Section: General Medicine

Original Research Article

PROGNOSTIC SIGNIFICANCE OF SERUM URIC ACID LEVELS IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION

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Received : 20/07/2026
Received in revised form : 18/08/2026
Accepted : 02/09/2026

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DOI: 10.70034/ijmedph.2026.3.690

Source of Support: Nil,
Conflict of Interest: None declared

Int J Med Pub Health
2026; 16 (3); 4407-4413

ABSTRACT

Background: Acute myocardial infarction (AMI) remains an important cause of morbidity and mortality worldwide. Early identification of patients at increased risk of hemodynamic compromise and left ventricular dysfunction is essential for appropriate risk stratification and management. Serum uric acid (SUA), a final product of purine metabolism, has been increasingly investigated as a marker of oxidative stress, inflammation, endothelial dysfunction and cardiovascular risk. The present study evaluated the prognostic significance of SUA in patients with acute myocardial infarction, particularly its association with Killip class and echocardiographic indices of left ventricular function.

Materials and Methods: This observational cross-sectional study was conducted over 12 months in patients aged >18 years diagnosed with acute myocardial infarction and fulfilling predefined inclusion and exclusion criteria. A total of 100 patients were included. Patients with chronic kidney disease, gout, hematological malignancy, hypothyroidism, chronic alcoholism, and use of drugs known to increase SUA were excluded. Clinical assessment included age, sex, diabetes, hypertension, smoking status and infarct localization. Laboratory investigations included serum uric acid, random blood sugar, renal function tests and fasting lipid profile. Electrocardiography and echocardiography were performed, with particular attention to Killip class, ejection fraction and severity of left ventricular dysfunction. Statistical analysis was performed using GraphPad Prism version 5. Categorical variables were expressed as number and percentage and continuous variables as mean ± standard deviation. A p value <0.05 was considered statistically significant.

Results: The study population consisted of 79 males and 21 females, with 66% belonging to the 41–60-year age group. Mean SUA increased significantly with age, being highest among patients aged >60 years (6.72 ± 1.62 mg/dL; p=0.028). SUA was higher in females than males, although the difference was not statistically significant (p=0.125). No significant association was observed with diabetes, hypertension or smoking. SUA differed significantly according to infarct localization, with the highest mean value in anterior wall myocardial infarction (6.27 ± 1.57 mg/dL; p=0.041). A strong progressive increase in SUA was observed across Killip classes, from 4.76 ± 1.15 mg/dL in Killip I to 7.81 ± 0.48 mg/dL in Killip IV (p<0.001). Patients with ejection fraction <40% had significantly higher SUA (7.03 ± 1.11 mg/dL; p<0.001). Severe echocardiographic dysfunction was associated with a mean SUA of 7.71 ± 0.46 mg/dL (p<0.001). Hyperuricemia was present in 40% of patients.

Conclusion: Elevated SUA was significantly associated with higher Killip class, reduced ejection fraction and greater echocardiographic severity in

patients with AMI. SUA may therefore serve as a simple adjunctive biomarker for early identification of patients with more severe myocardial dysfunction. Prospective studies incorporating clinical outcomes are warranted to establish its independent prognostic value.
Keywords: Acute myocardial infarction; serum uric acid; hyperuricemia; Killip class; ejection fraction; left ventricular dysfunction; prognosis.

INTRODUCTION

Acute myocardial infarction (AMI) remains a major cause of morbidity and mortality worldwide and represents an important manifestation of ischemic heart disease. Despite advances in reperfusion therapy, pharmacological treatment and secondary prevention, AMI continues to be associated with substantial morbidity, particularly among patients who develop heart failure and left ventricular systolic dysfunction. Early identification of patients at increased risk of complications is therefore essential for appropriate clinical management and prognostic assessment. The Killip classification, originally described by Killip and Kimball, is a simple bedside system for grading the severity of heart failure in patients with AMI and has subsequently become an established clinical tool for risk stratification.[1]

Serum uric acid (SUA), the end product of purine metabolism, has attracted considerable interest as a potential cardiovascular biomarker. Although uric acid has antioxidant properties at physiological concentrations, elevated SUA has been associated with oxidative stress, endothelial dysfunction, inflammation and adverse cardiovascular outcomes.[2-4] Contemporary evidence suggests that hyperuricemia is associated with an increased cardiovascular risk profile and may provide prognostic information beyond conventional risk factors.[5]

