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Renovascular Hypertension: Choice of Treatment Options

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Renovascular hypertension (RVH) is one of the most common causes of secondary hypertension which is responsible for renin-angiotensin system (RAS) blockade. It accounts for 1~5% of all causes of hypertension, and 5% of secondary hypertension in adults. RHV is usually asymptomatic before at least 60% of the luminal diameter of the renal artery that develops gradually in the elderly. The most common cause of RHV in the elderly is atherosclerotic renal artery stenosis in the context of systemic atherosclerosis related to plaque. It is found from the asymptomatic imaging on screening. Still, it can show early onset of hypertension, refractory hypertension, unilateral contracted kidney, progressive renal function decline, sudden hyperkalemia or election of serum creatinine after RAS inhibition, and recurrent acute pulmonary edema. Fibromuscular dysplasia (FMD) could cause RVH in young females or patients with connective tissue disease.

The treatment strategies for RVH depend on a characteristic of the disease. Patients with FMD or focal renal artery stenosis are amenable to percutaneous balloon angioplasty and/or endovascular stent application. However, elderly patients with diffuse atherosclerotic renovascular hypertension can apply multi-targeted control of hypertension, obesity, dyslipidemia, and metabolic syndrome including dyslipidemia and diabetes. There is still a lack of well-established benefits of angioplasty for atherosclerotic RHV, but the American College of Cardiology (ACC) and American Heart Association (AHA) recommend criteria that patients are most likely to gain benefit from renal artery stenting.

Table 1. Indications for renal artery stenting in RVH

Patients with RVH should be treated with multi-disciplinary and medical treatment, but patients with significant stenosis, refractory hypertension, and declining renal function are reasonable candidates for renal artery stenting. Solving end-organ ischemia is also an important strategy for protecting against hypertension and renal dysfunction, but further randomized trials for CKD would guide the optimal treatment of RVH.

Table 1

Class I	level of evidence B	Flash pulmonary edema, unstable angina with > 10 mmHg gradient
		Recurrent CHF with unilat. mod. stenosis > 10 mmHg (maybe appropriate)
Class IIa	level of evidence B	CKD with bilateral stenosis with > 10 mmHg gradient (kidney size > 7 cm)
		Resistant hypertension with bilateral stenosis
		Resistant hypertension with unilateral stenosis (maybe appropriate)
Class IIb	level of evidence B	CKD stage IV and global renal ischemia (severe stenosis)
		Asymptomatic unilateral or bilateral stenosis (rarely appropriate)