International Journal of Research in Medical Sciences
*Khan MH et al. Int J Res Med Sci. 2019 May;7(5):1392-1398*
www.msjonline.org
pISSN 2320-6071 | eISSN 2320-6012
DOI: http://dx.doi.org/10.18203/2320-6012.ijrms20191623

**Original Research Article**

# Correlation of troponin-I level with left ventricular systolic dysfunction after first attack of non-ST segment elevation myocardial infarction

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**Received:** 06 January 2019
**Revised:** 21 February 2019
**Accepted:** 05 March 2019

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## ABSTRACT

**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. Ventricular function is the best predictor of death after an ACS. It serves as a marker of myocardial damage and provides information on systolic function as well as diagnosis and prognosis. The study aimed at investigating the impact of LVEF on elevated troponin-I level in patients with first attack of NSTEMI.

**Methods:** This cross-sectional analytical study was conducted in the department of cardiology in Mymensingh Medical College Hospital from December, 2015 to November, 2016. Total 130 first attack of NSTEMI patients were included considering inclusion and exclusion criteria. The sample population was divided into two groups: Group-I: Patients with first attack of NSTEMI with LVEF: ≥55%. Group-II: Patients with first attack of NSTEMI with LVEF: <55%. Then LVEF and troponin-I levels were correlated using Pearson's correlation coefficient test.

**Results:** In this study mean troponin-I of group-I and group-II were 5.53±7.43 and 16.46±15.79ng/ml respectively. It was statistically significant ($p<0.05$). The mean LVEF value of groups were 65.31±10.30% and 40.17±4.62% respectively. It was statistically significant ($p<0.05$). The echocardiography showed that patients with high troponin-I level had low LVEF and patients with low troponin-I level had preserved LVEF. Analysis showed that patients with highest level of troponin-I had severe left ventricular systolic dysfunction (LVEF <35%) and vice versa-the patients with the lowest levels of troponin-I had preserved systolic function (LVEF ≥55%). In our study, it also showed that the levels of troponin-I had negative correlation with LVEF levels with medium strength of association ($r= -0.5394$, $p=0.001$). Our study also discovered that Troponin-I level ≥6.6ng/ml is a very sensitive and specific marker for LV systolic dysfunction.

**Conclusions:** The study has enabled the research team to conclude that the higher is the Troponin-I level the lower is the LVEF level and thus more severe is the LV systolic dysfunction in first attack of NSTEMI patients.

**Keywords:** Acute coronary syndrome, Coronary heart disease, Left ventricular dysfunction, LVEF, NSTEMI, Troponin-I

## INTRODUCTION

ACS describes the range of myocardial ischemic states that includes UA, NSTEMI or STEMI. The diagnosis and classification of ACS is based on a thorough review of clinical features, including ECG findings and biochemical markers of myocardial necrosis.[1] The term MI (myocardial infarction) is used when there is evidence of myocardial necrosis in the setting of acute myocardial ischemia. STEMI is differentiated from NSTEMI by the presence of persistent ECG findings of ST segment elevation.[2]

CHD is responsible for more than half of all cardiovascular incidence in individuals. During the past several years, the rates of hospitalization for MI and mortality associated with CHD have decreased. The decline in CHD mortality is partially reflective of the change in the pattern of clinical presentations of ACS.[3]

There has been a substantial reduction in the incidence of STEMI and a subsequent increase in the incidence of NSTEMI.[4] The research team believes that there is room for more improvement in the prevention and management of ACS.

After AMI, a patient's prognosis is closely related to the extent of irreversibly damaged myocardium.[5,6] In routine clinical practice, infarct size is estimated non- invasively by electrocardiography, imaging techniques and serological tests. Ventricular function is the best predictor of death after an ACS. It serves as a marker of myocardial damage, provides information on systolic function as well as diagnosis and prognosis.[7,8]

cTnI is 100% tissue-specific for the myocardium. cTnI has shown to be a very sensitive and specific marker for AMI.[9-12] The early release kinetics for cTnI is similar to those of CK- MB.[13]

cTnI peaks between 14 and 36 hrs after onset of AMI and remains elevated for five to seven days after AMI. The preferred cardiac biomarker is troponin, which has high clinical sensitivity and myocardial tissue specificity.

