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Original Research Article

Clinical Correlation of Hs Troponin I and Serum Uric Acid Levels in Acute Myocardial Infarction Patients: A Cross-Sectional Study

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Abstract:

Background: Acute myocardial infarction (AMI) remains a leading cause of morbidity and mortality worldwide. High-sensitivity Troponin I (Hs-TnI) is a key biomarker for early diagnosis of myocardial injury. Recent studies also suggest a possible link between elevated serum uric acid levels and cardiovascular events. This study aims to evaluate the levels of Hs-TnI and uric acid in patients presenting with myocardial infarction and assess their potential clinical correlation.

Aim and Objectives: To assess serum Hs-Troponin I and uric acid levels in patients diagnosed with AMI and determine their relationship with severity and clinical profile.

Materials and Methods: This hospital-based cross-sectional observational study was conducted over a 12-month period from April 2024 to March 2025 at the Department of Biochemistry, NMCH, Jamuhar, Sasaram. A total of 150 AMI patients were included. Serum Hs-TnI and uric acid levels were measured using chemiluminescence immunoassay and enzymatic methods respectively. Data were statistically analyzed for correlation with clinical parameters and risk factors.

Results: A significant elevation in serum Hs-TnI and uric acid levels was observed in all AMI patients. Hs-TnI levels were markedly higher in STEMI patients compared to NSTEMI. Elevated uric acid levels were more prevalent in patients with a history of hypertension and metabolic syndrome. A positive correlation was found between uric acid levels and severity of myocardial infarction, as evidenced by ECG and echocardiography findings.

Conclusion: Hs-Troponin I remains a sensitive and specific biomarker for AMI diagnosis. Elevated uric acid levels may serve as an adjunct marker indicating increased cardiovascular risk and worse outcomes in myocardial infarction. Dual assessment of Hs-TnI and uric acid may enhance early risk stratification and guide therapeutic decisions.

Keywords: High-sensitivity Troponin I, Uric Acid, Myocardial Infarction, Biomarkers, Cardiovascular Risk.

Introduction

Acute myocardial infarction (AMI), commonly known as a heart attack, continues to be a major global health burden, contributing significantly to morbidity and mortality, particularly in low- and middle-income countries like India [1]. It is characterized by the interruption of blood supply to a part of the heart, typically due to a thrombotic occlusion of a coronary artery, resulting in myocardial ischemia and necrosis. Rapid and accurate diagnosis is essential for initiating timely interventions to reduce infarct size, preserve left ventricular function, and improve patient outcomes [2].

Cardiac biomarkers play a pivotal role in the early and accurate diagnosis of AMI. Among them, cardiac troponins have emerged as the gold standard due to their high specificity and sensitivity for myocardial injury [3]. In particular, high-sensitivity troponin I (Hs-TnI) assays allow for the detection of even minor myocardial injury at very early stages of infarction. These assays enable the rapid rule-in or rule-out of AMI in emergency settings and are integral to current clinical protocols and algorithms, such as the ESC 0/1-hour rule-in rule-out strategy. Hs-TnI levels typically rise within 3–4 hours of symptom onset, peaking around 12–24 hours, and remain elevated for several days, thus offering a wide diagnostic window [4].

In addition to established cardiac biomarkers, there is growing interest in identifying novel biochemical indicators that could offer prognostic value or reflect underlying metabolic disturbances associated with cardiovascular disease. Uric acid, the end product of

purine metabolism, has recently emerged as a potential contributor to cardiovascular risk [5]. Hyperuricemia has been linked with endothelial dysfunction, oxidative stress, inflammation, and insulin resistance all of which are recognized mechanisms in atherogenesis and plaque instability. Elevated uric acid levels have been observed in various cardiovascular conditions, including hypertension, metabolic syndrome, heart failure, and ischemic heart disease [6]. However, its role as a marker or mediator in acute coronary events remains an area of active investigation.

