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Article

Evaluation of Left Ventricular Remodeling and Prognosis after PCI in Acute Myocardial Infarction Using Real-Time, Three-Dimensional Echocardiography Combined with Layer-Specific Strain Imaging

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Abstract

Aim: This study aimed to evaluate left atrial and left ventricular volumes, strains, and strain rates in patients with acute ST-segment elevation myocardial infarction (STEMI) treated with percutaneous coronary intervention (PCI) using real-time three-dimensional echocardiography and layer-specific strain techniques. The relationships between these parameters and left ventricular remodeling (LVR) and prognosis were also explored. **Methods**: The study included 217 patients with first-episode STEMI who underwent emergency PCI. Echocardiography and myocardial strain analyses were performed within 24 h post-PCI. Patients were categorized into early and non-early LVR groups based on the increase in left ventricular end-diastolic volume (LVEDV). The occurrence of major cardiovascular adverse events (MACE) was followed up for one-year post-PCI. Differences in clinical data and ultrasound parameters between the groups were compared, and the predictive value of myocardial strain indicators for late LVR was analyzed using a multivariate logistic regression model and receiver operating characteristic (ROC) curves. **Results**: Early LVR occurred in 54.8% of patients and was characterized by decreased left ventricular systolic function, more segments with abnormal movement, and reduced absolute strain values in the three layers of the left ventricular wall myocardium, with a compensatory increase in left atrial strain rate during the contraction phase. The early LVR group showed a higher incidence of MACE at the one-year follow-up. At 6 months post-PCI, 29.9% of patients developed late LVR, which was not completely related to early LVR. Late LVR was associated with a higher incidence of MACE. The longitudinal strain value of each layer of left ventricular myocardium obtained from layer-specific strain imaging showed predictive value for advanced LVR. **Conclusions**: More than half of patients with STEMI develop early LVR post-PCI, with a higher incidence of MACE during one-year of follow-up, necessitating attention to early LVR in clinical practice. Late LVR, which develops in some patients after 6 months, is also linked to a higher incidence of MACE. Accurate monitoring of the myocardial deformation function using layer-specific strain imaging is expected to become a reference indicator for clinical diagnosis and treatment. Monitoring the structure and function of the left atrium and mitral valve alongside the LVR is important for prognostic assessment and for formulating diagnostic and treatment plans.

Keywords

ST-elevation myocardial infarction (STEMI); left ventricular remodeling (LVR); echocardiography; left ventricular myocardial strain

Introduction

Acute ST-segment elevation myocardial infarction (STEMI) is a critical condition and one of the main causes of death and disability in patients with coronary heart disease. According to the classification of myocardial infarction, STEMI occurs because of atherosclerotic plaque rupture in the coronary artery, which erodes blood vessels and causes lumen blockage, leading to myocardial ischemia, injury, and necrosis. Timely diagnosis and treatment with reperfusion therapy are essential for patients with STEMI, to quickly and completely open the occluded coronary arteries. Percutaneous coronary intervention (PCI) is the most effective treatment. In addition to conducting clinical risk assessments for patients with STEMI during hospitalization, guidelines also recommend echocardiography to clarify their cardiac function status, infarct size, and complications [1]. Following myocardial ischemia-reperfusion therapy, patients with STEMI may experience different outcomes. Some patients may see functional recovery of the damaged myocardium [1], while others may undergo adverse remodeling of the left ventricular structure and function, together with major adverse cardiovascular events (MACE) [2,3], which significantly affect cardiac function and prognosis and increase the risk of long-

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term heart failure. The left ventricular remodeling (LVR) process after myocardial infarction has the characteristics of being time-dependent, dynamic, and complex. Reconstruction can be seen in both infarcted and non-infarcted areas, and involves the intercellular matrix, growth factors, cytokines, cell types, and left ventricular morphology and structure [4]. Therefore, LVR post-acute myocardial infarction (AMI) can be divided into early LVR (usually within 24–72 h after AMI) and late LVR, according to the disease stage [5]. However, previous studies have not paid sufficient attention to early LVR. Myocardial remodeling and dysfunction also occur in the left atrium owing to changes in left ventricular pressure and geometry, which are important links in the progression of cardiovascular diseases. This underscores the importance of long-term follow-up planning and medication rehabilitation guidance for such patients. In summary, corresponding evaluations should be performed for both early and late LVR, based on the progression of LVR. Effective early estimation of MACE risks and atrioventricular remodeling, coupled with timely and reasonable intervention plans, is crucial for improving patient prognosis.

In clinical practice, noninvasive cardiac imaging technology plays a vital role in predicting cardiovascular complications in patients with STEMI [6]. Echocardiography is used not only for routine AMI examinations but also for risk stratification assessments, especially for measuring the left ventricular ejection fraction (LVEF), a key factor in clinical decision-making [7]. Real-time three-dimensional echocardiography (RT-3DE) does not require a prior assumption of the geometric shape of the left ventricle and can accurately evaluate the overall and local left ventricular functions quickly and comprehensively [8]. Two-dimensional speckle tracking echocardiography (STE) and its derived layer-specific strain (LSS) can quantitatively evaluate the mechanical and contractile properties of different layers of the ventricular and atrial muscles. Ultrasound parameters of the left ventricular and atrial structures and functions are also powerful biomarkers of cardiovascular events [9]. Our study utilized multimodal quantitative echocardiography to evaluate the structural functions of the left ventricle and left atrium in patients with first-time STEMI within 24 h after AMI and PCI. We aimed to observe the impact of early LVR on cardiac function and follow-up on late-stage atrial and ventricular myocardial remodeling and the occurrence of MACE post-discharge, to explore the relationship between ultrasound parameters, myocardial remodeling, and prognosis.

