

Evaluation of the Elderly Patient with Acute Chest Pain

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In the United States, almost 6 million patients present to emergency departments each year with the chief complaint of chest pain [1–3]. Despite the frequency of this presentation, accurate diagnosis of the cause of chest pain remains challenging. In the majority of unselected patients, chest pain has a benign cause; however, the clinician must first consider potential life-threatening causes when evaluating patients with chest pain. Acute myocardial infarction (AMI), aortic dissection, pulmonary embolism, pneumothorax, esophageal rupture, and pericarditis with resultant cardiac tamponade may all present with chest pain. When these conditions are missed, significant patient morbidity and mortality can result. Misdiagnoses causing delayed treatment not only lead to poorer outcomes but also impose substantial emotional and financial stress. For example, missed AMI represents the greatest malpractice risk in emergency medicine, accounting for 10% of all lawsuits and almost 30% of monetary losses [3]. Claims for missed AMI occur with equal frequency to providers who care for elderly patients [4].

It is imperative that geriatricians be able rapidly to evaluate patients with acute chest pain and accurately exclude life-threatening causes. It is well known that serious causes of chest pain are more prevalent among older patients than in younger age groups. Unfortunately, many elderly patients who have a serious cause of chest pain present with atypical and nonspecific symptoms. To detect these life-threatening causes, the geriatrician must systematically evaluate every elderly patient with chest pain.

This article discusses the evaluation of the elderly patient with acute chest pain. Specific emphasis is placed on identifying acute coronary syndrome (ACS), pulmonary embolism (PE), acute aortic dissection (AD), and acute pericarditis with tamponade. By understanding the different presentations of these potentially life-threatening emergencies, the geriatrician will be

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better prepared to distinguish them from less dire conditions and initiate prompt treatment.

Pathophysiology

To develop an approach to chest pain, it is important first to review thoracic anatomy and the relationship between afferent innervation and symptom production. Chest pain arises from stimulation of visceral and somatic pain fibers. Visceral fibers originate from the heart, blood vessels, esophagus, and visceral pleura and enter the spinal cord at multiple levels. Stimulation of these fibers produces symptoms that are poorly localized and often difficult for patients to describe. Elderly patients are more likely to use terms such as *aching*, *pressure*, *discomfort*, or *heaviness* when describing pain of visceral origin. In addition, patients may report discomfort in other areas of the body, namely the neck, shoulders, back, and arms. In contrast, somatic pain fibers originate from musculoskeletal structures, parietal pleura, and dermis, producing symptoms that are well localized and typically easily described. Somatic fibers enter the spinal cord at specific levels and tend to produce symptoms that follow a dermatomal pattern.

Understanding these differences makes it easier for clinicians to interpret the descriptive terms patients use when reporting their symptoms. It is important to understand that patients' perception and description of pain are affected by a number of other factors, most notably age, level of education, comorbid medical or psychologic diseases, and cultural differences.

Initial evaluation

The key components of the initial evaluation of chest pain include an assessment of the patient's general appearance, a detailed history and focused physical examination, electrocardiogram, and chest radiograph. Additional components of the evaluation, such as laboratory studies, are obtained based on findings of the initial evaluation.

General appearance

Critically ill patients must be rapidly identified. Begin by noting the patient's general appearance. Patients who are pale, clammy, anxious, or diaphoretic need immediate attention. Obtain a complete set of vital signs: temperature, pulse, blood pressure, respiratory rate, and oxygen saturation. Following interpretation of vital signs, one should perform a rapid survey of the airway, breathing, and circulation (see later discussion). All unstable patients require an ECG and a chest radiograph as soon as possible. A more detailed history may be obtained from patients who are hemodynamically stable and in no acute distress.

History

Obtaining a detailed history is a crucial step in the formulation of an appropriate differential diagnosis in patients who have chest pain. A useful mnemonic for the key components of the history is “OLD CAAARS” (A. Mattu, MD, personal communication, 2003).

Onset

Determine the time of onset of the chest pain. Patients presenting immediately after the onset of chest pain generally view their condition as serious. Additionally, knowing when the pain began is helpful when one is interpreting specific test results such as cardiac biomarkers. Many patients presenting shortly after the onset of an AMI have normal cardiac biomarker levels initially. For this reason, it is important not to exclude ACS based on a single set of cardiac markers.

Location

Elicit the location of the pain. Lateralizing pain may be present with a PE or pneumothorax. Anterior chest pain can be associated with ascending AD, whereas interscapular back pain is typical of descending dissections. Although it is important, the location of pain should not be used by itself to exclude potential life-threatening diagnoses. For example, patients who have inferior myocardial infarction (MI) may complain of epigastric pain and report associated symptoms of nausea or emesis. These complaints can be misleading if the clinician does not consider ACS as a diagnosis.

Duration

Determine the duration of the pain. Neither fleeting pain lasting seconds nor constant pain that is unremitting for weeks is typical of myocardial ischemia. Unstable angina (UA) may present with intermittent, recurring pain over days to weeks; however, in AMI, pain typically lasts at least 5 to 15 minutes and will gradually worsen.

