DOI: 10.4274/forbes.galenos.2025.27880
Forbes J Med 2025;6(2):165-71

Original Article / Orijinal Araştırma

Assessing the Association Between Troponin I Changes Below the Diagnostic Cut-off with Mortality and Major Cardiac Events

Tanı Eşiğinin Altındaki Troponin I Değişimlerinin Mortalite ve Majör Kardiyak Olaylarla İlişkisinin Değerlendirilmesi

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Cite as: Torun A, Gönüllü H, Sağlam C, Parlak İ. Assessing the association between troponin I changes below the diagnostic cut-off with mortality and major cardiac events. Forbes Med J. 2025;6(2):165-71

ABSTRACT

Objective: In this study, we aimed to investigate the effect of variations in cardiac troponin I (cTnI) (delta troponin) levels-within the normal reference range-on 30-day mortality and non-fatal major adverse cardiac events (MACE) in patients who were monitored in the emergency department with a preliminary diagnosis of acute coronary syndrome.

Methods: After obtaining ethics committee approval, this retrospective study included patients who presented to the emergency department with chest pain and/or related symptoms, underwent serial cTnI measurements, and had measured cTnI levels ≤0.05 ng/mL. Patients were divided into two groups based on the presence or absence of delta troponin. The association between delta troponin and 30-day mortality and non-fatal MACE was examined.

Results: The group with positive delta cTnI had a significantly higher risk of non-fatal MACE compared to the group without delta cTnI (p<0.001). The presence of cardiac-type chest pain, ischemic ST-segment changes on electrocardiographic, and a history of hypertension, diabetes, hyperlipidemia, and coronary artery disease were all significantly associated with an increased risk of 30-day mortality and non-fatal MACE. Similarly, advancing patient age was significantly associated with these outcomes.

Conclusion: Our findings demonstrate that changes in delta troponin levels below the 99th percentile have a significant impact on the risk of 30-day mortality and non-fatal MACE.

Keywords: 99th percentile, delta troponin, chest pain, mortality, major adverse cardiac event

ÖZ

Amaç: Bu çalışmada, acil serviste akut koroner sendrom ön tanısıyla izlenen hastalarda, normal referans aralığı içindeki kardiyak troponin I (cTnI) düzeylerindeki değişimlerin (delta troponin) 30 günlük mortalite ve ölümcül olmayan majör kardiyak olaylar (MACE) üzerindeki etkisini araştırmayı amaçladık.

Yöntem: Bu çalışmaya, etik kurul onayı alındıktan sonra göğüs ağrısı ve/veya ilişkili semptomlar ile acil servise başvuran, seri cTnI takibi yapılan ve ölçülen cTnI ≤0,05 ng/mL olan hasta grubu geriye dönük olarak alındı. Hastalar delta troponin olan ve olmayan şeklinde iki gruba ayrıldı ve 30 günlük mortalite ve mortalite dışı MACE ile ilişkili durumları incelendi.

Bulgular: Delta cTnI pozitif olan grupta olmayan gruba göre mortalite dışı MACE riski daha yüksek bulundu (p<0,001). Kardiyak tipte göğüs ağrısı ve elektrokardiyogramda iskemik ST segment değişikliğinin olması, hasta özgeçmişinde hipertansiyon, diabet, hiperlipidemi ve koroner arter hastalığı öyküsünün olması, 30 günlük mortalite ve mortalite dışı MACE riskini anlamlı olarak artırdı. Benzer anlamlılık hasta yaşı arttıkça da görüldü.

Received/Geliş: 15.05.2025
Accepted/Kabul: 02.07.2025
Yayınlanma Tarihi/ Publication Date: 06.08.2025

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Sonuç: Çalışmamız bulguları, 99 persantil altındaki delta troponin değişiminin 30 günlük mortalite ve mortalite dışı MACE riski üzerinde etkisinin anlamlı olduğunu gösterdi.

