The triglycerides-glucose index and the triglycerides to high-density lipoprotein cholesterol ratio are both effective predictors of in-hospital death in non-diabetic patients with AMI

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Background: The triglycerides-glucose index (TyG) and the triglycerides to high-density lipoprotein cholesterol (TG/HDL-C) are simple indicators for assessing insulin resistance in epidemiological studies. We aimed to clarify the relationship between indicators of insulin resistance and prognosis in non-diabetic acute myocardial infarction (AMI) patients.

Methods: 1648 AMI patients without diabetes were enrolled from the Department of Cardiology, Zhongda Hospital, between 2012.03 and 2018.12. The medical history, laboratory and imaging data of patients were collected through the medical record system, and all-cause death events were recorded. Pearson analysis was used to study the correlation among different variables. Logistic regression analysis was used to analyze the predictive effect of TyG and TG/HDL-C in in-hospital death of AMI patients. **Results**: 1. In AMI group, the TyG index was significantly increased in death groups compared to no-death groups (P=0.025). TG/HDL-C was not significantly increased in the death group of AMI patients (P=0.588). The patients were respectively divided into Q1-Q4 groups and T1-T4 groups according to the quartiles of TyG and TG/HDL-C. The trends of in-hospital mortality in the Q4 group of TyG and T4 group of TG/HDL-C were higher than in other groups, although these differences were not significant. 2. Pearson correlation analysis showed that TyG was positively correlated with lipid-related markers, including ApoB (r=0.248, P<0.001), total cholesterol (TC) (r=0.270, P<0.001), low-density lipoprotein cholesterol (LDL-C) (r=0.238, P<0.001). Spearman analysis showed that TG/HDL-C was also positively associated with TC (r=0.107, P<0.001), ApoB (r=0.180, P<0.001) and LDL-C (r=0.164, P<0.001). 3. Logistic regression analysis showed that TyG (OR=3.106, 95%CI=2.122-4.547, P<0.001) and TG/HDL-C (OR=1.167, 95%CI=1.062-1.282, P=0.001) were both important factors to predict the in-hospital death of AMI patients without

diabetes. **Conclusions**: TyG index and TG/HDL-C, as emerged simple markers of insulin resistance, were both important predictors of in-hospital death in AMI patients without diabetes.

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21 **Abstract**

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23 lipoprotein cholesterol (TG/HDL-C) are simple indicators for assessing insulin resistance in
24 epidemiological studies. We aimed to clarify the relationship between indicators of insulin
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27 Cardiology, Zhongda Hospital, between 2012.03 and 2018.12. The medical history, laboratory
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29 death events were recorded. Pearson analysis was used to study the correlation among different
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31 TG/HDL-C in in-hospital death of AMI patients.
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33 to no-death groups (P=0.025). TG/HDL-C was not significantly increased in the death group of
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36 the Q4 group of TyG and T4 group of TG/HDL-C were higher than in other groups, although
37 these differences were not significant. 2. Pearson correlation analysis showed that TyG was
38 positively correlated with lipid-related markers, including ApoB (r=0.248, P<0.001), total

39 cholesterol (TC) (r=0.270, P<0.001), low-density lipoprotein cholesterol (LDL-C) (r=0.238,
40 P<0.001). Spearman analysis showed that TG/HDL-C was also positively associated with TC
41 (r=0.107, P<0.001), ApoB (r=0.180, P<0.001) and LDL-C (r=0.164, P<0.001). 3. Logistic
42 regression analysis showed that TyG (OR=3.106, 95%CI=2.122-4.547, P<0.001) and TG/HDL-
43 C (OR=1.167, 95%CI=1.062-1.282, P=0.001) were both important factors to predict the in-
44 hospital death of AMI patients without diabetes.
45 **Conclusions**: TyG index and TG/HDL-C, as emerged simple markers of insulin resistance, were
46 both important predictors of in-hospital death in AMI patients without diabetes.
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48

49 ## Introduction
50 Acute coronary syndrome (ACS) is a severe type of coronary heart disease (*Reed GW et al.,*
51 *2017*). The Global Registry of acute coronary events (GRACE) study showed that the 1-year
52 mortality of ACS patients was about 15%, and the cumulative 5-year mortality was about 20%
53 (*Fox KA et al., 2010*). The mortality of acute myocardial infarction (AMI) is still on the rise in
54 China. Diabetes and metabolic syndrome are important risk factors for AMI (*DeFilippis AP et*
55 *al., 2019*). Identifying high-risk patients, early diagnosis and timely intervention of patients with
56 AMI to reduce mortality and improve prognosis have been important research directions.
57
58 Insulin resistance is a significant risk factor for cardiovascular disease (CVD), which is
59 associated with decreased cardiac autonomic function in the elderly without diabetes (*Laakso M*
60 *et al., 2014; Ormazabal V et al., 2018; Tenenbaum A et al., 2007; Howard G et al., 1996*).
61 Insulin resistance is characterized by the loss of endogenous or exogenous insulin effect,
62 resulting in glucose uptake and utilization disorders (*James DE et al., 2021*). A study showed
63 that nearly 58.4% of non-diabetic Parkinson's patients have been diagnosed with insulin
64 resistance (*Hogg E et al., 2018*). Tissue-specific insulin resistance, such as abnormalities in the
65 liver, muscle and adipose tissue, can lead to endothelial dysfunction and macrophage
66 accumulation, causing severe CVD events (*Poon AK et al., 2020; Jia D et al., 2021; Ryu J et al.,*
67 *2021*).
68
69 The triglycerides-glucose index (TyG) and the triglycerides to high-density lipoprotein
70 cholesterol (TG/HDL-C) are simple indicators for assessing insulin resistance in epidemiological
71 studies. TyG is the product of fasting triglyceride and fasting blood glucose (*Wang S et al.,*
72 *2021*). There is a positive correlation between TyG index and insulin resistance (*Dikaiakou E et*
73 *al., 2020*). Compared with the homeostasis model assessment of insulin resistance (HOMA-IR)
74 index and the euglycemic hyperinsulinemic clamp test, TyG is a new, concise and reliable
75 alternative index for evaluating insulin resistance (*Wang S et al., 2021*). Moreover, studies have
76 shown that a higher TyG index is associated with an increased risk of type 2 diabetes (*Xuan X et*
77 *al., 2021; Dikaiakou E et al., 2020*). In addition, TyG index is positively correlated with

