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Case report

A case report of post-surgical page kidney due to extensive renal hematoma following percutaneous nephrolithotomy

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ABSTRACT

Introduction and importance: Post-surgical Page kidney due to large renal hematoma following percutaneous nephrolithotomy (PCNL) is a rare significant complication that may lead to loss of a kidney.
Case presentation: A 50-year-old lady underwent elective left side PCNL for a 3 cm renal pelvis stone, and one week later, she presented back with a massive renal hematoma with high blood pressure.
Clinical discussion: The ultrasound abdomen and computed tomography diagnosed a page kidney due to massive intrarenal and perirenal hematoma as a complication of PCNL. Angioembolization and percutaneous aspiration were failed, and the antihypertensives also failed to control the blood pressure. Therefore, she underwent a left-side simple nephrectomy and had an uneventful recovery with reversal of normal blood pressure.
Conclusion: Post-surgical page kidney needs to identify early to facilitate the percutaneous radiological interventions that may preserve the renal parenchyma and avoid further surgeries. Nevertheless, late cases or the failed radiologically intervened cases need open renal exploration and simple nephrectomy, which may be the bailed-out procedure to reverse the consequence of page kidney.

1. Introduction

Percutaneous nephrolithotomy (PCNL) is one of the standard treatment options for patients with renal stones. In this procedure, perioperative bleeding complications are relatively commoner (11%–30%), including intrarenal or perirenal hematoma and gross hematuria [1]. The massive intrarenal and perirenal hematoma are rare following PCNL, and they lead to compression of renal parenchyma and renal arteries and secondarily causing systemic hypertension, known as page kidney [2]. This paper aimed to present the identification, prevention, and management of post-surgical Page kidneys.

This case report has been written according to the SCARE 2020 guideline [3].

2. Case history

A 50-year-old, previously healthy lady underwent elective left side prone percutaneous nephrolithotomy via single lower pole puncture (30Fr) for a 3 cm renal pelvis stone. An experienced urologist did the procedure, and she did not have significant intraoperative or immediate postoperative bleeding complications. Therefore, the ureteric catheter and nephrostomy tubes were removed in 24 h, and she was discharged on postoperative day two. She was admitted with left side abdominal pain, distension, fever, and reduced urine output one week later. She was febrile, pale, profound hypertensive (190/110 mmHg), and palpable left side abdominal ballotable mass on examination. Unfortunately, she did not have any gross hematuria on discharge or during readmission. She does not take any anticoagulation or antiplatelets and is not known for other bleeding disorders or hypertension before this surgery.

The white cell count and C-reactive protein levels were elevated in serum 22.0 $\times$ 10^9/L and 350 mg/L, respectively. Further, the serum creatinine level raised to 212 μmol/L, and hemoglobin level also dropped to 5.5 g/dl, which were standard in limit while on discharge.

The ultrasound kidney ureter bladder (KUB) showed an extensive renal hematoma on the left side kidney with a compressive effect on the kidney. Later, she underwent contrast-enhanced computed tomography (CECT) abdomen after dropping of serum creatinine to the upperlimit of

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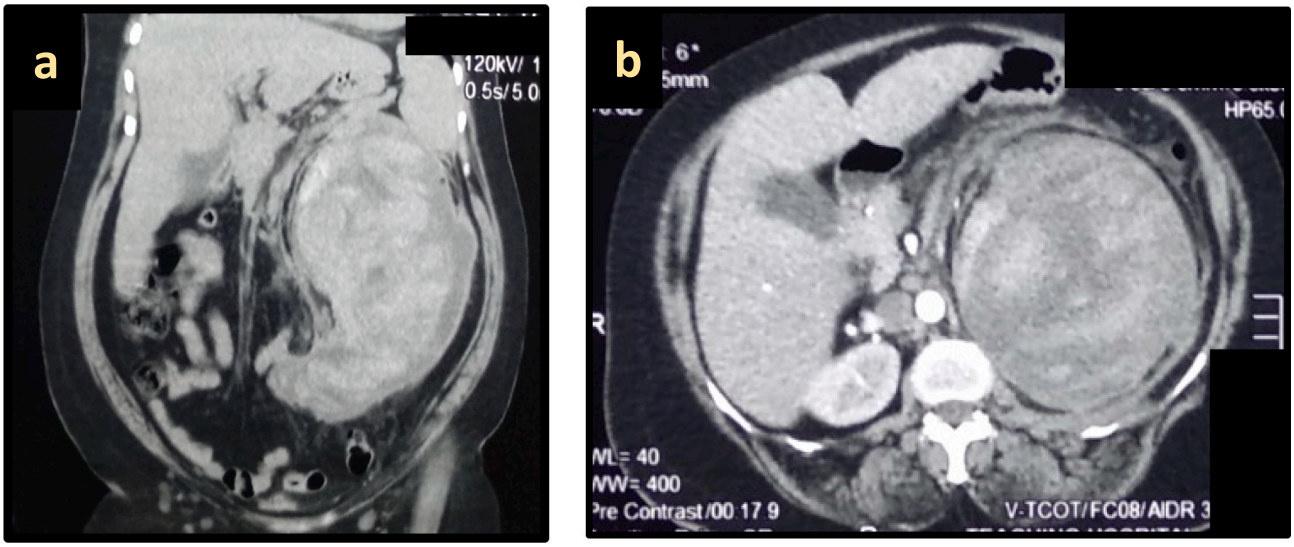

