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Evidence-Based Urgent Care

High-Yield Clinical Education • Practical Application

CLINICAL CHALLENGES:

- **What is the role** of the urgent care clinician in the evaluation, management, and disposition of patients with chest pain?
- **Which history and physical examination findings** are most important in determining risk for ACS in the urgent care setting?
- **Are clinical decision-making tools** for ACS risk appropriate for use in settings that do not have access to troponin testing?

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Prior to beginning this activity, see "CME Information" on page 2.

Identifying Urgent Care Patients With Chest Pain Who Are at Low Risk for Acute Coronary Syndromes

■ Abstract

As the popularity and accessibility of urgent care centers have expanded, patients frequently present to urgent care without a working knowledge of the center's clinical capabilities. Between 75% and 90% of patients seeking medical care for chest pain are not experiencing acute coronary syndromes, but it is imperative to quickly identify patients with true acute coronary syndromes and immediately disposition them to a higher level of care. Clinicians can avoid overtriage by using clinical findings and decision-making tools to identify patients with low-risk chest pain who can be safely evaluated in urgent care. This article reviews recommendations for evidence-based risk stratification of chest pain using decision-making tools that are appropriate for outpatient settings.

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Target Audience: This internet enduring material is designed for physicians, physician assistants, nurse practitioners, and residents in the urgent care and family practice settings.

Goals: Upon completion of this activity, you should be able to: (1) identify areas in practice that require modification to be consistent with current evidence in order to improve competence and performance; (2) develop strategies to accurately diagnose and treat both common and critical urgent care presentations; and (3) demonstrate informed medical decision-making based on the strongest clinical evidence.

CME Objectives: Upon completion of this activity, you should be able to: (1) identify patients who are at low risk of acute coronary syndromes and short-term major adverse cardiac outcomes; and (2) effectively apply clinical risk scores for better risk stratification of patients with chest pain in the urgent care setting, including when troponin testing is not available.

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Points & Pearls

NOVEMBER 2022 | VOLUME 1 | ISSUE 8

Identifying Urgent Care Patients With Chest Pain Who Are at Low Risk for Acute Coronary Syndromes

Points

- History cannot reliably rule in or rule out ACS.
- Features that indicate a higher likelihood of ACS include pain radiating to both arms or shoulders, pain similar to prior ischemia, exertional pain, pain associated with diaphoresis, and a change in pain pattern over the past 24 hours.
- Features that indicate a lower likelihood of ACS include pain described as pleuritic, positional, reproducible with palpation, sharp/stabbing, or nonexertional.
- Females, older adults, and patients with diabetes are more likely to present with atypical symptoms of ACS, such as nausea, dyspnea, lack of pain, or pain outside of the chest.
- In patients with chest pain, the physical examination is often normal, but is still useful in assessing overall hemodynamic function and uncovering alternative diagnoses.
- Physical examination features that may indicate a higher likelihood of ACS include hypotension, new mitral regurgitation murmur, and third heart sound.
- Clinicians must be proficient at recognizing ST segment changes, and understand that, when missed, subtle or early changes could have devastating consequences.
- Conventional troponin assays can detect myocardial infarction within 3 hours of ED arrival in most patients; they have excellent sensitivity but poor specificity for myocardial infarction. POC troponin testing is not available in most UC settings. It is important to remember that when they are used, these POC tests are not as sensitive as troponin assays performed in dedicated laboratories.

Pearls

- The majority of patients who present with chest pain do not have a cardiac etiology. The percentage of chest pain patients who are ultimately found to have ACS varies from 1.5% in primary care settings to up to 25% of ED patients.
- The 2 recommended scores for the outpatient evaluation of undifferentiated chest pain to identify low-risk cardiac pain are the Marburg Heart Score and the HEAR Score. Low risk scores alone should not determine medical decision making, but should be used along with the history and clinical assessment.
- For patients with potential ACS, an ECG should be obtained within 10 minutes of arrival. STEMI is defined as ST elevation at the J point of ≥1 mm (0.1 mV) in ≥2 contiguous leads (except in leads V2-V3 where ST elevation can be up to 1.5 mm in women, 2 mm in men aged ≥40 years, and 2.5 mm in men aged <40 years).
- ACS is one of UC medicine's "don't miss" diagnoses. All patients with chest pain should be quickly identified by staff so that a rapid initial assessment can be performed (including targeted history, physical examination, and ECG), and a risk stratification tool such as the Marburg Heart Score or the HEAR Score can be utilized.

Case Presentations

CASE 1

A 65-year-old man presents to urgent care after he experienced a 20-minute episode of dull, aching, left-sided chest discomfort...

- The pain began while he was doing yard work an hour ago. His wife reports that he has been having similar episodes on and off for the past 2 weeks.
- He is pain free on arrival, and his vital signs are unremarkable. He has a history of hypertension, diabetes, and prior myocardial infarction.
- His ECG and chest x-ray are normal.
- When you go back into the room to reassess him, he says he feels fine now and asks if he can leave. You hesitate, considering whether it is safe to send him home…

CASE 2

A 22-year-old man arrives at your urgent care with sharp, left-sided chest pain and shortness of breath...

- He states that he is concerned that he's having a heart attack.
- He recently returned from a spring break trip to Mexico. He reports that he had symptoms of an upper respiratory infection shortly after the trip.
- He says that he feels that his chest pain is worse when he is lying flat.
- His temperature is 37°C, blood pressure is 124/80 mm Hg, pulse is 115 beats/min, respiratory rate is 18 breaths/min, and pulse oximetry is 98% on room air.
- The physical examination is unremarkable, with no reproducible chest wall tenderness. He has no past medical history, no cardiac risk factors, and no family history of heart disease. His triage ECG and a chest x-ray are normal.
- ACS seems unlikely, but as you think through your differential diagnosis, you wonder if any other tests are needed to rule it out definitively…

CASE 3

A 20-year-old woman comes to urgent care complaining of chest pain that occurs with certain movements and when she takes a deep breath...

- She is a college student and has been preparing for final exams. She says she has been consuming energy drinks so she can stay up late to study.
- She denies injury but recalls that she first noticed the pain when she awoke after falling asleep in an awkward position in a chair in the student lounge. Her pain is reproducible with palpation.
- Her vital signs, ECG, and assessment are normal. She denies palpitations.
- She has no past medical history, no cardiac risk factors, and no family history of heart disease. She is not taking any exogenous estrogens.
- You wonder if any additional testing is needed to assess this young woman's chest pain…

Introduction

Each year in the United States, there are approximately 8 million emergency department (ED) visits for chest pain, but only 13% to 25% of these visits result in diagnosis of acute coronary syndromes (ACS).[1,2] In primary care settings, it is estimated that just 1.5% of the patients presenting with chest pain are experiencing ACS.[3] Many people with chest pain now choose to go to urgent care (UC) clinics, which are typically more convenient and less expensive than EDs. According to an Urgent Care Association benchmarking report, the number of UC clinics in the United States has been on a steady rise, from 6100 centers in 2013 to 9616 in 2019.[4] As many as 89 million patients now utilize these clinics each year.[5] Although there are differing levels of service provided among UC clinics, none have the capabilities to serve as fully functional EDs. The recommendations in this article are intended for clinicians practicing in settings that are not equipped to treat patients with ACS and who do not have access to diagnostics such as troponin levels.