Materials and Methods

General Data

The clinical data of 221 patients who experienced their first myocardial infarction (MI), were diagnosed with STEMI, and underwent emergency PCI at the Chest Pain Center of the First Hospital of Lanzhou University from September 2020 to September 2021 were selected for this study. After excluding two cases with missing clinical data and two cases lost to follow-up, 217 patients were finally included. All selected patients underwent echocardiography within 24 h after PCI, and images were retained for subsequent quantitative analysis. Baseline clinical data, including blood pressure, diabetes status, smoking and drinking status, N-terminal pro-brain natriuretic peptide (NT-ProBNP) concentration, troponin I (TnI) concentration, and myocardial enzyme values, were collected. The degree of coronary artery stenosis during PCI was determined by an experienced interventional cardiologist, and the medication and treatment plans during hospitalization were recorded. This study conforming to the Declaration of Helsinki [10] was approved by the Ethics Committee of the First Hospital of Lanzhou University, and all participants signed informed consent forms. The Ethics Committee Approval Letter Number was LDYYLL2021-16.

Inclusion and Exclusion Criteria

The inclusion criteria were as follows: an age of 18–80 years and admission with STEMI. The diagnostic criteria for STEMI were as follows: (1) persistent, severe, and typical ischemic chest pain; (2) elevated ST segment, with or without pathological Q and R wave reductions on electrocardiography; (3) elevated serum myocardial injury markers; and (4) imaging evidence of segmental wall motion abnormalities, if necessary.

The exclusion criteria were as follows: (1) previous history of MI; (2) history of coronary artery bypass grafting surgery; (3) organic heart diseases, such as valve disease, congenital heart disease, cardiomyopathy, and constrictive pericarditis; (4) severe liver and kidney diseases; (5) severe arrhythmia, including atrial fibrillation and atrioventricular block; and (6) poor echocardiographic image quality.

Methods

Echocardiography

The SIEMENS Acuson SC2000 color Doppler ultrasound system, equipped with a 4V1c two-dimensional ultrasound probe (probe frequency: 1–4 MHz) and a 4Z1c three-dimensional full-volume ultrasound probe (probe frequency: 1.75–4.25 MHz), was utilized. The velocity vector imaging (VVI) software (version VB10E, Siemens

Medical Solutions USA, Mountain View, CA, USA) integrated into the ultrasound system was used for data acquisition and analysis. Echocardiographic image collection and data analyses were conducted in accordance with the 2019 American Society of Echocardiography guidelines [11].

Acquisition of Two-Dimensional Echocardiographic Images

Patients assumed the left lateral decubitus position, maintaining peaceful and relaxed breathing, with synchronized electrocardiography. Data from at least three cardiac cycles were collected, including the 2D dynamic images of the apical 4-chamber, apical 2-chamber, apical 3-chamber, and long-axis left ventricular views.

Analysis of Conventional and Left Ventricular Volume Parameters on Echocardiography

The left atrial anteroposterior diameter (LAAPD) was measured using two-dimensional M-mode echocardiography on the long axis of the left ventricle view. Mitral regurgitation (MR) was semi-quantitatively graded according to the guidelines [12]; MR was considered mild if the jet area/left atrial area ratio was $<$20% and the vena contracta was $<$0.3 cm and was severe if the ratio was $>$40% and the vena contracta was $\geq$0.7 cm, with moderate MR falling between these criteria. After switching to the 3D full-volume probe, full-volume images of the apical 4-chamber view for at least three cardiac cycles were obtained. The "LVA" function in the software automatically measured LVEF, left ventricular end-systolic volume (LVESV), left ventricular end-diastolic volume (LVEDV), and maximum left atrial volume (LAVmax), to obtain the LAV index (LAVI), which was calculated as: LAVI = LAVmax/BSA.

Analysis of Infarct Site and Left Ventricular Segmental Wall Motion

The left ventricle was divided into 17 segments, and the myocardial motion of each segment was examined by two senior echocardiography physicians. The Wall Motion Score Index (WMSI) was used to semi-quantitatively assess the segmental myocardial motion. Scores of 1–5 represented normal movement, decreased movement, absence of movement, reverse movement, and formation of a ventricular aneurysm, respectively. The WMSI was calculated by adding the scores of each segment and dividing the total score by the total number of segments. Finally, abnormal motion segments observed on ultrasound were matched with the segments indicated by electrocardiography, and the patient's infarct site and segments were recorded.

GLS Analysis of Layer-Specific Strain

The VVI software performed layer-specific strain quantitative analysis of the stored 2D images. The left ventricular endocardium and epicardium were manually drawn point-by-point under the apical 4-chamber, apical 2-chamber, and apical 3-chamber views, respectively, with adjustments for the myocardial thickness of the region of interest (ROI), as needed. The software automatically divided the left ventricular wall into endocardial, mid-myocardial, and epicardial layers and acquired the global longitudinal strain (GLS) of each layer (GLSendo, GLSmid, and GLSepi, respectively) under each view.

Analysis of Left Atrial Strain Rate

After initiating the VVI software and reading the saved apical 4- and 2-chamber views, the electrocardiogram was replayed to the end of the left ventricular systolic phase to clearly display the left atrial endocardium. The left atrial endocardium was manually delineated point-by-point from the atrial septal mitral annulus, excluding the left atrial appendage and pulmonary vein ostia. After completing the delineation, the "analyze" button was pressed to obtain the left atrial strain rate (LASR) curve of the corresponding view. The left atrial strain rate was measured during three phases: reserve phase (LASRr), duration phase (LASRcd), and contraction phase (LASRct). The values from the two views were averaged to obtain the final LASR values for each phase.

Analysis of Repeatability

Thirty cases were randomly selected, and routine echocardiography and strain parameters were measured by two experienced attending physicians and more than two echocardiographic diagnostic physicians who were unaware of the experimental scheme (to avoid interobserver variability). One of the physicians remeasured these parameters at the same step after 2 weeks (to avoid intraobserver variability).

