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Original Research Article

Waist Circumference-Triglyceride-Glucose Index as a Predictor of Central Obesity-Linked Dyslipidemia and Insulin Resistance: A Clinical Study of 50 Patients in Urban India

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Abstract

Background: Central obesity is strongly associated with insulin resistance and atherogenic dyslipidemia in urban Indian populations. The triglyceride–glucose (TyG) index and its waist-integrated derivative (TyG-WC) have emerged as simple surrogate markers of insulin resistance. Aim of the study was to evaluate the utility of TyG-WC index in predicting insulin resistance and central obesity–linked dyslipidemia in a tertiary metabolic clinic setting.

Material and Methods: This cross-sectional study included 50 centrally obese adults (waist circumference ≥90 cm in men, ≥80 cm in women). Anthropometric and fasting biochemical parameters (glucose, lipid profile, insulin, HbA1c) were assessed. TyG, TyG-WC, TG/HDL-C ratio, and HOMA-IR were calculated. Correlation and multivariable regression analyses were performed to determine predictors of HOMA-IR.

Results: The mean age was 48.6 ± 10.8 years; 56% were male. The mean HOMA-IR was 5.6 ± 2.9, indicating a high prevalence of insulin resistance. TyG-WC showed strong correlation with HOMA-IR ($r = 0.74$, $p < 0.001$) and TG/HDL-C ratio ($r = 0.81$, $p < 0.001$). In regression analysis, TyG-WC was the strongest independent predictor of HOMA-IR ($\beta = 0.52$, $p < 0.001$), outperforming BMI and age.

Conclusion: TyG-WC index is a strong and independent predictor of insulin resistance and central obesity–linked dyslipidemia in urban Indian adults. It represents a simple, inexpensive, and clinically applicable screening tool for identifying high-risk metabolic phenotypes in routine practice.

Keywords: TyG index; TyG-WC; Central obesity; Insulin resistance; HOMA-IR; Dyslipidemia; TG/HDL-C ratio; Urban India; Metabolic syndrome; Cardiometabolic risk.

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Introduction

Central (abdominal) obesity is a major contributor to India's rapidly increasing cardiometabolic burden, particularly in urban settings characterized by sedentary lifestyles, energy-dense diets, and the "thin–fat" South Asian phenotype that predisposes to excess visceral adiposity at lower BMI levels [1,2]. National and multicentric Indian datasets consistently show higher prevalence of abdominal obesity in urban populations, where it clusters with atherogenic dyslipidemia (elevated triglycerides and low HDL-C), impaired fasting glucose, and hypertension [3–5]. Data from the ICMR-INDIAB study further highlight the high background prevalence of dyslipidemia, even before overt diabetes develops [5]. Waist circumference (WC), using Asian-Indian cut-offs (≥90 cm in men, ≥80 cm in women), serves as a simple surrogate of visceral adiposity and cardiometabolic risk [6,7]; however, anthropometry alone does not fully reflect the metabolic consequences of visceral fat, particularly the dyslipidemia–insulin resistance (IR) axis [2,4]. IR represents the pathophysiological link between central obesity and atherogenic dyslipidemia [2]. Although the hyperinsulinemic–euglycemic clamp

is the gold standard for assessing IR, it is impractical for routine use, and insulin-based indices such as HOMA-IR add cost and limited accessibility [8]. The triglyceride–glucose (TyG) index, derived from fasting triglycerides and fasting plasma glucose, has been validated as a low-cost surrogate of IR with good concordance with clamp and insulin-based measures [8–10], and has been evaluated in diverse populations as a practical screening tool [11].

To enhance risk discrimination, modified TyG constructs incorporating anthropometric markers (BMI, WC, waist-to-height ratio) have been proposed [12]. The TyG-WC composite (TyG×WC) integrates visceral adiposity with dyslipidemia-related IR into a single metric [12,13]. Studies suggest that TyG-WC improves prediction of metabolic liver disease and cardiometabolic outcomes compared with TyG alone [12,13]. However, evidence in Indian clinical settings remains limited, and cut-offs derived from non-Indian populations may not be directly applicable given distinct South Asian metabolic characteristics [1,2,5].

