

Original Research Article

Troponin I measurement after acute myocardial infarction and its correlation with left ventricular ejection fraction

Mohita Shah*, Prafful Kothari

Department of General Medicine, SMIMER, Surat, Gujarat, India

Received: 04 December 2021

Accepted: 03 January 2022

***Correspondence:**

Dr. Mohita Shah,

E-mail: mohita.shah15@gmail.com

Copyright: © the author(s), publisher and licensee Medip Academy. This is an open-access article distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.

ABSTRACT

Background: After acute myocardial infarction (MI), a patient's prognosis is closely related to the extent of irreversibly damaged myocardium. The evaluation of infarct size after acute MI (AMD) is important for predicting the subsequent clinical course. Cardiac troponin I (cTnI) is accepted as a highly reliable biochemical marker for detecting myocardial damage, and its use in the diagnosis of acute MI (AMI) is increasing. Its concentration is unaffected by thrombolysis after the first 12 hours, following which it shows a stable plateau for about 48 hours.

Methods: This study investigated the value of a single cTnI concentration, estimated 12-48 hours after admission, to provide an integrated measurement of the degree of cardiac damage following first acute MI, and its correlation with left ventricular ejection fraction (LVEF). This study of troponin I measurement after acute MI and its correlation with LVEF was conducted during the period between October 2019 and October 2021 at SMIMER hospital, Surat.

Results: This study shows a strong negative correlation between cTnI concentration measured between 12-48 hours post MI and echocardiographic LVEF. It was also found that cTnI concentration more than 3.8 ng/ml is a sensitive (100%) and specific (78.12%) indicator of LVEF <40% after a first acute MI. It can be considered as a significant prognostic marker.

Conclusions: In conclusion, cTnI shows excellent promise as a marker of infarct size, and for the assessment of LVEF; and may potentially replace the CPK-MB as the cardiac specific marker for AMI detection.

Keywords: MI, cTnI, LVEF

INTRODUCTION

After acute MI, a patient's prognosis is closely related to the extent of irreversibly damaged myocardium. The evaluation of infarct size after acute MI (AMD) is important for predicting the subsequent clinical course and to validate the effectiveness and clinical relevance of therapeutic interventions. In routine clinical practice, infarct size is estimated non-invasively by electrocardiographic mapping and scoring. imaging techniques (such as echocardiography, myocardial radionuclide imaging and magnetic resonance imaging), and serological tests enzymatic measurements).¹

Left ventricular function is the best individual predictor of mortality after acute MI. Late mortality and morbidity after MI are improved by treatment with angiotensin converting enzyme (ACE) inhibitors in patients whose infarction is complicated by heart failure and in patients whose ejection fraction is <40% LVEF may be the most easily assessed measurement of left ventricular function, and this measurement is extremely useful for risk stratification.²

Estimation of LVEF requires echocardiography or contrast or radionuclide left ventriculography echocardiography is probably the most widely available of the three methods but echocardiograms good enough

for ejection fraction measurement are not possible in all patients. Contrast and radionuclide left ventriculography are more expensive and are not widely available in district hospitals.³

Further prognostic information can be obtained by the accurate assessment of end-systolic volume, which is superior to ejection fraction for prediction of survival following AMI. In patients with low LVEF, the measurement of exercise capacity is useful for further identifying those patients at particularly high risk.

Biochemical markers of myocardial damage have long been used to estimate infarct size and closely correlate with five-year mortality. Infarct size can be estimated from creatine kinase MB and myoglobin, but repeated estimations are required during a small-time window'. Moreover, it is infarct size rather than residual left ventricular function that is measured, although they are related. Furthermore, thrombolytic treatment profoundly alters the kinetics of conventional cardiac markers. The increased release of creatine kinase after reperfusion is such that peak creatine kinase activity after thrombolysis can no longer be used as even an approximate indicator of myocardial infarct size. While infarct size will affect left ventricular function, the direct relation between creatine kinase or myoglobin and LVEF has not been assessed".

Recently, serum troponin T has emerged as a specific indicator of myocardial damage in acute MI '. cTnl T release closely relates to infarct size, but its relation to LVEF has not been determined.⁴ Serum troponin T concentration may inversely correlate with LVEF as a consequence of the inverse relation between infarct size and LVEF.

Cardiac troponin I (cTnl) is accepted as a highly reliable biochemical marker for detecting myocardial damage, and its use in the diagnosis of acute MI (AMI) is increasing". Unlike creatine kinase and myoglobin, its concentration is unaffected by thrombolysis after the first 12 hours, following which it shows a stable plateau for about 48 hours.

Objective

Objectives of the study was to find correlation between S. troponin I and LVEF after MI (anterior and inferior wall).

