Strain Echocardiography and Wall Motion Score Index Predicts Final Infarct Size in Patients With Non–ST-Segment–Elevation Myocardial Infarction

Christian Eek, MD; Bjørnar Grenne, MD; Harald Brunvand, MD, PhD; Svend Aakhus, MD, PhD; Knut Endresen, MD, PhD; Per K. Hol, MD, PhD; Hans-Jørgen Smith, MD, PhD; Otto A. Smiseth, MD, PhD; Thor Edvardsen, MD, PhD; Helge Skulstad, MD, PhD

Background—Infarct size is a strong predictor of mortality and major adverse cardiovascular events after myocardial infarction. Acute reperfusion therapy limits infarct size and improves survival, but its use has been confined to patients with ST-segment–elevation myocardial infarction. The purpose of this study was to assess the relationship between echocardiographic parameters of left ventricular (LV) systolic function obtained before revascularization and final infarct size in patients with non–ST-segment–elevation myocardial infarction, as well as the ability of these parameters to identify patients with substantial infarction.

Methods and Results—Sixty-one patients with non–ST-segment–elevation myocardial infarction were examined by echocardiography immediately before revascularization, 2.1±0.6 days after hospitalization. LV systolic function was assessed by ejection fraction, wall motion score index, and circumferential, longitudinal, and radial strain in a 16-segment LV model. Global strain represents average segmental strain values. Infarct size was assessed after 9±3 months by late-enhancement MRI, as a percentage of total LV myocardial volume. A good correlation was found between infarct size and wall motion score index (r=0.74, P<0.001) and global longitudinal strain (r=0.68, P<0.001). Global longitudinal strain >13.8% and wall motion score index >1.30 accurately identified patients with substantial infarction (≥12% of myocardium, n=13; area under the receiver operator curve, 0.95 and 0.92, respectively).

Conclusions—Echocardiographic parameters of LV systolic function correlate to infarct size in patients with non–ST-segment–elevation myocardial infarction. Global longitudinal strain and wall motion score index are both excellent parameters to identify patients with substantial myocardial infarction, who may benefit from urgent reperfusion therapy. (Circ Cardiovasc Imaging. 2010;3:187-194.)

Key Words: myocardial infarction ■ echocardiography ■ MRI ■ myocardial contraction

Infarct size is a strong predictor of mortality and major adverse cardiovascular events after myocardial infarction (MI).1 Reperfusion therapy by thrombolysis or percutaneous coronary intervention (PCI) salvages viable myocardium and preserves left ventricular (LV) function by reduction of infarct size.2 This strategy has improved clinical outcome preserves left ventricular (LV) function by reduction of coronary intervention (PCI) salvages viable myocardium and CE-MRI) is considered the gold standard for assessment of myocardial infarction. Acute coronary occlusion is followed by rapid changes in LV systolic function that can be quantified by echocardiography.7 In STEMI patients, echocardiographic parameters of LV systolic function are demonstrated to correlate to infarct size, both in the acute and chronic phases.8,9 Strain echocardiography is an accurate and validated measure of regional systolic LV function10,11 that has demonstrated excellent ability to differentiate between different levels of infarct size.8 Recently, strain echocardiography has been validated as a prognostic indicator.12,13 Contrast-enhanced MRI (CE-MRI) is considered the gold standard for assessment of final infarct size.14 We assessed the ability of strain echocardiography and established indices of LV systolic function to predict final infarct size and specifically the ability to stratify...
patients with regard to substantial as opposed to minor myocardial necrosis.

Methods

This study was conducted in a single tertiary coronary care center. Consecutive patients with documented NSTEMI on whom coronary angiography was planned within 3 days of index admission were prospectively enrolled from May 2007 to June 2008. All patients were enrolled and examined immediately on arrival at the invasive center.

Patients

The study population comprises 61 patients with a diagnosis of NSTEMI confirmed at the referring hospital by elevated troponin I or troponin T above the 99% percentile. Blood samples acquired immediately before coronary angiography were available in 56 patients (92%), and all subsequent reports of troponin T refer to this sample. Time from hospital admission to coronary angiography was 2.1±0.6 days (range, 1 to 3 days). Exclusion criteria were prior MI; evidence of STEMI (ST-elevation >0.1 mV (0.2 mV in precordial leads V1–V3) in 2 or more contiguous leads on any ECG during index admission; bundle-branch block with QRS >120 ms; severe valvular disease; previous heart surgery; atrial fibrillation with heart rate >100; and any condition interfering with the patient’s ability to comply and contraindications to MRI. All patients were considered clinically and hemodynamically stable during index admission, and none were referred for urgent coronary intervention. The study was approved by the regional ethics committee, and all patients gave written informed consent.