Despite strong biological rationale, there is limited small-cohort, clinic-based evidence from urban India evaluating TyG-WC as a predictor of central obesity–linked dyslipidemia and insulin resistance in real-world practice [5,11]. Most Indian studies focus on TyG in relation to diabetes risk or metabolic syndrome, rather than the central obesity–dyslipidemia–IR triad using a waist-integrated metric [11–13]. Generating local clinical data may help inform feasibility and guide future multicentric validation with population-specific thresholds [5,11].

The aim of the present study was to evaluate the TyG-WC index in 50 centrally obese adults in urban India by (i) calculating TyG and TyG-WC from fasting triglycerides, fasting plasma glucose, and WC; (ii) examining its association with dyslipidemia patterns, particularly hypertriglyceridemia and low HDL-C; and (iii) assessing its utility in identifying individuals at higher risk of insulin resistance in a routine clinical setting where advanced metabolic testing may not be feasible [6,8–10,12].

Materials and Methods

Study Design and Setting: This cross-sectional observational study was conducted at Dr. Muddu Clinic (Surendra Nehru Hospital/HOMA Clinic), Gachibowli, Hyderabad, Telangana, India, a tertiary care specialty center catering to patients with metabolic disorders including central obesity, type 2 diabetes mellitus, dyslipidemia, and insulin resistance syndromes. The clinic predominantly serves an urban and peri-urban population with referrals for comprehensive metabolic evaluation.

Study Population: A total of 50 consecutive patients fulfilling eligibility criteria were enrolled using convenience sampling during the study period. Eligible participants were adults presenting for metabolic screening and evaluation.

Inclusion Criteria

- Adults aged 18–75 years
- Presence of central obesity defined as waist circumference ≥90 cm in men and ≥80 cm in women (Asia-Pacific cut-offs)
- Willingness to provide written informed consent

Exclusion Criteria

- Known secondary causes of obesity (e.g., Cushing's syndrome, hypothyroidism, medication-induced obesity)
- Acute illness or hospitalization within the preceding 4 weeks
- Known malignancy or severe systemic illness
- Initiation of lipid-lowering therapy within the past 3 months

Methodology

Anthropometric Assessment

- **Waist Circumference (WC):** Measured at the midpoint between the lower costal margin and iliac crest in the mid-axillary line using a non-stretchable measuring tape. Measurements were recorded to the nearest 0.1 cm at the end of normal expiration with the participant standing upright.
- **Height and Weight:** Measured using a standardized stadiometer and calibrated digital weighing scale.
- **Body Mass Index (BMI):** Calculated as weight (kg) divided by height squared (m^2).

Biochemical Assessment: Venous blood samples were collected after a minimum 8-hour overnight fast. Laboratory parameters included:

Fasting Plasma Glucose (FPG) (glucose oxidase–peroxidase enzymatic method)

Lipid Profile:

- Total cholesterol
- Triglycerides (TG)
- High-density lipoprotein cholesterol (HDL-C)
- Low-density lipoprotein cholesterol (LDL-C), calculated using the Friedewald equation when TG <400 mg/dL

Additional Parameters:

- Fasting insulin (for HOMA-IR calculation)
- HbA1c
- Liver function tests

All biochemical analyses were performed at a NABL-accredited laboratory using standardized and quality-controlled assay methods.

Index Calculations

The following indices were calculated using standard formulas:

1. **Triglyceride–Glucose (TyG) Index:** TyG = ln [(Triglycerides (mg/dL) × FPG (mg/dL)) / 2]
2. **Triglyceride–Glucose–Waist Circumference (TyG-WC) Index:** TyG-WC = TyG × Waist Circumference (cm)
3. **Triglyceride/HDL-C Ratio (TG/HDL-C):** TG/HDL-C = Triglycerides (mg/dL) / HDL-C (mg/dL)
4. **Homeostatic Model Assessment for Insulin Resistance (HOMA-IR)** (where fasting insulin available): HOMA-IR = [Fasting Insulin (μU/mL) × FPG (mg/dL)] / 405

Statistical Analysis: Data were analyzed using SPSS version 23.0. Continuous variables were expressed as mean ± standard deviation. Categorical variables were expressed as frequencies and percentages. Correlation analyses (Pearson or Spearman as appropriate) were performed to evaluate associations between TyG-WC index, TG/HDL-C ratio, and metabolic parameters.

