Introduction

Acute Myocardial infarction (AMI) or Myocardial infarction (MI) (ie, heart attack) is the irreversible death (necrosis) of heart muscle due to prolonged lack of oxygen supply (ischemia). Cardiovascular disease (CVD) is a major global cause of mortality in the developed countries. Approximately 1.5 million cases of MI occur annually in the United States [1].

Biochemical basis of Acute Myocardial Infarction.: The elevated level of LDL concentration in blood signifies the risk for coronary artery disease. Elevated level of LDL or modified LDL (glycated LDL) or oxidized LDL enhances the proportion of cholesterol taken up by macrophages. LDL infiltrates through arterial wall and are taken up by macrophages or scavenger cells [2]. Then the ruptured endothelial lining stimulates adhesion molecules and pro-inflammatory chemokines. These attract T lymphocytes as well as monocytes. Plaque is formed which consist an active inflammatory cell and large lipid core. This is the starting events for atherosclerosis which gradually leads to the formation of atherosclerotic plaque, risk factor for the development of acute myocardial infarction [3].

Cardiac biomarkers: The term ‘biomarker’ abbreviated for “biological-marker” is defined as “a characteristic that is objectively measured and evaluated as an indicator of normal biological processes or pharmacological responses to a therapeutic intervention” [4]. Chemically cardiac markers are protein components of cells and released into circulation when myocardial injury occurs [5].

Myoglobin: Myoglobin is a heme protein found in cytoplasm of all muscles cells. The level increases within 1 to 3 hrs after myocardial infarction reach peak elevation within 6 to 9 h, and may become normal in, 24 hrs [6]. Myoglobin is the earliest cardiac marker to rise after AMI because of high cytoplasmic content and comparatively smaller size [7]. Myoglobin has limited specificity for for the diagnosis of AMI in cases with skeletal muscle trauma and renal disease [8]. Myoglobin may lead to normal values for patients who present 24 hrs after symptom of AMI onset [9].

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ABSTRACT

Introduction: Acute Myocardial infarction (AMI) or Myocardial infarction (MI) (ie, heart attack) is the irreversible death (necrosis) of heart muscle due to prolonged lack of oxygen supply (ischemia). Biochemically the study of Cardiac markers like myoglobin, troponin, Creatine –phosphokinase (CPK), Serum glutamate oxaloacetate transaminase (SGOT), Lactate dehydrogenase (LDH) can used in the assessment of Myocardial infarction.

Materials and methods: Total 70 patients (50 MI patient and 20 control (not diagnosed as MI) were enrolled for the study and the biochemical investigations like Total CPK,CPK-MB, Troponin-T & I, LDH and SGOT were conducted for the study. Result and discussion: Each of the following biochemical parameters examined were significantly increases in case of AMI/MI patients (case) when compared with control. Troponin -T shows significant difference than other cardiac markers (p value for Troponin-T is <.001). Conclusion: Appropriate study of cardiac markers like CPK, LDH, SGOT, Troponin is significant for the study and evaluation of myocardial infarction. Troponin –T is most appropriate and significant than other cardiac markers mention in the study.
Cardiac Troponin (cTn): The most widely accepted biomarker for myocardial injury is cTn. The Troponin complex is made up of 3 subunits—C, I, and T—that combinedely control calcium mediated interaction of actin and myosin filament, leading to the relaxation and contraction of striated muscle or voluntary muscles. However, the skeletal and cardiac sub forms for TnI and TnT are distinct, and immunoassay have been designed to differentiate between them with no cross-reactivity [10]. cTnT and cTnI assays can detect cardiac muscle injury with great sensitivity and specificity. cTnT and cTnI appear in serum within 4–8 hrs after onset of symptoms of AMI and remain elevated for as long as 7–10 days post-MI [11]. Assays for cTnT and cTnI have asensitivity of up to 100% for myocardial damage within 4–6 hrs and 6 hrs respectively [12].

Creatine Kinase (CK-MB)/Creatine phosphokinase (CPK-MB): CK catalyses the transfer of phosphate group from ATP to creatine to form creatine phosphate. CK, which is also known as CPK has three isoenzymes each having two subunits M (muscles) and B (brain) and exist as CPK-BB,CPK-MB,CPK-MM. Increased plasma CK activity signifies skeletal muscle damage and it is not AMI-specific [13]. However CPK-MB (also called CK-2) comprises about 40% of the CK activity in cardiac muscle and 2% or less of the activity in most muscle groups and other tissues is the first enzyme to be releasd in to circulation within 6-8 hrs after the infarction, reaches peak value within 24-30 hrs and return to normal level by 2\textsuperscript{nd} or 3\textsuperscript{rd} day and serves as a more sensitive and specific marker for MI [14].

Serum glutamate oxaloacetate transaminase (SGOT)/ Aspartate transaminase (AST): SGOT, commonly known as Aspartate transaminase (AST) is both cytoplasmic as well as mitochondrial enzymes arises sharply after CPK, and reaches a peak within 48 hrs of myocardial infarction. AST takes 4-5 days to return to normal level and it is not cardiac specific because this enzyme is also found in liver cells and also used to assay liver function test [15].