During myocardial ischemia, depletion of adenosine triphosphate results in increased degradation of adenine nucleotides to hypoxanthine and xanthine. Xanthine oxidoreductase catalyses the subsequent formation of uric acid, with concomitant generation of reactive oxygen species. Increased xanthine oxidase activity may therefore contribute to oxidative stress, endothelial dysfunction and impaired vascular homeostasis during ischemic injury.[6,7] Recent reviews have emphasized the potential role of uric acid and xanthine oxidoreductase-related pathways in the pathophysiology of AMI.[7,8]

Previous clinical studies have demonstrated an association between elevated SUA and adverse outcomes following AMI. Higher SUA has been associated with impaired coronary reperfusion, increased heart failure and increased mortality in selected AMI populations.[9,10] More recent evidence has strengthened this association, suggesting that elevated SUA may provide additional prognostic information when combined with established cardiovascular risk factors.

However, the relationship between SUA and the clinical and echocardiographic severity of AMI, particularly its association with Killip class, infarct localization, left ventricular ejection fraction and degree of ventricular dysfunction, requires further evaluation. Therefore, the present study was undertaken to evaluate the association of SUA with clinical and echocardiographic severity among patients with AMI. The study also assessed the prevalence of hyperuricemia and its relationship with selected cardiovascular risk factors.

Aim

1. To evaluate the prognostic significance of serum uric acid in patients with acute myocardial infarction.

Objectives

1. To assess the association between serum uric acid levels and Killip class in patients with acute myocardial infarction.
2. To evaluate the relationship between serum uric acid levels and left ventricular systolic function, particularly ejection fraction, on echocardiography.

MATERIALS AND METHODS

Study design and setting

This was an observational cross-sectional study conducted over a period of 12 months among patients diagnosed with acute myocardial infarction at Government Dindigul Medical College Hospital. The dissertation describes the study population predominantly in relation to ST-elevation myocardial infarction.

Study population

A total of **100 patients** Patients aged **>18 years** diagnosed with acute myocardial infarction based on clinical history, physical examination, electrocardiographic changes and biochemical evidence of myocardial injury were considered for inclusion.

Data Collection

After obtaining approval from the Institutional Ethics Committee and written informed consent from the patient or nearest relative, eligible patients diagnosed with acute myocardial infarction were enrolled during the 12-month study period. A structured proforma was used for systematic collection of demographic, clinical, laboratory, electrocardiographic and echocardiographic data. Demographic details including age and sex were recorded. A detailed history was obtained regarding diabetes mellitus, hypertension, smoking, dyslipidemia, chronic alcohol intake, previous

cardiovascular illness and relevant drug intake. Complete clinical examination was performed, including assessment of pulse rate, blood pressure and clinical evidence of heart failure. The severity of heart failure at presentation was graded according to the Killip classification. A standard 12-lead electrocardiogram was performed to confirm myocardial infarction and determine the infarct territory, which was categorized as anterior wall myocardial infarction (AWMI), inferior wall myocardial infarction (IWMI) or lateral wall myocardial infarction (LWMI). Blood samples were collected for estimation of serum uric acid, random blood sugar, renal function tests and fasting lipid profile. Patients with chronic kidney disease, gout, hematological malignancy, hypothyroidism, chronic alcoholism and those receiving drugs known to increase serum uric acid were excluded. Two-dimensional echocardiography was performed to assess left ventricular systolic function and ejection fraction. Ejection fraction was categorized as <40%, 41–60% and >60%, and the severity of left ventricular dysfunction was classified as normal, mild, moderate or severe according to the echocardiographic findings. Serum uric acid levels were subsequently compared with demographic characteristics, cardiovascular risk factors, infarct localization, Killip class, ejection fraction and echocardiographic severity. All observations were recorded in a standardized data collection sheet, entered into Microsoft Excel and subsequently analyzed using GraphPad Prism version 5.

Statistical Analysis

Data were coded and entered into Microsoft Excel 2010 and analyzed using **GraphPad Prism version 5**. Continuous variables were expressed as mean ± standard deviation, while categorical variables were expressed as number and percentage. The unpaired Student's *t* test was used for comparison of means between two groups, while Fisher's exact test was used for categorical comparisons where appropriate. For comparisons involving more than two groups, the reported analysis used the corresponding group-comparison approach. A **p value <0.05** was considered statistically significant.

