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Original Research Article

Serum Uric Acid in Type II Diabetes Mellitus Patients: A Cross-Sectional Study from a Tertiary Care Hospital, Gujarat

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Abstract:

Introduction: Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder associated with significant morbidity through micro- and macrovascular complications. Serum uric acid (SUA) has emerged as a potential biomarker linked to glycaemic dysregulation, dyslipidaemia, and early renal impairment. Despite its clinical relevance, SUA levels in T2DM patients remain understudied in the Indian context, particularly in Gujarat.

Materials and Methods: A cross-sectional study was conducted at a tertiary care hospital, Pramukh Swami Medical College, Karamsad, and enrolling 200 participants: 100 T2DM patients and 100 age-sex–matched healthy controls. Biochemical parameters including fasting plasma glucose (FPG), HbA1c, serum uric acid, lipid profile, serum creatinine, and urine albumin-to-creatinine ratio (UACR) were measured. Statistical analysis was performed using Epi info version 7.

Results: Serum uric acid was significantly elevated in T2DM patients (6.43 ± 1.19 mg/dL) compared to controls (4.84 ± 0.99 mg/dL; $p<0.001$). Hyperuricaemia was present in 34% of diabetic patients versus 8% of controls. SUA correlated positively with triglycerides ($r=0.46$), FPG ($r=0.34$), HbA1c ($r=0.29$), and UACR, and negatively with HDL-C ($r=-0.31$). ROC analysis revealed SUA >6.2 mg/dL as the optimal cut-off for predicting albuminuria (AUC=0.76; sensitivity 72%, specificity 68%).

Conclusion: Serum uric acid is significantly elevated in T2DM patients and independently correlates with atherogenic dyslipidaemia and early renal damage. Routine SUA monitoring may serve as an accessible and cost-effective biomarker for cardiorenal risk stratification in diabetic populations.

Keywords: Albuminuria, Dyslipidaemia, eGFR, Serum uric acid, Type 2 diabetes mellitus

Introduction

Type 2 diabetes mellitus (T2DM) constitutes one of the most significant public health challenges of the twenty-first century. India bears a disproportionate burden of this epidemic, with an estimated 101 million adults living with diabetes as of 2023, positioning the country as the global diabetes capital.[1] The progressive nature of T2DM leads to a spectrum of micro- and macrovascular complications, including diabetic nephropathy, retinopathy, neuropathy, and cardiovascular disease, which collectively contribute to premature mortality and disability.

Uric acid (UA), the terminal catabolite of purine metabolism in humans, has garnered considerable scientific attention beyond its classical association with gout and hyperuricaemia. Mechanistically, UA diminishes endothelial nitric oxide bioavailability, promotes oxidative stress, activates the renin-angiotensin-aldosterone system (RAAS), and stimulates pro-inflammatory cytokines such as tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6).[2] These pathways are intimately implicated in the pathogenesis of insulin resistance, endothelial dysfunction, and progression of diabetic kidney disease (DKD).

Several Indian studies have documented disparate findings regarding SUA in T2DM. Purohit et al. (2025) [3] from Uttarakhand documented significantly elevated SUA in T2DM patients (8.40 ± 2.80 mg/dL) compared to controls (5.04 ± 1.81 mg/dL). Conversely, Patil and Prathik (2019) [4] reported lower SUA in uncomplicated T2DM (3.49 mg/dL) versus controls (4.86 mg/dL), attributing this to the uricosuric effect of glycosuria. This apparent paradox—hypouricaemia in uncomplicated and hyperuricaemia in complicated T2DM—has been elucidated by Lalnunpuii et al. (2020),[5] who demonstrated that SUA was lower in T2DM without complications but significantly elevated in those with complications (6.04 ± 1.72 mg/dL vs. control 5.20 ± 1.41 mg/dL).

The relationship between SUA and atherogenic dyslipidaemia is particularly relevant. Elevated SUA has been correlated with increased triglycerides, reduced HDL-cholesterol, and a higher triglyceride-to-HDL ratio—key components of the metabolic syndrome phenotype common in South Asian populations.[6] A recent meta-analysis by Zhao and Zhao (2026)[7] confirmed that hyperuricaemia significantly increases the risk of diabetic kidney disease (OR=1.85; 95% CI: 1.52–2.26; p<0.001) and is associated with lower eGFR, underscoring SUA's utility as an early renal biomarker.

