A Case of Hyperreninemic Hypertension with Unilateral Hydronephrosis

Hidenori Urata, MD, Setsuo Masui, MD*, Munehito Ideishi, MD, Yoshihiro Kato, MD, Masaharu Ikeda, MD and Kikuo Arakawa, MD

A patient with unilateral ureteral obstruction by urolithiasis at the ureteropelvic junction was accompanied by hypertension. Plasma renin activity (PRA) was high in peripheral veins and was significantly higher in the renal vein drained from the affected kidney than the contralateral. Infusion of angiotensin II antagonist or an oral administration of captopril, an angiotensin converting enzyme (ACE) inhibitor, resulted in a prompt drop of blood pressure. After pyelolithotomy was successfully performed, both the blood pressure and peripheral PRA completely normalized. It is suggested that the renin-angiotensin system might have played a major role in the mechanism of the accompanied hypertension. Hyperreninemia could have been caused by both renal ischemic vasoconstriction, which might be due to uretero-renal reflex, and increased synthesis of prostaglandins resulting from ureteropelvic obstruction.

Key Words: Plasma renin activity, Prostaglandins, Captopril, Ketoprofen

Hyperreninemic hypertension associated with unilateral ureteral obstruction rarely occurs. Moreover, the mechanism of hypertension has not been fully defined. We reported a case of unilateral hydronephrosis with hyperreninemic hypertension. A relief of ureteral obstruction with pyelolithotomy improved the hypertension, which was accompanied with the normalization of PRA. There were also some clinical evidence to support the mechanism of hyperreninemic hypertension in this case.

CASE REPORT

A forty years old house wife had been quite healthy until six months before admission. She was attacked with severe dull pain on left hypochondrium and high grade fever on February 28, 1980. Her blood pressure was 208/122 mmHg. The attack occurred three times. Antibiotic therapy was repeated by her home doctor with good response each time. When she was re-attacked with sudden left abdominal colicky pain with high grade fever on July 19, she was referred to our hospital. Anticholinergic drugs were injected and she was relieved from pain. Blood pressure was 182/104 mmHg. Abdominal plain X-ray film revealed two large renal stone, one of which appeared to obstruct left uretero-pelvic junction.

Past history was negative. A younger brother had a nephrectomy for renovascular hypertension. Both parents died of hepatic cirrhosis.

On August 12, her blood pressure was 152/106 mmHg (right arm), 154/110 (left arm), and she had severe knock pain on left lumber region but there was no colicky pain. Cultures of urine yielded many pseudomonas (≥10^5 colonies/ml). Acute inflammatory signs, such as CRP (3+),
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accelerated erythrocyte sedimentation rate (27 mm/1 hr, 55 mm/2 hr) and leucocytosis (12,000/mm³) were observed. Numeral white blood cells were counted in the urinary sediment. Serum urea nitrogen level was 10 mg/dl. Serum creatinin level was 0.9 mg/dl. Value of creatinin clearance was 849 dl/day. Serum sodium content was 141 mEq/l; serum potassium 3.7 mEq/l; serum chloride 98 mEq/l. Values for twenty-four hours urinary excretion of catecholamines, vanillylmandelic acid, 17-OH-CS, 17-KS were all within normal limit. Peripheral Plasma renin activity was 10.5 ng/ml/hr (normal 0.3-1.4 ng/ml/hr at rest). Urinary aldosterone excretion for 24 hours was 21.5 μg. Because of her high fever, 150 mg/day of ketoprofen, an anti-inflammatory and prostaglandin synthesis inhibitor, was prescribed for two days. Her blood pressure decreased from 160/110 mmHg to 132/90 mmHg, together with a fall of PRA at peripheral vein from 10.5 ng/ml/hr to 3.2 ng/ml/hr. They elevated again after the drug was discontinued (Fig. 1).

Transverse section of enhanced abdominal computed tomography revealed moderate left hydronephrosis with dilated pelvis and preserved cortex. There was no evidence of the atrophy of left renal cortex (Fig. 2), suggesting that the hydronephrosis was in acute phase.

An abdominal aortogram and selective renal arteriograms were also performed (Fig. 3). The aorta and the right renal artery were normal. The branches of left renal artery was generally narrow, which might be due to uretero-renal reflex, but there was no evidence of segmented renal arterial stenosis.

Renogram with 131I-hippuran revealed normal uptake by right kidney and scarce uptake by left kidney.

PRA of renal vein 15 minutes after stimulation by diazoxide injection was 11.0 ng/ml/hr in the left, 7.25 ng/ml/hr in the right, and 7.5 ng/ml/hr in the inferior vena cava distal to the renal veins. The PRA ratio between the renal vein of the hydronephrotic and contralateral kidney was greater than 1.5, while that between the contralateral kidney and peripheral was less than 1.0.

By the infusion of [Sar⁴, Ile⁸]-angiotensin II, an angiotension II antagonist, on dose of
Fig. 3. (a) Selective left renal arteriogram showing generally small narrowing artery but no segmental narrowing. (b) Selective right renal arteriogram showing normal arteries and arterioles.

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Fig. 4. Effect of angiotensin II antagonist on blood pressure before operation.

An administration of 50 mg of captopril, an ACE inhibitor, resulted in a decrease of blood pressure from 160/120 mmHg to 120/85 mmHg (Fig. 5). PRA level elevated from 5.5 ng/ml/hr to 9.0 ng/ml/hr during infusion of angiotensin II antagonist, and from 4.0 ng/ml/hr to 12.5 ng/ml/hr due to administration of captopril, respectively.

