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Correlation of Serum Uric Acid and Troponin I Level in Patients with Acute Myocardial Infarction

Md. A. A. Mohimen[1]*, Nazia N. Ria[1], Zannatul Ferdousi[2], Sumaiya S. Zaman[3], Md. N. Sadat[4]
[1]Assistant Professor (CC), Department of Physiology, Rajshahi Medical College, Rajshahi, Bangladesh
[2]Assistant Professor (CC), Department of Anatomy, Rajshahi Medical College, Rajshahi, Bangladesh
[3]Assistant Professor (CC), Department of Biochemistry, Rajshahi Medical College, Rajshahi, Bangladesh
[4]Assistant Professor, Department of Biochemistry, Diabetic Association Medical College, Faridpur, Bangladesh

Abstract: ***Background:*** Acute myocardial infarction (AMI) is a significant contributor to global mortality, with ischemic heart disease being the predominant form. Risk factors for cardiovascular disease have been linked to elevated serum uric acid levels. This study investigates the relationship between AMI patients serum uric acid and troponin I levels. Objective: The study sought to determine the correlation between serum uric acid and troponin I levels in patients with AMI and to evaluate the difference in uric acid levels between different types of AMI. ***Methods:*** A cross-sectional comparative study was conducted at Dhaka Medical College over one year, involving 100 AMI patients aged 30 to 70. Serum uric acid and troponin I levels were measured, and demographic data, including age, gender, BMI, smoking status, and hypertension, were recorded. Statistical analyses were performed using SPSS software. ***Results:*** The mean serum uric acid level was significantly higher in AMI patients (6.03 ± 1.68 mg/dl) compared to healthy individuals (4.65 ± 1.19 mg/dl), with a positive correlation between uric acid and troponin I levels ($r=0.621$, $p < 0.001$). Additionally, serum uric acid levels were significantly higher in ST-segment elevated MI (STEMI) patients (7.45 ± 0.81 mg/dl) compared to non-STEMI patients (4.61 ± 0.96 mg/dl). ***Conclusions:*** Hyperuricemia appears to be a potential risk factor for AMI development. Regular screening of serum uric acid levels may aid in prognostic assessment and cardiovascular risk management. The results highlight the need for prompt action to detect and treat AMI in its early stages.

Keywords: Acute myocardial infarction, Serum uric acid and Troponin I.

Original Researcher Article

Correspondence:
Dr. A. A. Mohimen
Assistant Professor (CC), Department of Biochemistry, Rajshahi Medical College, Rajshahi, Bangladesh
Email: drmohimen@gmail.com
https://orcid.org/0009-0004-7091-6206

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Article at a glance:

Study Purpose: *Relationship between serum uric acid and the results emphasize the need of early detection and intervention to reduce AMI consequences.*

Key findings: *Serum uric acid levels in AMI patients were elevated (6.03 ± 1.68 mg/dl) and positively correlated with troponin I ($r=0.621$, $p < 0.001$). Significant differences were seen between ST-segment elevated MI (STEMI) and non-STEMI groups.*

Newer findings: *The study reinforces hyperuricemia as a potential risk factor for AMI and emphasizes the significance of regular screening for improved prognostic assessment and cardiovascular risk management.*

Abbreviations: *AMI: Acute myocardial infarction, cTnI: Cardiac Troponin I, HUA: Hyperuricemia, IHD: Ischemic heart disease, and ECG: Electrocardiogram.*

INTRODUCTION

Cardiovascular diseases (CVDs) stand as a formidable global health challenge, contributing significantly to worldwide mortality rates. Annually, one-third of global fatalities are attributed to CVDs, a perilous and potentially fatal condition on the rise, with projections reaching 23.6 million deaths by 2010.[1] Alarmingly, 80% of these deaths occur in impoverished and middle-income nations, highlighting the urgent need for effective preventive and management strategies.[2] While the precise incidence of coronary artery disease (CAD) in Bangladesh remains elusive, regional studies indicate rates of 3.4% in rural areas and a

substantial 19.6% in urban centers.3 Among the critical manifestations of CVDs, Acute Myocardial Infarction (AMI) takes a prominent position, necessitating emergency hospital admissions. Despite commendable advancements in medical care, including a 30% reduction in mortality rates over the last two decades, the impact of AMI remains significant. Particularly concerning is the sharp decline in survival percentages for individuals beyond the age of 70, with in-hospital death rates escalating to 21% compared to 2.8% for patients under 60. [2,3]

