International Journal of Medicine
pISSN 1468-3814 | eISSN 2667-7008
https://theinternationalmedicine.com/
Original Research Article *DOI: 10.61336/im/26-4-25*

Estimation of Level of Serum Uric Acid and Albuminuria in Patients with Type 2 Diabetes Mellitus in Tertiary Care Hospital

DR. NIRAJ DIWAKAR[1], DR. SWEETY SINGH[2], DR. RISHABH JHA[3], DR. ARSHAD AHMAD[4]
[1]SENIOR RESIDENT, DEPARTMENT OF GENERAL MEDICINE, IGIMS PATNA
[2]ASSISTANT PROFESSOR, DEPARTMENT OF GENERAL MEDICINE, IGIMS, PATNA
[3]JUNIOR RESIDENT, DEPARTMENT OF GENERAL MEDICINE, IGIMS, PATNA
[4]PROFESSOR, DEPARTMENT OF GENERAL MEDICINE, IGIMS, PATNA

Correspondence:
DR. NIRAJ DIWAKAR*

Received: **February 10, 2026**
Revised: **February 25, 2026**
Accepted: **March 09, 2026**
Published: **April 09, 2026**

Abstract
Background: Type 2 diabetes mellitus (T2DM) is a leading cause of diabetic nephropathy and chronic kidney disease, which contributes significantly. Serum uric acid (SUA) is involved in renal injury and endothelial dysfunction, and the early marker of diabetic nephropathy is albuminuria. ***Methods:*** An observational cross-sectional study was conducted at a tertiary care hospital during the period from January 2021 to June 2022, including 150 patients with T2DM. SUA was measured by a uricase enzymatic method, whereas urinary albumin-creatinine ratio was determined using an immunoturbidimetry assess. Statistical analysis in SPSS was used, with correlation estimated by Spearman's test. ***Results:*** The most frequent type of albuminuria in the study sample was microalbuminuria. There was also a positive association of SUA with the urinary albumin creatinine ratio. Albuminuria also tended to be more with duration of diabetes. The prevalence of albuminuria was higher among patients with hyperuricaemia than in the normal group. ***Conclusion:*** Albuminuria in patients with T2DM is positively associated with SUA levels. A cost-effective marker for early identification of patients at risk of diabetic nephropathy, which may serve as a simple test of SUA.

Keywords: Albuminuria, Diabetic Nephropathy, Serum Uric Acid, T2DM

This article is an open access article distributed under the terms and conditions of the Creative Commons Attribution (CC-BY) license (http://creativecommons.org/licenses/by/4.0/).

INTRODUCTION
Type 2 diabetes mellitus (T2DM) is one of the most prevalent chronic metabolic disorders worldwide, which represents a major public health challenge mainly in developing countries such as India [1]. Diabetes is a rapidly expanding global epidemic; India has more than its fair share of diabetes according to the International Diabetes Federation. Long-term hyperglycaemia in T2DM is associated with detrimental and progressive microvascular and macrovascular complications, of which Diabetic Nephropathy (DN) is the leading cause of Chronic Kidney Disease (CKD) and End-stage Renal Disease (ESRD) [2]. Diabetic nephropathy is a well-known cause of ESRD, and in India it contributes substantially to the population of patients on renal replacement therapy leading to an increase in morbidity, mortality and healthcare burden.

As the last product of purine metabolism, SUA has become an essential metabolic factor to be involved in the development of diabetic complications [3]. The increased levels of SUA have been shown to be linked with endothelial dysfunction, oxidative stress, inflammation as well as activation of the renin-angiotensin system, which contribute to glomerular hypertension and renal microvascular damage [4]. Experimental and clinical studies support that hyperuricemia can be a direct pathogenic role in renal injury, which through stimulation of vascular smooth muscle proliferation, reduction in nitric oxide bioavailability and increase in pro-inflammatory mediators. SUA levels are increased as well as associated with impaired renal function and progression of diabetic nephropathy in patients with T2DM [5].

