VIA MEDICA

ORIGINAL ARTICLE

Cardiology Journal
2021, Vol. 28, No. 5, 678–689
DOI: 10.5603/CJ.a2020.0010

ISSN 1897–5593
eISSN 1898–018X

The prognostic value of left atrial and left ventricular strain in patients after ST-segment elevation myocardial infarction treated with primary percutaneous coronary intervention

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Abstract

Background: *Global longitudinal strain (GLS) based on two-dimensional speckle-tracking echocardiography (2D-STE) might better reflect left ventricular (LV) contractile performance than conventional parameters. Recently, left atrial (LA) strain has been used as a more accurate alternative to assessing LA performance. The aim in this study was to assess the clinical prognostic value of left ventricular GLS (LV GLS) and peak atrial longitudinal strain (PALS) in patients after ST-segment elevation myocardial infarction (STEMI).*

Methods: *The study enrolled 199 patients who underwent primary percutaneous coronary intervention (pPCI) for first STEMI. Conventional and 2D-STE were performed within 48 h after pPCI. LV GLS and PALS were related to LV remodeling at 6-month follow-up and to adverse events.*

Results: *Diabetes mellitus, GLS and PALS independently predicted LV remodeling. With multivariable Cox proportional hazards, diabetes mellitus, GLS and PALS were predictive of adverse clinical outcomes. However, PALS did not add significant incremental value beyond LV GLS in the prediction of LV remodeling (increase in area under the receiver-operator characteristic curve [AUC]: 0.05, $p = 0.24$) and clinical events (even a decrease in AUC: 0.03, $p = 0.69$).*

Conclusions: *Both GLS and PALS provide independent prognostic value for adverse LV remodeling and clinical outcomes after STEMI. However, the ability of the combination of PALS and GLS to predict LV remodeling and clinical outcomes may not be superior to that of a single indicator.* (Cardiol J 2021; 28, 5: 678–689)

Key words: acute myocardial infarction, atrial strain, global longitudinal strain, echocardiography, remodeling, prognosis

Introduction

It is well known that outcomes of ST-segment elevation myocardial infarction (STEMI) have dramatically improved in recent years because of the introduction of modern thrombolytic drugs and percutaneous coronary intervention (PCI). However, left ventricular (LV) remodeling still occurs in 30–35% of patients [1, 2]. There is a progressive change in myocardial wall and ventricular structure, including expansion in the infarct region, wall thinning, and ventricular dilation in the non-infarcted region [3], which may be followed by adverse cardiovascular events and an increase mortality rate [4]. The introduction of two-dimensional speckle-tracking echocardiography (2D-STE) may

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Received: 1.08.2019 Accepted: 20.01.2020 Early publication date: 5.02.2020

contribute to quantification of LV global and regional systolic function [5]. Previous studies have shown that global longitudinal strain (GLS) can be used to predict LV remodeling and cardiovascular events after STEMI [6–9]. However, some studies showed that like GLS, global circumferential strain (GCS) and circumferential strain rate are independent predictors of LV remodeling [10].

Left atrial (LA) volumes and LA function have been recognized as significant predictors of adverse events in a range of cardiovascular diseases [11, 12]. Recently, 2D-STE is shown to be feasible for measuring LA deformations, thus allowing analysis of LA reservoir function (peak atrial longitudinal strain [PALS]) during the LV systolic phase [13]. More recently, LA reservoir function measured by PALS has shown good predictive value, even independently of LV GLS and LA volume [14, 15]. However, the additional value of PALS in patients with decreased LV GLS is questionable. A previous study proved that the prognostic value of PALS in patients with acute myocardial infarction (AMI) is dependent on LV GLS and LA size [16].

Accordingly, the purpose of this study was to examine patients with STEMI in: the clinical and prognostic importance of both LV GLS and PALS on LV remodeling and clinical outcome and prognostic information incremental of PALS to clinical data as well as reduced LV GLS.

Methods

Study population

In this prospective study, a total of 216 patients diagnosed with STEMI treated with primary PCI (pPCI) were enrolled from September 2017 to March 2018. The inclusion criteria were as follows: age 18 to 80 years, STEMI with onset of pain < 12 h before pPCI, and admission with STEMI based on present guidelines [17]. The exclusion criteria were: previous myocardial infarction or coronary artery bypass, significant valvular dysfunction, ventricular arrhythmia, atrial fibrillation or paced rhythm, and noncardiac disease with a life expectancy of < 1 year.

All patients were treated according to present cardiology guidelines. Before pPCI, they were given a loading dose of acetylsalicylic acid (ASA), 600 mg of clopidogrel, and 100 IU/kg of heparin (maximum 5,000 IU). This prospective study was approved by the Ethics Committee of the First Hospital of Lanzhou University. All patients signed informed consent forms.

Echocardiography

Echocardiographic data were obtained using the EPIQ 7C (Kininklijke Philips NV, Eindhoven, The Netherlands). Echocardiographic images were obtained by recording three consecutive heart cycles during apnea according to the guidelines of the American Society of Echocardiography [5]. Two experienced observers performed all patient views offline using an echocardiographic analysis system (QLAB Advanced Tissue Motion Quantification, Phillips).

