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Research Article

CPK-MB Vs Troponin I – Better Marker for Acute Myocardium Infarction

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Abstract

Acute myocardial infarction (AMI) occurs when blood flow is occulted by an atherosclerotic plaque present in the intima lining of a coronary artery. 50 patients with diagnostic features of AMI were recruited and constituted the study group and 25 healthy age and sex matched controls were recruited for comparison constituted the control group. Special investigations such as CPK-MB, Troponin-I and other routine investigation were assayed. We concluded from the results that whenever considering the role of cardiac Troponin-I and CPK-MB in the diagnosis of AMI, we required both CPK-MB and Troponin-I for the diagnosis of AMI. Troponin-I rises within 6 hours of chest pain, remains raised for 10 days and CPK-MB is raised within 6-12 hours of chest pain and comes back to normal levels within 48 hours. So, we can say that fresh attack will be assayed by serial CPK-MB whereas 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; Troponin I

ISSN: 2578-0204

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Submission: January 2, 2020
Published: March 06, 2020

Volume 3 - Issue 3

How to cite this article: Vivek Kumar Garg. CPK-MB Vs Troponin I – Better Marker for Acute Myocardium Infarction. Open J Cardiol Heart Dis.3(3). OJCHD.000562.2020.
DOI: 10.31031/OJCHD.2020.03.000562.

Introduction

Acute myocardial infarction (AMI) occurs when blood flow is occulted by an atherosclerotic plaque present in the intima lining of a coronary artery [1,2]. It spreads from the inner lining of the heart to the outer lining. The heart will be damaged if there is complete blockade for at least 15-20 minutes [3]. It mostly occurs at the area which is more at risk and when the obstruction is sustained for 4-6 hours. Most of the destruction takes place in the first 2-3 hours. The heart muscle will be relieved when regaining of blood flow is within 4-5 hours, but the salvage is more if blood flow is regained within 1-2 hours [4]. Risk factors for AMI are old age, male sex, smoking, alcohol, high LDL, low HDL, High cholesterol, diabetes, hypertension etc. [5]. In 1980s, SGOT and LDH enzyme assays were used to diagnose AMI but these enzymes are not tissue-specific [6]. So nowadays, cardiac Troponin-I or T and CPK-MB assays are used to diagnose AMI as these markers are tissue specific as well as rise very early after AMI. Thus, in the present study we evaluate the positive levels of Troponin-I in patients of AMI, (b) evaluate the levels of CPK-MB in patients of AMI, (c) compare and correlate the positive levels of Troponin-I and CPK-MB in these patients, and also (d) evaluate the sensitivity and specificity of cardiac Troponin-I Vs CPK-MB in these patients.

Material and Methods

50 patients reporting to the Department of Medicine (Indoor and Outdoor) of Rajindra Hospital, Patiala, Punjab, India with diagnostic features of AMI were recruited and constituted the study group and 25 healthy age and sex matched controls were recruited for comparison constituted the control group. Special investigations such as CPK-MB, Troponin-I and other routine investigation were assayed in the Department of Biochemistry, Government Medical College and Rajindra Hospital, Patiala. A detailed history was recorded from all the subjects.

Inclusion criteria: Patients recruited in the study group were adults' patients coming to the for the study group were all the patients Department of Medicine (Indoor and Outdoor) of Rajindra Hospital, Patiala, Punjab, India with chest pain or symptoms of AMI.

Exclusion criteria: Patients excluded from the study were pregnant women, lactating mothers, patients with renal failure and patients on hormonal therapy. Routine investigations were analyzed like Hemoglobin, bleeding time, clotting time, total leukocyte count, differential leukocyte count, erythrocyte sedimentation rate, Urine complete investigation, fasting blood sugar, Serum creatinine, Creatin kinase-MB and Troponin-I.

Special investigations

Specimen collection: 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.

