The Impact of Different Revascularization Strategies Implemented in Acute Myocardial Infarction on the Recovery of Left Ventricular Functional and Deformational Parameters

Abstract

Background: Despite well-established therapeutic techniques, such as direct revascularization through percutaneous coronary intervention (PCI), acute myocardial infarction (AMI) remains a leading cause of mortality and morbidity.

Objectives: In order to determine if two-dimensional speckle tracking echocardiography (STE) deformation parameters and the early recovery of left ventricular (LV) functions are affected by the timing of PCI in AMI.

Methods: A total of 200 cases with newly-onset acute myocardial infarction (AMI) who had a baseline left ventricular ejection fraction (LVEF) higher than 40% and received effective therapy with percutaneous coronary intervention (PCI) were included in this investigation. cases were categorized as either ST-elevation myocardial infarction (STEMI) or non-ST-elevation myocardial infarction (NSTEMI). cases were grouped into four groups according to the time between presentation and PCI. Using standard echocardiography and two-dimensional (2D) ST-elevation STE, individuals were re-evaluated three months later to find out if remodeling had taken place or if the LV function had returned.

Results: Of the 200 AMI patients, including 140 males (70%), improvement in global longitudinal strain (GLS) and harmed longitudinal strain (HLS) were better in STEMI and NSTEMI patients received urgent revascularization with PCI (groups I and III) versus patients with

pharmacoinvasive strategy or routine invasive strategy (Groups II and IV) ($P < 0.05$) while there was an insignificant difference between group I and III ($P = 0.79$). Of the 200 patients, 47 patients (23.5%) presented signs of LV remodeling at 3 months follow up. Age, smoking history, hypertension, dyslipidemia, Killip class, peak creatine phosphokinase - MB level, baseline left ventricular end diastolic volume (LVEDV), HLS, and harmed longitudinal strain rate (HLSR) were all factors that were found to be significantly associated with left ventricular remodeling ($P<0.05$) in the univariate logistic regression analysis. The following factors were identified as independent predictors of left ventricular remodeling in multivariate logistic regression analysis: damaged left ventricular ejection fraction (EF) and end-systolic volume, peak troponin I, Killip class, culprit left anterior descending (LAD), 2 and 3-vessel coronary artery disease (CAD), and wall motion score index (WMSI).

Conclusion: Earlier PCI in AMI helps earlier improvement in myocardial strain parameters. HLS and HLSR are excellent predictors for LV remodeling and may do better than global parameters.

Key Words: Speckle tracking echocardiography – LV Remodeling – Longitudinal strain – Myocardial infarction – PCI – Revascularization

Introduction

Despite the availability of advanced therapeutic methods, such as direct revascularization with PCI, AMI continues to rank as a top cause of death and morbidity globally **(1)**. Early PCI aims at achieving the approved benefit of lower morbidity and mortality and providing better prolonged outcomes. It is still possible to mostly abort the infarction when the time from pain to PCI is minimized to (< 3 to 4 hours) **(2)**.

Harmful LV remodeling still occurs in AMI patients even after revascularization by PCI, with an approximate incidence of around 30% **(3)**. Ventricular remodeling is a strong indicator of heart failure, which is why it is assigned a negative prognostic significance **(4)**. Reportedly, LV remodeling and clinical outcomes can be better predicted using the LVEF as evaluated by conventional echocardiography and WMSI **(5)**. Although left ventricular ejection fraction (LVEF) is a confirmed independent predictor of acute myocardial infarction (AMI) outcome, research on the recovery of LV function after PCI in cases with reduced LVEF is limited **(6)**.

The two-dimensional STE (2D-STE) methods of measuring LV strain and strain rate (SR) are more recent and more effective techniques for estimating myocardial performance after AMI. These methods are capable of highlighting minor changes in LV function, particularly in cases where LVEF is preserved or midrange. **(7) (8) (9)**.

This investigation set out to determine the impact of the time of PCI in AMI and the rate of left ventricular function recovery from revascularization, through different protocols adopted in acute STEMI and NSTEMI patients (either primary versus pharmaco-invasive in STEMI patients or early invasive versus routine invasive in NSTEMI patients) and to assess the parameters linked to left ventricular remodeling after an acute myocardial infarction (AMI) and a preserved or midrange

left ventricular ejection fraction (LVEF) using clinical, biochemical, echocardiographic, and angiographic aspects.

Patients and Methods

Study Design:

This prospective cohort investigation was carried out at a single center from October 2022 to December 2023 and involved 200 cases who were transported to the hospital for PCI after presenting with new-onset AMI to the emergency department at Benha University Hospital within 12 hours of the beginning of chest pain. Both the baseline and three-month follow-up echocardiograms were performed within two or three days after PCI.

Study Population:

We have initially enrolled 246 patients who had AMI (STEMI and NSTEMI) within 12 hours of chest pain onset of whom 46 patients have dropped out during follow up and not evaluated for remodeling either due to poor echogenicity, recurrence of ischemia, need for revascularization, missed follow up or completion of the predefined initial group. eventually, 200 patients were enrolled, with an equal distribution among the four categories.:

- **Group I (N=50):** Cases with acute STEMI who given urgent reperfusion with primary PCI.
- **Group II (N=50):** Cases with acute STEMI who were given fibrinolysis with streptokinase and then underwent invasive PCI within 3-24 hours (The Pharmaco-invasive strategy).
- **Group III (N=50):** Cases with NSTEMI to whom an early intrusive approach was administered (PCI within 24 hours of onset of chest pain).
- **Group IV (N=50):** Cases with NSTEMI who were managed with an invasive strategy (PCI within 24-72 hours of onset of chest pain).

DM and HTN were both assessed in all cases. **(10) (11)**. All of them were found to have no prior history of cerebrovascular or cardiovascular disorders. Baseline data was retrieved from the patient's medical record: gender, age, physical exam findings, laboratory data, 12-lead resting electrocardiogram (ECG), coronary angiography, and cardiovascular risk factors. All study subjects had a normal sinus rhythm and no known history of atrial fibrillation. Prior to inclusion in the investigation, all patients had transthoracic echocardiography performed to evaluate LV function, mass, and significant valvular anomalies.

We have excluded cases presenting late after 12 hours of chest pain, those with failed fibrinolysis, failed PCI or referred for coronary artery bypass graft surgery (CABG), cases suffering from cardiogenic shock, LVEF <35% after PCI, patients with poor image quality for STE and those who refuse to participate into the study.

