Association of Creatine kinase (MB) and troponin (I) with electrocardiographic changes, in acute myocardial infarction

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Abstract

72 (Seventy two) patients with complaints of chest pain and discomfort were clinically examined and evaluated by using ECG, Creatine kinase MB and cardiac Troponin I. Of these, 61 patients were had diagnosed to have acute myocardial ınfarction. 64% of them had elevated Creatine kinase MB whereas only 23% showed elevated Troponin I. Within the first six hours, Creatine kinase MB was elevated in 21% patients in contrast to 13% with elevated Troponin I. Elevated Creatine kinase MB was significantly associated with ECG changes ($p<0.005$)while Troponin I was not. Although, Troponin I was found to be 100% specific for Acute Myocardial Infarction, Creatine kinase MB was more sensitive. Diagnostic efficacy of Creatine kinase MB was also found to be more than Troponin I.

Key words: Chest pain, ECG, creatine kinase MB, troponin I, myocardial infarction

Introduction

Electrocardiogram is the diagnostic tool of first choice in acute myocardial infarction (AMI). However; it has a sensitivity of only 55-75 % [1]. In view of this, the diagnosis of AMI depends largely on cardiac markers in blood such as Creatine kinase-MB isoenzyme (CK-MB), Creatine kinase-total, troponins and myoglobin.

Reports received in recent years suggest that troponin is superior to CK-MB as the diagnostic marker for AMI the contrary, many clinical studies and statistical analyses report that CK-MB is the most appropriate test for diagnosis of AMI [3].

The present study aims at finding a possible association of cardiac troponin I (cTn I) positivity and raised CK-MB levels with ECG changes suggestive of AMI. Furthermore,

the study focuses on the question as to whether CK-MB or cTn I is a better indicator especially during the early hours, following an episode of AMI.

Materials and Methods

The present study is retrospective and observational investigation. The study population consisted of 72 patients who presented with chest pain and or discomfort during the period of study (Jan -Dec 2007) in our hospital.

A clinical evaluation followed by ECG, CK-MB and cTnI was done in all cases. Out of them, 61 cases were found to have AMI and depicted suggestive changes in ECG. Out of these 61 cases, 14 cases that had positive cTnI and showed elevated CK-MB levels, comprised group I. Group II had 25 cases, who were cTnI negative, but had high CK-MB levels. Group III comprised of 22 cases, that were cTnI negative and had normal levels of CK-MB. Ten cases were diagnosed to have non-cardiac causes of chest pain like gastro oesophageal reflux disease and were taken as controls (group IV), while one case had increased CK-MB levels but ECG was normal and cTn I tested negative, this comprised group V. Hence, the 72 cases were grouped as shown in Table 1.

Serum CK-MB levels were quantitated on fully automated random access chemistry analyzer and as per the International Federation of Clinical Chemistry (IFCC) recommended procedure. cTnI was detected in serum by the rapid assay (Single step membrane based qualitative immunoassay). Statistical analyses of the results were performed by using students' 't' test and chi-square test.

Results and Discussion

Finding an ideal and an unambiguous cardiac biomarker for evaluation of AMI has ever been a topic of active research and controversy. When new diagnostic tests are compared against the gold standard CK-MB, they were found to have decreased diagnostic efficiency, although they are more cardiospecific [3].

In this study, 64% of patients of AMI (as confirmed by ECG) had elevated levels of CK-MB while only 23% showed positive cTnI. Also, CK-MB was found to be elevated in 21.3% of patients within 6 hours and in 29% within 12 hours, whereas, cTnI was positive in only 13% and 18% within 6 hours and 12 hours respectively (Table 2).

Hence, according to this study, CK-MB appears to be a better indicator of AMI as compared to cTnI especially within the first few hours of AMI. The results are in agreement with an earlier study conducted by Collinson et al. [1] where during the initial 12 hours, CK-MB was a better indicator of AMI as compared to cardiac Troponin T. This assumes clinical significance since thrombolysis could be attempted. It was found by Wu et.al. that cTnT had clinical sensitivity of 63% only in the first 6 hours of chest pain which is insufficient for effective early diagnosis [4].

When the mean of CK-MB levels were compared between group I (117 IU/L ±85) and II (48 IU/L ±17) it was observed that in group I, CK-MB levels were significantly higher as compared to group II ($p<0.05$). CK-MB levels are known to correlate with the extent of myocardial damage. It is possible that group I patients had a greater myocardial damage as compared to those in group II. Quantitative determination of cTnI levels would have possibly thrown more light on this finding. However, this was not within the purview of this study.

Table 1: The study groups based on cardiac biomarkers and ECG findings

Groups	CK-MB	cTn I	ECG
I (n=14)	+ve	+ve	AMI
II (n=25)	+ve	-ve	AMI
III (n=22)	-ve	-ve	AMI
IV (n=10)	-ve	-ve	Normal
V (n=1)	+ve	-ve	Normal

Total number of cases, n=72

Table 2: Time dependent relevance of levels of positive cardiac biomarkers following an episode of AMI.

Time Parameter	0-6 hours	6-12 hours	0-12 hours
CK (MB)	13	5	18
cTn (I)	8	3	11

Table 3: Comparison of diagnostic indices of AMI

Cardiac Marker		
Diagnostic Indices	**cTnI**	**CK-MB**
Sensitivity	11%	63%
Specificity	100%	90.9%
Diagnostic efficiency	33.3%	68%
χ^2 value	2.86	7.009
Df	1	1

p value	>0.05	<0.01

Among group I patients, 14.3% mortality was observed (cTnI positive cases).These results demonstrate that elevated cTn levels are predictive of poor outcomes in patients with acute coronary syndrome and are in agreement with an earlier report [5].

On the contrary, the group III patients showed a mortality of 4.5%, though they had normal levels of CK-MB and tested negative for cTnI. This proves that negative cardiac markers do not necessarily predict better outcome for patients of AMI. This is corroborated by an earlier study performed by Mc Erlean et al. [6], which states that a negative cardiac marker value does not essentially confer low risk of complications in patients presenting with acute chest pain.

Also in this study, elevated levels of CK-MB were found to be associated with ECG changes which was statistically significant ($p<0.05$). No such statistical significance was observed in the association between positive cTnI and ECG changes (Table 3).

In this study, cTnI was found to be 100% specific while CK-MB was a more sensitive marker in diagnosis of AMI (Table 3).Diagnostic efficiency of CK-MB was found to be 68% whereas that of cTnI was only 33.3%. In conclusion, it was observed that CK-MB is a more sensitive marker and has better diagnostic efficiency for diagnosis of AMI as compared to cTnI, especially during the initial hours following an episode of AMI. Association of CK-MB and cTnI in acute myocardial infarction.

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