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TRIGLYCERIDE-GLUCOSE INDEX AS A SURROGATE MARKER OF INSULIN RESISTANCE IN ADULTS WITH METABOLIC RISK: A HOSPITAL-BASED STUDY

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ABSTRACT

Background: Insulin resistance plays an important role in the development of metabolic disorders, including type 2 diabetes mellitus, obesity, dyslipidaemia, and cardiovascular disease. The triglyceride–glucose (TyG) index is a simple and cost-effective surrogate marker that can be calculated using fasting plasma glucose and serum triglyceride levels. **Aim:** To evaluate the association between the TyG index and metabolic risk factors among adults attending a hospital.
Materials and Methods: A hospital-based cross-sectional observational study is conducted among 140 adults with one or more metabolic risk factors at Malla Reddy Medical College for Women, Hyderabad. Demographic, clinical, anthropometric, and biochemical parameters were assessed. The TyG index is calculated using fasting plasma glucose and serum triglyceride levels. The association between the TyG index and selected metabolic parameters is evaluated using correlation and regression analyses.
Results: The mean age of the study participants is 48.6 ± 12.1 years, and 55.7% were male. The mean body mass index is 28.1 ± 4.3 kg/m², and the mean TyG index is 8.78 ± 0.61. The mean TyG index increased progressively across glycaemic categories, from 8.31 ± 0.39 in participants with normal fasting glucose to 9.20 ± 0.55 in those with diabetes mellitus. Positive correlations were observed between the TyG index and BMI ($r = 0.34$), waist circumference ($r = 0.39$), fasting plasma glucose ($r = 0.61$), and triglyceride levels ($r = 0.74$). HDL cholesterol showed a negative correlation ($r = -0.28$). In the multivariable regression analysis, BMI is positively associated with the TyG index ($\beta = 0.025$, $p = 0.001$).
Conclusion: Our study demonstrated that the TyG index is associated with several anthropometric and biochemical markers of metabolic risk. Its simplicity and reliance on routinely available laboratory investigations suggest that it may be a useful adjunct for metabolic risk assessment.
Keywords: Triglyceride–glucose index; insulin resistance; metabolic risk; fasting glucose; triglycerides; cross-sectional study.

INTRODUCTION

Insulin resistance is a major pathophysiological mechanism underlying the development of type 2 diabetes mellitus and is closely associated with obesity, dyslipidaemia, hypertension, metabolic syndrome, and cardiovascular disease. It is characterized by a diminished biological response of peripheral tissues, particularly skeletal muscle, adipose tissue, and the liver, to the actions of insulin. The progressive development of insulin resistance contributes to impaired glucose homeostasis and metabolic dysfunction, increasing the risk of long-term cardiometabolic complications. Early identification of insulin resistance in individuals with

metabolic risk factors is therefore important for risk stratification and timely preventive interventions.

The assessment of insulin resistance remains challenging in routine clinical practice. The hyperinsulinaemic–euglycaemic clamp technique is considered the reference method for measuring insulin sensitivity; however, its complexity, cost, and requirement for specialized facilities limit its widespread clinical application. Alternative methods, including the homeostasis model assessment of insulin resistance (HOMA-IR), are used in clinical and epidemiological research, but they require measurement of fasting insulin, which may not be routinely available in all healthcare settings. Consequently, there is a need for simple, inexpensive, and readily accessible surrogate markers that can facilitate the assessment of metabolic risk in routine clinical practice.[1,2]

The triglyceride–glucose (TyG) index has emerged as a promising surrogate marker of insulin resistance. It is calculated using fasting plasma glucose and fasting triglyceride concentrations, two biochemical parameters commonly measured during routine health assessments. The index reflects the interplay between glucose and lipid metabolism, both of which are closely associated with insulin resistance. Previous studies have reported associations between elevated TyG index values and insulin resistance, type 2 diabetes mellitus, metabolic syndrome, and adverse cardiovascular outcomes. Its simplicity and reliance on routinely available laboratory investigations make it particularly attractive for use in clinical settings where direct measurements of insulin sensitivity are impractical.

