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Original Research Article

Assessment of serum cardiac troponin-I over serum CPK-MB in early diagnosis of acute myocardial infarction (AMI)

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ABSTRACT

Background: The early mortality rate from AMI is 30% with about half of them occurring within 1hour of disability. Although the mortality rate after admission for AMI has declined by 30% over the past decades, approximately 1 of every 25 patients who survive the initial hospitalization die in the first year after AMI. The gold standard for diagnosis of MI has been an elevated serum level of creatinine kinase – myocardial band (CK- MB), the cardiac-specific isoenzyme of CK. However, elevated CK-MB may not detect all myocardial necrosis. In patients who die suddenly after severe or silent episodes of ischemia, autopsies frequently reveal micronecrosis that was not reflected in routine CK-MB measurements. The present study was undertaken to know that serum Cardiac Troponin-I is more sensitive marker than serum CPK-MB in early diagnosis of acute myocardial infarction (AMI).
Methods: The study was carried out in tertiary care hospital in Gulbarga. The study was undertaken with an aim to study that serum cardiac troponin-I (cTnl) is more sensitive than serum CK-MB in early diagnosis of acute myocardial infarction (AMI). The study was conducted on patients admitted with history of chest pain suggestive of AMI as diagnosed by WHO criteria to medicine ward of Basaveshwar Teaching and General Hospital, Gulbarga. The period of study was from June 2012 to June 2014. The sample size included 100 patients with history of chest pain suggestive of AMI, selected by simple random method.
Results: Our results revealed that cardiac troponin I was more sensitive (62%) than CK-MB in overall cases admitted in between 6-24 hrs from the onset of chest pain. Maximum number (41%) of AMI patients were affected on the anterior wall followed by Inferior wall of AMI. 11 percent were affected with Antero lateral wall wereas 5 to 6 percent were affected with anteroseptal and global acute and right ventricular AMI was seen among 2 percent of patients. Anterior wall AMI was the significantly affected site with AMI (χ^2:12.5, P:0.0004). The maximum number of acute myocardial infarctions were ST elevation myocardial infarctions. 28% of cases where CKMB is normal, the cTnI detects the AMI cases indicating its sensitivity.
Conclusions: Cardiac troponin-I (cTnI) was more sensitive serum marker than CKMB in the early diagnosis of acute myocardial infarction (AMI). Anterior wall was the most significantly affected site of AMI. In the future, further improvements in analytical performance may open additional diagnostic windows.

Keywords: Acute myocardial infarction, Serum cardiac troponin-I, Serum CPK-MB

INTRODUCTION

Cardiovascular diseases are at present the leading causes of death in the developed countries. Coronary artery disease is the cause of 25-30% of deaths in most industrialized countries.[1] In India, there were approximately 29.8 million patients with cardiovascular disease by the year 2003, 1.5 million people die every year. The burden of Ischemic heart disease was about 2.4 million every year.[1] Compared with all other countries,

India suffers the highest loss in potentially productive years of life, due to deaths from cardiovascular disease in people with age 35-64 years (9.2 million years lost in 2000).[1] In hospital admitted patients, diagnosis of AMI according to WHO criteria was confirmed in only 30-50% cases. ECG changes are seen only in about ½ of AMI cases on presentation. Approximately ¼th of patients with AMI do not present with classic chest pain and the event would go unrecognized.[1]

The gold standard for diagnosis of MI has been an elevated serum level of creatinine kinase-myocardial band (CK- MB), the cardiac-specific isoenzyme of CK.[2] However, elevated CK-MB may not detect all myocardial necrosis. In patients who die suddenly after severe or silent episodes of ischemia, autopsies frequently reveal micro necrosis that was not reflected in routine CK-MB measurements.[2] In addition, myocardial biopsies taken during coronary artery bypass surgery in patients with unstable angina have shown platelet aggregates in the microvasculature, with associated myocardial necrosis, but without serum CK-MB elevation.[2] New cardiac marker, the Troponin-I with superior sensitivity and specificity for myocardial damage and greater ability to risk stratify patients with ischemic myocardial necrosis is now challenging the role of CK-MB.

Monoclonal antibody-based assays have been developed that are specific for the cardiac isoform of troponin-I[2]. Using such assays, data are now available confirming that troponins (I) can identify myocardial micronecrosis even when an AMI diagnosis has been excluded – according to the conventional definition.[2] In response to these and other issues, a new definition of MI was proposed which emphasizes the use of cardiac troponin (I) as the preferred marker of myocardial necrosis in the context of ischemic symptoms in routine clinical practice.[2]

The present study was undertaken to know that serum Cardiac Troponin-I is more sensitive marker than serum CPK-MB in early diagnosis of acute myocardial infarction (AMI).

