

Association of Serum Uric Acid with Metabolic Syndrome: A Cross-Sectional Study at a Tertiary Care Centre in Western GujaratCharmi N. Khant¹, Prem R. Lakhani²¹Senior Resident, Department of Internal Medicine, PDU Govt Medical College, Rajkot²Consultant, Department of Internal Medicine, Shree Giriraj Multi-specialty Hospital, Rajkot

Received: 01-09-2025 / Revised: 15-10-2025 / Accepted: 21-11-2025

Corresponding author: Dr. Charmi N Khant

Conflict of interest: Nil

Abstract

Background: Metabolic syndrome (MetS) - a combination of obesity, hypertension, dyslipidemia, and hyperglycemia - raises the risk of cardiovascular disease and type 2 diabetes. Serum uric acid (SUA) is increasingly linked to MetS through oxidative stress, inflammation, and insulin resistance. Hyperuricemia correlates with several MetS components, but data from Western Gujarat are limited. This study evaluates the association between SUA and MetS in a tertiary care setting, highlighting the regional need for routine SUA screening.

Material and Methods: This cross-sectional study was conducted over one year at a tertiary care centre in Western Gujarat, India. A total of 300 participants aged 30-65 years were enrolled, comprising 150 with MetS diagnosed using the harmonized International Diabetes Federation (IDF) criteria and 150 age- and sex-matched controls without MetS. Ethical approval was obtained from the institutional ethics committee, and informed consent was secured from all participants. Anthropometric measurements (body mass index, waist circumference), blood pressure, and fasting blood samples for glucose, lipid profile, and SUA were collected. Exclusion criteria included chronic kidney disease, gout, malignancy, or use of uric acid-altering medications. Data analysis involved descriptive statistics, independent t-tests for comparisons, Pearson correlation for associations, and logistic regression for odds ratios, using SPSS version 25.0. Significance was set at $p < 0.05$.

Results: The mean age of participants was 48.2 ± 9.5 years, with 55% males. Mean SUA levels were significantly higher in the MetS group (6.8 ± 1.4 mg/dL) compared to controls (4.5 ± 1.1 mg/dL; $p < 0.001$). Hyperuricemia (>7.0 mg/dL in men, >6.0 mg/dL in women) was prevalent in 62% of MetS cases versus 18% in controls. Positive correlations were found between SUA and waist circumference ($r = 0.42$, $p < 0.01$), triglycerides ($r = 0.38$, $p < 0.01$), systolic blood pressure ($r = 0.35$, $p < 0.01$), and fasting glucose ($r = 0.31$, $p < 0.05$), with an inverse correlation for HDL-cholesterol ($r = -0.29$, $p < 0.05$). Logistic regression revealed that elevated SUA increased the odds of MetS by 2.8-fold (95% CI: 1.9-4.1, $p < 0.001$), independent of age and sex.

Conclusion: Elevated SUA is strongly associated with MetS and its components in this Western Gujarat cohort, underscoring its potential as a biomarker for cardiometabolic risk. Routine SUA assessment could aid in early MetS detection and management.

Keywords Serum Uric Acid, Metabolic Syndrome, Hyperuricemia, Cardiovascular Risk, India.

This is an Open Access article that uses a funding model which does not charge readers or their institutions for access and distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/4.0>) and the Budapest Open Access Initiative (<http://www.budapestopenaccessinitiative.org/read>), which permit unrestricted use, distribution, and reproduction in any medium, provided original work is properly credited.

Introduction

Metabolic syndrome encompasses a group of conditions that heighten the risk for heart disease, stroke, and diabetes. It includes central obesity, high blood pressure, abnormal lipid levels, and impaired glucose tolerance. Over the past decade, the global burden of MetS has surged, particularly in developing nations like India, where dietary shifts and sedentary lifestyles contribute to its rise.

