The Egyptian Journal of Hospital Medicine (July 2025) Vol. 100, Page 4658-4666

Myocardial Longitudinal Strain in Prediction of LV Remodeling in Patients with Successfully Reperfused Anterior Wall ST-Segment Elevation Myocardial Infarction

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ABSTRACT

Background: LV remodeling and subsequent heart failure (HF) following acute myocardial infarction (AMI) are linked to unfavorable prognosis.

Objective: The present study was designed to evaluate the predictive value of global longitudinal strain (GLS), assessed using two-dimensional speckle tracking echocardiography (2D STE), for the occurrence of LV remodeling in cases with acute anterior wall ST-segment elevation myocardial infarction (ant-STEMI).

Subjects and methods: This investigation included 57 cases diagnosed with ant-STEMI who underwent successful primary coronary intervention and had two-dimensional speckle tracking echocardiography (2D STE) data, in addition to standard transthoracic two-dimensional echocardiographic assessment. All participants were prospectively followed for a period of six months, after which transthoracic echocardiographic evaluation was repeated.

Results: At the 6-month follow-up, left ventricular (LV) volumes and ejection fraction (EF) were reassessed. LV remodeling was defined as an increase in LV end-diastolic volume (EDV) of ≥ 20% compared to baseline measurements. Remodeling occurred in 12 cases (21.05%), while the remaining 45 cases (78.9%) exhibited no evidence of remodeling. Multivariate analysis of baseline echocardiographic parameters revealed no substantial variations between the two groups in LV end-diastolic diameter (LVEDD), LV end-systolic volume (LV ESV), or LV EDV. In contrast, the LV remodeling group demonstrated a statistically significant increase in LV end-systolic diameter (LV ESD) and a significantly lower EF. Moreover, this group exhibited markedly higher LV peak systolic GLS values, with the optimal cut-off identified as > –12.8 (sensitivity 91.6% & specificity 93.4%). Wall motion score index (WMSI) was also substantially elevated in remodeling group, with the best cut-off determined as > 1.59 (sensitivity 92.5% & specificity 88.3%). Independent predictors of LV remodeling were WMSI > 1.59, GLS > –12.5%, and total ischemic time.

Conclusion: GLS represents a robust echocardiographic indicator associated with the occurrence of LV remodeling at 6 months in cases with ant-STEMI who underwent successful reperfusion.

Keywords: Anterior wall myocardial infarction, Echocardiography, Strain, LV remodeling.

INTRODUCTION

Despite notable advancements in prognosis over the past decade, AMI continues to represent a major global contributor to morbidity and mortality. This improvement in clinical outcomes has been driven by several pivotal developments, including enhanced risk stratification, broader adoption of invasive management strategies, establishment of care pathways emphasizing prompt revascularization via PCI or thrombolytic therapy, significant progress in antiplatelet and anticoagulant pharmacotherapy, and increased implementation of secondary prevention measures such as statin therapy [(1)].

Systolic function of the LV arises from the synchronized contributions of circumferential shortening, radial wall thickening, and longitudinal contraction. Strain and strain rate have recently been established as sensitive diagnostic modalities for assessing ventricular performance following AMI [(2)].

Multiple studies have highlighted GLS as a more sensitive and reliable marker than LVEF for predicting cardiac events, assessing functional recovery, and identifying irreversible remodeling following AMI [(3)]. So, this study aimed to investigate the importance of GLS in predicting LV remodeling at six months in cases with successfully reperfused anterior Wall STEMI.

PATIENTS AND METHODS

The present study enrolled 57 cases who presented to the Cardiology Department at Benha University Hospital between November 2023 and November 2024 with a first episode of acute ant-STEMI and underwent primary PCI. All participants were subsequently followed for a duration of six months.

Inclusion criteria: Cases experiencing a first episode of anterior STEMI who underwent primary PCI with successful epicardial reperfusion and were confirmed by achieving TIMI grade III flow.

