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Predictive Value of the Inflammatory Burden Index Combined with Multimodality Imaging for Left Ventricular Remodeling After Primary PCI in Patients with STEMI

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Article

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Abstract

Background

Left ventricular remodeling (LVR) remains common after primary percutaneous coronary intervention (PPCI) for ST-segment elevation myocardial infarction (STEMI) and is linked to heart failure.

Objective

To determine whether the inflammatory burden index (IBI) adds predictive value to multimodality imaging for LVR after PPCI.

Methods

Consecutive STEMI patients undergoing PPCI (January–December 2024) were screened. Transthoracic echocardiography (TTE), 3-dimensional speckle-tracking echocardiography (3D-STE), and myocardial work (MW) were performed within 24 hours; cardiac magnetic resonance within 7 days quantified infarct size (IS). Imaging was repeated at 6 months. LVR was defined as a ≥ 20% increase in left ventricular end-diastolic volume (LVEDV).

Results

Ninety patients were included (LVR n = 22; nonLVR n = 68). IBI was higher in the LVR group ($p < 0.05$). IBI (AUC 0.898), left ventricular ejection fraction (LVEF; AUC 0.863), global longitudinal strain (GLS; AUC 0.754), global wasted work (GWW; AUC 0.861), and IS (AUC 0.809) predicted LVR (all $p < 0.05$). Adding IBI improved discrimination: IBI+LVEF (AUC 0.968), IBI + GLS (AUC 0.960), IBI + GWW (AUC 0.937), and IBI + IS (AUC 0.947) (all $p < 0.05$). LVEDV difference(ΔLVEDV) correlated linearly with IBI in the LVR group($r = 0.690$, $P < 0.001$).

Conclusions

IBI is a strong predictor of LVR after PPCI in STEMI, and combining IBI with multimodality imaging outperforms any single marker.

Introduction

According to the World Health Organization (WHO), ischemic heart disease (IHD) remains a leading cause of death worldwide. Its pathological basis is the progressive deposition of lipids, inflammatory cells, and other components within the coronary arterial wall, forming atherosclerotic plaques that cause luminal narrowing or functional abnormalities[1]. ST-segment elevation myocardial infarction (STEMI) is among the most lethal manifestations of IHD[2]. The pathogenesis of STEMI involves plaque rupture, platelet aggregation, and thrombus formation, leading to acute and complete coronary occlusion[3]. Early recognition of STEMI and timely myocardial reperfusion are crucial for reducing complications, morbidity, and mortality[4]. Primary percutaneous

coronary intervention (PPCI) is widely used and substantially improves survival; however, STEMI management remains challenging. Extensive myocardial injury can trigger ventricular remodeling, malignant arrhythmias, cardiogenic shock, and sudden death, resulting in persistently high in-hospital and long-term mortality[5]. Post-STEMI left ventricular remodeling (LVR) is a key determinant of long-term prognosis. Therefore, identifying risk factors for LVR after STEMI is clinically important.

Inflammation plays a pivotal role in STEMI onset and progression. The evolution of vulnerable plaques is characterized by inflammatory changes, and inflammatory mediators contribute to the development of atherosclerosis and plaque instability[6]. Local inflammation (e.g., monocyte infiltration) and systemic inflammation (e.g., elevated C-reactive protein [CRP]) may both promote STEMI[7]. The inflammatory burden index (IBI) is a composite measure integrating CRP, neutrophils, and lymphocytes, calculated as IBI = CRP × neutrophil count / lymphocyte count. Compared with single inflammatory biomarkers, IBI may better capture the systemic inflammatory state and has been used for risk stratification across multiple diseases[8,9]. Prior studies have reported associations between IBI and cardiovascular disease[10].

Transthoracic echocardiography (TTE) is the primary noninvasive imaging modality for acute coronary syndrome because of its availability, versatility, and safety. It can help relate regional wall-motion abnormalities to culprit coronary territories. However, TTE is operator-dependent and may not accurately capture complex early functional changes after STEMI[11]. Three-dimensional speckle-tracking echocardiography (3D-STE) enables three-dimensional assessment of myocardial deformation by tracking speckle motion, offering more comprehensive evaluation than conventional 2D echocardiography[12]. Myocardial work (MW) quantifies the mechanical energy generated during the cardiac cycle and reflects both contractile performance and energetic efficiency[13]. 3D-STE-derived MW provides clinically useful information in heart failure, cardiomyopathies, and ischemic heart disease.

Cardiac magnetic resonance (CMR) is highly sensitive for early ischemic signs in STEMI. Late gadolinium enhancement (LGE) can identify ischemic patterns accompanied by edema, helping estimate infarct age and localize infarction[14,15]. However, in very early STEMI, edema and partial-volume effects may enlarge abnormal tissue regions, and LGE may overestimate infarct size before scar formation[16].

