



Association of Uric Acid-to-High-Density Lipoprotein Cholesterol Ratio with Microvascular Complications in Patients with Type 2 Diabetes Mellitus: A Cross-Sectional Study

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ABSTRACT:

Background: Microvascular complications—chiefly diabetic nephropathy and diabetic retinopathy—accompany type 2 diabetes mellitus (T2DM). Integrating oxidative stress with vascular protection, the uric acid-to-high-density lipoprotein cholesterol ratio (UHR) has surfaced as a composite metabolic marker. How UHR relates to microvascular complications in T2DM patients was the focus of this study. **Methods:** One hundred T2DM patients at a tertiary care centre were enrolled in this hospital-based cross-sectional study. Measurements covered HbA1c, fasting and post-prandial blood sugar, serum uric acid, lipid profile, and urine albumin-creatinine ratio. Fundus examination served to assess retinopathy, while albuminuria status determined nephropathy classification. UHR values fell into low (≤ 0.10) or high (> 0.10) categories. Statistical work drew on the Chi-square test and Pearson's correlation. **Results:** UHR elevation appeared in 71% of participants. The high-UHR group carried a markedly greater retinopathy prevalence than the low-UHR group (49.3% vs 24.1%; $p=0.004$). Nephropathy followed a graded pattern with rising UHR, the high-UHR group showing larger proportions of both microalbuminuria and macroalbuminuria ($p<0.001$). Positive correlations connected UHR with HbA1c ($r=0.41$; $p<0.001$), post-prandial blood sugar ($r=0.32$), and fasting blood sugar ($r=0.29$). **Conclusion:** Diabetic retinopathy, nephropathy, and suboptimal glycaemic control in T2DM each show meaningful links to elevated UHR. As an adjunctive marker for microvascular risk assessment in routine clinical practice, UHR offers a straightforward and cost-effective option.

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MAIN POINTS

- In type 2 diabetes mellitus patients, an elevated uric acid-to-HDL cholesterol ratio (UHR) was present in 71% of cases and showed meaningful links with both diabetic nephropathy and diabetic retinopathy.
- Diabetic retinopathy prevalence ran markedly higher among those with UHR above 0.10 than among the low-UHR group (49.3% vs 24.1%; $p=0.004$).
- Diabetic nephropathy severity tracked elevated UHR in a clear graded fashion, with the high-UHR group displaying greater proportions of macroalbuminuria and microalbuminuria ($p<0.001$).
- Glycaemic parameters correlated positively with UHR—HbA1c most notably ($r=0.41$, $p<0.001$)—pointing to UHR as a reflection of chronic glycaemic burden.
- Derived from routine laboratory investigations and inexpensive to obtain, UHR offers a practical adjunctive tool for stratifying microvascular risk in everyday clinical work.

PLAIN LANGUAGE SUMMARY:

Patients with type 2 diabetes can develop complications that affect the small blood vessels in the eyes (retinopathy) and kidneys (nephropathy). This study tested whether a simple ratio—uric acid divided by HDL ("good") cholesterol, both measured by routine blood tests—can help identify patients at higher risk. In 100 adults with type 2 diabetes, those with a higher ratio were significantly more likely to have eye and kidney complications and to have poorer long-term blood sugar control. Because the ratio uses tests already done in everyday practice, it could be a low-cost way to flag patients who need closer monitoring.

INTRODUCTION:

Type 2 diabetes mellitus (T2DM) ranks among the foremost global health challenges, with prevalence climbing rapidly and a heavy load of chronic complications in its wake. Current projections suggest the number of adults living with diabetes will climb substantially in the decades ahead, with low- and middle-income countries like India bearing a disproportionate share, where suboptimal glycaemic control and early disease onset are routinely encountered.¹² Long-term morbidity in T2DM stems largely from microvascular complications—diabetic retinopathy and diabetic nephropathy in particular—which together drive chronic kidney disease, visual impairment, diminished quality of life, and escalating healthcare expenditure.^{3–5}

Among working-age adults worldwide, diabetic retinopathy continues to rank as a leading cause of preventable blindness.⁶ The pathogenesis traces back to chronic hyperglycaemia, with inflammation, oxidative stress, and endothelial dysfunction together producing progressive retinal microvascular damage.⁷ Diabetic nephropathy similarly stands out as a major contributor to chronic kidney disease and end-stage renal failure, marked by declining glomerular filtration rate, albuminuria, and elevated cardiovascular risk.⁸ Identifying individuals at heightened risk for these microvascular complications early therefore forms a cornerstone of comprehensive diabetes management.

