Renal Ostial Angioplasty and Stenting. Part 2: Pitfalls and Complications

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Renal ostial stenting (ROS) is the most common endovascular intervention for treatment of atherosclerotic renal artery stenosis. This article is the second in a two-part series dedicated to pitfalls and complications of renal stenting.

PREPROCEDURE

Patients should be seen in the clinic. One of the significant problems associated with renal revascularization is that despite a technically successful procedure, hypertension or renal insufficiency is unlikely to be “cured,” it may or may not improve, or it may even get worse. Therefore, it is critical to manage expectations of both physicians and patients.

IMAGING

The renal arteries typically do not arise at a 90 degree angle from the aorta in the axial plane. Therefore, the image intensifier should be angled to best depict the proximal aspect of the artery in profile. Cross-sectional imaging is reviewed with particular attention to the angle of the renal arteries with respect to the aorta. This information is used to properly position the image intensifier during the procedure. Because a renal stent should be positioned to slightly extend into the aorta, if the image intensifier is not orthogonal to the ostium, a stent may be positioned too far distally into the artery (Fig. 1).

PROCEDURE

Renal Insufficiency

Because this intervention is often being performed for declining renal function, patients with renal insuffi-

ciency are often the rule rather than the exception. In my practice, I use carbon dioxide routinely for this intervention. The use of carbon dioxide has been described in a previous issue.¹ If visualization is sub-optimal with carbon dioxide, I will use dilute iso-osmolar contrast. Even in difficult cases, the entire procedure can typically be performed using no more than 10 mL of iso-osmolar contrast when carbon dioxide and cross-sectional imaging studies are utilized.

Pitfalls with Renal Artery Catheterization

Renal artery catheterization is typically the most difficult and “dangerous” portion part of the procedure. As described in the first part of this article, I favor a short recursive catheter such as a visceral selective 1 (Cook Medical, Bloomington, IN), which is formed and then advanced cephalad with a soft tipped crossing guide wire to select the artery of interest. This catheter usually works well even in very high-grade stenoses and occlusions (Fig. 2).

I try to avoid C-shaped catheters such as a Cobra 2 or RC-1 for this intervention. These latter types are typically used to select the vessel of interest and then a guide wire is advanced out of the catheter into the vessel. In nondiseased vessels, this works well, but in arteries that have significant ostial plaque, the tip of the catheter may become embedded into the plaque rather than the lumen of the vessel. If this occurs, advancing any guide wire out of the catheter (even a relatively soft tipped one such as a Bentson wire) can cause proximal plaque disruption or even worse, a renal artery dissection (Fig. 3). (Incidentally, using a recursive catheter *without* a leading guide wire can do the same thing.)

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Figure 1 Importance of image intensifier positioning. Bilateral renal artery stent insertion at outside institution referred for recurrent hypertension 8 months after intervention. (A) Aortogram in slight left anterior obliquity shows ostial stenosis incompletely covered by deployed stent (arrow). (B) Unenhanced computed tomography (CT) scan shows incomplete coverage of renal artery ostium. Notice the stent may appear to be positioned at the ostium on frontal fluoroscopic image as the posterior proximal edge of the stent will project over the aorta.

Figure 2 Recanalization of nearly occluded right renal artery. (A) Aortogram shows near complete right renal artery occlusion. (B) Fluoroscopic image of catheterization using short recursive catheter. (C) Selective renal angiogram after successfully crossing occlusion. (D) Final image after stent insertion.

Figure 3 Illustration showing plaque disruption using C-shaped catheter. (A) C-shaped catheter in the aorta. (B) Catheter tip embedded in renal ostial plaque. (C) Guide wire being advanced out of catheter. (D) Plaque disruption by guide wire.

CATHETERIZING HIGH-GRADE STENOSES OR OCCLUSIONS

Occasionally, catheterization is unsuccessful using a recursive catheter and soft-tipped guide wire. If a wire is able to be advanced beyond the lesion, but not the catheter, I ask the patient to cough or take in a deep breath while maintaining gentle traction on the catheter. If this fails and I still have wire access, I switch to a 4 French hydrophilic catheter and position the guiding sheath closer to the ostium for added support. If I am able to pass the catheter beyond the lesion, I attempt

more distal catheterization. If this fails or wire access is lost, I switch to a larger recursive catheter such as a Simmons 2, which provides more mechanical advantage. It is crucial to attempt to determine why the shorter recursive catheter cannot be advanced into the artery—sometimes this indicates dissection rather than simply a very high-grade lesion. I usually attempt to spin the wire in between my fingers and rely on both tactile sensation and the configuration of the guide wire to make this assessment. Wires in a false channel often assume a spiral orientation.

