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Original Research Article

A Study of Prognostic Importance of Serum Uric Acid Level in Patients with Acute Myocardial Infarction

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Abstract

Introduction: Acute myocardial infarction (AMI) remains a major cause of morbidity and mortality worldwide despite advances in diagnostic and therapeutic strategies. Early identification of patients at increased risk of adverse outcomes is essential for effective risk stratification and optimal management. Serum uric acid, the final product of purine metabolism, has been implicated in oxidative stress, endothelial dysfunction, and inflammation, all of which contribute to cardiovascular injury. Several studies have suggested an association between elevated serum uric acid levels and poor clinical outcomes following AMI. However, its prognostic utility remains controversial across different populations. Therefore, the present study was undertaken to evaluate the prognostic significance of serum uric acid levels in patients presenting with acute ST-segment elevation myocardial infarction.

Materials and Methods: This prospective observational cohort study was conducted in the Department of General Medicine at Ozone Hospitals, Hyderabad, India, between June 2023 and January 2024. A total of 100 consecutive patients diagnosed with acute ST-segment elevation myocardial infarction (STEMI) were enrolled. Demographic characteristics, cardiovascular risk factors, clinical findings, laboratory investigations, electrocardiographic parameters, and echocardiographic findings were recorded at admission. Serum uric acid levels were measured using the enzymatic uricase method. Hyperuricemia was defined as serum uric acid >7.0 mg/dL in men and >5.6 mg/dL in women. Patients with conditions or medications known to influence serum uric acid levels were excluded. All patients were monitored throughout hospitalization for clinical outcomes including mortality, heart failure, and arrhythmias. Clinical characteristics were compared between normal uric acid and hyperuricemia groups. Multivariate logistic regression analysis was performed to identify independent predictors of mortality after adjustment for potential confounding variables. Statistical significance was considered at $p<0.05$.

Results: The study included 100 patients with STEMI, with a mean age of 55.0 ± 9.7 years; 75% were male. Hypertension, smoking, dyslipidemia, and diabetes mellitus were present in 38%, 45%, 32%, and 30% of patients, respectively. Anterior wall myocardial infarction (45%) was the most common presentation, followed by inferior wall myocardial infarction (41%). The mean serum uric acid level was 5.85 ± 1.85 mg/dL. Patients with hyperuricemia were significantly older and had higher serum creatinine levels than those with normal uric acid levels. Short-term mortality was significantly greater in the hyperuricemia group (19.4% vs. 6.4%, $p=0.020$). Non-survivors had significantly higher serum uric acid levels than survivors (6.71 ± 1.38 mg/dL vs. 4.45 ± 1.29 mg/dL, $p<0.001$). Multivariate logistic regression identified serum uric acid as an independent predictor of mortality (adjusted OR 6.76; 95% CI: 1.018–178.540; $p=0.015$).

Conclusion: Elevated admission serum uric acid levels are significantly associated with adverse outcomes in patients with acute ST-segment elevation myocardial infarction. Serum uric acid serves as an independent predictor of mortality and may be a useful, inexpensive biomarker for early risk stratification in acute myocardial infarction.

Keywords: Acute myocardial infarction; Serum uric acid; Hyperuricemia; Prognosis; Mortality.

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Introduction

Acute myocardial infarction (AMI) remains one of the leading causes of morbidity and mortality worldwide despite significant advances in diagnostic and therapeutic strategies. It results from acute interruption of coronary blood flow, leading to myocardial ischemia and necrosis. [1] Early identification of patients at high risk for adverse outcomes is essential for prompt intervention, appropriate risk stratification, and improvement of clinical outcomes. [2] Various biochemical and clinical markers have been investigated to predict prognosis in patients with AMI; however, the search for simple, readily available, and cost-effective prognostic indicators continues. [3]

Serum uric acid, the final product of purine metabolism, has gained considerable attention as a potential marker of cardiovascular risk and prognosis. [4] Elevated serum uric acid levels have been associated with oxidative stress, endothelial dysfunction, inflammation, and impaired myocardial perfusion, all of which contribute to the pathogenesis and progression of cardiovascular disease.5 Increased activity of xanthine oxidase during ischemia leads to excessive production of reactive oxygen species and uric acid, suggesting a possible link between hyperuricemia and adverse outcomes following acute myocardial infarction. [6]

Several studies have reported an association between elevated serum uric acid levels and increased mortality, heart failure, and major adverse cardiac events in patients with AMI. [7,8] However, the prognostic significance of serum uric acid remains controversial, with inconsistent findings across different populations. Considering its widespread availability and low cost, serum uric acid may serve as a useful biomarker for early risk assessment.

