RESEARCH PAPER

Serum Malondialdehyde and Uric Acid in Acute Myocardial Infarction: Comparative Levels and Inter-Marker Correlation

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Abstract

Background

Acute myocardial infarction (AMI) is one of the leading causes of morbidity and mortality worldwide. Oxidative stress plays an important role in myocardial injury and progression of cardiovascular disease. Malondialdehyde (MDA), a marker of lipid peroxidation, and serum uric acid, a metabolic antioxidant marker associated with oxidative injury, have been implicated in ischemic heart disease.

Aim

To assess serum uric acid and malondialdehyde levels in patients with acute myocardial infarction and to investigate their role as markers of oxidative stress in comparison with healthy individuals and correlation with troponin.

Methods

A hospital-based case–control study was conducted among 230 participants, including 115 AMI cases and 115 age- and sex-matched healthy controls. MDA measured by TBARS using HPLC, Troponin levels were estimated using a chemiluminescence-based immunoassay, Uric acid by uricase method. Since data distribution was non-normal, Mann–Whitney U test and Spearmen Rank Correlation Coefficient test were applied.

Results

Mann–Whitney U test was applied, Median age in control was 64 (54-73) and in cases 67 (54-75) years was not significant ($p = 0.313$), MDA level in cases was [0.632 (0.490–0.883) µmol/L vs 0.192 (0.126–0.316) µmol/L], uric acid [7.02 (5.10–8.70) mg/dL vs 3.80 (2.79–4.89) mg/dL], and Troponin I [1.234 (0.128–6.176) ng/mL vs 0.006 (0.003–0.012) ng/mL] compared with controls (all $p<0.001$). A strong positive correlation was observed between MDA and troponin levels ($r = +0.759$, $p <0.001$), MDA also showed a moderate positive correlation with uric acid ($r = +0.497$, $p <0.001$). Additionally, troponin demonstrated a moderate positive correlation with uric acid ($r = +0.597$, $p <0.001$)

Discussion:

Serum MDA and uric acid levels were significantly elevated in AMI patients compared with controls, indicating increased oxidative stress during myocardial injury. Both biomarkers showed significant positive correlations with troponin, suggesting an association with the severity of myocardial damage. These findings support the potential utility of MDA and uric acid as adjunctive biomarkers in AMI.

Conclusion

The present study demonstrated significant positive correlations between MDA, Troponin, and uric acid levels in acute myocardial infarction patients. The strong association between MDA and Troponin suggests that increased oxidative stress is closely related to the extent of myocardial injury. These findings support the potential role of oxidative stress biomarkers, particularly MDA, in the pathophysiology and assessment of acute myocardial infarction.

Keywords; Acute Myocardial Infarction; Spearman Rank Correlation; Mann Whitney U test; Oxidative stress; Malondialdehyde; Uric acid; Troponin I; Thiobarbituric acid Reactive Oxygen Species

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Introduction

Myocardial infarction, the most frequent type of coronary heart disease, accounts for more than 15% of

cardiovascular deaths each year.[1,2] The following is how its burden is distributed: 20% in high-income countries, 8% in upper-middle-income countries, 37% in lower-middle-income countries, and 35% in low-income countries like India.[3] In India, there are an estimated 64.37 myocardial infarctions for per 1,000 individuals.[4]

One of the leading causes of death and morbidity worldwide is acute myocardial infarction (AMI).[5] The most common cause of AMI is atherosclerotic coronary artery disease (CAD), which is defined by the erosion or rupture of a plaque that results in temporary, partial, or total arterial occlusion.[6] Troponin I is a dependable diagnostic sign for AMI since it is a highly specific indicator of cardiac muscle injury. After a cardiac attack, troponin I levels start to rise within hours and usually peak 24 to 48 hours later.[7]

