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Original Research Article

Correlation between Serum Uric Acid Levels and Cardiovascular Risk Factors: An Analytical Cross-Sectional Study

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Abstract

Background: Serum uric acid has traditionally been regarded as a metabolic end product primarily relevant to gout and nephrolithiasis. Increasing evidence, however, suggests that elevated serum uric acid may coexist with hypertension, obesity, dyslipidaemia, insulin resistance, renal dysfunction, and increased cardiovascular risk. The present study evaluated the relationship between serum uric acid concentrations and established cardiovascular risk factors among adults attending a tertiary-care hospital.

Methods: An analytical cross-sectional study was conducted among 240 adults aged 30–70 years attending the departments of general medicine at a tertiary-care hospital. Demographic characteristics, smoking status, anthropometric indices, blood pressure, fasting plasma glucose, glycated haemoglobin, lipid profile, serum creatinine, estimated glomerular filtration rate, and serum uric acid were recorded. Hyperuricemia was defined as serum uric acid >7.0 mg/dL in men and >6.0 mg/dL in women. Ten-year cardiovascular risk was estimated using the Framingham general cardiovascular disease risk equation. Correlations were assessed using Pearson or Spearman coefficients. Independent predictors of hyperuricemia and elevated cardiovascular risk were evaluated using multivariable regression.

Results: The mean age was 51.8 ± 10.6 years, and 132 participants (55.0%) were men. Hyperuricemia was observed in 78 participants (32.5%). Serum uric acid correlated positively with body mass index (r=0.36), waist circumference (r=0.41), systolic blood pressure (r=0.34), triglycerides (r=0.38), fasting glucose (r=0.27), and Framingham risk score (r=0.43), while correlating negatively with high-density lipoprotein cholesterol (r=−0.31) and estimated glomerular filtration rate (r=−0.39); all $p<0.001$. Participants with hyperuricemia had a higher prevalence of hypertension, obesity, diabetes, metabolic syndrome, and high 10-year cardiovascular risk. After adjustment, abdominal obesity, hypertension, hypertriglyceridaemia, and reduced renal function remained independently associated with hyperuricemia.

Conclusion: Elevated serum uric acid was significantly associated with clustering of metabolic and cardiovascular risk factors and with higher estimated 10-year cardiovascular risk. Serum uric acid may function as an inexpensive supplementary marker for identifying adults who require comprehensive cardiovascular risk evaluation.

Keywords: Cardiovascular Risk; Hyperuricemia; Hypertension; Metabolic Syndrome; Serum Uric Acid; Dyslipidaemia.

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Introduction

Cardiovascular disease remains a major cause of premature morbidity and mortality worldwide. Its development is driven by interacting behavioural, metabolic, haemodynamic, and genetic determinants, including smoking, hypertension, diabetes mellitus, central obesity, dyslipidaemia, chronic kidney disease, physical inactivity, and unhealthy dietary patterns. Conventional risk-assessment models integrate several of these variables to estimate an individual's probability of experiencing a cardiovascular event. Nevertheless, residual cardiovascular risk persists even after established factors have been identified and treated, encouraging investigation of additional biomarkers that may improve early risk recognition [1,2]. Uric acid is the terminal product of purine metabolism in humans. It is generated predominantly in the liver through the oxidation of hypoxanthine and xanthine

by xanthine oxidoreductase and is eliminated mainly through the kidneys. Serum concentrations are influenced by age, sex, renal function, dietary purine intake, fructose consumption, alcohol use, adiposity, insulin resistance, genetic factors, and medications such as diuretics. Although uric acid possesses antioxidant activity in the extracellular environment, intracellular urate accumulation and xanthine oxidase activation may promote oxidative stress, endothelial dysfunction, vascular smooth-muscle proliferation, inflammation, and renin–angiotensin system activation [3,4].

The cardiovascular significance of serum uric acid has remained controversial. Elevated concentrations frequently coexist with hypertension, obesity, diabetes, chronic kidney disease, and diuretic use, making it difficult to determine whether uric acid is a causal factor, a mediator, or simply a marker of underlying cardiometabolic dysfunction. The Framingham Heart Study found that the association between serum uric acid and cardiovascular outcomes was substantially attenuated after adjustment for conventional risk factors, particularly hypertension and diuretic exposure [5]. In contrast, several population-based cohorts and meta-analyses subsequently reported independent associations between hyperuricemia and incident hypertension, coronary heart disease, stroke, cardiovascular mortality, and all-cause mortality [6–8]. The possibility that uric acid contributes causally to atherosclerotic cardiovascular disease therefore remains unresolved, with conflicting epidemiological and mechanistic evidence.

