LETTERS TO THE EDITOR

Worsening of Felty's syndrome with methotrexate

Sir: Drs Hughes and Abdulla recently reported a favourable response to low dose oral methotrexate treatment in a patient with Felty's syndrome,1 confirming earlier reports.2 In contrast, we found such treatment failed, with worsening of neutropenia. A 55 year old man had had rheumatoid arthritis since 1980. He had received second line drug treatment, including gold compounds and d-penicillamine, which were discontinued because of proteinuria and inefficacy respectively. Haematological signs of toxicity were not noted with these treatments. A diagnosis of Felty's syndrome was established in 1988 when he developed a splenomegaly associated with polyarthritus and was found to have neutropenia (white blood cells 2.41x10^9/l; neutrophils 0.72x10^9/l). Prednisone treatment 30 mg daily was started, then decreased to 20 mg/day. In December 1989 he presented with acute polyarthritus and low peripheral neutrophils at 1.76x10^9/l. The erythrocyte sedimentation rate was 45 mm/h. Methotrexate 7.5 mg weekly was introduced, together with 15 mg prednisone daily. After one month no marked amelioration of clinical condition was seen, the spleen was unchanged, white blood cells control showed a drop of neutrophils to 1.636x10^9/l (WBC 2.8x10^9/l), platelets 231x10^9/l). Methotrexate was discontinued. Further follow up showed that after one month neutrophils had increased and stabilised. Treatment with corticosteroids was continued all alike.

In this patient methotrexate treatment failed to improve Felty's syndrome, and, on the contrary, induced a transient worsening of neutropenia. His condition was well defined,3 and no previous history of toxic neutropenia was found. He was not receiving any other neutropenic drug. His condition did not apparently differ from those of other patients who improved with methotrexate treatment.4 Our data suggest individual sensitivity to methotrexate treatment in Felty's syndrome, and further studies are needed to define the characteristics which would predict a positive response.

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Lympheodema complicating rheumatoid arthritis

Sir: I read with interest the report of Dacre, showing the dye penetrating the narrow cavity of the radius. This patient's oedema was rather refractory to treatment. I am unaware of this narrow penetration phenomenon being previously reported.

I thank Dr J D Jessop, University Hospital of Wales, Cardiff, for permission to reproduce the radiographs.

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Smoking and back pain

Sir: The recent study by Symmons and coworkers1 confirms previous work showing an association between smoking and back pain. The authors also (for the first time) show a similar association between back pain and oral contraceptives. These two findings might have a common mechanism as both smoking and oral contraceptives lead to disturbances in the flow properties of blood. Blood viscosity is a suitable ex vivo parameter for measuring this. It is roughly 50% higher in non-smokers than in heavy smokers (23.3 (SD 8.0) vs 31.2 (9.7) mPa.s).2 One might therefore speculate3 that this alteration in blood rheology leads to a malnourishment of the highly bradytrophic intervertebral disc, rendering it more vulnerable to injury. This would be an attractive explanation of the fact that these cardiovascular risk factors are also related to back problems. It seems tempting to test this speculation in more detail—for example, by reanalysing some of the numerous epidemiological studies on cardiovascular disease in terms of back problems.

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Rheumatology in Dar-es-Salaam, Tanzania

Sir: May I commend and support the views expressed by Dr Adeabajo.1 During a spell teaching at Muhimbili Hospital, Dar-es-Salaam, Tanzania, I found a dearth of rheumatological experience amidst a wealth of clinical material. The table shows those disorders encountered during two weeks, mainly by scouting through clinics and wards. Good...
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