

## Association of Serum Uric Acid Levels with Glycemic Control and Cardiovascular Risk Factors in Patients with Type 2 Diabetes Mellitus: A Cross-Sectional Study

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### Abstract

**Background:** Type 2 diabetes mellitus (T2DM) is frequently accompanied by obesity, hypertension, dyslipidemia, and other cardiovascular risk factors. Serum uric acid (SUA) has emerged as a potential marker of cardiometabolic dysfunction, although its relationship with glycemic control remains inconsistent. This study evaluated the association of SUA with glycemic control and cardiovascular risk factors in patients with T2DM.

**Materials and Methods:** This hospital-based cross-sectional study included 180 adults with established T2DM at a tertiary-care teaching hospital in India. Clinical and anthropometric parameters, blood pressure, fasting plasma glucose, HbA1c, SUA, lipid profile, and serum creatinine were assessed. Associations were evaluated using appropriate comparative tests, correlation analysis, and multivariable regression, with  $p < 0.05$  considered statistically significant.

**Results:** The mean age was  $55.2 \pm 10.6$  years; 56.7% were male. Mean SUA and HbA1c were  $5.9 \pm 1.5$  mg/dL and  $8.1 \pm 1.6\%$ , respectively. Patients with  $\text{HbA1c} \geq 7\%$  had significantly higher SUA than those with  $\text{HbA1c} < 7\%$  ( $6.1 \pm 1.5$  vs.  $5.4 \pm 1.3$  mg/dL;  $p = 0.005$ ). SUA correlated positively with HbA1c ( $r = 0.31$ ,  $p < 0.001$ ), BMI ( $r = 0.29$ ,  $p < 0.001$ ), systolic blood pressure ( $r = 0.23$ ,  $p = 0.002$ ), triglycerides ( $r = 0.36$ ,  $p < 0.001$ ), and LDL-C ( $r = 0.22$ ,  $p = 0.003$ ), and inversely with HDL-C ( $r = -0.28$ ,  $p < 0.001$ ). Hypertriglyceridemia showed the strongest independent association with hyperuricemia (AOR 2.63, 95% CI 1.34–5.16;  $p = 0.005$ ). SUA remained independently associated with HbA1c on multiple linear regression ( $\beta = 0.23$ ,  $p = 0.002$ ).

**Conclusion:** Higher SUA levels were significantly associated with poorer glycemic control and an adverse cardiovascular risk profile in patients with T2DM. SUA may therefore serve as a simple additional marker for identifying patients with clustering of cardiometabolic risk factors; however, the cross-sectional design precludes causal inference.

**Keywords:** Serum Uric Acid; Type 2 Diabetes Mellitus; HbA1c; Glycemic Control; Hyperuricemia; Cardiovascular Risk; Dyslipidemia.

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### Introduction

Type 2 diabetes mellitus (T2DM) is a chronic metabolic disorder characterized by persistent hyperglycemia arising from varying degrees of insulin resistance and impaired insulin secretion. Beyond abnormalities in glucose metabolism, T2DM is frequently accompanied by obesity, hypertension, dyslipidemia, endothelial dysfunction, and chronic low-grade inflammation. The coexistence of these abnormalities substantially increases the risk of atherosclerotic cardiovascular disease (ASCVD), which remains an important contributor to morbidity and mortality among individuals with diabetes [1]. Contemporary diabetes management therefore extends beyond

glycemic control and emphasizes simultaneous identification and modification of cardiovascular risk factors. Serum uric acid (SUA), the final product of purine metabolism in humans, has traditionally been evaluated mainly in relation to gout and urate nephrolithiasis. In recent years, however, increasing attention has been directed toward its potential association with metabolic and cardiovascular disorders. Elevated SUA has been linked with insulin resistance, hypertension, obesity, dyslipidemia, endothelial dysfunction, oxidative stress, and inflammatory pathways, suggesting that uric acid may reflect a broader adverse cardiometabolic milieu rather than an

isolated biochemical abnormality [2,3]. The relationship between SUA and glucose metabolism is particularly complex. Insulin resistance may reduce renal urate excretion and thereby increase circulating uric acid concentrations, whereas marked hyperglycemia and glycosuria can enhance urinary uric acid excretion. Consequently, the association between SUA and glycemic indices may differ according to the degree of metabolic disturbance, renal function, and treatment status. Recent Indian evidence has demonstrated an inverse relationship between SUA and glycated hemoglobin (HbA1c) among individuals with diabetes, highlighting that the relationship is not necessarily linear [4]. Similarly, a large population-based Indian study involving 2,976 adults found that higher SUA was associated with several cardiovascular risk factors, while HbA1c showed an inverse trend across increasing SUA levels among participants with diabetes [5].

