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Association of Hyperuricemia with Hypertension in Adult without Major Metabolic Comorbidities

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ABSTRACT

Introduction: Hypertension is a major global health problem and a leading cause of cardiovascular morbidity and mortality. Hyperuricemia has been increasingly recognized as a potential metabolic factor associated with elevated blood pressure, although its independent role remains unclear, particularly in individuals without obesity, diabetes mellitus, or chronic kidney disease (CKD).
Methods: Hypertension is a major global health problem and a leading cause of cardiovascular morbidity and mortality. Hyperuricemia has been increasingly recognized as a potential metabolic factor associated with elevated blood pressure, although its independent role remains unclear, particularly in individuals without obesity, diabetes mellitus, or chronic kidney disease (CKD).
Result: Hypertension occurred in 72.5% of participants with hyperuricemia compared with 25.0% without hyperuricemia. Hyperuricemia was significantly associated with hypertension ($p < 0.001$; contingency coefficient = 0.429). Participants with hyperuricemia had a 2.91-fold higher risk of hypertension (RR = 2.91; 95% CI: 1.64–5.13). This association persisted across age groups, with RR = 2.41 (95% CI: 1.31–4.44) in participants aged <60 years and RR = 3.73 (95% CI: 1.25–11.16) in those aged ≥60 years.
Conclusion: Hyperuricemia was associated with an increased risk of hypertension independent of major metabolic comorbidities and age, suggesting its potential role as an additional metabolic risk factor for hypertension.

Keywords: Hyperuricemia; hypertension; uric acid serum; metabolic risk factor

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Introduction

Hypertension remains a major global health problem and is a leading contributor to cardiovascular morbidity and mortality[1]. It is often referred to as a silent killer because it may progress without specific symptoms and is frequently diagnosed only after target organ damage occurs. Numerous risk factors have been implicated in the development of hypertension, including age, sex, genetic predisposition, smoking habits, body mass index, and metabolic disorders such as diabetes mellitus and obesity[2].

In addition to these well-established risk factors, hyperuricemia has increasingly attracted attention as a metabolic condition potentially associated with elevated blood pressure[3,4]. Uric acid exerts antioxidant effects at physiological concentrations; however, at elevated levels, it may act as a pro-oxidant, promoting oxidative stress, endothelial dysfunction, and vascular inflammation. These mechanisms are believed to contribute to increased peripheral vascular resistance and activation of the renin-angiotensin system (RAS), both of which play important roles in the pathogenesis of hypertension[5,6].

Previous studies have demonstrated an association between hyperuricemia and hypertension[7,8]. Nevertheless, many of these studies were conducted in populations with major metabolic comorbidities, particularly obesity, which is a strong and independent risk factor for hypertension. Obesity may confound the assessment of the role of hyperuricemia, making it difficult to determine whether elevated uric acid levels contribute independently to the development of hypertension[9,10]. Furthermore, several studies have focused primarily on statistical significance without emphasizing the quantitative risk estimation.

Therefore, studies evaluating the association between hyperuricemia and hypertension in non-obese populations are limited. Assessing this relationship in a non-obese population may provide a clearer understanding of the contribution of hyperuricemia within the context of other classical risk factors, such as smoking, genetic predisposition, and variations in body mass index[9]. In addition, the use of medical record–based data reflects real-world clinical conditions in which hyperuricemia is frequently encountered in routine healthcare settings.

This study aimed to assess the relative risk of hyperuricemia for the occurrence of hypertension in a non-obese clinical population at Sultan Agung Islamic Hospital, Semarang, using medical record data from 2016 to 2020. The findings of this study are expected to clarify the role of hyperuricemia as an additional risk marker for hypertension and support early screening and preventive strategies for hypertension in clinical practice.

Methods

Study Design and Ethical Approval

This study employed an analytic observational design with a retrospective cohort approach using medical record data from Sultan Agung Islamic Hospital, Semarang, which were collected between 2016 and 2020. Ethical approval for this study was obtained from the Health Research Ethics Committee of Sultan Agung Islamic Hospital (approval no. 55/EC/KEPK/2020).

Study Population

The target population consisted of both inpatients and outpatients at Sultan Agung Islamic Hospital who presented with joint pain. The accessible population included patients with and without hyperuricemia whose blood pressure measurements were recorded in the hospital medical records during the study period (2016–2020).

Sampling Technique

Sampling was performed using a consecutive sampling method, in which all patients who met the eligibility criteria were included sequentially until the required sample size was reached.

Inclusion and Exclusion Criteria

The inclusion criteria were patients aged ≥18 years with a documented history of hypertension or without hypertension. The exclusion criteria were coronary heart disease, obesity, diabetes mellitus, chronic kidney disease, and incomplete medical record data. Obesity was defined as a body mass index (BMI) of ≥30 kg/m².

