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Early prediction of myocardial viability after acute myocardial infarction by two-dimensional speckle tracking imaging

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Abstract

Background Identifying the transmural extent of myocardial necrosis and the degree of myocardial viability in acute myocardial infarction (AMI) is important clinically. The aim of this study was to assess myocardial viability using two-dimensional speckle tracking imaging (2D-STI) in patients with AMI. **Methods** 2D-STI was performed at initial presentation, three days, and six months after primary percutaneous coronary intervention (PCI) in 30 patients with AMI, who had a left anterior descending coronary artery (LAD) culprit lesion. In addition, 20 patients who had minimal stenotic lesions (< 30% stenosis) on coronary angiography were also included in the control group. At six months dobutamine echocardiography was performed for viability assessment in seven segments of the LAD territory. According to the recovery of wall motion abnormality, segments were classified as viable or non-viable. **Results** A total of 131 segments were viable, and 44 were nonviable. Multivariate analysis revealed significant differences between the viable and nonviable segments in the peak systolic strain, the peak systolic strain rate at initial presentation, and peak systolic strain rate three days after primary PCI. Among these, the initial peak systolic strain rate had the highest predictive value for myocardial viability (hazard ratio: 31.22, $P < 0.01$). **Conclusions** 2D-STI is feasible for assessing myocardial viability, and the peak systolic strain rate might be the most reliable predictor of myocardial viability in patients with AMI.

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Keywords: Acute myocardial infarction; Two-dimensional speckle tracking imaging; Viable myocardium

1 Introduction

Assessment of the viable myocardium forms is an important basis for revascularization in patients with acute myocardial infarction (AMI).[1–3] This can be assessed using dobutamine echocardiography, F18-fluorodeoxyglucose (FDG) positron emission tomography (PET), perfusion scans, 64-slice computed tomography (CT), and contrast-enhanced magnetic resonance imaging (MRI).[4–10] Although a variety of different imaging modalities have been used, these approaches are often limited by their availability, cost, technical difficulty, subjective character, or a combination of these factors. Echocardiography, on the other hand, is more easily available and feasible technique in the acute setting. The use of strain (deformation) by Doppler quantifies regional myocardial deformation, and can demonstrate abnormal myocardial function due to ischemia. Some investigators proposed that strain via tissue Doppler images (TVI) could serve as a marker of viability.[11–13] Although TVI and Doppler strain measurements have been used most commonly in this clinical setting, they are limited by the angle dependence in Doppler.

To eliminate the problem of angle-dependency, a method to measure strain based on two-dimensional speckle tracking imaging (2D-STI) has been developed, which provides quantitative and angle-independent measurements for assessing the myocardial strain.[11] However, there is no established echocardiographic method to identify viable myocardium using 2D-STI. The aim of this study was to assess myocardial viability using 2D-STI in patients with AMI.

2 Methods

2.1 Study population

This was a prospective observational study. Seventy six consecutive patients with first ST-segment elevation myocardial infarction (STEMI) in the anterior wall were prospectively enrolled. All patients had typical chest pain and sustained ST-segment elevation on electrocardiogram. Typ-

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Typical chest pain means that the chest pain lasted more than 30 min and was resistant to nitrates. ST-segment elevation means that electrocardiography revealed > 0.2 mV ST elevations in at least two contiguous leads. Among them, 30 patients (22 men, 63.6 years of age) who had left anterior descending artery (LAD) culprit lesion and thrombolysis in myocardial infarction (TIMI) flow grade 0 or 1 confirmed by coronary angiography were enrolled. Patients who had moderate to severe valvular heart disease, other significant systemic disease, significant arrhythmia, who received percutaneous coronary intervention (PCI) after 12 h of symptom onset, and who received thrombolytic therapy instead of PCI were excluded. In addition, 20 patients who had minimal stenotic lesions (< 30% stenosis) on coronary angiography were also included in the control group. The ethnical review board at Kyung Hee University (Seoul, South Korea) approved the study protocol. All subjects provided written informed consent.

Among the 30 STEMI subjects, 18 patients had stenosis in single LAD lesion, 6 patients in 2-vessel disease, and 6 individuals had three-vessel disease. After angioplasty, no patients had significant residual stenosis in the LAD, and none exhibited a no-reflow phenomenon. Drug-eluting stents were implanted in all patients.