78   coronary artery calcification and is an effective indicator for predicting the progress of CVD
79   (*Park K et al., 2019*).
80
81   TG/HDL-C is also a feasible indicator for evaluating insulin resistance (*Zhou M et al., 2016*). A
82   study that enrolled 9764 adults (average age 56 years old) who had participated in the
83   REACTION study in a Guangzhou community found that, compared with other blood lipid
84   markers, TG/HDL-C may be a better indicator for assessing insulin resistance and diabetes
85   among middle-aged and elderly Chinese populations (*Lin D et al., 2018*). Elahe Zare et al. used
86   TG/HDL-C as a surrogate index of insulin resistance in people with pulmonary hypertension
87   without a history of diabetes, and defined TG/HDL-C>3.0 as an indicator of insulin resistance.
88   This study found that insulin resistance was not associated with all-cause mortality in pulmonary
89   hypertension (*Zare E et al., 2022; Naderi N et al., 2014*).
90
91   Due to the crucial roles of TyG and TG/HDL-C in insulin resistance, this study aimed to use
92   TyG and TG/HDL-C to explore the effects of insulin resistance on cardiac function and in-
93   hospital death in non-diabetic AMI patients, and to provide guidance for the stratification of
94   clinical risk in patients with AMI.
95

Materials & Methods

Population

98   This design was an observational and retrospective study. 1648 AMI patients without diabetes,
99   consisting of 774 NSTEMI and 874 STEMI patients, were enrolled from the Department of
100 Cardiology, Zhongda Hospital between 2012.03 and 2018.12. Inclusion criteria: 1. Age > 18
101 years old; 2. The patient was diagnosed with AMI for the first time. Exclusion criteria: 1.
102 Pregnant or lactating women; 2. Patients with severe diseases, such as advanced malignant
103 tumors, and patients with an expected survival time of less than 3 months; 3. Diabetic patients.
104 The Ethics Committee of Zhongda Hospital affiliated to Southeast University
105 (2020ZDSYLL164-P01) approved this study and exempted the need for informed consent
106 signature.
107

Data collection

109 The demographic characteristics, medical history, laboratory and imaging data of patients were
110 collected by the medical record system (Yidu Cloud System, China), and death events were
111 recorded. In-hospital death was defined as all-cause death due to cardiovascular and non-
112 cardiovascular disease, and all deaths were reported with medical records (*Yusuf S et al., 2020;*
113 *Lee IM et al. 2019*). The demographic data included age, gender, smoking history and systolic
114 blood pressure (SBP). The medical history included history of hypertension history (HBP) and
115 diabetes. Blood routine examination included white blood cells (WBC), neutrophils,
116 lymphocytes, platelet (PLT) and hemoglobin (Hb). The biochemical test included alanine
117 aminotransferase (ALT), lactate dehydrogenase (LDH), aspartate aminotransferase (AST), blood

118   urea nitrogen (BUN), uric acid (UA), total cholesterol (TC), triglyceride (TG), albumin (ALB),
119   glucose (GLU), high-sensitivity C-reactive protein (hsCRP), low-density lipoprotein cholesterol
120   (LDL-C) and high-density lipoprotein cholesterol (HDL-C). Left ventricular ejection fraction
121   (LVEF) data were collected from echocardiography were collected. Fasting blood glucose and
122   lipid profiles for all participants were assessed upon admission. The TyG index was calculated
123   by ln(TG [mg/dL]*GLU [mg/dL]/2) (*Kahui Park et al., 2019*). TG/HDL-C was calculated as TG
124   dividing by HDL-C (*Wu Z et al., 2021*).
125
126   **Statistics**
127   Data were analyzed by SPSS 23.0 and GraphPad Prism 8 software. The Shapiro-Wilk test and Q-
128   Q plots were both used to test normality of continuous variables. For continuous data conforming
129   to a normal distribution, independent-sample T-test was used for comparison between two
130   groups. If the data did not follow a normal distribution, Mann-Whitney U was used for
131   comparison among groups. For categorical variables, comparison was performed by chi-square
132   test or Fisher's exact test. For variables conforming to normality, Pearson analysis were used to
133   test the correlation between TyG or TG/HDL-C and other variables. For rank data or data that do
134   not conform to a normal distribution, Spearman was used for correlation analysis. Univariate and
135   multivariate logistic regression analyses were used to analyze the predictive effect of TyG or
136   TG/HDL-C in in-hospital death of AMI patients. The variables included in logistic regression
137   must conform to independence and linearity.
138
139   # Results
140   **1. Basic characteristics of enrolled patients**
141   The Shapiro-Wilk test and Q-Q plots were both used to test the normality test of continuous
142   variables (Supplemental materials 1 and 2). homogeneity test for variance was performed
143   (Supplemental materials 3). Continuous variables with normality included age, SBP,
144   neutrophils, lymphocytes, PLT, Hb, LDL-C, HDL-C, UA, TC, ALB and LVEF. Two-sample
145   independent t-tests were used for comparison between death and no-death groups. Continuous
146   variables that did not conform to normality included WBC, ALT, AST, LDH, BUN, cTnI,
147   Creatinine, GLU, hsCRP and HbA1c. Rank sum test was used. The chi-square test was used to
148   compare categorical variables, including sex, smoking, medical history, and concomitant
149   medication. In the STEMI and NSTEMI groups, there were no differences in sex and
150   hypertension history between the death and no-death groups (P>0.05). The age in the death
151   groups was higher than in the no-death groups (P<0.001). The WBC count and neutrophils
152   were also significantly higher than that of no-death groups (P<0.05), indicating a severe
153   inflammatory state in death groups. There were no differences in ALT and AST between the
154   two groups (P>0.05). The ALB levels were significantly lower in death groups than in no-death
155   groups (P<0.001). The creatinine level was higher in death groups than in no-death groups
156   (P=0.001) (Table 1).
157

2. TyG and TG/HDL-C, both indicators of insulin resistance, are closely related to death after myocardial infarction

Normality of TyG was tested by the Shapiro-Wilk test and Q-Q plots. The absolute value of the sample kurtosis is less than 10 and the absolute value of the skewness is less than 3, TyG can be described as basically conforming to the normal distribution. Independent-sample T-test was used for comparison of TyG. The TyG index was significantly increased in the death groups compared to the no-death groups in AMI (8.86 ± 0.59 vs 9.22 ± 0.97, P=0.001), STEMI (8.90 ± 0.60 vs 9.24 ± 0.95, P=0.024) and NSTEMI (8.81 ± 0.58 vs 9.19 ± 1.00, P=0.022) patients (Figure 1A). Mann-Whitney U method was used for comparison of TG/HDL-C not conforming to normality. TG/HDL-C was not significantly increased in the death group of AMI patients (P=0.588) (Figure 1B). The patients were also divided into different groups according to the quartiles of TyG (Q1 <8.4543, Q2 8.4543-8.8418, Q3 8.8418-9.2294, Q4 ≥9.2294) and TG/HDL-C (T1 <2.0270, T2 2.0270-2.9487, T3 2.9487-4.3456, T4 ≥4.3456). The Chi-square test was used to test for differences in the trend of in-hospital mortality. The results showed that in-hospital mortality in the Q4 group of TyG (6.80%) and T4 group of TG/HDL-C (5.76%) was higher than in other groups (Q1=5.10%, Q2=3.64%, Q3=5.10%; T1=4.51%, T2=4.75%, T3=4.74%), despite the lack of statistically significant differences among these groups.

