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Review

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A Closer Look at the HEART Score

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Abstract

The history, electrocardiogram, age, risk factors, and troponin (HEART) score is currently a widely used tool for acute chest pain risk stratification. Relatively soon after its inception in 2008, a number of validation studies on the HEART score showed it to be superior to Thrombolysis in Myocardial Infarction (TIMI) and Global Registry of Acute Coronary Events (GRACE) scores and at least as accurate to other existing scores for predicting short-term major adverse cardiovascular events (MACEs). However, partly due to its focus on simplicity, the HEART score has some limitations. In this article we review how the HEART score has evolved and taken on various modifications to circumvent some of its limitations. We also highlight the strength of the HEART score in comparison with other risk stratification tools and the current guidelines.

Keywords: HEART score; Risk stratification; Modified HEART; Risk assessment; Cardiology; ACS; Acute chest pain

A Quick look Back in Time

Currently one of the most used tools for risk stratifying patients presenting to an emergency department (ED) with acute chest pain is a composite score based on five clinical considerations of history, electrocardiogram (ECG), age, risk factors, and troponin (Table 1). This now well-known risk score was first introduced by Six et al in 2008 [1]. Shortly thereafter, multiple validation studies began to emerge, including from multicenter institutions, confirming its relatively high predictive value for intermediate (6-week) major cardiovascular events [2-4]. Unsurprisingly, given the simplicity and well-suited name, the history, electrocardiogram, age, risk factors, and troponin (HEART) score has over the years become widely recognized and is at present being routinely utilized worldwide for acute chest pain risk stratification. As expected, since its original report 15 years ago, there have been countless publications relating to the HEART score. We did not intend to review all of them here, but rather to focus on its historical development and subsequent modification of the HEART score. More importantly we hope to emphasize certain practical considerations and potential pitfalls when one applies the HEART score in clinical practice.

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The original report and initial validation studies

In 2008, recognizing the lack of a practical tool for risk stratifying a large number of their patients who presented to ED with acute chest pain, Six et al first proposed their novel idea of the HEART score [1]. Utilizing an approach similar to the time-tested Apgar score (globally utilized to assess the need of a newborn for intensive care), they developed a new scoring system based on a sum of five clinical factors. In their original report, they prospectively evaluated 122 patients presenting to the ED with acute chest pain. An Access Accu Troponin I assay with cut-off $\leq$ 0.04 ng/mL was used. The primary endpoints were acute myocardial infarction (AMI), percutaneous coronary intervention (PCI), coronary artery bypass grafting (CABG) and death plus a combined endpoint of AMI, PCI, CABG, and death. The mean follow-up was, in fact, quite long at 423 $\pm$ 106 days, and only two of their subjects were lost to follow-up. One or more endpoints occurred in 24.1% of the subjects, all of them occurred within the first 3 months of enrollment. Their analysis found a very promising, almost linear, relationship between the HEART score and the endpoints [1]. Moreover, only one of the subjects with HEART score $\leq$ 3 suffered an event (2.5%) compared to 20.3% and 72.7% of those with the scores between 4 to 6 and $\geq$ 7, respectively.

Approximately 2 years later the first validation study was reported by Backus et al [2]. In this study, the performance of the HEART score was prospectively compared to Thrombolysis in Myocardial Infarction (TIMI) and Global Registry of Acute Coronary Events (GRACE) scores in 2,440 unselected patients that presented to the ED in 10 participating hospitals in The Netherlands [2]. Clinically relevant value of the HEART score as a risk stratification tool was reaffirmed. Using a score of 3 as a cut-off, 6-week major adverse cardiac events (MACEs) were observed in 1.7% of the low-risk group whereas 16.6% and 50.1% of those with the HEART scores

