Renal Hypertension due to Giant Perirenal Haematoma: Permanent Resolution by Percutaneous Ultrasound-guided Drainage

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We describe a case of renovascular hypertension accompanied by renal failure, arising in a young man with a solitary kidney 4 months after a blunt abdominal trauma. A giant haematoma was found around the right kidney and ultrasound-guided percutaneous drainage completely relieved the symptom complex. Nine years later, the patient is normotensive with normal renal function.

Key words: long-term follow-up, Page kidney, percutaneous drainage, renovascular hypertension

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In 1939 Page demonstrated experimentally that constriction of one or both kidneys by a cellophane-induced fibro-collagenous hull resulted in hypertension, probably secondary to renal ischaemia caused by compression of the small intraparenchymal arteries (10). In 1943 Braasch reported the first clinical observation of this phenomenon (1) and by 1991 a total of 80 cases were reported in the English language literature (8). Successful treatment of Page kidney is usually accomplished by nephrectomy, drainage and decapsulation or watchful waiting (9).

We describe a case of post-traumatic renal hypertension due to Page phenomenon, successfully treated by percutaneous ultrasound-guided drainage of a giant perirenal haematoma. During a 9-year follow-up period no recurrence of hypertension has been recorded and the patient is currently asymptomatic.

CASE REPORT

A 15-year-old white male came to our attention because of a rapidly growing palpable mass in his right flank and hypertension. Four months earlier, during a soccer match, the patient had accidentally received a violent blunt abdominal trauma that had apparently left no consequences. The patient specifically denied gross haematuria and his past medical history was unremarkable. He had been practising competitive sports for a long time and frequent medical check-ups had never highlighted any abnormality of either blood pressure or renal function.

At hospital admission his blood pressure was 170/110 mm Hg. Blood tests revealed increased serum creatinine (2.7 mg.%) and blood urea nitrogen (133 mg.%), although daily urinary output was still within the range of normality. Abdominal ultrasound evidenced a vast perirenal fluid collection pushing the right kidney anteriorly and medially (Fig. 1). The left kidney was congenitally absent. Plasma renin activity was 21 ng/ml per hour (normal 0.3–2.9 ng/ml) and ^{99m}Tc-DTPA renal scintigraphy with the captopril test confirmed the presence of decreased parenchymal perfusion with flattened time–activity curves. The patient received a percutaneous ultrasound-guided drainage, which yielded 3500 ml of unclotted blood. The catheter was removed 1 week after drainage. By the same time the patient became normotensive and recovered to normal renal function tests. During the following 2 months, the treatment was completed by 3 fine-needle aspirations that finally led to complete resolution of the perirenal haematoma. Control radioisotope renography evidenced a normal profile (Fig. 2).

After a follow-up period of 9 years the patient is normotensive, completely asymptomatic and has normal renal function tests.

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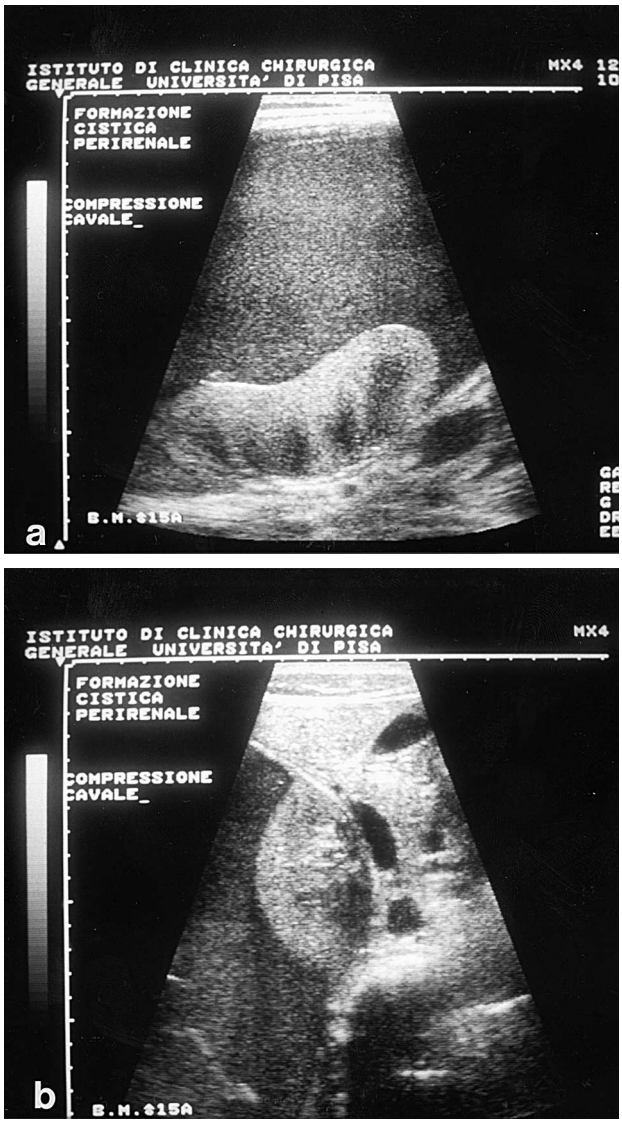

