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Speckle Tracking Echocardiography Predicts Left Ventricular Remodeling after Acute ST-Segment Elevation Myocardial Infarction in Patients Undergoing Primary PCI

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SOUHRN

Cíl studie: Remodelace levé komory (left ventricular remodeling, LVR) po infarktu myokardu s elevacemi úseku ST (STEMI) významně ovlivňuje prognózu. I když trojrozměrná echokardiografie (3D-echo) přesně změří objem levé komory, lze případnou LVR v časném období po STEMI obtížně predikovat. Cílem této studie bylo zjistit, zda vstupní celková longitudinální deformace myokardu (global longitudinal strain, GLS) jako marker neinvazivního echokardiografického vyšetření může u pacientů léčených primární perkutánní koronární intervencí (PPCI) predikovat remodelaci levé komory po šesti měsících od STEMI.
Metody: Do této prospektivní observační studie bylo zařazeno 53 pacientů po prvním prodělaném STEMI, u nichž byla provedena PPCI a u nichž po šesti měsících následovalo kontrolní echokardiografické vyšetření. Funkce levé komory se hodnotila do 48 hodin po příhodě a po šesti měsících od příhody pomocí dvourozměrné (2D) echokardiografie, „speckle tracking" echokardiografie (STE) a trojrozměrné echokardiografie (3D-echo). Remodelace levé komory byla definována jako ≥ 15% zvětšení objemu levé komory na konci diastoly (LV end-diastolic volume, LVEDV) při vyšetření metodou 3D-echo. Pacienti byli zařazováni do skupin podle (ne)přítomnosti LVR a potenciální prediktory LVR byly statisticky analyzovány.
Výsledky: K LVR došlo u 19 pacientů (35,8 %). Při vstupním vyšetření měli pacienti s LVR větší objem levé komory, nižší ejekční frakci, vyšší index kinetiky stěny levé komory a nižší hodnotu GLS (–12,3 % vs. –18,7 %; $p < 0{,}001$). Analýza ROC křivky prokázala, že vznik LVR nejspolehlivěji predikuje hodnota GLS > –14,8 % (AUC: 0,796; senzitivita 78,95 %, specificita 82,35 %). Jako nezávislé prediktory potvrdila multivariační analýza parametry GLS > –14,8 % ($p < 0{,}001$) a stupeň koronárního průtoku podle klasifikace TIMI ($p = 0{,}013$).
Závěr: Celková longitudinální deformace myokardu měřená do 48 hodin od STEMI predikuje (ne)přítomnost LVR po šesti měsících. Dostupnost měření uvedeného parametru a jeho predikční síla by mohly podporovat jeho využití ve stratifikaci rizika v období časně po STEMI.

Keywords:
Global longitudinal strain
left ventricular remodeling
Primary PCI
STEMI
3D echocardiography

ABSTRACT

Purpose: Left ventricular remodeling (LVR) following ST-segment elevation myocardial infarction (STEMI) significantly impacts prognosis. Although three-dimensional echocardiography (3D-echo) accurately assesses LV volume, early LVR prediction remains challenging. This study evaluated whether baseline global longitudinal strain (GLS), a non-invasive echocardiographic marker, predicts LVR six months post-STEMI in patients treated with primary percutaneous coronary intervention (PPCI).
Methods: This prospective observational study included 53 first-time STEMI patients who underwent PPCI and completed a six-month echocardiographic follow-up. LV function was assessed within 48 hours and at six months using 2D echocardiography, speckle tracking echocardiography (STE), and 3D-echo. LVR was characterized as a ≥15% rise in LV end-diastolic volume (LVEDV) during follow-up 3D-echo. Patients were grouped based on LVR status, and potential LVR predictors were statistically analyzed.
Results: LVR occurred in 19 patients (35.8%). At baseline, LVR patients had larger LV volumes, lower ejection fraction, higher wall motion score index, and lower GLS (–12.3% vs. –18.7%, $p < 0.001$). ROC analysis identified GLS > –14.8% as the optimal LVR predictor (AUC: 0.796, sensitivity: 78.95%, specificity: 82.35%). Multivariate analysis confirmed GLS > –14.8% ($p < 0.001$) and TIMI flow grade ($p = 0.013$) as independent predictors.
Conclusion: GLS measured within 48 hours of STEMI strongly predicts LVR at six months. Its accessibility and predictive power may support its role in post-STEMI early risk stratification.

