CLINICAL REPORTS

Hypertensive encephalopathy complicating transplant renal artery stenosis

R. J. S. McGONIGLE*  
M.R.C.P.

J. A. P. TRAFFORD†  
F.R.C.P.

M. BEWICK  
F.R.C.S.

V. PARSONS  
D.M., F.R.C.P.

King’s College Hospital Renal Unit, Dulwich Hospital, London SE22 and †Renal Unit, Royal Sussex County Hospital, Brighton BN2 5BE

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 μmol/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 μmol/l. A renal biopsy was considered to be contraindicated by the degree of hypertension but methylprednisone, 1 g 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
ranitidine, a new H₂-receptor antagonist, was insti-
gated.

Over the next 24 hr the patient became increas-
ingly more drowsy, developing neck stiffness, photo-
phobia and a mild hemiparesis with bilateral exten-
sor 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 µg/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 µmol/l, blood urea 53
mmol/l). Hyperkalaemia then developed and perito-
neal 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 saphe-
nous 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 conscious-
ness and complete resolution of abnormal neurologi-
cal signs. The blood pressure stabilized at 140/80
mmHg requiring only oral propranolol therapy and
renal function also returned to normal (plasma
creatinine 105 µmol/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 steno-
sis has been shown to have a high rate of success and
some authors have suggested early renal arteriogra-
phy 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

![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.](http://pmj.bmj.com/)

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.

References


(Accepted 25 April 1983)
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Postgrad Med J 1984 60: 356-358
doi: 10.1136/pgmj.60.703.356

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