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Original Research Article

ANALYSIS OF BIOCHEMICAL MARKERS IN DIABETIC AND NON-DIABETIC ACUTE MYOCARDIAL INFARCTION PATIENTS: A COMPARATIVE STUDY

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Abstract

Background: Diabetes is a metabolic condition that causes more free radicals to be produced, which causes oxidative stress. The purpose of this study was to compare the activity of cardiac and antioxidant enzymes in acute myocardial infarction (AMI) patients with and without diabetes. **Materials and Methods:** This cross-sectional study was conducted in the Department of Biochemistry, Index Institute of Medical Sciences and Hospital, Indore, Madhya Pradesh. There were 300 participants in this study, of whom 100 had normal blood glucose levels and normal ECGs (Normal, N), 100 had normal blood glucose levels and an AMI (Non-diabetic and AMI, N-AMI), and 100 had diabetes and an AMI (Diabetic and AMI, D-AMI). **Result:** Compared to N-AMI patients, D-AMI persons exhibited higher levels of TC, TG, LDL, and lower levels of HDL. According to a study, myocardial infarction patients with diabetes mellitus (DM) had significantly higher levels of cardiac markers such Troponin I, CPK, CK-MB, AST, LDH, and CRP than myocardial infarction patients without DM. Patients with D-AMI had lower levels of antioxidant SOD and GSH activity than those with N-AMI. MDA and CAT levels, however, were greater in D-AMI than in N-AMI controls. **Conclusion:** According to a study, D-AMI patients had lower antioxidant levels than N-AMI patients and have increased cardiac markers.

INTRODUCTION

Diabetes mellitus raises the risk of CVD-induced mortality and the incidence of cardiovascular illnesses in diabetic persons compared to non-diabetic subjects.[1] The majority of deaths worldwide were caused by myocardial infarction and heart failure, which were caused by coronary artery disease.[2] The development of reactive oxygen species, myocardial ischemia leading to myocardial necrosis, and coronary artery obstruction are all factors in acute myocardial infarction.[3] According to earlier research, hyperglycemia increases the risk of heart problems caused by reactive oxygen species by interacting with lipids, proteins, and DNA,[4] myocardial antioxidants can reverse this oxidative damage.[5] Numerous studies have shown that antioxidant function is reduced in diabetic subjects,[6] which may contribute to the pathogenesis of acute myocardial infarction caused by oxidative stress.[7] It is generally known that risk factors for the development of acute myocardial infarction include smoking, diabetes, dyslipidemia, hypertension, family history, obesity, and hypertension.[8] The aim of the study was to evaluate whether diabetic and non-diabetic acute myocardial infarction patients had cardiac damage brought on by oxidative stress. This study emphasises that, along with other signs like the ECG and cardiac biomarkers, an imbalance in antioxidants may be a crucial sign of diabetes-related myocardial damage.

MATERIALS AND METHODS

This cross-sectional study was conducted in the Department of Biochemistry, Index Institute of Medical Sciences and Hospital, Indore, Madhya Pradesh. This present study included 300 subjects, and categories in to three groups: Group-1: 100 subjects were with normal blood glucose level and with normal ECG (Normal, N), Group-2: 100 subjects were with normal blood glucose level and AMI (Non-diabetic and AMI, N-AMI) and Group-3: 100 subjects were with diabetes and AMI (Diabetic

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and AMI, D-AMI). Analysing the level of glycated haemoglobin (HbA1c>6.5%) allowed for the diagnosis of diabetes. After receiving written permission to participate from their carers, diagnosed cases of both diabetic and non-diabetic AMI patients were included in the study. Patients' biographical information, a thorough medical history, blood pressure readings, and electrocardiograms (ECG) were duly entered into questionnaires.

Inclusion and Exclusion Criteria

There were both male and female subjects having a history of AMI. Chest discomfort history, ECG abnormalities, and increased cardiac enzymes were used to diagnose AMI. This study covered both diabetic and non-diabetic AMI patients. The control subjects were chosen because they had a normal ECG and were not hypertensive. The study did not include participants with histories of smoking, obesity, or any other ailment.

Data Collection

All individuals had their cubital veins used to collect blood samples, which were then separated into serum and immediately transported in an icebox from the hospital to the lab. At 4oC, blood samples were centrifuged for 10 minutes at 2100 x g. In order to store the serum for examination at -20oC, it was aspirated.

