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Original Article

# Serum Uric Acid Levels in Type II Diabetes Mellitus Patients: A Cross-Sectional Study in Kodagu District

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## ABSTRACT

**Background**: Type 2 diabetes mellitus (T2DM) is a major global health problem because of its associated metabolic complications. Elevated Serum uric acid (SUA) levels have been linked to insulin resistance, cardiovascular disease and diabetic nephropathy. However, region-specific data on SUA levels in Indian patients with T2DM remain limited. Our study aimed to evaluate serum uric acid levels in patients with T2DM in the Kodagu district of Karnataka.
**Methods**: A hospital-based cross-sectional study was conducted at Kodagu Institute of Medical Sciences, Madikeri, from December 2022 to March 2025. A total of 256 participants were included in the study. Our study included 128 adults with T2DM and 128 age and sex matched healthy controls. Standardized laboratory methods were used to measure fasting blood glucose, postprandial blood glucose and serum uric acid. The data were analysed using SPSS software.
Results: The patients with T2DM had a significantly higher mean serum uric acid level (8.38 ± 1.40 mg/dL) compared to healthy control group (5.34 ± 1.19 mg/dL) ($p < 0.01$).
**Conclusion**: Serum uric acid levels were significantly higher in patients with T2DM than in healthy controls. This suggests that high serum uric acid may be associated with T2DM and could serve as a useful marker for identifying individuals at increased risk of diabetes-related complications. Further longitudinal studies are needed to understand the role of serum uric acid in the management of T2DM.

***Keywords***: *Type 2 diabetes mellitus; serum uric acid; cross-sectional study.*

## INTRODUCTION

Type 2 diabetes mellitus (T2DM) represents major non-communicable disease associated with worldwide morbidity and mortality. Current epidemiological estimates suggest that approximately 589 million adults were living with diabetes in 2024; however, projections indicate that this number may exceed 850 million by 2050[1]. This burden appears to be disproportionately concentrated in low- and middle-income countries, including India. T2DM is closely linked to metabolic syndrome and confers a markedly elevated risk of cardiovascular disease, chronic kidney disease and premature mortality [2].

Serum uric acid (SUA) is the end product of purine catabolism in humans. It has historically been linked to pathophysiology of gout. However recent evidences suggest that SUA may also be involved in cardiometabolic diseases. Hyperuricemia occurs due to increased uric acid production, reduced renal excretion or both. This can lead to increase cardiovascular risk through mechanisms such as endothelial dysfunction, oxidative stress and systemic inflammation. At the molecular level, elevated SUA may reduce nitric oxide bioavailability, activate the renin–angiotensin–aldosterone system and promote pro-inflammatory signaling cascades. These changes may contribute to metabolic dysregulation and vascular injury [3,4].

Elevated SUA levels may interefere with insulin signaling through mechanisms involving oxidative stress and inflammation. The mechanism may include activation of the NLRP3 inflammasome and stress kinase pathways, which can

lead to peripheral insulin resistance. On the otherhand, high insulin levels can increase renal reabsorption of uric acid, indicating a bidirectional relationship between uric acid metabolism and glucose homeostasis. Epidemiological studies further support an association between higher SUA levels and increased risk of developing T2DM, as well as poorer glycaemic control [5].

Among individuals with established T2DM, elevated SUA has been consistently linked to adverse cardiometabolic outcome and higher risk of complications. A recent meta-analysis has shown a link between SUA levels and diabetic nephropathy, suggesting its potential role as an early biomarker of renal involvement [6]. In addition, observational and genetic studies have explored the association between SUA and cardiovascular outcomes in patients with T2DM, although the causal nature of this association it remains uncertain [7]. Interventional evidence further indicates that sodium–glucose cotransporter-2 inhibitors exert uricosuric effects, reducing SUA levels and supporting the link between glucose and urate homeostasis [8].

Despite these findings, the relationship between SUA and T2DM is not fully understood. Variability in findings may be due to differences in glycaemic status, disease duration, renal function and population chraracteristics. Moreover, most previous studies have been conducted in urban or tertiary care settings, limiting thier applicability to rural populations. Dietary patterns, environmental exposures and genetic background can influence both uric acid metabolism and development of diabetes expression [9,10]. Therefore region-specific investigations are necessary.