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Introduction

Despite improved acute-phase mortality in ST-segment elevation myocardial infarction (STEMI) over the past three decades,[1] left ventricular remodeling (LVR) remains a significant long-term consequence, contributing to left ventricular (LV) dysfunction and heart failure (HF).[2] Subsequent myocardial damage causes several LV structural alterations that may end in changes in LV shape and volume, which eventually culminate in HF. These LVR changes[3] occur in 30–35% of patients due to increased wall stress caused by cardiomyocyte loss and infarct area distension, even after appropriate and effective reperfusion, either by primary percutaneous coronary intervention (PPCI) or fibrinolytic therapy.[4–6]

From a pathophysiological standpoint, LVR may lead to major adverse cardiac events – HF and ventricular arrhythmias, and consequently increased mortality.[7] However, there is clinical evidence that post-MI LVR can be prevented or reversed in some cases.[8] Therefore, identifying patients with a high probability of LVR early after STEMI is important prognostically for risk stratification in the acute phase[9,10] and clinically for the implementation of proper preventive and therapeutic management strategies, especially for high-risk patients, including anti-remodeling therapies (beta-blockers, angiotensin-converting enzyme inhibitors, and aldosterone antagonists).[11–13]

LVR prediction can be problematic.[12] Over the years, various cardiac imaging techniques and parameters were proposed: conventional and three-dimensional echo (3D-echo) – ejection fraction (EF), LV end-diastolic and end-systolic volumes (EDV, ESV),[14,15] two-dimensional speckle tracking echocardiography (2D-STE) – global longitudinal strain (GLS),[16] microvascular obstruction,[17] and cardiac magnetic resonance (CMR) imaging – LV global function index.[18]

After an acute MI, early LVEF assessment by echocardiography determines the extent of myocardial damage and predicts early and late complications.[11] However, it can be misleading in this context because of myocardial stunning and the inability to differentiate viable and nonviable myocardium.[19,20]

Over decades, two-dimensional echocardiography (2D-echo) has been used to analyze the LV volumes, geometry, and function.[21] However, this modality has limitations due to the restricted examined spatial planes, the geometric assumptions for volume calculations, and the possible foreshortening of the geometric shapes in the rendered images.[22] The structural examination of the heart from multiple spatial planes is made possible by the 3D-echo. The recent iterations in real-time 3D-echo have closed the gap with higher spatial resolution modalities such as 64-channel ultrarapid computed tomography and CMR and provided a more accurate analysis of LV size and function.[23,24] It eliminates the need for geometric assumptions regarding the shape of the LV, and image processing can reduce the underestimation of LV size caused by foreshortening. At present, 3D-echo evaluation of LV volumes and EF, using transthoracic or transesophageal approaches, is preferred over 2D-echo due to its superior accuracy and reproducibility.[25]

Several studies have highlighted the added value of deformation imaging parameters in predicting LVR, especially GLS.[26–28] However, the optimal GLS threshold for predicting LVR and its comparative performance against 3D-echo parameters remain uncertain.

This study aimed to assess the ability of baseline GLS, measured within 48 hours of first-time acute STEMI, in predicting patients at risk of developing LVR, as assessed by 3D-echo at six months. By comparing GLS with standard echocardiographic markers, we sought to establish its utility in routine post-STEMI risk stratification.

Patients and methods

Study design and population

This prospective observational study enrolled patients presenting with first-ever acute STEMI who underwent PPCI within 24 hours of symptom onset and achieved post-procedural coronary Thrombolysis In Myocardial Infarction (TIMI) flow grade II or III.[29] We excluded patients with prior MI or HF or previous coronary revascularization, significant valvular disease, atrial fibrillation or flutter, or suboptimal echocardiographic window from the study. The study was conducted following the principles of the Declaration of Helsinki. Approval was granted by our institution's Ethics Committee, and all patients gave their written informed consent.

Clinical and laboratory evaluation

All patients underwent a comprehensive medical history evaluation and physical examination. The presence of cardiovascular risk factors was documented based on standard clinical definitions:

- Hypertension: blood pressure ≥ 140/90 mmHg or the use of antihypertensive drugs.[30]
- Diabetes mellitus: fasting plasma glucose levels >126 mg/dL, random glucose levels >200 mg/dL, or use of anti-diabetic medications.[31]
- Family history of coronary artery disease (CAD): a first-degree relative with CAD or sudden death before 55 years of age (men) and 65 years (women).[32]
- Dyslipidemia: LDL >160 mg/dL, total cholesterol >200 mg/dL, TG >150 mg/dL, and HDL <40 mg/dL), or use of lipid-lowering therapy.[33]
- Smoking: current or previous smoking history, and its duration.

Electrocardiogram (ECG) and angioplasty protocol

A 12-lead ECG was obtained upon admission and immediately post-PPCI using a Schiller machine (25 mm/s, 10 mm/mV). STEMI was classified as anterior or non-anterior based on ECG findings. ST-segment amplitude was measured pre- and post-PPCI, with ST-segment resolution documented.

PPCI was performed as early as possible and was considered successful if the residual stenosis in the culprit lesion was <30% with TIMI grade flow ≥ II. The total ischemic time (symptom onset to balloon inflation) and the door-to-balloon time were also recorded in every patient.