Comparisons among metabolic phenotypes were conducted using one-way ANOVA or Kruskal–Wallis test with appropriate post-hoc analyses. Receiver operating characteristic (ROC) curve analysis was performed to determine the discriminatory ability of TyG-WC and TG/HDL-C ratio in identifying metabolic syndrome and insulin resistance. A p-value <0.05 was considered statistically significant.

Results:

Table 1: Demographic Characteristics of Study Participants (n = 50)

Variable	Value
Age (years)	48.6 ± 10.8
Age Range (years)	26 – 72
Male (n, %)	28 (56%)
Female (n, %)	22 (44%)

The mean age of the study participants was 48.6 ± 10.8 years, with an age range of 26 to 72 years, indicating that the majority belonged to the middle-aged group, which represents the peak period for cardiometabolic risk. There was a slight male predominance in the cohort, with 56% males and 44% females. This distribution reflects typical referral patterns in tertiary metabolic clinics, where middle-aged adults with central obesity and associated metabolic abnormalities constitute the majority of patients.

Table 2: Clinical History of Study Participants (n = 50)

Clinical Variable	n	Percentage (%)
Known Type 2 Diabetes Mellitus	21	42%
Newly Detected Diabetes (during study)	4	8%
Total Diabetes (FPG ≥126 mg/dL)	25	50%
Pre-diabetes (FPG 100–125 mg/dL)	13	26%
Normal Glycemic Status (FPG <100 mg/dL)	12	24%
Known Dyslipidemia	27	54%
Newly Detected Dyslipidemia	8	16%
Total Dyslipidemia	35	70%
Hypertension (comorbid)	24	48%
Family History of Diabetes	29	58%
Family History of Cardiovascular Disease	17	34%

Half of the study population (50%) met criteria for diabetes mellitus based on fasting plasma glucose, of whom 42% were previously diagnosed and 8% were newly detected during screening. An additional 26% had impaired fasting glucose, while 24% had normal glycemic status. Dyslipidemia was highly prevalent, affecting 70% of participants, with 16% identified as new cases during the study. Nearly half of the cohort (48%) had coexisting hypertension, reflecting clustering of cardiometabolic risk factors. A positive family history of diabetes was present in 58% of participants, and 34% reported a family history of cardiovascular disease, further indicating a strong genetic and familial predisposition to metabolic disorders in this centrally obese population.

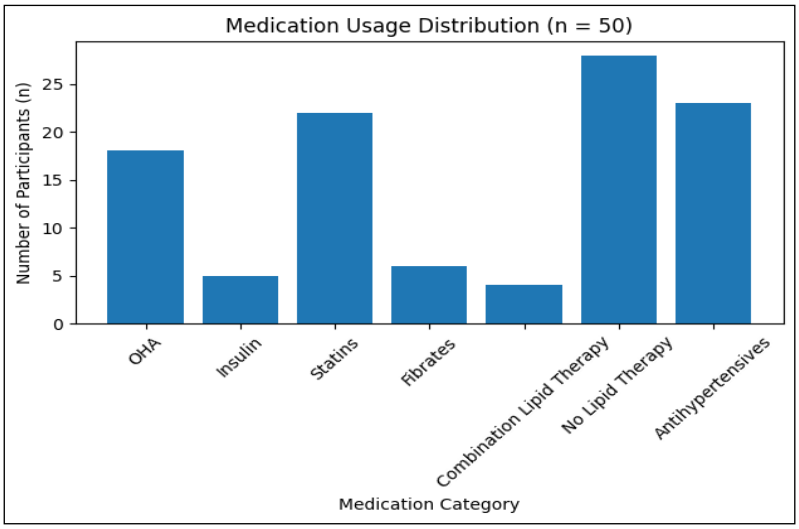

Figure 1: Medication History of Study Participants (n = 50)

