Section: Miscellaneous

Original Research Article

STUDY OF SERUM MALONDIALDEHYDE LEVEL IN PATIENTS WITH ACUTE MYOCARDIAL INFARCTION (AMI)

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ABSTRACT

Background: Acute Myocardial Infarction (AMI) results from blocked coronary blood flow, leading to myocardial ischemia. Reactive oxygen species (ROS) are key drivers in its pathogenesis. Malondialdehyde (MDA), a marker of oxidative stress, worsens AMI by promoting oxidative damage, inflammation, cardiac dysfunction, myocardial remodeling, and endothelial impairment. **Objectives:** To estimate and compare the levels of serum malondialdehyde in individuals with acute myocardial infraction and healthy subjects. **Materials and Methods:** This is a cross-sectional study was conducted in the Department of Biochemistry in collaboration with the Department of Cardiology, RIMS, Imphal. A total of 86 participants were enrolled, including 43 AMI patients and 43 healthy controls. Serum malondialdehyde levels were measured using ELISA (Enzyme-Linked Immunosorbent Assay). **Result:** In this study, we found that the majority of cases were in the age group above 60 years, accounting for 58.1% (25), followed by the 40–60 years age group with 34.9% (15), and the least in the under 39 years age group with 7.0% (3). It was evident that the majority of cases were men, comprising 58.1% (25) of the total. We also found that serum malondialdehyde levels in AMI cases (21.8 ± 4.5 ng/ml) were significantly higher ($P < 0.001$) compared to controls (4.4 ± 1.4 ng/ml). **Conclusion:** This study showed elevated serum malondialdehyde levels in acute myocardial infarction patients. MDA can serve as a useful marker for myocardial injury and oxidative damage, aiding early diagnosis, prognosis, and understanding of the disease's oxidative stress.

INTRODUCTION

Cardiovascular diseases (CVDs) are the leading global cause of death. An estimated 17.9 million people died from CVDs in 2019, representing 32% of all global deaths. Of these fatalities, 85% were attributed to heart attacks and strokes.[1] The incidence of myocardial infarction in India is estimated to be 64.37/1000 people.[2]

Acute Myocardial Infarction (AMI) is a critical event in cardiovascular diseases, occurring due to myocardial ischemia caused by a blockage in the blood flow of the coronary artery.[3] Troponin I (TnI) is a key biomarker for detecting cardiac injury. The rise in troponin I levels is directly associated with increased mortality following a myocardial infarction (MI).[4]

Reactive oxygen species degrade polyunsaturated lipids in cell membranes, leading to the formation of lipid peroxyl radicals and lipid hydroperoxides, which subsequently break down into various reactive aldehydes, such as malondialdehyde (MDA), 4-Hydroxynonenal (4-HNE). These aldehydes, being highly reactive, can form adducts with proteins, lipids, and DNA, which may result in functional impairment, mutations, inflammation, and cell death).[5]

Malondialdehyde (MDA) is an organic compound with the formula CH2(CHO)2. It is a naturally occurring reactive species and serves as a marker for

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oxidative stress. MDA is a sensitive indicator of lipid peroxidation.[6]

Malondialdehyde (MDA) is a widely recognized by product of lipid peroxidation, primarily formed through the breakdown of polyunsaturated fatty acids (PUFAs), particularly arachidonic acid and linoleic acid As a highly reactive aldehyde that forms adducts with cellular macromolecules like proteins, lipids, and DNA. These adducts can disrupt the structure and function of proteins and lipids in heart cells, leading to cellular dysfunction and damage.[5]

Malondialdehyde (MDA) exacerbates myocardial infarction by contributing to oxidative stress, inflammation, impaired cardiac function, myocardial remodelling, and endothelial dysfunction.15 Oxidative stress plays a key role in the progression of atherosclerosis.[7]

The use of malondialdehyde as an additional marker for evaluating myocardial injury and the degree of oxidative damage to the heart tissue. Assessing serum MDA levels in acute myocardial infarction could aid in early diagnosis, improve prognostication, and offer a deeper insight into the oxidative stress underlying the disease.

In this study, we aim to estimate and compare the levels of serum malondialdehyde in AMI patients and controls.

MATERIALS AND METHODS

This cross-sectional study was conducted in collaboration between the Department of Biochemistry and the Department of Cardiology at the Regional Institute of Medical Sciences (RIMS), Imphal, Manipur, India, from March 2023 to March 2025. The study was approved by the Research Ethics Board, RIMS, Imphal.

The study included individuals aged 18 years and above, comprising diagnosed cases of acute myocardial infarction (AMI) as well as age- and sex-matched individuals without AMI, serving as the control group. Participants were excluded if they had any of the following conditions: liver diseases such as cirrhosis or liver metastasis; renal diseases such as chronic kidney disease; diabetes mellitus; recent infections such as typhoid fever; chronic obstructive pulmonary disease (COPD); osteoarthritis; or any form of malignancy.