2.2 Echocardiography

Standard echocardiography with 2D-STI was performed at initial presentation, 3 days, and 6 months after PCI using a 3.5-MHz transducer with a commercially available system (Vivid 7, GE Vingmed, Horten, Norway) in both the AMI and control groups.[14] All echocardiography was performed by a certified sonographer who qualified from the American Registry for Diagnostic Medical Sonography (Registered Diagnostic Cardiac Sonographer) with 14 years of experience. After images acquisition, a cardiologist who is specialized professor on echocardiography confirmed the echocardiographic images. Digital loops were stored in the hard disk of the echocardiography machine for on-line and off-line analyses, and were also transferred to a workstation (EchoPac, GE Vingmed) for off-line analysis. Dobutamine echocardiography was also performed six months after PCI. The left ventricular (LV) was divided into six equal segments or regions, as defined by the American Society of Echocardiography.[14] According to the recovery of the regional wall motion abnormality at 6 months, segments of the LAD territory were classified as viable or non-viable. The strain and strain rate of the viable and non-viable segments were compared at initial presentation, 3 days, and 6 months after PCI (Figure 1).

The recovery of the regional wall motion abnormality was defined as an improvement in the regional wall motion from akinesia or dyskinesia to normal or mild hypokinesia. A viable segment was defined as recovery of the regional wall motion abnormality in echocardiography or dobutamine echocardiography six months after PCI. Conversely, a non-viable segment was defined as no recovery of the regional wall motion abnormality in either echocardiography or dobutamine echocardiography 6 months after PCI (Figure 1). The myocardial strain and strain rate were measured using 2D-STI in the seven mid- and apical segments

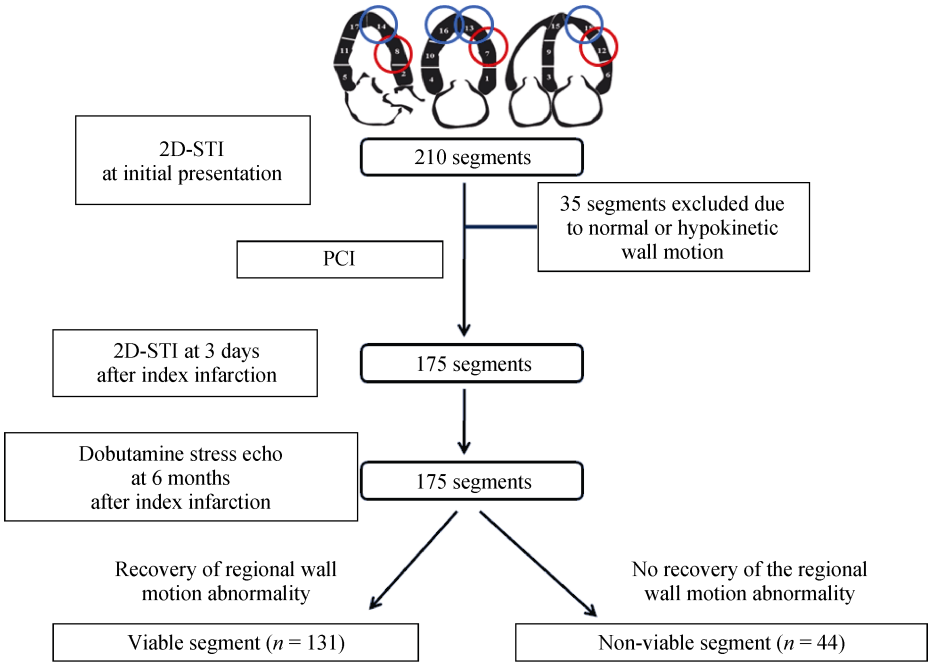

Figure 1. Study overview. PCI: percutaneous coronary intervention; 2D-STI: two-dimensional speckle tracking imaging.

