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Chest Pain or Discomfort

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Definition

Pain, pressure, tightness, or other discomfort originating in or radiating to the chest constitutes an important indicator of potentially serious cardiac or cardiovascular disorders.

Technique

Chest pain is one of the most common symptoms for which adults seek the care of a physician. Because the probable etiology often can be determined from the history alone, the clinician should systematically evaluate characteristics of the pain. Precise definition of the pain allows the list of possible causes to be narrowed and guides the physical examination and choice of diagnostic tests. A central goal is to determine whether the pain is likely caused by myocardial ischemia. Coronary arteriography studies have demonstrated that patients with pain typical of angina pectoris are more likely to have obstructive coronary disease than are patients with atypical pain syndromes. Angina pectoris is a recognizable pain syndrome, and careful attention to the features of the history aids the clinician in determining whether a patient's pain is typical of angina pectoris.

Patients should be asked to describe the quality of their discomfort in their own words. Anginal pain is typically described as dull, heavy, or crushing. The patient may describe a pressure sensation rather than a true pain. Sharp, stabbing, or burning pain is less typical of angina. Anginal pain is usually located substernally or across the anterior chest. Pain located exclusively in either the left or right chest is atypical. While radiation of the pain to the left arm is typical of angina, patients with coronary artery disease also frequently have pain that radiates to the right arm or neck.

The most important part of the chest pain history is to define what aggravates and relieves the pain. Angina usually becomes worse with exertion and is relieved by rest. If the patient notes that less exertion is required to cause the pain when going out in cold weather or after eating a large meal, then the pain is likely to be caused by coronary artery disease. Patients with coronary disease frequently report pain brought on by emotional stress or sexual intercourse. Pain that is pleuritic or is brought on by moving the arms or torso is less likely to be caused by coronary disease. Patients occasionally will attribute pain to exertion, but careful questioning will reveal that the pain comes and goes with deep breaths or body movements during heavy exercise. Such pain usually is not caused by coronary artery disease.

For patients reporting chest pain brought on by exertion, clarify the amount of exertion required to cause the pain. Patients with a history of recurrent pain that now occurs with decreasing levels of exertion may require urgent treatment. Similarly, improving exercise tolerance is strong evidence that a treatment regimen has been effective. Whether or not the pain is caused by exertion, its severity provides a clue to its etiology. Pain caused by coronary disease is more likely to require patients to stop their usual activities. The frequency and duration of pain should be defined, although these data are less helpful in distinguishing the cause of attacks. Episodes of pain caused by coronary disease usually last less than an hour. Coronary disease rarely causes episodes lasting more than 12 hours without electrocardiographic changes of acute myocardial infarction.

Symptoms accompanying chest pain often provide diagnostic clues. Cough or dyspnea suggests pulmonary disorders. Diaphoresis frequently occurs in acute myocardial infarction. Dysphagia, nausea, and vomiting suggest a gastrointestinal etiology. The patient should be asked to describe the temporal relationship between the pain and associated symptoms. Accompanying symptoms are most helpful diagnostically when they occur in close proximity to the episodes of pain. Not surprisingly, patients with a history of myocardial infarction are likely to be found to have coronary disease as the cause of recurrent episodes of pain. Other conditions that should be asked about include valvular or rheumatic heart disease, hypertension, peptic disorders, esophageal disorders such as achalasia, rheumatologic disease, chronic lung disease, hyperventilation syndrome, and anxiety states.

Basic Science

Pain may originate from several different structures within the chest, including the skin, ribs, intercostal muscles, pleura, esophagus, heart, aorta, diaphragm, or thoracic vertebrae. The pain may be transmitted by intercostal, sympathetic, vagus, and phrenic nerves. The innervations of the deep structures of the thorax follow common pathways to the central nervous system, making it difficult to localize the source of pain. In pursuing the cause of chest pain, a probabilistic approach to diagnosis will aid in making decisions about patients. As indicated in Chapter 61 the clinician can estimate the probability of a particular diagnosis for a particular patient.

