International Journal of Science and Research (IJSR)
ISSN (Online): 2319-7064
Impact Factor (2012): 3.358

Role of CK-MB and Troponin-I in Diagnosing Non-ST-Elevation Myocardial Infarction

Kiran R Bagale[1], Avinash S. Ingle[2]

[1]Department of Biochemistry, Pt. Jawaharlal Nehru Medical College, Jail Road, Raipur, India

[2]Department of Physiology, All India Institute of Medical sciences, GE Road, Tatibandh, Raipur, India

Abstract: ***It is now well known that, as compared to all other cardiac markers the cardiac Troponin-I and CK-MB are found to be more reliable for the diagnosis of myocardial infarction (MI). The aim of our study was to assess the comparative role of CK-MB and Troponin-I in the diagnosis of non-ST elevation MI (NSTEMI) where there is minimal cardiac damage. Design: The study comprised of 50 patients presenting with history of chest pain and no ST segment elevation on electrocardiogram. Troponin-I was estimated on admission and CK-MB was measured 12 hrs after admission by commercially available kit. The patients were divided into two groups i.e. Troponin-I positive and Troponin-I negative. Results: The Troponin-I was positive in 17 (34%) patients but CK-MB was elevated in 12 (24%) patients. The 5 (30%) patients were Troponin-I positive but CK-MB within normal limit. Conclusion: Therefore it can be concluded that Troponin-I can identify the minimal cardiac damage which will be useful for the physician to start immediate intervention.***

Keywords: Troponin-I, CK-MB, Non-ST-elevation MI, Unstable angina

1. Introduction

Coronary artery disease is a major health risk in most of the western population and is now becoming major cause of death in many developing countries like India with one in ten patients dying of myocardial infarction. As per projections of the Global Burden of Disease study, the burden of cardiovascular disease in India by the year 2020 will be the highest in world [1].

Ischemia refers to lack of oxygen due to inadequate perfusion of the myocardium, which causes an imbalance between oxygen supply and demand. The most common cause of myocardial ischemia is obstructive atherosclerosis of epicardial coronary arteries. Patients with ischemic heart disease fall into two large groups i.e. patients with stable angina secondary to chronic coronary artery disease and patients with acute coronary syndrome. Acute coronary syndrome encompasses patients with ST elevation myocardial infarction on their presenting ECG & those with unstable angina and non-ST elevation myocardial infarction.

Unstable angina is defined as angina pectoris (or equivalent type of discomfort) with at least one of the following features

1. Occurring at rest or at minimal exertion usually lasting for more than 20 min (if not interrupted by nitroglycerin administration).
2. Being severe & described as frank pain or of new onset (within one month).
3. Occurring with crescendo pattern (more severe, prolonged and frequent than the previous one).

The diagnosis of NSTEMI is established if patient with clinical features of unstable angina develop evidence of myocardial necrosis. Unstable angina and non-ST elevation myocardial infarction if not detected as well as not treated on time then it may lead to infarction [2].

Previously the enzymes like aspartate transaminase & lactate dehydrogenase have been of much supporting value but the gold standard in detection of myocardial infarction was an elevated level of creatine kinase [3]. The use of creatine kinase was superseded by the use of myocardium specific enzyme CK-MB which is a myocardium specific isoenzyme of creatine kinase enzyme. This marker satisfies the criteria for diagnosing myocardial infarction (MI) as proposed by WHO, European Cardiac Society [ECS], and American College Of Cardiology [ACC] for defining acute myocardial infarction there should be elevated levels of troponin in presence of appropriate clinical features [4].

Recently it has been documented that there is increase incidence of non-ST-elevation myocardial infarction with fewer patients presenting with classical acute myocardial infarction. So as per the suggestions of National Academy of Clinical Biochemists (NACB), while defining non-ST elevation myocardial infarction, ECG changes plus elevated troponin levels should be considered [5].

Certain markers like lactate dehydrogenase [LDH], aspartate transaminase [AST] and creatine phosphokinase [CPK] are already been replaced by Troponin-T and CK-MB. In some studies it has been noted that the Troponin-I has a potential to replace CK-MB as a cardiac marker. So the elevated levels of which can be useful to distinguish non-ST-elevation myocardial infarction from unstable angina [6].

Therefore the purpose of present study is to compare the role of CK-MB and Troponin-I for the diagnosis of Non ST elevation myocardial infarction.

Licensed Under Creative Commons Attribution CC BY

2. Material

The present study was approved by ethical committee of Narayana Medical College, Nellore. The study population included 50 patients admitted to intensive coronary care unit of the Medical College. Informed consent was taken from the patients. Both males and females of age between 35-65 years were included. Patient's case notes were inspected to establish the time of onset of chest pain and time of presentation to the hospital.

