Enthesis of the ligamentum teres during anklyosing spondylitis: histopathological report

We were interested to read the article by Inoue et al.1 on ossification of the medial acetabular floor, including the ligamentum teres as a possible mechanism of lateral subluxation in coxopathy associated with anklyosing spinal hyperostosis. A few years ago we saw a patient suffering from anklyosing spondylitis (AS) with right coxitis including inflammatory enthesiopathy of the ligamentum teres (ligamentum capitis femoris). A 57 year old man with a five year history of a painful right hip was first seen at the rheumatology clinic in 1976, when the diagnosis of AS was made (HLA B27 was present). Anteroposterior radiography of the pelvis demonstrated joint space narrowing in the right hip, sclerosis and new bone formation on the right acetabular rim, enthesophytes of the femoral head, and an ankylosis in the sacro-iliac joints. Due to persistent pain and major disability, an arthroplasty was performed on the right hip.

On opening the capsule, the synovium was inflated around the ligamentum capitis femoris although there was no obvious pannus the articular cartilage was considerably ulcerated at the femoral head. Histological examination showed an intense vascular proliferation in the central area of attachment of the femoral head ligament to the fovea capitis, especially in the deep subchondral perilunar area (figure). Lymphoplasmocytic cells had spread along the proliferating vessels which dissociate the vertical collagen fibres at the ligamentum teres attachment. In the narrow spaces surrounding the damaged area there was evidence of chondrocyte proliferation. Ossifying enthesitis was also present on the attachment of the capsule in the acetabular labrum.

Published reports are scant concerning the histopathology in the early stage of AS. Nevertheless, several papers have underlined the role of enthesis in the pathogenesis of AS and related spondylarthopathies.2 4 Ossifying enthesopathies on the great trochanter and capsular enthesophytes are common features in AS5 and Forester's disease6 with the resulting tendency to ankylosis of the hip. Enthesopathies include all changes, whether traumatic, degenerative, metabolic or inflammatory of enthesis.8 9 As enthesiopathies usually produce ossification, they are very useful diagnostically and nosologically, especially when they are diffuse or multiple.1 The most extreme example of metabolic enthesopathy is diffuse idiopathic skeletal hyperostosis (DISH, more commonly known as Forester's disease). Conversely, AS, which is the archetype of spondylarthopathies, is the more classic inflammatory enthesopathy.

The most distinctive feature of the natural history of ossifying enthesiopathies, suggests that during AS and DISH, enthesophyte of the ligamentum capitis femoris, may initiate intra-articular involvement, as observed in the knee with cruciate ligaments.3 The presence of an intra-ligamentary artery probably enhances its proclivity to promote coxitis during AS.

Plasma viscosity in giant cell arteritis

We read with great interest the paper by Gudmundsson et al.1 It was interesting that at follow up there was evidence that plasma viscosity and the erythrocyte sedimentation rate (ESR) parallelled clinical findings and predicted flare ups better than other variables.

In previous studies, we have tried to establish a definition of biological parameters for monitoring giant cell arteritis (GCA) and polymyalgia rheumatica (PMR).2 Our studies have shown that in patients with GCA or PMR there is a positive correlation between ESR, haptoglobin and orosomucoid during the acute phase of the disease before treatment.

Under corticosteroid treatment, a correlation persists between clinical symptoms and ESR. But a discrepancy exists between ESR and acute phase proteins: haptoglobin and orosomucoid. These two proteins remain elevated while clinical symptoms and ESR have returned to normal. In individual cases this elevation is linked to a persistence of the disease and could lead to a possible flare up when attempting to taper corticosteroid treatment. Moreover, an elevation of orosomucoid and haptoglobin can signal a clinical flare up.

Our data suggest that acute phase protein elevation that is present in spite of improvement in clinical symptoms and ESR is a contraindication for tapering corticosteroid doses or for withdrawing treatment.