Chest Pain
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Epidemiology

Every year 6.2 million people are seen in U.S. emergency departments (EDs) with complaints of chest pain, which accounts for roughly 6% of ED visits and is the second most common reason for such visits. The differential diagnosis of chest pain ranges from benign causes, such as muscle strain, to the immediately life-threatening ones, such as acute coronary syndrome, pulmonary embolism, and aortic dissection. Although the focus in patients with chest pain remains appropriately on life-threatening causes, a majority of patients have benign or indeterminate diagnoses after ED evaluation. In one study of ED patients with symptoms consistent with acute cardiac ischemia, only 8% had acute myocardial infarction (AMI) and 9% had unstable angina.1 Another investigation of patients evaluated in the ED for nontraumatic chest pain found that AMI was diagnosed in 4%, unstable angina or stable coronary disease in 7.5%, and pulmonary embolism or aortic dissection in less than 1%.2 Given the potentially lethal nature of conditions manifested as chest pain and the lack of sensitivity or specificity, in many instances, of the history and physical examination, the emergency physician (EP) must have an organized approach, a complete differential diagnosis, and a thorough understanding of assessment and management of this common complaint.

Pathophysiology

In the differential diagnosis of patients with chest pain, one must consider the five groups of structures in the thorax: cardiac (heart and pericardium), pulmonary (lungs and pleura), gastrointestinal (esophagus and upper abdominal contents), vascular (aorta and great vessels), and musculoskeletal (chest wall). Chest discomfort is experienced through three distinct pathways, as follows:

Visceral pain, from internal structures such as the heart, lungs, esophagus, and aorta, may be difficult for the patient to define or localize. It is experienced as discomfort or a vague sensation and is often difficult to pinpoint.

Somatic pain, from chest wall structures and the parietal pleura, is often easier to describe and localize. Somatic pain may be sharp or stabbing and exacerbated by movement or position.

Referred pain, from irritation or inflammation of the upper abdominal contents, is a form of visceral pain that may be perceived in the chest wall, shoulder, or upper part of the back.

A differential diagnosis based on anatomic structures within the chest is presented in Box 54.1.

Presenting Signs and Symptoms

Most patients with nontraumatic chest pain warrant high triage priority and an early electrocardiogram (ECG) (recommended within 10 minutes) to evaluate for AMI. Patient stabilization, evaluation of the history, physical examination, and diagnostic and therapeutic interventions proceed simultaneously. As assessment continues, interventions are refined (Box 54.2). Importantly, the history and physical findings alone are often inadequate to definitively establish or exclude life-threatening diagnoses.

The EP should keep the following points and issues in mind during assessment of a patient with chest pain:

- Use the term discomfort as opposed to pain to facilitate communication.
- Do not ascribe partially reproducible pain to a musculoskeletal cause. Pain arising from inflammation of the pericardium (secondary to AMI or pericarditis) or inflammation of the pleura (pulmonary embolism, pneumonia, or pleurisy) can be partially reproduced by palpation.
- Chest pain that is completely pleuritic (present on inspiration) or completely reproducible significantly decreases suspicion for cardiac causes and raises suspicion for pulmonary or musculoskeletal causes. Partially pleuritic (worse with inspiration) or partially reproducible chest pain has much less predictive value.

Observation and repeated testing are extremely valuable in a patient with chest pain in whom the diagnosis is unclear.

Rapid ruling out of acute myocardial infarction can be performed with serial cardiac marker testing once an appropriate interval after symptom onset has elapsed (8 hours for troponin I or T), although shorter intervals may be acceptable if immediate stress testing is performed.

Normal cardiac marker values do not exclude unstable angina.

Consider life-threatening diagnoses other than acute myocardial infarction in patients with chest pain, including aortic dissection, which is frequently missed and often manifested atypically.

KEY POINTS

- Observation and repeated testing are extremely valuable in a patient with chest pain in whom the diagnosis is unclear.
- Rapid ruling out of acute myocardial infarction can be performed with serial cardiac marker testing once an appropriate interval after symptom onset has elapsed (8 hours for troponin I or T), although shorter intervals may be acceptable if immediate stress testing is performed.
- Normal cardiac marker values do not exclude unstable angina.
- Consider life-threatening diagnoses other than acute myocardial infarction in patients with chest pain, including aortic dissection, which is frequently missed and often manifested atypically.