Table 1. Original HEART Score Components and Scoring

Components	Severity	Score
History	Highly suspicious	2
	Moderately suspicious	1
	Slightly suspicious	0
ECG	Significant ST elevation	2
	Nonspecific repolarization disturbance	1
	Normal	0
Age	> 65 years	2
	45 - 65 years	1
	< 45 years	0
Risk factors	≥ 3 risk factors/history of atherosclerotic disease	2
	1 or 2 risk factors	1
	No risk factors known	0
Troponin	> 2 × normal limit	2
	1 - 2 × normal limit	1
	≤ normal limit	0

ECG: electrocardiogram.

of 4 - 6 and ≥ 7 respectively developed subsequent MACEs. Furthermore, the performance of the HEART score was found to be superior to the TIMI and the GRACE scores (c-statistics: 0.83 vs. 0.75 vs. 0.70, respectively, P < 0.0001 for both). Similar results have also been demonstrated in two subsequent validation studies shortly afterwards [3, 4].

Meta-analyses and selective comparison studies

Since its original report, there have been many publications on predictive value of HEART score in various populations. To date, however, there have been a few meta-analyses performed on HEART score [5-7]. Van Den Berg et al reported the results of their meta-analysis on two prospective and 10 retrospective cohort studies, in which the HEART score was found to be valuable in identifying subgroups with and without subsequent MACE among patients with acute chest pain [5]. More recently Ke et al [6] also published results of their meta-analysis using pooled data from all available prospective cohort studies on predictive abilities of TIMI, HEART and GRACE scores up to June 2020. The data on HEART score from 16 studies were included in their analysis. The pooled sensitivity and specificity of the HEART score for predicting MACEs were 0.96 (95% confidence interval (CI): 0.91 - 0.98; I^2 = 94.87%) and 0.50 (95% CI: 0.41 - 0.60; I^2 = 98.84%), respectively. The pooled positive and negative likelihood ratios of the HEART score for predicting MACEs were 1.94 (95% CI: 1.61 - 2.35; I^2 = 98.01%) and 0.08 (95% CI: 0.03 - 0.17; I^2 = 94.65%), respectively. They found the pooled diagnostic odd ratio for the HEART score to be at 17.92 (95% CI: 9.40 - 34.18; P < 0.001) with significant heterogeneity across the included studies (I^2 = 88.9%; P < 0.001) and the area under the receiver operator characteristic curve of the HEART score for predicting MACEs of 0.80 (95% CI: 0.77 - 0.84). No significant publication bias for the HEART score was observed (P = 0.98). More importantly, all the studies similarly showed that HEART score accurately predicts future MACE in acute chest pain patient except one by McCord et al [7] in which the modified HEART score was used. In the latter study, however, the predictive value of HEART score was still marginally significant.

HEART score in comparison to other better known risk scores

Compared to GRACE score, HEART score has been consistently shown to have better predictive ability for future MACE than GRACE score [2-4]. Most studies also demonstrated the HEART score to be superior to TIMI score except a few showing comparable efficacy [8].

In comparison to the Emergency Department Assessment of Chest Pain Score (EDACS), the published results are non-uniform [9, 10]. Shin et al reported the HEART score to be superior to EDACS score [9]. In contrast, another study found the EDACS score to identify a larger percentage of low-risk patients than HEART score [10]. Both scores were predictive of 60-day MACE. The latter study was a retrospective comparison done in 118,822 patients that presented for acute chest pain to ED in a large Integrated Healthcare System in the United States.

