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Clinical Research Study

Implementation of High-Sensitivity Troponin for Early Rule-Out of Acute Myocardial Infarction in Emergency Department☆,☆☆

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ABSTRACT

Background/Purpose: Chest pain is a common reason for ED visits. Implementing a HEART score-based algorithm previously increased early discharges by 99%. This study aims to determine if the transition from cTnT to hs-cTnT assays affected patient disposition rates.
Methods: This retrospective observational study was conducted in a multi-site hospital system. Adults presenting to the ED with chest pain and a low HEART score (≤3) between November 9, 2020, and November 10, 2022, were included. The primary outcome was the change in patient disposition. Secondary outcomes included length-of-stay (LOS), rates of provocative testing, ED returns, and major adverse cardiovascular events (MACE).
Results: We evaluated 32,968 patients (17,173 in the cTnT group and 15,795 in the hs-cTnT group). Both groups had a similar median age, but the hs-cTnT group had a higher proportion of patients with baseline troponin elevations. The ED discharge rate was higher in the hs-cTnT group (87.5%) compared to the cTnT group (85.3%; $P < .001$), with a corresponding decrease in observation and inpatient admissions. Additionally, the implementation of hs-cTnT was associated with a reduced LOS and a decrease in patients undergoing further testing. Finally, there was a reduction in ED re-visits without a difference in 30- or 60-day MACE after the implementation of hs-cTnT.
Conclusions: Integration of hs-cTnT into our chest pain clinical pathway resulted in increased ED discharges, reduced LOS, and fewer additional tests without a change in MACE. This translates to a savings of almost 7,000 ED hours annually without compromising safety.

Clinical Significance

- High-sensitivity troponin (hs-cTn) assays can detect lower concentrations than conventional assays and enable rapid detection/exclusion of MI, and implementation of HEART score or hs-cTn into clinical pathways can decrease admission rate, LOS, and increase ED discharge.
- Implementation of hs-cTn within the HEART score pathway was associated with increased ED discharges, decreased admissions, decreased ED revisits, without a difference in MACE.
- Findings should prompt prospective research as there are potential QI changes, such as the broad adoption of a 1-hour hs-cTn decision algorithm within our health system.

Introduction

Chest pain and related syndromes were among the 10 leading reasons for emergency department (ED) visits in the United States during 2016-2019. On estimate, more than 8 million patients presented to the ED with chest pain annually.[1] Upon presentation, it is important to dis-

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tinguish cardiac causes, such as acute coronary syndrome (ACS), versus non-cardiac. Guidelines recommend a focused cardiovascular examination to aid in the diagnosis of ACS, including obtaining an electrocardiogram (ECG) within 10 minutes of arrival and measuring troponin as soon as possible after presentation.[2] In addition, risk stratification using validated clinical pathways can identify patients eligible for discharge without a requirement for further diagnostic testing.[3]

Both prospective and retrospective studies have determined that implementation of clinical pathways leads to an increase in safe discharge rates of low-risk chest pain patients and reductions in admissions to inpatient or observation units.[4-7] Between 2015 and 2016, a written consensus algorithm was designed and implemented in our 9-campus hospital system in Florida to establish the standard-of-care in adult patients presenting to the ED with undifferentiated chest pain. This incorporated the HEART (History, EKG, Age, Risk factors, Troponin) score as a stratification tool, using conventional troponin (cTnT) assays to measure this biomarker for myocardial damage. In a previous case study conducted within our health system, the implementation of this clinical pathway resulted in a 99% increase in chest pain patients discharged from the ED, 20% decrease in 'Observation' utilization, and 63% decrease in inpatient admissions ($P < .0001$).[8]

On November 10, 2021, high-sensitivity troponin (hs-cTnT) assays were incorporated into the chest pain clinical pathway in place of cTnT assays at our health system. Fifth generation hs-cTnT assays can detect troponin at concentrations 10- to 100-times lower than conventional assays.[9,10] For this reason, contemporary guidelines endorse hs-cTnT as the preferred biomarker for assessment of chest pain patients, as this assay can rapidly detect or rule out myocardial injury and improve diagnostic precision.[2]

