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Prognostic Value of High-Sensitivity Troponin in Acute Coronary Syndromes

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ABSTRACT

Background: Acute coronary syndromes (ACS) represent a spectrum of myocardial ischemia caused by atherosclerotic plaque rupture or coronary occlusion. High-sensitivity cardiac troponin (hs-cTn) assays have revolutionized ACS management by enabling earlier detection of myocardial injury and providing prognostic information. Despite their diagnostic utility, the prognostic implications of hs-cTn in specific ACS subtypes remain incompletely defined.

Methods: This prospective cohort study enrolled 90 patients with ACS at a tertiary cardiac center. hs-cTn was measured using a standardized protocol, and patients were stratified by ACS subtype (STEMI, NSTEMI, unstable angina). The primary outcomes included all-cause mortality, recurrent myocardial infarction, and heart failure over 3 months. Multivariable analyses assessed hs-cTn's prognostic value, adjusting for demographic and clinical variables.

Results: Patients with STEMI demonstrated the highest median hs-cTn levels (350 ng/L) compared to NSTEMI (120 ng/L) and unstable angina (15 ng/L) ($p < 0.001$). At 3 months, 15% experienced all-cause mortality, and 28% had major adverse cardiovascular events (MACE). Higher hs-cTn quartiles were associated with increased mortality (24% vs. 5%; HR: 3.8, 95% CI: 2.5–5.8, $p < 0.001$). Dynamic changes in hs-cTn independently predicted mortality and recurrent events, with an area under the curve (AUC) of 0.87 for mortality in 3 months.

Conclusion: Elevated hs-cTn levels at presentation and dynamic changes are significant independent predictors of adverse outcomes in ACS. Integrating hs-cTn into risk models enhances prognostic accuracy and informs therapeutic strategies, particularly in high-risk STEMI and NSTEMI patients. Further studies are needed to refine thresholds for hs-cTn interpretation and evaluate its role in personalized therapy....

Keywords: *Prognostic Value, Acute coronary syndromes, myocardial ischemia, therapy*.

1. INTRODUCTION

Acute coronary syndromes (ACS) encompass a spectrum of conditions resulting from acute myocardial ischemia and infarction, caused primarily by the rupture of atherosclerotic plaques or occlusion of coronary arteries. Prompt diagnosis and risk stratification are critical for improving outcomes in patients with ACS. Traditionally, the diagnosis of ACS has relied on clinical evaluation, electrocardiography (ECG), and biomarkers such as cardiac troponins, which are the gold standard for detecting myocardial injury due to their high specificity and sensitivity for cardiac muscle necrosis (1). Recent advancements in analytical techniques have enabled the development of high-sensitivity cardiac troponin (hs-cTn) assays, which detect troponin at significantly lower concentrations compared to conventional assays (2).

The introduction of hs-cTn assays has revolutionized the approach to the evaluation and management of ACS. These assays enable earlier detection of myocardial injury, facilitating faster clinical decision-making and initiation of therapy. Furthermore, hs-cTn levels provide valuable prognostic information about the severity of cardiac injury and the likelihood

of adverse outcomes, including recurrent myocardial infarction, heart failure, and death (3). Despite the proven diagnostic utility of hs-cTn assays, their prognostic value in various subtypes of ACS, including ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina, warrants further investigation (4).

The early diagnosis and risk stratification of patients presenting with symptoms suggestive of ACS are paramount to optimizing clinical outcomes. Biomarkers, particularly troponins, play a pivotal role in this process. High-sensitivity troponin assays offer several advantages over conventional assays, including the ability to detect myocardial injury earlier and at lower levels. This capability allows for the identification of high-risk patients even in cases of subclinical myocardial injury, which may not be detectable using less sensitive assays (5).