Several studies have reported that higher serum uric acid levels are associated with increased mortality and adverse outcomes in AMI patients. It is hypothesized that hyperuricemia may reflect an underlying pro-inflammatory and pro-oxidant state that predisposes individuals to atherothrombosis and myocardial ischemia [7]. Moreover, uric acid levels tend to rise in the context of ischemia-reperfusion injury, reduced renal perfusion, and increased tissue catabolism—all of which occur during acute myocardial events. Hence, assessing uric acid levels in conjunction with Hs-TnI may offer additional insights into the severity, prognosis, and metabolic background of AMI [8].

In this context, the present study aims to assess serum Hs-TnI and uric acid levels in patients presenting with AMI at a tertiary care center. The study further seeks to explore the clinical correlation of these biomarkers with the type of myocardial infarction, associated comorbidities, and potential implications for risk stratification and management.

Aim and Objectives

Aim: To assess the serum levels of high-sensitivity Troponin I (Hs-TnI) and uric acid in patients diagnosed with acute myocardial infarction (AMI) and evaluate their clinical correlation with the type and severity of myocardial infarction.

Primary Objectives:

1. To estimate serum Hs-Troponin I levels in patients presenting with AMI.
2. To estimate serum uric acid levels in the same patient cohort.
3. To compare Hs-TnI and uric acid levels between STEMI and NSTEMI patients.
4. To correlate serum uric acid levels with common cardiovascular risk factors such as hypertension, diabetes mellitus, and metabolic syndrome.

Secondary Objectives:

1. To evaluate the association between Hs-TnI and echocardiographic findings such as left ventricular ejection fraction (LVEF).
2. To determine whether elevated serum uric acid levels are predictive of worse clinical outcomes in AMI patients.
3. To explore the combined utility of Hs-TnI and uric acid as biochemical indicators in the diagnosis and risk stratification of AMI.

Materials and Methods

Study Design: A hospital-based, observational cross-sectional study.

Study Duration: April 2024 to March 2025 (12 months).

Study Location: Department of Biochemistry, Narayan Medical College and Hospital (NMCH), Jamuhar, Sasaram, Bihar, India.

Study Population: Patients aged 30 years and above who presented with acute myocardial infarction (STEMI or NSTEMI) and were admitted to the emergency or cardiology unit of NMCH, Jamuhar.

Sample Size: A total of 150 patients diagnosed with AMI were included in the study.

Inclusion Criteria:

- Patients aged ≥30 years diagnosed with AMI based on ECG, cardiac enzyme profile, and clinical history
- Willing to provide informed consent
- Patients within 24 hours of symptom onset

Exclusion Criteria:

- Known history of chronic kidney disease (CKD), gout, or hematological disorders
- Patients on uric acid-lowering therapy
- Chronic alcoholics or patients with liver failure
- Recent trauma, surgery, or malignancy

Methods

Detailed clinical and demographic data including age, sex, history of diabetes, hypertension, smoking, and alcohol use were recorded. Patients were classified into STEMI or NSTEMI based on ECG and cardiac enzyme findings.

Blood samples were collected within 24 hours of admission.

- **Serum Hs-Troponin I** was measured using chemiluminescence immunoassay (CLIA) on a high-sensitivity platform.
- **Serum uric acid** was estimated using the uricase enzymatic method on an automated biochemistry analyzer.

Echocardiography was performed in all patients to evaluate left ventricular ejection fraction (LVEF) and regional wall motion abnormalities (RWMA).

Statistical Analysis: All data were compiled and analyzed using SPSS version 26. Descriptive statistics were expressed as mean ± standard deviation (SD) or percentage. Independent t-test and chi-square test were used to compare means and proportions respectively. Pearson's correlation coefficient was used to assess relationships between variables. A p-value <0.05 was considered statistically significant.

Results

This observational study was conducted on 150 patients diagnosed with acute myocardial infarction (AMI) at the Department of Biochemistry, NMCH, Jamuhar, over a period of 12 months. The study aimed to evaluate the levels of high-sensitivity Troponin I (Hs-TnI) and serum uric acid and assess their correlation with the type and severity of myocardial infarction. Patients were categorized into two groups based on ECG findings: STEMI (n=92) and NSTEMI (n=58). Baseline demographic data, biochemical markers, comorbidities, and echocardiographic findings were recorded and statistically analyzed.

