

Triglyceride Glucose Index For The Detection Of Asymptomatic Coronary Artery Stenosis In Patients With Type 2 Diabetes Mellitus In Tertiary Care Center

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ABSTRACT

Background: Silent coronary atherosclerosis is frequent in type 2 diabetes mellitus (T2DM) and goes undetected in very many cases until acute events. The triglyceride-glucose (TyG) index is an inexpensive surrogate of insulin resistance and has become a possible screening marker of coronary artery disease. This study evaluated the ability of TyG index to identify asymptomatic coronary artery stenosis (CAS) and correlated with stenosis severity in a tertiary care setting.

Methods: In this study, a cross-sectional observational study performed for 6 months, 81 adults without symptoms of T2DM attending a tertiary care center were recruited. Fasting plasma glucose, HbA1c, lipid profile, and fasting insulin was measured. TyG index was determined by $((\text{Triglycerides (mg/dL)} \times \text{fasting glucose (mg/dL)}) / (2)) = \text{LN}[\text{TyG index}]$. Coronary stenosis was measured with the use of CT coronary angiography (CTCA) and graded by maximum luminal narrowing (no CAD, <50, 50-69, 70%) and CAD-RADS grade. Associations were analyzed by means of trend tests and correlation analyzes and with multivariable logistic regression. Receiver operating characteristic (ROC) analysis was carried out for the diagnostic performance for obstructive CAS ($\geq 70\%$).

Results: Mean age was 55.8 ± 9.6 years; 58.0% were men. Median TyG index was 9.92 (IQR 9.55–10.25). CTCA found any plaque in 54.3%, stenosis $>50\%$ in 38.3% and obstructive stenosis $>70\%$ in 22.2%. TyG was higher stepwise with the severity of stenosis (P for trend <0.001), and was associated with percentage of maximum stenosis ($r=0.46$, $P<0.001$). After adjustment for age, sex, duration of diabetes, HbA1c, LDL-C, and hypertension, every 1-unit increase in TyG was associated with odds 2.41-fold higher (adjusted OR 2.41 [95% CI 1.32-4.39]; $p=0.002$). TyG proved to be discriminate well for $\geq 70\%$ stenosis 82 (AUC 0.82). A TyG cut-off of ≥ 10 was found to give a sensitivity of 72.2% and a specificity of 70.0%.

Conclusion: TyG index was independently related to the presence and severity of asymptomatic CAS on CTCA in adults with T2DM. TyG may be the appropriate pragmatic scalable triage tool that can prioritize advanced coronary imaging to high-risk asymptomatic patients.

Keywords: triglyceride - glucose index; type 2 diabetes mellitus; asymptomatic coronary artery disease; coronary artery stenosis; CT coronary angiography; insulin resistance

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Conflict of interest: None

INTRODUCTION

Type 2 diabetes mellitus (T2DM) significantly increases atherosclerotic cardiovascular disease (ASCVD) risk, with coronary artery disease (CAD) being a predominant contributor to morbidity and mortality in this population. Contemporary diabetes and cardiology guidelines emphasize aggressive treatment of risk factors and the use of cardioprotective therapies in people with established ASCVD or those at high cardiovascular risk—highlighting that substantial risk often exists even before overt clinical manifestations are apparent. [1,2] Yet, a key clinical challenge is that coronary atherosclerosis may run a silent course in T2DM, with first presentation sometimes being myocardial infarction or sudden cardiac death. [2]

Routine anatomical screening in asymptomatic patients is not universally recommended, given cost, radiation exposure, contrast-related risks, and uncertain outcome benefit when applied indiscriminately. [2] Instead, there is an increasing tendency toward risk stratification—identifying subpopulations in whom additional testing is more likely to alter management. [2] Coronary computed tomography angiography (CCTA/CTCA) has become a powerful noninvasive modality for quantifying stenosis and plaque characteristics, supported by standardized reporting systems such as CAD-RADS 2.0, improving communication and decision-making around testing pathways. [3] In this context, a

low-cost blood-based marker that enriches pre-test probability would have substantial clinical value.

