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Original Research Article

Oxidative stress in myocardial infarction-does it correlate with the cardiac marker troponin?

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ABSTRACT

Introduction: A myocardial infarction (MI), often known as a heart attack, occurs when blood supply to a region of the heart is reduced or stopped, resulting in heart muscle damage. One of the elemental mechanisms responsible for the development of myocardial infarction is oxidative stress. The study aims to assess the Oxidative stress and Troponin I levels in patients with myocardial infarction (MI) and compare them with the level of these parameters in healthy controls. An attempt has been made to find if there is any correlation between oxidative stress and Troponin I levels in patients with myocardial infarction.
Materials and Methods: The Cardiac marker Troponin I and the marker of oxidative stress malondialdehyde were estimated in 30 patients with myocardial infarction and 30 healthy individuals who acted as controls.
Results: A statistically significant difference was observed between Troponin I and MDA in patients with MI as compared with controls. A significant positive correlation was also observed between MDA and Troponin I levels.
Conclusion: In our study there was a significant positive correlation between oxidative stress and Troponin I. Further studies with a larger number of subjects will be needed to find if oxidative stress plays a role in the pathogenesis of myocardial infarction.

1. Introduction

Coronary artery disease (CAD) is one of the leading causes of morbidity and mortality all over the world including India. Acute Myocardial Infarction (AMI) is the most crucial occurrence in cardiovascular diseases and it occurs as a result of myocardial ischemia which arises due to the obstruction of blood flow in the coronary artery.[1] India has one of the highest burdens of cardiovascular disease (CVD) worldwide. 24.8% of deaths in India are due to cardiovascular diseases as estimated by the Global Burden of Disease Study (2010). Overall, cardiovascular diseases contributed 28.1% of the total deaths in India in 2016, compared with 15.2% deaths in 1990. As per the World Health Organization (WHO) data, the incidence of CAD in India resumes to increase.

Troponin I is an ideal biomarker for the detection of cardiac injury. The increase in troponin I level and mortality after MI is directly related.[2] The cardiac TnI is exclusively found in the heart and is absolutely specific. The blood levels of troponin increase within 4 hours after the onset of symptoms of myocardial infarction peaks at 14-24 hours and remains elevated for 3 to 5 days after infarction (b) The cardiac damage level depends on the concentration peak of

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the liberated troponin I and it can be quantitatively used. (c) Troponin I have the highest stability in the blood (6 to 10 days) among other cardiac biomarkers.[3]

Myocardial infarction is due to obstruction to the flow of blood to the heart due to obstruction in the blood vessels. Increased levels of reactive oxygen species (ROS) are observed in vascular tissues including coronary endothelium in CVD, and thus are believed to cause coronary endothelial dysfunction, CAD, and infarction.[4,5] Oxidative stress stimulates vascular smooth muscle cell proliferation, hypertrophy, and collagen deposition, which causes thickening of the vascular media and narrowing of the vascular lumen. Also, an increase in oxidative stress can damage the endothelium, reduce endothelium-dependent vascular relaxation and increase vascular contractile activity. All these factors may explain how increased oxidative stress may play an etiological role in myocardial infarction. The direct measurement of the liberated ROS is strenuous due to their instability. Therefore, a stable lipid peroxidation end product, Malondialdehyde (MDA) is usually used as a marker of ROS production.[6]

2. Materials and Methods

The present case-control study was carried out in the Department of Biochemistry, Pushpagiri Institute of Medical Sciences and Research Centre, Thiruvalla, Kerala. The cases for the present study were selected from the Department of Cardiology. A total of 30 patients admitted with acute Myocardial Infarction were selected.

2.1. Inclusion criteria

Patients with confirmed acute myocardial infarction.

2.2. Exclusion criteria

Patients with any kind of serious illness are excluded.

The study was approved by the Institutional Ethics Committee, Pushpagiri Institute of Medical Sciences and Research Centre, Thiruvalla, Kerala.ethical committee (Registration No. PIMRSC/E1/388A/63/2021).

