Original Research Article

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Correlation between cardiac troponin I level with adverse in-hospital outcome in patients with ST-segment elevation myocardial infarction

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Abstract

Introduction: Cardiac troponin-I (cTnI) is a well-established biomarker for diagnosing myocardial infarction. However, its prognostic value in predicting adverse in-hospital outcomes in ST-segment elevation myocardial infarction (STEMI) patients remains underexplored in Bangladesh. The objective of the study is to assess the correlation between admission cTnI levels and adverse in-hospital outcomes among STEMI patients.

Methods: This cross-sectional observational study included 100 STEMI patients admitted to Sir Salimullah Medical College and Mitford Hospital, Dhaka. Admission troponin-I levels were measured and categorized into <0.034 ng/mL, 0.034–0.12 ng/mL, and ≥0.12 ng/mL groups. In-hospital outcomes, including arrhythmia, cardiogenic shock, heart failure, and hospital stay duration, were recorded. Statistical analyses were conducted using Chi-square tests.

Results: Arrhythmia occurred in 0%, 33.3%, and 46.1% of patients in increasing troponin-I groups ($P < 0.001$). Cardiogenic shock incidence rose from 3.7% to 23% ($P = 0.011$), and heart failure increased from 0% to 30.7% ($P < 0.001$). Hospital stay >5 days was required by 0%, 71.7%, and 100% of patients across ascending troponin-I groups ($P < 0.001$). All associations were statistically significant except cardiogenic shock, which showed a moderate positive association.

Conclusion: Higher admission troponin-I levels are strongly associated with adverse in-hospital outcomes in STEMI patients. Admission troponin-I assessment can aid in early risk stratification to optimize management in resource-limited settings.

Keywords: Cardiogenic shock, in-hospital outcomes, risk stratification, ST-segment elevation myocardial infarction, troponin-I

Introduction

Acute myocardial infarction (AMI), particularly the ST-segment elevation myocardial infarction (STEMI) subtype, remains a leading cause of mortality globally. It contributes significantly to worldwide cardiovascular mortality, accounting for nearly 31% of all deaths, with a disproportionate impact on low- and middle-income countries.[1] In these settings, STEMI represents the most severe and life-threatening manifestation of AMI, often characterized by extensive heart muscle damage and high mortality rates due to delayed presentation and limited healthcare infrastructure.[2]

In Bangladesh, the burden of STEMI is notably high, with substantial healthcare challenges such as late patient presentations, inadequate transportation facilities, and limited availability of advanced cardiac care, including timely percutaneous coronary interventions (PCI). Studies from both urban and rural Bangladeshi populations highlight these issues clearly. For instance, Akhtar *et al*. reported alarmingly high rates of major adverse cardiac events in-hospital and at 30 days post-STEMI admission in Dhaka hospitals, reflecting significant constraints in cardiac care delivery.[3] Similarly, Kim *et al*. found that in rural Bangladesh, cardiogenic shock complicated more than half (56.25%) of STEMI cases, significantly contributing to high in-hospital mortality rates.[4]

At a cellular level, STEMI typically occurs due to sudden, complete blockage of a major coronary artery, resulting in extensive damage and death of cardiac muscle cells.[5] This process, known as myocardial necrosis, releases specific cardiac biomarkers into the bloodstream. Among these, cardiac troponin-I (cTnI) is the gold-standard biomarker widely used for diagnosing myocardial injury because of its remarkable sensitivity and specificity. cTnI is typically detectable within 3–4 h after the onset of myocardial infarction symptoms, peaks between 12 and 24 h, and remains elevated for 7–10 days, providing a critical diagnostic window.[6,7]

Beyond its diagnostic role, the clinical significance of cTnI has been extensively investigated as a predictor of patient outcomes post-STEMI. Elevated admission cTnI levels correlate closely with larger infarct size, increased risk of the left ventricular dysfunction, and poorer long-term prognosis.[8] However, despite this evidence, significant variability exists in the threshold levels of cTnI used across different populations, complicating its use for risk stratification universally.[9]

