

the patient relapsed, developing oral and genital ulcers, again requiring treatment in hospital. However, the seric concentration of liver enzymes remained normal and the titre of antibodies did not increase. She received topical treatment and the dose of steroids was increased to 12 mg/day. Three weeks later she was discharged from hospital and, at present, remains clinically stable.

In the present report a correlation between the titre of antibodies to ribosomal P proteins and the development of hepatic disease was seen. The levels of liver enzymes increased and decreased in parallel with the titres of ribosomal antibodies. Correlation between the titres of autoantibodies and other clinical manifestations was not found as the titres remained unchanged despite two acute episodes with important articular and dermatological complications. We do not have a definitive explanation of the relation between hepatic disease and the presence of ribosomal antibodies. However, it has recently been shown that these antibodies may bind to liver membranes in cell cultures.⁸ It may be, therefore, that these autoantibodies cause hepatopathy by binding in vivo to certain proteins in the hepatocyte membrane. Further studies will help to confirm this suggestion.

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Correction

Can methotrexate be used as a steroid sparing agent in the treatment of polymyalgia rheumatica and giant cell arteritis?

van der Veen et al. (*Ann Rheum Dis* 1996; **55**: 218-24)

It is regretted that an incorrect affiliation was given for Dr H J Dinant, who is rheumatologist at the Jan van Breemen Instituut, Amsterdam.