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Incremental Value of the Triglyceride–Glucose Index Beyond Conventional Clinical Factors for SCORE2-Based Cardiovascular Risk Stratification in Patients with Dyslipidemia

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Abstract

Objective and Aim

To investigate the associations of the triglyceride–glucose (TyG) index, systemic immune-inflammation index (SII), and systemic inflammation response index (SIRI) with SCORE2-based cardiovascular risk and to evaluate their incremental predictive value beyond conventional clinical factors in patients with dyslipidemia.

Materials and Methods

In this retrospective cross-sectional study, 111 adults with dyslipidemia managed in a primary care setting were included. Cardiovascular risk was assessed using the SCORE2 algorithm, and participants were categorized into low–moderate and high–very high-risk groups. The TyG index, SII, and SIRI were calculated using routine laboratory parameters. Binary logistic regression models were constructed to determine the incremental contribution of these biomarkers to cardiovascular risk stratification. Model performance was evaluated using receiver operating characteristic (ROC) curve analysis.

Results

Patients with high–very high cardiovascular risk had significantly higher TyG index values compared with those at low–moderate risk (9.00 ± 0.64 vs. 8.66 ± 0.57, p=0.014). No significant differences were observed for SII (p=0.405) or SIRI (p=0.908). In multivariable analysis, TyG was independently associated with high cardiovascular risk (OR=2.21, 95% CI: 1.08–4.52, p=0.030), whereas SII and SIRI were not. The addition of TyG to the clinical model improved discrimination for high cardiovascular risk (AUC: 0.639 to 0.689), while subsequent inclusion of SII and SIRI resulted in only marginal improvement (AUC: 0.692).

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Conclusion

The TyG index was independently associated with SCORE2-based cardiovascular risk and provided incremental predictive value beyond conventional clinical variables in patients with dyslipidemia. In contrast, SII and SIRI showed limited utility for cardiovascular risk stratification. Larger prospective studies are warranted to confirm these findings.

Keywords: Dyslipidemia, Triglyceride-Glucose Index, Systemic Immune-Inflammation Index, Cardiovascular Risk.

1. Introduction

Cardiovascular diseases (CVDs) remain among the leading causes of mortality and morbidity worldwide and impose a substantial burden on healthcare systems. In addition to traditional risk factors such as age, hypertension, diabetes mellitus, and smoking, dyslipidemia is a major determinant in the development of atherosclerotic cardiovascular disease. Therefore, the early identification of high-risk individuals and the implementation of appropriate preventive strategies are of paramount importance. One of the most widely used tools for cardiovascular risk assessment is the Systematic Coronary Risk Evaluation 2 (SCORE2) algorithm developed by the European Society of Cardiology.[1] However, SCORE2 primarily relies on conventional risk factors and does not directly reflect the underlying metabolic and inflammatory processes involved in atherosclerosis.[2]

Insulin resistance is one of the key mechanisms contributing to the development of atherosclerosis. In recent years, the triglyceride-glucose index (TyG) has emerged as a practical surrogate marker of insulin resistance. Calculated from fasting plasma glucose and triglyceride levels, the TyG index is a simple and cost-effective parameter. Previous studies have demonstrated that an elevated TyG index is associated with coronary artery disease, major adverse cardiovascular events, and cardiovascular mortality.[3] Particularly in high-risk populations, such as individuals with dyslipidemia or metabolic syndrome, the TyG index has been reported to provide valuable information for cardiovascular risk stratification.[4,5]

Chronic inflammation also plays a crucial role in the pathogenesis of atherosclerosis. Endothelial dysfunction, oxidative stress, and inflammatory cell activation accelerate the formation and progression of atherosclerotic plaques. Consequently, inflammatory markers derived from routine complete blood count parameters have gained increasing attention in cardiovascular risk assessment. Among these, the systemic immune-inflammation index (SII) and the systemic inflammation response index (SIRI) are novel biomarkers calculated using combinations of neutrophil, platelet, monocyte, and lymphocyte counts. Elevated SII and SIRI levels have been associated with coronary artery disease, heart failure, and cardiovascular mortality in several studies.[6]

In patients with dyslipidemia, the atherosclerotic process is influenced by both metabolic and inflammatory mechanisms. Therefore, the combined evaluation of the TyG index, reflecting insulin resistance, and SII and SIRI, reflecting systemic inflammatory burden, may provide a more comprehensive assessment of cardiovascular risk. Nevertheless, studies investigating the relationship between these biomarkers and cardiovascular risk estimated by the SCORE2 algorithm remain limited. In particular, evidence regarding this association in dyslipidemic patients managed in primary care settings is scarce.