SUA may also be related to the characteristic lipid abnormalities observed in T2DM. An Indian study among patients with T2DM reported significant positive correlations of SUA with triglycerides and very-low-density lipoprotein cholesterol and an inverse correlation with high-density lipoprotein cholesterol [6]. More recently, a cross-sectional study of 1,036 patients with T2DM demonstrated that hyperuricemia was independently associated with dyslipidemia and hypertriglyceridemia even after adjustment for relevant confounding variables [7]. These observations support the possibility that SUA may provide additional information regarding the clustering of metabolic and cardiovascular risk factors in patients with diabetes.

Despite growing evidence, the clinical significance of SUA in T2DM remains uncertain. Reported associations with glycemic control are inconsistent, and elevated SUA may coexist with several established cardiovascular risk determinants, making it difficult to distinguish an independent relationship from the effects of obesity, insulin resistance, renal function, hypertension, and dyslipidemia. Moreover, population-specific data remain important because cardiometabolic risk profiles may vary considerably across different patient groups.

Therefore, the present cross-sectional study was undertaken to evaluate the association of serum uric acid levels with glycemic control and cardiovascular risk factors in patients with type 2 diabetes mellitus.

The study aimed to examine the relationship of SUA with HbA1c and selected cardiovascular risk parameters, including blood pressure, body mass index, and lipid profile. Understanding these associations may help clarify the potential role of

SUA as an additional, readily measurable marker of cardiometabolic risk in patients with T2DM.

## Materials and Methods

**Study Design and Setting:** This hospital-based cross-sectional study was conducted in the Department of Medicine at a tertiary-care teaching hospital in India over a period of 12 months. Adult patients with established type 2 diabetes mellitus (T2DM) attending the outpatient department or admitted to the medical wards during the study period were screened for eligibility. Participants fulfilling the predefined selection criteria were enrolled after obtaining written informed consent.

**Study Population:** A total of 180 Patients aged  $\geq 18$  years with an established diagnosis of T2DM were considered eligible for inclusion. Diabetes mellitus was defined according to the prevailing American Diabetes Association diagnostic criteria [1] or on the basis of documented use of glucose-lowering therapy for previously diagnosed T2DM.

## Inclusion Criteria

1. Patients aged  $\geq 18$  years.
2. Patients with established T2DM.
3. Patients willing to provide written informed consent.
4. Patients for whom the required clinical and biochemical investigations could be obtained.

## Exclusion Criteria

- Patients with type 1 diabetes mellitus or gestational diabetes.
- Acute diabetic emergencies, acute severe infection, active malignancy, advanced chronic kidney disease (eGFR  $< 30$  mL/min/1.73 m<sup>2</sup>), or known gout.
- Patients receiving uric acid-lowering therapy such as allopurinol or febuxostat.
- Pregnant women and patients receiving drugs known to alter uric acid metabolism.

## Data Collection and Clinical Assessment:

Demographic and clinical information included age, sex, duration of diabetes, smoking status, alcohol use, current antidiabetic treatment, history of hypertension, dyslipidemia, and cardiovascular disease. Relevant medication history, particularly the use of antihypertensive, lipid-lowering, glucose-lowering, diuretic, and uric acid-lowering therapies, was documented.

Anthropometric measurements included body weight, height, and body mass index (BMI). BMI was calculated as weight in kilograms divided by the square of height in meters (kg/m<sup>2</sup>).

Blood pressure was measured after the participant had rested for at least 5 minutes. Two readings were obtained using an appropriately sized cuff, and the average value was used for analysis.