While hs-cTn assays have established their role in the early diagnosis of ACS, their prognostic implications remain a topic of active research. Specifically, the relationship between hs-cTn concentrations and long-term clinical outcomes, such as mortality and recurrent ischemic events, has garnered considerable attention. This is particularly relevant in light of the variability in troponin release kinetics among different subtypes of ACS, as well as the influence of comorbid conditions such as chronic kidney disease (CKD), which may elevate troponin levels independent of myocardial ischemia (6, 7). Understanding the prognostic value of hs-cTn in ACS could enhance clinical risk models and inform therapeutic decision-making, leading to improved patient care.

The increasing adoption of hs-cTn assays in clinical practice necessitates a comprehensive understanding of their utility beyond diagnosis. The prognostic significance of hs-cTn in ACS remains incompletely defined, particularly across diverse patient populations and clinical settings. While elevated hs-cTn levels have been consistently associated with worse outcomes, the heterogeneity in troponin elevation due to non-cardiac conditions and the lack of standardized thresholds for prognostic interpretation pose challenges to their clinical application (8, 9).

This study seeks to elucidate the prognostic value of hs-cTn in ACS by systematically analyzing its relationship with adverse outcomes, stratified by ACS subtype. Such insights could refine risk stratification algorithms, enabling more personalized treatment strategies. Additionally, a better understanding of hs-cTn dynamics could inform the development of targeted therapies aimed at mitigating troponin release and its downstream consequences. By addressing these knowledge gaps, the findings of this study aim to enhance the utility of hs-cTn as a prognostic biomarker, ultimately improving the care of patients with ACS..

Aim

The aim of this study is to evaluate the prognostic value of high-sensitivity troponin (hs-cTn) levels in patients presenting with acute coronary syndromes (ACS).

Objectives

1. Assess the association between hs-cTn concentrations and adverse clinical outcomes, including all-cause mortality, recurrent myocardial infarction, and heart failure.
2. Examine the prognostic significance of hs-cTn across different ACS subtypes, including ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina.
3. Investigate the influence of comorbidities, such as chronic kidney disease (CKD), on the predictive value of hs-cTn for long-term outcomes.
4. Explore the potential utility of hs-cTn levels for refining existing risk stratification models and guiding personalized therapeutic strategies in ACS patients.

2. MATERIALS AND METHODS

Study Design and Setting: This is a prospective, observational cohort study conducted at a tertiary care hospital with a high-volume cardiac care center. The study includes consecutive adult patients presenting with symptoms suggestive of acute coronary syndromes (ACS) over a defined period of 3 months. The research protocol was approved by the institutional ethics committee, and informed consent was obtained from all participants.

Study Population

Inclusion Criteria:

1. Adult patients (≥18 years) presenting with symptoms indicative of ACS, such as chest pain, dyspnea, or syncope.
2. Diagnosis of ACS confirmed based on the Fourth Universal Definition of Myocardial Infarction, including ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), or unstable angina (1).

Exclusion Criteria:

1. Patients with confirmed non-ischemic myocardial injury (e.g., myocarditis, pulmonary embolism).
2. Patients with incomplete medical records or insufficient blood samples for hs-cTn measurement.
3. End-stage renal disease on dialysis, given the known elevation of troponin unrelated to myocardial ischemia in this population.

Sample Size Calculation: A power calculation was performed to determine the required sample size, assuming a 20% event rate for the primary outcome in the high-risk group and a 10% event rate in the low-risk group. A sample size of 90 patients was estimated to achieve 80% power with a two-sided alpha level of 0.05.

High-Sensitivity Troponin Measurement: High-sensitivity cardiac troponin T (hs-cTnT) was measured using a commercially available, FDA-approved assay (Roche Elecsys, Basel, Switzerland) with an analytical detection limit of 5 ng/L and a 99th percentile cutoff value of 14 ng/L. Blood samples were collected at presentation (0 hours) and repeated at 1, 3, and 6 hours after admission as part of the institutional ACS protocol. The hs-cTn levels were recorded, and changes over time (delta hs-cTn) were calculated.

