Hypertension due to Renal Artery Stenosis Caused by Abdominal Aortic Aneurysm

Successful Treatment by Nephrectomy


It is now more than a quarter of a century since Goldblatt et al. reported that partial occlusion of one renal artery could produce a sustained rise in blood pressure. Wilson and Byrom extended this work, and showed that this increase in blood pressure could sometimes be reversed by removal of the clamped kidney. Since that time there have been many reports of the cure of hypertension by nephrectomy when renal disease has been shown to be unilateral. Most of these successes, however, have been in patients with unilateral renal disease due to pyelonephritis, and much less commonly in patients with local obstruction of a renal artery. More recently, aortography has been widely employed to define stenotic lesions of the renal arteries, and since this technique has been introduced it has been shown that such lesions of the renal vessels are more common than was previously supposed. De Camp and Birchall reviewed the causes of renal artery stenosis, and found only one case that was associated with aneurysm of the abdominal aorta. Brown et al. added two further cases.

We report here a patient in whom this rare association occurred, and whose high blood pressure was successfully treated by removal of the affected kidney.

Case Report

The patient was a 21-year-old Nigerian houseboy who gave a history of three attacks of pain in the left loin, which occurred 11, 8, and 4 months before he came to the hospital. The pain was colicky, radiated around the abdomen and to the left groin, and lasted from 3 to 7 days. He denied both hematuria and frequency of micturition. He had noticed a pulsatile swelling in his abdomen after the first attack of pain and about this time he also began to have headaches. There were no other symptoms. The family history was irrelevant; nine siblings were alive and well. His blood pressure varied from 240/140 to 180/100 (the latter after sedation); there was some left ventricular enlargement with a soft ejection systolic murmur in the mitral area. A pulsatile swelling was palpable in the mid and lower abdomen, and a bruit was audible above and to the left of the umbilicus; the femoral pulses were present and equal. The ocular fundi showed grade-I changes.

The hemoglobin, white cell count, serum electrolytes, and urine were normal. Chest x-ray and electrocardiogram showed light left ventricular hypertrophy.

Intravenous pyelogram showed bilateral excretion of dye, possibly more concentrated on the left side; the left renal pelvis and calyces were smaller than the right, and the upper part of the left ureter was displaced away from the midline (fig. 1).

Aortogram was done by the Seldinger technique, retrograde from the right femoral artery. An aneurysm of the abdominal aorta was demonstrated that extended from the level of the tenth dorsal vertebra to the bifurcation of the aorta and involved the left common iliac artery. The right renal artery was normally filled, but the left renal artery could not be clearly seen. It proved impossible to catheterize the left renal artery, which seemed abnormal. The nephrogram showed that the right kidney was 12.5 cm. long, but the left kidney was less than 9 cm. long and of irregular contour (figs. 2, 3, and 4).

Renal biopsy on the right side showed no abnormality other than slight interstitial fibrosis. Eleven normal glomeruli were seen (fig. 5a). Bilateral ureteric catheterization was performed but technically was not altogether satisfactory. The only reliable results at 1 hour were: Right kidney, volume 224 ml., urea 280 mg. per cent, creatinine 0.3 mg. per cent; left kidney, volume 42 ml., urea 640 mg. per cent, creatinine 0.9 mg. per cent.

On this evidence a diagnosis of stenosis of the left renal artery with secondary ischemic contraction of the left kidney was made. Theoretically this could have been caused by direct pressure of the large abdominal aneurysm on the renal artery or by involvement of the mouth of that vessel in the aneurysmal process. It was thought more

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likely, however, that the enlarging aneurysm had stretched the left renal artery as it arose from the posterior aspect of the aorta, and that this stretching had predisposed to thrombosis and hence to infarction of the kidney.

Reconstruction of the aorta was not feasible, and since no abnormality of the right kidney could be demonstrated, it was decided to remove the left kidney. Nephrectomy was done through an abdominothoracic incision. The abdominal aneurysm extended to a level of 6 cm. above the diaphragm, and there was no clot surrounding it. At the level of the origin of the left renal artery there was a localized bulging, so that the left renal artery was, in fact, stretched and narrowed as it ran behind the aneurysm to the left kidney. There was also a piece of white thrombus in the lumen.

