



Association between Triglyceride-to-High-Density Lipoprotein Ratio and Urinary Albumin-to-Creatinine Ratio in Patients with Type 2 Diabetes Mellitus in Indonesia

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Abstract

Background: Dyslipidaemia and albuminuria are recognised indicators of diabetic nephropathy. The triglyceride-to-high-density lipoprotein (TG-HDL) ratio has been proposed as a potential predictor of renal complications in type 2 diabetes mellitus (T2DM).

Objectives: This study aimed to evaluate the association between TG-HDL ratio and urinary albumin-to-creatinine ratio (UACR) in patients with T2DM.

Methods: This retrospective cross-sectional study analysed medical records of hospitalised T2DM patients at Adam Malik Hospital between October 2024 and March 2025. Forty-six patients aged 18 years or older with complete clinical and laboratory data were included. Clinical variables, including TG-HDL ratio, lipid profiles, and UACR, were extracted. Associations were assessed using Fisher's Exact test and odds ratios (OR) with 95% confidence intervals (CI).

Results: The median TG-HDL ratio was 2.0 (IQR 1.0), and the median UACR was 158.5 mg/g. A TG-HDL ratio ≥ 2.76 was observed in 60.9% of patients. Macroalbuminuria (UACR ≥ 300 mg/g) occurred in 28.3% of patients, while 54.3% had moderate albuminuria (A₂) and 65.2% had dyslipidaemia. A significant association was identified between TG-HDL ratio and UACR ($p = 0.049$). Patients with TG-HDL ratio ≥ 2.76 were more likely to develop macroalbuminuria compared with those with lower ratios (OR 5.18; 95% CI 0.99–27.06).

Conclusion: An elevated TG-HDL ratio was significantly associated with increased UACR in patients with type 2 diabetes mellitus (T2DM). Individuals with a TG-HDL ratio ≥ 2.76 had a 5.18-fold higher risk of developing macroalbuminuria, underscoring its potential as a simple predictive marker for the early detection of diabetic nephropathy.

Keywords: albuminuria; diabetic nephropathy; type 2 diabetes mellitus; TG-HDL ratio; urinary albumin-to-creatinine ratio.

Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterised by hyperglycaemia resulting from insulin resistance and impaired insulin secretion, and it accounts for approximately 90% of diabetes cases worldwide (American Diabetes Association Professional Practice Committee et al., 2025; Perkumpulan Endokrinologi Indonesia, 2024). Complications of T2DM include both neurological

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disorders and vascular complications, the latter classified into macrovascular (affecting the heart, brain, and large vessels) and microvascular (involving the retina and renal microcirculation) types. Among these, diabetic kidney disease (DKD) is the leading cause of chronic kidney disease (CKD) in patients receiving renal replacement therapy, affecting about 40% of individuals with T2DM and often present at diagnosis (Perkumpulan Endokrinologi Indonesia, 2024; Suastika & Dwipayana, 2024). Experimental and clinical studies have shown that abnormal lipoprotein metabolism is closely linked to the onset and progression of CKD (Amelia et al., 2021; Xue et al., 2021). In diabetic patients with CKD, dyslipidaemia is commonly triggered by hyperglycaemia and insulin resistance (Chen & Tseng, 2013). This dyslipidaemia typically presents as low high-density lipoprotein (HDL) and high triglyceride (TG) levels, while total cholesterol and low-density lipoprotein (LDL) levels are usually within normal limits (Chen & Tseng, 2013; Suh & Kim, 2023). Hypertriglyceridaemia is considered an early finding of CKD and is the most frequent lipid abnormality in this population (Suh & Kim, 2023).

