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Page Kidney: A Curable Form of Arterial Hypertension

Case Report and Review of the Literature

Francesco Aragona, Walter Artibani, Antonio Calabrò, Giorgio Villi, Antonio Cisternino, Edoardo Ostardo

Institute of Urology, University of Padua, Italy

Key Words. Hypertension · Kidney, hemorrhage · Kidneys, wounds and injuries

Abstract. A case report of arterial hypertension which occurred 5 years after a blunt renal trauma is presented. The physiopathology of this type of hypertension along with the diagnostic features and the therapeutic aspects are extensively discussed. The sudden appearance of hypertension in a young patient following trauma must suggest a Page kidney as one of the diagnostic possibilities.

In 1939 Page [1] first created a persistent arterial hypertension in various laboratory animals by wrapping one or both kidneys with cellophan. The resultant perinephritis led to the formation of a thick fibrocollagenous hull that compressed the renal parenchyma without compromising the hilar vessels. Since then, the Page kidney refers to a secondary hypertension caused by compression of the renal parenchyma by a perirenal process without involvement of the main renal vessels.

As Page speculated and subsequently Waugh and Hamilton [2] have shown, compression of the parenchyma decreases blood flow through the kidney, producing the equivalent of the Goldblatt kidney: the renal ischemia, consequent to the parenchymal compression, activates the renin-angiotensin-aldosterone system resulting in hypertension [3]. Relief of extrarenal pressure or nephrectomy led to a normotensive state in experimental animals. Later, in 1955, Engle and Page [4] reported a 19-year-old patient with a calcified subcapsular hematoma and hypertension who became normotensive following nephrectomy. Since then, several other cases of hypertension caused by fibrous encasement of the kidney have been reported in the literature, mostly of traumatic origin. An additional case is reported herein and an extensive review of the literature is presented.

Case Report

A 25-year-old man was admitted in January 1986 for evaluation of arterial hypertension of 5 years' duration. Nine years before, the patient was hospitalized elsewhere because of a car accident resulting in a perinephric hematoma. The lesion was treated conservatively and the patient was asymptomatic until 1981 when he was noted to be hypertensive on a routine physical examination. There was no history of renal disease and no family history of hypertension. At admission, physical examination was normal except for elevated blood pressure (150/110 mm Hg). Urinalysis and routine laboratory studies were all within normal limits. On excretory urogram (IVP) (fig. 1) the upper calyces of the left kidney appeared distorted and displaced downward. A selective left renal arteriogram (fig. 2 A) showed a renal artery of normal caliber but splaying of the intraparenchymal vessels, consistent with mass effect. No tumor vessels were seen. A prominent capsular artery (fig. 2 A) was separated from the parenchyma by an avascular mass; in a later phase (fig. 2 B) nonhomogeneous stain and flattening of the lateral border of the kidney were also seen. Split renal vein renin (RVR) determinations

