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A STUDY ON SIGNIFICANCE OF CARDIAC ENZYMES AND SERUM ADENOSINE DEAMINASE LEVELS IN MYOCARDIAL INFARCTION.

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ABSTRACT

Background: Myocardial Infarction (MI) signifies the culmination of the acute coronary syndrome continuum, characterized by irreversible ischemic injury that results in cellular death and necrosis. The majority of MI cases are attributed to coronary atherosclerosis, which contains macrophages and activated T-cells that release cytokines. Previous studies have indicated the involvement of an immunological component in the development of atherosclerotic lesions associated with coronary heart disease. This study aims to assess the activity level of adenosine deaminase (ADA), a recognized non-specific marker of T lymphocyte activation, alongside Troponin-I and cardiac enzymes in patients diagnosed with Myocardial Infarction (MI). The research included 40 MI cases and 20 age- and sex-matched controls. Measurements of serum ADA, cardiac enzymes, and Troponin-I were conducted. The results demonstrated significantly elevated levels of serum ADA, CK-MB, LDH, AST, and Troponin-I in MI patients compared to healthy controls. These findings support the notion of T lymphocyte activation and proliferation in MI patients and propose ADA as a potential marker for elucidating the pathogenesis of MI.

INTRODUCTION

Acute myocardial infarction (AMI) is defined as an imbalance between myocardial oxygen supply and demand resulting in injury to and the eventual death of myocytes[1]. All most all MI cases result from coronary atherosclerosis generally with superimposed coronary thrombosis. It is the major cause of mortality in middle age and elderly population.

In MI there is a decrease in coronary flow or an equivalent abrupt increase in myocardial demand for oxygen due to ischemic necrosis caused by obstruction in the coronary artery. Thrombus or atherosclerotic plaque may obstruct the coronary flow. The atherosclerotic lesion contain macrophage and activated T-cells which secrete cytokines . This inflammatory response results in plaque rupture and thrombosis causing MI[2].

The diagnosis of MI is based on clinical symptoms, electrocardiographic (ECG) changes and characteristic pattern of changes in some serum enzymes such as creative kinase(CK), creatine kinase isoenzyme MB (CK-MB), lactate dehydrogenase (LDH) and cardiac specific proteins like Troponins. MI is diagnosed mostly based on the ECG findings because clinical symptoms are varied from subject to subject. But inconclusive ECG pattern puts clinician

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in a dilemma, created on their plays the role of serum biochemical markers of myocardial injury to confirm the diagnosis of MI[3]. Numerous biomarkers have been monitored to assess myocardial injury. The availability of serum cardiac markers with markedly enhanced sensitivity for myocardial damage enables clinicians to diagnose MI in approximately an additional one-third of the patients who would not have been fulfilled criteria for MI in the past[4]. Thus the aim of the study was drawn to study the relation between established cardiac enzyme markers like (CK-MB, AST, LDH, Troponin -I) and less established serum Adenosine Deaminase activity in acute myocardial infarction cases.

MATERIALS AND METHODS

The current investigation was carried out within the Department of Biochemistry in conjunction with the Department of General Medicine at SIMSRH, Karnataka. The focus of the study was on patients diagnosed with Acute Myocardial Infarction who were admitted to the intensive care unit of SIMSRH. Approval for the study was obtained from the Institutional Ethical Committee. The study population comprised 40 patients (aged between 30 and 50 years) suffering from myocardial infarction, while the control group included 20 healthy individuals matched for age and sex, all of whom had no prior history of any medical conditions. Each patient underwent a clinical examination, and data regarding age, sex, lifestyle habits, and overall health status were meticulously recorded in a designated case proforma. A total of 5 ml of venous whole blood samples were collected from both the control group and the myocardial infarction patients, allowed to clot, and subsequently centrifuged at 5000 RPM for 10 minutes to separate the serum.