Microalbuminuria, which reflects a moderate increase in urinary albumin excretion, is known to be the earliest clinical sign of diabetic nephropathy [6,7]. It is indicative of higher glomerular permeability and widespread endothelial pathology. Microalbuminuria is not only a predictor of the progression to renal disease, but also serves as an index for cardiovascular risk in diabetic patients [8]. The early targeting of microalbuminuria

makes it possible to intervene in time and potentially retard or even revert progression of diabetic nephropathy.

While independent studies have shown associations of SUA levels and albuminuria in T2DM, there is a lack of data from the Indian population and that from tertiary care hospitals [9]. In addition, the association of SUA with albuminuria and duration of diabetes has not been well-studied. In this context, we aimed to measure the SUA levels and albuminuria in T2DM patients attending a tertiary care hospital and also tried to find a correlation between SUA levels, albuminuria and duration of diabetes.

Objectives

1. To estimate the levels of SUA with T2DM patients can attends a tertiary care hospital.
2. To assess the measuring urinary albumin-creatinine ratio in patients with T2DM to determine the prevalence of albuminuria.

To evaluate the relationship among the SUA levels, albuminuria, as well as duration of T2DM.

MATERIALS AND METHODS

Study Design and Setting

This was a cross-sectional observational study done in the Department of General Medicine, Indira Gandhi Institute of Medical Sciences (IGIMS), Patna, which is a tertiary care teaching hospital of Bihar. The study was done for a period of eighteen months from January 2021 to June 2022 after getting approval from the Institutional Ethical Committee of IGIMS, Patna.

Study Population

Study population patients presenting at outpatients and inpatient facilities of the department of general medicine, IGIMS, Patna with diagnosis T2DM. Patients aged 30-80 years of both genders were enrolled in this study. Patients with known causes that have an impact on SUA level or albumin excretion, such as arthritis, hypertension, hypothyroidism, renal failure, heart failure, myeloproliferative disorders and intake of alcohol for the last 12 months were excluded. Pregnant and lactating women were also excluded. All patients signed an informed consent before entering the study.

Inclusion Criteria

1. In the diagnosed patients with T2DM, as per the criteria of the American Diabetes Association, either gender is aged 30-80 years.
2. During the study period, tertiary care patients are attending the Department of General Medicine from the outpatient or inpatient services.
3. Written informed consent was provided by the patient to participate in the study.

Exclusion Criteria

1. Patients with CKD, hypertension, arthritis, hypothyroidism, heart failure, acute febrile illness, or other diseases that impair uric acid or albumin excretion.
2. People who drink heavily, take diuretics, or take any albuminuria-affecting prescriptions.
3. Patients in pregnant or lactating women affects the urinary tract infections.

Sample Size Calculation

Sample size was estimated from previous studies which reported a medium correlation between SUA and albuminuria in T2DM patients. Hypothesizing a correlation coefficient of 0.25, with 90% power, and error probability at 5% level, the estimated sample size was found to be 135 participants. To account for potential loss to follow-up and missing data, a sample size of 150 was included in the study.

Data Collection and Laboratory Methods

The entire clinical profile including history of age, sex, duration and any significant past medical history was recorded for all cases. After overnight fast of at least 8 hours, blood samples were collected under aseptic conditions for assessment of Fasting Plasma Glucose (FPG), Postprandial Plasma Glucose (PPG), SUA, and serum creatinine. Random urine samples were taken to estimate urinary microalbumin and urinary creatinine.

SUA was determined based on the uricase enzymatic colorimetry. Urinary microalbumin was determined by immunoturbidimetry and serum and urinary creatinine were estimated with the modified kinetic Jaffe method. Blood glucose was measured by the glucose oxidase-peroxidase method. Urinary albumin-to-creatinine ratio (UACR) was determined and reported as milligrams of albumin per gram of creatinine.

