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Short-Term Prognostic Value of Strain Echocardiography in Patients with STEMI

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Objectives: Left ventricular (LV) systolic function is major predictor of outcome after acute myocardial infarction (AMI). In recent years, speckle-tracking echocardiography (STE) derived strain measurement has been demonstrated to directly reflect myocardial deformation patterns and to be superior to conventional echocardiographic measurements. Our study aimed to reveal the prognostic value of strain parameters for 30-day mortality in patients with ST elevation myocardial infarction (STEMI) after primary percutaneous coronary intervention (PCI).
Methods: We prospectively included patients with STEMI after primary PCI treatment. The primary endpoint was 30-day mortality. 2D STE was used to determine LV strain parameters.
Results: 414 patients were selected. 30-day mortality occurred in 15 patients (3.6%). LV global longitudinal strain (GLS) was significantly impaired in the 30-day mortality group compared with survivors (-8.0±2.6% vs. -15.4±3.8%, p<0.001). Multivariate regression revealed that LV GLS was independently associated with 30-day mortality (OR 1.71, 95% CI 1.23-2.38, p<0.001). Adding GLS into the clinical and echocardiographic model improved model performance.
Conclusion: The LV GLS parameter is an independent predictor of 30-day mortality in patients with STEMI after primary PCI treatment. Adding LV GLS into the prediction model improved its predictive performance.

Keywords: ST Elevation Myocardial Infarction, Prognosis, Left Ventricle

Introduction

The left ventricle (LV) is major pump in cardiovascular system, and its function is crucial to sustaining hemodynamics. It is well established that LV systolic function is a major predictor of outcome after acute myocardial infarction (AMI) [1-4]. ST elevation myocardial infarction (STEMI) is type of AMI which presents persistent ST segment elevation on an electrocardiogram [5]. During STEMI, LV function is greatly impaired because of extended ischemia caused by the

obstruction of the large epicardial vessels. Current guidelines for STEMI recommends an assessment of LV systolic function in all patients after STEMI [6, 7].

The echocardiography is a frequently used tool to assess LV structure and function. LV ejection fraction (LVEF) and wall motion score index (WMSI) are the most frequently used parameters for LV global and regional function assessment [8]. LVEF is a measurement of the percentage of blood leaving the heart each time it contracts, and the WMSI is a parameter of myocardial motion abnormality during a cardiac cycle. LVEF may be influenced by heart rate, preload, afterload, apical foreshortening, and poor endocardial border delineation [9]. Assessment of WMSI is usually experience dependent and could overestimate the amount of ischemic or infarcted myocardium because of tethering effect, disturbance of regional loading conditions, and stunning [10].

A new echocardiographic technique, strain echocardiography, has been developed to reflect true myocardial deformation not influenced by tethering effect [11]. Furthermore, strain echocardiography using a two dimensional (2D) speckle-tracking algorithm is angle-independent unlike the tissue Doppler derived measurements [12, 13]. In prior studies, 2D speckle-tracking strain echocardiography has shown good correlation with cardiac magnetic resonance imaging (MRI) to detect infarct size and microvascular obstruction and to predict LV global and regional functional recovery after AMI [14-19].

Despite therapeutic advances during 1999-2008, the 30-day mortality rate remained unchanged in STEMI patients, likely because of the more severe nature of STEMI due to the involvement of large epicardial coronary vessels. In contrast, the 30-day mortality rate of non-STEMI patients has significantly decreased during this period [20]. To reduce mortality caused by STEMI, we must work towards decreasing the 30-day mortality rate.

Previous studies have demonstrated the long-term prognostic relevance of strain echocardiography after AMI [21-29]. However, the short-term prognostic value of strain echocardiography in patients with STEMI has not been well evaluated. Therefore, our study was aimed to test the prognostic capacity of strain echocardiography for 30-day mortality in patients with STEMI.

