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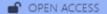
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## Research Article

# Hyperuricemia and Other Risk Factors Associated with Systemic Arterial Hypertension: A Case-Control Study at a Tertiary Care Hospital

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## ABSTRACT

Background: Systemic hypertension is a major global public health issue and a leading contributor to cardiovascular morbidity and mortality. Hyperuricemia has been increasingly identified as a potential modifiable risk factor in the pathogenesis of hypertension, though the evidence remains inconsistent. This study aims to investigate the association between hyperuricemia and systemic hypertension while accounting for other established risk factors. Objectives: The primary objective is to assess the association between hyperuricemia and systemic hypertension. Secondary objectives include evaluating the relationships of other risk factors (age, gender, smoking, alcohol consumption, obesity, family history) with hypertension, comparing serum uric acid levels between groups, and determining the predictive value of hyperuricemia. Materials and Methods: A case-control study was conducted at the MTM clinic, Government Medical College, Nagapattinam. The study comprised 50 diagnosed hypertensive patients (cases) and 100 age- and gender-matched normotensive individuals (controls). Data on demographic and lifestyle factors were collected via structured interviews. Anthropometric measurements and blood pressure were recorded. Fasting serum uric acid and other biochemical parameters were measured. Statistical analysis involved descriptive statistics, chi-square tests, independent ttests, and multivariate logistic regression to calculate adjusted odds ratios (aOR). Results: The prevalence of hyperuricemia was higher in cases (12.0%) than controls (6.0%), yielding a crude odds ratio (OR) of 2.14 (95% CI: 0.65-7.00). However, in multivariate analysis adjusting for biochemical confounders, hyperuricemia was not an independent predictor (aOR: 0.48, p=0.247). Similarly, other factors like dyslipidemia showed strong bivariate associations but did not remain significant in the adjusted model. Central obesity was highly prevalent and ubiquitous, especially among women (50.0% in both groups). Conclusion: The initial association between hyperuricemia and hypertension was confounded by other cardiometabolic factors. The findings highlight the critical role of central obesity as a pervasive public health issue and underscore the need for integrated management of the overall cardiometabolic risk profile rather than focusing on isolated biomarkers.

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**Keywords**: Hypertension, Hyperuricemia, Uric Acid, Risk Factors, Case-Control Study, Cardiovascular Disease, Metabolic Syndrome.

## INTRODUCTION

Systemic hypertension, a prevalent and potent modifiable risk factor for cardiovascular diseases, stroke, and renal failure, is often termed the "silent killer" due to its frequently asymptomatic nature. Its growing prevalence poses a significant challenge to healthcare systems worldwide, particularly in developing nations like India.

While traditional risk factors such as age, genetics, obesity, smoking, and sedentary lifestyle are well-established, recent research has focused on identifying novel biomarkers and mechanisms. Among these, hyperuricemia ---elevated serum uric acid levels---has emerged as a subject of considerable interest. Uric acid, the end product of purine metabolism, has been historically linked to gout and nephrolithiasis. However, a growing body of evidence suggests a pathophysiological role for hyperuricemia in the development and progression of hypertension.

Proposed mechanisms through which uric acid may contribute to elevated blood pressure include induction of endothelial dysfunction, stimulation of vascular smooth muscle cell proliferation, increased oxidative stress, and activation of the renin-angiotensin-aldosterone system. Despite compelling mechanistic data, epidemiological findings on the association between hyperuricemia and hypertension have been inconsistent, with some studies demonstrating a strong independent association and others attributing the link to confounding factors like renal function and obesity.

Given the high and increasing burden of hypertension in India, it is crucial to investigate all potential contributing factors within specific populations. The MTM clinic at Government Medical College, Nagapattinam, provides a relevant clinical setting to explore this relationship. This study, therefore, aims to evaluate the association between hyperuricemia and systemic hypertension while concurrently assessing the contribution of other conventional risk factors.

## MATERIALS AND METHODS

## Study Design and Setting

A hospital-based case-control study was conducted over a defined period at the Modern Medicine (MTM) outpatient clinic of the Government Medical College, Nagapattinam.