Grouping and Follow-Up

Definition of Early LVR and Grouping

In all patients, the LVEDV obtained by RT-3DE within 24 h after AMI and PCI exceeded the normal reference value from the guidelines by 20%, which is the primary endpoint for defining early LVR. Patients were divided into the early LVR (ELVR) and non-early LVR (NELVR) groups, and differences in clinical and echocardiographic parameters between the two groups were compared.

Follow-Up Status

Follow-up echocardiography was performed at least 6 months after discharge, utilizing RT-3DE to evaluate the LVEDV, LVESV, LVEF, and LAVI. Patients were grouped based on the degree of change in LVEDV. Patients with

a >20% increase in LVEDV compared to baseline were classified into the late left ventricular remodeling (LLVR) group [10]. Patients whose LVEDV enlargement did not meet this criterion were classified into the non-late left ventricular remodeling (NLLVR) group. At least one year after discharge (with an average follow-up time of one year), the follow-up center of our department followed up with these patients by phone. During these calls, the occurrence of MACE was recorded. MACE included the composite events of recurrent angina, MI, severe arrhythmia, ischemic stroke, or cardiovascular death.

Statistics

SPSS 26.0 statistical software (IBM Corp., Armonk, NY, USA) was used to analyze data that conformed to a normal distribution. The Shapiro-Wilk test was used to assess the normality of continuous variables. For between-group comparisons, ANOVA was used, and the LSD-*t* test was applied for pairwise comparisons. Measurement data that did not follow a normal distribution were represented by the median and interquartile range (M [Q1, Q3]), and the Kruskal-Wallis H rank-sum test was employed for intergroup comparisons. Count data are expressed as numbers or percentages, with intergroup comparisons conducted using the χ^2 test. Receiver operating characteristic (ROC) curves were plotted using GraphPad Prism software (version 9.0, Dotmatics, Boston, MA, USA). Univariate logistic regression analysis was performed to identify predictive factors of LVR. A p-value < 0.05 was considered statistically significant.

Results

Enrollment Status

A total of 217 patients with their first episode of STEMI who received PCI treatment were enrolled, with an average age of 64 $\pm$ 7 years. The cohort comprised 191 males (88%) and 26 females (12%). During hospitalization, 119 patients (54.8%) experienced early LVR, and at the 6-month follow-up, 65 patients (29.9%) exhibited an increase in LVEDV and late LVR. During the one-year follow-up, 22 patients (10.1%) experienced MACE.

Early and Late LVR

Within 24 h of AMI, 119 patients (54.8%) experienced early LVR, while 98 patients (45.2%) did not. The clinical baseline data and echocardiographic parameters of the two groups are shown in Table 1. The proportion of patients with a Killip grade $\geq$2 and anterior wall MI was higher in the ELVR group than in the NELVR group. The NT-proBNP and TnI concentrations were also higher in the ELVR group. As shown in Table 2, compared with the NELVR group, the ELVR group had larger LVEDV, LVESV, LAVI, WMSI, and LASRct, while GLSendo and GLSepi were smaller, with a higher proportion of moderate-to-severe MR. At the 6-month follow-up, the ELVR group had a significantly reduced LVEF and increased LVEDV, LVESV, and LAVI. At the one-year follow-up, the incidence of MACE had significantly increased and approaching statistical significance (p = 0.057). Compared to the NLLVR group, the LLVR group had a higher proportion of patients with a Killip grade $\geq$2, anterior wall MI, and extracorporeal membrane oxygenation (ECMO) assistance during hospitalization, along with higher NT-proBNP and TnI concentrations (Table 3). The LVEF and LVEDV of the LLVR group were lower than those of the NLLVR group, with an increase in LAVI and a significant increase in the proportion of moderate-to-severe MR. GLSendo, GLSmid, GLSepi, and LASRr were significantly reduced in the LLVR group. At the six-month follow-up, the LLVR group had a significant decrease in LVEF and a significant increase in LVEDV, LVESV, and LAVI (Table 4), leading to an increased incidence of MACE at the one-year follow-up (all, $p < 0.05$) (Table 3).

Binary Logistic Regression Analysis of Predictive Factors for Late LVR

Binary logistic regression analysis incorporating clinical and strain indicators showed that, after adjusting for clinical and echocardiographic parameters, GLSendo, GLSmid, and LASRr were predictive indicators, with odds ratios (OR) of 1.658, 1.196, and 0.292, respectively (Table 5).

ROC Curve Analysis

The area under the curve (AUC) of GLSendo, GLSmid, and GLSepi for predicting late LVR were 0.909, 0.896, and 0.855, respectively, with optimal cutoff values of –15.865%, –11.595%, and –9.73%, respectively; sensitivities of 87.7%, 81.5%, and 90.8%, respectively; and specificities of 90.8%, 88.2%, and 67.8%, respectively ($p < 0.001$). The predictive strengths of LASRr, LASRcd, and LASRct for late LVR were significantly lower than those of GLS, with AUCs of 0.596, 0.504, and 0.525, respectively. The optimal cutoff values were 0.72 (s^{-1}), –1.63 (s^{-1}), and –2.055 (s^{-1}), respectively, with sensitivities of 59.2%, 50.8%, and 58.5%, respectively; specificities of 92.1%, 94.7%, and 86.2%, respectively; and p-value of 0.026, 0.042, and 0.049, respectively (Fig. 1 and Table 6).

Discussion

Within hours of acute myocardial infarction (AMI) onset, myocardial cells undergo necrosis due to ischemia. With advancements in cardiovascular diagnosis and treat-

Table 1. Baseline clinical characteristics of early LVR patients.

