Correspondence

Angioimmunoblastic lymphadenopathy

SIR, We were interested to read the case report from McHugh *et al.*¹ We have been following up a patient who presented with similar clinical features, but in whom the findings of T lymphocyte subset analysis of synovial fluid (SF) and peripheral blood (PB) were contrasting (Table 1).

A 51 year old woman presented in August 1983 with generalised lymphadenopathy, fever, weight loss, and arthritis. She was admitted to her local hospital for a lymph node biopsy, which showed features suggestive of angioimmunoblastic lymphadenopathy. When she developed a fever and became anaemic she was transferred to Guy's where a repeat lymph node biopsy showed obliteration of normal lymph node architecture by cells (immunoblasts, plasma cells, and lymphocytes) and vascular components (including cells with endothelial hyperplasia), confirming the diagnosis of angioimmunoblastic lymphadenopathy. Examination at that time showed cervical and axillary lymphadenopathy without hepatosplenomegaly, symmetrical synovitis of metacarpophalangeal and proximal interphalangeal joints, ankles, and knees. Investigations showed haemoglobin 105 g/l, white blood cell count $11.2 \times 10^9/l$, erythrocyte sedimentation rate 116 mm/h, direct Coombs' test positive, and a diffuse increase in gammaglobulin (IgG 28 g/l (normal 9-18.5)). Rheumatoid factor, antinuclear antibodies, complement, hepatitis B surface antigen, thyroid function, chest X ray, and X ray of joints were all normal. Bone marrow showed a reactive marrow with no specific changes. Multitest (Institute Mierieux, Lyons, France) testing showed anergy, consistent with depressed cellular immune responses.

Clinically the patient responded well to prednisolone (20 mg daily) with an increase in haemoglobin and a loss of lymphadenopathy, synovitis, and fever. Joint pain dictated the rate of reduction of steroid dosage, and steroids were stopped in August 1986, since when she has remained well.

Table 1 T lymphocyte subsets

	Peripheral blood	Synovial fluid
Total WBC $\times 10^9/l$	14.2	6.8
Neutrophils $\times 10^9/l$	6.1	NA
Lymphocytes $\times 10^9/l$	3.5	NA
Monocytes $\times 10^9/l$	0.3	NA
OKT3 (%)	27.0	16.5
OKT4 (%)	17.7	4.5
OKT8 (%)	18.0	17.9
T4:T8	0.98	0.25

NA=not available; WBC=white blood cell count.

This patient presented in a similar fashion to the one previously reported.¹ The response to steroids and the subsequent benign course, however, are in contrast. Particularly noticeable is the difference in the findings of T lymphocyte subset analysis. Whereas McHugh *et al.* found a loss of cells of the suppressor phenotype in both PB and SF, these were maintained in the present case and in fact were relatively increased in the SF. Whether this increased number of suppressor cells had an important effect on the outcome of the disease is impossible to assess from an isolated case. There are, however, theoretical grounds for supposing that the numbers of suppressor cells at presentation could provide a prognostic guide, and measurement in future cases should allow testing of this hypothesis.

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1 McHugh N J, Campbell G J, Landreth J J, Laurent M. Polyarthritides and angioimmunoblastic lymphadenopathy. *Ann Rheum Dis* 1987; 46: 555-8.

Did Galen describe rheumatoid arthritis?

SIR, There has been a good deal of speculation and controversy about the antiquity of rheumatoid arthritis. The apparent paucity of convincing descriptions in early medical publications and the absence of typical rheumatoid erosions in skeletal palaeopathology have led some authors to speculate that the disease is of recent origin.^{1 2} Landre-Beavais is usually credited with the first description of the disorder, written in 1800.³ Others have highlighted the difficulties in interpreting either medical accounts of arthritis or skeletal changes, arguing that the negative evidence cited is of little value.⁴ Recent evidence of RA-like erosions in ancient American populations has led to the suggestion that RA may be a 'New World' disease, like syphilis, only transmitted to Europe in the 18th or 19th centuries.⁵

The Greek physician Galen (AD 128-200) classified joint disease according to its site (e.g., 'podagra', 'sciatica') and was the originator of the term 'rheumatism'.⁶ He was a prolific author, writing some 700 books. In his text, 'On the Preservation of Health', he writes about these diseases as follows:

'Now is it not a disgraceful state of affairs that a person who has had an excellent constitution should need to be