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ORIGINAL RESEARCH

Association of Serum Uric Acid with Glycemic Status in Type 2 Diabetes Mellitus Patients with and without Metabolic Syndrome

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ABSTRACT

Background: Serum uric acid (SUA) has been associated with several cardiometabolic abnormalities, but its relation to glycaemic status in established type 2 diabetes mellitus (T2DM) is complex and may be influenced by metabolic syndrome (MetS), renal urate handling, and glycosuria. **Objective:** To compare SUA and glycaemic indices between patients with T2DM and comparison participants, and to examine glycaemic and SUA measures according to MetS status and diabetes duration among patients with T2DM. **Methods:** This hospital-based cross-sectional study included 155 patients with confirmed T2DM and 155 comparison participants at Bhima Bhoi Medical College and Hospital, Balangir, Odisha, between August 2023 and January 2025. MetS was defined using the harmonised IDF/AHA-NHLBI criteria. Continuous variables were compared using Welch independent-samples t tests, categorical variables using Pearson chi-square tests with odds ratios (ORs) and 95% confidence intervals (CIs), and diabetes-duration categories using one-way analysis of variance. **Results:** Compared with the comparison group, patients with T2DM had higher mean fasting blood glucose (130.7 ± 48.67 vs 106.9 ± 35.03 mg/dL; p<0.001), HbA1c (6.8 ± 1.69% vs 6.1 ± 1.38%; p<0.001), and body mass index (23.6 ± 3.91 vs 21.5 ± 3.53 kg/m²; p<0.001), whereas mean SUA was similar (5.8 ± 1.69 vs 5.7 ± 1.27 mg/dL; p=0.556). MetS was present in 71 of 155 patients (45.8%). HbA1c ≥6.5% was observed in 54.9% of patients with MetS and 39.3% without MetS (OR 1.88, 95% CI 0.99–3.57; p=0.052). Fasting blood glucose ≥100 mg/dL was more frequent among patients with MetS (93.0% vs 60.7%; OR 8.54, 95% CI 3.11–23.43; p<0.001), although fasting glucose is itself a component of the MetS definition. SUA ≥7 mg/dL did not differ significantly between the MetS and non-MetS groups (21.1% vs 16.7%; OR 1.34, 95% CI 0.60–3.01; p=0.478). Mean HbA1c and SUA also did not differ significantly across diabetes-duration categories (p=0.508 and p=0.876, respectively). **Conclusion:** In this cross-sectional cohort, SUA did not differ significantly between patients with T2DM and comparison participants, or according to MetS status or diabetes duration within the T2DM group. The apparent association between MetS and fasting glucose should be interpreted cautiously because fasting glucose is incorporated into the MetS definition. The HbA1c threshold comparison was borderline but did not reach conventional statistical significance. Longitudinal studies with patient-level multivariable analysis are required to determine whether SUA contributes independently to glycaemic dysregulation.

Keywords: Serum uric acid; Type 2 diabetes mellitus; Metabolic syndrome; Glycaemic status; HbA1c; Fasting blood glucose

Introduction

Diabetes mellitus is a major metabolic disorder characterised by chronic hyperglycaemia resulting from impaired insulin secretion, impaired insulin action, or both [1]. Globally, an estimated 536.6 million adults aged 20–79 years were living with diabetes in 2021, and this number is projected to reach 783.2 million by 2045 [2]. In India, the ICMR-INDIAB national cross-sectional study estimated a weighted diabetes prevalence of 11.4% (95% CI 10.2–12.5), corresponding to approximately 101 million people [3].

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Type 2 diabetes mellitus (T2DM), which accounts for most cases of diabetes, develops through interactions among genetic susceptibility, adiposity, diet, physical inactivity, and other environmental and metabolic factors [4]. Metabolic syndrome (MetS) represents a clustering of central adiposity, elevated triglycerides, reduced high-density lipoprotein cholesterol, elevated blood pressure, and elevated fasting glucose [5]. Its presence identifies individuals with substantial cardiometabolic risk, including increased risks of diabetes and cardiovascular disease [6].