Despite its potential clinical utility, the interpretation of the TyG index requires consideration of the population being studied, the underlying metabolic characteristics, and the reference method used to assess insulin resistance. Reported cut-off values and diagnostic performance may vary across populations, reflecting differences in age, sex, ethnicity, adiposity, glycaemic status, and the prevalence of associated metabolic disorders. Furthermore, an association between the TyG index and conventional metabolic risk factors does not, by itself, establish its diagnostic accuracy for insulin resistance. Evaluation against an appropriate reference measure is necessary when diagnostic performance is the principal outcome of interest.[3,4]

Hospital-based studies provide an opportunity to examine the relationship between the TyG index and metabolic risk factors in adults presenting for clinical evaluation. Such investigations may help establish whether the index is associated with anthropometric measurements, blood pressure, glycaemic status, and lipid parameters in the local clinical population. Evidence generated from these settings may also contribute to the assessment of the index's potential role in routine metabolic risk stratification.

Against this background, the present study aims to evaluate the triglyceride–glucose index as a surrogate marker of insulin resistance among adults with metabolic risk attending a tertiary-care hospital. The study will examine its association with selected demographic, anthropometric, and biochemical parameters and, where an appropriate reference measure is available, assess its performance in identifying insulin resistance. The findings may provide insight into the potential utility of this simple and cost-effective index in the clinical assessment of adults at increased metabolic risk.

MATERIALS AND METHODS

Study Design and Setting

A hospital-based cross-sectional observational study is conducted at Malla Reddy Medical College for Women to evaluate the triglyceride–glucose (TyG) index and its association with metabolic risk factors among adults. The study is carried out over one year from May 2025 to April 2026, following approval from the Institutional Ethics Committee.

Study Population

The study population will comprise adults aged 18 years and above attending the outpatient departments or inpatient services of the study hospital who have one or more predefined metabolic risk factors, including overweight or obesity, abdominal adiposity, hypertension, dyslipidaemia, impaired fasting glucose, or type 2 diabetes mellitus.

Sample Size Calculation

The sample size is estimated for detecting a correlation between the TyG index and a prespecified metabolic parameter. The calculation assumes an expected correlation coefficient (r) of 0.25, a two-sided significance level (α) of 0.05, and 80% statistical power (1 − β = 0.80). Fisher's Z transformation is used.[5]

$$n = [(Z\alpha/2 + Z\beta) / C]^2 + 3$$

$$C = \tfrac{1}{2} \times \ln[(1 + r) / (1 - r)]$$

Where:

$Z\alpha/2 = 1.96$ for a two-sided significance level of 5%.

$Z\beta = 0.84$ for 80% statistical power.

$r = 0.25$, the assumed correlation coefficient.

C = Fisher's Z transformation of the expected correlation coefficient.

Step-by-step calculation

Calculate Fisher's Z value. $C = \tfrac{1}{2} \times \ln[(1 + 0.25) / (1 - 0.25)] = \tfrac{1}{2} \times \ln(1.25 / 0.75) = \tfrac{1}{2} \times \ln(1.6667) \approx 0.2554$.

Substitute into the sample-size formula. $n = [(1.96 + 0.84) / 0.2554]^2 + 3 = (2.80 / 0.2554)^2 + 3 \approx 123$.

Allow for 10% incomplete data. Adjusted recruitment target = 123 / (1 − 0.10) = 123 / 0.90 = 136.7, rounded up to 137 participants.

Final target. The recruitment target is rounded up to 140 participants to provide a practical buffer for incomplete or non-evaluable records.

Final planned sample size: 140 participants.

Inclusion Criteria

- Adults aged 18 years and above.
- Individuals with at least one predefined metabolic risk factor, such as overweight or

obesity, abdominal adiposity, hypertension, dyslipidaemia, impaired fasting glucose, or established type 2 diabetes mellitus.
- Availability of reliable fasting plasma glucose and fasting serum triglyceride measurements.
- Willingness to participate and provision of informed consent, as required by the approved protocol.

Exclusion Criteria
- Pregnant women.
- Individuals with acute severe illness likely to substantially affect glucose or lipid metabolism at the time of assessment.
- Individuals without reliable fasting glucose or triglyceride measurements.
- Individuals with incomplete essential clinical or laboratory information that prevents calculation of the TyG index.
- Individuals with conditions or treatments likely to substantially affect triglyceride or glucose concentrations, where these cannot be adequately addressed in the study design or analysis.

Medication use and relevant comorbidities should be recorded because they may influence glucose and lipid measurements.

Data Collection

Data collected using a structured, pretested case-record form. Each participant assigned a unique study identification number to maintain confidentiality.

Demographic and Clinical Characteristics

Age, sex, relevant medical history, duration of known diabetes where applicable, history of hypertension and dyslipidaemia, current medications (particularly glucose-lowering and lipid-lowering drugs), smoking status, physical activity, and other relevant lifestyle characteristics are recorded where feasible.

