

ORIGINAL ARTICLE

Estimation of Serum Troponin-I in ST Elevated MI Patient and Prediction of their in-Hospital Outcome

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ABSTRACT

Background: Troponin-I is a highly sensitive biomarker for myocardial injury and may predict in-hospital outcomes in ST-elevation myocardial infarction (STEMI) patients. This study aimed to estimate serum troponin-I levels in STEMI patients and evaluate their prognostic significance for in-hospital outcomes. **Methods & materials:** This cross-sectional observational study included 100 STEMI patients admitted to Sir Salimullah Medical College & Mitford Hospital, Dhaka, Bangladesh. Serum troponin-I levels were measured at admission and patients were categorized into three groups: <0.034 ng/mL, 0.034 – 0.12 ng/mL, and ≥ 0.12 ng/mL. In-hospital outcomes including arrhythmia, cardiogenic shock, heart failure, hospital stay duration, and mortality were recorded. **Results:** The mean age was 52.5 ± 13.1 years, with males comprising 59% of patients. Hypertension (63%), smoking (47%), and diabetes mellitus (42%) were the most common risk factors. Arrhythmia rates increased from 0% (<0.034 ng/mL) to 46.1% (≥ 0.12 ng/mL); cardiogenic shock rose from 3.7% to 23.0%; and heart failure rose from 0% to 30.7%. Hospital stays >7 days were observed in 76.9% of patients with troponin-I ≥ 0.12 ng/mL. Mortality was highest (38.5%) in this group, compared to 0% in the lowest troponin category. **Conclusion:** Elevated admission troponin-I levels are associated with increased in-hospital complications, prolonged hospitalization, and mortality among STEMI patients. Troponin-I measurement serves as a valuable tool for early risk stratification in acute STEMI management.

Keywords: Troponin-I, ST-elevation myocardial infarction (STEMI), In-hospital outcomes, Risk stratification

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INTRODUCTION

Coronary artery disease (CAD) continues to be the leading cause of mortality globally, representing a critical public health challenge. According to the World Health Organization (WHO), cardiovascular diseases (CVDs), predominantly ischemic heart disease, accounted for approximately 17.9 million deaths globally in 2019, representing 32% of all deaths worldwide. Remarkably, over three-quarters of these deaths occur in low- and middle-income countries (LMICs), underscoring the stark health inequities that exist between different global regions [1]. This disparity persists despite substantial advancements in therapeutic interventions and preventive cardiology measures, highlighting the urgent need for targeted research and interventions, especially in resource-limited settings [2]. Acute coronary syndrome (ACS),

a manifestation of coronary artery disease, encompasses a clinical spectrum ranging from unstable angina through non-ST elevation myocardial infarction (NSTEMI) to ST-elevation myocardial infarction (STEMI). STEMI, characterized by complete thrombotic occlusion of a coronary artery, remains associated with particularly high morbidity and early mortality rates if not promptly addressed with appropriate reperfusion strategies [3]. Indeed, untreated STEMI can result in up to 30% mortality within the initial hours to days following onset [4]. In contrast, NSTEMI and unstable angina typically involve partial coronary obstruction, resulting in comparatively lower immediate risk but substantial longer-term morbidity and mortality [5]. The rapid identification and stratification of patients based on their clinical severity and associated risk factors thus become critical steps in improving