Serum uric acid (SUA), the final oxidation product of purine metabolism in humans, has been associated with hypertension [7], incident chronic kidney disease [8], and broader metabolic dysfunction. Experimental work has implicated oxidative and metabolic pathways, including mitochondrial oxidative stress, in urate-related metabolic effects [9]. Prospective evidence has also linked higher SUA concentrations with subsequent development of T2DM [10].

The relationship between SUA and glycaemia in established diabetes, however, may not be linear. Glycosuria can increase urinary urate excretion and thereby lower circulating SUA, potentially attenuating associations observed at earlier stages of dysglycaemia [11]. In patients with established T2DM, a measured SUA concentration may therefore reflect the combined influence of insulin resistance, adiposity, renal urate handling, glycosuria, medications, and kidney function rather than glycaemia alone.

South Asian populations carry a high burden of MetS and cardiometabolic risk at comparatively lower body mass index (BMI), with a tendency toward central and visceral adiposity [12,13]. These characteristics may influence both glucose metabolism and serum urate concentrations. The present study therefore compared SUA and glycaemic indices between patients with T2DM and comparison participants and examined these measures according to MetS status and diabetes duration in a hospital-based cohort from Odisha, India.

Materials and Methods

Study design and setting

This hospital-based cross-sectional observational study was conducted in the Department of Biochemistry, Bhima Bhoi Medical College and Hospital (BBMCH), Balangir, Odisha, India, from August 2023 to January 2025. Ethical clearance was obtained from the Institutional Ethics Committee of BBMCH, and written informed consent was obtained from all participants before enrolment.

Study participants

The study included 155 patients with confirmed T2DM and 155 comparison participants. Patients with T2DM were recruited from the Medicine outpatient departments, whereas comparison participants were recruited from hospital staff volunteers and persons accompanying patients. T2DM was diagnosed according to American Diabetes Association criteria [1]. The two groups were treated as independent samples for between-group analyses; individual matching was not reported.

Eligibility criteria

Patients aged 19–60 years with T2DM were eligible irrespective of diabetes duration or glycaemic status and whether or not they were receiving oral antihyperglycaemic therapy. Exclusion criteria were type 1 diabetes mellitus, anaemia (haemoglobin <12 g/dL in men or <11 g/dL in women), gouty arthritis, use of medications known to materially alter SUA, diuretic therapy, chronic liver disease, chronic kidney disease, malignancy, pregnancy, insulin therapy, and comorbid hypothyroidism. These exclusions were intended to reduce major sources of confounding in the interpretation of SUA.

Clinical and anthropometric assessment

Demographic characteristics, medical history, medication use, and lifestyle information were recorded using a standardised questionnaire. Body weight, height, waist circumference, and blood pressure were measured using standard clinical procedures. BMI was calculated as weight in kilograms divided by height in metres squared. MetS was defined according to the harmonised International Diabetes Federation/American Heart Association-National Heart, Lung, and Blood

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Institute criteria, requiring any three of the following: central obesity (waist circumference ≥90 cm in men or ≥80 cm in women), triglycerides ≥150 mg/dL, reduced HDL cholesterol (<40 mg/dL in men or <50 mg/dL in women), blood pressure ≥130/85 mmHg or antihypertensive treatment, and fasting glucose ≥100 mg/dL or treatment for diabetes [5]. Because fasting glucose is itself one of the defining components of MetS, comparisons of fasting blood glucose ≥100 mg/dL according to MetS status were interpreted descriptively rather than as independent evidence of association.

Laboratory methods

Venous blood was collected after an overnight fast of at least 8 hours. Serum was separated for measurement of SUA and fasting blood glucose using the TBA-120 fully automated biochemical analyser (Toshiba, Japan) according to laboratory standard operating procedures. HbA1c was measured by a validated ion-exchange chromatographic method. Routine internal quality-control procedures were applied to all assays.