Method for Troponin-I

The Onsite Troponin-I Rapid Test a lateral flow chromatographic immunoassay was done on CTK Biotech cassette, CA (USA) in human serum. Adequate volume of test specimen is dispensed into the sample well of the cassette, the specimen migrates by capillary action across the cassette. Elevate Troponin-I present in the specimen will bind to the antibody conjugates. Immunocomplex is then captured on the membrane by the pre-coated anti-Troponin-I antibodies, forming a burgundy colored T band, Indicating a Troponin-I positive test result. Absence of T- band suggests a negative result. The test contains an internal control (C band) which should exhibit a burgundy colored band of goat anti-mouse IgG/mouse IgG-gold conjugate immunocomplex regardless of the present of Troponin-I in the specimen. Otherwise, the test is invalid, and the specimen must be retested with another device.

Creatin kinase-MB (CPK-MB)

CPK-MB was estimated on antoanalyzer by Kit method for quantitative measurement of CPK-MB in human serum/ plasma.

Principle: CPK-MB consists of subunits CK-M and CK-B. Specific antibodies against CK-M inhibit complete CK-MM activity (main part of the total CK-activity) and the CK-M subunit of CPK-MB. Only CK-B activity is measured, which is half of the CK-MB activity. The value was read at absorbance 340nm at room temperature 37 °C.

Result

Mean±SD values of study group and control group are 51.2±11.4 and 50.7±4.74 years which has p-value of 0.218 (non-significant). So, both the groups are comparable. Gender, Biochemical Investigations was also non-significant when compared both the groups. Study group was divided into two groups on the basis of time of collecting of sample shown in Table 1 and Figure 1. Comparison of Troponin-I between study and control group was done which is shown in Table 2 and Figure 2. Comparison of levels of CPK-MB between study and control group was done which is shown in Table 3 and Figure 3 (Figure 4 & 5) (Table 4-12).

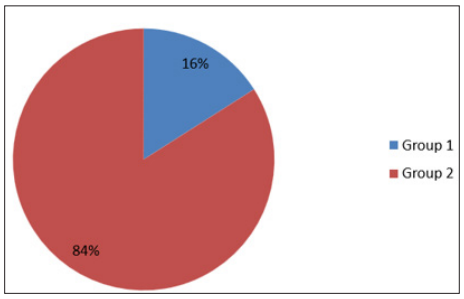


Figure 1: Distribution of cases depending upon time of collecting of sample.

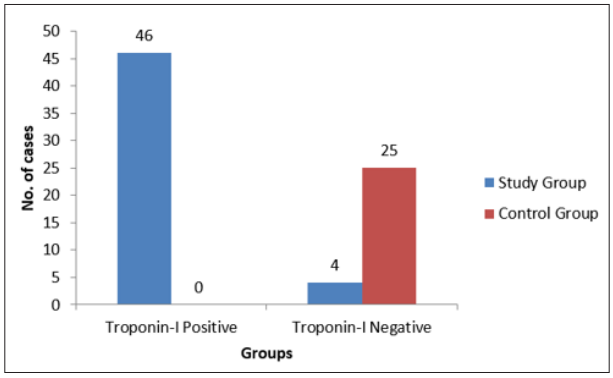
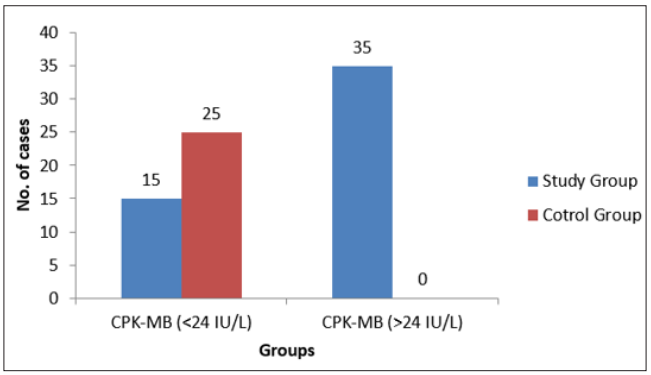


Figure 2:



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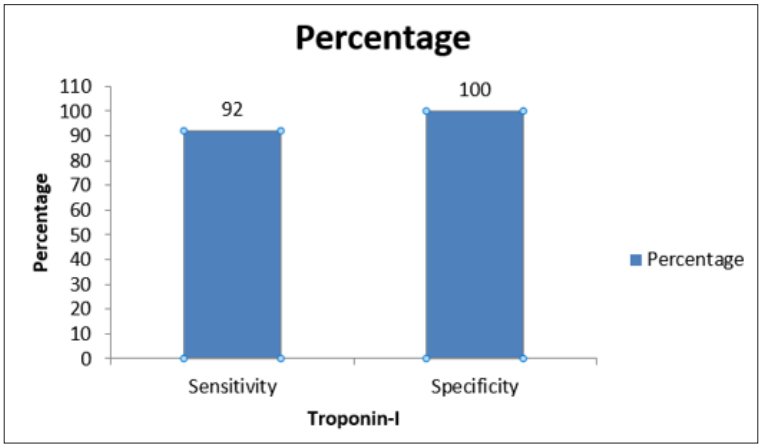

Figure 4:

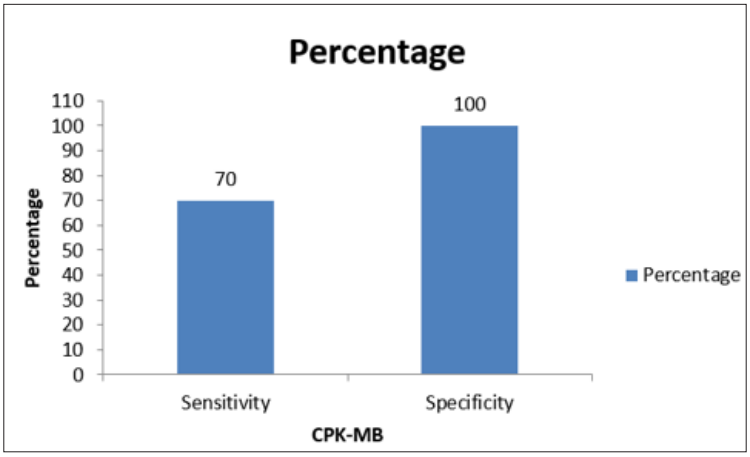

Figure 5:

Table 1: Distribution of cases depending upon time of collecting of sample.

Groups	Time Period	No of Patients	%Age
Group I	Within 6 hours of chest pain	8	16
Group II	Within 6-24 hours of chest pain	42	84
	Total	50	100

Table 2: Comparison of Troponin-I between study and control group was done which is shown in Table 2.

Groups	No of Cases	Troponin-I Positive	%Age	Troponin-I Negative	%Age	Significance
Study Group	50	46	92	4	8	$\chi 2=55.67$ $p<0.01$Highly significant
Control Group	25	0	0	25	100	

Table 3: Comparison of levels of CPK-MB between study and control group.

Groups	No of Cases	CPK-MB (<24 IU/L)	%Age	CPK-MB (>24 IU/L)	%Age	Significance
Study Group	50	15	30	35	70	Highly significant
Control Group	25	25	100	0	0	

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

Total No of Cases	Troponin-I		CPK-MB (IU/L)	
50	Positive	Negative	(>24 IU/L)	(<24 IU/L)
	46	4	35	15

Table 5: Comparison of Troponin-I in patients presenting within 6 hours of onset of chest pain.

Total No of Cases	Within 6 hours	
	Troponin-I	
8	Positive	Negative
	5	3

Table 6: Comparison of Troponin-I in patients presenting within 6-24 hours of onset of chest pain.

Total No of Cases	Within 6-24 hours	
	Troponin-I	
42	Positive	Negative
	41	1

Table 7: Comparison of CPK-MB levels in patients presenting within 6 hours 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)
8	2	28-60	45±16.09	5	Sep-23	16.4±5.36

Table 8: Comparison of CPK-MB levels in patients presenting within 6-24 hours 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)
42	32	26-478	139.2±113.07	10	14-23	19.1±3.66

Table 9: Comparison of Troponin-I Vs CPK-MB levels in patients presenting within 6 hours 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	5	3	3	5

Table 10: Comparison of Troponin-I Vs CPK-MB levels in patients presenting within 6-24 hours of onset of chest pain.

