International Journal of Research in Medical Sciences
Mala HR et al. Int J Res Med Sci. 2026 Dec;14(12):5768-5775
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: https://dx.doi.org/10.18203/2320-6012.ijrms20264481

Original Research Article

Association between serum uric acid levels and cardiovascular risk factors in adults

Hilal Razvi Mala[1], Mohammed Waqas[2], Hira Junejo[3], Zohaib Ansari[4], Syeda Ayesha Siddiqua[5]*

[1]Department of Cardiology, Freeman Hospital, United Kingdom
[2]Department of General Medicine, NCIC NHS Foundation Trust, Carlisle, United Kingdom
[3]Cardiology Trust Grade Doctor, NCIC NHS Foundation Trust, Cumbria, United Kingdom
[4]FY2 General Internal Medicine, NCIC NHS Foundation Trust, Cumbria, United Kingdom
[5]Deccan College of Medical Sciences, Santosh Nagar, Hyderabad, Telangana, India

Received: 11 September 2026
Revised: 14 October 2026
Accepted: 13 November 2026

***Correspondence:**
Dr. Syeda Ayesha Siddiqua,
E-mail: ayesharesearch123@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: Elevated serum uric acid has increasingly been associated with hypertension, obesity, dyslipidemia, and other cardiometabolic abnormalities. Its usefulness as an additional cardiovascular risk marker, however, remains incompletely established. Aim was to assess the association between serum uric acid levels and cardiovascular risk factors among adults.
Methods: This cross-sectional study included 150 adults aged ≥20 years. Demographic characteristics, body mass index, blood pressure, fasting blood glucose, and lipid profile were assessed. Participants were categorized into three SUA tertiles of 50 each. Cardiovascular risk parameters were compared using analysis of variance and chi-square tests. Pearson correlation and multivariable linear regression were performed.
Results: Mean SUA was 5.62±1.38 mg/dl. Across increasing SUA tertiles, systolic blood pressure increased from 123.26±10.89 to 132.62±11.16 mmHg, triglycerides from 124.83±38.99 to 165.78±34.29 mg/dl, and LDL-C from 104.58±19.48 to 129.48±20.29 mg/dl. HDL-C declined from 50.02±7.32 to 42.93±7.64 mg/dl. Hypertension increased from 20% to 58% ($p<0.001$). SUA correlated positively with systolic blood pressure, triglycerides, and LDL-C, and negatively with HDL-C.
Conclusions: Higher SUA levels were associated with clustering of adverse cardiovascular risk factors. SUA may serve as an accessible adjunctive marker of cardiometabolic risk.

Keywords: Cardiovascular risk, Dyslipidemia, Hypertension, Hyperuricemia, Serum uric acid

INTRODUCTION

Cardiovascular disease remains a major cause of morbidity, premature mortality, and healthcare burden worldwide. The development of CVD is multifactorial, with hypertension, diabetes mellitus, dyslipidemia, obesity, tobacco exposure, and physical inactivity representing established modifiable determinants. Identification of additional biochemical markers associated with clustering of these risk factors may facilitate earlier cardiovascular risk recognition.

Serum uric acid is the final metabolic product of purine degradation in humans and is generated principally through xanthine oxidoreductase-mediated oxidation of hypoxanthine and xanthine.[1] Although hyperuricemia has traditionally been considered primarily in relation to gout and urate nephrolithiasis, an expanding body of

epidemiological and experimental evidence has demonstrated associations between increased SUA concentrations and cardiovascular, renal and metabolic disorders.[1-3]

Hyperuricemia has been associated with hypertension, obesity, insulin resistance, metabolic syndrome, hypertriglyceridemia, chronic kidney disease and coronary artery disease.[2-5]

Consequently, interest has increased in the possibility that SUA may function as an additional marker of cardiometabolic risk.

