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ORIGINAL RESEARCH

The Association of C-Reactive Protein-Triglyceride Glucose Index with Major Adverse Cardiovascular Events in Patients with Acute Myocardial Infarction

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Background: The C-reactive protein-triglyceride glucose index (CTI), a novel marker that integrates metabolic dysfunction and inflammation, has an uncertain prognostic value for predicting major adverse cardiovascular events (MACEs) after acute myocardial infarction (AMI).

Methods: This retrospective study enrolled 578 patients with AMI admitted to Xuanwu Hospital from 2019 to 2022. All patients were categorized according to tertiles of CTI values. Receiver operating characteristic (ROC) curves analysis and restricted cubic spline (RCS) were applied to explore the relationships of CTI, triglyceride-glucose (TyG) index, and high-sensitivity C-reactive protein (hs-CRP) with MACEs. Furthermore, the association between CTI and MACEs was evaluated using Kaplan-Meier curves and Cox proportional hazards regression models, with subgroup analyses assessing this relationship across various clinical conditions.

Results: During a follow-up period with a median time of 34 months, 69 patients experienced MACEs, and higher CTI tertiles were associated with a greater cumulative risk of MACEs (Log rank test, $p = 0.003$). RCS analysis showed a positive linear association between CTI and MACEs risk (p-overall = 0.017). Compared with the TyG index (AUC = 0.562, 95% CI: 0.488–0.636) and hs-CRP (AUC = 0.596, 95% CI: 0.530–0.663), CTI demonstrated the highest AUC (0.612, 95% CI: 0.541–0.682). Importantly, the addition of CTI to the baseline model provided significant incremental prognostic value, with significant improvements in both net reclassification improvement (NRI) and integrated discrimination improvement (IDI), whereas neither the TyG index nor hs-CRP yielded significant improvements. Multivariate Cox regression analysis confirmed that the highest CTI tertile had a 3.09-fold increased risk of MACEs compared to the lowest tertile ($p < 0.001$) in the fully adjusted model, and the association remained stable across subgroups.

Conclusion: The CTI is a more promising indicator for predicting MACEs in AMI patients than the TyG index or hs-CRP alone. By integrating the core risk factors of inflammation and insulin resistance, CTI may provide additional value for post-discharge risk stratification, helping to identify high-risk patients who may benefit from precise management.

Keywords: acute myocardial infarction, C-reactive protein-triglyceride glucose index, high-sensitivity C-reactive protein, triglyceride glucose index

Introduction

Acute myocardial infarction (AMI), widely recognized as the most severe form of ischemic heart disease, remains one of the leading causes of global mortality and is a major contributor to disability.[1] Although advances in reperfusion therapies, together with improvements in regional coordination of care, have substantially decreased acute-phase mortality in patients with AMI, the burden of extended adverse clinical events remains considerable over time.[2,3] Therefore, identifying and managing modifiable risk factors holds substantial importance in the long-term prognosis of AMI patients.

Among the complex pathophysiological mechanisms of AMI, insulin resistance (IR) is recognized as one of the core metabolic disorders driving atherosclerotic progression.[4,5] Multiple investigations have demonstrated a significant association between IR and increased incidence of several cardiovascular events.[6–9] The triglyceride-glucose (TyG) index, composed of triglyceride (TG) and fasting blood glucose (FBG), is a well-validated indicator of IR and shows a strong association with

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adverse cardiovascular outcomes.[10–13] It is an easily obtainable and inexpensive tool that is anticipated to offer useful information for the clinical management of cardiovascular disease (CVD).

In addition to IR, recent findings have increasingly suggested that inflammation is a crucial factor driving recurrent cardiovascular events and poor outcomes after AMI.[14–16] High-sensitivity C-reactive protein (hs-CRP) concentration is among the most recognized and widely utilized biomarkers available for detecting low-grade systemic inflammation.[17] Elevated hs-CRP levels have been identified across multiple studies as a strong predictor of recurrent cardiovascular events and mortality following AMI.[18,19] Based on the concept of integrated systemic risk and the recognition that inflammation and IR jointly promote atherosclerotic progression, researchers have increasingly explored the value of assessing inflammatory markers together with the TyG index. The C-reactive protein–triglyceride glucose index (CTI) was first described in 2022 as a potential biomarker capable of predicting survival in patients with malignancies.[20] The index was created to provide a combined assessment of systemic inflammatory activity and IR severity. Previous studies have suggested that CTI may have predictive value for the incidence of CVD.[21,22] However, CTI is a relatively novel index, and its external validation across different cardiovascular populations remains limited. Moreover, AMI is a multifactorial disease, and residual cardiovascular risk cannot be fully explained by any single pathway alone. Recent evidence suggests that plaque characteristics and coronary physiology may also provide complementary prognostic information.[23] These findings further support the concept of integrated risk assessment after AMI and underscore the need to explore composite indices that can more comprehensively capture risk.

Our study first compared CTI and its two constituent indices in their ability to predict adverse cardiovascular events in patients with AMI. In addition, we conducted further analyses to verify the robustness and consistency of the observed relationship between CTI and the occurrence of major adverse cardiovascular events (MACEs).

Materials and Methods

Study Design and Participants

For this study, we enrolled 578 patients with AMI who presented at Xuanwu Hospital of Capital Medical University from 2019 to 2022. We analyzed the previous day's indicators for patients who were in stable condition and prepared for discharge. The diagnosis of AMI was determined according to the consensus document outlining the contemporary definitions of myocardial infarction.[24] The exclusion criteria were as follows: (1) missing data related to indicators, including hs-CRP, TG, and FBG; (2) severe conditions, including severe hepatic (alanine aminotransferase (ALT) or aspartate aminotransferase (AST) > 5.0 × the upper limit of normal) or renal (Creatinine > 3.0 × the upper limit of normal) dysfunction, critical structural heart disease requiring intervention, infectious diseases, bleeding predisposition, and cancer; and (3) previous coronary artery bypass grafting (CABG). Participants were categorized into one of three equal groups depending on their CTI levels: T1: CTI ≤ 9.06; T2: 9.06 < CTI ≤ 9.79; and T3: CTI > 9.79 (Figure 1). The studies involving humans were approved by Xuanwu Hospital Ethics Committee under document No. 2022-129. This investigation was conducted in full compliance with the ethical standards set forth in the Declaration of Helsinki. This study was reported in accordance with the RECORD (REporting of studies Conducted using Observational Routinely-collected Data) guidelines to promote transparency and completeness in the reporting of observational research.

