Usefulness of Two-Dimensional Longitudinal Strain Pattern to Predict Left Ventricular Recovery and In-Hospital Complications after Acute Anterior Myocardial Infarction Treated Successfully by Primary Angioplasty

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Background: The aim of this study was to test the usefulness of two-dimensional longitudinal strain pattern in segments with wall motion abnormalities to predict left ventricular recovery and in-hospital cardiac events as well as coronary microvascular impairment (CMI) in patients with recent acute anterior myocardial infarction.

Methods: Forty-nine consecutive patients with acute myocardial infarction (mean age, 59 ± 13 years) treated successfully with primary coronary angioplasty prospectively underwent transthoracic Doppler echocardiography 24 hours after angioplasty and during follow-up (6 months). A two-dimensional strain analysis, including measurement of the duration of systolic lengthening expressed as a percentage of systolic duration (SL % duration), the lengthening-to-shortening ratio, the postsystolic shortening index in segments with wall motion abnormalities, and global longitudinal strain and left anterior descending coronary artery territory strain, was performed. Cardiac events were defined as a composite of death, reinfarction, and heart failure. CMI was assessed noninvasively by transthoracic Doppler left anterior descending coronary artery investigation <24 h after angioplasty and was defined as coronary flow velocity reserve < 1.7 and/or a no-reflow pattern (mean coronary flow velocity reserve, 1.8 ± 0.6 in the whole group).

Results: At the segmental level, SL % duration, lengthening-to-shortening ratio, and postsystolic shortening index were correlated with recovery (defined as normalization of wall motion abnormalities), whereas in multivariate analysis, only SL % duration independently predicted recovery (threshold level, 40%; area under the curve, 0.76; $P < .01$). At the patient level, in univariate analysis, SL % duration, global longitudinal strain, left anterior descending coronary artery territory strain, and troponin peak were correlated with recovery (defined as an absolute improvement of left ventricular ejection fraction of >5%). In multivariate analysis, SL % duration was independently related to recovery (area under the curve, 0.78; $P < .01$). Furthermore, SL % duration was independently linked to cardiac events ($n = 13$) and CMI ($n = 24$) ($P < .01$ for all).

Conclusions: In patients with AMI treated by primary angioplasty, two-dimensional strain predicts left ventricular recovery independently of more traditional parameters and is independently linked to cardiac events and CMI. (J Am Soc Echocardiogr 2015;■:■-■.)

Keywords: Longitudinal strain, Systolic lengthening, Myocardial infarction, Heart failure, Coronary flow reserve

Some patients do not recover from left ventricular (LV) systolic dysfunction after acute myocardial infarction (AMI) despite being treated successfully with primary angioplasty and receiving optimal medical therapy according to current guidelines.[1] Indeed, the presence and extent of postischemic viable myocardium is an important determinant of functional recovery after AMI, and its quantification has prognostic value in this setting.[2-6] This is particularly true in anterior AMI, in which the area exposed to ischemia is large. Several tools can assess directly or indirectly the stunned myocardium after AMI, including dobutamine stress echocardiography, nuclear cardiac imaging, and magnetic resonance imaging (MRI).[3,4,7] However, nuclear cardiac imaging and MRI are not easily available, are expensive, and involve radiation and gadolinium exposure. Dobutamine stress echocardiography is a subjective test with currently very few indications in the acute phase of AMI. Myocardial contrast echocardiography is also a useful technique providing regional and transmural information, which is a critical determinant of prognosis after AMI.[8] However, this technique requires advanced training and expertise and an intravenous infusion of a microbubble contrast agent. Myocardial deformation imaging by two-dimensional strain (2DS) allows the evaluation of regional and global LV systolic

