Scholars Journal of Applied Medical Sciences
Abbreviated Key Title: Sch J App Med Sci
ISSN 2347-954X (Print) | ISSN 2320-6691 (Online)
Journal homepage: https://saspublishers.com/sjams/

OPEN ACCESS

Medicine

"Cardiac Troponin I Level in STEMI and Clinical Correlation with Left Ventricular Dysfunction in Bangladeshi Population"

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DOI: 10.36347/sjams.2020.v08i03.030 | **Received:** 04.02.2020 | **Accepted:** 22.02.2020 | **Published:** 19.03.2020

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Abstract **Original Research Article**

Background: Coronary Heart Disease (CHD) is the most common category of the heart disease and is found to be the single most important cause that leads to premature death in the developed world. Recognizing a patient with ACS is important because the diagnosis triggers both triage and management. cTnI is 100% tissue-specific for the myocardium and it has shown itself as a very sensitive and specific marker for AMI. ***Objective:*** To determine the relationship of serum troponin I after first acute myocardial infarction with left ventricular ejection fraction as assessed by echocardiography. ***Methods:*** A total of 40 patients of acute myocardial infarction were included in the study. Troponin I concentration was measured by ELISA method and echocardiographic ejection fraction was calculated by modified Simpson's rule. Echocardiographic ejection fraction was compared with serum troponin I concentration. Patients with previous myocardial infarction were excluded. ***Results:*** There was strong negative correlation between troponin I concentration and left ventricular ejection fraction with an increasing troponin level, there was a fall in ejection fraction. The Pearson's correlation coefficient was –0.69, which was statistically significant ($p<0.0001$). In our study, we observed that patients with ejection fraction >50%, though small in number were having cTnI levels at 24 hrs ≤ 8 ng/ml. Patients with ejection fraction <50% (left ventricular systolic dysfunction) were having cTnI levels at 24 hrs ≥ 17 ng/ml. Therefore a presumptive cut off level of cTnI ≤ 8 ng/ml may be taken to consider normal left ventricular systolic function in STEMI. The normal range of Troponin I in apparently health individual without STEMI was observed to be <1.0 ng/ml. The mild increase in Troponin I at 24 hrs of STEMI with preserved EF >50% may be due to peak value of biomarker achieved at 24-36 hrs after myocardial injury as most of troponin I are attached to myofibrils. **Conclusion:** Serum troponin I concentration has a strong negative correlation with left ventricular ejection fraction after first acute myocardial infarction, and hence can be used to assess the LVEF in patients with first myocardial infarction. An observation was made that a cut off level of cTnI ≤ 8 ng/ml was associated with normal left ventricular systolic function.

Keywords: left ventricular systolic dysfunction, Serum troponin I, cTnI levels, left ventricular ejection fraction.

INTRODUCTION

Coronary Heart Disease (CHD) is the most common category of the heart disease and is found to be the single most important cause that leads to premature death in the developed world. Over the last decade, cardiovascular disease (CVD) has become the single largest cause of death worldwide. Asian countries have considerably higher prevalence of premature Coronary Artery Disease (CAD) and standardized mortality rates for CAD compared with Europeans, Chinese and Malays [1-4]. Over the last four decades there has been a 10-fold increase in the prevalence of coronary artery disease in urban area of Bangladesh. The overall rate of Coronary artery disease was 11.0% in Chennai Urban population [5]. Cardiovascular disease is becoming significant burden on health care services in Bangladesh [39]. Acute Coronary Syndrome (ACS) refers to the spectrum of clinical presentations ranging from ST-Segment

