American College of Emergency Physicians®

FORESIGHT

RISK MANAGEMENT FOR EMERGENCY PHYSICIANS ISSUE 56 • FEBRUARY 2003

Noncardiac Chest Pain

OBJECTIVES

After reading this issue, you should be able to:

- List the life-threatening noncardiac etiologies for chest pain.
- Describe clues in a patient's history and physical examination that might indicate a life-threatening condition.
- Discuss the diagnostic tests that can help identify the cause of chest pain.
- Explain the importance of providing clear instructions for followup care for a patient being discharged from the emergency department.

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INTRODUCTION

Chest pain is the second most common chief complaint among adults who present to emergency departments.[1] Of the total dollar losses incurred in emergency department malpractice claims, 20% is attributed to cases involving patients with chest pain.[2] Chest pain is, as all emergency physicians know, a high-volume, high-risk presentation.

Of all patients who present to emergency departments with a chief complaint of chest pain, less than one third have myocardial ischemia or infarction as the cause of the pain. Patients with nonischemic causes (Figure 1) can be difficult to evaluate, and those with suspicious histories should undergo thorough evaluation before they can be safely discharged. The workup of many adult patients with unexplained chest pain should be focused on excluding both cardiac ischemia and other thoracic cavity catastrophes, making the correct diagnosis quickly, even with limited information, and initiating treatment and appropriate specialty consultation.

Of all successful lawsuits against emergency physicians, most are related to claims of either misdiagnosis or delay in treatment. Misdiagnosis of a patient with chest pain can have serious consequences—administering thrombolytics or anticoagulants, for example, to a patient who is presumed to have a myocardial infarction but who actually has the rare aortic dissection. A delay in definitive treatment, even when the diagnosis is correct, can also create risk. If, for example, the anticoagulation of a patient who is highly likely to have pulmonary embolism is delayed pending a definitive study to be performed the next morning, the consequences can be dire for that patient.

The following scenarios illustrate issues involved in the evaluation of chest pain and the diagnosis and treatment of life-threatening noncardiac etiologies.

ON SIGHT – 1

Phillip Wright, a 23-year-old man who is confined to a wheelchair, comes to the Preston Medical Center emergency department because he has had "stabbing" pain on the right side of his chest for 2 days. He tells Jordan Roberts, MD, the emergency physician on duty, that the pain gets worse when he moves and that he uses his upper body muscles in virtually all of his daily tasks. When asked, Mr. Wright says that he cannot remember injuring himself recently. He does not feel short of breath but does say that the pain keeps him from taking a deep breath. When asked about his disability, Mr. Wright explains that he had a serious car wreck 2 years earlier and suffered an aortic rupture that required cross clamping; hypoxic injury to his spinal cord resulted in near-complete paralysis of his lower extremities.

Vital signs as recorded on the triage sheet are blood pressure 135/89, pulse rate 105, respiratory rate 26, temperature 97.8°F (36.5°C), and oxygen saturation 95% on room air. There is a focal area of point tenderness near his right pectoralis muscle; range of motion testing of the shoulder and trunk reproduces the pain. Lung sounds are clear bilaterally. Mr. Wright's leg muscles are atrophied from disuse, but his legs are otherwise nontender with no swelling noted. A chest radiograph is obtained and read as normal. Dr. Roberts makes a diagnosis of pectoralis muscle strain and discharges Mr. Wright with instructions to take ibuprofen.

The next day Mr. Wright returns to the emergency department because the pain has gotten worse, especially with breathing, and he is also experiencing dizziness. Vital signs are blood pressure 135/79, respiratory rate 24, pulse rate 120, temperature 100.4°F (38.1°C), and oxygen saturation 92% on room air. While Mr. Wright is being examined, his oxygen saturation level suddenly drops to 87% and his respirations become more labored. Despite administration of high-flow oxygen, he remains hypoxic and significantly tachypneic. Rudy Franklin, MD, intubates Mr. Wright and orders a helical CT scan, which reveals multiple large pulmonary emboli. He transfers Mr. Wright to the ICU; his stay is complicated and prolonged, and mechanical ventilation is required. While Mr. Wright is hospitalized, his parents hire a lawyer and file a lawsuit against Dr. Roberts and Preston Medical Center for the delay in making the diagnosis of pulmonary embolism.