The American Diabetes Association (ADA) recommends annual screening for urinary albumin excretion, such as urinary albumin-to-creatinine ratio (UACR) and estimated glomerular filtration rate (eGFR), in all patients with T2DM (Baltaro, 2012). UACR provides an estimate of 24-hour urinary albumin excretion by comparing albumin concentration to creatinine levels in a spot urine sample (Sun et al., 2015). Albuminuria, particularly microalbuminuria, is a recognised marker of glomerular injury and is currently the gold standard for early detection of DKD (Amelia et al., 2021). However, access to UACR testing remains limited in many healthcare centres across Indonesia. Epidemiological studies have demonstrated that TG is a better indicator of microalbuminuria than other lipid parameters, showing significant associations with both micro- and macroalbuminuria in diabetic populations (Wen et al., 2017).

Wen et al. reported that non-traditional lipid indices, particularly the TG–HDL ratio, are superior predictors of eGFR decline compared with conventional lipid profiles (Wen et al., 2017). Similarly, a Japanese cohort study found an inverse correlation between the TG–HDL ratio and CKD (Tsuruya et al., 2015). This finding was reinforced by Sun et al. in China, who concluded that the TG–HDL ratio is a stronger predictor of elevated UACR than traditional lipid parameters (Sun et al., 2015).

To date, no study has specifically examined the association between the TG–HDL ratio and UACR in patients with T2DM in Indonesia. This gap in the literature forms the basis for the present research.

Materials and methods

This was an analytical study with a cross-sectional design, conducted using retrospective data from the electronic medical record system of Adam Malik Hospital, Medan, between October 2024 and March 2025. The study aimed to evaluate the association between the TG–HDL ratio and UACR in patients with type 2 diabetes mellitus (T2DM).

A total of 46 hospitalised patients aged 18 years or older with confirmed T2DM and complete clinical and laboratory data were included in the study. As this was a retrospective cross-sectional study, the sample size was determined by data availability during the study period rather than an a priori power calculation. Patients were excluded if they were undergoing regular haemodialysis, had CKD of non-diabetic origin, liver cirrhosis, urinary tract infections, congestive heart failure, or pregnancy.

Clinical and laboratory data collected comprised lipid profiles (TG, HDL, LDL, and total cholesterol), glycaemic parameters including fasting plasma glucose (FPG), random plasma glucose (RPG), and 2-hour post-prandial plasma glucose, glycated haemoglobin (HbA1c), eGFR, and UACR. The TG–HDL ratio was calculated by dividing TG by HDL concentration. The TG–HDL ratio was categorised using a cut-off value of 2.76, as previously reported by Ho et al (Ho et al., 2015).

Descriptive statistics were used to summarise baseline characteristics. Categorical variables were presented as frequencies and percentages, while continuous variables were expressed as mean $\pm$ standard deviation (SD) for normally distributed data or median with interquartile range (IQR) for non-normally distributed data. The Shapiro–Wilk or Kolmogorov–Smirnov test was applied to assess normality. Associations between the TG–HDL ratio and UACR were analysed using Fisher’s exact test. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated from 2×2 contingency tables. Logistic regression analysis was not performed due to the limited sample size. A p-value < 0.05 was considered statistically significant. Data analysis was performed using SPSS version 27.0 (IBM Corp., Armonk, NY, USA). This study received ethical approval from the Health Research Ethics Commission, Faculty of Medicine, Universitas Sumatera Utara/Adam Malik Hospital (Approval No: 223/KEPK/USU/2025).

Results

A total of 46 patients with T2DM were included in this study. The mean age was 57.6 ± 10.5 years, with slightly more males (56.5%) than females (43.5%). The median duration of diabetes was 60 months (IQR 96). Most patients (82.6%) were receiving insulin therapy, while smaller proportions were managed with oral agents (8.7%) or no treatment (8.7%). The most frequent comorbidities were hypertension (37.0%) and coronary artery disease (34.8%), followed by stroke (21.7%) and pulmonary tuberculosis (2.2%). Regarding nutritional status, 41.3% of patients had normal BMI, while obesity grade 1 (26.1%) and grade 2 obesity (15.2%) were also common. Dyslipidaemia was observed in 65.2% of participants.