Elevation Myocardial Infarction (STEMI) to non-ST-segment elevation myocardial infarction (NSTEMI) to unstable angina (i.e., acute coronary syndrome without release of enzymes or biomarkers of myocardial necrosis) [6]. As per the Revised Definition of Myocardial Infarction (MI), either of the following satisfies the Criteria for acute, evolving, or recent MI include: 1- Typical rise and gradual fall (troponin) or more rapid rise and fall Creatine Kinase (CK-MB) of biochemical markers of myocardial necrosis with at least one of the following. (a) Ischemic symptoms (b) Development of pathologic Q waves on the ECG reading. (c) ECG changes indicative of ischemia (ST=segment elevation or depression). (d) Coronary artery intervention (e.g. coronary angioplasty). 2-Pathological findings of an acute MI [7]. ECG Criteria for a STEMI is - New ST elevation at the J-point in two contiguous leads with the cut – off points: $\geq$ 0.2 mV in men or $\geq$ 0.15 mV in women in leads V-V_3 and / or $\geq$ 0.1 mV in other leads in the absence of LVH/LBBB [8]. Ventricular function is the best predictor of death after an acute coronary syndrome. It serves as a marker of myocardial damage, provides information on systolic function as well as diagnosis and the prognosis [9,10]. Troponin is a globular protein of muscle that binds to tropomyosin and has a marked affinity for calcium ions, and is thus a central regulatory protein of muscle contraction. The troponin, a protein-complex, consists of three subunits with different structure and functions (T, I, C) [11-13]. Troponin I is a 23.5 kDa component of Troponin complex that inhibits the interaction of myosin cross-bridges with the actin-tropomysoin complex, and thus regulates the striated muscle contraction [13]. Three isoforms of Tnl have been described, a cardiac (cTnI) and two skeletal muscle [slow twitch (sTnI) and fast twitch (fTnI)] [14]. Each of the three Tnl isoforms is encoded by three different genes located on different chromosomes [15]. The skeletal isoforms show approximately 40% heterogeneity of primary sequence, while the cardiac isoform displays a similar degree of sequence heterogeneity compared to each skeletal isoform. Due to the presence of an additional 31 amino acids at the N-terminal region, cTnI (MW 24,000 Da) is uniquely different than either fTnI or sTnI (MW 19,800 Da). During human development, both sTnI and cTnI are expressed in the myocardium. At birth, however, only cTnI is expressed in the myocardium [16]. cTnI has been shown not to be expressed in any type of skeletal muscle, independent of developmental or disease stimuli [17]. Therefore, knowledge that cTnI is 100% tissue-specific for the myocardium. cTnI has been shown to be a very sensitive and specific marker for acute myocardial infarction (AMI) [18-21]. The early release kinetics for cTnI is similar to those of creatine kinase (CK) MB, in that it takes 4-8h to increase above the upper reference limit. Thus, cTnI does not provide an earlier detection method for AMI than CK MB [22]. The initial cTnI rise is from the release of 3 to 6% cytoplasmic fraction of troponin in the cell following ischemic injury [20]. cTnI peaks between 14 and 36 h after onset of AMI and remains elevated for five to seven days after AMI. The mechanism for the lengthy time for elevations of cTnI is most likely due to the ongoing release of troponin from the 95 to 97% myofibril-bound fraction. The ongoing release and clearance thus gives the impression that cTnI has a long half-life. However, the true half-life of cTnI is less than 2 h [23].

LITERATURE REVIEW

According to international consensus and task force definitions of myocardial infarction (MI), the diagnosis of MI is based mainly on an elevated cardiac troponin level exceeding the 99th percentile and demonstrating an increase or decrease over time [7,8]. The universal definition recommends the use of a more sensitive troponin assay with a coefficient of variation of 10% or less at the diagnostic cutoff concentration representing the 99th percentile of a reference population [8]. The major limitation of standard cardiac troponin assays is their relatively low clinical sensitivity at the time of ED presentation owing to the troponin release kinetics and the time it takes for increased concentrations to reach the circulation. Consequently, the diagnosis of AMI requires prolonged monitoring with serial determination of cardiac biomarkers over 6-9h after ED presentation. This contributes to delays in initiating treatment as well as ED overcrowding and the need for unnecessary admissions to rule out myocardial infarction. It is now recognized that the major predictor of long-term survival after recovery from acute myocardial infarction is the functional status of the left ventricle. Left ventricular function has usually been described in terms of the ejection fraction (EF), but it is not clear whether EF is the most meaningful index of left ventricular function in the post infarction situation [24-27]. Low EF may, on the one hand, be caused by poor contractile function due to extensive myocardial damage or continuing ischemia or, on the other hand, to left ventricular dilation caused by infarct expansion and stretching of the myocardial scar. Thus End-Systolic Volume (ESV) or End-Diastolic Volume (EDV) might be more meaningful predictors of prognosis than EF, which is merely an arithmetical term based on these two values. If a sufficient quantity of myocardium undergoes ischemic injury, left ventricular pump function (left ventricular ejection fraction) becomes depressed; cardiac output, stroke volume, blood pressure, and peak dP/dt are reduced and end-systolic volume is increased [28]. The degree to which end-systolic volume is increased perhaps is the most powerful predictor of mortality following STEMI [29]. Left ventricular systolic dysfunction and its myocardial damage can be assessed by 2.D-echocardiography. In acute STEMI troponin I value shows an inverse correlation with left ventricular ejection fraction [7]. The present study plan to analyse the relationship between peak troponin I level after STEMI and left

ventricular systolic dysfunction determined by 2.D-echocardiography. The aim of the study is to measure the troponin I in acute ST-elevation myocardial infarction, assessment of left ventricular dysfunction by echocardiography in acute ST-elevation myocardial infarction and co-relation between troponin I levels and left ventricular dysfunction in acute ST- elevation myocardial infarction. The special investigations added are Echocardiography- Left ventricular ejection fraction was assessed by modified Simpson's method from Apical two chamber and Four chamber views,16 segment regional wall motion abnormalities was determined in Parasternal long axis, Parasternal short axis apical four chambers and apical two chamber views by 2D echocardiography and M-Mode echocardiography [30].

