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Original Research

Association of Metabolic Indices With Left Ventricular Remodeling After Percutaneous Coronary Intervention in Patients With Acute Myocardial Infarction

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Abstract

Background: Left ventricular (LV) remodeling after acute myocardial infarction (AMI) remains a key determinant of long-term prognosis. Insulin resistance (IR) has increasingly been recognized as an important metabolic mechanism influencing post-infarction myocardial recovery. This study aimed to evaluate the associations of three noninsulin-based metabolic indices, the triglyceride–glucose (TyG) index, triglyceride-glucose body mass index (TyG–BMI), and the metabolic score for insulin resistance (METS-IR)—with LV remodeling after percutaneous coronary intervention (PCI). **Methods:** A total of 1180 patients with AMI who underwent PCI and completed baseline and 6-month follow-up echocardiography were retrospectively analyzed. LV remodeling was defined as a ≥20% increase in LV end-diastolic volume. Multiple logistic regression and restricted cubic spline (RCS) analyses were used to assess linear associations, and stratified analyses were conducted across key clinical subgroups. **Results:** During the 6-month follow-up, 269 patients (22.8%) developed LV remodeling. After adjustment for confounders, higher levels of TyG, TyG–BMI, and METS-IR were independently and positively associated with the risk of LV remodeling (TyG: odds ratio (OR) = 1.67, 95% confidence interval (CI) 1.24–2.26; TyG–BMI: OR = 1.01, 95% CI 1.00–1.01; METS-IR: OR = 1.04, 95% CI 1.00–1.07). RCS analyses revealed linear positive associations with no evidence of nonlinearity. Stratified analyses showed consistent results across all subgroups (all interaction p-values > 0.05). **Conclusion:** Higher levels of TyG, TyG–BMI, and METS-IR are independently and positively associated with LV remodeling in patients with AMI undergoing PCI.

Keywords: acute myocardial infarction; left ventricular remodeling; TyG; TyG–BMI; METS-IR

1. Introduction

Acute myocardial infarction (AMI) remains one of the leading causes of morbidity and mortality worldwide, despite significant advances in reperfusion therapy, such as percutaneous coronary intervention (PCI) [1]. While the acute mortality rate has decreased significantly with PCI, a substantial proportion of patients continue to experience left ventricular (LV) remodeling after reperfusion, which can lead to heart failure, arrhythmias, and other adverse events, severely impacting long-term prognosis [2]. Therefore, early identification of and timely intervention in patients at high risk of left ventricular remodeling has become critical in clinical management.

The mechanisms underlying LV remodeling are complex and involve multiple factors, including the extent of myocardial injury, inflammation, neurohormonal activation, and metabolic abnormalities [3]. Among these, insulin resistance (IR) has emerged as a key metabolic driver of adverse LV remodeling [4]. Traditional measures of IR, such as homeostatic model assessment of insulin resistance (HOMA-IR), have certain predictive value, but their clinical applicability is limited by the need for insulin assays. Recently, several non-insulin-based metabolic indices have been proposed, such as the triglyceride-glucose (TyG) index, TyG-body mass index (TyG–BMI), and the metabolic score for insulin resistance (METS-IR), which are based on routine laboratory parameters and are both simple and reliable [5,6,7,8]. However, existing research has predominantly focused on long-term prognosis or ischemic outcomes, and very few studies have investigated whether these indices can predict post-AMI LV remodeling—a critical structural intermediate phenotype preceding heart failure. Moreover, to date, no study has systematically compared all three commonly used metabolic indices (TyG, TyG–BMI, and METS-IR) in the context of post-infarction LV remodeling, resulting in an important evidence gap regarding their relative performance and clinical applicability.

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Therefore, this study aimed to investigate the associations of TyG, TyG–BMI, and METS-IR with LV remodeling after an AMI and to evaluate their potential utility as simple, non–insulin-based markers for identifying patients at risk for adverse structural cardiac changes following PCI.

2. Methods

2.1 Study Population

This single-center, retrospective, cohort study consecutively enrolled 1180 patients diagnosed with an AMI who were hospitalized in the Department of Cardiology at Xuanwu Hospital, Capital Medical University, between January 2016 and July 2023. All enrolled patients underwent PCI and had complete echocardiographic data at baseline and at 6-month follow-up. This study was conducted in accordance with the Declaration of Helsinki and was approved by the Ethics Committee of Xuanwu Hospital, Capital Medical University (Approval No. 2022161). The requirement for written informed consent for research participation was waived by the Ethics Committee because of the retrospective nature of the study and the use of anonymized clinical data.