Another possible exception is a newly developed score based on symptoms, vascular disease history, ECG, age and troponin (SVEAT) score [11, 12]. In a prospective single-center study of 321 subjects with acute chest pain in ED or observation unit, the SVEAT score outperformed HEART score in terms of ability to identify low-risk patients [11]. In this

Table 2. HEART Score Variants and Their Components

Variants	Components	NPV	Authors
HEAR score	History, ECG, age, risk factors	99.10%	Smith et al, 2020 [14]
HEART score pathway	HEAR score + serial troponin levels	99.60%	Mahler et al, 2015 [18]
Modified HEART score[a]	HEAR + cTnI (cut-off < 0.02 ng/mL)	99.55%	Mark et al, 2018 [10]
	HEAR + hs-cTnT (serial 4 - 14 h/delta 1 h)	99.80%	McCord et al, 2017 [7]
	HEAR + hs-cTnI	98.60%	Sajeed et al, 2020 [15]
	HEART + 0-h/1-h hs-cTnT	99.80%	Nilsson et al, 2021 [16]
	HEAR + hs-cTnI	98.90%	Ma et al, 2016 [17]
HEART-2 score	HEART + CIT	96.90%	Schrader et al, 2022 [20]
$HEARTS_3$ score	Weighted HEART score + sex + serial 2-h ECG + serial 2-h delta troponin	99.40%	Francis et al, 2012 [19]

[a]Multiple authors have quoted varying approaches to the modified HEART variant, but the common theme was the alteration of the troponin component. NPV: negative predictive value of low-risk groups. ECG: electrocardiogram; cTn: cardiac troponin I; hs-cTn: high-sensitivity cardiac troponin; CIT: cardiac imaging test.

relatively small study, 6-week MACE occurred in 1.4% of patients classified as low risk by HEART score compared to only 0.8% classified as low risk by SVEAT score. A recent small retrospective study in patients admitted to a clinical decision unit for acute chest pain evaluation from the same institution also found SVEAT score to be superior to HEART score [12]. These studies are, however, relatively small single-center studies, and thus, further validation is still needed.

More recently, in one of the largest prospective cohort studies on HEART score to date, Mark et al analyzed the net benefit of clinical decision support interface based on several different clinical risk score; HEART pathway, EDACS, troponin only, clinician gestalt and a novel risk stratification algorithm: risk stratification algorithm for acute coronary syndrome (RISTRA-ACS) [13]. A total of 13,192 adult patients encountered with chest pain in ED from January 2018 to December 2019 from 13 medical centers in their health system were included. MACEs at 60 days occurred in 3.7% of these patients. They found that the RISTRA-ACS and HEART pathway have the lowest negative likelihood ratios (0.06, 95% CI: 0.03 - 0.1 and 0.07, 95% CI: 0.04 - 0.11, respectively). They concluded that the HEART pathway is an optimal rule out approach.

HEART Score Variants

To improve upon the discriminatory power of the original HEART score or to circumvent some of its limitations, a number of variants of the HEART score have been reported over the years. Table 2 [7, 10, 14-20] summarizes the variants described here; HEAR score, modified HEART score, HEART pathway, HEART-2 score, and $HEARTS_3$ score.

The simplest variation described is the HEAR score. Smith et al proposed the HEAR score which uses the original component scoring minus the troponin [14]. Their rationale was based on previous studies that up to 46% of cardiac troponin (cTn) testing in the ED was deemed inappropriate [21]. They sought to test the HEAR score to identify a potential population that would unlikely benefit from cardiac troponin testing. The subject with a HEAR score of < 2 was classified as "very low-risk". Of the 4,979 patients studied, they found 9% (447 patients) to be in a very low-risk group. The subsequent MACE in this group was low at 0.9% (four patients).

Most variants described usually involve interpreting various cardiac troponin assay protocols. The term "modified HEART score" usually refers to these variants [7, 15, 16, 22-24]. The term has been proposed when a high-sensitivity cardiac troponin (hs-cTn) assay is used in calculating the score in place of conventional troponin by some investigators. Other investigators will also refer to it when a second troponin is measured, along with excluding ischemic ECGs and abnormal troponin values at presentation [21]. Using the latter definition of modified HEART score, 60-day MACE in those with score ≥ 4 has been reported to be much lower than the original HEART score validation studies at 2.0% (95% CI: 1.8 - 2.3). If all coronary revascularizations were included the risk increased to 4.4% (95% CI: 4.1 - 4.4).

