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Chest Pain in Young Adults

Chest pain is one of the most common reasons that patients present to the emergency department (ED). The underlying disease processes can range from benign to life-threatening. It is critical for the provider to thoroughly evaluate and risk-stratify patients who present with this complaint to reduce the chances of missing a potentially serious condition.

The purpose of this article is to discuss the diagnosis, treatment, and management of the common causes of chest pain that can present in a young adult.

Introduction

About 8 million people are seen in the United States every year for chest pain.[1] In contrast to the population of elderly adults typically seen for chest pain in EDs, an increasing number of young men and women are evaluated for symptoms of acute chest pain every day.[2] Many of these patients have no cardiac risk factors. The prevalence of acute coronary syndrome has been found to be around 1% in people younger than 40 years of age, and the prevalence is about 5-10% in those younger than 50 years of age.[3] Therefore, ED providers must be aware of the various other causes of chest pain in the young population.

Chest pain can be caused by a variety of disease processes that can range from benign to life-threatening. Although protocols have been developed to consider every patient who presents with chest pain as a potentially serious condition based on acuteness, duration, and intensity of pain, these factors do not necessarily correlate with a life-threatening disorder. Providers often dismiss chest pain in the younger population because of the rare occurrence of significant morbidity and mortality. Patients who are evaluated for chest pain in the ED must undergo a thorough history and physical examination with proper diagnostic testing to avoid inappropriate patient evaluation, treatment, and disposition.

Evaluation of Chest Pain

In patients presenting to the ED with chest pain, the provider must perform a thorough history and physical examination to help guide the plan of care and treatment. It is imperative that the provider ask the proper questions and pay attention to key information obtained in the history, as this can assist with developing a broad differential diagnosis.[4] In addition, being aware of the characteristics of the pain also can be beneficial. The differential for chest pain involves noncardiac causes as well, so asking questions about the symptoms, pain quality, and other characteristics can differentiate between cardiac and noncardiac causes.[2] After gaining the necessary information, the provider can identify risk factors that will place the patient in a high-risk category.

On diagnostic evaluation, an electrocardiogram (ECG) is recommended

EXECUTIVE SUMMARY

- The most common causes of acute chest pain in young adults presenting to the emergency department are chest wall pain (costochondritis) and anxiety (panic disorder).
- A 12-lead ECG and chest radiograph are usually the only ancillary tests required to evaluate chest pain in otherwise healthy young adults.
- Risk-stratification tools can be used to reasonably exclude potentially serious causes of chest pain, such as pulmonary embolism or aortic dissection.
- Consider a therapeutic trial of of histamine -2 (H2) blockers in young adults discharged with undiagnosed chest pain after an appropriate evaluation.

within 10 minutes of arrival.[5] If the provider is concerned about potential acute coronary syndrome, the laboratory evaluation should include cardiac troponins. However, elevations in troponin T can be seen in other diseases, such as renal dysfunction, pulmonary embolism, heart failure, myocarditis, pericarditis, and septic shock.[6] A chest X-ray should be included for patients with chest pain to assist with the diagnosis. Chest radiography also is beneficial in life-threatening causes of chest pain, such as a pneumothorax, for which immediate intervention may be required. Other laboratory testing or specific imaging, such as CT or ultrasound, should be considered based on the suspected diagnosis. Disposition depends on either determining a specific cause or excluding potentially serious conditions to a reasonable degree. Thus, a majority of young adults with chest pain are sent home after an ED assessment. Some EDs have created observation units, where patients with fewer comorbidities and risk factors may be monitored and have additional testing performed before discharge.

Gastroesophageal Reflux Disease

Gastroesophageal reflux disease (GERD) is a frequent cause of chest pain in young people. It is caused by transient relaxation of the lower esophageal sphincter (LES) complex.[7] It can be seen in a wide variety of age groups, including neonates and the elderly. If GERD develops in childhood, the risk of developing it in adolescence and adulthood increases.[8]

When the LES relaxes, the acidic gastric contents enter the lower esophagus, causing irritation of the mucosa.[7] In turn, this causes a burning sensation to the substernal chest. Patients also may complain of dysphagia, odynophagia, nausea, vomiting, unrelenting cough, and increased discomfort with meals.[7]

Although usually harmless, the sensation often may be mistaken by patients for ischemia. This is due to the variability in the nature of the chest pain itself. Specifically, GERD can present with squeezing or pressure-like chest pain, radiation of pain to the arms and back, and diaphoresis.[7] Patients also may describe worsening pain while lying flat.

Because of the similar presentations of GERD and acute coronary syndrome, patients must be risk-stratified for a safe disposition. This includes a careful and thorough history and physical exam. If there is suspicion for acute coronary syndrome based on the patient's history, symptoms, and risk factors, a more aggressive approach may be taken, including lab testing, ECG, and radiologic studies.