2.2 Inclusion and Exclusion Criteria

Patients were eligible for inclusion if they met the diagnostic criteria for an AMI, including both ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI). The inclusion criteria required patients to be between 18 and 80 years of age, have undergone PCI at Xuanwu Hospital, and have completed both baseline and 6-month follow-up echocardiographic assessments.

Patients were excluded if they had a history of malignant tumors, were unable to undergo standard treatment, or had previously undergone heart surgery (except coronary artery bypass grafting). In addition, patients with significant structural heart diseases such as valvular heart disease, primary cardiomyopathy, congenital heart disease, or pericardial disease were excluded. Patients with missing critical clinical variables were also excluded from the study.

2.3 Clinical and Laboratory Data Collection

Baseline clinical data were retrospectively collected from the hospital information system (HIS), including age, sex, body mass index (BMI), smoking history, alcohol consumption, hypertension, diabetes, dyslipidemia, and medication history. Data on time from symptom onset to admission, time from symptom onset to vessel opening, infarct-related artery, number of diseased vessels, and PCI procedures were also recorded. Laboratory data, including fasting blood glucose (FPG), HbA1c, triglycerides (TG), total cholesterol (TC), low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), serum creatinine, urea nitrogen, alanine aminotransferase (ALT), aspartate aminotransferase (AST), and uric acid, were collected at baseline. Complete blood count data, including white blood cell count, hemoglobin, and platelet count, were also collected.

2.4 Metabolic Indices Calculation

The following metabolic indices were calculated using previously published standardized formulas: TyG index = ln[TG (mg/dL) × FPG (mg/dL) / 2] [9], TyG–BMI index = TyG × BMI [10], METS-IR = ln[(2 × FPG + TG) × BMI] / ln(HDL-C) [11]. When laboratory results were expressed in mmol/L, triglycerides were multiplied by 88.57, and FPG by 18.02 to convert to mg/dL before calculation.

2.5 Echocardiographic Assessment

All patients underwent echocardiographic assessments at baseline and 6 months after PCI, using a standardized echocardiographic protocol. Echocardiographic examinations were performed by experienced echocardiographers in accordance with the guidelines of the American Society of Echocardiography (ASE) [12]. Echocardiographers were blinded to patients' clinical information and outcome status during image interpretation. The primary parameters measured included left ventricular end-diastolic diameter (LVEDD), left ventricular end-systolic diameter (LVESD), interventricular septal thickness (IVST), and left ventricular posterior wall thickness (LVPWT). Left ventricular ejection fraction (LVEF) and left ventricular end-diastolic volume (LVEDV) were calculated using the Simpson's biplane method. In addition, baseline and follow-up echocardiographic parameters were compared between patients with and without LV remodeling, and the results are presented in **Supplementary Table 1**.

2.6 Left Ventricular Remodeling Definition

Left ventricular remodeling was defined as a ≥20% increase in LVEDV from baseline to 6 months, based on international guidelines and previous studies. Patients were divided into two groups: remodeling and non-remodeling, based on the occurrence of LV remodeling [13].

2.7 Statistical Analysis

All analyses were performed using R software, version 4.2.2 (R Foundation for Statistical Computing, Vienna, Austria), and Free Statistics software, version 2.1 (Beijing Free Clinical Medical Technology Co., Ltd., Beijing, China). Continuous variables were expressed as mean ± standard deviation ($\bar{x} \pm s$) or median (interquartile range) based on normality. Independent *t*-tests were used for normally distributed data, while the Mann–Whitney U test was used for non-normally distributed data. Categorical variables were expressed as counts (percentages), and group comparisons were performed using the χ^2 test. Multiple logistic regression was used to identify factors influencing LV remodeling in AMI patients. Covariates included in the multivariable logistic regression models were se-

lected based on clinical relevance, prior evidence on predictors of post-infarction left ventricular remodeling, results from univariable analyses, and a change-in-estimate approach. Specifically, variables were retained if their inclusion resulted in a clinically or statistically meaningful change (≥10%) in the estimated effect of the primary exposure, or if they were considered potential confounders based on clinical knowledge. Results were presented as odds ratios (ORs) with 95% confidence intervals (95% CIs). An RCS analysis based on the logistic regression model was used to explore whether metabolic indices had a nonlinear relationship with LV remodeling. Subgroup analyses assessed potential interaction effects between metabolic indices and clinical variables. Receiver operating characteristic (ROC) curve analyses were performed to assess the discriminatory ability of TyG, TyG–BMI, and METS-IR for left ventricular remodeling. The area under the curve (AUC) and 95% CIs were calculated. Multicollinearity among covariates was evaluated using the generalized variance inflation factor (GVIF), with adjusted GVIF values >5 considered indicative of substantial multicollinearity. All tests were two-tailed, and a $p < 0.05$ was considered statistically significant.

