



FROM BLUE TO PINK: SUCCESSFUL SALVAGE OF INTRAOPERATIVE RENAL ALLOGRAFT CYANOSIS DUE TO ACUTE RENAL ARTERY THROMBOSIS IN LIVING DONOR RENAL TRANSPLANTATION

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ABSTRACT

Vascular complications following renal transplantation, though uncommon, are serious and can jeopardize graft survival. Acute intraoperative renal artery thrombosis secondary to intimal injury is a rare but recognized cause of graft ischemia. We report a case of a 48-year-old male with end-stage renal disease who underwent live-related renal transplantation with his wife as the ABO-compatible donor. Following completion of the vascular anastomosis and release of clamps, the graft initially reperfused but progressively developed patchy and then complete cortical cyanosis over ten minutes, with loss of arterial pulsation at the hilum. Re-exploration revealed a thrombus at the arterial anastomosis with an underlying intimal injury of the donor renal artery. The clot was evacuated, the graft re-perfused in situ with cold preservation solution, the injured arterial segment trimmed, and a fresh anastomosis fashioned, restoring pink colour and turgor to the graft within minutes. The kidney functioned well postoperatively, with steady improvement in urine output and serum creatinine, and the patient was discharged on postoperative day 15 with a serum creatinine of 0.8 mg/dL. Prompt intraoperative recognition of graft cyanosis and loss of arterial pulsation, followed by immediate re-exploration and vascular salvage, rescued an otherwise threatened allograft.

KEYWORDS : Graft Salvage, Intimal Injury, Living Donor Renal Transplantation, Renal Artery Thrombosis, Vascular Complications

INTRODUCTION

Renal transplantation remains the definitive treatment for end-stage renal disease, offering superior survival and quality of life compared with maintenance dialysis. Vascular complications after renal transplantation occur in 3–15% of patients and, although uncommon, are among the most serious surgical complications, as improper or delayed management can cause irreversible graft damage or even threaten the patient's life. These complications are broadly classified as intraoperative or postoperative and include transplant renal artery stenosis, transplant renal artery or vein thrombosis, biopsy-induced vascular injuries, pseudoaneurysm formation, and perinephric haematoma.

Intimal tears of the donor renal artery are a recognised, though rare, cause of vascular complications and typically arise from iatrogenic trauma during organ procurement, perfusion, or vascular anastomosis. Such injuries frequently progress to secondary thrombosis of the vessel, jeopardising graft perfusion. Early intraoperative recognition of poor arterial inflow — through changes in graft colour, turgor, and pulsation — is essential, since prompt re-exploration and vascular salvage can rescue the graft and avoid the need for graft nephrectomy. We describe a case in which the transplanted kidney turned progressively cyanotic soon after reperfusion due to acute arterial thrombosis at the site of an intimal injury, and was successfully salvaged by re-exploration, thrombus evacuation, and re-anastomosis, with the graft regaining a healthy pink colour intraoperatively and excellent function thereafter.

Case Report

A 48-year-old male with end-stage renal disease, on maintenance haemodialysis (twice weekly) for one and a half years via a right brachiocephalic arteriovenous fistula, was worked up for live-related renal transplantation with his wife (38 years) as the donor. The recipient was blood group O positive and the donor blood group O negative — an ABO-compatible pair. Viral markers were negative in both recipient

and donor. Preliminary and final cross-match were negative (5–10%). Recipient evaluation, including a micturating cystourethrogram, computed tomography (CT) iliac vessel angiogram, and iliac vessel Doppler study, was unremarkable [Figure 1a].

Donor evaluation showed normal preliminary blood and urine analyses. Split renal function on ^{99m}Tc-DTPA renal scintigraphy was 48% (GFR 49.2 mL/min) on the left and 52% (GFR 53.3 mL/min) on the right, with a total GFR of 102.5 mL/min. CT renal angiography demonstrated bilateral single renal arteries and veins without pre-hilar branching or retro-aortic course; early segmental branching of the right renal artery was noted, and the left kidney (artery length ~28 mm, diameter 7 mm at origin; vein diameter ~19 mm) was therefore selected for donation [Figure 1b]. An open left donor nephrectomy was performed uneventfully, with a warm ischaemia time of 40 minutes and a cold ischaemia time of 45 minutes; both the renal artery and vein were of adequate length, calibre, and sharp cut ends.



Figure 1: (a) Preoperative three-dimensional computed tomography renal angiogram of the donor showing bilateral single renal arteries (RT RA, LT RA) of adequate calibre, the

left being selected for donation. (b) Preoperative computed tomography iliac vessel angiogram of the recipient showing normal calibre bilateral iliac arteries and veins.

