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PICTORIAL ESSAY

Pictorial Review of Paediatric Renal Transplant Vascular Complications

CWK Ng[1], JTH Yeung[2], KNY Pan[1], WH Luk[1], DCY Lui[1], ALT Ma[3], PC Tong[3], THF Chan[1]

[1]Department of Radiology, Princess Margaret Hospital, Laichikok, Hong Kong
[2]Department of Radiology, Yan Chai Hospital, Tsuen Wan, Hong Kong
[3]Department of Paediatrics, Princess Margaret Hospital, Laichikok, Hong Kong

INTRODUCTION

Renal transplantation offers the most effective long-term renal replacement therapy for paediatric patients with end-stage renal disease. It is associated with better survival rates and quality of life than dialysis. Given the technically demanding surgery with small-sized vessels, paediatric patients are particularly susceptible to vascular complications. They are an important cause of morbidity after paediatric renal transplantation affecting 5% to 10% of patients.[1] Imaging plays an important role in diagnosing these complications to facilitate timely management. In this article, we will review the imaging findings of paediatric renal transplant-related vascular complications. The vascular complications can be stratified into immediate (within a week), early (between a week to a month), or late (>1 month) [Table].

SURGICAL TECHNIQUE

The graft kidney is placed in the right or left iliac fossa. The graft renal arteries and veins are anastomosed to the ipsilateral common, external or internal iliac arteries and veins.

RENAL ARTERY THROMBOSIS

Renal artery thrombosis is an uncommon yet devastating major cause of early graft loss where flow in both the main and the intrarenal arteries is absent.[2] One-third of early graft loss is due to vascular thrombosis and occurs in 3% to 4% of all paediatric renal transplants.[3] Typically it is due to technical problems such as vessel kinking, dissection, or torsion. Additional risk factors include hypotension, multiple renal arteries, and unidentified intimal flaps, young age of the recipient, young age of the deceased donor, prolonged cold ischaemic time, history of transplantation, and presence of acute tubular necrosis.

Ultrasound is commonly used to diagnose renal infarcts. Renal artery thrombosis is characterised by absent colour flow and spectral Doppler waveforms within the main renal vasculature, associated with absent parenchymal perfusion on colour and power Doppler imaging (Figure 1a, b). Although poor parenchymal perfusion is also seen in hyperacute rejection and renal vein thrombosis (RVT), in renal artery thrombosis a normal

Correspondence: *Dr CWK Ng, Department of Radiology, Princess Margaret Hospital, Laichikok, Hong Kong*
Email: carol26@gmail.com

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Table. Timing of vascular complications after renal transplantation.

Immediate (<7 days)
• Renal artery thrombosis
• Segmental infarct
• Renal vein thrombosis
Early (1 week to 1 month)
• Renal vein thrombosis
Late (>1 month)
• Renal artery stenosis
Biopsy-related
• Arteriovenous fistula

graft artery cannot be identified. An adjunct ultrasound feature of renal artery thrombosis is a low resistive index (RI) <0.6.[4] In cases where ultrasonographic identification of the main renal artery is challenging due to technical factors such as postoperative gas limiting the acoustic window or a lack of operator experience, magnetic resonance (MR) or contrast computed tomography (CT) angiogram can provide a definitive diagnosis. The graft artery will show absent flow signal on MR imaging or a filling defect on contrast CT. Parenchymal perfusion of the graft kidney on CT is also absent (Figure 1c, d). Urgent thrombolysis or thrombectomy may occasionally