METHODS

The study was carried out in tertiary care hospital in Gulbarga. The study was undertaken with an aim to study that serum cardiac troponin-I (cTnl) is more sensitive than serum CK-MB in early diagnosis of acute myocardial infarction (AMI).

Selection of Patients: The study was conducted on patients admitted with history of chest pain suggestive of AMI as diagnosed by WHO criteria to medicine ward of Basaveshwar Teaching and General Hospital, Kalaburagi.

This was a hospital based cross sectional study carried out from June 2012 to June 2014. The sample size included patients attending Basaveshwar Teaching and General Hospital, Kalaburagi with history of chest pain suggestive of AMI, selected by simple random method. Of these, 100 patients were diagnosed as having AMI by using WHO criteria after exclusion of patients having acute pericarditis, severe heart failure, acute myocarditis, cardiac trauma, skeletal muscle disease and/or trauma, chronic renal failure.

Inclusion criteria

Patients attending Tertiary care hospital during the study period with proved acute myocardial infarction with history of chest pain suggestive of acute myocardial infarction studied.

Exclusion criteria

Patients with Acute pericarditis, Severe heart failure, Acute myocarditis, Cardiac trauma, Chronic renal failure, Skeletal muscle disease and or trauma, Hematologic malignancies and Cerebrovascular accident were excluded from the study.

Study protocol

Clinical history was taken either from patient or his/ her relatives or attendant after obtaining informed consent. While taking history importance was given regarding chest pain; its site, nature, duration, radiation, relation with exertion, sweating and vomiting, gastrointestinal system and central nervous system were examined in detail. Detailed investigations including blood hemoglobin, TC, DC, ESR, FBS, fasting lipid profile, Blood urea, serum creatinine, ECG, chest X-ray, serum CK-MB and serum cTnI were done.

Ethical clearance has been obtained from Institutional ethical committee of M. R. Medical college, Kalaburagi.

Method of study

All the 100 patients coming to ICCU with history of chest pain suggestive of acute myocardial infarction were subjected for the following investigations at the time of admission or 6 hours whichever is later within 24 hours of chest pain symptom. Acute myocardial infarction was confirmed by utilizing WHO criteria as follows: 1. Serial ECG changes: Q-wave or QS complex indicates myocardial necrosis.2. Serum CK-MB: ST segment elevation indicates myocardial injury 3. Serum cTnI.: T-wave inversion indicates myocardial ischemia. ST elevation indicates transmural infarction.

ECG Changes: AMI results in myocardial necrosis, injury and ischemia, each of which is reflected by a different and distinctive ECG manifestation. ST depression indicates subendocardial infarction.

From a clinical viewpoint, the division of AMI into ST segment elevation and non ST-elevation types is useful

since the efficacy of acute reperfusion therapy is limited to the former group.

Serial ECG tracings were studied because single ECG was not diagnostic in many cases.

Sernm CK-MB

2 ml of plain blood of the patients with history of chest pain at the time of admission was collected and sent immediately to the Basaveshwar Teaching and General Hospital Laboratory for estimation at room temperature.

B-subunit activity of CK-MB and CK-BB. Then we used CK method to determine the CK-B activity. The CK-MB activity was obtained by multiplying the CK-B activity by two. The estimation of the CK-MB was done by using immuno-inhibition methodology, the principle of which includes as follows:

Principle: This procedure involves measurement of CK-activity in the presence of antibody to CK-M monomer.

Cardiac Troponin-I (cTnI): Fresh, unhemolysed serum of the chest pain patients coming to ICCU

Department at the time of admission was used.Each kit contains the following: Troponin-I test unit and Plastic dropper. The troponin-I kit was stored at room temperature (below 3 0°C). It was not allowed to freeze.

Principle: The biocard troponin-I is a rapid immune-chromatographic qualitative test. The method employes a unique combination of monoclonal dye conjugate and polyclonal solid phase antibodies to identify troponin in the test samples with a high degree of sensitivity.

Procedure: Remove a testing device from the pouch and place it on a bench with aseptic precaution. Dropper is filled with specimen and by holding it vertically, dispense 3-4 free falling drops into the sample well(s). Results are read within 10-15 minutes after application of the specimen.

