
Leucopenia after gold and sulphasalazine treatment

Sir: I read the interesting report by Bliddal and colleagues of three patients who developed leucopenia after both gold and sulphasalazine treatment. HLA-DR3 is a marker for toxic reactions after treatment with gold or d-penicillamine could not be shown. Patient 1, however, was apparently a carrier of HLA-B7. It is well known that female patients with rheumatoid arthritis, who are HLA-B27 positive, have a significantly higher risk for drug induced agranulocytosis, not only after treatment with leva misole but also with other antirheumatic drugs. Thus under these conditions the possibility should be considered that in this patient, in the absence of HLA-D3, HLA-B27 may be the risk indicator for her relapsing leucopenia.

K L SCHMIDT
Department of Rheumatology and Physical Medicine
University of Giessen
Ludwigstrasse 37-39
D 6350 Bad Nauheim
West Germany


Sir: Thank you for your interesting comments on our article about HLA types of patients with leucopenia.

Our patients all had classical rheumatoid arthritis and none was positive for antinuclear antibodies. The observation in a small group of patients of HLA-B27 in just one case does not justify further conclusions.

Like many others we no longer use leva misole treatment for patients with rheumatoid arthritis.

HENNING BLIDDAL
Department of Rheumatology
Bispebjerg Hospital
DK-2400, Copenhagen NV
Denmark

Destructive spine lesions in ankylosing spondylitis

Sir: We read with interest the article by Audefurmarn entitled ‘Pathogenesis of square bodies in ankylosing spondylitis’. In this case report of a patient with ankylosing spondylitis the author provided histopathological evidence to suggest a primary acute and chronic inflammatory lesion, resulting in destruction of the vertebral bodies, followed by new bone formation.

It might be interesting to recall our case of destructive cervical vertebral lesions showing ankylosing spondylitis in a 19 year old woman. During her disease of 18 months’ duration she had inflammatory pain of the lumbar spine, sacroiliac joints, knees, and heels; radiographs showed bilateral sacroilitis; HLA typing was A3, B7, 12 and erythrocyte sedimentation rate 60 mm/1st h. From the start of her disease she had inflammatory pain of the cervical spine. Radiographs showed severe destructive lesions of the 6th cervical vertebra without discal lesions or cervical ankylosis, and a milder anteroposterior spondylitis of the 5th cervical vertebra (figure). There was no history of trauma. Surgical biopsy excluded a tumorous or infectious process. Spinal fusion (C4-7) was performed, and cervical pain disappeared with non-steroidal anti-inflammatories.

Histopathological examination of the 6th cervical vertebra biopsy specimen showed the absence of any inflammatory cells and the presence of spongiosa surrounded by fibrous tissue.

These findings are consistent with those of Audefurmarn; the inflammatory cervical pain, which was noted as an early symptom of the disease in our patient, might have been due to the inflammatory lesion of the vertebral body, and the present histopathological findings might represent the reparative stage with scar and new bone formation. It is noteworthy that this severe destructive cervical lesion was the first manifestation of ankylosing spondylitis in this patient.

A KAHAAN
C J MENKES
Service de Rhumatologie A
Hôpital Cochin
27 rue du Faubourg Saint-Jacques
75014 Paris, France

Low incidence of antinuclear antibodies in dermatomyositis with malignancy

Sir: In 1958 Walton and Adams first classified ‘polymyositis/dermatomyositis with malignancy’ as a distinct subset of polymyositis. Recent prospective controlled studies, however, cast doubt upon the increased incidence of malignancy in polymyositis/dermatomyositis. If the association of polymyositis/dermatomyositis with malignancy happens by mere coincidence, serological features in these diseases with and without malignancy should not be different. The purpose of our preliminary study was to clarify this point in 36 patients with dermatomyositis.

Dermatomyositis was diagnosed according to the diagnostic criteria of Bohan and Peter. Thirty six patients were diagnosed as having pure adult dermatomyositis. These patients were followed up for a period of time for malignant disease. Patients with dermatomyositis with malignancy (nine definite and three probable dermatomyositis) and 24 patients with dermatomyositis without malignancy (16 definite and eight probable dermatomyositis). Patients with dermatomyositis which overlapped with other connective tissue diseases were excluded from this analysis. Existence of malignancies was proved histologically or from cytology and/or MRI, or both. Antinuclear antibodies were tested by indirect immunofluorescence with HEp-2 cells (Kallstad, Chaska, Mn, USA) as substrates. Serum samples giving apparent fluorescence at a dilution of 1:40 were considered positive. The y2 test was used for the statistical analysis.

Positive antinuclear antibodies were found in 13/24 (54%) patients with dermatomyositis without malignancy, but they were found in only 2/12 (17%) of those with malignancy (p<0.03). The low incidence of antinuclear antibodies in dermatomyositis with malignancy suggests the presence of a different serological background from that in the dermatomyositis without malignancy. This conclusion supports the view that a distinct subset of dermatomyositis exists—dermatomyositis with malignancy.

MASAHIKO NISHIKAI
AKIO SATO
Departments of Internal Medicine
Second Tokyo National Hospital
Tokyo Japan


Inhibition of xanthine oxidase by allopurinol: its lack of effect on models of inflammation

Sir: In response to an article published in the *Annals*, entitled ‘Inhibition of xanthine oxidase by allopurinol: A therapeutic option for ischaemia induced pathological processes’1 we wish to report our findings on the effect of xanthine oxidase inhibition in models of joint