Several mechanisms have been proposed to explain these associations. Increased xanthine oxidoreductase activity may promote generation of reactive oxygen species, while elevated intracellular uric acid may contribute to oxidative stress, reduced nitric oxide bioavailability, endothelial dysfunction, vascular smooth-muscle proliferation and activation of inflammatory pathways.[3,6] These changes may promote vascular dysfunction and contribute to the development of hypertension and atherosclerosis.

The relationship between uric acid and hypertension is particularly well documented. Hyperuricemia may promote renal microvascular injury, oxidative stress and activation of the renin-angiotensin system, whereas hypertension and renal impairment may themselves reduce urate elimination.[1,3] Grayson et al demonstrated in a systematic review and meta-analysis that hyperuricemia was independently associated with incident hypertension.[7] Similarly, Wang et al reported that hyperuricemia was associated with an approximately 48% higher adjusted risk of developing hypertension, with evidence of a dose-response relationship.[8]

A more recent dose-response meta-analysis involving 321,716 adults demonstrated that each 1 mg/dl increase in SUA was associated with an approximately 10% increase in the risk of incident hypertension.[9] These findings indicate that increasing SUA concentration may reflect a progressively adverse vascular risk profile.

Obesity and insulin resistance represent additional important determinants of SUA. Hyperinsulinemia reduces renal urate clearance, while excessive consumption of fructose and energy-dense foods may simultaneously promote obesity, hepatic lipogenesis and uric acid production.[3,10] This may explain the frequent coexistence of hyperuricemia, increased BMI, central obesity and metabolic syndrome.

Serum uric acid has also been linked with dyslipidemia. Elevated triglycerides, increased LDL cholesterol and reduced HDL cholesterol are commonly observed among individuals with higher SUA concentrations.[3,10] A study of healthy young and middle-aged adults demonstrated that SUA was positively associated with BMI, waist circumference, LDL cholesterol and triglycerides, and negatively associated with HDL cholesterol.[11]

In addition to individual risk factors, prospective studies have investigated SUA in relation to overt cardiovascular outcomes. A systematic review and meta-analysis demonstrated a modest but statistically significant independent association between hyperuricemia and coronary heart disease incidence and mortality.[12] A later dose-response meta-analysis involving nearly one million participants similarly reported higher risks of coronary morbidity and mortality with increasing SUA.[13]

Nevertheless, the causal nature of these relationships remains controversial. Uric acid has antioxidant properties in the extracellular environment, and SUA concentrations are strongly influenced by renal function, obesity, diet, alcohol consumption, insulin resistance, medications, and genetic factors.[2,4] Therefore, elevated SUA may act partly as a marker of an unfavorable metabolic phenotype rather than as an independent causal cardiovascular risk factor.

Evidence from India is particularly relevant because the population has a substantial burden of hypertension, diabetes, dyslipidemia, and central adiposity. Kanieeth et al conducted a population-based cross-sectional study among 2,976 Indian adults and demonstrated significantly higher cardiovascular risk-factor values among participants in the highest SUA tertile.[14] Their multivariable analysis supported a positive relationship between SUA and several metabolic risk factors.

More recent Indian evidence has similarly suggested that elevated SUA is associated with adverse cardiovascular-kidney-metabolic characteristics. These observations support further evaluation of SUA within Indian adult populations, particularly because it is inexpensive, widely available and routinely measured in clinical practice.

Aim

The present study was therefore undertaken to evaluate the association between SUA concentrations and established cardiovascular risk factors among adults. The study particularly examined whether increasing SUA levels were accompanied by progressive changes in BMI, blood pressure, glycemic status and lipid parameters.

METHODS

Study design and population

A cross-sectional observational study was conducted for 12 months (January 2025 to January 2026), among 150 adults aged ≥20 years attending, Owaisi Hospital and Research Centre.

Participants who fulfilled the eligibility criteria and provided written informed consent were enrolled consecutively until the required sample size was achieved.

Inclusion criteria

Adults aged ≥20 years of either sex who consented to participate and for whom complete clinical, anthropometric and biochemical assessments were available were included.