On the recipient side, the graft was placed extraperitoneally in the right iliac fossa. The venous anastomosis was performed between the donor renal vein and the recipient's right external iliac vein (end-to-side), followed by the arterial anastomosis between the donor renal artery and the recipient's right internal iliac artery (end-to-end). Following completion of the anastomoses and release of the vascular clamps, the graft reperused and appeared satisfactory initially. However, over the next five minutes patchy cyanotic areas appeared over the cortical surface [Figure 2a], progressing to complete cortical cyanosis by ten minutes [Figure 2b]. Palpation revealed loss of pulsation in the renal artery distal to the anastomosis, near the hilum, raising a strong suspicion of acute renal artery thrombosis.

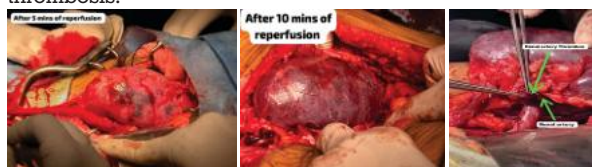


Figure 2: (a) The graft shows patchy cortical cyanosis within five minutes of reperfusion. (b) The graft becomes completely cyanotic by ten minutes, with loss of arterial pulsation at the hilum. (c) Re-exploration shows a thrombus (arrow) occluding the donor renal artery just proximal to the anastomotic site.

Immediate re-exploration was undertaken. The arterial anastomosis was taken down and a thrombus was evacuated from the donor renal artery just proximal to the anastomotic site [Figure 2c]. The excised arterial stump, on inspection, revealed a focal intimal injury at the anastomotic margin [Figure 3a]. The graft was then re-perfused in situ with cold histidine-tryptophan-ketoglutarate (HTK) solution for approximately 20 minutes (total secondary ischaemia time of about 30 minutes), with a venotomy fashioned to allow free egress of the perfusate; the kidney turned uniformly pale, confirming an adequate flush [Figure 3b]. The injured arterial edge was trimmed back to healthy tissue and a fresh end-to-side anastomosis was fashioned to the same recipient internal iliac artery, and the venotomy was closed. This entire salvage procedure was carried out by the urology team, without need for vascular or cardiothoracic surgical assistance. On release of the clamps, the graft reperused, regaining a uniformly pink colour and normal turgor within ten minutes [Figure 3c]. The immediate urine output at the end of the procedure was approximately 25 mL.

Postoperatively, urine output and renal function improved steadily (Table 1). Doppler ultrasonography of the transplant renal artery, performed on postoperative day 2, showed normal resistive indices at the main renal artery (at its origin and at the hilum) as well as at the interlobar, arcuate, and segmental arteries, with an impression of a normal transplant renal artery Doppler study. Ultrasonography also showed a perinephric collection measuring approximately $9.3 \times 7.7 \times 6.8$ cm with minimal free fluid in the pelvis, the tip of the double-J stent lying within the collection; this was managed conservatively. A subsequent contrast-enhanced CT scan, performed later during the admission in view of the perinephric collection, confirmed normal contrast opacification and vascularity of the transplant kidney with no evidence of active contrast extravasation at the anastomotic site, along with a reducing perinephric collection and bilaterally contracted native kidneys. The patient was discharged on postoperative day 15 with a serum creatinine of 0.8 mg/dL and satisfactory graft function.



Figure 3: (a) The excised arterial segment shows a focal intimal injury (arrow) at the anastomotic margin. (b) The graft turns pale following in-situ cold HTK re-perfusion prior to re-anastomosis. (c) The graft regains a healthy pink colour and turgor after re-anastomosis and reperfusion.

Table 1: Postoperative Urine Output and Serum Creatinine Trend

Postoperative Day	Urine Output (mL)	Serum Creatinine (mg/dL)
1	550	6.4
2	550	4.9
3	900	4.1
4	1500	3.8
5	2200	3.7
6	2400	3.2
7	3500	2.1
8	3200	1.9
9	3100	1.8
10	3600	1.7
11	3400	1.4
12	3600	1.3
13	3400	1.1
14	3200	0.9
15	3600	0.8

DISCUSSION

Renal transplantation is the definitive therapy for end-stage renal disease, and outcomes have improved considerably in the era of modern immunosuppression. Even so, posttransplant vascular complications, although not common, remain a severe and serious surgical problem; improper or delayed treatment can cause irreversible damage to graft function and even endanger the recipient's life. Vascular complications may relate to recipient factors (advanced age, diabetes mellitus, atherosclerosis, hypertension, a hypercoagulable state), donor factors (advanced age, marginal donor status, donor renal artery lesions), transplantation-related factors (prolonged cold ischaemia time, immune induction protocol, rejection), or surgery-related factors, such as intimal damage sustained during donor organ procurement, perfusion, or vascular anastomosis, and technical imperfections in the anastomosis itself. Early detection, diagnosis, and treatment of these complications are crucial to an effective outcome.