Materials and Methods

1. Study design

In this study, we used a prospective cohort study design. A total of 414 patients with STEMI were included from the Coronary Care Unit, State Third Central Hospital between August 2015 and January 2016. The diagnosis of STEMI was made on basis of currently available STEMI guidelines, and all patients were treated with primary percutaneous coronary intervention (PCI) according to the institutional STEMI care protocol, which includes pre-hospital care, in-hospital care, and follow-up monitoring [6, 7]. All the patients were followed-up for 30-day mortality occurrence or until 30 days after index admission for AMI. During the follow-up period, we contacted the patients using mobile phones and, if necessary, called them back to the hospital for follow-up examination. The study design was approved by the review board of the Mongolian National University of Medical Sciences.

2. Clinical variables

In this study, several clinical variables, such as age, gender, admission systolic blood pressure (SBP), admission heart rate (HR), previous co-morbidities, and door-to-balloon time (D2B time), were considered risk factors for adverse events. Data about these clinical variables were collected from medical records during admission. For cardiac enzymes, peak values were collected. For cardiac troponin I (TnI), levels were considered increased if above the 99th percentile reference level [5]. Coronary angiogram was collected during primary PCI. Both initial and final angiograms were collected. Coronary flow was graded using the standard thrombolysis in myocardila infarction (TIMI) flow grade [30].

3. Echocardiography

Patients were imaged in the left lateral decubitus position using a commercially available echocardiographic machine (iE33 xMATRIX, Philips). Measurements were obtained using a 3.5 MHz transducer at a depth of 16 cm with parasternal and apical views. M-mode and 2D images were obtained during breath hold and saved in a cine-loop format from three consecutive cardiac cycles. The analysis was performed in offline mode by two experienced observers (QLAB 9, Philips).

The LV end-diastolic volume (LVEDV) and end-systolic volume (LVESV) were measured using endocardial border tracing at the end-diastolic and end-systolic phase, respectively. LVEF was calculated by using the Simpson's biplane method [8].

As recommended by American Society of Echocardiography, LV was divided into 17 segments, and each segment was analyzed individually and scored based on its motion and thickening during a cardiac cycle. 1-normokinesis, 2-hypokinesis, 3-akinesis and 4-dyskinesis were considered in the scoring of each segment, and WMSI was calculated as an average value from the sum of the segment scores divided by the number of segments scored [8].

Pulsed-wave Doppler at the mitral valve inflow was obtained by placing the Doppler sample volume between the tips of the mitral leaflets. Peak early (E) and late (A) diastolic velocities and deceleration time (DT) were measured. The Ee' ratio was calculated by dividing E by e' which was measured by color-coded tissue Doppler imaging at the basal septal segment of LV [23].

4. Strain measurement

The LV strain parameters were measured from the apical four-chamber (4CH), two-chamber (2CH), and long-axis (APLAX) views using the 2D speckle-tracking algorithm [31]. Speckle-tracking analysis was constructed to track sequential frame-to-frame movement of natural acoustic markers of myocardium from ultrasonic images in two dimensions. According to the software manual, all images were recorded at a minimum rate of 40 frames per second and saved in cine-loop format for subsequent offline analysis. Offline analysis was performed