EPIDEMIOLOGY

PATHOPHYSIOLOGY

REFERENCES


ACUTE CORONARY SYNDROME

EPIDEMIOLOGY

Several risk stratification systems have been proposed for acute coronary syndrome. These systems have been shown to help in risk stratification, thereby enabling triage decisions. They have never been shown to improve the ability to formulate discharge decisions in comparison with practitioner judgment. The American College of Cardiology and the American Heart Association have published criteria to determine a patient’s risk for coronary artery disease and adverse outcomes from acute coronary syndrome. These guidelines are cumbersome and more appropriately applied to patients with documented disease than to undifferentiated ED patients. A simplified approach to stratifying risk is to determine whether the patient has definite acute coronary syndrome, probable acute coronary syndrome, or possible acute coronary syndrome, as follows:

• Patients with definite acute coronary syndrome are those with (1) changes diagnostic of ischemia or infarction on an ECG, (2) diagnostic elevation of serum cardiac markers, or (3) evidence of new heart failure or shock directly attributable to an acute ischemic event.
• Patients with probable acute coronary syndrome are those in whom suspicion for acute coronary syndrome is high but definitive criteria are lacking. An example is a patient with a classic history for acute coronary syndrome or whose cardiac marker values are slightly elevated but still below the diagnostic cutoff and who does not have clear ECG evidence of ischemia.
• Patients with possible acute coronary syndrome constitute the majority of patients with chest pain. They have atypical histories, their ECG findings are normal or unchanged from previous studies, or suspected alternative causes are triggering their symptoms.
This chapter is focused on patients with possible acute coronary syndrome. After chest radiography, a substantial proportion of such patients require further testing and observation, such as serial cardiac biomarker testing or other tests to evaluate for alternative diagnoses.

The challenge for the EP lies in determining when and which patients with possible acute coronary syndrome can be safely discharged home. At this time no definitive answer exists. A critical error, however, is failure to identify features that warrant further evaluation. Characteristics such as advanced age, known coronary artery disease, diabetes, pain similar to that of a previous myocardial infarction, worsening of typical angina, pressurelike or squeezing discomfort, and radiation of pain to the neck, left shoulder, or left arm have all been shown to increase the likelihood of AMI.

### PRESENTING SIGNS AND SYMPTOMS

The classic manifestation of AMI is discomfort that feels like an elephant sitting on one’s chest; radiates to the left shoulder, arm, or jaw; and is associated with shortness of breath, nausea, or diaphoresis. Patients may describe their discomfort with a clenched fist against their chest, a finding known as the Levine sign. Physical examination demonstrates tachycardia, diaphoresis, and if the infarction has compromised left ventricular function, findings of acute heart failure such as hypoxia, tachypnea, elevated jugular venous pulsations, and bilateral rales. The classic manifestation in patients with unstable angina is a sense of discomfort or pressure that is similar to that of AMI but transient in nature. Patients with unstable angina experience similar associated symptoms typically brought on by exertion and relieved with rest or nitroglycerin. In practice, these classic findings are the exception, not the norm.

Risk factors for coronary artery disease predict a patient’s risk for the development of ischemic heart disease over a period of many years but are only moderately predictive of acute coronary syndrome in the ED. Most important, it is well established that a lack of cardiac risk factors by itself does not place a patient at low risk for acute cardiac events.

Historical and examination features that raise or lower the likelihood of acute coronary syndrome are described in Box 54.3 and Table 54.1. It is important to remember that the presence of lower-likelihood features does not exclude the diagnosis of acute coronary syndrome. One study of patients with AMI found that 22% had sharp or stabbing pain and 13% had partially pleuritic pain.5

The physical examination should be thorough, and findings suggestive of an alternative diagnosis may be helpful but are often not adequately specific to exclude acute coronary syndrome. For example, in 7% of patients with AMI, the pain is fully reproduced by palpation.5

