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The Prognostic Value of the Triglyceride-Glucose Index in Forecasting Ten-Year Major Adverse Cardiovascular Events in Non-Diabetic Patients with Acute Myocardial Infarction Undergoing Percutaneous Coronary Intervention

Perkütan Koroner Girişim Uygulanan Akut Miyokard Enfarktüslü Diyabetik Olmayan Hastalarda On Yıllık Majör İstenmeyen Kardiyovasküler Olayları Öngörmede Trigliserit-Glukoz İndeksinin Değeri

ABSTRACT

Objective: This study aimed to explore the association between the triglyceride-glucose (TyG) index and major adverse cardiovascular events (MACE) over a ten-year period in non-diabetic patients with acute myocardial infarction (MI) undergoing primary percutaneous coronary intervention (PCI).

Methods: We included 375 consecutive non-diabetic patients presenting with acute MI who underwent primary PCI. The TyG index was calculated and patients were divided based on a cut-off value of ≥ 8.84 into high and low TyG index groups. The incidence of MACE, including all-cause mortality, target vessel revascularization, reinfarction, and rehospitalization for heart failure, was assessed over 10 years.

Results: Over the next 10 years, patients who underwent PCI for acute MI experienced a significantly higher incidence of MACE in the group with a high TyG index (≥ 8.84) (*P* = 0.004). Multivariable analysis revealed that the TyG index independently predicted MACE in these patients [odds ratio = 1.64; 95% confidence interval (CI): 1.22-2.21; *P* = 0.002]. Analysis of the receiver operating characteristic curve indicated that the TyG index effectively predicted MACE in patients with acute MI following PCI, with an area under the curve of 0.562 (95% CI: 0.503-0.621; *P* = 0.038).

Conclusion: This study established a correlation between high TyG index levels and an elevated risk of MACE in non-diabetic patients with acute MI. The findings suggest that the TyG index could be a reliable indicator of clinical outcomes for non-diabetic acute MI patients undergoing PCI.

Keywords: Acute myocardial infarction, major adverse cardiovascular events, percutaneous coronary intervention, triglyceride-glucose index

ÖZET

Amaç: Primer perkütan koroner girişim (PKG) uygulanan, diyabetik olmayan akut miyokard enfarktüslü (MI) hastalarda 10 yıllık takipte trigliserit glikoz (TyG) indeksi ile majör istenmeyen kardiyovasküler olay (MACE) insidansı arasındaki ilişkiyi araştırmayı amaçladık.

Yöntem: Akut MI ile başvuran ve primer PKG uygulanan toplam 375 ardışık diyabetik olmayan hasta çalışmaya dahil edildi. TyG indeksi değerlendirildi. TyG indeksi cut-off değerine göre ≥ 8,84 olanlar yüksek TyG indeksi grubu olarak kabul edildi. MACE için klinik sonuçlar 10 yıllık takiplerde değerlendirildi. MACE, tüm nedenlere bağlı ölüm, tekrarlayan MI, hedef damar revaskülarizasyonu ve kalp yetersizliği nedeniyle yeniden hastaneye yatış olarak tanımlandı.

Bulgular: Akut MI ile gelen PKG yapılan hastalarda sonraki 10 yıl içinde MACE insidansı, yüksek (≥ 8.84) TyG indeks grubunda anlamlı olarak daha yüksekti (*P* = 0,004). Çok değişkenli analizde TyG indeksi, akut MI hastalarında MACE için bağımsız öngördürücü olarak saptandı. (Odds oranı = 1,64; %95 GA: 1,22–2,21; *P* = 0,002). 'Receiver operating characteristic' eğrisi analizine göre PKG sonrası akut MI hastalarında TyG indeksi MACE oluşumunu tahmin edebilmiştir (MACE için eğri değerinin altındaki alan 0,562; %95 GA: 0,503-0,621; *P* = 0,038).

Sonuç: Bu çalışma, diyabetik olmayan akut MI hastalarında yüksek TyG indeks seviyeleri ile artmış MACE riski arasında bir ilişki olduğunu ve TyG indeksinin, diyabeti olmayan PCI uygulanan akut MI hastalarında klinik sonuçların geçerli bir göstergesi olabileceğini göstermiştir.