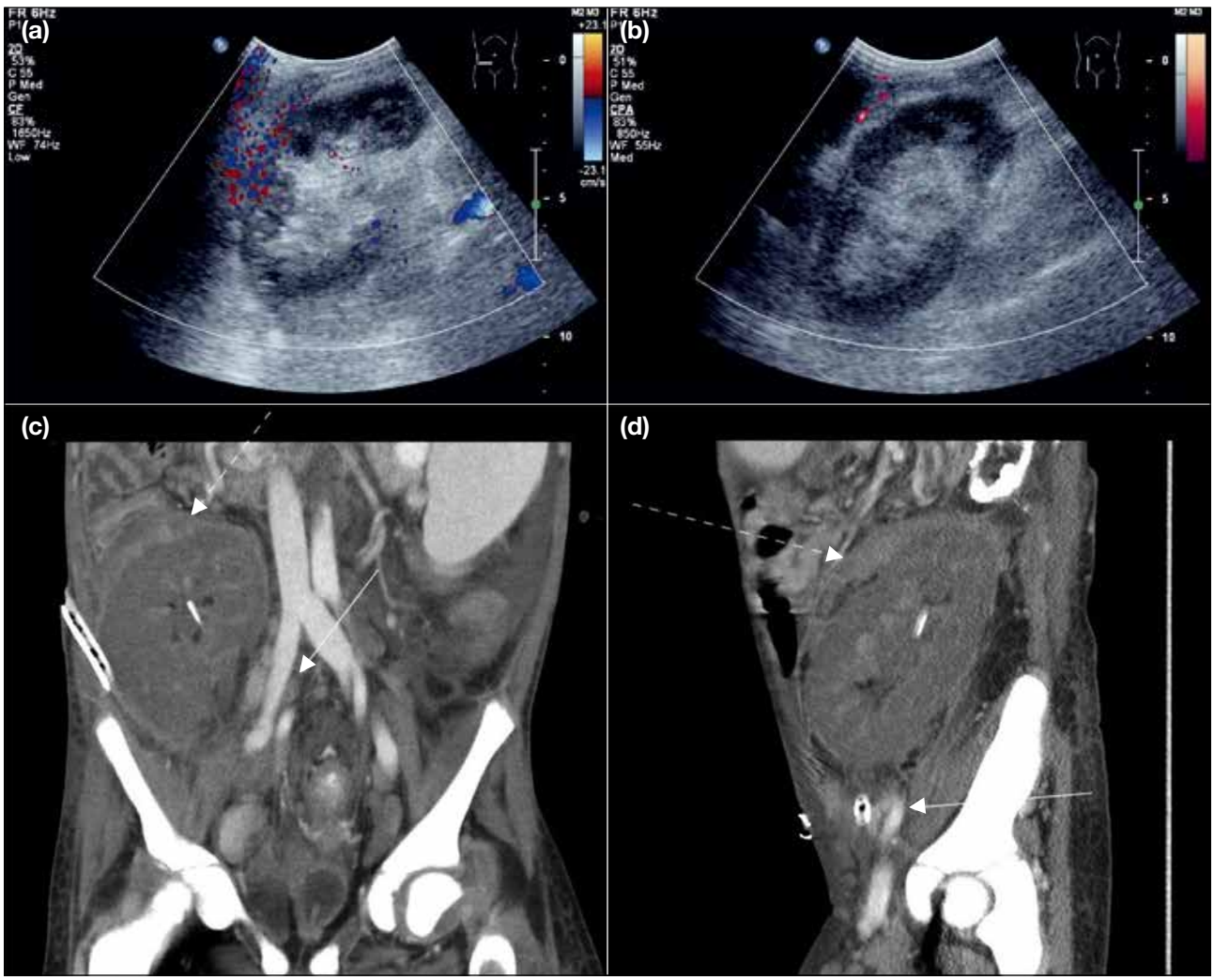

Figure 1. An 11-year-old girl with primary hyperoxaluria and end-stage renal failure complained of intense graft pain on day 10 after surgery; blood test results revealed she was oliguric with deteriorating renal function. Her previous ultrasound scan on day 5 showed normal renal vasculature and flow on colour Doppler. Ultrasound scan on day 10 showed no blood flow in the graft kidney on (a) colour and (b) power Doppler studies. (c, d) Contrast-enhanced computed tomography showed no contrast enhancement in the graft kidney while satisfactory contrast opacification of the inferior vena cava and aorta and bilateral iliac vessels were both seen (solid arrows). A concomitant rim-like hyperdense, non-enhancing mass compatible with a perinephric haematoma is present (dashed arrows). Intraoperative findings confirmed the presence of a perinephric haematoma and thrombosed graft kidney artery and vein with global infarction. The kidney was non-salvageable and graft nephrectomy was performed.

salvage the kidney, but nephrectomy due to graft failure is the more likely outcome.

