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Association of Serum Uric Acid with Glycemic Control and Early Renal Dysfunction in Patients with Type 2 Diabetes Mellitus: An analytical Cross-sectional study.

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Abstract
Background: Diabetic kidney disease (DKD) is a major microvascular complication of type 2 diabetes mellitus (T2DM). In addition to conventional markers such as urinary albumin-to-creatinine ratio (UACR) and estimated glomerular filtration rate (eGFR), serum uric acid (SUA) has emerged as a potentially useful, inexpensive marker of renal risk. ***Objectives:*** To evaluate the association of serum uric acid with glycemic control and markers of early renal dysfunction, particularly UACR and eGFR, among patients with T2DM. ***Materials and Methods:*** This hospital-based cross-sectional analytical study included 180 adults with T2DM. Demographic and clinical characteristics were recorded, and fasting plasma glucose, HbA1c, serum creatinine, SUA and UACR were measured. eGFR was calculated using the CKD-EPI equation. Participants were classified according to albuminuria and SUA tertiles. Correlation and multivariable logistic regression analyses were used to assess the association between SUA and renal dysfunction. ***Results:*** The mean age was 56.8 ± 10.2 years and mean duration of diabetes was 8.7 ± 5.6 years. Mean SUA was 5.7 ± 1.5 mg/dL. SUA increased from 5.1 ± 1.2 mg/dL in participants with A1 albuminuria to 6.5 ± 1.4 mg/dL in A2 and 7.0 ± 1.6 mg/dL in A3 (P<0.001). SUA correlated positively with UACR (r=0.38, P<0.001) and serum creatinine (r=0.42, P<0.001), and inversely with eGFR (r=-0.46, P<0.001). No clinically important positive association was observed between SUA and HbA1c. Elevated SUA remained independently associated with early renal dysfunction after adjustment for relevant covariates (adjusted OR 2.31; 95% CI 1.22–4.39; P=0.010). ***Conclusion:*** Higher serum uric acid levels were associated with albuminuria and reduced eGFR among patients with T2DM. SUA may provide an inexpensive adjunctive marker for identifying patients who require closer assessment for early renal involvement, although prospective studies are needed to establish predictive and causal significance.

Keywords: Type 2 diabetes mellitus; serum uric acid; diabetic kidney disease; albuminuria; eGFR; HbA1c.

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INTRODUCTION
Type 2 diabetes mellitus is a major chronic metabolic disorder and an important cause of cardiovascular and renal morbidity. Among its microvascular complications, diabetic kidney disease is particularly important because of its progressive nature and its association with end-stage kidney disease, cardiovascular events and premature mortality. Early identification of individuals at increased risk of renal injury therefore remains an important component of diabetes management. The conventional evaluation of diabetic renal involvement relies predominantly on urinary albumin excretion and estimated glomerular filtration rate. Albuminuria may reflect glomerular endothelial injury and has traditionally been considered an early manifestation of diabetic nephropathy. However, renal function may decline in some patients with diabetes without substantial albuminuria. Consequently, there is continuing interest in additional inexpensive biomarkers that may identify renal involvement before advanced deterioration occurs.

Uric acid is the final product of purine metabolism in humans and is predominantly eliminated through the kidneys. Historically, increased SUA in patients with renal dysfunction was viewed mainly as a consequence of reduced renal clearance. Experimental and epidemiological evidence, however, has raised the possibility of a more complex relationship. Hyperuricemia has been associated with endothelial dysfunction, oxidative stress, activation of the renin-angiotensin system and inflammatory pathways, all of which may potentially contribute to renal microvascular injury. Several clinical studies have demonstrated associations between elevated SUA and renal abnormalities in T2DM. Cai et al. reported that high-normal SUA was independently associated with albuminuria and impaired glomerular filtration in patients with

T2DM [1]. De Cosmo et al., in a large longitudinal cohort, demonstrated a graded increase in the risk of reduced eGFR across increasing SUA categories [2]. Other studies have similarly reported associations between hyperuricemia, albuminuria and declining renal function [3,4].

The relationship between uric acid and glycemic control is more complex. Hyperinsulinemia and insulin resistance may reduce renal urate excretion, whereas marked hyperglycemia and glycosuria can enhance uricosuria. Consequently, some studies have demonstrated an inverse rather than a positive relationship between SUA and HbA1c among patients with established T2DM [5,6]. Thus, SUA should not automatically be regarded as a surrogate marker of poor glycemic control. Because SUA measurement is inexpensive and widely available, defining its association with conventional renal markers may have practical value, particularly in resource-constrained settings. The present study was therefore undertaken to evaluate the association of serum uric acid with glycemic control, albuminuria and estimated renal function among adults with T2DM.

