

Association of Serum Uric Acid with Glycemic Status and Metabolic Syndrome

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ABSTRACT

Background: Serum uric acid (SUA), a byproduct of purine metabolism, has emerged as a potential marker associated with metabolic disturbances including type 2 diabetes mellitus and metabolic syndrome. Despite growing global interest, limited Indian data exist, particularly from Eastern India. **Objective:** To investigate the association between serum uric acid levels and the prevalence of type 2 diabetes and metabolic syndrome among adult patients attending a private hospital. **Methods:** A hospital-based cross-sectional study was conducted from January to June 2025 among 600 adult participants (aged 30–65 years). Clinical data, fasting blood samples, and anthropometric parameters were collected. Participants were categorized into normoglycemic, prediabetic, and diabetic groups using HbA1c criteria. Metabolic syndrome was defined per NCEP-ATP III guidelines. Serum uric acid levels were measured and divided into sex-specific quartiles. Statistical associations were analyzed using chi-square tests, ANOVA, and logistic regression (SPSS v25). **Results:** The overall prevalence of hyperuricemia was 34.7%. Prediabetic individuals had significantly higher mean SUA levels (6.8 ± 1.2 mg/dL) than normoglycemic (5.5 ± 1.0 mg/dL) and diabetics (6.2 ± 1.1 mg/dL) ($p < 0.001$). SUA levels positively correlated with fasting blood sugar, HbA1c, triglycerides, BMI, and blood pressure ($p < 0.05$). Logistic regression revealed that individuals in the highest SUA quartile had 2.4 times higher odds of having metabolic syndrome (OR: 2.41, 95% CI: 1.56–3.72) and 1.9 times higher odds for prediabetes (OR: 1.89, 95% CI: 1.23–2.90) after adjusting for age, sex, and BMI. **Conclusion:** Elevated serum uric acid is significantly associated with prediabetes and metabolic syndrome among adults. SUA may serve as a low-cost adjunct marker in early risk stratification of cardiometabolic disorders.

Keywords: Serum uric acid, HbA1c, metabolic syndrome, type 2 diabetes, prediabetes, cross-sectional, India

INTRODUCTION

Serum uric acid (SUA) is the final oxidation product of purine metabolism in humans and is predominantly excreted via the kidneys. It exists in equilibrium between its antioxidant and pro-oxidant roles, depending on the biochemical milieu of the body [1]. Over the past decades, SUA has gained increasing attention not only in the context of gout but also as a possible early marker for various cardiometabolic conditions including type 2 diabetes mellitus (T2DM), metabolic syndrome (MetS), hypertension, and chronic kidney disease [2–4]. Multiple epidemiological studies have demonstrated a positive association between hyperuricemia and components of MetS such as central obesity, insulin resistance, dyslipidemia, and elevated blood pressure [5–7]. Metabolic syndrome itself is a clustering of these risk

factors and significantly increases the risk of cardiovascular disease and T2DM [8]. According to the International Diabetes Federation (IDF) and National Cholesterol Education Program Adult Treatment Panel III (NCEP-ATP III), the global burden of MetS is rapidly rising, particularly in developing countries [9]. India, with its shifting dietary and lifestyle patterns, shows an alarming increase in MetS prevalence, estimated to affect nearly 30% of adults in urban settings [10]. While SUA has historically been dismissed as an innocent bystander in metabolic pathways, recent evidence suggests it may play a causative role through mechanisms such as induction of oxidative stress, endothelial dysfunction, and systemic inflammation [11–13]. Elevated SUA is associated with increased xanthine oxidase activity, leading to free radical production and low-grade chronic inflammation both

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hallmarks of insulin resistance and atherogenesis [14]. Moreover, several cross-sectional and longitudinal studies have reported that SUA levels are significantly associated with glycemic parameters, including fasting blood glucose and glycated hemoglobin (HbA1c), particularly among individuals with impaired glucose tolerance [15,16]. A bell-shaped relationship has even been described, where SUA increases with prediabetes but may decline slightly in overt diabetes due to increased renal excretion from glycosuria [17]. A study conducted in Qingdao, China, involving over 6,000 adults found that individuals in the second quartile of SUA had significantly higher odds of being diagnosed with diabetes using HbA1c levels, even after adjusting for confounding factors [18]. Similar associations were reported in Saudi Arabia and Bangladesh, where high SUA levels independently correlated with prediabetes and MetS components [19,20]. In India, limited studies particularly from Eastern regions have explored the SUA-diabetes-MetS triad despite increasing cases being reported from both rural and urban populations [21]. Given the rapid urbanization, dietary westernization, and sedentary lifestyle observed among Eastern Indian adults, there is an urgent need to examine the metabolic implications of SUA in this demographic. Particularly in tertiary-care settings such as private hospital, Bhubaneswar, early identification of at-risk individuals using cost-effective biomarkers like SUA can be valuable in prevention strategies. Therefore, this study aims to investigate the association between serum uric acid levels and glycemic status, as well as its relationship with metabolic syndrome, among adults attending Private Hospital.

