

Destructive arthritis of the shoulder triggered by trauma in a patient with polymyositis

Sir,

Arthritis or arthralgia occurs in 25-50% of patients with polymyositis (PM). However, the articular abnormalities are usually mild and without radiographic changes, erosion or deformity. These articular symptoms generally occur early in the course of PM. We report an atypical case of destructive arthritis of the shoulder following trauma in a patient with well-documented PM.

A 52-year-old Japanese male had a three-year history of PM. In 1995, he complained of proximal muscle weakness and polyarthralgia. He was diagnosed with PM on the basis of elevated creatine phosphokinase and aldolase levels, myogenic changes on electromyography, and muscle biopsy findings. RF, ANA and Jo-1 antibodies were negative. He received medical treatment with low dose corticosteroids for 2 years. He did not exhibit severe arthritis or joint destruction.

In April 1997 our patient bruised his right shoulder during a fall. A few days after the injury, he complained of painful swelling and impaired active motion in his right shoulder. Radiography showed no abnormal findings such as erosion, sclerotic changes or periarticular calcific deposition. The acute phase reactants CRP and ESR were elevated (CRP: 2.0 mg/dl, ESR: 36 mm/hr). Despite medical treatment with corticosteroids and NSAIDs, the arthritis continued in the shoulder. The patient complained of a disturbed sleeping patterns due to the shoulder pain.

Eight months after the injury, radiography showed severe glenohumeral joint destruction. A total shoulder arthroplasty was therefore carried out in November 1997. During the operation, severe synovitis and destruction of the articular surfaces were found. Histopathological findings at the humeral head indicated subchondral lymphoid infiltration (Fig. 1). No calcific crystals were detected in the joint fluid or synovium. After the operation, the shoulder pain disappeared. However, the patient's active range of motion did not improve sufficiently due to muscle weakness. In the one-year follow up period, no loosening or dislocation of the prosthesis was observed.

Articular manifestations are generally mild, with no radiographic changes, in PM patients. However, some previous studies have described severe erosive and deforming arthritis in the hands due to periarticular calcification that has been identified as hydroxyapatite (1, 2). The same pathogenesis is probably involved in apatite-associated destructive arthritis (3). The "Milwaukee shoulder" syndrome, which is caused by hydroxyapatite crystals, was defined as a combination of the elevation and collapse of the humeral head, a massive cuff tear, and damage to the glenohumeral joint with blood-stained effusions in elderly patients, predominantly females (4). In our case, periarticular calcific deposition was not seen on radiography, and neither the joint fluid nor the synovium contained calcium crystals.

Moreover, the diagnosis of Milwaukee shoulder syndrome or steroid-induced avascular necrosis were not supported in our case by the histopathological findings at the humeral head, which showed subchondral lymphoid follicles indicative of RA. Other cases of rap-

idly developing destructive arthritis following trauma in patients with rheumatic diseases have been reported (5-7). Although the pathogenesis of this phenomenon has not been elucidated, the exposition of self antigens after trauma could lead to an autoimmune reaction and, eventually, arthritis and joint destruction.

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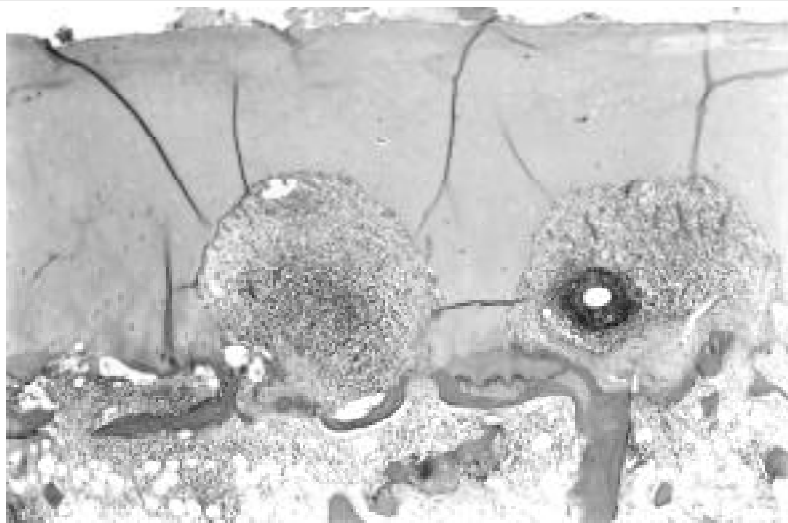


Fig. 1. Marked chronic inflammatory infiltrates, in the form of lymphoid follicles, can be noted in the subchondral bone (hematoxylin and eosin stained).