# Prognostic Value of Cardiac Troponin I for In-hospital Recovery in Patients with ST-Elevation Myocardial Infarction

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**Abstract:**

**Introduction:**
ST-elevation myocardial infarction (STEMI) remains a common cause of cardiovascular mortality, with accurate risk stratification for the appropriate management of the patient. Cardiac troponin I (cTnI) has been identified as a potential prognostic biomarker, as well as in its diagnostic role.

**Objective:**
This study aimed to evaluate the prognostic significance of cardiac troponin I level for in-hospital recovery and complications in patients with STEMI.

**Methods:**
A cross-sectional study was conducted on 100 STEMI patients aged 30-70 years. The patients were categorized into three groups based on serum troponin I concentration: <0.034 ng/mL (low risk), 0.034-0.12 ng/mL (intermediate risk), and ≥0.12 ng/mL (high risk). In-hospital mortality, complications (arrhythmia, heart failure, cardiogenic shock), and length of stay in the hospital were the primary outcomes of interest. Statistical analysis was performed on SPSS version 26 using chi-square tests and binary logistic regression.

**Results:**
The study population had a mean age of 52.5±13.1 years with 59% male preponderance. There was a clear-cut dose-response relation observed between troponin levels and adverse outcomes. In-hospital mortality increased step-wise in low, intermediate, and high-risk troponin groups (0%, 3.4%, 38.5% respectively; p<0.001). Cumulative complication rates also followed a similar pattern (3.7%, 68.4%, 100% respectively; p<0.001), with specific complications like arrhythmias (0%, 33.3%, 46.1%), heart failure (0%, 23.3%, 30.7%), and cardiogenic shock (1.3%, 11.6%, 23.0%). Hospital stay was also significantly related to the troponin levels, with prolonged hospitalization (>7 days) in 0%, 6.7%, and 76.9% in respective groups (p<0.001). Multivariate analysis also endorsed troponin ≥0.12 ng/mL as an independent predictor of mortality with a 49-fold increased risk (OR=49.8, 95% CI: 5.3-463.2, p<0.001).

**Conclusion:**
The study highlights the optimal prognostic value of cardiac troponin I levels in the prediction of in-hospital outcomes among STEMI patients. The proposed risk stratification system can be applied to inform decisions for clinical management, deployment of resources, and patient care approach in acute cardiac care units.

**Keywords:** Cardiac Troponin, ST-Elevation Myocardial Infarction (STEMI), Prognostic Biomarker.

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## Introduction:

ST-elevation myocardial infarction (STEMI) is a critical cardiovascular emergency characterized by complete coronary artery occlusion, resulting in transmural myocardial necrosis.[1] Despite advances in reperfusion therapy, STEMI remains associated with significant morbidity and mortality, making early risk stratification essential for optimizing clinical outcomes.[2] Identifying high-risk patients likely to experience adverse

events during hospitalization is a key challenge in modern cardiology. Cardiac troponin I (cTnI) has emerged as the gold standard biomarker for myocardial injury, offering greater diagnostic accuracy than traditional markers like creatine kinase-MB.[3] Its prognostic utility extends beyond diagnosis, with elevated levels correlating with infarct size, left ventricular dysfunction, and worse clinical outcomes.[4] Recent studies, such as by Docherty et al, have shown that higher admission troponin levels in STEMI patients undergoing primary PCI are associated with increased 30-day and 1-year mortality, independent of baseline risk.[5,6] High-sensitivity cardiac troponin I (hs-cTnI) levels, particularly peak values within 8 hours, are also independent predictors of mortality, reinfarction, and in-hospital heart failure.[7] However, its predictive power may be time-sensitive; one study noted that troponin's association with mortality and heart failure was significant during the initial 30 days but diminished over a 1-year follow-up.[8] Despite its proven role, standardized thresholds for prognostic use in STEMI are lacking. Furthermore, the association between cTnI levels and hospital length of stayis a key indicator of healthcare resource utilizationrequires further exploration. This study aimed to evaluate the prognostic value of admission cTnI levels in predicting in-hospital recovery and complications in STEMI patients. By establishing clear risk categories based on troponin levels, the research intends to support clinical decision-making and improve resource allocation in acute cardiac care.