	ELVR	NELVR	t*/U/x^2	*p*-value
	n = 119	n = 98		
Age (y)*	60.00 ± 10.96	61.00 ± 10.65	–0.567	0.493
Male (%)	108 (90.7)	83 (84.7)	1.873	0.171
Hypertension (%)	59 (49.6)	39 (39.8)	2.077	0.150
Diabetes mellitus (%)	22 (18.5)	20 (20.4)	0.127	0.722
Hyperlipidemia (%)	32 (26.9)	27 (27.6)	0.012	0.913
Smoking (%)	41 (34.5)	33 (33.7)	0.015	0.904
Killip grade ≥2 (%)	13 (10.9)	3 (3.1)	4.865	0.027
Prehospital thrombolysis (%)	37 (31.1)	25 (25.5)	0.821	0.365
D-to-W (min)	53 (35.0, 91.5)	66 (37.5, 99.0)	6191.000	0.302
Stent (%)	100 (84.0)	85 (86.7)	0.312	0.577
ECMO (%)	11 (9.2)	3 (3.1)	3.404	0.063
Anterior wall MI (%)	88 (73.9)	32 (32.7)	37.077	<0.001
MCAD (%)	78 (65.5)	60 (61.2)	0.434	0.510
Aneurysm (%)	8 (6.7)	5 (5.1)	0.251	0.617
NT-proBNP (pg/mL)	2110 (945.25, 4332.50)	1120 (442.50, 2065.00)	3808.500	<0.001
TnI (ng/mL)	25 (8.4, 25.0)	16 (4.55, 25.0)	4874.000	0.030
CK-MB (ng/mL)	9.75 (2.50, 43.69)	8.83 (2.50, 50.11)	5786.500	0.992
Myo (ng/mL)	104.17 (33.89, 249.38)	82 (30.0, 240.9)	5457.000	0.475
Medication (%)				
ARNI	26 (21.8)	10 (10.2)	5.266	0.022
Aspirin	114 (95.8)	91 (92.9)	0.890	0.346
Clopidogrel/Tegrelo	114 (95.8)	91 (92.9)	0.890	0.346
Statins	119 (100.0)	97 (99.0)	-	0.452
β-blockers	90 (75.6)	64 (65.3)	2.780	0.095
1-year follow-up				
MACE (%)	15 (12.6)	5 (5.1)	3.616	0.057

LVR, left ventricular remodeling; D-to-W, door-to-wire; ELVR, early left ventricular remodeling; NELVR, non-early left ventricular remodeling; ECMO, extracorporeal membrane oxygenation; MI, myocardial infarction; MCAD, multiple coronary artery disease; NT-proBNP, N-terminal pro-brain natriuretic peptide; TnI, troponin I; CK-MB, creatine kinase isomer MB; Myo, myoglobin; ARNI, angiotensin receptor neprilysin inhibitor; MACE, major adverse cardiovascular events.

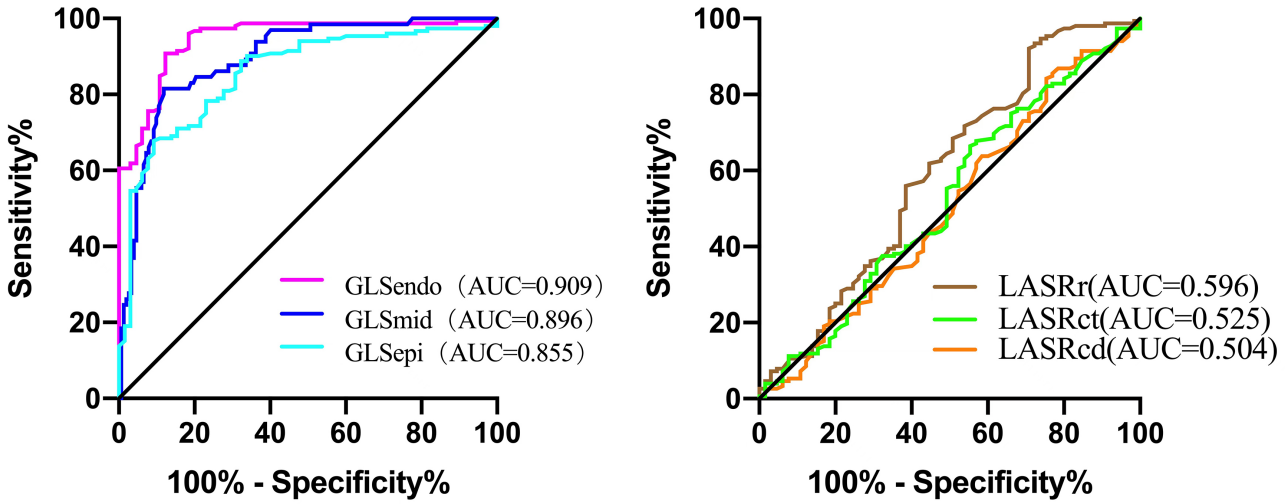

Fig. 1. Receiver operating characteristic (ROC) curves for predicting late LVR using GLS and LASR. AUC, area under curve; GLS, global longitudinal strain; LASR, left atrial strain rate.

Table 2. Echocardiographic parameters of early LVR patients.