2.8 Sensitivity Analysis

Left ventricular end-diastolic volume indexed to body surface area (LVEDVi) was used to redefine left ventricular remodeling. LVEDVi was calculated as LVEDV divided by body surface area, and indexed remodeling was defined as a ≥20% increase in LVEDVi at 6 months compared with baseline. Multivariable logistic regression analyses were repeated to assess the significance of the findings.

3. Results

3.1 Baseline Characteristics of Patients

A total of 1180 patients with an AMI were included in this study, of whom 911 (77.2%) did not develop LV remodeling and 269 (22.8%) did (Table 1). There were no significant differences between the two groups in age or sex distribution, with a median age of 62.0 years and approximately 80% being male. In terms of lifestyle factors, the proportion of current smokers was significantly higher in the LV remodeling group than in the non-remodeling group ($p = 0.031$), while no significant difference was observed in alcohol consumption between the two groups ($p = 0.522$). The prevalence of hypertension and diabetes mellitus was similar between the two groups, with no statistically significant differences (both $p > 0.05$). Patients in the remodeling group had markedly higher levels of ALT and AST, as well as elevated neutrophil counts, indicating greater hepatic and inflammatory stress (all $p < 0.01$). Other biochemical indices, including lipid profiles, glucose levels, and renal function, showed no significant differences between groups. The distribution of culprit vessels and infarct location was comparable between the groups (both $p > 0.05$), with the left anterior descending artery and an anterior wall infarction being the most frequent. The use of ACEI/ARB was significantly lower in the remodeling group than in the non-remodeling group ($p = 0.012$), whereas the use of aspirin and statins was nearly universal and did not differ significantly between groups. There were no significant differences in the metabolic indices, including the TyG index, TyG–BMI, and METS-IR (all $p > 0.05$). Baseline and 6-month echocardiographic parameters are summarized in **Supplementary Table 1**.

3.2 Association Between TyG-Related Indices and Left Ventricular Remodeling After AMI

Univariable logistic regression analyses were performed to evaluate the crude associations between clinical variables and left ventricular remodeling (**Supplementary Table 2**). In these analyses, TyG, TyG–BMI, and METS-IR were not significantly associated with left ventricular remodeling, which was consistent with the baseline comparisons presented in Table 1. However, after adjustment for potential confounding factors, these indices became significantly associated with left ventricular remodeling in the multivariable models.

After adjustment for age, sex, hypertension, diabetes mellitus, systolic blood pressure (SBP), diastolic blood pressure (DBP), alcohol consumption, uric acid, HDL-C, and infarct-related artery, all three insulin resistance-related indices were independently associated with LV remodeling. Each one-standard-deviation increase in the TyG index was associated with a 1.67-fold higher risk of LV remodeling (OR = 1.67, 95% CI: 1.24–2.26, $p = 0.001$). Similarly, higher TyG–BMI (OR = 1.01, 95% CI: 1.00–1.01, $p = 0.008$) and METS-IR (OR = 1.04, 95% CI: 1.00–1.07, $p = 0.048$) were also independently associated with increased odds of LV remodeling. These findings indicate that insulin resistance-related metabolic indices, particularly the TyG index, are independently associated with adverse LV remodeling after an AMI, irrespective of traditional cardiovascular risk factors (Table 2).

After further adjustment for the use of ACEI/ARB, the associations of TyG and TyG–BMI with left ventricular remodeling remained statistically significant, whereas the association for METS-IR was attenuated and no longer statistically significant (**Supplementary Table 5**). Notably, the direction of the association for METS-IR remained consistent with the primary analysis, although the effect size was reduced after additional adjustment. After further adjustment for infarct severity and baseline cardiac function, including peak CK-MB, Killip class, emergency PCI status, and baseline LVEF, the associations between TyG-related indices and left ventricular remodeling remained unchanged (**Supplementary Table 6**). Using BSA-indexed LV end-diastolic volume (LVEDVi) to define left ventricular remodeling, patients with remodeling showed significantly greater increases in indexed LV volumes (**Supple-**

Table 1. Baseline characteristics of patients with and without left ventricular remodeling after acute myocardial infarction.

