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Renal transplantation from HBsAg positive donors to HBsAg negative recipients

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There has recently 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 transplants performed² can barely keep up with the numbers of patients accepted for dialysis because of the scarcity of donor kidneys. We describe here our initial experience in transplanting kidneys 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

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Case	Donor	Recipient	Conversion to anti-HBsAg	Conversion to HBsAg positive	Hepatic dysfunction	Serum creatinine at follow up ($\mu\text{mol/l}$)
1	HBsAg+anti-HBeAg+	HBsAg-, anti-HBsAg-, anti-HBe+	Transient at 2-2½ years	No	No	276
2	HBsAg+anti-HBeAg+	HBsAg-, anti-HBsAg-, anti-HBe+	Persistent from 1 year onwards	No	No	136
3	HBsAg+anti-HBeAg+	HBsAg-, anti-HBsAg-, anti-HBe-	Persistent from 1 year onwards	No	No	186
4	HBsAg+anti-HBeAg+	HBsAg-, anti-HBsAg+, anti-HBe+	Persistent throughout	No	No	125

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 $136 \mu\text{mol/l}$. 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 $86 \mu\text{mol/l}$. 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 $125 \mu\text{mol/l}$. 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.³ 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 it contained) 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

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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.^{1,3} Two patients with thrombocytosis associated with rheumatoid arthritis who developed pseudohyperkalaemia are presented who 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 naproxen 500 mg twice daily was found to have a serum potassium concentration of 7.3 mmol/l (normal $3.5\text{--}5.0 \text{ mmol/l}$) on routine testing. Haemoglobin concentration was 108 g/l , platelet count $1260 \times 10^9/\text{l}$, and white cell count

$11.9 \times 10^9/\text{l}$. Bone marrow was normal. The naproxen was stopped and dextrose-insulin and calcium polystyrene sulphonate started, but she continued to have intermittent episodes of hyperkalaemia. Pseudohyperkalaemia was diagnosed and was confirmed by simultaneous measurements 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 $930 \times 10^9/\text{l}$, and white cell count $14.5 \times 10^9/\text{l}$. A bone marrow aspirate was normal. Pseudohyperkalaemia was diagnosed and confirmed by simultaneous measurement of serum and plasma potassium concentrations (6.3 and 4.8 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-inflammatory 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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