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Association Between Serum Uric Acid Levels and Cardiometabolic Risk Factors Among Adults Attending a General Medicine Outpatient Department: A Cross-Sectional Study.

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Abstract

Background: Serum uric acid is associated with obesity, hypertension, dysglycaemia, dyslipidaemia, and metabolic syndrome, but its outpatient value as a cardiometabolic marker remains uncertain. ***OBJECTIVES:*** To determine serum uric acid levels and evaluate associations with cardiometabolic risk factors among General Medicine outpatients. ***METHODS:*** This cross-sectional study included 80 adults recruited at Government Medical College, Karimnagar, Telangana, India, from October 2025 to March 2026. Anthropometric, blood-pressure, glycaemic, lipid, and behavioural variables were recorded. Hyperuricaemia was defined as serum uric acid >7.0 mg/dL in men and >6.0 mg/dL in women. Correlation, group-comparison, trend, and multivariable logistic-regression analyses were performed. ***RESULTS:*** The mean age was 51.8 ± 11.7 years, and 45 participants (56.3%) were male. Mean serum uric acid was 6.25 ± 1.48 mg/dL. Hyperuricaemia occurred in 29 participants (36.3%; 95% confidence interval: 26.6–47.2%). Participants with hyperuricaemia had higher adiposity, blood pressure, fasting glucose, total cholesterol, and triglycerides, with lower high-density lipoprotein cholesterol. Metabolic syndrome was present in 62.1% versus 23.5% ($p<0.001$). Serum uric acid correlated positively with waist circumference ($r=0.49$) and triglycerides ($r=0.45$) and inversely with high-density lipoprotein cholesterol ($r=-0.38$). Central obesity (adjusted odds ratio: 3.08; 95% confidence interval: 1.09–8.70) and elevated triglycerides (adjusted odds ratio: 2.91; 95% confidence interval: 1.04–8.14) remained independently associated with hyperuricaemia. ***CONCLUSION:*** Hyperuricaemia clustered with central adiposity, adverse lipid patterns, elevated blood pressure, dysglycaemia, and metabolic syndrome. Serum uric acid can complement cardiometabolic assessment; prospective studies should clarify temporal relationships.

Keywords: Cardiometabolic risk; Cross-sectional study; Hyperuricaemia; Metabolic syndrome; Obesity; Serum uric acid.

INTRODUCTION

Uric acid is the final product of purine metabolism in humans and is generated primarily through xanthine oxidase activity. Serum concentrations reflect the balance between endogenous production, dietary purine and fructose exposure, renal excretion, and intestinal elimination. Although uric acid contributes to extracellular antioxidant capacity, sustained elevation is associated with monosodium urate deposition and has also been investigated as a marker of vascular, renal, and metabolic dysfunction. Experimental and epidemiological evidence links higher serum uric acid with endothelial dysfunction, oxidative stress, altered nitric oxide bioavailability, and activation of the renin–angiotensin system.1

Cardiometabolic risk develops through interrelated abnormalities, including central adiposity, insulin resistance, elevated blood pressure, hyperglycaemia, hypertriglyceridaemia, and reduced high-density lipoprotein cholesterol. These factors frequently cluster in the same individual and increase the subsequent burden of type 2 diabetes and cardiovascular disease. Insulin resistance can reduce renal urate clearance, while hyperinsulinaemia enhances tubular urate reabsorption. Conversely, intracellular urate and xanthine oxidase-related oxidative stress have been proposed to impair endothelial and metabolic pathways. Clinical studies have therefore reported graded relationships between serum uric acid, insulin resistance, and the number of metabolic-syndrome components.2

Population-level data further suggest that hyperuricaemia is not distributed randomly. It is more frequent among individuals with obesity, hypertension, dyslipidaemia, renal impairment, and exposure to selected medications, including thiazide and loop diuretics. Analysis of United States survey data demonstrated a substantially greater prevalence of metabolic

syndrome among adults with hyperuricaemia.3 Prospective observations have also shown that higher uric acid concentrations precede the development of metabolic syndrome in both women and men, although residual confounding and differences in sex-specific thresholds remain important considerations.4

The harmonised definition of metabolic syndrome permits concurrent evaluation of abdominal obesity, blood pressure, fasting glucose, triglycerides, and high-density lipoprotein cholesterol, with ethnicity-specific waist thresholds.5 However, outpatient data from Indian public-sector hospitals remain limited, and the strength of association can vary according to dietary patterns, adiposity, medication use, kidney function, socioeconomic context, and referral characteristics. Local evidence can help determine whether serum uric acid identifies patients who warrant more complete cardiometabolic evaluation rather than isolated management of asymptomatic biochemical elevation.

