# Acute Chest Pain Care Guide

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CALIFORNIA CORRECTIONAL
HEALTH CARE SERVICES

*Information contained in the Care Guide is not a substitute for a health care professional's clinical judgment. Evaluation and treatment should be tailored to the individual patient and the clinical circumstances. Furthermore, using this information will not guarantee a specific outcome for each patient. Refer to "Disclaimer Regarding Care Guides" for further clarification.*

*https://cchcs.ca.gov/clinical-resources/*

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## GOALS

- ✓ **Determine clinical stability; assess need for transfer to emergency department (ED) for timely intervention**
- ✓ **Identify patients with acute coronary syndrome (ACS) and other life-threatening emergencies**
- ✓ **Identify and manage stable patients with non-cardiac chest pain precipitants**
- ✓ **Coordinate with cardiology to appropriately evaluate and manage patients diagnosed with chronic coronary disease (CCD), especially those presenting to Triage and Treatment Area (TTA) with recurrent chest pain**

## ALERTS

- **Women with chest pain are at risk for underdiagnosis, so history should emphasize accompanying symptoms**
- **Transfer clinical unstable patients directly to ED by EMS (i.e., do not delay transfer for electrocardiogram [ECG] or other evaluation)**
- **Emergent transfer for reperfusion therapy if evidence of ST-segment elevation myocardial infarction (STEMI)**
- **Patients with ACS symptoms, stable vital signs, and no evidence of STEMI should be transferred to ED for monitoring and serial cardiac troponin evaluation**

*This Acute Chest Pain Care Guide is based on the 2021 American Heart Association (AHA)/American College of Cardiology (ACC) Guideline for the Evaluation and Diagnosis of Chest Pain, which relies on cardiac troponin (cTn) as part of the assessment of patients with acute chest pain and suspected ACS. High-sensitivity cTn (hs-cTn) is preferred for a biomarker diagnosis. Acute troponin testing, which is typically done in the ED, cannot be done in our setting, so adaption of all AHA/ACC guideline recommendations is not possible.*

## INTRODUCTION

Approximately 1% of all ambulatory visits in primary care and approximately 4% of all emergency department visits were for acute chest pain. Within the United States, heart disease remains the leading cause of death since the early 1900s, yet only 2-4% of patients presenting to a primary care provider with chest pain will have ACS. National guidelines and cardiovascular societies increasingly recognize health disparities that exist among diverse patient populations and the need for cultural competency training to improve provider awareness of non-ACS causes of chest pain as well as anginal-equivalent symptoms that may indicate ACS.[1,2,3]

**Chest pain** is broadly defined as pressure, tightness, squeezing, heaviness, or burning that can occur in the chest, shoulder, arms, jaw, neck, and upper abdomen. Chest pain should be described as "cardiac", "possible cardiac", or "noncardiac" to determine the cause of chest pain. Furthermore, cardiac chest pain can be "ischemic" or "nonischemic" in etiology.[1]

## EVALUATION

The most common causes of chest pain in the primary care setting are chest wall pain, reflux esophagitis, and costochondritis.[2] However, acute chest pain can be caused by many life-threatening conditions, such as ACS, pulmonary embolism (PE), and acute chest syndrome, among others. Detailed evaluation of a patient's chest pain history (characteristics), cardiac risk factors, focused physical exam, and ECG findings can help differentiate acute chest pain etiologies, but the presence or absence of ACS can only be confirmed by cTn/hs-cTn results, which is not available at CCHCS for acute chest pain.[1]

The classic symptoms of ACS are substernal chest pain, often described as pressure or crushing, with radiation to the jaw and/or left arm. However, this classic presentation is not described by many groups, including elderly patients, patients with diabetes, and women, in whom cardiac ischemia may present as shortness of breath, nausea or vomiting, lightheadedness, confusion, presyncope or syncope, as well as vague abdominal symptoms. See **Health Equity Alert Box**.[1,4]

### HEALTH EQUITY ALERT

Ischemic heart disease represents the leading cause of death in women, who experience relatively worse outcomes compared to men. This is due to sex-specific differences from biological factors, as well as gender-specific differences from broader social, environmental, and community factors. There exists a knowledge gap regarding these differences in presentation, risk factors, pathophysiology, and response to treatment, which contributes to cardiovascular outcome disparities. In addition to traditional risk factors, sex-specific risk factors include autoimmune disease, pregnancy, menopause, and depression.[4]

Furthermore, there may be differences in the description and perception of chest pain and associated symptoms among various diverse patient groups, so consider sociocultural differences in the evaluation and management of patients presenting with acute chest pain.[1] Remember that some populations present with ACS differently, so consider these differences during rapid assessment (see **Appendix 1**).[1,2,3,4]

## Evaluation, cont'd

### STEP 1: CLINICAL STABILITY

When a patient presents with acute chest pain, a rapid assessment is needed, starting by determining clinical stability.

- Assess the patient's vital signs and level of consciousness. If the patient is clinically unstable or has altered level of consciousness, initiate EMS transfer to higher-level of care (HLOC).
- Unless a noncardiac cause is evident, obtain and review ECG **within 10 minutes** of arrival for all patients who present with acute chest pain, irrespective of setting. As per AHA/ACC national guidelines, target initial medical decision-making within 10 minutes of obtaining ECG and interpretation, which includes machine automatic interpretation ECG +/- provider review) in a clinically stable patient. If ECG shows ST elevation or new left bundle branch block (LBBB), assume STEMI ACS and initiate EMS transfer for STEMI. Notify EMS of STEMI so patient is directed to appropriate ED. Compare ECG to prior baseline ECG, if available. See **Appendix 2** for ECG Interpretation.
  - A normal ECG markedly reduces the probability that chest pain is due to acute myocardial infarction (MI), but it does not exclude another serious, life-threatening cardiac etiology.
- Emergently transfer the following patients to HLOC. While awaiting EMS transfer to HLOC, continue to monitor level of consciousness, vital signs, cardiac rhythm, and oxygen saturation:
  - Patient with unstable vital signs, such as heart rate (HR) ≤50 or ≥100, respiratory rate (RR) ≤12 or >20, systolic blood pressure (SBP) ≤90 mmHg, and $SpO_2$ <95% on ambient air.
  - Patient with ECG showing ST elevation or new LBBB. Notify EMS of STEMI so patient is directed to appropriate ED.
  - Patient with ECG showing high-grade heart block or unstable arrhythmia/bradycardia/tachycardia.
  - Patient with serious life-threatening conditions, such as STEMI/ACS, PE, acute chest syndrome, tension pneumothorax, cardiac tamponade, and esophageal rupture.
- For stable patients, continue monitoring and proceed to next steps in evaluation of chest pain.[1,2]