### DIAGNOSTIC TESTING

In adult patients with chest pain or acute coronary syndrome equivalents, an ECG is recommended within 10 minutes of ED arrival. Thirty percent to 50% of patients with AMI have diagnostic ECG findings, 40% to 70% have nonspecific ECG findings, and 1% to 10% have normal ECG findings. Nonspecific or unchanged ECG findings do not affect the likelihood of acute coronary syndrome; although a normal ECG does not exclude acute coronary syndrome, it significantly decreases the likelihood. Comparing the ECG with previous or serial ECGs can improve sensitivity and specificity. A right-sided ECG is recommended in all patients with inferior ST changes, and a posterior lead ECG is recommended if ST depression is present in septal leads V₄ through V₉. The ECG helps guide not only diagnosis but also therapy decisions (i.e., the presence of ST-segment elevation in AMI is a primary criterion for thrombolytic therapy). As with all tests, it is imperative that the ECG findings be interpreted in context.

An understanding of cardiac biomarkers is pivotal to excluding possible AMI in the ED. Currently, AMI is defined as the rise and fall of serum cardiac biomarkers in the presence of at least one of three other findings: ischemic symptoms, a pattern of progressive ischemic changes on ECG, or imaging evidence of a new regional wall motion abnormality.

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**Table 54.1** Features of Chest Pain That Lower the Likelihood of Acute Myocardial Infarction*

<table>
<thead>
<tr>
<th>Feature</th>
<th>Frequency in Patients with Acute Ischemia (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pleuritic pain</td>
<td>13</td>
</tr>
<tr>
<td>Pain that is reproducible with palpation or movement</td>
<td>7</td>
</tr>
<tr>
<td>Sharp, stabbing pain</td>
<td>22</td>
</tr>
<tr>
<td>Pain that lasts seconds or is constant for 24 hours or longer</td>
<td>NA</td>
</tr>
</tbody>
</table>

*NA, Not available
*Likelihood ratio of approximately 0.3.
Current guidelines recommend the use of cardiac troponins for the evaluation of all patients with suspected acute coronary syndrome. Troponins, regulatory proteins found in cardiac muscle, are composed of three subunits: I, T, and C. Cardiac subunits I and T are genetically distinct from the skeletal muscle forms, and no cross-reactivity occurs on immunoassays. Within 2 to 8 hours of AMI, troponin levels are abnormal and remain so for 7 to 10 days (Table 54.2). Detectable troponin but at a value below the diagnostic cutoff for AMI still portends a higher risk for adverse outcomes. Nonspecific elevations, especially of troponin T, can occur with renal dysfunction, pulmonary embolism, septic shock, decompensated heart failure, myocardial contusion, pericarditis, and myocarditis. Cardiac troponins are more sensitive and specific than creatinine kinase, MB fraction (CK-MB), and myoglobin for cardiac muscle damage, and contemporary troponin assays identify the majority of AMIs within 3 hours, thus limiting the utility of CK-MB and myoglobin.

CK-MB is an enzyme present at higher percentages in cardiac muscle than in skeletal muscle, and it is relatively specific for cardiac muscle damage. False-positive results occur in patients with renal failure and in those with large amounts of skeletal muscle injury, such as seen with rhabdomyolysis. The CK-MB index improves the specificity of the biomarker by comparing the ratio of CK-MB with total CK. CK-MB subforms CK-MB1 and CK-MB2 rise earlier than CK-MB and are detectable 4 to 6 hours after injury, with a sensitivity of 92% achieved at 6 hours. Unfortunately, laboratory testing for CK-MB1 and CK-MB2 is not widely available.

Myoglobin is a heme protein in skeletal and cardiac muscle whose levels rise rapidly within 2 to 4 hours and return to normal within 24 to 36 hours. Its utility is limited by inadequate sensitivity and specificity, and its measurement is primarily used in combination with that of CK-MB and troponin as a point-of-care “triple-marker” assay. Studies have demonstrated that specificity can be improved through evaluation of the rate of myoglobin elevation (delta myoglobin) over a 1- to 2-hour period. It is recommended that delta myoglobin cutoff values of 25% to 40% be used to indicate abnormality. Other cardiac markers are being investigated, and their roles are being determined.

