ELSEVIER

Techniques in Vascular and Interventional Radiology

Management of Renovascular Hypertension

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Renal artery stenosis is a potentially reversible cause of hypertension, and transcatheter techniques are essential to its treatment. Angioplasty remains a first-line treatment for stenosis secondary to fibromuscular dysplasia. Renal artery stenting is commonly used in atherosclerotic renal artery stenosis, although recent trials have cast doubts upon its efficacy. Renal denervation is a promising procedure for the treatment of resistant hypertension, and in the future, its indications may expand.
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Introduction

Hypertension, defined as systolic blood pressure greater than or equal to 140 mm Hg or diastolic blood pressure greater than or equal to 90 mm Hg, is a common condition, affecting approximately 25%-30% of the adult population in the United States.[1] Although most hypertension cases are "essential"—meaning that no primary cause can be identified—approximately 3%-5% of patients with hypertension have renovascular etiologies.[2] In these patients, renal artery stenosis (RAS), defined as narrowing of the renal arterial lumen by more than 50%, results in decreased blood flow to the kidneys and activation of the renin-angiotensin system, which in turn increases systemic blood pressure.[3] In addition, decreased renal perfusion can lead to decreased renal function and chronic kidney disease, an entity referred to as ischemic nephropathy.

Upwards of two-thirds of cases of RAS are caused by atherosclerosis.[4] Less commonly, RAS occurs secondary to fibromuscular dysplasia (FMD), which represents a range of pathologic diagnoses differentiated by the layer of the arterial wall that they affect. Medial fibroplasia is the most prevalent subset of FMD, constituting 75%-80% of all fibrous lesions of the renal artery. Less common entities include intimal fibroplasia, perimedial fibroplasia, medial hyperplasia, and adventitial hyperplasia.[5,6] Finally, other processes can lead to RAS including vasculitides such as Takayasu arteritis, neurofibromatosis, or irradiation.

Although medical treatment remains the standard of care for essential hypertension, angioplasty, stenting or both are often used in the treatment of renovascular hypertension. The indications for renovascular interventions are evolving and are dependent upon the etiology of RAS.[2] In general, stenosis secondary to FMD is treated with percutaneous renal artery transluminal angioplasty (PRTA), whereas stenosis secondary to atherosclerosis is treated with angioplasty or stenting or both. The indications for revascularization, however, are more controversial for atherosclerotic RAS than they are for RAS secondary to FMD.[7]

More recently, renal denervation (RDN), also known as catheter-based renal arterial sympathectomy, is emerging as a potentially useful procedure for the treatment of resistant hypertension. Resistant hypertension is defined as hypertension that persists despite adherence to an adequate and appropriate triple drug regimen.[2,8,9] Although the pathophysiology of resistant hypertension has not entirely been elucidated, there is growing consensus that excess sympathetic tone in the renal arterioles is contributory.[10] Transluminal ablation of the renal arterial sympathetic encircling the adventitia of the renal artery has been proposed as a potential treatment for this problem, although results, thus far, have been mixed.

This article reviews the presentation, workup, and management of patients with renovascular hypertension.

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Clinical Presentation

The recommendations of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC 7), last updated in 2003, argue against testing for identifiable causes of hypertension unless adequate blood pressure control cannot be achieved. However, the report further argues "reversible causes of renal failure always should be sought and treated."[1] Given this background, it is not always clear when to screen for RAS.

Certain clinical characteristics are classically taught to be associated with RAS and may warrant further workup, and include (1) the onset of hypertension in younger ($\leq$30 years) or older ($\geq$ 55 years) patients, (2) hypertension in patients with recurrent flash pulmonary edema, (3) and accelerated or resistant hypertension.[2] Hypertension associated with renal insufficiency suggests ischemic nephropathy, and hypertension in patients with known atherosclerotic disease suggests atherosclerotic RAS. Both scenarios warrant screening.[11] A physical examination finding that may point to RAS is the presence of an abdominal bruit, particularly one that continues into diastole and is lateralized.[2]

