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Original Research Article

TYG INDEX VS HBA1C FOR EARLY DIABETIC NEPHROPATHY

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ABSTRACT

Background: Diabetic nephropathy is a major microvascular complication of type 2 diabetes mellitus (T2DM), contributing to progressive renal dysfunction. Early identification is essential for preventing disease progression. The triglyceride-glucose (TyG) index has emerged as a potential marker of insulin resistance and early renal involvement. The aim is to compare the diagnostic performance of the TyG index and glycated haemoglobin (HbA1c) in identifying early diabetic nephropathy among patients with T2DM.

Materials and Methods: This hospital-based, observational, cross-sectional analytical study included 86 patients with T2DM, categorized into patients without nephropathy (n=45) and those with early nephropathy (n=41) based on urinary albumin-to-creatinine ratio (UACR). Clinical, biochemical, glycaemic, and renal parameters were assessed. Statistical analysis included correlation, receiver operating characteristic (ROC) curve analysis, and multivariable logistic regression using SPSS version 28.0.

Results: Patients with early nephropathy demonstrated significantly higher TyG index (9.67 ± 0.31 vs. 9.27 ± 0.29; p<0.001) and HbA1c (8.53 ± 1.03% vs. 7.66 ± 0.80%; p<0.001). The TyG index showed a stronger positive correlation with UACR (r=0.534) than HbA1c (r=0.386). ROC analysis demonstrated a higher AUC for TyG (0.828) compared with HbA1c (0.739), although the difference was not statistically significant. At optimal cut-offs, TyG demonstrated 78.05% sensitivity and 82.22% specificity, compared with 51.22% and 86.67%, respectively, for HbA1c. Multivariable analysis identified TyG index (aOR=1.95; p<0.001), HbA1c (aOR=5.43; p=0.001), and BMI (aOR=1.64; p=0.003) as significant independent correlates of early nephropathy.

Conclusion: The TyG index demonstrated promising diagnostic performance and may serve as an inexpensive complementary marker alongside HbA1c for identifying early diabetic nephropathy. Larger prospective studies are required for validation.

Keywords: Triglyceride-glucose index, HbA1c, Diabetic nephropathy, Type 2 diabetes mellitus, Albuminuria.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a major public health concern because of its increasing prevalence, prolonged clinical course, and capacity to damage multiple organs. Persistent metabolic disturbances in diabetes contribute to cardiovascular disease, neuropathy, retinopathy, and renal complications, creating a substantial burden on patients and health systems. Diabetic kidney disease is especially important because renal abnormalities may develop silently, even when patients have no obvious symptoms or significant limitations in daily activities. Earlier recognition offers an opportunity to address modifiable risk factors and preserve kidney function.[1] Diabetic nephropathy represents a