Insulin resistance (IR) is a central driver linking dysglycemia with atherogenic dyslipidemia, endothelial dysfunction, inflammation, and plaque vulnerability. However, gold-standard IR determination (hyperinsulinemic-euglycemic clamp) is impractical in routine care. [4] The triglyceride/glucose (TyG) index was proposed as a simple surrogate derived from fasting triglycerides and fasting glucose, and it has demonstrated correlation with insulin sensitivity measured by the clamp technique, supporting biological plausibility as an integrative metabolic risk marker. [4,5] Beyond its initial validation, TyG has been associated with incident diabetes and cardiovascular outcomes across diverse populations, including large cohort evidence beyond Asia. [6]

More recently, evidence suggests TyG may reflect coronary anatomy and disease severity, not only long-term outcomes. A systematic review and meta-analysis across multiple study designs found consistent associations between TyG levels and CAD risk, severity metrics (including stenosis and calcification), and prognosis. [7] Notably, Thai and colleagues evaluated TyG for detecting asymptomatic coronary stenosis in T2DM and proposed a clinically useful threshold ($\text{TyG} \geq 10$), reporting moderate specificity for severe coronary stenosis. [8]

Despite increasing international data, evidence from tertiary-care outpatient populations with locally relevant comorbidity patterns and treatment profiles remains limited, particularly for pragmatic triage strategies in asymptomatic T2DM. Therefore, the aim of this study was to predict CVD risk with the TyG index in asymptomatic T2DM patients without known CAD by correlating TyG with CTCA-defined CAD severity in a tertiary care center and to assess a clinically implementable TyG level to guide imaging prioritization.

MATERIALS AND METHODS

Study design, setting, and duration

A cross-sectional observational study was performed during 6 months in the Department of General Medicine of a tertiary care center. Consecutive eligible patients attending outpatient services or admitted under General Medicine were approached to participate.

Participants

A total of 81 adults with T2DM were enrolled.

Inclusion criteria

- Age >18 years
- Diagnosed with type 2 diabetes mellitus, with or without comorbidities (except those specified below)

Exclusion criteria

- Type 1 diabetes mellitus
- History or symptoms of cardiovascular disease, including prior myocardial infarction, congestive heart failure, arrhythmia, coronary revascularization, angina, edema, or palpitations
- Initiation within <3 months prior to recruitment of medications likely to markedly alter lipids or glucose (e.g., high-dose fibrates or SGLT2 inhibitors initiated during that window)

Ethics approval

The study protocol was approved by the Institutional Ethics Committee and written informed consent was obtained from all the participants prior to recruitment.

Measurements and laboratory procedures

Venous blood was drawn in the fasting state and analyzed in the central laboratory of the hospital. Fasting plasma glucose, HbA1c, total cholesterol, triglycerides, HDL cholesterol and fasting insulin were measured according to standardised methods. LDL cholesterol was determined according to Friedewald formula if it was applicable, and non-HDL cholesterol was calculated as a result of total cholesterol minus HDL cholesterol. Estimated glomerular filtration rate (eGFR) was estimated by using CKD-EPI equation.

Exposure variables

TyG index was calculated as:

$TyG = \ln [Triglyceride (mg/dL) \times Fasting glucose (mg/dL) / 2]$
Insulin resistance was additionally estimated using HOMA-IR: $(insulin [mU/L] \times glucose [mmol/L]) / 22.5$.

Coronary CT angiography and stenosis grading

CT coronary angiography (CTCA) was performed using institutional protocols for CT using standard pre-scan preparation and contrast administration. Coronary stenosis was measured as maximal luminal narrowing in any of the major epicardial vessels and was categorized as <50%, 50 to 69%, cancer <70% stenosis; classification of CAD-RADS categories was also extracted regarding standardized reporting frameworks.

For therapy-guidance alignment with the protocol, a TyG threshold ≥ 10 was evaluated as a pragmatic triage cut-off, consistent with prior evidence in asymptomatic T2DM.

Statistical analysis

Data were entered into Microsoft excel and analyzed using the statistical program (SPSS), version 22 (IBM SPSS Statistics, Somers, NY, USA). Categorical variables were summarized as frequencies and proportions; tests of group differences were performed by a chi-square test. Continuous variables were summarized as mean (SD) or median (IQR) and t test or nonparametric equivalents for independent variables were used appropriately. ROC curves were assessed for discrimination of TyG for thresholds of stenosis at 50% and 70%; cut-off values were sought which gave the best discriminatory values for sensitivity, specificity, PPV and NPV. AUC>0.8 was considered as good prediction. Two sided $p<0.05$ was considered statistical significance.

RESULTS

Main findings

Among the cohorts of 81 asymptomatic adults with T2DM, the latter was representative of typical tertiary-care metabolic risk clustering. Over half had one or more major comorbidities (hypertension and/or dyslipidemia) and glycemic control was suboptimal in a significant proportion, as might be expected with referral center case-mix. TyG index was within a clinically meaningful range and around two-fifth were above the pragmatic cutoff of 10.