Existing peer-reviewed literature evaluating the impact of implementation of a strategy including hs-cTnT at 0 and 1 hour combined with the HEART score determined that after the introduction of the new strategy, the admission rate decreased significantly from 59% to 33%.[11] Additionally, incorporation of hs-cTnT into the pathway significantly increased ED discharge and decreased median length of ED stay.[12] The primary purpose of this study was to determine whether the implementation of hs-cTnT assays as part of the HEART score pathway is associated with a significant change in the disposition decision rate of patients (ED discharge, Observation, Inpatient) as compared to cTnT assays in our hospital system.

Methods

This retrospective observational study was conducted in a multi-site hospital system in Florida, including 9 hospitals and 7 stand-alone emergency departments. Adult patients (≥18 years) presenting to the ED with a chief complaint of chest pain between November 9, 2020 and November 10, 2022, were included, which corresponds to 1 year before and after the transition to hs-cTnT assays (Figure 1). ED provider education was disseminated 2 weeks prior utilizing the assay manufacturer (Roche Diagnostics) recommendations for sex-specific hs-cTnT cutoffs as well as delta information. Additionally, the electronic health record (EHR) was updated to recognize Δ troponin results as well as to automatically calculate and input a HEART score when the pathway was initiated. To mirror the timing of the chest pain pathway with the c-TnT assay, a 0-3-hour testing strategy was maintained with the intent to evolve as the hs-cTnT literature adjusts. Patients must have had at least 1 troponin level and a documented HEART score for study inclusion. Outcomes were assessed among patients with presentation not highly concerning for ACS, with a documented HEART score ≤3. Patients with missing data, those who left without being seen, or against medical advice, duplicate or outpatient encounters were excluded, as shown in Figure 1. Data were extracted and cross-referenced for accuracy from centralized dashboards, including an internal system-wide chest pain dashboard and percutaneous coronary intervention (PCI) registry. The system chest pain database included all patient demographics, baseline characteristics, and outcomes, except for the provocative testing data in which the PCI registry was referenced. Race and ethnicity were included for internal and external validity of the sample and were reported as coded in the system EHR. This study was determined to be a qualitive improvement study and was deemed exempt from full review by the institutional review board.

The study consisted of 2 phases, i.e., before and after the implementation of hs-cTnT into the chest pain pathway. The conventional assay used across our hospital system was the Roche Elecsys Troponin T STAT, with a limit of detection of 0.01 μg/L, limit of quantitation (20% CV) of 6 ng/L, and 10% imprecision 11 ng/L. In the post-implementation group, the hs-cTnT assay used was the Elecsys Troponin T Gen 5 STAT, with a limit of detection 3-5 ng/L, and limits of quantitation and 10% imprecision consistent with those seen with our conventional assay. The clinical pathway for patients presenting with low-risk chest pain (HEART score ≤3) is shown in Figure 2. This strategy includes assessment of ECG changes, time elapsed between initial symptoms and presentation to the ED, measurement of troponin levels (cTnT or hs-cTnT) at presentation and within 3 hours, and other components of the HEART score. An initial troponin was below the upper limit of normal if hs-cTnT was ≤22 ng/L in males, hs-cTnT was ≤14 ng/L in females, or cTnT <0.03 ng/L regardless of sex. For patients presenting >6 hours after their last chest pain episode, a serial troponin was not needed. If the first value was within the normal reference range, the patient could safely be discharged. For patients presenting within 6 hours of their last chest pain episode, serial troponins were indicated within 3 hours and the absolute change in troponin (Δ value) was calculated. If the Δ value for hs-cTnT within 1 hour was ≥3 ng/L, hs-cTnT within or after 3 hours was ≥7 ng/L, or there was a ≥20% increase in the cTnT value at any subsequent time, the Δ value was considered positive. Patients with a low HEART score and a positive Δ value should be admitted to an observation unit. Conversely, if the Δ value was negative, a myocardial infarction (MI) was considered unlikely, and the patient could safely be discharged. Nevertheless, if the ED providers determine that it is clinically indicated, they could overrule pathway determinations. The change from cTnT to hs-cTnT assays was the only modification to the chest pain pathway. Due to our study design, we were unable to assess ECG changes or time between symptom onset and patient presentation and based the adherence to pathway disposition recommendations solely on troponin values.

