

Association of Serum Uric Acid and Microalbuminuria in Essential Hypertension: A Cross-Sectional Study

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ABSTRACT

Background: Hypertension is a leading contributor to cardiovascular and renal morbidity worldwide, and subclinical target-organ damage such as microalbuminuria (MAU) often precedes overt complications. Serum uric acid (SUA) has been implicated in endothelial dysfunction and nephrosclerosis, but data establishing its association with microalbuminuria in Indian patients with essential hypertension remain limited. **Methods:** This hospital-based cross-sectional study was conducted among 100 in-patients and out-patients with essential hypertension attending a tertiary care centre over 18 months. Patients aged 18–80 years were enrolled by purposive sampling after excluding those with chronic kidney disease, diabetes mellitus, hypothyroidism, renal artery disease, and other endocrine causes of hypertension. All participants underwent clinical evaluation, serum uric acid estimation, and urine protein–creatinine ratio testing for microalbuminuria, along with renal function tests and relevant imaging. Hyperuricemia was defined as SUA >7.0 mg/dl in males and >6.0 mg/dl in females. Association between hyperuricemia and microalbuminuria was assessed using the Chi-square test, and mean SUA levels between groups were compared using Student's t-test, with $p < 0.05$ taken as statistically significant. **Results:** The mean age of participants was 53.45 ± 11.657 years, with 71% males. Hyperuricemia and microalbuminuria were present in 22% and 24% of participants, respectively. Among participants with hyperuricemia, 86.4% had microalbuminuria compared with 6.4% of those without hyperuricemia, a statistically significant association ($p < 0.001$). The mean SUA level was significantly higher in participants with microalbuminuria (7.300 ± 0.4374 mg/dl) than in those without (5.108 ± 1.0179 mg/dl; $p < 0.001$). **Conclusion:** Serum uric acid is significantly associated with microalbuminuria in patients with essential hypertension. Hyperuricemia may serve as an accessible, low-cost marker to identify hypertensive patients at higher risk of renal and cardiovascular target-organ damage, supporting its role in risk stratification and early intervention.

Keywords: Serum uric acid; Microalbuminuria; Hyperuricemia; Essential hypertension; Target organ damage

INTRODUCTION

Hypertension is a major global health burden and a leading risk factor for stroke, cardiovascular disease, end-stage renal disease, and overall mortality, with a lifetime risk of nearly 90% among individuals who are normotensive at 55–65 years of age and survive to 80–85 years.[1,2] It is the third most common cause of death

worldwide, responsible for approximately one in every eight deaths, and its prevalence and associated morbidity rise steadily with age.[3]

Even in its asymptomatic phase, hypertension is associated with subclinical target-organ damage, including left ventricular hypertrophy, retinopathy, cognitive dysfunction, and microalbuminuria, which

frequently precede catastrophic events such as stroke, myocardial infarction, and renal failure. The majority of these patients have essential hypertension, a condition of elevated blood pressure without an identifiable secondary cause.

Microalbuminuria (MAU), defined as urinary albumin excretion of 30–300 mg/24 h or an albumin-to-creatinine ratio of 30–300 mg/g (not detectable by routine dipstick methods), is an independent risk factor for cardiovascular and cerebrovascular disease and an early marker of hypertensive and diabetic nephropathy.[4,5] Importantly, MAU has been shown to be at least partly reversible with optimal control of blood pressure and blood glucose.[5,6]

Serum uric acid (SUA), the end product of purine metabolism, has been linked to hypertension, diabetes, heart failure, diabetic kidney disease, and cerebrocardiovascular disease.[7–10] Elevated SUA correlates with rising blood pressure and incident hypertension, and hyperuricemia defined as SUA >7.0 mg/dl in adult males and >6.0 mg/dl in adult females has been associated with a fivefold higher risk of hypertension among individuals with untreated primary disease.[11,12] Proposed mechanisms include inhibition of nitric oxide, activation of the renin–angiotensin system, oxidative stress, vascular smooth muscle proliferation, and endothelial dysfunction.[13,14]

Although derangements of SUA and microalbuminuria have each been individually well documented in hypertensive populations, few studies — particularly in the Indian setting, where dietary patterns substantially influence uric acid metabolism have specifically examined their association. Because both parameters are simple and inexpensive to measure, establishing this relationship could support earlier risk stratification and more cost-effective surveillance. This study was therefore undertaken to assess the association between serum uric acid and microalbuminuria in patients with essential hypertension.

MATERIALS AND METHODS

Study design and setting

This was a hospital-based, cross-sectional, observational study conducted in the Department of General Medicine at a tertiary care teaching hospital, among in-patients and out-patients with essential hypertension.