Adverse in-hospital outcomes associated with STEMI include in-hospital mortality, cardiogenic shock, major arrhythmias such as ventricular tachycardia and ventricular fibrillation, and acute heart failure.[10] In resource-constrained regions, these complications occur at significantly higher rates due to systemic healthcare deficiencies. For example, cardiogenic shock following STEMI has a mortality rate approaching 50% in low-resource settings.[11] Similarly, data from Pakistan indicate considerable rates of STEMI-related complications such as acute heart failure (11.1%) and significant arrhythmias (12.9%), underscoring the regional challenges.[12]

Despite the existing body of knowledge linking cTnI levels with adverse outcomes in STEMI, several methodological limitations exist in the available literature. A significant proportion of studies are either retrospective or involve single-center designs with relatively small sample sizes, limiting their generalizability. Moreover, variability in assay types and timing of cTnI measurements further complicates interpretation and standardization of results.[13] Importantly, robust prospective studies analyzing the correlation between admission cTnI and in-hospital adverse events in the Bangladeshi population are notably lacking. Most local studies are limited to single-center experiences without adequate adjustment for confounding factors such as age, sex, comorbidities, and the time elapsed from symptom onset to hospital presentation.[14]

Given these gaps, there is a compelling need for well-designed studies evaluating the prognostic value of admission cTnI levels for predicting adverse in-hospital outcomes in Bangladeshi STEMI patients. Addressing these gaps could significantly improve clinical decision-making, help allocate scarce healthcare resources efficiently, and ultimately enhance patient survival and recovery in resource-limited settings.

Methods

This hospital-based cross-sectional observational study was conducted at the Department of Medicine and Cardiology, Sir Salimullah Medical College and Mitford Hospital, Dhaka, Bangladesh, over a 6-month from June to December 2018. The

study enrolled 100 patients diagnosed with STEMI based on the clinical assessment and electrocardiographic evidence, who fulfilled the inclusion and exclusion criteria. Inclusion criteria were patients aged between 30 and 70 years with confirmed STEMI, whereas exclusion criteria included patients with non-STEMI, previous myocardial infarction, history of coronary artery bypass grafting or PCI, valvular heart disease, cardiomyopathy, chronic pulmonary diseases, chronic kidney disease, malignancy, or terminal illness. A purposive sampling technique was used due to time and cost constraints. After obtaining informed written consent, detailed medical history, physical examination, and assessment of cardiovascular risk factors such as age, sex, diabetes mellitus, hypertension, smoking status, and family history of coronary artery disease were recorded. Venous blood samples were collected at admission to measure serum cTnI levels using an immunometric assay (Vitros ECI system, Johnson and Johnson, USA) following standard laboratory protocols, with cutoff values categorized as <0.034 ng/mL indicating no infarction, 0.034–0.12 ng/mL as high risk, and ≥0.12 ng/mL confirming infarction. Additional laboratory investigations included fasting blood glucose, serum creatinine, lipid profile, and other routine tests as required. Echocardiography and chest X-rays were performed as part of standard evaluation. Clinical outcomes assessed during hospitalization included the development of arrhythmias, acute heart failure, cardiogenic shock, duration of hospital stay, and in-hospital mortality. Data were collected using a structured case record form and checked for completeness and consistency. All data were entered into Statistical Package for the Social Sciences version 20 for analysis. Quantitative variables were presented as mean and standard deviation, while categorical variables were expressed as frequencies and percentages. Associations between serum troponin I levels and in-hospital outcomes were analyzed using Chi-square tests for categorical data and Student's *t*-test for continuous variables, with $P < 0.05$ considered statistically significant. Ethical clearance was obtained from the Institutional Review Board of Sir Salimullah Medical College and Mitford Hospital, ensuring patient confidentiality and adherence to ethical research conduct throughout the study.

Results

The mean age of patients was 52.5 years with a standard deviation of 13.1 years, ranging from 29 to 68 years. Majority of the patients were within the 41–50 years of age group, comprising 34% of the total sample. This was followed by 27% in the 51–60 years of age group and 21% in the 31–40 years of age group. Patients aged over 60 years accounted for 13% of the sample, while only 5% were aged 30 years or younger [Table 1].

Out of the total 100 patients included in this study, 59% were male and 41% were female, indicating a male predominance among STEMI cases [Figure 1].

Among the 100 patients included, the largest occupational group was businesspersons, accounting for 29% of the sample. This was followed closely by day laborers at 26% and homemakers at 25%, indicating a substantial representation of lower income and domestic worker groups. Patients employed in service occupations constituted 20% of the study population [Table 2].