Patients with suspected GERD may be treated in the ED with a variety of interventions. The goal of these treatments is to decrease acid production and risk factors.[7] The two most commonly prescribed medications for acute GERD include histamine-2 (H2) blockers and proton pump inhibitors (PPIs), which decrease acid production.[7]

Baclofen is a GABA-B agonist that also can be used. This has been shown to work by inhibiting the relaxation of the lower esophageal sphincter.[9] However, this medication has multiple side effects, including sleepiness, fatigue, and lightheadedness. A recent trial showed that baclofen may be beneficial for patients on PPI therapy who have frequent episodes of nighttime heartburn.[10] Prokinetic drugs, like mosapride, in combination with a PPI have shown no clear role in the treatment of GERD.[11]

In addition, it is important to counsel patients on lifestyle modifications, including weight loss and decreasing consumption of caffeine, alcohol, fatty foods, and chocolate.[7,11] It also may prove beneficial for patients to sleep with the head of the bed elevated at 30 degrees and to avoid eating within three hours of bedtime.[7,11] In patients who are refractory to drug therapy, surgical options such as laparoscopic antireflux surgery have been shown to be beneficial.[11] In patients with normal vital signs and who are well appearing, outpatient referral to a gastroenterologist for ambulatory pH monitoring or non-emergent endoscopy is appropriate. If left untreated, complications from GERD may develop. These include esophageal strictures, inflammatory esophagitis, and Barrett's esophagus.

Acute Pericarditis

Acute pericarditis is caused by inflammation of the pericardial sac. In one study it was found to occur in 4.4% of patients presenting to the ED with chest pain, and frequently is seen in young adults.[12] One study of 1,361 patients admitted to the hospital for chest pain showed that pericarditis was a significant cause for admission in young adults and was seen more frequently in males of younger age groups than in females.[12]

The inflammation from pericarditis results from an increase in lymphocytes and granulocytes around the pericardium, usually caused by infection, malignancy, drugs, or systemic rheumatic diseases.[13,14] (*See Table 1.*) Patients typically present with sharp, pleuritic chest pain, which is worse when the patient is lying flat.[13] Oftentimes, patients will lean forward to alleviate their symptoms. They may be febrile

Table 1. Common Causes of Acute Pericarditis
Infections: viral (coxsackie virus, echovirus), bacterial (staphylococcus and streptococcus)
Malignancy (i.e., breast or lung cancer, leukemia, lymphoma)
Drugs: procainamide, hydralazine
Systemic rheumatic diseases: systemic lupus erythematosus, rheumatoid arthritis, scleroderma
Miscellaneous: post-myocardial infarction, uremia, radiation, myxedema

and complain of myalgias, and a friction rub may be heard on physical exam.[13] Although the affected area may be generalized in the anterior chest wall, there may be referral of pain to the left trapezial ridge.[15]

Unfortunately, ancillary tests available in the ED lack accuracy for detecting acute pericarditis. For patients in whom the disease is suspected, an ECG should be ordered to look for specific patterns that correlate with the diagnosis. A single ECG may not be that helpful, but serial ECGs performed over several days may show serial changes in a characteristic pattern. The first changes will be seen in the initial hours to days after onset of the disease.[13] This includes ST segment elevation in leads I, II, and III, aVL, aVF, and V_2 to V_6 with reciprocal depressions in aVR and V_1.[13] PR segment depression also frequently is seen in leads II, aVF, and V_4-V_6.[13] After the initial stage, the ST and PR segments normalize, T waves flatten, and there is development of deep inverted symmetrical T waves.[13] Lastly, the ECG normalizes, but T wave inversions may become permanent.[13]

Although not required, a chest X-ray can be performed to rule out other causes if the diagnosis is unclear initially. Bedside echocardiogram may be used to rule out the presence of a pericardial effusion.[14] If present, admission may be warranted to investigate the underlying cause, and treatment may be necessary if the patient is hemodynamically unstable.

Laboratory studies generally are nonspecific and may be ordered to determine non-idiopathic causes of pericarditis.[14] If there is suspicion for associated myopericarditis, cardiac biomarkers may be ordered to assess for elevation.[14]

Treatment should be directed at the underlying cause. Nonsteroidal anti-inflammatory drugs (NSAIDs) are the mainstay of therapy in patients with idiopathic or viral pericarditis for up to one to three weeks.[14] Ibuprofen is recommended in the acute phase, and colchicine has been found to prevent recurrence.[15] In patients with resistance to colchicine, anakinra has been shown to reduce the risk of recurrence over a median of 14 months.[16]

Glucocorticoids have not been shown to be beneficial in patients with acute idiopathic pericarditis, as there is a high risk for recurrence when the steroids are stopped or tapered. These should be used only in patients with idiopathic pericarditis who are refractory to treatment or are unable to tolerate NSAIDs plus colchicine.[17]

Patients with acute pericarditis may be discharged home safely unless there is a high suspicion for myocarditis or the presence of a large effusion, fever, or immunosuppression.[14]

Myocarditis

Myocarditis is a condition that occurs as a result of inflammation of the myocardium.[18] The pathophysiology involves invasion of the myocardium by various cells, including lymphocytes, plasma cells, and histiocytes.[18] It is caused most commonly by an infectious process.[18] (*See Table 2.*) Although Chagas disease is the most common cause worldwide, viruses such as parvovirus B19, human herpes virus 6, and coxsackie B are responsible for the majority of cases in the United States.[19] Other common causes include toxins, drugs, and immunologic conditions. Sometimes myocarditis is associated with pericarditis; myocarditis is seen in an estimated 12% of patients with pericarditis.[20]

Patients diagnosed with myocarditis can present with a wide range of symptoms, varying from nonspecific chest pain to cardiogenic shock. Myocarditis and acute coronary syndrome share similar characteristics, and both must be considered in a young patient who presents with chest pain.[21] Patients generally complain of retrosternal chest pain, myalgias, and fever with disproportional sinus tachycardia. As with pericarditis, a friction rub may be auscultated on exam. The patient's appearance may help differentiate between myocarditis and pericarditis, as patients with myocarditis generally appear ill, as opposed to those with pericarditis, who often do not look ill.