	ELVR	NELVR	t*/U/x^2	*p*-value
	n = 119	n = 98		
LVEF (%)	47 (43, 50)	55 (52, 58)	10,026.500	<0.001
LVEDV (mL)	145 (131.25, 158.00)	108 (97.50, 119.00)	951.000	<0.001
LVESV (mL)	74 (66.00, 84.75)	49 (42.00, 54.00)	29.000	<0.001
WMSI	1.35 (1.25, 1.47)	1.12 (1.05, 1.29)	2435.500	<0.001
GLSendo (%)	–16.21 (–19.12, –14.12)	–17.01 (–19.14, –14.32)	6255.000	0.037
GLSmid (%)	–13.42 (–15.09, –10.91)	–12.43 (–14.38, –10.28)	6535.500	0.049
GLSepi (%)	–8.65 (–10.78, –7.41)	–9.74 (–10.92, –7.58)	6452.000	0.041
Moderate-to-severe MR (%)	41 (34.50)	18 (18.40)	7.025	0.008
LAD (mm)	33.19 (31.07, 35.31)	31.54 (29.45, 34.21)	4337.500	0.001
LAVI (mL/m^2)	32.21 (25.60, 38.80)	26.43 (21.33, 31.52)	4212.000	0.001
LASRr (s^{-1})	1.07 (0.89, 1.47)	1.05 (0.85, 1.44)	5597.500	0.515
LASRcd (s^{-1})	–0.91 (–1.15, –0.68)	–0.85 (–1.08, –0.54)	6543.000	0.122
LASRct (s^{-1})	–1.19 (–1.72, –0.86)	–1.17 (–1.82, –0.86)	5768.500	0.892
6-month follow-up				
LVEF (%)	50.5 (45, 56)	58 (54, 61)	8969.500	<0.001
LVEDV (mL)	150 (124.75, 180.50)	120 (103.00, 138.00)	3108.500	<0.001
LVESV (mL)	71 (55.25, 95.25)	50 (41.50, 61.00)	2572.500	<0.001
LAD (mm)	34 (32.00, 37.75)	34 (30.00, 35.50)	5048.000	0.088
LAVI (mL/m^2)	30.48 (24.93, 36.03)	21.68 (17.23, 26.13)	4127.000	0.001

LVEF, left ventricular ejection fraction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; WMSI, wall motion score index; GLS, global longitudinal strain; MR, mitral regurgitation; LAD, left atrial diameter; LAVI, left atrial volume index; LASR, left atrial strain rate.

ment technologies, the timely treatment and survival rates of patients with AMI have greatly improved. However, following the reperfusion of obstructed blood vessels and restoration of the myocardial blood supply, adverse outcomes can still occur. The myocardium undergoes a series of pathophysiological changes, including damage from reactive oxygen species after ischemia-reperfusion, infiltration by numerous inflammatory cells that promote myocardial scar formation, and activation of the neuroendocrine system during the acute phase [13]. The geometric structure, size, and function of the myocardium undergo remodeling to adapt to the adverse stimuli of ischemia and hypoxia. Even with standardized follow-up and postoperative medication, there is still a risk of heart failure and decreased cardiac function [14].

Currently, there is no unified standard for LVR post-AMI, and most researchers tend to use changes in left ventricular volume as an indicator. Cardiac magnetic resonance imaging (CMR) remains the gold standard for non-invasive measurement of cardiac cavity size, volume, and function [15]; however, its widespread application is limited by various factors. Echocardiography, which is recommended as a routine noninvasive imaging evaluation method [16], offers advantages in implementation beyond those of CMR. Cardiac quantification guidelines advocate the use of RT-3DE for assessing cardiac size and function when the echocardiographic image quality meets the standards, owing to its accuracy and repeatability [17]. The GLS obtained using STE is an accurate predictor of left ventricular size and remodeling after PCI [18,19]. However, studies using layer-specific strains to evaluate LVR and prognosis in post-PCI patients with STEMI are scarce. Given the close interrelationship between the left atrium and ventricle, the impact and role of left atrial remodeling have also garnered attention in recent years. Research focusing on left atrial function and its relationship with LVR in patients with STEMI is limited, and the mechanism of action between LVR and left atrial remodeling (LAR) is not yet fully understood.

Early LVR

Previous studies on LVR post-MI have primarily focused on late-stage (6-months post-MI) LVR and heart failure (HF), and less attention has been paid to left ventricular volume expansion and patient prognosis during hospitalization (within 72 h of MI). Through a controlled examination of early LVR in patients with first-time STEMI during hospitalization, we found that more than half (54.6%) experienced early LVEDV enlargement. This aligns with the findings of Barros-Gomes *et al*. [20] and helps exclude cardiac cavity enlargement caused by prior coronary artery disease or HF. Myocardial ischemic necrosis increases the ventricular wall tension and mechanical stretching of myocardial cells, leading to the release of natriuretic peptides (NPs).

Table 3. Baseline clinical characteristics of late LVR patients.

	LLVR	NLLVR	U/x^2	*p*-value
	n = 65	n = 152		
Age (y)	59 (50.5, 67.0)	61 (52.5, 68.5)	4591.500	0.410
Male (%)	61 (93.8)	130 (85.5)	2.988	0.084
Hypertension (%)	31 (47.7)	67 (44.1)	0.240	0.624
Diabetes mellitus (%)	15 (23.1)	27 (17.8)	0.824	0.364
Hyperlipidemia (%)	18 (27.7)	41 (27.0)	0.012	0.913
Smoke (%)	22 (33.8)	52 (34.2)	0.003	0.959
Killip grade ≥2 (%)	9 (13.8)	7 (4.6)	5.693	0.017
prehospital thrombolysis (%)	20 (30.8)	42 (27.6)	0.220	0.639
D-to-W (min)	54 (36.0, 89.5)	64 (37.5, 96.5)	0.164	0.456
Stent (%)	59 (90.8)	126 (82.9)	2.246	0.134
ECMO (%)	9 (13.8)	5 (3.3)	8.407	0.004
Anterior wall MI (%)	43 (66.2)	77 (50.7)	4.423	0.035
MCAD (%)	53 (81.5)	91 (59.9)	9.577	0.002
Aneurysm (%)	8 (12.3)	5 (3.3)	6.575	0.010
NT-proBNP (pg/mL)	1620 (702.5, 4250.0)	1560 (542.0, 3360.0)	5422.500	0.255
TnI (ng/mL)	25.00 (16.0, 25.0)	17.38 (4.8, 25.0)	6352.500	0.001
CK-MB (ng/mL)	7.7 (2.50, 50.13)	9.5 (2.50, 43.31)	4906.500	0.998
Myo (ng/mL)	111.57 (30.00, 386.56)	96.55 (30.00, 212.60)	5338.500	0.304
Medication (%)				
ARNI	19 (29.2)	17 (11.2)	10.716	0.001
Aspirin	59 (90.8)	146 (96.1)	2.433	0.119
Clopidogrel/Tegrelo	59 (90.8)	146 (96.1)	2.433	0.119
Statins	64 (98.5)	152 (100.0)	-	0.300
β-blockers	47 (72.3)	107 (70.4)	0.081	0.776
1-year follow-up				
MACE (%)	10 (15.4)	10 (6.6)	4.219	0.040