3. Pearson analysis of TyG and TG/HDL-C

Pearson was used for correlation analysis for the variables did not conform to normality, otherwise, Spearman was used. Correlation analysis showed that TyG was positively correlated with WBC (r=0.113, P<0.001), neutrophils (r=0.090, P<0.001, Hb (r=0.183, P<0.001), UA (r=0.113, P<0.001), TC (r=0.270, P<0.001), LDL-C (r=0.238, P<0.001), ApoB (r=0.248, P<0.001) and ALB (r=0.169, P<0.001). TyG was negatively associated with age (r=-0.217, P<0.001), BUN (r=-0.077, P=0.002), and not related to SBP (r=0.036, P=0.147) (Table 2). TG/HDL-C was positively associated with lymphocytes (r=0.240, P<0.001), Hb (r=0.227, P<0.001), ALB (r=0.165, P<0.001), TC (r=0.107, P<0.001), UA (r=0.185, P<0.001), ApoB (r=0.180, P<0.001), LDL-C (r=0.164, P<0.001) and LVEF (r=0.090, P=0.001), and negatively related to age (r=-0.326, P<0.001), BUN (r=-0.080, P=0.001), hsCRP (r=-0.103, P=0.024), ApoA (r=-0.311, P<0.001) (Table 2).

4. TyG and TG/HDL-C are important independent predictors of in-hospital death in AMI

Since this study only recorded the death status of enrolled subjects without recording the time of death, logistic regression instead of Cox regression was used to study the predictive ability of TyG and TG/HDL-C on in-hospital death of AMI patients. Due to a large number of variables (about 100) in this study and the sample size of 1648 patients, the inclusion of excess variables into the logistic model could affect the model's accuracy. Therefore, we chose to perform univariate logistic regression to screen out the significant variables that were subsequently included in multivariate logistic regression without omitting clinically important variables. Univariate logistic regression analysis showed that age (OR=1.079, 95%CI=1.056-1.102,

198   P<0.001), alcohol history (OR=2.234, 95%CI=1.019-4.896, P=0.045), smoking history
199   (OR=0.565, 95%CI=0.354-0.902, P=0.017), ALB (OR=0.847, 95%CI=0.815-0.881, P<0.001),
200   ALT (OR=1.001, 95%CI =1.001-1.002, P=0.001), BUN (OR=1.135, 95%CI=1.103-1.169,
201   P<0.001), Creatinine (OR=1.003, 95%CI=1.002-1.005, P<0.001), Hb (OR=0.970,
202   95%CI=0.961-0.978, P<0.001), TC (OR=0.991, 95%CI=0.985-0.996, P=0.001), SBP (OR=0.982,
203   95%CI=0.972-0.992, P<0.001), UA (OR=1.003, 95%CI=1.002-1.004, P<0.001), WBC
204   (OR=1.083, 95%CI=1.043-1.124, P <0.001) and TyG (OR=2.256, 95% CI=1.646-3.092,
205   P<0.001) were significant factors predicting in-hospital death in patients with AMI. The
206   significant variables in the univariate analysis were enrolled in the multivariate logistic
207   regression analysis. We used tolerance Tol and variance inflation factor VIF to evaluate whether
208   the collinearity of included variables was met (Supplemental materials 4). "Enter" method was
209   used for multivariate logistic regression analysis, and the results showed that age (OR=1.069,
210   95%CI=1.042-1.097, P<0.001), ALB (OR=0.926, 95%CI=0.879-0.976, P=0.004), ALT
211   (OR=1.001, 95%CI=1.000-1.002, P=0.028), BUN (OR=1.063, 95%CI=1.005-1.123, P=0.031),
212   SBP (OR= 0.982, 95%CI = 0.971-0.993, P = 0.002), WBC (OR=1.060, 95%CI =1.012-1.111,
213   P=0.014) and TyG (OR=3.106, 95%CI=2.122-4.547, P<0.001), were important factors
214   predicting the prognosis of AMI. "Forward" and "Backward" methods were also performed to
215   validate the result of "enter" method (Supplemental materials 5). The results of TyG by
216   "Forward" (OR=3.021, 95%CI=2.084-4.379, P<0.001) and "Backward" (OR=3.080,
217   95%CI=2.123-4.469, P<0.001) methods were similar with "enter" method. Based on the above
218   results, TyG was an important independent predictor of in-hospital death in AMI after adjusting
219   for multiple clinical confounders (Table 3, Figure 2A). When TG/HDL-C and significant factors
220   in univariate variables were included in to multivariate analysis, the result of "enter" method
221   showed that TG/HDL-C (OR=1.167, 95%CI=1.062-1.282, P=0.001) was also an important
222   predictor of in-hospital death (Table 4, Figure 2B). The results of TG/HDL-C by "Forward"
223   (OR=1.161, 95%CI=1.059-1.273, P=0.002) and "Backward" (OR=1.163, 95%CI=1.059-1.276,
224   P=0.001) methods were also similar with "enter" method (Supplemental materials 5).
225

Discussion

227   This study found that a higher TyG index and TG/HDL-C (markers of insulin resistance)
228   indicated poorer prognosis and higher mortality in AMI patients without diabetes. Higher TyG
229   and TG/HDL-C were also associated with higher inflammatory cells count and LDL-C, which
230   meant they were closely related to severe inflammatory states and dyslipidemia.
231

232   Insulin resistance is characterized by reduced insulin sensitivity and impaired oxidation and
233   glucose utilization (*Wu H et al., 2020*). Studies have shown that insulin resistance was found not
234   only in type 2 diabetes but also in a wide range of diseases with abnormal physiological
235   functions, triggering the proposal of the concept of insulin resistance syndrome (*Fletcher B et
236   al., 2004; Willard DL et al., 2016*). Insulin resistance was associated with the incidence of CVD
237   and could be used as a potential therapeutic target for CVD (*Rutter MK et al., 2008; Bersch-*