Diagnosis in the ED usually is clinical and is supported by clues on traditional cardiac workup. These include elevations in cardiac biomarkers, ECG abnormalities, and cardiomegaly on chest X-ray. The ECG may show nonspecific ST wave changes, and PR depression as is seen in pericarditis.[18]

Definitive diagnosis typically was done by cardiac catheterization and endomyocardial biopsy. The approach has fallen out of favor because of the risk of that invasive procedure and the advent of newer diagnostic strategies. These strategies include nuclear imaging with gallium-67-labeled or indium-111-labeled antimyosin antibodies and cardiac MRI.[18,21] These methods may assist in differentiating between acute coronary syndrome and myocarditis if the diagnosis is uncertain. These studies generally are time intensive and are performed on admission after the diagnosis is suspected.

The mainstay of treatment in idiopathic or viral myocarditis consists of conservative management, including hemodynamic support. In severe cases, immunosuppressive therapy with prednisone or azathioprine may be beneficial but generally is not started in the ED.[18] If diagnosed early, antiviral therapy with ribavirin or pleconaril may be initiated in cases of viral myocarditis.[19] If patients become unstable and are unresponsive to traditional support therapies such as vasopressors and inotropes, more aggressive care may be warranted. This includes left ventricular assist device (LVAD), extracorporeal membrane

Table 2. Some Common Causes of Myocarditis

Chagas disease (*Trypanosoma cruzi*)
Viruses
- Parvovirus B19
- Human herpes virus 6
- Coxsackie B

Bacteria
- *Corynebacterium diphtheriae*
- *Neisseria meningitidis*
- Rheumatic fever
- Lyme disease (*Borrelia burgdorferi*)

Toxins: ethanol, arsenic, iron, copper
Drugs: anthracycline chemotherapeutic agents (doxorubicin), antipsychotics (clozapine)
Immunologic conditions: scleroderma, systemic lupus, sarcoidosis

oxygenation (ECMO), or lastly cardiac transplantation.[19]

Mitral Valve Prolapse

Mitral valve prolapse (MVP) is a frequently unrecognized cause of chest pain, primarily in young females. Because of its subtle presentation and physical exam findings, it often is forgotten in the differential diagnosis of chest pain in the ED. It is the most common valvular disorder in industrialized countries, affecting approximately 2.4% of the population.[22]

Generally speaking, MVP is recognized as a benign condition. In rare instances, complications may develop and treatment may be necessary. If left undiagnosed, dysrhythmias, congestive heart failure, endocarditis, stroke, and sudden cardiac death may occur.[23]

MVP occurs when there is bulging of the valve leaflets into the left atrium during left ventricular contraction. The underlying cause involves abnormalities in connective tissue proteins, and it usually is inherited.[22]

Patients with MVP usually are asymptomatic, but may complain of atypical chest pain, palpitations, generalized fatigue, anxiety, and shortness of breath.[22] Increases in morbidity have been associated with symptoms during exercise.[24] On physical exam, the provider should pay close attention to auscultation of heart sounds. Classically, a mid-systolic click is heard at the apex.[22]

Diagnostic studies and treatment are not indicated in ED patients suspected to have MVP, but the provider should assess the patient carefully for its complications, depending on symptomatology.[22] Patients may be referred to outpatient cardiology for an echocardiogram to confirm the diagnosis and to assess other mitral valve dysfunction and complications. Patients sometimes are prescribed beta-blocker therapy if they are symptomatic with palpitations, but this is not performed routinely by the ED provider.[22]

Hypertrophic Cardiomyopathy

Hypertrophic cardiomyopathy is an inherited autosomal dominant disease that is characterized by ventricular hypertrophy, mostly affecting the interventricular septum. It has been shown to have a prevalence in the general population of approximately one in 500 people.[25] It can occur in any race or gender and is seen in any age, but it is diagnosed most commonly among people 30-40 years of age.[26] It is caused by mutations in sarcomere genes, which encode sections of the contractile proteins in the heart muscle.[26] The most common mutation occurs on the beta-myosin heavy chain.[25] Over time, this translates into development of asymmetrical left ventricular hypertrophy. The reduction in compliance of the hypertrophied left ventricle leads to poor diastolic function and restricted ventricular filling.[25]

Dyspnea on exertion is the most frequent initial complaint seen in the ED.[22] Chest pain, palpitations, syncope, and sudden cardiac death are other associated symptoms. In athletes, hypertrophic cardiomyopathy is the most common cause of sudden cardiac death.[27] It is important to ask the patient about family history of sudden cardiac death, as this can be an indicator of familial inheritance of the disease. The chest pain experienced is due to a mismatch between the oxygen demand of the hypertrophied muscle and the available rate of perfusion in the myocardium.[25]

On physical exam, a systolic ejection murmur may be auscultated, which is best heard at the apex of the heart. This murmur may be enhanced if the patient is asked to stand from the seated position or if a Valsalva maneuver is performed. In turn, this causes alterations in preload and afterload that accentuate the murmur.

