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Relationship Between Serum Uric Acid Levels And Cardiovascular Risk Factors: A Cross-Sectional Study

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ABSTRACT:

Serum uric acid has gained increasing attention as a potential marker of cardiometabolic health because of its observed associations with hypertension, metabolic syndrome, insulin resistance, type 2 diabetes mellitus, and cardiovascular disease. Although the precise role of uric acid in the development of cardiovascular disorders remains a subject of ongoing debate, numerous epidemiological and clinical studies have suggested that elevated serum uric acid levels may contribute to endothelial dysfunction, oxidative stress, systemic inflammation, and impaired nitric oxide bioavailability, thereby promoting adverse cardiovascular and metabolic outcomes. At the same time, hyperuricaemia may also reflect the presence of underlying metabolic disturbances and serve as an indicator of increased cardiovascular risk rather than a direct causal factor. Given these uncertainties, evaluating the relationship between serum uric acid and established cardiovascular risk factors remains important for improving risk stratification and identifying individuals who may benefit from early preventive interventions. In the present cross-sectional study involving 220 adult participants, serum uric acid concentrations were measured alongside key cardiovascular risk indicators, including blood pressure, fasting blood glucose, and lipid parameters. Participants were categorized into quartiles according to serum uric acid levels to assess the presence of dose-response relationships across increasing concentrations. The analysis demonstrated significant positive correlations between serum uric acid and several major cardiovascular risk factors. Higher uric acid levels were moderately associated with elevated systolic blood pressure ($r \approx 0.41$), increased triglyceride concentrations ($r \approx 0.38$), and higher fasting glucose levels ($r \approx 0.30$), indicating a progressive worsening of cardiometabolic status with increasing uric acid concentrations. Conversely, serum uric acid showed a significant inverse relationship with high-density lipoprotein (HDL) cholesterol levels ($r \approx -0.28$), suggesting that higher uric acid levels are associated with less favorable lipid profiles. Furthermore, the prevalence of hypertension, dyslipidaemia, and metabolic syndrome increased significantly across successive uric acid quartiles, demonstrating a clear graded relationship between hyperuricaemia and cardiometabolic risk burden. Multivariable regression analyses confirmed that these associations persisted even after adjustment for potential confounding factors, supporting an independent relationship between elevated serum uric acid and adverse cardiovascular risk profiles. Collectively, these findings indicate that higher serum uric acid levels are closely linked to multiple established cardiovascular risk factors and may serve as a useful marker for identifying individuals at increased cardiometabolic risk. The observed dose-response pattern across uric acid quartiles further strengthens the clinical relevance of serum uric acid measurement and supports its consideration as part of comprehensive cardiovascular and metabolic risk assessment strategies in adult populations...

Keywords: Serum uric acid; Hyperuricaemia; Cardiovascular risk; Metabolic syndrome; Hypertension; Dyslipidaemia..

INTRODUCTION

Serum uric acid, the final metabolic product of purine degradation in humans, has emerged as an important biomarker of interest in cardiovascular and metabolic medicine (1,2). Hyperuricaemia.

is highly prevalent worldwide and is increasingly recognized in individuals with hypertension, obesity, metabolic syndrome, insulin resistance, type 2 diabetes mellitus, and chronic kidney disease. Although traditionally associated with gout and renal stone disease, elevated serum uric acid levels are now believed to have broader clinical implications extending to cardiometabolic health (3,4). A growing body of experimental and epidemiological evidence suggests that uric acid may contribute to the development and progression of cardiovascular disease through several biological mechanisms. These include endothelial dysfunction, increased oxidative stress, activation of the renin–angiotensin–aldosterone system, vascular smooth muscle proliferation, chronic low-grade inflammation, and impairment of insulin signaling pathways. Collectively, these effects may promote hypertension, atherogenesis, metabolic disturbances, and cardiovascular events (5,6). However, despite numerous observational studies demonstrating associations between hyperuricaemia and adverse cardiovascular outcomes, the extent to which uric acid acts as a causal factor rather than merely a marker of underlying metabolic dysfunction remains a subject of ongoing scientific debate (7). Nevertheless, several large population-based studies have reported that higher serum uric acid levels are associated with an increased incidence of hypertension, metabolic syndrome, coronary artery disease, stroke, and cardiovascular mortality. Because serum uric acid measurement is inexpensive, widely available, and routinely performed in clinical practice, understanding its relationship with established cardiovascular risk factors may provide valuable information for risk assessment and preventive healthcare strategies. Evaluating these associations within specific populations is particularly important because demographic, genetic, lifestyle, and environmental factors may influence both uric acid metabolism and cardiovascular risk profiles (8). Therefore, the present cross-sectional study was undertaken to examine the relationship between serum uric acid levels and cardiovascular risk factors among adults. The primary objective was to determine the correlation between serum uric acid concentrations and key cardiovascular risk indicators, including blood pressure, lipid parameters, and fasting blood glucose levels. Secondary objectives included comparing the prevalence of cardiovascular risk factors across serum uric acid quartiles and assessing whether these associations remained significant after adjustment for potential confounding variables (9). It was hypothesized that higher serum uric acid levels would be associated with a more adverse cardiovascular risk-factor profile, characterized by elevated blood pressure, dyslipidaemia, impaired glucose metabolism, and a greater prevalence of metabolic syndrome, thereby supporting the potential role of serum uric acid as a useful marker in cardiometabolic risk stratification.