Data Collection and Definitions

Information on patient demographics, past medical conditions, laboratory indices, echocardiographic parameters, and coronary angiographic findings was obtained from the digital patient record system by qualified physicians. All participants provided fasting blood samples in the morning after an overnight fast, and the samples were analyzed on the same day in a central laboratory. The TyG index was quantified via the following formula: TyG index = ln [TG (mg/dL) × FBG (mg/dL)/2]. Previous cohort studies have established hs-CRP level as a key prognostic indicator in patients with atherosclerotic cardiovascular disease. Therefore, our study employed hs-CRP values to compute the CTI. The CTI was calculated as follows: CTI = TyG index + 0.412 × ln [hs-CRP (mg/L)].

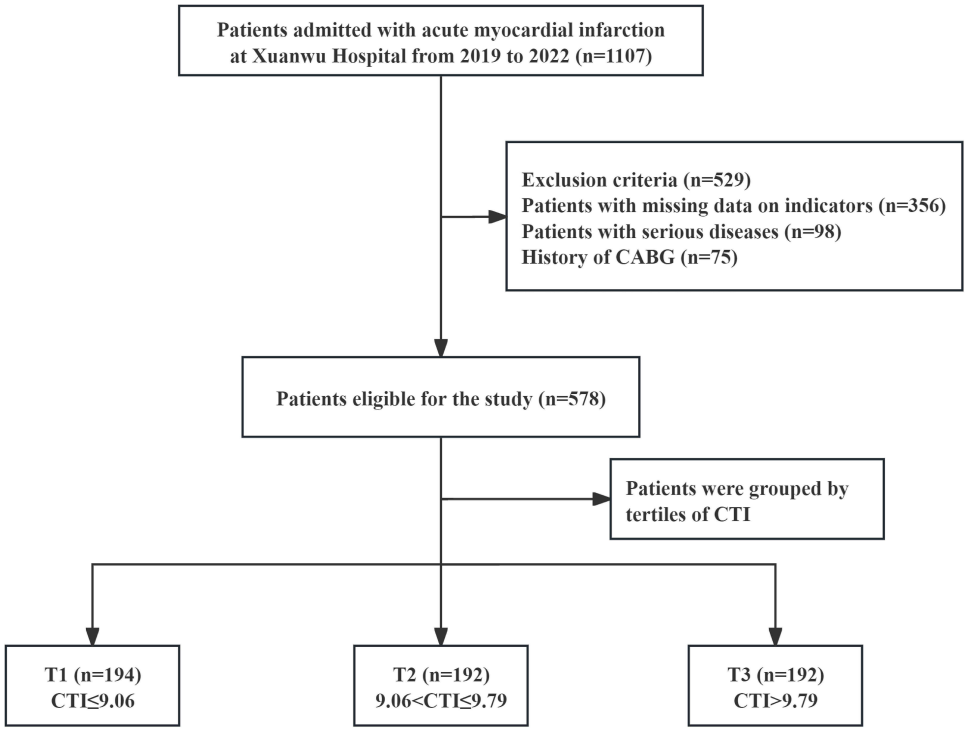

Figure 1 Flowchart of Patient Enrollment.
Abbreviations: CTI, C-reactive protein-triglyceride glucose index; CABG, coronary artery bypass grafting.

Follow-Up and Endpoint Events

Follow-up was conducted by telephone calls, text messages, and clinical visits. To standardize follow-up procedures and ensure data accuracy, all coordinators received professional training. The primary endpoint assessed in this investigation was the occurrence of composite MACEs, defined as all-cause death, nonfatal myocardial infarction, nonfatal stroke, or unplanned revascularization. Unplanned revascularization refers to a procedure performed due to worsening myocardial ischemia caused by in-stent restenosis, native coronary artery lesion progression, or new territory disease. If a patient experienced more than one endpoint event, only the first occurring event was used for the analysis.

Statistical Analysis

Continuous data based on distribution characteristics were reported as means ± standard deviations or medians (interquartile ranges) and compared using ANOVA or the Kruskal–Wallis test. We utilized restricted cubic splines (RCS) to analyze associations between CTI, TyG index, and hs-CRP with MACEs. Receiver operating characteristic (ROC) curves were used to assess discriminatory ability, and model improvement was evaluated using the net reclassification improvement (NRI) and integrated discrimination improvement (IDI). We plotted Kaplan–Meier survival curves to illustrate the cumulative incidence of MACEs in groups based on CTI tertiles and applied the Log rank test to compare the survival differences between these groups. Given the limited number of MACEs, we adopted a parsimonious modeling strategy for the primary multivariable Cox regression, with covariates pre-specified to include demographic variables and traditional cardiovascular risk factors. To quantify the association between the CTI and study outcomes, three sequential Cox proportional hazards models were constructed: an unadjusted model; a model adjusted for age, gender, and body mass index (BMI); and a fully adjusted model additionally including hypertension, low-density lipoprotein cholesterol (LDL-C) and high-density lipoprotein cholesterol (HDL-C). To address potential confounding from AMI- specific prognostic variables, a sensitivity analysis was performed using an extended Cox model that further adjusted for diabetes, hyperlipidemia, smoking status, GRACE score, left ventricular ejection fraction (LVEF), and percutaneous coronary intervention (PCI). Multicollinearity was assessed using variance inflation factors (VIF), with no substantial multicollinearity observed in the multivariable Cox models

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(Supplementary Table S1). Subgroup analyses stratified by demographic and clinical factors were conducted to test for interactions using likelihood ratio tests. Statistical analyses were performed using R Programming Language (version 4.5.1). All statistical tests were two-sided, and the significance level was set at 0.05.