Inclusion criteria: The diagnosis of non-ST elevation myocardial infarction was made in patients who presented with retrosternal chest pain radiating or not to the neck, jaw, epicardium, shoulder, left arm with a sudden onset and duration of 30 minutes or more. The ST-segment elevation which is one of the important features of myocardial infarction was absent in on admission ECG of a patient.

Exclusion criteria: The patients presenting on admission with ST elevation in any one of the twelve lead ECG, trauma, sepsis, renal failure, pulmonary embolism, muscular dystrophy, skeletal muscle injury were excluded from the study.

The blood sample was collected on admission for the estimation of Troponin-I. The blood was allowed to clot for 2 hours. Then the serum was separated by centrifugation for 10 minutes. Hemolysed and lipemic samples were avoided. Troponin-I was estimated by Hexagon Troponin plus kit [7]. It detects cardiac Troponin-I with sensitivity limit minimum of 0.5ng/ml. The second blood sample was collected after 12 hours to measure CK-MB levels. It was measured by immunoinhibition method by using Humastar 300 semi-autoanalyser. The CK-MB up to 25 units/ liter was considered normal (cut of point-25 units / liter). The patient who's Troponin-I was positive was diagnosed as NSTEMI and those with Troponin-I negative results were diagnosed as unstable angina.

3. Results

Total of 50 patients were studied, out of which 17 patients [34%] were Troponin-I positive and 33 patients [66%] were Troponin-I negative. CK-MB was elevated in 12 out of 17 Troponin-I positive cases where as 5 cases were Troponin-I positive and CK-MB within normal limit.

Data evaluation was done using SPSS programme by using student's 't' test. The results were expressed as a mean standard deviation. The p-value was used to compare between two groups. The p-value of < 0.05 was considered to be significant.

Table 1: Patients demographic characteristics

Particular	Troponin-I positive (n=17)	Troponin-I negative (n=33)	Total (n=50)
Age (Yrs.)	56.94 ± 7.79	51.85 ± 7.99	-
Male	14 (28%)	22 (44%)	36(72%)
Female	3 (6%)	11(22%)	14(28%)

The mean value of CK-MB was higher in Troponin-I positive group as compared to Troponin-I negative group, statistically significant difference was found (p-value<0.0001).

Table 2: CK-MB in Troponin-I groups

Particular	*Troponin-I positive*	*Troponin-I negative*	*P Value*
	Mean ± SD (n=17)	Mean ± SD (n=33)	
CK-MB (U/L)	102.53 ± 47.19	20.61 ± 7.31	<0.0001

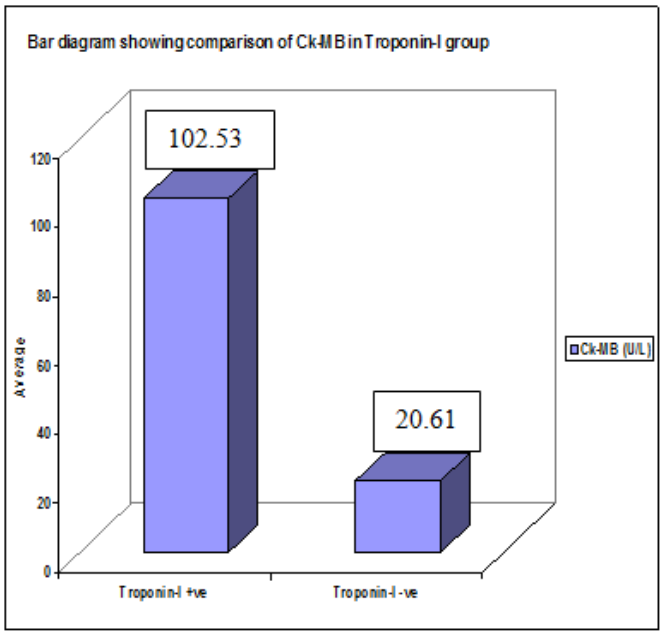


Figure 1: CK-MB levels in troponin-I group

4. Discussion

The early phase of Coronary artery disease with minimal myocardial damage usually manifested as unstable angina with no ST elevation in Electrocardiogram. In such cases biochemical cardiac markers play an important role to detect necrosis. Enzyme markers such as AST, LDH and CPK were used in the past but currently they are not used because their levels get affected by many other physiological and pathological factors. In our study Troponin-I was positive and CK-MB was within normal limit in 5 cases which account to be about 30% of positive cases.

