

Recurrent Non-Traumatic Page Kidney

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The Page kidney phenomenon refers to hypertension resulting from any external compression of a kidney by a hematoma, tumor, lymphocele or urinoma. Hypertension develops due to activation of the renin-angiotensin-aldosterone system induced by renal hypoperfusion and microvascular ischemia. As such, Page kidney is a rare cause of high renin hypertension. Subcapsular or perinephric hematoma due to traumatic or iatrogenic hemorrhage accounts for the majority of cases. The interval between injury and the development of hypertension may vary from days to years. Presentation, however, may be acute to the point of a hypertensive emergency.

We report a patient who presented with repeated acute episodes of hypertensive urgency/emergency secondary to Page kidney caused by a spontaneously recurring subcapsular renal hematoma. Between episodes, the hematoma was seen to completely resolve. The possible etiology is discussed and a brief overview of Page kidney is given.

PATIENT DESCRIPTION

A 40 year old man was first admitted in August 2006 complaining of visual disturbances; his blood pressure was 209/140 mmHg. Fundoscopy showed grade IV hypertensive retinopathy (Keith-Wagener-Barker classification). Laboratory data revealed a serum cre-

atinine of 1.2 mg/dl and potassium 3.5 mEq/L. An electrocardiogram showed left ventricular hypertrophy. Despite being advised to the contrary, the patient discharged himself immediately upon admission. Relevant past history was notable for ambiguous childhood hypertension during which ambulatory blood pressure monitoring was performed and the patient was diagnosed as having white coat hypertension for which no antihypertensive treatment was administered.

In January 2007, he was hospitalized due to the sudden onset of severe right flank pain simulating renal colic. There was no history of any abdominal trauma. Blood pressure was 193/112 mmHg with bilateral papilloedema. Serum creatinine had increased to 1.67 mg/dl and potassium levels ranged from 2.5 to 3.5 mEq/L. Thyroid-stimulating hormone was within normal limits. Renal sonography demonstrated a large right subcapsular hematoma (12 x 5.5 cm), a finding confirmed by computerized tomography. BP1 was controlled by a combination of bisoprolol 5 mg once daily, aldospirone 25 mg once daily and lercanidipine 10 mg twice a day. A repeat ultrasound of the kidneys a year later (January 2008) showed the right kidney to be 9.6 cm in length, the left kidney 11.5 cm, and complete resolution of the hematoma. The patient proved to be extremely non-compliant regarding an orderly follow-up and medications.

In October 2009, he was again admitted with right flank pain and a BP of 240/150 mmHg. Upon treatment with intravenous nitroprusside and combined beta blocker and calcium channel blockers, the patient's BP

BP = blood pressure

stabilized at 140–150/80–90 mmHg. Serum creatinine had risen to 2.3 mg/dl. An abdominal CT revealed a large right subcapsular hematoma. He was discharged on atenolol 50 mg once daily, clonidine 0.15 mg three times a day and amlodipine 10 mg once daily.

A month later (November 2009) he presented once more with a BP of 195/125 mmHg and grade III hypertensive retinopathy. Renal sonography showed the right kidney to be 8.6 cm in length and the left kidney 10 cm, and corroborated the presence of the right subcapsular hematoma seen on CT, measuring 6.5 x 2.5 cm [Figure A]. Doppler ultrasound did not show any findings supportive of renal artery stenosis. Plasma renin activity was > 50 ng/ml/hr (range 0.2–2.8 ng/ml/hr) with a plasma aldosterone concentration of 43.1 ng/dl. Urinary catecholamines were all within the normal range. The patient was discharged on treatment with ramipril 5 mg twice a day, bisoprolol 5 mg once daily, amlodipine 5 mg twice a day and furosemide 40 mg once daily. Once again, he proved to be extremely non-compliant regarding an orderly follow-up and medications. As a result, outpatient blood pressure recordings between the above acute episodes were scarce and were reported to be 140–160/90–100 on treatment. Since then, repeat ultrasounds of the kidneys (September 2010 and February 2011) have not demonstrated any subcapsular hematoma [Figure B].