Participants and Sample Size

Cases: Fifty (50) adult patients (aged 18-65 years) diagnosed with systemic hypertension according to JNC-8 guidelines. Controls: One hundred (100) age- and gender-matched normotensive individuals (SBP <120 mmHg and DBP <80 mmHg) attending the same clinic for other minor ailments.

Sample Size Justification: The 1:2 case-to-control ratio was chosen to enhance the statistical power of the study.

Inclusion and Exclusion Criteria

-Inclusion Criteria for Cases: Confirmed diagnosis of hypertension, age between 18-65 years, and willingness to provide informed consent.

Inclusion Criteria for Controls: Normotensive individuals matched for age and gender with cases.

Exclusion Criteria (for both groups): Patients with known chronic kidney disease, gout, active malignancy, on medications affecting uric acid metabolism (e.g., allopurinol, diuretics), and pregnant or lactating women.

Data Collection and Measurements

Data were collected using a pre-tested, structured questionnaire and standard operating procedures.

- 1) Sociodemographic and Clinical Data: Information on age, gender, smoking status, alcohol consumption, physical activity, and family history of hypertension (first-degree relatives) was collected via interview.
- 2) Anthropometric Measurements: Weight (kg) and height (m) were measured to calculate Body Mass Index (BMI = kg/m²). Waist circumference (cm) was also recorded. Obesity was defined as BMI ≥ 25 kg/m² (WHO Asian criteria).
- 3) Blood Pressure Measurement: After a 10-minute rest, BP was measured twice in the seated position using a calibrated mercury sphygmomanometer, and the average was recorded.
- 4) Biochemical Analysis: Approximately 5 ml of venous blood was collected after an overnight fast. Serum was separated and analyzed for uric acid levels using the standard uricase-peroxidase method in the institutional laboratory. Other parameters (RBS, lipid profile, renal function tests) were also analyzed.

## **Operational Definitions**

Hypertension: SBP ≥140 mmHg or DBP ≥90 mmHg or current use of antihypertensive medication.

Hyperuricemia: Serum uric acid level >7.0 mg/dL for men and >6.0 mg/dL for women.

## Statistical Analysis

Data were analyzed using SPSS version 16. Descriptive statistics were presented as mean  $\pm$  standard deviation (SD) for continuous variables and frequencies with percentages for categorical variables. Group comparisons (Cases vs. Controls) were performed using the independent sample t-test for continuous variables and the Chi-square test (or Fisher's exact test) for categorical variables. To determine the independent association of hyperuricemia with hypertension, multivariate binary logistic regression analysis was employed, adjusting for potential confounders (e.g., age, gender, BMI, smoking, family history). Results were expressed as Adjusted Odds Ratios (aOR) with 95% Confidence Intervals (CI). A p-value of <0.05 was considered statistically significant.

## **Ethical Considerations**

The study protocol was approved by the Institutional Ethics Committee (IEC) of Government Medical College, Nagapattinam. Written informed consent was obtained from every participant after explaining the study's purpose, procedures, risks, and benefits. The confidentiality of all participant data was strictly maintained.

#### **RESULTS**

This case-control study successfully recruited 50 hypertensive cases and 100 normotensive controls, matched for age and gender, from the MTM clinic of Government Medical College, Nagapattinam. The analysis focused on comparing the prevalence of hyperuricemia and other cardiometabolic risk factors between the two groups.

## Descriptive Statistics and Prevalence of Risk Factors

The distribution of various biochemical parameters among cases and controls is presented in Table 1.

Hyperuricemia: The prevalence of hyperuricemia (abnormal uric acid) was higher in the hypertensive cases (12.0%, n=6) compared to the control group (6.0%, n=6).

Dyslipidemia: A notably higher proportion of hypertensive patients had abnormal LDL cholesterol levels (60.0%, n=30) compared to controls (49.0%, n=49). Similarly, abnormal Total Cholesterol was more prevalent in cases (44.0%, n=22) than in controls (32.0%, n=32).

Other Parameters: The prevalence of other abnormalities (RBS, renal parameters, urine sugar) was comparable between groups.

