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Elevation of Troponin I in Ischemic Stroke of Outcomes in Patients with Acute Stroke

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ABSTRACT

Background: Stroke is the second leading cause of death in the world, comprising approximately 10% of all deaths. Troponin is a sensitive marker of myocardial injury. Rise in serum troponin is characteristic for myocardial ischemic injury; however it can rise in several other conditions (e.g. renal failure, heart failure, pulmonary edema, and sepsis). Stroke is the second leading cause of death worldwide, comprising approximately 10% of all deaths. A substantial number of stroke patients have elevated cardiac troponin levels and are associated with poorer prognosis.

Methods: This prospective observational study was done at the department of neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), Dhaka, Bangladesh from January to 100 acute ischemic stroke patients was enrolled. Data included vital signs, laboratory parameters, and clinical features evaluated at the time of admission. The National Institute of Health Stroke Scale (NIHSS) and modified Rankin Scale (mRS) were used to assess stroke severity and outcomes.

Results: A total of 100 patients with acute ischemic stroke were enrolled in the study, and clinical and demographic characteristics the mean age was 58.68 ± 17.36. The mean age of patients in both groups. Elevated troponin I (>0.034 mg/ml) was observed in eight (8.00%) patients. Compared to patients with normal troponin I, patients with elevated troponin I were older (mean age 61 *vs.* 59.68 years), had higher blood glucose(10.6 *vs.* 7.04 mmol/L), higher median white blood cells (9.3 *vs.* 8.9 1,000/m^3) and creatinine levels(118.5 µmol/L *vs.* 94.9 µmol/L), higher mean NIHSS scores on admission (15 *vs.* 8.6), and discharge (13.5 *vs.* 6.8), higher median mRS scores (4.13 *vs.* 1.8) at discharge (p^{13} (OR 15.902; 95% CI (3.6569.28), p=0.029 mg/mL (odds ratio (OR):28.451; 95% CI (2.785-290.6), p=0.005) were significant predictors of poor outcomes. Significant predictor of in hospital mortality only included troponin I level>0.04 ng/mL (OR 0.071; 95% CI (0.005-1.037), P=0.05).

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Conclusion: Elevation of troponin I in ischemic stroke independently predicted unfavorable functional outcomes at discharge and in hospital mortality. Compared to other laboratory parameters, troponin I is a better predictor of outcomes of stroke. Careful and prompt evaluation of patients with acute stroke is needed in the setting of elevated troponin I.

Keywords: Stroke; Troponin I; Hospital mortality; Outcome; Standard deviation

INTRODUCTION

Troponin is a sensitive marker of myocardial injury [1]. Rise in serum troponin is characteristic for myocardial ischemic injury; however it can rise in several other conditions (e.g. renal failure, heart failure, pulmonary edema, and sepsis) [2,3]. Stroke is the second leading cause of death in the world, comprising approximately 10% of all deaths [4]. Cardiac mortality accounts for 20% of deaths and is the second commonest cause of death in the acute stroke population, second only to neurologic deaths as a direct result of the incident stroke [5]. Cardiac troponin T or I is elevated in a substantial number of acute stroke patients. It is associated with a higher number of in hospital cardiac complications and deaths. Compared to cardiac troponins other cardiac markers like CK-MB or myoglobin and ECG are less accurate [6]. Cardiac troponins are important biomarkers of acute myocardial infarction and are routinely studied in ischemic heart disease [7]. Cardiac indicators in acute ischemic stroke were first documented in the late 1970's [8]. Prevalence of symptomatic and asymptomatic ischemic heart disease in acute stroke has been reported to be 20% to 30% and 40%, respectively [9]. Cardiac troponins are important biomarkers of acute myocardial infarction and are routinely studied in the setting of ischemic heart disease. Abnormal levels of cardiac troponins have also been reported to be associated with poor clinical outcome in patients with acute cerebrovascular diseases, including ischemic stroke. Increased troponin I after stroke is associated with elevated circulating levels of epinephrine through activation of the sympathetic nervous system [10]. The sympathetic activation may be responsible for myocardial damage in acute stroke [11]. Available laboratory parameters, clinical features, and biomarkers during acute stroke provide valuable information when investigating the clinical outcomes after stroke. Some studies have reported an independent association of elevated troponin I with both case fatality, and the combined poor outcomes of mortality or disability [12,13]. However, other investigators have found no independent association. Some researchers have found association between troponin level and location and size of infarction, severity of stroke (measured by National Institutes of Health Stroke Scale (NIHSS)), ischemic Electrocardiogram (ECG) changes and increased mortality [14,15]. The purpose of this study is to investigate the elevation of Troponin I in ischemic stroke of outcomes in patients with acute stroke.