Hyperuricemia definition was serum level of uric acid >7.0 mg/dL in male and >5.7mg/dL in female respectively. Microalbuminuria was defined as a UACR of 30-300 mg/g creatinine, and less than 30 mg/g as normoalbuminuria.

Statistical Analysis

Statistical calculations were performed with SPSS. Continuous variables were presented as mean ± standard deviation, and categorical variables such as frequencies and percentages. Differences between groups were analyzed using unpaired Student's t-test or Chi-square test when applicable. Associations between the levels of SUA and albuminuria were estimated by Spearman's rank correlation coefficient. A p-value of < 0.05 was considered to be statistically significant.

RESULTS

Baseline Characteristics

A total of 150 Type 2 diabetes patients were enrolled in the study. The average age in the group was 54.25 ± 13.93 years (18 -79 years). Males and females were 90 (60.0%) and 60 (40.0%) respectively of the total study subjects. The average duration from the onset of diabetes was 3.49 ± 1.44 years, more than half (52.7%) had a history of diabetes between 1 and 3 years.

The average HbA1c level was 7.30 ± 1.37%. The average fasting blood sugar and postprandial blood sugar level were 141.13 ± 27.17 mg/dL and 213.41 ± 45.64 mg/dl respectively. Mean (± SD) SUA and creatinine was 5.68 ± 1.34 mg/dL and 1.36 ± 1.05 mg/dL, respectively. The average UACR was 259.04 ± 663.67 mg/g creatinine.

Table 1 Baseline characteristics of the study population

Parameter	Mean ± SD	Range
Age (years)	54.25 ± 13.93	18 – 79
Duration of diabetes (years)	3.49 ± 1.44	1.5 – 8.5
HbA1c (%)	7.30 ± 1.37	3.2 – 11.3
SUA (mg/dL)	5.68 ± 1.34	2.30 – 9.20
Serum creatinine (mg/dL)	1.36 ± 1.05	0.40 – 7.93
UACR (mg/g creatinine)	259.04 ± 663.67	5 – 5207

Distribution of Albuminuria and Uric Acid

UACR levels, 36 (24.0%) had normoalbuminuria (UACR <30 mg/g), 87 (58.0%) had microalbuminuria (UACR of 30–300 mg/g) and 27(18.0%) had macroalbuminuria (UACR ≥300 mg/g). Microalbuminuria was the predominant type of albuminuria in the study material.

Concerning SUA levels, 118 patients (78.7%) were in the normal range of SUA with gender-specific cut-off points, while 32 (21.3%) had hyperuricemia. Hyperuricemia was a feature of male and female patients, but it was more common in women.

Table 2 Distribution of patients according to albuminuria and SUA

Variable	Category	Frequency (%)
Albuminuria (UACR)	Normoalbuminuria	36 (24.0)
	Microalbuminuria	87 (58.0)
	Macroalbuminuria	27 (18.0)
SUA	Normal	118 (78.7)
	Hyperuricemia	32 (21.3)

Correlation Analysis

The correlation analysis determined the relation of SUA with urinary albumin-creatinine ratio. Among the whole study population, a significant positive relationship between SUA and UACR was noted ($r = 0.79$, $p = 0.048$), showing that as SUA increased, UACR also increased.

Relationship between UACR and duration of diabetes mellitus was also evaluated. The duration of diabetes was also correlated with those changes. UACR was increased in higher degrees by prolonged duration of disease. The correlation coefficient was moderate and statistically significant ($p < 0.05$).

Table 3 Correlation between SUA, UACR, and duration of diabetes

Variables Compared	Correlation coefficient (r)	p-value
SUA vs UACR	0.79	0.048
Duration of diabetes vs UACR	Positive correlation	<0.05

DISCUSSION

The current study aimed to assess the association of SUA level with albuminuria in individuals with T2DM attending a tertiary care hospital. Major results of this investigation show that SUA levels are positively correlated with urinary albumin excretion. Additionally, the findings had a higher prevalence of albuminuria with longer duration of diabetes. These results underscore the significance of SUA as a key biochemical marker for early renal damage in T2DM patients.