Statistical analysis

Data were entered in Microsoft Excel 2019 and analysed using IBM SPSS Statistics version 29.0. Continuous variables are presented as mean ± standard deviation (SD), and categorical variables as number and percentage. Between-group comparisons of continuous variables were performed using Welch independent-samples t tests, whereas categorical variables were compared using Pearson chi-square tests. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated for binary threshold comparisons. Mean HbA1c and SUA across diabetes-duration categories were compared using one-way analysis of variance. All analyses were unadjusted, and a two-sided p value <0.05 was considered statistically significant. HbA1c ≥6.5% and SUA ≥7 mg/dL were used only as analytic cut-points and should not be interpreted as individualised treatment targets or as sex-specific clinical definitions of hyperuricaemia.

Results

Participant characteristics

The study included 310 participants: 155 with T2DM and 155 in the comparison group. Among patients with T2DM, 56.8% were men and 43.2% were women, compared with 59.4% and 40.6%, respectively, in the comparison group. The age-group distribution differed between groups (p=0.007), whereas sex distribution was similar (p=0.645). Mean age was 48.8 ± 9.11 years in the T2DM group and 46.4 ± 12.14 years in the comparison group (p=0.050). Hypertension was more common among patients with T2DM (43.9% vs 9.0%; p<0.001). Mean fasting blood glucose, HbA1c, and BMI were significantly higher in the T2DM group, whereas mean SUA was similar between groups (5.8 ± 1.69 vs 5.7 ± 1.27 mg/dL; p=0.556) (Table 1).

Table 1. Sociodemographic and clinical characteristics of study participants

Variable	Category	T2DM (n=155), n (%)	Comparison group (n=155), n (%)	p value
Age group (years)	19–30	6 (3.9)	23 (14.8)	0.007
	31–40	29 (18.7)	30 (19.4)	
	41–50	45 (29.0)	33 (21.3)	
	51–60	75 (48.4)	69 (44.5)	
Sex	Male	88 (56.8)	92 (59.4)	0.645
	Female	67 (43.2)	63 (40.6)	
History of hypertension	Yes	68 (43.9)	14 (9.0)	<0.001
	No	87 (56.1)	141 (91.0)	
Antihypertensive medication	Yes	60 (38.7)	14 (9.0)	<0.001
	No	95 (61.3)	141 (91.0)	
Antidiabetic medication	Yes	140 (90.3)	0 (0)	<0.001
	No	15 (9.7)	155 (100)	

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Variable	Category	T2DM (n=155), n (%)	Comparison group (n=155), n (%)	p value
Duration of hypertension	<5 years	31 (45.6)	—	—
	≥5 years	37 (54.4)	—	
Duration of diabetes	<5 years	66 (42.6)	—	—
	≥5 years	89 (57.4)	—	
Mean age, years		48.8 ± 9.11	46.4 ± 12.14	0.050
HbA1c, %		6.8 ± 1.69	6.1 ± 1.38	<0.001
Serum uric acid, mg/dL		5.8 ± 1.69	5.7 ± 1.27	0.556
Fasting blood glucose, mg/dL		130.7 ± 48.67	106.9 ± 35.03	<0.001
BMI, kg/m²		23.6 ± 3.91	21.5 ± 3.53	<0.001

Values are n (%) unless otherwise stated. Continuous variables are mean ± SD. BMI, body mass index; HbA1c, glycated haemoglobin; T2DM, type 2 diabetes mellitus. Continuous-variable p values are from Welch independent-samples t tests; categorical p values are from Pearson chi-square tests.