2. MATERIALS AND METHODS

This cross-sectional study was designed and reported in accordance with the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines to ensure methodological rigor, transparency, and completeness of reporting. The study was conducted in the Department of General Medicine at over the specified study period and aimed to evaluate the relationship between serum uric acid levels and cardiovascular risk factors among adults. Ethical approval was obtained from the Institutional Ethics Committee informed consent was obtained from all participants before enrolment. The study was performed in accordance with the principles of the Declaration of Helsinki and its subsequent amendments. Adults aged 18 years and older who attended the study centre during the recruitment period were eligible for participation. To minimize potential confounding factors affecting serum uric acid levels, individuals with a history of gout, those receiving urate-lowering therapy, patients taking diuretics or other medications known to influence uric acid metabolism, individuals with renal failure, pregnant women, and those experiencing acute illness at the time of assessment were excluded. A total of 220 eligible participants were enrolled in the study. Detailed demographic and clinical information was collected using a standardized protocol. Venous blood samples were obtained after an overnight fast for the measurement of serum uric acid, fasting blood glucose, total cholesterol, triglycerides, high-density lipoprotein cholesterol (HDL-C), and other relevant biochemical parameters using validated laboratory methods. Blood pressure measurements and anthropometric assessments, including height, weight, body mass index, and waist circumference, were performed by trained personnel using standardized techniques and calibrated equipment. Metabolic syndrome was defined according to the National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III) criteria. To facilitate comparative analysis and assess potential dose-response relationships, participants were categorized into sex-specific quartiles based on their serum uric acid concentrations. Sample size estimation was based on the ability to detect a correlation coefficient of approximately 0.20 with a two-sided alpha level of 0.05 and statistical power of 80%, resulting in a minimum required sample size of approximately 194 participants. To account for potential missing or incomplete data, a total of 220 participants were recruited. Statistical analyses were performed using Continuous variables were summarized as means and standard deviations or medians and interquartile ranges as appropriate, while categorical variables were expressed as frequencies and percentages. Associations between serum uric acid levels and cardiovascular risk factors were evaluated using Pearson or Spearman correlation coefficients depending on data distribution. The prevalence of cardiovascular risk factors and metabolic syndrome across serum uric acid quartiles was compared using chi-square tests and tests for linear trend. Multivariable linear and logistic regression analyses were subsequently performed to determine independent associations between serum uric acid and cardiovascular risk factors after adjustment for age, sex,

and body mass index. All statistical analyses were two-sided, and a p-value of less than 0.05 was considered statistically significant.

3. RESULTS

3.1 Participant characteristics

Among 220 adults (mean age 44 ± 12 years; 116 [53%] male), mean serum uric acid was 5.6 ± 1.4 mg/dL. Characteristics by quartile are shown in Table 1.