LLVR, late left ventricular remodeling; NLLVR, non-late left ventricular remodeling; LVR, left ventricular remodeling; D-to-W, door-to-wire; ECMO, extracorporeal membrane oxygenation; MI, myocardial infarction; MCAD, multiple coronary artery disease; NT-proBNP, N-terminal pro-brain natriuretic peptide; TnI, troponin I; CK-MB, creatine kinase isomer MB; Myo, myoglobin; ARNI, angiotensin receptor neprilysin inhibitor; MACE, major adverse cardiovascular events.

Consequently, the NT-proBNP concentration has been recognized as an effective serological indicator for assessing cardiac function and managing patients with HF. TnI, which is released after myocardial cell injury, is considered the gold standard for the serological diagnosis of AMI and can also reflect the severity of myocardial injury, with predictive value for HF occurrence [21,22]. NT-proBNP and TnI levels in the ELVR group were significantly higher than those in the NELVR group, indicating more severe myocardial injury based on serological indicators. Thus, early LVR leads to myocardial cell necrosis and stunning.

The ELVR group had significantly more patients with a Killip score ≥2, anterior wall MI, and multivessel coronary artery disease than the NELVR group. As previously reported, LV remodeling is significantly higher at the anterior MI location, and the proportion with anterior MI was larger in the remodeled group [23]. These results suggest that the ELVR group exhibits lower cardiac function, more severe clinical HF manifestations, and more severe and extensive coronary artery disease. After the loss of myocardial contractile tissue, myocardial cells undergo hypertrophy and lengthening to maintain the stroke volume (SV) as an early compensatory mechanism [24]. Echocardiographic parameter comparisons showed that the ELVR group had a reduced LVEF and significantly increased LVESV and LVEDV. The WMSI, which reflects the segmental myocardial contractions, was also significantly higher. The GLS of the three layers of the left ventricular myocardium measured using VVI was lower in the ELVR group, suggesting more segmental myocardial systolic dysfunction and a more extensive infarct area, consistent with the results of Barros-Gomes *et al*. [20]. Yalta *et al*. [5] noted that the infarct size is one of the prerequisites for the degree of early LVR and that LVEF undergoes a significant reduction during early LVR, which explains the observed results. The endocardial myocardium, which is most vulnerable to ischemia and hypoxia, is first affected, and the disease gradually extending to the epicardium. Lay-

Table 4. Echocardiographic parameters of late LVR patients.

	LLVR	NLLVR	U/x^2	*p*-value
	n = 65	n = 152		
LVEF (%)	49 (43.0, 53.0)	51 (46.5, 56.0)	3817.000	0.008
LVEDV (mL)	115 (100.0, 139.5)	130 (113.0, 151.0)	3570.000	0.001
LVESV (mL)	58 (46.0, 75.0)	63 (51.0, 76.5)	4344.000	0.159
WMSI	1.35 (1.21, 1.58)	1.23 (1.12, 1.41)	6388.000	<0.001
Moderate-to-severe MR (%)	26 (40.0)	33 (21.7)	7.693	0.006
LAD (mm)	33 (30.0, 35.5)	32 (29.0, 35.0)	5337.000	0.347
LAVI (mL/m^2)	29.82 (23.19, 36.45)	20.23 (15.80, 24.66)	3457.000	0.001
GLSendo (%)	–13.36 (–14.92, –11.62)	–18.69 (–20.26, –16.78)	9340.000	<0.001
GLSmid (%)	–9.83 (–11.28, –8.95)	–14.13 (–15.83, –12.26)	8848.000	<0.001
GLSepi (%)	–7.48 (–9.11, –6.27)	–10.73 (–11.88, –9.25)	8447.000	<0.001
LASRr (s^{-1})	0.87 (0.55, 1.29)	1.11 (0.89, 1.47)	4610.500	0.001
LASRcd (s^{-1})	–0.89 (–1.15, –0.54)	–0.87 (–1.10, –0.61)	4980.000	0.925
LASRct (s^{-1})	–1.13 (–1.77, –0.77)	–1.19 (–1.78, –0.89)	5192.500	0.551
6-month follow-up				
LVEF (%)	51 (45.0, 56.5)	56 (50.0, 60.5)	3256.500	<0.001
LVEDV (mL)	162 (140.0, 189.5)	120 (104.0, 141.0)	8158.500	<0.001
LVESV (mL)	78 (62.5, 104.0)	53 (42.0, 68.0)	7814.500	<0.001
LAD (mm)	35 (33.0, 37.0)	33 (30.0, 37.0)	5895.500	0.024
LAVI (mL/m^2)	30.23 (25.21, 35.25)	21.54 (16.71, 26.37)	4313.000	0.001

LLVR, late left ventricular remodeling; NLLVR, non-late left ventricular remodeling; LVEF, left ventricular eject fraction; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; WMSI, wall motion score index; GLS, global longitudinal strain; MR, mitral regurgitation; LAD, left atrial diameter; LAVI, left atrial volume index; LASR, left atrial strain rate.

ered strains can identify whether an MI is transmural [25]. Notably, in our study, in addition to the GLSendo reduction, the GLSmid and GLSepi were also reduced, indicating that more patients in the ELVR group experienced transmural MI, suggesting a propensity for early LVR in such cases.