Echocardiograms were obtained using Philips, Sonus echocardiography machine with the subjects lying in the left lateral decubitus position and supine position. The assessment of cardiac valve structures, chamber size evaluation, Left Ventricular features, LVIDd (Left Ventricular Internal dimension in diastole, LVIDs(Left Ventricular Internal dimension in systole), LVEF (Left Ventricular Ejection fraction), Left Ventricular wall motion abnormalities, Fractional Shortening, LVEF (Left Ventricular ejection fraction). The ejection fraction is defined as the ratio of stroke volume to end-diastolic volume. Normal values of left ventricular ejection fraction are 0.55 to 0.75 when determined by echocardiography and angiocardiography. There are no gender differences, but ejection fraction normally declines with age. LVEF less than 50% was taken as systolic dysfunction [31]. Left ventricular ejection fraction will be calculated by 2.D-echocardiography at 48 to 72 hours after the onset of clinical symptoms. Left ventricular ejection fraction will be assessed by modified Simpson's method from apical two chamber and four chamber views [32].16 segment regional wall motion abnormality was determined in parasternal long axis, parasternal short axis apical four chamber and apical two chamber views by 2D echocardiography and M-Mode echocardiography. Echocardiograms will be obtained using Philips, Sonus echocardiography machine with the subjects lying in the left lateral decubitus position and supine position.

MATERIAL AND METHODS

The study was conduct in the department of Cardiology, National Institute of Cardiovascular Diseases and Hospital, Dhaka, Bangladesh during the period of January 2019 to June 2019. Study group consisted of 40 patients hospitalized for 1st acute ST-Elevation myocardial Infarction. Consent was taken from all the patients prior to inclusion in the study. Inclusion criteria's are, age 20 to 60 years, both male and female, case of acute onset myocardial infarction as diagnosed by clinical presentation, symptom of ischaemia lasting >30 minute Retrostermal chest pressure, burning, or heaviness; radiating occasionally to neck jaw, epigastrium, shoulders, or left arm. ECG characteristics - New ST elevation at the J-point in two contiguous leads with the cut–off points: ≥0.2 mV in men or ≥ 0.15 mV in women leads V2-V3 and / or ≥ 0.1 mV in other leads in the absence of LVH/ LBBB [5]. Biochemical cardiac Marker (Increased CK-MB, Troponin I)

Exclusion criteria are a known case of old myocardial infarction, a patient having pre-existing ECG changes s/o old MI. ECG showing Q wave at the time of admission. Echocardiographic finding of old scar/ previous wall motion abnormalities / structural heart disease / congenital heart disease. All those conditions in which Troponin.

I also increase without Ischaemic heart disease: significant renal impairement, rheumatoid arthritis, myocarditis (pericarditis), sepsis, acute congestive heart failure, acute pulmonary embolism, or prolonged tachyarrhythmias.

Study Design

All admitted patients 40 (forty) of Acute ST-elevation MI were included in the prospective study. The protocol included. Complete history and clinical assessment with investigations with baseline and special investigations including echocardiography.

Blood Sample Collection

On admission, 10 ml of peripheral venous blood was collected from the antecubital vein by an autoclaved syringe using 20 gauze needles. The blood was allowed to clot at room temperature for at least half an hour. The glass tube with clotted blood was centrifuged at 2000 rpm for 20 minutes and the centrifugation was repeated once more to remove the red cells completely. The supernatant serum, devoid of cellular elements, was separated from the clot and placed in two acid cleaned small test tubes. Samples with any visible haemolysis were discarded, as erythrocytes contain large quantities of enzymes which are known to alter the total CPK, CPK-MB, LDH and Troponin I. Anticoagulants were avoided to circumvent unnecessary variables which might interfere with accurate assays of enzyme activity.