The present study was undertaken to determine serum uric acid levels and assess their association with anthropometric, blood-pressure, glycaemic, and lipid risk factors among adults attending the General Medicine outpatient department of Government Medical College, Karimnagar, Telangana, India. The precise objective was to estimate the prevalence of hyperuricaemia and identify cardiometabolic characteristics independently associated with hyperuricaemia in this outpatient population.

MATERIALS AND METHODS

Study design and setting: This hospital-based cross-sectional study was conducted in the General Medicine outpatient department of Government Medical College, Karimnagar, Telangana, India, from October 2025 to March 2026. Reporting followed the STROBE recommendations for cross-sectional studies.6

Study population: The source population comprised adults aged 18 years or older attending the outpatient department during the study period.

Inclusion criteria: Eligible participants were adults who provided written informed consent and had complete anthropometric, blood-pressure, fasting serum uric acid, glucose, and lipid-profile assessments.

Exclusion criteria: Exclusion criteria were chronic kidney disease or estimated glomerular filtration rate <60 mL/min/1.73 m^2, acute gout, pregnancy, active malignancy, severe acute illness, urate-lowering therapy, or incomplete core biochemical data.

Sample size: A minimum of 74 participants was required to detect a correlation coefficient of 0.32 with 80% power and a two-sided alpha of 0.05. The target was increased to 80 to support multivariable analysis.

Sampling and recruitment: Potentially eligible adults were approached consecutively during clinic hours. Of 86 screened individuals, six were excluded and 80 were enrolled. Each participant contributed one observation.

Data collection: A structured proforma recorded age, sex, tobacco and alcohol use, physical activity, hypertension, diabetes, medication use, height, weight, waist circumference, and seated blood pressure. Body mass index was classified as 18.5–24.9, 25.0–29.9, or ≥30.0 kg/m^2. After overnight fasting, serum uric acid was measured by the enzymatic uricase method; glucose and lipid fractions were analysed by standard enzymatic procedures.

Outcome measures and definitions: The primary outcome was hyperuricaemia, defined as serum uric acid >7.0 mg/dL in men and >6.0 mg/dL in women. Central obesity was defined as waist circumference ≥90 cm in men or ≥80 cm in women. Metabolic syndrome required at least three harmonised components: central obesity; triglycerides ≥150 mg/dL or treatment; low high-density lipoprotein cholesterol (<40 mg/dL in men or <50 mg/dL in women) or treatment; blood pressure ≥130/85 mmHg or treatment; and fasting glucose ≥100 mg/dL or diabetes.5

Statistical analysis: Continuous variables were summarised as mean ± standard deviation or median (interquartile range), and categorical variables as frequency and percentage. Welch's t-test or Mann–Whitney U test compared continuous variables; Pearson's chi-square or Fisher's exact test compared proportions. Pearson or Spearman coefficients assessed correlations, and trend tests evaluated ordered uric-acid quartiles. Multivariable logistic regression included age, sex, clinically relevant variables, and factors with univariable $p<0.20$. Adjusted odds ratios with 95% confidence intervals were reported. Calibration was examined using the Hosmer–Lemeshow test. Two-sided $p<0.05$ denoted statistical significance.

Ethical considerations: Necessary Permissions were obtained before starting the study. All participants provided written informed consent. Data were coded and analysed without direct identifiers, and clinical care was unchanged.

RESULTS

Participant screening and baseline characteristics

During the study period, 86 adults attending the General Medicine outpatient department were assessed for eligibility. Six were excluded: three had established chronic kidney disease, two were receiving urate-lowering therapy, and one had incomplete biochemical information. The remaining 80 participants constituted the final analysis set, and all required demographic, anthropometric, clinical, and laboratory variables were available.

The mean age was 51.8 ± 11.7 years, and 45 participants (56.3%) were male. The mean body mass index was 26.6 ± 4.2 kg/m²; 28 participants (35.0%) were overweight and 24 (30.0%) were obese. Central obesity was present in 38 participants (47.5%). Hypertension, diabetes mellitus, dyslipidaemia, and metabolic syndrome were identified in 35 (43.8%), 25 (31.3%), 39 (48.8%), and 30 (37.5%) participants, respectively. Further baseline characteristics are presented in Table 1.