Intimal tears of the renal artery are a recognised cause of secondary vascular thrombosis and typically result from traumatic mechanisms — crush injury from vascular clamps, mishandling of the vessel, or traumatic perfusion cannulae and infusion devices during organ procurement. In a previously reported case from our own experience, an intimal injury attributable to traumatic cold perfusion of the donor kidney led to poor flow across the anastomosis, which was salvaged using an interposition saphenous venous graft with the assistance of the cardiothoracic surgical team. In the present case, by contrast, the graft reperused satisfactorily at first and only became progressively cyanotic over the following ten minutes, with the intimal injury and resultant thrombus localised at the anastomotic margin itself rather than at a site of perfusion cannula trauma. This distinction is instructive: graft colour and arterial pulsation must continue to be monitored closely for several minutes after clamp release and completion of the anastomosis, since arterial compromise may declare itself only after an initial period of apparently adequate perfusion, rather than being evident immediately.

Once thrombosis was suspected on the basis of progressive cyanosis and loss of hilar arterial pulsation, immediate re-exploration allowed identification of the thrombus and the underlying intimal defect. Because the segment of donor artery requiring excision was short, re-perfusion of the graft in situ with cold preservation solution, trimming of the injured arterial edge, and a fresh anastomosis were sufficient to restore inflow, without recourse to an interposition graft or the involvement of a vascular/cardiothoracic surgical team, as had been necessary in the previously reported case. This highlights that the reconstructive strategy for such salvage should be tailored to the length and quality of the vessel remaining after debridement of the injured segment — a short defect may be bridged by direct re-anastomosis, whereas a longer defect, or one leaving insufficient donor artery length for a tension-free anastomosis, may necessitate an interposition graft and multidisciplinary vascular support.

The excellent recovery of graft function in this case — with progressive improvement in urine output and a fall in serum creatinine to 0.8 mg/dL by discharge — underscores the importance of a low threshold for suspecting vascular compromise intraoperatively and of prompt corrective action. Attempts should be made to minimise the risk of intimal injury through gentle vessel handling, use of atraumatic clamps and perfusion devices, careful inspection of the cut ends of donor vessels before anastomosis, and repeated reassessment of graft colour, turgor, and arterial pulsation before abdominal closure.

CONCLUSION

Vascular complications following renal transplantation are uncommon but, when they occur, are serious threats to graft survival. Acute intraoperative renal artery thrombosis secondary to intimal injury may present not at the moment of reperfusion but with progressive cyanosis and loss of arterial pulsation over the following minutes, and the operating team must remain vigilant for these signs even after an apparently successful initial reperfusion. Prompt re-exploration, thrombus evacuation, and vascular salvage — tailored to the length of donor vessel available after debridement — can rescue an otherwise threatened graft and secure an excellent functional outcome.

Declaration of Patient Consent

The authors certify that patient consent has been obtained for participation in the study and for publication of clinical details and images. The patient and donor understand that their names and initials will not be published, and that all standard protocols will be followed to conceal their identity.

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Conflicts of Interest

There are no conflicts of interest.

REFERENCES

1. Reyna-Sepúlveda F, Ponce-Escobedo A, Guevara-Charles A, Escobedo-Villarreal M, Pérez-Rodríguez E, Muñoz-Maldonado G, et al. Outcomes and surgical complications in kidney transplantation. *Int J Organ Transplant Med* 2017;8:78-84.
2. Di Carlo HN, Darras FS. Urologic considerations and complications in kidney transplant recipients. *Adv Chronic Kidney Dis* 2015;22:306-11.
3. Dimitroulis D, Bokos J, Zavos G, Nikiteas N, Karidis NP, Katsaronis P, et al. Vascular complications in renal transplantation: A single-centre experience in 1367 renal transplantations and review of the literature. *Transplant Proc* 2009;41:1609-14.
4. Mayhew M, Solomon R, LaGuardia H, Shaw K, Arenas J, Hranjec T. Vascular complications in renal transplantation: Surgical salvage of renal artery dissection. *Transplant Direct* 2022;8:e1340.
5. Dobrilovic N, Bennett S, Smith C, Edwards J, Luchette FA. Traumatic renal artery dissection identified with dynamic helical computed tomography. *J Vasc Surg* 2001;34:562-4.
6. Zhang J, Xue W, Tian P, Zheng J, Ding C, Li Y, et al. Diagnostic and therapeutic strategies for vascular complications after renal transplantation: A single-centre experience in 2,304 renal transplantations. *Front Transplant* 2023;2:1150331.
7. Nerli RB, Uplenchwar S, Shetty G, Patil S, Rai S, Patel K, et al. Intraoperative renal artery thrombosis of the transplanted kidney secondary to intimal injury. *Indian J Transplant* 2024;18:204-6.