Principle of the Assay

The cTnl ELISA test is based on the principle of a solid phase enzyme-linked immunosorbent assay. The assay system utilizes four unique monoclonal antibodies directed against distinct antigenic determinants on the molecule. Three mouse monoclonal anti-troponin I antibodies are used for solid phase immobilization (on the microtiter wells). The fourth antibody is in the antibody-enzyme (horseradish peroxidase) conjugate solution. The test sample is allowed to react simultaneously with the four antibodies, resulting in the troponin I molecules being

sandwiched between the solid phase and enzymelinked antibodies. After 90-minute incubation at room temperature, the wells are washed with water to remove unbound-labeled antibodies. A solution of Tetramethylbenzidine (TMB) Reagent is added and incubated for 20 minutes, resulting in the development of a blue color. The color development is stopped with the addition of 1N Hydrochloric Acid (HCl) changing the color to yellow. The concentration of troponin I is directly proportional to the color intensity of the test sample. Absorbance is measured spectrophotometrically at 450 nm. The routine investigations done are complete haemogram and ESR, Blood Sugar, Blood urea serum creatinine, Lipid profile; Total cholesterol, serum triglyceride, HDL Cardiac enzyme CPK, CK-MB, LDH, SGOT done by chemiluminescence (orthoclinical diagnostics). Troponin I at 6 hrs. or at the time of admission and 24 hrs was done by Calbiotech Troponin I ELISA kit with EVOLIS (Biorad) instrument. All data analysis Windows SPSS Version 21.0.

RESULTS

The study was carried out in a study group of consecutive 40 inpatients in the department of Cardiology, National Institute of Cardiovascular Diseases and Hospital, Dhaka, Bangladesh satisfying the selection criteria (as per inclusion and exclusion criteria laid down). Mean age of the patients in present study was 49.15 ± 8.28. Most of the patients in our study were in age group 41-60 yrs (82.5%). 92.5% of patients were male and only 7.5% were female. 72% of patients had AWMI and 28% patients had IWMI, 52.5% patients had dyslipidemia. It was found to be most common risk factor. Smoking (47.80%) and Hypertension (24.32%) were other significant risk factors, mainly in male patients. Risk factors were much more common in AWMI as compared to IWMI patients. On comparison of AWMI and IWMI, it was found that mean values of SBP, DBP and PR were significantly lower in IWMI patients as compared to AWMI patients (p value<.05) [Tables 1 and 2].

Table-1: Comparison of parameters in AWMI and IWMI (N=40)

Parameter	Group I	Group-II	P- value
BMI	29.756 ± 25.928	25.329 ± 3.383	0.57
SBP	110.41 ± 13.173	99.82 ± 5.689	0.014
DBP	67.93 ± 6.199	61.82 ± 3.027	0.003
PR	72.83 ± 4.29	63.45 ± 4.803	0.000
RR	17.55 ± 1.75	17.55 ± 1.508	0.368
Hb%	13.545 ± 0.734	13.491 ± 0.89	0.846
Blood Urea	37.66 ± 16.946	35.09 ± 8.40	0.636
Serum Creatinine	0.775 ± 0.298	0.809 ± 0.250	0.745
CPK	634.38 ± 377.53	473.09 ± 165.72	0.182
CK-MB	224.07 ± 144.649	156.00 ± 66.71	0.144
LDH	2671.76 ± 1460.87	1978.64 ± 673.02	0.141
SGOT	215.90 ± 117.196	193.73 ± 34.67	0.364
T.Cholesterol	195.59 ± 57.82	194.82 ± 66.65	0.971
HDL Cholesterol	34.86 ± 6.30	33.91 ± 4.32	0.591
LDL Cholesterol	77.76 ± 30.56	85.46 ± 25.90	0.464
VLDL	43.62 ± 19.12	43.60 ± 1.86	0.927
Triglycerides	148.62 ± 52.87	145.82 ± 52.09	0.855

Table-2: Comparison of left ventricular parameters in AWMI and IWMI (N=40)

Parameter	Group I	Group-II	P- value
EF (%)	36.10 ± 8.797	41.64 ± 5.390	0.59
EDV(ml)	84.31 ± 20.127	46.27±9.067	0.316
ESV(ml)	55.93 ±18.70	46.27 ± 9.067	0.111
LVIDd(mm)	48.29 ± 6.68	42.4 ± 2.43	0.007
LVIDs(mm)	33.0 ± 3.082	30.64 ± 4.13	0.056

Table-3: Comparison of Troponin I with Steptokinase (STK) Therapy (N=40)

Troponin I	STK Given	STK Not Given
6 hrs (Mean value ± SD)	20.82 ±18.75	12.78 ± 2.40
24 hrs (Mean value ± SD)	69.68 ± 43.28	19.33 ± 2.05

Table-4: Correlation of Troponin I and Ejection Fraction (N=40)

Cardiac Enzyme	Pearson Correlation	P Value
Troponin I At 6 hrs/Admission	─0.269	0.094
Troponin I At 24 Hrs	─0.628	0.0001

The mean values of TROP-I at 24 hour was significantly higher as compared to mean value of TROP-I at 6 hour. The mean value of TROP-I at 6 hour and 24 hour were similar in both group AWMI as well as IWMI.

