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Relationship Between Left Ventricular Myocardial Longitudinal Mechanics and In-hospital Heart Failure in Patients With Acute Myocardial Infarction: a Two-dimensional Speckle-tracking Study

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Objectives: In-hospital heart failure (HF) during acute myocardial infarction (AMI) is associated with adverse outcome. However, data about the relationship between left ventricular (LV) longitudinal myocardial mechanics and in-hospital HF in patients with AMI is limited. Thus, we aimed to determine the association between speckle-tracking derived global longitudinal strain (GLS) and in-hospital HF. **Methods:** We selected patients with AMI treated by primary percutaneous coronary intervention (PCI). In-hospital HF was defined by Killip class. Multiple logistic regression analysis was used to reveal the relationship between LV GLS and in-hospital HF. **Results:** A total of 414 patients (mean age 60 ±13 years, 84% male) were included and in-hospital HF presented in 93 patients (22.5%). LV GLS was significantly impaired in patients with in-hospital HF compared to patients without (-16.1 ±3.7% vs. -11.6 ±3.1%, p <0.001). After adjustment of possible predictors, GLS was independently associated with in-hospital HF (odds ratio 1.32, 95% CI: 1.16-1.50, p <0.001). In-hospital HF presented in 21 patients with preserved left ventricular ejection fraction (LVEF) and GLS was also significantly impaired (-17.7 ±3.2% vs. -12.7 ±2.2%, p <0.001). Separate multiple logistic regression models showed that GLS was still independently associated with in-hospital HF in this group. **Conclusion:** LV GLS is independently associated with in-hospital HF during AMI. This relationship is still evident for patients with preserved LVEF.

Keywords: Myocardial Infarction, Left Ventricular Function, Heart Failure, Prognosis

Introduction

In-hospital heart failure (HF) remains a significant predictor of short- and long-term prognosis of patients with acute myocardial infarction (AMI) [1, 2]. In-hospital HF during AMI is usually caused by significant loss of functioning myocardium and associated with subsequent adverse cardiac events [3]. However, it also can occur in patients who had minor loss of myocardial and normal or mildly reduced left ventricular ejection fraction (LVEF), and is also associated with increased risk of adverse outcomes [4]. Inconsistency between normal LVEF and clinical symptoms of HF is mainly explained by diastolic dysfunction because those patients are more likely to have co-morbid conditions such as, diabetes and hypertension, and these have been associated with diastolic dysfunction [5, 6]. However, current studies reported that patients with diastolic dysfunction did not always have clinical symptoms of HF and those patients had relatively normal LV global longitudinal strain (GLS) compared with patients who had HF-preserved LVEF (HFpEF) [7].

Deformation analysis using two-dimensional speckle-tracking is new way to assess LV longitudinal fiber shortening, which may be expressed as a global longitudinal strain (GLS). The longitudinal fibers in the subendocardial layer are more sensitive to ischemia and wall stress and can exhibit abnormal contraction patterns in the setting of apparently normal LVEF [8]. Furthermore, recent data suggest that patients with HFpEF are characterized by abnormal global LV longitudinal deformation [9, 10]. However, evidences of clinical significance of LV longitudinal function assessment in patients with AMI complicated by in-hospital HF, particularly in patient populations who had normal LVEF, is limited. In this study, we evaluated the clinical significance of left ventricular global longitudinal function assessment in patients who presented in-hospital HFpEF after AMI.

Materials and Methods

1. Study population

A total of 414 patients presenting with AMI treated with primary percutaneous coronary intervention (PCI) at the Third State Central Hospital between August 2015 and January 2016 were included in this study. Diagnosis of AMI was made on the basis of currently available guidelines [11, 12]. All patients were treated according to the institutional AMI care protocol, which includes pre-hospital, in-hospital care and follow-up monitoring. Patients who had paced heart rhythm and poor image quality for speckle-tracking analysis were excluded from the final patient population.

2. Clinical variables

Several clinical variables, such as age, gender, smoking status and previous co-morbidities, were considered as risk factors for adverse events in this study. Data about these clinical variables were collected from the patient's medical record during admission. For cardiac enzymes, peak values were collected. For cardiac troponin I (TnI), above the 99th percentile reference level was considered increased [13]. Coronary angiogram was collected during primary PCI. Both an initial and final angiogram was collected. Coronary flow was graded by the use of the standard thrombolysis in myocardial infarction (TIMI) flow grade [14].