Comparing left atrial parameters, we found that both the LAAPD and LAVI increased in the ELVR group compared to those in the NELVR group, suggesting that patients with early LVR also underwent left atrial structural and functional changes. Owing to the uncoordinated contraction of the left ventricular wall, the left ventricular blood cannot be effectively discharged, leading to an increased pressure load in the ventricle and corresponding changes in the left atrium [26]. The larger LASRct in the ELVR group aligns with the Frank-Starling mechanism, indicating left atrial expansion to adapt to and maintain cardiac output. However, no statistically significant differences in left atrial changes were observed between the groups. In early LVR, the left atrial reservoir and conduit functions may not be significantly weakened and may remain compensatory. Thus, early left atrium changes in AMI are linked to structural remodeling and alterations in myocardial contractility. As the heart remodels, the mitral annulus expands and flattens. These changes, in turn, can affect structurally normal valves, leading to insufficient leaflet coaptation [27]. The ELVR group showed a significant increase in moderate-to-severe MR, indicating that early LVR can cause adverse mitral hemodynamic changes that further affect cardiac function and confirming that early LVR is not just an isolated left ventricular phenomenon.

Analysis of the follow-up results revealed that patients with early LVR, despite undergoing timely coronary artery revascularization, still had larger left ventricular volumes and lower systolic function, with a higher incidence of MACE one year later than those without early LVR. Thus, early LVR has a sustained impact on patients [22], leading to decreased cardiac function and poor prognosis. Hence, the importance of the early detection and timely evaluation of LVR cannot be overstated.

Late LVR

Although early and late LVR present similar structural imaging findings in the heart, including increased cardiac volume and functional changes [28], they are fundamentally different in terms of the mechanism, pathophysiological changes, and clinical significance, representing relatively independent disease courses. At the 6-month follow-up in our study, 65 patients developed late LVR, of whom only 21 developed early LVR during hospitalization, accounting for just 18% of ELVR patients. The remaining 82% did not experience early LVR during hospitalization. We speculated that the main reasons were as follows. Early LVR refers to the expansion of the infarcted area, which

Table 5. Logistic regression analysis of influencing factors of late LVR.

	B	SE	Wald	DF	*p*-value	OR	95% CI	
							Lower limit	Upper limit
Male	0.736	0.795	0.857	1	0.355	2.088	0.439	9.918
Age	–0.020	0.021	0.907	1	0.341	0.980	0.940	1.022
Hypertension	–0.170	0.446	0.145	1	0.703	0.844	0.352	2.023
Diabetes mellitus	–0.474	0.534	0.786	1	0.375	0.623	0.218	1.775
GLSendo	0.506	0.107	22.198	1	0	1.658	1.344	2.047
GLSmid	0.179	0.088	4.164	1	0.041	1.196	1.007	1.420
GLSepi	0.058	0.072	0.642	1	0.423	1.059	0.920	1.219
LASRr	–1.230	0.599	4.218	1	0.040	0.292	0.090	0.945
LASRcd	–0.553	0.483	1.314	1	0.252	0.575	0.223	1.481
LASRct	–0.521	0.420	1.544	1	0.214	0.594	0.261	1.351
TnI	0.015	0.009	2.626	1	0.105	1.015	0.997	1.033
NT-proBNP	0	0	0.007	1	0.932	1.000	1.000	1.000
Constant	10.935	2.382	21.074	1	0	56,078.592		

B, Beta; OR, odds ratio; GLS, global longitudinal strain; LASR, left atrial strain rate; NT-proBNP, N-terminal pro-brain natriuretic peptide; SE, standars error; DF, degree of freedom; 95% CI, confidence interval; TnI, troponin I.

Table 6. Parameters for ROC curve analysis.

Variate	Optimum cutoff	Sensitivity	Specificity	AUC	Standard error	*p*-value	95% CI	
							Lower limit	Upper limit
LASRr	0.720	0.592	0.921	0.596	0.045	0.026	0.379	0.554
LASRcd	–1.630	0.508	0.947	0.504	0.044	0.042	0.417	0.591
LASRct	–2.055	0.585	0.862	0.525	0.044	0.049	0.439	0.612
GLSendo	–15.865	0.877	0.908	0.909	0.016	<0.001	0.914	0.976
GLSmid	–11.595	0.815	0.882	0.896	0.023	<0.001	0.851	0.940
GLSepi	–9.730	0.908	0.678	0.855	0.027	<0.001	0.801	0.909

LASR, left atrial strain rate; GLS, global longitudinal strain; AUC, area under the curve; CI, confidence interval.

can be observed several hours after MI onset, leading to left ventricular dilation, reduced ventricular wall thickness, and increased wall tension during diastole and systole. Myocardial cell necrosis and myocardial stunning may occur. After timely reopening of the coronary arteries to restore the blood supply, the degree of myocardial cell damage can be reduced, and stunned myocardial cells may be restored to some extent. Early LVR can also be effectively controlled. The occurrence of late remodeling is time-dependent, involving the entire left ventricle, and wall hypertrophy and ventricular distortion are closely related. Myocardial cells may undergo hypertrophy, apoptosis, and diffuse fibrosis. Studies have found that non-infarcted tissue remodeling occurs around the infarcted area 3 weeks after the MI [29]. Therefore, early and late LVR are not completely correlated in these patients. As Bolognese *et al.* [14] proposed, even after successful vascular patency, patients with a smaller LVEDV but larger infarct sizes within 12 h of AMI may still undergo progressive left ventricular enlargement during follow-up. Therefore, early assessment of the risk of late LVR is crucial.

