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Association Between Uric Acid Levels and Stenosis Severity in Patients With and Without Renal Insufficiency

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ABSTRACT

Hyperuricemia is associated with an increased risk of atherosclerosis and coronary stenosis, particularly in the presence of renal insufficiency. However, the independent relationship between serum uric acid levels and the severity of coronary stenosis, when stratified by renal function, remains poorly understood. This study aimed to evaluate the association between serum uric acid concentrations and the extent of coronary stenosis in patients with and without renal insufficiency. A cross-sectional study was conducted on 853 patients diagnosed with acute coronary syndrome (ACS) who underwent coronary angiography. Elevated uric acid was defined as ≥ 7.1 mg/dL, while renal insufficiency was defined as an estimated glomerular filtration rate (eGFR) < 60 mL/min/1.73 m^2. The associations between variables were analyzed using Chi-square tests and logistic regression models. The 853 patients, 386 (45.3%) presented with renal insufficiency. In the overall cohort, elevated uric acid levels were significantly associated with coronary stenosis of 50% or greater ($p = 0.005$). However, upon subgroup analysis, this association lost statistical significance in both the renal insufficiency ($p = 0.319$) and non-renal insufficiency ($p = 0.154$) groups. Conversely, traditional cardiovascular risk factors—specifically advanced age ($p < 0.001$), male sex ($p < 0.001$), and smoking history ($p = 0.008$)—remained significantly associated with coronary stenosis across both subgroups. While elevated serum uric acid levels correlate with coronary stenosis in the general ACS population, this relationship does not remain independently significant when patients are stratified by renal function. Traditional risk factors, such as age, sex, and smoking, serve as more robust predictors for the progression of coronary stenosis.

Key Messages:

- While elevated uric acid levels correlate with coronary stenosis in the general population, this association loses significance when stratified by renal status, suggesting that traditional risk factors like age, male sex, and smoking are more primary drivers of stenosis progression.

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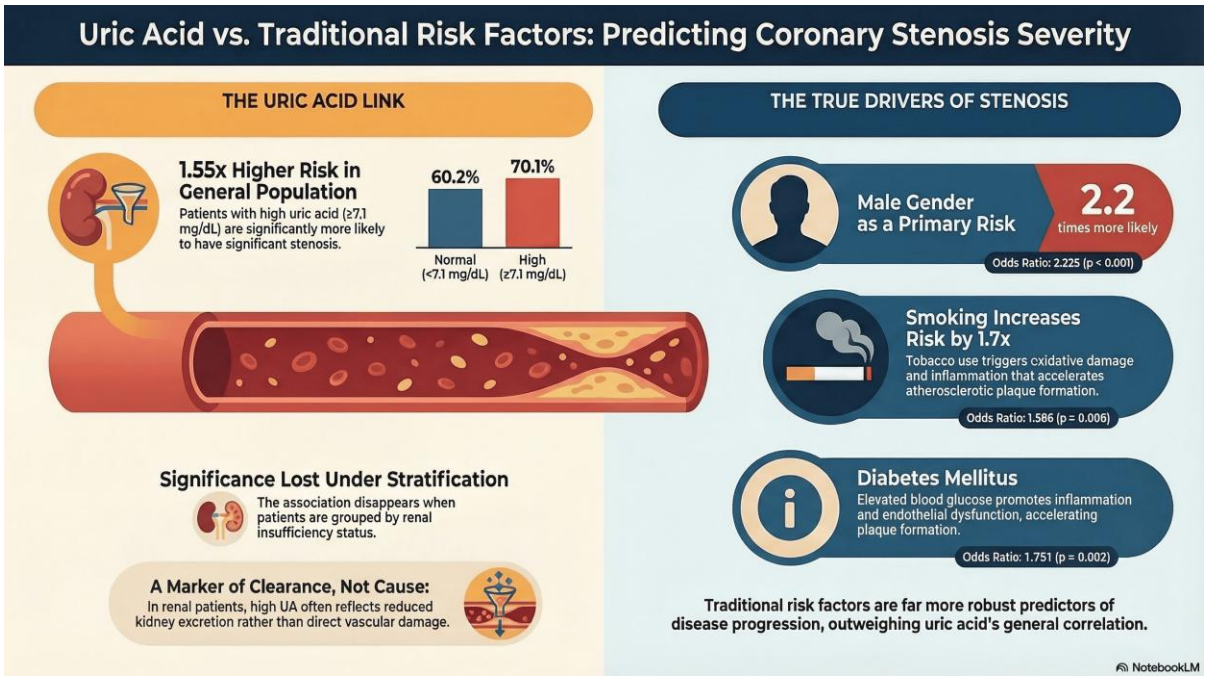
GRAPHICAL ABSTRACT



