Prognostic Value of Follow-up Measures of Left Ventricular Global Longitudinal Strain in Patients With ST-Segment Elevation Myocardial Infarction

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Introduction: After ST-segment elevation myocardial infarction (STEMI), follow-up imaging is currently recommended only in patients with left ventricular ejection fraction (LVEF) <40%. Left ventricular global longitudinal strain (LVGLS) was shown to improve risk stratification over LVEF in these patients but has not been thoroughly studied during follow-up. The aim of this study was to explore the changes in LVGLS after STEMI and their potential prognostic value.

Materials and Methods: Data were analyzed from an ongoing STEMI registry. Echocardiography was performed during the index hospitalization and 1 year after STEMI; LVGLS was expressed as an absolute value and the relative LVGLS change (ΔGLS) was calculated. The study end point was all-cause mortality.

Results: A total of 1,409 STEMI patients (age 60 ± 11 years; 75% men) who survived at least 1 year after STEMI and underwent echocardiography at follow-up were included. At 1-year follow-up, LVEF improved from 50% ± 8% to 53% ± 8% (*P* < .001) and LVGLS from 14% ± 4% to 16% ± 3% (*P* < .001). Median ΔGLS was 14% (interquartile range, 0.5%-32%) relative improvement. Starting 1 year after STEMI, a total of 87 patients died after a median follow-up of 69 (interquartile range, 38-103) months. The optimal ΔGLS threshold associated with the end point (derived by spline curve analysis) was a relative decrease >7%. Cumulative 10-year survival was 91% in patients with ΔGLS improvement or a nonsignificant decrease, versus 85% in patients with ΔGLS decrease of >7% (*P* = .001). On multivariate Cox regression analysis, ΔGLS decrease >7% remained independently associated with the end point (hazard ratio, 2.5 [95% CI, 1.5–4.1]; *P* < .001) after adjustment for clinical and echocardiographic parameters.

Conclusions: A significant decrease in LVGLS 1 year after STEMI was independently associated with long-term all-cause mortality and might help further risk stratification and management of these patients during follow-up. (J Am Soc Echocardiogr 2024;37:666-73.)

Keywords: ST-Segment elevation myocardial infarction, Left ventricular global longitudinal strain

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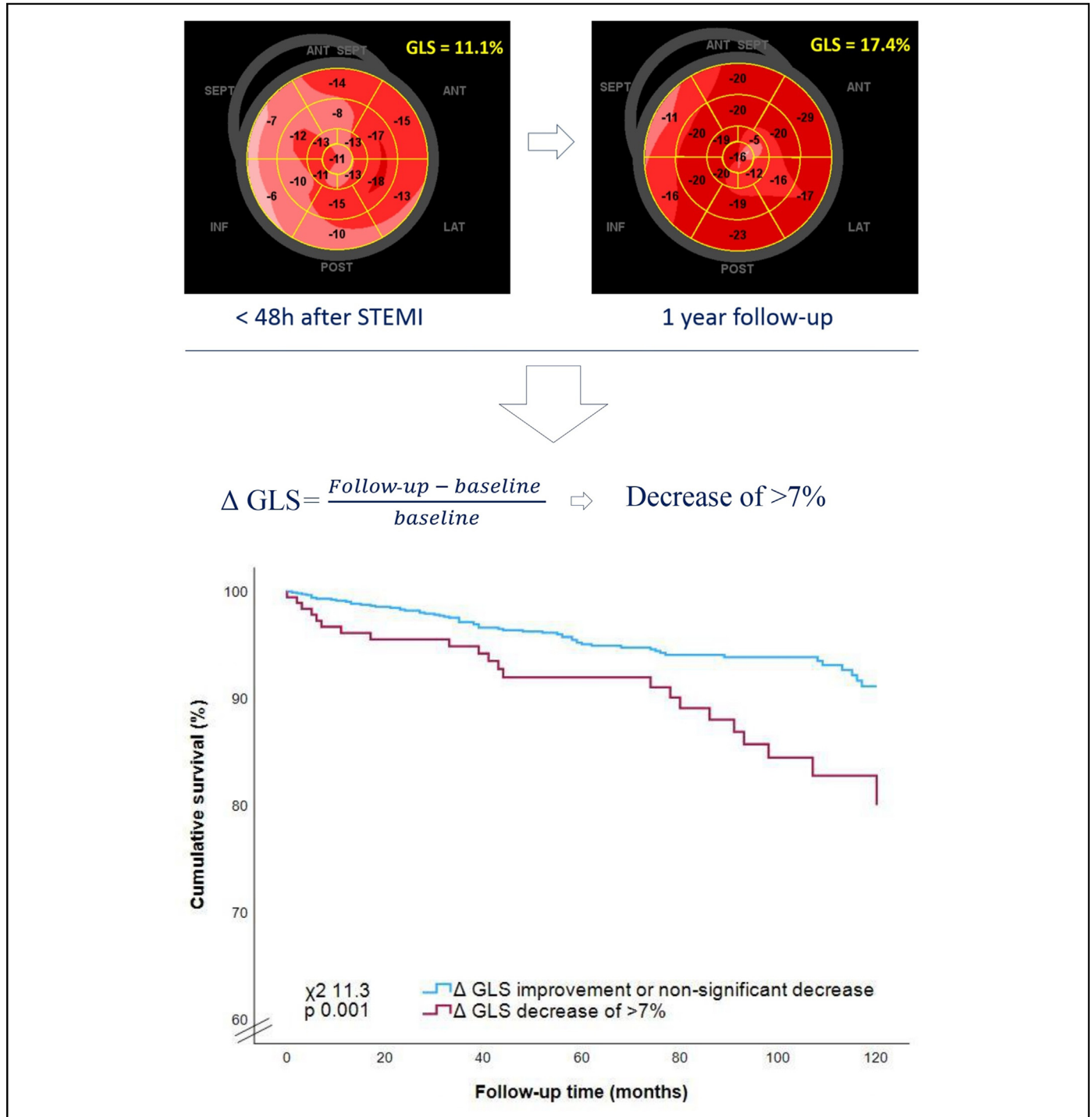
Central Illustration Prognostic value of change in LVGLSleft ventricular global longitudinal strain after STEMI-elevation myocardial infarction.

