

Torsion of Extraperitoneally Transplanted Kidney: An Unusual Complication

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Abstract

Torsion of the extraperitoneally transplanted kidney is rare complication with no clinical data in the literature. The authors present the case of a 44-year-old man with end-stage renal disease who received a kidney transplant from his father. On postoperative day 4, serum urea and creatinine levels increased and urine output decreased. Renal ultrasonography revealed the renal hilum to be rotated to the lateral pelvic border, causing mild pelvocaliectasis, and Doppler ultrasonography, the patients showed a poststenotic flow pattern. After the patient underwent urgent reoperation, all laboratory values and ultrasonography findings returned to normal. To the authors' knowledge, this is the first published case report of torsion of the extraperitoneally transplanted kidney. When posttransplant deterioration in renal function occurs, renal torsion should be considered in the differential diagnosis.

Key words: *Torsion, Kidney transplant, Vascular complication, Rejection*

Introduction

Incidences and causes of complications after kidney transplant are well documented. Common complications include renal artery stenosis, thrombosis, and aneurysm; renal vein thrombosis;

and anastomotic hemorrhage. Torsion of the transplanted kidney is a rare complication.¹⁻³ Such torsion occurs as the kidney rotates around the vascular hilum, and as the result of impaired circulation, infarction develops. With rapid diagnosis, graft detorsion can save the transplanted kidney. In the literature, graft torsion is reported in children with Prune belly (ie, Eagle-Barrett) syndrome^{4,5} and in adults who received intra-peritoneal renal transplants, especially double transplants of the pancreas and kidney.^{1,2,6,7}

There are no data about torsion of the extraperitoneally transplanted kidney in the literature. We present a case of renal graft torsion confirmed by laboratory and radiologic findings after kidney transplant.

Case Report

A 44-year-old man was admitted to the hospital with end-stage renal disease in July 2009. His medical history indicates that renal biopsy was performed because of hematuria and proteinuria 20 years ago, revealing mesangioproliferative glomerulonephritis. After medical treatment, the patient completely recovered. At the time of this writing, 7 years earlier, laboratory tests of his renal function revealed normal results except for proteinuria (24-hour urine protein excretion of 1 g/d). Medical treatment was then performed successfully.

Four months earlier, after use of 3 antibiotics subsequent to upper respiratory tract infection, the patient's renal failure progressed to the decompensated phase. Hemodialysis was initiated in May 2009 and repeated 3 times every week since then. After 3 months of dialysis, the patient became a candidate for live-donor transplant. In August 2009, the patient's father volunteered to donate his kidney. The father was 63 years old, with an

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unremarkable physical examination and no illness and surgery in his medical history. The patient's blood group was A Rh+ and his father's blood group was O Rh+. Human leukocyte antigen (HLA) tissue types for the patient were HLA-A 29/33, HLA-B 35/51, and HLA-DRB1 04/11, and for the father, HLA-A 02/33, HLA-B 51/51, and HLA-DRB1 04/16.

During surgery, a nephrectomy was performed without complications. There was no anatomic abnormality in the transplanted tissue, which had a single renal vein and single renal artery. The renal vein and artery of the transplanted kidney were anastomosed to the external iliac vein and artery using the end-to-side technique. The modified Lich-Gregoir technique was used for ureteroneocystostomy. There was no intraoperative complication. For immunosuppression, basiliximab (monoclonal antibody [IgG1k]) was given to the patient just before induction of anesthesia. Intravenous steroids were given immediately before the arterial clamp was removed. Postoperatively, tacrolimus, mycophenolate mofetil, and methylprednisolone were given daily. On postoperative day 4, basiliximab was administered.

Laboratory results on postoperative day 1 showed a serum urea level of 28.2 mmol/L, a serum creatinine level of 437.6 $\mu\text{mol/L}$, a potassium level of 5.2 mmol/L, an arterial pH of 7.4, and an HCO_3 level of 19.8 mmol/L. Twenty-four-hour urine output on postoperative day 1 was 2100 cc. On postoperative day 2, the patient's laboratory analysis showed a urea level of 32.1 mmol/L, a creatinine level of 361.2 $\mu\text{mol/L}$, a potassium level of 4.6 mmol/L, and 24-hour urine output was 2065 cc. After intravenous administration of 5 mCi of Tc-99m MAG3 (mercaptoacetyl triglycine), renography images (1 min perfusion for 30 min) and static radiograph images were taken. Renal size, perfusion, and function were found to be normal, but residual cortical activity was slightly elevated.