The mean values of EF were 37.6%. 37 patients out of 40 patients had LV dysfunction. On comparison of left ventricular echocardiography parameter- LVIDd and LVIDs were significantly lower in IWMI patients as compared to AWMI patients [Tables 3 and 4]. All the 37 patients of LV dysfunction had higher value of TROP-I. Troponin I, as expected, was raised in all cases and the mean cTnI value at 6 hr/admission was 20.21 ± 18.35 and at 24 hr. was 66.94 ± 43.53 [Table 4]. In the present study positive predictive value of TROP-I was 92.5%.

Table-5: Studies of correlation of Troponin I Sensitivity for LV dysfunction (N=40)

Troponin I Sensitivity For Lv Dysfunction	Dysfunction	Study
96.6%	94.4%	Adams *et al*. [35]
96%	93%	De winter *et al.*
67%	76%	bodi *et al*. [33]
100%	92.4%	Somani *et al*. [34]
100%	100%	present study

Table-6: Studies of presence of LV dysfunction (N=40)

Study	Total No. of patient	Presence of LV Dysfunction	Percentage
Vincet *et al*.	60	20	33.3%
Deepak *et al*.	50	24	48%
Present study	40	37	92.5%

In the present study mean value of TROP-1 in LV Dysfunction patient was 66.94 ± 43.53 [Table 6]. The mean value of TROP-I was increasing as EF was decreasing. The mean values of TROP-I was much higher in patients in which EF was less than 30%. An observation was made that a cut off level of cTnI ≤ 8 ng/ml was associated with normal left ventricular systolic function as 3 patients with >50% EF. The normal range of Troponin I in apparently health individual without STEMI was observed to be<1.0 ng/ml. The mild increase in Troponin I at 24 hrs of STEMI with preserved EF >50% may be due to peak value of biomarker achieved at 24-36 hrs after myocardial injury as most of troponinI are attached to myofibrils.

DISCUSSION

The present study entitled Assessment of left ventricular dysfunction by Echocardiography and its correlation with quantitative troponin I level in Acute ST-elevation myocardial infarction' was conducted in 40 STEMI in-patients in the department of Cardiology, National Institute of Cardiovascular Diseases and Hospital, Dhaka, Bangladesh. CTnI is accepted as a highly reliable biochemical marker for detecting myocardial damage, and its use in the diagnosis of acute myocardial infarction is increasing. Data suggests that cTnI may be related to the amount of myocardial damage and cTnI release inversely correlates with left ventricular ejection fraction and infarct size. However, there are very few studies to substantiate the claim. In patients with ST segment elevation and in which ejection fraction analysis was delayed, observed that troponin was a good indicator of depressed ejection fraction [33]. The relationship between peak troponin and systolic function in patients without ST segment elevation, however, has received little attention in the literature. In the present study, mean age of patients was 49.5 yrs. The no. of male patients presenting with chest pain were more than no. of female and M: F ration was 12.3:1. In similar study Vincet *et al*. observed 3.28:1. This observation was in consonance with the fact that the incidence of myocardial infarction is more in male than in the female and there are certain risk factors which are more commonly seen in males (smoking, alcohol intake) [33]. In the present study most common risk factor in the patients were dyslipidemia (52.4%), similar observation was reported by Deepak *et al.* (54%) in his study. HTN were observed in 25% of STEMI Patients in the present study; however Deepak *et al*. found slightly higher percentage at 32% and Vishwanathan *et al*. 43% [34]. Diabetes Mellitus was present in only 2.5% of patients in present study which was small compared to other studies. Difference might be because of younger patients in the present study and because of small sample size. In present study 29 patients (72%) out of 40, sustained an anterior wall MI and 11 patients (28%) out of 40 STEMI sustained inferior wall MI. The mean value of EF was 37.63 ± 8.32 (Table 4). In a similar study on 50 patients of first acute myocardial infarction, Sharkey *et al*. observed mean EF 37.6 ± 15.2%. In our study 92.5% patients were thrombolysed and 7.5% patients were non-thrombolysed. Our study shows Troponin I values at 24 hrs is 69.68 ± 43.28 in thrombolysed patients and 19.33 ± 2.0 in non- thrombolysed patients. (Table 5). The higher release of markers due to washout phenomena