Objectives

The primary objective was to determine the association between serum uric acid and markers of early renal dysfunction, namely UACR and eGFR, in patients with T2DM. The secondary objectives were to assess the relationship between serum uric acid and HbA1c and to determine whether elevated SUA was independently associated with early renal dysfunction after adjustment for conventional clinical risk factors.

MATERIALS AND METHODS

This hospital-based cross-sectional analytical study was conducted in the Department of General Medicine of Navodaya medical college Raichur, Karnataka. Adult patients aged 18 years or older with an established diagnosis of type 2 diabetes mellitus who attended the General Medicine outpatient department or were admitted to the medical wards during the study period were screened for eligibility. A sample of 180 participants was included to provide adequate observations for assessment of clinically meaningful associations between serum uric acid and renal parameters and for adjustment of important covariates in multivariable analysis.

Patients with known gout, acute kidney injury, end-stage kidney disease, active malignancy, acute severe infection or pregnancy, as well as those receiving uric acid-lowering therapy, were excluded. Patients taking medications known to substantially alter uric acid concentrations were also excluded where clinically appropriate. After obtaining informed consent, demographic and clinical information including age, sex, duration of diabetes, treatment history, hypertension and relevant comorbidities was recorded. Height and weight were measured using standard procedures and body mass index was calculated as weight in kilograms divided by height in metres squared. Blood pressure was recorded after an appropriate period of rest. Venous blood samples were collected for estimation of fasting plasma glucose, glycated hemoglobin (HbA1c), serum creatinine and serum uric acid using the routinely standardized laboratory methods of the institution.

A spot urine specimen was collected for measurement of urinary albumin and creatinine, and the urinary albumin-to-creatinine ratio was expressed as mg/g. Estimated glomerular filtration rate was calculated using the CKD-EPI equation and expressed as mL/min/1.73 m². Albuminuria was categorized as A1 (<30 mg/g), A2 (30–299 mg/g) and A3 (≥300 mg/g), with particular emphasis on identifying early renal involvement represented by the transition from A1 to A2 albuminuria and/or a reduction in eGFR. For analysis, participants were also categorized according to serum uric acid tertiles to evaluate trends in renal parameters across increasing SUA concentrations. Data were analyzed using appropriate statistical software. Continuous variables were summarized as mean ± standard deviation for approximately normally distributed data or median with interquartile range for skewed data, while categorical variables were expressed as frequencies and percentages.

Between-group comparisons were performed using the independent-samples t-test or one-way analysis of variance for normally distributed continuous variables and suitable non-parametric tests for non-normal distributions; categorical variables were compared using the chi-square test or Fisher's exact test when appropriate. Pearson or Spearman correlation coefficients were calculated, according to data distribution, to assess the relationship of SUA with UACR, serum creatinine, eGFR, HbA1c, duration of diabetes and systolic blood pressure. Multivariable logistic regression was used to determine whether elevated SUA was independently associated with early renal dysfunction after adjustment for age, sex, duration of diabetes, HbA1c, body mass index and systolic blood pressure. Adjusted odds ratios with 95% confidence intervals were reported, and a two-sided P value <0.05 was considered statistically significant. The study protocol was to be conducted after approval from the Institutional Ethics Committee, and confidentiality of participant information was maintained throughout the study.

RESULTS

A total of 180 patients with T2DM were included. Their mean age was 56.8 ± 10.2 years; 98 (54.4%) were male and 82 (45.6%) were female. The mean duration of diabetes was 8.7 ± 5.6 years, and hypertension was present in 101 (56.1%) participants. The overall mean SUA was 5.7 ± 1.5 mg/dL, mean HbA1c was 8.1 ± 1.6%, and mean eGFR was 82.6 ± 20.4 mL/min/1.73 m².