Character

A patient's description of the quality or character of the pain is an important historical element but must be used carefully. Classically, myocardial ischemia is usually described as a tightness, pressure, ache, or heavy weight pressing on the center of the chest. In practice, elderly patients, especially women and diabetics, often report less classic symptoms, such as shortness of breath, fatigue, indigestion, lightheadedness, dizziness, altered mental status, and palpitations. Sharp, tearing pain is classic of AD. Sharp pleuritic pain, caused by pleural irritation, is common in PE.

Aggravating factors

Information about factors that provoke or aggravate the pain is useful. Pain that is worsened by activity, emotional stress, sexual intercourse, or

exposure to cold suggests myocardial ischemia. Pleuritic pain, or pain worsened by deep breathing or position, suggests pneumothorax, pericarditis, or PE. Pain that is reliably and exactly reproduced with minor movements of the trunk or extremities is most likely musculoskeletal in nature.

Alleviating factors

Exertional pain that subsides with rest is classic of angina. Pain associated with pericarditis may diminish in an upright or leaning-forward position. Therapeutic trials of medications such as nitroglycerin or antacids should not be used to increase or decrease clinical suspicion that a serious disease exists. Studies have demonstrated that the relief of chest pain after nitroglycerin does not predict a cardiac source [5,6]. It is not uncommon for patients with chest pain to report relief after nitroglycerin, regardless of the cause. Shry and colleagues [6] highlighted this finding in a retrospective study of 251 emergency department patients with chest pain. Eighty-eight percent of patients who had cardiac chest pain and 92% of patients who had noncardiac chest pain reported some relief after receiving sublingual nitroglycerin. In addition, the symptoms of myocardial ischemia reportedly improve after administration of antacids [7]. Do not exclude ACS based on the relief of pain with antacids.

Associated symptoms

Any symptoms associated with the pain must be elicited. Patients who have myocardial ischemia may report nausea, vomiting, diaphoresis, dyspnea, or near-syncope. Elderly patients may report belching, indigestion, or even generalized fatigue when suffering MI. Dyspnea is the most common presenting symptom of angina in patients older than 85 years [8]. When chest pain is associated with syncope, the clinician must consider the possibility of a PE, AD with resultant cardiac tamponade, or ruptured aortic aneurysm. Chest pain associated with neurologic symptoms is AD until proved otherwise.

Radiation

Visceral pain often radiates or is referred to other locations. For example, the pain associated with myocardial ischemia classically radiates to the neck, shoulder, arm, or jaw. A wide extension of chest pain radiation or radiation of pain to both arms increases the probability of MI. In an excellent meta-analysis, Panju and colleagues [9] reported likelihood ratios for ACS based on clinical features (Table 1). The pain experienced with AD often radiates to the back and may migrate to the abdomen or legs as the dissection plane advances to the distal abdominal aorta.

Severity

Severity of pain is subjective. Regardless, temporal severity is useful information. Pain that is abrupt in onset and immediately becomes maximally

Table 1
Clinical features that increase the likelihood of acute myocardial infarction

Clinical feature	Likelihood ratio (95% confidence interval)
Pain in chest or left arm	2.7 ^a
Chest pain radiation	
Right shoulder	2.9 (1.4–6.0)
Left arm	2.8 (1.7–3.1)
Both left and right arms	7.1 (3.6–14.2)
History of myocardial infarction	1.5–3.0 ^b
Association with nausea or vomiting	1.9 (1.7–2.3)
Association with diaphoresis	2.0 (1.9–2.2)
Hypotension (systolic blood pressure \leq 80 mm Hg)	3.1 (1.8–5.2)

^a Data not available to calculate confidence interval.

^b In heterogeneous studies the likelihood ranges are reported as ranges.

Adapted from Panju AA, Hemmelgarn BR, Guyatte GH, et al. Is this patient having a myocardial infarction? JAMA 1998;280(14):1256; with permission.

severe suggests AD, spontaneous pneumothorax, or PE. Pain associated with myocardial ischemia classically begins more gradually, with intensity increasing over a 5- to 10-minute period.

Medical history

One should question patients specifically about traditional risk factors for PE, AD, and ACS (Boxes 1–3). However, the utility of traditional risk factor assessment for ACS has been debated. Because traditional risk factors are derived from population studies, risk factor assessment has minimal utility when one is attempting to predict the likelihood of ACS in a specific patient who has acute chest pain [1,3]. The ACS risk factors that appear to be most important include age, gender, and a family history of premature coronary artery disease (CAD) [10]. Both the prevalence and severity of CAD increase with age, as do mortality and complication rates.

The utility of AD risk factor assessment has been less debated. Among patients who have AD, there is a 3:1 male predominance. Peak incidence occurs between 50 and 70 years of age. A history of hypertension is common (70%) [1]. Abuse of sympathomimetic drugs such as methamphetamine and cocaine increases the risk for AD.

The list of risk factors associated with PE is extensive. Hospitalization for surgery, especially when secondary to trauma, or medical illness accounts for 46% of patients with pulmonary emboli [11]. Other factors frequently encountered in patients who have PE include malignant neoplasm (18%), nursing home residence (13%), congestive heart failure (10%), and central venous catheter or pacemaker placement (9%).