Several studies of coronary arteriography in patients referred for evaluation of recurrent chest pain have quantified the prevalence of coronary artery disease in different chest pain syndromes. These studies have found that 90% of patients with typical exertional angina have significant anatomic coronary disease. Patients with atypical angina have a 50 to 60% prevalence of coronary disease, while the prevalence in patients with nonanginal pain is 20 to 30%. These prevalence figures should be interpreted with caution, however. Arteriography studies consistently have shown that women have a lower prevalence of coronary disease than men. In the Coronary Artery Surgery Study, men with typical angina pectoris had a 93% prevalence of significant coronary disease, whereas women with typical angina had a 72% prevalence. Furthermore, the prevalence of coronary
artery disease is strongly influenced by age. In men, the risk of coronary disease increases steadily between the ages of 30 and 70, with little further increase above age 70. For women, the risk rises gradually until age 60 and then increases more rapidly between ages 60 and 80. Arteriography studies have reported disease prevalences in patients evaluated at referral centers, and these selected populations have a higher prevalence of coronary disease than the general population. In a study reported by Sox et al. (1981), patients who met specific criteria for nonischemic chest pain were followed to determine the cause of pain. None of these patients, who were seen at a walk-in clinic, had evidence of ischemic heart disease on follow-up. Clinicians should attempt to estimate the underlying rates of disease in their clinical settings when interpreting a patient's disease risk.

While several studies have provided data for estimating the risk of coronary artery disease, there has been less investigation of the prevalence of other disease in patients with chest pain. Of patients whose recurrent chest pain is not caused by coronary artery disease, approximately half have reflux esophagitis or esophageal contraction abnormalities. Other possible etiologies include chest wall disorders, hyperventilation, or pulmonary disease, which are relatively common in some populations.

Clinical Significance

Various schemes have been used to classify the etiologies of chest pain, but the most useful is to distinguish between acute and chronic patterns of pain. Patients with acute pain include those whose episodes are of recent onset or those who have had a recent increase in the intensity or frequency of recurrent pain. Patients with chronic pain include those who have recurrent episodes of pain occurring in a relatively stable pattern.

Causes of Chronic Chest Pain

The leading diagnostic consideration in patients with chronic chest pain is coronary artery disease. The commonest clinical presentation of coronary artery disease is recurrent angina pectoris. A helpful diagnostic feature of coronary artery disease is that the pain usually improves with specific medications. Relief of anginal pain within 3 minutes of taking sublingual nitroglycerin is strong evidence that coronary disease has caused the pain.

Decreased frequency of attacks after starting a beta blocker, calcium channel blocker, or long-acting nitrate preparation suggests that coronary artery disease is the cause.

Esophageal disease is a common cause of recurrent chest pain. Esophagitis, usually secondary to acid reflux from the stomach, frequently causes esophageal pain. The acid causes chemical damage and inflammation of the mucosa, resulting in pain that often has a burning quality. Tests to the presence of reflux esophagitis include a history of acid–peptic disease and symptoms of reflux, such as regurgitation or acid taste in the mouth. Chest pain caused by esophageal reflux tends to occur after meals and may be related to body position. Episodes of pain can be induced by bending over at the waist. They often occur at night, because the recurrent posture enhances reflux of acid into the esophagus. Relief of pain by antacids, topical lidocaine, or by specific maneuvers to reduce reflux suggests this diagnosis.

Esophageal motor disorders also commonly cause chest pain. While reflux esophagitis causes pain by irritation of the esophageal mucosa, motor disorders cause pain by contraction and spasm of the muscular wall of the esophagus. Esophageal spasm often occurs as a secondary manifestation of reflux esophagitis. As mucosal irritation and inflammation become more severe, the stimulation of local nerves leads to muscular spasm. Such patients will report a pattern of pain similar to that seen in reflux esophagitis, occurring after meals and aggravated by body position. Patients may report varying qualities of pain. With episodes of simple mucosal irritation, the pain may be reported as "heartburn," while the pain is reported as having a more severe, heavy quality during episodes of muscular spasm. Esophageal motor disorders can be independent of acid reflux disease, as in patients with achalasia or diffuse esophageal spasm. These patients show a different pattern of episodes than occurs in patients with acid reflux. The pain usually is unrelated to body position and may occur while eating instead of after meals. Dysphagia is frequently a prominent symptom in patients with primary motor disorders.