238   *Ferreira AC et al., 2018*). The gold standard for defining insulin resistance is the euglycemic
239   hyperinsulinemic clamp (*Geloneze B et al., 2006*). The HOMA-IR index is also an effective
240   marker for diagnosing insulin resistance (*Geloneze B et al., 2006*). However, both of them are
241   costly, and the identification of HOMA-IR was based on fasting insulin, which is not readily
242   available in a large-scale cross-sectional study or a retrospective design. A study that used TyG
243   and TG/HDL-C index as surrogate markers of insulin resistance to explore the longitudinal
244   association of insulin resistance with the progression of arteriosclerosis in the hypertensive
245   population found that the synergistic effect of hypertension and insulin resistance led to the
246   adverse progression of arteriosclerosis (*Wu Z et al., 2021*). Therefore, TyG and TG/HDL-C are
247   expected to perform as reliable and convenient markers, not inferior to the above two indicators.
248
249   Based on TyG and TG/HDL-C, we found insulin resistance was associated with inflammatory
250   states and dyslipidemia. Previous studies have demonstrated that the onset age of stroke and
251   hypertension decreased gradually with the increase of TyG index, which clarifies the negative
252   effects of TyG index on the onset of diseases (*Zhou Y et al., 2020; Gu S et al., 2020*). Insulin
253   resistance is always accompanied by chronic low-grade inflammation (*Perry BI et al., 2021*).
254   Increased WBC counts, neutrophil counts and lymphocyte counts are convenient markers for
255   evaluating systemic inflammation (*Perry BI et al., 2021*). In our study, the WBC counts and
256   lymphocyte counts were significantly associated with TyG index and TG/HDL-C, indicating that
257   insulin resistance may result from systemic inflammation. A higher TyG index was accompanied
258   by more severe inflammation. Moreover, the relationship between dyslipidemia and insulin
259   resistance was reciprocal (*Fassler CS et al., 2019*). On the one hand, lipid accumulation is one of
260   the leading causes of insulin resistance (*Wang S et al., 2021*). On the other hand, insulin
261   resistance is related to changes in lipoprotein and lipid metabolism (*van der Kolk BW et al.,
262   2019*). We found that TC and LDL-C levels were positively associated with higher TyG and
263   TG/HDL-C, which confirmed that insulin resistance was accompanied by dyslipidemia and other
264   metabolic disorders.
265
266   Insulin resistance was not only important in AMI patients with diabetes, but also in patients
267   without diabetes. Many clinical trials have determined that insulin resistance plays a vital role in
268   the development of CVD and can result in myocardial ischemia (*Klarin D et al., 2017; Masaki N
269   et al., 2019*). Furthermore, insulin resistance is currently considered an independent risk factor
270   for cardiovascular death (*Zhang Y et al., 2021*). In a large, multi-ethnic and prospective cohort
271   study of more than 15,000 postmenopausal women published in Circulation: Cardiovascular
272   Quality and Outcomes, insulin levels, HOMA-IR, blood glucose, and TG/HDL-C were measured
273   to assess insulin resistance. After adjusting for traditional cardiovascular risk factors such as age,
274   race/ethnicity, smoking, total cholesterol, systolic blood pressure, and treatment with
275   antihypertensive agents, insulin resistance was significantly associated with increased CVD risk
276   (*Schmiegelow MD et al., 2015*). Previous studies on TyG and TG/HDL-C and insulin resistance
277   mainly focused on AMI patients with diabetes (*Zhang Y et al., 2021*). However, we mainly

278 explored the role of TyG and TG/HDL-C in predicting the prognosis of AMI patients without
279 diagnosis of diabetes due to diabetes being an significant risk factor for AMI. It was interesting
280 and significant for us to found insulin resistance help predict short-term death in non-diabetes
281 AMI patients.
282
283 There are still some limitations of our study. First, the long-term follow-up was not performed,
284 limiting the survival analysis of high TyG and TG/HDL-C patients. Second, the roles of TyG and
285 TG/HDL-C in AMI patients with or without diabetes were not compared. Third, HOMA-IR and
286 serum insulin levels were not measured.
287

Conclusions

289 In conclusion, the emerged simple markers of insulin resistance, TyG index and TG/HDL-C are
290 important predictors of in-hospital death in AMI patients without diabetes after adjusting
291 significant variables such as age, SBP, BUN, WBC, ALT and ALB. For the population without
292 diabetes, it is also worth emphasizing the importance of screening for insulin resistance and
293 actively taking early intervention measures.
294

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297

References

299 **Bersch-Ferreira ÂC, Sampaio GR, Gehringer MO, Torres EAFDS, Ross-Fernandes MB,**
300 **da Silva JT, Torreglosa CR, Kovacs C, Alves R, Magnoni CD, Weber B, Rogero MM. 2018.**
301 Association between plasma fatty acids and inflammatory markers in patients with and without
302 insulin resistance and in secondary prevention of cardiovascular disease, a cross-sectional study.
303 Nutr J **17(1)**: 26 DOI 10.1186/s12937-018-0342-1.
304 **DeFilippis AP, Chapman AR, Mills NL, de Lemos JA, Arbab-Zadeh A, Newby LK,**
305 **Morrow DA. 2019.** Assessment and Treatment of Patients With Type 2 Myocardial Infarction
306 and Acute Nonischemic Myocardial Injury. Circulation **140(20)**: 1661-1678 DOI
307 10.1161/CIRCULATIONAHA.119.040631.
308 **Dikaiakou E, Vlachopapadopoulou EA, Paschou SA, Athanasouli F, Panagiotopoulos I,**
309 **Kafetzi M, Fotinou A, Michalacos S. 2020.** Triglycerides-glucose (TyG) index is a sensitive
310 marker of insulin resistance in Greek children and adolescents. Endocrine **70(1)**: 58-64 DOI
311 10.1007/s12020-020-02374-6.
312 **Fassler CS, Pinney SE, Xie C, Biro FM, Pinney SM. 2019.** Complex relationships between
313 perfluorooctanoate, body mass index, insulin resistance and serum lipids in young girls. Environ
314 Res **176**: 108558 DOI 10.1016/j.envres.2019.108558.
315 **Fletcher B, Lamendola C. 2004.** Insulin resistance syndrome. J Cardiovasc Nurs **19(5)**: 339-
316 345 DOI 10.1097/00005082-200409000-00009.

317 **Fox KA, Carruthers KF, Dunbar DR, Graham C, Manning JR, De Raedt H, Buysschaert I,**
318 **Lambrechts D, Van de Werf F. 2010.** Underestimated and under-recognized: the late
319 consequences of acute coronary syndrome (GRACE UK-Belgian Study). Eur Heart J **31(22)**:
320 2755-2764 DOI 10.1093/eurheartj/ehq326.

321 **Geloneze B, Tambascia MA. 2006.** Laboratorial evaluation and diagnosis of insulin resistance.
322 Arq Bras Endocrinol Metabol **50(2)**: 208-215 DOI 10.1590/s0004-27302006000200007.

323 **Gu S, Wang A, Ning G, Zhang L, Mu Y. 2020.** Insulin resistance is associated with urinary
324 albumin-creatinine ratio in normal weight individuals with hypertension and diabetes: The
325 REACTION study. J Diabetes **12(5)**: 406-416 DOI 10.1111/1753-0407.13010.

326 **Hogg E, Athreya K, Basile C, Tan EE, Kaminski J, Tagliati M. 2018.** High Prevalence of
327 Undiagnosed Insulin Resistance in Non-Diabetic Subjects with Parkinson's Disease. J
328 Parkinsons Dis **8(2)**: 259-265 DOI 10.3233/JPD-181305.

329 **Howard G, O'Leary DH, Zaccaro D, Haffner S, Rewers M, Hamman R, Selby JV, Saad**
330 **MF, Savage P, Bergman R. 1996.** Insulin sensitivity and atherosclerosis. The Insulin Resistance
331 Atherosclerosis Study (IRAS) Investigators. Circulation **93(10)**: 1809-1817 DOI
332 10.1161/01.cir.93.10.1809.