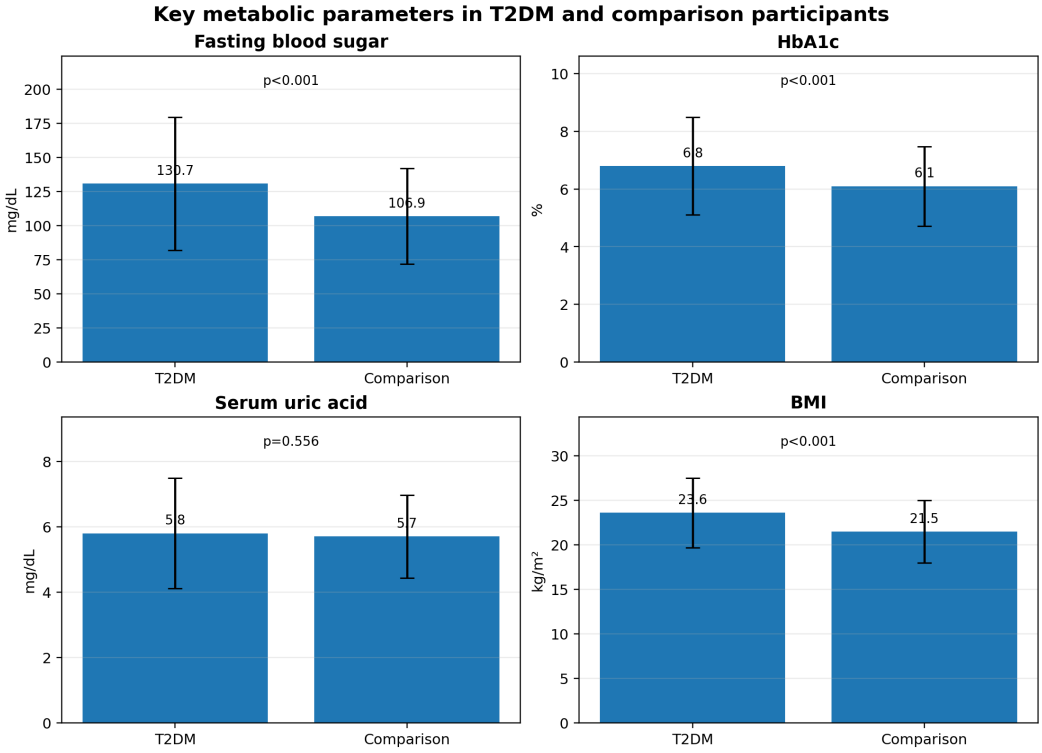


Figure 1. *Key metabolic parameters in patients with T2DM and comparison participants. Bars show mean ± SD; p values are from Welch independent-samples t tests.*

Glycaemic and serum uric acid profiles according to metabolic syndrome status

Among the 155 patients with T2DM, 71 (45.8%) met the criteria for MetS and 84 (54.2%) did not. HbA1c ≥6.5% was present in 39 of 71 patients with MetS (54.9%) and 33 of 84 without MetS (39.3%), corresponding to an OR of 1.88 (95% CI 0.99–3.57; p=0.052). Fasting blood glucose ≥100 mg/dL was observed in 66 of 71 patients with MetS (93.0%) and 51 of 84 without MetS (60.7%; OR 8.54, 95% CI 3.11–23.43; p<0.001); this comparison is partly definitional because fasting glucose is one of the criteria used to classify MetS. SUA ≥7 mg/dL occurred in 15 of 71 patients with MetS (21.1%) and 14 of 84 without MetS (16.7%; OR 1.34, 95% CI 0.60–3.01; p=0.478) (Table 2).

HbA1c and serum uric acid according to diabetes duration

Across diabetes-duration categories of ≤3 years, 3–6 years, and >6 years, mean HbA1c values were 7.01 ± 1.73%, 6.67 ± 1.50%, and 6.97 ± 1.89%, respectively; the overall difference was not statistically significant (one-way ANOVA p=0.508).

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Mean SUA values were 5.71 ± 1.50, 5.87 ± 1.80, and 5.82 ± 1.75 mg/dL, respectively, and likewise did not differ significantly across duration groups (p=0.876) (Table 3).