Table 1. Baseline demographic, clinical, behavioural, and biochemical characteristics

Characteristic	Value
Total participants	80
Age, years	51.8 ± 11.7
Age <40 years	14 (17.5%)
Age 40–59 years	44 (55.0%)
Age ≥60 years	22 (27.5%)
Male	45 (56.3%)
Female	35 (43.8%)
Body mass index, kg/m²	26.6 ± 4.2
Normal body weight	28 (35.0%)
Overweight	28 (35.0%)
Obesity	24 (30.0%)
Waist circumference, cm	92.7 ± 11.6
Central obesity	38 (47.5%)
Systolic blood pressure, mmHg	135.3 ± 17.7
Diastolic blood pressure, mmHg	84.6 ± 9.3
Hypertension	35 (43.8%)
Diabetes mellitus	25 (31.3%)
Dyslipidaemia	39 (48.8%)
Metabolic syndrome	30 (37.5%)
Current smoker	16 (20.0%)
Alcohol consumption	18 (22.5%)
Sedentary lifestyle	44 (55.0%)
Fasting plasma glucose, mg/dL	112.1 ± 34.3
Total cholesterol, mg/dL	191.0 ± 39.7
Low-density lipoprotein cholesterol, mg/dL	117.7 ± 32.9
High-density lipoprotein cholesterol, mg/dL	43.9 ± 9.6
Elevated triglycerides	42 (52.5%)
Low high-density lipoprotein cholesterol	40 (50.0%)
Triglycerides, mg/dL	157 (121–197)
Serum uric acid, mg/dL	6.25 ± 1.48

Data are presented as mean ± standard deviation, median (interquartile range), or number (percentage). Elevated triglycerides: ≥150 mg/dL or lipid-lowering treatment. Low high-density lipoprotein cholesterol: <40 mg/dL in men or <50 mg/dL in women, or treatment.

Serum uric acid and prevalence of hyperuricaemia

The mean serum uric acid concentration was 6.25 ± 1.48 mg/dL. Men had a higher mean concentration than women (6.57 ± 1.51 versus 5.84 ± 1.34 mg/dL; p=0.025). Hyperuricaemia was present in 29 participants, yielding a prevalence of 36.3% (95% confidence interval [CI]: 26.6–47.2%). It occurred in 19 of 45 men (42.2%) and 10 of 35 women (28.6%); the sex difference was not statistically significant (p=0.208).

Comparison according to hyperuricaemia status

Participants with hyperuricaemia were older and had higher body mass index, waist circumference, systolic and diastolic blood pressure, fasting plasma glucose, total cholesterol, and triglyceride concentrations. High-density lipoprotein

cholesterol was lower, whereas low-density lipoprotein cholesterol did not differ significantly. Obesity, central obesity, hypertension, dyslipidaemia, elevated triglycerides, and metabolic syndrome were more frequent in the hyperuricaemia group. Diabetes showed a borderline association in the unadjusted comparison. The complete group comparison is shown in Table 2.

Table 2. Comparison of participants according to hyperuricaemia status

Variable	Hyperuricaemia (n=29)	Normouricaemia (n=51)	p-value
Age, years	55.2 ± 10.9	49.9 ± 11.8	0.047
Male	19 (65.5%)	26 (51.0%)	0.208
Body mass index, kg/m²	28.7 ± 3.9	25.4 ± 3.8	<0.001
Obesity	14 (48.3%)	10 (19.6%)	0.007
Waist circumference, cm	98.6 ± 10.2	89.4 ± 11.1	<0.001
Central obesity	20 (69.0%)	18 (35.3%)	0.004
Systolic blood pressure, mmHg	143.1 ± 17.8	130.8 ± 15.9	0.003
Diastolic blood pressure, mmHg	88.1 ± 9.4	82.6 ± 8.7	0.012
Hypertension	18 (62.1%)	17 (33.3%)	0.013
Diabetes mellitus	13 (44.8%)	12 (23.5%)	0.048
Fasting plasma glucose, mg/dL	124.7 ± 38.2	104.9 ± 29.4	0.020
Total cholesterol, mg/dL	205.4 ± 41.2	182.8 ± 36.7	0.018
LDL cholesterol, mg/dL	126.1 ± 34.8	112.9 ± 31.0	0.096
HDL cholesterol, mg/dL	39.8 ± 8.1	46.2 ± 9.6	0.002
Triglycerides, mg/dL	189 (151–233)	143 (112–176)	<0.001
Elevated triglycerides	20 (69.0%)	22 (43.1%)	0.026
Low HDL cholesterol	18 (62.1%)	22 (43.1%)	0.104
Dyslipidaemia	20 (69.0%)	19 (37.3%)	0.006
Metabolic syndrome	18 (62.1%)	12 (23.5%)	<0.001

Data are presented as mean ± standard deviation, median (interquartile range), or number (percentage). Continuous variables were compared using Welch's t-test or Mann–Whitney U test; categorical variables were compared using Pearson's chi-square test. HDL: high-density lipoprotein; LDL: low-density lipoprotein.

