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Kidney Failure in a Transplant From an Identical Twin

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DISCUSSION

What is the differential diagnosis for transplant injury following identical twin transplantation?

Solid-organ transplantation between identical twins provides a unique circumstance in which posttransplantation immunosuppression can be withdrawn with little risk of organ rejection because the donor and recipient organ antigens are identical. Therefore, loss of transplant function in identical twin transplantation results from either recurrence of the primary disorder or de novo kidney pathology. This patient had benign urinary sediment and a kidney transplant ultrasound demonstrating chronic kidney injury without significant evidence for obstruction, making an underlying glomerulopathy or obstructive nephropathy unlikely. Recurrence of diabetes within the kidney transplant occurs, but is unlikely to result in the rapid kidney failure that this patient experienced.

What does the kidney biopsy demonstrate?

The kidney biopsy specimen shows chronic interstitial fibrosis, which, combined with the cortical thinning on ultrasound, carries a poor prognosis for kidney recovery. Multiple large calcium crystals can be seen within the tubules that intensify with polarized light (Fig 2), which are highly suggestive of oxalate nephropathy.

What is the most likely cause of the patient's kidney injury, and what tests would you perform to confirm the diagnosis?

Given the patient's history of Roux-en-Y gastric bypass and the evidence of oxalate nephropathy from the biopsy, enteric hyperoxalosis was suspected. A diagnosis of enteric oxalosis requires showing enteric fat malabsorption, hyperoxaluria, and enhanced urinary oxalate excretion following an oxalate load.¹ Serum oxalate levels are of limited value in patients with advanced kidney injury because they are elevated from reduced kidney

clearance. A 72-hour fecal fat collection and 24-hour urine collection confirmed the presence of fat malabsorption and hyperoxaluria (Table 2). Given the patient's advanced kidney failure and risk of exacerbating oxalate deposition, we did not pursue an oxalate load test for confirmation.

What treatment options are available?

Failure to treat early oxalate nephropathy results in irreversible kidney injury and progression to end-stage renal disease. Hemodialysis, especially intensive daily therapy with high-flux membranes, has been shown to effectively reduce serum oxalate levels.² Although hemodialysis is effective at removing oxalate, maintenance hemodialysis for the treatment of enteric oxalosis is not desirable. Long-term therapy involves a combination of measures aimed at reducing enteric oxalate levels and calcium oxalate precipitation within the renal tubules. A combination of low-oxalate diet, oral calcium, and probiotics are used to lower enteric oxalate levels.³ Ensuring adequate hydration with high urine flow is the primary method to reduce urinary oxalate concentrations; however, this treatment may be of limited value in patients with limited residual kidney function. Ultimately, reversing the underlying defect should be undertaken if possible. In this case, surgical reversal of a Roux-en-Y was not feasible.

FINAL DIAGNOSIS

Secondary oxalate transplant nephropathy from enteric hyperoxalosis following Roux-en-Y gastric bypass.

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CLINICAL PRESENTATION

A 61-year-old white man with end-stage renal disease secondary to diabetic nephropathy presented with a 2-month history of progressive fatigue and lower-extremity edema. The patient initially was evaluated for kidney transplantation 4 years earlier, but deferred due to body mass index of 42 kg/m². To achieve the required weight loss, he underwent Roux-en-Y gastric bypass, which resulted in a loss of 120 lb (corresponding to body mass index of 26.5 kg/m²) and cured his diabetes. He then received a living related kidney transplant from his identical twin brother, confirmed by genetic testing, 2 years before the current presentation. His posttransplantation course was remarkable for an episode of methicillin-sensitive *Staphylococcus aureus* discitis and *Clostridium difficile* colitis 3 months posttransplantation. He achieved a nadir serum creatinine level of 1.4 mg/dL (estimated glomerular filtration rate, 55 mL/min/1.73 m² by the 4-variable MDRD (Modification of Diet in Renal Disease) Study equation), but was lost to follow-up 6 months prior to presentation.

On presentation, the patient's medications included oral calcium, amlodipine, multivitamin, and gabapentin (Neurontin). Laboratory testing indicated significant loss of transplant function (Table 1). An ultrasound of the transplant kidney showed marked cortical thinning and mild hydronephrosis. A biopsy of the transplant was performed (Fig 1). Three days later, the patient developed pulmonary edema and dialysis therapy was initiated.

Figure 1. Kidney transplant biopsy. Light microscopy. There is marked interstitial fibrosis and tubular atrophy. Tubular lumen contains calcium oxalate crystals. Hematoxylin and eosin stain; original magnification, $\times 40$.

Figure 2.
Light microscopy under polarized light highlights calcium oxalate nephropathy.
Hematoxylin and eosin stain; original magnification, $\times 40$.

Table 1**Laboratory Data**

	6 mo Prior	Presentation
WBC count ($\times 10^3/\mu\text{L}$)	6	8.3
Hb (g/dL)	12.8	7.8
Hct (%)	37.3	22.8
Platelet count ($\times 10^3/\mu\text{L}$)	152	119
Sodium (mEq/L)	139	144
Potassium (mEq/L)	5.2	4.2
Chloride (mEq/L)	107	113
Carbon dioxide (mEq/L)	26	15
SUN (mEq/L)	23	81
SCr (mg/dL)	1.4	6.6
eGFR ^a (mL/min/1.73 m ²)	55	8
Glucose (mg/dL)	122	75
Calcium (mg/dL)	9.2	6.8
Phosphorus (mg/dL)	3.3	8.9
PTH (pg/mL)	195	848
HbA _{1c} (%)	5.6	5.6
Urine pH	5	5
Ketone in urine	ND	ND
Blood in urine	ND	ND
Protein in urine	ND	ND
Glucose in urine	ND	Trace

Abbreviations: eGFR, estimated glomerular filtration rate; Hb, hemoglobin; Hct, hematocrit; ND, not detectable; PTH, parathyroid hormone; SCr, serum creatinine; SUN, serum urea nitrogen; WBC, white blood cells.

^aCalculated using the 4-variable MDRD (Modification of Diet in Renal Disease) Study equation.

Table 2

Hyperoxaluria Workup

	Value	Reference Range
72-h fecal fat (g/d)	22	2–7
Serum oxalate level (μmol/L)	25.7	<1.8
Urine oxalate level (mmol)	1.7	0.11–0.47