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## **Predictive value of GNRI and TyG index for poor prognosis in NSTEMI-ACS patients post-PCI**

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## ABSTRACT

**Background and Objectives:** This study aimed to assess the predictive power of the Geriatric Nutritional Risk Index (GNRI) and the triglyceride-glucose (TyG) index for poor prognosis in non-ST segment elevation acute coronary syndrome (NSTE-ACS) patients' post-percutaneous coronary intervention (PCI). **Methods and Study Design:** A cohort of 393 NSTE-ACS patients who underwent PCI at the People's Hospital of Nanjing Jiangbei from 2016 to 2022 was analyzed. Major adverse cardiovascular events (MACEs), including death, non-fatal myocardial infarction, and target vessel revascularization, served as the primary outcome. Relationships between GNRI, TyG index, and MACEs were explored using univariate and multivariate logistic regression, with results presented as odds ratios (OR) and 95% confidence intervals (CI). The predictive value was further evaluated using the area under the curve (AUC) from the receiver operating characteristic (ROC) curve. **Results:** MACEs occurred in 34 patients. A TyG index  $\geq 1.359$  was associated with a significantly increased risk of MACEs (OR=5.07, 95%CI: 1.64-15.71), while a GNRI  $\geq 107.514$  indicated a decreased risk (OR=0.17, 95%CI: 0.04-0.68). These associations were consistent across various subgroups, including age, gender, and specific pre-existing conditions. The combined predictive value of TyG index and GNRI was higher than each alone (AUC=0.711, 95%CI: 0.642-0.779). **Conclusions:** In patients with NSTE-ACS after PCI, the TyG index and GNRI are significant predictors of MACEs, with the TyG index indicating higher risk and GNRI lower risk. Their combined use may enhance the predictive accuracy for MACEs in this patient population.

**Key Words:** Non-ST segment elevation myocardial infarction, percutaneous coronary intervention, major adverse cardiovascular and cerebrovascular events, Geriatric Nutritional Risk Index, triglyceride-glucose index

## INTRODUCTION

Non-ST segment elevation acute coronary syndrome (NSTE-ACS), encompassing unstable angina and non-ST elevation myocardial infarction (NSTEMI), represents a significant cause of morbidity and mortality worldwide.<sup>1</sup> The global cardiovascular disease burden has shifted to low and middle-income countries, accounting for more than 80% of cardiovascular deaths worldwide.<sup>2</sup> Despite the rapid development and extensive use of Percutaneous Coronary Intervention (PCI),<sup>3</sup> identifying patients at higher risk of poor prognosis, including major adverse cardiovascular events (MACEs), remains a clinical challenge.

The triglyceride-glucose (TyG) index and the Geriatric Nutritional Risk Index (GNRI) have emerged as potential prognostic markers in cardiovascular disease. TyG index, derived from fasting triglyceride (TG) and glucose levels, serves as a marker of insulin resistance and metabolic dysfunction. Elevated TyG index has been linked to heightened risks of coronary artery disease (CAD) and MACEs. Mao et al.<sup>4</sup> reported that the TyG index was closely related to the complexity of coronary lesions and the incidence of MACEs in a 12-month follow-up period of patients with NSTEMI-ACS. Luo et al. reported an elevated TyG index may be an indicator of poor prognosis in patients with ST elevation myocardial infarction (STEMI) after PCI.<sup>5</sup> Elevated TyG index also be found as an indicator of poor prognosis of patients with NSTEMI-ACS, and combined with other indicators could significantly improve risk prediction capabilities.<sup>6,7</sup>

The GNRI, initially developed to assess nutritional risk in elderly individuals, integrates serum albumin levels and body weights, reflecting both nutritional status and inflammatory burden.<sup>8</sup> Low GNRI scores have been associated with adverse clinical outcomes in several diseases.<sup>9,10</sup> Malnutrition is also common in patients with CAD. Basta et al. found nutritional status could affect the prognosis of elderly patients with ST-elevation myocardial infarction (MI), with almost 55% of the elderly being malnutrition.<sup>11</sup> GNRI was linked to the progression of cardiac rehabilitation in patients with heart failure, stroke, end-stage kidney disease, and cardiovascular surgery.<sup>9-14</sup> Lower GNRI was a significant predictive factor for adverse prognosis in patients with NSTEMI-ACS after PCI, and combined with other indicators also could improve risk prediction capabilities.<sup>15</sup>

However, there is no data regarding the predictive value of GNRI and TyG on clinical outcomes in patients with NSTEMI-ACS after PCI. Thus, our study aims to investigate the relationship between GNRI or TyG and MACEs on the poor prognosis in patients with NSTEMI-ACS after PCI.

## **MATERIALS AND METHODS**

### ***Study population***

Patients who have NSTEMI-ACS after PCI, visited Nanjing Jiangbei Hospital between January 2016 to January 2022, were enrolled in this retrospective cohort study. Participants were included of those having NSTEMI-ACS and underwent PCI. Patients would be excluded with meeting any of the following criteria: (1) patients with diagnosed or suspected type 1 diabetes, (2) impaired kidney function with estimated glomerular filtration rate  $<30 \text{ mL}/(\text{min}\cdot 1.73\text{m}^2)$  or receiving continuous renal replacement therapy, (3) severely impaired liver function with

alanine aminotransferase (ALT) or aspartate aminotransferase (AST) 5 times higher than the normal limit, (4) history of coronary artery bypass grafting (CABG), history of cardiogenic shock, chronic infectious disease, or malignant tumor, (5) PCI failure, complications, or death in hospital, (6) history of cerebrovascular disease and surgery, and (7) missing data on TG, fasting blood glucose (FBG), aspartate aminotransferase (ALB), height, and weight. Finally, a total of 393 eligible patients were included (Figure 1).

### ***Data collection***

Data were acquired according to medical records or computer tracking systems. The information collected included demographic information, disease history, complications, laboratory examinations, medication taken before and after admission, data on PCI, data on angiographic, and MACEs. Demographic information included age, gender, height, systolic blood pressure, diastolic blood pressure, pulse, heart rate, smoking, and drinking status. Disease history included family history of coronary heart disease (CHD), history of PCI/CABG, acute MI, and stroke. Complications information included type 2 diabetes mellitus, hyperlipemia, and CHD. Laboratory examinations were hemoglobin, red blood cell, white blood cell, neutrophil, monocyte, lymphocyte, platelet, platelet distribution width (PDW), red cell distribution width (RDW), creatine kinase isoenzymes, TG, total cholesterol (TC), low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, ALT, AST, ALB, FBG, hemoglobin A1c, serum creatinine, urinary acid, and fibrosis. Before and after admission, medication taken information was collected about angiotensin-converting enzyme inhibitors, angiotensin II receptor blockers, dual antiplatelet therapy, aspirin, clopidogrel, beta-blockers, statins, proton pump inhibitors, oral hypoglycemic drugs, and other drug.

### ***TyG and GNRI measurement***

TyG index =  $\ln$  [fasting TG (mg/dL) \* FBG (mg/dL)].<sup>16</sup>

GNRI =  $1.489 * \text{ALB (g/L)} + 41.7 * \text{present weight/ideal weight (kg)}$ .<sup>8,17</sup>

The ideal body weight was calculated based on the Lorentz equations:  $0.75 \times \text{height (cm)} - 62.5$  for men, and  $0.60 \times \text{height (cm)} - 40$  for women. When the present weight/ideal body weight was  $\geq 1$ , the ratio was set to 1. The cut-off values of TyG and GNRI were 1.359 and 107.514.

