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Pathophysiological Insights and Prognostic Value of the Triglyceride-Glucose Index in Patients with Chronic Total Occlusion

ABSTRACT

The triglyceride-glucose (TyG) index is a surrogate marker of insulin resistance (IR) associated with atherosclerosis, endothelial dysfunction, and cardiovascular disease (CVD). Chronic total occlusion (CTO) presents major clinical challenges, especially in patients undergoing percutaneous coronary intervention (PCI). This narrative review explores the role of the TyG index in predicting CTO development and adverse cardiovascular outcomes. A literature review of studies assessing the association between the TyG index and CTO, PCI outcomes, and contrast-induced nephropathy (CIN) was conducted. Pathophysiological mechanisms linking IR, TyG, and CTO progression were evaluated, and the predictive utility of the TyG index in risk stratification and post-PCI complications was analyzed. Multiple studies show that a higher TyG index is strongly associated with increased CTO risk, poor collateral circulation, CIN, and adverse outcomes after PCI. Elevated TyG values were independently predictive of impaired collateral formation in diabetic and non-diabetic patients, with stronger effects in metabolically vulnerable subgroups. Individuals with higher TyG levels had a greater likelihood of developing CIN, with analyses confirming its role as an independent predictor. Long-term prognosis in CTO patients was also worse with elevated TyG, with higher rates of major adverse cardiovascular events. The TyG index demonstrated consistent predictive capability compared with other metabolic markers, supporting its potential as a low-cost tool for risk stratification. The TyG index is a cost-effective biomarker for predicting adverse outcomes in CTO patients. Its incorporation into clinical assessment may improve early risk identification and support individualized PCI planning.

Keywords: Chronic total occlusion, insulin resistance, percutaneous coronary intervention, triglyceride-glucose index

INTRODUCTION

Coronary chronic total occlusions (CTOs) are defined as completely occluded coronary arteries with Thrombolysis in Myocardial Infarction grade 0 flow for ≥3 months.[1] The CTOs are encountered in up to 20% of patients undergoing coronary angiography and pose significant therapeutic challenges. The majority are managed with guideline-directed optimal medical therapy (OMT) targeting anginal relief and reduction of major adverse cardiovascular events.[1,2] While randomized clinical trials have not demonstrated mortality benefit with CTO-percutaneous coronary intervention (PCI) compared with OMT alone, evidence supports successful PCI is associated with improved symptoms, quality of life, and left ventricular function.[2-4]

Despite technological advances, CTO-PCI remains technically demanding, with lower procedural success rates and higher complication risks compared with non-CTO interventions.[5,6] Identifying high-risk patients is critical to optimizing outcomes. The triglyceride-glucose (TyG) index, calculated as ln [fasting triglyceride (mg/dL) × fasting glucose (mg/dL)/2], has emerged as a reliable surrogate of insulin resistance (IR).[2,6] The TyG index has demonstrated prognostic value in non-obstructive cardiovascular disease (CVD) populations across multiple observational studies, supporting its clinical utility as a cardiometabolic risk indicator.[8]

REVIEW

Rupak Desai[1]
Abhishek Prasad[2]
Jai Sivanandan Nagarajan[3]
Ananth Guddeti[4]
Navya Mandalapu[5]
Darsh Tusharbhai Patel[6]
Warda Shahnawaz[7]
Sourabh Khatri[8]
Abdul Aleem[9]
Adil Sarvar Mohammed[10]
Muhammad Usman Ghani[11]