Patients who present with chest pain and suspected hypertrophic cardiomyopathy should have an ECG and chest X-ray performed in the ED. Although not completely diagnostic, changes on either test may lead the provider to further suspect the disease. About 75-95% of ECGs in these patients will be abnormal and can show signs of left ventricular hypertrophy and left atrial enlargement.[26,27] Specifically, anterior, lateral, and inferior Q waves, known as "septal Q waves," with associated upright T waves may be present.[25] The chest X-ray may appear normal, but can show enlargement of the left ventricle or the left atrium.

If the diagnosis is suspected, disposition should be made in conjunction with a cardiologist, and arrangements made for an echocardiogram. The echocardiogram can be performed on an outpatient basis if the patient is stable and has reliable follow-up, unless the patient experienced a syncopal event prior to ED arrival. This can be an indicator of deadly dysrhythmia, for which patients need to be admitted to a monitored bed. This subset of patients requires a more extensive workup in addition to an echocardiogram, including a possible stress test, tilt testing, and Holter monitor placement.

Once performed, the echocardiogram can show disproportionate septal

hypertrophy, reduced septal motion, or left ventricular outflow tract narrowing.[26] In the instance in which the echocardiogram is inconclusive, cardiac MRI is recommended.[25,26,27] Genetic screening may be helpful to identify other family members who may carry the disease.

Beta-blockers are the first-line therapy in patients with hypertrophic cardiomyopathy who have episodic chest pain. Medicated patients have improved left ventricular filling, due to prolonged diastolic filling time. In addition, the negative inotropic effect of beta-antagonism reduces outflow tract obstruction.[26,29] Second-line therapy includes calcium channel blockers if the patient has no reduction in symptoms with beta-blockers.[29]

If the patient is unresponsive to medical management, septal myomectomy may be a last resort in patients with severe symptomatic disease.[27] Newer therapies also include percutaneous septal ablation, in which ethanol is injected into one of the septal branches, which causes a chemical necrosis of the tissue and thinning of the hypertrophied ventricle over time.[27] Nitroglycerin should be avoided in these patients, as it decreases preload, which the patient requires to maintain adequate cardiac output.[26,27]

Patients with increased risk for sudden cardiac death need to be evaluated for implantable cardioverter defibrillator placement. Risk factors for sudden death include a family history of sudden cardiac death in someone younger than 45 years of age, evidence of non-sustained ventricular tachycardia on Holter monitor, more than two incidences of syncope, abnormal blood pressure response during exercise, or LVH with wall thickness greater than 30 mm seen on echocardiogram. If the patient has two or more risk factors, implantable cardioverter defibrillator implantation is recommended.[27]

If the patient is discharged from the ED, close outpatient follow-up is critical. The provider must have the patient refrain from any strenuous activity until he or she is evaluated by a cardiologist.

Costochondritis

Costochondritis is defined as inflammation of costochondral junctions of ribs or chondrosternal joints of the anterior chest wall.[30] Although it is harmless, the condition's symptomatology can cause concern for patients who fear their pain is cardiac in nature. It is a common cause of chest pain seen in the ED and was responsible for 30% of ED visits in one prospective study.[31] It sometimes can be mistaken for Tietze syndrome, in which there is swelling of a single costal cartilage, usually of the second rib.[30] Costochondritis, on the other hand, usually involves multiple rib spaces and lacks the swelling seen in Tietze syndrome.

Patients with costochondritis can complain of chest pain of varying nature, although typically the pain is described as pressure-like, sharp, or achy. Pain can be worsened by movement, breathing, and exertion. Inciting factors include prior respiratory illness with forceful cough, recent strenuous exercise, and pulling, pushing, or reaching with the upper extremity. Ribs 3 and 4 are affected most often, and the pain is more frequently unilateral.[30] On physical exam, patients will have tenderness to palpation of the affected sites.

A diagnosis of costochondritis should be made only after other serious, life-threatening causes of chest pain have been ruled out, as cardiac-related chest pain also can be reproducible. A study published in 1994 showed that 6% of patients presenting to the ED with reproducible chest pain ultimately were diagnosed with myocardial infarction (MI).[31]

In young adults with symptoms and signs consistent with costochondritis, the only ancillary test needed is an ECG. In patients older than 35 years of age, those with risk factors for acute coronary syndrome, or those who have other cardiopulmonary symptoms, an ECG and chest X-ray should be performed.[30] Further imaging with CT should be reserved for patients in whom there is suspicion for costal mass or neoplastic process.[30]

The mainstay of treatment for costochondritis is pain relief with non-opiate analgesics. Acetaminophen and ibuprofen are appropriate treatment modalities, and the patient should be instructed to refrain from strenuous activity that reproduces symptoms. Heating pads applied to the area also may be helpful. Severe cases may be treated with intercostal lidocaine/corticosteroid combinations or other local anesthetics.[30]

Patients should be informed that the pain can resolve within weeks to months, but may last up to one year.[30,31] Nonetheless, costochondritis is a self-limiting condition in which symptomatic improvement is the mainstay of treatment.

