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Assessment of Cardiac Enzyme Level and Lipid Peroxidation in Patients with Actute Myocardial Infarction at the Tertiary Care Teaching Hospital of Chhattisgarh

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Abstract

Background: Oxidative stress has been implicated in the pathogenesis of acute myocardial infarction. In recent years, cardiac troponin (cT) has revolutionized the diagnosis and management of acute myocardial infarction (AMI). But in India, most tertiary care hospitals are depending on creatinine kinase (CK), CK-MB, asparte transaminase (AST), and MDA for the diagnosis of AMI due to the unavailability of cT. **Aim and objective:** Assessment of Cardiac Enzyme Level and Lipid Peroxidation in Patients with Actute Myocardial Infarction at the Tertiary Care Teaching Hospital of Chhattisgarh. **Material and method:** The study was carried out in the Department of Cardiac and Biochemistry, SSIMS, Bhilai, C.G. patients, and 30- to 70-year-old age and sex-matched healthy controls. The patients who were recruited from the Critical Care Unit (SSIMS) were brought to the emergency room with a history of chest pain. Patients with chest pain were diagnosed to have AMI according to the clinical criteria: chest pain that lasted for more than 3 hours, ECG changes (ST elevation of 2mm or more in at least two leads), and elevation in total CK, CK-MB, and trop-I+ve. **Result:** We have found a significant increase in MDA, total CK, CK-MB, and AST ($p< 0.001$) and a significant decrease in total thiols ($p< 0.001$) in AMI patients as compared to healthy controls. **Conclusion:** Reactive oxygen species play a role in the pathogenesis of atherosclerosis, thus leading to acute coronary events.

Keywords: *MDA, myocardial infarction, oxidative stress, cardiac enzymes.*

Introduction

Acute myocardial infarction (AMI) is one of the major causes of mortality and morbidity in the world. [1] The most common cause of AMI is atherosclerotic coronary artery disease (CAD) with the erosion or rupture of a plaque, thus causing

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transient, partial, or complete arterial occlusion. [2] Acute ischemic stroke has been shown to increase the incidence of acute coronary syndrome and vice versa [3]. Patients with transient ischemic attacks have a higher relative risk of myocardial infarction (MI) than the general population [4]. According to a study, 13.7% of individuals with acute ischemic stroke exhibited an increased cardiac biomarker [5]. In the absence of primary cardiac issues, myocardial damage was found following an ischemic stroke, the pathogenesis of which is unknown. More than 50% of coronary stenosis was found in up to one-third of patients with ischemic stroke who had no previous history of cardiac symptoms. Following an acute ischemic stroke, there is a substantial chance of recurrence; nevertheless, myocardial infarction is the major cause of death in these patients [6–7].

Previous studies have shown that reactive oxygen species (ROS) cause the initiation and progression of atherosclerosis, thus leading to coronary artery disease. [8] During AMI, two distinct types of damage occur to the heart: ischaemic injury and reperfusion injury. The heart can tolerate a brief exposure to ischaemia because of temporary protective mechanisms like anaerobic glycolysis, fatty acid utilization, an increase in glucose uptake, and decreasing contractility of heart muscles. Persistent ischaemia can lead to a severe ATP deficit and myocardial cell death. [9] During ischaemia, ROS can be produced both by the endothelial cells and the circulating phagocytes, and they are capable of damaging macromolecules, including nucleic acids, proteins, lipids, lipoproteins, and carbohydrates. [10] On interaction with unsaturated lipids, ROS are capable of initiating the self-perpetuating chain reactions of lipid peroxidation in the membranes. Malondialdehyde (MDA), a lipid peroxidation end product, is considered one of the markers of cell membrane damage. [11] The major antioxidant in the body fluids is cysteine-SH, which is bound to proteins, with the majority of it being found in albumin and glutathione (GSH). These SH groups (total thiols) play a major role along with other antioxidants in the body to ameliorate the lipid peroxidative effects of ROS. [12]

The aim of the study is to measure MDA, which is an important marker of lipid peroxidation, along with cardiac enzymes like total CK, CK-MB, and AST in AMI patients at 12 hours after the onset of chest pain, to compare their levels with those of age-matched healthy controls. We have also tried to establish a relationship between oxidative stress markers and cardiac enzymes.

Materials and Methods

The study was carried out in the Department of Cardiac and Biochemistry, SSIMS, Bhilai, with C.G. patients and 30- to 70-year-old age and sex-matched healthy controls. The patients who were recruited from the Critical Care Unit (SSIMS) were brought to the emergency room with a history of chest pain. Patients with chest pain were diagnosed to have AMI according to the clinical criteria: chest pain that lasted for more than 3 hours, ECG changes (ST elevation of 2mm or more in at least two leads), and elevation in total CK, CK-MB, and trop-I+ve. Informed consent was obtained from all the subjects who were involved, and ethical clearance was obtained from the Institutional Ethics Committee (IEC). The patients under treatment were also considered cases. Patients with renal failure and malignancies have been excluded from this study.

