

Salvaging the Blue Kidney

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ABSTRACT

One of the most serious complications following kidney transplantation is graft thrombosis. This can be associated with extra morbidity for the patient as well as the need for dialysis. We present a case of the late exploration in a poorly perfused kidney following living donor kidney transplantation, which was saved by explantation, reperfusion on the back table and re-transplantation.

Keywords

Kidney, Transplant, Thrombosis, Explantation, Re-perfusion, Re-transplantation.

Abbreviations

ICU: Intensive care unit; ATI: acute tubular injury; CT: Computerized Tomography; MMF: Mycophenolate Mofetil.

Introduction

Although graft thrombosis remains rare [1], it is one of the most serious complications following kidney transplantation. In one study, where 109 kidney allografts were lost out of 2447 in the first year following transplantation, 23 (34%) were due to venous thrombosis and 6 (9%) due to arterial thrombosis. Other causes included 14 (21%) due to primary non function, 10 (15%) due to rejection 4 (6%) due to recurrent disease [2]. We present a case of the late exploration in a poorly perfused kidney, which was saved by explantation, ex-vivo reperfusion on the back table and re-transplantation.

Case Presentation

A 65-year old gentleman underwent a compatible living unrelated donor (59-year old male) compatible kidney transplant for diabetic nephropathy. At the time of transplant, the patient was on peritoneal dialysis for 8 months with a residual urine output of 250ml/day. Past medical history included myocardial infarction and angioplasty in 2005 with a recent coronary angiography demonstrating diffuse

disease and moderate stenosis. He also had hypertension, bilateral carotid artery disease, a BMI of 26 and was smoking 10 cigarettes per day. He was on Aspirin 150mg, atorvastatin 10mg frusemide 80mg and ramipril 5mg once a day. Iliac Doppler scan demonstrated generalised atheromatous disease with a 75% stenosis of the right common iliac artery with monophasic waveform in the left external iliac artery. CT angiogram the day before surgery did not demonstrate any significant stenosis on the right side; therefore, the implantation of the kidney was planned for the right side.

During transplantation, the renal artery and vein were anastomosed to the right external iliac vessels. A smaller lower polar vein was tied off before transplantation. The cold and warm ischaemia times were 3hr 6min and 53min respectively. At perfusion, the graft looked well filled and pink apart from a part of the lower pole that took longer to perfuse. Ureterovesical anastomosis was uncomplicated using the Lich-Gregoir technique. Intraoperatively, there were episodes of recipient hypotension and these were managed with metaraminol and norepinephrine. The patient was therefore admitted overnight to ICU for continuous inotropic support and monitoring. Induction immunosuppression included basiliximab, and maintenance immunosuppression included tacrolimus, MMF and prednisolone.

Post-operative ultrasound on the same day demonstrated good graft perfusion (Figure 1a). Further ultrasound scan the next day, performed due to a temporary reduction in urine output, and was

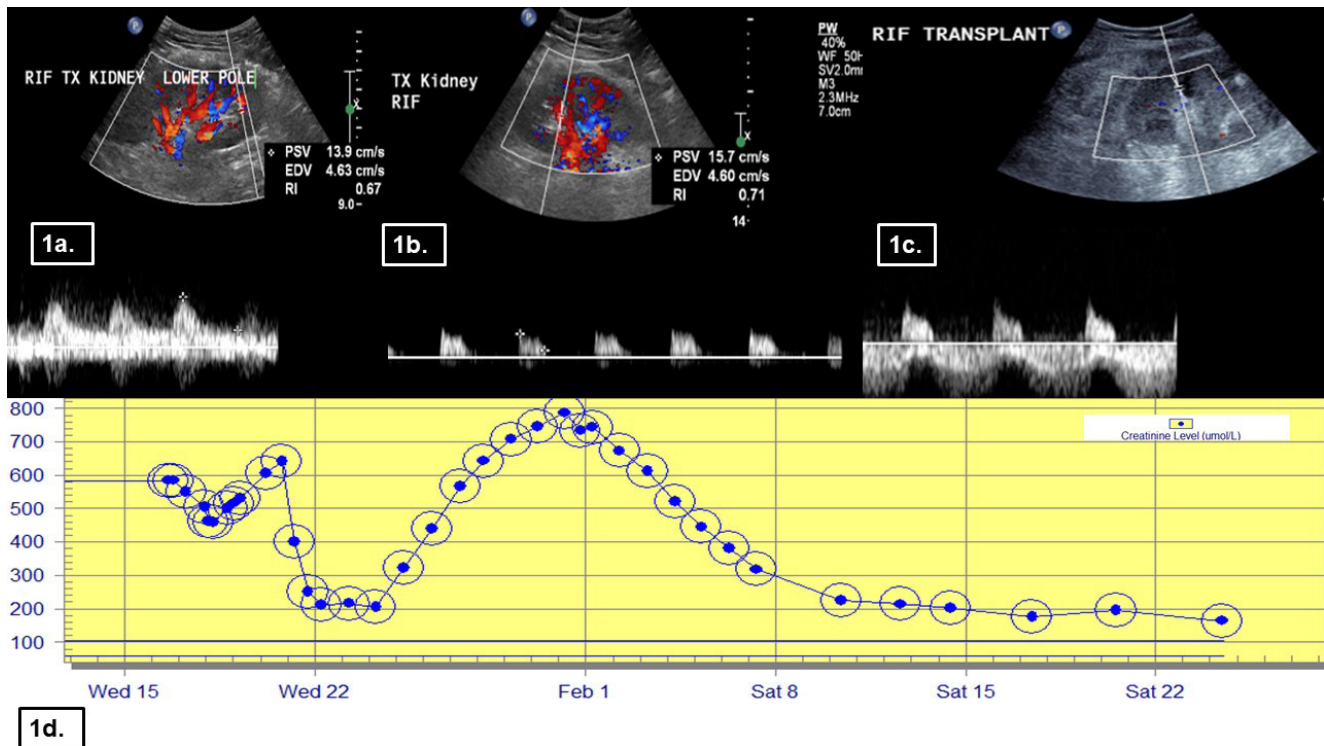
reported to show normal perfusion. However, retrospective review of the images revealed there was absent diastolic flow (Figure 1b). Time zero biopsy showed acute tubular injury (ATI) and Karpinski score [3] was 3.

