

## Demographic Profile of NSTEMI (Non ST Elevation Myocardial Infarction) Patients & Association of ST-Segment Depression and Level of Troponin I with NSTEMI Patient's In-Hospital Outcome

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### Abstract

Acute coronary syndrome (ACS) remains the leading cause of death in the developed world and second

leading cause of death in developing countries. Elevated troponin levels and extent of ST-segment depressions are clinically important because they may act as an effective prognostic marker. This cross-sectional study has been designed to see the correlation of ST-segment depression and level of troponin I with in-hospital outcome of NSTEMI patients. The study was conducted in the Department of Cardiology, Dhaka Medical College Hospital, Dhaka during the period of April, 2011-March, 2012. A total of 90 patients were selected by purposive sampling. In this study, the mean  $\pm$ SD age of the patients was  $55.9 \pm 9.1$  years with a range of 36-80 years with a male-female ratio of 2:1. Over all dyslipidaemia was the most common risk factor present in 55(61.10%) patients followed by smoking in 48(53.3%) patients then obesity in 32(35.60%) then hypertension in 31(34.4%) patients. Troponin I level was significantly high in patients who developed acute LVF ( $10.36 \pm 7.4$  vs  $7.0124 \pm 6.8$ ,  $p = .027$ ), and cardiogenic shock ( $13.72 \pm 11.37$  vs  $8.64 \pm 7.35$ ,  $p = .033$ ). Troponin I was significantly high in patients who developed complication ( $10.72 \pm 8.84$  vs  $6.24 \pm 5.41$ ,  $p = .005$ ) than the patients who were discharged without complication. ST segment depression was significantly more in patients who developed acute LVF ( $1.07 \pm 1.63$  vs  $.55 \pm .74$ ,  $p = .048$ ). Logistic regression analysis of acute LVF with Troponin I and ST-segment depression showed that ST-segment depression and level of Troponin I were important correlates of acute LVF.

**Keywords:** NSTEMI, Troponin I, ST-segment depression, In-hospital outcome

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### Introduction

Acute coronary syndrome (ACS) remains the leading cause of death in the developed world and second leading cause of death in developing countries<sup>1,2</sup>. Various studies have pointed out that South Asians have a higher prevalence of ACS as compared with other ethnicities, with a higher rate at younger ages<sup>3</sup>. Being a south Asian country Bangladesh is not immune to this higher prevalence of ACS. Non ST elevation myocardial infarction (NSTEMI) is an important part of ACS.

Elevated troponin levels are clinically important because they may act as an effective prognostic marker<sup>4</sup>. In the consensus document of the Joint ESC/ACC Committee, myocardial infarction is defined on the basis of pathological findings or on the basis of a typical rise and fall in biochemical markers of myocardial necrosis and the presence of at new assay least one of the following: ischemic signs and symptoms, electrocardiographic (ECG) signs of ischemia or necrosis, or a coronary artery intervention<sup>5</sup>. Diagnostic level for increased cardiac risk is troponin I > 0.25 ng/ml. A level of 0.1-0.25 ng/ml is considered intermediate. A level of < 0.1 ng/ml is considered negative. An elevated troponin indicates myocardial necrosis. It can occur in acute myocardial infarction and in other clinical settings where myonecrosis has occurred. The assay identifies patients who are at higher risk for cardiac events and mortality<sup>6,7</sup>. Each increase of 1.0 ng/ml in the cardiac troponin I level is associated with an increase in the relative risk of mortality. Lower limit of detection is 0.04 ng/ml. The 97.5 percentile for apparently healthy adults troponin I level is 0.06 ng/ml. A minimum of 0.3 ml of blood is required. Moderately hemolyzed specimens are acceptable. The test is available only on a STAT basis with a maximum 60 minute turn-around time. A troponin I level >2.0 ng/ml will be treated as a panic value<sup>6,7</sup>. In patients of ACS with ST depression  $\geq 2$  mm in 2 or more adjacent leads the probability of dying was 6 times higher within 1 year than it was the case with patients without ST depression in ACS<sup>8</sup>. In the presence of normal ECG appearance, NSTEMI is not excluded. In patients with ischemic symptoms and normal ECG, there may be approximately 1 - 6% patients presenting with evidence of myocardial necrosis (i.e., NSTEMI)<sup>9</sup>. About 30% of patients with NSTEMI, have an MI within 3 months of onset; sudden death is less common. Five clinical characteristics predict 90% of the mortality in patient with NSTEMI, these are older age, low systolic BP, Killip class >1, fast heart rate, anterior location on ECG<sup>10</sup>. A substantial number of patients get admitted to the Cardiology Department of Dhaka Medical College Hospital and diagnosed as NSTEMI. The aim of the study is to correlate in-hospital outcome of these patient by the level of troponin I and extent of ST-segment depression on ECG and to determine, between the level of troponin I and extent of ST-segment depression, which one is the better predictor regarding in-hospital outcome of NSTEMI patients. The objectives of the study was to find out the association between ST-segment depression and level of troponin I with in-hospital outcome of NSTEMI patients.