Materials and Methods

This prospective observational cohort study was conducted in the Department of General Medicine at Ozone Hospitals, Hyderabad, India, between June 2023 and January 2024. The study included 100 consecutive patients diagnosed with acute ST-segment elevation myocardial infarction (STEMI) who were admitted to the emergency department, intensive care unit, cardiology ward, or general medicine ward during the study period.

The diagnosis of STEMI was established based on a history of typical ischemic chest pain lasting more than 30 minutes, characteristic electrocardiographic changes in two contiguous leads, and elevated cardiac biomarkers. Written informed consent was obtained from all participants, and the study protocol was approved by the Institutional Ethics Committee. Patients with elevated renal parameters, gout, chronic alcoholism, previous ischemic heart disease on aspirin therapy, presentation later than 72 hours after symptom onset, hemolytic disorders, or use of medications known to alter serum uric acid levels such as aspirin, diuretics, ethambutol, and pyrazinamide were excluded. Demographic data, cardiovascular risk factors, clinical history, and baseline laboratory parameters were recorded at admission. Hypertension, diabetes mellitus, dyslipidemia, and smoking status were documented according to standard diagnostic criteria.

Venous blood samples were collected at admission under aseptic precautions for estimation of serum uric acid and other biochemical parameters. Serum uric acid levels were measured using the enzymatic uricase method on an automated analyzer. Additional investigations included complete blood count, blood glucose, renal function tests, serum electrolytes, and lipid profile. All patients underwent a standard 12-lead electrocardiogram and transthoracic echocardiography. Left ventricular ejection fraction (LVEF) was assessed using the biplane Simpson's method. Patients eligible for thrombolytic therapy received intravenous streptokinase followed by standard medical management according to institutional protocols.

Patients were stratified according to sex-specific serum uric acid levels. Hyperuricemia was defined as serum uric acid >7.0 mg/dL in men and >5.6 mg/dL in women. Clinical severity was assessed using the Killip classification at admission. Patients were followed throughout hospitalization and monitored for the development of heart failure, arrhythmias, and mortality. Continuous cardiac monitoring and serial electrocardiographic assessments were performed whenever clinically indicated. Coronary angiography and revascularization procedures were undertaken based on clinical requirements.

The primary outcome measures were in-hospital mortality and short-term mortality. Secondary outcomes included the occurrence of heart failure, arrhythmias, and the association between serum uric acid levels and Killip class. Continuous variables were expressed as mean ± standard deviation, whereas categorical variables were presented as frequencies and percentages.

Comparisons between groups were performed using Student's t-test, Chi-square test, or Fisher's exact test as appropriate. Multivariate logistic regression analysis was used to identify independent predictors of mortality after adjustment for potential confounding variables. A p-value of <0.05 was considered statistically significant.

Results

A total of 100 patients with acute ST-segment elevation myocardial infarction (STEMI) were

included in the study. The baseline demographic and clinical characteristics of the study population are presented in Table 1. The study population had a mean age of 55.0 ± 9.7 years and was predominantly male. Hypertension, smoking, dyslipidemia, and diabetes mellitus were the major cardiovascular risk factors observed. Previous coronary artery disease was present in more than one-third of the study population, while the mean left ventricular ejection fraction was 40.0 ± 9.1%.

Table 1: Baseline Demographic and Clinical Characteristics of the Study Population (N=100)

Variable	Category	n (%)	Variable	Category	Value
Gender	Male	75 (75.0)	Age (years)	Mean ± SD	55.0 ± 9.7
	Female	25 (25.0)	BMI (kg/m²)	Mean ± SD	27.14 ± 1.2
Hypertension	Present	38 (38.0)	Time from chest pain onset (hours)	Mean ± SD	2.5 ± 0.5
	Absent	62 (62.0)	Hospital stay (days)	Mean ± SD	7.1 ± 3.7
DM	Present	30 (30.0)	LVEF (%)	Mean ± SD	40.0 ± 9.1
	Absent	70 (70.0)	Previous PCI	Present	10 (10.0)
Dyslipidemia	Present	32 (32.0)	Previous CABG	Present	4 (4.0)
	Absent	68 (68.0)	Medical Management for CAD	Present	21 (21.0)
Smoking	Present	45 (45.0)	Previous CAD	Present	35 (35.0)
	Absent	55 (55.0)	Previous CAD	Absent	65 (65.0)

Values are expressed as mean ± standard deviation or frequency (percentage). CAD = coronary artery disease; PCI = Percutaneous coronary intervention; CABG = Coronary artery bypass grafting; LVEF = Left ventricular ejection fraction.