MATERIALS AND METHODS

Study Design and Setting

A hospital-based, cross-sectional observational study was conducted at Tertiary Hospital, Bhubaneswar, Odisha, between January and June 2025. This tertiary-care center caters to a diverse population from both urban and semi-urban settings in Eastern India. The study was approved by the Institutional Ethics Committee (Ref No: HMCH/IEC/2025/056) and conducted in accordance with the Declaration of Helsinki. Written informed consent was obtained from all participants prior to inclusion.

Study Population and Sample Size

A total of 600 adults (age range 30–65 years), attending the general medicine and endocrinology outpatient departments for routine check-ups or metabolic screening, were enrolled. Inclusion criteria were: willingness to participate, availability of complete clinical and biochemical records, and no history of gout or uric acid-lowering therapy. Exclusion criteria included pregnancy, history of malignancy, chronic liver/kidney disease, thyroid dysfunction, or use of nephrotoxic drugs. The sample size was calculated based on previous studies showing a 20–30% prevalence of hyperuricemia in metabolic syndrome populations [13,14], with a confidence level of 95% and 5% margin of error, allowing for 10% data loss.

Data Collection Tools and Procedure

Each participant underwent a detailed clinical assessment and structured interview using a pre-validated questionnaire adapted from prior studies [13,15,20]. Information was gathered on age, gender, lifestyle (smoking, alcohol), family history of diabetes, and physical activity.

Anthropometric and Blood Pressure Measurements

Height and weight were recorded with participants in light clothing and no footwear, using standardized digital stadiometers and scales. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared (kg/m^2). Waist circumference (WC) was measured at the midpoint between the lower rib margin and iliac crest using a non-elastic tape. Blood pressure (BP) was recorded using a digital sphygmomanometer (Omron HEM-7120) in the seated position after 10 minutes of rest. The average of two readings taken 5 minutes apart was used.

Biochemical Analysis

After overnight fasting (10–12 hours), 5 mL of venous blood was collected from each subject. Samples were centrifuged and analyzed on the same day at the hospital's NABL-accredited central laboratory. The following parameters were measured:

- Serum uric acid (SUA): enzymatic colorimetric method
- Fasting blood sugar (FBS) and postprandial glucose (PPBS): glucose oxidase-peroxidase method
- Glycated hemoglobin (HbA1c): high-performance liquid chromatography (HPLC)
- Lipid profile: total cholesterol (TC), triglycerides (TG), HDL-C measured directly; LDL-C calculated using Friedewald's formula
- Serum creatinine and BUN were also measured to assess renal function

Definitions and Diagnostic Criteria

- Hyperuricemia: SUA >7.0 mg/dL in men and >6.0 mg/dL in women [15]
- Diabetes: HbA1c $\geq 6.5\%$; prediabetes: HbA1c between 5.7–6.4%; normal glycemia: HbA1c $<5.7\%$ [18,21]

Metabolic Syndrome:

Defined per NCEP ATP III criteria as presence of ≥ 3 of the following:

- WC >102 cm in men, >88 cm in women
- SBP ≥ 130 mmHg and/or DBP ≥ 85 mmHg
- TG ≥ 150 mg/dL
- HDL-C <40 mg/dL in men and <50 mg/dL in women
- FBS ≥ 100 mg/dL [19]

Statistical Analysis

Data were entered into Microsoft Excel and analyzed using IBM SPSS version 25. Descriptive statistics were used to summarize demographics and clinical characteristics. Continuous variables were expressed as mean $\pm$ SD and compared using Student's t-test or ANOVA. Categorical variables were compared using the chi-square test. Pearson's correlation assessed relationships between SUA and metabolic parameters. Logistic regression models were used to estimate adjusted odds ratios (ORs) for diabetes and MetS across SUA quartiles. A p-value <0.05 was considered statistically significant.