### ***Outcomes and follow-up***

The outcome measured was the incidence of MACE during follow-up, including all-cause mortality, non-fatal MI, and target vessel revascularization (TVR).<sup>18</sup> MI was characterized by typical chest pain, ST-segment deviation, T wave changes, and creatine kinase-myocardial band levels exceeding three times the normal upper limit.<sup>19</sup> TVR, involving interventions on both target and non-target vessels through PCI or CABG, was conducted for patients with severe in-stent restenosis or newly developed coronary lesions (luminal diameter narrowing  $\geq 70\%$ ).<sup>18</sup> Patients were followed up through telephone or outpatient visits at 3, 6, 12, 18, and 24 months after hospital discharge. For analysis, the initial MACE occurrence during the follow-up period was selected. In cases where patients experienced multiple adverse outcomes simultaneously, the most severe event was prioritized (all-cause mortality > non-fatal MI > target vessel revascularization).

### ***Statistical analysis***

Continuous variable was shown as mean  $\pm$  standard deviation (S.D) or median [interquartile range: 25th to 75th percentiles]. The normality of continuous variable was assessed using skewness and kurtosis, while homogeneity was detected by the Levene test. The comparison between the two groups was performed according to Student's t-tests and Satterthwaite t-tests for normality distribution, with Wilcoxon rank sum tests for non-normality distribution. Categorical variable was reported as frequencies and percentages (%), Chi-square tests or Fisher exact tests were utilized to compare differences between two groups. Potential covariates were screened by univariate logistic regression model and two-way stepwise method. The relationships of TyG, GNRI, with MACE was explored using univariate and multivariate logistic regression models. Odds ratios (ORs) and 95% confidence intervals (CIs) were utilized to present results. Maximally selected test statistics were employed to determine the cut-off value of TyG and GNRI. The area under the curve (AUC) was performed to compare the predictive value of TyG, GNRI, and combined indexes. The Delong test was also performed. Restrict cubic spline (RCS) was used to show the non-linear relationship of TyG, GNRI, with MACE. The associations of TyG, GNRI, with MACE were further explored in different ages, genders, pre-diffuse lesion, pre-bifurcation lesion, pre-in stent restenosis, and pre-complete revascularization subgroups. All analyses were employed using Python 3.9.12 and R version 4.3.1. A *p* value <0.05 was considered statistically difference.

### ***Ethics statement***

The study was approved by the Ethics Committee of the People's Hospital of Nanjing Jiangbei (Approval number: 2021031). All patients provided an informed consent before the study.

## **RESULTS**

### ***Characteristics of patients with NSTEMI-ACS***

In total, 393 patients were enrolled in our study. Among them, the mean age was 64.70 (10.17) years, and 245 (62.34%) were males. The mean scores of TyG and GNRI were 1.58 (0.76) and 102.54 (6.11) respectively. Statistically significant between MACE and no-MACE groups were observed in PDW, RDW, TG, TC, pre-single vessel disease, pre-triple vessel disease, pre-diffuse lesion, pre-bifurcation lesion, pre-in stent restenosis, pre-complete revascularization, pre-number of supports, and TyG (all  $p < 0.05$ ). More characteristics of patients with NSTEMI-ACS were illustrated in Table 1.

### ***Associations between TyG, GNRI, and MACE in patients with NSTEMI-ACS***

The relationships between TyG, GNRI, and MACE were shown in Table 2. In model 2, we adjusted PDW, TC, pre-diffuse lesion, pre-bifurcation lesion, pre-in stent restenosis, and pre-complete revascularizations. TyG  $\geq 1.359$  was related to elevated odds of MACE (OR=5.07, 95%CI: 1.64-15.71) in patients with NSTEMI-ACS. While GNRI  $\geq 107.514$  was linked to increased incidence of MACE (OR=5.07, 95%CI: 1.64-15.71). Restricted cubic splines suggested non-linear relationships between TyG or GNRI and the incidence of MACE (Figure S1). A positive trend was found in the incidence of MACE as RCS increased. No obvious trend was observed in the occurrence of MACE as GNRI increased. Supplementary Figure 2 illustrates the distribution of MACE in different TyG and GNRI levels. In patients with different TyG levels, there was a statistical difference between the no-MACE and MACE groups ( $p < 0.05$ ).

### ***Associations between TyG, GNRI, and MACE in different subgroups***

The associations of TyG, GNRI, with MACE were also investigated in different ages, gender, pre-diffuse lesion, pre-bifurcation lesion, pre-in stent restenosis, and pre-complete revascularization subgroups (Figure 2 and Supplementary Table 1). Compared to patients with TyG  $< 1.359$ , higher TyG was linked to increased incidence of MACE in those aged  $\geq 65$  years (OR=6.44, 95%CI: 1.59-44.47), males (OR=4.94, 95%CI: 1.38-24.68), pre-diffuse

lesion (OR=4.06, 95%CI: 1.02-16.16), without pre-bifurcation lesion (OR=7.73, 95%CI: 1.89-55.49), without pre-in stent restenosis (OR=5.32, 95%CI: 1.73-23.31), and without pre-complete revascularization (OR=5.32, 95%CI: 1.56-25.24). When compared to patients with GNRI <107.514, GNRI  $\geq$ 107.514 was related to decreased incidence of MACE in those age  $\geq$ 65 years (OR=0.08, 95%CI: 0.00-0.59), males (OR=0.06, 95%CI: 0.00-0.36), without pre-bifurcation lesion (OR=0.15, 95%CI: 0.02-0.65), without pre-in stent restenosis (OR=0.21, 95%CI: 0.04-0.75), and without pre-complete revascularization (OR=0.08, 95%CI: 0.00-0.55).

### ***ROC curves of TyG, GNRI, and combination of two indexes***

We use TyG, GNRI, and the combination of TyG and GNRI to predict MACE in Figure 3. ROC curves show that the AUC of TyG, GNRI, and combined were 0.665 (95%CI:0.605-0.726), 0.559 (95%CI:0.506-0.612), and 0.711 (95%CI:0.642-0.779) respectively. Among the three indicators, the combination indicator had the highest AUC of 0.711 (compared with TyG alone and GNRI alone, both  $p < 0.01$ , DeLong test). The result indicates that a combination of TyG and GNRI can increase the predictive value of MACE.

## **DISCUSSION**

TyG combined with GNRI may be a valuable predictive indicator for MACE in patients with NSTEMI-ACS after PCI. We found the cut-off values were 1.359 and 107.514 for TyG and GNRI, respectively. The predictive value of TyG combined GNRI was superior to that of these indicators alone. By calculating these indicators in NSTEMI-ACS patients, we can identify those at high risk of MACE, and provide a basis for early intervention to improve prognosis.

Our results suggested the associations of increased incidence of MACE with elevated TyG or lower GNRI in patients with NSTEMI-ACS after PCI. A meta-analysis reported the association of increased TyG index with higher incidence of MACE in patients who underwent PCI.<sup>20</sup> The TyG index is an independent predictor of CAD severity and MACEs.<sup>4</sup> The increased risk of MACE in patients with elevated TyG index levels is likely due to insulin resistance (IR).<sup>21, 22</sup> IR and glucose metabolism disturbances lead to oxidative stress, inflammation, and impaired immune regulation, which accelerate arteriosclerosis and promoting plaque formation. The violin plot shows the TyG scores in the MACE group were higher than those in the non-MACE group, which further validates our findings. Similarly, the predictive value of GNRI on all-cause mortality and MACE in patients with CAD has been reported.<sup>23, 24</sup> Malnutrition, evaluated by the GNRI score upon admission, independently

predicted MACE in chronic artery occlusion patients after PCI.<sup>25</sup> The GNRI, which combines serum albumin and body weight, is frequently utilized to assess the nutritional status of hospitalized elderly patients. Malnutrition was related to all-cause mortality and MACE.<sup>26</sup> Notably, our analysis using restricted cubic splines indicated non-linear relationships between TyG or GNRI and the occurrence of MACEs. Specifically, there was a discernible positive trend in MACE incidence with increasing TyG values, suggesting a potential threshold effect beyond  $TyG \geq 1.359$ . While the relationship between GNRI and MACE incidence did not display a clear trend as GNRI values increased.

