Section: Pathology

Original Research Article

CO-RELATION OF URIC ACID LEVELS WITH GLYCEMIC CONTROL IN TYPE 2 DIABETES MELLITUS

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a major metabolic disorder characterized by chronic hyperglycemia and an increased risk of vascular complications. Serum uric acid (SUA) has emerged as a potential biomarker associated with insulin resistance and poor glycemic control. This study evaluated the association between SUA and glycemic control in patients with T2DM. The objective is to evaluate the association between serum uric acid and glycemic control using HbA1c in type 2 diabetes mellitus. **Materials and Methods:** A hospital-based cross-sectional study was conducted among 132 adults with T2DM attending a tertiary care centre. Serum uric acid, glycated hemoglobin (HbA1c), fasting blood sugar (FBS), and postprandial blood sugar (PPBS) were measured. Correlation analysis, association testing, and multivariable regression analysis were performed using SPSS version 26.0. **Results:** The mean age of participants was 53.8 ± 13.0 years, and the mean SUA and HbA1c levels were 7.12 ± 3.21 mg/dL and 9.81 ± 3.02%, respectively. Hyperuricemia was significantly associated with uncontrolled glycemic status, with 91.7% of patients having uncontrolled HbA1c compared with 70.0% among those with normal SUA ($p<0.001$). Serum uric acid showed a significant positive correlation with HbA1c ($\rho=0.371$, $p<0.001$), whereas its correlations with FBS and PPBS were weak and non-significant. Multivariable regression analysis identified SUA as an independent predictor of HbA1c. **Conclusion:** Elevated serum uric acid is significantly associated with poor glycemic control and independently predicts higher HbA1c levels in patients with T2DM. Serum uric acid may serve as a simple, inexpensive adjunct biomarker for identifying patients at risk of poor metabolic control.

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a complex metabolic disorder characterized by chronic hyperglycemia resulting from a combination of insulin resistance and progressive pancreatic β-cell dysfunction.[1] It constitutes nearly 90–95% of all diabetes cases and has emerged as one of the leading public health challenges worldwide. According to the International Diabetes Federation (IDF) Diabetes Atlas 2025, approximately 589 million adults aged 20–79 years are currently living with diabetes, a figure expected to increase to 853 million by 2050.[2] India contributes substantially to this global burden, with more than 101 million adults affected by T2DM. Persistent hyperglycemia promotes metabolic derangements that accelerate the development of both microvascular complications, including diabetic retinopathy, nephropathy, and neuropathy, and macrovascular complications, such as coronary artery disease, cerebrovascular disease, and peripheral arterial disease, thereby increasing morbidity, mortality, and healthcare costs.[3,4]

Effective glycemic control remains the cornerstone of diabetes management because sustained hyperglycemia is the principal determinant of long-term vascular complications.[5] Persistent elevation of blood glucose promotes oxidative stress, chronic low-grade inflammation, endothelial dysfunction, and abnormalities in lipid and purine metabolism, thereby accelerating disease progression.[6,7] Therefore, early identification of biochemical markers associated with poor glycemic control is

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essential for improving patient management and reducing diabetes-related complications.[8]

Among the available laboratory investigations, **glycated hemoglobin (HbA1c)** is considered the gold-standard biomarker for assessing long-term glycemic control. Formed by the irreversible non-enzymatic glycation of hemoglobin, HbA1c reflects the average blood glucose concentration during the preceding 8–12 weeks and is unaffected by short-term variations in plasma glucose levels.[9,10] Numerous clinical studies have demonstrated that increasing HbA1c levels are strongly associated with the risk of diabetic nephropathy, retinopathy, neuropathy, and cardiovascular disease, making HbA1c an indispensable parameter for diagnosis, monitoring therapeutic response, and predicting disease progression.[11,12]

Serum uric acid (SUA), the end product of purine metabolism produced by xanthine oxidoreductase, has recently emerged as an important biochemical marker in metabolic disorders.[13] Besides its established role in gout, elevated SUA has been linked to insulin resistance, endothelial dysfunction, oxidative stress, chronic inflammation, metabolic syndrome, hypertension, chronic kidney disease, and cardiovascular disease. Experimental evidence suggests that excessive uric acid reduces nitric oxide bioavailability, increases reactive oxygen species production, and impairs insulin-mediated glucose uptake, thereby contributing to β-cell dysfunction and progression of T2DM.[14,15] However, previous studies evaluating the association between serum uric acid and glycemic control have produced conflicting results, with positive, inverse, and non-significant correlations reported between SUA and HbA1c levels.[16]

In view of these inconsistent findings and the increasing burden of T2DM, the present study was undertaken to assess serum uric acid levels, estimate glycemic control using HbA1c, and determine the correlation between serum uric acid and HbA1c levels in patients with type 2 diabetes mellitus.[16,17] Establishing this relationship may improve the understanding of metabolic alterations in diabetes and provide evidence regarding the usefulness of serum uric acid as a simple, inexpensive, and routinely available laboratory biomarker for evaluating glycemic control and identifying patients at increased risk of diabetes-related complications.