The primary outcome was the change in patient disposition rate during the periods of utilization of either cTnT or hs-cTnT assays within the chest pain pathway among patients with a low (≤3) HEART score. The potential dispositions were ED discharge, admission to an observation unit, or admission to an inpatient unit. Secondary outcomes included LOS, rates of provocative testing with coronary computed tomography angiography (CCTA), diagnostic catheterizations, or percutaneous coronary intervention (PCI), return to the ED within 30 or 60 days, and major adverse cardiovascular events (MACE) within 30 or 60 days. MACE was defined as a composite of MI, stroke, or all-cause death.

Statistical Analysis

Descriptive statistics were used to summarize baseline characteristics of both the cTnT and hs-cTnT groups. Continuous variables were reported as median and interquartile range (IQR) for its decreased sensitivity to outliers. Categorical variables were reported as absolute frequency and corresponding percentage. Among patients with a low HEART score, categorical variables were analyzed using Chi-square test and continuous, non-parametric data were analyzed using the Mann-Whitney U test. All statistical comparisons were performed using SPSS Statistics version 29 (IBM Corp., Armonk, NY, USA).

Results

A total of 226,878 patient ED encounters with chest pain were screened for eligibility. Among them, 85,382 duplicate encounters were

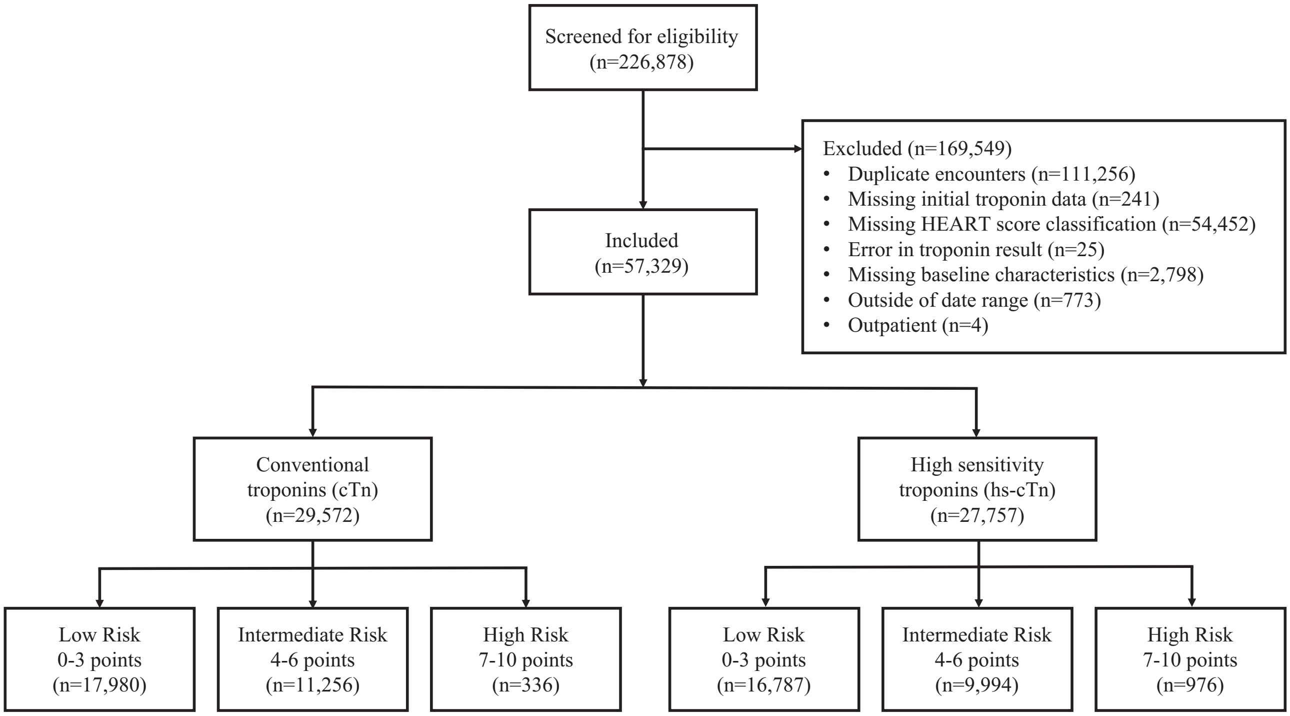

Figure 1. Participant flow diagram. *Abbreviations:* HEART, History, Electrocardiogram, Age, Risk factors, Troponin; LWBS, left without being seen; AMA, against medical advice; cTn, conventional troponin; hs-cTn, high-sensitivity troponin.

Table 1
Baseline characteristics of chest pain patients.