If the Bentson wire cannot be advanced across the lesion, I usually switch to a soft tipped hydrophilic guide wire (angled Glidewire[®], Terumo Medical Corporation, Somerset, NJ). This is a “high risk–high reward” wire in renal interventions and has a steep learning curve. If used incorrectly, iatrogenic dissection is not unusual using this guide wire (neither is capsular perforation) so all manipulations should be done very gingerly. If this fails, I switch to a smaller diameter wire or a hybrid wire such as a TAD II[®] (Abbott Cardiovascular Systems, Inc., Santa Clara, CA).

CAUDALLY ORIENTED RENAL ARTERIES

The majority of renal arteries can be catheterized from a femoral approach, although it is occasionally necessary to use a brachial access. I perform ultrasound-guided brachial puncture and use a long slightly tapered 6F sheath to perform intervention (Fig. 4). A monorail or “rapid exchange” system is advantageous for this intervention. It is important to avoid awkward “exchange length” guide wires.

IATROGENIC DISSECTION

This complication has been discussed in a previous Morbidity and Mortality (M&M) article in this journal.² In my experience, dissection is most common during

catheterization of the renal arteries, although it can also occur after aggressive angioplasty and/or stent insertion. It tends to occur more commonly with high-grade or heavily calcified lesions. If the dissection is not flow limiting, observation may be pursued. Otherwise, if access to the true lumen can be secured, the dissection flap should be tacked down with prolonged balloon dilation or a bare metal stent.

EMBOLIC PROTECTION DEVICES

Given the rapidly evolving types and numbers of embolic protection devices (and my relatively limited experience using them), a discussion of the pitfalls and complications of these devices is beyond the scope of this article. Clearly, passage of some of these devices into the distal renal artery causes more emboli than they prevent and can lead to dissection, spasm, or distal renal artery injury, similar to any other device. Some coronary devices may be undersized in some patients and cannot address emboli with early branching renal arteries. As discussed in Part 1 of this article,³ I currently prefer the SpiderRX (ev3 Inc. Plymouth, MN) embolic protection device because catheterization can be achieved with any guide wire and catheter combination.

Pitfalls to Maintaining Wire Access

After successfully crossing the lesion, the soft tipped crossing wire is switched for a working wire for the remainder of the procedure. If using conventional 0.035 systems, I use a Rosen wire as a working wire because it has a short tip and rigid body. If using smaller systems, I use a 0.018 inch McNamara wire, which has similar attributes. The tip of the working wire should be maintained in the distal main renal artery or in a proximal 2nd order branch. There is natural tendency to try to advance as much wire as possible into the renal artery to maintain wire access. **DO NOT DO THIS.** Smaller wires in particular can easily perforate the renal capsule leading to a large perirenal hematoma or occlude the distal vessel causing a renal infarct (Fig. 5). This complication may be fatal, particularly if the perforation is unrecognized and treatment is delayed.⁴

RENAL CAPSULE PERFORATION

Capsular perforation should be treated immediately when recognized (Table 1). If an angioplasty balloon has already been inserted, it should be reinflated to decrease bleeding. Immediate hypotension is usually due to a vasovagal response rather than massive blood loss. Intravascular fluids should nonetheless be administered. Severe bradycardia and hypotension may be treated with atropine (0.5 mg) if necessary. The perforation should be treated with superselective embolization (microcoils combined with Gelfoam [Pfizer, Inc., New

Figure 4 Renal stent insertion via brachial artery approach. (A) Fluoroscopic image shows stent deployment. (B) Final renal angiogram.

Figure 5 Pitfalls of distal placement of guide wire during renal intervention. (A) Aortogram showing distal infarct (arrow) in patient with previous renal angioplasty. (B) Renal angiogram in different patient obtained during renal stent insertion showing tip of guide wire coiled in distal branch of renal artery, perforating capsule. (C) Follow-up computed tomography (CT) scan of patient in (B) showing large perirenal hematoma.

York, NY] pledgets are suitable) and heparin should be reversed with protamine. In the event of endovascular treatment failure, surgical treatment will be mandated and even in the event of successful endovascular treatment, surgical consultation is advisable.

Table 1 Management of Renal Capsular Perforation

1. Balloon tamponade
2. Reverse anticoagulation
3. Treat hemodynamic instability (type and cross if not already done) with intravenous fluids $\pm$ atropine 0.5 mg
4. Embolization with coils and Gelfoam pledgets
5. Surgical consultation

HEPARIN REVERSAL

Be aware that protamine allergy may exist, especially in diabetic patients previously exposed to NPH (neutral protamine Hagedorn) insulin. Because heparin has a relatively short half-life, dosing of protamine is variable based on when heparin has been administered. In most cases, heparin will have been given between 30 minutes and 2 hours of the complication so ~ 0.5 mg of protamine per 100 units of heparin is administered (longer than 2 hours would be half the dose above). In a typical procedure, 5000 IU heparin are administered so ~ 25 mg of protamine would be given as a reversal agent. Protamine is given by *slow* intravenous injection with the calculated dose injected into a 100 mL 0.9% normal saline and run at 10 mL per minute. Protamine should not be mixed with other drugs and maximum dose is 50 mg.

