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Original Article

Predictive Values of the Blood CHG Index Combined with MHR or TG/HDL-C for Non-ST-Segment Elevation Acute Coronary Syndrome: A Cross-sectional Diagnostic Study

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Abstract

Background and objectives: This study aimed to evaluate the predictive value of cholesterol, high-density lipoprotein, and glucose index (CHG index) alone and combined with the monocyte-to-high-density lipoprotein cholesterol ratio (MHR) or triglyceride-to-high-density lipoprotein cholesterol ratio (TG/HDL-C) for NSTE-ACS.

Methods: This cross-sectional diagnostic study included 150 patients with NSTE-ACS and 76 healthy controls. Based on the median Gensini score, patients were divided into high-risk (Gensini score ≥51, n = 75) and low-risk groups (Gensini score <51, n = 75). MHR, TG/HDL-C, and CHG index were compared between patients and controls and between high- and low-risk groups. Their correlations with Gensini scores were assessed. Univariate and Multivariate binary logistic regression analysis was performed to identify factors independently associated with high-risk coronary lesions among patients with NSTE-ACS. The predictive performance of individual indicators (MHR, TG/HDL-C, and CHG index) and their combinations was evaluated using receiver operating characteristic curve analysis.

Results: MHR, TG/HDL-C, and CHG index were significantly higher in the patient group than in the control group (all P < 0.001) and the high-risk group than the low-risk group. Those indicators positively correlated with Gensini scores and were independently associated with high-risk coronary lesions among patients with NSTE-ACS (odds ratio (OR) = 16.051, 95% confidence interval (CI): 13.677-99.650 for MHR; OR = 3.562, 95% CI: 1.868-6.793 for TG/HDL-C; and OR = 2.455, 95% CI: 1.040-5.791 for CHG index). The areas under the curve (AUCs) were 0.803 (95% CI: 0.730-0.876) for MHR, 0.746 (95% CI: 0.666-0.826) for TG/HDL-C, and 0.659 (95% CI: 0.573-0.746) for CHG index. The combination of CHG index and TG/HDL-C achieved an AUC of 0.821 (95% CI: 0.755-0.887), while the combination of CHG index and MHR achieved the higher AUC of 0.872 (95% CI: 0.815-0.929).

Conclusions: MHR, TG/HDL-C, and CHG index are independently associated with high-risk coronary lesions among patients with NSTE-ACS. Combining CHG index with MHR or TG/HDL-C shows numerically higher AUCs for identifying high-risk coronary lesions.

Keywords: CHG index; MHR; TG/HDL-C; Non-ST-segment elevation acute coronary syndrome; Combined diagnosis; Gensini score; Biomarker.
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Introduction

Acute coronary syndrome (ACS) is a major threat to global health,[1,2] which includes ST-segment elevation acute myocardial infarction, non-ST-segment elevation acute myocardial infarction (NSTEMI), and unstable angina pectoris (UA). Among them, UA and NSTEMI are called non-ST-segment elevation acute coronary syndrome (NSTE-ACS),[2] accounting for a significant incidence

and mortality of cardiovascular disease.[3] Therefore, timely assessment and prediction of the condition in NSTE-ACS patients are particularly crucial.

Established studies have also demonstrated that inflammatory neutrophils (NEUTs), monocytes (MONOs), and white blood cells (WBCs) are closely associated with lipid metabolic disorders.[4] Cardiovascular diseases are markedly correlated with the quantity and activity of inflammatory cells in peripheral blood, particularly MONOs, which can participate in anti-inflammatory responses while also promoting the progression of atherosclerotic lesions.[5,6]

The monocyte-to-high-density lipoprotein cholesterol ratio (MHR) has emerged as a sensitive indicator of vascular inflammation,[7,8] integrating the pro-inflammatory activity of MONOs and the anti-inflammatory, atheroprotective properties of high-density lipoprotein cholesterol (HDL-C), thereby providing a multidimensional perspective on its relationship with the occurrence and development of ACS. Additionally, the triglyceride-to-high-density lipoprotein cholesterol (TG/HDL-C) ratio has become a reliable marker for insulin resistance or type 2 diabetes in patients.[9,10] Meanwhile, considerable clinical evidence has also documented that TG/HDL-C is useful for identifying angiographically defined coronary artery disease.[11] Thus, MHR and TG/HDL-C are established markers that capture aspects of inflammation and lipid metabolism.

