Chapter 1
Chest Pain in the ER

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1.1 The Scope of the Problem

Chest pain (CP) management is one of the biggest challenges in the emergency room (ER), being the second most common cause of ER presentation among adults in the United States [1, 2]. Causes of this symptom range from musculoskeletal CP to potentially life-threatening emergencies, such as coronary artery disease (CAD) [2]. For this reason, good clinical evaluation is mandatory; although most patients presenting with classical CP and accompanying symptoms are easily diagnostic-oriented, there is an important fraction of patients that will not have the typical presentation. It is essential to accurately stratify risk for this patients to improve ER efficiency and avoid unnecessary tests and admissions [3].

1.2 Prevalence

CP accounts for 5.5 million (9%) of all noninjury-related ER visits for adults in the United States each year [1]. This symptom accounts from 5% to 20% of all ER admissions [2]. Among those without diagnostic ECGs and/or cardiac biomarkers, only 1–4% have angiographic evidence of significant CAD [4], so although CP is related to very serious complications, most of its causes are non-cardiac of origin. Although one of the most urgent and treatable causes for CP is the acute coronary syndrome (ACS), it only accounts for a small percentage (9%) of all the ER visits with this symptom [1], and it is the cause with more fatal-preventable "management."

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1.3 High-Clinical Suspicion for Cardiac-Related Causes of CP in the ER

Clinicians in the ER must focus on the immediate recognition and exclusion of life-threatening causes of CP, although patients with life-threatening etiologies may appear deceptively well, manifesting neither vital sign nor physical examination abnormalities [5]. Therefore, the recognition of the cause of CP based on the patients' medical history and semiology of the symptom is imperative. Since there are several causes for CP, and some patients will have atypical signs, clinicians should be able to suspect a cardiac cause if it presents with coronary risk factors, typical pain characteristics, and ECG findings positive for ACS [6]. In all age ranges, an ischemic chest pain (see below) should suggest structural or nonstructural heart disease. In young, middle age, or elderly population, physicians in charge should be in warning about pulmonary arterial hypertension, hypertrophic cardiomyopathy, congenital coronary abnormalities, pulmonary embolism, ischemic heart disease, Takotsubo syndrome, etc.

1.4 Chest Pain and Risk Factors for Acute Coronary Syndromes

The coronary risk factors of CP of ACS origin are as follows [6].

Medical History

- A familiar history of myocardial infarction (MI), >60 years, smoking, high arterial blood pressure, dyslipidemia, < LDL cholesterol, diabetes mellitus, periphery vascular disease, prior history of MI, male sex

Risk Factors

- Obesity, hypertension, diabetes, dyslipidemia, visceral fat, insulin resistant, metabolic syndrome, low HDL cholesterol, stress, cocaine abuse

Triggers

- The sudden lowering of body temperature, traffic pollution, intense tobacco abuse, and infections

1.4.1 Pathophysiology

The underlying cause for CP is in relation with its cause, whether for the aortic dissection or the lack of oxygen in myocardial cells. Each of these causes will have a different onset and evolution of the signs and symptoms and different

pathophysiology. To cover all the different theories for the onset of CP for each given cause is far beyond the scope of this chapter, it will instead analyze which characteristics can guide us to a correct and prompt diagnosis. In subsequent chapters, we will address the cardiovascular pathologies seen in the ER.

1.4.2 Clinical Presentation

Since each cause of CP has a different clinical presentation, we will mainly focus on life-threatening cardiovascular causes.

Characteristics of Ischemic CP [6]

- Oppressive or sibling pain (from the chest to the back)
- Localization: precordial, retrosternal, anterior face of the neck, inferior mandible
- With or absent irradiation: left arm, both arms, scapulae, neck, dorsal region
- With or without adrenergic symptoms (nausea, vomit, diaphoresis)
- The sense of imminent death
- Length > 1 min

In Table 1.1, characteristics and related symptoms of CP are shown, along with their positive likelihood ratio and their association with an increased probability of MI [1].

The chest discomfort or pain that occurs in ACS is generally accompanied by an autonomic nervous system stimulation, which in turn makes the patient appear pale, cold, diaphoretic, and clammy to touch [7]. However, we can identify a similar chest pain in non-cardiac disorders such as aortic dissection [7]. Nausea and vomiting are associated with the cardiac cause of the CP. Nausea and vomiting associated with dyspnea are more frequent in women with MI, whereas sweating is more frequent in men. Associated symptoms should always be assessed together with signs of other diseases, such as infection, fever, anxiety, and nervousness [7].

