

P205

P205-Imaging challenges in transplant renal artery stenosis

Doctor David Baird¹, Doctor Jac Williams¹, Doctor Michaela Petrie¹, Doctor Shona Methven², Doctor Laura Clark², Professor Lorna Marson¹, Doctor Andrew Walker¹, Doctor James Smith²

¹Royal Infirmary of Edinburgh, Edinburgh, United Kingdom, ²Aberdeen Royal Infirmary, Aberdeen, United Kingdom

We present the case of a 47-year-old female with IgA nephropathy and controlled hypertension, who received a pre-emptive, DBD renal transplant from an 18-year-old female donor with a 0-0-1 mismatch and a cold ischaemic time of 20 hours. The donor died from anaphylactic shock, with no other medical history. In the context of initial delayed graft function, a doppler ultrasound (US) showed good transplant perfusion and graft biopsy on day 7 post transplant showed acute tubular necrosis only. Transplant function improved with a creatinine of 109micromol/L by day 32.

At routine post-transplant reviews she was well but noted to have hypertension and antihypertensives were titrated. Four months post-transplant she developed significant peripheral oedema with uncontrolled hypertension, severe headache, vomiting and transplant dysfunction. There was no audible bruit. Serum creatinine rose to 236micromol/l, urinary protein:creatinine ratio was 716mg/mmol and serum albumin fell to 29g/l. Initial US doppler imaging showed good perfusion to the graft, however a repeat scan showed low resistive indices and a flat Doppler trace. MR renal angiogram showed normal surgical anastomosis, a small filling defect at the renal hilum but good distal perfusion. Subsequently CT angiogram showed a caliber change at the renal hilum strongly suggestive of a distal transplant artery stenosis.

On full dose of amlodipine, doxazosin, bisoprolol, furosemide 80mg and methyldopa 250mg tds she remained hypertensive with a blood pressure (BP) of 170/109mmHg. In the context of nephrotic range proteinuria and transplant dysfunction, the patient underwent a transplant biopsy which showed acute tubular injury and mild microvascular disease but no other pathology. Subsequent invasive angiography demonstrated a smooth stenosis from the mid to distal portion of the transplant artery (figure 1) and angioplasty to 3.2mm was performed. This led to an immediate improvement in blood pressure and creatinine dropped from 216 pre-procedure to 157micromol/l after 12 hours and 131micromol/l at discharge four days later.

Discussion

Transplant Renal Artery Stenosis (TRAS) presents with hypertension, salt and water retention and transplant dysfunction in the first six months following transplant and is associated with an increased risk of graft loss and death¹. Most commonly it occurs close to the site of surgical anastomosis and relates to the suture line or post anastomotic turbulence of blood flow. Atherosclerotic disease may be evident in older donors, or develop many years post-transplant. The unusual distal location and relative length of stenosis in this case raises the possibility of fibromuscular dysplasia (FMD), a non-atherosclerotic, non-inflammatory disease which may have been pre-existent, but not yet clinically apparent in the 18-year-old donor. Assessment of living donors has shown that up to 2.6% had CT angiographic evidence of FMD, of whom 87% were female². Traction injury of the transplant artery at time of retrieval can cause distal transplant artery narrowing but would be expected to present earlier than this case³.

This case highlights the challenges of US Doppler imaging which may miss a mid or distal TRAS. Clinically silent donor FMD may have been responsible and may be more common in cadaveric donors than previously appreciated.