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Evaluation of LV Remodeling and Predictors of Heart Failure in Cases with Acute ST Elevation Myocardial Infarction with Preserved Left Ventricular Systolic Function after Successful Primary PCI

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Abstract:

Background: Left ventricular remodeling (LVR) after acute STEMI significantly affects long-term outcomes, even in patients initially presenting with preserved EF. Early detection of those at risk for adverse remodeling and heart failure is critical. Speckle-tracking echocardiography (STE) identifies myocardial deformation prior to EF reduction. This study evaluated LVR using STE in STEMI patients with preserved EF treated by primary PCI. **Methods:** Fifty STEMI patients with preserved EF following PCI underwent clinical and echocardiographic assessment at baseline and six months. **Results:** The reduced EF group (<50%) showed significantly lower LVEF, LVEDV, LVESV, and ILS ($P < 0.05$). No significant differences were found in SVI, WMSI, E/A ratio, GLS, GLSR, GCS, GCSR, GRS, GRSR, ILSR, or infarct-related segment count. At six months, LVEF, SVI, GLSR, and GCS were significantly decreased, while LVESV and WMSI were increased in the <50% group ($P = 0.009$ and < 0.001, respectively). Univariate logistic regression identified age, smoking, hypertension, hyperlipidemia, Killip class, NT-proBNP, ILS, and ILSR as associated factors. **Conclusion:** Patients developing reduced EF at six months had higher Killip class and worse clinical outcomes, including LVR, HF, MI, hospitalization, and MACCE. STE-derived ILS and ILSR are promising early predictors of remodeling post-PCI and may enhance risk stratification in preserved EF STEMI populations.

Keywords: STEMI; Left ventricular remodeling; Speckle-tracking Echocardiography; PCI; Ejection fraction.

Introduction

Heart failure (HF) remains a widespread clinical concern with substantial implications for public health and healthcare systems globally, affecting both developed and developing nations. It persists as a primary contributor to cardiovascular morbidity and mortality globally [1].

The etiological factors underlying HF differ according to ejection fraction categories. Acute ST-segment elevation myocardial infarction (STEMI) and chronic systemic hypertension (HTN) are the primary drivers of HF with reduced ejection fraction (HFrEF) and HF with mildly reduced ejection fraction (HFmrEF). Conversely, HF with preserved ejection fraction (HFpEF) is predominantly associated with HTN, non-STEMI events, and a diverse spectrum of conditions such as atrial fibrillation, cardiomyopathies, myocarditis, valvular heart disease, and metabolic disorders including diabetes mellitus [2].

Timely implementation of percutaneous coronary intervention (PCI) can effectively restore perfusion to infarct-related arteries and substantially mitigate early mortality. Nonetheless, a significant proportion of myocardial infarction survivors eventually experience adverse left ventricular remodeling (LVR), even following successful reperfusion, which may precipitate the onset of HF or death [3].

LVR describes the structural and functional alterations in the myocardium triggered by the initial myocardial injury and impaired contractile function. It is broadly recognized as a crucial prognostic marker in the early phases of cardiac recovery and rehabilitation, especially after acute myocardial infarction (AMI), given its strong correlation with HF progression [4].

Multiple investigations have aimed to elucidate predictors of LVR through the assessment of serological biomarkers, echocardiographic indices, cardiac magnetic resonance imaging (CMRI), and coronary angiography (CAG) [5].

Traditional echocardiography continues to serve as the cornerstone imaging modality in the assessment of cases with AMI. Parameters such as left ventricular ejection fraction (LVEF) and wall motion score index (WMSI) remain well-validated predictors of ventricular remodeling and case prognosis [6].

However, advanced modalities like two-dimensional speckle-tracking echocardiography (2D-STE), which quantify left ventricular strain (S) and strain rate (SR), offer enhanced sensitivity for detecting subclinical myocardial dysfunction, particularly in individuals with preserved or borderline LVEF [7].

This research aimed to employ 2D-STE in evaluating LVR and identifying early indicators of HF in cases with acute STEMI who initially presented with preserved ejection fraction and underwent primary PCI.