Although sustained hyperglycaemia drives microvascular injury principally, mounting evidence implicates additional metabolic factors in the process. Serum uric acid has come into focus as a possible mediator of vascular damage in diabetes. Experimental and clinical work indicates that raised uric acid levels track with endothelial dysfunction, reduced nitric oxide bioavailability, activation of inflammatory cascades, and oxidative stress.^{9–10} Hyperuricaemia is a common finding in T2DM, arising from insulin resistance-driven impairment of renal urate clearance together with heightened uric acid production.¹¹ Reports linking elevated serum uric acid to diabetic microvascular complications—nephropathy and retinopathy especially—have accumulated, though the findings have not been wholly uniform.^{12–14}

High-density lipoprotein cholesterol (HDL-C) has long been viewed as a protective lipid fraction, carrying endothelial-protective, antioxidative, and anti-inflammatory properties.¹⁵ HDL-C levels frequently fall in T2DM, and the particles themselves may become functionally compromised through glycation and oxidative modification, eroding their vasculoprotective capacity.¹⁶ Reduced HDL-C carries a recognised link with greater risk of both microvascular and macrovascular complications in diabetic patients.¹⁷

Attention has more recently turned toward composite metabolic indices capable of integrating multiple pathophysiological pathways at once. The uric acid-to-high-density lipoprotein cholesterol ratio (UHR) represents one such marker, capturing the equilibrium between a pro-inflammatory, pro-oxidant factor (uric acid) and a protective lipid component (HDL-C). Associations have been documented between UHR and metabolic

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syndrome, insulin resistance, and adverse cardiovascular outcomes.^{18–20} Drawing on two biochemical parameters that are already part of routine workup, UHR potentially offers a more rounded read on metabolic and vascular risk than either parameter taken alone.

For all the interest UHR now attracts, evidence regarding its link to diabetic microvascular complications stays sparse, especially within the Indian population. Macrovascular outcomes and general metabolic risk have dominated the published work to date, with comparatively little research dedicated to UHR and specific microvascular endpoints such as diabetic retinopathy and nephropathy. The heavy burden of T2DM and its complications across India, paired with a pressing need for simple, low-cost risk stratification tools, makes further evaluation of UHR in this setting both warranted and timely.

This study was therefore designed to examine how UHR relates to microvascular complications in T2DM patients attending a tertiary care hospital. The work set out to look at UHR alongside diabetic retinopathy and diabetic nephropathy, and to test its correlation with glycaemic parameters. Clearer understanding of these associations should help define what role UHR might play as an adjunctive marker for microvascular risk assessment in everyday diabetes care.

MATERIAL AND METHODS

Study Design and Setting: This was a hospital-based cross-sectional observational study conducted in the Department of General Medicine at a tertiary care teaching hospital (xxx) in India. The study was carried out from 1 September 2024 to 1 March 2025 after obtaining approval from the Institutional Ethics Committee.

Ethics Approval and Informed Consent: Ethical committee approval was received from the Ethics Committee of AVMCH (Approval no: AV/IHEC/01/2024/042, Date: 7 June 2024). Written informed consent was obtained from all patients who agreed to take part in the study. The study was conducted in accordance with the principles of the Declaration of Helsinki.

Use of Generative AI / LLM Tools: This study did not use AI and/or LLM tools during the preparation of this manuscript.

Study Population: The study included 100 adult patients diagnosed with T2DM, attending the outpatient and inpatient services during the study period. Diagnosis of T2DM was based on standard diagnostic criteria as per the American Diabetes Association guidelines.