Recommendations based on the best available evidence and consensus argue against using a single cardiac marker value within 6 hours of the onset of symptoms to exclude AMI. For patients initially seen more than 6 to 8 hours after onset of the most recent episode of pain, a single negative cardiac marker value is often adequate to exclude AMI (but not unstable angina) in those with possible acute coronary syndrome. A period of observation that includes repeated ECG and serum CK-MB and troponin measurements can be used to rapidly rule out AMI at 6 and 8 hours after the onset of symptoms, respectively (see Table 54.2). Some evidence shows that a more accelerated testing approach is appropriate when such testing is followed immediately by stress imaging. In fact, one investigation found that it was safe to test patients with chest pain on an exercise treadmill immediately without initially determining cardiac marker values. The patients involved in this study, however, were at extremely low risk, with normal or nearly normal ECG findings, no evidence of heart failure, and the ability to exercise, and they were found to have only a 1% rate of AMI.7

OBSERVATION UNITS AND PROTOCOLS

Increases in resource utilization, cost, and medicolegal concerns associated with patients evaluated in the ED for chest pain have led to the advent of rapid assessment protocols and chest pain units. These strategies aim to lower admission rates and cost of care while minimizing the inappropriate discharge of patients with unrecognized acute coronary syndrome. Approaches vary widely in these strategies, and most published methodologies involve immediate stress testing of low-to moderate-risk patients after a period of observation with serial ECGs and cardiac marker testing. Protocol-driven strategies increase the number of patients evaluated, accelerate the rate of evaluation, lower the number of missed events, and may save overall costs.

After a period of observation, repeated cardiac marker testing, and either continuous or intermittent ECG

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**Table 54.2 Characteristics of Cardiac Marker Levels After Myocardial Infarction (MI)***

<table>
<thead>
<tr>
<th>CARDIAC MARKER</th>
<th>TIME OF RISE (HOURS AFTER MI)</th>
<th>TIME OF PEAK (HOURS AFTER MI)</th>
<th>TIME OF RETURN TO BASELINE (AFTER MI)</th>
<th>TIME OF SECOND MEASUREMENT (HOURS AFTER MI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myoglobin</td>
<td>&lt;3</td>
<td>4-9</td>
<td>&lt;24 hr</td>
<td>---</td>
</tr>
<tr>
<td>Creatine kinase—MB form (CK-MB)</td>
<td>3-8</td>
<td>9-30</td>
<td>1-3 days</td>
<td>6-10</td>
</tr>
<tr>
<td>CK-MB subforms</td>
<td>1-3</td>
<td>4-6</td>
<td>18-24 hr</td>
<td>6-10</td>
</tr>
<tr>
<td>Troponin T</td>
<td>2-6</td>
<td>10-24</td>
<td>10-15 days</td>
<td>8-12</td>
</tr>
<tr>
<td>Troponin I</td>
<td>2-6</td>
<td>10-24</td>
<td>7-10 days</td>
<td>8-12</td>
</tr>
</tbody>
</table>

*The American College of Emergency Physicians recommends an initial measurement of cardiac markers and a second measurement at the given intervals for rapid exclusion of MI in low- to moderate-risk patients. It is unclear whether a second measurement is needed if the time between symptom onset and the patient’s arrival at the ED exceeds the recommended interval for the second measurement. See Fesmire FM, Decker WW, Diercks DB, et al. Clinical policy: critical issues in the evaluation and management of adult patients with non-ST-segment elevation acute coronary syndromes. Ann Emerg Med 2006;48:270-301.
monitoring, patients in whom the ECG findings are unremarkable and cardiac biomarker results are negative undergo stress testing. Guidelines recommend that the stress test be performed within 72 hours of ED discharge; a majority of published reports describe stress testing before discharge.1

The most common adjunctive test is the continuous ECG treadmill stress test (TST). Patients with normal TST results under these circumstances have been found to have acceptably low rates of missed ischemia and adverse events. Unfortunately, a reasonable percentage of patients are poor candidates for an ECG TST (18% in one study) because of either an inability to ambulate at a moderate (2.5 mph) pace or the presence of confounding baseline ECG findings, such as left ventricular hypertrophy, left bundle branch block, ventricular-paced rhythm, or preexcitation syndrome. ECG TSTs also have a 5% to 25% nondiagnostic rate, depending on the patient population and protocol used. Patients in whom the ECG TST cannot be used must undergo stress imaging studies. Patients with nondiagnostic or abnormal ECG TST results should undergo further evaluation, which usually requires admission.