The important finding of our study was the predictive value of TyG combined with GNRI exceeds that of TyG or GNRI alone in MACE in patients with NSTEMI-ACS after PCI. The superior predictive value of GNRI and TyG index for MACE in patients with NSTEMI-ACS after PCI likely stems from their complementary roles in different aspects of metabolic and nutritional status. TyG index reflects IR and metabolic dysfunction, both of which contribute significantly to the pathogenesis of cardiovascular diseases.<sup>4, 27</sup> Elevated TyG levels have been linked to increased arterial stiffness, endothelial dysfunction, and systemic inflammation, all of which are fundamental in the progression of atherosclerosis and subsequent adverse cardiovascular events.<sup>28, 29</sup> In addition, GNRI assesses nutritional status and overall health resilience, factors that influence post-PCI recovery and outcomes. Poor nutritional status is associated with impaired immune function, inflammation, and increased susceptibility to infections, all of which may exacerbate the incidence of MACE.<sup>30, 31</sup> By combining TyG and GNRI, clinicians gain a more comprehensive assessment of metabolic health, nutritional status, and systemic inflammation, which collectively enhance risk stratification and guide targeted interventions to reduce cardiovascular risk factors.

The combination of TyG and GNRI likely enhances predictive accuracy by capturing diverse aspects of patient vulnerability. TyG emphasizes metabolic dysregulation and underlying insulin resistance, while GNRI underscores the broader health status and nutritional adequacy critical for recovery and prognosis after PCI. Integrating these indicators yields a more comprehensive risk profile, enabling clinicians to identify high-risk patients who could benefit from intensified monitoring or targeted interventions to mitigate adverse cardiovascular outcomes.

Several limitations warrant consideration in the current study. Firstly, due to its retrospective nature, despite adjusting for potential covariates, residual or unmeasured confounding may still exist. Secondly, limited information was available regarding changes in patients' TyG index and GNRI levels during follow-up, indicating a need for future research



to explore the dynamics. Lastly, as a single-center study, this research may have selection bias, necessitating further studies to validate our findings.

### **Conclusions**

Higher TyG and lower GNRI levels were associated with an elevated incidence of MACCE in patients with NSTEMI-ACS after PCI. The combination of these indicators may be a reliable, simple, cost-effective, and accessible method for predicting MACCE incidence in patients with NSTEMI-ACS after PCI. This helps cardiovascular specialists in patients' risk stratification and reduces the incidence of MACE.

### **CONFLICT OF INTEREST AND FUNDING DISCLOSURE**

The authors declare no conflict of interest.

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**Table 1.** Characteristics of patients with NSTEMI-ACS after PCI

Variables	Total (n=393)	No-MACE (n=359)	MACE (n=34)	<i>p</i>
Age, Mean ( $\pm$ SD)	64.70 ( $\pm$ 10.17)	64.56 ( $\pm$ 10.20)	66.24 ( $\pm$ 9.94)	0.359 <sup>a</sup>
Gender, n (%)				0.318 <sup>c</sup>
Female	148 (37.66)	132 (36.77)	16 (47.06)	
Male	245 (62.34)	227 (63.23)	18 (52.94)	
Height, Mean ( $\pm$ SD)	1.64 ( $\pm$ 0.08)	1.64 ( $\pm$ 0.08)	1.64 ( $\pm$ 0.08)	0.773 <sup>a</sup>
Weight, Mean ( $\pm$ SD)	67.72 ( $\pm$ 10.80)	67.51 ( $\pm$ 10.52)	69.96 ( $\pm$ 13.38)	0.206 <sup>a</sup>
SBP, Mean ( $\pm$ SD)	139.20 ( $\pm$ 18.70)	139.01 ( $\pm$ 18.10)	141.24 ( $\pm$ 24.39)	0.607 <sup>b</sup>
DBP, Mean ( $\pm$ SD)	81.04 ( $\pm$ 11.38)	81.20 ( $\pm$ 11.39)	79.35 ( $\pm$ 11.32)	0.365 <sup>a</sup>
Pulse, Mean ( $\pm$ SD)	72.17 ( $\pm$ 13.42)	72.14 ( $\pm$ 13.60)	72.44 ( $\pm$ 11.61)	0.901 <sup>a</sup>
Heart rate, Mean ( $\pm$ SD)	72.00 ( $\pm$ 13.26)	71.96 ( $\pm$ 13.42)	72.44 ( $\pm$ 11.61)	0.840 <sup>a</sup>
NSTEMI-ACS, n (%)				0.922 <sup>c</sup>
NSTEMI	101 (25.7)	93 (25.91)	8 (23.53)	
UA	292 (74.3)	266 (74.09)	26 (76.47)	
Killip level, n (%)				1.000 <sup>c</sup>
I & II & III	65 (16.54)	59 (16.43)	6 (17.65)	
Unknown	328 (83.46)	300 (83.57)	28 (82.35)	
LVEF, Mean ( $\pm$ SD)	59.50 ( $\pm$ 6.02)	59.34 ( $\pm$ 6.19)	61.17 ( $\pm$ 3.25)	0.089 <sup>a</sup>
Smoking, n (%)				0.988 <sup>c</sup>
No	249 (63.36)	228 (63.51)	21 (61.76)	
Yes	144 (36.64)	131 (36.49)	13 (38.24)	
Drinking, n (%)				0.674 <sup>c</sup>
No	317 (80.66)	291 (81.06)	26 (76.47)	
Yes	76 (19.34)	68 (18.94)	8 (23.53)	
Family history CHD, n (%)				1.000 <sup>e</sup>
No	382 (97.2)	349 (97.21)	33 (97.06)	
Yes	11 (2.8)	10 (2.79)	1 (2.94)	
History PCI/CABG, n (%)				0.059 <sup>e</sup>
No	343 (87.28)	317 (88.3)	26 (76.47)	
Yes	50 (12.72)	42 (11.7)	8 (23.53)	
History AMI, n (%)				0.275 <sup>e</sup>
No	366 (93.13)	336 (93.59)	30 (88.24)	
Yes	27 (6.87)	23 (6.41)	4 (11.76)	
History stroke, n (%)				0.950 <sup>c</sup>
No	293 (74.55)	267 (74.37)	26 (76.47)	
Yes	100 (25.45)	92 (25.63)	8 (23.53)	
Type-II diabetes, n (%)				0.978 <sup>c</sup>
No	284 (72.26)	260 (72.42)	24 (70.59)	
Yes	109 (27.74)	99 (27.58)	10 (29.41)	
Hyperlipemia, n (%)				0.379 <sup>e</sup>
No	378 (96.18)	346 (96.38)	32 (94.12)	
Yes	15 (3.82)	13 (3.62)	2 (5.88)	
CHD, n (%)				0.518 <sup>e</sup>
No	8 (2.04)	7 (1.95)	1 (2.94)	
Yes	385 (97.96)	352 (98.05)	33 (97.06)	
Pre-ACEI, n (%)				1.000 <sup>e</sup>
No	374 (95.17)	341 (94.99)	33 (97.06)	
Yes	19 (4.83)	18 (5.01)	1 (2.94)	
Pre-ARB, n (%)				0.993 <sup>c</sup>
No	295 (75.06)	270 (75.21)	25 (73.53)	
Yes	98 (24.94)	89 (24.79)	9 (26.47)	
Pre-DAPT, n (%)				1.000 <sup>e</sup>
No	360 (91.6)	329 (91.64)	31 (91.18)	
Yes	33 (8.4)	30 (8.36)	3 (8.82)	

