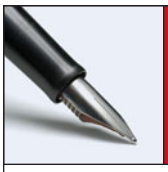

No Troponin, No Problem: Reimagining Chest Pain Assessment in Urgent Care

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Most urgent care providers loathe when a patient checks in with chest pain because, typically, they are presenting because they're worried about a heart attack, and *we're* worried we don't have the tools to exclude this diagnosis. It's no surprise that we're met with consternation when we suggest they may have come to the wrong place for care. But is unavailability of troponin testing a worthy scapegoat? And is the practice of ED referral for nearly every patient with chest pain appropriate?

We propose we reevaluate the typical approach to chest pain in UC.

Chest Pain Is Common, but MI Is Rare in Urgent Care

Chest pain is concerning to patients predominantly due to the possibility of myocardial infarction (MI), which represents between 1% and 3% of ambulatory visits for acute complaints.[1] While this is a small proportion of overall visits, it means we will see patients like this nearly every shift.

The vast majority of patients seeking care for acute chest pain aren't having a heart attack. In fact, only about 10%–12% of patients presenting to an ED with concerns for acute coronary syndrome (ACS) will go on to have a major adverse cardiac event (MACE), within the subsequent 30 days.[2,3] Rates of immediate ACS in ED populations are even lower (5%–10%).[4]

Frequency of short-term MACE and immediate ACS have not been specifically studied in U.S. urgent care populations, but are likely significantly less than those observed in the ED. The best estimate from the recent literature which can be extrapolated to UC comes from a European study of acute primary care visits, where the investigators found the 6-week risk of MACE to be <5%.[1]

Most Studies of Chest Pain Measure the Wrong Outcomes

Immediate risk of ACS and what to do with the patient in front of us reporting chest symptoms is our primary concern in UC. Unfortunately, most studies reporting outcomes of patients with acute chest pain fail to be directly relevant for the UC clinician not only because they're ED-based, but also because they report MACE over the subsequent weeks as the primary endpoint.

The concept of MACE was developed in the late 1990s by cardiologists as a composite endpoint for measuring outcomes after coronary interventions (PCI).[5] Patients are classified as having a MACE if they die, have an MI, or have a repeat PCI during some specified period of time, usually 4-6 weeks. While convenient for statistical analysis, these composite endpoints are difficult to interpret, as death and "needing to have a procedure" are far from equivalent outcomes. However, research using MACE counts these events equally.

A second problem is that the timeline for cardiac events in many studies is not relevant to our predicament.[6] We seek to know the near-term safety of the patient, ie, will they drop dead before they can make it to the ED if the chest pain comes back? If we knew they could make it to outpatient follow-up, we'd feel much more comfortable foregoing an immediate referral.

Unfortunately, a trend among many studies examining the various ACS clinical decision rules (CDR) is that they look at MACE over a longer time period (usually around 1 month) than is relevant

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to UC providers.[6] This is problematic because immediate risk for sudden death or serious MI in UC patients has not been specifically studied. In other words, it's certainly lower, but we really can't say how *much* lower without UC-specific data.

Clinicians Do Not Tolerate Uncertainty with ACS

A recent study by Samuels, et al found that half of 126 emergency providers of varying roles were uncomfortable with missing an acute myocardial infarction (MI) even 0.1% of the time.[7] Even though the American College of Emergency Physicians has stated that an ACS miss rate of 1% to 2% is acceptable—perhaps even unavoidable—acute care providers continue to approach patients with chest pain with an overabundance of caution. The rationale for this is related to fear of litigation, which is a valid concern as "failure to diagnose" MI remains a leading cause of U.S. malpractice claims.[8] But if we could say with confidence that there's a less than 2% chance of MI, we'd be well protected by the ACEP policy and the current stream of excessive ED referrals, testing, and admissions could be significantly mitigated. Over the past decade, several CDRs have been developed to address this very conundrum, with the HEART score being the most prominent and well validated. But there's a catch.

Most ACS Prediction Tools Don't Work in Urgent Care

Outpatient risk stratification tools for patients presenting with chest pain have been sought after for several decades. This is because clinician gestalt has been proven unreliable consistently in ruling out cardiac etiologies of chest symptoms. The aim of these CDRs was to take provider subjectivity out of the calculation; however, none have really met the needs of the UC clinician.

A number of these rules (eg, Marburg, Gencer, INTERCHEST) were developed for use in primary care. While it is helpful that these rules do not require troponin testing (or even an EKG), they were designed to predict whether patients' symptoms are due to coronary artery disease (CAD), not ACS. These tools not only fail to address the question we're trying to answer in UC, they also don't do an adequate job of even answering the question they were developed for (ie, CAD or not), with sensitivities ranging from 81% to 88%.[1]

The Bruins Slot rule is a unique tool developed with the aim ruling out ACS (rather than CAD) in an ambulatory setting without an EKG or troponin. While promising in concept, its real-world performance falls short of holy grail status with a sensitivity of ~90%.[1]

For ED patients, on the other hand, the recent development of the HEART and EDACS scores has proven to be highly useful in identifying a large proportion of patients presenting with concerns for ACS who can safely be discharged without further immediate work-up. These tools, especially the HEART score, have been widely adopted by emergency clinicians who now can discharge many more patients with chest pain and still sleep well at night.[9] The catch: these tools all require serum troponin testing, which is only available in <10% of U.S. urgent care centers.