Left ventricular end-systolic volume (LVESV), LV end-diastolic volume (LVEDV) and LV ejection fraction (LVEF) were determined using the biplane Simpson method in 4-, 3-, and 2-chamber views. The LV was divided into 16 segments, and segments were graded (1 = normokinetic, 2 = hypokinetic, 3 = akinetic, 4 = dyskinetic) according to subjective assessments of wall motion amplitude and changes in LV thickness at systole. The wall motion score index (WMSI) was defined as the sum of the segment score ratings divided by the number of segments scored. Pulsed-wave Doppler variables were measured by placing at the tip of the mitral valve (MV) leaflets from the apical 4-chamber view during diastole. The peak velocity of early (E) and late (A) diastole and the MV deceleration time were measured, and the E/A ratio was calculated. The measurements of myocardial peak early velocity (e') were performed at the lateral and medial mitral annulus. E/e' were obtained by dividing E by e'.

LV strain analysis

Two-dimensional echocardiographic images were obtained from 4-, 3-, and 2-chamber and midventricular short-axis views with frame rates of 60 to 90 frame/s. 2D-STE was performed using the commercially available software QLAB Advanced Tissue Motion Quantification (Philips) equipped with STE analysis. The LV endocardial and epicardial borders were initially traced at end-diastole, and the software automatically tracked the region of interest of the myocardium. Longitudinal peak systolic strain (LPSS), was obtained for each segment from which the software provided strain curves in all 16 segments. The GLS was calculated as the average of the observed segmental values of LPSS from the apical 4-, 3-, and 2-chamber view (Fig. 1A). For LV circumferential peak systolic strain and radial peak systolic, 2D-STE analyses were performed on the LV short-axis midventricular view. Global circumferential strain and global radial strain were calculated as the mean of values from LV short-axis views.

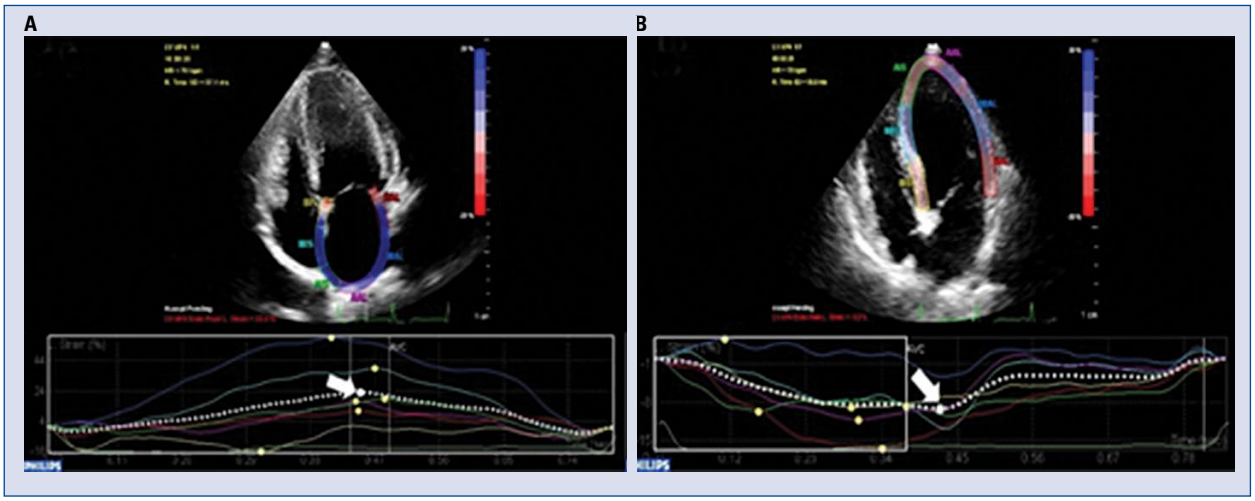

Figure 1. Two-dimensional speckle-tracking of the left ventricle (LV). The resulting strain curves for LV are shown with markings corresponding to peak global longitudinal strain (**A**); the resulting strain curves for left atrium are shown with markings corresponding to peak atrial longitudinal strain (**B**).

LA function analysis

The biplane Simpson method was used to analyze LA function. LA volume at LV end-systole (LAVmax), LA volume at LV end-diastole (LAVmin), and LA volume before atrial active contraction at the onset of the P-wave (LAVpreA) were obtained from apical 4- and 2-chamber views. All LA volumes were indexed to the body surface area [5]. From these volumes, the indexes of LA mechanical function were calculated: (1) total atrial emptying fraction: LA total ejection fraction = ((LAVmax – LAVmin) / LAVmax) × 100; (2) active atrial emptying fraction-an index of LA active contraction: LA active ejection fraction = = ((LAVpreA – LAVmin) / LAVpreA) × 100; (3) passive atrial emptying fraction-an index of LA conduit function: LA passive ejection fraction = ((LAVmax – LAVpreA) / LAVmax) × 100; (4) atrial expansion index of reservoir function: LA expansion index = = (LAVmax – LAVmin) / LAVmin × 100 [18].