This study aimed to evaluate the predictive value of IBI combined with multimodality imaging for LVR after PPCI in patients with STEMI.

Materials and Methods

Study population

This single-center observational study enrolled patients with first-episode STEMI hospitalized at Lianyungang First People's Hospital between January 2024 and December 2024. Inclusion criteria were: (1) age 18–75 years; (2) STEMI diagnosis according to current guideline definitions[17]; (3) PPCI within 12 h of symptom onset with post-procedural TIMI grade 3 flow. Exclusion criteria were: (1) prior myocardial infarction, prior PPCI, or coronary artery bypass grafting; (2) congenital heart disease, valvular heart disease, or severe cardiomyopathy; (3) persistent arrhythmia at admission (e.g., persistent atrial fibrillation/flutter); (4) prior use of medications known

to affect remodeling (e.g., certain corticosteroids or immunosuppressants); (5) severe systemic disease (e.g., end-stage renal disease, malignancy, acute infection) that could confound outcomes; (6) cardiac surgery or interventional therapy within the preceding 6 months. This study was approved by the the Ethics Committee of Lianyungang First People's Hospital (KY-20250213001-01). The requirement for informed consent was waived by the same committee because the study posed no additional risk to participants. All methods were performed in accordance with the relevant guidelines and regulations.

Clinical and laboratory data

Baseline data included sex, age, history of hypertension and diabetes, and post-procedural medications. Laboratory tests included high-sensitivity C-reactive protein (hsCRP), neutrophil count, lymphocyte count, lipid profile [TC, TG, LDL-C, HDL-C, Lp(a)], liver enzymes (ALT, AST), high-sensitivity troponin I, CK, CK-MB, and NT-proBNP. The culprit vessel was determined by coronary angiography.

Echocardiography, 3D-STE, and myocardial work

Within 24 h after PPCI, all patients underwent TTE, 3D-STE, and MW assessment using a GE Vivid E95 system (GE Vingmed Ultrasound, Horten, Norway). Parameters included left ventricular ejection fraction (LVEF), LV end-diastolic volume (LVEDV), LV end-systolic volume (LVESV), global longitudinal strain (GLS), global radial strain (GRS), global circumferential strain (GCS), global area strain (GAS), and MW indices: global work index (GWI), global constructive work (GCW), global wasted work (GWW), and global work efficiency (GWE).(Fig. 1A-F)

Cardiac magnetic resonance

Within 7 days after PPCI, CMR was performed on a 1.5-T scanner (Magnetom Avanto, Siemens, Germany) to quantify infarction size (IS).

Follow-up and definition of LVR

At 6 months, TTE/3D-STE/MW were repeated. LVR was defined as an increase in LVEDV ≥ 20% compared with baseline by TTE[18].

Statistical analysis

SPSS (version 22.0) and R(version 4.3.3) were used for statistical analyses. Continuous variables are presented as mean ± SD or median (interquartile range) as appropriate; categorical variables are presented as percentages. Between-group comparisons used independent-samples t-tests for normally distributed variables, Mann–Whitney U tests for non-normal variables, and χ^2 tests for categorical variables. Univariable and multivariable logistic regression identified independent predictors of LVR. Receiver operating characteristic (ROC) curves were constructed to estimate sensitivity, specificity, and AUC; optimal cut-offs were determined by the Youden index. Pearson correlation and linear regression were used to assess associations between variables. Restricted cubic splines (RCS) were used to explore the dose–response relationship between IBI and ΔLVEDV. A two-sided $P < 0.05$ was considered statistically significant.

Results

A total of 90 patients were included after excluding incomplete data and losses to follow-up. Patients were divided into the LVR group (n = 22) and the non-LVR group (n = 68) according to the change in LVEDV. Baseline demographics, laboratory results, angiographic findings, and medications are summarized in Table 1. NT-proBNP levels were higher in the LVR group. Angiography showed the left anterior descending artery (LAD) more frequently as the culprit vessel in the LVR group, whereas the right coronary artery (RCA) was more common in the non-LVR group (both $P < 0.05$). Neutrophil-to-lymphocyte ratio (NLR = neutrophils/lymphocytes), platelet-to-lymphocyte ratio (PLR = platelets/lymphocytes), and IBI (IBI = hsCRP × neutrophils/lymphocytes) were higher in the LVR group, with significant differences for NLR and IBI ($P < 0.05$).