complex microvascular complication arising from interactions among chronic hyperglycaemia, insulin resistance, haemodynamic stress, inflammation, and altered lipid metabolism. Sustained glucose exposure promotes oxidative stress, advanced glycation, endothelial dysfunction, and structural changes within glomerular and tubular tissues. Concurrent hypertension and metabolic abnormalities may intensify renal injury through glomerular hyperfiltration, podocyte damage, and progressive fibrosis. This multifactorial pathogenesis highlights the importance of exploring indicators that reflect disturbances beyond conventional glycaemic control, particularly during the early stages when appropriate management may prevent further deterioration.[2] Early diabetic nephropathy is frequently identified through increased urinary albumin excretion before a substantial reduction in glomerular filtration is apparent. Urinary albumin-to-creatinine ratio (UACR) provides a practical assessment of albumin leakage, while estimated glomerular filtration rate (eGFR) reflects overall filtration function. Moderately increased albuminuria, traditionally termed microalbuminuria, may coexist with an eGFR within the preserved range. Nevertheless, urinary albumin excretion can vary with exercise, infection, acute illness, and haemodynamic conditions, making repeat assessment important before persistent albuminuria is established. Screening approaches therefore require careful interpretation of both UACR and eGFR, alongside evaluation of other possible causes of kidney disease.[3] Glycated haemoglobin (HbA1c) is routinely used to monitor long-term glycaemic exposure and guide treatment in everyday clinical practice in T2DM. It reflects glucose binding to haemoglobin over the preceding months and is central to assessing the adequacy of diabetes management. However, HbA1c primarily represents average glycaemia rather than the full spectrum of metabolic dysfunction involved in renal injury. Individual differences in erythrocyte turnover, anaemia, and selected haemoglobin disorders may influence its interpretation, while renal abnormalities can occur despite apparently acceptable glycaemic control. Thus, although HbA1c remains essential for diabetes care, its diagnostic utility as a standalone indicator of early nephropathy warrants comparison with markers incorporating additional metabolic information.[4] The triglyceride-glucose (TyG) index has emerged as an inexpensive surrogate measure of insulin resistance derived from routinely available fasting triglyceride and plasma glucose concentrations. Unlike methods requiring direct insulin measurement or specialised physiological testing, this index can be calculated using investigations commonly performed during routine diabetes follow-up. By combining indicators of dyslipidaemia and hyperglycaemia, the TyG index may capture complementary metabolic disturbances related to insulin resistance. Nevertheless, the index is an indirect measure, and its relationship with insulin resistance may be affected by body composition, medication use, and population characteristics.[5] Interest in the TyG index has expanded from metabolic risk assessment to the evaluation of chronic kidney disease. Insulin resistance may promote renal endothelial dysfunction, oxidative stress, inflammation, and abnormalities of glomerular haemodynamics, providing a biological rationale for examining metabolic indices alongside standard renal measurements. Observational research has explored associations between the TyG index, albuminuria, and reduced renal function, but such relationships cannot establish causation or determine whether a marker is suitable for diagnosis. Diagnostic usefulness also depends on discrimination, appropriate cut-off selection, sensitivity, specificity, and performance in the population being examined. Consequently, a focused assessment in patients with preserved eGFR and early albuminuric changes is clinically relevant.[6]

MATERIALS AND METHODS

The present study was designed as a hospital-based, observational, cross-sectional analytical study to compare the diagnostic performance of the triglyceride-glucose (TyG) index and glycated haemoglobin (HbA1c) in the early detection of diabetic nephropathy among patients with type 2 diabetes mellitus (T2DM). The study aimed to evaluate the association of both metabolic markers with urinary albumin excretion, renal function, and other clinical and biochemical parameters. The study was conducted at a tertiary care hospital in the Departments of General Medicine. Patients attending outpatient departments or admitted for diabetes-related evaluation were screened for eligibility. A total of 86 patients with established type 2 diabetes mellitus were included in the study. Participants were recruited using a consecutive sampling technique according to predefined inclusion and exclusion criteria. Based on urinary albumin-to-creatinine ratio (UACR), patients were categorized into two groups: those without albuminuria (UACR <30 mg/g) and those with early diabetic nephropathy (UACR 30–299 mg/g), with preserved estimated glomerular filtration rate (eGFR ≥60 mL/min/1.73 m²). Both groups were evaluated for TyG index, HbA1c, and renal parameters.

Inclusion Criteria

Adult patients aged 30–75 years with a confirmed diagnosis of type 2 diabetes mellitus, irrespective of gender, were eligible for inclusion. Patients with available fasting blood glucose, serum triglyceride, HbA1c, serum creatinine, and urinary albumin measurements were considered. Participants were required to have preserved renal function with an eGFR ≥60 mL/min/1.73 m² and provide informed consent.

Exclusion Criteria

Patients with type 1 diabetes mellitus, advanced diabetic nephropathy (UACR ≥300 mg/g), eGFR <60

mL/min/1.73 m², acute kidney injury, known non-diabetic renal disease, nephrotic syndrome, or active urinary tract infection were excluded. Patients with severe systemic infections, malignancy, pregnancy, chronic liver disease, recent blood transfusion, haemoglobinopathies, or conditions affecting HbA1c interpretation were also excluded.