[1]*Independent Researcher, Outcomes Research, Atlanta, GA, USA*
[2]*Department of Anesthesiology and Perioperative Medicine, MD Anderson Cancer Center, Houston, TX, USA*
[3]*Department of Medicine, SUNY Upstate Medical University, Syracuse, New York, USA*
[4]*Department of Internal Medicine, SUNY Upstate Medical University, Syracuse, NY, USA*
[5]*Department of Internal Medicine, Bronxcare Health Sciences, Bronx, NY, USA*
[6]*Department of Medicine, Mercy Catholic Medical Center, Darby, PA, USA*
[7]*Internal Medicine, Mobile Infirmary Medical Center, USA*
[8]*Department of Medicine, Independence Health System, Greensburg, PA, USA*
[9]*Department of Pulmonary and Critical Care Medicine, Henry Ford Genesys Hospital Grand Blanc, Michigan, USA*
[10]*Department of Internal Medicine, Central Michigan University, Mt. Pleasant, MI, USA*
[11]*Central Michigan University College of Medicine, Mt. Pleasant, MI, USA*

Corresponding author:
Muhammad Usman Ghani
✉ ghani1mu@cmich.edu

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Insulin resistance promotes vascular injury through oxidative stress, endothelial dysfunction, and impaired collateral development, thereby exacerbating CAD progression.[18-20] Prior studies suggest that elevated TyG is associated with poor collateral circulation, contrast-induced nephropathy (CIN), and major adverse cardiac and cerebrovascular events (MACCE) in CTO patients.[11-16] In this review, we synthesize current evidence linking the TyG index with CTO pathophysiology, PCI outcomes, and long-term prognosis.

Methodology of Literature Review

A narrative review approach was used to synthesize current evidence addressing the relationship between the triglyceride–glucose (TyG) index and outcomes in patients undergoing PCI for CTO-PCI. A comprehensive literature search was performed using PubMed, Google Scholar, and Scopus to identify English-language studies published between January 2018 and January 2025, investigating the relationship between the TyG index and CTO-related outcomes. Search terms included "triglyceride–glucose index," "TyG," "chronic total occlusion," and "percutaneous coronary intervention." Studies were considered eligible if they examined associations between the TyG index and key CTO-related outcomes, including CIN, MACCE, collateral circulation, and mortality. Because this article serves as a narrative review rather than a systematic review, PRISMA-based procedures and formal risk-of-bias instruments were not utilized. Nonetheless, to enhance transparency, the databases searched, the key search terms, and the inclusion criteria for the studies were identified. Extracted data encompassed study design, sample size, TyG thresholds, outcome measures, and statistical indicators such as odds ratios (OR), hazard ratios (HR), and receiver operating characteristic area under the curve (ROC-AUC).

Across all included studies, the TyG index was derived using the standard formula: ln [fasting triglycerides (mg/dL) × fasting glucose (mg/dL) / 2]. Laboratory values for triglyceride and glucose were consistently reported in mg/dL and obtained after an overnight fast. The TyG index was analyzed either as a continuous variable (by tertiles or quartiles) or through predefined cutoffs (such as 8.65 for CIN; 9.10 for long-term cardiovascular risk), contributing to variability in reported optimal thresholds. Furthermore, differences in predefined cutoff thresholds (e.g., 8.65, 9.10, or tertile-based categorization) and variability in laboratory assay techniques likely contributed to the heterogeneity observed in reported findings. Given the heterogeneity of study designs and outcome definitions, findings were synthesized qualitatively to align with narrative methodology(Table 1).

HIGHLIGHTS

- In patients with chronic total occlusion (CTO), a higher triglyceride-glucose (TyG) index is associated with impaired coronary circulation.
- The TyG index serves as a reliable predictor for major adverse cardiac and cerebrovascular events and cerebrovascular accidents. For individuals undergoing CTO procedures, elevated TyG levels are independently associated with increased risk of developing contrast-induced nephropathy.
- As a practical and reproducible measure of insulin resistance, the TyG index offers superior clinical utility to the traditional Homeostatic Model Assessment of Insulin Resistance metric.
- The TyG is a cost-effective biomarker for pre-procedural risk stratification of CTO patients and adds strong prognostic ability.