METHODS

This study investigated the value of a single troponin I (cTnl) concentration, estimated 12-48 hours after admission, to provide an integrated measurement of the degree of cardiac damage following first acute MI, and its correlation with LVEF.⁵

This study of troponin I measurement after acute MI and its correlation with LVEF was conducted during the

period between October 2019 and October 2021 at SMIMER hospital, Surat.

Study population

Study population consisted of 50 patients with first acute MI diagnosed by clinical symptoms and signs, 12 lead ECG and biochemical markers (CK-MB).

Inclusion criteria

Acute MI was diagnosed if at least two of the following criteria were present and they included in the study: Cardiac chest pain, ST segment elevation of at least 2 mm in chest leads or 1 mm in limb leads and raised creatine kinase MB activity.^{6,7}

Exclusion criteria

Patients with significant renal impairment, history of heart failure (due to any cause) and previous MI were excluded from study.

Methodology used

After admission detailed history and clinical examination was carried out.

Following investigations were done in all patients: Hemogram, urine examination, random blood sugar, blood urea, serum creatinine, ECG, S.CPK-MB, S. troponin I and S. cholesterol.

Serum troponin I concentration was measured between the 12-48 hours after the onset of chest pain by the microparticle enzyme immunoassay (MEIA) on AXSYM System.⁸

At present there is no WHO standardization for troponin I due to the use of different troponin I antibodies in the test. In our study, the test kit was calibrated with Abbott's AXSYM troponin I ADV. The diagnostic cut off for the AMI patient was determined to be 0.4 ng/ml.

Echocardiography and tissue Doppler imaging patients were imaged in the left lateral decubitus position using a commercially available system (high resolution colour Doppler vivid 7 ultrasound machine). Images were obtained using a 3.5-MHz transducer, at a depth of 16 cm in the parasternal and apical views (standard long-axis and two-and four-chamber images).⁹

Left ventricular volumes (end-systolic, end-diastolic) and LVEF were calculated from the conventional apical two-and four-chamber images, using the biplane Simpson's technique.

Ethical approval was not required for the study along with a signed patient consent form in local language, for performing 2D ECHO and blood reports.

Study type was of observational cross-sectional study.

RESULTS

This Table shows the sensitivity and specificity of cTnI to predict LVEF in case of anterior wall MI. It was found that cTnI concentration >3.8 ng/ml predicted LVEF of <40% with a sensitivity of 100% and specificity of 66.67%.¹⁰

Table 1: Relation to LVEF in anterior wall MI.

cTnI (ng/ml)	LVEF <40%	LVEF >40%	Total
>3.8	14	05	19
<3.8	00	10	10
Total	14	15	29

Table 2: cTnI level in relation to LVEF in inferior wall MI.

cTnI (ng/ml)	LVEF <40%	LVEF >40%	Total
>3.8	04	02	06
<3.8	00	15	15
Total	04	17	21

This Table shows the sensitivity and specificity of cTnI to predict LVEF in case of inferior wall MI. It was found that cTnI concentration <3.8 ng/ml predicted LVEF of <40% with a sensitivity of 100% and specificity of 88.23%.¹¹

Final results

The 58% patients of AMI had anterior wall affection and 42% patients had inferior wall MI.

The 48% patients of anterior wall MI have LVEF less than 40%. While only 19% patients of inferior wall MI have LVEF less than 40%.

This study shows a strong negative correlation between cTnI concentration measured between 12-48 hours post MI and echocardiographic LVEF.¹²

It was also found that cTnI concentration more than 3.8 ng/ml is a sensitive (100%) and specific (78.12%) indicator of LVEF <40% after a first acute MI. It can be considered as a significant prognostic marker.

DISCUSSION

This study shows a strong negative correlation between cTnI concentration measured between 12-48 hours post MI and echocardiographic LVEF. It was also found that cTnI concentration more than 4.0 ng/ml is a sensitive (100%) and specific (78.12%) indicator of LVEF <40% after a first acute MI.

These results were little different to the results obtained by Somani et al.¹⁰ They evaluated 50 patients of AMI at Jodhpur medical college determine the relationship of serum troponin I after first acute MI with LVEF as assessed by echocardiography. Echocardiographic ejection fraction (measured by modified Simpson's rule) was compared with serum cTnI concentration (measured by ELISA method between 12-48 hrs after admission). There was a strong negative correlation between cTnI concentration and LVEF after first acute MI. It was also found that cTnI >6.6 ng/ml predicted LVEF <50% with a sensitivity of 100% and specificity of 92.4%.

Table 3: Comparative studies.