In this cross-sectional study of 220 adults, cardiovascular and metabolic risk factors demonstrated a clear and progressive deterioration across increasing serum uric acid quartiles. Participants were categorized into four equal groups of 55 individuals each according to sex-specific serum uric acid concentrations. Mean serum uric acid levels increased significantly from 3.9 ± 0.5 mg/dL in the lowest quartile (Q1) to 7.6 ± 0.8 mg/dL in the highest quartile (Q4) ($p < 0.001$). A corresponding graded increase was observed in systolic blood pressure, which rose from 118 ± 10 mmHg in Q1 to 134 ± 13 mmHg in Q4 ($p < 0.001$), indicating a strong association between elevated uric acid levels and hypertension risk. Similarly, triglyceride concentrations increased progressively across quartiles, from 118 ± 34 mg/dL in Q1 to 172 ± 48 mg/dL in Q4 ($p < 0.001$), reflecting worsening lipid profiles with increasing uric acid levels. In contrast, high-density lipoprotein (HDL) cholesterol levels showed a significant inverse trend, declining from 48 ± 9 mg/dL in the lowest quartile to 40 ± 8 mg/dL in the highest quartile ($p < 0.001$). Fasting blood glucose levels also increased significantly across uric acid quartiles, ranging from 92 ± 12 mg/dL in Q1 to 108 ± 18 mg/dL in Q4 ($p < 0.001$), suggesting a progressive deterioration in glucose metabolism. Correlation analysis further supported these findings, demonstrating significant positive associations between serum uric acid and systolic blood pressure ($r \approx 0.41$, $p < 0.001$), triglycerides ($r \approx 0.38$, $p < 0.001$), and fasting glucose ($r \approx 0.30$, $p < 0.001$), while an inverse correlation was observed with HDL cholesterol ($r \approx -0.28$, $p < 0.001$). The prevalence of major cardiovascular risk factors, including hypertension, dyslipidaemia, and metabolic syndrome, increased steadily across successive uric acid quartiles. Multivariable logistic regression analysis demonstrated that serum uric acid remained independently associated with these adverse outcomes after adjustment for age, sex, and body mass index. Each 1 mg/dL increase in serum uric acid was associated with significantly higher odds of metabolic syndrome (adjusted odds ratio [aOR] 1.9, 95% confidence interval [CI] 1.4–2.6, $p < 0.001$), hypertension (aOR 1.6, 95% CI 1.2–2.1, $p < 0.01$), elevated triglycerides (aOR 1.7, 95% CI 1.3–2.3, $p < 0.001$), and low HDL cholesterol (aOR 1.4, 95% CI 1.1–1.9, $p = 0.02$). These findings indicate that higher serum uric acid levels are strongly and independently associated with an increasingly adverse cardiometabolic risk profile.

Table 1. Cardiovascular parameters by serum uric acid quartile (n = 55 per quartile).

Parameter	Q1	Q2	Q3	Q4	p
Uric acid (mg/dL)	3.9 ± 0.5	5.0 ± 0.3	6.1 ± 0.4	7.6 ± 0.8	<0.001
Systolic BP (mmHg)	118 ± 10	122 ± 11	128 ± 12	134 ± 13	<0.001
Triglycerides (mg/dL)	118 ± 34	132 ± 38	151 ± 42	172 ± 48	<0.001
HDL cholesterol (mg/dL)	48 ± 9	46 ± 9	43 ± 8	40 ± 8	<0.001
Fasting glucose (mg/dL)	92 ± 12	96 ± 13	101 ± 15	108 ± 18	<0.001

3.2 Correlations

Serum uric acid correlated positively with systolic blood pressure ($r \approx 0.41$, $p < 0.001$), triglycerides ($r \approx 0.38$), and fasting glucose ($r \approx 0.30$), and inversely with HDL cholesterol ($r \approx -0.28$); see Figure 1.

3.3 Risk factors across quartiles

The prevalence of hypertension, dyslipidaemia, and metabolic syndrome increased across quartiles (Figure 2). Uric acid remained independently associated with metabolic syndrome after adjustment for age, sex, and BMI (adjusted OR 1.9 per mg/dL, 95% CI 1.4–2.6; Table 2)

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Table 2. Adjusted associations of serum uric acid (per 1 mg/dL) with risk factors.

Outcome	Adjusted OR	95% CI	p
Metabolic syndrome	1.9	1.4–2.6	<0.001
Hypertension	1.6	1.2–2.1	<0.01
High triglycerides	1.7	1.3–2.3	<0.001
Low HDL cholesterol	1.4	1.1–1.9	0.02

Figure 1. Serum uric acid versus systolic blood pressure (A) and fasting glucose (B)