AMI occurs when a coronary artery experiences a sudden reduction in blood flow due to thrombotic occlusion, disrupting the myocardial cellular healing mechanism and impeding cardiac activity to maintain homeostasis.4 Prolonged myocardial ischemia, reaching a critical threshold, leads to irreversible damage and cell death, underscoring the urgent need for effective diagnostic and management strategies. Uric Acid, a byproduct of purine metabolism, is a possible cardiovascular biomarker and therapeutic target. Elevated serum uric acid levels indicate hyperuricemia, a pathological condition with implications for metabolic and vascular health.[5] While uric acid exhibits antioxidant properties, imbalances are implicated in hypertension, renal disease, and atherosclerosis.[6,7]

A cardiovascular biomarker and therapeutic target have emerged. Conventional vascular risk factors, including advanced age, male sex, obesity, dyslipidemia, smoking, and insulin resistance, are substantially linked to uric acid levels.[8] Furthermore, hyperuricemia has been implicated in atherosclerosis through mechanisms involving lipid peroxidation and vascular endothelial dysfunction.[9] Activation of the renin-angiotensin-aldosterone system by hyperuricemia further exacerbates development of hypertension, complicating cardiovascular pathophysiology.[10] Cardiac Troponin I (cTnI) has emerged as a key biomarker for diagnosing and predicting AMI. As a contractile protein found exclusively in cardiac myocytes, cTnI levels rise rapidly following myocardial injury, reaching peak concentrations within 14 to 18 hours and remaining elevated for 5 to 7 days.[11,12] Early detection of cTnI within 4-6 hours of myocardial damage provides clinicians with a sensitive tool for timely intervention and risk stratification.[13]

Despite insufficient evidence establishing causation, routine evaluation and treatment of hyperuricemia are recommended to mitigate the incidence and severity of AMI and its associated morbidity and mortality rates.[14] However, further research is warranted to elucidate the intricate interplay between hyperuricemia, cTnI, and cardiovascular outcomes. Hypertension complicates cardiovascular pathophysiology, necessitating comprehensive strategies for prevention, diagnosis, and management. Hyperuricemia, characterized by elevated serum uric acid levels, Globally, AMI causes major morbidity and mortality. The association between hyperuricemia and traditional vascular risk factors underscores the complex interplay between metabolic and vascular conditions in cardiovascular pathophysiology.

Furthermore, the role of cTnI as a sensitive biomarker for myocardial injury highlights the importance of timely diagnosis and risk stratification in AMI management. While routine evaluation and treatment of hyperuricemia are recommended, further research is needed to elucidate the causal mechanisms underlying its association with cardiovascular outcomes. By addressing these gaps in knowledge, clinicians and researchers can advance our understanding of AMI pathophysiology and improve patient outcomes through targeted interventions and personalized treatment approaches.

METHODS

Study Design

This study employed a cross-sectional comparative design conducted at the Department of Biochemistry, Dhaka Medical College, from July 2018 to June 2019. The study investigated the relationship between serum uric acid and troponin I levels in patients with acute myocardial infarction. Cases involved individuals admitted to the National Institute of Cardiovascular Diseases (NICVD), Dhaka, diagnosed through 12-lead ECG and troponin I assessment. Purposive sampling was used to choose study participants after ethical permission.

Inclusion Criteria

- Patients diagnosed with acute myocardial infarction.
- Admission to the National Institute of Cardiovascular Diseases (NICVD), Dhaka.
- Confirmation of diagnosis through 12-lead ECG and troponin I assessment.
- Willingness to participate in the study and provide informed written consent.
- Availability of fasting blood samples for analysis.