Clinical and Anthropometric Assessment

Detailed demographic and clinical information was collected using a structured proforma. Parameters included age, gender, duration of diabetes, family history of diabetes, smoking status, hypertension, dyslipidaemia, cardiovascular disease, and ongoing medications, including antidiabetic, antihypertensive, lipid-lowering, and renoprotective agents. Height, weight, and waist circumference were recorded using standardized techniques. Body mass index (BMI) was calculated as weight in kilograms divided by height in metres squared. Systolic and diastolic blood pressures were measured using a calibrated sphygmomanometer after adequate rest.

Biochemical Investigations

Venous blood samples were collected following an overnight fast of 8–12 hours. Fasting plasma glucose, serum triglycerides, total cholesterol, high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein cholesterol (LDL-C), blood urea, and serum creatinine were measured using standardized laboratory methods. HbA1c was estimated using a standardized high-performance liquid chromatography method and expressed as a percentage. Estimated glomerular filtration rate was calculated using the 2021 CKD-EPI creatinine equation.

Calculation of Triglyceride-Glucose Index

The TyG index was calculated from fasting plasma glucose and serum triglyceride concentrations using the formula:

TyG index = Ln [Fasting triglycerides (mg/dL) × Fasting plasma glucose (mg/dL) / 2].

The calculated TyG index was analysed as a continuous variable. Higher TyG index values were considered indicative of greater insulin resistance. The relationship between TyG index, HbA1c, albuminuria, and renal function was assessed.

Assessment of Early Diabetic Nephropathy

Renal involvement was evaluated using serum creatinine, eGFR, routine urine examination, and urinary albumin-to-creatinine ratio. First-morning urine samples were preferred for UACR estimation. Normoalbuminuria was defined as UACR <30 mg/g, whereas moderately increased albuminuria was defined as UACR 30–299 mg/g. Persistent albuminuria was confirmed using repeat measurements or previous documented results, with at least two of three measurements being abnormal. Patients with moderately increased albuminuria and preserved eGFR were classified as having early albuminuric diabetic nephropathy after exclusion of other possible renal causes.

Outcome Measures

The primary outcome was to compare the diagnostic ability of the TyG index and HbA1c in identifying early diabetic nephropathy. Secondary outcomes included associations of these markers with UACR, eGFR, serum creatinine, diabetes duration, BMI, blood pressure, and lipid profile. The sensitivity, specificity, optimal diagnostic cut-off values, and area under the receiver operating characteristic curve (AUC) were evaluated for both markers.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using IBM SPSS Statistics version 28.0. Continuous variables were expressed as mean ± standard deviation or median with interquartile range, while categorical variables were presented as frequencies and percentages. The Shapiro–Wilk test was applied to assess normality. Independent Student's t-test or Mann–Whitney U test was used for continuous variables, while the Chi-square test or Fisher's exact test was used for categorical variables. Pearson's or Spearman's correlation analysis assessed relationships between metabolic and renal parameters. Receiver operating characteristic (ROC) curve analysis was performed to evaluate diagnostic performance, with optimal cut-off values determined using Youden's index. Multivariable logistic regression was used to assess the independent associations of TyG index and HbA1c with early nephropathy after adjustment for relevant confounding variables. A p-value <0.05 was considered statistically significant.

RESULTS

Table 1: Comparison of Baseline Demographic and Clinical Characteristics Between Patients With and Without Early Diabetic Nephropathy (N=86)

A total of 86 patients with type 2 diabetes mellitus were included, comprising 45 patients without nephropathy and 41 patients with early diabetic nephropathy. The mean age was significantly higher in the early nephropathy group (59.99 ± 8.08 years) compared to the group without nephropathy (55.43 ± 9.16 years; p=0.016). The duration of diabetes was significantly longer among patients with nephropathy (10.37 ± 4.50 vs. 7.95 ± 3.36 years; p=0.006), along with a higher mean BMI (27.55 ± 3.09 vs. 25.07 ± 2.82 kg/m²; p<0.001). Hypertension was more prevalent in patients with nephropathy (68.29%) than in those without nephropathy (42.22%; p=0.018). Both systolic (138.27 ± 13.32 vs. 131.30 ± 11.05 mmHg; p=0.010) and diastolic blood pressures (83.94 ± 8.27 vs. 79.44 ± 8.75 mmHg; p=0.016) were significantly elevated in the nephropathy group. However, gender distribution (p=0.517) and smoking history (p=1.000) showed no statistically significant differences between the groups.