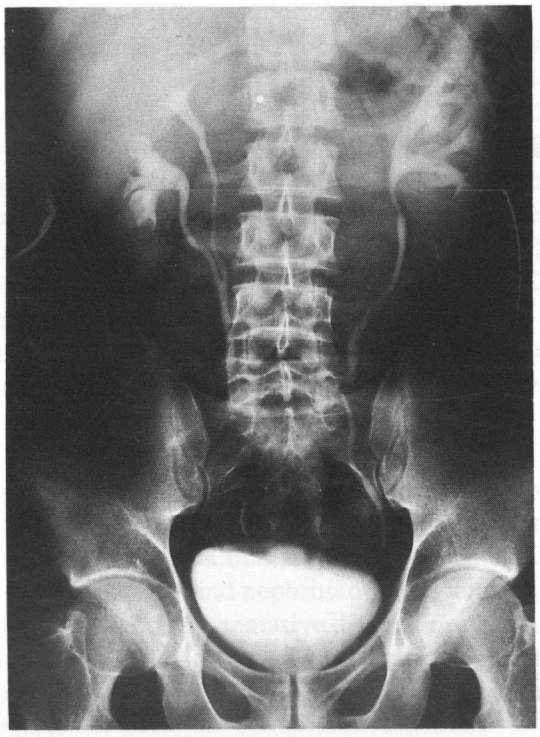
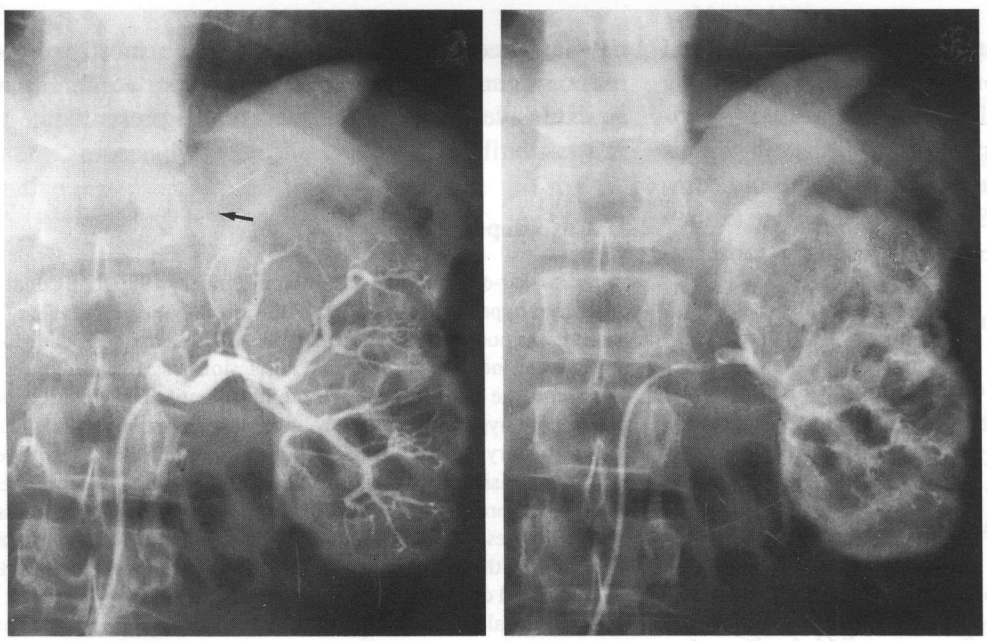


Fig. 1. IVP. Upper calyces of the left kidney appear distorted and displaced downward, showing extrinsic pressure changes. No definite left renal outline is visible.

indicated that the left kidney was responsible for the hypertension (RVR ratio right:left was 0.92:2.01; renin in the inferior vena cava below the renal veins: 1.02 ng/ml/h). A computed tomography (CT) scan (fig. 3) revealed a low density (+ 10 HU) fluid collection in the posterolateral aspect of the left kidney. The sudden appearance of hypertension in a young patient following trauma and the radiological finding were all characteristics of a Page kidney and a surgical exploration was carried out. A dense, fibrous capsule, containing approximately 200 ml of a relatively clear fluid, was found to compress the renal parenchyma without involvement of the renal hilar vessels (fig. 4). The cyst-like formation occupied two thirds of the posterior border of the kidney. The cyst had a 2-mm-thick wall and, since there was no obvious parenchymal involvement (fig. 5A, B), it was decorticated except for its fibrous base that remained on the kidney. Microscopic examination of the specimen showed a cystic wall consisting of fibrous tissue without epithelial lining (fig. 5C). The postoperative course was uneventful and the patient is normotensive without medication at a 3-year follow-up.

Discussion

Page kidney is an uncommon cause of hypertension. So far, about 70 cases have been reported, most of them (78%) occurring following perinephric hematoma secondary to blunt or penetrating injury to the kidney. In spite of a history of renal trauma, only 41% of these patients had gross hematuria and 4% microscopic hema-



A **B**

Fig. 2. Selective left remal arteriography **A** The arterial phase demonstrates no renal artery narrowing and stretching, splaying and attenuation of the intrarenal vessels, consistent with mass effect. Note the prominent capsular artery separated from the parenchyma by an avascular mass (arrow). **B** The nephrogram phase demonstrates a nonhomogeneous stain suggesting areas of ischemia or thinning of parenchyma.