Patient Baseline Characteristics	Entire Cohort			Low HEART Score (0-3)		
	cTn, n=27,884	hs-cTn, n=25,312	*p*-Value	cTn, n=17,173	hs-cTn, n=15,795	*p*-Value
HEART score classification						
Low (0-3 pts), n (%)	17,173 (61.6%)	15,795 (62.4%)	<0.001			
Intermediate (4-6 pts), n (%)	10,378 (37.2%)	8,638 (34.1%)	<0.001			
High (7-10 pts), n (%)	333 (1.2%)	879 (3.5%)	<0.001			
Age (years), median [IQR]	48 [36-60]	48 [35-61]	0.014	40 [31-49]	40 [31-49]	0.889
Sex, male, n (%)	11,617 (41.7%)	10,645 (42.1%)	0.358	6,819 (39.7%)	6,266 (39.7%)	0.945
Race[a]						
American Indian or Alaska Native, n (%)	112 (0.4%)	194 (0.8%)	<0.001	54 (0.3%)	93 (0.6%)	<0.001
Asian, n (%)	397 (1.4%)	448 (1.8%)	0.002	217 (1.3%)	264 (1.7%)	0.002
Black or African American, n (%)	5,903 (21.2%)	5,575 (22%)	NS	3,865 (22.5%)	3,701 (23.4%)	0.046
Multiple, n (%)	606 (2.2%)	387 (1.5%)	<0.001	370 (2.2%)	251 (1.6%)	<0.001
Native Hawaiian or Pacific Islander, n (%)	29 (0.1%)	25 (0.1%)	NS	17 (0.1%)	18 (0.1%)	NS
White, n (%)	16,433 (58.9%)	14,426 (57%)	<0.001	9,754 (56.8%)	8,618 (54.6%)	<0.001
Other, n (%)	4,404 (15.8%)	4,097 (16.2%)	NS	2,896 (16.9%)	2,850 (18%)	0.005
Ethnic Group						
Hispanic or Latino, n (%)	10,938 (39.2%)	7,735 (30.6%)	<0.001	7,241 (42.2%)	5,995 (38%)	<0.001
Non-Hispanic or Non-Latino, n (%)	16,765 (60.1%)	15,987 (63.2%)	<0.001	9,826 (57.2%)	9,710 (61.5%)	<0.001
Multiple, n (%)	181 (0.6%)	72 (0.3%)	<0.001	106 (0.6%)	40 (0.3%)	<0.001
Troponin						
Initial troponin >ULN, n (%)	652 (2.3%)	2,587 (10.2%)	<0.001	50 (0.3%)	223 (1.4%)	<0.001

Abbreviation: IQR, interquartile range; HEART, History, Electrocardiogram, Age, Risk factors, Troponin; cTn, conventional troponin; hs-cTn, high-sensitivity troponin; ULN, upper limit of normal; NS, not significant.