Table 1: Age-wise Distribution of Study Participants

Age Group (years)	STEMI (n=92)	NSTEMI (n=58)	Total (n=150)
30–40	8 (8.7%)	5 (8.6%)	13 (8.7%)
41–50	19 (20.6%)	12 (20.7%)	31 (20.7%)
51–60	28 (30.4%)	18 (31.0%)	46 (30.7%)
61–70	25 (27.2%)	14 (24.1%)	39 (26.0%)
>70	12 (13.0%)	9 (15.5%)	21 (14.0%)
Mean ± SD	**56.1 ± 9.3**	**57.3 ± 10.1**	**56.6 ± 9.6**

Table 2: Gender Distribution of Study Subjects

Gender	STEMI (n=92)	NSTEMI (n=58)	Total (n=150)
Male	69 (75.0%)	43 (74.1%)	112 (74.7%)
Female	23 (25.0%)	15 (25.9%)	38 (25.3%)

Table 3: Prevalence of Common Risk Factors

Risk Factor	STEMI (n=92)	NSTEMI (n=58)	Total (n=150)
Hypertension	64 (69.6%)	37 (63.8%)	101 (67.3%)
Diabetes Mellitus	56 (60.9%)	33 (56.9%)	89 (59.3%)
Smoking	48 (52.2%)	25 (43.1%)	73 (48.7%)
Alcohol Consumption	38 (41.3%)	20 (34.5%)	58 (38.7%)
Metabolic Syndrome	30 (32.6%)	18 (31.0%)	48 (32.0%)

Table 4: Mean Hs-Troponin I Levels in STEMI and NSTEMI Patients

Group	Mean Hs-TnI (ng/L)	SD	P-value
STEMI	4200.5	1120.3	<0.001
NSTEMI	1680.4	890.6	

Table 5: Serum Uric Acid Levels in Study Groups

Group	Mean Uric Acid (mg/dL)	SD	P-value
STEMI	7.48	1.33	0.048
NSTEMI	6.91	1.41	

Table 6: Hs-Troponin I Levels and Ejection Fraction

LVEF (%)	Mean Hs-TnI (ng/L)
>50	1620.6
41–50	2980.2
≤40	4625.8

Table 7: Uric Acid Levels and Number of Cardiovascular Risk Factors

No. of Risk Factors	Mean Uric Acid (mg/dL)
0–1	6.22
2–3	7.01
≥4	7.68

Table 8: Correlation Coefficient Matrix

Variables	Correlation (r)	Significance (p)
Hs-TnI vs. LVEF	-0.72	<0.001
Uric Acid vs. Risk	+0.59	0.002
Hs-TnI vs. Uric Acid	+0.35	0.024

Table 1 shows that the most affected age group was 51–60 years, followed by 61–70 years. Table 2 highlights a male predominance among AMI patients across both STEMI and NSTEMI categories. Table 3 indicates hypertension and diabetes as the most common risk factors. Table 4 reveals significantly elevated Hs-TnI levels in STEMI patients compared to NSTEMI, confirming the marker's diagnostic strength. Table 5 shows elevated serum uric acid levels in both groups, slightly higher in STEMI cases. Table 6 demonstrates an inverse relationship between Hs-TnI levels and ejection fraction, suggesting increased myocardial damage in patients with lower LVEF. Table 7 reflects a positive trend between uric acid levels and the number of cardiovascular risk factors. Finally, Table 8 confirms a strong inverse correlation between Hs-TnI and LVEF, and a moderate positive correlation between uric acid and both cardiovascular risk burden and Hs-TnI levels.

Discussion

The present study was undertaken to evaluate the serum levels of high-sensitivity Troponin I (Hs-TnI) and uric acid in patients presenting with acute myocardial infarction (AMI) and to determine the association of these biochemical parameters with the type and severity of myocardial damage. A total of 150 patients were studied, including both STEMI and NSTEMI cases, and multiple clinical, biochemical, and echocardiographic parameters were analyzed [9,10].

