



COMPARATIVE STUDY OF CARDIAC BIOMARKERS IN MYOCARDIAL INFARCTION AND ATYPICAL CHEST PAIN

Biochemistry

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ABSTRACT

Background: Myocardial infarction (MI), commonly known as a heart attack, occurs when blood flow decreases or stops to a part of the heart, causing damage to the heart muscle. The myocardium receives its blood supply from the two large coronary arteries and their branches. Occlusion of one or more of these blood vessels (coronary occlusion) is one of the major causes of MI.^[1]

Chest pain is the most common symptom of acute myocardial infarction (AMI) and is often described as a sensation of tightness, pressure, or squeezing. The pain associated with MI is usually diffuse, does not change with position and lasts for more than 20 minutes. [2] If an individual experiences chest pain that does not meet the criteria to be called as a classic anginal chest pain is termed as atypical chest pain (ACP). The symptoms experienced in such cases may be both cardiac as well as non-cardiac related. An anginal chest pain is a squeezing or pressure like sensation in the chest that is felt generally when an individual does any strenuous activity. This symptom is the classic presenting feature of MI. An atypical chest pain does not occur in the sternum and may radiate to other regions of the body as well. This pain is sudden sharp and short lived.

Material & method: This study was conducted at Department of Biochemistry PBM hospital in association with Department of Cardiology-HRMC, Sardar Patel Medical College Bikaner, Rajasthan from april 2018 to march 2019. There were 50 study and 50 controls in the age groups from 20 to 70 years. Fresh samples were taken and required tests were performed following standard protocol. Troponin-I level was estimated by two-site ELISA method and CK-MB level was quantitatively estimated on fully auto analyzers using Immuno-inhibition/UV Kinetic method.^[3]

Result: Troponin-I showed high significance with p-value < 0.0001***. For CK-MB p-value was 0.0001**

Conclusion: In this study the role of cardiac biomarkers- Troponin-I and CK-MB has been explored. Troponin-I is the most significant and preferred cardiac biomarker for early detection of MI.

KEYWORDS

Cardiac Biomarkers, Myocardial Infarction, Atypical Chest Pain

INTRODUCTION

MI is one of the five main manifestations of coronary heart disease (CHD), namely stable angina pectoris, unstable angina pectoris, MI, heart failure and sudden death.^[4] Cardiac biomarkers become detectable in the peripheral blood once the capacity of the cardiac lymphatics to clear the interstitium of the infarct zone is exceeded and spill over into venous circulation occurs. Troponin is an enzyme or protein that is used for the evaluation of heart injury. Cardiac troponins are proteins that are located in the heart muscle. Troponins are released into the bloodstream when damage has occurred to the heart muscle. Troponin is a sensitive marker that is used to make the diagnosis of a heart attack or other heart damage.^[5] It is highly specific to cardiac tissue and accurately diagnoses myocardial infarction with a history of ischaemic pain or ECG changes reflecting ischaemia. Cardiac troponin level is dependent on infarct size, thus providing an indicator for the prognosis following an infarction.^[6] CK-MB is found in cardiac and skeletal muscle. It has high sensitivity but poor specificity. It may be useful for the early detection of MI. It is imperative to identify a heart attack in its early stages because healthcare providers can then provide emergency treatment.

The early identification of MI is vital for the institution of anti-thrombotic therapy to limit heart muscle damage and preserve cardiac function. As we have seen from above discussion that the level of Troponin-I, CK-MB is increased in MI and not in ACP. In this proposal work the level of Troponin-I, CK-MB will be determined in MI subjects and will be compared with ACP controls.

MATERIAL AND METHOD

This study was carried out in Department of Biochemistry of Sardar Patel Medical College and attached Hospital, Bikaner for the necessary tests and investigations. Clinically diagnosed MI and ACP patients, registered in the OPD and wards of department of cardiology, P.B.M Hospital satisfying both the inclusion criteria and exclusion criteria were selected for this study.

It was an observational cross sectional study which was conducted on 100 subjects. Out of 100 subjects, 50 subjects were patients of MI and 50 were ACP patients having matched age and sex. Patients who had typical symptoms of MI like chest pain, sweating, breathlessness etc.

and specific abnormalities for MI on ECG and elevated cardiac markers, and patients who had atypical symptoms like chest pain, sweating, breathlessness etc. without any specific abnormalities on ECG and no elevated cardiac markers were included in the present study. All patients having previous heart diseases like congenital heart disease, diseases of heart valves and myocardium, confounding factors that could interfere in the biochemical analyses of study subjects and alter the results like renal insufficiency, hepatic disease, inflammatory disease, history of recent infection and febrile disorders etc were excluded.

