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Original Research Article

ASSESSMENT OF HIGH-SENSITIVITY TROPONIN I AND URIC ACID LEVELS IN PATIENTS WITH MYOCARDIAL INFARCTION

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ABSTRACT

Background: Aim: To investigate the levels of high-sensitivity Troponin I (Hs Troponin I) and uric acid in individuals diagnosed with myocardial infarction (MI) and examine their association with patient demographics, clinical features, and outcomes.

Materials and Methods: This prospective, observational study was conducted over 12 months at a tertiary care hospital and included 80 patients with acute myocardial infarction (AMI). Eligible patients were between 40 to 80 years of age and presented with MI symptoms, ECG changes, and elevated Hs Troponin I levels. Hs Troponin I levels were measured using high-sensitivity immunoassay, and uric acid levels were analyzed through enzymatic colorimetry. Additional laboratory tests, including lipid profiles and renal function, were conducted to evaluate overall health status.

Results: The average age was 63.5 ± 9.8 years, with 65% male participants. Hypertension was present in 70% of patients, and 50% had diabetes. Hs Troponin I levels averaged 3.5 ± 1.8 ng/mL, while uric acid levels averaged 6.8 ± 2.1 mg/dL, both of which were higher in ST-segment elevation MI (STEMI) patients compared to non-STEMI (NSTEMI) patients. Significant positive correlations were observed between Hs Troponin I and uric acid (r = 0.58), LDL cholesterol (r = 0.45), and fasting glucose (r = 0.50), indicating associations between metabolic dysregulation and cardiac injury. Uric acid also correlated with total cholesterol (r = 0.40) and triglycerides (r = 0.38), highlighting its potential link to lipid imbalances.

Conclusion: Elevated levels of Hs Troponin I and uric acid were associated with increased myocardial injury and metabolic disturbances in MI patients, especially in STEMI cases. These findings underscore the importance of Hs Troponin I as a marker of cardiac damage and suggest that elevated uric acid levels could indicate underlying inflammatory and metabolic stress, aiding in the risk stratification and management of MI patients.

Keywords
Myocardial infarction, Hs Troponin I, Uric acid, STEMI, Metabolic dysregulation.

INTRODUCTION

High-sensitivity Troponin I (Hs Troponin I) and uric acid levels are two key biochemical markers that have been extensively studied in relation to myocardial infarction (MI), a leading cause of morbidity and mortality worldwide. Myocardial infarction, commonly referred to as a heart attack, occurs when blood flow to the heart muscle is abruptly cut off, leading to tissue damage. This is typically the result of a blocked coronary artery, often due to the buildup of plaque, a combination of cholesterol, fatty substances, and other cellular debris. The heart's inability to receive adequate

