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LETTER TO THE EDITOR

New scoring system for acute chest pain risk stratification: Is it worth SVEAT-ing it?

Mahati Dasari, Pramukh Arun Kumar, Yuvaraj Singh, Eddison Ramsaran

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Abstract

The emergency room is a very potent environment in the hospital. With the growing demands of the population, improved accessibility to health resources, and the onslaught of the triple pandemic, it is extremely crucial to triage patients at presentation. In the spectrum of complaints, chest pain is the commonest. Despite it being a daily ailment, chest pain brings concern to every physician at first. Chest pain could span from acute coronary syndrome, pulmonary embolism, and aortic dissection (all potentially fatal) to reflux, zoster, or musculoskeletal causes that do not need rapid interventions. We often employ scoring systems such as GRACE/PURSUIT/TIMI to assist in clinical decision-making. Over the years, the HEART score became a popular and effective tool for predicting the risk of 30-d major adverse cardiovascular events. Recently, a new scoring system called SVEAT was developed and compared to the HEART score. We have attempted to summarize how these scoring systems differ and their generalizability. With an increasing number of scoring systems being introduced, one must also prevent anchorage bias; *i.e.*, tools such as these are only diagnosis-specific and not organ-specific, and other emergent differential diagnoses must also be kept in mind before discharging the patient home without additional workup.

Key Words: Chest pain; Acute coronary syndrome; SVEAT score; HEART score; TIMI score; Risk stratification scores

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Core Tip: Despite several studies, scoring systems, and artificial intelligence -guided tools available to triage symptoms of chest pain, physicians are often struck with the dilemma before discharging patients from the endoplasmic reticulum. The reason is that chest pain etiologies such as acute coronary syndromes (ACS) can present atypically and, when misdiagnosed, can lead to catastrophic consequences. Tools such as the HEART score and recently published SVEAT score are robustly validated methods of triaging this conundrum. However, while we delineate how they differ, one must be mindful that most patients with ACS could present with chest pain, but not every chest pain is due to ACS.

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TO THE EDITOR

We read with great interest the retrospective cohort study by Antwi-Amoabeng *et al*[1] entitled "SVEAT score outperforms HEART score in patients admitted to a chest pain observation unit." It is a well-written study that validated that the performance of the Symptoms, history of Vascular disease, Electrocardiography, Age, and Troponin (SVEAT) score is superior as compared to History, Electrocardiography, Age, Risk factors, and Troponin (HEART) score in stratifying acute chest pain in low to intermediate risk patients for 30-d major adverse cardiovascular events (MACE). The study assessed the potential usefulness of the SVEAT score developed by Roongsritong *et al*[2] in a prospective observational study by comparing it with HEART and TIMI (Thrombolysis In Myocardial Infarction) risk scores.

Acute chest pain is the second most common reason for adults presenting to the emergency department after trauma, of which only 5.1% of cases are caused by acute coronary syndrome (ACS)[3, 4]. Patients with ACS symptoms with less than a 1% probability of 30-d MACE or death are classified as low-risk chest pain[5]. High-sensitivity troponins are used to diagnose myocardial infarction and detect myocardial injury[6].

Before 2008, widely used risk scores for ACS like GRACE, PURSUIT, and TIMI mainly focused on high-risk patients[7-10]. In 2008, Six *et al*[11] developed the HEART score in a single-centric study to better guide ER physicians to triage acute chest pain in low-risk patients aiding in safe early discharge, which was further validated by Backus *et al*[12] in a multicentric study stating that low HEART scores had a low likelihood of an ACS and high HEART score predicted higher MACE in 6 wk. Of the currently available risk stratification scores commonly used, the HEART score clinical decision pathway is the most widely employed[13]. Head-to-head comparison studies between GRACE, TIMI, and HEART scores showed that HEART scores had better predictability of MACE in low-risk patients[14]. It is also proven to reduce objective cardiac testing in 30 d, reduce the length of hospital stay and increase early discharges compared to usual care as per ACC/AHA[15].

However, the HEART score includes traditional cardiac risks factors, such as hypertension, diabetes, smoking, and obesity, which have limited value in diagnosing ACS, especially in patients older than 40 [16]; hence, these have been eliminated from the SVEAT score. Instead, the history of vascular events was included in the SVEAT score, as shown in Table 1[2]. Using more objective data in the SVEAT score reduces uncertainty and inter-rater variability inherent to other scores caused by arbitrary, subjective criteria. In addition, as stated by the authors, the SVEAT score incorporates more points for factors with higher risk association and negative points for factors with a lower risk associated with acute coronary events, ranging from +5 to -2. This, in turn, provides a broader range of cumulative scores, helping achieve superior stratifications between subgroups.

