Section: General Medicine

Original Research Article

ASSOCIATION OF THE TRIGLYCERIDE GLUCOSE(TYG) INDEX WITH DIABETIC KIDNEY DISEASE AMONG PATIENTS WITH TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Diabetic kidney disease (DKD) is a leading cause of morbidity in patients suffering from type 2 diabetes mellitus (T2DM). The triglyceride–glucose (TyG) index is considered as reliable alternate marker of insulin resistance. It has emerged as a potential predictor of diabetic complications. We studied the association of the triglyceride glucose index with DKD and its correlation with conventional renal markers like eGFR and albuminuria.

Materials and Methods: A cross-sectional study was conducted with 135T2DMpatients attending a tertiary care hospital` in eastern India over 7 month period. Various parameters like demographic details, laboratory values [fasting blood sugar (FBS), 2-hour postprandial blood glucose (2hrPPBG), lipid profile, renal markers], and the TyG index were recorded. Patients were stratified based on the presence or absence of DKD, defined by eGFR< 60 mL/min/1.73 m² and/or urine albumin-to-creatinine ratio ≥ 30 mg/g. Both groups compared on various parameters. TyG index was corelated with eGFR and albuminuria was done and its association with DKD found out.

Results: In our study of out of total patients46.67% had DKD. The patients with DKD had significantly higher FBS (188.7 vs. 131.4 mg/dL), 2hrPPBG (276.0 vs. 189.5 mg/dL), and TyG index (5.28 vs. 4.95; all p< 0.0001) than non DKD patients. Renal markers like, including urea, creatinine, and urine albumin, with lower eGFR (all p< 0.0001)significantly deranged in the DKD group. The TyG index correlated positively with urine albumin (r = 0.38, p< 0.0001) and negatively with eGFR (r = –0.17, p = 0.04). ROC analysis demonstrated good predictive performance for both TyG index (AUC 0.773, cutoff 4.9, sensitivity 90.5%, specificity 51.4% and eGFR (AUC 0.754, cut off 61.5, sensitivity 90.3%, specificity 56%).

Conclusion: The triglyceride index is significantly associated with DKD in T2DM patients. It can be a simple and cost-effective yet promising marker for early detection of DKD.

Keywords: Consumer Protection Act, CPA 2019, medical negligence, informed consent, litigation, healthcare law, patient rights, defensive medicine, India.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a leading global health problem, associated with increased rates of morbidity, mortality as well as health system burden. Among microvascular complications of T2DM chronic kidney disease (CKD) affects 25–40% of patients.[1-3] CKD is clinically defined by a reduction in estimated glomerular filtration rate (eGFR<60 mL/min/1.73 m²) or an increase in urinary albumin-to-creatinine ratio (UACR ≥30 mg/g).4 Approximately 30% of patients develop end-stage renal disease (ESRD), which dramatically reduces survival and quality of life.3 T2DM is now the primary cause of ESRD in developed nations and the second most common cause in China.[5,6]

DKD markedly increases risk of cardiovascular disease (CVD), compounding both morbidity and

mortality.[7] Albuminuria is widely recognized as the most sensitive biomarker of early diabetic kidney disease (DKD).[8,9] Even at subclinical levels, increased albumin excretion reflects glomerular barrier dysfunction and is strongly predictive of CVD and non-alcoholic fatty liver disease (NAFLD).[10] Studies suggest that nearly 40% of diabetic patients develop albuminuria, highlighting its utility as a screening and prognostic tool.[11]

Insulin resistance (IR), a key feature of T2DM and an important contributor to DKD. It alters renal hemodynamics, damages podocytes, promotes glomerular hypertrophy, and leads to tubulo-interstitial fibrosis.[12] These processes accelerate albuminuria, hypertension, and CKD progression. Accurate assessment of IR is therefore crucial for predicting renal complications. The hyperinsulinemic–euglycemic clamp test is considered the gold standard for insulin resistance(IR) measurement, but it is complex and impractical in clinical settings.[13]

The triglyceride–glucose (TyG) index, calculated from fasting triglycerides and fasting glucose, has become a reliable and cost-effective surrogate marker of IR.[14] It correlates strongly with the clamp test and HOMA-IR.[14,15] Elevated TyG index values have been related to increased risk of T2DM, CVD, and diabetic complications, including retinopathy, neuropathy, and nephropathy.[16]

