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Triglyceride–Glucose (TyG) Index as a Predictor of Early Metabolic Alterations in a Tertiary Care Laboratory Population

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ABSTRACT

Background: The Triglyceride–Glucose (TyG) index, derived from fasting triglyceride and glucose levels, has emerged as a practical surrogate marker of insulin resistance. Growing evidence links TyG index with metabolic syndrome, type 2 diabetes mellitus, and cardiovascular disease; however, data from routine tertiary laboratory populations remain limited.
Objective: To evaluate the association between TyG index and early metabolic alterations in an adult tertiary care laboratory population.
Materials and Methods: A cross-sectional analytical study was conducted among 300 adults undergoing routine fasting investigations at a tertiary care center. TyG index was calculated as ln [(Triglycerides (mg/dL) × Fasting Blood Glucose (mg/dL)) / 2]. Associations between TyG index and metabolic parameters including body mass index (BMI), lipid profile, and glycemic indices were analyzed using Pearson correlation, one-way ANOVA, multiple linear regression, and receiver operating characteristic (ROC) curve analysis.
Results: The mean TyG index was 8.71 ± 0.62. TyG index showed moderate positive correlations with fasting blood glucose ($r = 0.401$), BMI ($r = 0.382$), total cholesterol ($r = 0.356$), and LDL cholesterol ($r = 0.341$), and a significant inverse correlation with HDL cholesterol ($r = –0.298$) (all $p < 0.001$). Individuals in the highest TyG tertile demonstrated significantly greater prevalence of moderate and high metabolic risk categories ($p < 0.001$). On multivariate regression analysis, BMI, LDL, and HDL emerged as independent predictors of TyG index ($R^2 = 0.42$). ROC curve analysis revealed good discriminatory performance of TyG index for identifying ≥2 metabolic abnormalities (AUC = 0.79).
Conclusion: The TyG index is significantly associated with adverse metabolic profiles and demonstrates good predictive capability for early metabolic risk. Given its simplicity and cost-effectiveness, it may serve as a valuable adjunct tool for routine metabolic risk assessment in clinical practice.

Keywords*: Triglyceride–glucose index; insulin resistance; dyslipidemia; metabolic risk; cardiometabolic biomarkers.*

INTRODUCTION

Metabolic disorders including insulin resistance, dyslipidemia, and obesity contribute substantially to cardiovascular morbidity and mortality worldwide[1,2]. Early detection of subclinical metabolic alterations remains critical for timely intervention. Insulin resistance is central to the pathogenesis of metabolic syndrome and type 2 diabetes mellitus[3]. However, direct measurement using hyperinsulinemic-euglycemic clamp is impractical in routine settings[4]. Consequently, surrogate indices derived from routine laboratory parameters have gained importance. The Triglyceride–Glucose (TyG) index, calculated from fasting triglycerides and glucose, has emerged as a reliable surrogate of insulin resistance[5]. Several studies have demonstrated its association with diabetes, metabolic syndrome, and cardiovascular disease[6–9]. Despite growing

international evidence, region-specific data from Indian tertiary laboratory populations remain limited. This study was undertaken to evaluate the association between TyG index and early metabolic alterations in an adult laboratory cohort.

MATERIALS AND METHODS

Study Design and Settings

A cross-sectional analytical study was conducted in the Clinical Biochemistry Laboratory of Sree Mookambika Institute of Medical Sciences, Kulasekharam, Tamil Nadu. The study was carried out over a period of six months from January to June 2025 using laboratory records and anthropometric measurements obtained during routine health evaluations.

Study Population

A total of 300 adult individuals aged ≥18 years who underwent fasting biochemical investigations during the study period were included. Participants were selected consecutively based on availability of complete laboratory and anthropometric data.

Inclusion and Exclusion Criteria

Records were included if fasting blood glucose, complete lipid profile (total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol), and body mass index (BMI) measurements were available on the same visit. Subjects with previously diagnosed diabetes mellitus, chronic inflammatory diseases, known renal or hepatic disorders, endocrine disorders, pregnancy, or those receiving lipid-lowering or antidiabetic medications were excluded to minimize confounding effects on metabolic parameters.

