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COMPARISON OF CREATININE PHOSPHO KINASE MYOCARDIAL BAND WITH TROPONIN I FOR MARKING ABILITY OF MYOCARDIUM INFARCTION DISEASE IN SOUTH INDIAN PATIENTS - A PROSPECTIVE COHORT STUDY

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ABSTRACT
A heart attack, or acute myocardial infarction, is a potentially fatal disorder that develops when the blood supply to the heart muscle is suddenly interrupted, resulting in tissue damage. A blockage in one or more of the coronary arteries frequently causes this. The study group consisted of 40 patients with diagnostic signs of AMI, and 20 healthy age and sex matched controls were enrolled for comparison. It was done to conduct standard investigations like CPK-MB, Troponin-I, and others. According to the study's findings, the diagnosis of acute myocardial infraction requires the use of both CPK-MB and Troponin-I.CPK-MB is up within 10 hours of chest pain and returns to normal levels in almost 2 days, while troponin-I is elevated within 8 hours of chest pain and remains elevated for almost a week. Troponin-I will not be able to indicate the fresh infarction, but Troponin-I is a better indicator of AMI.

KEYWORDS: Acute myocardium infarction; CPK-MB; Troponin I.

1. INTRODUCTION
Chest pain (Myocardial Infarction), the technical term for a heart attack, is a life-threatening medical emergency in which your heart muscle starts to deteriorate due to inadequate blood flow. A blockage in the arteries that feed blood to your heart is typically the cause of this. While chest discomfort and shortness of breath are the typical signs of a heart attack, there can be a wide range of other symptoms as well. The most typical signs of a heart attack are pressure or tightness in the chest, pain that lasts for more than a few minutes or that comes and goes from the chest, back, jaw, and other upper body areas, shortness of breath, sweating, nausea, vomiting, anxiety, feeling like you're going to faint, a rapid heartbeat, and a sense of impending doom. The most accurate and precise sign for the identification of an acute myocardial infarction is the creatine phosphokinase myocardial band (CPK-MB). A cardiac marker used to help with the diagnosis of an acute myocardial infarction, myocardial ischemia, or myocarditis is the CPK-MB test, sometimes referred to as the CK-MB test. The enzyme phosphocreatine kinase, which has two forms (isoenzymes CKM and CKB), is measured by the blood level of CK-MB (creatine kinase myocardial band). The troponin test has taken the place of the test in several areas. However, the test that involves calculating the ratio of the CK-MB1 to CK-MB2 isoforms has recently undergone advancements. The previous test discovered the existence of cardiac-related isoenzyme dimers, but the more recent test detects distinct B subunit isoforms specific to the myocardium. There have been numerous reports of CK-MB levels that are higher than the blood level of total CK, particularly in babies with heart abnormalities, particularly ventricular septal defects. The ratios are in favour of vasculitis or pulmonary emboli. A complex CK and IgG molecule produced by an autoimmune response should be taken into account. A class of cardiac and skeletal muscle proteins is called troponin I. It is a component of the troponin protein complex, which holds the actin-tropomyosin complex in place by binding to actin in delicate myofilaments. In relaxed muscle, troponin I prevents myosin from attaching to actin. Troponin I is dislocated as a result of conformational changes brought about by calcium's binding to troponin C.

After that, tropomyosin departs from the myosin-binding site on actin, causing the muscle to contract. The letter I is assigned because of its inhibiting nature.[1,2] It is a helpful sign for heart attack laboratory diagnosis. Both tests can be used to confirm cardiac muscle damage because they occur under the same conditions as troponin T but in different plasma concentrations. Laboratories typically offer one test or the other. The following list of three paralogs, along with their locations and OMIM accessions, is of paralogs expressed in

humans with distinct tissue-specific expression patterns are Cardiac troponin I, TNNI3, slow-twitch skeletal muscle isoform troponin I, TNNI2, and fast-twitch skeletal muscle isoform troponin I.[3-5]