Exclusion Criteria

- Chronic renal disease patients.
- Patients diagnosed with diabetes mellitus.
- Inability to provide informed written consent.
- Individuals with incomplete medical records or missing data.
- Patients on medications known to influence uric acid or troponin I levels.

Data Collection

Data collection for this cross-sectional comparative study occurred at the Department of Biochemistry, Dhaka Medical College, Dhaka, from July 2018 to June 2019. Acute myocardial infarction (AMI) patients admitted to the National Institute of Cardiovascular Diseases (NICVD), Dhaka, were identified using 12-lead ECG and troponin I confirmation. We used a purposive sampling strategy to pick 30 healthy people and 70 AMI patients. After obtaining informed written consent, a pre-designed data collection sheet captured patient particulars, medical history, and relevant investigations. We took blood samples from those who had fasted, analyzed them, and stored for subsequent analysis of uric acid and troponin I levels at the NICVD Clinical Biochemistry Department.

Blood Sample Collection

After obtaining aseptic precautions, a 5 ml venous Each participant had a disposable plastic syringe used to draw blood from them while they were fasting. The sample was immediately transferred to a clean, dry test tube and allowed to clot at room temperature. For AMI patients, blood samples were collected between twelve to twenty-four hours after the onset of chest pain. The samples were centrifuged at 3000 rpm for 10 minutes, and the clear serum was separated into sterile Eppendorf tubes. Fasting blood glucose and serum creatinine levels were initially assessed to exclude patients with diabetes mellitus and chronic kidney disease. Following the exclusion of these conditions, the separated serum samples were stored at -20ºC for further analysis.

Data Analysis

Data processing and analysis were carried out using the use of SPSS 24.0, a statistical package for social science research. The Unpaired t-test was used to compare the two groups' continuous variables, which were represented as mean ± SD. Using the Chi-Square Test, we compared categorical variables. The correlation between serum uric acid and troponin I levels was assessed using Pearson's correlation test. For all tests, a probability level of $p < 0.05$ was used to establish statistical significance.

Ethical considerations

Ethical approval was paramount throughout the study. Dhaka Medical College's Ethical Review Committee gave its approval before the study began. Informed written consent was secured from all participants, outlining the study's purpose and procedures. Confidentiality of participants' personal information was strictly maintained, adhering to ethical standards. The research prioritized respect for participants' autonomy, beneficence, and non-maleficence. All procedures were conducted following ethical guidelines to ensure the well-being and rights of the study participants.

RESULTS

The mean age of patients in Group A was 52.70±10.11 years; in Group B, it was 54.90±9.54 years, with no significant difference (p=0.313). Male patients constituted 81.4% in Group A and 76.7% in Group B, showing no statistically significant difference (p=0.595). However, BMI was significantly higher in Group A (23.94 ± 3.36 kg/m2) compared to Group B (22.18±1.91 kg/m2), and this difference was statistically significant (p=0.009).

Table 1: Demographic profile of the study subjects according to age, gender, and BMI in group A and group B (n=100)

Age (years)	Group A (n=70) n (%)	Group B (n=30) n (%)	p-value
30 – 39	8 (11.4)	2 (6.7)	[b]0.718
40 – 49	17 (24.3)	4 (13.3)	[b]0.228
50 – 59	15 (21.4)	9 (30.0)	[b]0.356
≥60	30 (42.9)	15 (50.0)	[b]0.511
Mean± SD	52.70±10.11	54.90±9.54	[a]0.313
Range	32-70	34-70	
Gender			
Male	57 (81.4)	23 (76.7)	[b]0.595
Female	13 (18.6)	7 (23.3)	
BMI (kg/m^2)			
Normal (18.5 - 24.9)	50 (71.4)	28 (93.3)	[b]0.015
Overweight (25.0 - 29.9)	15 (21.4)	2 (6.7)	[b]0.071
Obese (≥30.0)	5 (7.1)	0 (0.0)	[b]0.318
Mean ±SD	23.94 ± 3.36	22.18±1.91	[a]0.009
Range	18.0 – 34.7	19.4-26.3	

[b]Chi-square test and [a]Unpaired student t-test were done to measure the significance level. The value within the parenthesis indicates percentage (%).