Study Procedure and Laboratory Parameters

Venous blood samples were collected after an overnight fasting period of 8–12 hours. Fasting blood glucose was measured using the glucose oxidase–peroxidase method. Lipid profile parameters including total cholesterol, triglycerides, and HDL cholesterol were estimated by enzymatic colorimetric methods. LDL cholesterol was calculated using the Friedewald formula for samples with triglyceride levels <400 mg/dL. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m²).

The following parameters were analyzed: Fasting Blood Glucose (mg/dL), Triglycerides (mg/dL), Total Cholesterol (mg/dL), HDL Cholesterol (mg/dL), LDL Cholesterol (mg/dL), Body Mass Index (kg/m²) Calculation of TyG Index: The Triglyceride–Glucose (TyG) index was calculated using the formula: TyG = ln [(Triglycerides (mg/dL) × Fasting Blood Glucose (mg/dL)) / 2]

Participants were stratified into tertiles based on TyG distribution: T1 (Low TyG), T2 (Moderate TyG), T3 (High TyG) Metabolic Risk Classification: Metabolic risk was categorized based on the presence of predefined abnormal parameters: Fasting blood glucose ≥100 mg/dL, Triglycerides ≥150 mg/dL, HDL cholesterol <40 mg/dL (males) or <50 mg/dL (females), LDL cholesterol ≥130 mg/dL, BMI ≥25 kg/m².

Participants were grouped into: No Risk (0 abnormal parameters), Low Risk (1 abnormal parameter), Moderate Risk (2 abnormal parameters), High Risk (≥3 abnormal parameters).

Statistical Analysis

Data were expressed as mean ± standard deviation (SD) for continuous variables and percentages for categorical variables. Normality of distribution was assessed using the Kolmogorov–Smirnov test. Independent samples t-test was used to compare gender differences. One-way analysis of variance (ANOVA) followed by Tukey post-hoc analysis was applied to compare metabolic parameters across TyG tertiles. Pearson's correlation coefficient was used to evaluate relationships between TyG index and metabolic variables. Multiple linear regression analysis was performed to determine independent predictors of TyG index. Receiver Operating Characteristic (ROC) curve analysis was used to assess the discriminatory ability of TyG index in predicting metabolic risk. A p-value <0.05 was considered statistically significant. Statistical analyses were performed using SPSS version 25.0 (IBM Corp., Armonk, NY, USA).

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee of Sree Mookambika Institute of Medical Sciences. All data were anonymized prior to analysis, and confidentiality of participant information was strictly maintained.

RESULTS

A total of 300 adult laboratory records were analyzed. The mean age of the study population was 47.8 ± 14.6 years, with 158 males (52.7%) and 142 females (47.3%). The overall mean TyG index was 8.71 ± 0.62, with values ranging from 7.82 to 9.88. Participants were stratified into tertiles based on the distribution of their calculated TyG index to evaluate the metabolic risk gradient. The tertiles were defined as follows: Tertile 1 (T1 - Low TyG) included individuals with TyG index

values ranging from 7.82 to 8.34, with a group mean of 8.01±0.22. Tertile 2 (T2 - Moderate TyG) included individuals with values ranging from 8.35 to 8.99 (mean: 8.67±0.15), and Tertile 3 (T3 - High TyG) included individuals with values ranging from 9.00 to 9.88 (mean: 9.34±0.28). Gender-based comparison revealed marginally higher mean TyG values among males compared to females; however, this difference did not reach statistical significance ($p = 0.071$), suggesting that TyG distribution was comparable between genders. As shown in Table 1, individuals in the highest TyG tertile (T3) demonstrated significantly higher BMI, fasting blood glucose, triglycerides, total cholesterol, and LDL levels compared to those in T1 and T2 ($p < 0.001$ for all comparisons). Mean BMI increased from 23.1 ± 2.4 kg/m² in T1 to 27.6 ± 3.8 kg/m² in T3, indicating a strong association between TyG index and adiposity. Similarly, fasting blood glucose showed a graded rise from 86.4 ± 7.2 mg/dL in T1 to 108.6 ± 12.4 mg/dL in T3. Triglyceride levels nearly doubled across tertiles, rising from 118 ± 22 mg/dL in T1 to 198 ± 44 mg/dL in T3. LDL cholesterol also demonstrated a steady increase across tertiles. Conversely, HDL cholesterol showed a significant declining trend, decreasing from 52.3 ± 8.1 mg/dL in T1 to 41.8 ± 5.4 mg/dL in T3, reflecting worsening atherogenic lipid profile. One-way ANOVA revealed statistically significant differences across tertiles ($p < 0.001$), and post-hoc Tukey analysis confirmed that T3 differed significantly from both T1 and T2 for all major parameters. These findings indicate that higher TyG index is associated with progressive deterioration in metabolic profile.

