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# STUDY OF CARDIAC TROPONIN-I AS A DIAGNOSTIC MARKER IN COMPARISION WITH CREATINE KINASE-MB IN MYOCARDIAL INFARCTION

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## ABSTRACT

*BACKGROUND: Myocardial infarction is a medical emergency that most often caused by an exclusive coronary thrombus which results in prolonged myocardial ischemia and irreversible cell death. Early diagnosis of myocardial infarction is crucial in planning the treatment modalities, which if instituted in time reduces the morbidity and mortality considerably. OBJECTIVES: To evaluate the usefulness of cardiac troponin-I in the early diagnosis of myocardial infarction and in assesing the severity of the infraction. The study also compares cardiac troponin-I with cardiac marker creatine Kinase-MB. METHODS: The present study comprises of 100 subjects which includes 50 healthy controls and 50 subjects with myocardial infarction, in whom the following parameters were analysed on admission and after 24hours. The parameters are cardiac troponin- I (Chemiluminesence immunoassay method), creatine- kinase –MB (optimized DGKC method by semi auto ananlyzer). RESULTS: Cardiac troponin –I is better than creatine kinase –MB in diagnosing myocardial infarction .On admission the increase in serum cardiac troponin –I is highly significant in subjects with MI (p<0.001)when comparied to creatine kinase-MB .In our study the cutoff value of cardiac troponin –I is 0.60ng/ml by taking the mean of healthy controls .cardiac troponin-I level greater than 0.60ng/nl is used as positive value .The sensitivity and specificity of troponin-I is 96% and 98% respectively which is higher than creatine kinase-MB(62%and 68%)respectively ,within the first 6 hours of myocardial infarction .The diagnostic efficiency of cardiac troponin-I is 97%,and creatine kinase –MB is 63% in diagonosis of acute MI. CONCLUSION: Serum cardiac troponin-I is highly sensitive and specific cardiac marker which can substantially improve the early diagnosis of acute mycocardial infartion so that adequate treatment modalities can be initiated early in these patients to minimise the risk of death.The estimation of serum cardiac troponin-I level can be routinely employed to confirm or rule out acute myocardial infarction as a part of laboratory evaluation of acute myocardial infarction .*

**KEY WORDS**

*Cardiac troponin-I; Creatine kinase-MB; Myocardial infarction*

## INTRODUCTION

Cardiovascular diseases (CVD) comprise of a group of diseases of the heart and the vascular system which includes the major conditions like coronary heart disease (CHD), hypertension, congenital heart disease. Coronary heart disease is defined as “impairment of heart function due to inadequate blood flow to the heart compared to its needs, caused by obstructive changed in the coronary circulation to the heart”.

Coronary heart disease is now the leading cause of deaths in India accounting for 29% of all WHO has drawn attention to the fact that CHD is a mordern epidemic .CHD may manifest itself in many presentations such as myocardial infarction ,angina pectoris and sudden death ,of

which acute myocardial infarction is specific to CHD. [1] Acute myocardial infarction is the most dangerous manifestation of CHD.[2]

Myocardial infarction (MI) results from reduced coronary flow such that oxygen demand of the myocardium is not met resulting in irreversible cell death and necrosis. [3]

According to WHO criteria, diagnosis of MI is based on clinical symptoms ,ECG changes and characteristic changing pattern of serum cardiac markers .The complications after acute myocardial infarction are maximum in the first few hours .[4] The early detection and diagnosis of MI is vital for the institution of therapy to limit myocardial damage and preserve cardiac function.[5]

There is an urgent need for a rapid, sensitive and specific cardiac marker that can help clinicians to make early diagnosis of MI.Now the commercial avaliability of rapid, sensitive and cardiac specific troponin tests have revolutionized the cardiac biomarker utility in the differentiation of MI from other causes of chest pain. [6]

Troponin is a protien complex located on the thin filament of striated muscles having 3 subunits .Troponin –T(TnT),Troponin –I(TnI) and Troponin –C(TnC).cardiac troponin particularly troponin –I is the preferred marker for the diagnosis of MI.[6]

The present study is an attempt to elucidate the usefulness of troponin-I in the early detection of myocadial injury enabling adequate interventions to be given towards those who are likely to be benefited.