A HEAR(-T) Score for the Rest of Us

The HEART score, first developed in 2008,[10] is a clever acronym which combines 1) history, 2) EKG findings, 3) age, 4) CAD risk factors, and 5) troponin values to categorize patients as low, moderate, or high risk for ACS. It has been validated by multiple investigators and found to be a reliable means of risk stratifying patients with chest pain for risk of MACE over the subsequent weeks, with a sensitivity >98% for low HEART score patients.[11]

However, the necessity of troponin testing for the calculation of a HEART score has left UC providers feeling somewhat appropriately resigned to continue the status quo practice of near-automatic ED referrals for all but the lowest risk patients (read: anxious adolescents). This has resulted in an abundance of low-risk ED referrals with an accompanying line in the chart: "Cannot r/o ACS without troponin." But do we actually need a troponin to exclude ACS in low-risk patients with chest pain?

While the HEART score may be the most well-known clinical decision tool for chest pain presentations, its lesser-known cousin the "HEAR" or "HEAR(-T)" score has been validated with promising results. It seems the dogma of mandatory troponin testing when considering ACS may not be as ironclad as we've thought—especially for the very low-risk patients.

In 2020, Smith, et al first described the use of a HEART score without troponin testing applied retrospectively to over 4,000 ED patients from the original HEART score study population.[2] They found that a HEAR score of 0 or 1 occurred in 9% of patients and was 97.8% sensitive for ruling out 30-day MACE in this population. As ACEP has codified the 2% acceptable miss rate for ACS, this sensitivity almost exactly meets the minimum necessary for an acceptable "test" to be clinically useful in this situation. (Interestingly, the addition of a single troponin in this study did not improve the sensitivity of the rule either.)

More recently, O'Reilly and colleagues published the results of an external validation of the HEAR score.[12] They performed a secondary analysis of data collected in a prospective cohort study of 820 patients presenting in an urban Canadian ED with symptoms concerning for ACS. Improving on the clinical utility of the original HEAR study, they included patients with known CAD (who were excluded from the initial study) and used both 30-day MACE and immediate risk of MI diagnosed within 24 hours of ED presentation as co-primary endpoints. Importantly, patients with ischemic changes or new arrhythmia on EKG, advanced renal failure, MI within the prior month, and those under 25 years of age were excluded.

They found that nearly 25% of patients had a HEAR score of 0 or 1. Confirming that low-risk patients are indeed low risk for bad near-term outcomes, only one patient in the low-risk group (score of 0 or 1) had an MI or 30-day MACE event. This

yielded a sensitivity of HEAR <2 for 30-day risk of MACE or immediate MI of 98.3- 99.2%. Better yet, *for patients with a HEAR score of 0, the sensitivity was 100%.*

This study did not receive nearly the fanfare as the original HEART score studies among the EM community because troponin testing for chest pain patients in the ED is literally automatic. However, the authors failed to mention the potential utility of this decision rule for UC clinicians who don't have instant troponin testing.

Given that UC centers tend to see younger, healthier, lower acuity patients with chest pain compared to the ED population, it's likely that an even greater proportion of UC patients will actually fall into this low-risk (ie, score 0 or 1) group. This means that by applying the HEAR rule there is now an evidence base for discharging low-risk patients directly from UC. Coupled with the support of ACEP's clinical policy on acceptable ACS miss rates, UC providers should feel confident that this is a reasonable practice. Plus, this approach will be preferred by nearly every low-risk patient you see.

Cautions in Applying the HEAR Score

If this is your first introduction to the HEAR score, hopefully you're feeling more enthusiastic than skeptical at this point. For the enthusiasts, however, it is important to remember the limitations of CDRs in clinical practice.

First, CDRs, including the HEAR score, are developed to exclude conditions, rather than to make diagnoses.[13] Patients with HEAR scores of 0 or 1 can be safely presumed to be low enough risk for discharge from UC without immediate ED referral, but patients with scores >1 do not necessarily warrant immediate 911 activation. It is just not appropriate to use the HEAR score to justify your disposition decision in such patients. In other words, a "negative" HEAR score is meaningful but, a "positive" result is not. In fact, the specificity of a score >1 for one of the adverse cardiac outcomes was an unimpressive 19%–26% in the O'Reilly validation study.[12]

Secondly, a CDR can only be applied validly to the same type of patients as those who were included in the studies from which it was derived. For example, patients under 25 years and with end-stage renal disease were excluded in the HEAR validation study. Therefore, the rule can't be relied upon in these patients unless a subsequent study produces similar results and does not exclude these patients.

A New Approach When Considering ACS in UC

Hopefully at this point, you're reconsidering the "business as usual approach" to UC patients with chest pain. Although most patients with chest pain who present to UC are exceptionally low risk for ACS (and even more so for sudden cardiac death), providers are extremely intolerant of missing an MI. A recent ACEP policy statement, however, provides top cover for an approach to evaluation for ACS that results in a miss rate <2%.[14]

While the original HEART score is inaccessible to most UC clinicians due to lack of troponin testing, the ability to obtain an EKG is nearly universal. So, when patients present to your UC center with chest pain or symptoms that create concerns for ACS, they can be approached initially in the standard fashion: rapid rooming, vitals, and EKG. If the patient has a STEMI or other clear signs of ischemia, 911 activation is appropriate. However, this is rarely the case. For the vast majority of patients, the EKG will be reassuring and you'll be able to take some time to look up and apply the HEAR score.

With a reassuring history and EKG, a large proportion of patients can safely be ruled out for immediate and 30-day MACE (provided the HEAR is score <2).

For the rest of the patients, we can continue to use our clinical gestalt, appreciating its shortcomings, as well as shared decision-making regarding the necessity of immediate vs PRN ED referral and 911 activation.

Applying this strategy in chest pain management rather than quickly dismissing patients due to lack of troponin testing will be appreciated by your patients, who certainly want to avoid the ED if possible. Most importantly, it will achieve this in an evidence-based fashion—avoiding bad outcomes not only for our patients, but for ourselves as well. ■

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