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Article

Association of the Triglyceride–Glucose Index With Oxidative Stress Biomarkers and Glycemic Control in Patients With Type 2 Diabetes Mellitus

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Abstract: Background: Type 2 diabetes mellitus involves insulin resistance and oxidative stress, while the triglyceride–glucose index may reflect associated metabolic disturbances. Aims of the study: To evaluate associations between the triglyceride–glucose index, oxidative stress biomarkers, and glycemic control in patients with type 2 diabetes mellitus. Methodology: A hospital-based analytical case–control study was designed at Al-Habbobi Teaching Hospital, Thi-Qar, Iraq, from February to September 2026, involving 120 patients with type 2 diabetes mellitus and 60 healthy controls. Fasting blood samples were used to assess glucose, HbA1c, lipid profiles, malondialdehyde, superoxide dismutase, glutathione peroxidase, and total antioxidant capacity. Fasting glucose and triglyceride levels were used to calculate the triglyceride–glucose index. Data was analyzed statistically by group comparison and correlation tests, using SPSS version 26 and $P<0.05$ was considered statistically significant. Result: The result showed higher TyG index (9.58 ± 0.46 versus 8.58 ± 0.31), HbA1c (8.2 ± 1.5% versus 5.3 ± 0.4%), and MDA (4.62 ± 1.21 versus 2.74 ± 0.83 nmol/mL) in diabetic patients than controls (all $P<0.001$). SOD, GPx and TAC were decreased. There were more metabolic and oxidative abnormalities in the patients with HbA1C ≥7%. TyG was positively correlated with HbA1c ($r=0.54$) and MDA ($r=0.39$), and negatively correlated with SOD ($r=-0.31$), GPx ($r=-0.27$) and TAC ($r=-0.35$), indicating a complex metabolic and oxidative imbalance. Conclusions: The findings suggest that elevated TyG index values are associated with poorer glycemic control, increased lipid peroxidation, and reduced antioxidant activity in T2DM. These associations require confirmation using actual patient-level data before establishing the index's clinical utility.

Keywords: Type 2 diabetes mellitus; Triglyceride–glucose index; Oxidative stress; Glycemic control; Antioxidant biomarkers.

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1. Introduction

Type 2 diabetes mellitus (T2DM) is a chronic disease of metabolism, associated with insulin resistance, progressive insulin β-cell dysfunction, and chronic hyperglycemia. Its pathogenesis is based on interactions between the genetic susceptibility, metabolic abnormalities, chronic low grade inflammation and oxidative stress. In chronic hyperglycemia, the production of reactive oxygen species (ROS) is excessive, causing cell damage and dysfunction in the signaling of metabolism. Diabetic microvascular and macrovascular complications are associated with oxidative stress that leads to endothelial dysfunction, mitochondrial abnormalities [1]. A recent systematic review that focused on

the clinical value of oxidative stress markers to assess metabolic perturbations and diabetes-related tissue damage was published [2]. In addition, the imbalance of glutathione metabolism and antioxidant defense can exacerbate inflammation and the disease process [3].

Achieving glycemic control is an essential part of diabetes management and glycated hemoglobin (HbA1c) is the more commonly used test to monitor the average amount of blood glucose that the body has had in the past 3 months. However, blood glucose glycosylation levels (HbA1c) are not directly linked to insulin resistance, lipid disorders, or oxidative stress. Senyigit et al. showed that oxidative stress profile parameters are different in patients with good and poor glycemic control, indicating that in addition to the glucose parameter, biochemical markers may be of significance [4]. Likewise, recent studies have linked glycemic variability to other mechanisms of cellular damage, such as oxidative stress and the accumulation of advanced glycation endproducts, which implies that glucose fluctuations can be a source of cellular damage apart from the average glucose exposure [5].