Serum uric acid, traditionally linked to gout, is now recognized as a possible mediator in MetS due to its involvement in endothelial dysfunction and pro-inflammatory pathways. Studies have shown that

hyperuricemia often coexists with insulin resistance, a core feature of MetS, suggesting a bidirectional relationship. In Asian populations, where MetS prevalence is high, exploring such associations can inform preventive strategies. [1]

Research from various regions indicates a consistent link between elevated SUA and MetS components. For instance, in urban settings, higher SUA correlates with obesity and dyslipidemia, potentially exacerbating cardiovascular outcomes. However, regional variations exist, influenced by genetics, diet, and environment. In India, with its

diverse ethnic groups, data from northern and southern states highlight differing patterns, but Western Gujarat lacks comprehensive studies. This gap is significant, as local factors like high-salt diets and water quality might influence uric acid metabolism. Addressing this can help tailor region-specific guidelines for MetS management. [2,3]

The present study was justified to investigate the association of SUA with MetS in a tertiary care setting in Western Gujarat. Given the rising MetS incidence in this area, understanding SUA's role could enhance screening protocols. By comparing SUA levels between MetS patients and controls, we aimed to provide evidence for incorporating SUA as a routine marker. This could lead to earlier interventions, reducing long-term complications and healthcare burdens in resource-limited settings.

Materials and Methods

This cross-sectional observational study was carried out at a tertiary care centre in Western Gujarat, India, for one year. The research adhered to ethical standards, with approval from the Institutional Ethics Committee. All participants provided written informed consent prior to enrollment, ensuring confidentiality and voluntary participation. The study population included adults aged 30-65 years attending the outpatient department for routine check-ups or MetS-related complaints. Sample size was calculated based on prior prevalence data, aiming for 300 participants (150 with MetS and 150 controls) to achieve 80% power at 5% significance level. General information such as age, gender, medical history, and lifestyle factors was collected via structured questionnaires.

Inclusion criteria encompassed individuals diagnosed with MetS using the harmonized IDF criteria: central obesity (waist circumference ≥ 90 cm in men, ≥ 80 cm in women) plus any two of raised triglycerides (≥ 150 mg/dL), reduced HDL-cholesterol (< 40 mg/dL in men, < 50 mg/dL in

women), elevated blood pressure (systolic ≥ 130 mmHg or diastolic ≥ 85 mmHg), or raised fasting glucose (≥ 100 mg/dL). Controls were age- and sex-matched individuals without MetS or its components. Exclusion criteria included chronic renal disease (eGFR < 60 mL/min), active gout, malignancies, pregnancy, or use of medications affecting uric acid levels (e.g., allopurinol, diuretics). Participants with acute illnesses or incomplete data were also excluded to maintain data integrity.

Data collection involved anthropometric assessments (height, weight, waist circumference using standardized tools) and clinical measurements (blood pressure via sphygmomanometer).

Fasting venous blood samples were analyzed for glucose (enzymatic method), lipid profile (automated analyzer), and SUA (uricase-peroxidase method). Statistical analysis employed SPSS version 25.0. Descriptive statistics summarized variables as means \pm SD or frequencies. Independent t-tests compared groups, Pearson correlation assessed associations, and multivariate logistic regression evaluated odds ratios, adjusting for confounders like age and gender. P-values < 0.05 were considered statistically significant.

Results

The study enrolled 300 participants, with balanced demographics between the MetS and control groups. Mean SUA was markedly elevated in MetS patients, showing clear associations with all components. Descriptive analysis revealed a higher prevalence of hyperuricemia in the MetS cohort. Correlation coefficients indicated positive links with obesity, hypertension, hyperglycemia, and hypertriglyceridemia, alongside a negative link with HDL-cholesterol. Logistic models confirmed SUA as an independent predictor of MetS. These findings align with the hypothesis of SUA's involvement in MetS pathogenesis.

Table 1: Demographic Characteristics of Study Participants

Characteristic	MetS Group (n=150)	Control Group (n=150)	p-value
Age (years, mean \pm SD)	49.1 \pm 9.8	47.3 \pm 9.2	0.12
Males (%)	56	54	0.78
BMI (kg/m ² , mean \pm SD)	28.4 \pm 4.1	23.2 \pm 3.5	< 0.001
Waist Circumference (cm, mean \pm SD)	96.5 \pm 8.7 (males), 88.2 \pm 7.4 (females)	82.3 \pm 6.9 (males), 76.4 \pm 5.8 (females)	< 0.001

Table 2: Prevalence of Metabolic Syndrome Components

Component	MetS Group (%)	Control Group (%)	p-value
Central Obesity	100	12	< 0.001
Raised Triglycerides	82	15	< 0.001
Reduced HDL-Cholesterol	76	18	< 0.001
Elevated Blood Pressure	88	14	< 0.001
Raised Fasting Glucose	74	10	< 0.001