Exclusion criteria: History of prior CABG or PCI, the presence of another significant coronary lesion beyond

Received: 25/06/2025
Accepted: 29/08/2025

the LAD, post-reperfusion TIMI flow less than grade III and inadequate echogenic window, or if they underwent revascularization during the follow-up period.

Assessments*:*

Medical history assessment: History taking with emphasized personal data and risk factors for IHD, including HTN, DM, smoking status, dyslipidemia, and a positive family history. Clinical evaluation documented presenting symptoms and signs such as arterial blood pressure (ABP), HR, and Killip classification at admission, along with the presence of a third heart sound (S3), pulmonary congestion, mitral regurgitation (MR), and ventricular septal rupture on local examination.

Electrocardiograms (ECG): A 12-lead surface ECG was obtained within the first 10 minutes of presentation, repeated 90 minutes after catheterization, performed daily thereafter, and once more immediately prior to discharge.

Laboratory investigations: On admission, venous blood was obtained from each case for cardiac biomarker assessment, and additional results from standard laboratory investigations carried out at the same time were documented.

Coronary angiography and intervention procedural details:

Coronary intervention was carried out using either a transradial or transfemoral approach. All cases received an IV bolus of unfractionated heparin at a dose of 100 IU/kg prior to PCI to the LAD. Successful PCI will be defined by the achievement of TIMI grade 3 flow.

Echocardiographicy:

(A) Standard transthoracic 2D echocardiography was performed using a Vivid E9 Ultrasound System, adhering to established acquisition guidelines. Cases were examined in the left lateral decubitus position, with imaging obtained in parasternal long- and short-axis as well as apical four- and two-chamber views. The following measurements were documented:

- LV end-diastolic volume (EDV), LV end-systolic volume (ESV), and LVEF by biplane Simpson's method.
- E and A waves with the E/A ratio by Doppler.
- WMSI based on 16-segment LV stratification.

Segmental wall motion was evaluated using a semi-quantitative scale: (1) normal, (2) hypokinesia, (3) akinesia, and (4) dyskinesia. WMSI was derived by dividing the total segmental scores by the number of segments, as described by **Broderick *et al.*** [(4)].

(B) LV peak systolic GLS by 2D speckle tracking:

LV peak systolic GLS was measured using automated function imaging (AFI), a technique based on 2D longitudinal strain imaging. Longitudinal strain (%) was calculated as: longitudinal strain (%) = [L (end-systole) – L (end-diastole)]/L (end-diastole) x 100%; where L is the length of the RoI [(5)].

(C) Six-month echocardiographic follow-up: At 6 months, all cases underwent echocardiographic assessment of LV ESV, LV EDV, and LVEF. LV remodeling was defined as a ≥ 20% increase in LVEDV compared with baseline values [(6)].

Medications: Therapy began during admission and continued after discharge. Aspirin was prescribed indefinitely, clopidogrel 75 mg daily for 12 months, and β-blockers and ACEIs were started within 24 hours and titrated as tolerated. An aldosterone antagonist was added for patients with EF ≤ 40% and either HF or diabetes already on ACEI and β-blocker, unless contraindicated. High-intensity statins were prescribed in all eligible cases.

Ethical consideration: This investigation was conducted following approval from the IRB of the Faculty of Medicine, Benha University (Approval Code: MD 1.11.2023). The Helsinki Declaration was followed throughout the course of the investigation.

Statistical analysis

Statistical analyses were conducted with SPSS version 27 (IBM Corp., Armonk, NY, USA). ROC curve analysis was utilized to determine diagnostic accuracy, with the AUC representing the performance metric. AUC values above 50% were regarded as indicative of acceptable discrimination, whereas values closer to 100% reflected optimal accuracy. Specificity, sensitivity, PPV, and NPV were also estimated. Logistic regression models were applied to explore associations between outcome variables and potential predictors, using univariate and multivariate approaches. Normality was assessed by Shapiro–Wilk test and histogram review. Parametric variables were summarized as mean ± SD and compared with Student's t-test. Non-parametric variables were presented as median (IQR) and analyzed using the Mann–Whitney U test. Categorical data were expressed as frequency (%) and compared by Chi-square or Fisher's exact test. Significance was defined as $p \leq 0.05$ (two-tailed).

