



## The triglyceride-glucose (TyG) index as an independent predictor of coronary artery disease: A cross sectional study

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Insulin resistance (IR) is a significant risk factor for cardiovascular disease, which is the leading cause of death worldwide. A straightforward and reasonably priced surrogate indicator for IR and cardiovascular risk is the triglyceride-glucose (TyG) index. The purpose of this study was to assess the TyG index's correlation and diagnostic value in patients with confirmed coronary artery disease (CAD). 75 CAD patients and 75 healthy controls were compared in this hospital-based cross sectional study. The TyG index was evaluated as an independent predictor using logistic regression, which was adjusted for age, sex, and BMI. The ideal cutoff and diagnostic accuracy were determined by ROC curve analysis. The mean TyG index was significantly higher in the CAD group ( $4.94 \pm 0.34$ ) versus Controls ( $4.56 \pm 0.21$ ) ( $P < 0.001$ ). Multivariate analysis confirmed the TyG index as an independent predictor of CAD (OR: 18.49, 95% CI: 3.57-95.82,  $P=0.001$ ). ROC analysis yielded an AUC of 0.818. The optimal cutoff was 4.71, achieving 77% sensitivity and 80% specificity. The TyG index is a robust, independent predictor of CAD with high diagnostic utility, supporting its integration into routine clinical practice for early risk stratification.

**Keywords:** Coronary artery disease, Insulin Resistance, Triglyceride–glucose index

Cardiovascular diseases are a collection of disorders which affects the circulatory system which results in the myocardial infarction, stenosis of the blood vessels, and systemic dysfunction. Of these, coronary heart disease (CHD) continues to be the primary cause of death worldwide, accounting for 19.8 million fatalities in 2022<sup>1</sup>.

In India for the past 60 years coronary artery disease has become more common among both urban and rural populations<sup>2,3</sup>. It is found that the prevalence of CAD varied between 2.5% and 12.6% in urban areas and between 1.4% and 4.6% in rural areas<sup>4</sup>.

According to various studies, insulin resistance has been shown to be an independent risk factor for atherosclerosis, even in the absence of hyperglycemia or dyslipidemia. Finding insulin resistance indicators linked to atherosclerosis indicators may make it possible to identify patients early in the course of the disease and start preventative treatments sooner<sup>5</sup>.

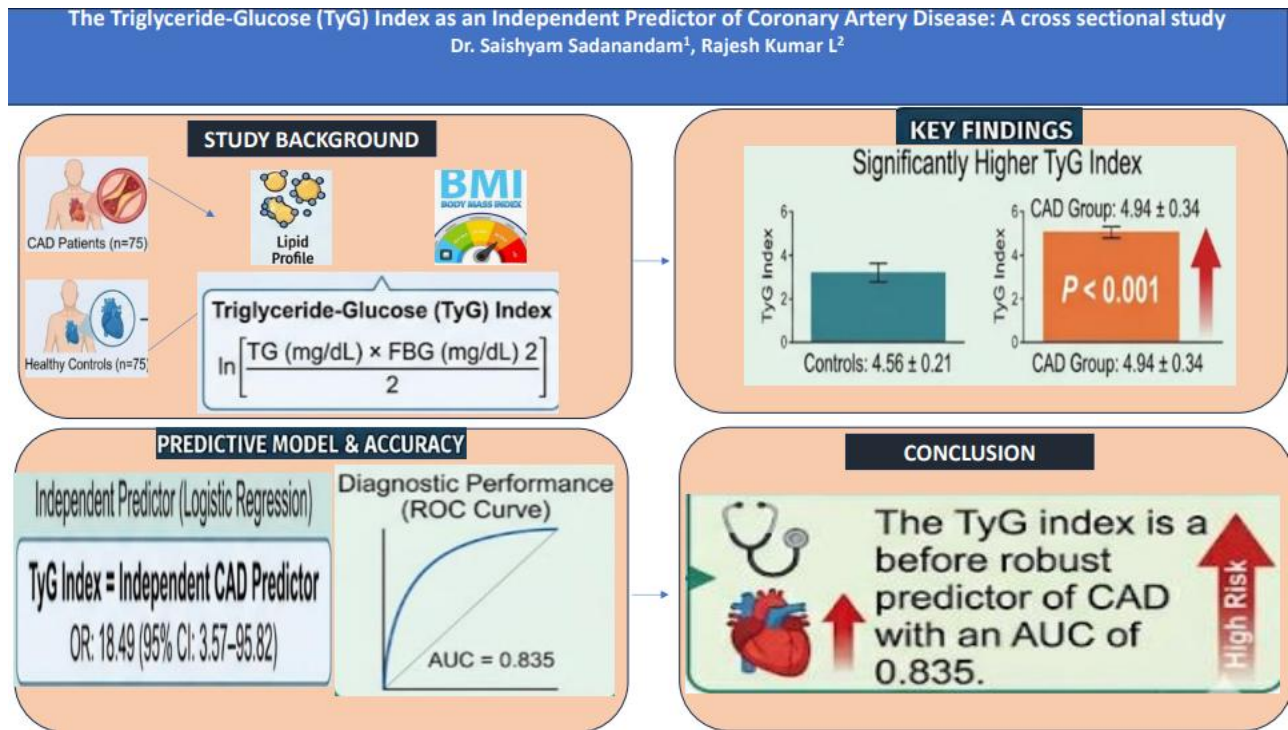
The development of atherosclerosis, which results in cardiovascular problems, is significantly influenced