INTRODUCTION

Comprehensive transthoracic echocardiography (TTE) after ST-segment elevation myocardial infarction (STEMI) is an integral part of clinical care and risk stratification. Current guidelines recommend TTE in all patients during the index hospitalization, but follow-up imaging only if baseline left ventricular (LV) ejection fraction (LVEF) is ≤40% in order to further optimize heart failure (HF) medications or assess the need for an implantable cardioverter defibrillator.[1] In the era of primary percutaneous coronary intervention and optimal medical therapy, patient survival, admission for HF, and impaired LVEF rates post-STEMI have improved significantly.[2,3] In the SWEDEHEART registry, the incidence of impaired LVEF after STEMI was reported as having decreased from 37% in 1997 to

HIGHLIGHTS

- At baseline after STEMI, LVEF and LVGLS are on average preserved or mildly reduced.
- At 1 year after STEMI LV systolic function has improved in most patients.
- A >7% relative decrease in LVGLS is linked to 2.5 times higher all-cause mortality.

Abbreviations
ΔGLS = Relative left ventricular global longitudinal strain
HF = Heart failure
HR = Hazard ratio
IQR = Interquartile range
LV = Left ventricular
LVEF = Left ventricular ejection fraction
LVGLS = Left ventricular global longitudinal strain
MI = Myocardial infarction
STEMI = ST-segment elevation myocardial infarction
TTE = Transthoracic echocardiography

26% in 2013.[2] Furthermore, LVEF has been described to improve during follow-up after STEMI,[4-7] most likely secondary to a gradual resolution of myocardial stunning and the positive effect of medical therapy.[8] However, a lack of LVEF improvement post-STEMI has been shown to be associated with worse outcome.[4,5]

Left ventricular global longitudinal strain (LVGLS) measured by speckle-tracking echocardiography is a more sensitive parameter of LV dysfunction than LVEF and has shown incremental prognostic value when measured immediately after STEMI.[9] Nevertheless, the evolution of LVGLS after STEMI and its prognostic value have not been extensively investigated. Accordingly, the aim of the current study was to (1) describe the relative changes of LVGLS (ΔGLS) from baseline to 1-year follow-up after STEMI in a large contemporary patient cohort; and (2) explore the prognostic value of ΔGLS in addition to baseline assessment.

METHODS

Study Population and Data Collection

Patients admitted with STEMI at the Leiden University Medical Center in the Netherlands from September 2004 to April 2021 were included from an ongoing registry.[10] All patients underwent primary percutaneous coronary intervention and were treated with guideline-directed medical therapy according to a standardized institutional protocol based on contemporary European Society of Cardiology guidelines.[1] Demographics and clinical characteristics, including cardiovascular risk factors and biochemistry, were collected during admission. Patients were followed up for the primary end point of all-cause mortality, and survival data were collected from the departmental information system (EPD-Vision, Leiden University Medical Center, The Netherlands), which is linked to municipal registries.