Lipid peroxidation, an oxidative modification of membrane lipids, is a crucial element in the pathogenesis of atherosclerosis and inflammatory conditions. The persistent cycle of free radical generation leads to ongoing peroxidative damage, and severe lipid peroxidation has been associated with the onset of ischemic heart disease, including angina and acute myocardial infarction (AMI).[8,9] Malondialdehyde (MDA) is a widely utilized biochemical marker of oxidative lipid damage in human research, serving as a stable terminal consequence of polyunsaturated fatty acid peroxidation. Numerous studies have demonstrated a correlation between oxidative stress and the pathogenesis of acute myocardial infarction (AMI), revealing significantly elevated serum malondialdehyde (MDA) levels in individuals with cardiac disease.[10,11] Reactive oxygen species (ROS) and compromised endogenous antioxidant defense systems cause myocardial injury, which is mostly caused by atherosclerosis.[12]

Elevated serum uric acid (SUA) levels are associated with pathogenic processes such as increased oxidative stress, systemic inflammation, and endothelial dysfunction.[13] Recent epidemiological studies suggest that elevated SUA may be associated with acute myocardial infarction. Uric acid levels usually increase during AMI due to accelerated cellular breakdown, particularly of nucleic acids, as a result of ischemic tissue injury. This increase may worsen oxidative stress and inflammation because it has been associated with worse clinical outcomes in people with AMI. Elevated uric acid during AMI may be a sign of increased cardiovascular risk and more severe myocardial injury.[14] Therefore, the current study's objectives were to measure and compare the blood levels of MDA and uric acid in AMI patients and healthy controls, evaluate their potential as AMI diagnostic biomarkers, and examine their association with high-sensitivity Troponin I.

Method

Study Design and Setting

Between December 2023 and December 2025, Shree Krishna Hospital, a tertiary care facility in Karamsad, Gujarat, India, carried out prospective case-control research. A total of 230 participants were enrolled, comprising 115 consecutive patients with AMI and 115 healthy controls. The emergency medicine and cardiology departments progressively recruited patients, regardless of gender, who had experienced their first episode of acute myocardial infarction (AMI) and were ≥18 years old. The diagnosis was made based on high serum Troponin I levels, characteristic pain in the chest, and abnormalities in ECG suggestive of myocardial infarction.

Individuals having a history of myocardial infarction, established cardiovascular disease, recent trauma or surgery, active infection or inflammatory disease, cancer, long-term liver or renal disease, pregnancy, chronic systemic illness, or drugs that influence oxidative stress biomarkers were excluded. Healthy, age-and sex-matched individuals without a history of cardiovascular disease met the inclusion criteria for the control group. The Institutional Ethics Committee (IEC/BU/153/Faculty/15/048/2025) authorized the study procedure, which was carried out in compliance with the Declaration of Helsinki. Because the study was observational and used samples obtained during standard clinical investigations, informed consent was waived.

Aseptic venous blood (around 5 mL) was drawn in plain tubes under aseptic circumstances. Samples of serum were allowed to clot at room temperature before being centrifuged for 20 minutes at 35000 RPM. the serum was separated into eppendorf tubes and kept at -80°C until analysis. A sandwich immunoassay based on flash chemiluminescence technology was used on the XPT analyzer to quantify troponin I. High-performance liquid chromatography (HPLC) in conjunction with ultraviolet detection (HPLC–UV) was used to measure serum malondialdehyde (MDA) as thiobarbituric acid reactive substances (TBARS).[15,16] The uricase–peroxidase enzymatic endpoint approach with spectrophotometric detection was used to quantify serum uric acid levels.

The data was analyzed using SPSS version 27 and STATA 19. To ascertain whether the data distribution was normal, the Q-Q plot, histogram, and Kolmogorov-Smirnov test were employed. Continuous variables were expressed using the median and interquartile range (IQR). Group comparisons were conducted using the Mann-Whitney U test (Wilcoxon rank-sum test), and correlations were evaluated using Spearman's rank correlation analysis. A p-value of less than 0.05 was considered statistically significant.

