Arterial Interventions for Renovascular Hypertension

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ABSTRACT

Renovascular hypertension is a major cause of secondary hypertension. It affects relatively younger patients. The unifying pathology is renal artery stenosis. The most common cause is atherosclerosis accounting for about 90% of cases with fibromuscular dysplasia being the second most common cause. Both of these are amenable to percutaneous interventional therapy. With the advent of new medical therapies, the control of blood pressure has improved significantly. In well-selected patients, renal arterial intervention has a good outcome. The intervention includes renal angioplasty and stenting. In this article, the authors review the role of percutaneous intervention and the techniques involved with renal angioplasty and stenting for the treatment of renovascular hypertension.

KEYWORDS: Renal artery stenosis, renovascular hypertension, atherosclerosis, fibromuscular dysplasia, renal artery angioplasty, renal artery stenting

Objectives: Upon completion of this article, the reader should be able to demonstrate an increase in, or affirmation of, their knowledge of renovascular hypertension, and define its medical and interventional management including the technique of intervention.

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Renal artery stenosis (RAS) defined as >50% stenosis of the renal artery lumen. It is recognized as a major cause of secondary hypertension, also referred to as renovascular hypertension, accounting for 1 to 6% of the 60 million cases of hypertension in the United States.¹

The rise in incidence parallels the increase in the atherosclerosis-induced peripheral vascular diseases (PVD) and coronary artery disease (CAD), particularly in the aging population (>50 years old). An increasing number of cases are recognized because of better screening and increasing longevity. Atherosclerosis is the major cause, accounting for ~90% of renovascular hyperten-

sion cases, with fibromuscular dysplasia (FMD) causing the rest.²

Nearly 50% of symptomatic RAS cases progress to renovascular hypertension and/or ischemic nephropathy (chronic renal insufficiency).³ Asymptomatic patients who solely have an anatomic abnormality with no pathophysiologic consequence go undocumented. As of now, it is not possible to predict accurately which patients will be symptomatic. The life expectancy of these subjects is mainly decided by their cardiovascular profile, which most often is the reason for early mortality.⁴ Therefore, what most of us encounter is a "tip of the iceberg."

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Imaging procedures for diagnosis of RAS have undergone a phenomenal change over the years. This has been possible through the development of noninvasive techniques like computed tomography angiography (CTA) with three-dimensional (3D) reconstruction, and duplex ultrasound scanning. Until recently, gadolinium-enhanced magnetic resonance angiography (MRA) was a very valuable technique. Unfortunately, with the advent of nephrogenic systemic sclerosis, this modality is declining in utility. Overall, conventional catheter angiography is considered the gold standard for diagnosis as it is close to 100% sensitive for proximal main renal artery, intrarenal, or accessory renal artery stenosis; helps in defining the subtle changes in the renal vascular bed in FMD; and allows for possible intervention.

Patients with accelerated hypertension resistant to medical therapy and those advancing to chronic renal failure are treated with percutaneous transluminal angioplasty (PTA), stenting, or other surgical revascularization techniques after weighing the pros and cons of these interventions with regards to overall and symptom-free survival.² Surgical bypass procedures and ultrasound-guided endarterectomy have only a few indications and carry higher associated risks.^{5,6} Understandably, the current standard of treatment is minimally invasive procedures in the form of PTA and vascular stent placement. These interventions have better technical and anatomical results, decrease the complication rates and hospital stay, and help to maintain a long-term arterial lumen patency particularly in patients with FMD where it is the procedure of choice.² Nevertheless, 20 to 30% of the patients may exhibit deterioration in renal function post PTA due to atheroembolism and ~10 to 20% may develop in-stent restenosis.⁷ This poses a concern in making PTA more readily accepted; however, with the advent of distal renal protection devices and drug-eluting stents this problem may lessen and show better outcomes.⁸

Due to compelling reasons, cardiac catheterization and peripheral arterial angiographies are being performed more aggressively and rigorously in patients with CAD and PVD. In the same group of patients who also have concurrent problems of resistant hypertension and renal dysfunction, "drive-by" renal angiographies are increasingly being performed for screening of RAS (15 to 22% cases of RAS diagnosed for all the coronary angiographies done for suspected cases of CAD).^{9,10} Although this protocol for screening and correction of RAS abounds with controversy with regards to its safety, accuracy, and availability, it works out more favorably in some patients who do not require an additional test to confirm diagnosis.