SEGMENTAL INFARCT

Segmental infarcts are common in the early postoperative period when the allograft has multiple renal arteries. It is associated with an elevated lactate dehydrogenase in blood tests although patients are often clinically asymptomatic.[5]

On ultrasound, a segmental infarct has the appearance of a wedge-shaped hypoechoic mass with poorly defined margins (Figure 2a) or a hypoechoic mass with a well-defined echogenic wall that shows absent Doppler flow signal. Another cause of a similar grayscale appearance is pyelonephritis, although colour Doppler will show increased flow.

Contrast-enhanced CT or scintigraphic study will show wedge-shaped hypoenhancement or reduced scintigraphic uptake at the segmental infarct (Figure 2b).[6] Contrast-enhanced ultrasound is an alternative to CT in diagnosing segmental infarct with no risk of contrast nephropathy.[7] Most patients with segmental infarct can be managed conservatively.

RENAL VEIN THROMBOSIS

Most cases of RVT occur within the first week of transplantation. Nearly all cases are reported within the first month. The incidence of RVT is 0.1% to 8.2%.[8] Risk factors for developing thrombosis are donor age <6 or >60 years, perioperative or postoperative haemodynamic instability, peritoneal dialysis, history of thrombosis, cadaver organ, cold ischaemic time >24 hours, and history of transplantation.[9] Patients usually present with pain, graft swelling, and oliguria. Timely diagnosis of RVT is crucial as there is a narrow therapeutic window for graft salvage before irreversible ischaemia sets in. Treatment includes prompt thrombolytic therapy, transvascular mechanical thrombectomy or surgical reoperation with thrombectomy. Beyond the therapeutic window, the non-salvageable infarcted kidney will require nephrectomy.

Ultrasound findings of RVT include diffuse swelling and abnormal echogenicity in the graft parenchyma with an elevated RI, main renal artery decreased peak systolic velocity (PSV), and reversed diastolic flow.

Although acute rejection and acute tubular necrosis can also present with a swollen kidney with elevated RI, a specific sign on ultrasound for RVT is the absence of venous flow on colour and spectral Doppler (Figure 3a). On greyscale ultrasound, a thrombosed renal vein is seen as a tubular structure in the renal hilum.

RI is used as an adjunct to colour Doppler ultrasound in graft kidneys. It is calculated using the following formula: (PSV – end diastolic velocity) / PSV. There is a variation of the upper limit of normal RI in the literature, ranging from 0.7 to 0.8.[10] RI is used as a marker of microcirculation injury and a sequela of interstitial oedema in all forms of graft dysfunction, and is non-specifically elevated in a range of conditions.[1] Elevated