INSIGHT – 1

Diagnosing pulmonary embolism is often very difficult because no constellation of signs or symptoms is consistently reliable. Because pulmonary embolism is relatively rare and the presentations are so various, not every "missed" case represents negligence. However, patients with known risk factors such as immobility, hematologic abnormalities, recent surgery, cancer, or pregnancy are at increased risk. (The patient in the scenario, for example, was in a much higher risk category than most emergency department patients.) Some patients who are young present with symptoms that closely, and simply, resemble musculoskeletal pain. The most common symptoms of pulmonary embolism—dyspnea and pleuritic chest pain—are absent in at least one fourth of patients with pulmonary embolism. The classic triad of hemoptysis, chest pain, and dyspnea is seen infrequently. The most common physical finding is tachypnea; it is seen in more than 90% of patients, but even this finding is absent in some. Rales, tachycardia, accentuated second heart sound, and fever are seen in half of patients. Chest wall tenderness may be a nonspecific finding, but it is present in a significant proportion of patients with pulmonary embolism.[3]

Figure 1. Nonischemic causes of chest pain.

Life-threatening

Pulmonary embolism
Thoracic aorta dissection
Boerhaave syndrome
Pneumothorax
Pericardial effusion
Pericarditis
Pneumonia

Non–life-threatening

Esophageal spasm
Gastroesophageal reflux disease
Rib fracture
Costochondritis
Pleurisy
Mitral valve prolapse

Figure 2. Wells criteria for establishing pretest probability for pulmonary embolism.[6]

Wells Score (out of 14)	Points
Clinical signs of deep vein thrombosis	+3
Alternative diagnosis less likely than pulmonary embolism	+3
Previous pulmonary embolism or deep vein thrombosis	+1.5
Heart rate >100 beats/min	+1.5
Recent surgery or immobilization	+1.5
Hemoptysis	+1
Cancer	+1

Clinical Probability

Low:	0-1
Intermediate:	2-6
High:	≥7

Among patients in whom pulmonary embolism is ultimately diagnosed, nearly one fourth will have had a normal chest radiograph initially. Abnormalities that may be found on chest radiographs include cardiac enlargement, an infiltrate or atelectasis, or an elevated hemidiaphragm, occasionally associated with a pleural effusion.[4] Although chest radiography usually is nonspecific for pulmonary embolism, the presence of two "classic" findings—Hampton hump and Westermark sign—while rarely seen, makes the diagnosis more likely. Hampton hump is an area of hemorrhagic infarction within the lung. The lung undergoes hemorrhagic infarction because of its dual vascular supply (pulmonary and bronchial). Hemorrhage into the lung causes a characteristic lesion that is opaque and has a convex contour, generally seen at the edge of the lung field. Westermark sign has been described as an abrupt pulmonary vascular cut-off as a result of an impacted embolus. This appears as a sparseness of vascular markings or relative oligemia generally seen near the hilum; this sign, however, is extremely subtle and can easily be missed.

The classic ECG finding for pulmonary embolism—$S_1Q_3T_3$—is rarely seen. The most common ECG findings, on the other hand, are tachycardia and/or nonspecific ST and T wave changes.[3]

Clinical risk stratification is the best method developed to assess how extensive a workup should be performed to rule out this diagnosis. The Prospective Investigation of Pulmonary Embolism Diagnosis,[5] or PIOPED, is the landmark study of the diagnosis of pulmonary embolism and is based on data obtained from review of almost 1,500 cases. According to this study, the pretest likelihood of having pulmonary embolism is 9%, 30%, and 68% for the low, moderate, and high pretest risk groups, respectively.[5] These findings indicate that categorizing patients into risk categories has serious implications for evaluation, treatment, and disposition.

