Left Ventricle Longitudinal Deformation Assessment by Mitral Annulus Displacement or Global Longitudinal Strain in Chronic Ischemic Heart Disease: Are They Interchangeable?

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Background: Increasing infarct mass is associated with impaired prognosis in chronic ischemic heart disease. Global strain by echocardiographic assessment relates closely to infarct mass assessed by delayed enhancement magnetic resonance imaging but requires deformation analysis in a 16-segment model of the left ventricular. Mitral annular (MA) displacement reflects longitudinal left ventricular deformation and could provide similar information.

Methods: Global longitudinal strain and MA displacement by Doppler tissue imaging were assessed in 61 patients 9 months after first myocardial infarctions and compared with global myocardial infarct mass assessed using contrast-enhanced magnetic resonance imaging.

Results: Both indices significantly separated medium-sized infarcts from small or large infarcts ($P < .05$) and correlated significantly with global infarct mass ($P < .01$ for both). There was a good correlation between global strain and MA displacement ($r = 0.65$, $P < .01$). The sensitivities and specificities to identify myocardial infarcts differed only slightly among the indices, but global longitudinal strain tended to be the best.

Conclusions: Longitudinal deformation by global strain or MA displacement correlated well with myocardial infarct mass and could discriminate between different extents of myocardial infarctions. Global longitudinal strain tended to be better, especially for the identification of the smallest infarcts. (J Am Soc Echocardiogr 2009;22:823-830.)

Keywords: Contrast-enhanced MRI, Strain, Mitral annulus, Displacement, Ischemic heart disease

The quantification of myocardial infarcts provides important diagnostic and prognostic information in patients with acute myocardial infarctions (MIs) and chronic ischemic left ventricular (LV) dysfunction, because mortality closely relates to infarct size and location.[1-4] Myocardial infarct size assessed by contrast-enhanced magnetic resonance imaging (CE-MRI) has been validated in animal models[1] and predicts cardiovascular events in patients,[2] but it is time consuming and expensive. Risk stratification therefore requires reliable and feasible clinical tools to measure the exact extent and location of myocardial necrosis.

Systolic LV long-axis deformation decreases with increasing myocardial scar load,[5-10] and the assessment of circumferential or radial deformation adds no additional information on global infarct mass.[9] The position of the LV apex is relatively stationary throughout the cardiac cycle, and mitral annular (MA) displacement in the longitudinal direction therefore reflects the global shortening deformation of the left ventricle.[11] MA displacement has been shown to correlate with LV ejection fraction (LVEF)[11-13] and to predict cardiovascular outcomes[14-16] and is widely used in the clinical setting. Analysis of MA displacement could therefore represent an alternative and easily available approach for the assessment of myocardial infarct mass in chronic ischemic heart disease.

Myocardial strain is a clinical index of myocardial deformation[17,18] and has been quantified and validated using tagged MRI, Doppler tissue imaging (DTI), and two-dimensional speckle-tracking echocardiography (2D-STE).[19-21] It has been found to correlate well with myocardial infarct in both the acute and chronic settings.[7-10] Global longitudinal strain reflects the averaged segmental myocardial long-axis relative shortening and has been introduced as a reliable and reproducible deformation index that correlates well with myocardial infarct mass assessed by CE-MRI.[7-9]

Longitudinal strain by 2D-STE is assessed along the LV curvature and reflects myocardial deformation regardless of changes in the curvature of the infarcted LV walls. The assessment of MA displacement,

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on the other hand, requires visualization of the basal LV parts only. Therefore, this method might potentially be more robust in the presence of reverberations or when other parts of the LV wall are poorly visualized.

The aim of this study was to compare myocardial infarct mass with MA displacement on tissue Doppler echocardiography and with global longitudinal strain on 2D-STE, to determine whether the two indices of longitudinal LV function are interchangeable. The exact distribution of myocardial infarct mass was assessed using CE-MRI in patients with chronic ischemic heart disease. The study also established reference values for MA displacement in chronic ischemic heart disease.

METHODS

Patients

Sixty-one patients (mean age 57 ± 10 years; 13 women) previously treated with primary percutaneous coronary intervention for acute ST elevation MIs were included in the study. Patients with contraindications to MRI were excluded, but no patients were excluded because of impaired echocardiographic image quality. The clinical data and infarct characteristics are displayed in Tables 1 and 2. Patients were examined with CE-MRI and echocardiography 9 months after the index MI, typically the same day. There were no reports of cardiovascular events after angiography at the index MI, and patients were hemodynamically stable at the time of the imaging studies. All study subjects were in sinus rhythm and had QRS widths <120 ms. None had significant valvular dysfunction as defined by echocardiography. The study was approved by the Regional Committee for Medical Research Ethics (REK Sør, Oslo, Norway), and all subjects gave written informed consent.