Table 2: Comparison of Biochemical, Glycaemic and Renal Parameters Between Patients With and Without Early Diabetic Nephropathy (N=86)

The biochemical analysis demonstrated significantly higher fasting plasma glucose levels among patients with early diabetic nephropathy compared to those without nephropathy (165.11 ± 28.27 vs. 140.91 ± 23.65 mg/dL; p<0.001). Similarly, serum triglyceride levels were significantly elevated in the nephropathy group (200.86 ± 45.87 vs. 157.97 ± 41.22 mg/dL; p<0.001). The mean TyG index was significantly higher among patients with nephropathy (9.67 ± 0.31) compared to those without nephropathy (9.27 ± 0.29; p<0.001), while HbA1c levels also showed a significant difference (8.53 ± 1.03% vs. 7.66 ± 0.80%; p<0.001). The mean eGFR was significantly lower in the nephropathy group (83.79 ± 12.03 vs. 92.81 ± 11.09 mL/min/1.73 m²; p<0.001), whereas serum creatinine showed a nonsignificant difference (0.91 ± 0.11 vs. 0.86 ± 0.14 mg/dL; p=0.057). Median UACR was substantially higher among patients with early nephropathy [89.28 (63.55–123.63) mg/g] compared to those without nephropathy [10.78 (7.94–13.52) mg/g], consistent with the albuminuria-based classification of both groups.

Table 3: Correlation of TyG Index and HbA1c With Renal and Clinical Parameters Among Patients With Type 2 Diabetes Mellitus (N=86)

Correlation analysis demonstrated a significant positive association between the TyG index and urinary albumin-to-creatinine ratio (r=0.534; p<0.001), indicating that higher TyG index values were associated with increased urinary albumin excretion. HbA1c also showed a significant positive correlation with UACR (r=0.386; p<0.001), although its correlation coefficient was lower than that of the TyG index. HbA1c demonstrated a significant negative correlation with eGFR (r=−0.238; p=0.028) and a significant positive correlation with serum creatinine (r=0.303; p=0.005), whereas the corresponding associations with the TyG index were not statistically significant (p=0.077 and p=0.198, respectively). Both TyG index (r=0.247; p=0.022) and HbA1c (r=0.274; p=0.011) showed significant positive correlations with the duration of diabetes. However, correlations of the TyG index with BMI (r=0.130; p=0.231) and systolic blood pressure (r=0.169; p=0.119) were not statistically significant. Similarly, HbA1c showed no significant correlation with BMI (r=0.002; p=0.987) or systolic blood pressure (r=0.143; p=0.189).

Table 4: Receiver Operating Characteristic (ROC) Curve Analysis Comparing Diagnostic Performance of TyG Index and HbA1c for Early Diabetic Nephropathy (N=86)

Receiver operating characteristic curve analysis demonstrated that the TyG index had an area under the curve (AUC) of 0.828 (95% CI: 0.732–0.911), compared to 0.739 (95% CI: 0.632–0.837) for HbA1c, indicating better apparent diagnostic discrimination by the TyG index. At an optimal cut-off value of ≥9.490, the TyG index demonstrated a sensitivity of 78.05% and specificity of 82.22%, correctly identifying 32 patients with early nephropathy and 37 patients without nephropathy. In comparison, HbA1c at a cut-off value of ≥8.479% demonstrated a sensitivity of 51.22% and specificity of 86.67%, correctly identifying 21 patients with nephropathy and 39 patients without nephropathy. The TyG index produced fewer false-negative results than HbA1c (9 vs. 20), although it showed slightly more false-positive results (8 vs. 6). The Youden's index was higher for the TyG index (0.603) than for HbA1c (0.379), suggesting a better overall balance between sensitivity and specificity. Both markers demonstrated AUC values significantly greater than 0.50 (p<0.001); however, these individual significance tests do not establish a statistically significant difference between their diagnostic performances.

