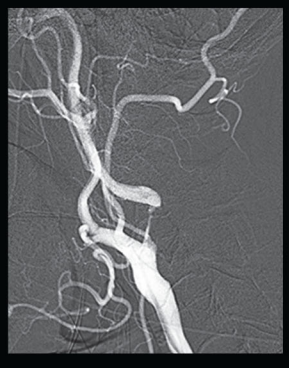
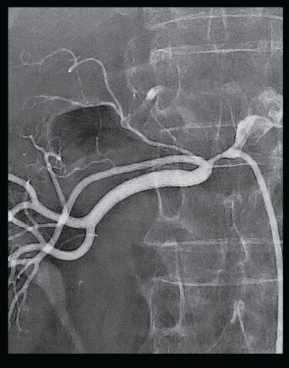
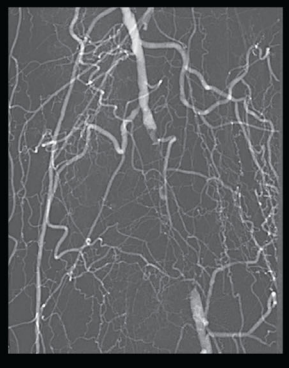
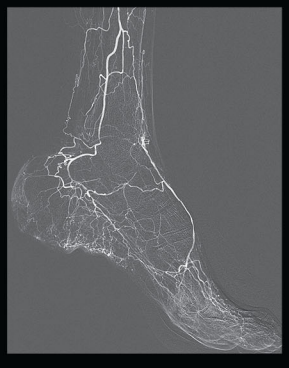
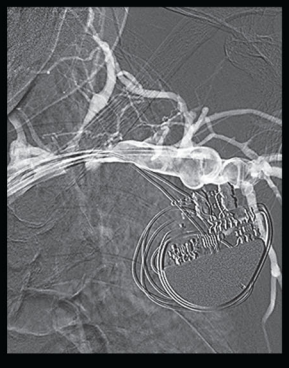
# Carotid and Peripheral Vascular Interventions Textbook

## Step-by-step technique

Thosaphol Limpijankit

CHAPTER 10

# Renal artery intervention

Thosaphol Limpijankit

## INTRODUCTION

Atherosclerotic renal artery stenosis (ARAS) is one of the common etiologies of secondary hypertension and is associated with resistant ischemic nephropathy, hypertension, and cardiac destabilization (1). This disorder remains underrecognized and undertreated because most patients have no symptoms or signs. Several studies examining the natural history of ARAS have shown that, without intervention, progressive vascular occlusion commonly occurs with worsening severity of stenosis by 50% at 5 years (2-4). If undiagnosed, ARAS usually leads to renal function deterioration and progressive renal atrophy and causes 12-14% of dialysis-dependent renal failures (5).

The diagnosis and early treatment of ARAS as a potentially correctable etiology of hypertension and renal insufficiency, has increased using non-invasive imaging modalities such as doppler ultrasound study (DUS), magnetic resonance angiography (MRA), and computerized tomography angiography (CTA). Although the incidence of ARAS recognized by DUS within a general population ranges between 0.5% and 7% (6), the prevalence increases to 14-39% in patients receiving coronary angiography (7) and 14-42% in patients undergoing peripheral artery angiography (8). The higher prevalence rates in these high-risk populations emphasize using a screening test to diagnose underlying asymptomatic renal artery stenosis.

Although ARAS-associated hypertension may be successfully managed with medication, but it has a propensity to be more resistance than primary essential hypertension, often requiring more medication. In addition, patients are more likely to suffer from shortened life span and progressive renal failure than those treated with renal artery revascularization.

The aims of renal artery revascularization are (1) to improve or cure renovascular hypertension or heart failure that is unresponsive to a combination therapy medication and (2) to stabilize or improve renal function. Renal revascularization using percutaneous transluminal renal artery stenting (PTRAS) in selected groups of population with severe ARAS has proven to be benefit (9,10) even though several randomized-controlled trials failed to demonstrate the superiority of PTRAS versus optimal medical therapy (OMT) (11-13).

## ANATOMIC CONSIDERATIONS

The two renal arteries arise from the lateral surface of the descending aorta at around the L1-L2 vertebral level, just below to the anterior origin of the superior mesenteric artery. The origin of the right renal artery is frequently slightly higher than the left renal artery, the take-off points are slightly posterior, and the main renal artery remains intact for a variable length (Fig. 10-1A). The proximal renal arteries have small inferior adrenal, ureteric and capsular branches, that are usually not visible during arteriography. At the renal hilum, the renal artery bifurcates into ventral and dorsal rami. These trunks branch into segmental arteries, lobar arteries, interlobar, arcuate, and interlobar arteries. Within the renal cortex, the arcuate and interlobular arteries branch into the smaller afferent arterioles which penetrate the renal cortex and medulla to supply the glomeruli. Importantly, in these small vessels atherosclerotic disease on top of hypertensive glomerular injury can explain deterioration of renal function and incurable hypertension, even following successful renal artery revascularization.

There are a number of differences in renal artery anatomy. The most frequent variation is the presence of one or more accessory renal arteries that are identified during angiography in about 25% to 35% of cases (14). Most accessory renal arteries are usually small caliber and typically supply the lower pole (Fig. 10-1B) or the upper pole of the kidney which may arise anywhere

between the suprarenal aorta and the iliac artery. However, in some instances, the accessory renal artery can have a caliber similar to the main renal artery (Fig. 10-1C), thus providing a large part of the renal blood supply. In this circumstance revascularization of the accessory renal artery stenoses should be performed. Normally, a main renal artery remains intact for several centimeters prior to dividing into a variable number of segmental branches. Early subdivision or bifurcation of the main renal artery (Fig. 10-1D) is the second most common anatomic variant, and it makes optimal percutaneous revascularization more challenging.