Kodagu district in Karnataka has local dietary habits and lifestyle patterns that may influence uric acid levels. In addition, the region also has relatively cooler climate, particularly during the winter and rainy seasons, may affect uric acid metabolism and its deposition. This could increase the risk of renal complications and urate deposition in peripheral joints, thereby influencing the clinical profile of patients with T2DM. Therefore, the present cross-sectional study was designed to evaluate serum uric acid levels among patients with Type 2 Diabetes Mellitus in Kodagu district may provide valuable regional evidence.

## MATERIALS AND METHODS

### 2.1 Study design and setting

This is a cross sectional study conducted at the Kodagu Institute of Medical Sciences, Madikeri, located in the Kodagu district of Karnataka, India. The study was carried out over a 27 month period from December 2022 to March 2025.

### 2.2 Study population

The case group included adults aged 30–70 years with a confirmed diagnosis of T2DM. Participants were enrolled from both outpatient clinics and inpatient medical wards. The control group consisted of healthy, non diabetic adults selected from the general population, including patient attendants and individuals attending routine health screening camps. Controls were matched with cases based on age and sex to reduce potential demographic confounding.

### 2.3 Eligibility criteria

**Inclusion criteria**

- T2DM group: Adults aged 30–70 years with a diagnosis of T2DM based on American Diabetes Association criteria who provided informed consent[11].
- Control group: Age and sex matched individuals without a history of diabetes with fasting plasma glucose <126 mg/dL and random blood glucose <140 mg/dL.

**Exclusion criteria**

Patients with known gout or currently receiving treatment for hyperuricemia, individuals with chronic kidney disease or impaired renal function, patients with chronic liver disease, individuals on medications affecting serum uric acid levels such as diuretics, low-dose aspirin, allopurinol, febuxostat, pyrazinamide were excluded from the study. Patients with acute infections, inflammatory conditions, or recent hospitalization, pregnant or lactating women, individuals with malignancy participants unwilling or unable to provide informed consent were also excluded from the study.

### 2.4 Sample size estimation

Sample size was calculated based on prior studies that assessed serum uric acid levels in individuals with T2DM in the Indian population [12]. Considering a 1:1 case control ratio, a two sided alpha error of 0.05 and a statistical power of 80%, the required minimum sample size was estimated to be 100 participants in each group to identify a clinically significant difference in serum uric acid levels. A total of 128 participants were ultimately included in each group.

### 2.5 Laboratory procedures

After an overnight fast of 10 to 12 hours, 5 mL of venous blood was collected aseptically from the antecubital vein. The blood samples were allowed to clot and then centrifuged at 4000 rpm for 15 minutes to separate the serum. Biochemical tests were carried out in the central laboratory of Kodagu Institute of Medical Sciences using standard laboratory methods with adherence to internal quality control protocols.

### 2.6 Biochemical measurements

Blood glucose was measured using the glucose oxidase–peroxidase (GOD-POD) method and serum uric acid was determined by the uricase–TOOS method using a cobas c311 analyser. Renal function parameters, including serum creatinine and urea, were assessed where indicated to exclude underlying renal dysfunction

### 2.7 Ethical considerations

The study protocol received approval from the Institutional Ethics Committee of Kodagu Institute of Medical Sciences. Written informed consent was obtained from all participants after providing detailed explanation of the study objectives, procedures, potential risks and anticipated benefits in the local language. Strict measures were taken to ensure the participant confidentiality.

### 2.8 Statistical analysis

Statistical analysis was done using SPSS version 23. Continuous variables were presented as mean ± standard deviation. The mean serum uric acid levels of the T2DM and control groups were compared using the independent sample t tests. A p value of less than 0.05 was considered statistically significant.

## RESULTS & DISCUSSION

### 3.1 Results

A total of 256 participants were enrolled in the study, including 128 patients with T2DM and 128 age and sex matched healthy controls. The diabetic group comprised of 90 males and 38 females, while the control group included 86 males and 42 females. The majority of patients with T2DM belonged to the 51–60 year age group (Table 1).