Anahtar Kelimeler: Akut miyokard enfarktüsü, majör istenmeyen kardiyovasküler olaylar, perkütan koroner girişim, trigliserit-glikoz indeksi

ORIGINAL ARTICLE
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Acute myocardial infarction (MI) represents the most critical manifestation of atherosclerotic cardiovascular disease and poses a significant socioeconomic challenge due to its substantial burden. Despite significant advancements in the prognosis of acute MI patients, including enhanced medical treatments and the adoption of modern revascularization techniques like percutaneous coronary intervention (PCI), the incidence of cardiovascular events remains high among this group.[1,2] Consequently, identifying risk factors in this specific group is crucial for the development of new therapeutic targets, a vital step for clinical progress.

Insulin resistance (IR) and the frequency of metabolic abnormalities, which have risen alongside improved living standards,[3,4] are substantial risk factors for atherosclerotic cardiovascular diseases and major adverse cardiovascular outcomes (MACE).[5-7]

Although the hyperinsulinemic-euglycemic clamp is the gold standard for assessing IR, its complexity and high cost limit its practical use in clinical settings.[8,9] Homeostatic model assessment for insulin resistance (HOMA-IR), another method of determining IR, is expensive because it relies on plasma insulin testing and is not available in most laboratories. It has been shown that the triglyceride glucose (TyG) index, calculated using the equation ln [fasting triglycerides (TGs, mg/dL) × fasting plasma glucose (FPG, mg/dL)/2], correlates significantly with both the hyperinsulinemic-euglycemic clamp test and HOMA-IR.[10,11] Studies have demonstrated that the TyG index is associated with the occurrence of cardiovascular diseases and adverse cardiac outcomes in diabetic patients.[12-15] However, there is limited data on the TyG index's effectiveness in predicting long-term MACE in non-diabetic patients with acute MI. This study explores the TyG index's predictive value for long-term MACE in non-diabetic patients who present with acute MI and undergo PCI.

Materials and Methods

Study Population

For this retrospective study, the minimum number of patients required to achieve an effect size of 0.2 with 90% power was determined, resulting in the inclusion of 375 acute MI patients. Inclusion criteria included non-diabetic patients presenting with acute MI and undergoing PCI within 12 hours of symptom onset. Exclusion criteria encompassed a diagnosis of diabetes or prediabetes, use of oral antidiabetic medication or insulin, use of triglyceride-lowering (TG-lowering) medication, severe liver or kidney disease, a body mass index over 45 kg/m^2, or insufficient clinical data. Acute MI was defined as ST-segment elevation myocardial infarction (STEMI) and high-risk non-ST-segment elevation myocardial infarction (NSTEMI) in patients who underwent percutaneous coronary intervention due to hemodynamic instability, recurrent or refractory chest pain despite medical therapy, life-threatening arrhythmias, and recurrent dynamic ST segment or T wave changes. This study adhered to the Declaration of Helsinki, and informed consent was obtained from all participants. The study was approved by the Karabük University Ethics Committee (Approval Number: 2022/1059, Date: September 29, 2022).

Laboratory Analyses

Blood samples were collected from peripheral veins at the time of acute MI diagnosis. A complete blood count and standard biochemical parameters were assessed. TG and fasting plasma glucose (FPG) concentrations were measured in the first blood samples taken after for a minimum of 12 hours of fasting during hospitalization. The TyG index was calculated using the formula: TyG index = ln (fasting TGs × FPG/2) based on the laboratory data obtained.

Angiographic Procedures

Coronary angiographic procedures were conducted using the Seldinger technique through either the femoral or radial arteries. Prior to angiography, all patients received a 300 mg loading dose of acetylsalicylic acid, P2Y12 inhibitors, and a standard dose of unfractionated heparin (50-70 U/kg). The administration of glycoprotein IIb/IIIa receptor blockers, such as tirofiban, was at the operator's discretion and not routinely used. Two cardiologists, who were unaware of the patients' medical histories, visually analyzed the angiographic data. The thrombolysis in myocardial infarction (TIMI) flow grade was assessed as previously described.[16] Success of the intervention was defined as achieving a TIMI 3 flow and reducing the stenosis in the infarct-related artery (IRA) to less than 30% post-primary angioplasty.