Results

Characteristics of the Study Population

All patients were categorized according to CTI tertiles (T1: CTI ≤ 9.06; T2: 9.06 < CTI ≤ 9.79; and T3: CTI > 9.79), and the baseline characteristics of the three groups are detailed in Table 1. A total of 578 patients were ultimately assessed in the study cohort, with the median age being 60 years. Among these individuals, 470 (81.31%) were male and 108 (18.69%) were female. 69 patients experienced MACEs during a follow-up period with a median time of 34 months. Patients in the higher CTI tertiles were more likely to have diabetes, hyperlipidemia and previous stroke ($p < 0.05$). No significant differences among the groups were observed in age, gender, hypertension, previous MI or revascularization, or GRACE score ($p > 0.05$). Patients with higher CTI levels had higher laboratory indices, including blood glucose, total cholesterol (TC), TG, serum uric acid, hs-CRP, glycated hemoglobin A1c (HbA1c), interleukin-6 (IL-6), and N-terminal pro-B-type natriuretic peptide (NT-proBNP) levels. The incidence of in-hospital ventricular tachyarrhythmias, atrial fibrillation, and cardiac arrest was low across all groups, with no statistically significant differences observed ($p > 0.05$). Additionally, the T3 tertile group showed the highest incidence of three-vessel disease (42.71% vs 31.25% vs. 27.32%; $p < 0.001$) and MACEs (17.71% vs. 10.94% vs. 7.22%; $p = 0.006$) when compared with the other two groups.

RCS Analysis

We employed RCS to visualize the associations of CTI, TyG index, and hs-CRP level with MACEs. The analysis demonstrated that both CTI (p-overall = 0.017) and hs-CRP level (p-overall = 0.020) were significantly associated with the risk of MACEs. The tests for nonlinearity were not significant for either variable (CTI: p-nonlinear = 0.733; hs-CRP level: p-nonlinear = 0.167), suggesting linear positive associations with risk. The RCS curves demonstrated that the hazard ratio (HR) continuously increased with increasing CTI (Figure 2A) and hs-CRP level (Figure 2C). In contrast, the analyses for the TyG index (p-overall = 0.394, p-nonlinear = 0.883) indicated a lack of statistical evidence for an association with MACEs (Figure 2B).

Comparative Predictive Ability of CTI, TyG Index and Hs-CRP Level for MACEs

We first assessed the individual discriminative ability of these indices for MACEs using ROC analysis (Figure 3). The CTI demonstrated the highest predictive value, with an AUC of 0.612 (95% CI: 0.541–0.682), compared with hs-CRP (AUC = 0.596; 95% CI: 0.530–0.663) and TyG (AUC = 0.562; 95% CI: 0.488–0.636). Based on the Youden index, the optimal CTI cut-off value was 9.46, with a sensitivity of 71.0% and a specificity of 52.7% (Supplementary Table S2). This value was close to the boundary between the second and third tertiles of CTI in our analysis (9.79). We then evaluated the incremental predictive value of each index when added to a baseline model, which was adjusted for conventional risk factors (age, gender, BMI, hypertension status, HDL-C and LDL-C). The performance of the models is detailed in Table 2. While adding CTI did not significantly change the AUC ($p = 0.370$), it resulted in significant improvements in reclassification and discrimination metrics, including continuous NRI (0.421; 95% CI: 0.177–0.665; $p < 0.001$), categorical NRI (0.131; 95% CI: 0.012–0.249; $p = 0.031$; Supplementary Table S4), and IDI (0.021; 95% CI: 0.005–0.038; $p = 0.011$). In contrast, the addition of the TyG index or hs-CRP did not significantly improve any of the evaluated predictive metrics.

Prognostic Value of the CTI for MACEs

On the basis of the preceding analysis, we found that the CTI demonstrated relatively greater predictive value for MACEs. Therefore, we further analyzed the relationship between CTI and MACEs. Kaplan–Meier analysis demonstrated a significant relationship between CTI tertiles and MACE incidence (log-rank, $p = 0.003$). As CTI tertile increased, the

Table 1 Baseline Characteristics of AMI Participants Divided by Tertiles of the CTI Value