Total Cases	Troponin-I		CPK-MB (IU/L)	
	Positive	Negative	Normal Range(>24 IU/L)	Raised(<24 IU/L)
42	41	1	32	10

Table 11: Sensitivity and Specificity of Troponin-I.

Groups	Total No of Cases	Troponin-I				Sensitivity	Specificity
		Positive	%Age	Negative	%Age		
Study Group	50	46	92	4	8	0.92	1
Control Group	25	0	0	25	100		

Table 12: Sensitivity and Specificity of CPK-MB.

Groups	Total No of cases	CPK-MB				Sensitivity	Specificity
		(>24 IU/L)	%Age	(<24 IU/L)	%Age		
Study Group	50	35	70	15	30	0.7	1
Control Group	25	0	0	25	100		

Discussion

AMI is one of the leading causes of death worldwide in both men and women. Heart tissue death occurs within 6-12 hours of attack. So, to diagnose AMI in early stage, several markers were used [7]. In the present study we used the reliability of cardiac Troponin-I and CPK-MB in these patients. In the past many studies also compare cardiac Troponin-I and CPK-MB. Thompson et al in 1988 studied 50 patients of AMI and concluded that >95% patients had abnormally raised CPK-MB (>24IU/L) and <5% patients had CPK-MB within normal range (<24IU/L) which was highly significant. They also concluded that 100% of the patients had Troponin-I positive and 0% had Troponin-I negative cases [8].

Hamm et al. [9] in 1997 studied 47 patients of AMI and revealed that 91% patients had abnormally increased levels of CPK-MB (>24IU/L) and 9% patients had CPK-MB levels within normal range (<24IU/L) and was highly significant. They also concluded

that 100% of the patients had Troponin-I positive and 0% had Troponin-I negative cases [9] like Thomson et al. In the present study, 70% cases had raised CPK-MB (>24IU/L) and 30% cases had (<24IU/L) levels of CPK-MB, and 92% cases had Troponin-I positive and 8% cases had Troponin-I negative while in control group 0% individuals had CPK-MB (>24IU/L) and 100% individuals had CPK-MB (<24IU/L) and 0% individuals had Troponin-I positive and 100% had Troponin-I negative. Statistical analyses comparing CPK-MB between study and control groups was significant. Our results comply with the results of the above-mentioned studies that CPK-MB and Troponin-I are highly sensitive (70% and 92%) and highly specific (100% and 100%) in patients of AMI.

We also compared the sensitivity and specificity of CPK-MB and Troponin-I in the present study with other studies in the study group. Sawhney et al in 2004 conducted a study in 18 patients and concluded that sensitivity and specificity of Troponin-I and CPK-MB was 83%, 87% and 93%, 100% respectively [9]. Meraz et al. [10] in 2006 also conducted a study on 40 patients of AMI and demonstrated that sensitivity and specificity of Troponin-I and CPK-MB was 95%, 95% and 40%, 50% respectively [10]. In the present study, we recruited 50 patients and concluded that sensitivity and specificity of Troponin-I and CPK-MB was 92%, 100% and 70%, 100% respectively from comparison of sensitivity and specificity of cardiac Troponin-I in the present study and other studies, it is concluded that our study contradicts the study done by Sawhney et al and but consistent with Meraz et al. [10]. But in the case of CPK-MB, our study consistent with the study done by Meraz et al. [10] and contradicts the study done by Sawhney et al.

Conclusion

In conclusion, whenever we consider the role of cardiac Troponin-I and CPK-MB in the diagnosis of AMI, we required both CPK-MB and Troponin-I for the diagnosis of AMI. Troponin-I rises within 6 hours of chest pain, remains raised for 10 days and CPK-MB is raised within 6-12 hours of chest pain and comes back to normal levels within 48 hours. So, we can say that fresh attack will be assayed by serial CPK-MB whereas Troponin-I will not be able to indicate the fresh infarction, but Troponin-I is a better indicator of AMI. The limitation of the study was small sample size, so I suggest a larger sample size to conclude the further result.

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