When patients with potentially life-threatening complaints such as chest pain present to UC, clinicians must determine whether patients are at low risk for cardiac events or need a higher level of care. There are national guidelines for the management of chest pain in the ED, but proper protocols for UC are not entirely clear. The 2021 American Heart Association/American College of Cardiology guidelines on the management and diagnosis of chest pain state that while most chest pain is not cardiac in origin, early identification or exclusion of life-threatening causes is the goal.[6] Risk stratification of patients with chest pain in the outpatient setting can optimize pa-

tient care on multiple levels, including cost savings for the patient and the health system, as well as reducing ED overcrowding.[7]

■ Definitions

Acute Coronary Syndromes

ACS are a group of potentially life-threatening conditions comprised of ST-segment elevation myocardial infarction (STEMI), non-ST-segment elevation myocardial infarction (NSTEMI), and unstable angina. For the evaluation of suspected ACS in the ED, consensus guidelines recommend obtaining electrocardiogram (ECG) and cardiac biomarker testing in addition to the basic history, physical examination, and chest radiography.[2,8-11] If these tests are unremarkable, guidelines then recommend further confirmatory testing. Despite the extensive testing typically performed for patients with chest pain from suspected ACS, a landmark study by Pope et al estimated that more than 2% of patients with ACS are mistakenly discharged from the ED, potentially leading to increased risk of harm.[12] Although this study is over 20 years old, more recent research has shown similar miss rates, suggesting that the ED evaluation of chest pain for suspected ACS remains challenging despite advances in knowledge and technology.[13-15]

Low-Risk Patient

The term *low-risk patient* is inherently unclear and can mean different things to different clinicians. In most literature, patients with chest pain who are described as being at low risk for ACS are those who: (1) are hemodynamically stable, (2) have no concerning features on history or examination, and (3) have no immediate, objective evidence of myocardial ischemia on initial ECGs and biomarker testing.[2] Current consensus guidelines further define the low-risk patient as a patient with a <1% risk of a major adverse cardiac event (MACE) or death at ≥30-days' follow-up.[16] For the purpose of this article, the low-risk patient will be defined more broadly as a patient who may be safely treated in the outpatient setting. In UC clinics where biomarkers are not available, low risk is defined using other clinical variables. Some useful risk stratification tools, such as the Marburg Heart Score and the HEAR Score, do not require troponin levels.[17,18]

■ Etiology and Pathophysiology

Myocardial ischemia results when myocardial oxygen supply and demand are mismatched. This mismatch activates free nerve endings of visceral afferent and vagal fibers originating in the myocardium and causes substernal chest discomfort referred to as angina.[19] Sensory afferents of the C1-C2 (neck and jaw) and C5-C6 (upper arm) dermatomes often overlap these fibers, which can cause referred pain from these areas. There is also considerable overlap from sensory afferents of the vagus nerve, phrenic nerve, intercostal nerves, and others. This can give rise to atypical symptoms of myocardial ischemia (known as anginal equivalents), such as shortness of breath or nausea. Alternatively, activation of these pathways by irritation of the esophagus, pleura, or aorta can lead to anginal-type pain from noncardiac sources.[20]

True anginal chest pain (ie, chest pain from myocardial ischemia) is most commonly due to atherosclerotic obstructive coronary artery disease (CAD). Once an atherosclerotic plaque reaches ≥70% total vessel diameter, blood flow through the vessel becomes limited at times of increased myocardial oxygen demand, causing myocardial ischemia.[21] Plaque rupture or endothelial erosion can cause thrombosis within the vessel, causing vessel occlusion and myocardial infarction (type 1 myocardial infarction).[22] However, nonobstructive processes such as coronary microvascular disease, coronary artery vasospasm, aortic stenosis, left ventricular hypertrophy, shock, and anemia can also lead to myocardial ischemia and infarction (type 2 myocardial infarction).

Most confirmatory tests focus on the diagnosis of atherosclerotic CAD. Stress testing indirectly detects CAD by assessing for cardiac wall motion abnormalities and perfusion deficits that are typically caused by flow-limiting stenoses. Coronary computed tomography angiography (CCTA) is a newer imaging technique that directly visualizes CAD and can measure the degree of stenosis. Computed tomography (CT) imaging can also measure coronary arterial calcification, which is associated with underlying CAD (sensitivity and specificity of coronary artery calcification scanning for predicting clinically significant CAD is estimated at 97%-100% and 54%-63%, respectively).[23]

■ Differential Diagnosis

The differential diagnosis of chest pain can be divided broadly into ischemic cardiac causes, nonischemic cardiac causes, and noncardiac causes. **(See Table 1, page 6.)** In addition to ACS, the immediately life-threatening causes of chest pain that must be considered in every patient include pulmonary embolism, aortic dissection, tension pneumothorax, perforating peptic ulcer, and esophageal perforation (Boerhaave syndrome). The complete differential diagnosis also includes other potentially serious causes such as pericarditis, pneumonia, pancreatitis, and hepatobiliary disease.

■ Urgent Care Evaluation

No single component of the history, physical examination, or initial diagnostic testing can reliably exclude ACS, but various clinical risk scores incorporate this information to identify patients at low risk for ACS

or serious short-term outcomes. The use of clinical decision pathways is advised by the 2021 American Heart Association/American College of Cardiology chest pain guideline.[6] The goal should be to promptly identify and assess patients presenting with chest pain in order to recognize those who are actively having ACS. Time matters in these patients. Intervention before myocardial damage is the desired outcome; most commonly used metric is a door-to-balloon time of less than 90 minutes.[24] It is reasonable to postpone the comprehensive patient check-in process until an initial assessment has been done, with high-risk patients expedited to a higher level of care. Patients who are identified as low risk can return to the standard check-in process and then undergo a full clinical evaluation.[25]

History

A focused history should be obtained from all stable patients. Historical features of a patient's chest pain cannot reliably rule in or rule out ACS but may be associated with a higher or lower likelihood of ACS. A 2015 review that included 58 studies found that pain radiating to both arms, pain similar to prior ischemia, and a change in the pattern of pain over the past 24 hours were the most helpful historical features in predicting ACS. These features had a positive likelihood ratio (LR) ≥2.0 and a 95% confidence interval (CI) excluding 1.0.[26] This review also found that pleuritic pain is less likely to be associated with ACS (positive LR, 0.35-0.61; 95% CI excluding 1.0). Using the same criteria, a 2005 review found that chest pain that radiates to the shoulders or arms, pain that is associated with exertion, or pain associated with diaphoresis was most predictive of ACS. Conversely, pain described as sharp or stabbing, pain not associated with exertion, and pain described as pleuritic, positional, or reproducible with palpation (colloquially referred to as "the 3 Ps") were least predictive.[27] Women, older adults, and patients with diabetes are more likely to present with "atypical" symptoms of ACS (eg, pain outside of the chest, lack of pain, or symptoms such as nausea or dyspnea).[28,29]

Table 1. Differential Diagnosis of Chest Pain[2,20]