Recent evidence has demonstrated significant associations between the TyG index and renal outcomes. Low et al. in their prospective cohort study reported that higher TyG values independently predicted CKD progression in T2DM, mediated partly by pigment epithelium-derived factor.[17] Fritz et al. further showed that the TyG index explained nearly half of the observed association between body mass index (BMI) and ESRD in the general population.[18] Gao et al. found that each one-unit increase in the TyG index conferred a 1.49-times higher risk of ESRD in T2DM patients with CKD.[19] Li et al. demonstrated that higherTyG index values were significantly associated with both albuminuria and CKD in T2DM.[20]Despite growing evidence, few studies have specifically examined the association of TyG index with both eGFR and albuminuria in T2DM patients. Understanding this could help in risk stratification and early treatment, ultimately improving renal and cardiovascular outcomes.

MATERIALS AND METHODS

This hospital-based cross-sectional study was conducted at the Institute of Medical Sciences and SUM Hospital, Bhubaneswar, after approval from the Institutional Ethics Committee. Adult patients with type 2 diabetes mellitus (T2DM), diagnosed per American Diabetes Association (ADA) criteria21, were recruited from outpatient and inpatient departments over seven months. The sample size was calculated as 132 using a 5% margin of error, 95% confidence interval, and 50% response distribution, based on an estimated population of 200 diabetic patients.

Inclusion criteria were Type 2 DM patients with age >18 years and informed consent. Exclusion criteria were end-stage renal disease, eGFR<15 mL/min/1.73 m² (CKD-EPI equation)22, acute infection, pregnancy, malignancy, or lipid-lowering drug use.

Demographic and clinical data (age, sex, BMI, blood pressure, diabetes duration, and comorbidities) were collected. Laboratory tests included fasting blood glucose (FBG), serum triglycerides, HbA1c, serum creatinine, lipid profile, and urinary albumin-to-creatinine ratio (UACR). Renal function was assessed by eGFR (CKD-EPI).22 The triglyceride-glucose (TyG) index was calculated as:TyG=ln((Triglycerides (mg/dL)×Fasting Glucose (mg/dL))/2.23

Statistical Analysis: Statistical analysis was done with IBM SPSS v26. Nominal variables were expressed as counts/percentages, ordinal as medians (IQR), and continuous as means ± SD. For comparisons, Chi-square, Fisher's exact, or McNemar tests were used for nominal data; Mann–Whitney U, Wilcoxon signed-rank, Kruskal–Wallis, or Friedman tests for ordinal data; and independent/paired t-tests or ANOVA for continuous data. Logistic regression assessed associations between TyG and renal parameters, while ROC curve analysis evaluated its diagnostic accuracy. A p-value <0.05 was considered significant.

RESULTS

Age and gender distribution of patients: Among 54 females, 22.22% (n=12) were aged 20–35 years, 40.74% (n=22) were 36–50 years, and 37.04% (n=20) were 51–65 years; no females were older than 65 years. Of 81 males, none were 20–35 years, 28.40% (n=23) were 36–50 years, 46.91% (n=38) were 51–65 years, and 24.69% (n=20) were >65 years. The mean age was 44.38 ± 10.94 years for females and 58.54 ± 8.93 years for males [Table 1 and Supplementary Figure 1].

Laboratory Parameters: The mean fasting blood sugar (FBS) was 158.17 ± 57.01, 2-hour postprandial blood glucose (2hrPPBG) 229.86 ± 101.32, and TyG index 5.1 ± 0.37. Renal markers included urea 40.45 ± 19.61, creatinine 1.24 ± 0.66, eGFR 77.62 ± 30.24, and urine albumin 182.74 ± 168.82. Lipid profile values were triglycerides 199.18 ± 110.63 and LDL 124.68 ± 59.18 [Table 2 and Supplementary Figure 2].

Diabetic Complications: Among the patients, 46.67% (63) had diabetic kidney disease, 5.93% (8) had diabetic retinopathy, and 29.63% (40) had diabetic neuropathy [Table 3 and Supplementary Figure 3].