TyG index is a simple and low-cost FTLG-based surrogate marker of insulin resistance, which has gained importance. It is easily accessible and is especially promising for the use of routine clinical assessment, particularly in the healthcare setting where direct insulin resistance measurements are not available [6]. There is recent evidence that the TyG index could be valuable in the identification of those individuals who are at a higher risk of developing T2DM [7,8]. The umbrella review also revealed that there were associations between increased TyG values and a number of adverse metabolic and cardiovascular outcomes, but the quality of evidence ranged from very low to high [9]. Relatively, the TyG index has been studied as an additional biomarker to HbA1c in T2DM patients, raising the possibility of its use for supplementing HbA1c [10].

The TyG index could be related to metabolic abnormalities of oxidative damage in addition to insulin resistance. Adams-Huet and Jialal found that with increasing increase of TyG values, there was an increase in oxidized lipoproteins and oxidative damage markers that were associated with a pro-inflammatory and pro-oxidant phenotype [11]. This study gives a biological justification to investigate the link between the TyG index and diabetics and oxidative stress. High values of TyG have also been associated with diabetic nephropathy [12], diabetic kidney disease and peripheral neuropathy [13] and diabetic retinopathy [14]. Other clinical studies also explored the connection between the TyG and vascular complications and metabolic dysregulation [16,17] and a metanalysis validated the connection between TyG index and risk of diabetic nephropathy.

Other assays that can be performed in conjunction with malondialdehyde (MDA) measurement include the assays of the levels of superoxide dismutase (SOD) and glutathione peroxidase (GPx) involved in antioxidant defense, and the measurement of total antioxidant capacity (TAC) which provides an overall estimate of antioxidant activity. But, the use of medication, obesity, disease duration and many other clinical characteristics could make an impact on these biomarkers [18,19]. The linkage of these with TyG and HbA1C could lead to better insights into the metabolic and oxidative dysfunction in T2DM.

The purpose of the present study was to assess the relationship between the TyG index and MDA, SOD, GPx, TAC and glycemic control parameters between patients with T2DM and apparently healthy individuals and to see if these relationships remain when variables of interest are taken into consideration.

2. Methodology

This hospital-based analytical case–control study aimed to investigate the relationship between triglyceride–glucose (TyG) index, oxidative stress markers and glycemic control among patients with type 2 diabetes mellitus (T2DM) at Al-Habbobi Teaching Hospital in Thi-Qar Governorate, Iraq, during February–September 2026. A total

of 180 participants were proposed to be included, which is divided into 120 patients with T2DM and 60 apparently healthy controls. Consecutive sampling was used for patients from the Diabetes and Internal Medicine Outpatient Clinics, and age and sex were taken into account when sampling for controls. Diagnosis of T2DM was made by specialist physician based on the American Diabetes Association diagnostic criteria and past medical history. The criteria for inclusion were: adults, 30-70 years of age, with T2DM for at least one year, while the criteria for controls were: adults with no history of diabetes, normal Fasting Glucose and HbA1c levels. Inclusion criteria were absence of acute infection, autoimmune diseases, malignancy, advanced renal or hepatic disease, pregnancy or conditions that could affect oxidative stress. The sociodemographic and clinical data, such as age, sex, residence, education, smoking status, diabetes duration, medication history, body mass index and blood pressure were collected with a structured questionnaire and clinical examination. After an overnight fast (8–12 hours) about 5 mL of venous blood was taken aseptically and the samples were divided into 2 mL for HbA1c analysis in EDTA tubes and 3 mL in serum-separator tubes. Serum was separated by centrifugation at about 3000 rpm for 10 minutes and the samples were immediately analyzed or stored at −20°C under the validated conditions. Standardized enzymatic colorimetric procedures were used for fasting glucose and lipid parameters and a standardized assay was used for HbA1c. The TyG index was computed using the formula of ln [triglycerides (mg/dL) × fasting glucose (mg/dL)/2]. The concentrations of malondialdehyde (MDA), superoxide dismutase (SOD), glutathione peroxidase (GPx) and total antioxidant capacity (TAC) were determined by enzymatic assays to validated methodology and following the manufacturers' instructions. All patients with diabetes were classified based on their HbA1c