RESULTS

Across the included studies, an elevated TyG index was consistently associated with adverse outcomes in patients with coronary CTO. In a 2025 cohort, Soner et al[13] reported that a TyG index ≥8.65 nearly doubled the risk of CIN (OR ≈2.17), although no independent association with mortality was observed. Zhu et al (2024)[11] and Gao et al (2021)[12] demonstrated a strong correlation between higher TyG values and impaired coronary collateral circulation, with ORs exceeding 5 and robust discrimination (AUC ~0.78). In terms of long-term prognosis, Yang et al[10], Song et al[9], Lin et al[15], and Li et al[14] consistently found that elevated TyG predicted MACCE. For example, Song et al[9] showed that patients with TyG >9.10 had a four-fold increased risk of cardiovascular death and a two-fold higher risk of MACCE. Xiao et al[16] also reported that higher TyG predicted increased likelihood of CTO presence in a large cross-sectional cohort. Collectively, these findings highlight that the TyG index provides strong predictive utility for CIN, impaired collateralization, and MACCE in CTO populations, with effect sizes generally larger in higher-risk subgroups such as diabetics and smokers.

DISCUSSION

Pathophysiological Insights

Insulin resistance, as reflected by the TyG index, plays a pivotal role in the cascade of pathophysiological changes that culminate in vascular dysfunction. Elevated TyG levels reflect a metabolic milieu of hyperglycemia and hypertriglyceridemia, which together promote vascular inflammation, oxidative stress, and endothelial injury.[7,18-20]

Insulin resistance, at the molecular level, disrupts PI3K/Akt signaling pathways responsible for activating endothelial nitric oxide (NO) synthase, leading to reduced NO production and impaired vasodilation.[18] In parallel, IR also increases endothelin-1 expression, further promoting vasoconstriction and exacerbating endothelial dysfunction.[18] Together, these alterations impair vascular homeostasis and contribute to early atherosclerotic changes.

Beyond these vasomotor effects, IR amplifies oxidative injury via the production of reactive oxygen species through activation of NADPH oxidase, leading to oxidative stress, lipid peroxidation, and progressive endothelial damage.[19] Consequently, this oxidative burden upregulates vascular adhesion molecules, including ICAM-1 (Intercellular Adhesion Molecule-1) and VCAM-1 (Vascular Cell Adhesion

Table 1. Association Between Triglyceride-Glucose Index and Clinical Outcomes in Chronic Total Occlusion: Summary of Observational Studies

Study	Sample Size	Study Population	Study Type	Follow-up	TyG Level	Main Results	Outcomes	Key Metrics
Soner et al, 2025	218	CTO patients undergoing PCI	Retrospective cohort	96 months	Cutoff ≥ 8.65	TyG index (Cutoff ≥ 8.65) was significantly associated with increased risk of CIN and mortality in CTO patients undergoing PCI.	CIN, Mortality	OR = 2.17
Zhu et al, 2024	681	CTO patients with varying glucose metabolism states	Cross-sectional	Cross-sectional—no follow-up	Continuous variable	Higher TyG index was associated with poor collateral circulation. ROC-AUC = 0.779 for prediction.	Collateral circulation	OR = 5.104, AUC = 0.779
Xiao et al, 2024	2691	General CTO patients	Cross-sectional	Cross-sectional – no follow-up	Quartiles (Q1-Q4)	Elevated TyG index was linked to a 2.09-fold increased risk of CTO. ROC-AUC = 0.643.	CTO Risk	OR = 2.09, AUC = 0.643
Yang et al, 2023	331	CTO patients post-successful PCI	Prospective cohort	44 months	Tertiles (T1-T3)	TyG index was an independent predictor of MACCE in CTO-PCI patients. ROC-AUC = 0.677.	MACCE	HR = 2.54, AUC = 0.677
Song et al, 2023	2740	CTO patients with angina	Prospective cohort	36 months	Cutoff >9.10	High TyG index (>9.10) predicted CV death/TVMI (HR = 4.23, $P < .001$) and MACCE (HR = 2.47, $P < .001$).	CV Death, TVMI, MACCE	HR = 4.23 (CV death), HR = 2.47 (MACCE), AUC = 0.623
Lin et al, 2023	681	CTO patients with type 2 diabetes mellitus	Retrospective cohort	24 months	Continuous	TyG index was significantly correlated with adverse events (HR = 1.699, $P = .001$). Adding TyG improved model AUC from 0.663 to 0.693.	Adverse Events	HR = 1.699, AUC = 0.693
Li et al, 2022	652	CTO patients undergoing revascularization	Retrospective cohort	22.8 ± 3.84 months	Tertile	TyG index (highest tertile) was associated with a 2.09-fold increase in MACCE risk.	MACCE	HR = 2.09
Gao et al, 2021	1093	CAD patients with CTO lesions	Cross-sectional	Cross-sectional—no follow-up	Tertile	High TyG index was strongly linked to impaired collateralization (OR = 5.72, $P < .001$). ROC-AUC demonstrated superior risk prediction.	Collateral circulation	OR = 5.72

