RESEARCH PAPER

Triglyceride-Glucose Index as a Predictor of Cardiovascular, Renal, and Mortality Outcomes in Patients with Type 2 Diabetes and Metabolic Syndrome

Lu Yali[1], I Nyoman Ehrich Lister[2*], Florenly[3]

[1,2,3] Faculty of Medicine, Dentistry, and Health Sciences, Prima Indonesia University, Indonesia.

[2*] Corresponding Author: I Nyoman Ehrich Lister, Email: nyoman@unprimdn.ac.id

Received: 28th Feb, 2026 | **Revised:** 14th Mar, 2026 | **Accepted:** 4th Apr, 2026 | **Available Online:** 20th Apr, 2026

ABSTRACT

A low-cost insulin resistance surrogate, derived from standard measurements of fasting triglyceride and fasting glucose, has gained interest as the triglyceride-glucose (TyG) index. Its clinical utility is based on its simplicity, particularly in environments where direct measurement of insulin, clamp experiments, or repeat assessment by expert functions are not feasible. This review aims to assess the capacity of the TyG index to predict cardiovascular, kidney, and mortality outcomes in individuals with type 2 diabetes mellitus (T2DM) and those with high-risk cardiometabolic states highly compatible with metabolic syndrome. An intensive literature review was conducted using recent peer-reviewed articles, with preference given to primary cohort analyses and meta-analytic syntheses. The findings section has selected 15 main studies that were critically evaluated for their design, population, adjustment strategy, endpoint definition, and clinical transferability. On the whole, these findings indicate that there is a uniform pattern of association: increased TyG scores, and in part increased cumulative exposure, are associated with increased major adverse cardiovascular events, accelerated renal failure, and increased either all-cause or cardiovascular mortality. Prediction, however, is not uniform across populations. These are most strongly associated in cohorts with existing coronary disease, acute coronary syndromes, chronic kidney disease, inadequate glycaemic regulatory control, or a longer duration of diabetes. Methodological flaws also recur, including retrospective designs, single baseline measurements, uncontrolled confounding, low discrimination, and a concentration of data in East Asian or North American samples. The paper concludes that TyG can only be construed as an applied risk-enrichment tool, rather than a test of its own prognostic value. It is most useful when combined with traditional measures of glycaemic burden, kidney disease, obesity, and already established cardiovascular risk, whether or not it is like that.

Keywords: Triglyceride-glucose index; type 2 diabetes; metabolic syndrome; cardiovascular outcomes; chronic kidney disease; mortality.

How to cite this article: Yali L, Lister INE, Florenly. Triglyceride-Glucose Index as a Predictor of Cardiovascular, Renal, and Mortality Outcomes in Patients with Type 2 Diabetes and Metabolic Syndrome. Int J Drug Deliv Technol. 2026;16(30s):731-741. DOI: 10.25258/ijddt.16.30s.71

Source of support: Nil.

Conflict of interest: The authors declare no conflict of interest.

INTRODUCTION

Type 2 diabetes is considered to be one of the rapidly increasing chronic diseases around the globe. According to the most recent IDF Diabetes Atlas (as of 2024), there were 589 million adults aged 20 to 79 years with diabetes, which is 1 in 9 adults, and 3.4 million deaths from diabetes in the same year.[1] This is augmented by the concomitant increase in metabolic syndrome, an aggregation of abdominal adiposity, dysglycaemias, hypertension, dyslipidaemias, which significantly increases the risk of heart and kidney disease.[2,11,12] A recent global analysis estimated that the number of adults living with metabolic syndrome was 1.54 billion in 2023, and that the prevalence has risen

significantly over the past 20 years in both sexes.[2] Figure 1 summarises these background trends because the clinical interest in TyG is inseparable from this rapidly enlarging cardiorenal-metabolic disease pool.[1,2]

The clinical difficulty with the high-volume diabetes care is that insulin resistance is the core of the disease development, which is inaccessible to direct measurement. The physiological reference standard is the hyperinsulinaemic-euglycaemic clamp test, which is time-intensive, costly, and unsuitable for routine risk stratification. Other surrogate indices, such as HOMA-IR, require insulin assays, which cannot routinely meet good-quality standards. A simpler mechanism suggested is the TyG index, originally defined as the natural log of the product of fasting triglyceride and fasting glucose (divided by two), and it provides a more direct measure, on the assumption that both fasting hypertriglyceridemia and fasting hyperglycemia are caused by altered insulin signaling, hepatic hyperproduction, and metabolic rigidities.[4,5]