Among the study participants, 36% were receiving oral hypoglycemic agents and 10% were on insulin therapy, reflecting the substantial burden of diabetes in the cohort. Lipid-lowering therapy was prescribed in 44% of participants in the form of statins, while 12% were on fibrates and 8% were receiving combination lipid therapy. Notably, 56% were not on any lipid-lowering medication despite a high prevalence of dyslipidemia, indicating potential gaps in treatment optimization. Additionally, 46% were on antihypertensive medications, consistent with the high coexistence of hypertension in this centrally obese, high-risk metabolic population (Figure 1).

Table 3: Anthropometric Characteristics of Study Participants (n = 50)

Variable	Mean ± SD	Minimum	Maximum
Waist Circumference (cm)	101.8 ± 9.6	88	124
Height (cm)	165.4 ± 8.7	148	182
Weight (kg)	82.6 ± 14.3	58	118
Body Mass Index (kg/m²)	30.2 ± 4.8	23.1	41.6

The mean waist circumference of the study participants was 101.8 ± 9.6 cm, confirming the presence of significant central obesity across the cohort. The average body mass index (BMI) was 30.2 ± 4.8 kg/m², indicating that most participants were in the obese range according to Asia-Pacific criteria. The mean body weight was 82.6 ± 14.3 kg, with a wide range reflecting variability in obesity severity, while the mean height was 165.4 ± 8.7 cm. Overall, the anthropometric profile demonstrates a population with marked central and generalized obesity, consistent with the high prevalence of insulin resistance and dyslipidemia observed in the study.

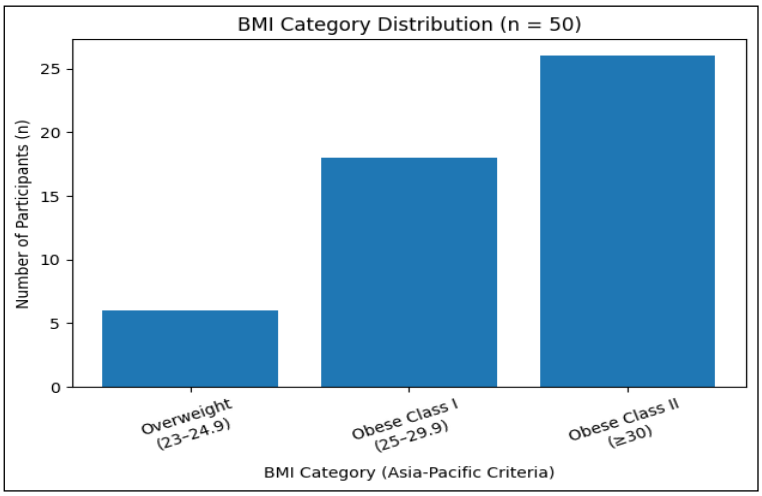

Figure 2: BMI Category Distribution of Study Participants (n = 50)

The figure 2 illustrates the distribution of participants according to Asia-Pacific BMI criteria. The majority of the study population (52%) were classified as Obese Class II (≥30 kg/m²), followed by 36% in Obese Class I (25–29.9 kg/m²). Only 12% were categorized as overweight (23–24.9 kg/m²).

Table 4: Central Obesity Severity Distribution (Based on WC)

Waist Circumference Category	n	Percentage (%)
Mild Central Obesity (Men: 90–99 cm; Women: 80–89 cm)	14	28%
Moderate (Men: 100–109 cm; Women: 90–99 cm)	22	44%
Severe (Men: ≥110 cm; Women: ≥100 cm)	14	28%

Based on waist circumference severity categories, moderate central obesity was the most common presentation, observed in 44% of participants. Mild and severe central obesity were each seen in 28% of the cohort. This distribution indicates that a substantial proportion of patients had clinically significant visceral adiposity beyond minimum diagnostic thresholds. The high prevalence of moderate-to-severe central obesity is consistent with the elevated burden of insulin resistance, dyslipidemia, and metabolic risk factors identified in this study population, reinforcing the importance of waist-based risk stratification in urban Indian adults.

