

Influence of Microvascular Obstruction on Regional Myocardial Deformation in the Acute Phase of Myocardial Infarction: A Speckle-Tracking Echocardiography Study

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Background: In the acute phase of myocardial infarction (MI), infarct size and microvascular obstruction (MVO) are important prognostic factors for cardiovascular outcome. MI size is a major determinant of myocardial function, but the specific effect of MVO is less documented. The aim of this study was to evaluate the impact of MVO on longitudinal myocardial strain assessed by speckle-tracking echocardiography.

Method: Speckle-tracking echocardiography and contrast-enhanced cardiac magnetic resonance studies were performed in 69 patients 72 hours after first acute MI. Segmental and global longitudinal systolic strain (ϵ_L) was measured using speckle-tracking echocardiography. Transmural extent of MI, MI size, and the presence or absence of MVO were assessed using contrast-enhanced cardiac magnetic resonance. Left ventricular (LV) ejection fraction was assessed at 6 months using echocardiography.

Results: The mean infarct size was $23 \pm 13\%$ of LV mass. MVO was present in 64% of patients. MVO was significantly associated with ϵ_L impairment ($-7.8 \pm 4.9\%$ vs $-16.3 \pm 6.4\%$, $P < .001$), and ϵ_L remained significantly worse in MVO-positive segments after adjustment for transmural extent of MI. A ϵ_L value $> -12.5\%$ predicted the presence of MVO with 83% sensitivity and 75% specificity. On multivariate analysis, global ϵ_L and MI size, but not MVO, were identified as independent predictors of LV ejection fraction at follow-up ($\beta = -0.9$, $P = .023$, and $\beta = -0.2$, $P = .034$, respectively).

Conclusion: In the acute phase of MI, segmental and global ϵ_L is significantly altered by the presence of MVO, in addition to MI size. However, MI size but not MVO independently predicts LV ejection fraction at follow-up. (J Am Soc Echocardiogr 2014;27:93-100.)

Keywords: Myocardial infarction, Speckle-tracking echocardiography, Microvascular obstruction, Contrast-enhanced magnetic resonance imaging

After reperfusion, the final infarct size and the presence of microvascular obstruction (MVO) are two major determinants of left ventricular (LV) remodeling and prognosis in patients with myocardial infarction (MI).¹⁻⁴

MVO is a complex and dynamic phenomenon that leads to impaired tissue perfusion at the microvascular level despite adequate restoration of epicardial vessel patency. Its presence is additional evidence of the severity of the myocardial injury caused by prolonged myocardial ischemia together with reperfusion injury.⁵ Also, MVO appears as an independent predictor of contractile recovery and adverse clinical events.⁵⁻⁹ There is a significant relationship between MI size and the presence of MVO, but the effect of their interaction on regional and global contractile function and recovery is not clearly defined.¹⁰

Recent experimental and clinical studies have shown that myocardial deformation determined using speckle-tracking echocardiography (STE) in the acute phase of MI is closely related to MI size as quantified by contrast-enhanced cardiac magnetic resonance (CMR).¹¹⁻¹⁴ Moreover, it has been shown that myocardial longitudinal systolic strain (ϵ_L) predicts LV remodeling and has additional prognostic value in the acute phase of MI.^{3,15} However, none of these studies has assessed the impact of MVO in addition to MI size on global and regional ϵ_L in the acute phase of MI.

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Abbreviations

CI = Confidence interval
CMR = Cardiac magnetic resonance
ϵ_L = Longitudinal systolic strain
LV = Left ventricular
LVEF = Left ventricular ejection fraction
MI = Myocardial infarction
MVO = Microvascular obstruction
STE = Speckle-tracking echocardiography
STEMI = ST-segment elevation myocardial infarction
TME = Transmural extent

The main objectives of our study were to assess the impact of MVO (1) on segmental myocardial strain in addition to MI transmural extent (TME) and (2) on global myocardial strain in addition to MI size in the acute phase of MI. We also sought to determine factors associated with myocardial systolic recovery as assessed by LV ejection fraction (LVEF) at 6-month follow-up.

METHODS**Study Population**

From December 2006 to February 2008, 69 consecutive patients were prospectively included with first acute ST-segment elevation MIs (STEMIs)

according to standard electrocardiographic and enzymatic criteria.¹⁶ Patients presented within 24 hours of symptom onset and were referred for primary percutaneous coronary intervention or thrombolysis followed by percutaneous coronary intervention with stent implantation.