RESULTS

Table 1: Distribution of patients according to age, sex and serum uric acid (N=100)

Variable	Category	Number, n (%)	Serum uric acid, mean ± SD (mg/dL)	p value
Age group	<40 years	13 (13.0)	5.33 ± 1.44	**0.028***
	41–60 years	66 (66.0)	5.89 ± 1.52	
	>60 years	21 (21.0)	6.72 ± 1.62	
Sex	Male	79 (79.0)	5.57 ± 1.49	0.125
	Female	21 (21.0)	6.11 ± 1.59	
Serum uric acid level	<7.0 mg/dL (Normal)	60 (60.0)	4.88 ± 0.95	**<0.001***
	≥7.0 mg/dL (Hyperuricemia)	40 (40.0)	7.66 ± 0.44	

Statistically significant at p<0.05. using Independent-samples t test and One way ANOVA

Among the 100 patients, the majority belonged to the 41–60-year age group (66.0%), followed by those aged >60 years (21.0%) and <40 years (13.0%). Mean serum uric acid increased progressively with age, from 5.33 ± 1.44 mg/dL in patients aged <40 years to 6.72 ± 1.62 mg/dL in those aged >60 years, with a statistically significant difference (p=0.028). Males constituted the majority of the study population (79.0%), while females accounted for 21.0%. Although females had a higher mean serum uric acid level than males (6.11 ± 1.59 vs. 5.57 ± 1.49 mg/dL), the difference was not statistically significant (p=0.125). Hyperuricemia was present in 40.0% of patients, with a mean serum uric acid level of 7.66 ± 0.44 mg/dL, compared with 4.88 ± 0.95 mg/dL among those with normal levels (p<0.001). [Table 1]

Table 2: Association of cardiovascular risk factors with serum uric acid

Risk factor	Category	Number (n)	Serum uric acid, mean ± SD (mg/dL)	p value
Diabetes	Present	35	5.90 ± 1.52	0.670
	Absent	65	6.04 ± 1.61	
Hypertension	Present	43	5.86 ± 1.56	0.216
	Absent	57	6.09 ± 1.58	
Smoking	Present	53	6.24 ± 1.48	0.098
	Absent	47	5.72 ± 1.65	
Dyslipidemia	Present	10	6.88 ± 0.92	0.060
	Absent	90	5.89 ± 1.60	

No statistically significant association was observed between serum uric acid and diabetes, hypertension, smoking, or dyslipidemia. The mean serum uric acid level was 5.90 ± 1.52 mg/dL among patients with diabetes compared with 6.04 ± 1.61 mg/dL among those without diabetes (p=0.670). Similarly, patients with hypertension had a mean level of 5.86 ± 1.56 mg/dL, compared with 6.09 ± 1.58 mg/dL in those without hypertension (p=0.216). Smokers had somewhat higher serum uric acid levels than non-smokers (6.24 ± 1.48 vs. 5.72 ± 1.65 mg/dL), but this difference did not reach statistical significance

(p=0.098). Patients with dyslipidemia showed a higher mean serum uric acid level (6.88 ± 0.92 mg/dL) than those without dyslipidemia (5.89 ± 1.60 mg/dL); however, the difference was not statistically significant (p=0.060). [Table 2]

Table 3: Association between infarct localization and serum uric acid

MI type	Number, n (%)	Serum uric acid, mean ± SD (mg/dL)	p value
AWMI	60 (60.0)	6.27 ± 1.57	**0.041***
IWMI	29 (29.0)	5.65 ± 1.55	
LWMI	11 (11.0)	5.40 ± 1.46	
Total	**100 (100)**	—	

Statistically significant at p<0.05. using One way ANOVA

AWMI was the most common infarct localization, accounting for 60.0% of cases, followed by IWMI (29.0%) and LWMI (11.0%). Patients with AWMI had the highest mean serum uric acid level (6.27 ± 1.57 mg/dL), followed by IWMI (5.65 ± 1.55 mg/dL) and LWMI (5.40 ± 1.46 mg/dL). The difference in serum uric acid levels according to infarct localization was statistically significant (p=0.041). [Table 3]

Table 4: Association between Killip class and serum uric acid

Killip class	Serum uric acid, mean ± SD (mg/dL)	p value
I	4.76 ± 1.15	**<0.001***
II	5.67 ± 0.83	
III	7.27 ± 0.56	
IV	7.81 ± 0.48	

Statistically significant at p<0.05. using One way ANOVA

A strong and statistically significant relationship was observed between Killip class and serum uric acid (p<0.001). Mean serum uric acid increased progressively with increasing Killip class, from 4.76 ± 1.15 mg/dL in class I to 5.67 ± 0.83 mg/dL in class II, 7.27 ± 0.56 mg/dL in class III, and 7.81 ± 0.48 mg/dL in class IV. Thus, higher serum uric acid levels were associated with increasing clinical severity of heart failure at presentation. [Table 4]