**Methods:**

This cross-sectional study was conducted from June 12 to December 11, 2018, in the Medicine and Cardiology departments of Sir Salimullah Medical College Mitford Hospital. It included patients aged 30–70 years admitted with ST-elevation myocardial infarction (STEMI), confirmed by ECG. Exclusion criteria were non-STEMI, prior cardiac events, valvular or cardiomyopathic diseases, chronic kidney/pulmonary disease, malignancies, and age outside the specified range. A calculated sample size of 298 (based on disease prevalence and hospital case load) was reduced to 100 due to time and cost constraints. Purposive sampling was employed. Data were collected using structured case record forms and analyzed with SPSS v20. Categorical variables were assessed using chi-square tests; continuous variables with unpaired Student's t-tests. Variables included demographics, risk factors (diabetes, hypertension, smoking, obesity), clinical features, complications (arrhythmia, heart failure, cardiogenic shock), and hospital stay duration. Serum Troponin-I was measured using immunometric assay and categorized for risk stratification: <0.034 ng/mL (low risk), 0.034–0.12 ng/mL (high risk), and ≥0.12 ng/mL (indicative of acute MI). Blood samples were collected aseptically and analyzed in hospital and external accredited labs. Ethical approval was obtained from the institutional review board. Informed consent was taken from all participants. Data confidentiality was maintained, and all procedures followed standard hospital protocols with minimal risk to participants.

**Results:**

The mean age of patients was 52.5±13.1 years, with 61% aged 41–60 years. Males were more affected (59%). The most common occupations were business (29%) and day labor (26%). Hypertension (63%) was the most prevalent risk factor, followed by smoking (47%) and diabetes (42%) (Table-I).

**Table-I: Distribution of the study population by demography (N=100)**

| Demographics | no. (%) |
|---|---|
| **Age (in years)** | |
| ≤30 | 5(5) |
| 31-40 | 21(21) |
| 41-50 | 34(34) |
| 51-60 | 27(27) |
| >60 | 13(13) |
| Mean±SD | 52.5±13.1 |
| **Sex** | |
| Male | 59(59) |
| Female | 41(41) |
| **Occupation** | |
| Business | 29(29) |
| Service | 20(20) |
| Day Labour | 26(26) |
| Housewife | 25(25) |
| **Comorbidity** | |
| Hypertension | 63(63) |
| Smoker | 47(47) |
| Diabetes Mellitus | 42(42) |
| Dyslipidemia | 22(22) |

Troponin I levels were strongly associated with in-hospital complications. No major complications occurred in the low troponin group (<0.034 ng/mL), while the high group (≥0.12 ng/mL) had the highest rates of arrhythmia (46.1%), heart failure (30.7%), and cardiogenic shock (23.0%). Length of hospital stay increased with troponin levels. All patients in the low-risk group had short stays (3–5 days). Most in the intermediate group stayed 5–7 days, while 76.9% of the high troponin group required hospitalizations >7 days. Adverse outcomes increased with higher troponin levels. Mortality rose from 0% (low group) to 38.5% (high group), while recovery declined from 100% to 61.5% (Table-II).

**Table-II: Prognostic value of Troponin I in STEMI regarding in-hospital complications, duration of hospital stay and outcome (N=100)**

| Complication | <0.034 ng/ml (n=27) no. (%) | 0.034–0.12 ng/ml (n=60) no. (%) | ≥0.12 ng/ml (n=13) no. (%) | p-value |
|---|---|---|---|---|
| Arrhythmia | 0(0) | 20(33.3) | 6(46.1) | 0.021 |
| Cardiogenic shock | 1(3.7) | 7(11.6) | 3(23.0) | 0.048 |
| Heart failure | 0(0) | 14(23.3) | 4(30.7) | 0.014 |
| Total complications | 1(3.7) | 41(68.4) | 13(100) | <0.001 |
| No complication | 26(96.2) | 19(31.6) | 0(0) | <0.001 |
| **Hospital stay duration** | | | | |
| 3–5 days | 27(100) | 17(28.3) | 0(0) | |
| 5–7 days | 0(0) | 39(65.0) | 3(23.0) | <0.001 |
| >7 days | 0(0) | 4(6.7) | 10(76.9) | |
| **Outcome** | | | | |
| Recovered | 27(100.0) | 58(96.6) | 8(61.5) | <0.001 |
| Death | 0(0) | 2(3.4) | 5(38.5) | <0.001 |

Reference category: Troponin <0.034 ng/Ml

Logistic regression confirmed troponin ≥0.12 ng/mL as a strong independent predictor of in-hospital mortality (OR=49.8, 95% CI: 5.3–463.2, p<0.001), while intermediate levels were not statistically significant.

