Sulphasalazine treatment in rheumatoid arthritis

Sir: We read with great interest the article by Englert and colleagues on their experiences with sulphasalazine treatment in rheumatoid arthritis (RA).¹ They noted the disappearance of rheumatoid nodules in four patients with RA in eight to 12 weeks after the start of treatment, parallel with a decrease in disease activity.

We also have used sulphasalazine regularly for the treatment of RA since 1983,² but we have not found any favourable effect on the extra-articular manifestations of the disease. After reading Englert's article, therefore, we decided to carry out a trial of sulphasalazine treatment in RA.

During a 24 week prospective trial we followed up seven patients with RA, who had subcutaneous rheumatoid nodules. They received 2 g/day sulphasalazine. Nine patients with RA and rheumatoid nodules treated with gold (sodium aurothiomalate) and six receiving various types of non-steroidal anti-inflammatory drugs (NSAIDs) alone served as controls.

Patients were randomly allocated to receive sulphasalazine, gold, or only NSAID. Intra-articular corticosteroids were not allowed during the course of the study, but some patients continued to receive oral prednisolone 7.5 mg daily or less, which had been started previously. All doses remained constant throughout the trial. There was no statistically significant difference in the characteristics of the patients at the start of the study among the three groups ($p > 0.05$, Mann-Whitney U test).

All patients had active classical or definite RA according to American Rheumatism Association criteria and all were seropositive for rheumatoid factor. The size of the rheumatoid nodules varied between 7 and 28 mm, their localisation was typical (over olecranon, extensor surface of the forearm, proximal interphalangeal joints, patella, Achilles tendon). To assess the disease activity we used the following indices: Ritchie articular index, visual analogue pain scale, duration of early morning stiffness, hand grip strength, and erythrocyte sedimentation rate. Disease activity was considered to be decreased when at least

four of the five variables improved by 50% or more.

At the end of the 24 week observation period no difference in the rate of regression of rheumatoid nodules could be detected between the patients taking sulphasalazine and those in the other two groups.

In the group taking sulphasalazine the size of the subcutaneous nodules had decreased in one patient only, but at the same time improvement in disease activity was seen in five patients. In the control group treated with gold we noticed a diminution of nodules in two patients and a decrease of inflammatory activity in six. In the group taking an NSAID alone the nodule size had decreased in three patients and the disease activity had diminished in four. The use of oral corticosteroids had no influence on the regression of the rheumatoid nodules. The table shows the results of our study.

Complete disappearance of nodules could not be found in any of the 22 patients. It is noteworthy that, like other investigators,^{3,4} we found no relation between the regression of nodules and the reduction of disease activity.

Thus our results failed to confirm the experiences of Englert *et al.* In our opinion the total disappearance of rheumatoid nodules during sulphasalazine treatment may be explained by spontaneous regression. This phenomenon, though rare, is well known in everyday clinical practice and in rheumatological reports.^{3,5}

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Characteristics of patients and results of treatment

Treatment	Sex	Age (years)	Disease duration (years)	Localisation of rheumatoid nodule	Reduction of disease activity	Decrease in nodule size
Sulphasalazine						
1	F	74	6	Olecranon	+	
2	M	54	12	PIP*		
3	F	71	9	Olecranon	+	
4	F	43	8	Achilles tendon	+	
5	M	28	4	Olecranon	+	
6	F	52	7	Forearm		+
7	F	48	6	PIP	+	
Gold						
1	F	40	8	Achilles tendon	+	
2	F	59	11	Olecranon, PIP	+	
3	M	52	15	Patella		
4	F	44	8	Olecranon, PIP	+	
5	F	67	20	Forearm, PIP	+	
6	F	73	9	Olecranon		
7	F	32	3	Forearm		+
8	M	63	8	Olecranon	+	+
9	M	50	7	Forearm, PIP	+	
NSAID*						
1	M	49	8	Olecranon	+	
2	F	32	6	PIP		+
3	F	63	14	Patella, PIP	+	+
4	F	41	1	Forearm	+	+
5	F	68	5	Olecranon		
6	M	72	9	Olecranon	+	

NSAID=non-steroidal anti-inflammatory drug; PIP=proximal interphalangeal joint.

Methotrexate treatment of Felty's syndrome

Sir: We report the response to low dose oral methotrexate treatment in a patient with Felty's syndrome.

A 65 year old white man developed Felty's syndrome in 1987 with polyarthritis, rheumatoid nodules, and splenomegaly. He was found to have neutropenia (white cell count $2.8 \times 10^9/l$; neutrophils 11%) and thrombocytopenia (platelets $123 \times 10^9/l$). Other laboratory indices included a high IgG, erythrocyte sedimentation rate 109 mm/h, rheumatoid factor 1 in 10 240. He also developed severe intermittent claudication. Angiography showed severe 'atherosclerotic' changes in the iliac arteries. Angioplasty was deferred in view of his blood picture. He developed a rash on treatment with penicillamine and treatment was started with prednisolone 7.5 mg daily. There was no improvement in his clinical or haematological status and he was referred to St Thomas's Hospital. In July 1988 treatment was started with methotrexate in an oral dose of 7.5 mg a week, while continuing the prednisolone at 7.5 mg a day. Prednisolone was eventually tapered to 2.5 mg/day.

He showed rapid clinical improvement. At three months his arthritis had subsided and after nine months of treatment the spleen was not palpable, the intermittent claudication had completely disappeared, and the neutrophil and platelet counts had increased: white cell count $5.4 \times 10^9/l$ (neutrophils 46.2%), platelets $150 \times 10^9/l$. Erythrocyte sedimentation rate was 8 mm/h and rheumatoid factor 1 in 2560. The rheumatoid nodules remained. He has had no side effects to date, one year after starting methotrexate.

This report confirms earlier reports of a response of the neutropenia of Felty's syndrome to methotrexate.^{1,2} It was also interesting to note the fall in rheumatoid factor titre and marked improvement in intermittent claudication in this patient, in addition to the response of his rheumatoid arthritis.

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Giant cell arteritis presenting as a supraclavicular nodule

Sir: Giant cell arteritis is a systemic vasculitis that usually affects cranial arteries. Many manifestations secondary to involvement of other territories have also been described, however.¹ We have recently seen an atypical case of giant cell arteritis located in subcutaneous tissue. A supraclavicular nodule simulating a lymphadenopathy was the main clinical expression of this disease.

An 81 year old woman was admitted to our hospital for evaluation of a right supraclavicular nodule. Her personal history was unremarkable. One month before admission she had noted a slightly tender nodule in the right supraclavicular fossa. She also complained of malaise, anorexia, and loss of 10 kg in