Although the percentage of low-risk chest pain patients in whom acute coronary syndrome is diagnosed during their hospital evaluation is low, 0.5% to 5%, the admission rate of patients who have been evaluated in a chest pain unit ranges from 10% to 50%. Patients discharged after a rapid assessment protocol or evaluation in a chest pain unit should receive outpatient follow-up soon.

**TREATMENT**

Patients with possible acute coronary syndrome should receive aspirin. In patients with ongoing ischemic symptoms, nitrates may be given. Nitrates have never been shown to improve outcomes in patients with acute coronary syndrome, and recently, the response to nitrates has been shown to lack predictive value in the diagnosis of acute coronary syndrome. Their use in these low-likelihood patients should be weighed against the risk for hypotension or even headache. Analgesics such as morphine are given to patients with discomfort unresponsive to nitrates. Controversy surrounds the use of β-adrenergic receptor blockers. Currently, routine administration of intravenous beta-blockers in the prehospital setting or ED is not recommended.4 Therapies such as heparin, clopidogrel, and glycoprotein IIb/IIIa inhibitors have been shown to be of benefit primarily in patients with definite acute coronary syndrome and therefore should not be used in this low-likelihood group.4

**DISPOSITION**

It is important to acknowledge that the clinician cannot obtain perfect sensitivity in the assessment of patients with any disease. An analysis of multiple studies on acute coronary syndrome found that clinicians missed fewer AMIs when they admitted more patients.5 Clearly, there is a limit to this strategy, although evidence does suggest that providing resources to increase the number of patients undergoing evaluation may reduce the proportion of acute coronary syndrome that is missed. This appears to be a cost-effective approach but depends on multiple factors that may be outside the clinician’s and even the institution’s control. Even when clinicians are confident of an alternative diagnosis, subsequent adverse cardiac events may occur, with a 2.8% rate documented in one large study.59 The acceptable “miss rate” depends on the following factors:

- Risk aversion of the clinician
- Risk aversion of the patient
- Resources available
- Perceived risk of litigation for an adverse outcome even when the care is appropriate

Even patients with chest pain who undergo thorough evaluation that yields unremarkable findings experience a low but meaningful rate of adverse events. On the basis of these considerations, clinicians must decide the level of acceptable risk for missed acute coronary syndrome while realizing there is a finite rate of adverse outcomes. It is best for the EP to explain these risks to the patient, clearly document the reasoning, clearly document the patient’s understanding, provide appropriate discharge instructions, and document the recommendations for follow-up.

**AORTIC DISSECTION**

Aortic dissection is a tear in the intimal lining of the aorta. It is a distinct entity from a dilated aortic aneurysm, which involves pathologic dilation of the intima, media, and adventitia and can result from traumatic aortic injury. The reported incidence is 2.9 cases per 100,000 patients per year, which corresponds to roughly 5000 new adult cases per year in the United States. Missing or incorrectly diagnosing this condition can be fatal, especially if anticoagulation or fibrinolysis is initiated. Risk factors for aortic dissection include hypertension, Marfan disease, pregnancy, valvular disease, syphilis, and cocaine use.

**PRESENTING SIGNS AND SYMPTOMS**

The classic manifestation of aortic dissection is acute (with maximum intensity at onset), severe, tearing chest pain that radiates to the back in patients with a history of hypertension. On examination, patients may exhibit pulse deficits or an aortic insufficiency murmur. Unfortunately, the classic manifestation is the exception and the clinical spectrum is broad (Table 54.3). Symptoms frequently mimic more common disorders, and the clinician must maintain a high index of suspicion.12

No single finding or combination of findings has been determined to be sensitive or specific enough to direct the evaluation for aortic dissection. Given that the diagnosis is frequently missed, the EP should have a low threshold for evaluating patients for aortic dissection when it is part of the differential diagnosis. Aortic dissection should be considered in a patient with any of the following features:

- Severe chest pain
- Pain that occurs in more than one anatomic distribution (chest and back, chest and abdomen)
- Pain accompanied by a focal neurologic complaint
**Table 54.3** Frequency of Symptoms and Physical Findings in Patients with Aortic Dissection