Box 1. Risk factors associated with pulmonary embolism*Inherited thrombophilia*

Elevated individual clotting factors (VIII, IX, XI)
Factor V Leiden mutation
Hyperhomocystinemia
Protein C, protein S, or antithrombin III deficiency
Prothrombin gene mutation

Acquired disorders

Age
Antiphospholipid antibody syndrome
History of PE or deep venous thrombosis
Hyperviscosity syndrome
 Multiple myeloma
 Waldenstrom's macroglobulinemia
Immobilization (bedridden condition, paralysis, or paresis)
Malignancy
Medical conditions:
 Congestive heart failure
 Obesity
 Nephrotic syndrome
 Tobacco
 Inflammatory bowel disease
Central venous catheter/pacemaker placement
Hormone therapy/oral contraceptives
Pregnancy
Surgery
Trauma
Long-distance travel

Approximately one quarter of patients diagnosed with PE lack known risk factors [11].

Physical examination

Although emphasis is placed on the cardiovascular system, one should perform a thorough physical examination in all patients who have chest pain. In unstable patients, rapid assessment of the airway, breathing, and circulation should precede history acquisition.

Airway

The airway may be assessed by asking simple questions and inspecting the oropharyngeal cavity for potential obstructions. An appropriate verbal response to questioning ensures a patent airway.

Box 2. Risk factors associated with aortic dissection*Inherited disease*

Marfan syndrome
Ehlers-Danlos syndrome
Annuloaortic ectasia
Polycystic kidney disease
Turner syndrome
Bicuspid aortic valve
Coarctation of the aorta

Acquired condition

Pregnancy
Syphilis
Cardiac catheterization
Cocaine use
Hypertension
Aortic valve replacement
Trauma

Breathing

The patient's breathing is assessed primarily through a combination of observation and auscultation. Observe the patient's respiratory effort, noting the rate and depth of respirations. Increased respiratory effort should immediately alert the physician to a serious disorder. Inspect the chest wall to ensure equal chest rise. Auscultation of equal bilateral breath sounds is useful to exclude rare, but life-threatening, causes of acute chest pain such as spontaneous tension pneumothorax.

Circulation

Rapid assessment of the circulatory system includes measurement of the patient's blood pressure and heart rate, inspection of skin color and temperature (eg, warm and pink versus cool and mottled), assessment of capillary refill, and palpation of peripheral pulses. Obtain intravenous access as early as possible. Any abnormalities found during this assessment must be addressed before one proceeds with other components of the evaluation.

Look for any findings that may be attributable to cardiopulmonary disease. Jugular venous distention, if present, may signify decompensated heart failure, right heart strain secondary to a large pulmonary embolus, cardiac tamponade, or tension pneumothorax. Pulses in the upper and lower extremities should be carefully assessed. A difference in pulse is seen in as many as 60% of patients who have AD [12]. Von Kodolitsch and colleagues [13] demonstrated that a blood pressure difference of 20 mm Hg or greater

Box 3. Risk factors associated with acute coronary syndrome*Major risk factors that cannot be changed*

Increasing age

Male gender

Family history of premature CAD or cerebrovascular accident
(male relative <55 years or female relative <65 years)

Race

Modifiable major risk factors

Cigarette smoking

Diabetes mellitus

Hypercholesterolemia

Low high-density lipoprotein

Hypertension

Obesity

Physical inactivity

Drug abuse (cocaine and methamphetamines)

Other factors

Hyperhomocysteinemia

Hyperfibrinogenemia

Elevated lipoprotein (a)

Elevated C-reactive protein

Systemic lupus erythematosus

HIV

Stress

between the arms was an independent predictor of AD. However, inter-arm blood pressure differences are highly variable, with as many as 19% of normal individuals having differences greater than 20 mm Hg [14,15]. Auscultate the lungs to detect any asymmetric findings of diminished breath sounds, wheezing, rhonchi, or rales. Auscultation of the heart may reveal the presence of a murmur, gallop, or rub. A new high-pitched diastolic murmur and acute chest pain should raise suspicion for AD. Hepatojugular reflux and bilateral dependent pitting edema may be additional signs of congestive heart failure. Unilateral lower-extremity edema or tenderness should raise suspicion for deep venous thrombosis.

Electrocardiogram

The ECG is often reported to be the single most important initial diagnostic test in the evaluation of patients who have chest pain and suspected ACS [1,3]. Medical negligence often results when clinicians fail to obtain an

ECG or misinterpret the result as normal or nonspecific changes. Despite its importance, the initial ECG is diagnostic in less than 50% of patients who have ACS [1–3]. Only a small percentage of patients who have AMI present with a normal ECG (ie, no ST segment or T wave changes, strain, ischemia, old infarcts, or pseudonormalization), whereas a normal ECG is more common in patients who have unstable angina. Techniques that may be used to increase the sensitivity of the standard 12-lead ECG when nondiagnostic findings are present include the addition of right-sided or posterior leads, serial ECGs, and continuous ST segment monitoring.

