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Renal Artery Intervention

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Renal artery stenosis (RAS), hypertension and renal insufficiency (RI) are each frequently present especially in the elderly population. RAS is often present without any clinical signs or symptoms and even when hypertension or renal insufficiency are also present, they may be coincidentally rather than causally related. However, when RAS is hemodynamically or physiologically significant, it is one of the few potentially reversible causes of RI and hypertension.

The challenge for physicians is to identify patients with RAS who would benefit from renal revascularization, whether by interventional techniques or open surgery. RAS is often clinically silent, at least until it becomes hemodynamically significant when it can produce renal vascular hypertension (RVH) or RI.

Selection for intervention must consider and integrate the clinical, anatomic and physiologic status of the patient and the risk and benefit of alternative medical and invasive therapies must be compared to each other and to the natural history of the disease. Prior to renal artery intervention the physiological significance of a stenosis should be confirmed by demonstrating a hemodynamically significant 10% peak systolic trans-stenotic pressure gradient, in addition to appropriate clinical and anatomic indications. RVH, especially if due to unilateral disease, is usually easily and well controlled by modern anti-hypertensive medications, especially angiotensin converting enzyme inhibitors (ACEI). Ischemic nephropathy, one of the few reversible causes of RI in adults is the most appropriate indication for intervention in renal artery occlusive disease.

All of these hemodynamic and humoral mechanisms contribute to major adverse cardiovascular events (MACE) such as death, dialysis, myocardial infarction and stroke. The terms RAS and atheromatous renal artery stenosis (ARAS), physiologically or hemodynamically significant RAS and ARAS and RVH are often used interchangeably. Others recommend intervention (stenting) for any RAS even if the stenosis is physiologically and clinically not significant, though Zeller recently acknowledged that antistatin therapy may be an important alternative. It has even been suggested by some in lectures, though not in writing, that any, even physiologically not significant RAS can produce adverse cardiovascular effects by mysterious hemodynamic and humoral effects. There is no evidence to support this concept.

Some advocate prophylactic intervention for asymptomatic and hemodynamically non significant RAS lesions based on the following:

1. ARAS is a progressive disease and "progressive worsening of ARAS stenosis occurs despite medical therapy that effectively controls blood pressure"
2. Therefore, unless prophylacticaly and aggressively treated by mechanical (stenting) intervention, ARAS will progress to cause severe hypertension and renal insufficiency with their attendant MACE at which point the disease is no longer reversible or controllable by interventional treatment.
3. Early prophylactic intervention in a less diseased renal artery and aorta is techni-

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cally easier and less likely to result in complications.

These arguments are at first persuasive, but are based on inadequate or incorrect assumptions or information much of which is no longer current.

Most of the studies on the progression of renal artery stenosis overestimate the progression of atheromatous renal artery disease; they predate the widespread aggressive use of statins, exquisite control of RVH by drugs specifically targeting renin dependent hypertension, the aggressive control of glucose and efforts at lifestyle modifications affecting smoking, diet, exercise and weight control especially in high risk populations. In fact, several large studies have recently confirmed the arrest of progression and in some cases regression of atheromatous plaque in the coronary and carotid arteries by aggressive treatment with statins; there is no reason to postulate that these beneficial effects do not apply to the renal circulation. It seems reasonable then to treat all patients with such asymptomatic and hemodynamically non significant ARAS lesions with aggressive statin therapy, aspirin and other appropriate medications and lifestyle modifications. This would likely produce all the potential benefits of aggressive interventional stent treatment whose very long term durability and fate is unknown and without any of its known major risks such as cholesterol embolization and without the 15-20 % restenosis all of which can occur even in "low risk" patients.

Statins and risk reduction are the new paradigm for the treatment of all atheroma, especially when clinically and hemodynamically not significant, but also for clinically and physiologically significant stenoses following interventional treatment.

In summary, appropriately selected patients with ARAS and RI benefit from interventional therapy, while those with clinically and hemodynamically non significant stenoses should be treated by aggressive medical therapy including statins and life style modifications.

ANGIOPLASTY IN NON-ATHEROMATOUS LESIONS

FIBROMUSCULAR DYSPLASIA (FMD)

Generally, percutaneous transluminal angioplasty is considered to be a method of choice for FMD treatment. Stents are usually not used as a primary measure, but they are helpful in renal angioplasty suboptimal result or failure.

The main clinical symptoms of FMD are hypertension and ischemic nephropathy, but this is less frequent. In technically successful cases, the positive clinical response occurs in over 90% of cases. Stent implantation is used rarely, mainly as a bail-out procedure in angioplasty complication or failure.

ARTERITIS

Stenoses usually affecting the longer segments of main renal artery respond favorably to angioplasty, and the dilation should not be performed during active period of the disease. Stents are not used very frequently.

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