[a] Race categories were documented as coded in the electronic health record

excluded. Additional exclusion reasons are outlined in Figure 1. A total of 53,196 encounters were assessed, with 27,884 in the cTnT group (pre-implementation) and 25,312 in the hs-cTnT group (post-implementation). The median age and proportion of males were similar between groups (48 years; 41.7% vs 42.1% males). Most patients had a low HEART score 0-3 (61.6% vs 62.4%; p=NS) and could potentially be discharged from the ED without further interventions. Patients in this group were younger, with a median age of 40 years (IQR 31-49 years), but other general baseline characteristics were similar (Table 1). Although the absolute frequency of patients with elevated troponin at baseline was lower in this subset of patients with a low HEART score, a trend was observed where a higher proportion of patients in the hs-cTnT group had troponin elevations at baseline, as compared to the cTnT group (0.3% in the cTnT group vs 1.5% in the hs-cTnT group; $P < .001$), which was also present when comparing pre- and post-implementation groups across the spectrum of HEART scores.

The ED discharge occurred in 85.3% in the cTnT group versus 87.5% in the hs-cTnT group ($P < .001$), an absolute difference of 2.2%. Ad-

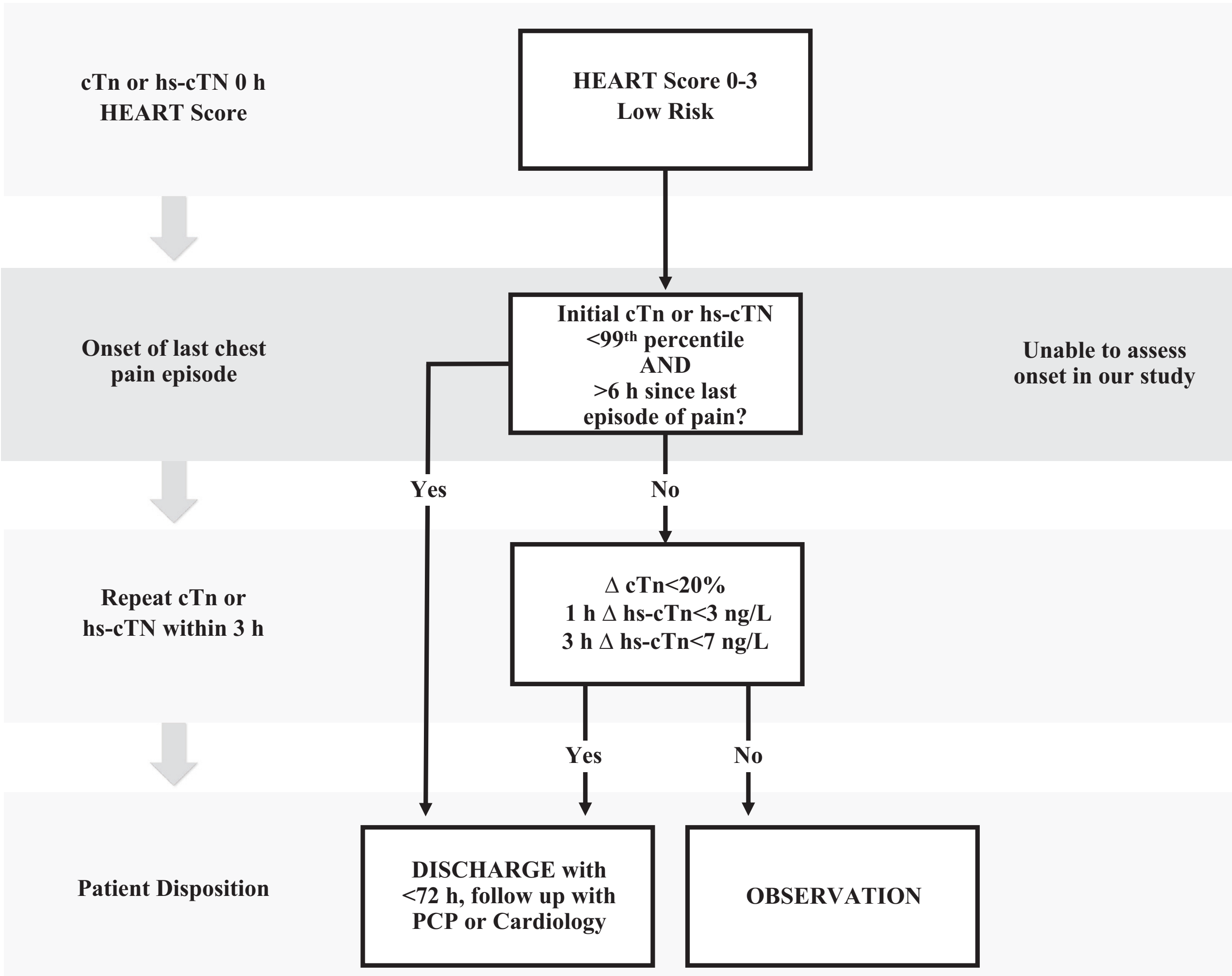

Figure 2. Chest pain pathway for patients with a low (≤3) HEART score. *Abbreviations:* HEART, History, Electrocardiogram, Age, Risk factors, Troponin; cTn, conventional troponin; hs-cTn, high-sensitivity troponin; PCP, primary care physician.

ditionally, there was a significant decrease in observation (12.8% vs 10.8%; $P < .001$) and inpatient admissions (2.0% vs 1.6%; $P = .02$). Moreover, after the implementation of hs-cTnT there was a reduced median LOS (4.87 vs 4.46 hours; $P < .001$) and a reduction in the proportion of patients undergoing further testing with CCTA (6.9% vs 5.6%; $P < .001$). There was no difference in the proportion of patients undergoing diagnostic catheterizations (0.06% vs 0.09%; $P = .318$) or PCI (0.05% vs 0.03%; $P = .361$) between groups. Despite a decrease in protocol adherence when hs-cTnT was implemented, primarily through increased ED discharges when Observation was indicated, there was a decrease in 30- and 60-day all-cause ED revisits (7.8% vs 6.9%, $P < .001$; 10.2% vs 8.9%, $P < .001$) without a significant difference in 30- and 60-day MACE (0.08% vs 0.06%, $P = .540$; 0.11% vs 0.09%, $P = 0658$) (Table 2).