## Evaluation, cont'd

### STEP 2: STABLE PATIENT WITH ABNORMAL ECG

- **ECG abnormalities other than STEMI or new LBBB:** Compare ECG to prior baseline ECG, if available. These non-STEMI/non-LBBB ECG abnormalities include ST depression, T-wave flattening, T-wave inversion, heart block, arrhythmia, or other changes from ECG baseline. Left ventricular hypertrophy (LVH), bundle branch blocks, and ventricular pacing may mask signs of ischemia.
- Continue evaluation with cardiac risk factor assessment, acute chest pain history, and focused physical examination, as detailed below. Consider other serious life-threatening conditions, such as non-ST elevation ACS (NSTE-ACS), PE, acute chest syndrome, tension pneumothorax, cardiac tamponade, and esophageal rupture.
- Utilize the patient's cumulative burden of traditional and nontraditional cardiac risk factors, acute chest pain history and descriptors, and ECG findings to determine ACS risk.
- Identify prior cardiac testing, if available.
- Unless clear non-serious, non-life-threatening cause of acute chest pain is identified, consider transfer to HLOC for cTn/hs-cTn and further evaluation.[1,2]
- Emergently transfer the following patients to HLOC:
  - Patient becomes clinically unstable during the course of their evaluation with unstable vital signs, defined as new or deteriorating HR ≤50 or ≥100, RR ≤12 or >20, SBP ≤90 mmHg, and $SpO_2$ <95% on ambient air.
  - Patient with ECG that evolves to showing ST elevation or new LBBB. Notify EMS of STEMI so patient is directed to appropriate ED.
  - Patient with ECG showing high-grade heart block or unstable arrhythmia/bradycardia/tachycardia.
  - Patient with suspected ACS and high-risk features, such as continuing chest pain, severe dyspnea, presyncope/syncope, palpitations, and/or evolving ECG findings.
  - Increased clinical suspicion for serious life-threatening conditions, such as ACS, PE, acute chest syndrome, tension pneumothorax, cardiac tamponade, and esophageal rupture.[1,2,3]
- Consider using clinical decision tools to stratify a patient's risk that acute chest pain is due to coronary artery disease (CAD), like INTERCHEST Score (see **Table 1)** or Marburg Heart Score, which are validated for patients seen in a primary care setting.[2,5,6] There are many clinical decision pathways used to aid in medical decision-making for disposition from ED that are mentioned. However, these clinical decision pathways necessitate cTn/hs-cTn results and are only validated for patients that present to ED with acute chest pain. Therefore, these clinical decision pathways cannot be used for CCHCS patient population.[1,2,5,6]
- In general, patients who have "not low" CAD risk by the INTERCHEST Score (2-5) may need to be sent to HLOC for cTn/hs-cTn and further evaluation.
- Consider repeating ECGs at 15-30 minute intervals for patients with baseline abnormal ECG (e.g. LVH, bundle branch block, ventricular pacing) or with new T-wave abnormalities in patients without ongoing chest pain and lower clinical suspicion for ACS (i.e. low INTERCHEST Score of ≤1).
  - NSTE-ACS may demonstrate progressive or evolving ST deviation on ECG, as well as other ECG abnormalities such as T-wave flattening or inversion. At initial presentation of NSTE-ACS, it may be difficult to distinguish between unstable angina and non-ST elevation myocardial infarction (NSTEMI). The distinction between these two types of NSTE-ACS is based on cTn/hs-cTn.

**Table 1: INTERCHEST Score**

This clinical prediction tool is used for acute chest pain in primary care settings to determine if diagnostic workup can be done non-urgently in a stable "low" risk patient or if further testing for ischemia should be done more urgently in a "not low" risk patient.

The INTERCHEST Score should not be used in patients with a readily apparent cause of chest pain (e.g. trauma, infection), clear anginal equivalent symptoms (e.g. jaw pain, dyspnea on exertion, arm pain), or if other testing (e.g. ECG) has suggested a clear cardiac etiology.

| INTERCHEST Score | |
|---|---|
| **Variable** | **Points** |
| History of CAD | 1 |
| Female ≥65 yo or male ≥55 yo | 1 |
| Chest pain related to effort | 1 |
| Pain reproducible by palpation | -1 |
| Provider initially suspected a serious condition | 1 |
| Chest discomfort feels like "pressure" | 1 |

| Score | CAD Risk | Probability of CAD |
|---|---|---|
| ≤1 | Low | 2.1% |
| 2-5 | Not low | 43.0% |

## Evaluation, cont'd

- Consider chest x-ray for further evaluation. Also consider urine drug screen for further evaluation of chest pain etiology, since stimulant use is common in our clinical setting, and patients younger than expected can present with ACS due to recent cocaine or methamphetamine use.[1,2]
- In addition to evaluating for NSTE-ACS, consider that patients with acute chest pain who are found to have PE may present with nonspecific ECG abnormalities. Consider assessing a patient's pretest probability of PE using the Wells Score for PE (see **Table 2**).
  - If a patient has a low probability of PE (Wells Score <2), check PE Rule-out Criteria (PERC) Rule (see **Table 3**) to determine if patient warrants further testing.
  - If a patient has an increased probability of PE (Wells Score ≥2) or PERC Rule is not fulfilled, then consider transfer to HLOC for further workup of PE.[1,2,3]

### Table 2: Wells Score

The Wells Score risk stratifies patients for PE and provides an estimated pre-test probability.

| Wells Score for Pulmonary Embolism (PE) | |
|---|---|
| **Variable** | **Points** |
| Clinical signs and symptoms of deep vein thrombosis (DVT) | 3 |
| PE is top diagnosis OR equally likely | 3 |
| Heart rate > 100 | 1.5 |
| Immobilization for at least 3 days OR surgery in the previous 4 weeks | 1.5 |
| Previous, objectively diagnosed DVT or PE | 1.5 |
| Hemoptysis | 1 |
| Malignancy | 1 |

| | **Risk of PE** | **Probability of PE** |
|---|---|---|
| <2 | Low | 1.3% |
| 2-6 | Moderate | 16.2% |
| >6 | High | 37.5% |

### Table 3: PERC Rule

The Pulmonary Embolism Rule-out Criteria (PERC) Rule was designed to identify patients with low pre-test probability of PE (by Wells Score <2) in whom the risk of unnecessary testing outweighs the risk of PE.

| Pulmonary Embolism Rule-Out Criteria |
|---|
| **Variable** |
| Age <50 |
| Heart rate <100 |
| Oxygen saturation on room air ≥95% |
| No hemoptysis |
| No estrogen use |
| No prior DVT or PE |
| No unilateral leg swelling |
| No surgery/trauma requiring hospitalization within the previous 4 weeks |

## Evaluation, cont'd

### STEP 3: STABLE PATIENT WITH NORMAL ECG

- Continue evaluation with cardiac risk factor assessment, acute chest pain history, and focused physical examination. Consider other serious life-threatening conditions, such as NSTE-ACS, PE, acute chest syndrome, tension pneumothorax, cardiac tamponade, and esophageal rupture.
- Utilize the patient's cumulative burden of traditional and nontraditional cardiac risk factors, acute chest pain history and descriptors, and ECG findings to determine ACS risk.
- Identify prior cardiac testing, if available. The warranty period of prior cardiac testing should be considered when symptoms are unchanged in quality, severity, and frequency from prior episodes of chest pain (see **Table 4**). In patients with recent cardiac testing and normal findings who do not have evidence of ACS, further testing is of limited value, provided that adequate exercise levels were achieved, or pharmacologic testing was performed, imaging was of sufficient quality, and there are no changes in symptom frequency or severity.

**Table 4: Warranty Period for Prior Normal Cardiac Testing**
In patients with recent cardiac testing with normal findings, who do not have evidence of ACS, provided there are no changes in symptom frequency or severity.

| Test Modality | Result | Warranty Period |
|---|---|---|
| Anatomic | Normal coronary angiogram<br>Coronary computed tomographic angiography | 2 years |
| Stress testing | Normal stress test (given adequate stress levels) | 1 year |

- Consider using clinical decision tools to stratify a patient's risk that acute chest pain is due to CAD, like INTERCHEST Score (see **Table 1** on page 5) or Marburg Heart Score, which are validated for patients seen in a primary care setting.[2,5,6] There are many clinical decision pathways used to aid in medical decision-making for disposition from ED that are mentioned. However, these clinical decision pathways necessitate cTn/hs-cTn results and are only validated for patients that present to ED with acute chest pain. Therefore, these clinical decision pathways cannot be used in our patient population.[1,2,5,6]
- In general, patients who have "not low" CAD risk by the INTERCHEST Score (2-5) may need to be sent to HLOC for cTn/hs-cTn and further evaluation. For patients with "low" CAD risk by the INTERCHEST SCORE (0-1), who have a normal ECG and a normal physical examination, consider close follow up at the institution with referral to specialists if they have remained symptom free throughout evaluation and monitoring.[3,4,5]
- If symptoms recur or evolve during a patient's evaluation and monitoring, consider repeating another ECG even if initial ECG was normal.[1,2,3] Compare ECGs to prior baseline ECG, if available. Left ventricular hypertrophy, bundle branch blocks, and ventricular pacing may mask signs of ischemia. On a repeat ECG, NSTE-ACS may demonstrate new ST deviation, as well as other ECG abnormalities such as T-wave flattening or inversion. Consider transfer to HLOC for cTn/hs-cTn and further evaluation for patient with suspected ACS and high-risk features, such as recurring/evolving chest pain, severe dyspnea, presyncope/syncope, palpitations, and/or new/evolving ECG findings.
- Consider chest x-ray for further evaluation. Also consider urine drug screen for further evaluation of chest pain etiology, since stimulant use is common in our clinical setting, and patients younger than expected can present with ACS due to recent cocaine or methamphetamine use.[1,2as]
- Consider assessing for PE using the Wells Score for PE (see **Table 2** on page 6), followed by (PERC) Rule (see **Table 3** on page 6) if Wells Score <2. If a patient has an increased probability of PE (Wells Score ≥2) or PERC Rule is not fulfilled, then consider transfer to HLOC for further workup of PE.