INTRODUCTION

Coronary heart disease (CHD) remains one of the leading causes of morbidity and mortality worldwide, including in Indonesia (1). The extent of coronary artery stenosis plays a critical role in determining prognosis and guiding treatment decisions in affected patients. The severity of atherosclerotic narrowing can be accurately assessed through coronary angiography, which remains the gold standard for evaluating coronary artery disease(2). Numerous risk factors have been identified for CHD, such as hypertension, diabetes mellitus, and dyslipidemia, all of which contribute to the progression of atherosclerosis.

In recent years, uric acid (UA) has gained attention as a possible non-traditional risk factor for cardiovascular disease. UA is the final product of purine metabolism and is linked to several cardiovascular and renal conditions, including hypertension and chronic kidney disease (CKD)(3)(4). An imbalance between uric acid synthesis and elimination primarily causes increased serum uric acid concentrations Elevated serum UA levels generally result from an imbalance between production and excretion, with the kidneys responsible for eliminating approximately two-thirds of total uric acid, while the remainder is excreted via the gastrointestinal tract (5). When renal function declines, uric acid clearance decreases, leading to hyperuricemia that may promote vascular inflammation, endothelial dysfunction, and atherosclerosis (6). These mechanisms suggest that uric acid may influence the degree of coronary stenosis, particularly among patients with impaired renal function.

Although several studies have reported an association between serum uric acid levels and coronary artery stenosis, the evidence remains inconsistent, particularly regarding whether this relationship persists independently of renal function (7)(8). Additionally, UA is linked to the onset of heart failure (HF), heightened mortality rates, and atrial fibrillation (6). In some populations, uric acid acts as a marker of reduced renal clearance rather than an independent contributor to vascular disease. Given these uncertainties, further research is needed to clarify the relationship between uric acid, renal function, and coronary stenosis severity. Therefore, this study aimed to evaluate the association between serum uric acid levels and the degree of angiographic stenosis in patients with and without renal insufficiency at Sultan Agung Islamic Hospital, Semarang, Indonesia.

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METHODS

This research was conducted at Sultan Agung Islamic Hospital, Semarang, using a cross-sectional observational analytical design. The study was a retrospective analysis based on secondary data obtained from existing patient medical records, which were collected between January 2018 and June 2024. The use of secondary data provides an efficient approach to evaluate associations within real-world clinical populations; however, it also carries inherent limitations related to the accuracy and completeness of recorded information and the inability to control for unmeasured confounding variables. This study aimed to evaluate the relationship between serum uric acid levels and the severity of coronary stenosis, particularly among patients with and without renal insufficiency.

The study population comprised all patients diagnosed with acute coronary syndrome (ACS) who underwent coronary angiography at Sultan Agung Islamic Hospital between January 2018 and June 2024. Patients with complete data on serum uric acid and estimated glomerular filtration rate (eGFR) were included, while those with incomplete medical records or a prior history of coronary artery bypass grafting were excluded.

The minimum required sample size was estimated using a single-proportion formula:

$$n = Z^2 \times p(1-p)/d^2$$

where Z = 1.96 for a 95% confidence level, p = 0.5 (conservative estimate in absence of prior data), and d = 0.05 (margin of error).