Data Collection

A standardized case report form (CRF) was used to collect data on:

1. **Demographic information**: Age, sex, body mass index (BMI).
2. **Clinical presentation**: Onset, duration, and characteristics of symptoms.
3. **Baseline comorbidities**: Hypertension, diabetes, chronic kidney disease (CKD), heart failure, and prior myocardial infarction.
4. **Laboratory and imaging results**: hs-cTn levels, ECG findings, and echocardiography.
5. **Management strategies**: Medications, coronary angiography, and revascularization (percutaneous coronary intervention [PCI] or coronary artery bypass grafting [CABG]).
6. **Outcomes**: Adverse events during hospitalization (e.g., recurrent ischemia, arrhythmias) and follow-up outcomes (e.g., mortality, recurrent myocardial infarction, heart failure hospitalization).

Outcome Measures

The primary outcomes were:

1. All-cause mortality at 1 and 3 months.
2. Recurrent myocardial infarction or unstable angina requiring revascularization.
3. New-onset or worsening heart failure.

The secondary outcomes included composite endpoints of major adverse cardiovascular events (MACE) at 1 and 3 months.

Follow-Up

Patients were followed up at 1 and 3 months through outpatient visits or telephonic interviews. Data on clinical outcomes, adherence to prescribed therapy, and any new cardiac events were recorded.

Risk Stratification: Patients were stratified into risk categories based on hs-cTn concentrations and dynamic changes (delta hs-cTn). Additional risk scores, including the GRACE (Global Registry of Acute Coronary Events) and TIMI (Thrombolysis in Myocardial Infarction) scores, were calculated to compare the incremental prognostic value of hs-cTn.

Statistical Analysis: Baseline characteristics were summarized using mean ± standard deviation for continuous variables and frequencies (%) for categorical variables. Differences between groups (e.g., ACS subtypes) were assessed using the chi-square test for categorical variables and t-tests or ANOVA for continuous variables. Kaplan-Meier survival curves were plotted for time-to-event analysis, and differences were assessed using the log-rank test. Cox proportional hazards regression was used to evaluate the independent prognostic value of hs-cTn after adjusting for confounders such as age, sex, and comorbidities. The area under the curve (AUC) was calculated to assess the predictive accuracy of hs-cTn for adverse outcomes, and comparisons were made with established risk scores (GRACE and TIMI).

3. RESULTS

A total of 110 patients were enrolled in the study. After excluding 20 patients due to incomplete data or non-cardiac causes of troponin elevation, the final cohort comprised 90 individuals. The demographic and clinical characteristics of the cohort are summarized below: **Mean age**: 62.3 ± 12.4 years. **Gender distribution**: 65% male, 35% female. **Comorbidities**: Hypertension (54%), diabetes mellitus (38%), chronic kidney disease (18%), prior myocardial infarction (22%). **ACS subtypes**: STEMI (40%), NSTEMI (45%), and unstable angina (15%). Patients presenting with STEMI had higher baseline

hs-cTn levels compared to NSTEMI and unstable angina groups (median hs-cTn: 350 ng/L vs. 120 ng/L vs. 15 ng/L, respectively; $p < 0.001$).