333 **James DE, Stockli J, Birnbaum MJ. 2021.** The aetiology and molecular landscape of insulin
334 resistance. Nat Rev Mol Cell Biol **22(11)**:751-771 DOI 10.1038/s41580-021-00390-6.

335 **Jia D, Zhang J, Liu X, Andersen JP, Tian Z, Nie J, Shi Y. 2021.** Insulin Resistance in
336 Skeletal Muscle Selectively Protects the Heart in Response to Metabolic Stress. Diabetes **70(10)**:
337 2333-2343 DOI 10.2337/db20-1212.

338 **Klarin D, Zhu QM, Emdin CA, Chaffin M, Horner S, McMillan BJ, Leed A, Weale ME,**
339 **Spencer CCA, Aguet F, Segrè AV, Ardlie KG, Khera AV, Kaushik VK, Natarajan P;**
340 **CARDIoGRAMplusC4D Consortium, Kathiresan S. 2017.** Genetic analysis in UK Biobank
341 links insulin resistance and transendothelial migration pathways to coronary artery disease. Nat
342 Genet **49(9)**: 1392-1397 DOI 10.1038/ng.3914.

343 **Laakso M, Kuusisto J. 2014.** Insulin resistance and hyperglycaemia in cardiovascular disease
344 development. Nat Rev Endocrinol **10(5)**: 293-302 DOI 10.1038/nrendo.2014.29.

345 **Lee IM, Shiroma EJ, Kamada M, Bassett DR, Matthews CE, Buring JE. 2019.** Association
346 of Step Volume and Intensity With All-Cause Mortality in Older Women. JAMA Intern Med
347 **179(8)**: 1105-1112 DOI 10.1001/jamainternmed.2019.0899.

348 **Lin D, Qi Y, Huang C, Wu M, Wang C, Li F, Yang C, Yan L, Ren M, Sun K. 2018.**
349 Associations of lipid parameters with insulin resistance and diabetes: A population-based study.
350 Clin Nutr **37(4)**: 1423-1429 DOI 10.1016/j.clnu.2017.06.018.

351 **Masaki N, Ido Y, Yamada T, Yamashita Y, Toya T, Takase B, Hamburg NM, Adachi T.**
352 **2019.** Endothelial Insulin Resistance of Freshly Isolated Arterial Endothelial Cells From Radial
353 Sheaths in Patients With Suspected Coronary Artery Disease. J Am Heart Assoc **8(6)**: e010816
354 DOI 10.1161/JAHA.118.010816.

355 **Naderi N, Boobejame P, Bakhshandeh H, Amin A, Taghavi S, Maleki M. 2014.** Insulin
356 resistance in pulmonary arterial hypertension, is it a novel disease modifier? Res Cardiovasc
357 Med **3(3)**: e19710 DOI 10.5812/cardiovascmed.19710.
358 **Ormazabal V, Nair S, Elfeky O, Aguayo C, Salomon C, Zuñiga FA. 2018.** Association
359 between insulin resistance and the development of cardiovascular disease. Cardiovasc Diabetol
360 **17(1)**: 122 DOI 10.1186/s12933-018-0762-4.
361 **Park K, Ahn CW, Lee SB, Kang S, Nam JS, Lee BK, Kim JH, Park JS. 2019.** Elevated TyG
362 Index Predicts Progression of Coronary Artery Calcification. Diabetes Care 42(8): 1569-1573
363 DOI 10.2337/dc18-1920.
364 **Perry BI, Burgess S, Jones HJ, Zammit S, Upthegrove R, Mason AM, Day FR, Langenberg**
365 **C, Wareham NJ, Jones PB, Khandaker GM. 2021.** The potential shared role of inflammation
366 in insulin resistance and schizophrenia: A bidirectional two-sample mendelian randomization
367 study. PLoS Med **18(3)**: e1003455 DOI 10.1371/journal.pmed.1003455.
368 **Poon AK, Whitsel EA, Heiss G, Soliman EZ, Wagenknecht LE, Suzuki T, Loehr L. 2020.**
369 Insulin resistance and reduced cardiac autonomic function in older adults: the Atherosclerosis
370 Risk in Communities study. BMC Cardiovasc Disord **20(1)**: 217 DOI 10.1186/s12872-020-
371 01496-z.
372 **Reed GW, Rossi JE, Cannon CP. 2017.** Acute myocardial infarction. Lancet **389(10065)**: 197-
373 210 DOI 10.1016/S0140-6736(16)30677-8.
374 **Rutter MK, Wilson PW, Sullivan LM, Fox CS, D'Agostino RB Sr, Meigs JB. 2008.** Use of
375 alternative thresholds defining insulin resistance to predict incident type 2 diabetes mellitus and
376 cardiovascular   disease.   Circulation   **117(8)**:   1003-1009   DOI
377 10.1161/CIRCULATIONAHA.107.727727.
378 **Ryu J, Hadley JT, Li Z, Dong F, Xu H, Xin X, Zhang Y, Chen C, Li S, Guo X, Zhao JL,**
379 **Leach RJ, Abdul-Ghani MA, DeFronzo RA, Kamat A, Liu F, Dong LQ. 2021.** Adiponectin
380 Alleviates Diet-Induced Inflammation in the Liver by Suppressing MCP-1 Expression and
381 Macrophage Infiltration. Diabetes **70(6)**: 1303-1316 DOI 10.2337/db20-1073.
382 **Schmiegelow MD, Hedlin H, Stefanick ML, Mackey RH, Allison M, Martin LW, Robinson**
383 **JG, Hlatky MA. 2015.** Insulin Resistance and Risk of Cardiovascular Disease in
384 Postmenopausal Women: A Cohort Study From the Women's Health Initiative. Circ Cardiovasc
385 Qual Outcomes **8(3)**: 309-16 DOI 10.1161/CIRCOUTCOMES.114.001563.
386 **Tenenbaum A, Adler Y, Boyko V, Tenenbaum H, Fisman EZ, Tanne D, Lapidot M,**
387 **Schwammenthal E, Feinberg MS, Matas Z, Motro M, Behar S. 2007.** Insulin resistance is
388 associated with increased risk of major cardiovascular events in patients with preexisting
389 coronary artery disease. Am Heart J **153(4)**: 559-565 DOI 10.1016/j.ahj.2007.01.008.
390 **van der Kolk BW, Vogelzangs N, Jocken JWE, Valsesia A, Hankemeier T, Astrup A, Saris**
391 **WHM, Arts ICW, van Greevenbroek MMJ, Blaak EE; DiOGenes consortium. 2019.**
392 Plasma lipid profiling of tissue-specific insulin resistance in human obesity. Int J Obes (Lond)
393 **43(5)**: 989-998 DOI 10.1038/s41366-018-0189-8.

