

Assessment of Hematological and Biochemical Markers in Saudi Patients with Diabetes and Non-Diabetic Acute Myocardial Infarction

Kamal Ahmad^{1,*}, Raju K Mandal², Mohd Wahid², Bincy Vincent², Majed Ali Suwaydi³, Sheela prabhudas², Elsie L Maguen⁴, Muhammad H. Khawaji³, Mathkour Hazem³, Mohammad Shane Alam³

¹Department of Botany, Islamia College, Darbhanga, Bihar, INDIA.

²Department of Nursing, College of Nursing and Health Sciences, Jazan University, Jazan, KSA.

³Department of Medical Laboratory Technology, College of Nursing and Health Sciences, Jazan University, Jazan, KSA.

⁴Department of Nursing, College of Nursing and Health Sciences, EMS Program, KSA.

ABSTRACT

Background: Diabetes Mellitus (DM) is a long-term metabolic disease that causes increased oxidative stress and a higher risk of cardiovascular problems, particularly Acute Myocardial Infarction (AMI). This paper tried to compare the biochemical, lipid, and oxidative stress parameters between diabetic and non-diabetic AMI patients in order to comprehend the metabolic changes associated with cardiac injury. **Purpose:** The study aimed to compare biochemical, lipid, and oxidative stress markers among normal controls, non-diabetic AMI patients, and diabetic AMI patients. It also aimed to assess how diabetes influences myocardial injury, inflammation, and oxidative stress in AMI. **Materials and Methods:** 600 subjects were assigned to three groups: Normal controls ($n=200$), Non-Diabetic AMI (ND-AMI, $n=200$), and Diabetic AMI (D-AMI, $n=200$). Blood samples collected after fasting were analyzed for cardiac markers (Troponin I, CK-MB, CRP), lipid profile (Total Cholesterol, Triglycerides, LDL, HDL), and oxidative stress markers (MDA, SOD, Catalase, GSH). The statistical analysis of the data was performed using ANOVA, t-tests, and correlation in SPSS v24. **Results:** The level of Troponin I, CK-MB, CRP, Total Cholesterol, Triglycerides, LDL, and MDA in the D-AMI patients was significantly higher than that of ND-AMI and Normals ($p<0.001$). On the other hand, there was a significant decrease in HDL, SOD, and GSH levels. There were positive correlations between Troponin I and CK-MB, CRP, LDL, MDA, and SOD, and GSH. **Conclusion:** The results have shown that diabetic patients with AMI have more myocardial damage and inflammation with increased oxidative stress levels in comparison with non-diabetic patients with AMI. Lipid abnormalities and low antioxidant defenses during diabetes increase myocardial injury. The use of oxidative and biochemical markers to monitor may enhance risk prediction and therapy selection in AMI, particularly in populations with diabetes.

Keywords: Antioxidants, Acute Myocardial Infarction, Cardiac Biomarkers, Diabetes Mellitus, Lipid Profile, Oxidative Stress.

Correspondence:

Kamal Ahmad

Department of Botany, Islamia College,
Darbhanga, Bihar, INDIA.

Email: kamalahmad8@gmail.com

Received: 21-07-2026;

Revised: 18-08-2026;

Accepted: 07-10-2026.

INTRODUCTION

The leading cause of death on a global scale is Cardiovascular Diseases (CVDs). One of the most risky and potentially fatal types of CVD that result in a considerable percentage of such deaths is Acute Myocardial Infarction (AMI) (Nesbitt *et al.*, 2025). An acute coronary occlusion leads to AMI, ultimately resulting in an ischemic injury and myocardial necrosis. ACS is a serious risk factor and predictor influenced by the severity

of Diabetes Mellitus (DM) (Stampouloglou *et al.*, 2023; Smith *et al.*, 1984). It is a chronic metabolic illness, which is defined by chronic hyperglycemia due to insulin resistance or insulin deficiency and predisposes diabetic patients to AMI two to four times (Ormazabal *et al.*, 2018). This leads to increased myocardial injury, recovery, and poor clinical outcome of such patients as compared to patients who are non-diabetic.

In India, much like in most of the developing countries, diabetes and AMI are on the rise rather fast due to urbanization, sedentary lifestyle, and dietary modification (Kumar *et al.*, 2020). This dual epidemic has imposed a major public health burden (Mohan *et al.*, 2010). The current research was carried out in the Darbhanga area, which has been characterized by a significant increase in heart-related complications brought about by diabetes. Knowledge of the biochemical differences between diabetic and



DOI: 10.5530/ijpi.20270109

Copyright Information :

Copyright Author (s) 2027 Distributed under
Creative Commons CC-BY 4.0

Publishing Partner : Manuscript Technomedia. [www.mstechnomedia.com]

non-diabetic patients with ACS in these regional populations is important for the design of specific prevention and management measures (Sabitha *et al.*, 2024; Ali *et al.*, 2016).

Biochemical and hematological markers are essential tools for identifying myocardial injury and metabolic alterations (Tidy *et al.*, 2019). Cardiac Troponin I and Creatine Kinase-MB (CK-MB) are specific indicators of myocardial cell damage (Daubert *et al.*, 2010; Wega *et al.*, 2021). Lipid profile parameters such as total cholesterol, triglycerides, LDL, and HDL reflect the extent of dyslipidemia, a major contributor to atherosclerosis and coronary artery disease (Puri *et al.*, 2023). Serum levels of inflammatory markers, such as C-Reactive Protein (CRP), and oxidative stress markers, such as Malondialdehyde (MDA), Superoxide Dismutase (SOD), and Glutathione (GSH), provide additional insight into the underlying oxidative and inflammatory stress that promotes myocardial injury, at least in diabetic individuals (Narres *et al.*, 2022; Cecerska-Heryć *et al.*, 2025).

This research is intended to evaluate and compare these hematological and biochemical parameters within the Normal, Non-Diabetic AMI (ND-AMI) and Diabetic AMI (D-AMI) samples. The study aims to determine the effects of diabetes on myocardial damage and metabolic imbalances by evaluating cardiac enzymes, lipid levels, and oxidative stress markers. It is hoped that the results of the research will contribute to improved diagnostic accuracy, earlier intervention, and the clinician's orientation toward personalized therapeutic interventions for AMI patients with and without diabetes.

MATERIALS AND METHODS

Study Design and Population

The current research is a hospital-based comparative study conducted in the Department of Biochemistry at Darbhanga Medical College and Hospital, Bihar. A group of 600 subjects was selected and then stratified into three groups, each of which had 200 subjects: (i) healthy controls; (ii) acute myocardial infarction in the Non-Diabetic (ND-AMI) group; (iii) acute myocardial infarction in the Diabetic (D-AMI) group. Diagnostic accuracy was achieved by combining clinical symptomatology, electrocardiographic findings, and elevated cardiac biomarker levels to diagnose acute myocardial infarction. Diabetic status was determined by fasting plasma glucose ≥ 126 mg/dL or $\text{HbA}_{1c} \geq 6.5\%$, according to American Diabetes Association (ADA) criteria (ADA, 2023).