The calculation yielded *n = 384.16*, rounded to *385*. To compensate for potential incomplete data (10%), the minimum sample required was *424* participants. In this study, a consecutive sampling technique was used, meaning that all eligible patients who met the inclusion criteria during the study period were included. Consequently, the final sample comprised 853 patients, exceeding the minimum required sample size and providing sufficient statistical power. Among them, 386 patients had renal insufficiency, and 467 did not.

Serum uric acid levels were classified as “Normal” (<7.1 mg/dL) and “High” (≥7.1 mg/dL). This threshold is consistent with recommendations from the American College of Rheumatology (ACR) and other international guidelines, which define hyperuricemia as serum uric acid levels exceeding 7.0 mg/dL in men and 6.0 mg/dL in women, based on the solubility limit of monosodium urate at physiological temperature. In this study, a uniform cutoff of ≥7.1 mg/dL was applied to simplify categorization and maintain consistency with prior cardiovascular research evaluating uric acid and coronary risk. The degree of coronary stenosis was determined angiographically and categorized as “Significant” (≥50%) or “Not significant” (<50%).

Independent Variable: Serum uric acid levels, categorized as “Normal” and “High.” Dependent Variable: Degree of coronary stenosis categorized as “Significant” (≥50%) and “Not Significant” (<50%). Stratification Variable: Renal function categorized as “Renal Insufficiency” (GFR <60 ml/min/1.73 m3) and “Non-Renal Insufficiency” (GFR ≥60 ml/min/1.73 m3). Confounding Variables for Multivariate Analysis: Age, sex, smoking, diabetes mellitus, hypertension, creatinine, body mass index (BMI), HDL, LDL, total cholesterol, triglycerides, and GFR.

Data analysis was conducted utilizing SPSS version 25.0. Univariate descriptive statistics were employed to characterize the sample population. The chi-square test was employed to assess the correlation between serum uric acid levels and the degree of stenosis. Analyses were conducted separately for patients with and without renal insufficiency to assess the impact of renal function on the correlation between uric acid and stenosis. The independent effect of serum uric acid on stenosis was assessed through logistic regression analysis to control for potential confounding variables. Additional variables, including age, gender, smoking status, diabetes mellitus, hypertension, BMI, HDL, LDL, total cholesterol, triglycerides, and renal insufficiency, were also controlled for in the analysis. Statistical analysis was conducted using the Chi-square test to evaluate associations between categorical variables and binary logistic regression to determine predictors of significant stenosis. Variables with $p < 0.25$ in bivariate analysis were included in multivariate modeling. A p-value < 0.05 was considered statistically significant.

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CODE OF HEALTH ETHICS

The Bioethics Committee of the Faculty of Medicine, Sultan Agung Islamic University (UNISSULA) accepted this study with reference number 307/VIII/2024/Bioethics Commission.

RESULTS

Table 1 shows the baseline demographic and clinical characteristics of a study cohort comprising 853 participants, stratified into Renal Insufficiency (RI, n=386) and Non-RI (n=467) groups. Demographically, the RI cohort is notably older (mean age 61.1 ± 8.3 vs. 52.9 ± 8.7 years) and has a slightly higher proportion of females (72.5% vs. 66.0%) compared to the Non-RI group. Clinically, the RI group demonstrates expectedly lower Glomerular Filtration Rates (47.2 vs. 77.0) and elevated serum creatinine (1.5 vs. 1.0), alongside higher mean serum uric acid levels (7.2 vs. 6.0), although the Non-RI group shows a higher categorical prevalence of 'High' uric acid (71.5% vs. 66.3%). While baseline BMI, lipid profiles (HDL and LDL), smoking status, and diabetes prevalence are relatively comparable between the two cohorts, the Non-RI group presents with a higher prevalence of certain cardiovascular comorbidities, specifically hypertension (77.1% vs. 54.9%) and significant stenosis (44.3% vs. 27.2%).