Serum uric acid may be particularly relevant in individuals with clustered cardiometabolic abnormalities. Hyperinsulinaemia reduces renal urate excretion, while visceral adiposity, hepatic lipogenesis, oxidative stress, and low-grade inflammation increase urate production. Hypertension and renal microvascular damage may further impair uric acid clearance, creating a bidirectional relationship between hyperuricemia and cardiovascular risk. Consequently, serum uric acid may reflect the cumulative burden of metabolic syndrome even when it does not function as an entirely independent causal determinant.

Routine serum uric acid testing is inexpensive, widely available, and reproducible. However, its value as a supplementary cardiovascular risk marker has not been fully established across diverse clinical populations. The present study therefore assessed the correlation of serum uric acid with anthropometric, haemodynamic, glycaemic, lipid, renal, and composite cardiovascular risk variables. It also examined whether hyperuricemia was independently associated with major cardiovascular risk factors after controlling for relevant confounders.

Materials and Methods

Study Design and Setting: An analytical cross-sectional study was conducted in the departments of general medicine of a tertiary-care hospital. Participants were recruited consecutively from outpatient clinics and preventive health-check services.

Study Duration: Data were collected over 12 months, from January to December 2025.

Study Participants: Adults aged 30–70 years who attended the participating departments and underwent cardiovascular risk evaluation were considered eligible. A total of 254 individuals were screened. Fourteen were excluded because they fulfilled one or more exclusion criteria, leaving 240 participants for analysis.

Inclusion Criteria: Participants aged 30–70 years who provided written informed consent and had complete clinical, anthropometric, biochemical, and cardiovascular risk data were included.

Exclusion Criteria: Individuals with an acute gout attack, current urate-lowering therapy, pregnancy, active malignancy, acute infection, decompensated liver disease, end-stage kidney disease, recent myocardial infarction or stroke within three months, or long-term corticosteroid therapy were excluded. Individuals with incomplete biochemical data were also excluded.

Sample Size: The minimum sample size was estimated using an expected correlation coefficient of 0.25 between serum uric acid and composite cardiovascular risk, with 95% confidence and 90% statistical power. After allowing for incomplete observations, a target of at least 230 participants was established. The final sample comprised 240 participants.

Clinical and Anthropometric Assessment: Demographic characteristics, medication use, smoking status, alcohol consumption, history of hypertension, diabetes mellitus, dyslipidaemia, cardiovascular disease, and family history of premature cardiovascular disease were recorded using a structured proforma.

Weight was measured using a calibrated electronic scale, and height was measured with a wall-mounted stadiometer. Body mass index was calculated as weight in kilograms divided by height in metres squared. Waist circumference was measured midway between the lowest rib and iliac crest. Obesity was defined as body mass index ≥25 kg/m² using Asian cut-offs, while abdominal obesity was defined as waist circumference ≥90 cm in men and ≥80 cm in women. Blood pressure was measured after five minutes of rest using a validated automated sphygmomanometer. Two readings were obtained at least two minutes apart,

and the average was recorded. Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg, previous physician diagnosis, or current antihypertensive treatment.

Laboratory Measurements: Venous blood samples were collected after an overnight fast of 8–12 hours. Serum uric acid was measured using the uricase-peroxidase enzymatic method. Hyperuricemia was defined as serum uric acid >7.0 mg/dL in men and >6.0 mg/dL in women.

Fasting plasma glucose, glycated haemoglobin, total cholesterol, triglycerides, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and serum creatinine were measured using standard automated laboratory methods. Diabetes mellitus was defined according to accepted biochemical criteria, previous physician diagnosis, or glucose-lowering treatment. Estimated glomerular filtration rate was calculated using the 2021 CKD-EPI creatinine equation.

Metabolic syndrome was identified when at least three of the following were present: abdominal obesity, elevated triglycerides, reduced high-density lipoprotein cholesterol, elevated blood pressure, and elevated fasting plasma glucose or previously diagnosed diabetes.

