Acute Page kidney after angioplasty in kidney transplant allografts

Elizabeth Canllavi¹, Julio Teigell², Hernando Trujillo¹, Eduardo Gutiérrez¹,³, Natalia Miranda-Utrera², Enrique Morales¹,³,⁴

¹Department of Nephrology, Hospital Universitario 12 de Octubre, Madrid, Spain
²Department of Urology, Hospital Universitario 12 de Octubre, Madrid, Spain
³Instituto de Investigación del Hospital Universitario 12 de Octubre (imas12), Madrid, Spain
⁴Department of Medicine, Universidad Complutense de Madrid, Madrid, Spain

Correspondence to: Enrique Morales; E-mail: emoralesr@senefro.org

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ABSTRACT

Acute Page kidney (APK) in kidney transplantation is a rare entity often related to interventional techniques. Percutaneous angioplasty remains an exceptional cause of APK. Herein, we describe the clinical course and outcome of APK following percutaneous angioplasty for transplant renal artery stenosis in 4 kidney transplant recipients where external compression of the graft was caused by subcapsular hematomas. All patients were treated with surgical drainage, after which 2 cases recovered baseline kidney function, 1 developed advanced chronic kidney disease, and 1 remained dialysis-dependent. To our knowledge, the present series is the largest to describe APK in kidney allografts after percutaneous angioplasty.

Keywords: acute kidney injury, acute Page kidney, angioplasty, kidney transplantation, transplant renal artery stenosis
BACKGROUND

Acute Page kidney (APK) is defined as an extrinsic compression of the parenchyma, usually caused by a subcapsular hematoma, which is capable of inducing hypoperfusion and tissue ischemia with subsequent activation of the renin-angiotensin-aldosterone system, producing systemic hypertension and in some cases acute kidney injury (AKI) (1). There are few descriptions in the literature regarding APK in kidney transplant (KT) recipients; the majority of which are individual case reports resulting from a complicated kidney allograft biopsy (2). The relationship between APK and percutaneous luminal angioplasty (PLA) as treatment of transplant renal artery stenosis (TRAS) has not been well described. TRAS represents the most common vascular complication after kidney transplantation, usually presenting with difficult-to-control hypertension and graft dysfunction. Currently, PLA has become the treatment of choice for TRAS on the basis of its efficacy and safety profile. Nonetheless, it is not exempt from complications, which may occur in up to 10% of cases (3). APK following PLA of the kidney graft represents a rare but serious complication, potentially reversible if diagnosed and treated early. We aimed to describe the clinical course, management and outcome of patients with APK following angioplasty of the transplant renal artery.

CASE REPORTS

We identified four cases of APK in KT recipients following PLA of the transplant renal artery. Ages varied between 38 and 72 years. Two patients were female and two were male. APK occurred in a first KT in one case while two cases were on a second transplant and one case in a third transplant. Diagnosis of...

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RAS was done between the 2nd and the 6th month after transplantation. Most cases presented with impaired graft function whilst one patient also exhibited difficult-to-control hypertension and anasarca. Doppler ultrasound was the preferred diagnostic technique and PLA was the treatment of choice in all cases. The suspicion of APK was based on clinical symptoms and imaging findings by computed tomography (CT) or Doppler ultrasound (Figure 1). The four cases were managed with urgent surgical decompression with capsulotomy and evacuation of the subcapsular hematoma. Clinical presentation, treatment, and outcomes of our study population are detailed in Table 1. During a mean follow-up of 14.6 months, 2 cases recovered baseline graft function, 1 case developed advanced chronic kidney disease, and 1 case progressed to end-stage kidney disease (ESKD) with the need for chronic dialysis. Of note, one patient presented complete thrombosis of the graft artery stent, requiring transplantectomy.

DISCUSSION

APK is a well-recognized clinical entity in the native kidney, however, only few case series have been reported in KT patients (2,4). To our knowledge, the present series is the largest to describe APK in kidney allografts following PLA. McCune et al reviewed 80 cases of APK where only 2 patients (2.5%) were KT recipients; 1 case secondary to a perioperative hematoma and 1 associated to a fibrotic capsule formed after lymphocele drainage (4). Subsequently, 12 additional cases were published by Dopson et al where the most common causes included transplant biopsy, lymphocele, and graft hemorrhage with
hematoma formation (2). To date, only 1 case of APK following angioplasty has been reported (5) (Table S1).

TRAS represents a common complication after kidney transplantation and an important cause of graft dysfunction with potential serious outcomes (6). Currently, PLA is the standard of care for treating TRAS due to its high efficacy and relative safety. Nevertheless, several mechanisms can lead to APK after PLA, including spontaneous bleeding, guidewire-induced arterial perforation, arterial rupture or dissection, sudden increase in blood flow and pressure following relief of the arterial stenotic area, rupture of an arteriovenous fistula, and parenchymal bleeding from sites where previous biopsies have been performed (5). In our series, all APK cases were secondary to subcapsular hematomas. Two patients presented pseudoaneurysms, whereas 2 cases presented mild dissection of an intrarenal artery, which probably favoured the onset of Page kidney.

