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Review Article

Renovascular Hypertension -

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ABSTRACT

Renovascular hypertension is the most common secondary hypertension.Atherosclerosis and fibromuscular dysplasia are the most common causes. Activation of the renin angiotensin aldesteron system secondary to renal artery stenosis is responsible for the pathogenesis. The first choice in diagnosis is Doppler ultrasound. The gold standard diagnostic method is conventional standard angiography.Lifestyle changes are recommended primarily in treatment. Low-dose aspirin and statin therapy is important in atherosclerotic stenosis. Angiotensin coverting enzyme inhibitors, thiazide diuretics and calcium channel blockers can be used in drug therapy. Revascularization is recommended in resistant hypertension, worsening renal function and recurrent episodes of pulmonary edema.

Keywords: Renovascular hypertension; Atherosklerosis; Fibromuscular dysplasia; Drug therapy; Revascularization

INTRODUCTION

The definition of Hypertension (HT) differs in American and European guidelines. In the American guideline, office/clinical/daily mean blood pressure is accepted as HT of 130/80 mmHg and above, whereas in the European guideline, office/clinical blood pressure is accepted as HT of 140/90 mmHg [1]. In addition, the average daily blood pressure of 135/85mmHg and above is accepted as HT in the European guideline [1]. The cause of 90% of HT is unknown. Secondary reasons are responsible for 10% of HT. The most common cause of secondary HT is Renovascular Hypertension (RVHT). It constitutes 1-2% of hypertensive cases [1,2]. Renovascular disease (RVH) is characterized by stenosis in one or more branches of the renal artery outside the kidney.

Common causes of stenosis are Atherosclerosis (ATS) and Fibromuscular Dysplasia (FMD) [1]. Other rare causes include renal artery dissection due to trauma, aortic dissection, and tumor. Atherosclerotic disease is responsible for 75-80% of the cases. It is especially seen in elderly men and involves the proximal 1/3 of the renal artery. FMD is mostly seen in young women and involves the distal 2/3 of the renal artery. 90% of the renal artery involves the media layer. Aneurysmatic dilatations in the form of a rosary bead are seen in renal angiography. FMD progresses more slowly than atherosclerotic lesions [1-3]. Bilateral renal artery stenosis is less common than unilateral renal artery stenosis. Now let's examine the pathogenesis of RVHT.

ETIOPATHOGENESIS

The most common causes of RVHT are ATS and FMD. ATS is seen equally in both sexes, while FMD is more common in women. ATS is more common over the age of 50, while FMD is more common between the ages of 20-40. While total occlusion is common in ATS, it is rare in FMD. While ischemic atrophy is common in ATS, it is rare in FMD. While the efficiency of percutaneous transluminal renal agioplasty is low in ATS, it is high in FMD [2-4]. The cause of high blood pressure in RVHT; It is the activation of the renin-angiotensin-esterone system, which starts with the release of renin from the juxtaglomerular cells in the afferent arterioles secondary to the decrease in renal blood flow. Sodium and water reabsorption increases as a result of this activation. Thus, body fluid volume and cardiac output increase. In addition, activation of this system causes all arterioles to contract and increase peripheral vascular resistance. However, in studies conducted, a gradient of 10-20 mmg is required between the aorta and the renal artery for renin release [2].

Studies have shown that 70-75% lumen narrowing is required for a decrease in renal perfusion pressure against an occlusive lesion [3]. This level of stenosis leads to a 40% reduction in perfusion pressure. This situation leads to the loss of renal autoregulation [4].

DIAGNOSIS

There are certain clinical clues that suggest RVHT. Sudden onset and progressive hypertension, papillary edema, oliguria, resistant hypertension, onset of hypertension over the age of 60, recurrent episodes of acute pulmonary edema, faster than expected deterioration of renal functions with angiotensin converting enzyme or angiotensin receptor blocker (increase in creatinine more than 30%) and cystolo-diastolic murmur in the renal artery trace in the abdomen on physical examination are the clues that suggest RVHT [4-6]. Screening for RVHT should be performed in patients with these characteristics.

For this screening, non-invasive renal Doppler ultrasonography with high accuracy should be used primarily. In this examination, the renal-aortic ratio is > 3.5, the peak systolic velocity is > 200 cm/sec, and the resistance index < 0.5 suggests a stenosis with a probability of 60-99% [7]. In addition, one kidney shrinking more than 1.5 cm in renal ultrasound is one of the findings of RVHT [4]. However, renal ultrasound is largely operator dependent and not diagnostic in obese patients. In these cases, it is more appropriate to use magnetic resonance angiography and spiral CT angiography [6,8,9].

The gold standard test in renal artery stenosis is conventional angiography. Digital subtraction angiography improves the image quality by distinguishing the contrast-filled vessel from bone structures and tissues [6]. Carbon dioxide digital subtraction angiography can be used in patients with renal failure and allergy to iodinated contrast material. In conventional standard renal angiography, stenosis of 70% or more is hemodynamically significant.

Certain parameters are used to determine the hemodynamic significance in strictures between 50-70%. The lesion is hemodynamically important if the resting translesional mean pressure gradient is > 10 mmHg, the hyperemic peak systolic pressure gradient is > 20 mmHg, or the renal fractional flow reserve is 0.8 or less. The translesional gradient should be measured with a nonobstructive catheter of 4F and below [6,7]. Hyperemia is performed with an intrarenal bolus of 30 mg papaverine or 50 microgram/kg dopamine. The sensitivity and specificity of captopril renal scintigraphy is low and it is not the first choice [6].

TREATMENT

Optimization of antihypertensive treatment and all cardiovascular risk factors constitutes the first step of treatment in RVHT. Smoking cessation, weight loss, diabetes control, statin therapy and low-dose aspirin are important in treatment [4]. Angiotensin converting enzyme inhibitors are safe and effective in unilateral renal artery stenosis. However, it should be started at low doses and the dose should be increased slowly, following renal functions. These drugs

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are contraindicated in bilateral renal artery stenosis. When blood pressure control is not achieved, thiazides and calcium channel blockers can be used.

Medical treatment is recommended with a class 1 indication in atherosclerotic renal artery stenosis [8]. Revascularization with class 2b indication is recommended in resistant hypertension, worsening of renal functions and/or resistant heart failure and non-atherosclerotic diseases (such as fibromuscular dysplasia) [8].

Percutaneous transluminal angioplasty is the treatment of choice in fibromuscular dysplasia. Despite its high success rate, 25% of restenosis is seen [10-12]. Stent implantation should not be performed unless necessary, such as vascular dissection. Surgery should be considered in case of failure or in cases of complex anatomy. The success rate of percutaneous transluminal angioplasty in atherosclerotic lesions is low and restenosis rates are high. For this reason, stenting procedure, which reduces stenosis rates, should be performed together with angioplasty. Patients who undergo percutaneous transluminal renal artery angioplasty, with or without stenting, are given dual antiplatelet therapy for 1 month.

In the STAR and ASTRAL studies, no difference was observed between atherosclerotic renal artery intervention and medical therapy in terms of serum creatinine values, reduction in systolic blood pressure, and frequency of renal events [10-13]. Stenting did not influence eGFR in participants with atherosclerotic RVHT receiving renin angiotensin aldesteron system inhibitor. Predictors of clinical events were traditional risk factors for chronic kidney disease and cardiovascular disease [14]. There was no difference between the two groups (medical and interventional) in terms of hospitalization for heart failure. As a result, if renal functions can be preserved, blood pressure control can be achieved, if renal artery stenosis is not too advanced and the person has a history of hypertension for a long time, drug therapy should be considered first [15,16].

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