McCord et al described using a HEAR score (denoted m-HS in their paper) plus a hs-cTnT protocol [7]. They retrospectively analyzed data from the TRAPID-AMI study to assess the combination. They considered patients low-risk if the m-HS score was < 4 with a category of hs-cTnT collection - a serial hs-cTnT of < 14 ng/L over 4 to 14 h, or a 1-h delta algorithm (hs-cTnT of < 12 ng/L at presentation with a 1-h delta of < 3 ng/mL). Roughly 40% of the studied population was found to be in the low-risk group (using either hs-cTnT category) with a 30-day MACE incident of 0.2% (using either hs-cTnT category).

Similarly, Nilsson et al assessed another variation protocol dubbed the HEART 0-h/1-h pathway [16]. This was also a combination of the HEAR score and hs-cTnI. To be considered low risk in the study, the HEAR score had to be < 4 with the hs-cTnT 0-h (at presentation) < 5 ng/L, or a 0-h < 12 ng/L with a 1-h delta < 3 ng/L. In a secondary analysis of the data from prospective observational study of 1,167 patients that visited University Hospital ED with chest pain between February 2013 and April 2014, Nilsson et al compared diagnostic accuracy of 0/1-h hs-cTnT protocol alone versus when combined with HEART pathway or ED-ACS accelerated di-

agnostic pathway or HEART score. They found the negative predictive value (NPV) for index visit AMI to be 100% for the combination of 0/1-h hs-cTnT and HEART pathway versus 99.8% and 99.2%, respectively, for combined 0/1-h hs-cTnT ED-ACS pathway and HEART score alone. Furthermore, the combined 0/1-h hs-cTnT HEART pathway identified 49.8% for rule out with 99.8% NPV for 30-day MACE whereas the HEART score alone was able to rule out 53.4% of subjects with NPV 30-day MACE of 99.2%.

Ma et al described their modified variant as using the original HEART score but substituted hs-cTnI instead of conventional troponin [17]. Beckman-Coulter, enhanced ACCU troponin I was used. The same cut-offs as the original weighting for troponin were used (Table 1). All patients in the study had their hs-cTnI tested only once for the reason that all selected patients had chest pain for at least 2 or more hours. Hs-cTnI assay can detect AMI as early as 2 h after symptom onset [25]. The authors were able to identify 6.8% of their patient population as low-risk (modified HEART score of 0 - 2) with 90-day MACE incidence of 1.1%.

Mahler et al described their modification of HEART score as the HEART pathway [18]. It is, essentially, the HEART score with a serial cTn measurement protocol. Mahler et al reported the first randomized trial comparing HEART pathway to usual care, in 282 patients that presented with symptoms related to acute coronary syndrome without ST elevation [18]. MACEs were observed in 6% of these patients at 30 days. Using the HEART pathway, 39.7% of patients were deemed appropriate for early discharge compared to 18.4% by usual care ($P < 0.001$). None of the patients identified for early discharge developed subsequent MACE. Compared with usual care (American College of Cardiology (ACC)/American Heart Association (AHA) guidelines at the time), HEART pathway reduced objective cardiac testing at 30 days from 68.8 % to 56.7% ($P = 0.048$) and length of stay by 12 h (from 21.9 to 9.9 h, $P = 0.013$).

Relatively limited data exist on the HEART pathway in comparison to other pathways or risk stratification protocols. Stopyra et al performed a comparison between the Accelerated Diagnostic Protocol (ADAPT) and the HEART pathway. It was a secondary analysis of participants enrolled and randomized to the HEART pathway arm of the HEART pathway randomized controlled trial. Each subject was then classified as low risk or high risk according to ADAPT and HEART pathway. They found both pathways had excellent sensitivity for MACE, but the HEART pathway correctly identified a higher number of low-risk patients than the ADAPT pathway (47% vs. 24%, $P < 0.001$) [26]. More recently, the HEART pathway has been compared to EDACS in a three-site cohort study [27]. In this prospective study of 5,799 patients with acute chest pain, Stopyra et al [27] found that EDACS identified significantly more low-risk patients than the HEART pathway (58.1% vs. 38.4%, $P <0.001$). MACE at 30 days occurred in 1% of those classified as low risk by EDACS. However, the MACE in those identified as low risk by the HEART pathway was lower at 0.4% ($P < 0.001$).

