

Allograft loss from acute Page kidney secondary to trauma after kidney transplantation

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Abstract

We report a rare case of allograft loss from acute Page kidney secondary to trauma that occurred 12 years after kidney transplantation. A 67-year-old Caucasian male with a past surgical history of kidney transplant presented to the emergency department at a local hospital with left lower abdominal tenderness. He recalled that his cat, which weighs 15 lbs, jumped on his abdomen 7 d prior. On physical examination, a small tender mass was noticed at the incisional site of the kidney transplant. He was producing a normal amount of urine without hematuria. His serum creatinine level was slightly elevated from his baseline. Computer tomography revealed a large subscapular hematoma around the transplant kidney. The patient was observed to have renal trauma grade II at the hospital over a period of three days, and he was finally transferred to a transplant center after his urine output significantly decreased. Doppler ultrasound demonstrated an extensive peri-allograft hypoechoic area and abnormal waveforms with absent arterial diastolic flow and a patent renal vein. Despite surgical decompression, the allograft failed to respond appropriately due to the delay in surgical intervention. This is the third reported case of allograft loss from acute Page kidney following kidney transplantation. This case reinforces that kidney care differs if the kidney is solitary or a transplant. Early recognition and aggressive treatments are mandatory, especially in a case with Doppler signs that are suggestive of compression.

Key words: Page kidney; Kidney transplantation; Trauma; Subcapsular hematoma; Doppler ultrasound

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Core tip: We experienced a rare case of allograft loss from acute Page kidney secondary to trauma that occurred 12 years after kidney transplantation. This case reinforces that care for a transplanted kidney differs from care of a native kidney. Early recognition and aggressive treatments are mandatory, especially when Doppler signs suggest there is compression of the transplanted kidney. To the best of our knowledge, our case is the third case of allograft loss from Page kidney following kidney transplantation.

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INTRODUCTION

The Page kidney (PK) phenomenon occurs with compression of the kidney by a hematoma or mass, leading to arterial hypertension^[1]. More than 100 cases have been described in the literature^[2-4]; however, no systematic review has focused on post-transplant PK. In this case, report, we describe a rare case of allograft loss from PK secondary to trauma that occurred 12 years after kidney transplantation. This is the third reported case of allograft loss from PK following kidney transplantation^[5,6]. We describe this case alongside a review of the literature.

CASE REPORT

A 67-year-old Caucasian male presented to the emergency department at a local hospital for left lower abdominal tenderness. The patient had undergone a living unrelated kidney transplant into his left iliac fossa 12 years prior due to chronic glomerulonephritis. His stable immunosuppression regimen included tacrolimus (1 mg every 12 h), mycophenolate mofetil (500 mg every 12 h), and prednisone (5 mg daily). Except for one episode of acute cellular rejection a month after kidney transplantation, he had been doing well with a baseline serum creatinine level of 2.0 mg/dL. On arrival, his body temperature was 36.6 °C, blood pressure was 163/54 mmHg, and pulse was 61 beats/min. He reported that he had been active until the day before without noticing any injuries, but he recalled his cat, weighing 15 lbs, jumped on his abdomen seven days prior. On physical examination, his abdomen was soft and flat without rebound or guarding, except for a small tender mass noticed at the incisional site of the kidney transplant. His hemoglobin was 7.1 g/dL. His serum creatinine level was elevated from his baseline to 2.5

mg/dL. He was producing a normal amount of urine without hematuria. Computed tomography (CT) without intravenous contrast revealed a 12 cm × 2.5 cm subcapsular hematoma around the transplanted kidney (Figure 1). Urology was consulted, and the decision was made to conservatively observe the patient, as he met criteria of a renal trauma grade II according to the renal trauma grading system by the American Association for the Surgery of Trauma.

On admission, the patient received a red blood cell transfusion and was started on labetalol for hypertension. His systolic blood pressure was controlled within a range of 110-140. Within three days, his serum creatinine level increased to 5.4 mg/dL and his urine output decreased. His blood pressure was elevated up to 156/80 mmHg. The patient was transferred to a transplant center for further treatment.

At the transplant center, Doppler ultrasound (US) demonstrated an extensive peri-allograft hypoechoic area, abnormal arterial waveforms with absent diastolic flow in the arcuate arteries and a patent renal vein (Figure 2). He underwent emergent laparotomy for hematoma decompression. A substantial portion of the hematoma was evacuated by capsulotomy. Concurrent kidney biopsy showed no evidence of rejection. His postoperative course was uncomplicated and uneventful. The patient resumed tacrolimus, mycophenolate mofetil, and prednisone. However, his kidney function continued to deteriorate and he became dependent on hemodialysis. He is currently maintained with mycophenolate mofetil monotherapy and is awaiting a second kidney transplant.