The majority of patients, 60%, had troponin-I levels between 0.034 and 0.12 ng/mL, whereas 27% of patients had levels below 0.034 ng/mL, suggesting absence or minimal elevation of cardiac injury

Table 1: Distribution of the study patients by age (*n*=100)

Age (years)	Number of patients (*n*)	Percentage
≤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 (min–max)	29–68	

SD: Standard deviation

markers in these individuals. A smaller proportion, 13%, had troponin-I levels equal to or >0.12 ng/mL, indicating confirmed myocardial infarction. The mean troponin-I level among the study population was 0.10 ng/mL with a standard deviation of 0.05 [Table 3].

Among patients with troponin-I levels below 0.034 ng/mL, none developed arrhythmias or heart failure, and only one patient (3.7%) developed cardiogenic shock. In contrast, patients with troponin-I levels between 0.034 and 0.12 ng/mL had higher rates of complications, with 33.3% developing arrhythmias, 11.6% experiencing cardiogenic shock, and 23.3% developing heart failure. The highest complication rates were observed in patients with troponin-I levels equal to or above 0.12 ng/mL, where 46.1% developed arrhythmias, 23.0% developed cardiogenic shock, and 30.7% developed heart failure. Regarding hospital stay duration, all patients with troponin-I levels below 0.034 ng/mL were discharged within 3–5 days. Among those with troponin-I levels between 0.034 and 0.12 ng/mL, 28.3% had a hospital stay of 3–5 days, 65.0% stayed for 5–7 days, and 6.7% stayed longer than 7 days. For patients with troponin-I levels equal to or >0.12 ng/mL, none were discharged within 3–5 days, 23.0% stayed for 5–7 days, and a significant majority (76.9%) had hospital stays exceeding 7 days. Notably, no complications were observed in 96.2% of patients with troponin-I levels below 0.034 ng/mL, whereas only 31.6% of patients with troponin-I levels between 0.034 and 0.12 ng/mL had no complications, and none of the patients with troponin-I levels equal to or above 0.12 ng/mL were free of complications [Table 4].

The graph shows that patients with troponin-I levels below 0.034 ng/mL did not develop arrhythmia or heart failure, while a very small number experienced cardiogenic shock. In contrast, patients with troponin-I levels between 0.034 and 0.12 ng/mL had notably higher incidences of complications, with 35 developing arrhythmias, 15 developing cardiogenic shock, and 25 developing heart failure. The highest complication rates were observed among patients with troponin-I levels equal to or >0.12 ng/mL, where 50 developed arrhythmias, 25 experienced cardiogenic shock, and 33 developed heart failure [Figure 2].

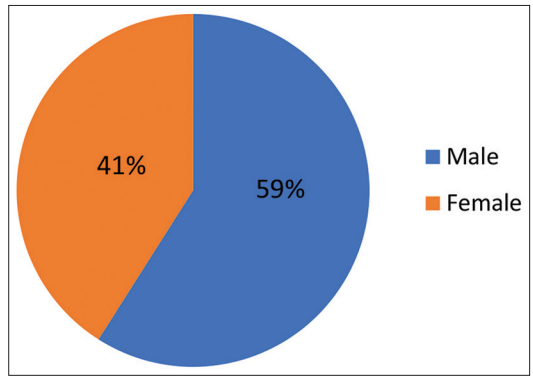

Figure 1: Pie chart showing sex distribution of the study patients

Table 2: Occupation status of the study population (*n*=100)

Occupation	Number of patients (*n*)	Percentage
Business	29	29.0
Service	20	20.0
Day labor	26	26.0
Homemaker	25	25.0

Table 3: Evaluation of troponin-I (ng/mL) among STEMI patient (*n*=100)

Troponin-I (ng/mL)	Number of patients (*n*)	Percentage
<0.034	27	27.0
0.034–0.12	60	60.0
≥0.12	13	13.0
Mean±SD	0.10±0.05	

STEMI: ST-segment elevation myocardial infarction, SD: Standard deviation

Table 5 presents the statistical correlation between cTnI levels, and adverse in-hospital outcomes among STEMI patients. For arrhythmias, there was a statistically significant upward trend in incidence with increasing troponin-I levels, with no cases observed in patients with levels