Echocardiography

All patients had baseline comprehensive conventional 2D-echo within 48 hours of admission using an EPIQ 7 machine (Philips Medical Systems, Andover, MA, USA) equipped with a 1–5 MHz X5-1 transducer. The whole study was done in the left lateral decubitus position. Images were acquired at end-expiration.

To acquire 2D-STE images, we adjusted the sector size, and depth to achieve optimal visualization of all LV myocardium in the 3 standard apical views (4-, 2-, and long-axis view) and parasternal short axis (basal, mid, and apical levels) at 60–100 frames per second. Three consecutive cardiac cycles were stored for offline analysis with QLab 10 software (cardiac motion quantification [CMQ]; Philips Medical Systems). LV end-diastolic and end-systolic diameters were measured according to the current guidelines, and the LVEF and LV volumes were calculated using the modified Simpson's biplane method.[21]

Regional wall motion was assessed using a 17-segment model – each segment is given a score according to its systolic function (1, normal; 2, hypokinesia; 3, akinesia, 4, dyskinesia; and 5, aneurysm). The wall motion score index (WMSI) was calculated by dividing the total wall motion scores of all segments by 17.[34] We measured peak early (e') and late (a') diastolic velocity at the mitral septal and lateral annulus using Tissue Doppler imaging.

LV deformation performance measurement by 2D-STE was done by analyzing the stored grayscale images offline using QLab 10 software (CMQ; Philips Medical Systems). The endocardial border was traced by placing markers on the mitral annulus and the apex at end-systole to automatically track the myocardium. Poor tracking quality was manually revised until it became acceptable. The width of the region of interest was adjusted to cover the entire myocardium. We calculated the global and segmental strain in 2 directions: the circumferential strain from the parasternal short-axis views at the three levels, and the longitudinal strain from the three apical views. Each of the apical or short-axis views was divided into 6 segments. GLS and global peak circumferential strain (GCS) were averaged from the total 17 segments. All these measurements were presented in a color-coded polar map. Any segment with longitudinal strain > –20% was considered abnormal.

We performed the 3D-echo studies using a wide-angle matrix array transducer, acquiring images from the apical windows. The entire LV cavity was carefully placed inside the pyramidal scan volume. We acquired the datasets over 4 cardiac cycles during a short breath-hold to avoid stitching artifacts. EF and LV volume measurements were computed offline using the QLab 10 software, which enables semi-automated identification of endocardial and epicardial borders. Three distinct modifiable cross-sections of each acquisition were displayed. The anatomical 2-chamber and 4-chamber images must be shown concurrently for the detection method to function correctly. The markers were then positioned at end-diastole and end-systole on the apex and mitral annulus. The program produced truncated ellipsoid end-diastolic and end-systolic 3D models of the LV using these markers. The LV cavity's papillary muscles were included in the volumes and corresponded to the LVEDV and LVESV. When necessary, manual adjustments were made.

Follow-up

We asked all patients to return for follow-up after six months to record clinical events (hospitalization or revascularization, adherence to medical therapy), echocardiographic examination using the same protocols, and detect any LVR compared with the baseline measurements (defined as >15% LVEDV increase from baseline measured by 3D-echo[10]).

Statistical analysis

The sample size was calculated using G*Power 3.1.[35] The study should include 52 patients to achieve 80% power to detect a 0.4 difference from the mean with $p = 0.05$.

Statistical analysis was performed using the Statistical Package for Social Science (SPSS) version 20 (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± SD or median (range) for normal or skewed distribution respectively, while categorical variables were presented as numbers (%). Univariate analysis of LVR occurrence was done using Chi-square (or Fisher Exact tests if appropriate) for categorical variables, and Student's T-test for continuous variables with normal distribution or Mann–Whitney test for skewed continuous variables. ROC analysis was used to determine the optimal GLS cutoff for LVR prediction. Multivariate regression analysis (forward stepwise method) identified the independent predictors of LVR among variables with $p < 0.1$ in univariate analysis. A two-tailed $p < 0.05$ was considered statistically significant.

Results

A total of 85 patients underwent PPCI and had both 2D- and 3D-echo examinations within 48 hours of admission. At the six-month follow-up, 56 patients returned, but only 53 had matching pairs of analyzable LV volume data at follow-up and were included in the final analysis (**Fig. 1**). The median follow-up duration was 195 days (6.5 months, range: 188–200 days).