Patients and methods:

Patients:

This prospective cohort study included 50 patients diagnosed with acute STEMI, all of whom underwent primary PCI and showed preserved LVEF on initial echocardiographic evaluation. Participants were admitted to the Cardiology Department of Benha Teaching Hospital in the duration between January 2024 and December 2024. Written informed consent was obtained from all enrollees after being fully briefed on the research objectives, with each case anonymized via a unique code. Ethical approval was granted by the Institutional Ethical Committee (Approval Code: MS 11-12-2023) of the Faculty of Medicine, Benha University Hospital.

Inclusion criteria encompassed cases aged 18 years or older, of both sexes, presenting with STEMI, managed with primary PCI, and exhibiting preserved LVEF (≥50%) post-intervention.

Exclusion criteria included cases with non-STEMI, prior myocardial infarction,

post-PCI reduced ejection fraction, significant valvular disease, arrhythmias, thyroid dysfunction (hyper- or hypothyroidism), alcohol misuse, active chemotherapy, renal impairment, multivessel disease necessitating coronary artery bypass grafting (CABG) as per CAG findings, immunosuppressive therapy, intolerance to key medications (aspirin, heparin, clopidogrel, ticagrelor, contrast media), and serious comorbidities such as cerebrovascular events, variceal hemorrhage, or pregnancy.

Methods:

All participants underwent a thorough clinical assessment beginning with detailed history-taking. This encompassed demographic data such as name, age, and sex, alongside a comprehensive CV risk profile including diabetes, obesity, HTN, smoking status, and dyslipidemia. A history of ischemic heart disease, prior revascularization procedures (either PCI or CABG), and details regarding the onset and nature of presenting symptoms were also recorded.

Physical examination followed, involving both general and targeted assessments. General examination included evaluation of vital signs and signs of volume overload, such as peripheral edema, while focused examination involved detailed cardiac and abdominal assessment to identify signs of HF or other relevant clinical findings.

Laboratory evaluations comprised a complete blood count alongside assessments of renal and hepatic function. High-sensitivity cardiac troponin levels were measured upon the patient's initial presentation to the emergency department to aid in the diagnosis of myocardial injury. Additionally, a comprehensive lipid profile was obtained, and NT-proBNP concentrations were measured at the time of admission to evaluate the extent of myocardial wall stress and hemodynamic burden.

A 12-lead electrocardiogram (ECG) was performed within the first ten minutes of emergency department arrival, in line with recommended acute care protocols. A follow-up ECG was conducted following successful revascularization via PCI to assess for dynamic ischemic alterations or evidence of reperfusion, such as resolution of ST-segment elevation or T-wave inversion.

Transthoracic echocardiography assessments and evaluations were conducted following the comprehensive guidelines set forth by the American Society of Echocardiography. Essential parameters including left ventricular (LV) size, volume, and systolic function were measured employing the Simpson's biplane method. Systolic function was deemed preserved when LVEF was equal to or exceeded 50%. A meticulous visual assessment of regional wall motion was performed across 17 myocardial segments utilizing a standardized scoring system ranging from 1 (normal motion) to 5 (aneurysmal motion). The wall motion score index (WMSI) was calculated as the mean of all segmental scores, providing a semi-quantitative evaluation of regional contractile performance. Diastolic function assessment was carried out through pulsed-wave Doppler analysis of mitral inflow, with particular emphasis on the E/A ratio as an indicator of LV filling dynamics.

Myocardial deformation imaging was executed using two-dimensional speckle-tracking echocardiography (2D-STE) to obtain detailed insights into myocardial mechanics. Global and directional strain (S) and strain rate (SR) parameters were measured in three spatial planes: longitudinal (L), circumferential (C), and radial (R). Circumferential and radial parameters—including global circumferential strain (GCS), strain rate (GCSR), radial strain (GRS), and strain rate (GRSR)—were derived from parasternal short-axis views obtained at basal, mid-ventricular, and apical levels. Longitudinal deformation indices (GLS and GLSR) were recorded from standard

apical two-, three-, and four-chamber views, ensuring comprehensive segmental coverage. Overall, deformation characteristics were analyzed across 18 myocardial segments. Systolic strain rate values in all dimensions were integrated to calculate global peak strain metrics, facilitating the detection of subtle myocardial dysfunction. Segments exhibiting longitudinal strain values less negative than −15% were categorized as infarcted, reflecting impaired contractile function. For these infarcted segments, mean strain and strain rate values were calculated and reported as HLS and HLSR, respectively. Additionally, the total number of segments meeting infarction criteria was documented to support quantitative and prognostic analyses.