of the LAD territory in the apical two-chamber, three-chamber, and four-chamber views (Figure 1). The images were processed using a workstation with software (EchoPAC Q-analysis, General Electric, Waukesha, WI, USA).[15,16] The endocardial border was traced in an end-systolic frame. The software selected six equidistant tissue-tracking regions of interest in the myocardium automatically, and the outer border was adjusted approximately to the epicardial border. The software selected suitable stable acoustic objects for tracking automatically, searched for them in the next frame using an absolute differences algorithm, and provided a tracking score to measure the degree of decorrelation of the block-matching (1: excellent; 3: poor). An assumption was made that the natural acoustic markers changed position from frame-to-frame in conjunction with the motion of the surrounding tissue.[15–17] All segments could be evaluated and had a tracking score of one, except for two segments that had scores of 1.1. The computer then provided a profile of the radial and circumferential strain (%) over time. Apical views revealed that the circumferential strain was deformed along the curvature of the LV. As previously reported, the inter- and intra-observer variability were well controlled.[18–20] For reproducibility of measurements, two independent observers repeated ten measurements of 2D-STI strain and strain rate parameters. Differences in measurements by the two observers were obtained for estimation of interobserver variability. The same observer repeated the ten measurements after three month interval, and intraobserver variability was calculated.

The outcome measurements included the lengthening strain (ε-L), end systolic strain (ε-ES), and peak systolic strain (ε-PS). The lengthening shortening (LS) ratio and postsystolic strain (PSS) were then calculated from these strain parameters. The peak systolic strain rate (SSR), early diastolic strain rate (ESR), and late diastolic strain rate (ASR) were also measured (Figure 2).

2.3 Statistics

Statistical analyses were carried out using the SPSS Statistics for Windows ver. 17.0 software package (SPSS Inc, Chicago, IL, USA). A two-sided $P < 0.05$ was considered significant. Continuous variables, presented as means ± SD, were compared using the independent *t*-test or Mann–Whitney *U* test. Categorical variables, presented as frequencies and percentages, were compared using the chi-square test or Fisher's exact test when appropriate. A logistic regression model was used to determine the predictive ability of the continuous variables for identifying myocardial viability. Correlations for reproducibility analysis were performed using the Pearson correlation coefficient. As an alternative to the logistic model, receiver operating characteristic (ROC) curves were also computed for prediction of myocardial viability among the different measurements.

3 Results

In the 30 patients with STEMI of the LAD culprit lesion, a total of 175 segments were evaluated, of which 131 segments were viable and 44 were non-viable. A total of 140 segments in the LAD territory were also analyzed in the 20

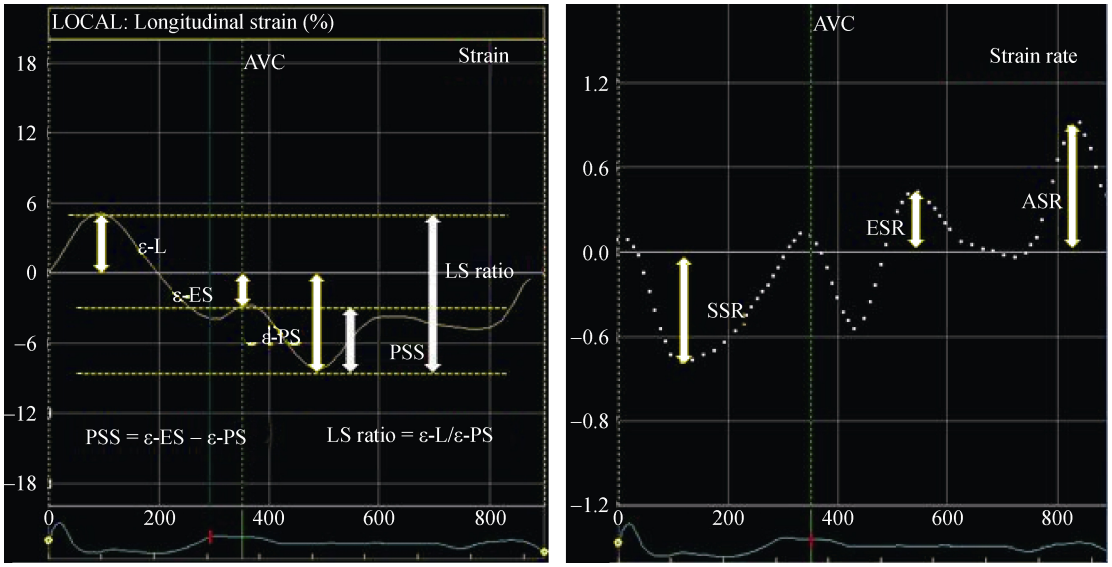

Figure 2. Strain and strain rate parameters. AVC: aortic valve closure; ε-L: lengthening strain; ε-ES: end systolic strain; ε-PS: peak systolic strain; LS ratio: lengthening–shortening ratio; PSS: postsystolic strain; SSR: peak systolic strain rate; ESR: early diastolic strain rate; ASR: late diastolic strain rate.