MATERIALS AND METHODS

This prospective observational study was done at the department of neurology, Bangabandhu Sheikh Mujib Medical University (BSMMU), and Dhaka, Bangladesh from January. A total of 100 patients were enrolled in the study. All adult patients who gave informed signed consent, with the diagnosis of acute ischemic stroke that was confirmed by clinical presentation and proof of an ischemic lesion and/or absence of a corresponding intracranial lesion other than infarction by brain computed tomography or magnetic resonance imaging were included in the study. Patients with history of myocardial infarction in the past 4 weeks and previous renal impairment were excluded from the study. A detailed history including the presence of hypertension, diabetes mellitus, smoking, and alcohol intake was obtained, NIHSS scoring was done and underwent cardiac enzyme testing including troponin I level at the time of admission/ within 48 hours of admission regardless of the stroke onset, 12 lead electrocardiograph, and other investigations at the time of admission according to our institute's protocol. An adult who has smoked 100 cigarettes in his or her lifetime and either quit at the time of interview (former smoker) or continue smoking (current smoker), both were included [16]. Blood pressure was recorded using the standard sphygmomanometer at brachial artery and radial artery was used to record the pulse manually.

Statistical Analysis

Data were analyzed using descriptive as well as analytical approaches. Continuous variables are presented as mean values and Standard Deviation (SD) for normally distributed data. Categorical variables are presented as absolute values and percentages. As most variables were normally distributed, comparisons between groups for continuous variables were made by Student t-test and ANOVA for independent samples, and Pearson's χ^2 test or Fisher's exact test for categorical variables (as appropriate). A p-value of less than 0.05 was considered to indicate statistical significance. All statistical analyses were performed using the statistical package SPSS.

RESULTS

A total of 100 patients with acute ischemic stroke were enrolled in the study, and clinical and demographic characteristics are presented below. The mean age was 58.68 ± 17.36. The mean age of patients in both groups of 100 stroke patients, 51 had large vessel atherosclerosis (51.00%), 24 had cardio embolic stroke (24.00), 13 patients had small

vessel stroke (13.00%), 11 had stroke due to undetermined cause (11.00%), and 1 patient had stroke due to other determined etiology (1%). The major risk factor for ischemic stroke was hypertension (n=21, 21.00%), followed by smoking seen in 13 cases (13.00%) and diabetes mellitus in 3 cases (3.00%). In 19.00% of cases, both smoking and HTN were present, while in 4 cases both DM and HTN were risk factors and in the other 4 cases, smoking, HTN, and DM were present. Troponin I was elevated in 8 cases (8.00%) of acute ischemic stroke, as shown in **Table 1**.

Table 1: Prevalence of troponin I in ischemic stroke.

Positive	8%
Negative	92%

Troponin was elevated in 8 cases and 5 cases were associated with ECG changes and 3 had normal ECG despite elevated troponin I (p=0.012). The most common ECG changes seen in acute ischemic stroke patients were atrial fibrillation (n=19, 19.00%), T wave inversion was seen in 6 cases (6.00%), ST elevation, LBBB, R BBB was seen in 1 case each and 3 cases had features of LVH(3%) as shown in **Table 2**.

Table 2: Relation between troponin I and ECG changes.