## MATERIALS AND METHOD

A study of serum cardia troponin-I, creatine kinase-MB activity in subjects of MI is carried out from May 2011 to May 2012. The study is carried out in subjects with myocardial infarction and healthy controls selected from Bapuji Hospital and Chigateri General Hospital, Davangere (both these hospitalsd are attached to the teaching institute, J J M Medical College, Davangere). Each subject gave an informed consent and this study was approved by the ethical and research committee of JJM Medical College, Davangere to use human subjects in the research study.

Based on the inclusion and exclusion criteria a total number of 100 subjects are selected for the present study which includes 50 cases with MI and 50 healthy controls.clinically proven cases of myocardial infarction in the age group of 30-80yrs, who are admitted to the cardiac ICU are included in the study. The diagnosis of myocardial infarction was confirmed by ECG changes.and controls are healthy age and sex matched individuals without any major illness and not on any medications. And myocardial infarction patients with cardiac trauma (cardioversion, pacing), hepatic disease, renal disease, critically ill, history of MI in past 1 year, severe sepsis, hypothyroidism and patients with angina, pericarditis and pulmonary embolus were excluded from study.

Under all aseptic precautions about 4ml of venous blood is collected in a sterile bulb after admission and also 24hours later. Serum is seperated after centrifugation and is used for the analysis. In the present study serum cardiac troponin-I and serum creatine kinase –MB were estimated.cardiac troponin-I is estimated using chemiluminesence immunoassay (CLIA) kit from Acculite in Lumax 4101 reader using principle of immunoenzymometric assay [7] with normal value in adults of < 1.3ng/ml, and creatine kinase-MB is analysed using kits from ERBA company in ERBA chem 5 semiautoanalyzer using immunoinhibition kinetic method.[8] with normal value of CK-MB < 25IU/L AT 37 $^{0}$ C.

## RESULTS

The results obtained in this present study are from the total number of 100 subjects. Out of

total 50 healthy controls, 36 were males and 14 females. And out of 50 cases 36 males and 14 females.serum troponin-I and creatine kinase – MB is estimated in all the subjects. Their results are shown in the following tables.

**Table 1: Age wise distribution of healthy controls and cases**

| | Controls | Cases |
|---|---|---|
| **No of Subjects** | 50 | 50 |
| **Age (yrs)**<br>**31-40** | 5 | 4 |
| **41-50** | 12 | 12 |
| **51-60** | 22 | 20 |
| **>60** | 11 | 14 |
| **Mean ± SD** | 52.7± 11.0 | 55.7 ±10.0 |
| **Range** | 30-75yrs | 38-75yrs |

