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Renal Artery Stenting Following Unsuccessful Balloon Angioplasty

As an interventional cardiologist I read with great interest the Rocha-Singh et al. (1) paper regarding renal artery stenosis and revascularization. I think an excellent job was done in delineating the usual indications. However, new ideas have recently surfaced regarding how to determine the significance of a lesion, and more information is now available regarding the physiology of a significant renal artery stenosis.

The radiologist and vascular surgeon, in general, are often not conversant with the adverse effects of a hyperrenin state, congestive heart failure, unstable angina, and progression of atherosclerosis. Unfortunately, many well-known experts, in the face of a hemodynamically significant lesion, have over the years, advocated delaying stenting until creatinine is elevated. However, when creatinine is ≥2 mg/dl, (12.5% of study patients) mortality has been shown to be grossly increased in studies where patients are followed over a four-year period (2,3). Half of renal function may be lost before creatinine is elevated, and there should be an obvious attempt to find these patients before renal function has deteriorated and mortality and morbidity have escalated (4). Renal artery Doppler, renal scan, and creatinine clearance will give subtle clues to early loss of kidney function, and beta-natriuretic peptide may be elevated.

The "unobvious" patients needing revascularization may be the patients with elevated creatinine without hypertension, often in the face of peripheral vascular disease and coronary disease; unilateral kidney shrinkage without significant hypertension; or sudden significant increase in blood pressure. The number of medications should be removed as a parameter for interventions if the patient is treated with clonidine, labetalol, or minoxidil, as moderate doses of these drugs may be equivalent to multiple medications.

Moreover, it needs to be emphasized that the significance of the lesion is often much greater than its angiographic appearance and can be confirmed by flow-wire measurement of pressure gradients and fractional flow reserve (FFR) (5), with these measurements perhaps supplanting 4F catheter gradient measurements. (French size of catheter used for gradient measurement is not discussed in the study.) Then there is the asymptomatic patient with normal creatinine, with a high-grade renal artery stenosis and an extremely high gradient. Obviously the patient with a hyperrenin state will undergo significant progression of diffuse atherosclerosis and progression of arteriolar nephrosclerosis in the contralateral kidney (6). This patient should be revascularized.

These comments should be read in context, as the ASPIRE-2 study was performed 8 to 10 years ago. It is hoped that new studies regarding further research into FFR with varying degrees of renal stenosis, cytokine release in the presence of renal artery stenosis, and the effects of progression of diffuse atherosclerosis, congestive heart failure, and unstable angina will be delineated.

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