Smoking (57.1% in Group A and 33.3% in Group B), hypertension (41.4% in Group A, 16.7% in Group B), SBP (127.5 ± 7.5 mmHg in Group A and 120.0 ± 7.2 mmHg in Group B) were significantly higher in group A than group B (p=0.029, p=0.017 and p < 0.001, respectively). However, there was no significant difference in terms of DBP between the two groups (p=0.114).

Table 2: Baseline characteristics of the study subjects in group A and group B (n=100)

Variable	Group A (n=70) n (%)	Group B (n=30) n (%)	p-value
Smoking	40 (57.1)	10 (33.3)	[b]0.029
Hypertension	29 (41.4)	5 (16.7)	[b]0.017
SBP	127.5 ± 7.5	120.0 ± 7.2	[a]<0.001
DBP	81.6 ± 3.1	80.0 ± 7.2	[a]0.114

[b]Chi-square test and [a]Unpaired student t-test were done to measure the significance level. The value within the parenthesis indicates percentage (%). SBP = Systolic Blood Pressure, DBP = Diastolic Blood Pressure. Mean serum uric acid level was 6.03 ± 1.68 mg/dl in Group A and 4.65 ± 1.19 mg/dl in Group B and it was statistically highly significant (p < 0.001),and troponin I was also higher in Group A (12.78 ± 9.89 ng/ml).

Table 3: Serum Uric acid and Troponin I level of the study subjects in groups A and B (n=100)

Parameters	Group A (n=70)	Group B(n=30)	p-value
	Mean ± SD		
Uric Acid level (mg/dl)	6.03 ± 1.68	4.65 ± 1.19	<0.001
Troponin I level (ng/ml)	12.78 ± 9.89	-	

An unpaired student t-test was done to measure the level of significance. There was a statistically highly significant correlation between uric acid and troponin I in Group A (r=0.621, p < 0.001) (Figure 1).

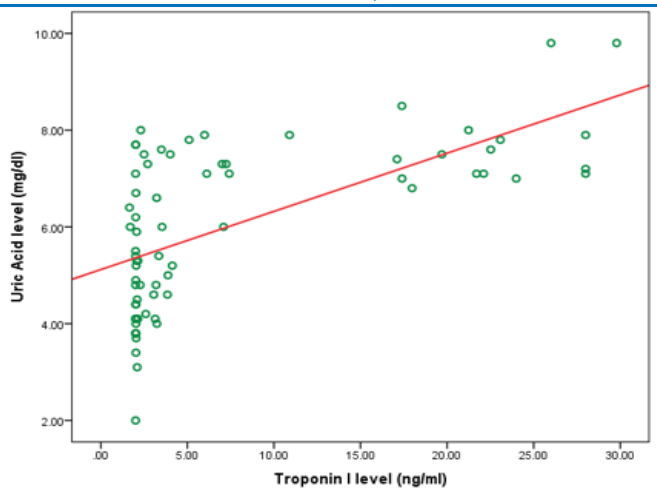

Figure 1: Scatter diagram showing the correlation of Uric Acid with Troponin I in Group A

The uric acid level was higher among STEMI patients (7.45 ± 0.81 mg/dl) than non-STEMI (4.61 ± 0.96 mg/dl), and it was statistically significant ($p < 0.001$).

Table 4: Serum Uric acid level of STEMI and Non-STEMI in group A(n=70)

	STEMI	Non-STEMI	p-value
Uric acid (mg/dl)	7.45 ± 0.81	4.61 ± 0.96	<0.001

Unpaired student t-test was done to measure the level of significance; STEMI= ST segment Elevated Myocardial Infarction; Non-STEMI Non-ST segment Elevated Myocardial Infarction.

