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 after MI but is generally confined to patients with ST-segment–elevation MI (STEMI). ST-segment elevation in the ECG has high specificity but suboptimal sensitivity for detection of MI and acute coronary occlusion3 and has demonstrated only modest correlation to infarct size.4 A number of patients with non–ST-segment–elevation MI (NSTEMI) have substantial infarction,5,6 but these patients rarely fulfill criteria for acute reperfusion therapy. In current clinical practice, no instant method for early assessment of final infarct size in patients with NSTEMI exists.

Clinical Perspective on p 194

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-V₄) 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; extensive comorbidity with short life expectancy; 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 immediately 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.15 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 parameters. The model as infarct volume divided by myocardial volume per segment, and segments with ≥50% contrast enhancement were judged transmurally infarcted. The relationship between infarct size and strain values in the different models was assessed by intraclass correlation coefficient in 16 randomly selected cases. 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 WMS 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).

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%). Thirty patients (49%) had final infarct size <5% of total LV mass. 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-
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 ≥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 ≥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±1.9% (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%
Our study demonstrates the use of a noninvasive imaging method to identify these patients.

**Table 3. Logistic Regression Model for Infarct Size ≥12%**

<table>
<thead>
<tr>
<th>Variable</th>
<th>Coefficient Estimate</th>
<th>Wald $\chi^2$</th>
<th>$P$</th>
<th>Odds Ratio (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intercept</td>
<td>-1.07</td>
<td>0.009</td>
<td>0.92</td>
<td></td>
</tr>
<tr>
<td>LVEF*</td>
<td>0.04</td>
<td>0.23</td>
<td>0.64</td>
<td>1.04 (0.89-1.22)</td>
</tr>
<tr>
<td>WMSI†</td>
<td>1.27</td>
<td>5.23</td>
<td>0.02</td>
<td>3.55 (1.20-10.47)</td>
</tr>
<tr>
<td>GLS*</td>
<td>1.26</td>
<td>6.46</td>
<td>0.01</td>
<td>3.51 (1.33-9.25)</td>
</tr>
</tbody>
</table>

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**Table 4. Classification Table**

<table>
<thead>
<tr>
<th>Predicted Infarct ≥12%</th>
<th>Observed</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0</td>
</tr>
<tr>
<td>Infarct ≥12%</td>
<td>47</td>
</tr>
</tbody>
</table>

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**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, 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. 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. Current reperfusion therapy is highly effective, and the relative reduction of infarct size achieved is typically 40% by thrombolysis and 60% by primary PCI. The reduced mortality rate observed in the reperfusion era is largely attributable to reduction of final infarct size. A treatment that results in a 3% to 5% absolute reduction of infarct size is associated with improved survival and reduction of clinical events. Previous studies have suggested that mortality and major adverse cardiovascular events can be reduced by 13% to 19% by revascularization therapy only in clinically unstable NSTEMI patients.
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 di-
agnosed with MI.23 Hence, the NSTEMI population com-
prises a large number of patients with only minimal myocard-
ial 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 my 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 anal-
ysis (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 param-
eter 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 echocardiogra-
phy 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 re-
ported in healthy individuals (GLS = \(-21.7 \pm 1.6\%\)).25 It can be
calculated using commercially available dedicated software
from several vendors. The calculation is semiautomatic; 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%</th>
<th>Infarct Size (\geq12%)</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.7 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.

Sources of Funding

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Disclosures

None.

References

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.
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