Results

Of the 230 patients in the study, 115 had experienced an acute myocardial infarction (AMI), and 115 were healthy controls. The Kolmogorov-Smirnov test showed that all research variables had non-normal distributions, hence non-parametric statistical methods

were employed. The IQR (interquartile range) and median of continuous variables were recorded, and the groups were compared using the Mann-Whitney U test. Serum levels of malondialdehyde (MDA), uric acid, and troponin were considerably higher in AMI patients than in controls ($p < 0.001$). Higher uric acid levels indicated altered antioxidant status and endothelial dysfunction, while higher MDA levels indicated increased oxidative damage and lipid peroxidation. Serious myocardial injury was indicated by the markedly raised troponin levels in patients with AMI. Overall, the results show that patients with AMI have a significant burden of oxidative stress and emphasize the critical role that oxidative damage plays in the development and course of acute myocardial infarction.

Table: 1 Gender-wise distribution of study participants among acute myocardial infarction (AMI) cases and control groups.

Gender	Controls	Cases	Total	p-value (χ^2)
Male	63	66	129	0.790
Female	52	49	101	
Total	115	115	230	

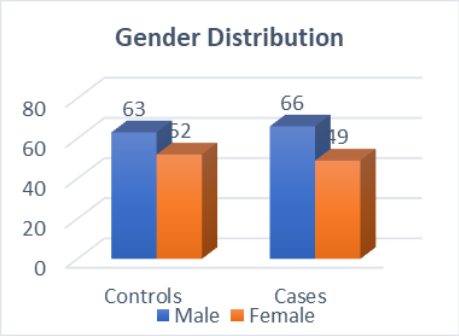


Table: 2 Group comparisons were analyzed using the Mann–Whitney U test. Values are expressed as median with interquartile range (Q1–Q3). Asterisks denote statistical significance (*p < 0.05; **p < 0.01; ***p < 0.001).

Study Variables	Median (IQR) in Controls	Median (IQR) in Cases	Z Value	Statistical Significance (p)
Age (years)	64 (54–73)	67 (54–75)	-1.010	0.313
Troponin (ng/mL)	0.006 (0.003–0.012)	1.234 (0.128–6.176)	-13.119	<0.001 ***
MDA (µmol/L)	0.192 (0.126–0.316)	0.632 (0.490–0.883)	-10.752	<0.001 ***
Uric Acid (mg/dL)	3.80 (2.79–4.89)	7.02 (5.10–8.70)	-8.083	<0.001 ***