It is essential to detect a rise and/or fall in cardiac biomarkers to distinguish acute from chronic elevations in troponin concentrations, which may be associated with structural heart disease. Troponin levels should be measured on first assessment, within 6 hours of the onset of pain, and in the 6-12hours after onset of pain.

It is now recognized that the major predictor of long-term survival after recovery from AMI is the functional status of the left ventricle which has usually been described in terms of the LVEF.

The study aimed to measure the troponin I in of those patients, assessment of LVEF by echocardiography in NSTEMI and co-relation between troponin I levels and LVEF.

## METHODS

This study was conducted in the department of Cardiology, Mymensingh medical college hospital, Mymensingh since December 2015 to November 2016. Study population comprised all the patients admitted into Cardiology department with chest pain. Sample population was selected on the basis of brief history, targeted physical examination, ECG, troponin-I level and on the basis of inclusion and exclusion criteria.

### *Inclusion criteria*

Patients with first attack of NSTEMI.

### *Exclusion criteria*

- Patients admitted with acute STEMI.
- Patients had previous history of MI.
- Patients with valvular heart disease, congenital heart disease and cardiomyopathy.
- Patients had major non-cardiovascular disorder causing elevation of Troponin-I such as severe renal impairment, prolonged immobilization, major surgery, chest trauma, myocarditis (pericarditis), acute pulmonary embolism, prolonged tachyarrhythmia.
- Any systemic infection.
- Patients were under chemotherapy on discovery of malignancy.
- Patient not willing to get themselves enrolled in study.

Considering inclusion and exclusion criteria; study population was divided into two groups.

Group-I: Patients with first attack of NSTEMI with LVEF: ≥55%.

Group-II: Patients with first attack of NSTEMI with LVEF: <55%.

Group-II was again subdivided into 03 groups:

- Mild left ventricular systolic dysfunction defined as LVEF: 45-54%.
- Moderate left ventricular systolic dysfunction defined as LVEF: 35-44%.
- Severe left ventricular systolic dysfunction defined as LVEF: <35%.

### *Statistical analysis*

Purposive sampling method was employed. The collected data were analyzed with the aid of computer software SPSS version 20 where quantitative data were expressed

as mean ±SD and Student's "t" test was used for analysis. Qualitative data were analyzed with $\chi 2$ test. Comparison between groups were made by unpaired t-test. Correlation was set up by using Pearson's correlation coefficient test. p value <0.05 was considered. Data was collected through a structured case record form. Data were collected from all respondents by direct face-to-face interviews. Informed written consent was obtained from all participants. The variable parameters like age, sex, smoking, hypertension, diabetes mellitus, dyslipidemia, F/H of CAD, BMI, ECG, troponin-I and LVEF were studied.

## RESULTS

Total sample population were 140. Follow-up of the patient was carried out clinically and by ECG. Among them 04 patients developed STEMI, 04 were discharged earlier on request of the patient, 01 was referred and 01 patient died. Finally, LVEF measurement by echocardiography was done in 130 patients and they were grouped into two groups; Group-I: NSTEMI with LVEF≥55%, n = 26 (male 18, female 8) Group-II: NSTEMI with LVEF<55%, n= 104 (male 77, female 27).