3. Echocardiography

Patients were imaged in the left lateral decubitus position using a commercially available echocardiographic machine (iE33 xMATRIX, Philips, Netherlands). Measurements were obtained by using a 3.5 MHz transducer (X5-1 xMATRIX Array, Philips) at a depth of 16 cm in the parasternal and apical views. M-mode and 2D images were obtained during a breath hold and saved in cine-loop format from three consecutive cardiac cycles. 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 by using endocardial border tracing at the end-diastolic and end-systolic phase, respectively. LVEF was calculated by using Simpson's biplane method [15]. As recommended by the American Society of Echocardiography, LV was divided into 17 segments and each segment was analyzed individually and scored based on its motion and thickening. The scoring of the segment was as follows: 1 = normokinesis, 2 = hypokinesis, 3 = akinesis and 4 = dyskinesis [15]. WMSI was calculated as the average value of the sum of the segment scores divided by the number of segments scored [15]. Pulsed-wave Doppler of 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 E/E′ ratio was calculated by dividing E by E′, which was measured by color-coded tissue Doppler imaging at the basal septal segment.

4. Strain and strain rate measurement

Strain and strain rate parameters were measured from apical four-chamber (4CH), two-chamber (2CH) and long-axis (APLAX) views by using 2D speckle-tracking analysis [16]. Speckle-tracking analysis is 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 least at a rate of 40 frames per second and saved in cine-loop format for subsequent offline analysis. Offline analysis was performed by using the issue motion quantification algorithm of the QLAB 9 software. At the beginning of the measurement, the LV endocardial border was manually traced and then the software automatically created a region of interest that was adjusted to the thickness of the myocardium but not covered epicardium. Systolic and peak strain was measured in all 17 segments of the three apical views. If the segment tracking was poor, this segment was excluded from measurement. The GLS value of LV was calculated as the average value of the peak systolic longitudinal strain in 17 segments that were taken from three apical views.

5. Study endpoint

The study endpoint was in-hospital HF after AMI. Data about occurrence of the endpoint was collected from the patient's medical chart and objective signs were evaluated and symptoms of HF which were defined according to the recommendation of the American College of Cardiology (ACC) and the American Heart Association (AHA) [17]. After that, each patient was graded according to the highest Killip class by using the Killip classification scheme [11, 12].

6. Statistical analysis

Continuous data were presented as the mean ±standard deviation (SD) and categorical data were presented as frequencies and percentages. Differences in baseline characteristics between patients with in-hospital HF and without in-hospital HF were evaluated by using the independent sample t-test and chi-squared test. Continuous variables that were not normally distributed (as evaluated by Kolmogorov–Smirnov tests) were presented as medians and 25th and 75th percentiles and were compared using the Wilcoxon's rank-sum test.

Univariate logistic regression analysis was performed to determine the relationship between individual parameters and the study endpoint. All continuous variables were assessed per 1 unit change in each variable.

Multivariate logistic regression analysis was performed to assess the independent association between GLS and in-hospital HF. Multivariate logistic regression analysis consisted of age, gender, smoking status, diabetes, hypertension, stable angina, previous coronary artery disease (CAD), previous myocardial infarction (MI), previous HF, chronic kidney disease (CKD), admission TnI level, left anterior descending coronary artery (LAD) culprit vessel, multivessel disease, final TIMI 3 flow, mitral inflow peak early velocity/mitral inflow peak late velocity (EA ratio), DT, LVEDV, LVESV, LVEF, mitral inflow peak early velocity/mitral annular peak early velocity (EE' ratio), wall motion score index (WMSI) and GLS.

Separate multivariate logistic regression analysis was performed to assess the independent relationship between GLS and in-hospital HF in patients who had preserved LVEFs (LVEF ≥55%). However, there were a relatively small number of patients in the preserved LVEF group. Therefore, we constructed a model which had a reduced number of covariates such as, age, diabetes, hypertension, admission TnI, LAD culprit vessel, multivessel disease, final TIMI flow, EE' ratio, and WMSI. Then GLS were separately added into that model and their independent association with in-hospital HF was tested. Preserved left ventricular function was defined as LVEF ≥55%. Therefore, LVEF was excluded from that separate multivariate regression model to prevent co-linearity.