Table 1: Age and Gender Distribution of Patients

Age Distribution (in years)	Female		Male		Total	
	No. of Patients	Percentage	No. of Patients	Percentage	No of patients	Percentage
20-35	12	22.22	0	0.00	12	8.89
36-50	22	40.74	23	28.40	45	33.33
51-65	20	37.04	38	46.91	58	42.96
>65	0	0.00	20	24.69	20	14.81
Total	54	100.00	81	100.00	135	100.00
Mean±SD	44.38±10.94		58.54±8.93		52.88±11.97	

The data in this table shows the distribution of patients based on gender as well as total number of patients in each age group in number as well as percentage form. The final row, 'Mean ± SD', provides the mean age and standard deviation for each group.

Table 2: Laboratory Parameters of the Study Population

		Mean	SD
Glucose Metabolism & Diabetes Markers	FBS	158.17	57.01
	2hrPPBG	229.86	101.32
	TyG Index	5.1	0.37
Renal Function Markers	Urea	40.45	19.61
	Creatinine	1.24	0.66
	eGFR (Estimated Glomerular Filtration Rate)	77.62	30.24
	Urine Albumin	182.74	168.82
Lipid Profile &Cardiovascular Risk Markers	Triglyceride	199.18	110.63
	LDL (Low-Density Lipoprotein)	124.68	59.18

This table presents descriptive statistics, including the Mean and Standard Deviation (SD), for a range of health markers.

Table 3: Prevalence of Diabetic Complications Among Patients

	No. of Patients	Percentage	No. of Patients	Percentage
Diabetic Kidney Disease	63	46.67	72	53.33
Diabetic Retinopathy	8	5.93	127	94.07
Diabetic Neuropathy	40	29.63	95	70.37

This table presents the prevalence of specific diabetic complications among a cohort of patients.

Glucose Metabolism and Diabetes Markers: Patients with diabetic kidney disease (DKD) had significantly higher fasting blood sugar (FBS: 188.73 ± 63.18 vs. 131.43 ± 33.09; overall 158.17 ± 57.01), 2-hour postprandial glucose (275.96 ± 104.11 vs. 189.52 ± 79.82; overall 229.86 ± 101.32), and TyG index (5.28 ± 0.33 vs. 4.95 ± 0.33; overall 5.1 ± 0.37) compared to the non-DKD group [Table 4 and Figure 4]. All differences were statistically significant ($p < 0.0001$).

Table 4: Comparison of Glucose Metabolism and Diabetes Markers in Patients with and Without Diabetic Kidney Disease

Diabetes Mar	Present (n=63)		Absent (n=72)				Value
	Mean	SD	Mean	SD	Mean	SD	
FBS (mg/dl)	188.73	63.18	131.43	33.09	158.17	57.01	<0.0001
2hrPPBG (mg/dl)	275.96	104.11	189.52	79.82	229.86	101.32	<0.0001
TyG Index	5.28	0.33	4.95	0.33	5.1	0.37	<0.0001

This table compares key glucose metabolism and diabetes markers in two groups of patients

Renal Function Markers: Patients with diabetic kidney disease (DKD) showed significantly higher urea (47.71 ± 23.15 vs. 34.09 ± 13.03; overall 40.45 ± 19.61) and creatinine (1.57 ± 0.77 vs. 0.94 ± 0.34; overall 1.24 ± 0.66), along with markedly elevated urine albumin (347.46 ± 95.68 vs. 38.61 ± 25.56; overall 182.74 ± 168.82) compared to non-DKD patients. Conversely, eGFR was lower in the DKD group (62.17 ± 31.93 vs. 91.13 ± 20.93; overall 77.62 ± 30.24) [Table 1 and Figure 5]. All differences were statistically significant ($p < 0.0001$).

Lipid Profile and Cardiovascular Risk Markers: Patients with diabetic kidney disease (DKD) had significantly lower triglyceride levels (172.33 ± 124.76 vs. 229.87 ± 82.65; overall 199.18 ± 110.63) and LDL cholesterol (98.54 ± 41.56 vs. 154.57 ± 62.38; overall 124.68 ± 59.18) compared to non-DKD patients [Table 5 and Figure 6]. Both differences were statistically significant ($p < 0.0001$).