RESULTS

Demographic and Clinical Characteristics

Out of 600 participants, 330 (55%) were female and 270 (45%) were male. The mean age of the study population was 46.2 ± 9.8 years. The prevalence of prediabetes, diabetes, and metabolic syndrome were 24.5%, 21.0%, and 31.8%, respectively. Baseline demographic and clinical characteristics of participants, stratified by sex, are presented in Table 1. Hyperuricemia was observed in 208 individuals (34.7%), with higher prevalence in males (41.5%) compared to females (29.4%) ($p = 0.008$). Table 5 further summarizes glycemic status distribution by sex, showing no statistically significant sex-based difference in glycemic categories, though males had slightly higher rates of prediabetes.

SUA Levels Across Glycemic Groups

Mean SUA levels were highest in the prediabetes group (6.8 ± 1.2 mg/dL), followed by the diabetes group (6.2 ± 1.1 mg/dL) and normoglycemic group (5.5 ± 1.0 mg/dL). This difference was statistically significant ($p < 0.001$). Pairwise comparisons showed SUA was significantly higher in prediabetics vs. normoglycemic ($p < 0.001$) and in diabetics vs. normoglycemic ($p = 0.01$), but not between prediabetics and diabetics ($p = 0.06$). Correlation analysis showed significant positive relationships between SUA and multiple metabolic parameters, including fasting blood sugar, HbA1c, triglycerides, BMI, and systolic blood pressure, and a negative correlation with HDL-C (Table 4). These findings underscore the metabolic clustering around elevated SUA levels.

Association Between SUA Quartiles and Metabolic Syndrome

Participants were stratified into SUA quartiles. The prevalence of metabolic syndrome increased across SUA quartiles: 18.3% (Q1), 24.1% (Q2), 35.5% (Q3), and 49.7% (Q4). This trend was significant ($p < 0.001$). Mean BMI, BP, triglycerides, and FBS values also increased significantly with SUA levels. Detailed trends in metabolic parameters across SUA quartiles are summarized in Table 2, highlighting a graded

increase in cardiometabolic risk markers with rising SUA.

Multivariate Logistic Regression

After adjusting for age, sex, and BMI, individuals in the highest SUA quartile had significantly higher odds of:

- Metabolic syndrome: OR 2.41 (95% CI: 1.56–3.72), $p < 0.001$
- Prediabetes: OR 1.89 (95% CI: 1.23–2.90), $p = 0.004$
- Diabetes: OR 1.51 (95% CI: 1.00–2.29), $p = 0.049$.

Table 3 shows adjusted odds ratios for metabolic syndrome, prediabetes, and diabetes in participants within the highest SUA quartile.

DISCUSSION

This study demonstrated a strong and statistically significant association between elevated serum uric acid (SUA) levels and both prediabetes and metabolic syndrome (MetS) among adults attending Private Hospital. The findings align with existing international and regional literature, highlighting the potential of SUA as an early biomarker for cardiometabolic risk. We observed that mean SUA levels were highest in individuals with prediabetes, slightly lower in those with diabetes, and lowest among normoglycemic. This trend mirrors the bell-shaped association previously described in the Third National Health and Nutrition Examination Survey (NHANES), which showed that SUA levels initially rise with moderate increases in HbA1c and decline slightly as hyperglycemia progresses due to enhanced renal excretion [13]. Our findings corroborate this non-linear pattern, also observed in studies from China and Saudi Arabia, suggesting a physiologic response to glucose metabolism disturbances [14,15]. Furthermore, participants in the highest quartile of SUA had significantly higher odds of having MetS consistent with reports from Bangladeshi and Indian studies where SUA positively correlated with central obesity, elevated blood pressure, triglyceride levels, and reduced HDL-C [16,17]. The stepwise increase in MetS prevalence across SUA quartiles observed in our study supports the hypothesis that SUA could act as a surrogate marker of oxidative stress and