Inclusion Criteria

The study included male and female participants aged 35-80 years. Healthy individuals without cardiovascular, diabetic, renal, or hepatic disorders were taken as controls. ND-AMI patients had acute myocardial infarction without diabetes ($\text{FPG} < 126$ mg/dL, $\text{HbA}_{1c} < 6.5\%$), whereas D-AMI patients had AMI with diabetes ($\text{FPG} \geq 126$ mg/dL or $\text{HbA}_{1c} \geq 6.5\%$) according to the ADA 2023

criteria (Cui *et al.*, 2012). All participants gave written informed consent before sample collection.

Exclusion Criteria

Patients with chronic liver or renal disease, thyroid disorders, and chronic inflammatory conditions were barred. No one taking antioxidant supplements, lipid-lowering medications, or anti-inflammatory medications was included, either. The study excluded subjects who were recently infected, had cancer lesions, autoimmune diseases, were pregnant women, lactating women, or had uncertain records or lacked the willingness to take part in the study (Ceriello *et al.*, 2009).

Sample Collection

Under aseptic precautions, 5 mL of venous blood was drawn from each participant after overnight fasting. The blood samples were divided into two parts:

- Serum was separated by centrifugation at 3000 rpm for 10 min and used for biochemical assays.
- EDTA-treated plasma was used for hematological parameters.

All analyses were performed within 2 hr of collection to maintain sample integrity.

Biochemical Analysis

The levels of creatine kinase-MB and serum cardiac troponin I were tested using an automated chemiluminescent immunoassay analyzer. The level of C-reactive protein was measured using a highly sensitive immunoturbidimetric method. The lipid profile parameters (total cholesterol, triglycerides, High-Density Lipoprotein (HDL), and Low-Density Lipoprotein (LDL)) were measured using enzymatic colorimetric kits (Erba Diagnostics, India).

Oxidative Stress Markers

The Thiobarbituric Acid Reactive Substances (TBARS) method, which assesses lipid peroxidation, was used to measure Malondialdehyde (MDA) levels. The pyrogallol autoxidation method, developed by Marklund and Marklund (1974), was used to assess Superoxide Dismutase (SOD) activity (Yao *et al.*, 2021). Ellman's reagent was used to measure Glutathione (GSH) concentration, and the Aebi method (1984) was used to assess Catalase (CAT) activity (De Leon *et al.*, 2020). A UV-visible spectrophotometer was used to take all absorbance measurements.

Hematological and Blood Pressure Measurement

An automated hematology analyzer was used to determine the Hemoglobin (Hb) concentration. The systolic and diastolic blood pressures (SBP, DBP) after 10 min of rest in a sitting position were measured using a routine mercury sphygmomanometer.

Statistical Analysis

The data was analyzed with SPSS version 24.0. The qualitative data were summarized in the form of frequencies or percentages, and the quantitative variables in the form of mean $\pm$ SD. We have used the Shapiro-Wilk test to test the normality of the continuous distributions. Intergroup comparison among the three research groups was conducted using one-way ANOVA, with the assumption of homogeneity of variances addressed by a Kruskal-Wallis test, followed by an independent-samples t-test between the study groups ND-AMI and D-AMI at the pairwise level. Pearson correlation coefficients were used to assess the relationships between Troponin I concentrations and biochemical markers, and multiple linear regression was performed to identify independent predictors of Troponin I levels. The statistical significance of *p*-values less than 0.05 (two-tailed) was considered significant.

RESULTS

The current research compared demographic and biochemical factors, as well as oxidative stress indicators, among the Normal, ND-AMI, and D-AMI groups. Continuous variables are presented as means with standard deviations, and categorical variables as frequencies and percentages. Depending on the characteristics of the variable distributions, one-way analysis of variance, the Kruskal-Wallis test, and independent-samples t-tests were used to test differences between the groups. In addition, the correlation and regression analyses were conducted to both test the relationship and to identify predictors of cardiac biomarkers. The most important findings have been summarized below.

Demographic and Clinical Characteristics

Table 1 shows that there was no statistically significant difference between the mean age and sex distribution between the three cohorts, and thus, the demographic profile was consistent. This homogeneity in age affirms that later biochemical differences were driven by disease rather than senescence. Although there was no significant difference between baseline hematological parameters like leukocyte and platelet counts, there was a significant, though mild, decrease in hemoglobin in the acute myocardial infarction arms. It is interesting to note that systolic and diastolic pressures increased steadily from the healthy control group to the non-diabetic AMI group and culminated in the diabetic AMI group, clearly indicating that hypertension is aggressive when combined with glycemic derangement in infarction (Figure 1A).

Biochemical, Lipid, and Oxidative Stress Parameters

Table 2 shows that there is a definite pathological gradient of the groups, and the AMI diabetic patients have the greatest biochemical load. The indices of cardiac injury- Troponin I, CK-MB, and C-reactive protein were significantly increased in diabetic patients in comparison to non-diabetic AMI patients

and healthy controls. The lipid panel reflected this degree of severity, with increased total cholesterol, triglycerides, and low-density lipoprotein, and reduced high-density lipoprotein, in the diabetic group. Moreover, antioxidant defenses such as superoxide dismutase, catalase, and reduced glutathione were significantly reduced in diabetic AMI, and malondialdehyde levels were elevated, all of which indicate that diabetes enhances oxidative injury and suppresses protective enzyme activity during myocardial infarction (Figure 1B).

Intergroup Statistical Comparisons and Normality Assessment

Table 3 results revealed that most continuous variables did not meet the Gaussian assumption, necessitating the use of non-parametric tests for several comparisons. The across-group analysis revealed significant differences in almost all cardiac, inflammatory, lipid, and oxidative stress parameters, whereas age, white blood cell count, and platelet count were not statistically different. These differences were further highlighted by pairwise comparisons between diabetic and non-diabetic ACS patients, which revealed statistically significant disparities in Troponin I, CRP, LDL, HDL, malondialdehyde, and superoxide dismutase. These results confirm that diabetes by itself exacerbates the metabolic and inflammatory disturbances in acute coronary syndromes (Figure 1C).

Correlation and Multiple Linear Regression Analysis

Bivariate analysis, as shown in Table 4, showed that an increase in Troponin I was correlated with increases in CK-MB, CRP, LDL, and malondialdehyde, but was anti-correlated with superoxide dismutase activity. Multivariate models further reduced these relations: CRP and malondialdehyde became strong positive determinants of myocardial injury, whereas HDL, glutathione, and superoxide dismutase had strong negative impacts, suggesting cardioprotective effects. After controlling for other biomarkers, low-density lipoprotein no longer had independent predictive value. Inflammation and oxidative stress seem to dominate the mechanistic environment of diabetic myocardial damage, with about 47% of the variance in Troponin I explained by the model (Figure 2).