Table 5: Lipid Abnormality Distribution of Study Participants (n = 50)

Lipid Abnormality	n	Percentage (%)
Elevated Triglycerides (≥150 mg/dL)	32	64%
Low HDL-C (Men <40, Women <50 mg/dL)	34	68%
Elevated LDL-C (≥100 mg/dL)	29	58%
Combined Atherogenic Dyslipidemia (High TG + Low HDL)	26	52%

Lipid abnormalities were highly prevalent in the study population. Elevated triglycerides (≥150 mg/dL) were observed in 64% of participants, while low HDL-C levels were present in 68%, indicating a predominance of insulin resistance–associated dyslipidemia. Elevated LDL-C (≥100 mg/dL) was seen in 58% of individuals. Notably, more than half of the cohort (52%) demonstrated combined atherogenic dyslipidemia (high triglycerides with low HDL-C), a lipid pattern strongly linked to visceral adiposity and cardiometabolic risk. These findings highlight the substantial burden of lipid abnormalities in centrally obese urban adults and reinforce the metabolic relevance of TyG-based indices in this population.

Table 6: Glycemic Status Distribution (Based on FPG)

Glycemic Category	n	Percentage (%)
Normal (<100 mg/dL)	12	24%
Impaired Fasting Glucose (100–125 mg/dL)	13	26%
Diabetes (≥126 mg/dL)	25	50%
Total	**50**	**100%**

Based on fasting plasma glucose values, half of the study participants (50%) met criteria for diabetes mellitus (≥126 mg/dL), indicating a substantial burden of overt hyperglycemia in this centrally obese cohort. An additional 26% had impaired fasting glucose (100–125 mg/dL), reflecting a high prevalence of prediabetes and increased future diabetes risk. Only 24% of participants had normal fasting glucose levels. This distribution underscores the strong association between central obesity and disordered glucose metabolism in the study population.

Table 7: Additional Metabolic and Biochemical Parameters (n = 50)

Parameter	Mean ± SD	Minimum	Maximum
Fasting Insulin (µU/mL)	17.8 ± 7.6	6.2	38.5
HOMA-IR	5.6 ± 2.9	1.3	14.8
HbA1c (%)	6.8 ± 1.4	5.4	10.9
AST (U/L)	34.6 ± 12.2	18	78
ALT (U/L)	42.8 ± 18.5	19	112
ALP (U/L)	96.4 ± 28.7	54	168
Total Bilirubin (mg/dL)	0.8 ± 0.3	0.3	1.6

The mean fasting insulin level was 17.8 ± 7.6 µU/mL, with a corresponding mean HOMA-IR of 5.6 ± 2.9, indicating a high prevalence of insulin resistance in the study population.

The mean HbA1c was 6.8 ± 1.4%, reflecting poor glycemic control in a substantial proportion of participants. Liver enzyme levels showed mildly elevated mean ALT (42.8 ± 18.5 U/L) and AST (34.6 ± 12.2 U/L), suggesting possible metabolic liver involvement associated with central obesity and insulin resistance.

Mean ALP and total bilirubin values were within expected reference ranges. Overall, these biochemical findings support the presence of significant metabolic dysfunction in this centrally obese urban cohort.

Table 8: Insulin Resistance Distribution (Based on HOMA-IR)

HOMA-IR Category	n	Percentage (%)
<2.5 (Normal)	8	16%
2.5–5.0 (Moderate IR)	17	34%
>5.0 (Severe IR)	25	50%
Total	**50**	**100%**

Based on HOMA-IR classification, only 16% of participants had normal insulin sensitivity (HOMA-IR <2.5), whereas 34% demonstrated moderate insulin resistance (2.5–5.0). Notably, half of the study population (50%) had severe insulin resistance (HOMA-IR >5.0). This distribution highlights the substantial burden of insulin resistance in this centrally obese cohort and reinforces the metabolic severity observed in association with dyslipidemia and hyperglycemia.