Table 3: Serum Uric Acid Levels by Group

Parameter	MetS Group (mean \pm SD, mg/dL)	Control Group (mean \pm SD, mg/dL)	p-value
Overall SUA	6.8 \pm 1.4	4.5 \pm 1.1	<0.001
Males	7.2 \pm 1.3	4.9 \pm 1.0	<0.001
Females	6.3 \pm 1.4	4.0 \pm 1.1	<0.001
Hyperuricemia Prevalence (%)	62	18	<0.001

Table 4: Correlations and Odds Ratios for SUA with MetS Components

Component	Correlation Coefficient (r)	p-value	Adjusted OR (95% CI)
Waist Circumference	0.42	<0.01	2.1 (1.4-3.2)
Triglycerides	0.38	<0.01	2.5 (1.7-3.7)
HDL-Cholesterol	-0.29	<0.05	0.5 (0.3-0.8)
Systolic BP	0.35	<0.01	2.3 (1.5-3.5)
Fasting Glucose	0.31	<0.05	1.9 (1.2-2.9)
Overall MetS	-	-	2.8 (1.9-4.1)

Discussion

The interplay between serum uric acid and metabolic syndrome has garnered attention in recent years, as hyperuricemia emerges as more than just a gout precursor but a potential contributor to cardiometabolic disorders. In our study, we observed significantly higher SUA levels in patients with MetS compared to controls, with strong correlations across all components. This suggests that SUA may amplify the oxidative stress and inflammatory responses inherent in MetS, leading to endothelial damage and insulin resistance. Such findings underscore the importance of monitoring SUA in clinical practice, especially in regions like Western Gujarat where lifestyle factors may exacerbate these risks. Overall, our results contribute to the growing body of evidence linking uric acid metabolism to systemic metabolic health. [1,4]

In terms of central obesity, our data showed a robust positive correlation between SUA and waist circumference, with elevated SUA increasing the odds by 2.1-fold. This aligns with a South Indian study where 92% of hyperuricemic MetS patients exhibited abdominal obesity, highlighting a similar regional pattern possibly due to shared dietary habits like high purine intake. Another Indian investigation in diverse areas found a positive association between SUA tertiles and obesity metrics, reinforcing the visceral fat-uric acid nexus through adipokine dysregulation. Internationally, a Chinese coastal study reported central obesity risk escalating with SUA levels, attributing it to fructose metabolism and renal urate handling. Likewise, Korean data indicated higher SUA quartiles linked to increased body mass index, suggesting a universal mechanism involving leptin and inflammation. These comparisons validate our observations, emphasizing obesity's role in hyperuricemia across populations. [5,7] For dyslipidemia, particularly hypertriglyceridemia, we

noted a correlation of 0.38 with SUA, and odds ratio of 2.5. This mirrors findings from a Chennai-based study¹ on diabetic MetS patients, where SUA positively correlated with triglycerides and LDL, likely via impaired lipid clearance. An Indian population-wide analysis also showed elevated triglycerides in higher SUA tertiles, pointing to shared pathways in lipid peroxidation. On the global front, a Beijing study confirmed strong associations between SUA and hypertriglyceridemia, with gender-specific variations. Similarly, the Chinese coastal research linked SUA to low HDL and high triglycerides, proposing uric acid's interference in lipoprotein metabolism. Our results thus echo these, suggesting targeted lipid management in hyperuricemic individuals to mitigate MetS progression. [5,1]

Hypertension's association with SUA in our cohort was evident, with a 0.35 correlation and 2.3-fold odds. This is consistent with a South Indian tertiary care study, where 78.5% of hyperuricemic MetS cases had hypertension, possibly due to uric acid-induced vascular stiffness. Another Indian report noted positive correlations in metabolic risk factors including blood pressure. Internationally, Korean⁸ epidemiology revealed higher SUA quartiles elevating hypertension risk in MetS. A Chinese single-centre analysis also found SUA linked to high blood pressure, with female predominance. These parallels highlight uric acid's renin-angiotensin system activation as a common thread, supporting antihypertensive strategies incorporating SUA control. [9]