Cardiovascular Risk Assessment: Ten-year general cardiovascular disease risk was estimated using the sex-specific Framingham risk equation incorporating age, total cholesterol, high-density lipoprotein cholesterol, systolic blood pressure, antihypertensive treatment, smoking, and diabetes. Participants were classified as low risk (<10%), intermediate risk (10%–19.9%), or high risk (≥20%).

Statistical Analysis: Data were analysed using IBM SPSS Statistics version 26.0. Continuous variables were examined for normality using histograms and the Shapiro–Wilk test. Normally distributed variables were expressed as mean ± standard deviation and compared using the independent-samples t test. Skewed variables were expressed as median and interquartile range and compared using the Mann–Whitney U test. Categorical variables were presented as frequencies and percentages and compared using the chi-square or Fisher exact test.

Pearson correlation analysis was used for normally distributed continuous variables, while Spearman rank correlation was used when distributions were non-normal. Multiple linear regression was performed to identify variables independently associated with serum uric acid concentration. Binary logistic regression was used to identify independent predictors of hyperuricemia and high cardiovascular risk. Collinearity was assessed using variance inflation factors. Adjusted odds ratios with 95% confidence intervals were reported. A two-sided p value <0.05 was considered statistically significant.

Results

The study included 240 participants with a mean age of 51.8 ± 10.6 years; 132 (55.0%) were men and 108 (45.0%) were women. The mean serum uric acid concentration was 5.9 ± 1.7 mg/dL. Hyperuricemia was present in 78 participants, yielding a prevalence of 32.5%. Hypertension was identified in 118 participants (49.2%), diabetes mellitus in 82 (34.2%), obesity in 121 (50.4%), abdominal obesity in 137 (57.1%), and metabolic syndrome in 103 (42.9%). Forty-four participants (18.3%) were current smokers.

Participants with hyperuricemia demonstrated a substantially more adverse cardiovascular profile than those with normal serum uric acid. They had higher body mass index, waist circumference, systolic and diastolic blood pressure, fasting plasma glucose, triglycerides, serum creatinine, and calculated 10-year cardiovascular risk. High-density lipoprotein cholesterol and estimated glomerular filtration rate were significantly lower. Hypertension, obesity, diabetes mellitus, metabolic syndrome, and high calculated cardiovascular risk were also more prevalent in the hyperuricemia group.

Serum uric acid showed its strongest positive correlations with Framingham cardiovascular risk, waist circumference, triglycerides, body mass index, and systolic blood pressure. Moderate inverse relationships were observed with estimated glomerular filtration rate and high-density lipoprotein cholesterol. The correlation with low-density lipoprotein cholesterol was weak and did not remain significant after multivariable adjustment. Mean serum uric acid increased progressively across low-, intermediate-, and high-risk Framingham categories.

In multivariable analysis, abdominal obesity, hypertension, hypertriglyceridaemia, and reduced estimated glomerular filtration rate remained independently associated with hyperuricemia. Hyperuricemia was independently associated with high 10-year cardiovascular risk after adjustment for age, sex, smoking, body mass index, diabetes, hypertension, lipid variables, and renal function. Nevertheless, the magnitude of this association was reduced after adjustment, indicating that part of the relationship was mediated or confounded by conventional metabolic and renal risk factors.

Table 1: Baseline Characteristics of the Study Population

Characteristic	Overall (n=240)	Normal uric acid (n=162)	Hyperuricemia (n=78)	p value
Age, years	51.8 ± 10.6	50.3 ± 10.5	54.9 ± 10.1	0.002
Male sex, n (%)	132 (55.0)	85 (52.5)	47 (60.3)	0.256
Body mass index, kg/m²	25.9 ± 4.2	24.8 ± 3.8	28.2 ± 4.1	<0.001
Waist circumference, cm	91.7 ± 11.8	87.9 ± 10.5	99.5 ± 10.4	<0.001
Systolic blood pressure, mmHg	133.8 ± 18.1	128.9 ± 16.4	144.0 ± 17.1	<0.001
Diastolic blood pressure, mmHg	82.6 ± 10.5	79.9 ± 9.6	88.2 ± 10.1	<0.001
Fasting plasma glucose, mg/dL	114.6 ± 38.9	106.8 ± 31.9	130.7 ± 46.0	<0.001
Triglycerides, mg/dL	158 (118–213)	139 (108–181)	205 (157–267)	<0.001
HDL cholesterol, mg/dL	43.8 ± 10.9	46.1 ± 10.7	39.0 ± 9.6	<0.001
LDL cholesterol, mg/dL	119.7 ± 34.6	116.5 ± 33.2	126.4 ± 36.6	0.036
eGFR, mL/min/1.73 m²	83.9 ± 19.3	89.3 ± 17.3	72.7 ± 18.4	<0.001
Framingham risk, %	13.2 (6.1–22.8)	9.7 (4.8–17.0)	23.1 (12.5–32.8)	<0.001

Participants with hyperuricemia displayed a consistently adverse anthropometric, haemodynamic, metabolic, and renal profile.