Evaluation of Cardiovascular Parameters

Using commercial test kits, the concentrations of TC, TG, and HDL in the blood were determined spectrophotometrically. Using the Fridewald formula, LDL was determined.[9]

Analysis of Cardiac Markers

Using commercially available kits, the levels of several cardiac enzymes, including troponin-I(TnI), CPK, CK-MB, LDH, AST, and CRP, were measured.

Estimation of Oxidative Stress

Using commercial kits, the serum concentrations of MDA, CAT, SOD, and GSH were examined to determine the level of oxidative stress.

Statistical Analysis

Software called Epi-info was used to do the statistical analysis. Three sets of quantitative variables were initially compared using one-way ANOVA, and the locations of the differences between the groups were then confirmed using the Bonferroni post hoc test. The means and standard deviations (SD) for all the data were displayed. Statistics were judged significant at P <0.05.

RESULTS

This study covered 300 patients in total. Three sets of study participants were used: group A (Control) consisted of healthy persons, group B (N-AMI) of AMI patients, and group C (D-AMI) of patients with both diabetes mellitus and AMI. In all three groups, the distribution of participants by age is shown in [Table 1].

In all groups, the average age and sex were comparable. In [Table2], D-AMI and N-AMI had higher systolic blood pressure (SBP) and diastolic blood pressure (DBP) than the normal group. When compared to the N-AMI and normal groups in [Table 3], the fasting blood glucose, postprandial blood glucose, and HbA1c values in the D-AMI group were all considerably higher (P 0.001).

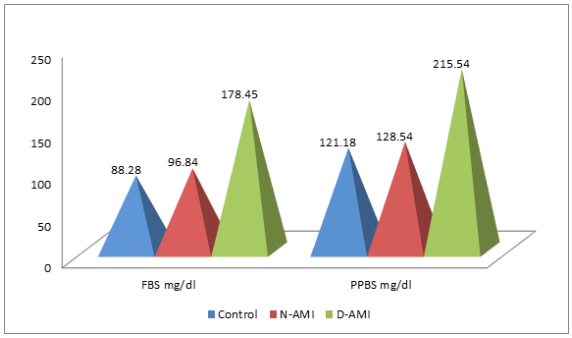

Figure 1: Shows the level of fasting and postprandial blood sugar in different groups.

The TC (274.65±21.24), TG (306.32±54.36), and LDL (294.32±24.36) levels in the D-AMI group were significantly higher than those in the N-AMI group for the corresponding values of TC (238.34±16.54), TG (232.21±20.14), and LDL (249.32±32.14). While N-AMI group had significantly higher levels of HDL (31.54±5.32) and N group had 41.21±7.38 correspondingly, D-AMI group had significantly lower levels (27.65±4.21). [Table 4]

Table 1: Distribution of subjects according to age group.

Age group	Control	N-AMI	D-AMI
35-50	32 (32.0%)	37 (37.0%)	36 (36.0%)
50-65	68 (68.0%)	63 (63.0%)	64 (64.0%)
Total	100 (100.0%)	100(100.0%)	100 (100.0%)

Table 2: Shows the general demographic characteristics.

Variables	Control	N-AMI	D-AMI	P value
Male : Female	64:36	67:33	62:38	0.25
Age in years	50.24±14.21	51.26±15.34	51.76±15.36	0.18
SPB in mm of Hg	122.14±26.04	134.23±28.54	150.32±31.25	0.01
DBP in mm of Hg	80.54±16.21	86.32±17.54	98.21±18.26	0.01

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Table3: Shows the comparison of sugar profile.

Sugar profile	Control	N-AMI	D-AMI	P value
FBS (mg/dl)	88.28± 15.25	96.84±25.34	178.45±32.32	0.01
PPBS (mg/dl)	121.18±25.06	128.54±27.21	215.54±45.26	0.01
HbA1c (%)	4.41±1.32	4.54±1.56	7.39±2.54	0.001

Table4: Shows the comparison of lipid profile.

Lipid profile	Control	N-AMI	D-AMI	P value
T. cholesterol (mg/dl)	184.45±14.21	238.34±16.54	274.65±21.24	0.001
Triglycerides (mg/dl)	152.21±15.32	232.21±20.14	306.32±54.36	0.001
HDL-c (mg/dl)	41.21±7.38	31.54±5.32	27.65±4.21	0.03
LDL-c (mg/dl)	138.24±14.65	249.32±32.14	294.32±24.36	0.001

Table 5: Shows the comparison of cardiac profile.

