

Case report

Monarticular rheumatoid-like arthritis of seven years' duration following fracture of the radial head

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SUMMARY We report a patient who developed rheumatoid-like arthritis in an elbow joint following a fracture. The arthritis has remained localised to that joint for seven years.

Case report

In August 1978 a previously fit 32 year old woman fell from a ladder and injured her right elbow. x Rays showed a comminuted fracture of the radial head (Fig. 1). She was treated with immobilisation in a plaster of Paris back-slab for three weeks followed by physiotherapy.

Initial progress was satisfactory, but the elbow never returned to normal, and four months later she complained of increasing pain on the lateral aspect of the elbow. When seen in February 1979 there was swelling of the lateral epicondylar region with a 30° fixed flexion deformity. Radiographs showed that the radial head had united in a good position.

Pain, swelling, and stiffness of the elbow persisted with synovial swelling, persistent effusion, and radiological erosion of the lateral epicondyle of the elbow (Fig. 2). At this stage, the erythrocyte sedimentation rate (ESR) varied between 11 mm and 32 mm/1st h. The latex fixation was negative. In view of these developments exploration of the lateral aspect of the elbow was undertaken in March 1981. At operation the synovial membrane was found to be markedly thickened and about 20 ml of synovial fluid was present. Articular surfaces of the capitellum and radial head were smooth and of normal reflective appearance. A synovial biopsy specimen was taken.

Histological examination of the excised material (EGLB) showed the two pieces of synovial membrane each to have undergone synovial cell hyperplasia with villus formation. There was practically

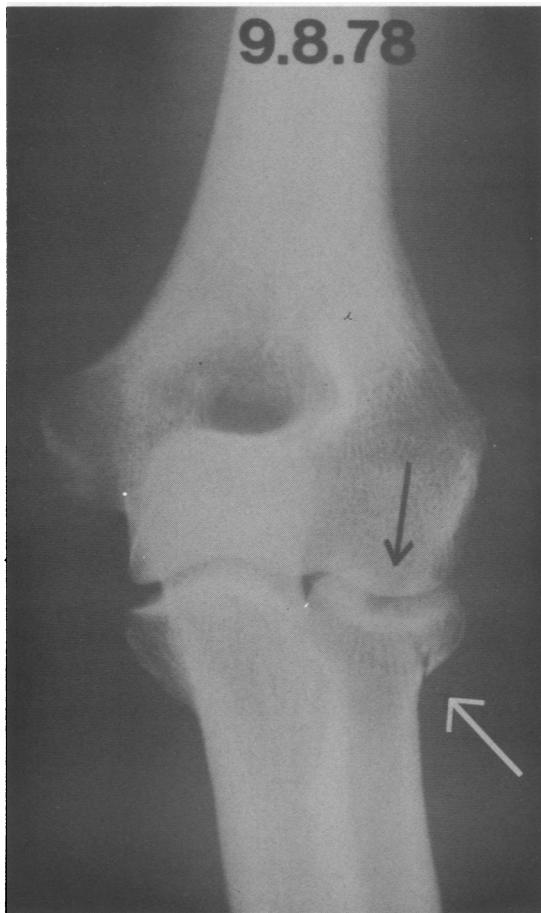


Fig. 1 AP view of right elbow showing fracture of radial head (arrowed).

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Fig. 2 AP view of right elbow showing erosion of the lateral epicondyle.

no fibrin and no giant cells, but the synovial layer did contain some polymorphs. The subsynovial layer was packed with collections of plasma cells surrounding lymphocytic foci: the latter did not include germinal centres. There were also polymorphs in the subsynovial layer but not in excess of those often found in acute rheumatoid arthritis. The process appeared to be of some duration because there was subsynovial thickening and fibrosis with an increase in the number of blood vessels. There were, however, no signs of trauma or bone erosion, i.e., no macrophages containing haemosiderin and no fragments of cartilage or bone. The appearance was felt to be typical of active acute rheumatoid arthritis. There was no histological evidence of infection or a post-traumatic synovitis (Fig. 3). Culture of the synovial membrane for acid fast bacilli was negative.