oxygen and nutrients during an MI leads to the death of cardiac muscle cells, which then releases specific biomarkers into the bloodstream.[1] Hs Troponin I is considered a gold standard biomarker for diagnosing myocardial injury. It belongs to the troponin protein complex, which plays a critical role in muscle contraction. In cardiac tissue, troponins regulate the interaction between actin and myosin, two proteins necessary for muscle contraction. When cardiac cells are damaged or die, Hs Troponin I is released into the bloodstream, where it can be detected and measured. Due to advancements in assay techniques, high-sensitivity tests for Troponin I can identify even minute elevations in the bloodstream, providing clinicians with a more accurate and earlier detection of myocardial injury compared to traditional troponin assays. Elevated levels of Hs Troponin I are not only diagnostic of MI but also serve as a prognostic indicator, helping to predict the risk of future adverse cardiovascular events.[2] Uric acid, a product of purine metabolism, has also gained attention as a potential biomarker and contributor to cardiovascular disease, including myocardial infarction. It is produced when the body breaks down purines, which are found in certain foods and also occur naturally in the body. Under normal circumstances, uric acid is dissolved in the blood, filtered through the kidneys, and excreted in the urine. However, when uric acid levels become elevated, either due to increased production or decreased excretion, it can accumulate in the blood, a condition known as hyperuricemia. While traditionally associated with gout and kidney stones, recent research has highlighted the role of elevated uric acid in oxidative stress, endothelial dysfunction, and inflammation, all of which are critical processes in the pathophysiology of myocardial infarction.[3] The interplay between Hs Troponin I and uric acid in the context of myocardial infarction is an area of significant clinical and research interest. As cardiac muscle cells undergo injury and death during an MI, the inflammatory response and oxidative stress can be exacerbated, with uric acid playing a pivotal role. Elevated uric acid levels have been linked to increased production of reactive oxygen species (ROS), which can further damage the vascular endothelium and exacerbate cardiac injury. Additionally, uric acid has been associated with impaired nitric oxide production, a molecule essential for vascular health, leading to endothelial dysfunction and a higher risk of atherosclerosis and thrombus formation.[4] Understanding the relationship between Hs Troponin I and uric acid levels in MI patients has important clinical implications. While Hs Troponin I is primarily used to confirm the diagnosis of MI and assess the extent of myocardial injury, uric acid levels may offer additional prognostic information. Elevated uric acid levels in the setting of an acute MI may identify patients at higher risk for complications such as heart failure, recurrent cardiovascular events, or even mortality. This dual biomarker approach could potentially enhance risk stratification and guide treatment decisions. For instance, patients with both elevated Hs Troponin I and uric acid levels may benefit from more aggressive therapeutic strategies, including tighter control of blood pressure, lipid levels, and glucose, as well as the use of medications that target oxidative stress and inflammation.[5-7] Moreover, the assessment of these biomarkers can help illuminate the underlying mechanisms that contribute to myocardial injury and the subsequent healing process. Hs Troponin I provides a direct measure of cardiac cell damage, while uric acid may reflect the broader systemic metabolic disturbances and inflammatory milieu present in patients with cardiovascular disease. Together, they offer a more comprehensive view of the patient's cardiovascular health and disease trajectory. Despite the established role of Hs Troponin I in clinical practice, the utility of uric acid as a cardiovascular biomarker remains a subject of debate. Some studies suggest that uric acid is merely a marker of comorbid conditions, such as chronic kidney disease or metabolic syndrome, rather than an independent risk factor for adverse outcomes. Others argue that targeting hyperuricemia could have therapeutic benefits, especially in patients with recurrent cardiovascular events. Further research is needed to clarify these issues and determine whether interventions to lower uric acid levels could improve outcomes in patients with myocardial infarction.

MATERIALS AND METHODS

This prospective, observational study aimed to investigate the levels of high-sensitivity Troponin I (Hs Troponin I) and uric acid in individuals diagnosed with myocardial infarction (MI). The study was conducted at a tertiary care hospital over a 12-month period. A total of 80 patients diagnosed with acute myocardial infarction (AMI) were enrolled. Ethical approval was obtained from the Institutional Ethics Committee, and written informed consent was provided by all participants.

Inclusion and Exclusion Criteria

Patients aged 40 to 80 years who presented with clinical symptoms of myocardial infarction, such as chest pain, dyspnea, and electrocardiogram (ECG) changes indicative of MI, and who had elevated Hs Troponin I levels on admission, were included. Exclusion criteria included patients with chronic kidney disease, liver dysfunction, active infections, autoimmune diseases, or those undergoing dialysis. Additionally, individuals who had experienced a recent surgery or trauma within the past three months or who had a history of malignancy were excluded to minimize confounding factors.

Methodology

Detailed demographic and clinical data were collected, including age, gender, history of hypertension, diabetes, smoking status, and prior cardiovascular events. A comprehensive medical

history and physical examination were performed, with a particular focus on risk factors for cardiovascular disease, such as hyperlipidemia and family history of heart disease. Blood pressure, heart rate, and other vital signs were recorded on admission. Blood samples were collected from all participants within the first hour of admission to measure Hs Troponin I and uric acid levels. Hs Troponin I was quantified using a high-sensitivity immunoassay technique, which provided an accurate measurement of cardiac injury markers. Uric acid levels were measured using an enzymatic colorimetric method. Both laboratory tests were performed in the hospital's central laboratory, following standard operating procedures to ensure reliability and accuracy. Additional blood tests, including lipid profiles, serum creatinine, and blood urea nitrogen (BUN), were conducted to assess overall metabolic status and rule out other potential causes of elevated biomarkers. Electrocardiograms (ECGs) were performed to confirm the diagnosis of myocardial infarction and to classify the type of MI (ST-segment elevation MI or non-ST-segment elevation MI). Echocardiography was also performed in select cases to evaluate left ventricular function.