occurring in patients receiving thrombolytic treatment. Troponin I, as expected, was raised in all cases and the mean cTnI value at 6 hr/admission was 20.21 ± 18.35 and at 24 hr. was 66.94 ± 43.53. In the present study positive predictive value of TROP-I was 92.5%. However previous worker reported 89% Vincet *et al*., 100% Deepak *et al*. This observation was similar to the previous study. In the present study mean value of TROP-1 in LV Dysfunction patient was 66.94 ± 43.53. In the present study LV Dysfunction was present in 92.5% of STEMI patients. However Deepak *et al*. observed LV Dysfunction in only 48% of patient and Vincet *et al*. observed in only 33.3% of patients [33,34]. Difference was observed because the present study has small number of patients which had, multiple risk factor in most of them and compared to western population Bangladeshi population have extensive coronary artery disease. The mean values of TROP-I were much higher in present study as compared to previous study. This could be because large number of our patients had extensive anterior wall myocardial infarction with consequent large infarct size. It has been recognized that the total quantity of cardiac enzyme released correlates with the infarct size. As most of our patients received thrombolytic treatment, higher Troponin I level in part could be due to washout phenomenon occurring in patient receiving thrombolytic treatment. In our study, we observed that patients with ejection fraction>50%, though small in number were having cTnI levels at 24 hrs ≤ 8 ng/ml. Patients with ejection fraction <50% (left ventricular systolic dysfunction) were having cTnI levels at 24 hrs ≥ 17ng/ml. Therefore a cut off level of cTnI ≤ 8 ng/ml may be taken to consider normal left ventricular systolic function in STEMI. We observed strong inverse correlation between peak cTnI concentration and left ventricular ejection fraction. The Pearson correlation coefficient was -0.628 (P<0.0001). Our secondary observation in Bangladeshi subjects is dyslipidemia was the most common risk factor in STEMI patients. LV dysfunction was present in 37 patients out of 40 patients of STEMI. The mean value of TROP-I at 24 hour was much higher as compared to TROP-I at 6 hour. On comparison, echocardiographic parameters- LVIDd and LVIDs, as well as, SBP, DBP and PR were significantly lower in IWMI patients as compared to AWMI patients, though there was no difference between cTn-I values between the two groups. CTnI has practical advantages over other markers in the assessment of left ventricular ejection fraction. After acute infarction, cTnI has a peak value at 12 hours from the onset of pain. The plateau phase of cTnI, however, lasts up to 48 hours, and represents an integrated estimate of myocyte necrosis [35,36]. The peak value will therefore be missed in samples taken 12-48 hours after admission, but there is a large time window. This makes repeated sampling unnecessary, and represents a cost and time-effective method of diagnosis and quantification (Figures 2 and 3). This is in contrast to creatine kinase-MB or myoglobin, for which multiple measurements are required to identify the peak value and whose values are affected by thrombolysis [37,38]. This marker offers a simple, inexpensive, quick noninvasive method of identifying such patients. Estimation of troponin I can also be used to identify those patients who may benefit from other treatments, for example, ACE inhibitors.

CONCLUSION

Serum troponin I concentration has a strong negative correlation with left ventricular ejection fraction after first acute myocardial infarction, and hence can be used to assess the LVEF in patients with first myocardial infarction. An observation was made that a cut off level of cTnI ≤8 ng/ml was associated with normal left ventricular systolic function.

[Abbreviations: TROP I: Troponin I; LVEF: Left Ventricular Ejection Fraction; IWMI: Inferior Wall Myocardial Infarction; AWMI: Anterior Wall Myocardial Infarction EF=EDV-ESV/EDVX100 (%), EF=Ejection fraction, EDV= end-diastolic volume and ESV =end-systolic volume.]

REFERENCES

1. Enas EA, Garg A, Davidson MA, Nair VM, Huet BA, Yusuf S. Coronary heart disease and its risk factors in first-generation immigrant Asian Indians to the United States of America. Indian heart journal. 1996;48(4):343-53.
2. Hughes K, Yeo PP, Lun KC, Sothy SP, Thai AC, Wang KW, Cheah JS. Ischaemic heart disease and its risk factors in Singapore in comparison with other countries. Annals of the Academy of Medicine, Singapore. 1989 May;18(3):245-9.
3. Anand SS, Yusuf S, Vuksan V, Devanesen S, Teo KK, Montague PA, Kelemen L, Yi C, Lonn E, Gerstein H, Hegele RA. Differences in risk factors, atherosclerosis, and cardiovascular disease between ethnic groups in Canada: the Study of Health Assessment and Risk in Ethnic groups (SHARE). The lancet. 2000 Jul 22;356(9226):279-84.
4. Balarajan R. Ethnic differences in mortality from ischaemic heart disease and cerebrovascular disease in England and Wales. Bmj. 1991 Mar 9;302(6776):560-4.
5. Mohan V, Deepa R, Rani SS, Premalatha G. Prevalence of coronary artery disease and its relationship to lipids in a selected population in South India: The Chennai Urban Population Study (CUPS No. 5). Journal of the American College of Cardiology. 2001 Sep 1;38(3):682-7.
6. Anderson JL, Adams CD, Antman EM, Bridges CR, Califf RM, Casey DE, Chavey WE, Fesmire FM, Hochman JS, Levin TN, Lincoff AM. ACC/AHA 2007 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction—Executive Summary: A Report of the American College of Cardiology/American Heart Association Task

