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Postangioplasty Infrequent Complication: A Page Kidney Case Report

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ABSTRACT

Page kidney phenomenon (PK) is hypereninemic hypertension triggered by any compressive mechanism on the kidney. Since it was first described in human beings in 1955, multiple causes of extrinsic compression of the renal parenchyma have been reported. However, to our knowledge, no cases related to arteriography with angioplasty as the cause of PK in a transplanted kidney has been reported. Clinical suspicion is always essential in making an accurate diagnosis. Treatment should be individualized. Patients who suffer PK should be monitored due to the possibility of chronic hypertension.

INTRODUCTION

In 1939, Irwin Page [1] demonstrated experimentally that wrapping dog kidneys in cellophane caused constrictive perinephritis, favoring microvascular ischemia of the renal parenchyma, and juxtaglomerular cell hyperplasia, thereby stimulating the renin-angiotensin-aldosterone system and reabsorption of sodium and water, generating hypertension.

Page kidney (PK) is an uncommon and severe clinical disease. Since 1955, when Engel and Irwin Page [2] first described a PK in human beings, a rugby player who had received blunt trauma to the side, very few cases have been reported in the literature.

We are presenting a case of PK treated at our hospital after an angioplasty in a kidney transplant patient, which is an uncommon complication and to our knowledge not described in the literature until now.

CASE REPORT

A 38-year-old male who received his first kidney transplantation from a donation after circulatory determination of death with the graft implanted in the left iliac fossa, posttransplant serum creatinine was 2 mg/dl. A progressive stricture of the renal artery, which caused impaired renal function, was diagnosed 6 months after kidney transplantation, whereupon a scheduled arteriography and angioplasty were performed. At the time of the angioplasty, the serum creatinine was 3.74 mg/dl, with a creatinine clearance of 27.2 mL/min, a urine output of 2800 cc, and the proteinuria was 0.73 g/24 hours.

Seventy-two hours after the angioplasty, the patient presented sudden pain, mass effect in the left iliac fossa, anuria, impaired renal function (serum creatinine 7.8 mg/dl), hypertension (171/107 mmHg), and severe anemization.

An urgent ultrasound of the renal graft showed a heterogeneous collection of 3 cm x 8 cm deforming the contour of the upper pole of the kidney, consistent with subcapsular hematoma. A computed tomography (CT) scan was requested to provide a better assessment of possible active bleeding, which was not confirmed (Figure 1 and Figure 2). The nephrographic phase showed a heterogeneous uptake of the renal parenchyma, suggesting poor perfusion. In view of the patient's acute severe clinical situation, a capsulotomy and urgent surgical drainage of the hematoma was performed. In the days postoperation, the patient's renal function improved up to his baseline values

KEYWORDS: Hypertension, page kidney, renal artery stenosis, renal transplantation, subcapsular hematoma, ultrasound

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Figure 1. Computed tomography scan showing hematoma surrounding the transplanted kidney.

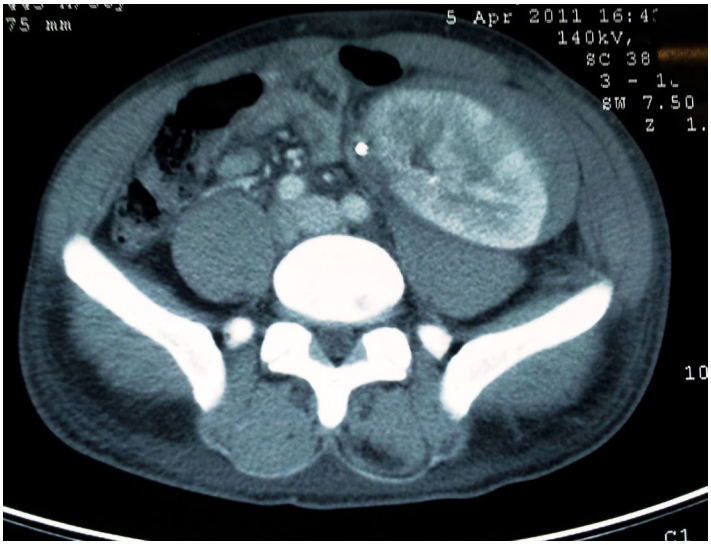
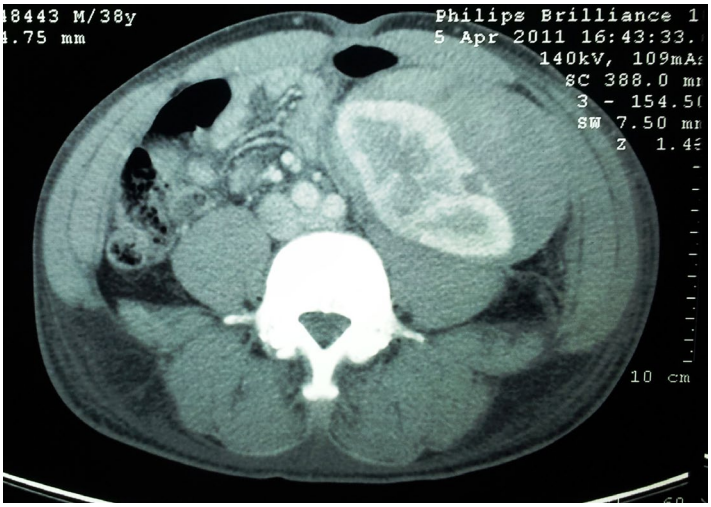