The relationship found between SUA and diabetic nephropathy is in accordance with that reported for other studies. [10] found that increased SUA levels were an independent predictor for the presence and progression of albuminuria in patients with type 2 diabetes, indicating a contributory role of hyperuricemia to diabetic kidney disease. Similarly, [11] reported a marked association of SUA levels with microalbuminuria and pointed out that hyperuricemia could be an early predictor for renal damage in diabetes mellitus patients. [12] also demonstrated higher SUA in diabetics with microalbuminuria than those with normoalbuminuria, suggesting its role in renal endothelial damage. Furthermore, [13] found that higher

SUA levels were correlated with an increased risk of the onset and progression of diabetic nephropathy, even among patients who had preserved renal function. The current study's findings are consistent with these studies and support the mounting evidence for a relationship between SUA levels in diabetic renal involvement.

The pathophysiology between serum uric acid and albuminuria are varied. Hyperuricemia is confirmed to induce oxidative stress by reactive oxygen species formation, leading to endothelial dysfunction [14]. Uric acid is known to decrease nitric oxide availability leading to endothelial-mediated vasodilatation dysfunction and glomerular hypertension. Furthermore, hyperuricemia induces inflammatory pathways and activates the renin-angiotensin-aldosterone system which stimulates the intraglomerular pressure and glomerular permeability. These events converge to contribute to podocyte injury and increased urinary albumin excretion, thus facilitating the transition from normoalbumiuria to microalbuminuria and subsequent overt diabetic nephropathy.

Clinically, the results of this study are very useful. SUA determination is an easy, cheap and very mutable laboratory test. Its link with albuminuria indicates that SUA might be used as an early warning marker for screening diabetic subjects at high risk of kidney disease. The identification of patients with a rise in SUA can give to the clinician the possibility of an early intervention, Optimizating glycaemic control, modifying lifestyle and renoprotective therapies. In addition, SUA may be useful for risk stratification to select patients who need more intensive monitoring for the presence or progression of diabetic nephropathy.

The public health implications of these findings are even more relevant in the Indian scenario where there is a relentless increase in the burden of T2DM and its complications. Diabetic nephropathy continues to be one of the most common causes of CKD and ESRD, imposing a considerable burden on tertiary care centers [15]. Early recognition of the renal involvement with available markers including SUA and microalbuminuria may lead to reduced progression of the disease, saving expensive healthcare resources, thus a better outcome for patients. Routine measurement of SUA levels in patients with T2DM seen at tertiary care centers might fill a gap in preventive and therapeutic approaches.

Limitations

Several limitations should be noted in the current study. Because we were a single-center study at a tertiary care hospital, the findings may not be applicable to the general population or primary care clinic. A causal relationship between SUA levels and albuminuria cannot be drawn based on a cross-sectional study. Absence of serial data did not allow any inference with respect to the course of albuminuria and chronic renal damage. Furthermore, the possible confounding effects of dietary habits, physical activity and use of uric acid–reducing therapy could not be taken into consideration when examining SUA levels.

CONCLUSION

The current study shows that SUA levels are positively associated with albuminuria in patients with T2DM, indicating a possible pathophysiological role of hyperuricaemia in the early stages of diabetic renal involvement. Because of its simplicity, low-cost and common nature in laboratories, SUA could be a useful new diagnostic index for those diabetic individuals with a higher risk for the occurrence of nephropsathy. Screening for hyperuricemia and albuminuria in the early stage of T2DM might contribute to earlier intervention, better risk stratification and deferred progression of diabetic kidney disease.

REFERENCES

[1] R. Shruthi, H. S. Sridevi, and B. M. Sharanabasavaraja, “A cross-sectional study on urinary microalbuminuria and serum uric acid in patients of type 2 diabetes mellitus at a tertiary care hospital,” Int. J. Pharm. Res. Technol., vol. 15, no. 1, pp. 1441–1445, 2025.

