The donor kidney came from a 36 year old woman who had died from a subarachnoid haemorrhage and who had not had any urinary problems. The recipient of the second kidney from this donor has had normal renal function and has shown no evidence of ureteric obstruction.

Intravenous urogram showing ureteric calculus originating in donor kidney.

Comment

Urological complications of renal transplantation are well recognised^{1,2} but, to our knowledge, this is the first case of ureteric obstruction resulting from a calculus in the donor kidney. The recipient's normal bone biochemistry and the time of presentation make it unlikely that the calculus arose de novo after surgery. Unless a calculus is particularly large it is unlikely to be recognised at retrieval surgery. Previous studies have emphasised the need for early detection in the management of the obstructed transplant ureter.³ The frequent use of an isotope renogram in preference to an intravenous urogram after transplantation may render early detection of such calculi more difficult.

¹ Sagalowsky AI, Ranster CW, *et al.* Urological complications in 505 renal transplants with early catheter removal. *J Urol* 1983;129:929-32.

² Bishop MC. Urological complications of renal transplantation. *Br Med J* 1982;285:394-5.

³ Helling TS, Thomas CU, Moore JD, Koontz PG Jr. The surgical approach to obstructive problems of the transplant ureter. *Transplant Proc* 1982;14:751-60.

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Hyponatraemia induced by a combination of hydrochlorothiazide and triamterene

Hyponatraemia has become recognised as an adverse effect of treatment with diuretic drugs.^{1,2} The combination of hydrochlorothiazide and amiloride has been particularly blamed in several reports,^{3,4} and Brooks and Ritch suggested that amiloride might have a synergistic effect with the thiazide in producing the effect,⁴ although the mechanism of any such synergism is not clear. Potassium supplements are relatively ineffective in preventing hypokalaemia caused by diuretics, and this has given rise to a trend towards prescribing combinations of potassium wasting and potassium sparing diuretics. Whether this trend will lead to an increase in the incidence of hyponatraemia is of great interest. We report a case of hyponatraemia related to the use of a combination of hydrochlorothiazide and triamterene.

Case report

A 44 year old woman with epilepsy was admitted as an emergency in a confused and agitated state. She was receiving phenytoin 300 mg a day, sodium valproate 500 mg four times a day, and a combined preparation of hydrochlorothiazide 25 mg and triamterene 50 mg (Dyazide, one tablet daily). Diazepam, piroxicam, and sex hormone replacement treatment had been stopped several weeks before admission. She had been taking diuretic drugs, including bendroflumazide and cyclopenthiiazide, regularly for several years for ankle swelling and shortness of breath. She had also received various non-steroidal anti-inflammatory agents and anticonvulsants but not carbamazepine. She experienced epileptic fits about once every month.

During the few days before admission she had become increasingly vague and confused. At the time of admission she was intermittently aggressive and unresponsive to verbal command and required parenteral sedation for management. Focal and postictal signs were absent, and there were no signs of cardiac failure or oedema. Blood pressure was 159/80 mm Hg supine with no postural change. On admission plasma concentrations were sodium 117 mmol(mEq)/l, chloride 74 mmol(mEq)/l, potassium 2.8 mmol(mEq)/l, bicarbonate 29 mmol(mEq)/l, and urea 5.4 mmol/l (32.4 mg/100 ml). Results of thyroid function tests and plasma cortisol concentration were normal. Plasma phenytoin concentration was at the lower end of the therapeutic range (11 µmol/l (27.75 µg/100 ml)), and no sodium valproate was detectable in the plasma. A chest radiograph was clear.

Management consisted simply of restricting fluid to 500 ml a day and stopping the diuretic. She was fed normally. A 24 hour urine collection, started on the day after withdrawal of the diuretic drug, contained less than 2 mmol sodium and 18 mmol potassium and showed a creatinine clearance of 92 ml/min. Over the next three days her electrolyte abnormality corrected and she became lucid and returned to normal both mentally and physically.

Comment

We are confident that this patient's presenting syndrome was hyponatraemia induced by the diuretic that she was taking. The mode of presentation and the clinical and biochemical findings were similar to those previously reported. The speed with which the effect was reversed and the results of our investigations excluded other causes of hyponatraemia and the syndrome of inappropriate secretion of anti-diuretic hormone.

The main point of interest in this case is the drug treatment. The diuretic drugs taken previously by the patient had not caused appreciable electrolyte abnormalities but were not combinations of proximally and distally acting drugs. Thus the combination of triamterene and hydrochlorothiazide may well have been responsible for the profound electrolyte abnormality. This supposition is supported by one well documented case report of the occurrence of hyponatraemia related to this drug combination.⁵ On the other hand, Brooks and Ritch suggested that amiloride is specifically implicated.⁴

The exact mechanism by which this adverse effect of diuretic treatment arises remains a source of debate. The most plausible explanation is that a minor degree of sodium depletion together with the drugs' actions in the diluting segment of the nephron impair the capacity to excrete free water.^{2,3} Hypokalaemia has also been implicated in exacerbating the effect. Why, then, the addition of a potassium sparing diuretic should potentiate the problem remains a mystery.

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² Roberts CJC, Mitchell JV, Donley AJ. Hyponatraemia: adverse effect of diuretic treatment. *Br Med J* 1977;1:210.

³ Sunderam SG, Mankikar GD. Hyponatraemia in the elderly. *Age Ageing* 1983;12:77-80.

⁴ Brooks RWS, Ritch AES. New drugs: modern diuretic treatment. *Br Med J* 1983;286:1971-2.

⁵ Hamburger S, Koprivica B, Ellerbeck E, Covinsky JO. Thiazide-induced syndrome of inappropriate secretion of anti-diuretic hormone. *JAMA* 1981;246:1235-6.

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