Exclusion criteria

Participants with known gout, severe chronic kidney disease, active malignancy, severe hepatic disease, pregnancy, or acute critical illness were excluded. Individuals receiving urate-lowering therapy were also excluded. Drugs known to substantially alter SUA were documented.

Clinical assessment

Demographic data including age and sex were recorded using a structured proforma. Relevant history regarding hypertension, diabetes mellitus, smoking, medication use and other cardiovascular risk factors was obtained.

Height was measured to the nearest 0.1 cm using a stadiometer, and body weight was recorded using a calibrated digital weighing scale. BMI was calculated as:

BMI = weight (kg)/height^2 (m^2).

Blood pressure measurement

Blood pressure was recorded after the participant had remained seated and rested for at least five minutes. An appropriate cuff size was used. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were recorded in mmHg.

Hypertension was defined according to documented hypertension, current use of antihypertensive medication or blood pressure values meeting the prespecified diagnostic criteria.

Biochemical assessment

Venous blood was obtained following an overnight fast. Laboratory investigations included: serum uric acid; fasting blood glucose; serum creatinine; total cholesterol; triglycerides; LDL cholesterol; and HDL cholesterol.

Serum uric acid was measured using an enzymatic uricase-based method and expressed in mg/dl.

Serum uric acid classification

Participants were classified into three equal SUA tertiles containing 50 participants each. tertile 1: 3.12-5.01 mg/dl, tertile 2: 5.04-6.21 mg/dl, tertile 3: 6.27-8.80 mg/dl.

This tertile-based approach was selected to evaluate trends in cardiovascular risk factors across the SUA distribution, consistent with previous population-based research.[14]

Assessment of cardiovascular risk factors

The principal cardiovascular risk variables evaluated were BMI, SBP, DBP, hypertension, fasting blood glucose, diabetes mellitus, total cholesterol, triglycerides, LDL-C, HDL-C and smoking.

Overweight/obesity was defined as BMI≥25 kg/m^2 for the principal categorical analysis. Hypertriglyceridemia was defined as triglycerides ≥150 mg/dl, elevated LDL-C as LDL-C ≥130 mg/dl, and low HDL-C as <40 mg/dl in men and <50 mg/dl in women.

Statistical analysis

Data were analysed using standard statistical software. Continuous variables were expressed as mean±standard deviation (SD), whereas categorical variables were reported as number and percentage. One-way analysis of variance (ANOVA) was used to compare continuous cardiovascular risk variables across SUA tertiles. Categorical variables were compared using the chi-square test. Pearson correlation analysis was performed to examine relationships between SUA and continuous cardiovascular risk parameters. Multivariable linear regression analysis was subsequently performed with SUA as the dependent variable. Age, sex, BMI, SBP, DBP, fasting glucose, triglycerides, LDL-C, HDL-C and serum creatinine were entered as potential explanatory variables. Regression coefficients were presented with 95% confidence intervals. A two-sided p value <0.05 was considered statistically significant.

Ethical considerations

The study was conducted in accordance with the principles of the Declaration of Helsinki. Institutional ethics committee approval was obtained before commencement of the study. Written informed consent was obtained from all participants, and confidentiality was maintained throughout.

RESULTS

A total of 150 adults were included. The mean age was 46.91±10.45 years, and 71 (47.3%) participants were male. The overall mean SUA concentration was 5.62±1.38 mg/dl. The mean BMI was 24.58±2.81 kg/m^2. Mean SBP and DBP were 127.95±12.10 mmHg and 80.35±7.42 mmHg, respectively. Hypertension was present in 57 (38.0%) participants, while 23 (15.3%) had diabetes mellitus.

Table 1: Baseline characteristics of the study population (n=150).