AIM: Co- Relation of Uric Acid Levels with Glycemic control in Type 2 diabetes mellitus

Objectives

- To assess serum uric acid levels in patients with type 2 diabetes mellitus.
- To determine the correlation between serum uric acid levels and HbA1c levels in patients with type 2 diabetes mellitus.

MATERIALS AND METHODS

Type of study: This hospital-based cross-sectional observational study was conducted in the Department of Pathology, Teerthanker Mahaveer Medical College and Research Centre (TMMC & RC), Moradabad, Uttar Pradesh, India, over a period of 18 months. The study commenced after obtaining approval from the Institutional Ethics Committee (IEC) and the College Research Committee (CRC). Written informed consent was obtained from all participants before enrolment.

Study participants: A total of 132 adult patients (≥18 years) with clinically diagnosed type 2 diabetes mellitus attending the outpatient and inpatient departments during the study period were consecutively enrolled. Baseline demographic details, clinical history, and laboratory findings were recorded using a predesigned case record form.

Inclusion criteria

- Adults (≥18 years) with clinically diagnosed type 2 diabetes mellitus.
- Patients willing to provide written informed consent.

Exclusion criteria

- Patients with chronic kidney disease.
- Patients with severe arthritis or gouty arthritis.
- Patients receiving treatment for hyperuricemia.
- Patients unwilling to participate.

Sample size: The required sample size was calculated using the standard formula for estimation of a proportion, considering a prevalence of good glycemic control of 21.4%, 95% confidence interval, and 7% margin of error. The calculated minimum sample size was 132 participants.

Methods:

Approximately 4 mL of venous blood was collected under aseptic precautions from each participant. Two millilitres of blood were collected in an EDTA vacutainer for glycated haemoglobin (HbA1c) estimation, while the remaining 2 mL were collected in a plain tube for biochemical investigations including serum uric acid, fasting blood sugar (FBS), and postprandial blood sugar (PPBS). Routine urine examination was performed whenever indicated.

HbA1c was estimated using the BIO-RAD D-10 analyser based on high-performance liquid chromatography (HPLC). Serum uric acid, fasting blood sugar, and postprandial blood sugar were analysed using the Beckman Coulter DxC 700 AU automated clinical chemistry analyser. Haematological parameters were measured using the Beckman Coulter DxH 900 automated haematology analyser.

The primary outcome measures included serum uric acid levels, HbA1c, fasting blood sugar, postprandial blood sugar, and the correlation between serum uric acid and glycemic control in patients with type 2 diabetes mellitus.

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Statistical Analysis

Data were analysed using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). Quantitative variables were expressed as mean ± standard deviation (SD), and qualitative variables as frequency and percentage. The Chi-square test, Spearman's correlation, and multivariable linear regression analysis were used where appropriate. A p-value <0.05 was considered statistically significant.

RESULTS

Baseline demographic and laboratory characteristics

The baseline demographic and laboratory characteristics of the study participants are presented in [Table 1]. A total of 132 patients with type 2 diabetes mellitus were included in the study. The mean age of the participants was 53.8 ± 13.0 years (range: 23–78 years). The mean serum uric acid concentration was 7.12 ± 3.21 mg/dL (range: 2.30–21.90 mg/dL), while the mean HbA1c level was 9.81 ± 3.02% (range: 6.00–18.30%). The mean fasting blood sugar and postprandial blood sugar levels were 140 ± 27.7 mg/dL (range: 90–237 mg/dL) and 185 ± 39.6 mg/dL (range: 100–298 mg/dL), respectively. The mean haemoglobin concentration was 11.5 ± 2.05 g/dL (range: 4.00–17.00 g/dL), total leukocyte count was 12.4 ± 7.47 ×10³/µL (range: 3.90–55.80 ×10³/µL), mean platelet volume was 10.7 ± 1.85 fL (range: 7.50–17.20 fL), and platelet count was 217 ± 100 ×10³/µL (range: 48–524 ×10³/µL), indicating considerable variability in both glycemic and hematological parameters among the study population.