Furthermore, a separate baseline clinical model which consisted of age, gender, smoking status, diabetes, hypertension, stable angina, previous CAD, previous MI, previous HF, CKD, admission TnI level, LAD culprit vessel, multivessel disease, and final TIMI flow was constructed and the incremental value of echocardiagraphic indices, WMSI, LVEF and GLS were tested in the following sequence: (1) baseline clinical model, (2) baseline clinical model + echocardiographic indices which consisted of the EA ratio, DT, LVEDV, LVESV and EE' ratio, (3) baseline clinical model + echocardiographic indices + WMSI, (4) baseline clinical model + echocardiographic indices + WMSI + LVEF, (5) baseline clinical model + echocardiographic indices + WMSI + LVEF + GLS. The incremental value of model performance was tested by estimation of the model chi-squared. Also receiver-operating characteristic curve analysis was used to evaluate model c-statistics.

Finally, 20% of all patients were randomly selected to test the intra- and inter-observer variability for strain measurements

by Bland–Altman analysis. All statistical tests were two-sided, and a p-value of <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

A total of 414 patients were included in this study after using exclusion criteria. The mean age was 60 ±13 years and the majority of the patients were men (84%, n = 347). During hospital admission, HF occurred in 93 patients (22%) as defined by Killip class >1. Of patients with in-hospital HF, 43 were in Killip class II, 21 were in Killip class III and 29 were in Killip class IV. Patients with in-hospital HF were older, more likely to be hypertensive, their release of TnI was higher, they often had LAD culprit artery and final TIMI 3 flow was not achieved more than patients without in-hospital HF. The baseline characteristics of the patient population are described in Table 1.

Table 1. Baseline characteristics

Variables	With in-hospital HF (n = 93) (n (%))	Without in-hospital HF (n = 321) (n (%))	p-value
Age (years)[a]	63 ±12	59 ±13	<0.01
Male	17 (18)	50 (16)	0.533
Current smoker	40 (43)	156 (48)	0.342
Diabetes	27 (29)	95 (29)	0.917
Hypertension	62 (67)	176 (55)	<0.05
Stable angina	6 (6)	8 (2)	0.063
Previous CAD ≥50%	17 (18)	34 (11)	<0.05
Previous MI	7 (7)	22 (7)	0.823
Previous HF	5 (5)	7 (2)	0.106
CKD	9 (10)	18 (6)	0.162
Admission TnI level (μg/L)[b]	94 (7; 218)	54 (13; 104)	<0.01
LAD culprit vessel	66 (71)	165 (51)	<0.001
Total occlusion	66 (71)	212 (66)	0.373
Multivessel disease	63 (68)	198 (62)	0.302
Final TIMI 3 flow	74 (79)	293 (91)	<0.01
EA ratio[b]	0.9 (0.6; 1.2)	0.8 (0.7; 1.2)	0.966
DT (ms)[b]	157 (126; 190)	182 (155; 219)	<0.001
LVEDV (mL)[a]	94 ±32	83 ±26	<0.001
LVESV (mL)[a]	53 ±26	38 ±18	<0.001
LVEF (%)[a]	45 ±13	54 ±11	<0.001
EE' ratio[b]	13 (11; 18)	11 (9; 14)	<0.001
WMSI[b]	1.8 (1.3; 2.2)	1.2 (1.0; 1.6)	<0.001
GLS (%)[a]	-11.6 ±3.1	-16.1 ±3.7	<0.001

[a]Mean ±SD [b]Median (25th percentile; 75th percentile)

2. Echocardiographic variables associated with in-hospital HF

Patients who presented in-hospital HF had significantly lower LVEF (45 ±13% vs. 54 ±11%, p <0.001) and higher WMSI (1.8 vs. 1.2, p <0.001). Also diastolic dysfunction was more common in patients with in-hospital HF; they had a higher EE' ratio (13 vs. 11, p <0.001) and lower DT (157 ms vs. 182 ms, p <0.001, Table 1).