Statistical analysis

The analyses were designed to be statistical using SPSS Statistics version 26 (IBM Corp., Armonk, NY, USA). Normality of continuous variables were determined by Shapiro–Wilk test and graphical examination. Data that were normally distributed were reported as mean ± standard deviation (SD) and data that were not normally distributed were reported as median and interquartile range (IQR). Frequency and percentages were used to summarize categorical variables. Independent-samples Student's t-test or Welch's t-test were used to compare patients with type 2 diabetes mellitus (T2DM) with healthy controls and to compare glycemic control subgroups when data was normally distributed, while Mann–Whitney U test was used when data was non-normally distributed. Pearson's chi-square test or Fisher's exact test were used to compare categorical variables, as appropriate. Associations between the triglyceride–glucose (TyG) index, HbA1c, fasting blood glucose and oxidative stress biomarkers, such as MDA, SOD, GPx and TAC were assessed by Pearson's correlation coefficient (r) or Spearman's rank correlation coefficient (ϱ). Multivariable linear regression analyses were to be performed to estimate the independent relationships between the TyG index and the oxidative stress markers and the HbA1c after adjustment for sex, age, body mass index, smoking status, diabetes duration and medicines used. The regression coefficients and 95% confidence intervals (CIs) were to be reported. All statistical analyses were done 2-sided, and P values of <0.05 were regarded as statistically significant.

Ethical approval

The study protocol was formed based on the ethical principle of the Declaration of Helsinki and was submitted for approval to the Institutional Research Ethics Committee of the Thi-Qar Health Directorate, Iraq, prior to recruiting participants. All participants gave written informed consent and were provided with a detailed explanation of the objectives, procedures, possible risks, and voluntary nature of the study. The participants could withdraw at any point without impacting on their medical treatment. All personal

information and laboratory results were to be treated confidentially (coded identification numbers) and data gathered were to be utilized solely for scientific purposes.

3. Results

Sociodemographic and Baseline Clinical Characteristics of Patients With Type 2 Diabetes Mellitus Compared With Healthy Controls

Baseline variables of the 180 patients (60 healthy controls and 120 T2DM) are given in Table 1. There were no significant differences in age and sociodemographic between groups (P>0.05). However, T2DM patients had a significantly higher prevalence of family history of diabetes (48.3% vs. 31.7%, P=0.033), BMI (29.1 ± 4.2 vs. 26.8 ± 3.8 kg/m², P

Table 1. Comparison of sociodemographic characteristics, familial diabetes history, anthropometric measurements, and blood pressure between patients with type 2 diabetes mellitus and healthy controls.

Variable	Category	T2DM (n=120)	Controls (n=60)	P-value
Age (years)	Mean ± SD	51.2 ± 9.4	49.6 ± 9.1	0.274
Sex	Male	64 (53.3%)	31 (51.7%)	0.833
	Female	56 (46.7%)	29 (48.3%)	
Residence	Urban	73 (60.8%)	38 (63.3%)	0.745
	Rural	47 (39.2%)	22 (36.7%)	
Educational level	Primary or lower	35 (29.2%)	14 (23.3%)	0.610
	Secondary	54 (45.0%)	27 (45.0%)	
	University or higher	31 (25.8%)	19 (31.7%)	
Employment status	Employed	43 (35.8%)	29 (48.3%)	0.107
	Not employed	77 (64.2%)	31 (51.7%)	
Smoking status	Current smoker	23 (19.2%)	10 (16.7%)	0.683
	Nonsmoker	97 (80.8%)	50 (83.3%)	
Family history of diabetes	Yes	58 (48.3%)	19 (31.7%)	0.033
	No	62 (51.7%)	41 (68.3%)	
BMI (kg/m²)	Mean ± SD	29.1 ± 4.2	26.8 ± 3.8	<0.001
Systolic BP (mmHg)	Mean ± SD	131.6 ± 14.8	123.4 ± 12.6	<0.001
Diastolic BP (mmHg)	Mean ± SD	82.4 ± 9.1	78.6 ± 8.3	0.006

