Case Report

A Case of Hypertension Due to the Left Renal Vascular Stenosis with Aplasia of the Right Kidney

Kazuo SHIRASAKI, M.D., Akiyuki OKUBO, M.D.,
Hiroshi KURIHARA, M.D., and Masao IKEDA, M.D.

SINCE the report on experimental hypertension by Goldblatt in 1934,¹⁾ renal vascular stenosis has been one of the most wellknown causes of hypertension both in human beings and in experimental animals. Increasing attention has been focused on unilateral renal hypertension in recent years concerning its pathogenesis and surgical treatment.

With the advance of adequate diagnostic methods, the reports of cases with hypertension due to stenosis of renal arteries^{2), 3)} has also shown a steady increase. This report illustrates a case of severe hypertension probably due to the stenosis of the left renal arteries with aplasia of the right kidney. Such a case has been of an extremely rarity in the literature up to this time.

CASE REPORT

A 26-years-old man was admitted to University of Tokyo Hospital on December 25, 1962, for a detailed examination on the cause of hypertension which was found by a physician approximately 1 month prior to admission.

His relatives are free of renal and cardiovascular diseases. His birth was normal at full term. His development was uneventful, without any history of abdominal trauma.

On November 26, 1962, he had attacks consisting of headache, slight disturbance in speech and impaired sensation on the left arm. On account of the blood pressure of 220 mm. Hg in systolic and 120 mm. Hg in diastolic, hospitalization was recommended. Physical examination on admission revealed a well nourished and developed man. Pulse was regular, 72 per min. and respiration was normal. Blood pressure was 190 mm. Hg in systolic and 118 mm. Hg in diastolic.

No significant difference of blood pressure was noted between the upper and lower or the right and left extremities. Examination of ocular fundi revealed slight arterial narrowing without hemorrhages and papilledema. There was no edema on the face and legs. The forceful cardiac apical beat was localized in an area in

From the Third Department of Internal Medicine (Prof. K. Nakao), Faculty of Medicine, University of Tokyo, Tokyo.

the left 5th intercostal space 2 cm. lateral from the midclavicular line. The cardiac dullness was extended to the left and grade I systolic murmur was heard at the apex. The percussion over the lung fields revealed no abnormality and the breath sound was normal. A systolic ejection murmur was heard on the left side of the abdomen. The kidney was palpable on the left side but not on the right side. Neither liver nor spleen was palpable and there was no ascites. Neurologic examination was normal.

Laboratory findings: Urinalysis; Specific gravity 1.024, albumin negative, sugar negative, urobilinogen negative. Sediments were normal.

Hemogram; Hemoglobin 98%, RBC 532×10^4 , WBC 6,400 with normal differential count, hematocrit 51%, erythrocyte sedimentation rate 4 mm. per hour.

Blood chemistry; Total serum protein 7.2 Gm./100 ml., A/G ratio 0.23, serum urea nitrogen 17 mg., serum creatinine 1.0 mg, total cholesterol 180 mg. per 100 ml. Sodium, potassium and chloride were all within normal range. Liver function tests revealed no disturbance. Regitine test was negative and catecholamine in urine was within normal range. C-reactive protein and serological tests for syphilis were negative.

PSP excretion was 35% in 15 min. and 65% in 120 min. Fishberg's concentration test was normal. The urea clearance was 69.0 ml. and creatinine clearance was 108 ml. per minute. GFR was 127 ml., RPF 657 ml. per min. and FF 0.187. Intravenous indigocarmine dye appeared in 3 min. at the left ureteral orifice but did not appear at any time in the right side.

The electrocardiogram (Fig. 1) showed the pattern of left ventricular hypertrophy with regular sinus rhythm.

Fig. 1. Electrocardiogram showing left ventricular hypertrophy pattern.

Roentgenogram; Chest X-ray (Fig. 2) showed cardiac enlargement with the prominence of left ventricular contour. Intravenous pyelogram (Fig. 3) revealed an enlargement of the left kidney with good concentration of the dye, but no excretion of dye was seen in the right kidney. Retroperitoneum (Fig. 4) also revealed enlarged outline of the left kidney and absence of the outline of the right kidney. Translumbar aortogram (Fig. 5) disclosed stenosis of the left renal artery,

Fig. 2. P-A chest X-ray showing cardiac enlargement with the prominence of left ventricular contour.

Fig. 3. Intravenous pyelogram showing enlarged left renal outline with good dye concentration and no excretion of dye in the right side.

Fig. 4. Retropneumoperitoneum showing enlarged outline of the left kidney and absence of the right renal outline.

whereas the right renal artery was not demonstrated. Snaky collateral circulations were observed from the abdominal aorta to the left kidney.

Renoscintigram using Hg^{203} -Neohydrin as well as retropneumoperitoneum showed an enlarged outline of the left kidney and absence of the right renal outline. In I^{131} -radioisotope renogram with I^{131} tagged Hippuran (Fig. 6), a normal curve was obtained except for a slight prolongation of the plateau for the left kidney but the curve for the right side indicated the absence of the kidney.

During the hospitalization, his blood pressure, ranging from 190 to 180 mm. Hg in systolic and 130 to 110 mm. Hg in diastolic, did not respond to various anti-

Fig. 5. Translumbar aortogram demonstrating stenosis of the left renal artery, absence of the right renal artery, and snaky collateral circulation from abdominal aorta to the left kidney.