394 **Wang S, Schianchi F, Neumann D, Wong LY, Sun A, van Nieuwenhoven FA, Zeegers MP,**
395 **Strzelecka A, Col U, Glatz JFC, Nabben M, Luiken JJFP. 2021.** Specific amino acid
396 supplementation rescues the heart from lipid overload-induced insulin resistance and contractile
397 dysfunction by targeting the endosomal mTOR-v-ATPase axis. Mol Metab **53**: 101293 DOI
398 10.1016/j.molmet.2021.101293.
399 **Wang S, Shi J, Peng Y, Fang Q, Mu Q, Gu W, Hong J, Zhang Y, Wang W. 2021.** Stronger
400 association of triglyceride glucose index than the HOMA-IR with arterial stiffness in patients
401 with type 2 diabetes: a real-world single-centre study. Cardiovasc Diabetol **20(1)**: 82 DOI
402 10.1186/s12933-021-01274-x.
403 **Willard DL, Stevenson M, Steenkamp D. 2016.** Type B insulin resistance syndrome. Curr
404 Opin Endocrinol Diabetes Obes **23(4)**: 318-323 DOI 10.1097/MED.0000000000000263.
405 **Wu H, Ballantyne CM. 2020.** Metabolic Inflammation and Insulin Resistance in Obesity. Circ
406 Res **126(11)**: 1549-1564 DOI 10.1161/CIRCRESAHA.119.315896.
407 **Wu Z, Zhou D, Liu Y, Li Z, Wang J, Han Z, Miao X, Liu X, Li X, Wang W, Guo X, Tao L.**
408 **2021.** Association of TyG index and TG/HDL-C ratio with arterial stiffness progression in a non-
409 normotensive population. Cardiovasc Diabetol **20(1)**: 134 DOI 10.1186/s12933-021-01330-6.
410 **Xuan X, Hamaguchi M, Cao Q, Okamura T, Hashimoto Y, Obora A, Kojima T, Fukui M,**
411 **Yuan G, Guo Z, Luo Z, Qin Y, Luo X, Xie X. 2021.** U-shaped association between the
412 triglyceride-glucose index and the risk of incident diabetes in people with normal glycemic level:
413 A population-base longitudinal cohort study. Clin Nutr **40(4)**: 1555-1561 doi:
414 10.1016/j.clnu.2021.02.037.
415 **Yusuf S, Joseph P, Rangarajan S, Islam S, Mente A, Hystad P, Brauer M, Kutty VR,**
416 **Gupta R, Wielgosz A, AlHabib KF, Dans A, Lopez-Jaramillo P, Avezum A, Lanas F, Oguz**
417 **A, Kruger IM, Diaz R, Yusoff K, Mony P, Chifamba J, Yeates K, Kelishadi R, Yusufali A,**
418 **Khatib R, Rahman O, Zatonska K, Iqbal R, Wei L, Bo H, Rosengren A, Kaur M, Mohan**
419 **V, Lear SA, Teo KK, Leong D, O'Donnell M, McKee M, Dagenais G. 2020.** Modifiable risk
420 factors, cardiovascular disease, and mortality in 155 722 individuals from 21 high-income,
421 middle-income, and low-income countries (PURE): a prospective cohort study. Lancet
422 **395(10226)**: 795-808 DOI 10.1016/S0140-6736(19)32008-2.
423 **Zare E, Kafshbani P, Chenaghlou M, Noori M, Ghaemmaghami Z, Amin A, Taghavi S,**
424 **Naderi N. 2022.** Prognostic significance of insulin resistance in pulmonary hypertension. ESC
425 Heart Fail **9(1)**: 318-326 DOI 10.1002/ehf2.13752.
426 **Zhang Y, Ding X, Hua B, Liu Q, Gao H, Chen H, Zhao XQ, Li W, Li H. 2021.** Predictive
427 effect of triglyceride-glucose index on clinical events in patients with type 2 diabetes mellitus
428 and acute myocardial infarction: results from an observational cohort study in China. Cardiovasc
429 Diabetol **20(1)**: 43 DOI 10.1186/s12933-021-01236-3.
430 **Zhou M, Zhu L, Cui X, Feng L, Zhao X, He S, Ping F, Li W, Li Y. 2016.** The triglyceride to
431 high-density lipoprotein cholesterol (TG/HDL-C) ratio as a predictor of insulin resistance but not
432 of β cell function in a Chinese population with different glucose tolerance status. Lipids Health
433 Dis **15**: 104 DOI 10.1186/s12944-016-0270-z.

434 **Zhou Y, Pan Y, Yan H, Wang Y, Li Z, Zhao X, Li H, Meng X, Wang C, Liu L, Wang Y.**
435 **2020.** Triglyceride Glucose Index and Prognosis of Patients With Ischemic Stroke. Front Neurol
436 **11**: 456 DOI 10.3389/fneur.2020.00456.
437

Table 1 Basic characteristics of AMI patients.

For continuous data conformed to normality, data was described as mean±standard deviation (X±SD). For data did not conform to normality, data was described by quartiles [50% (25%, 75%)]. *P<0.05; **P<0.01; ***P<0.001. Systolic blood pressure (SBP), Lactate dehydrogenase (LDH), Alanine aminotransferase (ALT), Aspartate aminotransferase (AST), Low-density lipoprotein cholesterol (LDL-C), High-density lipoprotein cholesterol (HDL-C), Blood urea nitrogen (BUN), Uric acid (UA), Total cholesterol (TC), Albumin (ALB), Glucose (GLU), White blood cells (WBC), Hemoglobin (Hb), Glycosylated hemoglobin (HbA1C), Left ventricular ejected fraction (LVEF), Hypertension (HBP), Chronic kidney disease (CKD), Platelet (PLT).

