Transplant renal artery stenosis: Narrow and prone to closure

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Renal transplant dysfunction: over-reliance on subjective investigations can contribute to diagnostic uncertainty.

Background

Complete anuria is an uncommon clinical presentation that is usually due to an obstructive (postrenal) cause. Less frequent aetiologies include catastrophic vascular compromise (prerenal), massive rhabdomyolysis or antiglomerular basement membrane disease (intrinsic renal).[1] Two-dimensional ultrasonography is the initial imaging investigation of choice in these patients, as it is non-invasive, can identify the presence of obstruction (manifesting as hydronephrosis) and when combined with colour Doppler can provide information on renal perfusion.[2] We present a case of severe transplant renal artery stenosis causing acute renal failure with complete anuria, where a premature closure in diagnostic reasoning caused a delay in diagnosis. We discuss the approach to formulating the differential diagnosis in patients presenting with anuria, the potential limitations of ultrasonography and the value of multidisciplinary team meetings in preventing diagnostic errors.

Case report

A 44-year-old renal transplant patient with a baseline creatinine of 220 μmol/L self-presented on a Friday evening with 24 h of complete anuria. He had developed end stage renal failure from reflux nephropathy 13 years previously and received a pre-emptive deceased donor transplant with a 0-0-0 mismatch. Three years prior to the current presentation, a transplant biopsy performed for a 'creeping creatinine' revealed changes consistent with calcineurin-inhibitor toxicity and as a result, his ciclosporin was stopped and azathioprine switched to mycophenolate mofetil with stabilization of his graft function. His medical history included hypertension requiring four agents and anaemia treated with darbepoeitin.

On examination he was alert, oriented and appeared comfortable. His pulse was 60 beats per minute and regular, and his blood pressure 129/73. Dual heart sounds were auscultated, with no additional sounds or murmurs present. There was mild peripheral oedema bilaterally. His chest was clear and oxygen saturation was 99% on room air. Examination of the abdomen revealed a non-tender transplant kidney in the left iliac fossa, with no masses or organomegaly. The bladder was not palpable. Normal bowel sounds were audible with no bruits evident. His creatinine was elevated at 500 μmol/L.

As postrenal (obstructive) aetiologies are the most common cause for complete anuria, an urgent ultrasound scan was obtained and reported to demonstrate an 11 cm transplant kidney, no hydronephrosis and Doppler evidence of perfusion (Figure 1). His renal function continued to worsen and, due to refractory hyperkalaemia, he was commenced on dialysis. As it was early in the course of obstruction, dilation of the ureters or renal pelvis may not be obvious.[3] The following day a non-contrast computed tomography scan was requested; however, there was no

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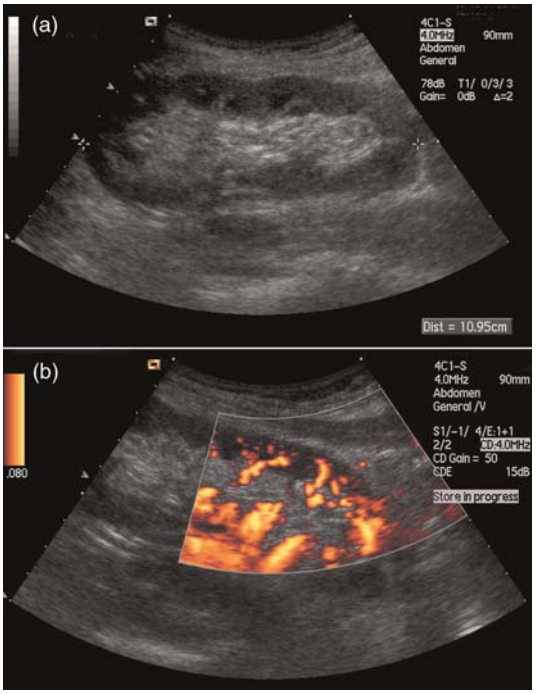
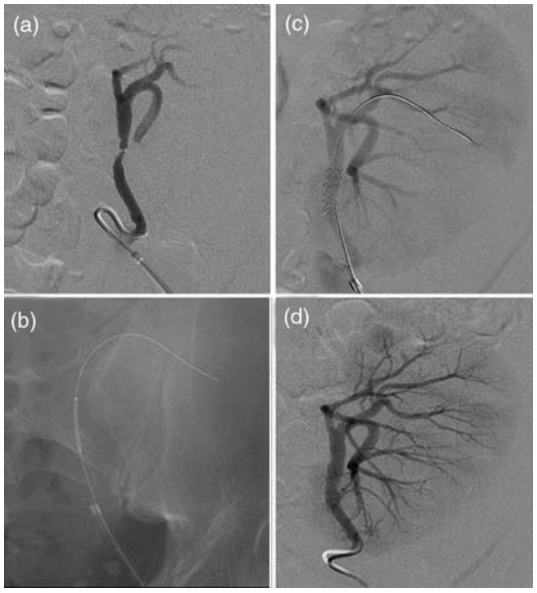
Figure 1
Ultrasound scan from the day of admission. (a) Two-dimensional ultrasound and (b) Doppler imaging of the transplant