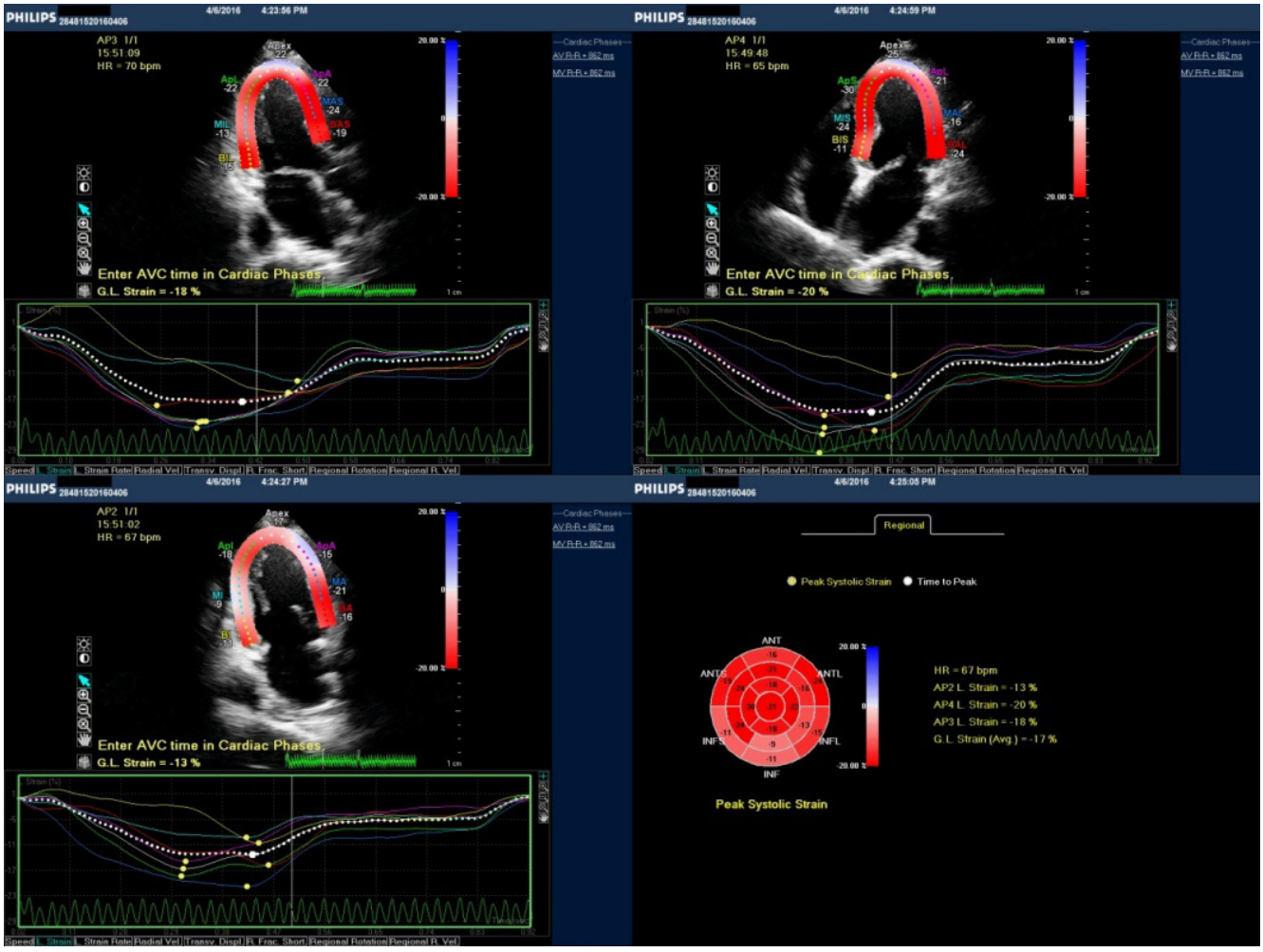

Figure 1. Measurement of GLS from the three apical views of the LV.

The infarct related artery was the right coronary artery, and the strain value was diminished in the inferior septum and the inferior segments.

using QLAB 9.0. At the beginning of the measurement, the LV endocardial border was manually traced and then the software automatically created the region of interest, which was adjusted to the thickness of myocardium but not the epicardium.

Peak strain was measured in all 17 segments from the three apical views. If tracking was poor for a particular segment, that segment was excluded from the measurement. Global longitudinal strain (GLS) of the LV was defined as the average value of the all measured segments from the three apical views (Figure 1).

5. Study endpoint

Our study endpoint was 30-day mortality after index STEMI. Data on the occurrence of our endpoint was collected from the hospital registry. 30-day mortality was defined according to the recommendation of the American College of Cardiology and the American Heart Association [32].