Table 2. Glycaemic indices and serum uric acid thresholds in patients with T2DM according to metabolic syndrome status

Variable	Category	With MetS (n=71), n (%)	Without MetS (n=84), n (%)	p value	OR (95% CI)
HbA1c, %	<6.5	32 (45.1)	51 (60.7)	0.052	1.88 (0.99–3.57)
	≥6.5	39 (54.9)	33 (39.3)		
Fasting blood glucose, mg/dL	<100	5 (7.0)	33 (39.3)	<0.001	8.54 (3.11–23.43)
	≥100	66 (93.0)	51 (60.7)		
Serum uric acid, mg/dL	<7	56 (78.9)	70 (83.3)	0.478	1.34 (0.60–3.01)
	≥7	15 (21.1)	14 (16.7)		

MetS, metabolic syndrome; CI, confidence interval; OR, odds ratio. Percentages are within MetS-status groups. p values are from Pearson chi-square tests. The fasting-glucose comparison is descriptive because fasting glucose is part of the MetS definition. The SUA threshold of 7 mg/dL is an analytic cut-point rather than a sex-specific clinical definition of hyperuricaemia.

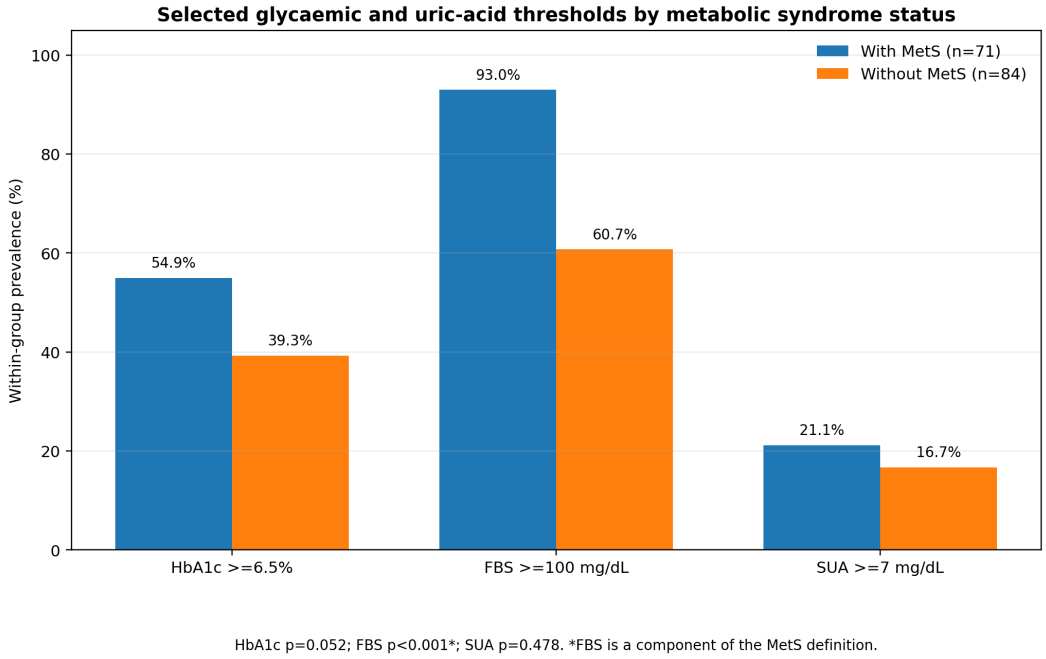


Figure 2. *Prevalence of selected glycaemic and serum uric acid thresholds in patients with T2DM with and without metabolic syndrome.*

The fasting-glucose comparison is descriptive because fasting glucose is a component of the MetS definition.