Moreover, cholesterol, high-density lipoprotein, and glucose index (CHG index) is an index integrating cholesterol (TC), HDL-C, and fasting blood glucose (FBG).[12] Although it is a simple combination of metabolic markers, it reflects the overall glucolipid metabolic status and serves as an effective tool for revealing metabolic disorders. CHG index has been associated with an increased risk of cardiovascular disease.[13] However, CHG index does not incorporate inflammatory components, which occupy a critical position in the pathogenesis of ACS. Given that NSTE-ACS is caused by interactions between metabolic disturbances and inflammatory processes, we hypothesized that combining a comprehensive metabolic index, such as the CHG index, with the inflammatory marker MHR or insulin resistance marker TG/HDL-C would yield a more powerful tool for assessing disease severity. While the individual utility of these markers has been explored, their combined predictive value in NSTE-ACS patients has not been investigated. Therefore, the aim of the present study was to explore the predictive values of CHG index alone and in combination with MHR or TG/HDL-C for NSTE-ACS.

Materials and methods

Participant recruitment

This single-center, prospective, cross-sectional study enrolled 150 NSTE-ACS patients at the Department of Cardiovascular Medicine, Jiangsu Provincial Hospital of Traditional Chinese Medicine, from January 2023 to January 2025. A formal pre-experimental sample size calculation was not performed for this exploratory analysis. The research received approval from the Ethics Committee of the Affiliated Hospital of Nanjing University of Chinese Medicine (Approval No. 2021NL-073-02) in accordance with the principles of the Declaration of Helsinki (as revised in 2024), and written informed consent was obtained from all participants prior to enrollment.

Based on a median Gensini score of 51, participants were categorized into a high-risk group (score ≥51, n = 75) and a low-risk group (score <51, n = 75). This cross-sectional design evaluated disease severity at a single time point (first hospitalization). A study flowchart is provided in Supplementary Fig. 1.

Inclusion criteria: (1) Diagnosis of UA or NSTEMI based on clinical symptoms, electrocardiographic findings, and dynamic changes in myocardial injury markers; (2) coronary angiography performed during hospitalization. Exclusion criteria: (1) Patients with other valvular heart diseases, cardiomyopathy, pericarditis, or pulmonary heart disease; (2) presence of stroke, diabetes, glucose metabolism disorders, malignant tumors, systemic infection, hematological disorders, or immune diseases; (3) organ transplantation or use of immunosuppressive medications. Additionally, we recruited 76 healthy controls from the Health Examination Center of the hospital during the same period.

Angiography and Gensini scoring

Standard percutaneous techniques were used to perform coronary angiography via the femoral or radial artery. Adequate visualization of all major coronary vessels and their branches was achieved through multiple angiographic projections. To ensure objective assessment, all angiograms were independently interpreted by two experienced interventional cardiologists who were blinded to participants' clinical and laboratory information. The Gensini scoring system was applied to quantify the extent and severity of coronary atherosclerosis.[14] Stenosis severity was categorized as 1–25% (score 1), 26–50% (score 2), 51–75% (score 4), 76–90% (score 8), 91–99% (score 16), or total occlusion (score 32). Each lesion score was then adjusted by a coefficient corresponding to the specific coronary segment's vascular territory. The cumulative Gensini score, representing the sum of all weighted segmental scores, served as the final measure of coronary disease burden. In instances of discordant readings, a third senior cardiologist was consulted to achieve consensus.

Specimen collection and processing

Venous blood samples were collected using EDTA-K_2 anticoagulant tubes and gel-separator serum tubes. Serum was promptly separated, and biochemical parameters were measured using a Beckman-Coulter 5800 automatic biochemical analyzer. Blood cell counts (MONO, NEUT, WBC) were determined using a Mindray automated hematology analyzer. All analyses were performed at the hospital's accredited Clinical Laboratory, which adheres to strict internal quality control procedures. Laboratory technicians were blinded to participants' clinical information. The intra- and inter-assay coefficients of variation for all tests were below 5%, ensuring high precision for the calculation of MHR, TG/HDL-C, and CHG index.

Calculation formulas

MHR = MONO (×10⁹/L) / HDL-C (mmol/L), neutrophil to high-density lipoprotein cholesterol ratio (NHR) = NEUT (×10⁹/L) / HDL-C (mmol/L), white blood cell to high-density lipoprotein cholesterol ratio (WHR) = WBC (×10⁹/L) / HDL-C (mmol/L),[7] CHG index = ln [TC (mg/dL) × FBG (mg/dL)/(2 × HDL-C (mg/dL))],[13] TG/HDL-C = TG (mg/dL) / HDL-C (mg/dL).[11] Additionally, 1 mg/dL= (10/Molecular) ×1 mmol/L.

