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 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 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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-20°C until being assayed for serum and plasma potassium concentrations by flame photometry (within batch SD=0.03 mmol/l). The Mann-Whitney U test and Spearman's rank correlation coefficient were used in statistical analysis.

The median (range) difference in potassium concentration between serum and plasma in the 43 patients with rheumatoid arthritis was 0.3 (0.1-1.3) mmol/l, which was significantly greater ($p<0.001$) than that in the 21 controls with arthritis (0.2 (0.0-0.4) mmol/l) and the 28 normal subjects (0.2 (0.0-0.4) mmol/l). Serum creatinine and plasma potassium concentrations and the white cell count were similar in the three groups, but the platelet count was significantly ($p<0.001$) higher in the patients with rheumatoid arthritis (median (range) 366 (165-1260) $\times 10^3/l$ v 287 (122-444) $\times 10^3/l$ in the controls with arthritis and 305 (192-440) $\times 10^3/l$ in the normal subjects). The difference in potassium concentration between serum and plasma in the patients with rheumatoid arthritis correlated with their platelet count (figure) ($r=0.64$, $p<0.001$).

Relation between platelet count and difference in potassium concentration between serum and plasma (potassium difference) in 43 patients with rheumatoid arthritis. Area enclosed by broken line indicates reference ranges for both variables in normal subjects

Comment

We are not aware of other reports of pseudohyperkalaemia occurring in conditions such as rheumatoid arthritis. We found that in normal subjects the median serum potassium concentration was 0.2 mmol/l higher than the plasma potassium concentration, which confirms a previous report.¹ We found a similar difference in our control group of patients with arthritis who were taking non-steroidal anti-inflammatory drugs, which excludes these as the cause of the pseudohyperkalaemia. In contrast, the difference in concentrations was significant in the patients with rheumatoid arthritis, with values of 1.5 mmol/l being recorded (case 2). The pseudohyperkalaemia may have been due to the raised platelet count in view of the correlation between it and the difference between serum and plasma potassium concentrations.

Failure to recognise pseudohyperkalaemia in our patients led to errors in management: one patient was given treatment to lower her potassium concentration

and one had her treatment with a non-steroidal anti-inflammatory drug stopped unnecessarily. Our data suggest that pseudohyperkalaemia may be common and often overlooked in active rheumatoid arthritis. It may easily be confirmed by concurrent measurements of serum and plasma potassium concentrations, a difference >0.40 mmol/l being diagnostic.

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Relative risks of different types of contact lenses

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There have been few studies to determine the size of the problem of eye disease related to contact lenses in a community or to compare the risks of complications associated with different types of lenses.^{1,2} We therefore performed a case-control study to estimate the relative risks.

Patients, methods, and results

All patients who wore contact lenses and attended the High Holborn branch of Moorfields Eye Hospital between 1 January and 31 March 1986 were included in the study. Disorders related to contact lenses were diagnosed from previously agreed clinical criteria; the conditions were grouped for analysis according to pathogenesis.

Of 393 patients (152 men, 241 women) who wore contact lenses, 329 had problems related to wearing lenses; 64 patients who wore lenses attended for an

unrelated ocular problem. One hundred and ninety patients wore daily wear soft lenses, 45 extended wear soft lenses, 100 polymethylmethacrylate hard lenses, and 58 gas permeable hard lenses. Two hundred and seventeen patients were aged 20-29. Of the 393, 367 patients obtained their lenses privately and 26 were fitted by hospital eye departments. Ten patients wore lenses for aphakia, six for keratoconus, and 14 for high myopia. The rest wore lenses in preference to spectacles for low refractive errors.

The main groups of conditions seen were metabolic (principally corneal hypoxia), toxic and hypersensitivity reactions, corneal abrasions, and infections. Five cases of bacterial keratitis were diagnosed, all in patients who wore soft lenses. Problems related to contact lenses accounted for 10% of all attendances at the casualty department.

The relative risk associated with each type of lens for a disorder was estimated by calculating the odds ratio from contingency tables. Hard lenses were hypothesised to have the least risk of complications before the analysis, for which the polymethylmethacrylate hard lens was taken as the referent, with a baseline risk of 1.0. The risks for other lenses were estimated as multiples of this. The significance of the increasing risks was estimated with the Mantel-Haenszel χ^2 test of trend.³ The overall risk for soft lenses compared with