Pneumothorax

A pneumothorax occurs when free air enters the intrapleural space. It is a potential life-threatening cause of chest pain encountered in the ED. It can be due to trauma or can occur spontaneously with or without underlying lung disease. A pneumothorax that occurs in the absence of pulmonary pathology is termed a primary pneumothorax. This usually occur in males, smokers, those with a history of Marfan syndrome or mitral valve prolapse, and changes in ambient pressure.[32] In addition, familial patterns indicate an inherited association.[32,33]

Spontaneous pneumothorax occurs in approximately 15 patients per 100,000 population per year in males and five patients per 100,000 population per year in females.[34,35] It develops when there is rupture of a subpleural bleb, forming a passage for free air into the pleural space.[32] This usually involves the apex of the lung.[32,34]

Patients will present with sudden-onset unilateral pleuritic chest pain and dyspnea. Sinus tachycardia is the most common physical finding. Many primary pneumothoraces often are small in size and may not be evident on physical exam. While breath sounds classically are absent or significantly diminished with a large pneumothorax, in cases of small primary pneumothoraces, bilateral breath sounds can be present. Tracheal deviation generally is not seen, as the occurrence of a spontaneous tension pneumothorax is rare.[36]

Diagnosis is made with an upright posteroanterior chest X-ray. It is recommended to obtain a lateral view as well, as one study showed this view led to the diagnosis of an additional 14% of cases that were not identified initially.[32,37]

Table 3. Treatment of Small Primary Pneumothorax

Small pneumothorax (< 20%) in otherwise healthy young adult:
- Observe and administer oxygen therapy.
- Observe in ED or observation unit for 4-6 hours for small, minimally symptomatic pneumothorax.
- Obtain repeat chest X-ray prior to discharge to ensure there is no increase in size.
- Advise patient to avoid air travel and underwater diving until complete resolution.

Findings on X-ray include loss of lung markings in the periphery and a pleural line that runs parallel to the chest wall.[32]

The management and disposition is dependent on the size of the pneumothorax, as well as the patient's clinical picture. Measurement of the pneumothorax can be done using a variety of different techniques; for ED management purposes, the important distinction is small vs. not small. A small primary pneumothorax can be determined if the distance from the lung apex to the cupula is 3 cm or less on an upright chest radiograph.[35] If a chest X-ray is inconclusive but suspicion remains high, a chest CT without contrast can be obtained.

For a small pneumothorax (classified as less than 20%) occurring in an otherwise healthy young adult, observation with oxygen therapy is recommended.[32,35,38] The addition of 100% oxygen has been shown to increase the absorption rate four-fold per day compared to observation alone.[32,35] This treatment works by decreasing the partial pressure of nitrogen, which in turn increases the rate that air diffuses across the pleural-alveolar barrier.[35]

Patients may be observed in the ED or a traditional observation unit for four to six hours in the case of a small, minimally symptomatic pneumothorax. A repeat chest X-ray should be obtained prior to discharge to ensure that there is no increase in size.

Patients should be advised to avoid air travel and underwater diving until complete resolution of the deficit. Other options include aspiration of air with a small catheter or small chest tube placement.[32] (*See Table 3.*)

Anxiety Reaction

ED providers are seeing an increasing number of patients with complaints of chest pain who have no risk factors for coronary artery disease and a non-diagnostic or normal evaluation. Many of these patients are found to have similar features, including life stressors, restlessness, fatigue, insomnia, and poor eating habits.[39] This symptomatology, known as a stress reaction or panic disorder, can be responsible for causing chest pain and should be included in the differential diagnosis.[39,40]

Panic disorder affects more than 6 million Americans and usually develops in young adulthood.[39] A significant percentage of young adults evaluated in the ED for chest pain are found to have panic disorder.[39-42] When symptoms noted above are present, the provider should attempt to avoid unnecessary and costly workups.[39]

Patients who typically experience a stress reaction or panic disorder may make multiple visits to the ED for fear they are experiencing a serious cardiac event. This is because the underlying anxiety can cause symptoms that resemble those found in acute coronary syndrome, including tingling, palpitations, and disordered breathing in addition to chest pain.[39] It is crucial to obtain a thorough history in these patients, as this is what likely will lead the provider to the diagnosis of anxiety. In a young patient without significant medical history, no workup is required. If the patient has risk factors for acute coronary syndrome, further testing with an ECG, chest X-ray, and laboratory studies is necessary.

Treatment varies, but the majority of studies support the use of conservative management with outpatient cognitive therapy. Because the spectrum of psychological therapy can be time-consuming and progressive, its use is not practical for imitation in the ED. With multiple sessions, cognitive therapy discussions may help the patient understand the triggers behind the attacks and how to prevent them. It is important for the ED physician to validate the patient's concern and reassure the patient that the symptoms are treatable.

If calming the patient in the ED fails, medical therapy may be required. Small doses of oral benzodiazepines, such as lorazepam, diazepam, or alprazolam, can be used to alleviate anxiety.[39] If the patient receives medication in the ED and the chest pain remains unchanged, further workup is recommended. On discharge, patients should be referred to professional mental health personnel for long-term care and treatment.

Cocaine-associated Chest Pain

Cocaine abuse is a common problem in the United States, with more than 14% of the population 12 years of age and older having used it once in their lifetime.[6] Acute MI occurs in approximately 6% of patients presenting to the ED with chest pain after using cocaine.[6,43] One study found most patients with cocaine-induced MI were young, nonwhite tobacco smokers who had used cocaine within the past 24 hours prior to arrival.[6,44] Chest pain is the most common complaint in patients presenting to the ED after cocaine use. Cocaine users are at increased risk of myocardial ischemia because of its effects on coronary vasoconstriction, platelet aggregation, and myocardial oxygen demand.[6,45]

The nature of the chest pain may vary, and no specific historical factor is more highly associated with cocaine-induced MI than an alternative diagnosis. Vital signs may be normal, but an acute rise in blood pressure commonly is seen.[6] Chest pain may be accompanied by tachycardia, diaphoresis, and tremulousness.

