

MATTERS ARISING

Enthesitis of the ligamentum teres during ankylosing 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 ankylosing spinal hyperostosis. A few years ago we saw a patient suffering from ankylosing spondylitis (AS) with right coxitis including inflammatory enthesopathy 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 inflamed 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 perivascular 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 enthesitis in the pathogenesis of AS and related spondylarthropathies.²⁻⁴ Ossifying enthesopathies on the great trochanter and capsular enthesiophytes are common features in AS⁵ and Forestier's disease⁶ with the resulting tendency to ankylosis of the hip.

Enthesopathies include all changes, whether traumatic, degenerative, metabolic or inflammatory of enthesitis.^{7,8} As enthesopathies usually produce ossification, they are very useful diagnostically and nosologically, especially when they are diffuse or multiple.⁵ The most extreme example of metabolic enthesopathy is diffuse idiopathic skeletal hyperostosis (DISH, more commonly known as Forestier's disease). Conversely, AS, which is the archetype of spondylarthropathies, is the more classic inflammatory enthesopathy.

The most distinctive feature of the natural history of ossifying enthesopathies, suggests that during AS and DISH, enthesopathy 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.

P GILLET
P PERÉ
J Y JOUZEAU
J FLOQUET
A GAUCHER
Departments of Rheumatology and Pathology,
URA CNRS 1288, Faculté de Médecine, BP 184,
54505 Nancy,
France

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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) paralleled 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).^{2,3} 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.

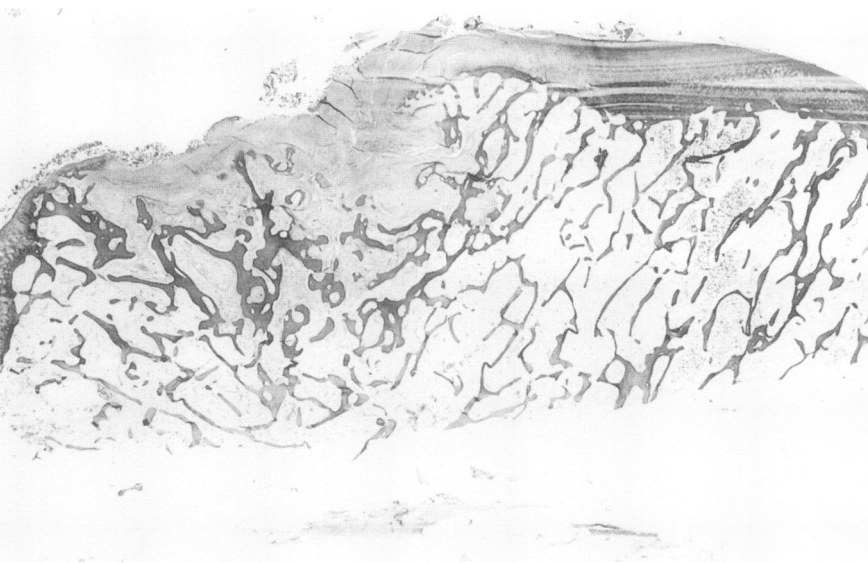
Under corticosteroid treatment, a correlation persists between clinical symptoms and ESR. But a discrepancy exists between ESR and acute phase proteins: haptoglobin and orosomucoid. These two proteins remain elevated while clinical symptoms and ESR have returned to normal. In individual cases this elevation 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.

J B PAOLAGGI
Service of Rheumatology,
Hôpital A. Paré, 9 avenue Charles de Gaulle, 92100
Boulogne, France

D CHAOUAT
Service of Internal Medicine, Fondation A de
Rothschild, 25-29 rue Manin, 75019 Paris, France

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Histological specimen of femoral head showing enthesitis of the ligamentum teres with subchondral osteitis (haematoxylin and eosin stain).