Table-1: Baseline Characteristics by TyG Tertiles

Parameter	T1 (Low)	T2 (Moderate)	T3 (High)
TyG index	8.01 ± 0.22	8.67 ± 0.15	9.34 ± 0.28
BMI	23.1 ± 2.4	25.3 ± 3.1	27.6 ± 3.8
FBG	86.4 ± 7.2	94.8 ± 9.3	108.6 ± 12.4
TG	118 ± 22	152 ± 30	198 ± 44
HDL	52.3 ± 8.1	47.2 ± 6.3	41.8 ± 5.4
LDL	108 ± 24	126 ± 28	149 ± 32

ANOVA showed statistically significant differences across tertiles ($p < 0.001$). Participants in the highest TyG tertile demonstrated significantly higher BMI, fasting glucose, triglycerides, and LDL levels, along with lower HDL levels compared to lower tertiles ($p < 0.001$).

Correlation Analysis

Pearson correlation analysis demonstrated moderate but statistically significant associations between TyG index and metabolic variables. As depicted in Table 2, TyG index showed: Moderate positive correlation with fasting blood glucose ($r = 0.401$; $p < 0.001$) Moderate positive correlation with BMI ($r = 0.382$; $p < 0.001$), Positive correlation with total cholesterol ($r = 0.356$; $p < 0.001$), Positive correlation with LDL cholesterol ($r = 0.341$; $p < 0.001$), Significant inverse correlation with HDL cholesterol ($r = –0.298$; $p < 0.001$). The strongest association was observed with fasting glucose, reinforcing TyG's physiological link to glycemic regulation and insulin resistance. The inverse relationship with HDL suggests that higher TyG index corresponds to a more atherogenic lipid pattern. All correlations were statistically significant ($p < 0.001$), indicating a consistent relationship between TyG and adverse metabolic parameters.

Table 2: Pearson Correlation with TyG

Parameter	r	p-value
BMI	0.382	<0.001
FBG	0.401	<0.001
Total Cholesterol	0.356	<0.001
LDL	0.341	<0.001
HDL	–0.298	<0.001

TyG index showed moderate positive correlations with fasting glucose, BMI, LDL, and total cholesterol, and a significant inverse correlation with HDL cholesterol (all $p < 0.001$).

Multiple Linear Regression Analysis

To determine independent predictors of TyG index, multiple linear regression analysis was performed. As shown in Table 3, BMI ($\beta = 0.29$, $p < 0.001$), LDL cholesterol ($\beta = 0.21$, $p = 0.002$), and HDL cholesterol ($\beta = –0.18$, $p = 0.004$) emerged as independent determinants of TyG index. The overall regression model was statistically significant ($p < 0.001$) with an R^2 value of 0.42, indicating that approximately 42% of the variability in TyG index could be explained by the included metabolic variables. This suggests that TyG index reflects a composite metabolic burden rather than being dependent on a single parameter.

Table-3: Multiple Linear Regression Analysis for Determinants of TyG Index

Variable	β Coefficient	Standard Error	t value	p value	95% CI

BMI	0.29	0.05	5.82	<0.001	0.19–0.39
LDL	0.21	0.06	3.45	0.002	0.08–0.34
HDL	–0.18	0.05	–2.92	0.004	–0.30– –0.06

(Model Statistics: R^2 = 0.42, Adjusted R^2 = 0.40, F = 36.5, p < 0.001. BMI, LDL, and HDL cholesterol emerged as independent predictors of TyG index, explaining 42% of the variability (R^2 = 0.42, p < 0.001).)