Data Collection

Data collection was conducted between October and December 2020. Relevant variables, including hyperuricemia status and blood pressure measurements, were obtained from medical records. The collected data were reviewed for completeness, coded, tabulated, and entered into the Statistical Package for the Social Sciences (SPSS) software for analysis.

Statistical Analysis

Statistical analyses were performed using IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA). The association between hyperuricemia and hypertension was analyzed using the non-parametric Contingency Coefficient test. Statistical significance was determined based on the Approximate Significance (Approx. Sig.) Statistical significance was set at $p < 0.05$.

Potential confounding factors were minimized through restriction at the study design stage by excluding participants with obesity, diabetes mellitus, chronic kidney disease, a smoking history, and a family history of hypertension. For non-modifiable risk factors that could not be equally distributed between the study groups, particularly age, an additional stratified analysis was conducted to evaluate the association between hyperuricemia and hypertension across age categories (<60 and ≥60 years).

Result

Eighty participants were included in the analysis. The baseline characteristics of the study population according to hyperuricemia status are presented in **Table 1**. Among participants with hyperuricemia (n = 40), the mean age was 54.4 ± 12.2 years, whereas participants without hyperuricemia had a mean age of 41.5 ± 13.5 years. Participants aged ≥60 years constituted 35.0% of the hyperuricemia group compared with 12.5% of the non-hyperuricemia group, indicating a higher proportion of older adults among those with hyperuricemia.

The mean body mass index was comparable between the two groups, with values of 21.78 ± 2.18 kg/m² in the hyperuricemia group and 21.70 ± 2.42 kg/m² in the non-hyperuricemia group. All participants were classified as non-obese (BMI <30 kg/m²). Male participants predominated in both groups, accounting for 70.0% of the hyperuricemia group and 52.5% of non-hyperuricemiacemia group. No participant in either group was diagnosed with diabetes mellitus or chronic kidney disease, indicating a relatively homogeneous metabolic profile of the study population.

Table 1. Baseline Characteristics of Study Participants (n = 80)

Variable	Hyperuricemia (n=40)	Non-hyperuricemia (n=40)
Age (years)	54.4 ± 12.2	41.5 ± 13.5
Age <60 years	26 (65.0%)	35 (87.5%)
Age ≥60 years	14 (35.0%)	5 (12.5%)
BMI (kg/m²)	21.78 ± 2.18	21.70 ± 2.42
Sex		
Male	28 (70.0%)	21 (52.5%)
Female	12 (30.0%)	19 (47.5%)
Hypertension		
Yes	29 (72.5%)	10 (25.0%)
No	11 (27.5%)	30 (75.0%)
Diabetes Mellitus		
No	40 (100%)	40 (100%)

Chronic Kidney Disease		
No	40 (100%)	40 (100%)
Smoking History		
No	40 (100%)	40 (100%)
Family history of hypertension		
No	40 (100%)	40 (100%)

Data are presented as mean ± standard deviation or numbers (percentages). All participants had a normal body mass index (BMI <30 kg/m²). None of the participants had diabetes mellitus, chronic kidney disease, a smoking history, or a family history of hypertension.

The association between hyperuricemia and hypertension is presented in **Table 2**. Among participants with hyperuricemia, hypertension was observed in 29 individuals (72.5%), while 11 (27.5%) were normotensive. In contrast, among participants without hyperuricemia, hypertension was present in 10 individuals (25.0%), whereas 30 individuals (75.0%) were hypertensive. Statistical analysis demonstrated a significant association between hyperuricemia and hypertension (P < 0.001). The contingency coefficient value of 0.429 indicates a moderate positive association between hyperuricemia and hypertension. Relative risk analysis showed that participants with hyperuricemia had a 2.91-fold higher risk of hypertension than those without hyperuricemia (RR = 2.91; 95% CI: 1.64–5.13).

Table 2. Association Between Hyperuricemia and Hypertension

		Hypertension n (%)	Normotension n (%)	Total n (%)	CC value	p-value	RR (95% CI)
Hyperuricemia	Yes	29 (36.3)	11 (13.8)	40 (50.0)	0.429	<0.001	2.91 (1.64–5.13)
	No	10 (12.5)	30 (37.5)	40 (50.0)			

CC: Contingency Coefficient.

To further evaluate the potential influence of age on the association between hyperuricemia and hypertension, a stratified analysis by age group was performed (Table 3). Among participants aged <60 years, hypertension was observed in 18 of 26 participants (69.2%) with hyperuricemia compared with 9 of 35 participants (25.7%) without hyperuricemia, corresponding to a relative risk of 2.41 (95% CI: 1.31–4.44).