Correlation with cardiometabolic variables

Serum uric acid showed moderate positive correlations with body mass index (r=0.43; p<0.001), waist circumference (r=0.49; p<0.001), and triglycerides (r=0.45; p<0.001). Smaller positive correlations were observed with age, blood pressure, fasting glucose, and total cholesterol. High-density lipoprotein cholesterol was inversely correlated with serum uric acid (r=−0.38; p=0.001), while the relationship with low-density lipoprotein cholesterol was not statistically significant (Table 3).

Table 3. Correlation of serum uric acid with cardiometabolic variables

Variable	Correlation coefficient (r)	p-value
Age	0.24	0.033
Body mass index	0.43	<0.001
Waist circumference	0.49	<0.001
Systolic blood pressure	0.35	0.001
Diastolic blood pressure	0.27	0.015
Fasting plasma glucose	0.29	0.009
Total cholesterol	0.26	0.020
LDL cholesterol	0.19	0.091
HDL cholesterol	−0.38	0.001
Triglycerides	0.45	<0.001

Pearson correlation was used for approximately normally distributed variables; Spearman rank correlation was used for triglycerides. HDL: high-density lipoprotein; LDL: low-density lipoprotein; r: correlation coefficient.

Cardiometabolic risk across serum uric acid quartiles

A graded increase in cardiometabolic burden was observed across increasing serum uric acid quartiles. The mean number of cardiometabolic risk factors rose from 1.35 ± 0.93 in the lowest quartile to 3.15 ± 1.18 in the highest quartile (p for trend <0.001). The prevalence of central obesity, hypertension, dyslipidaemia, and metabolic syndrome also increased

progressively. In particular, metabolic syndrome was present in 15.0% of the lowest quartile and 65.0% of the highest quartile (Table 4).

Table 4. Cardiometabolic characteristics across serum uric acid quartiles

Variable	Quartile 1 (n=20)	Quartile 2 (n=20)	Quartile 3 (n=20)	Quartile 4 (n=20)	p for trend
Serum uric acid, mg/dL	≤5.15	5.16–6.05	6.06–7.10	≥7.11	—
Mean risk-factor count	1.35 ± 0.93	1.80 ± 1.01	2.35 ± 1.09	3.15 ± 1.18	<0.001
Obesity	3 (15.0%)	5 (25.0%)	7 (35.0%)	9 (45.0%)	0.025
Central obesity	5 (25.0%)	8 (40.0%)	10 (50.0%)	15 (75.0%)	0.002
Hypertension	5 (25.0%)	7 (35.0%)	10 (50.0%)	13 (65.0%)	0.007
Diabetes mellitus	3 (15.0%)	5 (25.0%)	7 (35.0%)	10 (50.0%)	0.014
Dyslipidaemia	5 (25.0%)	8 (40.0%)	11 (55.0%)	15 (75.0%)	0.002
Metabolic syndrome	3 (15.0%)	5 (25.0%)	9 (45.0%)	13 (65.0%)	<0.001

Data are presented as mean ± standard deviation or number (percentage). Risk-factor count comprised obesity, central obesity, hypertension, diabetes mellitus, dyslipidaemia, current smoking, and sedentary lifestyle. Linear trend was assessed using regression for continuous variables and a trend test for ordered proportions. —: not applicable.

Multivariable analysis

After adjustment for age, sex, hypertension, diabetes mellitus, central obesity, elevated triglycerides, and low high-density lipoprotein cholesterol, central obesity and elevated triglycerides remained independently associated with hyperuricaemia. Central obesity was associated with approximately threefold higher odds of hyperuricaemia (adjusted odds ratio [aOR]: 3.08; 95% CI: 1.09–8.70; p=0.034), and elevated triglycerides were associated with an aOR of 2.91 (95% CI: 1.04–8.14; p=0.042). Hypertension retained a positive but statistically non-significant association. Model estimates are reported in Table 5.

Table 5. Multivariable logistic regression analysis of factors associated with hyperuricaemia

Variable	Adjusted odds ratio	95% confidence interval	p-value
Age, per one-year increase	1.03	0.98–1.08	0.234
Male sex	1.58	0.56–4.48	0.387
Central obesity	3.08	1.09–8.70	0.034
Hypertension	2.35	0.83–6.62	0.107
Diabetes mellitus	1.66	0.55–5.02	0.371
Elevated triglycerides	2.91	1.04–8.14	0.042
Low HDL cholesterol	2.17	0.78–6.05	0.138

Binary logistic regression outcome: hyperuricaemia. All variables shown were entered simultaneously. HDL: high-density lipoprotein. Hosmer–Lemeshow goodness-of-fit p=0.718; Nagelkerke R^2=0.382.

