Longitudinal Myocardial Strain Alteration Is Associated with Left Ventricular Remodeling in Asymptomatic Patients with Type 2 Diabetes Mellitus

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Background: In normal subjects, left ventricular (LV) dimensions have been shown to decrease over time, while wall thickness is increasing. The aim of this study was to investigate LV remodeling in a cohort of patients with type 2 diabetes mellitus during a 3-year follow-up period and its potential association with decreased longitudinal systolic strain (ε_L).

Methods: One hundred seventy-two patients with type 2 diabetes without overt heart disease were prospectively enrolled and underwent echocardiography with speckle-tracking imaging to assess global LV ε_L at baseline and at 3 years. The associations between alteration in ε_L (defined as $|\varepsilon_L| < 18\%$), LV geometry at baseline, and LV remodeling over time were evaluated.

Results: Among the 172 enrolled patients, 154 completed 3-year follow-up. At baseline, patients with ε_L alteration had higher LV end-systolic volumes (28 $\pm$ 11 vs 23 $\pm$ 9 mL, $P < .001$) and relative wall thicknesses (RWT; 0.44 $\pm$ 0.06 vs 0.40 $\pm$ 0.07, $P = .008$) compared with those with normal ε_L. At 3-year follow-up, RWTs remained stable in both groups. LV volumes significantly decreased in patients with normal ε_L but not in patients with ε_L alteration. Multivariate analysis showed that ε_L alteration was independently associated with LV end-systolic volume ($\beta = 5.0$, $P = .006$) and RWT ($\beta = 0.03$, $P = .03$) at baseline and with changes in both LV end-diastolic volume ($\beta = 19.1$, $P = .001$) and LV end-systolic volume ($\beta = 2.6$, $P = .047$) over 3 years.

Conclusions: In patients with type 2 diabetes, ε_L alteration was associated with higher RWT and LV volumes and with the absence of decreases in LV volumes over time, which might be an early sign of adverse LV remodeling. (J Am Soc Echocardiogr 2014;27:479-88.)

Keywords: Left ventricular remodeling, Myocardial strain, Diabetes mellitus

Diabetes mellitus is associated with an increased risk for heart failure, even in the absence of coronary artery disease or hypertension, because diabetes itself is responsible for the development of diabetic cardiomyopathy.[1,2] This pathology is responsible for increases in both cardiovascular morbidity and mortality.[1,2]

We and others have shown that patients with diabetes exhibit decreased left ventricular (LV) systolic strain compared with euglycemic subjects.[3-6] It was speculated that such an abnormality could be considered an early marker of diabetic cardiomyopathy. However, the potential impact of these subtle abnormalities on the evolution of LV function and LV geometry remains unknown. Indeed, the association between abnormal systolic strain in patients with diabetes and cardiac remodeling over time has never been investigated.

Recently, large cohort studies have underlined the influence of diabetes on cardiac LV remodeling over the lifetime.[7-9] Although in normal subjects, the aging process is associated with a progressive

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Abbreviations

ACE = Angiotensin-converting enzyme

ARB = Angiotensin receptor blocker

ε_L = Longitudinal systolic strain

HbA_{1c} = Glycosylated hemoglobin

LV = Left ventricular

LVEDV = Left ventricular end-diastolic volume

LVEF = Left ventricular ejection fraction

LVESV = Left ventricular end-systolic volume

RWT = Relative wall thickness

increase in LV wall thickness and a decrease in LV cavity dimensions, the presence of diabetes induces a more pronounced increase in LV wall thickness but the absence of a proportional decrease in cavity dimensions.[7]

We hypothesized that in patients with type 2 diabetes mellitus, alterations in longitudinal systolic deformation are associated with LV remodeling. Therefore, the aim of this study was to investigate LV remodeling in a cohort of patients with type 2 diabetes mellitus during a 3-year follow-up period and its potential association with alteration in longitudinal systolic strain (ε_L).

