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Association of Serum Uric Acid Levels with Disease Severity and Short-Term Outcomes in Patients with Ischemic Heart Disease: A Prospective Observational Study

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ABSTRACT

Background: Serum uric acid (SUA) has emerged as a potential marker of cardiovascular risk and adverse outcomes in patients with ischaemic heart disease (IHD). However, its association with clinical severity, cardiac dysfunction, and short-term prognosis remains incompletely understood in hospitalized patients.
Objective: To evaluate the association of serum uric acid levels with disease severity, cardiac biomarkers, cardiac function, and short-term clinical outcomes among patients with ischaemic heart disease.
Methods: This prospective observational study was conducted among 80 adults diagnosed with angina, ST-segment elevation myocardial infarction (STEMI), or non-ST-segment elevation myocardial infarction (NSTEMI). Patients were stratified into three SUA categories: 4.0–5.5 mg/dL, 5.6–7.0 mg/dL, and >7.0 mg/dL. Clinical severity was assessed using Killip class, New York Heart Association (NYHA) functional class, and Global Registry of Acute Coronary Events (GRACE) risk score. Cardiac biomarkers, electrocardiographic findings, echocardiographic parameters, and short-term outcomes were compared across SUA categories.
Results: The majority of participants were aged ≥61 years (52.6%), and 72.5% were male. Higher SUA levels were significantly associated with greater clinical severity, including higher Killip class, worse NYHA functional class, and elevated GRACE risk scores

throughout hospitalization (all p<0.001). Cardiac biomarkers, including troponin-T, troponin-I, and CK-MB, increased progressively with rising SUA levels (all p<0.001). Patients with SUA >7.0 mg/dL demonstrated more severe electrocardiographic abnormalities, greater left ventricular dysfunction, and significantly lower left ventricular ejection fraction values (all p<0.001).

Conclusion: Elevated serum uric acid levels are strongly associated with increased disease severity, myocardial injury, impaired cardiac function, and adverse short-term outcomes in patients with ischaemic heart disease.

Keywords: Serum uric acid; Ischaemic heart disease; Acute coronary syndrome; Prognosis; Left ventricular dysfunction.

INTRODUCTION

Cardiovascular disease, particularly ischaemic heart disease (IHD), remains a major cause of premature mortality and disability in India. [1,2] IHD encompasses a spectrum of clinical conditions, including angina, non-ST-segment elevation myocardial infarction (NSTEMI), and ST-segment elevation myocardial infarction (STEMI). The underlying pathological process involves obstruction of coronary blood flow, resulting in myocardial ischaemia and, when prolonged, irreversible myocardial necrosis.[3] A comprehensive understanding of the mechanisms involved in IHD is essential for improving diagnosis, treatment, risk stratification, and prognostic assessment.[4] Despite advances in cardiovascular care, acute coronary syndromes continue to place a considerable burden on healthcare systems. Current risk assessment relies largely on clinical evaluation, electrocardiographic findings, cardiac biomarkers, and imaging studies. Therefore, there is growing interest in identifying simple, cost-effective biomarkers that can assist in the early identification of high-risk patients without delaying treatment. [1]

Serum uric acid (SUA) has attracted increasing attention as a potential biomarker in cardiovascular disease because of its relationship with oxidative stress, endothelial dysfunction, and cardiorenal impairment. However, its precise clinical significance remains a subject of debate. Several observational studies and clinical cohorts have demonstrated an association between elevated SUA levels and an increased risk of adverse cardiovascular events and mortality following myocardial infarction, although some reports have shown that these associations become less pronounced after adjustment for conventional cardiovascular risk factors. [5]

Uric acid is the final product of purine metabolism and is generated through the enzymatic action of xanthine oxidase. [6] Hyperuricaemia may arise from increased uric acid production, impaired renal excretion, or a combination of both, and is traditionally recognized as an important factor in the development of gout and nephrolithiasis. [7] More recently, elevated uric acid levels have been linked to a variety of cardiovascular and metabolic disorders, including hypertension, atrial fibrillation, chronic kidney disease, heart failure, coronary artery disease, and cardiovascular mortality. [8,9] During uric acid production, xanthine oxidase generates reactive oxygen species that may contribute to oxidative stress and vascular injury. Experimental evidence suggests that elevated uric acid levels can promote inflammation, endothelial dysfunction, and enhanced vasoconstrictor activity, thereby facilitating the progression of cardiovascular disease. [10]

Evidence from high-risk populations indicates that elevated admission SUA levels may be associated with increased short-term mortality and adverse in-hospital outcomes following acute myocardial infarction. [5] Similarly, higher uric acid concentrations have been linked with

impaired myocardial reperfusion and poorer outcomes after coronary intervention, potentially reflecting microvascular dysfunction and reperfusion-related injury. [6,10] These findings support the possibility that SUA may provide prognostic information in patients with acute coronary syndromes.