Cardiac profile	Control	N-AMI	D-AMI	P value
Troponin I (ng/ml)	0.96±0.36	1.86±0.21	3.35±0.38	0.01
CPK IU/L	131.21±16.35	342.54±26.54	1078.04±45.68	0.01
CPK-MB (IU/L)	43.21±8.32	109.26±12.56	244.28±18.24	0.01
LDH (IU/L)	112.36±15.42	614.32±48.36	1008.2±54.62	0.01
CRP (mg/L)	1.26±0.52	4.58±1.24	7.94±2.54	0.01
SGOT (IU/L)	22.42±6.20	64.34±9.21	101.6.2±23.52	0.01

Table 6: Shows the comparison of oxidative stress.

Oxidative stress variables	Control	N-AMI	D-AMI	P value
MDA (nmol/ml)	0.05±0.002	0.06±0.0021	0.9±0.013	0.01
CAT (MU/L)	1.05±0.035	0.85±0.008	0.92±0.041	0.01
SOD (units/ml)	0.087±0.004	0.061±0.01	0.055±0.02	0.01
GSH (µM)	0.11±0.004	0.06±0.002	0.05±0.002	0.01

DISCUSSION

Due to increased ROS production,[10] activation of pro-inflammatory responses,[11] decreased antioxidant activity,[12] increased lipid peroxidation, and myocardial ischemia,[10] AMI is brought on. All of these incidents cause plaque to become activated, coronary blockages to form, and ultimately heart attacks. AMI affects a significant portion of the population.[13] Many risk factors, including diabetes, dyslipidemia, hypertension, smoking, obesity, and advancing age, are connected to the emergence of AMI.[14] According to Bartels et al.'s research,[15] those with diabetes have a higher chance of developing CVD than people without the condition. The current investigation discovered the impact of diabetes, hypertension, and dyslipidemia in D-AMI patients. It has been discovered that type 2 diabetes alters the way lipids and lipoproteins are used and causes atherogenic dyslipidemia.[16] Our findings indicate that D-AMI patients had significantly lower levels of HDL while significantly higher levels of TC, TG, and LDL. This shows that atherogenic dyslipidemia plays a significant role in the development of AMI in diabetic people. Inducing both local and systemic inflammatory reactions, atherogenic dyslipidemia favours the oxidative alteration of proteins along with lipids, especially LDL.[17] By detecting the CRP level, cardiac tissue harm brought on by these inflammatory reactions can be identified. In fact, CRP is a marker of systemic inflammation and provides predictive data for cardiovascular events like CAD and atherosclerosis.[18] In this study, D-AMI patients' CRP levels were higher than those of N-AMI patients. By assessing the myocardial tissue-specific protein Trop I, which is involved in cardiac contractility, heart contractility is assessed. Previous research suggested that the myocardial damage marker Trop I is extremely sensitive and specific, making it a useful diagnostic tool for AMI.[19] This study demonstrated that D-AMI patients had considerably higher levels of Trop I than N-AMI patients, indicating that diabetic persons have a higher rate of cardiac muscle cell death. CPK and CPK-MB are two crucial markers of myocardial necrosis,[20] and this study's D-AMI group had significantly elevated levels of both markers. Additionally, we discovered a statistically significant difference in LDH and AST values between the D-AMI and N-AMI groups, the two indicators that have previously been recommended for the diagnosis of infarct. It has been suggested that oxidative stress brought on by hyperlipidemia and hyperglycemia contributes to the development of AMI.[2] The disruption between free radicals and antioxidant defence mechanisms is brought on by oxidative stress. Superoxide radicals are broken down into oxygen or hydrogen peroxide by SOD, one of the vital defence enzymes.[21] Glutathione peroxidase, often known as CAT, catalyses the conversion of hydrogen peroxide into water; GPX, on the other hand, depends on reduced glutathione for this reaction.[22] Additionally, GSH prevents lipid peroxidation. Previous studies indicated that lipid peroxidation increased in AMI patients,[23] and that this enhanced lipoperoxidation is a result of the oxidative stress caused by hyperglycemia.

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CONCLUSION

The research we performed demonstrates the importance of atherosclerosis and its related consequences in D-AMI patients, including dyslipidemia and inflammation. In conclusion, our research shows that D-AMI patients had significantly higher levels of non-traditional cardiac markers like CRP compared to NAMI patients as well as standard cardiac markers including CPK, CK-MB, LDH, and AST. The study demonstrates that while levels of the antioxidants CAT, SOD, and GSH are decreased in D-AMI patients, the oxidative stress measure MDA significantly increases. Clinicians treating MI patients with DM will find these outcomes useful.

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