2. MATERIAL AND METHODS

40 patients reporting to the Department of Medicine out patients and inpatients of Department of Cardiology, Shadan Institute of Medical Sciences, Peerancheruvu, Telangana, India with diagnostic features of Acute Myocardial Infraction were recruited and constituted the study group and 20 healthy age and sex matched controls were recruited. Special investigations such as CPK-MB, Troponin-I and other routine investigations were carried out like hemoglobin, bleeding time, clotting time, total leukocyte count, differential leukocyte count, erythrocyte sedimentation rate, Urine complete investigation, fasting blood sugar, Serum creatinine, CPK-MB and Troponin in the department of biochemistry, Shadan Institute of Medical Sciences, Peerancheruvu, Telangana, India. A detailed history was recorded from all the subjects. Inclusion criteria is patients recruited in the study group were adult out patients and inpatients of Department of Cardiology, Shadan Institute of Medical Sciences, Peerancheruvu, Telangana , India with chest pain or symptoms of acute myocardial infraction. Excluded patients from the study group were pregnant women, lactating mothers, patients with renal failure and patients on hormonal therapy. 5ml of blood were collected under aseptic conditions from the cubital vein of the patients and serum/ plasma were separated by centrifugation from further analysis.

2.1 Method for estimation of Troponin-I

On a CTK Biotech cassette, CA (USA), a lateral flow chromatographic immunoassay for the Onsite Troponin-I Rapid Test was performed by using human serum. A sufficient amount of test material is poured into the cassette's sample well, where it is carried across the cassette by capillary action. Elevated Troponin-I will bind to the antibody conjugates if it is present in the specimen. The pre-coated anti-Troponin-I antibodies subsequently trap the immunocomplex on the membrane, generating a burgundy-colored T band that indicates a positive Troponin-I test result. Absence of the T-band indicates a failure. Regardless of the presence of Troponin-I in the specimen, the test includes an internal control (C band) that should show a burgundy coloured band of goat anti-mouse IgG/mouse IgG-gold conjugate immunocomplex. Otherwise, the test is invalid.

2.2 Method for estimation of CPK-MB

For the purpose of measuring CPK-MB quantitatively in human serum or plasma, the Kit method was used to estimate CPK-MB on an autoanalyser. The principle of estimation involves CK-M and CK-B subunits of CPK-MB make up the whole protein. Certain anti-CK-M antibodies completely block both the CK-M subunit of CPK-MB and the majority of overall CK-activity. Just CK-B activity, which is only half of CK-MB activity, is measured. At 340 nm absorbance and 37 °C ambient temperature, the value was read.

3. RESULTS

The study group's mean age is 48.1±09.2 years, while the control group's is 49.1±1.23 years, with a p-value of 0.282. (non-significant). Therefore, both groups can be compared. When comparing the two groups, gender and biochemical investigations were similarly not significant. Based on the time required to collect the sample, as shown in Table 3 and Figure 1, the study group was split into two groups. Troponin-I was compared between the study and control groups, as indicated in Table 1 and Figure 2. The comparison of CPK-MB levels between the study and control groups is depicted in Table 2 and Figure 3.

Table 1: Sensitivity and Specificity of Troponin-I and CPK-MB.

Groups	Total no of cases	Troponin-I				Sensitivity	Specificity
		Positive	% Age	Negative	% Age		
Study group	40	36	90	4	10	0.95	1
Control group	20	0	0	20	100		
Groups	Total no of cases	CPK-MB				Sensitivity	Specificity
		(>24 IU/L)	% Age	(<24 IU/L)	% Age		
Study group	40	30	75	10	25	0.9	1
Control group	20	0	0	20	100		

Table 2: Comparison of Troponin-I Vs CPK-MB levels in patients presenting within 8 h, 8-24 h of onset of chest pain.

Within 8h of of onset of chest pain				
Total Cases	Troponin-I		CPK-MB (IU/L)	
	Positive	Negative	Normal Range (>24 IU/L)	Raised (<24 IU/L)
8	6	2	2	6
Within 8-24h of onset of chest pain				
Total Cases	Troponin-I		CPK-MB (IU/L)	
	Positive	Negative	Normal Range (>24 IU/L)	Raised (<24 IU/L)
32	31	1	23	9

Table 3: Distribution of cases depending upon time of collecting of sample

Group	Time Period	No of Patients	% Age
Group-I	Within 8 hrs of Chest Pain	6	15
Group-II	Within 8-24 hours of Chest Pain	34	85
	Total	40	100