Table 4: In-hospital outcomes of the study population (*n*=100)

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

Table 5: Correlation between cardiac troponin-i levels and adverse in-hospital outcomes in STEMI patients (*n*=100)

Outcome	Troponin-I group (ng/mL) (%)	Incidence (%)	χ^2 (Chi-square)	*P*-value	Observed trend
Arrhythmia	<0.034: 0 0.034–0.12: 33.3 ≥0.12: 46.1	Proportional increase with Troponin-I level	21.6	<0.001	Statistically significant upward trend
Cardiogenic shock	<0.034: 3.7 0.034–0.12: 11.6 ≥0.12: 23.0	Progressive rise across increasing Troponin-I categories	8.94	0.011	Moderate positive association
Heart failure	<0.034: 0 0.034–0.12: 23.3 ≥0.12: 30.7	Gradual increase with elevated Troponin-I concentrations	15.7	<0.001	Strong positive association
Hospital stay >5d	<0.034: 0 0.034–0.12: 71.7 ≥0.12: 100	Markedly higher rates observed with higher troponin-I levels	61.3	<0.001	Highly significant positive association

STEMI: ST-segment elevation myocardial infarction

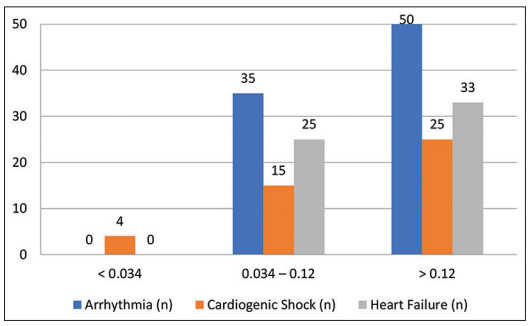

Figure 2: Correlation between cardiac Troponin-I level with adverse in-hospital outcome in patients with ST-segment elevation myocardial infarction (*n*=100)

below 0.034 ng/mL, compared to 33.3% in the 0.034–0.12 ng/mL group and 46.1% in patients with levels equal to or >0.12 ng/mL (χ^2 = 21.6, $P < 0.001$). A similar pattern was observed for cardiogenic shock, where incidence increased progressively from 3.7% in the lowest troponin-I group to 11.6% in the middle group and 23.0% in the highest troponin-I group, showing a moderate positive association (χ^2 = 8.94, P = 0.011). For heart failure, there was a strong positive association with troponin-I levels, with no cases reported in the lowest group, while 23.3% of patients in the 0.034–0.12 ng/mL group and 30.7% of patients

with troponin-I levels ≥0.12 ng/mL developed heart failure (χ^2 = 15.7, P < 0.001). In addition, the duration of hospital stay showed a highly significant positive association with troponin-I levels, as none of the patients in the <0.034 ng/mL group stayed longer than 5 days, compared to 71.7% in the 0.034–0.12 ng/mL group and all patients (100%) in the ≥0.12 ng/mL group requiring hospital stays exceeding 5 days (χ^2 = 61.3, P < 0.001) [Table 5].