<table>
<thead>
<tr>
<th>FEATURE</th>
<th>FREQUENCY (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptoms</td>
<td></td>
</tr>
<tr>
<td>Pain</td>
<td>95</td>
</tr>
<tr>
<td>Severe or worst ever</td>
<td>90</td>
</tr>
<tr>
<td>Abrupt onset</td>
<td>85</td>
</tr>
<tr>
<td>Location in chest</td>
<td>75</td>
</tr>
<tr>
<td>Location in chest and back or back alone</td>
<td>50</td>
</tr>
<tr>
<td>Tearing or ripping</td>
<td>50</td>
</tr>
<tr>
<td>Syncope</td>
<td>10</td>
</tr>
<tr>
<td>Physical Findings</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td>50</td>
</tr>
<tr>
<td>Aortic insufficiency murmur</td>
<td>30</td>
</tr>
<tr>
<td>Pulse deficit (pulse differences in the four extremities)</td>
<td>15</td>
</tr>
<tr>
<td>Hypotension</td>
<td>5</td>
</tr>
</tbody>
</table>

**Diagnostic Testing**

Chest radiography alone is insufficient to exclude aortic dissection. However, normal findings on chest radiography significantly decrease the level of suspicion—as long as they are truly normal; only 12% of chest radiographs in patients who do have aortic dissection are retrospectively considered normal. In 78% of patients with aortic dissection, chest radiography demonstrates either a widened mediastinum or abnormal aortic contour. If possible, the EP should inform the radiologist that aortic dissection is under consideration to direct examination of the radiograph toward the pertinent abnormalities.

The following features are found on the chest radiographs of patients with aortic dissection:

- Wide mediastinum or abnormal aorta (in 78% of cases)
- Normal mediastinum and aorta (12.5%)
- Wide paraspinal shadow
- Pleural effusion
- Tracheal shift
- Calcification displacement
- “Lump” distal to vessels

ECG findings are neither sensitive nor specific for the diagnosis. In fact, as many as one in six patients with aortic dissection have evidence of ischemia or AMI on an ECG—presumably resulting from occlusion of the coronary vessels by an intimal flap or thrombosis—and 70% have normal or nonspecific findings.

Helical computed tomography and echocardiography provide definitive testing for aortic dissection. Either diagnostic test is 95% to 100% sensitive; echocardiography is preferred when the patient is unstable because it can be performed in the critical care setting. Transthoracic echocardiography is extremely sensitive in detecting abnormalities of the aortic root and ascending aorta, whereas the transesophageal approach is required to exclude involvement of the arch or descending aorta (Fig. 54.1).

**Treatment**

The goal of initial ED treatment of patients with aortic dissection is to decrease shearing stress on the aorta with negative inotropic and chronotropic agents, such as intravenous beta-blockers or calcium channel blockers. Further blood pressure control can be achieved with intravenous nitroprusside or nitroglycerin. Desired values are a heart rate of 50 to 60 beats/min and a systolic blood pressure of 100 to 110 mm Hg.

**Disposition**

Once aortic dissection is confirmed, ED medical management proceeds in parallel with emergency surgical evaluation, and if surgery is deemed appropriate, arrangements for intervention should not be delayed.

**Cocaine-Associated Chest Pain**

The U.S. Department of Health and Human Services reported in 2002 that 33 million people 12 years and older (14.4% of the U.S. population) reported using cocaine at least once in their lifetimes. Cocaine abuse is not limited to a specific subset of the population and is frequently seen in ED patients, as demonstrated by an urban ED report that 2% of the institution’s patients 60 years and older tested positive for cocaine.
PATHOPHYSIOLOGY

Chest pain, the most common complaint of ED patients with cocaine-associated visits, results from myocardial ischemia, trauma, pulmonary damage, and probably nonspecific vasospasm. Cocaine raises the risk for myocardial ischemia via multiple factors, including α-adrenergic receptor–mediated coronary vasoconstriction, platelet aggregation, direct myocardial toxicity, accelerated atherosclerosis, and increased myocardial oxygen demand. Therefore, acute coronary syndrome may be present in individuals who would otherwise be considered to have a very low risk for the disorder.