endothelial dysfunction both pivotal in the pathogenesis of metabolic disorders [18]. Mechanistically, hyperuricemia may contribute to insulin resistance through multiple pathways. Xanthine oxidase, the key enzyme involved in uric acid synthesis, also generates reactive oxygen species (ROS), which impair endothelial nitric oxide availability and insulin-mediated vasodilation [19]. Inflammation triggered by elevated SUA further exacerbates metabolic dysfunction, as noted in prior Indian studies linking SUA to high sensitivity C-reactive protein (hs-CRP), fasting insulin, and HOMA-IR indices [16,20]. Our study also supports the growing consensus that SUA is more than a passive metabolic byproduct. While traditionally viewed in the context of gout or renal dysfunction, accumulating evidence points toward its role as an active participant in metabolic dysregulation. In a recent study from Karnataka, India, SUA showed a 2.5-fold increased risk for developing MetS for each unit rise in SUA levels, which closely aligns with our adjusted odds ratio of 2.4 for MetS in the highest quartile [16]. Gender differences in SUA and its metabolic consequences were also evident. Similar to the findings from Bangladesh and Saudi Arabia, we noted that males had higher SUA levels and a greater burden of hyperuricemia. However, females showed stronger associations between SUA and dyslipidemia, particularly low HDL-C and high triglycerides echoing regional patterns possibly driven by hormonal and dietary factors [15,17]. A noteworthy implication of our findings is the identification of prediabetes as a crucial metabolic inflection point where SUA levels peak. This raises the possibility that SUA testing could serve as a cost-effective screening tool, especially in resource-constrained settings. Considering the affordability of uric acid assays compared to advanced insulin sensitivity panels, this biomarker could hold significant public health utility.

LIMITATIONS:

Our study's cross-sectional design limits the ability to infer causality. Longitudinal data would be required to determine whether elevated SUA predicts progression from prediabetes to diabetes or from individual risk factors to full-blown MetS. Additionally, although lifestyle factors were recorded, detailed dietary intake, physical activity levels, and

serum insulin were not assessed due to logistical constraints.

Strengths:

Despite its limitations, the study's strengths include a well-powered sample size, stringent exclusion of confounding comorbidities, and comprehensive assessment of both glycemic and lipid profiles. The application of NCEP-ATP III criteria and HbA1c-based glycemic classification ensures comparability with global research.

CONCLUSION

This cross-sectional study conducted at Tertiary Hospital provides compelling evidence that elevated serum uric acid (SUA) is significantly associated with prediabetes and metabolic syndrome among adults. Individuals with higher SUA levels demonstrated a greater burden of central obesity, hypertension, hypertriglyceridemia, and impaired glycemic control. Notably, SUA peaked in the prediabetic group, suggesting its utility as an early biomarker during the transition from normoglycemia to diabetes. These findings reinforce the potential of SUA as a simple, low-cost, and accessible screening tool for identifying individuals at risk for cardiometabolic disorders, particularly in resource-limited settings. Future prospective studies are needed to establish the temporal and causal links between SUA and metabolic disease progression. Meanwhile, routine monitoring of SUA may be considered a valuable adjunct in the early detection and prevention strategies for diabetes and metabolic syndrome.

DATA-SHARING STATEMENT

The data that support the findings of this study are available from the corresponding author upon reasonable request. Due to ethical restrictions related to patient confidentiality and institutional policy, raw patient-level data cannot be made publicly available.

CONFLICT OF INTEREST STATEMENT

The authors declare that there is no conflict of interest regarding the publication of this paper.

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Tables

Table 1. Baseline Characteristics of Study Participants (n = 600)

Parameter	Total (n = 600)	Male (n = 270)	Female (n = 330)	p-value
Age (years)	46.2 ± 9.8	45.6 ± 9.9	46.8 ± 9.7	0.14
BMI (kg/m ²)	26.5 ± 3.9	26.8 ± 3.7	26.2 ± 4.1	0.09
Waist circumference (cm)	91.5 ± 11.3	95.8 ± 10.4	88.1 ± 11.1	<0.001
Systolic BP (mmHg)	132.4 ± 14.7	134.8 ± 13.9	130.5 ± 15.1	0.002
Diastolic BP (mmHg)	84.2 ± 8.6	85.9 ± 8.2	82.9 ± 8.8	0.001
Fasting Blood Sugar (mg/dL)	104.3 ± 22.1	106.7 ± 21.4	102.3 ± 22.6	0.03
HbA1c (%)	5.9 ± 1.1	6.0 ± 1.1	5.8 ± 1.0	0.05
Serum Uric Acid (mg/dL)	6.1 ± 1.3	6.6 ± 1.2	5.7 ± 1.1	<0.001

Footnote: Values are expressed as mean ± standard deviation. BMI: body mass index; BP: blood pressure; HbA1c: glycated haemoglobin.