## PATHOPHYSIOLOGY

Significant ARAS is generally a luminal stenosis >70% that leads to reduced renal perfusion pressure and stimulates the renin-angiotensin-aldosterone system (RAAS) (Fig. 10-2) (15). The net effect of this activation results in sodium retention, peripheral vasoconstriction, aldosterone secretion, vascular remodeling, inflammation and triggering of additional vasopressor mechanisms including endothelin and sympatho-adrenergic pathways (16,17).

ARAS has two principle pathophysiological consequences: 1) RAAS activation (in unilateral stenosis) and 2) reduced glomerular filtration and water and salt excretion (i.e., bilateral artery stenosis or Pickering syndrome (18) or renal artery stenosis of a solitary kidney). Although the post-stenotic kidney has less perfusion, the contralateral kidney experiences glomerular hyperfiltration and hyperperfusion associated with RAAS activation by the stenotic kidney. With prolonged exposure, there is progression of arteriosclerotic lesions and parenchymal injury to the contralateral kidney that may lead to persistent proteinuria and result in pathologic changes from secondary focal segmental glomerulosclerosis of the contralateral kidney. Revascularization in patients with severe unilateral renal artery stenosis may prevent atrophy of the affected kidney and protect the contralateral kidney.

About 20% of patients have a single functioning solitary kidney or bilateral RAS disease. These worse case scenarios create a state of sodium and fluid retention so that volume-dependent hypertension develops which then aggravates heart failure symptoms in patients who have impaired left ventricular function and causes progressive worsening of renal function. These subgroups of ARAS are two of the few absolute indications for endovascular revascularization.

## ETIOLOGY

Renal artery stenosis has many etiologies (Table 10-1). The most common is atherosclerosis that is progression of aortic atherosclerotic plaque which affects the proximal segments of the renal arteries and the renal ostia (Fig. 10-3A).

Fibromuscular dysplasia (FMD) is the 2nd most frequent etiology and is a nonatherosclerotic, noninflammatory disorder with unknown etiology that typically affects women aged between 15-50 years (19). FMD commonly involves on the mid to distal portions of the renal arteries and causes the angiographic appearance of "string of beads" aneurysmal appearance (Fig. 10-3B). Contrasting



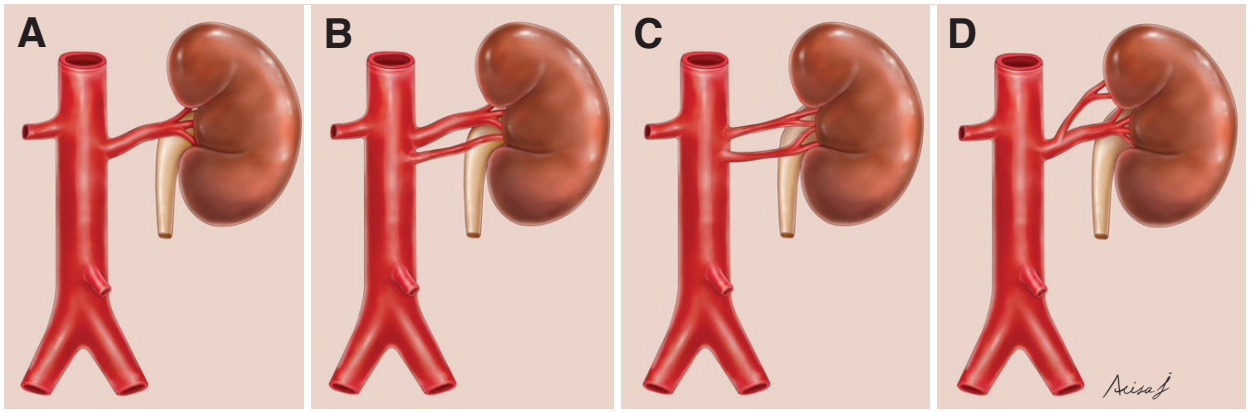



**Figure 10-1.** Anatomical variations of renal artery. A: Normal single renal artery. B: An accessory inferior pole renal artery. C: Two main renal artery. D: Early bifurcation of renal artery. (Redrawn with permission from Omar R, Kisansa M, Dehnavi AD. The prevalence of anatomical variants of the coeliac trunk and renal arteries on contrast-enhanced abdominal computed tomography scans at Dr George Mukhari Academic Hospital. *SA J Radiol.* 2021;25:1990.)

with ARAS, the major clinical manifestation of renovascular FMD is hypertension which hardly ever causes recurrent pulmonary edema or renal impairment. Although medical management of hypertension is frequently successful, the high rates of procedural success, elimination of hypertension, and low recurrence rate (10%) of percutaneous transluminal angioplasty (PTA) have resulted in a low threshold for intervening in FMD patients (19).

Takayasu's arteritis is an uncommon systemic vasculitis principally effecting the aorta and its major branches, including the renal artery. Nearly half of the Asian Takayasu's arteritis patients suffer from renal artery involvement (20).