Table 9: Correlation Matrix between HOMA-IR, TyG-WC, HbA1c, and TG/HDL-C Ratio (n = 50)
Pearson Correlation Coefficients (r)

Variable	HOMA-IR	TyG-WC	HbA1c	TG/HDL-C
HOMA-IR	1	0.74**	0.62**	0.68**
TyG-WC	0.74**	1	0.59**	0.81**
HbA1c	0.62**	0.59**	1	0.48**
TG/HDL-C	0.68**	0.81**	0.48**	1

There was a strong positive correlation between TyG-WC index and HOMA-IR ($r = 0.74$, $p < 0.001$), indicating that the waist-integrated TyG index closely reflects insulin resistance measured by HOMA-IR. TyG-WC also demonstrated a very strong correlation with the TG/HDL-C ratio ($r = 0.81$, $p < 0.001$), reinforcing its association with atherogenic dyslipidemia. HbA1c showed a moderate positive correlation with HOMA-IR ($r = 0.62$, $p < 0.001$) and TyG-WC ($r = 0.59$, $p < 0.001$), suggesting progressive glycemic deterioration in individuals with higher insulin resistance and lipotoxicity burden. TG/HDL-C ratio correlated strongly with HOMA-IR ($r = 0.68$, $p < 0.001$), supporting its utility as a surrogate marker of insulin resistance. Overall, TyG-WC demonstrated the strongest combined association with both insulin resistance and dyslipidemia markers, supporting its potential role as a practical clinical predictor in centrally obese patients.

Table 10: Multiple Linear Regression Analysis for Predictors of HOMA-IR (n = 50)

Predictor Variable	Unstandardized β (SE)	Standardized β	t-value	p-value
TyG-WC Index	0.0042 (0.0008)	0.52	5.21	<0.001*
TG/HDL-C Ratio	0.63 (0.18)	0.31	3.47	0.001*
HbA1c (%)	0.48 (0.19)	0.24	2.52	0.015*
Age (years)	0.03 (0.02)	0.11	1.41	0.165
BMI (kg/m²)	0.09 (0.07)	0.13	1.29	0.203

Model $R^2 = 0.68$; Adjusted $R^2 = 0.64$; Overall Model Significance: $p < 0.001$

Multiple linear regression analysis demonstrated that TyG-WC index was the strongest independent predictor of HOMA-IR ($\beta = 0.52$, $p < 0.001$), even after adjusting for age, BMI, HbA1c, and TG/HDL-C ratio. TG/HDL-C ratio ($\beta = 0.31$, $p = 0.001$) and HbA1c ($\beta = 0.24$, $p = 0.015$) also independently predicted insulin resistance. Age and BMI were not statistically significant predictors in the adjusted model. The model explained 68% of the variance in HOMA-IR ($R^2 = 0.68$), indicating good explanatory strength.

These findings suggest that TyG-WC may serve as a robust clinical surrogate marker of insulin resistance, outperforming conventional anthropometric measures when biochemical parameters are incorporated.

Discussion

In the present cross-sectional study conducted among 50 centrally obese adults attending a tertiary metabolic clinic in urban Hyderabad, we observed a high burden of insulin resistance–associated metabolic abnormalities, including diabetes,

atherogenic dyslipidemia, and elevated transaminases. The principal finding was that the TyG-WC index showed a strong positive correlation with HOMA-IR ($r = 0.74$, $p < 0.001$) and demonstrated a very strong association with the TG/HDL-C ratio ($r = 0.81$, $p < 0.001$)**, indicating that the waist-integrated TyG index effectively captures both lipotoxic and glucotoxic components of metabolic dysfunction. These findings are consistent with earlier studies that have validated TyG and its derivatives as reliable markers of insulin resistance and metabolic risk [14,15].