Metabolic Risk Categorization

Based on predefined criteria, participants were categorized into metabolic risk groups: No Risk: 72 (24%), Low Risk: 108 (36%), Moderate Risk: 78 (26%) and High Risk: 42 (14%). A significantly higher proportion of individuals in the highest TyG tertile fell into moderate and high metabolic risk categories compared to lower tertiles (χ^2 test, p < 0.001). This indicates that TyG index effectively stratifies individuals according to metabolic risk burden.

ROC Curve Analysis

Receiver Operating Characteristic (ROC) curve analysis was performed to assess the discriminatory ability of TyG index in identifying individuals with ≥2 metabolic abnormalities (Figure 1). The TyG index demonstrated good predictive performance with: Area Under Curve (AUC): 0.79 (95% CI: 0.74–0.83), Sensitivity: 78%, Specificity: 72%, Optimal cut-off value: 8.85. An AUC of 0.79 indicates good diagnostic accuracy, supporting the utility of TyG index as a screening tool for early metabolic alterations in routine clinical practice.

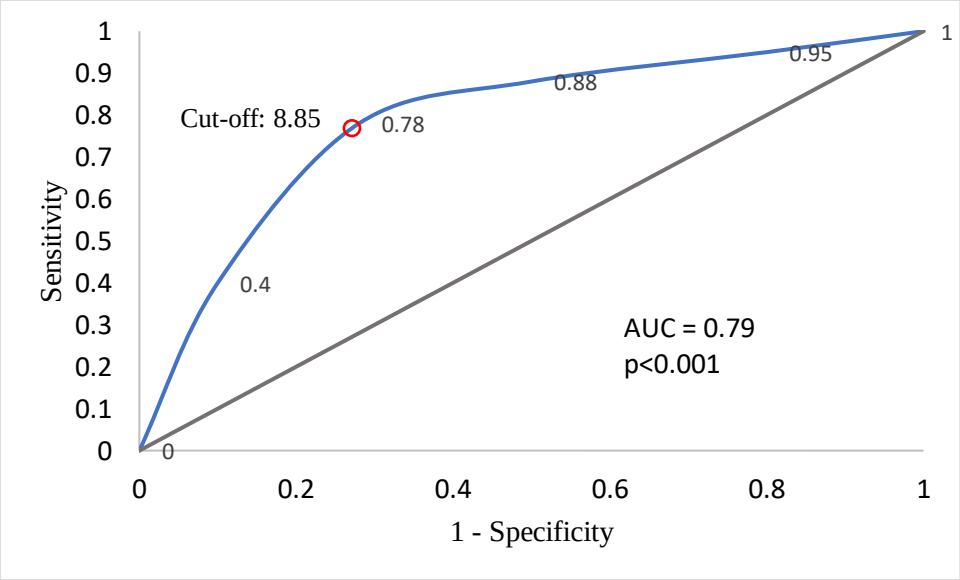

Figure 1: ROC Curve of TyG Index

The blue curve represents the trade-off between sensitivity and 1-specificity (AUC = 0.79). The diagonal grey line represents the reference line of no discrimination (AUC = 0.5). The red marker indicates the optimal predictive cut-off point of 8.85 for TyG index.

DISCUSSION

The present study demonstrates a significant association between the Triglyceride–Glucose (TyG) index and early metabolic alterations in an adult tertiary care laboratory population. Our findings indicate that TyG index was moderately and consistently associated with indices of adiposity, glycemic status, and atherogenic lipid abnormalities, supporting its potential role as an integrated metabolic risk marker. In the current study, TyG index showed a moderate positive correlation with fasting blood glucose (r = 0.401; p < 0.001). This finding reinforces the biological premise that TyG reflects underlying insulin resistance, as both fasting hyperglycemia and hypertriglyceridemia arise from impaired insulin-mediated glucose uptake and increased hepatic lipogenesis. Guerrero-Romero et al.[5] first proposed TyG as a reliable surrogate marker for insulin resistance, demonstrating strong concordance with the hyperinsulinemic-euglycemic clamp technique. Similarly, Lee et al.[7] reported that elevated TyG index independently predicted incident type 2 diabetes in a large prospective cohort. Navarro-González et al.[8] further confirmed its association with metabolic syndrome components, highlighting its clinical utility in risk stratification.

