International Journal of Medical and Health Research
ISSN: 2454-9142
Impact Factor: RJIF 5.54
www.medicalsciencejournal.com
Volume 4; Issue 3; March 2018; Page No. 188-190

Study of troponin-T, troponin-I and CK-MB in acute myocardial infarction

Dr. Santosh Kumar[1], Dr. Archana Bharti[2], Dr. Mani Bhushan[3], Dr. Sude Kumar Singh[5]
[1] MBBS MD, Tutor, Department of Biochemistry, Darbhanga Medical college and Hospital, Darbhanga, Bihar, India
[2] MBBS MD, Ex-Senior Resident, Department of Biochemistry, AIIMS Patna, Bihar, India
[3] MBBS MD, Assistant Professor, Department of Pediatrics, ANMCH Gaya, Bihar, India
[4] MBBS MD, HOD, Department of Biochemistry, Darbhanga Medical college and Hospital, Darbhanga, Bihar, India

Abstract
Measuring cardiac biomarkers can be a step toward making a diagnosis for a condition. Whereas cardiac imaging often confirms a diagnosis, simpler and less expensive cardiac biomarker measurements can advise a physician whether more complicated or invasive procedures are warranted.
The study group includes the 25 patients with symptoms of myocardial infarction and history of vascular complications. The control group includes the 25 non-hypertensive patients with no renal failure, no diarrhoea, novomiting and with no history of myocardial infarction. The estimation of the Troponin T, Troponin I & CKMB was done by various biochemical technique.
Hence from the above study it is found that appropriate study of cardiac markers like Total CPK, CPK-MB, LDH, SGOT, cTn-T, cTn-I is significant for the study and evaluation of myocardial infarction. Males are at higher risk than female for cardiac disease and also from my study it is found that cTn –T is the reliable markers. Troponin-T is the best cardiac marker for detection of Myocardial Infarction (MI).

Keywords: cardiac markets, myocardial infarction, troponin T, troponin I & CKMB

Introduction
Acute 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 most common symptom is chest pain or discomfort which may travel into the shoulder, arm, back, neck, or jaw. Often it occurs in the center or left side of the chest and lasts for more than a few minutes. The discomfort may occasionally feel like heartburn. Other symptoms may include shortness of breath, nausea, feeling faint, a cold sweat, or feeling tired [1]. About 30% of people have atypical symptoms. Women more often have atypical symptoms than men. Among those over 75 years old, about 5% have had an MI with little or no history of symptoms. An MI may cause heart failure, an irregular heartbeat, cardiogenic shock, or cardiac arrest [2].
There are a number of different biomarkers used to determine the presence of cardiac muscle damage. Troponins, measured through a blood test, are considered to be the best, and are preferred because they have greater sensitivity and specificity for measuring injury to the heart muscle than other tests [3]. A rise in troponin occurs within 2–3 hours of injury to the heart muscle, and peaks within 1–2 days. The level of the troponin, as well as a change over time, are useful in measuring and diagnosing or excluding myocardial infarctions, and the diagnostic accuracy of troponin testing is improving over time. One high-sensitivity cardiac troponin is able to rule out a heart attack as long as the ECG is normal [4].
Other tests, such as CK-MB or myoglobin, are discouraged [69]. CK-MB is not as specific as troponins for acute myocardial injury, and may be elevated with past cardiac surgery, inflammation or electrical cardioversion; it rises within 4–8 hours and returns to normal within 2–3 days. Copeptin may be useful to rule out MI rapidly when used along with troponin.
Cardiac markers are used in the diagnosis and risk stratification of patients with chest pain and suspected acute coronary syndrome (ACS). The cardiac troponins, in particular, have become the cardiac markers of choice for patients with ACS. Cardiac markers are biomarkers measured to evaluate heart function. They are often discussed in the context of myocardial infarction, but other conditions can lead to an elevation in cardiac marker level. Most of the early markers identified were enzymes, and as a result, the term "cardiac enzymes" is sometimes used. However, not all of the markers currently used are enzymes. For example, in formal usage, troponin would not be listed as a cardiac enzyme [1].
Measuring cardiac biomarkers can be a step toward making a diagnosis for a condition. Whereas cardiac imaging often confirms a diagnosis, simpler and less expensive cardiac biomarker measurements can advise a physician whether more complicated or invasive procedures are warranted. In many cases medical societies advise doctors to make biomarker measurements an initial testing strategy especially for patients at low risk of cardiac death.
Many acute cardiac marker IVD products are targeted at nontraditional markets, e.g., the hospital ER instead of traditional hospital or clinical laboratory environments. Competition in the development of cardiac marker diagnostic products and their expansion into new markets is intense. Recently, the intentional destruction of myocardium by

alcohol septal ablation has led to the identification of additional potential markers [5].