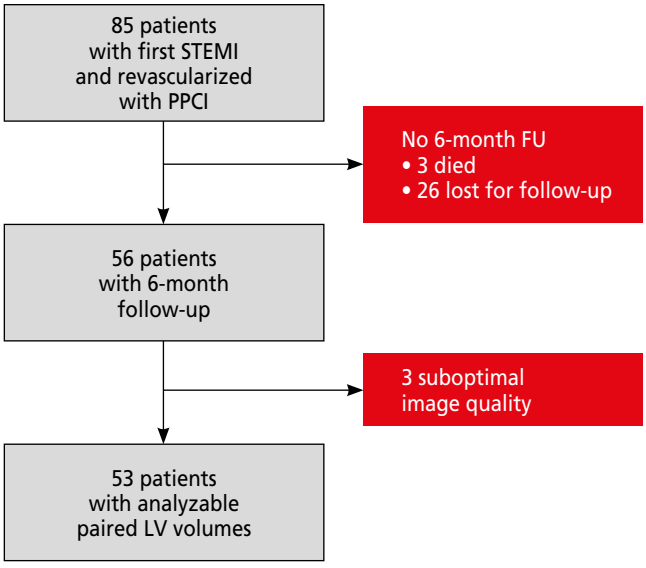

Fig. 1 – Flow diagram of the patient selection process and follow-up.

Table 1 – Baseline clinical characteristics of the study population

Variable	No (%)
Demographic data	
Age (years)	50.8 ± 10.8
Weight (kg)	79.7 ± 12.7
Height (m)	1.6 ± 0.08
BMI (kg/m^2)	27.9 ± 4.4
BSA (m^2)	1.8 ± 0.15
Male	42 (79.2%)
Female	11 (20.8%)
Cardiovascular risk factors	
Hypertension	13 (24.5%)
Diabetes	15 (28.3%)
Smoking	42 (79.2%)
Family history of CAD	5 (9.4%)
Dyslipidemia	13 (24.5%)
STEMI type	
Anterior Non-anterior (inferior, lateral, posterior)	30 (56.6%) 23 (43.4%)
Culprit vessel	
LAD Non-LAD	30 (56.6%) 23 (43.4%)
TIMI flow	
TIMI II TIMI III	6 (11.3%) 47 (88.7%)
Key time intervals	
Total ischemic time (hours)	6.3 ± 4.4
Door to balloon (hours)	1.6 ± 1.1
ST-segment elevation	
Pre-PCI ST amplitude (mm)	3.3 ± 1.7
Post-PCI ST amplitude (mm)	1.2 ± 1.1
Change in ST amplitude (mm)	2.0 ± 1.6

BMI – body mass index; BSA – body surface area; CAD – coronary artery disease; LAD – left anterior descending coronary artery; PCI – percutaneous coronary intervention; STEMI – ST-elevation myocardial infarction; TIMI – thrombolysis in myocardial infarction. Data are presented as mean ± SD or number and (%).

Baseline characteristics of all patients

The mean age of the study population was 50.8 ± 10.8 years, and 42 (79.2%) were males, Smoking was the most prevalent CAD risk factor, and 30 (56.6%) patients presented with anterior STEMI. The mean total ischemic time was 6.3 hours, and the median door-to-balloon time was 1.6 hours (**Table 1**).

At baseline, the patients had moderate reduction in LVEF (44.3 ± 10.2%), with a mean GLS of –15.86 ± 4.5%. The baseline echocardiography parameters of the patients are presented in **Table 2**.

Table 2 – Baseline echocardiographic parameters of the study population

Variable	No (%)
2D echocardiography	
LVEDD 2D (cm)	4.75 ± 0.5
LVESD 2D (cm)	3.57 ± 0.5
LVEDV 2D (ml)	89.02 ± 19.7
LVESV 2D (ml)	50.35 ± 20.8
EF "Simpson's" (%)	44.32 ± 10.2
WMSI	1.58 ± 0.4
E/E' ratio	9.82 ± 4.7
Speckle tracking echocardiography	
GLS	–15.86 ± 4.5
GCS	–19.52 ± 6.8
3D echocardiography	
LVEDV 3D (ml)	87.80 ± 17.3
LVESV 3D (ml)	50.03 ± 17.6
EF 3D (%)	44.13 ± 9.5
SV 3D (ml)	37.79 ± 6.7

EF – ejection fraction; GCS – global circumferential strain; GLS – global longitudinal strain; LVEDD – LV end-diastolic diameter; LVEDV – LV end-diastolic volume; LVESD – LV end-systolic diameter; LVESV – LV end-systolic volume; SV – stroke volume; WMSI – Wall Motion Score Index.
Data are presented as mean ± SD or number and (%).

Follow-up data

Clinical

During the six-month follow-up period, only three patients were hospitalized – one for HF, and two for recurrent acute coronary syndromes. Six patients underwent additional revascularization for non-infarct-related arteries. Compliance with optimal medical therapy was high (94.33%).