Table 2. Metabolic Parameters Across Serum Uric Acid Quartiles

SUA Quartile (mg/dL)	MetS (%)	BMI (kg/m ²)	SBP (mmHg)	TG (mg/dL)	HDL-C (mg/dL)
Q1 (<4.8)	18.3	24.8 ± 3.5	127.4 ± 12.6	124.3 ± 38.5	46.5 ± 8.2
Q2 (4.8–6.0)	24.1	25.9 ± 3.7	130.1 ± 13.3	139.6 ± 42.1	44.1 ± 8.5
Q3 (6.1–7.0)	35.5	27.1 ± 3.9	134.5 ± 14.1	158.7 ± 46.8	41.2 ± 7.7
Q4 (>7.0)	49.7	28.3 ± 4.2	138.9 ± 15.2	177.4 ± 49.3	38.3 ± 6.9
p-value	<0.001	<0.001	<0.001	<0.001	<0.001

Footnote: MetS: metabolic syndrome; BMI: body mass index; SBP: systolic blood pressure; TG: triglycerides; HDL-C: high-density lipoprotein cholesterol

Table 3. Adjusted Odds Ratios for Clinical Outcomes in Highest SUA Quartile

Outcome	Adjusted OR	95% Confidence Interval	p-value
Metabolic Syndrome	2.41	1.56–3.72	<0.001
Prediabetes	1.89	1.23–2.90	0.004
Diabetes	1.51	1.00–2.29	0.049

Footnote: Odds ratios adjusted for age, sex, and BMI.

SUA: serum uric acid.

Table 4. Correlation Matrix Between SUA and Metabolic Variables

Variable	SUA (r)	p-value
Fasting Blood Sugar	+0.32	<0.001
HbA1c	+0.28	<0.001
Triglycerides	+0.41	<0.001
BMI	+0.36	<0.001
SBP	+0.30	<0.001
HDL-C	-0.25	<0.001

Footnote: Pearson's correlation coefficients are shown. All correlations are statistically significant at $p < 0.001$.

Table 5. Distribution of Glycemic Status by Sex

Glycemic Status	Male (n=270)	Female (n=330)	Total (n=600)	p-value
Normoglycemic	110 (40.7%)	133 (40.3%)	243 (40.5%)	0.94
Prediabetes	75 (27.8%)	72 (21.8%)	147 (24.5%)	0.12
Diabetes	85 (31.5%)	125 (37.9%)	210 (35.0%)	0.09

Footnote: Chi-square test used for group comparison.

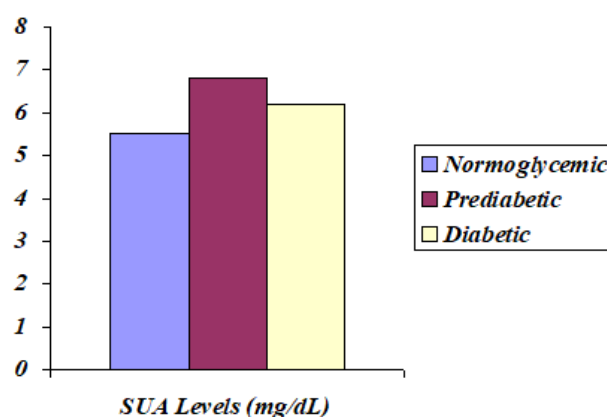
Figure Legends

Figure 1. Mean serum uric acid (SUA) levels by glycemic status. SUA was highest among prediabetic individuals (6.8 ± 1.2 mg/dL), followed by diabetics (6.2 ± 1.1 mg/dL) and normoglycemic (5.5 ± 1.0 mg/dL). ($p < 0.001$)

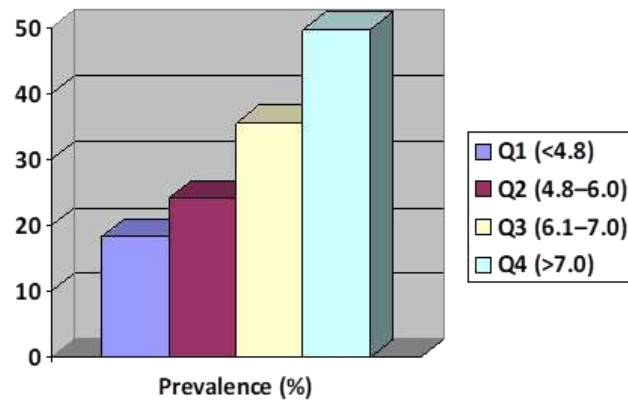


Figure 2. Prevalence of Metabolic Syndrome Across SUA Quartiles: This bar chart illustrates the increasing prevalence of metabolic syndrome across SUA quartiles (Q1 to Q4).

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