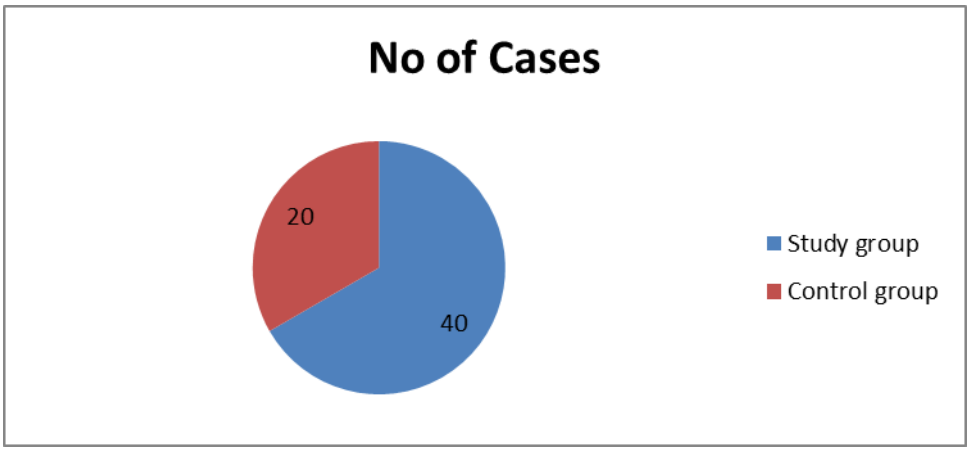

Figure 1: Study population details.

Table 4: Comparison of Troponin-I and CPK-MB levels in Cases.

Total no of cases	Troponin-I		CPK-MB (IU/L)	
40	Positive	Negative	(>24 IU/L)	(<24 IU/L)
	36	4	31	9

Table 5: Comparison of Troponin-I in patients presenting within 8 h, 8-24 h of onset of chest pain.

Total	Within 8 hours	
7	Troponin-I	
	Positive	Negative
	4	3
Total	**Within 8-24 hours**	
33	Troponin-I	
	Positive	Negative
	31	2

Figure 2: Troponin-I positivity.

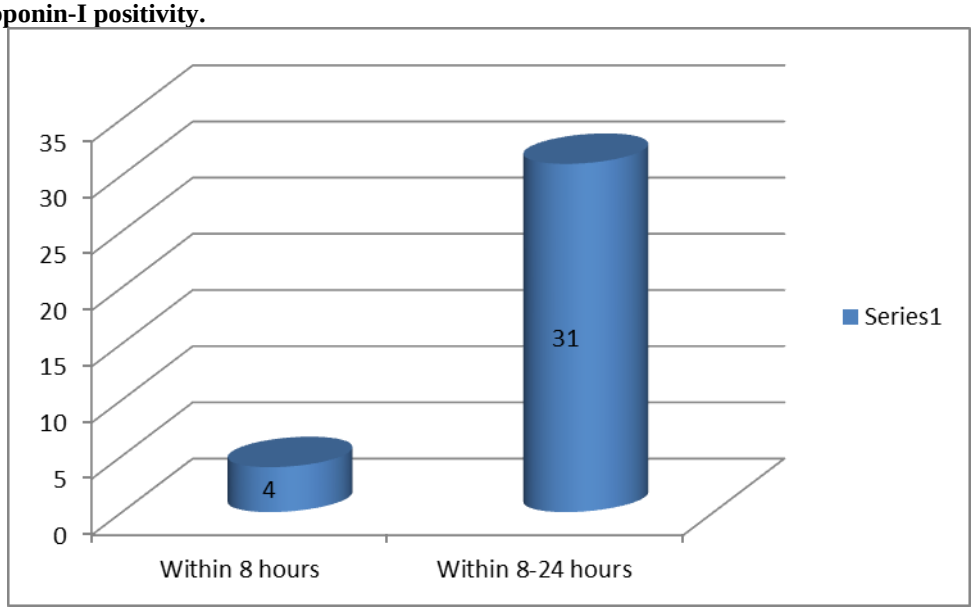

Table 6: Comparison of Troponin-I,CPK-MB between study and control group.