According to the institutional protocol, TTE was performed within 48 hours of the index hospitalization and repeated after 1 year. The following exclusion criteria were applied: (1) previous myocardial infarction (MI) and/or a known history of HF, (2) severe aortic stenosis

at baseline echocardiography, (3) missing TTE at baseline or at 1-year follow-up, or (4) suboptimal image quality for LVGLS assessment.

All data used in the present study were collected for routine clinical purposes and handled anonymously. The requirement for written informed consent was waived by the institutional review board on a patient level due to the retrospective design of the study.

Transthoracic Echocardiography

Transthoracic echocardiography was performed in the left lateral decubitus position using a commercially available ultrasound system (Vivid 7, Vivid E9 or E95; GE Vingmed Ultrasound). Acquisitions included M-mode, two-dimensional, color, pulsed-wave, and continuous-wave Doppler traces. Echocardiographic loops were digitally archived to allow offline analysis (EchoPac 202, 203 and 204, GE Vingmed Ultrasound), and measurements were performed retrospectively. According to current recommendations[11] LVEF, LV end-diastolic volume, and LV end-systolic volume were calculated using Simpson's biplane method. Left ventricular end-diastolic diameter, LV end-systolic diameter, interventricular septal thickness, and posterior wall thickness were measured in the parasternal long-axis view at the level of the mitral valve leaflet tips. In addition, the left atrial volume index was calculated using the biplane area–length method and indexed to body surface area. Right ventricular fractional area change and tricuspid annular plane systolic excursion were calculated from the focused apical 4-chamber view. Severity of mitral regurgitation and tricuspid regurgitation was obtained from the official report, where it was graded based on a multiparametric approach.[12] Mitral and tricuspid regurgitations were considered significant if above grade II. Pulmonary artery systolic pressure was estimated from the maximal tricuspid regurgitation velocity and inferior vena cava diameter during respiration.

Speckle-tracking strain analysis was used to calculate LVGLS from apical 2-, 3- and 4-chamber views as the mean peak systolic strain of 17 segments (Figure 1). Left ventricular global longitudinal strain is expressed as absolute values, with higher values indicating better myocardial contraction.

Statistical Analysis

Continuous data are presented as mean ± SD or median and interquartile range (IQR), as appropriate. Categorical data are presented as frequencies and percentages. Paired samples t tests were used to compare continuous baseline versus follow-up parameters, and McNemar tests to compare categorical variables. The ΔGLS was calculated as follow-up LVGLS minus baseline LVGLS divided by baseline LVGLS and expressed as a percentage (Central Illustration). Penalized spline curve analysis was performed to dichotomize ΔGLS using a hazard ratio (HR) of 1. The ΔGLS is a well-validated measure in cardio-oncology, with a relative decrease of >15% recommended in the guidelines as a threshold for increased risk for future LVEF worsening.[13] Therefore, in addition to the threshold identified by spline curve analysis, ΔGLS was also dichotomized at >15 decrease.

To demonstrate survival differences between the groups, Kaplan-Meier curves were constructed and compared with log-rank tests. The association between ΔGLS and mortality was explored by univariate Cox regression analysis, and a multivariate Cox regression model was constructed using variables with a P value < .05 on univariate analysis. Clinical parameters were first analyzed in a separate multivariate model, and statistically significant ones were included in additional multivariable models. P value < .05 was considered
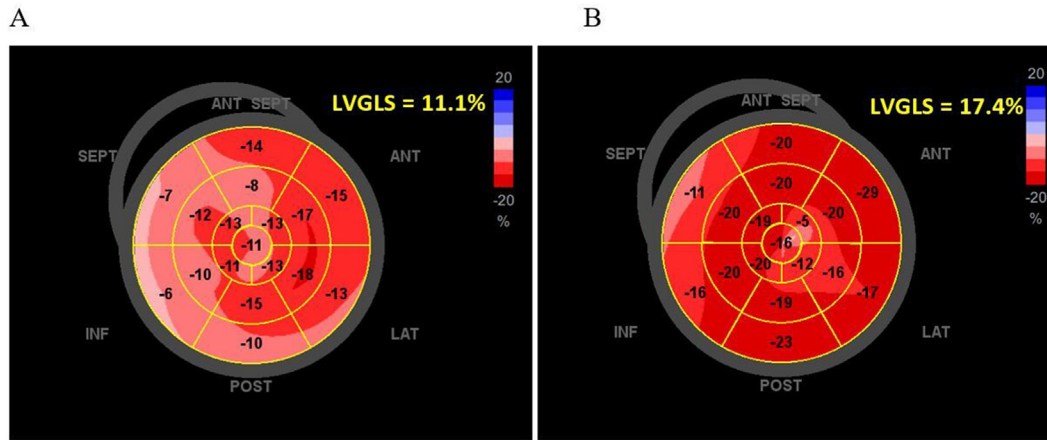
Figure 1 Example of LVGLS measured at baseline **(A)** and at 1-year follow-up **(B)** in the same STEMI patient. In panel A, the 17-segment bull's-eye shows an LVGLS of 11.1%. Panel B shows significant LVGLS improvement from 11.1% to 17.4%, corresponding to a 57% relative increase of LVGLS (ΔGLS). *ANT*, Anterior LV wall; *INF*, inferior LV wall; *LAT*, lateral LV wall; *POST*, posterior LV wall; *SEPT*, septal wall.