#### Materials and Methods

This Cross sectional study was in the Department of Cardiology, Dhaka Medical College, Dhaka for 1 year during the period of April, 2011 to March, 2012. All the patients of acute coronary syndrome admitted in the Coronary care unit, Dhaka Medical College Hospital within the study period were included in this study.

Newly diagnosed NSTEMI Patients who have not any valvular or congenital heart diseases and not suffering from any severe co morbid condition and willing to participate in providing data and sample of blood, were selected. Patients having previous history of PCI, CABG, or old MI were excluded from the study. Sample size was 90. Data was collected by using a structured data sheet. After recording patients profile, risk factors of IHD like hypertension, smoking, dyslipidaemia, diabetes mellitus, family history of premature CAD and obesity was noted. Then the patients ECG were done and troponin I was tested within 6 to 24 hours of symptoms. Baseline laboratory investigation e.g., serum creatinine, blood sugar, lipid profile, Echocardiography was done for each patients. Then all the patients were grouped according to serum Troponin I and ST-segment depression status. Then in-hospital outcome of all patients like acute LVF, Cardiogenic were noted and compared by the level of Troponin I and ST-segment depression. Data was analyzed by using SPSS (statistical package for the social science) version 16. Continuous data were expressed as mean  $\pm$  standard deviation of mean and categorical data as percentages. Categorical variables were analyzed by chi-square test. Quantitative variables were analyzed t test or ANOVA. Correlation between magnitude of ST-segment depression and level of Troponin I with in-hospital outcome was measured by regression analysis. P value  $p < 0.05$  was considered as significant.

#### Results

This study was conducted in the Department of Cardiology, DMCH, Dhaka for 1 year starting from April 2011 to March 2012. A total 90 patients with NSTEMI were followed up during hospital period. Patients were categorized into 4 groups on the basis of ST-segment depression and another 4 groups by troponin I. In-hospital outcome was taken. The descriptive and inferential statistics are described below by tables and graphs. Table-1 shows, the mean  $\pm$ SD age of the patients was 55.9 $\pm$ 9.1 years with a range of 36-80 years. The highest number of patients are within the group '51-60' years (33.3%) followed by 41-50 years group (31.1%) and >60 years group (30.0%)

Table -I. Distribution of the patients by age (n=90)

Age (years)	n(%)
31-40	5 (5.6)
41-50	28(31.1)
51-60	30(33.3)
>60	27(30.0)
Total	90(100)
Mean $\pm$ SD	55.9 $\pm$ 9.1
Range	36-80

Among the patients, 60(66.7%) were male and 30 (33.3%) were female. Male female ratio was 2:1 (Fig.1)

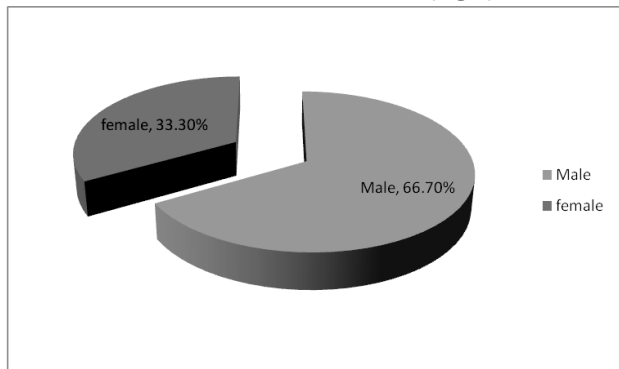


Figure 1: Distribution of patients by sex (n=90)