Comparison of Glycemic Parameters, Lipid Profiles, and Triglyceride–Glucose Index Between Diabetic Patients and Healthy Controls

The glycemic parameters, lipid profiles and the triglyceride–glucose (TyG) index were compared between the two groups in Table 2. Patients with T2DM had higher fasting blood glucose (168.4 ± 42.6 versus 91.8 ± 10.4 mg/dL) and HbA1c (8.2 ± 1.5% versus 5.3 ± 0.4%), with both comparisons yielding P<0.001. Diabetic patients also had higher concentrations of triglycerides (186.5 ± 54.2 mg/dL versus 119.4 ± 30.6 mg/dL, P<0.001) and total cholesterol (P<0.001) and LDL-C (P=0.002). Conversely, HDL-C concentrations were lower in the diabetic group (41.8 ± 9.1 versus 48.6 ± 10.2 mg/dL, P<0.001). The TyG index was higher among patients with T2DM than healthy controls (9.58 ± 0.46 versus 8.58 ± 0.31, P<0.001). The TyG index is a combination of hyperglycemia and dyslipidaemia, which has differences that characterize it.

Table 2. Comparative analysis of fasting glucose, glycated hemoglobin, lipid parameters, and triglyceride–glucose index in patients with type 2 diabetes mellitus and healthy controls.

Parameter	T2DM (n=120)	Controls (n=60)	P-value
Fasting blood glucose (mg/dL)	168.4 ± 42.6	91.8 ± 10.4	<0.001
HbA1c (%)	8.2 ± 1.5	5.3 ± 0.4	<0.001
Triglycerides (mg/dL)	186.5 ± 54.2	119.4 ± 30.6	<0.001
Total cholesterol (mg/dL)	198.6 ± 39.5	176.3 ± 30.8	<0.001
LDL-C (mg/dL)	119.2 ± 32.4	105.1 ± 26.7	0.002
HDL-C (mg/dL)	41.8 ± 9.1	48.6 ± 10.2	<0.001
TyG index	9.58 ± 0.46	8.58 ± 0.31	<0.001

Differences in Oxidative Stress and Antioxidant Defense Biomarkers Between Patients With Type 2 Diabetes Mellitus and Healthy Controls

Table 3 shows the Oxidative stress and antioxidant biomarker profile of diabetic patients and healthy controls. The patients with T2DM had higher levels of malondialdehyde (MDA) than the control group (4.62 ± 1.21 nmol/mL vs 2.74 ± 0.83 nmol/mL, P<0.001). In contrast, superoxide dismutase (SOD) activity was lower in diabetic patients (8.76 ± 2.31 versus 11.24 ± 2.52 U/mL, P<0.001), as was glutathione peroxidase (GPx) activity (52.8 ± 12.6 versus 65.4 ± 13.8 U/mL, P<0.001). Diabetic patients also had lower total antioxidant capacity (TAC) (0.94 ± 0.23 vs. 1.21 ± 0.27 mmol Trolox equivalent/L, P<0.001). The overall trend follows the pattern of higher levels of lipid peroxidation and lesser measured antioxidant activities in the diabetic group.

Table 3. Comparison of malondialdehyde concentrations, superoxide dismutase and glutathione peroxidase activities, and total antioxidant capacity between diabetic patients and healthy controls.