Coronary angiography was performed via radial or femoral arterial access depending on clinical context and operator preference. Visualization of the infarct-related artery was achieved using standard coronary projections before and after PCI. Each angiographic study was conducted with meticulous technical precision to optimize image quality, ensuring unobstructed views of the catheter tip, minimal foreshortening, and avoidance of vessel overlap. At least one post-PCI acquisition encompassed the full myocardial perfusion territory to enable calculation of the TIMI frame count. Particular caution was exercised when interpreting lesions with less than 60% stenosis to prevent artifacts caused by suboptimal angulation or projection.

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Statistical Analysis:

All statistical analyses were performed using IBM SPSS Statistics for Windows, Version 26.0 (IBM Corp., Armonk, NY, USA). Quantitative variables were reported as means ± standard deviations (SD) and analyzed using the unpaired Student's t-test or one-way asnalysis of variance (ANOVA, F-test), depending on the distribution and comparison requirements. Categorical variables were expressed as absolute frequencies and corresponding percentages. Comparisons between categorical groups were conducted using the Chi-square test or Fisher's exact test when expected frequencies were low. A two-tailed p-value of less than 0.05 was considered to indicate statistical significance throughout the analysis. [8]

Results:

The age of the enrolled population ranged from 45 to 79 years, with a mean of 63.02 ± 9.2 years. Of the total 50 cases, 39 (78%) were male and 11 (22%) were female. Case weight ranged from 60 to 110 kg, with a mean of 86.7 ± 15.3 kg. Heights ranged from 1.60 to 1.79 meters (mean: 1.70 ± 0.06 m), yielding a BMI range of 20.76 to 40.53 kg/m² and a mean BMI of 29.9 ± 4.95 kg/m². **(Table 1)**

At baseline, LVEF, LVEDV, LVESV, and ILS were significantly diminished in the group with LVEF <50% compared to those with preserved function (LVEF ≥50%) (P<0.05). Other echocardiographic indices—including stroke volume index, WMSI, E/A ratio, GLS, GLSR, GCS, GCSR, GRS, GRSR, ILSR, and infarct segment count—did not show statistically significant differences between the two groups. At 6-month follow-up, LVEF, stroke volume index, GLSR, and GCS remained significantly diminished in the reduced LVEF group (P<0.05). In contrast, LVESV and WMSI were significantly elevated in this group (P=0.009 and <0.001, respectively), while LVEDV, E/A ratio, GCSR, and GRS did not differ significantly. (**Table 2)**

One-vessel CAD was significantly less frequent in the reduced LVEF group (P=0.001). Conversely, two- and three-vessel CAD were more prevalent among cases with preserved LVEF. No significant differences were observed in culprit vessel distribution (LAD, LCX, RCA) or TIMI flow grades between groups. (**Table 3)**

Table 1: Baseline characteristics of the enrolled cases

		Total (n=50)
Age (years)	**Mean± SD**	63.02 ± 9.2
	Range	45 - 79
Sex	**Male**	39 (78%)
	Female	11 (22%)
Weight (Kg)	**Mean± SD**	86.7 ± 15.3
	Range	60 - 110
Height (m)	**Mean± SD**	1.70 ± 0.06
	Range	1.6 - 1.79
BMI (Kg/m^2)	**Mean± SD**	29.9 ± 4.95
	Range	20.76-40.53

BMI: body mass index.