6. Statistical analysis

Continuous data are presented as mean±standard deviation, and categorical data are presented as frequencies and percentages. Differences in baseline characteristics between the patients who died and the survivors were evaluated using the independent sample t-test and chi-square test. Univariate logistic regression analysis was used to determine the relationship between individual variables and the study endpoint. All continuous variables were assessed per 1 unit change in each variable.

The number of co-variables was limited because of the relatively small number of endpoint occurrence. Therefore, the chi-square test was used to select independent variables from categorical variables (Table 3, supplemental), while the Pearson's correlation was used to select independent variables from continuous variables (Table 4, supplemental). The final multivariate logistic regression model consisted of age, diabetes, previous heart failure (HF), chronic kidney disease (CKD), Killip class greater than 1, cardiac arrest at admission, left anterior descending (LAD) culprit vessel, final TIMI 3 flow, HR at admission, D2B time, EA ratio, DT, LVEDV, LVEF, Ee' ratio, WMSI and GLS.

The prognostic capacity of GLS was assessed using the area under the receiver operating characteristic curve (c-statistic)

Table 3. Pearson's correlation between continuous variables.

	Age	Admission SBP	Admission HR	D2B time	Peak TnI level	EA ratio	DT	LVEDV	LVESV	LVEF	Ee' ratio	WMSI
Age	1	0.072	0.101[a]	0.119[a]	0.014	-0.291[b]	0.107[a]	-0.147[b]	-0.038	-0.113[b]	0.327[b]	0.098[a]
Admission SBP		1	0.449[b]	0.002	-0.163[b]	-0.205[b]	0.137[b]	0.053	0.035	-0.014	0.084	-0.064
Admission HR			1	0.124[a]	-0.043	-0.057	-0.125[a]	0.061	0.144[b]	-0.206[b]	0.130[b]	0.228[b]
D2B time				1	-0.160[b]	-0.064	0.069	-0.017	-0.026	0.051	0.000	0.178[b]
Peak TnI level					1	0.062	-0.319[b]	0.002	0.096[a]	-0.255[b]	0.089	0.225[b]
EA ratio						1	-0.230[b]	0.206[b]	0.155[b]	-0.035	0.151[b]	0.018
DT							1	-0.089	-0.224[b]	0.304[b]	-0.095	-0.358[b]
LVEDV								1	0.865[b]	-0.311[b]	0.071	0.300[b]
LVESV									1	-0.707[b]	0.170[b]	0.441[b]
LVEF										1	-0.201[b]	-0.419[b]
Ee' ratio											1	0.175[b]
WMSI												1

[a]Correlation is significant at <0.05 level.

[b]Correlation is significant at <0.01 level.

SBP, systolic blood pressure; HR, heart rate; D2B time, door-to-balloon time; TnI, troponin I; EA, mitral inflow peak early velocity (E)/mitral inflow peak late velocity (A); DT, deceleration time; LVEDV, left ventricular end diastolic volume; LVESV, left ventricular end systolic volume; LVEF, left ventricular ejection fraction; Ee' mitral inflow peak early velocity (E)/mitral annular peak early velocity (e'); WMSI, wall motion score index.

Table 4. Multivariate logistic regression of 30-day mortality.

Variables	Univariate regression			Mutlivariate regression		
	OR	95% CI	p-value	OR	95% CI	p-value
Age	1.05	1.01-1.10	<0.05			
Admission HR (per min)	1.00	0.98-1.03	0.652			
Diabetes	2.86	1.01-8.06	<0.05			
Previous HF	5.98	1.19-30.1	<0.05			
CKD	3.90	1.03-14.8	<0.05			
Killip class >1	3.61	1.26-10.4	<0.05			
Cardiac arrest	12.2	2.88-51.8	<0.001			
D2B time (min)	0.99	0.99-1.00	0.698			
LAD culprit vessel	5.39	1.20-24.2	<0.05			
Final TIMI 3 flow	0.09	0.03-0.27	<0.001	0.11	0.02-0.64	<0.05
EA ratio	2.62	1.24-5.51	<0.05			
DT (msec)	0.98	0.97-0.99	<0.001			
LVEDV (ml)	0.99	0.97-1.01	0.331			
LVEF (%)	0.92	0.88-0.96	<0.001			
Ee' ratio	1.15	1.08-1.22	<0.001	1.18	1.04-1.34	<0.05
WMSI	7.18	2.81-18.3	<0.001			
GLS (%)	1.90	1.50-2.41	<0.001	1.73	1.28-2.33	<0.001