Methods of data and sample collection

Before the commencement of the study, written informed consent was obtained from all participants. Eligible individuals with acute myocardial infarction (AMI) were conveniently recruited from the casualty, medicine or cardiology wards, and the Intensive Coronary Care Unit (ICCU) of RIMS, Imphal. For each AMI patient enrolled, one age- and sex-matched eligible individual without AMI was also recruited from the outpatient department (OPD) attendees or patient attendants in the medicine or cardiology departments. Blood samples were collected after obtaining informed consent. A 5 ml venous blood sample was drawn into a plain vial, centrifuged for 10 minutes at 2000–3000 rpm, and the serum malondialdehyde level was estimated using the ELISA method.

Statistical Analysis

The collected data were analyzed using IBM SPSS version 26.0 for Windows. Descriptive statistics, including the mean, standard deviation, frequency and proportions, were used to summarize the findings. Age of the participants and serum malondialdehyde level were expressed in mean and standard deviation. To compare the mean serum malondialdehyde level between patients with AMI and the healthy individuals, student's t-test was used. A p-value of less than 0.05 was considered statistically significant for all parameters.

RESULTS

Table 1 and Figure 1 show that the highest number of cases occurred in the age group above 60 years, with 25 cases (58.1%), followed by 15 cases (34.9%) in the 40–60 years age group, and 3 cases (7.0%) in the age group below 39 years. Table 2 and Figure 2 indicate that more cases were found in the male population, with 25 cases (58.1%), compared to the female population, which had 18 cases (41.9%). Table 3 and Figure 3 show that the mean serum MDA level of the study participants was significantly higher in the AMI group (21.8 ± 4.5 ng/ml) compared to the control group (4.4 ± 1.4 ng/ml). This difference was statistically significant ($p < 0.001$).

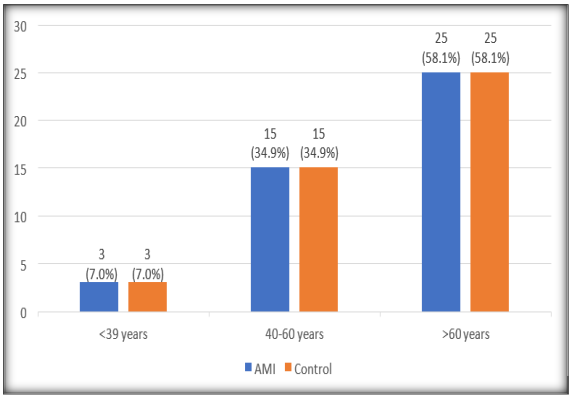

Figure 1: Age wise distribution of the study population

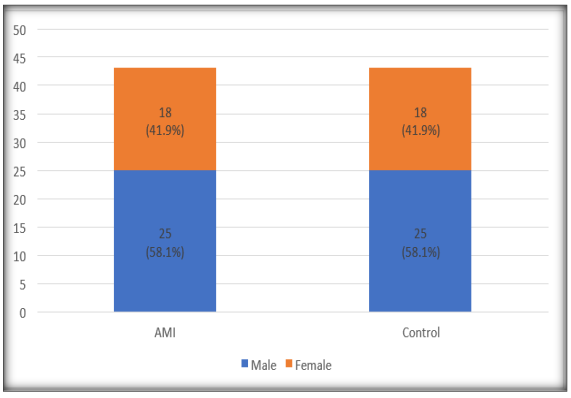

Figure 2: Distribution of participants by sex (N=86)

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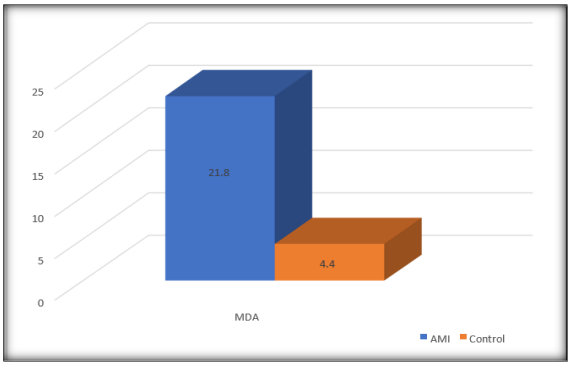

Figure 3: Comparison of the mean serum MDA level between the two groups (N=86)

Table 1: Age wise distribution of study population (N=86)

Age in years	AMI (n=43)	Control (n=43)
< 39 years	3(7.0%)	3(7.0%)
40-60 years	15(34.9%)	15(34.9%)
> 60 years	25(58.1%)	25(58.1%)
Total	43(100%)	43(100%)

Table 2: Distribution of participants by sex (N=86)

Sex	AMI (n=43)	Control (n=43)
Male	25(58.1%)	25(58.1%)
Female	18(41.9%)	18(41.9%)

