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The Association of Cardiac Troponin I Level on Admission with the Angiographic Severity of Coronary Artery Disease in Acute STEMI Patients

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ABSTRACT

Objective: In this study our main goal is to find out the association of cardiac troponin I level on admission with the angiographic severity of coronary artery disease in acute STEMI patients.

Method: This cross-sectional observational study was done in the NICVD, Dhaka from October 2010 to September 2011. A total of 100 consecutive patients were included. Study populations were sub-divided into two groups on the basis of cTn I level. In group I cTn I level ≥20ng/ml and in group II cTn I level <20ng/ml. 50 patients were included in group I and 50 patients were included in group II.

Results: Most of the patients presented with more than 4 hours chest pain which was 68% vs. 60% patients in group I and group II respectively. Where mean heart rate was higher in group I than group II (83.0±8.4 vs. 78.2±10.6). It was statistically significant (p=0.01) in student-test. Vessel score 2 and 3 together formed the main bulk of the patients (68%) in group I, while vessel score 0 and 1 were frequently common in group II (80%). In group I there was no patient with vessel score 0, in group II 12(24%) patients had vessel score 0 (5 patients had normal coronaries and 7 patients had insignificant lesions, p-value from Chi square test).

Conclusion: From our study we can say that, estimation of serum cardiac troponin I might facilitate the triage of patients with acute myocardial infarction by clinicians and more aggressive approach to promote myocardial reperfusion might be warranted in the patients with high cardiac troponin I level.

Keywords: Coronary Heart Disease (CHD), Diabetes Mellitus, Dyslipidaemia, cTn I.

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INTRODUCTION

Coronary heart disease (CHD) is now the leading cause of death worldwide; it is on the rise and has become a true pandemic that respects no borders. This statement, made in 2009, can be found on the Web site of the World Health Organization (WHO, 2009), and is not that different from the warning issued in 1969 by the executive board of the World Health Organization: "Mankind's greatest epidemic: coronary heart disease has reached enormous proportions striking more and more at younger subjects. It will result in coming years in the greatest epidemic mankind has faced unless we are able to reverse the trend by concentrated research into its cause and prevention" (WHO, 1973).CHD has received great attention because its epidemic development after World War II initially struck the industrialized countries. Nowadays, however, the burden of CHD involves the whole world; the age-standardized death rates for CHD are declining in many developed countries but are increasing in developing and transitional countries—partly as a result of demographic changes, urbanization, and lifestyle changes. Today, approximately 3.8 million men and 3.4 million women worldwide die each year from CHD.[1-3]

In recent study it was found that Cardiac troponin I is a highly accurate, sensitive and specific marker for determination of myocardial injury in acute STEMI patients. It is a powerful predictor of outcome and benefit of invasive strategy. Higher cardiac troponin I levels are associated with worse outcomes and complex coronary lesions in STEMI patients. Cardiac troponin I level also showed a predictive capacity to assess angiographic severity of coronary artery disease that is required for subsequent decision making, appropriate referral and early intervention to

improve myocardial reperfusion.[4] Therefore, this simple, affordable and widely available noninvasive test may likely to increase clinicians` ability to rapidly and accurately assess risk and tailor effective management in acute STEMI patients.[5]

In this study our main goal is to find out the association of cardiac troponin I level on admission with the angiographic severity of coronary artery disease in acute STEMI patients.

OBJECTIVE
General Objective
- To find out the association of cardiac troponin I level on admission with the angiographic severity of coronary artery disease in acute STEMI patients.

Specific Objective
- To identify clinical presentations of patients.
- To detect coronary angiographic severity of patients.

METHODOLOGY
Study Type
It was a cross sectional study.
Place and Period of the Study
This study was carried out in NICVD, Dhaka from October 2010 to September 2011.
Study Population
All patients diagnosed as acute STEMI admitted in NICVD and undergoing coronary angiography during hospital admission were taken as study population. A total of 100 consecutive patients were included. Study populations were sub-divided into two groups on the basis of cTn I level. In group I cTn I level ≥20ng/ml and in group II cTn I level <20ng/ml. 50 patients were included in group I and 50 patients were included in group II.