Many authors have attempted to develop criteria that would make risk stratification more accurate. One of the more thoroughly studied strategies is the Wells criteria,[6] which use historical factors to stratify risk probability (Figure 2). In this approach, key items from a patient's signs and symptoms are assigned point values, and the total score is directly proportional to the patient's likelihood of having pulmonary embolism. Having more than one of the criteria increases a patient's clinical pretest probability from low to intermediate; having several positive findings increases the probability to high. Patients who fall into an intermediate risk profile present the greatest diagnostic challenge.

Identifying the pretest probability of pulmonary embolism enables the emergency physician to guide the workup appropriately. Results of testing should then be interpreted in light of the clinical probability. Currently, it is not clear whether spiral CT scanning of the chest can be used in the same manner as ventilation/perfusion scanning, and the results of this test should be interpreted with caution.[7]

Another diagnostic laboratory test, the ELISA D-dimer, has been studied for its usefulness in the evaluation of the risk of pulmonary embolism in an individual patient. The ELISA D-dimer has been found to be nonspecific but highly sensitive for the detection of pulmonary embolism. Thus, a negative D-dimer in patients with a low risk profile may be sufficient to rule out pulmonary embolism in this population.[8] Using a combination of tests is the best way to increase the likelihood of ruling out or discovering pulmonary embolism as the cause of a patient's symptoms.

HINDSIGHT – 1

In the Wright family's case against Jordan Roberts, the plaintiff's expert argued that Dr. Roberts did not have enough information to effectively rule out the diagnosis of pulmonary embolism clinically, and that Phillip Wright should have been considered at higher risk because of his history of chronic lower extremity stasis. The plaintiff's expert asserted that an immobilized patient with acute chest pain who is tachycardic (105) and has a low oxygen saturation level (95%) should be considered as having pulmonary embolism until proved otherwise. If Dr. Roberts had diagnosed pulmonary embolism, the plaintiff's expert contended, Phillip Wright would not have had such a prolonged or complicated hospital stay. The defense expert agreed that, because additional testing was justified but not done during the initial visit, the case would be difficult to defend in court. The hospital and Dr. Roberts agreed to settle the case.

- The symptoms of pulmonary embolism can be subtle. There is no "classic" presentation.
- Abnormal vital signs such as a persistent tachycardia must be addressed and explained.
- Emergency physicians should stratify patients with possible pulmonary embolism into risk groups and use an effective diagnostic strategy to make or rule out the diagnosis.

ON SIGHT – 2

Jerry Wilson, 42, has come to the emergency department at Forums Hospital many times, and each time his concern has been the same: extreme anxiety and a sudden sensation that he is "about to die." When he presents again on a Wednesday afternoon, he describes these same symptoms, just as he did on his last visit 4 months earlier, but he tells the triage nurse that he also has a headache on the right side of his head and that he is dizzy and "can't stand this light" in the triage room. "And my arm feels weird," he says.

Kellen Smith, MD, reviews the nurse's notes and asks Mr. Wilson if anything else is bothering him. Mr. Wilson then remembers that he had some pain in his chest and back a few hours earlier, but that it lasted only about 15 minutes then went away. He adds that he might have had some muscle spasms in his back, and points to an area of his lower thoracic spine.

On physical examination, vital signs are blood pressure 150/85, pulse rate 88, respiratory rate 18, temperature 98.4°F (36.89°C), and oxygen saturation 97%. Significant left side weakness on grip strength, flexion, and extension of his arm is noted on examination. An ECG shows normal sinus rhythm with no acute ST or T wave abnormality. Chest radiograph reveals no evidence of infiltrate or pneumothorax, and a normal mediastinum. The preliminary reading of the CT scan of the head is negative for acute bleeding.

After making a diagnosis of acute stroke, Dr. Smith talks with the neurologist on call, who admits Mr. Wilson to the neurology unit. Heparin therapy is started. When Mr. Wilson arrives on the floor 8 hours later, a nurse conducting a neurologic check notes that he has a worsening dense left hemiparesis. While she is on the telephone with the neurologist, Mr. Wilson suffers cardiac arrest and dies. Postmortem examination reveals a dilated aortic arch with a dissection that progressed into the right carotid artery. A large clot is noted extending from the ascending arch and completely obstructing the right carotid artery. The final reading of the initial CT scan obtained in the emergency department notes early ischemic changes of the right hemisphere.