The HEART score has a threshold of 3 for stratifying as low risk, while the SVEAT score of 4 was chosen as a cut-off for low risk to achieve a 30-d MACE of 0.8%, calculated retrospectively in the index article. The HEART score identified less than 60% of the low-risk patients, whereas an additional 28% of low-risk patients were identified using the SVEAT score as compared to the HEART score[2,11,12]. Moreover, the HEART score allocates the highest score of '2' for troponins, while the cut-off for low-risk stratification is 3. Hence, with the HEART score, there is a disclaimer that if there is positive high sensitivity troponin despite the score being less than or equal to 3, *i.e.*, low-risk score, experts recommend further workup and admission[13]. However, a score of '5' with the SVEAT score system is allocated if the troponin level is over 0.7 ng/mL. This, by default, ensures that the patient is not in the low-risk group if troponin is significantly elevated. Also, vascular disease has one of the most quantifiable associations with cardiac mortality, which was given a higher individual score in the SVEAT score system[17].

Table 1 Summarizing differences between the HEART and SVEAT scores			
Scoring variables	HEART score	SVEAT score	
Symptom- Chest pain	Stratifies symptoms subjectively, <i>i.e.</i> , based on level suspicion. (This is open to bias based on the provider)	Stratifies symptoms more objectively by using well-defined terminologies for chest pain, hence being less open to bias	
Risk factor	Includes hyperlipidemia, hypertension, diabetes mellitus, smoking, and a family history of obesity, and scoring is based on their frequency. Does not take recent coronary disease into account	Includes recent myocardial infarction, PCI/CABG, or any prior vascular event	
EKG	Positively scores any EKG changes. If none are present, score 0. No negative scores	Gives a score of 3 for dynamic ST or T wave changes, higher than HEART (2). It also assigns a negative score when there are no EKG changes in the presence of ongoing chest pain	
Age	Assigns a score of 2 for all patients over 65 yr	Assigns a score of 2 for all patients over 75 yr. It also assigns a negative score when the patient is < 30 yr	
Troponin	Is applicable for both Troponin I and T assays. No negative scores for a normal Troponin	Validated for the 4 th generation ultra-sensitive Troponin I assay only. Assigns negative scores for normal Troponin levels after > 4 h of chest pain	

CABG: Coronary artery bypass grafting; EKG: Electrocardiographic; PCI: Percutaneous interventions; ST: Subthemes; SVEAT: Symptoms, history of Vascular disease, Electrocardiography, Age, and Troponin; HEART: History, Electrocardiography, Age, Risk factors and Troponin.

> Both scores examine the risk stratifying of patients presenting with chest pain due to coronary artery disease. They do not consider other life-threatening illnesses in patients with chest pain, such as aortic dissection, pulmonary embolism, or esophageal rupture. This is important to note since chest pain can cause anchorage bias, and a low score can create a false sense of security, leading to premature discharge. While promising, the SVEAT score has several limitations, as mentioned by the authors, including the need for further validation in multicentric studies with diverse populations. Additionally, we need comprehensive follow-up data regarding prognostication for a longer duration. As the authors state, the individual scores are assigned rather arbitrarily than using a more formally weighted logistic regression model. Further studies could also be performed to validate if a combination of scores can increase reliability and precision in identifying low-risk patients with acute chest pain.

> Acute chest pain etiology also differs in a gender-specific manner, with conditions such as coronary artery spasm, subacute coronary artery dissection, and takotsubo being significantly more prevalent in women, unlike obstructive CAD, which is more prevalent in men[18-21]. However, given the absence of conventional risk factors and ECG changes in the conditions mentioned above, screening and specific stratification remain challenging with any available scoring system, including the SVEAT system.

> In conclusion, we would like to reiterate that using a well-validated scoring system is crucial to educate patients about chest pain, its implications, and key management measures. Before discharging someone with a low HEART or SVEAT score, patients must be asked if they live alone, have access to phones, are ambulatory, and how far they are from a tertiary medical facility. If these resources are unavailable, the patient should be considered a non-low risk and admitted to the hospital for further workup.

FOOTNOTES

Author contributions: Dasari M conceptualized the idea and designed the research; Dasari M and Arun Kumar P wrote initial draft of manuscript; Singh Y and Ramsaran E proof-read and suggested changes in manuscript, Singh Y checked for scientific accuracy, plagiarism and table creation; Dasari M, Arun Kumar P, Singh Y, Ramsaran E made further edits and reviewed the final version of the manuscript.

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