Table 1
Baseline Characteristics

Variables	nonLVR(n = 68)	LVR(n = 22)	P
Male(female)	56(82.35%)	17(77.27)	0.597
Age	59.06 ± 10.32	57.23 ± 10.69	0.475
Hypertension	41(27)	12(10)	0.634
Diabetes	25(43)	10(12)	0.467
TC(mmol/L)	4.93 ± 1.37	4.87 ± 1.42	0.856
TG(mmol/L)	1.365(0.7975, 2.035)	1.7(1.1925, 2.86)	0.073
HDL-C(mmol/L)	1.03(0.89, 1.3125)	1.02(0.8775, 1.125)	0.295
LDL-C(mmol/L)	3.14 ± 0.97	3.01 ± 0.86	0.597
LP(a)(mmol/L)	218.07 ± 173.97	195.41 ± 152.74	0.586
ALT(U/L)	61.26 ± 51.44	66.23 ± 36.17	0.676
AST(U/L)	248.54 ± 154.01	288.23 ± 163.87	0.304
hs-cTnI(pg/ml)	23421.19 ± 5793.68	23115.63 ± 5698.39	0.83
NT-proBNP(pg/ml)	526.00(268.75,944.25)	712.00(459.5,2085)	0.038
CK(U/L)	2727.74 ± 1743.47	3125.18 ± 2208.01	0.387
CK-MB(ng/ml)	261.05 ± 247.64	295.21 ± 370.48	0.622
hsCRP(mg/L)	14.51 ± 6.43	18.36 ± 6.11	0.16
Neutrophil(10^9 /L)	10.96 ± 3.39	12.36 ± 3.82	0.107
Lymphocyte(10^9/L)	1.16 ± 0.45	0.99 ± 0.2	0.086
IBI	132.95 ± 36.5	225.1 ± 69.28	<0.001
NLR	10.27 ± 3.79	13.19 ± 5.29	0.006
PLR	209.64 ± 83.21	233.25 ± 82.34	0.249
IRA-LAD	25(36.76%)	19(86.36%)	<0.001
IRA-RCA	38(55.88%)	1(4.55%)	<0.001
IRA-LCX	5(7.35%)	2(9.09%)	0.791

TC: Total Cholesterol; TG: Triglycerides; HDL: High-Density Lipoprotein (Cholesterol); LDL: Low-Density Lipoprotein (Cholesterol); Lp(a): Lipoprotein(a); ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; hs-cTnI: High-Sensitivity Cardiac Troponin I; CK: Creatine Kinase; CK-MB: Creatine Kinase-MB (Isoenzyme); NLR: Neutrophil-to-Lymphocyte Ratio; PLR: Platelet-to-Lymphocyte Ratio; IRA: Infarct-Related Artery; LAD: Left Anterior Descending (Artery); RCA: Right Coronary Artery; LCX: Left Circumflex (Artery); MRA: Mineralocorticoid Receptor Antagonist

Variables	nonLVR(n = 68)	LVR(n = 22)	P
SGLT2i	34(50%)	14(63.63%)	0.265
MRA	10(17.24%)	3(13.64%)	0.901
Aspirin	65(95.59%)	21(95.45%)	0.979
Ticagrelor	66(97.06%)	22(100%)	0.416
Statins	66(97.06%)	22(100%)	0.416
β-blockers	57(83.82%)	21(95.45%)	0.163
ACEI/ARNI/ARB	54(79.41%)	20(90.91%)	0.220

TC: Total Cholesterol; TG: Triglycerides; HDL: High-Density Lipoprotein (Cholesterol); LDL: Low-Density Lipoprotein (Cholesterol); Lp(a): Lipoprotein(a); ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; hs-cTnI: High-Sensitivity Cardiac Troponin I; CK: Creatine Kinase; CK-MB: Creatine Kinase-MB (Isoenzyme); NLR: Neutrophil-to-Lymphocyte Ratio; PLR: Platelet-to-Lymphocyte Ratio; IRA: Infarct-Related Artery; LAD: Left Anterior Descending (Artery); RCA: Right Coronary Artery; LCX: Left Circumflex (Artery); MRA: Mineralocorticoid Receptor Antagonist

Comparison of imaging parameters

At baseline, LVEF was lower in the LVR group ($P < 0.05$). For 3D-STE, GLS and GAS were lower in the LVR group ($P < 0.05$). For MW, GWI and GWE were lower and GWW was higher in the LVR group (all $P < 0.05$). IS measured by CMR was higher in the LVR group ($P < 0.05$, Table 2).