Temporal changes in echocardiographic parameters

At six months, significant improvements in GLS and WMSI were observed in the entire cohort. GLS improved from –15.7% (–19.7% to –12.3%) at baseline to –17.5% (–20.9% to –13.8%) at follow-up ($p < 0.001$). WMSI also improved significantly from 1.47 (1.24–1.88) to 1.35 (1.12–1.82) ($p = 0.005$). These findings suggest partial recovery of myocardial function, particularly in non-remodeling patients.

Comparison between remodeling and non-remodeling groups

LVR, defined as a >15% increase in LVEDV by 3D-echo at six months, occurred in 19 patients (35.8%). The LVR group had a significantly higher body mass index (BMI). Anterior STEMI was significantly associated with LVR, as was lower TIMI flow post-PPCI. Patients with LVR had sig-

Table 3 – Distribution of the studied parameters according to the occurrence of LV adverse remodeling

Variable	No remodeling (n = 34)	Remodeling (n = 19)	*p*
Demographic data			
Age (years)	51.9 ± 11.2	48.8 ± 10	0.337
Gender Male Female	 27 (79.4%) 7 (20.6%)	 15 (78.9%) 4 (21.1%)	1.000
BMI	26.7 (24.2–28.3)	30.8 (24.2–34)	0.047
Cardiovascular risk factors			
Hypertension	10 (29.4%)	3 (15.8%)	0.334
Diabetes	9 (26.5%)	6 (31.6%)	0.756
Smoking	27 (79.4%)	15 (78.9%)	1.000
Family history of CAD	4 (11.8%)	1 (5.3%)	0.643
Dyslipidemia	10 (29.4%)	3 (15.8%)	0.334
STEMI type			
Non-anterior Anterior	19 (55.9%) 15 (44.1%)	4 (21.1%) 15 (78.9%)	0.021
Culprit vessel			
Non-LAD LAD	19 (55.9%) 15 (44.1%)	4 (21.1%) 15 (78.9%)	0.021
TIMI flow			
TIMI II TIMI III	1 (16.7%) 33 (70.2%)	5 (83.3%) 14 (29.8%)	0.018
Key time intervals			
Total ischemic time (hours)	5 (3–7)	6 (5–9)	0.168
Door to balloon (hours)	1 (1–2)	1 (1–2)	0.771
ST-segment elevation			
Pre-PCI ST amplitude (mm)	3 (2–4)	4 (3–5)	0.090
Post-PCI ST amplitude (mm)	1 (0–1)	2 (1–3)	0.007
Change in ST amplitude (mm)	2 (1–2)	2 (1–3)	0.931

BMI – body mass index; CAD – coronary artery disease; LAD – left anterior descending coronary artery; PCI – percutaneous coronary intervention; STEMI – ST-elevation myocardial infarction; TIMI – Thrombolysis In Myocardial Infarction score.
Data are presented as mean ± SD or number and (%) or median and (25.–75. percentile).

nificantly higher maximal ST-segment elevation after PCI, however, the change in ST-segment amplitude between pre-PCI and post-PCI was not significant. There was no significant difference between the two groups in terms of age, CAD risk factors, total ischemic time, or door-to--balloon time (**Table 3**).

Compared to the non-remodeling group, patients with LVR had significantly larger baseline LV volumes, lower LVEF, higher WMSI, and lower LV GLS values (**Table 4**).

GLS prediction ability of LVR

In ROC curve analysis, GLS > –14.8% was identified as the optimal threshold for predicting LVR, yielding an AUC of 0.796 (p <0.001), with a sensitivity of 78.95% and a specificity of 82.35% (**Fig. 2**).

Prediction of LVR

Multivariate regression analysis confirmed GLS >–14.8% (OR: 0.015, 95% CI: 0.002–0.152, p <0.001) and TIMI

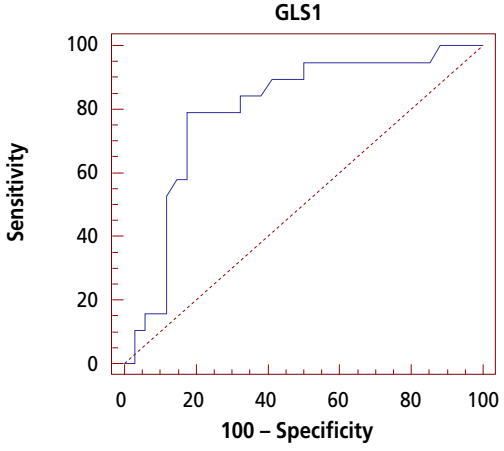

Fig. 2 – ROC curve analysis for GLS cut-off prediction of LV adverse remodeling, where GLS >–14.8% provided optimal sensitivity (78.95%) and specificity (82.35%).