A plausible biological explanation for this relationship exists. During myocardial ischaemia and hypoxia, accelerated degradation of adenine nucleotides results in increased production of uric acid. Adenosine released from cardiac myocytes during ischaemic stress undergoes rapid metabolism by endothelial cells, ultimately contributing to elevated circulating uric acid levels. [11] Consequently, serum uric acid may reflect the extent of myocardial injury and systemic metabolic stress, making it a potentially useful marker of disease severity and prognosis in IHD. [12] The relevance of this issue is particularly important in India, where substantial regional differences exist in the burden of IHD and its associated risk factors. Variations in socioeconomic conditions, healthcare accessibility, lifestyle patterns, and comorbid diseases contribute to marked heterogeneity in cardiovascular outcomes across the country. Such diversity may limit the universal applicability of existing risk assessment tools and highlights the need for affordable and widely available biomarkers with proven clinical value. [2,13] Furthermore, nationally representative studies have documented a high prevalence of diabetes mellitus and hypertension, both of which influence uric acid metabolism and substantially affect cardiovascular risk. [14] State-level analyses have demonstrated considerable variation in IHD-related mortality and disability across India, including Odisha, emphasizing the importance of validating potential biomarkers within local populations. [2] National mortality studies have also reported differing trends in IHD and stroke-related deaths across regions, further underscoring the need for region-specific evaluation of cardiovascular risk factors and prognostic indicators. [13]

Although serum uric acid estimation is inexpensive, readily available, and routinely performed in clinical practice, its value in assessing the severity of ischaemic heart disease remains incompletely defined. Additional evidence is required to determine whether SUA provides clinically meaningful information beyond conventional laboratory and diagnostic parameters.

Therefore, the present study was undertaken to evaluate the relationship between serum uric acid levels and the severity of ischaemic heart disease. The study also aimed to assess the association of SUA with biochemical parameters, electrocardiographic findings, and echocardiographic characteristics among patients with angina, NSTEMI, and STEMI. By examining these relationships, the study seeks to clarify the potential utility of serum uric acid as a readily available marker of disease severity and prognosis in patients with IHD.

MATERIALS AND METHODS

Study Design and Participants:

This prospective observational study was conducted in the Departments of General Medicine and Cardiology, Hi-Tech Medical College and Hospital, Bhubaneswar, Odisha, from January 2024 to December 2025. Eighty consecutive adult patients diagnosed with ischaemic heart disease (IHD), including angina, ST-segment elevation myocardial infarction (STEMI), and non-ST-segment elevation myocardial infarction (NSTEMI), were enrolled after obtaining written informed consent. Ethical approval was obtained from the Institutional Ethics Committee, Hi Tech Medical College & Hospital, Bhubaneswar Vide Approval No. HMCH/IEC/2024/230(28) prior to study initiation.

Inclusion and Exclusion Criteria: Patients presenting with chest pain and diagnosed with IHD based on clinical assessment, electrocardiography (ECG),

echocardiography, cardiac biomarkers, treadmill testing, or coronary angiography (where indicated) were included. Patients with non-ischaemic heart disease, those receiving medications known to elevate serum uric acid levels, and patients undergoing treatment for hyperuricaemia were excluded. And the final analysed dataset included 80 participants (n=80).

Data Collection:
Baseline demographic and clinical characteristics, cardiovascular risk factors, comorbidities, and examination findings were recorded using a structured study proforma. All participants underwent serum uric acid (SUA) estimation, cardiac biomarker assessment (troponin T, troponin I, and CK-MB), routine biochemical investigations, 12-lead ECG, and two-dimensional echocardiography. Treadmill testing and coronary angiography were performed in selected patients when clinically indicated.

Clinical Assessment and Outcomes:
Disease severity was assessed using Killip classification, New York Heart Association (NYHA) functional class, and Global Registry of Acute Coronary Events (GRACE) risk stratification. Clinical outcomes evaluated included requirement for oxygen support, duration of intensive care unit stay, total hospital stay, in-hospital complications, and mortality.