Such an argument has been extended in recent reviews. Insulin resistance facilitates endothelial dysfunction, oxidative stress, low-grade inflammation, ectopic lipid deposition, plaque instability, glomerular hyperfiltration, and progressive fibrosis.[5,6,11] These connections can give a biologically plausible case on why an increased TyG index may be an indicator of not only the incidence of diabetes but also subsequent cardiovascular outcomes, kidney failures, and mortality.[5,6] Figure 2 presents an adapted conceptual pathway linking elevated TyG to these outcome domains.[5,6,11] Notably, TyG cannot be viewed as a disease-specific biomarker. Instead, it seems to describe a common pathophysiological burden across cardiometabolic diseases.

The medical base has grown at a tremendous rate. Meta-analytic studies now indicate that higher TyG scores are directly associated with increased cardiovascular event risk in large cohort studies, and newer mortality syntheses indicate positive dose-response relationships for all-cause and cardiovascular mortality.[8-10] Important heterogeneity is, however, also reflected in these pooled findings. Associations seem to be greater in certain cohorts than in others and vary across cohorts by diabetes status, country income, pre-existing cardiovascular disease, age structure, and endpoint.[8-10] Such variation is of clinical significance, since a statistically significant association does not necessarily lead to useful discrimination or bedside changes in treatment.

A certain difference also exists in the application of TyG evidence in individuals with proven T2DM and metabolic syndrome. Lots of patients in diabetes departments already have a combination of several overlapping risk factors, such as obesity, hypertension, albuminuria, and previous atherosclerotic disease. The question of interest in that environment is not whether TyG is associated with unfavourable biology, but whether it provides useful prognostic information beyond conventional measurements such as HbA1c, estimated glomerular filtration rate, albuminuria, blood pressure, lipid profile, and established cardiovascular history.[3,33] Recent primary studies indicate that it might, especially when cumulative exposure or variability is factored in, but others have only found small discrimination or threshold effects.[9,10]

This paper thus examines TyG as a marker of cardiovascular and kidney outcomes and mortality in individuals with T2DM and phenotypes of overlapping metabolic syndromes. It is not merely a goal to provide recitations of findings, but rather to compare the methods used in studies, highlight strengths and weaknesses, and determine how far the existing body of literature can support the routine use of TyG in clinical risk stratification.

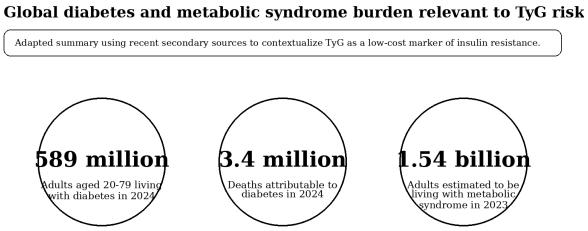

Figure 1. Global burden context for TyG-related risk assessment. Adapted from secondary data in the IDF Diabetes Atlas (2025) and Noubiap et al. (2025).[1,2]

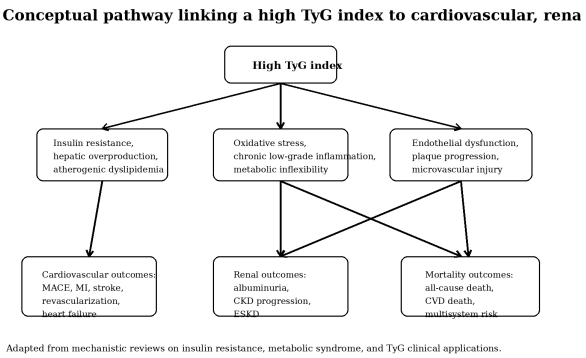

Figure 2. Conceptual pathway linking higher TyG to cardiorenal and mortality outcomes. Adapted from mechanistic reviews of insulin resistance, TyG, and metabolic syndrome.[5,6,11]