significant. Statistical analysis was performed using SPSS version 25.0 (IBM) and R version 4.2.1 (R Foundation for Statistical Computing, packages "foreign", "stats", "survival", "rms", and "Greg").

RESULTS

Patient Population

A total of 1,756 patients were included. Eighty-five patients were excluded due to a previous MI and/or HF, 2 patients because of severe aortic stenosis, and 19 patients because they died before the 1-year follow-up visit. In addition, 241 patients were lost to follow-up at 1 year or had suboptimal image quality for LVGLS measurement. The final population consisted of 1,409 patients (mean age, 60 ± 11 years; 1,059 [75%] men). Baseline clinical characteristics are presented in Table 1. Arterial hypertension was present in 523 (37%), diabetes mellitus in 136 (10%), and chronic obstructive pulmonary disease in 49 (4%) patients; 734 (52%) patients had multivessel disease.

Baseline echocardiographic characteristics are summarized in Table 2. Overall LV size was normal, and LV function was only mildly impaired, according to LVEF and LVGLS. Significant mitral and tricuspid regurgitations were infrequently seen, and, on average, right ventricular function was preserved.

Median time to follow-up echocardiography was 364 (IQR, 361-67) days. As shown in Table 2, LV end-diastolic volume, LV end-diastolic diameter, and left atrial volume index slightly increased at follow-up, while LVEF improved from 50% ± 8% to 53% ± 8% (P < .001) and LVGLS improved from 14% ± 4% to 16% ± 3% (P < .001). The median ΔGLS was 14% (IQR, 0.5%-32%) improvement.

Association Between Evolution of LVGLS at Follow-up and Long-Term Outcome

After a median follow-up of 69 (IQR, 38-103) months, 87 (6.2%) patients died. On spline curve analysis (Figure 2), the optimal threshold value for ΔGLS was identified as −7%, indicating that more than a 7% relative decrease of LVGLS from baseline was associated with an increased risk of all-cause mortality. An improvement or a nonsignificant decrease in ΔGLS was observed in 1,221 (87%) patients, while a ΔGLS decrease of >7% was observed in 188 (13%).

When applying the other cutoff value of >15% relative decrease in ΔGLS, an improvement or a nonsignificant decrease was observed in 1,325 (94%) patients and a decrease in 84 (6%) patients.

Table 1 Baseline clinical characteristics of the total population

Variable	All patients (N = 1,409)
Age, years	60 ± 11
Gender, male	1,059 (75.2)
Arterial hypertension	523 (37.1)
Hyperlipidemia	309 (22)
Positive family history	645 (46.2)
Diabetes mellitus	136 (9.7)
Current smoker	553 (39.2)
COPD	49 (3.5)
Atrial fibrillation	27 (1.8)
BMI, kg/m²	26.8 ± 3.9
Peak troponin T, ng/L	3,115 (1,300-6,251)
Peak CK, U/L	1,193 (578-2,318)
eGFR, mL/min/1.73 m²	86 ± 17
Multivessel disease	734 (52.1)
Systolic blood pressure on admission, mm Hg	134 ± 24
Heart rate at discharge, bpm	69 ± 12
QRS duration >120 ms at discharge	77 (5.5)
Medication recommendations on discharge	
RAS inhibitors	1,284 (91.1)
Beta-blockers	1,325 (94)
Aldosterone antagonists	28 (2)
Diuretics	104 (7.4)

BMI, Body mass index; *CK*, creatine phosphokinase; *COPD*, chronic obstructive pulmonary disease; *eGFR*, estimated glomerular filtration rate; *RAS*, renin-angiotensin system.
Data are presented as mean ± SD, median (IQR), or n (%).