Table 2: Baseline Echocardiography of the enrolled groups

Baseline Echocardiography		**Total (n=50)**	**LVEF ≥50% (n=37)**	**LVEF <50% (n=13)**	**P**
LVEF (%)	**Mean± SD**	57.8 ± 5.21	58.7 ± 5.39	55.2 ± 3.81	**0.039***
LVEDV (mL)	**Mean± SD**	102.02 ± 10.4	104.6 ± 9.5	94.8 ± 9.72	**0.003***
LVESV (mL)	**Mean± SD**	41.8 ± 5.66	43.8 ± 4.81	36 ± 3.56	**<0.001***
Stroke volume index (mL/m^2)	**Mean± SD**	40.8 ± 5.94	41.1 ± 6.06	40.1 ± 5.75	0.605
WMSI	**Mean± SD**	2.2 ± 0.15	2.19 ± 0.14	2.27 ± 0.15	0.088
E/A ratio	**Mean± SD**	1.12 ± 0.36	1.13 ± 0.35	1..08 ± 0.39	0.653
GLS (%)	**Mean± SD**	-18.1 ± 1.95	-18.4 ± 1.86	-17.3 ± 2.06	0.089
GLSR (s^{-1})	**Mean± SD**	-1.28 ± 0.37	-1.3 ± 0.38	-1.1 ± 0.28	0.079
GCS (%)	**Mean± SD**	-17.4 ± 3.07	-17.5 ± 3.12	-17.2 ± 3.03	0.799
GCSR (s^{-1})	**Mean± SD**	-1.58 ± 0.32	-1.5 ± 0.33	-1.7 ± 0.27	0.144
GRS (%)	**Mean± SD**	35.3 ± 7.49	35.6 ± 7.95	34.5 ± 6.2	0.666
GRSR (s^{-1})	**Mean± SD**	1.86 ± 0.34	1.8 ± 0.36	1.9 ± 0.26	0.383
ILS (%)	**Mean± SD**	-12.78 ± 0.78	-12.97 ± 0.8	-12.2 ± 0.35	**0.002***
ILSR (s^{-1})	**Mean± SD**	-0.75 ± 0.04	-0.75 ± 0.04	-0.73 ± 0.04	0.101
Number of infarct-related segments	**Mean± SD**	5.93 ± 2.33	5.97 ± 2.45	١.٦ ± ٦.٥٠	0.472
Echocardiography after 6 months					
LVEF (%)	**Mean± SD**	54.9 ± 7.35	58.3 ± 4.85	45.2 ± 3.59	**<0.001***
LVEDV (mL)	**Mean± SD**	107 ± 16.51	105.2 ± 16.03	112.1 ± 17.46	0.201
LVESV (mL)	**Mean± SD**	47.2 ± 8.01	45.5 ± 7.01	52.2 ± 8.91	**0.009***
Stroke volume index (mL/m^2)	**Mean± SD**	47.1 ± 4.63	48.3 ± 4	43.7 ± 4.77	**0.001***
WMSI	**Mean± SD**	2.03 ± 0.09	1.98 ± 0.03	2.18 ± 0.02	**<0.001***
E/A ratio	**Mean± SD**	1.05 ± 0.18	1.07 ± 0.19	1.0 ± 0.16	0.247
GLS (%)	**Mean± SD**	-19.3 ± 2.36	-20.1 ± 1.65	-17.1 ± 2.66	**<0.001***
GLSR (s^{-1})	**Mean± SD**	-1.38 ± 0.16	-1.41 ± 0.17	-1.30 ± 0.12	**0.038***
GCS (%)	**Mean± SD**	-17.9 ± 2.71	-18.4 ± 2.52	-16.5 ± 2.82	**0.027***
GCSR (s^{-1})	**Mean± SD**	-1.58 ± 0.32	-1.54 ± 0.33	-1.69 ± 0.27	0.144
GRS (%)	**Mean± SD**	31.2 ± 8.39	31.5 ± 8.1	30.2 ± 9.43	0.613

LVEF: left ventricular ejection fraction, LVEDV: left ventricular end diastolic volume, LVESV: left ventricular end systolic volume, WMSI: wall motion score index, E/A: early to atrial filling velocity ratio, GLS: global longitudinal strain, GLSR: global longitudinal strain rate, GCS: global circumferential strain, GCSR: global circumferential strain rate, GRS: global radial strain, GRSR: global radial strain rate, ILS: infarct-related longitudinal strain, *: statistically significant as p <0.05.

Table 3: Coronary angiography of the enrolled groups

		Total (n=50)	**LVEF ≥50% (n=37)**	**LVEF<50% (n=13)**	**P**
CAD	**1-vessel**	28 (56%)	25 (67.56%)	3(23.07%)	**0.001***
	2-vessel	10 (20%)	6 (16.21%)	4 (30.76%)	0.558
	3-vessel	12 (24%)	6 (16.21%)	6 (46.15%)	0.121
Culprit vessel	**LAD**	25 (50%)	19 (51.35%)	5 (38.46%)	0.747
	LCX	7 (14%)	5 (13.51%)	2 (15.38%)	1
	RCA	18 (36%)	13 (35.14%)	6 (46.15%)	0.83
TIMI FLOW	**2**	4 (8%)	3 (8.1%)	1 (7.6%)	0.173
	3	46 (92%)	34 (91.9%)	12 (92.3%)	0.172

LAD: left anterior descending artery, RCA: right coronary artery, LCX: left circumflex artery *: statistically significant as p <0.05.