Left ventricular longitudinal function was significantly impaired in patients with in-hospital HF compared to patients without in-hospital HF (-11.6 ±3.1% vs. -16.1 ±3.7%, p <0.001). The GLS was significantly increasing along through Killip class such as, from Killip class 2 to 3 (-13.2 ±2.8% vs. -11.0 ±2.8%, p <0.05) and from Killip class 2 to 4 (-13.2 ±2.8% vs. -9.7 ±2.6%, p <0.001), but there was no difference between Killip class 3 and 4 (-11 ±2.8% vs. -9.7 ±2.6%, p = 0.199) in multiple comparisons with the least significant difference post hoc test.

Multivariate logistic regression analyses with odds ratio (OR) and 95% confidence intervals (CI) are shown in Table 2. Univariate logistic regression analysis showed that every 1 unit increase of GLS was associated with a 1.4 times increased probability of in-hospital HF (OR: 1.43, 95% CI: 1.31-1.55, p <0.001). Based on multivariate logistic regression analysis, GLS was an independent predictor of in-hospital HF (Table 2).

Table 2. Multivariate logistic regression of in-hospital HF

Variables	Univariate regression			Multivariate regression		
	OR	95% CI	p-value	OR	95% CI	p-value
Age	1.03	1.01-1.05	<0.005	1.02	0.99-1.05	0.103
Male	1.21	0.66-2.22	0.534	1.06	0.46-2.48	0.886
Current smoker	0.80	0.50-1.27	0.342	1.18	0.61-2.29	0.623
Diabetes	0.97	0.58-1.62	0.917	0.77	0.39-1.53	0.452
Hypertension	1.65	1.02-2.67	<0.05	1.90	0.99-3.66	0.054
Stable angina	2.70	0.91-7.98	0.073	2.50	0.42-14.9	0.314
Previous CAD ≥50%	1.88	1.00-3.56	0.050	1.94	0.50-7.53	0.339
Previous MI	1.11	0.46-2.68	0.823	0.37	0.07-1.92	0.237
Previous HF	2.55	0.79-8.23	0.118	2.94	0.50-17.3	0.233
CKD	1.80	0.78-4.16	0.167	0.83	0.27-2.54	0.751
Admission TnI level	1.00	1.00-1.01	<0.001	1.00	1.00-1.01	0.094
LAD culprit vessel	2.31	1.40-3.80	<0.005	1.29	0.61-2.75	0.504
Total occlusion	1.26	0.76-2.08	0.374	0.85	0.43-1.67	0.630
Multivessel disease	1.29	0.79-2.11	0.302	0.84	0.45-1.58	0.592
Final TIMI flow	0.37	0.20-0.70	<0.005	0.87	0.36-2.07	0.749
EA ratio	1.39	0.88-2.18	0.152	1.66	0.90-3.04	0.102
DT	0.99	0.98-0.99	<0.001	1.00	0.99-1.01	0.290
LVEDV	1.01	1.01-1.02	<0.005	0.98	0.94-1.03	0.518
LVESV	1.03	1.02-1.04	<0.001	1.03	0.96-1.11	0.430
LVEF	0.93	0.91-0.95	<0.001	1.01	0.94-1.08	0.813
EE' ratio	1.06	1.03-1.11	<0.005	0.97	0.92-1.02	0.234
WMSI	8.33	4.81-14.4	<0.001	2.30	0.97-5.46	0.059
GLS	1.43	1.31-1.55	<0.001	1.32	1.16-1.50	<0.001

3. GLS in patients with preserved ejection fraction

A total of 181 patients (43.7%) had preserved left ventricular function (mean age 58 ±12 years, 81% male) and 21 of them (11.6%) experienced in-hospital HF. GLS was significantly impaired in patients with in-hospital HFpEF compared to HF with a reduced EF (HFrEF) (-12.7 ±2.2% vs. -17.7 ±3.2%, p <0.001). Differences of clinical variables are described in Table 3. Multivariate logistic regression analysis including age, diabetes, hypertension, admission TnI, LAD culprit vessel, multivessel disease, final TIMI flow, EE' ratio and WMSI showed that GLS was significantly associated with in-hospital HF in patients who had preserved ejection fraction (Table 4). In this analysis, GLS showed highest significance within independent predictors based on the Wald chi-squared test ($\chi^2 = 7.85$, p <0.001).