The kidney itself was small, weighing only 80 Gm.; it was 8.3 cm. long and 5 cm. wide at its widest point. The capsule stripped easily, and there were three depressed scarred areas on its surface. Injection of the renal artery with radio-opaque dye showed a reduction in the number and caliber of the arteries and arterioles, especially in the upper pole; this particularly affected the arcuate arteries (fig. 6). When the kidney was cut, it was seen that the cortex was shallow and its depth varied from 4 to 12 mm., averaging about 6 mm. The medulla was about 12 mm. deep throughout. The pyramids and calyces appeared healthy.

Histologically, the cortex was normal between the scars, but there was a greater number of glomeruli and tubules per unit area than normal. The scars consisted of crowded glomeruli in various stages of fibrosis most of whose associated tubules had disappeared. The sear tissue was infiltrated with lymphocytes, histiocytes, and occasional plasma cells. The arteries and arterioles appeared normal, and endarteritis was not seen in the vessels in the sears. This was thought to be a hypoplastic kidney with focal pyelonephritis (fig. 5b).

The postoperative course was uneventful. The day after operation the blood pressure fell to normal; there was a transient rise on the second postoperative day lasting about 24 hours, following which normal tension persisted. When last seen, 6 months after nephrectomy, the blood pressure was 140/95 (standing).

Discussion

Gellman found that renal infarction was common in patients with hypertension due to lesions of the renal arteries. Our patient had three episodes of pain consistent with renal infarctions, and yet, although the sears found on the surface of the kidney confirmed that this complication had occurred, histology

Figure 1
Intravenous pyelogram. The left renal pelvis and calyces are smaller than the right, and the concentration of dye from the left kidney is greater than that from the right. The upper part of the left ureter is displaced laterally.

Figure 2
The upper limit of the aneurysm lies at the level of the tenth dorsal vertebra.
of the kidney also showed pyelonephritis. That there was disordered function of this kidney was suggested by the smaller volume of urine secreted and the greater concentration of creatinine in the urine; these are among the characteristic features of the kidney affected by renal artery stenosis.²

It is well known that stenosis of the renal artery, whether acquired or produced experimentally, frequently serves to protect the kidney distal to the stenosis from the consequences of the raised blood pressure. The extent of this protection seems to be governed by the degree of stenosis. But when this stenosis is severe the kidney will be the seat of ischemic instead of secondary hypertensive vascular changes. The histologic picture in our patient suggests that the stenosis was extreme, because of the lack of secondary hypertensive vascular changes, and that the kidney may have had a poor arterial supply for years, resulting in its being hypoplastic. The absence of hypertensive changes in the contralateral “normal” kidney, however, strongly suggests that the rise in blood pressure was of recent origin, possibly due to recent pyelonephritis.

The large abdominal aneurysm was particularly interesting in this patient, but its
etiology is obscure. Atherosclerosis is extremely uncommon in the Nigerian, and has not been reported in this age group. The negative Kahn test makes syphilis extremely unlikely, and there were no other stig mata of a connective-tissue disorder such as the Marfan syndrome. Abrahams and Cockshott, however, have recently described a series of young Nigerians in whom they found multiple nonsyphilitic aneurysms of the great vessels of uncertain etiology; the extensive abdominal aneurysm in this patient may well be an expression of the same condition. Isaacsen has reported an idiopathic aortitis giving rise to aneurysm formation in young Africans, and five of his six patients had hypertension due to renal artery obstruction. It is thus possible that our patient had the same disease as Isaacsen described, which he thought was a new condition with similar lesions to those found in the pulseless disease/giant-cell arteritis complex.

The response to nephrectomy has been gratifying, although the blood pressure is not absolutely normal. There may have been minimal irreversible changes due to hypertension in the right kidney that were not seen in the biopsy specimen, and that could cause the minimal rise of pressure 6 months after operation.

Summary

An extensive aneurysm of the thoracic and abdominal aorta involving the left renal artery was found in a 21-year-old Nigerian man who presented with abdominal pain and hypertension. Nephrectomy was followed by a significantly lower pressure. The etiology of the aortic aneurysm is discussed briefly.

Acknowledgment

We wish to thank Dr. C. J. Barton for doing the aortogram, Mr. E. J. M. Weaver for the ureteric catheterization, Professor W. W. Davey for the nephrectomy, and Professor I. Donich for his report on the kidney. Mr. Frank Speed prepared the illustrations.

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Circulation. 1962;26:104-107
doi: 10.1161/01.CIR.26.1.104

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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