Clinical presentation of APK in the kidney graft is more expressive than in native kidneys since there is no contralateral kidney that can compensate for the function of the graft. Typical clinical features of APK in kidney allografts include pain in the graft area, AKI, and hypertension (2,4,7).

There is no definitive consensus on the treatment of APK. However, large subcapsular hematomas require an aggressive approach to preserve as much kidney function as possible and prevent long-term complications (8). Although hematomas or collections that cause APK can be drained percutaneously, surgical capsulotomy is mandatory in cases of uncontrolled pain, signs of arterial ischemia or rapidly growing hematomas due to active bleeding (4,7,9).
After surgical intervention, 50% of our patients exhibited complete clinical resolution.

In conclusion, APK in kidney transplantation is a rare but serious clinical entity, capable of compromising the graft. Awareness of this uncommon disease is key in order to establish an early diagnosis and prompt treatment which might be crucial to restore kidney function.

CONFLICT OF INTEREST STATEMENT

None declared.

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PATIENT CONSENT

The patients gave informed consent to publish their case.
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Table 1. Case series of APK in kidney transplant patients at our institution (2012 – 2019)

| Nº | Etiology                          | Age (years) | Sex | KT number | Baseline SCr (mg/dL) | APK symptoms                                      | Peak SCr (mg/dL) | Imagine techniques                  | Treatment                               | Outcome                     | Last SCr (mg/dL) |
|----|-----------------------------------|-------------|-----|-----------|----------------------|--------------------------------------------------|-----------------|-------------------------------------|----------------------------|-----------------|------------------|
| 1  | Angioplasty                        | 72          | Female | 2<sup>nd</sup> | 1.1                  | HTA, pain, hemodynamic instability, anuria        | 1.7             | CT                                  | Capsulotomy               | Recovery        | 1.1              |
| 2  | Angioplasty and stent placement    | 65          | Female | 2<sup>nd</sup> | 1.5                  | HTA, pain, oliguria                                | 7.5             | Doppler ultrasound, Percutaneous embolization of pseudoaneurysm + capsulotomy | Partial recovery       | 3.4             |
| 3  | Angioplasty and stent placement    | 69          | Male  | 3<sup>rd</sup> | 2.5                  | Anuria                                            | 7.1             | Ultrasound, CT, Capsulotomy          | Stent thrombosis + transplantectomy + chronic HD | 7.1             |
| 4  | Angioplasty                        | 38          | Male  | 1<sup>st</sup> | 2                    | HTA, pain, anemia                                  | 3.7             | Ultrasound, CT, Capsulotomy          | Recovery                  | 2               |

Abbreviations: APK: acute Page kidney; CT: computed tomography; HD: hemodialysis; HTA: hypertension; SCr: serum creatinine;
Legendes for Figures

- **Figure 1.** Doppler ultrasound findings in acute Page Kidney.

  **Figure A.** Grayscale ultrasound: kidney transplant in the left iliac fossa with a subcapsular hematoma. Hypoechoic collection (*) measuring 9.8×6.7 cm of subcapsular location that compresses the kidney graft parenchyma (r).

  **Figure B.** Spectral Doppler ultrasound: pseudoaneurysm. Inside the hematoma, a 14 mm vascular saccular lesion (white arrow) is identified with a communicating neck dependent of an intrarenal artery with characteristic “to and fro” pattern in spectral Doppler trace.

  **Figure C.** Contrast-Enhanced Ultrasound (CEUS), Computed tomography angiography (**Fig. D**) and selective kidney angiography (**Fig. E**), confirming the Doppler findings. Pseudoaneurysm (white arrow); subcapsular hematoma (*); compressed kidney graft parenchyma (r).

  **Figure F.** Post-embolization kidney angiography. Endovascular treatment was performed with supraselective embolization with microcatheter and Onix, attaining pseudoaneurysm occlusion (black arrow).

  **Figure G.** Color and spectral Doppler at 64 hours after embolization and surgical drainage showing a kidney graft measuring 10 cm, with normal echostructure and resolution of both the pseudoaneurysm and the hematoma, with recovery of normal intrarenal flow in spectral Doppler (resistive index 0.77, acceleration time 70 ms).

  **Figure H.** CEUS showing a regular and homogeneous uptake of kidney parenchyma, without areas of infarction.

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Figure 1. Doppler ultrasound findings in acute Page Kidney

940x529mm (150 x 150 DPI)