Page kidney - A review of the literature

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ABSTRACT: Page kidney was first described in animal experiments in 1939. In the 1950s and 1960s the human counterpart became evident. In this review we examine the modest literature on this rare but important renal/urological complication, summarize the clinical features, and discuss the best approach to diagnosis and management.

Key words: *Page kidney, Hematoma, Hypertension, Ischemia, Renal failure*

INTRODUCTION

This review concerns a rare clinical situation: "Page kidney" phenomenon. We first review briefly the history of this disease, then we will describe its pathophysiology and review all (around 75) the cases of Page kidney reported in the world literature. In addition we will describe all the available diagnostic modalities and end by suggesting modes of treatment and suggestions for future studies.

History of the Page kidney phenomenon

In 1939 Page published his experiment of induced hypertension where he wrapped a canine kidney with cellophane (1) and described an intense inflammatory reaction to this foreign material producing constrictive perinephritis, compression of the kidney parenchyma and hypertension. Page proved that extirpating the affected kidney could cure this high blood pressure. Page's observations were experimental until 1955 when he and a colleague reported a case of an American football player who suffered a blunt injury to the kidney producing a renal hematoma and renin-mediated hypertension (2). From then on cases of hypertension secondary to kidney compression were referred to in the literature as "Page" kidney. Since then there have been many case reports of secondary hypertension associated with acute or chronic unilateral subcapsular or perinephric hematomas in the literature. The most common scenario is a healthy young person with new onset hypertension with a history of, acute or remote, blunt trauma to the kidney.

Pathophysiology of the Page kidney

Any significant external compression of the kidney causes renal hypoperfusion and ischemia which activates the renin-angiotensin-aldosterone axis resulting in excess salt and water retention ultimately leading to hypertension (1, 3-7). This mechanism is similar to what Goldblatt et al reported in 1934 (8) where they demonstrated that hypertension and ischemia of the kidney ensue after deliberate ligation / constriction of a renal artery. Hypertension can be acute, secondary, to direct compression of the kidney by the hematoma or remote after the hematoma organizes into restrictive fibrous capsule ultimately compressing the kidney, interfering with intrarenal blood flow and causing ischemia. Although hypertension is the most common presenting disease, renal insufficiency can occur in the setting of diseased contralateral kidney. The im-

plications for renal function are usually much more severe in the context of a single functional kidney, as compression of renal parenchyma not only causes hypertension but also a decrease in glomerular filtration rate (GFR) of the ischemic kidney, which in the setting of a normal contralateral kidney that compensates by hyperfiltration, does not lead to a rise in creatinine. In patients with a single functional kidney – e.g. after renal transplantation – an acute Page kidney is usually associated with acute renal failure and hypertension (9).

Aetiology of Page kidney

Table I classifies the many different aetiological factors. In the past the most common presentation is a young male athlete with a history of blunt flank/abdominal trauma presenting with new onset hypertension. Most of the time the history of trauma is obscure or presumptive. American football is the most common reported type of sport implicated in Page kidney and this is for three main reasons – first, due to the large number of players and the popularity of this game in the USA. Second, due to a move in this sport called "spearing" where a helmeted player runs headfirst into his opponent and by hitting him in the abdomen or back causes him to fall to the ground (3, 10, 11). The third reason is the sheer bulk of most American footballers (typically 250 - 350 lbs.).

TABLE I - CAUSES OF PAGE KIDNEY

Bleeding secondary to trauma
American football
Other contact sports
Motor vehicle accidents
Bleeding secondary to interventions
Postoperative
Kidney biopsy
Extra corporeal shock wave lithotripsy
Sympathetic nerve block
Bleeding spontaneous
Pancreatitis
Warfarin therapy
Polyarteritis nodosa
Tumor
Non bleeding causes
Pararenal lymphoceles
Large simple cysts
Retroperitoneal paraganglioma
Urinoma
Perirenal pseudocysts
Peritransplant lymphocele

Page kidney is reported also in other contact sport such as judo, soccer, rugby and baseball (12-15). Particularly since ultrasound and CT imaging have been widely available, both for diagnostic and for interventional procedures, a growing number of reports in the literature were published describing Page phenomenon secondary to medical diseases and interventions. It is reported as complicating lithotripsy, kidney biopsy, and sympathetic nerve block (4, 5, 16). However, it can be spontaneous as in polyarteritis nodosa, warfarin therapy, pancreatitis, cancer and in hemodialysis patient with acquired cysts (3, 17-24).

In addition to bleeding there are reports of urinoma (urine under the capsule), large simple cysts, pararenal lymphoceles, perirenal pseudocysts and from retroperitoneal paraganglioma causing Page kidney (6, 7, 25-29). Finally it is reported as one of the causes of pseudorejection in kidney transplant patients (19, 29, 30).

Anatomically the kidney is a poorly protected retroperitoneal organ that is surrounded by two envelopes. The first is Gerota's fascia, which is a fat filled space that consists as the only shock absorbing system of the kidney. It is a large space that may communicate with the intraperitoneal cavity at the ureterovesical junction. A large hematoma is usually needed in this space to compress the kidney, which is the usual scenario after trauma. The second, is the kidney capsule which is a potential space that allows only small amount of blood to seep into it before compressing the kidneys and manifesting as hypertension or worsening renal function. Usually bleeding into this space does not cause hemodynamic instability as opposed to bleeding into Gerota's fascia. Subcapsular bleeding most commonly complicates renal biopsy and extracorporeal shock wave lithotripsy (ESWL). Transplanted kidneys in the cyclosporin era have particularly tough and rigid renal capsules.