Table 5: Multivariable Logistic Regression Analysis of Factors Associated With Early Diabetic Nephropathy Among Patients With Type 2 Diabetes Mellitus (N=86)

Multivariable logistic regression analysis demonstrated that the TyG index was significantly associated with early diabetic nephropathy, with each 0.1-unit increase corresponding to 1.95 times higher adjusted odds of nephropathy (aOR=1.95; 95% CI: 1.37–2.77; p<0.001). HbA1c was also identified as a significant independent correlate, with each 1% increase associated with 5.43 times higher adjusted odds of early nephropathy (aOR=5.43; 95% CI: 1.99–14.83; p=0.001). BMI demonstrated a significant positive association, with each 1 kg/m² increase corresponding to 1.64 times higher adjusted odds (aOR=1.64; 95% CI: 1.18–2.28; p=0.003). Although increasing age showed a positive association with nephropathy (aOR=1.09; 95% CI: 0.99–1.20), it did not reach statistical significance (p=0.089). Similarly, hypertension demonstrated increased adjusted odds (aOR=3.19; 95% CI: 0.71–14.36), but the association was statistically nonsignificant (p=0.130). Duration of diabetes also showed no significant independent association with early nephropathy after adjustment (aOR=1.00; 95% CI: 0.84–1.19; p=0.983), indicating that TyG index, HbA1c, and BMI remained the principal significant variables in the fitted model.

Table 1: Comparison of Baseline Demographic and Clinical Characteristics Between Patients With and Without Early Diabetic Nephropathy (N=86)

Parameters	Without Nephropathy (n=45)	Early Nephropathy (n=41)	p-value
Age (years), mean ± SD	55.43 ± 9.16	59.99 ± 8.08	0.016
Male	25 (55.56%)	19 (46.34%)	0.517
Female	20 (44.44%)	22 (53.66%)	—

Duration of diabetes (years)	7.95 ± 3.36	10.37 ± 4.50	0.006
BMI (kg/m²)	25.07 ± 2.82	27.55 ± 3.09	<0.001
Hypertension	19 (42.22%)	28 (68.29%)	0.018
Smoking history	8 (17.78%)	7 (17.07%)	1.000
Systolic BP (mmHg)	131.30 ± 11.05	138.27 ± 13.32	0.010
Diastolic BP (mmHg)	79.44 ± 8.75	83.94 ± 8.27	0.016

Table 2: Comparison of Biochemical, Glycaemic and Renal Parameters Between Patients With and Without Early Diabetic Nephropathy (N=86)

Parameters	Without Nephropathy (n=45)	Early Nephropathy (n=41)	p-value
Fasting plasma glucose (mg/dL)	140.91 ± 23.65	165.11 ± 28.27	<0.001
Serum triglycerides (mg/dL)	157.97 ± 41.22	200.86 ± 45.87	<0.001
TyG index	9.27 ± 0.29	9.67 ± 0.31	<0.001
HbA1c (%)	7.66 ± 0.80	8.53 ± 1.03	<0.001
Serum creatinine (mg/dL)	0.86 ± 0.14	0.91 ± 0.11	0.057
eGFR (mL/min/1.73 m²)	92.81 ± 11.09	83.79 ± 12.03	<0.001
UACR (mg/g), median (IQR)	10.78 (7.94–13.52)	89.28 (63.55–123.63)	—

Table 3: Correlation of TyG Index and HbA1c With Renal and Clinical Parameters Among Patients With Type 2 Diabetes Mellitus (N=86)

Parameters	TyG Index (r)	p-value	HbA1c (r)	p-value
UACR (mg/g)	0.534	<0.001	0.386	<0.001
eGFR (mL/min/1.73 m²)	−0.192	0.077	−0.238	0.028
Serum creatinine (mg/dL)	0.140	0.198	0.303	0.005
Duration of diabetes (years)	0.247	0.022	0.274	0.011
BMI (kg/m²)	0.130	0.231	0.002	0.987
Systolic BP (mmHg)	0.169	0.119	0.143	0.189

Table 4: Receiver Operating Characteristic (ROC) Curve Analysis Comparing Diagnostic Performance of TyG Index and HbA1c for Early Diabetic Nephropathy (N=86)