Anahtar Kelimeler: 99 persantil, delta troponin, göğüs ağrısı, mortalite, majör kardiyak olay

INTRODUCTION

Chest pain is one of the most common complaints presented to the emergency department. In the majority of cases, the underlying cause is benign, non-cardiac in nature, and does not require hospitalization. However, a smaller yet potentially life-threatening subset of these patients is diagnosed with acute coronary syndromes (ACS).[1,2] According to the literature, approximately 2% of ACS patients are mistakenly discharged from the emergency department, and this group demonstrates increased 30-day morbidity and mortality rates.[3] This poses a significant concern for both emergency physicians and cardiologists, complicating the overall management of patients presenting with chest pain.[1]

One of the key challenges lies in the subjective nature of chest discomfort, which may not be clearly articulated by certain patient populations, including women, the elderly, individuals with diabetes, and those with mental health conditions. In such groups, ACS may present with atypical or equivalent symptoms such as dyspnea, fatigue, epigastric pain, dizziness or nausea and vomiting, rather than classic chest pain.[1,2]

In ST-elevation myocardial infarction (STEMI), which accounts for a small proportion of ACS, the diagnosis can be made rapidly through electrocardiographic (ECG) evaluation. The identification of non-STEMI (non-STEMI) cases-characterized by ischemic ECG changes and elevated cardiac enzymes-is relatively straightforward. However, the primary challenge lies in detecting and managing low-risk ACS cases in which cardiac enzyme levels are either within normal limits or show only minimal elevation.[2,4]

The diagnostic approach to ACS relies on a combination of clinical history, ECG, and cardiac biomarkers. While the interpretation of history and ECG findings has remained largely unchanged over the years, cardiac biomarkers have undergone frequent updates in recent years.[2,4] Among cardiac troponin T (cTnT) and troponin I (cTnI) are considered reliable markers for both the diagnosis and exclusion of ACS.[2,4-6] cTnI, a myocardial-specific marker of infarction, begins to rise within 2 to 4 hours following acute myocardial injury and typically peaks at around 24 hours.[7]

In current ACS guidelines when neither STEMI nor non-STEMI is suspected, the evaluation of chest pain is recommended to incorporate validated risk-stratification tools.[8,9] This approach is particularly important in emergency departments that rely on conventional cTn assays rather than high-sensitivity cardiac troponin (hs-cTn), as risk scores improve the identification of low-risk chest pain patients in these settings.[9] Notably, the History, ECG, Age, Risk Factors, and Troponin (HEART) score-encompassing HEART, its successor, the HEARTS[3] score, facilitates the early discharge of chest pain patients without necessitating exercise testing, advanced cardiac investigations, or imaging.[5] Moreover, these tools permit reliable estimation of 30-day major adverse cardiac event (MACE) risk-including non-fatal myocardial infarction, revascularization, and out-of-hospital cardiac arrest-thereby optimizing patient management in the emergency department.[4,9]

Delta troponin refers to the difference between two troponin values evaluated at different times during a patient's follow-up. In our study, we aimed to evaluate whether changes in troponin values, even when below the normal limits, could be clinically predictive. In this study, we aimed to evaluate the impact of an increase in delta troponin levels on 30-day mortality and non-fatal MACE among patients monitored in the emergency department with a provisional diagnosis of ACS and normal-range cTnI values.

METHODS

This retrospective cross-sectional study was approved by the Clinical Research Ethics Committee of University of Health Sciences Türkiye, İzmir Bozyaka Training and Research Hospital (decision number: 7, date: 04.07.2018). We included patients aged ≥18 years who presented to the emergency department of this tertiary center between January 1, 2017, and December 31, 2017, with chest pain or chest pain-related symptoms (dyspnea, fatigue, epigastric pain, dizziness, nausea/vomiting), and whose serial cTnI measurements remained within the normal range (≤0.05 ng/mL). Exclusion criteria comprised pregnancy; diagnoses of STEMI, non-STEMI, sepsis, acute or chronic renal failure, pulmonary thromboembolism, or severe anemia; and presentation due to trauma.

Demographic characteristics, presenting complaints, duration and character of chest pain, and ECG findings were extracted retrospectively from manually maintained patient files and the hospital's electronic health record system. Chest pain type and ECG abnormalities were classified according to the HEART risk score.

The first cTnI level measured upon arrival in the emergency department was defined as the baseline troponin.