Origin of Pain	Organ System	Condition	Typical Features
Ischemic cardiac chest pain	Cardiac	Stable angina	Substernal aching, pressure, or burning; referred pain in the neck, jaw, or arms; triggered with exertion, improved with rest or nitroglycerin
		Unstable angina	Same as stable angina, but with a change in the pattern of pain (eg, episodes are more frequent, prolonged, severe, or occurring at rest)
		Acute myocardial infarction	Same as angina, but more severe and sustained
Nonischemic cardiac chest pain	Cardiac	Pericarditis	Sharp, sustained, pleuritic; worse when supine
		Myocarditis	Variable symptoms; may mimic angina or pericarditis
		Mitral valve prolapse	Sharp, stabbing; unchanged with activity; persistent and chronic
Noncardiac chest pain	Vascular	Aortic dissection	Intense, "tearing," sudden onset; variable location (type A often felt in chest, type B often felt in back)
	Pulmonary	Pulmonary embolism	Often pleuritic, sudden onset; associated with dyspnea
		Tension pneumothorax	Pleuritic, sudden in onset; associated with dyspnea, typically unilateral; increased risk in smokers and patients with Marfan syndrome
		Pneumonia	Pleuritic; typically unilateral; gradual onset
		Bronchospastic disease	Tightness, pleuritic; may be reproducible with palpation
		Pleuritis	Pleuritic, worse with forceful breathing (eg, coughing or sneezing); often associated with symptoms of autoimmune disease
	Gastrointestinal	Esophageal rupture (Boerhaave syndrome)	Severe retrosternal pain after vomiting; may be associated with subcutaneous emphysema
		Perforated peptic ulcer	Sudden, severe epigastric pain, gradually becoming generalized; peritoneal abdominal findings; may radiate to chest
		Gastroesophageal reflux disease	Burning retrosternal discomfort; postprandial; worse when supine, may mimic angina
		Esophageal dysmotility	Intermittent retrosternal chest pain; may mimic gastroesophageal reflux disease; associated with dysphagia
	Musculoskeletal	Costal inflammation	Anterior chest wall pain that is reproducible on palpation
	Hematologic	Acute chest syndrome (sickle cell disease)	Chest pain associated with tachypnea, fever, hypoxia, and infiltrate on chest x-ray
	Miscellaneous	Herpes zoster	Burning, throbbing pain in a dermatomal distribution; may be constant or intermittent; triggered by light touch (allodynia); pain may precede rash
		Panic disorder	Chest tightness associated with dyspnea and anxiety

Several landmark studies have shown that patients' age and gender and their description of symptoms are associated with the presence of clinically significant CAD.[30-32] However, these studies examined patients who had undergone invasive angiography, a population that differs from most patients presenting to EDs or UCs with chest pain. A more recent study of patients with chest pain who underwent noninvasive CCTA has suggested that these historical features greatly overestimate the actual prevalence of CAD.[33]

In general, classic cardiac risk factors (hypertension, hyperlipidemia, diabetes, smoking, and family history of CAD) are not independently predictive of ACS in patients presenting to the ED with chest pain;[34,35] however, these classic cardiac risk factors may be more useful in younger patients. A prospective analysis of nearly 11,000 patients found that among those aged <40 years, the presence of zero risk factors had a negative LR of 0.17 for ACS (95% CI, 0.04-0.66), and the presence of 4 or more risk factors had a positive LR of 7.39 (95% CI, 3.09-17.67).[36]

Physical Examination

The physical examination in patients with chest pain is often normal, and abnormalities found on examination are often nonspecific for ACS. Hypotension, the presence of a new mitral regurgitation murmur, and the presence of a third heart sound all increase the likelihood of ACS.[9] Chest pain that is reproducible on palpation is perhaps the most useful finding in lowering the likelihood of ACS; a systematic review showed that this finding had a LR of 0.28 for ACS (95% CI, 0.14-0.54).[26] However, none of these features can be used to reliably rule in or rule out ACS. As such, the physical examination is perhaps more important for assessing overall hemodynamic function and the likelihood of alternative diagnoses of chest pain. For example, the examination findings of oxygen saturation < 95% or unilateral leg swelling are strongly associated with pulmonary embolism.[37] A prospective cohort study of 250 patients found that an aortic regurgitation murmur, pulse differential (absence of unilateral carotid or upper extremity pulse), or blood pressure differential >20 mm Hg between the arms are independent predictors of thoracic aortic dissection. Focal neurologic signs may also suggest dissection but were seen in only 13% of patients in this study.[38] A brief dermatologic examination may uncover vesicular lesions suggestive of herpes zoster.

■ Diagnostic Studies

Electrocardiogram

For patients with suspected ACS, an ECG should be obtained within 10 minutes of arrival.[2] This initial ECG should accompany any patient sent to the ED, as it will serve as the baseline for sequential ECGs. It is recommended that patients sent to the ED for evaluation of potential ACS should go by ambulance and not private vehicle; these patients are at risk for sudden decompensation and should be appropriately monitored by skilled personnel. A small prospective study showed that 12.5% of prehospital ECGs had clinically significant abnormalities (ST elevation or depression, T-wave inversion, or arrhythmia) that were not seen on the initial ED ECG, leading to a change in physician management nearly two-thirds of the time.[39] This underscores the importance of using assessment tools in addition to the ECG when risk stratifying patients.

In the United States, 29% to 38% of patients with ACS present with STEMI.[40] STEMI is defined as new ST elevation at the J point of ≥1 mm (0.1 mV) in ≥2 contiguous leads, with the exception that some degree of ST elevation is considered normal in leads V2-V3 (up to 1.5 mm in women, 2 mm in men aged ≥40 years, and 2.5 mm in men aged <40 years).[22] Significant ST elevation typically signifies transmural ischemia from acute coronary artery occlusion (type 1 myocardial infarction).[41] New horizontal or downsloping ST depression ≥0.5 mm (0.05 mV) and T-wave inversion ≥1 mm (0.1 mV) in ≥2 contiguous leads can also indicate myocardial ischemia, though this typically signifies subendocardial ischemia. A large retrospective review found these abnormalities in 22.9% and 14% of patients with NSTEMI, respectively.[42] T waves and other ECG features can vary from minute to minute in an ischemic event. **(See Figure 1, page 8.)** Serial ECGs at 5- to 10-minute intervals are recommended if the initial ECG is nondiagnostic but the patient still has concerning symptoms.

Errors in ECG interpretation can lead to a missed diagnosis of ACS. Pope et al found that 11% of patients with missed ACS actually had subtle ST elevation of 1 to 2 mm.[12] Other factors, such as left bundle branch block, left ventricular hypertrophy, electrolyte abnormalities, or digoxin use may further confound ECG interpretation in patients with suspected ACS. The ECG also often shows nonspecific abnormalities that may indicate an increased risk of adverse outcome. In a recent retrospective study of over 2300 patients, Knowlman et al found that even commonly seen nonspecific changes (such as isolated T-wave inversion in lead III or V1) confer an increased likelihood of MACE at 30 days.[43] **(See Table 2, page 8.)** This knowledge is incorporated into some clinical risk scores, such as the HEART Score (HEART is an acronym for history, ECG, age, risk factors, troponin).[44] For more information on the HEART Score, see the "Risk Stratification and Clinical Risk Scores" section.

Ultimately, a normal ECG does confer a lower risk of ACS and MACE, but as with all components

of the evaluation of chest pain, the ECG cannot be used alone to reliably rule out ACS. In a multicenter prospective study of nearly 400,000 patients with myocardial infarction, 7.9% had a normal initial ECG,[45] and in the study by Knowlman et al, 5% of patients with chest pain and a normal ECG had a MACE within 30 days.[43] Sequential ECGs are useful tools in the ED to detect early changes but are not utilized often in UC settings because patient lengths of stay are considerably shorter in UC.

Biomarkers

Cardiac biomarkers are the most objective tests for myocardial injury. Troponin, a protein specific to myocardial cells, is the preferred biomarker.[9] Two forms of troponin, cardiac troponin I and cardiac troponin T, are used by modern immunoassays to detect myocardial injury, and most guidelines make no distinction between the 2 forms.[46] The timing of troponin testing in relation to the onset of symptoms is important. In EDs, where troponin levels are easily obtainable, observational data have shown that when using conventional sensitive assays, troponin is detectable within 3 hours of arrival in nearly all patients with myocardial injury, regardless of when symptoms began.[47,48] Therefore, most guidelines recommend that if symptoms are suggestive of ACS but troponin is negative on arrival, a second value should be obtained in 3 to 6 hours.[8,16] Negative troponin testing does not entirely rule out ACS, due to the possibility of unstable angina, which is a purely clinical diagnosis. Therefore, interpretation of troponin values should always be done in conjunction with clinical risk stratification tools.