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Sample collection

Blood samples were drawn into plain vacutainers from the antecubital veins of AMI patients immediately after admission. Similarly, samples were also obtained from age- and sex-matched healthy controls. Total CK, CK-MB, AST, and MDA levels were measured in all the obtained samples after proper processing.

Measurement of cardiac enzymes:

Reagent kits for total CK, CK-MB, and AST were obtained from MeRLIN, India**.** Cardiac enzymes like total CK, CK-MB, AST, and MDA were measured by an enzymatic assay using an Erba fully automated analyzer.

Statistics Analysis

Mean ± SD were calculated for all the parameters to examine and were differentiated by the student's *t*-test using SPSS 23. *P*-values considered significant were as follows: – $P < 0.05$– a Significant and $P > 0.001$ –a highly Significant.

Observation and Result

Table No. 1: Showing the comparative changes between the demographics between the demographics of acute myocardial infraction patients and normal healthy control subjects.

Variable	**Case (mean ± SD)**	**Control (mean ± SD)**	**P-value**
Age	55.3±12.26	**50.33±12.17**	**0.121**
Sex (M:F)	**19:11**	**17:13**	
Weight	**82.46±10.78**	**75.76±9.88**	**0.015**

The mean age, sex, and weight of the patients were 55.3±12.26 years, with 19 males and 11 females and 82.46±10.78 years, **and** that of the controls was 50.33±12.17 years, with 17 males and 13 females and **75.76±9.88 years,** respectively.

Figure No. 1 shows the difference between the case group and the control group in mean ± SD.

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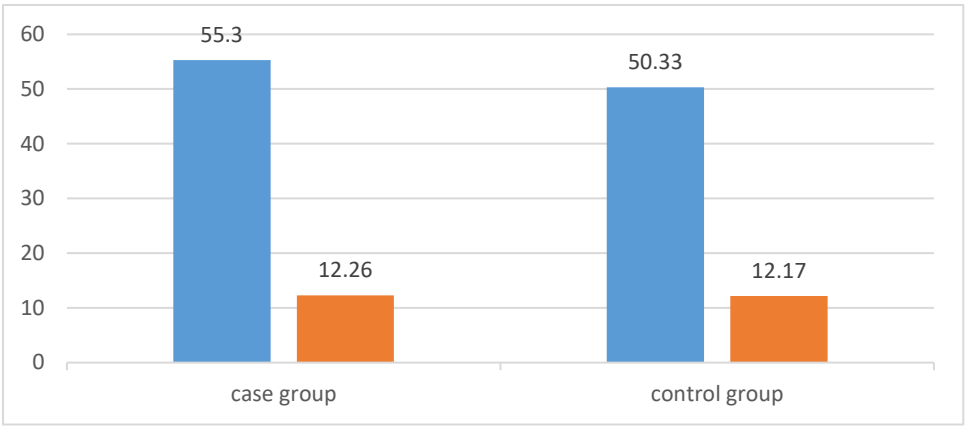
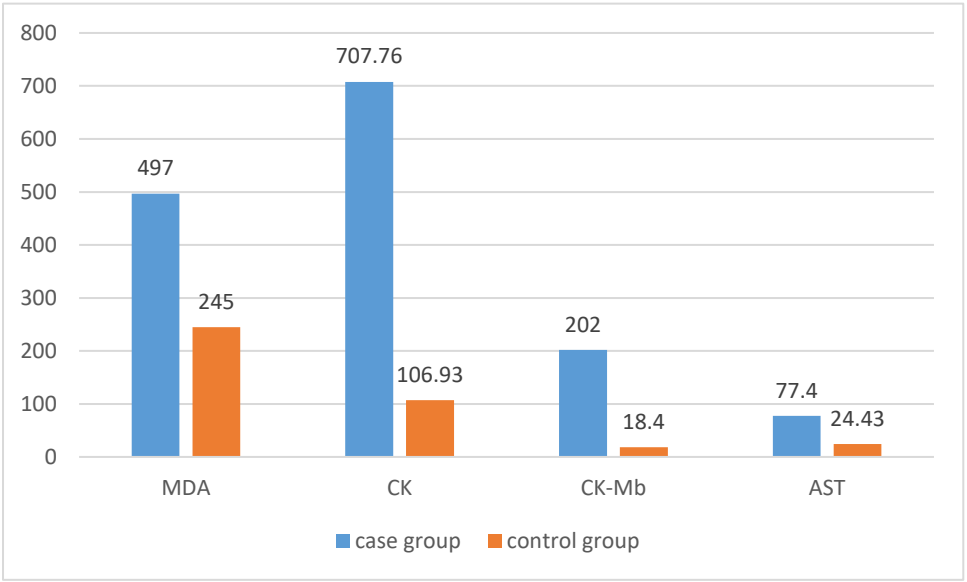


