Resistant hypertension after kidney transplantation

Roberta Callus,^{1,2} Maria Bugeja,¹ Lara Delicata,¹ Adrian Mizzi³

DESCRIPTION

¹Department of Medicine, Mater

²Faculty of Health and Science,

Teaching University of Liverpool.

Dei Hospital, Msida, Malta

Institute of Learning and

³Department of Radiology,

Mater Dei Hospital, Msida,

Correspondence to

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Dr Roberta Callus, robertacallus@yahoo.co.uk

Liverpool, UK

Malta

A 50-year-old man with known hypertension, diabetes controlled on diet and end-stage kidney disease of unknown aetiology received a donor after brain death renal allograft from an extended criteria donor. There was initial delayed graft function, followed by a reduction in serum creatinine to a nadir of 115 μ mol/L. Maintenance immunosuppression included tacrolimus, mycophenolic acid and corticosteroids.

Four months following transplantation, he presented with worsening blood pressure control and bilateral lower limb oedema. This occurred despite escalation of antihypertensive medication, which included a calcium channel blocker, loop diuretic, beta-blocker and alpha-blocker. Compliance to treatment was ensured. Ambulatory blood pressure monitoring recorded a mean blood pressure of 168/98 mm Hg with no nocturnal dipping. Serum creatinine at this stage was 130 μ mol/L. On introduction of an ACE inhibitor, creatinine rose by approximately 30% to 179 μ mol/L. A faint bruit was audible over the allograft. In view of this, CT renal angiography was performed (figure 1).

Percutaneous angioplasty and stenting was undertaken (figure 2). Serum creatinine fell to $120 \,\mu$ mol/L



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Figure 1 Multiplanar oblique reconstruction of the CT renal angiography confirmed a tight focal stenosis approximately 2.5 cm distal to the anastomosis of the transplant artery to the right external iliac artery (arrow).

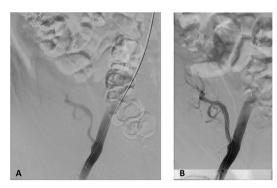


Figure 2 Transplant renal artery stenosis as visualised on angiography (**A**) was subsequently ballooned and stented successfully via a percutaneous approach through the right femoral artery (**B**).

after 6 months. Repeat ambulatory blood pressure monitoring gave a mean blood pressure reading of 130/85 mm Hg, although he continued on the same antihypertensive therapy.

Transplant renal artery stenosis (TRAS) is a recognised cause of post-transplant refractory hypertension, renal dysfunction and allograft loss. The reported incidence varies from 1% to 23%.^{1 2} Presentation most commonly occurs in the first 6 months following transplantation but may present at a later stage.¹ Hypertension and fluid retention secondary to activation of the reninangiotensin–aldosterone system are characteristic findings. Prompt diagnosis is crucial since it is potentially curable.

Comorbidities associated with TRAS include diabetes mellitus, hypertension, hyperlipidaemia and smoking.³ Proposed causes include donor or recipient artery atheroma, arterial trauma during procurement or transplantation, different suture techniques with end-to-end anastomosis being more liable to error, immune-mediated vascular damage, prolonged cold ischaemia time and rarely extrinsic mechanical compression.¹ Cytomegalovirus infection and delayed graft function are considered to be risk factors.^{2–4}

Duplex ultrasonography is a useful non-invasive screening tool for TRAS; however, it remains operator-dependent and transplant vessels may be difficult to visualise. Indeed, our patient was initially screened with Doppler ultrasound of the allograft; however, it was non-diagnostic in this case. More accurate tools for diagnosis include CT or magnetic resonance angiography.

Treatment includes conservative medical therapy, percutaneous transluminal angioplasty (PTA) or surgical therapy. In cases with uncontrolled hypertension, deteriorating renal function or worsening stenosis, revascularisation is advised.¹ PTA with stent

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placement remains the treatment of choice despite no randomised trial comparing its efficiency and safety with other treatment options.^{1 2} Potential complications include renal artery dissection, stent restenosis, thromboembolism, haematoma, stent migration, pseudoaneurysms, nephrotoxicity and rarely allograft loss.³ Surgical correction is technically challenging and is considered a rescue therapy when PTA fails or if severe kinking is present.²

Learning points

- Transplant renal artery stenosis should be suspected in all patients with resistant hypertension following renal transplantation.
- Early recognition and prompt intervention in selected cases will lead to improved outcomes.
- Percutaneous transluminal angioplasty is the preferred treatment, but surgical correction may be required for technically challenging cases.

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Competing interests None declared.

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