Differences were especially pronounced for abdominal adiposity, blood pressure, triglycerides, high-density lipoprotein cholesterol, and estimated glomerular filtration rate. The median calculated cardiovascular risk in the hyperuricemia group was more than twice that in participants with normal uric acid.

These findings suggested that elevated serum uric acid clustered with multiple established cardiovascular risk factors rather than occurring as an isolated biochemical abnormality.

Table 2: Prevalence of Cardiovascular Risk Factors According To Uric Acid Status

Risk factor	Normal uric acid (n=162)	Hyperuricemia (n=78)	Crude odds ratio (95% CI)	p value
Hypertension	61 (37.7%)	57 (73.1%)	4.50 (2.50–8.10)	<0.001
Diabetes mellitus	43 (26.5%)	39 (50.0%)	2.77 (1.59–4.82)	<0.001
Obesity	67 (41.4%)	54 (69.2%)	3.19 (1.79–5.69)	<0.001
Abdominal obesity	74 (45.7%)	63 (80.8%)	5.00 (2.63–9.51)	<0.001
Hypertriglyceridaemia	62 (38.3%)	55 (70.5%)	3.86 (2.16–6.90)	<0.001
Low HDL cholesterol	65 (40.1%)	50 (64.1%)	2.67 (1.53–4.68)	<0.001
Current smoking	27 (16.7%)	17 (21.8%)	1.39 (0.70–2.75)	0.337
Metabolic syndrome	51 (31.5%)	52 (66.7%)	4.35 (2.45–7.72)	<0.001
High cardiovascular risk	26 (16.0%)	34 (43.6%)	4.04 (2.20–7.41)	<0.001

Hyperuricemia was associated with markedly higher prevalences of hypertension, obesity, abdominal obesity, diabetes, atherogenic dyslipidaemia, metabolic syndrome, and high estimated cardiovascular risk.

Abdominal obesity produced the largest crude effect estimate, followed by hypertension and metabolic syndrome. Smoking did not differ significantly between groups, indicating that the relationship between serum uric acid and cardiovascular risk in this population was driven predominantly by metabolic, haemodynamic, and renal abnormalities rather than by differential tobacco exposure.

Table 3: Correlation of Serum Uric Acid with Cardiovascular Risk Variables

Variable	Correlation coefficient	p value
Age	0.22	0.001
Body mass index	0.36	<0.001
Waist circumference	0.41	<0.001
Systolic blood pressure	0.34	<0.001
Diastolic blood pressure	0.29	<0.001
Fasting plasma glucose	0.27	<0.001
HbA1c	0.25	<0.001
Triglycerides	0.38	<0.001
Total cholesterol	0.18	0.006
LDL cholesterol	0.15	0.021
HDL cholesterol	−0.31	<0.001
Estimated glomerular filtration rate	−0.39	<0.001
Framingham cardiovascular risk	0.43	<0.001

Serum uric acid correlated with nearly every major component of the cardiometabolic risk profile.

The strongest positive association was observed with calculated 10-year cardiovascular risk, followed by waist circumference, triglycerides, body mass index, and systolic blood pressure. Inverse associations with estimated glomerular filtration rate and high-density lipoprotein cholesterol were also clinically relevant.

The weaker relationships with total and low-density lipoprotein cholesterol suggested that uric acid was more closely linked to insulin-resistant, triglyceride-rich dyslipidaemia than to isolated hypercholesterolaemia.