**Table 1: Age distribution of the study population (n=130).**

| Age group (years) | Group-I (n=26) | | Group-II (n=104) | | p-value |
|---|---|---|---|---|---|
| | Number | % | Number | % | |
| 20-30 | 1 | 3.8 | 0 | 0 | |
| 31-40 | 0 | 0 | 12 | 11.5 | |
| 41-50 | 7 | 26.9 | 28 | 26.9 | |
| 51-60 | 12 | 46.2 | 38 | 36.5 | |
| 61-70 | 5 | 19.2 | 16 | 15.4 | |
| 71-80 | 1 | 3.8 | 8 | 7.7 | |
| 81-90 | 0 | 0 | 2 | 1.9 | |
| Mean ±SD | 55.85±10.00 | | 57.18±10.55 | | 0.968$^{ns}$ |

Unpaired t-test was done, ns means not-significant

The Table 1 shows majority of patients were in the age range of 51-60 years in group-I and group II. But 19.2% and 15.4% patients were in 61-70 age group also. The mean age of groups were 55.85±10.00 and 57.18±10.55 years respectively. Analysis revealed no statistical significance (p>0.05).

**Table 2: Gender status of the study population (n=130).**

| Parameters | Group-I (n=26) | Group-II (n=104) | p-Value |
|---|---|---|---|
| **Gender** | | | |
| Male | 18 (69%) | 77 (74%) | 0.621$^{ns}$ |
| Female | 08 (31%) | 27 (26%) | |

Chi square test was done to measure the level of significance

The Table 2 shows sex distribution of the study population which shows majority of the study population were male.

**Table 3: Anthropometric status of the study population (n=130).**

| Parameters | Group-I (n=26) | Group-II (n=104) | p-Value |
|---|---|---|---|
| BMI Range | 18.9-32.5 | 16.5-37.7 | 0.417$^{ns}$ |
| Mean ±SD | 25.07±3.55 | 24.65±4.21 | |

Unpaired t-test was done to measure the level of significance

The observed BMI of the groups were 25.07±3.55 and 24.65±4.21. From Table 3, it was statistically insignificant (p>0.05).

**Table 4: Distribution of risk factors of the study population (n=130).**

| Risk factors | | Study subjects | | | | p-value |
|---|---|---|---|---|---|---|
| | | Group-I (n=26) | | Group-II (n=104) | | |
| | | No. | % | No. | % | |
| Smoking | Smoker | 14 | 53.8 | 88 | 84.6 | 0.78$^{ns}$ |
| | Non-smoker | 12 | 46.2 | 16 | 15.4 | |
| HTN | Yes | 12 | 46.2 | 63 | 60.6 | 0.23$^{ns}$ |
| | No | 14 | 53.8 | 41 | 39.4 | |
| DM | Yes | 15 | 57.7 | 62 | 58.2 | 0.59$^{ns}$ |
| | No | 11 | 42.3 | 42 | 41.8 | |
| F/H CAD | Yes | 06 | 23.1 | 28 | 26.9 | 0.69$^{ns}$ |
| | No | 20 | 76.9 | 76 | 73.1 | |

Chi-square test was done

The Table 4 shows distribution of risk factors of the study population. Considering risk factors, smoking had high percentage in both groups (Group-I, 53.8% and group-II, 84.6%) and those were not statistically significant (p>0.05).

**Table 5: Lipid profile of the study population (n=130).**

| Parameters | Group-I (n=26) (Mean ±SD) | Group-II (n=104) (Mean ±SD) | p-value |
|---|---|---|---|
| TC | 196.81±47.90 | 181.53±51.27 | 0.171$^{ns}$ |
| LDL-C | 124.85±44.40 | 113.20±40.70 | 0.202$^{ns}$ |
| HDL-C | 35.15±7.95 | 34.61±7.53 | 0.743$^{ns}$ |
| TG | 236.81±201.66 | 184.32±95.31 | 0.061$^{ns}$ |

Unpaired t-test was done to measure the level of significance

On the other hand, other risk factors like hypertension, DM and F/H of CAD were also prevalent in both the study groups but also statistically insignificant (p>0.05).

From the lipid profile of the study population it was found that the mean HDL-C and TG levels were elevated with normal mean total cholesterol and LDL-C levels.

But they were also not statistically significant (p>0.05) (Table 5).