The age-wise distribution showed that the majority of patients belonged to the 51–60 and 61–70 year age groups, indicating a higher incidence of AMI in the middle-aged to elderly population. Male predominance was evident, consistent with established literature that suggests males are at higher risk for ischemic cardiac events due to a combination of hormonal, behavioral, and lifestyle-related factors [11].

In terms of cardiovascular risk factors, hypertension and diabetes mellitus emerged as the most prevalent, followed by smoking and alcohol consumption. These comorbidities are known contributors to endothelial dysfunction, atherogenesis, and plaque instability, thereby increasing the likelihood of acute coronary syndromes. The presence of metabolic syndrome further amplified the cardiovascular risk in a significant portion of patients [12].

The mean serum Hs-Troponin I levels were found to be significantly higher in STEMI patients than NSTEMI, reaffirming its utility as a sensitive and specific biomarker for myocardial injury. Elevated levels corresponded with more severe myocardial damage and were inversely related to left ventricular ejection fraction (LVEF), highlighting the prognostic significance of this marker. This inverse correlation underscores that patients with higher myocardial injury had greater impairment in ventricular function, as assessed via echocardiography [13,14].

Serum uric acid levels were also elevated in a substantial number of patients and were marginally higher in STEMI cases. Uric acid, though traditionally not a primary cardiac biomarker, is increasingly being recognized for its association with adverse cardiovascular outcomes [15]. The study revealed a significant positive correlation between uric acid levels and the number of risk factors, suggesting that elevated uric acid may reflect an underlying pro-inflammatory and pro-oxidant state in individuals with metabolic burden. Moreover, a moderate positive correlation was noted between uric acid and Hs-TnI levels, indicating that higher uric acid concentrations may be linked with greater myocardial injury [16].

The analysis also showed that uric acid levels increased progressively with the accumulation of cardiovascular risk factors such as hypertension, diabetes, and smoking, supporting its role as a potential secondary marker of risk stratification. Although causality cannot be established due to the cross-sectional design, the observed associations align with previous research advocating the inclusion of uric acid assessment in high-risk cardiovascular profiles [17,18].

Correlation matrices demonstrated a strong negative association between Hs-TnI and LVEF and a moderate positive correlation between uric acid and both cardiovascular risk burden and troponin levels. These findings reinforce the complementary nature of these biomarkers: Hs-TnI being a primary indicator of acute myocardial injury, while uric acid may serve as a marker of systemic cardiovascular stress and risk [19,20].

Overall, the study substantiates the clinical relevance of combining Hs-TnI with uric acid estimation in AMI patients. While Hs-TnI continues to serve as a gold standard in the diagnosis and severity assessment of myocardial infarction, the addition of uric acid analysis may enhance early risk profiling, guide clinical decision-making, and potentially predict outcomes more effectively.

Conclusion

This observational study highlights the diagnostic and prognostic significance of Hs-Troponin I and serum uric acid in patients with acute myocardial infarction. Hs-TnI levels were significantly higher in STEMI patients and showed an inverse correlation with left ventricular ejection fraction, reflecting greater myocardial damage and functional compromise. Serum uric acid, although traditionally not classified as a cardiac biomarker, demonstrated elevated levels in AMI patients, with a significant association with metabolic risk factors and a moderate positive correlation with Hs-TnI. These findings suggest that serum uric acid may act as an adjunct marker for cardiovascular stress and risk stratification. Early assessment of both Hs-TnI and uric acid can aid in evaluating myocardial injury severity, optimizing therapeutic interventions, and improving clinical outcomes. Incorporating uric acid estimation alongside conventional cardiac markers may enhance the overall assessment strategy for patients presenting with acute coronary syndromes, especially in resource-limited settings. Further large-scale prospective studies are recommended to validate the clinical utility of serum uric acid as a prognostic indicator in myocardial infarction.

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