Table 2 Baseline and 1-year-follow-up echocardiographic characteristics of the total population

Variable	Baseline (N = 1,409)	Follow-up (N = 1,409)	P value
LV EDD, mm	49 ± 5	50 ± 6	<.001
LV ESD, mm	32 ± 6	32 ± 7	.47
IVST, mm	11.3 ± 1.6	10.8 ± 1.5	<.001
PWT, mm	9.9 ± 1.4	9.5 ± 1.3	<.001
LV EDV, ml	142 ± 45	144 ± 49	<.001
LV ESV, ml	71 ± 28	69 ± 31	<.001
LVEF, %	50 ± 8	53 ± 8	<.001
LVGLS, %	14 ± 3.5	16.1 ± 3.2	<.001
LAVi, ml/m²	29 ± 9	31 ± 10	<.001
Significant MR	6 (0.4)	11 (0.8)	.33
RV FAC, %	43 ± 9	44 ± 8	<.001
TAPSE, mm	20 ± 3	21 ± 3.1	<.001
Significant TR	6 (0.4)	7 (0.5)	1
PASP, mm Hg	17.2 ± 11.4	17.1 ± 5.7	.98

IVST, Interventricular septal thickness; *LAVi*, left atrial volume index; *LV EDD*, LV end-diastolic diameter; *LV EDV*, LV end-diastolic volume; *LV ESD*, LV end-systolic diameter; *LV ESV*, LV end-systolic volume; *MR*, mitral regurgitation; *PASP*, pulmonary artery systolic pressure; *PWT*, posterior wall thickness; *RV FAC*, right ventricular fractional area change; *TAPSE*, tricuspid annular plane systolic excursion; *TR*, tricuspid regurgitation.
Data are presented as mean ± SD or *n* (%).

Cumulative 1-, 5-, and 10-year survival was 99%, 95%, and 91%, respectively, in patients with ΔGLS improvement or a nonsignificant decrease. In contrast, the survival rates for those with a ΔGLS decrease of >7% were 96%, 94%, and 85% at 1, 5, and 10 years, respectively Central Illustration Figure 3A; log-rank χ^2 = 11.3; P = .001). When ΔGLS was dichotomized by a threshold of 15% decrease, 1-, 5-, and 10-year survival was 99%, 95%, and 91% for the group with an improvement or a nonsignificant decrease and 95%, 91%, and 77% for those with a decrease of >15%, respectively (Figure 3B; log-rank χ^2 = 16.3; P < .001).
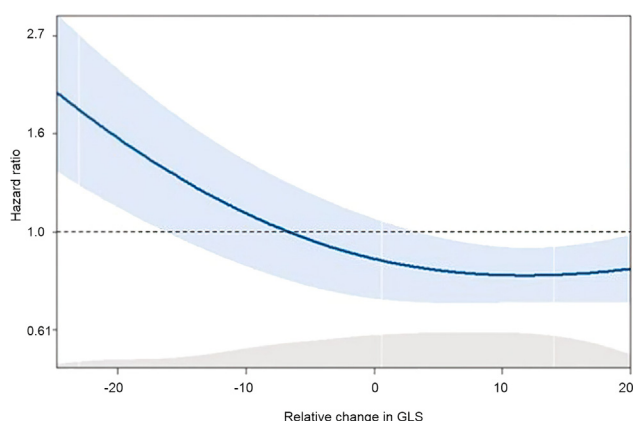
Figure 2 A ΔGLS spline curve for all-cause mortality across a range of relative change in LVGLS. Negative values of relative change signify the decrease in GLS at 1-year follow-up. The *gray area* above the x axis represents the distribution frequency.

On the univariate Cox regression analysis of clinical characteristics, age, arterial hypertension, chronic obstructive pulmonary disease, diabetes mellitus, multivessel disease, heart rate upon discharge from hospital, QRS duration >120 ms, troponin T level, creatine kinase, and estimated glomerular filtration rate were significant predictors of the outcome. These variables (creatine kinase was not included due to collinearity with troponin T) were used in a separate multivariate Cox regression model, where age, chronic obstructive pulmonary disease, QRS duration >120 ms, troponin T level, and heart rate remained significantly associated with outcome. These variables were subsequently included in multivariate analyses, which included echocardiographic parameters (Table 3).