	Positive	Negative	Total
RBBB	0	1	1
LVH	1	2	3
LBBB	1	0	1
Atrial fibrillation	2	17	19
T wave inversion	1	5	6
ST elevation	0	1	1
Normal	3	66	69

Patients with abnormal troponin I had significantly higher blood glucose (p=0.033). However, patients with abnormal troponin I and normal troponin I had similar baseline heart rate (p=0.339), white blood cell count (p=0.689), creatinine (p=0.129), and age (p=0.843). However, heart rate controlling drugs were being taken in the few patients (n=9) in the normal troponin I group and none in the elevated troponin group. Hemoglobin (p=0.620) and hematocrit (p=0.1360) were also not statistically different between the groups. In addition, patients with abnormal troponin I had a higher mean NIHSS score on admission (15) and on discharge (13.5) than patients with normal troponin I levels (8.66 and 6.89). The mean MRS score was higher (4.13) in patients with abnormal troponin I than in patients with normal troponin I levels (1.84, p=<0.001), as shown in **Tables 3 and 4**.

Table 3: Relation between troponin I and type of stroke.

	Normal Troponin I	Abnormal Troponin I	Total
Small vessel stroke	12	1	13
Large vessel atherosclerosis	48	3	51
Cardioembolism	21	3	24
Special cause	1	0	1
Undetermined etiology	10	1	11

Table 4: Comparison of clinical features, laboratory data, severity of stroke, and outcomes of patients with different levels of troponin.

Characteristics	Troponin I test				P-Value
	Normal (n=92)		Abnormal (n=8)		
	Mean	S.D	Mean	S.D	
Mean age (years)**	58.68	17.36	60	12.98	0.843
Systolic pressure (mmHg)**	134.58	24.55	120.25	20	0.127
Diastolic pressure (mmHg)**	83.19	13.98	79	16.72	0.456
Heart rate (Bpm)**	82.58	10.61	78.5	10.33	0.339
White blood cells (1000/mm^3)*	8906.74	2860.83	9367.75	4894.69	0.683
Glucose(mmol/L)**	7.04	3.71	10.6	10.01	0.033
Creatinine (µmol/L)*	94.98	41.44	118.5	28.73	0.129
NIHSS score (on admission)**	8.66	4.21	15	7.61	<0.001
NIHSS score (on discharge)**	6.89	4.32	13.5	7.94	<0.001
Modified rankin scale**	1.84	1.36	4.13	1.24	<0.001
Death*	3		2		0.006
Poor outcome mRS>2*	26		8		<0.001

Data presented as *number and ** Mean ± S.D

Abnormal troponin I were common in patients with stroke due to large artery atherosclerosis (3/51=5.8%) and cardio embolism (3/24=12.5%) than in patients with stroke due to small vessel disease and other determined etiology according to TOAST classification (p=0.890). Poor outcomes were observed in 34 (34.00%) patients of 100 patients and death occurred in 5 (5.00%) patients. Patients with abnormal troponin I had poorer outcomes than normal troponin I level patients (p<0.001), as shown in **Table 5** and significantly higher deaths (p=0.006), as shown in **Table 6**. Length of hospital stay was similar in both groups (10.24 *vs*. 11, p=0.50). Univarate analysis of continuous variables revealed that patients with poor outcomes compared to good outcomes had higher troponin levels (0.029 *vs*. 0.013 ng/ mL, p=0.001), creatinine levels (112.58 *vs*. 88.86 µmol/L, p=0.007), NIHSS score on admission (12.4 *vs*. 5.10, p<0.001), discharge (11.4 *vs*. 5.1, p<0.001), and higher mRS scores at discharge (3.71 *vs*. 1.16, p<0.001). Other variables including age, total count, hematocrit, blood glucose, and heart rate were similar between both the groups analysis. Dichotomous variables revealed that male sex and cardio embolic stroke were not associated with poor outcomes.

Table 5: Correlation of the factors affecting the outcomes of ischemic stroke.

Characteristics	Poor outcomes (mRS>2)				P value
	Yes (n=34)		No (n=66)		
	Mean	S.D	Mean	S.D	
Mean age (years)**	57.97	17.74	60.38	15.61	0.527
Systolic pressure (mmHg)**	135.7	23.64	131	25.74	0.212
Diastolic pressure (mmHg)**	83.63	13.49	83.35	15.52	0.479
Heart rate (Bpm)**	82.94	10.94	80.91	11.64	0.406

