is more effective than inogatran, a low-molecular weight thrombin inhibitor in suppressing ischemia and recurrent angina in unstable coronary artery disease. Am J Cardiol 1998; 81: 939–44.


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‘The heart doesn’t pump, the kidneys suck’

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The amusing quote that serves as a headline for this editorial was told to me some years ago by a nephrology colleague of mine. Of course, he was trying to be humorous but perhaps there was a bit of truth in what he said. The heart and the kidneys are closely allied in maintaining the body’s circulatory homeostasis, a fact well known to physiologists for many decades. Of course, the circulation of the blood depends upon the heart’s ability to pump, but blood pressure regulation is a major component of normal renal function. Thus, the partnership between the heart and the kidneys is a critical one for normal circulatory function.

In this issue Sørenson et al. report their observations on more than 6000 patients with previous myocardial infarction[1]. The authors were interested in determining whether abnormal renal function affected mortality following a myocardial infarct. Their analysis clearly demonstrated that abnormal renal function increased the likelihood of dying after a myocardial infarction. However, mild renal impairment had no influence on prognosis after statistical adjustment was made for other variables that affect post-myocardial infarction prognosis.

Only patients with creatinine clearances less than or equal to 40 ml . min$^{-1}$ had an increased risk of dying following an myocardial infarction.

Sørensen and co-workers are not the first investigators to observe that abnormal renal function affects long-term prognosis in patients with arteriosclerotic coronary artery disease. Aronow prospectively studied approximately 1400 elderly patients with a variety of chronic illnesses. Most of these elderly patients also had atherosclerotic heart disease. He noted that patients with hypertension and/or diabetes mellitus combined with an increased serum creatinine had a markedly increased likelihood of developing a new coronary event during 3-5 years of follow-up[2]. Similarly, Johannes et al. observed in the HOPE trial (Heart Outcomes and Prevention Evaluation) that patients with pre-existing vascular disease or diabetes mellitus combined with one other coronary risk factor had a significantly increased risk for a subsequent coronary event if they had mild, moderate, or severe renal insufficiency[3]. ACE inhibitor therapy with ramipril reduced the coronary event rate irrespective of whether the patients had renal insufficiency or not. And finally, it has been known for decades that abnormal renal function markedly worsens prognosis in patients hospitalized with acute myocardial infarction[4].

What could be the mechanism by which renal insufficiency worsens prognosis in patients with
atherosclerotic coronary artery disease? The simplest explanation would seem to be that abnormal renal function in these individuals confirms that the patient’s atherosclerotic vascular disease is affecting the renal as well as the coronary circulation. It seems logical, therefore, that patients with coronary artery disease and renal insufficiency have more diffuse and more severe vascular disease when compared with patients who have normal renal function.

Other explanations for the correlation between renal impairment and worsened prognosis in patients with coronary artery disease are also possible. For example, the metabolic status of patients with mild, moderate, or severe renal insufficiency might affect long-term prognosis in these individuals by furthering the atherosclerotic process. There is indeed evidence for the latter contention. Sechi et al. noted that patients with mild renal impairment had elevated serum lipoprotein(a) levels and a greater prevalence of cardiovascular disease. Catena et al. studied 382 patients with primary hypertension. Individuals with mildly decreased creatinine clearance had increased levels of fibrinogen, D-dimer, and prothrombin fragments 1+2, thereby demonstrating the presence of intravascular coagulation in these subjects. Moreover, Kennedy et al. compared 129 patients with chronic renal failure with 82 matched control patients with normal renal function. Patients with renal failure had greater evidence of carotid arterial atherosclerosis as well as abnormal arterial endothelial function. Thus, it would appear that factors favouring the development of atherosclerosis are enhanced in patients with renal insufficiency. Furthermore, atherosclerotic vascular disease is more severe in these patients than in comparable individuals without renal insufficiency.

The ‘take home’ messages for the clinician that derive from the investigation of Sørenson et al. as well as from the work of others in this field are as follows: 1. Any level of renal insufficiency worsens the short- and long-term prognosis for a patient who already has clinical coronary artery disease; 2. Any level of renal insufficiency worsens the short- and long-term prognosis for a patient who already has clinical coronary artery disease; 3. Aggressive medical management of the underlying atherosclerotic process with ACE inhibition, would appear to be a rational therapeutic approach for these patients, particularly since a number of different investigators have documented the beneficial effect of ACE inhibitors on both the progression of renal insufficiency and the likelihood of developing an acute coronary event.

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References