HR, heart rate; HF, heart failure; CKD, chronic kidney disease; D2B time, door-to-balloon time; LAD, left anterior descending artery; TIMI, thrombolysis in myocardila infarction; EA, mitral inflow peak early velocity (E)/mitral inflow peak late velocity (A); DT, deceleration time; LVEDV, left ventricular end diastolic volume; LVEF, left ventricular ejection fraction; Ee' mitral inflow peak early velocity (E)/mitral annular peak early velocity (e'); WMSI, wall motion score index, GLS, global longitudinal strain.

[33]. The c-statistic reflects the concordance of predictions with actual outcomes in rank order, with a c-statistic of 1.0 indicating perfect discrimination.

Furthermore, a baseline clinical model, which consisted of age, admission SBP, diabetes, previous HF, CKD, peak cTnI level, LAD culprit vessel, and final TIMI 3 flow, was constructed, and incremental values of echocardiographic indices and GLS were tested following order: 1) baseline clinical model, 2) baseline clinical model + echocardiographic indices which consisted EA ratio, LVESV, LVEF, Ee' ratio and WMSI, 3) baseline clinical model + echocardiographic indices + GLS. Incremental values of model performance was tested by an estimation of model chi-square.

All statistical tests were two-sided, and p-value <0.05 was considered to be statistically significant. All statistical analyses were performed with SPSS software (version 22.0, SPSS Inc., Chicago, IL, USA).

Results

1. Baseline characteristics

414 patients with STEMI were selected for this study. The mean age was 60±13, and the majority of patients were male (n=347, 84%). Within 30-days after index STEMI, 15 patients (3.6%) had died, and the median time of death was 9 days (interquartile range [IQR] 3; 15) after hospital admission. At admission, cardiac arrest occurred in 11 patients (2.7%). Patients with 30-day mortality were older, had lower admission SBP, were more likely diabetic, had extensive myocardial damage as evaluated by troponin I (TnI) levels, often had LAD coronary artery involvement, and coronary flow grade was more likely impaired as assessed by coronary TIMI flow grade. Baseline characteristics of the study population are shown in Table 1.

2. Predictors of 30-day mortality

All the patients were followed-up until 30-day mortality occurrence or until 30 days after index admission for AMI. Patients with 30-day mortality had significantly impaired LV GLS (-8.0±2.6% vs. -15.4±3.8%, p<0.001), lower LVEF (39±13% vs. 53±12%, p<0.001) and higher WMSI (2.03±0.53 vs. 1.44±0.46, p<0.001). Diastolic dysfunction was more often present in patients with 30-day mortality as assessed by DT (136±39msec vs. 184±50msec, p<0.001) and Ee' ratio (22.6±14 vs. 12.9±5.0, p<0.001).

The results of the univariate and multivariate logistic regression analysis are shown in Table 2. Univariate logistic regression analysis showed that every 1 unit increase of GLS was associated with 1.90 times increased probability of 30-day mortality (OR 1.90, 95% CI 1.50-2.41, p<0.001).