Table 1

Test	Sensitivity and specificity	Approximate peak	Description
Troponin test	The most sensitive and specific test for myocardial damage. Because it has increased specificity compared with CK-MB, troponin is a superior marker for myocardial injury.	12 hours	Troponin is released during MI from the cytosolic pool of the myocytes. Its subsequent release is prolonged with degradation of actin and myosin filaments. Isoforms of the protein, T and I, are specific to myocardium. Differential diagnosis of troponin elevation includes acute infarction, severe pulmonary embolism causing acute right heart overload, heart failure, myocarditis. Troponins can also calculate infarct size but the peak must be measured in the 3rd day. After myocyte injury, troponin is released in 2–4 hours and persists for up to 7 days.
Creatine Kinase (CK-MB) test	It is relatively specific when skeletal muscle damage is not present.	10–24 hours	The CK-MB isoform of creatine kinase is expressed in heart muscle. It resides in the cytosol and facilitates movement of high energy phosphates into and out of mitochondria. Since it has a short duration, it cannot be used for late diagnosis of acute MI but can be used to suggest infarct extension if levels rise again. This is usually back to normal within 2–3 days.

Improper diagnosis of patients with chest pain often leads to inappropriate admission of patients without myocardial infarction and vice versa. In addition to clinical history, physical examination, accurate electro cardiogram findings and assessment of cardiac biomarkers have an important role in the early diagnosis of acute ischemia. Hence based on above literature findings the present study was planned to assess the details of various cardiac biomarkers released during the event of a myocardial infarction.

Methodology

This study was conducted in patients admitted in Darbhanga Medical college and Hospital, Total 50 patients were enrolled into the study. The approval of the institutional ethic committee had been taken before the study. All the patients were informed consent. The aim and the objective of the study are conveyed to all patients. The 50 patients were divided in two group as study group and control group.

The study group includes the 25 patients with symptoms of myocardial infarction and history of vascular complications. The control group includes the 25 non-hypertensive patients with no renal failure, no diarrhoea, no vomiting and with no history of myocardial infarction.

Estimation of serum cardiac Troponin T as done by chemiluminescence immunoassay (CLIA) on Lumax hormone analyser. Creatine kinase (CKMB) was evaluated by kinetic kit method, urea by diacetymonoxime and creatinine by Jiffs kit method, sodium and potassium were measured by flame photometry (Bio-Lab Diagnostic Kit).Serum troponin I concentration was measured between 12 - 48 hours after the onset of chest pain by enzyme linked Immuno sorbent assay using polyvinyl micro titre plates (micro-ELISA).

Results & Discussion

The data from the total 50 patients were collected and presented as below. The 25 cases of the myocardial infarction patients and 25 non hypertensive patients were evaluated of the present study.

Table 2: Sex and Age distribution

	Control Group	Myocardial infarction group
Male	18	20
Female	7	5
Age group	35- 60 years	36 – 58 years

Table 3: Levels of the Cardiac Markers in Study group

	Myocardial infarction group	Control Group
Troponin T	12.6 ± 3.6 ng/ml	0.3 ± 0.15 ng/ml
Troponin I	Positive	Negative
CKMB	235.3 ± 56.5 IU/L	6.3 ± 2.5 IU/L
Urea	34.5 ± 18	16.5 ± 2.1
Creatinine	1.2 ± 0.8	0.75 ± 0.12
Sodium	130 ± 3.8	145 ± 2.3
Potassium	3.8 ± 0.7	4.6 ± 0.15

From the above generated data level of the Troponin T and CKMB is increased significantly in the patients suffered from the myocardial infarctions. The levels of the other serum markers like Urea, and Creatinine is also showing significant increase in there levels. The levels of the serum electrolytes were decreased in the myocardial infarction patients.

In the present study, the established markers, Trop. T&CKMB were significantly elevated in AMI as in previou studies [6-7]. This study showed serum sodium levels significantly decreased ($P<0.0001$) in AMI cases comparedto normal healthy group. These observations are inconsistent with that of Hadeel Rashid Faraj [8]. Rakesh Mudarraddi [9] and Flear and Singh [10]. In AMI no osmotic release of vasopressin may occur due to acute development of left ventricular dysfunction due to pain &stress or may be due to use of analgesics or diuretics.