PRESENTING SIGNS AND SYMPTOMS

Inquiry should be made about recent cocaine use in all patients seen in the ED with chest pain. Patients who have used cocaine recently often have significant elevations in blood pressure in addition to their chest pain. They may be jittery and somnolent at the same time after having binged on crack cocaine. Studies have documented the incidence of AMI in patients with cocaine-associated chest pain to be approximately 6%. One study found that patients with cocaine-associated AMI were young (mean age, 38 years), tobacco smokers (91%), and nonwhite (72%) and had used cocaine within the proceeding 24 hours (88%). Nevertheless, a significant proportion of patients with cocaine-associated chest pain are older, and their risk for myocardial ischemia, though greatest in the first hours after the drug use, remains elevated for at least 2 weeks after discontinuation of the drug.

MEDICAL DECISION MAKING AND DIFFERENTIAL DIAGNOSIS

The chest pain or dyspnea associated with cocaine use may stem from a variety of causes. In addition to acute coronary syndrome, aortic dissection has been reported to be associated with cocaine use.14 The barotrauma induced by smoking crack cocaine results from deep inhalation followed by the Valsalva maneuver or severe coughing and leads to pneumothorax, pneumomediastinum, and pneumopericardium. Pulmonary diseases associated with smoking cocaine include noncardiogenic pulmonary edema, pneumonia, asthma, interstitial lung disease, bronchiolitis obliterans–organized pneumonia, parenchymal hemorrhage, and pulmonary vascular disease. Musculoskeletal trauma may also occur.

DIAGNOSTIC TESTING

The initial evaluation of a patient with chest pain is the same regardless of whether cocaine is involved. The EP should order laboratory testing of blood, an ECG, and chest radiography for similar indications. The ECG findings do not depend on whether the AMI is cocaine related. In both those with and those without cocaine-related AMI, ECG findings are normal in 1% to 10%, nondiagnostic but abnormal in 30% to 50%, and diagnostic in 50% to 60%. Nonspecific abnormalities and normal variations often found in young persons, such as J-point elevation and left ventricular hypertrophy, are common.

Testing for the myocardial markers troponin and CK-MB is the cornerstone of evaluation for AMI in patients with cocaine-associated chest pain. Troponins are the markers of choice because unlike CK-MB, they are not affected by recent cocaine use. The extent to which skeletal muscle breakdown from cocaine use affects the diagnostic accuracy of CK-MB measurement has not been established.

Stress testing in patients with cocaine-associated chest pain after appropriately timed myocardial marker testing to evaluate for AMI is considered safe. The utility of exercise ECGs, myocardial perfusion, or stress echocardiography may be limited, however, given the low rate of reversible coronary artery lesions in these patients (2% to 14%) and the significant false-positive rate.

TREATMENT AND DISPOSITION

Initial management (cardiopulmonary monitoring and aspirin) of patients with cocaine-associated chest pain is similar to that for patients with typical chest pain. In addition, the use of short-acting benzodiazepines, such as lorazepam, 1 mg intravenously repeated as necessary, in combination with nitroglycerin is recommended to counteract the sympathomimetic effects of cocaine. Hypertension usually responds to the preceding treatments. Additional blood pressure control is occasionally required because of suspicion for end-organ damage. β-Adrenergic blockade raises the theoretic concern of worsening hypertension as a result of vasospasm from unopposed α-adrenergic stimulation; however, little evidence supports this potential complication. Finally, for patients with evidence of cardiac ischemia or infarction, cardiac catheterization is beneficial and is preferred over thrombolytics, which should be used with caution.

There is some controversy regarding the disposition of patients with cocaine-associated chest pain. The EP should maintain a low threshold in evaluating for aortic dissection if the symptoms are severe and persistent. For patients in whom the findings are unremarkable—no ECG evidence of ischemia, no elevation in serial cardiac markers, and symptoms that resolve with treatment during observation—many authorities would argue that discharge is safe. Preliminary evidence has shown that this population is at low risk for subsequent complications. Until this issue is studied systematically, however, whether patients with cocaine-associated chest pain should be admitted for further evaluation is unclear.

REFERENCES

References can be found on Expert Consult @ www.expertconsult.com.
REFERENCES