**Table 6: ECG patterns of the study population (n=130).**

| ECG | Study Subjects | | | | p-value |
|---|---|---|---|---|---|
| | Group-I (n=26) | | Group-II (n=104) | | |
| | Number | % | Number | % | |
| ST-depression | 14 | 53.8 | 40 | 38.5 | 0.235$^{ns}$ |
| T-inversion | 10 | 38.5 | 44 | 42.3 | |
| Normal | 2 | 7.7 | 20 | 19.2 | |
| Total | 26 | 100.0 | 104 | 100.0 | |

Chi-square test was done

Table 6 shows, type of ECG changes among the study population. Majority (53.8%) in Group-I presented with ST-segment depression. But in Group-II majority (42.3%) presented with T- wave inversion. There was no significant difference of the ECG patterns between the groups.

**Table 7: Troponin-I level of the study population (n=130).**

| Parameter | Group-I (n=26) (Mean ±SD) | Group-II (n=104) (Mean ±SD) | p-value |
|---|---|---|---|
| Troponin-I | 5.53±7.43 | 16.46±15.79 | 0.003$^{s}$ |

Unpaired t-test was done, $^{S}$-Significant

The mean troponin-I of group-II was more than that of group-I. It was statistically significant (p<0.05) (Table 7).

Group-II was again divided into three sub-groups:

- Mild LV systolic dysfunction (45-54%)
- Moderate LV systolic dysfunction (35-44%)
- Severe LV systolic dysfunction (<35%)[16]

**Table 8: Ejection fraction in echocardiography in the NSTEMI with LV systolic dysfunction population (n=104).**

| LVEF in ECHO group-II (n=104) | | |
|---|---|---|
| **Subgroups** | **Number** | **%** |
| 45-54% | 78 | 75 |
| 35-44% | 19 | 18 |
| <35% | 07 | 07 |

The Table 8 shows ejection fraction in echocardiography in the NSTEMI with LV systolic dysfunction group. Majority of patients were with Ejection Fraction 45-54% range in this group.

The Table 9 shows majority of the study subjects of group-I had troponin-I level <6.6ng/ml and majority of the study subjects of group-II had troponin-I level ≥6.6ng/ml. Here, the difference between the two groups was statistically significant (P<0.05).

**Table 9: Comparison of troponin-I level and Left Ventricular Ejection Fraction (LVEF) between the groups (n=130).**

| | Ejection Fraction | | p-value |
|---|---|---|---|
| Troponin-I (ng/ml) | Group-I (n=26) | Group-II (n=104) | <0.00001$^{s}$ |
| ≥6.6 | 03 | 95 | |
| <6.6 | 23 | 09 | |

Chi-Squire test was done

**Table 10: Relationship between troponin-I level and Left Ventricular Ejection Fraction (LVEF) (n=130).**

| Grouping of LVEF (%) | Troponin-I ng/ml (Mean ±SD) | LVEF (Mean ±SD) | p-value |
|---|---|---|---|
| Group-I ≥55% (n=26) | 5.53±7.43 | 65.31±10.30 | 0.001$^{s}$ |
| Group-II 45-54% (n=78) | 15.06±14.50 | 50.39±3.22 | |
| 35-44% (n=19) | 16.52±14.31 | 39.84±3.21 | |
| <35% (n=07) | 17.04±14.74 | 30.29±1.44 | |
| Unpaired t-test | | | |

Patients in the group with severe left ventricular dysfunction (<35%) had the highest level of troponin-I and vice versa-the patients with the preserved systolic function (≥55%) had the lowest levels of troponin-I. Here, the difference between the two groups was statistically significant (P<0.05) (Table 10).