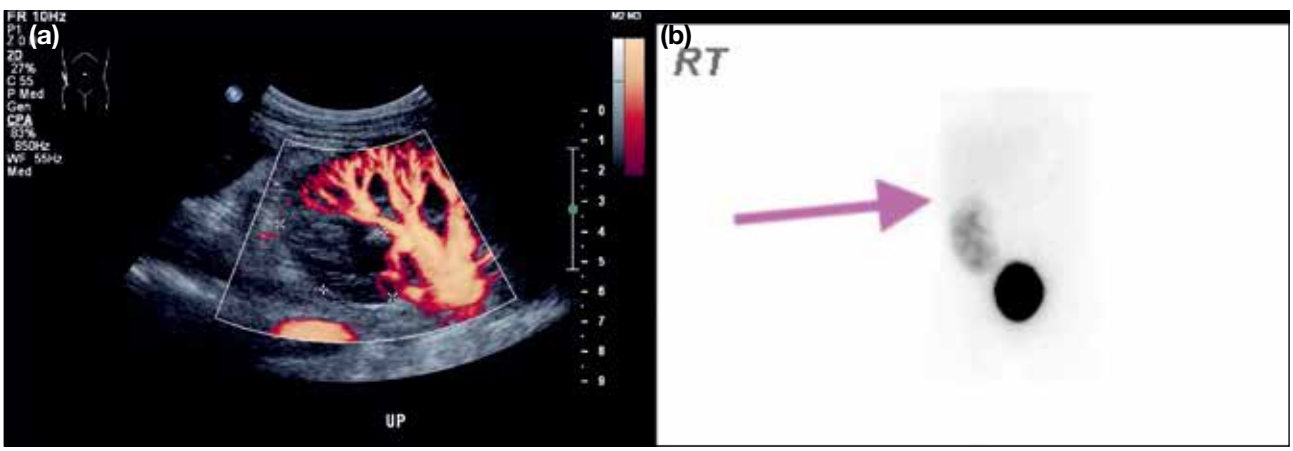

Figure 2. An 8-year-old girl with end-stage renal failure due to severe renal parenchymal disease underwent a cadaveric renal transplant. The graft kidney had double renal arteries. The small upper pole renal artery was too small for anastomosis. (a) Power Doppler ultrasound demonstrated a hypoechoic avascular wedge-shaped area in the upper pole of the graft kidney. (b) Technetium-labelled mercaptoacetyltriglycine study demonstrated a focal wedge-shaped well-demarcated area of decreased perfusion and parenchymal extraction (pink arrow) at the upper pole. Imaging findings were compatible with a focal segmental renal infarct in the upper pole.

RI is correlated with early allograft dysfunction, but does not show a correlation with long-term allograft survival.[11]

RVT on scintigraphy is revealed as decreased perfusion and delayed cortical uptake, with prolonged cortical retention and reduced excretion. A similar pattern can be seen in parenchymal pathologies such as acute rejection or pseudorejection.

If in doubt, CT with contrast (Figure 3b, c) can be employed but further deterioration of renal function is a concern for patients with raised creatinine. MR venography can help confirm this complication but is not as readily available as CT.

RENAL ARTERY STENOSIS

The incidence of transplant-related arterial stenosis (TRAS) in paediatric patients ranges from 5% to 9%.[8] Prolonged cold ischaemia time, occurrence of delayed graft function, and cytomegalovirus infection are recognised risk factors.[12,13] An adjunctive risk factor in young patients may be the diameter of the small vessels and mismatch of anastomosed vessel size due to the disproportional body weight of donor and recipient.[14] Patients usually present clinically with new-onset hypertension or worsening of pre-transplant hypertension. Patients with TRAS often require high dosages of antihypertensives for blood pressure control. A small portion of TRAS can be clinically silent so regular screening with Doppler ultrasound is essential.

Ultrasound is free of radiation and easily available, but CT and MR angiograms can more accurately detect the extent and site of arterial stenosis. Ultrasound will show focal areas of colour aliasing due to increased flow velocity. The ultrasound criteria for diagnosis of TRAS in paediatric patients are based on studies in adults since no paediatric-specific data have been published. A wide range of PSV thresholds have been published for detection of haemodynamically significant (>50%) transplant-related renal artery stenosis (RAS) with a range of sensitivities and specificities. Practically, a threshold of 200 cm/s is often used with a sensitivity 90% to 100% and specificity of 67% to 88%[1] (Figure 4a).