Discussion

This study evaluated the correlation between admission cTnI levels and adverse in-hospital outcomes in STEMI patients in a tertiary care setting in Bangladesh. The study found that 59% of patients were male and 41% were female, with a mean age of 52.5 ± 13.1 years, and the highest proportion in the 41–50 years of age group. These findings align with Qamar *et al*., who reported a male predominance (80.8%) and the mean age of 55 years in the NORIN-STEMI registry in India, though the male percentage was slightly higher in their cohort.[15] Similarly, Bortnick *et al*. observed a male predominance of 67% with a median age of 59 years in a low-income urban US STEMI cohort, supporting the global trend of higher STEMI incidence among middle-aged males.[16] Regarding occupational status, businesspersons formed the largest group (29%), followed by day laborers (26%), homemakers (25%), and service holders (20%). Sadeghi *et al*. reported comparable occupational distributions in an Iranian STEMI cohort, where manual workers, business owners, and service employees comprised the majority, highlighting socioeconomic risk factors as major contributors to STEMI.[17] In terms of troponin-I level distribution, this study found that 27% had levels <0.034 ng/mL, 60% had levels between 0.034 and 0.12 ng/mL, and 13% had levels ≥0.12 ng/mL, with a mean troponin-I of 0.10 ± 0.05 ng/mL. Ahmad *et al*. reported much higher mean troponin-I levels (~9.54 ng/mL) in an Indian STEMI cohort, suggesting possible differences in assay sensitivity, presentation timing, or infarct size between populations.[18] Wanamaker *et al*. also found that higher troponin levels were significantly associated with worse in-hospital outcomes, which is consistent with the current study findings.[19] Importantly, this study observed that arrhythmia occurred in 0% of patients with troponin-I <0.034 ng/mL, 33.3% with 0.034–0.12 ng/mL, and 46.1% with ≥0.12 ng/mL (P < 0.001), showing a significant upward trend. Khullar *et al*. similarly reported that peak troponin-T levels were predictive of arrhythmias and mortality in STEMI patients.[20] Cardiogenic shock incidence rose from 3.7% to 23% across troponin categories (P = 0.011), indicating a moderate positive association, whereas Coelho-Lima *et al*. also demonstrated significant predictive value of admission hs-troponin for shock and mortality.[21] For heart failure, incidence increased from 0% (<0.034 ng/mL) to 30.7% (≥0.12 ng/mL) with a strong positive correlation (P < 0.001). Similar results were observed by Wanamaker *et al*., where higher troponin levels predicted in-hospital heart failure.[19] In addition, this study found that hospital stay >5 days was required by 0% with <0.034 ng/mL, 71.7% with 0.034–0.12 ng/mL, and 100% with ≥0.12 ng/mL (P < 0.001), highlighting a highly significant positive association between elevated troponin-I and prolonged hospitalization. Overall, all associations between troponin-I levels and in-hospital adverse outcomes were statistically significant, except for cardiogenic shock, which showed a moderate significance (P = 0.011). These findings are consistent with existing literature showing that higher troponin levels at admission are robust predictors of in-hospital complications and mortality.[19-21]

Limitations of the study

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

Conclusion

This study demonstrated that higher admission cTnI levels are significantly associated with adverse in-hospital outcomes among STEMI patients, including increased rates of arrhythmia,

cardiogenic shock, heart failure, and prolonged hospital stay. These findings highlight the utility of troponin-I not only as a diagnostic marker but also as an effective prognostic tool for early risk stratification in STEMI management. Implementing admission troponin-I-based risk assessment in clinical practice can assist physicians in identifying high-risk patients promptly and allocating appropriate intensive care resources, ultimately improving patient outcomes in resource-limited settings like Bangladesh. Further large-scale prospective studies are recommended to validate these findings and establish standardized troponin-I cutoffs for prognostic use in the regional population.

Funding

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Conflict of Interest

None declared.

Ethical Approval

The study was approved by the Institutional Ethics Committee.