Table 1. Baseline Characteristic

Variable	RI (n=386)	Non-RI (n=467)	All (n=853)
Continuous variable mean (S.D.)			
Age	61,1 (8,3)	52,9 (8,7)	56,6 (9,5)
Serum Uric Acid	7,2 (5,7)	6,0 (3,8)	6,5 (4,8)
Creatinine	1,5 (0,5)	1,0 (0,2)	1,2 (0,4)
BMI	26.8 ± 4.7	25.9 ± 4.3	26.3 ± 4.9
HDL	37,1 (9,6)	38,5 (10,5)	37,9 (10,2)
LDL	115,1 (39,6)	112,9 (37,7)	113,9 (38,5)
GFR	47,2 (9,6)	77,0 (13,6)	63,5 (19,1)
Categorical variable, n (%)			
Gender			
- Male	106 (27,5)	159 (34)	265 (31,1)
- Female	280 (72,5)	308 (66)	588 (68,9)
Smoking Status			
- Yes	209 (54,1)	258 (55,2)	467 (54,7)
- No	177 (45,9)	209 (44,8)	386 (45,3)
Uric Acid Category			
- High	256 (66,3)	334 (71,5)	590 (69,2)
- Normal	130 (33,7)	133 (28,5)	263 (30,8)
DM Status			
- Yes	195 (50,5)	260 (55,7)	455 (53,3)
- No	191 (49,5)	207 (44,3)	398 (46,7)
Hypertension			
- Yes	212 (54,9)	360 (77,1)	572 (67,1)
- No	174 (45,1)	107 (22,9)	281 (32,9)
Stenosis Category			
- Significant	105 (27,2)	207 (44,3)	312 (36,6)
- Non-Significant	281 (72,8)	260 (55,7)	541 (63,4)

Note: RI: Renal Insufficiency, S.D.: Standard Deviation, BMI: Body Mass Index, HDL: High Density Lipoprotein, LDL: Low Density Lipoprotein, GFR: Glomerulus Filtration Rate.

Table 2 illustrates the relationship between serum uric acid levels and the degree of stenosis within the overall study population. Individuals with high serum uric acid levels demonstrated a notably higher prevalence of significant stenosis (70.1%, n=197) than those with normal uric acid levels (60.2%, n=344). Statistical analysis confirms that this observed difference is significant (P-value = 0.005). Furthermore, the Prevalence Ratio (PR) of 1.554, supported by a 95% Confidence Interval (CI) of 1.145 to

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2.110, indicates that patients with elevated uric acid are approximately 1.55 times more likely to exhibit significant stenosis than those with normal levels.

Table 2. Relationship Between Uric Acid Levels and Stenosis in the Overall Population

Category		Degree of Stenosis		Total	P Value	PR	95% CI	
		Non-Significant	Significant				Lower	Upper
Serum Uric Acid	Normal	228	344	572	0.005	1,554	1,145	2,110
		39,8%	60,2%	100%				
	High	84	197	281				
		29,9%	70,1%	100%				

Note:PR: Prevalence Ratio, CI: Confidence Interval

Table 3. Subgroup Analysis: Patients with Renal Insufficiency

Category		Degree of Stenosis		Total	P Value	PR	95% CI	
		Non-Significant	Significant				Lower	Upper
Serum Uric Acid	Normal	62	150	212	0.358	1,259	0,800	1,980
		29,25 %	70,75 %	100%				
	High	43	131	174				
		24,71 %	75,29 %	100%				

Note: PR: prevalence ratio, CI: confidence interval

Table 3 shows a subgroup analysis evaluating the relationship between serum uric acid levels and the degree of stenosis specifically within patients diagnosed with renal insufficiency. Among this restricted cohort, individuals with high serum uric acid exhibited a slightly higher clinical prevalence of significant stenosis (75.29%, n=131) compared to those with normal uric acid levels (70.75%, n=150). However, unlike the findings observed in the overall population, this association does not reach statistical significance (p-value = 0.358). Furthermore, while the calculated prevalence ratio (PR) is 1.259, the 95% confidence interval (CI) ranges from 0.800 to 1.980; because this interval crosses the null value of 1.0, it confirms that within the specific subgroup of patients with renal insufficiency, elevated serum uric acid is not statistically proven to be a reliable predictor for an increased likelihood of significant stenosis