Table 2
Baseline Imaging Parameters

Variables	nonLVR(n = 68)	LVR(n = 22)	P
TTE			
LVEF(%)	52.59 ± 4.81	45.32 ± 3.66	<0.001
LVEDV(ml)	122.97 ± 13.61	126.55 ± 11.76	0.272
LVESV(ml)	59.51 ± 10.17	64.31 ± 9.93	0.056
3D-STE(%)			
GLS	-10.35 ± 1.53	-8.5 ± 2.11	<0.001
GCS	-12.32 ± 4.46	-11.23 ± 1.57	0.263
GAS	-19.41 ± 4.73	-16.05 ± 2.87	0.002
GRS	28.25 ± 7.77	24.82 ± 4.8	0.054
MW(mmHg)			
GWI	1453.43 ± 265.67	1208.36 ± 144.03	<0.001
GCW	1563.81 ± 509.31	1378.59 ± 294.96	0.11
GWW	162.47 ± 77.99	261.14 ± 48.16	<0.001
GWE(%)	86.12 ± 6.44	80.23 ± 4	<0.001
CMR			
IS(%)	22.15 ± 5.16	28.83 ± 6.16	<0.001

TTE: transthoracic echocardiography; LVEF: left ventricular ejection fraction; LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume; GLS: global longitudinal strain; GCS: global circumferential strain; GAS: global area strain; GRS: global radial strain; MW: myocardial work; GWI: global work index; GCW: global constructive work; GWW: global wasted work; GWE: global work efficiency; CMR: cardiac magnetic resonance; IS: infarction size.

At 6 months, the LVR group had lower LVEF and higher LVEDV and LVESV ($P < 0.05$). GLS remained lower in the LVR group ($P < 0.05$). MW indices showed lower GWI, GCW, and GWE and higher GWW in the LVR group (all $P < 0.05$, Table 3).

Table 3
Followup Imaging Parameters

Variables	nonLVR(n = 68)	LVR(n = 22)	P
TTE			
LVEF(%)	51.09 ± 5.98	41.09 ± 3.7	<0.001
LVEDV(ml)	126.71 ± 13.87	172.68 ± 7.09	<0.001
LVESV(ml)	66.62 ± 11.71	87.65 ± 6.7	<0.001
3D-STE(%)			
GLS	-11.15 ± 1.83	-9.95 ± 2.04	0.011
GCS	-12.84 ± 4.4	-12.32 ± 1.89	0.593
GAS	-20.21 ± 4.42	-18.45 ± 4.52	0.112
GRS	29.72 ± 6.1	28 ± 5.48	0.304
MW(mmHg)			
GWI	1570.65 ± 236.98	1296.45 ± 120.56	<0.001
GCW	1753.93 ± 454.51	1472.86 ± 263.96	0.007
GWW	135.46 ± 67.42	172.41 ± 46.3	0.019
GWE(%)	88.06 ± 4.61	81.55 ± 3.19	<0.001

Single and multiple logistic regression analysis

In univariable logistic regression, hsCRP, NLR, IBI, LVEF, GLS, GAS, GWI, GWW, GWE, and IS were associated with LVR ($P < 0.05$, Fig. 2A). In multivariable analysis, IBI, LVEF, GLS, GWW, and IS were independent predictors of LVR ($P < 0.05$, Fig. 2B).

ROC analysis

ROC analysis showed AUCs of 0.898 for IBI (95%CI 0.828–0.969), 0.863 for LVEF (95%CI 0.774–0.953), 0.754 for GLS (95%CI 0.621–0.887), 0.861 for GWW (95%CI 0.785–0.937), and 0.809 for IS (95%CI 0.691–0.928) (Fig. 3, Table 4). Joint models incorporating IBI with LVEF, GLS, GWW, or IS showed higher AUCs than individual predictors (Fig. 4, Table 5).

Table 4
ROC curve analysis of each parameter to predict LVR

Variables	AUC	Cutoff	Sensitivity	Specificity	Youden's index	PPV	NPV	95%CI	P
IBI	0.898	148.79	0.909	0.706	0.615	0.500	0.960	0.828–0.969	<0.001
LVEF	0.863	50.5	0.955	0.662	0.617	0.583	0.981	0.774–0.953	<0.001
GLS	0.754	-7.5	0.409	1	0.409	1.000	0.840	0.621–0.887	<0.001
GWW	0.861	199	0.909	0.735	0.644	0.526	0.962	0.785–0.937	<0.001
IS	0.809	27.65	0.727	0.868	0.595	0.640	0.908	0.691–0.928	<0.001
PPV: positive predictive value; NPV: negative predictive value									

Table 5
ROC curve analysis of combined parameters to predict LVR

Variables	AUC	Sensitivity	Specificity	95%CI	P
IBI+LVEF	0.968	0.955	0.882	0.937–0.999	<0.001
IBI + GLS	0.96	0.909	0.882	0.924–0.996	<0.001
IBI + GWW	0.937	0.909	0.809	0.886–0.989	<0.001
IBI + IS	0.947	0.955	0.809	0.904–0.991	<0.001

Correlation between IBI and ΔLVEDV.