Serum Uric Acid Categorization:
Patients were categorized according to SUA levels as 4.0–5.5 mg/dL, 5.6–7.0 mg/dL, and >7.0 mg/dL. Associations between SUA levels and disease severity, biochemical parameters, ECG findings, echocardiographic characteristics, and short-term clinical outcomes were analyzed.

Statistical Analysis: Data were analyzed using SPSS version 22. Continuous variables were expressed as mean ± standard deviation, while categorical variables were presented as frequencies and percentages. Group comparisons were performed using one-way ANOVA, Chi-square test, or Fisher's exact test, as appropriate. A p-value <0.05 was considered statistically significant.

RESULTS

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

Age Group in years	Frequency	Percent
31-40	03	3.74
41-50	15	18.75
51-60	20	25.0
61-70	21	26.3
> 70	21	26.3
Gender		
Male	58	72.5
Female	22	27.5

Table 2. Clinical Profile and Cardiovascular Risk Factors Among Patients with Ischaemic Heart Disease (n = 80)

Diagnosis	Frequency	Percent
NSTEMI	33	41.25
STEMI	34	42.5
Angina	13	16.25
Diabetis mellitus		
Yes	32	40.0
No	48	60.0
Hypertension		
Yes	45	56.3
No	35	43.8
Dyslipidemia		

Yes	42	52.5
No	38	47.5
Family History of Ischemic Heart Disease		
Yes	18	22.5
No	62	77.5

Table 3. Association of Serum Uric Acid Levels with Clinical Severity Scores During Hospitalization in Patients with Ischaemic Heart Disease

Severity Score	Category	SUA 4–5.5	SUA 5.6–7	SUA >7	p-value
Killip Class (Day 0)	I	16	14	4	<0.001*
	II	0	9	21	
	III	0	1	11	
	IV	0	0	4	
Killip Class (Day 3)	I	20	20	11	<0.001*
	II	0	5	10	
	III	0	1	5	
	IV	0	0	4	
Killip Class (Day 5)	I	23	16	12	0.001*
	II	0	2	8	
	III	0	1	5	

* P < 0.05 was considered statistically significant.

Table 4. Association of Serum Uric Acid Levels with NYHA Functional Class During Hospitalization in Patients with Ischaemic Heart Disease

NYHA Class	Day 0 SUA 4–5.5	Day 0 SUA 5.6–7	Day 0 SUA >7	Day 3 SUA 4–5.5	Day 3 SUA 5.6–7	Day 3 SUA >7	Day 5 SUA 4–5.5	Day 5 SUA 5.6–7	Day 5 SUA >7
I	8	5	0	15	11	1	20	11	6
II	8	13	8	4	12	10	3	7	9
III	0	6	25	0	3	15	0	1	5
IV	0	0	7	0	0	4	0	0	5
p-value	<0.001*			<0.001*			<0.001*		

* P < 0.05 was considered statistically significant.

Table 5. Association of Serum Uric Acid Levels with GRACE Risk Classification During Hospitalization in Patients with Ischaemic Heart Disease

GRACE Risk Category	Day 0 SUA 4–5.5	Day 0 SUA 5.6–7	Day 0 SUA >7	Day 3 SUA 4–5.5	Day 3 SUA 5.6–7	Day 3 SUA >7	Day 7 SUA 4–5.5	Day 7 SUA 5.6–7	Day 7 SUA >7
Low	11	7	0	14	4	0	23	1	2
Intermediate	5	2	1	5	2	1	0	7	7
High	0	15	39	0	20	29	0	11	16
p-value	<0.001*			<0.001*			<0.001*		

Table 6. Comparison of Biochemical and Cardiac Biomarkers According to Serum Uric Acid Categories

Parameter	SUA 4–5.5	SUA 5.6–7	SUA >7	p-value
Blood Urea (Day 0)	39.9 ± 7.9	47.2 ± 9.5	54.2 ± 11.1	<0.001*
Creatinine (Day 0)	1.09 ± 0.19	1.36 ± 0.40	1.46 ± 0.34	0.002*
SGOT (Day 0)	54.3 ± 15.8	118.4 ± 61.0	132.0 ± 52.9	<0.001*
SGPT (Day 0)	48.5 ± 12.5	61.1 ± 19.7	71.5 ± 19.7	<0.001*
Troponin-T (Day 0)	37.3 ± 35.3	657.4 ± 563.3	1475.0 ± 453.9	<0.001*
Troponin-I (Day 0)	64.8 ± 52.9	842.8 ± 717.7	1592.5 ± 498.9	<0.001*
CK-MB (Day 0)	88.1 ± 15.1	133.6 ± 49.3	160.6 ± 46.3	<0.001*
Troponin-T (Day 3)	67.2 ± 14.9	2059.4 ± 1192.8	2644.8 ± 616.2	<0.001*
Troponin-T (Day 7)	45.4 ± 13.9	712.7 ± 399.8	727.3 ± 397.5	<0.001*

* P < 0.05 was considered statistically significant.

