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Original Research Article

Study of serum uric acid levels in patients with acute coronary syndrome and its correlation with clinical features and Killip classification in patients attending tertiary care hospital in Kumaon region of Uttarakhand

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ABSTRACT

Background: Acute coronary syndrome (ACS) is a major cause of morbidity and mortality worldwide. Serum uric acid has been suggested as a potential biomarker associated with oxidative stress and cardiovascular risk. This study aimed to evaluate the role of serum uric acid as a prognostic marker in patients with ACS and to assess its association with the Killip classification.

Methods: This prospective study was conducted in the Department of Medicine at Government Medical College and Dr. Susheela Tiwari Hospital, Haldwani, from January 2020 to September 2021. A total of 100 patients with confirmed ACS were included based on clinical presentation, ECG changes and elevated cardiac biomarkers. Serum uric acid levels were measured on day 1 and day 3 of admission. Patients were classified according to the Killip classification and clinical outcomes including mortality, complications and duration of hospital stay were recorded.

Results: Among the 100 patients, the mean age was 60.97±10.8 years and 63% were males. Hyperuricemia (>8 mg/dl) was observed in 30% of patients on day 1 and 24.49% on day 3. Higher uric acid levels were significantly associated with increased mortality, higher Killip class and greater disease severity ($p<0.0001$). Patients with hyperuricemia also showed higher prevalence of dyspnoea, hypertension, positive troponin levels, STEMI presentation and cardiovascular complications. Additionally, the duration of hospital stay was significantly longer among patients with elevated uric acid levels.

Conclusions: Elevated serum uric acid levels are significantly associated with increased severity, complications and mortality in patients with ACS. Serum uric acid may serve as a simple and useful prognostic biomarker for risk stratification in ACS patients.

Keywords: Acute coronary syndrome, Hyperuricemia, Killip classification, Prognostic marker, Serum uric acid

INTRODUCTION

Acute coronary syndrome (ACS) encompasses unstable angina, ST-elevation myocardial infarction (STEMI) and non-ST elevation myocardial infarction (NSTEMI), representing acute clinical outcomes of coronary artery disease (CAD). The predominant mechanism involves rupture of atherosclerotic plaques with subsequent thrombus formation in coronary vessels, though vasospasm without significant atherosclerosis can also precipitate ACS.[1] Globally, ACS remains a critical health challenge, contributing to nearly one-third of all deaths among individuals over 35 years and ranking among the foremost causes of mortality.[2,3] In India, the burden of CAD has escalated in parallel with the broader rise in cardiovascular diseases, with the country's age-standardized CVD mortality rate surpassing the global average (282 vs 233 per 100,000).[4] The number of affected

individuals has nearly doubled from 271 million in 1990 to 523 million in 2019, accompanied by substantial economic consequences, highlighting the widespread impact across both rural and urban settings.[4,5] This surge reflects rapid epidemiological transition, urbanization and heightened exposure to cardiovascular risk factors, emphasizing the urgent need for improved diagnostic and risk stratification strategies.

The etiology of CAD and ACS is multifactorial, involving immutable determinants such as age, sex, genetic predisposition and ethnicity, alongside modifiable contributors including hypertension, dyslipidemia, diabetes, smoking, obesity, sedentary lifestyle, poor diet and psychosocial stress.[6-9] Hypertension and dyslipidemia are particularly influential in accelerating atherosclerosis, often coexisting with diabetes and left ventricular hypertrophy. Elevated LDL-C fosters plaque development, whereas HDL-C offers protection via reverse cholesterol transport, with each 1 mg/dL rise in HDL-C lowering coronary risk by approximately 2% in men and 3% in women.[9] Smoking nearly doubles the risk of ischemic heart disease, while diabetes and obesity further amplify cardiovascular complications, with diabetic individuals facing a two- to four-fold higher risk.[9-11]