Discussion

To our knowledge, this is 1 of the largest retrospective cohort studies assessing outcomes upon switching from cTnT to hs-cTnT assays, specifically in low-risk chest pain.[13,14] The implementation of hs-cTnT assays in the chest pain pathway in low-risk patients was associated with a significant increase in the proportion of patients discharged from the ED (85.3% to 87.5%; $P < .001$). We observed a decrease in utilization of observation units, indicating that implementation of hs-cTnT may have resulted in more direct ED discharges without the need for prolonged monitoring or further diagnostic testing. This coincides with the finding of reduced LOS and decreased CCTA utilization, supporting the notion that hs-cTnT enables more rapid detection or exclusion of myocardial injury and facilitates efficient patient management. When applied to our system's population of low-risk chest pain patients, the adoption of hs-cTnT assay was associated with a savings of almost 7,000 hours of ED bed-time annually. Overall, these findings are congruent with other peer-reviewed published literature. A previous prospective, multicenter study found that implementation of the HEART score and hs-cTnT assays led to a decrease in admission rate from 59% to 33% (RR 0.55 [95% CI 0.48 to 0.63], reduced LOS from 23.2 to 4.7 hours, and reduced healthcare related costs.[6] Another real-world study conducted at a large academic medical center also observed a comparable reduction in ED LOS from 6.2 (IQR 4.7-8.4) to 5.3 (IQR 4.0-7.2) hours among low-risk chest pain patients directly discharged to home, demonstrating a similar clinical impact of hs-cTnT implementation.[14]

Consistent with smaller prospective studies, we found no difference in new presentations to the ED or MI after discharge.[11] Furthermore, our results on provocative testing were aligned with those of other large studies, which showed no difference in invasive cardiac testing.[15] A retrospective analysis comparing outcomes before and after implementation of the HEART pathway within the EHR found that while low-risk chest pain patients were discharged in 82.3% of encounters, 1.6% of patients experienced death or MI at 1 year, representing a negative predictive value of 97.5% for this clinical decision tool among low and non-low risk patients.[6] Factors including differences in baseline char-

Table 2
In-hospital, 30-day and 60-day outcomes of low-risk chest pain patients.