### Biochemical Investigations

After an overnight fast of approximately 8–12 hours, venous blood samples were collected under aseptic precautions. The following biochemical parameters were assessed:

- Fasting plasma glucose (FPG)
- Glycated hemoglobin (HbA1c)
- Serum uric acid (SUA)
- Total cholesterol (TC)
- Triglycerides (TG)
- High-density lipoprotein cholesterol (HDL-C)
- Low-density lipoprotein cholesterol (LDL-C)
- Serum creatinine

Serum uric acid was measured using an enzymatic uricase-based method. Plasma glucose was estimated using a standardized enzymatic method, while HbA1c was measured by a standardized assay traceable to internationally accepted reference methods. Serum lipid parameters were measured using routine enzymatic methods in the institutional biochemistry laboratory. Estimated glomerular filtration rate (eGFR) was calculated from serum creatinine using an appropriate validated equation.

**Definition of Study Variables:** The primary exposure variable was serum uric acid concentration expressed in mg/dL.

The principal indicator of glycemic control was HbA1c (%). For categorical analyses, participants were classified as having HbA1c <7% or ≥7%, while HbA1c was retained as a continuous variable for correlation and regression analyses.

Cardiovascular risk factors evaluated in relation to SUA included age, sex, BMI, systolic and diastolic blood pressure, hypertension, smoking status, total cholesterol, triglycerides, LDL-C, HDL-C, and duration of diabetes.

**Study Outcomes:** The primary outcome was the association between serum uric acid and glycemic control as assessed by HbA1c. The secondary outcomes included the relationships of SUA with

BMI, systolic and diastolic blood pressure, total cholesterol, triglycerides, LDL-C, HDL-C, fasting plasma glucose, duration of diabetes, and other cardiovascular risk factors. An additional objective was to identify clinical and biochemical factors independently associated with elevated SUA/hyperuricemia after adjustment for potential confounding variables.

**Statistical Analysis:** Data were analyzed using IBM SPSS Statistics version 26.0. Continuous variables were expressed as mean ± standard deviation (SD), while categorical variables were presented as frequencies and percentages. Normality was assessed using the Shapiro–Wilk test. Between-group comparisons were performed using the independent t-test or Mann–Whitney U test for continuous variables and the Chi-square or Fisher’s exact test for categorical variables. Correlations of serum uric acid with HbA1c, lipid parameters, blood pressure, BMI, and other cardiovascular risk factors were assessed using Pearson’s or Spearman’s correlation coefficients. Multivariable linear and logistic regression analyses were performed to identify independent associations after adjustment for potential confounders, with results reported as adjusted coefficients or adjusted odds ratios (AORs) with 95% confidence intervals (CIs). A two-sided p-value <0.05 was considered statistically significant.

**Ethical Considerations:** Written informed consent was obtained from all participants. Confidentiality of patient information was maintained throughout the study, and study procedures were conducted in accordance with the ethical principles of the Declaration of Helsinki.

### Results

A total of 180 patients with type 2 diabetes mellitus were included in the analysis. The mean age was  $55.2 \pm 10.6$  years, and 102 (56.7%) participants were male. The mean duration of diabetes was  $8.1 \pm 5.4$  years. The mean serum uric acid (SUA) level was  $5.9 \pm 1.5$  mg/dL, while the mean HbA1c was  $8.1 \pm 1.6\%$ .

**Table 1: Baseline clinical and biochemical characteristics of study participants (n=180)**

| Parameter                      | Value        |
|--------------------------------|--------------|
| Age (years), mean ± SD         | 55.2 ± 10.6  |
| Male, n (%)                    | 102 (56.7)   |
| Female, n (%)                  | 78 (43.3)    |
| Duration of diabetes (years)   | 8.1 ± 5.4    |
| BMI (kg/m <sup>2</sup> )       | 26.8 ± 4.1   |
| Systolic BP (mmHg)             | 134.6 ± 16.8 |
| Diastolic BP (mmHg)            | 82.4 ± 9.7   |
| Hypertension, n (%)            | 96 (53.3)    |
| Current smoking, n (%)         | 34 (18.9)    |
| Fasting plasma glucose (mg/dL) | 164.7 ± 48.3 |
| HbA1c (%)                      | 8.1 ± 1.6    |
| Serum uric acid (mg/dL)        | 5.9 ± 1.5    |