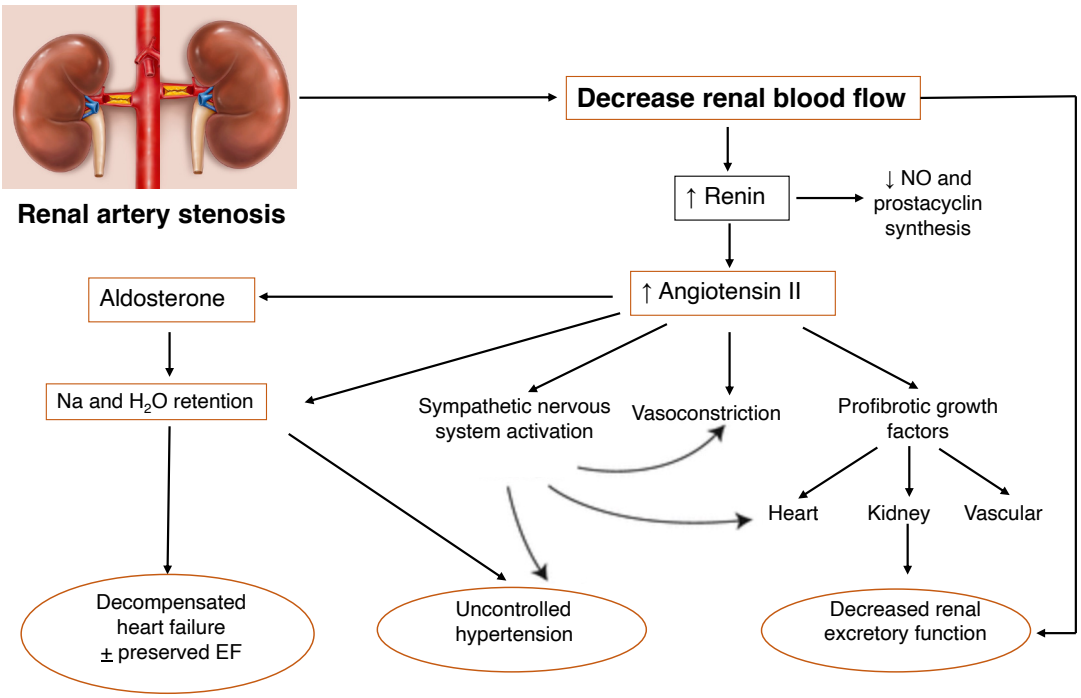



**Figure 10-2.** Pathophysiology of renal artery stenosis (Reprinted from Fernando D, Garasic J. Percutaneous intervention for renovascular disease: rational and patient selection. *Curr Opin Cardiol.* 2004;19:582-588, with permission from Wolters Kluwer Health, Inc.).
EF, ejection fraction; H2O, water; Na, sodium; NO, nitric oxide



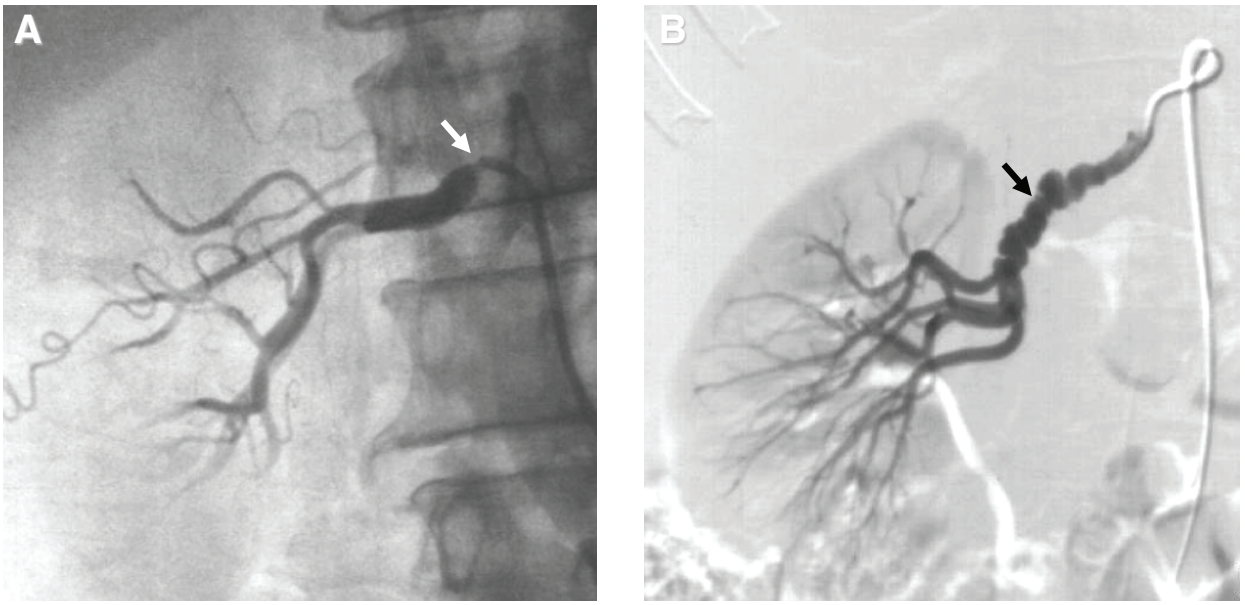



**Figure 10-3.** A: Atherosclerotic renal artery stenosis at the renal ostium (white arrow). B: Fibromuscular dysplasia with 'string of beads' stenosis (black arrow) at the mid to distal part of the renal artery.

The location is often bilateral and frequently ostial or proximal with perirenal aortic wall involvement. Aneurysmal lesions have rarely been reported. Immunosuppressive therapy is important to slow the inflammatory process and prevent restenosis after revascularization.

Extrinsic compression of the renal artery caused by a fibromuscular band beginning from the diaphragm is a rare condition. This disorder causes systemic hypertension associated with kinking of the renal artery and stenosis. Surgical decompression is mandatory in all cases (21). Other causes of renal artery stenosis such as dissection or embolus can also be treated with endovascular techniques.

**Table 10-1.** Etiologies of renal artery stenosis.

| |
|---|
| Atherosclerosis |
| Fibromuscular dysplasia (FMD) |
| Extrinsic fibrous band |
| Renal artery dissection |
| Aortic dissection |
| Arterial embolus |
| Aortic endograft occluding the renal artery |
| Miscellaneous: |
| Autoimmune diseases |
| (e.g., Takayasu's arteritis, polyarteritis nodosa) |
| Hypercoagulable state with renal infarction |
| (e.g., Lupus anticoagulant) |
| Malignancy encircling the renal artery |
| (e.g., Renal cell carcinoma, pheochromocytoma) |