On univariate analysis of echocardiographic parameters, baseline LVEF, LVGLS, tricuspid annular plane systolic excursion, pulmonary artery systolic pressure, and both thresholds of ΔGLS were significantly associated with all-cause mortality. In different multivariate models, a ΔGLS decrease of >7 % remained associated with mortality (HR = 2.5 [95% CI, 1.5-4.1]; P < .001 when adjusted for baseline LVGLS; and HR = 2.1 [95% CI, 1.3-3.4]; P = .003 when adjusted for baseline LVEF) together with a number of clinical variables and tricuspid annular plane systolic excursion. When adding to the model the changes in LVEF (Δ) over time, a ΔGLS decrease remained significantly associated with the outcome (Supplemental Table 1). Similar results were obtained when including a ΔGLS decrease of >15% (HR = 2.4 [95% CI, 1.3-4.5]; P = .004 when adjusted for baseline LVGLS; and HR = 2.0 [95% CI, 1.1-3.6]; P = .017 when adjusted for baseline LVEF).

DISCUSSION

The main findings of this study can be summarized as follows: (1) when measured at baseline and at 1-year follow-up after STEMI, LVGLS improved in the majority of patients, (2) a significant worsening of LVGLS at follow-up, defined as a relative decrease of either >7% or >15%, was independently associated with a 2.0 to 2.5 times higher risk of all-cause mortality, after adjusting for clinical parameters and baseline LV and right ventricular function.

Risk Stratification After STEMI

Current guidelines recommend long-term risk assessment before hospital discharge in all patients experiencing an acute MI.[1] Left ventricular ejection fraction has long been the cornerstone of risk stratification,[14] reflecting the extent of myocardial damage, and it is firmly linked to all-cause mortality.[15,16] Irreversible myocardial loss by acute MI can be limited by prompt revascularization. Nevertheless, ischemia and reperfusion result in myocardial stunning due to metabolic changes in cardiomyocytes, such as rapid depletion of energy reserves and abnormal calcium metabolism.[8] Some function is therefore regained within the first few hours after reperfusion,[17] while further recovery takes place over days[18] and months after MI.[4] Antoni *et al.*[19] reported LV systolic function improving in 72% patients within the first year after MI, the majority of which (54% of patients) occurred within the first 3 months. A longer time to reperfusion,[18,20] diabetes mellitus, higher troponin levels, a culprit lesion in the left anterior descending artery, female sex, smoking, and worse LV function at baseline have all been associated with lack of subsequent improvement in LV systolic function.[4,19,21-23] Therefore, current guidelines recommend follow-up echocardiography in patients with baseline
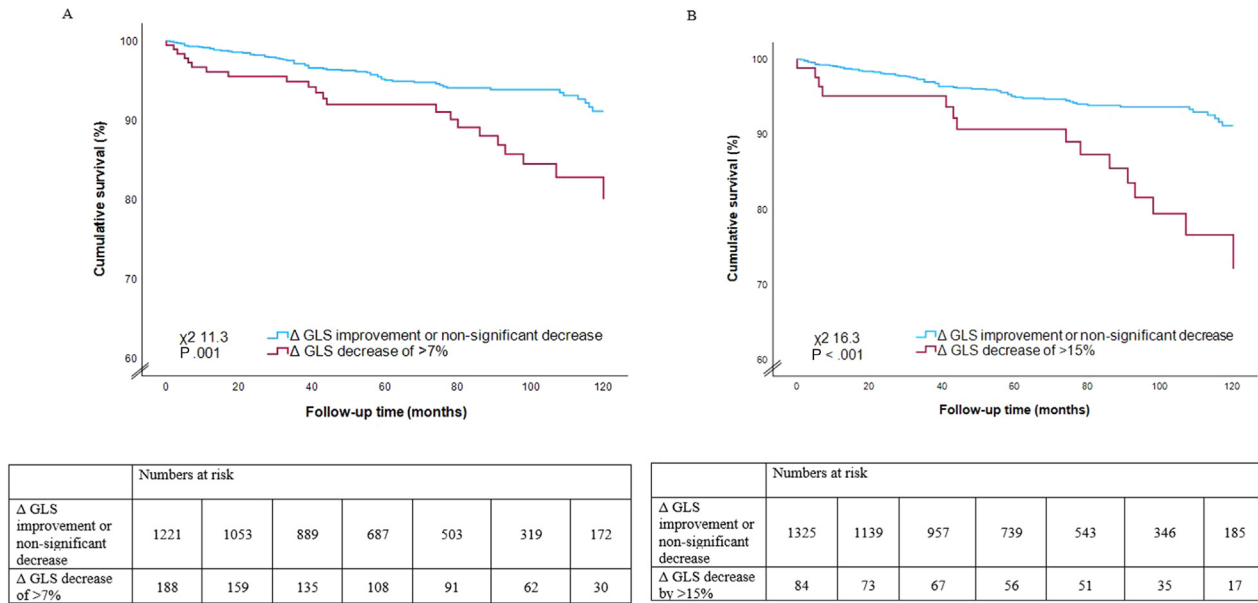
Figure 3 Kaplan-Meier curve for all-cause mortality, stratified according to **(A)** ΔGLS of >7% and **(B)** ΔGLS of >15%.