RESULTS

Of the 93 cases initially enrolled, 36 were excluded from evaluation of LV remodeling for the following reasons: eight had suboptimal echogenic image quality, ten required revascularization procedures during the six-month follow-up, sixteen exhibited post-reperfusion TIMI flow less than grade III, and two cases died. The final cohort consisted of 57 cases with a first episode of acute ant-STEMI who underwent primary PCI to the LAD, with no substantial disease in other coronary arteries.

All cases had follow-up echocardiography at six months. The analysis was restricted to cases who achieved successful epicardial reperfusion, defined as TIMI grade 3 flow post-reperfusion. These cases were categorized into two groups based on the occurrence of LV remodeling: Group I comprised 45 cases who did not develop LV remodeling, whereas group II consisted of 12 cases in whom LV remodeling occurred.

Cases demographics and personal data:

Both groups were comparable in age, with a mean of 51.06 ± 5.56 years in the LV non-remodeling group and 50.83 ± 5.90 years in the LV remodeling group. They were also matched for gender distribution, with males comprising 88.9% of the LV non-remodeling group and 83.3% of LV remodeling group. As presented in table (1), these differences were not statistically significant between groups.

Table (1): Age and gender distribution between both groups

		No-remodeling	remodeling	t/ X2	P
		45	12		
Age		51.06±5.56	50.83±5.90	0.127	0.899
Sex	F	5 (11.1%)	2(16.7%)	0.27	0.602
	M	40 (88.9%)	10 (83.3%)		

Risk factors and previous medical history between studied groups:

In the LV non-remodeling group, 17 cases (37.8%) were hypertensive, 15 (33.3%) had diabetes mellitus, 10 (22.2%) had dyslipidemia, 29 (64.4%) were smokers, and 8 (17.8%) reported a positive family history of premature CAD. In the LV remodeling group, 3 cases (25%) were hypertensive, 6 (50%) had DM, 2 (16.7%) had dyslipidemia, 8 (66.7%) were smokers, and 2 (16.7%) had a positive family history of premature CAD.

As summarized in table (2), the distribution of CAD risk factors did not differ statistically between LV non-remodeling and LV remodeling groups.

Table (2): Risk factors of CAD distribution between both groups

		No remodeling	Re-modeling	t/ X2	P
		45	12		
Hypertension	-VE	28 (62.2%)	9 (75.0%)	0.67	0.41
	+VE	17 (37.8%)	3 (25.0%)		
DM	-VE	30 (66.7%)	6 (50.0%)	1.13	0.28
	+VE	15 (33.3%)	6 (50.0%)		
Dyslipidemia	-VE	35 (77.8%)	10 (83.3%)	0.17	0.67
	+VE	10 (22.2%)	2 (16.7%)		
Smoker	-VE	16 (35.6%)	4 (33.3%)	0.02	0.88
	+VE	29 (64.4%)	8 (66.7%)		
Family History	-VE	37 (82.2%)	10 (83.3%)	0.008	0.92
	+VE	8 (17.8%)	2 (16.7%)		

Pain to door time between studied groups: Analysis of pain-to-door time revealed a highly substantial variation between groups, with longer delays in the LV remodeling cohort. Mean pain-to-door time was 3.39 ± 0.96 hours in the non-remodeling group versus 4.90 ± 1.42 hours in the remodeling group. As shown in table (3). Prolonged pain-to-door intervals were associated with a greater likelihood of LV remodeling.