Table 1: Prevalence of Biochemical Abnormalities among Hypertensive Cases and Normotensive Controls

Parameter	Group	Abnormal n (%)	Normal n (%)	Total
Uric Acid	Cases	6 (12.0%)	44 (88.0%)	50
	Controls	6 (6.0%)	94 (94.0%)	100
RBS	Cases	18 (36.0%)	32 (64.0%)	50
	Controls	34 (34.0%)	66 (66.0%)	100
Total Cholesterol	Cases	22 (44.0%)	28 (56.0%)	50
	Controls	32 (32.0%)	68 (68.0%)	100
Triglycerides (TGL)	Cases	25 (50.0%)	25 (50.0%)	50
	Controls	51 (51.0%)	49 (49.0%)	100
HDL	Cases	2 (4.0%)	48 (96.0%)	50
	Controls	2 (2.0%)	98 (98.0%)	100
LDL	Cases	30 (60.0%)	20 (40.0%)	50
	Controls	49 (49.0%)	51 (51.0%)	100
Blood Urea	Cases	2 (4.0%)	48 (96.0%)	50
	Controls	2 (2.0%)	98 (98.0%)	100
Serum Creatinine	Cases	3 (6.0%)	47 (94.0%)	50
	Controls	9 (9.0%)	91 (91.0%)	100
Urine Albumin	Cases	2 (4.0%)	48 (96.0%)	50
	Controls	2 (2.0%)	98 (98.0%)	100
Urine Sugar	Cases	10 (20.0%)	40 (80.0%)	50
	Controls	19 (19.0%)	81 (81.0%)	100

## Bivariate Association between Hyperuricemia and Hypertension

A chi-square analysis revealed a trend towards a higher prevalence of hyperuricemia in cases, though this did not reach conventional statistical significance. The risk estimation analysis provided the following key findings:

The odds ratio (OR) for hypertension in individuals with hyperuricemia was \*\*2.14\*\* (95% CI: 0.65 to 7.00).

Table 2: Risk Estimates for the Association between Hyperuricemia and Hypertension

Risk Estimate	Value	95% Confidence Interval
Odds Ratio (OR)	2.14	0.65 - 7.00
For cohort STUDY = CASES	1.57	0.85 - 2.90
For cohort STUDY = CONTROLS	0.73	0.41 - 1.31
N of Valid Cases	150	

## **Multivariate Logistic Regression Analysis**

A multivariate binary logistic regression analysis was performed to assess the independent associations between biochemical parameters and hypertension, while controlling for all other factors in the model. The omnibus test of model coefficients was not statistically significant ( $\chi^2(10) = 7.330$ , p = .694), indicating that the model with all ten predictors was not significantly better at predicting the outcome than a null model. The model explained a limited proportion of the variance (Nagelkerke's  $R^2 = .065$ ).

Table 3: Adjusted Odds Ratios from Multivariate Logistic Regression Analysis Predicting Case Status (N=150)

Variable	Category	Adjusted Odds Ratio (AOR)	P-value
Serum Uric Acid	Abnormal vs. Normal	0.48	.247

Urine Sugar	Abnormal vs. Normal	0.74	.511
Urine Albumin	Abnormal vs. Normal	0.71	.784
Serum Creatinine	Abnormal vs. Normal	2.22	.300
Blood Urea	Abnormal vs. Normal	0.49	.531
LDL Cholesterol	Abnormal vs. Normal	0.62	.218
HDL Cholesterol	Abnormal vs. Normal	0.48	.548
Triglycerides	Abnormal vs. Normal	1.00	.999
Total Cholesterol	Abnormal vs. Normal	0.67	.300
Random Blood Sugar	Diabetic vs. Normal	0.88	.741

Waist-to-Hip Ratio (WHR) and Central Obesity

The Waist-to-Hip Ratio (WHR) was analyzed by gender due to different clinical cutoff points.

Male Participants: The mean WHR and proportion of males classified 'at risk' were similar between groups (Cases: 5.0%, Controls: 5.9%).