Study duration and sample size

The study was carried out over a period of 18 months. A sample size of 100 was calculated using the standard finite-population formula for a 95% confidence level with a margin of error of 0.05.

Inclusion criteria

- Patients with essential hypertension, aged 18–80 years
- Willingness to provide written informed consent

Exclusion criteria

- Chronic kidney disease
- Diabetes mellitus
- Hypothyroidism
- Prior treatment for hyperuricemic disorders
- Renal artery disease
- Endocrine causes of secondary hypertension

Sampling and procedure

Patients were enrolled by purposive sampling after obtaining Institutional Ethics Committee approval and written informed consent. Each participant underwent a detailed medical history, general physical examination (including pulse and blood pressure), and systemic examination of the cardiovascular, respiratory, gastrointestinal, and nervous systems. Relevant investigations included electrocardiography, serum uric acid, urine routine examination, urine protein-creatinine ratio, renal function tests, random/fasting/postprandial blood sugar, thyroid function tests, serum cortisol, ultrasonography of the abdomen and pelvis, and renal artery Doppler where indicated. Hyperuricemia was defined as SUA >7.0 mg/dl in males and >6.0 mg/dl in females, and microalbuminuria as a urinary albumin-creatinine ratio of 30–300 mg/g.

Statistical analysis

Data were entered and analysed using standard statistical software. Categorical variables are presented as frequencies and percentages and were compared using the Chi-square test. Continuous variables are presented as mean $\pm$ standard deviation and were compared using Student's t-test. A p value <0.05 was considered statistically significant.

RESULTS

A total of 100 patients with essential hypertension were included in the study. The mean age of participants was 53.45 ± 11.657 years, with the largest proportion (30%) in the 61–70-year age group. Males constituted 71% of the study population. A family history of hypertension was present in 12% and a history of smoking in 37% of participants. The mean BMI was 24.739 ± 2.4616 kg/m², mean systolic blood pressure 161.70 ± 15.359 mmHg, and mean diastolic blood pressure 101.44 ± 9.441 mmHg.

Table 1. Baseline demographic and clinical characteristics (n=100)

Characteristic	Value	Percentage/SD
Mean age (years)	53.45	± 11.657
Male sex	71	71.0%
Family history of hypertension	12	12.0%
Smoking history	37	37.0%
Mean BMI (kg/m ²)	24.739	± 2.4616
Mean SBP (mmHg)	161.70	± 15.359
Mean DBP (mmHg)	101.44	± 9.441

The mean random blood sugar was 107.20 ± 30.175 mg/dl, mean blood urea 28.99 ± 6.008 mg/dl, mean serum creatinine 0.934 ± 0.2061 mg/dl, and mean serum uric acid 5.634 ± 1.3095 mg/dl. With regard to target-organ involvement, left ventricular hypertrophy was present in 15%, coronary artery disease in 29%, cerebrovascular accident/transient ischaemic attack in 16%, and peripheral arterial disease in 1% of participants. Hypertensive retinopathy of any grade was noted in 46% of participants, and 69% had evidence of target-organ damage overall. Hyperuricemia was present in 22% and microalbuminuria in 24% of the study population.

Table 2. Target-organ damage and prevalence of hyperuricemia/microalbuminuria (n=100)

Parameter	n	%
Left ventricular hypertrophy	15	15.0
Coronary artery disease	29	29.0
CVA/TIA	16	16.0
Peripheral arterial disease	1	1.0
Hypertensive retinopathy (any grade)	46	46.0
Target organ damage (overall)	69	69.0
Hyperuricemia	22	22.0
Microalbuminuria	24	24.0

A statistically significant association was found between hyperuricemia and microalbuminuria. Among participants with hyperuricemia, 86.4% (19/22) had microalbuminuria compared with only 6.4% (5/78) of those without hyperuricemia ($p < 0.001$).

Table 3. Association of hyperuricemia with microalbuminuria

Hyperuricemia	MAU present, n (%)	MAU absent, n (%)	p value
Yes (n=22)	19 (86.4%)	3 (13.6%)	<0.001
No (n=78)	5 (6.4%)	73 (93.6%)	

The mean serum uric acid level was significantly higher among participants with microalbuminuria (7.300 ± 0.4374 mg/dl) than among those without (5.108 ± 1.0179 mg/dl; $p < 0.001$).