White blood cells (1000/mm^3)**	8730.04	2937.88	9363.41	3228.4	0.325
Glucose (mmol/l)**	7.23	4.22	7.5	5.16	0.784
Creatinine (µmol/L)**	88.86	24.32	112.58	59.55	0.007
Troponin I (ng/mL)**	0.013	0.0032	0.029	0.038	0.001
NIHSS score (on admission)**	7.1	3.51	12.44	4.65	<0.001
NIHSS Score (on discharge)**	5.1	3.085	11.4	4.43	<0.001
Modified rankin scale**	1.16	0.751	3.71	1.06	<0.001
Length of stay (days)**	9.43	4.18	11.09	7.54	0.025
Cardioembolism*	2		12	0.158	
Male sex*	9		30	0.291	

Data presented as *number (percent) and **mean ± S.D

In hospital, deaths were associated with higher total counts (11903, p=0.02), creatinine levels (157.6, p=0.001), and troponin I levels (0.043 ng/mL, p=0.016). NIHSS score on admission did not show a significant association with in hospital mortality. Length of stay was also similar between the groups. Dichotomous analysis showed a significant correlation between death and cardioembolic stroke. Age and sex were not associated with in hospital death, as shown in **Table 6**.

Table 6: Correlation of clinical features and in hospital mortality in 100 acute ischemic stroke patients.

Characteristics	**Death**				**P value**
	Yes (n=5)		**No (n=95)**		
	Mean	S.D.	Mean	S.D.	
Mean age (years)**	68	18.33	58.3	16.91	0.242
Systolic pressure (mmHg)**	127	18.235	133.78	24.74	0.564
Diastolic pressure (mmHg)**	83	22.02	82.85	13.82	0.983
Heart rate (Bpm)**	84.6	4.09	82.14	10.77	0.643
White blood cells (1000/mm^3)**	11903	5442.97	8789.1	2824.07	0.025
Glucose (mmol/l)**	5.94	1.11	7.39	4.64	0.22
Creatinine (µmol/L)**	157.6	97.28	93.68	34.24	0.001
Troponin I (ng/mL)**	0.043	0.037	0.01743	0.022	0.016
NIHSS score (on admission)**	10.8	7.25	9.16	4.824	0..470
Modified rankin scale**	6		1.81	1.199	<0.001
Length of stay (days)**	11	11.787	10.24	5.15	0.5

Cardioembolism*	2 (40%)	22 (23.15%)	<0.001
Male gender*	3 (60%)	46 (48.42%)	0.598

Data presented as *number (percent) and **mean ± S.D

Multiple regression analysis of the significant dichotomous variables was done with cut-off levels according to the mean values of poor outcomes. Multiple logistic regression analysis revealed that NIHSS score on admission >13 (odds ratio (OR) 15.902; 95%CI (3.65-69.28), p=<0.001) and abnormal troponin I level, troponin I>0.029 mg/mL) (odds ratio (OR): 28.451; 95% CI (2.785-290.6), p=0.005) were significant predictors of poor outcomes; however, creatinine levels did not show any significance in predicting the outcomes, as shown in **Table 7**.

Table 7: Regression model of factors influencing outcomes in 100 patients with acute ischemic stroke.

Characteristics	B	S.E.	Wald	Df	P value	Expo (B)	95% CI for EXP (B)	
							Lower	Upper
NIHSS score on admission >13	2.766	0.751	13.572	1	0	15.902	3.65	69.287
Creatinine> 113 µmol/L	1.047	0.87	1.45	1	0.229	2.849	0.518	15.661
Troponin I>0.029 ng/mL	3.348	1.186	7.974	1	0.005	28.451	2.785	290.63
Constant	9.417	1.943	23.501	1	0	0		4

Only troponin I level>0.04 ng/mL (odds ratio (OR) 0.071; 95% CI (0.005-1.037), P=0.05) was a significant predictor of in-hospital mortality, as shown in **Table 8**.

Table 8: Regression model of factors influencing deaths in patients with acute ischemic stroke.

