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Biochemical Study of Cardiac Markers in Acute Myocardial Infarction

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ABSTRACT *Biochemical markers are the primary diagnostic test used to evaluate patients with inconclusive electrocardiograms for possible acute coronary syndrome. Measurements of cardiac enzymes allow a clear distinction between patients with and without myocardial infarction. For many years, creatine kinase (CK) and its isoenzyme MB (Muscle Brain) have been the markers of choice for diagnosing acute myocardial infarction. The scope of this study is to create a general awareness regarding the importance of Troponin T, cardiac biomarker in identifying the cardiovascular disease (CAD) and also in order to make a comparative analysis of the change of bio-chemical enzymes and other constituents in troponin positive myocardial infarction patients. Finally this study proved that the elevated troponin level above the normal range is the perfect biomarker for the diagnosis of acute MI.*

Introduction

Annually, several million patients seek care in the emergency department because of chest pain or other symptoms suggesting an acute coronary syndrome (ACS), but only about 10% are subsequently confirmed to have acute myocar-dial infarction (AMI).1 The criteria for the diagnosis of myocardial infarction have been redefined recently, as reported in a consensus document of the European Society of Cardiology (ESC) and the American College of Cardiology (ACC),2 and require at least 2 of the 3 following characteris-tics: (1) typical symptoms; (2) characteristic rise-and-fall pattern of a cardiac marker (eg, MB isoenzymes of creatine kinase) or, preferably, serum troponins (T or I); or (3) a typical electrocardiogram (ECG) pattern involving the devel-opment of Q waves.

Current cardiac marker technologies, particularly the serum troponins, can now detect extremely small amounts of myocardial necrosis (<1.0 g), which, in the setting of an ACS, may be associated with increased risk of complications compared with patients without myonecrosis. The increased risk associated with even minor amounts of myonecrosis has led to the concept that any amount of myocardial necrosis should be defined as a myocardial infarction2 with the caveat that some patients previously designated as having severe stable or unstable angina may be diagnosed as having had a small AMI. This change in perspective will lead to an increase in the number of cases of AMI that are recognized (improved sensitivity). Presumably, fewer false-positive diag-noses will occur (improved specificity) owing to the improved performance of newer diagnostic technologies. Current criteria for the diagnosis of AMI using the joint ESC/ACC consensus definition are shown in Table 1. In contrast with the older World Health Organization criteria, the ESC/ACC criteria place a much greater emphasis on the

Table 1
European Society of Cardiology/American College of Cardiology Definition of Acute, Evolving, or Recent Myocardial Infarction2

role of biochemical cardiac markers in the diagnosis of AMI. However, the selection of the most optimal cardiac marker (or combination of markers) remains controversial.

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

Troponin is a protein complex located on the thin filament of striated muscles having 3 subunits. Troponin-T(TnT),Troponin-I(TnI),Troponin-C(TnC).Both cardiac Troponin-T and Troponin-I are stored in two compartment distribution in the myocyte,including a small cytosolic pool(4-6%),with the majority of the remaining troponin in the sarcomere.Thus TnT and TnI have similar release kinetics from damaged myocardium.Both Troponins increase in serum with in 4-9 hours after AMI,peak at 12-24 hours and remain elevated for upto 14 days.

The CK-MB isoenzyme can be separated by high-voltage electrophoresis into 2 isoforms: CK-MB 1 and CK-MB 2.3 CK-MB 1 is the plasma form and arises as a result of cleavage of a terminal lysine from the M peptide. CK-MB 2 is the tissue form that is released from damaged myocardium. The CK-MB 2 isoform begins to increase 2 hours after AMI and peaks at 4 to 6 hours. The measurement of CK-MB isoforms can improve early diagnosis of AMI in some situa-tions.4False-positive results have been reported in a variety of other clinical conditions, such as certain muscle diseases, congestive heart failure, and pulmonary edema. The method for measuring CK-MB isoenzymes is technically difficult because it relies on the use of a semiautomated elec-trophoresis system. Consequently, few hospitals offer CK-MB isoforms as part of chest pain evaluation pathways.

Materials and Methods

A study of serum cardiac troponin-T, creatine kinase-MB activity in subjects of MI is carried out in the year 2014. The study is carried out in subjects with myocardial infarction and healthy controls selected from Government General Hospital,Kurnool.

Based on the inclusion and exclusion criteria a total num-

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ber 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-T and serum creatine kinase –MB were estimated. cardiac troponin-T is estimated using chemiluminesence immunoassay (CLIA) kit using principle of immunoenzymometric assay [5] 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.[6] with normal value of CK-MB < 25IU/L AT 37 0 C.

Results

Table 1: Serum levels of CK-MB and cTnT among healthy controls and MI cases on admission

Groups	n		CK-MB (IU/L)	CTnT(ng/ml)
Controls	50	Mean± SD (Range)	14.7±4.2	0.60±0.26
			(7.5-22.4)	(0.20-1.35)
Cases	50	Mean± SD (Range)	17.5±4.2	3.16±1.02
			(9.2- 25.2)	(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: comparision of CK-MB and cTnT 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
cTnT(ng/ml)	<0.01	3.16±1.02

Discussion

Table 1 shows levels (mean ± SD and range) of serum CK-MB and cardiac troponin-T 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-T 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-Tlevel 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 2 shows the comparative analysis of serum levels of CK-MB and Cardiac troponin-T 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-T as 7.0-24.0IU/L and <0.01ng/ml respectively.

It is evident that cardiac troponin-T 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.

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.The present study found a statistically highly significant increase in cardiac troponin-T levels in subjects with MI at the time of admission to the cardiac ICU when compared to CK-MB.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-T is superior to creatine kinase-MB as an indicator of myocardial ischemia. The routine use of cardiac troponin-T 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-T can make the diagnosis of MI accurately rather than waiting for serial changes of serum cardiac markers.

The estimation of serum cardiac troponin-T 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-T 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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