Characteristics	STEMI			NSTEMI		
	Without death (n=829)	Death (n=45)	P value	Without death (n=734)	Death (n=40)	P value
Demographic characteristics						
Sex (female)	175(21.11%)	10(22.22%)	0.859	225(30.65%)	18(45.00%)	0.057
Age (years)	67.65±14.01	78.87±14.40	<0.001***	73.37±13.21	85.90±6.38	<0.001***
Smoking	426(51.39%)	18(40.00%)	0.137	280(38.15%)	9(22.50%)	<0.046*
SBP (mmHg)	127.67±21.37	121.23±25.80	0.054	134.87±22.18	122.87±27.51	0.001**
Blood routine examination						
Neutrophils (10⁹/L)	8.19±4.38	10.56±6.47	0.022*	6.05±3.45	8.38±5.67	0.014*
Lymphocytes (10⁹/L)	1.39±0.66	1.34±0.90	0.620	1.45±0.69	1.14±0.73	0.006**
WBC (10⁹/L)	9.34(7.08, 12.19)	11.21(7.14, 16.93)	0.043*	7.32(6.00, 9.28)	8.60(6.49, 11.76)	<0.001***
PLT (10⁹/L)	207.79±64.95	195.53±93.16	0.399	194.60±70.01	178.38±80.78	0.158
Hb (g/L)	133.31±20.11	119.72±26.36	0.002**	130.76±22.32	110.18±26.49	<0.001***
Biochemical test						
ALT (U/L)	39.00(24.00, 61.00)	34.00(15.50, 84.50)	0.655	24.00(16.00, 35.00)	30.00(13.00, 38.75)	0.223
AST (U/L)	98.00(36.00, 207.75)	57.00(28.00, 306.50)	0.483	33.00(22.00, 62.00)	47.50(21.25, 150.25)	0.057
LDH (U/L)	426.50(251.25, 737.50)	405.00(272.50, 877.50)	0.407	235.00(178.00, 311.00)	300.00(195.75, 546.50)	0.010*
LDL-C (mmol/L)	2.80±0.86	2.51±1.06	0.084	2.71±0.91	2.31±0.90	0.008**
HDL-C (mmol/L)	1.10±0.26	0.98±0.26	0.007**	1.09±0.27	1.00±0.34	0.106
BUN (mmol/L)	5.10(4.20, 6.70)	8.80(6.05, 15.50)	<0.001***	5.90(4.70, 7.90)	10.35(8.18, 15.08)	<0.001***
UA (μmol/L)	349.80±111.27	434.98±211.36	0.010*	362.79±121.54	385.80±157.95	0.370
cTnI (ng/ml)	3.82(0.57, 12.00)	2.84(0.11, 11.50)	0.219	0.67(0.13, 2.92)	1.60(0.15, 7.00)	0.165
TC (mmol/L)	4.48±1.11	4.09±1.52	0.099	4.39±1.16	3.94±1.23	0.017*
ALB (g/L)	37.56±4.65	32.43±6.58	<0.001***	37.23±4.96	32.41±6.64	<0.001***
Creatinine (μmol/L)	80.00(66.00, 96.50)	119.00(91.50, 191.00)	<0.001***	86.50(69.00, 109.00)	128.50(103.50, 212.75)	0.009*
GLU (mmol/L)	6.41(5.53, 7.62)	7.56(5.44, 9.41)	0.027*	6.13(5.37, 7.19)	6.68(5.61, 8.29)	0.042*
hsCRP (mg/L)	16.35(4.27, 49.98)	24.50(12.00, 95.90)	0.083	9.56(2.33, 37.38)	44.10(12.18, 81.45)	0.002**
HbA1c (%)	5.70(5.40, 6.30)	6.10(5.95, 6.90)	0.081	5.80(5.50, 6.20)	6.25(5.85, 6.88)	0.105

Echocardiography						
LVEF	0.55±0.12	0.49±0.17	0.093	0.60±0.13	0.51±0.17	0.001**
Medical history						
HBP	464(55.97%)	24(53.33%)	0.729	522(71.12%)	32(80.00%)	0.225
CKD	30(3.61%)	6(13.33%)	0.001**	57(7.77%)	8(22.00%)	0.007**
Concomitant medication						
Hypotensive drugs	601(72.50%)	11(24.44%)	<0.001***	524(71.39%)	16(40.00%)	<0.001***
Lipid-lowering drugs	783(94.45%)	34(75.56%)	<0.001***	704(95.91%)	28(70.00%)	<0.001***
Aspirin	787(94.93%)	33(73.33%)	<0.001***	710(96.73%)	28(70.00%)	<0.001***
Diuretics	333(40.17%)	10(22.22%)	0.016*	280(38.15%)	8(20.00%)	0.021*
β-blockers	704(84.92%)	20(44.44%)	<0.001***	605(82.43%)	20(50.00%)	<0.001***

Table 2 Correlations analysis about TyG and TG/HDL-C.

For variables conforming to normality, Pearson was used for correlation analysis, otherwise, Spearman was used. Correlation analysis among age, SBP, WBC, neutrophils, Hb, ALB, TC, UA, hsCRP, ApoB, ApoA, LDL-C, LVEF and TyG were performed by Pearson. Correlation analysis among lymphocytes, creatinine, BUN and TyG were performed by Spearman. Correlation analysis between TG/HDL-C and other factors were performed by Spearman. *P<0.05; **P<0.01; ***P<0.001. White blood cells (WBC), Systolic blood pressure (SBP), Total cholesterol (TC), Uric acid (UA), Blood urea nitrogen (BUN), Albumin (ALB), Hemoglobin (Hb), Low-density lipoprotein cholesterol (LDL-C), Left ventricular ejected fraction (LVEF), high sensitivity C reactive protein (hsCRP).

Variables	Correlation analysis of TyG		Correlation analysis of TG/HDL-C	
	r	p	r	p
Age (years)	-0.217	<0.001***	-0.326	<0.001***
SBP (mmHg)	0.036	0.147	0.007	0.794
WBC (10^9/L)	0.113	<0.001***	0.034	0.192
Neutrophils (10^9/L)	0.090	<0.001***	-0.018	0.488
Lymphocytes (10^9/L)	0.172	<0.001***	0.240	<0.001***
Hb (g/L)	0.183	<0.001***	0.227	<0.001***
ALB (g/L)	0.169	<0.001***	0.165	<0.001***
Creatinine (μmol/L)	-0.029	0.238	0.009	0.706
TC (mmol/L)	0.270	<0.001***	0.107	<0.001***
UA (μmol/L)	0.113	<0.001***	0.185	<0.001***
BUN (mmol/L	-0.077	0.002***	-0.080	0.001***
hsCRP (mg/L)	-0.028	0.538	-0.103	0.024*
ApoB (g/L)	0.248	<0.001***	0.180	<0.001***
ApoA (g/L)	0.001	0.968	-0.311	<0.001***
LDL-C (mmol/L)	0.238	<0.001***	0.164	<0.001***
LVEF	0.030	0.280	0.090	0.001***

Table 3 Logistic regression of predicting in-hospital death in AMI patients without diabetes.

*P<0.05; **P<0.01; ***P<0.001. Albumin (ALB), Alanine aminotransferase (ALT), Blood urea nitrogen (BUN), Systolic blood pressure (SBP), Total cholesterol (TC), Uric acid (UA), White blood cells (WBC).

