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Permalink

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Journal

Journal of surgical case reports, 2017(5)

ISSN

2042-8812

Authors

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Publication Date

2017-05-01

DOI

10.1093/jscr/rjx033

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Peer reviewed



doi: 10.1093/jscr/rjx033 Case Report

CASE REPORT

Post-transplantation nephroptosis causing recurrent episodes of acute renal failure and hypertension secondary to intermittent vascular torsion of intraperitoneal renal allograft

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Abstract

Nephroptosis is a rare complication in renal transplantation, but one with significant associated risk. Due to non-specific clinical features, there may be a substantial delay in diagnosis and loss of the transplanted kidney due to renal pedicle thrombosis. We present a case of post-transplantation nephroptosis after simultaneous pancreas and kidney transplant, which resulted in accelerated hypertension and reversible acute kidney injury >1 year after transplantation. Prompt detection of this rare entity leading to expeditious surgical intervention is necessary to preserve viability of the renal allograft.

INTRODUCTION

Traditionally, Nephroptosis has been referred to as 'floating kidney syndrome'. This was thought to be due to congenital deficiency of Gerota's fascia allowing for excessive, retro-peritoneal renal mobility. Severe complications can result including ureteral obstruction, intermittent vascular occlusion or traction of hilar nerves causing flank pain. Episodic hypertension can develop due to activation of the renin–angiotension–aldosterone axis that occurs with reduced renal blood flow from intermittent arterial occlusion. Symptomatic patients are treated with nephropexy that prevents free rotation of the kidney around its fixed vascular pedicle. While this phenomenon is well-described in the general population, there are few reports of nephroptosis in renal transplantation. Renal allografts are typically transplanted

in the iliac fossa which limits potential for migration, although intra-abdominal transplantation is performed in simultaneous kidney-pancreas transplant (SPK) or dual renal transplantation. Most patients develop sufficient intra-abdominal scar tissue so allograft mobility is restricted; however, this does not always occur. We present a case report with pathologic post-transplantation nephroptosis after SPK successfully treated with nephropexy.

CASE PRESENTATION

A 43-year-old Caucasian man with Type I diabetes mellitus and subsequent end-stage renal disease underwent uncomplicated simultaneous deceased donor renal and pancreas transplant with systemic venous and enteric drainage in January 2014.

Received: October 23, 2016. Accepted: April 26, 2017

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Fifteen months after the procedure, he presented to the emergency room with hypertensive emergency and acute kidney injury (AKI). He was admitted for blood pressure management and discharged after hypertension was controlled and renal function normalized. Ultrasound was performed and found to be normal. One month later, he returned to the emergency room with AKI and refractory hypertension requiring esmolol drip for blood pressure control. An abdominal computed tomography (CT) scan was obtained showing a 1.2 cm adrenal nodule that prompted evaluation for functional adenoma, however workup was negative. Antihypertensive regimen was adjusted and he was discharged after normalization of blood pressure and serum Cr. One month later, he again presented with hypertensive emergency, AKI and new-onset substernal chest pain. He underwent repeat ultrasound showing increase in flow velocities of the arterial anastomosis suggestive of transplant artery stenosis (Fig. 1). CT scan was reviewed and showed migration and rotation of the transplanted allograft

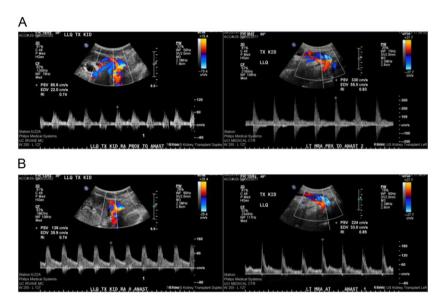


Figure 1: Comparison of duplex ultrasound of transplanted kidney in August 2015 compared to September 2015. Elevated velocities proximal and at the main renal artery anastomosis to 330 cm/s from 85.6 cm proximal to anastomosis (A) and 224 cm/s from 139 cm/s at the site of the anastomosis (B).