by endothelial dysfunction. Furthermore, endothelial dysfunction is caused by selective insulin resistance in the phosphoinositide 3-kinase/Akt/endothelial nitric oxide (NO) synthase pathway in endothelial cells, which results in decreased NO production and increased endothelin-1 production from the endothelium<sup>6</sup>.

Because of its accuracy, the hyperinsulinemic-euglycemic clamp is regarded as the gold standard method for evaluating insulin resistance; however, its extensive clinical use is restricted by its complexity. In order to estimate insulin resistance and aid in early detection and risk stratification in metabolic disorders, surrogate indices based on fasting and post-load glucose and insulin levels have been developed<sup>7</sup>.

A thorough statistical metric that takes into account both fasting triglyceride and fasting glucose levels is the Triglyceride-Glucose Index (TyG index). Because of its high sensitivity and specificity, studies have shown that it can be used as a useful alternative biomarker for insulin resistance (IR). TyG index reflects the combined impact of hypertriglyceridemia and hyperglycemia. The importance of the TyG index in a variety of medical conditions has been highlighted by numerous excellent clinical studies<sup>8</sup>.

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Graphical abstract

The findings indicate that in patients with CAD, an elevated TyG index is linked to a higher risk of Major Adverse Cardiovascular Events (MACEs), cardiovascular mortality, all-cause mortality, myocardial infarction, revascularization procedures, and stroke<sup>9</sup>. However, data specifically focusing on the diagnostic utility of the TyG index in discriminating between patients with established CAD and healthy controls, particularly in localized populations, remain varied. Furthermore, establishing a locally relevant, optimal cutoff value that maximizes sensitivity and specificity is crucial for translating this research into clinical practice.

## Materials and Methods

### Study Design and Population

This was a hospital-based, non-interventional cross sectional study conducted at a tertiary care center. The study population was divided into two groups: Case group (CAD, n=75): Patients diagnosed with established Coronary Artery Disease (CAD), Control group (Healthy, n=75): Healthy individuals recruited from the general population who were age- and sex-matched to the case group and had no history or clinical evidence of CAD or other major chronic diseases. The study received ethical approval from the

Institutional Human Ethics Committee (Proposal Id: {IHCC-II/0550/24}).

