Ventricular Remodeling Does Not Accompany the Development of Heart Failure in Diabetic Patients After Myocardial Infarction

To the Editor:

Scott D. Solomon and colleagues report that the increased incidence of heart failure (HF) after myocardial infarction (MI) in patients with diabetes cannot be explained by an increased propensity for left ventricular enlargement. Although HF develops at twice the rate in diabetic patients than in non-diabetic patients after MI, and although the development of HF is associated with increased ventricular enlargement in both groups, the incidence and extent of left ventricular (LV) enlargement are significantly less in diabetic patients. Increased risk of HF in diabetes without MI is associated with poor glycemic control, neurohormonal activation, and increased oxidative stress.

Studies have identified dyssynchrony between right and left ventricular contraction and relaxation as an independent predictor of HF and cardiac mortality in patients with HF.

The sequelae of ventricular dyssynchrony include loss of coordination of contraction and relaxation, leading to increases in regional and global wall stress, reductions in stroke volume and in the rate of rise of left ventricular pressure, diminished diastolic filling time, prolongation of mitral regurgitation, and diminished effective ejection time.

We evaluated whether abnormalities of echocardiographic parameters of ventricular dyssynchrony were associated with differences in glycemic control in 31 type 2 diabetic patients (16 men and 15 women) aged 59.2 ± 8.7 years (mean ± SD) 97 ± 19 days after uncomplicated first MI. Myocardial performance index, transmitral Doppler flow, and pulmonary venous flow analysis were performed to assess ventricular dyssynchrony. Compared with patients without ventricular dyssynchrony (n = 17), patients with ventricular dyssynchrony (n = 14) had increased HbA1c levels (7.2 ± 1.7% versus 9.8 ± 1.2%, P < 0.01) and fasting plasma glucose (7.7 ± 1.1 versus 8.9 ± 1.3 mmol/L, P < 0.02), whereas body mass index, duration of diabetes, and use of β-blocker therapy were similar in both groups.

Moreover, echocardiographic determination of LV areas, infarct segment length, and ejection fractions showed no difference between the groups. Echocardiographic parameters for ventricular dyssynchrony might be seen as an early indicator of left ventricular dysfunction in diabetic patients after MI, particularly in those with poor glycemic control.

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Response

Marfella et al have suggested an alternative mechanism by which diabetes may modify ventricular remodeling. In a small number of type 2 diabetics, they have shown that worsening measures of diabetes, including glucose intolerance and HbA1c, were associated with ventricular dyssynchrony. These data raise the possibility that ventricular dyssynchrony might further distinguish diabetics from non-diabetics and might influence ventricular remodeling after myocardial infarction. Unfortunately, we have no specific data to support this hypothesis from the Survival And Ventricular Enlargement (SAVE) trial patients presented in our analysis. Specifically, we have not looked at measures of dyssynchrony, either echocardiographic or electrocardiographic, and do not have fasting plasma glucose levels or HbA1c levels for these patients. Nevertheless, the fact that diabetics developed heart failure after myocardial infarction at twice the incidence of non-diabetics while not demonstrating increased ventricular remodeling argues that inherent functional abnormalities, including possibly ventricular dyssynchrony, likely contribute to these differences. Assessment of these and other functional differences between diabetics and non-diabetics is worthy of further investigation.

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