INSIGHT – 2

Thoracic aorta dissection is rare, with an estimated incidence of 1,500 to 8,500 cases per year in the United States, as compared to 4.6 million annual emergency department visits for chest pain. The diagnosis is initially suspected in only 15% to 43% of patients in whom it is eventually diagnosed. With dissection, hypertension is a major but nonspecific risk factor; abnormality of the aortic valve, including bicuspid and replacement valves, is an important risk factor. Thoracic aorta dissection is very uncommon in patients who are younger than 40 unless it is associated with specific diseases such as Marfan, Turner, or Ehlers-Danlos syndrome.[9]

Thoracic aorta dissection is often diagnosed incidentally during the workup of chest pain. The classic signs and symptoms include severe tearing chest pain, radiation of pain to the back, focal neurologic deficit, diastolic murmur consistent with aortic regurgitation, blood pressure differences of 20 mm Hg systolic between arms, and differences in pulse strength. However, most patients do not present with all of these classic findings. Nearly 90% of patients report a sharp pain that is most severe at onset and often radiates to the back.[9] Having more than one of the classic symptoms increases the likelihood of the diagnosis, so emergency physicians should ask specifically about the quality, onset, and radiation of the pain; omission of one or more of these questions might hinder the physician's ability to make the diagnosis. The absence of clinical findings, however, does not rule out this condition.

An extremely small number (2%) of patients with thoracic aorta dissection present with stroke symptoms.[10] Extension of the dissection into the carotid artery will cause various neurologic findings; Horner syndrome (ipsilateral miosis with ptosis and anhydrosis), for

example, is one such neurologic finding. Dissection should be considered in any patient who presents with classic chest pain and associated stroke symptoms.

In a patient with dissection, chest radiography typically will reveal a widened mediastinum, an indistinct aortic knob, or an abnormal aortic arch. However, in 15% to 20% of cases, the chest radiograph will be normal.[9]

Several other diagnostic modalities are now used to make a definitive diagnosis of thoracic aorta dissection. Computed tomography is the most commonly used study; it has shown a sensitivity of 83% to 94% with a specificity of 87% to100%.[11] In most cases, the detection rate using transesophageal echocardiography, or TEE, is reported to be better than CT scanning, with a sensitivity of 97% to 99% and specificity of 97% to 100%. Magnetic resonance imaging has greater than 90% sensitivity and greater than 95% specificity; some believe MRI is superior to TEE because it can show the specific site of the intimal tear, type and extent of dissection, and presence of aortic insufficiency. There are limitations to both of these diagnostic studies. Although faster and less expensive, TEE is not available in many areas for emergent diagnosis; MRI cannot be used in patients with metallic foreign bodies and also is limited by availability. Aortic angiography, previously considered the gold standard for the diagnosis of thoracic aorta dissection, has a sensitivity of 80% to 90% and a specificity of 90% to 100% with a positive predictive value of about 95%.[11]

Although a CT scan of the chest is very good at picking up the diagnosis, using it as a screening test in every patient with any type of chest pain to rule out aortic disease defies logic and is not cost-effective. To indiscriminately use CT scanning in patients with a low pretest probability of disease is likely to significantly increase the false-positive rate of the test and increase the complication rate from the resulting testing for normal patients. A patient who presents with chest pain, especially pain radiating to the back, associated with stroke symptoms should undergo further diagnostic testing (commonly a CT scan) to evaluate the aorta. If the patient has a normal CT scan and a very high clinical likelihood of dissection, a second study such as angiography may be needed to adequately rule out this condition.