## CLINICAL PRESENTATIONS

RAS is assoiciated with a variety of clinical manifestations as shown in Fig. 10-4. Symptoms can range from mild-to-severe uncontrolled hypertension to worsening renal function and accelerated cardiovascular (CV) events, such as unstable angina and congestive heart failure (CHF). Many patients with RAS persist clinically asymptomatic because of the large kidney functional reserve and present as an incidental finding during imaging of other vessels. Hypertension with sudden onset in persons <30 years of age usually is a manifestation of FMD. If the diagnosis of hypertension is initially made in a patient >55 years of age who present with malignant, accelerated or resistant hypertension, ARAS should be considered. Patients developing progressive azotemia secondary to ischemia (a rise in serum creatinine of >30%), or unexplained hypokalemia (secondary aldosteronism), or azotemia that is drug-induced (administration of ACEI or ARBs), should be examined for RAS (22). The presence of an abdominal or flank bruit or atherosclerotic disease appearing elsewhere in the body can increase the likelihood of RAS. Kidney size is also important. Prolonged decrease of blood flow with tissue hypoxia causes irreversible kidney damage and fibrosis, so-called "ischemic nephropathy". An atrophic kidney (length <7cm) or a size discrepancy between the two kidneys of larger than 1.5 cm are important clues of a possible severe stenosis of the renal artery supplying the small kidney. Finally, recurrent episodes of CHF, or impaired left ventricular systolic function, or unexplained flash pulmonary edema without significant myocardial ischemia may result from stenosis of the renal artery of a solitary kidney, or severe bilateral RAS (Pickering syndrome). These conditions should be considered a hypertensive emergency that will benefit from an urgent endovascular revascularization. Table 10-2 summarizes the clinical indicators for the diagnosis of RAS (23).

**Table 10-2.** Clinical clues to the diagnosis of renal artery stenosis.

- Onset of hypertension before the age of 30 years
- Onset of severe hypertension after the age of 55 years, when associated with CKD or heart failure
- Hypertension and abdominal bruit
- Rapid and persistent worsening of previously controlled hypertension
- Resistant hypertension (i.e., other secondary causes unlikely and target blood pressure not achieved despite four drug classes including a diuretic and a mineralocorticoid-receptor antagonist in appropriate doses)
- Hypertensive crisis (i.e., acute renal failure, acute heart failure, hypertensive encephalopathy, or grade 3-4 retinopathy)
- New azotemia or worsening of renal function after treatment with RAAS blockers
- Unexplained atrophic kidney or discrepancy in kidney size (>1.5cm) or unexplained renal failure
- Flash pulmonary edema

CKD, chronic kidney disease; RAAS, renin-angiotensin-aldosterone system

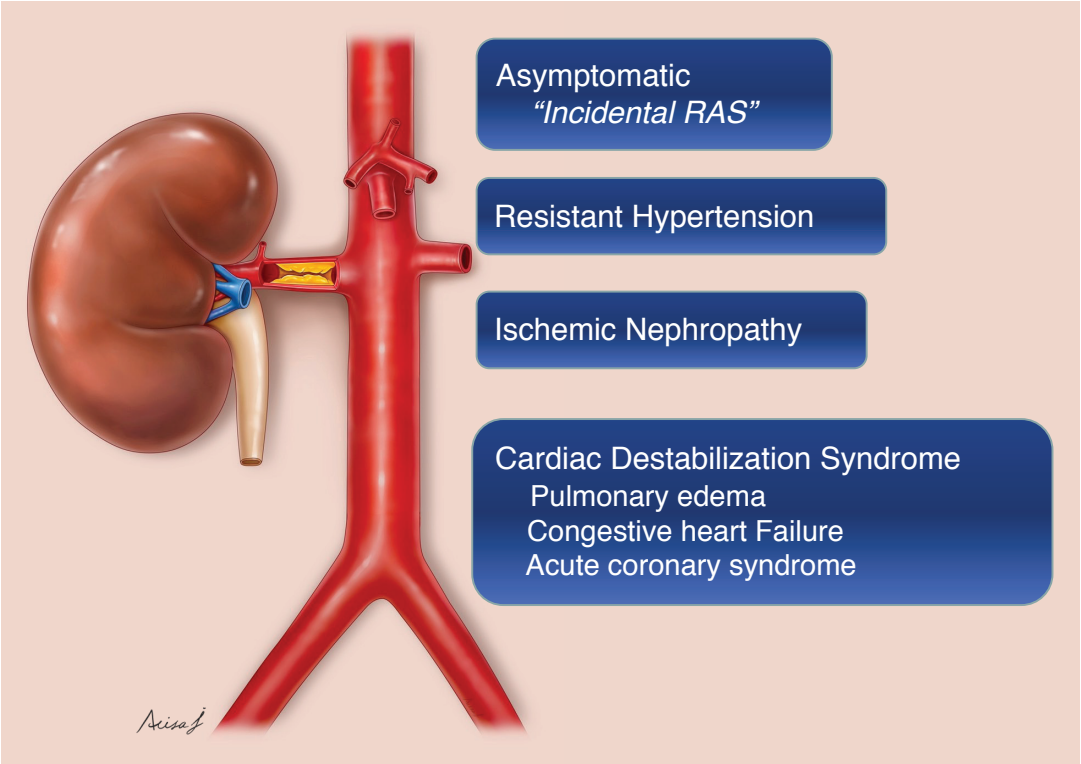



**Figure 10-4.** A spectrum of clinical presentation of renal artery stenosis.

## IMAGING STUDIES

The ideal imaging study should assess both the main and accessory renal arteries, evaluate the hemodynamic significance of a lesion, recognize the severity and location of the stenosis, and recognize related perirenal pathology, such as renal or adrenal masses or the appearance of an abdominal aortic aneurysm. DUS is the first-line imaging test to screen for a significant (>60%) stenosis (24). This can be repeated to evaluate stenosis progression and its hemodynamic consequence (e.g., renal vascular resistance and flow velocity). Moreover, the renal resistive index (RRI) can assist to recognize a more severe stenosis and give additional information on response to intervention (Fig. 10-5). The RRI is defined as (peak systolic velocity - end diastolic velocity) /peak systolic velocity. The normal range is 0.50-0.70. High resistive indices (>0.8) in native kidneys are associated with poor response to intervention.

