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Hypertensive encephalopathy complicating transplant renal artery stenosis

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Summary

A 26-year-old female diabetic patient developed hypertensive encephalopathy with gross neurological abnormalities complicating renal artery stenosis of her transplant kidney. The elevated blood pressure was unresponsive to medical treatment. Surgical correction of the stenoses in the renal artery cured the hypertension and renal failure and led to the patient's complete recovery.

KEY WORDS: diabetes mellitus, renal failure, gastrointestinal haemorrhage.

Introduction

Hypertension is a frequent complication of renal transplantation. Transplant renal artery stenosis has a reported incidence ranging from 3 to 12% of cases of hypertension (Lacombe, 1975; Lindsey *et al.*, 1975; Munda *et al.*, 1977; Smellie, Vinik and Hume, 1969). Although the causes are multiple the differential diagnosis between rejection of the graft and stenosis of the renal artery is the most important and difficult to establish. Rejection itself may lead to stenosis of the renal artery (Doyle *et al.*, 1975; Kauffman *et al.*, 1977). In this case report an unusual presentation of renal artery stenosis in the transplanted kidney and its surgical treatment is described.

Case report

A 26-year-old woman with chronic renal failure due to insulin-dependent diabetes mellitus received a

live-related kidney transplant from the patient's father in November 1980. An end-to-end anastomosis of the renal artery to the recipient internal iliac artery was performed. At the time of operation both arteries were noticed to be very atheromatous. The patient's postoperative recovery was uneventful and renal function quickly returned to normal (plasma creatinine concentration 125 $\mu\text{mol/l}$). In February 1981, the patient was admitted having suffered an epileptic seizure of grand mal type, at which time her blood pressure was raised at 180/110 mmHg. The patient's immunosuppression on admission was prednisone 20 mg/day and azathioprine 150 mg/day. Renal function was initially unchanged and no proteinuria was detected. However over the next few days the plasma creatinine concentration progressively increased from 106 to 290 $\mu\text{mol/l}$. A renal biopsy was considered to be contraindicated by the degree of hypertension but methylprednisone, 1 mg daily for 3 days, was added to the patient's baseline immunosuppression. On the third day after admission the patient developed severe upper abdominal pain associated with several episodes of haematemesis and melaena. Investigations showed that the haemoglobin had decreased from 10.8 g/dl on admission to 5.5 g/dl and that there was a marked disturbance in the patient's diabetic state. The blood glucose concentration had increased to 54 mmol/l (972 mg/100 ml) and blood urea concentration had increased to 51.0 mmol/l (306 mg/100 ml). The patient was rehydrated, the hyperglycaemia was controlled with an intravenous infusion of insulin and the patient was transfused four units of blood. Subsequent endoscopy showed severe inflammation of the oesophageal and gastric mucosae with several shallow ulcers on the lesser curvature of the stomach; the duodenum was normal. Intravenous therapy with

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ranitidine, a new H_2 -receptor antagonist, was instigated.

Over the next 24 hr the patient became increasingly more drowsy, developing neck stiffness, photophobia and a mild hemiparesis with bilateral extensor plantar responses. The patient became oliguric. Examination of the fundi showed a severe diabetic retinopathy with some new haemorrhages and the blood pressure was 230/170 mmHg. There was no bruit or abdominal tenderness over the transplant kidney. Two lumbar punctures were performed to exclude intracerebral bleeding as a cause for the neurological signs and they were both normal. During this time the blood pressure remained very difficult to control and was eventually lowered to 170/80 mmHg by a combination of repeated boluses of intravenous diazoxide and infusions of labetalol, 2 mg/min, and sodium nitroprusside, 1.5 μ g/kg/min. During the following 3 days the patient remained very drowsy with labile blood pressure recordings and her renal function continued to deteriorate (plasma creatinine 390 μ mol/l, blood urea 53 mmol/l). Hyperkalaemia then developed and peritoneal dialysis was consequently started. Although excess body fluid was removed, the patient remained hypertensive. A renal arteriogram was performed and demonstrated significant stenoses at the origin of the right internal iliac artery and at the site of anastomosis of the transplant and internal iliac arteries (Fig. 1). At operation the tight renal artery

stenosis was confirmed and repaired using a saphenous vein patch graft from the common iliac artery along the length of the internal iliac artery to the renal artery. Postoperatively the patient rapidly improved with full recovery of her level of consciousness and complete resolution of abnormal neurological signs. The blood pressure stabilized at 140/80 mmHg requiring only oral propranolol therapy and renal function also returned to normal (plasma creatinine 105 μ mol/l, blood urea 9.4 mmol/l).

Discussion

The aetiology of renal artery stenosis in the transplanted kidney includes atheromatous plaques, external compression, malrotation, technical failure and most commonly intimal hyperplasia of the donor vessel distal to the anastomosis which has been postulated to be secondary to rejection. It is the differential diagnosis between rejection of the graft and stenosis of the renal artery which is critical. Both may have similar presentations but require very different treatments. Surgical correction of the stenosis has been shown to have a high rate of success and some authors have suggested early renal arteriography for those patients with persistent hypertension despite adequate medical treatment and in the absence of overt signs of rejection. The incidence of renal artery stenosis varies considerably (Lacombe, 1975; Lindsey *et al.*, 1975; Munda *et al.*, 1977; Smellie

FIG. 1. Renal arteriogram of transplant kidney showing stenoses at the origin of the right internal iliac artery and at the site of anastomosis of the transplant and internal iliac arteries.

et al., 1969) and depends to some extent on the type of arterial anastomosis. Bewick *et al.* (1976) reported an overall incidence of 2.8%, but this was considerably higher (6.1%) in end-to-end anastomoses. Hypertension associated with stenosis of the renal artery is characteristically refractory to medical treatment, slow in onset, associated with an abdominal bruit and resulting in only moderate and gradual loss of renal function. In this report the diagnosis was delayed due to an absence of an abdominal bruit on auscultation possibly related to the severity of the stenosis and by the rapid development of malignant hypertension with complicating encephalopathy and loss of renal function.

Delayed diagnosis and treatment may have serious consequences for the survival of the graft although some investigators have reported that renal artery stenosis may reverse spontaneously (Steensma-Vegter *et al.*, 1981). Dipyridamole has been shown to afford protection against distal segmental renal arterial stenosis by inhibiting platelet aggregation and intravascular fibrin deposition suggesting segmental stenosis may be a manifestation of rejection (Kauffman *et al.*, 1977). The renal artery stenosis in this case was due to atheromatous plaques. It is of interest that renal artery stenosis and gastrointestinal haemorrhage have been previously reported in association (Lee *et al.*, 1972).

In conclusion we have described a diabetic patient with an unusual presentation of renal artery stenosis in the transplant kidney manifested by hypertensive

encephalopathy and severe loss of renal function. Surgical correction of the stenosis reversed both the hypertension and renal failure and the patient made a complete recovery from the neurological complications.

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