Despite the growing body of evidence, there remains a paucity of data from Gujarat, a state with a high prevalence of T2DM and metabolic syndrome driven by dietary patterns rich in refined carbohydrates and saturated fats.[8] The present study was therefore designed to evaluate serum uric acid levels and their correlations with lipid profile, glycaemic indices, and markers of renal function in T2DM patients attending a tertiary care hospital in Gujarat, India.

Materials and Methods

Study Design and Setting: This cross-sectional observational study was conducted at Shree Krishna Hospital tertiary care hospitals, attached to Pramukh Swami Medical College, Karamsad from January 2023 to December 2023. The study protocol was approved by the Institutional Ethics Committee (IEC/HMPCMCE/65/Faculty/10/33/16) and conducted in accordance with the Declaration of Helsinki principles. Written informed consent was obtained from all participants prior to enrollment.

Study Population and Sample Size: The study included 200 participants divided into two groups: 100 patients with established T2DM (cases) and 100 age-matched healthy controls. Sample size calculation was based on an expected dyslipidemia prevalence of 70% in T2DM patients versus 25% in controls, with 80% power and 5% significance level. Participants were recruited through systematic random sampling from outpatient departments and inpatient wards.

Inclusion criteria: The study included adults aged 30–70 years diagnosed with Type 2 Diabetes Mellitus according to American Diabetes Association (ADA) criteria (FPG ≥126 mg/dL or HbA1c ≥6.5%) with disease duration >1 year and who provided informed consent. Age-matched healthy controls with normal fasting glucose (<100 mg/dL), HbA1c <5.7%, and no family history of diabetes were also enrolled after consent.[11]

Exclusion Criteria: Type 1 diabetes, gestational diabetes, patients on insulin therapy, active thyroid disorders, chronic kidney disease (eGFR <60 mL/min/1.73m²), liver dysfunction, acute infections, pregnancy, patients on lipid-lowering medications within 6 weeks of study entry, and history of cardiovascular events within 3 months.

Data Collection and Clinical Assessment: Standardized questionnaires were used to collect demographic data, medical history, medication details, and lifestyle factors.

Anthropometric Measurements: Height and weight were recorded using a wall-mounted stadiometer and calibrated digital scale, respectively. BMI was calculated as weight (kg)/height (m²). Obesity was defined as BMI ≥25 kg/m² according to Asian criteria

Blood Pressure Measurements: Blood pressure was measured in the right arm after 5 minutes of rest using a mercury sphygmomanometer; three readings taken at 5-minute intervals were averaged. Hypertension was defined as systolic BP ≥140 mmHg, diastolic BP ≥90 mmHg, or current use of antihypertensive medication.

Laboratory Investigations: After 12-hour overnight fasting, venous blood samples (5 mL) were collected in four vacutainers - plain for lipid profile, sodium fluoride for Fasting plasma glucose,and EDTA for HbA1c. Samples were collected with an aseptic blood collection technique by use of sterile gloves and thorough disinfection of venipuncture site with 70% ethyl alcohol. All the samples were collected in sitting position. Samples were centrifuged within one hour at 1500 rpm for 15 minutes. These would be processed to obtain serum/plasma for the estimation of serum lipid profile and fasting plasma glucose. Estimation of fasting lipid profile (Triacylglycerol, Total Cholesterol, and HDL-C), Fasting plasma glucose, and HbA1c were carried out on fully automated COBAS INTEGRA 400 PLUS clinical chemistry analyzer. During the course of the study there was no change in the equipment, reagent, Calibration standards and controls. Before starting the analysis, the instrument was calibrated using calibrators and the controls were checked at different concentrations of the analytes.

HbA1c is measured on COBAS INTEGRA systems using monoclonal antibodies attached to latex particles. Fasting plasma glucose (FPG) was measured using the glucose oxidase method. Comprehensive lipid profile analysis included:

- Total cholesterol (TC) - enzymatic colorimetric method with Endpoint CHOD-POD Method
- Triglycerides (TG) - Colorimetric Endpoint GPO-PAP Method
- High-density lipoprotein cholesterol (HDL-C) - Homogenous Enzymatic Colorimetric Assay
- Low-density lipoprotein cholesterol (LDL-C) - Friedewald formula
- Very-low-density lipoprotein cholesterol (VLDL-C) - calculated as TG/5

Diagnostic Criteria[11]**:**

T2DM: It was diagnosed according to American Diabetes Association criteria: FPG ≥126 mg/dL or 2-hr PG ≥200 mg/dL or HbA1c ≥6.5%.