Table 7. Electrocardiographic and Echocardiographic Findings According to Serum Uric Acid Categories

A. Association of Serum Uric Acid Levels with Electrocardiographic Findings During Hospitalization

Assessment Time	χ^2	p-value
Baseline ECG	65.59	<0.001*
Day 3 ECG	70.61	<0.001*
Day 7 ECG	65.69	<0.001*

* P < 0.05 was considered statistically significant.

B. Association of Serum Uric Acid Levels with Left Ventricular Function at Baseline

LV Function	SUA 4–5.5 mg/dL	SUA 5.6–7.0 mg/dL	SUA >7.0 mg/dL
Normal LV Function	11	4	0
Mild LV Dysfunction	5	15	9
Moderate LV Dysfunction	0	5	19
Severe LV Dysfunction	0	0	12
p-value	<0.001*		

* P < 0.05 was considered statistically significant.

C. Comparison of Left Ventricular Ejection Fraction (LVEF) According to Serum Uric Acid Categories

Mean LVEF (%)	SUA 4–5.5	SUA 5.6–7	SUA >7	p-value
Day 0	55.81 ± 2.56	49.96 ± 5.32	39.35 ± 8.41	<0.001*
Day 3	57.00 ± 1.89	51.23 ± 4.74	41.48 ± 7.29	<0.001*
Day 7	57.83 ± 1.92	51.33 ± 4.46	42.33 ± 7.08	<0.001*

* P < 0.05 was considered statistically significant.

Table 8. Association of Serum Uric Acid Levels with Short-Term Clinical Outcomes in Patients with Ischaemic Heart Disease

Outcome	SUA 4–5.5	SUA 5.6–7	SUA >7	p-value
Oxygen Requirement, n (%)	6	8	29	0.003*
PCI/Thrombolysis Required, n (%)	0	2	19	<0.001*
Mortality by Day 3, n (%)	0	0	5	0.015*
Mortality by Day 7, n (%)	0	4	9	0.029*
ICU Stay (days)	0.19 ± 0.40	1.38 ± 1.10	3.78 ± 1.64	<0.001*

*P < 0.05 was considered statistically significant.

Among the 80 study participants, the majority were aged ≥60 years (52.6%), and males constituted 72.5% of the cohort. STEMI (42.5%) and NSTEMI (41.3%) were the most common clinical presentations. Hypertension (56.3%), dyslipidemia (52.5%), and diabetes mellitus (40.0%) were the predominant cardiovascular risk factors (Tables 1 and 2).

Higher serum uric acid (SUA) levels were significantly associated with greater disease severity throughout hospitalization. Patients with SUA >7 mg/dL were predominantly classified in higher Killip, NYHA, and GRACE risk categories, whereas those with lower SUA levels were more frequently observed in lower-risk categories. These associations remained statistically significant at all assessment time points (all p<0.001) (Tables 3–5).

Biochemical and cardiac biomarker analysis demonstrated a progressive increase in renal function markers (blood urea and creatinine), liver enzymes (SGOT and SGPT), and myocardial injury markers (Troponin-T, Troponin-I, and CK-MB) with increasing SUA levels. Patients in the highest SUA category consistently exhibited the greatest biomarker elevations, indicating more severe myocardial injury and systemic involvement (all p≤0.002) (Table 6).

Electrocardiographic and echocardiographic findings also showed significant associations with SUA levels. Higher SUA categories demonstrated more severe ECG abnormalities and poorer recovery patterns during follow-up (all p<0.001). Baseline left ventricular dysfunction was significantly more common among patients with elevated SUA, with moderate and severe dysfunction largely confined to the >7 mg/dL group

(χ^2=57.20, p<0.001). Mean left ventricular ejection fraction (LVEF) decreased progressively with increasing SUA levels at baseline and during follow-up (all p<0.001) (Table 7).

Short-term clinical outcomes were significantly worse among patients with elevated SUA. Individuals with SUA >7 mg/dL had higher oxygen requirements, greater need for PCI/thrombolysis, increased mortality by day 3 and day 7, and longer ICU stays compared with those having lower SUA levels. These findings indicate that elevated serum uric acid is strongly associated.

