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Original Article

Association of Troponin-I Level with Left Ventricular Systolic Dysfunction after First Episode of Non- ST Elevation Myocardial Infarction

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Key Words : *Troponin-I, Left Ventricular Systolic Dysfunction, Non- ST Elevation Myocardial Infarction, Coronary artery disease.*

Abstract:

Background: *Serum troponin is an exquisitely sensitive marker of myocardial injury and is necessary for establishing the diagnosis of Myocardial Infarction particularly non-ST elevation myocardial infarction (NSTEMI). However, the relation of troponin-I level with left ventricular systolic dysfunction following first episode of NSTEMI are still not examined in our population. Therefore, to determine the relationship of serum troponin I with left ventricular ejection fraction after first episode of NSTEMI was set as an objective of the study.*

Methods: *This cross sectional analytical study was conducted on 160 patients. They were divided into the two groups (80 patients in each group): Group-A (Troponin I level e" 0.4 ng/ml) high risk group and Group-B (Troponin I level < 0.4 ng/ml) low risk group. Left ventricular systolic dysfunction was defined as ejection fraction (EF%) < 50% calculated by modified Simpson's method on transthoracic echocardiography. Association of troponin-I level with left ventricular systolic function was determined.*

Results: *Among a total 160 patients, no significant variation across the group A and B in terms of age, gender and occupation (p>0.05 in all cases). Median serum troponin I of group A was 7.24 (range: 0.41-58.25) and group B was 0.21(range: 0.12-0.39). Left ventricular EF was significantly lower in higher troponin I group (Group A-45.95±10.28 vs. Group B-56.30±7.78; p<0.05) and it was associated with higher proportion of left ventricular dysfunction (p<0.05).*

Conclusion: *Serum troponin-I can be considered as a predictor of the left ventricular systolic dysfunction in first episode of Non-ST elevation myocardial infarction patients.*

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Introduction:

Epidemiological transition which is taking place in every part of the world, among all races, ethnic groups and cultures has resulted in the global rise in cardiovascular disease (CVD). Cardiovascular disease is the leading cause of morbidity and mortality throughout the world.[1,2] Important modifiable risk factors of CVDs are unhealthy diet, physical inactivity, tobacco use and the effects insinuate abnormal blood lipid profile and obesity.[3]

Risk assessment scores and clinical prediction algorithms using clinical history, physical examination, ECG, and cardiac troponins have been developed to help identify patients with ACS at increased risk of adverse outcomes. Common risk assessment tools include the TIMI (Thrombolysis in Myocardial Infarction) risk score, the GRACE (Global Registry of Acute Coronary Events) risk score. Assessment of prognosis guides initial clinical evaluation and treatment and is useful for selecting the site of care (coronary care unit, monitored step-down unit, or outpatient monitored unit), antithrombotic therapies and invasive management. There is a strong

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relationship between indicators of ischemia due to coronary artery disease and prognosis. Patients with a high likelihood of ischemia due to coronary artery disease are at greater risk of a major adverse cardiac event than patients with a lower likelihood of ischemia.[4]

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. Study reported that serum troponin I concentration has a strong negative correlation with left ventricular ejection fraction of acute myocardial infarction, and hence can be used to predict the LVEF in patients with first episode of myocardial infarction.[5] The aim of this study was to determine the Troponin-I level with left ventricular ejection fraction in patients after First Episode of non-ST elevation myocardial infarction.