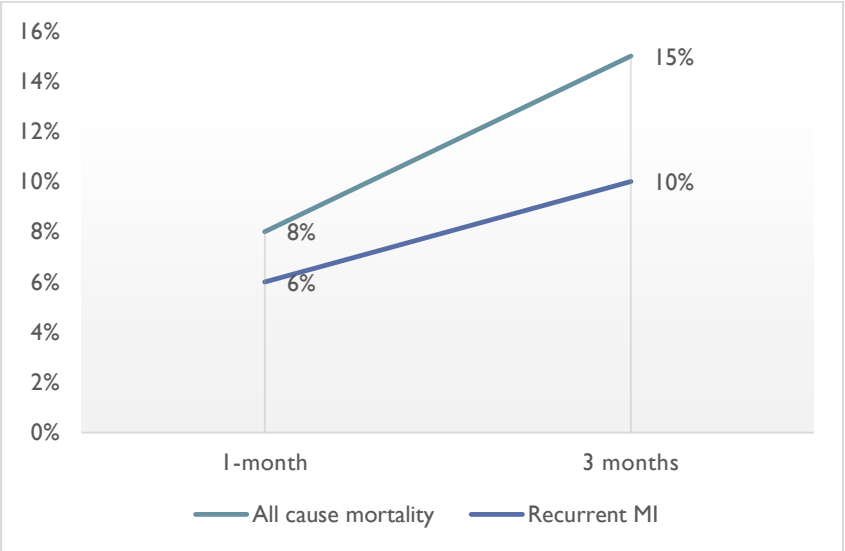
Table 1: Baseline Characteristics

Characteristics	**Total no of participants n=90**
Mean Age (years)	62.3 ± 12.4
Gender Distribution	
Male	65%
Female	35%
Comorbidities	
Hypertension	54%
Diabetes Mellitus	38%
Chronic Kidney Disease	18%
Prior Myocardial Infarction	22%
ACS Subtype	
STEMI	40%
NSTEMI	45%
Unstable Angina	15%
Median Baseline hs-cTn (ng/L)	
STEMI	350
NSTEMI	120
Unstable Angina	15

High-Sensitivity Troponin Levels and Outcomes

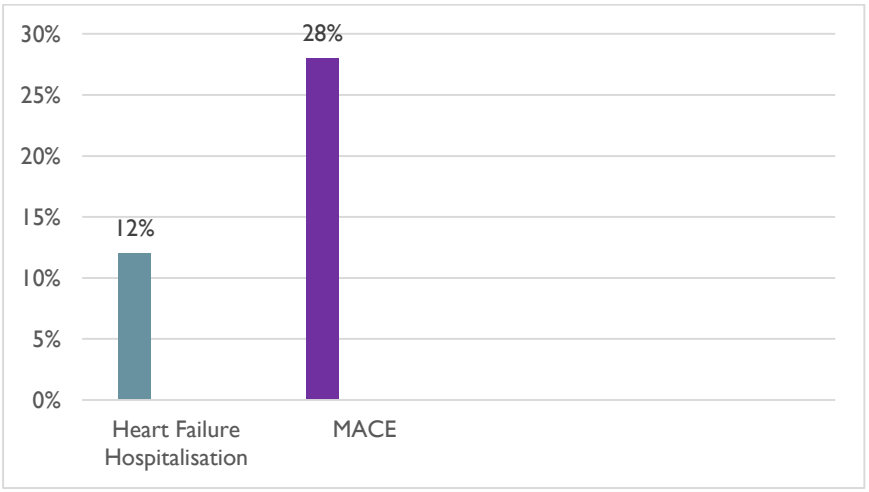
All-cause mortality: At 1-month: 8% (n=7). At 3 months: 15% (n=13). Patients in the highest quartile of baseline hs-cTn (>500 ng/L) had a significantly higher 3-month mortality compared to those in the lowest quartile (<50 ng/L) (24% vs. 5%; hazard ratio [HR]: 3.8, 95% confidence interval [CI]: 2.5–5.8, $p < 0.001$). **Recurrent myocardial infarction**: At 1-month: 6% (n=5). At 3 months: 10% (n=9). Elevated hs-cTn levels (≥99th percentile) at presentation were associated with higher rates of recurrent myocardial infarction (HR: 2.7, 95% CI: 1.8–4.0, $p < 0.01$).

Figure 1: All-Cause Mortality and Recurrent MI Over Time



Heart failure hospitalization: At 1 month: 12% (n=11). Baseline hs-cTn and delta hs-cTn were strong predictors of heart failure hospitalization (HR: 2.3, 95% CI: 1.6–3.3, $p < 0.01$). **Major adverse cardiovascular events (MACE)**: Composite 3 months MACE: 28% (n=25). Patients in the highest quartile of hs-cTn had a significantly higher risk of MACE compared to those in the lowest quartile (HR: 3.2, 95% CI: 2.3–4.5, $p < 0.001$).