  Proceed with further evaluation and management if there is a clear diagnosis of noncardiac chest pain or if the patient is no longer symptomatic and can be safely managed at the institution with appropriate follow up chest pain.[1,2,3]

## Evaluation, cont'd

### HISTORY

Conduct a history including both personal and family, paying particular attention to the following:

- **Cardiac risk factors:** Identify both traditional risk factors (male, age >45 yo, diabetes mellitus (DM), dyslipidemia, hypertension (HTN), smoking, family history of CAD, prior cardiac history, and/or known CAD, and nontraditional risk factors (see **Table 5**). Also identify prior cardiac testing, such as prior coronary angiogram or stress testing.

  In our clinical setting, stimulant use is common, and patients younger than expected can present with ACS due to recent cocaine or methamphetamine use.[1,2]

**Table 5: Cardiac Risk Factor Assessment**

These are common traditional and non-traditional cardiac risk factors for CAD. In our setting, consider stimulant use as a cause of chest pain, especially among younger patients without traditional risk factors present with ACS. Patients with known CAD, especially those who had recent cardiac revascularization procedures are high risk for recurrent ischemia.[1,2,4,8]

| **Traditional Risk Factors for Coronary Artery Disease** | | | | |
|---|---|---|---|---|
| Male | Age >45 yo | Diabetes | Hypertension | Dyslipidemia |
| Overweight and obesity | Family history of premature CAD | History of stroke/Transient ischemic attack (TIA) | Known CAD | History of myocardial infarction (MI) |
| Smoking history | Poor diet | Sedentary lifestyle | Known peripheral artery disease (PAD) | Prior PCI and/or CABG |
| **Nontraditional Risk Factors for Coronary Artery Disease** | | | | |
| CKD | Premature menopause (before age 40 yo) | Coronary artery spasm | History of chest radiation | Estrogen or oral contraceptive pill use |
| HIV | NAFLD/MASLD | Thyroid disease | Cardiomyopathy | Sickle cell disease (SCD) |
| Chronic inflammatory conditions (e.g. lupus, rheumatoid arthritis) | Stimulant use, e.g. cocaine, methamphetamine | History of pregnancy-associated conditions, e.g. preeclampsia | Elevated biomarkers, e.g. hs-CRP, Lp(a), apoB, ABI | History of COVID-19 |

*Percutaneous coronary intervention (PCI), coronary artery bypass graft (CABG), chronic kidney disease (CKD), human immunodeficiency virus (HIV), non-alcoholic fatty liver disease (NAFLD), metabolic dysfunction-associated steatotic liver disease (MASLD), high-sensitivity C-reactive protein (hs-CRP), lipoprotein (a) (Lp[a]), apolipoprotein B (apoB), ankle-brachial index (ABI), coronavirus disease 2019 (COVID-19)*

- **Chest pain history and descriptors:** Obtain details on the following information that can help better identify potential chest pain etiology (see **Figure 1** and **Table 6** on page 9).[1]

  Remember that some populations present with ACS differently, so consider these differences during rapid assessment (see **Appendix 1**).[1,2,3,4]

## Evaluation, cont'd

**Figure 1: Probability of Ischemia**
Like most visceral discomfort, the sensation produced by cardiac ischemia is characteristically deep, difficult to localize, and usually diffuse. Point tenderness renders cardiac ischemia less likely. Reported symptoms lie somewhere on a continuum of higher or lower probability of cardiac ischemia based on the presence or absence of specific characteristics.[1]

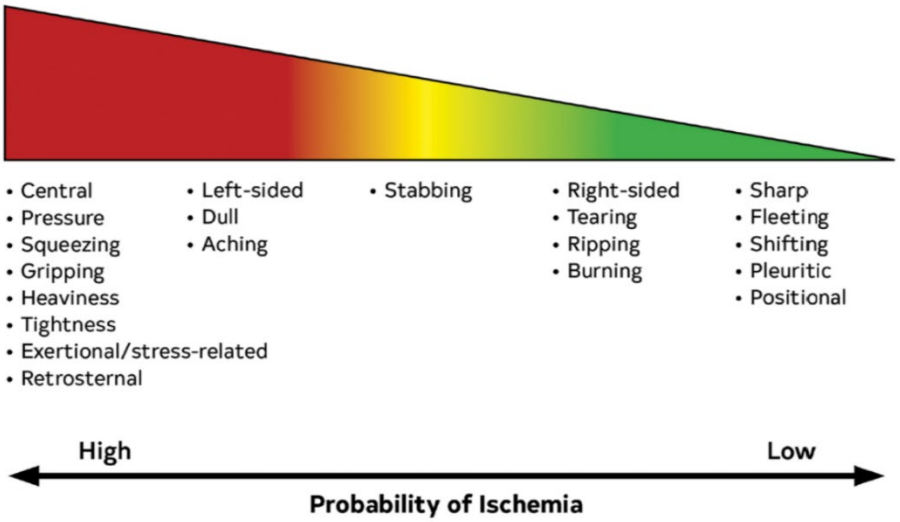



**Table 6: Chest Pain Characteristics**[1]

| Characteristic | Common Corresponding Causes |
|---|---|
| **Nature and Quality** | • Anginal symptoms are perceived as retrosternal chest discomfort (e.g. pain, discomfort, heaviness, tightness, pressure, construction, squeezing).<br>• Sharp chest pain that increases with inspiration and lying supine is unlikely related to cardiac ischemia (i.e. these symptoms usually occur with acute pericarditis). |
| **Onset and Duration** | • Anginal symptoms gradually build in intensity over a few minutes.<br>• Sudden onset of ripping chest pain with radiation to the upper or lower back is unlikely to be anginal but is suspicious for an acute aortic syndrome, which is a serious life-threatening condition.<br>• Fleeting chest pain, which lasts a few seconds in duration, is unlikely to be related to cardiac ischemia. |
| **Location and Radiation** | • Pain that can be localized to a very limited area and pain radiating to below the umbilicus or hip are unlikely related to cardiac ischemia. |
| **Severity** | • Ripping chest pain, often described as "the worst chest pain of my life", especially when sudden in onset and occurring in a patient with uncontrolled hypertension or a patient with a known bicuspid aortic valve or aortic dilation, is suspicious for an acute aortic syndrome, which is a serious life-threatening condition. |
| **Precipitating Factors** | • Physical exercise or emotion stress are common triggers for anginal symptoms.<br>• Occurrence at rest or with minimal exertion associated with anginal symptoms, usually indicates cardiac ischemia.<br>• Positional chest pain is usually nonischemic. |
| **Relieving Factors** | • Relief with nitroglycerin is not necessarily diagnostic of myocardial ischemia and should not be used as a diagnostic criterion. |
| **Associated Symptoms** | • Common symptoms associated with cardiac ischemia include, but are not limited to, dyspnea, palpitations, diaphoresis, lightheadedness, presyncope or syncope, jaw and/or neck pain, and upper abdominal pain with heartburn unrelated to meals or nausea/vomiting.<br>• Symptoms on the left or right side of the chest, stabbing chest pain, sharp chest pain, or discomfort in the throat or abdomen may occur in patients with diabetes, women, and elderly patients. |
| **Special Populations** | • Some populations of patients are less likely to have a "classic" presentation of ACS. For example, patient ≥75 yo may present with shortness of breath, syncope, mental impairment, abdominal pain, or an unexpected fall. See Appendix 1. |

## Evaluation, cont'd

### PHYSICAL EXAMINATION

In patients with acute chest pain, a focused cardiovascular and pulmonary examination should be performed to aid in the diagnosis of ACS or other serious life-threatening causes of chest pain. These focused examinations also help to identify complications and to facilitate rapid implementation of appropriate treatment of serious life-threatening conditions. A normal physical examination does not exclude ACS or other serious life-threatening conditions. Specific findings may be useful in establishing the correct diagnosis (see **Table 7** on page 11-13 and **Table 8** on page 13).