Baseline Characteristics	Total (n = 578)	T1 (n = 194)	T2 (n =192)	T3 (n =192)	*p* Value
Age (years)	60.00 (52.00,66.00)	61.00(56.00,65.00)	60.00(50.00,66.00)	59.00(51.00,66.00)	0.362
Male, n (%)	470(81.31)	163(84.02)	161(83.85)	146(76.04)	0.072
BMI, kg/m^2	25.44(23.66,27.72)	25.14(23.00,27.34)	25.71(24.22,27.77)	25.25(23.53,28.23)	0.008
Medical History					
Current smoker, n (%)	371(64.19)	115(59.28)	137(71.35)	119(61.98)	0.035
Hypertension, n (%)	275(47.58)	89(45.88)	92(47.92)	94(48.96)	0.827
Diabetes, n (%)	87(15.05)	13(6.70)	20(10.42)	54(28.12)	<0.001
Hyperlipidemia, n (%)	86(14.88)	19(9.79)	31(16.15)	36(18.75)	0.039
Previous Stroke, n (%)	51(8.82)	6(3.09)	21(10.94)	24(12.50)	0.002
Previous MI, n (%)	46(7.96)	20(10.31)	9(4.69)	17(8.85)	0.107
Previous revascularization, n (%)	46(7.96)	19(9.79)	14(7.29)	13(6.77)	0.502
Types of AMI					
STEMI, n (%)	365(63.15)	128(65.98)	127(66.15)	110(57.29)	0.120
NSTEMI, n (%)	213(36.85)	66(34.02)	65(33.85)	82(42.71)	
Acute Myocardial Infarction Score					
GRACE Score	133.24± 27.80	131.10 ± 27.06	135.67 ± 29.44	133.12 ± 26.77	0.304
Laboratory test					
Hemoglobin, g/L	141.00(130.00,150.00)	141.00(130.00,149.75)	141.50(130.00,149.00)	141.00(130.00,152.25)	0.975
Platelet count, $10^9/L$	219.00(186.75,263.25)	212.00(181.00,242.75)	224.00(187.00,269.75)	223.00(194.50,277.25)	<0.001
Blood glucose, mg/dL	92.88(84.60,105.66)	87.57(81.54,96.30)	91.62(84.06,99.94)	102.78(91.08,136.84)	<0.001
ALT, U/L	33.00(20.00,52.00)	31.00(20.00,48.00)	35.50(20.00,57.00)	36.00(21.00,56.00)	0.310
AST, U/L	48.00(29.00,125.00)	45.50(29.00,130.00)	47.00(29.00,122.75)	51.00(27.00,102.00)	0.929
Creatinine, umol/L	70.50(62.00,81.00)	67.00(60.25,77.50)	74.00(64.00,84.00)	71.00(62.75,83.00)	0.002
TC, mmol/L	4.31(3.64,4.93)	4.18(3.51,4.67)	4.35(3.63,4.94)	4.50(3.80,5.15)	<0.001
LDL-C, mmol/L	2.69(2.19,3.18)	2.62(2.15,2.97)	2.79(2.26,3.26)	2.74(2.18,3.23)	0.016
HDL-C, mmol/L	0.99(0.84,1.19)	1.07(0.93,1.27)	0.95(0.80,1.18)	0.94(0.79,1.13)	<0.001
TG, mg/dL	127.94(91.20,185.93)	92.52(72.60,123.96)	138.12(95.40,175.53)	187.70(125.51,268.94)	<0.001
Serum uric Acid, umol/L	353.00(295.00,418.00)	327.50(284.25,401.00)	362.00(301.75,418.25)	366.00(311.00,431.50)	0.006
hs-CRP, mg/L	5.11(1.85,14.31)	1.52(0.69,2.54)	5.72(2.84,9.59)	18.09(8.06,33.30)	<0.001
HbA1C (%)	5.70(5.40,6.00)	5.60(5.30,5.80)	5.70(5.40,6.00)	5.80(5.50,6.40)	<0.001
IL-6, pg/mL	16.53(9.83,31.32)	12.14(7.53,19.82)	18.25(10.88,30.12)	23.84(12.02,52.07)	<0.001
NT-ProBNP, pg/mL	279.00(90.10,752.00)	210.00(75.85,480.00)	279.00(89.65,744.00)	531.00(148.00,1149.50)	<0.001
Peak hs-cTnI, ng/mL	7.46(1.14,27.00)	10.50(0.74,33.10)	5.05(0.71,20.20)	6.79(1.65,27.10)	0.079
Echocardiogram					
LVEF (%)	58.00(52.00,64.00)	59.00(51.00,65.00)	59.00(53.25,65.00)	58.00(51.00,62.00)	0.098
Left atrial diameter, mm	37.00(34.00,40.00)	36.00(32.00,40.00)	38.00(35.00,40.00)	38.00(35.00,41.00)	<0.001
LVEDD, mm	51.00(48.00,54.00)	51.00(48.00,53.00)	51.0 (48.00,55.00)	51.00(48.00,54.00)	0.621
In-hospital complications					
Ventricular tachyarrhythmias, n (%)	20(3.46)	6(3.09)	10(5.21)	4(2.08)	0.232
Atrial fibrillation, n (%)	17(2.94)	6(3.09)	6(3.13)	5(2.60)	0.994
Cardiac arrest, n (%)	6(1.04)	0(0.00)	2(1.04)	4(2.08)	0.090
Number of diseased vessels, n (%)					
1-vessel disease	241(41.70)	83(42.78)	88(45.83)	70(36.46)	0.164
2-vessel disease	142(24.57)	58(29.90)	44(22.92)	40(20.83)	0.095
3-vessel disease	195(33.74)	53(27.32)	60(31.25)	82(42.71)	0.004
PCI, n (%)	481(83.22)	152(78.35)	169(88.02)	160(83.33)	0.039
MACEs, n (%)	69(11.94)	14(7.22)	21(10.94)	34(17.71)	0.006

Notes: Data are median (interquartile range), or n (%).

Abbreviations: AMI, acute myocardial infarction; ALT, alanine aminotransferase; AST, aspartate aminotransferase; BMI, body mass index; GRACE, Global Registry of Acute Coronary Events; HDL-C, high-density lipoprotein cholesterol; hs-CRP, high-sensitivity C-reactive protein; HbA1c, glycated hemoglobin A1c; IL-6, interleukin-6; LDL-C, low-density lipoprotein cholesterol; LVEF, left ventricular ejection fraction; LVEDD, left ventricular end-diastolic diameter; MI, myocardial infarction; MACEs, major adverse cardiovascular events; NT-proBNP, N-terminal pro-B-type natriuretic peptide; NSTEMI, non-ST-segment elevation myocardial infarction; PCI, percutaneous coronary intervention; STEMI, ST-segment elevation myocardial infarction; TC, total cholesterol; TG, triglycerides; hs-cTnI, high-sensitivity cardiac troponin I.

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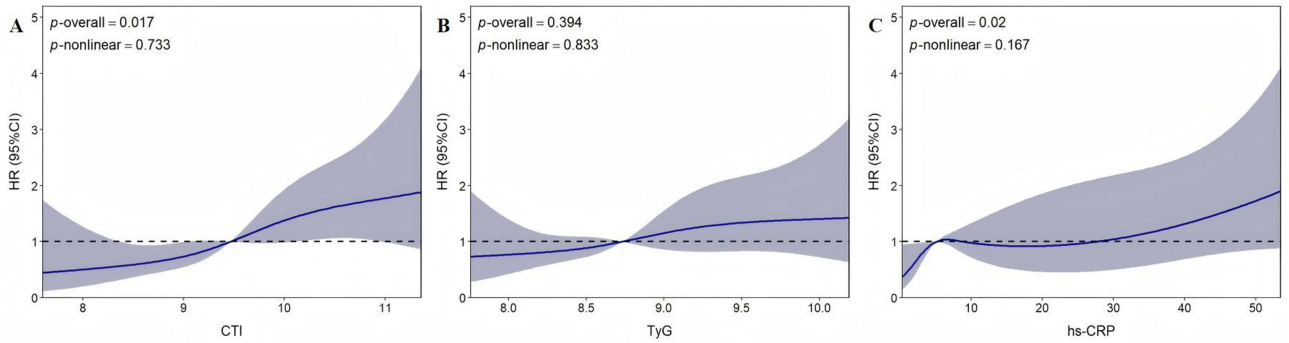

Figure 2 Restricted cubic spline curves for the associations between the CTI (**A**), TyG index (**B**) and hs-CRP (**C**) with the incidence of MACEs among patients with AMI.
Abbreviations: AMI, acute myocardial infarction; CI, confidence interval; CTI, C-reactive protein-triglyceride glucose index; HR, hazard ratio; hs-CRP, high-sensitivity C-reactive protein; MACEs, major adverse cardiovascular events; TyG, triglyceride-glucose.