### Data Collection and Biochemical Measurements

Detailed clinical history, demographic data (age, sex, BMI), and established risk factors (history of diabetes mellitus, history of hypertension) were collected for all participants. Fasting blood samples were drawn after a minimum 10–12 h fast. The following biochemical parameters were measured: fasting glucose, triglyceride, LDL, HDL, total cholesterol, HbA1c. The Triglyceride-Glucose ({TyG} index was calculated for all participants using the standard formula:  $\text{TyG Index} = \{\text{Ln}\}\{\text{Fasting TGL (mg/dL)}\} \times \{\text{Fasting Glucose (mg/dL)}\} / \{2\}$ <sup>9</sup>.

### Statistical Analysis

Data analysis was performed using a statistical software package SPSS. Continuous variables were presented as Mean and Standard Deviation and categorical variables as frequencies and percentages. Continuous variables were compared using the non-parametric Mann-Whitney U test, given the non-normal distribution of several key variables (*e.g.*, TGL, Troponin I). Categorical variables were compared using the Chi-square test. The Spearman's rank correlation coefficient was used to assess the association between the TyG index and other continuous metabolic and cardiac markers. A

Table 1 — Comparison of demographic data for the study participants

Characteristic	Cases (n=75)	Controls (n=75)	p-value
Age (years)	59.84 ± 13.49	58.88 ± 12.45	0.653
BMI (kg/m <sup>2</sup> )	27.00 (26.00 - 28.00)	24.00 (23.00 - 24.00)	< 0.001
Sex (Male), n (%)	46 (62.2)	40 (53.3)	0.355

Table 2 — Comparison of baseline characteristics for the study participants

Characteristic	Cases (n=75)	Controls (n=75)	p-value
HbA1c (%)	6.80 (5.90 - 8.65)	5.60 (5.25 - 5.80)	< 0.001
TyG Index	4.94 ± 0.34	4.56 ± 0.21	< 0.001
Triglycerides (mg/dL)	112.00 (89.25 - 153.75)	89.00 (83.00 - 121.50)	< 0.001
Glucose (mg/dL)	159.00 (123.00 - 231.25)	97.00 (89.00 - 116.00)	< 0.001
Total Cholesterol (mg/dL)	182.50 (123.75 - 224.50)	112.00 (111.00 - 119.00)	< 0.001
HDL (mg/dL)	40.00 (32.00 - 48.00)	44.00 (37.00 - 52.00)	0.008
LDL (mg/dL)	107.00 (67.75 - 150.50)	49.60 (40.30 - 64.00)	< 0.001
VLDL (mg/dL)	26.00 (19.25 - 36.25)	17.80 (16.60 - 24.30)	< 0.001

Receiver Operating Characteristic (ROC) curve analysis was conducted to evaluate the discriminatory power of the TyG index for CAD diagnosis. Logistic Regression analysis was performed to determine the association of the TyG index with CAD (dependent variable). A univariate model was first run, followed by a multivariate model adjusted for the known confounders: age, sex, and BMI.

## Results and Discussion

Table 1 provides a comprehensive overview of the baseline demographic and Table 2 provides metabolic characteristics of our study population, comparing the case group and the control group. A correct matching of Age (p=0.653) and Sex (p=0.355) among the cases and control was done in our study. As seen in the table, there were no statistically significant differences between the two groups. Males were comparatively more than the females in case group.

The BMI was found to be high in the case group (Median 27.0) compared to the control group (Median 24.0). A p value of less than 0.001 indicated that the difference was statistically significant. This discrepancy emphasizes the part that obesity plays. Weight is not the only problem here; visceral fat is a metabolically active organ that releases inflammatory cytokines, which exacerbates the group's insulin resistance and dyslipidemia. This finding is consistent with the study done by Held C *et al*, in their study they have concluded that when people with stable coronary

heart disease fell into the overweight or obese BMI categories (BMI > 25 kg/m<sup>2</sup>), their inflammatory and cardiometabolic health markers gradually worsened<sup>10</sup>.

