Correspondence

Angioimmunoblastic lymphadenopathy

Sirs, We were interested to read the case report from McHugh et al. 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 x 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 g/l)). 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

<table>
<thead>
<tr>
<th></th>
<th>Peripheral blood</th>
<th>Synovial fluid</th>
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<tbody>
<tr>
<td>Total WBC x 10^9/l</td>
<td>14.2</td>
<td>6.8</td>
</tr>
<tr>
<td>Neutrophils x 10^9/l</td>
<td>6.1</td>
<td>NA</td>
</tr>
<tr>
<td>Lymphocytes x 10^9/l</td>
<td>3.5</td>
<td>NA</td>
</tr>
<tr>
<td>Monocytes x 10^9/l</td>
<td>0.3</td>
<td>NA</td>
</tr>
<tr>
<td>OKT3 (%)</td>
<td>27.0</td>
<td>16.5</td>
</tr>
<tr>
<td>OKT4 (%)</td>
<td>17.7</td>
<td>4.5</td>
</tr>
<tr>
<td>OKT8 (%)</td>
<td>18.0</td>
<td>17.9</td>
</tr>
<tr>
<td>T4:T8</td>
<td>0.98</td>
<td>0.25</td>
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NA = not available; WBC = white blood cell count.

This patient presented in a similar fashion to the one previously reported. 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 increase in 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 further measurement in future cases should allow testing of this hypothesis.

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Reference

Did Galen describe rheumatoid arthritis?

Sirs. 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. Landre-Beavais is usually credited with the first description of the disease, written in 1800. Others have highlighted the difficulties in interpreting medical accounts of arthritis or skeletal changes, arguing that the negative evidence cited is of little value. 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.

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'. 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..."
carried about by other people owing to gout . . . Is it not disgraceful that a person should, by reason of that extraordinary thing arthritis, be unable to use his hands and should need somebody else to bring food to his mouth and to perform his toilet necessities for him . . . And even if one overlooks the disgraceful aspect of this, yet one cannot overlook the pain these people suffer, night and day, as though their maladies were torturers twisting them on the rack . . . And the cause of all this must be referred to dissipation or ignorance or both.6

Modern rheumatologists may recognise their rheumatoid patients in this powerful description, which clearly separates generalised arthritis from gout. It can obviously be argued that several other arthropathies could result in similar pain and disability, emphasising the difficulty in interpreting early descriptions of arthritic diseases.

Perhaps the most important aspect of this quotation is not in its diagnostic value, or its contribution to the debate on the antiquity of rheumatoid arthritis. Galen appears to be making a political as well as a medical point—"Is it not disgraceful" is a question that remains pertinent today, and should not be purely rhetorical. At the recent 11th European Congress of Rheumatology, held in Athens, this little known quote was highlighted by Professor Bartsocas of Greece and by me. The continuing plight of those with chronic rheumatic disease, the inadequacies of treatment, and relative lack of funding of rheumatology were also addressed at this conference. If Galen had been allowed to return to his home country as a conference delegate what would he have thought of 1800 years of "progress"? What adjective, other than 'disgraceful' would he have been tempted to use? Would he be accusing us of dissipation, or ignorance, or both?

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References
6 Brook A J. Greek medicine. London: Dent, 1929.