Table 1. Comparison of basic data between the control group and the NSTE-ACS group

Variable	Control group (*n* = 76)	NSTE-ACS group (*n* = 150)	t/Z/X^2	*P*
Age (years)	66.5(60, 72)	65(57, 75)	0.057	0.954
Male, *n* (%)	50(65.8%)	106(70.7%)	0.561	0.454
TC (mmol/L)	3.45(2.37, 4.26)	4.6(3.88, 5.62)	-6.994	<0.001
TG (mmol/L)	1.32(0.92, 1.75)	1.61(1.23, 2.14)	-3.573	<0.001
LDL-C (mmol/L)	2.34(1.77, 3.1)	2.44(1.66, 2.98)	-0.25	0.803
HDL-C (mmol/L)	1.25(1.07, 1.42)	1.02(0.9, 1.22)	5.343	<0.001
FBG (mmol/L)	5.01(4.59, 5.41)	5.7(4.84, 6.87)	-4.836	<0.001
MONO (×10^9/L)	0.4(0.31, 0.5)	0.54(0.4, 0.7)	-4.278	<0.001
NEUT (×10^9/L)	3.45(2.8, 4.18)	4.86(3.99, 6.41)	-6.621	<0.001
WBC (×10^9/L)	6.3(5.24, 7.38)	8.21(7.13, 9.69)	-6.477	<0.001
MHR	0.33(0.22, 0.45)	0.53(0.36, 0.74)	-5.482	<0.001
NHR	2.83(2.15, 3.66)	4.88(3.69, 6.58)	-7.528	<0.001
WHR	4.96(4.13, 6.7)	7.81(6.62, 9.59)	-6.964	<0.001
TG/HDL-C	1.14(0.65, 1.62)	1.61(1.08, 2.33)	-4.954	<0.001
CHG index	2.34(1.92, 2.62)	2.65(2.28, 2.92)	-4.694	<0.001

CHG index, cholesterol, high-density lipoprotein, and glucose index; FBG, fasting blood glucose; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MHR, monocyte-to-high-density lipoprotein cholesterol ratio; MONO, monocyte; NEUT, neutrophil; NHR, neutrophil-to-high-density lipoprotein cholesterol ratio; NSTE-ACS, non-ST-segment elevation acute coronary syndrome; TC, total cholesterol; TG, triglyceride; TG/HDL-C, triglyceride-to-high-density lipoprotein cholesterol ratio; WBC, white blood cell; WHR, white blood cell-to-high-density lipoprotein cholesterol ratio.

Statistical analysis

SPSS version 25.0 was utilized for all statistical computations. The Kolmogorov-Smirnov test was employed to examine the distributional properties of continuous variables. Depending on normality or non-normality, data were expressed either as mean ± standard deviation (analyzed by independent samples t-test) or as median with interquartile range (analyzed by Mann-Whitney U test). Categorical variables were presented as percentages and compared using the χ^2 test. Spearman's rank correlation was used to assess relationships between variables. Univariate logistic regression identified variables with $P < 0.05$, which were then considered for entry into separate multivariable binary logistic regression models. Variables with $P < 0.05$ in the multivariate analysis were considered independent influencing factors. Variance inflation factors (VIF) were used to assess multicollinearity among the independent variables, with a VIF < 5. Three separate models were used to evaluate CHG index, MHR, and TG/HDL-C individually, and direct component variables of the corresponding composite biomarker were not adjusted in the same model. For combined biomarker analyses, predicted probabilities generated from the CHG index + MHR and CHG index + TG/HDL-C logistic regression models were analyzed using receiver operating characteristic (ROC) curves to assess their discriminatory capacity, as well as that of the individual markers. Optimal cut-off values were determined using the Youden index. Because no pairwise area under the curve (AUC) comparison was reported, differences between AUCs were interpreted descriptively. A significance threshold of $P < 0.05$ (two-tailed) was applied for all tests.

Results

The clinical characteristics and laboratory parameters

There were no significant differences between the NSTE-ACS group and control group in age ($P = 0.954$) and sex ($P = 0.454$). The NSTE-ACS group and healthy subjects differed significantly in TC, triglyceride (TG), FBG, HDL-C, MONO, NEUT, WBC, MHR, NHR, WHR, TG/HDL-C, and CHG index ($P < 0.001$ for all) (Table 1).

As detailed in Table 2, the high-risk group displayed significantly higher levels of TC, FBG, TG, low-density lipoprotein cholesterol (LDL-C), MONO, NEUT, MHR, NHR, WHR, TG/HDL-C, and CHG index, whereas HDL-C levels were significantly lower ($P < 0.05$).

Spearman correlation analysis between MHR, NHR, WHR, TG/HDL-C, CHG index, and Gensini score

Correlation analysis revealed that MHR, NHR, WHR, TG/HDL-C, and CHG index were significantly correlated with Gensini scores. The most notable correlation was observed for MHR ($r_s = 0.759$, $P < 0.001$), followed by NHR ($r_s = 0.625$, $P < 0.001$), TG/HDL-C ($r_s = 0.468$, $P < 0.001$), WHR ($r_s = 0.466$, $P < 0.001$), and CHG index ($r_s = 0.268$, $P = 0.0137$) (Table 3). These data indicated that higher values of these inflammatory and metabolic markers are associated with coronary atherosclerotic disease.