Echocardiography

Echocardiographic examinations were performed with a Vivid 7 scanner (GE Vingmed, Horten, Norway), using a phased array transducer. The patients were examined before coronary angiography. Three consecutive cycles in 3 apical planes (4-chamber, 2-chamber, and long axis) and 3 short-axis planes (mitral valve, papillary muscle, and apical) were obtained by conventional 2D gray-scale echocardiography, using second harmonic imaging. Loops were digitally stored and later analyzed off-line using Echo-Pac version 7.0.0. (GE Vingmed). All examinations were performed by 1 operator. LV ejection fraction (LVEF) was calculated from 4-chamber and 2-chamber images, using the modified Simpson rule. Wall motion score was assessed in a 16-segment model.15 Segmental wall motion was judged by an experienced cardiologist as normal=1, hypokinetic=2, akinetic=3, and dyskinetic=4. Wall motion score index (WMSI) represents the average value of analyzed segments.

Strain Analyses

Longitudinal, circumferential, and radial strain was measured by speckle tracking echocardiography in a 16-segment LV model.10,13 Peak negative systolic strain, which represents the maximum systolic longitudinal or circumferential shortening, was noted for each segment. For radial strain, the peak positive systolic value was used. As a measure of global systolic function, values of all segments were averaged to obtain global longitudinal strain (GLS), global circumferential strain (GCS), and global radial strain (GRS). Torsion was assessed as the difference in rotation between the basal and apical planes. All echocardiographic and strain analyses were performed separately and blinded to other patient data.

ECG

For ST-segment analyses, the ECG obtained in the emergency room at referring hospital was used. Evidence of ischemia was defined as any ST-deviation >0.5 mm or symmetrical T-wave inversion >3 mm in 2 or more contiguous leads (10 mm/mV). The sum of all ST-segment deviations exceeding 0.5 mm was recorded.

CE-MRI

Final infarct size was quantified by MRI at follow-up 9±3 months after inclusion, using a 1.5-T unit (Magnetom Sonata, Siemens, Erlangen, Germany) on 29 patients and a 3-T unit (Philips Medical Systems, Best, The Netherlands) on 32 patients. Late-enhancement images were obtained 10 to 20 minutes after intravenous injection of 0.1 to 0.2 mmol/kg gadopentetate dimeglumine (Magnevist, Schering, Berlin, Germany) in multiple short-axis slices covering the entire LV. Typical image parameters were slice thickness, 8 mm; gap, 2 mm; and inversion time, 270 ms. On each short-axis image, total myocardial area as well as area of infarcted myocardium was manually drawn (PACS, Sectra, Sweden). Final infarct size was calculated as infarct volume as a percentage of total myocardial volume. Segmental transmurality was calculated in a 16-segment LV model as infarct volume divided by myocardial volume per segment, and segments with ≥50% contrast enhancement were judged transmurally infarcted.16 Both short- and long-term mortality rates have been demonstrated to be increased in patients with infarct size ≥12%.1,17 Therefore, patients were dichotomized by infarct size using 12% as cutoff. Figure 1 is an illustration of MRI images and strain values from 1 patient.

Coronary Angiography

Coronary angiography was performed by standard (Judkins) technique, using digital image acquisition and storage. Revascularization was not part of the study protocol and was performed on clinical indication. Compliant to current guidelines, complete revascularization was attempted. All analyses were performed in retrospect by a single experienced invasive cardiologist, blinded to the results of the other imaging studies.

Statistical Analyses

Continuous variables are presented as mean±SD or median (interquartile range [IQR]). The relationship between infarct size and echocardiographic parameters were analyzed by bivariate correlation. Differences between groups were analyzed with the Student t test, Mann–Whitney U test, or Kruskal-Wallis test. Categorical variables are presented as numbers (percentage) and were analyzed by χ² test or Fisher exact test. We used receiver operator analysis (ROC) curves to identify the cutoff for optimal sensitivity and specificity to detect infarct size ≥12% of total LV myocardial volume.17 Area under the curve (AUC), negative predictive value, positive predictive value, and accuracy (overall fraction correct) are reported. The AUC is a measure of discriminating power and represents the probability of the model to assign a higher probability to a correct case than to an incorrect case for all possible pairs of cases.