Table (3): Comparing both groups for pain to door time

	No remodeling	**Remodeling**	**t**	**P**
Pain to door hours	**3.39 ± 0.96**	**4.90 ± 1.42**	**4.341**	**0.00****

Clinical examination and LAB data between studied groups: As shown in table (4), Killip class distribution did not differ substantially between groups; 41 cases (91.1%) in the LV non-remodeling group were Killip class I and 4 (8.9%) were class II, while all 12 cases (100%) in the LV remodeling group were class I. Mean SBP and DBP were slightly higher in the non-remodeling group (121.11 ± 7.44 mmHg and 74.86 ± 8.44 mmHg respectively) compared to the remodeling group (116.33 ± 8.23 mmHg and 72.16 ± 9.20 mmHg respectively), though the differences were not statistically significant.

In contrast, discharge HR was significantly elevated in the remodeling group (79.25 ± 5.24 vs. 75.0 ± 2.16 beats/min).

Table (4): Comparing both groups for clinical examination

		No remodeling	Remodeling	t	P
		45	12		
SBP		121.11±7.44	116.33±8.23	1.732	0.058
DBP		74.86±8.44	72.16±9.20	1.651	0.036
Discharge HR		75.0±2.16	79.25±5.24	3.561	0.001**
KILLIP class	I	41(91.1%)	12(100.0%)	1.14	0.28
	II	4(8.9%)	0(0.0%)		
	III	0(0.0%)	0(0.0%)		
	IV	0(0.0%)	0(0.0%)		

Laboratory parameters showed no significant intergroup differences (Table 5).

Table (5): LAB data distribution between both groups

	No remodeling	Remodeling	t	P
Creatinine (mg/dl)	0.98±0.30	0.87±0.12	1.248	0.217
HB(g/dl)	12.68±1.31	12.95±1.98	0.578	0.565
LDL(mg/dl)	132.48±8.65	129.91±5.63	0.972	0.335
HDL(mg/dl)	38.6±3.72	37.33±3.20	1.075	0.287
TG(mg/dl)	127.28±42.63	115.75±22.52	1.497	0.184
Cholesterol (mg/dl)	208.0±18.47	209.25±20.77	0.203	0.840

Coronary angiographic data between both groups:

As shown in table (6), regarding LV non-remodeling group the site of lesion was osteal LAD in 3 cases (6.7%), proximal LAD in 16 cases (35.6%), mid LAD in 25 cases (55.6%) and distal LAD in 1 case (2.2%). In the LV remodeling group, the site of lesion was proximal LAD in 7 cases (58.3 %), and mid LAD in 5 cases (41.7 %) and these variations were non substantial between both groups. Regarding thrombus grading, there was a non substantial variation between both groups being of higher grade in LV remodeling group.

In the LV non-remodeling group, 2 cases had thrombus grade 3 (4.4%), 9 cases had thrombus grade 4 (20%) and 34 cases had thrombus grade 5 (75.6%). In the LV remodeling group, 12 cases had thrombus grade 5 (100%). Myocardial blush grade (MBG) analysis revealed a statistically highly substantial variation between the two groups, as presented in table (6). In the LV non-remodeling group, MBG was grade 1 in 3 cases (6.7%), grade 2 in 12 cases (26.7%) and grade 3 in 30 cases (66.6%). Conversely, in the LV remodeling group, 4 cases (33.3%) had MBG grade 1 and 8 cases (66.7%) had MBG grade 2, with no cases achieving grade 3.