DISCUSSION

The present study demonstrated that elevated serum uric acid (SUA) levels were significantly associated with greater disease severity and poorer short-term outcomes among patients with ischaemic heart disease (IHD). The study population was predominantly older, with more than half of the patients aged ≥61 years, and showed a marked male predominance. Similar demographic patterns have been reported by Banerjee et al. (2025) and Liu et al. (2021), reflecting the higher burden of coronary artery disease among older adults and men. [15,16] Previous studies have also highlighted sex-related differences in IHD presentation and healthcare utilization, which may explain the predominance of male patients in hospital-based cohorts. [17,18]

Traditional cardiovascular risk factors were highly prevalent in the present cohort. Diabetes mellitus, hypertension, and dyslipidaemia were observed in 40.0%, 56.3%, and 52.5% of patients, respectively, supporting the established role of cardiometabolic disorders in the development and progression of coronary artery disease. [14,19-21] Furthermore, STEMI and NSTEMI together accounted for over 80% of admissions, indicating a predominantly acute coronary syndrome population, comparable to previously reported cohorts. [22,23] The relatively low prevalence of positive family history suggests that acquired risk factors contributed more substantially to disease occurrence in this population. [13,24]

A key finding of the study was the strong association between higher SUA levels and worsening clinical severity. Patients with SUA >7 mg/dL consistently demonstrated higher Killip class, poorer NYHA functional status, and higher GRACE risk scores throughout hospitalization. These observations are in agreement with previous studies showing that elevated uric acid levels are associated with increased heart failure severity, adverse cardiovascular events, and higher risk stratification scores in acute coronary syndromes. [16,25-27] The findings support the concept that SUA reflects underlying oxidative stress, endothelial dysfunction, inflammation, and impaired myocardial perfusion, all of which contribute to disease progression.

Higher SUA levels were also associated with significantly elevated cardiac biomarkers, including troponin-T, troponin-I, and CK-MB. This progressive rise in biomarker levels across SUA categories suggests a greater extent of myocardial injury among patients with hyperuricaemia. Similar relationships between uric acid and myocardial necrosis markers have been reported in studies evaluating risk assessment in acute myocardial infarction. [28,29] Thus, SUA may serve as an indirect indicator of infarct severity and myocardial damage.

Electrocardiographic and echocardiographic findings further supported the adverse prognostic significance of elevated SUA. Patients with higher SUA levels exhibited more severe ECG abnormalities, greater degrees of left ventricular dysfunction, and significantly lower left ventricular ejection fraction during follow-up. These findings are consistent with previous evidence demonstrating an inverse relationship between serum uric acid levels and cardiac function. [30] Elevated SUA may therefore identify patients at increased risk of ventricular dysfunction and adverse cardiac remodeling.

Importantly, elevated SUA was associated with worse short-term clinical outcomes, including increased oxygen requirement, greater need for PCI or thrombolysis, prolonged ICU stay, and higher in-hospital mortality. These findings indicate that hyperuricaemia is not only associated with disease severity but also reflects increased healthcare resource utilization and poorer prognosis. Collectively, the results suggest that SUA, an inexpensive and readily available laboratory parameter, may be useful for early risk stratification and identification of high-risk patients with IHD. While SUA should not replace established clinical scoring systems, its significant association with multiple indicators of disease severity supports its complementary role in routine clinical assessment.

CONCLUSION

The present study demonstrates that elevated serum uric acid levels are significantly associated with greater disease severity, increased myocardial injury, impaired cardiac function, and poorer short-term clinical outcomes in patients with ischaemic heart disease. Patients with serum uric acid levels >7.0 mg/dL consistently exhibited higher Killip, NYHA, and GRACE risk scores, greater elevations in cardiac biomarkers, more severe electrocardiographic and echocardiographic abnormalities, longer ICU stays, and increased in-hospital mortality.

These findings suggest that serum uric acid is a simple, inexpensive, and readily available biomarker that correlates closely with clinical severity and prognosis in hospitalized patients with ischaemic heart disease. Although it should not replace established risk assessment tools, serum uric acid may serve as a valuable adjunct for early risk stratification, identification of high-risk patients, and guiding closer monitoring and timely therapeutic intervention. Further large-scale multicentre studies are warranted to validate its prognostic utility and determine its role in routine cardiovascular risk assessment.

Declaration by Authors

Ethical Approval: The study was conducted after obtaining approval from the Institutional Ethics Committee.

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