4. Incremental value of GLS

The incremental value of GLS was assessed by using five modeling steps which are described in Figure 1. Adding GLS to the model that included baseline clinical variables, echocardiographic indices, WMSI and LVEF produced an association with a significantly increased model chi-squared ($\chi^2 = 133$, p <0.001). Furthermore, receiver-operating characteristic curve analysis showed that the c-statistic value of GLS was significantly higher than LVEF (0.83, 95% CI: 0.78-0.87 vs. 0.70, 95% CI: 64-76, p

Table 3. Clinical characteristics of patients with HFpEF and patients with HFrEF

Variables	Patients with HFpEF (n = 21) (n (%))	Patients with HFrEF (n = 160) (n (%))	p-value
Age (years)[a]	62 ±14	57 ±12	0.077
Male	14 (67)	132 (82)	0.084
Current smoker	9 (43)	77 (48)	0.649
Diabetes	5 (24)	43 (27)	0.765
Hypertension	18 (86)	80 (50)	<0.01
Stable angina	2 (9)	6 (4)	0.226
Previous CAD ≥50%	4 (19)	16 (10)	0.214
Previous MI	1 (5)	8 (5)	0.962
Previous HF	1 (5)	3 (2)	0.397
CKD	2 (9)	5 (3)	0.153
Admission TnI level (μg/L)[b]	126 (4; 298)	27 (4; 67)	<0.05
LAD culprit vessel	16 (76)	78 (49)	<0.05
Total occlusion	15 (71)	90 (57)	0.195
Multivessel disease	14 (67)	90 (57)	0.380
Final TIMI 3 flow	16 (76)	174 (92)	<0.001
EA ratio[b]	0.7 (0.6; 1.1)	0.8 (0.7; 1.2)	0.065
DT (ms)[b]	182 (136; 212)	189 (162; 228)	0.246
LVEDV (mL)[a]	72 ±19	77 ±22	0.332
LVESV (mL)[a]	28 ±9	29 ±9	0.648
LVEF (%)[a]	61 ±5	63 ±6	0.226
EE' ratio[b]	14 (11; 18)	11 (9; 13)	<0.05
WMSI[b]	1.8 (1.3; 2.1)	1.1 (1.0; 1.4)	<0.001
GLS (%)[a]	-12.7 ±2.2	-17.7 ±3.2	<0.001

[a]Mean ±SD [b]Median (25th percentile; 75th percentile)

Table 4. Multivariate logistic regression

Variables	Wald χ^2	OR	95% CI	p-value
Age	0.11	0.99	0.92-1.06	0.744
Diabetes	2.67	0.20	0.03-1.37	0.102
Hypertension	7.03	17.7	2.12-148	<0.01
Admission TnI level	3.95	1.01	1.00-1.01	<0.05
LAD culprit vessel	0.68	2.24	0.33-15.1	0.408
Multivessel disease	0.02	1.12	0.26-4.72	0.876
Final TIMI flow	1.43	0.40	0.09-1.80	0.232
EE' ratio	0.004	1.00	0.85-1.19	0.952
WMSI	4.89	8.54	1.28-57.1	<0.05
GLS	7.85	1.68	1.17-2.41	<0.001