Multidetector MRA or CTA (without or with gadolinium) shows comparable high specificities (92-98% and 85-93%) and sensitivities (64-100% and 94-97%) to detect significant RAS (25). CTA provides better spatial resolution than MRA (Fig. 10-6) and likely being more readily available, although the need to use iodinated contrast makes it an unappealing modality for impaired renal function patients. Gadolinium-enhanced MRA produces excellent anatomy of the renal mass, renal arteries, the surrounding vessels, as well as renal excretion function. However, it has a propensity to overestimate stenosis, and it may be less helpful in patents with renal artery stents due to artifacts. In impaired renal function patients , the cut-off levels of estimated glomerular filtration rate (eGFR) suitable for CTA or MRA study are >60 mL/min and >30 mL/min, respectively (23).

Digital subtraction angiography (DSA) is still the gold standard for diagnosing RAS (25). Besides better resolution, the major benefit of DSA is the potential to measure the pressure gradient over the lesion, particularly for a moderate stenosis. Renal artery fractional flow reserve (FFR) measurement during maximum hyperemia triggered by dopamine or papaverine is another way to evaluate the severity of RAS that may predict the clinical response to intervention (26). Because of invasive procedure and potential risks of contrast exposure or atheroemboli, angiography is normally limited to quantification and visualization of the stenosis prior to vascular intervention

or to clarify inconclusive results from noninvasive study. Venous renin measurements, plasma renin measurements before and after angiotensin converting enzymes inhibitor (ACEI) provocation, and renal scintigraphy are no longer used for diagnosing atherosclerotic RAS (1,23,27).

## TREATMENT

Patients with RAS with or without end-stage CKD have a shortened life expectancy (28). Most of them died from acute CV events (29). Risk factor assessment, life-style modification, and medications (e.g., antiplatelet and statin) are important and should follow current guidelines of primary prevention for CAD (30,31). Most antihypertensive drugs [diuretics, beta-blockers, calcium channel blockers, and ACEIs, angiotensin renin blockers (ARBs)] are effective for managing hypertension associated with renal artery disease and can cause slowing of its progression. In two observational studies ARBs and ACEIs have shown advantages in decreasing morbidity and mortality in RAS patients (32,33). However, these drugs may decrease glomerular capillary hydrostatic pressure and result in a transient reduction in eGFR and increase serum creatinine. From the 2017 ESC guidelines (23), ACEIs or ARBs are recommended for hypertension treatment related to unilateral RAS. ARBs or ACEIs can also be administered in bilateral severe RAS as well as a single functioning kidney stenosis if well-tolerated with close observation. Nearly all patients having significant RAS tolerate ARBs or ACEIs without any problem. However, optimal target blood pressure (BP) for the setting of RAS, is still unknown.