Variables	Total (n = 1180)	Without remodeling (n = 911)	With remodeling (n = 269)	*p* value
Demographics				
Age, years	62.0 (55.0, 69.0)	62.0 (55.0, 69.0)	62.0 (53.8, 69.0)	0.486
Male, n (%)	950 (80.5)	734 (80.6)	216 (80.3)	0.921
Lifestyle factors				
Current smoking, n (%)	502 (60.3)	376 (58.3)	126 (67.0)	0.031
Alcohol drinking, n (%)	606 (73.2)	474 (73.7)	132 (71.4)	0.522
Comorbidities				
Hypertension, n (%)	653 (57.2)	502 (56.7)	151 (58.8)	0.562
Diabetes mellitus, n (%)	399 (33.8)	320 (35.1)	79 (29.4)	0.079
Laboratory findings				
Uric acid, μmol/L	347.0 (287.0, 416.0)	343.0 (287.0, 411.0)	365.0 (287.0, 434.0)	0.057
HDL-C, mmol/L	1.054 ± 0.292	1.048 ± 0.296	1.072 ± 0.280	0.255
LDL-C, mmol/L	2.705 ± 0.815	2.705 ± 0.833	2.707 ± 0.752	0.964
ApoA-I, g/L	1.112 ± 0.207	1.107 ± 0.207	1.129 ± 0.208	0.121
ApoB, g/L	0.919 ± 0.245	0.921 ± 0.254	0.911 ± 0.215	0.574
Triglycerides, mmol/L	1.505 (1.040, 2.170)	1.480 (1.035, 2.150)	1.580 (1.050, 2.220)	0.275
Total cholesterol, mmol/L	4.338 ± 0.996	4.334 ± 1.020	4.354 ± 0.914	0.768
Fasting glucose, mmol/L	6.2 (5.2, 8.2)	6.1 (5.2, 8.0)	6.4 (5.3, 8.6)	0.115
HbA1c, %	6.5 ± 1.5	6.5 ± 1.5	6.5 ± 1.6	0.597
ALT, U/L	28.0 (18.0, 49.0)	27.0 (18.0, 46.0)	35.0 (19.0, 65.0)	<0.001
AST, U/L	65.0 (28.0, 164.0)	56.0 (27.0, 147.5)	97.0 (36.0, 235.0)	<0.001
Urea, mmol/L	5.6 (4.5, 7.1)	5.5 (4.4, 7.0)	5.7 (4.7, 7.2)	0.173
hs-CRP, mg/L	5.2 (1.9, 16.7)	5.1 (1.9, 16.8)	5.4 (1.9, 16.6)	0.703
Neutrophil count, $\times 10^9$/L	6.8 (5.0, 9.3)	6.7 (4.9, 9.2)	7.5 (5.4, 9.8)	0.006
Platelet count, $\times 10^9$/L	219.0 (181.0, 262.2)	219.0 (181.0, 260.0)	218.0 (182.0, 265.0)	0.879
Lymphocyte count, $\times 10^9$/L	1.6 (1.1, 2.1)	1.6 (1.1, 2.2)	1.6 (1.1, 2.0)	0.429
Peak CK-MB, U/L	41.9 (11.1, 135.8)	34.0 (10.0, 119.0)	76.0 (17.8, 204.0)	<0.001
Infarct-related features				
Infarct-related artery, n (%)				0.784
LAD	502 (55.5)	383 (55.3)	119 (56.4)	
RCA	300 (33.2)	229 (33.0)	71 (33.6)	
LCX	102 (11.3)	81 (11.7)	21 (10.0)	
Number of diseased vessels, n (%)				0.579
Single-vessel	132 (42.7)	105 (44.3)	27 (37.5)	
Double-vessel	85 (27.5)	64 (27.0)	21 (29.2)	
Triple-vessel	92 (29.8)	68 (28.7)	24 (33.3)	
Infarct location, n (%)				0.855
Anterior wall	324 (49.3)	242 (49.8)	82 (48.0)	
Inferior wall	299 (45.5)	220 (45.3)	79 (46.2)	
Lateral wall	34 (5.2)	24 (4.9)	10 (5.8)	
Medication at discharge				
Aspirin, n (%)	720 (99.9)	540 (99.8)	180 (100.0)	1.000
Clopidogrel, n (%)	665 (93.9)	500 (94.0)	165 (93.8)	0.910
Statins, n (%)	717 (100.0)	542 (100.0)	175 (100.0)	1.000
ACEI/ARB, n (%)	622 (98.9)	472 (99.6)	150 (96.8)	0.012
SGLT2i, n (%)	14 (21.2)	12 (26.7)	2 (9.5)	0.195
MRA, n (%)	60 (55.6)	44 (57.1)	16 (51.6)	0.601
β-blockers, n (%)	553 (96.3)	416 (96.7)	137 (95.1)	0.374
Metabolic indices				
TyG index	9.0 (8.5, 9.4)	9.0 (8.5, 9.4)	9.1 (8.6, 9.5)	0.175

Table 1. Continued.