patients for the control group. The baseline characteristics of all study subjects are shown in Table 1. The Killip class, left ventricle ejection fraction (LVEF), and wall motion score index (WMSI) were significantly worse in patients with AMI compared with controls. There were no significant differences between groups in the other characteristics, including age, gender, and past medical history (angina, diabetes, hypertension, dyslipidemia, and smoking).

Table 1. Baseline characteristics.

Variable	STEMI (n = 30)		Control (n = 20)	P value
	Viable (n = 21)	Non-viable (n = 9)		
Age, yrs	62.6 ± 11.9	66.0 ± 9.4	63.2 ± 10.2	NS
Male gender, n (%)	16 (76%)	6 (67%)	14 (70%)	NS
Systolic blood pressure, mmHg	129.0 ± 25.8	123.3 ± 30.4	130.7 ± 21.3	NS
Diastolic blood pressure, mmHg	74.8 ± 12.5	74.4 ± 15.9	80.2 ± 7.7	NS
Heart rate, time/min	78.2 ± 15.0	72.4 ± 28.3	79.8 ± 15.7	NS
Killip class (> 2), n (%)	5 (24%)	6 (67%)	0 (0%)	< 0.05
Symptom-to-door time, min	203 ± 145	184 ± 155	-	-
Door to balloon time, min	82 ± 27	82 ± 17	-	-
Past medical history, n (%)				
Angina	1 (5%)	1 (11%)	2 (10%)	NS
Diabetes	5 (24%)	4 (44%)	3 (15%)	NS
Hypertension	12 (57%)	6 (67%)	8 (40%)	NS
Dyslipidemia	2 (9%)	2 (22%)	4 (20%)	NS
History of smoking	13 (62%)	6 (67%)	8 (40%)	NS
Echocardiographic findings				
LV end-diastolic volume, mL	94.5 ± 30.4	96.5 ± 22.9	82.2 ± 31.5	NS
LV end-systolic volume, mL	45.5 ± 23.4	52.9 ± 22.9	30.4 ± 14.7	< 0.01
LV ejection fraction, %	51.5 ± 12.0	44.8 ± 13.9	63.1 ± 10.2	< 0.001
Wall motion score index	1.39 ± 0.31	1.61 ± 0.25	1.00 ± 0.00	< 0.001
E wave, cm/s	61.9 ± 22.2	67.6 ± 21.6	69.7 ± 20.3	NS
Deceleration time, ms	204.6 ± 43.7	169.5 ± 77.9	219.7 ± 73.7	NS
E/A ratio	0.8 ± 0.4	1.2 ± 0.7	1.0 ± 0.4	NS
E/E′ ratio	12.9 ± 3.8	16.4 ± 5.7	13.2 ± 5.3	NS
Angiographic findings				
IRA (LAD), n (%)	21 (100%)	9 (100%)	-	-
Associated lesion, n (%)				
LCX	5 (24%)	4 (44%)	-	-
RCA	4 (19%)	3 (33%)	-	-

Data are presented as means ± SD or n (%). P values were calculated for comparisons between the AMI and control groups. AMI: acute myocardial infarction; A wave: late diastolic inflow velocity; E wave: early diastolic inflow velocity; E′: early diastolic medial mitral annular velocity; IRA: infarct-related artery; LAD: left anterior descending artery; LCX: left circumflex artery; LV: left ventricular; NS: not significant; STEMI: ST segment elevation myocardial infarction; RCA: right coronary artery.

3.1 Strain and strain rate at initial presentation

Comparisons of the strain and strain rate at initial presentation in patients with AMI are shown in Table 2. There were significant differences in all strain parameters between the viable and non-viable segments at initial presentation. Specifically, ε-L, the LS ratio, and PSS were increased significantly in the non-viable segments of patients with AMI. In addition, the absolute values of ε-ES and ε-PS were reduced significantly in the non-viable segments. When the strain rate was compared, there were significant reductions in the absolute values of SSR, ESR, and ASR in the non-viable segments. There were no differences in the strain and strain rate between the control segment group and the viable segment group.