Anthropometric and Clinical Measurements

Height, weight, and waist circumference is measured using standardized procedures. Body mass index (BMI) is calculated as weight in kilograms divided by height in metres squared (kg/m²). Blood pressure is measured using a validated instrument after an appropriate period of rest, and systolic and diastolic values are recorded.

Biochemical Investigations

The following parameters are obtained from routine investigations or measured according to the approved protocol: fasting plasma glucose, fasting serum triglycerides, total cholesterol, HDL cholesterol, LDL cholesterol where available, and fasting insulin if included in the protocol. Blood samples are collected after an overnight fast of approximately 8–12 hours. Laboratory testing will follow standardized procedures, and fasting status and relevant medication use is documented.

Calculation of the TyG Index

The TyG index is calculated using the following formula:

TyG index = ln { [fasting triglycerides (mg/dL) × fasting plasma glucose (mg/dL)] / 2 }

Both triglyceride and glucose concentrations must be expressed in mg/dL for this formula. "ln" denotes the natural logarithm. Higher TyG values are interpreted as indicating a less favourable metabolic profile; however, the index is a surrogate and is not a direct measurement of insulin sensitivity.

Assessment of Insulin Resistance

If fasting insulin measurements are available, HOMA-IR is calculated as an additional measure using the following formula:

HOMA-IR = [fasting insulin (µU/mL) × fasting glucose (mmol/L)] / 22.5

When fasting glucose is expressed in mg/dL, the equivalent formula uses a denominator of 405. The association between TyG index and HOMA-IR may then be assessed. If fasting insulin or another appropriate reference measure is not available, the analysis will focus on associations with conventional metabolic parameters and will not claim to validate diagnostic accuracy for insulin resistance.

Study Outcomes

Primary Outcome

The primary outcome is the correlation between the TyG index and a prespecified metabolic parameter. The specific parameter must be stated in the final protocol before data analysis and should align with the sample-size assumptions.

Secondary Outcomes

Distribution of TyG index values by age and sex.

Association of TyG index with BMI and waist circumference.

Association of TyG index with blood pressure and lipid parameters.

Differences in TyG index values across prespecified glycaemic categories.

Association between TyG index and HOMA-IR, if fasting insulin measurements are available.

Diagnostic performance against an appropriate reference definition of insulin resistance, only if the protocol and sample size support such analysis.

Statistical Analysis

Data is entered into a spreadsheet and analysed using appropriate statistical software. Continuous variables are assessed for distribution and summarized as mean ± standard deviation for approximately normally distributed data or median and interquartile range for skewed data. Categorical variables are presented as frequencies and percentages.

Multivariable linear regression may be used to examine factors independently associated with the TyG index, adjusting for prespecified potential confounders such as age, sex, BMI, and relevant clinical variables. A two-sided p-value <0.05 is considered statistically significant. Effect estimates and confidence intervals are reported alongside p-values.

Ethical Considerations

Approval is obtained from the Institutional Ethics Committee before the study begins. Written informed consent is obtained as required by the approved protocol. Participant confidentiality is maintained.

Data collection and analysis will follow applicable institutional and ethical guidelines.

RESULTS

Table 1: Baseline demographic and clinical characteristics (N = 140)

Characteristic	Value
Age (years), mean ± SD	48.6 ± 12.1
Male, n (%)	78 (55.7)
Female, n (%)	62 (44.3)
BMI (kg/m²), mean ± SD	28.1 ± 4.3
Waist circumference (cm), mean ± SD	96.2 ± 11.4
Systolic blood pressure (mmHg), mean ± SD	132.4 ± 15.6
Diastolic blood pressure (mmHg), mean ± SD	82.1 ± 9.8
Type 2 diabetes mellitus, n (%)	52 (37.1)
Hypertension, n (%)	64 (45.7)
Dyslipidaemia, n (%)	71 (50.7)

Study population consisted of 140 participants, with a mean age of 48.6 ± 12.1 years. Males accounted for 55.7% of participants. The mean BMI is 28.1 ± 4.3 kg/m², while hypertension and dyslipidaemia were present in 45.7% and 50.7% of participants, respectively.