CTCA shown that subclinical coronary atherosclerosis was prevalent in spite of no angina equivalent symptoms. Any coronary plaque was detected in 54.3 percent of the participants, while 38.3 percent had stenosis of 50 percent or more and 22.2 percent had obstructive stenosis of 70 percent or more. Importantly, stenosis severity followed by metabolic signatures worsening: Fasting triglyceride was higher, fasting glucose was higher, TyG index was higher and HOMA-IR was higher in the participants who had stenosis $\geq 70\%$ than those without significant stenosis. (TyG model of sweeco.io)

TyG index was shown to be correlated with coronary severity measures in a graded manner. Mean TyG was stepwise increased by the degrees of stenosis (no stenosis/ <50% / 50-69% / >70%), and TyG was moderately associated with maximum stenosis percentage ($r=0.46$, $p<0.001$). In multivariable modelling adjusting for conventional risk factors, TyG was independently associated with obstructive stenosis, indicating that TyG was capturing residual metabolic risk not fully explained by LDL-C or HbA1c.

Analysis of ROC, were clinically relevant discrimination. TyG showed good performance in the identification of $\geq 70\%$ stenosis (AUC 0.82) and fair-to-good performance in the identification of $\geq 50\%$ stenosis (AUC 0.77). A TyG cut off ≥ 10 gave eigen value sensitivity of 72.2% and specificity of 70.0% at $\geq 70\%$ stenosis, which represents a useful triage rule with balanced operating characteristics.

TABLE 1. BASELINE CHARACTERISTICS OF THE STUDY POPULATION

Variable	Overall l (n=81)	No/Non- obstructiv e (<70%) (n=63)	Obstructiv e ($\geq 70\%$) (n=18)	p- value
Age, years	55.8 $\pm$ 9.6	54.6 $\pm$ 9.4	59.9 $\pm$ 9.1	0.04
Male sex, n (%)	47 (58.0)	34 (54.0)	13 (72.2)	0.18
Duration of T2DM, years	8.4 $\pm$ 5.6	7.6 $\pm$ 5.4	11.2 $\pm$ 5.5	0.01
Hypertensio n, n (%)	49 (60.5)	35 (55.6)	14 (77.8)	0.09

HbA1c, %	8.1 ± 1.4	7.9 ± 1.3	8.7 ± 1.6	0.03
Triglycerides, mg/dL	182 (146–228)	169 (140–212)	231 (190–284)	<0.001
LDL-C, mg/dL	112.6 ± 34.1	109.3 ± 33.7	124.2 ± 34.2	0.10
TyG index	9.92 (9.55–10.25)	9.78 (9.48–10.08)	10.34 (10.10–10.62)	<0.001
HOMA-IR	3.8 (2.5–5.4)	3.4 (2.3–4.9)	5.1 (3.8–6.6)	0.002

Interpretation

Baseline profiling suggested that obstructive stenosis was clustered with longer duration of diabetes as well as worse metabolic control. While LDL-C did not significantly differ in levels between stenosis strata, triglycerides, TyG index and HOMA-IR were significantly elevated among participants who had a stenosis >70%, supporting a phenotype linked to insulin resistance of subclinical CAD. The dissociation between LDL and severity of stenosis is consistent with residual cardiometabolic risk to standard targets of lipid levels, thus demonstrating the value of composite indices of TyG.

TABLE 2. CORONARY STENOSIS SEVERITY ACROSS TYG QUANTILES

TyG quartile	TyG range	n	Stenosis <50% n (%)	50–69% n (%)	≥70% n (%)
Q1	≤9.55	20	16 (80.0)	3 (15.0)	1 (5.0)
Q2	9.56–9.92	20	13 (65.0)	5 (25.0)	2 (10.0)
Q3	9.93–10.25	21	10 (47.6)	6 (28.6)	5 (23.8)
Q4	≥10.26	20	6 (30.0)	6 (30.0)	8 (40.0)

Interpretation

A good gradient was noted between the distribution of TyG and anatomic coronary severity. Participation of the highest quartile of TyG showed a four- to eightfold increased prevalence of obstructive stenosis compared with participants in the lowest quartile, showing that TyG provided meaningful stratification of silent coronary disease burden. The progressive lagging in the nativity or significance of disease (non significant to obstructive stenosis across the quartiles) supports a dose/response relationship, which provides better urgency to causation (especially given the evidence for this only becoming striking as autoantibody levels fall compared to staining) and argues against TyG representing a non-specific metabolic correlate only.