Esophageal motor disorders may be relieved by nitrates and calcium channel blockers, via relaxation of the smooth muscle wall of the esophagus. Because these agents also relieve the chest pain caused by coronary artery disease, the clinician may have difficulty using medication response as a clue to the cause of undiagnosed chest pain. The clinician must carefully interpret the results of a therapeutic trial, particularly the rapidity of response. Relief of pain within 3 minutes of a sublingual dose of nitroglycerin is more consistent with coronary artery disease than esophageal motor disorders. If relief occurs only after 10 to 15 minutes, esophageal disease is more likely. Nitrates and calcium channel blockers can relax the lower esophageal sphincter and aggravate esophageal reflux, thereby increasing symptoms of reflux esophagitis.

Myocardial ischemia sometimes occurs in the absence of fixed obstructions of the coronary arteries, resulting in recurrent chest pain. Obstructive disease of the intramural small vessels can cause ischemia. Although such lesions occur more frequently in diabetics, small vessel disease is an infrequent cause of chest pain. It should be considered only after more likely etiologies have been excluded. Valvular aortic stenosis, hypertrophic cardiomyopathy, and thyrotoxicosis can also cause myocardial ischemia. The quality and pattern of pain in these conditions usually is similar to that of coronary artery disease. These entities nearly always are accompanied by physical examination findings typical of the underlying disease, and so their detection usually is not difficult.

Coronary vasospasm can cause myocardial ischemia in the absence of obstructive coronary disease. The pain usually has a quality similar to the pain of obstructive coronary disease, but it tends to occur in an unpredictable pattern. The pain typically is not induced by exertion and may awaken the patient from sleep. Some patients report emotional stress as a trigger. The pain frequently responds to sublingual nitroglycerin, and the frequency of episodes decreases after therapy with calcium channel blockers or long-acting nitrates. Approximately 90% of patients with coronary vasospasm have ECG changes during episodes of pain. Thus, the absence of electrocardiographic changes during pain makes coronary vasospasm unlikely.

Mitral valve prolapse (MVP) is a controversial etiology of chronic chest pain. Clinical and echocardiographic studies have demonstrated that MVP is a common finding in oth-
Clinical diagnosis of chest pain is often not an option in the frequency or severity of recurrent chest pain. Because the clinician must make immediate management decisions, echocardiography may be necessary to obtain echocardiography to exclude mitral valve prolapse unless a patient has findings on physical examination and/or electrocardiogram that suggest a risk of complications.

The chest wall can cause recurrent chest pain, but the clinical diagnosis of chest wall syndromes has not been described well. The quality and location of chest wall pain vary greatly; precipitating factors are useful for diagnosis. Chest wall pain is often pleuritic and tends to be aggravated by moving the arms or torso. In most patients the pain can be reproduced by palpation or manipulation of the chest wall.

Causes of Acute Chest Pain

The clinician must make immediate management decisions for patients having new onset of chest pain or an increase in the frequency or severity of recurrent chest pain. Because some of the disorders are life threatening, it must be decided whether to admit the patient. It often is not an option to assess response to therapeutic interventions in these patients. A systematic approach to management is essential.

The leading diagnostic consideration in acute chest pain is myocardial infarction, a life-threatening condition requiring immediate medical management. The pain of myocardial infarction is similar in quality and location to angina pectoris, although usually more intense, severe, and longstanding. The pain often occurs at rest and is not relieved by nitroglycerin. It may be accompanied by diaphoresis, dyspnea, or nausea. The initial electrocardiogram shows new Q waves or ST segment elevations in only 70% of patients, so absence of these findings does not exclude infarction. Patients who are suspected of having a myocardial infarction usually require hospital admission.