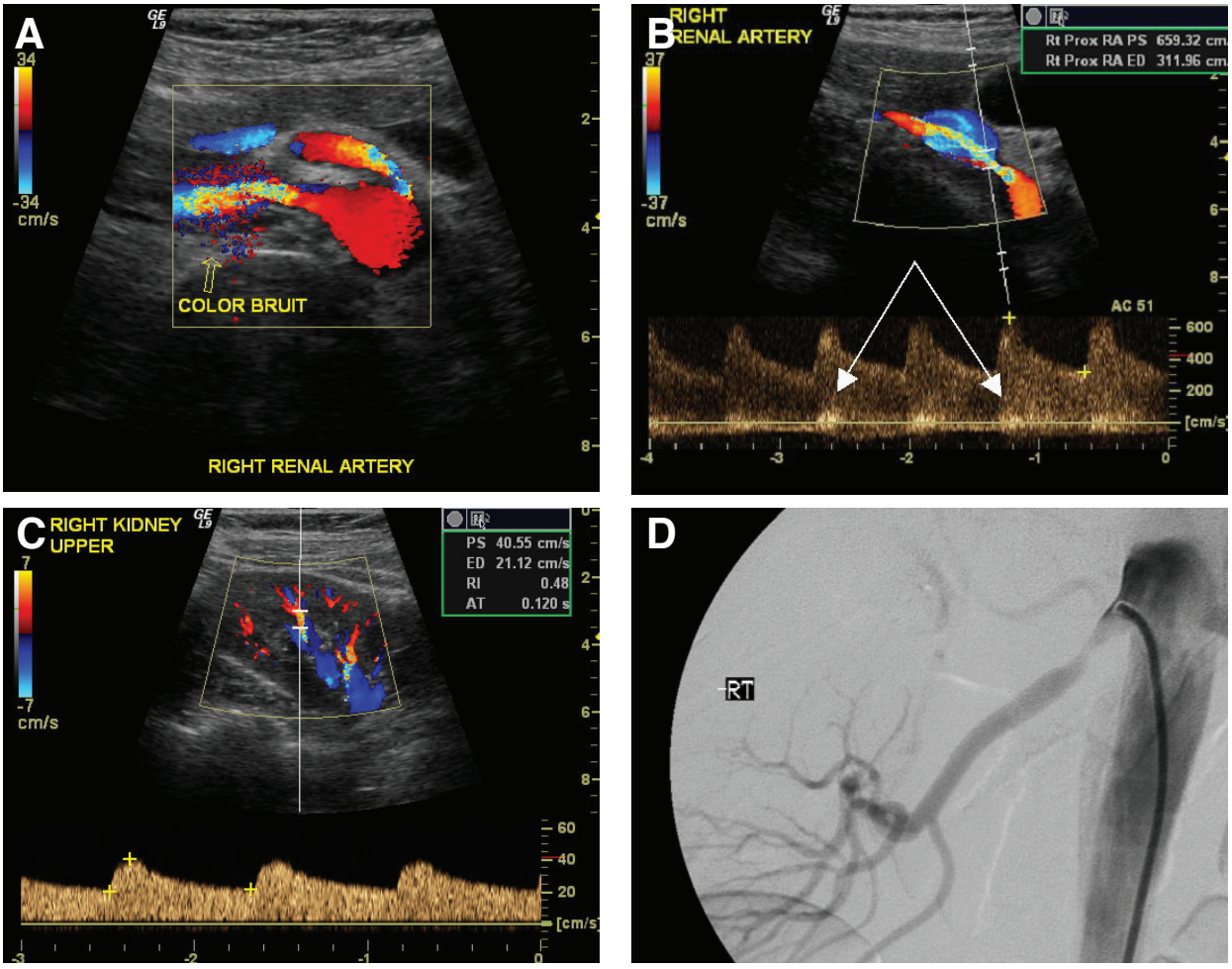



**Figure 10-5.** Doppler ultrasound of right renal artery stenosis. A: Color doppler image showing a color bruit of the post stenotic turbulence. B: Doppler waveform obtained near the renal artery origin showing velocities over 600 cm/s in systole and over 300 cm/s in diastole consistent with a high-grade stenosis. C: Doppler waveform obtained from the segmental renal arteries within the right kidney showing delayed systolic acceleration with absence of the early systolic peak. The calculated renal resistive index (RRI) is normal (=0.48) with good prognosis. D: Subsequent digital subtraction angiogram before undergoing renal stenting revealed a high-grade stenosis of proximal right renal artery.

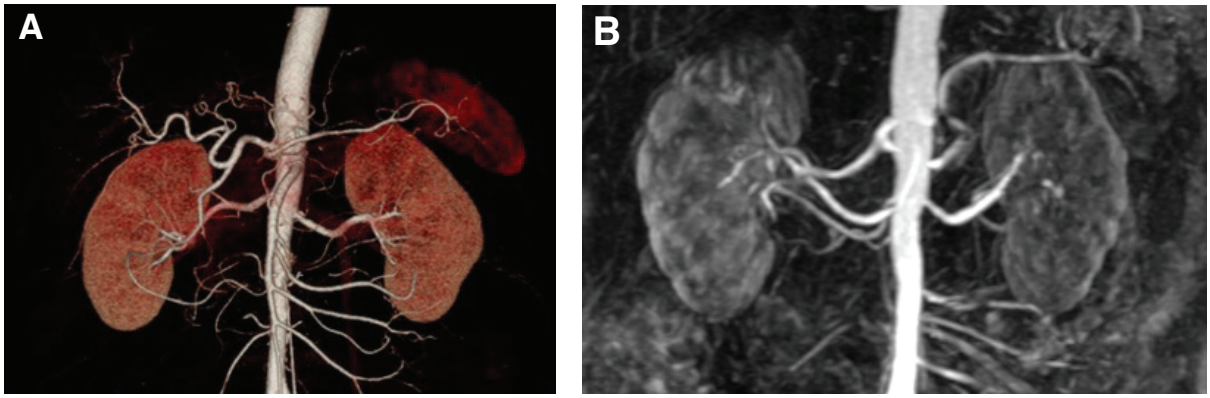



**Figure 10-6.** Comparison imaging resolution between computerized tomography angiography (A) and magnetic resonance angiography (B) of renal arteries.

## INDICATION FOR REVASCULARIZATION

The following organizations have published guidelines for the treatment of RAS: 2013 American College of Cardiology (ACC)/American Heart Association (AHA) (34), 2014 Society of Cardiovascular Angiography and Interventions (SCAI) (35), and 2017 European Society of Cardiology (23). There are some discrepancies among these guidelines, but all guidelines favor medical therapy for the primary management of RAS.

The 2013 ACC/AHA guidelines recommend PTRAS in patients who have hemodynamically significant RAS together with any of the following (34) :

- Recurrent CHF or sudden unexplained pulmonary edema (Class I, LOE B)
- Unstable angina (Class IIa, LOE B)
- Accelerated, resistant, or malignant hypertension or hypertension with unexplained unilateral small kidney and hypertension with medication intolerance (Class IIa, LOE B)
- RAS and chronic renal insufficiency (CRI) with bilateral RAS or RAS to solitary functioning kidney (Class IIa, LOE B)
- Asymptomatic bilateral or solitary viable kidney with a hemodynamically significant RAS (Class IIb, LOE C)
- Asymptomatic unilateral hemodynamically significant RAS in a viable kidney (Class IIb, LOE C)
- RAS and CRI with unilateral RAS (2 kidneys present) (Class IIb, LOE C)

The 2017 ESC guidelines recommended balloon angioplasty, without or with stenting, only selected RAS patients and unexplained repeated congestive heart failure or sudden pulmonary edema (Class IIb) (23). Both ACC/AHA and ESC also recommended balloon angioplasty together with bailout stenting in case of FMD with hypertension and/or indications of renal impairment (Class IIa) (23,34).

In 2018, ACC/AHA/SCAI/SIR/SVM produced an appropriate use criteria (AUC) statement for PTRAS (36). Using an expert panel to scientifically review data, they concluded that patients with the following were most likely to gain advantage from PTRAS (36,37).

1. Cardiac destabilization syndromes: flash pulmonary edema with severe hypertension
2. Resistant (refractory) hypertension
3. Rapidly progressive ischemic nephropathy, CKD with eGFR less than 45 cc/min/$m^2$, and global renal ischemia.

Table 10-3 summarizes the AUC for treatment of ARAS in different indications (36). Both clinical and anatomical lesion criteria have to be met. Severe RAS is defined as a stenosis diameter ≥70-99% or a stenosis 50-69% with a translesional peak pressure gradient (measured with a ≤ 5-Fr catheter or pressure wire) ≥20 mmHg or a resting mean pressure gradient ≥10 mmHg, a hyperemic mean gradient ≥10mmHg or FFR performed with dopamine (50 µg/kg) or papaverine (32 mg) of <0.8 or any stenosis ≥70% diameter by intravascular ultrasound (36).

## SURGICAL REVASCULARIZATION

Although surgical renal revascularization is associated with more durable long-term results, the high surgical risk limits its use. Surgical revascularization should be appraised only for patients who have complex renal arteries' anatomy, following a failed endovascular procedure, or during open aortic surgery (i.e., presence of abdominal aortic aneurysm, renal artery dissection or aneurysm) (23,34).