Patient demographics data can help determine the etiology of RAS. Atherosclerotic RAS should be suspected in older patients, and those with known atherosclerotic disease elsewhere. FMD should be suspected in younger patients, particularly in younger females.[12]

Imaging Studies

Once RAS is suspected, the diagnosis is confirmed with imaging, as laboratory tests such as plasma renin concentration lack specificity and are no longer recommended.[13]

Ultrasound

Duplex ultrasonography is commonly employed as a first-line imaging modality in the workup of RAS, because it is noninvasive and does not use ionizing radiation (Fig. 1). Its major drawback is its operator dependence. Grayscale ultrasonography may allow the direct visualization of a stenosis. More often, a nonvisualized stenosis can be inferred by analyzing the adjacent spectral waveforms. Proximal (upstream) stenosis should be suspected when the renal artery peak systolic velocity (PSV) is increased to 100-200 cm/s, or the renal-to-aortic PSV ratio is greater than 3.5. Turbulent flow may be seen in the poststenotic area. Severe stenosis can result in parvus-tardus (weak and delayed) waveforms, and visualization of the renal artery without detectable Doppler signal indicates renal artery occlusion.[14] Distal stenosis should be suspected when early systolic peak acceleration is blunted, characterized by an acceleration index of less than 4 m/s^2, an increased time to PSV of less than 0.07 seconds, or greater than a 5% difference in renal resistive indices between kidneys.[15]

Computed Tomographic and Magnetic Resonance Angiography

Computed tomographic angiography (CTA) and magnetic resonance angiography (MRA) are additional noninvasive modalities that are useful in the workup of suspected RAS. CTA has superior spatial resolution to MRA, but uses ionizing radiation, is contraindicated in patients with contrast allergies, and has been reported to cause contrast-induced nephropathy in patients with poor renal function. Severely calcified lesions may be underestimated on CTA.[4] MRA does not use ionizing radiation, but it is contraindicated in patients with certain implantable devices and can induce claustrophobia. The use of gadolinium-based contrast agents must be avoided in patients with poor renal function because of the risk of nephrogenic systemic fibrosis.

Digital Subtracted Angiography

Diagnostic angiography remains the gold standard for the diagnosis of RAS because of its superior spatial resolution; however, any catheter-based procedure carries inherent risks. The Society of Interventional Radiology recommends catheter-based angiography whenever the previously mentioned screening indications for RAS are met, in addition to 1 or more of the following: (1) RAS previously diagnosed on noninvasive imaging, (2) noninvasive

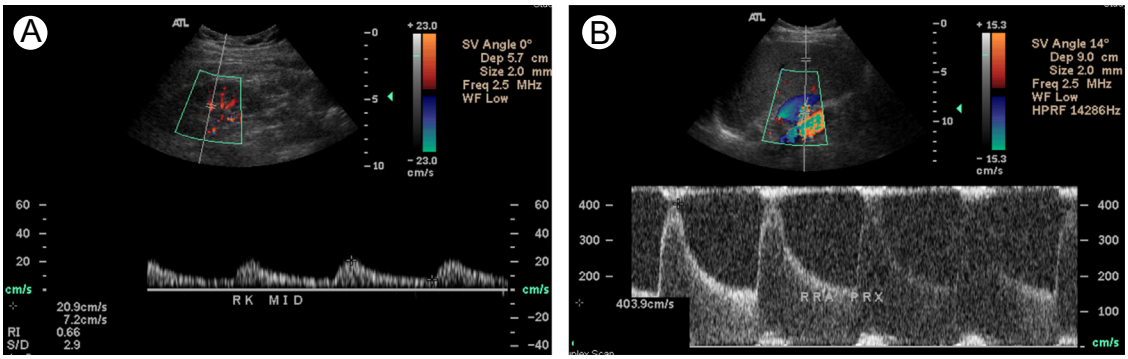

Figure 1 Renal duplex ultrasonography in this patient with right renal artery stenosis demonstrating (A) parvus-tardus waveforms in the intraparenchymal renal artery branches and (B) an elevated peak systolic velocity. (Color version of figure is available online.)