Outcomes	cTn, n=17,173	hs-cTn, n=15,795	*p*-Value
Patient disposition			
ED discharge, n (%)	14,644 (85.3%)	13,826 (87.5%)	<0.001
Observation, n (%)	2,192 (12.8%)	1,713 (10.8%)	<0.001
Inpatient, n (%)	337 (2.0%)	256 (1.6%)	0.02
LOS (h), median, [IQR]	4.87 [3.85-6.58]	4.46 [3.24-6.48]	<0.001
Provocative testing			
CCTA, n (%)	1,180 (6.9%)	887 (5.6%)	<0.001
PCI, n (%)	9 (0.05%)	5 (0.03%)	0.361
Diagnostic catheterization	11 (0.06%)	15 (0.09%)	0.318
MACE			
30-day MACE, n (%)	14 (0.08%)	10 (0.06%)	0.540
60-day MACE, n (%)	19 (0.11%)	15 (0.09%)	0.658
All Cause Return to the ED			
30-day ED return, n (%)	1,346 (7.8%)	1,084 (6.9%)	<0.001
60-day ED return, n (%)	1,744 (10.2%)	1,401 (8.9%)	<0.001
Protocol Adherence*	11,733 (69.2%)	8,611 (54.6%)	<0.001
Initial troponin ≤1 h	12,539 (74%)	12,017 (76.3%)	<0.001
Repeat troponin within 3 h	16,954 (100%)	14,204 (90.1%)	<0.001
Disposition in accordance with pathway guidelines	15,818 (93.3%)	11,517 (73.1%)	<0.001

Abbreviations: ED, emergency department; LOS, length-of-stayI; IQR, interquartile range; CCTA, coronary computed tomography angiography; PCI, percutaneous coronary intervention; cTn, conventional troponin; hs-cTn, high-sensitivity troponin; MACE, major adverse cardiovascular events.
* Protocol Adherence sample size: cTn 16,955, hs-cTn 15,757

acteristics or the temporal overlap with the Coronavirus Disease 2019 (COVID-19) pandemic and restrictions lifting could have had an impact on our results. Additionally, these could have been affected by decreased protocol adherence in the hs-cTnT group, due in part to patient dispositions differing from pathway recommendations. As a retrospective observational study, we cannot establish a causal relationship between the implementation of hs-cTnT and the observed changes in patient outcomes.

It is important to acknowledge the limitations of our study. Due to its retrospective design, many patient encounters were excluded due to missing data. Patients without a second troponin level were assumed to be low risk. Patients with a past medical history of heart failure, renal dysfunction, or arrhythmias may have chronically elevated troponin and an assessment of the troponin delta is necessary to determine if an acute process is occurring. Due to limitations in troponin data available to be extracted, assumptions had to be made to allow for the calculation of the Δ value. In addition, while manufacturer-specific cutoffs were used to describe a positive Δ value in the hs-cTnT group, an absolute change of Δ value ≥20% was used for the cTnT group.

Our assessment of adherence to pathway disposition was based solely on troponin data. Interpretation of the Δ value should be performed in a clinical context, accounting for other aspects such as medical history, changes in ECG, onset of chest pain or other symptoms, and imaging.[16] While robust data supports using 1 hs-cTnT value below the limit of quantitation to rule out an MI in patients presenting >2 hours after symptom onset[17-20], our study design was unable to capture symptom onset time. Algorithms may be less reliable <2 hours or >12 hours after symptom onset due to troponin level dynamics.[20,21] In addition, although the finding of elevated cardiac troponin is highly sensitive for detecting myocardial injury, the specificity for the acute myocardial infarction (AMI) is moderate, due to non-ACS causes for troponin elevations.[22] In this study, it is unclear what the predictive effect of the hs-cTnT assays had on the rest of the HEART score, as all patients were required to have a documented HEART score and at least 1 troponin level to be included. Additional studies are warranted to determine if management of low-risk chest pain should be further subdivided into HEART scores of ≤1 and 2-3 subgroups to ascertain the potential added benefit of a hs-cTnT in low-risk chest pain. There exists subjectivity of the HEART score with the scoring of chest pain history and risk factors, particularly in the emergency department where patients may receive their first care contact for undiagnosed disease states. The chest pain dashboard utilized in this study did not have detailed data that comprises the HEART score nor the ability to control for individual institution-specific workflow changes, such as nurse triage processes.