METHODS

Study Population

Between February 2006 and June 2009, 172 consecutive patients with type 2 diabetes referred to the outpatient Department of Endocrinology at our institution were prospectively included. The inclusion criteria were (1) age between 35 and 75 years, (2) oral antidiabetic or insulin treatment, and (3) LV ejection fraction (LVEF) > 50%. Exclusion criteria were (1) symptoms, signs (clinical or electrocardiographic), or history of heart disease; (2) presence of regional LV wall motion abnormalities; (3) absence of sinus rhythm; (4) history of cardiomyopathy, coronary artery disease, or valvular heart disease; (5) severe renal failure, defined as creatinine clearance < 30 mL/min; (6) echocardiographic images unsuitable for quantification; (7) severely uncontrolled diabetes, defined as glycosylated hemoglobin (HbA_{1c}) > 12% or glycemia > 3 g/L; and (8) uncontrolled blood pressure at rest (defined as blood pressure > 180/100 mm Hg). All patients underwent exercise stress tests, stress echocardiography, or myocardial perfusion scintigraphy within the month before inclusion to exclude silent ischemia.

Among the 172 enrolled patients, seven declined to repeat the echocardiographic examination, two were censored because of cancer treated with chemotherapy, two died (one sudden death and one pancreatic cancer), one had stress cardiomyopathy, and six had nonfatal myocardial infarctions and/or underwent revascularization. The remaining study population consisted of 154 patients.

All subjects provided informed consent to participate, and the study was approved by the ethics committee of our institution.

Study Design

All patients underwent physical examinations, standard echocardiography, and biochemical analysis on the same day at baseline and after 3 years.

Echocardiography

Resting transthoracic echocardiography was performed in the left lateral decubitus position using a commercially available ultrasound system (Vivid 7 or 9; GE Medical Systems, Oslo, Norway). All acquisitions were digitally stored in raw-data format from at least three consecutive heartbeats for offline analysis (EchoPAC; GE Vingmed Ultrasound AS, Horten, Norway), which was performed by two experienced observers blinded to the other data (L.E., C.B.).

LV wall thickness was measured from M-mode images from the parasternal long-axis view according to the recommended criteria.[10] Total LV wall thickness was calculated as the sum of septal wall thickness and posterior wall thickness.[7] LV mass was determined as recommended[10] using Devereux's formula[11] and indexed to body surface area. Relative wall thickness (RWT) was calculated as (2 × posterior wall thickness at end-diastole)/LV end-diastolic diameter.[10]

LV end-diastolic volume (LVEDV) and LV end-systolic volume (LVESV) and LVEF were calculated from the apical four-chamber and two-chamber views using the modified biplane Simpson's method.[10]

Using pulsed-wave Doppler, mitral inflow velocities, peak early (E) and late (A) diastolic velocities, the E/A ratio, and E-wave deceleration time were measured. The annular early diastolic velocity (e′) was assessed at the lateral and septal sites of the mitral annulus using pulsed-wave Doppler tissue imaging. The average e′ value (from the lateral and septal sites) was used to calculate the E/e′ ratio. Left atrial area was measured in an apical four-chamber view using planimetry, and left atrial volume was assessed as previously described.[12]

Strain Analysis by Speckle-Tracking Imaging

Speckle-tracking analysis was performed using a dedicated software package (EchoPAC).[4] Peak ε_L was measured in the apical four-chamber and two-chamber views (frame rate, 70–80 frames/sec) The endocardial border was manually traced from an end-systolic frame. The software automatically detected the epicardial border, and the region of interest was manually adjusted to include the entire myocardial wall. Thus, the software tracked the contour throughout the entire cardiac cycle frame by frame. The quality of tracking was verified both automatically and visually, and the region of interest was modified and corrected by the observer if judged necessary to obtain optimal tracking. The software automatically divided the LV walls into six segments for each view and calculated the segmental strain values. Each segmental peak ε_L value was collected, and the average of longitudinal segmental strain values was calculated for each patient and presented as ε_L.

Alteration in ε_L was defined as $|\varepsilon_L| < 18\%$ and normal ε_L as $|\varepsilon_L| \geq 18\%$ (mean value − 2 standard deviations in normal subjects), as previously published by our group,[4] in accordance with others[13] and as reported in the American Society of Echocardiography and European Association of Echocardiography consensus statement on techniques for the quantitative evaluation of cardiac mechanics (Figure 1).[14]

Reproducibility

To define reproducibility, 15 patients were randomly selected in the population study. In these patients, LV dimensional measurements by M-mode echocardiography, LV volumes, and ε_L analysis were repeated 3 months apart by the same observer and performed by a second observer. The first observer (L.E.) was blinded to previous measurements during the second analysis, and the second observer (C.B.) was blinded to measurements of the first observer. A minimum of six cardiac cycles were available for each measurement (three cardiac cycles per loop and at least two loops for each view), and the reader was allowed to select the best cardiac cycle each time and to repeat and average the measurement if judged necessary.

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