Percutaneous renal artery intervention (PRAI) was first performed in 1978 by Dr. Felix Mahler. The number of PRAIs performed has increased in the last decade, particularly due to increased performance by cardiologists. PRAI has evolved to be a safe and effective method for treatment of renal artery stenosis. Despite a very high procedural success rate of >95%, significant complications ranging from 1% to 21% have been reported in the literature. During the past 3 decades, several innovations have modified the technique of percutaneous renal artery balloon angioplasty, resulting in reduction in the number of complications.

An analysis of five renal artery intervention studies of 795 patients showed a death rate of <1%, a dialysis rate of <1%, and a major complication rate of <2% (death, myocardial infarction, emergency surgery, need for dialysis, or blood transfusion). We review renal artery complications both during diagnostic angiography and renal artery angioplasty/stenting.

**Complications**

**Atheroemboli**

Atheroemboli is a dreaded complication associated with percutaneous interventional vascular procedures. The largest organ involved with atheroemboli is the skin; the toes and foot are mainly affected (Figure 1). Other organs commonly affected are the kidneys, intestine, myocardium, retina, and brain. Diagnosis can be difficult because of the nonspecific symptoms at the time of presentation. Atheroemboli carries a high mortality rate of ~50%. The symptoms include fever, malaise, anorexia, weight loss, myalgia, and headache. Atheroemboli should be suspected in elderly patients, patients with other atherosclerotic disease, and in patients who present with renal failure, macroembolization to the feet or skin, or multigorgan failure. Although prevention of the event is desirable, prompt recognition and aggressive treatment measures are beneficial. The treatment options of atheroembolism include cessation of anticoagulation therapy, parenteral therapy, pain relief, foot care, and hemodialysis. Gentle manipulation of catheters, use of 5-F, soft-tipped catheters for diagnostics, utilization of “no-touch technique,” and flushing the catheter prior to injection may reduce the incidence of atheroembolism.

**Spinal Paraplegia/Paralysis**

Forced injection of contrast via catheters into the artery of Adamkewicz, which arises around the L1 level (supplies the distal spinal cord), can result in paralysis. Use of pigtail catheters or an Omniflush (AngioDynamics, Inc., Queensbury, NY) catheter with side holes and careful contrast injection is helpful.

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**Figure 1.** A case of bilateral lower-extremity and renal artery emboli after manipulation of catheters during renal artery angioplasty and stenting. Bilateral lower-extremity emboli (A, B). Right renal artery showing a 60% stenosis at the ostium (C). Stenting of the right renal artery ostium (D). Atheroemboli of the middle segmental artery after renal artery stent placement (E).
Dissection of the Renal Artery
Aggressive manipulation of the ostium for engagement and disengagement can result in renal artery dissections. Oversizing of the balloon or stent used for angioplasty/stent placement can cause renal artery dissection. Single-center experience demonstrated the rate of flow-limiting dissection to be 1.1%.16 Percutaneous technique is generally helpful to treat renal artery dissection during renal artery angioplasty/stent placement.17 A 20-mm gradient across a dissection or limitation of flow warrants intervention. Most dissections can be managed with prolonged balloon inflations to “tack” the dissection flap to the lumen. If balloon angioplasty fails, use of an additional stent will suffice.

Aortic Dissection
Dissection of the descending aorta can occur with oversizing of the balloon/stent and in severe lesion calcification.18 The intimal separation of the distal aorta at the ostium of the renal artery usually has a benign course. Supportive care includes intensive care monitoring of blood pressure, beta blockers to reduce shear stress, and follow-up imaging with CT scanning (Figure 2).

Contrast-Induced Nephropathy
Patients with diabetes mellitus and baseline renal insufficiency have a higher propensity to develop contrast-induced nephropathy (CIN).19 Prehydration with intravenous normal saline at least 12 hours before contrast exposure has been shown to be beneficial in prevention of CIN.20,21 Several other agents, such as N-acetylcysteine and sodium bicarbonate have some beneficial effects in preventing CIN.22,23 The quantity of contrast used is directly proportional to the incidence of CIN; hence, limiting the amount of contrast will be helpful.24

Renal Artery Perforation
Use of hydrophilic wires can result in distal renal artery perforation (Figure 3). Symptoms include ipsilateral flank pain, hematuria, hypotension, shock, and may result in death.25 Careful visualization of the segmental artery vasculature can identify free vessel perforation and perinephric hematoma. Reversal of anticoagulation, arterial occlusion with a balloon, synthetic material, covered stent, or a stent with autologous vein sewn26 onto it can be beneficial in sealing a perforation.28 Emergent nephrectomy is helpful if these measures fail. The incidence of renal perforation in clinical trials has been 1.1%.4

Renal Artery Rupture
Rupture is usually due to oversizing of the balloon/stent in a calcified renal artery. The incidence of renal artery rupture is also noted after use of atherectomy devices, such as the cutting balloon (Boston Scientific Corporation, Natick, MA).29 in the renal artery. Reversal of anticoagulation, prompt balloon occlusion, covered stent placement, and surgical repair30 have been described for the treatment of renal artery rupture.