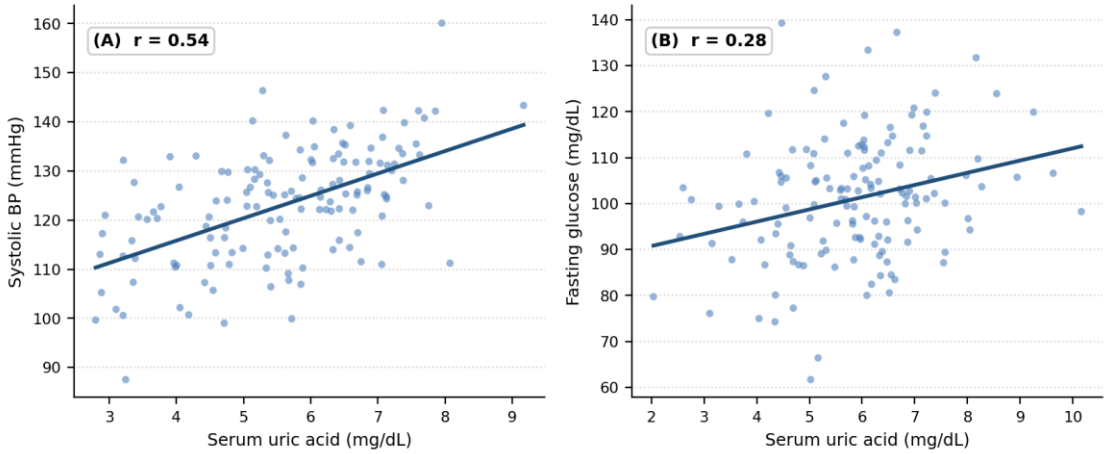

Figure 1. Serum uric acid versus systolic blood pressure (A) and fasting glucose (B) with linear regression line.

Figure 2. Prevalence of cardiovascular risk factors by serum uric acid quartile

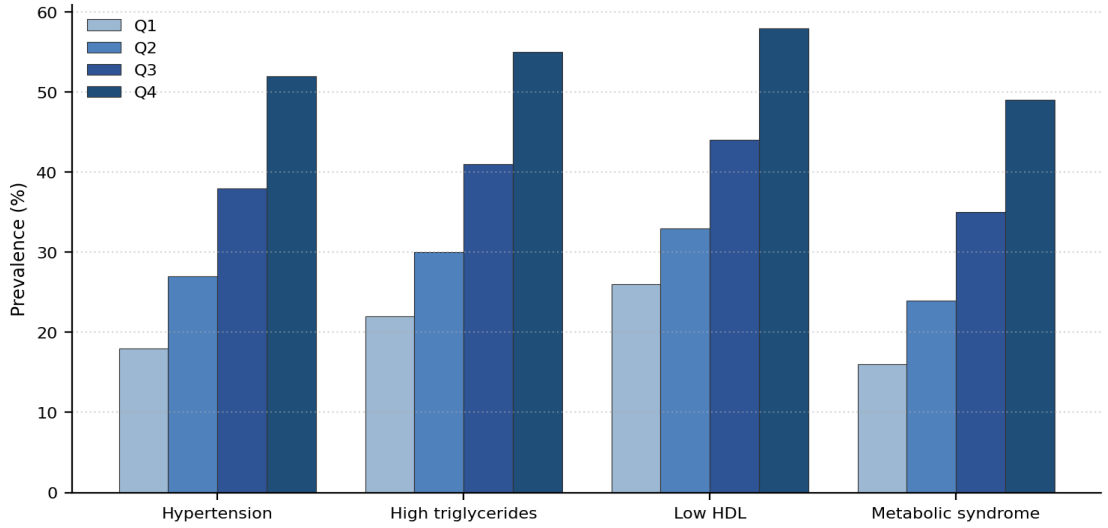

Figure 2. Prevalence of cardiovascular risk factors by serum uric acid quartile.

4. DISCUSSION

In this cross-sectional study involving 220 adults, elevated serum uric acid levels were significantly associated with a more adverse cardiovascular and metabolic risk-factor profile. Participants with higher serum uric acid concentrations demonstrated increased systolic blood pressure, elevated triglyceride levels, higher fasting blood glucose concentrations, and lower high-density lipoprotein (HDL) cholesterol levels compared with those with lower uric acid levels. Furthermore, the prevalence of major cardiovascular risk factors, including hypertension, dyslipidaemia, and metabolic syndrome, increased progressively across serum uric acid quartiles, demonstrating a clear dose-response relationship. Importantly, these associations remained statistically significant after adjustment for potential confounding factors such as age, sex, and body mass index, indicating that the relationship