Despite an increase in urine output to 110 ml/hr by day 3, renal function deteriorated. Serum creatinine rose from 459 $\mu\text{mol/L}$ on day 1, to 642 $\mu\text{mol/L}$ on day 3. Further ultrasound on that day reported reversed diastolic flow within the renal artery and no flow within the renal vein, consistent with renal vein thrombosis (Figure 1c). Therefore, emergency exploration was performed. This revealed a mottled, blue kidney. At the time, the renal artery had a good pulse and no obvious clot in the vein. The decision was taken to explant the renal allograft to assess on the back table. Vascular clamps were applied and the vessels divided. No intraluminal clots were demonstrated. The ureter was also divided and the kidney was placed in ice and perfused with cold soltran (Baxter International Inc) preservation fluid.

On re-transplantation, the kidney did not show great improvement and still looked blue. As the renal artery had a good pulse and the renal vein felt soft, it was thought that the cause of the problem was microvascular thrombosis. Therefore, a biopsy was taken

and heparin infusion was started. Ultrasound on day 4 showed cortical perfusion but poor peripheral perfusion. Kidney biopsy revealed ATI, small foci of early interstitial infarction, interstitial haemorrhage suggestive of focal incipient infarction and cellular rejection. As the patient was being treated for pneumonia at the time, antirejection treatment was not commenced. The patient needed haemofiltration until day 8 post-transplant.

When the patient was clinically better, a further kidney allograft biopsy was performed. This showed cellular and vascular rejection and therefore a 10-day course of Anti-Thymocyte Globulin was started. Over the next few days, the patient's creatinine fell from 642 $\mu\text{mol/L}$ to 164 $\mu\text{mol/L}$ (Figure 1d). The patient recovered well and was discharged home on day 21. His renal function remained stable with a creatinine of 157 $\mu\text{mol/L}$ and eGFR 38.5 mL/min/1.73m² at 12 months post-transplant. A quality of life questionnaire performed nine months post-transplant. This showed that he perceived his health to be excellent at the time, that his kidney problem did not affect his life at all, and that he strongly agreed that he was satisfied with his life. He also felt it was beneficial to have received his kidney transplant that his new kidney met his expectations, that his life changed for the better and that he did not regret having this.



Discussion

Kidney transplantation, especially in high-risk patients with multiple comorbidities, is not always without complications. In particular, correct identification of kidney transplant perfusion abnormalities following transplantation is vital in allowing early assessment and exploration [4]. It is important for the team looking after the patient to have access to scanning when the

clinical picture is not as expected, even out of hours. In addition, it is helpful to have knowledge and understanding of abnormal imaging and to discuss cases with the radiology team when the clinical picture does not fit. Abnormal results can be pursued by doing further imaging, such as a contrast CT, or return to theatre to inspect the kidney graft. Successful salvage surgery is possible, but graft nephrectomy remains common.

Venous complications successfully treated with re-perfusion and re-transplantation have been described previously but these were detected intraoperatively or on early post-operative Doppler [5]. Explanations for these complications included renal vein stenosis or renal vein laceration. Technical factors may result in allograft thrombosis but nothing obvious was identified at the time of graft explantation in our case. Other reasons that can predispose to graft thrombosis include genetic coagulation abnormalities or the presence of antiphospholipid antibodies [6]. There was no prothrombotic history in our patient. Finally, atherosclerosis, haemodynamic instability at the time of transplantation as well as diabetic nephropathy and in some studies peritoneal dialysis have been described as possible risk factors for renal graft thrombosis [6] and our patient had all these.

Another study tried to identify risk factors that could predict graft thrombosis by comparing patients who received deceased donor kidneys that resulted in early thrombosis with patients who received the contralateral kidneys from the same donor [7]. There was an increased risk for vascular thrombosis in kidneys with multiple veins or organ retrieval complications such as vascular lesions or abnormal perfusion. In our case, there was no obvious organ retrieval damage but there was a smaller second vein that was tied.

Conclusion

This paper intends to raise awareness of critical cases where identifying possible risk factors prior to surgery, having a high index of suspicion when things are not progressing as planned, developing knowledge of the correct renal graft doppler waveforms and acquiring a low threshold for re-exploration, play an extremely important part in potentially saving a kidney graft. Explantation,

re-perfusion and re-transplantation, even after three days following transplantation, can then be utilised to salvage a blue kidney. This approach allowed our patient to have a functioning allograft with very good function as well as excellent quality of life.

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