Total Renal Artery Occlusion: Recovery of Function After Revascularization

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Current trends in managing atherosclerotic renal artery stenosis favor medical therapy, on account of negative results from prospective trials of revascularization, such as CORAL and ASTRAL. One result of this trend has been encountering occasional patients with progressive disease, sometimes leading to total arterial occlusion. We illustrate a case of accelerated hypertension with complete renal artery occlusion in which the patient recovered function after surgical bypass and we review the clinical approach used and the advanced imaging modalities available to us. A high index of suspicion and careful radiologic imaging play important roles in selecting patients who may have residual function and may benefit from revascularization. This case illustrates an example whereby restoring renal artery perfusion for carefully selected patients can be life changing, with recovery of kidney function and improved blood pressure, pill burden, and overall quality of life.

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Am J Kidney Dis. 71(5): 748-753. Published online February 8, 2018.

doi: 10.1053/j.ajkd.2017.11.014

Teaching Cases focus on interpretation of pathology findings, laboratory tests, or imaging studies to educate readers on the diagnosis or treatment of a clinical problem.

Introduction

Occlusive atherosclerotic renovascular disease (ARVD) is a leading cause of secondary hypertension and renal ischemic disease.[1] One of the principal causes of renal arterial occlusion is atherosclerosis. The prevalence of ARVD is estimated to be 6.8% from community-based studies of people older than 65 years using an ultrasound threshold of elevated peak systolic arterial velocities suggestive of 60% occlusion.[2] Some patients with progressive vascular occlusion develop advanced kidney failure on that basis, and recently concern has been raised for a possible increase in ARVD-related kidney failure.[3] Considering that many patients have renovascular disease undiagnosed, it is difficult to estimate the real incidence of end-stage kidney disease from ARVD.

We know from the coronary circulation that gradual complete arterial occlusion can occur with preservation of organ viability due to collateral arterial supply.[4] Studies using blood oxygen level–dependent magnetic resonance (BOLD-MR) demonstrate that renovascular occlusion of 70% to 80% ultimately produces demonstrable cortical ischemia.[5] This hypoxia can lead to inflammatory changes in the kidney, resulting in fibrosis and subsequent decreased kidney function.[6,7] Salvage of such a hypoxic kidney must be achieved before failure of compensatory mechanisms. We present the case of a 69-year-old patient who developed total occlusion of her left renal artery and underwent a bypass revascularization procedure of the occluded vessel, resulting in improvement in blood pressure and recovery of kidney function sufficient to be removed from the transplant wait-list.

Case Presentation

Clinical History and Initial Laboratory Data

A 69-year-old white woman was referred to our Hypertension Clinic for evaluation of accelerated hypertension and decreased kidney function. She had hypertension diagnosed 10 years earlier and was maintained on a thiazide diuretic with good blood pressure control. She had no other significant medical conditions. Serum creatinine concentration at that time was 1.4 mg/dL.

Six months before presentation, the patient was hospitalized near home with a hypertensive emergency. Blood pressure was 250/110 mm Hg associated with a refractory headache, confusion, and creatinine concentration elevation to 2.5 mg/dL. Protein-creatinine ratio was 1.6 g/g. A kidney ultrasound identified probable left renal artery stenosis. An angiogram identified a patent right renal artery, but total occlusion of the left renal artery. No endovascular procedure was possible. Her antihypertensive therapy was intensified, with resolution of central nervous system symptoms. However, blood pressure continued to be labile and difficult to control, and kidney function progressively deteriorated. She underwent biopsy of the right nonstenotic kidney, which showed evidence of severe arteriosclerosis, focal segmental glomerular scarring, and mild effacement of podocyte foot processes suggestive of secondary focal segmental glomerulosclerosis, likely

secondary to vascular disease. She came to Mayo Clinic for a second opinion.

Upon presentation to our institution, the patient's blood pressure averaged 165/79 mm Hg despite administration of amlodipine, 10 mg, daily; carvedilol, 12.5 mg, daily; clonidine, 0.1 mg, twice a day; and torsemide, 10 mg, daily. Initial blood work showed a creatinine concentration of 3.5 mg/dL, plasma renin activity of 6.3 ng/mL/h, and aldosterone concentration of 10 ng/dL. Urinalysis showed no hematuria or cellular elements. She had proteinuria with protein excretion of 3.5 g/d and a measured creatinine clearance of 13 mL/min/1.73 m^2. She was started on treatment with a low dose of losartan, and her other medications were adjusted. She was evaluated and accepted as a transplantation candidate. Blood pressure continued to be difficult to control. The possibility of removal of the left kidney as a "pressor" kidney was considered.