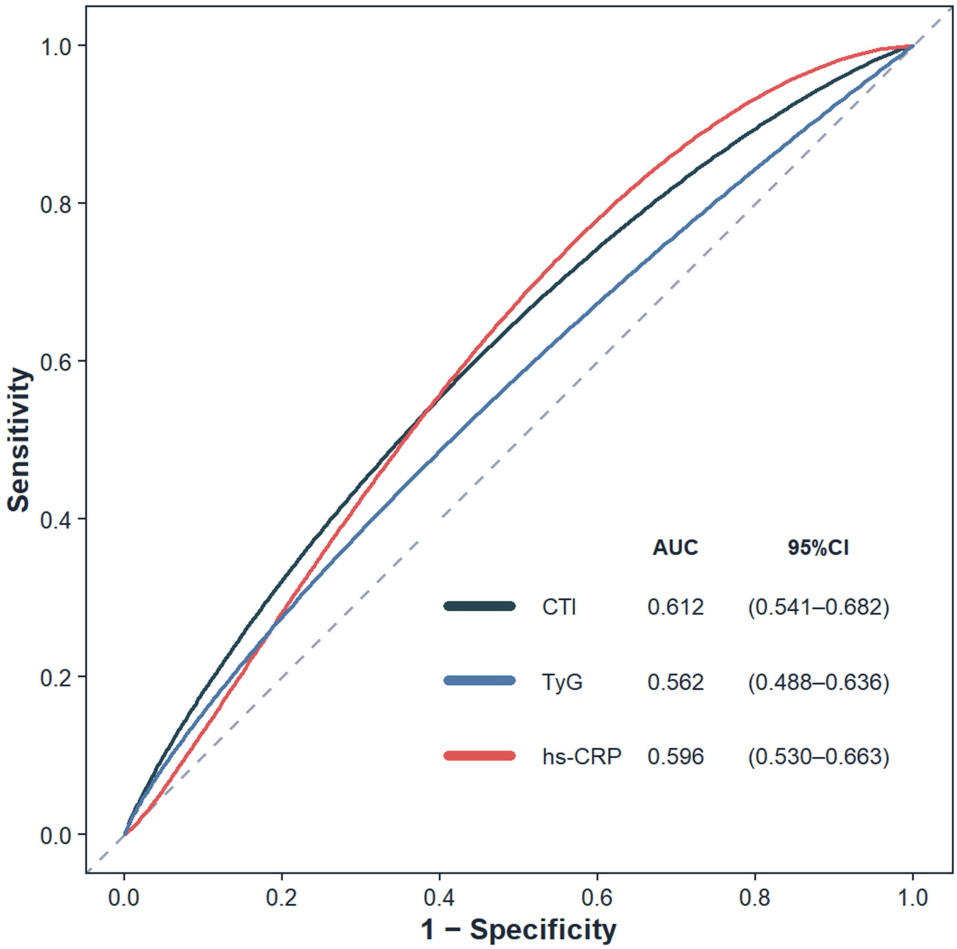

Figure 3 Receiver operating characteristic curve analysis of the CTI, TyG index and hs-CRP for MACEs.
Abbreviations: CTI, C-reactive protein-triglyceride glucose index; hs-CRP, high-sensitivity C-reactive protein; MACEs, major adverse cardiovascular events; TyG, triglyceride-glucose.

cumulative risk of MACEs gradually increased. The group with the highest CTI had the highest risk during the 36-month follow-up (Figure 4). Multivariate Cox regression analysis (Table 3) revealed that the association between CTI and MACEs remained significant after progressive adjustment for confounders, including age, gender, BMI, hypertension status, HDL-C level, and LDL-C level. In Model 3, patients in the highest CTI tertile had a 3.09-fold increased risk of

Table 2 The Discriminative Value of the CTI, TyG, Hs-CRP for the Risk of MACEs

	AUC (95% CI)	*p* Value	Continuous NRI (95% CI)	*p* Value	IDI (95% CI)	*p* Value
Baseline	0.702(0.635 to 0.770)		Reference		Reference	
+CTI	0.716(0.649 to 0.783)	0.370	0.421(0.177 to 0.665)	<0.001	0.021(0.005 to 0.038)	0.011
+TyG	0.704(0.636 to 0.772)	0.847	0.143(−0.108 to 0.394)	0.263	0.005(−0.003 to 0.012)	0.241
+hs-CRP	0.708(0.641 to 0.774)	0.663	0.214(−0.025 to 0.453)	0.080	0.012(−0.001 to 0.025)	0.053

Notes: The baseline model was adjusted for age, gender, BMI, hypertension status, HDL-C, and LDL-C.
Abbreviations: AUC, area under the curve; CI, confidence interval; CTI, C-reactive protein-triglyceride glucose index; hs-CRP, high-sensitivity C-reactive protein; IDI, integrated discrimination improvement; MACEs, major adverse cardiovascular events; NRI, net reclassification index; TyG, triglyceride-glucose.