Table (6): Coronary angiographic data distribution between both groups

		Remodeling		X2	P
		-VE	+VE		
		45	12		
LAD Site	ostial	3(6.7%)	0(0.0%)	2.6	0.45
	Proximal	16(35.6%)	7(58.3%)		
	Mid	25(55.6%)	5(41.7%)		
	Distal	1(2.2%)	0(0.0%)		
Thrombus grade	III	2(4.4%)	0(0.0%)	3.63	0.16
	IV	9(20.0%)	0(0.0%)		
	V	34(75.6%)	12(100.0%)		
MBG	I	3(6.7%)	4(33.3%)	17.8	0.00**
	I	12(26.7%)	8(66.7%)		
	II	30(66.6%)	0(0.0%)		

Baseline Echocardiographic data between studied groups:

As shown in table (7), analysis of the transmitral inflow pattern revealed no substantial variations between both groups in mitral E wave, mitral A wave, or the E/A ratio. In the LV non-remodeling group, the mitral E wave was 79.59 ± 9.75 cm/s, the mitral A wave was 84.14 ± 14.71 cm/s, and the E/A ratio was 1.12 ± 0.25. In the LV remodeling group, corresponding values were 82.15 ± 11.31 cm/s for the mitral E wave, 80.22 ± 21.24 cm/s for the mitral A wave, and 1.19 ± 0.30 for the E/A ratio.

Similarly, there was no substantial variation in the distribution of LV diastolic dysfunction grades between the groups. In the LV non-remodeling group, 12 cases (26.7%) had normal diastolic function, 20 (44.4%) had an impaired relaxation pattern, and 13 (28.9%) exhibited a pseudo-normal pattern. In the LV remodeling group, 1 case (8.3%) had normal diastolic function, 4 (33.3%) had an impaired relaxation pattern, 6 (50%) exhibited a pseudo-normal pattern, and 1 (8.3%) had a restrictive pattern. In contrast, WMSI showed a highly substantial variation between groups, being higher in LV remodeling group.

Table (7): Comparing both groups for baseline echocardiographic data

		No remodeling	Remodeling	t	P
		45	12		
EF		49.04±4.34	37.50±3.60	8.448	0.00**
LVEDD		50.24±2.89	50.41±2.10	0.192	0.848
LVESD		35.02±3.2	40.59±3.46	5.262	0.00**
LVEDV		103.35± 17.30	99.75± 12.33	0.675	0.503
LVESV		53.01±8.94	61.31±9.40	1.6	0.2
MITRAL_E		79.59±9.75	82.15±11.31	0.783	0.437
MITRAL_A		84.14±14.71	80.22±21.24	0.745	0.460
E/A ratio		1.12±0.25	1.19±0.30	1.261	0.128
Grade of Diastolic dysfunction	Normal	12 (26.7%)	1 (8.3%)	6.7 Chi square	0.082
	I	20 (44.4%)	4 (33.3%)		
	II	13 (28.9%)	6 (50%)		
	III	0	1 (8.3%)		
WMSI		1.45±0.12	1.69±0.55	2.690	0.009*
GLS		-15.2o±1.70	-11.o2±1.44	7.758	0.00**

In the LV non-remodeling group, the WMSI was 1.45 ± 0.12. Whereas in the LV remodeling group, it was 1.69 ± 0.55. As presented in table (8) and figure (1), the optimal cut-off value for WMSI in predicting LV remodeling was > 1.59, yielding a sensitivity of 92.5%, specificity of 88.3%, PPV of 64.7%, and NPV of 97.5%. For LV diameters, LVEDD did not differ markedly between groups, whereas LVESD was greater in the remodeling cohort. LVEDD and LVESD measured 50.24 ± 2.89 mm and 35.02 ± 3.20 mm respectively in the non-remodeling group versus 50.41 ± 2.10 mm and 40.59 ± 3.46 mm respectively in the remodeling group.

With respect to volumes, LVEDV was comparable between groups, while LVESV was significantly larger in the remodeling group (103.35 ± 17.30 ml and 53.01 ± 8.94 ml vs. 99.75 ± 12.33 ml and 61.31 ± 9.40 ml). Biplane Simpson's LVEF showed a marked difference, being higher in non-remodeling group (49.04 ± 4.34%) than in the remodeling group (37.50 ± 3.60%). Also, mean LV peak systolic GLS was substantially reduced in the non-remodeling group relative to the remodeling group. In the LV non-remodeling group, average LV peak systolic GLS was 15.20 ±1.70. In LV remodeling group, average LV peak systolic GLS was -11.02 ± 1.44.