Methods:

This cross-sectional analytical study was conducted in Department of Cardiology, Sir Salimullah Medical College & Mitford Hospital. Study group consisted of 160 patients hospitalized for first acute non-ST-elevation myocardial Infarction. Consent was taken from all the patients prior to inclusion in the study. Inclusion criteria were, all ages, both male and female, case of acute onset myocardial infarction as diagnosed by clinical presentation, symptom of ischaemia lasting >30 minute. ECG characteristics were - ST segment depression and/or inverted T-wave and/ or flat T-wave or normal. Exclusion criteria were a known case of old myocardial infarction or a patient having pre- existing ECG changes of old MI, ECG showing ST-elevation myocardial infarction, echocardiographic finding of old scar/ previous wall motion abnormalities / structural heart disease/ congenital heart disease. All admitted patients of acute non-ST-elevation MI included in this study. Complete history and clinical assessment with baseline and special investigations including serum troponin-I and echocardiography were done. Data were collected by direct interview from patient or attendant and by collecting blood sample from patients for laboratory investigations or collecting investigation results.

Left ventricular systolic function assessment: Echocardiographic ejection fraction was calculated by bi plane modified Simpson's rule. 16 segment regional wall motion abnormalities were determined in Parasternal long axis, Parasternal short axis, apical four chambers and apical two chamber views by 2D and M-Mode echo-cardiography. The assessment of cardiac LVIDd (Left ventricular internal dimension in diastole, LVIDs (Left ventricular internal dimension in systole), Fractional Shortening (FS) were calculated by Teichholz method. LVEF: <50% was considered LV systolic dysfunction. Echo-cardiography was done by GE Vivid E95 machine within 24 – 72 hours of onset of chest pain of index hospitalization. All patients were conservatively treated. Troponin-I assay method: Serum Troponin-I concentration was determined by immunometric assay (Vitros ECi Cube System-Troponin-I; Johnson & Johnson; USA). Serum Troponin-I value was measured within 12 to 24 hours of onset of chest pain. The Troponin kit reagent used in this study has a cut-off value of 0.12 ng/ml for diagnosis of acute myocardial infarction.

Then patients were allocated into two groups-Group-A (Troponin I level e"0.4 ng/ml) high risk group and Group-B (Troponin I level <0.4 ng/ml) low risk group.[6] In each group total 80 patients were included. All data were presented in suitable table or graph according to their affinity. A description of each table and graph were given to understand them clearly. All statistical analysis was performed using the statistical package for social science (SPSS) program, version 25 for Windows 10. Continuous parameters were expressed as Mean±SD and categorical parameters as frequency and percentage. Comparisons between groups (continuous parameters) were done by student-t test. Categorical parameters were compared by Chi-Square test. Correlation analyses will be done by Pearson correlation coefficient. The significance of the results as determined in 95.0% confidence interval and value of $P<0.05$ was consider to be statistically significant.

Results:

Total 160 study population of non-ST elevated MI was included in the study. Among them 80 patients

had serum troponin I level ≥0.4 ng/ml (group A) and another 80 patients had serum troponin I <0.04 ng/ml (group B). Mean age of all patients was 58.86±13.44 years, of group A patients was 59.24±12.92 years and of group B patients was 58.48±14.02 years. The difference between groups was not statistically significant (p>0.05). Majority patients belonged to age group 51 – 60 years (29.4%).

Total 65.6% patients were male and 34.4% patients were female. There was no statistically significant difference between groups in relation to sex (p>0.05).

Table-I

Age distribution of study population (N160).

Age (in years)	Group A (n=80) No. (%)	Group B (n=80) No. (%)	Total (n=160) No. (%)	p-value
31 – 40	9 (11.2)	17 (21.2)	26 (16.2)	0.135*NS
41 – 50	14 (17.5)	8 (10.0)	22 (13.8)	
51 – 60	24 (30.0)	23 (28.8)	47 (29.4)	
61 – 70	18 (22.5)	18 (22.5)	36 (22.5)	
71 – 80	9 (11.2)	13 (16.2)	22 (13.8)	
81 – 90	6 (7.5)	1 (1.2)	7 (4.4)	
Mean±SD	59.24± 12.92	58.48± 14.02	58.86± 13.44	0.721NS

Chi-squared Test and Unpaired Student's t-test was performed to compare between two groups. Group A: Patients who had serum troponin I ≥0.4 ng/ml. Group B: Patients who had serum troponin I <0.4 ng/ml. NS= Not significant (p > 0.05).