The diagnosis of STEMI and NSTEMI was predicated on the presence of typical angina for a duration exceeding twenty minutes.; ECG interpretation upon presentation and elevated cardiac biomarkers as per guidelines **(1)** (**2) (12)**. Based on the clinical examination conducted upon presentation, the AMI cases were assigned to one of the Killip classes. **(13)**.

Biochemical evaluations that include creatine phosphokinase MB isoenzymes (CK-MB), a renal profile, a complete blood count, Troponin I. To find the estimated glomerular filtration rate (eGFR), the CKD-EPI equation was used. **(14)**.

PCI procedure

It was done through transfemoral or transradial approach as per standard techniques. For every patient, the following information was documented: (A) Culprit vessel (B) Number of diseased vessels (C) TIMI Grade Flow Previous to and following the procedure: during PCI, values of coronary blood flow range from 0 to 3 **(15)**.

The presence of left main coronary artery stenosis greater than 50% and left circumflex, right coronary, or left anterior descending artery stenosis greater than 70% were criteria for severe coronary stenosis. If the remaining stenosis was less than 30% and the culprit vessel's flow was grade 2 or 3, as measured by the TIMI flow score, a percutaneous coronary intervention (PCI) was considered successful **(16)**. Complete revascularization was attempted during index procedure or planned as staged PCI according to clinical situation and current recommendations **(17) (18)**.

Conventional echocardiography:

With the use of the S5-1 probe and a Philips EPIQ 7C machine, we conducted thorough transthoracic echocardiographic assessments while simultaneously recording ECG data. To conduct the examination, the subjects were put in the left lateral decubitus position. Doppler pictures were obtained in 2D, color, pulsed-wave, and continuous-wave mode. We obtained and documented all echocardiographic assessments offline. We followed the guidelines while measuring the diameters and wall thicknesses of the LVs. Following the prescribed procedure, LVEF was evaluated using modified biplane Simpson's approach. **(19)**.

The 17-segment model that was suggested was utilized to assess the motion of the walls in the region. There were seventeen sections made of LV. Every section is given a score that indicates whether it is normal (1), hypokinesia (2), akinesia (3), dyskinesia (4), or aneurysmal (5). To get the WMSI, we divided all of the scores by 16. To find the WMSI, we divided the total by 16 (after dropping the apical cap). Abnormalities were characterized as WMSI > 1. By indexing the left atrial volume, which was acquired from a biplane calculation to the body surface area, the left atrial volume index (LAVI) was computed **(19)**.

In addition to the standard 2D imaging, M-mode, pulsed and continuous Doppler flow measurements across various cardiac valves and Doppler tissue imaging (DTI) were obtained,

following the recommendations of the American Society of Echocardiography. The septal and lateral mitral annuli were used to quantify the DTI velocities, which include e′ (early diastole) and a′ (late diastole). The averages of these velocities were then determined. Standard criteria were used to categorize patients into 4 categories based on diastolic function **(19) (20)**.

Two-dimensional speckle tracking echocardiography:

The apical four-chamber (A4CH), apical two-chamber (A2CH), and long axis views were employed to acquire three end-expiratory cardiac cycles in sequence at a frame rate of (60-80 frames/sec). harmonic imaging. The 2D-STE analysis was carried out without an internet connection using these views' grayscale LV pictures. During end-systole, the endocardial border was manually traced, and the software monitored the myocardial region of interest automatically. Following optimization of the regions of interest, strain curves for various myocardial parts are automatically generated by the software. Through the basal, mid, and apical antero-lateral wall parts, as seen from the A4CH perspective, LS and longitudinal SR (LSR) were evaluated. Looking at it from the A2CH view, it passes through the following segments: basal, mid, apical inferior wall, basal, mid, to apical anterior wall; and looking at it from the apical long axis perspective, it passes through the following segments: basal, mid, apical infero-lateral wall, basal, mid, and apical anterior septal segments. We measured LS and LSR for every segment. To create a single bull's-eye picture that shows the analysis for all segments and the LV GLS value, the average LS of all three planes were combined. By averaging the strain rates of all parts, the global longitudinal strain rate (GLSR) was determined. Damaged (infarct-related) segments were identified as segments with longitudinal strains of less than 15%. Damaged longitudinal strain (HLS) and damaged longitudinal systolic strain rate (HLSR) were used to determine the mean LS and LSR of the

damaged parts. A larger negative value demonstrates a larger extent of longitudinal strain; in general, values are expressed as negative values for longitudinal strain.

Follow up:

All cases were examined using 2D-STE and echocardiography after three months. An increase of 20% in LVEDV from baseline to the 3-month follow-up was considered LV remodel. Two categories were formed: one that underwent modification and one that did not. **(22)**.

Statistical Analysis:

Categorical data was described using percentages and frequency, whilst quantitative data was shown using range and Mean ± Standard Deviation (SD). The analysis was conducted using the SPSS v28 statistical tool, which was developed and is maintained by IBM in Armonk, New York, USA. For the purpose of establishing a normal distribution for the data, histograms and the Shapiro-Wilks test were employed. Utilizing standard deviation (SD) and mean, quantitative parametric data was assessed with a Tukey post hoc test and an ANOVA (F) test. The qualitative variables' percentages and frequencies were examined with the help of the Chi-square test. A statistically significant result was defined as a two-tailed P value less than 0.05. If the association is multivariate or univariate, logistic regression can be used to assess the association among the dependent variable and one or more independent variables.

Results

The baseline characteristics of the groups that were studied as well as the risk factors in **Tables 1 & 2**. The groups that were initially examined did not significantly different in terms of the following: age, sex, weight, height, BMI, and BSA. In terms of cardiovascular risk factors, the categories did not exhibit any statistically significant differences. The number of diseased vessels

and the Culprit vessels (LAD, LCX, and RCA) did not differ significantly across the categories. **Figures 1 &2**.