Table 5: Comparison of Lipid Profile and Cardiovascular Risk Markers in Patients with and Without Diabetic Kidney Disease.

Cardiovascular markers	Present(n=63)		Absent(n=72)				Value
	Mean	SD	Mean	SD	Mean	SD	
Triglyceride	172.33	124.76	229.87	82.65	199.18	110.63	<0.0001
LDL (Low-Density Lipoprotein)	98.54	41.56	154.57	62.38	124.68	59.18	<0.0001

This table compares key cardiovascular risk markers—Triglyceride and LDL levels—between two groups

Correlation Analysis: Pearson's correlation analysis revealed a moderate positive corelation between the TyG index and urine albumin ($r = 0.38$, $p < 0.0001$), indicating that higher TyG index values are significantly linked with elevated urine albumin levels [Table 2 and Figure 1].

Correlation Analysis with eGFR: Pearson's correlation analysis demonstrated a weak negative correlation between the TyG index and eGFR ($r = -0.17$, $p = 0.04$), indicating a slight but statistically significant inverse relationship between insulin resistance and renal function [Table 3 and Figure 1B].

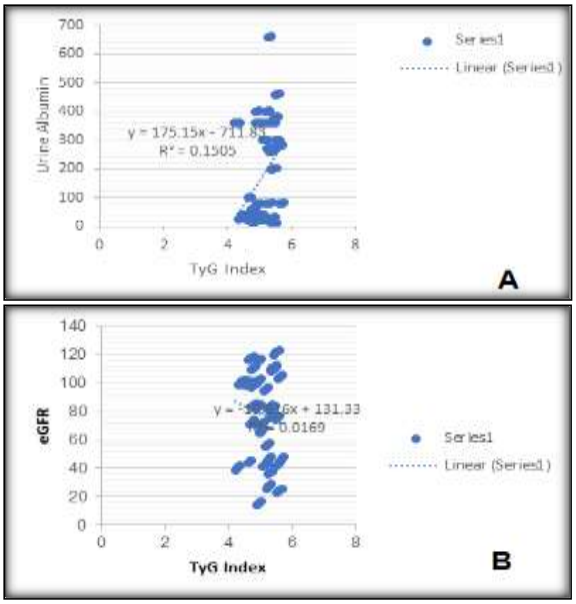

Figure 1: Correlation Analysis and ROC analysis of Dib (a) between TyG Index and Urine Albumin (b) TyG Index and eGFR.

ROC Analysis for eGFR: Receiver operating characteristic (ROC) analysis for eGFR yielded an AUC of 0.754 (95% CI: 0.666–0.841; $p < 0.0001$), indicating good discriminatory ability. The optimal cutoff value of 61.5 demonstrated a sensitivity of 90.3% and specificity of 56% [Table 4 and Figure 2a].

ROC Analysis for TyG Index: The ROC analysis for the TyG index demonstrated an AUC of 0.773 (95% CI: 0.690–0.857; $p < 0.0001$), reflecting robust discriminatory performance. The optimal cutoff value of 4.9 provided a sensitivity of 90.5% and a specificity of 51.4% [Table 5 and Figure 2b].

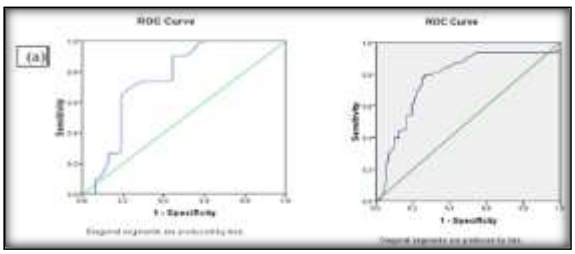

Figure 2: ROC correlation Analysis of Diabetic kidney patients (a) eGFR for Diabetic Kidney Disease Prediction (b) TyG Index for Diabetic Kidney Disease Prediction.

DISCUSSION

The triglyceride-glucose (TyG) index considered as a marker of insulin resistance is gaining importance for its role in predicting renal dysfunction in type 2 diabetes mellitus (T2DM). We got the following observations in our study.