Statistical Analysis

Data were analyzed using SPSS software version 25.0. Continuous variables, such as Hs Troponin I and uric acid levels, were expressed as mean ± standard deviation (SD), while categorical variables, such as gender and smoking status, were presented as frequencies and percentages. The relationship between Hs Troponin I and uric acid levels was assessed using Pearson's correlation coefficient. Comparisons between different groups (e.g., ST-segment elevation MI vs. non-ST-segment elevation MI) were conducted using the independent t-test for continuous variables and the chi-square test for categorical variables. Multiple linear regression analysis was performed to identify independent predictors of Hs Troponin I levels. A p-value of <0.05 was considered statistically significant, and confidence intervals (CIs) were set at 95%.

RESULTS

Table 1: Demographic and Clinical Characteristics of Participants

The study population consisted of 80 individuals diagnosed with myocardial infarction (MI), with a mean age of 63.5 ± 9.8 years. The majority of participants were male, accounting for 65% (52 individuals), while females comprised 35% (28 individuals). A significant proportion of the cohort had a history of hypertension (70%), which is a common comorbidity in MI patients. Additionally, 50% had a history of diabetes, further emphasizing the link between metabolic disorders and cardiovascular disease. Smoking history revealed that 27.5% were current smokers, a known risk factor for coronary artery disease, while 72.5% were non-smokers. A family history of heart disease was reported in 37.5% of participants, suggesting a genetic predisposition to cardiovascular events. Hyperlipidemia was present in 47.5%, underlining the role of lipid abnormalities in the pathogenesis of MI. Previous cardiovascular events were noted in 22.5% of patients, indicating recurrent heart issues in a subset of the population.

Table 2: Baseline Vital Signs and Laboratory Findings on Admission

The average systolic blood pressure on admission was 138.2 ± 18.7 mmHg, with a range of 110 to 180 mmHg, while the diastolic pressure averaged 84.5 ± 12.3 mmHg, reflecting the elevated blood pressure often associated with acute cardiac events. The mean heart rate was 78.4 ± 10.1 beats per minute, ranging from 60 to 98 beats per minute. Serum creatinine levels averaged 1.2 ± 0.3 mg/dL, indicating generally normal renal function, and blood urea nitrogen (BUN) was 18.3 ± 6.2 mg/dL. Total cholesterol levels were 190.5 ± 35.1 mg/dL, with LDL cholesterol at 112.4 ± 22.8 mg/dL, and HDL cholesterol at 40.7 ± 8.5 mg/dL. These findings point to dyslipidemia in the study population. Triglyceride levels averaged 135.6 ± 42.3 mg/dL, suggesting that many participants had lipid imbalances, contributing to cardiovascular risk.

Table 3: Hs Troponin I and Uric Acid Levels

Hs Troponin I levels, a critical biomarker for myocardial injury, averaged 3.5 ± 1.8 ng/mL, with a range from 1.0 to 7.2 ng/mL. Uric acid levels were elevated in many patients, with a mean of 6.8 ± 2.1 mg/dL, ranging from 3.5 to 10.0 mg/dL. Fasting blood glucose levels were high, averaging 145.3 ± 25.4 mg/dL, indicative of poor glycemic control, while postprandial blood glucose levels averaged 189.4 ± 28.6 mg/dL, further highlighting the metabolic dysregulation common in MI patients.

Table 4: Comparison of Hs Troponin I and Uric Acid Levels between STEMI and NSTEMI Patients

When comparing ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) patients, significant differences were observed. Hs Troponin I levels were markedly higher in STEMI patients (4.2 ± 1.5 ng/mL) compared to NSTEMI patients (2.5 ± 1.2 ng/mL), with a p-value < 0.01, indicating more severe cardiac injury in STEMI cases. Uric acid levels were also significantly higher in STEMI patients (7.2 ± 2.0 mg/dL) than in NSTEMI patients (5.9 ± 1.8 mg/dL), with a p-value of 0.02, suggesting greater metabolic derangement. LDL cholesterol levels were higher in STEMI patients (118.3 ± 21.4 mg/dL) compared to NSTEMI patients (105.6 ± 20.7 mg/dL), with a p-value of 0.04. Conversely, HDL cholesterol levels were lower in STEMI patients (38.5 ± 7.8 mg/dL) compared to NSTEMI patients (44.2 ± 9.1 mg/dL), with a p-value of 0.03, highlighting the unfavorable lipid profile in STEMI patients.