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Abbreviations

AMI = Acute myocardial infarction

AUC = Area under the curve

CFVR = Coronary flow velocity reserve

CMI = Coronary microvascular impairment

GLS = Global longitudinal strain

LAD = Left anterior descending coronary artery

LV = Left ventricular

LVEF = Left ventricular ejection fraction

MRI = Magnetic resonance imaging

OR = Odds ratio

SL = Systolic lengthening

SL % duration = Systolic lengthening as a percentage of systolic duration

2DS = Two-dimensional strain

WMA = Wall motion abnormality

WMSI = Wall motion score index

function at rest with high reproducibility and a shallow learning curve, without the limits of Doppler tissue imaging due to its angle dependency.[9-21] This is of particular importance when assessing viability in anterior AMI, in which the apex is always involved. Some studies have used the quantitative method of 2DS to assess myocardial recovery after AMI, using a global or regional approach.[11-13,16,22] However, with regard to the qualitative evaluation of 2DS and when focusing on the segmental strain pattern aiming to predict recovery and prognosis, no available data currently exist. One study using Doppler tissue imaging before reperfusion showed a positive correlation between longitudinal systolic lengthening (SL) and transmural extent of necrosis in anterior AMI,[19] but such an evaluation has never been performed by 2DS. In a preliminary study involving 21 patients with AMI compared with 21 patients with typical takotsubo syndrome, we found an independent relationship between SL and segmental recovery.[21] The strain pattern analysis by 2DS may offer a new diagnostic capability in patients with AMI, particularly by studying in impaired segments the presence of SL, which identifies a passive myocardium. However, whether this specific analysis could contribute to the important topic of myocardial viability and prognosis in patients with AMI is unclear. Therefore, our objective was to estimate the diagnostic value of the longitudinal strain pattern in segments with wall motion abnormalities (WMAs) to predict recovery after anterior AMI and test its link with in-hospital events, as well as with coronary microvascular impairment (CMI), as already shown in terms of diagnostic and prognostic value in this setting.[23-25]

METHODS

Population Studied

Forty-nine consecutive patients with anterior AMI who underwent successful primary angioplasty at our institution <12 hours after symptom onset and comprehensive transthoracic Doppler echocardiography <24 hours of admission and at follow-up, including 2DS analysis, were prospectively included in the study. All patients were in normal sinus rhythm. Significant valvular disease (at least moderate regurgitation or stenosis), left bundle branch block, and poor echogenicity were exclusion criteria. The diagnosis of AMI was based on chest pain lasting >30 min, ST-segment elevation > 2 mm in at least two contiguous precordial electrocardiographic leads, and increase in serum troponin T.[1] Diagnostic coronary angiography using the radial or femoral approach and coronary angioplasty were performed by standard techniques. During the procedure, glycoprotein IIb/IIIa inhibitors, thrombectomy (Export; Medtronic, Inc, Minneapolis, MN), and intracoronary vasodilators (adenosine and verapamil) were used at the discretion of the interventional cardiologist. Successful angioplasty, required for inclusion in the study, was defined as a final angiographic Thrombolysis In Myocardial Infarction flow grade of 3 with a residual stenosis <30% in the left anterior descending coronary artery (LAD). All patients received aspirin (250–500 mg), a $P2Y_{12}$ inhibitor (a loading dose of clopidogrel, prasugrel, or ticagrelor) and intravenous bolus of heparin (5,000 U) before angiography. After the procedure, all patients received medical therapy according to current guidelines for ST-segment elevation myocardial infarction.[1] All patients gave informed consent for the protocol.

Conventional Echocardiography

Comprehensive transthoracic Doppler echocardiography was performed <24 hours after angiography and at 6-month follow-up, and all echocardiograms were digitized online and stored on a workstation (EchoPAC 7 version 108 for PC; GE Medical Systems, Waukesha, WI) for subsequent offline analysis by two observers blinded to patient data. LV end-diastolic volume and end-systolic volume were measured from the apical four- and two-chamber view and LV ejection fraction (LVEF) was calculated using the modified biplane Simpson rule. Left atrial volume index was measured according to the area-length method and LV mass according to the American Society of Echocardiography formula.[26] Wall motion score index (WMSI) was measured using the 16-segment four-point scaling model from the apical four-, two-, and three-chamber views. WMSI was derived as the sum of all scores divided by the number of segments visualized. Segment scores were as follows: 1 = normal, 2 = hypokinesia, 3 = akinesia, and 4 = dyskinesia. Conventional Doppler parameters were also measured according to a standardized examination: early (E) and late (A), diastolic transmitral flow velocity, and deceleration time of E, average of the septal and lateral annular mitral early diastolic (e′), late diastolic (a′), and systolic (Sa) pulsed-wave tissue Doppler velocity, and the E/e′ ratio. Pulmonary artery systolic pressure was calculated using the modified Bernoulli equation from tricuspid regurgitant peak jet velocity and estimated right atrial pressure (from respiratory variation of inferior vena).

Myocardial Deformation Imaging by 2DS

From the apical long-axis and four- and two-chamber views, LV global longitudinal strain (GLS) by 2DS was quantified as previously described.[14-16] Briefly, the left ventricle was divided into six segments in each apical view, and tracking quality was validated for each segment. Careful attention was paid to cover all the thickness of the myocardium for tracking. Then myocardial motion was analyzed by speckle-tracking within the region of interest. The automated algorithm provided peak systolic longitudinal strain for each LV segment (total, 18 segments). LAD strain was also measured and was defined as the mean of peak systolic longitudinal strain values from the 11 segments assigned to the LAD territory (see Figure 1).The corresponding strain curves were stored for segmental strain analysis in segments with WMAs, focusing, in addition to peak systolic segmental strain, on the strain pattern, measuring the duration and amplitude of longitudinal SL, as well as the occurrence of postsystolic shortening. The duration of SL (when present) was measured, as previously described,