patient outcomes. In recent decades, cardiac biomarkers have emerged as crucial diagnostic and prognostic tools in ACS. Among these biomarkers, the cardiac troponins; regulatory proteins consisting of T, I, and C subunits have demonstrated superior specificity and sensitivity for myocardial injury compared to previously used markers such as creatine kinase-MB (CK-MB). Troponin-I, in particular, is highly specific to cardiac muscle injury, with diagnostic sensitivity that surpasses CK-MB significantly, making it a gold-standard marker in diagnosing myocardial infarction [6,7]. The kinetics of troponin-I also underscore its clinical utility; it typically becomes detectable in serum 4 to 12 hours after myocardial injury, peaks between 14 to 36 hours, and remains elevated for approximately 5 to 7 days, providing a robust window for diagnosis and prognostication in ACS patients [8]. Beyond its diagnostic role, troponin-I plays a significant role in risk stratification and prognostication among patients with ACS. Landmark clinical trials, including the Fragmin and Fast Revascularization during Instability in Coronary artery disease (FRISC-II) and the Global Use of Strategies to Open Occluded Coronary Arteries (GUSTO-III), have firmly established the prognostic importance of troponin measurements. Elevated troponin levels at admission correlate strongly with adverse outcomes such as increased mortality, arrhythmias, cardiogenic shock, and heart failure, both at 30-day and 6-month follow-up periods [9,10]. Clinically validated cut-offs of troponin levels have therefore been implemented in practice to classify patients into low, intermediate, and high-risk categories, guiding timely clinical decisions, aggressive interventions, and resource allocations [11]. In Bangladesh, cardiovascular diseases have increasingly emerged as leading contributors to morbidity and mortality. According to data from the INTERHEART study, Bangladesh reported the highest prevalence of key risk factors for CVD in the South Asian region, including hypertension (14.3%), smoking (59.9%), and low fruit and vegetable intake (8.6%) [12]. Rapid urbanization, combined with significant lifestyle changes such as increasing sedentary behavior, dietary shifts, and escalating prevalence of obesity, diabetes mellitus, and dyslipidemia, has exacerbated the burden of CAD in the country [13]. Moreover, despite this growing burden, Bangladesh still lacks comprehensive, large-scale, population-based studies focusing specifically on ACS outcomes. Most existing studies tend to be hospital-based, single-center analyses with limited generalizability, highlighting a critical knowledge gap that impedes the formulation of effective national health policies and preventive strategies [14]. Recognizing these gaps, this current research aims to examine the predictive value of serum troponin-I measurement at admission for in-hospital outcomes among STEMI patients in Bangladesh. It is hypothesized that higher levels of troponin-I at admission will correlate significantly with increased risk of arrhythmias, cardiogenic shock, heart failure, prolonged hospitalization, and mortality. By addressing the prognostic implications of serum troponin-I in a Bangladeshi cohort, this study seeks to contribute vital evidence to the existing literature, potentially influencing clinical practices and

guiding future research and policy initiatives in the context of ACS management in Bangladesh.

METHODS AND MATERIALS

This cross-sectional observational study was conducted in the Department of Medicine and Cardiology at Sir Salimullah Medical College & Mitford Hospital, Dhaka, Bangladesh, over a period of six months from June 12, 2018, to December 11, 2018. The study population included adult patients aged 30 to 70 years admitted with ST-elevation myocardial infarction (STEMI) confirmed by clinical evaluation and electrocardiographic (ECG) evidence. Patients were selected using purposive sampling after applying inclusion and exclusion criteria. Patients with non-STEMI, previous cardiac events including old MI, history of coronary artery bypass grafting (CABG) or percutaneous coronary intervention (PCI), valvular heart disease, cardiomyopathy, malignancy, chronic pulmonary diseases such as bronchitis, asthma, old pulmonary tuberculosis, chronic kidney disease, and those aged below 30 or above 70 years were excluded. A total of 100 patients were enrolled. After obtaining informed written consent, detailed medical history and complete physical examination were performed for each patient, recording cardiovascular risk factors such as hypertension, diabetes mellitus, smoking status, dyslipidemia, obesity, and family history of premature coronary artery disease. Body mass index (BMI) and blood pressure were measured. Venous blood samples were collected at admission for routine investigations including fasting blood glucose, serum creatinine, lipid profile, and serum troponin-I estimation. Serum troponin-I concentration was determined using an immunometric assay (Vitros Troponin-I ES Reagent Pack; Johnson & Johnson, USA) performed on the Vitros ECI Cube System. Samples were collected in plastic tubes without anticoagulant, centrifuged at 4000 rpm for 10 minutes, and processed within standard laboratory protocols. The assay used a cut-off value of ≥ 0.12 ng/mL to diagnose acute myocardial infarction, with risk stratification categories defined as < 0.034 ng/mL (low risk), $0.034-0.12$ ng/mL (average risk), and ≥ 0.12 ng/mL (high risk). All patients were monitored during hospital stay for outcomes including duration of hospitalization, development of arrhythmia, cardiogenic shock, heart failure, or in-hospital mortality. Data were collected using a structured case record form and entered into Microsoft Excel, then analysed with SPSS version 20.0 (IBM Corp., Armonk, NY, USA). Quantitative data were expressed as mean and standard deviation, while qualitative data were presented as frequencies and percentages. Comparisons were performed using Chi-square test for categorical variables and Student's t-test for continuous variables. A p-value < 0.05 was considered statistically significant. The study protocol was reviewed and approved by the Institutional Review Board of Sir Salimullah Medical College & Mitford Hospital, and all procedures were performed in accordance with the Declaration of Helsinki.