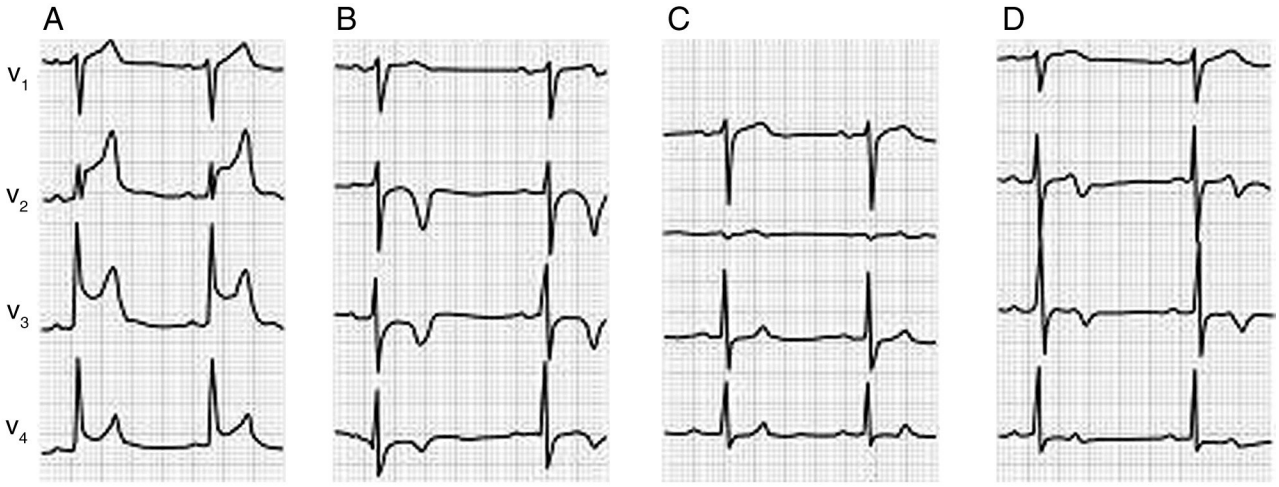
In those UC settings where point-of-care (POC) troponin assays are available, clinicians can facilitate detection of cardiac ischemia prior to hospital arrival.[49] A recent randomized trial showed that use of prehospital POC troponin testing resulted in earlier ED disposition.[50] However, in addition to not being uniformly available in UC, POC troponin

Table 2. Electrocardiographic Classification and Likelihood of 30-day Major Adverse Cardiac Event[43]

Electrocardiographic Classification	Positive LR for 30-day MACE[a]
Normal	0.4
Nonspecific ST and/or T-wave changes	1.2
Abnormal, but not diagnostic of ischemia	1.2
Ischemia or prior infarction known to be old	2.6
Ischemia or prior infarction not known to be old	9.7
Consistent with acute myocardial infarction	15.8

[a]Major adverse cardiac events include acute myocardial infarction, cardiovascular death, unstable angina, or revascularization.
Abbreviations: LR, likelihood ratio; MACE, major adverse cardiac event.

Figure 1. Serial Electrocardiograms in a Patient With Acute Left Anterior Descending Artery Occlusion



(A) Note initial ST elevation in V_1-V_4 during acute LAD artery occlusion. (B) Spontaneous reperfusion of the LAD causes deep T-wave inversions. (C) Reocclusion of the LAD now causes "pseudo-normalization" of T waves. (D) Spontaneous reperfusion again causes T-wave inversions.
Abbreviation: LAD, left anterior descending artery.
Reprinted from *Journal of Electrocardiology*. Volume 43, Issue 2. Kjell Nikus, Olle Pahlm, Galen Wagner, et al. Electrocardiographic classification of acute coronary syndromes: a review by a committee of the International Society for Holter and Non-Invasive Electrocardiology. Pages 91-103. Copyright 2010, with permission from Elsevier.

assays are less sensitive and less reliable than in-hospital assays and cannot be used to rule out ACS;[51] while a prehospital or POC troponin test is helpful when positive, it should not be used to rule out ACS when negative. When rapid troponin testing is not available, clinicians should consider the use of pathways that employ risk stratification tools to guide disposition and treatment of patients.

Risk Stratification and Clinical Risk Scores

Risk stratification enables clinicians to categorize patients into subgroups of high, medium, and low risk.[52] Identifying level of risk assists clinicians in choosing more efficient and cost-effective plans of care for individual patients. Many risk stratification tools for acute coronary artery syndrome emerged in the early 2000s, including the GRACE Score, the ADAPT Score, the Vancouver Chest Pain Rule, the North American Chest Pain Rule, and others. The Thrombolysis in Myocardial Infarction (TIMI) Score, HEART Score, and GRACE scores have been externally validated and are widely implemented in the United States.[53] The TIMI Score and the HEART Score are perhaps the most useful scores for the ED evaluation of undifferentiated chest pain from suspected ACS.[54] Both of these risk stratification tools were designed for use in the ED; because they utilize troponin levels in their scoring, they are not as useful in UC settings.

Unfortunately, there is no perfect clinical risk score. Many scores were derived without the use of contemporary biomarkers that are recommended by international consensus guidelines.[22] Study design, outcome measures, and performance on subsequent validation studies vary significantly.[56,62,63] Risk must also be considered in terms of the outcome(s) and follow-up periods used to define it (eg, nonfatal myocardial infarction vs cardiac arrest, or 48 hours vs 30 days).These factors can make it difficult to compare clinical risk scores directly or to draw firm conclusions about their accuracy.

The TIMI Score

The TIMI Score was initially designed to predict 14-day mortality in patients with confirmed NSTEMI or unstable angina.[55] It has since been validated in several studies for use in the undifferentiated chest pain patient in the ED, but a meta-analysis of these validation studies showed that patients in the lowest-risk group (TIMI Score = 0) still had a 30-day incidence of cardiac events of 1.8%, which may be unacceptably high.[56] The addition of serial biomarker measurements may increase the accuracy of the TIMI score. Two observational studies, ASPECT and ADAPT, used the TIMI Score and biomarkers (troponin I, creatine kinase MB-isoenzyme [CK-MB], and myoglobin in ASPECT; troponin I only in ADAPT) at 0 and 2 hours, and they showed sensitivity and negative predictive value (NPV) of 99.3% to 99.7% and 99.1% to 99.7%, respectively.[57,58]

The HEART Score

The HEART Score, developed specifically for risk stratification of patients with undifferentiated chest pain, has been validated nationally and internationally and performs similarly to the TIMI Score, with low-risk patients having a 0.9% to 1.7% risk of MACE at 6-week follow-up.[59-61] As with the TIMI Score, the addition of serial biomarker measurements may also increase accuracy; Mahler et al used the HEART Score with biomarkers (troponin I, CK-MB) at 0 hours and 4 to 6 hours and demonstrated a sensitivity and NPV of 100%.[64]

An online tool for calculating the HEART Score is available at: https://www.mdcalc.com/calc/1752/heart-score-major-cardiac-events

Risk Stratification Scores Used in Settings Without Troponin Testing

Most UC clinics do not have access to rapid troponin levels, and even at clinics that do have POC rapid troponin testing available, results are less reliable than troponin testing done in a hospital laboratory. Research on risk stratification tools is not as robust for the outpatient population as it is for ED patients, but there are some validated options.

