Letters to the Editor

unreliable for the detection of antibodies to CMV. The latex agglutination test will be of particular value for screening patients before marrow transplantation. Those found to have no CMV antibody could then be given CMV negative blood products or CMV immunoglobulin, or both.

The disappearance of antibody from one patient over a few months is worthy of note. During November and December 1985, this patient had received 104 units of platelet concentrate. We would suggest that the two positive sera were due to passively acquired antibody. If screening for CMV antibody is to be undertaken on patients awaiting marrow transplantation it should be performed as long as possible after any previous transfusion likely to transfer antibody. A single negative serum is probably a reliable indicator that the patient is not immune to CMV infection.

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References


Coagulation changes in homozygous sickle cell disease in Nigeria

Abnormalities in haemostasis have been described in sickle cell disease, and recently increased fibrinogen concentrations1 and increased viscosity2 have been observed. These have been related to possible abnormalities in plasma proteins in patients with sickle cell disease (SCD). No study has been designed to examine such changes in Nigerian patients, despite the large numbers of patients with the disease in our population.

The patients studied included 70 (20 men and 50 women) patients with SCD attending the University of Benin Teaching Hospital, Nigeria. Their routine checks included haemoglobin concentration or packed cell volume, administration of antimarial drug (proguanil or pyrimethamine), and folic acid supplementation. The presence of joint pains, fever, and bone pains of sufficient gravity to warrant immediate admission were considered to constitute a crisis. Seventy five healthy non-sicklers served as controls. A venous blood sample from each patient was assessed for platelet count, fibrinogen, factors V and VIII.

The results are shown in the table. There was an increased platelet count (p < 0.05), fibrinogen concentration, (p < 0.005), and factor VIII p < 0.01, but a reduced factor V value (p < 0.0005) in patients with SCD in stable state compared with non-sicklers.

SCD in crisis also showed an increased platelet count (p < 0.0005), fibrinogen concentration (p < 0.0005), higher factor VIII (p < 0.0005) and lower factor V value (p < 0.0005) than patients with SCD in stable state.

The cause of the increased factor VIII value may be fever, stress, and infections which are common complications during crisis. The changes in factor V and factor VIII values observed in this study are similar to the findings of Leslie et al3 and Green et al,4 who compared steady state patients with normal age matched black controls. Raised factor VIII values have been reported in other haemolytic anaemias5 and may reflect increased reticuloendothelial cell activity due to hypoxia during crisis. As vascular occlusion resulting from sickled erythrocytes is a common occurrence in sickle cell disease the possibility exists that stasis combined with an increase in factor VIII may lead to thrombotic complications. In other words increased factor VIII values could produce a detrimental hypercoagulable state. Factor V values dropped during crisis, perhaps due to consumptive coagulopathy. Impaired or subclinical derangement of liver functions might also account for the reduced values of factor V.

This study has established that there is evidence of continuous activation of the coagulation system together with thrombocytosis and a hyperfibrinogenemia in SCD. A more extensive longitudinal study which may help to establish the role of coagulation studies in the prediction of crisis in SCD is underway in our laboratory.

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References

1 Famodu AA, Reid HL. Fibrinogen level in sickle-cell disease (HbSS). Tropical and Geographical Medicine 1987;33:36–9.

Table 1 Mean (SD) coagulation changes in non-sicklers compared with those in sicklers in steady state and sicklers in crisis episodes

<table>
<thead>
<tr>
<th></th>
<th>Platelets (x 10^9/l)</th>
<th>Fibrinogen (g/l)</th>
<th>Factor V (%)</th>
<th>Factor VIII (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Control non-sicklers</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>206.21 (62-0)</td>
<td>3.12 (1.04)</td>
<td>100.47 (15.68)</td>
<td>141.90 (47.32)</td>
</tr>
<tr>
<td>Range</td>
<td>(150–320)</td>
<td>(1.90–5.25)</td>
<td>(75–150)</td>
<td>(85–300)</td>
</tr>
<tr>
<td>Sicklers in steady state</td>
<td>222.41 (40.5)</td>
<td>5.25 (2.00)</td>
<td>93.14 (12.01)</td>
<td>216.10 (59.86)</td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>(160–320)</td>
<td>(3.0–11.5)</td>
<td>(70–130)</td>
<td>(100–350)</td>
</tr>
<tr>
<td>Range</td>
<td>(210–400)</td>
<td>(3.0–1.05)</td>
<td>(50–120)</td>
<td>(180–300)</td>
</tr>
<tr>
<td>Sicklers in crisis</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>302.51 (50.3)</td>
<td>6.24 (2.05)</td>
<td>76.75 (15.67)</td>
<td>247.00 (40.75)</td>
</tr>
<tr>
<td>Range</td>
<td>(210–400)</td>
<td>(3.0–1.05)</td>
<td>(50–120)</td>
<td>(180–300)</td>
</tr>
</tbody>
</table>
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