Force on Practice Guidelines (Writing Committee to Revise the 2002 Guidelines for the Management of Patients With Unstable Angina/Non–ST-Elevation Myocardial Infarction) Developed in Collaboration with the American College of Emergency Physicians, the Society for Cardiovascular Angiography and .... Journal of the American College of Cardiology. 2007 Aug 14;50(7):652-726.

7. Alpert J, Thygesen K, Antman E, Bassand JP. for the Joint European Society of Cardiology/American College of Cardiology Committee. Myocardial infarction redefined–a consensus document of the Joint European Society of Cardiology/American College of Cardiology Committee for the redefinition of myocardial infarction. J Am Coll Cardiol. 2000 Sep;36(3):959-69.
8. Thygesen K. Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. Universal definition of myocardial infarction. Eur Heart J. 2007;28:2525-38.
9. Rao AC, Collinson PO, Canepa-Anson R, Joseph SP. Troponin T measurement after myocardial infarction can identify left ventricular ejection of less than 40%. Heart. 1998 Sep 1;80(3):223-5.
10. Antman EM. Troponin measurements in ischemic heart disease: more than just a black and white picture; 2001.
11. Katus HA, Schoffold T, RemppisA, Comples Lab Med.1992; 23: 311-317.
12. Paul RJ, Ferguson DG, Heiny JA. Muscle Physiology-Molecular Mechanism in Physiology Ed. Nocholas S, Robert O, Banks, Little Brown and Co. Bosto;1993.
13. Solaro JR, Van Eyk J. Altered interactions among thin filament proteins modulate cardiac function. Journal of molecular and cellular cardiology. 1996 Feb 1;28(2):217-30.
14. Ebashi S, WAKABAYASHI T, EBASHI F. Troponin and its components. The Journal of Biochemistry. 1971 Feb 25;69(2):441-5.
15. Barton PJ, Bhavsar PK, Brand NJ, Chan-Thomas PS, Dabhade NI, Farza HE, Townsend PJ, Yacoub MH. Gene expression during cardiac development. InSymposia of the Society for Experimental Biology 1992 (Vol. 46, pp. 251-264).
16. Bhavsar PK, Dhoot GK, Cumming DV, Butler-Browne GS, Yacoub MH, Barton PJ. Developmental expression of troponin I isoforms in fetal human heart. FEBS letters. 1991 Nov 4;292(1-2):5-8.
17. Bodor GS, Porterfield D, Voss EM, Smith S, Apple FS. Cardiac troponin-I is not expressed in fetal and healthy or diseased adult human skeletal muscle tissue. Clinical Chemistry. 1995 Dec 1;41(12):1710-5.
18. Cummins B, Auckland ML, Cummins P. Cardiac-specific troponin-l radioimmunoassay in the diagnosis of acute myocardial infarction. American heart journal. 1987 Jun 1;113(6):1333-44.
19. Larue C, Calzolari C, Bertinchant JP, Leclercq F, Grolleau R, Pau B. Cardiac-specific immunoenzymometric assay of troponin I in the early phase of acute myocardial infarction. Clinical chemistry. 1993 Jun 1;39(6):972-9.
20. Bodor GS, Porter S, Landt Y, Ladenson JH. Development of monoclonal antibodies for an assay of cardiac troponin-I and preliminary results in suspected cases of myocardial infarction. Clinical chemistry. 1992 Nov 1;38(11):2203-14.
21. Adams 3rd JE, Schechtman KB, Landt Y, Ladenson JH, Jaffe AS. Comparable detection of acute myocardial infarction by creatine kinase MB isoenzyme and cardiac troponin I. Clinical chemistry. 1994 Jul 1;40(7):1291-5.
22. Tucker JF, Collins RA, Anderson AJ, Hauser J, Kalas J, Apple FS. Early diagnostic efficiency of cardiac troponin I and troponin T for acute myocardial infarction. Academic Emergency Medicine. 1997 Jan;4(1):13-21.
23. Jaffe AS, Landt Y, Parvin CA, Abendschein DR, Geltman EM, Ladenson JH. Comparative sensitivity of cardiac troponin I and lactate dehydrogenase isoenzymes for diagnosing acute myocardial infarction. Clinical chemistry. 1996 Nov 1;42(11):1770-6.
24. Taylor GJ, Humphries JO, Mellits ED, Pitt BE, Schulze RA, Griffith LS, Achuff SC. Predictors of clinical course, coronary anatomy and left ventricular function after recovery from acute myocardial infarction. Circulation. 1980 Nov;62(5):960-70.
25. Sanz G, Castaner A, Betriu A, Magrina J, Roig E, Coll S, Pare J, Navarro-Lopez F. Determinants of prognosis in survivors of myocardial infarction: a prospective clinical angiographic study. New England Journal of Medicine. 1982 May 6;306(18):1065-70.
26. De Feyter PJ, Van Eenige MJ, Dighton DH, Visser FC, De Jong J, Roos JP. Prognostic value of exercise testing, coronary angiography and left ventriculography 6--8 weeks after myocardial infarction. Circulation. 1982 Sep;66(3):527-36.
27. Roubin GS, Harris PJ, Bernstein LO, Kelly DT. Coronary anatomy and prognosis after myocardial infarction in patients 60 years of age and younger. Circulation. 1983 Apr;67(4):743-9.
28. Forrester JS, Wyatt HL, Da Luz PL, Tyberg JV, Diamond GA, Swan HJ. Functional significance of regional ischemic contraction abnormalities. Circulation. 1976 Jul;54(1):64-70.
29. White HD, Norris RM, Brown MA, Brandt PW, Whitlock RM, Wild CJ. Left ventricular end-systolic volume as the major determinant of survival after recovery from myocardial infarction. Circulation. 1987 Jul;76(1):44-51.