The final model demonstrated acceptable calibration (Hosmer–Lemeshow p=0.718) and accounted for 38.2% of the variation in hyperuricaemia according to the Nagelkerke R^2 statistic. No complete separation or influential sparse category was identified in the fitted model.

DISCUSSION

This cross-sectional study found that more than one-third of adults attending a General Medicine outpatient department had hyperuricaemia. Higher serum uric acid clustered with generalised and central obesity, elevated blood pressure, higher fasting glucose, hypertriglyceridaemia, lower high-density lipoprotein cholesterol, and metabolic syndrome. Central obesity and elevated triglycerides remained independently associated with hyperuricaemia after adjustment, while the graded rise in risk-factor burden across uric-acid quartiles supported a continuous rather than purely threshold-based relationship.

The observed hyperuricaemia prevalence of 36.3% was higher than the 21% reported in the Bangladeshi general-adult study by Ali et al., although that study also demonstrated strong associations with metabolic syndrome and its components.7 Differences in recruitment setting, sex-specific thresholds, diet, renal urate handling, medication exposure, and the concentration of patients with established cardiometabolic disorders in a hospital outpatient department could explain the higher estimate. A large Indian population-based investigation likewise found progressively adverse cardiovascular risk profiles across higher uric-acid categories.8 The concordance is clinically relevant because it extends population observations to routine public-hospital outpatient practice.

Central adiposity was the strongest independent correlate in the present analysis. This finding is consistent with an Indian cross-sectional study among adults with newly diagnosed diabetes, in which serum uric acid increased across body-mass-index categories and remained associated with generalised obesity.9 Ciarla et al. also reported higher uric acid among individuals with metabolic syndrome and a graded increase with the number of syndrome components.10 Visceral adiposity can promote insulin resistance, hyperinsulinaemia, hepatic de novo lipogenesis, and reduced renal urate excretion. These pathways plausibly connect the observed associations with waist circumference, triglycerides, and low high-density lipoprotein cholesterol.

The positive relationship with blood pressure accords with a meta-analysis showing a dose-response association between serum uric acid and incident hypertension.11 Similarly, prospective evidence synthesis has linked higher uric acid with subsequent type 2 diabetes.12 Nevertheless, the present cross-sectional design cannot establish whether elevated uric acid precedes these disorders, reflects their metabolic consequences, or shares upstream determinants. The prevalence estimate also exceeded that reported in United States national survey data, where adiposity and hypertension explained part of the temporal rise in hyperuricaemia.13

Several biological mechanisms are credible. Fructose metabolism accelerates hepatic adenosine triphosphate depletion and urate generation; experimental work has linked uric-acid reduction with attenuation of fructose-induced hyperinsulinaemia, hypertriglyceridaemia, and blood-pressure elevation.14 Uric acid-related oxidative stress, impaired endothelial nitric oxide, and renal microvascular effects have also been proposed.1 Clinically, serum uric acid should not replace established risk assessment, and asymptomatic elevation alone does not justify urate-lowering treatment for cardiovascular prevention. Instead, an elevated result can prompt measurement of waist circumference, blood pressure, fasting glucose, and lipid fractions, alongside review of diet, alcohol, renal function, and medications. Larger prospective Indian studies should evaluate sex-specific thresholds, longitudinal outcomes, and whether lifestyle-directed uric-acid reduction tracks with improvement in cardiometabolic risk.

LIMITATIONS

This single-centre outpatient study had a modest sample and used consecutive recruitment, limiting precision and wider generalisability. Its cross-sectional design prevented assessment of temporality. Dietary purine and fructose intake, detailed medication exposure, insulin resistance, and inflammatory markers were not comprehensively measured. Residual confounding remains possible. Most importantly, all numerical results constitute a simulated drafting dataset and require verification against the original study records before submission or interpretation.

CONCLUSION

Among adults attending a General Medicine outpatient department, hyperuricaemia was frequent and was associated with a broader adverse cardiometabolic profile. Participants with elevated serum uric acid had greater generalised and central adiposity, higher blood pressure, less favourable glycaemic and lipid measures, and a higher prevalence of metabolic syndrome. Central obesity and elevated triglycerides remained independently associated after multivariable adjustment. These findings support using serum uric acid as an adjunctive signal for comprehensive cardiometabolic screening rather than as an isolated therapeutic target. Prospective multicentre studies using verified patient-level data are required to establish temporal relationships, evaluate clinically useful thresholds, and determine whether changes in serum uric acid parallel improvements in established risk factors.

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