- Vital Signs
- Jugular venous pressure elevation
- Cardiac examination, such as auscultating for S3 gallop, new murmurs, friction rub, etc.
- Chest wall examination, such as looking for new rash over area of pain, point tenderness to palpation, etc.
- Pulmonary examination, such as increased work of breathing, crackles, absent breath sounds, etc. [1,2,3,9]

### CHEST X-RAY

In patients presenting with acute chest pain, a chest radiograph is useful to evaluate for other potential cardiac, pulmonary, and thoracic causes. Do not delay transfer to HLOC to obtain chest x-ray. Typically, chest x-ray is normal in ACS, unless heart failure is present. This can be done to identify other serious life-threatening causes of chest pain, such as acute chest syndrome in patients with SCD.[3,8] See **Health Equity Alert Box**.

### MEDICAL DECISION-MAKING

Transfer to HLOC for patients who are clinically unstable, patients who have altered level of consciousness, patients with ECG showing STEMI, patients with ECG showing new LBBB, and patients in whom there is a high clinical suspicion for a serious life-threatening cause of chest pain.

Also consider transfer to HLOC for patients with chest pain of uncertain etiology who may benefit from cTn/hs-cTn and further urgent evaluation. The following table summarizes many of the important identifiers and findings of various causes of serious life-threatening acute chest pain
(see **Table 8** on page 13).

### HEALTH EQUITY ALERT

SCD affects 1 out of 365 Black Americans and 1 out of 16,300 Latinos in the United States (US). As these populations are overrepresented in US carceral settings, it is essential that providers in these setting understand SCD and its complications.

An acute complication of SCD is acute chest syndrome, which is the leading cause of mortality among patients with SCD and accounts for a quarter of deaths. It can be caused by vaso-occlusion, ischemic, and endothelial injury within the pulmonary microvasculature.

A diagnosis of acute chest syndrome in a patient with SCD is made with the following criteria:

- New pulmonary infiltrates on chest x-ray involving at least one lung segment

and one of the following symptoms:

- Chest pain
- Temperature more than 38.5°C
- Tachypnea, wheezing, rales, coughing, evidence of increased work of breathing
- Hypoxemia relative to baseline, which is more than 2% decrease in oxygen saturation at room air[8]

Clinically stable patients who have acute chest pain who are determined to have a "low" risk of CAD may not require immediate transfer to HLOC, and further outpatient evaluation is generally safe and appropriate. Outpatient evaluation for CAD for these "low" risk patients may be done with an exercise or pharmacology stress test. Consider outpatient evaluation for other non-ischemic and non-cardiac etiologies, as clinically indicated.

If there is a readily apparent cause of chest pain, further evaluation, treatment, and management should be directed to these causes as clinically appropriate.

Shared decision-making is crucial, and patients should be actively involved in treatment decisions based on their individual needs, preferences, and understanding of the benefits, risks, and alternatives of various treatment options.[1,2,3,9] See patient education attachments PE-1 through PE-2 for details.

## Evaluation, cont'd

**Table 7: Characteristics of Serious Life-Threatening Chest Pain**[1]

| Differentiation of Serious Life-Threatening Cause of Acute Chest Pain | | | | |
|---|---|---|---|---|
| **Diagnosis** | **Chest Pain Descriptors** | **Physical Examination Findings** | **Electrocardiogram** | **Chest X-ray** |
| **ACS** | • Substernal/left sided chest pressure or tightness<br>• Onset often gradual<br>• Pain radiating to shoulders or pain with exertion increases relative risk<br>• "Nonclassic" symptoms (e.g., dyspnea, weakness) more common in elderly, women, diabetics<br>• Assume symptoms of ACS or mechanical complication of MI within days or a few weeks of PCI or CABG is from an occluded artery or graft | • Diaphoresis<br>• Tachypnea<br>• Tachycardia, hypotension, crackles, S3, mitral regurgitation murmur<br>• Examination may be normal in uncomplicated cases | • ST segment elevations in ≥2 contiguous leads, Q waves, new LBBB are evidence of acute MI<br>• Single ECG is not sensitive for ACS and does NOT exclude ACS if initially normal<br>• Prominent R waves with ST segment depressions in V1 and V2 strongly suggest posterior MI | • Nonspecific<br>• May show evidence of heart failure |
| **Mechanical complications of MI** | • Complications of MI declined with advances in reperfusion, but mortality remains high between 10-40%<br>• Ventricular septal rupture can occur 24 hours to 2 weeks after MI; free wall rupture can occur 5 days to 2 weeks after MI; and papillary muscle rupture can occur 2-7 days after MI<br>• Presentation ranges from acute or recurrent chest pain with dyspnea on exertion to severe cardiogenic shock<br>• Assume symptoms of ACS or mechanical complication of MI within days or a few weeks of PCI or CABG is from an occluded artery or graft | • Ventricular septal rupture can present as hypotension, tachycardia, with new murmur that is harsh, loud, and holosystolic<br>• Free wall rupture can present as sudden profound right heart failure and shock causing death, but subacute rupture can present as hypotension, tachycardia<br>• Papillary muscle rupture can present as acute onset hypotension, severe pulmonary edema, and new mitral regurgitation murmur | • Ventricular septal rupture may show heart block, ST segment elevation<br>• Free wall rupture may range from features of pericarditis to sudden bradycardia then pulseless electrical activity from tamponade<br>• Papillary muscle rupture may show ST segment elevation | • Ventricular septal rupture may reveal left ventricular enlargement and pulmonary edema<br>• Free wall rupture may show cardiomegaly with clear lung fields<br>• Papillary muscle rupture may reveal pulmonary edema |
| **Cardiac tamponade** | • Pain from pericarditis often sharp anterior chest pain made worse by inspiration or lying supine, leaning forward<br>• Pain relieved by sitting forward<br>• Dyspnea, fatigue common<br>• Presyncope or syncope<br>• Pericardial effusion and pericarditis in renal failure and dialysis patients | • Tamponade creates obstructive shock and hemodynamic instability with findings of tachycardia, hypotension, elevated jugular venous pressure, pulsus paradoxus<br>• Muffled heart sounds<br>• Pericardial effusion can cause friction rub | • Decreased QRS amplitude and electrical alternans can appear with significant effusions<br>• Diffuse PR segment depressions with or without ST segment elevations in pericarditis | • May reveal enlarged cardiac silhouette |

## Evaluation, cont'd

| Differentiation of Serious Life-Threatening Cause of Acute Chest Pain | | | | |
|---|---|---|---|---|
| **Diagnosis** | **Chest Pain Descriptors** | **Physical Examination Findings** | **Electrocardiogram** | **Chest X-ray** |
| **Acute aortic syndrome, such as aortic dissection** | • Sudden onset of severe, sharp, tearing, or ripping pain<br>• Maximal severity at onset<br>• Most often begins in chest radiating to the back<br>• >10% present as syncope<br>• Can mimic stroke, mesenteric ischemia, ACS, kidney stone, among others, depending on branch arteries involved<br>• Consider in connective tissue disorders (e.g. Ehlers-Danlos or Marfan syndrome) | • Absent pulse in upper extremity or carotid pulse<br>• Extremity pulse discrepancy in SBP > 20 mmHg between right and left arm (30% of patients, type A>B)<br>• Up to 30% with neurologic findings<br>• Findings vary depending on branch arteries involved<br>• Aortic regurgitation murmur in 40-75% (type A) | • Ischemic changes in 15%<br>• Nonspecific ST and T changes in 30% | • Up to 76% with wide mediastinum or loss of normal aortic knob contour<br>• 10% with normal chest x-ray<br>• Abrupt onset, severe pain + pulse differential + wide mediastinum has >80% probability of aortic dissection |
| **Infective endocarditis** | • Suspect in patients with bacteremia, relevant cardiac risk factors (prior IE, history of valvular or congenital heart disease), or other predisposing conditions (intravenous drug use, indwelling cardiac device or intravenous catheter, immunosuppression, or a recent dental or surgical procedure)<br>• Fever, chills, anorexia, weight loss, night sweats, dyspnea | • Fever patterns vary<br>• Up to 85% with new or changing murmur<br>• Supportive signs: splenomegaly, cutaneous manifestations such as petechiae or splinter hemorrhages | • May demonstrate new or evolving conduction disease, such as first or second-degree atrioventricular block, bundle branch block, or complete heart block | • Nonspecific<br>• May show opacities suggestive of underlying pneumonia and septic pulmonary embolic<br>• May show pulmonary congestion in patients with left sided valve leaflet destruction |
| **Pulmonary embolism** | • Many possible presentations, including pleuritic chest pain and painless dyspnea<br>• Often sudden onset<br>• Dyspnea often dominant symptom | • No finding is sensitive or specific<br>• Tachycardia<br>• Extremity exam may be normal<br>• Lung exam nonspecific, but tachypnea common<br>• Focal wheezing may be present | • Usually abnormal but nonspecific<br>• Evidence of right heart strain (e.g. RAD, RBBB, RAE)<br>• Sinus tachycardia, T wave inversion in anterior leads with pathognomonic S1Q3T3 (S wave in lead I, Q wave in lead III, T wave inversion in lead III) | • Majority normal<br>• May show atelectasis, hemidiaphragm, pleural effusion |

