Transient ‘stress lymphocytosis’

Sir,

We wish to draw attention to a little-recognized clinical condition termed ‘stress lymphocytosis’ which is not mentioned in standard haematology texts; it may be defined as a lymphocyte count greater than 4·0 × 10⁹/L, lasting less than 24 h and following trauma, an acute medical event or possibly just fear.

A short-lived lymphocytosis has been described following injection of adrenaline (Steel et al., 1971), trauma, wounds or stress-related conditions (Thommasen et al., 1986; Teggtatz et al., 1987; and Soppi et al., 1982). We have detected 17 such instances in a 2-year period, drawn from one emergency casualty department. Total white cell counts were performed by a Coulter Model S Plus™ or Model S Plus IV™. In all but one case a differential count was done on 100 white cells. The clinical conditions of the 17 patients were: cardiac arrest or myocardial infarct (5), seizures (5), trauma, bleeding (4), abdominal pain (1), brain stem infarct (1) and gastroesophageal reflux (1). The median age of the patients was 61 years (range 24–81). A further two patients are not included because they had a second lymphocyte count only after 24 h (Table 1).

Changes occurred in other leukocyte classes, not shown in the table. Mean neutrophil count of the first samples was 4·9 × 10⁹/L (range 0·2–9·2), and increased to 11·9 × 10⁹/L (range 3·6–29·4) in the second sample. In five cases, the number of eosinophils was increased in the first sample (0·6–1·5, mean 0·9 × 10⁹/L) and returned to normal in the second blood sample. No patient had evidence of lymphoproliferative disorder or infection. One received an adrenaline injection prior to the first blood sample.

No relationship was detected between the degree of lymphocytosis and clinical outcome. The lymphocytes were normal, pleomorphic (resembling lymphoma cells) and ‘atypical’, suggestive of a viral infection. In five patients, the immunological phenotype was determined by a dual laser Epic V™ flow cytometer. T-cells showed an increase, ‘T8’ cells more so than ‘T4’, thus the T4/T8 ration was reduced.* Three of the five showed an increase in B cells. All these five showed normal lymphocyte morphology.

Table 1  Blood counts in 17 patients with transient lymphocytosis

<table>
<thead>
<tr>
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<th>Leukocytes × 10⁹/L</th>
<th>Lymphocytes × 10⁹/L</th>
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<tbody>
<tr>
<td></td>
<td>range</td>
<td>mean</td>
</tr>
<tr>
<td>1st sample</td>
<td>6·2–21·9</td>
<td>14·6</td>
</tr>
<tr>
<td>2nd sample</td>
<td>5·4–30·6</td>
<td>14·2</td>
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</table>

* T₄/T₈ mean 1·12, range 0·6–1·6, normal mean 2·0, SD. ± 0·25.

Only one of our clinical colleagues in the Emergency Department or the Haematology service was aware of this phenomenon, recognition of which may avoid unnecessary investigation.

This was not a prospective study. Some patients with lymphocytosis were not admitted and had no second blood test. Some patients had their first blood count only
after being admitted and any lymphocytosis could have subsided. It is probable, therefore, that transient lymphocytosis occurs more frequently than we had observed. The source of the lymphocytes is possibly a re-distribution from various organs, thoracic duct and lymph nodes mediated by catecholamine. The transient lymphocytosis in our patients probably reflects a stress-related increased endogenous catecholamine level (Davies et al., 1984). Further studies are necessary to assess their clinical significance.

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REFERENCES