Cases with LVEF <50% exhibited significantly poorer clinical outcomes, including elevated rates of LVR, MACCE, HF, recurrent MI, and hospital readmission (P<0.05). **(Table 4)**

Receiver operating characteristic (ROC) analysis demonstrated that Killip class predicted remodeling with an AUC of 0.856 (*P*<0.001), at a cut-off >2, yielding 95% sensitivity, 60% specificity, PPV of 61.3%, and NPV of 94.7%. LVEF predicted remodeling with an AUC of 0.828 (P<0.001), cut-off ≤52%, with 80% sensitivity and specificity. GLS exhibited strong predictive capacity (AUC 0.941, *P*<0.001) at a threshold of <−18%, achieving 95% sensitivity, 66.7% specificity, PPV of 65.5%, and NPV of 95.2%. GCS (AUC 0.678, *P*=0.022) and GRS (AUC 0.682, *P*=0.016) showed modest predictive value. ILS (AUC 0.804, *P*<0.001) was also a significant predictor, with a cut-off >−13% yielding 85% sensitivity and 73.3% specificity. **(Table 5)**

Univariate analysis identified age, smoking, HTN, hyperlipidemia, Killip class, NT-proBNP, ILS, ILSR, and extent of CAD as significant predictors of LVR. In multivariate analysis, only Killip class, ILS, ILSR, and CAD remained independent predictors. **Table 6**

Table 4: Outcome of the enrolled groups

	Total (n=50)	LVEF ≥50% (n=37)	LVEF <50% (n=13)	P
LV remodelling	21(42%)	10 (27.02%)	11 (84.62%)	**<0.0 01***
MACCE	12 (24%)	6 (16%)	6 (46%)	**0.026***
HF	5 (10%)	1 (2.7%)	4 (30.77%)	**0.003***
MI	7 (14%)	3 (8.11%)	4 (30.77%)	**0.042***
Hospitalization	12 (24%)	5 (13.51%)	7 (53.85%)	**0.004***
Life threating arrhythmia	2 (4%)	1 (3%)	1(8%)	**0.038***

LV: left ventricle, MACCE: major adverse cardiac and cerebrovascular events, HF: heart failure, MI: myocardial infarction, *: statistically significant as p <0.05.

Table 5: Diagnostic accuracy for prediction of LVR

	Cut off	Sensitivity	Specificity	PPV	NPV	AUC	P
Killip class	>2	95.00	60.00	61.3	94.7	0.856	**<0.001***
LVEF (%)	≤52	80.00	80.00	66.7	84.6	0.828	**<0.001***
GLS (%)	<-18	95.00	66.67	65.5	95.2	0.941	**<0.001***
GCS (%)	≤-17	75.00	56.67	53.6	77.3	0.678	**0.022***
GRS (%)	≤36	75.00	46.67	48.4	73.7	0.682	**0.016***
ILS (%)	>-13	85.00	73.33	68.0	88.0	0.804	**<0.001***

LVEF: left ventricular ejection fraction, GLS: global longitudinal strain, GCS: global circumferential strain, GRS: global radial strain, ILS: infarct-related longitudinal strain, PPV: positive predictive value, NPV: negative predictive value, AUC: area under the curve, *: statistically significant as p <0.05.

Table 6: Logistic regression analysis for prediction of LV remodelling

major adverse cardiac and cerebrovascular event, BMI: body mass index, HTN: hypertension, DM: diabetes mellitus, MI: myocardial infarction, PCI:

	Univariate			Multivariate		
	OR	**95%CI**	**P**	**OR**	**95%CI**	**P**
Age (years)	1.1256	1.0385 to 1.2201	**0.004***	1.811	0.4704 to 6.975	0.388
Sex	0.8214	0.2058 to 3.2792	0.781	0.101	0.000 to 282.761	0.571
BMI (Kg/m^2)	1.0616	0.9427 to 1.1955	0.324	0.489	0.1123 to 2.1270	0.340
Smoking	4.3333	1.2976 to 14.471	**0.017***	1.768	0.8930 to 3.2246	0.418
HTN	38.00	4.4289 to 326.03	**0.001***	0.005	0.00 to 187.172	0.417
DM	1.1515	0.3600 to 3.6830	0.812	12.14	0.080 to 183.424	0.330
Hyperlipidaemia	7.4286	2.0604 to 26.783	**0.002***	0.831	0.5233 to 1.332	0.369
Obesity	2.7500	0.8341 to 9.0662	0.096	0.068	0.005 to 21.019	0.533
Family history of CAD	0.4286	0.1298 to 1.4151	0.164	1.126	0.001 to 13.651	0.974
B-blockers	0.4444	0.0880 to 2.2451	0.326	0.332	0.0559 to 1.9787	0.226
ACEI/ ARBs	0.8000	0.1864 to 3.4340	0.764	0.839	0.1765 to 3.9882	0.825
Statin	2.6667	0.7146 to 9.9509	0.144	2.730	0.7018 to 10.620	0.147
HR (beat/min)	1.0292	0.9539 to 1.1104	0.457	1.478	0.7279 to 3.0018	0.280
SBP (mmHg)	1.4548	0.8414 to 2.5153	0.179	1.041	0.7102 to 1.5245	0.839
DBP (mmHg)	1.8153	0.8288 to 3.9760	0.136	1.199	0.7144 to 2.0105	0.493
Killip class	1.1395	1.0533 to 1.2329	**0.001***	1.002	1.0005 to 1.0037	**0.008***
Hb (g/dL)	1.2185	0.5585 to 2.6583	0.619	2.514	0.8240 to 7.6675	0.105
PLT ($*10^9/L$)	1.0053	0.9935 to 1.0173	0.379	1.007	0.9896 to 1.0237	0.454
WBCs ($*10^9/L$)	0.8967	0.6140 to 1.3095	0.572	0.878	0.5057 to 1.5232	0.643
Serum creatinine (mg/dL)	0.8312	0.5269 to 1.3112	0.427	0.696	0.3798 to 1.2742	0.240
ALT(U/L)	0.9450	0.8605 to 1.0379	0.237	0.881	0.7640 to 1.0153	0.080
AST(U/L)	1.0343	0.9331 to 1.1466	0.520	1.134	0.9586 to 1.3415	0.142
NT-proBNP (pg/mL)	1.0017	1.0004 to 1.0030	**0.009***	1.261	0.5763 to 2.7596	0.561
LVEF (%)	0.9517	0.8512 to 1.0642	0.385	0.954	0.7632 to 1.1915	0.676
LVEDV (mL)	0.9481	0.8934 to 1.0062	0.079	1.009	0.8933 to 1.1394	0.887
LVESV (mL)	1.5134	0.5565 to 5.6763	0.098	0.384	0.6276 to 3.0555	0.264
Stroke volume index (mL/m^2)	1.0384	0.9427 to 1.1437	0.445	0.995	0.8372 to 1.1832	0.957
WMSI	16.0087	0.3166 to 809.53	0.166	0.021	0.0000 to 9.0635	0.367
E/A ratio	1.1505	0.2300 to 5.7541	0.864	0.088	0.0009 to 8.8674	0.302
GLS (%)	1.2559	0.9267 to 1.7019	0.142	1.855	0.8179 to 4.2079	0.139
GLSR (s^{-1})	0.6335	0.1327 to 3.0246	0.567	0.191	0.0066 to 5.4978	0.334
GCS (%)	2.608	0.3054 to 22.268	0.381	0.424	0.1795 to 1.0030	0.051
GCSR (s^{-1})	0.6594	0.1092 to 3.9811	0.650	0.204	0.0021 to 19.939	0.496
GRS (%)	0.9335	0.8605 to 1.0127	0.097	0.828	0.6543 to 1.0489	0.118
GRSR (s^{-1})	3.3894	0.5932 to 19.366	0.170	5.156	0.1218 to 21.252	0.391
ILS (%)	1.1446	1.0554 to 1.2414	**0.001***	0.805	0.6570 to 0.9876	**0.037***
ILSR (s^{-1})	0.8066	0.7031 to 0.9253	**0.002***	0.646	0.4436 to 0.9391	**0.022***
Number of infarct-related segments	0.8627	0.6692 to 1.1120	0.254	0.856	0.6404 to 1.1453	0.296
CAD	2.4319	1.1506 to 5.1398	**0.019***	2.222	1.0155 to 4.8614	**0.046***
Culprit vessel	1.3200	0.7105 to 2.4525	0.379	1.422	0.7089 to 2.8517	0.322