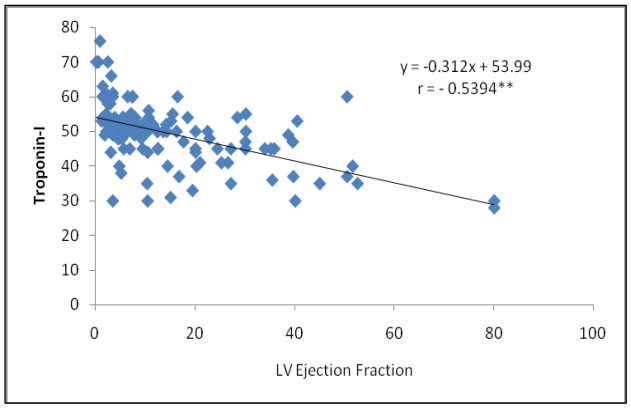


**Figure 1: Correlation between troponin-I and LVEF level of the study population (n=130).**

Figure 1 shows statistically significant moderate negative correlation with medium strength of association – correlation coefficient between troponin-I and LVEF (r = -0.5394, p=0.001) suggesting that the higher was the level of troponin-I, the lower was the LV ejection fraction level in first attack of NSTEMI patients.

The multivariate regression analysis was done for the variables studied which showed regression co-efficient for troponin-I and LVEF were statistically significant ($p<0.05$) but the other parameters revealed no statistical significance (Table 11).

**Table 11: Multivariate regression analysis of the risk factors of the study population (n=130).**

| Parameter | $\beta$ | p-value |
|---|---|---|
| Age | 1 | $0.57^{ns}$ |
| Sex | -0.06 | $0.723^{ns}$ |
| BMI | 0.10 | $0.12^{ns}$ |
| Smoking | 0.142 | $0.813^{ns}$ |
| HTN | -0.194 | $0.325^{ns}$ |
| DM | 0.233 | $0.435^{ns}$ |
| F/H of CAD | -0.005 | $0.565^{ns}$ |
| TLC | -0.229 | $0.113^{ns}$ |
| LDL-C | -0.198 | $0.187^{ns}$ |
| HDL-C | -0.051 | $0.652^{ns}$ |
| TG | -0.213 | $0.091^{ns}$ |
| ECG | 0.004 | $0.324^{ns}$ |
| Troponin-I | 0.261 | $0.002^{s}$ |
| LVEF | -0.182 | $0.001^{s}$ |

Multivariate linear regression analysis was done

## DISCUSSION

Age is an important predictor of survival after AMI.[15] Mean ages of the present study were 55. 85 (±10.00) and 57.18 (±10.55), those were not statistically significant. Majority of patients of both groups belonged to 41-50 age group and 51-60 age group. In Group-I, 07 (26.9%) were of 41-50 years and 12 (46.2%) were of 51-60 years. On the other hand; in Group-II, 28 (26.9%) were in 41-50 years and 38 (36.5%) in 51-60 years. A study of similar type reported the mean age of the patients were 55 (±9) years.[16] This finding is almost similar to the present study.

In our study, out of 130 cases 95 were male, among them 18 (69%) were in Group-I and 77 (74%) were in Group-II. Male-female ratio was 2.7:1. In one study the researchers found, male were 34 and female 16. Therefore, like other studies, males were predominant in our study.[17]

A study in NICVD, Dhaka, reported that the most common risk factor of AMI was smoking and it was 73.33%.[18] In this study smoking status of the study population was 14 (53.8%) in Group-I and 88 (84.6%) in Group-II which was statistically insignificant ($p>0.05$).

DM in patients after AMI was shown as a strong predictor of short- and long-term mortality.[19] In this study, DM was found in group-I 57.7% and in group-II it was 58.2%, which was also statistically insignificant ($p>0.05$).

Hypertension is a recognized risk factor for coronary artery disease and LV systolic dysfunction. In this study hypertensive status of the study population was in group-I, 46.2% and in group-II, 60.6% which was not statistically significant ($p>0.05$).