DISCUSSION

PK was first described by Irvine Page in 1939, when he wrapped animal kidneys with cellophane and observed the development of acute hypertension^[1]. The typical presentation of PK is distinguished by the presence of acute renal dysfunction in conjunction with hypertension. Trauma, spontaneous hemorrhage in patients with predisposing factors (anticoagulation), bleeding after interventions (surgery, biopsy, nephrostomy, and lithotripsy), tumors, renal cysts, urinoma, and lymphocele have been proposed as etiological factors^[1-4]. Hypoperfusion and microvascular ischemia in the kidney are considered to stimulate the renin-angiotensin-aldosterone system and cause hypertension^[1]. If the involved kidney is solitary, or if the contralateral organ is damaged, renal failure may ensue. There are a variety of treatment options, including conservative management as the hematoma is absorbed^[7]; surgical decompression by capsulotomy as part of a laparoscopic intervention^[8]; and, in extreme cases, nephrectomy^[9,10]. Improvement of renal function after evacuation of the hematoma, in the absence of rejection or ureteral obstruction, confirms the diagnosis. In our case, CT demonstrated a large subcapsular hematoma compressing the parenchyma with a significant Doppler US finding of absent arterial diastolic

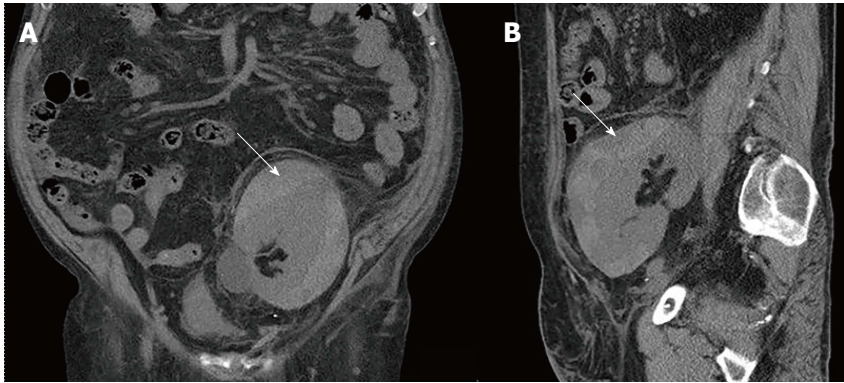


Figure 1 Computed tomography without intravenous contrast of the transplanted kidney. A: Coronal view; B: Sagittal view. A subscapular hematoma 12 cm × 2.5 cm in size was compressing the transplanted kidney (arrows).

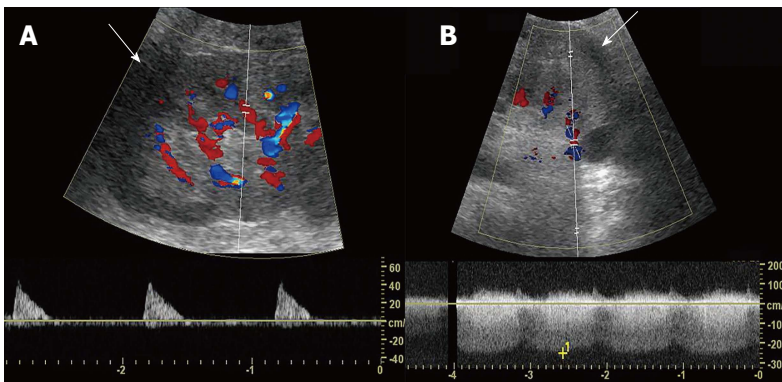


Figure 2 Presence of peri-allograft hematoma and Doppler ultrasound findings. A: Transplant arterial flow. Peri-allograft hypoechoic area (arrows) with absent diastolic flow in the arcuate arteries; B: Transplant venous flow. The transplant renal vein was patent.

flow with patent renal vein". Furthermore, a kidney biopsy failed to demonstrate evidence of rejection, which further supported the diagnosis of PK.

PK after kidney transplantation was first described in 1976 as "pseudorejection" by Cromie *et al*^[11]. This was because PK causes acute deterioration of graft function, which resembles rejection. Since then, 30 cases of post-transplant PK have been reported in the literature (Table 1)^[4-6,10-31]. The most common causes are iatrogenic (kidney biopsy in 18 cases, renal artery stenting in 1 case, ureteral stenting in 1 case, and nephrostomy in 1 case); trauma (3 cases)^[5,6]; spontaneous (2 cases)^[15,27]; and postoperative bleeding (2 cases)^[11,12]. Surgical decompression with capsulotomy and evacuation of hematoma have been performed in most cases (25 cases), and interventional radiographic drainage was performed in 1 case^[14], while 3 cases were conservatively observed with complete improvement of kidney function^[19,26]. The diagnosis is most commonly made by Doppler US findings of "absent arterial diastolic flow, reversible arterial diastolic flow, or significant increase of arterial resistive index, with a large peri-allograft hypoechoic area," suggesting extrinsic compression of renal parenchyma and subsequent cortical ischemia. In most cases, these findings have prompted surgical or radiographic intervention. On the other hand, allograft losses have been reported in 2 cases^[10,21]. One allograft was saved with a surgical intervention performed 2 d after the onset^[11], while one allograft was lost despite immediate intervention^[21]. In our case, the patient was observed at a local hospital and noted to have renal trauma grade II; the patient did not undergo Doppler US evaluation for the first three days of hospitalization. He