## Evaluation, cont'd

| Differentiation of Serious Life-Threatening Cause of Acute Chest Pain | | | | |
|---|---|---|---|---|
| **Diagnosis** | **Chest Pain Descriptors** | **Physical Examination Findings** | **Electrocardiogram** | **Chest X-ray** |
| **Tension pneumothorax** | • Often sudden onset<br>• Initial pain often sharp and pleuritic<br>• Dyspnea often dominant<br>• Can be traumatic from blunt or penetrating injury<br>• Can be atraumatic | • Ipsilateral diminished or absent breath sounds<br>• Labored breathing or accessory muscle use<br>• Hemodynamic compromise with tachycardia, hypotension<br>• Subcutaneous emphysema uncommon | • Nonspecific<br>• May demonstrate shift of axis | • Demonstrates air in pleural space<br>• May show rib fracture from blunt or penetrating trauma |
| **Esophageal rupture causing mediastinitis** | • Forceful vomiting often precedes esophageal rupture<br>• Recent upper endoscopy or instrumentation increases risk | • Ill-appearing, shock, fever<br>• May hear Hamman's crunch over mediastinum<br>• 20% with pneumothorax with unilateral diminished or absent breath sounds<br>• Subcutaneous emphysema uncommon | • Nonspecific | • Large majority have abnormal chest x-ray<br>• Pneumomediastinum<br>• Pleural effusion<br>• Pneumothorax |
| **Acute chest syndrome** | • Chest pain, shortness of breath, and fever in patient with SCD<br>• Cough | • Fever ≥38.5°C<br>• >3% decrease in oxygen saturation ($SpO_2$)<br>• Tachypnea with intercostal retractions, nasal flaring, or accessory muscle use<br>• Wheezing, rales | • Nonspecific | • New pulmonary radiodensity involving at least one complete lung segment |

*Acute coronary syndrome (ACS), myocardial infarction (MI), percutaneous coronary intervention (PCI), coronary artery bypass graft (CABG), left bundle branch block (LBBB), electrocardiogram (ECG), systolic blood pressure (SBP), infective endocarditis (IE), right axis deviation (RAD), right bundle branch block (RBBB), right atrial enlargement (RAE), sickle cell disease (SCD)*

**Table 8: Characteristics of Other Chest Pain Etiologies to Consider in Patients with Acute Chest Pain[1]**

| Other Chest Pain Etiologies | Characteristics |
|---|---|
| Noncoronary cardiac: AS, AR, HCM | AS: Characteristic systolic murmur, tardus or parvus carotid pulse<br>AR: Diastolic murmur at right of sternum, rapid carotid upstroke<br>HCM: Increased or displaced left ventricular impulse, prominent A wave in jugular venous pressure, systolic murmur |
| Pericarditis | Fever, pleuritic chest pain, increased in supine position, friction rub |
| Myocarditis | Fever, chest pain, heart failure, S3 |
| Esophagitis, peptic ulcer disease, gallbladder disease | Epigastric tenderness or right upper quadrant tenderness, Murphy sign |
| Pneumonia | Fever, localized chest pain, may be pleuritic, friction rub may be present, regional dullness to percussion, egophony |
| Pneumothorax | Dyspnea and pain on inspiration, unilateral absence of breath sounds |
| Costochondritis, Tietze syndrome | Tenderness of costochondral joints |
| Herpes zoster | Pain in dermatomal distribution, triggered by touch; characteristic rash (unilateral and dermatomal distribution) |

*Aortic stenosis (AS), aortic regurgitation (AR), hypertrophic cardiomyopathy (HCM), third heart sound (S3)*

## Evaluation, cont'd

### DOCUMENTATION

When documenting a complaint of chest pain, distinguish **acute chest pain** from **stable chest pain**. Chest pain is considered acute when it is new in onset or involves a change in pattern, intensity, or duration compared to previous episodes in a patient with recurrent symptoms. Chest pain is considered stable when symptoms are chronic and associated with consistent precipitants, such as exertion or emotional stress.

Next, document chest pain is **cardiac**, **possibly cardiac**, or **noncardiac** because these terms are more specific to the potential underlying diagnosis. Avoid describing chest pain as "atypical" because it is not helpful in determining the cause and can be misinterpreted as benign in etiology.

Then, cardiac chest pain can be further differentiated as **ischemic** or **nonischemic**. Ischemic chest pain can be caused by stable angina, NSTE-ACS, or STEMI. Nonischemic cardiac chest pain may be due to aortic pathology, pericardial effusion, endocarditis, or other etiologies.[1,9]

Finally, clear, accurate, and timely documentation of medical decision making to relay the events, and the thought process allows the entire care team, including those at HLOC, to seamlessly follow through with an appropriate care plan.

## INITIATION OF THERAPY DURING EVALUATION PROCESS

1. Get STAT ECG, and monitor cardiac rate, cardiac rhythm, and oxygen saturation.
2. Provide supplemental O2 at 2-6 L/minute via nasal cannula, 6-10 L/minute via face mask, or 15 L/minute via non-rebreather mask to maintain oxygen saturation ≥95% (See **Table 9** on page 15). Note the limited accuracy of pulse oximetry on darker pigmented skin. See **Health Equity Alert Box**.
3. Establish IV access with large bore needle and infuse 0.9% sodium chloride intravenous solution at 30 cc/hour.
4. Chew non-enteric-coated aspirin 325 mg 1 tablet unless the patient is allergic to aspirin or is actively bleeding.
5. Nitroglycerin 0.4 mg 1 tablet sublingually every 5 minutes for 3 doses. If symptoms do not resolve after 1st dose, consider transfer to HLOC. Avoid nitroglycerin if SBP <90 mmHg, if SBP >30 mmHg below baseline blood pressure, if recent use of 5'-phosphodiesterease (PDE) inhibitors within the past 24-48 hours, or if right ventricular (RV) infarction is suspected.
6. For patients awaiting transfer to HLOC by EMS, print emergency transfer report completed by nursing (chest pain history, vital signs, physical exam, and treatment given), recent ECG, and previous ECG (if available) to send with patient.
7. For patients managed at institution who do not have a clinical indication for transfer to HLOC, consider repeat ECG at 15-30 minute intervals if clinically indicated.
8. If there is a readily apparent cause of chest pain, treatment and management should be directed to these causes as clinically appropriate.[1,9]

### HEALTH EQUITY ALERT

Pulse Oximeters and Skin Pigmentation

- ✓ Be aware that pulse oximeters have limited accuracy, which can be worsened in those with poor circulation, current tobacco use, or more skin pigmentation.
- ✓ Think of pulse oximeter results as an estimate with a 4-5% margin of error on both sides of the result. For example, a pulse oximeter saturation of 90% may represent an arterial blood saturation of 86-94%. This range is wider for those with more skin pigment.
- ✓ Do not be reassured by a normal oxygen saturation if the clinical picture does not match. Assess for other signs of desaturation.
- ✓ If possible, make clinical decisions based on trends of pulse oximeter readings over time rather than point in time results.