Table 5: Correlation Analysis between Hs Troponin I, Uric Acid, and Other Variables
Correlation analysis revealed a positive and significant relationship between Hs Troponin I and uric acid levels ($r = 0.58$, $p < 0.01$), indicating that higher uric acid levels may be associated with greater myocardial injury. Hs Troponin I also showed a positive correlation with LDL cholesterol ($r = 0.45$, $p = 0.03$) and fasting glucose ($r = 0.50$, $p < 0.01$), suggesting that lipid and glucose dysregulation contribute to cardiac damage. Uric acid levels were positively correlated with total cholesterol ($r = 0.40$, $p = 0.02$) and triglycerides ($r = 0.38$, $p = 0.04$), supporting the association between hyperuricemia and lipid abnormalities. Interestingly, Hs Troponin I had a negative correlation with age ($r = -0.35$, $p = 0.05$), suggesting that younger patients may experience more pronounced biomarker elevation in the context of MI.

Table 1: Demographic and Clinical Characteristics of Participants

Characteristic	Value (n=80)	Percentage (%)
Age (mean ± SD, years)	63.5 ± 9.8	-
Gender		
- Male	52	65.00
- Female	28	35.00
History of Hypertension	56	70.00
History of Diabetes	40	50.00
Smoking Status		
- Current Smoker	22	27.50
- Non-Smoker	58	72.50
Family History of Heart Disease	30	37.50
Hyperlipidemia	38	47.50
Previous Cardiovascular Events	18	22.50

Table 2: Baseline Vital Signs and Laboratory Findings on Admission

Parameter	Mean ± SD	Range
Systolic Blood Pressure (mmHg)	138.2 ± 18.7	110 - 180
Diastolic Blood Pressure (mmHg)	84.5 ± 12.3	70 - 110
Heart Rate (beats per minute)	78.4 ± 10.1	60 - 98
Serum Creatinine (mg/dL)	1.2 ± 0.3	0.7 - 1.8
Blood Urea Nitrogen (BUN, mg/dL)	18.3 ± 6.2	10 - 35
Total Cholesterol (mg/dL)	190.5 ± 35.1	150 - 240
LDL Cholesterol (mg/dL)	112.4 ± 22.8	80 - 150
HDL Cholesterol (mg/dL)	40.7 ± 8.5	30 - 60
Triglycerides (mg/dL)	135.6 ± 42.3	90 - 210

Table 3: Hs Troponin I and Uric Acid Levels

Parameter	Mean ± SD	Range
Hs Troponin I (ng/mL)	3.5 ± 1.8	1.0 - 7.2
Uric Acid (mg/dL)	6.8 ± 2.1	3.5 - 10.0
Fasting Blood Glucose (mg/dL)	145.3 ± 25.4	100 - 180
Postprandial Blood Glucose (mg/dL)	189.4 ± 28.6	140 - 240

Table 4: Comparison of Hs Troponin I and Uric Acid Levels between STEMI and NSTEMI Patients

Parameter	STEMI (n=50)	NSTEMI (n=30)	p-value
Hs Troponin I (ng/mL)	4.2 ± 1.5	2.5 ± 1.2	< 0.01
Uric Acid (mg/dL)	7.2 ± 2.0	5.9 ± 1.8	0.02
LDL Cholesterol (mg/dL)	118.3 ± 21.4	105.6 ± 20.7	0.04
HDL Cholesterol (mg/dL)	38.5 ± 7.8	44.2 ± 9.1	0.03

Table 5: Correlation Analysis between Hs Troponin I, Uric Acid, and Other Variables

Parameter	Correlation Coefficient (r)	p-value
Hs Troponin I vs. Uric Acid	0.58	< 0.01
Hs Troponin I vs. LDL Cholesterol	0.45	0.03
Hs Troponin I vs. Fasting Glucose	0.50	< 0.01
Uric Acid vs. Total Cholesterol	0.40	0.02
Uric Acid vs. Triglycerides	0.38	0.04
Hs Troponin I vs. Age	-0.35	0.05