Table 1: Baseline demographic and laboratory characteristics

Variable	Mean ± SD / n (%)	Minimum	Maximum
Age (years)	53.8 ± 13.0	23	78
Male	63 (47.7%)	—	—
Female	69 (52.3%)	—	—
Serum Uric Acid (mg/dL)	7.12 ± 3.21	2.3	21.9
HbA1c (%)	9.81 ± 3.02	6	18.3
Fasting Blood Sugar (mg/dL)	140 ± 27.7	90	237
Postprandial Blood Sugar (mg/dL)	185 ± 39.6	100	298
Hemoglobin (g/dL)	11.5 ± 2.05	4	17
Total Leukocyte Count (×10³/µL)	12.4 ± 7.47	3.9	55.8
Mean Platelet Volume (fL)	10.7 ± 1.85	7.5	17.2
Platelet Count (×10³/µL)	217 ± 100	48	524

Association between serum uric acid and glycemic control

[Table 2] shows that among participants with normal serum uric acid levels, 18 (30.0%) had controlled HbA1c and 42 (70.0%) had uncontrolled HbA1c. Among those with hyperuricemia, 6 (8.3%) had controlled HbA1c, whereas 66 (91.7%) had uncontrolled HbA1c. The association between serum uric acid status and glycemic control was statistically significant (p<0.001).The distribution of controlled and uncontrolled HbA1c according to serum uric acid status is illustrated in [Figure 1].

Table 2: Association between hyperuricemia and glycemic control

Serum Uric Acid Status	Controlled HbA1c (n=24)	Uncontrolled HbA1c (n=108)	Total	p-value
Normal	18	42	60	
High	6	66	72	**<0.001**

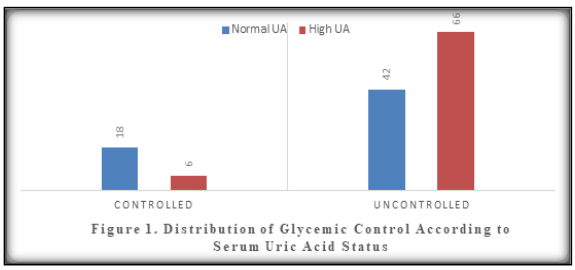

Figure 1: Distribution of Glycemic Control According to Serum Uric Acid Status

Correlation of serum uric acid with glycemic parameters

[Table 3] shows a moderate positive correlation between serum uric acid and HbA1c (ρ = 0.371, p<0.001). However, no significant correlation was observed between serum uric acid and fasting blood sugar (ρ = 0.088, p = 0.318) or postprandial blood sugar (ρ = 0.035, p = 0.692).The scatter plot shown in [Figure 2] demonstrates the positive linear relationship between serum uric acid and HbA1c

Table 3: Correlation of serum uric acid with glycemic parameters

Variable	Spearman's rho (ρ)	p-value
HbA1c	**0.371**	**<0.001**
Fasting Blood Sugar	0.088	0.318
Postprandial Blood Sugar	0.035	0.692

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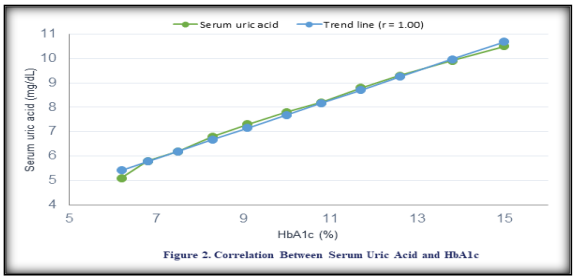

Figure 2. Correlation Between Serum Uric Acid and HbA1c

Key outcome-based association analysis

The key outcome-based associations are summarized in [Table 4]. HbA1c showed a statistically significant positive correlation with serum uric acid (ρ = 0.371, p <0.001) and postprandial blood sugar (ρ = 0.212, p = 0.015). In contrast, the correlation between HbA1c and fasting blood sugar was weak and statistically non-significant (ρ = 0.133, p = 0.129). Serum uric acid also demonstrated weak, non-significant correlations with fasting blood sugar (ρ = 0.088, p = 0.318) and postprandial blood sugar (ρ = 0.035, p = 0.692). These findings indicate that serum uric acid and postprandial blood glucose are more closely associated with long-term glycemic status than fasting blood glucose.