For 2D-STE analysis of LA function, 2D gray-scale images were obtained in apical 4- and 2-chamber views, consistent with software and version for analyzing LV strain. To measure PALS (LA reservoir function), the beginning of QRS wave of the electrocardiogram was used as a reference point [13]. After selecting the cardiac cycle, the LA endocardial border was manually traced, automatically creating a region of interest to cover the thickness of LA myocardium from a total of 12 atrial segments (Fig. 1B). PALS values were estimated in each LA segment from two apical views, and the mean of global PALS was calculated. Patients in whom more than two segments with poor images could not be analyzed were excluded [2].

Follow-up and endpoint definition

At least 6 months after STEMI (18.3 ± 5.0 months), conventional echocardiography was performed. LV remodeling assessed by echocardiography was defined as an LVEDV increase of > 20% compared with baseline echocardiographic data [2]. Cardiovascular medical professionals completed follow-up phone calls in all patients each month after discharge from the hospital. Major adverse clinical events were a composite of death from any cause, hospitalization for heart failure and reinfarction, which were determined by both clinical visits and telephone calls. Hospitalization for heart failure occurring because of exacerbation of exertional dyspnea, with typical symptoms of pulmonary congestion and initiation of intravenous diuretics. Reinfarction was defined as a typical sign of chest pain, elevated cardiac enzyme levels, and obvious changes on the electrocardiogram [19].

Statistical analysis

Data for continuous variables are presented as the mean ± standard deviation or median and interquartile range, and categorical variables are presented as frequencies and percentages. Continuous variables are compared using the

independent-samples t test. Categorical variables were compared by the χ^2 test. To examine determinants of LV remodeling as a dependent variable, logistic forward regression analysis was applied. Univariate analysis was performed to choose the independent variables, and those variables with borderline values ($p < 0.10$) were submitted for multivariate analysis. The ability of clinical and echocardiographic parameters to predict adverse events were tested in univariate Cox proportional hazards models. To estimate the independent prognostic value of the above parameters, multivariate Cox proportional hazards analysis was also performed. Receiver operating characteristic (ROC) curve analysis were constructed, and areas under curves (AUC) were measured to determine cutoff values with maximum sensitivity and specificity. All statistical tests were two-sided, and a p value < 0.05 was considered statistically significant.

Results

Subject characteristics

A total of 216 patients with their first acute STEMI treated with pPCI were initially evaluated. Seventeen patients were excluded: before echocardiographic examination, 2 (0.9%) patients died during hospitalization, and 5 (2.3%) patients were not available to undergo echocardiography due to poor cooperation. Another 10 (4.6%) patients did not have sufficient image quality for tracking of the LV and LA walls. No patients were lost to follow-up. Thus, 199 patients were enrolled in the present study. Mean age was 57.4 ± 10.7 years, and 150 were males.

Prediction of LV remodeling at 6 months

At 6-month follow-up, the incidence of adverse LV remodeling was 25%. The baseline characteristics and echocardiographic parameters of both the LV remodeling group and the non-LV remodeling group are summarized in Table 1. Except for diabetes mellitus, the incidence of risk factors associated with cardiovascular disease did not differ significantly between the two groups. Anterior wall STEMI appeared in 106 (52%) patients and was the most common (76%) kind of adverse LV remodeling. After immediate pPCI therapy, a comparison of echocardiographic data showed larger LVEDV, LVESV and LA volume index (LAVI); lower LVEF, LA total ejection fraction, LA active emptying fraction and LA reservoir function and higher WMSI were observed in the LV remodeling group. There were significant reductions in both LV GLS and GCS, as well as in PALS, regardless of myocardial infarction location.

Univariate analysis demonstrated the variables to be correlated to the LV remodeling, namely diabetes mellitus, creatinine kinase-MB, LAVI, LA total ejection fraction, LA active emptying fraction, LA reservoir function, PALS, WMSI, GLS and GCS. Therefore, these parameters were included in a forward stepwise multivariate analysis, and diabetes mellitus, GLS and PALS were demonstrated to independently predict LV remodeling (Table 2).

The AUC for LV GLS and PALS were 0.86 and 0.89, respectively. However, PALS did not add significant incremental value beyond LV GLS (AUC increased from 0.86 to 0.91; $p = 0.24$) in the prediction of LV adverse remodeling. The best cutoff values of LV GLS and PALS for LV remodeling were –11.3% (sensitivity: 71.4%, specificity: 84.0%) and 28.9% (sensitivity: 72.7%, specificity: 87.8%) (Fig 2A–C).

Clinical events during follow-up

During a mean follow-up of 18.3 ± 5.0 months, 23 (11.6%) patients reached one or more composite endpoints: 3 (1.5%) patients died, 9 (4.5%) patients had reinfarction, and 11 (5.5%) patients required hospital admission to control heart failure symptoms, who were in the event group; the other 176 patients were divided into the event-free group. Comparison of clinical and echocardiographic features between patients who achieved the composite endpoint and those who did not are displayed in Table 3.