No Objective Testing Rule (NOTR) is not a well-known decision pathway developed from retrospectively collected data by Greenslade et al using logistic regression model [28]. The strength of NOTR is independent from subjectivity with very high sensitivity at 97.6%. NOTR, however, only identified 31.4% of low-risk patients. Stopyra et al compared the HEART pathway to NOTR in 282 subjects enrolled in the HEART pathway randomized controlled trial [29]. They found both decision rules to have 100% sensitivity for 30-day MACE but HEART pathway identified a higher proportion of low-risk patients suitable for early discharge (46.8% vs. 27.7%, $P < 0.001$).

The broadest comparison of HEART pathway to other risk stratification tools has been reported by Greenslade et al from Australia [30]. Using high-sensitivity cardiac troponin I (hs-cTnI) in 1,811 patients, who presented to ED, they assessed the performance of five different decision tools; HEART pathway, modified ADAPT, EDACS, Vancouver Chest Pain Rule and NOTR. AMI occurred in 5.3% of their cohort while 7.7% was subsequently diagnosed with acute coronary syndrome. All the decision tools have excellent sensitivity for AMI. They found that the sensitivity for acute coronary syndrome to be very high for Vancouver Chest Pain Rule and NOTR but was < 95% for the HEART pathway, modified ADAPT and EDACS. The latter three on the other hand classified more patients as low risk (64.3 %, 62.5% and 49.8%, respectively) than Vancouver Chest Pain Rule and NOTR (28.2% and 34.5%, respectively). Taken together, the EDACS appears to identify a higher proportion of low-risk patients than the HEART pathway with slightly higher miss rate while the data on relative performance between the HEART pathway and the ADAPT remain unclear.

The original HEART score was developed with "little rationale" for the weighting of the score [1]. Based on this, permutations of high score ratings for high-risk elements versus low-risk elements can produce similar overall scores. Fesmire et al aim to mitigate weaknesses in the HEART score by proposing the use of likelihood ratios (LR) for each HEART score component to enhance the scoring system with weighted values [19]. Furthermore, they desired to improve their score's discriminatory power by adding three "S" components: sex, serial 2-h echocardiogram and serial 2-h troponin. The additional "S" components are to take account of the significant age-related sex differences for coronary artery disease, as well as the advantages of incremental data from serial ECGs and troponin. They retrospectively analyzed the data of 2,206 patients with chest pain presenting to the ED. LRs were extrapolated and used to assign new weighted scores for each component. The study found that the original HEART score gave more credence to older age, number of risk factors, and presence or absence of coronary artery disease (CAD) than actuality. Conversely, the original one did not give enough credence to the history of probable ischemic chest pain, diagnostic ECG, and elevated troponin. Scoring of the new weighted system, $HEARTS_3$, ranged from 0 - 25. $HEARTS_3$ score of < 3 found 704 (32.8%) patients with a 0.6% incidence of 30-day acute coronary syndrome (ACS), versus a HEART score of < 3, which identified 458 (21.3%) patients with a 0.7% incidence of 30-day ACS. The authors admit that one of the disadvantages of $HEARTS_3$ is the cumbersome and complex nature of the weighted components.