On postoperative day 3, the patient's laboratory analysis showed a urea level of 38.6 mmol/L, a creatinine level of 344.8 $\mu\text{mol/L}$, and a potassium level of 4.3 mmol/L, and 24-hour urine output was 2285 cc. On postoperative day 4, laboratory analysis revealed a urea level of 51.4 mmol/L, a creatinine level of 407.5 $\mu\text{mol/L}$, a potassium level of 4.8 mmol/L, and a tacrolimus level of 18.7 ng/mL. The patient's day-four 24-hour urine output was 1990 cc with intravenous injection of 3 doses of

furosemide 20 mg/2 mL. On postoperative day 5, the patient's laboratory analysis showed a urea level of 64.9 mmol/L, a creatinine level of 488.8 $\mu\text{mol/L}$, a potassium level of 4.3 mmol/L, and a tacrolimus level of 5.1 ng/mL. The day-five 24-hour urine output was 1680 cc with intravenous injection of 5 doses of furosemide 20 mg/2 mL.

Because of low tacrolimus levels, biopsy was planned to determine the cause of increased urea and creatinine (Figure 1). Before biopsy, renal ultrasonography showed the renal hilum rotated to the lateral pelvic border and mild pelvocaliectasis. Doppler ultrasonography indicated that perfusion of parenchyma was extremely reduced. Renal artery flow at the level of anastomosis was slightly increased, but flow at the middle and proximal levels were markedly increased (300 cm/s, 350 cm/s). The distal level of the renal artery had prolonged acceleration of flow, including a poststenotic waveform pattern. The flow rate from branches of the renal artery to the arcuate artery was markedly decreased. At the level of the arcuate artery, the flow rate was characterized by low resistance, acceleration was decreased, and acceleration time was prolonged (pulsatility index, 0.61; resistance index, 0.48; acceleration, 57.34 cm/s²; acceleration time, 95 ms), with a poststenotic waveform pattern. The renal vein flow rate was normal (Figure 2).

It was determined that the poststenotic flow pattern may have been caused by malposition of the graft kidney, and repeat surgery was scheduled. Perioperative evaluation revealed that the transplanted kidney was rotated to lateral on its vertical axis (Figure 3). The kidney was replanted to its normal axis, and 2 biopsies were taken from its parenchyma using a Tru-Cut needle (Cardinal Health; Dublin, OH, USA). The operation was finished by suturing the kidney from its poles.

Figure 1. Plasma Levels of Urea and Creatinine in the Patient, From Before Surgery to Postoperative Day 5

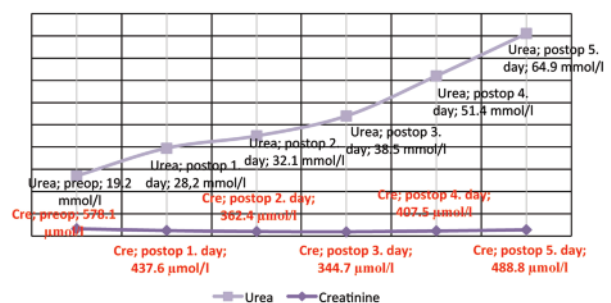
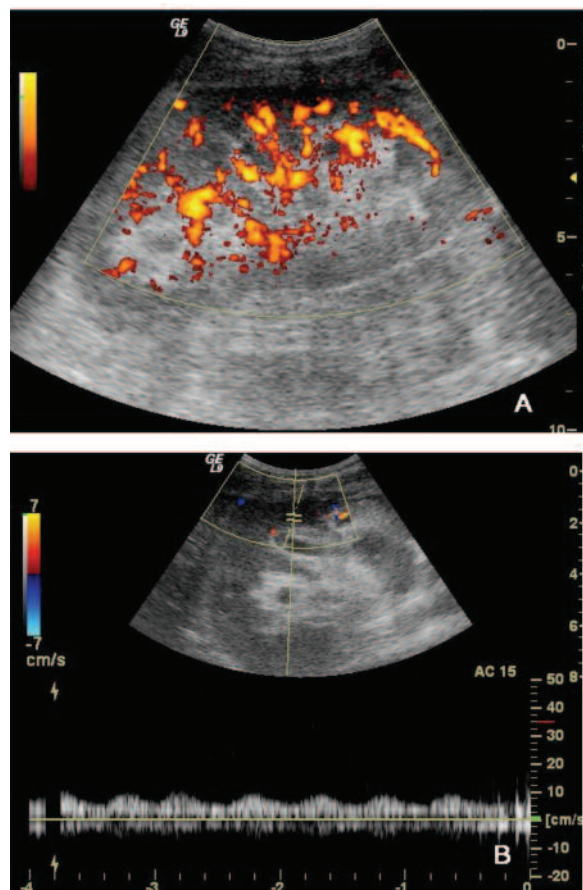
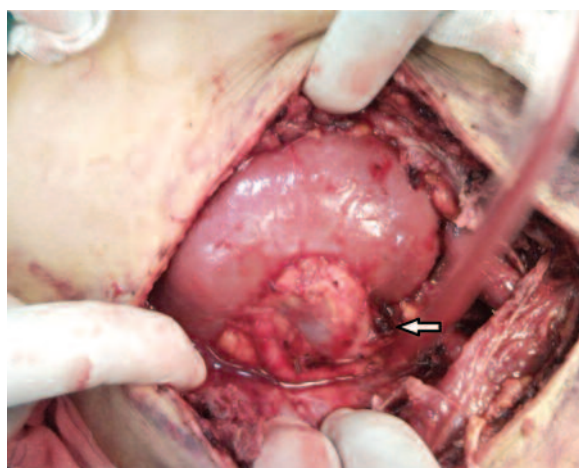