Imaging modalities used in diagnosis

All imaging modalities have been tried to diagnose Page kidney. The main preference of any diagnostic modality over the others is a combination of the following: its sensitivity and specificity in detecting a hematoma or a fibrous capsule, its invasiveness and risk to the patients, cost and accessibility (Tab. II). Ultrasound has the advantage of being cheap, easy to perform and non-invasive but because it is operator dependent it can miss small compressive subcapsular hematomas (13, 31, 32). CT of the abdomen is a preferred modality, it can give a better resolution of the retroperitoneal space. It can diagnose small

hematomas and is relatively cheap, easy to perform and readily accessible in any small center (11, 15, 31). Magnetic resonance imaging (MRI) has the advantage of estimating the age of hematomas, which has therapeutic implications, as we will see later. It can also demonstrate patency of renal vessels (4). Renal arteriography can be used to evaluate hematoma and fibrous capsule by detecting vascular tears and calcifications around kidney. It may detect also parenchymal injury (33). The disadvantages of this modality are its low sensitivity, invasiveness and use of radiocontrast, which is nephrotoxic. Captopril renography has generally not been a useful diagnostic modality (34). Figure 1 demonstrates an example of Page kidney.

Treatment of Page kidney

Treatment of Page kidney is controversial and depends on many factors. The aim of any modality of treatment or procedure is to try to spare the kidney and cure hypertension. Spontaneous resolution of hematoma and hypertension was reported after 3 weeks of injury (35). In other cases where hematoma was old these could only be treated by nephrectomy (10). The major factor that affects the preferred modality of treatment is the age of hematoma, which can be estimated only by MRI.

In situations where blood pressure can be controlled easily by oral antihypertensives, and renal function is preserved, careful observation for a few months can be tried. ACE inhibitors (ACEI) have made a tremendous change in the management of Page kidney. Before this era surgery was the preferred modality because of the repetitive failures in controlling blood pressure using other oral antihypertensives (4). But with the use of these highly potent anti-hypertensives a few months trial of observation to control blood pressure can often be tolerated hoping that the acute hematoma can resolve by itself. Usually large hematomas are less likely to remit spontaneously.

The second line of treatment if hypertension persists, the hematoma is very large, or renal function deteriorates is to try to evacuate the hematoma percutaneously. Percutaneous drainage is a tempting modality of treatment as it is less invasive than surgery and less risky to the patient. The only problem is that old organized hematomas (fibrotic) are non-amenable to percutaneous drainage. Laparoscopy has been employed to try to evacuate the compressive hematoma with variable success rates depending on the expertise of surgeon (11, 32). If a fibrous pseudocapsule has formed then capsulectomy or stripping of the fibrotic area from the kidney can be tried but sometimes it is

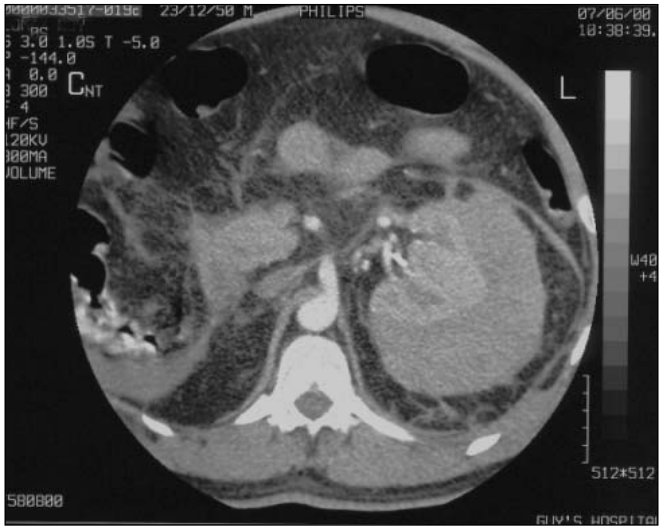

Fig. 1 - A large subcapsular renal hematoma following extracorporeal lithotripsy. In this case there was hypertension and acute renal failure which recovered with surgical extirpation of the hematoma.

TABLE II - DIAGNOSTIC IMAGING MODALITIES FOR PAGE KIDNEY

Modality	Cost	Advantages	Disadvantages
Ultrasound	+	Not invasive Readily accessible	Operator dependent Can miss small hematomas
CT scan	++	Not invasive Can detect small hematomas Readily accessible	
MRI	+++	Estimate age of hematomas Demonstrate patency of renal vessels	Not present in all centres Not readily accessible
Renal arteriography	++/+++	Evaluate renal arteries	Invasive Low sensitivity Use of radiocontrast

hard to remove it all and then a partial, or even total, nephrectomy is indicated (36). Nephrectomy used to be the most common modality of treatment of hypertension but often failed to treat hypertension. This might be due to one of two explanations. The first is that the fibrotic capsule was not significantly compressing the renal parenchyma and Page kidney cannot be accused of causing hypertension. The second is a long standing capsule and secondary hypertension can cause arteriolar damage of the non affected kidney and even after nephrectomy renin-angiotensin-aldosterone axis continue to be activated and hypertension persists. This is analogous to the difference between atherosclerotic, as opposed to fibromuscular dysplasia-induced, renal arterial disease. No test (even renal vein renin sampling) can adequately predict the success of nephrectomy and ultimately therefore predict a cure for hypertension (36, 37).

Due to the rarity of the disease and lack of controlled trials no clear recommendations can be followed and therapy of Page kidney should be individualized for each case bearing in mind the estimated time the kidney was compressed.

Conclusions

Page kidney is a rare but important cause of hypertension, and in the context of a single functional kidney, it may also be associated with acute renal failure. The commonest cause is blood compressing the renal parenchyma. Quick diagnosis and intervention can lead to successful resolution of hypertension and renal dysfunction.

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