To assess the value of the potential combined use of echocardiographic parameters, a logistic regression model (enter) was used. LVEF and WMSI are conventional parameters of LV systolic function and were entered as covariates along with the strain parameter that demonstrated the best correlation to infarct size. The predicted probabilities from this model were used to calculate the AUC (equivalent to the c-statistic) of the combination of variables. Interobserver variability and intraobserver variability were calculated by intraclass correlation coefficient in 16 randomly selected patients. Comparison between ROC curves was performed according to the method described by Hanley and McNeil,18 using dedicated software (MedCalc Software version 10.3.1.0, Mariakerke, Belgium). All other statistical analyses were performed on SPSS version 13 (SPSS Inc, Chicago, Ill). The authors had full access to the data and take responsibility for its integrity. All authors have read and agree to the manuscript as written.

Results

Patient characteristics, risk factors, and medications are listed in Table 1.
Feasibility and Reproducibility

No patients were excluded because of suboptimal image quality on echocardiography. Longitudinal strain values could be obtained in 960 (98.4%), circumferential strain values in 877 (90.0%), radial strain in 864 (89%), and WMSl in 973 (99.7%) of all LV segments. LVEF could be calculated in all patients and LV torsion in 60 patients (98%). Intraclass correlation coefficients for interobserver and intraobserver variability of GLS were 0.94 and 0.96, respectively.

Angiographic Findings and Revascularization

All patients underwent coronary angiography. Significant stenosis was found in 54 patients (89%). Of these, 15 (28%) underwent surgical revascularization and 37 (69%) underwent PCI. Stents were used in 33 (89%) of PCI patients, and TIMI 3 flow in all attempted vessels was achieved in 32 (97%). Three patients who underwent PCI of the culprit lesion in addition had significant lesions in other vessels that were not amenable to PCI because of small vessel diameter. Between revascularization and follow-up, 1 patient had reinfarction with minimal enzyme release (troponin T, 0.18 μg/L) in the same vessel as was initially treated.

Infarct Size and Transmurality by CE-MRI

The distribution of infarct size is demonstrated in Figure 2. Median infarct size was 5.4% (IQR, 1.7% to 11.4%) of total LV myocardial volume. Thirteen patients (21%) had infarct size ≥12%, and 13 patients (21%) had no visible late enhancement on CE-MRI (infarct size, 0%). Of 976 analyzed segments, evidence of infarction by late enhancement was seen in 249 (26%). Of these, 199 (80%) demonstrated subendocardial and 50 (20%) transmural infarction.

Echocardiography and Infarct Size

A significant correlation was found between all parameters of LV systolic function and final infarct size (Figure 3). Importantly, the echocardiographic parameters of LV systolic function were able to discriminate between patients with final infarct size <12% or ≥12% (Table 2). To further elucidate whether the relationship between myocardial systolic func-