Figure 2. Doppler Ultrasonographic Images of Renal Graft on Postoperative Day 5



(A) Poor tissue perfusion (B) poststenotic waveform pattern

Figure 3. Transplanted Kidney Rotated to Lateral on Its Vertical Axis



After the second operation, all laboratory values for the patient rapidly returned to normal levels. Doppler ultrasonography on day 2 after the second operation showed the renal hilum faced to medial. In addition, renal parenchyma perfusion was normal,

and correction of the renal artery stream Doppler index was observed (peak systolic velocity, 29.37 cm/sec; end-diastolic velocity, 10.44 cm/sec; renal artery stream velocity, 100/34 cm/sec; resistance index, 0.64; acceleration time, 0.17 msec).

Discussion

The reported incidence of vascular complications of renal allografts varies between 0.5% and 3.5%.⁸⁻¹⁰ Renal artery or renal vein thrombosis might lead to catastrophic results for grafts; more than 90% of patients with these conditions lose their grafts.^{9,10} Causes of renal artery or renal vein thrombosis include technical failure, use of older donors, prolonged total ischemic time, problematic coagulation status of the recipient, treatment with cyclosporine, anatomic variants of the donor kidney.¹ Torsion of the transplanted kidney is a rare complication, but rapid diagnosis followed by graft detorsion can save the transplant.

In 1990, Abbitt and associates⁵ reported a case of acute torsion of a renal transplant in a patient, aged 2.5 years, who had Prune belly syndrome. That patient had received a living-donor renal transplant that was placed intraperitoneally. In 1995, Marvin and associates⁴ reported renal allograft torsion in a child diagnosed as having Prune belly syndrome and end-stage renal disease who had received intraperitoneal implantation of an adult deceased-donor renal graft. Because of the small size of the extraperitoneal space in children, an adult renal graft is more favorable for intraperitoneal placement, and the graft that not fixed is an unforeseeable complication. However, displacement of the graft related to Prune belly syndrome must be kept in mind. According to Marvin and associates,⁴ Prune belly syndrome is characterized by the triad of weakness of abdominal wall musculature, cryptorchidism, and congenital urinary tract abnormalities. The lack of normal tonus and abnormal elasticity of abdominal wall muscles may cause excessive mobilization of the renal graft, leading to subsequent torsion of the vascular pedicle or compromise of the ureter.⁴ In the case of our patient, his history was not meaningful for any muscle or collagen tissue diseases associated with torsion.

In 1998, Wong-You-Cheong and associates¹¹ reported adult patients diagnosed as having renal

graft torsion. In a review of 981 patients who received renal grafts, torsion occurred in 5 of the transplants (0.5%) in their series. It must be emphasized that all 5 transplants were placed intraperitoneally. The authors believed that the intraperitoneal location, as well as a long transplanted segment from insufficient trimming of the donor ureter, likely allowed more transplant mobility and ultimate torsion. They further noted that coexisting ascites may have exacerbated organ mobility.

Roza and associates² described renal graft torsion in 2 patients who had received combined intraperitoneal kidney and pancreas transplants. In combined kidney-pancreas transplants, the renal allograft is often placed intraperitoneally. According to the authors, the mechanism of this complication was the intra-abdominal placement of the kidney, length of the vascular pedicle, excess ureteral length, and paucity of adhesions secondary to steroid administration.

Several authors have reported cases in which renal graft dysfunctions were caused by torsion of the vascular pedicle of intra-abdominally placed renal grafts.^{1,3,6,7,12} West and associates¹ emphasized that the discrepancy (≥ 2 cm) between lengths of the renal artery and vein may lead to "kinking" of the renal pedicle.¹³⁻¹⁵ This kinking of the renal pedicle, associated with increased mobility of the kidney secondary to intra-abdominal placement, might be the inciting reason for the torsion. Additional study is needed to determine whether such a discrepancy in length—or the total length of the vascular pedicle—may predispose recipients to torsion.¹¹ In the case of our patient, for whom the left kidney was used from the donor, the length of the left renal vein was 6 cm and that of the renal artery was 3 cm.

