Successful Treatment of Transplant Renal Artery Thrombosis With Systemic Infusion of Recombinant-Tissue-Plasminogen Activator After Renal Transplant

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Abstract
A 24-year-old man with end-stage renal disease secondary to congenital renal hypoplasia underwent a preemptive renal transplant. Although a vascular complication occurred during surgery, the operation was completed satisfactorily. However, postoperative Doppler ultrasound showed no perfusion of the renal artery, vein, and parenchyma, indicating a transplant renal artery thrombosis. A reoperation was promptly performed, with systemic infusion of recombinant-tissue-plasminogen activator during graft nephrectomy, followed by a reimplant that resulted in a salvage allograft. Immediate thrombolysis using systemically infused recombinant-tissue-plasminogen activator may be an effective treatment option for transplant renal artery thrombosis after renal transplant.

Key words: Recombinant-tissue-plasminogen activator, Systemic infusion, Thrombolysis, Transplant renal artery thrombosis

Introduction
Transplant renal artery thrombosis (TRAT) is a rare, but severe, complication, with a prevalence of 0.5% to 3.5% that often results in graft loss after a renal transplant.\(^1\) Intervention for graft artery thrombosis has been successful in a few patients,\(^2-4\) making managing TRAT uncertain. This report describes the first successful treatment of TRAT using systemically infused recombinant-tissue-plasminogen activator (rt-PA) with subsequent reimplant after a renal transplant.

Case Report
A 24-year-old man presented with chronic kidney disease secondary to congenital renal hypoplasia and a serum creatinine concentration of 7.46 mg/dL. The patient was scheduled for a preemptive renal transplant from his father, who was ABO incompatible and had zero HLA mismatches. The patient had no history of thrombotic disease or medication that could potentially induce a thrombus. Immunosuppression including tacrolimus, mycophenolate mofetil, and methylprednisolone, was initiated 1 week before surgery. The right kidney of the donor was selected for transplant, because his left kidney had multiple renal arteries.

A 15-cm Gibson incision was made in the right lower quadrant of the abdomen. The external iliac vein and the internal iliac artery were dissected to prepare for anastomosis. The right kidney allograft measured 9.68 × 4.21 cm and had a single renal artery and a single renal vein. An end-to-side renal vein anastomosis to the external iliac vein was created with 5-0 Prolene (Ethicon, West Somerville, NJ, USA) running sutures, and an end-to-end renal artery anastomosis to the internal iliac artery was created with 6-0 Prolene interrupted sutures.

After release of the vessel clamps, uncontrolled bleeding occurred from linear tears in the donor renal vein. The renal vein was restored by reclamping the external iliac vein and internal iliac...
artery for 40 minutes. Subsequent intraoperative Doppler ultrasound showed slightly weak flow in the renal artery and vein. Systemic heparinization was begun to prevent vessel thrombosis. A ureteroneocystostomy was created, and the skin incision was closed, confirming that there was no bleeding at any surgical site. Postoperative Doppler ultrasound showed no perfusion of the renal artery, vein, and parenchyma, indicating TRAT (Figure 1).

To salvage the donor graft, a graft nephrectomy was promptly performed, consisting of surgical thrombectomy and systemic infusion of rt-PA for thrombolysis. Forty-four minutes after the initiation of thrombolysis, the graft nephrectomy was completed, and infusion of rt-PA was stopped. There was no evidence of visible occlusive thrombus requiring thrombectomy in the renal artery. Cold preservation solution was perfused easily into the allograft, confirming that thrombolysis was successful. The donor kidney was reimplanted without difficulty, and Doppler ultrasound demonstrated restoration of flow throughout the entire allograft (Figure 2). Despite administration of rt-PA, intraoperative bleeding was well controlled with partial use of Surgical Nu-Knit (Ethicon). The total warm and cold ischemic times were 99 and 266 minutes. A kidney biopsy showed acute tubular necrosis, with no evidence of fibrin thrombus or acute rejection.

After the patient was transferred to the intensive care unit, his vital signs remained stable, with urine output greater than 100 mL/hour. Doppler ultrasound of the transplanted kidney showed good blood flow, with a resistive index of approximately 0.65. Hemodialysis and anticoagulant drugs were not required postoperatively. At 6-month follow-up, his serum creatinine concentration had gradually declined and is currently 2.94 mg/dL.

Discussion

Transplant renal artery thrombosis in this patient may have been due to the 40-minute reclamping needed to control bleeding during the initial renal transplant. Other conditions that can lead to blockage of blood flow in the renal artery, including torsion or kinking of the anastomosis and dissection of the arterial wall, were not observed in this patient.

Generally, transplanted renal allografts diagnosed with renal artery thrombosis are lost. Once TRAT is confirmed, surgical thrombectomy with repair of the anastomosis is usually performed; however, many patients require a transplant nephrectomy because of graft loss. Therefore, it is likely that surgical thrombectomy alone is not adequate treatment for TRAT.

Successful treatments of TRAT have included intraarterial catheter-guided fibrinolysis treatment using rt-PA or urokinase for delayed TRAT, pharmacomechanical thrombectomy with catheter-directed thrombolysis and stent placement, and intraoperative direct infusion of rt-PA into the external iliac artery used to create the renal artery anastomosis after an unsuccessful surgical thrombectomy. Catheter-directed thrombolysis could not be performed on our patient on the day of surgery, because immediate thrombolysis that allowed time for transplant nephrectomy was required, rt-PA was administered systemically. Renal flow could not be
evaluated before kidney removal because of intraoperative technical difficulties.

Recombinant-tissue-plasminogen activator is a powerful thrombolytic agent frequently used in patients with myocardial infarction, acute ischemic stroke, and acute pulmonary embolism.\(^6-8\) The rt-PA dosage and route of administration in our patient were the same as those recommended in Japan for patients with acute ischemic stroke: an intravenous bolus of 0.06 mg/kg (10% of a 0.6 mg/kg dose) for 1 minute followed by continuous infusion of 0.54 mg/kg (90% of a 0.6 mg/kg dose) for 60 minutes.\(^9\)

Major surgery within the previous 14 days is a relative contraindication to using rt-PA because of the increased risk of bleeding.\(^10\) However, uncontrolled bleeding was not seen intraoperatively in this patient after the initiation of rt-PA. This may have been because we used an optimal hemostatic technique during surgery or to the short half-life of rt-PA (ie, 6 minutes) indicating that the effects of this drug immediately disappear after its withdrawal. These findings indicate that systemically infused rt-PA is safe in selected situations, even during surgery.

In conclusion, renal artery thrombosis after renal transplant can be successfully treated by immediate thrombolysis using systemically infused rt-PA, depending on the patient’s condition and comorbidities. To our knowledge, this is the first case of successful allograft salvage using systemically infused rt-PA followed by reimplant after renal transplant.

References


