

Case report

Osteonecrosis at multiple sites in a patient with systemic lupus erythematosus

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SUMMARY Osteonecrosis of multiple joints is described in a patient with systemic lupus erythematosus.

Key word: avascular necrosis.

A 50-year-old woman presented in 1971 with seronegative, symmetrical, peripheral arthritis, a rash, dyspnoea with left subcostal pain, proximal myopathy, neutropenia, and thrombocytopenia. Tests for rheumatoid factor were negative but those for antinuclear factor and DNA binding were strongly positive. A diagnosis of systemic lupus

erythematosus (SLE) was made with five positive criteria for SLE.¹

Corticosteroid therapy was begun in 1972 and large doses were used: 20–60 mg daily at first, followed by maintenance therapy of 15 mg per day. Azathioprine was introduced in 1973 at a dose of 100 mg per day.



Fig. 1 *Osteonecrosis of left ankle (note left tibial periosteal reactions).*

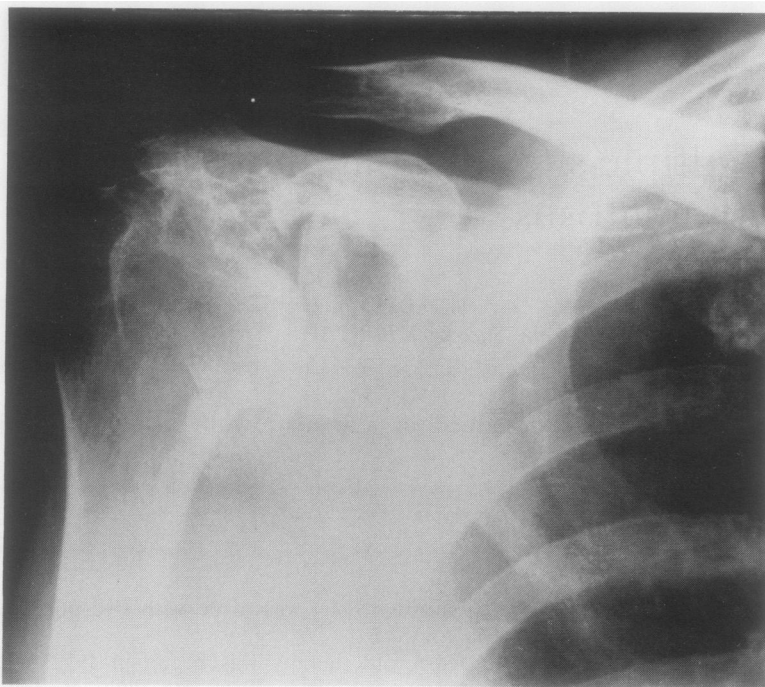


Fig. 2 Osteonecrosis of right humerus and glenoid articulating surface.

On presentation to this Unit in 1982 the patient had continuing arthritic pain, and routine radiological assessment showed twelve osteonecrotic lesions: left 3rd and 4th metacarpal heads, right 4th metacarpal head, proximal row of carpal bones bilaterally, particularly involving both lunates, both-medial femoral condyles, left talus (Fig. 1), right humeral head, right glenoid articulating surface (Fig. 2), and left humeral head.

Discussion

The association of osteonecrosis with SLE was described in 1960 by Dubois and Cozen² and further documented in numerous publications.³⁻⁶ The most commonly reported incidence is approximately 5-6% but with estimates as high as 40%,⁷ and, although the femoral head is the most typical site of abnormality, other and multiple sites have been reported.⁸

Ruderman and McCarty⁸ report a case with osteonecrosis involving six joints, but such multiple joint involvement is uncommon, and the case of our patient with 12 osteonecrotic lesions was therefore considered to be worth reporting.

Osteonecrosis in SLE is usually, though not invariably, associated with high dose corticosteroid

therapy. Other possible pathogenic factors, either singly or in combination, include increased activity of previously weakened bone during remission, vasculitis and capillary fat globules.

References

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