A second blood sample for cTnI was drawn at the fourth hour. Patients whose baseline and fourth-hour cTnI values both remained below the assay cut-off yet differed from each other were designated the "delta+positive" group. Those whose cTnI levels remained unchanged and below the assay cut-off at four hours were classified as the "delta-negative" group.

All cTnI assays were performed on the Beckman Coulter Access 2 analyzer (Beckman Coulter, Canada, USA) using the manufacturer's original Access AccuTnI kit. To assess the association between changes in troponin values below the assay cut-off and 30-day outcomes-mortality and non-fatal MACE-patients were contacted by telephone and their electronic medical records were reviewed. Collected data were recorded on a standardized research form, and 30-day mortality and MACE information were obtained via telephone follow-up or hospital database review.

There is no previously established delta threshold reported in the literature for this context. Therefore, the 0.005 ng/mL cutoff for delta cTnI was determined empirically through receiver operating characteristic (ROC) curve analysis. Using Youden's index, we identified the optimal threshold that maximizes the sum of sensitivity and specificity for predicting both 30-day mortality and MACE. This value represents the point on the ROC curve where the trade-off between sensitivity and specificity is most favorable.

Statistical Analysis

Descriptive statistics were used to summarize the data. Continuous variables were presented as mean ± standard deviation (SD) when normally distributed, or median, with interquartile range when non-normally distributed. Categorical variables were expressed as counts and percentages.

Comparisons of delta troponin status, age groups, chest pain type, ECG findings, and sex were made with respect to 30-day mortality and non-fatal MACE using the chi-square test for categorical data.

All statistical analyses were conducted using IBM SPSS Statistics version 25 (SPSS Inc., Chicago, IL, USA). A two-tailed p value of <0.05 was considered statistically significant. ROC curve analysis was employed to evaluate the association between delta troponin and 30-day mortality or non-fatal MACE. ROC curves were generated to assess the discriminatory performance of troponin change for survival and non-fatal MACE. Optimal cut-off values, along with their 95% confidence intervals (CI) and areas under the curve (AUC), were determined using Youden's index and the DeLong method in MedCalc Statistical Software (trial version 15.8; MedCalc Software bvba, Ostend, Belgium; http://www.medcalc.org).

RESULTS

During the study period, troponin measurements were performed in 4,650 patients. Of these, 1,259 were excluded because at least one troponin value (baseline or 4-hour) exceeded 0.05 ng/mL, and 410 met predefined exclusion criteria. Consequently, 2,981 patients were included in the analysis: 43.7% (n=1,304) in the delta+positive group and 56.3% (n=1,677) in the delta-negative group.

There were statistically significant differences in both sex distribution and age groups between the delta+positive and delta-negative cohorts (Table 1). The prevalence of risk factors, symptom characteristics, ECG findings, and comorbidities is also detailed in Table 1. Overall, hypertension was the most common risk factor (50.6%). When comparing risk factors by sex, only coronary artery disease was significantly more frequent in men than in women (p=0.036); there were no significant sex differences for diabetes mellitus, hypertension, or hyperlipidemia (all p>0.05).

Baseline cTnI levels were 0.009±0.012 ng/mL (mean ± SD; range 0.000–0.050), and 4-hour cTnI levels were 0.014±0.015 ng/mL (range: 0.000-0.060). The mean delta troponin was 0.006±0.008 ng/mL (range: 0.000-0.050). Compared to the delta-negative group, the delta+positive group had significantly higher rates of non-fatal MACE (p<0.001) and mortality (p=0.013) (Table 2). A total of 112 patients (8.6%) in the delta+positive group and 100 patients (6%) in the delta-negative group required admission to the coronary intensive care unit.

Among patients presenting with cardiac-type chest pain, there was no statistically significant difference in 30-day mortality between the two groups (p=0.173), whereas non-fatal MACE rates differed significantly (p<0.001). A history of hypertension, diabetes mellitus, hyperlipidemia, or coronary artery disease was significantly associated with non-fatal MACE within 30 days (p<0.001), but none of these risk factors showed a significant relationship with mortality (all p>0.05).