Results of laboratory data, abdominal echogram and abdominal computed tomography suggested that the function of the left kidney preserved and correctable, since the hydronephrosis was apparently acute phase. Left pyelolithotomy was performed on September 12, 1980. Renal cortex was well preserved without microabscess and discoloration. One of the two stone was fixed and completely obstructed the ureteropelvic junction. The other was mobile in the pelvis. Sodium content of residural urine in the pelvis was 88 mEq/l, potassium content was 9 mEq/l. Creatinin level was 10 mg/dl. Fractional excretion of sodium (FENa) of affected kidney was 9%, fractional excretion of potassium (FEK) 21%.
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V/GFR (Pcu/Ucr) 9% which suggested that affected kidney might be reversible.

After operation, blood pressure fell to around 130/80 mmHg with concomitant decrease of plasma renin activity (Fig. 1). Intravenous pyelogram performed one month later revealed the efflux of contrast medium from the affected kidney and ureter. Renogram also showed apparent improvement of initial slope (vascular phase) of the left kidney.

On thirtieth day after operation, her blood pressure was 140/88 mmHg but both the infusion of angiotensin II antagonist and an administration of ACE inhibitor failed to decrease in blood pressure any further.

DISCUSSION

Hydronephrosis as a cause of high blood pressure was suspected by Friedrichs as early as in 1835, as Abeshouse reviewed. Ritch noted a reversal of hypertension after surgical dilatation of the ureter in patient with hydronephrosis. Ever since there have been several reports that unilateral hydronephrosis complicated with hypertension was surgically corrected and consequently blood pressure normalized. However, Wesson suggested that hypertension was uncommon and the peripheral PRA was within normal limits in most of hypertensive patients with unilateral hydronephrosis. Vaughan et al. reported thirteen cases of unilateral hydronephrosis. In their cases, PRA was high in one case (8%), normal in 10 (76%) and low in 2 (16%). However, Belman et al. first reported the hyperreninemic hypertension with unilateral ureteral obstruction. Similar case reports were followed by Stockit et al., Nemoy et al., Squitiery et al., Wise, Andarolo, Carella and Silver, Chapman and Douglas, Weidmann et al., Gabriel and Heslop, and Pak et al.

Even though the level of peripheral PRA was normal, increased renal vein renin activity of affected kidney was often observed, suggesting that renin-angiotensin system might play an important role in these cases. As to renovascular hypertension, measurement of renal vein renin activity may be useful in predicting surgical curability of the hypertension. The ratios of plasma renin activity of hydronephrotic kidney to that of contralateral greater than 1.5, and one of contralateral kidney to that of peripheral vein less than 1.2 have been shown to be a good surgical curability index of the hypertension. Weidmann et al. successfully applied this index to the surgical curability of unilateral hydronephrosis with hypertension.

Pak et al. indicated participation of renin-angiotensin system in associated hypertension with unilateral hydronephrosis based on high level of renal renin activity on an affected side and depressor response to angiotensin II antagonist. There have been no report on diagnostic use of captopril to the hypertensive patient with unilateral hydronephrosis. The present paper clearly showed that captopril is as useful as angiotensin antagonist for screening angiotensinogenic hypertension associated with urolithiasis.

In the present case, the role of renin-angiotensin system in hypertension was suspected by three reasons. First, increased renin activity in both peripheral and renal vein from the affected kidney was obvious (PRA ratio of hydronephrotic to contralateral kidney being 1.52, and that of contralateral kidney to peripheral vein 0.96). Second, both the infusion of angiotensin antagonist and an administration of captopril significantly lowered blood pressure. Third, after surgical relief from ureteral obstruction, blood pressure normalized and plasma renin activity simultaneously became within normal range.

In experimental animals, Klein et al. and Vaughan et al. showed increased plasma renin activity concomitant with hypertension after artificial ureteral obstruction. In spite of these elaborated experimental and clinical observation, there have been few reports on the definite role of renin-angiotensin system in hypertension with unilateral ureteral obstruction. The mechanism of enhanced renin secretion during unilateral ureteral obstruction is not known. But it may be related to such conditions as ischemia, activating renin baroreceptors, alterations of the macula densa receptors, or uretero-renal reflex. Uretero-renal reflex is thought to be sympathetic reflex back to the kidney to constrict the renal arterioles. In the present case,
left renal arteriogram showed that renal artery and arterioles were generally narrowing without segmental stenosis, renal scintigraphy revealed low uptake by the affected kidney and the first phase of renogram was depressed. These observations suggested that the uretero-renal reflex might result in high secretion of renin and this might caused hyperreninemic hypertension in this case.

Recently it is also noted that the prostaglandins and their metabolites might play a major role in both vasoconstriction (TX A2) and vasodilation (PG E2, PG I2) in obstructed kidney. A possible relationship between renin-angiotensin system and prostaglandins was revealed in experimental study. PRA level increased from three to five fold after acute unilateral obstruction in rabbit with concomitant-hypertension, which was parallel with output of immunoreactive PG E2 and PG F1 α that increased from three to fivefold without changes in creatinine clearance. Increased PRA was suppressed by prostaglandin synthesis inhibitor.

In our case, because of fever and concomitant pyelonephritis, ketoprofen, a prostaglandin synthesis inhibitor, was prescribed for two days. Incidentally, her blood pressure lowered from 160/110 to 132/90 mmHg with decreased plasma renin activity and both elevated again after the drug was discontinued. These findings coincide with the previous report that prostaglandins might partially induce high secretion of renin and it has not been proved in the clinical case of unilateral hydronephrosis.

In conclusion, both renal ischemia, which might be due to uretero-renal reflex, and increased synthesis of prostaglandins resulting from ureteropelvic obstruction could lead to hyperreninemic hypertension in this case.

REFERENCES

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