LVEF <40% to assess the changes in LV function over time and possibly optimize HF treatment. In initial studies, a lack of improvement in LVEF has been associated with worse outcomes: a 7 to 8 times higher risk of cardiovascular mortality has been reported in patients who did not recover LVEF above 50% at follow-up, compared with patients with a normal baseline LVEF[4] and/or recovered LVEF.[5] In our study, LVEF at baseline was preserved (mean 50%) and at 1-year follow-up increased significantly to 53% in line with previous studies. Still, identification of those patients with increased risk for further events, even in case of an LVEF >50%, remains crucial for proper risk stratification and patient management.

Over the last decade, LVGLS has been studied extensively as a more sensitive tool for evaluating LV systolic function. When measured at baseline, it has shown additional incremental value over LVEF in predicting all-cause mortality, sudden cardiac death, HF hospitalization, and other adverse cardiovascular events postinfarct.[24-27] In addition, LVGLS holds the advantage of better reproducibility than LVEF—among both experienced and novice echocardiographers.[28] Technical advances in postprocessing software allow for rapid, automated measurement of LVGLS, as well as improved intervendor agreement.[29,30] However, the changes in LVGLS during follow-up after STEMI have not been extensively studied especially in relation with outcome.

Evolution of LVGLS: Prognostic Implications

Recently, Iwahashi et al.[31] performed serial echocardiographic examinations in a small cohort of patients with STEMI and showed that three-dimensional LVGLS measured at follow-up had incremental value over LVEF change for predicting the combined end point of long-term cardiovascular mortality and HF hospitalization; however, due to the small sample size, the independent value of this specific echocardiographic approach could not be robustly tested. We expand on these results with a large, real-world cohort of STEMI patients, demonstrating that ΔGLS was independently associated with all-

cause mortality, in addition to known clinical risk factors and baseline echocardiographic parameters and on top of the changes in LVEF at follow-up.

In our study population, a ΔGLS threshold of >7% decrease was the optimal cutoff value associated with outcome. This finding suggests that since an improvement in LV function after primary percutaneous coronary intervention is expected in most patients, any lack of improvement in function identifies patients with potentially worse prognosis. When applying a more restrictive threshold of 15% decrease for ΔGLS, the independent association with worse long-term survival was maintained. This additional threshold for ΔGLS was investigated, since it is routinely applied in the setting of cardio-oncology,[13] where timely identification of LV function deterioration is of utmost importance. However, in STEMI patients, an even lower threshold may be clinically meaningful to identify patients who remain at higher risk, despite a normal or near-normal LVEF at baseline. Although our findings are hypothesis generating and prospective large studies would be required as confirmation, they suggest that repeating GLS measurements at 1-year follow-up may be useful to identify those patients at higher risk, especially among those with LVEF still within normal ranges and who deserve more frequent surveillance and possibly more intensive HF treatment.

Study Limitations

The primary limitations of this study are related to its retrospective design, as, for example, for the number of patients excluded because of loss at follow-up. However, for the patients included, clinical and echocardiographic data within the first year of follow-up were very complete as per institution protocol; in addition, the exclusion criteria chosen for this study may limit the applicability of our findings to the general post-STEMI population. Information regarding the exact cause of death was not systematically available and could not be used in the outcome analysis. Since the cutoff value for ΔGLS was derived from a single-center population and 1 echocardiography vendor, further studies are warranted to confirm our findings.