To mitigate variation in PSV due to the difference in systemic blood flow velocities, another Doppler parameter used is the post-stenosis PSV: pre-stenosis PSV ratio, that is, renal-to-iliac artery PSV, normally <1.8 to 2.0.[15] This is again based on adult studies as no relevant paediatric data have been published. The

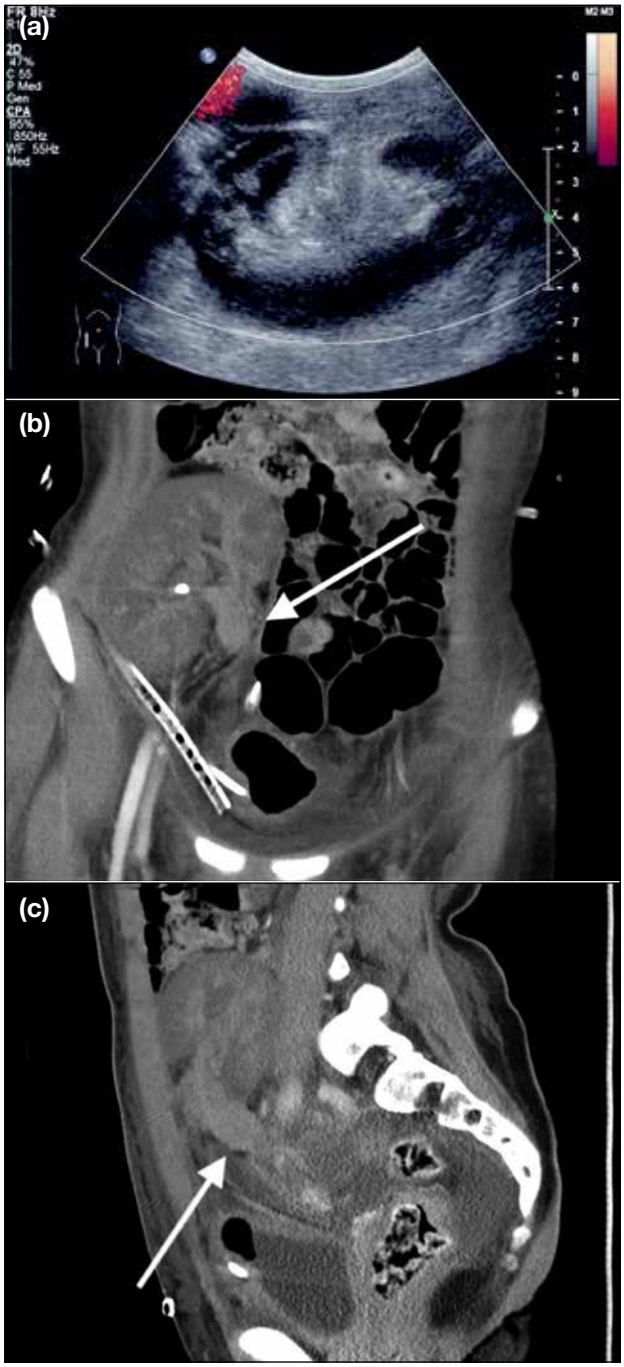

Figure 3. A 15-year-old boy with Noonan syndrome, cystic dysplastic kidney, and end-stage renal failure underwent a cadaveric renal transplant. Creatinine was persistently elevated. Ultrasound showed markedly reduced graft kidney perfusion. Renal vein colour flow was absent. (a) A thrombosed renal vein was seen as a hypoechoic tubular structure in the renal hilum. (b, c) Contrast computed tomography showed a hyperdense non-enhancing, distended renal vein (white arrows) compatible with renal vein thrombosis. The graft kidney showed markedly reduced perfusion. Intraoperative findings confirmed renal vein thrombosis, the graft kidney was severely congested with multiple superficial ruptures. The graft could not be salvaged and graft nephrectomy was performed.

intra-arterial waveform of tardus-parvus pattern with a slow rise in velocity is another sign of RAS.

Catheter-directed angiogram remains the gold standard for evaluation of transplant renal artery stenosis (Figure 4c). MR angiogram involves no radiation but is less readily available than CT angiogram (Figure 4b). Additional sedation may be required for MR compared with CT due to the longer scan time. Renal scintigraphy after angiotensin-converting enzyme inhibition has fallen out of favour for diagnosing RAS owing to its low sensitivity and high radiation exposure.