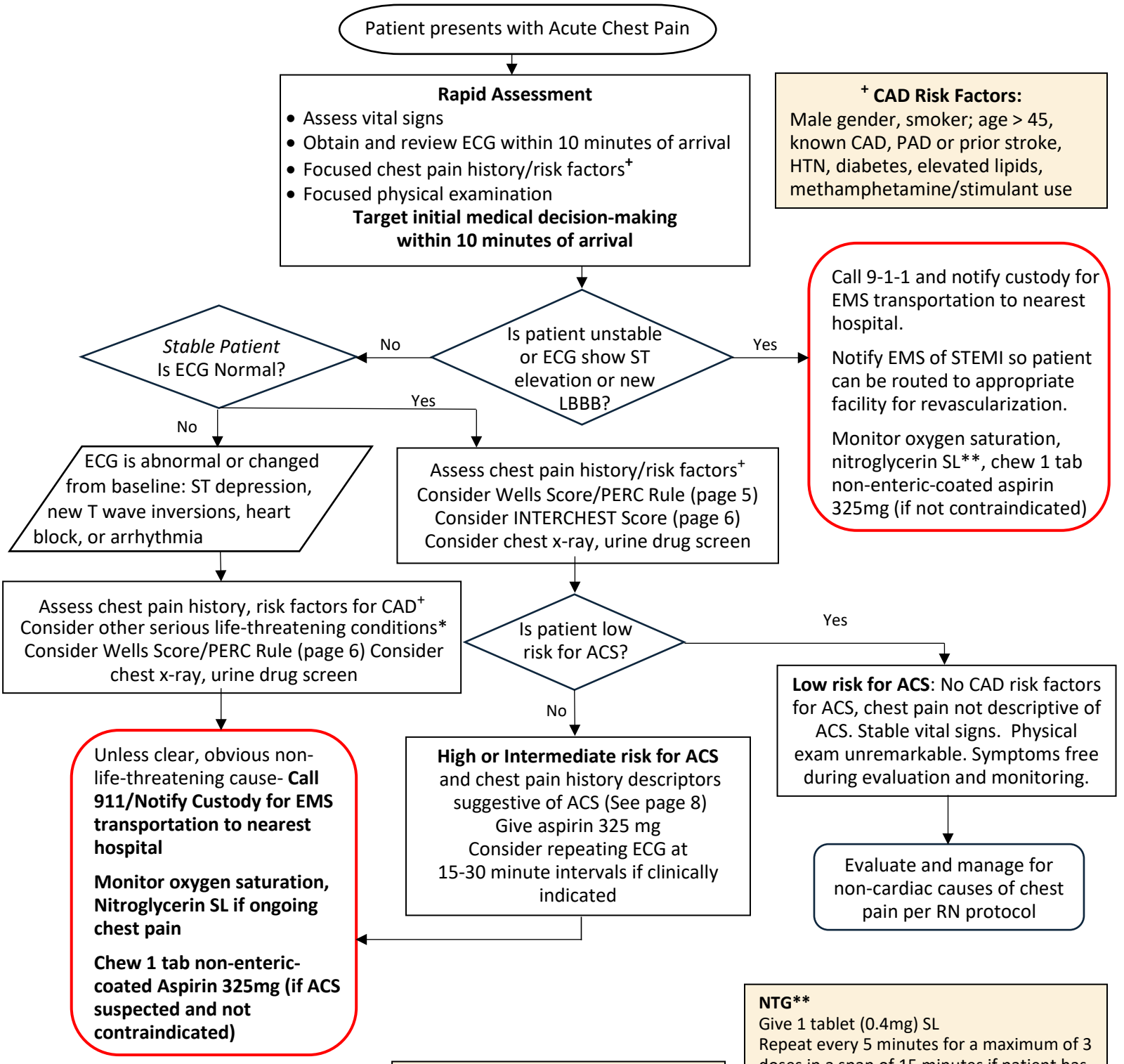
## Initiation of Therapy During Evaluation Process, cont'd

**Table 9: Therapies to Initiate During Evaluation Process**[1,9]

| Treatment | Dose and Treatment Goals | Notes |
|---|---|---|
| Aspirin (acetylsalicylic acid)<br><br>Non-Enteric Coated Oral Tablet: 325 mg | Emergency Management of ACS<br>• Loading Dose: 325 mg non-enteric coated tablet, chewed and swallowed immediately (including patients taking chronic low-dose aspirin therapy and irrespective of treatment strategy)<br>• May use rectal suppository (if available) for patients who cannot take orally<br>• Give within minutes of arrival<br>• Higher doses (1000 mg) interfere with prostacyclin production and may limit positive benefits | • Administer to all patients with suspected ACS unless contraindicated<br>• Use with caution in patients with active ulcer disease<br>• Contraindicated in patients with known hypersensitivity to aspirin |
| Nitroglycerin (Nitrostat®)<br><br>Sublingual Tablet: 0.4 mg | • Take one tablet under the tongue every 5 minutes as needed for chest pain<br>• May repeat every 5 minutes<br>• No more than 3 tablets are recommended during a 15-minute period<br>• If symptoms do not resolve after 1st dose, consider transfer to HLOC | • Initial anti-anginal for suspected ischemic pain<br>• No mortality benefit<br>• Hypertensive urgency with ACS.<br>Precautions:<br>• Patient with evidence of acute MI, limit systolic blood pressure to drop 10% if patient is normotensive, 30% drop if hypertensive, and avoid drop below 90 mm Hg<br>• Patient should sit or lie down with receiving this medication<br>Contraindications:<br>• Hypotension (SBP <90 mmHg or SBP >30 mmHg below baseline blood pressure)<br>• Recent (within 24-48 hours) 5'-PDE inhibitor use (e.g., sildenafil)<br>• Severe bradycardia or severe tachycardia<br>• Suspected RV infarction |
| Oxygen | • Keep oxygen saturation ≥95% | • Any suspected cardiopulmonary emergency, especially (but not limited to) complaints of shortness of breath and suspected ischemic chest pain<br>• Pulse oximetry provides a useful method of titrating oxygen administration to maintain physiologic oxygen saturation<br>Precautions:<br>• Pulse oximetry inaccurate in low cardiac output states or with vasoconstriction.<br>• Observe closely when using with pulmonary patients known dependent on hypoxic respiratory drive (rare)<br>• Routine use of oxygen did not decrease the individual risks of all-cause death, recurrent ischemia or MI, heart failure, or occurrence of arrhythmia events. |

See prescribing information for complete description of contraindications/precautions, adverse effects, and drug interactions.

## ACUTE CHEST PAIN ALGORITHM




**ACS:** Acute coronary syndrome
**CAB:** Circulation, airway, breathing
**CAD:** Coronary artery disease **DM:** diabetes mellitus
**EMS:** Emergency medical services
**LBBB:** Left bundle branch block
**NSTE-ACS:** Non-ST elevated ACS
**PAD:** Peripheral artery disease

***Suspect other serious life-threatening conditions**
Acute coronary syndrome/STEMI
Acute aortic dissection
Pulmonary embolism
Tension pneumothorax
Pericardial tamponade
Mediastinitis (e.g., esophageal rupture)

**NTG****
Give 1 tablet (0.4mg) SL
Repeat every 5 minutes for a maximum of 3 doses in a span of 15 minutes if patient has persistent chest discomfort, HTN, or signs of heart failure AND there is no sign of hemodynamic compromise (e.g., right ventricular infarction, drop in SBP ≤ 90 mm Hg, HR ≤ 50 or ≥ 100 min, RR ≤ 12 or > 20 min) (See page 4 CLINICAL STABILITY) and no use of 5'-PDE inhibitors (e.g., for erectile dysfunction and pulmonary HTN)