The syndrome of unstable angina pectoris includes a diverse group of patients having chest pain due to coronary artery disease. Patients with unstable angina have a short-term risk of myocardial infarction, and therapeutic interventions have been shown to reduce this risk. The syndrome includes patients with new onset angina pectoris. These patients have exertional pain relieved by rest or sublingual nitroglycerin. The syndrome also includes patients with stable exertional pain but a changing pain pattern. Such patients report pain occurring at a lower level of exertion or at rest. Nitroglycerin may no longer provide relief. Various pathophysiologic mechanisms may be responsible for unstable angina. The most common mechanism is progression of the severity of atherosclerotic disease. Vasospasm aggravating atherosclerotic disease can also cause unstable angina pectoris. An important cause of unstable angina in patients with prior stable angina is alteration of the patient's medication. The patient may misunderstand the physician's prescribed regimen, or the patient may not be compliant with the regimen. Nitroglycerin can become outdated and ineffective. A careful review of the patient's medications can provide clues.

It is difficult to differentiate between unstable angina and acute myocardial infarction. Hospitalization is often required to clarify the diagnosis and facilitate treatment interventions. Serial electrocardiograms and cardiac enzymes will confirm or exclude myocardial infarction. If infarction is excluded, the patient's response to medications should be carefully evaluated. If the pain episodes do not improve substantially with cardiac medications, then the patient has either refractory coronary artery disease ("preinfarction angina") or another cause of pain. Thus, further diagnostic evaluation with imaging studies or coronary arteriography may be necessary.

Dissection of the thoracic aorta is a serious and potentially catastrophic cause of acute chest pain. Because dissection requires immediate, specific treatment, the clinician must exclude this diagnosis early. The two primary types of dissection have different clinical presentations. Proximal dissections arise in the ascending aorta but sometimes extend beyond the aortic arch. Nearly all patients with proximal dissection have pain in the anterior chest, and approximately half report radiation to the posterior chest. Approximately two-thirds have evidence of previous hypertension. Most have pulse deficits, a murmur of aortic regurgitation, or neurologic deficits, and so the physical examination is important when considering this diagnosis.

Distal dissection of the thoracic aorta arises in the descending aorta below the great vessels. It occurs predominantly in men older than age 40 who have a history of hypertension, a group also at risk for acute myocardial infarction. Nearly all patients with acute myocardial infarction have anterior chest pain, but the majority of patients with distal dissection have posterior chest pain. The pain of distal dissection may be described as "tearing" or "ripping" and does not improve with nitroglycerin. Physical findings are less frequent in distal than in proximal dissection, so the diagnosis of distal dissection may be subtle and should be considered in patients who have histories of hypertension, are hypertensive at the time of pain, or have atypical pain. Radiographic studies of the aorta may be necessary to exclude the diagnosis.

Pericarditis is a moderately common disorder, and chest pain is a frequent symptom. There are many causes of pericarditis. The pain is often retrosternal and may radiate to the left arm, suggesting an ischemic etiology. The pain is usually pleuritic or affected by body position, however, aggravated by lying down and relieved by sitting up. A pericardial friction rub suggests the diagnosis, but a rub may be absent. The electrocardiogram can show ST elevations in multiple leads, a finding that may be confused with acute myocardial infarction. Not all patients with pericarditis have ECG changes, and uricemic pericarditis is notable for the absence of ECG findings. Chest x-rays often show a normal cardiac silhouette. Because of its diverse presentation, pericarditis is difficult to exclude by routine clinical examination, but the diagnosis should be suspected if the patient does not have other likely causes of pain and has clinical evidence of a precipitating cause.

Pneumonia and pneumothorax also cause acute chest pain, but these disorders are usually easy to diagnose. The pain is characteristically unilaterial and pleuritic. Chest x-ray will establish the diagnosis.
A more difficult diagnostic problem is hyperventilation. Hyperventilation can cause pain that is atypical of angina pectoris. It may vary in location and may not be pleuritic. A useful clue is that the pain is often brought on by anxiety or emotions. The most useful tool for confirming hyperventilation is to demonstrate that the pain can be reproduced by rapid breathing.

References