We observed a decrease in protocol adherence with the change to hs-cTnT assays, when compared to cTnT, primarily through reductions in a repeat troponin within 3 h and adherence to pathway disposition. The chest pain pathway, like other clinical decision-making tools, is designed to conduct care at a high level, and deviations from provider behavior exist. Providers could use shared decision-making for protocol violations, but this is not able to be observed with our study design. Additionally, our system has a safety net with guaranteed cardiovascular specialist follow-up in these ED chest pain patients within 72 hours of disposition, which may also affect MACE and protocol violations. Despite the decrease in adherence, we did not find any difference in MACE when transitioning from cTnT to hs-cTnT.

The decision to use all-cause mortality in our MACE composite outcome was to conservatively capture all deaths during the study period. During independent MACE adjudication, we found that 8 patients in each group were included as MACE, but the cause of death was from complications of COVID-19 or other non-cardiac causes. Another limitation is that our design would fail to capture MACE or ED revisits outside of our hospital system. Nevertheless, our design had advantages of feasibility and cost-effectiveness, which provided a robust sample size. Further prospective trials that control for more confounding variables should be performed to explore these findings.

The adoption of a 1-hour hs-cTnT decision algorithm, as recommended by the European Society of Cardiology and American College of Cardiology Expert Consensus Decision Pathway, may further enhance the efficiency of AMI diagnosis and ACS rule-out. Shortening the period for repeat troponin measurements from 3-hours to 1-hour could lead to more timely diagnoses, further reducing patient LOS and optimizing resource utilization.[16,20,23] In addition, promising directions for further investigation include re-calibration of troponin thresholds within the HEART score using limit of detection or quantification instead of the 99th percentile.[24] This approach represents a viable strategy that could increase safe early ED discharges for patients presenting with chest pain, although further studies are needed before implementation. The information provided by the clinical pathway should be used as guidance, but individual circumstances should be considered when stratifying patients and determining appropriate dispositions.[3]

Conclusions

In patients presenting to the ED with chest pain, integration of hs-cTnT into the HEART score was associated with an increase in the proportion of patients discharged from the ED, decreased all-cause ED revisits, reduced LOS, decrease in CCTA utilization, without a difference in MACE, as compared with cTn. Deviations from the pathway were significant in the hs-cTnT cohort, but without a difference in MACE, shining light on the importance of the rest of the HEART score as a unified risk management tool for safe disposition. Factors such as differences in baseline characteristics and overlap with the COVID-19 pandemic could have influenced these results. Further prospective studies are needed to explore these findings.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

CRediT authorship contribution statement

Alexandra M. Cruz Pabón: Writing – original draft, Validation, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Eric Pyles:** Writing – original draft, Validation, Software, Methodology, Formal analysis, Data curation, Conceptualization. **Daniel Peach:** Writing – original draft, Visualization, Validation, Supervision, Software, Resources, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **Sarfraz Ahmad:** Writing – review & editing, Visualization, Validation, Supervision, Software, Resources, Methodology, Investigation, Formal analysis. **Paul Blake O'Brien:** Writing – review & editing, Visualization, Validation, Supervision, Resources, Project administration, Methodology, Investigation, Conceptualization. **Michael Kuhlman:** Writing – review & editing, Visualization, Validation, Software, Methodology, Investigation, Formal analysis, Data curation. **Sarah Steiner:** Writing – review & editing, Visualization, Validation, Software, Methodology, Investigation, Formal analysis, Data curation. **Lara Crown:** Writing – review & editing, Visualization, Validation, Software, Methodology, Investigation, Formal analysis, Data curation. **Elizabeth Purinton:** Writing – review & editing, Visualization, Validation, Resources, Methodology, Investigation, Formal analysis, Data curation, Conceptualization. **James Priano:** Writing – review & editing, Visualization, Validation, Software, Resources, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

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