Renal Artery Occlusion Due to Angiotensin-Converting Enzyme Inhibition and Reintervention for in-Stent Stenosis

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Renal artery angioplasty and stenting is the first-line therapy for many patients with renal insufficiency or hypertension caused by atherosclerotic renal artery stenosis. Unfortunately, like all stents, renal artery stents are prone to restenosis. When this process occurs, repeated intervention is usually necessary.

CASE REPORT

A 67-year-old woman with a solitary functioning kidney underwent uneventful renal artery stenting for hypertension and renal insufficiency. Both blood pressure and renal function improved and she remained well for 2 years. She was then admitted to an outside hospital with chest pain. She was hypertensive on admission and placed on an angiotensin enzyme inhibitor. While in the hospital being treated for myocardial infarction, she developed flank pain and became anuric. A computed tomography scan was obtained (not shown) revealing thrombosis of the solitary renal artery stent.

The patient was referred to Interventional Radiology and catheter-directed thrombolysis was initiated using 1 mg/h of tissue plasminogen activator. After 24 hours, the renal artery was partially recanalized revealing a high-grade in-stent stenosis (Fig. 1A). The outside report noted that there was "great difficulty passing angioplasty balloons beyond the stenosis" and dilatation led to "repeated balloon rupture" (Fig. 1B). Antegrade flow remained poor. Thrombolysis was continued for a total of 48 hours with minimal improvement. The patient was then transferred to our hospital for additional evaluation. She remained anuric.

The patient was referred to our Interventional Radiology section and abdominal aortography was per-

formed (Fig. 2A), revealing complete rethrombosis of the renal artery stent. Five thousand units of heparin were administered intra-arterially and the pigtail catheter was exchanged for a 5-Fr VS-1 catheter and 0.035-inch Bentson guide-wire (Boston Scientific, Natick, MA). Initial attempts to cross the stent were unsuccessful. A magnified fluoroscopic image demonstrated the proximal portion of the stent crimped closed at the renal artery origin (Fig. 2B).

The Bentson wire was exchanged for a TAD-II guide wire (Mallinckrodt, Hazelwood, MO) that was used to probe the stent. The orifice of the stent could not be catheterized because it was crimped shut. Instead, the wire preferentially passed through the interstices of the stent and into the more distal renal artery. The decision was made to attempt to create a rent in the stent interstices. A thrombectomy catheter was then used to remove a clot from the main renal artery. A 5-mm high-pressure balloon catheter was advanced into the stent and used to create a large rent in the stent interstices. Final angiogram revealed antegrade flow through the main renal artery and into the lower pole of the right kidney (Fig. 2C).

The patient began making small amounts of urine but remained on hemodialysis.

DISCUSSION

Angiotensin-converting enzyme (ACE) inhibitors are known to exacerbate renal insufficiency in patients with renovascular hypertension. In this case, initiation of this therapy probably also incited renal artery thrombosis due to systemic hypotension. This complication has been described in patients with bilateral renal artery stenoses,

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Figure 1 Outside hospital angioplasty of right in-stent renal stenosis. (A) Initial angiogram shows proximal in-stent renal stenosis. (B) Angiogram after angioplasty shows irregularity of the proximal aspect of the stent.

Figure 2 Angioplasty of stent interstices. (A) Initial aortogram shows complete thrombosis of right renal artery. (B) Magnified fluoroscopic image shows proximal aspect of renal stent crimped closed. (C) Final angiogram shows antegrade flow through rent created in stent.

a high-grade stenosis in a solitary kidney, and a transplant kidney with a high-grade renal artery stenosis. ACE inhibition is best avoided in patients with renovascular hypertension, particularly in a patient with history of treated renovascular hypertension.

Renal angioplasty has become the initial treatment of choice for atherosclerotic renovascular hypertension in many hospitals, including my own. Because atherosclerotic lesions tend to be ostial, renal stents are used in the overwhelming majority of procedures. These stents enable a very high technical success rate but may later suffer from in-stent restenosis, which can significantly limit the long-term patency of the intervention. At 1 year, 11 to 39% of stents suffer from restenosis. This process is usually caused by migration of smooth muscle cells into the zone of vascular injury that leads to vessel remodeling and restenosis. Myointimal hyperplasia can be exacerbated by incomplete stent expansion or incomplete plaque coverage.

The optimal treatment of stent restenosis is currently unclear. Strategies include angioplasty, cutting balloon dilatation, laser-assisted angioplasty, rotational atherectomy, surgical end-atherectomy, restenting, and brachytherapy. In my hospital, we typically use simple angioplasty for stent redilatation, reserving cutting balloons for refractory cases. Of course, a better strategy would be to inhibit the process altogether, and drug eluting or β -emitting stents are being investigated for this purpose. Bax et al reported the results of repeat interventions in 15 patients with 20 stenosed renal stents. Eighteen stents were treated with angioplasty and two with a second stent. Cumulative patency rates were 93 and 76% after 6 and 12 months, respectively. Renal function remained stable or improved in 80% and hypertension was improved or cured in 47% after 1 year. There were two complications in this series: one patient sustained an iatrogenic dissection, which progressed to a pseudoaneurysm and another patient suffered transient contrast-induced nephropathy.

Repeated intervention on renal stents may be technically difficult due to the fact that the stent typically protrudes from the renal artery several millimeters into the aorta. Thus catheterizing the stent can be quite challenging and often more difficult than negotiating a severe renal artery stenosis. This is especially true with recurrent in-stent stenosis. In this case, the initial reintervention at the outside hospital appears to have been complicated by the guide wire passing through the interstices of the existing stent (Fig. 3) instead of through the stent orifice. Thus when angioplasty was performed, the orifice of the stent was crimped shut and several angioplasty balloons ruptured. To my knowledge, this complication has not been previously reported. Nonetheless, in my experience, guide wire passage through stent interstices is not rare and the lesson

Figure 3 Illustration showing guide wire passing under stent and through stent interstices in the more distal renal artery.

learned is clear. Whenever catheterizing a previously deployed stent, it is important to remain vigilant. A guide wire can easily pass through one of the interstices of a stent, sometimes more readily than through the stent orifice. Sometimes the only indication that this has occurred is when difficulty is encountered in passing a catheter across the stent. A catheter is much more difficult to pass through stent interstices and moderate resistance is common.

My decision to expand the rent in this patient even further was a last-ditch effort to increase flow to the kidney. Another option would have been renal bypass but this was thought to be a poor choice given the patient's recent myocardial infarction and thrombosis of the renal artery. The warm ischemia time for a kidney is thought to be several hours; however, a patient with a severe renal artery stenosis may develop enough collateral arterial supply to preclude infarction even with a main renal artery occlusion. Despite the fact that the patient in this report had been anuric for more than 24 hours with a total occlusion of the renal artery, recanalization of the artery restored enough function for the kidney to begin making urine again.

In summary, ACE inhibitors are best avoided in a patient with a solitary kidney and previously treated renal artery stenosis. Furthermore, when reintervening on stenotic renal artery stents, it is imperative to ensure that catheterization is through the orifice of the stent and not through one of the stent interstices.

SUGGESTED READINGS

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