In Group-I: 14 (53.8%) ST-depression, 10 (38.5%) T-inversion and 02 (7.7%) were presented with normal ECG pattern. In Group-II: 40 (38.5%) ST-depression, 44 (42.3%) T-inversion and 20 (19.2%) were presented with normal ECG pattern. Patients with ST-depression were commonest in Group-I, whereas T-inversion were commonest in Group-II.

In this study mean LVEF value of group-I and group-II were 65.31±10.30% and 40.17±4.62% respectively that was statistically significant ($p<0.05$). On the other hand, the mean troponin-I of group-I and group-II were 5.53±7.43 and 16.46±15.79ng/ml respectively, was also statistically significant ($p<0.05$).

In this study, in group-I: 100% were with EF≥55%. In group-II, 78% were with EF 45-54%, 18% were with EF 35-44% and 7% were with EF<35%. All of the patients in group-I were with EF ≥55% (100%) and majority (78%) were with EF range 45-54% in group II. Here, the difference is statistically significant ($p<0.05$). In this study, all patients were divided according to their LVEF in 4 groups: Group I-preserved LV function (EF: ≥55%), Group II-mild LV dysfunction (EF: 45-54%), Group III-moderate LV dysfunction (EF: 35-44%) and Group IV-severe LV dysfunction (EF: <35%). Patients in the group with severe LV dysfunction had the highest level of troponin-I and vice versa-the patients with the preserved systolic function had the lowest levels of troponin-I. Our data are consistent with those of other study.[20] This finding has implications for the potential use of troponin-I as a marker for LV dysfunction following NSTEMI.

In our study, it was shown that the levels of LVEF had negative correlation with troponin-I with medium strength of association ($r= -0.5394$, $p=0.001$). In literature it was reported that there is negative correlation between LVEF and troponin-I, strength of association was, $r = -0.44$.[21] In a study they found negative correlation of LVEF and troponin-I levels in Acute MI patients, strength of association was $r = -0.269$.[22] All these findings are consistent with our finding.

From the above discussion researchers detected that troponin-I level had a negative correlation with LVEF as well as a very sensitive and specific marker for systolic dysfunction in first attack of NSTEMI patients.

Several limitations of our study must be acknowledged:

- The sample size was small.
- This study was conducted in only one center and majority of the study population were male.

- Troponin-I level estimation have become easier and more sensitive by using the newer methods. Owing to infrastructural constraints, blood sample of patients was analyzed with the aid of traditional technique.
- LVEF assessment with newer techniques have become more reliable. The team opted for only one method.

## CONCLUSION

The present study concluded that the higher was the troponin-I level the lower was the LVEF level in first attack of NSTEMI patients and thus it serves as a very sensitive and specific marker for LV systolic dysfunction.

***Recommendations***

Based on the findings of the study, research team has been able to suggest the following measures for diagnosis, prognosis and treatment of the patients afflicted with NSTEMI.

In perspective of Bangladesh, troponin-I is an available test for making diagnosis and to see prognosis in acute MI patients. Troponin-I level has an impact over LEVF in patients with NSTEMI.

Troponin-I level provides a note of warning about the outcomes of the patients after NSTEMI. A number of studies were conducted in past for AMI patients, mostly on STEMI. Few studies were conducted regarding NSTEMI. As, LVEF was correlated well with troponin-I levels; So, troponin-I alone can serve dual purpose-for both diagnosis and prognosis of NSTEMI Patients.

The study also recommends that aggressive treatment strategy or intervention including early PCI and closer surveillance should be applied to NSTEMI patients with high Troponin-I levels.

For validity of this study-results needs further confirmation through a randomized large scale, multi-center prospective cohort study.

*Funding: No funding sources*
*Conflict of interest: None declared*
*Ethical approval: Not required*

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**Cite this article as:** Khan MH, Islam MMN, Islam MS, Khan KN, Chowdhury S, Rahman R. Correlation of troponin-I level with left ventricular systolic dysfunction after first attack of non-ST segment elevation myocardial infarction. Int J Res Med Sci 2019;7:1392-8.