RESULTS

Table I shows the baseline demographic and clinical characteristics of the study population. The mean age of patients was 52.5 ± 13.1 years, ranging from 29 to 68 years. The majority of patients were aged between 41 and 50 years (34%), followed by 51–60 years (27%). Males comprised 59% of the study population, while females accounted for 41%. Regarding occupation, 29% were involved in business, 26% were day labourers, 25% were housewives, and 20% were service holders. [Table I]

Table – I: Baseline Demographic and Clinical Characteristics of Patients (n = 100)

Characteristic	n	%
Age Group (years)		
≤30	5	5.0
31–40	21	21.0
41–50	34	34.0
51–60	27	27.0
>60	13	13.0
Mean ± SD	52.5 ± 13.1	
Range (years)	29–68	
Sex		
Male	59	59.0
Female	41	41.0
Occupation		
Business	29	29.0
Service	20	20.0
Day laborer	26	26.0
Housewife	25	25.0

Table II presents the distribution of cardiovascular risk factors among STEMI patients. Hypertension was the most

common risk factor, present in 63% of patients. Diabetes mellitus was noted in 42% of patients, while 47% were smokers. Dyslipidemia was present in 22% of the study population, and 27% had a family history of coronary artery disease. [Table II]

Table – II: Cardiovascular Risk Factors among STEMI Patients

Risk Factor	Yes n (%)	No n (%)
Hypertension	63 (63.0)	37 (37.0)
Diabetes Mellitus	42 (42.0)	58 (58.0)
Smoking	47 (47.0)	53 (53.0)
Dyslipidemia	22 (22.0)	78 (78.0)
Family History of CAD	27 (27.0)	73 (73.0)

Table III shows the distribution of serum troponin-I levels among STEMI patients. The majority of patients (60%) had troponin-I levels between 0.034 and 0.12 ng/mL, while 27% had levels below 0.034 ng/mL. A total of 13% of patients had troponin-I levels equal to or greater than 0.12 ng/mL. The mean troponin-I level in the study population was 0.1 ± 0.05 ng/mL. [Table III]

Table – III: Serum Troponin-I Levels in STEMI Patients

Troponin-I Level (ng/mL)	n	%
<0.034	27	27.0
0.034–0.12	60	60.0
≥0.12	13	13.0
Mean ± SD	0.1 ± 0.05	

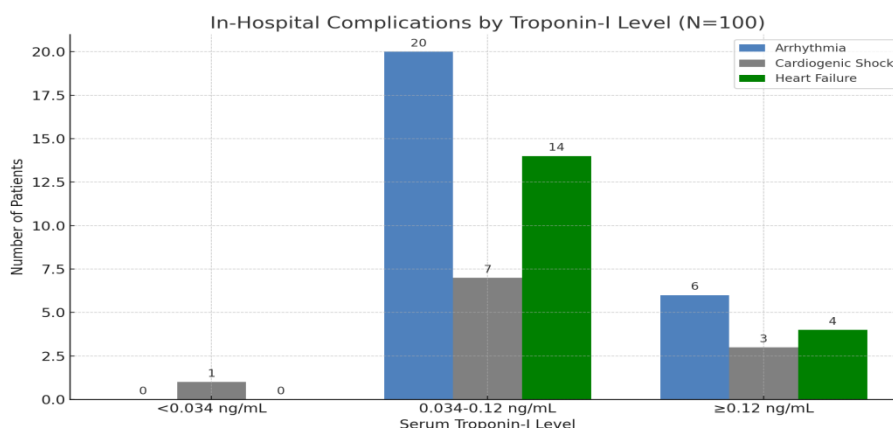


Figure – 1: In-Hospital Complications by Troponin-I Level

Figure 1 illustrates the distribution of in-hospital complications according to serum troponin-I levels. Patients with troponin-I levels between 0.034 and 0.12 ng/mL had the highest frequency of arrhythmia (20 patients), cardiogenic shock (7 patients), and heart failure (14 patients). In the group with troponin-I levels ≥ 0.12 ng/mL, 6 patients developed arrhythmia, 3 experienced cardiogenic shock, and 4 developed heart failure. Minimal complications were observed among patients with troponin-I levels < 0.034 ng/mL.