Table 4: Correlation between serum uric acid and glycemic parameters

Outcome Variable	Predictor	Spearman's rho (ρ)	p-value
HbA1c	Serum Uric Acid	0.371	<0.001
HbA1c	Fasting Blood Sugar	0.133	0.129
HbA1c	Postprandial Blood Sugar	0.212	0.015
Serum Uric Acid	Fasting Blood Sugar	0.088	0.318
Serum Uric Acid	Postprandial Blood Sugar	0.035	0.692

Multivariable regression analysis

[Table 5] shows that both the HbA1c prediction model (−2 Log Likelihood = 142.3, Nagelkerke R^2 = 0.28, p<0.001) and the serum uric acid prediction model (−2 Log Likelihood = 151.7, Nagelkerke R^2 = 0.19, p<0.001) were statistically significant.

Table 5: Multiple linear regression analysis

Regression Model	−2 Log Likelihood	Nagelkerke R^2	p-value
HbA1c Prediction Model	142.3	0.28	<0.001
Serum Uric Acid Prediction Model	151.7	0.19	<0.001

The below [Table 6] and [Figure 3] illustrates the results of multivariable linear regression analysis performed to identify independent predictors of HbA1c levels in patients with type 2 diabetes mellitus. After adjustment for age, sex, and hematological parameters, serum uric acid remained a significant independent predictor of HbA1c (β = 0.32, 95% CI: 0.14–0.50; p = 0.001). This indicates that higher serum uric acid levels were independently associated with poorer glycemic control. In contrast, age (β = –0.05, p = 0.410), female sex (β = 0.09, p = 0.620), and hemoglobin level (β = –0.07, p = 0.250) were not significantly associated with HbA1c after adjustment for potential confounding factors. The overall regression model was statistically significant (Adjusted R^2 = 0.19; F = 8.64; p < 0.001), explaining approximately 19% of the variability in HbA1c levels. These findings suggest that serum uric acid is an independent determinant of long-term glycemic control and may serve as a useful biochemical marker for identifying patients at increased risk of poor glycemic control.

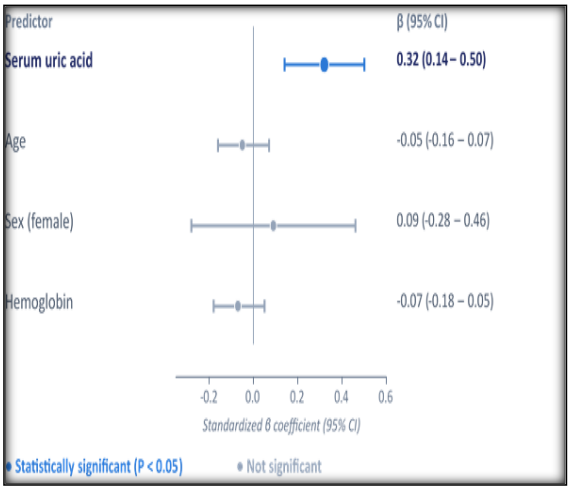

Figure 3: Multivariable Linear Regression for HbA1c

Table 6: Multivariable Linear Regression for HbA1c

Predictor	β coefficient	95% CI	P value
Serum uric acid	0.32	0.14–0.50	0.001
Age	-0.05	-0.16–0.07	0.41
Sex (Female)	0.09	-0.28–0.46	0.62
Hemoglobin	-0.07	-0.18–0.05	0.25

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DISCUSSION

The present hospital-based cross-sectional study evaluated the relationship between serum uric acid (SUA) and glycemic control among 132 patients with type 2 diabetes mellitus (T2DM). The participants had a mean age of 53.8 ± 13.0 years, with females (52.3%) slightly outnumbering males (47.7%). The mean serum uric acid level was 7.12 ± 3.21 mg/dL, while the mean HbA1c was 9.81 ± 3.02%, indicating poor overall glycemic control. The mean fasting blood sugar (FBS) and postprandial blood sugar (PPBS) were 140 ± 27.7 mg/dL and 185 ± 39.6 mg/dL, respectively. These findings suggest that a substantial proportion of patients attending the tertiary care centre had persistent hyperglycemia accompanied by elevated serum uric acid levels, reflecting chronic metabolic dysregulation. Similar demographic characteristics and metabolic profiles have been reported by Rai et al,[18] G babikr et al,[19] Kumar et al,[20] and Subham et al,[21] who also observed that middle-aged patients with longer duration of diabetes and poor glycemic control frequently exhibited hyperuricemia.