MATERIALS AND METHODS

A selective literature review was conducted to generalise current evidence on the predictive capacity of the TyG index in T2DM and in populations closely linked to metabolic syndrome. The sources of literature were defined as peer-reviewed journal articles indexed in major biomedical databases and publisher platforms, focusing on results published since 2020, as well as essential mechanistic or methodological articles required to build context. The combination of search terms included triglyceride-glucose index (TyG) and type 2 diabetes, metabolic syndrome, cardiovascular, renal, kidney, mortality, cohort, and outcomes. The framing of the epidemiological context and theoretical interpretation was based on meta-analyses, umbrella reviews, and guideline documents, but the foundations section was limited to 15 separate primary research papers. Of these 15 articles, none were reused as stand-alone evidence anchors for the paper.

The published studies prioritized those that described hard clinical endpoints, that is, major adverse cardiovascular events, chronic kidney disease progression, end-stage kidney disease, all-cause mortality, or cardiovascular mortality. Cohort analyses with multivariate adjustment, a defined endpoint, and a population applicable to T2DM or metabolic syndrome were considered a priority. Design, sample, follow-up, key findings, and methodological aspects that influence interpretability were the basis for data extraction: TyG was measured once or longitudinally, restricted cubic splines or subgroup interactions were used, and whether TyG added predictive power compared to standard models was assessed. Since the existing literature is not a homogeneous sample with respect to population type and outcome definitions, a formal meta-analysis was not conducted in this case. Rather, comparisons of studies were presented narratively, and assessments of benefits and constraints were made explicitly.

RESULTS

The 15 identified primary studies were published since 2020 and generally show a wide, uneven signal that increased TyG levels are associated with poor cardiovascular, renal, and survival outcomes in T2DM or cardiometabolically similar groups. The majority of the studies were observational cohort studies. They had some strong points: their large sample sizes, clinically meaningful endpoints, and increasing modeling complexity. Their limitations were a retrospective recruitment method, recruitment at one center, residual confounding, and incremental discrimination of modest intensity across multiple analyses. The lists of studies included are summarised in Table 1.

Study	Design and population	Main finding	Critical appraisal
Ma 2020	Retrospective PCI cohort, 776 T2DM + ACS	Composite CV events over median 30 months; highest vs lowest tertile HR 2.17	Tight post-PCI phenotype and hard endpoint. Single-setting and procedure-specific cohort limits generalisability.
Wang 2020	Prospective/observational ACS cohort, 2531 diabetes	TyG independently predicted future MACE in diabetes + ACS	Large, clinically relevant ACS population. More heterogeneity and residual treatment confoundin

			g than procedure-specific cohorts.
Pan 2021	Hospitalized T2DM cross-sectional study	Higher TyG is associated with nephric microvascular damage and vascular stenosis.	Captures complication clustering. A cross-sectional design is weak for prognosis and vulnerable to reverse causation.
Tai 2022	Post hoc ACCORD analysis, 5695 T2DM	Cumulative TyG predicted MACE, CVD death, and non-fatal MI; HR 1.59 for overall MACE in the highest exposure group.	Repeated measures strengthen causal ordering and reduce dilution bias. Requires serial data and remains observational.
Zhang H 2022	Retrospective CABG cohort, 386 T2DM	Higher TyG linked with 5-year MACE, death, and HF rehospitalization	Post-surgical relevance and nomogram development are useful. Small single-center cohort without external validation.
Lin 2023	Retrospective CTO after PCI cohort, 681 T2DM	High TyG associated with adverse CV events after CTO intervention	Targets a genuinely high-risk niche. Selection bias likely because only treated CTO cases were studied.
Duan 2023	Longitudinal T2DM renal cohort	Higher TyG is associated with CKD progression, especially in earlier-stage disease.	Temporal renal endpoint stronger than simple prevalence studies. Residual confounding by therapy and diet remains likely.
Zhao 2023	NHANES T2DM cohort, 2998 middle-aged/older adults	Threshold effect for mortality; TyG >=8.95 related to higher all-cause death and >=9.0 to higher CVD death	Nationally representative with spline modeling. Single baseline TyG may misclassify long-term exposure.
Abudu aini 2023	Ischemic cardiomyopathy + T2DM cohort	TyG predicted MACCEs in advanced	Useful in severe disease where simple