Pitfalls Related to Stent Deployment

STENT DISPLACEMENT

This complication was much more common when operators mounted expandable stents on balloons themselves

prior to deployment. Current premounted stents track very well around acute curves and are rarely displaced from balloons. Rarely, when using a very short stent in an eccentric stenosis, the stent may “slip” or “watermelon seed” proximally or distally. Although a detailed discussion of

Figure 6 Illustration showing strategy to capture displaced renal stent. (A) Renal stent displaced off of balloon in renal artery. (B) Snare advanced over balloon and guide wire. (C) Snare capturing stent. (D) Snare pulling stent proximally.

Figure 6 (Continued) (E) Stent deployment in ipsilateral iliac artery.

this complication is beyond the scope of this article, the general strategy is to pass a snare over the wire alongside the balloon and lost stent. The stent may be captured using the snare and then repositioned with the angioplasty balloon catheter. If it cannot be removed via the sheath, it can usually be deployed in the distal renal artery or ipsilateral iliac artery (Fig. 6). It may be necessary to exchange the indwelling sheath for a larger one to perform retrieval.

RENAL ARTERY RUPTURE

Renal artery rupture is a feared complication that occurs rarely. Like any other iatrogenic arterial rupture, patients typically complain of pain which continues after the angioplasty balloon is deflated. The treatment algorithm is the same as for renal capsular perforation except that the rupture is usually treated with a covered stent instead of embolization. A surgical consultation is warranted. The most important aspect of dealing with this complication is recognizing it early! The angioplasty balloon should be reinflated across the rent for ~5 to 10 minutes followed by repeat angiography to assess the injury. Heparin should be reversed with protamine unless the patient has a known allergy and a covered stent can be prepared in the event balloon tamponade is unsuccessful. Currently, we stock a balloon expandable covered stent (iCast[®], Atrium Medical Corporation, Hudson, NH) for this complication. This is a stainless steel stent covered with ultrathin polytetrafluoroethylene (PTFE), which can be slightly overdilated as necessary. It comes in

a variety of diameters (5 to 10 mm) and lengths (16, 22, 38, 59 mm). For renal artery intervention, a 5 to 6 mm covered stent may be deployed via a 6F sheath.

If extravasation is arrested, close imaging follow-up is warranted to exclude development of pseudoaneurysms. If bleeding continues despite covered stent insertion, the angioplasty balloon should be reinflated across the rent; urgent surgical intervention is necessary. In general, a kidney becomes nonviable after 1 to 2 hours of total circulatory arrest; kidneys that are chronically ischemic may tolerate longer periods of proximal renal arterial occlusion, especially if collateral flow to the kidney has developed.

POSTPROCEDURE PITFALLS

Renal Artery Thrombosis

Renal arterial thrombosis is a rare complication and may indicate an unrecognized renal artery dissection. It is important to realize that renal dissection may occur during catheterization, balloon angioplasty, or stent insertion. Other possible etiologies of acute thrombosis include arterial perforation or even rupture. One special circumstance that may lead to thrombosis is reintervention on a previously deployed stent. Because most stents are positioned with their trailing ends in the aorta, during catheterization, a guide wire may be inadvertently advanced through the interstices of a stent and then into the more distal renal artery. Balloon angioplasty in this circumstance may lead to folding the stent at the renal artery orifice and subsequent thrombosis. This complication has also been subject of prior M&M article in this journal.⁵

Acute renal arterial thrombosis may be treated with mechanical or pharmacologic thrombolysis or both. As noted above, in general, a kidney becomes nonviable after 1 to 2 hours of total circulatory arrest, but kidneys that are chronically ischemic may tolerate longer periods of proximal renal arterial occlusion. Given the need for rapid resolution of occlusion, mechanical methods are desirable. Percutaneous aspiration thrombectomy has been used for many years and is a simple and inexpensive method to debulk thrombosis and reestablish flow in the artery.⁶ This technique is performed using a thin-walled end hole catheter or guiding sheath (6 or 7 French) embedded in the thrombus. Continued suction is applied with a 30 to 60 mL syringe while pulling the catheter into a femoral sheath. Remember that a smaller syringe generates higher “pushing pressure” on inflation while a larger syringe provides more “suction.” Wire access can be maintained using a Tuohy-Borst Y-shaped adaptor. Intrarenal vasospasm is common so liberal use of vasodilators (nitroglycerine 200 µg aliquots given intraarterially) is recommended. Suction thrombectomy may be combined with catheter directed thrombolytic infusion.

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SUGGESTED READINGS

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