Fig. 6. I¹³¹-tagged Hippuran renogram showing normal curve for the left kidney and the tracing indicating the right renal agenesis.

hypertensive drugs including rauwolfia, ganglionic blocking agents, thiazide derivatives and α -methyl DOPA.

DISCUSSION

Hypertension due to the stenosis of the left renal artery with aplasia of the right kidney as reported by Decamp et al.,⁸⁾ has not yet been reported in Japan to date.

According to Campbell,⁷⁾ renal aplasia is seen in approximately 1% of the autopsy cases. Since various laboratory findings in this case were consistent with aplasia of the right kidney, hypertension in this case seemed to be the result of the vascular stenosis of the left kidney.

In their review of 104 translumbar aortographies, Poutasse and Dustan⁹⁾ found stenosis of the renal arteries in 30 cases. Translumbar aortography is of value in: 1) unexplained disparity of size or function of the kidneys by intravenous urography, 2) hypertensive young people, 3) elderly patients with sudden acceleration of malignant hypertension, 4) those patients with symptoms suggesting previous renal infarction. Selective renal angiography by retrograde arterial catheterization instead of translumbar aortography has been recommended in recent years by other investigators.

Brest et al.¹⁰⁾ classified renal vascular hypertension as seen in Table I. Hunt et al.¹¹⁾ described the frequent occurrence of beaded shadows in renal arteriogram in cases with fibrous or fibromuscular stenosis of the renal arteries. A few small round shadows were noted in the left renal artery and its branches on renal arteriogram besides the picture of stenosis in the present case. It was very difficult actually to decide whether these shadows represent aneurysms or poststenotic enlargement owing to fibrous or fibromuscular stenosis of renal arteries. O'Connor¹²⁾ mentioned that intrarenal aneurysm was quite rare in comparison with extrarenal one, and renal aneurysm was frequently found bilaterally.

Table I.
Anatomic Lesions Responsible for Renovascular Hypertension

A. Congenital
1. Renal artery stenosis (fibromuscular hyperplasia)
2. Abdominal aortic coarctation (with or without renal artery stenosis)
3. Renal artery aneurysm
B. Acquired
1. Atherosclerosis
a. Involving renal artery
b. Involving aorta (with ascending thrombotic occlusion)
2. Thrombosis or embolism of renal artery (with or without infarction)
3. Renal arteritis
4. Trauma to renal artery (with thrombosis, perirenal hematoma, arteriovenous fistula, foreign body occlusion)
5. Tumor or fibrous bands involving renal artery

(Brast, A.N. et al.¹⁰⁾)

Ueda et al.¹³⁾ reported that pan-aortitis was the most frequent cause of renovascular hypertension in Japan in contrast to western countries. The absence of laboratory findings indicating inflammation such as accelerated

erythrocyte sedimentation rate, leucocytosis as well as the radiologically normal appearance of the aortic wall make the association or etiological role of pan-aortitis in this case most unlikely, although the possibility of renovascular stenosis due to the scar of burnt out inflammation cannot be entirely ruled out. The beaded shadows in the left renal artery in this case is probably consistent with congenital fibrous or fibromuscular change. The relationship between the aplasia of the right kidney and the vascular abnormality on the left side remains in doubt.

As for the treatment of unilateral renovascular hypertension, arterial repair or bypass has been recommended. Decamp et al.⁹⁾ successfully treated a case of hypertension caused by left renal vascular stenosis with absence of the right kidney with end-to-side splenorenal arterial anastomoses. However, the probable extension of fibrous or fibromuscular lesion to the small branches of the left renal artery and the preservation of a good renal function make the indication of such operation in this case quite doubtful.

SUMMARY

A case of severe hypertension caused by the left renal vascular stenosis with aplasia of the right kidney was presented. The causative lesion of this renovascular stenosis is considered to be fibrous or fibromuscular hyperplasia of the renal artery.

ACKNOWLEDGMENT

Authors are indebted to Prof. K. Nakao, the Director of our Department for the revision of this manuscript.

REFERENCES

1. Goldblatt, H., Lynch, J., Hanzal, R.F., and Summervill, W.W.: *J. Exper. Med.* **59**: 347, 1934.
2. Donaraj, T.J. and Wong, H.O.: *Circulation* **20**: 856, 1959.
3. Goodman, H.L.: *New Engl. J. Med.* **246**: 8, 1952.
4. Cordonnier, J.J.: *J. Urol.* **82**: 1, 1959.
5. Wylie, E.J. and Wellington, J.S.: *Am. J. Surg.* **100**: 183, 1960.
6. Platt, W.R.: *J. Urol.* **85**: 24, 1961.
7. Campell, M.: *Urology* **1**: 239, 1957.
8. DeCamp, P.T. and Birchall, R.: *Surgery* **43**: 134, 1958.
9. Poutasse, E.F. and Dustan, H.P.: *J.A.M.A.* **165**: 1521, 1957.
10. Brest, A.N., Heider, C., and Moyer, J.H.: *Am. J. Cardiol.* **9**: 748, 1962.
11. Hunt, J.C., Harrison, E.G., Kincaid, O.W., Bernatz, P.E., and Davis, G.D.: *Proc. Staff Meet. Mayo Clin.* **37**: 181, 1962.

12. O'Connor, V.J.: New Engl. J. Med. **262**: 456, 1960.
13. Ueda, H., Wada, T., Takeda, T., Shintani, F., and Nakamura, H.: Jap. Heart J. **4**: 497, 1963.