Multivariate logistic regression analysis was revealed that GLS was independently associated with 30-day mortality (OR 1.73, 95% CI 1.28-2.33, p<0.001). Other independent predictors of 30-day mortality were final TIMI 3 flow (OR 0.11, 95% CI 0.02-0.64, p<0.05) and Ee' ratio (OR 1.18, 95%

Table 1. Baseline characteristics

Variables	All patients (n=414)	30-day mortality (n=15)	Survivors (n=399)	p-value
Age	60±13	67±11	59±13	<0.05
Male gender	347 (84%)	11 (73%)	336 (84%)	0.261
Admission SBP (mmHg)	128±37	88±48	130±36	<0.001
Admission HR (per min)	75±22	78±37	75±21	0.654
Diabetes	122 (29%)	8 (53%)	114 (28%)	<0.05
Hypertension	238 (57%)	11 (73%)	227 (57%)	0.206
Previous CAD ≥50%	51 (12%)	1 (6.7%)	50 (12%)	0.497
Previous MI	31 (7.5%)	2 (13%)	29 (7.3%)	0.311
Previous HF	12 (2.9%)	2 (13%)	10 (2.5%)	0.066
CKD	27 (6.5%)	3 (20%)	24 (6%)	0.066
Killip class >1	126 (30%)	9 (7.1%)	6 (2.1%)	<0.05
Cardiac arrest	11 (2.7%)	3 (20%)	8 (2%)	<0.01
D2B time (min)	53 (40; 79)	69 (42; 146)	53 (40; 78)	0.290
Peak TnI level (ng/ml)	57 (12; 120)	316 (70; 500)	54 (11; 115)	<0.001
Total occlusion	278 (67%)	10 (67%)	268 (67%)	0.957
LAD culprit vessel	231 (56%)	13 (87%)	218 (54%)	<0.05
Multivessel disease	261 (63%)	12 (80%)	249 (62%)	0.169
Final TIMI 3 flow	367 (88%)	7 (47%)	360 (90%)	<0.001
EA ratio	0.99±0.48	1.32±0.73	0.98±0.46	<0.01
DT (msec)	182±50	136±39	184±50	<0.001
LVEDV (ml)	85±28	78±38	86±28	0.333
LVESV (ml)	42±21	48±30	41±20	0.215
LVEF (%)	52±12	39±13	53±12	<0.001
Ee' ratio	13.3±5.9	22.6±14	12.9±5.0	<0.001
WMSI	1.46±0.47	2.03±0.53	1.44±0.46	<0.001
GLS (%)	-15.1±4.0	-8.0±2.6	-15.4±3.8	<0.001

SBP, systolic blood pressure; HR, heart rate; CAD, coronary artery disease; MI, myocardial infarction; HF, heart failure; CKD, chronic kidney disease; D2B time, door-to-balloon time; TnI, troponin I; LAD, left anterior descending artery; TIMI, thrombolysis in myocardila infarction; EA, mitral inflow peak early velocity (E)/mitral inflow peak late velocity (A); DT, deceleration time; LVEDV, left ventricular end diastolic volume; LVESV, left ventricular end systolic volume; LVEF, left ventricular ejection fraction; Ee' mitral inflow peak early velocity (E)/mitral annular peak early velocity (e'); WMSI, wall motion score index, GLS, global longitudinal strain.

Table 2. Chi-square estimation between categorical variables.

Variables	30-day mortality (n=15)	Survivors (n=399)	p-value[a]
Male gender	11 (73%)	336 (84%)	0.261
Diabetes	8 (53%)	114 (28%)	<0.05
Hypertension	11 (73%)	227 (57%)	0.206
Previous CAD ≥50%	1 (6.7%)	50 (12%)	0.497
Previous MI	2 (13%)	29 (7.3%)	0.311
Previous HF	2 (13%)	10 (2.5%)	0.066
CKD	3 (20%)	24 (6%)	0.066
Killip class >1	9 (7.1%)	6 (2.1%)	<0.05
Cardiac arrest	3 (20%)	8 (2%)	<0.01
Total occlusion	10 (67%)	268 (67%)	0.957
LAD culprit vessel	13 (87%)	218 (54%)	<0.05
Multivessel disease	12 (80%)	249 (62%)	0.169
Final TIMI 3 flow	7 (47%)	360 (90%)	<0.001

[a]Chi-square test

CAD, coronary artery disease; MI, myocardial infarction; HF, heart failure; CKD, chronic kidney disease; LAD, left anterior descending artery; TIMI, thrombolysis in myocardila infarction.