		diabetic heart disease	markers matter. A modest sample size reduces stability and transferability.
Liu 2024	Young US adults with diabetes, NHANES	U-shaped association between TyG and all-cause/CVD mortality	Important warning against linear assumptions. Mortality outcome susceptible to frailty and reverse causation at low TyG values.
Yu 2024	ACCORD renal analysis in T2DM	Linear association with worsening renal function and albuminuria after extensive adjustment	Strong covariate adjustment, including UACR and creatinine. The late renal failure endpoint is less robust.
Tao 2024	T2DM + coronary heart disease cohort	TyG-related metabolic indices predicted incident CV outcomes	Comparative design tests TyG against related markers. Also shows TyG is not always uniquely superior.
Sbriscia 2025	Long-term T2DM cohort	TyG predicted all-cause mortality and MACE; the strongest mortality signal in ages 57-74	Long follow-up and clinically meaningful endpoints. A specialized cohort may limit the applicability to primary care.
Kwak 2025	Nationwide diabetes cohort	TyG is associated with both CVD and mortality, especially with poor glycaemic control or longer duration	Large-scale supports precision and subgroup analysis. Administrative data reduces phenotypic depth.
Kim 2025	T2DM cohort evaluating ESRD	Higher TyG quartiles are associated with greater ESRD risk, with the association stronger with longer diabetes duration.	Hard renal endpoint is clinically compelling. Competing risk from death and treatment heterogeneity complicate interpretation.

Table 1. Summary and critical appraisal of the 15 primary studies included in the findings section.

The most common were cardiovascular outcome studies. Ma et al. found that in a targeted cohort of 776 T2DM patients with acute coronary syndrome, those in the highest TyG tertile were at greater risk of the composite cardiovascular endpoint than those in the lowest tertile after multivariate adjustment (HR 2.17, 95% CI 1.45 to 3.24).[13] The strength was that they were defined by a tight phenotype following the PCI, which enhanced internal validity; the narrow effect of external generalisability was caused by the specificity of the cohort used. A larger sample of 2,531 individuals, followed by Wang et al., once again identified TyG as an independent predictor of future major adverse cardiovascular events, and the broader inclusion criteria resulted in greater clinical heterogeneity. Collectively, these studies further substantiate the role of TyG in the risk enrichment of post-ACS. Still, they do not resolve whether TyG provides additional information beyond more recent revascularisation-era scores.

The cardiovascular cohorts followed by changes in exposure concepts. A 5,695-patient post hoc ACCORD study of patients free of prior events during the landmark period demonstrated that very high cumulative TyG exposure predicted overall major adverse cardiovascular events, cardiovascular death, and non-fatal myocardial infarction in the highest vs lowest exposure group, at a median of 5.09 years.[16] This is a strong study methodologically in the field since repeated measurements decrease regression dilution and are more indicative of chronic metabolic burden. Trade-offs: Cumulative exposure assumes serial data and thus is less practical to use in low-resource or fragmented care environments. The study by Zhang et al. included 386 patients following coronary artery bypass grafting and observed that 5-year MACE was higher in the high-TyG group, particularly in all-cause death and heart failure rehospitalization.[17] The fact that they added their nomogram by integrating TyG with left ventricular ejection fraction and NYHA class made them significant at the bedside. Still, the retrospective single-center design and the lack of external validation also limit confidence in their generalizability.

Similar trends are apparent in studies of anatomically or clinically complex coronary disease, which also reveal the frailty of observational prognostic studies. Lin et al. retrospectively recruited 681 T2DM patients who experienced a chronic TOTO following PCI, and the results showed that patients with a high TyG index had a higher number of adverse cardiovascular events during follow-up.[18] Clinical utility of the study is that CTO patients are truly high risk and difficult to stratify. Still, the study would also be susceptible to selection bias, as none of the revascularised patients were excluded from the cohort, according to Abuduaini et al. TyG was found to predict major adverse cardiac and cerebral events in ischemic cardiomyopathy with T2DM, which, once again, indicated value at an advanced disease stage.[21] In this case, the benefit was concentration on a severely ill diabetic subpopulation on which simple biomarkers are desirable; the cost was a relatively small, specialized sample population that does not yield as stable effect sizes. Tao et al. extended the question by comparing a range of triglyceride-derived metabolic indices in patients with T2DM and coronary heart disease. They identified TyG-related measures associated with incident cardiovascular outcomes.[24] The origin of the comparative design is a strength of the study as it not only demonstrates that single-marker superiority is by no means ensured, but it also tests the hypothesis about whether TyG is among the many correlated markers. The clinical implication of the message is that TyG is not a vacuum but rather part of a family of insulin-resistance surrogates.

