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Evaluation of Serum Uric Acid Levels as an Independent Biomarker for Essential Hypertension Severity and Preclinical Target Organ Damage

Dr. Vedanth Vasudevan, MBBS
Viswabharathi Medical college Kurnool, Andhra Pradesh

Dr. Arsalaan Ahmad Khan, MBBS
Dhaka National Medical College

Ms. Elia Kulsum, MBBS 2nd year student
College of Medicine and Sagore Dutta Hospital, Kolkata

Dr. Mohammed Khaja Moinudeen, MBBS
AL- Falah School of Medical Science and Research Center, Dhauj, HR -121004

Dr. Faraj Khan, MBBS
AL- Falah School of Medical Science and Research Center, Dhauj, HR -121004

Abstract

Background: The pathological intersection between purine metabolism and vascular hemodynamics remains an area of critical clinical investigation. This study evaluates the relationship between serum uric acid (SUA) concentrations, the clinical staging of essential hypertension, and the presence of subclinical target organ damage (TOD).

Methods: A cross-sectional observational analysis was conducted on 250 adult patients diagnosed with essential hypertension. Patients were stratified into Stage 1 and Stage 2 hypertension based on standard clinical guidelines. Serum uric acid levels, glycemic indices, microalbuminuria, and left ventricular mass index (LVMI) via echocardiography were comprehensively evaluated.

Results: The mean SUA concentration was significantly elevated in patients with Stage 2 hypertension compared to Stage 1 (7.12 ± 1.45 mg/dL vs. 5.45 ± 1.12 mg/dL, $p < 0.001$). Pearson correlation coefficient models revealed a strong positive correlation between SUA levels and both systolic blood pressure ($r = 0.44$, $p < 0.001$) and diastolic blood pressure ($r = 0.32$, $p = 0.002$). Furthermore, hyperuricemia was strongly linked to an increased prevalence of microalbuminuria and left ventricular hypertrophy ($p < 0.01$).

Conclusion: Elevated serum uric acid is significantly correlated with the severity of essential hypertension and serves as a reliable marker for preclinical target organ damage. Routine baseline screening of SUA is highly recommended to improve cardiovascular risk stratification.

Introduction

Essential hypertension remains a paramount global public health challenge, driving substantial morbidity and mortality through its long-term cerebrovascular, cardiovascular, and renal complications[1]. While mechanical stress on the vascular wall is a primary driver of systemic deterioration, secondary metabolic biomarkers are increasingly recognized for their predictive value in identifying accelerated vascular damage. Among these, serum uric acid (SUA), the final metabolic byproduct of purine degradation via the xanthine oxidase pathway, and breakdown of ATP from fructose and alcohol metabolism and because of having a mutated nonfunctional uricase enzyme humans are unable to metabolize and degrade uric acid this mutation likely served as a protective mechanism during prolonged periods of starvation as uric acid enhances fat storage and maintains blood pressure but in the recent times the mechanism which worked for the survival of humans has increased the risk for development of hyperuricemia, gout, obesity and hypertension which has shifted uric acid from being viewed as an inert metabolic waste product to a clinically significant mediator of vascular homeostasis.[2]

The physiological solubility limit of monosodium urate in human serum is approximately 6.4 to 7.0 mg/dL. When concentrations exceed this threshold, hyperuricemia develops, increasing the risk of crystalline precipitation within tissue structures[1]. Beyond articular deposition, elevated intracellular urate acts as a pro-inflammatory stimulus that suppresses endothelial nitric oxide synthase (eNOS), and this intracellular stress activates renin angiotensin system inducing profound vasoconstriction and endothelial dysfunction, localized oxidative stress, and the proliferation of vascular smooth muscle cells within the renal microcirculation.[2 3]

The duality of uric acid indicates that under physiological limits it acts as a potent antioxidant which neutralizes free radicals, reduces oxidative stress and provide neuroprotective benefits in conditions like Parkinson's and Alzheimer's disease. Alternatively at elevated concentrations it becomes pro inflammatory and triggers immune response by activating toll like receptors (TLRs) causing release of inflammatory cytokines endothelial dysfunction and reduction in nitric oxide.[10]

While large-scale epidemiological data show a clear link between hyperuricemia and incident hypertension, clinical debates continue regarding whether SUA is an independent causative agent or a downstream marker of metabolic syndrome and subclinical renal impairment.[4] In limited-resource clinical settings, advanced diagnostic screening for subclinical target organ damage (TOD), such as echocardiography or microalbuminuria assays, can be cost-prohibitive or inaccessible.[5] Identifying a reliable, cost-effective, and widely available laboratory biomarker like SUA could significantly improve early risk stratification. This study investigates the direct correlation between serum uric acid levels, the clinical severity of essential hypertension, and indicators of early target organ involvement.

Materials and Methods

A cross-sectional observational study was carried out over a 12-month period in a tertiary care hospital setting. A total of 250 adult patients aged 20 to 65 years with established or newly diagnosed primary essential hypertension were enrolled.

Selection Criteria

- ***Inclusion Criteria***: Consecutive patients diagnosed with essential hypertension under regular follow-up or presenting as new cases; aged between 20 and 65 years; willingness to participate.