Diabetes mellitus, LAVI, LA total ejection fraction, LA active emptying fraction, LA reservoir function, PALS, LVEF, LV GLS and GCS were univariable predictors of adverse events. All these parameters were included in a multivariate Cox proportional hazards model, and diabetes mellitus, LV GLS and PALS were independently associated with the composite events (Table 4)

The AUC for LV GLS and PALS were 0.86 and 0.83, respectively. Similarly, PALS did not add significant incremental value beyond LV GLS (AUC decreased from 0.86 to 0.83; $p = 0.69$) in the prediction of the composite event. The best cutoff values of LV GLS and PALS for LV remodeling were –12.3% (sensitivity: 95.7%, specificity: 67.0%) and 28.9% (sensitivity: 88.1%, specificity: 65.2%) (Fig. 3A–C).

Figure 4A, B showed survival curves by the Kaplan-Meier analysis for patients divided by the best value of LV GLS and PALS: patients with LV

Table 1. Baseline characteristics of patients with and without left ventricular remodeling.

Parameter	Non-remodeling (n = 150)	Remodeling (n = 49)	P
Clinical parameters			
Number	150 (75%)	49 (32%)	
Male	73%	75.5%	0.76
Age [years]	57.9 ± 10.5	55.9 ± 11.0	0.26
BMI [kg/m^2]	24.5 ± 3.7	24.7 ± 3.9	0.75
Diabetes	18 (12.0%)	19 (38.8%)	0.001
Hypertension	65 (43.3%)	18 (36.7%)	0.42
Hyperlipidemia	53 (35.3%)	18 (36.7%)	0.86
Smoking	102 (68%)	32 (65.3%)	0.73
Systolic BP [mmHg]	111.2 ± 16.9	110.5 ± 17.3	0.82
Diastolic BP [mmHg]	73.1 ± 14.8	69.1 ± 9.6	0.07
Heart rate [bpm]	74.4 ± 15.3	74.5 ± 7.4	0.95
QRS width [ms]	97.9 ± 16.4	102.2 ± 21.1	0.14
S-TO-B [min]	328.0 ± 174.4	383.9 ± 175.6	0.053
D-TO-B [min]	49.1 ± 19.1	53.2 ± 21.2	0.20
eGFR [mL/min/1.73 m^2]	92.1 ± 27.3	99.0 ± 28.8	0.13
Creatinine [μmol/L]	71.9 ± 26.3	67.6 ± 11.5	0.19
Grace (scores)	95.8 ± 26.7	98.7 ± 22.6	0.49
Crusade (scores)	22.7 ± 13.2	19.9 ± 11.8	0.26
CK-MB [ng/mL]	332.2 ± 143.4	436.2 ± 117.9	0.001
CK-MB peak time after onset [h]	15.5 ± 5.1	19.4 ± 5.2	0.001
Killip class ≥ II	14 (9.3%)	6 (12%)	0.56
Anterior wall MI	66 (44.0%)	37 (75.5%)	0.001
ST max before PCI [mm]	3.8 ± 2.0	4.5 ± 2.4	0.07
Multivessel coronary disease	42 (28%)	20 (41%)	0.09
Medication during hospitalization			
ASA	150 (100%)	49 (100%)	1
Clopidogrel/Ticagrelor	150 (100%)	49 (100%)	1
Beta-blockers	113 (75%)	35 (71%)	0.59
ACEI/ARB	89 (59%)	29 (59%)	0.99
Statins	135 (90%)	45 (92%)	0.70
Initial LV function			
LVESV [mL]	86.9 ± 21.6	104.4 ± 28.7	0.001
LVEDV [mL]	41.3 ± 13.2	56.6 ± 17.6	0.001
LVEF [%]	52.9 ± 4.5	46.3 ± 3.8	0.001
WMSI	1.31 ± 0.1	1.37 ± 0.1	0.001
Deceleration time [ms]	171.3 ± 39.2	159.0 ± 53.4	0.09
E/A ratio	0.9 ± 0.3	0.9 ± 0.5	0.47
E/E′	11.8 ± 3.1	12.5 ± 3.8	0.21
Moderate or severe MR	6 (4%)	4 (8%)	0.06
GLS [%]	–14.7 ± 2.9	–10.6 ± 2.4	0.001
GCS [%]	–14.5 ± 3.5	–12.7 ± 2.9	0.001
GRS [%]	39.1 ± 8.6	38.7 ± 7.8	0.75
LA function			
LAVI [mL/m^2]	26.8 ± 5.0	32.8 ± 7.5	0.001
LA total ejection fraction [%]	54.9 ± 6.0	52.4 ± 5.4	0.01

→

Table 1 (cont.). Baseline characteristics of patients with and without left ventricular remodeling.