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1. Writing Committee Members, et al. "2021 AHA/ACC/ASE/CHEST/SAEM/SCCT/SCMR guideline for the evaluation and diagnosis of chest pain: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines." Journal of the American College of Cardiology 78.22 (2021): e187-e285.
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3. McConaghy, John R., Malvika Sharma, and Hiten Patel. "Acute chest pain in adults: outpatient evaluation." *American Family Physician* 102.12 (2020): 721-727.
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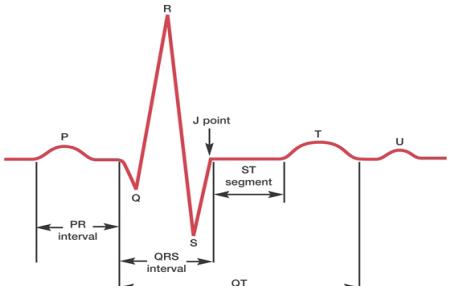
6. Manten, A., et al. "Evaluation of the Marburg Heart Score and INTERCHEST score compared to current telephone triage for chest pain in out-of-hours primary care." Netherlands Heart Journal 31.4 (2023): 157-165.
7. Damluji, Abdulla A., et al. "Mechanical complications of acute myocardial infarction: a scientific statement from the American Heart Association." Circulation 144.2 (2021): e16-e35.
8. Bhasin, Neha, and Ravi Sarode. "Acute Chest Syndrome in Sickle Cell Disease." Transfusion Medicine Reviews (2023): 150755.
9. Amsterdam, Ezra A., et al. "2014 AHA/ACC guideline for the management of patients with non–ST-elevation acute coronary syndromes: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines." Journal of the American College of Cardiology 64.24 (2014): e139-e228.

## Appendix 1

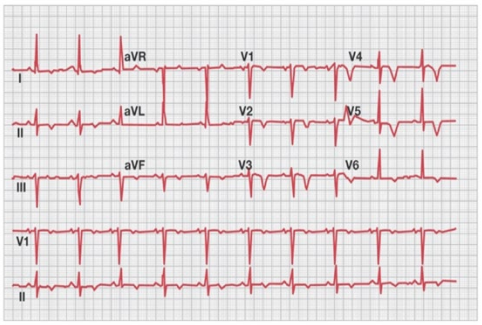
| **Presentation of Acute Coronary Syndrome in Special Populations (AHA/ACC 2021)[1,3]** | |
|---|---|
| **Elderly age > 75 years[1]** | ➢ In patients with chest pain who are > 75 years of age, ACS should be considered when accompanying symptoms such as shortness of breath, syncope, or acute delirium are present, or when an unexplained fall has occurred. |
| **Women[1,4]** | ➢ Women who present with chest pain are at risk for underdiagnosis, and potential cardiac causes should always be considered.<br>➢ In women presenting with chest pain, it is recommended to obtain a history that emphasizes accompanying symptoms that are more common in women with ACS. While chest pain remains the predominant symptom reported by women among those ultimately diagnosed with ACS, women are less likely to have timely and appropriate care.<br>• This could be explained by the fact that women are more likely to experience prodromal symptoms when they seek medical care.<br>• Women may also present with accompanying symptoms (e.g., nausea, fatigue, and shortness of breath) more often than men.<br>• In the PROMISE study, women commonly presented with chest pain symptoms similar to men but also had a greater prevalence of other symptoms such as palpitations, jaw and neck pain, as well as back pain. |
| **Diverse Patient Populations with Chest Pain[1]** | ➢ Cultural competency training is recommended to help achieve the best outcomes in patients of diverse racial and ethnic backgrounds who present with chest pain.<br>➢ Among patients of diverse race and ethnicity presenting with chest pain in whom English may not be their primary language, addressing language barriers with the use of formal translation services is recommended.<br>➢ Young people (age < 55 yrs) presenting to the ED with chest pain, women, and people of color waited longer to be evaluated by a provider, independent of other clinical features, compared with men and White adults, respectively. |
| **Patient with Sickle Cell Disease and on Hemodialysis[1,8]** | ➢ Patients with sickle cell disease and hemodialysis who experience chest pain, ACS is associated with significant morbidity and mortality rates. These patients should be transferred to an acute care setting by EMS when there is clinical suspicion of ACS. |
| **Patients with history of stimulant use cocaine and methamphetamine [1]** | ➢ Chest pain is a common complaint, nonspecific, significant tachycardia, BP may be high but can be low once cases progress to cardiogenic shock.<br>➢ Have significantly greater odds of multiple readmissions (3 or more admissions in less than 6 months).<br>➢ Clinical evidence of other methamphetamine related, noncardiac findings, including cachexia, diaphoresis, track marks or excoriations, excessive talking, dry mouth with tooth decay.<br>➢ ECG: tachyarrhythmia, right axis deviation, lateral T-wave inversion, P pulmonale, LV hypertrophy, QT prolongation, and inferior Q waves. |
| **COVID-19 patients[4]** | ➢ Symptoms of chest pain or tightness are common in patients with an active COVID-19 infection. Pain is usually not well localized and may be associated with shortness of breath due to COVID-19 pneumonia.<br>➢ Potential mechanisms of cardiovascular injury include direct myocardial injury from hemodynamic derangement or hypoxemia, inflammatory myocarditis, stress cardiomyopathy, microvascular dysfunction or thrombosis due to hypercoagulability, or systemic inflammation (cytokine storm), which may destabilize coronary artery plaques.[4]<br>➢ ECG criteria are not specific for coronary artery thrombosis, particularly in COVID-19 patients in whom ST elevation may occur with stress cardiomyopathy or possibly myocarditis. Thus, noncoronary artery COVID-19-associated myocardial injury needs to be carefully considered as a diagnostic possibility before reperfusion therapy is considered.<br>➢ In most patients with ST elevation, the diagnosis will be thrombotic occlusion of a coronary artery. |

## Appendix 2

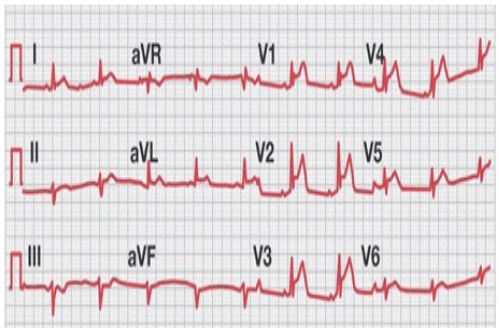
### Important ECG Changes



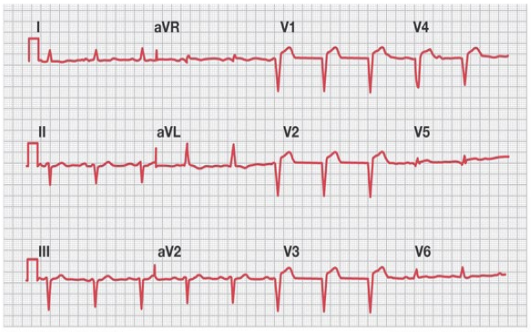

Features of ECG changes that increase probability of ACS in patients with chest pain: New ST segment elevation, Q waves, conduction defect, ST segment depression or T wave inversion