was finally transferred to a transplant center after his urine output significantly decreased. His graft loss may have been preventable if he had been evaluated with Doppler US upon presentation to the local hospital as well as if timely surgical intervention or transfer to a transplant center had been requested earlier. This case reinforces that care of the kidney differs if the kidney is solitary or a transplant. Early recognition of PK and aggressive treatments are mandatory, especially when Doppler findings suggest compression of a solitary or transplanted kidney.

We recommend the following care for acute PK. Patients without pre-existing kidney disease who have unilateral PK need to be admitted for monitoring of vitals, including blood pressure, heart rate, urine output, serum creatinine levels and hemoglobin levels. Abdominal/pelvic CT scan is preferable for accurate initial staging and diagnosis of the etiology of PK. Ongoing hemorrhage in a stable patient can be controlled by embolization with interventional radiology. After the initial diagnosis has been made, follow-up with US is appropriate. An initial attempt should be made to stabilize hypertension with antihypertensive medication. Conservative management and evaluation of the etiology are recommended as part of first-line treatment. Unstable patients might be more appropriate for surgery.

In the case of transplant patients, patients with a single kidney, and patients with bilateral PK, the patient should be transferred to a transplant center or a center capable of caring for the patient with acute PK and the underlying etiology. Vitals, including the blood pressure, heart rate and urine output, serum creatinine level and hemoglobin levels, should be carefully

Table 1 Acute Page kidney after kidney transplantation

Year	Ref.	Age/sex	Onset after transplant	Cause	Modality for diagnosis	Positive US sign ¹	Type of intervention	Intervention time after onset	Result
2016	Takahashi	67/M	12 yr	Trauma	US/CT	Yes	Surgical decompression	3 d	AL
2015	Sedigh <i>et al</i> ^[6]	67/M	12 yr	Trauma	US	Yes	Surgical decompression	12 h	CR
2015	Ay <i>et al</i> ^[12]	50/M	1 d	Postoperative bleeding	US	Yes	Surgical decompression	Immediately	CR
2014	Adjei-Gyamfi <i>et al</i> ^[13]	12/M	7 wk	Txp kidney biopsy	US/CT	No	Surgical decompression	Immediately	CR
2014	Adjei-Gyamfi <i>et al</i> ^[13]	18/F	1 yr	Txp kidney biopsy	US	No	Surgical decompression	Immediately	CR
2013	Hamidian Jahromi <i>et al</i> ^[14]	19/M	5 wk	Txp renal arterial stenting	US/Angio	Yes	IR drainage	6 h	CR
2012	Gandhi <i>et al</i> ^[15]	46/M	17 yr	Spontaneous	US	Yes	Surgical decompression	Immediately	CR
2011	Maurya <i>et al</i> ^[16]	30/M	7 d	Txp kidney biopsy	US/CT	Unknown	Surgical decompression	Immediately	CR
2011	Okecgukwu <i>et al</i> ^[17]	32/M	8 d	Txp ureter stenting	US	Unknown	Surgical decompression	Immediately	CR
2010	Butt <i>et al</i> ^[4]	61/F	24 d	Spontaneous	CT	-	Surgical decompression	Immediately	CR
2010	Posadas <i>et al</i> ^[18]	55/M	3 mo	Txp kidney biopsy	US	Yes	Surgical decompression	Immediately	CR
2009	Kamar <i>et al</i> ^[19]	47/M	1 yr	Txp kidney biopsy	US	Yes	Observation	-	CR
2009	Kamar <i>et al</i> ^[19]	59/M	1 yr	Txp kidney biopsy	US	Yes	Observation	-	CR
2009	Caldés <i>et al</i> ^[20]	60/M	1 mo	Percutaneous nephrostomy	US	Yes	Surgical decompression	24 h	CR
2008	Chung <i>et al</i> ^[21]	27/F	11 d	Txp kidney biopsy	US/CT	Yes	Surgical decompression	Immediately	CR
2008	Chung <i>et al</i> ^[21]	39/F	Several days	Txp kidney biopsy	US	Yes	Surgical decompression	Immediately	CR
2008	Chung <i>et al</i> ^[21]	35/M	4 d	Txp kidney biopsy	US/CT	Unknown	Surgical decompression	Immediately	AL
2008	Chung <i>et al</i> ^[21]	33/F	9 mo	Txp kidney biopsy	US	Yes	Surgical decompression	Immediately	CR
2008	Heffernan <i>et al</i> ^[22]	64/M	4 mo	Txp kidney biopsy	US	Yes	Surgical decompression	Immediately	CR
2007	Patel <i>et al</i> ^[23]	69/M	7 yr	Txp kidney biopsy	US/CT	Unknown	Surgical decompression	Immediately	CR
2005	Gibney <i>et al</i> ^[24]	32/M	1 yr	Txp kidney biopsy	US/Angio	Unknown	Surgical decompression	Immediately	CR
2000	Rea <i>et al</i> ^[25]	34/M	3 yr	Txp kidney biopsy	US	Yes	Surgical decompression	Immediately	CR
1996	Machida <i>et al</i> ^[26]	32/M	4 mo	Txp kidney biopsy	CT/Scinti	-	Observation	-	PR
1996	Goyal <i>et al</i> ^[5]	41/M	12 yr	Trauma	CT/MRI/Scinti	-	Unknown	Unknown	Unknown
1994	Nguyen <i>et al</i> ^[27]	26/M	12 h	Spontaneous	Scinti	-	Surgical decompression	Immediately	CR
1993	Dempsey <i>et al</i> ^[28]	19/F	2 yr	Txp kidney biopsy	US	Yes	Surgical decompression	Immediately	CR
1993	Ben Hamida <i>et al</i> ^[29]	32/M	7 mo	Heparin after renal vein thrombosis	US	Yes	Observation	-	CR
1991	Kliwer <i>et al</i> ^[10]	56/F	2 wk	Txp kidney biopsy	US	Yes	Nephrectomy	Unknown	AL
1988	Figuroa <i>et al</i> ^[30]	40/F	11 mo	Txp kidney biopsy	CT/Angio	-	Surgical decompression	30 h	CR
1988	Yussim <i>et al</i> ^[31]	40/F	5 mo	Postoperative lymphocele	US	Unknown	Surgical decompression	Unknown	CR
1976	Cromie <i>et al</i> ^[11]	35/M	10 d	Postoperative bleeding	US	Unknown	Surgical decompression	2 d	CR