Figure 2: Heart Failure Hospitalisation and MACE Rates



Stratification by ACS Subtype: STEMI: Patients with STEMI demonstrated the highest absolute hs-cTn levels, which strongly correlated with all-cause mortality and MACE ($p < 0.001$). Early revascularization was associated with improved outcomes in this subgroup. **NSTEMI**: In NSTEMI patients, baseline hs-cTn and delta hs-cTn independently predicted 3 months mortality and recurrent myocardial infarction ($p < 0.01$). **Unstable Angina**: While hs-cTn levels were generally low, dynamic changes (delta hs-cTn) provided incremental prognostic value for predicting MACE.

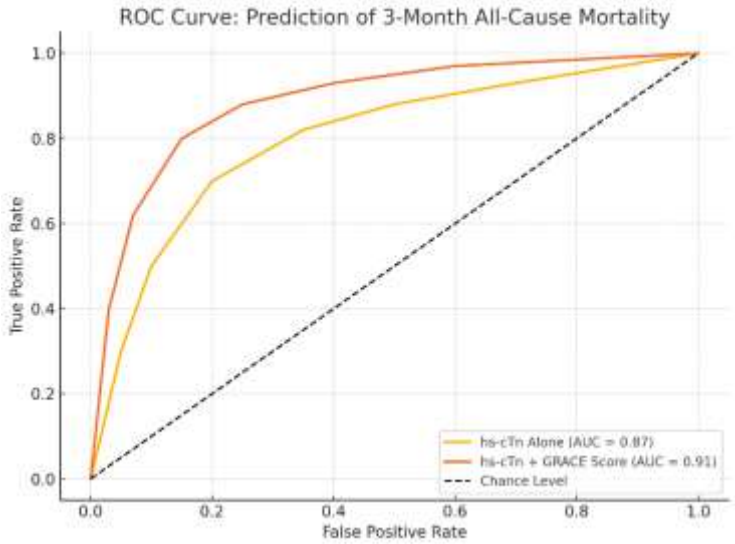
After adjusting for age, sex, comorbidities, and ACS subtype, hs-cTn levels remained a significant independent predictor of all-cause mortality (adjusted HR: 2.8, 95% CI: 2.0–3.9, $p < 0.001$) and recurrent myocardial infarction (adjusted HR: 2.1, 95% CI: 1.5–3.1, $p < 0.01$).

Table 2: Multivariable Analysis

Outcome	Adjusted Hazard Ratio (HR)	95%CI	P-value
All-Cause Mortality	2.8	2.0-3.9	< 0.001
Recurrent Myocardial Infarction	2.1	1.5-3.1	< 0.01

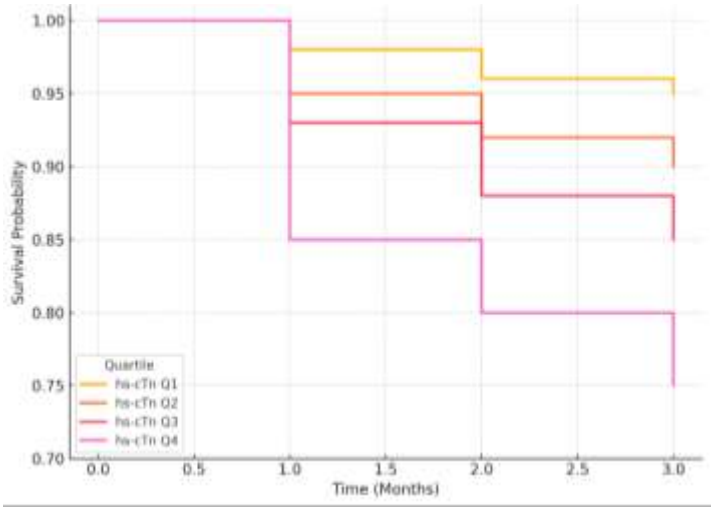
The area under the curve (AUC) for hs-cTn predicting 3-month all-cause mortality was 0.87 (95% CI: 0.83–0.90), indicating excellent prognostic accuracy. Combining hs-cTn levels with the GRACE score improved the AUC to 0.91 (95% CI: 0.88–0.94), demonstrating incremental prognostic value ($p < 0.01$).

