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Abraam M. Rezkalla
St. Joseph's University Medical Center, 703 Main St, Paterson, NJ 07503, abraamrezkalla@gmail.com

Minha Naseer
St. Joseph's University Medical Center, 703 Main St, Paterson, NJ 07503

Adil Afzal
St. Joseph's University Medical Center, 703 Main St, Paterson, NJ 07503

Andrew Nashed
St. Joseph's University Medical Center, 703 Main St, Paterson, NJ 07503

Patrick Michael
1. St. Joseph's University Medical Center, 703 Main St, Paterson, NJ 07503

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CASE REPORT

A Rare Case of Intravenously Managed Hypertensive Emergency Arising from a Perirenal Hematoma Subsequent to a Native Kidney Biopsy

Abraam Rezkalla*, Minha Naseer, Adil Afzal, Andrew Nashed, Patrick Michael

St. Joseph's University Medical Center, 703 Main St, Paterson, NJ 07503, USA

Abstract

Page kidney is a pathologic and rare occurrence caused by a compression of renal parenchyma leading to hypertension. When infiltrated or engulfed by extrinsic matter, the subcapsular region surrounding the renal tissue may cause blanket compression, leading to the activation of the renin-angiotensin-aldosterone system secondary to renal hypoperfusion. While most cases of Page kidney are secondary to blunt trauma to the costovertebral angle, herein we present a case of Page kidney due to renal parenchymal core needle biopsy. The rarity of our case is not due to the cause of such an incidence but because our case resulted in a hypertensive emergency treated with dual intravenous infusions.

Keywords: Page kidney, Acute kidney injury, Hypertensive emergency

1. Introduction

Page kidney or Page phenomenon, first described by Dr. Irvine Page in 1939,[1] is a rare treatable cause of secondary hypertension. It is attributed to extrinsic compression of renal parenchyma by subcapsular hematoma or other sources leading to alteration of small segmental vessel's hemodynamics.[2,3] The etiologies of Page kidney can be traumatic, nontraumatic, or idiopathic.[3] Traumatic etiologies include blunt abdominal injuries or iatrogenic trauma from procedures like renal biopsy, extracorporeal shock wave lithotripsy, and ureteral-laparoscopic procedures.[3] Nontraumatic etiologies include spontaneous renal bleeding as a complication of anticoagulation, underlying tumor, cyst rupture, glomerulonephritis, vasculitis, or from arteriovenous malformations.[3] Most reported cases of acute Page Kidney in the literature presented as secondary hypertension requiring treatment of underlying cause and oral antihypertensive agents. Our case is unique because the patient presented as a hypertensive emergency after undergoing renal biopsy requiring critical care admission and treatment with intravenous infusions of antihypertensive medications.

2. Case presentation

A 23-year-old African American female with a past medical history of Chronic Kidney Disease stage 3b secondary to uncontrolled hypertension presented to the Emergency Department (ED) with complaints of left-sided flank pain lasting one day. She described the pain as sharp in nature and severity. The patient also described a rapid onset of diaphoresis and dizziness which lasted until her arrival at the Emergency Department. Of note, the patient had undergone a renal biopsy at an external institution three days prior to the onset of symptoms. During this time, the patient complained of improving tenderness at the site of biopsy, until the day of presentation.

Through further history, the patient reported working as a retail store manager for many years. She participates in daily marijuana use however no tobacco products. She also drinks alcohol occasionally.

3. Investigation

On examination, she was afebrile, with a blood pressure of 296/160 mmHg, heart rate of 84 beats per minute, respiratory rate of 20 breaths per minute, and was saturating 100% of oxygen on room air. Her

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* Corresponding author.
E-mail address: Abraamrezkalla@gmail.com (A. Rezkalla).

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physical exam was significant for acute severe distress. The patient's breathing was unlabored, and her abdominal exam was significant for tenderness, particularly in her left flank. There was no suprapubic tenderness present. Laboratory studies revealed an elevated Blood Urea Nitrogen (BUN) level (35 mg/dL) and Creatinine level (2.47 mg/dL). Baseline kidney function was unknown. The patient's estimated Glomerular Filtration Rate of an African American was significantly decreased (29 mL/min/1.73mˆ2). She also had leukocytosis (17.2 × 10ˆ3/mm3) without left shift or bandemia and a hemoglobin level on presentation of 11.1 g/dL decreasing as low as 6.7 g/dL within two days [Table 1]. A CT abdomen and pelvis without intravenous contrast was performed, suggesting the right kidney was replaced by a hematoma, measuring 11 × 11 × 20 cm. The left kidney was within the center of this hemorrhage with mass effect displacing the pancreas anteriorly. Free intraperitoneal hemorrhage was also noted in the left flank and retroperitoneum (Figs. 1 and 2). The patient was admitted for suspected Page Kidney Phenomenon due to significant compression of the kidney from the hematoma causing a hypertensive crisis.