The study showed clear biochemical and physiological differences among the three groups. Both ND-AMI and D-AMI patients had higher cardiac and inflammatory markers compared to the normal group. Diabetic AMI patients, in particular, showed the most significant changes, with much higher levels of Troponin I, CK-MB, CRP, LDL, and MDA, along with much lower activity of antioxidant enzymes (SOD, GSH, and Catalase). Blood pressure readings were also significantly higher in the D-AMI group, indicating a greater prevalence of hypertension. These findings suggest that diabetes worsens heart damage, inflammation, and oxidative stress (Figure 3).

DISCUSSION

The age structure of the Normal, ND-AMI, and D-AMI samples was examined to determine whether age differences might affect biochemical or oxidative measurements. The mean age of the three groups did not differ significantly according to our results. This indicates that the study groups were age-matched, and the observed differences in biochemicals were not due to age but rather to disease status.

We find that the previous clinical research is consistent with ours. According to the FINAMI Study, diabetic and non-diabetic individuals were not significantly different in the mean age, which was the time at which the myocardium was first struck, and this suggests that diabetes changes the severity and outcome of AMI and not its age (Pajunen *et al.*, 2005; Tipparapu *et al.*, 2025). A recent systematic review also found no statistically significant age difference between diabetic and non-diabetic AMI cohorts across multiple global studies (Alawaji *et al.*, 2024). Both studies support our observation that metabolic disturbances, not age, primarily influence the biochemical and oxidative patterns associated with AMI.

We compared the general demographic and clinical characteristics among the Normal, ND-AMI, and D-AMI groups to establish baseline comparability. Our data showed that gender distribution was nearly identical across groups, with male-to-female ratios of 106/94 in Normal, 106/94 in ND-AMI, and 109/91 in D-AMI ($p=0.941$). The mean age was also comparable, with values of 70.52 ± 8.91 years in Normal, 69.45 ± 9.19 years in ND-AMI, and 68.97 ± 9.38 years in D-AMI ($p=0.2258$), indicating that age and gender did not differ significantly. However, systolic and diastolic blood pressures were significantly higher in the AMI groups. The average systolic blood pressure rose from 130.49 ± 15.03 mmHg in normal subjects to 145.55 ± 18.88 mmHg in ND-AMI and then

increased to 149.26 ± 17.11 mmHg in D-AMI. Similarly, diastolic blood pressure rose from 80.10 ± 10.39 mmHg in Normal to 90.90 ± 10.77 mmHg in ND-AMI and 96.15 ± 58.36 mmHg in D-AMI (both $p<0.001$). These findings show that the severity of blood pressure among AMI patients, especially diabetics, is progressively increasing.

Similar trends were described in recent research. Another study found the level of hypertension in myocardial infarction in type 2 diabetic patients to be significantly higher than that of people without diabetes, which substantiates our results that diabetes intensifies the hypertensive impact during myocardial infarction (Yen *et al.*, 2022). Likewise, Karpay *et al.* (2022) reported elevated systolic and diastolic pressures in diabetic AMI subjects, linking this rise to endothelial dysfunction and metabolic stress (Karpay *et al.*, 2022). Together with our past data, our results confirm the fact that both age and gender were well balanced, but hypertension is a determining clinical feature in AMI, especially in diabetic patients.

We compared the parameters of biochemical stress, oxidative stress, and ND-AMI and D-AMI groups. We found increases in Cardiac Troponin I, CK-MB, CRP, Total Cholesterol, Triglycerides, LDL, and MDA, and significant decreases in HDL, SOD, Catalase, and GSH. These tendencies suggest that the level of myocardial damage, inflammation, and oxidative stress rises dramatically in the case of AMI, particularly in diabetic patients.

Another study reported similar levels of Troponin I, CK-MB, and CRP in AMI patients, supporting our evidence of elevated cardiac and inflammatory markers (Karpay *et al.*, 2022; Geeshma *et al.*, 2025). It also observed increased MDA levels and reduced antioxidant enzyme activities in AMI, consistent with our oxidative stress data (Zhao *et al.*, 2023). Likewise, another study found higher LDL and triglyceride levels and lower HDL levels in diabetic AMI cases, consistent with our lipid profile results.

Table 1: Demographic and Clinical Characteristics of the Study Groups.

Parameter	Normal (n=200)	ND-AMI (n=200)	D-AMI (n=200)	p-value
Age, Years				
Mean $\pm$ SD	70.52 $\pm$ 8.91	69.45 $\pm$ 9.19	68.97 $\pm$ 9.38	0.2258
50-59, n	31	40	51	—
60-69, n	72	73	63	—
70-79, n	60	54	54	—
Gender				
Male, n (%)	106 (53.0)	106 (53.0)	109 (54.5)	0.9415
Female, n (%)	94 (47.0)	94 (47.0)	91 (45.5)	
SBP (mmHg)	130.49 $\pm$ 15.03	145.55 $\pm$ 18.88	149.26 $\pm$ 17.11	<0.001
DBP (mmHg)	80.10 $\pm$ 10.39	90.90 $\pm$ 10.77	96.15 $\pm$ 58.36	<0.001
Hemoglobin (g/dL)	13.83 $\pm$ 1.15	13.42 $\pm$ 1.50	13.40 $\pm$ 1.32	0.0088
WBC (million/m ³)	7.18 $\pm$ 1.84	7.28 $\pm$ 1.67	7.41 $\pm$ 1.74	0.313
Platelet Count ($\times 10^3/\mu$ L)	247.85 $\pm$ 44.54	250.53 $\pm$ 47.88	246.67 $\pm$ 51.87	0.809