Additional Investigations

An ultrasound of the kidneys with spectral Doppler analysis was obtained. The origin of the left renal artery was not well visualized, but dampened tardus parvus Doppler waveforms in the rest of the left renal artery and segmental arteries were consistent with the proximal left renal artery occlusion or high-grade stenosis. The right renal artery was widely patent. Remarkably, cortical thickness was relatively preserved in the left kidney, with overall length of 10.1 cm as compared to the right kidney of 12.6 cm. The segmental resistive indexes were also normal, at 0.53 (Fig 1B). A computed tomography angiogram confirmed total occlusion of flow in the proximal left renal artery, but with visualization of distal vessels and kidney (Fig 1A).

Renal BOLD-MR imaging was performed before and after intravenous furosemide administration. The BOLD-MR image of the left kidney showed elevated cortical R2* suggestive of cortical hypoxia and increased fractional tissue hypoxia (Fig 2) with a modest response to furosemide. Unexpectedly, a nuclear mercaptoacetyltriglycine (MAG3) renal scan identified that the majority of glomerular filtration (57.1%) appeared to derive from the occluded left kidney, while the other 42.9% came from the right kidney.

Diagnosis

Accelerated hypertension associated with total left renal artery occlusion with evidence of residual function in a chronically occluded left kidney.

Clinical Follow-up

The patient was evaluated for possible left renal artery revascularization with a focused computed tomography angiogram that confirmed left renal artery occlusion proximally (Fig 1). A viable distal vessel was identified, with patency of most intrarenal arterial branches. She underwent left abdominal aortorenal bypass using a left nonreversed saphenous vein. At the time of the surgery, creatinine concentration was 4.8 mg/dL with blood pressure of 165/82 mm Hg. Two weeks after the surgery, blood pressure was 122/76 mm Hg without losartan and a reduced dose of diuretic, and creatinine concentration had decreased to 2.9 mg/dL, with electrolyte concentrations within the reference ranges. After 3 months, blood pressure was 108/56 mm Hg with occasional symptoms of dizziness, and antihypertensive drugs were further reduced to amlodipine, 5 mg, daily; metoprolol succinate, 25 mg, daily; and torsemide, 2.5 mg, daily (Fig 3). Creatinine concentration had stabilized at 2.9 mg/dL, and proteinuria had decreased to protein excretion of 500 mg/d. After 6 months, anemia had resolved, appetite improved, and measured glomerular filtration rate was 26 mL/min/1.73 m^2.

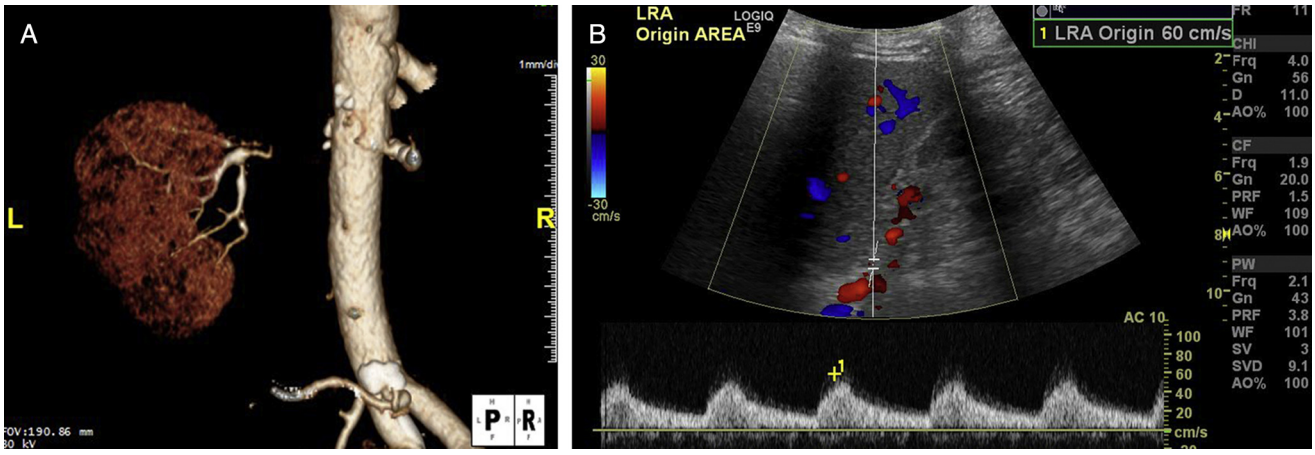

Figure 1. Renal imaging. (A) Posterior view computed tomography angiogram with total occlusion of flow in the proximal left renal artery (LRA) with visualization of distal vessels and kidney by angiography suggestive of evidence of preserved distal perfusion. Collaterals cannot be visualized. (B) Ultrasound of the LRA with spectral Doppler analysis shows a waveform with a delayed upstroke and a dampened tardus parvus.