MACEs ($p < 0.001$), compared with patients in the lowest tertile. Furthermore, when analyzed as a continuous variable, the CTI remained a robust independent predictor (HR: 1.64; 95% CI: 1.23–2.18; $p < 0.001$), indicating a 64% increase in MACEs risk for each 1-unit increase in the CTI. To further address potential confounding from AMI-specific prognostic variables, we constructed an extended Cox model incorporating diabetes, hyperlipidemia, smoking status, GRACE score, LVEF, and PCI. The association between CTI and MACEs remained robust across this extended adjustment (Supplementary Table S3), supporting the independence of CTI as a prognostic marker beyond established AMI risk variables.

Subgroup Analysis

No significant interactions were observed across the subgroups stratified by age, gender, BMI, hypertension status, diabetes status, or smoking status. These results demonstrate that the prognostic value of the CTI for MACEs across these diverse clinical subpopulations is robust and consistent (Figure 5).

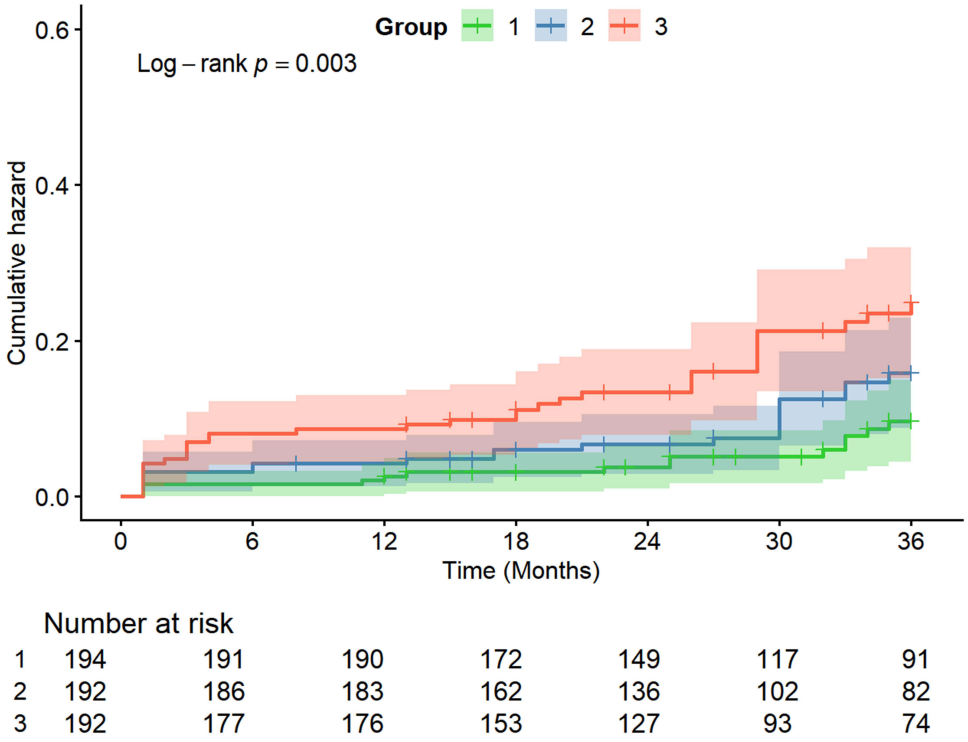

Figure 4 Kaplan–Meier survival curves to illustrate the cumulative risk of MACEs in groups based on CTI tertiles.
Abbreviations: CTI, C-reactive protein-triglyceride glucose index; MACEs, major adverse cardiovascular events.

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Table 3 Cox Regression Models for the Relationship Between the CTI and MACEs

Variables	Model 1		Model 2		Model 3	
	HR (95% CI)	*P* Value	HR (95% CI)	*P* Value	HR (95% CI)	*p* Value
CTI	1.53 (1.18–1.99)	<0.001	1.62 (1.23–2.14)	<0.001	1.64 (1.23–2.18)	<0.001
T1	Reference		Reference		Reference	
T2	1.62 (0.82–3.19)	0.161	1.66 (0.84–3.26)	0.144	1.74 (0.87–3.49)	0.120
T3	2.75 (1.48–5.13)	<0.001	2.81 (1.51–5.26)	<0.001	3.09 (1.60–5.97)	<0.001
p for trend	<0.001		<0.001		<0.001	

Notes: Model 1: unadjusted for any confounders; Model 2: adjusted for age, gender, and BMI; Model 3: adjusted for age, gender, BMI, hypertension, HDL-C, and LDL-C.
Abbreviations: CI, confidence interval; CTI, C-reactive protein-triglyceride glucose index; HR, hazard ratio; MACEs, major adverse cardiovascular events.

Discussion

This research first compared the CTI, hs-CRP level, and TyG index in predicting MACEs in patients with AMI. Our results demonstrate that the CTI is a more promising indicator for identifying high-risk individuals. Further analysis revealed that the CTI remained significantly associated with MACEs after controlling for confounding factors, whether analyzed as a continuous or categorical variable. These results indicate that the CTI may be a practical biomarker for identifying high-risk patients with AMI.

Although secondary prevention has made substantial advances, MACEs still occur after AMI.[25] Identifying and managing residual risks effectively is essential for enhancing patient outcomes. The CTI reflects two core components: systemic inflammation (hs-CRP level) and IR (TyG index). Each contributes substantially to residual cardiovascular risk and is linked to an increased incidence of CVD.[26–28]