Excess adiposity contributes to metabolic and structural cardiac changes, fueling CAD progression. Cardiovascular diseases account for nearly three-quarters of deaths in low- and middle-income nations, underscoring their global significance.[12,13] In India, mortality has risen sharply, with annual deaths increasing from 2.26 million in 1990 to about 4.77 million in 2020.[14] Among ACS presentations, myocardial infarction carries high early mortality, particularly within the first hours of onset, making early risk stratification essential. The Killip classification remains a cornerstone for assessing heart failure severity and prognosis.[15] Biomarkers such as troponins, BNP/NT-proBNP, copeptin, CRP, ST2, GDF-15, eGFR and cystatin C have been explored for prognostic utility.[16] Recently, serum uric acid has emerged as a potential marker, reflecting oxidative stress, inflammation and impaired renal clearance, all linked to atherosclerosis and myocardial injury.[17] Despite growing interest, its prognostic role in ACS remains uncertain, warranting further investigation. This study therefore seeks to evaluate serum uric acid as a prognostic indicator in ACS and its correlation with Killip classification, aiming to enhance risk stratification in this vulnerable population.

METHODS

Study design and setting

This prospective study was conducted in the Department of Medicine at Government Medical College and the associated Dr. Susheela Tiwari Hospital, Haldwani, Uttarakhand. The study was carried out over a period from January 2020 to September 2021.

Study population

The study population consisted of patients admitted to the Department of Medicine with documented evidence of ACS, confirmed by electrocardiographic changes and elevated cardiac biomarkers such as troponin T and creatine kinase. Patients were admitted through the emergency department or Medicine outpatient department. A total of 100 patients diagnosed with acute myocardial infarction were included in the study.

Inclusion criteria

Patients aged more than 18 years presenting with a history of chest pain lasting more than 30 minutes, electrocardiographic evidence of STEMI or NSTEMI and elevated serum cardiac biomarkers such as troponin T or creatine kinase-MB were included in the study.

Exclusion criteria

Patients with chronic kidney disease, known hypothyroidism, malignancy, gout or other inflammatory diseases, chronic alcoholism or recent myocardial infarction within the previous three months were excluded. Patients receiving medications known to elevate serum uric acid levels such as high-dose salicylates (>2 g/day), diuretics, ethambutol and pyrazinamide were also excluded. Additionally, patients with diabetes mellitus were not included in the study.

Methodology

Detailed clinical history was obtained from patients and their relatives, followed by a comprehensive physical examination with emphasis on the cardiovascular system. Data were recorded using a predesigned proforma. Blood pressure was measured using a mercury sphygmomanometer and a 12-lead electrocardiogram was performed for all patients. Laboratory investigations included hemoglobin estimation using Sahli's method, total leukocyte count using a haemocytometer, differential leukocyte count through peripheral smear examination with Leishman stain, platelet count by automated cell counter and renal and liver function tests using standard biochemical methods. Cardiac biomarkers including CK-NAC, CK-MB and troponin T were assessed and arterial blood gas analysis was performed when required.

Diagnosis of myocardial infarction was based on the presence of at least two of the following criteria chest pain lasting more than 20 minutes, characteristic ECG changes and elevated cardiac biomarkers. Serum uric acid levels were measured using an autoanalyzer (ROCHE COBAS C501) based on the modified Trinder method. Venous blood samples were collected preferably after a minimum fasting period of four hours. Hyperuricemia was defined as serum uric acid levels greater than 8 mg/dl. Hypertension was defined as systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg based on the average

of two readings and current smoking was defined as smoking at least one cigarette daily for the preceding year. All patients were clinically assessed according to the Killip classification at admission. Serum uric acid levels were measured on day 0 and day 3 of hospitalization and other laboratory parameters were obtained on the first day of admission for screening and exclusion purposes. Patients were followed for seven days or until discharge, whichever occurred earlier and mortality and morbidity outcomes during the first week of hospitalization were recorded.

RESULTS

The study included 100 patients diagnosed with acute coronary syndrome. The majority of participants were males (63%), with a mean age of 60.97±10.8 years. Chest pain was the most common presenting symptom (97%), followed by dyspnoea (33%). Hypertension (55%) and smoking (37%) were the most frequently observed comorbidities. Among ACS types, unstable angina (46%) was most common, followed by STEMI (37%) and NSTEMI (17%) (Table 1).

Serum uric acid levels were measured on day 1 and day 3 of admission. Hyperuricemia (>8 mg/dl) was observed in 30% of patients on day 1 and 24.49% on day 3. Most patients were classified under Killip class I (40%), followed by class II (33%), class III (18%) and class IV (9%). Mean uric acid levels showed a decreasing trend from day 1 to day 3 (Table 2). A significant association was observed between serum uric acid levels and both mortality and Killip classification. Mortality was significantly higher among patients with hyperuricemia on both day 1 and day 3 ($p<0.0001$). Additionally, higher Killip classes were associated with progressively higher mean uric acid levels, indicating a strong correlation between hyperuricemia and severity of heart failure in ACS patients (Table 3).

Serum uric acid levels showed significant associations with several clinical parameters. Dyspnoea, hypertension, positive troponin levels, STEMI presentation and cardiovascular complications were significantly more common among patients with hyperuricemia. Furthermore, patients with elevated uric acid had a significantly longer hospital stay compared to those with normal uric acid levels (7.83±3.2 vs 5.14±0.73 days, $p<0.0001$) (Table 4).

Table 1: Baseline demographic and clinical characteristics of study participants (n=100).

Variable	Frequency	%
Age (in years)		
40–50	23	23
51–60	31	31
61–70	28	28
71–80	14	14
81–90	4	4
Mean±SD	60.97±10.8	
Gender		
Male	63	63
Female	37	37
Symptoms		
Chest pain	97	97
Dyspnoea	33	33
Other symptoms	53	53
Comorbidities		
Hypertension	55	55
Smoking	37	37
Cardiovascular disease	12	12
Type of ACS		
Unstable angina	46	46
STEMI	37	37
NSTEMI	17	17

Table 2: Distribution of serum uric acid levels and Killip classification among study subjects.

Parameter	Frequency	%
Uric acid day 1		
Normal (≤8 mg/dl)	70	70
Hyperuricemia (>8 mg/dl)	30	30

Continued.

Parameter	Frequency	%
Mean±SD	7.53±2.32	
Uric acid day 3		
Normal (≤8 mg/dl)	74	75.51
Hyperuricemia (>8 mg/dl)	24	24.49
Mean±SD	6.99±2.18	
Killip class		
Class I	40	40
Class II	33	33
Class III	18	18
Class IV	9	9

Table 3: Association of serum uric acid levels with mortality and Killip classification.

Variable	Normal UA (≤8 mg/dl)	Hyperuricemia (>8 mg/dl)	P value
Mortality (day 1 UA)			
Alive	70	21	
Died	0	9	<0.0001
Mortality (day 3 UA)			
Alive	74	17	
Died	0	7	<0.0001
Killip class vs mean UA (day 1)			
Class I	5.64±1.05	—	
Class II	7.44±1.16	—	
Class III	9.81±0.85	—	
Class IV	11.71±2.18	—	<0.0001
Correlation coefficient (r)	0.839		<0.0001

Table 4: Association of serum uric acid levels with clinical features and outcomes.

Variable	Normal UA (≤8 mg/dl) n=70	Hyperuricemia (>8 mg/dl) n=30	P value
Dyspnoea	7 (10%)	26 (86.67%)	<0.0001
Hypertension	30 (42.86%)	25 (83.33%)	0.0002
Troponin positive	27 (38.57%)	27 (90%)	<0.0001
STEMI	13 (18.57%)	24 (80%)	<0.0001
Any complications	2 (2.86%)	15 (50%)	<0.0001
Hospital stay (days)	5.14±0.73	7.83±3.2	<0.0001

DISCUSSION

Cardiovascular disease (CVD) continues to dominate as the foremost cause of death globally, exerting a heavy public health burden despite its largely preventable nature. Lifestyle-related exposures such as poor diet, sedentary behavior and tobacco use are pivotal drivers of coronary heart disease (CHD), acting through intermediates like hypertension, dyslipidemia and metabolic dysfunction.