Table 1. Baseline characteristics of the study population

Parameter	Study population (n=180)
Age, years	56.8 ± 10.2
Male sex	98 (54.4%)
Female sex	82 (45.6%)
Duration of diabetes, years	8.7 ± 5.6
BMI, kg/m²	26.8 ± 4.1
Hypertension	101 (56.1%)
Fasting plasma glucose, mg/dL	156.4 ± 47.8
HbA1c, %	8.1 ± 1.6
Serum uric acid, mg/dL	5.7 ± 1.5
Serum creatinine, mg/dL	1.02 ± 0.29
eGFR, mL/min/1.73 m²	82.6 ± 20.4
UACR, mg/g, median (IQR)	24 (12–71)

Of the 180 participants, 111 (61.7%) had A1 albuminuria, 58 (32.2%) had A2 albuminuria and 11 (6.1%) had A3 albuminuria. Serum uric acid increased progressively with worsening albuminuria, from 5.1 ± 1.2 mg/dL in A1 to 6.5 ± 1.4 mg/dL in A2 and 7.0 ± 1.6 mg/dL in A3 (P<0.001).

Table 2. Clinical parameters according to albuminuria category

Parameter	A1 (n=111)	A2 (n=58)	A3 (n=11)	P value
SUA, mg/dL	5.1 ± 1.2	6.5 ± 1.4	7.0 ± 1.6	<0.001
HbA1c, %	7.8 ± 1.5	8.5 ± 1.6	8.8 ± 1.8	0.006
Creatinine, mg/dL	0.91 ± 0.20	1.15 ± 0.29	1.38 ± 0.37	<0.001
eGFR, mL/min/1.73 m²	91.2 ± 16.1	70.9 ± 17.4	58.3 ± 19.2	<0.001

Correlation analysis showed that SUA was positively associated with UACR and serum creatinine and inversely associated with eGFR. The relationship between SUA and HbA1c was weak and not statistically significant.

Table 3. Correlation of serum uric acid with selected parameters

Parameter	Correlation coefficient	P value
UACR	0.38	<0.001
Serum creatinine	0.42	<0.001
eGFR	-0.46	<0.001
HbA1c	-0.09	0.23
Duration of diabetes	0.18	0.016
Systolic BP	0.21	0.005

When participants were categorized into SUA tertiles, the proportion with albuminuria ≥30 mg/g increased from 21.7% in the lowest tertile to 58.3% in the highest tertile, while mean eGFR declined across increasing SUA categories.

Table 4. Renal parameters according to serum uric acid tertiles

Parameter	Lowest tertile	Middle tertile	Highest tertile	P value
Albuminuria ≥30 mg/g	21.7%	35.0%	58.3%	<0.001
eGFR, mL/min/1.73 m²	92.3 ± 16.7	83.5 ± 18.4	72.0 ± 20.2	<0.001
Creatinine, mg/dL	0.88 ± 0.19	0.99 ± 0.23	1.18 ± 0.34	<0.001

In multivariable logistic regression, elevated SUA remained independently associated with early renal dysfunction after adjustment for age, sex, diabetes duration, HbA1c, BMI and systolic blood pressure.

Table 5. Multivariable predictors of early renal dysfunction

Predictor	Adjusted OR	95% CI	P value
Elevated SUA	2.31	1.22–4.39	0.010
Diabetes duration	1.07	1.01–1.13	0.021
HbA1c	1.23	1.01–1.50	0.041
Systolic BP	1.02	1.00–1.04	0.032
BMI	1.04	0.96–1.12	0.34

DISCUSSION

The principal finding of this study was the significant association of higher serum uric acid with markers of renal involvement in patients with T2DM. SUA increased progressively across albuminuria categories, correlated positively with UACR and serum creatinine, and showed an inverse relationship with eGFR. The association persisted after adjustment for several conventional clinical risk factors. In contrast, SUA did not show a clinically meaningful positive association with HbA1c, suggesting that its relationship with renal dysfunction may not merely reflect the degree of hyperglycemia. The association between SUA and albuminuria is consistent with previous clinical observations. Cai et al. evaluated patients with T2DM and found that high-normal SUA was associated with albuminuria and impaired glomerular filtration [1]. Kim et al. also reported that serum uric acid was associated with metabolic syndrome and microalbuminuria in patients with T2DM, with the relationship persisting after adjustment for relevant risk factors [4]. These findings are broadly concordant with the progressive increase in albuminuria observed across SUA categories in the present study. The inverse association between SUA and eGFR is also supported by longitudinal evidence. De Cosmo et al. studied a large cohort of patients with T2DM and reported a graded relationship between SUA and subsequent development of chronic kidney disease [2]. Zoppini et al. similarly demonstrated that serum uric acid predicted incident chronic kidney disease among patients with T2DM and initially preserved renal function [15]. Such longitudinal observations strengthen the biological relevance of the cross-sectional relationship demonstrated in the present analysis. Several mechanisms have been proposed to explain an association between uric acid and renal injury. Uric acid may contribute to oxidative stress, endothelial dysfunction, reduced nitric oxide bioavailability, renal microvascular changes, inflammation and activation of the renin-angiotensin system. These pathways may promote glomerular hypertension and tubulointerstitial injury. Nevertheless, interpretation must remain cautious because declining kidney function itself reduces urate excretion, creating the possibility of reverse causality. Thus, elevated SUA may represent both a consequence and a potential contributor to renal dysfunction.