In the LVR group, IBI was positively correlated with the change in LVEDV(ΔLVEDV). Shapiro–Wilk test suggested approximate normality for IBI and ΔLVEDV (IBI: P = 0.205, ΔLVEDV = 0.188). In the primary analysis, Pearson correlation showed a significant linear association (r = 0.690, P < 0.001), simple linear regression yielded: ΔLVEDV = 26.62 + 0.0867× IBI(R^2 = 0.476, P < 0.001). Consistently, the association remained significant using Spearman rank correlation (ρ = 0.570, P = 0.005), indicating robustness to potential non-normality or outliers. In addition, LOESS smoothing demonstrated an overall increasing trend of ΔLVEDV with higher IBI, without an obvious departure from linearity. Restricted cubic spline (RCS) analysis demonstrated a significant dose–response association between IBI and ΔLVEDV (overall P < 0.001), while there was no evidence of a nonlinear relationship (P for nonlinearity = 0.210), suggesting that the association was largely consistent with a linear trend.(Fig. 5A-5D).

Discussion

STEMI is a high-risk form of coronary heart disease characterized by abrupt onset, severe myocardial injury, and poor prognosis[19]. Although advances in PPCI have improved infarct-artery patency[20], a substantial proportion of patients still develop complications such as arrhythmias and heart failure. LVR is a central pathological

process influencing outcomes after acute myocardial infarction[21]. Thus, early identification of predictors of post-STEMI LVR is crucial.

In this study, IBI and multimodality imaging (TTE, 3D-STE/MW, and CMR) were evaluated for predicting LVR after PPCI. We found that IBI, LVEF, GLS, GWW, and IS were independent predictors of LVR, and that IBI was linearly associated with the extent of remodeling (ΔLVEDV). These findings support early risk stratification and proactive optimization of therapeutic strategies.

Inflammation is a key driver of post-infarction remodeling. Ischemia–reperfusion injury triggers systemic inflammatory responses and local immune activation[22]. Inflammatory cell infiltration and cytokine release promote cardiomyocyte apoptosis, interstitial fibrosis, and disordered collagen metabolism, leading to ventricular dilation, spherical remodeling, and functional decline[22]. As an integrated peripheral inflammatory biomarker, IBI captures inflammatory–immune imbalance more comprehensively than single indices such as NLR and PLR and may provide superior value for cardiovascular risk stratification[23]. In our cohort, NLR differed between groups but was not an independent predictor in multivariable models, whereas elevated IBI was strongly associated with LVR risk, consistent with prior literature.

Imaging plays a complementary role in detecting early myocardial dysfunction and structural change. TTE is convenient and widely available but may miss subtle early remodeling and is operator-dependent[24]. 3D-STE quantifies multidimensional myocardial deformation without relying on geometric assumptions and can identify subclinical dysfunction even when LVEF is preserved[25]. In our study, early impairment in GLS and GAS was observed in the LVR group, and GLS remained an independent predictor of LVR, underscoring its prognostic utility.

MW reflects myocardial energetic efficiency and integrates deformation with estimated afterload. After STEMI, MW abnormalities correlate with ventricular dilation and adverse remodeling[26]. Here, MW indices differed between groups at both baseline and follow-up, and GWW emerged as an independent predictor, highlighting the relevance of wasted work to adverse remodeling.

CMR with LGE provides high-resolution quantification of infarct size and is regarded as a noninvasive reference standard[27]. We found that IS was an independent predictor of LVR, supporting the role of CMR for risk stratification. Nevertheless, limited availability, cost, and patient tolerance restrict broader use.

Importantly, combining IBI with imaging markers substantially improved predictive performance compared with single indicators, suggesting strong complementarity between systemic inflammatory burden (IBI), functional impairment (strain and MW), and myocardial injury burden (CMR-derived IS). This multidimensional approach may help identify high-risk patients early and inform the timing and intensity of anti-remodeling interventions.

Limitations

This was a single-center observational study with a modest sample size, which may explain extreme estimates in some metrics (e.g., specificity/PPV for GLS). Dynamic monitoring of IBI was not performed, and long-term major adverse cardiovascular events were not assessed. Larger multicenter prospective studies incorporating

serial inflammatory measurements are needed to refine prediction models and evaluate clinical benefits of inflammation-targeted and cardioprotective therapies.