Renal evidence has become more convincing in the last three years. When Pan et al. compared patients hospitalized with T2DM, they concluded that a higher TyG index was associated with nephric microvascular damage and lower-limb vascular stenosis.[15] Since this study involved clustering of complications as opposed to a single endpoint, it represented the systemic insulin resistance. It suffers because it is a cross-sectional inpatient context, which is problematic for association but less effective for prognosis. Duan et al. made a contribution to the field by showing that a higher TyG index was associated with the progression of chronic kidney disease in T2DM, with stronger correlations in earlier stages of CKD.[19] One of the most notable benefits of this work is that it captures the decline in renal status over time, rather than merely baseline albuminuria. Nevertheless, this paper continued to rely heavily on observational follow-up. It could not rule out the effects of treatment intensification, diet, or the duration of untreated dyslipidemia as confounding factors. Using the ACCORD cohort, Yu et al. demonstrated that TyG is linearly associated with adverse renal outcomes. In fully adjusted models, increasing TyG categories were all associated with deteriorating renal function (HR 1.24, 95% CI 1.14 to

1.35) and albuminuria (HR 1.19, 95% CI 1.03 to 1.37), though the renal failure endpoint showed a positive but non-significant directionality after complete adjustment.[23] This methodologically outmuscles most of the previous renal businesses since adjustment incorporated serum creatinine, urinary albumin-creatinine ratio, and renal renin-angiotensin system therapy. Despite this, baseline TyG was a one-occasion exposure, which can underrate or misrepresent chronic metabolic stress.

Kim et al. also extended renal prognosis to end-stage kidney disease. They discovered that higher TyG quartiles and longer duration of diabetes predisposed individuals to a higher risk of ESRD in T2DM.[27] This paper has clinical importance since ESRD is a patient-relevant and hard endpoint, rather than a biochemical surrogate. It is also consistent with the biological hypothesis that glomerular injury in patients with long-standing diabetes is mediated more rapidly by insulin resistance. However, ESRD researchers are at risk of death and treatment heterogeneity, and therefore the interpretation of outcomes must be approached with caution. In general, renal research has smaller sample sizes than cardiovascular literature, yet may frequently exhibit greater biological consistency, since the transition from insulin resistance to albuminuria, hyperfiltration, and fibrosis is conceptually straightforward. [3,5,11]

Mortality investigations yield a more nuanced picture, since a few indicate non-linear rather than purely linear connections. This NHANES-based study analyzed 2,998 middle-aged and older US adults with T2DM and found threshold effects. For each 1-unit increase in TyG, all-cause mortality was 33% more likely above TyG 8.95, and cardiovascular mortality was 50% more likely above TyG 9.0.20. The advantage is that Liu et al. subsequently showed that TyG had a U-shaped association with both all-cause and cardiovascular mortality among younger adults with diabetes, suggesting that very low values may reflect illness burden, malnutrition, or reverse causality.[22] This plays a vital remedial role in the face of the mere adaptations of higher is always worse. In comparison, Sbriscia et al. found that the TyG index predicted long-term all-cause mortality and MACE in patients with T2DM, with the strongest mortality signal among patients aged 57 to 74.25. The study is useful as it examines long follow-up in an adequately characterized cohort of patients with diabetes. However, the rather specific setting might not be representative of primary care. Kwak et al. reported nationwide cohort outcomes indicating that TyG was a predictor of both cardiovascular events and mortality in diabetes, and that patients with worse glycaemic regulation and those with longer disease duration had TyG.[26] The benefits of such a large-scale method are statistical power and subgroup exploration. The downside is that administrative or claims-linked cohorts tend to lose phenotypic detail, implying that residual confounding by obesity pattern, medication adherence, and inflammatory state is still likely.