Parameters	Univariate regression			Variate regression		
	OR	95%CI	P value	OR	95%CI	P value
Age (years)	1.079	1.056-1.102	<0.001***	1.069	1.042-1.097	<0.001***
Alcohol	2.234	1.019-4.896	0.045*	1.243	0.488-3.165	0.648
Smoking	0.565	0.354-0.902	0.017*	1.097	0.607-1.981	0.760
ALB (g/L)	0.847	0.815-0.881	<0.001***	0.926	0.879-0.976	0.004***
ALT (U/L)	1.001	1.001-1.002	0.001**	1.001	1.000-1.002	0.028*
BUN (mmol/L)	1.135	1.103-1.169	<0.001***	1.063	1.005-1.123	0.031*
Creatinine (µmol/L)	1.003	1.002-1.005	<0.001***	1.000	0.998-1.003	0.887
Hb (g/L)	0.970	0.961-0.978	<0.001***	0.991	0.979-1.003	0.144
TC (mg/dl)	0.991	0.985-0.996	0.001***	0.999	0.993-1.005	0.698
SBP (mmHg)	0.982	0.972-0.992	<0.001***	0.982	0.971-0.993	0.002**
UA (µmol/L)	1.003	1.002-1.004	<0.001***	1.000	0.998-1.002	0.828
WBC (10^9/L)	1.083	1.043-1.124	<0.001***	1.060	1.012-1.111	0.014**
TyG	2.256	1.646-3.092	<0.001***	3.106	2.122-4.547	<0.001***

Table 4 Logistic regression of predicting in-hospital death in AMI patients without diabetes.

*P<0.05; **P<0.01; ***P<0.001. Albumin (ALB), Alanine aminotransferase (ALT), Blood urea nitrogen (BUN), Hemoglobin (Hb), Systolic blood pressure (SBP), Total cholesterol (TC), Uric acid (UA), White blood cells (WBC).

Parameters	Univariate regression			Variate regression		
	HR	95%CI	P value	HR	95%CI	P value
Age (years)	1.079	1.056-1.102	<0.001***	1.069	1.041-1.098	<0.001***
Alcohol	2.234	1.019-4.896	0.045*	1.023	0.406-2.574	0.962
Smoking	0.565	0.354-0.902	0.017*	1.101	0.611-1.983	0.748
ALB (g/L)	0.847	0.815-0.881	<0.001***	0.935	0.887-0.986	0.011*
ALT (U/L)	1.001	1.001-1.002	0.001**	1.001	1.000-1.002	0.020*
BUN (mmol/L)	1.135	1.103-1.169	<0.001***	1.066	1.005-1.130	0.033*
Creatinine (μmol/L)	1.003	1.002-1.005	<0.001***	1.001	0.998-1.003	0.638
Hb (g/L)	0.970	0.961-0.978	<0.001***	0.991	0.978-1.003	0.133
TC (mg/dl)	0.991	0.985-0.996	0.001***	1.002	0.996-1.008	0.564
SBP (mmHg)	0.982	0.972-0.992	<0.001***	0.983	0.972-0.995	0.005**
UA (μmol/L)	1.003	1.002-1.004	<0.001***	0.999	0.997-1.001	0.389
WBC (10⁹/L)	1.083	1.043-1.124	<0.001***	1.070	1.022-1.120	0.004**
TG/HDL-C	1.075	1.000-1.155	0.050	1.167	1.062-1.282	0.001**

Figure 1

Figure 1. Expression of TyG and TG/HDL-C in no-death and death groups of AMI, STEMI and NSTEMI.

(A) Independent-sample T-test was used for comparison of TyG conforming to normality. The TyG index was significantly increased in death groups compared to no-death groups in AMI (8.86±0.59 vs 9.22±0.97, P=0.001), STEMI (8.90±0.60 vs 9.24±0.95, P=0.024) and NSTEMI (8.81±0.58 vs 9.19±1.00, P=0.022) patients. (B) Mann-Whitney U method was used for comparison of TG/HDL-C not conforming to normality. TG/HDL-C was not significantly increased in the death group of AMI patients (P=0.588), as well as STEMI (P=0.961) and NSTEMI (P=0.456) subgroups. Data was presented by mean with SD (TyG) and median with range (TG/HDL-C). *P<0.05, #P>0.05.
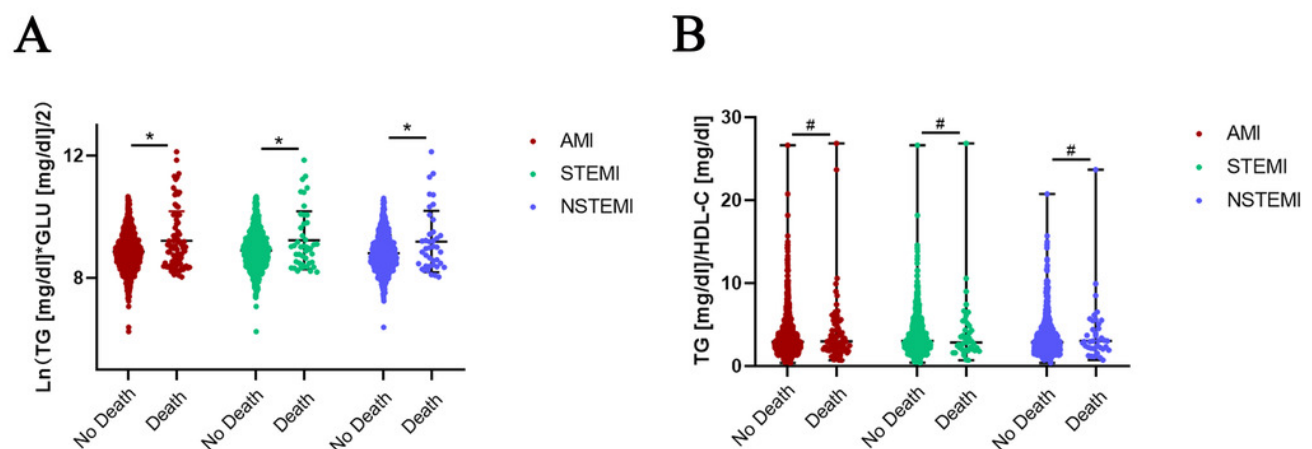
Figure 2

Figure 2. Forest plots of TyG and TG/HDL-C in predicting in-hospital death in AMI without diabetes.

(A) The results showed that after adjusting age (OR=1.069, 95%CI=1.042-1.097, P<0.001), ALB (OR=0.926, 95%CI=0.879-0.976, P=0.004), ALT (OR=1.001, 95%CI=1.000-1.002, P=0.028), BUN (OR=1.063, 95%CI=1.005-1.123, P=0.031), SBP (OR= 0.982, 95%CI = 0.971-0.993, P = 0.002) and WBC (OR=1.060, 95%CI =1.012-1.111, P=0.014), TyG (OR=3.106, 95%CI=2.122-4.547, P<0.001) was an important factor to predict the prognosis of AMI. (B) After adjusting age (OR=1.069, 95%CI=1.041-1.098, P<0.001), ALB (OR=0.935, 95%CI=0.887-0.986, P=0.011), ALT (OR=1.001, 95%CI=1.000-1.002, P=0.020), BUN (OR=1.066, 95%CI=1.005-1.130, P=0.033), SBP (OR=0.984, 95%CI =0.972-0.995, P=0.005) and WBC (OR=1.070, 95%CI =1.022-1.120, P=0.004), TG/HDL-C (OR=1.167, 95%CI=1.062-1.282, P=0.001) was an important factor to predict the prognosis of AMI.