Case Report

A Case of Hyperreninemic Hypertension after Extracorporeal Shock-Wave Lithotripsy

Manabu SASAGURI, Keita NODA, Takemasa MATSUMOTO, Kazuyuki SHIRAI, Emiko TSUJI, Yuji TSUJI*, and Kikuo ARAKAWA

A 53-year-old male was found to have hypertension caused by the significant secretion of renin from an atrophic left kidney. He had undergone extracorporeal shock-wave lithotripsy (ESWL) for left renal lithiasis 11 years previously. A renal dynamic study with 99mTc-diethylenetriaminepentaacetic acid (DTPA) indicated that the rate of renal excretion and uptake was decreased in the left kidney and normal in the right kidney. Renal angiography demonstrated a normal right renal artery and a small but nonstenotic left renal artery. The ratio of PRA in the left renal vein to that in the right renal vein was 1.7. Blood pressure could be lowered to the range of 140-150/80-90 mmHg with imidapril, an ACE inhibitor. ESWL may cause hypertension via the well-known Page kidney effect. In this case, the kidney, atrophic probably due to ESWL, released a significant amount of renin. (Hypertens Res 2000; 23: 709-712)

Key Words: hyperreninemic hypertension, renovascular hypertension, extracorporeal shock-wave lithotripsy, Page kidney

Introduction

Hypertension in unilateral renal disease can be caused by renovascular hypertension, but also by chronic atrophic pyelonephritis, congenital hypoplasia, tuberculosis, renal cyst, renal encystment (Page kidney), hydronephrosis, and tumors (1). Renovascular hypertension may give rise to renal artery stenosis or renal ischemia in response to atherosclerosis, fibromuscular dysplasia, Takayasu's arteritis, middle artery syndrome, neurofibromatosis, aortic dissection, renal artery aneurysm, dissection of the renal artery, renal arteriovenous fistula, radiation arteritis, renal artery embolus, renal transplantation or extrinsic obstruction (2).

Extracorporeal shock-wave lithotripsy (ESWL) is widely applied for the treatment of renal lithiasis. The side effects of ESWL include hemorrhage, edema, and acute tubular necrosis of the kidney. ESWL is also associated with hypertension, which can either occur immediately or be delayed by several weeks or months (3). The present report describes a case of hyperreninemic hypertension after ESWL.

Case Report

A 53-year-old male was seen in our hospital for elevated blood pressure (168/110 mmHg) on June 22nd, 1999. He had come to our hospital in 1988 due to general malaise. At that time, high blood pressure (150/100 mmHg) and a stone-like shadow on abdominal X ray had been found. Plasma renin activity (PRA) was 2.1 ng/ml/h and the plasma aldosterone concentration was 110 pg/ml. On ultrasonography, hydronephrosis due to ureterolithiasis was observed in the left kidney; however, the size of the left kidney was the same as that of the right kidney. Subsequently, the patient underwent extracorporeal shock-wave lithotripsy for left renal lithiasis at another hospital. Although he had been taking a 2-mg dose of trichlormethiazide once a day for 2 months, an antihypertensive
drug was not administered until this visit from 1988 to 1999. Since 1997, blood pressure has been found to be elevated at regular annual check-ups. According to his family history, only his elder brother has exhibited signs of hypertension.

In our outpatient clinic, the patient’s blood pressure was found to be 170/104 mmHg. Left ventricular hypertrophy was observed on electrocardiogram (SV1+RV5 = 4.87 mV) and echocardiography (interventricular septum = 12 mm, left ventricular posterior wall thickness = 12 mm, left ventricular end-diastolic diameter = 39 mm). Five milligrams of imidapril, an ACE inhibitor, was initially administered. Three weeks later, the daily dose of imidapril was increased to 10 mg. Blood pressure decreased to a range of 150-156/80-90 mmHg. A renal dynamic study with 99mTc-diethylenetriaminepentaacetic acid (DTPA) was performed because the plasma renin activity was 3.0 ng/ml/h and the plasma aldosterone concentration was 261 pg/ml on the patient’s first visit, indicating that renal uptake and the excretion rate were decreased in the left kidney.