Table 2. Comparison of data among patients with different degrees of coronary artery lesions

Variable	Low-risk group (*n* = 75)	High-risk group (*n* = 75)	t/Z/X^2	*P*
Age (years)	66.51±11.13	64.87±13.21	0.822	0.412
Male, *n* (%)	48(64%)	58(77.3%)	3.216	0.073
Systolic pressure (mmHg)	128(118,142)	129(123,141)	-0.621	0.535
Diastolic pressure (mmHg)	84(73,97)	87(78,98)	-1.311	0.190
Hypertension	32(42.7%)	29(38.7%)	0.249	0.618
Smoking	43(57.3%)	38(50.7%)	0.671	0.413
BMI (kg/m^2)	24(21,29)	25(23,28.6)	-1.892	0.058
TC (mmol/L)	4.31±1.24	5.31±1.24	-4.95	<0.001
TG (mmol/L)	1.42(1.05, 1.84)	1.91(1.43, 2.68)	-4.065	<0.001
LDL-C (mmol/L)	2.21(1.56, 2.77)	2.53(2.07, 3.42)	-2.738	0.006
HDL-C (mmol/L)	1.11(0.94, 1.29)	0.93(0.87, 1.09)	3.645	<0.001
FBG (mmol/L)	5.22(4.52, 6.26)	6.11(5.24, 7.67)	-4.137	<0.001
MONO ($\times10^9$/L)	0.43(0.33, 0.58)	0.63(0.51, 0.8)	-5.848	<0.001
NEUT ($\times10^9$/L)	4.26(3.2, 5.69)	5.4(4.61, 7.17)	-4.817	<0.001
WBC ($\times10^9$/L)	7.09(5.62, 8.67)	8.43(7.11, 9.69)	-0.839	0.402
MHR	0.41(0.29, 0.5)	0.69(0.54, 0.88)	-6.407	<0.001
NHR	3.8(3.18, 4.95)	5.98(4.85, 7.49)	-6.247	<0.001
WHR	7.38(6.12, 8.54)	8.42(7.3, 10.56)	-3.793	<0.001
TG/HDL-C	1.21(0.95, 1.62)	2.08(1.5, 2.53)	-5.2	<0.001
CHG index	2.52(2.13, 2.85)	2.78(2.48, 3.11)	-3.37	0.001

BMI, body mass index; CHG index, cholesterol, high-density lipoprotein, and glucose index; FBG, fasting blood glucose; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; MHR, monocyte-to-high-density lipoprotein cholesterol ratio; MONO, monocyte; NEUT, neutrophil; NHR, neutrophil-to-high-density lipoprotein cholesterol ratio; TC, total cholesterol; TG, triglyceride; TG/HDL-C, triglyceride-to-high-density lipoprotein cholesterol ratio; WBC, white blood cell; WHR, white blood cell-to-high-density lipoprotein cholesterol ratio.

Table 3. Spearman's correlation analysis

	MHR	NHR	WHR	TG/HDL-C	CHG index
r_s	0.759	0.625	0.466	0.468	0.268
P	<0.001	<0.001	<0.001	<0.001	0.0137

CHG index, cholesterol, high-density lipoprotein, and glucose index; MHR, monocyte-to-high-density lipoprotein cholesterol ratio; NHR, neutrophil-to-high-density lipoprotein cholesterol ratio; TG/HDL-C, triglyceride-to-high-density lipoprotein cholesterol ratio; WHR, white blood cell-to-high-density lipoprotein cholesterol ratio.

Analysis of those indicators for high-risk coronary lesions among patients with NSTE-ACS

We performed binary logistic regression analysis to examine the relationship between biomarkers and coronary artery disease severity. Univariate logistic regression of Table 4, significant associations were observed for TG, LDL-C, MONO, NEUT, CHG index, and smoking status. After adjustment via multivariate logistic regression, TG (odds ratio (OR) = 2.446, 95% confidence interval (CI): 1.353-4.422), MONO (OR = 13.055, 95% CI: 5.946-68.683), and CHG index (OR = 2.455, 95% CI: 1.040-5.791) remained significant. Univariate analysis of Table 5 revealed significant associations for TG, LDL-C, MHR, NEUT, FBG, TC, and smoking status. Following multivariate regression, TG (OR = 2.504, 95% CI: 1.317-4.762), MHR (OR = 16.051, 95% CI: 13.677-99.650), FBG (OR = 1.428, 95% CI: 1.076-1.895), and TC (OR = 1.665, 95% CI: 1.144-2.424) were identified as significant independent factors. Univariate logistic regression of Table 6 identified significant variables including LDL-C, MONO, TG/HDL-C, NEUT, FBG, TC, and smoking status. After multivariate adjustment, MONO (OR = 18.681, 95% CI: 3.750-61.951), TG/HDL-C (OR = 3.562, 95% CI: 1.868-6.793), FBG (OR = 1.458, 95% CI: 1.093-1.945), and TC (OR = 1.693, 95% CI: 1.162-2.468) remained statistically significant. Therefore, CHG index, MHR and TG/HDL-C were independently associated with high-risk coronary lesions among patients with NSTE-ACS. Additionally, the results showed that all significantly independent variables had VIF values between 1 and 5, indicating no significant multicollinearity among them.