Parameters	Value (%)
Age (in years)	46.91±10.45
Gender	
Male	71 (47.3)
Female	79 (52.7)
BMI, kg/m^2	24.58±2.81
Systolic BP, mmHg	127.95±12.10
Diastolic BP, mmHg	80.35±7.42
Hypertension	57 (38.0)
Diabetes mellitus	23 (15.3)
Current smoking	23 (15.3)
Serum uric acid, mg/dl	5.62±1.38
Fasting blood glucose, mg/dl	100.84±15.42
Total cholesterol, mg/dl	188.46±24.63
Triglycerides, mg/dl	143.71±40.53
LDL-C, mg/dl	115.96±22.69
HDL-C, mg/dl	46.37±7.90
Serum creatinine, mg/dl	0.96±0.13

Table 2: Comparison of clinical parameters across serum uric acid tertiles.

Parameters	Tertile 1 (n=50)	Tertile 2 (n=50)	Tertile 3 (n=50)	P value
SUA, mg/dl	4.05±0.47	5.62±0.32	7.19±0.64	<0.001
Age (in years)	44.73±8.98	48.27±10.40	47.73±11.65	0.189
BMI, kg/m^2	23.85±3.20	24.69±2.77	25.22±2.26	0.048
SBP, mmHg	123.26±10.89	127.96±12.54	132.62±11.16	<0.001
DBP, mmHg	75.72±6.48	82.05±6.53	83.28±7.02	<0.001
Fasting glucose, mg/dl	99.32±15.69	101.17±14.86	102.03±15.88	0.671

There was no significant difference in age or fasting glucose across SUA tertiles.

Table 3: Lipid parameters across serum uric acid tertiles.

Parameters	Tertile 1 (n=50)	Tertile 2 (n=50)	Tertile 3 (n=50)	P value
Total cholesterol, mg/dl	184.00±23.57	183.79±24.24	197.59±23.94	0.005
Triglycerides, mg/dl	124.83±38.99	140.51±37.79	165.78±34.29	<0.001
LDL-C, mg/dl	104.58±19.48	113.82±21.23	129.48±20.29	<0.001
HDL-C, mg/dl	50.02±7.32	46.17±7.24	42.93±7.64	<0.001

Table 4: Cardiovascular risk-factor prevalence according to SUA tertile.

Risk factors	Tertile 1 N (%)	Tertile 2 N (%)	Tertile 3 N (%)	P value
Male gender	24 (48)	21 (42)	26 (52%)	0.602
BMI ≥25 kg/m^2	19 (38)	19 (38)	27 (54%)	0.176
Hypertension	10 (20)	18 (36)	29 (58%)	<0.001
Diabetes mellitus	5 (10)	6 (12)	12 (24%)	0.110
Hypertriglyceridemia	12 (24)	20 (40)	35 (70%)	<0.001
Elevated LDL-C	5 (10)	9 (18)	26 (52%)	<0.001
Low HDL-C	15 (30)	24 (48)	29 (58%)	0.017
Current smoking	7 (14)	8 (16)	8 (16%)	0.950

Cardiovascular risk parameters across SUA tertiles

Significant differences were observed in several cardiovascular risk variables across increasing SUA tertiles.

Mean BMI increased progressively from 23.85±3.20 kg/m^2 in tertile 1 to 25.22±2.26 kg/m^2 in tertile 3 (p=0.048).

Mean SBP increased from 123.26±10.89 mmHg in tertile 1 to 132.62±11.16 mmHg in tertile 3 (p<0.001). A similar trend was observed for DBP, which increased from 75.72±6.48 mmHg to 83.28±7.02 mmHg (p<0.001).

Association between SUA and lipid parameters

A clear deterioration in the lipid profile was observed with increasing SUA levels. Mean triglyceride concentration increased from 124.83±38.99 mg/dl in tertile 1 to 165.78±34.29 mg/dl in tertile 3 (p<0.001).

LDL-C increased from 104.58±19.48 mg/dL to 129.48±20.29 mg/dL (p<0.001), whereas HDL-C decreased from 50.02±7.32 mg/dl to 42.93±7.64 mg/dl (p<0.001).