Physicians in charge must be in warning about that the severity of symptoms and the outcome are not related in some cases of ACS. Also, the clinician must have in mind that women suffering from MI have been reported to have pain more frequently in the back, in the neck, and in the jaw [7].

Table 1.1 Chest pain characteristics and related symptoms that are associated with increased odds of MI

Characteristic	+ LR
Pain radiation to both arms	7.1
Right shoulder	2.9
Left arm	2.3
Chest pain as most important symptom	2.0
Diaphoresis	2.0
Nausea or vomiting	1.9

MI myocardial infarction, *+ LR* positive likelihood ratio

Table 1.2 Causes of chest pain and its characteristics

Cause	Characteristic of chest pain
Acute aortic dissection	Pain most often occurs in the chest and most often present as a sharp, severe pain with changing localization, described by patients as tearing, or ripping; auscultation of aortic valve regurgitation
Pulmonary embolism	Pulmonary infarction: worsen with inspiration, anterior or lateral chest wall, associated with transitory or persistent dyspnea. Submassive or massive: retrosternal oppression without irradiation accompanied by persistent dyspnea, tachypnea, desaturation
Pneumothorax	<50%: ipsilateral chest pain bound to respiration, initially sharp and pleuritic, but may become dull or achy over time. >50% retrosternal oppression, in hypertensive modality a circulatory collapse is a clinical presentation. Sudden dyspnea is the main symptom in both conditions
Pericarditis	Classically positional worsening when lying supine and relieved when leaning forward; also, it is possible to identify a friction sound
Musculoskeletal cause	Sharp, well localized, reproduced with movement or palpation
Esophageal rupture	Can cause identical symptoms as cardiac disease but more commonly cause burning pain in the chest and epigastrium

In Table 1.2, other probable causes for CP and the characteristics that can help differentiate the underlying pathology are listed [5, 7].

1.4.3 Physical Examination

For most cases, a physical examination is not helpful distinguishing patients with ACS from those with non-cardiac CP [5]. Although, the approach for a stable and an unstable patient should be different to guide our clinical diagnoses. Physical examination findings associated to MI are shown in Table 1.3 [1].

1.4.4 Electrocardiogram

An ECG is mandatory in all patients with suspected CP from cardiac origin. The findings in the ECG may variate depending on the underlying cause.

1.4.4.1 Acute Coronary Syndromes

ECG remains the best immediately available test for detecting ACS, but its sensitivity for MI is low; a single ECG performed during the initial clinical presentation detects fewer than 50% of AMIs. Patients with normal or nonspecific ECGs have a 1–5% incidence of MI and a 4–23% incidence of unstable angina. The ECG must

Table 1.3 Physical examination findings associated with increased or decreased likelihood of MI

Characteristic	+ LR
Increase probability of MI	
Include a third heart sound on auscultation	3.2
Hypotension with a systolic blood pressure of 80 mmHg or lower	3.1
Pulmonary crackles on auscultation	2.1
Decrease probability of MI	
Pleuritic chest pain	0.2
Pain that is sharp or stabbing	0.3
Pain that is positional	0.3
Pain reproduced by palpation	0.2–0.4

MI acute myocardial infarction, *+ LR* positive likelihood ratio

repeat every 10 minutes when it is not diagnostic and in symptomatic patients with high-clinical suspicion for MI. Prior ECGs are important for determining whether abnormalities shown are new [5].

1.4.4.2 Pulmonary Embolism

ECG has a high sensitivity to pressure overload but low specificity. The most common findings in patients with severe pulmonary hypertension are sinus tachycardia, "S1Q3T3", prominent S wave in lead I, Q wave in lead III, and inverted T wave in lead III (right heart strain). Also aVR ST elevation (right ventricular ischemia), V1 qR and ST elevation (right atrial dilatation and right myocardial infarction), V1 to V4, ST dynamic changes as elevation or depression and V1 to V4 negative T waves, or complete or incomplete right bundle branch, (right ventricular ischemia), and atrial fibrillation as consequence of right ventricular strain [8]. Patients with acute pulmonary embolism (PE) rarely have a normal ECG, but a wide range of abnormalities are possible, and most are equally likely to be seen in other patients [5].

1.4.4.3 Pericarditis and Pericardial Tamponade

Pericarditis, or inflammation of the pericardium, has typical ECG findings. These findings occur in progressive stages, all of which are seen in about 50% of cases of pericarditis.