Clinical Follow-Up and Study Outcomes

Hospital records were used to collect patients' clinical, laboratory, and demographic data. Survival information was sourced from digital national databases. Data on readmissions and adverse cardiac events in surviving patients were gathered through telephone interviews with the patient or their relatives. The clinical outcomes associated with MACE were assessed over a 10-year period. MACE included all-cause mortality, target vessel revascularization (TVR), reinfarction, and hospital readmission due to heart failure. Reinfarction was identified based on the criteria set forth in the fourth universal MI guidelines.[17] TVR was characterized as the necessity for coronary artery bypass surgery or PCI due to narrowing or re-occlusion of the IRA.

Statistical Analysis

The statistical analysis was conducted using the IBM Statistical Package for the Social Sciences (SPSS) program (IBM SPSS Statistics for Windows, Version 21.0, IBM Corp., Armonk, New York, USA). Continuous variables with normal distribution were presented as mean (± standard deviation [SD]), whereas those with non-normal distribution were presented as the median (interquartile range). Categorical data were expressed in numbers and percentages. Univariate and multivariate logistic regression analyses were used to identify independent predictors of MACE,

ABBREVIATIONS

CVD	Cardiovascular disease
FPG	Fasting plasma glucose
HDL-C	High-density lipoprotein cholesterol
HOMA-IR	Homeostatic model assessment for insulin resistance
IR	Insulin resistance
IRA	Infarct-related artery
MACE	Major adverse cardiovascular events
MI	Myocardial infarction
NSTEMI	Non-STsegment elevation myocardial infarction
PCI	Percutaneous coronary intervention
ROC	Receiver operating characteristic
TC	Total cholesterol
TIMI	Thrombolysis in myocardial infarction
TVR	Target vessel revascularization
TyG	Triglyceride glucose

Table 1. Demographics and Baseline Clinical Characteristics

Variables	Low TyG Index (n = 209)	High TyG Index (n = 166)	*P*
Age, Years	55.1 ± 12.1	53.5 ± 11.7	0.187
Male	174 (83.3)	139 (83.7)	0.901
SBP, mmHg	136.9 ± 81.7	139.5 ± 32.2	0.881
DBP, mmHg	80.6 ± 19.8	84.4 ± 19.5	0.096
Heart Rate, bpm	78.1 ± 16.9	78.1 ± 19.5	0.993
Killip Class > 1	10 (4.8)	12 (7.3)	0.316
Hospital Stay, days	3.11 ± 0.83	3.5 ± 0.92	**<0.001**
Previous History			
Smoking	254 (75.1)	126 (75.9)	0.861
Hypertension	62 (29.7)	45 (27.1)	0.586
Hyperlipidemia	23 (11.0)	26 (16.1)	0.212
Myocardial infarction	31 (14.8)	24 (14.5)	0.919
PCI	30 (14.4)	21 (12.7)	0.633
Coronary Angiography			
Infarct-Related Artery			0.769
LAD	100 (47.8)	77 (44.6)	
CX	26 (12.4)	20 (12.0)	
RCA	83 (39.7)	72 (43.4)	
Diseased Vessels			0.780
1-Vessel	102 (48.8)	77 (46.4)	
2-Vessel	63 (30.1)	54 (32.5)	
3-Vessel	44 (21.1)	35 (21.1)	
Initial TIMI Grade 0-1	188 (90.0)	151 (91.0)	0.741
Final TIMI Grade 3	192 (91.9)	157 (94.6)	0.304
Direct Stenting	43 (20.6)	46 (27.7)	0.107
Laboratory Findings			
White Blood Cells, 10^9/L	13.1 ± 8.3	13.0 ± 3.6	0.843
Hemoglobin, g/L	14.5 ± 1.81	14.5 ± 1.57	0.701
Creatinine, mg/dL	0.93 ± 0.48	0.95 ± 0.26	0.585
Troponin I, ng/mL	12.8 ± 12.4	12.7 ± 32.7	0.981
LDL-C, mg/dL	124.6 ± 32.9	146.0 ± 36.0	**<0.001**
HDL-C, mg/dL	43.4 ± 11.5	38.0 ± 8.6	**<0.001**
Total Cholesterol, mg/dL	185.5 ± 39.5	211.3 ± 44.3	**<0.001**
Triglycerides, mg/dL	76.0 ± 28.8	177.1 ± 76.1	**<0.001**
FPG, mg/dL	99.1 ± 10.6	102 ± 12.4	**0.016**