Regarding hyperglycemia, our study demonstrated a 0.31 correlation between SUA and fasting glucose, with 1.9-fold odds for impairment. This resonates with Chennai research showing positive links between SUA and both fasting and postprandial glucose in diabetic MetS. However, an Indian study observed an inverse SUA-HbA1c relation in diabetics, contrasting our general cohort

but indicating context-specific dynamics. Globally, the Korean study associated higher SUA with hyperglycemia components in MetS. Chinese data from coastal areas similarly tied SUA to high fasting glucose, implicating beta-cell dysfunction. Our findings thus contribute to understanding glucose-urate interactions, advocating for integrated diabetes screening. [8,10]

The inverse correlation with HDL-cholesterol (-0.29) in our results further cements SUA's dyslipidemic impact, with protective HDL diminished in hyperuricemia. This is supported by South Indian evidence of dyslipidemia in 57% of hyperuricemic cases. Chennai's diabetic study showed no significant HDL correlation but overall lipid derangements. Internationally, Beijing research strongly linked SUA to low HDL. The coastal Chinese study echoed this, with SUA elevating low HDL risk. These consistencies suggest antioxidant depletion by uric acid, warranting lifestyle interventions to boost HDL in MetS patients. [6]

Limitations of this study include its cross-sectional design, which precludes causality inference, and the relatively small sample from a single centre, potentially limiting generalizability. Future longitudinal studies could overcome these.

Conclusion

In conclusion, this study shows a clear association between elevated serum uric acid (SUA) and metabolic syndrome (MetS) in Western Gujarat, with hyperuricemia present in over 60% of MetS cases. SUA correlated with central obesity, high triglycerides, hypertension, and hyperglycemia, and inversely with HDL, highlighting its role in cardiometabolic risk. These findings support SUA as a simple, useful biomarker for early detection of MetS. Routine SUA screening and targeted management of hyperuricemia may help reduce the long-term cardiovascular impact of MetS, especially in resource-limited settings.

Bibliography

1. Rohith N, Anil Kumar T, Ashwin Kulkarni, Nagarjun Subhash. Study of serum uric acid

levels in patients of metabolic syndrome in a tertiary care centre in South India. *J Assoc Physicians India*. 2022;70(4):11-12.

2. Rajadhyaksha A, Sarate N, Raghorte N, Ingawale S. A clinical profile of patients with hyperuricemia and the relationship between hyperuricemia and metabolic syndrome: a cross-sectional study at a tertiary hospital in the Indian population. *J Assoc Physicians India*. 2022;70(5):11-12.
3. Remedios C, Shah M, Bhasker AG, Lakdawala M. Hyperuricemia: a reality in the Indian obese. *Obes Surg*. 2012;22(6):945-948.
4. Mundhe SA, Mhasde DR. The study of prevalence of hyperuricemia and metabolic syndrome in type 2 diabetes mellitus. *Int J Adv Med*. 2016;3(2):241-249.
5. Ni W, Wang R, Liu Z, Yuan X, Chi H, Lv D, Sun Y, Liu P, Xu J. Association of serum uric acid with metabolic syndrome and its components: a cross-sectional study in Chinese coastal population. *Metab Syndr Relat Disord*. 2020;18(2):103-109.
6. Yang T, Chu CH, Bai CH, You SL, Chou YC, Chou WY, Chien KL, Hwang LC, Su TC, Tseng CH, Sun CA. Uric acid level as a risk marker for metabolic syndrome: a Chinese cohort study. *Atherosclerosis*. 2012;220(2):525-531.
7. Jeong J, Suh YJ. Association between serum uric acid and metabolic syndrome in Koreans. *J Korean Med Sci*. 2019;34(48):e307.
8. Lee JK, Ryoo JH, Choi JM, Park SK. Serum uric acid level and the incidence of metabolic syndrome in middle-aged Korean men: a 5-year follow-up study. *J Prev Med Public Health*. 2014;47(6):317-326.
9. Mukhopadhyay P, Ghosh S, Pandit K, Chatterjee P, Majhi B, Chowdhury S. Uric acid and its correlation with various metabolic parameters: a population-based study. *Indian J Endocrinol Metab*. 2019;23(1):134-139.
10. Billa G, Dargad R, Mehta A. Prevalence of hyperuricemia in Indian subjects attending hyperuricemia screening programs-a retrospective study. *J Assoc Physicians India*. 2018;66(5):19-23.