SD: standard deviation; M: median; Q<sub>1</sub>: 1st quartile; Q<sub>3</sub>: 3rd quartile, PCI: percutaneous coronary intervention, SBP: systolic blood pressure, DBP: diastolic blood pressure, NSTEMI-ACS: non-ST elevation acute coronary syndromes, NSTEMI: non-ST-elevation myocardial infarction, UA: unstable angina, LVEF: left ventricular ejection fraction, CHD: coronary heart disease, CABG: coronary artery bypass grafting, AMI: acute myocardial infarction, ACEI: angiotensin-converting enzyme inhibitor, ARB: angiotensin II receptor blockers, DAPT: dual antiplatelet therapy, RBC: red blood cell count, WBC: white blood cell count, PDW: platelet distribution width, RDW: red cell distribution width, CK-MB: creatine kinase isoenzymes, TG: triglyceride, TC: total cholesterol, LDL-C: low density lipoprotein cholesterol, HDL-C: high density lipoprotein cholesterol, ALT: alanine aminotransferase, AST: aspartate aminotransferase, ALB: albumin, FBG: fasting blood glucose, HbA<sub>1c</sub>: hemoglobin A<sub>1c</sub>, Scr: serum creatinine, FIB: fibrosis, TyG: triglyceride-glucose index, GNRI: Geriatric Nutritional Risk Index, MACE: major adverse cardiac event.

<sup>a</sup> student's t test; <sup>b</sup> Satterthwaite t test; <sup>c</sup> Chi-square test; <sup>d</sup> Wilcoxon rank sum test; <sup>e</sup> Fisher's exact test.

**Table 1.** Characteristics of patients with NSTEMI-ACS after PCI (cont.)

Variables	Total (n=393)	No-MACE (n=359)	MACE (n=34)	<i>p</i>
Pre-aspirin, n (%)				0.794 <sup>e</sup>
No	340 (86.51)	311 (86.63)	29 (85.29)	
Yes	53 (13.49)	48 (13.37)	5 (14.71)	
Pre-clopidogrel, n (%)				1.000 <sup>e</sup>
No	390 (99.24)	356 (99.16)	34 (100)	
Yes	3 (0.76)	3 (0.84)	0 (0)	
Pre-beta blocker, n (%)				0.599 <sup>e</sup>
No	340 (86.51)	309 (86.07)	31 (91.18)	
Yes	53 (13.49)	50 (13.93)	3 (8.82)	
Pre-statins, n (%)				0.996 <sup>c</sup>
No	318 (80.92)	291 (81.06)	27 (79.41)	
Yes	75 (19.08)	68 (18.94)	7 (20.59)	
Pre-proton pump inhibitor, n (%)				0.312 <sup>e</sup>
No	380 (96.69)	348 (96.94)	32 (94.12)	
Yes	13 (3.31)	11 (3.06)	2 (5.88)	
Pre-oral hypoglycemic drug, n (%)				0.858 <sup>c</sup>
No	311 (79.13)	285 (79.39)	26 (76.47)	
Yes	82 (20.87)	74 (20.61)	8 (23.53)	
Pre-other drug, n (%)				0.810 <sup>c</sup>
No	233 (59.29)	214 (59.61)	19 (55.88)	
Yes	160 (40.71)	145 (40.39)	15 (44.12)	
Hemoglobin, M (Q <sub>1</sub> , Q <sub>3</sub> )	134 (122-144)	134 (122-144)	131.5 (121-146.75)	0.959 <sup>d</sup>
RBC, M (Q <sub>1</sub> , Q <sub>3</sub> )	4.43 (4.06-4.78)	4.42 (4.06-4.78)	4.44 (4.06-4.78)	0.911 <sup>d</sup>
WBC, M (Q <sub>1</sub> , Q <sub>3</sub> )	6.3 (5.2-7.7)	6.28 (5.2-7.75)	6.65 (5.73-7.58)	0.419 <sup>d</sup>
Neutrophil, M (Q <sub>1</sub> , Q <sub>3</sub> )	4.17 (3.31-5.31)	4.16 (3.28-5.38)	4.61 (3.65-5.07)	0.495 <sup>d</sup>
Monocyte, M (Q <sub>1</sub> , Q <sub>3</sub> )	0.38 (0.31-0.49)	0.38 (0.31-0.5)	0.38 (0.32-0.45)	0.895 <sup>d</sup>
Lymphocyte, M (Q <sub>1</sub> , Q <sub>3</sub> )	1.49 (1.17-1.85)	1.48 (1.12-1.86)	1.52 (1.27-1.79)	0.463 <sup>d</sup>
Platelet, Mean (±SD)	183.01 (±54.19)	182.39 (±54.26)	189.65 (±53.81)	0.456 <sup>a</sup>
PDW, Mean (±SD)	28.20 (±18.59)	27.34 (±18.22)	37.34 (±20.20)	0.003 <sup>a</sup>
RDW, Mean (±SD)	31.32 (±14.26)	31.93 (±14.13)	24.88 (±14.28)	0.006 <sup>a</sup>
CK-MB, M (Q <sub>1</sub> , Q <sub>3</sub> )	10 (2.95-14)	10 (2.78-14)	10 (4.12-14)	0.473 <sup>d</sup>
TG, M (Q <sub>1</sub> , Q <sub>3</sub> )	1.51 (1.01-2.17)	1.41 (0.97-2.1)	2 (1.61-3.02)	<0.001 <sup>d</sup>
TC, Mean (±SD)	4.61 (±1.26)	4.55 (±1.25)	5.18 (±1.14)	0.005 <sup>a</sup>
LDL-C, Mean (±SD)	2.62 (±0.95)	2.61 (±0.96)	2.77 (±0.84)	0.331 <sup>a</sup>
HDL-C, M (Q <sub>1</sub> , Q <sub>3</sub> )	1.08 (0.92-1.26)	1.07 (0.92-1.26)	1.1 (1-1.31)	0.282 <sup>d</sup>
ALT, M (Q <sub>1</sub> , Q <sub>3</sub> )	19 (14-29)	19 (14-29)	21 (13-34)	0.520 <sup>d</sup>
AST, M (Q <sub>1</sub> , Q <sub>3</sub> )	21 (17-29)	21 (17-28.5)	23.5 (17-33.75)	0.456 <sup>d</sup>
ALB, Mean (±SD)	41.21 (±3.82)	41.27 (±3.83)	40.59 (±3.60)	0.323 <sup>a</sup>
FBG, M (Q <sub>1</sub> , Q <sub>3</sub> )	5.73 (5.03-7.45)	5.73 (5.01-7.41)	5.77 (5.19-7.61)	0.468 <sup>d</sup>
HbA1c, Mean (±SD)	6.41 (±1.36)	6.40 (±1.38)	6.56 (±1.12)	0.499 <sup>a</sup>
SCr, Mean (±SD)	73.73 (±17.68)	73.49 (±17.60)	76.26 (±18.64)	0.382 <sup>a</sup>
Uric acid, Mean (±SD)	334.31 (±97.20)	333.51 (±96.85)	342.74 (±101.96)	0.598 <sup>a</sup>
FIB, Mean (±SD)	3.30 (±0.90)	3.28 (±0.91)	3.54 (±0.83)	0.110 <sup>a</sup>
Pre-left main disease, n (%)				1.000 <sup>e</sup>
No	369 (93.89)	337 (93.87)	32 (94.12)	
Yes	24 (6.11)	22 (6.13)	2 (5.88)	
Pre-single vessel disease, n (%)				0.015 <sup>c</sup>
No	268 (68.19)	238 (66.3)	30 (88.24)	
Yes	125 (31.81)	121 (33.7)	4 (11.76)	
Pre-bivessel disease, n (%)				0.878 <sup>c</sup>
No	267 (67.94)	243 (67.69)	24 (70.59)	
Yes	126 (32.06)	116 (32.31)	10 (29.41)	