Predictive value of MHR, TG/HDL-C, CHG index, and combinations

ROC curve analysis demonstrated that the combination of CHG

Table 4. Logistic regression model including CHG index in NSTE-ACS patients

Variable	univariate-OR (95%CI)	uni-*P*	Multi-OR (95%CI)	multi-*P*	VIF
TG (mmol/L)	2.529 (1.523-4.198)	<0.001	2.446(1.353-4.422)	0.003	1.048
LDL-C (mmol/L)	1.530 (1.066-2.197)	0.021	1.590(0.983-2.574)	0.059	1.175
WBC ($\times10^9$/L)	0.513 (0.334-2.554)	0.510			
MONO ($\times10^9$/L)	11.168 (3.653-96.249)	<0.001	13.055(5.946-68.683)	0.001	1.432
Systolic pressure (mmHg)	1.006 (0.982-1.030)	0.635			
Diastolic pressure (mmHg)	1.017 (0.990-1.045)	0.225			
BMI (kg/m^2)	1.065 (0.990-1.145)	0.092			
NEUT ($\times10^9$/L)	1.630 (1.306-2.034)	<0.001	1.143(0.881-1.483)	0.313	1.479
CHG index	3.427 (1.691-6.945)	<0.001	2.455(1.040-5.791)	0.040	1.182
Gender	1.919 (0.937-3.932)	0.075			
Age	0.989 (0.963-1.015)	0.410			
Smoking	2.047 (1.030-4.066)	0.041	1.838(0.781-4.327)	0.163	1.021
Hypertension	0.847 (0.441-1.626)	0.618			

BMI, body mass index; CHG index, cholesterol, high-density lipoprotein, and glucose index; CI, confidence interval; LDL-C, low-density lipoprotein cholesterol; MONO, monocyte count; NEUT, neutrophil count; NSTE-ACS, non-ST-segment elevation acute coronary syndrome; OR, odds ratio; TG, triglyceride; VIF, variance inflation factor; WBC, white blood cell count.

Table 5. Logistic regression model including MHR in NSTE-ACS patients

Variable	univariate-OR (95%CI)	uni-*P*	Multi-OR (95%CI)	multi-*P*	VIF
TG (mmol/L)	2.529 (1.523-4.198)	<0.001	2.504(1.317-4.762)	0.005	1.054
LDL-C (mmol/L)	1.530 (1.066-2.197)	0.021	1.670(0.986-2.831)	0.057	1.158
MHR	16.732 (14.221-88.910)	<0.001	16.051(13.677-99.650)	<0.001	1.384
Systolic pressure (mmHg)	1.006 (0.982-1.030)	0.635			
Diastolic pressure (mmHg)	1.017 (0.990-1.045)	0.225			
BMI (kg/m^2)	1.065 (0.990-1.145)	0.092			
WBC ($\times10^9$/L)	0.513 (0.334-2.554)	0.510			
NEUT ($\times10^9$/L)	1.630 (1.306-2.034)	<0.001	1.052(0.804-1.377)	0.710	1.403
FBG (mmol/L)	1.526 (1.220-1.908)	<0.001	1.428(1.076-1.895)	0.014	1.118
Gender	1.919 (0.937-3.932)	0.075			
Age	0.989 (0.963-1.015)	0.410			
TC (mmol/L)	1.912 (1.425-2.565)	<0.001	1.665(1.144-2.424)	0.008	1.184
Smoking	2.047 (1.030-4.066)	0.041	2.297(0.854-6.173)	0.099	1.027
Hypertension	0.847 (0.441-1.626)	0.618			

BMI, body mass index; CI, confidence interval; FBG, fasting blood glucose; LDL-C, low-density lipoprotein cholesterol; MHR, monocyte-to-high-density lipoprotein cholesterol ratio; NEUT, neutrophil count; NSTE-ACS, non-ST-segment elevation acute coronary syndrome; OR, odds ratio; TC, total cholesterol; TG, triglyceride; VIF, variance inflation factor; WBC, white blood cell count.

and MHR showed a numerically higher predictive efficacy, with an AUC of 0.872 (95% CI: 0.815-0.929, $P < 0.001$). The combination of CHG and TG/HDL-C exhibited high predictive capacity, with an AUC of 0.821 (95% CI: 0.755-0.887, $P < 0.001$). Among individual markers, MHR performed best, with an AUC of 0.803 (95% CI: 0.730-0.876, $P < 0.001$) and a cut-off value of 0.504, followed by TG/HDL-C with an AUC of 0.746 (95% CI: 0.666-0.826, $P < 0.001$) and a cut-off value of 1.623, and CHG index with an AUC of 0.659 (95% CI: 0.573-0.746, $P < 0.001$) and a cut-off value of 2.574 (Table 7 and Fig. 1).