Table 4. Subgroup Analysis: Patients Without Renal Insufficiency

Category		Degree of Stenosis		Total	P Value	PR	95% CI	
		Non-Significant	Significant				Lower	Upper
Serum Uric Acid	Normal	166	194	360	0.183	1,377	0,886	2,142
		46,11%	53,89%	100%				
	High	41	66	107				
		38,32%	61,68%	100%				

Note: PR: Prevalence Ratio, CI: Confidence Interval

Table 4 shows a subgroup analysis examining the association between serum uric acid levels and the degree of stenosis, specifically among patients without renal insufficiency. Within this cohort, individuals with high serum uric acid levels had a higher proportion with significant stenosis (61.68%, n=66) than those with normal uric acid levels (53.89%, n=194). However, statistical analysis indicates that this observed difference does not reach statistical significance (p-value = 0.183). This lack of significance is further corroborated by a calculated Prevalence Ratio (PR) of 1.377 alongside a 95% Confidence Interval (CI) ranging from 0.886 to 2.142; because this confidence interval encompasses the null value of 1.0, it statistically demonstrates that elevated serum uric acid is not a reliable predictor for an increased likelihood of significant stenosis within the specific subgroup of patients who do not have renal insufficiency.

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Table 5. Subgroup Analysis: Patients Without Renal Insufficiency

Variable	Wald χ^2	p-value (Sig.)	Odds ratio (Exp(B))	95% C.I. for Exp(B) (Lower – Upper)
Age	15.242	.000*	1.046	1023 - 1070
Gender	12.626	.000*	2.225	1431 - 3458
Serum Uric Acid	1.298	.255	1.032	.977 - 1091
Creatinine	.098	.754	1.137	.509 - 2537
Smoking Status	7.016	.008	1.686	1145 - 2481
Diabetes Mellitus	9.941	.002*	1.751	1236 - 2480
Hypertension	3.888	.049*	1.362	1002 - 1852
BMI	.070	.792	1.000	1000 - 1000
HDL	.019	.890	.999	.982 - 1016
LDL	3.718	.054	1.007	1000 - 1015
Total Cholesterol	.033	.856	1.001	.994 - 1007
Triglycerides	.001	.970	1.000	.999 - 1001
GFR	.616	.432	.993	.976 - 1011

Note: *p:* power, CI: Confidence Interval, BMI: Body Mass Index, HDL: High Density Lipoprotein, LDL: Low Density Lipoprotein, GFR: Glomerulus Filtration Rate.

Table 5 shows a subgroup analysis identifying the independent clinical and demographic predictors of stenosis among patients without renal insufficiency. The regression analysis reveals several statistically significant risk factors, notably age ($p < 0.001$, OR = 1.046), gender ($p < 0.001$, OR = 2.225), smoking status ($p = 0.008$, OR = 1.686), diabetes mellitus ($p = 0.002$, OR = 1.751), and hypertension ($p = 0.049$, OR = 1.362), indicating that these traditional cardiovascular comorbidities substantially increase the likelihood of stenosis in this cohort. Crucially, and consistent with the univariate findings in Table 4, serum uric acid does not demonstrate a statistically significant independent association with the outcome in this specific subgroup ($p = 0.255$; OR = 1.032, 95% CI: 0.977–1.091). Furthermore, other renal and metabolic parameters—including creatinine, GFR, BMI, and lipid profiles (HDL, LDL, Total Cholesterol, and Triglycerides)—also failed to reach statistical significance (all $p > 0.05$). This suggests that for patients with normal renal function, established risk factors like diabetes, smoking, and hypertension are much more robust predictors of stenosis than isolated uric acid levels or basic lipid metrics.