Variables	Total (n = 1180)	Without remodeling (n = 911)	With remodeling (n = 269)	*p* value
TyG–BMI index	228.8 (206.9, 253.9)	228.0 (206.0, 253.0)	233.2 (208.9, 259.3)	0.177
METS-IR	39.1 (34.8, 43.9)	39.1 (34.7, 43.7)	39.2 (35.2, 44.1)	0.464

Abbreviations: HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; ApoA-I, apolipoprotein A-I; ApoB, apolipoprotein B; HbA1c, glycated hemoglobin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; hs-CRP, high-sensitivity C-reactive protein; CK-MB, creatine kinase-MB; LAD, left anterior descending artery; RCA, right coronary artery; LCX, left circumflex artery; ACEI, angiotensin-converting enzyme inhibitor; ARB, angiotensin II receptor blocker; SGLT2i, sodium-glucose cotransporter 2 inhibitor; MRA, mineralocorticoid receptor antagonist; TyG, triglyceride-glucose index; TyG-BMI, triglyceride-glucose body mass index; METS-IR, metabolic score for insulin resistance.

Note: Percentages were calculated using the number of participants with available data for each corresponding variable as the denominator. Available sample sizes varied across variables because of missing baseline information and incomplete prescription records. Medication use was calculated among patients with available prescription records; therefore, denominators may differ across medications and may be smaller than the total cohort. Continuous variables are presented as mean ± SD or median (IQR), and categorical variables as number (percentage). *p*-values were derived from Student's *t*-test, Mann–Whitney U test, or χ^2 test, as appropriate.

Table 2. Logistic regression analysis of the association between TyG-related indices and left ventricular remodeling after AMI.

Variable	Total (n)	Events, n (%)	Crude OR (95% CI)	*p* value	Adjusted OR (95% CI)	*p* value
TyG	1180	269 (22.8)	1.18 (0.98–1.42)	0.086	1.67 (1.24–2.26)	0.001
TyG–BMI	1180	269 (22.8)	1.00 (1.00–1.01)	0.115	1.01 (1.00–1.01)	0.008
METS-IR	1180	269 (22.8)	1.01 (0.99–1.03)	0.492	1.04 (1.00–1.07)	0.048

Adjusted for age, gender, hypertension, diabetes, SBP, DBP, alcohol consumption, uric acid, HDL-C, and infarct-related artery.

Abbreviations: AMI, acute myocardial infarction; TyG, triglyceride-glucose index; TyG-BMI, triglyceride-glucose body mass index; METS-IR, metabolic score for insulin resistance; OR, odds ratio; CI, confidence interval; SBP, systolic blood pressure; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol.

mentary Table 3). In sensitivity analyses, the associations between TyG-related indices and left ventricular remodeling remained consistent with the primary analysis (**Supplementary Table 4**). ROC curve analyses showed that TyG, TyG–BMI, and METS-IR had similar, albeit modest, discriminatory ability for left ventricular remodeling, with AUCs of 0.655 (95% CI, 0.593–0.716), 0.660 (95% CI, 0.599–0.720), and 0.652 (95% CI, 0.592–0.713), respectively (**Supplementary Table 7**).

3.3 Dose–Response Relationship Between TyG-Related Indices and Left Ventricular Remodeling After AMI

RCS analysis was conducted to evaluate the dose–response relationships between TyG-related indices and the risk of LV remodeling following an AMI. TyG and TyG–BMI exhibited approximately linear associations with LV remodeling (*p* for nonlinearity = 0.629 and 0.222, respectively). For METS-IR, although there was no evidence of nonlinearity (*p* for nonlinearity = 0.429), the overall association with LV remodeling was not statistically significant (*p* for overall association = 0.135) (Fig. 1).

3.4 Subgroup Analyses of the Associations Between TyG-Related Indices and Left Ventricular Remodeling

Subgroup analyses (Fig. 2) showed that all three insulin resistance-related metabolic indices (TyG, TyG–BMI, and METS-IR) were consistently and positively associated with LV remodeling in most clinical subgroups. No significant interactions were observed for sex, age, smoking, alcohol consumption, diabetes, hypertension, or other baseline characteristics (all *p* for interactions> 0.05).

4. Discussion

LV remodeling following an AMI remains a major determinant of adverse prognosis despite advances in early reperfusion therapy [3,14]. Although infarct size, inflammation, and neurohormonal activation are well-established contributors, emerging evidence indicates that IR and metabolic dysregulation play a pivotal role in maladaptive cardiac remodeling [4]. In this context, non–insulin-based metabolic indices such as the TyG index, TyG–BMI, and the METS-IR have attracted growing attention as simple yet powerful markers of systemic metabolic stress [15,16,17].