¹ Absent diastolic flow, reversible flow, high resistive index at the transplant renal arteries, or increase in the RI from baseline by Doppler US. US: Ultrasound; CT: Computed tomography; IR: Interventional radiography; Txp: Transplant; AL: Allograft loss; CR: Complete resolution; Angio: Angiography; Scinti: Scintigraphy.

monitored. In addition to CT scanning for staging and diagnosis, Doppler US should be performed to evaluate parenchymal compression. Hypertension should be managed using antihypertensive medication and strict fluid balance. If the patient has an elevated serum creatinine level or a decrease in urine output as well as positive Doppler signs, prompt surgical intervention should be considered.

We experienced a rare case of allograft loss from acute PK secondary to trauma after kidney transplantation. The care of PK in a transplant kidney differs from PK in the native kidney. Early recognition and aggressive treatments are mandatory, especially in a case with positive Doppler signs.

COMMENTS

Case characteristics

A 67-year-old male with a past surgical history of kidney transplantation (12 years prior) presented to the emergency department for left lower abdominal tenderness after a cat jumped on his abdomen (seven days prior).

Clinical diagnosis

The abdomen was soft and flat without rebound or guarding, except for a small tender mass noted at the incision site of the kidney transplant.

Differential diagnosis

Lymphocele, urinoma, seroma, hematoma, renal cell cancer, renal cyst.

Laboratory diagnosis

On initial presentation, all labs were normal except for a hemoglobin of 7.1 g/dL and serum creatinine level of 2.5 mg/dL.

Imaging

Computed tomography without intravenous contrast revealed a 12 cm × 2.5 cm subcapsular hematoma around the transplanted kidney.

Pathological diagnosis

The transplant kidney biopsy showed no evidence of rejection.

Treatment

Emergent laparotomy for decompression of the hematoma.

Related reports

A renal trauma grade II is usually observed according to the renal trauma grading system of the American Association for the Surgery of Trauma.

Term explanation

The Page kidney phenomenon occurs from kidney compression by a hematoma or a mass, leading to arterial hypertension. If the involved kidney is solitary, or the contralateral organ is damaged, renal failure may ensue.

Experiences and lessons

This case reinforces that kidney care differs if the kidney is solitary or transplanted. Early recognition and aggressive treatments are mandatory, especially in a case with Doppler signs suggestive of compression.

Peer-review

The topic is very interesting. The authors presented their experience with Page kidney phenomenon after kidney transplantation. It is relatively infrequent complication but with possible serious complications on graft.

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