A significant association between TyG index and body mass index (r = 0.382) was also observed in our study. Adiposity contributes to insulin resistance through increased free fatty acid flux, chronic low-grade inflammation, and altered adipokine secretion[15]. Er et al.[16] demonstrated that higher TyG values were significantly associated with central obesity and

visceral adiposity. The correlation between TyG and BMI observed in our cohort suggests that TyG may reflect adiposity-driven metabolic stress even in individuals without overt diabetes.

With regard to lipid parameters, TyG index showed positive correlations with total cholesterol and LDL cholesterol, along with an inverse correlation with HDL cholesterol. This pattern indicates a strong association between TyG and atherogenic dyslipidemia. Sánchez-Íñigo et al.[9] reported that elevated TyG was independently associated with increased cardiovascular events. Similarly, da Silva et al.[11] demonstrated that TyG index predicted coronary artery calcification and subclinical atherosclerosis. The inverse relationship with HDL cholesterol observed in our study aligns with findings by Zhang et al.[12], who suggested that TyG reflects a pro-atherogenic lipid environment characterized by reduced anti-atherogenic HDL fractions. The pathophysiological basis underlying these associations likely involves shared mechanisms of insulin resistance, oxidative stress, and chronic inflammation. Insulin resistance promotes hepatic overproduction of very low-density lipoproteins (VLDL), leading to elevated triglycerides and small dense LDL particles[17]. Concurrently, impaired insulin signaling reduces lipoprotein lipase activity, lowering HDL cholesterol levels. Thus, TyG index may serve as a composite indicator integrating disturbances in glucose metabolism and lipid handling. Importantly, our ROC curve analysis demonstrated good discriminatory performance of TyG index for identifying individuals with ≥2 metabolic abnormalities (AUC = 0.79). This finding is comparable to previous studies reporting AUC values ranging from 0.72 to 0.84 for predicting metabolic syndrome and insulin resistance[13,18]. Park et al.[13] demonstrated that TyG outperformed HOMA-IR in certain populations for metabolic risk detection. The relatively high sensitivity and specificity observed in our study further support its potential role as a practical screening tool in routine laboratory settings.

Another important observation in our study was the graded increase in metabolic risk prevalence across TyG tertiles. Individuals in the highest tertile exhibited significantly higher rates of moderate and high metabolic risk categories. Similar dose-response relationships have been described by Ding et al.[14] and Irace et al.[10], who reported stepwise increases in cardiovascular risk with increasing TyG quartiles. Such graded associations strengthen the argument that TyG is not merely correlated with metabolic disturbances but may reflect cumulative cardiometabolic burden. The clinical appeal of TyG index lies in its simplicity, cost-effectiveness, and universal availability. Unlike insulin-based indices, TyG does not require additional laboratory testing. As emphasized by Vasques et al.[19], TyG represents a feasible alternative for large-scale screening, particularly in resource-limited settings. In the Indian context, where metabolic syndrome and prediabetes prevalence are rapidly increasing, incorporation of TyG index into routine laboratory reports may facilitate early identification of high-risk individuals.

Strengths of the Study

The present study benefited from a relatively robust sample size (n = 300), inclusion of comprehensive metabolic parameters, and dual risk classification using both TyG tertiles and predefined metabolic criteria. The use of multiple statistical approaches, including regression and ROC analysis, enhanced the reliability of our findings.

Study Limitations

However, certain limitations should be acknowledged. The cross-sectional design precludes causal inference. Direct measures of insulin resistance, such as HOMA-IR or hyperinsulinemic–euglycemic clamp studies, were not performed. Inflammatory biomarkers were not included, which could have provided additional mechanistic insights. Prospective longitudinal studies are warranted to evaluate the predictive value of TyG index for incident diabetes and cardiovascular outcomes.

CONCLUSION

The TyG index demonstrated significant association with adverse metabolic parameters and effectively discriminated individuals with early metabolic risk. Overall, our findings support growing evidence that the TyG index serves as an integrated marker of insulin resistance, adiposity, and atherogenic dyslipidemia. Given its simplicity and reproducibility, the TyG index may serve as a valuable adjunctive tool for early metabolic risk assessment in routine clinical biochemistry practice.

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Conflict of Interest: Nil

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