Table 1. Patient Characteristics, Risk Factors, and Medical Treatment

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Infarct Size &lt;12% (n=48)</th>
<th>Infarct Size ≥12% (n=13)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, y</td>
<td>57.8±7.2</td>
<td>57.2±13.6</td>
<td>0.81</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>27.2±3.0</td>
<td>28.5±3.2</td>
<td>0.20</td>
</tr>
<tr>
<td>Risk factors</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Male sex</td>
<td>37 (77)</td>
<td>11 (85)</td>
<td>0.72</td>
</tr>
<tr>
<td>Current smoker</td>
<td>20 (42)</td>
<td>7 (54)</td>
<td>0.43</td>
</tr>
<tr>
<td>Hypercholesterolemia</td>
<td>18 (38)</td>
<td>2 (15)</td>
<td>0.19</td>
</tr>
<tr>
<td>Hypertension</td>
<td>19 (40)</td>
<td>5 (39)</td>
<td>1.00</td>
</tr>
<tr>
<td>Diabetes mellitus</td>
<td>6 (13)</td>
<td>1 (8)</td>
<td>1.00</td>
</tr>
<tr>
<td>History of CAD</td>
<td>3 (6)</td>
<td>0 (0)</td>
<td>1.00</td>
</tr>
<tr>
<td>Medication</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Aspirin</td>
<td>48 (100)</td>
<td>13 (100)</td>
<td>1.00</td>
</tr>
<tr>
<td>Clopidogrel</td>
<td>48 (100)</td>
<td>13 (100)</td>
<td>1.00</td>
</tr>
<tr>
<td>LMWH</td>
<td>47 (98)</td>
<td>13 (100)</td>
<td>1.00</td>
</tr>
<tr>
<td>β-blocker</td>
<td>35 (73)</td>
<td>10 (77)</td>
<td>1.00</td>
</tr>
<tr>
<td>Statin</td>
<td>44 (92)</td>
<td>12 (92)</td>
<td>1.00</td>
</tr>
<tr>
<td>Warfarin</td>
<td>1 (2)</td>
<td>0 (0)</td>
<td>1.00</td>
</tr>
<tr>
<td>ACE inhibitor or ARB</td>
<td>14 (29)</td>
<td>3 (23)</td>
<td>1.00</td>
</tr>
<tr>
<td>GpIIbIIIa inhibitor</td>
<td>1 (2)</td>
<td>0 (0)</td>
<td>1.00</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD or n (%). Hypercholesteremia was defined as use of statins or cholesterol >240 mg/dL. Hypertension was defined as use of antihypertensive medication. BMI indicates body mass index; CAD, coronary artery disease; LMWH, low-molecular-weight heparin; ACE, angiotensin-converting enzyme, ARB, angiotensin receptor blocker; GpIIbIIIa, glycoprotein IIbIIIa.
tion and final MI size is time-dependent, we dichotomized patients into those arriving early (day 1, n = 9) and those arriving late (days 2 to 3, n = 52). For patients arriving early, the correlation between myocardial systolic function and final infarct size was excellent (GLS, \( r = 0.94, P < 0.001 \); WMSI, \( r = 0.88, P = 0.002 \)). For patients arriving late, the correlation was good (GLS, \( r = 0.65, P < 0.001 \); WMSI, \( r = 0.72, P < 0.001 \)).

Among strain parameters, GLS demonstrated the closest correlation to infarct size and was therefore entered as a covariate in the logistic regression model, along with LVEF and WMSI. Details are reported in Tables 3 and 4. WMSI and GLS remained significant predictors, LVEF did not. ROC analysis (Figure 4 and Table 5) demonstrate that GLS and WMSI had excellent ability to identify patients with infarct size \( \geq 12\% \), superior to that of GCS, GRS, LVEF, and torsion. Interestingly, the AUC from the logistic regression model (AUC, 0.98; 95% confidence interval, 0.90 to 1.00) was not significantly different compared with WMSI or GLS alone. None of the predictor variables were affected by age or sex.

LV systolic function was reassessed by GLS at follow-up. A persistent impairment was found in patients with infarct size \( \geq 12\% \) compared with those with smaller infarcts (GLS, 14.1±2.0% versus 17.5±2.2%; \( P < 0.001 \)). A small but significant improvement of systolic function was observed, as mean change in GLS was \(-1.0\pm1.9\% \) (\( P < 0.001 \) by paired Student \( t \) test). The observed change in GLS did not correlate to final infarct size (\( r = -0.1, P = 0.47 \)).

**Angiographic Findings and Infarct Size**

Infarct size was similar in patients with single-vessel versus multivessel disease (6.3% [IQR, 2.5% to 14.7%] versus 6.0% [IQR, 2.1% to 12.0%]; \( P = 0.86 \)). However, infarct size was dependent on patency of the infarct related artery (IRA). In patients with an occluded IRA, median infarct size was 8.3%.

![Figure 2. Infarct size. Bar graph demonstrating the distribution of final infarct size.](image)

![Figure 3. Scatterplots of infarct size by CE-MRI and echocardiographic parameters of LV systolic function. Dashed lines indicate the cutoff value to identify infarct size \( \geq 12\% \) (Table 5). Pearson correlation coefficients are reported in the figure. Rank correlations were similar (WMSI–Spearman \( \rho = 0.65, P < 0.001 \); GLS–Spearman \( \rho = 0.59, P < 0.001 \)).](image)
Table 2. Echocardiographic Findings by Infarct Size