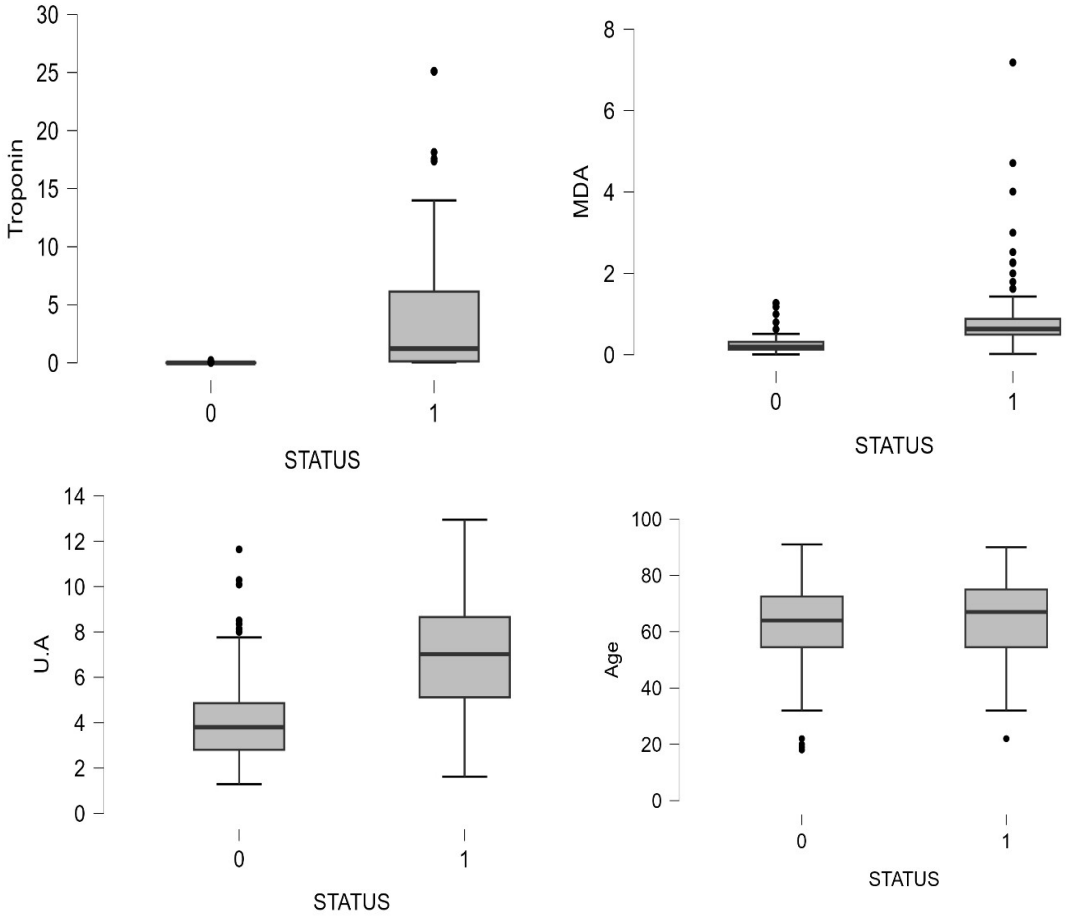
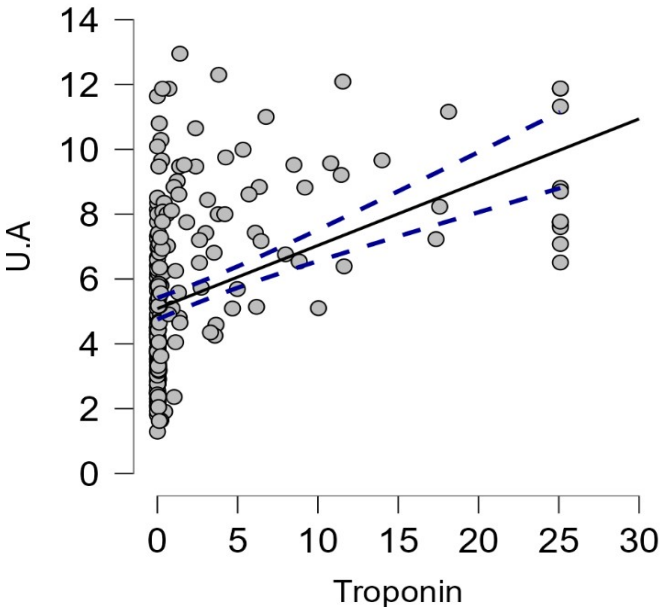


Figure:1 Whisker Box Plot of Troponin, Malondialdehyde, Uric Acid and Age (0 = control, 1= cases).

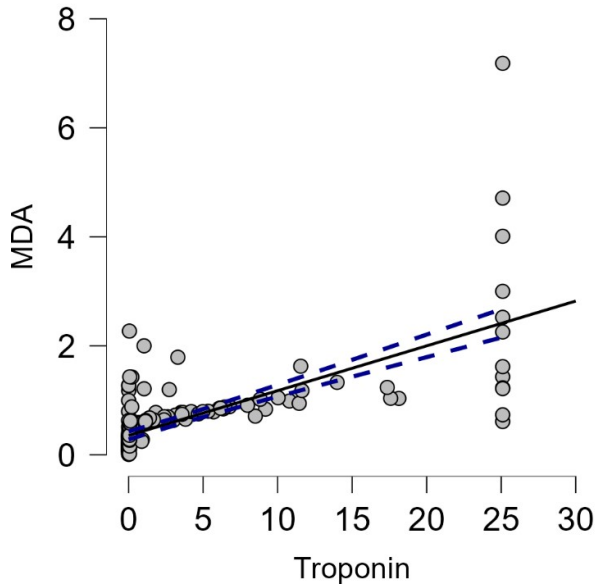
Table: The relationships between serum troponin, uric acid (UA), and malondialdehyde (MDA) were evaluated using Spearman's Correlation test (individual data points overlaid * p <.05, ** p <.01, *** p <.001).