All patients who are evaluated for cocaine-related chest pain should have a full cardiac workup because of the increased risk of myocardial ischemia and aortic dissection.[6] This includes

ECG, chest radiography, and cardiac biomarkers.

Treatment of chest pain related to cocaine use is similar to treatment for chest pain due to atherosclerosis, with the addition of benzodiazepines such as lorazepam. The patient should be placed on cardiopulmonary monitoring, and aspirin should be given. The combination of a benzodiazepine with sublingual nitroglycerin is effective by counteracting the sympathomimetic effects of cocaine, and also may be beneficial for those with significant elevations in blood pressure.[46] Honderick et al found that the combination of the two drugs was more effective in relieving chest pain caused by cocaine use than either drug alone.[47] If the patient is unresponsive to this therapy, calcium channel blockers are a second option.[46]

It has been stated that providers should refrain from administering beta-blockers in cocaine-related chest pain, but this conventional wisdom is controversial. Theoretically, beta-blockade can worsen myocardial tissue damage by allowing unopposed alpha-receptor stimulation. A randomized study published in 2010 in which beta-blockers and calcium channel blockers were given to patients with cocaine-related chest pain found that there were no significant adverse outcomes.[46,48]

In patients with cocaine-related chest pain who have evidence of ischemia on ECG and who do not show improvement on repeat ECG after medical therapy, percutaneous coronary intervention is warranted and is considered superior to fibrinolytic therapy.[46] Fibrinolytic therapy may be necessary if timely transport for percutaneous coronary intervention is not an option.

Several novel approaches have emerged in the literature for additional approaches when treating cocaine-related chest pain. Phentolamine administration has been shown to decrease pain and coronary vasoconstriction, but further studies are needed to validate its effectiveness.[46] Dexmedetomidine is a sympatholytic drug that may be effective in decreasing cocaine-associated hypertension because of its ability to counteract cocaine's effect on central sympathetic upregulation.[46] In this specific study, a low dose of 0.4 μg/kg was used, which proved to be non-sedating.[46,49] Lastly, apocynin has the ability to decrease cardiac oxidative stress, although its NADPH oxidase inhibition and may be useful in decreasing poor outcomes.[46]

Table 4. Wells Score

Variable	Score
Clinically suspected deep vein thrombosis	3
Alternate diagnosis is less likely than pulmonary embolism	3
Tachycardia (heart rate > 100 bpm)	1.5
Immobilization (≥ 3 days) or surgery in previous four weeks	1.5
History of deep vein thrombosis or pulmonary embolism	1.5
Hemoptysis	1
Malignancy (with treatment within 6 months) or palliative	1

Traditional interpretation:
Score > 6.0 – High (probability 59%)
Score 2.0-6.0 – Moderate (probability 29%)
Score < 2 – Low (probability <4%)

Adapted from: Reda LV, Choi A. Pulmonary embolism. *Emerg Med Rep* 2015;36:54.

Pulmonary Embolism

Pulmonary embolism (PE) occurs when a thrombus occludes a specific site in the pulmonary vasculature. Most pulmonary emboli stem from a deep venous thrombosis of the extremities or inferior vena cava.[50] About 200,000 new cases of PE are diagnosed in the United States each year.[50] PE is the second leading cause of non-traumatic death in outpatients.[50] In otherwise healthy patients younger than 50 years of age who are hemodynamically stable, the fatality rate is 1%.[50,51]

Pulmonary emboli occur when a venous clot in the periphery dislodges and travels through the right side of the heart into the pulmonary circulation. Patients who are predisposed to developing venous thromboembolisms include women taking oral contraceptives containing estrogen because of their procoagulant effect. Patients with prolonged immobility, such as those who recently have undergone surgery or extended travel, are at an increased risk. Other risk factors include pregnancy, malignancy, trauma, and obesity.[50,52] Acquired risk factors include systemic lupus erythematosus, factor V Leiden, and antiphospholipid antibody syndrome.[50]

The most common symptom in patients with PE is shortness of breath, followed by chest pain (often pleuritic in character) and cough (sometimes with hemoptysis).[50] The patient also may complain of extremity pain, redness, or swelling, which could indicate underlying deep venous thrombosis as the source of the PE. Pulmonary embolism can present similarly to pleurisy, in which the patient experiences pain with deep inspiration. This may occur after an upper respiratory infection that causes inflammation of the parietal pleura.

Although uncommon, patients may present with the initial complaint of syncope, as this was seen in 3-4% of patients ultimately diagnosed with PE.[51] Patients may be tachycardic, tachypneic, hypoxic, and febrile with a low-grade fever.[50]

Providers must be aware that vital signs may be completely normal in young, healthy patients who present with shortness of breath and a history concerning for PE.[50] The diagnosis of PE should be considered in a patient with dyspnea or hypoxemia and clear lung sounds on physical exam.[50] Routine labs, ECG, and chest X-ray should be ordered if the diagnosis is suspected. No specific

Table 5. Simplified Revised Geneva Score

Variable	Score
Age > 65 years	1
Previous DVT or PE	1
Surgery or fracture within 1 month	1
Active malignancy	1
Unilateral lower limb pain	1
Hemoptysis	1
Pain on deep vein palpation of lower limb and unilateral edema	1
Heart rate 75-94 bpm	1
Heart rate > 94 bpm	2

Score ≤ 2 unlikely to have a current PE (probability 3% with negative D-dimer)
Adapted from: Reda LV, Choi A. Pulmonary embolism. *Emerg Med Rep* 2015;36:53.