We need more robust data to guide the selection and feasibility of interventional procedures for the correction of atherosclerotic renal artery stenosis (ARAS),

especially in older patients for whom this problem is becoming more rampant and challenging. Angiotensin-converting enzyme (ACE) inhibitors have been shown to increase renal function and decrease mortality in RAS patients over the long term.¹¹ Medical regimens, employing newer antihypertensive medications for the management of right ventricular hypertrophy (RVH), have increased the likelihood of achieving good blood pressure control from 46% in the past to higher than 90%, thus decreasing the role of invasive management. However, more randomized trials are warranted to study the best medical therapy versus the interventional treatments for RAS patients. One such study is being funded by the National Institutes of Health (NIH), the Cardiovascular Outcomes for Renal Atherosclerotic Lesions (CORAL) Study, with expected results by 2010.⁹ One of our aims as we set out to write this review article was to refresh our knowledge about the basic understanding and role of percutaneous revascularization procedures for renovascular hypertension from the perspective of interventional radiology.

PREVALENCE AND PROGRESSION OF RAS AND ITS RELATED OUTCOMES

The burgeoning problem of RAS is evident from the Medicare data, which shows that from 1996 to 2000 the number of PTAs performed jumped from 7660 to 18,520, thus increasing the cost for renal artery interventions by 242%.¹² Despite the fact that RAS cases account for a miniscule 1 to 6% of all the hypertensive cases, the problem is growing in an aging population with atherosclerosis. Twenty-seven percent of all autopsies, 50% of all peripheral angiographies, 12 to 14% of new patients entering renal dialysis programs, 25 to 30% of patients aged over 60 years on dialysis programs have been documented to have RAS.¹⁰ Additionally, in a set of 297 patients who underwent coronary angiography and also a drive-by renal angiography, 28% had RAS with less than 50% narrowing and 19.2% had RAS with at least 50% narrowing.¹³

The disease is more prevalent in the white population with two studies showing the occurrence of RAS in 27 to 45% of cases of severe hypertension as opposed to only 8 to 19% in African American people.

The two most common causes of RAS are atherosclerosis and fibromuscular dysplasia (FMD) (Fig. 1). Other less well-known causes of RAS may be congenital stenosis, aortoarteritis (e.g., polyarteritis nodosa, Takayasu's arteritis), renal artery aneurysm, transplant arteriopathy, trauma, aortic dissection, extrinsic compression by a mass (e.g., tumor), neurofibromatosis, and stenosis due to a fibrous band.¹⁴ RAS may progress to the commonly encountered clinical entities of renovascular hypertension and/or ischemic nephropathy. Several studies have been undertaken in the past to estimate the disease progression

Figure 1 Right renal angiogram showing irregular beaded appearance of the proximal renal artery of fibromuscular dysplasia.

and follow-up of RAS cases. Tollefson and Ernst showed in a cohort of 48 patients followed up on arteriography for 7.3 years (mean) a progression of 53% and occlusion rates of 9%.³ This study was open for criticism for its overestimation of the actual numbers: as arteriography was the only reliable tool for follow-up of RAS then this might have resulted in a patient selection bias because angiographic studies are done in patients with deteriorating clinical features only. Another study by Zieler et al done on a 132-patient cohort followed up with non-invasive duplex ultrasound over 3 years showed progressive RAS in 7% cases per year on an average; however, the accuracy of results are subject to operator expertise and past experience.¹⁵

ARAS is a progressive disease especially in patients with >60% stenosis at the onset. A large body of observational studies show that progression precedes renal failure, which may be assessed by decreasing renal size, declining glomerular filtration rates (GFR), rising proteinuria, and rising renal functional parameters like BUN (blood urea nitrogen) and creatinine on follow-up studies.^{9,16,17} The best predictor of disease progression is the baseline GFR and /or biopsy proven renal fibrosis score.⁹

RAS is an important cause of hypertension in postrenal transplant patients. Currently, it may be seen in 10% of transplant cases. However, its diagnosis may be complicated by the presence of a concurrent graft rejection, which invariably leads to a vascular disease in the renal arteries, especially during the first year of

surgery. Hypertension and rising creatinine is a common feature to both RAS and rejection; hence, it may be confounding to reach the correct etiology and plan a definitive treatment. With the rising number of renal transplantations due to a varied number of reasons, it may be important to consider RAS in cases of a deteriorated kidney-function posttransplant.

