Stress fracture of base of the acromion

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Ann Rheum Dis 2002;**61**:944–945

Fractures of the scapula occur infrequently, with a fracture of the acromion being an even rarer entity. Acromial fractures constitute 9% of fractures of the scapula, which amounts to 3–5% of shoulder girdle injuries.¹ Fractures of the acromion are generally secondary to trauma, with only a few cases of stress fracture having been reported.^{2–5}

Reports published in English have described stress fractures of the acromion at the base of the acromion extending to the spine of the scapula,² neck of the acromion,³ medial aspect of the acromion,⁴ and the base of the acromion only.⁵ These cases occurred in young to middle aged patients and were associated with a single violent muscle contraction or repetitive subcritical load to the shoulder. We present a case of an atraumatic osteoporotic stress fracture at the base of the acromion associated with chronic rotator cuff tear arthropathy.

CASE REPORT

The patient, an 82 year old woman, was admitted to the medical ward with heart failure and varicose ulcers. She was referred to the orthopaedic department because of a three day history of left shoulder pain without trauma. She was mostly wheelchair bound but did walk indoors with a Zimmer frame.

Examination showed mild tenderness and crepitus at the base of the acromion where a fracture gap could be palpated. Active shoulder movements were restricted to 90° of both flexion and abduction. She also had a senile osteoporotic kyphosis of her thoracic spine. Anteroposterior and axillary radiographs of the shoulder showed a displaced fracture of the base of the acromion with superior subluxation of the head of the humerus, suggesting chronic rotator cuff tear arthropathy (figs 1 and 2). A radiograph of her spine confirmed the osteoporotic kyphosis of the thoracic vertebrae. Blood tests including full blood count, erythrocyte sedimentation rate (ESR), bone biochemistry, and myeloma screen were normal. She was treated conservatively in a broad arm sling with gradual mobilisation as her pain subsided. She recovered nearly full movements of her shoulder in four weeks. A follow up x ray examination at six months showed a non-union of the fracture, which was not painful.

Figure 1 Anteroposterior view of the right shoulder showing fracture of the base of acromion.

Figure 2 Axillary view of the right shoulder showing fracture of the base of the acromion.

DISCUSSION

Osteoporotic fractures without a history of trauma usually occur in the legs, pelvis or spine and rarely in the arms. Repetitive subcritical trauma or single violent muscular pulls have occasionally been associated with fractures of the acromion.^{1–5} Of the previous four cases of a similar type of injury reported, two were professional sports players,⁵ one was a car mechanic who felt a sudden crack in his shoulder while applying torque with a screwdriver,³ and the fourth was a woman playing golf, who suddenly felt acute pain in her shoulder as she hit the ball.² It was not possible to correlate our patient's injury with any trauma, indeed she recalled waking up in the morning with sudden pain, which gradually got worse. Dennis *et al* reported three cases of stress fracture of the anterior impingement zone of the acromion.⁶ All three patients had severe rotator cuff arthropathy and two had steroid dependent rheumatoid arthritis.⁶ Surgical excision of the fragments was carried out in all three of his patients when conservative treatment had failed, with one patient eventually requiring shoulder replacement. Pain improved in the other two patients, but there was no improvement in the range of motion. All the fractures reported united after conservative treatment except in the series reported by Dennis *et al*.⁶

It is sometimes difficult to visualise this fracture without adequate penetration of radiographs in the appropriate plane. These fractures are best visualised in the axillary view in our opinion (fig 2). All previous cases have been either linear undisplaced fractures or diagnosed by an isotope bone scan. One case did show sclerosis at the base of the acromial arch on x ray examination, which on subsequent bone scan was confirmed to be a fracture.⁵ We believe that abnormal pressure from the humeral head on the acromion, due to the rotator cuff arthropathy, leads to excessive movement at the fracture site causing a non-union of the fractured acromion.

Shoulder pain is a common complaint in elderly people. A stress fracture of the acromion should be considered in patients with chronic rotator cuff tear arthropathy who

have osteoporosis and whose shoulder pain increases spontaneously. Routine use of good quality axillary radiographs in such patients should lead to a higher rate of diagnosis of such injuries.