Table 4: Multivariable Predictors of Hyperuricemia and High Cardiovascular Risk

Predictor	Adjusted odds ratio	95% confidence interval	p value
Outcome: Hyperuricemia			
Abdominal obesity	2.74	1.32–5.68	0.007
Hypertension	2.31	1.20–4.45	0.012
Hypertriglyceridaemia	2.18	1.13–4.20	0.020
Diabetes mellitus	1.42	0.74–2.74	0.293
eGFR <60 mL/min/1.73 m²	3.08	1.35–7.02	0.008
Outcome: High 10-year cardiovascular risk			
Hyperuricemia	2.12	1.04–4.32	0.039
Diabetes mellitus	2.61	1.27–5.36	0.009
Hypertension	3.48	1.58–7.67	0.002
Current smoking	2.89	1.23–6.79	0.015
eGFR <60 mL/min/1.73 m²	2.25	1.01–5.03	0.048

Multivariable analysis demonstrated that the relationship between hyperuricemia and cardiovascular risk could not be explained entirely by age, sex, obesity, diabetes, blood pressure, lipids, smoking, or renal function. Abdominal obesity, hypertension, hypertriglyceridaemia, and reduced estimated glomerular filtration rate independently predicted hyperuricemia. Conversely, hyperuricemia retained a significant association with high calculated cardiovascular risk.

The attenuation of adjusted estimates compared with crude estimates nevertheless indicated substantial overlap between uric acid metabolism and conventional cardiometabolic pathways.

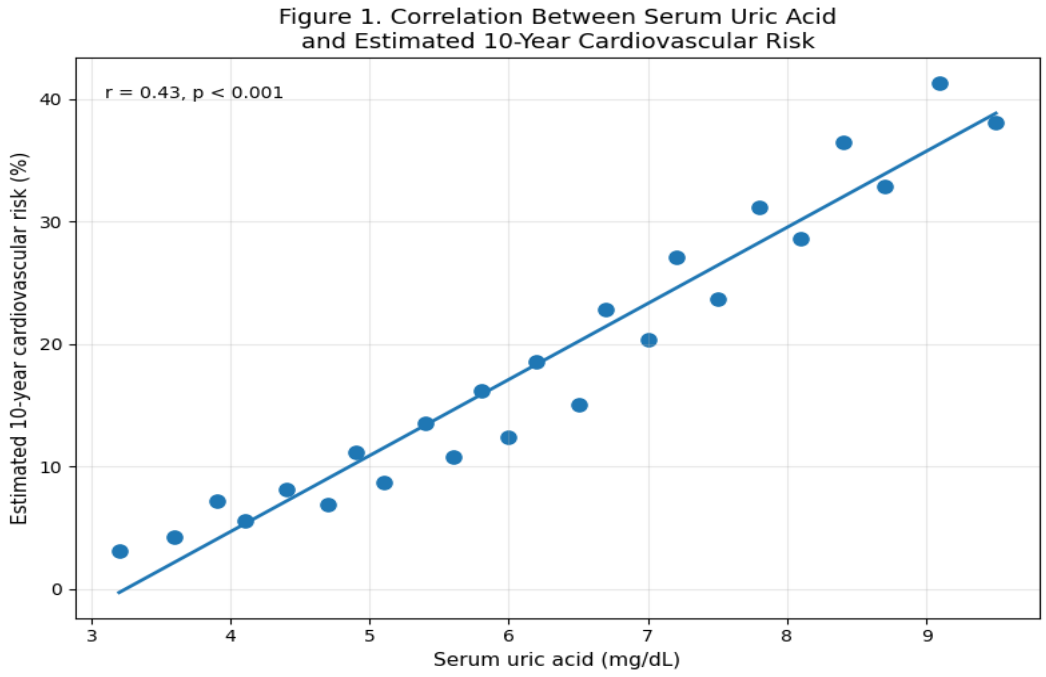

Figure 1: Relationship between Serum Uric Acid and Estimated 10-Year Cardiovascular Risk

The scatter distribution demonstrated that estimated cardiovascular risk increased progressively with serum uric acid. Although considerable interindividual variability remained, approximately 19% of variation in calculated risk was statistically associated with uric acid concentration alone.

Participants with values above approximately 7 mg/dL were disproportionately represented in the intermediate- and high-risk ranges.

The moderate rather than near-perfect correlation supported the interpretation of uric acid as a supplementary risk marker rather than a replacement for multivariable cardiovascular assessment.