In our cohort of 1180 AMI patients treated with PCI, 269 (22.8%) developed LV remodeling at 6-month follow-

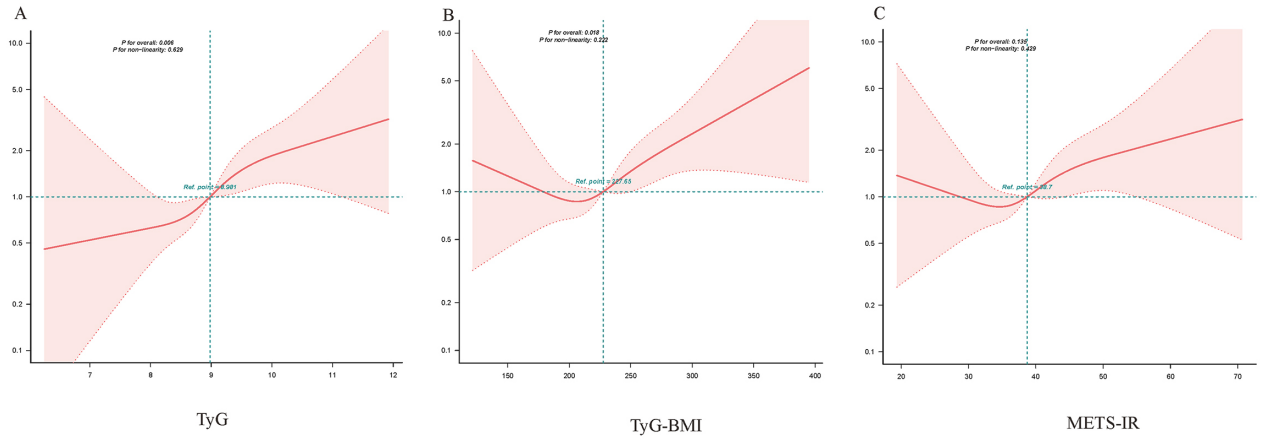


Fig. 1. Restricted cubic spline (RCS) curves showing the dose–response relationship between TyG-related indices and the risk of left ventricular remodeling after AMI. Models were adjusted for age, gender, hypertension, diabetes, SBP, DBP, alcohol consumption, uric acid, HDL-C, and infarct-related artery. (A) TyG index (B) TyG–BMI (C) METS-IR.

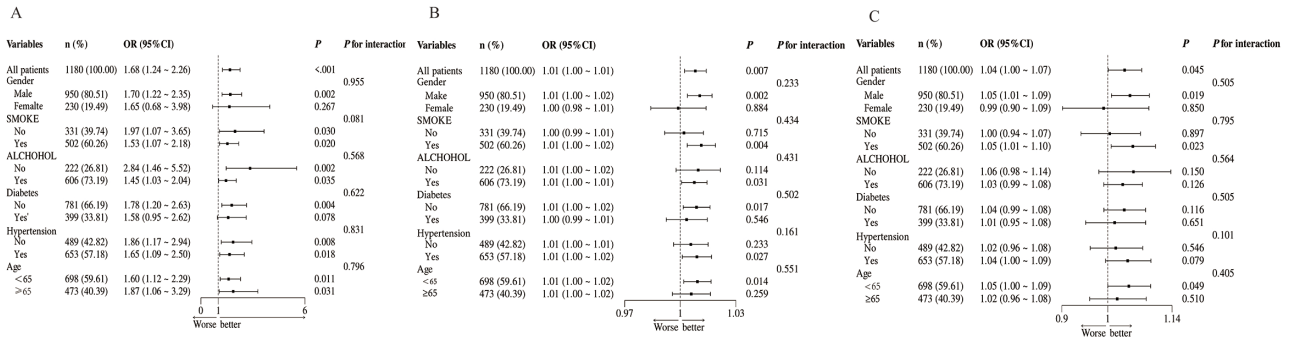


Fig. 2. Subgroup analyses showing the associations between (A) TyG, (B) TyG–BMI, and (C) METS-IR with the risk of left ventricular remodeling after acute myocardial infarction, stratified by baseline clinical characteristics.

up. Higher TyG, TyG–BMI, and METS-IR levels were independently associated with a higher risk of LV remodeling, consistent with previous evidence linking IR-related indices to adverse cardiovascular outcomes [18]. These associations were linear and significant across all clinical subgroups, suggesting that systemic insulin resistance contributes to post-infarction myocardial adaptation independent of traditional cardiovascular risk factors. The absence of statistically significant between-group differences in TyG-related indices in the unadjusted baseline comparison does not preclude their independent association with LV remodeling, since crude comparisons do not account for potential confounding factors. After multivariable adjustment, TyG, TyG–BMI, and METS-IR remained significantly associated with LV remodeling, indicating that these indices may capture metabolic risk not fully reflected in unadjusted analyses. In addition, sensitivity analyses using BSA-indexed LVEDV yielded consistent results, further supporting the significance of the observed associations and indicating that these findings were not materially influenced by differences in body size. Furthermore, after additional adjustment for the use of ACEI/ARB, the associations of TyG and TyG–BMI with left ventricular remodeling remained significant, whereas the association for METS-IR was attenuated.

