products' in ankylosing spondylitis denotes complement consumption and may represent an altered immune response to a persistent antigen in this disease.

References


Lymphocyte proliferation to artery antigen as a positive diagnostic test in polymyalgia rheumatica. By B. L. HAZLEMAN, I. C. M. MACLENNAN, and M. M. ESIRI (Oxford). Published in full in the Annals, 1975, 34, 222.

Evidence of impaired cell mediated immunity in the seronegative arthritides. By R. D. STURROCK, K. FROEBEL, R. N. M. MACSWEEN, and W. C. DICK (Centre for Rheumatic Diseases, Royal Infirmary and Department of Pathology, Western Infirmary, Glasgow)

Lymphocyte responses to plant mitogens have previously been reported to be depressed in ankylosing spondylitis (Escanilla, Alepa, and Reeve, 1970). In view of this, lymphocyte responses to phytohaemagglutinin (PHA), Conconavilin A (Con A), and poke-weed mitogen (PWM) have been studied in twenty patients with ankylosing spondylitis, twelve patients with Reiter's disease, and eleven patients with psoriatic arthritis*. The results were compared with a group of fourteen normal controls and twenty patients with osteoarthritis. Skin testing was performed using 40 units streptokinase-streptodornase variant and dinitrochlorobenzene sensitization. The Table summarizes the effect of the plant mitogens on lymphocytes from the various groups. PHA responses at a submaximal dose were significantly depressed in the ankylosing spondylotic and Reiter's groups and occurred particularly in those patients with clinically severe disease. An increased response occurred in the psoriatic group at higher doses of PHA and a similar effect was observed in response to PWM among the Reiter's patients. No correlation was found between in vivo skin testing and in vitro lymphocyte response, and the pattern of skin testing was not significantly different from that of controls.

* None of these patients were on anti-inflammatory treatment at the time.

A serial study of eosinophilia and raised IgE antibodies during gold therapy. By P. DAVIS and G. R. V. HUGHES (Post-graduate Medical School, Hammersmith Hospital, London)

Gold therapy is known to be effective therapy for rheumatoid arthritis (RA) but to be associated with a high incidence of mucocutaneous side effects (Empire Rheumatism Council, 1961). Little is known about the mechanism of gold reactions nor is there a satisfactory method of monitoring gold reactions. A recent report has shown that patients with gold reactions also have raised levels of IgE antibodies (Davis and others, 1973) and that this was usually associated with raised eosinophil counts. The aim of this study was to perform serial IgE levels and eosinophil counts in patients receiving gold for arthritis and to correlate raised levels with gold reactions.

Case and methods

47 patients with RA and 3 patients with psoriatic arthritis have been studied. All patients were receiving gold in a standard regimen and had normal eosinophil counts and IgE levels before starting their therapy. Patients with a known atopic history were excluded. Eosinophil counts were measured by routine full blood counts and IgE levels by the radioimmunosorbent technique.

Results

14 patients developed a reaction to their gold therapy, i.e. rash, pruritus, or mouth ulcers. 11 of these patients had concurrent eosinophilia which preceded the side effect in 6 instances. In addition, a further 9 patients had eosinophilia without clinical side effect. IgE levels were raised in 22 of the 50 patients receiving gold therapy, the relationship to eosinophilia and clinical side effect is shown in the Table.

Table

Lymphocyte responses to PHA, PWM, and Con A found to be significantly different (P < 0.05) from normal

<table>
<thead>
<tr>
<th></th>
<th>PHA (2.5 μg/ml)*</th>
<th>Psoriatic arthritis</th>
<th>Reiter's arthritis</th>
<th>Osteoarthritis</th>
<th>Normals</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ankylosing spondylitis</td>
<td>$ t = 2.84$</td>
<td>$P &lt; 0.05$</td>
<td>$ t = 2.989$</td>
<td>$P &lt; 0.05$</td>
<td>$ t = 2.84$</td>
</tr>
<tr>
<td>Con A (250 μg/ml)*</td>
<td>$ x = 32.921 \pm 4.461$</td>
<td>$ t = 5.12$</td>
<td>$P &lt; 0.005$</td>
<td>$ x = 33.268 \pm 3.991$</td>
<td>$ t = 2.947$</td>
</tr>
<tr>
<td>PWM (1:10)*</td>
<td>$ x = 28.138 \pm 3.601$</td>
<td>$ t = 3.218$</td>
<td>$P &lt; 0.01$</td>
<td>$ x = 13.341 \pm 2.099$</td>
<td>$ t = 2.2416$</td>
</tr>
</tbody>
</table>

* The concentration of the stock solution of mitogen. The final concentration in the culture medium is 10% of the stock solution.
† The mean dpm per culture for the group ± SEM.
‡ The values of t and P are derived using Student's 't' test.
**Proceedings: Evidence of impaired cell mediated immunity in the seronegative arthritides.**

R D Sturrock, K Froebel, R N MacSween and W C Dick

*Ann Rheum Dis* 1975 34: 203
doi: 10.1136/ard.34.2.203-a

Updated information and services can be found at:
[http://ard.bmj.com/content/34/2/203.1.citation](http://ard.bmj.com/content/34/2/203.1.citation)

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

**Notes**

To request permissions go to:
[http://group.bmj.com/group/rights-licensing/permissions](http://group.bmj.com/group/rights-licensing/permissions)

To order reprints go to:
[http://journals.bmj.com/cgi/reprintform](http://journals.bmj.com/cgi/reprintform)

To subscribe to BMJ go to:
[http://group.bmj.com/subscribe/](http://group.bmj.com/subscribe/)