SD: standard deviation; M: median; Q<sub>1</sub>: 1st quartile; Q<sub>3</sub>: 3rd quartile, PCI: percutaneous coronary intervention, SBP: systolic blood pressure, DBP: diastolic blood pressure, NSTEMI-ACS: non-ST elevation acute coronary syndromes, NSTEMI: non-ST-elevation myocardial infarction, UA: unstable angina, LVEF: left ventricular ejection fraction, CHD: coronary heart disease, CABG: coronary artery bypass grafting, AMI: acute myocardial infarction, ACEI: angiotensin-converting enzyme inhibitor, ARB: angiotensin II receptor blockers, DAPT: dual antiplatelet therapy, RBC: red blood cell count, WBC: white blood cell count, PDW: platelet distribution width, RDW: red cell distribution width, CK-MB: creatine kinase isoenzymes, TG: triglyceride, TC: total cholesterol, LDL-C: low density lipoprotein cholesterol, HDL-C: high density lipoprotein cholesterol, ALT: alanine aminotransferase, AST: aspartate aminotransferase, ALB: albumin, FBG: fasting blood glucose, HbA1c: hemoglobin A1c, Scr: serum creatinine, FIB: fibrosis, TyG: triglyceride-glucose index, GNRI: Geriatric Nutritional Risk Index, MACE: major adverse cardiac event.

<sup>a</sup> student's t test; <sup>b</sup> Satterthwaite t test; <sup>c</sup> Chi-square test; <sup>d</sup> Wilcoxon rank sum test; <sup>e</sup> Fisher's exact test.

**Table 1.** Characteristics of patients with NSTEMI-ACS after PCI (cont.)

Variables	Total (n=393)	No-MACE (n=359)	MACE (n=34)	<i>p</i>
Pre-triple vessel disease, n (%)				0.006 <sup>c</sup>
No	253 (64.38)	239 (66.57)	14 (41.18)	
Yes	140 (35.62)	120 (33.43)	20 (58.82)	
Pre-chronic complete occlusion, n (%)				0.160 <sup>e</sup>
No	346 (88.04)	319 (88.86)	27 (79.41)	
Yes	47 (11.96)	40 (11.14)	7 (20.59)	
Pre-diffuse lesion, n (%)				<0.001 <sup>c</sup>
No	277 (70.48)	265 (73.82)	12 (35.29)	
Yes	116 (29.52)	94 (26.18)	22 (64.71)	
Pre-bifurcation lesion, n (%)				0.046 <sup>c</sup>
No	293 (74.55)	273 (76.04)	20 (58.82)	
Yes	100 (25.45)	86 (23.96)	14 (41.18)	
Pre-in stent restenosis, n (%)				0.049 <sup>e</sup>
No	376 (95.67)	346 (96.38)	30 (88.24)	
Yes	17 (4.33)	13 (3.62)	4 (11.76)	
Pre-Drug eluting stent implantation, n (%)				0.518 <sup>e</sup>
No	8 (2.04)	7 (1.95)	1 (2.94)	
Yes	385 (97.96)	352 (98.05)	33 (97.06)	
Pre-use balloon, n (%)				1.000 <sup>e</sup>
No	10 (2.54)	10 (2.79)	0 (0)	
Yes	383 (97.46)	349 (97.21)	34 (100)	
Pre-complete revascularization, n (%)				0.010 <sup>c</sup>
No	200 (50.89)	175 (48.75)	25 (73.53)	
Yes	193 (49.11)	184 (51.25)	9 (26.47)	
Pre-number of supports, Mean (±SD)	1.33 (±0.59)	1.30 (±0.58)	1.59 (±0.66)	0.018 <sup>b</sup>
Post-ACEI, n (%)				0.403 <sup>e</sup>
No	347 (88.3)	315 (87.74)	32 (94.12)	
Yes	46 (11.7)	44 (12.26)	2 (5.88)	
Post-ARB, n (%)				0.245 <sup>c</sup>
No	205 (52.16)	191 (53.2)	14 (41.18)	
Yes	188 (47.84)	168 (46.8)	20 (58.82)	
Post-DAPT, n (%)				0.609 <sup>e</sup>
No	11 (2.8)	11 (3.06)	0 (0)	
Yes	382 (97.2)	348 (96.94)	34 (100)	
Post-aspirin, n (%)				1.000 <sup>e</sup>
No	391 (99.49)	357 (99.44)	34 (100)	
Yes	2 (0.51)	2 (0.56)	0 (0)	
Post-clopidogrel, n (%)				1.000 <sup>e</sup>
No	385 (97.96)	351 (97.77)	34 (100)	
Yes	8 (2.04)	8 (2.23)	0 (0)	
Post-beta blocker, n (%)				0.111 <sup>c</sup>
No	148 (37.66)	140 (39)	8 (23.53)	
Yes	245 (62.34)	219 (61)	26 (76.47)	
Post-statins, n (%)				0.609 <sup>e</sup>
No	11 (2.8)	11 (3.06)	0 (0)	
Yes	382 (97.2)	348 (96.94)	34 (100)	
Post-proton pump inhibitor, n (%)				0.987 <sup>c</sup>
No	248 (63.1)	226 (62.95)	22 (64.71)	
Yes	145 (36.9)	133 (37.05)	12 (35.29)	

SD: standard deviation; M: median; Q<sub>1</sub>: 1st quartile; Q<sub>3</sub>: 3rd quartile, PCI: percutaneous coronary intervention, SBP: systolic blood pressure, DBP: diastolic blood pressure, NSTEMI-ACS: non-ST elevation acute coronary syndromes, NSTEMI: non-ST-elevation myocardial infarction, UA: unstable angina, LVEF: left ventricular ejection fraction, CHD: coronary heart disease, CABG: coronary artery bypass grafting, AMI: acute myocardial infarction, ACEI: angiotensin-converting enzyme inhibitor, ARB: angiotensin II receptor blockers, DAPT: dual antiplatelet therapy, RBC: red blood cell count, WBC: white blood cell count, PDW: platelet distribution width, RDW: red cell distribution width, CK-MB: creatine kinase isoenzymes, TG: triglyceride, TC: total cholesterol, LDL-C: low density lipoprotein cholesterol, HDL-C: high density lipoprotein cholesterol, ALT: alanine aminotransferase, AST: aspartate aminotransferase, ALB: albumin, FBG: fasting blood glucose, HbA1c: hemoglobin A1c, Scr: serum creatinine, FIB: fibrosis, TyG: triglyceride-glucose index, GNRI: Geriatric Nutritional Risk Index, MACE: major adverse cardiac event.

<sup>a</sup> student's t test; <sup>b</sup> Satterthwaite t test; <sup>c</sup> Chi-square test; <sup>d</sup> Wilcoxon rank sum test; <sup>e</sup> Fisher's exact test.