Figure 2. Computed tomography scan showing hematoma surrounding the transplanted kidney.



(creatinine 3.3 mg/dl 9 days after the capsulotomy) and to normal urine output. However, a certain degree of hypertension persisted, which required medical treatment to control it.

DISCUSSION

Page kidney phenomenon is hypereninemic hypertension triggered by any compressive mechanism on the kidney. Multiple causes of extrinsic compression of the renal parenchyma have been reported. Blunt trauma used to be the most common cause of subcapsular hematoma, but its incidence has tapered off in recent years and it currently accounts for only 30% (classic PK) [3]. In recent decades, the kidney biopsy is undoubtedly the main etiological agent of subcapsular hematoma [3-6] with an estimated incidence of 35% [3]. Many other less frequent causes of compression have been described in the literature: percutaneous nephrostomy, percutaneous radiofrequency ablation, partial nephrectomy, ureteroscopy, shock-wave lithotripsy, lymphoceles, renal cysts, pararenal tumors, urinomas, and unknown etiology [7-9]. To date, we have no knowledge of any reported case of arteriography with angioplasty on one of the main renal arteries as the cause of PK in a transplanted kidney.

The clinical manifestations of KP will depend on the patient characteristics and how rapidly the compressive effect on the renal parenchyma kicks in. Hypertension is the constant and characteristic sign of PK, mainly due to the pathophysiology of the phenomenon. In double-kidney patients, or those in whom the compressive effect is progressive and not acute, the hypertension may go unnoticed on being confused with essential hypertension, particularly if PK is not suspected. In single-kidney patients, the clinical manifestations are more striking because they have no other kidney to compensate for the functional deficit of the affected kidney. Thus, oligoanuria and acute renal failure with impaired creatinine and glomerular filtration rate will be more evident in these patients. Acute anemization presents mainly in cases in which the cause of compression is hemorrhage. In our case, and as the patient has a kidney transplantation, we observed all these clinical manifestations acutely: anemization, oligoanuria and impaired renal function, and sudden hypertension with poor drug control. Moreover, pain in the graft area and the mass effect in the left iliac fossa were observed.

Clinical suspicion is always essential in making the diagnosis. Ultrasound is the technique of choice because it is quick and harmless for the patient, and it is also highly sensitive and specific for identifying renal abnormalities [3]. Moreover, CT scan provides greater detail and precision of the compressive cause, its nature, and anatomical relationships. There are no guidelines or consensus-based recommendations on the procedure for the treatment of PK. Therefore, treatment should be individualized.

The different authors do seem to agree that one of the first measures is pharmacological control of hypertension. Drug treatment will only suffice in 18% of cases in which there is only hypertension [3,10]. Kidney decompression will be necessary in all other cases. The use of angiotensin-converting enzyme inhibitors is more than justified [11]. However, if they are

contraindicated, as was the case in our patient who presented renal artery stricture, diuretics, beta-blockers, and calcium channel blockers may be used. Decompression can be performed through percutaneous drainage or by surgical drainage and capsulotomy, as was necessary in our case due to its severity. In cases in which the cause of the extrinsic compression is not a hematoma, this cause can be treated specifically. Nephrectomy should only be considered in selected cases, as it has been proven that conservative management and adequate drainage of the hematoma can solve acute cases. These patients must be closely monitored, as it is known that up to one third of patients with PK, with no previous history of hypertension, will develop chronic hypertension despite receiving suitable initial treatment [11].

CONCLUSIONS

PK is a rare and serious condition that can lead to loss of the kidney. Clinical suspicion is essential in all patients with a history of kidney manipulation, like an arteriography with angioplasty. Diagnosis by imaging, ultrasound, or a CT scan is fast and accessible for most centers. Surgical drainage of the hematoma and capsulotomy are the most effective options, but treatment should be individualized because some cases could be subsidiary to drug treatment and percutaneous drainage. Patients who suffer PK should be monitored due to the possibility of chronic hypertension.

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