Parameter	Non-remodeling (n = 150)	Remodeling (n = 49)	P
LA passive emptying fraction [%]	28.3 ± 8.1	28.2 ± 5.6	0.95
LA active emptying fraction [%]	36.9 ± 6.6	33.7 ± 4.9	0.002
LA reservoir function [%]	125.7 ± 31.2	112.8 ± 25.7	0.01
PALS [%]	32.5 ± 5.9	23.0 ± 4.8	0.001
Follow-up LV function			
LVESV [mL]	88.8 ± 23.1	131.2 ± 35.1	0.001
LVEDV [mL]	39.1 ± 15.3	74.2 ± 23.4	0.001
LVEF [%]	56.5 ± 5.8	43.9 ± 3.9	0.001
Composite endpoint during follow-up			
Total number of complications	9 (6.0%)	14 (29%)	0.001

Data are expressed as mean ± standard deviation or number (%). ACEI — angiotensin-converting enzyme inhibitors; ARB — angiotensin II receptor blocker; ASA — acetylsalicylic acid; BMI — body mass index; BP — blood pressure; CK — creatine kinase; D-TO-B — door-to-balloon time; E/A — mitral inflow peak early velocity/mitral inflow peak late velocity; E/E′ — mitral inflow peak early velocity/mitral annular peak early velocity; eGFR — estimated glomerular filtration rate; GCS — global circumferential strain; GLS — global longitudinal strain; GRS — global radial strain; LA — left atrium; LAVI — left atrium volume index; LV — left ventricular; LVEDV — left ventricular end-diastolic volume; LVEF — left ventricular ejection fraction; LVESV — left ventricular end-systolic volume; MI — myocardial infarction; MR — mitral regurgitation; PALS — peak atrial longitudinal strain; ST max — maximum ST-segment elevation from a single lead; S-TO-B — symptom-to-balloon time; WMSI — wall motion score index

Table 2. Factors predicting adverse left ventricular remodeling after 6-month follow-up in univariate and multivariate analysis.

Parameters	OR	95% CI	P
Univariate analysis			
Diabetes	4.64	2.18–9.90	0.001
CK-MB [ng/mL]	1.01	1.0–1.01	0.001
LA function			
LA max [mL/m^2]	1.18	1.11–1.26	0.001
LA total ejection fraction [%]	0.93	0.88–0.98	0.01
LA active emptying fraction [%]	0.92	0.87–0.97	0.003
LA reservoir function [%]	0.98	0.97–0.99	0.01
PALS [%]	0.71	0.64–0.79	0.001
LV function			
WMSI	10.70	1.95–58.82	0.006
GLS [%]	1.81	1.50–2.18	0.001
GCS [%]	1.21	1.06–1.37	0.004
Multivariate analysis			
Diabetes	4.93	1.63–14.87	0.005
PALS [%]	0.77	0.68–0.87	0.003
GLS [%]	1.36	1.11–1.67	0.001

CI — confidence interval; CK — creatine kinase; GCS — global circumferential strain; GLS — global longitudinal strain; LA — left atrium; LV — left ventricular; OR — odds ratio; PALS — peak atrial longitudinal strain; WMSI — wall motion score index

GLS > –12.3% (log-rank χ^2 = 37.3, p = 0.001) and PALS < 23.8% (log-rank χ^2 = 47.0, p = 0.001), and had composite event rates of 3% and 4%, respectively.

Discussion

The major results of this study showed the prognostic value of LV GLS and PALS measured

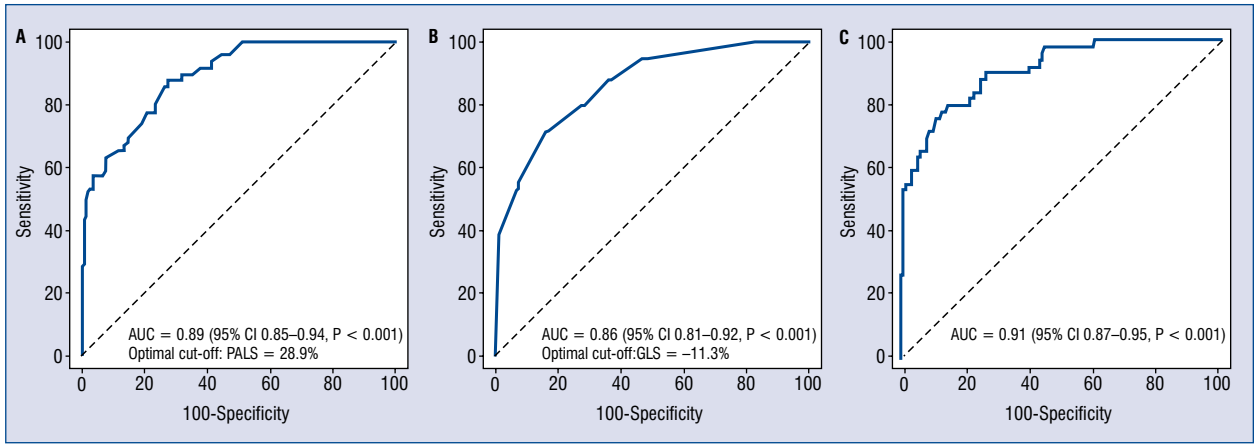

Figure 2. Receiver operating-characteristic curve for prediction of left ventricular remodeling 6 months after acute myocardial infarction using the independent variable peak atrial longitudinal strain (PALS) (**A**), left ventricular global longitudinal strain (LV GLS) (**B**) and PALS combined with GLS (**C**); AUC — area under curve.

Table 3. Baseline characteristics of patients, event and event-free.

