JACC: CARDIOVASCULAR INTERVENTIONS
VOL. 2, NO. 3, 2009
© 2009 BY THE AMERICAN COLLEGE OF CARDIOLOGY FOUNDATION
ISSN 1936-8798/09/$36.00
PUBLISHED BY ELSEVIER INC.
DOI: 10.1016/j.jcin.2008.10.014

STATE-OF-THE-ART PAPERS

Refining the Approach to Renal Artery Revascularization

Robert D. Safian, MD, FACC, Ryan D. Madder, MD

Royal Oak, Michigan

Renal artery stenosis (RAS) is caused by a heterogenous group of diseases with different pathophysiology, clinical manifestations, treatment approaches, and outcomes. The 2 most common forms of RAS are fibromuscular dysplasia (FMD) and atherosclerosis (ARAS). Renovascular syndromes are broadly classified into renovascular hypertension and ischemic nephropathy, but these terms are misleading, because they imply a causal relationship between RAS, hypertension, and renal dysfunction, which is difficult to prove in humans. Data supporting renal revascularization are limited by heterogeneous causes of hypertension and renal dysfunction, insufficient understanding of the relationship between RAS and nephropathy, inconsistent techniques for revascularization, ambiguous terminology and end points to assess benefit, and lack of large-scale randomized trials. The purpose of this review is to enhance understanding of the epidemiology, clinical markers, and diagnosis of RAS; the relationship between RAS and important disease states; the distinction between renal ischemia and nephropathy; optimal revascularization techniques; and avoidance of renal injury. (J Am Coll Cardiol Intv 2009;2: 161–74) © 2009 by the American College of Cardiology Foundation

Renal artery stenosis (RAS) is caused by a heterogenous group of diseases with different pathophysiology, clinical manifestations, treatment approaches, and outcomes. The 2 most common forms of RAS are fibromuscular dysplasia (FMD) and atherosclerosis (ARAS), whereas inflammatory disease of the arterial circulation and congenital abnormalities are far less common (Fig. 1). Traditionally, renovascular syndromes have been broadly classified into 2 categories: renovascular hypertension and ischemic nephropathy. These categories are potentially misleading, because they imply a causal relationship between RAS and hypertension or renal dysfunction, respectively. Although causal relationships are evident in experimental models of RAS, they are more difficult to prove in human diseases. Furthermore, a causal relationship suggests that revascularization of RAS should favorably impact blood pressure and renal function, yet available clinical data have failed to demonstrate unequivocal benefits of renal revascularization. The purpose of this review is to place RAS in appropriate perspective, particularly with regard to renal revascularization and the importance of renal ischemia and nephropathy. This

See page 183

perspective should incorporate understanding of the epidemiology, clinical markers, and diagnosis of RAS; establish a relationship between RAS and important disease states; distinguish renal ischemia and nephropathy; use optimal revascularization techniques; and avoid renal injury.

Epidemiology of RAS

FMD. Fibromuscular dysplasia is an uncommon disease of unknown etiology; typically occurs in women <30 years of age; and often affects the renal, carotid, and femoral arteries. Fibromuscular dysplasia should be considered in young patients if severe hypertension is not associated with obesity, oral contraceptives, or

From the Department of Cardiovascular Medicine, William Beaumont Hospital, Royal Oak, Michigan.

Manuscript received July 18, 2008; revised manuscript received September 29, 2008, accepted October 10, 2008.

JACC: CARDIOVASCULAR INTERVENTIONS, VOL. 2, NO. 3, 2009
MARCH 2009:161–74

known renal parenchymal disease. Unilateral or bilateral renal FMD might cause renovascular hypertension, but renal failure is unusual (1).

ARAS. Atherosclerotic renal artery stenosis is a common clinical entity, affecting 7% of patients older than age 65 years and 60% of patients with hypertension, coronary or peripheral artery disease, and renal insufficiency (2). Unlike FMD, ARAS rarely causes renovascular hypertension but is commonly associated with renal dysfunction (3).