The aim of the present study was to evaluate the association between the TyG index, SII, and SIRI and cardiovascular risk estimated using the SCORE2 algorithm in adult patients with dyslipidemia. Additionally, we sought to investigate the potential utility of these biomarkers in identifying individuals at high cardiovascular risk.

2. Materials and Method

Study Design and Population

This retrospective cross-sectional study was conducted using electronic medical records of patients registered at a family medicine unit. Adult patients (between 40-69 years) with a diagnosis of dyslipidemia who had complete demographic, biochemical, and hematological data available during the study period were included. No additional laboratory tests, interventions, or direct patient contact were performed for the purpose of the study. Only visits in which patients were clinically asymptomatic and presented for routine follow-up or screening purposes were considered. The required sample size was calculated based on the primary objective of evaluating the association between the TyG index, SII, SIRI, and SCORE2-based cardiovascular risk. Assuming a moderate correlation effect size of r=0.30, a two-tailed alpha level of 0.05, and 80% statistical power, the minimum required sample size was calculated as 84 patients. During the study period, 156 adult patients with dyslipidemia were identified. Patients with established atherosclerotic cardiovascular disease were excluded from the study, leaving 125 eligible patients. After excluding patients with missing demographic, clinical, biochemical, or hematological data required for SCORE2, the TyG index, SII, and SIRI calculations, a total of 111 patients were included in the final analysis.

Ethical Considerations

The study was conducted in accordance with the principles of the Declaration of Helsinki. Ethical approval was obtained from the Xxx Ethics Committee prior to study initiation. As the study was based exclusively on retrospective review of anonymized medical records, no direct patient contact or intervention was performed. Patient confidentiality was maintained throughout the study, and all data were analyzed anonymously.

Data Collection

Demographic characteristics, clinical information, and laboratory findings were obtained retrospectively from electronic medical records. The collected variables included age, sex, smoking status, systolic blood pressure, plasma glucose, triglycerides, total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), and complete blood count parameters. Hematological variables included neutrophil, lymphocyte, monocyte, and platelet counts. All laboratory measurements were obtained from routine clinical assessments and no additional tests were performed for the purpose of the study.

Calculation of the TyG index, SII and SIRI

The TyG index was calculated using the following formula: TyG = Ln [Triglyceride (mg/dL) × Glucose (mg/dL) / 2]. SII was calculated as: SII = Platelet count × Neutrophil count / Lymphocyte count. SIRI was calculated as: SIRI = Neutrophil count × Monocyte count / Lymphocyte count. These indices were calculated for all participants using laboratory values obtained from routine clinical practice.[7]

Assessment of Cardiovascular Risk

Cardiovascular risk was assessed using the SCORE2 algorithm recommended by the European Society of Cardiology. SCORE2 estimates the 10-year risk of first fatal and non-fatal cardiovascular events based on age, sex, smoking status, systolic blood pressure, total cholesterol, and HDL-C levels.[1] For each participant, SCORE2 risk was calculated according to current European guidelines. Patients were subsequently categorized according to their estimated cardiovascular risk levels. For comparative analyses, individuals classified as high cardiovascular risk were compared with those classified as low-to-moderate cardiovascular risk.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics. Continuous variables were presented as mean ± standard

deviation and median (interquartile range). Categorical variables were summarized as frequencies and percentages. Continuous variables were assessed for normality using the Shapiro–Wilk test. Comparisons between groups were performed using the independent samples t-test or Mann–Whitney U test for continuous variables, as appropriate. Categorical variables were compared using the chi-square test or Fisher's exact test. To identify independent predictors of high cardiovascular risk, binary logistic regression analysis was performed. The area under the curve (AUC), odds ratios (ORs) with 95% confidence intervals (CIs) were calculated. Receiver operating characteristic (ROC) curve analysis was conducted to evaluate the discriminative ability of TyG index, SII, and SIRI for identifying patients with high cardiovascular risk. A two-sided p value of <0.05 was considered statistically significant.

