Renovascular Hypertension Due to Buerger's Disease

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SUMMARY

In a 42-year-old man with severe hypertension, stenosis of the left renal artery at its origin and occlusion of the abdominal aorta below the level of the renal arteries were observed. His past history and clinical and laboratory findings suggested that the renal artery stenosis was due to Buerger's disease which was reported to be a rare cause of renovascular hypertension.

Additional Indexing Words:
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M ANY kinds of vascular diseases have been noticed as the causes of renal arterial stenosis, which produce hypertension. Buerger's disease, if the lesion occurs in the renal vessels, may also produce hypertension, but only a few of such cases of renovascular hypertension have been reported. The present report is concerned with a patient whose sustained hypertension is considered to be due to Buerger's disease (thromboangiitis obliterans) affecting the left renal artery.

CASE REPORT

A 42-year-old man was admitted to the hospital in May, 1975, complaining of severe headache. He has smoked more than 20 cigarettes a day since 17 years of age. At the age of 30, he felt so strong pain in both legs as to stop walking and found a small painful ulcer on the left little toe. He underwent lumbar sympathectomy under the diagnosis of Buerger's disease, but the operation brought him no favorable effect. Three months later, his left little toe was amputated for gangrene. The right big toe was amputated in 1964, and the distal portions including metatarsi of both feet were amputated during the following 2 years. Amputation of the left thigh and ablation of the adrenal medulla were performed in 1967, then amputation of the right thigh in 1968. The histological findings of the specimens obtained at these operations were not available. In 1963, numbness of the right hand on exercise appeared, to which cyanosis was added on exposure to cold. These

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symptoms continued until the time of admission. He was noticed, for the first time, to have hypertension (210/120 mmHg) in 1973, then proteinuria in 1975. He had 4 brothers and none of them suffered from such symptoms as he did.

He had twin children of 3 years of age.

Physical examination on admission showed a well nourished man with moderate stature in no acute distress. Blood pressure of the right arm was 208/108 mmHg and that of the left arm was 212/112 mmHg. Radial arterial pulsation, which was 80/min and regular, was normally palpable on the left side, but somewhat less on the right side. The chest was clear to percussion and no murmur was noted. In the abdomen, vascular bruit, which was high-pitched and continuous, was heard in the area by some 5 cm on the left and upward to the navel. Both lower extremities were amputated at the middle of the thigh. Femoral arterial pulsation was absent on both sides.

Laboratory examination revealed proteinuria of 0.8 Gm/day without abnormal sediments. Anemia was absent. The serum sodium was 143 mEq/L, potassium 4.4 mEq/L, and chloride 105 mEq/L. The PSP excretion was 30% in 15 min and the creatinine clearance was 92.4 ml/min. Diabetic pattern was not noted in the oral glucose tolerance test with 50 Gm of glucose. The plasma renin activity in recumbent position in the morning was 35 ng/ml, the normal range of which was 3 to 5 ng/ml under daily sodium intake of 6 Gm in our laboratory. The erythrocyte sedimentation rate was 3 mm/hour.

C-reactive protein, antinuclear factor, and LE cells were negative. The ocular fundi showed angiosclerosis and dotted hemorrhages. The thermogram showed hypothermia of the skin of the right hand (Fig. 1). The rapid sequence pyelography demonstrated a normal pyeloureter tract on the right and faintly visible calyces on the left 3 min after the injection of dye, but after 20 min the left pelvis shadow was more opaque than the right (paradoxical hyperconcentration, Figs. 2, 3).

The renogram showed delayed accumulation and excretion of the isotope in
Fig. 2 (left). Rapid sequence pyelography demonstrated a normal pyeloureter tract on the right and faintly visible calyces on the left 3 min after the injection of dye.

Fig. 3 (right). Rapid sequence pyelography after 20 min showed that the left pelvis shadow was more opaque than the right (paradoxical hyperconcentration).

the left kidney. The translumbar aortography showed that the aorta was almost completely occluded below the origin of the renal arteries and the origin of the left renal artery was markedly stenosed. However, the size of the nephrogram was equal on both sides, 11.3 × 6 cm on the left side and 11.5 × 6 cm on the right side, respectively (Fig. 4). The other portion of the aorta looked normal without any signs of atheromatous sclerosis. Infusion of 500 ng/Kg/min of 1-Sar-8-Ile-angiotensin II was performed for 45 min. The blood pressure was 185 mmHg systolic and 108 mmHg diastolic before the start of the antagonist infusion. The systolic blood pressure temporarily dropped in the beginning of infusion, then rose up to 190 mmHg, and gradually declined again, reaching 160 mmHg around 30 min after the start of infusion. It remained at that level during the period of infusion. The diastolic blood pressure began to drop in the beginning of infusion and reached 84 mmHg after 30 min, remaining at that level thereafter (Fig. 5).

