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COMPARATIVE STUDY OF SERUM AST, CK-MB, IN MYOCARDIAL INFARCTION WITH SURVIVAL AND MYOCARDIAL INFARCTION WITHOUT SURVIVAL

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ABSTRACT

Background: Myocardial infarction has varied presentation and associated high mortality. Patient's identification with acute myocardial infarction (MI) is very critical for their management and the prognosis. The goal of present study was to correlate the diagnostic value of cardiac biomarkers like AST (SGOT) and CK-MB in MI patient with and without survival. **Materials and methods:** This study was conducted on 50 MI patients admitted to the Emergency Department of Government DB Hospital Churu within 12 hours of acute chest pain and 50 healthy volunteers (age and gender matched) as a control group. Serum creatine kinase-MB (CK-MB), and aspartate transaminase (AST) were measured. For Analyzing the Data, Statistical software SYSTAT version 12 was used. The results were revealed in mean ± standard deviation. Comparisons of cases and control groups were done by applying Z test. Student's t-test at 5% ($p = 0.05$) and 1% ($p = 0.01$) level of significance was used for correlation. **Results:** Mean levels of serum CK-MB and AST levels were significantly higher ($p < 0.01$) in patients with MI in contrast with healthy volunteers. Serum levels of above cardiac biomarkers were significantly elevated ($p < 0.01$) in MI patients without survival as compared with MI with survival. **Conclusion:** The serum levels of biomarkers were raised in MI without survival as compared with MI with survival. These study data manifest that these changes might be useful to obtain a comprehensive view of the infarct size and severity of vascular stenotic lesions.

Keywords: Acute myocardial infarction, Creatine kinase-MB, Myocardial infarction, AST (SGOT).

INTRODUCTION

Coronary Vascular disease (CVD) is defined as an acute or chronic cardiac disability arising from imbalance between the myocardial supply and demand for oxygenated blood. **(1)** It is a multifactorial disease in etiology and has a spectrum of presentations ranging from stable angina, acute coronary syndrome to completely asymptomatic disease. **(2)** CVD is the largest killer disease in developed countries and is rapidly assuming a similar role in developing countries. The World Health Organization (WHO) has drawn attention to the fact that CVD is our modern epidemic, not an unavoidable attribute of aging. **(3)** According to WHO 17.7 million deaths (i.e., 31% of total deaths) of CVD occurred in 2017 globally. **(4)** More than 75% of CVD death occur in low- income and middle-

income countries. According to the Global Burden of Disease study age-standardized estimates (2010), nearly a quarter (24.8%) of all deaths in India are attributable to CVD. **(5)**

According to a previous study, it was concluded that the cascade of thrombotic events following atherosclerotic plaque rupture cause occlusion of the coronary artery, which interrupts blood supply and oxygen to the myocardium. **(6)** Myocardial necrosis with subsequent infarction is followed by heart failure, myocardial rupture, or arrhythmias. Myocardial infarction is the main pathophysiological characteristic of CVD. It occurs due to a lack of nutrients and oxygen reaching the heart muscle by reduction of blood supply to one area of the heart. **(7)** Early treatment like fibrinolysis, coronary artery bypass grafting, and percutaneous coronary intervention of MI help to prevent necrosis. However, for well-timed diagnosis, biomarkers play important roles to help us improve our diagnostic of the MI. **(8)** The manifestations of the MI are varied and multiple like chest pain, epigastric or arm discomfort, breathlessness, nausea, and vomiting. However, these symptoms may be tenuous and are not recognized. Because of the varied presentations and associated high mortality, the identification of MI and early diagnosis of CVD are one of the bottlenecks in medical practice of cardiology. Only about 22% patients admitted to cardiac care centers with chest pain actually suffer from MI, which is the acute clinical pattern of CVD. **(9)**

The blood test is a noninvasive method and The CK-MB is the most universal marker of myocardial necrosis, but the specificity of CK-MB for diagnosing MI is within limits. It is not sole to the myocardial, but also high in the setting of muscle trauma. **(10)**

CK-MB is an important enzyme in energy metabolism that catalyses the transfer of phosphate group of creatine phosphate to ADP to form ATP. It is the first enzyme to be elevated in circulation within 3 to 6 hours after symptomatic onset of MI.

In the Year 1954, AST was the first cardiac Biomarker to be used. AST is present in liver, Heart, Skeleton Muscles, Brain, and Kidney. Due to its lack of specificity, it is no longer used for diagnosis of acute MI, can be used for prognostic purposes.