3. Results

The baseline demographic and clinical characteristics of the study population are presented in Table 1. The mean age of the participants was 57.14 ± 7.56 years, and 64.9% were female. Current smokers accounted for 20.7% of the cohort, whereas 34.2% had diabetes mellitus and 44.1% had hypertension. Lipid-lowering treatment was being used by 64.9% of patients. Regarding physical activity status, 74.8% of participants were physically inactive. The mean body mass index (BMI) was 31.01 ± 6.11 kg/m^2, indicating that the majority of patients were overweight or obese.

Laboratory characteristics and calculated biomarker indices are shown in Table 2. The mean total cholesterol concentration was 201.29 ± 42.73 mg/dL, while median triglyceride and fasting plasma glucose levels were 125 mg/dL and 95 mg/dL, respectively. The mean TyG index was 8.87 ± 0.62. The median SII and SIRI values were 445.71 and 0.75, respectively.

The distribution of SCORE2 cardiovascular risk categories is summarized in Table 3. Among the study population, 22 patients (19.8%) were classified as having low cardiovascular risk, 46 (41.4%) as moderate risk, 33 (29.7%) as high risk, and 10 (9.0%) as very high risk. Overall, 43 patients (38.7%) were categorized as having high-to-very high cardiovascular risk.

Comparisons between low-to-moderate risk and high-to-very high risk groups are presented in Table 4. Patients in the high-risk group were significantly older than those in the low-risk group (63 vs. 56 years, p<0.001). Male sex was more prevalent among high-risk individuals (48.8% vs. 26.5%, p=0.016), whereas the proportion of never-smokers was lower (46.5% vs. 72.1%, p=0.017). Diabetes mellitus was markedly more frequent in the high-risk group (60.5% vs. 17.6%, p<0.001). Systolic and diastolic blood pressure values were also significantly higher among high-risk patients (p<0.001 and p=0.019, respectively).

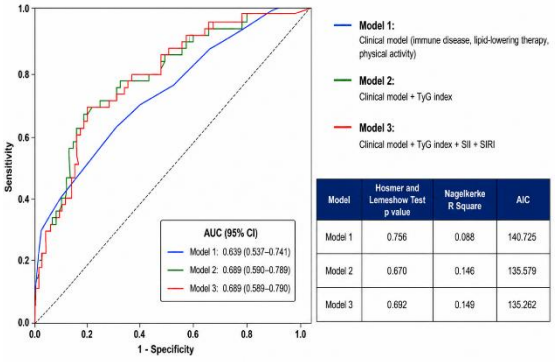

Figure 1: Receiver operating characteristic (ROC) curves of sequential prediction models for identifying patients with high-very high cardiovascular risk

Regarding biochemical findings, glucose levels were significantly elevated in the high-risk group (114 vs. 93 mg/dL, p<0.001). No statistically significant differences were observed for total cholesterol, LDL cholesterol, HDL cholesterol, triglycerides, or hematological parameters. Importantly, the TyG index was significantly higher among patients with high-to-very high cardiovascular risk compared with those with low-to-moderate risk (9.00 ± 0.64 vs. 8.66 ± 0.57, p=0.014). In contrast, SII and SIRI values did not differ significantly

between the groups (p=0.405 and p=0.908, respectively).