Table 1: Baseline characteristics of the studied groups

		Group I (n=50)	Group II (n=50)	Group III (n=50)	Group IV (n=50)	P value
Age (years)	**Mean± SD**	57.26 ± 8.58	58.62 ± 7.48	60.0 ± 8.67	59.84 ± 9.25	0.343
	Range	43 – 73	44 – 70	47 – 78	45 - 77	
Sex	**Male**	34 (68%)	31 (62%)	39 (78%)	36 (72%)	0.356
	Female	16 (32%)	19 (38%)	11 (22%)	14 (28%)	
Weight (Kg)	**Mean± SD**	82.78 ± 6.09	80.46 ± 6.49	81.26 ± 4.4	83.06 ± 6.85	0.100
	Range	72 – 94	70 – 90	74 – 89	73 – 96	
Height (m)	**Mean± SD**	1.68 ± 0.05	1.69 ± 0.05	1.7 ± 0.05	1.7 ± 0.05	0.125
	Range	1.60 - 1.78	1.61 - 1.77	1.62 - 1.79	1.60 - 1.76	
BMI (Kg/m^2)	**Mean± SD**	29.34 ± 2.66	28.13 ± 2.56	28.23 ± 2.26	28.66 ± 3.04	0.091
	Range	23.36 - 35.1	23.1- 33.2	23.62 - 33.53	23.9 - 35.1	
BSA (m^2)	**Mean± SD**	2.0 ± 0.09	2.0 ± 0.08	2.02 ± 0.09	2.03 ± 0.09	0.165
	Range	1.84 - 2.36	1.84 - 2.16	1.86 - 2.18	1.87 - 2.18	

Table 2: Risk factors of the studied groups

	Group I (n=50)	Group II (n=50)	Group III (n=50)	Group IV (n=50)	P value

Smoking	29 (58%)	19 (38%)	24 (48%)	20 (40%)	0.172
HTN	32 (64%)	28 (56%)	33 (66%)	26 (52%)	0.437
DM	24 (48%)	21 (42%)	22 (44%)	20 (40%)	0.870
Dyslipidemia	11 (22%)	10 (20%)	16 (32%)	13 (26%)	0.524
Family history of CAD	13 (26%)	9 (18%)	7 (14%)	10 (20%)	0.496

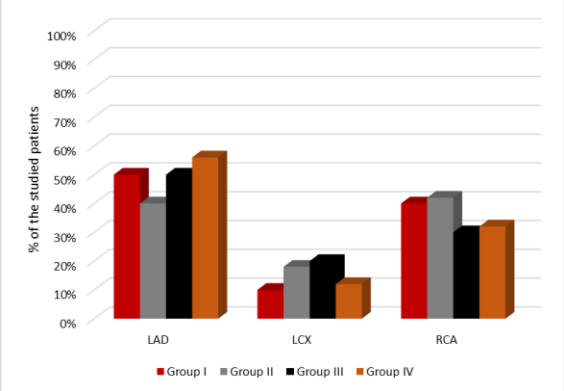

Figure 1: Culprit vessel distribution

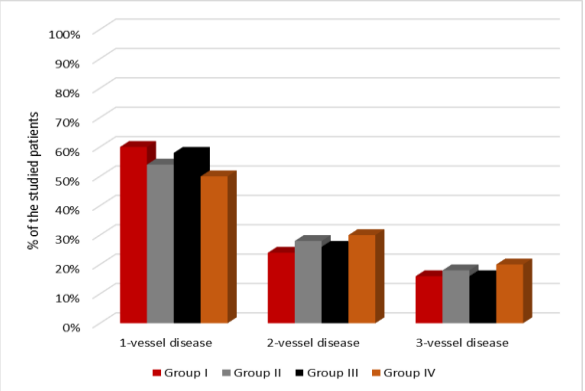

Figure 2: Number of diseased vessels

Cases with STEMI had significantly greater levels of Peak CK-MB and Troponin I compared to cases with NSTEMI (P<0.05). In comparison to categories II and IV, WMSI was significantly lower in groups I and III (P<0.05). Although categories I and III did not differ significantly from one another, Table 3.

Table 3: Baseline echocardiographic data

		Group I (n=50)	Group II (n=50)	Group III (n=50)	Group IV (n=50)	P value
EF (%)	**Mean ± SD**	53.04 ± 6.29	53.12 ± 8.33	51.7 ± 7.61	52.58 ± 9.2	0.798
	Range	47 – 61	43 – 59	46 – 60	40 – 58	
LVEDV (ml)	**Mean ± SD**	94.22 ± 16.7	100.6 ± 18.5	96.1 ± 19.01	100.1 ± 13.9	0.180
	Range	64 – 122	62 – 133	66 – 132	74 – 135	
LVESV (ml)	**Mean ± SD**	52.0 ± 6.5	56.0 ± 7.2	55.0 ± 7.8	58.0 ± 8.5	0.059
	Range	39 – 65	43 – 72	40 – 74	45 – 79	
LVMI (g/m^2)	**Mean ± SD**	104.2± 26.9	99.5± 15.85	106.4 ± 17.7	102.7 ± 17.1	0.358
	Range	64.9 - 162.6	75.5 - 130.2	75 - 130.5	76.6 - 130.4	
E/A ratio	**Mean ± SD**	1.18 ± 0.29	1.09 ± 0.37	1.09 ± 0.39	1.14 ± 0.25	0.435
	Range	0.7 - 2.1	0.6 - 2.2	0.5 - 2.2	0.7 - 1.8	

E/e´ ratio	**Mean ± SD**	11.6 ± 2.2	13.2 ± 2.28	11.99 ± 1.7	15.1 ± 0.81	**<0.001***
	Range	8.1 – 17	8.5 - 17.2	9 - 14.5	13.5 - 16.5	
	Post hoc	**P1=0.001*,** P2=0.356, **P3<0.001*, P4=0.003***, **P5<0.001***, **P6<0.001***				
	Range	144 – 262	141 – 266	137 – 260	142 – 266	
WMSI	**Mean ± SD**	1.73 ± 0.35	2.02 ± 0.36	1.77 ± 0.28	1.95 ± 0.29	**<0.001***
	Range	1.21 - 2.26	1.33 - 2.54	1.28 - 2.31	1.35 - 2.46	
	Post hoc	**P1<0.001*,** P2=0.571, **P3=0.001*, P4<0.001***, P5=0.285, **P6=0.002***				
SV (ml)	**Mean ± SD**	55.86 ± 9.81	60.7 ± 11.09	56.8 ± 10.49	57.1 ± 10.27	0.100
	Range	40 – 81	35 – 81	40 – 81	40 – 77	

*: statistically significant as P value <0.05, P1: P value between groups I & II, P2: P value between groups I & III, P3: P value between groups I & IV, P4: P value between groups II & III, P5: P value between groups II & IV, P6: P value between groups III & IV.