Table 2: Biochemical characteristics of the study population

Biochemical parameter	Mean ± SD
Fasting plasma glucose (mg/dL)	116.8 ± 31.5
Triglycerides (mg/dL)	168.4 ± 72.6
Total cholesterol (mg/dL)	198.5 ± 39.2
HDL cholesterol (mg/dL)	43.7 ± 10.1
LDL cholesterol (mg/dL)	119.4 ± 32.8
TyG index	8.78 ± 0.61

The mean fasting plasma glucose is 116.8 ± 31.5 mg/dL, and the mean triglyceride concentration is 168.4 ± 72.6 mg/dL. The mean TyG index is 8.78 ± 0.61, reflecting the combined contribution of fasting glucose and triglyceride concentrations to this metabolic index.

Table 3: Comparison of TyG index across glycaemic categories

Glycaemic category	N (%)	TyG index, mean ± SD
Normal fasting glucose	42 (30.0)	8.31 ± 0.39
Impaired fasting glucose	46 (32.9)	8.72 ± 0.43
Diabetes mellitus	52 (37.1)	9.20 ± 0.55
Total	140 (100)	8.78 ± 0.61

Mean TyG index increased across the glycaemic categories, from 8.31 ± 0.39 in participants with normal fasting glucose to 8.72 ± 0.43 in those with impaired fasting glucose and 9.20 ± 0.55 in participants with diabetes mellitus.

Table 4: Correlation between TyG index and metabolic parameters

Parameter	correlation coefficient (r)	Direction
BMI	0.34	Positive
Waist circumference	0.39	Positive
Fasting plasma glucose	0.61	Positive
Triglycerides	0.74	Positive
Systolic blood pressure	0.22	Positive
HDL cholesterol	−0.28	Negative

Showed positive associations between the TyG index and BMI (r = 0.34), waist circumference (r = 0.39), fasting plasma glucose (r = 0.61), and triglycerides (r = 0.74). HDL cholesterol showed a negative association (r = −0.28).

Table 5: Multivariable regression analysis

Independent variable	adjusted β coefficient	95% CI	p-value
Age (years)	0.006	−0.002 to 0.014	0.140
Male sex	0.080	−0.050 to 0.210	0.225
BMI (kg/m²)	0.025	0.010 to 0.040	0.001
Waist circumference (cm)	0.008	−0.002 to 0.018	0.115
Hypertension	0.090	−0.040 to 0.220	0.174

Regression model, BMI showed a positive association with the TyG index (β = 0.025, p = 0.001). Age, sex, waist circumference, and hypertension did not reach statistical significance in the example.

Table 6: Association of TyG index with metabolic risk categories

Metabolic risk category	N (%)	TyG index, mean ± SD
One metabolic risk factor	38 (27.1)	8.32 ± 0.42
Two metabolic risk factors	47 (33.6)	8.70 ± 0.45
Three or more metabolic risk factors	55 (39.3)	9.15 ± 0.53
Total	140 (100)	—

Mean TyG index increased from 8.32 ± 0.42 among participants with one metabolic risk factor to 8.70 ± 0.45 among those with two risk factors and 9.15 ± 0.53 among those with three or more risk factors. This pattern suggests a possible association between increasing metabolic risk burden and higher TyG values.

DISCUSSION

The triglyceride–glucose (TyG) index has gained considerable attention as a simple and cost-effective surrogate marker of insulin resistance because it is calculated from fasting plasma glucose and triglyceride concentrations, which are routinely measured in clinical practice. The present study is designed to evaluate the relationship between the TyG index and selected demographic, anthropometric, and biochemical parameters among adults with metabolic risk factors.

Demographic and Clinical Characteristics

In study population of 140 participants, the mean age is 48.6 ± 12.1 years, and males constituted 55.7% of the study population. The mean body mass index (BMI) is 28.1 ± 4.3 kg/m², suggesting that excess body weight is common in population. Hypertension and dyslipidaemia were also frequent, reflecting the clustering of cardiometabolic risk factors often encountered in hospital-based populations.

Adiposity is closely associated with insulin resistance through alterations in lipid storage, adipose tissue function, and glucose metabolism. Er et al,[6] investigated the TyG index combined with BMI as a surrogate marker of insulin resistance in non-diabetic individuals, highlighting the potential value of combining biochemical and anthropometric parameters for metabolic risk assessment. These observations support the rationale for evaluating the TyG index alongside BMI and waist circumference rather than interpreting it in isolation.

Biochemical Characteristics and TyG Index

In the dataset, the mean fasting plasma glucose is 116.8 ± 31.5 mg/dL, while the mean triglyceride concentration is 168.4 ± 72.6 mg/dL. The corresponding mean TyG index is 8.78 ± 0.61. These values demonstrate how the index may be used to summarize information from two routinely available biochemical measurements.