Elderly patients with ACS are more likely to have nonspecific findings on ECG than are younger patients. They are less likely than younger patients to present with ST segment elevation (31.4% versus 50.1%) and more likely to present with left bundle branch block (8.0% versus 0.6%) [16]. Elderly patients are also more likely to have paced rhythms and evidence of previous MI, both of which lead to more frequent nondiagnostic ECGs in the presence of AMI [17]. Comparison with a previous ECG is crucial. All elderly patients with new changes on the ECG require further evaluation to exclude ischemia.

ECG changes are common in patients who have PE; however, these changes are neither sensitive nor specific for the diagnosis. Classic ECG changes found in patients with PE include new-onset incomplete or complete right bundle branch block, atrial fibrillation, T-wave inversion across the anterior precordial leads, and the S1Q3T3 pattern (ie, an S wave in lead I, a Q wave in lead III, and an inverted T wave in lead III). These changes occur in a minority of patients. More common ECG findings in patients who have PE include sinus tachycardia and findings that reflect pre-existing cardiopulmonary disease.

Chest radiograph

The chest radiograph (CXR) is a cost-effective diagnostic test used to evaluate patients who have chest pain. It has been reported that CXRs influence management in 14% to 23% of emergency department patients with chest pain [1]. In cases of pneumothorax or pneumomediastinum, the CXR may be diagnostic.

Radiographs can be helpful in identifying AD. The most notable radiographic findings in patients who have AD are mediastinal widening greater than 8 cm and the presence of an abnormal aortic contour (Fig. 1). These findings are present in 61.6% and 49.6% of patients who have AD, respectively [18]. Other abnormalities that suggest AD include an abnormal aortic knob, left apical cap, tracheal deviation, depression of the left main stem bronchus, esophageal deviation, and loss of the paratracheal stripe. However, the CXR can be completely normal in as many as 12.4% of patients who have AD [18].

Classic radiographic findings in patients who have PE include Hampton's hump (a wedge-shaped, pleural-based density with the apex pointing toward

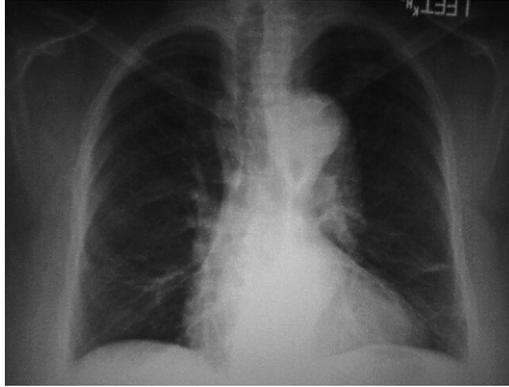


Fig. 1. Anterior–posterior chest radiograph demonstrating a widened mediastinum in a 67-year-old woman with chest pain and acute aortic dissection.

the hilum) and Westermark’s sign (a prominent pulmonary hilum caused by pulmonary vessel dilatation, with relative peripheral oligemia resulting from collapse of vessels distal to the embolus). However, these late findings are neither sensitive nor specific for the diagnosis of PE. More commonly, the CXR demonstrates progressive atelectasis and volume loss of the affected pulmonary segment. Data from the International Cooperative Pulmonary Embolism Registry suggest that 82% of patients older than 70 who have PE exhibit abnormal chest films. Radiographic abnormalities that occur with increased frequency in older patients who have PE include cardiomegaly and pulmonary venous congestion [19].

Radiographic findings in patients who have cardiac tamponade depend on the rate of pericardial fluid accumulation and the volume of the effusion. Because of the relative indistensibility of the pericardium, rapidly accumulated effusions can lead to cardiac tamponade even at small volumes. In this case, the CXR may appear normal. Pericardial effusions that occur more slowly allow the pericardium to distend gradually, producing an apparently “globular” enlarged cardiac silhouette on the CXR. As much as 300 mL of pericardial fluid may be required before significant cardiac enlargement is appreciated [20].

Differential diagnosis

It is imperative to generate initial broad differential diagnoses in elderly patients who have chest pain. This task is accomplished by assimilating all available historical and clinical information into a list of likely disorders. Priority must be given to potentially serious disorders. **Box 4** lists serious and benign causes of acute chest pain. Consider benign causes once life-threatening ones have been excluded.

Box 4. Differential diagnosis of chest pain*Life-threatening causes*

Acute myocardial infarction
Unstable angina
Pulmonary embolism
Aortic dissection
Myocarditis
Tension pneumothorax
Acute chest syndrome (sickle cell disease)
Esophageal rupture

Non-life-threatening causes

Gastroesophageal reflux
Peptic ulcer disease
Pneumonia
Pleurisy
Costochondritis
Herpes zoster
Musculoskeletal pain
Anxiety disorders
Mitral valve prolapse

When formulating the differential diagnosis in patients who have chest pain, it may be helpful to use an anatomic “inside-out” thought process. This approach forces the physician to focus on the cardiopulmonary system—the system responsible for the majority of serious disorders that produce pain in the chest. It also reduces the chance that a serious disorder will be overlooked.

Specific disease entities

The remainder of this article is dedicated to the diagnostic evaluation of specific disease entities that have been selected for further discussion because of their life-threatening nature. Prompt recognition and treatment of these conditions is crucial to minimize patient morbidity and mortality.