Fig. 1. Coronal (a) and transverse (b) right subcostal ultrasound scans, demonstrating a fairly echogenic giant perirenal fluid collection. The kidney is displaced in an anteromedial position. The inferior vena cava is pushed anteriorly and clearly flattened.

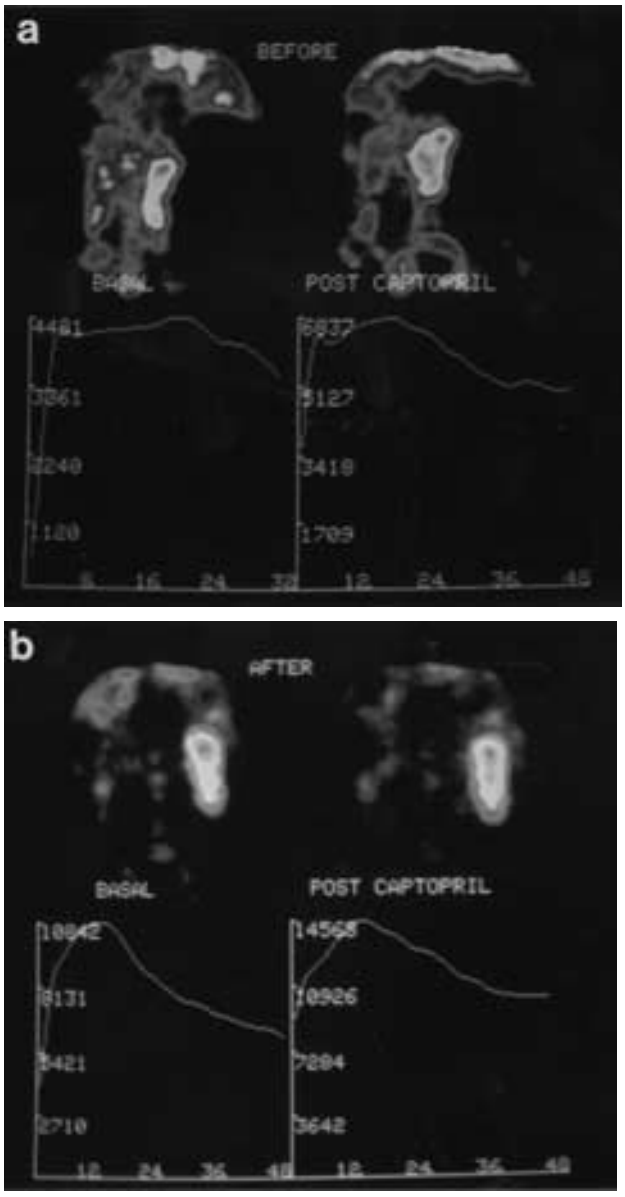

Fig. 2. ^{99m} Technitium-DTPA renal scintigraphy with captopril test. Predrainage scans (a) show inhomogenous radionuclide uptake and flattened time–activity curves. Post-drainage scans (b) show normalization of both radionuclide uptake and time–activity curves.