Fig. 1. a: Coronal section CT abdomen shows the left side large intrarenal and perirenal hematoma with vertical extension up to the pelvic brim.
b: The axial section reveals large renal hematoma (intrarenal and perinephric hematoma) 20 × 27 cm with distorted renal parenchyma, and it is confined to the Gerotas fascia.

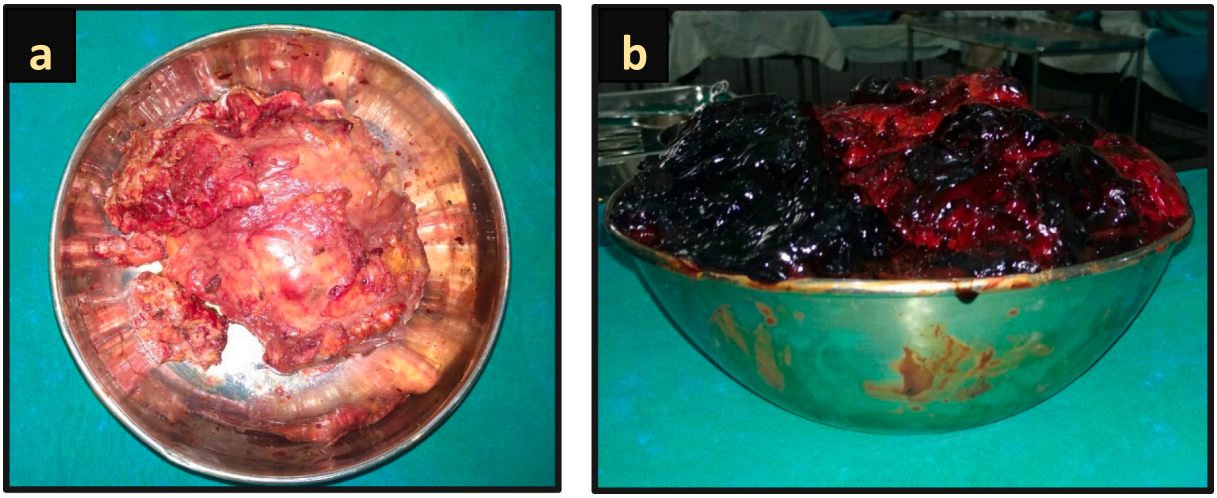

Fig. 2. a: This image shows the macroscopic appearance of nephrectomy with Gerotas fascia. b Shows the clots removed after dividing the Gerotas fascia during surgery to facilitate the dissection.

baseline, and it revealed a large perinephric and intrarenal hematoma (20 cm × 27 cm) with compressed and distorted renal parenchyma (Fig. 1). Although the initial angiogram did not reveal an obvious bleeder, therapeutic lower segmental angioembolization was done.

Despite embolization, hemoglobin levels continued to drop, needing repeated blood transfusions attributed to consumption coagulopathy. Urosepsis was managed with intravenous meropenem leading to recovery from both sepsis and acute kidney injury. However, blood pressure remained persistently high despite a total dose of oral angiotensin-converting enzyme (ACE) inhibitors. The patient was also frustrated due to a lengthy hospital stay and multiple procedures. A subsequent Diethylenetriaminepentaacetic acid (DTPA) scan revealed a non-functioning kidney, at which point it was decided to go ahead with exploration and nephrectomy after the multidisciplinary meeting and family counseling. The surgery was performed via left subcostal incision by a senior consultant urologist and two specialist urology registrars, and a large amount of perinephric hematoma was evacuated. Much fibrotic adhesion was noted around the kidney, and finally, a simple nephrectomy was done (Fig. 2) with two pints of blood transfusion.