The Marburg Heart Score

A cross-sectional study by Bösner et al enrolled 1249 patients with chest pain from 74 primary care practices in Germany and developed a 5-point questionnaire to be used as a prediction tool for acute CAD.[65] **(See Table 3, page 10.)** The endpoint in this study, and most other predictive tools without troponin (such as INTERCHEST), also used CAD as the endpoint rather than ACS.[66] The determinants used to develop the predictive rule were age/sex, known clinical vascular disease, pain worsening with exercise, pain not reproducible with palpation, and patient assumption that the pain is cardiac. The Marburg Heart Score awards 1 point for each determinant found. Scores of 3 to 5 points were treated as positive predictors for CAD, while scores of ≤2 points were treated as negative. The score had a sensitivity of 86.4%, with a NPV of 97.3% and a false negative rate of 2.7%. The original study included a validation cohort in Switzerland that had an NPV of 97% (95% CI, 96.4%-99.1%), and other external validation studies have been done.[17,67-69] As with all clinical prediction tools, the Marburg Heart Score cannot be used to rule out CAD, but has been shown to be useful as a triage tool.[25] The Marburg Heart Score does not include ECG, which is an essential tool for assessing patients with chest pain. Clinicians must utilize the tool in conjunction with patient assessment and history as well as clinical gestalt.

An online tool for calculating the Marburg Heart Score is available at: https://www.mdcalc.com/calc/4022/marburg-heart-score-mhs

The HEAR Score

As previously discussed, the HEART Score is one of the most validated and utilized tools in the United States;[53,70] The HEAR score is a modification of the HEART Score that omits the troponin level. This makes it an ideal tool for UC clinics that do not have troponin testing capabilities, and the College of Urgent Care Medicine has embraced the use of the HEAR Score in UC settings.[66] Initial research on the validity and specificity of the score for identifying low-risk cardiac pain has been supportive.[71] A study of 1150 chest pain patients concluded that a HEAR Score ≤1 can identify the estimated 17% of all patients who are at very low risk for acute myocardial infarction and would be unlikely to benefit from troponin testing.[72] As with any clinical prediction tool, the HEAR Score does not completely rule out ACS and should be used as a tool in a decision-making pathway and in conjunction with clinical gestalt.

Chest Radiography

Most chest x-rays performed in the chest pain population are normal. One prospective study of over 500 ED patients with nontraumatic chest pain found that >90% of chest x-rays performed in this population were normal, though 2.1% had abnormalities requiring acute intervention, including pulmonary edema, consolidation, or large pleural effusions.[73] Reducing the number of chest x-rays performed in this population could decrease radiation exposure and decrease costs for patients and health care organizations. Unfortunately, attempts to validate a clinical decision rule designed for this purpose have been unsuccessful.[74]

Confirmatory Testing

Confirmatory testing requires diagnostics beyond the capabilities of nonspecialized outpatient clinics. The general purpose of confirmatory testing is to identify obstructive CAD (either by direct visualization or indirectly by inducing regional ischemia) that would benefit from further treatment. Confirmatory testing may take the form of exercise electrocardiography and/or various types of noninvasive imaging, including exercise or chemical myocardial perfusion imaging, rest or stress echocardiography, and cardiac magnetic resonance imaging. Confirmatory testing is conducted in settings such as the ED, inpatient facilities, and cardiac-specialized outpatient settings.

Table 3. Marburg Heart Score Criteria

1 point each:[a]

- Age/sex (men aged ≥55 years, women aged ≥65 years)
- Known vascular disease
- The symptoms are induced by exercise
- The pain cannot be induced by palpation
- The patient suspects that heart disease is the cause

Points	Likelihood of Coronary Artery Disease
0-1	<1% (very low)
2	5% (low)
3	25% (intermediate)
4-5	65% (high)

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■ Treatment

Patients with chest pain who are at low risk for ACS or MACE may be candidates for interventions related to primary prevention and lifestyle modification, though benefits specific to this population have not been studied.

Aspirin is beneficial for primary prevention of cardiovascular disease. A meta-analysis including 95,000 patients showed aspirin use was associated with a 12% proportional reduction in serious vascular events, including first myocardial infarction, though the benefits of its use must be balanced against the risk of bleeding.[75] The 2022 United States Preventive Services Task Force guidelines recommend the initiation of 81 mg per day of aspirin in patients aged ≥60 years (Grade D recommendation). For those between aged 40 to 59 years with ≥10% 10-year risk of cardiovascular disease, the decision to start aspirin for primary prevention should be made on an individual basis, weighing risk and benefit (Grade C recommendation).[76]

Initiation of antihypertensive treatment from the ED in at-risk populations is safe and effective in lowering mean blood pressure at short-term follow-up, and it is not unreasonable to extrapolate this strategy to UC.[77] A 2013 American College of Emergency Physicians Clinical Policy supported the initiation of antihypertensive therapy in at-risk populations (eg, those with poor follow-up care) with markedly elevated blood pressure in the ED, though this is a Level C consensus recommendation and the benefits of this practice are not evidence-based.[78]

■ Special Populations

Chest Pain in Women

Several important differences must be considered during the evaluation of women with chest pain. First, compared with men of the same age, women have an overall lower prevalence of obstructive

CAD and a higher likelihood of atypical symptoms, which can lead to delayed diagnosis and increased likelihood of being discharged with missed myocardial infarction.[12,79] Second, ECG criteria for detection of myocardial infarction are different in women. Minor J-point elevation in V2-V3 can be a normal variant in men and women, but a study of 1332 healthy volunteers established that the upper limit of normal in women is lower than that of men.[80] Third, exercise stress testing is less accurate in women; a meta-analysis including 4113 women found that the sensitivity of exercise electrocardiography and exercise myocardial perfusion imaging for the detection of CAD was 0.61 and 0.78, respectively, compared to 0.68 and 0.85 in similar meta-analyses of studies that included a majority of men.[81]

Younger Patients

The prevalence of ACS is <2% in patients aged <40 years,[36] but 4% to 8% of myocardial infarctions still occur in this age group.[82] Nonetheless, observational research has shown that, among patients in this age group with chest pain, those who have no known history of heart disease and no cardiac risk factors (hypertension, elevated cholesterol, tobacco use, diabetes, and family history of premature CAD) are at <1% risk of ACS and MACE at 30 days[82] and 1 year.[83] Another observational study showed that even when cardiac risk factors are present, if the patient had a normal ECG and negative initial biomarker (either CK-MB or conventional troponin I), the risk of ACS or MACE at 30-day follow-up was 0.14%.[84] Not surprisingly, several retrospective observational studies have shown that confirmatory testing is very low-yield in this age group; out of 1650 stress tests performed in a total of 1993 patients, 20 were positive, of which only 4 were judged to be true positives via coronary angiography.[85-88]

Older Adult Patients

The evaluation of the older adult patient with chest pain is especially challenging. Patients aged ≥75 years have increased incidence, prevalence, and severity of CAD and ACS.[40] Older adult patients often have atypical and nonspecific symptoms, the ECG is less accurate,[89] and elevated troponin is less specific for myocardial infarction.[90] Most clinical risk scores include age as an independent risk factor, decreasing their ability to identify low-risk patients who may be suitable for discharge.[91]

Patients With Known Coronary Artery Disease or Previous Cardiac Testing

Patients with known CAD who present with chest pain have a higher pretest probability of ACS; one systematic review found that prior CAD conferred a positive LR of 2.0 (95% CI, 1.4-2.6) for ACS in patients presenting with chest pain.[26] Pre-existing CAD can also make the clinical evaluation more challenging. For example, the ECG may be more difficult to interpret due to baseline evidence of prior myocardial infarction, bundle branch block, conduction abnormalities, or arrhythmia.[92] Older patients with known CAD will have a baseline score of 2 points on both the Marburg Heart Score and the HEAR Score, so clinicians should keep in mind that this population will be less likely to be deemed low-risk.