Table 1. Baseline Demographic and Clinical Characteristics of the Study Population

Variable	n (%)	Mean ± SD	Median (IQR)
Age (years)		57.14 ± 7.56	60.0 (52.5–63.0)
Gender			
Female	72 (64.9)		
Male	39 (35.1)		
Smoking status			
Current smoker	23 (20.7)		
Former smoker	19 (17.1)		
Never smoker	69 (62.2)		
Diabetes mellitus	38 (34.2)		
Hypertension	49 (44.1)		
Immune disease	33 (29.7)		
Lipid-lowering therapy	72 (64.9)		
Physical activity level			
Physical inactivity	83 (74.8)		
Low	6 (5.4)		
Moderate	16 (14.4)		
High	6 (5.4)		
Height (cm)		161.79 ± 9.06	161 (155–169)
Weight (kg)		80.49 ± 13.11	80 (73–87)
BMI (kg/m^2)		31.01 ± 6.11	29.2 (26.7–33.7)
Obesity Classification			
Normal	10 (10.8)		
Overweight	48 (43.2)		
Obesity Class 1	27 (24.3)		
Obesity Class 2	13 (11.7)		
Obesity Class 3	11 (9.9)		
Systolic blood pressure (mmHg)		132.32 ± 18.49	130 (120–144)
Diastolic blood pressure (mmHg)		80.27 ± 11.10	80 (70–89.5)

The results of multivariable logistic regression analyses are shown in Table 5. In Model 1, immune disease was inversely associated with high cardiovascular risk (OR=0.353, 95% CI: 0.139–0.900, p=0.029). After the addition of the TyG index in Model 2, TyG emerged as an independent predictor of high cardiovascular risk (OR=2.207, 95% CI: 1.084–4.492, p=0.029). The association remained significant after further adjustment for SII and SIRI in Model 3 (OR=2.209, 95% CI: 1.079–4.521, p=0.030). Neither SII nor SIRI independently predicted high cardiovascular risk (p=0.617 and p=0.961, respectively).

The ROC curve analyses were performed to evaluate the discriminative performance of the sequential prediction models for identifying patients with high-to-very high cardiovascular risk (Figure 1). The baseline clinical model, including immune disease status, lipid-lowering therapy, and physical activity level (Model 1), demonstrated modest discriminative ability, with AUC of 0.639. Following the addition of the TyG index (Model 2), the predictive performance improved, yielding AUC of 0.689. However, further incorporation of SII and SIRI into the model (Model 3) did not result in any additional improvement in discrimination, with AUC remaining unchanged at 0.689.

Table 2. Laboratory Characteristics and Calculated Biomarker Indices of the Study Population

Variable	Mean ± SD	Median (IQR)
Total cholesterol (mg/dL)	201.29 ± 42.73	195 (175–223)
Triglycerides (mg/dL)	153.79 ± 87.36	125 (96–199)
Glucose (mg/dL)	112.77 ± 51.22	95 (88–118)
LDL cholesterol (mg/dL)	129.62 ± 34.35	133 (107–149)
HDL cholesterol (mg/dL)	39.93 ± 11.31	39 (32–47)
White blood cell count (×10^3/μL)	7.62 ± 2.14	7.42 (6.00–8.95)
Platelet count (×10^3/μL)	274.76 ± 66.66	260 (227–311)
Neutrophil count (×10^3/μL)	4.40 ± 1.77	4.05 (3.17–5.13)
Monocyte count (×10^3/μL)	0.49 ± 0.27	0.45 (0.37–0.57)
Lymphocyte count (×10^3/μL)	2.49 ± 0.79	2.40 (1.96–2.84)
TyG index	8.87 ± 0.62	8.76 (8.41–9.32)

SII	542.50 ± 411.16	445.71 (306.13–635.10)
SIRI	1.08 ± 1.66	0.75 (0.48–1.15)

Discussion

In the present study, we investigated the associations of the TyG index, SII, and SIRI with SCORE2-based cardiovascular risk among patients with dyslipidemia. The principal findings of our study were as follows: (1) patients classified as having high-to-very high cardiovascular risk had significantly higher TyG index values than those with low-to-moderate risk; (2) SII and SIRI were not significantly associated with cardiovascular risk categories; (3) the addition of TyG to a clinical prediction model improved cardiovascular risk discrimination, increasing the AUC from 0.639 to 0.689; and (4) the subsequent inclusion of SII and SIRI provided no further improvement in model performance (AUC 0.689). These findings suggest that the TyG index may offer incremental value beyond conventional clinical variables for cardiovascular risk stratification in patients with dyslipidemia, whereas the additional contribution of inflammatory indices appears limited.