Table 3 Univariate and multivariate Cox regression analysis for prediction of all-cause mortality

| | Univariate analysis | | Multivariate analysis | | | | | | | |
| | | | Model 1 | | Model 2 | | Model 3 | | Model 4 | |
Variable	HR (95% CI)	P value	HR (95% CI)	P value	HR (95% CI)	P value	HR (95% CI)	P value	HR (95% CI)	P value
Age, years	1.1 (1 –1.1)	<.001	1.1 (1.0 – 1.1)	<.001	1.1 (1.0 – 1.1)	<.001	1.1 (1.0 – 1.1)	<.001	1.1 (1.0 – 1.1)	<.001
COPD	3.7 (1.9 – 7.1)	<.001	2.9 (1.5-5.7)	.002	2.7 (1.4-5.3)	.004	2.8 (1.4-5.6)	.003	2.6 (1.3-5.2)	.006
QRS > 120 ms	3.5 (1.9-6.3)	.001	2.2 (1.2-4.1)	.014	2.3 (1.2-4.3)	.009	2 (1.0-3.8)	.035	2.3 (1.2-4.3)	.009
Troponin, ng/L	1.1 (1.04-1.1)	<.001	1 (0.99-1.1)	.19	1 (1.0-1.1)	.018	1 (1.0-1.1)	.13	1 (1.0-1.1)	.023
Heart rate, bpm	1 (1.01-1.04)	<.001	1 (1.0-1.03)	.14	1 (1.0-1.03)	.06	1 (1.0-1.0)	.15	1 (1.0-1.03)	.062
LV EDV, mL*	1 (0.99-1)	.33								
LV ESV, mL*	1 (1.0-1.01)	.28								
LVEF, %*	0.96 (0.93-0.98)	<.001			0.98 (0.95-1.0)	.2			0.98 (0.96-1.0)	.22
LAVi, mL/m²*	1 (0.99-1.0)	.37								
LVGLS, %*	0.9 (0.8-0.9)	<.001	0.9 (0.85-0.98)	.013			0.92 (0.86-1.0)	.037		
Significant MR*	3.2 (0.4-23)	.25								
RV FAC, %*	1 (0.97-1.0)	.81								
TAPSE, mm*	0.9 (0.8-0.97)	.006	0.97 (0.9-1.0)	.42	0.97 (0.9-1.05)	.42	0.97 (0.9-1.1)	.5	0.97 (0.9-1.05)	.45
Significant TR*	0.1 (0-513033)	.72								
PASP, mm Hg*	1 (1.0-1.1)	.003								
ΔGLS decrease of >7%	2.4 (1.5-3.8)	<.001	2.5 (1.5-4.1)	<.001	2.1 (1.3-3.3)	.003				
ΔGLS decrease of >15%	2.8 (1.6-4.9)	<.001					2.4 (1.3-4.5)	.004	2 (1.1-3.6)	.01

COPD, Chronic obstructive pulmonary disease; *LAVi*, left atrial volume index; *LV EDV*, LV end-diastolic volume; *LV ESV*, LV end-systolic volume; *MR*, mitral regurgitation; *PASP*, pulmonary artery systolic pressure; *RV FAC*, right ventricular fractional area change; *TAPSE*, tricuspid annular plane systolic excursion; *TR*, tricuspid regurgitation.

Model 1: clinical variables + baseline TAPSE + baseline LVGLS + ΔGLS decrease of >7%. Model 2: clinical variables + baseline TAPSE + baseline LVEF + ΔGLS decrease of >7%. Model 3: clinical variables + baseline TAPSE + baseline LVGLS + ΔGLS decrease of >15%. Model 4: clinical variables + baseline TAPSE + baseline LVEF + ΔGLS decrease of >15%.

*TTE measurements performed at the index hospitalization.

CONCLUSION

At 1-year follow-up after STEMI, LVGLS improved in the majority of patients. A relative decrease of LVGLS of >7% or of >15%, however, was associated with worse prognosis after adjusting for clinical risk factors and baseline LV and right ventricular function. Speckle-tracking strain echocardiography therefore has the potential to refine the risk stratification of STEMI patients, even when they have normal or near-normal LVEF at baseline and follow-up.

REVIEW STATEMENT

Given her role as *JASE* Editor-in-Chief, Patricia A. Pellikka, MD, and given her role as JASE Associate Editor, Nina Ajmone Marsan, MD, PhD, had no involvement in the peer review of this article and have no access to information regarding its peer review. Full responsibility for the editorial process for this article was delegated to Neil J. Weissman, MD.

CONFLICTS OF INTEREST

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.echo.2024.03.007.

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