The prognostic utility of previous cardiac testing in patients presenting with new chest pain is unclear. Annualized rates of myocardial infarction or cardiac death in outpatients who have had a negative stress test are 0.80% for exercise treadmill, 0.65% for exercise myocardial perfusion imaging, and 1.78% for pharmacologic myocardial perfusion imaging (the higher event rate in pharmacologic myocardial perfusion imaging is attributed to greater comorbidities in patients who are unable to exercise).[93] Patients with normal or nearly normal cardiac catheterizations have excellent long-term prognosis and very low risk of MACE at 5- and 10-year follow-up.[94,95] A relatively recent normal cardiac catheterization makes obstructive CAD a very unlikely etiology for chest pain.

■ Controversies and Cutting Edge

High-Sensitivity Troponin Testing

The ability of modern troponin assays to detect smaller and smaller levels of circulating troponin continues to improve. The United States Food and Drug Administration approved the first high-sensitivity troponin T assay for use in the United States in January 2017, though several other high-sensitivity assays have been used outside of the United States since 2009.[96] These high-sensitivity assays have been shown to lead to earlier diagnosis of myocardial infarction, potentially decreasing ED length of stay and time to disposition or definitive treatment.[97] They also increase the NPV of a normal troponin level, improving risk stratification and prognostication.[98-100]

However, as discussed previously, many conditions other than ACS can cause release of troponin, and high-sensitivity troponin assays are even less specific for ACS than their conventional counterparts. Delta troponin can increase specificity for ACS, though the optimal delta for high-sensitivity assays has not yet been defined. Tiny changes in troponin due to biologic or analytic variation become detectable with high sensitivity assays and may be higher than the 20% threshold used to define myocardial infarction with conventional sensitive assays in patients with an elevated baseline level.[101]

High-sensitivity troponins could reduce or even eliminate the diagnosis of unstable angina.[102] Unstable angina is distinguished from NSTEMI on the basis of normal cardiac biomarkers, so as high-

sensitivity assays detect smaller levels of troponin that would otherwise be missed by conventional assays, increasing numbers of patients are likely to be diagnosed with NSTEMI instead of unstable angina. A prospective study of 1124 patients found that the introduction of high-sensitivity troponin led to a relative increase in the diagnosis of myocardial infarction (attributable to increased NSTEMI diagnoses) by 22% and a corresponding relative decrease in the diagnosis of unstable angina by 19%.[103]

Triple-Rule-Out Computed Tomography

The "triple-rule-out" (TRO) CT combines CT angiography of the coronary arteries concurrently with the pulmonary arteries and aorta, thereby simultaneously evaluating for CAD, pulmonary embolism, and aortic dissection.[104] TRO CT typically requires additional contrast volumes and radiation exposure over dedicated CT imaging alone, with one study citing a mean difference of 38 mL and 4.84 mSv, respectively.[105] There may be incremental diagnostic yield to TRO CT over dedicated CT imaging alone, but at present, there are no clearly defined populations of ED patients that benefit from TRO CT,[106] and the data are sparse on TRO CT in the outpatient population.

Disposition

In outpatient settings such as UC, it is important to identify patients at high risk of ACS as quickly as possible, ideally within 10 minutes of presentation. These patients should be sent by ambulance, along with their UC ECG, to the nearest ED for evaluation. All patients with ACS are at high risk of deterioration and the sooner patients with STEMI and other occlusive MI patterns are reperfused (via interventional cardiac catheterization, coronary artery bypass grafting, or tissue plasminogen activator), the greater the chance that the impacted myocardial muscle will be preserved without damage.

In ED settings, many institutions are incorporating accelerated diagnostic protocols that aim to identify low-risk patients in a safe and efficient manner. For example, the University of Maryland Medical System developed an evidence-based Accelerated Diagnostic Protocol in 2015 that uses conventional troponin assays, risk stratification using the HEART Score, and defined shared decision-making between the clinician and patient.[107] Patients with a single negative troponin and a HEART Score of 0 to 3 may choose from 3 options for disposition: (1) discharge home with outpatient follow-up (<2% risk of MACE at 4 weeks); (2) stay for repeat troponin at 3 hours and, if negative, discharge home with outpatient follow-up (<1% risk of MACE at 4 weeks); or (3) be placed in observation for further testing or treatment as indicated. This accelerated diagnostic protocol highlights a number of variables that may differ from other validated accelerated diagnostic protocols and between individual institutions and clinicians, but its core principles of focused testing with contemporary cardiac biomarkers, risk stratification with a validated clinical risk score, and shared decision-making between the clinician and patient are crucial to every safe disposition.

Summary

By using a combination of the clinical history, physical examination, ECG, and validated clinical risk scores, many patients who present to UC with chest pain can be confidently identified as being at low risk for ACS and MACE, and can be appropriately managed as outpatients. The use of high-sensitivity troponin assays can improve the detection of myocardial infarction, but these assays are not available in most outpatient settings. The Marburg Heart Score and the HEAR Score are decision-making tools that can be used in UC settings in conjunction with other assessment components to risk stratify patients with chest pain without the use of cardiac biomarkers. While it is critical that UC clinicians identify patients with potentially life-threatening cardiac disease correctly and promptly, it is also possible to practice good stewardship of healthcare resources by managing low-risk chest pain in the outpatient setting.

5 Things That Will Change Your Practice

1. Patients who present to UC with chest pain should receive prompt assessment, bypassing the registration process if needed, but can be returned to the queue if they are determined to be at low risk for ACS.
2. The Marburg Heart Score and the HEAR Score can help to identify low-risk cardiac pain in settings without access to troponin testing.
3. Maintain suspicion for ACS in elderly, diabetic, or female patients who present with atypical symptoms (eg, pain outside of the chest, lack of pain, or symptoms such as nausea or dyspnea).
4. Patients with suspected ACS should be transported to the ED by ambulance, not private vehicle (even if the patient is stable in UC), due to the risk of sudden decompensation in these patients.
5. If a patient is sent to the ED for further evaluation, the initial ECG obtained at UC should accompany the patient.

Case Conclusions

CASE 1

For the 65-year-old man who had a 20-minute episode of chest pain…

He had a high pretest probability of ACS, given his age, history, comorbidities, and description of symptoms. His initial ECG was normal but was repeated to detect transient ischemic changes. Troponin testing was not available in your clinic, so you decided to utilize the HEAR Score. His score was 6 (2 points for highly suspicious symptoms, 2 points for age, and 2 points for prior history of myocardial infarction), placing him at moderate risk (12%-16.6%) for MACE within 6 weeks. You discussed options with the patient for shared decision making, but based on his HEAR score and risk factors, you strongly advised him to go to the ED for additional evaluation. With encouragement from his wife, he agreed but refused to go by ambulance, which is the standard of care for patients with a potential life-threatening event such as myocardial infarction. You discussed with the patient and his wife the possible risks (including death) of going to the ED in a private vehicle. The patient continued to refuse an ambulance but agreed to go directly to the ED and signed the "Against Medical Advice" form regarding ambulance refusal. He had a relatively uneventful ED evaluation and was admitted to the observation unit, where a stress echocardiogram was positive. Coronary angiography was performed and showed 2-vessel disease; 2 coronary stents were placed, and the patient was discharged shortly thereafter.