HINDSIGHT – 2

Months later, Kellen Smith and the neurologist and Forums Hospital were named in a wrongful death lawsuit. Experts testified during their depositions that, although the diagnosis of thoracic aorta dissection is difficult to make, both physicians should have asked the patient about his chest pain. The plaintiff's attorney contended that further evaluation of this component of the history would have likely led to the correct diagnosis. Because the correct diagnosis was not made until the postmortem examination, and because the patient was given heparin, the hospital's attorney did not want to see the case go to trial. The case was weakened by the delayed interpretation of the CT scan by radiology and was ultimately settled before trial. Since that time, the hospital has changed its system for prompt reading of CT scans, especially in patients who will be receiving anticoagulant or thrombolytic therapy.

- Chest pain associated with focal neurologic deficits should raise the suspicion for an aortic etiology.
- History-taking that includes specific questions about the patient's chest pain (quality, severity at onset, radiation) may elicit information that will raise the suspicion for thoracic aorta dissection.
- A normal-sized mediastinum is seen on posteroanterior chest radiographs in 15% of patients with aortic dissection. Echocardiography, CT scanning, MRI, or angiography may be required to make the diagnosis.

ON SIGHT – 3

For the past 5 weeks, 38-year-old Julia Wolf has been having chest pain. She has already been to the emergency department at Fran Kerwin Hospital twice in the past month, but on a Tuesday evening when the pain seems to be worse than usual, she decides to go again. Mrs. Wolf tells the triage nurse that she has a "stabbing" pain in the middle of her chest, along with some pain in her left shoulder. "Those other doctors did a bunch of tests and still couldn't find out what was wrong," she tells the nurse. "And now it hurts a lot worse, and I don't have any energy, and if I do anything I can't catch my breath." When asked, she says that she does not have any cold symptoms and has not been around anyone who is sick.

In her notes, the triage nurse records Mrs. Wolf's vital signs as blood pressure 110/75, pulse rate 85, respiratory rate 20, and temperature 97.7°F (36.5°C). Seth Taylor, MD, notes that Mrs. Wolf is obese and in no distress, but impatient. Examination of the head and neck is not remarkable. Chest wall and left shoulder are nontender to palpation. Lungs are clear, and heart rate is regular with no murmur. Abdomen is soft, and extremities show no signs of edema. Mrs. Wolf reports some dizziness when she stands, but her neurologic examination is nonfocal.

Dr. Taylor reviews the charts from Mrs. Wolf's two previous emergency department visits. On both of these visits the diagnosis was musculoskeletal chest wall pain, and she was sent home with pain medications. The second physician had documented that Mrs. Wolf did not follow up with her primary care physician as directed, and he wrote that he advised her again to schedule an appointment with her doctor.

Concerned about Mrs. Wolf's multiple visits for the same complaint, Dr. Taylor decides to pursue a more complete workup than had been done previously. He gets an ECG and notices that the height of the R wave of the QRS complex varies slightly every other beat, consistent with electrical alternans. When he initially reviewed her chest radiograph, he noticed that the heart was mildly prominent. But when he compares it to her chest radiograph obtained 4 weeks earlier, he sees that the heart size is now significantly enlarged. He orders a CT scan of the chest, which reveals a large pericardial effusion and a lung mass. Dr. Taylor admits Mrs. Wolf. She undergoes a pericardial window and a thoracotomy that confirm the presence of a lung carcinoma.

INSIGHT – 3

Patients with chronic pericardial effusion may present with anorexia, dyspnea, cough, and, occasionally, chest pain. Specific signs of chronic pericardial effusion—tachycardia, jugular venous distention, hepatomegaly, peripheral edema—are seen in less than half of patients.[12] The classic presentation of cardiac tamponade, Beck triad (hypotension, distended neck veins, muffled heart sounds), is not always present; in fact, it is seen in only 10% to 30% of patients. The presence of a "water-bottle"–shaped heart on a chest radiograph is suggestive of disease, although not specific. Electrical alternans may be seen on ECG. Pulsus paradoxus (accentuation of the normal decrease in the systolic blood pressure during inspiration by greater than 20 mm Hg) is seen in patients with pericardial tamponade. Although pulsus paradoxus is often mentioned in discussions of chronic pericardial effusion, assessment for it is often done incorrectly; it may be absent if the patient is hypovolemic, making this sign less useful.

