Successful Repair of Kidney Graft Artery Rupture Using External Stenting


Escherichia coli

Transplantation

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Kidney graft view at days 0 (A) and 10 (B). C, doppler ultrasound.

Two main graft arteries sutured to internal iliac artery (light blue arrow) and abdominal aorta (blue arrow). Graft vein (black arrow) anastomosed to suprarenal inferior vena cava. Green arrow indicates the site of future rupture.

Artery rupture secondary to infection is a rare and usually fatal complication of organ transplantation. In most cases, infection-related damage to the vascular wall is linked to the site of anastomosis and leads to vessel rupture and bleeding. If the patient survives, development of a pseudoneurym may cause symptoms that enable diagnosis. Here, we report the use of an external stent to repair the vessel wall in an 18-month-old girl with catastrophic bleeding from a graft artery that fissured 10 days after kidney transplantation.

CASE REPORT

An 18-month-old girl with congenital nephrotic syndrome received a maternal left kidney transplant on her right side (Fig. 1A) simultaneously with native nephrectomy. Early graft function was excellent. Preoperative examinations of both donor and recipient, including culturing of urine, dialysate, and throat and nasal mucosa, revealed no infections. The patient had been maintained on peritoneal dialysis since the third month of life and had experienced two episodes of dialysis peritonitis at 13 and 15 months of age.

Immunosuppression with 1-mg tacrolimus three times per day (trough level, 8–10 until day 10 and 2–8 thereafter) and 90-mg mycophenolate mofetil per day was initiated on day −1. No steroids were administered except for 125 mg of methylprednisolone before graft reperfusion. Routine antibiotic prophylaxis consisted of one intravenous dose of vancomycin (10 mg/kg) and cefotaxime (1 g) before incision and was followed with cefotaxime (1 g, twice a day) until drain removal (day 1 after transplantation). No complications occurred during the first week after transplantation. A small dehiscence of the midabdominal wound where the dialysis catheter was removed appeared on day 7, and the wound discharge was cultured. Urine was cultured daily and remained clear until day 7.

During a routine dressing procedure on day 10, the child became pale and lost consciousness, and the pulse and heart tones were no longer detectable. A considerable amount of scarlet blood appeared between the sutures on the right-side incision. Cardiopulmonary resuscitation was started, and the child was immediately moved to the operating room (on the same floor, 50 m from the dressing room). The large wound on the right side was opened, and the abdominal aorta was pressed by finger to stem the bleeding. Examination of the graft site revealed a 1-cm fissure in the branch of the renal artery responsible for supplying blood to approximately one-third of the kidney. The branch was clamped, and pressure on the abdominal aorta was relaxed, allowing blood to flow to the lower body and the remaining portion of the graft. An attempt to close the fissure with separate sutures was unsuccessful because the edges of the defect were too soft. The microbiology laboratory reported that cultures of the urine and wound discharge collected 3 days previously were positive. The same strain of Pseudomonas aeruginosa was detected in both specimens. This strain was resistant to all tested antibiotics except fosfomycin and polymyxin E.

A decision was made to wrap the affected artery in a piece of synthetis tissue. A knitted polyester vascular graft was slashed longitudinally, the excess was cut off to tailor the graft to the dimensions of the affected artery, and the remaining part was immersed into a solution of polymyxin for 1 min. The patch was then sewn around the fissured vessel by means of a running polypropylene suture (Fig. 1B). Imipenem/clastatin (250 mg) and vancomycin (90 mg) were administered for 5 days as prophylaxis for wound contamination. Fosfomycin (1 g) was administered through the nasogastric tube as the culture results were received and, subsequently, was given orally four times each 4 days until two consecutive sterile urine cultures were obtained. Immunosuppression was suspended for 3 days, and both drugs were reintroduced on day 13 after transplantation. The dosage of mycophenolate mofetil was increased to 90 mg two times per day on day 59 after transplantation because of improved intestinal tolerance.

The patient’s postoperative course was rather favorable. Acute graft failure required a 20-hr venovenous hemodiafiltration, after which the patient’s urine output increased and her blood creatinine dropped to a baseline level (~30 μM/L) within 5 days. Doppler

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ultrasound clearly indicated a high systolic flow and a low acceleration index within the repaired artery (Fig. 1C-2) and a moderate depression of the resistance index downstream (Fig. 1C-3) of the repair. At 12 weeks after transplantation, the child was free of infection, normotensive with no antihypertensive drugs, and showed excellent graft function.

**DISCUSSION**

The survival of the patient described in this report was the result of a fortunate combination of circumstances. However, the technique used to repair the infection-induced artery fissure deserves attention. Infection-induced vascular damage is typically localized at the site of anastomosis. In our patient, subadventitial blood collecting at the site of future vascular wall melting (Fig. 1A, green arrow) indicates that the branch of the renal artery was ruptured, likely as a result of wound contamination. We decided to save the graft despite the suspected multidrug-resistant *Pseudomonas* infection because we had at our disposal both of the effective antibiotics: fosfomycin for systemic administration and polymyxin E for soaking the knitted tissue.

Of the few reports in the literature involving graft rescue in cases of infection-induced artery damage, most describe surgical treatment of the pseudoaneurysm. Saving the affected recipient’s artery is often difficult to achieve. Taghavi et al. (1) reported good results after an 18-month follow-up in a case of artery repair involving a saphenous vein patch. Albano et al. (2) described two patients with aneurysms of the graft or iliac artery caused by *Candida* arteritis in which the graft was saved after arterial patching. Osman et al. (3) summarized the data for 24 patients with pseudoaneurysms related to kidney graft. The grafts were saved in only 5 of the patients (21%), and 9 of 17 transplantotomized patients lost the iliac or femoral artery, with or without subsequent bypass. Benson (4) was the first to describe wrapping of an aortic aneurysm with synthetic material. A similar technique can be used to successfully repair smaller vessels when bypass is not suitable because of infection-induced wall melting and when internal stenting may favor advance of the infection.

**REFERENCES**


**Effects of Oral Paricalcitol on Secondary Hyperparathyroidism and Proteinuria of Kidney Transplant Patients**

Secondary hyperparathyroidism (SHPT) persists up to 15% to 50% of patients at 1 year of kidney transplantation (1). This persistent SHPT contributes to bone mass loss, a higher risk of fracture, hypercalcemia, hypophosphoremia, and vascular calcifications in transplanted patients. On the contrary, the magnitude of proteinuria is a factor of paramount importance for the rate of progression in many kidney diseases (2, 3). Several studies have clearly indicated that the same correlation can be observed in kidney transplant patients and that the sensitivity of transplanted kidney to the level of proteinuria could be even higher than that of native kidneys (4–7).

Paricalcitol is a selective activator of vitamin D receptor that has demonstrated a significant improvement of SHPT in patients with chronic kidney disease while inducing less hypercalcemia and hyperphosphoremia than other vitamin D analogues (8). Recent experimental and clinical studies have demonstrated a reduction in proteinuria and less structural damage after paricalcitol treatment in diabetic and nondiabetic nephropathies (9, 10). These renoprotective influences have been corroborated by the Vitamin D activation with Paricalcitol for Reduction of Albuminuria in Patients with Type 2 Diabetes Trial study, which demonstrated a significant reduction in albuminuria in type 2 diabetic patients treated with paricalcitol (11).

Information about paricalcitol treatment for SHPT after kidney transplantation is remarkably scarce. The aim of this study was to analyze our experience with paricalcitol in the treatment of transplanted patients with SHPT.

We included all the transplanted patients who had received paricalcitol treatment in the period 2009–2010. Criteria for paricalcitol treatment were the

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