Inclusion Criteria: Patients aged ≥ 18 years; diagnosed cases of type 2 diabetes mellitus; willingness to participate and provide informed consent.

Exclusion Criteria: Patients with type 1 diabetes mellitus; pregnant women; patients with known chronic liver disease, gout, malignancy, or acute infections; patients on uric acid-lowering therapy; patients with non-diabetic causes of chronic kidney disease.

Clinical and Laboratory Assessment: Detailed clinical evaluation was performed for all participants, including demographic details and relevant medical history. Venous blood samples were collected after an overnight fast for biochemical analysis. The following laboratory investigations were performed: fasting blood sugar (FBS) and post-prandial blood sugar (PPBS) measured using standard enzymatic methods; glycated haemoglobin (HbA1c) estimated using high-performance liquid chromatography; serum uric acid measured by enzymatic colorimetric method; lipid profile, including HDL cholesterol; and urine albumin-creatinine ratio (ACR) measured from a spot urine sample.

Calculation of Uric Acid-to-HDL Cholesterol Ratio (UHR): UHR was calculated for each participant using the formula: $\text{UHR} = \text{Serum uric acid (mg/dL)} / \text{HDL cholesterol (mg/dL)}$. Based on predefined cut-offs, participants were categorised into low UHR (≤ 0.10) and high UHR (> 0.10).

Assessment of Microvascular Complications: Diabetic retinopathy was assessed by fundus examination performed by an ophthalmologist and classified as present or absent. Diabetic nephropathy was assessed using urine albumin-creatinine ratio and categorised as normoalbuminuria, microalbuminuria, or macroalbuminuria.

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Statistical Analysis: Data were entered into Microsoft Excel and analysed using JASP, Version 0.97.0 (JASP Team, University of Amsterdam; Amsterdam, Netherlands). Continuous variables were expressed as mean $\pm$ standard deviation, while categorical variables were expressed as frequency and percentage. Associations between UHR categories and microvascular complications were analysed using the Chi-square test. Correlation between UHR and glycaemic parameters (FBS, PPBS, HbA1c) was assessed using Pearson's correlation coefficient. A p-value <0.05 was considered statistically significant.

RESULTS

The study enrolled 100 T2DM patients in total. Mean age stood at 55.46 ± 12.34 years, and males formed the larger share of the cohort (62% males, 38% females). The glycaemic profile pointed to poor metabolic control overall: mean HbA1c was $8.42 \pm 1.56\%$, mean post-prandial blood sugar reached 232.50 ± 61.30 mg/dL, and mean fasting blood sugar measured 163.49 ± 43.97 mg/dL (Table 1).

Table 1. Baseline Demographic and Glycaemic Characteristics of the Study Participants (n=100)

Variable	Value
Age (years), mean $\pm$ SD	55.46 ± 12.34
Sex	
Male	62 (62.0%)
Female	38 (38.0%)
Fasting blood sugar (mg/dL), mean $\pm$ SD	163.49 ± 43.97
Post-prandial blood sugar (mg/dL), mean $\pm$ SD	232.50 ± 61.30
HbA1c (%), mean $\pm$ SD	8.42 ± 1.56

Abbreviations: SD, standard deviation; HbA1c, glycatedhaemoglobin.

Turning to biochemical parameters, serum uric acid was raised (>7.0 mg/dL) in 44% of participants, while the remaining 56% fell within the normal range. HDL cholesterol levels were reduced in 72% of the cohort, leaving 28% with normal HDL values. Once the uric acid-to-HDL cholesterol ratio was calculated, 71% of participants fell into the high UHR category (>0.10) and 29% into the low UHR category (≤ 0.10) (Table 2).