Table 3: Comparison of the mean serum MDA level between the two groups (N=86)

	AMI Mean ± SD	Control Mean ± SD	p-value
MDA (ng/ml)	21.8 ± 4.5	4.4 ± 1.4	<0.001

Independent t test

DISCUSSION

The study showed the majority of AMI cases were in the age group above 60 years, accounting for 58.1% (25), followed by the 40-60 years age group with 34.9% (15), and the least in the under 39 years age group with 7.0% (3). This study confirms the observation made by Tanna NA et al,[8] which involved 208 CAD patients, with 32.21% in the 60-69 age group and only 4.32% aged 80 or older. In contrast to our study, Baby N et al,[9] found a higher susceptibility to AMI in individuals aged 51-55 compared to other age groups. The findings of our study is similar to that of Salari N et al,[10] who reported a higher prevalence of MI in individuals over 60 years. Aging is associated with various factors, including increased oxidative stress, inflammation, apoptosis, and overall myocardial deterioration and degeneration.[11] With aging, the heart undergoes structural changes such as ventricular hypertrophy and fibrosis, which impair its function.[12] Moreover, a decline in endothelial function limits coronary artery dilation and oxygen delivery.[13] Thus, aging increases the risk of an imbalance between myocardial oxygen demand and supply.

In the study the majority of cases were men, comprising 58.1% (25) of the total. Similar observations were made by Kocabaş R et al[14] who found that males were more predominant than females, suggesting male gender as one of the risk factors for cardiovascular events. This is attributed to the cardioprotective effect of estrogen (E2) in women of reproductive age, which binds to estrogen receptor alpha (ERα), estrogen receptor beta (ERβ), and G protein-coupled estrogen receptor (GPR30). These interactions promote vasodilation, angiogenesis, and a reduction in reactive oxygen species production, thereby improving overall survival in premenopausal women.[15]

The study displayed that the highest mean of MDA was recorded in AMI group (21.8 ± 4.5 ng/ml) as compared with the control group (4.4 ± 1.4 ng/ml). The result was significant ($p< 0.001$), as shown in the Table 3. In agreement with this finding, Kathyaini R et al,[16] found that the mean serum MDA was significantly increased in patients with acute myocardial infarction compared to healthy individuals. Ibrahim DA et al,[17] study demonstrated that MDA levels were significantly elevated in the AMI group compared to the control group. Several studies have reported higher levels of serum MDA in MI patients which is due to increased oxidative stress.[18,19] Moreover, Jain AP et al,[20] reported that plasma levels of MDA and nitrite were significantly elevated in AMI patients compared to the control group, indicating that oxygen free radicals cause endothelial damage in these patients. Oxidative stress is a condition where oxidant metabolites cause toxic effects due to increased production or altered cellular

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protective mechanisms. This leads to the oxidation of thiol groups and lipid peroxidation, initially causing reversible damage and eventually progressing to necrosis.[21] Oxidative stress is considered one of the key factors in the progression of atherosclerosis.[22] Studies have reported that oxidative stress in acute myocardial disease results from reperfusion and an imbalance between antioxidants and pro-oxidants.[23,24]

Free radicals can oxidize lipids, proteins, and carbohydrates, damaging cell membranes and DNA, which alters cellular structure and function. Cell membranes are rich sources of polyunsaturated fatty acids, which are more prone to be attacked by oxidizing radicals causing lipid peroxidation.[25] Consequently, Malondialdehyde (MDA), an end product of lipid peroxidation, is widely used as a marker of oxidative stress. MDA reflects the free radical mediated peroxidation of polyunsaturated fatty acids and auto-oxidation.[26] MDA levels increase with the accumulation of oxygen free radicals, leading to lipid peroxidation in cell membranes, acute cardiac injury, mitochondrial dysfunction, reduced myocardial systolic function, and eventually induced AMI.[27] These findings confirm our study results that serum MDA levels are notably higher in AMI patients. This can be attributed to the fact that oxidative stress is a major contributor to acute myocardial infarction (AMI), with elevated malondialdehyde (MDA) levels serving as an indicator of increased oxidative stress and diminished antioxidant defenses in AMI patients. Therefore, MDA is one of the most commonly recognized biomarkers of oxidative stress. Its ability to interact with organic molecules such as DNA and proteins, enables it to disrupt various biological pathways.[9]

CONCLUSION

The study demonstrates that serum MDA levels were higher in AMI patients compared to healthy participants. Elevated serum malondialdehyde (MDA) levels are strongly linked to cardiovascular risk and are significantly higher in AMI, indicating increased oxidative stress and reduced antioxidant defense. The observed link between MDA and AMI suggests that serum MDA levels could serve as a adjuvant marker for predicting AMI risk. Therefore, incorporating antioxidant supplements into AMI management may offer benefits, though further studies are needed to confirm their effectiveness.

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Conflict of interest: Nil

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