Method
All patients admitted in Cardiology department of NICVD, Dhaka, fulfilling the inclusion criteria and exclusion criteria was considered for study. Informed written consent was taken from all patients or from legal guardian before enrollment. Acute STEMI was diagnosed by ESC/ACC guideline 2004.Initial evaluation of the patients by history and clinical examination was performed and recorded in patients' data collection sheet. Demographic profile, and pulse, blood pressure, body weight were recorded. Serum cTn I level was estimated and recorded by Immulite 1000 Troponin I. (SIEMENS Medical Solutions Diagnostic, Los Angeles, CS, USA). Echocardiographic ejection fraction was recorded on second or third day of hospitalization. Coronary angiogram was done during hospital admission. Angiographic severity of coronary artery disease was assessed by Vessel score and Stenosis score.

Statistical Analysis
Data were processed and analyzed using computer-based software SPSS (Statistical Package for Social Sciences) for windows version 22. Unpaired t-test was used to compare quantitative variables. Variables were expressed as range and mean $\pm$ SD. p value < 0.05 were taken significant. Students' t test, Pearson's correlation coefficient test, multivariate logistic regression analysis and Fisher's exact test as applicable.

Table 1: Age distributions of the patients

Age (in years)	Group -1 (n= 50)		Group -2 (n = 50)		P value
	Number	%	Number	%	
< 40	8	16.0	7	14.0	
40 – 50	21	42.0	20.0	40.0	
> 50	21	42.0	23	46.0	0.63^{ns}
Mean ±SD	49.8 ± 9.8		50.7 ± 10.4		

Table 2: Distribution of patients by clinical presentations (n=100)

Clinical presentations	Group-1 (n = 50)		Group -2 (n = 50)	
Duration of chest pain in hours	Number	%	Number	%
< 2	5	10.0	2	4.0
2 – 4	11	22.0	18	36.0
> 4	34	68.0	30	60.0

Table 3: Distribution of the patients by heart rate

Variable	Group -1 (n = 50)	Group -2 (n = 50)	P value
Heart rate	83.0±8.4	78.2±10.6	0.01^{s}

Table 4: Comparison of coronary angiographic severity between two groups

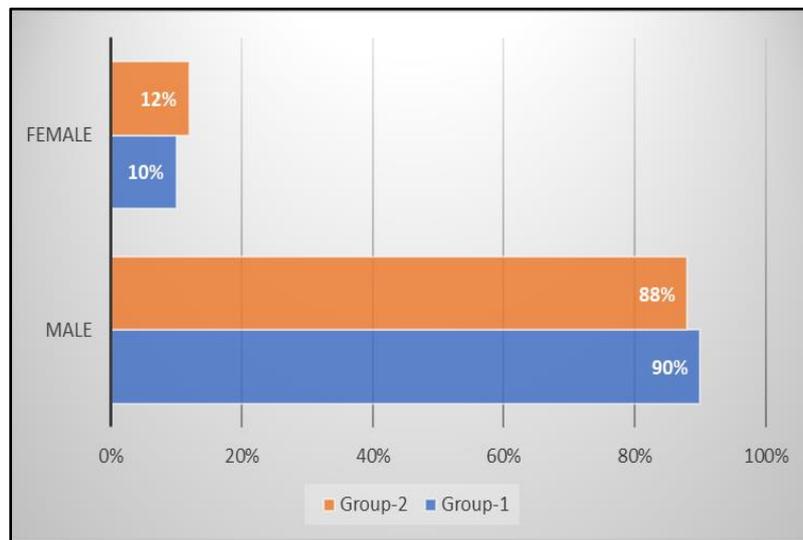| Coronary angiograms	Group I (n= 50)		Group II (n =50)		p-value
Vessel score	Number	%	Number	%	
Score – 0	0	0.0	12	24.0	
Score – 1	12	24.0	28	56.0	0.001^{s}
Score – 2	16	32.0	10	20.0	0.17^{ns}
Score – 3	18	36.0	4	8.0	0.001^{s}
Mean $\pm$ SD	2.24±0.85		1.16±0.44		0.001^{s}