Table 2. Distribution of Serum Uric Acid, HDL Cholesterol, and Uric Acid-to-HDL Cholesterol Ratio (UHR)

Parameter	Category	n (%)
Serum uric acid (mg/dL)	≤ 7.0	56 (56.0)
	> 7.0	44 (44.0)
HDL cholesterol (mg/dL)	Low	72 (72.0)
	Normal	28 (28.0)
UHR	≤ 0.10 (Low)	29 (29.0)
	> 0.10 (High)	71 (71.0)

Abbreviations: HDL, high-density lipoprotein; UHR, uric acid-to-HDL cholesterol ratio.

Forty-two percent of the cohort had diabetic retinopathy, with the remaining 58% showing no retinopathy. On renal involvement, the breakdown was 46% normoalbuminuria, 34% microalbuminuria, and 20% macroalbuminuria—meaning over half of participants carried some degree of diabetic nephropathy (Table 3).

Table 3. Prevalence of Diabetic Microvascular Complications

Complication	Category	n (%)
Diabetic retinopathy	Present	42 (42.0)
	Absent	58 (58.0)
Nephropathy (ACR)	Normoalbuminuria	46 (46.0)
	Microalbuminuria	34 (34.0)
	Macroalbuminuria	20 (20.0)

Abbreviations: ACR, albumin-to-creatinine ratio.

Table 4 and Fig. 1 lay out how UHR aligned with diabetic retinopathy. Retinopathy occurred in 49.3% of high-UHR participants against 24.1% of those with low UHR, a difference that reached statistical significance ($p=0.004$). UHR also tracked meaningfully with diabetic nephropathy (Table 4; Fig. 1). In the low-UHR group, 72.4% had normoalbuminuria, 20.7% had microalbuminuria, and 6.9% had macroalbuminuria. The high-UHR group looked rather different: just 35.2% had normoalbuminuria, while microalbuminuria accounted for 39.4% and macroalbuminuria for 25.4%. The way nephropathy severity distributed across UHR categories was statistically significant ($p<0.001$).

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Table 4. Association Between Uric Acid-to-HDL Cholesterol Ratio (UHR) and Microvascular Complications

Complication	UHR ≤ 0.10 (n=29)	UHR > 0.10 (n=71)	p-value
Diabetic retinopathy	7 (24.1%)	35 (49.3%)	0.004
Diabetic nephropathy			
Normoalbuminuria	21 (72.4%)	25 (35.2%)	<0.001
Microalbuminuria	6 (20.7%)	28 (39.4%)	
Macroalbuminuria	2 (6.9%)	18 (25.4%)	

Abbreviations: UHR, uric acid-to-HDL cholesterol ratio. p-values from Chi-square test.

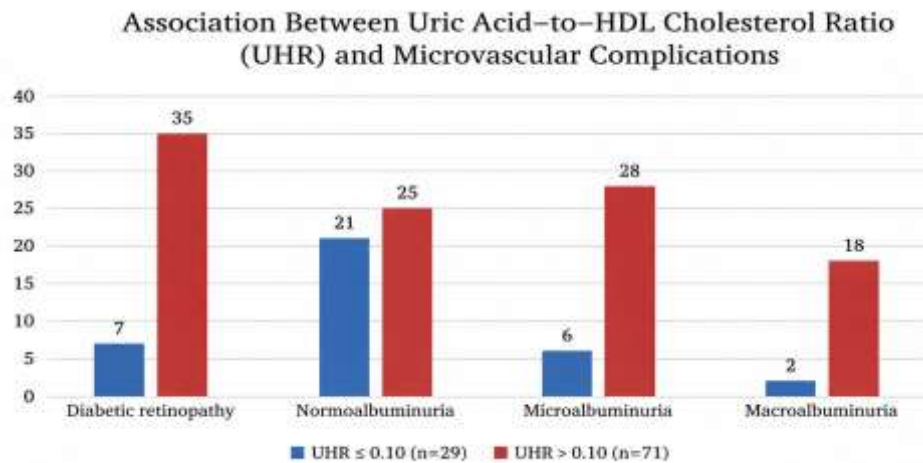


Fig. 1. Association between uric acid-to-HDL cholesterol ratio (UHR) categories and microvascular complications (diabetic retinopathy and nephropathy severity).