Table 6. Pulmonary Embolism Rule-out Criteria (PERC)

- Age ≥ 50 years?
- Heart rate ≥ 100 bpm?
- O_2 saturation on room air < 95%?
- Prior history of DVT/PE?
- Recent trauma or surgery?
- Hemoptysis?
- Exogenous estrogen?
- Unilateral leg swelling?

If no to all criteria and with clinician's pre-test probability < 15%, PERC Rule criteria are satisfied and no further workup needed.
Adapted from: Reda LV, Choi A. Pulmonary embolism. *Emerg Med Rep* 2015;36:55.

abnormality may be seen, but the ECG can demonstrate sinus tachycardia or nonspecific ST- and T-wave changes.[50]

The evaluation of a patient for PE has undergone extensive study in the past 20 years with the development of risk stratification and diagnostic decision-making tools. The first step is to risk-stratify the patient using one of several tools. The most studied tools are the Wells and Geneva scores. (*See Tables 4 and 5.*) If the patient is considered to be of low risk for pulmonary embolism by either tool or by clinical gestalt, then the two approaches are of equal efficacy. The first approach is to apply the PERC criteria to determine if the potential of PE is so low as to render further testing unnecessary. (*See Table 6.*) The second approach is to measure the D-dimer, and if the D-dimer is not elevated above a validated threshold, then no further testing is necessary. Either approach reduces the potential for a missed pulmonary embolism to less than 1 in 100 to 200, with an even lower risk for subsequent sudden death.

Patients judged to be of moderate-risk also can be assessed using the D-dimer, but the risk of missing a pulmonary embolism if the D-dimer is not elevated is greater because of the heightened pre-test probability. Some experts deem this risk of missing a pulmonary embolus acceptable when balanced against the complications of prolonged oral anticoagulation. An alternative approach is to image all moderate-risk patients without D-dimer testing, similar to the recommendation for all high-risk patients.

If the patient fails to satisfy all PERC criteria, has an elevated D-dimer, or is considered at high-risk for PE, imaging is needed in the form of either a CT angiography of the chest or, less commonly, a V/Q scan. In patients who present with hemodynamic instability, a bedside echocardiogram performed by the ED provider may quickly assess for right ventricular strain, further supporting the diagnosis of PE.

Once the diagnosis is confirmed, the mainstay of therapy includes anticoagulation to prevent further clot formation.[50,52] Both low molecular-weight heparin (LMWH) and unfractionated heparin traditionally are used, and studies have shown that LMWH may have fewer side effects, which include bleeding and death.[50] Direct-acting oral anticoagulants (DOACs), such as rivaroxaban, apixaban, and dabigatran, also may be considered.[50]

If patients with diagnosed or suspected PE become hemodynamically unstable at any point, a massive clot burden must be considered. In these instances, patients may benefit from fibrinolytic therapy, especially in the situation of cardiopulmonary arrest.[55] In young patients who are found to have a large proximal PE with associated hypotension, surgical embolectomy may be performed.[50] New studies are emerging which show that low-risk patient populations with hemodynamically stable PEs may be discharged safely with close follow-up on a DOAC.[56]

Acute Aortic Dissection

Acute aortic dissection (AAD) is a rare cause of acute chest pain in patients presenting to the ED; it is about 100 to 1,000 times less common than acute MI. Most patients with AAD are middle-aged and have a history of hypertension.[57] This disorder is rare in young adults and most often is associated with predisposing conditions such as a connective tissue disorder (Marfan and Ehlers-Danlos syndromes), family history of aortic disease, known aortic valve disease, recent aortic manipulation, or known thoracic aortic aneurysm.

Table 7. Aortic Dissection Detection Risk Score

Characteristic	Criteria	Score
High-risk conditions	Marfan syndrome Family history of aortic disease Known aortic valve disease Recent aortic manipulation Known thoracic aortic aneurysm	1
High-risk pain features	Chest, back, or abdominal pain described as any of the following: • Abrupt onset • Severe intensity • Tearing or ripping	1
High-risk physical examination features	Evidence of perfusion deficit: pulse deficit, systolic BP differential, focal neurologic deficit Murmur of aortic insufficiency Hypotension or shock	1
Total Score		0-3
Interpretation 0 = low risk 1 = moderate risk 2-3 = high risk		

The most sensitive clinical feature is the patient's report of an abrupt onset of pain that is maximal at symptom onset.[58] The most useful ancillary test is a chest radiograph looking for a widened mediastinum or obscured aortic knob. Other clinical features, such as the description of the pain as tearing or ripping, a pulse deficit in one wrist, hypotension, or the auscultation of a new murmur (assumed to be caused by acute aortic regurgitation/insufficiency) generally have low sensitivity (< 25%) but high specificity (97-99%).