Table-II

Sex distribution of study population (N=160).

Sex	Group I (n=80) No. (%)	Group II (n=80) No. (%)	Total (n=160) No. (%)	p-value
Male	56 (70.0)	49 (61.2)	105 (65.6)	0.244NS
Female	24 (30.0)	31 (38.8)	55 (34.4)	

Chi-squared Test was performed to compare between two groups. Group A: Patients who had serum troponin I ≥0.4 ng/ml. Group B: Patients who had serum troponin I <0.4 ng/ml. NS= Not significant (p > 0.05)

Among all 51.9% had hypertension, 32.5% had DM, 36.2% had dyslipidemia, 63.1% had smoking habit, 14.4% led sedentary life and 42.5% had family history of CAD. There was no statistically significant difference between groups in relation to different risk factors of ACS (p>0.05).

Table-III

Distribution of risk factors among study population (N=160).

Risk factors	Group I (n=80) No. (%)	Group Ii (n=80) No. (%)	Total (n=160) No. (%)	p-value
Hypertension	41 (51.2)	42 (52.5)	83 (51.9)	0.874NS
Diabetes Mellitus	23 (28.8)	29 (36.2)	52 (32.5)	0.311NS
Dyslipidemia	26 (32.5)	32 (40.0)	58 (36.2)	0.324NS
Smoking habit	56 (70.0)	45 (56.2)	101 (63.1)	0.071NS
Sedentary lifestyle	13 (16.2)	10 (12.5)	23 (14.4)	0.499NS
Family history of CAD	32 (40.0)	36 (45.0)	68 (42.5)	0.422NS

Chi-squared Test was performed to compare between two groups. Group A: Patients who had serum troponin I ≥0.4 ng/ml. Group B: Patients who had serum troponin I <0.4 ng/ml. NS= Not significant (p > 0.05)

Among all patients respectively 95.6, 71.2, 68.8, 56.2 and 5.6% had central chest pain, nausea and/or vomiting, palpitation, breathlessness and loss of consciousness. Nausea and/or vomiting, palpitation and breathlessness were significantly more common among group A patients than group B patients (p<0.05). Increased respiratory rate and basal crepitation were significantly more common findings among group A patients than group B patients (p<0.05). Rests of the signs were similarly distributed among groups.

Table-IV

Distribution of study population according to their symptoms (N=160).

Symptoms	Group A (n=80) No. (%)	Group B (n=80) No. (%)	Total (n=160) No. (%)	p-value
Central chest pain	75 (93.8)	78 (97.5)	153 (95.6)	0.246NS
Nausea and/or vomiting	69 (86.2)	45 (56.2)	114 (71.2)	<0.001S
Palpitation	63 (78.8)	47 (58.8)	110 (68.8)	0.006S
Breathlessness	52 (65.0)	38 (47.5)	90 (56.2)	0.026S
Loss of consciousness	4 (5.0)	5 (6.2)	9 (5.6)	0.732NS

Chi-squared Test was performed to compare between two groups. Group A: Patients who had serum troponin I ≥0.4 ng/ml. Group B: Patients who had serum troponin I <0.4 ng/ml.
S= Significant (p < 0.05). NS= Not significant (p > 0.05)

Serum troponin I was measured within 12 to 24 hours after onset of chest pain. Median serum troponin I of group A patients was 7.24 ranging from 0.41 to 58.25 and of group B patients was 0.21 ranging from 0.12 to 0.39.

Mean LVEF and FS was significantly lower in group A patients then group B patients (p<0.001). LVIDd and LVIDs was significantly higher in group A patients in comparison to group B patients (p<0.05).

Table-V

Distribution of study population according to their signs (N=160).