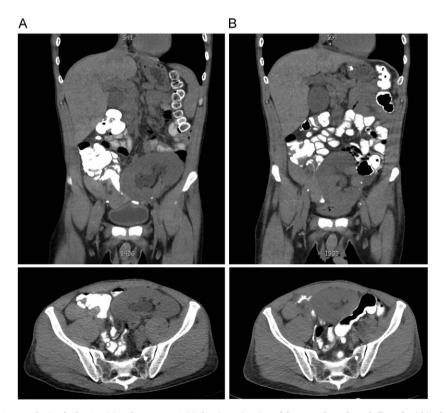


Figure 2: Comparison of CT scans obtained July 2015 (A) and August 2015 (B) showing migration of the transplanted renal allograft within the peritoneal cavity.

when compared with prior imaging (Fig. 2). On this basis, he underwent exploratory laparotomy. Intraoperatively, there was a noted paucity of adhesions with a freely mobile allograft. Manipulation of the transplanted kidney resulted in significant intraoperative blood pressure elevation. Suture nephropexy of the renal allograft to the lower abdominal wall was performed. An intraoperative kidney biopsy was obtained which confirmed the absence of rejection. Since the procedure, renal and pancreatic function have been stable with no further episodes of AKI or accelerated hypertension.

DISCUSSION

Nephroptosis in a transplanted renal allograft is an uncommon occurrence. When complete torsion of the renal axis occurs, AKI can develop and be easily confused with more common causes of acute renal failure such as rejection or obstruction. This can delay diagnosis and lead to irreversible allograft damage secondary to prolonged ischemia. A high index of suspicion is necessary to facilitate diagnosis of this rare phenomenon. Intraperitoneal renal transplant places the allograft into a larger compartment than the iliac fossa, which may predispose patients to developing post-transplantation nephroptosis [1]. In addition, patients receiving sirolimus have been shown to have impaired wound healing and presumably are also deficient in inflammatory cytokines necessary to form intra-abdominal adhesions, allowing free rotation to occur [2].

Presenting symptoms of nephroptosis most commonly include AKI, abdominal pain, decreased urine output and nausea/vomiting [3]. Doppler studies may reveal elevated flow velocities or complete occlusion of the vascular pedicle, but may also be normal if there is no active torsion of the renal allograft. Most common changes seen in a single study were shown to be reversed arterial diastolic flow, often with vascular thrombosis [1]. Identifying positional variations in renal blood flow may also be useful, although this has not been extensively studied. Sequential CT scans may also demonstrate migration of the renal allograft or change in axis, as was the case described above and has been reported previously as well [4].

Prophylactic suture nephropexy may be undertaken at the time of transplantation to reduce the risk of post-operative nephroptosis [3]. We now routinely perform this during intraabdominal renal transplants at our institution.

Post-transplant nephroptosis is a rare but potentially severe complication of intraperitoneal kidney transplant that can be successfully managed with appropriate surgical intervention [5]. It should be suspected in all patients with acute vascular compromise or recurrent episodes of renal failure without discernable cause. Transplant surgeons and nephrologists should be familiar with this unusual but surgically manageable cause of AKI.

CONFLICT OF INTEREST STATEMENT

None declared.

REFERENCES

- 1. Wong-You-Cheong JJ, Grumbach K, Krebs TL, Pace ME, Daly B, Chow CC, et al. Torsion of intraperitoneal renal transplants: imaging appearances. AJR Am J Roentgenol 1998;171:1355-9.
- 2. Dean PG, Lund WJ, Larson TS, Prieto M, Nyberg SL, Ishitani MB, et al. Wound-healing complications after kidney transplantation: a prospective, randomized comparison of sirolimus and tacrolimus. Transplantation 2004;77:1555-61.
- 3. Lucewicz A, Isaacs A, Allen RD, Lam VW, Angelides S, Pleass HC. Torsion of intraperitoneal kidney transplant. ANZ J Surg 2012;82:299-302.
- 4. Dewan R, Dasyam AK, Tan H, Furlan A. Renal allograft torsion: US and CT findings of a rare posttransplant complication. Case Rep Radiol 2016; 4273780.
- 5. Kaynar K, Sonmez B, Kutlu O, Ulusoy S, Cansiz M, Turkyilmaz S, et al. A case of recurrent episodes of acute renal allograft failure caused by renal pedicle tortion. Ren Fail 2013;35:556-9.