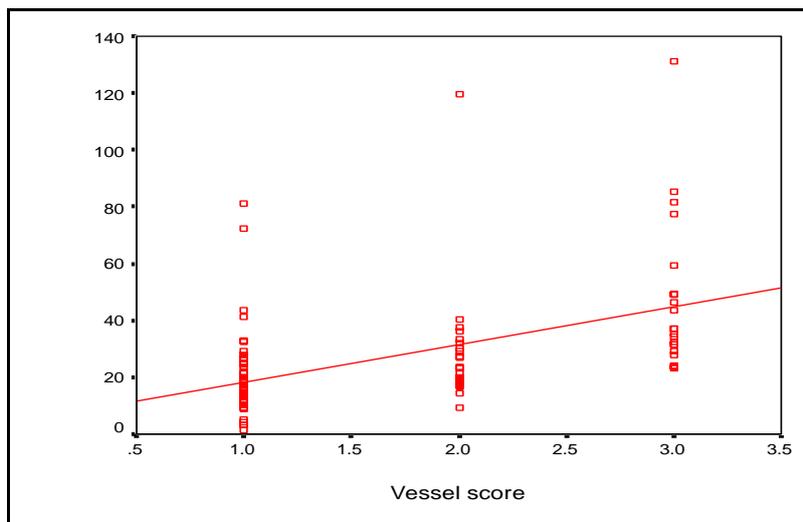**Figure 1: Gender distribution of the patients**

Fig 2: Correlation between serum cardiac troponin I level and vessel score

RESULTS

In table-1 shows age distributions of the patients where mean age of the patients of Group I was 49.8 ± 9.8 years and that of Group II was 50.7 ± 10.4 years. Though the mean age of group II was somewhat higher than group I the difference was not statistically significant (p=0.63) by Student's t-test.

In figure-1 shows gender distribution of the patients where among the study population the male and female patients were identical in both the groups which was statistically insignificant (p=0.74) by χ^2 (Chi square) test.

In table-2 shows distribution of patients by clinical presentations where most of the patients presented with more than 4 hours chest pain which was 68% vs. 60% patients in group I and group II respectively. The mean difference of chest pain was statistically significant (p=0.04) between two groups in student t-test.

In table-3 shows distribution of the patients by heart rate where mean heart rate was higher in group I than group II (83.0±8.4 vs. 78.2±10.6). It was statistically significant (p=0.01) in student-test.

In table-4 shows association between cardiac troponin I level and coronary angiographic findings where vessel score 2 and 3 together formed the main bulk of the patients (68%) in group-1, while vessel score 0 and 1 were frequently common in group -2 (80%). In group -1 there was no patient with vessel score 0, in group -2 12(24%) patients had vessel score 0 (5 patients had normal coronaries and 7 patients had insignificant lesions, p-value from Chi square test). The mean vessel score for group-1 patients were 2.24±0.85 and that of group-2 patients was 1.16±0.44 and the mean difference was statistically significant (p=0.001) in unpaired t-test.

In figure-2 shows Correlation between serum cardiac troponin I level and vessel score where the two variables exhibit significantly positive correlation (r=0.49, p=0.01).

DISCUSSION

Before introducing cTn I, the conventional markers in diagnosing acute myocardial infarction, creatine kinase (CK), lactate dehydrogenase (LDH) and their isoenzymes CKMB and LDH-1 are neither perfectly sensitive nor absolutely heart-specific. The development of a highly specific assay for cTn T was a diagnostic breakthrough for the laboratory diagnosis of myocardial damage in clinical practice. Although superior to CKMB and LDH-1, the specificity of cTn T for cardiac damage is not so far delineated.

cTn-T is expressed in fetal skeletal muscle and its re expression may occur in regenerating skeletal muscle. By contrast, cTn I is not expressed in skeletal muscle in any phase of ontogenesis. cTn I (molecular weight 22500 Da) is the only isotype present in human myocardium, and make this analyte as a cardiac-specific laboratory marker for myocardial damage.[6,7]