The postoperative period was uneventful, and her blood pressure returned to normal with one ACE inhibitor from postoperative day two onwards. She was discharged on postoperative day ten and reviewed at the clinic with a histology report that revealed the large hematoma with inflammatory changes in renal parenchyma.

3. Discussion

Page kidney is caused by the accumulation of blood in the perinephric or subcapsular space, resulting in compression of the involved kidney, renal ischemia, and high renin hypertension [2]. The causes of mass effects can be hematomas, urinomas, tumors, cysts, lymphoceles, and aneurysms [1]. Irving Page described in a study regarding the induction of hypertension in dogs by wrapping one or both kidneys by cellophane in 1939, followed by Engel and Page in 1955 reported the first clinical scenario of page kidney due to renal subcapsular hematoma in a football player who had sustained blunt trauma to the flank [4].

Perioperative bleeding following PCNL is one of the rare causes for the page kidney, and the post PCNL bleeding rate depends on the

technique of renal puncture, tract caliber, stone size, and procedure time [1]. One study found that there was significantly more bleeding (9.4% vs. 6.7%, respectively) and more transfusions (7.0% vs. 4.9%, respectively) in balloon dilatation compared with the telescopic/serial dilator group in the PCNL Global Study [5]. Also, Ketsuwan et al. revealed a blood transfusion rate of 9.29% among 226 patients who underwent PCNL for renal stones due to perioperative bleeding [6].

However, the occurrence of Page kidney is directly rated to the anatomy of the kidney. A large hematoma within the perinephric space or a small subcapsular hematoma could compress the parenchyma and compromise the intrarenal blood flow. Subsequently, this triggers the activation of the renin-angiotensin-aldosterone axis leading to systemic hypertension [2].

Hypertension is the main clinical presentation of the Page kidney, with refractory hypertension immediately after the causative event [2]. Patients may also present with features of infected hematoma or features of disseminated intravascular coagulopathy (DIC). In most cases, the global renal function is normal, except in compression to the single functioning kidney, transplanted kidney, global renal function will be compensated by the contralateral kidney.

Angiogram may be beneficial in the acute onset of page kidney to visualizes the bleeding artery and embolization [7]. Renal arterial Doppler studies can demonstrate the reduction of blood flow within the renal artery and renal parenchyma. The subcapsular hematoma will show a lack of blood flow by color Doppler imaging and spectral Doppler. Renal vein renin determination, which is not frequently done, can prove activation of the renin-angiotensin mechanism as the causative mechanism of hypertension in doubtful cases [2]. Only Isotope studies with split renal function would confirm severe functional deterioration of the affected kidney with the Page phenomenon.

The goal of treatment is to relieve compression of the kidney, restore normal perfusion, and return the patient to a normotensive state. Treatment options include medical management, percutaneous drainage of hematomas, surgical drainage via capsulotomy or capsulectomy, and even nephrectomies.

Small hematoma with hypertension can be treated with angiotensin-converting enzyme (ACE) inhibitors for a short period without drainage and observing hematoma resolution with serial imaging [4]. Exceptionally large subcapsular hematomas are probably less likely to spontaneously remit and form a fibrous capsule with a threat of permanent loss of renal function requiring some form of surgical drainage, which could be laparoscopic or open surgical decompression with capsulectomy. A nephrectomy would be the final solution if the hematoma is massive, as found in the present case, and hypertension continues with non-viable parenchyma.

4. Conclusion

Post-surgical page kidney is one rare but significant bleeding complication of PCNL surgeries even though PCNL is a minimally invasive treatment. Therefore, this case scenario gives some learning points to the readers, such as refining the techniques of PCNL during the learning curve, early suspicious for renal hematomas when there is an unexplained postoperative hemoglobin drop, early imaging combined with therapeutic radiological interventions, and a low threshold for early exploration after failed initial embolization to save the kidney.

Declaration of competing interest

All authors disclose any financial and personal relationships with other people or organizations that could inappropriately influence their work.

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Ethical approval

Not applicable.

Consent

Informed written consent was obtained from the patient for publication of the data and clinical images. A copy of the written consent is available for review by the Editor-In-Chief of this journal on request.

Author contribution

M. Sivashankar, B. Balagobi, PGN Ruvinda have equally contributed to the concept, design, data collection, and writing of this case report. In addition, Professor Neville D Perera was the operated surgeon and clinical supervisor of this study.

Registration of research studies

Not applicable.

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