Acute coronary syndrome

CAD remains the leading cause of death in the United States. ACS, including unstable angina and AMI, are important disease entities that must be considered in all elderly patients with chest pain. Despite heightened

awareness of ACS, it is estimated that between 0.4% and 10% of patients who have AMI are incorrectly discharged from the emergency department [1,3,21]. The reasons clinicians fail to recognize serious illness are numerous; however, even experienced, competent physicians can miss AMIs. The reason is often the clinician's failure to consider the possibility of ACS and therefore failure to initiate the appropriate diagnostic work-up. In the short term, patients sent home from the emergency department with ACS have a mortality nearly twice that of ACS patients who are admitted to the hospital [3].

Investigations involving patients who have AMI indicate specific subgroups that are at greater risk for misdiagnosis. These groups include the very young and very old, women, and diabetics [22]. In the case of the very young patient, the diagnosis is often not considered because of the very low prevalence of CAD in this group. The remaining three groups of patients have one thing in common—the propensity to present with atypical symptoms. Women presenting with ACS tend to be older than men who have ACS, typically postmenopausal; they are more likely to have comorbid diseases, such as diabetes or hypertension, and to have a family history of premature coronary heart disease [23]. Women who have AMI are also more likely to present with neck and shoulder pain, nausea, fatigue, and dyspnea than are men [23]. Diabetic patients are more likely to experience silent ACS than are their age-matched nondiabetic counterparts. Therefore, late presentations are common, a pattern that partially explains the increase in morbidity and mortality among diabetic patients who have AMI. Diabetic patients are also more likely to present with exertional dyspnea, severe fatigue, or lightheadedness [24].

In elderly patients, chest pain accompanies AMI much less frequently. In patients aged 85 years or older, dyspnea, not chest pain, is the single most common presenting symptom of angina [10]. Additionally, elderly patients present more frequently with complaints of fatigue, lightheadedness, worsening congestive heart failure, altered mental status, and syncope [2]. Elderly patients are also more likely to have comorbid diseases such as diabetes, hypertension, and coronary heart disease. At autopsy, approximately 50% of elderly women and 70% to 80% of elderly men have demonstrable obstructive CAD [25]. Elderly patients tend to have more severe and widespread CAD than younger patients [25–27].

Nonetheless, elderly patients who have ACS tend to receive less aggressive medical therapy and fewer revascularization procedures than do younger patients. One reason may be that elderly patients tend to wait before seeking medical attention. In a study by Tresch and colleagues [27], elderly patients with ischemic chest pain delayed seeking medical attention for more than 6 hours after the onset of pain, even though more than 50% of them had a documented history of CAD.

Finally, elderly patients who have AMI fare far worse than younger patients. Although octogenarians constitute less than 5% of the United

States population, they account for 30% of all MI-related hospital deaths [25].

Diagnostic evaluation

The cornerstone of the diagnosis of ACS has long been the ECG. Ideally, all patients who have suspected ACS should undergo an ECG within 10 minutes of arrival. Accurate interpretation of the ECG and prompt consultation with a cardiologist, when appropriate, are necessary to avoid delays in management. A CXR should be obtained before the administration of anticoagulants to minimize the chances of inadvertent administration of these medications to a patient who has AD.

Because elderly patients are more likely to present with non-ST-elevation MI (NSTEMI) and nondiagnostic ECGs, the diagnosis of AMI often relies heavily on measuring serum cardiac biomarkers. Conventional biomarkers, including creatine kinase MB isoenzyme, troponin I, and troponin T, are indicators of myocardial necrosis. These markers are released in the bloodstream following MI. The levels of these serum markers tend to rise 3 to 4 hours after the onset of an AMI. Serial sampling over a 12- to 24-hour period will detect the majority of AMIs. Although serial troponin sampling is the current gold standard for the diagnosis of NSTEMI, this test is not 100% sensitive or specific. Elevated troponin levels are seen in other disease states, including renal failure, subarachnoid hemorrhage, and severe sepsis. In addition, serial troponin sampling is only approximately 30% sensitive for the detection of UA [28]. Further evaluation with provocative stress testing must be performed when UA is a possibility.

Aortic dissection

AD is one of the most common and lethal diseases of the aorta. It is estimated that unrecognized AD carries a 1% to 2% mortality per hour for the first 48 hours [2]. If the diagnosis remains unrecognized, the mortality reaches 90% at 1 year [29]. When the disease is promptly diagnosed and treated, the 30-day survival rate is 80% to 90%, and the 10-year survival rate is 55% [29].

Despite advances in imaging techniques, diagnosing AD remains difficult. Because of the variety of symptoms attributable to AD, a high clinical index of suspicion is required. It is estimated that physicians correctly suspect the diagnosis in as few as 15% to 43% of patients at the time of presentation [2]. Diagnostic delays are common. Not infrequently, the correct diagnosis is made during advanced imaging procedures intended to assess other diagnoses [29]. Even worse, when AD is misdiagnosed as ACS, the consequences of giving anticoagulants or thrombolytics can be disastrous.