Dyslipidemia: Dyslipidemia was defined according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria as total cholesterol (TC) >200 mg/dL, triglycerides (TG) >150 mg/dL, low HDL-C (<40 mg/dL in males, <50 mg/dL in females), or high LDL-C (>130 mg/dL). Mixed dyslipidemia was defined as the presence of two or more abnormalities.

Atherogenic Indices: Atherogenic indices were calculated using the TC/HDL-C ratio (Castelli Risk Index I) and TG/HDL-C ratio (Atherogenic Index of Plasma).

Definitions

Hyperuricaemia: was defined as SUA >7.0 mg/dL in males and >6.0 mg/dL in females.[10] Microalbuminuria was defined as UACR 30–300 mg/g creatinine, and macroalbuminuria as UACR >300 mg/g. Atherogenic dyslipidaemia was defined as TG >150 mg/dL, HDL-C <40 mg/dL (males) or <50 mg/dL (females), and/or LDL-C >100 mg/dL.

Statistical Analysis

Data were entered into Microsoft Excel and analysed using SPSS version 23.0 (IBM Corp., USA). Continuous variables were expressed as mean ± standard deviation (SD) and compared using the independent-samples t-test. Categorical variables were expressed as frequencies and percentages and compared using the chi-square (χ^2) test. Pearson's correlation coefficient (r) was used to assess correlations between SUA and other continuous variables. Receiver operating characteristic (ROC) curve analysis was performed to determine the optimal SUA cut-off for predicting albuminuria. A p-value of <0.05 was considered statistically significant.

Results

Table 1 presents the baseline demographic and clinical characteristics of the study population. The two groups were well-matched for age (p=0.42) and sex distribution. Diabetic patients had significantly higher BMI (25.9 ± 3.4 vs. 23.2 ± 2.1 kg/m²; p<0.001), higher rates of obesity (37% vs. 16%; p<0.01), and significantly elevated blood pressure parameters and hypertension prevalence (42% vs. 18%; p<0.01).

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population

Parameter	Control (n=100)	Diabetes (n=100)	Total (n=200)	p-value
Age (years, mean ± SD)	49.8 ± 11.6	51.3 ± 10.4	50.6 ± 10.9	0.42
Male/Female	50/50	50/50	100/100	1
BMI (kg/m²)	23.2 ± 2.1	25.9 ± 3.4	24.5 ± 3.0	<0.001
Obesity – n (%)	16 (16%)	37 (37%)	53 (26.5%)	<0.01
SBP (mmHg)	121.4 ± 9.2	132.5 ± 12.3	126.9 ± 11.5	<0.001
DBP (mmHg)	78.3 ± 7.4	83.9 ± 8.6	81.1 ± 8.3	<0.01
Hypertension – n (%)	18 (18%)	42 (42%)	60 (30%)	<0.01
Duration of Diabetes (yrs)	—	7.1 ± 4.8	—	—
Smokers – n (%)	10 (10%)	14 (14%)	24 (12%)	0.38
Alcoholism – n (%)	9 (9%)	17 (17%)	26 (13%)	0.12

As shown in Table 2, FPG was markedly elevated in the diabetic group (162.0 ± 44.5 mg/dL vs. 98.2 ± 6.8 mg/dL; p<0.001), as was HbA1c (7.88 ± 1.48% vs. 5.78 ± 0.33%; p<0.001). Diabetic patients exhibited significantly lower haemoglobin levels (12.8 ± 1.6 g/dL vs. 13.9 ± 1.4 g/dL) with a higher prevalence of anaemia (38% vs. 14%; p<0.001) and elevated ESR (24.8 ± 11.3 vs. 14.2 ± 6.7 mm/h; p<0.001).