Parameter	Event-free	Event	P
Clinical parameters			
Male	72%	87%	0.21
Age [years]	57.4± 10.5	57.7 ± 11.4	0.90
BMI [kg/m^2]	24.4 ± 3.5	25.3 ± 5.0	0.25
Hypertension	74 (42.0%)	9 (39.1%)	0.83
Hyperlipidemia	59 (35.5%)	12 (52.2%)	0.10
Smoking	116 (65.9%)	18 (78.2%)	0.34
Systolic BP [mmHg]	110.4 ± 16.3	115.6 ± 21.3	0.17
Diastolic BP [mmHg]	72.0 ± 13.7	73.1 ± 14.2	0.71
Heart rate [bpm]	74.6 ± 14.1	73.0 ± 11.3	0.61
QRS width [ms]	97.4 ± 15.9	110.9 ± 25.2	0.001
S-TO-B [min]	334.5 ± 176.4	397.2 ± 162.3	0.11
D-TO-B [min]	48.9 ± 19.5	56.1 ± 19.0	0.10
eGFR [mL/min/1.73 m^2]	93.5 ± 26.2	96.2 ± 38.3	0.66
Creatinine [μmol/L]	71.4 ± 24.7	66.6 ± 11.9	0.36
Grace (scores)	96.6 ± 25.4	95.2 ± 28.9	0.79
Crusade (scores)	21.8 ± 12.9	22.7 ± 12.4	0.78
Killip class ≥ II	14 (8.0%)	6 (26.1%)	0.007
Anterior wall MI	83 (47.2%)	20 (87.0%)	0.001
CK-MB [ng/mL]	347.4 ± 146.5	437.2 ± 98.5	0.005
CK-MB peak time after onset [h]	16.2 ± 5.3	18.4 ± 5.1	0.06
ST max before PCI [mm]	4.0 ± 2.1	3.7 ± 1.9	0.59
Multivessel coronary disease	54 (30%)	8 (35%)	0.81
LA function			
LA max [mL/m^2]	27.7 ± 5.8	33.1 ± 7.5	0.001
LA total ejection fraction [%]	54.7 ± 5.9	50.7 ± 5.3	0.002
LA passive emptying fraction [%]	28.6 ± 7.6	25.6 ± 7.9	0.07
LA active emptying fraction [%]	36.4 ± 6.5	33.6 ± 4.4	0.04

→

Table 3 (cont.). Baseline characteristics of patients, event and event-free.

Parameter	Event-free	Event	P
LA reservoir function [%]	124.8 ± 30.6	105.8 ± 22.4	0.003
Moderate or severe MR	8 (5%)	2 (9%)	0.07
PALS [%]	31.1 ± 5.9	22.7 ± 5.7	0.001
Initial LV function			
LVESV [mL]	88.3 ± 21.7	113.5 ± 34.0	0.001
LVEDV [mL]	42.9 ± 13.4	61.5 ± 17.6	0.001
LVEF [%]	51.9 ± 5.1	46.6 ± 4.0	0.002
GLS [%]	–14.1 ± 3.1	–10.2 ± 1.9	0.001
GCS [%]	–14.2±3.3	–12.6 ± 3.5	0.03

Data are expressed as mean ± standard deviation or number (%). BMI — body mass index; BP — blood pressure; CK — creatine kinase; D-TO-B — door-to-balloon time; eGFR — estimated glomerular filtration rate; GCS — global circumferential strain; GLS — global longitudinal strain; LA — left atrium; LV — left ventricular; LVEDV — left ventricular end-diastolic volume; LVEF — left ventricular ejection fraction; LVESV — left ventricular end-systolic volume; MI — myocardial infarction; MR — mitral regurgitation; PALS — peak atrial longitudinal strain; PCI — percutaneous coronary intervention; ST max — maximum ST-segment elevation from a single lead; S-TO-B — symptom-to-balloon time

Table 4. Factors predicting adverse events according to Cox proportional hazards regression model using univariable and multivariate analysis.

Parameters	HR	95% CI	P
Univariate analysis			
Diabetes	4.96	2.18–11.2	0.001
CK-MB [ng/mL]	1.01	1.00–1.01	0.007
LA max [mL/m^2]	1.14	1.08–1.20	0.001
LA total ejection fraction [%]	0.90	0.84–0.96	0.01
LA active emptying fraction [%]	0.93	0.87–0.99	0.04
LA reservoir function [%]	0.97	0.96–0.99	0.01
PALS [%]	0.82	0.76–0.88	0.001
LVEF [%]	0.82	0.76–0.89	0.001
GLS [%]	1.55	1.31–1.83	0.001
GCS (%]	1.08	1.01–1.16	0.02
Multivariate analysis			
PALS [%]	0.88	0.78–0.99	0.04
GLS [%]	1.30	1.01–1.66	0.03
Diabetes	4.61	1.50–14.19	0.008

CI — confidence interval; CK — creatine kinase; GCS — global circumferential strain; GLS — global longitudinal strain; HR — hazard ratio; LA — left atrium; LV — left ventricular ejection fraction; PALS — peak atrial longitudinal strain

by 2D-STE in patients with STEMI after pPCI, as follows: (1) reductions in PALS and LV GLS are both strongly correlated to LV remodeling and the composite event; (2) however, PALS does not add significant incremental prognostic value to LV GLS.