Table 4 – Baseline echocardiographic parameters in the dichotomized LVR groups

Variable	No remodeling (n = 34)	Remodeling (n = 19)	*p*
2D echocardiography:			
LVEDD 2D (cm)	4.65 (4.30–4.80)	4.8 (4.50–5.30)	0.152
LVESD 2D (cm)	3.3 (3.2–3.6)	3.7 (3.4–4.1)	0.046
LVEDV 2D (ml)	81.1 (73.4–92.0)	91.3 (82.0–103.0)	0.011
LVESV 2D (ml)	39.8 (32.3–46.0)	60.0 (49.2–70.7)	<0.001
EF "Simpson's" (%)	49.7 (45.2–54.0)	35.0 (31.4–43.0)	**<0.001**
WMSI	1.35 (1.12–1.59)	1.94 (1.59–2.18)	**<0.001**
E/E' ratio	8.5 (7.4–10.2)	11.0 (8.0–12.2)	0.031
Speckle tracking echocardiography			
GLS	–18.7 (–20.5 – –15.0)	–12.3 (–14.4 – –10.4)	<0.001
GCS	–21.98 ± 5.809	–15.46 ± 6.616	0.001
3D echocardiography			
LVEDV 3D (ml)	84.9 (74.9–90.8)	89.3 (77.9–99.7)	0.201
LVESV 3D (ml)	43 (35.8-49)	56.8 (47.8–67)	0.001
EF 3D (%)	49.4 (44 -54.3)	36.4 (31.4-42.2)	<0.001
SV 3D (ml)	40.826 ± 5.4614	32.379 ± 5.2768	<0.001

EF – ejection fraction; GCS – global circumferential strain; GLS – global longitudinal strain; LA – left atrium; LVEDD – LV end-diastolic diameter; LVEDV – LV end-diastolic volume; LVESD – LV end-systolic diameter; LVESV – LV end-systolic volume; SV – stroke volume; WMSI – Wall Motion Score Index.
Data are presented as mean ± SD or median and (25.–75. percentile).

flow grade as the only independent predictors of LVR. Although 3D-echo provides a more precise assessment of LV volumes, none of the 3D parameters independently predicted LVR in multivariate analysis, reinforcing the superior prognostic value of GLS. Patients with remodeling also had a significantly higher E/e' ratio, suggesting a relationship between LV filling pressures and adverse remodeling.

Discussion

Prediction of LV adverse remodeling

In the present study, we found that **an average LV peak systolic GLS >–14.8% independently predicted LVR in** STEMI patients treated with PPCI**,** with an AUC of 0.796, sensitivity of 78.95%, and specificity of 82.35%. These findings reinforce the role of GLS as a superior early predictor of post-MI ventricular remodeling compared with conventional echocardiographic markers.

Following STEMI, longitudinal strain is often affected by the dysfunction in the shortening of myocardial longitudinal fibers: the greater the myocardial damage, the more pronounced the impairment in longitudinal strain, resulting in lower GLS values. This explains why GLS is a more sensitive marker of early myocardial dysfunction than the traditional volumetric parameters such as LVEF and LVEDV. Our study underscores this advantage, as has been previously reported by Joyce et al.[36] identified a similar GLS threshold (–14.9%) that was predictive of LVR. However, the cutoff was lower in Bastawy et al.[37] (>–12.5% with 87% sensitivity, and 85% specificity) and Sabry et al.[38] (LVGLS <–9.0% with 77% sensitivity, and 93.2% specificity); probably because they included anterior MI patients only, who tend to have more myocardial damage, and consequently worse LV systolic dysfunction and shape deformity. While early LVEF assessment is a widely accepted tool for estimating myocardial damage,[39] especially during early evaluation after MI, its reliability is limited by myocardial stunning, lack of differentiation between viable and non-viable segments,[19,20] and compensatory hyperkinesis in remote myocardial regions, which may mask underlying dysfunction. In contrast, GLS provides a more objective and reproducible measure of myocardial deformation by differentiating between active myocardial contraction and passive segmental motion.[11,19,20,38] Park et al.[26] first reported that strain strongly predicted LVR and prognosis after MI, particularly anterior MI, and Lacalzada et al.[40] illustrated that GLS could predict LVR in STEMI patients using a cutoff >–9.27%. So, the strain was a better predictor of LVR than WMSI or LVEF which is similar to our finding. In contrast, Mele et al.[41] found that GLS and EF lost their predictive power in multivariate analysis and observed a significant correlation only in severely altered longitudinal strain (r = 0.80, *p* <0.001).