**Table 1. Demographic Characteristics of the Study Population**

| **Age group in years** | **Diabetic Patients (n= 128)** | **Normal Subjects (n= 128)** |
|---|---|---|
| 30 - 40 | 15 | 20 |
| 41 - 50 | 19 | 27 |
| 51 - 60 | 58 | 49 |
| 61 - 70 | 36 | 32 |
| **Gender** | | |
| Male, n (%) | 90 (70.31%) | 86 (67.19%) |
| Female, n (%) | 38 (29.69%) | 42 (32.81%) |

The diabetic group demonstrated a mean fasting blood sugar (FBS) level of 144 mg/dL and a mean postprandial blood sugar (PPBS) level of 238 mg/dL. In comparison, the control group had a mean FBS of 98 mg/dL and mean PPBS of 146 mg/dL (Table 2). The mean serum uric acid concentration in the T2DM group was 8.38 ± 1.40 mg/dL, compared with 5.34 ± 1.19 mg/dL in the control group. This difference was statistically significant ($p < 0.01$), indicating a significantly higher serum uric acid levels among individuals with T2DM in the Kodagu district (Table 3)

**Table 2. Comparison of fasting and postprandial blood glucose levels between T2DM patients and controls**

| | **Diabetic Patients (n= 128)** | **Normal Subjects (n= 128)** |
|---|---|---|
| Mean Fasting blood sugar (mg/dL) ± Standard deviation | 144 ± 15 | 98 ± 14 |
| Mean Postprandial blood sugar (mg/dL) ± Standard deviation | 238 ± 24 | 146 ± 18 |

**Table 3: Comparison of serum uric acid levels between diabetic patients and normal subjects**

| **Group** | **Mean Serum Uric acid Level (mg/dL) ± Standard Deviation** | **p value** |
|---|---|---|
| Diabetic Patients (n= 128) | 8.38 ± 1.40 | < 0.01* |
| Normal Subjects (n= 128) | 5.34 ± 1.19 | |

*Statistically significant at p<0.01

## DISCUSSION

The present cross-sectional study evaluated serum uric acid levels in patients with type 2 diabetes mellitus (T2DM) in the Kodagu district. In our study, we found that patients with T2DM had significant higher serum uric acid levels than healthy individuals. Although uric acid levels are increased in both males and females, significant hyperuricemia is noted in males.

The elevated serum uric acid levels in T2DM patients may be explained by reduced renal uric acid clearance associated with insulin resistance along with increased oxidative stress [13]. The complication of diabetes manifested by nephropathy may further impair uric acid excretion resulting in elevation in serum uric acid levels [14]. In addition, the lower uric acid in females may be attributed to the uricosuric effect of estrogen [15].

The present findings are consistent with those of Yang et al., who, in a case-control study, observed that serum uric acid levels were significantly higher among patients with type 2 diabetes mellitus than among non-diabetic controls [16]. Patel et al. also reported significantly elevated serum uric acid levels in patients with diabetes when compared to healthy controls and demonstrated a positive correlation between serum uric acid and HbA1c, suggesting that elevated serum uric acid level may be associated with poorer glycaemic control [17].

This study was done in Kodagu Institute of Medical Sciences, Madikeri where the food habits and lifestyle of local population may be closely associated with hyperuricemia. Furthermore, Madikeri has relatively cool climatic conditions particularly during winter and rainy season. These climatic conditions may contribute to increased risk of deposition of uric acid in the kidney and peripheral joints, thereby predisposing to renal and articular complications. Further studies are needed to evaluate the effects of food, lifestyle habits and climate on uric acid levels in this population. Since this is a cross-sectional study, it does not establish a causal relationship between increased serum uric acid levels and diabetes-related complications. In addition, potential confounding factors such as dietary intake, medication use and detailed renal function parameters were not comprehensively assessed. Despite these limitations, our study included age and sex matched healthy controls and provides region specific data on serum uric acid levels among patients with T2DM in the kodagu district. Overall our study has demonstrated that hyperuricemia may be associated with diabetes mellitus and may serve as a marker for increased risk of complications.

## CONCLUSION

In conclusion, our study has demonstrated significantly higher serum uric acid levels in patients with type 2 diabetes mellitus than in healthy controls from the Kodagu district. Further large scale prospective studies are required to understand the relationship between serum uric acid levels and development of complications in diabetes mellitus, particularly renal impairment. Future studies assessing serum uric acid levels alongside glycemic control and treatment outcomes may help determine its clinical utility in the management and prognostic evaluation of patients with T2DM.

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**Conflict of interest**

We wish to confirm that there are no known conflicts of interest associated with this publication and there has been no significant financial support for this work that could have influenced its outcome.

**Funding information**

This research received no external funding.

**Ethical information**

The study is approved by Institutional ethics committee of Kodagu Institute of Medical Sciences, Madikeri. Written informed consent was obtained from all study participants.

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