Conclusions

IBI is a useful biomarker for predicting LVR after PPCI in STEMI. Combining IBI with multimodality imaging (TTE, 3D-STE/MW, and CMR) yields superior predictive value and may support noninvasive, efficient early risk stratification and individualized management.

Declarations

Competing interests

No conflicts of interest were declared by the authors.

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Author Contribution

Mingzhu Li: Study Design, Data Collection, Statistical Analysis, Data Interpretation, Literature Search. Jinshan Li: Statistical Analysis, Data Interpretation, Manuscript Preparation, Literature Search. Pengying zhang: Data Collection, Data Interpretation, Manuscript Preparation, Literature Search. Xiaoxuan Gong: Manuscript Preparation, Literature Search, Funds Collection. Kun Liu: Study Design, Data Collection, Statistical Analysis, Data Interpretation, Manuscript Preparation.

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Figures

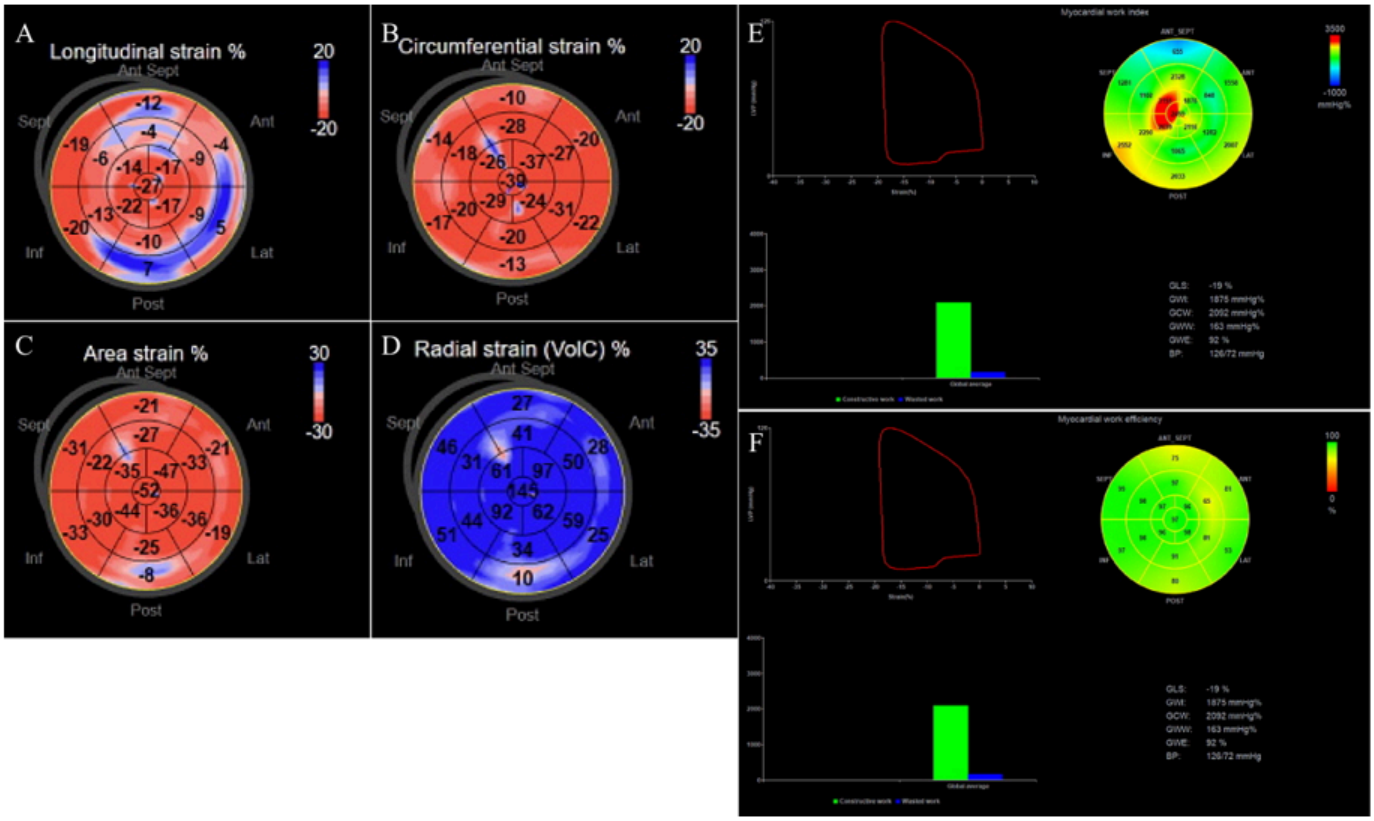


Figure 1

A-D: The strain parameters(GLS,GCS,GAS,GRS) are calculated by the software and presented in the form of 17-segements

E-F: The left ventricular pressure-strain loop (LV-PSL) was used to obtain global myocardial work indices (GWI, GCW, GWW, and GWE).