ECG abnormalities were significantly associated with both non-fatal MACE (p<0.001) and mortality (p=0.003). Multivariable logistic regression analysis identified independent predictors of 30-day mortality and non-fatal MACE; the results are presented in Table 2.

In ROC curve analysis, a delta troponin threshold ≥0.005 ng/mL yielded a sensitivity of 63.4% and specificity of 37.7% for predicting non-fatal MACE (AUC: 0.635; 95% CI, 0.607-0.664). For predicting 30-day mortality, the same threshold demonstrated a sensitivity of 61.2% and specificity of 41.1% (AUC: 0.609; 95% CI, 0.528-0.690). The ROC curves for delta troponin versus non-fatal MACE and mortality are shown in Figure 1.

Table 1. Demographic and general clinical characteristics of the patients						
		Delta+group (%)		Delta-group (%)		p
Sex	Female	616 (47.2)		861 (51.3)		0.026
	Male	688 (52.8)		816 (48.7)		
Age groups	<45 y	186 (14.3)		365 (18.5)		<0.001
	45-65 y	481 (36.9)		711 (42.4)		
	>65 y	637 (48.8)		601 (35.8)		
Symptoms	CPRS	528 (40.5)		677 (40.4)		0.016
	NCCP	706 (54.1)		945 (56.4)		
	CCP	70 (5.4)		50 (3.3)		
ECG	Normal	927 (71.1)		1365 (81.4)		<0.001
	NSC	352 (27)		292 (17.4)		
	ST-segment depression	25 (1.9)		20 (1.2)		
Comorbidities	DM	Present	301 (23.1)	Present	337 (20.1)	0.051
		Absent	1003 (76.9)	Absent	1340 (79.9)	
	HT	Present	718 (55.1)	Present	790 (47.1)	<0.001
		Absent	586 (44.9)	Absent	887 (52.9)	
	HL	Present	465 (35.7)	Present	489 (29.2)	<0.001
		Absent	839 (64.3)	Absent	1188 (70.8)	
	CAD	Present	325 (25.9)	Present	293 (17.5)	<0.001
		Absent	929 (75.1)	Absent	1384 (82.5)	

CPRS: Chest pain–related symptoms, NCCP: Non-cardiac chest pain, CCP: Cardiac chest pain, NSC: Non-specific ECG changes, ECG: Electrocardiographic, DM: Diabetes mellitus, HT: Hypertension, HL: Hyperlipidemia, CAD: Coronary artery disease

Table 2. Factors affecting non-fatal major adverse cardiac events and mortality				
Variable	**Non-fatal MACE odds ratio (95% CI)**	**p**	**Mortality odds ratio (95% CI)**	**p**
Cardiac chest pain	7.27 (4.43-11.95)	<0.05		
ECG-non-specific changes	4.36 (3.38-5.63)	<0.05	4.36 (3.38-5.63)	<0.05
ST-segment depression	11.75 (2.96-46.64)	<0.05	32.21 (13.29-78.07)	<0.05
Age >65			12.34 (2.87-53.08)	<0.05
Hyperlipidemia	4.16 (2.92-5.91)	<0.05		
Absence of hypertension			2.72 (1.29-5.77)	<0.05
Absence of CAD	1.53 (1.10-2.14)	<0.05		
Presence of delta troponin	1.61 (0.89-2.91)	0.12	2.67 (2.09-3.43)	<0.05

CAD: Coronary artery disease, MACE: Major adverse cardiac events, CI: Confidence interval, ECG: Electrocardiographic

DISCUSSION

Studies on delta troponin have largely focused on hs-cTn assays, while data regarding delta changes in conventional troponin I (cTnI) levels remain limited in the literature. In our study, we found that even minor changes in cTnI values within the normal reference range were statistically associated with 30-day MACE. Among patients presenting to the emergency department with chest pain and monitored for cardiac biomarkers, temporal variations in cTnI-despite remaining below the diagnostic threshold-appeared to be associated with subsequent cardiac events. These findings suggest that even modest delta increases in cTnI may warrant closer clinical monitoring due to their potential prognostic relevance.