Fibromuscular dysplasia (FMD) needs a special mention here because of its contrasting features to ARAS. It accounts for 10% of all the RAS cases. It is an idiopathic vascular disorder causing early-onset accelerated hypertension in patients 15 to 50 years of age, more commonly in women. About 30% cases of the “medial” variety of FMD progress and rarely cause dissection or thrombosis; hence, these cases rarely advance to ischemic nephropathy.¹⁷

SYMPTOMATIC RAS: HINTS FROM THE HISTORY AND CLINICAL EXAM

ARAS typically presents in an older population who also have other vascular conditions such as chest pain, shortness of breath with acute “flash” pulmonary edema (CAD), transient ischemic attacks, paralysis or carotid bruits (cerebrovascular accident), claudication, abdominal aortic aneurysm, impotence in males, or bowel angina (ischemic bowel disease).^{2,17} The presence of multivessel coronary disease is an important clue leading to the diagnosis of RAS. These patients essentially have multiple predisposing risk factors for atherosclerosis as a part of their clinical profile as discussed previously.

New onset of diastolic hypertension before 35 years or after 55 years of age, rising blood pressure not amenable to medical management (refractory hypertension defined as poor control of blood pressure despite three or more different classes of antihypertensive drugs, one of which is a diuretic) previously well controlled with the same drugs, malignant hypertension with end organ damage (left ventricular hypertrophy with congestive heart failure, visual or neurologic disturbances, and/or grade 4 retinopathy), absent family history of essential hypertension, family history of ARAS, abdominal bruits in the flanks heard in both phases of the cardiac cycle, unexplained hypokalemia and metabolic alkalosis, azotemia accompanied with accelerated hypertension, worsening creatinine levels after treatment of hypertension with ACE inhibitors or angiotensin-2 receptor blocker and with a pole-to-pole size difference of >1.5 cm between the two kidneys on ultrasonography are all suggestive of an underlying RAS.^{2,10} As per the Dutch Renal Artery Stenosis Intervention Co-operative (DRASTIC) Study the most reliable predictors for the recognition of RAS were age, symptomatic vascular disease, elevated low-density lipoprotein (LDL) cholesterol and evidence of an abdominal bruit on clinical examination.¹⁸

Due to the low sensitivity or specificity of the above-mentioned clues, the Joint National Committee (JNC) does not recommend extensive workup for RAS until and unless blood pressure control is not achieved adequately with drugs.¹⁹

In the early and the progressive stages of RAS, GFR and creatinine remain within normal limits.² Their values become dysfunctional (fall in GFR and rise in creatinine) only during the advanced stage of the disease when blood flow has significantly decreased to both kidneys and renal atrophic changes have set in.² Parameters like fractional blood flow and renal size may start declining during the progressive stage even before changes in GFR and creatinine are noticed. Therefore, stress should be placed on renal function with better functional and perfusion studies because GFR and creatinine levels remain unchanged until 50% of the total renal mass is compromised.

It is crucial to label the developing hypertension in a patient as primary (essential) or secondary (as in RAS). This is vital as any revascularization procedure performed to reverse the developing hypertension may only be useful in secondary hypertension cases. Most patients with ARAS have an element of both primary and secondary hypertension present and do not benefit significantly from PTA and stenting procedures. They may however benefit if ARAS is the cause of their ischemic nephropathy and renal failure.²

WORKUP FOR RAS: LABORATORY AND IMAGING STUDIES

An ideal renal imaging modality is one that clearly defines the renal artery course and its accessory branches; detects stenosis and poststenotic dilatation; demonstrates any calcification or thrombus in the renal artery; assesses the renal cortex and renal size; and measures flow and pressure changes in and around the stenotic segment, which may help predict the hemodynamic and functional significance of RAS. To evaluate a patient with suspected RAS, it is best to begin with noninvasive screening tools and investigations rather than invasive methods as these carry added morbidity. Invasive methods are increasingly being reserved for patients who would also need an interventional procedure at the same time.