The Aortic Dissection Detection Risk Score (ADD risk score) has been developed to assist in the evaluation of patients with acute chest pain.[59] (*See Table 7.*) The value of the ADD risk score in clinically excluding AAD in ED patients is unsettled because this tool was derived from a population with a much higher prevalence of AAD than the typical ED population; even with a score of zero, the incidence of AAD was 4.3%.[59]

Because clotting is part of the dissecting hematoma in AAD, the D-dimer has been studied to see if it is a sensitive test for AAD, and a non-elevated value can be used to exclude dissection.[60] The overall sensitivity is excellent (98%), but even at that high value, the D-dimer alone has not been considered adequate to exclude a disorder such as AAD because of the high potential for a catastrophic outcome if not promptly detected and treated. The combination of a low-risk clinical assessment (prevalence of 6% or less) and a non-elevated D-dimer has the potential to exclude AAD to a rate of less than 1 in 300 (0.3%).[60,61] Although not zero, it seems a reasonable approach to exclude AAD in young adults.

Conclusion

The ED provider must be aware of the many causes of chest pain. It is important that the life-threatening causes are ruled out and patients are managed and dispositioned properly. The differential for chest pain is broad and not limited to only cardiac causes, especially in the younger population.

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CME/CE Questions

1. Patients with gastroesophageal reflux disease are at risk for which of the following long-term complications?
   a. Barrett's esophagus
   b. Pancreatic cancer
   c. Boerhaave syndrome
   d. Plummer-Vinson syndrome
2. Which of the following is a risk factor for development of a pulmonary embolism?
   a. Pneumonia
   b. Prolonged immobility
   c. Hypertrophic cardiomyopathy
   d. High altitude exposure
3. In addition to myocardial infarction, patients who present to the ED with chest pain after cocaine use can have which other underlying disease process?
   a. Cardiac tamponade
   b. Esophageal rupture
   c. Aortic dissection
   d. Pericarditis
4. Patients with a recently diagnosed pneumothorax should refrain from which of the following until full resolution has occurred?
   a. Coughing
   b. Light lifting
   c. Extended ground travel
   d. Diving
5. Which of the following is a recommended first-line therapy for costochondritis?
   a. Fentanyl
   b. Ibuprofen
   c. Dilaudid
   d. Tramadol
6. Which of the following is the most common cause of myocarditis worldwide?
   a. Chagas disease
   b. Parvovirus B19
   c. Adenovirus
   d. Malaria
7. What is the first ECG change seen in patients with acute pericarditis?
   a. PR prolongation
   b. Flat T waves
   c. Diffuse ST-segment elevation
   d. Delta wave formation
8. What is the most common initial complaint seen in the emergency department in patients who ultimately are diagnosed with hypertrophic cardiomyopathy?
   a. Syncope
   b. Chest pain
   c. Palpitations
   d. Dyspnea on exertion
9. What is the recommended treatment in a young patient with diagnosed pulmonary embolism with minimal symptoms and normal vital signs?
   a. tPA
   b. Clopidogrel
   c. Intra-arterial thrombectomy
   d. Low molecular-weight heparin

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Upon completion of this educational activity, participants should be able to:

- recognize specific conditions in patients presenting to the emergency department;
- apply state-of-the-art diagnostic and therapeutic techniques to patients with the particular medical problems discussed in the publication;
- discuss the differential diagnosis of the particular medical problems discussed in the publication;
- explain both the likely and rare complications that may be associated with the particular medical problems discussed in the publication.

10. Patients with suspected anxiety should be referred to which outpatient service upon discharge?
    a. Professional mental health
    b. Cardiology
    c. Neurology
    d. Drug and rehabilitation clinic

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EMERGENCY MEDICINE REPORTS

Chest Pain in Young Adults

Table 1. Common Causes of Acute Pericarditis

Infections: viral (coxsackie virus, echovirus), bacterial (staphylococcus and streptococcus)
Malignancy (i.e., breast or lung cancer, leukemia, lymphoma)
Drugs: procainamide, hydralazine
Systemic rheumatic diseases: systemic lupus erythematosus, rheumatoid arthritis, scleroderma
Miscellaneous: post-myocardial infarction, uremia, radiation, myxedema

Table 2. Some Common Causes of Myocarditis

Chagas disease (*Trypanosoma cruzi*)
Viruses
- Parvovirus B19
- Human herpes virus 6
- Coxsackie B

Bacteria
- *Corynebacterium diphtheriae*
- *Neisseria meningitidis*
- Rheumatic fever
- Lyme disease (*Borrelia burgdorferi*)

Toxins: ethanol, arsenic, iron, copper
Drugs: anthracycline chemotherapeutic agents (doxorubicin), antipsychotics (clozapine)
Immunologic conditions: scleroderma, systemic lupus, sarcoidosis

Table 3. Treatment of Small Primary Pneumothorax

Small pneumothorax (< 20%) in otherwise healthy young adult:
- Observe and administer oxygen therapy.
- Observe in ED or observation unit for 4-6 hours for small, minimally symptomatic pneumothorax.
- Obtain repeat chest X-ray prior to discharge to ensure there is no increase in size.
- Advise patient to avoid air travel and underwater diving until complete resolution.

Table 7. Aortic Dissection Detection Risk Score

Characteristic	Criteria	Score
High-risk conditions	Marfan syndrome Family history of aortic disease Known aortic valve disease Recent aortic manipulation Known thoracic aortic aneurysm	1
High-risk pain features	Chest, back, or abdominal pain described as any of the following: • Abrupt onset • Severe intensity • Tearing or ripping	1
High-risk physical examination features	Evidence of perfusion deficit: pulse deficit, systolic BP differential, focal neurologic deficit Murmur of aortic insufficiency Hypotension or shock	1
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Interpretation 0 = low risk 1 = moderate risk 2-3 = high risk		

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