3.2 Serial change of strain and strain rate

Serial changes in the strain and strain rate at initial presentation, 3 days after PCI, 6 months after PCI, and after 6 months using dobutamine echocardiography in patients with AMI are shown in Figure 3. There were significant differences in all strain parameters between viable and non-viable segments. The difference in the LS ratio remained 3 days after PCI, 6 months after PCI, and after 6 months using dobutamine echocardiography. The difference in PSS continued, except for the measurements recorded 3 days after PCI. There were also significant differences in SSR, ESR,

Table 2. Comparison of the strain and strain rate at initial presentation in patients with AMI.

		Control segment (n = 140)	AMI		P value
			Viable segments (n = 131)	Non-viable segments (n = 44)	
Strain	ε-L, %	0.2 ± 0.7	1.4 ± 2.3	3.5 ± 4.0	< 0.001
	ε-ES, %	−21. 3 ± 5.6	−10.1 ± 4.7	−1.9 ± 4.2	< 0.001
	ε-PS, %	−21.6 ± 5.6	−12.3 ± 3.5	−7.2 ± 3.7	< 0.001
	LS ratio	0.01 ± 0.03	0.09 ± 0.15	0.30 ± 0.23	< 0.001
	PSS, %	0.28 ± 0.77	2.14 ± 2.79	5.38 ± 4.12	< 0.001
Strain rate	SSR, s^{-1}	−1.2 ± 0.4	−0.9 ± 0.4	−0.5 ± 0.2	< 0.001
	ESR, s^{-1}	1.6 ± 0.7	0.8 ± 0.4	0.5 ± 0.4	< 0.001
	ASR, s^{-1}	1.1 ± 0.5	1.0 ± 0.4	0.8 ± 0.4	< 0.01

Data are presented as means ± SD. P values were calculated for comparisons between viable and non-viable segments in AMI patients. AMI: acute myocardial infarction; ASR: late diastolic strain rate; ε-L: lengthening strain; ε-ES: end systolic strain; ε-PS: peak systolic strain; ESR: early diastolic strain rate; LS ratio: lengthening shortening ratio; PSS: postsystolic strain; SSR: peak systolic strain rate; STEMI: ST segment elevation myocardial infarction.