**GRAPH 1: Age wise distribution of healthy controls and cases**

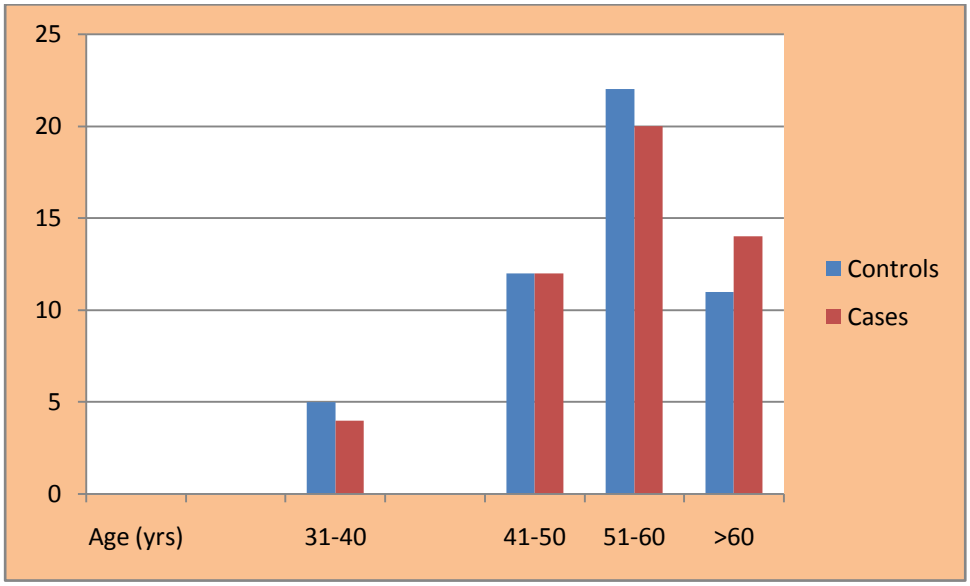


**Table 2: Serum levels of CK-MB and cTnI among healthy controls and MI cases on admission**

| Groups | n | | CK-MB (IU/L) | CTnI(ng/ml) |
|---|---|---|---|---|
| **Controls** | 50 | Mean± SD (Range) | 14.7±4.2<br>(7.5-22.4) | 0.60±0.26<br>(0.20-1.35) |
| **Cases** | 50 | Mean± SD (Range) | 17.5±4.2<br>(9.2- 25.2) | 3.16±1.02<br>(1.30-5.95) |
| **Mean diff.** | | | 2.8 | 2.56 |
| **t-value*** | | | 3.30 | 17.26 |
| **p-value** | | | <0.01, S | <0.001, HS |

***Unpaired t-test**

**Table 2** shows levels (mean ± SD and range) of serum CK-MB and cardiac troponin-I in healthy controls and subjects with acute myocardial infarction at the time of admission.

It is seen from the table that the estimated levels (mean ± SD) of serum CK-MB and Cardiac troponin-I in healthy controls were in the range of 14.7±4.2 IU/L and 0.60±0.26ng/ml respectively.In MI patients on admission, the mean value of serum CK-MB and cardiac troponin-I were in the range of 17.5±4.2 IU/L and 3.16±1.02ng/ml respectively.

The statistical analysis by unpaired t-test shows that cardiac troponin-I level is increased in patients with MI on admission when compared to healthy controls and it is statistically highly significant (p<0.001).

The serum CK-MB level is slightly increased in patients with MI on admission and is statistically significant (p<0.01).

**Table 3: Serum level of CK-MB and cTnI on admission and after 24 hrs amongs MI cases**

| Parameters | On admission Mean ± SD | After 24 hrs Mean ± SD | Difference | t-value* | p-level |
|---|---|---|---|---|---|
| CK-MB (IU/L) | 17.5±4.2 | 147.0±37.1 | 129.5 | 24.54 | <0.001,HS |
| CTnI(ng/ml) | 3.16±1.02 | 16.50±3.38 | 13.34 | 31.65 | <0.001,HS |

***Paired t-test**

**Table 3** shows the level (mean SD) of serum CK-MB and Cardiac troponin-I levels on admission and after 24 hours in MI patients.

On admission serum levels of CK-MB and Cardiac troponin-I levels were found to be 17.5±4.2 IU/L and 3.16 ±1.02ng/ml respectively.

After 24hours the levels (mean ±SD) of serum CK-MB and cardiac troponin-I levels were in the range of 147.0±37.1 IU/L and 16.50±3.38 ng/ml respectively.

The statistical analysis by paired t-test shows that the difference between th e values of serum CK-MB and Cardiac troponin-I levels in subjects with MI on admission and after 24 hours is 129.5 and 13.34 respectively. It shows that CK-MB and Cardiac troponin-I levels are significantly increased after 24 hours which is statistically significant (p<0.001).