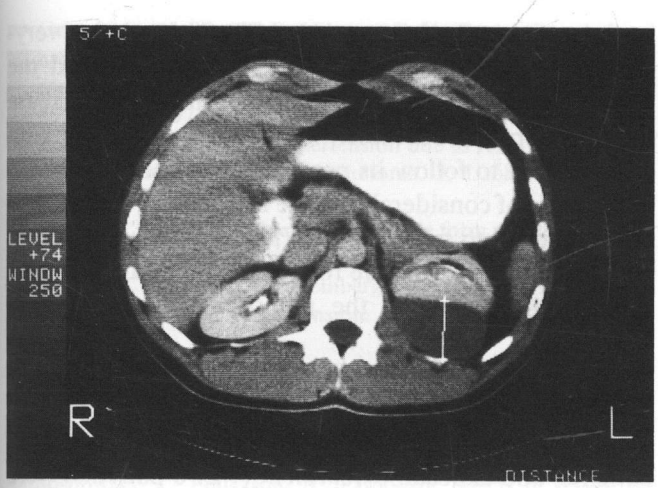


Fig. 3. CT shows compression of the renal parenchyma by an elliptically shaped (6.6 × 3.5 cm in diameter), low-density (+10 HU) fluid collection lying lateral and posterior to the left kidney and confined by the perirenal fascia.

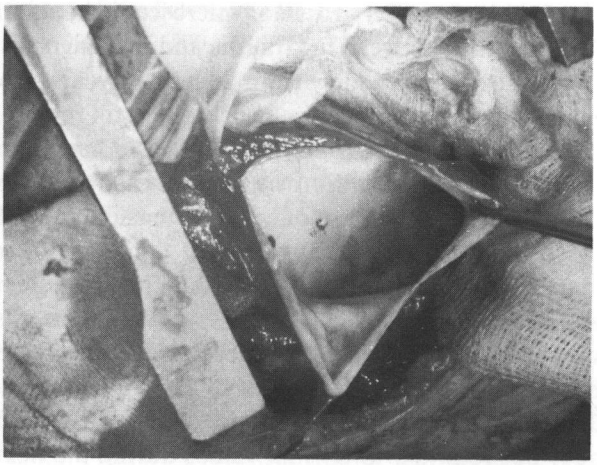
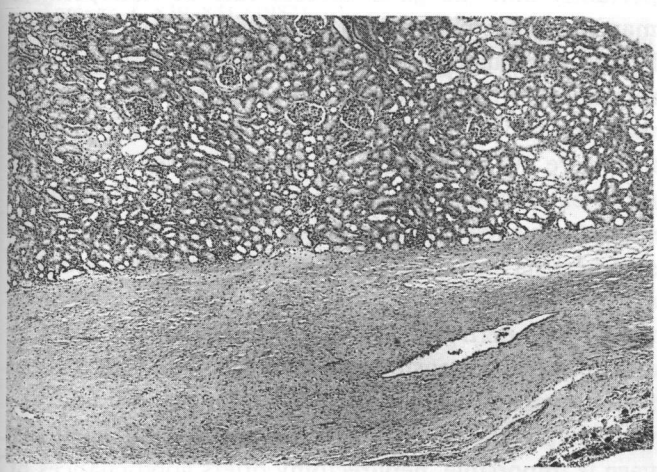


Fig. 4. Intraoperatively, a dense fibrous capsule, containing about 200 ml of clear fluid, was found to compress the renal parenchyma.



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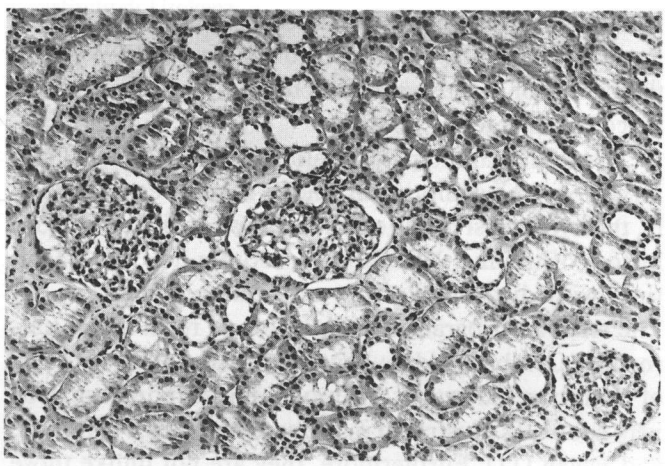

