The Surgical Significance of Aberrant Renal Arteries in Relation to Systemic Hypertension

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In a previous paper we reported a strong statistical correlation between the incidence of aberrant renal arteries and the incidence of systemic hypertension.1 We have since seen three clinical cases of systemic hypertension, the origin of which could be traced directly to segmental renal ischemia secondary to insufficiency of an aberrant renal artery.

Case Reports

Case 1

A 7-year-old boy was first seen by his local pediatrician in October 1956, with complaints of polydipsia, polyuria, and enuresis. Blood pressure recordings at this time were equal in all extremities and were elevated to as high as 280/150 mm. Hg. Intravenous pyelography and a phentolamine (Regitine) test were reported as negative. In November 1956, the patient underwent exploratory laparotomy. The findings were negative.

He was referred to the University of Texas Medical Branch in December 1956, at which time a translumbar aortographic study was attempted without success. Antihypertensive therapy was instituted with equivocal results, the blood pressure remaining at levels around 160/110 mm. Hg.

In April 1959, selective arteriography of the renal arteries was performed.2 Premature arteriization of the right renal artery was apparent, with an area of stenosis and poststenotic dilatation in a small branch that entered the superior segment of the kidney (figs. 1 and 2).

On April 25, 1959, the right renal artery was exposed at laparotomy. The artery branched into several small vessels almost immediately after its point of origin from the aorta, and a small branch to the superior segment was stenotic at its origin from the larger vessel. A significant pressure gradient was found over the stenotic area (fig. 3). The area of stenosis was resected, and the vessel was reanastomosed primarily.

The patient’s postoperative course was characterized by a progressive drop in blood pressure to normotensive levels. The polyuria, polydipsia, and enuresis disappeared. Two years after the date of operation the blood pressure was 115/80 to 100/70. The patient is completely asymptomatic.

Case 2

A 58-year-old man was seen by his local physician because of persistent throbbing headaches of approximately 2 months’ duration. There was no history of hypertension. He was referred to the University of Texas Medical Branch for evaluation because of the headaches, a history of nervousness, 9 months of insomnia, and a blood pressure of 280/150 mm. Hg. At the time of physical examination, his blood pressure was 270/128 mm. Hg. A grade-I systolic murmur was audible over the base of the heart. At this time the heart was considered to be enlarged. Intravenous pyelograms were negative. On selective arteriograms of the renal arteries, bilateral aberrant renal arteries to the lower pole of each kidney were demonstrable.

In March 1960, laparotomy was performed. Aberrant renal arteries were found to be supplying both lower poles. There was a significant pressure gradient across the orifice of the right aberrant renal artery. The orifice was severely compromised by diffuse subintimal atheromatous thickening. Both aberrant renal arteries were 1.5 mm. in diameter, and were, therefore, considered to be too small for grafting. Silk ligatures were used to tie off both of the aberrant arteries.

Postoperatively, the blood pressure declined gradually, from 160/90 mm. Hg, without antihypertensive therapy. In the 9 months the patient has been followed, his blood pressure has varied from 180/90 to 210/100 without antihypertensive therapy.

Case 3

A 12-year-old Negro girl was first seen in February 1958, at the University of Texas Medical Branch, because of frontal headaches and left flank discomfort. On physical examination, blood pressure was 140/100 mm. Hg in all extremities, and there was grade-2 hypertensive retinopathy. Findings were normal on intravenous pyelography.

The patient received antihypertensive therapy with little benefit. Blood pressure ranged from 130/100 to 160/120 mm. Hg. On translumbar aortography in October 1958, opacification of both...
kidneys was normal, but the right renal artery was not visible. The blood pressure dropped to a level of 120/80 with bed rest, low-salt diet, and treatment with antihypertensive drugs.

Moderate caliectasis of the right kidney was discernible on intravenous pyelograms, and findings on urinalysis were consistent with pyelonephritis. After antibiotic therapy urine cultures were sterile. With ambulation the blood pressure again rose to hypertensive levels.

On selective renal arteriograms on April 4, 1960, the right renal artery was well visualized, and the lower pole of the right kidney did not fill (figs. 4 and 5). On subsequent injection, a small artery to the lower pole of the right kidney was visible.

On May 6, 1960, laparotomy was performed. A hypoplastic aberrant renal artery to the lower pole of the right kidney was found. There was no pressure gradient across the right main renal artery, but the small aberrant vessel accommodated only a 22-gage needle. Although there was a large pressure gradient between this vessel and the aorta, recordings were severely damped. The vessel was believed to be too small to permit revascularization, and segmental renal resection was not considered feasible at the time; hence, the aberrant vessel was ligated in the hope that conversion of the area of ischemia to one of infarction would alleviate the hypertension.

Blood pressure recordings 10 to 12 days post-operatively ranged from 130/90 to 118/70. During the 7-month follow-up period, the patient's blood pressure has remained 130/90. The continued mildly elevated blood pressure may indicate a zone of relative ischemia between the infarcted area and the adjacent kidney parenchyma.