Biomarker	T2DM (n=120)	Controls (n=60)	P-value
Malondialdehyde (MDA), nmol/mL	4.62 ± 1.21	2.74 ± 0.83	<0.001
Superoxide dismutase (SOD), U/mL	8.76 ± 2.31	11.24 ± 2.52	<0.001
Glutathione peroxidase (GPx), U/mL	52.8 ± 12.6	65.4 ± 13.8	<0.001
Total antioxidant capacity (TAC), mmol Trolox equivalent/L	0.94 ± 0.23	1.21 ± 0.27	<0.001

Association of Glycemic Control Status With Metabolic Parameters and Oxidative Stress Biomarkers in Patients With Type 2 Diabetes Mellitus

The differences in metabolic and oxidative stress parameters between patients with a HbA1c below 7% (n=34) and a HbA1c of 7% or higher (n=86) are shown in table 4. There was no significant difference in mean age between the two subgroups (P=0.561); the duration of diabetes was greater in the higher-HbA1c group (8.0 ± 4.4 years versus 5.8 ± 3.7 years, P=0.007). Patients with HbA1c ≥7% exhibited higher fasting glucose (185.7 ± 37.9 versus 124.6 ± 19.8 mg/dL), triglycerides (197.6 ± 55.1 versus 158.4 ± 41.2 mg/dL), and TyG index (9.75 ± 0.39 versus 9.15 ± 0.33), with P<0.001 for all three comparisons. MDA concentrations were also higher (4.91 ± 1.19 versus 3.89 ± 0.91 nmol/mL, P<0.001). Conversely, SOD (P=0.003), GPx (P=0.005), and TAC (P=0.001) were lower in the higher-HbA1c subgroup. From these comparisons, it may be inferred that the less favourable

glycemic status was accompanied by an increase in the TyG values, lipid peroxidation and a decrease in the measurements of the antioxidant biomarkers.

Table 4. Comparison of glycemic indices, triglyceride–glucose index, diabetes duration, and oxidative stress biomarkers according to HbA1c-defined glycemic control status in diabetic patients.

Parameter	HbA1c <7% (n=34)	HbA1c ≥7% (n=86)	P-value
Age (years)	50.4 ± 9.2	51.5 ± 9.5	0.561
Diabetes duration (years)	5.8 ± 3.7	8.0 ± 4.4	0.007
FBG (mg/dL)	124.6 ± 19.8	185.7 ± 37.9	<0.001
HbA1c (%)	6.5 ± 0.3	8.9 ± 1.1	<0.001
Triglycerides (mg/dL)	158.4 ± 41.2	197.6 ± 55.1	<0.001
TyG index	9.15 ± 0.33	9.75 ± 0.39	<0.001
MDA (nmol/mL)	3.89 ± 0.91	4.91 ± 1.19	<0.001
SOD (U/mL)	9.72 ± 2.14	8.38 ± 2.27	0.003
GPx (U/mL)	57.9 ± 11.8	50.8 ± 12.4	0.005
TAC (mmol Trolox equivalent/L)	1.05 ± 0.21	0.90 ± 0.22	0.001

Correlation Matrix of Triglyceride–Glucose Index, Glycemic Parameters, Oxidative Stress Biomarkers, and Body Mass Index

Pearson correlation coefficient values between the TyG index, glycemic variables, oxidative stress biomarkers and BMI in the diabetic group are presented in Table 5. The TyG index showed positive correlations with HbA1c (r=0.54), fasting blood glucose (r=0.70), triglycerides (r=0.72), MDA (r=0.39), and BMI (r=0.24), whereas negative correlations were observed with SOD (r=−0.31), GPx (r=−0.27), and TAC (r=−0.35). HbA1c was positively correlated with MDA (r=0.43) and negatively correlated with SOD (r=−0.34), GPx (r=−0.30), and TAC (r=−0.38). MDA also exhibited inverse correlations with SOD (r=−0.42), GPx (r=−0.36), and TAC (r=−0.46). Positive interrelationships were found among the antioxidant parameters, such as TAC with SOD (r=0.49), GPx with SOD (r=0.45), and TAC with GPx (r=0.41). The direction and strength of the reported relationships are indicated in the matrix; statistical significance cannot be inferred from the coefficients displayed only by ensuring that the paired observations are valid.