Table 2: Comparison of Biochemical Parameters between control and diabetic patients

Parameter	Control	Diabetes	Total	p-value
FPG (mg/dL)	98.2 ± 6.8	162.0 ± 44.5	130.1 ± 45.0	<0.001
HbA1c (%)	5.78 ± 0.33	7.88 ± 1.48	6.83 ± 1.5	<0.001
Hemoglobin (g/dL)	13.9 ± 1.4	12.8 ± 1.6	13.4 ± 1.6	<0.001
Anemia – n (%)	14 (14%)	38 (38%)	52 (26%)	<0.001
ESR (mm/h)	14.2 ± 6.7	24.8 ± 11.3	19.5 ± 9.8	<0.001
ESR ≥ 20 mm/h – n (%)	18 (18%)	56 (56%)	74 (37%)	<0.001

Table 3 summarises RFT parameters. Mean SUA was significantly higher in the diabetic group (6.43 ± 1.19 mg/dL) compared to controls (4.84 ± 0.99 mg/dL; p<0.001). Hyperuricaemia was observed in 34% of T2DM patients versus 8% of controls. eGFR was significantly lower in the diabetic group (74.6 ± 25.7 vs. 92.4 ± 22.8 mL/min/1.73 m²; p<0.001), and 22% of diabetic patients had eGFR <60 mL/min/1.73 m² compared to only 6% of controls. UACR was significantly elevated in diabetic patients (56.1 ± 42.2 vs. 19.4 ± 9.3 mg/g Cr; p<0.001), with 38% demonstrating microalbuminuria and 8% macroalbuminuria (Figure 1).

Table 3: Comparison of RFT between control and diabetic patients

Parameter	Control (n=100)	Diabetes (n=100)	Total (n=200)	p-value
Serum Uric Acid (mg/dL)	4.84 ± 0.99	6.43 ± 1.19	5.64 ± 1.35	<0.001
Normal SUA – n (%)	92 (92.0%)	66 (66.0%)	158 (79.0%)	<0.001
Hyperuricemia – n (%)	8 (8.0%)	34 (34.0%)	42 (21.0%)	
Serum Creatinine (mg/dL)	0.73 ± 0.19	0.74 ± 0.22	0.74 ± 0.21	0.97
eGFR (mL/min/1.73 m²)	92.4 ± 22.8	74.6 ± 25.7	83.5 ± 26.3	<0.001
eGFR <60 mL/min/1.73 m² – n (%)	6 (6%)	22 (22%)	28 (14%)	<0.01
Urine Albumin/Creatinine Ratio (mg/g Cr)	19.4 ± 9.3	56.1 ± 42.2	37.8 ± 35.5	<0.001

