Correspondence

Sulphasalazine and hepatic transaminases

SIR, Subsequent to the letter from Drs MacGilchrist and Hunter¹ we have analysed further our data on the effect of sulphasalazine on transaminases. Of 150 rheumatoid patients receiving sulphasalazine, 104 remained on treatment for at least six months. One of those who discontinued therapy was the patient described by Drs MacGilchrist and Hunter, and one other patient stopped because of nausea and was found to have markedly abnormal liver function tests at the time of stopping. This patient has already been described.²

At the six month visit one patient had a serum aspartate transaminase (AST) concentration of 310 U/l (normal range (NR) 12-48 U/l) and alanine transaminase (ALT) concentration of 282 U/l (NR 3-55 U/l). When tests were repeated two weeks later while still taking sulphasalazine, however, this patient's transaminases were normal, and we suspect a laboratory error. When this patient was excluded from our analysis transaminase levels rose in the remaining 103 patients with a high level of statistical significance (Table 1). In five instances the ALT rose above the normal range to a maximum of 69 U/l and in three cases the AST concentration rose above the normal range to a maximum of 80 U/l. Treatment was continued in all these patients, and follow up for at least two years has shown no clinical sequelae and no further rise in transaminases. Thus it would appear that, in addition to the known hepatotoxicity with sulphasalazine, a small rise in transaminases of no clinical significance commonly occurs and is not an indication that therapy should be stopped.

Table 1 Serum transaminase concentration (median and range) before treatment and after six months' sulphasalazine treatment (n=103)

	Before treatment	After six months' treatment	Wilcoxon p value
AST (U/l)	16 (5-39)	19 (7-80)	<0.001
ALT (U/l)	12 (3-40)	16 (5-69)	<0.001

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Sulphasalazine in rheumatoid arthritis: desensitising the patient with a skin rash

SIR, We were pleased to read the paper by Bax and Amos describing seven rheumatoid patients with a skin rash while receiving sulphasalazine who had been desensitised to the drug.¹ This confirms our original report in 1982 when we desensitised a seronegative rheumatoid patient who responded well to the drug but after four months developed a rash.² After desensitisation using Holdsworth's regimen³ she successfully reached a dose of 1.5 g daily and again had a good therapeutic response. Three months later, however, she developed a more severe pruritic, erythematous, desquamating rash on her limbs, face, and trunk which was accompanied by severe buccal and genital ulceration. She had no oedema or other associated reactions. Skin biopsy was non-specific. The drug was stopped, and the rash and ulcers cleared up.

Since then we have attempted desensitisation in two more rheumatoid patients who developed rashes early in the course of therapy (within the first month). The first patient successfully reached a dose of 1.5 g daily with only occasional minor skin irritation. He responded well to the drug and continues at this dose. The second patient's rash recurred before going on to receive standard tablets (500 mg).

Sulphasalazine is a valuable second line drug in rheumatoid arthritis and desensitisation is a useful procedure for enabling patients responding well to it to continue therapy. We would emphasise, however, that a rash may recur after desensitisation and, as in one of our patients, in a more severe form. Continued observation of these patients is essential.

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