CONCLUSIONS

Renovascular hypertension is a well-known complication after either blunt or penetrating renal injuries (2, 9). Reported incidences diverge extremely in different series, and a documented normotensive state before the injury is mandatory for the diagnosis (9). Post-traumatic renal hypertension may be transitory but tends to become persistent once definite damage to the renal parenchyma has occurred (2). The majority of renal trauma patients are young adults and hypertension has an ominous prognostic significance in this age group (2). Blunt trauma may lead to hypertension by means of two separate mechanisms. First, renal artery stenosis or occlusion may result from subintimal tears, external compression or pedicle avulsion. Second, the renal parenchyma may be compressed by subcapsular or perirenal fluid collections that subsequently organize into a constricting fibrous capsule sparing hilar vessels (2, 9). These mechanisms have their experimental counterparts in the animal models proposed by Goldblatt (4) and Page (10). Alternatively, post-traumatic renal hypertension may result from a penetrating lesion with arteriovenous fistula formation and decreased blood flow (2, 9). In all cases, hypertension is supposed

to originate from renal ischaemia with stimulation of the renin–angiotensin system (2, 9).

The exact number of reported cases of hypertension caused exclusively by renal compression without associated narrowing of major renal vessels (Page kidney) is difficult to determine because of incomplete information on many of them. In 1991 McCune reviewed the English language literature on this topic and found only 80 cases with sufficient documentation (8). Blunt abdominal trauma represents the most common aetiological agent. Accordingly, most patients suffering from Page kidney are young males who underwent motor vehicle accidents or sport-related injuries. Other less-frequent causes include needle biopsy, cyst puncture, lumbar sympathetic nerve block, renal tumour, bleeding diathesis, anticoagulant therapy and polyarteritis nodosa (2, 9, 12). In a small fraction of cases the cause may go unrecognized (7, 11). Time lag from renal injury to development of significant hypertension may vary from 24 h to more than 20 years (9). However, a limit of 5 years has been suggested by several authors in order to establish a rational connection between the two events (2, 9). Hypertension may result from either early parenchymal compression from large fluid collections or late renal constriction from thick fibrous perinephritis (9). Renal function may remain normal because of contralateral compensation, or it may significantly worsen in cases of either pre-existing decreased renal reserve or solitary kidney (5, 13).

Currently recommended therapeutic approaches include observation, drainage and decapsulation or nephrectomy (9, 12). Small subcapsular or perirenal haematomas may be managed conservatively until complete resolution (2, 9). Acute hypertension due to large perirenal haematomas can be effectively managed by early evacuation (9). However, therapy is usually surgical and we found only one published case of percutaneous drainage as definitive therapy of renal hypertension (6). In the remaining cases nephrectomy or renal decapsulation represented the most common therapeutic alternatives (2, 9, 12).

In our case, a previously normotensive young man developed a palpable right flank mass and hypertension 4 months after a blunt abdominal trauma. The left kidney was congenitally absent and renal failure became a prominent clinical feature. Abdominal ultrasound promptly highlighted the presence of a giant perirenal haematoma. Renal scintigraphy confirmed the presence of decreased renal function and the captopril test proved positive for renal hypertension. Ultrasound-guided drainage of the perirenal haematoma was easily accomplished. Renal function and blood pressure promptly recovered to normality. A few fine-needle aspirations were required to completely clear the perirenal space and avoid late constrictive scaring. After a 9-year follow-up period the patient is asymptomatic with no sign of either hypertension or renal disease. Reviewing the literature we found one case in which a fibrotic perirenal hull had already developed by the time a percutaneous fine-needle aspiration was attempted. Although initially successful, the procedure failed to persistently relieve hypertension (3).

In conclusion, the excellent short- and long-term results obtained in this case suggest that percutaneous drainage should be considered a safe and effective alternative to surgical therapy in the management of selected cases with renovascular hypertension due to Page phenomenon. In order to avoid a late constrictive scarring reaction with permanent kidney damage, care should be given to achieve complete clearance of the perirenal space.

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