In laboratory parameters, the mean eGFR was 55.05 ± 30.93 mL/min/1.73 m², and the median UACR was 158.5 mg/g (IQR 282.7). The median HbA1c was 7.9% (IQR 5.3). The mean FPG was 192.6 ± 97.6 mg/dL, and the mean post-prandial glucose was 223.8 ± 94.2 mg/dL. The median RPG was 224.5 mg/dL (IQR 182). For lipid profiles, the mean total cholesterol was 165.7 ± 49.7 mg/dL, the mean LDL was 95.8 ± 46.8 mg/dL, the mean HDL was 39.3 ± 14.7 mg/dL, and the median triglycerides were 138.5 mg/dL (IQR 120) (Table 1).

Albuminuria was common in this cohort, with 54.3% of patients presenting moderate albuminuria (A2), 28.3% with macroalbuminuria (A3), and 17.4% with normoalbuminuria (A1) (Table 2). The median TG–HDL ratio was 2.0 (IQR 1.0). Based on the cut-off of 2.76, 60.9% of patients had an elevated TG–HDL ratio, while 39.1% were below this threshold (Table 3).

A significant association was found between TG–HDL ratio and UACR ($p = 0.049$). Patients with TG–HDL ratio ≥ 2.76 were more likely to develop macroalbuminuria compared with those with lower ratios, with an odds ratio of 5.18 (95% CI 0.99–27.06) (Table 4).

Table 1. Baseline characteristics of study participants

Characteristics	Results
Age (years), mean $\pm$ SD	57.59 $\pm$ 10.5
Gender, n (%)	
- Male	26 (56.5%)
- Female	20 (43.5%)
Duration of diabetes (months), median (IQR)	60 (96)
Antihyperglycemic drugs, n (%)	
- No	4 (8.7%)
- Oral	4 (8.7%)
- Injection	38 (82.6%)
Comorbidity	
- Hypertension	17 (37%)

-	CAD	16 (34.8%)
-	Stroke	10 (21.7%)
-	Pulmonary TB	1 (2.2%)
BMI (kg/m ²), n (%)		
-	Underweight	1 (2.2%)
-	Normoweight	19 (41.3%)
-	Overweight	7 (15.2%)
-	Obesity grade 1	12 (26.1%)
-	Grade 2 obesity	7 (15.2%)
Dyslipidaemia		
-	Yes	30 (65.2%)
-	No	16 (34.8%)

Laboratory Parameters

eGFR (mL/min/1.73 m ²), mean ± SD	55.05 ± 30.93
UACR (mg/g), median (IQR)	158.5 (282.7)
HbA1c (%), median (IQR)	7.9 (5.3)
FPG (mg/dL), mean ± SD	192.6 ± 97.6
Post-prandial plasma glucose (mg/dL), mean ± SD	223.8 ± 94.2
RPG (mg/dL), median (IQR)	224.5 (182)
Total Cholesterol (mg/dL), mean ± SD	165.7 ± 49.7
LDL (mg/dL), mean ± SD	95.8 ± 46.8
HDL (mg/dL), mean ± SD	39.3 ± 14.7
Triglycerides (mg/dL), median (IQR)	138.5 (120)

BMI: body mass index; CAD: coronary artery disease; DM: diabetes mellitus; eGFR: estimated glomerular filtration rate; FPG: fasting plasma glucose; HbA1c: Glycated haemoglobin; HDL: high-density lipoprotein; LDL: low-density lipoprotein; SD: standard deviation.

SI conversion factors: to convert glucose from mg/dL to mmol/L, multiply by 0.0555; cholesterol from mg/dL to mmol/L, multiply by 0.0259; triglycerides from mg/dL to mmol/L, multiply by 0.0113.

Table 2. Distribution of albuminuria categories among T2DM patients

Variables	Results
Degrees of albuminuria, n (%)	
- A1	8 (17.4 %)
- A2	25 (54.3%)
- A3	13 (28.3%)

UACR: urinary albumin- to - creatinine ratio.