The level of serum potassium found to be low ($P<0.0001$) in AMI cases than that of healthy group. This is in accordance with Vinod Wail and Singi Yatira jstudies [11]. Such lowering of potassium is an acute stress effect and is due to shift of potassium from extracellular to intracellular space and is a result of stimulation space and is a result of stimulation of beta

-2 adrenoceptor agonis linked to sodium – potassium ATPase [12]. Urea is the end product of protein metabolism. Rise of serum urea is observed in AMI cases of this study. Only few studies are done about association of urea with AMI. Elevated levels of urea indicate renal response to systemic hypoperfusion with respect to reduced cardiac output in decompensated heart failure [13]. The elevated levels of serum creatinine are associated with impaired myocardial flow [14]. as seen in this study. From this study we concluded in patients with AMI hyponatremia was evident with statistical significance. The routine measurement of renal function test (Urea & Creatinine) along with electrolytes (Sodium Potassium) thus can be used as adjuvant, affordable markers in diagnosis and treatment of AMI. Further studies are needed to establish the utility of these markers as effective cardiac biomarkers.

Conclusion

Hence from the above study it is found that appropriate study of cardiac markers like Total CPK, CPK-MB, LDH, SGOT, cTn-T, cTn-I is significant for the study and evaluation of myocardial infarction. Males are at higher risk than female for cardiac disease and also from my study it is found that cTn –T is the reliable markers. Troponin-T is the best cardiac marker for detection of Myocardial Infarction (MI).

References

1. What Are the Signs and Symptoms of Coronary Heart Disease? www.nhlbi.nih.gov. Archived from the original on Retrieved, 2014-2015.
2. What Is a Heart Attack? www.nhlbi.nih.gov. December 17, 2013. Archived from the original on. Retrieved, 2015.
3. Reed Grant W, Rossi Jeffrey E, Cannon Christopher P. Acute myocardial infarction". The Lancet. 2017; 389(10065): 97–210. doi:10.1016/S0140-6736(16)30677-8.
4. Chapman AR, Lee KK, McAllister DA, Cullen L, Greenslade JH, Parsonage W, et al. Association of High-Sensitivity Cardiac Troponin I Concentration With Cardiac Outcomes in Patients With Suspected Acute Coronary Syndrome". JAMA. 2017; 318(19):1913-1924. doi:10.1001/jama.2017.17488. PMC 5710293 Freely accessible. PMID 29127948.
5. Rao SP, Miller S, Rosenbaum R, Lakier JB. Cardiac troponin I and cardiac enzymes after electrophysiologic studies, ablations, and defibrillator implantations". Am. J. Cardiol. 1999; 84(4):470, A9. doi:10.1016/S0002-9149(99)00337-9. PMID 10468091.
6. Ralf Labugger, Lenny organ, et al. Extensive Tropinin I and T modification detected in serum of patients with AMI. Circulation 2000: 102;1221-1226
7. Mohammed A, Al-Otaiby, Hussein S, Al - Amri, Abdulrahaman M. Moghairi Journal of Saudi Heart Association. 2011; 23:3-11.
8. Haded Rashid Faraj. Clinical study of someelectolytes with patients in acute syndrome (ACS) in Thi-Qur Governorate, Iraq. Int. J. Curr. Microiol. App. Sci. 2015; 4(3):700-705.
9. Rakesh Mudarraddi, Shrirang Pralhad Kulkarni, Dhiraj Jayantilal Trivedi. Association of serum electrolytes and urea levels with cardiac markers in AMI. International Journal of Clinical Biochemistry & Research. 2015; 2(4):233-235.
10. Flear CTG, Singh C M. - Hyponatremia and sickcells. Br. J. Anesth. 1973; 45:976.
11. Vinod Wali, Singi Yatiraj. Study of serumsodium & potassium in Acute Myocardial infarction, Journal of clinical and Diagnostic Research. 2014 8(11):cc07- cc09.
12. Diuretics, Hypokalemia Cardiac arrhythmias, Acritical analysis, Vasilios Papademetrious, Am Heart Journal. 1986; 111:1217-24.
13. Doron Aronson, Haim Hammerman, *et al*. Serumblood urea nitrogen & long term mortality in Acute ST-elevation myocardial infarction, International Journal of Cardiology. 2008; 127:380-385.
14. Zhao L, *et al*. Elevated admission serum creatinine predicts poor myocardial blood flow and one-year mortality in ST- segment elevation myocardial infarction patients undergoing primary percutaneous coronary intervention.