Table 6. Logistic regression model including TG/HDL-C in NSTE-ACS patients

Variable	univariate-OR (95%CI)	uni-*P*	Multi-OR (95%CI)	multi-*P*	VIF
LDL-C (mmol/L)	1.530 (1.066-2.197)	0.021	1.641(0.970-2.777)	0.065	1.151
WBC (×10^9/L)	0.513 (0.334-2.554)	0.510			
MONO (×10^9/L)	11.168 (3.653-96.249)	<0.001	18.681(3.750-61.951)	0.003	1.472
TG/HDL-C	2.995 (1.847-4.858)	<0.001	3.562(1.868-6.793)	<0.001	1.074
Systolic pressure (mmHg)	1.006 (0.982-1.030)	0.635			
Diastolic pressure (mmHg)	1.017 (0.990-1.045)	0.225			
BMI (kg/m^2)	1.065 (0.990-1.145)	0.092			
NEUT (×10^9/L)	1.630 (1.306-2.034)	<0.001	1.093(0.811-1.475)	0.558	1.505
FBG (mmol/L)	1.526 (1.220-1.908)	<0.001	1.458(1.093-1.945)	0.010	1.113
Gender	1.919 (0.937-3.932)	0.075			
Age	0.989 (0.963-1.015)	0.410			
TC (mmol/L)	1.912 (1.425-2.565)	<0.001	1.693(1.162-2.468)	0.006	1.183
Smoking	2.047 (1.030-4.066)	0.041	2.128(0.795-5.698)	0.133	1.025
Hypertension	0.847 (0.441-1.626)	0.618			

BMI, body mass index; CI, confidence interval; FBG, fasting blood glucose; LDL-C, low-density lipoprotein cholesterol; MONO, monocyte count; NEUT, neutrophil count; NSTE-ACS, non-ST-segment elevation acute coronary syndrome; OR, odds ratio; TC, total cholesterol; TG/HDL-C, triglyceride-to-high-density lipoprotein cholesterol ratio; VIF, variance inflation factor; WBC, white blood cell count.

Table 7. ROC curve parameters

Variable	AUC	95% CI	Youden	Cut-off	Sensitivity	Specificity	*P*
MHR	0.803	0.730-0.876	0.587	0.504	81.3%	77.3%	<0.001
TG/HDL-C	0.746	0.666-0.826	0.480	1.623	72.0%	76.0%	<0.001
CHG index	0.659	0.573-0.746	0.280	2.574	70.7%	57.3%	<0.001
CHG + TG/HDL-C	0.821	0.755-0.887	0.533	0.379	84.0%	69.3%	<0.001
CHG + MHR	0.872	0.815-0.929	0.613	0.349	92.0%	69.3%	<0.001

AUC, area under the ROC curve; CHG index, cholesterol, high-density lipoprotein, and glucose index; CHG + MHR, combination of CHG index and MHR; CHG + TG/HDL-C, combination of CHG index and TG/HDL-C; CI, confidence interval; MHR, monocyte-to-high-density lipoprotein cholesterol ratio; ROC, receiver operating characteristic; TG/HDL-C, triglyceride-to-high-density lipoprotein cholesterol ratio.

Discussion

NSTE-ACS is a serious disease that endangers life and health and has a poor prognosis. It is an important problem that needs to be urgently addressed in the medical field. Hence, we must find accurate and consistent criteria for patients with NSTE-ACS.

Emerging evidence highlights CHG as a multiparameter index that integrates glycolipid metabolism to comprehensively reflect the pathophysiological mechanisms underlying coronary artery disease. Mo *et al*.[13] demonstrated through the CHARLS database study involving thousands of middle-aged and elderly participants that each 1-unit increase in CHG index was associated with a 35% elevated risk of coronary heart disease, suggesting that the CHG index may serve as a useful predictor of cardiovascular risk.

Furthermore, MONOs evolve into macrophages and further impact the pathogenesis of ACS by infiltrating the vascular endothelium and promoting the inflammatory response and the formation and progression of plaques.[6] Although HDL-C typically exerts protective effects by suppressing MONO activation and facilitating reverse cholesterol transport, the chronic inflammatory state associated with ACS may impair HDL-C functionality, thereby diminishing its antiatherogenic capacity.