Study Variables			**Sample Size**	**Spearman Correlation Coefficient**	**Lower Confidence Limit (95% CI)**	**Upper Confidence Limit (95% CI)**	**Fisher's z Value**	**SE of Fisher's z**
Troponin	-	U. A	230	0.597***	0.522	1.000	0.688	0.070
Troponin	-	MDA	230	0.759***	0.709	1.000	0.994	0.071
U. A	-	MDA	230	0.497***	0.410	1.000	0.545	0.069



ρ = 0.597

95% CI: [0.522, 1.000]



ρ = 0.759

95% CI: [0.709, 1.000]

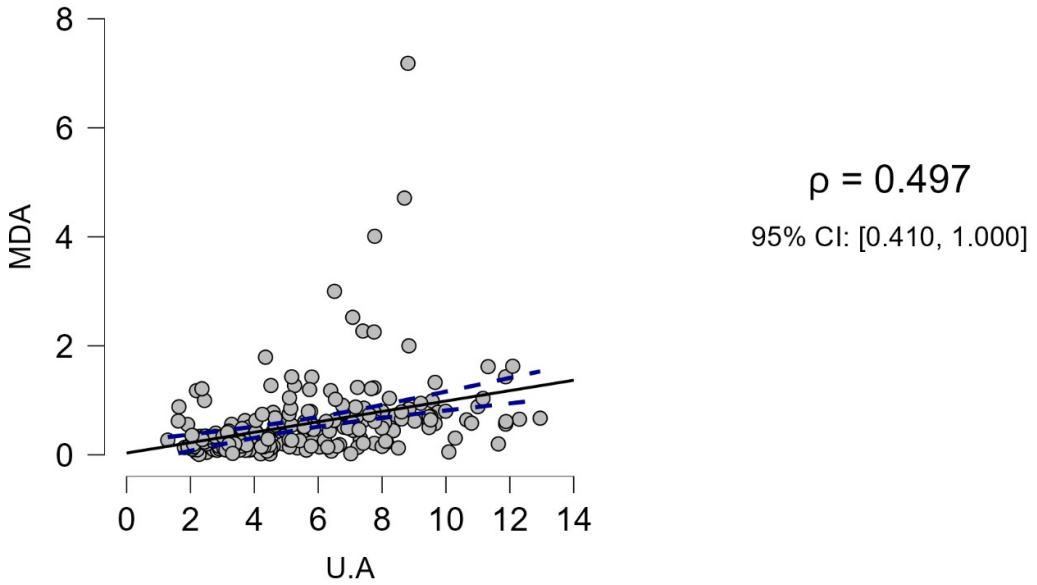


Figure: 2 Scatter Plot for Spearman Rank Correlation

Discussion

Reactive oxygen species (ROS) production, endothelial dysfunction, cytokine release, complement activation, and leukocyte-mediated myocardial injury are just a few of the complex oxidative and inflammatory processes that are triggered by ischemia-reperfusion injury in acute myocardial infarction (AMI). These processes promote irreversible cardiomyocyte injury, lipid peroxidation, and the instability of atherosclerotic plaques.[17–20] Malondialdehyde (MDA), a persistent consequence of the peroxidation of lipids, is thought to be a trustworthy indicator of oxidative stress in cardiovascular disease.[21]

MDA levels were 3.3 times more in AMI patients [0.632 µmol/L (IQR: 0.490–0.883)] than in controls [0.192 µmol/L (IQR: 0.126–0.316), $p < 0.001$]. These results are in line with those of Kumar and Sivakanesan[22], Kaur et al.[23], Raghuvanshi et al.[24], Cavalca et al.[25] and Tamer et al.[26] who also found that patients with coronary artery disease, atherosclerosis, and acute myocardial infarction had significantly higher MDA concentrations. Furthermore, Jain et al.[27] found that MDA and nitrite plasma levels were considerably higher in AMI patients than in the healthy controls, suggesting that oxygen free radicals harm patients endothelium. Bashar and Akhter[28] demonstrated the temporal endurance of this oxidative injury by showing that although MDA concentrations drop with therapy, they remain above normal reference values, indicating a condition of sustained oxidative stress beyond the initial ischemic episode.