evidence of obstruction and no cause for his anuria was identified. As the diagnosis remained unclear, a transplant biopsy was performed, revealing moderate interstitial fibrosis and tubular atrophy (similar appearance to the biopsy three years earlier), but otherwise normal appearing nephrons. He remained anuric and dialysis dependent.

On the third day of his admission, his imaging was presented at a radiology multidisciplinary team meeting attended by subspecialists in uroradiology and interventional radiology. Review of the ultrasound images revealed that power Doppler (Figure 1b) had been used by the sonographer to obtain adequate perfusion images, a finding consistent with impaired perfusion. A repeat ultrasound using colour Doppler, confirmed reduced flow to the kidney with an increased peak systolic velocity proximal to the renal pelvis, suggestive of transplant renal artery stenosis.

Figure 2
(a) Selective catheterization of the transplant renal artery showing a high-grade stenosis. (b,c) Balloon angioplasty and stenting of the stenotic segment. (d) Poststenting appearance



Urgent angiography verified the presence of high-grade transplant renal artery stenosis, and the patient proceeded to angioplasty and stenting (Figure 2). Within 15 min of the procedure he began producing urine, and within 24 h his creatinine level had improved to 236 μmol/L and his antihypertensive regimen could be reduced. His transplant function remains stable with a most recent creatinine level of 173 μmol/L, 15 months postprocedure.

Discussion

The differential diagnoses for a patient presenting with renal impairment are traditionally considered anatomically and divided into prerenal, intrinsic renal or postrenal aetiologies.[2] Although postrenal causes are the most common explanation for complete anuria, in part reflecting the relative probability of occluding one tube (the urethra) versus two tubes (the ureters or renal arteries), when there is only a single functioning kidney present, these *a priori* probabilities change

and a higher index of suspicion for prerenal causes should be entertained.

Although the admitting team considered a renovascular cause because of the patient's history of hypertension, this was (prematurely) excluded from the differential diagnosis due to the absence of a bruit on auscultation and the initial ultrasound report, which described normal perfusion. However, bruits are an inconstant finding in transplant artery stenosis[4,5] and adequate assessment of the vasculature by ultrasound is technically challenging and highly operator dependent.[6] Although the incidence of functionally significant transplant renal artery stenosis varies widely between studies due to the different imaging modalities used, it has been reported to be as high as 23%.[7] While colour Doppler ultrasound has a sensitivity and specificity for the detection of transplant renal artery stenosis of between 87 and 94%, and 86 and 100%, respectively,[8] the gold standard remains renal angiography.[9]

Fortunately, like many units, we hold regular multidisciplinary team meetings to review the results of imaging investigations and discuss complex cases. At this meeting, the abnormal perfusion in the initial ultrasound was readily identified; repeat imaging promptly organized and a therapeutic intervention undertaken. The rapid return of renal function following the angioplasty and stenting, highlights the delicate balances that exist within the kidney, where sufficient perfusion had been maintained to prevent significant tubular injury (which would have been evident on the biopsy), but was insufficient to establish pressures necessary for glomerular filtration.

Diagnostic reasoning is a complex process with premature closures, the exclusion of diagnoses without sufficient verification being the commonest cause of diagnostic error.[10] Our case highlights the need to appreciate the limitations of investigations that are requested, the importance of differentially weighting diagnoses depending on clinical circumstances and the value of external discussion and re-evaluation of previously discarded diagnoses when diagnostic uncertainty persists.

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