Noninvasive Functional Evaluation

Basic laboratory tests may be performed to assess the renal function. They include serum creatinine levels, estimation of renal clearance, 24-hour urine collection for obtaining total protein and protein-creatinine ratio values (rules out nephrotic range proteinuria), urine microscopy for red blood cell casts (rules out glomerulonephritis), and serological tests for antinuclear and

antineutrophil cytoplasmic antibodies (rules out systemic lupus erythematosus and other vasculitis).^{2,20}

Physiologic studies to assess the rise of renin are mainly of an academic interest and have limited usefulness. Such studies are useful in FMD-related hypertension in young patients. The studies include peripheral plasma renin activity, captopril-stimulated renin activity, and renal vein renin activity. Renal vein sampling, in particular in a case of unilateral RAS, may help us to lateralize and would be able to show benefits on revascularization, but is again not specific or sensitive.²

Renal scintigraphy scans help in the more accurate diagnosis of RAS and assessment of the overall renal function and differential blood flow to each kidney. Captopril renography with technetium-99 labeled mercaptide (^{99m}Tc-MAG3) and technetium-99-labeled pentetic acid (DTPA) are the more popular nuclear scans. They are more useful in patients with unilateral RAS or RAS in a solitary kidney and/or worsening azotemia because their sensitivity and specificity falls abruptly with bilateral RAS.²¹ A ^{99m}Tc-MAG3 study, if normal, excludes any renovascular hypertension due to RAS; however, its utility is limited in cases of advanced ARAS with creatinine levels >2 mg/dL as it may result in overt renal failure.² A DTPA scan helps in calculating single kidney GFR. Renal scintigraphy scans are best used for the functional assessment of kidneys with RAS and not as the first-line investigation to diagnose RAS in patients with high clinical suspicion.²¹

Noninvasive Anatomical Evaluation of RAS

DUPLEX ULTRASOUND SCAN

A duplex ultrasound scan has the advantage of low operational cost, wider availability, portability, repeatability, and no risk of contrast-induced nephrotoxicity. This makes it ideal to follow up a large number of patients enrolled in prospective randomized trials to study the progression of RAS. It helps to study the "lumenology" and the hemodynamic disarray by measuring parameters like blood flow velocity, distal flow turbulence, Renal Resistive Index (RRI), renal pole-to-pole length and cortical thickness thereby predicting the degree of stenosis and renal atrophic changes.²² Systolic and diastolic velocity peaks, poststenotic spectral wave broadening, and the Renal Aortic Ratio form the most important criteria for the diagnosis of RAS on color Doppler.²³ Computation of the bilateral resistive indices and recognition of the "Parvus-tardus" sign from an early systolic peak is critical to help decide between a conservative or interventional approach to therapy.

COMPUTERIZED TOMOGRAPHY ANGIOGRAPHY

CTA imaging is the next best imaging test for screening for RAS with a high detection rate. With the inception

of spiral and multislice CT scanners it is possible to scan a larger region of interest with a single breath-hold. 3D reconstruction of image sequences using maximal intensity projection (MIP) techniques has further increased the accuracy of reporting proximal main renal artery stenosis with high degrees of confidence at par with invasive angiographies. Rapid injection rates of contrast enable better image quality in the detection of in-stent restenosis CTA scores than an MRA due to artifacts with the latter. CTA is however less reliable than conventional angiography in detecting arterial dysplasia and accessory renal arterial and distal intrarenal lesions with good accuracy. The disadvantages with CTA are few, but important, including limited use in the determination of poor renal function based on the need for a high volume of contrast, the risk of high radiation dose, and artifacts in the case of calcified ARAS.

MAGNETIC RESONANCE ANGIOGRAPHY

MRA may be used as an intelligent substitute for CTA in patients with renal dysfunction. Gadolinium-enhanced (TRICKS: time resolved imaging of contrast kinetics) or phase-contrast MRA delineates the proximal renal artery anatomy with high accuracy.²¹ With cardiac synchronization 3D MRA shows even better results. MRA carries almost the same diagnostic accuracy as CTA and is fast becoming a clinical standard as it is devoid of radiation dose and the risk of contrast-induced nephrotoxicity. Gadolinium-enhanced MRA also showed better characterization of accessory renal artery stenosis.²¹ The combination of phase-contrast sequence with gadolinium-enhanced MRA better its image quality.²¹ Furthermore, measurement of renal flow by a phase-contrast MRA and renal volume size on morphologic imaging yields a Renal Flow Index, which helps to predict the outcomes of interventional revascularization for RAS preprocedure.²¹ This information is beneficial as otherwise one has to rely only on the clinical profile to predict such outcomes.