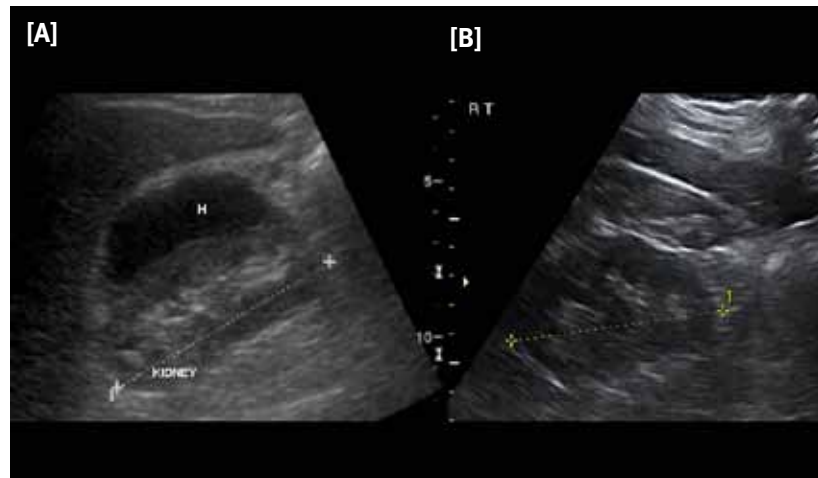
COMMENT

The entity known as Page kidney was originally described in 1939 by Page [1], who developed an animal model

of hypertension created by wrapping canine kidneys in cellophane. In 1955, Page followed up his original description with a clinical report of a male footballer found to be hypertensive, with a subcapsular renal hematoma and blood pressure that normalized after nephrectomy. The resulting hypertension of Page kidney is due to activation of RAAS (the renin-angiotensin-aldosterone system). Page kidney is, therefore, somewhat akin to the Goldblatt model of renovascular hypertension, except that in the latter there is compression or stenosis of major renal vessels whereas in Page kidney there is an ischemic renal vasculature. Hypokalemia, as seen in our patient, may develop due to secondary hyperaldosteronism.

Until 2008, a total of 108 cases of Page kidney were described [2]. In most cases compression of the kidney was caused by a traumatic subcapsular or perinephric hematoma. The literature review by McCune et al. included 80 cases [3]. Prior to 1991, the resultant hematoma was mainly a sports or motor vehicle accident-related injury. Since then, iatrogenic causes, in particular following kidney allograft biopsies (10/28), have increased in frequency.

Our case presents several unique features. First, the subcapsular hematoma was a recurring phenomenon with complete resolution between the acute episodes. Secondly, its occurrence was spontaneous and unrelated to any trauma or bleeding diathesis. Suspicion was therefore raised of a renal vascular malformation. Vo and co-authors [4] described a 16 year old boy whose congenital solitary functional kidney was complicated by a sports-related Page kidney. However, the recurrent nature of the hematoma eventually led to the discovery of dysplastic capsular arteries off the mid to lower pole of the kidney. Selective embolization of the cluster of these dysplastic arteries resulted in definitive resolution of the hematoma and easily manageable hypertension.



[A] Renal ultrasound (November 2009): longitudinal view of the right hyperechoic kidney surrounded by a sonolucent subcapsular hematoma (H)
[B] Renal ultrasound (February 2011): longitudinal view of the same right kidney showing complete disappearance (resolution) of the subcapsular hematoma

Although our patient was scheduled for a renal angiogram, he refused the procedure. Finally, our patient's recurrent episodes of acute Page kidney were manifested by the development of a hypertensive emergency. Over time, his renal function was seen to decline with progressive shrinkage of the affected kidney. It is noteworthy in this regard that compression-induced interstitial nephritis has been reported to play a role in both the hypertension and deterioration of renal function [5]. This factor as well as the ischemic renal vasculature probably account for the contraction of the affected right kidney. The left kidney also experienced some reduction in size compatible with the decline in renal function as a result of poorly controlled hypertension.

In the past, definitive treatment of Page kidney involved radical nephrectomy or open surgery to evacuate the hematoma or perform a decapsulating procedure. The current approach is toward less invasive procedures such as mesh hood fascial closure or medical management utilizing RAAS blockade medications.

In summary, Page kidney is a cause

RAAS = renin-angiotensin-aldosterone system

of secondary high renin hypertension that is easily ignored. Previously mainly related to trauma, the etiology has shifted to iatrogenic procedures, particularly kidney transplant biopsies. In the evaluation of recurrent non-traumatic Page kidney, renal angiogram should be considered.

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