DISCUSSION

This study's findings indicate a complex correlation between stenosis severity and blood uric acid levels in individuals, particularly in populations with and without renal insufficiency. Pearson's Chi-square test ($\chi^2 = 8.069$, $p = 0.005$) showed a statistically significant association between increased uric acid levels and significant stenosis (≥50%) in the general population. This study is consistent with previous studies that have linked hyperuricemia to atherosclerosis and vascular dysfunction (6). According to Bao Yang (7)Uric acid is one of the most significant independent predictors of the Genshini score. Oxidative stress and inflammatory responses induced by uric acid are recognized mechanisms that elevate uric acid levels and exacerbate coronary heart disease. Uric acid generates numerous free radicals via the actions of xanthine dehydrogenase and xanthine oxidase (9,10). These radicals participate in a sequence of inflammatory processes and enhance the endothelium-mediated vasodilation function, resulting in vascular endothelial dysfunction (11,12). Furthermore, uric acid can release various cytokines, activate the coagulation system, and exacerbate platelet adhesion and aggregation, thereby elevating the risk of thrombosis, leading to coronary embolism and increasing the incidence of cardiovascular events(13). Uric acid has the capacity to inhibit nitric oxide (NO) synthesis, reduce NO bioavailability, stimulate the renin-angiotensin system, promote vascular smooth muscle cell proliferation, and increase platelet aggregation (7). This pathway elucidates the impact of hyperuricemia on the formation and progression of atherosclerosis, while also providing a robust theoretical foundation for the prognostic value of serum uric acid in assessing the severity of coronary heart disease.

However, several other studies have shown that uric acid has a low to insignificant OR (14). Additional cardiovascular risk factors, including hypertension, hyperlipidaemia, diabetes, and the

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administration of antihypertensive and lipid-lowering medications, may affect the weak correlation between serum uric acid levels and cardiovascular disease. The subgroup analysis of this study indicated no significant association between uric acid levels and stenosis in patients with renal insufficiency (GFR <60 ml/min/1.73 m^2) (χ^2 = 0.991, p = 0.319). Although renal insufficiency was a major focus of this study, its effect on coronary stenosis was not statistically significant. This lack of significance may be explained by the complex pathophysiological mechanisms in chronic kidney disease (CKD), where multiple comorbidities, such as hypertension, diabetes mellitus, and dyslipidemia act synergistically to accelerate atherosclerosis. These comorbid factors can overshadow or mediate the independent role of uric acid on vascular injury, resulting in a weaker or non-significant direct association in regression analysis. Several studies have also reported that uric acid loses its predictive value for cardiovascular outcomes once renal function declines, because uric acid becomes more of a marker of reduced kidney clearance rather than a direct causal factor of vascular damage (4,15). Elevated uric acid levels in patients with reduced GFR may primarily indicate diminished excretory capacity rather than an independent atherogenic mechanism. This explanation also supports the finding that both uric acid and GFR categories were not significant predictors in the present study. Consistent with prior research, traditional cardiovascular risk factors such as age, sex, smoking, diabetes, and hypertension appear to have a stronger influence on stenosis severity than uric acid in populations with impaired renal function (16). In patients without renal insufficiency (GFR ≥60 ml/min/1.73 m^2), the lack of association may reflect preserved renal clearance that maintains uric acid homeostasis despite metabolic variations. Other unmeasured factors, including genetic predisposition, medication effects, and lifestyle habits, may also contribute to these results (17).

When many risk factors were considered, uric acid's effect on stenosis was reduced to the point that it was not statistically significant (p = 0.255). The univariate analysis revealed a correlation between uric acid and stenosis, but its predictive value declined when other risk factors were considered. These results are consistent with studies showing that uric acid can function as a secondary indicator of cardiovascular risk, not a primary factor (3,18). Age, sex, smoking status, diabetes mellitus, and high blood pressure played a stronger role as predictors of stenosis. Cubrilo et al. (19) found that age was a strong predictor of stenosis, in line with the significance we found for the age variable (Wald χ^2 = 15.242, p < 0.001). Each additional year increased the odds of stenosis by 6.6% (OR = 1.066, 95% CI: 1.023–1.070). Men were twice as likely to have significant stenosis compared to women (OR = 2.225, 95% CI: 1.431–3.458). The difference in lower physiological estrogen levels had a negative effect on men of all ages. In men, the incidence of cardiovascular disease was higher, with lower estrogen levels accelerating the development of heart failure and death (20).