Stage I (Acute Phase)

- Diffuse concave upward ST elevation in most leads, PR depression in most leads (maybe subtle), and sometimes nothing at the end of the QRS complex

Stage II

- ST elevation and PR depression have resolved, and T waves may be normal or flattened.

Stage III

- T waves are inverted, and the ECG is otherwise normal.

Stage IV

- T waves return to the upright position, and thus the ECG is back to normal [5, 9, 10].

The ECG changes with pericarditis must be distinguished from those of early repolarization. The ST elevation seen in early repolarization is very similar: diffuse and concave upward. However, three things may help to distinguish pericarditis from early repolarization [5, 9, 10]:

- The ratio of the T wave amplitude to the ST elevation should be greater than four if early repolarization is present, meaning the T wave in early repolarization is usually four times the amplitude of the ST elevation. Another way to describe this would be that the ST elevation is less than 25% of the T wave amplitude in early repolarization.
- The ST elevation in early repolarization resolves when the person exercises.
- Early repolarization, unlike pericarditis, is a benign ECG finding that should not be associated with any symptoms.

Also, ECG findings in patients with pericarditis may mimic MI. ST dynamic changes suggest an acute coronary syndrome. ECG findings suggestive of tamponade include low voltage and electrical alternans [5].

1.4.4.4 Acute Aortic Dissection

ECG tracing can range from completely normal, left ventricular hypertrophy or ST elevation if the dissection involves the origin of the right coronary artery [5].

1.5 Imaging Studies

1.5.1 Chest X-ray

It is one of the most taken studies in the ER when CP is present; the findings may vary depending on the underlying cause.

1.5.1.1 Acute Coronary Syndromes

A normal chest X-ray is characteristic. Signs of the pulmonary capillary wedge pressure rises and ACS complicated with heart failure [5].

1.5.1.2 Acute Aortic Dissection

Widened mediastinum or aortic knob occurs in up to 76% of patients; if we add high-clinical suspicion, these three findings give an odds ratio of 11 (95% CI 6.1–19.8) for aortic dissection. Displacement of the aorta and pleural effusion may also have a finding. Around 90% show some abnormality [5].

1.5.1.3 Acute Pulmonary Embolism

The study could be normal in low-risk PE (segmental or subsegmental); however, it is always abnormal in lobar, submassive, and massive PE. Main pulmonary artery dilatation and right ventricular dilatation are infrequent, mainly in those who early arrival after onset symptoms. It is possible to identify classic radiographic findings such as the Westermark sign (a clarified area with diminished vascularity), Hampton sign (a triangle with a base to the pleura and the vertex directed to a branch of the pulmonary artery), elevated diaphragm, and small pleural effusion which are findings related with pulmonary infarction [8].

When the pulmonary obstruction is >25%, acute pulmonary arterial hypertension occurs inducing pulmonary artery and right ventricular remodeling; its radiographic expression is right and/or left pulmonary artery dilatation, main pulmonary artery dilatation, as well as right ventricular dilatation. Also, left or right elevated diaphragms are findings. Most chest X-rays are bedside in submassive or massive PE patients, so it is not easy to identify classic signs. However in this condition chest radiograph allows to exclude another clinical situation mimicking PE (acute pulmonary edema, COPD exacerbation, cardiac tamponade, extensive pneumothorax, etc.) [8].

1.5.2 Echocardiogram

ED clinicians should perform a bedside echocardiogram study in every patient with acute CP and clinical instability, hypotension, severe respiratory failure, aborted cardiac arrest, or acute pulmonary edema if it is available [5]. This non-expensive and accessible tool provides unique insight into the pathophysiology of the CP extending our clinical sensitivity beyond the usual clinical perception. Bedside transthoracic echocardiography can rapidly differentiate conditions inducing clinical instability as PE, myocardial infarction, aortic dissection, and pericardial tamponade, also allowing a rapid lifesaving treatment. Since it is an "operator dependent" tool, is mandatory a clinicians with experience in its use in stable and unstable patients.