AD is largely a disease of old age. Younger patients who have Marfan's syndrome typically account for only about 5% of cases of AD [18]. The classic patient with AD is a man in the seventh decade of life. Seventy-two

percent of patients have a history of hypertension [18]. Five percent have a history of aortic valve repair or replacement [18]. Ninety-five percent of patients who have AD report some pain. Most often, the pain is abrupt in onset (84.8%) and located in the anterior chest (60.9%), posterior chest (35.9%), or back (53.2%) [18]. The pain is frequently described as severe or “worst ever” (90.6%) or sharp (64.4%). Although they are highly specific for AD, the descriptive terms *tearing* and *ripping* are only used by 50% of patients [18,29]. The pain is migratory in 16.6% of patients [18]. AD is associated with syncope in as many as 13% of patients [18,30], and this may be the only presenting symptom in as many as 3% of patients [30]. Therefore, AD should be included in the differential diagnosis of elderly patients who have syncope, even when chest symptoms are lacking.

On physical examination, approximately 50% of patients who have AD are hypertensive (systolic blood pressure [SBP] ≥ 150 mm Hg), whereas 16% are hypotensive (SBP ≤ 100 mm Hg) or in shock (SBP ≤ 80 mm Hg) [18]. The blowing, decrescendo, diastolic murmur of aortic insufficiency is present in less than one third of patients, and its absence should not be used to exclude the diagnosis [18,29]. Likewise, the presence of electrocardiographic changes suggestive of AMI does not exclude AD. In an excellent comprehensive literature review, Klompas [29] found that 7% of patients with acute AD had electrocardiographic evidence suggestive of AMI. Focal neurologic deficits suggestive of a cerebrovascular accident are found in 4.7% to 17% of patients [18,29]. Evidence of congestive heart failure is found in 6.6% of patients [18].

It is critical to obtain a detailed history from patients who have chest pain and suspected AD. Patients must be asked to describe the quality of the pain, its intensity at onset, and whether the pain radiates. A retrospective chart review of patients who had confirmed thoracic AD revealed that only 42% of conscious patients were asked these three simple questions [31]. Clinicians must also screen for known risk factors, including hypertension, aortic valve replacement, and recent cardiac catheterization. It is estimated that iatrogenic AD complicates approximately 2% of cardiac catheterizations [18].

Diagnostic evaluation

The initial evaluation of a patient who has suspected AD begins with prompt chest radiography. Most patients who have AD have abnormal findings on CXR. A completely normal CXR reduces the probability of AD (negative likelihood ratio 0.3) [18]. However, given that chest radiographs are normal in 12.4% of patients who have AD, CXR alone is insufficient to exclude AD.

The diagnosis of AD necessitates the use of advanced imaging modalities, most frequently CT (Fig. 2). With the advent of multidetector row CT (MDCT) scanners, aortic disorders are diagnosed with increased accuracy. In a recent retrospective review of 373 cases of suspected AD in the emergency setting, Hayter and colleagues [32] found the sensitivity of MDCT

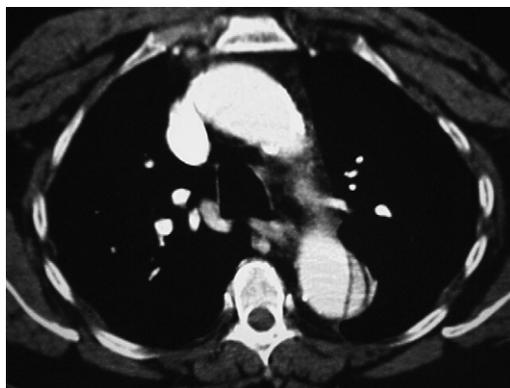


Fig. 2. Multidetector chest CT of the patient shown in Fig. 1, demonstrating a Stanford Type B (descending) thoracic aortic dissection.

for detecting AD to be 99%. Other advanced imaging modalities that are used less frequently include transthoracic and transesophageal echocardiography, MRI, and aortography.

To date, laboratory testing has played a limited role in the evaluation of AD. Several studies demonstrated that highly sensitive quantitative D-dimer assays have a sensitivity of 100% for the diagnosis of acute AD [33–35]. However, given this test's poor specificity and the lack of a large prospective trial, it remains to be seen whether it will play a significant role in the evaluation of patients who have suspected AD. Other laboratory tests for AD that are under investigation include smooth muscle myosin heavy chains and soluble elastin fragments [36,37]. These tests, while highly sensitive and specific for AD, are not readily available and lack current clinical utility.

Pulmonary embolism

PE is a challenging disease, particularly in elderly patients. It has been estimated that the diagnosis of PE is missed 400,000 times annually, leading to 100,000 preventable deaths [38]. The mortality of patients who have unrecognized PE is nearly seven times greater than that of appropriately treated patients [39]. Presenting signs and symptoms of PE vary greatly, and the coexistence of obstructive lung disease, pneumonia, or congestive heart failure can make diagnosis more difficult.