The etiology of most pericardial effusions is unknown. Most known causes are related to viral illnesses, uremia, or metastatic lesions. Symptoms of tamponade occur when the fluid accumulation in the pericardial space attains a critical volume that produces sufficient elevation of pressure to impair diastolic filling and to lower stroke volume and cardiac output.[13] If the patient is unstable, an emergent pericardiocentesis can be lifesaving. In some facilities, sonography- or CT-guided pericardiocentesis can be used to relieve the fluid. The stable patient can be sent for elective drainage of the fluid, during which a surgeon may perform a pericardial window.

HINDSIGHT – 3

All three emergency physicians who evaluated Julia Wolf's chest pain were summoned for a review with the hospital's peer review committee. All three encounters had been documented thoroughly, as had Mrs. Wolf's noncompliance with the recommended followup care. As a result, when a case was brought against the physicians, they were found to have treated the patient appropriately, and that any perceived delay in diagnosis did not affect care or outcome. A defense verdict was returned.

- Clearly and carefully document in the medical decision-making discussion why another, more dangerous diagnosis is unlikely.
- Review of charts from previous emergency department visits, if available, may be critical to the diagnostic evaluation of a patient.
- If a patient is being discharged home, make sure that the instructions for followup care are clear, that the patient understands them, and that both the instructions and the discussion are documented in the chart.

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1. Which of the following is the most likely presenting sign or symptom in a patient with a pulmonary embolism?
   A. chest pain
   B. dyspnea on exertion
   C. shortness of breath
   D. tachypnea
   E. unilateral swelling of an extremity

2. Which of the following is not a risk factor for pulmonary embolism?
   A. cancer
   B. chronic immobility
   C. history of hypertension
   D. pregnancy
   E. venous stasis

3. In a patient with a low clinical index of suspicion for pulmonary embolism, what is the likelihood of detecting the condition on a ventilation/perfusion study?
   A. 9%
   B. 15%
   C. 30%
   D. 45%
   E. 75%

4. Which study is most likely to be abnormal in a patient with an acute aortic dissection?
   A. arterial blood gas
   B. chest CT scan with contrast
   C. chest radiograph
   D. lower extremity doppler
   E. ventilation/perfusion scan

5. What is the most sensitive sign or symptom of a pericardial effusion causing tamponade?
   A. anorexia
   B. chest pain
   C. electrical alternans
   D. jugular venous distention
   E. water-bottle–shaped heart

6. Which of the following best describes electrical alternans as an ECG finding?
   A. beat-to-beat variation of the P wave axis
   B. fluctuating beat-to-beat variations of the QRS amplitude
   C. multifocal sinus tachycardia
   D. reduction of the QRS amplitude
   E. $S_1Q_3T_3$ pattern

7. In a patient with an aortic dissection, which of the following is the least likely presenting sign or symptom?
   A. chest pain radiating to the back
   B. continuous murmur
   C. focal neurologic deficit
   D. Horner syndrome
   E. pulsus paradoxus

8. Which of the following statements regarding aortic dissection is correct?
   A. chest pain is constant and does not resolve
   B. chest x-ray is abnormal in 95% of patients with dissection
   C. CT scanning is as sensitive as TEE for detecting dissection
   D. diagnosis of dissection is commonly made on the first visit
   E. sudden sharp pain is reported by almost all patients

ANSWERS

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Record your answers here:

Question	A	B	C	D	E
1	☐	☐	☐	☐	☐
2	☐	☐	☐	☐	☐
3	☐	☐	☐	☐	☐
4	☐	☐	☐	☐	☐
5	☐	☐	☐	☐	☐
6	☐	☐	☐	☐	☐
7	☐	☐	☐	☐	☐
8	☐	☐	☐	☐	☐

Answers from Issue 55, October 2002

1-C, 2-A, 3-B, 4-A, 5-E, 6-A, 7-B, 8-A

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