The age and sex of the subjects were taken and recorded. After taking written informed consent, 2ml of venous blood was collected. The blood was allowed to clot and serum was separated. Troponin I was estimated by high sensitivity Troponin I (hsTnI) Immunoassay method using Access immunoassay analyser. 1ml serum was used for estimation of Malondialdehyde by Thiobarbituric acid method. According to Sato's method, the reaction of MDA with thiobarbituric acid (TBA), forming an MDA-TBA2 adduct.[7]

Results were expressed as mean and standard deviation. An Independent sample 't' test was performed for comparing the quantitative variables between two groups. Relationships between quantitative variables were analyzed by Pearson correlation. A p-value of <0.05 is considered statistically significant. Statistical analysis was done by using MS Excel.

3. Results

The mean and standard deviation of MDA in cases and controls were calculated and are shown in Table 1. The comparison of MDA between cases and controls. In comparison, the mean value of MDA is found to be significantly higher in cases than in the control groups and the difference is statistically significant (P-value ≤ 0.05). The mean and standard deviation of Troponin I in cases and controls were calculated and are shown inTable 2. The comparison of Troponin I between cases and controls. In comparison, the mean value of Troponin I is found to be significantly higher in cases than in the control groups and the difference is found to be statistically significant (P-value ≤ 0.05). The correlation coefficient of Troponin I with MDA was calculated in the study subjects and statistical data is given in Table 3. A statistically significant correlation was found between Troponin I and MDA in the study subjects.

Table 1: Comparison of MDA between cases and controls

Parameters (U/L)	Cases	Controls	P value
MDA	3.89 ± 1.13	1.45 ± 0.13	< 0.00001

The values are given in mean± standard deviation.
The mean difference is significant at the 0.05 level.

Table 2: Comparison of Troponin I between cases and controls

Parameters (ng/ml)	Cases	Controls	P value
Troponin I	2986.1 ± 4033.66	4.29 ± 2.27	0.00035

The values are given in mean± standard deviation.
The mean difference is significant at the 0.05 level.

Table 3: Pearson correlation between Troponin I and MDA

Parameter	Pearson Correlation (r)	P value
Troponin I V/S MDA	0.970	< 0.00001

The mean difference is significant at the 0.05 level.

4. Discussion

Oxidative stress is one of the fundamental factors that contribute to the development of myocardial infarction. Myocardial Infarction is associated with increased ROS formation which in reaction with polyunsaturated fattyacids of lipid membranes results in lipid peroxidation. Malondialdehye (MDA) which is an end product of lipid

peroxidation is used as a marker of oxidative stress. Many studies have reported the association of MDA with Myocardial Infarction.[8,9] Several studies have reported higher levels of serum MDA in MI patients which is due to increased oxidative stress.[10,11] A recent study by Romuk et al 2019 observed significantly higher levels of MDA in MI cases compared to controls.[12] In the present study MDA levels were significantly ($p<0.05$) higher among MI patients (3.89 ± 1.13) compared to normal controls (1.45 ± 0.13) which was in agreement with the findings of previous studies. This suggests that Oxidative stress may play an important role in the pathogenesis of Myocardial Infarction.

Troponin I is a standard used in the diagnosis of Myocardial Infarction. The measurement of serum cTnI is relevant due to its sensitivity and specificity to cardiac muscle enzyme measurements in the diagnosis of cardiac muscle damage. Increased cardiac troponin concentrations are currently acceptable as biochemical markers for MI. Several studies have suggested an increase in Troponin I in individuals with Myocardial Infarction.[13,14] In the present study, the mean values of Troponin I were found to be significantly high (2986.1 ± 4033.66) in MI patients whereas the mean values of Troponin I in the normal healthy controls were significantly low (4.29 ± 2.27). This was in agreement with the results of previous studies.

From these findings it can be concluded that there is a positive correlation between Troponin I and oxidative stress in individuals with Myocardial Infarction. Lifestyle modifications such as exercise, diet and drugs may help to reduce oxidative stress Whether addition of antioxidants to the routine treatment will have any beneficial effect remains to be looked into.

5. Source of Funding

None.

6. Conflict of Interest

The authors declare no conflict of interest.

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