4.1 Insulin Resistance as a Driver of Post-Infarction Remodeling

Mechanistically, IR impairs myocardial energy metabolism by reducing glucose uptake and enhancing fatty acid oxidation, leading to mitochondrial dysfunction, lipotoxicity, and accumulation of reactive oxygen species (ROS) [19,20,21]. These alterations promote cardiomyocyte injury and extracellular matrix remodeling, contributing to ventricular dilation—hallmarks of adverse remodeling. In addition, IR contributes to endothelial dysfunction and microvascular inflammation [22], which may impair perfusion of the peri-infarct zone and promote myocardial fibrosis [5,23,24,25,26].

Our study found that patients in the LV remodeling group exhibited significantly higher levels of liver enzymes (ALT and AST) and neutrophil counts compared

with those without remodeling, suggesting enhanced inflammatory and metabolic stress in these patients. Elevated ALT and AST levels may indicate greater myocardial injury as well as hepatic metabolic overload, both of which are exacerbated by insulin resistance. The higher neutrophil count observed in the remodeling group underscores the contribution of systemic inflammation—a well-recognized consequence of IR—to adverse ventricular structural changes. These findings highlight the close interplay between metabolic stress and inflammation in the development of post-infarction LV remodeling.

4.2 Evidence From Clinical Studies

Recent cohort studies and meta-analyses have validated TyG-related indices as reliable surrogate markers of IR, which are strongly associated with cardiovascular morbidity and mortality [17,18,27]. A meta-analysis by Wang et al. [18] reported that an elevated TyG index was associated with higher risks of major adverse cardiovascular events (MACE) and all-cause mortality in patients undergoing PCI. Li et al. [17] reported that TyG–BMI independently predicted the incidence of cardiovascular disease in middle-aged and elderly adults. Our study extends these findings by linking TyG, TyG–BMI, and METS-IR directly to structural LV remodeling assessed by echocardiography, reflecting earlier stages of post-AMI cardiac adaptation before overt heart failure.

4.3 Biological Plausibility

Hyperinsulinemia enhances sympathetic drive and activates the renin–angiotensin–aldosterone system (RAAS), increasing afterload and promoting myocardial hypertrophy [28]. Chronic inflammation further promotes extracellular matrix turnover and fibrosis [29,30]. These pathways provide a biologically plausible explanation for the association between insulin resistance and adverse LV remodeling. Taken together, these indices may be best interpreted as simple adjunctive metabolic markers that can complement conventional clinical assessment rather than as standalone predictors of post-AMI remodeling.

4.4 Clinical Implications

The clinical implications of these findings are noteworthy. Unlike HOMA-IR, which requires insulin assays, TyG, TyG–BMI, and METS-IR are derived from routine fasting laboratory parameters, making them practical for large-scale screening. Integrating these indices into post-AMI risk stratification frameworks may provide complementary information for identifying patients at risk for adverse remodeling. However, given the relatively modest effect sizes observed for some indices, particularly TyG–BMI and METS-IR, these markers should be viewed as adjunctive rather than standalone tools. Given the modest discriminatory performance of these indices, TyG, TyG–BMI, and METS-IR may be more appropriately interpreted as supplementary metabolic markers rather than stand-alone predictive tools, and may provide additional information when combined with established clinical risk factors.

Recent randomized trials have demonstrated that improving insulin sensitivity—via sodium-glucose cotransporter-2 inhibitors (SGLT2i), glucagon-like peptide-1 (GLP-1) receptor agonists, or structured exercise programs—attenuates LV remodeling and lowers the incidence of heart failure [31,32]. However, only 21.2% of our cohort received SGLT2i, suggesting a significant opportunity for therapeutic optimization. In addition, we observed that the use of ACEI/ARB was significantly lower in the remodeling group (96.8% vs 99.6%, $p = 0.012$), despite guideline recommendations for its universal use in post-AMI patients. These observations highlight the need for integrated management strategies that combine optimal pharmacologic therapy with metabolic risk control to mitigate adverse LV remodeling.