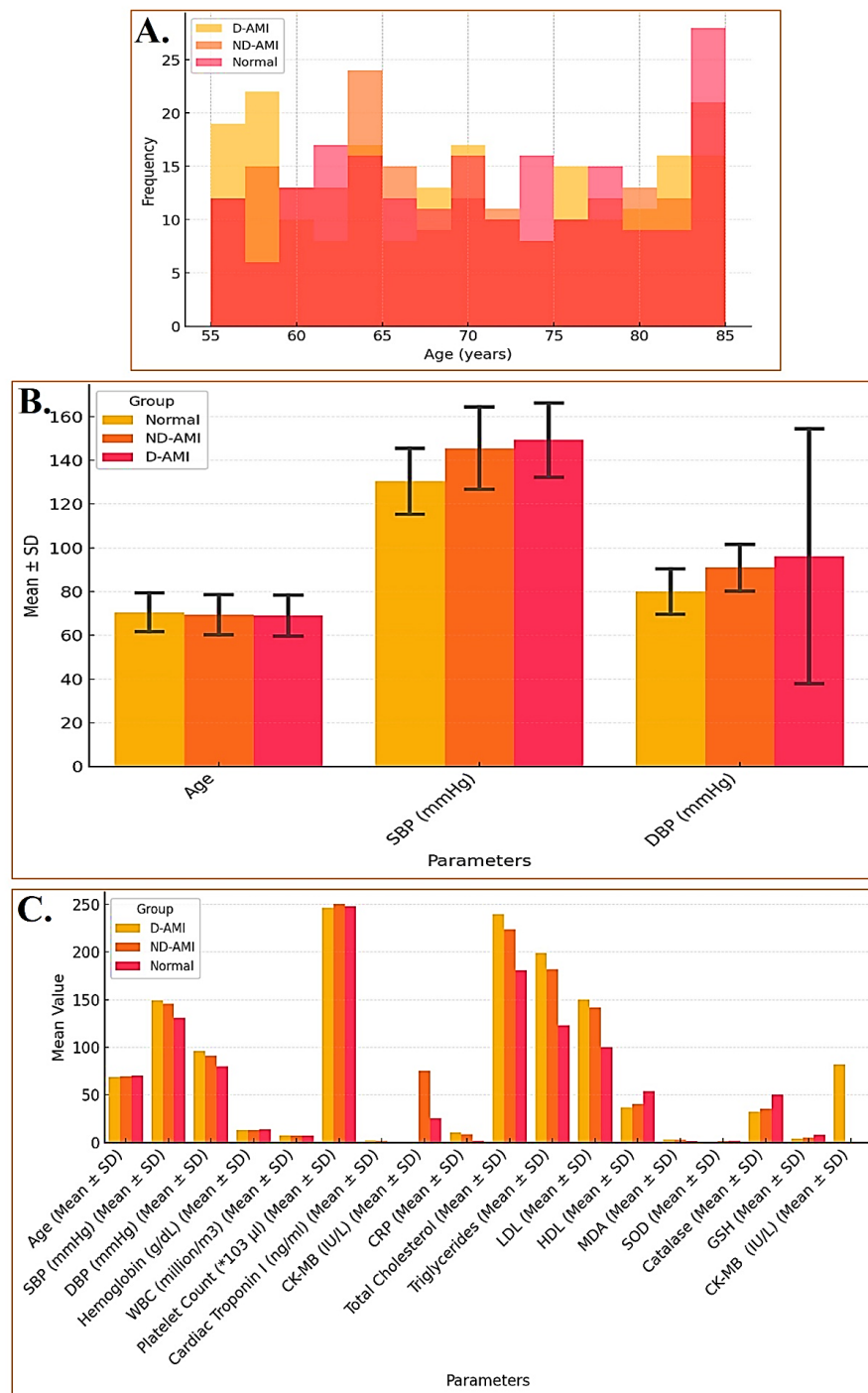


Figure 1: Age Distribution among Normal, ND-AMI, and D-AMI Groups. Demographic Characteristics Across Study Groups (C) Comparison of Biochemical Parameters Across Groups.

To assess the distribution of all biochemical and oxidative parameters, we used the Shapiro-Wilk test to determine which statistical test to use to compare the groups. The majority of variables, such as Troponin I, CRP, Triglycerides, and MDA, were not normally distributed ($p < 0.05$), whereas the Age, SBP, and DBP parameters were normally distributed ($p > 0.05$). These results were used to apply non-parametric (Kruskal-Wallis) tests to skewed data and parametric (ANOVA, t -test) tests to normally distributed data.

We find the same results as in the study, which observed that lipid peroxidation and inflammatory biomarkers are not normally distributed in patients with AMI (Jovanovic *et al.*, 2024). Similarly, observed that oxidative and lipid variables in AMI cohorts exhibited substantial variability due to disease severity and metabolic differences. These studies confirm that non-normality is common in clinical datasets involving AMI and diabetes because of biological heterogeneity and differential oxidative responses.

One-way ANOVA was used to test whether the variables were normally distributed across the three groups, namely Normal, ND-AMI, and D-AMI. The analysis found significant differences ($p < 0.05$) in most cardiac, lipid, and oxidative stress markers. The levels of cardiac Troponin I, CK-MB, and CRP were significantly higher in both ACS groups, with the highest mean values observed in D-AMI, indicating greater myocardial injury and inflammation in diabetic patients. The lipid parameters were no different, with AMI patients having considerably higher Total Cholesterol, Triglycerides, and LDL, and lower HDL compared to the Normal group.

Our results are consistent with reports of higher Troponin I and CK-MB levels in AMI subjects compared with healthy

controls (Karpay *et al.*, 2022). Similarly, Sabharwal *et al.* (2019) documented elevated CRP and dyslipidemia in diabetic AMI cases, highlighting a strong metabolic link between inflammation and lipid disturbance. Moreover, Hussien *et al.* (2023) demonstrated significantly higher LDL and lower HDL in patients with diabetic cardiac disease, further supporting our findings.

The factors that did not follow a normal distribution, such as SBP, DBP, Hemoglobin, Age, and WBC count, were analyzed using the Kruskal-Wallis's test. The findings showed statistically significant differences ($p < 0.001$) in SBP, DBP, and Haemoglobin among the three groups, but no significant differences in Age and WBC. The mean ranks of systolic and diastolic blood pressure were the highest in the D-AMI group and then in ND-AMI and normal,