Lactate dehydrogenase (LDH). : LDH catalyses the interconversion of lactate and pyruvate by NAD\textsuperscript{+} dependent reaction has five isoenzymes LDH\textsubscript{1},LDH\textsubscript{2},.....,LDH\textsubscript{5} and is tetrameric having two types of subunits namely M (for muscles) and H (for Heart).LDH\textsubscript{1} (having H\textsubscript{4} subunits) found in heart muscles. In case of myocardial infarction the activity of LDH\textsubscript{1} is much more greater .its activity rises in serum within 12-24 hrs after AMI, reaches to peak level after 48-72 hrs after AMI. That’s why considered as late marker [16].

Materials and methods

The study was carried out at Escorts Heart Institute and research centre LTD.(EHIRCL), Okhala Road, New Delhi, India in a clinical Biochemistry laboratory. The study was conducted from August 2009 to January 2010. The study includes the data of both the indoor and the out door patients. Total of 50 Myocardial Infarction (MI) cases diagnosed and 20 control (not diagnosed as MI) were studied. The blood sample of the MI patients for the following parameters were studied within few hrs after they had been admitted to ICU for the following parameters. Those parameters are Total CPK, CPK-MB, LDH, SGOT, Troponin-T and Troponin-I. All these parameter are cardiac markers. Other biochemical parameters of those patients were also there which has been excluded for the study. The reference range for all the parameters on which the study is based on are as follows. (I) Total CPK- <195 -U/L (II) CPK-MB- 1.90-25-U/L (III) LDH-240-480-U/L (IV) SGOT- <37 1.U/L. Immunoassay for the in vitro quantitative determination of Troponin-T based on electrochemiluminescence (ELECSYS) is performed. Solid phase chromatographic immunoassay technique is performed for the qualitative determination of Troponin-I. Enzymes activity is measured by end point essay technique.

Result

20 controls and 50 MI cases were considered. The mean age was 67 ± 9.93 (mean + standard deviation) in case of control and 62.66 ± 12.26 in case of MI patients. There were 17 males and 3 females in control. 41 males and 9 females were screened in abnormal cases (myocardial infarction). The blood investigation for cardiac markers are given below for both control and cases.

<table>
<thead>
<tr>
<th>S.NO</th>
<th>Variables</th>
<th>Variables</th>
<th>Mean ± Std. Deviation</th>
<th>N</th>
<th>Mean ± Std. Deviation</th>
<th>p-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Total CPK</td>
<td>20</td>
<td>148.8 ±279.1</td>
<td>50</td>
<td>648.84 806.7±</td>
<td>.002</td>
</tr>
<tr>
<td>2</td>
<td>CPK-MB</td>
<td>20</td>
<td>20.9± 9.58</td>
<td>50</td>
<td>89.4 ± 118.07</td>
<td>.002</td>
</tr>
<tr>
<td>3</td>
<td>LDH</td>
<td>20</td>
<td>510±199.4</td>
<td>50</td>
<td>836.6 ± 553.05</td>
<td>.002</td>
</tr>
<tr>
<td>4</td>
<td>SGOT</td>
<td>20</td>
<td>26.4±10.35</td>
<td>50</td>
<td>80.82 ± 100.03</td>
<td>.015</td>
</tr>
<tr>
<td>5</td>
<td>cTn-T</td>
<td>20</td>
<td>.01± &lt;.0001</td>
<td>50</td>
<td>1.66 ± 2.47</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>6</td>
<td>cTn- I</td>
<td>20</td>
<td>All -VE</td>
<td>50</td>
<td>16 + ve</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
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<td>34 -ve</td>
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</tr>
</tbody>
</table>

NOTE : CPK- Creatine phosphokinase, CPK-MB- Creatine phosphokinase- MB,LDH-Lactate dehydrogenase, SGOT-Serum glutamate oxaloacetate transaminase. cTn-cardiac Troponin.
Interpretation of the result for Troponin-I

- **Negative**- one red line appears in the control line region.
- **Positive**- two distinct red lines appear, one control and another line test line region.
- **Invalid**- lines or control line fail to appear.

![Fig: Positive result for Troponin I](image)

**Discussion**

Cardiac enzymes showed significant difference between controls and cases being higher in cases of myocardial infarction patients (cases). There is slight increase in the value of LDH in control patients. Total CPK was 148 ± 279.1 in control and 648 ± 806.7 in MI cases. CPK-MB was 20.9 ±9.58 in control and 89.4 ± 118.07 in MI cases. LDH was 510 ± 199.4 in control and 836.6 ± 553.1 in MI cases. SGOT was 26.4 ± 10.35 in control and 80.8 ±100.1 in MI cases. Troponin –T was <0.01 ± <.0001 in control and 1.7± 2.4 in MI cases. Also the Troponin –I is negative in all the control patients where as negative in 34 MI cases and positive in 16 MI cases. Study of above cardiac markers is significant in MI case. Out of 50 MI cases 41 is male and 9 is female. Though the cases are randomly selected , it shows that male are higher in risk than female for MI. According to the data the level of significance is very less for cTn-T (P <0.001) than other cardiac markers. So cTn –T may be considered as reliable cardiac markers for MI than other cardiac markers. Troponin- T is released in the serum earlier than other cardiac markers(4-6 hrs after MI) and remains for long time in the serum (up to 7 days ).So follow up of this parameter is also significantly diagnostic.

**Conclusion**

Hence from the above study it is found that appropriate study of cardiac markers like Total CPK, CPK-MB, LDH, SGOT, cTn-T, cTn-I is significant for the study and evaluation of myocardial infarction. Males are at higher risk than female for cardiac disease and also from my study it is found that cTn –T is the reliable markers . Troponin-T is the best cardiac marker for detection of Myocardial Infarction (MI).

**References**