In our study we found that there is a significant difference in the lipid parameters between of the case group and the control group. The median Triglyceride levels in cases is 112.00 (89.25 - 153.75) when compared with the controls 89.00 (83.00 - 121.50) with a p value of < 0.001. This finding corroborates with the finding of the study done by Reiner Ž, which revealed higher levels of triglycerides (fasting or nonfasting) or, more precisely, triglyceride-rich lipoproteins and their by products, are independently linked to an increased risk of cardiovascular disease (CVD)<sup>11</sup>.

The median total cholesterol and LDL in cases is 182.50 (123.75 - 224.50) and 107.00 (67.75 - 150.50) when compared with the control 112.00 (111.00 - 119.00) and 49.60 (40.30 - 64.00) which is statistically significant (p value <0.001). This pattern, which is strongly linked to CAD, is characterized by significantly higher levels of Triglycerides, LDL ("bad cholesterol"), and VLDL, coupled with significantly lower levels of protective HDL ("good cholesterol") (p=0.008). This finding is in agreement with the study done by Khan SW *et al*<sup>12,13</sup>. This combination fosters an inflammatory and pro-thrombotic environment conducive to atherosclerotic plaque formation<sup>14</sup> (Table 2).

There is a noticeable difference in glycemic control. The case group's fasting glucose level

Table 3 — Logistic Regression

Model	Variable	Odds Ratio (OR)	95% CI	P-value
Univariate	TyG Index	15.35	5.49 - 42.92	0.001
Multivariate	TyG Index	18.49	3.57 - 95.82	0.001
(Adjusted)	BMI	4.22	2.44 - 7.29	0.001
	Age	1.02	0.98 - 1.07	0.359
	Sex (Male)	2.31	0.62 - 8.65	0.209

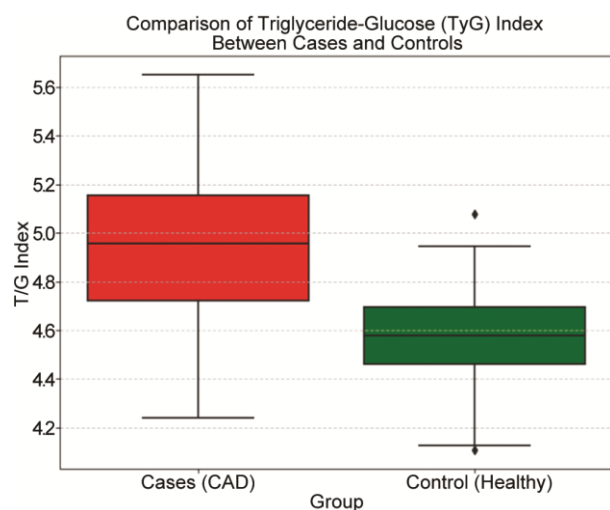


Fig. 1 — Box Plot comparison of triglyceride glucose index between cases and controls

(median 159 vs. 97 mg/dL) and HbA1c (median 6.8% vs. 5.6%) were both noticeably higher. This study finding is in relation with the study done by Kayali Y *et al.* They have concluded that an independent risk factor for CAD was HbA1c, the risk of stenosis increases by up to 12.4 times for every unit increase in HbA1c. This suggests widespread insulin resistance and impaired glucose metabolism, which alone are known risk factors for endothelial dysfunction and cardiovascular disease<sup>15</sup>.

This brings us to our study's main variable, the TyG Index. The problem is summarized by the highly significant difference ( $P < 0.001$ ), which is not just another isolated finding. The CAD Case group had a significantly higher TyG Index ( $4.94 \pm 0.34$ ) than the Healthy Control group ( $4.56 \pm 0.21$ ) ( $P < 0.001$ ). This finding is similar to the study done by Wang X *et al.*, which revealed that an increased TyG index was associated with a higher risk of multi-vessel CAD and TyG as an estimation index for evaluating IR could be a valuable predictor of CAD severity<sup>16</sup>. The TyG Index is a proxy for insulin resistance that is derived from both fasting triglycerides and glucose. Although TGL and glucose are both high on their own, our table