1.5.3 Immediate Exercise Stress Echocardiogram

Immediate exercise stress echocardiogram in the ER is a suggestion that has been made by certain studies [11]. Usual common management of a patient who presents with CP to the ER, with a low-risk score, includes a 23-h observation unit admission, with serial biomarkers to rule out MI. Stress echocardiography has several advantages as an imaging modality for low-risk CP patients. Studies report sensitivity 86% and specificity 81% for detecting coronary disease via stress echocardiography, which is superior to an exercise ECG and comparable to myocardial perfusion scintigraphy. Stress echocardiography can also provide findings to diagnose nonischemic causes of CP, including PE, valvular heart disease, pericardial disease, and cardiomyopathy. A final consideration is that there is no radiation. One disadvantage in this technique is the fact that the echocardiography, similar to ECG interpretation, is "operator dependent" [11]. We recommended this approach to patients with risk factors, ischemic chest pain, and normal or non-specific ECG.

1.5.4 Cardiac Computed Tomography (CCT) and Other Imaging Tests

The increase in CCT use is appropriate, given the finding of three major randomized trials that included ER patients with CP. CCT to evaluate patients with this symptom in the ER is performed as a so-called triple rule-out examination; it can be used to exclude other causes of acute CP, such as PE, acute aortic dissection, cardiac tamponade, pericardial effusion, and pneumothorax. Myocardial perfusion imaging and stress echocardiography are not widely accepted for this purpose [12].

Several modalities diagnose acute aortic dissection with high sensitivity, including computed tomography (98%), magnetic resonance imaging (98%), and transesophageal echocardiography (94%) [5].

Computed tomography is the most widely used study for the diagnosis of PE, and it will also provide information about alternative etiologies of CP. On the downside, it exposes patients to radiation and contrast dye, which can limit its use [5].

1.6 Laboratory Evaluation

1.6.1 Cardiac Biomarkers in the Context of Acute Coronary Syndromes

Cardiac Troponins

- Elevate within 3 hours, peak at 12 hours, and remain elevated for 7 to 10 days.
- Preferred test for the diagnosis of MI.

- Highly sensitive troponin assays become detected more rapidly including unstable angina [5].
- In the majority of cases, a single set of negative cardiac biomarkers is insufficient to rule out MI; however, using the high-sensitivity troponin assays, this approach is now possible in select patients [5].

D-dimer

- In patients with a low pretest probability for PE, this test that has high sensitivity can rule out the diagnosis, obviating the need for further testing [5].
- The utility of the D-dimer test depends upon both, patient baseline characteristics and the sensitivity and specificity of the test employed [5].
- Precaution at interpreting this test may be needed in recent major surgery, trauma, pregnancy, and those with malignancy because they are likely to have an elevated D-dimer at baseline [5].

Complete Blood Count

- White blood cell count elevated in any of the inflammatory or infectious etiologies, such as myocarditis, pericarditis, ST-elevation MI, PE, mediastinitis, and pneumonia [5].
- Anemia in exertional CP is suggestive of myocardial ischemia but also consistent with aortic rupture [5].

B-Type Natriuretic Peptide and N-Terminal Pro-BNP

- B-type natriuretic peptide levels >100 pg/mL are highly sensitive for acute heart failure. Levels <50 pg/mL have high negative predictive value for heart failure [5].
- N-terminal pro-BNP levels >500 pg/mL are highly sensitive for acute heart failure. Levels <500 pg/mL also have a high negative predictive value for heart failure [5].

1.6.2 Differential Diagnosis

In all patients with acute onset of CP, ACS must be ruled out; however, other more frequent clinical conditions should be considered and excluded. In Table 1.4, we can find the final diagnosis found in a multicenter registry [1] with suspected ACS that includes 15,608 patients (being CP the main complaint in the 71% of ACS visits).

1.6.3 Clinical Approach

When confronted with a patient suffering from acute CP, the first important task is to decide whether the patient has a life-threatening disease or not, so judgment is based on the patient's previous history, actual symptoms, and clinical signs on admission [7]. We will consider an unstable patient when it presents these characteristics:

Table 1.4 Final diagnosis of the Internet Tracking Registry of Acute Coronary Syndrome

Final diagnosis	Percentage
Chest pain not otherwise specified + another diagnostic	70%
Unstable angina	6.3%
Congestive heart failure	4.0%
STEMI	1.6%
Pneumonia	1.5%
Stable angina	1.2%
NSTEMI	1.0%
Pulmonary embolism	0.4%
Pericarditis	0.3%
Dissecting aneurysm	0.1%

STEMI ST-elevation myocardial infarction, *NSTEMI* non-ST-elevation myocardial infarction

- Blood pressure < 90 mmHg
- Severe respiratory distress
- Oxygen saturation < 90%
- Tachycardia > 100 bpm

When approaching this unstable patient, immediate actions are required, to stabilize airway, breathing, and circulation; start assessing the probable cause according to the presentation, ECG, and characteristics of CP; and treat accordingly.