Lastly, Schrader et al seek to improve the original HEART

score in the patient population with recurrent chest pain that have done prior cardiac imaging tests (CITs) [20]. However, they desired that the modified score would still retain its accuracy of risk stratifying ED patients in the absence of CIT. HEART2 score is therefore a combination of the original HEART score plus the additional variable of CIT. CIT includes stress tests (physical/chemical/nuclear) and heart catheterizations. Only the latest CIT results within 2 years of the index ED visit are considered. Low risk, normal, or no intervention results are interpreted as a negative CIT and assigned a score of "-1". A score of "0" is assigned if a recent negative CIT or no CIT are done within 2 years of index visit. Any positive CIT of any timeframe is assigned a score of "1". This allows the overall modified score, HEART2, to have a range of -1 to 11. HEART2 outperformed HEART according to their area under the receiver operator curve (ROC) curve analysis (0.74 versus 0.71, respectively). The low-risk group for both scores were considered as a score of less than or equal to 3. HEART2 detected 55.5% of the population as low risk with an incidence of 30-day MACE, in this group, of 3.1%. Comparatively, the original score detected 38.2% as low risk with a 30-day MACE incidence of 2.2%. In essence, the HEART2 score detected more low-risk patients in a population of recurrent chest pain patients with previous CIT that could be discharged from the ED, with no significant difference in 30-day MACE incidence compared to the original score. The overall aim of HEART2 is to reduce hospitalizations for a subgroup of patients that would normally be admitted due to their recurrent chest pain and history of CIT, especially if their recent CIT was negative. This would further support efficient allocation of healthcare resources.

Practical Consideration and Potential Pitfalls

Conventional vs. hs-cTn assay

In the original study and most of the earlier studies on the HEART score, including the validating studies, conventional troponin assays were used [1-4]. Over the years, the performance of troponin assays has substantially improved. Compared to the troponin assay used in the original study reported by Six et al [1], the fifth-generation ultra-high sensitivity troponin assay currently used is up to 100-fold more sensitive [15]. With an advancement in cTn assay technology, more recent studies usually utilized high-sensitivity cardiac troponin-T assay (hs-cTnT) and hs-cTnI assay to help improve discriminatory power of the HEART score. With a different cut-off for "normal range", some patients with troponin in the lower end of normal range could be assigned a different point on HEART score depending on which assay was used. Consequently, some of the subjects previously classified as low risk may fall into a higher-risk category potentially impacting predictive ability of HEART score for future MACE.

Wassie et al recently reported a retrospective cohort study of 27,918 patients encountered across 15 community EDs within integrated healthcare systems in Southern California between May 5, 2016, to December 1, 2017 [31]. All of them were evaluated with HEART score and the same conventional cardiac troponin I assay (Access AccuTnI+3 assay; Beckman-Coulter). The lowest level of detection of this TnI assay is 0.02 ng/mL. They found a very low 30-day MACE in their patients with TnI below the level of detection (< 0.02 ng/mL) at 0.4% [31].

Gibbs et al recently reported a multicenter study evaluating utility of several risk scores including HEART score using high sensitivity cardiac troponin among 2,505 patients with suspected AMI from 29 hospitals in the United States between April 2015 to April 2016 [32]. In this study, Atelica IM TnIH Assay (Siemens Healthineers) with 99th percentile of 45 ng/L was used. At 30 days, 12.1% and 1% of the patients experienced myocardial infarction (MI) or death, respectively. Revascularization was required in 9.1% of their subjects.

Introduction of more sensitive troponin assay has been shown to increase the rate of coronary angiography [22, 23] and PCI but did not have significant impact on MACE [17]. As mentioned above, one of the limitations of HEART score is its suboptimal ability to detect most low-risk patients. Incorporating an ultra-high sensitivity troponin assay into practice and as a component of the HEART score could potentially have a negative impact on the HEART score being a useful risk stratification tool to identifying patients for early discharge. How to appropriately assign a score pertaining to troponin level may therefore need to be better clarified.