Table 5: Association between ejection fraction and serum uric acid

Ejection fraction	Serum uric acid, mean ± SD (mg/dL)	p value
<40%	7.03 ± 1.11	<0.001*
41–60%	4.66 ± 0.79	
>60%	4.19 ± 0.73	

Statistically significant at p<0.05. using One way ANOVA

Patients with reduced ejection fraction had substantially higher serum uric acid levels. The mean serum uric acid was 7.03 ± 1.11 mg/dL among patients with an ejection fraction <40%, compared with 4.66 ± 0.79 mg/dL among those with an ejection fraction of 41–60% and 4.19 ± 0.73 mg/dL among those with an ejection fraction >60%. The difference was highly statistically significant (p<0.001), indicating an inverse relationship between serum uric acid and left ventricular systolic function. [Table 5]

Table 6: Association between echocardiographic severity and serum uric acid

Echocardiographic status	Serum uric acid, mean ± SD (mg/dL)	p value
Normal	4.30 ± 0.80	0.001*
Mild dysfunction	4.67 ± 0.77	
Moderate dysfunction	6.25 ± 1.14	
Severe dysfunction	7.71 ± 0.46	

***Statistically significant at p<0.05. using One way ANOVA**

Serum uric acid demonstrated a significant increasing trend with worsening echocardiographic dysfunction (p<0.001). The mean serum uric acid increased from 4.30 ± 0.80 mg/dL in patients with normal echocardiographic findings to 4.67 ± 0.77 mg/dL in mild dysfunction, 6.25 ± 1.14 mg/dL in moderate dysfunction, and 7.71 ± 0.46 mg/dL in severe dysfunction. This finding suggests that higher serum uric acid levels were associated with greater severity of cardiac dysfunction. [Table 6]

Table 7: Comparison of serum uric acid and ejection fraction according to hyperuricemia status

Hyperuricemia status	Number, n (%)	Serum uric acid, mean ± SD (mg/dL)	Ejection fraction, mean ± SD (%)	p value
Hyperuricemia present	40 (40.0)	7.66 ± 0.44	31.00 ± 5.35	**<0.001***
Hyperuricemia absent	60 (60.0)	4.88 ± 0.95	44.58 ± 11.72	
Overall	**100 (100)**	**5.99 ± 1.58**	—	

Statistically significant at p<0.05. using Independent-samples t test.

Hyperuricemia was present in 40 (40.0%) patients. The mean serum uric acid level was significantly higher in hyperuricemic patients than in those without hyperuricemia (7.66 ± 0.44 vs. 4.88 ± 0.95 mg/dL; p<0.001). Furthermore, hyperuricemic patients had a significantly lower mean ejection fraction (31.00 ± 5.35%) compared with patients without hyperuricemia (44.58 ± 11.72%; p<0.001). These findings demonstrate a significant association between hyperuricemia and impaired left ventricular systolic function. [Table 7]

DISCUSSION

The present study evaluated the association between serum uric acid (SUA) and clinical and echocardiographic severity among 100 patients with acute myocardial infarction (AMI). The major findings were a significant increase in SUA with advancing age and significant associations of SUA with infarct localization, Killip class, left ventricular ejection fraction and echocardiographic severity. Hyperuricemia was present in 40% of patients and was associated with substantially lower ejection fraction. These findings indicate that SUA may reflect the severity of myocardial injury and left ventricular dysfunction in patients presenting with AMI.

In the present study, 79% of patients were male and 66% belonged to the 41–60-year age group. SUA increased significantly with age, from 5.33 ± 1.44 mg/dL in patients aged <40 years to 6.72 ± 1.62 mg/dL in those aged >60 years (p=0.028). However, no significant association was observed between SUA and sex. Similarly, diabetes mellitus, hypertension, smoking and dyslipidemia were not significantly associated with SUA. Borghi et al. emphasized that hyperuricemia frequently accompanies cardiovascular and metabolic disorders and may reflect the complex interaction between uric acid metabolism, vascular dysfunction and cardiovascular risk.[11]

A significant association was observed between infarct localization and SUA, with the highest mean SUA among patients with AWMI (6.27 ± 1.57 mg/dL; p=0.041). More importantly, SUA showed a strong progressive relationship with Killip class. Mean SUA increased from 4.76 ± 1.15 mg/dL in Killip class I to 7.81 ± 0.48 mg/dL in class IV (p<0.001). Lazzeri et al. reported that SUA measured early during STEMI may contribute to risk stratification, supporting the possibility that SUA reflects the severity of acute myocardial injury.[12] Liu et al. further demonstrated an interaction between hyperuricemia and Killip class in patients with STEMI undergoing primary PCI, with higher mortality observed among hyperuricemic patients in selected Killip categories.[13]

