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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.¹ HLA-DR3 as 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-B27. 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 levamisole^{2,3} but also with other antirheumatic drugs.⁴ 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.

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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 levamisole treatment for patients with rheumatoid arthritis.

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Destructive spine lesions in ankylosing spondylitis

Sir: We read with interest the article by Aufdermaur entitled 'Pathogenesis of square bodies in ankylosing spondylitis'.¹ 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

Severe destructive lesions of C6 and anterosuperior spondylitis of C5.

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.² During her disease of 18 months' duration she had inflammatory pain of the lumbar spine, sacroiliac joints, knees, and heels; radiographs showed bilateral sacroiliitis; HLA typing was A3, 9, B7, 12 and erythrocyte sedimentation rate 60 mm/1st h. From the start of her disease she had 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 anterosuperior 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-inflammatory drugs. 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 Aufdermaur; 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.

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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.¹ Recent prospective controlled studies, however, cast doubt upon the increased incidence of malignancy in polymyositis/dermatomyositis.^{2,3} 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.⁴ Thirty six patients were diagnosed as having pure adult dermatomyositis. These patients were further divided into two groups: 12 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 by biopsy or necropsy, or both. Antinuclear antibodies were tested by indirect immunofluorescence with HEp2 cells (Kallstad, Chaska, Mn, USA) as substrates. Serum samples giving apparent fluorescence at a dilution of 1:40 were considered positive. The χ^2 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.

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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