Thus, in view of above information and several risks of complication, it is worthwhile to study the various biomarkers in MI. Thus, the aim of our study was to determine serum level of cardiac markers for early diagnosis of MI in patients presenting with symptoms of acute chest pain. The study also tried to correlate the serum level of the biomarker in MI with survival and MI without survival.

MATERIALS AND METHODS

The present study was conducted from September 2017 to April 2018, in the Department of Biochemistry of Govt. Medical College, CHURU and attached Dedraaj Bharatiya Hospital Churu, Rajasthan, India.

The study was approved by the Institutional Ethics Committee of Government Medical College and DB hospital, Churu, Rajasthan, India with all participants providing informed consent, and utmost care was taken during the experimental procedure according to the Declaration of Helsinki 1975.

Study Design

Type: Analytical case-control study.

Population: Totally, 100 subjects were enrolled in the present study of which MI patients were 50 and controls were 50.

Sampling: Simple random sampling. In the present study, the population was not universal. The study was carried out on available individuals that served as the accessible population.

Control Group

Totally, 50 healthy age- and sex-matched individuals without any evidence of MI as per clinical examinations were taken as control subjects.

Patients Group

The study included totally 50 patients between the age group 35 to 75 years of MI and had been taken

from the intensive cardiac care unit (ICCU) having chest pain. The patients were diagnosed by physicians, blinded to the results of markers; data included history, physical examination, serial 12-lead electrocardiogram, and cardiac markers measurement.

Inclusion Criteria

The diagnosis of all patients of MI was made by physicians. Patients who had typical symptoms of MI like chest pain, sweating, breathlessness etc., and specific abnormalities for MI on electrocardiogram and elevated cardiac markers were included in the present study.

Exclusion Criteria

All patients having heart diseases like congenital heart disease, diseases of heart valves, and myocardium. Confounding factors that could interfere in the biochemical analyses of study subjects and alter the results were diabetes mellitus, renal insufficiency, hypertension, hepatic disease, inflammatory disease, history of recent infection, and febrile disorders.

After taking informed consent, all subjects were screened for inclusion and exclusion criteria. All the subjects were categorized into different groups as follows.

Collection of Specimen

Criteria for blood collection were different for different groups:

For control, 5 mL blood was collected between 9.00 and 11.00 am by using 20G disposable needle from cubital vein with aseptic precaution. For MI, 5 mL blood was collected within 12 hours after admission in the ICCU. Plain vaccutainer (Randox Diagnostic) was used for estimation of CK-MB, AST. After an hour, the samples were centrifuged at 3000 rpm for 10 minutes to separate serum. The separated serum was collected in polythene tube with cork and stored at 20°C (precautions were taken to avoid the hemolysis) and used for analysis of respective parameters.

Methods

Determination of CK-MB by Liquid Stable Optimized UV Method/immune-inhibition Methods

The procedure included the measurements of CK activity in the presence of antibody to CK-MB monomer12. This antibody completely inhibits the activity of CK-MM and half of the activity of CK-MB, while not affecting the B-subunit activity of CK-MB and CK-BB monomers. The CK-MB activity was obtained by multiplying the CK-B activity by two.

Estimation of Aspartate Aminotransferase by Modified UV Kinetic Assay15

Aspartate aminotransferase catalyses the transamination of L-aspartate and α-ketoglutarate to form L-glutamate and oxaloacetate. Malate Dehydrogenase (MDH) reduces oxaloacetate to malate with simultaneous oxidation of Nicotinamide Adenine Dinucleotide (reduced) NADH to Nicotinamide Adenine Dinucleotide (NAD). The rate of oxidation of NADH was expressed kinetically by monitoring the decrease in absorbance at 340 nm and was directly proportional to AST activity in the sample. Lactate Dehydrogenase is added to enzyme system to prevent endogenous pyruvate interference which is normally present in the serum.

Ethics

The study was approved by the Institutional Ethics Committee of Government Medical College and attached Dedraaj Bharatiya Hospital, Churu, Rajasthan, India

Statistical Analysis

Statistical software SYSTAT version 12 was used to analyze the data. The results were expressed in mean ± standard deviation (mean ± SD). Data were analyzed by descriptive statistics as mean, SD, percentage, etc. Comparisons of study groups and study groups with control groups were done by applying Z test of the difference between two sample means at 5% (p=0.05) and1% (p=0.01)levels of significance.