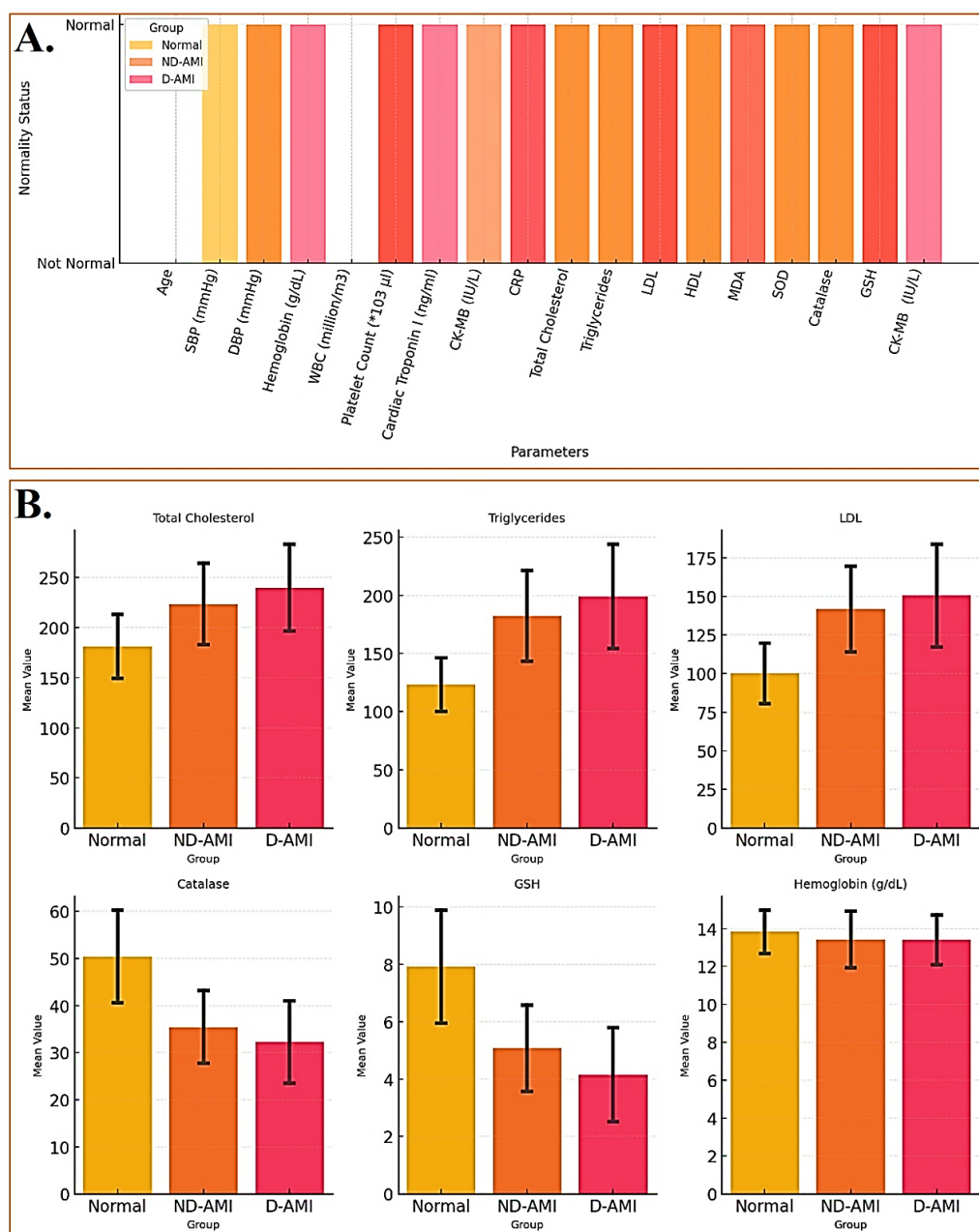


Figure 2: (A) Normality Status Across Groups. (B) Comparison of Significant Biochemical Parameters Across Groups.

Table 2: Biochemical, Lipid, and Oxidative Stress Parameters among Study Groups.

Parameter	Normal (n=200)	ND-AMI (n=200)	D-AMI (n=200)
Cardiac Troponin I (ng/mL)	0.01±0.01	1.74±1.01	2.14±0.91
CK-MB (IU/L)	25.16±5.08	74.97±28.98	81.88±30.38
CRP	1.92±0.99	8.60±4.29	10.57±4.40
Total Cholesterol	181.21±31.81	223.42±40.73	239.93±43.49
Triglycerides	123.08±23.00	182.17±39.02	199.03±44.93
LDL	99.99±19.64	141.78±27.85	150.44±33.44
HDL	54.23±9.95	40.78±8.18	36.98±9.61
MDA	1.48±0.32	2.84±0.88	3.18±0.94
SOD	1.79±0.41	1.18±0.30	0.98±0.32
Catalase	50.41±9.88	35.43±7.70	32.28±8.74
GSH	7.91±1.98	5.08±1.51	4.15±1.65

Table 3: Intergroup Statistical Comparisons and Normality Assessment.

Parameter	Normality Status	ANOVA F (p)	K-W H (p)	t-test D-AMI vs ND-AMI (p)	Significance
Age	Not Normal	—	3.091 (0.213)	—	NS
SBP	Not Normal	—	122.123 (<0.001)	—	Sig
DBP	Not Normal	—	130.574 (<0.001)	—	Sig
Hemoglobin	Not Normal	6.645 (0.001)	9.467 (0.009)	—	Sig
WBC	Not Normal	0.942 (0.391)	2.323 (0.313)	—	NS
Platelet	Normal	—	0.424 (0.809)	—	NS
Troponin I	Not Normal	418.449 (<0.001)	412.719 (<0.001)	4.129 (<0.001)	Sig
CK-MB	Not Normal	—	—	—	NR
CRP	Not Normal	317.622 (<0.001)	337.415 (<0.001)	4.541 (<0.001)	Sig
Total Cholesterol	Not Normal	120.605 (<0.001)	174.111 (<0.001)	—	Sig
Triglycerides	Not Normal	234.473 (<0.001)	286.042 (<0.001)	—	Sig
LDL	Normal	191.573 (<0.001)	251.080 (<0.001)	2.813 (0.005)	Sig
HDL	Not Normal	190.835 (<0.001)	230.221 (<0.001)	-4.256 (<0.001)	Sig
MDA	Not Normal	275.713 (<0.001)	335.941 (<0.001)	3.732 (<0.001)	Sig
SOD	Not Normal	295.470 (<0.001)	287.035 (<0.001)	-6.417 (<0.001)	Sig
Catalase	Not Normal	241.174 (<0.001)	261.443 (<0.001)	—	Sig
GSH	Normal	259.783 (<0.001)	276.773 (<0.001)	—	Sig

thus far confirming the severity of hypertension in diabetic AMI patients. There was also variation in hemoglobin levels, indicating mild anemia in AMI cases compared with normal individuals.

Our findings are consistent with those of those who discovered that diabetic patients with hypertension had much higher blood pressure and cardiovascular load during myocardial infarction. Similarly, it has been demonstrated that AMI patients tend to have reduced hemoglobin and elevated blood pressure due to oxidative damage and vascular inflammation. These findings strengthen our conclusion that non-normally distributed clinical parameters, such as BP and Hemoglobin, show clear pathological escalation in diabetic AMI subjects (Karpay *et al.*, 2022).

We used an independent t-test to compare the biochemical parameters between D-AMI and ND-AMI groups. The findings showed statistically significant differences across most parameters ($p < 0.05$). Cardiac Troponin I, CRP, LDL, and MDA were significantly greater in D-AMI, but HDL and SOD were significantly less. These results show that diabetic AMI patients experience more heart damage, inflammation, lipid imbalance, and oxidative stress than non-diabetic AMI patients.

Several recent studies back up our findings. found that LDL and triglyceride levels were significantly higher, while HDL levels were significantly lower in patients with diabetic AMI. This explains how diabetes worsens lipid metabolism. (Hussien *et al.*, 2023). Similarly, elevated Troponin I and CRP levels

Table 4: Correlation and Multiple Linear Regression Analysis of Cardiac Troponin I.