Troponin-I						
Groups	**No of Cases**	**Troponin-I positive**	**Percentage**	**Troponin-I Negative**	**Percentage**	**Significance**
Study group	40	36	90	4	10	$\chi 2=54.23$ $p<0.01$Highly significant
Control group	20	0	0	20	100	
CPK-MB						
Groups	No of Cases	CPK-MB positive	Percentage	CPK-MB Negative	Percentage	Significance
Study group	40	10	25	30	75	Highly significant
Control group	20	20	100	0	0	

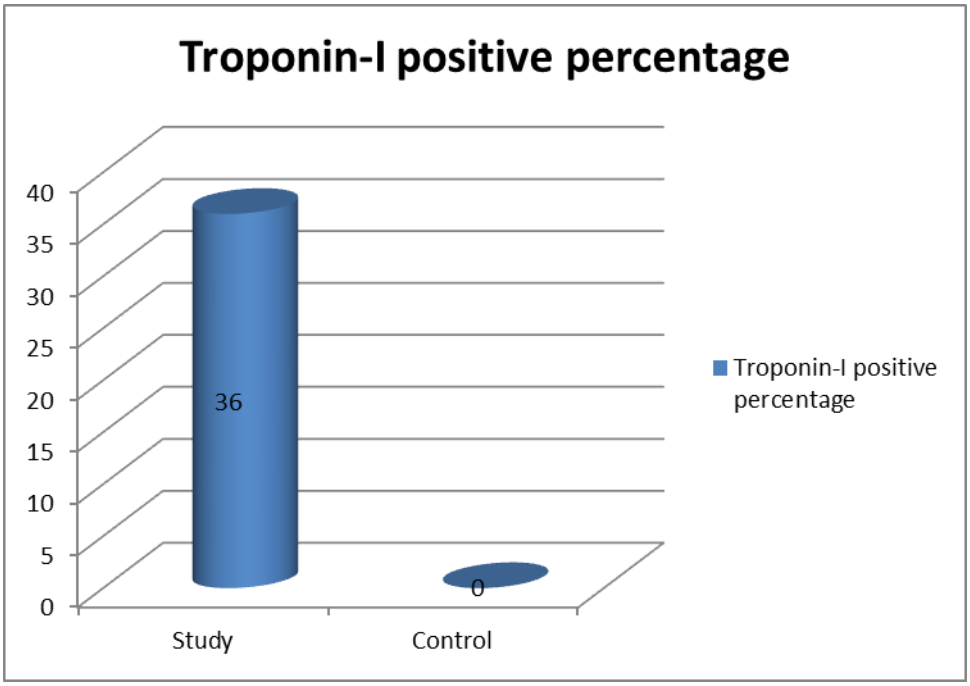

Fig. 3: Troponin-I positive percentage

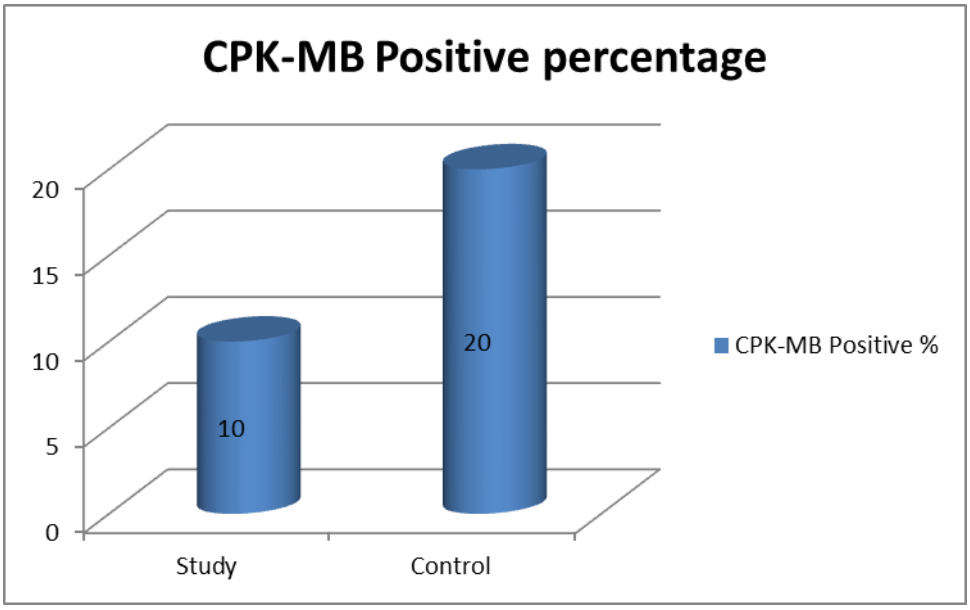

Fig. 4: CPK-MB positive percentage.