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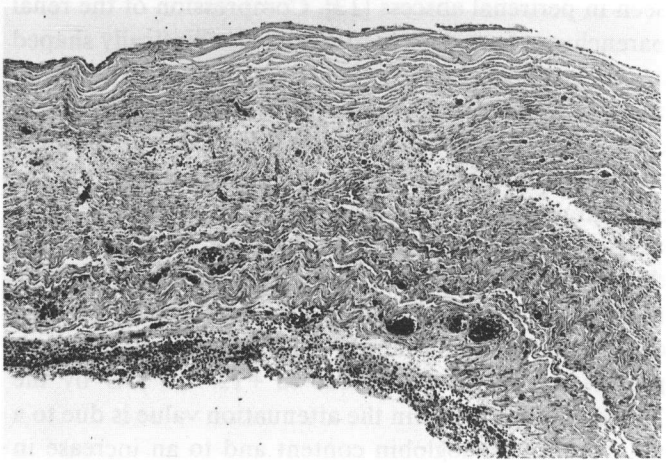

C

Fig. 5. A Renal biopsy close to the fibrous capsule does not reveal a reduction in number or regressive changes in glomerular morphology. H-H. × 10. **B** A higher magnification confirms the absence of glomerular alterations. H-H. 125 ×. **C** Cystic wall consists of fibrous tissue devoid of epithelial lining. H-H. × 25.

turia; 55% had neither gross nor microscopic hematuria [5].The time lag between the trauma and the diagnosis of hypertension varied widely from 24 h to 12 years. However, as the development of a Page kidney still remains a rare complication, it is likely that only the hematomas who cause subcapsular fibrosis and parenchymal compression are prone to produce hypertension [6]. This may contribute to explaining the low incidence of secondary hypertension in patients with perirenal hematomas following percutaneous renal biopsy. In the cases reviewed by Lingardh and Schonebeck [7] a heavy perirenal bleeding occurred in 7 out of 390 renal biopsies: only in 1 case did the blood pressure increase after biopsy but returned to normal values during the next 5 days. In recent years, extracorporeal shockwave lithotripsy (ESWL) has changed our therapeutic approach to the urolithiasis. However, experimental [8] and clinical [9, 10] studies have demonstrated that ESWL, at least in the acute stage, produces renal damage, mainly edema and extravasation of urine and blood in the interstitial space. Most of these lesions are reversible but others may be permanent and contribute to the development of secondary hypertension with a mechanism similar to the Page kidney. Today, hypertension following ESWL is in the center of extensive research but its clinical importance remains to be assessed.

The radiographic pattern along with the clinical history allow a presumptive diagnosis in most cases. Usually, the hypertensive patient is young and with a previous history of trauma. IVP shows a definite delay in transit time with distortion of the calyceal configuration [6]. The renal arteriogram demonstrates no renal artery narrowing (Goldblatt kidney) but attenuation and stretching of the intrarenal vessels. However, these findings may be very subtle, as illustrated in the case reported by Amparo and Fagan [11]. Furthermore, they are not unlike those seen in perirenal abscess [12]. Compression of the renal parenchyma by a sharply marginated, elliptically shaped area, lying lateral and posterior to the kidney and confined to the perirenal space, is the main CT finding in perinephric subcapsular hematomas [13]. The attenuation characteristics of the hematoma depend on the age and duration of the bleeding, freshly extravasated blood having a higher attenuation value (from +30 to +50 HU) than a more chronic hematoma [14]. Experimentally, Moss et al. [15] demonstrated that the attenuation value of a fresh hematoma ranged from +20 to +40 HU with progressive decrease in value to +15/+20 HU by the 28th day. The change in the attenuation value is due to a reduction in hemoglobin content and to an increase in water content. In our case, the CT finding of a very-low-density subcapsular mass (+10 HU) indicated the presence of a chronic liquefied hematoma. The decrease in the absorption coefficient of the perirenal hematoma may be used to follow its progression toward resolution [14]; this is of considerable value in adopting a conservative approach to treatment. RVR activity is elevated in all cases and confirms the role of the renin-angiotensin-aldosterone system in the pathogenesis of hypertension.

Treatment of the Page kidney has included observation, drainage and decortication, or nephrectomy. In 29 cases treated by Sufrin [5], both nephrectomy (15 of 17 patients) and expectant treatment (7 of 8 patients) were successful (88%) while drainage and decapsulation cured 2 of 4 patients (50%).