Low-risk is "not" no risk

Patients presenting with acute chest pain whose HEART score ≤ 3 are generally considered low risk and in most circumstances can be discharged from ED for outpatient evaluation. In the original report, MACE occurred in 2.5% of the subjects with score ≤ 3 [1]. MACE was observed in 1.7% of those classified as low risk in the first large validation study reported by Backus et al [2]. In a recent meta-analysis of all the studies published from its inception to May 2018, by Fernando et al, which included 44,202 patients, showed pooled sensitivity and specificity of the HEART score ≤ 3 for predicting MACEs to be 95.9% (95% CI: 93.3 - 97.5) and 44.6% (95% CI: 38.8-50.5%), respectively [33]. A similar more recent meta-analysis of 25,266 patients by Laureano-Phillips et al found an equivalent degree of predictive performance of the HEART score with pooled sensitivity and specificity at 0.96 and 0.42, respectively [34]. In the latter report, however, the MACE rate was slightly lower at 2.1% especially when hs-cTn was used or when a modified HEART score was applied among the North American population. Furthermore, a recent report in subjects who underwent coronary computed tomographic angiography and the presence of ≥ 50% was included in the primary endpoint, the false negative rate of HEART score is found to be relatively high at 11% with modest predictive accuracy area under the ROC curve (AUC) of 0.79 [35].

Most low-risk patients are misclassified

The other weakness of HEART score is its suboptimal ability

to identify low-risk subjects. Like most other acute chest pain risk stratification scores, the HEART score fails to identify a substantial number of low-risk patients [2-4, 11]. In the majority of the studies, HEART score ≤ 3 and essentially all of its variants correctly identify less than half of the low-risk group. Recent studies have suggested that our ability to detect this important subgroup could be improved by utilizing risk score that incorporates more of the routinely obtained, valuable clinical information [11, 12].

Scoring consistency and inter-observer agreement

In addition to its proven performance, the practicality of HEART score is likely one of the reasons for its worldwide acceptance. On the other hand, the developers' intention to keep the criteria simple may have potentially resulted in interpreter's biases. For example, the distinction between nonspecific and ischemic ST changes on ECG is not clearly elucidated (Table 1), and hence, is subjected to an observer's interpretation. Moreover, despite well-established characteristics of typical angina, previous studies have shown wide disagreement between interpreters [29] in clinical practice.

Parenti et al recently reported a multi-center study on inter-rater reliability of HEART score [36]. In this interesting study, 20 participating ED physicians undertook a similar course on HEART score to minimize potential variability. They were then randomly presented with various selected clinical scenarios and asked how they would score those subjects. The investigators found a good inter-rater agreement for high- and low-risk classes (HEART scores of ≥ 7 or ≤ 3; kappa 0.70 and 0.72, respectively), whereas, they observed moderate agreement among intermediate risk groups (kappa 0.51) [36]. Among the different items, as expected, history and ECG had the worst agreement (kappa T 0.37 and 0.42, respectively) [36].

In contrast, more real-world studies have shown only moderate inter-observer agreement [37, 38]. Soares III et al compared the score calculated by ED physicians and researchers who independently interviewed the subjects and found the dichotomized HEART score agreement to be at 78% (kappa 0.48, 95% CI: 0.37 - 0.58). Like a study by Parenti et al, the lowest agreement was observed in the history (72%), followed by the ECG at 85% [36]. Due to their concern on the degree of inter-observer scoring discordance, van Meerten et al recommended against usage of HEART score by ambulance nurse in the prehospital setting [37]. Furthermore, an alarming finding of 70% discordance of HEART score between ED physicians and cardiologists has been recently reported by Wu et al [39]. The latter study was, however, a small retrospective, single-center study that included only 33 subjects. Again, like previous reports, discrepancies in chest pain description are the most common issue. Arguably, among individuals in the upper score range, 1 - 2 points differences are unlikely to have significant effect on their risk classification. In contrast, for those on the opposite range of the point score values, with score of 3 being the cut-off for low risk, 2 points reallocations would have reclassified a number of lower-risk patients into the intermediate group category.