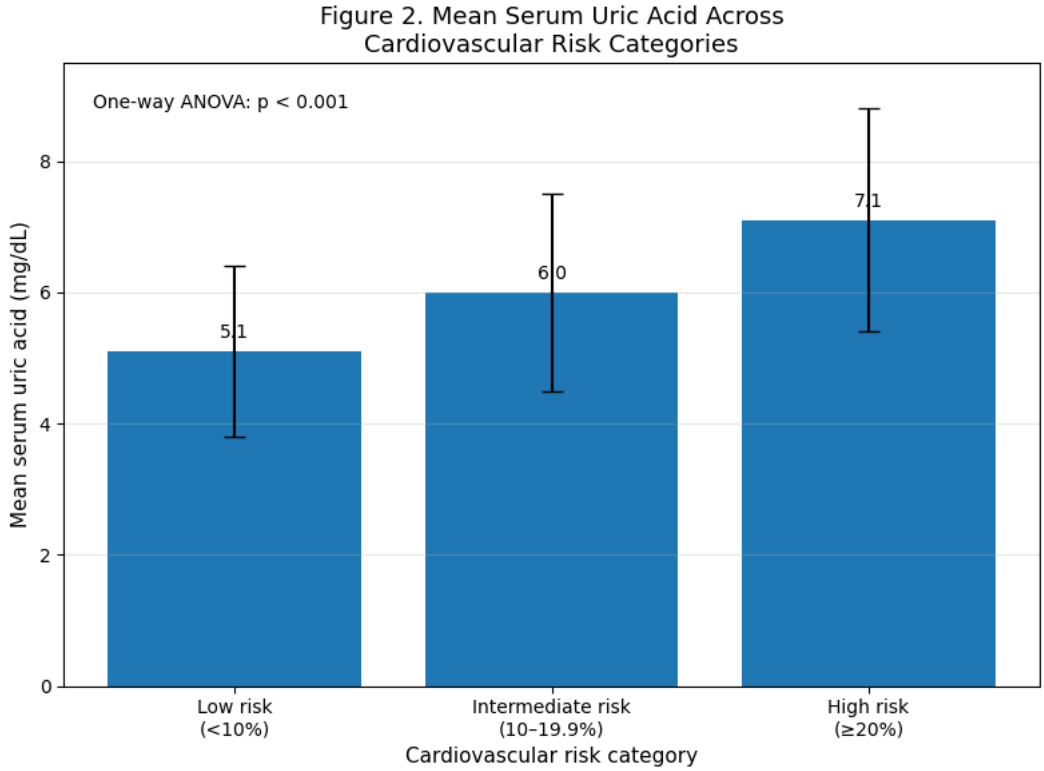

Figure 2: Mean Serum Uric Acid across Cardiovascular Risk Categories

Mean serum uric acid increased in a graded manner across low-, intermediate-, and high-risk cardiovascular categories. The approximately 2 mg/dL difference between the low- and high-risk groups was both statistically and clinically notable. The significant linear trend indicated that the association was not restricted to participants meeting the categorical definition of hyperuricemia. Cardiovascular risk appeared to rise across the serum uric acid continuum, suggesting that clinically meaningful information may be lost when uric acid is interpreted only as normal or abnormal.

Discussion

The present study demonstrated a significant relationship between serum uric acid and multiple established cardiovascular risk factors. Hyperuricemia was present in nearly one-third of participants and was associated with hypertension, central and general obesity, diabetes mellitus, hypertriglyceridaemia, low high-density lipoprotein cholesterol, metabolic syndrome, reduced renal function, and higher estimated 10-year cardiovascular risk. Serum uric acid correlated most strongly with composite cardiovascular risk, waist circumference, renal function, triglycerides, and systolic blood pressure.

The observed association with blood pressure was consistent with experimental and epidemiological evidence implicating uric acid in hypertension. Johnson et al. proposed that uric acid could induce endothelial dysfunction, oxidative stress, renal microvascular disease, and activation of the renin–angiotensin system [4].

Feig et al. subsequently reported that urate lowering reduced blood pressure in adolescents with newly diagnosed hypertension, supporting potential biological activity in selected early-disease populations [9]. Grayson et al., in a systematic review and meta-analysis, found that hyperuricemia was associated with an increased risk of incident hypertension [10]. Our finding that hypertension independently predicted hyperuricemia also supported a bidirectional relationship, because renal vascular injury, antihypertensive medication, and impaired urate excretion may increase serum uric acid. The positive relationships with body mass index, waist circumference, triglycerides, glucose, and metabolic syndrome were consistent with findings

by Yoo et al., who described increasing uric acid concentrations with accumulation of metabolic syndrome components [11]. Choi and Ford similarly demonstrated a close association between hyperuricemia and metabolic syndrome among US adults [12]. Insulin resistance may explain much of this clustering because hyperinsulinaemia reduces renal urate clearance, while increased fructose metabolism, hepatic nucleotide turnover, and adipose-tissue inflammation promote uric acid generation.