The HLS and HLSR were significantly weaker (less negative) in both groups II and IV in in comparison to either category I and III (P<0.05), with no significant difference when comparing categories I to III and also when comparing groups II. However, regarding baseline LV GLS and GLSR, with respect to both GLS and GLSR, the groups that were part of the study did not differ significantly from one another as shown in **table 4**.

Table 4: Baseline 2D STE parameters among studied categories

		Group I (n=50)	Group II (n=50)	Group III (n=50)	Group IV (n=50)	P value
GLS (%)	**Mean ± SD**	-15.64 ± 1.27	-15.39 ± 1.42	-15.4 ± 1.35	-15.77 ± 1.31	0.400
	Range	-18.03 - - 13.29	-18.04 - - 13.22	-18 - - 13.26	-18.1- - 13.28	
GLSR (s^{-1})	**Mean ± SD**	-1.06 ± 0.08	-1.09 ± 0.08	-1.07 ± 0.07	-1.08 ± 0.07	0.344
	Range	-0.95 - - 1.2	-0.96 - -1.19	-0.95 - - 1.19	-0.95 - -1.2	
HLS (%)	**Mean ± SD**	-12.51 ± 1.68	-11.39 ± 1.42	-12.08 ± 1.3	-11.49 ± 1.4	< **0.001***
	Range	-14.99 - -9.41	-13.91 - -9.1	-15.1 - -9.6	-13.91 - - 8.9	
	Post hoc	**P1<0.001*,** P2=0.151, **P3=0.001*, P4=0.014***, P5=0.713, **P6=0.034***				
HLSR (s^{-1})	**Mean ± SD**	-0.85 ± 0.14	-0.73 ± 0.09	-0.81 ± 0.13	-0.74 ± 0.07	< **0.001***

	Range	-1.09 - -0.6	-0.85 - -0.6	-1.08 - -0.6	-0.84 - -0.6	
	Post hoc	**<0.001*,** P2=0.819, **P3<0.001*, P4<0.001***, P5=0.333, **P6<0.001***				

*: statistically significant as P value <0.05, P1: P value between groups I & II, P2: P value between groups I & III, P3: P value between groups I & IV, P4: P value between groups II & III, P5: P value between groups II & IV, P6: P value between groups III & IV.

Upon follow up Echocardiography after 3 months, it was noticed that LVEF has also demonstrated not significantly difference among the categories that were studied. Total incidence of remodelling was 23.5% among all patients, as in **Table 5**.

Table 5: Follow up 2D echocardiographic data

		Group I (n=50)	**Group II (n=50)**	**Group III (n=50)**	**Group IV (n=50)**	**P value**
EF (%)	**Mean ± SD**	57.67± 5.92	55.35 ± 7.50	56.9 ± 6.44	54.89 ± 8.44	0.388
	Range	38-64	33-60	39-65	35-61	
LVEDV (ml)	**Mean ± SD**	108.4 ± 18.6	111.4 ± 16.8	112.6 ± 17.3	113.9 ±20.9	0.489
	Range	81-171	84-178	78-173	86-176	
LVESV (ml)	**Mean ± SD**	64.8 ± 10.6	69.5 ± 10.1	65.78 ± 11.32	70.87 ± 11.5	0.070
	Range	38-102	39-105	40-101	41-105	
Remodelling	**Number / Percentage**	29 / 29%		18 / 18%		**0.027***
		13 (26%)	16 (32%)	8 (16%)	10 (20%)	0.252
WMSI	**Mean ± SD**	1.54 ± 0.27	1.79 ± 0.43	1.50 ± 0.23	1.68 ± 0.39	**<0.001***
	Range	1.2-1.94	1.3-2.35	1.12-1.8	1.18-2.58	
	Post hoc	**P1<0.001***, P2=0.798, **P3=0.039***, P4<0.001*, P5=0.183, **P6<0.001***				

*: statistically significant as P value <0.05, P1: P value between groups I & II, P2: P value between groups I & III, P3: P value between groups I & IV, P4: P value between groups II & III, P5: P value between groups II & IV, P6: P value between groups III & IV.

In STEMI cases (categories I and II), the prevalence of remodeling was considerably more than that of NSTEMI patients (categories III and IV) (29% vs. 18%) (P= 0.027). Follow up GLS, GLSR, HLS and HLSR showed significant difference among the categories at the three-month follow up. Improvement in GLS (Δ GLS), was significantly better in categories I and III if contrasted with either categories II or IV. A nearly similar correlation was noticed regarding improvement in HLS (Δ HLS), as in **Table 6.**

Table 6: Follow up 2D STE of the studied groups

		Group I (n=50)	Group II (n=50)	Group III (n=50)	Group IV (n=50)	P value
GLS (%)	**Mean ± SD**	-17.9 ± 1.79	-15.9± 1.83	-18.1± 1.70	--17.2±1.65	**<0.001***
	Range	-20.8 _ -9.8	-19.2 _ -8.3	-21.11 _ -10.5	-20.2 _ -9.1	
	Post hoc	P1<**0.001*,** P2=0.128, P3=0.089, P4<**0.001**, P5<**0.001***, P6=0.068				
GLSR (s^{-1})	**Mean ± SD**	-1.14 ± 0.09	-1.08 ± 0.14	-1.19 ± 0.11	-1.11 ± 0.11	**<0.001***
	Range	-1.38 _ -0.97	-1.23 _ - 0.81	-1.35 _ -0.94	-1.31 _ -0.89	
	Post hoc	P1<**0.001*,** P2=0.051, P3=0.233, P4<**0.001**, P5=0.059, P6<**0.001***				
HLS (%)	**Mean ± SD**	-16.35 ± 1.59	-13.29 ± 1.92	-16.98 ± 1.42	-14.19 ± 1.54	**< 0.001***
	Range	-19.6 _ -9.8	-17.9 _ -7.9	-18.9 _ -10.2	-17.6 _ -8.4	
	Post hoc	P1<**0.001*,** P2=0.595, P3<**0.001***, P4<**0.001**, P5=0.089, P6<**0.001***				
HLSR (s^{-1})	**Mean ± SD**	-1.04 ± 0.14	-0.91 ± 0.12	-1.07 ± 0.11	-0.97 ± 0.09	**< 0.001***
	Range	-1.29 _ -0.82	-1.17 _ -0.72	-1.23 _ -0.85	-1.22 _ -0.76	
	Post hoc	P1<**0.001*,** P2=0.799, P3<**0.001***, P4<**0.001**, P5<**0.001***, P6=**0.089**				
Δ GLS (%)	**Mean ± SD**	-2.26 ± 0.52	-0.51 ± 0.41	-2.7 ± 0.35	-1.43 ± 0.34	**<0.001***
	Post hoc	P1<**0.001*,** P2=0.367, P3<**0.001***, P4<**0.001**, P5<**0.001***, P6<**0.001***				
Δ HLS (%)	**Mean ± SD**	-3.84 ± -0.09	-1.9 ± 0.5	-4.9 ± 0.12	-2.7 ± 0.14	**< 0.001***
	Post hoc	P1<**0.001*,** P2=0.295, P3<**0.001***, P4<**0.001**, P5<**0.001***, P6<**0.001***				

*: statistically significant as P value <0.05, P1: P value between groups I & II, P2: P value between groups I & III, P3: P value between groups I & IV, P4: P value between groups II & III, P5: P value between groups II & IV, P6: P value between groups III & IV.