**Table 4: Diagnostic value of cTnI and CK-MB in the diagnosis of AMI**

| Markers Cut-off value | CK-MB (>16.0 IU/L) | CtnI (>1.3ng/ml) |
|---|---|---|
| Sensitivity | 62% | 96% |
| Specificity | 64% | 98% |
| PPV | 63% | 98% |
| NPV | 63% | 96% |
| Diagnostic efficiency | 63% | 97% |

**GRAPH 2: Diagnostic value of cTnI and CK-MB in the diagnosis of AMI**

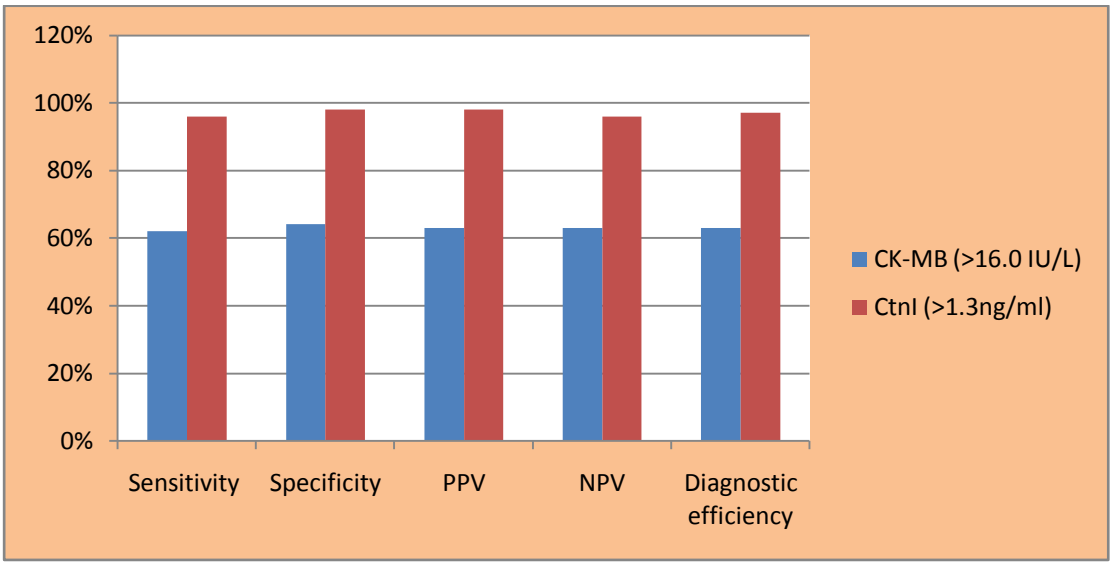


**Table 4 and graph 2** shows the diagnostic utility of cardiac biomarkers (serum CK-MB and C ardiac troponin-I) in the diagnosis of MI in patients admitted to the emergency department within 3 to 6 hours after onset of chest pain. Median of the combined groups is used as the cutoff value for the serum CK-MB and Cardiac troponin-I for the diagnosis of MI.It is evident that cardiac troponin-I is the most sensitive and specific cardiac biomarker with highest diagnostic utility in the early diagnosis of myocardial infarction.

**Table 5: comparision of CK-MB and cTnI between MI cases on admission and the standard reference value**

| Parameter | Normal reference value | On admission (mean ±SD) |
|---|---|---|
| **CK-MB(IU/L)** | 7.0-24.0 | 17.5±4.2 |
| **cTnI(ng/ml)** | 0.20-1.35 | 3.16±1.02 |

**Table 5** shows the comparative analysis of serum levels of CK-MB and Cardiac troponin-I in patients with MI on admission are 17.5±4.2 and 3.16±1.02ng/ml respectively.

The table shows the normal reference value of serum CK-MB and cardiac troponin-I as 7.0-24.0IU/L and 0.20-1.35ng/ml respectively.

It is evident that cardiac troponin-I is elevated well above the normal reference value in patients with MI on admission to the emergency department. Serum CK-MB levels are within the normal reference range in patients with MI on admission.

## DISCUSSION

Myocardial Infarction is the most serious complication of coronary artery disease which occurs due to the perfusion imbalance between supply and demand within the coronary arteries as a result of an acute thrombotic process. [5],[9].