Acute myocardial infarction is characterized by regional myocardial damage that results in systolic and diastolic dysfunction with a risk of adverse LV remodeling. For several decades, previous researchers have focused on the pathophysiology and prognosis of LV systolic dysfunction after AMI and have shown that LV remodeling mostly occurs in cases of transmural infarction and if at least 20% of LV mass is destroyed [3]. Although LVEF and WMSI have traditionally been used to evaluate the degree of myocardium injury and even WMSI is considered an independent predictor of LV remodeling [20, 21], either of them has

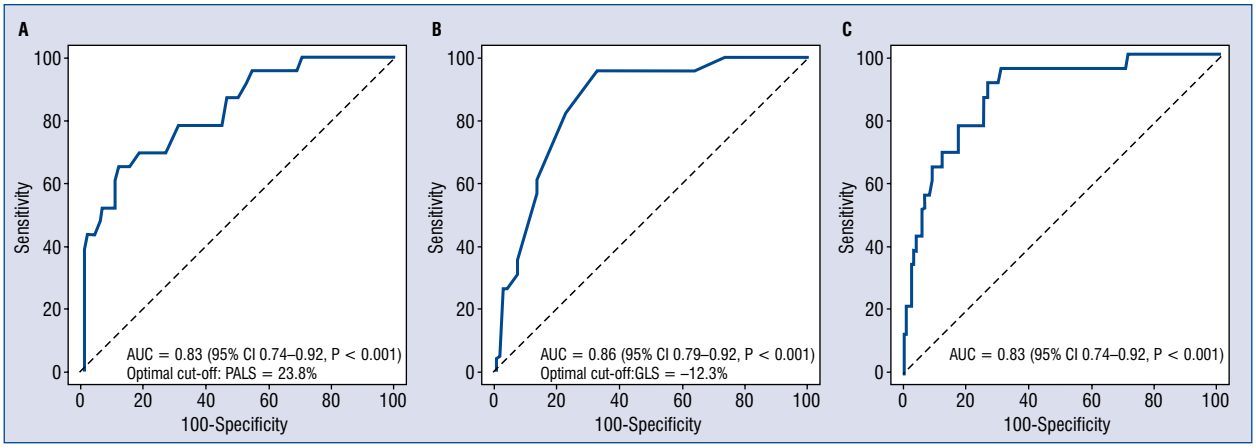

Figure 3. Receiver operating-characteristic curve for prediction of clinical adverse events using the peak atrial longitudinal strain (PALS) (**A**), left ventricular global longitudinal strain (LV GLS) (**B**) and PALS combined with GLS (**C**).

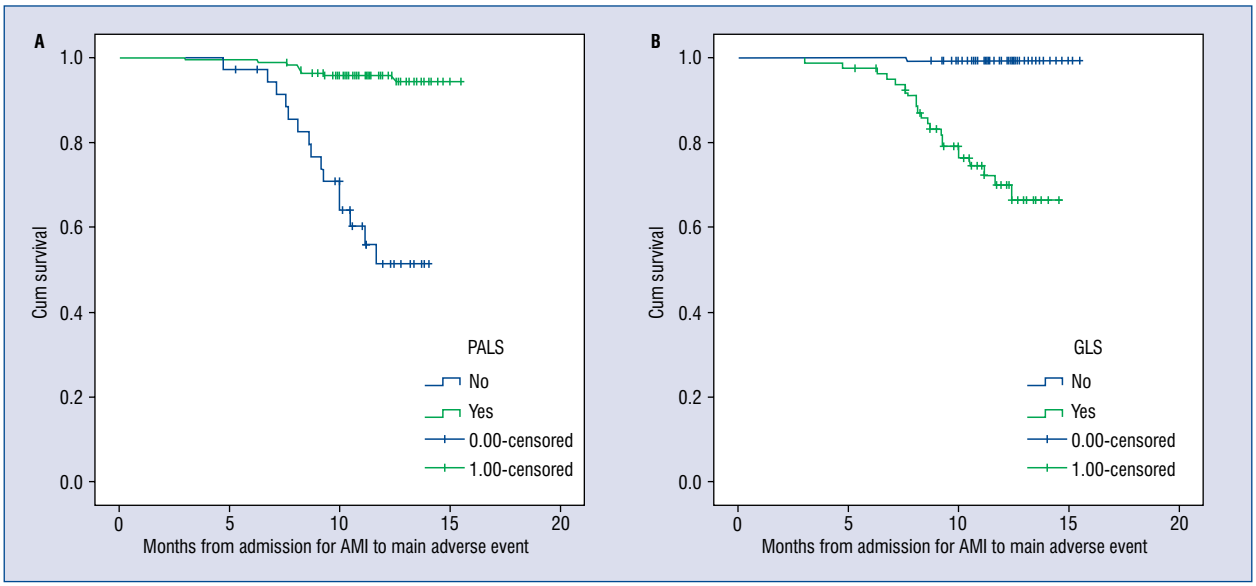

Figure 4. Survival analysis according to peak atrial longitudinal strain (PALS) and global longitudinal strain (GLS) values Kaplan-Meier survival curves for patients according to PALS (the optimal cutoff 23.8%) (**A**) and left ventricular GLS (the optimal cutoff –12.3%) (**B**); AMI — acute myocardial infarction.