Signs	Group A (n=80) No. (%)	Group B (n=80) No. (%)	Total (n=160) No. (%)	p-value
Tachycardia	19 (23.8)	10 (12.5)	29 (18.1)	0.065^{NS}
High Blood Pressure	26 (32.5)	23 (28.8)	49 (30.6)	0.607^{NS}
Increased Respiratory rate	17 (21.2)	5 (6.2)	22 (13.8)	0.006^{S}
Raised JVP	3 (3.8)	4 (5.0)	7 (4.4)	0.699^{NS}
Diastolic murmur	1 (1.2)	1 (1.2)	2 (1.2)	1.000^{NS}
Basal Crepitation	24 (30.0)	4 (5.0)	28 (17.5)	<0.001^{S}
Rhonchi	6 (7.5)	4 (5.0)	10 (6.2)	0.514^{NS}

Chi-squared Test was performed to compare between two groups. Group A: Patients who had serum troponin I ³0.4 ng/ml. Group B: Patients who had serum troponin I <0.4 ng/ml

S= Significant (p < 0.05). NS= Not significant (p > 0.05)

Among all patients 34.4% had mild left ventricular systolic dysfunction, 13.1% had moderate dysfunction and 2.5% had severe systolic dysfunction. Group A patients had significantly higher proportion of severe, moderate and mild left ventricular dysfunction as measure by EF than group B patients (p<0.001).

Table-VI

Serum troponin I level of both groups of study population (N=160).

Troponin I level (ng/ml)	Group A (n=80)	Group B (n=80)	Total (n=160)	p-value
Median (min-max)	7.24 (0.41-58.25)	0.21 0.40 (0.12-0.39)	<0.001^{S} (0.12-58.25)	

Mann-Whitney U test was performed to compare between two groups. Group A: Patients who had serum troponin I ≥0.4 ng/ml. Group B: Patients who had serum troponin I <0.4 ng/ml

S= Significant (p < 0.05). NS= Not significant (p > 0.05)

Table-VII

Echocardiographic findings in both group of study population (N=160).

Echocardio-graphic findings	Group A (n=80) Mean±SD	Group B (n=80) Mean±SD	Total (n=160) Mean±SD	p-value
LVEF (%)	45.36±10.91	55.98±8.27	50.67±11.02	<0.001^{S}
LVIDd (mm)	49.99±6.78	45.85±7.95	47.92±7.65	0.001^{S}
LVIDs (mm)	37.84±7.42	32.24±7.02	35.04±7.73	<0.001^{S}
FS (%)	23.92±6.51	28.69±5.54	26.15±6.51	<0.001^{S}

LVEF: Left ventricular ejection fraction; LVIDd: Left ventricular internal diameter during diastole: LVIDs: Left ventricular internal diameter during systole; FS: Fractional Shortening; Unpaired Student's t test was performed to compare between two groups. Group A: Patients who had serum troponin I ≥0.4 ng/ml. Group B: Patients who had serum troponin I <0.4 ng/ml

S= Significant (p < 0.05). NS= Not significant (p > 0.05)

Table-VIII

Severity of left ventricular dysfunction in study population (N=160).

Severity of Left Ventricular Dysfunction	Group A (n=80) No. (%)	Group B (n=80) No. (%)	Total (n=160) No. (%)	p-value
Normal (EF 50-70%)	25 (31.2)	55 (68.8)	80 (50)	<0.001^{S}
Mild (EF 40-49%)	31 (38.8)	24 (30)	55 (34.4)	
Moderate (EF 30-31%)	20 (25.0)	1 (1.2)	25 (13.1)	
Severe (EF <30%)	4 (5.0)	0	4 (2.5)	

Chi-squared Test (χ^2) was performed to compare between two groups. Group A: Patients who had serum troponin I ≥0.4 ng/ml. Group B: Patients who had serum troponin I <0.4 ng/ml

S= Significant (p < 0.05). NS= Not significant (p > 0.05)

A significant negative linear correlation was found between serum troponin I and LVEF (Pearson's r= -0.411, p<0.001). This indicates that LVEF decreased significantly with increasing troponin I level of patients.