Data are presented as mean ± SD or number (%). CX, Circumflex; DBP, Diastolic Blood Pressure; FPG, Fasting Plasma Glucose; HDL-C, High-Density Lipoprotein Cholesterol; LAD, Left Anterior Descending Coronary Artery; LDL-C, Low-Density Lipoprotein Cholesterol; PCI, Percutaneous Coronary Intervention; RCA, Right Coronary Artery; SBP, Systolic Blood Pressure; TIMI, Thrombolysis in Myocardial Infarction.

with the results reported as 95% confidence intervals (CI) and odds ratios (OR). The Kaplan-Meier curves demonstrated the survival rate without MACE, while the predictive value of the TyG index for MACE was illustrated using receiver operating characteristic (ROC) curves. All tests were two-tailed, and a p-value of less than 0.05 was considered to indicate statistical significance.

Results

A total of 375 patients who met the inclusion criteria were initially categorized into two groups based on their TyG index levels. Subsequently, these patients were also divided into two groups based on the presence of MACE. Demographic and basic clinical characteristics of the groups according to the TyG index are detailed in Table 1, showing statistically significant differences in FPG, TG, total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), and low-density lipoprotein cholesterol (LDL-C) values between the groups (all $P < 0.05$). No statistically significant differences were found for other parameters.

Demographic and baseline clinical data for groups with and without MACE are presented in Table 2. The group with MACE

Table 2. Baseline Characteristics of the MACE and MACE-Free Groups

Variables	MACE-Free Group (n = 207)	MACE Group (n = 168)	*P*
Age, Years	51.1 ± 9.7	58.4 ± 13.2	**<0.001**
Male	175 (84.5)	138 (82.1)	0.534
SBP, mmHg	132.8 ± 28.8	132.3 ± 32.9	0.882
DBP, mmHg	82.4 ± 19.7	81.4 ± 19.1	0.603
Heart Rate, bpm	76.8 ± 16.6	79.8 ± 19.7	0.107
Killip Class > 1	6 (2.9)	16 (9.5)	**0.007**
Hospital Stay	3.13 ± 0.67	3.46 ± 1.07	**<0.001**
Previous History			
Smoking	174 (84.1)	109 (64.9)	**<0.001**
Hypertension	48 (23.2)	59 (35.1)	**0.011**
Hyperlipidemia	25 (12.1)	24 (14.3)	0.479
Myocardial Infarction	24 (11.6)	34 (20.2)	**0.021**
PCI	20 (9.7)	31 (18.5)	**0.014**
Coronary Angiography			
Infarct-Related Artery			0.168
LAD	96 (46.4)	78 (46.4)	
CX	31 (15.0)	15 (8.9)	
RCA	80 (38.6)	75 (44.6)	
Diseased Vessels			**0.028**
1-Vessel	102 (49.8)	62 (36.7)	
2-Vessel	65 (31.7)	48 (28.3)	
3-Vessel	40 (19.5)	58 (35.0)	
Initial TMI Grade 0-1	184 (88.9)	155 (92.3)	0.270
Final TIMI Grade 3	200 (96.6)	149 (88.7)	**0.003**
Direct Stenting	50 (24.2)	39 (23.2)	0.831
Laboratory Findings			
White Blood Cells, 10^9/L	12.7 ± 3.6	13.4 ± 9.1	0.284
Hemoglobin, g/L	14.5 ± 1.81	14.5 ± 1.60	0.748
Creatinine, mg/dL	0.92 ± 0.44	0.96 ± 0.34	0.585
Troponin I, ng/mL	10.5 ± 11.3	13.2 ± 12.8	**0.032**
LDL-C, mg/dL	136.1 ± 35.7	131.9 ± 36.1	0.258
HDL-C, mg/dL	39.9 ± 10.5	41.1 ± 9.4	0.255
Total Cholesterol, mg/dL	199.2 ± 43.1	199.4 ± 44.6	0.291
Triglyceride, mg/dL	125.5 ± 80.4	116.1 ± 66.1	0.235
FPG, mg/dL	99.1 ± 10.8	100.9 ± 12.3	0.399
TyG Index	8.86 ± 0.70	9.09 ± 0.89	**0.004**

