Challenging Stent Placement in the Renal Artery

Analyzing red flags for treating renal artery stenosis.

BY JAMES A. M. SMITH, DO

When asked to submit one of my most difficult renal cases, I knew immediately which case to present. It was 1992, and as coprincipal investigator for the Johnson & Johnson (Miami, FL) PS-204 trial, I had to be proctored for the first three cases. At the Wichita Heart Center, we were very tuned in to renal artery stenosis (RAS) and its viability for treating resistant hypertension and progressive renal insufficiency.[1] Unfortunately, the lesions often recurred, and elastic recoil after balloon angioplasty alone was almost always predictable. For that reason, we were extremely eager to be part of this trial, offering balloon-expandable stents to provide better scaffolding for the angioplasty site.

It is well known that most atherosclerotic lesions involving the renal arteries begin in the juxtarenal aorta,[2] and they are easily recognized by looking at the aorta at the level of the renal arteries. Typically, the aorta shows some degree of calcification near the renal artery origin,[3] and theoretically, we felt that the stent must extend into the aorta itself to cover the lesion adequately. Atherosclerotic disease always involves the first centimeter of the renal artery, as opposed to fibrous dysplasia, which occurs beyond the ostium.[4]

PATIENT DETAILS

The patient was a 79-year-old woman who had never smoked. Her blood pressure was poorly controlled, and an electrocardiogram showed she had left ventricular hypertrophy.

She was on four medications for hypertension, including a loop diuretic, calcium channel blocker, beta blocker, and central alpha-adrenergic inhibitor. Her serum creatinine was 1.9, and her blood urea nitrogen was 37. Her body mass index was 25, and she was in normal sinus rhythm with a ventricular heart rate of 67 bpm. Her arm blood pressures were equal at 182/84. She had no known diabetes, but she was recently admitted with an acute hypertensive crisis in pulmonary edema, the second such episode in 6 months. Her cholesterol was elevated with an LDL of 122 and triglycerides of 114 mg/dL. Her HDL was 41 mg/dL, and she did not have proteinuria or other abnormalities on her urinalysis.

The proctor for the case was a radiologist from the San Antonio Veterans Administration Medical Center. The case

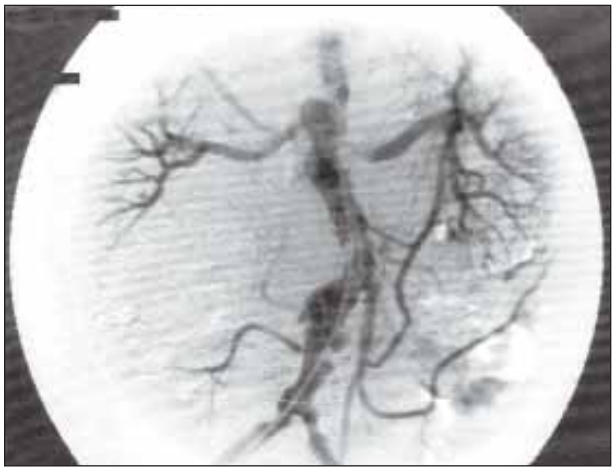

Figure 1. The aorta with a solitary renal artery supplying each kidney. There is advanced calcification in the aorta and considerable tortuosity of the infrarenal segment.

Figure 2. The right renal artery showing severe occlusive disease.

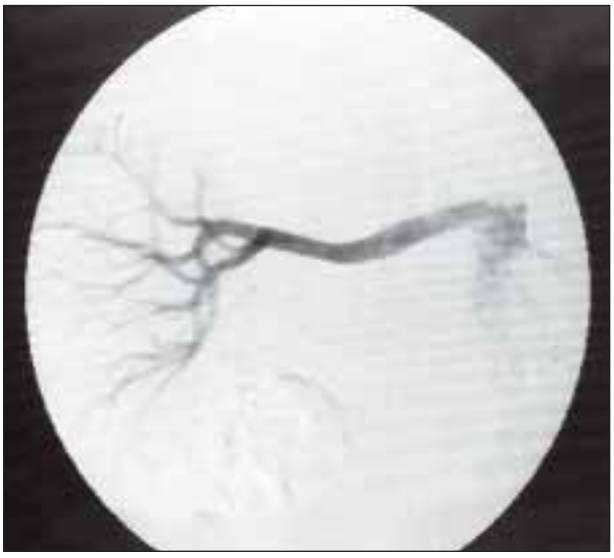

Figure 3. The right renal artery after implantation of the PS 204 articulated stent. There is no residual narrowing and the stent position is perfectly placed in the aorta. The nephrogram is preserved.

was presented to him along with four others. However, he rejected it on the basis of several issues, but primarily because of a hostile aorta due to tortuosity, diffuse calcification, and potentially high risk for atheroembolization and renal infarction during the intervention.

Figure 1 shows the aorta with a solitary renal artery supplying each kidney. There was advanced calcification in the aorta and considerable tortuosity of the infrarenal segment. The degree of severity of occlusive disease in the right renal artery is shown in Figure 2. The downward angulation of the left renal artery makes a femoral retrograde approach somewhat more difficult, and consideration to a percutaneous brachial entry was given for staged treatment of the left renal artery stricture.