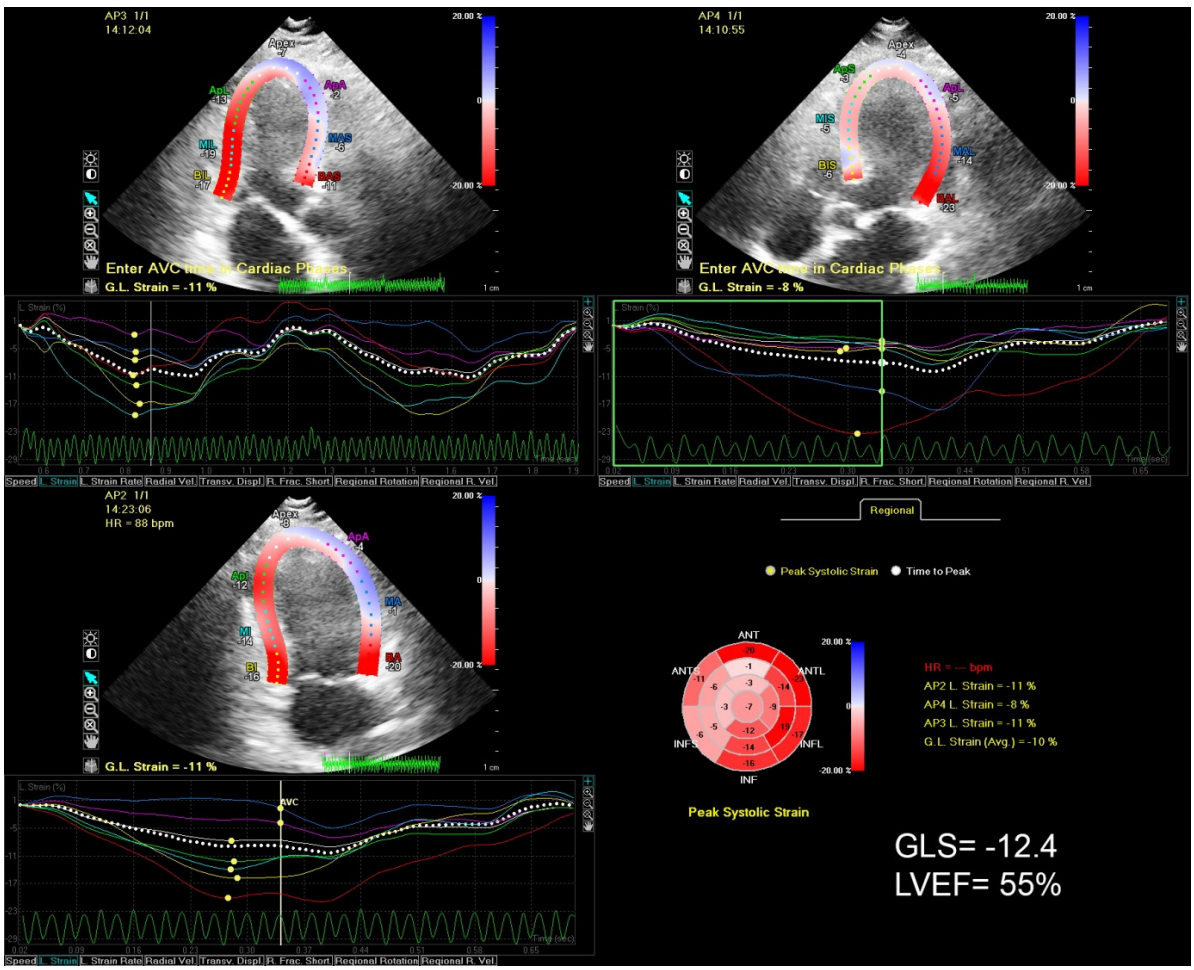

Figure 1. Example of left ventricular GLS in patients with coronary TIMI 2 flow. LVEF was preserved but GLS was severely impaired.