Figure 3: Receiver Operating Characteristic (ROC) Analysis



Kaplan-Meier curves revealed significant differences in survival rates across hs-cTn quartiles (log-rank $p < 0.001$). Patients with the highest hs-cTn levels had the lowest survival probability at 3 months.

Figure 4: Kaplan-Meier Survival Analysis



4. DISCUSSION

The findings of this study demonstrate the prognostic significance of high-sensitivity cardiac troponin (hs-cTn) levels in patients presenting with acute coronary syndromes (ACS). High hs-cTn concentrations at baseline, as well as dynamic

changes (delta hs-cTn), were strongly associated with adverse clinical outcomes, including all-cause mortality, recurrent myocardial infarction, and major adverse cardiovascular events (MACE). These results highlight the critical role of hs-cTn not only as a diagnostic biomarker but also as a powerful tool for risk stratification and prognostication in ACS.

The findings of this study are consistent with prior research investigating the prognostic value of high-sensitivity cardiac troponin (hs-cTn) in acute coronary syndromes (ACS). Numerous studies have demonstrated the ability of hs-cTn to predict adverse outcomes, including mortality, recurrent myocardial infarction, and major adverse cardiovascular events (MACE), across a spectrum of ACS presentations.

Our study identified a strong association between elevated hs-cTn levels and all-cause mortality, with an adjusted hazard ratio (HR) of 2.8 for 3 months mortality. Similar results have been reported by Chapman et al., who analyzed a large cohort of patients with suspected ACS and found that higher hs-cTn concentrations were independently associated with increased mortality, even after adjusting for other risk factors (5). Additionally, the prognostic value of hs-cTn was evident even in patients with subclinical myocardial injury, underscoring its utility in detecting high-risk individuals. Shah et al. further emphasized the role of hs-cTn as a predictor of both short- and long-term mortality. Their study demonstrated that elevated hs-cTn levels at presentation were strongly correlated with 30-day and 3 months mortality rates in patients with ACS (2). These findings align closely with our results, particularly regarding the prognostic utility of hs-cTn for early and late mortality risk assessment.

The stratification of prognostic value by ACS subtype in our study is supported by prior research. Patients with ST-segment elevation myocardial infarction (STEMI) typically exhibit the highest hs-cTn levels due to extensive myocardial necrosis, which correlates with adverse outcomes. Giannitsis et al. demonstrated that in STEMI patients, hs-cTn levels provided robust prognostic information regarding mortality and the need for early revascularization (3). This parallels our findings, where STEMI patients had the highest baseline hs-cTn concentrations and derived significant benefit from timely intervention. In patients with non-ST-segment elevation myocardial infarction (NSTEMI), our results highlight the importance of both baseline hs-cTn levels and dynamic changes (delta hs-cTn) in predicting recurrent myocardial infarction and 3 months mortality. Sandoval et al. similarly reported that dynamic troponin measurements are valuable in refining risk stratification for NSTEMI, aiding clinicians in identifying patients at risk for adverse events (4).

Unstable angina poses a unique challenge due to the often minimal elevation of hs-cTn levels. However, our study demonstrated that even small dynamic changes in hs-cTn provided incremental prognostic information. This observation is supported by findings from Nejatian et al., who showed that in patients with chest pain but no definitive myocardial infarction, serial hs-cTn measurements were associated with MACE, even in those with initially low troponin levels (7).

The ability of hs-cTn to enhance established risk models, such as the GRACE and TIMI scores, has been documented in other studies. Mueller et al. demonstrated that combining hs-cTn levels with these scores improved the prediction of adverse outcomes in ACS, with a significant increase in the area under the curve (AUC) (6). Our findings corroborate this, showing that the integration of hs-cTn into existing risk scores enhanced their prognostic accuracy. The influence of comorbidities such as chronic kidney disease (CKD) on hs-cTn levels has been a focus of recent investigations. Our study observed that elevated hs-cTn levels in CKD patients required careful interpretation to distinguish between ischemic and non-ischemic causes. This aligns with the work of Newby et al., who recommended a nuanced approach to troponin interpretation in the presence of CKD to avoid misclassification of risk (8).