The diagnosis of myocardial infarction has been clasically based on a history of chest pain and echocardiographic documentation of new Q waves and evolving ST-T wave changes. It has been demonstrated that 4-8% of patients with MI may be missed, as some patients have atypical symptomsor suffer from “silent MI” .A

significant number of false positive and false negative results have been reported on ECG. [10] As a result, clinical laboratories have become increasingly relied upon either to establish or rule out the diagnosis of acute myocardial infarction.[11]

Appropriate interventions like thrombolytic therapy and primary percutaneous angioplasty can limit the myocardial necrosis thus limiting myocardial damage which will improve the prognosis in patients with MI. There is a considerable difference in the outcum if these treatments can be administered early in the course of the illness.[6]

According to the WHO, the diagnosis of MI requires the presence of atleast two of the following three criteria: (1) a history of ischemia type of chest pain (2) evolutionary changes on serial electrocardiograms (3) rise and fall in the serum cardiac markers. Today the cornerstone of these diagnostic criteria is the evaluation of serial cardiac markers.[12]

The present study is conducted to determine the diagnostic performance of cardiac troponin-I for early diagnosis of acute MI in patients at the time of presentation to the emergency department.

In this study the estimated levels (mean±SD) of serum creatine kinase-MB in healthy controls is and 14.7±4.2 IU/L in MI patients on admission it is 17.5± 4.2 IU/L respectively.

In patients with MI, on admission the serum creatine kinase –MB level is 17.5±4.2 and after 24hours the level is 147.0±37.1 IU/L respectively. The statistical analysis by unpaired t-test shows that serum CK-MB level is slightly increased in patients with MI on admission is statistically significant ($p< 0.01$)

The statistical analysis by paired test shows that the difference between the values of serum CK-MB in subjects with MI on admission and after 24 hours is 129.5. It shows that serum CK-MB level is significantly increased after 24 hours which is highly signiificant (<0.001).

This is in accordance with Jesse E Admas et al [13],Wille Gerhaldt [14] , John Griffiths [15] , and John H Alexendaer et al [16].

Serum creatine-kinase activity starts to rise within 6-8 hours arter an acute attack of MI. It reaches a peak of two to ten folds in 24 hours and decline to the range within 3 to 4 days following the onset of chest pain.[2],[3],[17].

Serum CK-MB elevation following myocardial ischemia is indicative of myocardial necrosis. It is a reflection of enzymes release from the myocardial cells undergoing necrosis. The relationship between changes in the serum enzymes activity and infarct size appears to be quantitative. Thus analysis of changes in the serum CK-MB activity following MI helps in the accurate assessment of the extent of myocardial necrosis and its progression.[18],[19],[20].

Complements of CK-MB are found in skeletal muscle, brain and thyroid. So CK-MB is markedly increased in most patients with muscular dystrophy, inflammatory disease of muscle, alcohol intoxication and intramuscular injections because of the enzyme release from the muscle. The B-chain subunit of creatine kinase is predominantly present in the skeletal muscle during fetal and neonatal development. Expression of this B-chain is suppressed in adult skeletal muscle. After skeletal injury there is increased synthesis of this B-chain subunit by re-expression of previously suppressed B-subunit gene. it is also found increased in myxedema due to diminished catabolism of circulating enzyme.[19],[20],[21].

In this study the estimated levels (mean ±SD) of serum cardiac troponin-I in healthy controls is 0.60±0.26 ng/ml and in MI patients on admission is 3.16 ±1.02 ng/ml respectively.

The statistical analysis by un-paired t-test shows that cardiac troponin –I level is increased in

patients with MI on admission when compared to healthy controls and is statistically highly significant ($p<0.001$).

In patients with MI, on admission the serum cardiac troponin-I level is 3.16± 1.02ng/ml and after 24hrs the level is 16.50±3.38 ng/ml respectively. The statistical analysis by paired t-test shows that the difference between the values of serum cardiac troponin –I levels in subjects with MI on admission and after 24 hours is 13.34. it shows that serum cardiac troponin -I levels is significantly increased after 24 hours whch is statistically highly significant ( $p< 0.001$). This is in accordance with the study of Kristin Newby et al [22], Daylily S et al [23], Jan Ravkilde et al [24]. The troponin complex (troponin-T, troponin-I, and troponin-C) along with tropomyosin is located on the actin filament and is essential for calcium for the calcium mediated regulation of skeletal and cardiac muscle contraction. Troponin –I is a regulatory protein that binds to actin and inhibits calcium-mediated actin-myosin interaction. [2], [3], [25].