However, in contrast to larger studies such as the VALIANT Echo study,[42] which found that both GLS and GCS were predictive of LVR, our study did not identify GCS as an independent predictor. This disagreement could be due to sample size differences and the inclusion of patients with relatively preserved LVEF (>40%) in the

VALIANT Echo study. Other studies also found no value for GCS in LVR prediction.[27,43]

LVR prevalence

The prevalence of post-infarction LVR varies according to the used definition, which incorporates the magnitude of change in a certain parameter, and the imaging modality. Despite the lack of a consensus definition, the most cited is a 15% to 20% rise in LVEDV.[4,44] In our study, we defined LVR as an LVEDV increase >15% using 3D-echo in the follow-up visit compared with the baseline and found that LVR occurred in 35.8% of patients, which is concordant with many previously published studies.[4–6,45] Similarly, two other 3D-echo studies produced similar LVR prevalence: in one study,[24] post-STEMI LVR was observed in 38% of patients using the definition of >15% increase in LVEDV by 3D-echo after 6 months of the STEMI, and in the other study,[46] the prevalence was 39%. Also, in a CMR study,[41] LVR was defined as a 15% increase in LVEDV, 6 months from the baseline value. However, the CMR cut-off bar for LVEDV and LVESV changes was recently lowered to 12%.[47] By 2D-echo, the standard definition of post-STEMI LVR is ≥20% LVEDV increase from baseline,[48,49] and that of LV reverse remodeling is ≥10% decrease in LVESV,[50] but these cut-offs were first suggested in echocardiographic research before the PPCI era.

Comparison with 3D echocardiography

Although the 3D-echo avoids the limitations of 2D-echo analysis of the volumes, the geometry, and LV function as previously mentioned, and is comparable to CMR, none of the 3D-echo parameters independently predicted LVR in our multivariate analysis, despite LVESV, EF, and SV being significantly associated with LVR in univariate analyses. This might be because volumetric changes in LVR develop gradually, whereas myocardial deformation abnormalities appear sooner in the disease course. GLS, being a functional marker, can detect subclinical dysfunction long before measurable changes in LV volumes occur, making it a valuable tool for early risk stratification.

Other univariate predictors of LVR

In our study, risk factors did not influence the occurrence of LVR, except BMI which was higher in the LVR group. Similarly, Mele et al. found no differences in baseline characteristics between patients with and without LVR[41] and Sabry et al.[38] Anterior STEMI was present in 56.6% of our patients – a similar incidence was found in the study by Rodriguez-Palomares et al. (56.4%).[51] Those anterior STEMI patients had significantly more LVR (p = 0.021), and this LVR was associated with significantly higher maximal ST-segment elevation after PCI (p = 0.007). However, the change in ST-segment amplitude was not significantly different pre- and post-PCI. In contrast, in the study by Tawfik et al.,[52] the anterior STEMI represented 52.9% of all patients, and ST-segment resolution was complete in only 51%.

The total ischemic time in our patients was non-significantly longer in the LVR group (median 6 vs 5 hours). This was in line with Bolognese et al.,[4] Zaliaduonyte- Peksiene et al.,[53] and Barberato et al.[54] that demonstrated that LVR tended to be associated with delayed reperfusion. In contrast, Bastawy et al.[37] showed that the LVR group had significantly longer pain-to-door, door-to-balloon, and total ischemic times. This may be due to more myocardial damage owing to delayed reperfusion and highlights the importance of reducing pain-to-door time through raising public awareness of MI symptoms and door-to-balloon time through improving the healthcare system.

Other associations with LVR in our study included a lesion in the left anterior descending artery (LAD) as the culprit and TIMI flow grade II, the latter was a significant independent predictor in multivariate analysis. Similarly, Zaliaduonyte-Peksien1e et al.[55] stated that LAD and left circumflex as infarct-related arteries were significant predictors of LVR comparing the LV remodeling and non-remodeling groups (p <0.01 & <0.05, respectively), but not the right coronary artery, while Galiuto et al. concluded that TIMI flow < 3 after reperfusion was an independent predictor of LVR (OR: 5.6, p = 0.015).

Age and gender were not confounders –the mean age of our patients was 50.8 ± 10.9 years, and 79.2% were males, which is comparable to the results of Lacalzada et al.,[49] who showed that LVGLS can predict LVR and subsequent cardiac events in STEMI patients treated with PPCI with a mean age of 56 ± 12 years, and 79 % males. In many other similar studies,[38,47,56] male gender was predominant.

Our study showed that patients who developed postinfarction LVR had lower baseline EF and larger 2D LVEDV, and LVESV than those who did not develop LVR. Patients with a larger baseline LVEDV have a higher risk for LVR, as the percent change in LVEDV will be smaller in an already dilated LV before remodeling. In addition, patients with reduced LVEF are more likely to develop progressive LVEDV and LVESV dilation during follow-up. This is comparable to the findings of R. Ola et al.[46] in which the 3D-echo analysis of the LV global and regional contractility revealed that patients who developed postinfarction LVR had a lower EF (48% vs. 51%, p = 0.001), larger EDVs (99.8 ml vs. 87.1 ml, p = 0.037), and a higher 3D sphericity index (0.41 vs 0.31, p <0.001) at baseline than those who did not develop LVR.