Table (8): Best cut off value of WMSI

Area	Cut off value	P	Sensitivity	Specificity	PPV	NPV
0.910	>1.59	0.00**	**91.7%**	**86.7%**	**64.7%**	**97.5%**

As shown in table (9) and figure (2), the best cut-off value for average LV peak systolic GLS was >-12.8, with specificity of 93.4%, sensitivity of 91.6%, PPV of 78.5% and NPV of 97.6%. Regarding grade of MR, there was substantial variation between both groups, being of higher grade in LV remodeling group. In the LV non-remodeling group, 30 cases did not have MR (6.67%) and 15 cases had mild MR (33.3%). In LV remodeling group, 4 cases did not have MR (33.3%) and 8 cases had mild MR (66.7%).

Table (9): Best cut of value for average LV peak systolic GLS

Area	Cut off value	P	Sensitivity	Specificity	PPV	NPV
0.978	>-12.8	0.00**	91.7%	93.3%	78.5%	97.6%

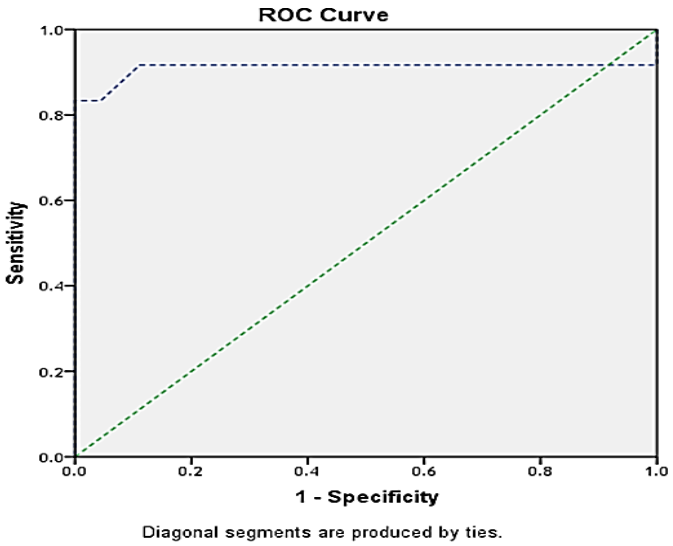

Figure (1): Best cut off value of WMSI

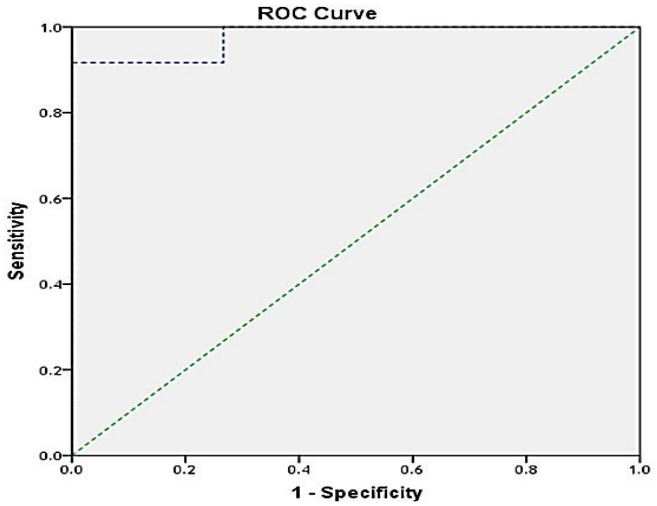

Figure (2): Best cut of value for average LV peak systolic GLS

Independent predictors of LV remodeling after AMI: Multivariate logistic regression analysis was performed to identify predictors of LV remodeling at six months following AMI (Table 10). Three variables emerged as independent predictors: a WMSI >1.59 (p = 0.002; OR 7.369, 95% CI 2.654–11.369), prolonged pain-to-door time (p = 0.002; OR 7.645, 95% CI 3.212–11.632), and GLS > −12.8% (p < 0.001; OR 10.984, 95% CI 3.745–14.323). These findings highlighted the prognostic significance of both clinical and ECG parameters in anticipating adverse LV remodeling.