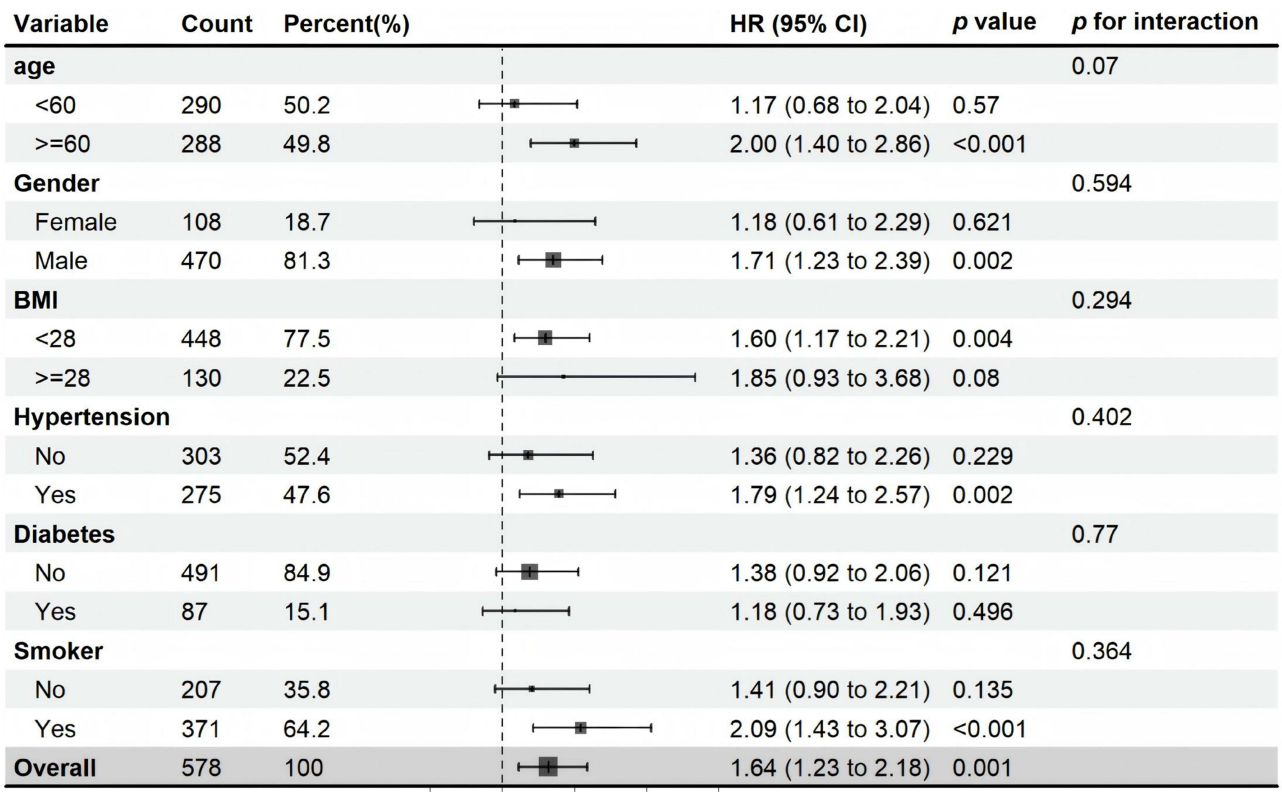

Figure 5 Subgroup and interaction analysis for the association between the CTI and MACEs. Adjusted model included age, gender, BMI, hypertension, HDL-C and LDL-C.
Abbreviations: BMI, body mass index; CI, confidence interval.

IR refers to an inability of the liver, muscles, and adipose tissue to effectively use insulin, resulting in chronic hyperglycemia and significantly increasing the long-term risk of developing type 2 diabetes.[29,30] The gold standard for evaluating IR is currently considered to be the hyperinsulinemic–euglycemic clamp. The clamp technique is laborious and costly, limiting its use in large epidemiological studies and routine clinical practice.[31] Alternatively, the TyG index is often used as a method for assessing IR. Nevertheless, the ability of the TyG index to predict adverse cardiovascular events across different populations remains controversial. According to a study by Luo et al, elevated TyG index levels serve as a robust and independent prognostic marker for MACEs in STEMI patients within one year following percutaneous coronary intervention (PCI).[32] In contrast, Rokicka et al reported that the TyG index had a low predictive capacity for MACEs within 12 months in a population of 1706 AMI patients.[33] In this study, the ability of the TyG index to predict MACEs in patients with AMI was insufficient. Several studies have indicated that the integration of the TyG index and measurement of inflammatory biomarkers provides greater predictive significance for MACEs after AMI.[34,35]

Post-AMI inflammation is characterized by distinct temporal phases and is subject to multifactorial regulation. It commences with a proinflammatory phase, characterized by neutrophil and macrophage infiltration within 72 h to clear necrotic tissue. This is followed by a reparative phase. However, disruption of this regulation can result in chronic low-grade inflammation and poor prognosis.[36,37] This state of persistent low-grade inflammation represents a critical target for assessing residual cardiovascular risk after AMI. Hs-CRP level is an important biomarker for assessing this persistent, low-grade inflammatory state.[25,27] In our study, we found that the CTI, a combination of hs-CRP level and TyG index, exhibited better predictive performance. In particular, although CTI did not significantly improve the AUC of the baseline model, it was associated with significant improvements in both continuous and categorical NRI, as well as IDI. The categorical NRI of 0.131 indicated significant improvement in category-based risk reclassification when CTI was added to the baseline model. Given the relatively limited sample size and number of MACEs in our cohort, the magnitude of these improvements should be interpreted with caution. Nevertheless, the overall findings still support the potential value of integrating information on inflammation and IR for risk stratification after AMI. Gao et al also found that the most reliable indicator among IR and inflammatory indicators for predicting the recurrence of cardiovascular risk in patients treated with PCI was CTI.[38] A nationwide cohort study investigated the combined association of the TyG index and hs-CRP levels with CVD. The results demonstrated that these markers exerted a synergistic effect on cardiovascular risk. The risk of CVD was highest among patients with high TyG and hs-CRP levels when compared to individuals with low levels of these indicators.[39] In addition, a multicenter cohort study involving 9421 patients with chronic coronary syndrome investigated the combined effect of TyG and hs-CRP. The study found that among patients with a high TyG index, the risk of MACE was significantly increased only when hs-CRP levels were also elevated.[40] These findings indicate that combining assessment of inflammation and IR appears to be a more powerful predictive strategy than using either marker alone.