CASE 2

For the 22-year-old man with left-sided chest pain and shortness of breath…

He was already at very low risk for ACS or future MACE given his age, lack of cardiac risk factors or previous cardiac history, lack of family history of premature cardiac disease, the atypical nature of his symptoms, and his normal ECG. His Marburg Heart Score was calculated at 2, with 1 point for pain not reproducible with palpation and 1 point for patient suspicion of a cardiac cause. His HEAR score was 0. Although risk-stratification tools placed him in a low-risk category, his resting tachycardia persisted. He was sent to the ED for further evaluation due to concerns for potential pulmonary embolism and pericarditis. In the ED, the patient had a negative d-dimer. His high-sensitivity troponin T was slightly above the hospital's accepted normal range. A bedside ultrasound showed a small pericardial effusion; a diagnosis of viral pericarditis was made, and the patient was successfully treated with colchicine and NSAIDs.

CASE 3

For the 20-year-old woman with chest pain that was reproducible with palpation…

She had no risk factors or family history of heart disease. An ECG was performed and interpreted as normal. Her Marburg Heart Score was 1, with the point given for the patient's perception that the pain is cardiac. Her HEAR Score was 0. Both scores placed her in the low-risk category. You determined that her pain most likely had a musculoskeletal etiology, and she was counseled on the causes and management of suspected chest wall pain. The patient was reassured, instructed to take NSAIDs, and directed to see her primary care provider for follow-up care. Symptoms that would prompt a return to care were discussed; she was comfortable with the plan at discharge. By the time of her follow-up appointment with her primary care provider, her symptoms had resolved.

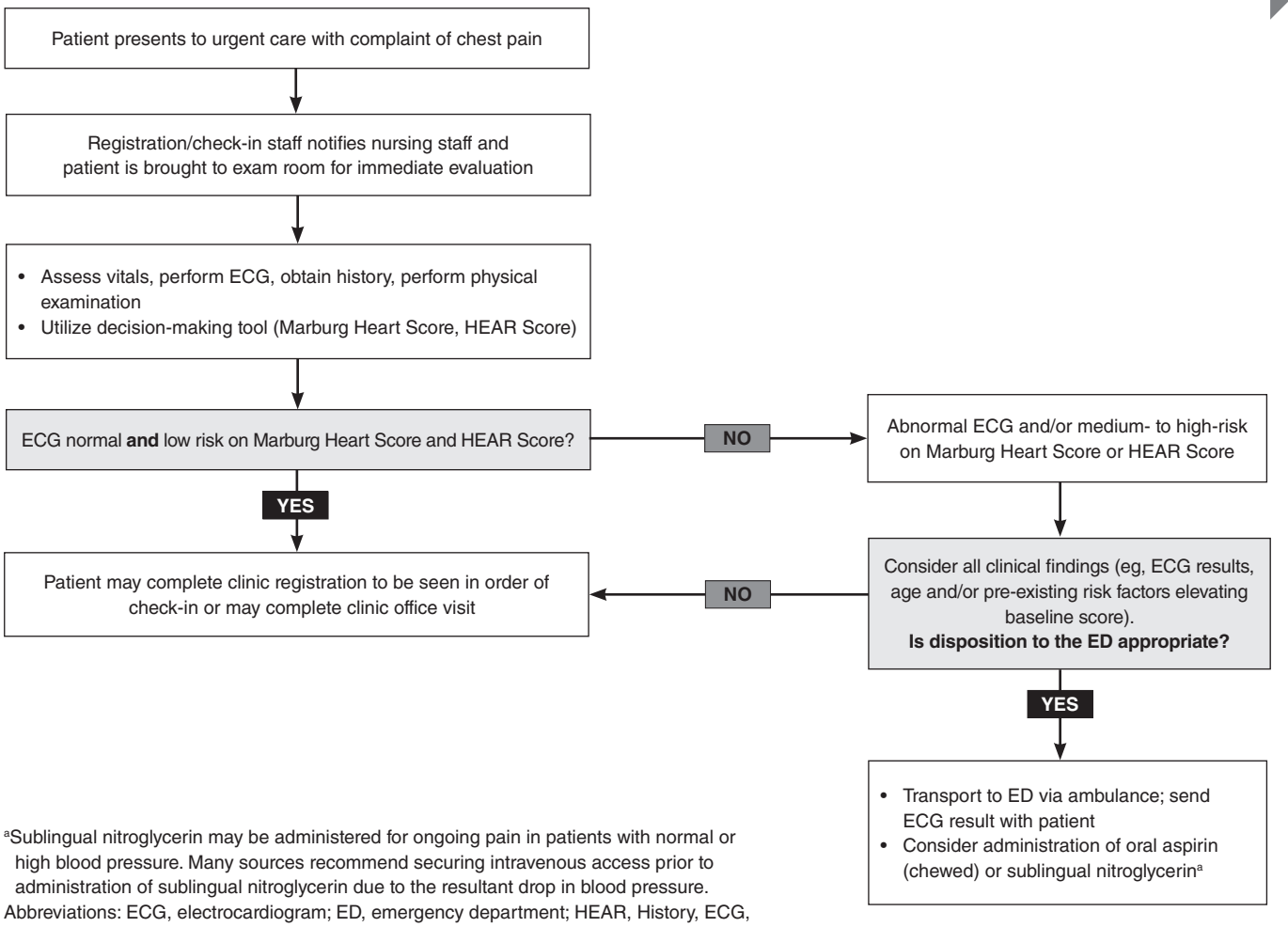
■ Critical Appraisal of the Literature

A large body of research is available on the evaluation and management of undifferentiated chest pain in the ED. Narrowing this work to chest pain only from presumed ACS yielded >1000 articles (using the search terms chest pain, acute coronary syndrome, and emergency department). Among these, articles from the following categories were reviewed: *low risk, risk stratification, clinical decision rules, stress testing, cardiac imaging*, and *disposition*. The Cochrane Library was searched using the term chest pain and acute coronary syndrome, but none were directly applicable to this topic. A National Guideline Clearinghouse search using the terms *low risk, chest pain*, and *acute coronary syndrome* yielded 104 articles, 8 of which were applicable. Additional references were gathered by reviewing the bibliographies of selected articles generated from these searches. The review of literature was later expanded to include *urgent care, outpatient clinics, Marburg Score*, and *HEAR Score*. Relevant guidelines and statements from various professional groups were also reviewed, with emphasis placed on more recent guidelines and statements that supersede older versions.

■ References

Evidence-based medicine requires a critical appraisal of the literature based upon study methodology and number of subjects. Not all references are equally robust. The findings of a large, prospective,

Clinical Pathway for Urgent Care Evaluation of Patients With Chest Pain



[a]Sublingual nitroglycerin may be administered for ongoing pain in patients with normal or high blood pressure. Many sources recommend securing intravenous access prior to administration of sublingual nitroglycerin due to the resultant drop in blood pressure.

Abbreviations: ECG, electrocardiogram; ED, emergency department; HEAR, History, ECG, Age, Risk Factors.

This clinical pathway is intended to supplement, rather than substitute for, professional judgment and may be changed depending upon a patient's individual needs. Failure to comply with this pathway does not represent a breach of the standard of care.

randomized, and blinded trial should carry more weight than a case report.

To help the reader judge the strength of each reference, pertinent information about the study is included in bold type following the reference, where available. In addition, the most informative references cited in this paper, as determined by the authors, are noted by an asterisk (*) next to the number of the reference.

