Renal transplantation from HBsAg positive donors to HBsAg negative recipients

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There has been a rapid increase in the numbers of people undergoing dialysis in Hong Kong owing to an active programme of continuous ambulatory peritoneal dialysis.1 The number of renal transplant patients has been increasing steadily with the growing numbers of patients accepted for dialysis because of the scarcity of donor kidneys. We describe here our initial experience in transplanting kidney recipients from donors positive for hepatitis B surface antigen to recipients negative for hepatitis B surface antigen.

Patients, methods, and results

Four patients who were negative for hepatitis B surface antigen, as assessed by a reversed passive haemagglutination test, received kidneys from donors positive for the antigen (table). They all received azathioprine and prednisolone as immunosuppressive agents.

Case 1—A 30 year old man received a kidney from his sister. Hyperimmune gammaglobulin was given in a dose of 3 ml immediately before the operation and another dose three months later. Good graft function was achieved. Fifty months after transplantation cyclosporin was substituted for azathioprine because of a slow but steady decline in renal function. The patient remained negative for hepatitis B surface antigen three years after transplantation. Antibodies to hepatitis B surface antigen were detected at two years but persisted for only six months. At the last review the patient's serum creatinine concentration was 276 μmol/l, and his transaminase activities were normal.

Case 2—A 36 year old man received a kidney from his mother. Before operation and immediately afterwards he was given 4 ml of hyperimmune gammaglobulin. Another dose was given three months later. Good graft function was achieved. The patient developed antibodies to hepatitis B surface antigen at
### Antibody state of donors and recipients of renal transplants

<table>
<thead>
<tr>
<th>Case</th>
<th>Donor</th>
<th>Recipient</th>
<th>Conversion to anti-HBsAg positive</th>
<th>Conversion to anti-HBcAg positive</th>
<th>Hepatitis</th>
<th>Serum creatinine at follow up (µmol/l)</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>HBsAg+anti-HBsAg+</td>
<td>HBsAg-, anti-HBsAg-, anti-HBc+</td>
<td>Transient at 2-2½ years</td>
<td>No</td>
<td>No</td>
<td>276</td>
</tr>
<tr>
<td>2</td>
<td>HBsAg+anti-HBsAg+</td>
<td>HBsAg-, anti-HBsAg-, anti-HBc+</td>
<td>Persistent from 1 year onwards</td>
<td>No</td>
<td>No</td>
<td>136</td>
</tr>
<tr>
<td>3</td>
<td>HBsAg+anti-HBsAg+</td>
<td>HBsAg-, anti-HBsAg-, anti-HBc+</td>
<td>Persistent from 1 year onwards</td>
<td>No</td>
<td>No</td>
<td>186</td>
</tr>
<tr>
<td>4</td>
<td>HBsAg+anti-HBsAg+</td>
<td>HBsAg-, anti-HBsAg-, anti-HBc+</td>
<td>Persistent throughout</td>
<td>No</td>
<td>No</td>
<td>123</td>
</tr>
</tbody>
</table>

one month, though these could not be detected at three and six months. At one year, however, he again became positive for antibodies to hepatitis B surface antigen, and the antibodies persisted. Serum creatinine concentration three years after transplantation was 136 µmol/l. Serum transaminase activities remained normal throughout.

_Case 3_—An 18 year old woman received a kidney from her mother. Hyperimmune gammaglobulin was given preoperatively in a dose of 5 ml, and a total of three doses were given at three monthly intervals. Good graft function was achieved, and the serum creatinine concentration three years after renal transplantation was 86 µmol/l. One year after operation the patient developed antibodies to hepatitis B surface antigen, which persisted. Transaminase activities were normal throughout.

_Case 4_—A 24 year old woman received a kidney from her mother. Hyperimmune gammaglobulin was given only before the operation. Good graft function was achieved, and the serum creatinine concentration two and a half years after transplantation was 125 µmol/l. Because of excessive alopecia azathioprine was replaced with cyclosporin 24 months after the operation. The patient remained positive for antibodies to hepatitis B surface antigen throughout, and her transaminase activities were normal.

### Comment

To discard kidneys from donors positive for hepatitis B surface antigen means discarding kidneys from 10% of potential donors, for such is the rate of carriage of hepatitis B surface antigen among the general population in Hong Kong. Our patients did not receive donor specific blood transfusions, and the blood transfused at the time of the operation was negative for hepatitis B surface antigen. The donor kidney was therefore the only parenteral source of hepatitis B surface antigen to which the patients were exposed.

None of our donors were positive for e antigen, which suggests that the donor kidneys may not have been particularly infectious since the presence of e antigen is an index of infectivity. Nevertheless, all except one of the recipients had evidence of exposure to the hepatitis B virus before renal transplantation since they had antibodies to the core antigen. Furthermore, two patients developed persistent and one transient antibodies to hepatitis B surface antigen, indicating that the donor kidney (or the blood containing) had been an important source of the viral surface antigen.

Our experience suggests that with the use of hyperimmune gammaglobulin it is safe to use kidneys from donors positive for hepatitis B surface antigen but negative for e antigen for transplantation in patients who are negative for hepatitis B surface antigen.

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### Rheumatoid arthritis: an unrecognised cause of pseudohyperkalaemia

Stuart H Ralston, Matthew Lough, R D Sturrock

Hyperkalaemia may be incorrectly diagnosed in patients with severe thrombocytosis or leucocytosis because potassium is released in vitro from platelets and leucocytes during coagulation of blood.1,2 Hitherto, pseudohyperkalaemia has been reported only in patients with myeloproliferative disease.3,4 Two patients with thrombocytosis associated with rheumatoid arthritis who developed pseudohyperkalaemia prompted us to look for this condition in a group of patients with rheumatoid arthritis and controls.

### Patients, methods, and results

_Case 1_—A 79 year old woman with active rheumatoid arthritis who was taking ibuprofen 400 mg thrice daily was found to have a serum potassium concentration of 5-7 mmol/l on routine testing. This raised concentration persisted after ibuprofen was withdrawn. Haemoglobin concentration was 149 g/l, platelet count 930×10^9/l, and white cell count 14×5×10^9/l. A bone marrow aspirate was normal. Pseudohyperkalaemia was diagnosed and confirmed by simultaneous measurement of serum and plasma potassium concentrations (4-7 and 3-4 mmol/l respectively; difference 1-3 mmol/l).

_Case 2_—A 70 year old woman with active rheumatoid arthritis who was taking ibuprofen 400 mg thrice daily was found to have a serum potassium concentration of 5-7 mmol/l on routine testing. This raised concentration persisted after ibuprofen was withdrawn. Haemoglobin concentration was 149 g/l, platelet count 930×10^9/l, and white cell count 14×5×10^9/l. A bone marrow aspirate was normal. Pseudohyperkalaemia was diagnosed and confirmed by simultaneous measurement of serum and plasma potassium concentrations (4-7 and 3-4 mmol/l respectively; difference 1-5 mmol/l).

Subsequently we studied 43 patients with rheumatoid arthritis, 40 of whom were taking non-steroidal anti-inflammatory drugs; 21 controls taking non-steroidal anti-inflammatory drugs for non-steroidal arthritis; and 28 normal subjects. None was taking other drugs known to affect potassium homeostasis. Paired samples of venous blood were collected into plain and lithium heparin containers, separated under routine conditions, and stored at

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