Furthermore, AMI patients had dramatically greater uric acid levels [7.02 mg/dL (IQR: 5.10–8.70)] than controls [3.80 mg/dL (IQR: 2.79–4.89), $p < 0.001$].This clear distinction highlights the paradoxical nature of uric acid in cardiovascular biology—a molecule that, at physiological concentrations, acts as a potent mediator of oxidative and endothelial damage when serum levels exceed homeostatic thresholds.[29] High levels of hyperuricemia trigger inflammatory reactions in the endothelium, deplete bioavailable nitric oxide, generate xanthine oxidase-dependent superoxide anions, and trigger downstream pathways that hasten the development of atherosclerosis.[30-32]

The abrupt rise in xanthine oxidase activity caused by nucleotide catabolism during ischemia in the setting of acute myocardial ischemia offers a mechanistically sound explanation for the hyperuricemia seen in our AMI cohort. Because of this metabolic relationship, serum uric acid is positioned as a potentially helpful supplemental marker in the spectrum of acute coronary syndrome. The results presented here are consistent with those of Kojima et al.[33] Bickel et al.[34] along with Jacobs[35] who found elevated uric acid was an independent predictor of myocardial infarction, cardiovascular problems, and higher mortality risk; this suggests that uric acid may actively contribute to the pathobiology of acute coronary events rather than merely being an epiphenomenon of metabolic disruption. The hyperuricemia observed in our AMI group can be explained mechanistically by the abrupt rise in xanthine oxidase activity, which is brought on by nucleotide catabolism during ischemia. uric acid is positioned as a potentially useful additional measure across the range of acute cardiac disease due to this metabolic link.

The presence of Troponin I in the systemic circulation is a recognized indication for the diagnosis of AMI and a dependable indicator of myocardial injury because it is almost exclusively expressed in heart muscle; serum levels peak between 24 and 48 hours after the beginning of ischemia and begin to climb within hours.[7] In the current investigation, troponin values were substantially higher in AMI patients [1.234 ng/mL (IQR: 0.128–6.176)] than in controls [0.006 ng/mL (IQR: 0.003–0.012); $p < 0.001$]. This result is in line

with research by Reichlin et al.[36] and Keller et al.[37] that showed the exceptional diagnostic accuracy of high-sensitivity troponin testing in AMI.

MDA and troponin showed a substantial positive correlation ($\rho = 0.759$, $p < 0.001$), suggesting a connection between the degree of myocardial damage and oxidative stress. Similar results were reported by Mehri et al.[38] and Pallavi et al.[39], who found a significant relationship between cardiac biomarkers and oxidative stress markers in people with AMI. Additionally, there was a significant association ($\rho = 0.597$, $p < 0.001$) between serum uric acid and troponin, which is in line with findings by Sarna et al.[40] that indicate higher uric acid levels are linked to increased myocardial necrosis. Additionally, the coexistence of oxidative damage linked to purine metabolism and lipid peroxidation during acute ischemia is supported by the moderately positive association between MDA and uric acid ($\rho = 0.497$, $p < 0.001$), which is in line with results published by Iliesiu et al.[41]

Conclusion

The current investigation showed that individuals with acute myocardial infarction (AMI) had considerably higher levels of MDA, troponin, and uric acid as well as strong positive associations between these biomarkers. The strong association between MDA and troponin highlights the important role of oxidative stress in myocardial injury. While troponin remains the gold standard biomarker for AMI diagnosis, MDA and uric acid may serve as valuable adjunctive biomarkers by providing additional insight into oxidative and metabolic disturbances associated with acute coronary events. These findings suggest that oxidative stress biomarkers, particularly MDA, may have potential utility in the assessment and risk stratification of AMI patients.

Limitations

There are some limitations in the current investigation. The results may not be as broadly applicable as they could be, because it was carried out at a single center with a moderate sample size. Furthermore, a causal association between oxidative stress indicators and AMI cannot be established due to the cross-sectional methodology. Since there was no long-term monitoring, it was unable to determine the prognostic importance of MDA and uric acid. Moreover, dietary practices and drugs that affect serum uric acid levels were not assessed.

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