Successful revascularization for acute renal allograft thrombosis after 32 hours of ischaemia

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Introduction

Acute thrombosis of the renal transplant artery is a known complication of renal transplantation, with incidences ranging between 0.5 and 3.5% [1–4]. Severe damage can be expected after periods of ischaemia of 1–2 h [5]. We report on one case of successful revascularization by percutaneous transluminal angioplasty (PTA) despite a prolonged period of ischaemia.

Case

A 55-year-old woman had been undergoing haemodialysis since January 1988 for chronic renal failure due to interstitial nephritis. She received a cadaver transplant in May 1989. An end-to-end anastomosis of the donor artery to the recipient left hypogastric artery was performed. The surgeon reported that the transplant artery had atheromatosis. After 5 days of hemodialysis for acute tubular necrosis, renal function improved and stabilized at a serum creatinine of 1.4 mg/dl. Baseline immunosuppressive therapy consisted of azathioprine, prednisone and cyclosporin A. At the end of the first post-operative month, a biopsy-proven acute rejection episode occurred that resolved after a short course of high dose methylprednisolone therapy. The patient was discharged from the hospital in July 1989, with a serum creatinine of 1.1 mg/dl.

In November 1989, she developed hypertension and was treated with beta-blockers and furosemide. On September 30, 1992, she developed acute anuria but came to the hospital only on October 1, 1992, after a period of anuria of 32 h. She was severely hypertensive (180/120 mmHg) and her serum creatinine had risen to 3.9 mg/dl. The graft ultrasonography was unremarkable.

Serum lactate dehydrogenase (LDH) levels were increased to 2710 IU/l (normal <350). A radionuclide DMSA scan disclosed reduced uptake of the isotope by the renal transplant, suggesting severe hypoperfusion. Renal angiography demonstrated a high-grade (90%) stenosis of the donor artery just beyond the anastomosis, with a nearly complete thrombosis (Figure 1).

As there was a minimal residual blood flow, an attempt at renal salvage seemed warranted. The angiographic catheter was exchanged for a 5 mm Bard balloon catheter, which was positioned selectively in the stenotic portion of the renal artery. The balloon was inflated to ~8 atmospheres and then deflated. An immediate post-procedure angiography showed that the PTA was successful (residual stenosis <50%).

Discussion

Acute deterioration of renal allograft function most commonly is due to rejection. However, renal artery stenosis also may lead to allograft renal failure and mimic acute or chronic rejection. According to published series, renal artery stenosis is reported in 8.5% of patients—ranging between 3.3 and 12.4% [2,3,6,7]. Stenosis may result from atheromatous narrowing of the host internal iliac artery, kinking attributable to...
Fig. 2. After PTA, only mild residual stenosis remains. The procedure was complicated by an infarction of the upper pole (arrow).

Hypertension in renal allograft recipients is due to multiple causes [10]; stenosis of the graft artery is responsible for 3–12% of post-transplant arterial hypertension [6–9]. The percentage reduction of the arterial lumen at which a stenosis becomes haemodynamically significant is unknown; reported figures vary from 50 to 80% [9].

Our patient presented several risk factors: immunosuppression with cyclosporin, pre-existing arterial hypertension and atheromatosis, previous allograft rejection and end-to-end anastomosis of the donor renal artery. Renovascular disease was suspected on the basis of rapidly increasing arterial hypertension and because acute anuria developed without any evidence of obstructive uropathy.

Angiography was the essential diagnostic procedure that also allowed immediate revascularization. The site of the arterial puncture for PTA varies according to the type of anastomosis. The stenotic site is approached and/or perfusion, periarterial fibrosis or immune injury (rejection) [8]. In addition to hypertension and impaired function of the allograft, poor flow through a stenotic renal artery may also lead to thrombosis. The reported incidence of transplant arterial thrombosis is 1.8% and ranges between 0.5 and 3.5% [1–4]. Eighty per cent of the thromboses occur within the first month and 93% within the first year after transplantation [4].

Most interestingly, in the present case, PTA was successful despite a period of anuria of 32 h. Occlusion of the artery was almost complete, but angiography had demonstrated some residual arterial flow around the thrombus into the renal parenchyma.

PTA is the first choice treatment of renal graft artery stenosis (75%) [7,11,12] because of its technical simplicity, good tolerance and effectiveness, whereas surgery
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is associated with significant morbidity and mortality (5%) [10–13] and prolonged hospitalization time. Complications of PTA include arterial spasm, intimal flap, extravasation of radiocontrast, haematomas, small segment infarctions, arterial thrombosis [1,2,12–14] and dissection by the balloon catheter [12,15,16]; no deaths have been reported. Complications appear to be less frequent after angioplasty than with surgery [12,15,16].

In conclusion, angioplasty of transplant renal artery stenosis is safe and effective and should be the procedure of choice for treatment of hypertension refractory to medical treatment and/or decline in renal function due to transplant renal artery stenosis.

References


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