**Table 10-3.** ACC/AHA/SCAI/SIR/SVM 2018 Appropriate Use Criteria (AUC) for Treatment of ARAS-. (Modified from Bailey SR, Beckman JA, Dao TD, et al. ACC/AHA/SCAI/SIR/SVM 2018 Appropriate use criteria for peripheral artery intervention: A report of the American College of Cardiology Appropriate Use Criteria Task Force, American Heart Association, Society for Cardiovascular Angiography and Interventions. *J Am Coll Cardiol*. 2019;73:214-237, with permission from Elsevier).

| Indications | | AUC Score | |
|---|---|---|---|
| | | Continue or Intensify Medical Therapy | Renal Stent Placement (Primary Stenting) – Atherosclerotic Lesions |
| **Hemodynamically Significant RAS (With a Severe [70-99%] RAS or 50-69% RAS with hemodynamic Significance)** | | | |
| **Chronic Kidney Disease*** | | | |
| 1. | • Bilateral RAS or a solitary viable† kidney with RAS<br>• Accelerating decline in renal function | | Appropriate (Class IIa, LOE B) |
| 2 | • Unilateral RAS<br>• Accelerating decline in renal function | Appropriate (Class I, LOE A) | May Be Appropriate (Class IIb, LOE C) |
| 3 | • Unilateral smaller kidney (<7cm pole to pole) | | Rarely Appropriate |
| **Hypertension** | | | |
| 4 | • Failure to control BP on 3 maximally tolerated medications, 1 of which is a diuretic | | May Be Appropriate (Class IIa, LOE B) |
| 5 | • Uncontrolled on <3 antihypertensive medications | Appropriate (Class I, LOE A) | Rarely Appropriate |
| 6 | • Well-controlled BP on ≥2 antihypertensive medications | | |
| 7 | • New onset<br>• No medical management | | |
| **Cardiac Destabilization** | | | |
| 8 | • Sudden-onset flash pulmonary edema | | Appropriate (Class I, LOE B) |
| 9 | • Recurrent ADHF requiring hospitalization<br>• Uncontrolled on maximal medical therapy | | May Be Appropriate (Class I, LOE B) |
| 10 | • Uncontrolled unstable angina despite maximal medical therapy | | May Be Appropriate (Class IIa, LOE B) |
| **Incidentally Discovered RAS** | | | |
| 11 | • Bilateral RAS or a solitary viable† kidney with RAS | Appropriate (Class I, LOE A) | Rarely Appropriate |
| 12 | • Unilateral RAS | | |
| **Borderline (50-69 %) RAS Without Hemodynamic Confirmation of Severity** | | | |
| 13 | • Unilateral RAS, bilateral RAS, or a solitary viable† kidney with RAS | Appropriate (Class I, LOE A) | Rarely Appropriate |

*Chronic kidney disease was defined as a decrease in estimated GFR rate <60mL/min/1.73 $m^2$ or serum creatinine >1.5mg/dl that persisted for at least 3 months.
†Viable is pole to pole kidney length ≥7cm.
ADHF, acute decompensated heart failure; BP, blood pressure; RAS, renal artery stenosis

# STEP BY STEP TECHNIQUE

## PATIENT SELECTION

With technological improvements of imaging and percutaneous intervention, our capability to diagnose and to treat RAS has increased considerably. However, this procedure still carries some risks which include cholesterol embolization, renal artery occlusion, contrast induced nephropathy (CIN), as well as restenosis, which altogether may further impair renal function. Identifying those patients that will probably gain the most benefit from intervention by using the AUC will spare others from unnecessary procedural risks.

In general, rapidly declining renal function is indicative of a favorable result after intervention (38). PTRAS improves or preserves renal function for most patients who have normal to moderately renal dysfunction. Patients who have a serum creatinine >2.8-3.0mg/dL are less likely to benefit (39) and have increased procedural-related complications. Bilateral ARAS or a solitary functioning kidney with a significant stenosis is a strong predictor of favorable response (40). Interventions should only be performed in patients with dialysis-dependent end-stage CKD in the setting of clinical study protocols due to rare data in this population. In borderline cases of severe chronic ischemic nephropathy when chronic dialysis is imminent, discussion of the planned intervention with a nephrologist is recommended.



## PATIENT PREPARATION

Before intervention, patients should be pre-treated with aspirin 81-325 mg/day and clopidogrel 75 mg once daily (although an advantage has not yet been proven) beginning at least 5-7 days or with a loading dose one day prior to the planned intervention. In patients with documented renal insufficiency, sufficient hydration and limiting contrast volume are useful to prevent contrast-induced nephropathy (CIN). Oral N-acetylcysteine did not significantly reduce the incidence of CIN and is not recommended (41). As with any other intervention, the patient should be fasting for at least 6 hours and receive intravenous normal saline hydration prior to arriving in the catheterization lab.

Once arterial access has been obtained, unfractionated heparin 50-100 units/kg should be administered to obtain an activated clotting time (ACT) of 250-300 seconds. The routine use of platelet glycoprotein (GP) IIb/IIIa inhibitors is not advised, although these agents may be useful in a complicated case with acute thrombus or distal embolization.

## VASCULAR ACCESS SITE

The common femoral artery (CFA) is still the traditional access site for renal artery intervention because of the ease of catheter manipulation and because it is close to the RAS lesion. It may be useful to use the right CFA approach to perform right renal artery stenosis and the left CFA approach to perform left sided stenosis because of a better adaptation of the shape of the catheter to the anatomic course of the iliac artery and aorta. If there is severe atherosclerotic disease iliac arteries or in the aorta, infrarenal abdominal aortic aneurysm (AAA), or kinking of the pelvic arteries, a long sheath (i.e., about 35 cm) may provide better support and avoid additional trauma to these vessels during catheter exchanges.