Table 3. Mean HbA1c and serum uric acid according to diabetes duration

Diabetes duration	n	Mean HbA1c, % ± SD	Mean SUA, mg/dL ± SD	ANOVA p value
≤3 years	55	7.01 ± 1.73	5.71 ± 1.50	HbA1c: 0.508; SUA: 0.876
3–6 years	60	6.67 ± 1.50	5.87 ± 1.80	
>6 years	40	6.97 ± 1.89	5.82 ± 1.75	

HbA1c, glycated haemoglobin; SUA, serum uric acid; SD, standard deviation. Overall p values are from one-way analysis of variance.

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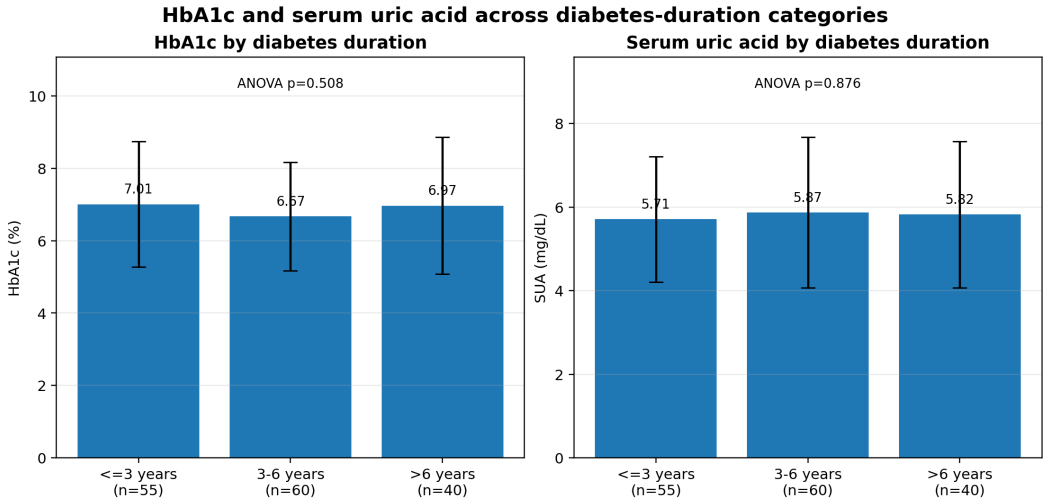


Figure 3. *Mean HbA1c and serum uric acid across diabetes-duration categories. Bars show mean ± SD; overall p values are from one-way analysis of variance.*

Discussion

This hospital-based cross-sectional study examined SUA and glycaemic measures in patients with T2DM and a comparison group and further described these measures according to MetS status and diabetes duration. Three findings merit emphasis. First, mean SUA did not differ significantly between patients with T2DM and comparison participants. Second, within the T2DM cohort, the proportion with SUA ≥7 mg/dL was not significantly different between patients with and without MetS. Third, neither mean SUA nor HbA1c differed significantly across diabetes-duration categories. By contrast, fasting blood glucose and HbA1c were higher in the T2DM group than in the comparison group, as expected from the clinical classification of the study groups.

MetS was present in 45.8% of the T2DM cohort, consistent with the substantial burden of clustered metabolic risk reported in South Asian populations [12]. The marked difference in fasting blood glucose ≥100 mg/dL between the MetS and non-MetS groups should not be interpreted as an independent biological association, because elevated fasting glucose or treatment for diabetes forms part of the harmonised MetS definition [5]. The HbA1c ≥6.5% threshold was numerically more frequent among patients with MetS (54.9% vs 39.3%), but the p value was 0.052 and the 95% CI for the OR included unity. It is therefore more appropriately interpreted as a borderline, statistically non-significant difference. Insulin resistance remains a biologically plausible common pathway linking MetS and dysglycaemia [14], but the present unadjusted cross-sectional data cannot establish causality.

The lack of a significant difference in SUA between patients with T2DM and comparison participants, and between MetS strata within T2DM, does not exclude a role for urate metabolism earlier in the development of dysglycaemia. Prospective studies have associated higher SUA with subsequent T2DM [10,15,16]. Once diabetes is established, however, the relation may be modified by renal and metabolic factors. Glycosuria can increase urinary urate excretion and lower circulating SUA [11], potentially attenuating cross-sectional associations between urate and glycaemic indices.