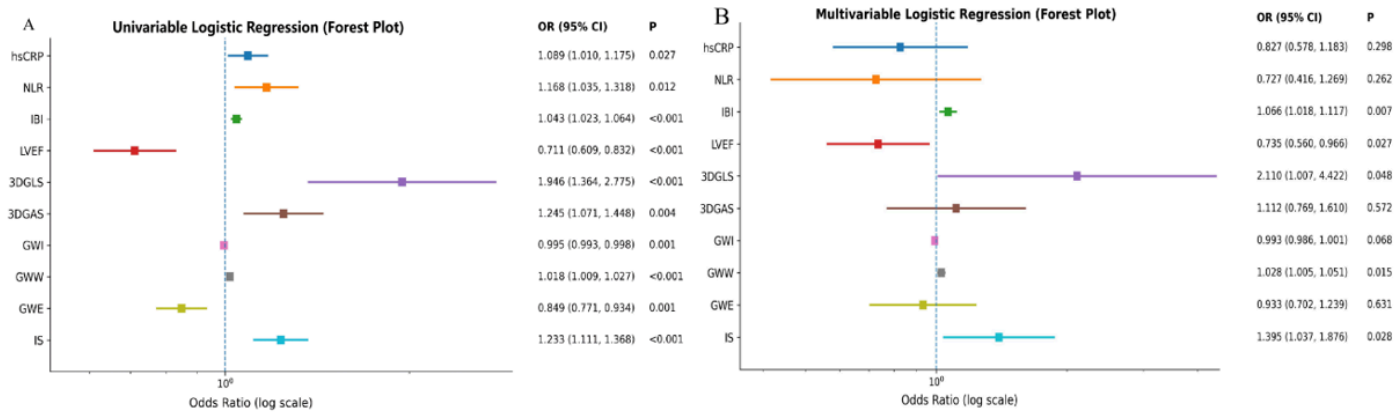


Figure 2

Univariable Logistic(A) and Multivariate Logistic(B)