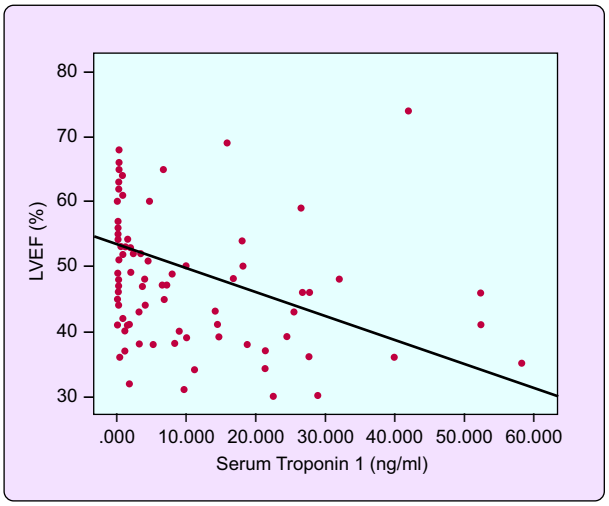

Fig.-1: *Correlation of serum troponin I with left ventricular ejection fraction.*

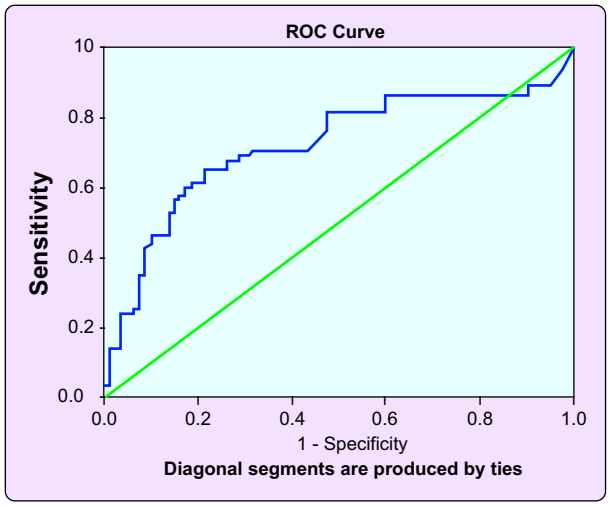

Fig.-2: *Receiver-Operator Characteristics curve analysis of troponin I in the prediction of LV dysfunction.*

Table-IX

Complication of NSTEMI in both group of study population (N=160).

Complications	Group A (n=80) No. (%)	Group B (n=80) No. (%)	Total (n=160) No. (%)	p-value
Left ventricular failure	31 (38.8)	8 (10.0)	39 (24.4)	<0.001[S]
Arrhythmia	9 (11.2)	4 (5.0)	13 (8.1)	0.148[NS]

Chi-squared Test was performed to compare between two groups. Group A: Patients who had serum troponin I ≥0.4 ng/ml. Group B: Patients who had serum troponin I <0.4 ng/ml

S= Significant ($p < 0.05$). NS= Not significant ($p > 0.05$)

Group A patients had significantly more cases of left ventricular failure than group B patients ($p<0.001$). Frequency of arrhythmia was statistically similar across groups ($p>0.05$).

ROC curve analysis of troponin I detected that a cut-off value of ≥0.40 ng/ml in the prediction of LV dysfunction among patients gives a sensitivity and specificity of 68.7% each. While a more accurate cut-of value of ≥0.37 gives a sensitivity and specificity of 70 and 68.7% respectively.