Correlation analysis pointed to statistically significant positive links between UHR and glycaemic parameters (Table 5). Positive correlations emerged with post-prandial blood sugar ($r=0.32$, $p=0.001$; Fig. 3), fasting blood sugar ($r=0.29$, $p=0.003$; Fig. 2), and HbA1c ($r=0.41$, $p<0.001$; Fig. 4). Of these, the tightest correlation was with HbA1c.

Table 5. Correlation of Uric Acid-to-HDL Cholesterol Ratio with Glycaemic Parameters

Parameter	Correlation coefficient (r)	p-value
Fasting blood sugar	0.29	0.003
Post-prandial blood sugar	0.32	0.001
HbA1c	0.41	<0.001

Abbreviations: HbA1c, glycatedhaemoglobin. p-values from Pearson's correlation analysis.

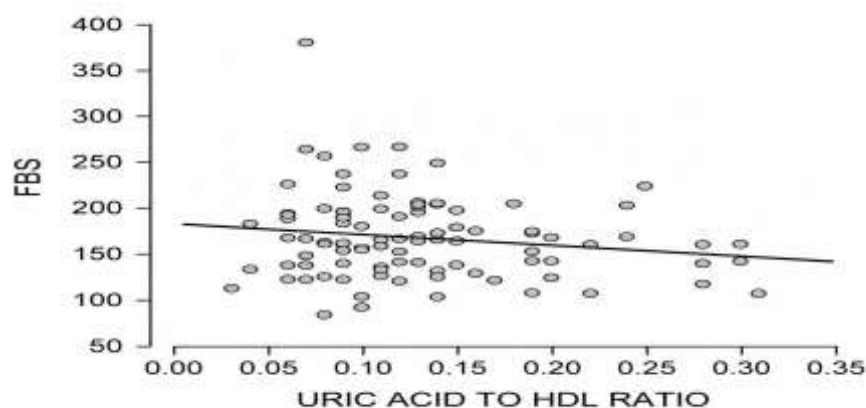


Fig. 2. Scatter plot of uric acid-to-HDL cholesterol ratio (UHR) versus fasting blood sugar (FBS) ($r=0.29$, $p=0.003$).

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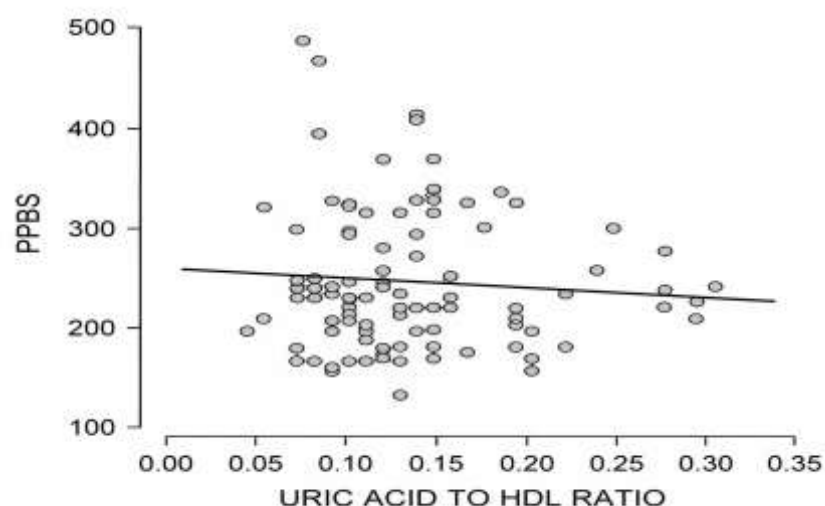


Fig. 3. Scatter plot of uric acid-to-HDL cholesterol ratio (UHR) versus post-prandial blood sugar (PPBS) ($r=0.32$, $p=0.001$).