The patient was treated with continuous Esmolol at a rate of 50/mcg/kg/min and within hours, an additive with continuous Nicardipine at a rate of 15 mg/h. She also received 1 unit of packed Red Blood cells given the significant drop in hemoglobin. The patient was taken for diagnostic angiography of the abdominal aorta, left renal artery, and distal branches which demonstrated active bleeding from distal branches of the left renal artery. The bleeding vessel was successfully embolized with detachable coils and a percutaneous drain was placed. The patient was admitted into the Medical Intensive Care Unit and transitioned to a continuous Labetalol infusion at a rate of 22.5 mL/h. The patient remained in the MICU until blood pressure improved to 144/90 mmHg and was transitioned to oral Labetalol 200 mg daily and oral Nifedipine 90 mg Extended Release daily. The patient underwent secondary hypertension workup, such as an aldosterone renin ratio, which was within normal limits. Moreover, the final diagnosis of Reflex hypertensive crisis secondary to renal parenchymal compression secondary to Page Phenomena was made. The patient was discharged under the care of Nephrology with proper follow-up.

Table 1. Admission laboratory values.

Test Name	Results	Reference Ranges
Sodium	139 mEq/L	135–145
Potassium	3.9 mEq/L	3–5
Magnesium	2.0 mEq/L	1.7–2.5
BUN	35 mg/dL	7–23
Creatinine	2.47 mg/dL	0.6–1.3
WBC	17.3 K cells/μL	4.5–11.0 K cells/μL
Hgb	11.1 g/dL	12.0–16.0 g/dL
Hct	33.5%	41–50%
Platelets	241K cells/μL	50–450K cell/μL
Prothrombin time	14.3 s	12.2–14.9
INR	1.1	
PTT	33.8 s	21.3–35.1
Fibrinogen	448 mg/dL	183–503
Renin	4.7	0.167–5.380
Aldosterone:renin	1	0–30

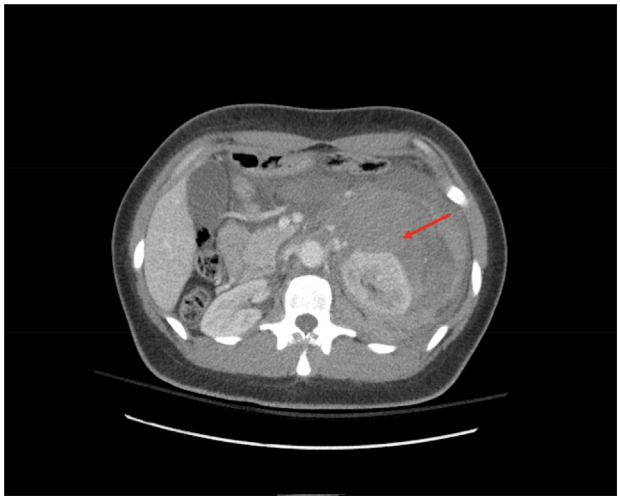

Fig. 1. The left kidney is replaced by a hematoma, measuring 11 × 11 × 20 cm. The left kidney is within the center of this hemorrhage demonstrating a large left perirenal hematoma. There is a mass effect, displacing the pancreas anteriorly. Free intraperitoneal hemorrhage is also noted in the left flank and retroperitoneum. The right kidney is normal.

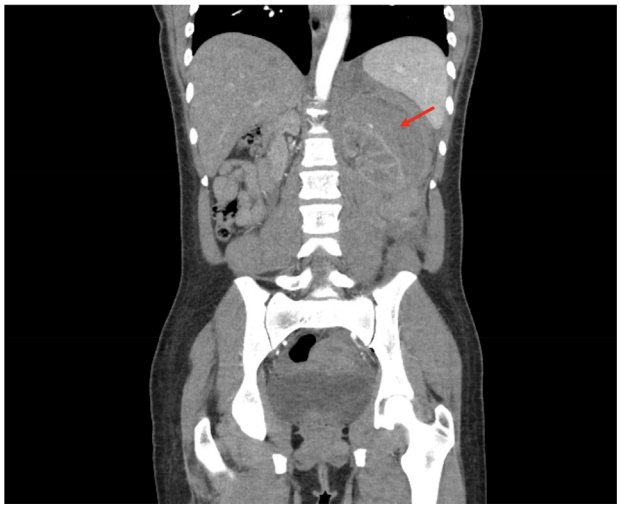

Fig. 2. The left kidney is replaced by a hematoma, measuring 11 × 11 × 20 cm. The left kidney is within the center of this hemorrhage demonstrating a large left perirenal hematoma. There is a mass effect, displacing the pancreas anteriorly. Free intraperitoneal hemorrhage is also noted in the left flank and retroperitoneum. The right kidney is normal.

