




EXCEPTIONAL CASE

Acute Page kidney after angioplasty in kidney transplant allografts

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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 four kidney transplant recipients, where external compression of the graft was caused by subcapsular haematomas. All patients were treated with surgical drainage, after which two cases recovered baseline kidney function, one developed advanced chronic kidney disease and one 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 haematoma, that is capable of inducing hypoperfusion and tissue ischaemia 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 ~10% of cases [3]. APK following PLA of the kidney graft represents a rare but serious complication that is 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

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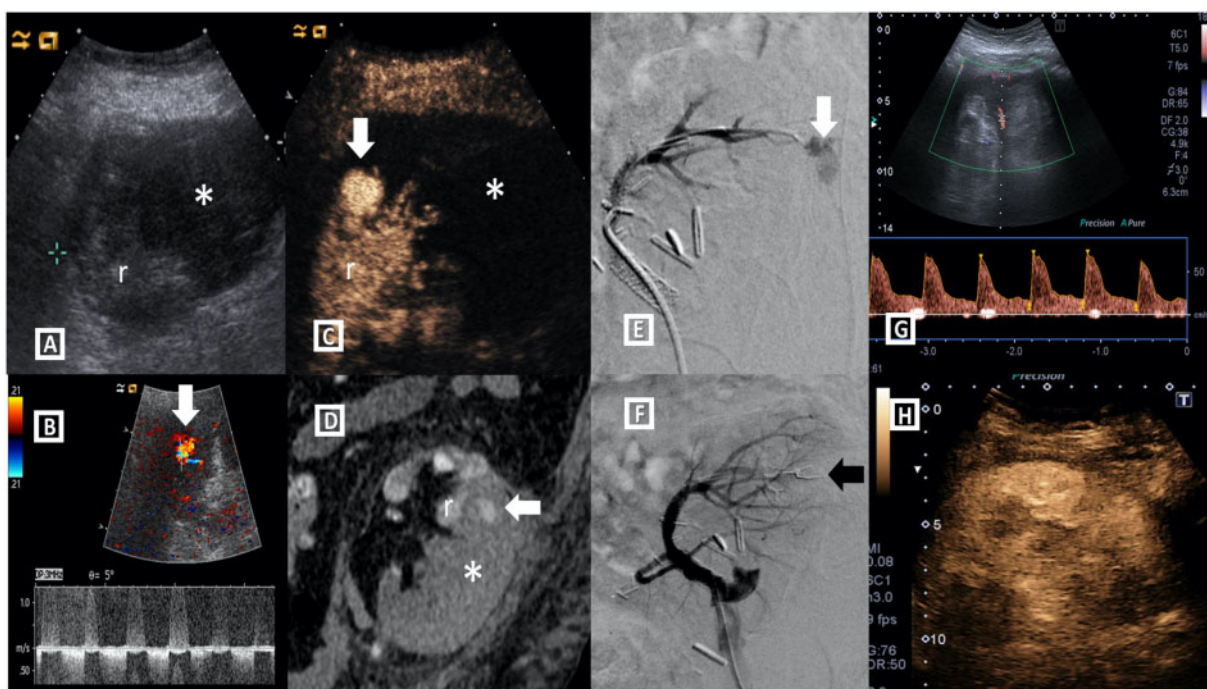


FIGURE 1: Doppler ultrasound findings in APK. (A) Grayscale ultrasound: KT in the left iliac fossa with a subcapsular haematoma. Hypoechoic collection (*) measuring 9.8×6.7 cm of a subcapsular location that compresses the kidney graft parenchyma (r). (B) Spectral Doppler ultrasound: pseudoaneurysm. Inside the haematoma, 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. (C) Contrast-enhanced ultrasound (CEUS), (D) CT angiography and (E) selective kidney angiography confirming the Doppler findings. Pseudoaneurysm (white arrow); subcapsular haematoma (asterisk); compressed kidney graft parenchyma (r). (F) Post-embolization kidney angiography. Endovascular treatment was performed with supraselective embolization with a microcatheter and Onix, attaining pseudoaneurysm occlusion (black arrow). (G) Colour and spectral Doppler at 64h after embolization and surgical drainage showing a kidney graft measuring 10 cm, with normal echostructure and resolution of both the pseudoaneurysm and the haematoma, with recovery of normal intrarenal flow in spectral Doppler (resistive index 0.77, acceleration time 70 ms). (H) CEUS showing a regular and homogeneous uptake of kidney parenchyma, without areas of infarction.

72 years. Two patients were female and two were male. APK occurred in a first KT in one case, while two cases were in a second transplant and one case in a third transplant. Diagnosis of RAS was done between Months 2 and 6 after transplantation. Most cases presented with impaired graft function, while 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 haematoma. Clinical presentations, treatments and outcomes of our study population are detailed in Table 1. During a mean follow-up of 14.6 months, two cases recovered baseline graft function, one case developed advanced chronic kidney disease and one case progressed to end-stage kidney disease 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 a 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.* [4] reviewed 80 cases of APK where only 2 patients (2.5%) were KT recipients; 1 case secondary to a

perioperative haematoma and 1 associated with a fibrotic capsule formed after lymphocele drainage. Subsequently 12 additional cases were published by Dopson *et al.* [2], where the most common causes included transplant biopsy, lymphocele and graft haemorrhage with haematoma formation. To date, only one case of APK following angioplasty has been reported [5] (Supplementary data, 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, a 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 haematomas. Two patients presented pseudoaneurysms, whereas two 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 haematomas require an aggressive approach to preserve as much kidney function as possible and

Table 1. Case series of APK in KT patients at our institution (2012–19)

Patient	Aetiology	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	1.1	HT, pain, haemodynamic instability, anuria	1.7	CT	Capsulotomy	Recovery	1.1
2	Angioplasty and stent placement	65	Female	2	1.5	HT, pain, oliguria	7.5	Doppler ultrasound	Percutaneous embolization of pseudoaneurysm + capsulotomy	Partial recovery	3.4
3	Angioplasty and stent placement	69	Male	3	2.5	Anuria	7.1	Ultrasound, CT	Capsulotomy	Stent thrombosis + transplantectomy + chronic HD	7.1
4	Angioplasty	38	Male	1	2	HT, pain, anaemia	3.7	Ultrasound, CT	Capsulotomy	Recovery	2

HD, haemodialysis; HT, hypertension; SCr, serum creatinine.

prevent long-term complications [8]. Although haematomas or collections that cause APK can be drained percutaneously, surgical capsulotomy is mandatory in cases of uncontrolled pain, signs of arterial ischaemia or rapidly growing haematomas due to active bleeding [4, 7, 9]. After surgical intervention, 50% of our patients exhibited complete clinical resolution.

In conclusion, APK in KT 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 that might be crucial to restore kidney function.

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CONFLICT OF INTEREST STATEMENT

None declared.

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