Radial access is more being used to perform percutaneous coronary intervention due to better post-procedural patient comfort and the lower access site bleeding complications. The radial approach for renal stenting is technically feasible and safe and can be considered as an alternative to the traditional transfemoral approach (Fig. 10-7) (42,43). This vascular access has obvious benefits in patients with difficult femoral access, bilateral aortoiliac disease, infrarenal AAA, or caudally angulated renal arteries. Nevertheless, an operator requires specific technical skill together with understanding about device compatibility. For renal intervention, either the right or left radial artery can be used. Normally, left radial access permits less distance to the renal arteries which depends on aortic arch tortuosity so it is preferable for taller patients. The right radial approach is more comfortable for the operator with less radiation exposure compared with the left radial approach. Using a 125 cm long guiding catheter with a 150 cm balloon or stent shaft are suitable for most patients where a 100 cm long catheter and 135 cm long balloon or stent shaft might not extend to the renal arteries for those patients who have excessive aortic arch tortuosity or taller patients . Brachial access is also feasible, but has higher risk of access site complications.

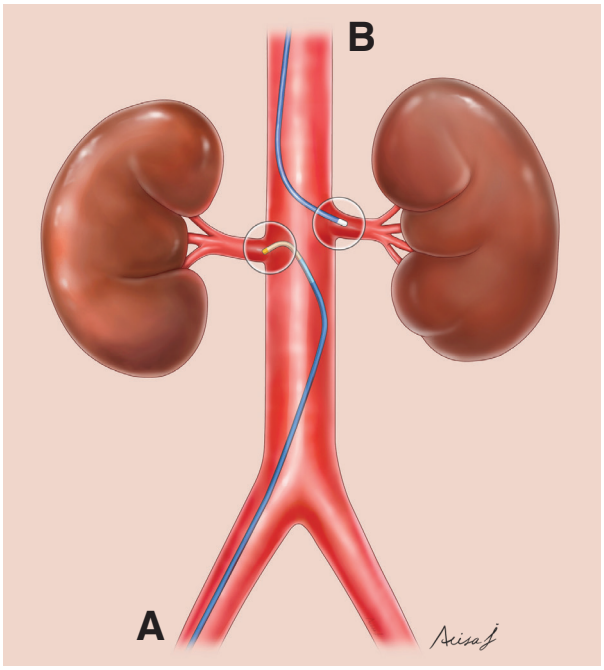



**Figure 10-7.** Two different vascular access approaches for renal artery intervention. A: Femoral approach. B: Radial or brachial approach.

## DIAGNOSTIC ANGIOGRAPHY

If there is no pre-procedural CTA or MRA study, an abdominal aortogram should be conducted before selective renal artery cannulation using a 5-Fr or 6-Fr straight pigtail or Omni™ Flush catheter (Fig. 10-8). Because the renal arteries originate at L1, the side holes of the catheter should be positioned slightly above at the T12-L1 interspace. DSA with an injection of 10-15mL of contrast (at rate 10-15 mL/sec) allows visualization of the beginning of the main renal arteries that normally arise between the mid L1 and the mid L2 level as well as accessory renal arteries if present.

The angle for the origin of the renal arteries from the aorta is important since frequently an anterior-posterior (AP) angiogram fails to display an ostial lesion accurately. Unwarranted stent extension into the aorta or geographic miss with a floating stent positioned within the post-stenotic segment can occur if not clearly defined. In the transverse plane the origin for the right renal artery has a tendency to be anterolateral, and the origin of the left renal artery tends to be posterolateral or lateral (44). If non-selective abdominal angiography is performed, then it is better to position a catheter in the L1 level to conduct imaging in a 20° LAO projection (Fig. 10-9). When selective angiography is conducted, then obliquity should be customized as explained above; and when the mid to distal portion of the renal artery has to be visualized, use of an ipsilateral oblique projection is recommended.

To engage the renal arteries selectively, the angle by which the renal artery comes off the aorta determines the choices of diagnostic catheters. The three types of take-off angulation include horizontal, downward (or caudal), or upward (Fig. 10-10). If renal arteries take-off horizontally, selective cannulation may be conducted with a Judkins Right (JR)-4 catheter (commonly used), internal mammary artery (IMA), Amplatz right (AR) 1, or Cobra catheter, (Fig. 10-11). However, renal arteries with a downward take-off might require cannulating with a SOS Omni, Cobra, RC 2, or HK 1 catheter (Fig. 10-12). Selective renal angiograms are performed with 3-4 mL of contrast, injected by hand using DSA with an ipsilateral oblique projection. If there is posterior take-off point of



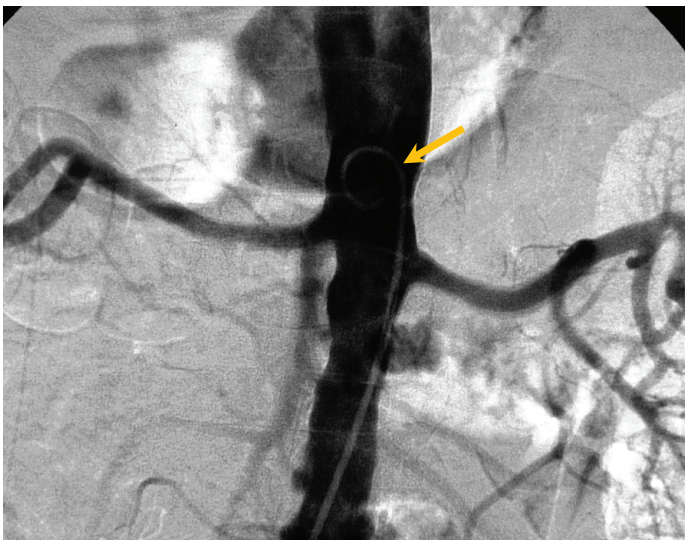


**Figure 10-8.** Abdominal aortogram using a 5-Fr pigtail catheter (orange arrow) placed above at the T12-L1 interspace.