Interpretation of results

- Negative: Appearance of only one pink-rose coloured band in the control window.
- Positive: Appearance of two pink-rose coloured bands both in test window
- Quality Control: The biocard troponin-I is provided with an inbuilt control to validate test results. A pink rose colored band in the control (C) window indicates proper performance of the test.

Statistical analysis

The data was analyzed using SPSS 20 version. Descriptive and analytical statistics was used to find the association. Chi square, P value and Adjusted Odds ratio were used to check the association. P value less than 0.05 was considered as significant.

RESULTS

Total 100 cases with history of chest pain suggestive of acute myocardial infarction admitted to Basaveshwar Teaching and General Hospital were studied. Present results revealed that cardiac troponin I was more sensitive than CK-MB in overall cases admitted in between 6-24 hrs from the onset of chest pain. 6-8 hours of onset of chest pain to admission, cTnI is more sensitive than CKMB. Majority 78% of the AMI patients were admitted to ICCU department within 12 hours of onset of chest pain.

Table 1: Distribution of acute myocardial infarction cases according to site of heart involved.

Site AMI Type	No. of cases	Percentage	Chi square and p value
Anterior wall acute myocardial infarction	37	41.11	χ^2: 12.5, P: 0.0004
Anterolateral wall acute myocardial infarction	10	11.11	χ^2: 1.635 P: 0.201
Anteroseptal acute myocardial infarction	5	5.55	χ^2: 1.716 P: 0.190
Inferior wall acute myocardial infarction	30	33.33	χ^2: 3.392 P: 0.066
Global acute myocardial infarction	6	6.66	χ^2: 3.244 P: 0.072
Right ventricular acute myocardial infarction	2	2.22	χ^2: 2.998, P: 0.083
Type of AMI			
STEMI	90	90.00	
NSTEMI	10	10.00	

Table 1 shows that as per the site of Myocardial Infarction maximum number 41% were at the anterior wall of acute myocardial infarctions followed by Inferior wall of AMI. 11 percent were affected with Antero lateral wall wereas 5 to 6 percent were affected with anteroseptal and global acute and right ventricular AMI was seen among 2 percent of patients. Anterior wall AMI was the significantly affected site with AMI (χ^2:12.5, P: 0.0004) ST elevation myocardial infarctions (90%) was the more common type of AMI affected.

Table 2 shows that in 45 percent of AMI cases were positive for CKM B (sensitivity of 45%) were as 62% of AMI cases were positive for cTnI (sensitivity 62%). This

reveals that cTnI was more sensitive as compared to CKM B in detecting AMI among patients admitted between 6-24 hours of chest pain. Further the sensitivity of cTnI was better compared with CKM B during 6 to 8 hours after onset of chest pain (64%) and after 12 hours of onset of chest pain (sensitivity 75%) 28% of cases where CKMB is normal, the cTnI detects the AMI cases indicating its sensitivity. After 12 hours of onset of chest pain, the sensitivity of both the cardiac biomarkers increases but more in favour of cTnI. 28% of cases where CKMB is normal, the cTnI detects the AMI cases indicating its sensitivity.

Table 2: Overall sensitivity of CKMB and cTnI in proved AMI cases admitted in between 6-24 hours of chest pain.

Variable	Total No. of AMI cases	Positive cases	Sensitivity percentage	Chi square and p value
Admitted between 6-24 hours of chest pain				
CKMB	100	45	45.00	Λ^2: 5.697, P: 0.011 AOR: 1.526 (1.077-2.161)
cTnI	100	62	62.00	
6-8 hours of onset of chest pain				
CKMB	65	29	44.60	Λ^2: 3.511, P: 0.036 AOR: 1.144 (0.998-1.311)
cTnI	65	42	64.60	
After 12 hours of onset of chest pain				
CKMB	20	12	60	Λ^2: 3.390, P: 0.041 AOR: 1.251 (0.989-1.581)
cTnI	20	15	75	

Present results revealed that the cTnI positivity was 1.526 more as compared to CKM B positivity among AMI patients admitted between 6-24 hours of chest pain. Were as positivity with cTnI was 1.144 times more than CKM B among AMI patients admitted with chest pain between 6-8 hours and positivity with cTnI was 1.251 times more than CKM B among AMI patients admitted after 12 hours of chest pain.