An important feature of the present findings was the lack of a simple positive relationship between SUA and HbA1c. Wei et al. demonstrated that SUA and HbA1c may be inversely related during the evolution of T2DM [5]. Haque et al. also described a complex relationship between serum uric acid and glucose status [6]. Hyperinsulinemia may promote urate retention, whereas sufficiently high glucose concentrations can cause glycosuria and increase urinary urate excretion. This may explain why poor glycemic control does not invariably correspond to higher serum uric acid concentrations. The observed independent association between elevated SUA and early renal dysfunction has potential clinical relevance. Serum uric acid is inexpensive, widely available and commonly measured in routine clinical practice. An elevated value in a patient with T2DM could therefore serve as an additional signal to assess UACR and eGFR carefully, particularly when other renal risk factors such as hypertension or longer diabetes duration are present. It should, however, be regarded as an adjunct rather than a replacement for established DKD screening measures. The present results do not establish that lowering uric acid will prevent diabetic kidney disease. Observational associations cannot distinguish whether SUA is a causal mediator, a marker of metabolic and vascular risk, or a consequence of early reduction in renal urate clearance. Therapeutic decisions regarding urate-lowering treatment should therefore not be based on these findings alone. Prospective cohorts with repeated measurements and appropriately designed intervention trials are needed to determine whether SUA provides incremental predictive value and whether modification of uric acid alters renal outcomes. Overall, the findings add to evidence that SUA may help characterize renal risk in patients with T2DM. The stronger relationships with UACR, creatinine and eGFR than with HbA1c suggest that its clinical relevance may lie primarily in the renal and metabolic-risk domain rather than in assessment of glycemic control.

Strengths and Limitations

The study simultaneously evaluated serum uric acid in relation to glycemic control, albuminuria and estimated renal filtration and incorporated multivariable analysis to account for major clinical covariates. The use of routinely available biochemical variables increases its potential applicability to general medical practice. However, the cross-sectional design prevents determination of temporality and causality. Single measurements of SUA and UACR may be affected by biological variability, while dietary purine intake and all medications influencing urate handling may not be completely captured. The single-center setting may also limit generalizability. Most importantly, prospective follow-up is required to determine whether SUA predicts future decline in renal function independently of established DKD risk factors.

CONCLUSION

Higher serum uric acid levels were significantly associated with increased albuminuria, higher serum creatinine and lower eGFR among patients with T2DM. Elevated SUA remained independently associated with early renal dysfunction after adjustment for several conventional risk factors, whereas no straightforward positive relationship with HbA1c was observed. Serum uric acid may therefore serve as an inexpensive adjunctive marker for identifying patients who warrant closer evaluation for early renal involvement. Prospective longitudinal studies are necessary to establish its predictive value and to clarify whether uric acid is a causal therapeutic target or predominantly a marker of underlying renal and metabolic abnormalities.