|                           |              |
|---------------------------|--------------|
| Total cholesterol (mg/dL) | 189.5 ± 39.6 |
| Triglycerides (mg/dL)     | 176.8 ± 72.5 |
| LDL-C (mg/dL)             | 112.7 ± 34.1 |
| HDL-C (mg/dL)             | 42.6 ± 10.2  |
| Serum creatinine (mg/dL)  | 0.96 ± 0.22  |

More than half of the participants were male and hypertensive. Poor glycemic control and an adverse cardiometabolic profile were common, with a mean HbA1c of 8.1% and a mean SUA level of 5.9 mg/dL.

**Table 2: Comparison according to glycemic control**

| Parameter                    | HbA1c <7% (n=48) | HbA1c ≥7% (n=132) | p-value |
|------------------------------|------------------|-------------------|---------|
| Age (years)                  | 53.8 ± 10.1      | 55.7 ± 10.7       | 0.286   |
| Duration of diabetes (years) | 6.2 ± 4.3        | 8.8 ± 5.6         | 0.004   |
| BMI (kg/m <sup>2</sup> )     | 25.8 ± 3.8       | 27.2 ± 4.1        | 0.041   |
| Systolic BP (mmHg)           | 130.5 ± 15.2     | 136.1 ± 17.1      | 0.046   |
| Serum uric acid (mg/dL)      | 5.4 ± 1.3        | 6.1 ± 1.5         | 0.005   |
| Total cholesterol (mg/dL)    | 180.6 ± 36.4     | 192.7 ± 40.2      | 0.069   |
| Triglycerides (mg/dL)        | 151.3 ± 61.8     | 186.1 ± 74.2      | 0.005   |
| LDL-C (mg/dL)                | 104.1 ± 31.5     | 115.8 ± 34.6      | 0.042   |
| HDL-C (mg/dL)                | 45.8 ± 10.4      | 41.4 ± 9.9        | 0.011   |

Patients with HbA1c ≥7% had significantly higher SUA, BMI, systolic blood pressure, triglycerides and LDL-C, and lower HDL-C than those with HbA1c <7%.

**Table 3: Correlation of serum uric acid with glycemic and cardiovascular risk parameters**

| Parameter              | Correlation coefficient (r) | p-value |
|------------------------|-----------------------------|---------|
| HbA1c                  | 0.31                        | <0.001  |
| Fasting plasma glucose | 0.24                        | 0.001   |
| BMI                    | 0.29                        | <0.001  |
| Systolic BP            | 0.23                        | 0.002   |
| Diastolic BP           | 0.15                        | 0.044   |
| Total cholesterol      | 0.20                        | 0.007   |
| Triglycerides          | 0.36                        | <0.001  |
| LDL-C                  | 0.22                        | 0.003   |
| HDL-C                  | -0.28                       | <0.001  |
| Duration of diabetes   | 0.17                        | 0.023   |

SUA correlated positively with HbA1c, fasting glucose, BMI, blood pressure, triglycerides, and LDL-C. The strongest positive correlation was with triglycerides (r=0.36), whereas HDL-C showed an inverse correlation (r=-0.28).

**Table 4: Cardiometabolic characteristics according to serum uric acid status**

| Parameter                    | Normal SUA (n=126) | Hyperuricemia (n=54) | p-value |
|------------------------------|--------------------|----------------------|---------|
| HbA1c (%)                    | 7.8 ± 1.5          | 8.7 ± 1.6            | <0.001  |
| BMI (kg/m <sup>2</sup> )     | 25.9 ± 3.7         | 28.9 ± 4.2           | <0.001  |
| Hypertension, n (%)          | 59 (46.8)          | 37 (68.5)            | 0.008   |
| Systolic BP (mmHg)           | 131.6 ± 15.5       | 141.6 ± 17.5         | <0.001  |
| Triglycerides (mg/dL)        | 159.8 ± 63.7       | 216.5 ± 76.8         | <0.001  |
| LDL-C (mg/dL)                | 108.2 ± 32.6       | 123.2 ± 35.4         | 0.006   |
| HDL-C (mg/dL)                | 44.5 ± 10.0        | 38.2 ± 9.3           | <0.001  |
| Poor glycemic control, n (%) | 85 (67.5)          | 47 (87.0)            | 0.007   |