WMSI is another feasible and practical method of myocardial assessment that can be used even in patients with poor acoustic windows without post-processing. In the present study, WMSI was significantly higher in remodelers than non-remodelers. This is concordant with the previously published data by Bastawy et al.,[37] Zaliaduonyte-Peksiene et al.,[53] and Mannaerts et al.[46] who reported a statistically significant higher WMSI in the LVR group. This makes sense –the higher the WMSI, the larger the akinetic regions with more muscle loss, and thus the susceptibility to infarct expansion and LVR development.

We also showed that remodelers had a significantly higher E/e' ratio than non-remodelers; this could be related to the dependence of LVEDV on structural remodeling and LV filling pressures –the higher LV filling pressure marked by a higher E/e' ratio, the larger the LVEDV. This was similar to Barberato et al.[54] in which the lateral e' and E/e' ratios were higher in the LVR group (6.9 ± 2 vs 8.5 ± 2 cm/s, p = 0.02, and 13 ± 4 vs 8.5 ± 2, p <0.001 respectively), but only E/e' ratio was an independent predictor of LVR.

Using 2D-STE, we showed that patients with LVR had lower LVGLS and LVGCS, which is concordant with the previously published studies.[40] Similarly, in another study,[38] although the two groups did not differ in the conventional echocardiographic parameters (LV volumes, LVEF, and WMSI), STE revealed that patients with LVR had lower LVGLS and LVGCS (–7.97 ± 3.85 vs –12.8 ± 2.48% and –11.76 ± 6.87 vs -18.35 ± 2.94 respectively, p <0.001).

Strengths

In this study, we used 3D-echo, which is the gold standard technique for quantifying LV volumes and has a better spatial resolution and finer proximity than 2D-echo. Recent advances in 3D-echo allowed comparable accuracy to CMR.[45] By producing a mathematical model of the LV, semi-automated endocardial detection software increases the precision of volume calculations and enables cardiac output and stroke volume measurements.[57] Despite that, none of the 3D parameters independently predicted LVR, reinforcing the early predictive advantage of GLS.

We recognize that there are several definitions for LVR and that results may vary according to the one used. However, the one we used in our study has been used in previous studies[10,46] and has the advantage of being clinically applicable.

Clinical implications

Our results highlight the potential utility of GLS in routine post-STEMI assessment. Unlike advanced imaging modalities such as cardiac MRI, which offer superior tissue characterization but are less accessible, GLS is a bedside tool that is widely available, cost-effective, and reproducible across different clinical settings. Integrating GLS into routine echocardiographic evaluations may facilitate the early identification of patients at high risk of adverse remodeling, enabling prompt initiation of cardioprotective therapies such as beta-blockers, and renin-angiotensin-aldosterone system inhibitors.

Furthermore, given the emerging role of artificial intelligence in echocardiography, automated strain analysis could enhance the clinical application of GLS by improving reproducibility and reducing interobserver variability. Future research should explore the integration of machine learning models with GLS assessment to further refine risk prediction algorithms.

Limitations

Our study has some limitations – it was a single-center experience with a relatively small sample size, which may limit the generalizability of our findings. We used surrogate endpoints rather than hard clinical endpoints such as mortality or hospitalization, which should be addressed in future research. The echocardiographic analysis was done by a single operator, without inter- or intra-observer variability assessment; this may introduce bias and limit reproducibility. Additionally, we defined LVR based on a single follow-up time point at six months, potentially overlooking the dynamic nature of the remodeling process, including early reverse remodeling. A longer follow-up period (12–24 months) would be beneficial to assess the persistence of these findings and their correlation with clinical outcomes. Finally, while our study focused on echocardiographic predictors of remodeling, incorporating additional biomarkers, such as NT-proBNP and high-sensitivity troponins, may provide a more comprehensive risk stratification approach. Further multicenter studies are necessary to validate our findings and study the combined predictive value of GLS and circulating biomarkers. The echocardiographic analysis was done by a single operator without assessing the inter- and intra-observer variability.

Conclusion

GLS measured within the first 48 hours after acute STEMI is a strong independent predictor of LVR at six months. Its ease of use, cost-effectiveness, and superior predictive ability over conventional echocardiographic markers make it a valuable tool for risk stratification in post-STEMI patients. Future research should focus on integrating GLS into standardized post-MI management protocols and exploring its role in guiding personalized therapeutic strategies.

Conflict of interest
None to declare.

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Ethical statement and consent to participate
This study was performed in line with the principles of the Declaration of Helsinki. Approval was granted by the Ethics Committee of Cairo University (Date 25-02-2018). All patients provided their written informed consent to participate in the study.

Consent for publication.
Not applicable.

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