Cardiac troponin-I has unique amino acid sequence that is produced only in myocardium throughout development. It is a regulatory protein with a high specificity for cardiac injury. Cardiac troponin-T is expressed in the skeletal muscle during fetal development. Troponin-I is not found in skeletal muscle during neonatal develpoment or during adulthood.

Thus cardiac troponin-I does not rise in skeletal muscle disease or injury where as cardiac troponin-T will be elevated in these conditions. [2],[3],[5].Cardiac troponin-I is tightly complexed to the contractile apparatus. It forms complexes with troponin-C and is released into the blood following MI predomintaly as a binary complex. Circulating levels are normally low , but they rise relatively rapidly after acute MI suggesting the pressure of troponins in the cytosol of the myocyte. The normal level of serum cTnI is 1.0-1.3ng/ml due to yhe continuous microscopic loss of cardiomyocytes during normal life.[5]

Troponin starts to rise within 4-6 hours of onset of myocardial necrosis because of rapid leak of the cytosolic pool (3% of troponin-I) of troponin-i. In any cardiac myocyte injury , this unbound pool of troponin-I os realeased first . thius is followed by slow release and degradation of the troponin bound to myofibrils (96% of the troponin-I). Troponin level peaks at 18-24 hours and declines over 10-14 days due to the continuous leashing of troponin-I from yhe neccrotic cell. The half life of troponin is 2 hours.[25]

Studies have shown that patients with positive troponin-I marker at the time of admission have an increased risk of re-infarction and that recurrent events are associated with increased risk of death. The mortality risk appears to co-relate with the level of troponin rise. These events may represent evidence of a refractorey pathophysiological process that progresses despite ongoing medical therapy. Our study is in accordance with the study of John F Tucker et al[26], Judd E Hollander[27] . To summarize an initial troponin-I determination drawn at the time of the patients presentation is a powerful diagnostic tool for a rapid diagnosis rather than serial CK-MB determination. This reflects the greater sensitivity and specificity of troponin-I for minor myocardial necrosis resulting from microembolization from active plaques, which predispose to subsequent major clinical events, but those activities may not be detectable by less sensitive markers like CK-MB until a large event occurs. [23]

## CONCLUSION

Acute myocardial infarction is the major cause of death and premature disability in the developing society .Serum cardiac biomarker testing is now the cornerstone in the diagnosis of MI.An ideal

Serum cardiac marker should have high sensitivity and high specificity so that the diagnosis of MI is not missed.

The present study found a statistically highly significant increase in cardiac troponin-I levels in subjects with MI at the time of admission to the cardiac ICU when compared to CK-MB,.cardiac troponin-I showed a high diagnostic efficiency of 97%,with a very sensitivity(96%) and specificity(98%) and positive predictive value(96%).It is the earliest marker for confirmation and exclusion of acute MI which is detected as early as 3 hours after the infarction.

The results of the study found that cardiac troponin-I has excellent sensitivity and specificity and it is superior to creatine kinase-MB as an indicator of myocardial ischemia. The routine use of cardiac troponin-I in the evaluation of patients with suspected MI, can eliminate the estimation of CK-MB in the diagnosis of MI. A single estimation of cardiac troponin-I can make the diagnosis of MI accurately rather than waiting for serial changes of serum cardiac markers.

The estimation of serum cardiac troponin-I should be made as an essential part of the evaluation in all subjects with signs and symptoms of chest pain. The combined use of cardiac troponin-I along with ECG and clinical history of chest pain will help in early and accurate diagnosis of MI leading to early diagnosis, initiation of treatment modalities thus resulting in better prognosis of patients with chest pain.

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