Combined, the results demonstrate three general trends. First, TyG works most effectively in cohorts where preexisting vascular disease, kidney disease, or long-duration diabetes is present, and the underlying insulin-resistant phenotype is already clinically apparent.[13-27] cc. The advantages of a large-scale approach include statistical power and subgroup discovery. The disadvantage is that administrative or claims-based cohorts are likely to lose phenotypic detail, meaning they are still likely to have residual confounding from obesity patterns, drug adherence, and inflammatory state.

Combined, the results demonstrate three general trends. First, TyG is best applied to cohorts in which preexisting vascular disease, kidney disease, or long-duration diabetes is present, and the underlying insulin-resistant phenotype is already clinically evident.[16] Third, the mortality relationships, in contrast to the cardiovascular or renal relationships, tend to be more complex, with multiple studies revealing that the mortality relationships are thresholds or U-shaped relationships, not a simple monotonic gradient.[20,22] These are not the weaknesses of this literature only. They can also inform that death is a composite overall outcome that is susceptible to frailty, intensity of treatment, multimorbidity, and reverse causation more than more proximate outcomes (e.g., albuminuria or post-PCI events).

DISCUSSION

The analyzed evidence demonstrates that the TyG index correlates not only with negative outcomes in an epidemiological context but is also consistently linked to mechanisms that the current cardiorenal-metabolic theory can explain. The most explanatory model is insulin resistance. Impaired insulin signaling causes elevated hepatic glucose production, heightened adipose

tissue lipolysis, increased production of triglyceride-laden lipoproteins, and impaired endothelial nitric oxide signaling. In that regard, the TyG index is a practical aggregation of the identical terrain of metabolic dysfunction found by more sophisticated indices of insulin resistance.[4,5]

These cardiovascular findings are consistent with prior pooled evidence indicating that higher TyG levels predict incident or recurrent major adverse cardiovascular events in large populations.[8,10] The major studies examined in this case, however, indicate that the association is not as strong in all clinical scenarios. This signal is typically higher in secondary-prevention populations, such as ACS, PCI, CABG, CTO, or ischemic cardiomyopathy, than in less-selected populations.[13,14,17,18,21,24] That trend is medically reasonable. The multiplication of already active plaque, thrombosis, and ventricular dysfunction is likely increased by insulin resistance in advanced atherosclerotic disease, thereby making TyG more predictive. In less risky or less homogenous populations, the absolute effect of TyG can be diluted by alternative determinants like smoking, age, blood pressure, or access to medication.

The renal results are also consistent with prior theories and recent reviews that identify insulin resistance as the central driver of diabetic kidney injury.[3,5,6,11] Albuminuria and structural renal failure can be precipitated by hyperinsulinemia, glomerular hyperfiltration, lipid toxicity, and inflammatory signaling. The relative consistency of direction is striking during primary renal studies. Regardless of the endpoint of nephric microvascular or CKD improvement, albuminuria or ESRD, high TyG tended to follow the line of worse renal prognosis.[15,19,23,27] This, perhaps, lies at the core of the increased discussion of TyG in the context of the larger cardiovascular-kidney-metabolic construct, rather than as a diabetes-only biomarker.[3,6] Even now, however, renal evidence is primarily observational, which would be reinforced by analyses that explicitly address competing-risk, repeated-exposure, and change in treatment.

Evidence of mortality should be interpreted with greater attention. As a meta-analytic result over the last few years points to a positive net relationship between TyG and all-cause and cardiovascular death, the T2DM studies addressing this that were reviewed here show that non-linearity is widespread.[9,10,20,22,25,26] The two patterns are U-shaped or U-threshold, which probably indicate two counter-processes. Increased TyG levels worsen insulin resistance, dyslipidaemia and glycaemic toxicity and exert risks at higher levels. Frailties and sarcopenia, diminished caloric intake, liver dysfunction, or overtreatment may be more pertinent at inordinately low levels, particularly in older or frailer populations. This implies that TyG cannot be comprehended independently. It is not always a good sign to have a low value when accompanied by weight loss, an extreme chronic condition, or deteriorating renal functioning. The index has a clinical meaning specific to the situation, not general.