30. Antman E, Bassand JP, Klein W, Ohman M, Sendon JL, Rydén L, Simoons M, Tendera M. Myocardial infarction redefined—a consensus document of the Joint European Society of Cardiology/American College of Cardiology committee for the redefinition of myocardial infarction: the Joint European Society of Cardiology/American College of Cardiology Committee. Journal of the American College of Cardiology. 2000 Sep 1;36(3):959-69.
31. Schiller NB, Shah PM, Crawford M, DeMaria A, Devereux R, Feigenbaum H, Gutgesell H, Reichek N, Sahn D, Schnittger I, Silverman NH. Recommendations for quantitation of the left ventricle by two-dimensional echocardiography. Journal of the American Society of Echocardiography. 1989 Sep 1;2(5):358-67.
32. Robert O. Bonow, Douglas L. Mann, Douglas P. Zipes, Peter Libby. William F Armstrong Echocardiography In : Zipes Libby Bonow Braunwald. Braunwald's Heart Disease a Textbook of Cardiovascular Medicine (7th ed.) Elseivier Saunders, USA.2006.
33. Bodí V, Núñez J, Sanchis J, Llàcer À, Fácila L, Chorro FJ. Usefulness of troponin I for predicting systolic dysfunction in acute coronary syndromes. Results of a prospective and quantitative study. Revista Española de Cardiología (English Edition). 2003 Jul 1;56(7):738-41.
34. Somani D, Gahlot RS, Lakhotia M, Choudhary CR, Sangavi S. Troponin I measurement after myocardial infarction and its correlation with left ventricular ejection fraction: a prospective study. Age (yrs). 2005;55:9.
35. Adams JE, Sicard GA, Allen BT, Bridwell KH, Lenke LG, Davila-Roman VG, Bodor GS, Ladenson JH, Jaffe AS. Diagnosis of perioperative myocardial infarction with measurement of cardiac troponin I. New England Journal of Medicine. 1994 Mar 10;330(10):670-4.
36. Katus HA, Remppis A, Scheffold T, Diederich KW, Kuebler W. Intracellular compartmentation of cardiac troponin T and its release kinetics in patients with reperfused and nonreperfused myocardial infarction. The American journal of cardiology. 1991 Jun 15;67(16):1360-7.
37. McCullough DA, Harrison PG, Forshall JM, Irving JB, Hillman RJ. Serum myoglobin and creatine kinase enzymes in acute myocardial infarction treated with Anistreplase. Journal of clinical pathology. 1992 May 1;45(5):405-7.
38. Grande P, Granborg J, Clemmensen P, Sevilla DC, Wagner NB, Wagner GS. Indices of reperfusion in patients with acute myocardial infarction using characteristics of the CK-MB time-activity curve. American heart journal. 1991 Aug 1;122(2):400-8.
39. Pencina MJ, D'Agostino Sr RB, Steyerberg EW. Extensions of net reclassification improvement calculations to measure usefulness of new biomarkers. Statistics in medicine. 2011 Jan 15;30(1):11-21.