

Long-term Graft Survival After Kidney Allograft Torsion: Rapid Diagnosis and Surgical Management Key to Reversibility of Injury

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ABSTRACT

Introduction. Kidney allograft torsion (KAT) is a rare complication of kidney transplantation (KT) that occurs when the transplanted kidney rotates around its vascular pedicle, which may result in a catastrophic compromise of the graft's blood supply, deterioration of kidney function, and eventually premature graft death.

Case Report. We report the case of a patient who had an acute kidney injury (AKI) episode from KAT. Her diagnosis was ascertained expeditiously and she had prompt surgical management. Five years after the KAT event, her baseline creatinine (Cr) stabilized around 1.6 mg/dL and she has achieved >8-year graft survival.

Discussion. This case illustrates the reversibility of injury that can occur after a KAT event with a commensurate return to baseline kidney function when KAT is promptly diagnosed and treated. A high index of suspicion of this uncommon but catastrophic complication of KT must be maintained to achieve desirable long-term outcomes. A diagnosis of KAT must be considered when routine etiologies of an acute deterioration of kidney allograft function have been excluded. Finally, prophylactic nephropexy must be strongly considered with intraperitoneal placement of a kidney allograft to avoid KAT.

KIDNEY allograft torsion (KAT) is a rare complication of kidney transplantation (KT) that occurs when the transplanted kidney rotates around its vascular pedicle [1,2]. Although a rare complication, it may result in a catastrophic compromise of the graft's blood supply, deterioration of kidney function, and eventually premature graft death. Acute KAT was first reported in a patient with prune-belly syndrome in 1990 [3]. Although commonly seen in intraperitoneally placed allografts [1,4–6], which have more mobility compared with extraperitoneal KT, the incidence of KAT is unknown because it often goes unrecognized [4].

The diagnosis of KAT is difficult to establish without a high level of suspicion [1,7]. Its clinical presentation can include nonspecific symptoms like fever, abdominal pain, decreased renal function, oliguria, diarrhea, vomiting, and edema, all of which are more often associated with KT dysfunction, such as rejection or ureteric obstruction. This makes diagnosis difficult and intervention delayed. Urgent doppler ultrasound (US) is the study of choice to demonstrate a decrease, reversal, or absence of flow in graft

vasculature [1]. However, decreased or reversed diastolic flow is nonspecific and may also be seen in acute rejection, acute tubular necrosis, or renal vein thrombosis [1]. KAT is difficult to detect on US because the position and axis of the renal allograft in the iliac fossa vary from patient to patient, with an inconstant relationship to its vascular pedicle [8]. Serial USs may show variations in the position and axis of the graft when compared with baseline, supporting a diagnosis of KAT [4], but this approach is highly user-dependent. If KAT is considered in the differential, it is recommended that an emergency exploratory laparotomy be performed to assess the graft and its vascular pedicle [1]. Once a diagnosis of KAT is ascertained, a nephropexy to the anterior abdominal wall is recommended to prevent further torsion [4].

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Fig 1. Coronal sections of kidney allograft torsion CT scan on admission (**A**) and 7 days later (**B**) demonstrating the rotation of the transplanted kidney around its vascular pedicle. *Indicates the upper pole of the kidney allograft.



The long-term outcomes after an acute KAT event are unknown. We found no case reports in the literature that report long-term (>1-year) graft survival after KAT. Herein, we report >5-year survival on a patient who had an acute kidney injury (AKI) episode from KAT with a post-KAT baseline creatinine (Cr) level of 1.6 mg/dL.

CASE REPORT

A 49-year-old woman with end-stage renal disease (ESRD) secondary to diabetes mellitus (DM) type 1 received a kidney transplant in 1983 and suffered its subsequent loss due to chronic rejection in 2008. In 2008, she underwent a simultaneous living donor kidney transplantation (LDKT) and deceased donor pancreas transplant (DDPT) with excellent baseline kidney allograft function (Cr, 0.7–1.0 mg/dL). In late 2011, she presented with a prominent lump in her abdomen over her renal allograft incision associated with nausea and one episode of vomiting. On further questioning, she disclosed that her abdominal lump flopped intermittently. Her examination was remarkable for a blood pressure of 130/85 and a heart rate of 66. The abdomen was soft with mild tenderness over the allograft. Lower extremities were without edema. She was noted to have an acute increase in Cr level to 5.91 mg/dL (1.01 mg/dL 6 days prior). Of note, 5 months prior to presentation, she had one episode of AKI from which she recovered

completely, although this recovery was not fully explained by simple volume depletion or immunologic insult.

The patient was admitted for hydration and further investigation. On US assessment, there was no blood flow seen within the transplanted kidney. This prompted a nuclear scan of the allograft that showed normal blood flow to the kidney allograft with delayed secretory and excretory function of the allograft. As a result, a computed tomography (CT) scan was obtained (Fig 1A), which noted no abnormalities. Her pancreatic enzymes and liver function test results were within normal limits. A biopsy was performed that showed mild resolving acute tubular necrosis without evidence of rejection.

She was discharged home with close follow-up, daily laboratory tests, and home intravenous infusions to maintain her hydration due to her bladder-drained pancreas. After 7 days of treatment, her Cr level slowly decreased to 4.68 mg/dL, however, she continued to experience abdominal discomfort for which she was seen and another abdominal CT scan was obtained, which was remarkable for 180-degree inversion of the kidney allograft (Fig 1B). A diagnosis of KAT was made and the patient was taken for operative exploration.

Upon surgical exploration through the previously made midline incision, the kidney was found to be congested, slightly darker than usual, and rotated 180 degrees around its pedicle with the lower pole up and the hilum directed laterally (Fig 2A). The kidney was detorsed and pexied with noticeable change in color from reperfusion (Fig 2B). An

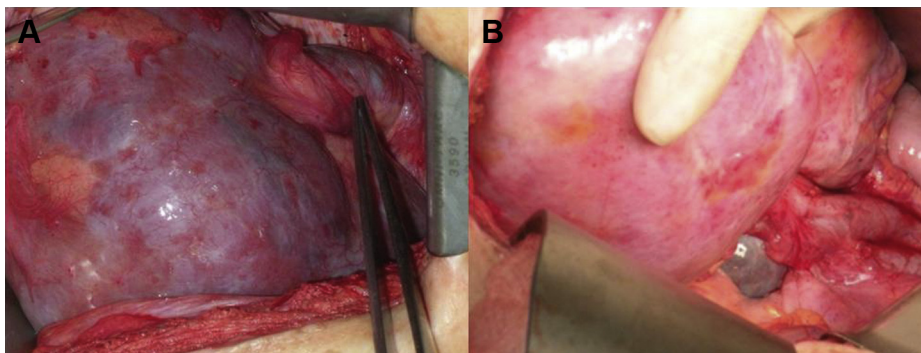


Fig 2. Intraoperative pictures of kidney allograft torsion. Upon exploration the kidney was found to be congested, slightly darker than usual, and rotated 180 degrees around its pedicle with the lower pole up and the hilum directed laterally (**A**). The kidney was detorsed and pexied with noticeable change in color from reperfusion (**B**).

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