Prevalence of cardiovascular risk factors

The prevalence of hypertension increased progressively from 20.0% in the lowest SUA tertile to 58.0% in the highest tertile (p<0.001).

Hypertriglyceridemia was present in 24.0%, 40.0% and 70.0% of participants across tertiles 1, 2 and 3, respectively (p<0.001).

Similarly, elevated LDL-C increased from 10.0% to 52.0% (p<0.001), while low HDL-C increased from 30.0% to 58.0% (p=0.017).

Correlation between SUA and cardiovascular risk parameters

SUA demonstrated a significant positive correlation with SBP (r=0.318, p<0.001), DBP (r=0.405, p<0.001), total cholesterol (r=0.238, p=0.003), triglycerides (r=0.398, p<0.001) and LDL-C (r=0.434, p<0.001).

Table 5: Correlation of serum uric acid with cardiovascular risk parameters.

Variables	Pearson r	P value
Age	0.115	0.162
BMI	0.155	0.058
Systolic BP	0.318	<0.001
Diastolic BP	0.405	<0.001
Fasting glucose	0.093	0.259
Total cholesterol	0.238	0.003
Triglycerides	0.398	<0.001
LDL-C	0.434	<0.001
HDL-C	−0.402	<0.001
Serum Creatinine	0.357	<0.001

A significant negative correlation was observed between SUA and HDL-C (r=−0.402, p<0.001).

The correlation with BMI was positive but did not reach conventional statistical significance (r=0.155, p=0.058). SUA was not significantly correlated with fasting glucose.

Multivariable analysis

Multivariable linear regression demonstrated that DBP, triglycerides, LDL-C, HDL-C and serum creatinine remained independently associated with SUA after simultaneous adjustment for age, sex, BMI, SBP and fasting glucose.

The overall model explained approximately 50.6% of the variation in SUA (R^2=0.506; adjusted R^2=0.470; model p<0.001).

Table 6: Multivariable linear regression for factors associated with serum uric acid.

Variable	β coefficient	95% CI	P value
Age (in years)	−0.004	−0.021 to 0.012	0.604
Male Gender	−0.194	−0.546 to 0.158	0.278
BMI, kg/m^2	0.037	−0.023 to 0.098	0.224
Systolic BP	0.012	−0.003 to 0.026	0.108
Diastolic BP	0.045	0.021 to 0.068	<0.001
Fasting glucose	0.007	−0.004 to 0.018	0.240
Triglycerides	0.006	0.001 to 0.010	0.011
LDL-C	0.015	0.008 to 0.023	<0.001
HDL-C	−0.043	−0.065 to −0.021	<0.001
Serum creatinine	2.493	1.103 to 3.882	<0.001

DISCUSSION

The present study evaluated the association between SUA concentrations and established cardiovascular risk factors among 150 adults. Increasing SUA was accompanied by a progressively less favourable cardiovascular risk profile, particularly with respect to blood pressure and lipid parameters. Participants in the highest SUA tertile demonstrated significantly higher SBP, DBP, total cholesterol, triglycerides and LDL-C, together with significantly lower HDL-C. Furthermore, hypertension, hypertriglyceridemia, elevated LDL-C and low HDL-C were substantially more prevalent in the highest SUA tertile.

These findings are broadly consistent with those reported by Kanieeth et al in a population-based study of 2,976 Indian adults.[14] Their study demonstrated significantly higher cardiovascular risk-factor values in participants belonging to the highest SUA tertile, and multivariable analyses showed positive associations between SUA and several metabolic risk variables. The present findings therefore support the concept that SUA may reflect the clustering of cardiovascular and metabolic abnormalities in Indian adults.

The overall mean SUA in the present study was 5.62±1.38 mg/dl. Participants in the highest tertile had a mean SUA of 7.19±0.64 mg/dl compared with 4.05±0.47 mg/dl in the lowest tertile. The use of tertiles rather than a single threshold allowed evaluation of graded changes in cardiovascular risk variables across the SUA distribution.