Clinical Manifestations of RAS

Hypertension and cardiovascular manifestations. Hypertension manifestations include onset of severe hypertension at age <30 years (FMD) or at age >55 years (ARAS) and resistant, accelerated, or malignant hypertension (3). Cardiovascular manifestations usually occur in the setting of malignant hypertension. The classic manifestation is "flash" pulmonary edema not explained by coronary artery or valvular disease, especially if left ventricular function is normal (4). Other cardiovascular manifestations include severe hypertension associated with acute coronary syndromes, acute aortic syndromes, stroke, transient cerebral ischemia, intracranial hemorrhage, encephalopathy, and papilledema.

Renal manifestations. Renal ischemia might present as acute renal failure, with a rise in serum creatinine within 14 days of initiation of angiotensin-converting enzyme inhibitors or angiotensin receptor blockers. Although considered a marker for bilateral RAS, this observation is neither sensitive nor specific for RAS (5). Other renal manifestations are subtle or insidious, including unexplained chronic renal failure, small kidney, and asymmetry in renal dimensions (2). Ischemic nephropathy is an important cause of chronic kidney disease and end-stage renal disease, representing the primary etiology of end-stage renal disease in 5% to 15% of patients initiating dialysis each year (6).

Abbreviations and Acronyms

ACC = American College of Cardiology
AHA = American Heart Association
ARAS = atherosclerotic renal artery stenosis
CTA = computerized tomography angiography
FMD = fibromuscular dysplasia
GFR = glomerular filtration rate
MRA = magnetic resonance angiography
RAS = renal artery stenosis
RRI = renal resistive index
TLG = translesional pressure gradient
^{99M}Tc-DTPA = technetium-labeled pentetic acid

Assessment of RAS and Its Clinical Significance

Screening for RAS. There are no guidelines for routine screening for RAS. In some patients, the diagnosis of RAS is made incidentally during angiographic evaluation of lower extremity arterial diseases, whereas in others a high index of suspicion is required, on the basis of existing guidelines (Table 1). The mere presence of angina, congestive heart failure, coronary artery disease, and peripheral artery disease are not strong indications for evaluation of RAS in the absence of other considerations mentioned in the preceding text. Impromptu "drive-by" renal arteriography during unrelated angiographic procedures is not recommended.

Establish the diagnosis of RAS. If clinical manifestations suggest RAS, the contemporary approach is to use renal duplex ultrasound, magnetic resonance angiography (MRA), or computerized tomography angiography (CTA) to identify RAS. Assessment of the renin-angiotensin system is not recommended (2). Invasive angiography is sometimes recommended to confirm the diagnosis of RAS; determine the etiology; identify dual, accessory, or aberrant renal arteries; identify diseases of the abdominal aorta; and evaluate the nephrogram. The angiographic technique is important to minimize renal injury, prevent atheroembolization, and obtain high-quality images. In most cases, abdominal aortography with digital subtraction provides superb images of the abdominal aorta and renal circulation, with a power injector and 10 to 15 cc of contrast (Fig. 2). Because 30% of patients have dual, accessory, or aberrant renal arteries (Fig. 3), selective angiography alone might preclude complete assessment of the renal arteries. Once "anatomic" RAS is recognized, it is important to establish a relationship between RAS and vital organ injury. The complete evaluation of patients with ARAS and vital organ injury must include a baseline assessment of nephropathy and renal ischemia.

Relationship Between RAS and Renal Dysfunction

The renal artery, the kidney, and renal function. In simplistic terms, the kidney is a filter with inflow (renal arteries), outflow (renal veins), and a reservoir (renal pelvis, ureters, and bladder) (Fig. 4). Apart from diseases of outflow (renal vein obstruction) and collection (obstructive uropathy), filter dysfunction might be due to inflow impairment (RAS and renal ischemia) or filter impairment (nephropathy) or both. When RAS and nonvascular etiologies of renal dysfunction co-exist, it might be difficult to establish RAS as the culprit. Patients with nephropathy might not improve after renal artery revascularization, depending on the extent of baseline nephropathy before revascularization and the degree of renal injury after revascularization (2,7). The relationship between renal ischemia and nephropathy is central to understanding published studies and ongoing trials of RAS, and failure to do so is the most important source of ambiguity about the benefits of renal revascularization.

Clinical evaluation of nephropathy. The clinical evaluation for nephropathy includes serum creatinine, urinalysis, renal duplex ultrasound to assess renal dimensions and renal resistive

Download English Version:

https://daneshyari.com/en/article/2941670

Download Persian Version:

https://daneshyari.com/article/2941670

Daneshyari.com