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Case Report

Page kidney: an unusual complication of image-guided native renal parenchymal biopsy - case report and literature review☆,☆☆

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ABSTRACT

Page kidney is a pathologic phenomenon in which extrinsic compression of renal parenchyma from a subcapsular collection causes secondary systemic hypertension, via activation of the renin-angiotensin-aldosterone system. Following the first description of Page kidney, the condition was most often recognized following blunt trauma to the flank. Increasingly, non-traumatic and iatrogenic etiologies of Page kidney have been described. We present a case of Page kidney as a complication of image-guided native renal parenchymal core needle biopsy. The current literature on etiologies, pathophysiology, and treatment options for Page kidney are summarized.

Introduction

Page kidney is a rare cause of secondary hypertension arising from extrinsic compression of the renal parenchyma. Increasingly, iatrogenic injury to the kidney is recognized in the literature as a leading cause of Page kidney. Percutaneous renal biopsy is a common procedure performed by Interventional Radiologists and Nephrologists which is commonly complicated by hemorrhage from the renal parenchyma. If a subcapsular hematoma forms following a renal biopsy, Page kidney may develop. In this case, we describe a case of Page kidney arising from the percutaneous biopsy of a native kidney, the imaging features of this entity, and subsequent management of the patient. This is followed by a brief literature review summarizing the current available literature on the etiology, pathophysiology, and management of Page kidney.

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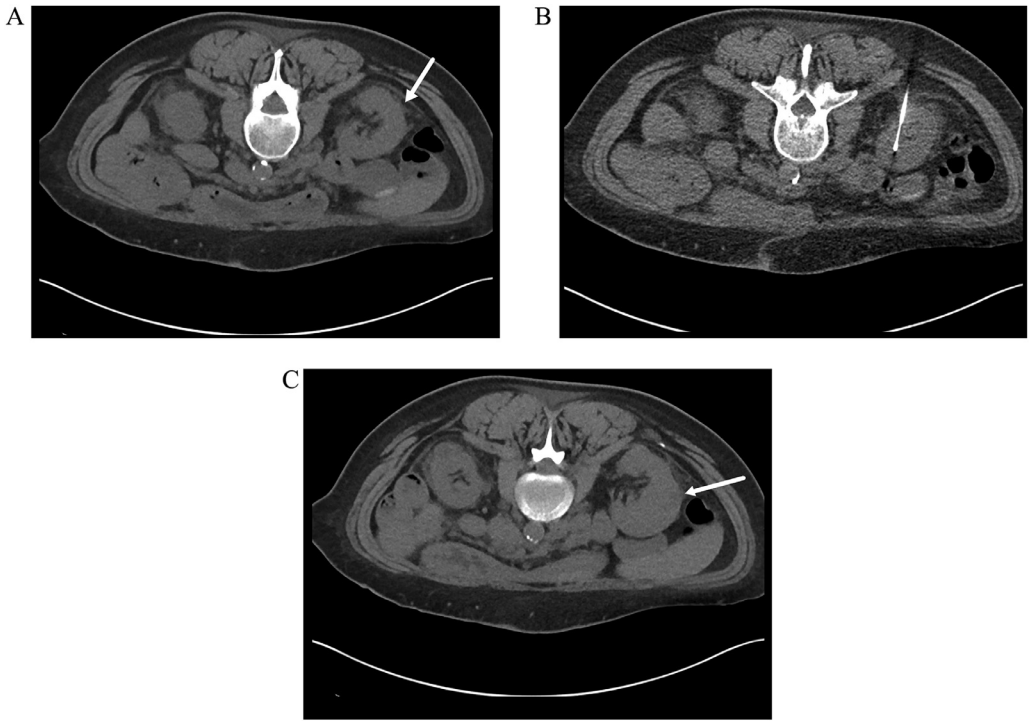

Fig. 1 – Preprocedural limited CT (A) demonstrating biopsy target with substantial peri-renal fibrosis (white arrow). Intra-procedural CT (B) demonstrating position of the biopsy needle. Post-biopsy CT (C) demonstrating new right subcapsular hematoma (white arrow).

Case report

A 58-year-old female with Stage III chronic kidney disease developed progressive renal dysfunction with nephrotic-range proteinuria. Her medical history included active tobacco use, poorly controlled Type-2 diabetes mellitus, and hypertension controlled with Lisinopril 5 mg and Amlodipine 2.5 mg daily. Antinuclear antibody titers, antineutrophil cytoplasmic antibodies, C3/C5 complement, anti-phospholipase A2 receptor antibodies, serum immuno-electrophoresis, and hepatitis B/C titers were negative. Her nephropathy was thought to be secondary to poorly controlled diabetes, however renal biopsy was indicated for tissue diagnosis. The patient presented for outpatient image-guided renal parenchymal biopsy to be performed by Interventional Radiology. On the day of biopsy, the patient's serum creatinine was 1.5 mg/dL and pre-procedure blood pressure was 136/63 mmHg. She received 24 mcg of desmopressin IV pre-procedurally, to reduce bleeding risk per institutional protocol.

