

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