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Accepted 4 April 2002

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α_1 Antitrypsin deficiency in a patient with systemic vasculitis and primary Sjögren's syndrome

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Ann Rheum Dis 2002;**61**:945-946

The internal homeostasis in man is critically dependent on regulation of proteolytic enzymes in tissues and fluids by endogenous inhibitors. α_1 Antitrypsin is the most abundant protease inhibitor (Pi) in plasma, and controls tissue degradation by proteases such as trypsin, neutrophil elastase, and proteinase 3.¹ Homozygous α_1 antitrypsin deficiency is known to predispose to emphysema and chronic liver disease.^{2,3} Recently, a strong correlation has been found between systemic small vessel necrotising vasculitis and both heterozygous and homozygous α_1 antitrypsin deficiency.⁴ Also, such deficiency has been found to confer a more disseminated disease and worse prognosis to patients with antineutrophil cytoplasmic antibody (ANCA) positive vasculitis.⁵

However, there is disagreement about the clinical implication of an intermediate α_1 antitrypsin deficiency: is it an accidental finding or does it imply susceptibility to autoimmune disease? Thus, reports have demonstrated an increased incidence of α_1 antitrypsin deficiency in patients with acute anterior uveitis, a finding that was refuted by others.⁶ Similarly, several reports have suggested an increased frequency of α_1 antitrypsin deficiency in patients with rheumatoid arthritis, whereas others found no such association in similar patients.⁷

CASE REPORT

Here, we report a severe vasculitic episode in a patient with primary Sjögren's syndrome (pSS) and heterozygous α_1 antitrypsin deficiency.

The patient, a white woman, was diagnosed with pSS at the age of 44 years. The diagnosis fulfilled the Copenhagen and the European community classification criteria for pSS.^{8,9} Five years after the start of sicca symptoms, arthritis suddenly appeared in the knee and ankle joints, as well as purpuric eruption on the lower legs. Laboratory studies showed a raised erythrocyte sedimentation rate (ESR 78 mm/1st h) and C reactive protein (CRP 42 mg/l; normal <10 mg/l) and on urine analysis proteinuria and microscopic haematuria. Oral prednisolone treatment was started, but a week later the patient suddenly experienced headache and loss of vision. Decreased wakefulness and generalised seizures ensued. Computed tomography of the brain showed three small haemorrhagic cortical infarctions, and an eye examination disclosed cortical blindness (complete visual loss with normal light reflexes). Two of three tests for circulating immune complexes were positive, and measurement of

complement factors C3 and C4 showed relative deficiency of C4. C4 isotyping disclosed the phenotype C4A3B1. Positive serological tests (rheumatoid factor, antinuclear antibodies, and anti-SSA/SSB) were unchanged compared with before the acute episode, while an ANCA test and a test for antiphospholipid antibodies were negative.

Intravenous (IV) injection of methylprednisolone and bolus IV cyclophosphamide produced a rapid response and the arthritis disappeared and eyesight gradually improved. Urine findings and ESR/CRP normalised. Oral prednisolone and intermittent IV cyclophosphamide were given for one year.

A retrospective chart review has on two separate occasions shown low plasma levels of α_1 antitrypsin (0.7 and 0.81 g/l, respectively; normal 0.9-1.7 g/l). Also, during the inflammatory episode, α_1 antitrypsin levels remained normal, while other acute phase proteins were raised. Therefore a genetically determined heterozygous α_1 antitrypsin deficiency was suspected. This was confirmed by α_1 antitrypsin phenotyping using monoclonal antibody specific for the PiZ mutant in an enzyme linked immunosorbent assay (ELISA).¹⁰ Isoelectric focusing confirmed the PiMZ phenotype.¹¹ The frequency of PiZ heterozygosity in the Swedish general population is 0.047.

The diagnosis of vasculitis in this patient was not verified by biopsy, but the clinical evidence is compelling. The α_1 antitrypsin deficiency in this patient is possibly related to the severe systemic vasculitis. Low levels of complement factor C4 have been reported in association with deficient elimination of circulating immune complexes in Sjögren's syndrome.¹² In the present case the relative deficiency of C4, in addition to α_1 antitrypsin deficiency, may hypothetically have contributed to an increased risk of immune complex mediated vasculitis.

DISCUSSION

The clinician should be observant about low levels of α_1 antitrypsin in patients with vasculitis because they may indicate a poor prognosis. α_1 Antitrypsin is an acute phase protein and therefore deficiency might be masked by the presence of inflammation. As in this case, normal α_1 antitrypsin levels may be consistent with deficiency of the protein. For definite diagnosis it is essential to perform phenotype identification.¹¹

ACKNOWLEDGEMENT

We thank Dr L Truedsson, Department of Microbiology, University Hospital, Lund, Sweden, for performing the C4 isotyping.