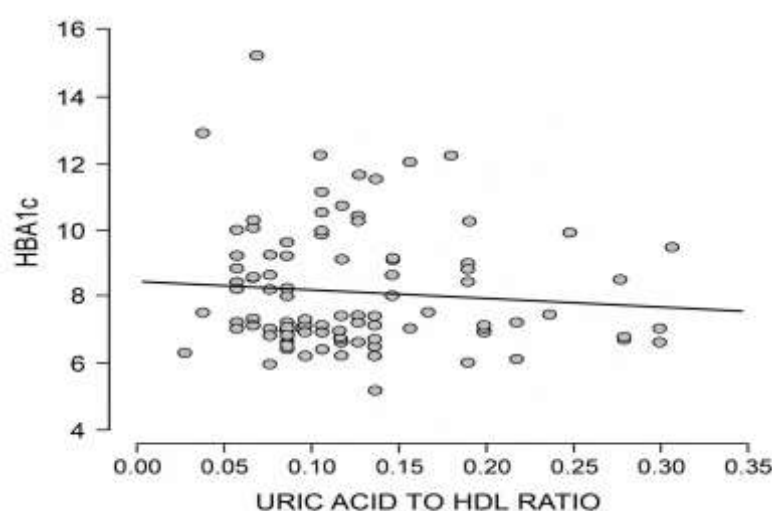


Fig. 4. Scatter plot of uric acid-to-HDL cholesterol ratio (UHR) versus glycatedhaemoglobin (HbA1c) ($r=0.41$, $p<0.001$).

DISCUSSION:

This study examined how UHR relates to microvascular complications in T2DM patients. Three main findings emerged: elevated UHR was highly prevalent, it linked meaningfully to both diabetic retinopathy and diabetic nephropathy, and it correlated positively with glycaemic parameters—HbA1c most strongly. Taken together, these observations point to UHR as a reflection of an integrated metabolic risk state with relevance to diabetic microvascular disease.

Middle-aged individuals with poor glycaemic control made up the bulk of the cohort, with mean HbA1c reaching $8.42 \pm 1.56\%$ (Table 1). This profile fits the broader Indian diabetes picture, where delayed diagnosis and suboptimal long-term glycaemic control are recurring themes, as documented by the International Diabetes Federation and the ICMR–INDIAB study.¹² Sustained hyperglycaemia is firmly established as the central driver of microvascular injury, working through endothelial dysfunction, activation of inflammatory pathways, and oxidative stress.^{3–5}

Diabetic retinopathy was present in 42% of participants in the current cohort, and over half showed evidence of diabetic nephropathy in the form of microalbuminuria or macroalbuminuria (Table 3). These figures sit comfortably alongside reports from tertiary-care settings and capture the heavy microvascular burden that

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accompanies T2DM.⁶⁻⁸

One noteworthy observation was that elevated UHR (71%) was far more prevalent than overt hyperuricaemia (44%) (Table 2). The contribution of low HDL cholesterol—seen in 72% of participants—largely accounts for this gap. HDL cholesterol normally provides endothelial-protective, anti-inflammatory, and antioxidative effects; in T2DM, however, levels often drop and the particles themselves may be functionally compromised through glycation and oxidative modification.¹⁵¹⁶ By bringing both pro-oxidant and protective metabolic components into one figure, UHR may track net vascular risk more faithfully than either serum uric acid or HDL cholesterol viewed in isolation.

Earlier work has linked UHR to insulin resistance and adverse cardiometabolic outcomes. Kocak et al. found UHR to be a strong predictor of metabolic syndrome in T2DM patients,¹⁸ and population-based investigations have tied higher UHR to elevated cardiovascular risk.¹⁹²⁰ The current study carries these observations forward into the domain of diabetic microvascular complications.

Diabetic retinopathy occurred significantly more often among participants with high UHR than among those with low UHR (49.3% vs 24.1%, $p=0.004$) in this study (Table 4). Such a pattern lends support to a connection between raised UHR and retinal microvascular injury.