Table 3. Distribution of SCORE2-Based Cardiovascular Risk Categories Among Patients with Dyslipidemia

Variable	**n (%)**	**Mean ± SD**	**Median (IQR)**
SCORE2 (%)		10.24 ± 6.56	8.5 (5.6–15.1)
SCORE2 Risk Categories			
Low risk	22 (19.8)		
Moderate risk	46 (41.4)		
High risk	33 (29.7)		
Very high risk	10 (9.0)		

Our findings regarding the TyG index are consistent with a growing body of evidence demonstrating the relationship between insulin resistance and cardiovascular risk. The TyG index, a simple surrogate marker derived from fasting triglyceride and glucose levels, has been increasingly recognized as a useful predictor of adverse cardiovascular outcomes. Tao et al. reported that elevated TyG levels were associated with an increased risk of major adverse cardiovascular events among patients with acute coronary syndrome undergoing percutaneous coronary intervention.[3] Similarly, higher TyG values were independently associated with increased cardiovascular risk in population-based studies conducted by Kwak et al. and Chen et al..[4,5] Furthermore, Mavraganis et al. demonstrated that incorporating the TyG index into conventional risk assessment models improved cardiovascular risk stratification in community-based populations.[8] In addition, Li et al. supported the association between elevated TyG levels and subclinical atherosclerosis.[9] The present study extends these findings to asymptomatic patients with dyslipidemia followed in primary care settings and demonstrates that TyG is associated with SCORE2-based cardiovascular risk even after adjustment for potential confounders.

Table 4. Comparison of Clinical, Biochemical, and Inflammatory Parameters According to SCORE2 Cardiovascular Risk Categories

Variable	**Low–Moderate Risk (n=68)**	**High–Very High Risk (n=43)**	**p-value**
Age (years)	56 (52–61)	63 (58–65)	<0.001*
Female sex, n (%)	50 (73.5)	22 (51.2)	0.016**
Male sex, n (%)	18 (26.5)	21 (48.8)	
Never smoker, n (%)	49 (72.1)	20 (46.5)	0.017**
Diabetes mellitus, n (%)	12 (17.6)	26 (60.5)	<0.001**
Hypertension, n (%)	28 (41.2)	21 (48.8)	0.428**
Immune disease, n (%)	25 (36.8)	8 (18.6)	0.041**
Physical inactivity, n (%)	48 (70.6)	35 (81.4)	0.232**
Obesity, n (%)	32 (47.1)	19 (44.2)	0.767**
Height (cm)	160 (154–168)	162 (156–169)	0.481*
Weight (kg)	78 (72–87)	81 (74–87)	0.787*
BMI (kg/m^2)	29.3 (26.6–33.9)	29.0 (26.8–33.1)	0.622*

Systolic blood pressure (mmHg)	123 (120–135)	137 (125–155)	<0.001*
Diastolic blood pressure (mmHg)	80 (70–84)	84 (76–90)	0.019*
Glucose (mg/dL)	93 (86–108)	114 (95–145)	<0.001*
Total cholesterol (mg/dL)	197 ± 34.8	206 ± 53.0	0.323***
Triglycerides (mg/dL)	123 (94–198)	133 (102–201)	0.519*
LDL cholesterol (mg/dL)	128 ± 31.4	132 ± 38.9	0.572***
HDL cholesterol (mg/dL)	40 ± 11.2	37 ± 11.3	0.153***
White blood cell count ($\times10^3$/μL)	7.05 (6.0–8.8)	7.60 (6.3–9.0)	0.776*
Platelet count ($\times10^3$/μL)	274 ± 70.7	257 ± 58.4	0.132***
Neutrophil count ($\times10^3$/μL)	4.00 (3.17–5.12)	4.14 (3.31–5.30)	0.918*
Monocyte count ($\times10^3$/μL)	0.44 (0.36–0.56)	0.46 (0.38–0.57)	0.458*
Lymphocyte count ($\times10^3$/μL)	2.41 (1.95–2.80)	2.36 (2.01–2.92)	0.580*
TyG index	8.66 ± 0.57	9.00 ± 0.64	0.014***
SII	493 (329–672)	444 (346–671)	0.405*
SIRI	0.73 (0.51–1.02)	0.77 (0.57–1.03)	0.908*