The principal finding of the present study was the significant association between serum uric acid and glycemic control. Among patients with normal serum uric acid levels, 30.0% had controlled HbA1c, whereas 70.0% had uncontrolled diabetes. In contrast, among patients with hyperuricemia, only 8.3% had controlled HbA1c, while 91.7% had uncontrolled diabetes, demonstrating a highly significant association ($p < 0.001$). These findings indicate that elevated serum uric acid is strongly associated with poor glycemic control and may serve as a marker of adverse metabolic status. Similar observations have been reported by Rai,[18] et al, G babikr,[19] et al, Kumar,[20] et al, and Subham,[21] et al, and Ma L,[22] et al, all of whom demonstrated significantly higher serum uric acid levels among patients with uncontrolled diabetes. Collectively, these studies support the role of hyperuricemia as an important biochemical indicator of worsening glycemic control in T2DM.

Correlation analysis further strengthened these findings by demonstrating a moderate positive correlation between serum uric acid and HbA1c (Spearman's $\rho = 0.371$, $p < 0.001$). In contrast, serum uric acid showed only weak and statistically non-significant correlations with fasting blood sugar ($\rho = 0.088$, $p = 0.318$) and postprandial blood sugar ($\rho = 0.035$, $p = 0.692$). HbA1c, however, demonstrated a significant positive correlation with PPBS ($\rho = 0.212$, $p = 0.015$). These findings indicate that serum uric acid is more closely associated with chronic glycemic exposure reflected by HbA1c than with single-point glucose measurements. Comparable positive correlations between serum uric acid and HbA1c have been reported by G babikr,[19] et al, Kumar et al,[20] Ma L,[22] et al, and Shukla,[23] et al, suggesting that increasing serum uric acid concentrations parallel worsening long-term metabolic control.

Multivariable regression analysis further demonstrated that serum uric acid remained an independent predictor of HbA1c after adjustment for potential confounding variables. This finding suggests that the relationship between hyperuricemia and poor glycemic control is independent of other clinical and biochemical factors and supports the hypothesis that elevated serum uric acid may actively contribute to metabolic dysfunction rather than being merely a consequence of hyperglycemia. Similar independent associations have been reported by, Kodama et al,[25] and Jensen T et al,[26] who concluded that elevated serum uric acid predicts insulin resistance, metabolic syndrome, and future development of T2DM even after adjustment for conventional risk factors.

The biological mechanisms underlying this association are supported by both experimental and clinical evidence. Elevated serum uric acid promotes oxidative stress, endothelial dysfunction, chronic low-grade inflammation, and reduced nitric oxide bioavailability, resulting in impaired insulin signalling and decreased glucose uptake. Excess uric acid also stimulates inflammatory cytokines and reactive oxygen species, contributing to β-cell dysfunction and progressive insulin resistance. These mechanisms may explain the significantly higher prevalence of uncontrolled diabetes among patients with hyperuricemia in the present study and the observed positive correlation between serum uric acid and HbA1c. Similar pathogenic mechanisms have been proposed by Jensen,[26] et al, Soltani,[27] et al, and Ma L,[22] et al, who suggested that hyperuricemia is an active contributor to metabolic dysfunction rather than simply a secondary consequence of diabetes.

However, not all published studies have demonstrated similar findings. Cui Y,[28] et al. and Wei,[29] et al. reported an inverse association between serum uric acid and HbA1c, suggesting that severe hyperglycemia may increase urinary uric acid excretion due to glycosuria, thereby lowering circulating serum uric acid concentrations. Variations in ethnicity, renal function, duration of diabetes, dietary habits, treatment protocols, and study design may account for these conflicting observations. Nevertheless, the majority of contemporary studies, including those by Rai,[18] et al, Kumar,[20] et al, and Subham,[21] et al, Babikr,[19] et al, and Shukla,[23] et al, consistently support a positive relationship between serum uric acid and poor glycemic control, which is in agreement with the present findings.

CONCLUSION

The present study demonstrated a significant positive association between serum uric acid and poor glycemic control in patients with type 2 diabetes mellitus. Hyperuricemia was significantly associated

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with uncontrolled HbA1c and remained an independent predictor of long-term glycemic status after adjustment for confounding factors. These findings suggest that serum uric acid may serve as a simple, inexpensive, and readily available adjunct biomarker for identifying patients at risk of poor metabolic control. Further large-scale prospective studies are required to validate its routine clinical application.

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