On initial CT scan, significant fibrosis was noted in the perirenal fat surrounding both kidneys (Fig. 1A). After sterile skin preparation, IV moderate sedation, and 1% lidocaine local anesthesia, CT-guided placement of a coaxial 18G Temno biopsy needle (Merit Medical, South Jordan, UT) into the periphery of the right inferior renal pole was performed (Fig. 1B). The renal capsule was noted to be densely fibrotic, requiring extra effort to enter the renal parenchyma. Two core biopsy samples were obtained with the needle noted to deflect more superiorly toward renal sinus fat than intended. After biopsy, brisk bleeding was noted from the introducer needle. The tract was therefore embolized with absorbable gelatin sponge slurry through the introducer needle. Post-procedural CT demonstrated a new large subcapsular hematoma adjacent to the right kidney with expansion seen on repeat imaging 5 minutes later (Fig. 1C). Although the patient remained hemodynamically stable, the hematoma expansion prompted the decision to emergently transfer the patient for renal angiography and possible embolization. The patient subsequently underwent right renal artery angiogram via common femoral arterial access, which showed no arterial extravasation or other vascular abnormality, although there was substantial extrinsic compression of the right kidney (Fig. 2A). No intervention was performed. The arteriotomy was closed with a 6F Angioseal device (Terumo Medical, Somerset, NJ).

Post-procedural ultrasound showed an 8.1 cm x 6.0 cm x 2.8 cm subcapsular hematoma along the lateral border of the right kidney (Fig. 3A). Renal artery peak systolic velocity was elevated at 191 cm/s, with diastolic arterial flow reversal secondary to hematoma mass effect (Fig. 3B). On postoperative day one, the patient's hematocrit was unchanged from baseline. Her creatinine however increased from 1.8 to 2.2 mg/dL. She had new onset worsening of her hypertension, with systolic blood pressures of 148-189 mmHg. The patient was therefore diagnosed with Page kidney. Non-contrast abdominal CT demonstrated a persistent right renal nephrogram from prior angiography (Fig. 4). A multidisciplinary team involving Urology, Nephrology, and Interventional Radiology reviewed the patient's medical and procedural management

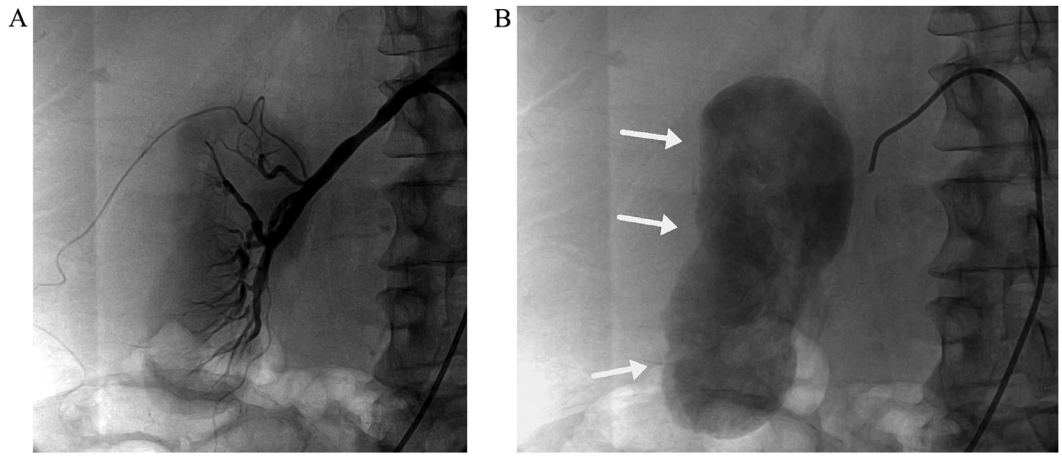

Fig. 2 – Arterial phase (A) and parenchymal phase (B) images of a right renal angiogram demonstrate no active arterial abnormality, but there is substantial extrinsic compression of the renal parenchyma by the subcapsular hematoma (white arrows).