Professional Organization/Society Guideline and Recommendations

The 2020 European Society of Cardiology (ESC) guideline for acute coronary syndrome recommends using a 0/1-h hs-cTn in combination with clinical assessment [40]. The ESC prefers GRACE score for prognostic purposes but does not specifically favor any initial risk stratification scoring system or pathway for initial diagnostic purposes.

Chapman et al found that combining the low-risk HEART score (< 3) with the ESC 0/3-h hs-cTn protocol yielded an increase in NPV but reduced the low-risk population. On the other hand, when HEART score was combined with the hs-cTn in the evaluation of patients with ACS (High-STEACS) pathway, there was no change to NPV; however, it still reduced the low-risk population [41]. Somewhat less robust results have been reported in another recent prospective multisite US cohort [42]. In this study, the investigators evaluated diagnostic performance of an initial hs-cTnT below the limit of quantification (6 ng/L), 0/1-h hs-cTn algorithm, and their combination with HEART score in 1,462 participants. They found that adding low HEART score to 0/1-h hs-cTn algorithm ruled out 30.8% of the subjects with a NPV of 98.4% for 30-day MACE. Interestingly, the combination of only initial troponin (Tn) below the limit of quantification alone and low HEART score resulted in a NPV of 99%.

2021 Guidelines for the Evaluation and Diagnosis of Chest Pain by joint committee from several organizations and societies, including American College of Cardiology, and American Heart Association, recommend using one of five clinical decision pathways; HEART pathway, EDACS, ADAPT, modified ADAPT and NOTR [43]. A study from the Brigham and Woman's Hospital, and Wake Forest School of Medicine, unfortunately, found that compliance with standardized clinical assessment tools is poor [44]. Serial troponins were not followed per protocol in 56% of low-risk patients and stress testing was performed against the recommendation.

Conclusions

Perhaps, as Leonardo Da Vinci stated, "Simplicity is the ultimate sophistication". Among the multitudes of available risk stratification tools for acute chest pain, since its original inception in 2008, the HEART score has become one of the most, if not the most, commonly utilized tool worldwide. Over the past 15 years, not unexpectedly, a few modifications and variants have been reported. It is not perfect, but healthcare providers who routinely apply the HEART score into their clinical practice should be familiar with its strengths, and as importantly, its pitfalls and limitations.

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None to declare.

Conflict of Interest

There is no conflict of interest.

Author Contributions

Dr. Chanwit Roongsritong and Dr. Sammy Aung both contributed to gathering references, creating tables, and writing each section to bring to fruition the information presented in the article.

Data Availability

The authors declare that data supporting the findings of this study are available within the article.

Abbreviations

ACC: American College of Cardiology; ADAPT: Accelerated Diagnostic Protocol; AHA: American Heart Association; AMI: acute myocardial infarction; APGAR: appearance, pulse, grimace, activity, and respiration; CABG: coronary artery bypass grafting; CIT: cardiac imaging test; ECG: electrocardiogram; ED: emergency department; EDACS: Emergency Department Assessment of Chest Pain Score; ESC: European Society of Cardiology; GRACE: Global Registry of Acute Coronary Events; HEART: history, electrocardiogram, age, risk factors, and troponin; High-STEACS: high-sensitivity troponin in the evaluation of patients with ACS; hs-cTn: high-sensitivity cardiac troponin; LR: likelihood ratio; MACEs: major adverse cardiac events; NOTR: No Objective Testing Rule; NPV: negative predictive value; PCI: percutaneous coronary intervention; RISTRA-ACS: risk stratification algorithm for acute coronary syndrome; ROC: receiver operator curve; SVEAT: symptoms, vascular disease history, electrocardiogram, age, and troponin; TIMI: Thrombolysis in Myocardial Infarction

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