**Table 1.** Characteristics of patients with NSTEMI-ACS after PCI (cont.)

Variables	Total (n=393)	No-MACE (n=359)	MACE (n=34)	<i>p</i>
Post-oral hypoglycemic drug, n (%)				1.000 <sup>c</sup>
No	300 (76.34)	274 (76.32)	26 (76.47)	
Yes	93 (23.66)	85 (23.68)	8 (23.53)	
Post-other drug, n (%)				0.610 <sup>e</sup>
No	57 (14.5)	51 (14.21)	6 (17.65)	
Yes	336 (85.5)	308 (85.79)	28 (82.35)	
TyG, Mean (±SD)	1.58 (±0.76)	1.55 (±0.75)	1.94 (±0.74)	0.004 <sup>a</sup>
GNRI, Mean (±SD)	102.54 (±6.11)	102.58 (±6.19)	102.07 (±5.32)	0.640 <sup>a</sup>
TyG, n (%)				<0.001 <sup>c</sup>
<1.359	165 (41.98)	161 (44.85)	4 (11.76)	
≥1.359	228 (58.02)	198 (55.15)	30 (88.24)	
GNRI, n (%)				0.153 <sup>c</sup>
<107.514	316 (80.41)	285 (79.39)	31 (91.18)	
≥107.514	77 (19.59)	74 (20.61)	3 (8.82)	

SD: standard deviation; M: median; Q<sub>1</sub>: 1st quartile; Q<sub>3</sub>: 3rd quartile, PCI: percutaneous coronary intervention, SBP: systolic blood pressure, DBP: diastolic blood pressure, NSTEMI-ACS: non-ST elevation acute coronary syndromes, NSTEMI: non-ST-elevation myocardial infarction, UA: unstable angina, LVEF: left ventricular ejection fraction, CHD: coronary heart disease, CABG: coronary artery bypass grafting, AMI: acute myocardial infarction, ACEI: angiotensin-converting enzyme inhibitor, ARB: angiotensin II receptor blockers, DAPT: dual antiplatelet therapy, RBC: red blood cell count, WBC: white blood cell count, PDW: platelet distribution width, RDW: red cell distribution width, CK-MB: creatine kinase isoenzymes, TG: triglyceride, TC: total cholesterol, LDL-C: low density lipoprotein cholesterol, HDL-C: high density lipoprotein cholesterol, ALT: alanine aminotransferase, AST: aspartate aminotransferase, ALB: albumin, FBG: fasting blood glucose, HbA1c: hemoglobin A1c, Scr: serum creatinine, FIB: fibrosis, TyG: triglyceride-glucose index, GNRI: Geriatric Nutritional Risk Index, MACE: major adverse cardiac event.

<sup>a</sup> student's t test; <sup>b</sup> Satterthwaite t test; <sup>c</sup> Chi-square test; <sup>d</sup> Wilcoxon rank sum test; <sup>e</sup> Fisher's exact test.

**Table 2.** Associations between TyG, GNRI, and MACE in patients with NSTEMI-ACS after PCI

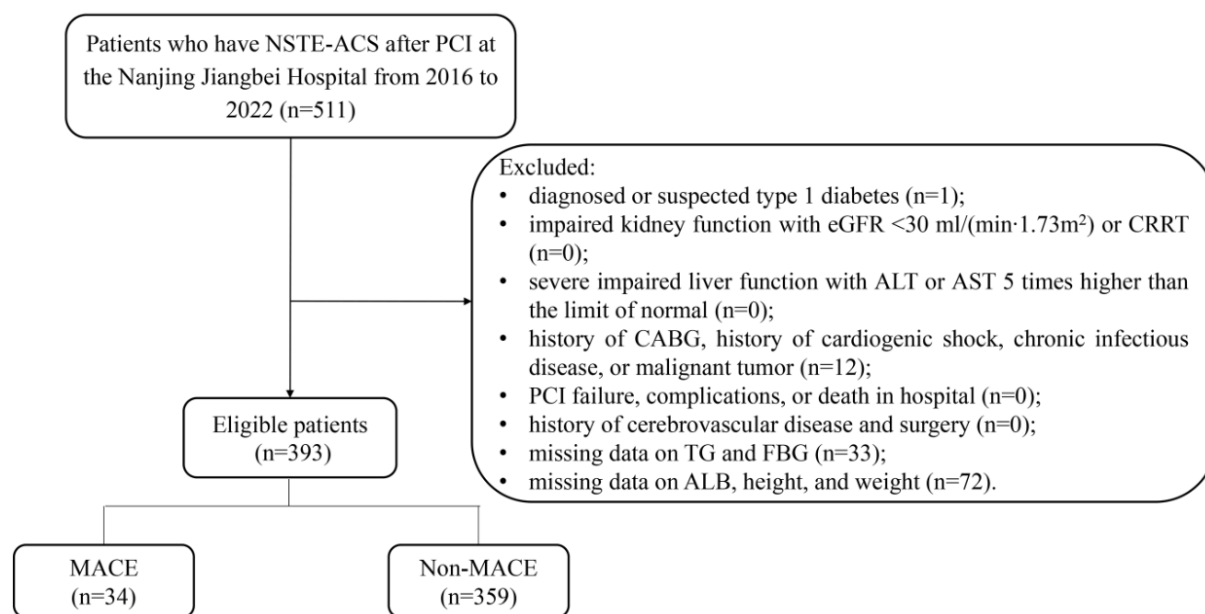
Variables	n (%)	Model 1		Model 2	
		OR (95% CI)	<i>p</i>	OR (95% CI)	<i>p</i>
TyG					
<1.359	165 (41.98)	Ref		Ref	
≥1.359	228 (58.02)	6.10 (2.35-20.85)	0.001	5.07 (1.64-15.71)	0.005
GNRI					
<107.514	316 (80.41)	Ref		Ref	
≥107.514	77 (19.59)	0.37 (0.09-1.08)	0.110	0.17 (0.04-0.68)	0.013

Ref: reference; OR: odds ratio; CI: confidence interval.

Model 1 was crude model.

Model 2 adjusting PDW, TC, pre-diffuse lesion, pre-bifurcation lesion, pre-in stent restenosis, and pre-complete revascularizations.