4.5 Strengths and Limitations

This study has several strengths. It enrolled a relatively large cohort of AMI patients, all treated with PCI, ensuring a strong clinically representative group of patients. Detailed metabolic and clinical data were systematically collected, and standardized echocardiographic assessments were performed both at baseline and 6-month follow-up to ensure consistency in evaluating LV remodeling. The consistent associations across three independent IR-related indices reinforce the significance and generalizability of our conclusions.

Nonetheless, several limitations warrant mention. As a single-center retrospective study, external validation in larger and more diverse populations is required. Despite significant statistical associations, causality cannot be inferred, and residual confounding by unmeasured variables cannot be excluded. Although no substantial multicollinearity was detected based on GVIF assessment, potential model dependence should still be considered when interpreting the multivariable results. Some covariates included in the adjusted models may be biologically related; therefore, the observed associations may partly depend on model specification, and residual model-dependent uncertainty cannot be completely excluded. Formal intra-observer and inter-observer reproducibility analyses were not performed in the present study, which may have introduced measurement variability. Specifically, some clinically relevant variables, including the precise time from symptom onset to reperfusion, the intensity of guideline-directed medical therapy, and complete medication records for certain therapies—particularly some antidiabetic medications—were not consistently available in the retrospective database, which may also contribute to residual confounding. In addition, mechanistic biomarkers, such as inflammatory cytokines, oxidative stress markers, or metabolomic signatures, were not measured, limiting

insight into the underlying biological pathways. In conclusion, increasing evidence, including our findings, supports the concept that insulin resistance-related metabolic indices are independently associated with LV remodeling after an AMI. These indices may reflect systemic metabolic and inflammatory stress and have potential clinical utility in patients following an AMI.

5. Conclusion

These findings indicate a positive association between TyG-related metabolic indices and left ventricular remodeling after an AMI, suggesting that higher insulin resistance may be associated with an increased risk of adverse structural cardiac changes.

Abbreviations

AMI, Acute myocardial infarction; AST, Aspartate aminotransferase; ALT, Alanine aminotransferase; ACEI, Angiotensin-converting enzyme inhibitor; ARB, Angiotensin II receptor blocker; BMI, Body mass index; CI, Confidence interval; DBP, Diastolic blood pressure; FPG, Fasting plasma glucose; HDL-C, High-density lipoprotein cholesterol; IR, Insulin resistance; IVST, Interventricular septal thickness; LDL-C, Low-density lipoprotein cholesterol; LAD, Left anterior descending artery; LCX, Left circumflex artery; LV, Left ventricle/left ventricular; LVEDD, Left ventricular end-diastolic diameter; LVEDV, Left ventricular end-diastolic volume; LVESD, Left ventricular end-systolic diameter; LVEF, Left ventricular ejection fraction; MACE, Major adverse cardiovascular events; METS-IR, Metabolic score for insulin resistance; MRA, Mineralocorticoid receptor antagonist; NSTEMI, Non–ST-segment elevation myocardial infarction; OR, Odds ratio; PCI, Percutaneous coronary intervention; RCA, Right coronary artery; RCS, Restricted cubic spline; ROS, Reactive oxygen species; SBP, Systolic blood pressure; SD, Standard deviation; SGLT2i, Sodium–glucose cotransporter 2 inhibitor; STEMI, ST-segment elevation myocardial infarction; TC, Total cholesterol; TG, Triglycerides; TyG, Triglyceride-glucose index.

Availability of Data and Materials

The data used in this study were obtained from an institutional research database. De-identified data may be available from the corresponding author upon reasonable request and with appropriate institutional approval.

Author Contributions

YYZ contributed to the study design, data collection, data analysis, and drafting of the manuscript. ZL contributed to the conception and design of the study, supervision, and critical revision of the manuscript. YLZ, QCY, SW, SX, HDJ, YLW, ZXF, and FYL contributed to data collection, interpretation of the results, and revision of the manuscript. All authors read and approved the final manuscript. All authors have participated sufficiently in the work and agreed to be accountable for all aspects of the work.

Ethics Approval and Consent to Participate

The study was carried out in accordance with the guidelines of the Declaration of Helsinki and approved by the Ethics Committee of Xuanwu Hospital, Capital Medical University (Approval No. 2022161). All patients provided routine informed consent for their clinical treatment. As this was a retrospective study using fully anonymized clinical records, the requirement for written informed consent for research participation was waived in accordance with institutional guidelines.

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Conflicts of Interest

The authors declare no conflicts of interest.

Supplementary Material

Supplementary material associated with this article can be found, in the online version, at https://doi.org/10.31083/RCM50053.

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