The association between SUA and left ventricular systolic function was particularly striking. Patients with EF <40% had a mean SUA of 7.03 ± 1.11 mg/dL compared with 4.66 ± 0.79 mg/dL among those with EF 41–60% and 4.19 ± 0.73 mg/dL among those with EF >60% (p<0.001). Similarly, SUA increased progressively from 4.30 ± 0.80 mg/dL in patients with normal echocardiographic findings to 7.71 ± 0.46 mg/dL in those with severe dysfunction (p<0.001). Nakanishi et al. demonstrated an association between higher SUA and subclinical left ventricular dysfunction in a community-based cohort, supporting a possible relationship between uric acid metabolism and myocardial functional impairment.[14]

Hyperuricemia was present in 40% of the present study population. Hyperuricemic patients had a mean SUA of 7.66 ± 0.44 mg/dL compared with 4.88 ± 0.95 mg/dL in patients without hyperuricemia (p<0.001). Importantly, hyperuricemic patients also had significantly lower mean EF (31.00 ± 5.35% vs. 44.58 ± 11.72%; p<0.001). Uric acid may therefore act not merely as a metabolic marker but also as an indicator of the biological processes accompanying myocardial ischemia, including oxidative stress, endothelial dysfunction and inflammatory activation.[15,16]

The prognostic relevance of elevated SUA has also been demonstrated in patients with AMI. Kim et al. reported that high SUA provided additional long-term prognostic information when combined with traditional cardiovascular risk factors, supporting its potential role as an adjunctive risk marker.[17] Mandurino-Mirizzi et al. similarly demonstrated that elevated SUA was associated with a greater inflammatory response and adverse outcomes in patients with STEMI undergoing primary PCI.[18] These findings are consistent with the present observation that patients with higher SUA had greater clinical and echocardiographic severity.

The evidence is further strengthened by recent systematic reviews and meta-analyses. Rong et al., in an updated meta-analysis involving 41 studies and 225,600 patients with MI, reported significantly higher short-term mortality, short-term MACE, long-term mortality and long-term MACE among patients with elevated SUA.[19] More recently, Sun et al. analyzed 40 cohort studies involving 105,609 patients with ACS and found that high SUA was associated with increased all-cause mortality, cardiovascular mortality, MACE and heart failure. A nonlinear relationship between SUA and mortality was also identified, suggesting that SUA may have prognostic value across different levels of cardiovascular risk.[20]

The findings of the present study therefore support the potential usefulness of SUA as a simple, inexpensive and readily available adjunctive marker of AMI severity. The progressive increase in SUA across Killip classes and the inverse relationship between SUA and ejection fraction are particularly noteworthy. However, the present study was observational and involved only 100 patients. In addition, renal function, medications and other

metabolic factors may influence SUA concentrations. Mortality and long-term MACE were not directly assessed. Therefore, elevated SUA should be regarded as an adjunctive prognostic marker rather than an established independent predictor of outcome.

CONCLUSION

The present study demonstrates that serum uric acid (SUA) is significantly associated with clinical and echocardiographic severity in patients with acute myocardial infarction (AMI). SUA increased progressively with advancing age and was significantly higher among patients with anterior wall myocardial infarction. A strong relationship was observed between SUA and Killip class, with progressively higher levels in patients with increasing clinical severity. Similarly, elevated SUA was significantly associated with reduced left ventricular ejection fraction and worsening echocardiographic dysfunction.

Hyperuricemia was present in 40% of the study population. Patients with hyperuricemia had significantly lower mean ejection fraction than those without hyperuricemia (31.00 ± 5.35% vs. 44.58 ± 11.72%; $p<0.001$), suggesting that elevated SUA may identify patients with greater myocardial and ventricular dysfunction.

SUA is an inexpensive, routinely available and easily measurable biomarker that may provide useful additional information for early risk assessment in AMI. However, because this was a relatively small observational study and potential confounding factors such as renal function and medications may influence SUA, it should be considered an adjunctive prognostic marker rather than an independent determinant of outcome. Larger prospective studies with serial SUA measurements, multivariable analysis and long-term follow-up are warranted to establish its independent prognostic value and clinical utility in AMI.

Funding: Nil

Conflict of interest: The authors wish to confirm that there is no conflict of interest associated with this paper and there has been no significant financial support for this study.

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