

Page Kidney – Rare but Correctable Cause of Hypertension

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ABSTRACT

A case of Page kidney as a result of injury followed by hypertension is presented. An abdominal ultrasound revealed soft tissue mass suggestive of a hematoma, resulting in a nephrectomy during which there were dense adhesions on the posterior surface of the kidney due to a large subcapsular hematoma. This presentation aims to bring attention to Page kidney's causes associated with hypertension and its correctable treatment.

KEYWORDS: Page kidney; Subcapsular hematoma; Hypertension

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INTRODUCTION

Page kidney is a rare phenomenon of hyperreninemic hypertension caused by compression of the renal parenchyma leading to a subcapsular hematoma. It has been reported in healthy individuals after abdominal trauma and after invasive nephrological interventions. We present a rare case of Page kidney following a trivial trauma. He underwent nephrectomy and he had a complete recovery.

CASE REPORT

A 53-year-old gentleman had dull, aching, left loin pain for 10 days. He was not a known hypertensive or diabetic. On examination, his BP was 180/110 mm of Hg and systemic examination was unremarkable. He was clinically diagnosed with left ureteric colic. An abdominal ultrasound revealed a soft tissue mass of 23 mm thickness in the perinephric space suggestive of a hematoma (Figure 1), and the color Doppler showed no flow within the mass. Upon probing, he remembered a trivial road traffic accident 6 months back but he remained asymptomatic after trauma. His renal parameters

were normal. A clinical diagnosis of left Page kidney was made on the basis of a presence of a triad of hypertension, perinephric hematoma, and left loin pain. A DTPA renogram was done, which showed no cortical tracer uptake in the left kidney. A medical consult was obtained for uncontrolled hypertension and he was started on ACE inhibitors, and his BP was controlled. He underwent nephrectomy. Intraoperatively, there were dense adhesions on the posterior surface of the kidney due to the presence of a large subcapsular hematoma (Figure 2). Following nephrectomy, his blood pressure was normal and he did not require further antihypertensives. A CT scan of the specimen kidney was done for academic interest (Figure 3).

DISCUSSION

Page kidney is the external compression of a kidney usually caused by a subcapsular hematoma [1]. It was named after Dr. Irvin Page (1901–1989) who first produced a renin-dependent model of hypertension by wrapping a dog kidney in cellophane. The clinical equivalent of this hypertensive model is the kidney compressed by a subcapsular or perirenal process causing renal ischemia leading to unilateral hypersecretion

Figure 1. USG picture showing perinephric hematoma

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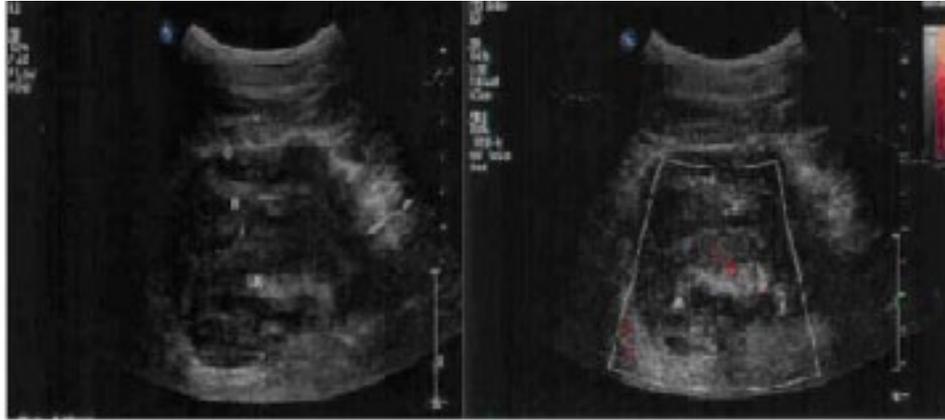