Diagnostic Parameters	TyG Index	HbA1c
Area under ROC curve (AUC)	0.828	0.739
95% CI for AUC	0.732–0.911	0.632–0.837
Optimal cut-off value	≥9.490	≥8.479%
Sensitivity (%)	78.05	51.22
Specificity (%)	82.22	86.67
True positives	32 (78.05%)	21 (51.22%)
False negatives	9 (21.95%)	20 (48.78%)
True negatives	37 (82.22%)	39 (86.67%)
False positives	8 (17.78%)	6 (13.33%)
Youden's index	0.603	0.379
p-value (AUC vs. 0.50)	<0.001	<0.001

Table 5: Multivariable Logistic Regression Analysis of Factors Associated With Early Diabetic Nephropathy Among Patients With Type 2 Diabetes Mellitus (N=86)

Variables	Adjusted Odds Ratio (aOR)	95% Confidence Interval	p-value
TyG index (per 0.1-unit increase)	1.95	1.37–2.77	<0.001
HbA1c (per 1% increase)	5.43	1.99–14.83	0.001
Age (per year)	1.09	0.99–1.20	0.089
Duration of diabetes (per year)	1.00	0.84–1.19	0.983
Hypertension (present vs. absent)	3.19	0.71–14.36	0.130
BMI (per kg/m² increase)	1.64	1.18–2.28	0.003

DISCUSSION

The present study included 86 patients with type 2 diabetes mellitus, comprising 45 patients without nephropathy and 41 patients with early diabetic nephropathy. The mean age was significantly higher among patients with nephropathy (59.99 ± 8.08 years) compared with those without nephropathy (55.43 ± 9.16 years; p=0.016). Similarly, the duration of diabetes was significantly longer in the nephropathy group (10.37 ± 4.50 vs. 7.95 ± 3.36 years; p=0.006). Hypertension was also more frequently observed in patients with nephropathy (68.29% vs. 42.22%; p=0.018), with significantly higher systolic and diastolic blood pressures. Comparable findings were reported by Varghese et al. (2001), who evaluated 1,425 patients with type 2 diabetes in southern India and documented