- ***Exclusion Criteria:*** Patients with secondary hypertension; history of gout or current treatment with urate-lowering therapies (e.g., allopurinol, febuxostat); chronic kidney disease (estimated glomerular filtration rate < 60 mL/min/1.73m²); ongoing treatment with loop or thiazide diuretics known to alter urate excretion; active malignancies; chronic hepatic disease; or pregnancy; Refusal or inability to provide informed consent.

Ethical Considerations

Patient confidentiality was maintained throughout the study. The study received prior approval from the Institutional Ethics Committee (IEC), and all ethical principles were adhered to throughout the research.

Clinical and Laboratory Assessments

Standardized mercury sphygmomanometers were utilized to record blood pressure in a seated position after a 10-minute rest period. Hypertension severity was graded according to standard clinical criteria: Stage 1 (Systolic Blood Pressure [SBP] 140–159 mmHg or Diastolic Blood Pressure [DBP] 90–99 mmHg) and Stage 2 (SBP ≥ 160 mmHg or Diastolic Blood Pressure [DBP] ≥ 100 mmHg).

Venous blood samples were collected following a 12-hour overnight fast. SUA was measured using the enzymatic colorimetric method on an automated clinical chemistry analyzer. Hyperuricemia was defined as SUA > 7.0 mg/dL in men and > 6.0 mg/dL in women (Kim et al., 1986). Preclinical target organ damage was assessed via spot morning urine collections for microalbuminuria (defined as a urinary albumin-to-creatinine ratio of 30–300 mg/g) and transthoracic echocardiography to calculate the Left Ventricular Mass Index (LVMI), with left ventricular hypertrophy (LVH) defined as an LVMI > 115 g/m² for men and > 95 g/m² for women as according to ASE guidelines[11].

Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics version **27.0** (IBM Corp., Armonk, NY, USA). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as frequencies and percentages. Pearson's correlation coefficient (r) was used to evaluate the strength and direction of the linear relationship between serum uric acid levels and systolic as well as diastolic blood pressure. Correlation coefficients (r) and corresponding p-values were calculated. A p-value of <0.05 was considered statistically significant.

Results

The baseline epidemiological and biochemical characteristics of the study cohort, stratified by hypertension severity, are organized below.

Table 1: Baseline Demographics and Clinical Characteristics of the Study Cohort

Parameter	*Stage 1 Hypertension (n=135)*	*Stage 2 Hypertension (n=115)*	*p-value*
Age (years)	51.4 ± 8.6	53.8 ± 9.2	0.084
Sex (Male/female)	78/57	69/46	0.542
Body Mass Index (kg/m2)	26.2 ± 3.4	27.8 ± 3.9	0.002
Systolic Blood Pressure(mmHg)	148.4 ± 6.2	166.5 ± 8.4	<0.001
Diastolic Blood Pressure(mmHg)	93.1 ± 4.1	104.2 ± 6.3	<0.001
Fasting Blood Glucose (mg/dL)	104.2 ± 18.5	112.6 ± 22.4	0.005
Serum Creatinine (mg/dL)	0.88 ± 0.16	0.94 ± 0.19	0.012

Analysis of purine metabolic status demonstrated a significant escalation in both mean urate concentration and the overall incidence of hyperuricemia as blood pressure status worsened.

Table 2: Prevalence of Hyperuricemia Across Hypertension Severity Groups

Uric Acid Status	*Stage 1 Hypertension (n=135)*	*Stage 2 Hypertension (n=115)*	*Total (n=250)*	*p-value*
Normal Uric Acid, n(%)	112(82.96%)	62(53.91%)	174(69.60%)	-
Hyperuricemia, n(%)	23(17.04%)	53(46.09%)	76(30.40%)	<0.001

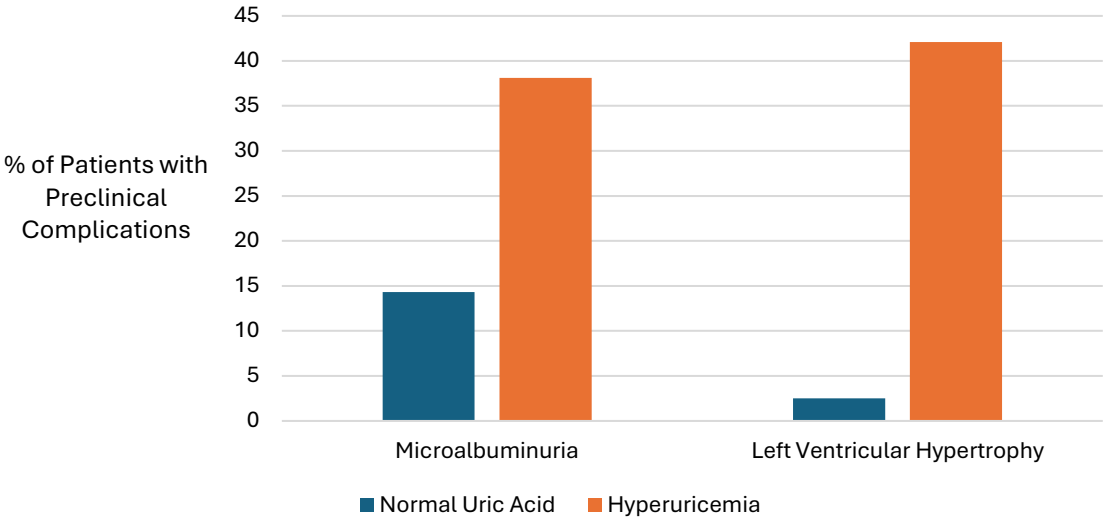
Pearson correlation coefficient models confirmed a strong, statistically significant positive linear relationship between baseline serum uric acid values and systemic blood pressure profiles. The correlation was more pronounced for systolic readings ($r = 0.44$, $p < 0.001$) than for diastolic measurements ($r = 0.32$, $p = 0.002$).