Table 3. TG-HDL ratio in T2DM patients

Variables	Results
TG-HDL ratio, median (IQR)	2.0 (1.0)
TG-HDL ratio, n (%)	
- <2.76	18 (39.1%)
- ≥2.76	28 (60.9%)

HDL: high-density lipoprotein; TG: triglyceride.

Table 4. Association between TG-HDL Ratio and UACR in T2DM Patients

Variables	UACR		P	OR (IK 95%)
	< 300 mg/g, n (%)	≥ 300 mg/g, n (%)		

TG-HDL ratio					
-	<2.76	16 (88.9%)	2 (11.1%)	0.049	5.18 (0.99–27.06)
-	≥ 2.76	17 (60.7%)	11 (39.3%)		

UACR: urinary albumin- to - creatinine ratio; OR: odds ratio; CI: confidence intervals.

Discussion

This study investigated the association between the TG-HDL ratio and UACR in hospitalised patients with T2DM in Indonesia. The majority of the subjects were male, with a mean age of 57.22 years, consistent with previous reports indicating a higher risk of renal complications among male diabetics (Kramer, 2003). In contrast, the national health data survey from 2023 suggested a higher prevalence among older females, indicating potential regional or hospital-based differences (Badan Kebijakan Pembangunan Kesehatan, 2023). Sex-related differences in diabetic complications may be influenced by hormonal factors, including the effects of oestrogen and testosterone on vascular tone, oxidative stress, and inflammatory responses (Kautzky-Willer et al., 2023; Tang et al., 2022).

Hypertension and CAD were the most prevalent comorbidities in this study, reflecting the close relationship between T2DM, cardiovascular disease, and diabetic kidney disease. Hypertension accelerates renal damage through increased intraglomerular pressure, endothelial dysfunction, and activation of the renin–angiotensin–aldosterone system (RAAS), while chronic hyperglycaemia promotes atherosclerosis and coronary artery stenosis, thereby increasing the risk of CAD (Naseri et al., 2022; Petrie et al., 2018). These findings are consistent with previous studies reporting a high burden of cardiovascular comorbidities among patients with T2DM (Petrie et al., 2018).

The high prevalence of obesity and dyslipidaemia observed in this cohort further supports the role of metabolic dysfunction in the progression of diabetic nephropathy. Excess adiposity exacerbates insulin resistance and chronic low-grade inflammation, leading to atherogenic lipid profiles characterised by elevated triglycerides and reduced HDL levels (Chen et al., 2013). In this study, dyslipidaemia was observed in more than half of the participants, a prevalence comparable to that of previous population-based studies. However, variability across regions may reflect differences in lifestyle, dietary patterns, and physical activity. Insulin resistance promotes increased free fatty acid release, hepatic triglyceride synthesis, and very-low-density lipoprotein production, while reducing lipoprotein lipase (LPL) activity, thereby aggravating dyslipidaemia (Taskinen & Borén, 2015).

Most participants had a relatively long duration of diabetes and suboptimal glycaemic control, as reflected by elevated HbA1c levels. Prolonged exposure to hyperglycaemia contributes to microvascular complications through the formation of advanced glycation end-products, oxidative stress, and endothelial injury, all of which are closely linked to increased albuminuria and declining renal function (Hawale et al., 2021; Xu et al., 2024). Consistent with these mechanisms, reduced eGFR and elevated UACR were common findings in this study, highlighting the substantial burden of diabetic kidney disease among hospitalised patients with T2DM (Fenta et al., 2023; Sugahara et al., 2021).

The median UACR in this study was 158.5 mg/g, with more than half of the patients classified in category A2 based on KDIGO 2024 (UACR 30–300 mg/24 h) (Stevens et al., 2024). This finding indicates that most patients exhibited elevated urinary albumin excretion, reflecting underlying endothelial and glomerular dysfunction. Increased UACR in patients with T2DM is primarily driven by chronic hyperglycaemia, which induces glomerular endothelial injury, oxidative stress, activation of the RAAS, and inflammatory pathways, ultimately leading to increased glomerular permeability to albumin. Insulin resistance and dyslipidaemia, which are commonly observed in T2DM, further exacerbate vascular injury and accelerate the progression of diabetic nephropathy towards end-stage renal disease (Jha et al., 2024).