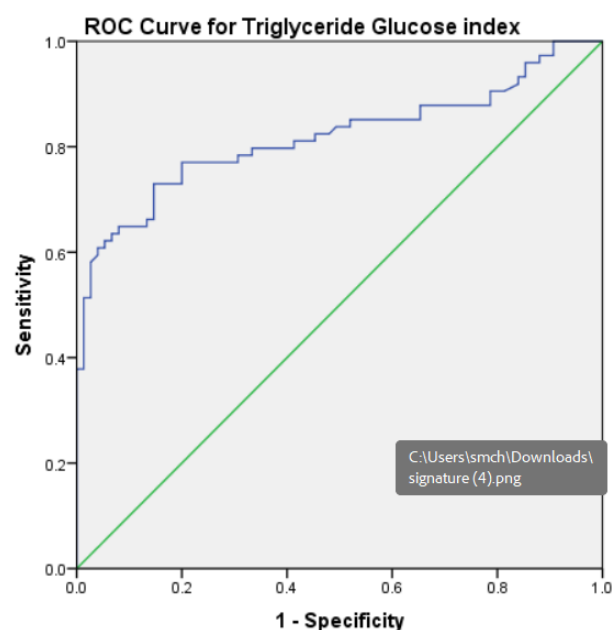


Fig. 2 — ROC curve analysis for TyG Index

demonstrates that their product, the TyG Index, offers a single, potent metric that captures this whole state of metabolic dysregulation. It implies that the underlying pathophysiology of CAD in our sample is a condition of insulin resistance, in which cells in the body do not react to insulin, resulting in a series of glucotoxicity (high glucose) and lipotoxicity (high TGL). Our main hypothesis—that the TyG Index is a clinically meaningful indicator that combines these two important metabolic insults—is strongly supported by this finding<sup>17-19</sup>.

To determine the cutoff value, sensitivity, and specificity, ROC curve analysis was performed for the triglyceride glucose index in cases and controls (Figs. 1 & 2). The TyG Index for CAD has good discriminatory power, as indicated by its AUC of 0.818. With 77% Sensitivity and 80% Specificity, the ideal cutoff point is 4.71, which maximizes diagnostic accuracy.

Regression analysis was done to find out the predictive value of TyG, one-unit increase in the TyG

Index is associated with over a 15-fold increased odds of having CAD ( $P < 0.001$ ). After adjusting for Age, Sex, and BMI, the TyG Index remains a highly significant and independent predictor of CAD, with an 18.49-fold increase in odds for every one-unit increase ( $P = 0.001$ ). BMI also emerged as an independent predictor (OR 4.22,  $P < 0.001$ ) (Table 3).

Our study has certain limitations; study was conducted with a relatively limited sample size. This study was conducted in a single centre limiting external validity. Furthermore, disease severity was not assessed by using angiogram reports.

Future research should focus on evaluating whether pharmacological therapy and lifestyle intervention that reduce the triglyceride glucose index can improve cardiovascular outcomes.

### Conclusion

In conclusion, there is strong evidence from this study that the triglyceride-glucose (TyG) index is a reliable and independent indicator of coronary artery disease (CAD). Our results show that the mean TyG index is significantly higher in CAD patients than in healthy controls, indicating the widespread involvement of metabolic dysfunction, most likely caused by insulin resistance, in the etiology of CAD.

The TyG index is a better clinical marker because it is non-invasive, inexpensive, and relies on commonly available fasting lipid and glucose parameters. In order to support early risk stratification and direct focused preventative interventions, especially in settings with limited resources, we advise including it in routine metabolic screening panels. In order to verify its predictive value for unfavorable cardiovascular outcomes, future research should concentrate on validating this 4.80 cutoff in prospective longitudinal studies.

### Acknowledgement

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### Conflict of interest

All authors declare no conflict of interest.

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