Data are presented as mean ± SD or number (%). CX, Circumflex; DBP, Diastolic Blood Pressure; FPG, Fasting Plasma Glucose; HDL-C, High-Density Lipoprotein Cholesterol; LAD, Left Anterior Descending Coronary Artery; LDL-C, Low-Density Lipoprotein Cholesterol; PCI, Percutaneous Coronary Intervention; RCA, Right Coronary Artery; SBP, Systolic Blood Pressure; TIMI, Thrombolysis in Myocardial Infarction; TyG, Triglyceride-Glucose Index.

showed higher incidences of a Killip class greater than 1, smoking history, hypertension, MI, and PCI compared to the MACE-free group (all $P < 0.05$). The final TIMI-3 flow grade was lower in the MACE group than in the MACE-free group. Significant differences were also observed between the MACE and MACE-free groups in terms of age, troponin levels, and TyG index.

At the 10-year follow-up, the observed rates in the overall population were as follows: all-cause mortality at 29.8% (n = 112), reinfarction at 16.8% (n = 63), TVR at 10.9% (n = 41), and rehospitalization for heart failure rate at 31.2% (n = 117) in the overall population. The 10-year all-cause mortality rates were similar between the two groups (High TyG index: 28.9% vs. Low TyG index: 30.6%; P = 0.720). Although TVR and reinfarction occurred more frequently in the high TyG index group, these

Table 3. Multivariable Regression Analysis for Predictors of 10-Year MACE

Variables	Odds Ratio	95% CI	*P*
Age (per 1 year)	1.055	[1.03, 1.07]	**<0.001**
PCI History	2.61	[1.32, 5.15]	**0.006**
Killip > 1	3.69	[1.08, 12.53]	**0.036**
High TYG Index	1.64	[1.22, 2.21]	**0.002**
Post TIMI < 3	3.36	[1.21, 9.37]	**0.020**

CI, Confidence Interval; OR, Odds Ratio; PCI, Percutaneous Coronary Intervention; TIMI, Thrombolysis in Myocardial Infarction; TyG, Triglyceride-Glucose Index.

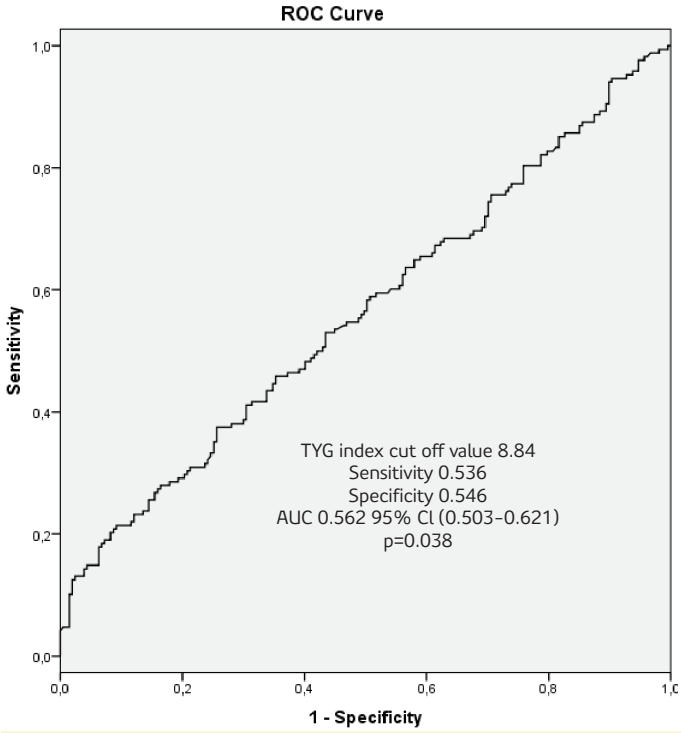

Figure 1. Receiver operating characteristics curve analysis for determining the predictive value of the TyG index for 10-year MACE incidence.