Smoking was shown to significantly affect the degree of stenosis (p = 0.008, OR = 1.686, CI: 1.145–2.481). Smokers had almost 1.7 times greater risk of developing stenosis than non-smokers. This can be explained by oxidative damage and inflammation induced by chemical components in cigarettes, such as nicotine and carbon monoxide, which accelerate the formation of atherosclerotic plaque (21). Nicotine's ability to accelerate the oxidative stress process causes damage and inflammation to the endothelium (21). Dysregulated immune responses and chronic (low-grade) inflammation have a significant role in the formation and progression of atherosclerosis and, ultimately, cardiovascular disease (22). This study's findings emphasize the importance of addressing modifiable lifestyle factors in the prevention of coronary artery disease. Hypertension showed marginal significance (p = 0.049), indicating that the effect may be less severe in this group due to overlapping risk variables (23).

The logistic regression did not demonstrate statistical significance for other variables, including creatinine, BMI, or cholesterol levels (HDL, LDL, and total cholesterol). However, we cannot ignore the role of these variables in the pathogenesis of cardiovascular disease. Creatinine and GFR, as markers of renal function, may be more relevant in subpopulations of patients with renal insufficiency. Cardiovascular events are common in individuals with CKD; previous studies have found that 50% of patients in stages 4 to 5 suffer from cardiovascular disease (24). Unfortunately, in this study we did not categorize our subjects into CKD stages. Meanwhile, previous lipid therapy use may affect lipid profiles (HDL, LDL, and total cholesterol).

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This study demonstrated that uric acid levels did not exert a significant independent effect on the degree of stenosis in patients, regardless of renal insufficiency status. However, the overall Chi-Square test results indicated a significant relationship. Additional risk factors, including age, male gender, smoking status, and diabetes mellitus, significantly influence the development of coronary artery stenosis. Uric acid levels may serve as an additional risk factor; however, they do not represent the primary determinant of stenosis severity in patients with acute coronary syndrome.

Based on these findings, clinicians should continue to prioritize the management of established cardiovascular risk factors, such as hypertension, diabetes, dyslipidemia, and smoking cessation when addressing coronary artery disease, while considering uric acid only as a potential adjunct marker rather than a therapeutic target. Regular assessment of renal function remains essential to interpret uric acid levels appropriately in clinical settings. Further prospective and mechanistic studies are recommended to clarify the causal pathways linking uric acid, renal function, and atherosclerosis progression. Larger, multi-center cohorts and interventional trials assessing the impact of uric acid–lowering therapy on coronary outcomes in different GFR categories would provide stronger evidence regarding its clinical relevance.

CONCLUSION

This study demonstrates that while elevated serum uric acid levels exhibit a significant correlation with the severity of coronary stenosis in the general population of patients with acute coronary syndrome (ACS). However, this association loses its independent statistical significance when the cohort is stratified based on renal function.

Multivariate analysis reveals that traditional cardiovascular risk factors—specifically advanced age, male sex, smoking history, diabetes mellitus, and hypertension—remain the most robust and primary predictors of significant coronary stenosis. These results suggest that in patients with declining renal function, hyperuricemia may serve more as a marker of reduced renal clearance rather than a direct independent driver of atherosclerotic progression.

Consequently, clinical management should continue to prioritize the mitigation of established risk factors, such as smoking cessation and the control of diabetes and hypertension, while regarding serum uric acid as a secondary adjunct marker rather than a primary therapeutic target for preventing coronary artery disease.

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CONFLICTS OF INTEREST

The authors declare no conflicts of interest.

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