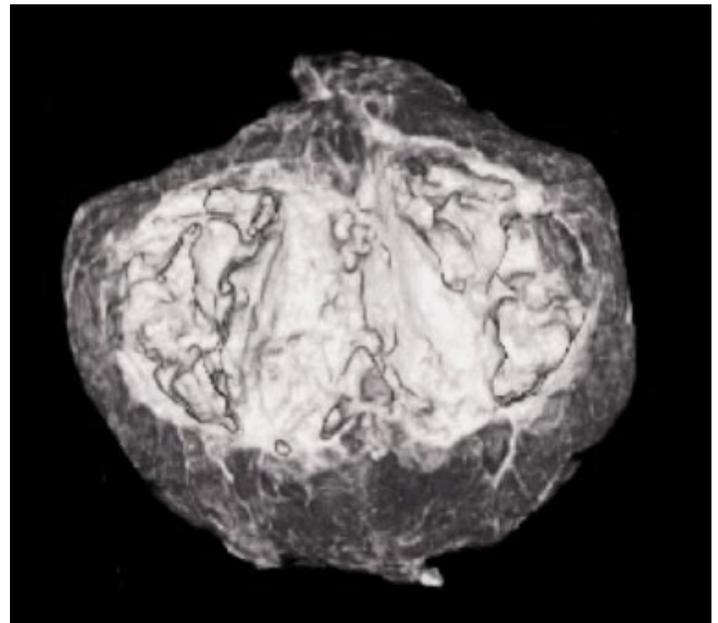
Figure 2. Gross specimen of Page kidney

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Figure 3. CT confirming thinned out renal parenchyma with thickened perinephric fat

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of renin and contralateral suppression. Subcapsular bleeding complicates renal biopsy and ESWL but usually does not cause hemodynamic instability. Although hypertension is the most common feature, renal insufficiency can occur in the setting of a diseased contralateral kidney, single functioning kidney, or a renal allograft [1]. Following injury, the onset of hypertension ranges from days to decades, with a mean of 36 months [2]. The pathophysiology of hypertension associated with renal

compression from perinephric hematoma or chronic perirenal scarring results from an intrarenal artery or segmental branch stenosis [3]. One explanation is that rapid deceleration stretches the renal artery, causing an intimal injury that leads to arterial obstruction and ischemia. The infarcted kidney develops collateral blood flow from the ureteric

and perinephric capsular systems and the direct exchange between perforating capsular vessels. Collateralization results in a small but critical amount of renal perfusion. This leads to a preservation of the endocrine function of the kidney, activating an increase in the secretion of renin (normal, 1.9–3.7 ng/mL/h) and eventually causing hypertension [4, 5]. The diagnosis of Page kidney depends on the presence of either a surrounding hematoma or an encasing fibrous pseudocapsule. CT abdomen is the preferred modality as it is noninvasive and can detect even very small hematomas [6]. An MRI may be helpful in assessing the age of the hematomas and patency of renal blood vessels. The treatment of Page kidney aims to preserve renal function and cure hypertension [7]. The gold standard is selective renal arteriography with renal-vein renin assays. Lesions in the renal vasculature are readily identified and renal-vein renin values will confirm the functional significance of an identified lesion. Elevated renin production from the responsible kidney and suppressed renin production from the contralateral kidney in a ratio >1.5:1 or 2:1 will predict which lesions will respond to surgical treatment in more than 90% of cases [5].

Antihypertensive therapy and observation, percutaneous evacuation of the perirenal hematoma, open drainage of the hematoma, capsulectomy, partial nephrectomy, and nephrectomy have been used. The appropriate duration of medical therapy before resorting to more aggressive treatment is unclear, but irreversible parenchymal changes are likely to occur if the hypertension does not resolve within 1 to 2 years [8]. Direct renin inhibitor (i.e., aliskiren) can be used to adequately control BP until surgery can be performed. There are no published reports of the use of aliskiren in these types of cases, however direct renin inhibitors are known to control the hypertension. A hematoma may reabsorb, relieving the parenchymal compression without forming an adhesive fibrotic pseudocapsule. In acute cases, therapy starts with ACE inhibitors to control blood pressure while waiting for the local hematoma to be absorbed. Surgery is the treatment of choice when a documented renovascular obstruction or damage is present. Large liquid hematomas may respond to percutaneous drainage. Patients who present with old hematoma or severely impaired kidney function may need active intervention, including capsulectomy, partial nephrectomy (if compression is to a polar area), or a total nephrectomy [9]. Watts and Hoffbrand [10] reported in a review paper that nephrectomy cured hypertension in 89% of 18 patients. Although technically challenging, Page kidney has been treated laparoscopically with a dissection of the perinephric fibrosis [9].

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