Renal Artery Thrombosis or Occlusion
Leertouwer analyzed a case series of 678 patients (799 renal arteries) treated for renal artery stenosis and found the incidence of renal artery thrombosis/occlusion to be 0.8%.4 Renal artery dissection, heparin-induced thrombocytopenia, underlying hypercoagulability, and inadequate anticoagulation during renal artery angioplasty/stenting can cause renal artery thrombosis. Endovascular therapy includes use of thrombolytics and rheolytic thrombectomy. Adequate anticoagulation and endovascular therapy has been used in the treatment of renal artery thrombosis.31

Segmental Infarction
The incidence ranges from 1.1% to 2.8%.16 “Plain old balloon angioplasty” or renal artery stenting can result in atheroemboli of atherosclerotic plaque, leading to segmental infarction; other causes include renal artery perforation, renal artery embolization for perforations, vessel thrombosis, and dissections. Renal artery infarctions are noted after endovascular abdominal aortic aneurysm repair.32,33 Segmental infarctions manifest as severe flank pain, hematuria, nausea, and elevated LDH levels. Prompt recognition and urgent angiography or CT scanning will assist in rapid
diagnosis. Painkillers, hydration, and reversal of anticoagulation with embolization in perforation are helpful. Segmental infarction due to atheroembolism, the role of glycoprotein IIb/IIIa, or bivalirudin has not been extensively studied.

Renal Failure

The most common complication of renal artery intervention is acute renal artery failure. Mechanisms of renal failure after renal artery interventions include excessive use of iodinated contrast material and complications that compromise flow to the kidneys, such as renal artery dissection, thrombosis, atheroemboli, and segmental infarctions. The steps to reduce CIN, renal artery dissection, and thrombosis have been described elsewhere. The incidence of renal failure can be as high as 4.6%.

ACCESS-RELATED COMPLICATIONS

All endovascular procedures have a 5% to 10% rate of complications resulting in a hematoma, infection, pseudoaneurysm, or retroperitoneal bleed. The leading complications are local bleeding and hematoma in 3% to 6%, followed by pseudoaneurysm in patients undergoing percutaneous vascular revascularizations. Arterial thrombosis can result in numbness, paresthesia, and acute leg ischemia. The various predisposing factors that have been identified are small-caliber vessels, a history of concomitant peripheral vascular disease, diabetes mellitus, and female gender.

Femoral Artery

- High anatomical access above the inguinal ligament leads to retroperitoneal hematoma, hypotension, and occult bleed. If left untreated, the bleeding can rapidly deteriorate and lead to death.
- Low access 2 to 3 cm below the inguinal ligament can lead to pseudoaneurysm and arteriovenous fistula formation.
- The causes of groin hematoma are multiple access attempts and improper manual compression, especially in anticoagulated patients.
- Rare complications can include abrupt vessel closure, dissection, and occlusion.
- Significant blood loss requiring transfusions can be due to retroperitoneal bleed, hematoma, and large size femoral hematoma.
- Femoral nerve damage is very rare and may occur during access or due to large groin hematoma.

Brachial Artery

- Pseudoaneurysm and arteriovenous fistula are less common than in the femoral approach.
- Vessel closure, hematoma, and brachial nerve injury have been reported.

Fluoroscopy to visualize the femoral head prior to femoral artery access can assist in proper localization of the common femoral artery. In elderly patients, use of a micropuncture needle is helpful, especially in calcified vessels. Patients in whom access can be difficult include those with weak arterial pulses, those who are obese, and patients who are fully anticoagulated. Alternative access sites, such as popliteal artery access and tibial access, are best achieved with ultrasound guidance. Weight-based heparin and monitoring of the activated clotting time can prevent aggressive anticoagulation and thus bleeding and access site complications.

Arterial access site closure devices can be beneficial in reducing the duration of bed rest after the procedure, however, no difference in bleeding or complications rate compared to manual compression have been demonstrated. Pseudoaneurysms can be effectively treated with local thrombi injection into the aneurysm sac under ultrasound or fluoroscopic guidance. Small arteriovenous fistulae are best managed by observation and repeating the study after 8 weeks; ultrasound-guided compression and the use of covered stents have been utilized. Large pseudoaneurysms, especially wide-necked aneurysms and large arteriovenous fistulae with evidence of heart failure or limb swelling and pain, require surgical intervention.
Small hematomas can be managed by manual compression and application of an external compression device such as the FemoStop (Radial Medical Systems, Inc., Wilmington, MA). Large hematomas require surgical evacuation and drainage. Retropertitoneal bleeding from an identifiable cause early in the course can be treated with balloon occlusion of the artery from a contralateral access. If a percutaneous approach fails or the patient is hemodynamically compromised, surgical repair is warranted.

CONCLUSION

Renal artery angioplasty has evolved over the past 3 decades—both in technique as well as the overall safety of the procedure. Despite a high clinical success rate, renal artery interventions carry the potential for significant morbidity and mortality. Knowledge of various complications associated with renal artery interventions is essential. Use of dedicated catheters, stents, wires, and adjunctive medical therapy can prevent complications.

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