The Page kidney phenomenon secondary to a traumatic fall

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Page kidney is a rare phenomenon of hyperreninemic hypertension caused by compression of the renal parenchyma. It has been reported in healthy individuals after blunt abdominal or flank trauma, and in patients after invasive nephrological interventions. We present a case of acute on chronic renal failure and Page kidney phenomenon in an elderly male after a traumatic fall, who underwent effective medical management until spontaneous recovery to baseline was observed. A brief discussion on the Page kidney phenomenon is provided with a suggested algorithmic approach towards the management of this process. ***European Journal of Emergency Medicine*** **17:24–26 © 2010 Wolters Kluwer Health | Lippincott Williams & Wilkins.**

European Journal of Emergency Medicine 2010, **17:**24–26

Keywords: aldosterone, angiotensin, hypertension, kidney, Page, renin

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Received 6 March 2009 **Accepted** 14 April 2009

Introduction

The Page kidney (PK) phenomenon was first described by Dr Irvine H. Page in 1939 [1]. It is defined as the external compression of a kidney by a subcapsular hematoma, cyst, or tumor, which results in renal hypoperfusion and consequently activates the renin–angiotensin–aldosterone axis causing severe hypertension. This phenomenon is an extremely rare occurrence with no prescribed standard management. With the variety of antihypertensive drugs available currently, optimum medical therapy should be implemented before any surgical or endovascular intervention, if possible.

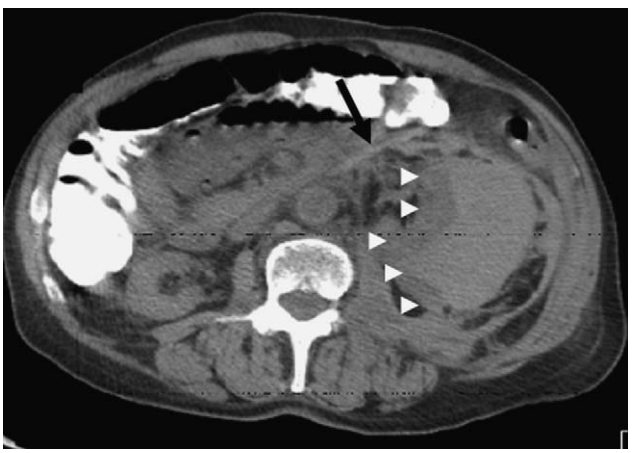
Case report

An 89-year-old diabetic, hypertensive male with a long-standing history of chronic renal insufficiency (serum creatinine 1.14, creatinine clearance 44.74 ml/min) and coronary artery disease, status – postmyocardial infarction within the past year, presented to our hospital with complaints of left-sided abdominal pain, nausea, and vomiting 3 weeks after a fall. He showed severe hypertension (220/110 mmHg) and tenderness to deep palpation in the left lower quadrant and costovertebral angle region. Labs revealed anemia (hematocrit 23%), hypokalemia (serum potassium, 3.1 mEq/l), and an elevated serum creatinine level (5.7). An abdominal nonintravenous contrast computed tomography (CT) scan revealed a large left subcapsular renal hematoma measuring 15 × 10 × 5 cm (Fig. 1). Acute-on-chronic renal failure and PK phenomenon was identified and the patient was treated with labetolol, felodipine, furosemide, and a nitroprusside drip. A packed red blood cell transfusion was provided with an appropriate response. The patient's blood pressure (Fig. 2) and renal function returned to his near-baseline values (creatinine 1.8 mg/dl) gradually over the next week. A repeat CT, 2 weeks after the initial, showed an unchanged hematoma and the patient was discharged home with normal blood pressure on clonidine, felodipine, and carvedilol. The patient was deemed an unsuitable candidate for operative interventions because of severe comorbidities. At a 9-month follow-up, the patient remains hemodynamically stable without worsening of his renal disease.

Discussion

PK is a rare condition resulting in hypertension secondary to renal compression. The phenomenon was initially described by Dr Irvine H. Page in an animal model in

Fig. 1



A nonintravenous contrast computed tomography of the abdomen shows a large subcapsular left renal hematoma (white arrowheads) compressing the renal pedicle. In addition, the left renal vein pushed away from the renal pelvis is seen (black solid arrow).

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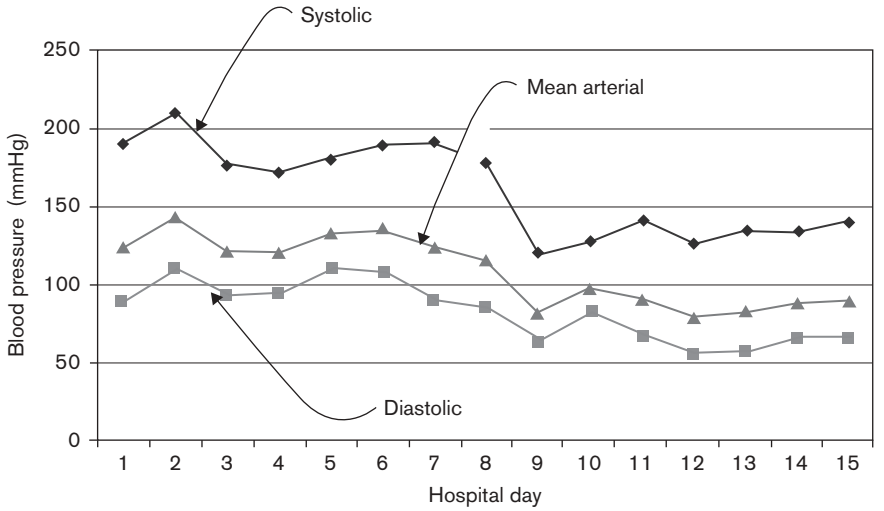
Fig. 2



Decrements in blood pressure with medical management in our patient.

1939 [1], and the first clinical case (1955) was that of a football player who had sustained a blunt renal injury [2]. Since then, a few cases of PK-mediated hypertension have been published.

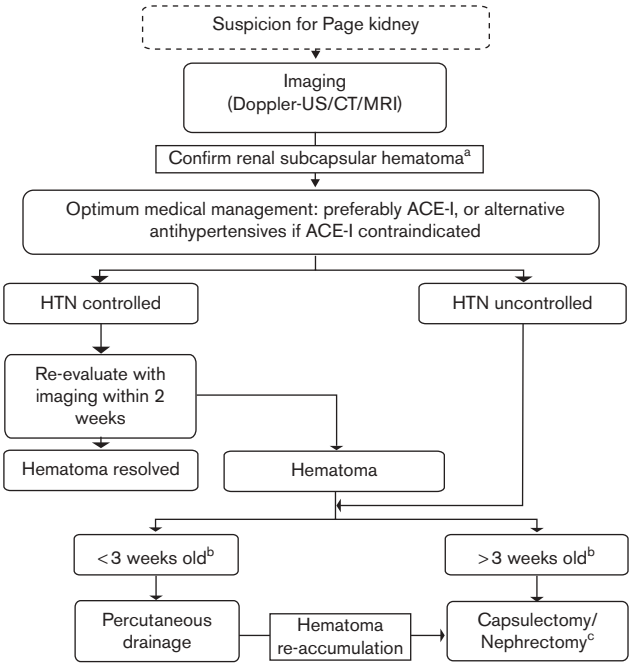
External compression of the renal parenchyma causes ischemia which is similar in mechanism to that described by Goldblatt *et al.* [3] who hypothesized that renal artery constriction leads to renal ischemia and subsequent renin-induced hypertension [4]. Interestingly, this phenomenon has been reported frequently in renal transplant patients who develop a perinephric hematoma [5] or lymphocele [6]. The most common cause of a compressive subcapsular renal hematoma is blunt trauma, although warfarin therapy and kidney biopsy have also been implicated in some cases [7].

Persistent activation of the renin–angiotensin–aldosterone axis may also cause chronic renal insufficiency. In the case of a normal contralateral kidney, compensation occurs without an apparent rise in creatinine. On the contrary, in the case of a single kidney or in those with chronic renal disease (as was seen in our patient), a decrease in glomerular filtration rate and acute renal failure may be pronounced [8].