The novel inflammatory marker MHR integrates both inflammatory and lipid metabolic pathways, captures the balance between MONO-driven inflammation and HDL-C-mediated protection, and accurately reflects the level of systemic inflammation.[15]

Previous studies demonstrated significant positive correlations between MHR and Gensini scores.[15] Furthermore, patients with elevated MHR demonstrated a significantly higher incidence of major adverse cardiovascular events, including recurrent myocardial infarction and mortality, during both short- and long-term follow-up.[15,16] Multivariable regression analysis confirmed

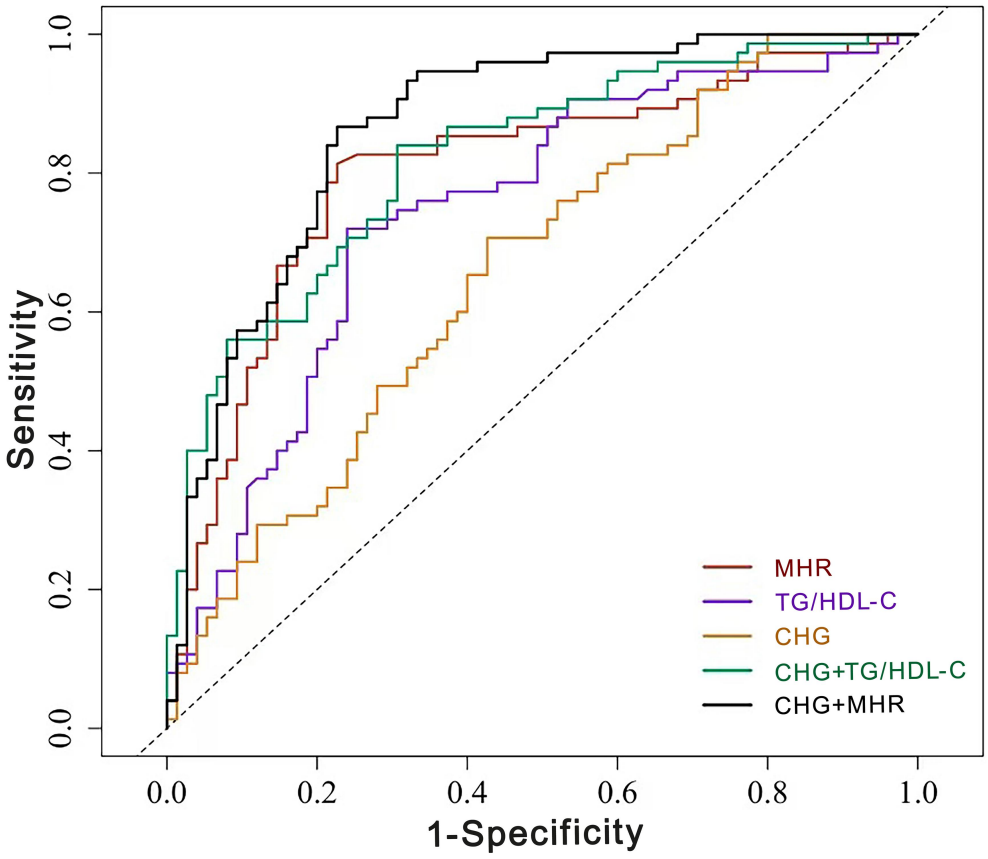

Fig. 1. The AUCs of MHR, TG/HDL-C, CHG index, CHG + TG/HDL-C, CHG + MHR. AUC, area under the ROC curve; CHG + MHR, the combination of CHG and MHR; CHG + TG/HDL-C, the combination of CHG and TG/HDL-C; CHG index, cholesterol, high-density lipoprotein, and glucose index; MHR, monocyte-to-high-density lipoprotein cholesterol ratio; TG/HDL-C, triglyceride-to-high-density lipoprotein.

MHR as an independent predictor of adverse cardiovascular events.[15] Thus, MHR can serve as a simple, inexpensive tool for enhancing risk stratification in ACS.

Previous studies have shown that TG/HDL-C can be regarded as a powerful index for atherosclerosis and insulin resistance,[17,18] and its positive correlation with the Gensini score in patients with coronary heart disease supports its use for assessing severe coronary artery lesions.[19]

As presented in our results, patients in the high-risk group exhibited significantly elevated levels of MHR, TG/HDL-C, and CHG index compared with those in the low-risk group. Correlation analysis revealed positive associations between all three biomarkers and Gensini scores, indicating that higher levels of MHR, TG/HDL-C, and CHG index are consistent with the severity of coronary artery lesions. We also found that MHR, TG/HDL-C, and CHG index were associated with severe coronary artery stenosis in NSTE-ACS patients, which was also in line with previous research findings. It should be noted that the large differences between groups naturally led to large ORs in the logistic regression analysis. For example, the OR for MHR was 16.051 (95% CI: 13.677–99.650), which, despite its wide CI, reflects the strong association with high-risk coronary lesions. The wide CI is likely due to the relatively small sample size and the skewed distribution of the data confirming the stability of the association.