This indicates that Troponin-I is more sensitive indicator for detecting acute myocardial infarction along with unstable angina and NSTEMI [8].

Dawson et al and Harris et al in separate studies have reported that Troponin-I is more specific and sensitive than CK-MB specifically in case of minor myocardial damage [9], [10], [11]. Similar to our findings some researchers like S. Gupta et al and Murray et al also noted that Troponin-I is better indicator as a cardiac marker [12], [13]. Robert et al also observed that troponin help to predict future events in unstable angina cases [14].

This signifies that Troponin-I is a sensitive cardiac marker as compared to CK-MB to detect the less severe myocardial infarction with no ST segment elevation on ECG So it can be ascertained that Troponin-I estimations may be more useful as compared to other cardiac markers in unstable angina cases to establish the status of the disease i.e. whether

Licensed Under Creative Commons Attribution CC BY

the patient with minor myocardial damage has entered in the phase of myocardial necrosis or not.

5. Conclusion

Troponin-I is found to be more useful cardiac marker for the diagnosis of non-ST-elevation myocardial infarction as compared to CK-MB. So it will useful for the early diagnosis and treatment of the patient.

6. Future Scope

As the present work is limited the larger studies are required in future, proving the specificity and sensitivity of Troponin-I to support our findings.

References

[1] Rajiv Gupta et al, Coronary artery disease epidemiology in India: lessons learnt and future directions. Cardiology today, vol. no.4 July-August 2001:225 -232.
[2] Harrison's principles of Internal medicine. Kasper, Fraucy, Braunwald et al, Ischemic Heart Disease.16th edition. vol.2:1434-1447.
[3] Jack H. Landenson et al, A personal history of markers of myocyte injury. clinica chemica acta, 2007,381:3-8.
[4] Paul O Collinson et al, British cardiac society on redefinition of myocardial infarction: a basis for discussion. Ann. of clinical biochemistry; 2004, 41:258-259.
[5] Kaa Fox, J. Birkhed et al, British cardiac society group on the definition of myocardial infarction. Ann. of clinical biochemistry, 2004:4-6.
[6] Paul O Collinson Frances G Boa et al Measurement of cardiac troponin-I.Ann of cl biochemistry2001:3-8.
[7] Jaffe A S, Lindahal B et al Sensitive Troponin-I assay in patients with suspected acute coronary syndrome J AM M Association.2011:306 [5];488-489.
[8] Sidney B Rosalky, Roberts et al, Cardiac biomarkers for detection of myocardial infarction: perspective from past to present, clinical chemistry 2004;50: (11): 2205-2213.
[9] Dawsons S E. The cardiac troponins AACE Review, clinical chemistry, 1997; 43; (s6):- 106-108.
[10] Fred S. Apple et al, Improved detection of minor ischemic myocardial injury with measurement of serum cardiac Troponin-I. clinical chemistry 1997; 43; 11. 2047-2051.
[11] B.M. Harris et al, Comparison of cardiac troponin-T and Troponin-I and CK-MB for the detection of minor myocardial damage during interventional cardiac procedures, analysis of clinical biochemistry; 37:764-769.
[12] S. Gupta, K .N. Singh. Diagnosis of acute myocardial infarction: CK-MB versus cardiac troponin-T in Indian patients, Indian journal of clinical Biochemistry, 2008, 23 (1) 89-91.
[13] Murray A. Varat, Donald W. Mercer. Cardiac specific creatine phosphokinase isoenzyme in the diagnosis of acute myocardial infarction, Circulation, May 1975;51.
[14] Robert J. D. Winter et al, Prognostic value of troponin-T, myoglobin and CK-MB mass in patients presenting with chest pain without myocardial infarction. Heart, 1996; 75:235-239

Author Profile



Dr Kiran R. Bagale (MBBS, MD, Biochemistry) has obtained the MBBS degree from ACPM medical college, Dhule, MS, India in 2004 and MD from NMC Nellore in 2009. She has worked as assistant professor in ASRAM Eluru, AP, GMC Dhule and GMC Yavatmal MS, India. She is currently working as an assistant professor in Pt. J.N. medical college, Raipur, Chhattisgarh, India.

Dr. Avinash S. Ingle, (MBBS, MD Physiology), has obtained MBBS from PDMMC Amaravati, and MD from TNMC Mumbai, MS, India in 2003. Currently working as an associate professor at All India Institute of Medical sciences Raipur, Chhattisgarh, India.

Licensed Under Creative Commons Attribution CC BY