CASE REPORT
Jurnal Penyakit Dalam Udayana
Udayana Journal of Internal Medicine
Volume 8, No. 2: 2024; 51-54

Print ISSN: 2580-2925
Online ISSN: 2580-2933

The page kidney phenomenon: Rarely found causes of secondary hypertension *subcapsular hematoma renal* in post-trauma cases

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DOI : 10.36216/jpd.v8i2.251

Page Kidney Phenomenon is a rare condition caused by extra-renal processes that cause the kidney parenchyma to compress, thus activating the renin-angiotensin-aldosterone system (RAAS). The manifestation that occurs is secondary hypertension. The etiology of Page Kidney can be caused by trauma and non-trauma. Initial management to treat secondary hypertension can be divided between antihypertensive treatment and combined with surgical approaches to drainage of hematoma subcapsular and in extreme cases nephrectomy total. An 18-year-old man was taken to the emergency room after a motorbike accident. The patient's blood pressure continued to increase to 178/91 mmHg. Abdominal ultrasonography revealed a subcapsular hematoma of the right kidney measuring 0.9 x 2.9 x 4.6 cm. The patient was diagnosed with Page Kidney. This patient was given antihypertensive therapy with a single pill combination (SPC) which is a combination of valsartan 80 mg and amlodipine 5 mg 1 x 1, with this antihypertensive therapy, secondary hypertension is controlled.

Keywords: *Secondary hypertension, Page Kidney, subcapsular hematoma, renin angiotensin system, post trauma*

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Received : 03 April 2024
Accepted : 05 June 2024
Published : 10 August 2024

INTRODUCTION

Page Kidney phenomenon is a rare condition of secondary hypertension caused by extra-renal processes that cause the kidney parenchyma to be compressed, causing hypoperfusion and then developing ischemia. After that, the renin-angiotensin-aldosterone system (RAAS) will be activated.[1] Page Kidney was introduced by Goldblatt et al. in 1934, who experimented with inducing hypertension in dogs by narrowing the main artery in the kidney with clamps and causing ischemia in the kidney. This may indicate that the release of renin can cause hypertension. If the narrowing process is severe enough and affects only one kidney, hypertension develops without a decrease in kidney function. This is known as Goldblatt's Kidney.[2-4]

In 1939 Dr. Irvin Page induced hypertension in animals by wrapping the kidneys in cellophane. Within a few days, this causes tissue formation that narrows the renal parenchyma, thereby pressing on the parenchymal blood vessels instead of the main renal arteries and causing ischemia and renovascular hypertension.[2] When one or both kidneys go through persistent compression, hypertension can occur. Page also noted that the removal of the compressed kidney can eliminate hypertension. Induction of hypertension through compression of the kidney is known as Page kidney.[5-8] Page Kidney is typically diagnosed in young men who present with previously unexplained high blood pressure, a history of trauma, and radiological findings that show a subcapsular hematoma.

Below, we report a case of an 18-year-old male with Page Kidney due to post-traumatic subcapsular hematoma. We took this case because it is a very rare case, and to be able to learn how to make a diagnosis and carry out appropriate management.

CASE ILLUSTRATION

An 18-year-old male patient experienced a loss of consciousness after a motorbike accident. Vital signs were checked and a physical examination revealed BP when in the emergency room 153/100 mmHg, HR 121 x/m RR 22 x/m S 36 C SpO2 96% RA. On physical examination, there was a right frontal cephalhematoma, extensive lacerations on the upper and lower lips down to the gums, and several abrasions on the hands and feet. Supporting examinations were carried out in the form of a non-contrast head CT scan, which revealed a skull base fracture, and subarachnoid hemorrhage in the brain, and a thorax x-ray examination revealed a lung contusion. A complete blood test revealed

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hemoglobin 13 g/dl, hematocrit 38.1%, erythrocytes 4.4 10^{5}/ul, PlateletS 230,000 10^{3}/ul, Leukocytes 21,800 10^{3}/ul, Neutrophils 78% Lymphocytes 12% Bleeding Time 2" Clotting time 5", Current Blood Glucose 101 mg/dl, Urea 35.70 mg/dl, Creatinine 1.27 mg/dl, Sodium 140 mmol/L, Potassium 4.6 mmol/L, Chloride mmol/L, SGOT 170 U/L, SGPT 403 U/L. The upper and lower lip wounds were then debrided in the operating room, and the patient was subsequently treated in the intensive care unit (ICU) following the procedure.