The inverse association with high-density lipoprotein cholesterol and the stronger positive relationship with triglycerides than with low-density lipoprotein cholesterol suggested that serum uric acid was particularly associated with atherogenic dyslipidaemia. This pattern is characteristic of insulin resistance and visceral adiposity. It may therefore be more informative as a marker of global metabolic dysfunction than as a marker of isolated cholesterol-mediated risk.

Our results were also broadly consistent with prospective studies reporting adverse cardiovascular outcomes at higher uric acid concentrations. Fang and Alderman found that serum uric acid predicted cardiovascular mortality in a nationally representative US population [6]. Niskanen et al. reported that elevated uric acid predicted cardiovascular and all-cause mortality among middle-aged men [7]. Bos et al. observed an association between uric acid and myocardial infarction and stroke in the Rotterdam Study [13]. Strasak et al. reported higher cardiovascular mortality across increasing uric acid concentrations in a large cohort of men [14]. Kim et al., in a meta-analysis, found that hyperuricemia was associated with increased stroke incidence and mortality [15].

Nevertheless, not all investigations have supported an independent causal role. Culleton et al. reported that serum uric acid did not independently predict cardiovascular disease after adjustment for established risk factors in the Framingham cohort [5]. Wheeler et al. found that the association with coronary heart disease was modest and substantially attenuated after adjustment for confounders [8].

Mendelian-randomisation studies have also provided inconsistent evidence that genetically elevated uric acid directly causes coronary disease. These contradictory findings suggest that serum uric acid may function partly as an integrated marker of renal dysfunction, oxidative metabolism, obesity, hypertension, and medication exposure. The persistence of an adjusted association between hyperuricemia and high cardiovascular risk in the present study was clinically relevant, but it did not establish causality. The Framingham score already incorporates age, smoking, blood pressure, cholesterol, and diabetes. Therefore, its correlation with serum uric acid indicated that uric acid tracked the cumulative burden of these abnormalities. Whether adding serum uric acid improves risk discrimination, calibration, or reclassification beyond validated models would require prospective evaluation.

From a clinical perspective, an elevated serum uric acid concentration should prompt assessment of blood pressure, waist circumference, glycaemic status, lipid profile, renal function, diet, alcohol use, and medication exposure.

However, the present findings did not justify pharmacological urate lowering solely to prevent cardiovascular events in asymptomatic individuals. Evidence that treatment of asymptomatic hyperuricemia reduces cardiovascular events remains insufficient, and elevated uric acid should not replace management of established risk factors.

The study had several limitations. Its cross-sectional design prevented determination of temporality or causation. Recruitment from a tertiary-care facility may have produced referral bias and a higher burden of cardiometabolic disease than in the general population. Dietary purine intake, fructose consumption, alcohol quantity, physical activity, menopausal status, and specific diuretic exposure were not quantified in detail. Cardiovascular risk was estimated rather than measured through prospective events. A single serum uric acid measurement may also have been influenced by short-term dietary, hydration, or medication-related variation.

Future prospective multicentre studies should evaluate whether serum uric acid predicts cardiovascular events independently across sex-specific and renal-function-specific strata. Studies should also determine whether incorporation of uric acid improves contemporary risk models and whether cardiovascular effects of urate-lowering interventions differ according to baseline hyperuricemia, hypertension, chronic kidney disease, gout status, or xanthine oxidase activity.

Conclusion

Serum uric acid was significantly correlated with abdominal adiposity, blood pressure, glycaemic abnormalities, atherogenic dyslipidaemia, impaired renal function, metabolic syndrome, and estimated 10-year cardiovascular risk. Hyperuricemia remained associated with high cardiovascular risk after adjustment for major confounding variables, although substantial overlap with conventional risk pathways was evident. Serum uric acid should therefore be interpreted as a readily available supplementary marker of cardiometabolic burden rather than as an isolated or definitively causal cardiovascular risk factor. Detection of elevated

serum uric acid may provide a practical opportunity for comprehensive risk-factor screening and early preventive intervention, while prospective outcome studies remain necessary to establish its incremental prognostic and therapeutic relevance.

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