While there was no statistically significant difference in cardiovascular risk factors, the remodeling category did show significantly older individuals (P=0.038), as well as significantly higher peak CK-MB and peak troponin levels (P=0.001 and P=0.001, respectively). **Table 7**

Table 7: Baseline demographic, clinical and laboratory characteristics categorized by remodeling at 3 months

		Non remodeling group (n=153)	Remodeling group (n=47)	P value
Age (years)	**Mean± SD**	58.84 ± 8.27	61.66 ± 7.43	**0.038**
	Range	43 – 73	48 – 72	
Sex	**Male**	105 (68.6%)	35 (74.5%)	0.445
	Female	48 (31.4%)	12 (25.5%)	
Weight (Kg)	**Mean± SD**	82.06 ± 7.99	82.91±6.78	0.121
	Range	70 – 96	72 – 94	
Height (m)	**Mean± SD**	1.69 ± 0.06	1.70 ± 0.04	0.424
	Range	1.60 - 1.79	1.63 - 1.77	
CK-MB (IU/L)	**Mean± SD**	140.75±53.22	208.89±73.17	**0.004**
	Range	59 – 258	83 – 353	
Troponin I (ng/mL)	**Mean± SD**	3183 ± 2316	8745 ± 4987	**0.001**
	Range	823 – 22461	2146 – 33685	
eGFR (mL/min/1.73m^2)	**Mean± SD**	91.78 ± 10.58	91.47 ± 6.38	0.806
	Range	70.5 – 116	80.5 - 103.8	
SBP (mmHg)	**Mean± SD**	128 ± 20.87	135 ± 22.43	0.834
	Range	90 – 160	95 – 180	
DBP (mmHg)	**Mean± SD**	76 ± 8.05	78 ± 8.99	0.071
	Range	65 – 100	70 – 105	
BMI (Kg/m^2)	**Mean± SD**	28.90 ± 3.59	28.88 ± 2.64	0.963
	Range	23.12 - 37.11	24.34 - 34.11	
BSA (m^2)	**Mean± SD**	2.01 ± 0.09	2.02 ± 0.09	0.529
	Range	1.84 - 2.36	1.86 - 2.18	

*: statistically significant as P value <0.05.

There was a considerably higher occurrence of advanced Killip class III in the remodeling category (P <0.001). The culprit vessels were significantly different among the two groups of people. While RCA participation was considerably lower in the non-remodeling category (P=0.005), LAD involvement was higher in the remodeling category (P=0.020) as shown in **Table 8**.

Table 8: Culprit vessel and killip class distribution categorized by remodeling

		Non remodeling group (n=153)	Remodeling group (n=47)	P value
LAD		68 (44.4%)	30 (63.8%)	**0.020***
LCX		22 (14.4%)	8 (17%)	0.657
RCA		63 (41.2%)	9 (19.1%)	**0.005***
Killip class	**I**	53 (34.6%)	7 (14.9%)	**0.009***

	II	90 (58.8%)	17 (36.2%)	**0.001***
	III	10 (6.5%)	23 (48.9%)	**<0.001***

*: statistically significant as P value <0.05.

When comparing the two categories at baseline 2D STE, there was no notable difference in GLSR between them. However, when comparing the remodeling category to the non-remodeling group, GLS was considerably lower (less negative) (P<0.001). Similarly, the remodeling category showed considerably weeker HLS and HLSR. compared to the non-remodeling category (P<0.001, <0.001). High WMSI at cutoff > 1.75 predicts the remodeling with AUC 0.775 and P value of <0.001, with 93.62% sensitivity, 60.13% specificity, 41.9% PPV and 96.8% NPV, as shown in **Table 9**.

Table 9: Baseline 2D echocardiography and STE categorized by remodeling

		Non remodeling group (n=153)	Remodeling group (n=47)	P value
EF (%)	**Mean± SD**	55.04 ± 3.9	52.09 ± 4.91	0.059
	Range	46 – 61	40 – 60	
LVEDV (ml)	**Mean± SD**	88.08 ± 14.75	110.4 ± 12.83	**<0.001***
	Range	68 – 110	88 – 135	
LVESV (ml)	**Mean± SD**	51.4 ± 5.9	64.5 ± 7.2	**<0.001***
	Range	39 – 62	56 – 79	
	Range	130.85 - 311.75	160.5 - 289.9	
LVMI (g/m^2)	**Mean± SD**	101.06 ± 22.24	104.93 ± 17.6	0.178
	Range	64.93 - 162.66	75 - 130.5	
LAVI (mL/m^2)	**Mean± SD**	32.22 ± 4.43	38.03 ± 2.83	**<0.001***
	Range	28.1 - 40.9	34 - 42.9	
E/A ratio	**Mean± SD**	1.14 ± 0.34	1.10 ± 0.31	0.374
	Range	0.6 - 2.2	0.5 - 2.2	
E/e` ratio	**Mean± SD**	12.36 ± 2.26	14.1 ± 2.28	**<0.001***
	Range	8.1 - 16.1	9.5 - 17.2	
	Range	137 – 262	141 - 266	
WMSI	**Mean± SD**	1.83 ± 0.23	1.97 ± 0.32	**0.002***
	Range	1.21 - 2.05	1.45 - 2.54	
	Range	35 – 81	40 – 81	
GLS (%)	**Mean± SD**	-15.98 ± 1.28	-14.98 ± 1.35	**<0.001***
	Range	-18.1 - -14.08	-17.72 - -13.2	
GLSR (s^{-1})	**Mean± SD**	-1.11 ± 0.07	-1.08 ± 0.08	0.089
	Range	-0.95 - -1.2	-0.95 - -1.2	
HLS (%)	**Mean± SD**	-13.56 ± 0.87	-12.02 ± 1.19	**<0.001***
	Range	-15 - -12	-14 - -10.1	
HLSR (s^{-1})	**Mean± SD**	-0.85 ± 0.1	-0.73 ± 0.07	**<0.001***
	Range	-1.18 - -0.7	-0.85 - -0.6	

*: statistically significant as P value <0.05.