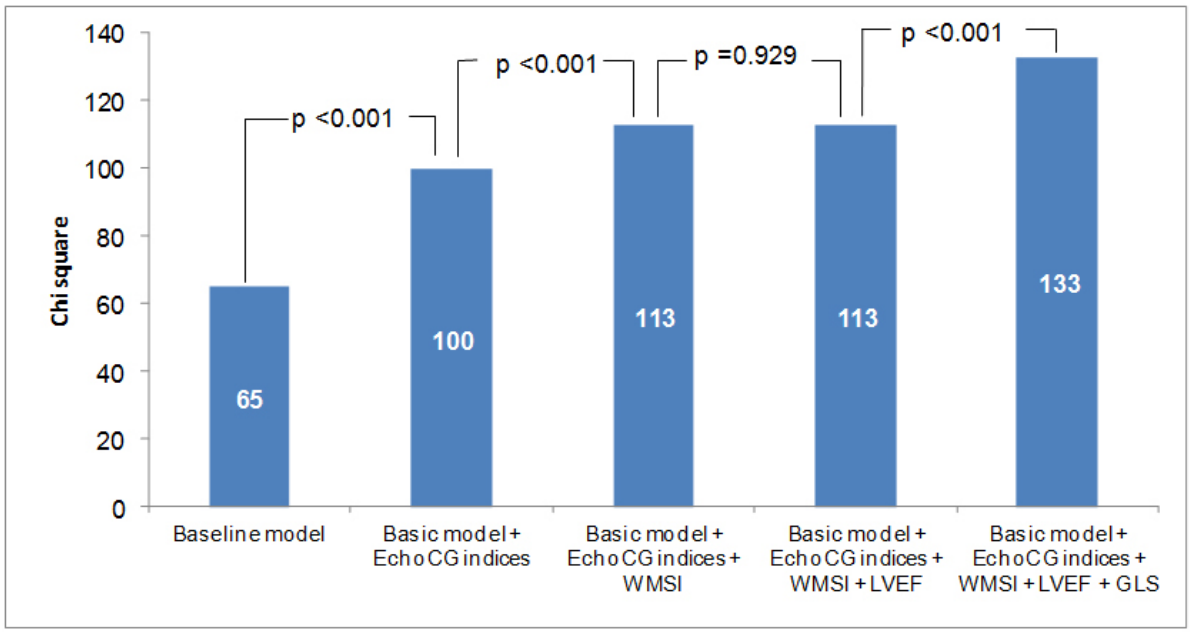

Figure 2. Incremental value of GLS in model performance assessed by chi-square estimation.

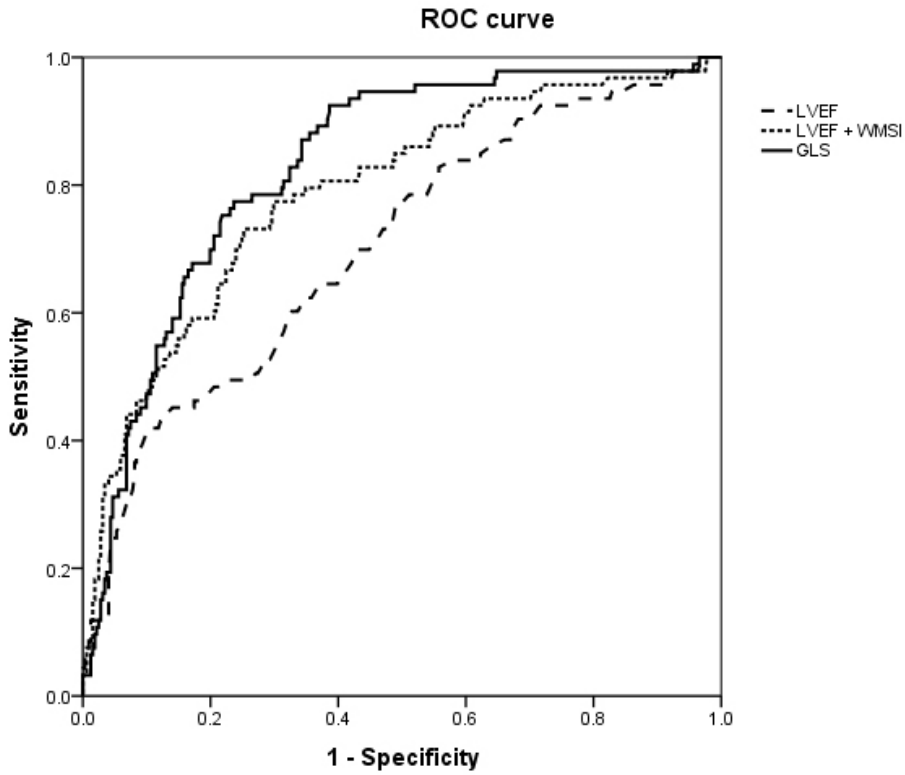

Figure 3. Receiver-operating characteristic curve analysis depicting model performance to predict in-hospital HF.

<0.001). Adding WMSI to LVEF increased the model c-statistic, although its c-statistic was still lower than GLS (0.83, 95% CI: 0.78-0.87 vs. 0.79, 95% CI: 0.73-0.84, p <0.001, Figure 2).

5. Intra and inter-observer variability

Bland-Altman analysis demonstrated a good intra- and inter-observer agreement with a non-significant small difference for the GLS parameter. The mean difference for global strain was

-0.59% (95% CI: -9.55 to 8.36, p = 0.309) for intra-observer agreement and 1.27% (95% CI: -9.50 to 11.94, p = 0.072) for inter-observer agreement.

Discussion

The major findings of this study can be summarized as follows: (1) after adjusting previously-determined clinical and routine echocardiographic predictors, speckle-tracking derived GLS is significantly and independently associated in-hospital HF after AMI; (2) association between GLS and in-hospital HF is still evident in patients who had preserved LVEF.