The mainstay of treatment for TRAS is percutaneous angioplasty. Surgery is reserved for failed percutaneous angioplasty with or without stenting and for anastomotic stenoses.[14]

ARTERIOVENOUS FISTULA

A percutaneous renal biopsy is invaluable for surveillance and for histologically diagnosing the aetiology of graft dysfunction. Arteriovenous fistula (AVF) after biopsy is more common in graft than native kidneys with an incidence of 15% to 16%.[16] AVF after biopsy has an incidence of around 7% in transplanted kidneys, of which 0.3% to 0.4% are symptomatic. Risk factors for AVF development include renal medullary disease, nephrocalcinosis, hypertension, renal insufficiency, increased number of attempts, and depth of the biopsy. 80% to 95% of them resolve spontaneously without treatment in 2 to 31 months.[17]

Small AVFs constitute the majority of cases and most close spontaneously. Larger AVFs are less common and can be symptomatic. Treatment is required for symptomatic cases with haematuria, high-output cardiac failure, vascular steal, and hypertension. Transarterial embolisation is the treatment of choice. It is safe and effective, with minimal loss of renal parenchyma. Long-term graft survival is not affected by embolisation. Late recurrence rate is low after treatment.[18]

The ultrasound appearance of AVFs is colour aliasing in a circumscribed area of the renal parenchyma showing turbulent flow with high-velocity arterial flow with a reduced systolic-diastolic difference and high arterialised venous flow on spectral analysis (Figure 5).

CONCLUSION

Vascular complications can range from those with poor clinical outcomes such as renal artery thrombosis or RVT, to less serious complications such as AVF or segmental infarct. Renal artery thrombosis typically occurs in the period immediately after the transplant.