<table>
<thead>
<tr>
<th>Infarct Size</th>
<th>LVEF, %</th>
<th>WMSI</th>
<th>GLS, %</th>
<th>GCS, %</th>
<th>GRS, %</th>
<th>Torsion, degrees</th>
<th>EDV, mL</th>
<th>ESV, mL</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt;12% (n=48)</td>
<td>57.5±7.7</td>
<td>1.08</td>
<td>-16.9±2.2</td>
<td>-22.1±3.8</td>
<td>35.0±13.2</td>
<td>13.2±6.7</td>
<td>112±25</td>
<td>48±14</td>
</tr>
<tr>
<td>≥12% (n=13)</td>
<td>48.5±9.3</td>
<td>1.44</td>
<td>-12.6±1.7</td>
<td>-18.2±5.5</td>
<td>27.6±8.3</td>
<td>10.2±8.2</td>
<td>121±27</td>
<td>62±22</td>
</tr>
<tr>
<td>P Value</td>
<td>0.001</td>
<td>&lt;0.001</td>
<td>0.030</td>
<td>0.018</td>
<td>0.173</td>
<td>0.226</td>
<td>0.001</td>
<td>0.031</td>
</tr>
</tbody>
</table>

Data are presented as mean±SD or median (IQR). EDV indicates end-diastolic volume; ESV, end-systolic volume.

Infarct size was not significantly different dependent on coronary territory (Table 6), but, numerically, smaller infarcts were found in the right coronary artery (RCA) territory. Choice of surgical versus percutaneous revascularization was not significantly associated with final infarct size.

**ECCG, Troponin T, and Clinical Findings Versus Infarct Size**

Twenty-two patients (36%) had evidence of ischemia on the ECG, as defined above. Compared with patients without ischemic changes, these had larger infarcts (7.3% [IQR, 4.4% to 17.5%] versus 3.2% [IQR, 0% to 10.4%], P=0.008). However, ischemic ECG changes were found in only 7 of 13 patients (54%) with infarct size ≥12%, and prevalence of ischemic ECG findings was not statistically different from patients with smaller infarcts (54% versus 31%, P=0.19). The sum of ST-segment deviations did not correlate to infarct size (r=0.15, P=0.26). Serum levels of troponin T obtained immediately before coronary angiography correlated significantly to infarct size (r=0.64, P<0.001) and could identify patients with infarct size ≥12% with sensitivity 63% and specificity 91%, using 1.4 ng/mL as cutoff. No significant differences were found in clinical parameters listed in Table 7, nor in risk factors or medication listed in Table 1.

**Discussion**

Our study demonstrates the use of a noninvasive imaging modality applied before revascularization to predict final infarct size in NSTEMI patients. We found a significant correlation between echocardiographic parameters of LV systolic function and final infarct size. Because acute coronary occlusion is accompanied by very rapid alterations in myocardial systolic function,7 the method may be applicable even in the very early phase in the development of MI. This has important clinical implications because it may provide a means for early identification of patients who have substantial infarction. Current guidelines recommend acute reperfusion therapy only in clinically unstable NSTEMI patients.19 This strategy may be inadequate in the subgroup of NSTEMI patients who have substantial infarction. These patients have a high prevalence of total occlusions and are likely to benefit from acute reperfusion therapy, as it may reduce infarct size and salvage viable myocardium. Importantly, in current clinical practice, echocardiography is the only bedside, noninvasive method to identify these patients.

**Infarct Size and Prognosis**

Final infarct size is a strong predictor of mortality and major adverse cardiovascular events.1,17,20,21 Current reperfusion therapy is highly effective, and the relative reduction of infarct size achieved is typically 40% by thrombolysis and 60% by primary PCI.22 The reduced mortality rate observed in the reperfusion era is largely attributable to reduction of final infarct size.2 A treatment that results in a 3% to 5% absolute reduction of infarct size is associated with improved survival and reduction of clinical events.20 Previous studies

![Figure 4. ROC analysis set to identify infarct size ≥12% of total LV myocardial volume.](Image)
have demonstrated increased mortality rate to be associated with infarct size $\geq 12\%$.\(^{1,17}\) In addition, 1 large trial found 12% to be median infarct size in STEMI patients treated by acute reperfusion.\(^{17}\) Hence, we chose 12% as cutoff to identify patients at increased risk in our study.

### Infarct Size in NSTEMI Patients

Since the introduction of more sensitive biochemical markers of myocardial necrosis, an increasing number of patients with non-ST-segment elevation acute coronary syndrome are diagnosed with MI.\(^{23}\) Hence, the NSTEMI population comprises a large number of patients with only minimal myocardial necrosis, leaving “typical” infarct size in this group relatively small. Accordingly, in our study, 13 patients (21%) had no visible area with late enhancement, and 30 patients (49%) had small infarcts (<5% of total LV volume).