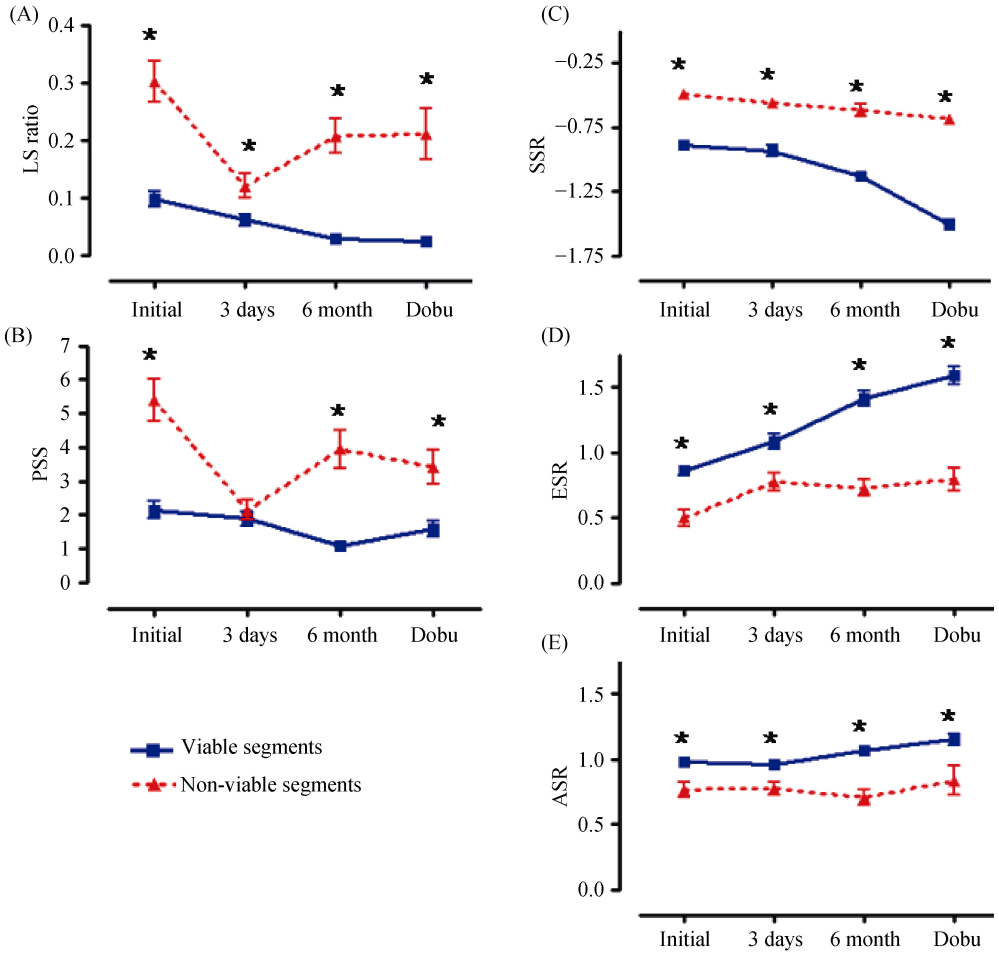

Figure 3. Serial changes in the strain and strain rate and a comparison of the strain in the viable and non-viable segments. (A): LS ratio; (B): SSR; (C): PSS; (D): ESR; (E): ASR. **P* <0.05 between the two groups. ASR: late diastolic strain rate; Dobu: dobutamine; ESR: early diastolic strain rate; LS ratio: lengthening shortening ratio; PSS: postsystolic strain; SSR: peak systolic strain rate.

and ASR between the viable and non-viable segments; these differences continued for 6 months. There were no differences in the serial changes in the strain and strain rate between the control segments and the viable segments.

3.3 Prediction of viable segments

Table 3 and Figure 4 summarize the 2D-STI strain and strain rate parameters that were predictive of myocardial viability. Univariate analysis revealed that all strain and strain rate parameters were significant predictors of myocardial viability in patients with AMI. Then, multivariate regression analysis showed that PSS [hazard ratio (HR): 1.29, 95% confidence interval (95%CI): 1.05–1.58], SSR (HR: 22.46, 95%CI: 2.25–224.49) at initial presentation, and SSR 3 days after PCI (HR: 31.22, 95%CI: 3.29–295.67) were meaningful parameters (Table 3). Among these, ROC curves revealed that initial SSR had the greatest ability to assess segmental myocardial viability (Figure 4). Using a cutoff of 0.72, initial SSR had a sensitivity of 88% and specificity of 69% for distinguishing viable from non-viable segments. In addition, the highest area under roc curves (AUCs) for ROC curves were obtained using initial SSR (AUC = 0.85), initial PSS (AUC = 0.78), and SSR 3 days after PCI (AUC = 0.79). The AUC for initial SSR was significant greater than was that for PSS and SSR 3 days after PCI. In addition, The SSR at 3 days after PCI had an incremental value for predicting myocardial viability (Figure 5).

The correlation coefficients of interobserver variability for ε-L, ε-ES, ε-PS, SSR, ESR and ASR were 0.92, 0.95, 0.91, 0.94, 0.92 and 0.90, respectively. The correlation coefficients of intraobserver variability for ε-L, ε-ES, ε-PS, SSR, ESR and ASR were 0.98, 0.94, 0.91, 0.98, 0.91 and 0.92, respectively.

4 Discussions

Viable myocardium demonstrates greater absolute ε-ES, ε-PS, SSR, ESR, and ASR values and a shorter ε-L, LS ratio,

Table 3. Prediction of viable segment.