cTn became popular for ACS diagnosis in the early 1990s and are now considered fundamental laboratory parameters alongside clinical assessment and ECG.[10] The delta cTn value is defined as the difference between an

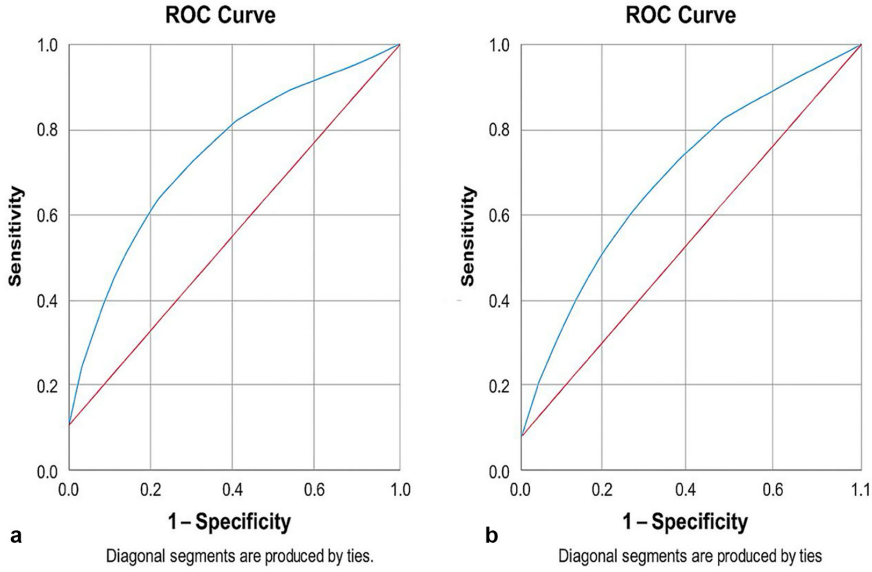

Figure 1. (a) Delta troponin-MACE ROC curve (b) delta troponin-mortality ROC curve

MACE: Major adverse cardiac events, ROC: Receiver operating characteristic

initial cTn concentration and a subsequent measurement; expressing this change as a percentage has been recommended.[11] High-sensitivity troponin I (hs-TnI) and its delta rate (delta hs-TnI) have recently been adopted for early ACS diagnosis with excellent performance. Studies have shown that a negative hs-TnI result safely facilitates discharge of chest pain, patients, whereas elevated hs-TnI values-irrespective of ACS-signal poorer prognosis and necessitate further evaluation.[12,13] Weir et al.[13] reported that delta hs-TnI changes of less than 20% can classify patients as low risk for ACS.

Because hs-TnI assays are not universally available, many emergency departments continue to rely on conventional cTnI measurements, which require longer monitoring intervals.[11,12] In our center, cTnI is expected to rise between 2 and 4 hours following myocardial injury. Accordingly, we compared baseline (0-hour) and 4-hour cTnI values and examined their relationship with 30-day mortality and non-fatal MACE.

In a prospective observational study, Suh et al.[14] evaluated the 0-to 1-hour delta hs-TnI alongside the HEART score to predict 30-day MACE. They designated patients as low risk if they had a modified HEART score of 0-3, symptom onset >3 hours before presentation, and hs-TnI <6 ng/L, and discharged them after a single hs-TnI measurement. Women with hs-TnI ≤14 ng/L and men with hs-TnI ≤22 ng/L had a follow-up hs-TnI obtained, and those with an hourly delta hs-TnI increase of <2.5 ng/L were deemed low risk and discharged. Conversely, patients exhibiting hourly increases >2.5 ng/L or elevated follow-up hs-TnI (>22 ng/L in men, >14 ng/L in women) were considered high risk.

Age-related increases in chest pain presentations and ACS incidence have also been reported.[15,16] Advanced age serves as a risk factor partly because other cardiovascular risk factors become more prevalent over time, increasing overall disease burden. In our cohort, delta+positive and delta-negative groups differed significantly by age and sex ($p=0.020$ and $p<0.001$, respectively); older age correlated with greater delta cTnI changes. Nevertheless, the rising incidence of ACS among individuals under 45-likely due to sedentary lifestyles and urbanization-should not be overlooked. In our study, male sex was independently associated with delta troponin elevation, irrespective of age.

