Coexistent tophaceous gout and ankylosing spondylitis

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There has recently been considerable interest in the rare coexistence of gout and other inflammatory arthridites.[1-3] We report the first case of tophaceous gout and anklyosing spondylitis.

CASE REPORT

A 62 year old man was admitted in June 1982 with persistently active polyarticular tophaceous gout and pronounced pedal oedema due to congestive cardiac failure.

The first attack of right podagra in 1969 was followed by progression to a symmetrical chronic arthritis, initially affecting only the lower limbs, and he was treated with various non-steroidal agents as well as colchicine. In 1976 arthritis developed in several predominantly distal interphalangeal joints of both hands associated with tophi. Allopurinol was added to his treatment regimen, resulting in exfoliative dermatitis. Upper limb involvement progressed to wrists, elbows, and shoulders, sparing all metacarpophalangeal joints. Further symptomatic deterioration followed a myocardial infarction in 1978 with the introduction of diuretic treatment. There was persistent hyperuricaemia and enlargement of tophi. He was placed on high doses of probenicid, which was changed to sulphinpyrazone after a poor response.

In 1965 he had suffered from low back pain radiating to his thighs. Although symptoms were attributed to mechanical back pain and a corset was prescribed, review of the radiographs showed bilateral sacroiliitis. Pain improved over the next 18 months and axial symptoms had only recurred in the past few years, within neck stiffness and a stooped posture.

On examination he was overweight with a pronounced dorsal kyphosis. Movements of the cervical and lumbar spine were considerably limited. Chest expansion was 1 cm. Most joints except metacarpophalangeal and hip joints showed chronic inflammatory arthritis. The interphalangeal joint of his right thumb was acutely inflamed. Tophi were present on several digits and on the pinna of the ears. There was cardiomegaly but no evidence of aortic incompetence or heart failure. Pitting oedema to his knees was associated with eczematous skin changes.

X-ray films of the hand showed tophaceous gouty arthritis and the sacroiliac joints showed typical changes of progressive ankylosing spondylitis. Before admission the serum uric acid was persistently raised—for example, 0·778 mmol/l—at admission it was 0·484 mmol/l. 24 hour urate excretion was 4·8 mmol (3·5–4·2 mmol) without purine restriction and taking sulphinpyrazone 200 mg four times daily. Blood urea was raised (9·4 mmol/l (2·5–6·7 mmol/l)) and creatinine clearance decreased (45 ml/min (105 ml/min)). He was HLA-B27 positive.

The absence of previous reports of the coexistence of these two diseases is surprising considering their high incidence in men, their relative frequency in the population and the fact that neither joint involvement nor serological examination would result in the diagnosis of one condition in preference to the other.

References

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Normal response to monosodium urate (MSU) crystals by patients with rheumatoid arthritis

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It has been suggested that the negative correlation between rheumatoid arthritis and gout might result from inability of patients with rheumatoid arthritis to respond to monosodium urate (MSU) crystals, perhaps due to crystal coating by rheumatoid factors with subsequent exclusion from interaction with cellular and non-cellular mediators of inflammation.[1] To test the validity of this hypothesis we investigated the