The clinical presentation and angiographic characteristics of the study participants are summarized in Table 2.

Anterior wall myocardial infarction was the most common STEMI presentation, followed by inferior wall myocardial infarction. The majority of patients belonged to Killip Class I at admission. Coronary angiography was performed in 41% of patients, among whom significant left anterior descending artery involvement and multivessel coronary artery disease were frequently observed.

Table 2: Clinical Presentation and Angiographic Characteristics of the Study Population (N=100)

Variable	Category	n (%)	Variable	Category	n (%)
Type of MI	AWMI	45 (45.0)	Coronary Angiography	Performed	41 (41.0)
	IWMI	41 (41.0)		Not Performed	59 (59.0)
	ASMI	6 (6.0)	Angiographic Findings*	Significant LAD Disease	30 (73.2)
	LWMI	4 (4.0)		Multivessel Disease	21 (51.2)
	IPMI	4 (4.0)	Patients Undergoing Angiography*	Male	33 (80.5)
Killip Class	I	73 (73.0)		Female	8 (19.5)
	II	27 (27.0)			

Values are expressed as frequency (percentage). *Percentages for angiographic findings and sex distribution were calculated among patients who underwent coronary angiography (n=41). AWMI = Anterior Wall Myocardial Infarction; IWMI = Inferior Wall Myocardial Infarction; ASMI = Anteroseptal Myocardial Infarction; LWMI = Lateral Wall Myocardial Infarction; IPMI = Inferoposterior Myocardial Infarction; LAD = Left Anterior Descending artery.

The distribution of serum uric acid levels according to sex is shown in Table 3. Most patients had serum uric acid concentrations between 6.0 and 7.9 mg/dL. Male patients demonstrated slightly higher mean serum uric acid levels than female patients. Overall, the mean serum uric acid level of the study population was 5.85 ± 1.85 mg/dL.

Table 3: Distribution of Serum Uric Acid Levels According to Sex (N=100)

Serum Uric Acid (mg/dL)	Male (n=75) n (%)	Female (n=25) n (%)	Total (n=100) n (%)
3.0–3.9	4 (5.3)	2 (8.0)	6 (6.0)
4.0–4.9	5 (6.7)	1 (4.0)	6 (6.0)
5.0–5.9	10 (13.3)	5 (20.0)	15 (15.0)
6.0–6.9	20 (26.7)	9 (36.0)	29 (29.0)
7.0–7.9	21 (28.0)	4 (16.0)	25 (25.0)
8.0–8.9	10 (13.3)	3 (12.0)	13 (13.0)
9.0–9.9	5 (6.7)	1 (4.0)	6 (6.0)
Mean Serum Uric Acid (mg/dL)	5.92 ± 1.81	5.64 ± 1.72	5.85 ± 1.85

Values are expressed as frequency (percentage) unless otherwise specified. Percentages for males and females were calculated using their respective group totals (Male n=75, Female n=25), while total percentages were calculated using the overall study population (N=100).

A comparison of clinical characteristics according to serum uric acid status is presented in Table 4. Patients with hyperuricemia were significantly older and had significantly higher serum creatinine levels compared with patients having normal serum uric acid levels. No significant differences were observed with respect to body mass index, cardiovascular risk factors, previous coronary artery disease, symptom-to-hospital presentation time, or duration of hospitalization. However, short-term mortality was significantly more frequent among patients with hyperuricemia.

Table 4: Comparison of Clinical Characteristics According to Hyperuricemia Status

Variable	Normal Uric Acid Group	Hyperuricemia Group	P value
Age (years)	55.1 ± 8.9	62.9 ± 10.9	0.010
BMI (kg/m²)	26.9 ± 0.49	27.6 ± 0.41	0.790
Serum Creatinine (mg/dL)	1.07 ± 0.21	1.19 ± 0.22	0.010
Time from symptom onset (hours)	2.58 ± 0.51	2.74 ± 0.38	0.432
Hospital Stay (days)	6.59 ± 2.71	7.51 ± 2.81	0.310
Hypertension (%)	44.7	37.0	0.130
Diabetes Mellitus (%)	25.5	19.4	0.810
Dyslipidemia (%)	53.2	48.1	0.862
Smoking (%)	66.0	55.0	0.100
Previous CAD (%)	57.4	50.0	0.521
Short-term Mortality (%)	6.4	19.4	0.020

