Open surgical treatment of renovascular disease changed with the introduction of renal artery angioplasty and stenting. More effective antihypertensive medications have also changed the practice. At Mayo Clinic, the number of primary open renal artery reconstructions for atherosclerotic disease decreased from 80 to 120 per year prior to 2002, to about 30 to 40 per year now. Most of these reconstructions are complex, and are relegated to patients with recurrent in-stent stenosis for atherosclerotic disease, complex fibromuscular disease, renal artery aneurysm, children and adolescents with renal artery stenosis, or renal reconstructions needed as part of aortic repair for aneurysms or occlusive disease.

The primary indication for intervention for atherosclerotic renal artery stenosis is failure of medical therapy. Intervention currently is offered to patients with accelerated, resistant, or poorly-controlled hypertension combined with high-grade renal artery stenosis and renal function decline. Other indications include cardiac destabilization syndromes such as flash pulmonary edema, congestive heart failure, and unstable angina. The most common indication for open surgical renal revascularization in our practice is failure of endovascular interventions. Most patients with stent failure have had multiple endovascular interventions, and the stents often extend to the bifurcation of the main renal artery. Open repair is challenging because of periarterial inflammation around the stent, and the need to carry a bypass to the distal main renal artery or its primary branches.

Between 1998 and 2013, 878 open renal artery reconstructions were done at Mayo Clinic for a variety of pathologies or reasons. Among these patients, 53 had reconstructions done subsequent to a failed endovascular intervention, of which 37 (70%) had 41 kidneys treated specifically for stent failure. Twenty-six were female, 11 were male, and the mean age was 63 ± 10 years. Operative reconstruction included 28 aortorenal bypasses, of which 18 were prosthetic, 9 were vein, and 1 was hypogastric artery. Two had renal arteries directly reimplanted onto the aorta. Eight kidneys were reconstructed from the hepatic, iliac, or gastroduodenal artery. Two others required ex vivo reconstructions, and the remaining patient had an endarterectomy and removal of the stent. Conduit for bypass was based on size of the renal artery. Saphenous vein or hypogastric artery was used as a conduit when the reconstruction was carried to the primary branches.

The complexity of the reconstruction determines the method of renal protection. Some patients with solitary kidneys or chronic kidney disease who require reconstruction carried to the primary branches, have a modified ex vivo technique used to minimize warm ischemia time. As described by the Wake Forest group, the renal vein is clamped, the renal arteries are divided, and a small venotomy is made in the renal vein. Collateral blood flow to the kidney via perireteral vessels can be controlled with a large silastic vessel loop. Cold renal perfusion solution is instilled into the artery until the kidney blanches white and the effluent from the renal vein is clear. Topical slush may also be used.

The rare patient with stent failure requires ex vivo repair, a technique more often used in select patients with renal artery aneurysms. Such a patient usually has a solitary kidney, chronic kidney disease, a deep body habitus, and needs revascularization to the primary branches. In contrast to the modified ex vivo repair, this type of reconstruction requires full mobilization of the kidney, done by making a T-incision in Gerota’s fascia. The renal artery and vein are isolated. A large silastic loop is placed around the ureter and its adjacent soft tissues to control collateral flow. The artery and vein are divided, and the
kidney is elevated to the abdominal wall, leaving the ureter intact. On the right side, a cuff of the vena cava is taken with the renal vein, which lowers the risk of anastomotic stenosis when the vein is sewn back to the cava. For the left renal vein, an oblique venotomy is made. The kidney is laid in an ice slush bath, and it is infused with a cold perfusion solution comprised of saline, heparin, and albumin, until the effluent through the renal vein is clear. Approximately 300 ml of the perfusion solution is usually needed. The modified and full ex vivo techniques allow for meticulous anastomoses to primary or secondary branches without the worry of warm renal ischemia. Sometimes, a heel and toe stitch facilitates the anastomosis to the distal main renal artery or primary branch. If the artery is small, we use interrupted suture for part or all of the anastomosis.

After the arterial reconstructions to the branch arteries are completed, the kidney is placed back in its bed. The renal vein anastomosis is done with continuous suture, leaving growth factor when tying the knot to avoid a purse string at the anastomosis. The vein or hypogastric artery graft is then sewn to the aorta or iliac artery. Urine flow is stimulated by intravenous Lasix and Mannitol after the reconstruction is completed. I often inject 30 mg of Papaverine into the renal bypass graft and bathe the kidney in warm saline to reverse the vasoconstriction which occurs from the cooling. Intraoperative completion duplex ultrasound imaging of the reconstructed renal arteries is then performed to assure technical adequacy of the reconstruction. If abnormal, the technical defect is corrected in the operating room. This imaging technique has been a routine part of our practice for more than 15 years, and has reduced our overall early perioperative graft failure rate for all patients who have renal revascularization.

Our early and late outcome analysis of patients who had renal revascularization for stent failure is ongoing. There have been two early deaths at 48 and 65 days after operation. Twelve patients had some type of perioperative complication, with cardiac, gastrointestinal, and pulmonary complications most frequent. Concomitant aortic reconstruction increased the risk of a major adverse event (MAE). One patient required nephrectomy because of renal infarction from a venous outflow problem. Three patients needed temporary dialysis and two permanent dialysis, all with either chronic kidney disease or a MAE. The remaining 32 patients had no early renal function decline at 3 months.

Open surgical renal artery reconstructions are done with a variety of techniques and for a wide range of pathologies. Intervention for occlusive lesions is best done before patients sustain irreversible renal parenchymal damage. However, the optimum timing of renal revascularization remains a challenge because there is no single test or battery of tests that reliably predicts response to operation. In our practice, open surgical reconstructions have become more complex since the advent of renal artery stenting. The key to successful outcome is careful surgical planning, choice of incision to expose the kidney and arterial inflow source, and measures to minimize warm ischemia to the kidney. We prefer inflow from the hepatic, iliac, or splenic arteries for individuals with calcified or diseased aortas, and for patients at high risk from the physiologic stress of an aortic clamp. Our early data analysis suggests an extra-aortic source of inflow is safer. Completion intraoperative duplex imaging is a useful technique to assess technical outcome in the operating room, and has improved our early outcomes.

References:


