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Original Research Article

CORRELATION OF SERUM URIC ACID WITH KILLIP GRADING IN ACUTE MYOCARDIAL INFARCTION

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ABSTRACT

Background: Acute myocardial infarction (AMI) is the most common cause of morbidity and mortality globally. The Killip classification is a straightforward and recognized clinical instrument utilized to evaluate the severity of heart failure in individuals with AMI. Uric acid, a terminal product of purine metabolism, is widely acknowledged as a biomarker for oxidative stress, inflammation, and endothelial dysfunction, all of which have a role in the development and consequences of AMI. The present study was undertaken to evaluate the correlation between serum uric acid levels and Killip class in patients presenting with acute myocardial infarction.

Materials and Methods: This prospective observational study was conducted over an 8-month duration in the Departments of General Medicine and Cardiology at a tertiary care teaching hospital. A total of 120 patients diagnosed with AMI, determined through clinical presentation, electrocardiographic alterations, and cardiac biomarkers, were enrolled following the acquisition of informed permission. A comprehensive clinical history and physical examination were documented. The Killip class was determined upon admission according to the clinical manifestations of heart failure. Serum uric acid levels were assessed within 24 hours of admission by enzymatic colorimetric techniques. Data were evaluated utilizing suitable statistical techniques, to evaluate the association between blood uric acid levels and Killip class.

Results: The review found that litigation under the CPA is more frequent in private healthcare settings, with a high prevalence in surgical and obstetric specialties. Key causes of legal action include lack of informed consent, inadequate documentation, and poor communication. The CPA 2019 amendment introduced new challenges by increasing the scope of jurisdiction and expediting complaint procedures. Defensive medical practices, increased insurance claims, and institutional legal preparedness were also found to be evolving trends in response to rising litigation.

Conclusion: The study revealed a notable positive correlation between blood uric acid levels and Killip class in individuals with AMI. Increased serum uric acid may function as a straightforward, cost-effective, and valuable biomarker for predicting the degree of heart failure and clinical outcomes in AMI.

Keywords: Acute Myocardial Infarction, Heart Failure, Killip Class, Serum Uric Acid.

INTRODUCTION

Acute myocardial infarction (AMI) continues to be a serious worldwide health issue, contributing to considerable morbidity and mortality despite improvements in diagnostic techniques and treatment approaches.[1] The World Health Organization (WHO) estimates that ischemic heart disease accounts for approximately one-third of global mortality, with its incidence increasing in developing nations due to the escalating prevalence of risk factors including diabetes, hypertension, smoking, dyslipidemia, and sedentary habits. Timely

diagnosis, early risk assessment, and suitable care are essential for enhancing survival rates and minimizing morbidity related to AMI.[2,3]

Risk stratification instruments have traditionally been utilized in clinical settings to classify patients according to sickness severity and to forecast prognosis. The Killip classification, first in 1967, has consistently served as a straightforward, practical, and well endorsed bedside instrument.[4]

The Killip classification, based only on clinical examination findings of pulmonary congestion and cardiogenic shock, categorizes patients into four groups: Class I indicates no evidence of heart failure, Class II denotes mild heart failure, Class III signifies significant pulmonary edema, and Class IV represents cardiogenic shock.[5] Elevated Killip classes are consistently linked to poorer outcomes, encompassing increased in-hospital and long-term mortality rates. Notwithstanding its simplicity, it remains integral to prominent risk assessments like the GRACE and TIMI scores, emphasizing its prognostic significance.[6]

In conjunction with clinical grading systems, biochemical markers are being extensively investigated to improve prognostic precision in AMI. Serum uric acid, the final result of purine metabolism facilitated by the enzyme xanthine oxidase, has garnered interest in this context.[7]

Historically associated with gout and renal impairment, uric acid is now acknowledged as an indicator of oxidative stress, inflammation, and endothelial dysfunction, which are integral to the pathogenesis of atherosclerosis and cardiac damage. Increased blood uric acid concentrations are linked to hypertension, metabolic syndrome, chronic renal disease, and heightened cardiovascular risk.[8,9]

During AMI, tissue hypoxia and ischemia induce heightened ATP breakdown and enhanced purine metabolism, resulting in high uric acid concentrations.[10] Xanthine oxidase activity also produces reactive oxygen species, exacerbating oxidative stress and leading to myocardial injury. Numerous studies indicate that excessive serum uric acid levels are associated with adverse clinical outcomes, increased incidence of heart failure, arrhythmias, and heightened mortality in individuals with AMI.[11]

Despite these correlations, the association between blood uric acid levels and recognized clinical categories of heart failure, such as the Killip class, has not been thoroughly investigated across various populations. Establishing such a link could yield a straightforward, cost-effective, and readily accessible biomarker to enhance clinical evaluation and refine risk categorization.

Aims and Objectives: To evaluate the correlation between serum uric acid levels and Killip class in patients presenting with acute myocardial infarction.

MATERIALS AND METHODS

This prospective observational study was carried out in the Departments of General Medicine and Cardiology at Sree Mookambika Institute of Medical Sciences, Kulasekharam, over a period of eight months from January 2025 to August 2025. The study population consisted of 120 patients admitted with AMI. The diagnosis of AMI was determined based on clinical presentation, electrocardiographic alterations, and increased cardiac biomarkers, consistent with the global criteria of myocardial infarction.

Patients were consecutively enrolled upon meeting the inclusion criteria, which included persons aged 18 years and older, of either gender, presenting within 24 hours of the beginning of symptoms indicative of AMI. Individuals with chronic renal illness, gout, hematological malignancies, chronic liver disease, autoimmune disorders, or cancers, along with those receiving drugs that modify uric acid metabolism, including allopurinol, febuxostat, or thiazide diuretics, were excluded. Patients who could not complete baseline laboratory investigations or were critically ill and unfit for assessment were eliminated from the study.

Informed consent was obtained from all participants before recruitment, and the confidentiality of patient data was maintained throughout the study. Upon admission, demographic information, clinical history, and data concerning risk factors including diabetes mellitus, hypertension, tobacco use, alcohol intake, and familial history of coronary artery disease were documented.

All patients received a comprehensive clinical evaluation, and baseline vital signs were recorded. The intensity of heart failure was categorized at admission utilizing the Killip classification. Patients were categorized into Killip Class I in the absence of heart failure, Class II with mild heart failure indicators such as S3 gallop, basal rales affecting less than 50% of lung fields, or elevated jugular venous pressure, Class III with evident pulmonary edema, and Class IV if the patient exhibited cardiogenic shock characterized by a systolic blood pressure below 90 mmHg accompanied by signs of hypoperfusion.

Venous blood samples were obtained within 24 hours of admission before the commencement of uric acid-modifying therapy. Serum uric acid concentrations were measured in the central laboratory with the enzymatic colorimetric uricase technique. Standard examinations, including complete blood count, fasting blood glucose, renal and hepatic function tests, lipid profile, electrocardiography, echocardiography, and cardiac biomarkers such as troponins and CK-MB, were conducted in accordance with hospital procedure.

All data were inputted into Microsoft Excel and analyzed with SPSS software version 20.0. Continuous variables were represented as mean ±

standard deviation, whereas categorical variables were denoted as percentages. The relationship between serum uric acid levels and Killip class was evaluated using Pearson's correlation coefficient. The mean serum uric acid levels among several Killip classes were compared using one-way analysis of variance (ANOVA). A p-value that was lower than 0.05 was regarded as statistically significant.

RESULTS

The age distribution of the study population revealed that the mean age was 58.4 ± 10.7 years, with the youngest patient being 35 years old and the oldest 82 years old. The maximum number of cases (46.7%) were in the 51–60 years old age group. [Table 1]

In terms of gender distribution, males predominated with 82 patients (68.3%), while females constituted 38 patients (31.7%). The male-to-female ratio was approximately 2.1:1. The majority of patients presented with no or mild heart failure (Classes I and II), while a smaller proportion presented with severe heart failure or cardiogenic shock (Classes III and IV). [Table 2]

Serum uric acid levels increased significantly with worsening Killip class ($p < 0.001$). The progressive elevation in serum uric acid levels with higher Killip classes indicates a strong relationship between worsening heart failure status and increased uric acid concentrations. [Table 3]

Table 1: Age-wise Distribution of Patients

Age Group (years)	Number of Patients (n=120)	Percentage (%)
31–40	12	10.0
41–50	26	21.7
51–60	56	46.7
61–70	18	15.0
>70	8	6.6

Table 2: Distribution of Patients According to Killip Class

Killip Class	Number of Patients	Percentage (%)
I	55	45.8
II	36	30.0
III	18	15.0
IV	11	9.2

Table 3: Mean Serum Uric Acid Levels in Relation to Killip Class

Killip Class	Number of Patients	Percentage (%)	p value
I	5.2 ± 1.1	3.8 – 7.0	<0.001
II	6.4 ± 1.3	4.5 – 8.5	
III	7.3 ± 1.5	5.6 – 9.4	
IV	8.1 ± 1.6	6.0 – 10.2	

The association between uric acid levels and Killip class was statistically significant ($p < 0.001$), confirming that higher uric acid levels correlate with more severe heart failure in AMI. [Table 4]

Table 4: Distribution of Patients by Serum Uric Acid Categories and Killip Class

Serum Uric Acid Level	Killip I (n=55)	Killip II (n=36)	Killip III (n=18)	Killip IV (n=11)	Total (n=120)	p-value
<6 mg/dL	40	12	2	0	54 (45.0%)	<0.001
6–8 mg/dL	12	18	10	4	44 (36.7%)	
>8 mg/dL	3	6	6	7	22 (18.3%)	

DISCUSSION

Myocardial infarction, also known as AMI, continues to be one of the leading causes of death. Timely risk categorization and prognostic evaluation are essential for enhancing patient outcomes. The mean age of the study population was 58.4 ± 10.7 years, with the majority of patients (46.7%) falling within the 51–60 age bracket.

This correlates with other Indian and international studies indicating that middle-aged and elderly individuals face an elevated risk of developing AMI due to cumulative exposure to cardiovascular risk factors, including hypertension, diabetes mellitus, smoking, dyslipidemia, and a sedentary lifestyle. The diminished percentage of patients over 70 years may indicate early mortality before hospital admission, reduced hospital attendance among the very elderly, or survival bias.

The gender distribution indicated a majority of males (68.3%) compared to females (31.7%), resulting in a male-to-female ratio of 2.1:1. This finding corresponds with established epidemiological data indicating that males are at an elevated risk for coronary artery disease and AMI at a younger age, likely due to increased exposure to behavioral and metabolic risk factors, as well as the cardioprotective effects of estrogen in premenopausal women.

Shahithya KA et al,[12] reported in their study that the majority of patients, specifically 75.86% of instances of AMI, were within the age category of 51-70 years,

with a mean age of 59.83±8.29 years, ranging from 29 to 80 years. Among the study population, there were 72 male patients and 44 female patients. This was analogous to the current investigation.

In the present study at admission, the distribution of patients by Killip class revealed that 45.8% were classified as Class I, 30% as Class II, 15% as Class III, and 9.2% as Class IV. This implies that although almost half of the patients exhibited no clinical indications of heart failure, a substantial minority presented with mild to severe heart failure. The percentage of patients in Killip Class III and IV is clinically significant, as these individuals exhibit elevated death rates and necessitate intensive care management.

Serum uric acid levels were seen to increase progressively with ascending Killip class. Statistical analysis revealed that the variations in uric acid levels among Killip classes were statistically significant ($p < 0.001$). The findings demonstrate a robust correlation between elevated blood uric acid levels and heightened severity of heart failure in patients with AMI.

The study conducted by Kumar N et al,[13] revealed that the mean serum uric acid levels were 4.4 mg/dl in Killip class I, 7.01 mg/dl in class II, 8.29 mg/dl in class III, and 9.87 mg/dl in class IV on day 0; 4.46 mg/dl in class I, 7.09 mg/dl in class II, 8.53 mg/dl in class III, and 9.43 mg/dl in class IV on day 3; and 4.72 mg/dl in class I, 6.62 mg/dl in class II on day 7. A statistically significant increase in uric acid levels was observed with ascending Killip class on days 0, 3, and 7.

A study conducted by Mehrpooya M et al,[14] found that the mean uric acid levels were 8.2±0.19 mg/dl in the Killip class I group, 9.02±0.34 mg/dl in the Killip class II group, 9.91±0.23 mg/dl in the Killip class III group, and 10.59±0.28 mg/dl in the Killip class IV patients ($P<0.001$).

The pathophysiological foundation for this correlation may be multifaceted. During AMI, ischemia accelerates the breakdown of adenine nucleotides, leading to heightened uric acid generation through xanthine oxidase activity. This process also produces reactive oxygen species, leading to oxidative stress, endothelial dysfunction, and further cardiac damage.[15] Hyperuricemia serves as both an indicator and a possible mediator of myocardial injury and heart failure. The noted incremental increase in blood uric acid levels with elevated Killip classes corroborates the concept that uric acid serves as an indicator of the severity of hemodynamic compromise in AMI.[16]

Similar to the present study, Shahithya KA et al,[12] noted that increased blood uric acid levels in AMI patients correlated with poorer in-hospital outcomes and raised Killip class. The study revealed that the majority of cases were classified as Killip class I (57), followed by class II (26), class III (18), and class IV (15). The mortality rate was 15.51% (18 instances), with 15 patients in Killip class IV and only 3 patients in Killip class III. The average SUA for released patients was 6.91±2.54 mg/dl, while for deceased patients it was 15.63±1.33 mg/dl.

Shivakumar BG et al,[17] also observed that hyperuricemia independently forecasted mortality and worse cardiovascular outcomes in individuals with acute coronary syndrome. Conversely, certain studies have indicated weaker or non-significant relationships, maybe attributable to variations in sample size, timing of uric acid assessment, patient demographics, or co-morbid conditions. Nonetheless, the preponderance of evidence, including the current study, corroborates the predictive relevance of serum uric acid in AMI.

The study indicated that the majority of patients in Killip Class I exhibited uric acid levels below 6 mg/dL, but those in Killip Classes III and IV often had values exceeding 7 mg/dL, with a significant number surpassing 8 mg/dL. This categorical analysis corroborates the trend of elevated uric acid levels correlating with increased clinical severity and indicates that uric acid may serve as a straightforward, cost-effective biomarker to augment Killip classification in risk stratification.

In the research by Shahithya KA et al,[12] uric acid levels were markedly elevated in patients classified as class IV (16.04) and class III (11.49) compared to those in class II (8.38) and class I (4.98), ($p=0.001$). Biswas K et al,[18] observed that serum uric acid levels were diminished in patients with a lower Killip class and elevated in those with a higher Killip class. A lower uric acid level correlated with an increased survival rate, whereas a higher uric acid level was associated with an elevated death rate.

CONCLUSION

The current study revealed that in patients with AMI, blood uric acid levels and Killip class significantly positively correlate. Elevated uric acid indicates greater severity of heart failure and may function as a valuable prognostic biomarker. Integrating blood uric acid measurement into the standard evaluation of AMI patients may assist doctors in early risk classification and treatment decision-making. Future extensive, multicentric investigations with prolonged follow-up are necessary to corroborate these findings and investigate the possible therapeutic implications of uric acid-lowering therapies in acute coronary syndromes.

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