Parameter	Pearson Correlation r (p)	Regression B (SE)	t -value	p -value	Interpretation
CK-MB	0.556 (<0.001)	—	—	—	Strong positive correlation
CRP	0.539 (<0.001)	0.0489 (0.0090)	5.43	<0.001	Positive predictor
LDL	0.446 (<0.001)	0.002 (0.0013)	1.582	0.114	NS predictor
MDA	0.529 (<0.001)	0.2178 (0.0436)	4.995	<0.001	Positive predictor
SOD	-0.531 (<0.001)	-0.4576 (0.0958)	-4.775	<0.001	Negative predictor
GSH	—	-0.0909 (0.0196)	-4.646	<0.001	Negative predictor
HDL	—	-0.0096 (0.0038)	-2.563	0.011	Negative predictor
Constant	—	1.6901 (0.3449)	4.9	<0.001	—
Model Fit	F=87.76, p <0.001, R^2 =0.470, Adjusted R^2 =0.460				

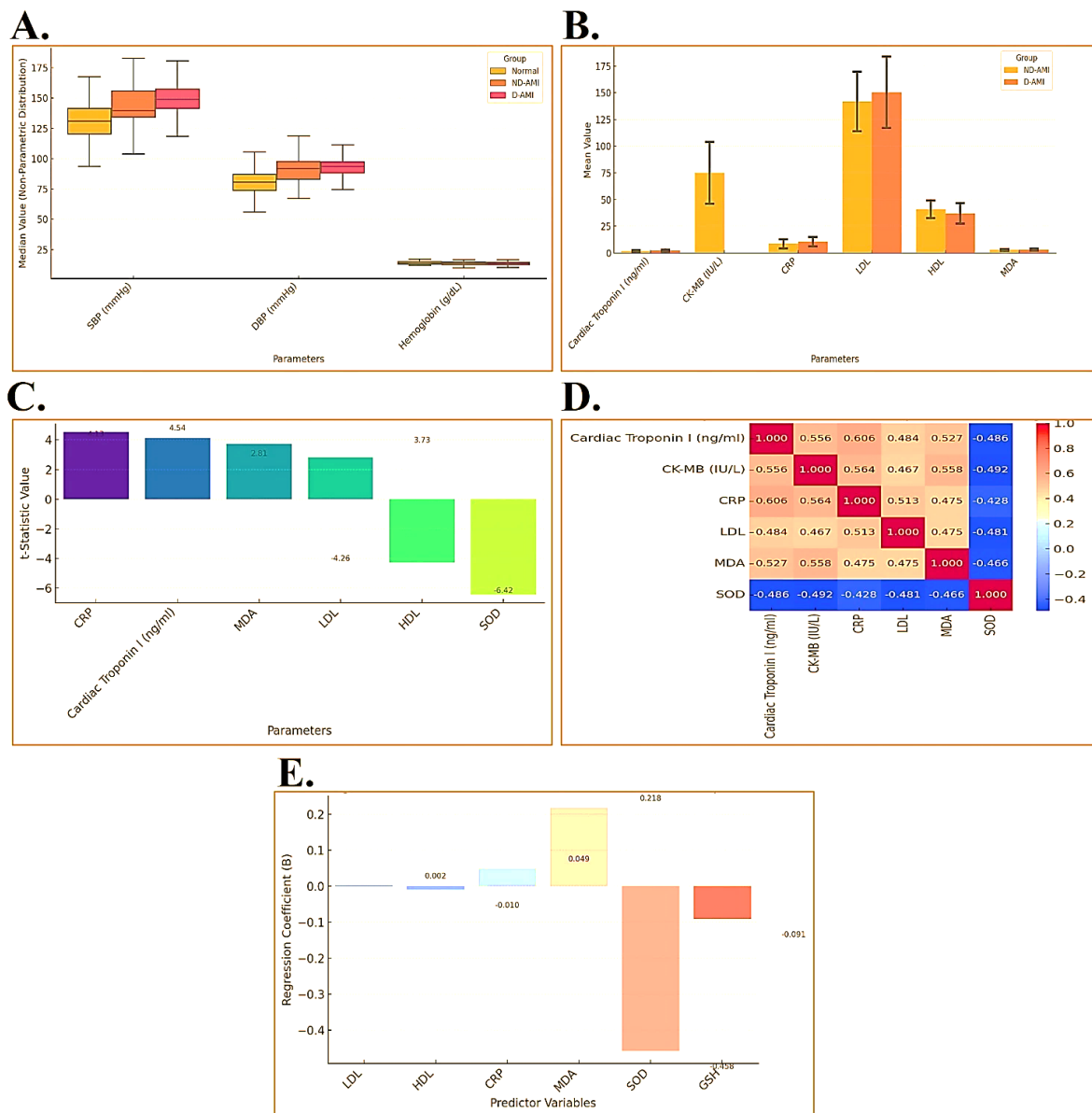


Figure 3: (A) Significant Non-Parametric Parameters Across Groups. (B) Comparison of Biochemical Parameters Between D-AMI and ND-AMI. (C) Statistic Values for Biochemical Parameters (D-AMI vs ND-AMI). (D) Correlation Heatmap Between Cardiac Troponin I and Key Biochemical Markers. (E) Regression Coefficients of Predictors for Cardiac Troponin I.

were reported in AMI subjects, confirming enhanced cardiac injury and inflammation. They also found that oxidative stress markers, such as MDA, were elevated in diabetic AMI cases. In contrast, antioxidant enzymes, such as SOD, were lower than in non-diabetics.

We conducted a correlation analysis to look at the relationship between Cardiac Troponin I and key biochemical parameters: CK-MB, CRP, LDL, MDA, and SOD. Our results showed strong, statistically significant correlations ($p < 0.001$). Troponin I showed positive correlations with CK-MB ($r = 0.556$), CRP ($r = 0.539$), LDL ($r = 0.446$), and MDA ($r = 0.529$), while a significant negative correlation was found with SOD ($r = -0.531$). These results indicate that inflammation, dyslipidemia, and oxidative stress are directly correlated with the extent of myocardial injury and the activities of antioxidant enzymes, respectively.

Similar relationships are reported in the recent literature. A significant positive correlation between Troponin I and CRP has also been observed in ACS patients by another study, suggesting concomitant elevation of cardiac infarction and systemic inflammation.^{23, 29} Similarly, Hussien and Abdinibi (2023) observed strong positive correlations between Troponin I and LDL, linking lipid abnormalities to myocardial damage in subjects with diabetic AMI. Jovanović *et al.* (2024) also reported that Troponin I positively correlates with MDA and inversely correlates with antioxidant enzymes such as SOD, reinforcing our results.

Collectively, these results indicate that inflammation, lipid imbalance, and oxidative stress are strongly associated with myocardial injury in AMI, and the extent of the rise in Troponin I indicates the cumulative biochemical stress on the myocardium.

A multiple linear regression was conducted to identify the biochemical variables that affect Cardiac Troponin I values. LDL, HDL, CRP, MDA, SOD, and GSH were used as independent variables. The model was significant ($F = 87.76$, $p < 0.001$), with an R^2 of 0.470 and an adjusted R^2 of 0.460, explaining nearly 47% of the variation in Troponin I.