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Up to 90% of patients seeking medical care for chest pain in an outpatient are not having ACS; therefore, most of these cases will result in a diagnosis of some other source of the chest pain symptom.[1,2] The following tips are provided for documentation and billing of encounters with this patient population.

A common mistake is to simply document the assessment of "chest pain," without further clarification. While there is a generic billing code for "chest pain," in reality the clinician is reporting a symptom rather than a true diagnosis. Just as patients with appendicitis are not diagnosed with "abdominal pain," and patients with pneumonia are not diagnosed with "cough," the assessment should state what the clinician believes is the source of the symptom (eg, pericarditis). One reason it has become common for outpatient clinicians to use symptoms for diagnoses is the Centers for Medicare and Medicaid Services guidelines for coding and reporting. The guidelines state that if "a definitive diagnosis has not been established by the end of the encounter, it is appropriate to report codes for sign(s) and/or symptom(s) in lieu of a definitive diagnosis."[108] This is also the reason clinicians working in the outpatient setting are often taught to avoid terms such as *probable, suspected, questionable, rule out, compatible with, consistent with, working diagnosis*, or similar terms that suggest uncertainty. The interpretation of what constitutes a "definitive diagnosis" is left to the clinician. If you are certain enough to pursue a specific treatment plan for the diagnosis, such as prescribing ibuprofen for costochondritis or antibiotics for pneumonia, it can be argued that the diagnosis is definitive for coding purposes.

Consider the case presented in this article of the young woman who had chest pain that was reproducible with palpation, and who had also been consuming energy drinks. Instead of a diagnosis of "chest pain," the clinician might state, "Acute musculoskeletal chest pain (R07.89), less likely an adverse reaction to caffeine (T43.615A) or GERD (K21.9)." The clinical presentation in this case is highly suggestive of a musculoskeletal source. It is a definitive diagnosis, based on the chosen treatment plan of NSAIDs. The billable diagnosis is musculoskeletal chest pain, which is code R07.89 in the International Classification of Diseases, 10th Revision, Clinical Modification (ICD-10). However, other etiologies are still possible, and the clinician has recommended reassessment if symptoms progress or new symptoms appear. Including the other potential diagnoses in the assessment demonstrates the complexity of the clinician's medical decision making, and provides a road map for the next steps in management if this patient were to return. See **Table 4** for a list of diagnoses often associated with chest pain, along with the corresponding ICD-10 codes.

Be sure to also document and capture any secondary diagnoses, such as hyperlipidemia or diabetes mellitus type 2. If abnormalities are present on the ECG, those should be documented as well; see **Table 5** for common diagnoses and their codes.

Table 4. ICD-10 Codes for Diagnoses Often Associated With Chest Pain[108]

Diagnosis	ICD-10 Code
Acute coronary syndrome	I24.9
Acute costochondritis	M94.0
Gastroesophageal reflux	K21.9
Musculoskeletal chest pain	R07.89
NSTEMI	I21.4
Panic attack	F41.0
Pericarditis	I31.0
Pleurisy	R09.1
Pulmonary embolism	I26.99
STEMI	I21.3

Abbreviations: ICD-10, International Classification of Diseases, 10th Revision, Clinical Modification; NSTEMI, non-ST-segment elevation myocardial infarction; STEMI, ST-segment elevation myocardial infarction.

Table 5. ICD-10 Codes for Abnormal Electrocardiogram Findings[108]

ECG Finding	ICD-10 Code
Bifascicular block	I45.2
Left bundle branch block	I44.7
Prolonged QT interval	R94.31
Right bundle branch block	I45.10

Abbreviations: ECG, electrocardiogram; ICD-10, International Classification of Diseases, 10th Revision, Clinical Modification.

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Risk Management Pitfalls for Urgent Care Evaluation of Patients With Chest Pain

1. **"My patient was young and healthy, so I didn't suspect ACS."** Younger patients are at lower risk of ACS, but 4% to 8% of myocardial infarctions still occur in patients <40 years old. While traditional cardiac risk factors are generally not useful in the management of undifferentiated chest pain, a high risk-factor burden is more predictive of ACS in younger patients. Validated clinical risk scores can identify very-low-risk patients in this age group with excellent accuracy.

2. **"Her symptoms didn't sound like angina, so ACS wasn't even in my differential diagnosis."** A patient's history cannot reliably exclude ACS. Atypical symptoms are often present and are more common in women, older adults, and people with diabetes. Additional testing, especially in these population groups, should be considered to reliably rule out ACS.

3. **"The ECG was normal, so I didn't think further testing was indicated."** A normal ECG lowers the risk of ACS but does not adequately exclude it, and nearly 8% of patients with myocardial infarction have a normal ECG. Misinterpretation of the ECG is also a factor associated with missed diagnosis of ACS. Accuracy is increased by obtaining serial ECGs.

4. **"The pain was reproducible on palpation, so I ruled out ACS."** Pain that is reproducible on palpation lowers the risk of ACS but does not exclude it.

5. **"My patient had a Marburg Heart Score of 2, so I excluded ACS and discharged him without further testing."** A Marburg Heart Score of 2 risk stratifies a patient as nonurgent, or low risk, but clinical decision making cannot be based on the Marburg Heart Score alone. History, physical assessment, and ECG results must also be considered, as well as clinical gestalt.

6. **"She had a negative stress test 6 months prior, and the ECG was normal, so I thought it was safe to rule out ACS."** The annual event rate (myocardial infarction or cardiac death) is about 1% after any stress test. Any patient presenting with chest pain should be evaluated with ECG and risk stratified using a validated clinical risk score, despite the recent negative stress test.

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■ CME Questions

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1. **Which of the following historical features is most likely to INCREASE the likelihood of ACS?**
   a. Chest pain that is associated with exertion
   b. Chest pain that is pleuritic
   c. Chest pain that is described as "stabbing"
   d. Chest pain that has been present for several hours

2. **Which of the following physical examination findings is most likely to indicate a DECREASE in the likelihood of ACS?**
   a. Presence of a third heart sound
   b. Hypotension
   c. Pulmonary rales
   d. Chest pain that is reproducible on palpation

3. **What is the goal time frame to obtain an ECG on a patient with chest pain from the time they enter the clinic?**
   a. 10 minutes
   b. 15 minutes
   c. 30 minutes
   d. 1 hour

4. **Which of the following ECG findings meets the criteria for STEMI?**
   a. J-point elevation of 0.2 mV in V2-V3 in a 30-year-old woman
   b. J-point elevation of 0.15 mV in V2-V3 in a 50-year-old man
   c. J-point elevation of 0.2 mV in V2-V3 in a 30-year-old man
   d. J-point elevation of 0.05 mV in V5-V6 in any adult

5. **Which of the following predictive tests for chest pain DO NOT require a troponin level?**
   a. HEART Score and TIMI Score
   b. GRACE Score and HEART Score
   c. Marburg Heart Score and HEAR Score
   d. North American Chest Pain Rule and Vancouver Chest Pain Rule

6. **Regarding younger patients and ACS, which of the following statements is CORRECT?**
   a. Traditional cardiac risk factors are not helpful for risk stratification in this group.
   b. Up to 8% of myocardial infarctions occur in patients aged <40 years.
   c. Confirmatory testing is beneficial in younger patients with an unremarkable ED workup.
   d. Biomarkers should not be obtained for younger patients.

7. **All of the following factors make the evaluation of suspected ACS in older adults more challenging EXCEPT:**
   a. Elevated troponins are less specific for myocardial infarction in older adults.
   b. Atypical symptoms are more common in older adults.
   c. Most clinical risk scores incorporate age, making it more difficult to identify low-risk patients.
   d. Confirmatory testing cannot be performed in older adults.

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