According to WHO's definition, a myocardial infarction occurs if at least two of the three criteria fulfilled: typical ischemic chest pain; raised concentrations of creatine kinase-MB in serum; and typical electrographic findings, including development of pathological Q-waves. Creatine kinase-MB, however, is not a sensitive marker of myocardial necrosis. Therefore, application of the WHO definition in clinical practice results in several patients erroneously diagnosed with non-myocardial infarction, since actually irreversible myocardial damage had occurred. Indeed, WHO definition aimed for high specificity for purposes of risk stratification and subsequent treatment, a sensitive detection of cardiac injury is needed. Much more sensitive detection of (minimum) myocardial damage, assays for the cardiac troponin T and I, are also highly specific, formed the basis of a revised definition of myocardial infarction as recently proposed by the joint ESC/ACC task force fits with the patient's clinical course.[8]

Cardiac-specific troponins are gaining acceptance as the primary biochemical cardiac marker in patients with acute coronary syndromes. They have greater diagnostic sensitivity due to their ability to identify patients with lesser amount of myocardial damage. Elevated levels of cTn I convey prognostic information beyond that supplied by the clinical characteristics of the patient or the ECG at presentation. Furthermore, among patients without ST-segment elevation and normal CK-MB levels, elevated cTn I or troponin T concentrations identify those at an increased risk of death and are thought to represent microinfarctions that result from microemboli from an unstable plaque.[8]

One study done in Dhaka, showed that bedside troponin-T test had better sensitivity, similar or better specificity and improved efficiency over CK-MB in the diagnosis of AMI at the time of admission and also useful in thrombolytic therapy or early coronary angioplasty may be in order, rather than regulating these patients to conservative management.[9]

Another study found the greater prevalence of angiographically significant stenosis in ACS patients with high cTn I level group than low cTn I level group.[10] The current study was intended to find whether cTn I on admission could assess any association with coronary angiographic severity in STEMI patients that may provide an integrated and improved delineation of the spectrum of acute STEMI patients.

A total of 100 patients with diagnosis of acute ST segment elevation myocardial infarction who were admitted in the CCU of NICVD, were selected for the study after considering inclusion and exclusion criteria. All the patients had ECG on admission. Of all the patients, serum cardiac troponin I level were estimated after hospital admission. Coronary angiogram for above mentioned patients were done during index hospital admission. On the basis of serum cardiac troponin, I level on admission patients were categorized into two groups. Group-I, cTn I level ≥20 ng/ml, and Group-II, cTn I level <20 ng/ml. 50 patients were included in group I and 50 patients were included in group II.

In clinical examination of the patients it was observed that the mean duration of chest pain from symptom onset to presentation was 9.1 hours and 7.0 hours in group I and group II respectively. It was slightly greater in group I than group II, which was statistically significant (p=0.04). When duration of chest pain >4 hours was considered as delayed presentation 68.0% were found in group I and 60.0% in group II. One study found the mean duration of chest pain was 9.4 hours in group I and 7.2 hours in group II, which closely resembled with our study.[10]

In this study the mean cTn I level in group I was 37.2±14.6 ng/ml and in group II 13.8±4.7 ng/ml, the difference was statistically significant (p=0.001). One article reported in their study the mean cTn I levels in the two groups as 46.74 ng/ml and 1.88 ng/ml.[11] These findings were consisted with the present study.

Correlation between cardiac troponin I level and stenosis score (Modified Gensini Score) showed that the two variables exhibit significantly positive high correlation (r=0.91, p=0.01), one study found similar correlation in their study.[12]

In this study it was also found that 31 (62%) patients in group I and no patients in group II had more severe stenosis, score ≥ 40. 19 (38%) patients in group I and 50 (100%) patients in group II had less severe stenosis, score <40. The difference between the two groups was statistically highly significant (p=0.001), according to their angiographic severity by Modified Gensini Score.

LIMITATIONS
- A single Centre study with a relatively small cohort size.
- Serial cardiac troponin I level estimation was not done.
- Difference of CAG findings between thrombolytic recipients and non-recipients was not compared.
- Coronary angiography was evaluated by visual estimation, so there was chance of inter-observer and intra-observer variation of interpretation of the severity of stenosis.

CONCLUSION
From our study we can say that, estimation of serum cardiac troponin I might facilitate the triage of patients with acute myocardial infarction by clinicians and more aggressive approach to promote myocardial reperfusion might be warranted in the patients with high cardiac troponin I level.

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