		Univariate analysis		Multivariate analysis	
		***P* value**	**Hazard ratio (95% CI)**	***P* value**	**Hazard ratio (95% CI)**
Initial presentation	LS ratio	< 0.001	186.41 (26.23–1324.83)	0.29	4.72 (0.27–82.05)
	PSS, %	< 0.001	1.37 (1.16–1.46)	< 0.05	1.29 (1.05–1.58)
	SSR, s^{-1}	< 0.001	416.71 (52.07–3334.60)	< 0.01	22.46 (2.25–224.49)
	ESR, s^{-1}	< 0.001	0.11 (0.04–0.31)	0.22	0.45 (0.12–1.64)
	ASR, s^{-1}	< 0.001	0.25 (0.09–0.65)	0.07	0.28 (0.07–1.09)
Three days after PCI	LS ratio	< 0.05	37.59 (2.62–539.77)	0.74	2.26 (0.02–264.9)
	PSS, %	0.55	1.04 (0.91–1.19)	0.12	0.80 (0.61–1.06)
	SSR, s^{-1}	< 0.001	42.77 (9.24–197.88)	< 0.01	31.22 (3.29–295.67)
	ESR, s^{-1}	< 0.01	0.35 (0.17–0.74)	0.39	0.62 (0.21–1.85)
	ASR, s^{-1}	< 0.05	0.29 (0.11–0.78)	0.66	1.38 (0.32–5.93)

ASR: late diastolic strain rate; ESR: early diastolic strain rate; LS ratio: lengthening shortening ratio; SSR: peak systolic strain rate; PCI: percutaneous coronary intervention; PSS: postsystolic strain.

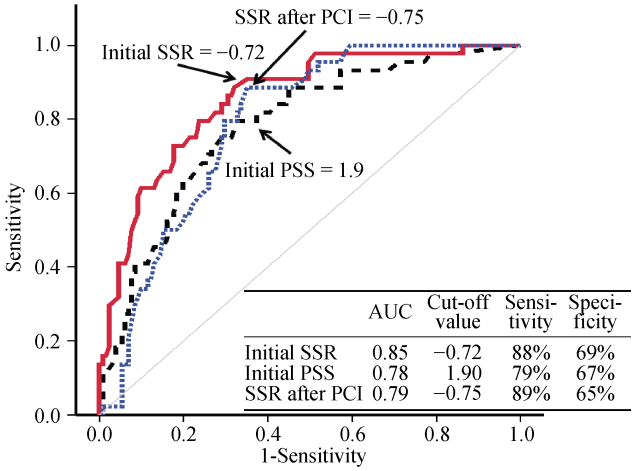

Figure 4. ROC curves, including the AUC and relevant cut-off values for the most pertinent strain and strain rate parameters. AUC: area under roc curve; SSR: peak systolic strain rate; PCI: percutaneous coronary intervention; PSS: postsystolic strain; ROC: receiver operating characteristic.

and PSS than non-viable myocardium does. The 2D-STI strain and strain rate parameters had high sensitivity but moderate specificity for detecting viability in patients with AMI. This study demonstrated that 2D-STI is useful for identifying viable myocardium early in patients with AMI. Among the strain and strain rate parameters measured, SSR 3 days after PCI had the highest predictive value. Therefore, SSR 3 days after PCI was proposed as a novel echocardiographic index with potential applications in AMI to differentiate between viable and non-viable myocardium.

Assessing viability in patients with AMI is important for determining patient prognosis and deciding whether revascularization is appropriate.[4,21,22] Several modalities—such as 2-D echocardiography, contrast-enhanced MRI, 64-slice CT, and F18-FDG PET—can be used to assess myocardial viability.[4–10] The sensitivity and specificity of these modalities are comparable.[9,10,23,24] However, the contrast-enhanced MRI and F18-FDG PET are expensive and not available to patients with acute conditions such as AMI. The 64-slice CT has the radiation hazard even this modality has been widely performed recently. Assessing myocardial viability based on WMSI during dobutamine echocardiography is difficult and subjective. Therefore, more feasible, inexpensive, quantitative, and rapid methods to assess myocardial viability are needed, especially in patients with AMI. Tissue Doppler imaging allows the non-invasive measurement of myocardial strain in the LV. Measuring strain and strain rate using low-dose dobutamine echocardiography was feasible, and their use in combination with assessing WMS improves the sensitivity of viability assessments made using dobutamine echocardiography.[25] Doppler-derived PSS represented a clinical method for identifying actively contracting and hence viable myocardium in dog models.[13] In addition, the LS ratio calculated using Doppler echocardiography identified areas of active contraction and necrosis in dog models of acute coronary occlusion.[12] In addition, a previous study found an inverse relationship between the segmental strain and the transmural extent of infarction in each segment, as determined using contrast-enhanced MRI after coronary reperfusion.[1] Analyzing segments using the peak systolic strain rate, as calculated from automated strain rate imaging analysis from the dobutamine stress echocardiography response, offers prognostic information that is independent from and incremental to standard WMSI.[26] However, tissue Doppler imaging is limited by angle dependency.[27] The resulting strain rate profiles also tend to be noisy, and measurements can be difficult.[17]