Table 7: Comparison of CPK-MB levels in patients presenting within 8 h, 8-24h of onset of chest pain.

Within 8h of of onset of chest pain						
Total no. of cases	**CPK-MB (>24 IU/L)**	**Range (IU/L)**	**Mean±SD (IU/L)**	**CPK-MB (<24 IU/L)**	**Range (IU/L)**	**Mean±SD (IU/L)**
6	2	32-60	46±17.12	4	12-23	11.1±3.09
Within 8-24h of onset of chest pain						
Total no. of cases	CPK-MB (>24 IU/L)	Range (IU/L)	Mean±SD (IU/L)	CPK-MB (<24 IU/L)	Range (IU/L)	Mean±SD (IU/L)
40	31	26-478	141.6±109.26	9	14-23	18.9±2.09

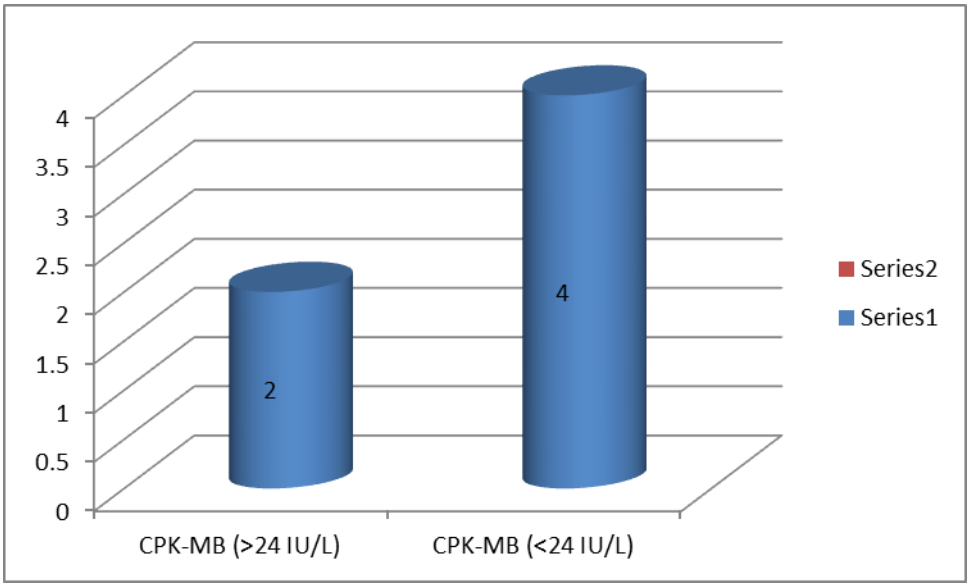

Fig. 5: CPK-MB within 8 h of chest pain

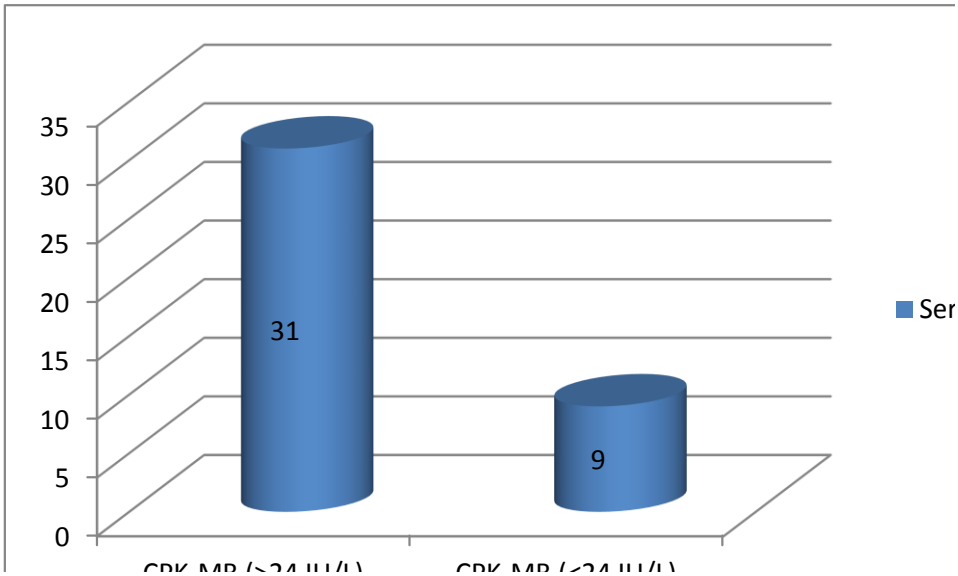

Fig. 6: CPK-MB within 8 -24 h of chest pain

4. DISCUSSION

Diagnosis of acute myocardial infarction relies upon the clinical history, interpretation of the electrocardiogram, and measurement of serum levels of cardiac enzymes. Newer biochemical markers of myocardial injury, such as cardiac troponin I and cardiac troponin T, are now being used instead of or along with the standard markers, the MB isoenzyme of creatine kinase (CK-MB) and lactate dehydrogenase. One of the main causes of death for both men and women worldwide is AMI. Within 8 to 24 h following the attack, heart tissue starts to die. Therefore, a number of indicators were employed to detect AMI at an early stage. The validity of cardiac Troponin-I and CPK-MB in these individuals was used in the current study. Numerous studies have already compared cardiac Troponin-I with CPK-MB.[6-9]

Thompson et al. evaluated 50 AMI patients in 1988 and came to the extremely significant conclusion that >95% of patients had abnormally increased CPK-MB (>24IU/L) and 5% had CPK-MB within normal range (24IU/L). Additionally, they came to the conclusion that there was no Troponin-I negative cases and that all patients had Troponin-I positive cases. In a 1997 study of 47 AMI patients, Hamm et al. found that 91% of patients had excessively elevated CPK-MB levels (>24IU/L), and 9% of patients had levels that were within the normal range (24IU/L). This difference was very significant. They came to the same conclusion as Thomson et al., that all patients had Troponin-I positive cases and none had Troponin-I negative instances. In the current study, 65% of cases had elevated CPK-MB levels (>24IU/L), 35% of cases had CPK-MB levels (24IU/L), and 90% of cases had positive Troponin-I levels, 10 % of cases had negative Troponin-I levels, compared to controls.