For a stable patient, the use of a fast stratification is necessary for their management, mainly to identify those with immediate risk of complications, as those with ACS. The HEART score in low-risk patients allows to rule out a cardiac cause without further planned cardiac testing. In several studies, this score has been accurate in predicting a low risk of 60-day MACE (>99% NPV) [4]. Further evidence suggests that the use of HEART score obtains a higher diagnostic value than troponin or clinical evaluation solely [13]. Tables 1.5 and 1.6 describe the variables of the HEART score and how to interpret each value, respectively.

The currently most used risk scores are the TIMI score and the GRACE score; each can give an idea of the 30-day mortality for the patient varying its prognostic value whether if there is an ST-elevation myocardial infarction or a non-ST-elevation myocardial infarction.

Where the clinician should always focus their attention first on are the patient's history, comorbidities, and description of symptoms, to help narrow the scope of potential diagnosis and to stratify patient's risk for life-threatening disease. Physical examination focuses on vital sign abnormalities and cardiac or pulmonary findings [5].

Any patient without a clear explanation for their CP even after the initial workup including chest X-ray and ECG will be considered to have an ACS until proven; otherwise, in these patients, serial ECGs and risk assessment (HEART, TIMI scores) are cornerstones for management [5].

Table 1.5 HEART score for chest pain patients at the ER

Variable		Points
History (anamnesis)	Highly suspicious	2
	Moderately suspicious	1
	Slightly or non-suspicious	0
ECG	Significant ST-depression	2
	Non-specific repolarization disturbance	1
	Normal	0
Age	≥65 years	2
	45–65 years	1
	≤45 years	0
Risk factors	≥3 risk factors, or history of atherosclerotic disease	2
	One or two risk factors	1
	No risk factors are known	0
Troponin	≥3x normal limit	2
	1–3x normal limit	1
	≤normal limit	0

Score: low risk, <4; intermediate risk, 4–6; high risk, >7

Table 1.6 How to interpret the HEART score

HEART score	MACE	Death	Decision
0–3	1.9%	0.05%	Discharge
4–6	13%	1.3%	Observation with noninvasive stress testing or imaging, risk management
7–10	50%	2.8%	Early invasive diagnostics and treatment

MACE major adverse cardiac event

Another pathology outcome time depending on that we must rule-out acute aortic dissection. In a prospective observational study, its probability significantly increases with the presence of the following variables [14]:

- Abrupt onset of thoracic or abdominal pain with a sharp, tearing, and/or ripping character
- Variation in pulse (absence of a proximal extremity or carotid pulse) and/or blood pressure (>20 mmHg difference between the right and left arm)
- Mediastinal and/or aortic widening in the chest X-ray

Acute aortic dissection occurs in approximately 83% of patients with variables 1 and 3 and approximately 92% of patients with variables 1 and 2. When all three variables coexist, diagnosis of acute aortic dissection is present in all patients; when no variable is present, approximately 7% of patients were found with the diagnosis [14].

Clinicians frequently overlook acute PE in the ER, and it always should be considered in the acute chest discomfort or dyspnea who lacks a firm alternative diagnosis. The approach is based on risk stratification, with symptoms suggestive of PE and right ventricular heart dysfunction or hemodynamic instability are at high risk. Several scoring systems exist to characterize patient risk for PE, including the Wells score, the Charlotte criteria, the revised Geneva score, and the PERC rule [5].

For diagnosing or rule-out cardiac tamponade, a bedside echocardiogram is an ideal tool, especially in any patient with suggestive historical, examination, or electrocardiogram findings [5].

Tension pneumothorax is diagnosed clinically combining: a suggestive history, hemodynamic compromise, and unilateral diminished breath sounds. This triad is the usual presentation. Treatment should not be delayed while awaiting confirmation from chest X-ray. This tool or bedside echocardiogram may be used to make the diagnosis in patients without signs of tension. The treatment is immediate needle thoracostomy, followed by tube thoracostomy [5].

The initial chest X-ray is almost always abnormal in patients with esophageal perforation and mediastinitis and usually reveals mediastinal or free peritoneal air as the initial radiologic manifestation. CT scan may show extraesophageal air, periesophageal fluid, mediastinal widening, and air and fluid in the pleural spaces, retroperitoneum, or lesser sac. The diagnosis is confirmed with the oral administration of a water-soluble contrast agent followed by chest X-ray looking for extravasation [5].