MRI

MRI was performed using 1.5-T units (Magnetom Vision Plus or Magnetom Sonata, Siemens Medical Systems, Erlangen, Germany) and a phased-array body coil. Corresponding cine and late enhancement images in two long-axis and multiple short-axis views to cover the left ventricle (average, 8.7; range, 6-12) were acquired in the same session (time resolution < 50 ms). Late-enhancement images were obtained 10 to 20 minutes after the intravenous injection of 0.1 or 0.2 mmol/kg gadopentetate dimeglumine (Magnevist; Schering AG, Berlin, Germany), with a slice thickness of 7 or 8 mm, an increment of 10 mm, and in-plane spatial resolution of 1.17 × 1.17 to 1.50 mm. A breath-hold segmented magnetization-prepared turbo gradient-echo sequence was used, with an inversion time chosen to null the signal of the normal myocardium, typically 210 to 260 ms.

Short-axis slices were manually assigned to the basal, mid, or apical LV region and divided into sectors to fit a 16-segment model.[22] Epicardial and endocardial borders and the infarcted area were manually delineated (PACS; Sectra Imtec AB, Linköping, Sweden),[23] and cross-correlation between short-axis and long-axis images was performed. When in doubt, areas were considered infarcted if the pixel intensity was ≥ 2 standard deviations above the mean pixel intensity of adjacent normal myocardium.[1,24,25] Myocardial and infarct masses were converted from volume by multiplying by 1.05 g/mL,[26] and the total myocardial volume and absolute and relative infarct volumes were calculated. Patients were divided into groups depending on global infarct mass: small infarcts of < 30 g, medium-sized infarcts of 30 to 50 g, and large infarcts of ≥ 50 g (Figure 1).[2] Segments were classified as transmural when ≥50% segmental tissue was infarcted, and infarcts were considered transmural when ≥1 segment was classified as transmural.[27]

Table 1 Patient characteristics (n = 61)

Variable	Value
Age (y)	57 ± 10
Men/women	48/13
Heart rate (beats/min)	57 ± 10
Systolic blood pressure (mm Hg)	127 ± 22
Diastolic blood pressure (mm Hg)	79 ± 13
Medications	
β-blockers	61 (100%)
ACE inhibitors or ARBs	49 (80%)
LV mass indexed for BSA (g/m^2)	84 ± 20
TIMI flow grade before PTCA	0.9 ± 1.2
TIMI flow grade after PTCA	2.9 ± 0.3
Stents implanted	1.5 ± 0.9
LAD stenosis/culprit lesion	56/54
LCx stenosis/culprit lesion	15/1
RCA stenosis/culprit lesion	15/6
Q wave on ECG	21

ACE, Angiotensin-converting enzyme; *ARB*, angiotensin receptor blocker; *BSA*, body surface area; *ECG*, electrocardiography; *LAD*, left anterior descending coronary artery; *LCx*, left circumflex coronary artery; *PTCA*, percutaneous transluminal coronary angioplasty; *RCA*, right coronary artery.
Data are expressed as mean ± SD or as number. Stenosis was considered significant for ≥50% luminal diameter obstruction.

Table 2 Absolute and relative numbers of patients with any infarcts and transmural infarcts in each of the 16 LV segments and median (range) transmural distribution among the infarcted segments

Segment	Infarcted segment	Transmural infarct	Transmural distribution
Basal anterior	20 (33%)	4 (7%)	12% (2%-83%)
Basal anteroseptal	23 (38%)	4 (7%)	27% (3%-89%)
Basal inferoseptal	14 (23%)	1 (2%)	18% (1%-66%)
Basal inferior	10 (16%)	4 (7%)	44% (15%-100%)
Basal inferolateral	9 (15%)	2 (3%)	31% (2%-83%)
Basal anterolateral	8 (13%)	1 (2%)	17% (3%-65%)
Mid anterior	47 (77%)	18 (30%)	40% (3%-100%)
Mid anteroseptal	46 (75%)	21 (34%)	44% (3%-100%)
Mid inferoseptal	40 (66%)	4 (7%)	19% (1%-100%)
Mid inferior	23 (38%)	3 (5%)	12% (4%-100%)
Mid inferolateral	14 (23%)	2 (3%)	10% (4%-74%)
Mid anterolateral	29 (48%)	3 (5%)	21% (2%-100%)
Apical anterior	49 (80%)	30 (49%)	58% (3%-100%)
Apical septal	50 (82%)	30 (49%)	55% (4%-100%)
Apical inferior	47 (77%)	7 (11%)	28% (2%-100%)
Apical lateral	35 (57%)	10 (16%)	28% (3%-100%)

Echocardiography

Patients were examined in the left lateral decubitus position. The study examinations were performed with Vivid 5 or 7 scanners (GE Vingmed Ultrasound AS, Horten, Norway), using phased-array transducers. Three consecutive heart cycles from the 3 standard apical views (4 chamber, 2 chamber, and long axis) were obtained by conventional 2-dimensional grayscale echocardiography and DTI,