CAD, coronary artery disease; CIN, contrast-induced nephropathy; CTO, chronic total occlusion; CV, cardiovascular; HR, hazard ratio; OR, odds ratio; MACCE, major adverse cardiac and cerebrovascular events; PCI, percutaneous coronary intervention; ROC-AUC, receiver operating characteristic-area under the curve; T2DM, type 2 diabetes mellitus; TVMI, target vessel myocardial infarction; TyG, triglyceride-glucose index.

Molecule-1), which facilitate monocyte adhesion and promote vascular inflammation.[20] These inflammatory and oxidative processes combined accelerate the formation and destabilization of atherosclerotic plaques—features that are characteristically pronounced in CTO lesions.

Furthermore, the development of collateral vessels, a key determinant of CTO prognosis, becomes severely limited in patients with IR. Diminished NO bioavailability, impaired macrophage-dependent arteriogenesis, and dysregulated angiogenic signaling collectively restrict the ability of collateral networks to mature fully.[18] These mechanistic impairments parallel clinical findings demonstrating that higher TyG index values correlate with poorer collateral vessel formation.

Finally, the CTO microenvironment exhibits persistent chronic hypoxia, which activates HIF-1α (Hypoxia-induced Factor 1 Alpha) and promotes inflammatory signaling, endothelial apoptosis, and microvascular rarefaction.[21] In the setting of IR, angiogenic responses are further compromised by attenuated VEGF signaling, making collateral development even more difficult.[21] The convergence of these mechanisms explains why CTO patients with elevated TyG index levels exhibit impaired collateralization, increased susceptibility to CIN, and worse cardiovascular outcomes.

Application in Special Populations

The TyG index shows variable relationships with cardiovascular risk across patients who have CTO. The metabolic characteristics of certain groups lead to a greater impact on these populations. For instance, individuals with type 2 diabetes mellitus (T2DM) typically present with more severe IR and endothelial dysfunction. These overlapping abnormalities may intensify the vascular pathways through which TyG influences CTO outcomes. Research findings from Lin et al. demonstrate that diabetic CTO patients with higher TyG values experience worse prognosis than non-diabetic patients with similar TyG levels.[15] A similar trend is seen in patients with pre-diabetes due to impaired regulation of glucose and lipid metabolism in this population. Even though these individuals have not developed overt diabetes, their metabolic profiles predispose them to poor collateral growth and higher post-PCI complications. In this context, the TyG index shows potential to function as an initial indicator of vascular vulnerability.[22]

Smoking and tobacco exposure promote oxidative stress and endothelial inflammation, further potentiating the metabolic disturbances reflected by the TyG index. When combined with IR, these smoking-related vascular insults contribute to impaired collateral development and accelerated atherosclerotic progression. Consistent with this, several studies have reported stronger associations between TyG and early signs of atherosclerosis among active smokers.[23]

Moreover, patients with obesity and metabolic syndrome—characterized by chronic low-grade inflammation, dyslipidemia, and visceral adiposity—cumulatively demonstrate elevated TyG values, which in turn promote vascular damage and accelerate atherosclerosis. Research by Guo et al[24] indicates that these factors may explain why CTO occurs more frequently in this subgroup and why revascularization success rates remain poor in these individuals.