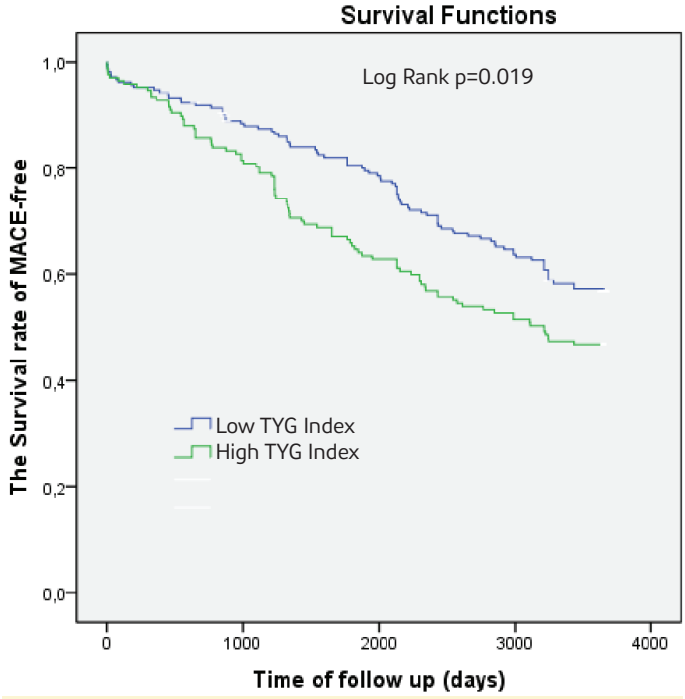

Figure 2. MACE-free survival curve showing the follow-up outcomes of two groups.

differences did not reach statistical significance (High TyG index: 16.9% vs. Low TyG index: 11.0%, *P* = 0.100; and High TyG index: 20.5% vs. Low TyG index: 13.9%, *P* = 0.089, respectively). The primary distinction between the two groups was evident in the rehospitalization rates for heart failure (High TyG index: 37.3% vs. Low TyG index: 26.3%; *P* = 0.022).

The univariate and multivariate analyses that identify independent predictors of 10-year MACE in non-diabetic patients with acute MI who underwent PCI are presented in Table 3. Since the TyG index calculation includes FPG and TG, these variables were excluded from both the univariate and multivariate analyses. The multivariate analysis revealed that age, PCI history, a Killip class greater than 1, TyG index, and a post-procedure TIMI less than 3 were independent predictors of MACE in non-diabetic patients with acute MI who underwent PCI (all *P* < 0.05).

Figure 1 displays the ROC curves for the TyG index's ability to predict MACE in non-diabetic acute MI patients undergoing PCI. The area under the curve (AUC) for the TyG index, indicating the development of MACE in non-diabetic acute MI patients undergoing PCI, was 0.562 (95% CI 0.503-0.621; *P* = 0.038).

Figure 2 displays the follow-up of TyG index groups without MACE (MACE-free) using the Kaplan-Meier curve. In non-diabetic patients who presented with acute MI and underwent PCI, the incidence of MACE was significantly higher within 10 years post-PCI in the group with a high TyG index (*P* = 0.019).

Discussion

Our study aimed to identify independent predictors of 10-year MACE in non-diabetic acute MI patients undergoing PCI, with a particular focus on the TyG index. The results can be summarized as follows: (1) The TyG index, with a value of 1.64 (95% CI 1.22-2.21; *P* = 0.002), was significantly associated with an increased risk of MACE at 10 years in non-diabetic acute MI patients after PCI; (2) The ROC curve analysis demonstrated that the TyG index has a moderate predictive value for MACE in this patient group.

IR is defined as a diminished biological response to insulin, which disrupts glucose metabolism and results in chronic hyperglycemia. This condition leads to inflammation and oxidative stress, causing cellular damage. Furthermore, IR may impact lipid metabolism, leading to dyslipidemia, and plays a significant role in the development of cardiovascular disease (CVD). It promotes the formation of atheroma plaque, ventricular hypertrophy, and diastolic abnormalities.[18] There is a well-established strong correlation between IR and increased risk of CVD.[19] Research by Bonora et al.[20] demonstrated the link between CVD and IR independent of other atherosclerosis risk factors. Similarly, Eddy et al.[21] highlighted IR's critical role in the development of coronary artery disease. Moreover, IR is known to affect the prognosis of patients with existing cardiovascular conditions.[22,23] Consequently, the quantitative assessment of IR may improve prognosis by aiding in risk stratification and informing the development of treatment strategies for individuals at risk of or diagnosed with cardiovascular disease.