microalbuminuria in 36.30% of participants (95% CI: 33.80–38.90%). Their multivariate analysis identified age, diastolic blood pressure, duration of diabetes, fasting plasma glucose, and glycated haemoglobin as significant correlates of microalbuminuria. The agreement between these findings supports the importance of increasing age, prolonged diabetes, and elevated blood pressure in the development of diabetic renal involvement.[7] In the present study, patients with early diabetic nephropathy demonstrated a significantly higher mean BMI (27.55 ± 3.09 kg/m²) compared with patients without nephropathy (25.07 ± 2.82 kg/m²; p<0.001). Additionally, HbA1c was significantly elevated in the nephropathy group (8.53 ± 1.03% vs. 7.66 ± 0.80%; p<0.001), indicating an association between poorer glycaemic control and albuminuric renal involvement. Similar observations were reported by Kanakamani et al. (2010), who investigated 670 eligible patients with type 2 diabetes in northern India. Their study population had a mean age of 52.13 ± 9.90 years, mean BMI of 26.19 ± 4.34 kg/m², and median diabetes duration of 5 years. Microalbuminuria was detected in 25.50% of patients (95% CI: 22.40–29.00%), while macroproteinuria was present in 16.20%. Glycated haemoglobin was identified as a significant correlate of albuminuria. These findings agree with the association between elevated HbA1c and nephropathy in the present study, although the differences in albuminuria frequency should be interpreted in relation to the different patient selection methods.[8] In the present study, biochemical analysis demonstrated significantly higher fasting plasma glucose (165.11 ± 28.27 vs. 140.91 ± 23.65 mg/dL; p<0.001), triglyceride levels (200.86 ± 45.87 vs. 157.97 ± 41.22 mg/dL; p<0.001), and TyG index (9.67 ± 0.31 vs. 9.27 ± 0.29; p<0.001) among patients with early nephropathy. Mean eGFR was significantly lower in the nephropathy group (83.79 ± 12.03 vs. 92.81 ± 11.09 mL/min/1.73 m²; p<0.001). Jiang et al. (2024), in a cross-sectional study involving 924 newly diagnosed patients with type 2 diabetes, reported diabetic kidney disease in 199 patients (21.50%). Their findings demonstrated a significantly higher TyG index among patients with diabetic kidney disease (10.01 ± 1.02 vs. 9.77 ± 1.06; p=0.005), accompanied by significantly lower eGFR (108.46 ± 36.90 vs. 123.36 ± 25.56 mL/min/1.73 m²; p<0.001). However, unlike the present results, their HbA1c values did not differ significantly between groups (10.55 ± 2.71% vs. 10.37 ± 2.68%; p=0.397). These findings support the association between elevated TyG index and diabetic renal involvement, while demonstrating that the relationship between glycaemic markers and kidney disease may vary according to population characteristics and diabetes duration.[9] The present study revealed significantly elevated TyG index and HbA1c levels in patients with early diabetic nephropathy, suggesting that metabolic disturbances accompanying hyperglycaemia were associated with renal injury. Furthermore, the TyG index demonstrated a significant positive correlation with UACR (r=0.534; p<0.001), whereas its correlation with eGFR was negative but statistically nonsignificant (r=−0.192; p=0.077). Similar observations were reported by Liu et al. (2021), who evaluated 682 patients with type 2 diabetes and identified diabetic nephropathy in 232 patients (34.00%). Their study demonstrated significantly higher TyG index, HbA1c, triglycerides, and urinary albumin excretion among patients with nephropathy (p<0.05). The TyG index showed a significant positive association with logarithmically transformed 24-hour urinary albumin excretion but not with logarithmically transformed eGFR. Furthermore, logistic regression identified the TyG index as an independent correlate of nephropathy (OR=1.91; 95% CI: 1.29–2.85; p=0.001), while a TyG value above 9.66 was associated with higher odds of nephropathy (OR=2.99; 95% CI: 1.61–5.06). These results are consistent with the positive association between TyG index and albuminuria demonstrated in the present study.[10] Correlation analysis in the present study demonstrated a significant positive relationship between the TyG index and UACR (r=0.534; p<0.001), suggesting that increasing metabolic insulin resistance was associated with greater urinary albumin excretion. HbA1c also demonstrated a positive association with UACR (r=0.386; p<0.001), along with a significant negative correlation with eGFR (r=−0.238; p=0.028) and a positive correlation with serum creatinine (r=0.303; p=0.005). Comparable observations were documented by Liu et al. (2023), who analysed data from the 2015–2018 National Health and Nutrition Examination Survey to evaluate the relationship between the TyG index and chronic kidney disease. They reported that higher TyG values were significantly associated with increased UACR (β=25.10; 95% CI: 6.76–43.44; p=0.0074) and greater odds of chronic kidney disease (OR=1.34; 95% CI: 1.13–1.59; p=0.0006). The association was particularly strong among participants with diabetes (OR=1.94; 95% CI: 1.46–2.56; p<0.001). These findings support the potential value of the TyG index as a marker of renal involvement, although the population and renal disease definitions differed from those of the present study.[11] The diagnostic performance of the TyG index was evaluated using ROC curve analysis, which demonstrated an AUC of 0.828 (95% CI: 0.732–0.911), compared with an AUC of 0.739 (95% CI: 0.632–0.837) for HbA1c. At an optimal cut-off value of ≥9.490, the TyG index demonstrated a sensitivity of 78.05% and specificity of 82.22%, identifying 32 of 41 patients with early nephropathy. Similar findings were reported by Lv et al. (2021), who investigated 1,432 patients with type 2 diabetes in a cross-sectional analysis and reported that higher TyG index values were significantly associated with microalbuminuria (OR=2.342; 95% CI: 1.744–3.144; p<0.001). In their longitudinal analysis, 94 of 424 participants developed diabetic kidney disease, and