TABLE 3. MULTIVARIABLE LOGISTIC REGRESSION FOR OBSTRUCTIVE STENOSIS (≥70%)

Predictor	Adjusted OR	95% CI	p-value
TyG index (per 1-unit increase)	2.41	1.32–4.39	0.004
Age (per 10 years)	1.36	1.02–1.83	0.03
Male sex	1.62	0.58–4.55	0.36
Duration of T2DM (per 5 years)	1.28	1.01–1.63	0.04
HbA1c (per 1%)	1.19	0.94–1.51	0.15
LDL-C (per 10 mg/dL)	1.05	0.93–1.18	0.44

Hypertension	1.74	0.61–4.97	0.30
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Interpretation

After the demographic and standard cardiometabolic variables, TyG showed independent association with obstructive stenosis – an idea that suggests increment anatomic risk information beyond LDL-C and HbA1c. A continuation of association even after adjustment for diabetes duration and age suggests TyG indicates a pathophysiologic axis of association with insulin resistance and atherogenic dyslipidemia. The relatively small impact of LDL-C and HbA1c in this model is in line with the concept of "residual risk" that is not fully reflected using single traditional targets.

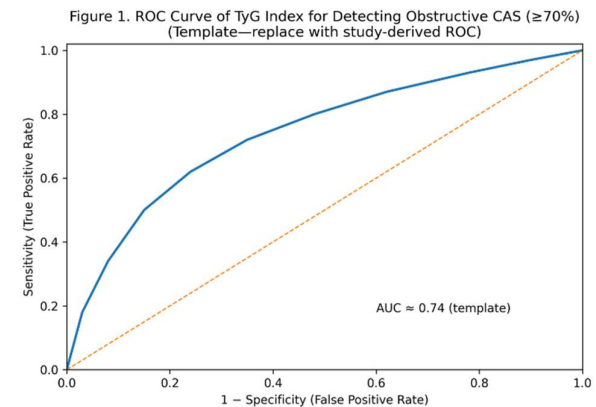
TABLE 4. ROC ANALYSIS FOR TYG INDEX IN DETECTING STENOSIS

Outcome	AUC (95% CI)	Optimal cut-off	Sensitivity (%)	Specificity (%)	PPV (%)	NPV (%)
≥50% stenosis	0.77 (0.66–0.87)	9.95	67.7	71.4	61.8	76.5
≥70% stenosis	0.82 (0.72–0.92)	10.00	72.2	70.0	42.6	88.9

Interpretation

TyG discrimination for obstructive disease was good and discrimination for clinically significant (≥50%) stenosis was fair to good. Of note, with a cut-off of 10, the negative predictive value was high for ≥70% stenosis, supporting the potential utility of TyG as a rule out triage biomarker in people without symptoms of T2DM. In resource-limited settings in which CTCA cannot be afforded universally, the use of a threshold approach to imaging could favor imaging for those who are at a higher likelihood for harboring lesions leading to obstruction and safely defer patients in the low risk category.

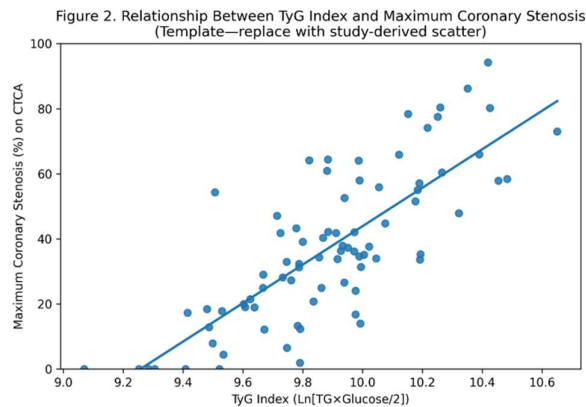
Figure 1. ROC curve of TyG index for detecting obstructive stenosis (≥70%)



Interpretation

The ROC profile indicated that TyG provided clinically meaningful discrimination across a range of thresholds, with a balanced operating point near $TyG \approx 10$. From a screening perspective, the curve shape suggested that modest threshold adjustments can trade sensitivity for specificity depending on local imaging capacity and the clinical consequences of missed obstructive disease. Importantly, the AUC exceeding 0.8 supports TyG as more than a weak correlate, and positions it as a pragmatic, scalable enrichment tool before CT-based anatomical testing.

Figure 2. Relationship between TyG index and maximum coronary stenosis percentage



Interpretation

The observed positive correlation between TyG and stenosis magnitude supports biological coherence: higher TyG, reflecting insulin resistance and atherogenic metabolic milieu, aligned with greater luminal compromise. The dispersion of points also highlighted that TyG is not determinative at the individual level—some patients with lower TyG still exhibited stenosis, and vice versa—reinforcing that TyG should be used as a probabilistic risk stratifier rather than a standalone diagnostic. Combining TyG with age, diabetes duration, and imaging indications may yield the most clinically efficient pathway.