Figure-2 shows among the traditional risk factors for the cardiovascular diseases, smoking or chewing tobacco was present in 48(53.3%) patients, obesity in 32(35.6%), hypertension in 31 (34.4%), diabetes mellitus in 18(20.0%), dyslipidaemia in 55(61.1%), family history of premature CAD in 11(12.2%) and sedentary life style in 8(8.9%) patients

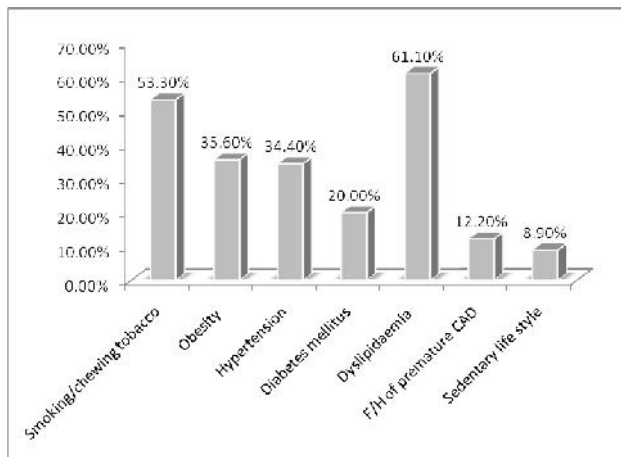


Figure- 2: Distribution of patients by traditional risk factors (n=90)

Figure 3 shows Troponin I was catagorised 2(2.2%) patients in group 1, 3(3.3%) patients in group II, 35(38.9%) patients in group III and in group IV 50(55.6%) patients.

**Distribution of patients by troponin I level (n=90)**

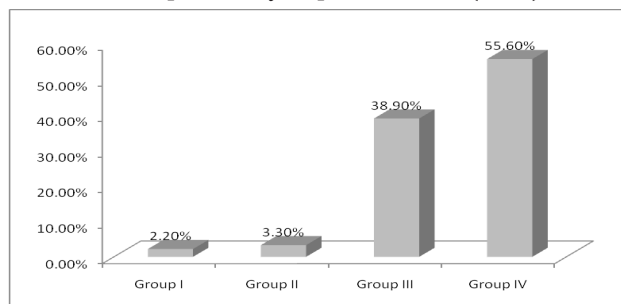


Figure - 3: Group I = troponin I level up to 0.5 ng/ml, Group II = troponin I level >0.5 to 1.0 ng/ml , Group III = troponin I level >1.0 to 5.0 ng/ml, Group IV = troponin I level >5.0 ng/ml

Figure - 4 shows distribution of patients by measurement of ST segment depression. In group I 51(56.7%) patients, group II 34(37.8%) patients, in group III 5(5.6%) and in group IV no patients .

**Distribution of patients by ST segment depression (n=90)**

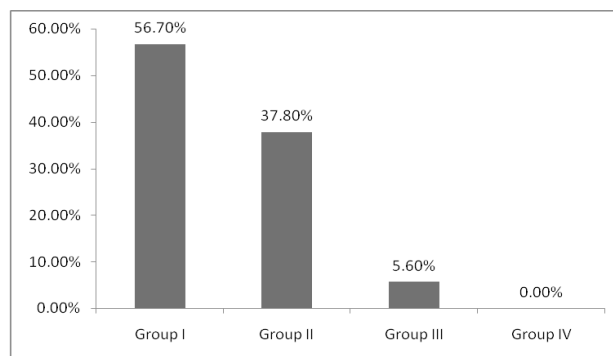


Figure-4: Group I = ST-segment depression 0 mm, Group II = ST-segment depression 1-2 mm, Group III = ST-segment depression >2 to 3mm, Group IV = ST-segment depression >3 mm.

Table 2 shows troponin I level was significantly high in patients who developed acute LVF (10.36±7.4 vs 7.012±6.8, p=.027), and cardiogenic shock (13.72±11.37 vs 8.64±7.35, p=.033). Troponin I was significantly high in patients who developed complication (10.72±8.84 vs 6.24±5.41, p=.005) than the patients who were discharged without complication. No significant difference was observed in Troponin I level between patients with or without development of arrhythmia, conduction defect and death (p>0.05).

