Response to Mildly Decreased Glomerular Filtration Rate Is Associated With Poor Coronary Collateral Circulation in Patients With Coronary Artery Disease

Kadi et al. *Clin Cardiol.* 2011;34:617–621.

To the Editor:

We read with great interest the recently published article by Kadi et al¹ related to coronary collateral circulation in patients with coronary artery disease and mildly decreased renal function. In their study, the authors demonstrated that patients with mildly decreased glomerular filtration rate (GFR) had worse coronary collateral circulation compared to patients with normal GFR. In general, patients with worsening renal function have an increased frequency of comorbidities, more severe left ventricular dysfunction, and worse coronary artery disease. Additionally, in patients with renal dysfunction mortality rates increase up to 60% during the 1st year after myocardial infarction.² In obstructive coronary artery disease, collateral arteries are important compensatory mechanisms for significant coronary arterial

occlusion because they have been shown to have beneficial effects on infarct size, ventricular aneurysm formation, and ventricular function.^{3–6} Although the significance of stenosis and duration of angina were found to be correlated with the collateral formation in patients with coronary artery disease,^{7,8} significant differences were seen in patients with a similar degree of coronary artery disease. In a recent study by Guray et al, a negative correlation was found between the coronary collateral development and inflammation.⁹ On the other hand, Yilmaz et al demonstrated that metabolic syndrome is associated with poor coronary collaterals in patients with occluded right coronary artery.¹⁰

In the Kadi et al study,¹ although coronary collateral circulation was evaluated noninvasively, mildly decreased GFR was found to impair the coronary collateral circulation in patients with coronary artery disease. Impaired collateral development was thought to be associated with the presence of endothelial dysfunction and increased inflammation. Moreover, increased cardiovascular mortality in patients with chronic renal disease was thought to be related to the presence of poor coronary collateral vessel development. On the other hand, in a recent study, an independent relation was found between erythropoietin (EPO) level and collateral grade in patients with coronary artery disease, and it was demonstrated that serum EPO levels were significantly higher in patients with better coronary collateral vessel grade.¹⁰ Recently, it was shown that the increase in blood viscosity and endothelial shear stress due to EPO stimulation triggers the production of endothelial nitric oxide leading to the development of collateral vessels.¹² Although EPO levels were expected to decrease in patients with chronic renal disease, in the Kadi et al study neither the serum levels nor the effect of EPO on coronary collateral circulation was investigated.

In conclusion, although serum EPO levels were not investigated in the Kadi et al study, in addition to the possible endothelial dysfunction and increased inflammation in patients with decreased GFR, coronary collateral circulation may also be affected by the decreased serum EPO levels. We think that further studies are required to clarify this subject.

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