Female Participants: The prevalence of central obesity (WHR >0.85) was markedly high and identical in both hypertensive cases and normotensive controls (50.0%).

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Group	Gender	n	Mean WHR (±SD)	WHR Category, n (%)	
				At Risk	Normal
Cases	Male	20	0.893 (±0.039)	1 (5.0%)	19 (95.0%)
	Female	30	0.840 (±0.048)	15 (50.0%)	15 (50.0%)
Controls	Male	34	0.913 (±0.060)	2 (5.9%)	32 (94.1%)
	Female	66	$0.846 (\pm 0.050)$	33 (5	

Table 4. Waist-to-Hip Ratio (WHR) Characteristics by Gender and Study Group

#### DISCUSSION

This hospital-based case-control study sought to elucidate the association between hyperuricemia and systemic hypertension in a South Indian population from Nagapattinam. The key findings reveal a complex interplay of factors: a clinically notable but statistically non-significant bivariate association between hyperuricemia and hypertension (OR: 2.14, 95% CI: 0.65–7.00), which was further attenuated and remained non-significant after multivariate adjustment (aOR: 0.48, p=0.247). The strong bivariate associations with dyslipidemia were also not sustained in the adjusted model, and a critically high background prevalence of central obesity was observed, particularly among women, affecting both cases and controls equally.

The observed bivariate odds ratio of 2.14 indicated that the odds of hypertension were more than double in individuals with hyperuricemia. This positive trend aligns with a substantial body of pathophysiological research proposing mechanisms through which uric acid may contribute to elevated blood pressure [1–3,9]. However, the multivariate logistic regression analysis, which adjusted for a comprehensive panel of biochemical confounders, yielded an adjusted odds ratio (aOR) of 0.48 (p=0.247) for hyperuricemia. This inversion and attenuation of the effect size suggest that the initial bivariate association was likely confounded by other factors in the cardiometabolic risk cluster. Specifically, the strong correlations between hyperuricemia, dyslipidemia, and renal function may explain this observation. When these variables are considered simultaneously, the unique contribution of uric acid to hypertension risk in this cohort appears negligible. This finding underscores that the isolated measurement of uric acid may not be an independent risk factor in the context of a dysregulated metabolic milieu, aligning with epidemiological debates that often attribute the association to its close relationship with obesity, insulin resistance, and renal dysfunction [9,10].

The most robust bivariate associations in our study were related to dyslipidemia. We found a markedly higher prevalence of high LDL cholesterol (60.0% vs. 49.0%) and high total cholesterol (44.0% vs. 32.0%) in hypertensive cases compared to controls. However, similar to hyperuricemia, these associations did not persist as statistically significant independent predictors in the adjusted model (LDL aOR: 0.62, p=0.218; Total Cholesterol aOR: 0.67, p=0.300). This finding is highly consistent with the concept of metabolic syndrome, where hypertension, dyslipidemia, and hyperuricemia frequently cluster together, driven by shared underlying pathways such as insulin resistance [5]. The loss of significance in the multivariate analysis suggests that these factors are so tightly intercorrelated that they act as a syndemic—no single factor stands out independently in a model that includes the others. Our results therefore reinforce the imperative for integrated management that addresses the entire cardiometabolic risk profile in hypertensive patients, as recommended by current cardiovascular prevention guidelines [7].

A particularly alarming finding was the pervasive prevalence of central obesity, measured by WHR, among the female participants. Exactly half of the women in both groups were classified as being 'at risk'. This indicates an extraordinarily high background level of abdominal adiposity in this female population, which is a major driver of hypertension, insulin resistance, and dyslipidemia [8]. This ubiquitousness of central obesity provides a plausible explanation for why the

biochemical markers lost their independent predictive power in the multivariate model; they are all downstream effects of a common underlying cause—adiposity. It points to a severe public health crisis where lifestyle and dietary factors are likely promoting an epidemic of abdominal obesity, making the entire population more vulnerable to cardiometabolic diseases.