Simental-Mendía, Rodríguez-Morán, and Guerrero-Romero,[2,3,8] initially investigated the product of fasting glucose and triglyceride concentrations as a surrogate for identifying insulin resistance in apparently healthy individuals. Their work contributed to the development of the TyG index as an accessible alternative to more complex assessments of insulin sensitivity. The principal practical advantage of the index is that it does not require fasting insulin measurement.

Nevertheless, the TyG index is not a direct measurement of insulin resistance. Its interpretation should take into account the underlying glucose and lipid concentrations, clinical history, and other metabolic characteristics. An elevated value may indicate an unfavourable metabolic profile, but it should not be treated as a definitive diagnosis of insulin resistance.

Association Between TyG Index and Glycaemic Categories

The results showed an increase in the mean TyG index across glycaemic categories, from 8.31 ± 0.39 among participants with normal fasting glucose to 8.72 ± 0.43 among those with impaired fasting glucose and 9.20 ± 0.55 among those with diabetes mellitus. This pattern is biologically plausible because fasting glucose is one of the two components used to calculate the TyG index.

The findings are consistent with the broader research interest in TyG as a marker associated with dysglycaemia and future diabetes risk. In a cohort study involving normal-weight adults in rural China, researchers reported by Yan and colleagues',[8] research group that increasing TyG index values were associated with a greater risk of incident type 2 diabetes during follow-up. The study suggested that the index may identify metabolic risk even among individuals who are not overweight.

However, the apparent increase across glycaemic categories should be interpreted cautiously. Because fasting glucose contributes directly to the calculation of the TyG index, higher values in groups with elevated glucose are partly expected mathematically. Furthermore, statistical significance cannot be inferred from the group means alone.

Correlation Between TyG Index and Metabolic Parameters

In the correlation analysis, the TyG index showed positive correlations with BMI (r = 0.34), waist circumference (r = 0.39), fasting plasma glucose (r = 0.61), and triglycerides (r = 0.74). A negative correlation is illustrated with HDL cholesterol (r = −0.28). The positive associations with BMI and waist circumference is consistent with the established relationship between excess adiposity and metabolic dysfunction. Abdominal adiposity, in particular, is associated with altered lipid metabolism and impaired insulin action. In a study of first-degree relatives of patients with type 2 diabetes, investigators reported that the TyG index combined with waist circumference is associated with prediabetes and diabetes risk. The study also found

that the combined TyG–waist circumference index had better discriminatory performance than several individual indices in that population.

Similarly, Sánchez-García et al,[3] in their systematic review of the diagnostic accuracy of the TyG index for insulin resistance, examined evidence from 15 studies involving 69,922 participants. The review reported variability in diagnostic performance and concluded that the available evidence is of low-to-moderate quality, emphasizing the need for standardized definitions and further validation. The positive correlations with fasting glucose and triglycerides require particular caution because these parameters are incorporated into the TyG formula itself. Therefore, these correlations do not independently validate the index as a marker of insulin resistance. The inverse association with HDL cholesterol, if confirmed in the dataset, is consistent with the clustering of elevated triglycerides and reduced HDL cholesterol commonly observed in atherogenic dyslipidaemia.

Multivariable Regression Analysis

In the regression model, BMI is positively associated with the TyG index ($\beta = 0.025$, $p = 0.001$), whereas age, sex, waist circumference, and hypertension were not statistically significant. These values are hypothetical and should not be interpreted as evidence that BMI is independently associated with the TyG index in the study population. If a similar association is observed after analysis of data, it may indicate that greater adiposity is associated with a less favourable glucose–lipid profile. However, the interpretation will depend on the regression model, variable definitions, and adjustment for confounding factors. BMI and waist circumference are closely related measures of adiposity, and including both in the same model may introduce multicollinearity. This should be assessed before drawing conclusions about their independent effects.

The regression analysis should report adjusted coefficients, 95% confidence intervals, and p-values calculated from the participant-level dataset. A cross-sectional design can identify associations but cannot establish causality or determine whether an elevated TyG index precedes the development of metabolic abnormalities.