RESULTS

Table 1 show mean ± SD levels of CK-MB and AST in MI patients as compared with healthy controls. In the same way, Table 2 illustrates that mean levels of CK-MB, and AST were significantly ($p<0.01$) increased in MI without survival when compared with MI with survival. The normal level of CK-MB in a healthy person is 0.0 - 5.5 ng/ml and normal level of AST (SGOT) is12-38U/l.

DISCUSSION

Cardiovascular diseases, generally coronary artery disease, have the potential of causing sudden death, which is described as a natural death due to cardiac causes through abrupt loss of consciousness within one hour of the onset of acute symptoms. **(1)**

Atherosclerosis is the main cause of CVD, having a prevalence of more than 90%, while other causes are responsible for less than 10% cases of CVD. The MI is due to an acute or subacute primary reduction of myocardial oxygen supply evoked by the destruction of an atherosclerotic plaque associated with inflammation, thrombosis, vasoconstriction, and micro embolization. Myocardial infarction is the main pathophysiological characteristic of CVD. It mainly happens due to lack of nutrients and oxygen reaching the cardiac muscle by reduction of blood supply to one area of the heart. **(7)** Identification of MI is extremely important for the management and prognosis of CVD. **(9)** Hence, in clinical practice, extreme attention has been paid to measure myocardial markers in the diagnosis of acute MI, stratification of acute coronary syndrome risk, and differential diagnosis of reversible vs irreversible MI and acute chest pain.

The current study showed highly significant ($p < 0.01$) mean levels of CK-MB, and AST in MI as compared with healthy volunteers. In the current study, we also tried to compare the MI with survival and MI without survival. The mean levels of CK-MB (41.10% increased) in MI without survival in contrast with MI with survival.

Pasupathi11 have shown that biochemical marker "CK-MB" is significantly altered with MI patients. The CK-MB enzyme normally exists in cellular compartment and leaks out into plasma during myocardial injury due to disintegration of contractile element and sarcoplasmic reticulum. **(12)**

In the current study, our findings confirm and expand upon previous reports, **(13, 14)** In the current study, serum levels of CK-MB and AST were significantly increased in MI. Peppes et al **(15)** have suggested that raised serum levels of myocardial enzymes with the coronary vascular disease in Greek patients. Our results were precisely matched to this outcome. Abdullah **(16)** had demonstrated in his study that prolonged ischemia originates in the accumulation of non-esterified fatty acid intra and extracellularly, which might change the permeability of plasma membrane of heart leading to the leakage of cellular substance and enzyme outside the cells.

CONCLUSION

The CVD is invariably caused by diseases affecting the coronary arteries. The MI is mainly due to an acute or subacute primary reduction of myocardial oxygen supply provoked by the destruction of an atherosclerotic plaque associated with inflammation, thrombosis, vasoconstriction, and micro embolization. Myocardial infarction is the main pathophysiological characteristic. The biochemical marker of MI should have the properties like a considerable concentration in the myocardium, absence from non- myocardial tissue and normal serum, rapid release into the blood at the time of ischemia, relationship to extent of injury, and persistence in the blood for a sufficient length of time to provide a diagnostic window.

The core of the present study takes into consideration that measurement of serum cardiac marker AST and CK-MB levels might make a diagnosis of MI in patients with ongoing ischemic pain admitted to the emergency department. The serum levels of biomarkers are raised in MI without survival in contrast with MI with survival. These study data may prove that these changes might be useful to obtain a comprehensive view of the infarct size and severity of vascular stenotic lesions.

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TABLES

"Table -1 Biochemical changes in MI patients and Controls "

Variables	Controls(n= 50) Mean ± SD	MI patients(n=50) Mean ± SD
CK-MB (IU/L)	0.081 ± 0.15	56.78 ± 25.09*
AST (IU/L)	12.59 ± 3.96	52.91± 20.98*

* P value < 0.001

"Table-2. Biochemical changes in MI with survival and MI without survival"

Variables	MI with survival (n=44)	MI without survival (n=6)	P - value	% increased
CK-MB (IU/L)	56.03 ± 23.87	79.06 ± 30.07	P < 0.01 HS	41.10
AST (IU/L)	52.98 ± 20.25	60.99 ± 31.87	P < 0.01 HS	15.11