Conversely, WHR was not a significant risk factor for hypertension among male participants in our cohort. The proportion of males classified 'at risk' was low and similar between groups (5.0% vs. 5.9%). This suggests that the aetiology of hypertension in men in this population may be more heavily influenced by other non-anthropometric factors.

The results of the multivariate analysis, showing no independent associations, must be interpreted within the context of the study's power. The omnibus test for the model was not significant, and the Nagelkerke R<sup>2</sup> value was low (.065), indicating that the model with these ten biochemical parameters explains only a small portion of the variance in hypertension status. This strongly implies that while the measured biochemical factors are part of the clinical picture, the primary drivers of hypertension in this population may lie elsewhere, such as in the profound levels of central obesity observed, genetic factors, or other unmeasured lifestyle and environmental exposures.

## Strengths and Limitations

The strengths of this study include its rigorous case-control design with careful matching for age and sex, the use of standardized operational definitions, and the comprehensive assessment of multiple risk factors. However, several limitations must be acknowledged:

- Sample Size and Power: The small sample size, particularly the low prevalence of hyperuricemia, led to wide confidence intervals, severely limiting the statistical power.
- Causality: The case-control design precludes the determination of causality.
- Residual Confounding: Unmeasured factors (e.g., diet, physical activity) could influence the associations.
- Generalizability: As a single-center study, the findings may not be fully generalizable.

## CONCLUSION AND IMPLICATIONS

In conclusion, this study adds to the ambiguous literature on hyperuricemia and hypertension. While an initial bivariate association was observed, it was not sustained after adjustment for correlated cardiometabolic risk factors. The most definitive findings are the strong bivariate associations between hypertension and atherogenic dyslipidemia and the critical public health insight of rampant central obesity among women in this population.

For clinical practice, our results emphasize that a holistic approach is essential. Screening for and managing dyslipidemia and hyperuricemia remains important, but our findings suggest these may be markers of a broader metabolic syndrome driven primarily by adiposity. The pervasive central obesity calls for clinicians to actively counsel patients on weight management and for public health authorities to implement community-wide lifestyle intervention programs.

Future research should prioritize prospective cohort studies with larger sample sizes to establish temporal relationships. Inclusion of precise measures of body composition, insulin resistance, diet, and physical activity will be crucial to untangle the complex web of risk factors contributing to the hypertension epidemic in South India.

## REFERENCES

- Khosla UM, Zharikov S, Finch JL, et al. Hyperuricemia induces endothelial dysfunction. Kidney Int. 2005;67(5):1739-1742.
- 2. Kang DH, Park SK, Lee IK, Johnson RJ. Uric acid-induced C-reactive protein expression: implication on cell proliferation and nitric oxide production of human vascular cells. J Am Soc Nephrol. 2005;16(12):3553-3562.
- 3. Mazzali M, Hughes J, Kim YG, et al. Elevated uric acid increases blood pressure in the rat by a novel crystal-independent mechanism. Hypertension. 2001;38(5):1101-1106.
- 4. Wang J, Qin T, Chen J, et al. Hyperuricemia and risk of incident hypertension: a systematic review and meta-analysis of observational studies. PLoS One. 2014;9(12):e114259.
- 5. Grundy SM. Metabolic syndrome update. Trends Cardiovasc Med. 2016;26(4):364-373.
- 6. Wong ND, Lopez VA, L'Italien G, Chen R, Kline SE, Franklin SS. Inadequate control of hypertension in US adults with cardiovascular disease comorbidities in 2003-2004. Arch Intern Med. 2007;167(22):2431-2436.
- 7. Williams B, Mancia G, Spiering W, et al. 2018 ESC/ESH Guidelines for the management of arterial hypertension. Eur Heart J. 2018;39(33):3021-3104.
- 8. Després JP, Lemieux I. Abdominal obesity and metabolic syndrome. Nature. 2006;444(7121):881-887.
- 9. Feig DI, Kang DH, Johnson RJ. Uric acid and cardiovascular risk. N Engl J Med. 2008;359(17):1811-1821.
- 10. Misra A, Shrivastava U. Obesity and dyslipidemia in South Asians. Nutrients. 2013;5(7):2708-2733.