The prognostic significance of serum uric acid is shown in Table 5. Patients who died during follow-up had significantly higher serum uric acid levels than survivors. Multivariate logistic regression analysis further demonstrated that serum uric acid was an independent predictor of mortality after adjustment for age, cardiovascular risk factors, and serum creatinine. These findings suggest that elevated admission serum uric acid is associated with adverse clinical outcomes and may serve as a useful prognostic marker in patients with acute ST-segment elevation myocardial infarction. The comparison of serum uric acid levels between survivors and non-survivors is presented in Figure 2. The mean serum uric acid level among survivors was 4.45 ± 1.29 mg/dL, whereas non-survivors had a significantly higher mean serum uric acid level of 6.71 ± 1.38 mg/dL. This difference was statistically significant ($p<0.001$), indicating that elevated admission serum uric acid levels were strongly associated with mortality in patients with acute ST-segment elevation myocardial infarction.

Table 5: Multivariate Logistic Regression Analysis for Mortality

Predictor	Adjusted Odds Ratio	95% Confidence Interval	P value
Age	1.147	0.995–1.275	0.058
Hypertension	0.346	0.048–2.780	0.341
Diabetes Mellitus	3.365	0.428–26.740	0.224
Dyslipidemia	4.011	0.876–17.600	0.076
Smoking	0.767	0.102–1.645	0.105
Serum Creatinine	6.780	0.096–521.800	0.397
Serum Uric Acid	6.760	1.018–178.540	0.015

Discussion

The present study demonstrated that elevated admission serum uric acid levels were significantly associated with increased short-term mortality in patients with acute ST-segment elevation myocardial infarction (STEMI).

Furthermore, serum uric acid emerged as an independent predictor of mortality after adjustment for conventional cardiovascular risk factors and clinical variables. These findings support the growing body of evidence suggesting that serum uric acid is an important prognostic biomarker in acute myocardial infarction. Similar findings were reported by Sardar et al. [9] (2021), who observed significantly higher rates of acute left ventricular failure and mortality among AMI patients with elevated serum uric acid levels. Kaya et al. [10] (2024) also demonstrated that admission serum uric acid independently predicted in-hospital major adverse cardiovascular events and six-month mortality following myocardial infarction. Likewise, Kang et al. [11] (2024) reported significantly higher uric acid levels among STEMI patients with poor prognosis after complete revascularization, while Kim et al. [12] (2022) found hyperuricemia to be an independent predictor of long-term mortality in a cohort of 5,888 patients with acute myocardial infarction. The consistency of these findings across different populations indicates that serum uric acid may be a reliable marker of adverse cardiovascular outcomes. Elevated uric acid likely reflects increased xanthine oxidase activity, oxidative stress, endothelial

dysfunction, and inflammatory activation during acute ischemic injury, thereby contributing to myocardial damage and worse prognosis.

A major finding of our study was the independent prognostic value of serum uric acid even after adjustment for other clinical variables. Similar observations have been reported by several investigators. Kaya et al. [10] (2024) showed that each incremental increase in serum uric acid significantly increased the risk of in-hospital complications and mortality after AMI. Xu et al. [13] (2025), in a retrospective cohort study of chronic heart failure patients, identified elevated uric acid as an independent predictor of poor prognosis and demonstrated significantly worse survival among patients with uric acid levels above the identified cut-off value. Liu et al. [14] (2021) similarly reported that hyperuricemia independently predicted in-hospital mortality among patients hospitalized with acute cerebral infarction, particularly in those with impaired renal function. These findings suggest that serum uric acid reflects multiple pathophysiological pathways simultaneously, including oxidative stress, renal dysfunction, neurohormonal activation, and systemic inflammation. Therefore, elevated uric acid appears to represent more than a simple biochemical abnormality and may function as an integrated marker of cardiovascular risk. The independent predictive value observed in our study supports the inclusion of serum uric acid in routine risk stratification models for patients presenting with acute myocardial infarction.