One of the most prominent findings was the association between SUA and blood pressure. Mean SBP increased from 123.26±10.89 mmHg in tertile 1 to 132.62±11.16 mmHg in tertile 3, while DBP increased from 75.72±6.48 mmHg to 83.28±7.02 mmHg. Correspondingly, hypertension prevalence increased from 20% to 58%.

Correlation analysis further demonstrated significant positive relationships between SUA and both SBP and DBP. These findings are consistent with previous evidence suggesting a relationship between uric acid and hypertension. Grayson et al reported a 41% greater adjusted risk of incident hypertension among individuals with hyperuricemia.[7] Wang et al subsequently reported an adjusted relative risk of 1.48 for incident hypertension in individuals with hyperuricemia.[8]

A later dose-response meta-analysis involving 321,716 adults reported a pooled relative risk of approximately 1.10 for incident hypertension for each 1 mg/dL increase in SUA.[9] Collectively, these observations support the presence of a graded relationship between uric acid concentrations and blood pressure.

Potential mechanisms include oxidative stress, reduction in endothelial nitric oxide bioavailability, vascular smooth-muscle proliferation, activation of the renin-angiotensin system and renal microvascular injury.[1,3,6] These mechanisms may result in increased systemic vascular resistance and contribute to the initiation or maintenance of hypertension.

The lipid findings were similarly noteworthy. Triglycerides increased from 124.83±38.99 mg/dl in the lowest SUA tertile to 165.78±34.29 mg/dl in the highest, while LDL-C increased from 104.58±19.48 to 129.48±20.29 mg/dl. Conversely, HDL-C declined progressively from 50.02±7.32 to 42.93±7.64 mg/dl.

SUA demonstrated significant positive correlations with triglycerides and LDL-C and a significant inverse correlation with HDL-C. These findings suggest that elevated SUA frequently occurs in conjunction with an atherogenic lipid phenotype.

Similar findings have been reported in previous studies. Healthy young and middle-aged adults with higher SUA concentrations have been shown to exhibit greater adiposity, increased LDL-C and triglycerides and reduced HDL-C.[11] A recent Indian hospital-based study among adults with essential hypertension similarly found higher triglycerides and LDL-C and lower HDL-C among participants with hyperuricemia.[15]

Insulin resistance may provide a common mechanistic link between hyperuricemia and dyslipidemia. Hyperinsulinemia can reduce renal urate clearance, while hepatic insulin resistance promotes increased triglyceride production.[3,10] Furthermore, fructose metabolism can increase both hepatic lipogenesis and uric acid generation, potentially contributing to the simultaneous development of hypertriglyceridemia and hyperuricemia.

BMI increased from 23.85±3.20 kg/m^2 in the lowest tertile to 25.22±2.26 kg/m^2 in the highest tertile, with an overall significant difference across tertiles. Nevertheless, the correlation between SUA and BMI was relatively weak and narrowly failed to reach conventional statistical significance. This suggests that adiposity may contribute to the SUA-associated cardiometabolic phenotype but may not fully explain it within this sample.

Obesity and SUA are biologically closely related. Adiposity is commonly accompanied by insulin resistance and hyperinsulinemia, which reduce renal urate excretion. Increased dietary purine and fructose consumption may additionally raise uric acid concentrations.[3,10] The relatively modest association observed in the present study may reflect the limited sample size, BMI distribution or the multifactorial determinants of SUA.

Interestingly, fasting blood glucose did not differ significantly across SUA tertiles and showed no significant linear correlation with SUA. Diabetes prevalence was numerically higher in the highest tertile but the difference did not achieve statistical significance.

The relationship between SUA and glycemia is complex. Kanieeth et al reported an inverse relationship between increasing SUA and HbA1c particularly among participants with established or newly diagnosed diabetes.[14] Their observation suggests that the relationship between uric acid and glucose metabolism may not be uniformly positive across different stages of metabolic disease.