The i-STAT cardiac Troponin I (cTnI) test cartridge which uses a two-site ELISA method was used for estimation of troponin-I.^[7] Creatine Kinase-MB was quantitatively estimated on fully auto analyzers using Immuno-inhibition/UV Kinetic method.^[8]

RESULT

The blood samples of control group as well as study group were withdrawn and analyzed for troponin-I and ck-mb level. Table shows mean Troponin-I (ng/ml) and mean CK-MB concentration (u/l) in myocardial infarction (study group) compared with that of atypical chest pain (control group). Mean Troponin-I concentration and standard deviation (SD) was (0.018 ± 0.013) in ACP and (5.639 ± 9.439) in MI subjects showing a large variation. Calculated degree of freedom (DF) and t-value were 98, 4.210 respectively. Troponin-I showed high significance with p-value < 0.0001***. Mean CK-MB (U/L) and SD in study and control group was (46.340 ± 17.915) and (10.460 ± 2.815) respectively. The DF and t-value was 98 and 13.990 respectively. CK-MB is not as significant as troponin-I because its p-value was 0.0001** (Table 1, figure 1 and 2).

TABLE

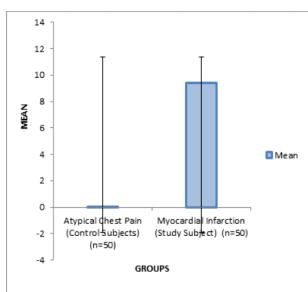
Mean Troponin (ng/ml) And Ck-mb (u/l) Concentration In Myocardial Infarction (study Group) Compared With That Of Atypical Chest Pain (control Group)

BLOOD PARAMETRES	ATYPICAL CHEST PAIN (CONTROL SUBJECTS) (n=50)	MYOCARDIAL INFARCTION (STUDY SUBJECT) (n=50)	SIGNIFICANCE		
			MEAN ± SD	MEAN ± SD	DF t-value p-value

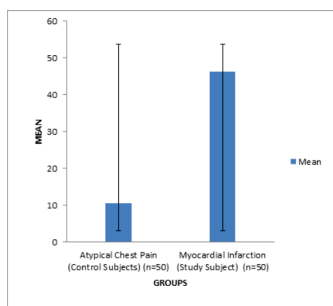
TROPONIN-I	0.018 ± 0.013	5.639 ± 9.439	98	4,210	<0.0001***
CK-MB	10.460 ± 2.815	46.340 ± 17.915	98	13,990	0.0001**

GRAPH-I

Mean Troponin Concentration (ng/ml) In Myocardial Infarction (study Group) Compared With That Of Atypical Chest Pain (control Group)

**GRAPH-II**

Mean Ck-mb Concentration (u/l) In Myocardial Infarction (study Group) Compared With That Of Atypical Chest Pain (control Group)

**DISCUSSION**

According to table mean Troponin-I concentration was found to be 0.018 in ACP and 5.639 in MI subjects. Standard deviation (SD) showed a large variation, 0.013 in ACP and 9.439 in MI. Calculated degree of freedom (DF) and t-value were 98, 4.210. Troponin-I showed high significance with p-value < 0.0001***. Hence troponin-i is the most significant and preferred cardiac biomarker for early detection of MI.

This result is supported by original research by Kiran R Bagale et al in 2014 that indicated that Troponin-I is more sensitive indicator for detecting acute myocardial infarction along with unstable angina and NSTEMI.^[9]

Table showed that mean CK-MB (U/L) and SD in study and control group was 46.340, 10.460 and 17.915, 2.815 respectively. The DF and t-value was 98 and 13.990 respectively. CK-MB is not as significant as troponin-I because its p-value was 0.0001**. These results are similar to research by Newby L. Kristin et al in 2006 which concluded that in patients with non-ST elevation ACS, an elevated troponin is associated with an increased risk of death regardless of the CKMB value, but an elevated CKMB alone is of little prognostic value.^[10]

CONCLUSION

The mean Troponin-I concentration was found to be increased significantly in MI subjects as compared to control group. The increased serum Troponin-I level is due to cardiac muscle damage because of hypoxia which release troponin protein in blood stream. Troponin-I is highly significant and most sensitive cardiac biomarker. There was significant increase in CK-MB level in MI subjects as compare to control group. But an elevated CK-MB alone is of little prognostic value.

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