**Table-III: Binary logistic regression – effect of Troponin I on in-hospital mortality (N=100)**

| Variable | B (β coefficient) | SE | Wald | OR (Exp(B)) | 95% CI for OR | p-value |
|---|---|---|---|---|---|---|
| Troponin 0.034–0.12 | 1.25 | 1.01 | 1.53 | 3.49 | 0.48 – 25.6 | 0.216 |
| Troponin ≥ 0.12 | 3.91 | 1.15 | 11.56 | 49.8 | 5.3 – 463.2 | <0.001 |
| Constant | -6.52 | 1.54 | 17.98 | 0.001 | - | <0.001 |

Reference category: Troponin <0.034 ng/Ml

**Discussion:**

This study demonstrates a strong correlation between cardiac troponin I (cTnI) levels and in-hospital outcomes in STEMI patients, confirming its role as a powerful prognostic marker. A dose-response relationship was evident, with cTnI ≥0.12 ng/mL associated with a 49-fold higher mortality risk compared to <0.034 ng/mL,

consistent with Sandoval et al.[9] Stratifying patients into three groups by troponin level effectively predicted outcomes, aligning with Hall et al., who showed cTnI after PCI independently predicted cardiac function.[10] Our findings extend this value to admission troponin levels, emphasizing its utility in early therapeutic decisions. Complication rates increased stepwise with troponin categories. The highest-risk group had universal complications, including arrhythmia (46.1%), heart failure (30.7%), and cardiogenic shock (23.0%), mirroring Jolly et al, who reported higher arrhythmia, arrest, and shock risks with rising troponin.[11] Conversely, 96.2% of patients with <0.034 ng/mL had no complications, suggesting this threshold can identify candidates for early discharge and reduced healthcare costs. Length of stay also correlated with troponin. All low-risk patients were discharged within 3–5 days, while 76.9% of the high group stayed >7 days. This highlights troponin's value for both clinical outcomes and resource planning. Other studies also support admission but not post-reperfusion cTnI as a predictor of mortality.[12] The mortality gradient in our study (0%, 3.4%, and 38.5% across groups) demonstrates excellent discriminatory ability, consistent with Khullar et al.[13] While Cediel et al suggested newer assays offer limited prognostic value,[14] our data confirm significant predictive strength even with conventional assays. Binary logistic regression confirmed troponin ≥ 0.12 ng/mL as an independent predictor of death (OR=49.8), providing a quantitative tool for risk stratification. Elevated cTnI also predicted heart failure development, supporting prior studies of troponin's prognostic value in acute heart failure.[15] The consistent trend across all outcomes mortality, complications, and length of stay ($p<0.001$) strengthens its clinical utility. Similar to Wanamaker et al, we found elevated troponin independently predicted in-hospital mortality.[16] Clinically, troponin stratification can guide management: Our findings have important clinical implications for STEMI management. Low-risk patients (troponin <0.034 ng/mL) may be eligible for early discharge strategies and facilitation of the decrease in health costs, while high-risk patients (troponin ≥0.12 ng/mL) need to undergo intensive surveillance and more intense therapeutic strategies. Intermediate-risk patients (0.034-0.12 ng/mL) require close monitoring and individualized management.

**Limitations:**

This study was limited by its single-center recruitment and relatively small sample size (n=100), which can affect generalizability to other populations. Cross-sectional study design does not permit analysis of longer-term outcomes after hospital admission, and the application of purposive sampling can create selection bias.

**Conclusion:**

In this study, cardiac troponin I is set squarely as an excellent prognostic biomarker in STEMI patients with clear dose-response relations between troponin concentrations and adverse outcomes. The proposed three-level risk stratification confidently predicts mortality, complications, and hospital stay and provides the clinician with a straight forward measure of employability for early risk assessment and decision-making in acute coronary care. Future studies would need to validate these troponin-based risk levels in larger, multi-center cohorts with longer follow-up to establish long-term prognostic value. Investigation of troponin kinetics and serial sampling may provide additional prognostic data. Creation of combined risk prediction models that include troponin level along with clinical covariates and other biomarkers might enhance prognostic accuracy and clinical applicability.

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