As noted above, the major current limitation of MRA concerns patients with renal insufficiency and the risk of nephrogenic systemic fibrosis. Additional limitations of MRA include its high cost, availability, motion artifacts due to longer acquisition time, and the presence of metallic implants in the body including renal stents (except nitinol and platinum stents, which do not cause much artifacts). MRA like CTA is not reliable for detecting distal intrarenal lesions and FMD versus invasive arteriography. It is crucial to recognize this classical "ring-of-beads appearance" of FMD on vascular imaging as it shows very promising results with percutaneous interventions.²

Invasive Anatomical Evaluation of RAS

Conventional catheter angiography is the current gold standard for the diagnosis of RAS. It is particularly

helpful in diagnosing RAS in the setting of variant renal artery anatomy with distal intrarenal lesions and stenosis due to FMD (Fig. 1). It has the advantage of allowing the performance of an endovascular therapeutic procedure in the same sitting. Postcontrast angiographic phase marks out the details of renal circulation and nephrographic phase depicts the kidney size and shape. Thus, it gives us information both about the anatomic and functional consequence of RAS. A pressure gradient can be measured, which provides a guide for therapy. The high amount of contrast used poses a major concern in patients with borderline renal function. An arterial puncture site may bleed profusely and the procedure involves a risk of cholesterol embolism as well. Fluoroscopic radiation dose may be an additional side effect.¹⁰ Complication rates range between 6 and 14% for most studies. Employment of low osmolar contrast media, digital subtraction angiography, gadolinium and carbon dioxide angiography may help in curbing the adverse affects.

PERCUTANEOUS INTERVENTION

Angioplasty and stenting are the percutaneous techniques used in the treatment of obstructive lesions of the renal artery that cause hypertension.

If possible, long-acting antihypertensive medications should be stopped or the dosage decreased. Patients with renovascular hypertension can have some impairment of renal function, thus renal protection from contrast-induced nephropathy (CIN) is of utmost importance. Instructions should be given for proper oral hydration. Intravenous hydration with normal saline can be pursued at a rate of 1 to 1.5 cc/kg/hour starting 12 hours prior to the procedure and extending 12 hours beyond it.

N-acetyl cysteine (Mucomyst®; Bristol Myers Squibb, Anagni, Italy) in a dose of 600 mg bid for 2 days can be given to reduce CIN. Sodium bicarbonate may reduce the risk of CIN although this remains unproven. Fenoldopam a specific dopamine A1 receptor agonist has shown to benefit in some circumstances. In addition, judicious use of dilute 1/2 to 1/3 strength) nonionic, isoosmolar contrast may be used. Alternative contrast agents such as CO² are available, but image quality is not as good as conventional contrast agents. Patients receive 325 mg of aspirin the day prior to the procedure. In patients with atherosclerotic RAS, Plavix® (Bristol-Myers Squibb, New York, NY) 300 mg is initiated the day before and 75 mg on the day of procedure. Patients who receive stents are maintained on 325 mg aspirin for life and 75 mg Plavix for 3 months.

A variety of balloon catheters exist based upon 0.035-inch, 0.018-inch, and 0.014-inch platforms. Smaller systems are more flexible, lower profile, can track better, and are available in both coaxial and monorail

(rapid exchange) systems. However, most of the stenting data published are based on a 0.035-inch system. With the rapid exchange system, shorter guidewire can be used, thus making the procedure easy to perform as well as having better fluoroscopic visibility. Previous generations of 0.014-/0.018-inch system stents had less hoop strength in comparison to a 0.035-inch system although current systems are more robust.

A femoral artery approach is most common; however, a brachial or radial approach can be used in special circumstances. 6- to 8-French guiding sheaths are used. They can be preshaped, have soft radiopaque

tips, tapered dilators, and have side ports to allow for contrast injection around guidewires. With a brachial or radial approach, long (60- to 80-cm) guiding sheaths are used. Abdominal aortography is typically performed, although the same information can be gleaned from cross-sectional imaging studies. In general, aortography should be imaged in 15 to 20 degrees left anterior oblique (LAO) position to profile the origins of the renal arteries.