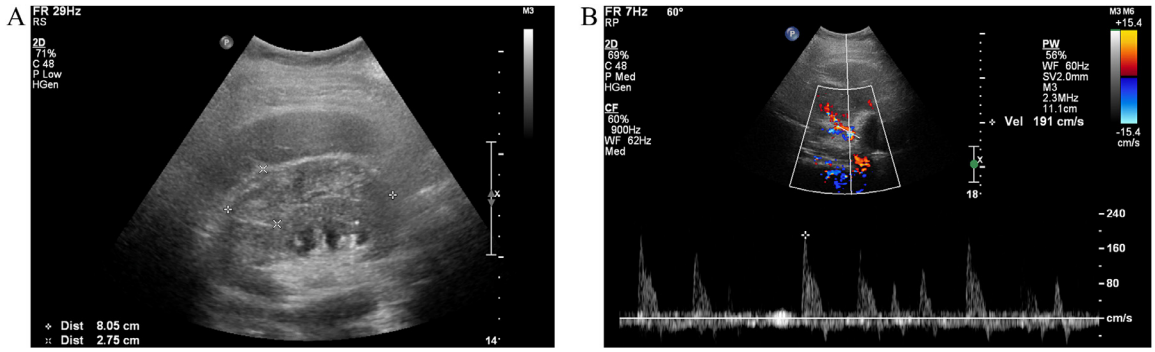

Fig. 3 – Grayscale (A) and Color/Spectral Doppler images (B) of the right kidney re-demonstrate the large right subcapsular hematoma, with elevated peak systolic velocity and reversed diastolic flow in the main renal artery.

options. Given stable hemodynamics and the risks associated with procedural management, conservative management was pursued with renal function monitoring and blood pressure control. The patient was started on amlodipine 10 mg and lisinopril 5 mg daily with hydralazine 50 mg as needed to maintain systolic blood pressure below 160 mmHg. Repeat ultrasound on postoperative day two demonstrated interval growth of the subcapsular hematoma, measuring 12.1 cm in largest dimension with persistent elevation of peak systolic renal artery velocities. From postoperative day two through five, the patient's serum creatinine rose to 3.6 mg/dL. With her serum creatinine stabilized and her hypertension controlled, the patient was discharged to home on postoperative day five.

On postoperative day seven, serum creatinine improved to 2.6 mg/dL. Three weeks after biopsy, the patient had a blood pressure of 119/65 mmHg. Thus, lisinopril was discontinued and she was maintained on amlodipine 2.5 mg daily. Six weeks following biopsy, she was having episodes of systolic blood pressures elevated to 172 mmHg requiring amlodipine titration to 10 mg daily with addition of losartan 50 mg daily and hydrochlorothiazide 12.5 mg daily. Eight weeks following her biopsy, the patient's creatinine level had returned to near baseline at 1.6 mg/dL and blood pressure was well-controlled.

Literature review

Etiology

Page kidney was first described as hypertension in young athletes following flank trauma during contact sports [1]. Blunt trauma may lead to the rupture of peripheral parenchymal vessels in the kidney, accumulation of blood in the narrow subcapsular space, and compression of the kidney from subcapsular hematoma. However, bleeding by any cause may lead to Page kidney. Indeed, iatrogenic causes are the most commonly reported following percutaneous and surgical renal interventions [2,3]. Rarely, spontaneous subcapsular hemorrhage can be seen with tumor hemorrhage, vascular malformations, or vasculitis [4]. Non-hemorrhagic extrinsic compression of the kidney can be seen with large renal cysts or other perinephric mass lesions [5].

Pathophysiology

The kidney is a solid organ surrounded by a fibrous capsule separating underlying parenchyma from perirenal fat. The

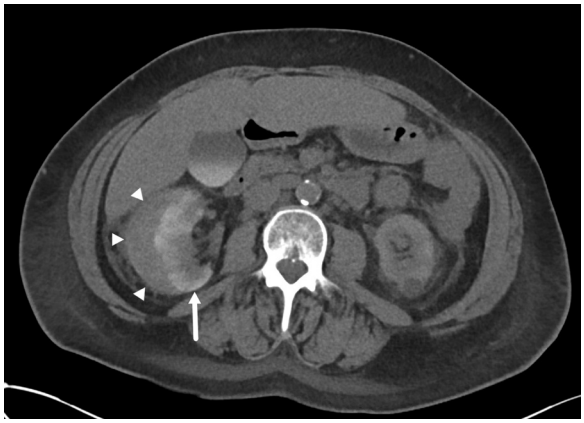

Fig. 4 – Non-contrast CT of the abdomen re-demonstrating the right renal subcapsular hematoma with associated persistent right renal nephrogram from prior angiography (white arrow). Also noted is the well-circumscribed subcapsular hematoma (white arrowheads).

subcapsular space between the capsule and the parenchyma is a potential space. Even small accumulations of fluid can cause substantial renal compression. In contrast, a large perinephric hematoma may accumulate within the perirenal space bounded by the perirenal fascia, without causing renal compression. In 1939, Dr Irvine Page first described the physiologic effects of renal compression in canines when he produced secondary hypertension after wrapping the kidney in cellophane, causing an inflammatory response leading to extrinsic renal compression [6]. Page pathophysiology develops when extrinsic compression of renal parenchyma is translated to the segmental arteries in the kidney. The juxtaglomerular apparatuses of the kidney sense decreased afferent blood flow, activating the renin-angiotensin-aldosterone system (RAAS) cascade. Increased renin release leads to increased conversion of angiotensin-II to angiotensin-I by angiotensin converting enzyme (ACE) [5]. Direct vasoconstrictive effects of angiotensin I and an upregulation of aldosterone release from the adrenal cortex result, leading to increased renal sodium reabsorption as a secondary means to increase systemic vascular resistance.