**Figure 1.** Selection process of the patients with NSTEMI-ACS

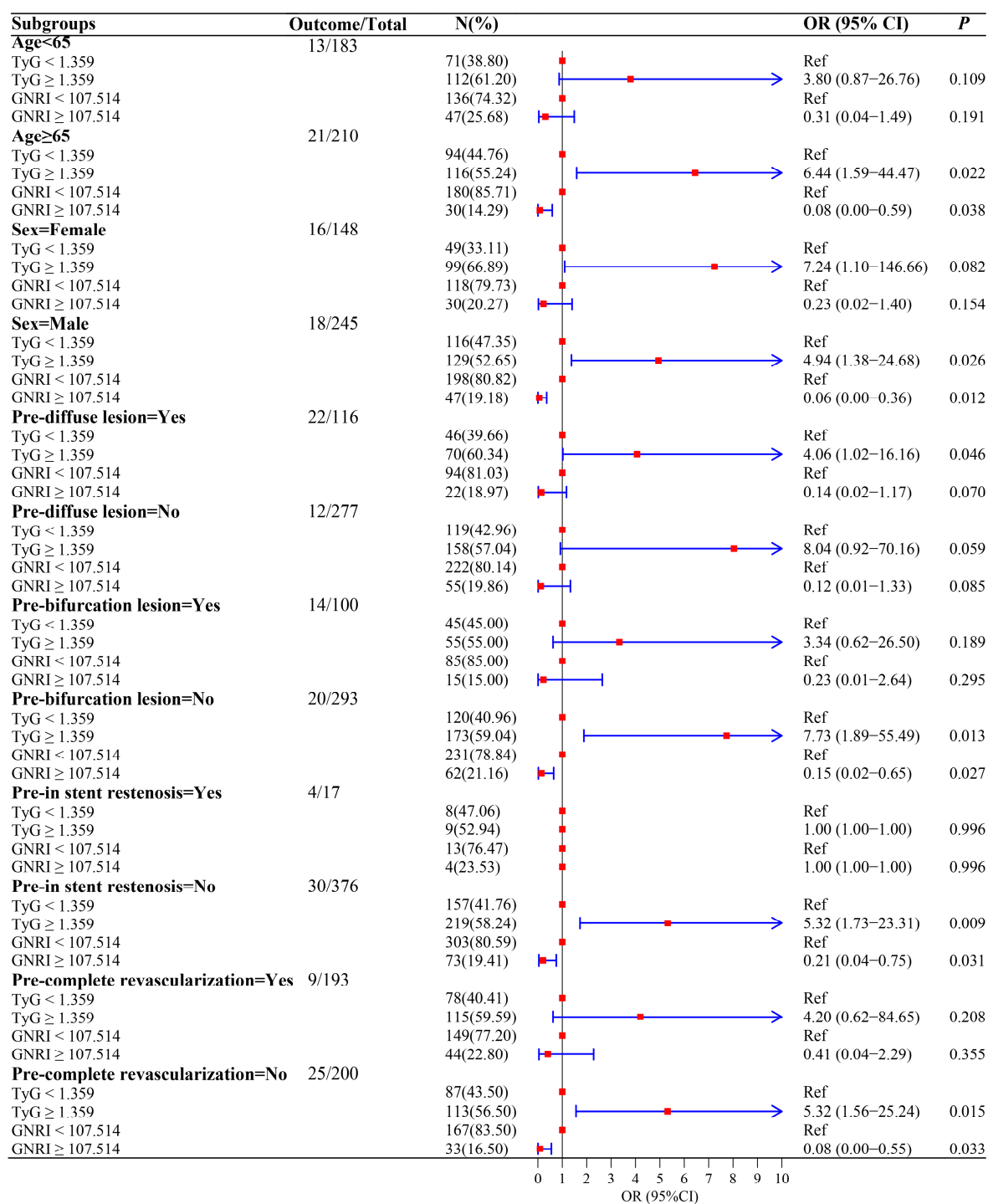
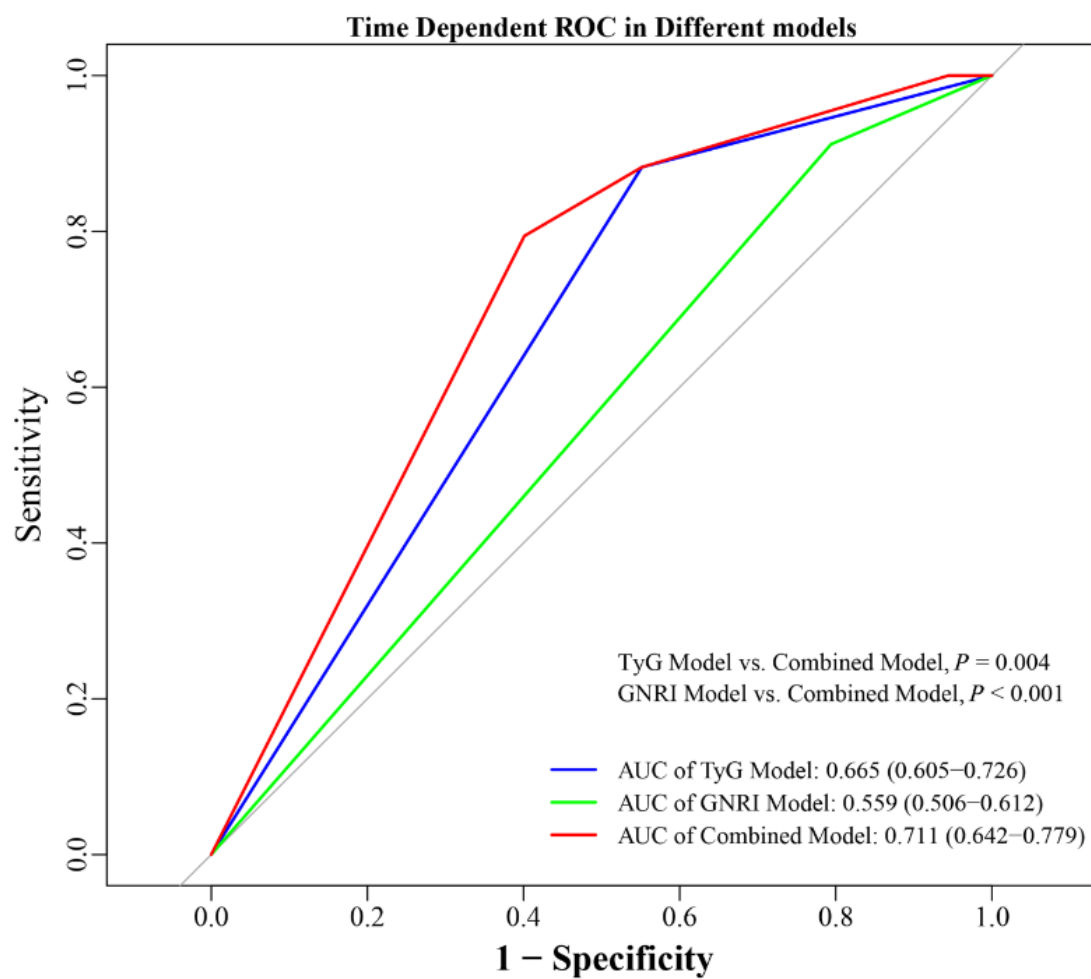


Figure 2. Associations between TyG, GNRI, and MACE in different subgroups



**Figure 3.** The ROC curves of predictive indicators in predicting the incidence of MACE

**Supplementary Table 1.** Associations between TyG, GNRI, and MACE in different age, gender, pre-diffuse lesion, pre-bifurcation lesion, pre-in stent restenosis, and pre-in stent restenosis subgroups

Subgroups (Outcome/Total)	n (%)	OR (95% CI)	<i>p</i>
Age<65 (n=13/183)			
TyG			
<1.359	71 (38.80)	Ref	
≥1.359	112 (61.20)	3.80 (0.74-19.45)	0.109
GNRI			
<107.514	136 (74.32)	Ref	
≥107.514	47 (25.68)	0.31 (0.05-1.80)	0.191
Age≥65 (n=21/210)			
TyG			
<1.359	94 (44.76)	Ref	
≥1.359	116 (55.24)	6.44 (1.32-31.57)	0.022
GNRI			
<107.514	180 (85.71)	Ref	
≥107.514	30 (14.29)	0.08 (0.01-0.87)	0.038
Gender=Female (n=16/148)			
TyG			
<1.359	49 (33.11)	Ref	
≥1.359	99 (66.89)	7.24 (0.78-67.54)	0.082
GNRI			
<107.514	118 (79.73)	Ref	
≥107.514	30 (20.27)	0.23 (0.03-1.74)	0.154
Gender=Male (n=18/245)			
TyG			
<1.359	116 (47.35)	Ref	
≥1.359	129 (52.65)	4.94 (1.22-20.09)	0.026
GNRI			
<107.514	198 (80.82)	Ref	
≥107.514	47 (19.18)	0.06 (0.01-0.53)	0.012
Pre-diffuse lesion=Yes (n=22/116)			
TyG			
<1.359	46 (39.66)	Ref	
≥1.359	70 (60.34)	4.06 (1.02-16.16)	0.046
GNRI			
<107.514	94 (81.03)	Ref	
≥107.514	22 (18.97)	0.14 (0.02-1.17)	0.070
Pre-diffuse lesion=No (n=12/277)			
TyG			
<1.359	119 (42.96)	Ref	
≥1.359	158 (57.04)	8.04 (0.92-70.16)	0.059
GNRI			
<107.514	222 (80.14)	Ref	
≥107.514	55 (19.86)	0.12 (0.01-1.33)	0.085
Pre-bifurcation lesion=Yes (n=14/100)			
TyG			
<1.359	45 (45.00)	Ref	
≥1.359	55 (55.00)	3.34 (0.55-20.20)	0.189
GNRI			
<107.514	85 (85.00)	Ref	
≥107.514	15 (15.00)	0.23 (0.01-3.64)	0.295
Pre-bifurcation lesion=No (n=20/293)			
TyG			
<1.359	120 (40.96)	Ref	
≥1.359	173 (59.04)	7.73 (1.53-39.07)	0.013
GNRI			
<107.514	231 (78.84)	Ref	
≥107.514	62 (21.16)	0.15 (0.03-0.80)	0.027

Ref: reference; HR: hazard ratio; CI: confidence interval.

