Case report

Coexistent rheumatoid arthritis and tophaceous gout: a case report

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SUMMARY Rheumatoid arthritis and gout are both common rheumatic diseases, but their coincidence is rare. We report the case of a 67-year-old Caucasian woman with rheumatoid arthritis who later developed tophaceous gout. The tophi disappeared with remarkable rapidity on treatment with allopurinol.

Case report

A married woman aged 67 was first referred to our department in May 1972 with a history of pain in many of her joints with increasing morning stiffness in her hands. General examination and systemic review were normal. Both ulnar styloids were

prominent, and there was synovial thickening of the index and middle metacarpophalangeal (MCP) joints in each hand. There was a small effusion in the right knee and periarticular thickening at each ankle. Her feet were normal. Investigations at that time showed an erythrocyte sedimentation rate (ESR) of 77 mm/1 h (Westergren), positive tests for rheumatoid factor with a Rose-Waaler titre of 1/256, and a normal serum uric acid of 0.29 mmol/l. X-rays of the hands showed periarticular osteoporosis at both carpi and small erosions at the left ulnar

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Fig. 1 Gouty tophi in the fingers at presentation. The left middle finger was the site of biopsy for polarised light microscopy.

Fig. 2 After seven months' treatment with allopurinol the tophi have completely disappeared.

styloid, the left thumb carpometacarpal joint and the left little MCP joint, while those of the feet showed erosion of the left second metatarsophalangeal (MTP) joint. She was treated with enteric-coated phenylbutazone 100 mg t.d.s. with the later addition of enteric-coated aspirin 3.25 g daily. In February 1974 she had a flexor synovectomy of the index and middle fingers of the left hand. Subsequent follow-up was undertaken by her general practitioner.

In March 1979 she was referred again with 'a multiplicity of nodules on the fingers causing her considerable pain and reducing her dexterity' (Fig. 1). These nodules had developed over the preceding 7 months and clinically appeared to be tophi rather than rheumatoid nodules. Polarised light microscopy of the contents of one of them confirmed the presence of abundant urate crystals. Other investigations showed her ESR was 91 mm/h (Westergren), rheumatoid factor positive with a Rose-Waaler titre of 1/128, and serum uric acid 0.95 mmol/l. Further studies of her urate status were undertaken and showed serum urate levels of 1.0, 1.0, and 0.94 mmol/l on 3 consecutive days. Creatinine clearance was 36 ml/min, and 24-hour urate excretion was 2.72 mmol/l. Fresh X-rays of the hands did not reveal any progression of the erosive changes noted earlier. In the feet she had developed more widespread erosions with marked destruction of the metatarsophalangeal joint of the left big toe. Her aspirin was stopped, and she was given 300 mg of allopurinol daily in addition to her phenylbutazone. The latter was discontinued after a few months. On review in

November 1979 serum urate estimations were 0.26, 0.24, and 0.23 mmol/l respectively on 3 consecutive days, and the tophi in her hands had all disappeared (Fig. 2).

Discussion

The coexistence of rheumatoid arthritis and gout is very rare. The first well authenticated case was reported as recently as 1966,¹ with further single case reports in 1979 associated with a comprehensive review of the literature,² and in 1980.³ Our own patient presented with seropositive erosive arthritis which satisfied the American Rheumatism Association criteria for classical rheumatoid arthritis.⁴ Her serum urate was normal at that time, but when she reappeared with extensive 'rheumatoid nodules' 7 years later she was floridly hyperuricaemic, though she had never experienced an acute attack of gout.

This is only the fourth report of the unequivocal association of gout and rheumatoid arthritis, despite the calculation by Wallace *et al.*² that there should be about 10 000 cases in existence. The reason for this paucity of cases remains obscure. Lussier and de Medicis⁵ found that hyperuricaemia appeared to exert an immunosuppressive effect on adjuvant arthritis in rats. They suggested that this effect might prevent the expression of a rheumatoid polyarthritis in man, though this effect would not explain the absence of gout in patients such as ours with pre-existing rheumatoid arthritis. Analgesic nephropathy, which is probably commoner than many rheumatologists believe,⁶ may affect the renal

handling of uric acid and precipitate hyperuricaemia, which it has been estimated⁷ occurs in 10% of patients with rheumatoid arthritis. Although doses of aspirin over 4 g daily promote uric acid excretion by inhibiting tubular resorption, lower doses suppress tubular secretion of urate and hence produce hyperuricaemia.⁸ This would seem a plausible mechanism in our own patient, as she not only became normouricaemic very rapidly once her aspirin was stopped and a modest dose of allopurinol introduced but also experienced very rapid resolution of her multiple tophi. We could identify no reason why this mechanism should have been so potent in our patient whereas a similar development of tophaceous gout is virtually unreported in the thousands of apparently similar patients with rheumatoid arthritis exposed to the same drug regimen.

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