Finally, older adults exhibit a distinct risk profile in which TyG may hold added prognostic relevance. The combination of diminished endothelial regenerative capacity and increased arterial stiffness predisposes this population to more pronounced TyG-related vascular injury. Research demonstrates that older adults with elevated TyG values show stronger correlations with cardiometabolic risk and microvascular damage.[25]

Taken together, these subgroup differences underscore the importance of interpreting TyG within a broader clinical context. The TyG index provides its best prognostic information when healthcare providers use it to make decisions about patient treatment based on their complete medical situation and metabolic health. The understanding of TyG performance in various patient populations will lead to better application of this test for CTO risk assessment and personalized treatment approaches.

Heterogeneity in Triglyceride-Glucose Thresholds Across Studies

The TyG thresholds which researchers measured in their studies displayed significant differences because their studies used different participant groups and research techniques. Among these influences, the most significant factor seems to be the characteristics of the population. Most existing research data originates from East Asian study groups, which show distinct metabolic patterns, visceral fat distribution, and IR rates compared to Western populations. As a result, the initial values between groups will affect how TyG values distribute and which threshold defines significant clinical risk.[26]

In addition to population differences, the way different conditions appear together in patients determines how their symptoms will reach the threshold for diagnosis. Research shows that people with diabetes, obesity, and metabolic syndrome often start with elevated TyG values, which makes it harder to distinguish between high-risk groups and requires separate risk threshold values for different studies. Furthermore, the risk of vascular disease which TyG measures becomes more complex because of how smoking and physical inactivity affect a person's lifestyle.

Methodologic differences also contribute to threshold variation. The research methods used in studies create additional variations between the groups investigated. Research studies about TyG used two different approaches—either treating the variable as a continuous value or dividing it into specific categories through tertiles, quartiles, and predefined cutoff points—which resulted in various threshold values. In turn, the reported effect sizes in studies become inconsistent because of different methods used to confirm fasting status, perform laboratory tests, and establish study endpoints, including collateral quality, CIN, and MACCE.

Given these variations, the present review uses study-defined TyG thresholds because the research methods and narrative approach of this review prevent us from creating standardized cutoff points. The evaluation of TyG results between different groups requires knowledge about all factors that affect test results. Looking ahead, future research should perform combined studies to create reference values that will work for different population groups.

Limitations of Triglyceride-Glucose Index

The TyG index provides convenient benefits as an easy-to-use indicator of IR, but healthcare providers need to understand its restrictions when using this test for CTO patients. The TyG values become unstable because of transient factors such as dietary changes, acute clinical conditions or infectious processes, and prescription medications. These factors create transient changes in blood glucose and triglyceride levels.[26] A single measurement becomes unreliable for the same reason, as these short-term influences cause TyG values to fluctuate. The TyG index reflects only hepatic IR and does not measure peripheral insulin sensitivity or β-cell function, which restricts its ability to represent the entire metabolic profile.[27]

The results of TyG tests between studies depend on differences in study populations. The metabolic health of patients, their comorbid conditions, and their abdominal fat distribution patterns influence their initial TyG test results, particularly because most research data originate from East Asian participants.[26] The available data from these cohorts create challenges for determining clinical risk thresholds, as different populations display varying metabolic characteristics. The research included observational studies and single-center investigations, which introduce potential risks from confounding factors and publication bias.

The TyG index operates as a standalone measurement, but researchers need to use it together with other variables to achieve its full potential. Research indicates that TyG measurements combined with additional metabolic indicators such as glycated hemoglobin, non-high-density lipoprotein cholesterol, and specific inflammatory markers enhance risk evaluation by detecting multiple metabolic dysfunction pathways.[24,28] However, current evidence does not provide sufficient data regarding the specific performance of these combination tests in patients with CTO.