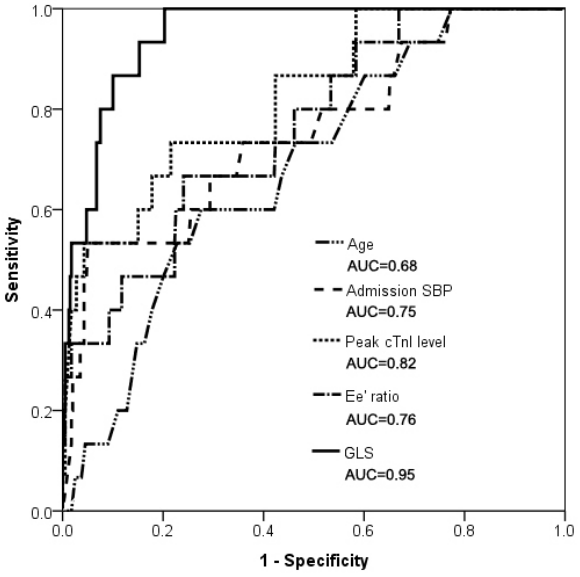

Figure 2. ROC curve estimation of predictive capacity.
GLS parameter (solid line) showed better predictive capacity (AUC=0.95) compared with other measurements.

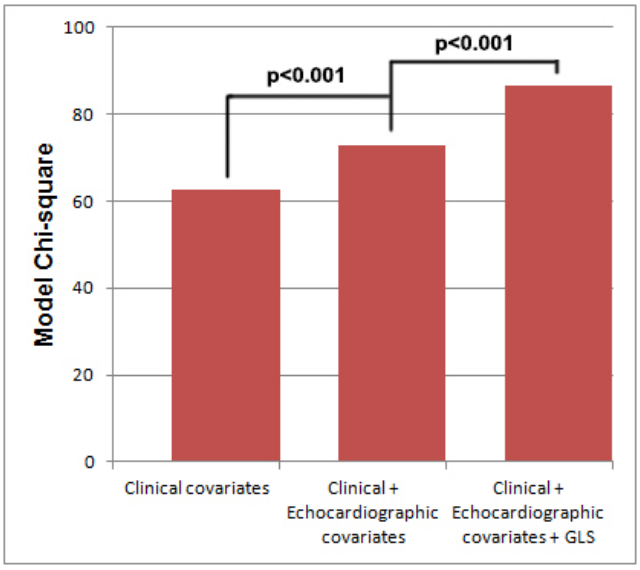

Figure 3. Incremental improvement in model performance.
Addition of echocardiographic measurements (EA ratio, LVESV, LVEF, Ee' ratio and WMSI) significantly improved the model which also included clinical variables (age, admission SBP, diabetes, previous HF, CKD, Peak cTnI level, LAD culprit vessel, final TIMI 3 flow). Furthermore, adding GLS into the combined clinical and echocardiographic model yielded significantly better model performance.

CI 1.04-1.34, p<0.05). Furthermore, the c-statistics of GLS outperformed all other independent predictors (0.95, 95% CI 0.91-0.98, p<0.001) for 30-day mortality (Figure 2).

Incremental value of GLS was evaluated using three modeling steps, as described in Figure 3. Adding GLS into the combined clinical and echocardiographic model significantly increased the model chi-square (p<0.001).

Discussion

The main findings of our study can be summarized as follows: 1) LV GLS was a significant independent predictor of 30-day mortality after STEMI, 2) the predictive capacity of GLS outperformed previously determined risk factors, 3) adding GLS into the currently available risk prediction model yielded better predictive capacity.