The 2D-STI method described in the current study overcomes these limitations. It is a novel method for estimating motion based on tissue tracking using time-domain proc-

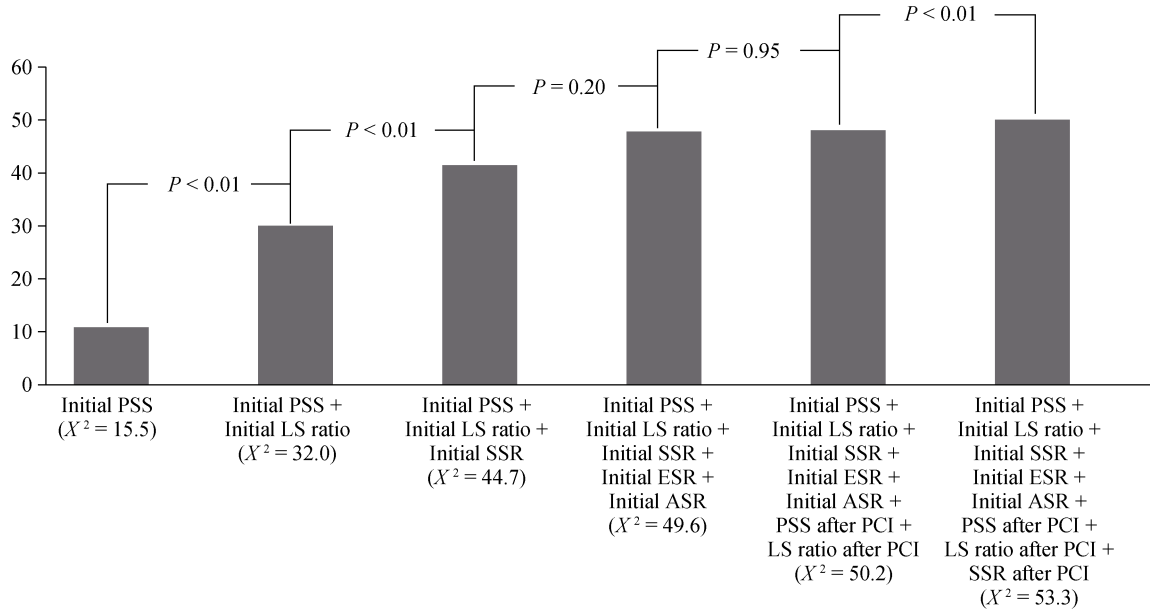

Figure 5. The incremental value of the strain and strain rate to measure myocardial viability in patients with acute myocardial infarction. ASR: late diastolic strain rate; ESR: early diastolic strain rate; LS ratio: lengthening shortening ratio; SSR: peak systolic strain rate; PCI: percutaneous coronary intervention; PSS: postsystolic strain.

essing, and measures strain independent of both cardiac translation and angle dependency.[15,28,29] The accuracy of 2D-STI was confirmed previously using sonomicrometry and MRI tagging as reference methods.[11] In a previous study, 2D-STI-derived strain measurements were useful for distinguishing infarcted from viable myocardium in a rat ischemia-reperfusion model.[30] The current study demonstrates for the first time that 2D-STI is useful for detecting myocardial viability early in patients with AMI.

Several limitations of the present study should be noted. This study was an observational study, and only a small number of patients were enrolled. The possibility of selection bias and low statistical power with residual confounding must be considered when interpreting our findings. 2D-STI should be tested largely to determine whether the SSR at 3 days after PCI by 2D-STI might add unique diagnostic information in AMI. We could not assess myocardial viability by cardiac MRI which was currently accepted as standard method. Finally, we did not assess the long term clinical outcomes.

In conclusion, 2D-STI was a feasible and quantitative method of assessing myocardial viability. In addition, SSR 3 days after PCI might be the most reliable predictor of myocardial viability in patients with acute myocardial infarction.

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