Table 4. Mean serum uric acid according to microalbuminuria status

Microalbuminuria	Mean SUA (mg/dl)	p value
Present (n=24)	7.300 ± 0.4374	<0.001
Absent (n=76)	5.108 ± 1.0179	

DISCUSSION

Essential hypertension accounts for 90–95% of all hypertension cases and carries substantial risk for stroke, renal failure, and cardiovascular disease. In the present study of 100 patients with essential hypertension, hyperuricemia was strongly and significantly associated with microalbuminuria: 86.4% of participants with hyperuricemia had microalbuminuria compared with 6.4% of those without ($p < 0.001$), and mean SUA was significantly higher in patients with microalbuminuria (7.300 ± 0.4374 mg/dl) than in those without (5.108 ± 1.0179 mg/dl; $p < 0.001$).

These findings are consistent with several previously published studies. Li et al. reported that more than a quarter of 404 patients with essential hypertension had microalbuminuria, and identified uric acid, among other variables, as a predictor of MAU.[15] Bjornstad et al., in obese adolescents with type 2 diabetes, found that a higher baseline SUA independently increased the risk of incident hypertension (hazard ratio 1.19 per 1 mg/dl increase) and elevated urinary albumin excretion (hazard ratio 1.24).[16] Similarly, Chen et al. demonstrated that hyperuricemia was significantly associated with incident hypertension even after adjusting for known confounders in a large prospective cohort, and Yu et al. reported a significantly higher prevalence of hyperuricemia among hypertensive compared with non-hypertensive individuals in a northern Chinese population.[17,18]

A cross-sectional study by Viazzi et al. similarly found that elevated SUA was independently associated with microalbuminuria in hypertensive patients, suggesting hyperuricemia as a marker of early renal damage, while Jalal et al. showed that urate-lowering therapy with allopurinol reduced microalbuminuria in patients with coexisting hyperuricemia and hypertension, supporting a potential therapeutic pathway.[19,20] In contrast, Zhang et al., in a longitudinal cohort, found that baseline microalbuminuria predicted future hypertension more robustly than baseline hypertension predicted future microalbuminuria, underscoring that the temporal relationship between these markers may be bidirectional and warrants further longitudinal evaluation.[21]

Taken together, these findings support the biological plausibility of a shared pathway linking hyperuricemia and microalbuminuria in essential hypertension, most likely mediated through endothelial dysfunction, activation of the renin–angiotensin system, oxidative stress, and nephrosclerosis. Because both SUA and urine albumin-creatinine ratio are inexpensive and widely available tests, their combined use may offer a practical, low-cost strategy for identifying hypertensive patients at higher risk of renal and cardiovascular complications, particularly in resource-limited settings.

Strengths of the Study

The study evaluated the relationship between serum uric acid and microalbuminuria in patients with essential hypertension using predefined biochemical criteria. The study also evaluated multiple indicators of hypertensive target-organ damage, allowing the observed association to be considered within a broader clinical context. Both serum uric acid and urinary albumin assessment are relatively accessible investigations, which may enhance the potential clinical relevance of the findings.

Future Directions

Future multicentre prospective studies with larger sample sizes should evaluate the temporal relationship between serum uric acid and microalbuminuria while adjusting for major demographic, clinical, dietary, renal, and treatment-related confounders. Interventional studies are also required to determine whether lowering serum uric acid can reduce microalbuminuria and improve long-term renal and cardiovascular outcomes.

Limitations

This study has several limitations. Its cross-sectional design precludes conclusions regarding causality or temporal sequence between hyperuricemia and microalbuminuria. The study was conducted at a single centre with a modest sample size of 100 patients, which may limit generalisability. Dietary intake, a known modifier of serum uric acid, was not formally quantified. Larger, multicentric, longitudinal studies are needed to confirm these findings and clarify the directionality of the association.

CONCLUSION

Serum uric acid is significantly associated with microalbuminuria in patients with essential hypertension, with both hyperuricemia and higher mean SUA levels closely linked to the presence of microalbuminuria. Given that both parameters are simple, inexpensive, and widely accessible, routine screening for hyperuricemia and microalbuminuria in hypertensive patients may aid early identification of those at risk for renal and cardiovascular target-organ damage. Addressing hyperuricemia through lifestyle modification and, where indicated, urate-lowering therapy, alongside optimal blood pressure control, may help reduce the risk of progression to overt renal and cardiovascular disease. Further longitudinal and interventional studies are warranted to establish causality and evaluate the impact of urate-lowering strategies on renal outcomes in this population.

DECLARATIONS

Ethical Approval

Obtained from the Institutional Ethics Committee prior to initiation of the study; written informed consent was taken from all participants.

Conflict of Interest

None declared.

Source of funding

Nil.

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Author contributions

Jashwanth Gowda S: study concept, data collection, analysis, and manuscript drafting. Karthik K H: study supervision, critical revision, and final approval of the manuscript.

Data Availability Statement

The datasets generated and/or analysed during the current study are available from the corresponding author on reasonable request, subject to institutional and ethical restrictions.

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