CRP ($B = 0.0489$, $p < 0.001$) and MDA ($B = 0.2178$, $p < 0.001$) were strong positive predictors, while SOD ($B = -0.4576$, $p < 0.001$), GSH ($B = -0.0909$, $p < 0.001$), and HDL ($B = -0.0096$, $p = 0.011$) showed negative associations, suggesting that inflammation and oxidative stress elevate Troponin I, whereas antioxidants and HDL exert a protective effect. LDL ($p = 0.114$) was not significant.

Our findings align with those of others who reported that CRP and MDA strongly correlate with myocardial injury, while antioxidant enzymes mitigate damage (Karpay *et al.* 2022; Hussien 2023). Also found that low HDL aggravates cardiac injury in diabetic AMI (Polyakova *et al.*, 2020; Srivastava *et al.*, 2018). Together, these studies confirm that inflammation and oxidative stress are key predictors of Troponin I elevation.

CONCLUSION

This paper has shown that diabetes is a serious aggravator of myocardial injury, lipid imbalance, and oxidative stress among patients with acute myocardial infarction. The AMI subjects with and without diabetes exhibited high cardiac biomarkers and inflammatory indices, although the levels were high in diabetic subjects. High levels of Troponin I, CK-MB, CRP, LDL, and MDA, and decreased HDL, SOD, and GSH concentrations indicate that diabetes amplifies the effects of oxidative and inflammatory stressors on cardiac tissue.

We have highlighted in our findings that oxidative stress and inflammation play a significant role in maintaining the intensity of myocardial infarction, particularly in diabetic subjects. The use of oxidative stress and lipid parameters, along with conventional cardiac parameters, would enable the timely diagnosis of high-risk patients and the prescription of more effective interventions. The work supports the use of drug therapies involving antioxidants and lipid management in the management of diabetic AMI to enhance the prognosis and lower the complications.

To conclude, diabetes is an important condition that often occurs alongside myocardial infarction. Oxidative stress and metabolic imbalance are critical issues to tackle in order to improve heart outcomes for these patients.

ACKNOWLEDGEMENT

The authors extend their appreciation to the Deanship of Research and Graduate Studies at King Khalid University, KSA, for its support.

ABBREVIATIONS

DM: Diabetes mellitus; **AMI:** Acute Myocardial Infarction; **MDA:** Malondialdehyde; **SOD:** Superoxide Dismutase; **GSH:** Reduced Glutathione; **LDL:** Low-Density Lipoprotein; **HDL:** High-Density Lipoprotein; **CK-MB:** Creatine Kinase Myocardial Band; **CRP:** C-Reactive Protein; **ANOVA:** Analysis of Variance; **CVDs:** Cardiovascular diseases; **ADA:** American Diabetes Association; **TBARS:** Thiobarbituric acid reactive substances; **CAT:** Catalase; **Hb:** Hemoglobin; **ND-AMI:** Non-Diabetic Acute Myocardial Infarction; **D-AMI:** Diabetic Acute Myocardial Infarction; **ACS:** Acute Coronary Syndrome; **FPG:** Fasting Plasma Glucose; **HbA_{1c}:** Glycated Hemoglobin; **EDTA:** Ethylenediaminetetraacetic Acid; **SBP:** Systolic Blood Pressure; **DBP:** Diastolic Blood Pressure; **WBC:** White Blood Cell; **UV:** Ultraviolet; **SPSS:** Statistical Package for the Social Sciences.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

FUNDING

The authors extend their appreciation to the Deanship of Research and Graduate Studies at King Khalid University for funding this work through the Large Research Project under grant number RGP2/361/46.

ETHICAL APPROVAL

This work follows ethical principles of honesty, respect, and responsibility. We have ethical approval with REC-43/02/017.

GENERATIVE AI STATEMENT

The author declares that generative AI (Artificial Intelligence) tools, including Grammarly and QuillBot, were used to enhance the language and clarity of this work. The author takes full responsibility for the accuracy and integrity of the content.