Characteristics	Std. Error	Wald	Sig.	Expo (B)	95% Confidence interval for Expo (B)	
					Lower	Upper
Total WBC>11900	1.117	3.037	0.081	0.143	0.016	1.275
Creatinine>158 µmol/L	1.328	3.347	0.067	0.088	0.007	1.189
Troponin I>0.04 ng/mL	1.368	3.737	0.05	0.071	0.005	1.037

DISCUSSION

Stroke is the second fourth most common cause of death, after Ischemic Heart Disease (IHD); meanwhile, IHD is the second most common cause of death after stroke. This study demonstrates the prognostic significance of elevated troponin in patients with ischemic stroke. Patients with increased troponin I had increased chances of unfavorable functional outcomes at discharge and mortality. Troponin I is a highly sensitive and specific marker of acute myocardial infarction. Hence other cardiac biomarkers were not considered for evaluation in this study. Elevated troponin I am characteristic of a number of cardiac diseases as well such as heart failure, pericarditis, myocarditis, atrial fibrillation, and tachycardia. Elevated troponin I have also been found in patients with chronic renal failure, sepsis, critical illness, pulmonary embolism, chronic obstructive pulmonary disease, and stroke [17]. Elevated levels of cardiac troponin have been reported in 10-34% of patients with acute stroke. Some studies reported that elevated troponin levels were more common in patients with stroke due to cardio embolism, who also had evidence of atrial fibrillation, ischemic heart disease, or heart failure [18,19]. Abnormal troponin I levels were observed in 8.00% of the patients in our study. We found that patients with

abnormal troponin I was more likely to have large artery atherosclerosis and cardio embolism. Patients with elevated troponin I levels were older and had higher creatinine levels and blood glucose levels than patients with normal troponin I levels. Patients with elevated troponin I presented with more severe initial stroke severity. Worse outcomes and higher in hospital mortality were observed in patients with abnormal troponin I, similar to those reported by Di Angel Antonio [20]. Increase in catecholamine release as a result of hypo perfusion of the posterior hypothalamus occurs in neurologic causes resulting in autonomic nervous system imbalance and increased sympathetic output. Involvement of the insular cortex is known to be associated with more frequent occurrences of cardiac complications after acute stroke. Brain magnetic resonance imaging to identify the precise location of the stroke was not performed in all patients; therefore, we were not able to analyze the involvement of the insular or parietal cortex. Higher troponin levels were associated with the severity of stroke rather than the location of stroke. Abnormal troponin levels were more likely to be due to cardiac and renal causes than the cerebral causes. The average age of patients with abnormal troponin I, patients with poor outcomes, and patients who died in the hospital in this study was approximately 65 years. reported that age ≥ 76 years was independently associated with elevated troponin levels in patients with acute ischemic stroke. But in our study, age was not significantly different between the groups. In our study, blood glucose was significantly elevated in the positive troponin I group however was not associated with poor outcomes. Similarly, elevated creatinine level >112 µmol/L at the time of admission was associated with poor outcomes of stroke. Showed that elevated creatinine levels at the time of admission were associated with increased mortality in stroke patients. With the exception of the NIHSS score on admission, only elevated troponin I was a strong independent predictor of both poor outcomes and death. Abnormal troponin I had an OR of 28.451 for poor outcomes and an OR of 0.071 for in hospital mortality. The American stroke association recommends the routine checking of markers of cardiac ischemia during acute stroke. Dynamic changes in the troponin levels (>50%) within 24 hour in stroke patients were better indicator for in hospital mortality than the patients whose troponin levels were elevated but remain stable. Elevation of troponin level has been associated with increased risk of mortality and disability compared to other factors as shown in multiple studies. In the present study, abnormal troponin I was a predictor of both poor outcomes and in hospital mortality. An NIHSS score ≥ 12 on admission was also a strong predictor of both poor outcomes and in hospital mortality. This study has a number of limitations. Cardiac illness like cardiomyopathy and heart failure were not excluded from the study. Troponin was only examined in a subgroup of patients and was not systematically assessed in all patients presenting with ischemic stroke. Troponin I was checked only once in each patient in the emergency room without a known lag time between exact time period of onset of symptoms and troponin measurement, without dynamic assessment.

CONCLUSION

Elevation of troponin I in ischemic stroke independently predicted unfavorable functional outcomes at discharge and in hospital mortality. Compared to other laboratory parameters, troponin I is a better predictor of outcomes of stroke. Careful and prompt evaluation of patients with acute stroke is needed in the setting of elevated troponin I.

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