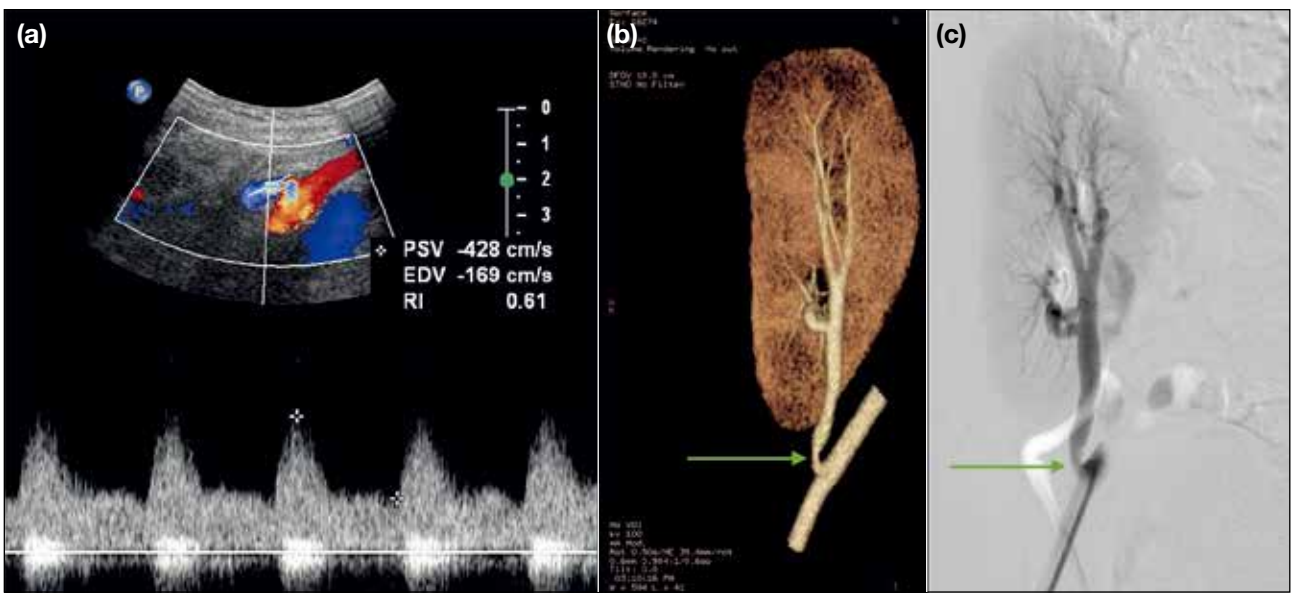

Figure 4. A 13-year-old girl with end-stage renal failure due to Goodpasture syndrome received a cadaveric renal transplant. She developed refractory hypertension 6 months after transplant. (a) Ultrasound scan revealed elevated peak systolic velocity >200 cm/s at the proximal graft renal artery. (b) Computed tomography angiography and (c) digital subtraction angiography scans showed moderate (around 50%) short segment stenosis at the proximal graft artery (green arrows), confirming the presence of graft renal artery stenosis. Angioplasty and stenting were performed.
Abbreviations: EDV = end diastolic velocity; PSV = peak systolic velocity; RI = resistive index.

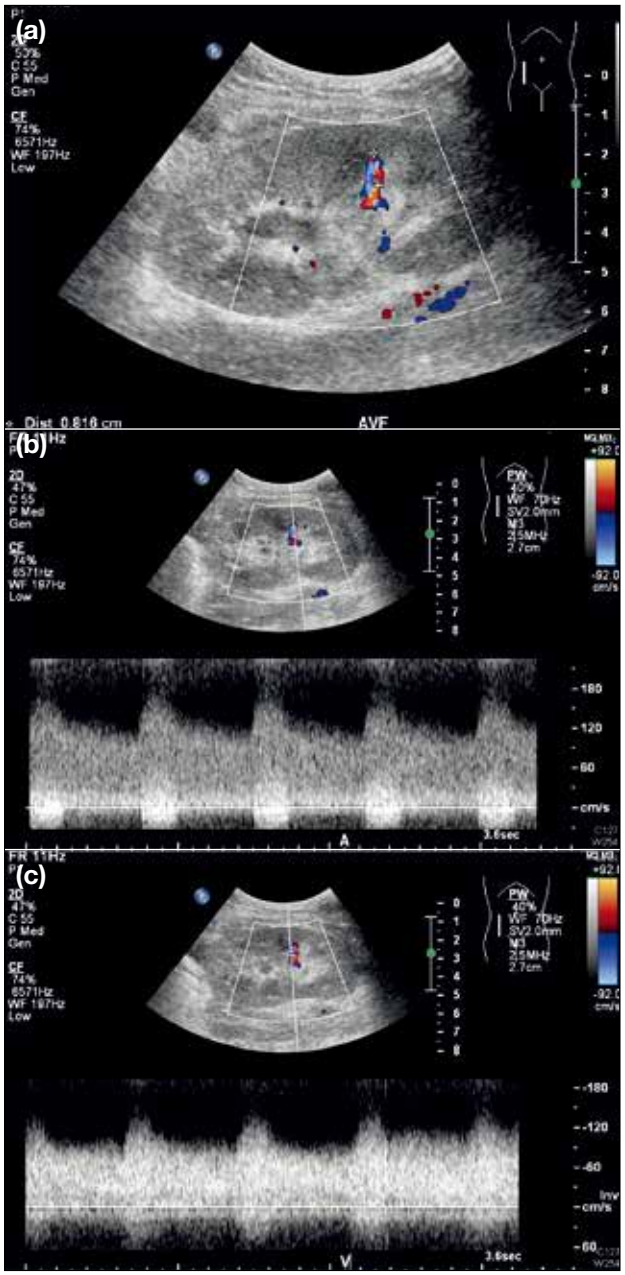

Figure 5. An 18-year-old girl with spina bifida, tethered cord syndrome and neurogenic bladder, bilateral vesicoureteral reflux, and end-stage renal failure. She received a cadaveric renal transplant but remained oliguric with high creatinine. Renal biopsy performed on day 5 after surgery demonstrated acute tubular necrosis. (a) Ultrasound scan on day 7 showed a circumscribed area of colour aliasing in the lower pole. Spectral Doppler showed (b) turbulent flow with high-velocity arterial flow and reduced systolic-diastolic difference and (c) high arterialised venous flow. Findings were compatible with an arteriovenous fistula.

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