Table (10): Independent predictors of LV remodeling after AMI

	Wald	P	A oR	95% C.I.	
				Lower	Upper
Discharge HR	1.557	0.276	2.522	0.865	12.417
Pain to _door hours	3.425	0.002*	7.645	3.212	11.632
EF	2.117	0.107	3.658	0.742	17.365
LVESD	1.632	0.223	2.992	0.632	9.687
LVESV	1.582	0.264	2.162	0.625	12.361
EA/ratio	1.784	0.121	2.804	0.875	29.635
GLS	4.487	0.00**	10.984	3.745	14.323
WMSI	3.214	0.002*	7.369	2.654	11.369
MBG	1.974	0.125	2.071	0.923	16.578
MR	2.135	0.074	1.473	0.745	18.574

Six months follow up comparison of LV volumes and LVEF between both groups: At six months, comparison of LV volumes revealed significantly larger dimensions in cases with remodeling. Mean LVEDV and LVESV were 125.36 ± 15.2 ml and 81.09 ± 15.25 ml respectively in the remodeling group compared to 106.0 ± 18.01 ml and 53.72 ± 7.79 ml respectively in the non-remodeling group. Similarly, systolic function assessed by the biplane Simpson's method showed a substantial reduction in remodeling cases, with mean LVEF of 35.08 ± 3.91% compared to 50.11 ± 4.51% in the non-remodeling cohort (Table 11).

Table (11): 6 months follow up comparison of LV volumes and LVEF between both groups

	No remodeling	Remodeling	t	P
LVEDV	106.0±18.01	125.36±15.2	3.410	0.001**
LVESV	53.72±7.79	81.09±15.25	8.639	0.00**
LVEF	50.11±4.51	35.08±3.91	10.510	0.00**

DISCUSSION

STEMI is the most critical manifestation of atherosclerotic CAD and usually results from complete coronary occlusion. Primary PCI is the preferred reperfusion strategy when performed promptly by an experienced team. LV remodeling following AMI is a strong predictor of mortality and a harbinger of clinical HF. In this study, remodeling developed in 21.1% of cases despite successful reperfusion with TIMI 3 flow. These results are consistent with **Bochenek *et al.*** [7] who reported LV remodeling in 42% of anterior STEMI cases. Likewise, **Hamdan *et al.*** [8] observed remodeling in 34.6% of cases with TIMI 3 flow and 17.6% with MBG 2–3, emphasizing the importance of myocardial perfusion beyond epicardial reperfusion.

CAD risk factors and LV remodeling: The remodeling and non-remodeling groups were similar with respect to age, sex, and CAD risk factors. Smoking and hypertension showed no association with remodeling. This is consistent with prior observations by **Symons *et al.*** [9] and **Parodi *et al.*** [10]. Diabetes was likewise not linked to remodeling, which is in agreement with **Lamblin *et al.*** [11] who found it predictive of HF but not of remodeling.

Pain to door time and LV remodeling: In this study, pain-to-door and total ischemic times were substantially longer in the LV remodeling group. This likely reflects greater myocardial injury from delayed reperfusion, highlighting the importance of minimizing both case-related delays through public awareness of AMI symptoms and system-related delays through optimizing healthcare response. Our results align with those of **Bolognese *et al.*** [12], **Zaliaduonyte-Peksiene *et al.*** [13] and **Barberato *et al.*** [14], who described a non-significant trend toward prolonged reperfusion times among cases who developed LV remodeling.