Exclusion criteria were prior MI, underlying cardiomyopathy, hemodynamic instability, atrial fibrillation, significant valvular heart disease, and contraindications to contrast-enhanced CMR. All patients received medical treatment according to guidelines.

The local ethics committee approved the study protocol. Written informed consent was obtained from all patients.

CMR Imaging

CMR imaging studies were performed <72 hours after admission using a 1.5-T whole-body scanner (Magnetom Avanto; Siemens Medical Solutions, Erlangen, Germany).

LV function at rest was assessed using retrospective electrocardiographically gated steady-state free-precession pulse cine sequences in long-axis and short-axis views (repetition time, 3.2 msec; echo time, 1.6 msec; slice thickness, 6.5 mm; temporal resolution, 35–50 ms; matrix size, 256 × 184). The short-axis scans covered the whole left ventricle with contiguous slices.

Contrast enhancement was assessed 10 min after an intravenous dose of 0.2 mmol/kg gadolinium-DOTA (Dotarem; Guerbet, Roissy, France) using a three-dimensional gradient spoiled turbo fast low-angle shot sequence with a selective 180° inversion recovery prepulse, in the short axis, covering the whole ventricle (repetition time, 1.6 msec; echo time, 1.4 ms; inversion time individually determined to null the myocardial signal, range 180–250 msec; slice thickness, 5 mm; matrix size, 256 × 192). Two or three additional long-axis views with a similar two-dimensional sequence were performed.

Contrast-Enhanced CMR Imaging Analysis

LVEF, LV end-diastolic volume, LV end-systolic volume, and absolute myocardial mass were calculated for each study using Argus postprocessing software (Siemens Healthcare, Erlangen, Germany) using short-axis volumetry.

MI was identified by contrast enhancement within the myocardium, defined quantitatively by a myocardial postcontrast signal intensity > 2 standard deviations above that within a reference region placed in the remote noninfarcted myocardium in the same slice. Infarct size was quantified on the three-dimensional data sets by manual planimetry of the hyperenhanced myocardium using the postprocessing imaging software Osirix (Osirix Foundation, Geneva, Switzerland). For all slices, absolute infarct size in grams was measured according to the following formula: infarct size (g) = $\Sigma(\text{hyperenhanced area [cm}^2]) \times \text{slice thickness (cm)} \times \text{myocardial specific density (1.05 g/cm}^3)$.¹⁷

For segmental infarct extension analysis, the myocardium was divided into 16 segments.¹⁸ The TME of each segment was assessed. Subendocardial infarct was defined as TME ≤ 50% of the segmental myocardial area, whereas transmural infarct was defined when TME > 50%.

The presence of MVO was defined in each segment by the presence of hypoenhancement within the hyperenhanced area on the contrast-enhanced studies. MVO size was also measured by manual planimetry of the hypoenhanced myocardium according to the same method used for the infarct size measurement. Two typical examples from our CMR study data set demonstrating a patient with MVO and another without are presented in Figure 1.

Echocardiography

Examinations were performed within 72 hours after reperfusion using a commercially available system (Vivid 7; GE Vingmed Ultrasound AS, Horten, Norway). Three cardiac cycles were stored for each view of the left ventricle: parasternal short-axis view at the papillary muscle level and apical four-chamber, two-chamber, and long-axis views. Particular care was taken to obtain optimal quality recordings of all LV walls. Two-dimensional grayscale images were obtained at frame rates of 76 to 90 frames/sec and stored for further strain analysis.

Echocardiographic Image Analysis

Speckle-tracking analysis was performed offline using dedicated software (EchoPAC; GE Vingmed Ultrasound AS). A 16-segment LV model was obtained from the four-chamber, two-chamber and long-axis recordings for quantitative analysis. Longitudinal systolic strain was measured from apical views. Strain was measured in the entire segment as obtained by the automatic EchoPAC segmentation during one cardiac cycle. Selection of the cardiac cycle was left to the reader's decision (best quality of the tracking process). Longitudinal systolic strain was defined as the maximal peak value during systole or at the time of aortic valve closure (end-systole) if the peak value occurred at aortic valve closure (Figure 2). The tracking process was automated from the end-systolic frame and corrected manually if necessary to obtain an optimal strain curve result. In case of poor acoustic signal, and loss of reproducibility (>10% between two consecutive measurements), segments were excluded from analysis. Peak systolic strain measurements were averaged from apical views to obtain global ϵ_L .

Follow-Up

All patients were scheduled for follow-up at 6 months to assess their clinical status as well as their LVEFs using transthoracic echocardiography within our institution or outside our institution by referring cardiologists.

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