The hyperinsulinemic-euglycemic clamp is considered the gold standard for evaluating IR.[8] Nevertheless, the use of hyperinsulinemic-euglycemic clamping in clinical practice is difficult due to its time-consuming nature, high cost, and procedural complexity.[9] Another method for evaluating IR is the HOMA-IR. Since it is determined using fasting insulin and glucose values, and because fasting insulin is not typically measured in routine laboratory tests, especially in patients without diabetes, it is considered unsuitable for clinical practice.[10] The TyG index, calculated from fasting TG and glucose, serves as an alternative marker for IR. The calculation of the TyG index is straightforward and practical for clinical use because it relies on commonly used parameters and does not require specialized techniques. Furthermore, the TyG index has demonstrated a significant correlation with both the hyperinsulinemic-euglycemic clamp test and the HOMA-IR.[10,11,24]

Previous studies have demonstrated the relationship between the incidence of diabetes, prediabetic status, and the TyG index. The TyG index is also recognized for its role in the early diagnosis of individuals prone to diabetes and prediabetes.[25,26] Additionally, the TyG index has been linked to cardiovascular diseases independently of diabetes and other cardiovascular risk factors. This indicates that assessing the TyG index could play a crucial role in the early diagnosis of individuals at risk for developing cardiovascular disease.[12,27] Furthermore, the TyG index has been shown to have a significant role in predicting poor prognosis in patients with cardiovascular disease. The relationship between the TyG index and recurrent adverse cardiovascular events in patients with stable coronary artery disease, regardless of their diabetic status, is well-established. Additionally, a significant correlation has been demonstrated between the TyG index and recurrent adverse cardiovascular events in patients with ACS, whether they have diabetes or not.[28-30]

Additionally, in studies focused exclusively on non-diabetic patients, a significant association between the TyG index and MACE was demonstrated in a study involving 1,510 patients. These patients presented with ACS and underwent primary PCI, with follow-ups conducted over a 4-year period.[31] However, another study that included 5,489 patients with stable coronary disease who had stent implantations found no significant association between the TyG index and MACE over a 2-year follow-up period.[32] Similarly, no significant relationship was found between the TyG index and MACE in a 1-year follow-up of 1,340 patients presenting with acute MI.[33] The impact of the TyG index on 10-year MACE outcomes in non-diabetic patients with acute MI has not been conclusively evaluated. In our study, we assessed the impact of the TyG index on the incidence of MACE over a 10-year period in non-diabetic patients who presented with acute MI and underwent PCI. We discovered a significant association between the TyG index and the risk of MACE in these non-diabetic patients during the 10-year follow-up after PCI.

The association between the TyG index and cardiovascular disease can be explained as follows: the TyG index has been proven to correlate effectively with IR in liver and adipose cells, and reflects overall body IR.[34] IR is linked to several physiological disruptions, including inflammatory responses, oxidative stress, disorders in coagulation, compromised myocardial reperfusion, and cardiovascular remodeling. All these elements mediate the development of cardiovascular disease and adversely affect prognosis.[35,36] However, these observations do not confirm causality. Randomized controlled studies are necessary to definitively prove this relationship.

Our study faced several limitations: 1) It had relatively low power due to being a single-center and retrospective study. 2) There were insufficient repeated measurements of the TyG index post-discharge, preventing further analysis. 3) There was a lack of data on hyperinsulinemic-euglycemic clamp and HOMA-IR, thus comparisons with the TyG index were not possible.

Conclusion

An elevated TyG index is a long-term predictor of MACE in non-diabetic patients who are undergoing PCI and have been diagnosed with acute MI. Consequently, assessing IR using the TyG index in this demographic may contribute to the risk classification and aid in formulating targeted treatment approaches.

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Ethics Committee Approval: The study was approved by the Karabük University Ethics Committee (Approval Number: 2022/1059, Date: September 29, 2022).

Informed Consent: Informed consent was obtained from all participants.

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