Table no. 2: Showing the comparative changes of MDA, total CK, CK-MB, and AST levels in healthy controls and myocardial infarction patients at 12 hours after the onset of chest pain

Variable	Case (mean ± SD)	Control (mean ± SD)	P-value
MDA(nM/L)	497±79.57	245±42.09	0.000
CK(U/L)	707.76±163.63	106.93±17.84	0.000
CK-Mb(U/L)	202±19.69	18.4±6.7	0.000
AST(U/L)	77.4±10.0	25.43±5.44	0.000

Table show that the parameters [MDA (497±79.57), 707076±163.63), CK-Mb (2022±19.69) and AST (77.4±10)] of cardiac enzyme and lipid peroxidation were significantly increased compared with the control group. Statistically significant increases in MDA, total CK, CK-MB, and AST levels in myocardial infarction patients compared with the control group. ($p<0.05$)

Figure no.2: showing different between case and control group of Mean



Discussion

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The present study found a significant increase in the cardiac enzymes in AMI patients, which rose in parallel to the extent of myocardial injury. The characteristic pattern of the rise in the serum cardiac enzymes was that they started to increase 4–6 hours after injury, reaching peak concentrations after 12–24 hours and returning to the baseline after 48–72 hours. [13] The rise in CK-MB levels was seen in all the AMI patients, and they were elevated about 8 times more than normal. This may indicate the extent of cardiac muscle damage during the ischaemic event, which can further damage the adjacent tissue by generating free radicals. Several previous studies have shown the presence of oxidative stress and the increased generation of reactive oxygen species during myocardial injury. [14]

One of the best markers of lipid peroxidation in serum is MDA, which indicates the amount of membranes that are being damaged by the reactive oxygen species. Being a lipid peroxidation product, the elevation of MDA in AMI patients is an indicator of increased oxidative stress. [14] We have found a significant elevation in MDA levels after myocardial infarction as compared to healthy controls.

In our study, we found that parameters [MDA (497±79.57), 707076±163.63), CK-Mb (2022±19.69) and AST (77.4±10)] of cardiac enzyme and lipid peroxidation were significantly increased compared with the control group. Our study was also supported by Dr. Jeevan Kumar Shetty et al. ROS is responsible for the pathogenesis of AMI and their production are enhanced during ischaemia due to decreased antioxidant defense mechanisms. [15] The introduction of cardiac troponins has been so successful that they have emerged as preferred biomarkers for use in the diagnosis and management of AMI. Previous studies have suggested that troponin is superior to CK-MB as the diagnostic marker for AMI. On the other hand, previous publications report that CK-MB is the most appropriate test for the diagnosis of AMI. [16-17] In India, cardiac troponins are not very frequently used in the diagnosis and management of AMI due to the unavailability of methods and proper equipment to measure them in most peripheral health care centers, and another important issue is the high cost involved in deterring it. CK, CK-MB, and AST are used as important markers in the majority of hospitals due to their easy availability of kits based on simple colorimetric methods and also their cheaper price. In the present study, we have measured CK, CK-MB, and AST in acute chest pain patients in a tertiary care hospital to look for their role in the early diagnosis of AMI in patients with acute chest pain and also to check for their sensitivity and specificity.

This finding further substantiates the role of free radicals in damaging the myocardial membrane. There is growing evidence that an increase in free radicals is relevant to atherosclerotic plaque formation and activation. Furthermore, it has been shown that lipid peroxidation and MDA generation are enhanced by the ischaemic event itself. In ischaemia, the ATPs are drastically reduced and are degraded to hypoxanthine and then into uric acid by xanthine oxidase. During this process, enormous amounts of superoxide radicals are formed, which can stimulate the Haber-Weiss reaction by generating ROS, which enhance the lipid peroxidation process. [18] We have also observed that increased MDA levels after reperfusion correlated positively with the cardiac marker enzyme, CK-MB, which may again explain the ROS-mediated

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damage to the myocyte membranes, thereby increasing the release of the cardiospecific marker, the CK-MB fraction. [19]

Strengths and Limitations of the Present Study

There are a few limitations to the study. In the present study, only 30–70-year-old subjects participated in the research. Hence, in the feature, we would like to include an increase in the number of participants to reach a concrete conclusion. The present study had an impact on understanding how the increased concentration of reactive oxygen species plays a role in the pathogenesis of atherosclerosis, thus leading to acute coronary

Conclusion

Reactive oxygen species play a role in the pathogenesis of atherosclerosis, thus leading to acute coronary events, and their levels are further elevated by the ischaemic event itself. We have also observed that increased MDA levels after reperfusion correlated positively with the cardiac marker enzyme, CK-MB, which may again explain the ROS-mediated damage to the myocyte membranes, thereby increasing the release of the cardiospecific marker, the CK-MB fraction.

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