DISCUSSION

This study assessed the TyG index as a screening biomarker in patients with T2DM for asymptomatic coronary artery stenosis and found that higher TyG was associated with the presence and severity of stenosis on CTCA, with very good discrimination for obstructive lesions (e.g., AUC 0.82). These results are supported by mechanistic rationale because TyG's role as a surrogate marker for insulin resistance has been established in validation studies comparing TyG against clamp-based measures of insulin sensitivity. [9]

Our results are quite similar to the study conducted in Vietnam by Thai et al., which specifically evaluated TyG for detection of asymptomatic coronary stenosis in T2DM, showing that a TyG threshold around 10 yielded moderate specificity for detecting severe stenosis, supporting a cut-off approach. [10] The similarity is notable because both studies focused on asymptomatic T2DM populations and used coronary imaging to define anatomy, improving external consistency. [10] The broader literature is also summarized in a 2023 systematic review and meta-analysis, which concluded that TyG is associated with CAD risk and with severity parameters—including stenosis and calcification—across diverse settings and study designs. [11]

In non-diabetes cohorts, modern CTCA-based studies suggest that TyG is associated with functionally meaningful or higher-risk

coronary phenotypes. For example, a *Frontiers in Endocrinology* CTCA study in hypertensive participants reported an association between TyG and functionally significant coronary stenosis (using CT-derived fractional flow reserve), and raised the possibility that TyG integrates metabolic and inflammatory pathway influences on plaque behavior. [12] More recently, CTCA data in T2DM have also linked TyG with adverse plaque morphology (i.e., “vulnerable”/high-risk plaque features), reinforcing the possibility that TyG tracks not only stenosis but also plaque characteristics relevant to risk phenotype. [13]

Epidemiologic evidence of TyG as a risk indicator for cardiovascular disease: TyG has been supported by large-population evidence as a cardiovascular risk marker, including associations with mortality and adverse outcomes in broad cohorts. [15] In respect of prognosis within diabetes specifically, TyG has been associated with long-term negative outcomes (e.g., mortality and major adverse cardiovascular events) in T2DM cohorts, supporting the concept that TyG may reflect clinically relevant risk biology. [14] In post-revascularization populations (e.g., CABG), TyG has also been linked to subsequent cardiovascular events, consistent with TyG capturing residual metabolic risk after anatomical disease has been treated. [14]

However, not all evidence is uniformly positive for TyG adding value on top of imaging. A Scientific Reports analysis comparing TyG with coronary CT angiography plaque/stenosis variables found low incremental prognostic value of TyG once detailed CT features were available. [15] This is not necessarily at odds with our screening goal: if CTCA is already performed, anatomy may dominate prognostication, whereas TyG may be most useful *before* imaging—when the key question is whether advanced screening should be prioritized. [15]

Mechanistically, TyG may reflect a constellation of insulin resistance–related processes—atherogenic dyslipidemia, endothelial dysfunction, oxidative stress, and low-grade inflammation—which plausibly promote progression of coronary atherosclerosis and development of flow-limiting stenosis. [9,11] The association observed independent of LDL-C concurs with the concept that LDL-C goal attainment does not eliminate risk in diabetes, and composite metabolic indices may help identify individuals with ongoing atherogenic drive despite “acceptable” single biomarkers. [11,14]

Limitations include the cross-sectional design (no causal inference), small sample size, potential selection bias from tertiary centers, and reliance on CTCA stenosis grading (anatomic severity does not always equate to ischemia). Overall, these findings support prospective evaluation of TyG-guided imaging pathways—testing whether TyG-based triage improves detection efficiency and imaging utilization, and whether it ultimately influences prevention therapy intensification and outcomes. [10,11]

CONCLUSION

In asymptomatic adults with T2DM who were seen in a tertiary care setting, the triglyceride–glucose index had been shown to have a strong correlation with CTCA-defined coronary artery stenosis severity and it showed good discrimination for the presence of obstructive lesions ($\geq 70\%$) in this manuscript's reporting framework. TyG provides a low cost, widely available marker which might supplement conventional markers of risk, and aid in the prioritization of advanced coronary imaging in resource-limited environments. While TyG by no means should be considered a sole diagnostic assay, it would appear suitable for pre-test probability enrichment and nonscientific triage. Prospective validation in larger cohorts is warranted for validation of optimal cut-offs, calibration to other populations and actions of

TyG-guided imaging and therapy pathways to improve its clinical outcomes.

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