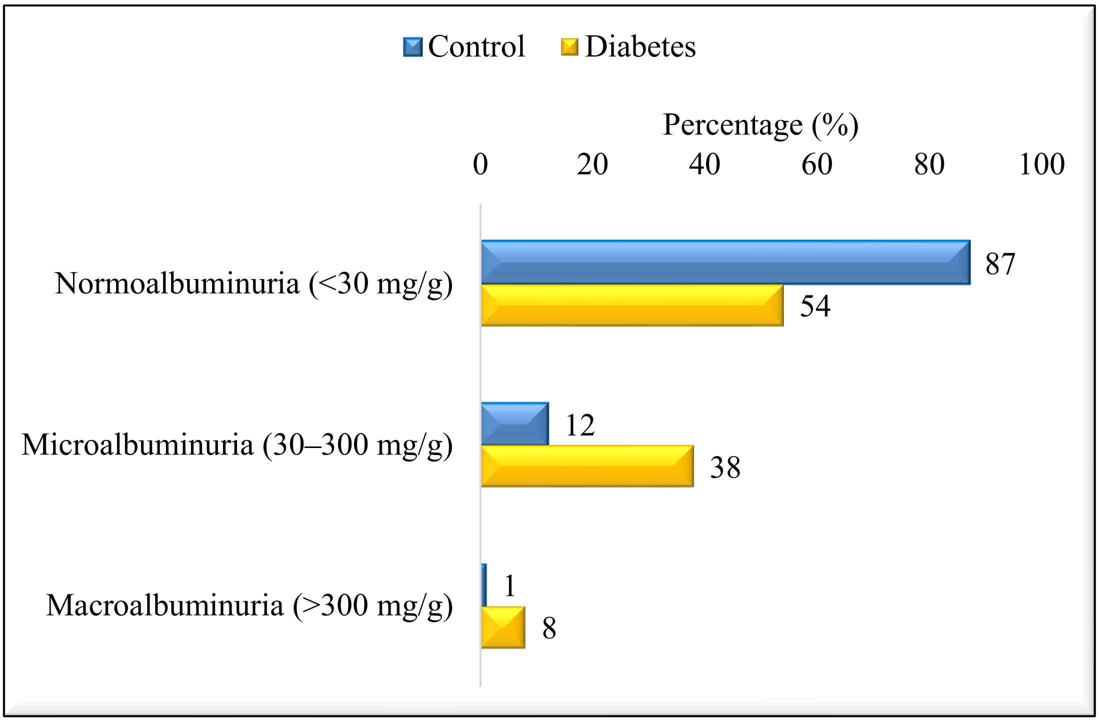

Figure 1: Comparison of albuminuria between control and diabetic patients

Among diabetic patients, those with albuminuria (UACR ≥30 mg/g; n=46) had significantly higher SUA (7.12 ± 1.04 vs. 5.78 ± 0.89 mg/dL; p<0.001), HbA1c (8.31% vs. 7.42%; p=0.002), longer diabetes duration (8.8 vs. 5.6 years; p<0.001), lower eGFR (58.3 vs. 88.5 mL/min/1.73 m²; p<0.001), and higher triglycerides (212 vs. 164 mg/dL; p=0.001) compared to those with normoalbuminuria (Table 4).

Table 4: Comparison Between Normoalbuminuria and Albuminuria Groups

Parameter	Normoalbuminuria (n=54)	Albuminuria (n=46)	p-value
Serum Uric Acid (mg/dL)	5.78 ± 0.89	7.12 ± 1.04	<0.001
HbA1c (%)	7.42 ± 1.11	8.31 ± 1.36	0.002
Duration (years)	5.6 ± 3.4	8.8 ± 4.9	<0.001
eGFR (mL/min/1.73 m²)	88.5 ± 19.6	58.3 ± 24.2	<0.001
Triglycerides (mg/dL)	164 ± 52	212 ± 64	0.001

ROC analysis identified an SUA cut-off of 6.2 mg/dL as the optimal threshold for predicting albuminuria in T2DM patients, with an AUC of 0.76 (95% CI: 0.66–0.85; p<0.001), sensitivity of 72%, and specificity of 68% (Table 5).

Table 5: ROC Curve Analysis of Serum Uric Acid for Predicting Albuminuria

Parameter	AUC	95% CI	Optimal Cut-off (mg/dL)	Sensitivity	Specificity	p-value
Serum Uric Acid	0.76	0.66–0.85	6.2	72%	68%	<0.001

Males demonstrated significantly higher SUA (6.05 ± 1.18 vs. 5.23 ± 1.39 mg/dL; p<0.01) and serum creatinine (0.85 ± 0.19 vs. 0.62 ± 0.15 mg/dL; p<0.001) compared to females. However, eGFR and UACR did not differ significantly by gender (Table 6).

Table 6: Gender-wise Comparison of Serum Uric Acid and Lipid Parameters

Parameter	Male (n=100)	Female (n=100)	p-value
Serum Uric Acid (mg/dL)	6.05 ± 1.18	5.23 ± 1.39	<0.01
Serum Creatinine (mg/dL)	0.85 ± 0.19	0.62 ± 0.15	<0.001
eGFR (mL/min/1.73 m²)	78.4 ± 25.2	70.8 ± 26.3	0.08
eGFR <60 mL/min/1.73 m² – n (%)	13 (26%)	9 (18%)	0.31
Urine Albumin/Creatinine Ratio (mg/g Cr)	61.2 ± 44.5	51.4 ± 39.1	0.19
Normoalbuminuria (<30 mg/g)	23 (46%)	31 (62%)	0.26
Microalbuminuria (30–300 mg/g)	22 (44%)	16 (32%)	
Macroalbuminuria (>300 mg/g)	5 (10%)	3 (6%)	

In the diabetic group, SUA demonstrated significant positive correlations with triglycerides (r=0.46; p<0.001), TG/HDL-C ratio (r=0.48; p<0.001), FPG (r=0.34; p=0.001), HbA1c (r=0.29; p=0.003), LDL-C (r=0.32; p=0.001), and BMI (r=0.25; p=0.01), and a significant negative correlation with HDL-C (r=−0.31; p=0.002). These correlations were weak and largely non-significant in the control group (Table 7).