Adiposity is another important consideration. Mean BMI was higher in the T2DM group, and prior work has demonstrated close relationships between central or visceral adiposity and metabolic risk in Asian Indian populations [13]. Mendelian-randomisation evidence has also suggested that greater adiposity may contribute to higher SUA rather than SUA being a primary cause of adiposity [17]. At the same time, diabetes in South Asian populations can occur in lean or non-obese phenotypes [18]. These observations reinforce the need to account for body composition and central adiposity when examining the relation between SUA and glycaemic status.

No validated patient-level analysis of the continuous relationship between SUA and HbA1c was available in the present dataset. Accordingly, the findings should not be interpreted as evidence for or against an independent continuous SUA-HbA1c association. Future analyses should examine continuous SUA in relation to continuous HbA1c and fasting glucose

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using correlation and multivariable regression, with adjustment for age, sex, BMI or waist circumference, blood pressure, renal function, medication exposure, and other relevant metabolic covariates.

Neither mean HbA1c nor mean SUA differed significantly across the three diabetes-duration categories. Because the design was cross-sectional, these comparisons describe differences between groups of patients with different disease durations rather than within-person changes over time. Longitudinal follow-up would be required to determine whether glycaemic control or urate concentrations change systematically as diabetes progresses.

Taken together, the present data support cautious interpretation of SUA as a cross-sectional metabolic marker in established T2DM. Clinical assessment should continue to consider the broader cardiometabolic profile rather than interpreting SUA in isolation. Future studies incorporating estimated glomerular filtration rate, urinary urate excretion or fractional urate excretion, fasting insulin or HOMA-IR, medication classes, and multivariable modelling would provide a more rigorous assessment of the relationship among SUA, MetS, and glycaemic status.

Limitations

Its cross-sectional design precludes temporal or causal inference. The comparison group should not be interpreted as a strictly healthy normoglycaemic control group, because its group-level fasting glucose and HbA1c values indicate that dysglycaemia may have been present within the group. In addition, insulin-treated patients and those with chronic kidney disease were excluded; although these exclusions reduce important sources of confounding for SUA, they also limit the generalisability of the findings to patients with more advanced or complicated T2DM.

Fasting glucose is incorporated into the harmonised MetS definition, creating incorporation bias in the comparison of fasting glucose ≥100 mg/dL by MetS status. The fixed SUA threshold of 7 mg/dL was used as an analytic cut-point and is not a sex-specific clinical definition of hyperuricaemia. Patient-level continuous analyses of SUA against HbA1c and fasting glucose were not available, and kidney-function indices, urinary urate handling, and direct measures of insulin resistance were not included.

Conclusion

In this hospital-based cross-sectional cohort, patients with T2DM had higher fasting blood glucose, HbA1c, and BMI than comparison participants, whereas mean SUA was similar between groups. Within the T2DM cohort, the proportion with SUA ≥7 mg/dL did not differ significantly according to MetS status, and mean SUA did not differ across diabetes-duration categories. HbA1c ≥6.5% was more frequent among patients with MetS, but the difference did not reach conventional statistical significance. The strong MetS-fasting-glucose relationship is partly definitional because elevated fasting glucose is itself a component of MetS. These findings do not establish SUA as an independent determinant of glycaemic status. Longitudinal studies using patient-level multivariable analyses are needed to clarify the independent relationship between urate metabolism and glycaemic dysregulation in T2DM.

Declarations

Ethical approval and consent to participate: Ethical clearance was obtained from the Institutional Ethics Committee, BBMCH, Balangir. Written informed consent was obtained from all participants before enrolment.

Conflict of interest: The authors declare no conflict of interest.

Funding: No external funding was received for this study.

Data availability: Data are available from the corresponding author upon reasonable request, subject to applicable ethical and institutional requirements.

HbA1c and serum uric acid according to diabetes duration

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