[10-13] Troponin-I positivity was 0% and Troponin-I negativity was 100% among the participants, with 0% having CPK-MB (>24IU/L) and 100% having CPK-MB (24IU/L). The results of statistical comparisons of CPK-MB between the study and control groups were noteworthy. Our findings confirm that CPK-MB and Troponin-I are highly sensitive (65 and 90 percent, respectively) and specific (100 and 100 percent, respectively) in patients with AMI.[13-19] I found that troponin I is a better cardiac marker than CK-MB for myocardial infarction because it is equally sensitive yet more specific for myocardial injury; troponin T is a relatively poorer cardiac marker than CK-MB because it is less sensitive and less specific for myocardial injury; and both troponin I and troponin T may be used as independent prognosticators of future cardiac events. Troponin I is a sensitive and specific marker for myocardial injury and can be used to predict the likelihood of future cardiac events. It is not much more expensive to measure than CK-MB. Overall, troponin I is a better cardiac marker than CK-MB and should become the preferred cardiac enzyme when evaluating patients with suspected myocardial infarction.

5. CONCLUSION

Elevated troponin levels indicate myocardial injury but may occur in critically ill patients without evidence of myocardial ischemia. An elevated troponin alone cannot establish a diagnosis of myocardial infarction (MI), yet the optimal methods for diagnosing MI in the intensive care unit (ICU) are not established. A troponin test measures the levels of troponin T or troponin I proteins in the blood. These proteins are released when the heart muscle has been damaged, such as occurs with a heart attack. The more damage there is to the heart, the greater the amount of troponin T and I there will be in the blood.

In conclusion, whenever cardiac Troponin-I and CPK-MB are taken into account for the diagnosis of AMI, we found that both substances were necessary. When experiencing chest pain, troponin-I increases within eight hours and stays elevated for ten days, while CPK-MB increases during eight to sixteen hours and returns to normal levels within 48 hours. Therefore, fresh infarction cannot be detected by troponin-I, while troponin-I is a stronger diagnostic of AMI. Fresh attack will be detected by serial CPK-MB. Overall, troponin I is a better cardiac marker than CK-MB and should become the preferred cardiac enzyme when evaluating patients with suspected myocardial infarction.

6. ACKNOWLEDGEMENT

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7. Conflict of Interest

Author declares no conflict of interest.

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