Additionally, ROC analysis further evaluated the efficacy of MHR, TG/HDL-C, CHG index, and their combinations for assessing coronary artery severity, demonstrating that the combination of MHR and CHG index yielded the numerically higher AUC of 0.872, and the AUC of the combined CHG index with TG/HDL-C was 0.821. Among the individual indicators, MHR had an AUC of 0.803, TG/HDL-C had an AUC of 0.746, and CHG index had an AUC of 0.659. It is notable that while CHG index was identified as an independent risk factor, its individual predictive value for severe coronary artery lesions was lower. This relatively weak discriminatory capacity as an independent marker may be attributed to its composition, which reflects metabolic status. However, it does not directly incorporate specific inflammatory mediators, such as MONOs, which can contribute to the instability of atherosclerotic plaques in NSTE-ACS. Thus, we suspected that inflammatory responses have a more intense acute-phase reaction than metabolic disorders. In our combined analysis, CHG with these more dynamic markers provided a more comprehensive assessment and showed

numerically higher AUCs. These findings indicated that combining CHG index with either MHR or TG/HDL-C showed numerically higher discriminatory capacity for predicting coronary heart disease severity than using any of these indicators alone.

Our findings extend previous research in several ways. While Mao *et al*.[20] focused on the triglyceride-glucose index, referring to TG and FBG in NSTE-ACS, CHG index offers a broader metabolic perspective by including TC and HDL-C. More importantly, we demonstrated the added value of pairing this metabolic index with inflammatory markers. Compared with imaging-based approaches, such as layer-specific strain echocardiography used by Zhang *et al.*[21] to predict complex coronary artery disease, our method offers a complementary, economical, and easily accessible alternative using routine blood parameters. The predictive performance of our combined models is comparable to these imaging techniques, highlighting the utility of blood-based biomarkers. Furthermore, while Eggers *et al.*[22] identified NT-proBNP as a dominant prognostic marker reflecting cardiac stress, our study explored the critical roles of inflammatory and metabolic pathways, suggesting that a multidimensional biomarker approach may provide the most comprehensive assessment.

Notably, FBG, TG, TC, and HDL-C are all easily accessible clinical parameters. Compared with invasive coronary angiography, they offer the advantages of simple calculation and low cost. If routinely applied in the clinical management of NSTE-ACS patients, these markers may help further identify individuals at high risk of complications and support early risk stratification and management of NSTE-ACS.

Limitations

Several limitations warrant consideration. First, the exclusion of patients with diabetes, while intended to avoid confounding by glucose-lowering medications, limits the generalizability of our findings to this substantial patient subgroup. Second, this was an exploratory, single-center study with a relatively modest sample size and no formal pre-experimental sample size calculation. Although consecutive patients were enrolled, the potential for selection bias exists, and the findings require validation in larger, more diverse populations. Third, despite multivariate adjustment, residual confounding from unmeasured variables such as medication use, disease duration, and lifestyle factors cannot be ruled out. Fourth, the lack of external validation is a significant constraint. Our results should be confirmed in independent multicenter cohorts. Fifth, we did not compare all possible combinations, as our primary aim was to compare CHG combined with MHR or CHG combined with TG/HDL-C against each single marker. The optimal biomarker panel should be determined in future large-scale studies. Sixth, we did not calculate formal inter-rater agreement statistics for Gensini scoring, as discrepancies were resolved by consensus. We acknowledge this methodological limitation, and future studies should report such metrics to improve scoring repeatability. Seventh, we did not perform cross-validation or calibration analyses due to the exploratory design and modest sample size. Our estimation of AUC may be overly optimistic. Future multicenter studies should include assessments of discrimination and calibration to validate our findings. Finally, the Gensini score, while widely used, is a semi-quantitative tool, and the median cut-off value of 51 derived from this specific cohort may not be universally applicable.

Future directions

In the future, a large-scale, multicenter prospective study is needed to assess generalizability through external validation. Such a study should fully consider all potential confounding factors that may affect MHR, TG/HDL-C, and CHG index to further validate the value of these indicators in predicting the severity of coronary stenosis.

Conclusions

Elevated MHR, TG/HDL-C, and CHG index correlate with coronary lesion severity in NSTE-ACS patients and were independently associated with high-risk coronary lesions, while their combined measurement showed numerically higher AUCs.

Supporting information

Supplementary material for this article is available at https://doi.org/10.14218/ERHM.2026.00005.

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Conflict of interest

The authors declare that there are no conflicts of interest.

Author contributions

Data collection, research analysis (JY, RC, XL), statistical analysis (JY, YL), supervision (PL, JH), and writing of the manuscript (JY, PL). All authors conducted the revision of the manuscript.

Ethical statement

The research received approval from the Ethics Committee of the Affiliated Hospital of Nanjing University of Chinese Medicine (Approval No. 2021NL-073-02) in accordance with the principles of the Declaration of Helsinki (as revised in 2024), and written informed consent was obtained from all participants prior to enrollment.

Data sharing statement

The data used to support the findings of this study are available

from the corresponding author at the email address (lipengfei@njucm.edu.cn) upon request.

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