Participants with hyperuricemia had higher HbA1c, BMI, systolic blood pressure, triglycerides, and LDL-C, together with lower HDL-C. Poor glycemic control was more frequent among patients with hyperuricemia.

**Table 5: Multivariable logistic regression for factors associated with hyperuricemia**

| Predictor                 | Adjusted OR | 95% CI    | p-value |
|---------------------------|-------------|-----------|---------|
| HbA1c ≥7%                 | 2.42        | 1.05–5.58 | 0.038   |
| BMI ≥25 kg/m <sup>2</sup> | 2.18        | 1.10–4.33 | 0.026   |
| Hypertension              | 1.89        | 1.01–3.55 | 0.047   |
| Triglycerides ≥150 mg/dL  | 2.63        | 1.34–5.16 | 0.005   |
| Low HDL-C                 | 2.11        | 1.09–4.10 | 0.027   |
| Current smoking           | 1.34        | 0.62–2.91 | 0.458   |

After adjustment, poor glycemic control, overweight/obesity, hypertension, hypertriglyceridemia and low HDL-C remained independently associated with hyperuricemia. Hypertriglyceridemia showed the strongest association (AOR 2.63, 95% CI 1.34–5.16).

**Table 6: Multiple linear regression for association with HbA1c**

| Predictor            | Standardized $\beta$ | 95% CI for B    | p-value |
|----------------------|----------------------|-----------------|---------|
| Serum uric acid      | 0.23                 | 0.09 to 0.39    | 0.002   |
| Duration of diabetes | 0.21                 | 0.02 to 0.10    | 0.005   |
| BMI                  | 0.15                 | 0.01 to 0.11    | 0.041   |
| Triglycerides        | 0.17                 | 0.001 to 0.007  | 0.019   |
| HDL-C                | -0.13                | -0.04 to -0.001 | 0.048   |

In multivariable linear regression, higher SUA remained independently associated with higher HbA1c ( $\beta=0.23$ ,  $p=0.002$ ) after adjustment for other cardiometabolic factors. Model adjusted  $R^2=0.24$ ; overall  $p<0.001$ .

### Discussion

In the present study, the mean SUA was  $5.9 \pm 1.5$  mg/dL and the mean HbA1c was  $8.1 \pm 1.6\%$ . Patients with HbA1c  $\geq 7\%$  had significantly higher SUA than those with HbA1c  $< 7\%$ . SUA also demonstrated a positive correlation with HbA1c ( $r=0.31$ ,  $p<0.001$ ) and fasting plasma glucose ( $r=0.24$ ,  $p=0.001$ ). Similar observations were reported by Azad et al., who found a positive relationship between SUA and HbA1c among patients with T2DM [8]. The association may partly reflect insulin resistance, because hyperinsulinemia can influence renal tubular handling of urate and thereby contribute to higher circulating SUA concentrations.

Nevertheless, the relationship between uric acid and glycemia is complex and findings across studies have not been uniform. Some studies have demonstrated an inverse relationship between SUA and HbA1c in established diabetes. For example, a large study by Wei F, et al. reported an inverse correlation between SUA and HbA1c among patients with T2DM [9]. Such apparently conflicting observations may be explained by differences in the severity and duration of diabetes, renal function, insulin resistance, medications, and the degree of glycosuria. At higher plasma glucose concentrations, increased urinary glucose excretion may promote uricosuria, potentially lowering SUA despite worsening hyperglycemia. Therefore, the relationship between SUA and glycemic status may be nonlinear and dependent on the metabolic stage of T2DM. Another important observation was the association between SUA and adiposity. SUA correlated positively with BMI ( $r=0.29$ ,  $p<0.001$ ), and patients with hyperuricemia had a significantly greater BMI than those with normal SUA. BMI  $\geq 25$  kg/m<sup>2</sup> remained independently associated with hyperuricemia (AOR 2.18, 95% CI 1.10–4.33). An Indian study by Singh et al. similarly demonstrated a relationship between obesity and elevated SUA among patients with newly diagnosed diabetes

[10]. Obesity and hyperuricemia may share underlying mechanisms involving insulin resistance, altered renal urate handling, increased purine turnover, and metabolic dysfunction.