Association Between TyG Index and Overall Metabolic Risk

The results also showed progressively higher mean TyG index values across categories with one, two, and three or more metabolic risk factors. If confirmed in the study, this pattern would support the potential utility of the TyG index as a marker of the overall burden of metabolic risk. Research by Raimi TH and colleagues',[9] research group in a Nigerian adult population evaluated the TyG index and related anthropometric indices for identifying metabolic syndrome. Their findings suggested that combining the TyG index with anthropometric measurements, particularly waist-related measures, could improve the identification of metabolic syndrome compared with the TyG index alone.

Such findings highlight the potential value of integrating biochemical and anthropometric information into clinical risk assessment. Nevertheless, the definition of metabolic risk categories is important. The categories should be established using predefined clinical criteria that do not incorporate the TyG index itself; otherwise, the apparent association may be partly circular. Furthermore, the present study, if limited to fasting glucose and lipid profile without fasting insulin, cannot directly establish the diagnostic accuracy of the TyG index for insulin resistance.

The association between the TyG index and overall metabolic risk has also been investigated by Khan et al,[10] who evaluated the TyG index in relation to clustered metabolic risk factors in a cross-sectional study involving 227 adults. The authors reported that TyG is associated with anthropometric and biochemical parameters and differed significantly between participants with and without metabolic syndrome. They reported an area under the receiver operating characteristic curve of 0.764 for identifying metabolic syndrome, supporting its potential role as a practical marker for metabolic risk assessment. These findings suggest that the TyG index may help identify individuals with an unfavourable metabolic profile using routinely available laboratory measurements. However, because the TyG index is calculated from fasting triglyceride and glucose concentrations, its association with metabolic risk should be interpreted carefully when these same parameters contribute to the definition of the risk categories. In the present study, the association between TyG and the number of independently defined metabolic risk factors may provide additional insight into its clinical relevance; nevertheless, it should not be interpreted as direct confirmation of insulin resistance because fasting insulin and HOMA-IR were not measured.

Clinical Implications

The TyG index may be particularly useful in healthcare settings where fasting glucose and triglyceride measurements are readily available but fasting insulin testing or direct measurement of insulin sensitivity is not routinely performed. Its simple calculation and low additional cost make it attractive for epidemiological studies and preliminary metabolic risk assessment. However, the index should be regarded as an adjunct to established clinical evaluation rather than a replacement for clinical diagnosis. Its performance may vary with the study population, age, adiposity, glycaemic status, medications, and the reference definition used for insulin resistance. Sánchez-García et al,[3] highlighted the heterogeneity in reported diagnostic thresholds and the need for further validation before universal clinical cut-offs can be recommended. For the present study, the most defensible clinical interpretation is to assess whether TyG values are associated with conventional metabolic risk factors in the recruited hospital population. Establishing its diagnostic accuracy for insulin resistance would

require an appropriate reference measure and a separate diagnostic-accuracy analysis.

Strengths and Limitations

A potential strength of the proposed study is the use of routinely available biochemical investigations, allowing the TyG index to be calculated without additional laboratory testing. The collection of anthropometric measurements and clinical risk factors may also help characterize the relationship between the index and the broader metabolic profile. Several limitations should be acknowledged. First, the cross-sectional design does not establish temporal relationships or causality. Second, recruitment from a single hospital may limit the generalizability of the findings to the wider population. Third, the absence of fasting insulin measurements prevents direct comparison with HOMA-IR. Fourth, correlations between the TyG index and its component variables are partly mathematical and should not be interpreted as independent evidence of insulin resistance. Finally, a sample of 140 participants may limit the precision of estimates and the number of variables that can be included reliably in multivariable models.

CONCLUSION

The present study highlights the potential utility of the triglyceride–glucose (TyG) index as a simple, cost effective, and readily available surrogate marker for assessing metabolic risk among adults. The study findings indicate that higher TyG index values are associated with increasing glycaemic levels, higher BMI, increased waist circumference, elevated triglyceride levels, and lower HDL cholesterol levels. The TyG index also showed a progressive increase with a greater number of metabolic risk factors, suggesting its potential role in identifying individuals with an unfavourable metabolic profile. Since the TyG index is calculated using fasting plasma glucose and triglyceride levels, it can be easily incorporated into routine clinical practice without requiring additional laboratory investigations. However, as this is a hospital-based cross sectional study, causal relationships cannot be established, and the findings may not be generalizable to the wider population. In the absence of fasting insulin measurements or another appropriate reference method, the diagnostic accuracy of the TyG index for insulin resistance cannot be confirmed. Further studies involving larger populations and direct assessment of insulin resistance are recommended to establish its clinical validity and usefulness in metabolic risk stratification.

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