Letter

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Lack of Association between Serum Cystatin C Levels and Coronary Artery Disease in Diabetic Patients (*Korean Diabetes J* 2010;34:95-100)

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Cardiovascular disease, including coronary artery disease (CAD), is the major cause of morbidity and mortality for individuals with diabetes and the largest contributor to the direct and indirect costs of diabetes [1,2]. Patients with diabetes are affected by other risk factors such as dyslipidemia, hypertension, prothrombic and proinflammatory factors, and therefore atherosclerosis in type 2 diabetes mellitus is often accelerated [3]. However, the pathophysiologic mechanisms responsible for the substantially increased risk for CAD in adult type 2 diabetes remain unclear. Inflammation plays a pivotal role in atherosclerosis, and inflammatory markers such as C-reactive protein, platelet-derived growth factor, transforming growth factor-beta, and granulocyte-macrophage colony stimulating factor may help clinicians to identify high risk patients [4,5]. In many previous studies, the association between inflammatory markers and coronary atherosclerosis was weak and mostly explained by the concomitant burden of CAD risk factors.

Cystatin C is a novel endogenous marker of kidney function that may be more sensitive than inflammatory markers for detection of mild to moderate decrements in glomerular filtration rate [6]. However, few data exist on the relationship between serum cystatin C level and cardiovascular disease in diabetic patients with or without diabetic nephropathy. Maahs et al. [7] reported a significant relationship between serum cystatin C level and CAD in type 1 diabetic patients, but they assessed surrogate markers of CAD rather than CAD events or death and more importantly did not consider urinary albumin excretion.

In contrast with previous studies, Kim et al. [8] reported that they found no association between serum cystatin C level and CAD in diabetic patients and that serum cystatin C levels were significantly higher in patients with diabetic nephropathy than in patients without both in CAD patients and non-CAD patients. However, the study used a retrospective case-control design, the sample was relatively small, and they did not evaluate the relationship between cystatin C levels and other markers (inflammatory, structural, and functional), body mass index (BMI) or other variables. Recently, Maahs et al. [9] investigated whether the relationship between cystatin C and progression of CAD differed between individuals with type 1 diabetes and without diabetes. In results, the univariate associations of cystatin C to CAD progression were similar in type 1 diabetic patients and without diabetes mellitus. The association of cystatin C to progression of CAD differed in the expected direction (increased cystatin C as a biomarker of worsening renal function associated with CAD progression) by type 1 diabetes status ($P = 0.01$) after adjustment for other risk factors of cardiovascular disease. Therefore, Maahs et al. [7] suggested, which a complex relationship exists among cystatin C, BMI, and CAD progression, which requires further study.

I appreciate the devotion of study investigators, who are conducting important research about the relationship between

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cystatin C and diabetes, and hope that they will continue to do so in the future.

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