The incidence of PE rises with age. It is not clear whether this risk should be attributed to advanced age itself or simply to an increased incidence of conditions associated with venous thromboembolic disease. What is clear is that elderly patients who have PE suffer increased morbidity and mortality compared with younger patients [39,40]. Additionally, complications associated with anticoagulation (eg, life-threatening bleeding) may increase with age.

Most patients who have PE present with complaints of dyspnea or acute chest pain [41]. The pain is typically sharp and pleuritic. Patients may report painless dyspnea occurring at rest or on exertion. Decreased exercise tolerance accompanied by nonspecific T-wave changes may be noted in some patients who have PE, prompting an evaluation for CAD. Less frequently, patients with PE report persistent cough, hemoptysis, presyncope, or syncope. Clinicians must be aware of the significance of leg symptoms (unilateral pain or swelling) and dyspnea or chest pain. Clinical signs associated with PE such as tachypnea and tachycardia are common and appear to occur with equal frequency in elderly and younger patients [42]. However, elderly patients may be more likely to ignore new symptoms or to attribute them to existent cardiopulmonary disease.

Diagnostic evaluation

The diagnostic evaluation of patients who have suspected PE presents a considerable challenge to physicians. Most often, the evaluation begins with some assessment of clinical pretest probability. Few other diseases exist in which pretest probability has been so extensively investigated. In the time since the Prospective Investigation of Pulmonary Embolism Diagnosis study compared clinical estimates of disease prevalence with actual disease rates, considerable effort has been invested in combining clinical pretest probability estimates with other forms of testing to diagnose or exclude PE. In current practice, the evaluation of patients who have suspected PE most commonly incorporates a clinical scoring system in combination with D-dimer testing and some form of advanced imaging (MDCT, compression venous duplex, or ventilation-perfusion scans) [43–45]. Fig. 3 is a CT scan that demonstrates a large intraluminal filling defect within the pulmonary artery representative of PE.

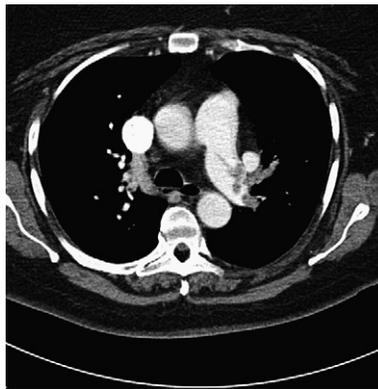


Fig. 3. Multidetector chest CT demonstrating an intraluminal filling defect within the pulmonary artery, characteristic of a pulmonary embolism. (Courtesy of Henry Kim, MD, University of Maryland School of Medicine.)

Although these clinical prediction models have been validated prospectively, the utility of D-dimer testing in elderly patients in whom PE is suspected is controversial. The main utility of D-dimer testing in patients with PE is its high sensitivity and negative predictive value (NPV). In patients who have low clinical suspicion for PE, a low D-dimer level (<500 ng/mL) makes the probability of PE so small that it is safe to withhold further testing or anticoagulation [46]. In a prospective cohort study, Tardy and colleagues [47] evaluated the use of an ELISA D-dimer in 96 consecutive outpatients older than 70 years who had suspected PE. In this geriatric population, conventional ELISA D-dimer testing had sensitivity and NPV of 100%; however, owing to advanced age and comorbid conditions, only a few of the 96 study patients presented with D-dimer levels less than 500 ng/mL. It is not difficult to imagine that the utility of D-dimer testing in geriatric inpatients with suspected PE would be even lower.

Because of this finding, additional imaging is frequently required in the work-up of elderly patients who have suspected PE. When evaluating elderly patients for PE, one must make the decision to use lower-extremity duplex ultrasound, ventilation-perfusion scanning, spiral CT, or angiography on an individual basis. Nevertheless, when PE is a possibility, definitive diagnosis or exclusion is required to minimize patient harm.

Acute pericarditis and cardiac tamponade

Acute pericarditis is the most common disorder of the pericardium. Like other inflammatory processes, acute pericarditis results from (1) local vasodilatation, (2) increased vascular permeability, and (3) leukocyte exudation [48]. The causes of acute pericarditis are numerous (Box 5). Viral infection is probably the most common cause of acute pericarditis in young, otherwise

Box 5. Causes of acute pericarditis

Idiopathic

Infectious

Viral

Tuberculous

Pyogenic bacteria

Noninfectious

Postmyocardial infarction

Uremia

Neoplastic disease

Radiation-induced

Connective tissue diseases

Drug-induced

healthy individuals. Uremic pericarditis may be seen in as many as one third of patients who have end-stage renal disease [49]. Neoplastic pericarditis, documented in as many as 10% of patients who have known malignancy at autopsy, may be responsible for as many as 35% of cases of acute pericarditis [50]. Malignancies commonly associated with pericardial involvement include breast cancer, lung cancer, leukemia, lymphoma, and mesothelioma.

Common symptoms of acute pericarditis include chest pain, fever, malaise, and myalgias. The chest pain is typically substernal with radiation to one or both trapezius muscle ridges. The pain is often described as positional and may be relieved by sitting up or bending forward. Exacerbation may occur with breathing, coughing, movement, or supine position. Dyspnea is common, particularly when a large pericardial effusion or cardiac tamponade is present. Fever is common in younger individuals but may be absent in elderly patients. Other common signs include tachypnea and tachycardia. Hypotension and cardiovascular shock may occur in patients who have pericarditis and cardiac tamponade.