Findings on the role of serum uric acid in diabetic retinopathy have been mixed across earlier studies. In a prospective cohort of Japanese men with T2DM, Kuwata et al. found higher serum uric acid levels carried an increased risk of newly developed diabetic retinopathy.¹² A positive link between uric acid levels and diabetic retinopathy was likewise suggested by a systematic review from Guo et al.¹⁴ Xia et al., on the other hand, failed to identify an independent association between serum uric acid and diabetic retinopathy in their cohort.¹³

What distinguishes this study from those earlier reports is the use of UHR in place of serum uric acid taken alone. The combined effect of low HDL cholesterol and raised uric acid—both feeding into oxidative stress and endothelial dysfunction within the retinal microvasculature—may account for the stronger association seen here.⁷⁹¹⁰ This combined effect could explain why UHR produced a clearer association with retinopathy than serum uric acid alone managed in some earlier studies.

The strongest association in the present analysis involved UHR and diabetic nephropathy. Within the high-UHR group, only 35.2% had normoalbuminuria, contrasted with 72.4% in the low-UHR group; macroalbuminuria, meanwhile, was present in 25.4% of the high-UHR group versus 6.9% of those with low UHR ($p<0.001$) (Table 4). This graded pattern suggests elevated UHR tracks not just the presence but also the severity of renal involvement.

These observations align with prior literature on the role of uric acid in diabetic kidney disease. Both experimental and clinical work indicate that uric acid contributes to tubulointerstitial inflammation, endothelial dysfunction, and glomerular hypertension.⁹¹⁰ Xia et al. documented an independent link between serum uric acid and diabetic nephropathy in T2DM patients.¹³ The current findings build on this evidence by showing that UHR, drawing on both uric acid and HDL cholesterol, displays a strong association with nephropathy severity.

Significant positive correlations between UHR and glycaemic parameters were apparent—HbA1c ($r=0.41$, $p<0.001$), post-prandial blood sugar ($r=0.32$, $p=0.001$), and fasting blood sugar ($r=0.29$, $p=0.003$) (Table 5). That the strongest correlation was with HbA1c suggests UHR captures chronic glycaemic burden rather than short-term glucose variability. Biologically this fits, since sustained hyperglycaemia drives metabolic dysregulation and oxidative stress, both of which can influence HDL cholesterol levels and uric acid metabolism.⁴¹⁶

Because HDL cholesterol and serum uric acid are routine investigations, UHR can be derived without additional expense. In resource-limited settings, the ratio may serve as a useful adjunctive marker for identifying patients who could benefit from closer monitoring for microvascular complications. Even so, UHR is best regarded as complementary to established diagnostic tools rather than a substitute for them.

LIMITATIONS:

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Several limitations apply to this study. To begin with, the cross-sectional design rules out causal inference regarding elevated UHR and microvascular complications. The single-centre setting paired with a modest sample size also places limits on how broadly the findings can be generalised. In addition, potential confounders—renal function variables, blood pressure control, treatment regimens, and diabetes duration among them—were not stratified within the analysis. Larger prospective multicentre studies are needed to confirm these results and to clarify what role UHR may play in long-term microvascular risk prediction.

CONCLUSION:

A meaningful link between elevated UHR and microvascular complications in T2DM patients emerges from this study. Both the prevalence and severity of diabetic retinopathy and diabetic nephropathy climbed with rising UHR, and the ratio correlated positively with poor glycaemic control, HbA1c in particular. Drawing together pro-oxidant and protective metabolic components into a single measure, UHR captures a composite metabolic risk state with relevance to diabetic microangiopathy. Since both of its components form part of routine laboratory testing, UHR holds promise as a straightforward, cost-effective adjunct for stratifying microvascular risk in everyday clinical practice.

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DECLARATIONS

Data Availability Statement: The data that support the findings of this study are available on request from the corresponding author.

ARTIFICIAL INTELLIGENCE USAGE STATEMENT: The author declared that no Artificial Intelligence tool was used in the preparation of the manuscript.

INFORMED CONSENT: Written informed consent was obtained from the patients who agreed to take part in the study.

CONFLICT OF INTERESTS: The author has no conflicts of interest to declare.

FUNDING: The author declared that this study has received no financial support.

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