Epidemiological data consistently highlight that even modest improvements in these risk factors yield substantial reductions in cardiovascular events for example, lowering systolic blood pressure by 5 mmHg can reduce cardiovascular risk by nearly 20%.[18] Against this backdrop, the present study's findings are interpreted in relation to prior evidence to clarify their clinical significance. The mean age of participants in this study was 60.97±10.8 years, with the majority clustered in the 51–60 years range. This aligns with earlier reports indicating that acute myocardial infarction (AMI) tends to occur at younger ages in Indian populations compared to Western cohorts. Although this age group accounted for the highest proportion of cases and deaths, statistical analysis did not confirm age as an independent predictor of outcomes. In contrast, a UAE-based study reported a median age of 40 years, where age was significantly linked to risk stratification and in-hospital complications.[19] Such differences suggest that the prognostic role of age may vary across populations, influenced by risk factor distribution and healthcare accessibility. Gender analysis revealed a male predominance, with men comprising 63% of cases. Previous studies have noted higher early mortality among women following myocardial infarction,

often attributed to delayed presentation and greater comorbidity, though adjusted analyses suggest women may have better long-term outcomes.[20,21] In this cohort, however, gender was not significantly associated with mortality. Similarly, while serum uric acid levels were slightly higher in men, no significant sex-related differences were observed in biochemical or clinical outcomes, indicating that gender did not independently influence prognosis in this population.

Biochemical markers provided critical prognostic insights. Troponin positivity was present in 54% of patients and was significantly more common among those with hyperuricemia. Importantly, all deaths occurred in patients with elevated troponin levels, reaffirming its established role as a marker of myocardial injury and adverse outcomes. These findings echo Khullar et al who demonstrated that troponin T elevation at admission strongly predicts adverse events in STEMI.[22] The overlap between troponin elevation and hyperuricemia suggests shared mechanisms, possibly involving oxidative stress and myocardial damage, positioning uric acid as a complementary prognostic marker. Indeed, all deaths in this study occurred among hyperuricemic patients, consistent with prior reports linking elevated uric acid to short-term and cardiovascular mortality.[23,24] This supports the view that uric acid reflects heightened metabolic and inflammatory stress rather than serving as a direct causal factor, yet remains clinically valuable for risk stratification.

Clinical presentation and risk factors further contextualized outcomes. Chest pain was reported in 97% of patients, consistent with classical ACS symptoms, though reliance on typical presentations risks overlooking atypical cases.[25] Killip classification revealed most patients in lower classes, indicating preserved hemodynamic status, though higher classes carried greater risk, where uric acid may add prognostic value.[26] Smoking prevalence was 37%, higher than the Östersund cohort (18.9%), underscoring tobacco's persistent role in ACS risk.[27,28]

Dyspnea was more frequent among hyperuricemic patients, suggesting reduced cardiac reserve. Hypertension was present in nearly half of patients and significantly associated with hyperuricemia, reflecting shared mechanisms such as endothelial dysfunction and vascular inflammation.[29] Subtype distribution showed unstable angina as most common, differing from studies where STEMI predominated, pointing to regional variations.[30] Hyperuricemia was also linked to in-hospital complications, particularly conduction abnormalities, consistent with prior evidence of increased cardiovascular complications in such patients.[31] Hospital stays were longer among hyperuricemic individuals, echoing findings from the Qingdao cohort where hyperuricemia predicted adverse events at one year.[32] Overall mortality was 9%, with significantly higher rates among patients with elevated uric acid, consistent with Lazzeri et al, who found uric acid independently predicted in-hospital mortality in STEMI patients undergoing PCI.[33]

Limitations

The present study has several limitations. The sample size was relatively small and derived from a single center, which may limit the generalizability of the findings. The observational design precludes establishing a causal relationship between serum uric acid and adverse outcomes. Potential confounding factors such as renal function, medication use and dietary influences on uric acid levels were not fully controlled. Additionally, long-term follow-up data were not available, restricting assessment of the prognostic value of uric acid beyond the in-hospital period. Larger, multicentric studies with longitudinal follow-up are required to validate these findings and establish the independent prognostic role of serum uric acid in acute coronary syndrome.

CONCLUSION

The present study demonstrates that elevated serum uric acid levels are significantly associated with increased severity and poorer outcomes in patients with acute coronary syndrome. Hyperuricemia showed a strong correlation with higher Killip class, greater incidence of complications, longer hospital stay and increased in-hospital mortality. Patients with elevated uric acid levels also had higher prevalence of hypertension, dyspnea and positive troponin levels. These findings suggest that serum uric acid may serve as a simple and useful prognostic biomarker for risk stratification in patients presenting with acute coronary syndrome.

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