Letters to the Editor

Leucocytic alkaline phosphatase activity, marker of evolution in leprosy?

Lepromatous leprosy is an interesting disease regarding macrophage function and the host's failure to control the disease. The hypothesis that impaired non-specific defences might have a role in leprosy has become increasingly plausible, with speculation that macrophages are unable to present lepromatous antigens to the immune system. As impaired phagocytosis has been implicated, we undertook a survey of the whole phagocyte population. We chose to study leucocytic alkaline phosphatase (LAP) activity because of the similarity between lepromatous leprosy and mucocutaneous candidiasis: this latter disease, characterised by the persistence of Candida in spite of high titres of anticanada antibodies had been reported as showing a substantial decrease in LAP activity.

Blood specimens collected on lithium heparinate were obtained in the French West Indies from 31 patients with leprosy aged between 4 and 73, and 11 healthy controls. All 31 patients had major cutaneous or neurological lesions. They were classified according to Ridley and Jolpling's criteria as follows: 14 cases of lepromatous leprosy (eight receiving treatment and six not yet treated); nine with tuberculosis (seven had been treated); three borderline cases under treatment; and five with indeterminate leprosy (not yet treated). Healthy controls were of the same race and social background as the patients.

The blood smears were fixed in formalin (10% in methanol) and stored at 4°C, and processed within three days for LAP activity (technique adapted from Kaplow). The slides were examined microscopically for a positive reaction by semiquantitative evaluation: the formation of a brown intracytoplasmic precipitate was scored from 0 to 4. The final result was expressed as the mean scores obtained from the cytological examination of 100 polymorphonuclear cells. The distribution of the different values showed a significant difference (p < 0.001) between patients with leprosy (mean (SEM) score 33.8 (7.3)) and healthy controls (109.8 (12.5)). As there was no significant difference between patients who had been treated and those who had not, they were grouped in the same category as the 14 with lepromatous leprosy and the nine with tuberculosis. These two groups each showed significantly different values compared with healthy controls (p < 0.001). Furthermore, there was a significant difference (p < 0.001) between the group with tuberculosis (47.2 (11.4)) and the group with lepromatous leprosy (20.6 (9.3)). Although the borderline group is too small to permit statistical analysis, it is interesting to note that its mean score (31) places borderline cases between the scores for tuberculosis and lepromatous leprosy. Finally, the LAP score of those with indeterminate leprosy matches a value distribution which spans all the leprosy types.

This work shows a correlation between LAP activity and leprosy with a progressive decrease of the score from tuberculosis to lepromatous leprosy. As the role of LAP is unknown, we cannot speculate whether it is a cause or consequence of infection. The role of granulocytes in protection against leprosy is dubious, and changes in granulocyte enzyme activity are likely to be epiphenomena, but we think that the LAP score might have a predictive value for the evolution of indeterminate leprosy. In this respect it is interesting to note that the lowest score we obtained was from a patient initially diagnosed with indeterminate leprosy that proved later to be lepromatous. This test is cheap and easy to perform, and it might be interesting to evaluate LAP score in those with indeterminate leprosy as well as in the follow up of those with borderline leprosy.

Table Mean LAP score for each type of leprosy and for controls

<table>
<thead>
<tr>
<th>Samples</th>
<th>(n)</th>
<th>Mean (SEM)</th>
<th>t=0.05 Sm</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tuberculoid</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treated</td>
<td>7</td>
<td>44.8 (9.8)*</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Untreated</td>
<td>2</td>
<td>55.5 ND</td>
<td></td>
</tr>
<tr>
<td>Total</td>
<td>9</td>
<td>47.2 (11.4)*</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Lepromatous</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treated</td>
<td>8</td>
<td>16.5 (1.4)*</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Untreated</td>
<td>6</td>
<td>26.1 (20.9)*</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Total</td>
<td>14</td>
<td>20.6 (9.3)*</td>
<td>p &lt; 0.001</td>
</tr>
<tr>
<td>Borderline</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Treated</td>
<td>3</td>
<td>31 ND</td>
<td></td>
</tr>
<tr>
<td>Indeterminate</td>
<td>5</td>
<td>48.2 ND</td>
<td></td>
</tr>
<tr>
<td>Controls</td>
<td>11</td>
<td>109.1 (12.5)</td>
<td></td>
</tr>
</tbody>
</table>

*p < 0.001

References

Troublesome Romanowsky stain deposits

These deposits may be removed from blood films by the following simple method. A sheet of cellulose tissue is placed on the slide overlapping one end. Two or three drops of a mixture of xylene, three parts, and 74% ethanol, one part, are run on to the tissue so that it adheres to the slide. The tissue is then drawn off the slide by pulling in the plane of the film. In this way the whole film is wetted and gently wiped by the tissue. The xylene appears to temper the solvent action of the ethanol so that staining of the blood film is unaffected even by repeated treatments.

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Nosocomial outbreak of Achromobacter xylosoxidans associated with a diagnostic contrast solution

Achromobacter xylosoxidans is a Gram negative rod, strictly aerobic, oxidase and catalase positive, motile by peritrichous flagella and which does not attach to carbohydrates. Taxonomically, it is recognised by the Centers for Disease Control as a genus and is subdivided into two species, Achromobacter xylosoxidans and an unnamed species designated Vd. Bergey's Manual of Systematic Bacteriology still does not accept Achromobacter as a genus name and it is listed as Alcaligenes denitrificans biotype xylosoxidans. The natural habitat of this organism remains unknown, but it has been isolated from many clinical sources (cerebrospinal fluid, blood, urine, pleural and peritoneal fluid, faeces, wounds, pharynx, and sputa), various hospital and
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