Table-2: Comparison of troponin I level between patients with or without development of in- hospital outcome :

In hospital outcome	Troponin I (ng/ml) (Mean ± SD)	P value
Acute LVF		
Yes (n=41)	10.36 ± 7.4	0.027*
No(n=19)	7.012 ± 6.8	
Cardiogenic Shock		
Yes (n=14)	13.72 ± 11.37	0.033*
No (n=76)	8.64 ± 7.35	
Arrhythmia		
Yes (n=07)	9.58 ± 11.08	0.958
No (n=85)	9.41 ± 8.03	
Conduction defect		
Yes (n=01)	1.22 ± 1.05	0.319
No (n=89)	9.52 ± 8.23	
Discharge with complication		
Yes (n=64)	10.72 ± 8.84	0.005*
No (n=26)	6.24 ± 5.41	
Death		
Yes (n=1)	10.00 ± .01	0.945
No (n=89)	9.42 ± 8.27	

P value was derived from t test, \* = Statistically significant

Table 3 shows ST segment depression was significantly more in patients who developed acute LVF ( $1.07 \pm 1.63$  vs  $.55 \pm .74$ ,  $p=.048$ ). ST segment depression was not statistically different in patients with or without development of Cardiogenic shock ( $.57 \pm 1.38$  vs  $.801 \pm 1.23$ ,  $p=.810$ ), arrhythmia ( $.57 \pm .98$  vs  $.69 \pm .91$ ,  $p=.749$ ), conduction defect ( $0.00$  vs  $.69 \pm .91$ ,  $p=.457$ ), discharge without complication ( $.57 \pm .70$  vs  $.71 \pm .98$ ,  $p=.308$ ) and death ( $0.00$  vs  $.69 \pm .91$ ,  $p=.457$ ).

Table-3: Comparison of ST-segment depression between patients

with or without development of in- hospital outcome :

In hospital outcome	ST-segment depression (Mean $\pm$ SD)	P value
<b>Acute LVF</b>		
Yes (n=41)	$1.07 \pm 1.63$	<b>.048</b>
No (n=49)	$.55 \pm .74$	
<b>Cardiogenic Shock</b>		
Yes (n=14)	$.71 \pm 1.38$	
No (n=76)	$.801 \pm 1.23$	<b>.810</b>
<b>Arrhythmia</b>		
Yes (n=07)	$.57 \pm .98$	<b>.749</b>
No (n=83)	$.69 \pm .91$	
<b>Conduction defect</b>		
Yes (n=01)	$0.00$	<b>.457</b>
No (n=89)	$.69 \pm .91$	
<b>Discharge with complication</b>		
Yes (n=64)	$.71 \pm 1.40$	<b>.308</b>
No (n=26)	$.57 \pm .70$	
<b>Death</b>		
Yes (n=1)	$0.00$	<b>.457</b>
No (n=89)	$.69 \pm .91$	

P value was derived from t test

## Discussion

The mean  $\pm$ SD age of our patients was  $55.9 \pm 9.1$  years (table-I) and male participants were 66.7% and female 33.3%. The mean age of the patients of a meta-analysis by Heidenreich *et al* (2001) was 63 years and male was 67% in their study. The findings of the study by Heidenreich *et al* (2001)<sup>4</sup> are similar to this study findings. In the present study smoking or chewing tobacco was present in 53.3% patients, obesity in 35.6%, hypertension in 34.4%, diabetes mellitus in 20.0%, dyslipidaemia in 61.10%, family history of premature CAD in 12.2% and sedentary life style in 8.9% patients (figure-2). The participant of Heidenreich *et al* (2001) meta-analysis had a history of hypertension in 42%, diabetes in 17% and smoking in 41%<sup>4</sup>. All these findings are similar to this study findings. Among all patients 6(6.6%) patients' pulse was feeble. The remaining 84(93.4%) patients had mean $\pm$ SD pulse  $86.75 \pm 14.27$  beats/min with a range of 50-120 beats/min indicating the presence of arrhythmia including bradycardia and tachycardia. Among all patients 4(4.4%)