The relationship between serum uric acid and the severity of cardiac dysfunction has been highlighted in several recent studies and may explain the adverse outcomes observed in hyperuricemic patients. [15] Sathvik et al. [15] (2024) demonstrated a progressive rise in serum uric acid levels with increasing Killip class and reported significantly higher values among patients with Killip class III and IV compared with those in lower Killip classes. Similarly, Kaya et al. [10] (2024) observed that elevated uric acid levels were associated with higher Killip class, reduced ejection fraction, and a more severe clinical presentation. Kang et al. [11] (2024) reported that patients with poor prognosis had significantly higher uric acid concentrations and lower survival rates than those with favorable outcomes. These findings support the hypothesis that elevated serum uric acid reflects greater myocardial injury and ventricular dysfunction. Increased myocardial ischemia leads to accelerated ATP degradation and activation of xanthine oxidase, resulting in excessive production of uric acid and reactive oxygen species. [16] Consequently, elevated serum uric acid may serve as an indirect indicator of infarct severity, ventricular remodeling, and hemodynamic compromise, thereby explaining its strong association with mortality. [17]

Although most contemporary studies support the prognostic significance of serum uric acid, conflicting findings have occasionally been reported. Mohammed and Rasool [18] (2021) evaluated STEMI patients and found no significant association between serum uric acid levels and 30-day mortality. However, their study involved a relatively smaller sample size and fewer outcome events, which may have limited statistical power. In contrast, larger studies have consistently demonstrated a significant relationship between hyperuricemia and adverse cardiovascular outcomes. Kim et al. [12] (2022) reported that elevated uric acid significantly improved long-term mortality prediction when added to traditional cardiovascular risk factors. Similarly, Kang et al. [11] (2024) demonstrated that both uric acid and NT-proBNP possessed significant prognostic value for long-term survival after STEMI. These findings suggest that discrepancies among studies may be attributable to differences in patient characteristics, treatment strategies, sample size, and duration of follow-up. Overall, the current evidence strongly favors the role of serum uric acid as a clinically relevant prognostic marker in acute myocardial infarction.

Further support for our findings comes from studies evaluating novel uric acid-based prognostic indices. Kalkan et al. [19] (2022) demonstrated that the uric acid-to-albumin ratio (UAR) was independently associated with mortality among STEMI patients undergoing primary PCI. Similarly, Selçuk et al. [20] (2022) reported that UAR was a powerful predictor of new-onset atrial fibrillation during hospitalization and showed superior predictive performance compared with serum uric acid alone. These studies suggest that combining uric acid with other biomarkers reflecting inflammation, nutritional status, or cardiac dysfunction may further enhance prognostic accuracy.[19,15] Importantly, even when used as a standalone biomarker, serum uric acid showed significant prognostic utility in our study. The growing interest in uric acid-based risk models highlights the clinical relevance of this inexpensive and widely available laboratory parameter. The strongest evidence supporting the prognostic importance of serum uric acid originates from large-scale meta-analyses and population-based studies. [22] He et al. [21] (2019), in a meta-analysis involving 8,776 patients with acute coronary syndrome, demonstrated that hyperuricemia significantly increased the risk of major adverse cardiovascular events, all-cause mortality, and cardiovascular mortality. More recently, Rong et al. [23] (2023) performed an updated systematic review and meta-analysis including 41 studies and over 225,000

myocardial infarction patients. They reported that hyperuricemia was associated with significantly higher short-term mortality, long-term mortality, and incidence of major adverse cardiovascular events following myocardial infarction. These findings remained significant even after adjustment for traditional risk factors. The results of our study are therefore strongly supported by contemporary high-quality evidence and contribute further data supporting the clinical utility of serum uric acid in risk stratification. Taken together, the available literature indicates that elevated serum uric acid is consistently associated with poorer outcomes across a wide spectrum of cardiovascular diseases and may serve as an important prognostic biomarker in patients with acute myocardial infarction.

Limitations

The present study has several limitations. First, it was conducted at a single center with a relatively small sample size, which may limit the generalizability of the findings. Second, female patients constituted a smaller proportion of the study population, limiting sex-specific analyses. Third, long-term follow-up outcomes were not assessed. Additionally, serial measurements of serum uric acid and the influence of medications affecting uric acid metabolism were not evaluated.

Conclusion

Elevated admission serum uric acid is an important independent predictor of adverse outcomes in patients with acute ST-segment elevation myocardial infarction. Hyperuricemia appears to reflect increased oxidative stress, inflammation, endothelial dysfunction, and myocardial injury, thereby contributing to worse prognosis. Given its low cost, wide availability, and consistent prognostic value demonstrated across multiple studies, serum uric acid may serve as a useful adjunctive biomarker for early risk stratification and identification of high-risk patients with acute myocardial infarction.

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