The mean age of participants was 52.88 ± 11.97 years, with most (42.96%) in the 51–65 years group. The mean age was 44.38 ± 10.94 years for females and 58.54 ± 8.93 years for males. Kunutsor et al. reported a similar mean age of 53 years,[24] while Lv L et al.[25] described mean age of 61 years17 and Gao et al. also had older cohorts,[19] reflecting older people as more affected. In our study, 60% were male, consistent with some prior findings, though other studies have shown balanced or female-predominant distributions, [26,27] again underscoring population differences in diabetes and CKD burden.

Nearly half of the patients (46.7%) had diabetic kidney disease, followed by neuropathy (29.6%) and retinopathy (5.9%). Jawa et al. reported higher retinopathy prevalence in nephropathy patients,[27] while Lv et al. showed greater DKD risk in higher TyGtertiles,[25] aligning with our findings that elevated TyG is closely linked to renal complications. Fasting glucose, postprandial glucose, and TyG index were significantly higher in the DKD patients than in non-DKD patients. This is consistent with studies by Low,[17] Gao,[19] Li,[20] and Jiang,[28] all of which confirmed higher TyG index values in patients with CKD or adverse outcomes, supporting its role as a metabolic predictor of renal injury.

Patients with DKD exhibited significantly higher urea, creatinine, and urinary albumin, and lower eGFR. Similar findings were reported by Low et al,[17] and Hussain etal,[29] who observed progressive worsening of renal function with rising albuminuria. Lv et al,[25] also demonstrated that higher TyG index was associated with incident microalbuminuria and eGFR decline. Oh et al,[30] noted only modest reductions across TyG index.

In our study triglycerides and LDL were lower in DKD patients which was not in agreement with prior reports. Low et al,[17] found higher triglycerides but no LDL differences. Gao et al,[19]observed higher LDL-C in adverse outcome groups. These differences may be due to difference in statin use, dietary factors, or disease stage.

We found a moderate positive correlation between TyG index and urinary albumin ($r = 0.38$) and a mild negative correlation with eGFR ($r = -0.17$). Li HF et al,[31] and Li X et al,[32] confirmed significant associations of TyG with albuminuria, creatinine, and reduced eGFR, reinforcing its link with renal dysfunction.

The TyG index showed good predictive value for DKD (AUC 0.773), with high sensitivity (90.5%) but modest specificity (51.4%). Li HF et al. and Tam et al,[33] similarly reported AUC ~0.67, though at a higher cutoff (>9.66). Variations in cutoff values may

be likely due differences in ethnicity, disease stage, and population risk.

There are some limitations in our study which we need to highlight. The cross-sectional design prevents causal inference, and prospective studies are needed to confirm whether elevated TyG predicts CKD progression. Residual confounding from factors such as diet, exercise, and medications are not excluded.[28] The single-center, moderate sample size (n = 132) may limit generalizability in various population groups. TyG was derived from a single fasting measurement, subject to short-term variability.

Our study has important clinical implications. If we detect DKD early timely treatment with RAAS blockade, SGLT2 inhibitors, and GLP-1 receptor agonists can delay progression.[34,35] TyG index may especially be used in resource-limited settings where advanced biomarkers are unavailable.[36] Future research should be done to give validation to these findings in larger, multi-ethnic cohort studies.

CONCLUSION

We found a strong association between the triglyceride–glucose (TyG) index and renal dysfunction in patients with type 2 diabetes mellitus (T2DM) in our study. Raised TyG index values were linked to reduced estimated glomerular filtration rate (eGFR) and increased albuminuria. So, it can be considered as a reliable marker of early diabetic kidney disease (DKD). TyG index combines both glycemic and lipid-related metabolic stress, providing a comprehensive measure of insulin resistance and its impact on renal function. DKD is a leading cause of end-stage renal disease (ESRD) and cardiovascular morbidity. So early identification of high-risk patients is essential. The TyG index, being inexpensive and easily calculated holds significant promise for use in everyday clinical practice. While the cross-sectional design of this study limits causal inference but TyG index can considered in risk stratification for diabetic nephropathy. Future longitudinal and interventional studies are needed to establish its predictive utility in DKD. We also need to explore whether therapeutic modulation of insulin resistance can improve renal outcomes in T2DM.

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