4. Discussion

In 1939, Dr. Irvine Page first described the effects of renal compression leading to the induction of secondary hypertension after wrapping the kidney in cellophane, causing an inflammatory response leading to external renal compression.[1]

The kidney is a solid organ surrounded by a fibrous capsule that separates the underlying parenchyma from perirenal fat. The subcapsular area is a potential space where even small accumulations of fluid can cause renal compression.[2] The phenomenon of page kidney develops when a sustained extrinsic compression of renal parenchyma alters small segmental vessel hemodynamics resulting in the activation of the Renin-angiotensin-aldosterone system (RAAS) cascade.[2,3] This leads to increased direct vasoconstrictive effects of angiotensin I and upregulation of aldosterone release, leading to increased renal sodium reabsorption with a rise in systemic blood pressure.[2,3]

Typically, an acute rise of creatinine is not common in patients with Page Kidney due to compensation by the normal kidney[2] but our patient presented with a questionable Acute Kidney Injury on Chronic Kidney Disease, which can be explained by the acuity of the event and a component of chronic kidney disease.

Page kidney can be caused by traumatic and nontraumatic etiologies.[3] The most common underlying mechanism includes the traumatic causes leading to subcapsular hematoma. Previously mentioned traumatic causes mostly include contact and non-sport-related blunted abdominal injury.[3] After 1991, there have been case reports stating that Page kidney can also be caused by iatrogenic trauma following procedures, including renal biopsies and post-extracorporeal shock wave lithotripsy or uretero-nephroscopic procedures.[3]

Nontraumatic causes are relatively uncommon and include spontaneous renal hemorrhage secondary to underlying tumors, arteriovenous malformations, cyst rupture, glomerulonephritis, or vasculitis. In some cases, Page kidney can be idiopathic as well.[3]

Acute page kidney post-native renal biopsy is a rare condition and it is of high significance to further investigate patients presenting with acute flank pain, acute changes in blood pressure, and reduction in urine output following renal biopsy.[4] It is also important to note that these patients can have a significant delay in presentation post-trauma, ranging between days and decades having been reported.[3]

Our patient presented with a hypertensive emergency following a native kidney biopsy. The hypertensive emergency was severe enough to require critical care management, resistant to single antihypertensive medication in the form of continuous infusion and acute kidney injury. Previously very few cases have been reported stating that Page kidney can present as a hypertensive emergency that is systolic blood pressure >220 and diastolic blood pressure >110 accompanied by acute organ failure, resistant to single drug therapy.

Previously, management of Page Kidney included nephrectomy but with medical improvement, antihypertensives that particularly target the renin aldosterone angiotensin system (RAAS) are used.[3,5] Most preferred antihypertensive agents include enalapril or lisinopril, a drug that blocks the renin aldosterone angiotensin system, and nicardipine, a calcium channel blocker, to control hypertension.[5] However, there is currently no standardization of optimal antihypertensive regimen and how to approach patients presenting with this condition; management mostly depends on the underlying cause of Page Kidney and the patient's clinical condition. Patients with subcapsular hematomas usually undergo radiological or laparoscopic guided percutaneous drainage.[3,5] In some cases of page kidney, patients may require chronic antihypertensive management, regardless of resolution.

Our patient was initially admitted to the intensive care unit and initiated on two anti-hypertensive continuous infusions to control the hypertension. She ultimately underwent embolization and radiology-guided percutaneous drain insertion and was discharged with oral antihypertensives including Labetalol and nifedipine.

5. Conclusion

Hypertensive emergency is a rare presentation of a traumatic Page kidney which may be unresponsive to oral medication or single-drug therapy. In these cases, prompt diagnosis and surgical intervention are warranted to control the hypertensive emergency; this may decrease the rate of persistent hypertension and complications of hypertensive emergency. Early recognition of the page kidney is also of high importance, to prevent any ischemic changes to the kidney and preserve its function.

Author contribution

Abraam Rezkalla and Minha Naseer are the article guarantors. Abraam Rezkalla, Minha Naseer, Adil Afzal and Andrew Nashed performed the literature review and wrote the manuscript. All authors assisted in the collection of the patient's

clinical data. All authors took part in the medical management of the patient and edited the final manuscript for submission. All work was performed at St. Joseph's University Medical Center at the following address:

St. Joseph's University Medical Center
Department of Internal Medicine
703 Main Street
Paterson, NJ USA 07503
(973) 754-2000

Consent

As this is a case report, consent was obtained for the purpose of this paper.

Conflict of interest

The authors report no conflict of interest. Ethical review is not necessary, because this is a case report. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

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