limitations for risk stratification after AMI [22]. 2D-STE, as a semiautomatic method, is not only applied to estimate the motion of the myocyte but also can distinguish the passive and active motility of LV segments, suggesting it is a more sensitive measurement of LV function [23]. The present results showed that LV GLS not LVEF and WMSI is an independent predictor of LV remodeling, and the AUC was 0.86, and the best cutoff value was –11.3%, which is similar to the –12.46% reported by Lacalzada et al. [24]. This may be because strain can better distinguish between passive and active motion of each segment of LV, and hence GLS appears to be more useful than LVEF and WMSI in predicting LV remodeling. Hung et al. [10] found that not only GLS but also GCS and circumferential strain rate are independent predictors of LV remodeling at 20 months after adjusting for clinical variables. It seems that circumferential function plays an essential role in maintaining LV structure, so circumferential dysfunction would lead to LV dilatation. In the current study, GCS

was not an independent predictor by multivariate analysis. The reason for the contradictory data in predicting LV remodeling by GCS may be the different follow-up periods after AMI.

Park et al. [7] demonstrated that not only GLS showed good predictive value for LV remodeling in patients with anterior wall AMI but also predicted death or heart failure as composite events, indicating that GLS was also a good predictor of adverse clinical events. A previous study confirmed that LV strain and strain rate were superior to LVEF and WMSI in risk stratification for long-term outcome, and a GLS value > –15.1% was an independent predictor of all-cause mortality [25]. However, the VALIANT Echo study, in a sample of 603 patients with LV dysfunction, heart failure, or both 5 days after myocardial infarction, showed that both longitudinal and circumferential strain and strain rate are the independent prognostic indicators in patients with high-risk myocardial infarction [10]. In the present study, it was shown that GLS is an independent predictor and the optimal GLS cutoffs for predicting composite events is > –12.3%, with a sensitivity and specificity of 95.7% and 67.0%.

Currently, LA function is assessed by LA volume, mechanical function and strain. Previous observation reported that LA volume is significantly related to cardiovascular disease and is independently correlated to death or heart failure [26]. LA mechanical function consists of the reservoir function, conduit and contractile function. LA reservoir function, which reflects LA relaxation, is particularly important during acute ischemia [27]. However, assessing changes in LA volume during different periods of the cardiac cycle is highly time-consuming; in addition, applying a simple geometric model to an asymmetric chamber may affect the estimation of LA volume [28]. Recently, by directly evaluating LA myocardial deformation to assess LA reservoir function post-AMI, clinically relevant information can be provided. PALS, which is evaluated by speckle-tracking derived strain, shows the direct evaluation of the atrial myocardium and may better reflect the properties of LA [29, 30]. Antoni et al. [31] confirmed the value of PALS to predict adverse events in patients after AMI treated with PCI, since only 48 of 320 patients (15%) reached the composite endpoint. This event rate was higher than the rate herein, where 23 of 199 patients (11.6%) experienced these events, perhaps due to a significantly shorter follow-up time. However, Ersboll et al. [16] found that the magnitude of PALS during the reservoir phase depends on the GLS and LA size, and measurement of PALS has no independent prognostic value. In patients with post-AMI, LA relaxation may be damaged by myocyte loss and LV filling pressure may also increase, both of which may be present, possibly limiting atrial expansion independently of LV longitudinal contraction damage, consequently increasing the risk of LV remodeling and adverse events [31, 32]. In the present study, PALS, like LV GLS, was found to be another independent predictor of LV remodeling; and a higher PALS value < 23.8%, with a sensitivity and specificity of 88.1% and 65.2%, was shown to be an independent predictor of a composite event.

In the current study, the independent prognostic value of PALS and LV GLS in patients with STEMI after pPCI was observed. Additionally, PALS did not add significant incremental value beyond LV GLS in the prediction of LV remodeling (AUC: 0.05, $p = 0.24$) and clinical events (even a decrease in AUC: 0.03, $p = 0.69$). The highly predictive values of GLS and PALS are further underscored.

Limitations of the study

A number of limitations of this study should be acknowledged. First, this is a single-center experience. In addition, the enrolled population was limited to patients with their first STEMI treated with pPCI, with low-risk AMI, and patients who died before completing their 6-month echocardiogram were excluded. Therefore, selection bias and potential selection bias should be taken into account when interpreting the findings. Finally, although the longitudinal, circumferential and radial strain of LV was analyzed, the impairment of right ventricular function was not assessed, which needs further study.

Conclusions

In conclusion, in patients with STEMI in any location treated with pPCI, both LV GLS and PALS are both more sensitive to myocardial damage and provide independent prognostic value for adverse LV remodeling and clinical events. However, the ability of the combination of PALS and GLS to predict LV remodeling and clinical outcomes may not be superior to that of a single indicator.

Conflict of interest: None declared

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