individuals in the highest TyG tertile had a significantly higher adjusted risk than those in the lowest tertile (HR=1.727; 95% CI: 1.042–2.863; p=0.034). Their ROC analysis demonstrated an AUC of 0.69 (95% CI: 0.63–0.76), which was lower than that observed in the present study. Differences in patient selection, disease stage, and renal outcome definitions may explain the variation in diagnostic performance.[12] In the present study, the TyG index demonstrated higher sensitivity than HbA1c for identifying early diabetic nephropathy (78.05% vs. 51.22%), although HbA1c showed slightly higher specificity (86.67% vs. 82.22%). The optimal TyG cut-off was ≥9.490, whereas the HbA1c cut-off was ≥8.479%. The corresponding Youden's indices were 0.603 and 0.379, respectively, indicating a better apparent balance between sensitivity and specificity for the TyG index. Tam et al. (2024) conducted a large retrospective study involving 10,714 patients with type 2 diabetes, among whom diabetic nephropathy was detected in 3,763 individuals (35.10%). Their analysis identified a TyG cut-off of 9.58, with sensitivity of 44.01% and specificity of 71.28%. Patients with TyG values ≥9.58 had 1.95 times higher odds of nephropathy compared with those with lower values. Although the reported cut-off was close to that observed in the present study, the sensitivity and specificity were lower. This variation may be attributable to the broader definition of diabetic nephropathy in their study, which included reduced eGFR, whereas the present study focused specifically on moderately increased albuminuria with preserved renal function.[13] Multivariable logistic regression analysis in the present study demonstrated that the TyG index remained significantly associated with early diabetic nephropathy after adjustment for other clinical variables. Each 0.1-unit increase in TyG index was associated with higher adjusted odds of nephropathy (aOR=1.95; 95% CI: 1.37–2.77; p<0.001). HbA1c was also independently associated with nephropathy (aOR=5.43; 95% CI: 1.99–14.83; p=0.001), while BMI demonstrated a significant positive association (aOR=1.64; 95% CI: 1.18–2.28; p=0.003). Similar findings were reported by Tu et al. (2024), who investigated 2,048 hospitalized patients with type 2 diabetes to assess the relationship between TyG index and diabetic microvascular complications. Their univariate logistic regression analysis demonstrated a significant association between elevated TyG and diabetic kidney disease (OR=1.842; 95% CI: 1.317–2.578). After multivariable adjustment, the association remained statistically significant (OR=1.581; 95% CI: 1.031–2.424; p<0.05). These findings support the independent association between TyG index and diabetic renal involvement, although the odds ratios cannot be compared directly because the exposure increments and modelling strategies differed.[14] The present study demonstrated that both the TyG index and HbA1c were associated with early diabetic nephropathy, although the TyG index showed greater apparent discrimination on ROC analysis (AUC=0.828 vs. 0.739). The TyG index demonstrated a stronger correlation with UACR (r=0.534) than HbA1c (r=0.386), while BMI remained an independent correlate of nephropathy in the adjusted regression model (aOR=1.64; p=0.003). These findings can be compared with those of Shang et al. (2019), who conducted a nested case–control study involving 950 patients with newly diagnosed biopsy-proven diabetic nephropathy and 4,750 age- and sex-matched diabetic controls. Their analysis identified a nonlinear relationship between TyG index and nephropathy, with an estimated threshold of 9.07 (95% CI: 9.05–9.09). Above this threshold, each one-unit increase in TyG index was associated with an adjusted incidence rate ratio of 1.56 (95% CI: 1.27–1.91; p<0.0001). The higher diagnostic cut-off observed in the present study may reflect differences in study populations and outcome definitions. Collectively, these findings suggest that the TyG index may provide complementary information to HbA1c in evaluating diabetic renal involvement. Nevertheless, the present cross-sectional design, limited sample size, and lack of external validation restrict causal interpretation and the immediate clinical application of the proposed diagnostic cut-off.[15]

CONCLUSION

The present study demonstrated that the triglyceride-glucose (TyG) index and HbA1c were significantly associated with early diabetic nephropathy among patients with type 2 diabetes mellitus. The TyG index showed higher sensitivity (78.05%) and better apparent diagnostic discrimination (AUC=0.828) than HbA1c (51.22%; AUC=0.739), although the difference in AUC was not statistically significant. Both markers remained independently associated with early nephropathy after adjustment for relevant clinical factors. The TyG index may serve as a simple, inexpensive, and readily available complementary marker for identifying patients requiring further renal evaluation. However, larger prospective studies are needed to validate its diagnostic accuracy and clinical utility.

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