The speckle-tracking derived LV strain parameter directly reflects myocardial stretching and contraction patterns throughout a cardiac cycle. All other conventional echocardiographic measurements are based on an indirect measurement of myocardial contraction, such as of geometrical changes or myocardial movement during a cardiac cycle. Therefore, speckle-tracking derived LV GLS may be a more accurate indicator of myocardial function.

In this study, 30-day mortality occurred in 15 patients (3.6%), which is comparable with recent registry studies with primary PCI as the choice method of reperfusion [34]. Despite dramatically improved outcomes of AMI, which is a consequence of early reperfusion therapy, the prognosis of short-term patients is worse in STEMI compared with NSTEMI [35, 36]. Therefore, early risk stratification is crucial managing patients with STEMI.

The previous studies have demonstrated possible predictors of 30-day mortality after AMI [37]. Risk score based prediction models, such as TIMI and GRACE risk score, are frequently used in daily clinical practice [38, 39]. The advantage of a risk score model is that the calculation is derived from simple clinical factors and readily usable at the bedside. Based on a patient's risk score, clinicians can make appropriate decisions about need for transfer to a tertiary care center, level of care, and use of treatment strategies such as initial thrombolysis or early invasive strategy.

In the last decade, the preferred treatment strategy of STEMI has dramatically changed into direct transfer to PCI capable hospital and early primary PCI, regardless of baseline condition. Therefore, the influence of risk score on treatment strategy has been limited.

In our study, we tested the prognostic value of GLS for the prediction of 30-day mortality after STEMI. Recent clinical trials have combined traditional clinical variables, angiographic measurements, and echocardiographic measurements for the prediction model of mortality in AMI. Therefore, we included the following in our in multivariate logistic regression: traditional clinical variables, such as age, Killip class, blood pressure, HR, elevated cardiac enzymes, and prior co-morbodities; coronary angiography results such as degree of stenosis, culprit vessel, and final TIMI flow; and routine echocardiographic parameters such as LVEF, WMSI, and Doppler indices. Our multivariate logistic regression analysis demonstrated that GLS is an independent predictor of 30-day mortality after STEMI. A ROC curve analysis showed that the prognostic capacity of GLS is better than of traditional clinical variables and echocardiographic measurements.

The long-term prognostic value of LV strain measurement is well studied. Several studies have determined that 2D speckle-tracking derived LV strain parameters provides valuable prediction of subsequent cardiac events and outcome in patients with AMI who have been treated by primary PCI [25, 26]. However, data about the early prognostic value of LV strain parameter is lacking. Saito et al. have demonstrated that LV GLS is an independent and incremental parameter to predict readmission and 30-day mortality of patients during first admission of HF [40]. However, a major limitation of their findings was that the etiology of HF was not only caused by AMI but also by other etiologies of HF. Meimoun et al. have revealed an independent correlation between LV strain measurements and in-hospital cardiac events after acute anterior MI [41]. They have also demonstrated a relationship between LV strain parameters and LV functional recovery after AMI. All their results were consistent with previous studies and the findings of our study [42].

A limitation of our study was the relatively small number of deaths that occurred during follow-up. This may have weakened the statistical power of our study. Therefore, a further study with a larger number of patients and long term follow-up is needed to confirm the above mentioned results. Furthermore,

our sample was collected from a single center, which created the possibility for patient selection bias. Thus, subsequent studies should use multi-center study design to prevent patient selection bias.

The findings of this study identified that the LV GLS parameter is a strong and independent predictor of 30-day mortality in patients with STEMI who were treated by primary PCI. Adding GLS to the risk prediction model improved the model's predictive performance. Based on our findings, we recommend further studies to clarify whether the measurement of LV GLS during early stage of STEMI improves the risk stratification compared with currently available score based models such as TIMI and GRACE score. Further studies should particularly investigate whether adding LV GLS to traditional clinical predictive model can improve the early identification of high risk patients among those with STEMI.

Conflict of Interest

The authors state no conflict of interest.

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