It is important to recognize that the difference between STEMI and NSTEMI by definition is electrophysiological and not pathophysiological. As this and other studies show, a subgroup of NSTEMI patients have substantial infarction,\(^{5,6}\) and there is a significant overlap in final infarct size between STEMI and NSTEMI patients.\(^{6}\) Even patients with a normal ECG may develop large infarctions.\(^{5}\) The latter corresponds to our findings, as only 7 of 13 patients (54%) with infarct size $\geq 12\%$ had any evidence of ischemia in the ECG.

As several factors affect LV systolic function in the acute ischemic setting, no absolute relationship between final infarct size and LV systolic function can be expected. In the present study, we demonstrate correlation coefficients of 0.74 and 0.68 for WMSI and GLS, respectively. Hence, variability in infarct size can explain approximately 50% of the observed variability in LV systolic function assessed by GLS or WMSI. Stunning, hibernation, ongoing ischemia without necrosis, and differences in loading conditions are likely to account for much of the remaining variability. The relatively small improvement in LV systolic function from baseline to follow-up may indicate that stunning or hibernation do not play major roles and that the observed reduction in LV systolic function largely persists despite revascularization. As the observed improvement of myocardial systolic function did not correlate to infarct size, stunning and hibernation were evenly distributed between different levels of infarct size and are not likely to have confounded our results.

### Prediction of Infarct Size by Echocardiography

In the present study, WMSI and GLS were independent predictors of infarct size $\geq 12\%$ by logistic regression analysis (Table 3). However, the discriminating power of both parameters used alone was excellent, and the combined use did not significantly increase power. WMSI is an established parameter of LV systolic function, validated as a prognostic indicator after MI.\(^{24}\) However, it is semiquantitative, experience-dependent, and based on subjective interpretation of myocardial motion. Longitudinal strain by speckle tracking echocardiography is a relatively new parameter that has not yet reached widespread use in clinical practice. The reported values of GLS were substantially lower compared with those previously reported in healthy individuals (GLS $\leq 21.7 \pm 1.6\%)$.\(^{25}\) It can be calculated using commercially available dedicated software from several vendors. The calculation is semiautomated; it provides an objective measure of segmental systolic shortening on a linear scale and has also previously demonstrated excellent feasibility and reproducibility.\(^{26}\) Recently, it has also been shown to be superior to LVEF and WMSI for prediction of mortality.\(^{13}\)

As previously demonstrated by others, circumferential strain is less accurate than longitudinal strain in predicting subendocardial infarction.\(^{27}\) In NSTEMI, the infarction is predominantly subendocardial, and 80% of infarcted seg-

### Table 6. Infarct Size by Infarct Location

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Infarct Size $&lt; 12%$ (n=48)</th>
<th>Infarct Size $\geq 12%$ (n=13)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure, mm Hg</td>
<td>144±26</td>
<td>141±28</td>
<td>0.75</td>
</tr>
<tr>
<td>Diastolic blood pressure, mm Hg</td>
<td>81±16</td>
<td>77±16</td>
<td>0.42</td>
</tr>
<tr>
<td>Heart rate, beats/min</td>
<td>64±10</td>
<td>69±11</td>
<td>0.17</td>
</tr>
<tr>
<td>Recurrent chest pain</td>
<td>6 (13)</td>
<td>1 (8)</td>
<td>1.00</td>
</tr>
<tr>
<td>Ischemic ECG</td>
<td>15 (31)</td>
<td>7 (54)</td>
<td>0.19</td>
</tr>
<tr>
<td>Sum of ST-deviation, mm</td>
<td>0.0 (0.0–1.0)</td>
<td>1.0 (0.0–2.5)</td>
<td>0.18</td>
</tr>
</tbody>
</table>

Values are mean±SD, median (IQR) or n (%).
ments were subendocardial in our study. Subendocardial layers of the myocardium contain predominantly longitudinal fibers, which may explain why longitudinal strain was more accurate. Second, longitudinal strain tracks motion parallel to the ultrasound beam, where resolution is optimal, in contrast to circumferential and radial strain, where motion is tracked in all directions. In addition, circumferential strain and radial strain are calculated from short-axis images, where image planes are not well standardized, and there is considerable through plane motion in basal parts of the heart. The reason why circumferential and radial strains were less accurate may therefore partly be caused by methodological reasons.