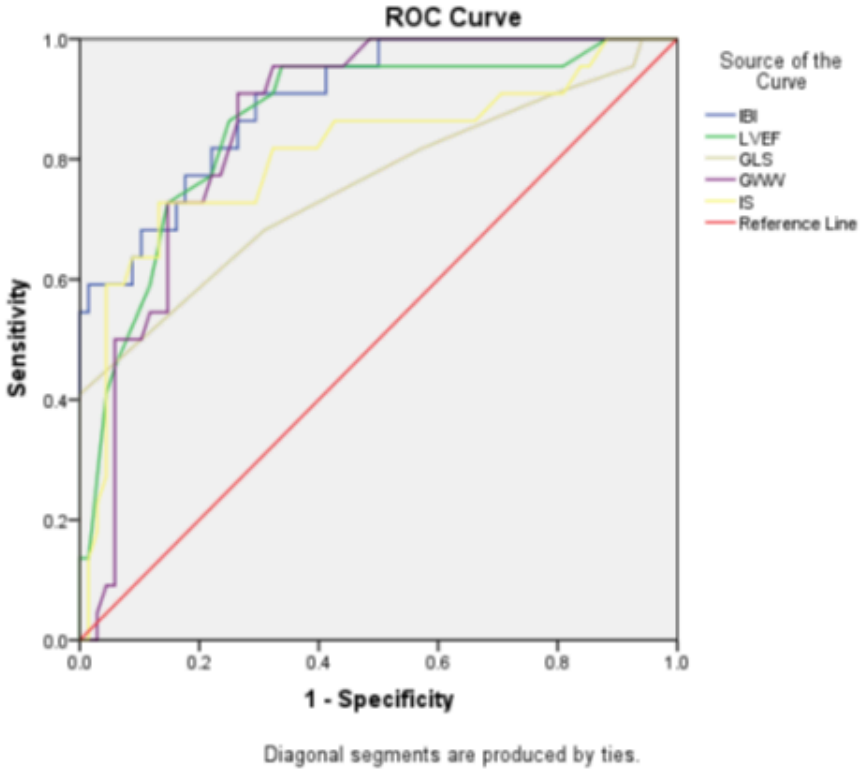


Figure 3

ROC curves of each parameter in predicting LVR

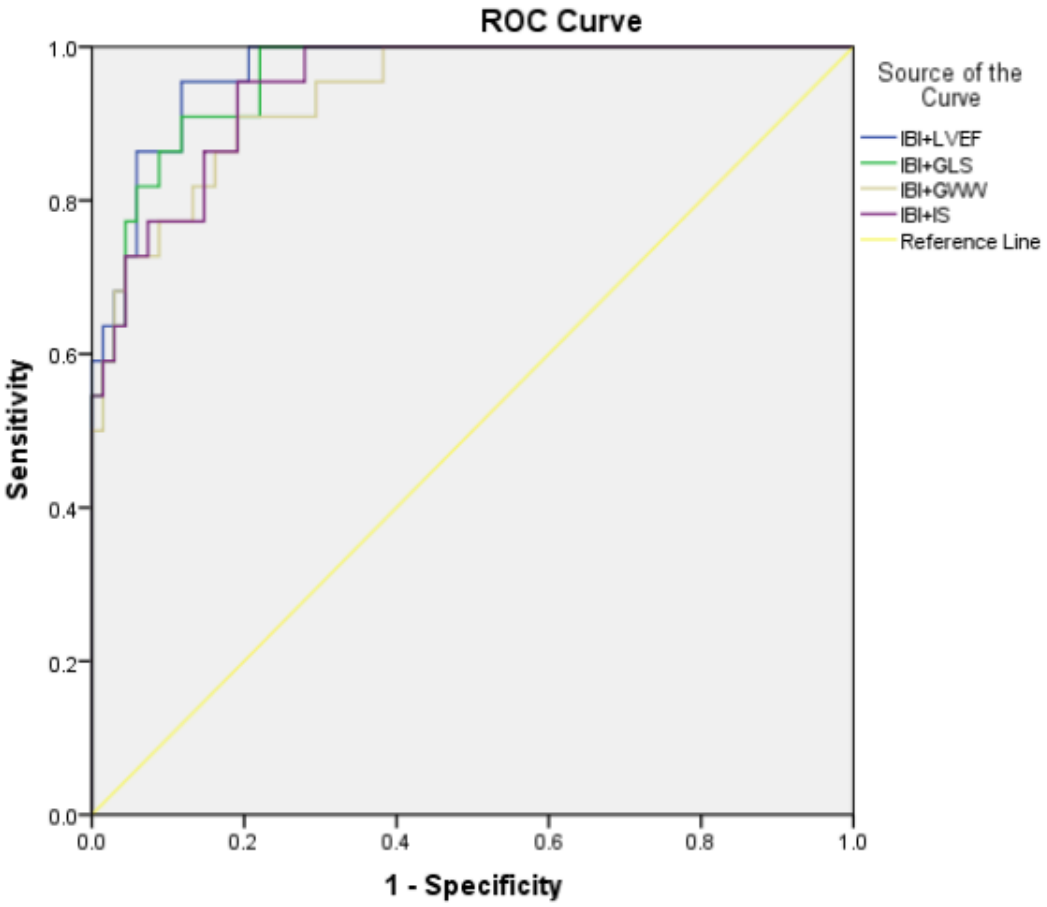


Figure 4

ROC curves of combined parameters in predicting LVR

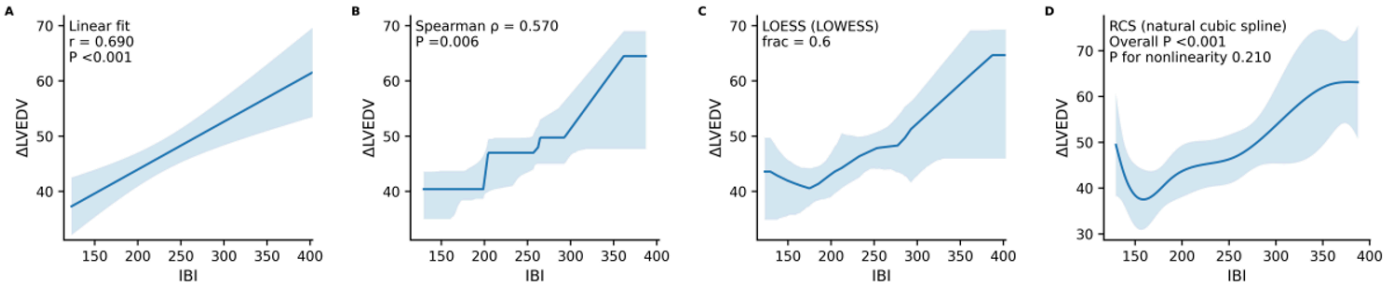


Figure 5

Relationship between IBI and ΔLVEDV in the LVR group.
(A) Linear regression line and Pearson correlation, (B) Rank-based association with Spearman correlation, (C) LOESS-smoothed curve, (D) Dose-response relationship from RCS analysis.(shaded area indicates 95% CI)