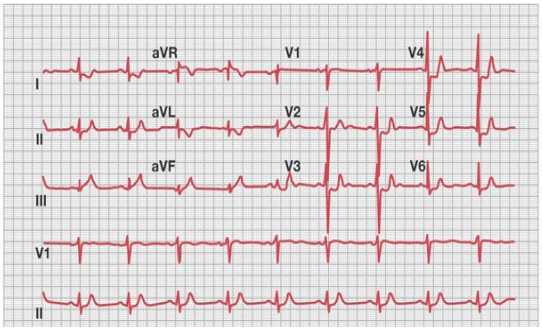

**T wave inversion**



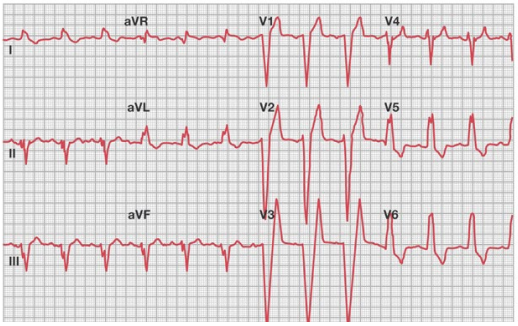

**Anteroseptal MI showing ST elevation**



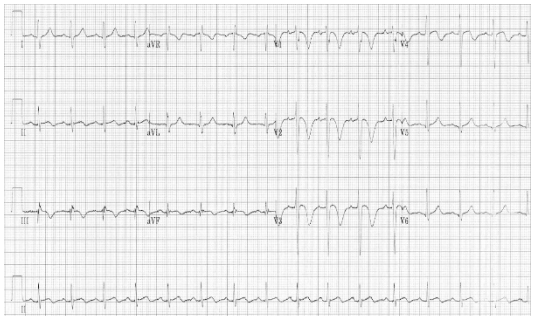

**Q waves in V1-V4**



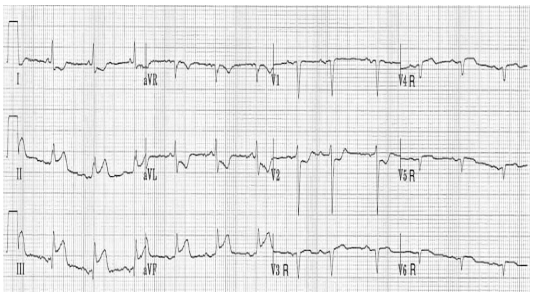

**ST segment depression I, V3-V6**




**Left Bundle Branch Block**



**Pulmonary embolus**:
Sinus tachycardia. Simultaneous T-wave inversions in the anterior (V1-4) and inferior leads (II, III, aVF). Non-specific ST changes – slight ST elevation in III and aVF.




**RV infarction is diagnosed:**
There is an inferior STEMI with ST elevation in lead III > lead II. V1 is isoelectric while V2 is significantly depressed. There is ST elevation throughout the right-sided leads V3R-V6R.

Conquering the EKG Cardiology Explained. Ashley EA, Niebauer J

# PATIENT EDUCATION

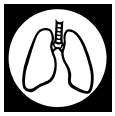
## CHEST PAIN: WHAT YOU SHOULD KNOW

**Q: Does all chest pain come from your heart?**

**A: No, chest pain can come from different parts of your body, including your muscles, bones, digestive tract, lungs, or heart.**

- Chest pain often comes from injuries or strains to the muscles, ribs, and bones in your chest.
  - "Musculoskeletal" pain is more common after a lot of exercise or doing something physical you are not used to doing.
  - Musculoskeletal chest pain is usually in one spot in the chest and hurts more if you move that area.

- Your lungs can also cause chest pain. Lung or "pulmonary" chest pain can come from inflammation (irritation) of the lungs
  - Often you may have cough, fever or you may be coughing up phlegm (sputum).



- Your digestive tract (including your esophagus-or food pipe which travels from your mouth to your stomach) can cause chest pain.
  - When chest pain is caused by a lot of acid in your stomach it is called GERD or heartburn.
  - Chest pain from your digestive tract is often felt under your breastbone (sternum) and gets better with antacid medication.
  - If you are short of breath or sweaty from the pain, you should contact medical right away even if you think the pain is heartburn

**Q: Am I having a heart attack?**

**A: A heart attack occurs when heart muscle dies because of blockage of the blood supply to the heart, usually because of a build-up of cholesterol. Most people think a heart attack is sudden and intense, like a "movie heart attack." The fact is that many heart attacks start slowly as mild pain or discomfort, and those who experience it may not know what is wrong.**

**Warning signs of a heart attack include:**

- Chest pain or discomfort (pressure, squeezing, fullness, or pain in the center of the chest).
- Pain or discomfort in one or both arms, back, neck, jaw, or stomach.
- Shortness of breath (often comes with or before chest discomfort).
- Breaking out in a cold sweat, nausea, or light headedness.
- **Waiting is dangerous. Minutes matter**. Anyone with heart attack symptoms should not wait more than five minutes to get medical help.

**Q: What if I have a heart attack?**

**A: Most people do not die when they have a heart attack. New medications and treatments work well. The faster you get medical help the better chance you will live and will do well.**

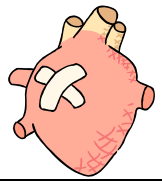
**Coronary Heart Disease**

Coronary heart disease (CHD) is the name for when your heart muscle does not get enough blood. CHD can give you chest pain.

CHD comes from a blockage in the arteries of the heart (most often caused by cholesterol related blockages).

You likely have CHD if:

- You have had a heart attack.
- You have angina (chest pain that comes from your heart).
- You have had a "balloon" reopen one of your coronary arteries.
- You have had coronary bypass surgery.



## EDUCACIÓN DEL PACIENTE

## DOLOR DE PECHO: LO QUE DEBE SABER

**P: ¿Todo el dolor de pecho viene de su corazón?**

**R: No, el dolor de pecho puede venir de diferentes partes de su cuerpo, incluyendo susu músculos, huesos, aparato digestivo o corazón.**

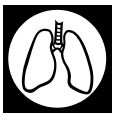
♦El dolor de pecho a menudo proviene de lesiones o distensiones en los músculos, las costillas y los huesos del pecho.

- El dolor "musculoesquelético" es más común después de hacer mucho ejercicio o algo físico a lo que no está acostumbrado.

- El dolor de pecho musculoesquelético generalmente se presenta en un punto del pecho y duele más si mueve esa área.

♦Sus pulmones también pueden causar dolor en el pecho. El dolor de pecho "pulmonar" puede provenir de la inflamación (irritación) de los pulmones

- A menudo puede tener tos, fiebre o puede estar tosiendo flema (esputo).



♦Su aparato digestivo (incluido el esófago o tubo de alimentación que viaja desde la boca hasta el estómago) puede causar dolor en el pecho.

- Cuando el dolor de pecho es causado por una gran cantidad de ácido en el estómago, se denomina ERGE o acidez estomacal.
- El dolor de pecho del aparato digestivo a menudo se siente debajo del esternón y mejora con medicamentos antiácidos.
- Si le falta el aire o suda por el dolor, debe comunicarse con un médico de inmediato, incluso si cree que el dolor es acidez estomacal.

**P: ¿Me está dando un ataque al corazón?**

**R: Un ataque al corazón ocurre cuando el músculo del corazón muere debido a la obstrucción del suministro de sangre al corazón, generalmente debido a una acumulación de colesterol. La mayoría de la gente piensa que un ataque al corazón es repentino e intenso, como un "ataque al corazón de película". El hecho es que muchos ataques al corazón comienzan lentamente como un dolor o una molestia leves, y es posible que quienes los experimentan no sepan qué es lo que está mal.**

**Los síntomas de alerta de un ataque al corazón incluyen:**

- Dolor o malestar en el pecho (presión, opresión, plenitud o dolor en el centro del pecho).
- Dolor o malestar en uno o ambos brazos, espalda, cuello, mandíbula o estómago.
- Dificultad para respirar (a menudo viene con o antes del malestar en el pecho).
- Inicio repentino de sudor frío, náuseas o mareos.
- **Es peligroso esperar. Minutos pueden hacer la diferencia**. Cualquier persona con síntomas de un ataque al corazón no debe esperarmás de cinco minutos para obtener ayuda médica.

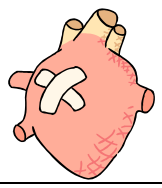
**P: ¿Qué pasa si me está dando un ataque al corazón?**

**R: La mayoría de las personas no mueren cuando les da un ataque al corazón. Los Nuevos medicamentos y tratamientos funcionanbien. Mientras más rápido obtenga ayuda médica, mayor será su probablilidad de vivir yque le vaya bien.**

**Cardiopatía coronaria**

La cardiopatía coronaria (CHD, por sus siglas en inglés) es el nombre que se le da cuando el músculo cardíaco no recibe suficiente sangre. CHD puede causarle dolor en el pecho. CHD proviene de un bloqueo en las arterias del corazón (casi siempre causado por bloqueos relacionados con el colesterol).

Es probable que tenga CHD si:



- Tuvo un ataque al corazón
- Tiene angina (dolor en el pecho que proviene del corazón).
- Se la ha puesto un "globo" para volver a abrir una de sus arterias coronarias.
- Ha tenido una cirugía de bypass de la arteria coronaria.