Renal handling of urate, glycosuria, insulin concentrations and medication exposure may modify the SUA-glycemia relationship. Consequently, the absence of a significant association between fasting glucose and SUA in the present study does not contradict the broader association between uric acid and cardiometabolic risk.

Another relevant finding was the positive relationship between SUA and serum creatinine. Creatinine remained independently associated with SUA in multivariable analysis. This is physiologically plausible because renal excretion is a principal route of urate elimination. Reduced renal clearance may therefore increase SUA concentrations even before clinically overt renal impairment becomes apparent. The National Kidney Foundation scientific workshop has highlighted the complex interrelationship between hyperuricemia, renal disease, hypertension and cardiovascular disease.[16]

Multivariable regression demonstrated that DBP, triglycerides, LDL-C, HDL-C and serum creatinine remained independently associated with SUA. The model explained approximately 50.6% of the variation in SUA concentrations. These findings suggest that the association between SUA and cardiovascular risk is not attributable to a single risk factor but rather reflects an interconnected metabolic and vascular phenotype.

The observed associations are clinically relevant because prospective evidence has linked hyperuricemia not only with cardiovascular risk factors but also with overt coronary outcomes. Kim et al reported modest independent increases in coronary heart disease incidence and mortality associated with hyperuricemia.[12] Li et al, in a larger dose-response meta-analysis, similarly reported increased coronary morbidity and mortality with elevated SUA.[13]

Nevertheless, these epidemiological relationships should not be interpreted as proof that uric acid itself is an independent causal cardiovascular risk factor. Ndrepepa highlighted that elevated SUA may contribute to oxidative stress, endothelial dysfunction, inflammation and metabolic dysregulation, but also emphasised that causality remains unproven.[2]

Similarly, Borghi et al. noted that hyperuricemia is closely associated with cardiovascular, metabolic and renal disorders and may exert pro-inflammatory, pro-oxidative and vasoconstrictive effects.[4] However, the extent to which pharmacological reduction of SUA improves cardiovascular outcomes in individuals without gout remains uncertain.

Accordingly, SUA may presently be most useful as an easily obtainable biochemical marker identifying individuals with an adverse cardiometabolic profile rather than as a stand-alone therapeutic target for cardiovascular prevention. Patients with elevated SUA may warrant more careful evaluation of blood pressure, body weight, lipid profile, glycemic status and renal function.

The present study has several limitations. First, its cross-sectional design does not permit determination of temporality or causation. Secondly, the relatively modest sample size of 150 participants limits subgroup analyses and may reduce the ability to detect weaker associations. Thirdly, dietary purine intake, fructose consumption, alcohol intake, physical activity, socioeconomic factors and genetic determinants were not comprehensively evaluated.

Fourthly, although serum creatinine was included in the multivariable analysis, more detailed evaluation of renal function using estimated glomerular filtration rate would strengthen interpretation. Finally, the study was conducted at a single centre and therefore may not be fully representative of the broader population.

Despite these limitations, the study demonstrates a clear clustering of cardiovascular risk factors with increasing SUA concentrations. The most consistent relationships involved blood pressure, triglycerides, LDL-C and HDL-C, with several associations persisting following multivariable adjustment. These observations reinforce the potential value of SUA as an adjunctive marker of cardiovascular and metabolic risk.

CONCLUSION

Higher serum uric acid concentrations were associated with an unfavourable cardiovascular risk profile among adults. Increasing SUA tertiles were accompanied by significantly higher blood pressure, total cholesterol, triglycerides and LDL-C and lower HDL-C. The prevalence of hypertension and atherogenic lipid abnormalities also increased substantially across SUA tertiles.

Serum uric acid demonstrated positive correlations with blood pressure, triglycerides and LDL-C and an inverse relationship with HDL-C. Several of these associations remained significant in multivariable analysis.