In May 1981 she noticed swellings in the right axilla and an excision biopsy confirmed the presence of three lymph nodes, the largest being 3×1.5 cm. All showed florid reactive lymphoid hyperplasia with some fat and haemosiderin pigment in their medullary spaces. No acid fast bacilli were seen and cultures were negative.

Since then the right elbow has remained her only problem. She has been treated with soluble aspirin with some relief. Her right elbow is now stiff for one hour each morning. There is soft tissue swelling laterally and a range of 70° – 120° , but pronation and supination are full. The muscles in her upper arm are wasted. Other joints and her spine are clinically normal. There are no extra-articular features of

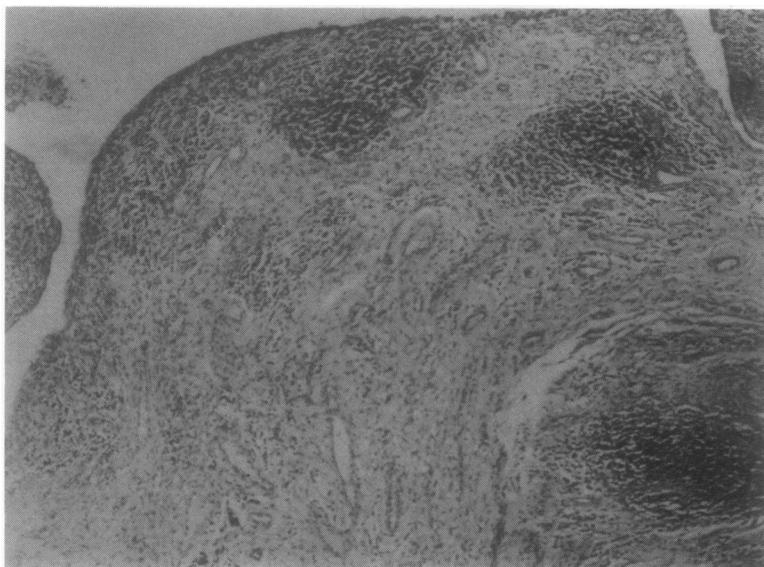


Fig. 3 Synovial membrane showing lymphocytic foci. (H and E).

rheumatoid arthritis. Current investigations show a haemoglobin of 12.2 g/dl (122 g/l), ESR 10 mm/1st h, and latex test, antinuclear antibody test, and HLA-B27 negative.

Discussion

Trauma has been postulated as a precipitation factor in the development of rheumatoid arthritis,¹ psoriatic arthritis,^{1,2} and Reiter's disease.^{3,4} Jacoby and colleagues found that 6% of patients with early rheumatoid arthritis gave a history of recent trauma.⁵ Julkunen and coworkers followed up 270 patients who were treated for trauma in an intensive care unit and found that 2.9% developed rheumatoid arthritis in the next two years, while none of the controls did so.⁶ On the other hand, there appears to be no definite evidence that ankylosing spondylitis is related to previous trauma.⁷ These few reports suggest that trauma may sometimes 'trigger' inflammatory arthropathy, but that this is relatively rare. If trauma can trigger immunologically mediated types of arthritis then this is clearly of interest conceptually and also of importance medicolegally. Individual reports are inevitably anecdotal, but it seems worth recording such cases in order to accumulate information.

The differential diagnosis in this patient must include tuberculosis and atypical mycobacterial infections. The negative biopsies and cultures and the subsequent progress in the absence of definitive treatment, however, must make these diagnoses

unlikely. It may be of interest to note that a history of trauma has been obtained in 45% of patients with atypical mycobacterial infections.⁸

The present case appeared to evolve directly from a traumatic intra-articular fracture into a rheumatoid-like (but seronegative) arthritis in the same joint. This has persisted as monarticular arthritis for seven years.

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