Two dimensional speckle-tracking (2D STE) derived strain measurement is a novel echocardiographic parameter which directly indicates myocardial global and longitudinal contractile function. It can overcome limitations of traditional tissue Doppler imaging such as angle dependency and tethering effect [18]. Previous studies demonstrated that GLS has good correlation with findings of cardiac magnetic resonance (CMR) to detect infarct size, microvascular obstruction and LV global and regional functional recovery after AMI [19-23].

Previously, the relationship between clinical HF and normal LVEF was mainly explained by diastolic dysfunction and impaired filling [6]. However, recent studies demonstrated that LV longitudinal deformation was impaired in patients with HFpEF but not in patients with diastolic dysfunction [7].

Cardiac muscle consists of inner and outer longitudinal fibers and middle circumferential fibers [24]. By the Frank-Starling law, forceful synchronized contraction of longitudinal and circumferential fibers increases elastic energy in the cardiomyocyte and interstitium. During diastole, elastic energy is released from the cardiomyocyte and interstitium and causes rapid filling at the early diastole. Therefore, impaired longitudinal fiber shortening decreases synchronized cardiac contraction and decreases elastic energy. This is a possible explanation of why GLS out-performed tissue Doppler parameters such as EE' ratio and DT in multivariate logistic regression analysis.

Previous co-morbid conditions such as diabetes, hypertension, previous CAD, previous MI and previous HF were causes of decreased LV longitudinal function [7, 25-27]. Therefore, the effect of overall co-morbid conditions to in-hospital HF could be implicated by GLS. According to the precise correlation between LV longitudinal function and infarct size, GLS could be a superior predictor of in-hospital HF compared with peak TnI level [20-23]. Estimation of LVEF and WMSI were based on the endocardial border definition and myocardial thickness and there is a collinear correlation between GLS and those parameters [15]. All these associations suggested that GLS could be a superior predictor of in-hospital HF. Therefore, we performed multivariate logistic regression analysis which adjusted for the above-mentioned variables and revealed that GLS is the only significant and independent variable which is associated with in-hospital HF. This association was also evident in patients who had normal LVEF.

GLS may implicate information about acute myocardial injury and previous co-morbid conditions which were associated with impaired LV longitudinal function. Therefore, we hypothesized that GLS may have an incremental value on prediction of in-hospital HF. We tested the incremental value of GLS by using four modeling steps which was comprised of baseline clinical variables, routine echocardiographic indices, LVEF and WMSI and adding GLS into this model was associated with increased chi-squared value. Additional receiver-operating characteristic analysis showed that GLS had the greatest c-statistic value compared with both LVEF and WMSI as well as in combination. All these data confirmed an independent relationship between GLS and in-hospital HF and the incremental value of GLS.

In this study, we did not distinguish between patients with ST segment elevation myocardial infarction (STEMI) and non-STEMI (NSTEMI). It could be a significant limitation of the current study because STEMI is a transmural infarction caused by total occlusion of the epicardial vessel while NSTEMI is a subendocardial infarction caused by partial occlusion of epicardial vessel. Also, patients with AMI in Mongolia were more likely referred to delayed primary PCI. During acute ischemia, however, the most vulnerable part of the myocardium is the inner longitudinal fiber which has a major role in LV longitudinal function and it could be impaired in both STEMI and NSTEMI.

Quantitative assessment of LV systolic function in the early stage of AMI is crucial in prognostic evaluation. Currently available echocardiographic measurements are not accurate to estimate HFpEF. During AMI, the most vulnerable part of myocardium is the subendocardial longitudinal fibers. Assessment of LV longitudinal fiber shortening by using deformation analysis may have strong diagnostic and predictive capacity of diagnosing HF. Modern revascularization strategies significantly decreased loss

of viable myocardium and the patient population with preserved LVEF after AMI is continuously increasing. Therefore, GLS could be important to reveal those patients with preserved LVEF at the high risk of subsequent adverse events. Furthermore, the long-term prognostic relevance of GLS should be assessed in patients with AMI. Left ventricular GLS is independently associated with in-hospital HF during AMI. This relationship is still evident for group of patients who had preserved LVEF.

Conflict of Interest

The authors state no conflict of interest.

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