Management

Early in the history of Page kidney management, radical nephrectomy was often the definitive treatment of choice. In Page's original case report, the patient's hypertension resolved following radical nephrectomy [1]. With removal of the affected kidney, renin overproduction is eliminated, and systemic vascular resistance often returns to baseline. However, given the invasiveness of nephrectomy, management of Page kidney evolved into more conservative protocols with the advent of medications in the 1980s targeting the components of the RAAS pathway [7].

Medical management of Page kidney centers around pharmacologic manipulation of the RAAS cascade. Secondary hypertension results from the vasculature's response to angiotensin-I and the effect of aldosterone on the renal collecting system. Treatment with angiotensin converting enzyme inhibitors (ACEIs) or aldosterone receptor blockers (ARBs) is typically effective at counteracting renin's downstream effects. Caution must be exercised when using ACEIs in the setting of acute kidney injury, however, as they can precipitate further renal function decline [8].

Some patients fail medical management and require more invasive intervention, often when hematomas are organized, large, or actively expanding [9]. Surgical management may involve capsulotomy, hematoma evacuation, or nephrectomy. In a retrospective review of 26 cases of Page kidney, 17 patients were managed surgically with nephrectomy (n = 9), hematoma evacuation (n = 6), and capsulotomy (n = 4). All 17 patients demonstrated improved post-intervention glomerular filtration rate and hypertension [7].

Less invasive percutaneous maneuvers may also be considered in select patients. Successful management of Page kidney refractory to medical therapy using percutaneous image-guided drainage of acute subcapsular collections has been described [10]. Percutaneous drainage has been shown to have lower rates of major complications compared to surgical approaches, with avoiding general anesthesia and decreasing length of hospital stay. A retrospective review of 24 patients demonstrated 5 major complications in patients that underwent open surgical drainages procedures compared to no major complications in patients undergoing percutaneous image-guided drainage. Patients that underwent percutaneous drainage also demonstrated shorter duration of hospital admission compared to their surgical counterparts [11].

Discussion

Parenchymal bleeding is a recognized complication of percutaneous renal biopsy. The rate of clinically minor bleeding after percutaneous biopsy has been reported at 11%, with rates of blood transfusion and procedural intervention for major bleeding following renal biopsy reported at 1.6% and 0.3%, respectively [12]. The incidence of Page kidney pathophysiology after renal biopsy is rare.

Treatment goals in the Page kidney scenario emphasize normalization of systemic blood pressure and preservation of renal function. The primary strategy in this case was ACEI administration to mitigate RAAS-induced hypertension. The patient was started on a minimum lisinopril dose with simultaneous monitoring of blood pressure and serum creatinine, with adjuvant ARB treatment not required for management. Tight control of the patient's systolic blood pressure became especially important in this case: forward flow in the kidney with high systolic blood pressures could have led to further hematoma expansion, whereas substantial drops in perfusion pressure could have aggravated the patient's acute kidney injury. Typically, a rise in serum creatinine is not seen in Page kidney because of a compensatory increase in filtration by the unaffected contralateral kidney [13]. However, acute rise in this patient's serum creatinine revealed her left kidney's inability to compensate with glomerular filtration, a result of her chronic kidney disease.

Reversal of renal diastolic arterial flow is a rare phenomenon in native kidneys and has only recently been described as a possible diagnostic indicator of physiologically significant Page kidney [14]. Such flow reversal is typically found with renal vein thrombosis of a renal allograft. In native kidneys, this finding is less specific and less likely to represent renal vein thrombosis, due to rapid formation of capsular venous collaterals [14]. In a patient with appropriate clinical history, reversed diastolic arterial flow on spectral doppler imaging should prompt consideration of the diagnosis of Page kidney.

Non-invasive management of Page kidney can be a safe and effective treatment of this form of secondary hypertension, through the pharmacologic manipulation of the RAAS biochemical cascade that underlies this condition. More invasive surgical or percutaneous interventions should be reserved for specific clinical situations. However, there is no standardized management protocol for Page kidney. Multidisciplinary management with early involvement by Urology, Nephrology, and Interventional Radiology is ideal to optimize the outcomes of patients suffering from Page kidney.

Patient consent

Informed written consent from the patient was obtained for the publication of this case report. This study was exempt from institutional IRB approval.

Trial Registration

N/A.

Material Presented at Prior Meeting

N/A

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