James et al.[17] found that patients with mildly elevated cTnT levels (0.01-0.1 µg/L) faced a higher risk compared to those with completely normal values. Cullen et al.[11] examined cTnI changes at 2 and 6 hours post-presentation, reporting that delta changes exceeding the 99th percentile were highly indicative of ACS. Consistent with these findings, our delta+positive group experienced significantly higher non-fatal MACE rates ($p<0.05$).

Although hs-TnI measurement allows rapid risk stratification-particularly useful in busy emergency departments-our center's reliance on conventional cTnI necessitated longer observation periods. Given increasing patient volumes and operational challenges in Turkish

emergency departments, broader adoption of hs-TnI assays could enhance efficient and accurate patient management.

Several scoring systems support safe discharge of low-risk ACS patients by demonstrating <1% 30-day MACE risk; these incorporate hypertension, hyperlipidemia, diabetes mellitus, and coronary artery disease history among their variables.[18,19] The Thrombolysis in Myocardial Infarction score, developed by Antman et al.[18] in 2000, similarly employs these risk factors. We evaluated these risk factors, chest pain characteristics, and ECG findings across delta+positive and delta-negative groups. Our observations regarding chest pain character and ECG ischemic changes (ST-segment depression and other ischemic signs) aligned with existing literature. However, in contrast to previous studies, our findings revealed that the absence of a history of coronary artery disease was associated with a higher incidence of non-fatal MACE, while the absence of hypertension was linked to increased mortality. This discrepancy may be attributed to the exclusion of STEMI and non-STEMI patients from our study, as well as the likelihood that individuals with known coronary artery disease or hypertension are under regular cardiology follow-up and receive optimal medical therapy, potentially mitigating the impact of these risk factors on mortality. Future prospective studies are warranted to further investigate the influence of hypertension on mortality in these populations.

Fesmire et al.[19] reported that a cTnI increase ≥0.2 ng/mL predicted MACE with 87.7% sensitivity and 61.4% specificity. That study included all emergency department presentations and focused on changes above the cut-off. Mueller et al.[20] examined patients with at least one troponin value above the assay cut-off, finding 88% specificity and 60% sensitivity for delta troponin in predicting MACE. Our study differs by investigating delta changes below the assay cut-off and their association with 30-day MACE.

Although delta troponin changes in our study were found to be clinically significant, the obtained AUC values indicate a limited discriminatory power. Therefore, combining delta troponin changes with established risk scoring systems in the literature (e.g., the HEART score) may enhance predictive accuracy. Prospective, multicenter studies are needed to test this hypothesis.

Study Limitations

The retrospective nature of the study limited the comprehensive evaluation of dynamic parameters and clinical decision-making in patient management. Key limitations include the inability to determine the precise onset time of symptoms, the lack of standardized follow-up procedures, and the absence of data on certain established risk factors such as family history, central obesity, and smoking status. Prospective follow-up studies are needed to validate our findings. Furthermore, the single-center design of the study restricts the generalizability of the results.

CONCLUSION

In conclusion, although delta changes in cTnI within the normal range did not predict 30-day mortality, they were significantly associated with non-fatal MACE. Monitoring delta cTnI in chest pain patients may thus aid in early identification of those at higher risk for adverse cardiac events.

Ethics

Ethics Committee Approval: This study was approved by the Clinical Research Ethics Committee of University of Health Sciences Türkiye, İzmir Bozyaka Training and Research Hospital (decision number: 7, date: 04.07.2018).

Informed Consent: Retrospective study.

Note: This study is derived from a medical specialty thesis.

Footnotes

Authorship Contributions

Concept: A.T., H.G., C.S., İ.P., Design: A.T., H.G., C.S., İ.P., Data Collection or Processing: A.T., Analysis or Interpretation: A.T., Literature Search: A.T., Writing: A.T., H.G., C.S.

Conflict of Interest: No conflict of interest was declared by the authors.

Financial Disclosure: The authors declared that this study received no financial support.

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