As compared to the non-remodeling category, the remodeling category had significantly lower LVEF, GLS, and GLSR at 3-month follow-up (P<0.05). On the other hand, the remodeling category had significantly higher LVEDV, LVESV, and WMSI (P<0.05), and significantly weaker HLS and HLSR (P<0.001, <0.001) than the non-remodeling category, as shown in **Table 10.**

Table 10: Follow up 2D echocardiography and STE categorized by remodeling

		Non remodeling group (n=153)	Remodeling group (n=47)	P value
EF (%)	**Mean± SD**	58.88 ± 3.84	44.43 ± 6.88	**<0.001***
	Range	53 – 65	33 – 55	
LVEDV (ml)	**Mean± SD**	103.58 ± 13.13	148.6 ± 18.92	**<0.001***
	Range	78 – 126	115 – 182	
LVESV (ml)	**Mean± SD**	46.93 ± 9.61	78.55 ± 17.44	**<0.001***
	Range	31.7 - 64.2	49.8 – 108	
WMSI	**Mean± SD**	1.47 ± 0.21	1.72 ± 0.25	**0.039***
	Range	1.15 - 1.91	1.43 - 2.58	
GLS (%)	**Mean± SD**	-18.0 ± 1.92	-14.49 ± 2.26	**<0.001***
	Range	-21.11 - -11.94	-18 - -8	
GLSR (s^{-1})	**Mean± SD**	-1.17 ± 0.12	-1.02 ± 0.13	**<0.001***
	Range	-1.38 - -0.98	-1.23 - -0.81	
HLS (%)	**Mean± SD**	-16.89 ± 1.85	-13.08 ± 1.24	**<0.001***
	Range	-20 - -14	-15 - -11	
HLSR (s^{-1})	**Mean± SD**	-1.06 ± 0.13	-0.93 ± 0.09	**<0.001***
	Range	-1.29 - -0.8	-1.05 - -0.72	

*: statistically significant as P value <0.05.

Results from the univariate logistic regression study indicated that age, smoking, HTN, dyslipidemia, Killip class, multivessel coronary artery disease (2 and 3- vessels), peak CK-MB, and baseline LVEDV, HLS, and HLSR were significant predictors of occurrence of remodeling, as shown in **Table 11**.

Table 11: Univariate logistic regression analysis for prediction of remodeling

	OR	95% CI	P value
Age	1.0480	1.0119 to 1.0854	**0.009***
Sex	1.3065	0.7127 to 2.3950	0.387
BMI (Kg/m^2)	0.9744	0.9019 to 1.0527	0.510
BSA (m^2)	14.7417	0.5809 to 374.1136	0.102
Smoking	1.0480	1.0119 to 1.0854	**0.011***
HTN	52.6592	1.5802 to 174.8109	**0.027***
DM	0.8321	0.4695 to 1.4748	0.529
Dyslipidemia	0.397	0.1830 to 0.8615	**0.019***
Family history of CAD	1.3509	0.6648 to 2.7449	0.405
Killip class	2.3655	1.0234 to 5.4676	**0.044***
Culprit vessel	0.9128	0.6730 to 1.2381	0.557
Multivessel CAD	1.0483	1.0120 to 1.0860	**0.013***
HR (beats/min)	0.9768	0.9472 to 1.0072	0.133

SBP (mmHg)	0.9958	0.9852 to 1.0066	0.445
DBP (mmHg)	0.9962	0.9853 to 1.0072	0.495
CK-MB (IU/L)	1.1851	1.0519 to 1.3351	**0.005***
Troponin I (ng/mL)	0.9767	0.9020 to 1.0576	0.561
S. creatinine (mg/dL)	3.9523	0.8527 to 18.3178	0.079
eGFR (mL/min/1.73m^2)	0.9960	0.9651 to 1.0279	0.805
Baseline			
EF (%)	0.9960	0.9651 to 1.0279	0.804
LVEDV (ml)	1.7954	1.5080 to 2.1376	**<0.001***
LVESV (ml)	0.9636	0.9048 to 1.0262	0.247
LVM (g)	1.0035	0.9942 to 1.0129	0.463
LVMI (g/m^2)	1.0091	0.9903 to 1.0283	0.3430
LAVI (mL/m^2)	1.0048	0.9958 to 1.0138	0.300
E/A ratio	0.6764	0.2862 to 1.5987	0.373
E/e` ratio	0.9517	0.8928 to 1.0145	0.128
Deceleration time (ms)	1.0021	0.9945 to 1.0097	0.596
WMSI	0.9754	0.9001 to 1.0570	0.543
SV (ml)	0.9794	0.9541 to 1.0053	0.118
GLS (%)	0.9773	0.9474 to 1.0081	0.146
GLSR (s^{-1})	1.3378	0.6028 to 2.9690	0.474
HLS (%)	3.9324	2.6665 to 5.7991	**<0.001***
HLSR (s^{-1})	1.0566	1.0102 to 1.1051	**<0.001***

*: statistically significant as P value <0.05.

The univariate logistic regression study showed that Killip class, Culprit vessel (LAD), multivessel coronary artery disease (2 and 3- vessels), peak Troponin I, baseline LVEF, LVESV, WMSI, GLS, HLS and HLSR were considered significant predictors of remodeling, as shown in **Table 12**.