Creatine kinase (Ck-MB)[5], Aspartate transaminase (AST)[6], Lactate dehydrogenase (LDH)[7] were estimated by IFCC Kinetic method using Tulip-evolution 3000 semi autoanlyzer. Troponin-I was estimated using Instant View Troponin-I cards based on principle of Immunoassay [8]. Serum ADA levels were estimated by Guisti and Galanti colorimetric method [9]. Statistical analyses were done using student t-test and p-value significance. P-value

<0.01 were considered as significant.

RESULTS

The present study included a total number of 60 subjects comprising of 20 normal individuals ((controls)Group-I) and 40 myocardial infarction cases (group-II). The mean±SD of CK-MB in group-I is 18.43±2.92 and in group –II 107.8±19.9 . CK-MB was significantly increased in group- II when compared with group-I with p-value < 0.001.The levels of AST in group-I (Mean±SD is 21.2±4.75) and group-II (Mean±SD is 141.3±21.01) .Group-II values were significantly increased with p-value <0.001.The mean±SD of LDH in group-I is 328.53± 48.6 and in group – II 798.53±60.2 with p-value <0.01 . LDH was significantly increased in group-II when compared with group-I.The CK-MB , AST , LDH values are significantly increased in group - II when compared with group-I.The Mean±SD of serum ADA in group –I and II is 17.49±3.83 and 52.06±7.77 (p-value <0.01) .ADA values are significantly increased in group –II when compared with group-I. Troponin-I values are negative in controls which are compared with MI patients with positive values (Qualitatively).

Sl. No.	Parameters	Group I Mean ± SD	Group II Mean ± SD	P-Value
1	CK – MB	18.43±2.92	107.8±19.9	<0.001

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2	AST	21.2±4.75	141.3±21.01	<0.001
3	LDH	328.53±48.6	798.53±60.2	<0.01
4	ADA	17.49±3.83	52.06±7.77	<0.01

DISCUSSION

Myocardial infarction represents a significant global health challenge, underscoring the necessity for prompt diagnosis of this condition. Laboratory parameters are crucial in the identification of acute myocardial infarction (AMI) and are instrumental in monitoring the progression and extent of the infarction. Among the various biomarkers currently utilized, Troponins stand out as the most promising indicators. Creatine kinase-MB (CK-MB) can be detected within 4 to 8 hours following the initial onset of chest pain, reaching its peak between 18 to 24 hours, and subsequently returning to normal levels within 2 to 3 days. Abnormal levels of aspartate aminotransferase (AST) can be observed between 6 to 8 hours after the onset of chest pain, peaking on average at 24 hours, and normalizing within 3 to 6 days. Lactate dehydrogenase (LDH) levels typically rise within 8 to 12 hours and peak between 24 to 72 hours. Troponin-I levels are detectable 2 to 8 hours post-myocardial infarction, with peak levels occurring at 18 to 24 hours. In our study, CK-MB, AST, LDH, and Troponin-I were found to be significantly elevated in cases of myocardial infarction compared to control subjects. Adenosine deaminase (ADA) is an enzyme that plays a role in purine metabolism via the salvage pathway, converting adenosine to inosine. Its activity is essential for the proliferation, maturation, and functionality of lymphocytes, particularly T-lymphocytes. ADA serves as a marker for evaluating cell-mediated immunity in conditions characterized by T-lymphocyte proliferation and maturation. Adenosine has the potential to enhance coronary artery blood flow during periods of stress and hypoxia, thereby helping to balance oxygen supply and demand. However, the benefits of adenosine may be compromised if it is rapidly metabolized by ADA, leading to the production of inosine, which generates superoxide radicals and exacerbates ischemic injury. The findings of the current study indicate a highly significant increase in ADA levels among patients with myocardial infarction when compared to control subjects. This observation aligns with the research conducted by A. Jyothi et al. and Neela Patil, who attributed the elevation to increased T-lymphocyte activation and proliferation in the pathogenesis of myocardial infarction.

CONCLUSION

Our results suggest increase in cardiac markers along with increased ADA activity in MI. It is also suggested that ADA can serve as inflammatory marker which is poorly studied with respect to MI.

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