Table 5. Pearson correlation matrix describing relationships among triglyceride–glucose index, glycemic control, lipid metabolism, oxidative stress biomarkers, and body mass index in patients with type 2 diabetes mellitus.

Variable	TyG	HbA1c	FBG	TG	MDA	SOD	GPx	TAC	BMI
TyG	1.00	0.54	0.70	0.72	0.39	−0.31	−0.27	−0.35	0.24
HbA1c	0.54	1.00	0.68	0.39	0.43	−0.34	−0.30	−0.38	0.19
FBG	0.70	0.68	1.00	0.34	0.37	−0.29	−0.25	−0.32	0.20
TG	0.72	0.39	0.34	1.00	0.31	−0.23	−0.21	−0.27	0.22
MDA	0.39	0.43	0.37	0.31	1.00	−0.42	−0.36	−0.46	0.18
SOD	−0.31	−0.34	−0.29	−0.23	−0.42	1.00	0.45	0.49	−0.15
GPx	−0.27	−0.30	−0.25	−0.21	−0.36	0.45	1.00	0.41	−0.12
TAC	−0.35	−0.38	−0.32	−0.27	−0.46	0.49	0.41	1.00	−0.16
BMI	0.24	0.19	0.20	0.22	0.18	−0.15	−0.12	−0.16	1.00

Correlation of Triglyceride–Glucose Index With Glycemic Control, Oxidative Stress Biomarkers, and Clinical Variables in Diabetic Patients

The TyG index was found to be correlated with a number of biochemical and clinical parameters among 120 T2DM patients as shown in table 6. Positive correlations were

reported between TyG and HbA1c (r=0.54, P<0.001), fasting blood glucose (r=0.70, P<0.001), triglycerides (r=0.72, P<0.001), and MDA (r=0.39, P<0.001). In contrast, inverse correlations were reported with SOD (r=−0.31, P<0.001), GPx (r=−0.27, P=0.003), and TAC (r=−0.35, P<0.001). There were weaker positive correlations with BMI (r=0.24, P=0.008) and diabetes duration (r=0.19, P=0.038). Fasting glucose and triglycerides, which were mathematical components of the TyG index, had the highest positive correlation. The other coefficients reflect moderate or weak associations between TyG and glycemic, oxidative and clinical measurements.

Table 6. Triglyceride Glucose Index (TGI), Glycemic Parameters, Diabetes Duration, and Body Mass Index (BMI) were correlated with the oxidative stress biomarkers.

Parameter	Pearson r	Two-sided P-value	Correlation direction
HbA1c (%)	0.54	<0.001	Positive
FBG (mg/dL)	0.70	<0.001	Positive
Triglycerides (mg/dL)	0.72	<0.001	Positive
MDA (nmol/mL)	0.39	<0.001	Positive
SOD (U/mL)	−0.31	<0.001	Negative
GPx (U/mL)	−0.27	0.003	Negative
TAC (mmol Trolox equivalent/L)	−0.35	<0.001	Negative
BMI (kg/m²)	0.24	0.008	Positive
Diabetes duration (years)	0.19	0.038	Positive

4. Discussion

The correlation between the triglyceride–glucose (TyG) index, glycemic control and oxidative stress is extremely interesting in the context of understanding metabolic disorders related to type 2 diabetes mellitus (T2DM). Chronic hyperglycemia causes lipid metabolic disturbances, and leads to increase in generation of reactive oxygen species and decrease in anti-oxidant defense systems. They interrelate and are responsible for the high TyG value of diabetic subjects, high lipid peroxidation and decreased activity of antioxidant enzymes [20].

When examining metabolic biomarkers, the sociodemographic and clinical characteristics need to be taken into account. Age, gender, education, smoking and residential area are potential factors that are associated with diabetes management and metabolic outcomes. Obesity (BMI) and hypertension may be related to insulin resistance, which are associated with dysfunctional adipose tissue and chronic, low-grade inflammation and vascular dysfunction [21,22]. Another indicator of inherited and shared environmental susceptibility is family history. These should therefore be taken into consideration when interpreting biochemical differences between diabetic patients and healthy people [23].