Importantly, 60.9% of patients had a TG–HDL ratio ≥ 2.76 , and the cohort median was 2.0 (IQR 1.0). This accords with Sultana et al., who reported that most patients with T2DM had a TG–HDL ratio > 2 , with a mean of 4.19 ± 1.11 (Sultana et al., 2024). A raised TG–HDL ratio reflects atherogenic dyslipidaemia, which is commonly observed in T2DM and is widely recognised as a marker of insulin resistance and endothelial dysfunction. Mechanistically, insulin resistance promotes adipose tissue lipolysis with increased free fatty acid release, stimulates hepatic triglyceride and very-low-density lipoprotein (VLDL) synthesis, suppresses LPL activity, and impairs HDL biogenesis, ultimately resulting in hypertriglyceridaemia accompanied by low HDL levels (Kalra & Raizada, 2024). Evidence from a Taiwanese study by Ho et al. demonstrated that a TG–HDL ratio > 2.76 was associated with an approximately 1.4-fold increased risk of chronic kidney disease (CKD) in men and a 1.74-fold increased risk in women, as well as reduced kidney function and higher albuminuria levels (Ho et al., 2015). Consistently, this study demonstrated a significant association between TG–HDL ratio and UACR ($p = 0.049$), with most patients who had a high TG–HDL ratio also exhibiting UACR ≥ 300 mg/g. Supporting these findings, Zhang et al. reported that the TG–HDL ratio can serve as a risk indicator for microalbuminuria and diabetic nephropathy, while Shin et al. identified it as a potential early predictor of albuminuria in T2DM (Shin et al., 2016; Zhang et al., 2024). Furthermore, a Japanese cohort study demonstrated an inverse correlation between the TG–HDL ratio and CKD, and Sun et al. concluded that the TG–HDL ratio is a more informative index for assessing the risk of elevated UACR than conventional lipid parameters (Sun et al., 2015).

From a pathophysiological perspective, insulin resistance increases lipolysis in adipose tissue, leading to elevated free fatty acid flux to the liver, enhanced triglyceride synthesis, and impaired HDL metabolism. Triglyceride-rich lipoproteins can induce oxidative stress, activate transforming growth factor- β (TGF- β) signalling pathways, and promote extracellular matrix deposition in the glomeruli and tubulointerstitium, thereby increasing glomerular permeability to albumin (Chen & Tseng, 2013; Wen et al., 2017). The kidney damage associated with these metabolic disturbances is progressive and irreversible, accelerating nephron loss and increasing the risk of chronic kidney disease (Magliano & Boyko, 2021). These mechanisms provide biological plausibility for the observed association between elevated TG–HDL ratio and increased UACR.

study is among the first to investigate the association between the TG-HDL ratio and UACR in patients with T2DM in Indonesia, utilising real-world hospital data. Both TG–HDL ratio and UACR are simple and widely available laboratory parameters, making them practical tools for early risk stratification in resource-limited settings. Nevertheless, this study has several limitations. The cross-sectional design precludes causal inference, and the relatively small sample size may limit generalisability. In addition, the absence of a formal power calculation and potential confounders, such as obesity-related glomerulopathy, hypertensive nephropathy, smoking, and the use of renin–angiotensin system blockers, should be considered when interpreting the findings. Future longitudinal studies with larger cohorts are warranted to confirm these results.

Conclusion

An elevated TG–HDL ratio was significantly associated with UACR in patients with T2DM. As a simple and widely available lipid-based index, the TG–HDL ratio may be useful for identifying patients at higher risk of renal involvement and supporting early risk stratification in clinical practice. Future prospective and longitudinal studies with larger sample sizes are warranted to confirm these findings and to elucidate further the causal relationship between dyslipidaemia and diabetic kidney disease progression.



Conflict of interest

None declared.

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