DISCUSSION

Pain is the most common presenting symptom in patients with myocardial infarction. The pain is severe and in some instances intolerable. The pain is prolonged usually lasting for more than 30 minutes and frequently for a number of hours. Pain of acute myocardial infarction is thought to arise from nerve endings in ischemic or injured, but not necrotic myocardium.[3]

Nausea, vomiting and diarrhea occur in more than 50% of patients with transmural myocardial infarction and severe chest pain. These symptoms occur more commonly in patients with inferior myocardial infarction than in those with anterior myocardial infarction.[4] Most commonly the pulse is rapid and regular initially [sinus tachycardia (at 100-110 beats/minutes)]. Slowing as the patient's pain and anxiety are relieved. Premature ventricular beats are common occurring in more than 95% of patients evaluated within the first 4 hours after the onset of symptoms.[5]

It is common for previously hypertensive patients to be normotensive without treatment following acute MI although many of these previously hypertensive patients eventually regain their elevated levels of blood pressure, generally 3-6 months after infarction. Patients in cardiogenic shock by definition have systolic pressure below 90 mm Hg and evidence of end organ hypoperfusionl22. Palpation of the precardium may yield normal findings, but in patients with transmural AMI, it more commonly reveals a presystolic pulsation, synchronous with an audible 4th heart sound reflecting a vigorous left atrial contraction filling a ventricle with reduced compliance. In the presence of left ventricular systolic dysfunction on outward movement of the left ventricle may be palpated in early-diastole coinciding with a S3. During auscultation First sound is frequently muffled. Sometimes S3 and S4 are heard. The mortality of patients who manifest S3 during acute phase of MI is higher than that of patients without such a sound.6 Almost all myocardial infarction-result from coronary atherosclerosis generally with superimposed coronary thrombosis.[7]

Important advances have occurred in our understanding of the pathophysiology of AMI leading to a reorganization of clinical presentations into what is now referred to as the acute coronary syndrome-the spectrum of which includes unstable angina, NSTEMI and STEMI and exposure of substances that promote platelet activation and thrombin generation. The resultant thrombus interrupts blood flow and leads to an imbalance between oxygen supply and demand and if this imbalance is severe and persistent may lead to myocardial necrosis fibrous and inflammatory elements in the arterial wall of

the coronary tree leads to progressive lumen narrowing with subsequent ischemia.[8-10]

In Mario D'Costal et al study, the overall peak performance of cTnI testing in samples received within 24 hours of admission indicated high sensitivity of 97%.[4] In the same study, the sensitivity for cTnI was 79% and for CKIVIB was 44% in AMI patients first specimen obtained at admission.[11]

A single CK-MB value in emergency department patients with acute chest pain had a sensitivity for detecting AMI of 34%5, a single value of cTnI had a sensitivity of about 40%75. Zurich SW et al found using a single troponin-I determination, that 46% of patients with confirmed MI had an abnormal cTnl and normal CKMB initially. In present study, a single CKMB and cTnI value in ICCU ward patients with history of acute chest pain had a sensitivity of 45% and 62% respectively for detecting acute myocardial infarction.[12]

Present study also showed that cTnI sensitivity increased (62% to 64.6%) and CKMB sensitivity remained almost same (44.6%) in patients admitted in between 6-8 hours of chest pain. This indicates that cTnI is more sensitive than CKMB in earlier AMI cases. Single values of CKMB and cTnI drawn more than 12 hours after the onset of symptoms had sensitivities for AMJ in the range of 70-90%.[13-15]

In the present study, the single value of CKMB and cTnI had the sensitivities of 60% and 75% drawn more than 12 hours after the onset of chest pain symptoms respectively. In a study by Kliemam S et al, 31% of the patients with normal CKMB (mass) had elevated cTnI levels, whereas in present study, 35% of the patients were having elevated cTnI levels with normal CKMB values.[16]

Mario D'Costa et al in his study showed the sensitivity of cTnT as 79% for cut-off value of 0.6 µg/L, 96.7% for cut-off value of 1.0 µg/L and 73.8% for cut-off value of 1.5 µgfL. Thus, the cTnI sensitivity varies with the cut-off values used.[11]

CONCLUSION

Cardiac troponin-I (cTnI) was more sensitive serum marker than CKMB in the early diagnosis of acute myocardial infarction (AMI). It seems reasonable for clinicians to measure cardiac troponin-I (cTnI) in patients with suspected AMI which could result in the more cost-effective use of intensive care facilities. In the future, further improvements in analytical performance may open additional diagnostic windows.

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