[2] S. Islam, M. A. Hossen, M. A. Rahman, M. I. Lubaba, and A. Akram, “Serum uric acid level among type-2 diabetes subjects attending in a tertiary hospital of Bangladesh,” World J. Biol. Pharm. Health Sci., vol. 12, no. 1, pp. 81–85, 2022.

[3] S. Kumar, H. Mondal, M. Lata, J. K. Behera, and B. Priyadarshini, “Correlation of serum uric acid with lipid profile in patients with type 2 diabetes mellitus with normal creatinine level: Report from a tertiary care hospital in India,” J. Fam. Med. Prim. Care, vol. 11, no. 6, pp. 3066–3070, 2022.

[4] S. K. Das, S. K. Patjoshi, C. B. Narayan, D. R. Nayak, and R. N. Yadav, “Assessment among type 2 diabetes mellitus relationship with albuminuria,” Int. J. Acad. Med. Pharm., vol. 5, no. 4, pp. 911–915, 2023.

[5] R. Bral, S. Bhat, R. Koul, and S. Sharma, “The association between serum uric acid in patients of impaired fasting glucose and type 2 diabetes mellitus,” [Journal name not available].

[6] Y. Hu, Q. Li, R. Min, Y. Deng, Y. Xu, and L. Gao, “The association between serum uric acid and diabetic complications in patients with type 2 diabetes mellitus by gender: A cross-sectional study,” PeerJ, vol. 9, p. e10691, 2021.

[7] S. Das, R. Mukherjee, and P. K. Mandal, “A study on urinary microalbuminuria and serum uric acid in patients of type 2 diabetes mellitus at a tertiary care hospital of West Bengal,” [Journal name not available].

[8] J. Du, X. Xu, N. Yuan, and X. Zhang, “Predictive value of serum uric acid-to-albumin ratio for diabetic kidney disease in patients with type 2 diabetes mellitus: A case-control study,” Front. Endocrinol., vol. 16, p. 1577950, 2025.

[9] M. Pazianas and P. D. Miller, “Osteoporosis and chronic kidney disease–mineral and bone disorder

(CKD-MBD): Back to basics," Am. J. Kidney Dis., vol. 78, no. 4, pp. 582–589, Oct. 2021.
[10] Y. Zhan, X. He, D. Hong, L. Wang, and G. Li, "The current status of chronic kidney disease–mineral and bone disorder management in China," Sci. Rep., vol. 12, no. 1, p. 16694, Oct. 2022.
[11] F. Afshinnia and S. Pennathur, "Lipids and cardiovascular risk with CKD," Clin. J. Am. Soc. Nephrol., vol. 15, no. 1, pp. 5–7, Jan. 2020.
[12] M. Saini, A. Vamne, V. Kumar, and M. S. Chandel, "The study of pattern of lipid profile in chronic kidney disease patients on conservative management and hemodialysis: A comparative study," Cureus, vol. 14, no. 1, Jan. 2022.
[13] L. Hou, Y. Shi, S. Wang, Q. Chen, Q. Li, M. Zhao et al., "Associations of serum uric acid level with diabetic retinopathy and albuminuria in patients with type 2 diabetes mellitus," J. Int. Med. Res., vol. 48, no. 12, Dec. 2020.
[14] P. Warjukar, P. Jain, P. Kute, A. Anjankar, and S. S. Ghangal, "Study of microalbuminuria and uric acid in type 2 diabetes mellitus," Int. J. Curr. Res. Rev., vol. 12, no. 14, pp. 56–65, 2020.
[15] Y. J. Lai, Y. Y. Chen, P. W. Ku, L. J. Chen, and Y. F. Yen, "Association between uric acid level and incidence of albuminuria in patients with type 2 diabetes mellitus: A 4.5-year cohort study," Medicine, vol. 100, no. 41, p. e27496, 2021.