DISCUSSION

In evaluating the correlation between serum uric acid in those suffering from a sudden heart attack, particularly troponin I, this cross-sectional study was conducted to monitor the levels of these two markers. The participants in the AMI group had an average age of 52.70±10.11 years, while those in the comparison group had an average age of 54.40±9.54 years. The results of this study were corroborated,[15] which found that 42.9% of patients with acute myocardial infarction were 60 years of age or older. Regarding the gender breakdown in this study, there were 80% male and 20% female participants. Males comprised 81.4% of the AMI group and females 18.6%, while in the normal healthy group, the ratio was 76.7% to 23.3%. Neither group differed significantly from the other about age or sex. This finding agreed with what Ahmadi *et al*. found.[16] The average body mass index (BMI) for the AMI group was 23.94 kg/m2, while the comparison groups was 22.18 kg/m2. In line with the study conducted by Kokane *et al*., the AMI group had a noticeably higher body mass index ($p=0.009$).[17]

In the present study, regarding smoking history, 57.1% of smokers were in AMI, and 33.3% were in the healthy group. There was a significantly higher number of smokers in the AMI group. This result was supported by Akanda *et al*. Concerning hypertension, 41.4% were hypertensive in the case, and 16.7% were in the comparison group.[18] There was also significantly higher hypertension in the AMI group, consistent with the study by Kabiruzzaman *et al*., Regarding blood pressure, mean systolic blood pressure (SBP) and diastolic blood pressure were 127.5 and 81.6 mm of Hg in the case and 120 and 80 mm of Hg in the comparison group.[19] SBP was significantly higher in AMI than in the healthy group. But in the case of DBP, there was no significant difference between the groups.

The average uric acid level in the AMI group was 6.03 mg/dl, while it was 4.65 mg/dl in the control group. Compared to the healthy group, those with AMI had substantially higher serum uric acid levels. Dambal *et al*. found the same thing.[20] Several cardiovascular events can be attributed to atherosclerosis, which uric acid facilitates.[21] According to the research, coronary artery disease patients had substantially greater SUA levels than

healthy controls.[22,23] Consistent with the work conducted by Hasic *et al*., the mean troponin I level in this present investigation was 12.78 ng/dl.[24] A statistically significant positive connection ($r = 0.621$, $p < 0.001$) between uric acid and troponin I in AMI patients was observed in this study using Pearson's correlation test (r). One investigation by Hasic *et al*. showed that serum uric acid and troponin I were positively correlated in patients with acute myocardial infarction and unstable angina pectoris. Subjects with STEMI (7.45 mg/dl) had a substantially greater mean blood uric acid level compared to those without STEMI (Non-STEMI) in this study (4.61). [25]

The study observed a positive correlation between serum uric acid and troponin I, indicating a potential link between uric acid levels and cardiac damage. Subgroup analysis further confirmed this correlation, showing significantly higher uric acid levels in ST-segment elevation myocardial infarction (STEMI) patients compared to non-STEMI patients. These results align with previous research supporting the role of uric acid in cardiovascular diseases. There was a positive correlation between myself and patients suffering from unstable angina pectoris and sudden myocardial infarction. Being a single hospital-based study with a small sample size and using purposive sampling may limit the generalizability of the findings. Future research with more diverse populations is warranted to validate and extend these observations. The study contributes to the growing evidence linking uric acid to cardiovascular health. The positive correlation with troponin I highlights the potential utility of serum uric acid as a biomarker for assessing cardiovascular risk in AMI patients. Regular screening of uric acid levels could provide clinicians with additional information for risk assessment and personalized management strategies.

CONCLUSION

This study underscores the importance of exploring novel biomarkers for cardiovascular risk assessment. Further research and prospective studies are needed to establish a causal relationship between uric acid and cardiac events, paving the way for potential interventions and preventive measures in the management of acute myocardial infarction and related conditions.

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Authors contributions

MAAM: Study and questionnaire design, data collection, data analysis and interpretation, writing the manuscript, and drafting the article. NNR and ZF: Analysis plan, data analysis, and interpretation, manuscript writing, and article drafting. SSZ: Manuscript writing and editing. MNS: Supervision. All authors revised the manuscript and approved it for publication.

Declarations

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