percutaneous coronary intervention, CABG: Coronary artery bypass grafting, AF: atrial fibrillation, CAD: Coronary artery disease, ACEI: angiotensin-converting enzyme, ARBs: angiotensin receptor blockers, HR: heart rate, SBP: systolic blood pressure, DBP: diastolic blood pressure, Hb: haemoglobin, PeLT: platelet, WBCs: white blood cells, NT-proBNP: B-type natriuretic peptide, LVEF: left ventricular ejection fraction, LVEDV: left ventricular end diastolic volume, LVESV: left ventricular end systolic volume, WMSI: wall motion score index, E/A: early to atrial filling velocity ratio, GLS: global longitudinal strain, GLSR: global longitudinal strain rate, GCS: global circumferential strain, GCSR: global circumferential strain rate, GRS: global radial strain, GRSR: global radial strain rate, ILS: infarct-related longitudinal strain, LAD: left anterior descending artery, RCA: right coronary artery, LCX: left circumflex artery, *: statistically significant as p <0.05.

Discussion:

Left ventricular remodeling following AMI remains a major determinant of both short- and long-term prognosis despite successful revascularization via primary PCI [9].In this context, speckle-tracking echocardiography offers superior sensitivity compared to LVEF in detecting subclinical myocardial dysfunction and estimating infarct size through GLS and ILS assessment[10].

Our cohort had a mean age of 63.02 years, with male predominance (78%) and a mean BMI of 29.9 kg/m². These findings align with Heidari and colleagues [11], who reported similar demographic and anthropometric characteristics in 629 cases post-STEMI with preserved LVEF.

Our research demonstrated that baseline echocardiographic parameters—including LVEF, LVEDV, LVESV, and ILS—were significantly diminished in cases with reduced LVEF (<50%) in contrast with those with preserved function (≥50%) (P<0.05). Other echocardiographic measures such as stroke volume index, WMSI, E/A ratio, GLS, GLSR, GCS, GCSR, GRS, GRSR, ILSR, and the number of infarct-related segments were comparable between the two groups. At six months, LVEF, stroke volume index, GLSR, and GCS remained significantly diminished in the reduced LVEF group, while LVESV and WMSI were significantly elevated (P=0.009 and P<0.001, respectively). LVEDV, E/A ratio, GCSR, or GRS were comparable between groups at follow-up.

These findings are consistent with Hammouda and colleagues[12], who reported significantly elevated LVESV and WMSI in cases with reduced LVEF (75.1 ± 5.03 mL and 1.9 ± 0.05 points, respectively) compared to those with preserved LVEF (53.2 ± 5.46 mL and 1.5 ± 0.09 points; *P*<0.001).

Coronary angiographic analysis revealed that single-vessel disease was significantly less frequent in the reduced LVEF group (*P*=0.001), while two- and three-vessel disease were more common in the preserved LVEF group. However, the culprit vessel (LAD, LCX, or RCA) and TIMI flow grades were comparable between the groups. These findings are in agreement with Hwang and colleagues [13], who observed that a greater number of infarct-related coronary arteries were associated with more pronounced reductions in LVEF post-PCI.

Similarly, Liu and colleagues[14], found no significant differences in culprit vessel distribution or TIMI flow between cases with preserved and reduced LVEF (P=0.304 and 0.775, respectively).

Clinical outcomes were notably worse in the reduced LVEF group, which exhibited elevated rates of LVR, MACCE, HF, recurrent MI, and hospital readmission (P<0.05). These findings support the results of Okuhara and colleagues[15], who identified LVEF reduction as an independent predictor of adverse cardiac events (HR 5.79; 95% CI 2.49–13.2; *P*<0.001).

In terms of predictive accuracy, Killip class demonstrated a strong ability to predict LVR, with an AUC of 0.856 (P<0.001) at a cut-off >2, yielding 95% sensitivity, 60% specificity, a PPV of 61.3%, and an NPV of 94.7%. Similarly, ILS was a robust predictor (AUC 0.804; P<0.001), with a cut-off of >−13% providing 85% sensitivity, 73.3% specificity, 68.0% PPV, and 88.0% NPV.