Adjusting PDW, TC, pre-diffuse lesion, pre-bifurcation lesion, pre-in stent restenosis, pre-complete revascularization. The corresponding covariates are not processed in their own subgroups.

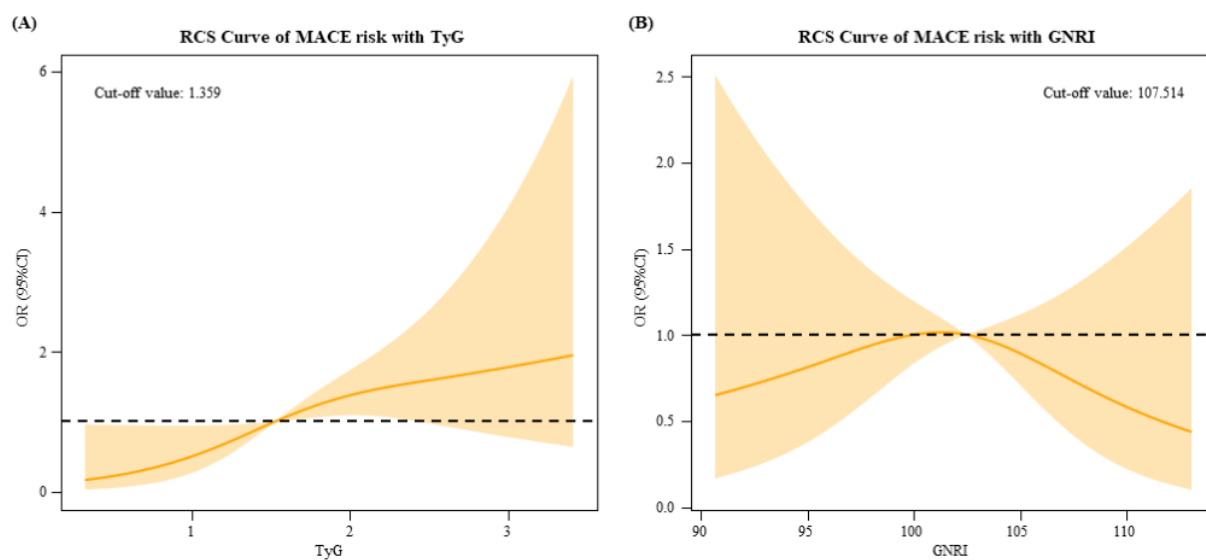
**Supplementary Table 1.** Associations between TyG, GNRI, and MACE in different age, gender, pre-diffuse lesion, pre-bifurcation lesion, pre-in stent restenosis, and pre-in stent restenosis subgroups

Subgroups (Outcome/Total)	n (%)	OR (95% CI)	<i>p</i>
Pre-in stent restenosis=Yes (n=4/17)			
TyG			
<1.359	8 (47.06)	Ref	
≥1.359	9 (52.94)	0.00 (0.00-Inf)	0.996
GNRI			
<107.514	13 (76.47)	Ref	
≥107.514	4 (23.53)	0.00 (0.00-Inf)	0.996
Pre-in stent restenosis=No (n=30/376)			
TyG			
<1.359	157 (41.76)	Ref	
≥1.359	219 (58.24)	5.32 (1.51-18.72)	0.009
GNRI			
<107.514	303 (80.59)	Ref	
≥107.514	73 (19.41)	0.21 (0.05-0.87)	0.031
Pre-complete revascularization=Yes (n=9/193)			
TyG			
<1.359	78 (40.41)	Ref	
≥1.359	115 (59.59)	4.20 (0.45-39.11)	0.208
GNRI			
<107.514	149 (77.20)	Ref	
≥107.514	44 (22.80)	0.41 (0.06-2.73)	0.355
Pre-complete revascularization=No (n=25/200)			
TyG			
<1.359	87 (43.50)	Ref	
≥1.359	113 (56.50)	5.32 (1.38-20.49)	0.015
GNRI			
<107.514	167 (83.50)	Ref	
≥107.514	33 (16.50)	0.08 (0.01-0.81)	0.033

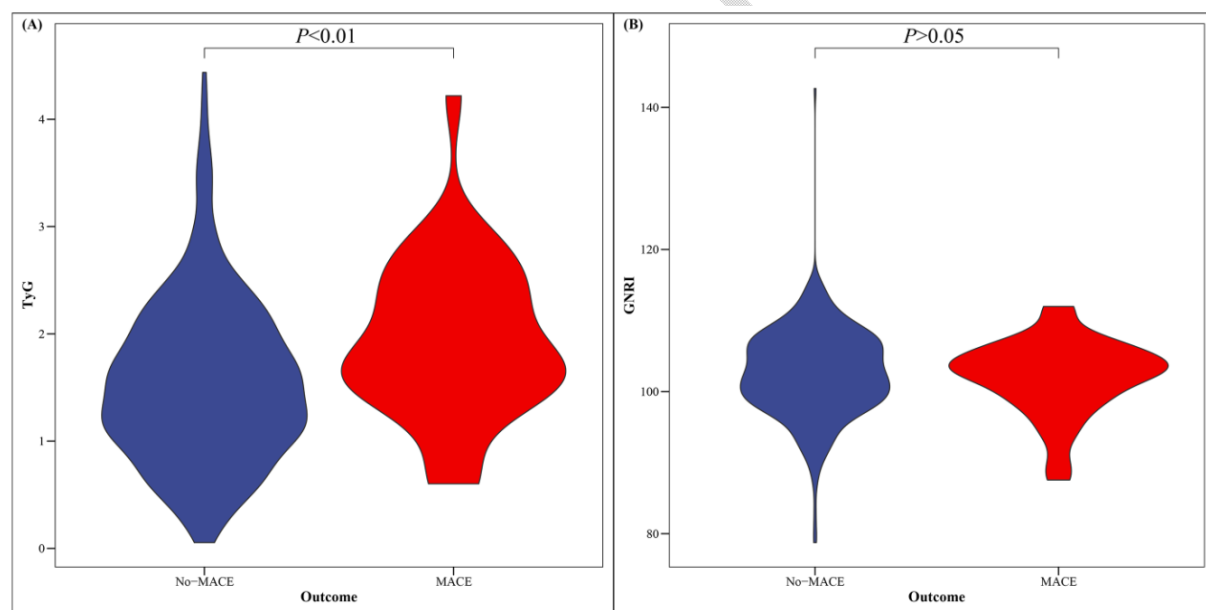
Ref: reference; HR: hazard ratio; CI: confidence interval.

Adjusting PDW, TC, pre-diffuse lesion, pre-bifurcation lesion, pre-in stent restenosis, pre-complete revascularization. The corresponding covariates are not processed in their own subgroups.

<sup>†</sup>*p*<0.05 compared with Gp1; <sup>‡</sup>*p*<0.05 compared with Gp2; <sup>§</sup>*p*<0.05 compared with Gm1; <sup>¶</sup>*p*<0.05 compared with Gm2; <sup>††</sup>*p*<0.05 compared with Gm3.



Supplementary Figure 1. Association between TyG, GNRI and incidence of MACE using RCS analysis



Supplementary Figure 2. Violin plots of TyG and GNRI in the MACE and no-MACE groups