The results from these studies demonstrate that healthcare providers need to approach TyG value interpretation with careful consideration in clinical practice. Research should further elucidate whether different population groups require their own specific TyG threshold values and should systematically evaluate the predictive value of TyG when combined with established cardiovascular risk indicators.

Clinical Utility of Triglyceride-Glucose

In CTO patients, metabolic dysfunction contributes to impaired collateral circulation, a vital compensatory mechanism.[11,12] Hyperglycemia and hypertriglyceridemia inhibit NO production and promote endothelial inflammation, thereby reducing collateral vessel development. This is clinically relevant, as robust collateral circulation is a major determinant of long-term outcomes in CTO. Moreover, renal microvascular injury and oxidative stress offer plausible explanations for the strong association between TyG and CIN in PCI patients.[13]

High TyG is consistently associated with adverse CTO outcomes, including CIN,[13] poor collateralization,[11,12] and MACCE.[9,10,14,15] Soner et al[13] (2025) showed that a TyG index ≥8.65 independently predicted CIN and was associated with worse clinical outcomes in CTO patients, while Yang et al (2023)[10] and Song et al[9] (2023) confirmed TyG as a strong predictor of MACCE and cardiovascular death. Zhu et al (2024)[11] demonstrated its value in identifying poor collateral circulation, and Lin et al (2023)[15] showed its incremental benefit in diabetics when added to conventional risk models. Collectively, these findings suggest that TyG may serve as an adjunct prognostic marker across diverse CTO outcomes. Its low cost, simplicity, and reproducibility make TyG a practical tool for pre-procedural evaluation. Elevated TyG may identify patients who require enhanced hydration, nephroprotective measures, and intensified metabolic management.

The TyG index compares favorably with Homeostatic Model Assessment of Insulin Resistance (HOMA-IR) and stress hyperglycemia ratio. It avoids insulin measurement (HOMA-IR) and is less influenced by acute illness Stress Hyperglycemia Ratio (SHR).[7] Adding TyG to conventional models improves predictive accuracy, as demonstrated in diabetic CTO patients.[15]

Despite promising findings, important limitations remain. The included studies vary in design (retrospective vs prospective), TyG thresholds (≥8.65 vs. >9.10), and endpoint definitions, which introduces heterogeneity and complicates comparisons. Most studies were single-center and limited to Asian populations, raising concerns about external validity,[11,13] limiting generalizability. Furthermore, no interventional trial has confirmed that lowering TyG improves outcomes, reinforcing its role as a prognostic marker only at present.

Clinical and Public Health Implications

The TyG index's accessibility makes it appealing for integration into cardiovascular risk assessment, particularly in resource-limited settings.[7] An elevated TyG index is a significant predictor of increased heart failure risk in patients with H-type hypertension, with the effect being particularly pronounced among those with diabetes.[17] Large, multicenter prospective studies with standardized cut-offs and outcome definitions are needed. Interventional trials assessing whether lowering TyG through pharmacologic or lifestyle modification improves CTO outcomes are also warranted.

Future Directions

Future research should focus on three key areas: (1) standardization of TyG thresholds across populations; (2) validation in large, multicenter, prospective cohorts with diverse ethnic representation; and (3) interventional studies testing whether lowering TyG through pharmacologic (e.g., GLP-1 receptor agonists, SGLT2 inhibitors, statins) or lifestyle

interventions improves CTO and PCI outcomes. Additionally, subgroup analyses by sex, age, and comorbidity burden could provide more nuanced insights into TyG's role as a prognostic biomarker.

CONCLUSIONS

The TyG index is a cost-effective and reproducible biomarker with promising clinical relevance in CTO patients, although further validation is needed to establish its definitive role. Elevated TyG predicts impaired collateralization, CIN, and MACCE, highlighting its potential role in pre-procedural risk stratification and long-term prognosis. Future prospective validation and interventional studies are required before routine guideline integration.

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