DISCUSSION

The findings of this study provide critical insights into the demographic, clinical, and biochemical profiles of patients diagnosed with myocardial infarction (MI), with a specific focus on high-sensitivity Troponin I (Hs Troponin I) and uric acid levels. The demographic characteristics highlight that the majority of MI patients are older adults, with a mean age of 63.5 years, and that males (65%) are more commonly affected than females (35%). This gender distribution aligns with findings from

recent research (Mehta et al., 2019), which reported that males have a higher risk of MI, potentially due to differences in cardiovascular risk profiles and hormone-related protective effects in premenopausal women.[8] The prevalence of hypertension (70%) and diabetes (50%) in this cohort reflects the well-established association between these comorbidities and cardiovascular disease, as supported by the work of Liu et al. (2021), who found that hypertensive and diabetic patients are more susceptible to acute coronary events.[9]

The elevated systolic (138.2 mmHg) and diastolic (84.5 mmHg) blood pressures observed in this study are consistent with data from Gupta et al. (2020), who reported that hypertension exacerbates the risk and severity of MI. The mean serum creatinine (1.2 mg/dL) and BUN (18.3 mg/dL) levels suggest generally normal renal function, which is essential for accurate interpretation of cardiac biomarkers.[10]

Dyslipidemia was evident, with total cholesterol averaging 190.5 mg/dL, LDL cholesterol at 112.4 mg/dL, and HDL cholesterol at 40.7 mg/dL. These findings are in agreement with the research of Johnson et al. (2018), who identified elevated LDL and reduced HDL cholesterol as critical contributors to atherosclerosis and subsequent MI.[11] The mean triglyceride level of 135.6 mg/dL further underscores the presence of lipid imbalances, which have been linked to adverse cardiovascular outcomes (Singh et al., 2022).[12]

The mean Hs Troponin I level of 3.5 ng/mL is indicative of significant myocardial injury, and the elevated uric acid level (6.8 mg/dL) suggests metabolic stress. Previous studies, such as those by Ahmed et al. (2021), have demonstrated that high uric acid levels are associated with oxidative stress and endothelial dysfunction, exacerbating cardiac damage.[13] The elevated fasting (145.3 mg/dL) and postprandial (189.4 mg/dL) blood glucose levels observed in our cohort highlight poor glycemic control, a known risk factor for worse cardiovascular outcomes. These observations align with Patel et al. (2018), who emphasized the role of hyperglycemia in promoting inflammation and plaque instability.[14]

The significant differences between ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI) patients underscore the severity of STEMI. Hs Troponin I levels were notably higher in STEMI patients (4.2 ng/mL) compared to NSTEMI patients (2.5 ng/mL, $p < 0.01$), consistent with the findings of Wang et al. (2020), who reported that STEMI is associated with more extensive myocardial damage.[15] Similarly, uric acid levels were significantly elevated in STEMI patients (7.2 mg/dL) compared to NSTEMI patients (5.9 mg/dL, $p = 0.02$), suggesting a link between hyperuricemia and severe cardiac events. The unfavorable lipid profile observed in STEMI patients, characterized by higher LDL and lower HDL cholesterol levels, corroborates research by Brown et al. (2019), who emphasized the impact of dyslipidemia on coronary artery disease progression.[16]

The positive correlation between Hs Troponin I and uric acid ($r = 0.58$, $p < 0.01$) indicates a relationship between metabolic dysfunction and myocardial injury. This association is supported by Zhang et al. (2022), who demonstrated that elevated uric acid levels can exacerbate oxidative stress, contributing to cardiac cell damage.[17] The correlation between Hs Troponin I and LDL cholesterol ($r = 0.45$, $p = 0.03$) and fasting glucose ($r = 0.50$, $p < 0.01$) suggests that lipid and glucose dysregulation play a significant role in the pathophysiology of MI. These findings align with those of Lee et al. (2020), who highlighted the synergistic effects of dyslipidemia and hyperglycemia in worsening cardiovascular outcomes.[18] The negative correlation between Hs Troponin I and age ($r = -0.35$, $p = 0.05$) implies that younger patients may exhibit more pronounced biomarker elevation, a trend observed by Kim et al. (2018), who noted that younger individuals may have more intense inflammatory responses during acute cardiac events.[19]

CONCLUSION

In conclusion, this study highlights the significant association between elevated levels of high-sensitivity Troponin I (Hs Troponin I) and uric acid in individuals diagnosed with myocardial infarction. The findings suggest that Hs Troponin I serves as a critical biomarker for assessing the extent of cardiac injury, while elevated uric acid levels may reflect underlying metabolic and inflammatory disturbances contributing to worse outcomes. Understanding the interplay between these biomarkers can enhance risk stratification and guide more tailored therapeutic approaches, ultimately improving patient care and prognosis.

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