Acute crescentic glomerulonephritis as a complication of a Staphylococcus aureus abscess of hip joint prosthesis

We report a case of acute diffuse proliferative glomerulonephritis following a coagulase positive staphylococcal (S aureus) abscess around a hip joint prosthesis. A standard work on renal pathology describes only three personally observed cases of staphylococcal septicaemia without endocarditis, in association with proliferative glomerulonephritis, and cites a description of two cases associated with staphylococcal pneumonia. Furthermore, we are not aware of a previous case of acute diffuse proliferative glomerulonephritis occurring after a Staphylococcus aureus infection of a hip joint prosthesis.

A 75 year old man had a right total hip replacement followed by a transurethral resection of prostate two months later. Over the following month he became increasingly confused and feverish. Blood and urine cultures persistently showed Staphylococcus aureus infection. He was treated with vancomycin, velosef, and amikacin, but developed uirementa despite peritoneal dialysis and died three months after the first operation.

At necropsy about 100 ml of purulent material were found in a loculated thick walled abscess around the hip prosthesis. Blood and hip abscess culture showed Staphylococcus aureus infection. Histological examination of the kidney showed diffuse proliferative glomerulonephritis. The glomeruli contained numerous crescents, thrombi, neutrophil polymorphs and areas of necrosis.

Previous studies of diffuse proliferative glomerulonephritis due to coagulase positive staphylococci indicate an immune complex aetiology rather than a bacterial embolic one. Electron microscopy has shown distinct subepithelial deposits, immunofluorescence has shown granular deposits of C3 and IgG within the glomerulus, and staphylococcal antigen and serum complement have been repeatedly reported to be reduced. The localised nature of the abscess in this case would make the propagation of large septic emboli much less likely than immune complexes, further confirming the latter theory of pathogenesis.

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References

Latex agglutination test for detecting CMV antibodies in patients awaiting bone marrow transplantation

Cytomegalovirus (CMV) infection in patients undergoing bone marrow transplantation is a major cause of morbidity and mortality. Patients who do not possess cytomegalovirus antibodies are particularly at risk if given blood or blood products from seropositive donors. The use of intravenous CMV immunoglobulin has been shown to be an effective prophylactic agent in this group, but the serum is expensive and so its use has to be restricted to seronegative patients. The availability of a rapid and reliable test that can show antibodies to CMV is therefore necessary.

To assess the sensitivity and specificity of the latex agglutination test an initial study of 100 sera was made. These sera were all from known homosexuals presenting to a genitourinary clinic. In a further study of patients awaiting bone marrow transplantation 13 sera were examined. All sera had been stored at -80°C before examination. In addition, three further sera from one patient, taken over a period of five months were examined—the patient was a 39 year old woman suffering from relapsed acute myeloid leukaemia. The sera were examined, both by a latex agglutination test (CMV Scan, Becton and Dickinson, Baltimore, Maryland) and an IgG ELISA test (Virenz G-CMV, Northumbria Biologicals Limited, Cramlington, Northumberland). The tests were performed exactly as stated in the instructions.

In the initial study of 100 sera from homosexuals there was 99% correlation between the two tests. Eighty sera gave positive results by both methods and 19, negative results. The remaining serum gave a positive result by latex but a negative result by ELISA. When both tests were repeated on this serum, both gave positive results. Of 100 sera examined by the ELISA test, there was one false negative result. In the study of sera from patients awaiting marrow transplantation there was a 100% correlation between the two tests. Of the transplant recipients, eight (62%) were positive and five (38%) negative.

The initial serum from the patient who had serial studies was received on 31 December 1985 and was positive by both methods. A further serum was received on 24 April 1986 and was still positive by both methods, though the ELISA was only weakly positive. Two further sera, received on 21 and 29 May 1986, were negative by both methods. These results were confirmed on retesting of the stored sera.

We agree with the findings of previously published studies that the latex agglutination test for CMV is both sensitive and specific. Furthermore, the test is rapid and easy to perform, making it suitable for use in non-specialist laboratories. Even in virology laboratories it compares favourably with other methods, particularly complement fixation tests which are known to be
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 antimalarial 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 steady 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 al4, who compared steady state patients with normal age matched control. Raised factor VIII values have been reported in other haemolytic anaemias5 and may reflect increased reticulo-endothelial 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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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 (× 10^11/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 (15-68)</td>
<td>141 (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></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Mean (SD)</td>
<td>222.41 (40-5)</td>
<td>5.25 (2.00)</td>
<td>93 (12-01)</td>
<td>216 (59-86)</td>
</tr>
<tr>
<td>Range</td>
<td>(160-320)</td>
<td>(3.0-11.5)</td>
<td>(70-130)</td>
<td>(100-350)</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 (2.50)</td>
<td>76 (15-67)</td>
<td>247 (40-75)</td>
</tr>
<tr>
<td>Range</td>
<td>(210-400)</td>
<td>(3.0-12-5)</td>
<td>(50-120)</td>
<td>(180-350)</td>
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</table>
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