The plasma renin activity in recumbent position was 34.5 ng/ml before infusion and 20.1 ng/ml at the end of infusion. In the separate kidney function test, the urinary creatinine and sodium concentrations were 124 mg/100 ml and 76 mEq/L on the left, and 63 mg/100 ml and 136 mEq/L on the right, respectively. The tubular ejection fraction ratio was 2.285, also being suggestive of the presence of ischemia in the left kidney.
From the above-mentioned findings, the patient was diagnosed as having renovascular hypertension due to stenosis of the left renal artery, probably caused by Buerger's disease. After the completion of examinations, 120–180 mg a day of

Fig. 4. Trans-lumbar aortography showed that the aorta was almost completely occluded below the level of the renal arteries and the origin of the left renal artery was markedly stenosed. However, the size of the nephrogram was equal on both sides, 11.3×6 cm on the left and 11.5×6 cm on the right side.
Fig. 5. Infusion of 500 ng/Kg/min of 1-Sar-8-Ile-angiotensin II was performed for 45 min. The blood pressure was 185 mmHg systolic and 108 mmHg diastolic before the start of infusion. The systolic pressure temporarily dropped in the beginning of infusion, then rose up to 190 mmHg, and gradually declined again, reaching 160 mmHg around 30 min after the start of infusion. It remained at that level during the infusion thereafter. The diastolic pressure began to drop in the beginning of infusion and reached 84 mmHg after 30 min, remaining at that level thereafter.

metoprolol, a cardioselective beta-blocking agent, was orally administrated. Smoking has been strictly prohibited and the clinical course of this patient is now followed up. The blood pressure at rest is maintained around 160 mmHg systolic and 90 mmHg diastolic.

DISCUSSION

Buerger’s disease (thromboangiitis obliterans) is a clinical entity which represents an obliterative disease or syndrome, probably inflammatory in origin type, the lesions of which are chiefly in peripheral arteries and veins. The lesions have been scarcely found in vessels in the central viscera, including the abdominal aorta.\textsuperscript{1,2}

In 1951 Malisoff et al.\textsuperscript{3} however, reported the first curable case of a 34-year-old patient suffering from hypertension which resulted from thromboangiitic occlusion of the right renal artery. This patient had shown a history of thromboangiitis obliterans in the lower limbs and was preoperatively diagnosed as having thrombosis of the renal artery and resultant hypertension. After
right nephrectomy, the blood pressure returned to normotensive level (125/70). The histological examination of the extirpated renal artery revealed the changes typical of thromboangiitis obliterans. In this report, Malisoff et al reviewed several previously reported cases and pointed out that the renal vessels could be affected more frequently than generally believed.

In reviewing all of the 18 autopsy cases of Buerger’s disease in the literatures up to 1934, Sprunt\(^4\) stated that one or both renal arteries were involved in 8 cases. Podoba\(^5\) reported a case of 36-year-old man with severe hypertension (200/140), in whom the postmortem study revealed the stenosis of renal arteries of both sides due to Buerger’s disease. However, Gilkes and Dow\(^6\) called attention that earlier case reports of Buerger’s disease should be carefully differentiated from atherosclerosis in identifying the lesions of visceral arteries.

In the present patient, the disease began with intermittent claudication and painful dolent ulcer without muscular atrophy of the lower extremities as early as 30 years of age. The symptoms were not relieved by lumbar sympathectomy and ablation of adrenal medulla, and finally amputation of both thighs were performed. Since the beginning of the disease, he also suffered from numbness on exercise and pain on exposure to cold in the right upper extremity, which have continued up to the admission. The thermogram of the right upper extremity revealed hypothermia of the skin of the right hand.

Although the histological examination of arterial tissue was not possible in the present study, the aortography demonstrated no atheromatous plaques in the remaining portion of the aorta. When the clinical course, signs and symptoms of the patient and the aortographic findings were taken into consideration together, it seemed likely that hypertension of the patient was caused by renal artery stenosis on the left side, affected by Buerger’s disease.

In addition, the high PRA, the decrease in blood pressure during the infusion of angiotensin II antagonist and the favorable hypotensive effect of a beta-blocking agent suggested that the hypertension was renin-dependent.

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References