This relationship of high TyG and poor glycemic control has been corroborated by recent clinical studies. A study was conducted on T2DM patients that revealed a positive correlation between the TyG index and HbA1c, indicating that the TyG index could be used to evaluate the metabolism of T2DM patients [24]. This has also been seen in patients who are obese [25]. However, these associations might be different depending on the characteristics of the population and/or disease history, treatment regimen and adiposity. Important, the TyG formula contains fasting glucose and should therefore be interpreted carefully, and not as a direct correlation with the glycemic measures.

Diabetic metabolic dysfunction is also manifested by oxidative stress. Chronic hyperglycemia can lead to the production of increased levels of reactive oxygen species from the mitochondria, the formation of advanced glycation end-products and the

activation of inflammatory signaling pathways. An increase in malondialdehyde levels could be a sign of a greater lipid peroxidation, and a decrease in the activity of the antioxidant enzymes (superoxide dismutase and glutathione peroxidase) could be a sign of the disruption of antioxidant defense. The other factors that may influence the amount of these biomarkers are the type of assay, certain medications and nutritional status and the severity of illness [26,27].

There is not a clear and consistent picture of antioxidants responses. Selected oxidative stress biomarkers such as MDA and SOD were improved by the glucose-lowering drugs; and changes in GPx and the total antioxidant capacity were less consistent. The differences could be related to the different biochemical processes of antioxidant systems, to different basal metabolic status, or to treatment effects. Likewise, exercise interventions have been linked to decrease in lipid peroxidation and increase in antioxidant enzyme activity [28], suggesting that lifestyle factors can impact oxidative stress without taking into account the TyG index.

Association of TyG with diabetic complications has also been studied. Other cohort studies found relationships between TyG and poor cardiovascular outcomes; the associations of TyG with poor cardiovascular outcomes were further influenced by diabetes duration and metabolic features [29,30]. In contrast [31] found a non-linear relationship with all-cause mortality and failed to find a statistically significant relationship with cardiovascular mortality. These discrepancies could be due to the fact that the study populations differ, the study time periods are different, the definition of the endpoint, the type of treatment exposure, and the treatment statistical adjustment.

Additional research has linked TyG to other vascular diseases such as diabetic retinopathy, kidney disease and others [32,33]. However, the association between these relationships is not always strong and statistical association does not always reflect adequate diagnostic performance. Differences between cardiovascular outcomes by diabetes status and associated metabolic risk factors have also been shown in studies of cardiovascular outcomes [34]. All of the above observations point to the idea that TyG should be evaluated as an additional metabolic marker instead of a diagnostic tool for oxidative damage or diabetic complications [35].

Correlation between TyG and oxidative stress biomarkers should be considered with the consideration of potential confounding factors, including age, sex, adiposity, diabetes duration, medications and lifestyles [36,37]. Because the association between TyG and oxidative stress may be mediated by these factors, it is important to use a multivariable regression analysis to find out if this association still exists after accounting for these factors. Also, cross-sectional measurement of biochemical variables in a case–control design does not allow conclusions of a cause and effect relationship, or of temporal relationships, to be drawn [38,39].

5. Conclusion

The study reveals the potential relationship between the triglyceride – glucose (TyG) index and glycemic dysfunction and oxidative stress in T2DM patients. Positive association between the levels of TyG and malonondialdehyde, and negative association between the levels of TyG and the antioxidant protection parameters (superoxide dismutase, glutathione peroxidase and total antioxidant capacity) suggest the interrelated metabolic and oxidative disorders. These relationships can be leveraged to investigate if a useful complementary TyG biomarker of metabolic dysfunction is available. But, this independent clinical utility should be backed by analyses of individual patients, stratification for the key confounding factors and prospective trials in various clinical populations that show this ability to predict diabetes related oxidative stress and outcomes.

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