Variables	Present study (cTnI>4 ng/ml to detect LVEF <40%)	Somani et al study (cTnI>6.6 ng/ml to detect LVEF <50%) ¹⁰	Panteghini et al study (cTnI>14.8 µg/L to detect LVEF <40%) ⁸
Sensitivity	100	100	100
Specificity	78.12	92.4	65

The results are almost similar to Panteghini et al study, CTnI measurements were performed 12 and 48 h after admission in 63 consecutive AMI patients.⁸ LV function was evaluated by gated single photon emission computed tomography (SPECT) and infarct size was estimated by CK-MB peak and SPECT myocardial perfusion. Significant correlations were found between cTnI at 12 and the perfusion defect size at SPECT. CTnI at 12 and 48 h were inversely related to LV ejection fraction (LVEF). cTnI >14.8 µg/L at 48 h predicted an LVEF <40% at 3 months with a sensitivity of 100%, specificity of 65%, and a negative predictive value of 100%

Limitation

A large study sample size is ideally required for statistical significance of the results, their implications and the validity to extrapolate results and to suggest recommendations on the basis of the same. It was a point study and so the results could not be used to prognosticate as it would require long term prospective trial.

The 2D ECHO calculation of LVEF was done, which is always smaller than that determined by angiography, thus chances of technical error are there. In present study, patients of only first attack of acute MI were included. So prognostic importance of cTnI could not be measured in subsequent attacks of MI.

CONCLUSION

In conclusion, cTnI shows excellent promise as a marker of infarct size, and for the assessment of LVEF; and may

potentially replace the CPK-MB as the cardiac specific marker for MI detection.

CTnI provide a reasonable approach to select patients with left ventricular dysfunction who may require further interventions. Estimation of cTnI can also be used to identify those patients who may benefit from other treatments.

Funding: No funding sources

Conflict of interest: None declared

Ethical approval: The study was approved by the Institutional Ethics Committee

REFERENCES

1. Mair J, Friendrich L, Galzolari C. cTnI release correlates with myocardial infarct size. *Eur J Clin Biochem.* 1996;335(18):1342.
2. Geltman EM, Ehanni AA, Campbell MK. The influence of location and extent of myocardial infarction on long term ventricular contractile dysfunction and mortality. *Circulation.* 1976;60:805-14.
3. The acute infarction ramipril efficacy (AIRE) study Investigators. Effect of ramipril on mortality and morbidity of survivors of acute myocardial infarction with clinical evidence of heart failure. *Lancet.* 1993;342:821-8.
4. Nicolosi GL, Latini R, Marino P. The prognostic value of predischARGE quantitative two-dimensional echocardiographic measurements and the effects of early lisinopril treatment on left ventricular structure and function after acute myocardial infarction in the GISSI-3 trial. Gruppo Italiano per lo Studio della Sopravvivenza nell'infarcto Miocardico. *Eur Heart J.* 1996;17:1646.
5. Adams JE III, Abendschein DR, Jaffe AS. Biochemical markers of myocardial injury. Is MB creatine kinase the choice for the 1990s? *Circulation.* 1993;88:750.
6. Adams JE III, Schechtman KB, Landt Y. Comparable detection of acute myocardial infarction by creatine kinase MB isoenzyme and cardiac troponin I. *Clin Chem.* 1994;40:1291.
7. Katus HA, Remppis A, Scheffold T. Intracellular compartmentation of cardiac troponin T and its release kinetics in patients with reper fused and non reperfused myocardial infarction. *Am J Cardiol.* 1991;67:1360.
8. Panteghini M, Bonetti G, Pagani F, Stefini F, Giubbini R, Cuccia C. Measurement of troponin I 48 hours after admission as a tool to rule out impaired left ventricular function in patient with a first myocardial infarction. *Clin Chem Lab Med.* 2005;43(8):848-54.
9. Mair J, Friendrich L, Galzolari C. cTnI release correlates with myocardial infarct size. *Eur J Clin Biochemistry.* 1996;335(18):1342
10. Somani D, Gahlot RS. Troponin I measurement after myocardial infarction and its correlation with left ventricular ejection fraction: a prospective study. *JIACM.* 2005;6(1):38-41.
11. Sharkey SW. Assessment of left ventricular function using serum cardiac troponin I measurement following myocardial infarction. *Clin Chim Acta.* 1998;272(1):59-67.
12. Hara S, Horinaka S. Cardiac troponin I level correlates with chronic phase left ventricular function after successful direct percutaneous transluminal coronary angioplasty in patients with acute myocardial infarction. *J Cardiol.* 2000;36(5):303-10.

Cite this article as: Shah M, Kothari P. Troponin I measurement after acute myocardial infarction and its correlation with left ventricular ejection fraction. *Int J Adv Med* 2022;9:113-6.