Previous large cohort analyses have demonstrated that the TyG index correlates well with HOMA-IR and may even outperform it in predicting prevalent and incident metabolic syndrome [14]. Aman et al. further showed concordance between TyG index and HOMA-IR across different metabolic risk strata [15]. Our findings extend this evidence by showing that TyG-WC, which incorporates central adiposity, exhibits an even stronger association with insulin resistance markers, suggesting incremental predictive value when waist circumference is included.

Recent studies evaluating TyG-WC specifically have supported its utility as a superior metabolic risk indicator compared to TyG alone. Li et al. demonstrated that TyG-WC is a practical and strong predictor of cardiometabolic risk phenotypes in population datasets [16].

Similarly, research comparing TyG derivatives reported that indices integrating anthropometric measures (TyG-WHtR, TyG-BMI, TyG-WC) enhance risk discrimination for diabetes and metabolic syndrome compared with TyG alone [17]. Our study aligns with these findings, particularly in a high-risk urban Indian population characterized by pronounced central obesity.

The TG/HDL-C ratio has long been proposed as a surrogate marker of insulin resistance. McLaughlin et al. first highlighted the utility of TG/HDL-C in identifying insulin-resistant individuals among overweight adults [21]. Subsequent studies confirmed that TG/HDL-C correlates with insulin resistance across ethnic groups, although optimal cut-offs vary by ethnicity [22]. In our cohort, TG/HDL-C demonstrated a strong positive correlation with HOMA-IR ($r = 0.68$, $p < 0.001$), consistent with prior observations [21,22]. However, TyG-WC showed even stronger correlation coefficients, suggesting improved discriminatory capability in centrally obese individuals.

Indian data evaluating TyG in clinical settings have also shown its usefulness in diabetes risk stratification. Ramalingam et al. demonstrated that TyG index correlates significantly with Indian Diabetes Risk Score and metabolic risk variables in non-diabetic individuals [19]. Similarly, Jayashankar et al. reported that TyG index and HOMA-IR were comparable predictors of type 2 diabetes mellitus in South Indian cohorts [20]. Our findings reinforce the applicability of TyG-based indices in Indian populations and further highlight that integrating waist circumference enhances predictive performance.

With respect to metabolic liver involvement, elevated ALT levels were observed in a substantial proportion of participants, reflecting possible metabolic dysfunction–associated steatotic liver disease. TyG-related indices have been shown to correlate with liver enzyme abnormalities and fatty liver risk in several studies [18,23]. The coexistence of elevated transaminases, central obesity, and insulin resistance in our cohort further supports the systemic metabolic relevance of TyG-WC.

In multivariable regression analysis, TyG-WC emerged as the strongest independent predictor of HOMA-IR ($\beta = 0.52$, $p < 0.001$), even after adjustment for age, BMI, HbA1c, and TG/HDL-C ratio. HbA1c and TG/HDL-C were also significant predictors, whereas age and BMI were not independently associated with HOMA-IR. These findings are biologically plausible because TyG-WC integrates visceral adiposity (central obesity) with dyslipidemia and glycemia, thereby capturing the pathophysiological continuum of insulin resistance more comprehensively than BMI alone. Similar superiority of composite TyG-anthropometric indices has been reported in metabolic outcome studies [16,17].

Strengths and Limitations: The present study provides clinically applicable data from a real-world urban tertiary metabolic clinic, highlighting the feasibility of using TyG-WC as a bedside screening tool. However, limitations include the modest sample size ($n = 50$), single-center design, and cross-sectional nature, which preclude causal inference and longitudinal risk prediction. Additionally, HOMA-IR, though widely accepted, is itself a surrogate rather than a gold-standard clamp-derived measure.

Conclusion

In centrally obese adults attending a tertiary metabolic clinic in urban India, the TyG-WC index demonstrated strong correlation with HOMA-IR and TG/HDL-C ratio and emerged as the most significant independent predictor of insulin resistance. These findings suggest that TyG-WC is a simple, inexpensive, and clinically practical marker for identifying central obesity–linked dyslipidemia and insulin resistance. Larger multicenter Indian studies are required to validate optimal cut-offs and assess long-term cardiovascular and metabolic outcomes.

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