In our opinion, the management of subcapsular hematomas must be individualized, the choice of therapy depending upon the clinical setting, the degree of hypertension and the duration of subcapsular hematoma. Small, asymptomatic hematomas may be managed expectantly and the good success rate of nonoperative management emphasizes that a constricting perirenal hematoma causing hypertension can be reabsorbed with remission of hypertension [16–18]. Some perirenal hematomas fail to be reabsorbed: factors leading to chronicity are not clear: although it is unproved, coexisting urine extravasation may contribute to fibrosis [19]. If spontaneous reabsorption does not occur and hypertension persists for more than 2 months [18], simple drainage of the fluid is often insufficient and decortication of the fibrous perirenal process is neccessary in order to ameliorate hypertension and preserve renal function before irreversible damage occurs. In long-standing cases, when hypertension cannot be controlled and renal salvage is impossible, nephrectomy offers good chances of definitive treatment. A Page kidney is one of the diagnostic possibilities in the work-up of a young patient who suddenly develops hypertension following a renal trauma. Appropriate and timely treatment will lead to a normotensive state in the majority of cases.

References

1 Page, I.H.: The production of persistent arterial hypertension by cellophane perinephritis. J. Am. med. Ass. *113:* 2046–2048 (1939).

2 Waugh, W.H.; Hamilton, W.F.: Physical effect of increased venous and extrarenal pressure on renal vascular resistance. Circulation Res. *6:* 116–121 (1958).

3 Grim, C.E.; Mullins, M.F.; Nilson, J.P: Unilateral 'Page kidney' hypertension in man: studies of the renin-angiotensin-aldosterone system before and after nephrectomy. J. Am. med. Ass. *231:* 42–45 (1975).
4 Engle, W.J.; Page, I.H.: Hypertension due to renal compression resulting from subcapsular hematoma. J. Urol. *73:* 735–739 (1955).
5 Sufrin, G.: The Page kidney: a correctable form of arterial hypertension. J. Urol. *113:* 450–454 (1975).
6 Scott, P.L.; Yune, H.Y.; Weinberger, M.N.: Page kidneys: an unusual cause of hypertension. Radiology *119:* 547–548 (1976).
7 Lingardh, G.; Schonebeck, J.: Acute hypertension and perirenal hematoma, a new aspect of Page's syndrome. Br. J. Urol. *47:* 359–361 (1975)
8 Newmann, R.; Hackett, R.; Senior, D.; Brock, K.; Feldman, J.; Sosnowsky, J.; Finlayson, B.: Pathologic effects of ESWL on canine renal tissue. Urology *29:* 138–141 (1987).
9 Kande, J.V.; Williams, C.M.; Millner, M.R.; Scott, K.N.; Finlayson, B.: Renal morphology and function immediately after ESWL. AJR *145:* 305–313 (1985).
10 Colombo, R.; Centemero, A.; Trabucchi, E.; Francesca, F.; Di Girolamo, V.; Rigatti, P.: Lesioni renali acute dopo trattamento di litotrissia extracorporea ad onde d'urto. Valutazioni istologiche ed ultrastrutturali. Acta. urol. ital. *2:* 73–77 (1988).
11 Amparo, E.G.; Fagan, C.J.: Page kidney. J. Comput. assist. Tomogr. *6:* 839–841 (1982)
12 Pollack, H.M.; Popky, G.L.: Roentgenographic manifestations of spontaneous renal hemorrhage. Radiology *110:* 1–6 (1974).
13 Schaner, E.G.; Balow, J.E.; Doppman, J.L.: CT in the diagnosis of subcapsular and perirenal hematoma. AJR *129:* 83–88 (1977).
14 Sagel, S.S.; Siegel, M.J.; Stanley, R.J.; Jost, R.G.: Detection of retroperitoneal hemorrhage by CT. AJR *129:* 403–407 (1977).
15 Moss, A.A.; Korobkin, M.; Price, D.; Brito, A.C.: CT of splenic subcapsular hematomas: an experimental study in dogs. Investve. Radiol. *14:* 60–64 (1979).
16 Massumi, R.A.; Andrada, A.; Kramer, N.: Arterial hypertension in traumatic subcapsular perirenal hematoma (Page kidney). Ann. J. Med. *46:* 635–637 (1969).
17 Jameson, R.M.: Transient hypertension associated with closed renal injury. Br. J. Urol. *45:* 482–484 (1973).
18 Noble, M.J.; Novick, A.C.; Straffon, R.A.; Stewart, B.M.: Renal subcapsular hematoma: a diagnostic and therapeutic dilemma. J. Urol. *125:* 157–160 (1981).
19 Sufrin, S.: Editorial comment. J. Urol. *125:* 160 (1981).

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Francesco Aragona, MD
Istituto di Urologia
via Giustiniani 2
I–35100 Padova (Italy)