In the present study, as CTI tertile increased, the cumulative risk of MACEs gradually increased. The group with the highest CTI showed the highest risk during the 36-month follow-up. Previous research in middle-aged and elderly adults revealed that both cumulative CTI and dynamic CTI are independently linked to an elevated risk of CVD.[41] Elevated CTI has also been significantly linked to the incidence of CVD.[22] Additionally, higher CTI is significantly linked to increased all-cause mortality at 30-day and 1-year follow-ups, along with longer hospital stays in critically ill patients.[42] We also found that patients in the highest CTI group had a higher prevalence of three-vessel disease. Previous studies have demonstrated that both the TyG index and hs-CRP are associated with the severity of coronary artery disease, and our findings indicate that combining these indicators similarly reveals this association.[43,44] In our Cox proportional hazards regression analyses, the association between CTI and MACEs remained significant across the main models, and this finding was further supported by the extended model. No significant interactions were detected across the subgroups stratified by age, gender, BMI, hypertension status, diabetes status, or smoking status. These results demonstrate that the prognostic value of CTI for MACEs across these diverse clinical subpopulations is robust and consistent.

There are possible underlying mechanisms by which the CTI is linked to poor clinical outcomes in patients with AMI. First, the CTI reflects two core components, systemic inflammation (hs-CRP level) and IR (TyG index), which together make the evaluation of metabolic-inflammatory status more comprehensive.[42,45,46] Second, these two pathways are closely linked. IR itself acts as a proinflammatory state that promotes cytokine release. This systemic inflammation in turn exacerbates IR by affecting its signaling pathways.[47–49] Finally, considering the substantial inter-individual variability in biomarker levels, the

TyG index and hs-CRP levels respectively reflect insulin sensitivity and inflammatory status, thus providing complementary biological information.[39,50]

CTI is calculated from three simple laboratory parameters, including TG, FBG, and hs-CRP, all of which are routinely recommended for assessment in AMI patients,[2,25] making CTI calculation feasible without additional cost or specialized equipment. Clinicians can use this composite index, which integrates both inflammatory and IR status, to assess prognostic risk in AMI patients. However, current research on CTI lacks a validated risk threshold for clinical application. In our cohort, the optimal CTI cut-off value was 9.46, yielding a sensitivity of 71.0% and a specificity of 52.7% for predicting 3-year MACEs. This statistically derived threshold closely approximates the boundary between the second and third tertiles of CTI in our analysis, supporting the internal consistency of CTI tertile-based stratification. These findings provide a preliminary reference for CTI-based risk stratification in AMI patients. Nevertheless, in our study, CTI components were measured before discharge, a timepoint influenced by multiple factors that may systematically affect biomarker values. Specifically, hs-CRP levels may remain elevated due to incomplete resolution of the acute-phase response following AMI, whereas TG levels may be reduced by in-hospital dietary restriction, fasting protocols, and medications. These opposing influences could result in systematic bias in CTI values. Therefore, both the optimal timing of CTI measurement and a validated risk threshold in AMI patients warrant further investigation in future prospective and multicenter studies.

Residual cardiovascular risk after AMI arises from multiple factors. Although CTI reflects an important metabolic-inflammatory dimension of residual risk, biomarker-based assessment alone cannot fully capture the complexity of long-term adverse outcomes after AMI. Recent evidence has shown that high-risk plaque morphology and impaired coronary physiology are independently associated with adverse prognosis in patients with coronary artery disease.[23] In this context, CTI should be regarded as a simple and readily available complementary biomarker rather than a standalone prognostic tool. Nevertheless, our findings suggest that CTI may provide additional value for post-discharge risk stratification, helping to identify high-risk patients who may benefit from precise management.

Limitations

This study has several limitations. First, this was a retrospective single-center study, and therefore selection bias and residual confounding cannot be completely excluded. Second, the relatively limited sample size and number of MACEs constrained the extent of confounder adjustment in the multivariable analyses. In particular, because NRI is sensitive to sample size and event rate, the precision of the observed reclassification improvement may be limited. Third, the biomarkers used to calculate CTI were measured at the pre-discharge stage rather than at admission. Although this timing was intended to reflect a more stable condition after the acute phase of AMI, these values may still be influenced by acute-phase physiology, dietary restriction, and in-hospital treatment. Although standard pharmacological therapies, including statins, renin-angiotensin-aldosterone system (RAAS) inhibitors, beta-blockers, and antiplatelet agents, were applied as part of routine clinical care in all enrolled patients, these in-hospital treatments could have systematically affected the pre-discharge biomarker values. Therefore, the absolute CTI values and proposed threshold levels identified in this cohort may have limited generalizability to other clinical settings or measurement time points. Finally, larger prospective multicenter studies with longer follow-up and serial biomarker measurements across different disease stages are needed to further validate and refine the prognostic value of CTI.

Conclusion

The CTI is a more promising indicator for predicting MACEs in AMI patients than the TyG index or hs-CRP alone. By integrating the core risk factors of inflammation and insulin resistance, CTI may provide additional value for post-discharge risk stratification, helping to identify high-risk patients who may benefit from precise management. Future studies integrating additional residual risk indicators may further improve prognostic prediction in AMI patients.

Data Sharing Statement

The data that support the findings of this study are available on request from the corresponding author.

Ethics Statement and Informed Consent

The study involving human participants was approved by the Ethics Committee of Xuanwu Hospital (Document No. 2022-129). The study was conducted in accordance with local legislation and institutional requirements. The requirement for informed consent was waived by the Ethics Committee given the retrospective nature of the study.

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Author Contributions

Tao Wang: Conceptualization, Formal analysis, Data curation, Methodology, Software, Supervision, Validation, Visualization, Writing - original draft. Guoheng Zhang: Data curation, Methodology, Software, Validation, Writing - original draft. Qinxue Li: Conceptualization, Data curation, Methodology, Validation, Writing - original draft. Yayun Liu: Data curation, Methodology, Software, Validation, Writing - original draft. Tao Zhao: Data curation, Methodology, Validation, Writing - original draft. Wenjing Che: Data curation, Methodology, Validation, Writing - original draft. Jinggang Xia: Conceptualization, Funding acquisition, Methodology, Project administration, Resources, Supervision, Visualization, Writing - review & editing. All authors reviewed and approved the final version of the manuscript and have agreed to its submission. All authors accept responsibility for the integrity and accuracy of the entire work.

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Disclosure

The authors declare no conflicts of interest.

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