A STUDY ON CARDIAC ENZYMES AND SERUM ADENOSINE DEAMINASE IN MYOCARDIAL INFARCTION

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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 which is associated with abnormal blood flow and atherosclerotic plaque which obstruct the coronary flow. The atherosclerotic lesion contains macrophages and activated T-cells which secrete cytokines. The presence of immunological component in the progression of atherosclerotic lesions of coronary heart disease has been suggested in the earlier studies. The present study is one such attempt to estimate the level of adenosine deaminase (ADA) activity (an accepted non-specific marker of T lymphocyte activation) along with Troponin-I and cardiac enzymes in patients with Myocardial Infarction (MI). 40 cases presenting with MI and 20 age and sex matched controls were included in the study. Serum ADA, cardiac enzymes and Troponin-I were estimated. The serum ADA levels, CK-MB, LDH, AST and Trop-I levels were significantly increased in MI patients when compared with normal healthy controls. The observation of the present study provides evidence for T lymphocyte activation and proliferation in MI patients and suggests ADA as one of the markers to elucidate the pathogenesis of MI.

MATERIALS AND METHODS

The present study was conducted in the Department of Biochemistry in collaboration with Department of General Medicine of SVS medical college and Hospital, Yenugonda, Mahabubnagar. The study was conducted on patients with acute Myocardial Infarction admitted in intensive care unit of SVS Hospital and medical college. This study was approved by the Institutional Ethical Committee. Study population consisted of 40 patients (age range 30-50 yrs) with MI and control group consisted of 20, age and sex matched healthy individuals with no known history of any disease. All the patients were examined clinically and information pertaining to age, sex, habits and health status were recorded in a special case proforma. 5ml of venous whole blood samples were collected from both controls and MI patients and allowed to clot, centrifuged at 5000 RPM for 10 minutes and serum was separated.

Keywords:
Acute Myocardial infarction (AMI)
Adenosine deaminase (ADA)
Cardiac markers.
Creatine kinase (CK-MB)[5], Aspartate transaminase (AST)[6], Lactate dehydrogenase (LDH)[7] were estimated by IFCC Kinetic method using Tulip-evolution 3000 semi auto analyzer. 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 analysis 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-I 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).

DISCUSSION

Myocardial infarction is the leading health problem all over the world so it has become important to achieve early diagnosis of MI. The laboratory parameters play a major role in diagnosing AMI. They also monitor the course and size of infarction. Among the currently available markers Troponins are the most promising currently available markers Troponins are the most promising. They also monitor the course and size of infarction. Among the currently available markers Troponins are the most promising ones[3,10]. CK-MB is detectable in 4-8 hrs after 1st onset of chest pain and peaks at 18-24 hrs and declines to normal within 2-3 days[11]. Abnormal AST levels are observed between 6-8 hrs of onset of chest pain and peaks of AST on an average of 24 hours and finally returns to normal levels in 3-6 days[12]. LDH values is raised in 8-12 hrs and peaks at 24-72 hrs[13]. Troponin-I are observed at 2-8 hrs post MI and peaks at 18-24 hrs[14]. CK-MB, AST, LDH, Troponin-I were significantly elevated in MI cases compared to controls in our study.

ADA is an enzyme involved in purine metabolism through salvage pathway which catalyzes adenosine to inosine. Its activity is necessary for proliferation , maturation and function of lymphocytes, specifically T-lymphocytes. It is a marker for assessing cell mediated immunity in diseases characterized by T-lymphocyte proliferation and maturation[15].

Adenosine can increase coronary artery blood flow during stress and hypoxia to balance oxygen supply and demand .The advantages of adenosine will be lost if it is rapidly metabolized by ADA. It is catalyzed to inosine which produce superoxide radicals and exaggerate ischemic injury[16]. The results of the present study showed highly significant increase in ADA levels in patients with MI as compared to controls. This is in agreement with A.Jyothi et al., neela patil. They attributed to the increased T-lymphocyte activation and proliferation in pathogenesis of MI[17].

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.

REFERENCES


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