**Discussion**

The concept of systemic hypertension secondary to renal ischemia is well accepted. Any process that causes ischemia of the renal parenchyma may produce systemic hypertension by the formation of the pressor substance, angiotensin. The kidney is divided into five distinct arterial segments, between which there is no appreciable anastomosis. Since aberrant renal arteries are truly segmental renal arteries with different origins, it is logical to conclude that vascular insufficiency of an aberrant renal artery may produce segmental renal ischemia and subsequent systemic hypertension. Indeed, cases of segmental renal ischemia causing systemic hypertension have been reported. A return of the blood pressure to normotensive levels has occurred after segmental resection of the affected kidney.

Partial occlusion of an aberrant renal artery that results in decreased blood supply to one or more renal segments may be congenital in origin, manifesting itself as an isolated stenosis of that artery. Case 1 is an example of right renal arterial morphology as seen by selective arteriography in case 1.
of such a stenosis. In addition, the entire aberrant vessel may be hypoplastic, as Howard et al.\textsuperscript{10} and Isaac et al.\textsuperscript{11} have reported (case 3). Segmental renal ischemia on the basis of partial occlusion of an aberrant renal artery may result from atheromatous encroachment upon that artery's lumen, and thus may occur as an acquired form. Gyori\textsuperscript{8} has reported 10 such cases. Our second case is an example of diffuse atheromatous deposition that caused constriction at the aortic orifice of an aberrant renal artery.

The effect of the most advanced lesion of arterial insufficiency, that is, infarction, is much less well delineated in relation to segmental renal involvement. Systemic hypertension rarely occurs as a result of infarction of an entire kidney,\textsuperscript{12} and, when it does occur, it is usually transient. The relationship between segmental renal infarcts and the production of systemic hypertension, is, however, much less clear. Gross\textsuperscript{13} stated that, in surgery for ureteropelvic obstruction secondary to an aberrant renal artery, it is possible to divide the offending vessel with impunity. He had seen only one case of hypertension thought to have occurred from this mechanism. Hoxie and Coggin\textsuperscript{14} were unable to correlate hypertension with segmental renal infarcts in their study of 205 cases; yet Arnold et al.\textsuperscript{15} have reported two cases of transient hypertension after ligation of aberrant renal arteries, and have suggested that such transient hypertension may be common. Numerous authors\textsuperscript{15-19} have reported cases of sustained severe systemic hypertension after segmental renal infarction. It seems, therefore, that complete occlusion of a segmental renal artery, specifically an aberrant renal artery, may result in no hypertension, transient hypertension, or sustained systemic hypertension, and, at present, there is no way to discern preoperatively whether a segmental renal infarct is the offending entity in the production of hypertension.

Until better diagnostic methods are devised, we consider that any hypertensive patient in whom no other cause for hypertension can be found, and who has, potentially, 15 to 20 good years left, should have the benefit of renal arteriography in an attempt to delineate the etiology of his hypertension. The technic of renal arteriography should be of such quality.
that all five renal arterial segments may be visualized.\textsuperscript{21} We have used primarily the technic of selective visceral arteriography\textsuperscript{2}—retrograde arterial catheterization—with good results. There is need, however, for defining more clearly the radiologic criteria for arterial narrowing that is sufficient to cause renal ischemia.

In the choice of surgical procedures for relief of hypertension secondary to segmental renal ischemia, revascularization of the ischemic area should be considered first. Morris et al.\textsuperscript{22} has emphasized the fact that the "ischemic" portion of the kidney may actually be the better-functioning portion, after revascularization, since it has been protected from the nephro sclerotic effect of the hypertension it produced. Revascularization of an ischemic renal segment may be accomplished in a number of ways. We have used primary resection of the stenotic area of an aberrant renal artery, as in case 1, and transplantation of the origin of an aberrant vessel, as in case 2. If the aberrant vessel in question is too small to consider revascularization, as in case 3, the artery may be tied off and the ischemic area converted to a frank infarct, or a segmental resection of that ischemic portion of the kidney may be made. A small incidence of hypertension after segmental renal infarcts is unavoidable; hence, ligation of the vessel may not relieve the hypertension. Total nephrectomy should, however, be avoided whenever it is possible.

**Summary**

Three cases of hypertension associated with vascular insufficiency of aberrant renal arteries are presented. Two patients were subjected to revascularization procedures. The types of pathologic processes associated with aberrant renal arteries in production of segmental renal ischemia are discussed. Considerations of diagnosis and therapy of such lesions are reviewed.

**References**


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One drawback of the present popular confidence in research is that the donors of research funds have overlooked or underestimated the importance of the medical education that will provide an adequate supply of good researchers in the future. The donors have also ignored the importance of giving first-rate research men today the salaries and tenure they deserve. As between donor and recipient, the relationship, especially in short-term grants, suggests a grim variant of the declaration that it is more blessed to give than to receive—it is certainly more comfortable. For the plain fact of the matter is that most of our medical schools often find that the full cost of research is not covered by the grants which are supposed to pay for it. The schools cannot afford any longer to accept the full moral responsibility, but only part of the full cost, of many research projects.—Alan Gregg, M.D., Challenges to Contemporary Medicine. New York, Columbia University Press, 1956, p. 72.
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