A reverse-curved catheter (SOS 2) is used to select the renal artery and the lesion is crossed with a soft-tip guidewire (Bentson; Cook, Inc., Bloomington, IN) or a

Figure 2 (A) Aortogram showing bilateral renal artery stenosis. On the right is ostial stenosis and on the left is midrenal artery stenosis. (B) Right renal stent placement. (C) Left balloon angioplasty. (D) Postprocedure aortogram of the same patient shows normal caliber of bilateral renal arteries. Also seen is an aortic stent graft.

steerable guidewire (Terumo Medical Corp., Somerset, NJ), keeping the distal tip of the wire in the main renal artery as the catheter is advanced across the lesion to avoid perforation. At this point or prior to crossing lesions, 3000 to 5000 IU of heparin is given intravenously. Once across the lesion, a decision to use a 0.035-inch or 0.014-/0.018-inch system is made. We usually use a 0.035 Rosen guidewire (Cook, Inc., Bloomington, IN) and a 6-French guide sheath is advanced over the guidewire followed by balloon dilatation of the lesion (Fig. 2). If a 0.018-inch system is used, a platinum tipped guidewire is inserted and advanced through the reverse-curved catheter and guiding sheaths that come with the system. The procedure is similarly performed as a 0.035 system using the rapid exchange system.

Predilatation of the lesion allows the determination of the appropriately sized balloon, the resistance and stability of the balloon, and the amount of discomfort to the patient with balloon inflation. In FMD and some atherosclerotic lesions, angioplasty alone is therapeutic. Primary stenting may be performed when there is a recurrence of lesion shortly after percutaneous transluminal renal angioplasty. Embolic protection systems are also being studied to reduce cholesterol embolization. Double guidewire access and a "kissing balloon" technique may be used in early bifurcating arteries. When using double guidewires, accessing both the femoral artery may be required, alternatively two 0.014-inch monorail systems can be advanced through a 6-French guiding sheath.

In lesions involving a small renal artery, tortuous vessel, difficult access, and a branch vessel, a 0.014-/0.018-inch system is useful. For an atherosclerotic ostial lesion, balloons are slightly undersized to minimize the risk of dissection. Eventually, the lesion is overdilated by ~20% for an optimum approximation of stent to the vessel wall and to optimize the poststent minimal lumen diameter (MLD). To minimize vasospasm, 100 to 200 μ g of nitroglycerine may be given directly into the renal artery.

For resistant lesions like intimal fibroplasia and neurofibromatosis, a smaller diameter balloon with a gradual increase of balloon size is done to minimize the risk of rupture. In such cases, the use of a cutting balloon has been reported. In cases of borderline RAS, intraarterial pressure measurements are obtained and a peak systolic gradient of 10% or more or a 20 mm Hg absolute pressure gradient is an indication of hemodynamically significant stenosis. A 50% stenosis corresponds to a 22 mm Hg systolic pressure gradient. Other supporting tests are lateralizing of renin, an abnormal renal Doppler study, or ACE inhibitor nuclear scan. Following intervention, a residual gradient of pressure of less than 5 mm Hg is acceptable.

Ostial lesions usually require stent placement. Most often a balloon-expandable stent is used. For an

ostial lesion, they are typically 5 to 7 mm in diameter and 1.5 to 2 cm in length. Deployed stents should protrude by 1 to 2 mm into the aorta for optimum results.

The most serious complication is renal artery rupture with an incidence of less than 1%. Once identified, reinflation of the balloon is life saving and after the patient is stabilized, the options are stent-graft placement or surgery. Other complications include atheroembolism, renal function derangement, and infection. Restenosis is sequelae that may require attention. The mean incidence of complication is 14%, most of which is not life threatening. The combined incidence of 30-day mortality, loss of kidney, renal artery rupture, occlusion of main renal artery, and need of surgery should be less than 4%.

Results of intervention depend on the cause of renovascular hypertension. FMD is more amenable to intervention than atherosclerotic RAS. The technical success of angioplasty for hypertension ranges from 40 to 90% and clinical success ranges from 50 to 85% depending on the cause. Stenting for renovascular hypertension has a technical success of 95% and a primary clinical success rate of 70 to 80%. Restenosis is higher in women, in patients with a smaller renal artery, and in patients who smoke poststenting.

In summary, angioplasty appears to be of benefit in the treatment of renovascular hypertension in a defined subset of patients. The group that is most likely to benefit are those with easily controlled hypertension, older patients without renal insufficiency, and patients with RAS of less than 50%. This makes it highly controversial and the CORAL is in progress to answer these queries. Stenting is beneficial for ostial atherosclerotic RAS, causing hypertension in 70 to 80% cases.

CONCLUSION

Arterial intervention in patients with renovascular hypertension is a safe and effective procedure. It requires appropriate patient selection for good results in the present setting of improved blood pressure control with the newer antihypertensive regimens.

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