DIAGNOSIS OF ACUTE MYOCARDIAL INFARCTION: CK- MB VERSUS cTn-T IN INDIAN PATIENTS

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ABSTRACT

The comparative diagnostic efficacy of two cardiac markers : CK-MB and cTn-T , has scarcely been investigated in Indian patients of acute myocardial infarction. The present study was conducted for the same objective. The present study comprised of 59 patients . Males were 44 (75%) and females were 15 (25 %). The age of patients ranged from 32-84 years with mean age of 62.8 yrs. The mean age of males and females were 60 and 63 yrs respectively. All patients presented with history of chest pain with a 12 leads ECG proven MI (ST Elevation, discordant T- waves). CK-MB was estimated in peripheral blood samples at 0,24,48 and 72 hours by an autoanalyzer. Following 12 hours of admission bed side Troponin –T test was done employing cTn-T marker kit. Initially (0 hr), in 50% patients CK-MB was elevated. By end of 24 hours all the patients were CK-MB positive and peak level was attained at 24 hrs. Then it tended to decline over next 48 hrs. There were no false positive or negative results. The cTn-T test was positive only in 22 % of ECG positive infarctions. However, the cTn-T positive cases were always accompanied by a higher CK-MB levels. A significantly lower cTn-T positive cases in Indian patients can only be attributed to some difference in amino acid sequence of Indian cTn-T and occidental cTn-T. A larger study from other Indian cardiac centers can either substantiate or contradict our results.

KEY WORDS

Acute Myocardial Infarction, CK-MB, cTn-T

INTRODUCTION

Ischemic heart disease (IHD) culminating in myocardial infarction happens to be prime killer in Western Countries (1). People of India and Indian subcontinent are nearly 4 times more prone to IHD as compared to other countries due to genetic and metabolic differences (2-5). Early diagnosis and management is therefore, imperative to prevent mortality and morbidity in these patients . Most of diagnostic investigations pivot around hypoxia induced electrical alteration in the heart i.e. electrocardiogram(ECG) and structural damage of cardiac myocytes and its sub cellular organelles with massive efflux of muscle enzymes viz creatine kinase (CK), Aspartate transaminase(AST), Lactate dehydrogenase (LDH) and

Creatine kinase MB isoform (CK-MB). Late degradation of myofibrillar proteins myosin light chain (9,10,11) and regulatory proteins troponin T and troponin I (4 – 24 hours) was demonstrated (12) and was attributed to action of Lysosomal enzymes Cathepsin D (13) and Cathepsin B (14). However, Srivastava et al (15,16) observed degradation of myofibrillar proteins as early as 30 to 60 min of total invitro ischemia in dog heart and troponin T and alpha actinin were the chief candidates. Subsequently, compartmentisation of cTnT within myocytes was done (17) and monoclonal antibodies against cTnT were developed (18). This led to assay technique for detection of cTnT (18) in peripheral blood following acute myocardial infarction. Currently, CK-MB and cTnT are two major biochemical tools for early diagnosis of acute myocardial infarction and the present study was undertaken to test their relative diagnostic efficacy in myocardial infarction patients.

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MATERIALS AND METHODS

The present study comprised of 59 patients admitted to intensive coronary care unit of SRN Hospital and Heart Line

cardiac care center, Allahabad. Males were 44 (75%) and females were 15 (25%). The age ranged from 32-84 years with mean age of 62.8 years. The mean age of males and females were 60 and 63 years respectively. All patients presented with history of chest pain and clinical diagnosis of acute myocardial infarction was confirmed by 12 leads ECG exhibiting ST elevation and discordant T waves. CK-MB was estimated at different time points (Table 1), using Enzo pak reagent Kit and Auto mod AMES technicon (Bayer's RA 50) chem semi Autoanalyzer. The cut off point for the CK-MB was $5\mu\text{g/L}$.