Area Under the Curve			
Area	P value	Asymptotic 95% Confidence Interval	
		Lower Bound	Upper Bound
.717	.000	.634	.799

Discussion:

Acute coronary syndrome (ACS) remains the leading cause of death in the developed world and second leading cause of death in developing countries.[7] One study found that NSTEMI accounts for approximately 70% cases and the proportion is rising with availability of advanced testing instruments.[8] NSTEMI is usually caused by unstable atherosclerotic plaque, resulting in the formation of either a non-occlusive thrombus or complete thrombosis of a vessel supplying a well collateralized area.[9] This may lead to myocardial damage and heart failure. Troponin I is a good marker for minor cardiac damage and can predict the development of heart failure in patients with myocardial infarction.[10,11] Therefore, this study was carried out with an aim to determine the association of troponin-I level with left ventricular systolic dysfunction after first episode of NSTEMI.

Total 160 patients of NSTEMI were enrolled. Group A and B consisted of 80 patients each with serum troponin I level of ≥0.4 ng/ml and <0.4 ng/ml respectively. Mean age of all patients was 58.86±13.44 years which was similar studies conducted in Bangladeshi population.[12,13]

Most of the patients in this study were male. Nearly similar finding was reported previously by other studies.[12,13] A meta-analysis reported 67% men among total 5360 NSTEMI patients.[14]

Although risk factors for myocardial infarction are common among both genders the higher prevalence of men among NSTEMI cases could be explained by higher risk among younger men than women.[15]

Among all of patients, 63.1% had smoking habit, 51.9% had hypertension, 46.7% were overweight/ obese, 42.5% had family history of CAD, 36.2% had dyslipidemia, 32.5% had DM and 14.4% led sedentary lifestyle. This is concordant with the findings of other studies conducted in Bangladesh and Indian subcontinent. The largest study to date to determine the risk factors of MI in the world, the INTERHEART study, has found that smoking, hypertension, diabetes, abdominal obesity, psychosocial factors, and irregular physical activity are important determinants of MI alongside alcohol intake, dietary habit and psychosocial factors.[16]

Patients with acute myocardial infarction may present with a myriad of typical and atypical symptoms and signs. Central compressive chest pain is the most common and typical feature of ACS and which is associated with various other symptoms at different proportions[17]. In the present study central chest pain was the most common symptoms at presentation (95.6%). Group A patients had significantly more proportion of breathlessness, and basal crepitation than group B patients indicating a higher proportion of heart failure among the former which was indeed the case in this study.

In this study 34.4% had mild left ventricular systolic dysfunction and 13.1% had moderate dysfunction and 2.5% had severe dysfunction. In comparison a study conducted in NICVD by Rahman and his colleagues found that 24.6% had mild dysfunction, 14.4% had moderate dysfunction and 1.7% had severe dysfunction.[18]

Left ventricular ejection fraction was significantly lower in patients with troponin I level ≥0.4ng/ml than those with troponin level <0.4ng/ml. Also, the severity of LV dysfunction was worse in the former group of patients. In addition, proportion of clinical left ventricular failure was significantly high among group A patients than group B patients indicating that patients with serum troponin I level ≥0.4ng/ml at presentation developed heart failure significantly more than those with troponin I level <0.4ng/ml. Univariate analysis followed by multivariate analysis also showed that serum troponin I is an independent predictor of left ventricular dysfunction among NSTEMI patients. Also, a significant inverse linear correlation was found between serum troponin I and LVEF indicating that LVEF decreased significantly with increasing troponin I level of patients. All these findings replicated the findings of Khan and his colleagues who did a same study on patients of NSTEMI.[13] Like some other studies, there was a significant negative correlation between LVEF and troponin I.[19]

Limitations of the study:

Sampling was purposive. Long terms follow up and repeated Echo could not be evaluated. Single sample of troponin-I was done.

Conclusion:

This study demonstrated that in non-ST segment elevated myocardial infraction patients, almost half of the patients develop either mild or moderate degree of ventricular dysfunction assessed by echocardiography. And this left ventricular dysfunction was associated with higher troponin value at admission. Moreover, there complications were also more. So, on admission troponin I level can help us to predict the prognosis of NSTEMI.

Conflict of Interest - None.

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