To find more about each specific treatment of pathologies causing CP, go to the corresponding chapter of your suspected diagnosis in this book.

To find a more visual way to the approach to CP patients, look for the algorithm in Fig. 1.1.

1.7 Additional Clinical Practice Takeaway

- CP is one of the most common complaints in the ER; its wide variety of causes forces a well-structured workup to find its diagnosis.
- The first step is to detect stable and unstable patients; in some cases, the underlying cause is obvious, for example, in trauma-related CP.
- It is necessary to determine whether CP is from a cardiac, pulmonary, musculoskeletal, or another source and do it with proper speed.
- The clinician should have a structured approach when encountered with CP and know if there is a code response team available at the hospital.
- Precaution at interpreting D-dimer in patients with recent major surgery, trauma, pregnancy, and those with malignancy.
- We recommended stress test in ER in those with risk factors, ischemic chest pain, and normal or non-specific ECG.
- An echocardiogram provides unique insight into the pathophysiology of the CP extending our clinical sensitivity beyond the usual clinical perception.

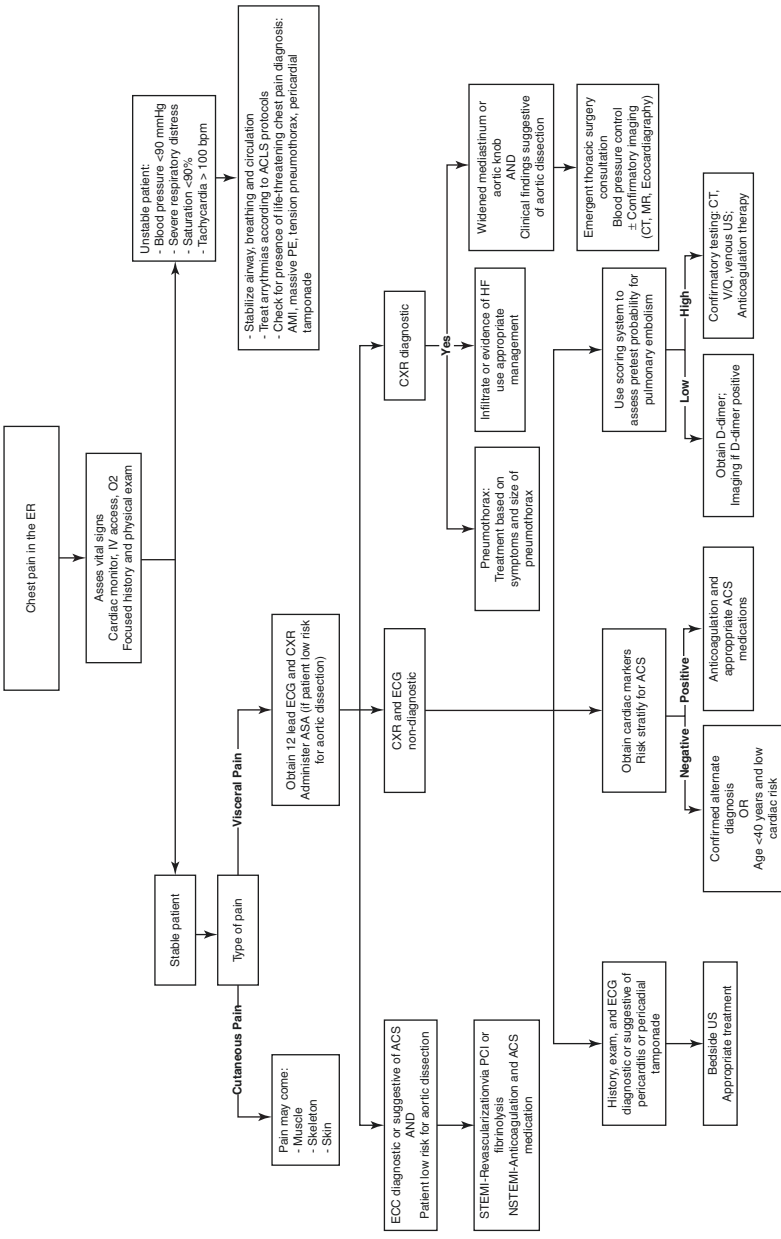

Fig. 1.1 Approach to the patient with chest pain in the emergency room

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