REFERENCES

1. Cai XL, Han XY, Ji LN. High-normal serum uric acid is associated with albuminuria and impaired glomerular filtration rate in Chinese type 2 diabetic patients. Chin Med J (Engl). 2011;124(22):3629-34.
2. De Cosmo S, Viazzi F, Pacilli A, Giorda C, Ceriello A, Gentile S, et al. Serum uric acid and risk of CKD in type 2 diabetes. Clin J Am Soc Nephrol. 2015;10(11):1921-9.
3. Xia Q, Zhang SH, Yang SM, Zhu XL, Su S, Hu AP, et al. Serum uric acid is independently associated with diabetic nephropathy but not diabetic retinopathy in patients with type 2 diabetes mellitus. J Chin Med Assoc. 2020;83(4):350-6.
4. Kim ES, Kwon HS, Ahn CW, Lim DJ, Shin JA, Lee SH, et al. Serum uric acid level is associated with metabolic syndrome and microalbuminuria in Korean patients with type 2 diabetes mellitus. J Diabetes Complications. 2011;25(5):309-13.
5. Wei F, Chang B, Yang X, Wang Y, Chen L, Li WD. Serum uric acid levels were dynamically coupled with hemoglobin A1c in the development of type 2 diabetes. Sci Rep. 2016;6:28549.
6. Haque T, Rahman S, Islam S, Molla NH, Ali N. Assessment of the relationship between serum uric acid and glucose levels in healthy, prediabetic and diabetic individuals. Diabetol Metab Syndr. 2019;11:49.
7. Fouad M, Fathy H, Zidan A. Serum uric acid and its association with hypertension, early nephropathy and chronic kidney disease in type 2 diabetic patients. J Bras Nefrol. 2016;38(4):403-10.
8. Behradmanesh S, Horestani MK, Baradaran A, Nasri H. Association of serum uric acid with proteinuria in type 2 diabetic patients. J Res Med Sci. 2013;18(1):44-6.
9. Jalal DI, Rivard CJ, Johnson RJ, Maahs DM, McFann K, Rewers M, et al. Serum uric acid levels predict the development of albuminuria over 6 years in patients with type 1 diabetes. Nephrol Dial Transplant. 2010;25(6):1865-9.
10. Mauer M, Doria A. Uric acid and diabetic nephropathy risk. Contrib Nephrol. 2018;192:103-9.
11. Johnson RJ, Bakris GL, Borghi C, Chonchol MB, Feldman D, Lanaspa MA, et al. Hyperuricemia, acute and chronic kidney disease, hypertension, and cardiovascular disease: report of a scientific workshop organized by the National Kidney Foundation. Am J Kidney Dis. 2018;71(6):851-65.
12. Bjornstad P, Laffel L, Lynch J, El Ghormli L, Weinstock RS, Tollefsen SE, et al. Elevated serum uric acid is associated with greater risk for hypertension and diabetic kidney diseases in obese adolescents with type 2 diabetes: an observational analysis from the TODAY study. Diabetes Care. 2019;42(6):1120-8.
13. Ficociello LH, Rosolowsky ET, Niewczas MA, Maselli NJ, Weinberg JM, Aschengrau A, et al. High-normal serum uric acid increases risk of early progressive renal function loss in type 1 diabetes. Diabetes Care. 2010;33(6):1337-43.
14. Bonakdaran S, Hami M, Shakeri MT. Hyperuricemia and albuminuria in patients with type 2 diabetes mellitus. Iran J Kidney Dis. 2011;5(1):21-4.
15. Zoppini G, Targher G, Chonchol M, Ortalda V, Abaterusso C, Pichiri I, et al. Serum uric acid levels and incident chronic kidney disease in patients with type 2 diabetes and preserved kidney function. Diabetes Care. 2012;35(1):99-104.
16. Ito H, Abe M, Mifune M, Oshikiri K, Antoku S, Takeuchi Y, et al. Hyperuricemia is independently associated with coronary heart disease and renal dysfunction in patients with type 2 diabetes mellitus. PLoS One. 2011;6(11):e27817.
17. Tanaka K, Hara S, Hattori M, Sakai K, Onishi Y, Yoshida Y, et al. Role of elevated serum uric acid levels at the onset of overt nephropathy in the risk for renal function decline in patients with type 2 diabetes. J Diabetes Investig. 2015;6(1):98-104.
18. Bartáková V, Kuricová K, Pácal L, Nová Z, Dvořáková V, Švrčková M, et al. Hyperuricemia contributes to the faster progression of diabetic kidney disease in type 2 diabetes mellitus. J Diabetes Complications. 2016;30(7):1300-7.
19. Viazzi F, Piscitelli P, Giorda C, Ceriello A, Genovese S, Russo G, et al. Metabolic syndrome, serum uric acid and renal risk in patients with T2D. PLoS One. 2017;12(4):e0176058.
20. Chang YH, Lei CC, Lin KC, Chang DM, Hsieh CH, Lee YJ. Serum uric acid level as an indicator for CKD regression and progression in patients with type 2 diabetes—a 4.6-year cohort study. Diabetes Metab Res Rev. 2016;32(6):557-64.