Keeping in view the kinetics of cTnT that negative test before 8 hours of onset of symptoms does not exclude myocardial infarction (17), its appearance in patient peripheral blood was screened after 12 hours of onset of pain employing cTnT marker kit (Boehringer Mannheim). This generation of kit had a cut off reference level of $0.3\text{-}0.2\mu\text{g/L}$.

RESULTS AND DISCUSSION

CK-MB levels were found to be elevated in initial samples in 50 % of cases where as after 24 hours CKMB levels of all patients were found to be more than normal, peak CK-MB level was attained at 24 hours after the onset of chest pain in most of the patients. The serum level showed a gradual fall after that and after 72 hours of onset of chest pain only 10% of patients showed a raised CK-MB level.

Antman and Braunwald (19) have emphasized that clinicians should not rely on single measurement of CK-MB instead should evaluate the temporal rise and fall of serially obtained values as done in the present study. Muscle released CK-MB remains elevated for longer time than myocardial release of CK-MB and thus produces a plateau pattern over several days in contrast to short time course of myocardial CK-MB elevation (19), we have also observed the same. As reported by others

Table 2 : Cardiac Troponin - T Test in ECG positive Acute Myocardial Infarction Patients.

S.No.	Test	No. of Cases	Percentage
1	Positive	13	22.03
2	Negative	46	77.97

Table 3 : Comparative CK-MB and Troponin -T Estimation in ECG positive Acute Myocardial Infarction Patients.

S.N.	Troponin Test	Mean CK-MB Levels	SD
1	Positive	$140.7\mu\text{g/L}$	113.5
2	Negative	$93.2\mu\text{g/L}$	101.3

there were no false positive and false negative cases detected in the present study (19). The association of cTnT positive cases with significantly higher CK-MB as compared to negative cTnT (Table 3) predisposes the patient to greater risk of serious outcome (19). However, longer follow up was not undertaken in the present investigation.

Since cTnT is also distributed in myocyte's cytosol (6%) few investigators have expressed skepticism whether serum rise of cTnT is due to leakage from cytosolic pool or damage to myofibrillar troponin units. However, Srivastava et al (15, 16) on the basis of their gel electrophoretic studies of myofibrils isolated from dog hearts within 30 to 60 minutes of total invitro ischemia suggested that Troponin T and /or alpha actinin presumably get degraded. In such a situation by 3-12 hours enough troponin units will be degraded to raise cTnT level in serum for positive cTnT test (19).

Contrary to others opinion that cTnT may be more sensitive to detect minor infarction which could not be detected by CK-MB (20,21), our study reveals only 22% cTnT positive cases while it was over 46% (22) and 90 % (23) in other studies; Although cTn-T positive cases had higher CK-MB (Table 3).

Table 1 : Serum CK-MB Estimation in ECG positive Acute Myocardial Infarction Patients

S.N.	Serum CK-MB Levels	Initial Sample		24 Hour		48 Hour		72 Hour	
		No. of Cases	%	No. of Cases	%	No. of Cases	%	No. of Cases	%
1	0-25	29	49.1	0	0.0	7	11.8	53	89.8
2	26-75	22	37.2	26	44.1	39	66.1	4	6.7
3	76-125	7	11.8	21	35.60	11	18.6	1	1.6
4	126-175	1	1.6	3	5.08	1	1.6	1	1.6
5	176-225	0	00	2	3.39	0	00	0	00
6	>226	00	0.00	4	6.78	1	1.6	00	00
Mean		29.4 $\mu\text{g/L}$		100.5 $\mu\text{g/L}$		68.3 $\mu\text{g/L}$		26.0 $\mu\text{g/L}$	

The reason for only 22 % positive cTnT in our study is difficult to explain. However, a wild speculation could be that there may be some difference in aminoacid constitution of Indian cTnT in comparison to cTnT in Western population, as it is seen in haemoglobin S and haemoglobin A among blacks and non blacks respectively (24). A larger study from other Indian cardiac centers can either substantiate or contradict our comparatively more negative cTnT findings.

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