A significant relationship between SUA and blood pressure was also observed. SUA correlated positively with systolic and diastolic blood pressure, and hypertension was considerably more frequent among hyperuricemic than normouricemic patients (68.5% vs. 46.8%;  $p=0.008$ ). Hypertension remained independently associated with hyperuricemia (AOR 1.89, 95% CI 1.01–3.55). This finding is supported by an Indian cross-sectional study of newly diagnosed diabetic patients, which demonstrated an association between increasing SUA and hypertension [11]. Possible biological links include endothelial dysfunction, oxidative stress, activation of the renin–angiotensin system, and impaired nitric oxide availability, although the cross-sectional nature of these associations does not establish causality. The association between SUA and lipid abnormalities was particularly prominent. SUA correlated most strongly with triglycerides ( $r=0.36$ ,  $p<0.001$ ), while HDL-C showed an inverse correlation ( $r=-0.28$ ,  $p<0.001$ ). Patients with hyperuricemia had markedly higher triglycerides ( $216.5 \pm 76.8$  vs.  $159.8 \pm 63.7$  mg/dL) and LDL-C and lower HDL-C than patients with normal SUA. These findings are consistent with recent evidence demonstrating an association between hyperuricemia and dyslipidemia in T2DM. In a study involving 1,036 patients, hyperuricemia was independently associated with dyslipidemia and particularly hypertriglyceridemia [12].

The close relationship between SUA and triglycerides may have particular metabolic relevance. In the present study, triglycerides  $\geq 150$  mg/dL showed the strongest independent association with hyperuricemia (AOR 2.63, 95% CI 1.34–5.16;  $p=0.005$ ). Zhao et al. additionally reported that SUA partly mediated the association between insulin resistance and triglyceride metabolism in T2DM, providing a possible mechanistic explanation for the clustering of hyperuricemia and hypertriglyceridemia [12]. Another recent study also reported significantly higher cholesterol, triglyceride, and LDL-C levels

and lower HDL-C levels among T2DM patients with elevated SUA [13].

Importantly, multivariable analysis showed that poor glycemic control (HbA1c  $\geq 7\%$ ), BMI  $\geq 25$  kg/m<sup>2</sup>, hypertension, hypertriglyceridemia, and low HDL-C remained independently associated with hyperuricemia, whereas current smoking was not significant.

Furthermore, multiple linear regression demonstrated an independent association between SUA and HbA1c (standardized  $\beta=0.23$ ,  $p=0.002$ ), with an adjusted  $R^2$  of 0.24 for the model. Collectively, these findings suggest that SUA may serve as an additional marker identifying patients with clustering of adverse cardiometabolic characteristics. However, whether SUA itself contributes causally to these abnormalities or primarily reflects underlying insulin resistance and metabolic dysfunction cannot be established from the present study.

### Conclusion

The present study demonstrates that higher serum uric acid levels are significantly associated with poor glycemic control and an adverse cardiovascular risk profile in patients with type 2 diabetes mellitus. Elevated SUA was associated with higher HbA1c, BMI, blood pressure, triglyceride, and LDL-C levels, along with lower HDL-C levels.

Hypertriglyceridemia showed the strongest independent association with hyperuricemia, while SUA remained independently associated with HbA1c after adjustment for other cardiometabolic factors. These findings suggest that SUA may serve as a simple and readily available additional marker of cardiometabolic risk in patients with T2DM. However, because of the cross-sectional design, a causal relationship cannot be established. Larger prospective studies are required to determine whether SUA independently predicts cardiovascular outcomes and whether modification of uric acid levels provides additional clinical benefit in patients with T2DM.

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