Table 12: Multivariate logistic regression analysis for prediction of remodeling

	OR	**95% CI**	**P value**
Age	0.5781	0.3262 to 1.0245	0.061
Sex	1.4345	0.7439 to 2.7661	0.281
BMI (Kg/m^2)	0.9719	0.8941 to 1.0563	0.502
Smoking	1.0031	0.5712 to 1.7616	0.991
BSA (m^2)	16.5277	0.6229 to 438.5088	0.093
HTN	0.5683	0.3192 to 1.0120	0.055
DM	1.9445	0.8140 to 4.6454	0.135
Dyslipidemia	0.4239	0.1758 to 1.0222	0.056
Family history of CAD	1.6084	0.7688 to 3.3648	0.207
Killip class	1.0480	1.0116 to 1.0857	**0.003***
Culprit vessel	1.0483	1.0120 to 1.0860	**0.008***
Coronary artery disease	1.0483	1.0120 to 1.0860	**0.009***

HR (beats/min)	0.9768	0.9472 to 1.0074	0.136
SBP (mmHg)	1.0070	0.9880 to 1.0263	0.474
DBP (mmHg)	1.0103	0.9808 to 1.0407	0.498
CK-MB (IU/L	1.1478	1.0128 to 1.3007	0.031*
Troponin I (ng/mL)	1.1626	1.0434 to 1.2955	**0.006***
S. creatinine (mg/dL)	2.5942	0.5292 to 12.7183	0.239
eGFR (mL/min/1.73m^2)	0.9997	0.9666 to 1.0340	0.987
Baseline			
EF (%)	0.9597	0.8992 to 1.0242	**0.013***
LVEDV (ml)	0.9295	0.8547 to 1.0109	0.088
LVESV (ml)	1.1320	1.0927 to 1.1726	**0.019***
LVM (g)	1.0044	0.9947 to 1.0141	0.374
LVMI (g/m^2)	1.0094	0.9903 to 1.0288	0.339
LAVI (mL/m^2)	1.0454	0.9997 to 1.0932	0.051
ST$_d$ (cm)	0.5618	0.0760 to 4.1525	0.572
E/A ratio	1.0193	0.9240 to 1.1244	0.703
E/e` ratio	1.0081	0.9163 to 1.1091	0.868
Deceleration time (ms)	1.0023	0.9945 to 1.0100	0.567
WMSI	1.5037	1.3395 to 1.6879	**<0.001***
SV (ml)	0.9812	0.9556 to 1.0075	0.160
GLS (%)	0.1949	0.0637 to 0.5961	**0.004***
GLSR (s^{-1})	1.2953	0.5786 to 2.8997	0.529
HLS (%)	1.9377	1.5986 to 2.3488	**<0.001***
HLSR (s^{-1})	1.0484	1.0052 to 1.0934	**0.027***

*: statistically significant as P value <0.05.

Discussion

The most crucial treatment for AMI is reperfusion therapy, which decreases infarct size and promotes heart function and hence improving the clinical outcomes **(16)**. The myocardium is suddenly overloaded after an acute injury, which starts the ventricular remodeling process and worsens the prognosis. **(17)**. Negative LV remodeling possesses an increased risk of heart failure and death. Its prevalence is approximately 30% following AMI **(3)**.

According to 2D-STE, global longitudinal strain is a useful predictor of LV remodeling. **(23)**. In cases of AMI, particularly in individuals with an LVEF above 40%, LS is a more accurate and non-invasive predictor of hemodynamic worsening than LVEF and WMIS. **(24)**. We have

combined both global and local echocardiographic and deformational parameters in our study to provide a wider scale of evaluation of those critical patients either at baseline or upon follow up.

Comparable to rates reported in earlier research, our investigation demonstrated LV remodeling in 23.5% of the cohort **(8) (25) (26).** Our investigation might explain the lower rate of LV remodeling compared to others because we have included cases with both STEMI and NSTEMI, and their baseline LV systolic functions were minimally affected to relatively preserved **(8) (27)**.

We have found that age was significantly higher in remodeling group which was in line with findings of **Bordejevic et al.,** where patients with LV remodeling at 6 months were older and correlated with male gender **(23)**. Other studies have reported no significant difference regarding baseline demographic and clinical data **(28) (29)**, while **Hsiao et al.,** has reported a correlation with female gender **(30)**.

There was no statistically significant change among the categories for the cardiovascular risk variables (smoking, HTN, DM, dyslipidemia, and family history of CAD) between the beginning and end of the remodeling process. Also, **Eldeeb et al.** and **Tawfik et al.** showed similar results regarding remodeling and traditional CV risk factors **(28) (29)**. While **D'Andrea et al.,** DM was found to be a strong independent predictor of unfavorable LV remodeling at 6 months in 70 individuals with acute NSTEMI, according to the study. **(8)**.

Acute STEMI cases compared to NSTEMI cases and remodeling categories had substantially higher peak CK-MB and Troponin I level in the current investigation. Also **Hsiao et al.,** and **Hendriks et al**., have concluded correlations between found peak CK-MB and Troponin T with adverse remodeling **(30) (31)**. Higher cardiac biomarkers usually correlate with infarction size and longer time to reperfusion.

At baseline, the culprit vessel (LAD, LCX and RCA) found not significantly difference between the categories examined in cases with STEMI and those without. But considering occurrence of remodeling, the culprit vessel LAD was more commonly involved in remodeling group (P=0.020) while RCA was more in the non-remodeling category (P=0.005).

In line with this, **Loboz-Grudzien´ et al.,** have found that culprit LAD was among the significant baseline predictors for LV remodeling at 6 months, the investigation found that primary PCI was effective in treating 88 cases with first-ever STEMI. **(32)**. Also, **Park et al.**, demonstrated the value of STE-measured longitudinal strain at seven LV parts in the LAD territor as a predictor of LV remodelingy **(27)**. Nearly similar findings were also reported by **Aboelkasem et al.** However, they have reported a non-significant difference regarding number of diseased vessels **(33)**.

Also, **Zaliaduonyte-Peksiene et al.** compared the LV remodeling and non-remodeling categories and found that LAD and LCX, as infarct-related arteries, were significantly relevant determinants in 82 AMI cases **(34)**.

While in a study done by **Hassan Shah et al.**, culprit vessel sThe categories that underwent remodeling and those that did not show significant difference (P= 0.468). Three vessel disease was significantly correlated with remodeling group (P < 0.001), but not single or two vessels disease **(35)**. On the other hand, **Xu et al.**, We looked at 110 cases who had an ST-elevation myocardial infarction (STEMI) and then had primary percutaneous coronary intervention and revascularization. At the 3-month follow-up, LV remodeling was observed in 26 cases, or 24% of the total. A rise of 20% in LVEDV was considered to be this. A statistically significant variation was found in the case with three-vessel disorder **(22)**.

While **Hsiao et al.,** have found that the proportion of LAD to non-LAD culprit artery was 52.5% in the non-remodeling category and 37.5% in the remodeling category (P= 0.43), with no significant difference regarding single or multivessel lesions **(30)**.