References

1. Zeidan RK, Farah R. Myocardial infarctions in developing countries. In: Handbook of Medical and Health Sciences in Developing Countries. Cham: Springer; 2023. p. 1-30. Available from: https://link.springer.com/rwe/10.1007/978-3-030-74786-2_5-1 [Last accessed on 2025 Jul 09].
2. Chowdhury IZ, Amin MN, Chowdhury MZ, Rahman SM, Ahmed M, Cader FA. Pre hospital delay and its associated factors in acute myocardial infarction in a developing country. PLoS One 2021;16:e0259979.
3. Akhtar Z, Aleem MA, Ghosh PK, Islam AK, Chowdhury F, MacIntyre CR, *et al*. In-hospital and 30-day major adverse cardiac events in patients referred for ST-segment elevation myocardial infarction in Dhaka, Bangladesh. BMC Cardiovasc Disord 2021;21:85.
4. Kim DY, Wala Z, Islam S, Islam R, Ahn M. Clinical characteristics and outcomes of ST-segment elevation myocardial infarction in a low income setting in rural Bangladesh. Int J Cardiol Heart Vasc 2019;23:100376.
5. Ferraro S, Biganzoli E, Marano G, Santagostino M, Boracchi P, Panteghini M, *et al*. New insights in the pathophysiology of acute myocardial infarction detectable by a contemporary troponin assay. Clin Biochem 2013;46:999-1006.
6. Fathil MF, Arshad MK, Gopinath SC, Hashim U, Adzhri R, Ayub RM, *et al*. Diagnostics on acute myocardial infarction: Cardiac troponin biomarkers. Biosens Bioelectron 2015;70:209-20.
7. Laugaudin G, Kuster N, Petiton A, Leclercq F, Gervasoni R, Macia JC, *et al*. Kinetics of high-sensitivity cardiac troponin T and I differ in patients with ST-segment elevation myocardial infarction treated by primary coronary intervention. Eur Heart J Acute Cardiovasc Care 2016;5:354-63.
8. Sheikh KU, Sarfaraz A, Sarfaraz S, Bano T, Ikram R. Assessment of the co-relation between 1st set of Troponin I, age, duration of chest pain and LVEF in patients presenting with first stemi and treatment protocol followed. Pak J Pharm Sci 2020;33 6 Suppl:2793-9.
9. Alnemer KA. In-hospital mortality in patients with acute myocardial infarction: A literature overview. Cureus 2024;16:e66729.
10. Gall E, Pezel T, Lattuca B, Hamzi K, Puymirat E, Piliero N, *et al*. Profile of patients hospitalized in intensive cardiac care units in France: ADDICT-ICCU registry. Arch Cardiovasc Dis 2024;117:195-203.
11. Koolwal P, Mangal K. Cardiogenic shock in resource-limited settings. J Nepal Soc Crit Care Med 2024;2:9-13.
12. Akhter N, Khan AR, Farooq H, Maqbool S, Mahmood N, Samad A. Total neutrophil count and neutrophil/lymphocyte ratio in predicting in-hospital mortality and complications after ST segment elevation myocardial infarction. Ann Punjab Med Coll 2023;17:195-203.
13. Shrestha B, Shrestha DB, Sedhai YR, Shtembari J, Oli PR, Shikhrakar S, *et al*. Differences in treatment and outcomes among patients with ST-segment elevation myocardial infarction with and without standard modifiable risk factors: A systematic review and meta-analysis. Ann Med Surg (Lond) 2023;85:2916-23.
14. Rahman MM, Joty SM, Jeshan T, Ghosh TP, Ahmed MU, Hossain S, *et al*. Safety and efficacy of tenecteplase among patients with ST elevated myocardial infarction - experience in a tertiary care hospital in Bangladesh. Cardiol Cardiovasc Med 2023;7:343-8.
15. Qamar A, Bhatia K, Arora S, Hendrickson M, Gupta P, Fatima A, *et al*. Clinical profiles, outcomes, and sex differences of patients with STEMI. JACC Asia 2023;3:431-42.
16. Bortnick AE, Shahid M, Shitole SG, Park M, Broder A, Rodriguez CJ, *et al*. Outcomes of ST-elevation myocardial infarction by age and sex in a low-income

urban community: The Montefiore STEMI registry. Clin Cardiol 2020;43:1100-9.

17. Sadeghi M, Saririyan M, Kermani-Alghoraishi M, Ghahramani R, Jamalian M, Roohafza H, *et al*. Evaluation of factors influencing return to work in STEMI patients: A case-control study. Medicine (Baltimore) 2025;104:e41839.
18. Ahmad M, Yadaw B, Sharma N, Varshney A, Sharma L, Singh R, *et al*. Cardiac troponin I level in STEMI and clinical correlation with left ventricular dysfunction in Indian population. J Cardiovasc Dis Diagn 2013;1:116.
19. Wanamaker BL, Seth MM, Sukul D, Dixon SR, Bhatt DL, Madder RD, *et al*. Relationship between troponin on presentation and in-hospital mortality in patients with ST-segment-elevation myocardial infarction undergoing primary percutaneous coronary intervention. J Am Heart Assoc 2019;8:e013551.
20. Khullar N, Buckley AJ, O'Connor C, Ibrahim A, Ibrahim A, Ahern C, *et al*. Peak troponin T in STEMI: A predictor of all-cause mortality and left ventricular function. Open Heart 2022;9:e001863.
21. Coelho-Lima J, Georgiopoulos G, Ahmed J, Adil SE, Gaskin D, Bakogiannis C, *et al*. Prognostic value of admission high-sensitivity troponin in patients with ST-elevation myocardial infarction. Heart 2021;107:1881-8.