During treatment, the patient's blood pressure showed an increase to 178/91 mmHg (**Figure 1**). It was previously reported that the patient had no history of high blood pressure. Head CT scan imaging and ICP monitoring showed no signs of increased intracranial pressure or signs of herniation. The patient complained of sudden severe right abdominal pain and this patient underwent further examination in the form of abdominal ultrasonography and found a subcapsular hematoma of the right kidney measuring 0.9 x 2.9 x 4.6 cm (**Figure 2** and **Figure 3**). The left kidney is normal. There is no decrease in GFR. The patient also underwent urinalysis and found Blood +2, Erythrocytes 7-9, and Bacteria+. This patient was given a single pill combination (SPC) in the form of a combination of CCB (Calcium Channel Blocker) and ARB (Angiotensin II Receptor Blocker) antihypertensives (amlodipine 5 mg + valsartan 80 mg) 1 x 1, the patient was treated for 3 weeks with antihypertensive therapy, secondary hypertension was controlled. At the first control at the polyclinic after hospitalization, the patient's blood pressure ranged from systolic 120-110 to diastolic 80-70 mmHg.

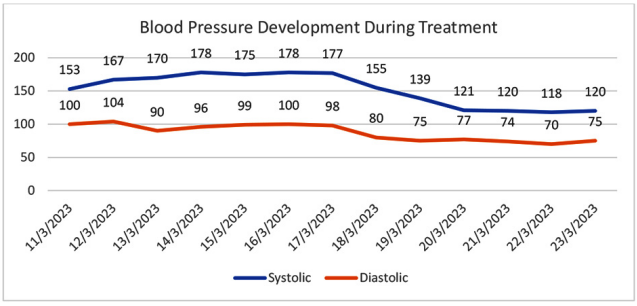

Figure 1. Blood Pressure Development During Treatment

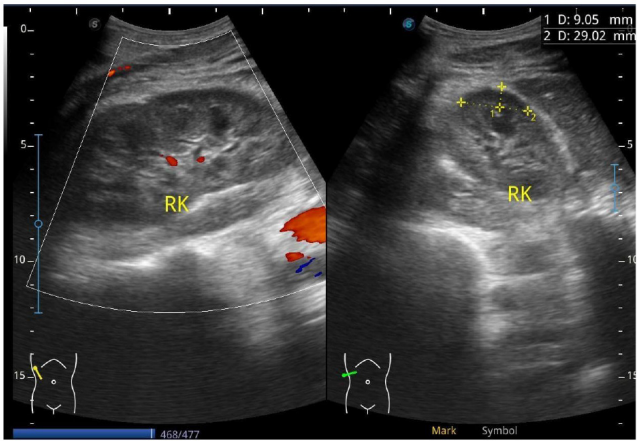

Figure 2. A sonographic picture of the right renal subcapsular hematoma 0.9 x 2.9 x 4.6 cm shows a fluid-filled mass pressing on the renal parenchyma

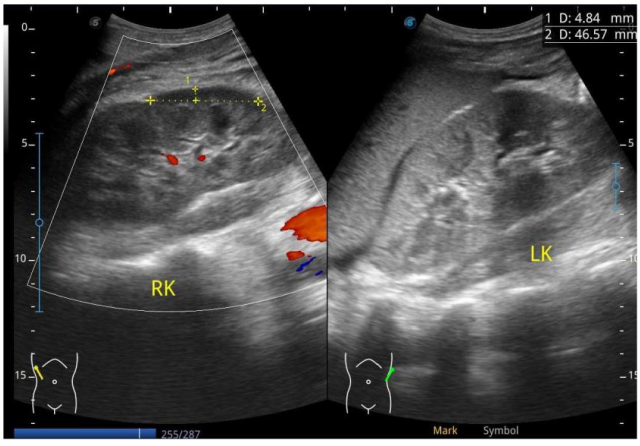

Figure 3. Subcapsular hematoma of the right kidney and normal left kidney

DISCUSSION

The Page Kidney phenomenon is rare, with only 20 cases reported in the United States from 1991-2008.[4,6] In Indonesia itself, there have been no reported cases of Page Kidney. Page Kidney phenomenon is most often caused by blunt abdominal trauma, it can also be due to surgical complications, spontaneous bleeding, and not from bleeding such as malignancy.[1,7,9,10] Page Kidney occurs due to the anatomy and structure of the kidneys that surround it. The kidneys are located retroperitoneally and have limited ability to withstand shocks. In the kidneys, there are 2 layers covering the renal parenchyma which, if filled with fluid, can cause compression in the kidneys. The first layer is the Gerota fascia, a layer of fat as a barrier if there is a shock to the kidneys. A large hematoma can cause compression of the renal parenchyma resulting in ischemic hypertension. The second layer is a narrow potential space in the form of a capsule. If there is fluid, it will cause compression on the kidneys and cause the renin-angiotensin-aldosterone system (RAAS) to become active, giving rise to secondary hypertension. This mechanism is supported by Goldblatt's research on Page Kidney.[1,4]

Goldblatt and Page demonstrated that when the renal arteries, including both major arteries and the microvasculature, are compressed, it leads to reduced blood flow to the kidneys, triggering the release of renin into the bloodstream. This results in the conversion of Angiotensin I to Angiotensin II and stimulates the adrenal glands to secrete Aldosterone. This is known as the renin-angiotensin-aldosterone system (RAAS). Activation of this system causes vasoconstriction of blood vessels and increased sodium and water reabsorption by the renal tubules into the blood,