Our research revealed that the baseline LVEDV, LVESV, E/e′ ratio, and WMSI did not exhibit a significant difference in relation to the baseline LVEF. However, the remodeling category exhibited substantially greater values than the non-remodeling category.

Eldamanhory et al., have demonstrated that LV remodeling was connected to higher levels of LVEDV and LVESV, whereas non-remodeled category had higher levels of LVEF **(36)**. While **Hsiao et al.,** have discovered that the LVEF, E/A ratio, and E/e' ratio did not differ significantly. In the category that had LV remodeling, WMSI was noticeably greater (P= 0.03). In contrast, lower initial LVEDV and LVESV values were seen in the remodeling category **(30)**. **Sugano et al.** in this investigation of 71 patients STEMI patients who underwent primary PCI, found no significant difference regarding baseline LVEDV, LVESV, LVEF and E/e′ ratio for occurrence of LV remodeling **(37)**.

According to our study, the remodeling category had considerably lower baseline GLS compared to the non-remodeling category (P<0.001). There was no statistically significant difference between the two categories at baseline when it came to GLSR, however HLS and HLSR were significantly weaker in the remodeling category (P<0.001, <0.001).

Also, **Hassan Shah et al.,** demonstrated a decrease in baseline LV GLS in cases that underwent LV remodeling in comparison to those that did not **(35)**. **Tawfik et al.** studied 130 STEMI patients with successful PCI and 6 months follow up for the occurrence of LV remodeling. LV GLS was significantly different between groups with a mean baseline GLS of −15.7 ± 3.6% **(29)**.

Park et al. have initially documented the predictive value for left ventricular remodeling of LS in the LAD area using STE at seven LV segments. Out of fifty cases with anterior-wall AMI, twenty-two underwent left ventricular remodeling **(27)**.

Hsiao et al., have showed that regarding STE indices, at baseline, only injured LS (Infarct related, cut-off level < −15% for LS) and injured longitudinal SR were significantly worse in the remodeling category, while GLS and GLSR showed no significant difference **(30)**. Also, **Bordejevic et al.,** have concluded that, baseline No significant difference was observed in the remodeling category with respect to GLS or GLSR, whereas HLS and HLSR were substantially weaker **(23)**.

In the present study, it was found in univariate logistic regression analysis that age, smoking, HTN, dyslipidemia, Killip class, coronary artery disease (2 and 3- vessels), CK-MB, baseline LVEDV, HLS, and HLSR were significant predictors of remodeling. However, the multivariate logistic regression analysis showed that Killip class, Culprit vessel (LAD), coronary artery disease (2 and 3- vessels), Troponin I, baseline LVEF, LVESV, WMSI, GLS, HLS and HLSR were the only significant predictors of remodeling.

In line with our results, **Hsiao et al.,** At 6-month follow-up, 83 patients with first AMI had LV remodeling that was independently predicted by male gender, CK-MB, and damaged LS, according to multivariate analysis **(30)**.

Na et al., reviewed 208 cases who had a low-risk STEMI and had a PCI. Of these cases, 53 (or 25.5%) had LV remodeling. A correlation between LV remodeling and LVEDV, LVESV, CK-MB, and LV GLS was found in the univariate study. Individually, LVEDV, GLS, and CK-MB have all been found to predict remodeling in multivariate analysis **(38)**.

Bordejevic et al., We have identified 15 predictors of remodeling in AMI cases who have undergone PCI and have midrange or preserved LVEF, as determined by univariate logistic regression analysis. Included in this category were age, HTN, dyslipidemia, smoking history, systolic and diastolic blood pressure, Killip class, eGFR, peak CK-MB, 2- and 3-vessel CAD, LVEDV and LVESV, as well as HLS and HLSR. Only five independent predictors for remodeling were chosen by the multivariate logistic regression: Killip class, baseline LVESV, 3-vessel CAD, and HLS **(23)**.

Cong et al., A threshold value of -10.85 yielded an 89.7 percent sensitivity and 91.7% specificity in multivariable logistic regression analysis, indicating that the GLS was a significant predictor of left ventricular remodeling. LV remodeling is defined as a rise of more than 15% in LVESV at 6 months. **(39)**. Also, **Tawfik et al.,** demonstrated a cutoff value of baseline GLS > −12.5% as a predictor of LV remodeling (64.5% sensitivity and 89% specificity). In multivariate logistic regression analysis baseline GLS > −12.5% was a significant predictor for remodeling (OR 0.704, 95% CI 0.597-0.829, P < .001) **(29)**.

Abdelhakam et al. concluded that Killip class, baseline LVEF and LVESV were significantly correlated with remodeling in a univariate analysis but failed to show significant difference in multivariate regression analysis. With a 6-month follow-up, they examined 107 cases who had their first acute STEMI managed by primary PCI or thrombolysis followed by PCI. The remodeling rate was approximately 34% **(40)**. While **Bastawy et al.** The independent factors that were determined to be a predictor of LV remodeling following anterior STEMI by multivariate logistic regression analysis were a baseline WMSI greater than 1.8, a baseline LVEF less than 40%, a GLS greater than -12.5%, a peak CK-MB, and total ischemic heart disease **(41)**.

A number of drawbacks are present in the research. This investigation is limited to individuals with AMI who are in sinus rhythm; this selection process likely includes people with smaller infarctions. The trial is conducted in a single center. As we have excluded patients with severely impaired LVEF, in cardiogenic shock and those with severe valvular affection. Although myocardial contractility may largely recover within two days following revascularization, echocardiography was conducted within 2-3 days after the PCI, not immediately. There are multiple definitions for remodeling, volumes are more accurately assessed by cardiac magnetic resonance imaging and longer follow up may be warranted. Also, reverse LV remodeling was not specified. We did not evaluate circumferential or radial strain, but they are relatively preserved in AMI and still there is lack of suggestions for cut-off values to define infarcted segments. It is possible that residual significant ischemia contributed to the development of LV remodeling; however, we did not assess myocardial perfusion following PCI to rule this out. But these findings support using 2D-STE for AMI risk classification.

Conclusion:

Earlier intervention is crucial in AMI that allows earlier improvement in myocardial performance. 2D-STE is an efficient, practical and reliable noninvasive prognostic procedure after AMI. HLS, HLSR and WMSI are excellent predictors for LV remodeling and may do better than global parameters like LVEF and GLS.

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