Table IV shows the distribution of in-hospital outcomes according to serum troponin-I levels. Arrhythmia occurred in 33.3% of patients with troponin-I levels between 0.034 and 0.12 ng/mL and in 46.1% of those with levels ≥ 0.12 ng/mL, while no cases were reported in the < 0.034 ng/mL group. Cardiogenic shock was observed in 3.7% of patients with troponin-I < 0.034 ng/mL, 11.6% in the 0.034–0.12 ng/mL group, and 23.0% in the ≥ 0.12 ng/mL group. Heart failure occurred in 23.3% of patients with troponin-I levels between

0.034 and 0.12 ng/mL and in 30.7% of those with levels ≥ 0.12 ng/mL, with no cases in the <0.034 ng/mL group. Regarding hospital stay, all patients with troponin-I <0.034 ng/mL were

discharged within 3–5 days, while the majority in the ≥ 0.12 ng/mL group stayed >7 days (76.9%). Overall, 45% of patients had no complications during hospitalization. [Table IV]

Table – IV: In-Hospital Outcomes by Serum Troponin-I Levels

Outcome	<0.034 ng/mL (n=27)	0.034–0.12 ng/mL (n=60)	≥ 0.12 ng/mL (n=13)	Total (N=100)
Arrhythmia	0 (0%)	20 (33.3%)	6 (46.1%)	26 (26.0%)
Cardiogenic shock	1 (3.7%)	7 (11.6%)	3 (23.0%)	11 (11.0%)
Heart failure	0 (0%)	14 (23.3%)	4 (30.7%)	18 (18.0%)
Hospital stay: 3–5 days	27 (100.0%)	17 (28.3%)	0 (0%)	44 (44.0%)
Hospital stay: 5–7 days	0 (0%)	39 (65.0%)	3 (23.0%)	42 (42.0%)
Hospital stay: >7 days	0 (0%)	4 (6.7%)	10 (76.9%)	14 (14.0%)
No complication	26 (96.2%)	19 (31.6%)	0 (0%)	45 (45.0%)

Table V presents recovery and mortality rates according to serum troponin-I levels. All patients with troponin-I levels <0.034 ng/mL recovered without any deaths. In the 0.034–0.12 ng/mL group, 96.6% recovered while 3.4% died. Among patients with troponin-I levels ≥ 0.12 ng/mL, 61.5% recovered and 38.5% died. Overall, the total mortality rate in the study population was 7%. [Table V]

Table – V: Recovery and Mortality Rates by Troponin-I Group

Troponin-I Level	Recovered n (%)	Death n (%)
<0.034 ng/mL	27 (100.0)	0 (0.0)
0.034–0.12 ng/mL	58 (96.6)	2 (3.4)
≥ 0.12 ng/mL	8 (61.5)	5 (38.5)
Total	93 (93.0)	7 (7.0)

Note. Recovery defined as discharge without death;

DISCUSSION

The present study evaluated the predictive value of serum troponin-I levels measured at admission in determining the in-hospital outcomes among STEMI patients in Bangladesh. Key findings of this study revealed a middle-aged cohort (mean age 52.5 ± 13.1 years), predominantly male (59%), and characterized by diverse occupational backgrounds, including businesspersons (29%), day labourers (26%), housewives (25%), and service holders (20%). This demographic profile aligns closely with other regional studies. Akhtar et al. reported a similar mean age of 52.6 ± 10.8 years and higher male predominance (88.7%) in their Bangladesh-based STEMI registry [15]. Similarly, Azad et al. (2020) found the mean age in a Dhaka STEMI cohort to be 53.25 ± 9.65 years, with males comprising 75% of their sample [16]. The cardiovascular risk profile identified in our study was notably high, with hypertension (63%), smoking (47%), and diabetes mellitus (42%) being most prevalent. This observation is consistent with other regional literature; Rabbani et al. reported hypertension in 56% and smoking in 49% of their Bangladeshi cohort, while Roy et al. noted even higher prevalence rates of smoking (59.5%) and hypertension (61.9%) among STEMI patients [17,18]. The prevalence of diabetes mellitus in our study aligns closely with Roy et al. (35.7%) and Hossen et al., emphasizing the substantial cardio metabolic risk burden among Bangladeshi STEMI patients