Assessment of subclinical target organ metrics indicated that individuals with hyperuricemia exhibited substantially higher rates of preclinical complications. Microalbuminuria was detected in 38.1% of hyperuricemia subjects compared to only 14.3% of those with normal urate ranges ($p < 0.001$). Similarly, echocardiographic confirmation of left ventricular hypertrophy was significantly more prevalent in the hyperuricemia subgroup relative to normouricemic individuals (42.1% vs. 18.4%, $p < 0.001$) as shown in Table 3.

Table 3: Association of Hyperuricemia with Preclinical Complications

Variable	Normal Uric Acid (n=174)	*Hyperuricemia (n=76)*	*p-value*
Microalbuminuria, n(%)	25(14.3%)	29(38.1%)	<0.001
Left ventricular hypertrophy, n(%)	32(18.4%)	32(42.1%)	<0.001

Figure 1: Association of Hyperuricemia with Preclinical Complications



Discussion

This study confirms a clear, direct correlation between elevated serum uric acid levels and the clinical severity of essential hypertension. Patients classified with Stage 2 hypertension showed substantially higher mean SUA levels and more than double the prevalence of hyperuricemia compared to those with Stage 1 disease. These findings match recent observations by Sarma et al. (2023)[3], who noted a step-wise increase in hyperuricemic frequency as blood pressure shifted into higher stages. Similar findings are also mentioned by Brahma et al. (2026)[6] that higher serum uric acid levels are positively correlated with hypertension severity and duration. In contrast, Golla and Yerraguntla (2022)[7] reported that although elevated serum uric acid levels were associated with hypertension, they were not significantly associated with the severity or duration of hypertension. The authors suggested that serum uric acid may serve as an early and more sensitive biomarker of hypertension than serum creatinine, potentially explaining why its association is more evident during the early stages of the disease. Differences in study population, sample size, duration of hypertension, and methodology may also account for the discrepancy between their findings and those of the present study.

Several interrelated pathological pathways support the biological plausibility of these findings. At the cellular level, soluble uric acid entering vascular smooth muscle cells causes activation of the local renin-angiotensin-aldosterone system (RAAS), downregulates nitric oxide production, and drives intracellular oxidative stress.[2] This initial phase of vasoconstriction leads to permanent vascular remodeling, downstream microvascular injury, and arteriolopathy over time.[4] Additionally, the reduction in renal blood flow typical of sustained systemic hypertension impairs the tubular clearance of urate, establishing a pathophysiological loop where hyperuricemia and hypertension exacerbate one another.[3]

Crucially, our data highlights a strong link between elevated SUA levels and preclinical target organ damage, independent of structural renal decline. The significantly higher rates of microalbuminuria and left ventricular hypertrophy seen in hyperuricemic individuals underscore the systemic nature of urate-mediated endothelial damage.[5,9] As microalbuminuria reflects systemic endothelial dysfunction and serves

as an early sign of nephropathy, its strong correlation with elevated SUA suggests that monitoring urate levels offers clear prognostic value.[5] These results match the findings of Padma et al. (2015), which demonstrated that hyperuricemia significantly tracks with heightened cardiovascular and microvascular risks. Furthermore, our findings support those of Gupta et al. (2025)[9], indicating that serum uric acid may potentially be a valuable marker for the early identification of target organ damage in patients with hypertension.

Study Limitations: This study is limited by its single-center, cross-sectional design, which prevents drawing definitive causal conclusions regarding whether hyperuricemia directly drives or follows advanced hypertension. Longitudinal clinical trials are necessary to determine whether early, targeted urate-lowering therapy can slow the progression of essential hypertension and delay target organ damage.

Conclusion

Serum uric acid levels are directly tied to the severity of essential hypertension and the presence of subclinical target organ damage, including early renal microvascular changes and cardiac hypertrophy. Given its low cost and widespread availability, routine screening for serum uric acid represents a potentially useful tool for risk stratification. Major guidelines such as ESH-ESC and 2020 ISH on hypertension have included SUA as a risk factor for cardiovascular disease in hypertensive populations. Integrating this biomarker into standard hypertension panels can help clinicians identify high-risk patients earlier, guiding more protective cardiovascular interventions[12].

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