Diagnostic Tools for Early Assessment of Infarct Size

After acute occlusion of a coronary artery, the time window for myocardial salvage is narrow. Therefore, a tool to predict infarct size should be based on information that can be obtained in the emergency room. Recent data suggest that 24% of all NSTEMI patients have total occlusion of the infarct-related artery, and the urgent need to identify patients who have substantial infarction has recently been pointed out.24 In the present study, clinical characteristics and ECG were unable to identify patients with substantial infarction. Biochemical markers of myocardial necrosis have demonstrated significant correlation to final infarct size in this and numerous other studies. However, due to the slow release of these biomarkers, only peak values or values from a sample taken at a fixed time point minimum 12 hours after onset of chest pain have been validated.20 Elevated biochemical markers from a very early sample obtained in the emergency room are certainly evidence of myocardial necrosis, but not the extent of such, and are not likely to be useful in identification of patients with substantial infarction. Myocardial systolic function, on the other hand, is dependent on a continuous blood supply and deteriorates within seconds of acute coronary occlusion,7 even before the onset of necrosis. Hence, an important advantage of echocardiography is that it can identify patients with developing substantial infarction at a time point when intervention can still achieve significant myocardial salvage. In our study, a stronger relationship between final infarct size and myocardial systolic function was found in patients arriving early, compared with those arriving late. This indicates that the association is present at an early stage during development of MI. ST-segment deviations in the ECG, when present, also appear rapidly after onset of ischemia. However, in our study neither the presence nor magnitude of ST-segment deviation was able to identify patients with substantial infarction.

Limitations

Echocardiography was not performed in the emergency room, but on arrival at the invasive center 1 to 3 days later. However, as acute coronary occlusion is accompanied by very rapid alterations in myocardial systolic function,7 similar findings can be expected even at a very early stage. Nevertheless, infarct expansion and edema developing during the first hours may be accompanied by alterations in systolic function, and our model requires prospective validation applied in the emergency room. We do not know how well our discrimination model will fit new data. Nevertheless, because 2 different methods (GLS and WMSI), assessed by 2 different observers, both demonstrated excellent discrimination, it is unlikely that this is a chance finding. Prior MI was an exclusion criterion in this study. Distinguishing between acute and chronic MI is difficult and was not the aim of this study. However, echocardiography may still be of great value for patients with chronic MI. Development of new areas with reduced systolic function might indicate high risk and lend support to choice of an aggressive treatment strategy.

Conclusion

Echocardiographic parameters of LV systolic function obtained before revascularization correlate to final infarct size in NSTEMI patients. Our findings demonstrate that patients with a potential for substantial myocardial damage may be identified with high accuracy. Because echocardiography is easily performed in the emergency room, it may serve as a tool for selection of NSTEMI patients who should be considered for urgent revascularization to limit infarct size.

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Disclosures

None.

References


**CLINICAL PERSPECTIVE**

Infarct size is a strong prognostic indicator after myocardial infarction. In patients with ST-segment elevation myocardial infarction, reperfusion therapy is effective in reducing infarct size and has improved survival. However, a proportion of non–ST-segment–elevation myocardial infarction patients also experience substantial myocardial infarction, but these patients are rarely eligible for acute reperfusion therapy. Severe myocardial ischemia and necrosis is not always reflected in ST segment elevation, and these patients often do not reveal clinical signs of instability. Hence, there is a need for other modalities to identify these patients. Abundant evidence has demonstrated that myocardial ischemia causes rapid deterioration of myocardial systolic function. Echocardiography is a fast and available tool for estimation of myocardial systolic function. Left ventricular ejection fraction and wall motion score index are conventional parameters used to estimate left ventricular systolic function. Recently, strain measurement by speckle tracking has emerged as a new modality. The present study demonstrates that non–ST-segment–elevation myocardial infarction patients with substantial myocardial infarction can be identified by echocardiographic parameters of left ventricular systolic function. Global longitudinal strain and wall motion score index both demonstrate excellent discriminating power. Echocardiography and particularly strain echocardiography may facilitate the evaluation of interventions that impact outcomes in patients with non–ST-segment–elevation myocardial infarction.
Strain Echocardiography and Wall Motion Score Index Predicts Final Infarct Size in Patients With Non–ST-Segment–Elevation Myocardial Infarction


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