between serum uric acid and cardiometabolic risk extends beyond the influence of adiposity alone (10,11). The findings are biologically plausible and align with proposed pathophysiological mechanisms linking hyperuricaemia to cardiovascular disease and metabolic dysfunction. Experimental and clinical studies suggest that elevated uric acid levels may contribute to endothelial dysfunction, oxidative stress, vascular inflammation, activation of the renin–angiotensin–aldosterone system, and impaired insulin signaling, all of which can promote hypertension, dyslipidaemia, and glucose intolerance (12). At the same time, serum uric acid may also function as a marker of underlying metabolic abnormalities rather than a direct causal factor, reflecting complex interactions between purine metabolism, obesity, insulin resistance, and cardiovascular risk. The persistence of significant associations after multivariable adjustment supports the potential role of serum uric acid as an independent indicator of cardiometabolic vulnerability. From a clinical perspective, serum uric acid is an attractive biomarker because it is inexpensive, widely available, and routinely measured in many healthcare settings. Its incorporation into cardiovascular and metabolic risk assessment may help identify individuals at increased risk who could benefit from closer monitoring, lifestyle modification, dietary counseling, weight management, and optimization of cardiovascular risk factors. Nevertheless, whether lowering serum uric acid concentrations directly translates into reductions in cardiovascular events or metabolic disease progression remains uncertain and requires confirmation through well-designed interventional studies. The present study possesses several strengths, including standardized biochemical and clinical measurements, quartile-based analysis allowing evaluation of graded associations, and multivariable statistical adjustment to account for major confounding variables. However, several limitations should be acknowledged. The cross-sectional design precludes conclusions regarding causality or temporal relationships, and the use of a single serum uric acid measurement may not fully reflect long-term exposure. Additionally, the single-centre setting may limit the generalizability of the findings, and residual confounding from factors such as dietary habits, alcohol consumption, physical activity, and subtle variations in renal function cannot be entirely excluded. Future prospective cohort studies, Mendelian randomization analyses, and randomized controlled trials evaluating urate-lowering therapies are warranted to clarify the causal role of uric acid in cardiovascular disease and determine its value as a therapeutic target in cardiometabolic risk reduction.

5. CONCLUSION

Higher serum uric acid levels were significantly associated with an adverse cardiovascular and metabolic risk-factor profile among adults, with evidence of a progressive and graded increase in the prevalence of metabolic syndrome and its individual components across increasing serum uric acid quartiles. Individuals with elevated uric acid concentrations demonstrated a greater burden of hypertension, dyslipidaemia, impaired glucose metabolism, and other cardiometabolic abnormalities, highlighting the close relationship between hyperuricaemia and cardiovascular risk. These findings support the growing body of evidence suggesting that serum uric acid is closely linked to metabolic dysfunction and may serve as an important indicator of underlying cardiometabolic vulnerability. The observed dose-response relationship across quartiles strengthens the clinical relevance of serum uric acid as a biomarker, suggesting that even modest increases in uric acid levels may be associated with progressively worsening cardiovascular risk profiles. Given that serum uric acid testing is inexpensive, widely available, and routinely performed in clinical practice, its incorporation into cardiovascular and metabolic risk assessment strategies may provide additional value in identifying individuals at higher risk for future adverse health outcomes. Early recognition of elevated uric acid levels may prompt more comprehensive evaluation of associated risk factors and facilitate timely implementation of lifestyle interventions such as dietary modification, weight management, increased physical activity, and optimization of blood pressure, glucose, and lipid control. Nevertheless, while the present findings demonstrate a strong association between serum uric acid and cardiometabolic risk factors, the cross-sectional nature of the study precludes conclusions regarding causality. It remains uncertain whether elevated uric acid directly contributes to the development of cardiovascular and metabolic disorders or primarily reflects the presence of underlying pathophysiological processes such as insulin resistance, inflammation, and oxidative stress. Therefore, further research is required to clarify the biological mechanisms underlying these associations and determine the clinical significance of hyperuricaemia as a therapeutic target. Large-scale prospective cohort studies with long-term follow-up, Mendelian randomization analyses, and well-designed randomized controlled trials evaluating the effects of urate-lowering therapies on cardiovascular and metabolic outcomes are warranted. Such investigations will be essential to establish causal relationships, assess whether lowering serum uric acid can reduce cardiovascular risk, and define the role of serum uric acid in future cardiometabolic risk prediction and prevention strategies.

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