Clinical examination and LV remodeling: In this investigation, discharge HR was substantially lower in the LV non-remodeling group. This observation is consistent with **Joyce *et al.*** [15], who identified discharge HR as an independent predictor of adverse remodeling in a cohort of 964 STEMI cases.

Coronary angiographic data and LV remodeling: Proximal LAD occlusion was more frequent in remodeling cases, whereas mid-LAD occlusion predominated in non-remodeling cases, which is consistent with the larger infarct size typically associated with proximal LAD lesions. **Masci *et al.*** [16] confirmed that anterior AMI is linked to greater infarct size and worse LV function than in non-anterior infarctions. Myocardial blush grade (MBG) differed significantly between groups, being lower in cases with remodeling. Remodeling was observed in 54.7% of MBG 0–1 cases compared to 16% of MBG 2–3 cases. These findings align with **Hamdan *et al.*** [8] who demonstrated lower remodeling rates in cases achieving successful myocardial reperfusion (MBG 2–3) than in those without reperfusion (MBG 0–1) (17.6% vs. 66.6%, p = 0.012).

Echocardiographic data and LV remodeling: LVEDV was comparable between groups without statistical significance, whereas LVESV was higher in the remodeling group but not substantial. LVEF by Simpson's method was significantly reduced in remodeling cases. These findings concur with **Bolognese *et al.*** [12] and **Zaliaduonyte-Peksiene *et al.*** [13].

WMSI was higher in the remodeling cohort, with the variation reaching statistical significance. On multivariate regression, WMSI >1.59 independently predicted remodeling (p = 0.002; OR 7.369, 95% CI 2.654–11.369). The best cut off value for WMSI was found to be 1.59 with sensitivity of 92.5%, specificity of

88.3%, PPV of 64.7% and NPV of 97.5%. This aligns with **Zaliaduonyte-Peksiene *et al.* [13]**, **Mannerts *et al.* [18]** , and **Galiuto *et al.* [19]** who found higher WMSI to be associated with larger akinetic areas, greater myocardial loss, and increased susceptibility to expansion and remodeling.

Average peak systolic GLS was markedly higher in LV remodeling group. Multivariate analysis confirmed GLS > −12.8% as an independent predictor of remodeling ($p < 0.001$; OR 10.984, 95% CI 3.745–14.323). The optimal cut-off value for GLS was > −12.8, yielding sensitivity of 91.6%, specificity of 93.4%, PPV of 78.5%, and NPV of 97.6%. These results are consistent with **Bochenek *et al.* [7]**, **Zaliaduonyte-Peksiene *et al.* [13]**, and **Joyce *et al.* [15]** who similarly identified GLS as an independent predictor with comparable cut-off values.

In the present study, three independent predictors of LV remodeling were identified. A WMSI >1.59 was significantly associated with remodeling ($p = 0.002$; OR 7.369, 95% CI: 2.654–11.369). Prolonged pain-to-door time also emerged as an independent predictor ($p = 0.002$; OR 7.645, 95% CI: 3.212–11.632). In addition, a GLS value > –12.8% demonstrated the strongest association with remodeling ($p < 0.001$; OR 10.984, 95% CI: 3.745–14.323).

LIMITATIONS

This study was conducted at a single medical center with a relatively small sample size of 57 cases. The analysis was limited to cases with single-vessel disease that involved LAD occlusion and those who achieved TIMI III flow following reperfusion. Cases presented in Killip class IV (cardiogenic shock) were excluded, as bedside measurement of LV peak systolic GLS was not feasible. Furthermore, the investigation focused exclusively on LV remodeling at 6 months post-MI and did not address early remodeling occurring in the immediate period after infarction.

CONCLUSION

Average peak systolic GLS measured early after myocardial infarction was an independent predictor of LV remodeling following anterior STEMI and may be used to anticipate its occurrence.

Conflict of interest: None.
Funding: None.

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