*Mann Whitney U test, **Chi-square test, ***Student-T test

The observed improvement in predictive performance after adding the TyG index to the baseline clinical model further supports its clinical utility. In our study, the inclusion of TyG increased the AUC from 0.639 to 0.689, while logistic regression analyses showed that TyG remained an independent predictor of high cardiovascular risk. These findings suggest that TyG captures aspects of cardiometabolic dysfunction not fully reflected by traditional cardiovascular risk factors. Since SCORE2 was developed using conventional risk factors, including age, sex, smoking status, systolic blood pressure, and lipid parameters, the addition of an index reflecting insulin resistance may provide complementary information regarding the pathophysiological mechanisms underlying atherosclerosis.[1] This observation may be particularly relevant in patients with dyslipidemia, in whom insulin resistance frequently coexists and contributes to residual cardiovascular risk.

Table 5. Multivariable Logistic Regression Models for Predicting High Cardiovascular Risk

	B (SE)	Z	p	OR	95% CI
MODEL 1					
Immune disease	-1.040 (0.477)	-2.181	0.029	0.353	0.139–0.900
Lipid-lowering therapy	-0.366 (0.419)	-0.873	0.383	0.693	0.305–1.577
Physical activity	-0.748 (0.489)	-1.529	0.126	0.473	0.181–1.235
Constant	0.236 (0.384)	0.613	0.540	1.266	0.596–2.686
MODEL 2					
Immune disease	-0.958 (0.491)	-1.954	0.051	0.383	0.147–1.003
Lipid-lowering therapy	-0.117 (0.445)	-0.263	0.793	0.890	0.372–2.128
Physical activity	-0.896 (0.510)	-1.755	0.079	0.408	0.150–1.110
TyG index	0.792 (0.363)	2.183	0.029	2.207	1.084–4.492
Constant	-6.957 (3.318)	-2.097	0.036	0.001	0.000001–0.635
MODEL 3					
Immune disease	-0.902 (0.503)	-1.795	0.073	0.406	0.151–1.087
Lipid-lowering therapy	-0.090 (0.452)	-0.200	0.842	0.914	0.377–2.215
Physical	-0.866 (0.514)	-1.687	0.092	0.420	0.154–1.151

activity					
TyG index	0.792 (0.366)	2.168	0.030	2.209	1.079–4.521
SII	-0.000328 (0.000657)	-0.500	0.617	0.9997	0.998–1.001
SIRI	0.00724 (0.147)	0.049	0.961	1.007	0.755–1.344
Constant	-6.835 (3.359)	-2.035	0.042	0.001	0.000001–0.778

Contrary to our expectations, neither SII nor SIRI differed significantly between SCORE2 risk groups, nor did they improve the discriminative ability of the prediction models. Although previous studies have demonstrated associations between elevated SII and adverse cardiovascular outcomes, these investigations were predominantly conducted in patients with established cardiovascular disease, acute coronary syndromes, obesity, or diabetes mellitus. A systematic review and meta-analysis by Zhang et al. demonstrated that elevated SII levels were associated with cardiovascular disease and mortality.[6] Data from a national cohort study also indicated that SII may have predictive value for coronary heart.[10] Moreover, Agyeman et al. reported an association between higher SII values and cardiovascular disease risk in the general population.[11]

Regarding SIRI, Liu et al. demonstrated significant associations between SIRI and cardiovascular disease among adults with obesity.[7] Similarly, Wang et al. reported that elevated SIRI levels were associated with an increased risk of heart failure, whereas Wang et al. identified both SII and SIRI as independent risk factors for coronary heart disease in young adults.[10] In contrast, our study population consisted of asymptomatic individuals without established atherosclerotic cardiovascular disease who attended primary care facilities for routine follow-up and screening purposes. Therefore, the relatively lower inflammatory burden in this population may partly explain the absence of significant associations observed in our analyses.