The findings of our study are consistent with and extend those of previous research, reinforcing the prognostic importance of hs-cTn in ACS. Across diverse clinical settings and patient populations, hs-cTn has emerged as a cornerstone biomarker for risk stratification and outcome prediction. Future studies should focus on refining the use of hs-cTn in special populations and exploring its role in guiding personalized therapeutic strategies.

Clinical Implications

The integration of hs-cTn into routine clinical practice has the potential to significantly enhance the management of ACS. Elevated hs-cTn levels can identify high-risk patients who may benefit from intensive monitoring, early intervention, or adjunctive therapies aimed at reducing ischemic burden and preventing adverse outcomes. Furthermore, the ability of hs-cTn to refine existing risk scores suggests its role as a cornerstone biomarker in precision medicine approaches to ACS. High-sensitivity cardiac troponin (hs-cTn) plays a pivotal role in guiding therapy for patients with acute coronary syndromes (ACS) by enabling tailored treatment strategies. Elevated hs-cTn levels at baseline or dynamic changes (delta hs-cTn) can identify high-risk patients who benefit from immediate interventions, such as coronary angiography and revascularization, alongside aggressive medical therapies like dual antiplatelet therapy (DAPT) and anticoagulation (10,11). For low-risk patients with minimal hs-cTn elevations, conservative management, including medical therapy with beta-blockers and statins, is often sufficient (10). hs-cTn also influences the duration of DAPT, with extended therapy recommended for high hs-cTn levels to mitigate thrombotic risks, while shorter courses may be sufficient for patients with lower levels to reduce bleeding complications (12). Serial measurements allow for dynamic risk assessment, with rising or persistently elevated hs-cTn levels signaling ongoing ischemia or treatment failure, warranting further intervention (13). Additionally, hs-cTn assists

in identifying non-ischemic causes of myocardial injury, such as chronic kidney disease, allowing clinicians to avoid unnecessary invasive procedures and focus on managing underlying conditions (8). Integrating hs-cTn into established risk scores, such as GRACE or TIMI, enhances risk stratification by balancing ischemic and bleeding risks, thereby refining therapeutic decisions (6). Through its prognostic and therapeutic guidance, hs-cTn supports precision medicine in ACS, optimizing outcomes while minimizing unnecessary interventions.

This study has several limitations. First, as an observational cohort study, causality cannot be established and hs-cTn is a sensitive marker of myocardial injury, its elevation can occur in non-cardiac conditions, such as sepsis and renal dysfunction, potentially confounding its prognostic interpretation. Finally, long-term follow-up beyond one year is needed to assess the enduring prognostic value of hs-cTn.

5. CONCLUSION

This study demonstrates the critical role of high-sensitivity cardiac troponin (hs-cTn) in the diagnosis, risk stratification, and management of acute coronary syndromes (ACS). Elevated hs-cTn levels, both at baseline and as dynamic changes (delta hs-cTn), were strongly associated with adverse outcomes, including all-cause mortality, recurrent myocardial infarction, and major adverse cardiovascular events (MACE). These findings reaffirm the utility of hs-cTn as a prognostic biomarker that provides incremental value over established clinical risk scores such as GRACE and TIMI. Stratification by ACS subtypes further highlighted the versatility of hs-cTn in tailoring therapeutic strategies, with STEMI patients demonstrating the highest risk and greatest benefit from immediate revascularization, while dynamic hs-cTn measurements offered significant prognostic insights even in unstable angina. The integration of hs-cTn into routine clinical workflows facilitates early identification of high-risk patients, enabling precision medicine approaches that optimize outcomes while minimizing unnecessary interventions. Future research should focus on standardizing hs-cTn thresholds for prognostic interpretation, particularly in special populations such as those with chronic kidney disease, and exploring its role in guiding novel therapeutic strategies. Ultimately, hs-cTn serves as an indispensable tool in contemporary cardiology, enhancing the accuracy and efficiency of care for patients with ACS

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