[18,19]. Serum troponin-I levels provided clear risk stratification in the current study. Most patients (60%) had intermediate troponin-I levels (0.034–0.12 ng/mL), while 13% exhibited high troponin-I levels (≥ 0.12 ng/mL). The significance of troponin-I levels in stratifying STEMI patients was also documented by Daniel et al. and Bularga et al., who highlighted troponin as a critical biomarker for predicting acute complications [20,21]. Our findings indicate that complications, including arrhythmia, cardiogenic shock, and heart failure, increased significantly with rising troponin-I levels. Arrhythmia prevalence climbed sharply from 0% in the lowest troponin group to 46.1% in the highest group. Cardiogenic shock and heart failure showed similar stepwise escalations (3.7% to 23.0% and 0% to 30.7%, respectively). These findings are consistent with Tambarta et al. and Polyzogopoulou et al., who found similarly strong correlations between elevated troponin-I levels and adverse cardiac outcomes such as cardiogenic shock and heart failure [22,23]. Moreover, hospital stay duration mirrored the troponin-I level stratification. Patients with low troponin levels (<0.034 ng/mL) uniformly had shorter hospital stays (3–5 days), whereas prolonged hospitalizations (>7 days) were predominantly observed among patients with troponin levels ≥ 0.12 ng/mL (76.9%). Similar observations were reported by Wanamaker et al. and Thielmann et al., who demonstrated prolonged hospitalization associated with higher admission troponin levels, reflecting the severity of myocardial injury and subsequent complications [24,25].

Recovery and mortality rates further emphasized the prognostic utility of troponin-I. Patients in the lowest troponin group exhibited complete recovery with no mortality, whereas those in the highest troponin group had markedly elevated mortality (38.5%). These results align closely with findings by Wanamaker et al., who demonstrated a significantly higher mortality rate (19.5%) among patients with elevated troponin levels [24]. Similarly, Cediell et al. confirmed that elevated troponin levels at admission independently predicted increased in-hospital and long-term mortality, underscoring troponin-I as a robust predictor of patient outcomes [26]. Overall, the current study reinforces the clinical importance of admission troponin-I measurement as a predictive biomarker for risk stratification, enabling clinicians to identify high-risk patients early and prioritize targeted interventions. The observed relationships between elevated

troponin-I levels, prolonged hospitalization, increased complication rates, and heightened mortality suggest a compelling case for routine use of troponin-I testing in resource-limited settings such as Bangladesh. Future large-scale multicenter studies are recommended to further validate these findings and inform national guidelines on STEMI management.

Limitations of the Study:

The study was conducted in a single hospital with a small sample size. So, the results may not represent the whole community.

CONCLUSION

In conclusion, this study demonstrated that serum troponin-I levels measured at admission are strong predictors of in-hospital outcomes among STEMI patients in Bangladesh. Higher troponin-I levels were associated with significantly increased rates of arrhythmia, cardiogenic shock, heart failure, prolonged hospital stays, and mortality. Patients with troponin-I levels ≥ 0.12 ng/mL experienced the highest complication and mortality rates, whereas those with levels < 0.034 ng/mL had no mortality and minimal complications. These findings underscore the utility of serum troponin-I as an effective biomarker for early risk stratification in STEMI, enabling clinicians to identify high-risk patients promptly and tailor management strategies accordingly. Routine assessment of troponin-I levels at admission, particularly in resource-limited settings, may enhance prognostication and optimize clinical outcomes. Further large-scale, multicenter studies are recommended to validate these findings and integrate troponin-based risk stratification into national STEMI management guidelines.

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