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Renal disease

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Renal insufficiency is a well-recognized coronary risk factor. Experimental studies have shown that even a mild impairment in renal function causes a dramatic acceleration in atherosclerosis (1). Coronary artery disease (CAD) is the leading cause of death (50%) in patients with end-stage renal disease (ESRD) and typically occurs within the first year of hemodialysis (2,3).

Conventional risk factors such as diabetes, hypertension, dyslipidemia, and advanced age are prevalent in patients with ESRD but they do not sufficiently account for the high incidence of cardiovascular disease in this patient population. Other unconventional risk factors such as oxidative stress, endothelial dysfunction, and vascular calcification are involved in the process of cardiovascular disease in patients with renal insufficiency.

The progression of CAD is rapid in patients with chronic renal failure, particularly those with ESRD who are undergoing hemodialysis. Consequently, early diagnosis of CAD may be extremely important in determining the prognosis and appropriate therapy in patients with ESRD, as well as in renal transplant patients. Unfortunately, screening for CAD in patients with ESRD is not yet the standard of practice, unless there is clinical or electrocardiographic evidence of CAD.

Although coronary angiography remains the gold standard for the diagnosis of ischemic heart disease, it is invasive, expensive and nephrotoxic and therefore not useful as a screening procedure. The coronary lesions are usually multiple, complex, and calcified, such that computed tomography angiography assessment of CAD is difficult. In addition, it provides anatomic but not functional information. In patients with ESRD, myocardial ischemia may also be caused by microangiopathy, a functional imbalance between vasoconstricting and vasodilating substances due to uremia and inappropriate remodeling of intramyocardial arterioles in left ventricular hypertrophy.

Several non-invasive functional diagnostic tools are available. The high prevalence of abnormal baseline ECG due to left ventricular hypertrophy, repolarisation abnormalities related to electrolyte disturbances, left bundle branch block and use of digitalis in patients with ESRD hampers the diagnosis of cardiac ischemia by ECG. Further, poor exercise tolerance in these patients limits the utility of stress exercise testing. Approximately 80% of ESRD patients fail to reach the target of 85 % of maximal predicted heart rate, due to deconditioning, joint problems, myopathy, polyneuropathy, peripheral vascular disease, chronic anemia, etc.

As a consequence, pharmacological stress testing should be considered as a primary modality of choice in the detection of CAD in this population of patients. Some small-scale studies have suggested that the sensitivity, the specificity and the predictive value of dipyridamole stress testing may be limited in patients with ESRD (4). The underlying pathophysiological substrate is said to be the blunted vasodilatory response to adenosine, due to high resting adenosine levels, and impaired response to endothelium-dependent as well as endothelium-independent vasodilators in this patient population. The use of an inotropic stress such as dobutamine may be more accurate in ESRD patients. Dobutamine stress echocardiography has been used to detect CAD in patients with chronic renal failure and appeared to have a better diagnostic accuracy (5). No data currently exist regarding the utility of dobutamine-myocardial perfusion imaging for the diagnosis of ischemic heart disease in ESRD patients.

In limited studies to date SPECT (5) myocardial perfusion imaging scans are abnormal in 22% to 50% of patients with ESRD and have both diagnostic and prognostic power, even in asymptomatic subjects with ESRD. Large prospective multicenter trials will be needed to change evidence-based guidelines and practice patterns to include screening of patients with ESRD.

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