Table 7: Correlation of Serum Uric Acid with Glycaemic and Lipid Parameters

Parameter	Diabetic (r)	Diabetic p value	Control (r)	Control p value
Fasting Plasma Glucose (mg/dL)	0.34	0.001	0.11	0.28
HbA1c (%)	0.29	0.003	0.07	0.42
Total Cholesterol (mg/dL)	0.27	0.006	0.09	0.33
Triglycerides (mg/dL)	0.46	<0.001	0.21	0.04
HDL-C (mg/dL)	−0.31	0.002	−0.17	0.09
LDL-C (mg/dL)	0.32	0.001	0.14	0.16
VLDL-C (mg/dL)	0.39	<0.001	0.19	0.06
Serum Creatinine (mg/dL)	0.22	0.03	0.10	0.29
BMI (kg/m²)	0.25	0.01	0.08	0.37
TC/HDL-C Ratio	0.34	0.001	0.13	0.19
TG/HDL-C Ratio	0.48	<0.001	0.21	0.04

Discussion

The present study provides robust evidence that serum uric acid is significantly elevated in T2DM patients attending a tertiary care centre in Gujarat, with a mean SUA of 6.43 ± 1.19 mg/dL compared to 4.84 ± 0.99 mg/dL in healthy controls (p<0.001). Hyperuricaemia was detected in 34% of the diabetic group versus only 8% of controls. These findings are concordant with several recent Indian studies. Purohit et al. (2025) [3] reported mean SUA of 8.40 ± 2.80 mg/dL in T2DM patients against 5.04 ± 1.81 mg/dL in controls, with significant positive correlations with both fasting and postprandial blood sugar. Similarly, Kalyani et al. (2019) [11] reported mean SUA of 5.08 ± 1.42 mg/dL in T2DM patients versus 3.55 ± 0.62 mg/dL in controls, while Periyandavar et al. (2020) [6] documented mean SUA of 5.26 ± 1.39 mg/dL in diabetics against 3.54 ± 0.62 mg/dL in controls.

A crucial nuance in the SUA-T2DM relationship is the influence of glycaemic control and disease complications. The apparent contradiction—some studies reporting low SUA and others high SUA in T2DM—is explained by the stage and complexity of disease. Patil and Prathik (2019) [4] observed hypouricaemia in uncomplicated T2DM (mean 3.49 mg/dL), attributing this to glycosuria-induced uricosuria: when blood glucose exceeds the renal threshold (~180 mg/dL), excess glucose in tubular fluid competitively inhibits urate reabsorption, promoting urinary uric acid excretion. In contrast, Lalnunpuii et al. (2020) [5] demonstrated that T2DM patients with complications had significantly higher SUA (6.04 ± 1.72 mg/dL) compared to those without complications (4.82 ± 1.85 mg/dL). The present study's population, drawn from a tertiary care centre, likely includes a higher proportion of patients with established complications and longer disease duration (mean 7.1 ± 4.8 years), explaining the elevated SUA observed.

The relationship between SUA and glycaemic parameters observed in this study—significant positive correlations with FPG (r=0.34) and HbA1c (r=0.29)—is consistent with the existing literature. Shirsath et al. (2019) [12] reported highly significant associations between SUA and HbA1c (p<0.001) and FBG (p=0.004) in T2DM patients. The Solanki et al. (2021) [13] study further demonstrated that SUA rises progressively with diabetes duration, with mean SUA increasing from 6.80 ± 0.89 mg/dL in patients with 2–6 years of disease to 7.72 ± 2.90 mg/dL in those with 7–10 years (r=+0.7; p=0.001). This aligns with the present finding of higher SUA in the albuminuria subgroup, which also had longer disease duration (8.8 vs. 5.6 years).