These results align with those of Sabry and colleagues[16], who found that 26% of cases experienced LVR at 3-month follow-up. While baseline characteristics were largely similar between remodeling and non-remodeling groups, cases with remodeling had significantly diminished baseline GLS and GCS (P<0.001). In multivariate analysis, baseline GLS emerged as the sole independent predictor (HR 1.68; 95% CI 1.35–2.09; P=0.001), with cut-off values of GLS <−9.0% and GCS <−11.1% offering strong predictive accuracy.

In our research, univariate logistic regression identified several variables as significant predictors of LVR : age, smoking, HTN, hyperlipidemia, Killip class, NT-proBNP, ILS, ILSR, and CAD. However, on multivariate analysis, only Killip class, ILS, ILSR, and CAD remained statistically significant. These findings are in accordance with Bordejevic and colleagues[6], who reported that in 253 AMI cases, age, comorbidities, Killip class, and multi-vessel CAD were associated with remodeling, while multivariate analysis identified HLS, HLSR, Killip class, three-vessel CAD, and LVEDV as independent predictors. ROC curve analysis from their research revealed AUCs of 0.85 and 0.77 for HLS (<−11%) and HLSR (<−0.65 s^{-1}), respectively.

This research has some limitations. First, it involved a relatively small number of cases from a single center, which may limit the generalizability of the results. Second, the follow-up period was limited to six months, so long-term outcomes could not be assessed. Third, some variables such as medication adherence, lifestyle changes, and genetic factors were not evaluated and could have influenced the outcomes.

Conclusion:

Left Ventricular Remodeling occurred in 42% of cases following successful PCI for STEMI. Cases with reduced LVEF (<50%) exhibited a elevated prevalence of CV risk factors (smoking, HTN, hyperlipidemia), elevated systolic and diastolic blood pressure, elevated Killip class, and elevated NT-proBNP levels. These individuals also experienced significantly worse clinical outcomes, including elevated rates of LVR, MACCE, HF, recurrent MI, and hospitalization. Echocardiographically, the reduced LVEF group showed significantly diminished baseline values of LVEF, LVEDV, LVESV, and ILS, and at six months, diminished LVEF, stroke volume index, GLSR, and GCS. Predictive modeling identified age, smoking, HTN, hyperlipidemia, Killip class, NT-proBNP, ILS, ILSR, and CAD as significant predictors of LVR, with multivariate analysis confirming Killip class, ILS, ILSR, and CAD as independent predictors.

List of Abbreviations

Abbreviation	Full Term
ALVR	Adverse Left Ventricular Remodeling
AMI	Acute Myocardial Infarction
CABG	Coronary Artery Bypass Graft
CAD	Coronary Artery Disease
CAG	Coronary Angiography
CMRI	Cardiac Magnetic Resonance Imaging
CV	Cardiovascular
ECG	Electrocardiogram
GCS	Global Circumferential Strain
GCSR	Global Circumferential Strain Rate
GLS	Global Longitudinal Strain
GRS	Global Radial Strain
GRSR	Global Radial Strain Rate
HF	Heart Failure
HFmrEF	Heart Failure with Mid-Range Ejection Fraction
HLS	Harmed Longitudinal Strain
HLSR	Harmed Longitudinal Systolic Strain Rate
HTN	Hypertension
ILS	Indexed Longitudinal Strain
ILSR	Indexed Longitudinal Strain Rate
LAD	Left Anterior Descending
LCX	Left Circumflex
HFPEF	Heart failure with preserved ejection fraction
LDL	Low-Density Lipoprotein
HFREF	Heart failure with reduced ejection fraction
LVEDV	Left Ventricular End Diastolic Volume
LVEF	Left Ventricular Ejection Fraction
LVESV	Left Ventricular End Systolic Volume
LVR	Left Ventricular Remodeling
MACCE	Major Adverse Cardiac and Cerebrovascular Events
MI	Myocardial Infarction
NSTEMI	Non-ST-Elevation Myocardial Infarction
NT-proBNP	B-type Natriuretic Peptide
PCI	Percutaneous Coronary Intervention

PPCI	Primary Percutaneous Coronary Intervention
RCA	Right Coronary Artery
ROC	Receiver Operating Characteristic
SR	Strain Rate
STE	Speckle Tracking Echocardiography
STEMI	ST-Elevation Myocardial Infarction
TIMI	Thrombolysis in Myocardial Infarction
WMSI	Wall Motion Score Index
2D-STE	Two-Dimensional Speckle-Tracking Echocardiography

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