Table 3. Stratified Analysis of the Association Between Hyperuricemia and Hypertension by Age Group

Age Group	Hyperuricemia with Hypertension n/N (%)	Non-hyperuricemia with Hypertension n/N (%)	RR	95% CI
< 60 years	18/26 (69.2%)	9/35 (25.7%)	2.41	1.31–4.44
≥ 60 years	11/14 (78.6%)	1/5 (20.0%)	3.73	1.25–11.16

In the elderly group (≥60 years), hypertension was observed in 11 of 14 participants (78.6%) with

hyperuricemia, whereas only one of five participants (20.0%) without hyperuricemia had hypertension. The relative risk of hypertension associated with hyperuricemia in this age group was 3.73 (95% CI: 1.25–11.16). Overall, the stratified analysis demonstrated that the association between hyperuricemia and hypertension persisted across both age groups.

Discussion

The present study identified an association between hyperuricemia and hypertension in a non-obese population without diabetes mellitus or chronic kidney disease. By restricting the study population to individuals without major metabolic comorbidities, this study aimed to minimize the influence of well-established risk factors and better elucidate the role of hyperuricemia in relation to hypertension.

This association is consistent with previous epidemiological studies that reported a relationship between elevated serum uric acid levels and hypertension. Fuchs et al. demonstrated that higher uric acid levels were associated with an increased risk of hypertension, while Thangadurai et al. reported a high prevalence of hyperuricemia among hypertensive adults, suggesting a close link between these conditions across different populations[9,11]. More recent clinical studies have also shown significant correlations between serum uric acid levels and both systolic and diastolic blood pressure, supporting the concept that hyperuricemia is an emerging metabolic factor associated with blood pressure elevation[7].

Several biological mechanisms may explain the association between hyperuricemia and hypertension. At elevated concentrations, uric acid may act as a pro-oxidant, promoting oxidative stress and endothelial dysfunction[2,12]. Increased production of reactive oxygen species reduces nitric oxide bioavailability, resulting in impaired vasodilation and increased peripheral vascular resistance. In addition, hyperuricemia has been shown to stimulates smooth vascular muscle cell proliferation through the activation of mitogen-activated protein kinase pathways and platelet-derived growth factors, contributing to vascular remodeling and arterial stiffness[6,13]. These vascular changes are key contributors to the development and persistence of hypertension.

Hyperuricemia may also affect blood pressure regulation via renal mechanisms. Experimental studies have demonstrated that elevated uric acid levels can induce renal microvascular injury, leading to impaired sodium handling and increased salt sensitivity[14,15]. Activation of the renin–angiotensin system and upregulation of inflammatory mediators, such as cyclooxygenase-2, further contribute to sustained increases in blood pressure. These renal and vascular mechanisms provide a plausible pathophysiological basis for the association observed in the present study, even in the absence of overt chronic kidney disease[16,17].

Stratified analysis by age showed that the association between hyperuricemia and hypertension persisted in both younger and older participants. This finding suggests that the observed relationship is

not solely explained by age, a well-known, non-modifiable risk factor for hypertension. Although the relative risk appeared higher among elderly participants, the wider confidence interval in this group indicates limited precision due to the smaller sample size and warrants a cautious interpretation. Nevertheless, the persistence of the association across age strata supports the robustness of these findings.

In this study, hypertension was more frequently observed among male participants, which is in line with previous reports that indicated a higher prevalence of hypertension in men than in women. Sex-related differences in lifestyle factors, occupational stress, and hormonal influences may contribute to these patterns. Estrogen exerts protective effects on vascular function in premenopausal women, whereas estrogen deficiency after menopause may increase susceptibility to blood pressure elevation. However, smoking status and family history of hypertension were absent in the study population, reducing the potential confounding effects.

Several limitations should be acknowledged when interpreting the results of this study. The retrospective approach and reliance on secondary medical record data restricted the evaluation of several variables, including dietary habits, physical activity levels, and psychosocial stress. Furthermore, the relatively limited number of participants may reduce the broader applicability of these findings. In addition, serum uric acid levels were classified into categories rather than analyzed as continuous values, which made it impossible to evaluate the potential dose–response relationship. Nevertheless, the application of restriction and stratified analyses helped improve the internal validity of the study by reducing the potential impact of major confounding variables.

In conclusion, this study provides evidence of an association between hyperuricemia and hypertension in a nonobese population without major metabolic comorbidities. These findings support the consideration of hyperuricemia as an additional metabolic factor associated with hypertension and highlight the potential value of early identification and monitoring of elevated uric acid levels in clinical practice.

Conclusion

This study demonstrated that hyperuricemia was associated with an increased risk of hypertension in a nonobese clinical population without diabetes mellitus or chronic kidney disease. This association remained evident after age stratification, suggesting that the relationship between hyperuricemia and hypertension was not solely explained by age. These findings support the consideration of hyperuricemia as an additional metabolic factor associated with hypertension. Further studies with larger sizes and prospective designs in non-obese populations are warranted to elucidate the causal relationship and the role of serum uric acid in the prevention and management of hypertension.

Conflicts of Interest

There is no conflict of interest.

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