The LLVR group had a higher proportion of patients with Killip scores $\geq$2, a need for ECMO assistance during hospitalization, and higher concentrations of TnI, confirming that patients with cardiogenic shock and severe myocardial cell ischemia and necrosis during hospitalization were more likely to experience late LVR. Previous study have shown that patients with early ventricular aneurysms within 48 h after AMI are more likely to experience severe remodeling of the ventricular anatomy and function, and their long-term prognoses is also worse [30]. The proportions of patients with anterior wall MI, multivessel coronary artery disease, and ventricular aneurysm during hospitalization were greater in the LLVR group. Our study suggests that ventricular aneurysm formation, anterior wall MI, and the degree and extent of coronary artery disease are related to late LVR.

During hospitalization, the late LVR group exhibited lower LVEF, larger LVEDV and LVESV, and higher WMSI values. The LVEF and WMSI, which are classic parameters for evaluating left ventricular systolic function and myocardial motion, can quickly and accurately assess myocardial injury. Previous studies have confirmed a correlation

between these two factors and the prognosis of AMI patients [31]. Although many studies have reported the prognostic value of the GLS obtained using STE in AMI patients [32], traditional STE techniques cannot distinguish the heterogeneity of the left ventricular myocardium [33]. Our study provided a more accurate hierarchical assessment of myocardial ischemia and damage by observing the strain of the three myocardial layers separately. The GLS of the three layers of the left ventricle in the LLVR group were lower, and the diagnostic and predictive value of GLSendo, GLSmid, and GLSepi in the LLVR group was higher according to regression models and ROC curve analysis. These findings suggest that the peak systolic strain of each left ventricular layer is highly correlated with late LVR.

Notably, GLSepi had predictive value for late LVR; thus, a decrease in epicardial myocardial strain suggests the occurrence of transmural MI. Early studies by Pfeffer Pfeffer and Braunwald [34] suggested that at least 20% of the myocardial fibers must be damaged during transmural MI for LVR to occur. Sharma *et al*. [35] proposed that both GLSendo and GLSepi provide valuable prognostic information on heart disease outcomes and that GLSepi is a better predictor for patients with acute coronary syndrome (ACS). Our study similarly concluded that GLSepi has a higher sensitivity in predicting late LVR than GLSendo and GLSmid. Thus, in the evaluation of LVR, the value of accurately assessing the deformation of the three myocardial layers is evident.

Additionally, the proportion of patients with moderate-to-severe MR significantly increased in the LLVR group, along with a significant increase in the LAVI and a decrease in the LASRr. This suggests that weakened left ventricular myocardial contractility leads to impaired compliance, increased filling pressure, and higher left atrial afterload. Consequently, there is increased volume expansion and obstruction of pulmonary venous blood flow, which affect the left atrial emptying and increase the residual blood. This leads to relaxation and stretching of the myocardium, which weaken the compensatory effect of the Frank-Starling mechanism and result in diminished left atrial diastolic function. Chu *et al*. [36] proposed that left atrial strain (LAS) is an independent predictor of late LVR. Our study found that although LASR can be used as a predictor of late LVR, its diagnostic efficacy is lower than that of left ventricular GLS. This may be because the LASR represents the degree of myocardial deformation per unit time, and the superposition of strain values with time factors could affect the results. Therefore, further research is required to determine the deviation of the LAS and LASR.

At the one-year follow-up, the incidence of MACE was higher in the LLVR group, with lower LVEF values and larger left ventricular and atrial volumes, indicating a lower likelihood of reverse remodeling of the atrioventricular volume in patients with late LVR. This may be related to myocardial fibrosis and irreversible remodeling during late LVR and is closely associated with poor patient prognosis.

Study Limitations

This was a single-center study with a small sample size and a short follow-up period. Early and late LVR were discussed separately, and their relationship was not further explored. Although our study included a one-year follow-up, this period may not be sufficient to fully evaluate the occurrence of long-term cardiovascular events and the long-term impact of LVR. Long-term follow-up is needed to observe the long-term prognosis of patients and to explore the long-term impact of early and late LVR on patient outcomes.

Conclusions

After undergoing PCI, more than half of patients with STEMI develop early LVR. Using echocardiography to evaluate these patients, we found that early LVR is associated with lower left ventricular systolic function, more segments with abnormal movement, and lower absolute strain values in the three layers of the left ventricular wall myocardium. However, there was a compensatory increase in the absolute LASRct value. Patients with early LVR also exhibited a higher incidence of MACE at the one-year follow-up, underscoring the need for clinical vigilance regarding early LVR.

Notably, some patients developed late LVR after 6 months, which was not completely correlated with early LVR. The incidence of MACE was also higher in patients with late LVR. The three-layer myocardial strain of the left ventricular wall, or a combination with the LASRr, has predictive value for advanced LVR. Accurate monitoring of myocardial deformation function using echocardiographic layered strain technology is expected to become a reference indicator for clinical diagnosis and treatment.

LVR is not an isolated phenomenon; it is closely associated with the left atrium and mitral valve. In addition to focusing on LVR, it is crucial to monitor the structure and function of the left atrium and mitral valve. This approach holds significant value for prognostic assessment and in the formulation of diagnostic and treatment plans.

Availability of Data and Materials

The data that support the findings of this study are included in this manuscript, and the original files are available from the corresponding author upon reasonable request.

Author Contributions

LZ, MB, ZZ carried out the study. LZ, AD performed the analysis. ZZ supervised the project and wrote the manuscript. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work to take public responsibility for appropriate portions of the content and agreed to be accountable for all aspects of the work in ensuring that questions related to its accuracy or integrity.

Ethics Approval and Consent to Participate

Written informed consent and research authorizations were obtained preoperatively from all participants. The study was performed in accordance with the Declaration of Helsinki of Good Clinical Practice. An institutional review committee approved the study protocol at the First Hospital of Lanzhou University. The Ethics Committee Approval Letter Number was LDYYLL2021-16.

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Conflict of Interest

The authors declare no conflict of interest.

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