The best studies in this review were those that transcended the simple baseline-association method. Repeated measures and incremental predictive value demonstration are distinctive features of Tai et al., whereas ACCORD-based renal analysis properly adjusted covariates, except perhaps for cohort size.[16,23] These methods are more in line with the real-life development of diabetes risk. In comparison, most smaller retrospective studies were statistically convincing but less clinically certain, as they employed single-center recruitment, single baseline TyG measures, or composite endpoints, in which limited events such as rehospitalization or repeat revascularization predominantly occur.[17,18,21] These techniques are not ineffectual, but may be artificial and exaggerated in their functionality when moved out of the context in which they originated. The replication of problems across all the studies is not that TyG is linked to outcomes, but rather that it enhances decision-making to a level at which practice can be changed.

Practically speaking, TyG is appealing mainly due to its low cost, reproducibility, and construction out of standard laboratory data. This is mainly applicable in environments where insulin assays, state-of-the-art imaging, or new biomarker panels are not accessible.[1,4,6] It can be particularly applicable in triaging patients who seem to be stable based on HbA1c alone but still display a significant burden of residual dyslipidaemic and insulin-resistant risk. The existing literature, however, does not warrant replacing standard risk tools with TyG. The index ought to be used in combination with HbA1c, blood pressure, LDL cholesterol, the body habitus, albuminuria, estimated GFR, and previous cardiovascular status.[3,33] That is, TyG has an opportunity to enhance risk profiling, yet it cannot be a single gatekeeper to treatment escalation.

Several research priorities are based on this review. First, the cut-off values are not yet stable across studies, in part due to ethnic heterogeneity, variation in fasting states, and varying outcome structures.6,9,10 Second, many prediction models, which include TyG, have yet to be externally validated. Third, studies in populations with evident metabolic syndrome but without established cardiovascular disease are required since most current data are secondary prevention cohorts, based on hospitals. Fourth, intervention studies must be able to test the hypotheses that reducing TyG or reducing the metabolic burden it represents leads to quantifiable improvements in cardiorenal outcomes. So far, TyG can be regarded as a strong epidemiological and clinical indicator of a high metabolic load, but not as a universal prognostic threshold with agreed-upon action points.

Another interpretive observation is that TyG appears most effective in areas focused on metabolic syndrome. In numerous cases where formal metabolic syndrome was not the entry criterion, central obesity, hypertension, dyslipidemia, and impaired renal function were high burdens in many of the included cohorts.[2,11,12] It is this overlap that makes TyG potentially serve as a transitional marker between diabetes- and metabolic syndrome-centered risk assessment. Nevertheless, since the vast majority of studies failed to carefully stratify by formal criteria for metabolic syndrome, the incremental value of TyG in a diagnostically verified case of metabolic syndrome has not been fully determined.

CONCLUSION

The triglyceride-glucose index may be a useful and clinically applicable indicator of unfavorable cardiovascular, renal, and mortality outcomes in patients with type 2 diabetes and metabolic syndrome. Throughout the body of evidence reviewed, increased TyG scores were repeatedly linked to increased risk of developing major adverse cardiovascular events, worsening kidney function, albuminuria development, and all-cause or cardiovascular mortality. This lends credence to the argument that TyG is not merely a surrogate for insulin resistance but also a valuable predictor of the overall decline in metabolism and vascularity.

A strong aspect of the index is its simplicity. Since it is determined from routine fasting triglyceride and glucose measurements, it can be implemented in routine clinical practice without additional costs or complex tests. This is particularly useful in risk stratification, where more detailed biomarkers might be unavailable. Nevertheless, observational studies continue to dominate the evidence base, limiting their ability to support causal inference and making them prone to residual confounding, population heterogeneity, and variation in outcome definitions.

All in all, TyG can be regarded as a promising adjunct marker to detect high-risk patients with type 2 diabetes and metabolic syndrome. To determine the best cut-off value and demonstrate its influence on long-term clinical decision-making, future large-scale prospective and interventional studies are required.

Acknowledgements

No external funding was used for this review. The paper was prepared for academic purposes, and all interpretations remain the responsibility of the author.

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