Another explanation for these findings may be related to the nature of SCORE2 itself. SCORE2 estimates the 10-year risk of first fatal and non-fatal cardiovascular events in apparently healthy individuals without previous cardiovascular disease.[1,2] Unlike studies evaluating hard clinical endpoints such as myocardial infarction, heart failure, or cardiovascular mortality, our investigation assessed the relationship between inflammatory biomarkers and a risk prediction algorithm. Thus, inflammatory indices derived from complete blood count parameters may have limited value in differentiating risk categories before overt cardiovascular disease develops. This interpretation is supported by the findings of Bayraktar et al., who reported that SII and SIRI were more closely related to coronary artery disease severity than to cardiovascular risk estimation in otherwise stable populations.[12]

The present findings have important implications for primary care practice. Family physicians are uniquely positioned to identify high-risk individuals before the occurrence of cardiovascular events. The TyG index can be calculated easily from routinely obtained fasting triglyceride and glucose measurements without additional cost or laboratory testing. Therefore, its incorporation into cardiovascular risk assessment strategies may enhance the identification of dyslipidemic patients who require more intensive preventive interventions. In contrast, although SII and SIRI are inexpensive and widely available biomarkers, their routine use for SCORE2-based risk stratification in asymptomatic dyslipidemic patients does not appear to provide substantial clinical benefit based on the current findings.

Study of Limitations

This study has several limitations that should be considered when interpreting the findings. First, due to its retrospective and cross-sectional design, causal relationships between the investigated biomarkers and cardiovascular risk cannot be established. Second, the study was conducted in a single primary care center and included a relatively

limited sample size, which may restrict the generalizability of the results to other populations. Third, TyG, SII, and SIRI were calculated using a single set of laboratory measurements obtained from medical records and therefore may not fully reflect long-term metabolic and inflammatory status. Finally, although major clinical variables were included in the regression models, the potential influence of unmeasured confounding factors such as dietary habits, socioeconomic status, and medication adherence cannot be completely excluded.

Conclusion

In conclusion, the present study demonstrated that the TyG index was significantly associated with SCORE2-based cardiovascular risk among patients with dyslipidemia and provided incremental predictive value beyond conventional clinical variables. The addition of the TyG index to the clinical model improved cardiovascular risk discrimination. In contrast, the inclusion of the SII and SIRI resulted in only marginal improvements in model performance and did not independently predict high cardiovascular risk.

These findings suggest that the TyG index, a simple and inexpensive marker derived from routinely available laboratory parameters, may serve as a useful adjunct to conventional cardiovascular risk assessment in primary prevention settings. Given its ease of calculation and widespread availability, incorporation of the TyG index into cardiovascular risk evaluation strategies may help identify high-risk individuals more accurately among patients with dyslipidemia. However, larger prospective studies are required to determine whether the integration of the TyG index into established risk prediction algorithms can improve the prediction of future cardiovascular events and clinical outcomes.

Acknowledgements

None

Conflict of Interest

The authors declare no conflict of interest.

Abbreviations:

AUC: Area Under the Curve, **BMI:** Body Mass Index, **CI:** Confidence Interval, **CVD:** Cardiovascular Disease, **ESC:** European Society of Cardiology, **HDL-C:** High-Density Lipoprotein Cholesterol, **LDL-C:** Low-Density Lipoprotein Cholesterol, **OR:** Odds Ratio, **ROC:** Receiver Operating Characteristic, **SCORE2:** Systematic Coronary Risk Evaluation 2**, SII:** Systemic Immune-Inflammation Index, **SIRI:** Systemic Inflammation Response Index, **TyG:** Triglyceride–Glucose Index

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