patients' systolic and diastolic blood pressure was not recordable. The remaining 86(95.6%) patients had mean $\pm$ SD systolic blood pressure  $118.12 \pm 19.42$  mm of Hg with a range of 60-200 mm of Hg and mean $\pm$ SD diastolic blood pressure  $75.82 \pm 11.14$  mm of Hg with a range of 40-100 mmHg indicating the presence of hypertension and cardiogenic shock. ST segment depression was significantly more in patients who developed acute LVF ( $1.07 \pm 1.63$  vs  $.55 \pm .74$ ,  $p=.048$ ). But ST segment depression was not statistically different in patients with or without development of cardiogenic shock ( $.57 \pm 1.38$  vs  $.801 \pm 1.23$ ,  $p=.810$ ), arrhythmia ( $.57 \pm .98$  vs  $.69 \pm .91$ ,  $p=.749$ ), conduction defect ( $0.00$  vs  $.69 \pm .91$ ,  $p=.457$ ), discharge without complication ( $.57 \pm .70$  vs  $.71 \pm .98$ ,  $p=.308$ ) and death ( $0.00$  vs  $.69 \pm .91$ ,  $p=.457$ ). One (2.0%) patient of ST segment depression group' died during hospital stay. However there was no significant difference in mortality among the groups ( $p=0.457$ ) The findings of the study was supported by Birnbaum *et al* (1996), Atar *et al* (2007), Kaul *et al* (2001), Peterson *et al* (1996)<sup>11-14</sup>. Atar *et al* (2007) collected electrocardiographic (ECG) and clinical data from 6,770 patients with NSTEMI. The authors found that ST-segment depression in any of the ECG locations was associated with higher mortality compared with patients without ST-segment depression<sup>12</sup>. Kaul *et al* (2001) described that ST segment depression was the strongest predictor of one-year mortality. Patients with ST segment depression  $\geq 2$  mm were almost 6 times more likely to die within one year than patients with no ST segment depression<sup>13</sup>. Troponin I level was significantly high in patients who developed acute LVF ( $10.36 \pm 7.4$  vs  $7.0124 \pm 6.8$ ,  $p=.027$ ), and cardiogenic shock ( $13.72 \pm 11.37$  vs  $8.64 \pm 7.35$ ,  $p=.033$ ). Troponin I was significantly high in patients who developed complication ( $10.72 \pm 8.84$  vs  $6.24 \pm 5.41$ ,  $p=.005$ ) than the patients who were discharged without complication. No significant difference was observed in Troponin I level between patients with or without development of arrhythmia, conduction defect and death ( $p>0.05$ ). These observations are in consistent with the findings of the trial by Acharji *et al* (2012), Ben-Dor *et al* (2006), Diderholm *et al* (2002), Heidenreich *et al* (2001)<sup>15-17</sup>. Diderholm *et al* (2002) observed that troponin I level is an effective prognostic marker and its level predicts in-hospital outcome in NSTEMI. The in-hospital outcome they observed were heart failure, arrhythmia, cardiogenic shock, heart block, even death etc.<sup>17</sup>. In a meta-analysis of seven clinical trials and 19 cohort studies Heidenreich *et al* (2001) found that patients with positive troponin (I or T) had significantly higher mortality than those with a negative test. They also found that there was no significant difference for mortality between troponin T and troponin I level<sup>4</sup>. The study was not supported by Nikus *et al* (2004). They found that the troponin levels did not differ significantly according to ST level depressions<sup>18</sup>. In the present study cardiac troponin I level and ST

segment depression has significant correlation with some in-hospital outcome like acute LVF and also significant correlation with patients with complication of NSTEMI during hospital stay. This finding is in line with the findings of Ciric-Zdravkovic (2007). Ciric-Zdravkovic (2007) proved that serum cardiac troponin level is related to ST depression size changes. Troponin I levels are much more elevated with ST depression depth increase<sup>19</sup>. The study was supported by Mia M E H, In 2011 one cross sectional study was done in NICVD, Bangladesh, which found that magnitude of ST-segment depression positively correlate with the severity of coronary artery disease.<sup>20</sup>

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