REFERENCES

- Alawaji, R., Musslem, M., Alshalahi, E., Alanzan, A., Sufyani, A., Alhati, M., Almutairi, A., Alqaffas, M., Alattas, B., & Alselmi, A. (2024). A systematic review and meta-analysis of the effect of hyperglycemia on admission for acute myocardial infarction in diabetic and non-diabetic patients. *Diabetology and Metabolic Syndrome*, 16(1), Article 224. <https://doi.org/10.1186/s13098-024-01459-w>
- Ali, F., Naqvi, S. A. S., Bismillah, M., & Wajid, N. (2016). Comparative analysis of biochemical parameters in diabetic and non-diabetic acute myocardial infarction patients. *Indian Heart Journal*, 68(3), 325–331. <https://doi.org/10.1016/j.ihj.2015.09.026>
- Cecerska-Heryć, E., Engwert, W., Michałow, J., Marciniak, J., Birger, R., Serwin, N., Heryć, R., Polikowska, A., Goszka, M., Wojciuk, B., Wiśniewska, M., & Dołęgowska, B. (2025). Oxidative stress markers and inflammation in type 1 and 2 diabetes are affected by BMI, treatment type, and complications. *Scientific Reports*, 15(1), Article 23605. <https://doi.org/10.1038/s41598-025-05818-z>
- Ceriello, A., & Testa, R. (2009). Antioxidant anti-inflammatory treatment in type 2 diabetes. *Diabetes Care*, 32(Suppl. 2), S232–S236. <https://doi.org/10.2337/dc09-S316>
- Cui, J., & Zhao, N. (2012). Consideration in selection of the control group in diagnostic tests. *International Journal of Biological Sciences*, 8(10), 1418–1419. <https://doi.org/10.7150/ijbs.5459>
- Daubert, M. A., & Jeremias, A. (2010). The utility of troponin measurement to detect myocardial infarction: Review of the current findings. *Vascular Health and Risk Management*, 6, 691–699. <https://doi.org/10.2147/vhrm.s5306>
- De Leon, J. A. D., & Borges, C. R. (2020). Evaluation of oxidative stress in biological samples using the thiobarbituric acid reactive substances assay. *Journal of Visual. Experiments*, 159, Article 61122.
- Geeshma, K. (2025). C-reactive protein as a predictor of infarct size in acute myocardial infarction. *European Journal of Cardiovascular Medicine*, 15, 458–462.
- Hussien, F., & Abdinibi, K. (2023). Evaluation of lipid parameters in diabetic and non-diabetic acute myocardial infarction patients in Al-Bayda. *AlQalam J. Med Appl. Sci.*, 327–330.
- Jovanović, J., Pantić, K., Milojević, D., Milojevic, A., Canovic, P., Zaric, M., Zdravkovic, N., Andjelkovic, M., Ravic, M., & Miloradovic, V. (2024). Oxidative stress in acute myocardial infarction patients. *Experimental and Applied Biomedical Research*, 25(1), 51–56. <https://doi.org/10.2478/sjecr-2021-0008>
- Karpay, S., Sarada, C. V., Kondu, D., Pavuluri, P., Gadepalli, R., & Naresh, B. (2022). Evaluation of biochemical parameters in acute myocardial infarction and angina patients. *Journal of Ideas in Health*, 5(2), 664–668. <https://doi.org/10.47108/jidhealth.Vol5.Iss2.213>
- Kumar, A. S., & Sinha, N. (2020). *Cardiovascular disease in India: A 360-degree overview* (pp. 1–3). Elsevier.
- Mohan, V., Venkatraman, J. V., & Pradeepa, R. (2010). Epidemiology of cardiovascular disease in type 2 diabetes: The Indian scenario. *Journal of Diabetes Science and Technology*, 4(1), 158–170. <https://doi.org/10.1177/193229681000400121>
- Narres, M., Kvitkina, T., Claessen, H., Ubach, E., Wolff, G., Metzendorf, M.-I., Richter, B., & Icks, A. (2022). Incidence of myocardial infarction in people with diabetes compared to those without diabetes: A systematic review protocol. *Systematic Reviews*, 11(1), Article 89. <https://doi.org/10.1186/s13643-022-01962-z>
- Nesbitt, K., Champion, S., Pearson, V., Gebremichael, L. G., Dafny, H., Ramos, J. S., Suebkinorn, O., Pinero de Plaza, M. A., Gulyani, A., Du, H., Clark, R. A., Beleigoli, A., & NHMRC Country Heart Attack Prevention (CHAP) Partnership Project Team. (2025). The effectiveness of interactive cardiac rehabilitation web applications versus usual care on programme completion in patients with cardiovascular disease: A systematic review and meta-analysis of randomised controlled trials. *Journal of Telemedicine and Telecare*, 31(4), 475–486. <https://doi.org/10.1177/1357633X231201874>
- Ormazabal, V., Nair, S., Elfeky, O., Aguayo, C., Salomon, C., & Zuñiga, F. A. (2018). Association between insulin resistance and the development of cardiovascular disease. *Cardiovascular Diabetology*, 17(1), Article 122. <https://doi.org/10.1186/s12933-018-0762-4>
- Pajunen, P., Koukkunen, H., Ketonen, M., Jerkkola, T., Immonen-Räihä, P., Kärjä-Koskenkari, P., Kuulasmaa, K., Palomäki, P., Mustonen, J., Lehtonen, A., Arstila, M., Vuorenmaa, T., Lehto, S., Miettinen, H., Torppa, J., Tuomilehto, J., Kesäniemi, Y. A., Pyörälä, K., & Salomaa, V. (2005). Myocardial infarction in diabetic and non-diabetic persons with and without prior myocardial infarction: The FINAMI Study. *Diabetologia*, 48(12), 2519–2524. <https://doi.org/10.1007/s00125-005-0019-0>
- Polyakova, E. A., & Mikhaylov, E. N. (2020). The prognostic role of high-sensitivity C-reactive protein in patients with acute myocardial infarction. *Journal of Geriatric Cardiology*, 17(7), 379–383. <https://doi.org/10.11909/j.issn.1671-5411.2020.07.007>
- Puri, R., Mehta, V., Duell, P. B., Wangnoo, S. K., Rastogi, A., Mohan, V., Zargar, A. H., Kalra, S., Sahoo, A. K., Iyengar, S. S., Yusuf, J., Mukhopadhyay, S., Singla, M. K., Shaikh, A., Kohli, S., Mathur, S., Jain, S., Narasingan, S. N., Gupta, V., (2023). Management of diabetic dyslipidemia in Indians: Expert consensus statement from the Lipid Association of India. *Journal of Clinical Lipidology*, 17(2), e1–e14. <https://doi.org/10.1016/j.jacl.2022.11.002>
- Sabharwal, R., Singh, L., & Bhatia, A. (2019). Dyslipidemia in diabetic patients with acute myocardial infarction. *JK Science*, 21, 160–164.
- Sabitha, S., Hegde, S. V., Agarwal, S. V. et al. (2024). Biomarkers of oxidative stress and their clinical relevance in type 2 diabetes mellitus patients. *Cureus*, 16.
- Smith, J. W., Marcus, F. I., Serokman, R., & Group, M. P. R. (1984). Prognosis of patients with diabetes mellitus after acute myocardial infarction. *The American Journal of Cardiology*, 54(7), 718–721. [https://doi.org/10.1016/S0002-9149\(84\)80196-4](https://doi.org/10.1016/S0002-9149(84)80196-4)
- Srivastava, R. A. K. (2018). Dysfunctional HDL in diabetes mellitus and its role in the pathogenesis of cardiovascular disease. *Molecular and Cellular Biochemistry*, 440(1–2), 167–187. <https://doi.org/10.1007/s11010-017-3165-z>
- Stampouloulou, P. K., Anastasiou, A., Bletsas, E., Lygkoni, S., Chouzouri, F., Xenou, M., Katsarou, O., Theofilis, P., Zisimos, K., Tousoulis, D., Vavuranakis, M., Siasos, G., & Oikonomou, E. (2023). Diabetes mellitus in acute coronary syndrome. *Life*, 13(11), Article 2226. <https://doi.org/10.3390/life13112226>
- Tidy, C., & Jarvis, S. (2019). Cardiac enzymes and markers for myocardial infarction. Professional articles. *Cardiovascular Diseases*.
- Tipparapu, B. (2025). Prevalence and impact of diabetes mellitus in patients with acute myocardial infarction. *CME Journal Geriatric Medicine*, 17, 109–114.
- Wega, A. B., Wabalo, E. K., Edae, C. K., & Awgichew, G. B. (2021). Cardiac troponin-I status of type-2 diabetic patients. *Research Reports in Clinical Cardiology*, 1–7.
- Yao, Y., Chen, S., & Li, H. (2021). An improved system to evaluate superoxide-scavenging effects of bioflavonoids. *ChemistryOpen*, 10(4), 503–514. <https://doi.org/10.1002/op.en.202100013>
- Yen, F.-S., Wei, J. C.-C., Chiu, L.-T., Hsu, C.-C., & Hwu, C.-M. (2022). Diabetes, hypertension, and cardiovascular disease development. *Journal of Translational Medicine*, 20(1), Article 9. <https://doi.org/10.1186/s12967-021-03217-2>
- Zhao, C., Liu, T., Wei, H., & Li, J. (2023). Serum oxidative stress factors predict myocardial ischemia reperfusion injury after percutaneous coronary intervention in patients with acute myocardial infarction and type 2 diabetes mellitus. *Advances in Interventional Cardiology*, 19(4), 333–342. <https://doi.org/10.5114/aic.2023.133475>

Cite this article: Ahmad K, Mandal RK, Wahid M, Vincent B, Suwaydi MA, Prabhudas S, et al. Assessment of Hematological and Biochemical Markers in Saudi Patients with Diabetes and Non-Diabetic Acute Myocardial Infarction. *Int. J. Pharm. Investigation*. 2027;17(1):471–80.