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resulting in increased blood pressure and renin-dependent hypertension. In-Page Kidney, there is a narrowing of the renal parenchymal arteries. If compression occurs in both kidneys or one kidney but the contralateral kidney is not functioning then referring to the Goldblatt II model there will be a decrease in renin, but there will be an increase in intrarenal Angiotensin II so that in this model non-dependent renin hypertension will arise.[11,12]

In the renal parenchyma, there must be compression to cause ischemia. Subcapsular hematomas, despite their small size, are more likely to lead to renal compression and ischemia because they are contained, whereas extracapsular bleeding can spread into the peritoneal space without exerting pressure on the kidneys.[11-13]

To diagnose Page Kidney, it's essential to conduct a detailed patient history to identify any past abdominal trauma. Additionally, imaging studies are necessary to detect a fibrous capsule or hematoma exerting pressure on the kidney. Sonography and abdominal CT scans are commonly utilized to identify subcapsular hematomas, but sonography offers the added advantage of evaluating renal vascular perfusion. A capsular hematoma can be identified by the absence of blood flow on Doppler imaging. Sonographic assessment offers benefits such as lower costs and the absence of ionizing radiation. However, its effectiveness can be limited by the operator's experience. In contrast, CT scans provide superior resolution for visualizing minor subcapsular and retroperitoneal hematomas, particularly in cases of perinephric hematoma. Additionally, MRI scans have the advantage of assessing the age of the hematoma.[1,14]

Treatment aims to relieve or eliminate kidney compression, restore kidney perfusion to normal, and return to a normotensive state. At Page Kidney, there is no standard treatment but the tendency is to use non-invasive treatment. Oral medications that block the renin-angiotensin-aldosterone system can be effective in managing the condition, giving the hematoma time to heal on its own. This approach allows for natural resolution of the hematoma.[15,16] Treatment options for Page Kidney involve surgical procedures such as capsulectomy, hematoma removal, and radical nephrectomy. The effectiveness of these treatments seems to be affected by the age of the subcapsular hematoma, with more aggressive approaches needed as the hematoma advances.[11,17,18]

Apart from being caused by trauma, Page kidney can also be caused by non-trauma such as kidney tumors, ruptured kidney cysts, vasculitis, and various interventional procedures. Most trauma cases are characterized by lower systolic blood pressure compared to causes unrelated to trauma.[10] In this patient, hypertension is characterized by high systolic blood pressure (systolic blood pressure above 170 mmHg) which is responsive to the administration of two antihypertensive therapies.

In this case, it was found that a young patient aged 18 years had hypertension (blood pressure 178/91 mmHg) during treatment in the ICU after a motorbike accident. It was previously stated that the patient had no history of hypertension. Ultrasonography or CT scan with contrast can generally confirm the diagnosis of Page Kidney. In this patient, an abdominal ultrasound was performed and a subcapsular hematoma of the right kidney was obtained measuring 0.9 x 2.9 x 4.6 cm. The left kidney is normal. There is no decrease in GFR. Kidney function is generally normal if the kidney compression is unilateral because the normal contralateral kidney can compensate. Kidney function can be impaired if kidney compression is bilateral.

The patient was given a single pill combination (SPC) in the form of a combination of CCB (Calcium Channel Blocker) and ARB (Angiotensin II Receptor Blocker) antihypertensives (amlodipine 5 mg + valsartan 80 mg) 1 x 1, with this antihypertensive therapy, the patient's blood pressure be controlled. Tight control of the patient's systolic blood pressure is very important in this case because if the systolic blood pressure is too high it can cause further expansion of the hematoma while an extreme decrease in blood pressure can worsen acute kidney injury. Non-invasive management of Page Kidney cases can be a safe and effective treatment for secondary hypertension based on RAAS activation. Interventions such as percutaneous drainage, capsulotomy, and nephrectomy are carried out if a poor clinical response is found to prevent persistent hypertension in the future.

CONCLUSION

Page Kidney phenomenon was diagnosed in an 18-year-old male who experienced secondary hypertension with subcapsular hematoma of the right kidney after a motorbike accident. This case is very rare. It is hoped that an appropriate therapeutic approach can prevent persistent secondary hypertension. Page Kidney caused by trauma can cause very high blood pressure so you can immediately make a diagnosis and provide appropriate therapy. This patient was given conservative therapy with antihypertensive drugs and the patient's blood pressure was controlled.

CONFLICT OF INTEREST

The authors have no conflict of interest in the preparation of this case report.

ETHICAL CONSIDERATION

This case report has obtained informed consent from the patient and their family and has followed the COPE guidelines on ethics in scientific publication.

FUNDING

The preparation of this case report did not involve any sponsorship or other funding sources.

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AUTHOR CONTRIBUTIONS

All authors contributed to the writing of this manuscript, starting from the patient examination, data collection, and the writing of the report.

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