One of the diagnostic difficulties associated with renal torsion is its lack of specific clinical features. A high level of suspicion is needed to diagnose renal transplant torsion, because it may mimic rejection and venous thrombosis. Percutaneous biopsy can easily be undertaken to obtain greater specificity regarding possible immunologic or vascular causes of rejection. However, the duration of pathologic investigation is likely too long to wait for effective treatment. Therefore, imaging studies play a crucial role in renal torsion diagnosis. Renal allograft ultrasonography, with color flow Doppler imaging, can detect many vascular complications.^{16,17} The overall sensitivity of this method when evaluating

the renal pedicle is 95%, with specificity of 92%.^{16,18} For the patient in our case, ultrasonography with color flow Doppler imaging played a major role in suspicion of torsion. No additional imaging method was needed.

To the best of our knowledge, the present case report is the first in the literature to describe torsion of the extraperitoneally transplanted kidney. Our patient's history was not meaningful for any muscle or collagen tissue disease that could cause torsion. Rather, we conclude that torsion could be caused by leaving the graft vessels long, an excess discrepancy in length between the renal artery and vein, and the small size of the graft compared to the large size of the area prepared extraperitoneally at the iliac fossa. When deterioration of previously improved renal function is established after transplant, renal torsion must be considered in the differential diagnosis.

References

1. West MS, Stevens RB, Metrakos P, et al. Renal pedicle torsion after simultaneous kidney-pancreas transplantation. *J Am Coll Surg*. 1998;187(1):80-87.
2. Roza AM, Johnson CP, Adams M. Acute torsion of the renal transplant after combined kidney-pancreas transplant. *Transplantation*. 1999;67(3):486-488.
3. Nangia S, Saad ER. Torsion of renal transplant 10 years after simultaneous kidney-pancreas transplantation: imaging as a diagnostic tool. *Transplantation*. 2009;87(10):1590.
4. Marvin RG, Halff GA, Elshihabi I. Renal allograft torsion associated with prune-belly syndrome. *Pediatr Nephrol*. 1995;9(1):81-82.
5. Abbitt PL, Chevalier RL, Rodgers BM, Howards SS. Acute torsion of a renal transplant: cause of organ loss. *Pediatr Nephrol*. 1990;4(2):174-175.
6. Badet L, Petruzzo P, Lefrancois N, Colombel M, Fassi-Fehri H, Martin X. Torsion of the renal pedicle after kidney luxation after simultaneous double kidney and pancreas transplantation [in French]. *Prog Urol*. 2003;13(4):675-678.
7. Knight RJ, Matsumoto C. Renal pedicle torsion as a cause of acute allograft failure after kidney-pancreas transplantation. *Acta Chir Austriaca*. 2001;33(suppl 174):1-9.
8. Palleschi J, Novick AC, Braun WE, Magnusson MO. Vascular complications of renal transplantation. *Urology*. 1980;16(1):61-67.
9. Hashimoto Y, Nagano S, Ohsima S, et al. Surgical complications in kidney transplantation: experience from 1200 transplants performed over 20 years at six hospitals in central Japan. *Transplant Proc*. 1996;28(3):1465-1467.
10. Rijkse JF, Koolen MI, Walaszewski JE, Terpstra JL, Vink M. Vascular complications in 400 consecutive renal allotransplants. *J Cardiovasc Surg (Torino)*. 1982;23(2):91-98.
11. Wong-You-Cheong JJ, Grumbach K, Krebs TL, et al. Torsion of intraperitoneal renal transplants: imaging appearances. *AJR Am J Roentgenol*. 1998;171(5):1355-1359.
12. Rodrigues P, Hering F, Gil A. A well-documented case of chronic renal failure due to misplacement of the transplanted kidney. *Clinics (Sao Paulo)*. 2008;63(1):147-148.
13. Smith RB, Ehrlich RM. The surgical complications of renal transplantation. *Urol Clin North Am*. 1976;3(3):621-646.
14. van Roye SF, van der Vliet JA, Hoitsma AJ, Reinaerts HH, Buskens FG. Causes of early vascular complications in renal transplantation. *Transplant Proc*. 1993;25(4):2609.

15. Benedetti E, Fryer J, Matas AJ, et al. Kidney transplant outcome with and without right renal vein extension. *Clin Transplant*. 1994;8(4):416-417.
16. Grenier N, Douws C, Morel D, et al. Detection of vascular complications in renal allografts with color Doppler flow imaging. *Radiology*. 1991;178(1):217-223.
17. Plainfosse MC, Calonge VM, Beyloun-Mainardi C, Glotz D, Duboust A. Vascular complications in the adult kidney transplant recipient. *J Clin Ultrasound*. 1992;20(8):517-527.
18. Tublin ME, Dodd GD 3rd. Sonography of renal transplantation. *Radiol Clin North Am*. 1995;33(3):447-458.