Both Doppler-ultrasound (US) and CT are the preferred initial imaging modalities for suspected PK. In addition to demonstrating subcapsular hematoma, Doppler-US can assess renovascular perfusion (calculate resistive index) and in cases of transplant recipients, it can allow differentiation between rejection and mechanical causes of allograft failure [4,5]. US is safe and inexpensive, but limited in its resolution [9]. CT may be able to assess concomitant perinephric hematomas and may better define the subcapsular hematoma [4,9], however, increased serum creatinine may prohibit intravenous contrast administration. Magnetic resonance imaging (MRI) has an added advantage of the ability to estimate the age of the hematoma and determine the patency of the renal vessels [9] especially in cases where above-mentioned modalities may not be adequate [6]. Selective renal arteriography to exclude renovascular lesions and renal vein renin assay to confirm hyperreninemia is the gold standard diagnostic test for PK [4,9]. It is suggested that radioisotope renal scan may be helpful in assessing renal flow and excretory function [4].

The treatment of PK remains controversial. Relief of compression and restoration of renal perfusion normally leads to recovery of renal function and controls hypertension in most cases. The major factor affecting treatment is the age of the hematoma. Hematomas that are less than 3 weeks old usually resolve spontaneously. Hypertension usually does not ensue until the hematoma organizes into a restrictive fibrous pseudocapsule that compresses the kidney [10]. Hence, a trial period of observation may be warranted if the blood pressure is controlled with oral hypertensives, specifically angiotensin-converting enzyme inhibitors [9]. The use of angiotensin-converting enzyme inhibitors may not be a viable option in some patients, as in our case, that have underlying renal disease. However, if hypertension remains uncontrolled despite medical therapy, the hematoma should be drained percutaneously, laparoscopically, or with an open procedure. Hematomas that are more than 3 weeks old and have transformed into a pseudocapsule are usually not amenable to percutaneous drainage and may require a capsulectomy or nephrectomy. It must be noted that percutaneous drainage and capsulectomy are

Fig. 3



Suggested management algorithm for Page phenomenon secondary to renal subcapsular hematoma. ACE-I, angiotensin-converting enzyme inhibitor; CT, computed tomography; HTN, hypertension; MRI, magnetic resonance imaging; US, ultrasound.
[a]Assuming contained hematoma without continued hemorrhage; otherwise, primary goal in management is achievement of hemostatis (endovascular or open surgical).
[b]Age of hematoma.
[c]Should be individualized according to patient's clinical and hemodynamic status, and comorbidities.

not definitive treatment procedures, as reaccumulation of fluid may occur or there may be incomplete excision of the fibrous pseudocapsule [4]. As mentioned earlier, no standard management of PK exists, and anecdotal reports of this phenomenon secondary to subcapsular hematoma yield several different successful treatment strategies. These strategies range from medical therapy alone to radiological drainage of subcapsular hematoma to capsulectomy or consequent nephrectomy in patients failing to respond to antihypertensives and/or radiological drainage. Figure 3 shows our suggested management algorithm for PK phenomenon secondary to renal subcapsular hematoma.

In conclusion, PK is a rare clinical entity and its diagnosis is based on a high index of suspicion. Although the etiology leading to this phenomenon can be evaluated with Doppler-US and MRI, CT scan (with/without contrast) should be the initial diagnostic modality of choice given its less-operator dependency, image resolution capabilities, and availability in emergency/trauma centers. The goal of treatment is to preserve renal function and achieve blood pressure control. As suggested in the algorithm (Fig. 3), selection of treatment strategy should be individualized for each patient with renal subcapsular hematoma causing PK phenomenon.

Acknowledgements

Authors declare that there are no conflicts of interest, and they accepted no financial sponsorship in producing and presenting this article. They are in agreement with and approve the content of the manuscript. In addition, the manuscript is not under consideration for publication elsewhere in a similar form, in any language.

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