The dyslipidaemic profile in T2DM patients corroborated by the present study—characterised by elevated triglycerides, reduced HDL-C, and elevated LDL-C—mirrors the classical atherogenic pattern of insulin resistance. The strong positive correlation of SUA with triglycerides (r=0.46) and TG/HDL-C ratio (r=0.48), and negative correlation with HDL-C (r=−0.31) in the diabetic group, but not in controls, suggests that SUA is specifically linked to the atherogenic milieu of T2DM. These associations are mechanistically plausible: insulin resistance impairs lipoprotein lipase activity, elevating circulating TG and reducing HDL-C, while simultaneously reducing renal urate excretion through enhanced proximal tubular reabsorption.[6] Kalyani et al. (2019) [11] and Periyandavar et al. (2020) [6] similarly documented statistically significant associations between SUA, dyslipidaemia, and central obesity (elevated BMI and waist-hip ratio) in their study populations.

The relationship between SUA and renal function is a cornerstone finding of this study. Patients with albuminuria had significantly higher SUA (7.12 mg/dL vs. 5.78 mg/dL; p<0.001) and lower eGFR (58.3 vs. 88.5 mL/min/1.73 m²; p<0.001). The ROC analysis identified an SUA cut-off of 6.2 mg/dL (AUC=0.76) as a useful predictor of albuminuria. These findings are strongly supported by the meta-analysis of Zhao and Zhao (2026),[7] which demonstrated that hyperuricaemia in T2DM patients was associated with significantly lower eGFR (MD=4.40 mL/min; 95% CI: 0.66–8.14; p=0.02) and an 85% higher risk of DKD (OR=1.85; 95% CI: 1.52–2.26; p<0.001). The pathophysiological mechanisms involve urate crystal deposition in the renal interstitium causing local inflammation, RAAS activation leading to glomerular hypertension, and direct tubulotoxic effects of soluble uric acid.[7] Maniappan et al. (2017)[14] demonstrated in 100 T2DM patients that elevated SUA was significantly associated with abnormal renal ultrasonography (p=0.016), fundus changes (p=0.001), and peripheral neuropathy (p=0.001), confirming its role as a marker of microvascular damage.

The gender-specific pattern observed—higher SUA in males—is consistent with the established physiological role of oestrogen in promoting renal urate excretion.[6] post-menopausal women, however, lose this protective effect and may experience SUA levels approaching those of males, a consideration warranting sex-stratified analyses in future studies. The fact that eGFR and UACR did not differ significantly by gender in the present study suggests that renal functional impairment may be more dependent on glycaemic control and disease duration than on gender-related SUA differences.

Clinically, the findings advocate for the integration of SUA measurement into routine biochemical monitoring of T2DM patients. As affirmed by Kalyani et al. (2019) [11] and Periyandavar et al. (2020),[6] SUA >4 mg/dL in T2DM patients should be regarded as a 'red flag' for cardiovascular risk stratification. The accessibility, low cost, and reproducibility of SUA measurement make it an ideal candidate biomarker in resource-constrained tertiary care settings prevalent across India. Furthermore, as highlighted by Zhao and Zhao (2026),[7] there is an urgent need for phase III randomised controlled trials evaluating whether urate-lowering therapies (allopurinol or febuxostat) can delay eGFR decline and reduce cardiovascular events in T2DM patients with hyperuricaemia and microalbuminuria.

The primary limitation of this study is its cross-sectional design, which precludes causal inference. The relatively modest sample size of 100 diabetic patients, while adequate for initial analyses, may limit the power to detect associations in subgroup analyses. Selection bias inherent to tertiary care settings and the absence of dietary history—a key determinant of SUA—represent additional limitations. Future studies should adopt prospective designs with larger cohorts, incorporate dietary purine assessment, and include post-menopausal female subgroup analyses.

Conclusion

Serum uric acid is significantly elevated in T2DM patients compared to healthy controls, with a prevalence of hyperuricaemia of 34% in the diabetic cohort. SUA demonstrates significant positive correlations with atherogenic dyslipidaemia—particularly triglycerides and the TG/HDL-C ratio—and with indices of glycaemic dysregulation. Furthermore, elevated SUA is strongly associated with albuminuria and reduced eGFR, highlighting its role as an early and accessible biomarker of diabetic kidney disease. An SUA cut-off of 6.2 mg/dL provides clinically useful sensitivity and specificity for predicting renal injury in T2DM. Routine measurement of SUA should be incorporated into the standard biochemical panel for T2DM patients in tertiary care settings, facilitating early identification of cardiorenal risk and timely intervention.

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