Figure 3 shows the right renal artery after implantation of the PS 204 articulated stent. There is no residual narrowing, and the stent position is perfectly placed in the aorta. The nephrogram is preserved.

Unfortunately, because the case was performed in 1992, I could not provide pictures after the left renal artery stent placement, which was completed the next day using a femoral approach as well.

DISCUSSION

One must realize that this was one of the first renal artery stent implantations that we performed, and that the tools utilized were far less user-friendly than the smaller delivery systems that are currently available. However, considering the alternative to renal intervention, which was performed for the purpose of improved blood pressure control and for maintenance of renal mass, there was little alternative to this risky intervention. For that reason, we waited until our proctor left, having signed off on our three proctored cases. The case was performed 1 day later.

> "First and foremost, one must consider the alternatives to treatment and understand the natural history of atherosclerotic RAS."

The case illustrates several interesting issues. First and foremost, one must consider the alternatives to treatment and understand the natural history of atherosclerotic RAS. In retrospect, flash pulmonary edema is now a recognized complication of RAS, and in the setting of recurrent admissions for pulmonary edema, the threshold for searching for RAS is now lower than before. This presentation should alert the clinician to the possibility of underlying RAS.

From the technical standpoint, the tortuosity of the infrarenal aorta may interfere with the engagement of the delivery system in the renal ostium and make the case more technically challenging. One must always consider the nature of the atherosclerotic lesion at the level of the ostium, because certain lesions are very soft and friable, making it easy to push plaque into the artery, resulting in a total occlusion and renal infarction. If staining of the aorta occurs with a small contrast injection, one must be extremely cautious about engaging the

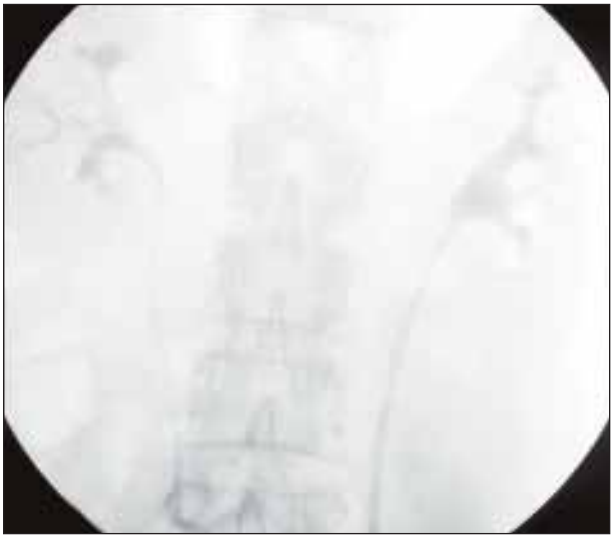

Figure 4. Nephrogram after 45 minutes of contrast, showing late hyperconcentration of contrast in the left kidney. This is frequently a finding in RAS.

ostium.

Figure 4 shows another caveat with regard to identifying patients who may have underlying RAS. The hyperconcentration in the left renal artery is a clinical clue that RAS is present on the left. This finding was originally described in the workup of these patients with the hypertensive rapid sequence intravenous pyelogram as one of the possible criteria for diagnosing RAS. The finding is especially important after an intervention in another vascular territory (eg, percutaneous transluminal coronary angioplasty). If the intervention requires more than 30 minutes to complete, a brief view of the nephrograms is literally a free piece of valuable information. If asymmetry is visible, it may justify flush aortography and select bilateral renal angiography. The kidney with dye retention may frequently be the one where renal artery narrowing is present.

This approach will lead to a higher yield of positive renal angiograms than the so-called drive-by renal angiogram, which has been recommended by some cardiologists.

CLINICAL ASSESSMENT

The patient lived another 7 years but ultimately died from a colon malignancy. She had no further episodes of pulmonary edema, and she did not experience any additional vascular territory insults. Her blood pressure was eventually controlled on three different medications, and her quality of life was notably improved.

The issue of renal artery intervention remains controversial among nephrologists. However, it is known that approximately 30% to 40% of patients on chronic hemodialysis have renal artery atherosclerosis as the etiology for their renal failure. Many of these patients are never evaluated for the condition, which is easily detected on renal duplex examination. With the minimally invasive nature of CT angiography, the diagnosis can be confirmed on an outpatient basis using very little amounts of contrast. Another point of contention is that the salvagability of renal function is generally limited once renal atrophy beyond 7 cm to 8 cm in pole-to-pole length of the kidney is seen.

To diagnose RAS, one must have a certain degree of clinical suspicion based on other cardiovascular risk factors, the degree of satisfaction with the patient's blood pressure control, the serum creatinine level, and the number of medications required for adequate control. To optimize treatment and avoid dialysis, we must engage in a concerted effort with the nephrologists to detect this problem and to intervene for two specific indications: the control of hypertension and the preservation of renal mass.[5,6] ■

The author wishes to thank Greg Downing, MD, for his input and assistance with this case.

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