**Fig. 1. Renal dynamic study with 99mTc-diethylenetriaminepentaacetic acid (DTPA). (A) Uptake of 99mTc-DTPA was low in the left kidney. (B) Uptake and the excretion curve for 99mTc-DTPA were decreased in the left kidney.**

ESWL is widely applied in the treatment of renal lithiasis. The side effects of ESWL include hemorrhage, edema, and acute tubular necrosis of the kidney. This form of renal trauma is associated with an immediate decrease in renal function of the treated kidney. ESWL is also associated with hypertension, which may occur immediately or can be delayed by several weeks or months. Approximately 8% of patients develop hypertension requiring pharmacological treatment 1 year after ESWL (3, 4). Recently, Ashida et al. have shown that hypertension (>160 mmHg) can develop in 3 of 97 patients as a late complication associated with ESWL (5).

However, the mechanism of hypertension after ESWL is unclear. Williams *et al.* have suggested that it could be due to a long-term decrease in effective renal plasma flow caused by renal fibrosis, as has been observed in canine kidneys after ESWL (6). Peterson and Finlayson (7) have suggested that renal trauma caused by ESWL may cause hypertension as a result of a perirenal hematoma via the well-known Page kidney effect. Page has observed that constriction of the renal parenchyma itself without compromise of the main renal vessels alters intrarenal hemodynamics to produce renal ischemia leading to renin release (8). When Grant *et al.* followed 33 patients with renal trauma, they observed hypertension in 13 of these 33 from 9 weeks to 16 years. Four of these 13 patients had subcapsular hematomas (9). The mechanism of hypertension in these cases is the same as that in the Page kidney. It has actually been shown by sensitive MRI methods that 63% of post-ESWL patients exhibit some abnormalities such as a loss of corticomедullary differentiation, perirenal fluid, subcapsular hematoma, and hemorrhage into a renal cyst (10, 11). In our case, the left kidney was atrophic, probably due to ESWL, since atrophy of the left kidney was not observed on ultrasono-
Ashida et al. have observed renal atrophy in 5 of 97 patients as a late complication associated with ESWL (5).

In our case, PRA was 13.6 ng/ml/h in the left renal vein and 8.0 in the right renal vein, with a ratio of 1.7. A renal vein renin ratio over 1.5 reportedly indicates that the affected kidney secretes renin into the total circulation, resulting in elevations in blood pressure (12, 13). In our case, the atrophic left kidney secreted a significant amount of renin. In the Page hypertension model using rabbits, i.e., one wrapped kidney, PRA in the peripheral vein was not elevated, but rather was observed to increase only when a low-salt diet was administered (14). However, three patients presenting with a remediable form of hypertension caused by Page kidney showed a significant increase in the renal venous renin ratio, suggesting that hypertension in these cases was of renovascular origin (15-17). Moreover, their blood pressure returned to normal after nephrectomy of the affected kidney. Patients with hypertension caused by the same mechanism have been shown to exhibit hydronephrosis and renal infarction, where the affected kidney secretes a significant amount of renin (13, 18). One of the underlying mechanisms of hypertension in our case is similar to

Fig. 2. (A) Aortography. There was no stenosis in either of the main renal arteries. The left kidney was atrophic. In digital subtraction arteriography (B, C), instead of being stenotic, the left renal artery was atrophic in appearance, while there were no abnormalities in the right renal artery or kidney.
that in these cases because the co-existence of essential hypertension cannot be ruled out. The atrophy of the left kidney was probably due to ESWL, as other causes such as stenotic renal artery and hydronephrosis were not observed.

In conclusion, ESWL may cause hypertension via the well-known Page kidney effect. In the case reported here, the kidney, which was probably atrophic due to ESWL, released a significant amount of renin, resulting in hyperreninemic hypertension. At present, the blood pressure in this patient can be controlled within the range of 140-150/80-90 mmHg with an ACE inhibitor.

**References**