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Percutaneous transluminal angioplasty in a transplanted kidney with fibromuscular dysplasia

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Sir: This is a report of incidental findings of fibromuscular dysplasia (FMD) in a living related donor kidney recipient who developed recurrent stenosis of the renal artery anastomosis.

The kidney recipient was a 28-year-old woman with chronic renal failure due to chronic membranous glomerulonephritis. She received a kidney from her 60-year-old mother. The donor had no history of arterial hypertension. During the preoperative donor work-up, renal arteriography demonstrated the medial form of FMD in both main renal arteries, but the arteriogram was misinterpreted as normal. The pre-existent FMD of the renal artery was discovered 2 years after transplantation. The patient had stable and well-preserved renal function for 2 years.

Twenty-five months after transplantation, renal function deteriorated and blood pressure increased from 140/85 mmHg to 190/120 mmHg. At that time arteriography revealed a tight stenosis of the right hypogastric artery at the site of anastomosis and FMD in the distal part of the renal artery. The severity of FMD was compared with that demonstrated by donor angiography before transplantation. The decision was made to dilate only the stenosis at the anastomosis. The lesion was dilated with a 6-mm high-pressure balloon catheter. Following percutaneous transluminal angioplasty (PTA), renal function improved: serum creatinine fell from 350 μmol/l before dilatation to 220 μmol/l 3 and 6 months after PTA. Three months after PTA, the patients blood pressure was 150/90 mmHg and it was well controlled with low doses of oral hypotensive drugs. She was fully ambulatory when discharged from the hospital.

Twenty months later the patient was readmitted to the hospital with a decreased urine output, high blood pressure (180/120 mmHg), and a raised serum creatinine concentration of up to 487 μmol/l. Renal angiography of the transplanted kidney confirmed a 60 % stenosis of the renal artery at the site of the anastomosis and demonstrated a typically "beaded" appearance of the distal part of the artery. Following dilatation of both lesions with a 7-mm balloon catheter, the renal artery was widely patent. Doppler ultrasound and clinical examination 6 months after the last PTA showed no recurrence of stenosis. The patient recovered completely. Her blood pressure has been normal and her serum creatinine stable at about 120 μmol/l.

Since transplantation the donor has been well and her blood pressure has remained well under control. The donor's last blood pressure, recorded 6 years after transplantation, was 150/90 mmHg.

McCormack's classification of FMD includes: intimal fibroplasia, medial fibroplasia, subadventitional fibroplasia, and fibromuscular hyperplasia [3, 4]. According to Meaney et al., the medial form of FMD is the most common variant of this disease; the symptoms are frequently mild and can even be absent [5]. FMD is a relatively frequent disease, occurring in 2 %–3 % of potential kidney donors; and it is more common in females than in males, [9, 10]. The medial form of FMD, although associated with a higher rate of transplantation complications, is not regarded as an absolute contraindication to transplantation [2]. Linder et al. [2] and Nghiem et al. [6] have recommended resection of the stenotic part of the renal artery prior to transplantation. Most symptomatic patients with the medial form of FMD show good initial and long-term response to PTA [1]. In transplant renal artery stenosis, PTA is the treatment of choice because it carries a lesser risk for the allograft than does surgical treatment [7, 8].

The question arises, however, as to whether the PTA technique, which has proven successful in the treatment of renal artery stenosis due to FMD, can also be applied to a lesion in a transplant renal artery. A review of the literature shows that four cases of FMD in a renal allograft have been reported. One of the grafts was from a living related donor and three were from cadaveric donors [2, 6]. To our knowledge, only one case of FMD treated by PTA in a renal allograft has been reported [3]. The case described indicates that PTA is a clinically effective procedure for the treatment of renovascular hypertension due to FMD in a transplanted kidney.

There are several lessons to be learned from that case, the most important of which is that donor angiograms should be carefully reviewed by the entire transplant team; one should not solely rely on the radiologists report. FMD of renal arteries in an asymptomatic or symptomatic living donor with bilateral changes should be regarded as an absolute contraindication to transplantation. The decision is more difficult in a living donor with unilateral changes. We believe that one should never consider using such a kidney from a young potential donor when the possibility exists that FMD might also occur on the contralateral side.

However, we think most would agree that FMD in only one kidney from an older donor could be used with excision of the diseased area and reconstruction of the renal artery.

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Victims of cyanide poisoning make suitable organ donors

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Sir: Renal transplantation is the elective treatment for a majority of patients suffering from end-stage renal disease. Despite the progressive growth in the number of renal transplantations presently being performed in most developed countries, the gap between the number of patients receiving a renal graft and the number of those on the waiting list is increasing. This shortage of organs makes it necessary for us to consider any and all potential donors and then to carefully evaluate each one. We report here on the results at 24 months of two renal transplantations performed using the kidneys from a donor who died of cyanide poisoning.

The donor was a 56-year-old male with no other significant past medical history than a long-standing depressive syndrome and two prior attempts at suicide. The patient was brought to the emergency room 20 min after a respiratory arrest occurred at his home as a consequence of the ingestion of a substance later identified as potassium cyanide.

Cardiorespiratory resuscitation techniques resulted in the rapid recovery of satisfactory cardiac function, although secondary, irreversible, hypoxic brain lesions ensued. We did not find the patient to be a suitable candidate for dicobalt edetate treatment and so we decided to implement the potential donor maintenance protocol. The donor rapidly reached hemodynamic stability with blood pressure of 140/80 mmHg and diuresis between 1 and 2 ml/min. Glycemia, electrolytes, GOT, GPT, hemogram, and coagulation parameters were normal and remained stable. Serum creatinine and urea were 1.3 mg/dl and 50 mg/dl, respectively. A qualitative determination of blood cyanide was negative 24 h later. Both kidneys were removed and preserved with Euro-collins solution.

The recipients of the kidneys were two men, aged 27 and 28 years, with end-stage renal disease secondary to IgA nephropathy and nephroangiosclerosis, respectively. Cold ischemia times were 16 h and 27 h, respectively. Both of the recipients were negative for lymphocytotoxic antibodies. They had three and two HLA antigen mismatches, respectively, with the donor. Immunosuppression consisted of prophylactic OKT3 and cyclosporin with low doses of prednisone. Both had immediate good renal function and were uneventfully discharged 9 days post-transplantation. Twenty-four months later, the first recipient has a serum creatinine of 1.6 mg/dl. The second recipient went back to dialysis 9 months after transplantation because of an irreversible acute rejection episode related to the patient's own decision to stop the immunosuppressive treatment. His serum creatinine was 2.2 mg/dl when last monitored.

Cyanide intoxication may occur in three different ways [8]: by hydrocyanic acid inhalation, which may prove fatal in less than 1 min [4]; by potassium, sodium, or calcium cyanide ingestion, which shows