These findings suggest that SUA may serve as an inexpensive and readily accessible adjunctive marker for identifying individuals with clustering of cardiovascular risk factors. However, because of the cross-sectional nature of the study, causality cannot be established. Larger prospective studies are required to determine the independent prognostic significance of SUA and whether

modification of uric acid levels translates into improved cardiovascular outcomes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Feig DI, Kang DH, Johnson RJ. Uric acid and cardiovascular risk. N Engl J Med. 2008;359(17):1811-21.
2. Ndrepepa G. Uric acid and cardiovascular disease. Clin Chim Acta. 2018;484:150-63.
3. Sharaf El Din UAA, Salem MM, Abdulazim DO. Uric acid in the pathogenesis of metabolic, renal, and cardiovascular diseases: a review. J Adv Res. 2017;8(5):537-48.
4. Borghi C, Agabiti-Rosei E, Johnson RJ, Kielstein JT, Lurbe E, Mancia G, et al. Hyperuricemia and gout in cardiovascular, metabolic and kidney disease. Eur J Intern Med. 2020;80:1-11.
5. Kanbay M, Segal M, Afsar B, Kang DH, Rodriguez-Iturbe B, Johnson RJ. The role of uric acid in the pathogenesis of human cardiovascular disease. Heart. 2013;99(11):759-66.
6. Puddu P, Puddu GM, Cravero E, Vizioli L, Muscari A. Relationships among hyperuricemia, endothelial dysfunction and cardiovascular disease: molecular mechanisms and clinical implications. J Cardiol. 2012;59(3):235-42.
7. Grayson PC, Kim SY, LaValley M, Choi HK. Hyperuricemia and incident hypertension: a systematic review and meta-analysis. Arth Care Res. 2011;63(1):102-10.
8. Wang J, Qin T, Chen J, Li Y, Wang L, Huang H, et al. Hyperuricemia and risk of incident hypertension: a systematic review and meta-analysis of observational studies. PLoS One. 2014;9(12).
9. Tian X, Wang A, Zuo Y, Chen S, Zhang L, Wu S, et al. Dose-response association between serum uric acid levels and incident hypertension: a systematic review and meta-analysis of 17 prospective cohort studies of 32 thousand participants. Acta Cardiol. 2021;76(7):748-57.
10. Soltani Z, Rasheed K, Kapusta DR, Reisin E. Potential role of uric acid in metabolic syndrome, hypertension, kidney injury, and cardiovascular diseases: is it time for reappraisal? Curr Hypertens Rep. 2013;15(3):175-81.
11. Cicero AFG, Fogacci F, Giovannini M, Grandi E, D'Addato S, Borghi C. Serum uric acid levels are associated with cardiometabolic risk factors in healthy young and middle-aged adults. Clin Exp Hypertens. 2018;40:1-7.
12. Kim SY, Guevara JP, Kim KM, Choi HK, Heitjan DF, Albert DA. Hyperuricemia and coronary heart disease: a systematic review and meta-analysis. Arth Care Res. 2010;62(2):170-80.
13. Li M, Hu X, Fan Y, Li K, Zhang X, Hou W, et al. Hyperuricemia and the risk for coronary heart disease morbidity and mortality: a systematic review and dose-response meta-analysis. Sci Rep. 2016;6:19520.
14. Kanieeth D, Swaminathan K, Velmurugan G, Ramakrishnan A, Alexander T, Raghupathy AK, et al. Association between serum uric acid levels and cardiovascular risk factors among adults in India. Nutr Metab Cardiovasc Dis. 2023;33(7):1330-8.
15. Suyambulingam A, Arun K, Venkatesan A, Jayakaran V, Mathisha EP, Anbalagan S, et al. From purines to plaques: serum uric acid as a cardiometabolic marker and its correlation with dyslipidemia in essential hypertension. Cureus. 2025;17(9).
16. 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 organised by the National Kidney Foundation. Am J Kidney Dis. 2018;71(6):851-65.

Cite this article as: Mala HR, Waqas M, Junejo H, Ansari Z, Siddiqua SA. Association between serum uric acid levels and cardiovascular risk factors in adults. Int J Res Med Sci 2026;14:5768-75.