Cardiac tamponade is an acute life-threatening emergency. It results from increased pressure within the pericardial space secondary to a high-pressure pericardial effusion. The increased pericardial pressure compresses the cardiac chambers, impairs diastolic filling, and reduces stroke volume and cardiac output. As blood return to the heart is impaired, the systemic and pulmonary venous pressures become elevated, leading to signs of right-heart failure (jugular venous distension, hepatomegaly, peripheral edema). Untreated, the resultant decline in cardiac output leads to hemodynamic compromise, hypotensive shock, and death.

Tamponade can result from any process that causes fluid to accumulate in the pericardial space. In the United States, the most common cause of cardiac tamponade is malignant pericardial effusion. Other common causes include trauma, catheter-based procedures, radiation exposure, postviral conditions, and uremic pericarditis.

Diagnostic evaluation

The ECG is abnormal in as many as 90% of patients who have acute pericarditis. Electrocardiographic changes associated with pericarditis reflect inflammation of the epicardium. In patients who have acute pericarditis, the ECG classically evolves through four characteristic stages:

Stage 1: Accompanies the onset of chest pain and is characterized by diffuse concave-upward ST-segment elevation with T-wave concordance, ST-segment depression in aVR and V1, and PR-segment depression (Fig. 4).

Stage 2: Occurs several days to weeks later and is characterized by ST-segment and PR-segment normalization and T-wave flattening.

Stage 3: Characterized by diffuse T-wave inversions (not always present).

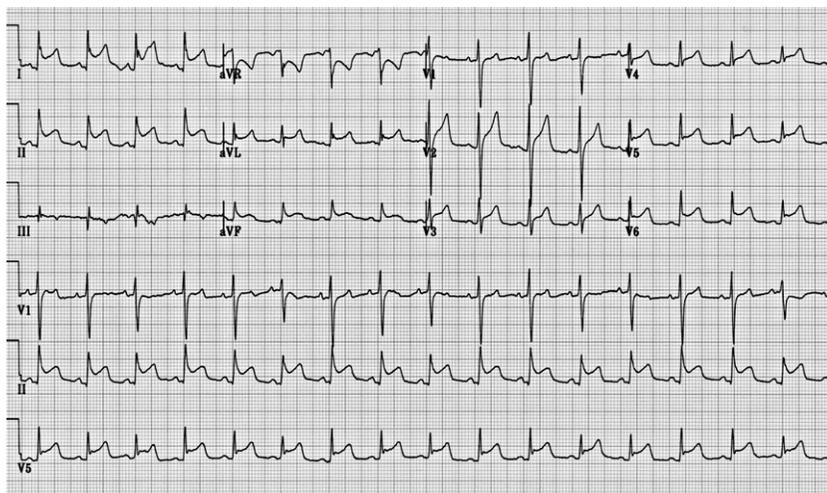


Fig. 4. Twelve-lead ECG in a patient with acute pericarditis. Note the diffuse concave upward ST-segment elevation. ST-segment depression is limited to lead aVR. Slight PR-segment depression is best appreciated in lead II. (Courtesy of Amal Mattu, MD, University of Maryland School of Medicine.)

Stage 4: Characterized by complete electrocardiographic normalization or persistence of T-wave inversions.

Other electrocardiographic findings that may be seen in patients who have pericarditis and large pericardial effusions include low-voltage QRS complexes and electrical alternans.

It is important to differentiate the electrocardiographic changes in patients who have acute pericarditis from those associated with more serious causes of ST-segment elevation. Two conditions commonly confused with pericarditis are benign early repolarization and AMI. As a rule, benign early repolarization is an electrocardiographic finding present only in younger individuals. The temptation to ignore ST-segment changes by labeling them as “early repolarization” can be disastrous in the elderly.

The most readily identifiable ECG feature distinguishing acute pericarditis from AMI is the lack of reciprocal ST-segment depression. With the exception of leads aVR and V1, ST-segment depression should not occur in patients who have acute pericarditis. The presence of ST-segment depression in leads other than aVR and V1 in association with ST-segment elevation suggests the presence of an AMI. Other electrocardiographic findings that strongly suggest AMI instead of acute pericarditis are convex ST segments, ST-segment elevation in specific areas that correlate with coronary artery anatomy, and the loss of R-wave voltage.

Cardiac tamponade must be suspected in any patient with known pericarditis who develops signs and symptoms consistent with systemic vascular

congestion and hemodynamic compromise. Vital sign derangements, including tachypnea, tachycardia, and hypotension, are common. On examination, one may observe Beck's triad, a complex of physical findings consisting of jugular venous distension, systemic hypotension, and diminished heart sounds. Jugular venous distension, the most prominent physical finding, may be absent in patients with severe volume depletion. Pulmonary rales resulting from pulmonary venous congestion may be appreciated. Other classically described physical findings that are not specific for cardiac tamponade include pulsus paradoxus (decrease in SBP ≥ 10 mm Hg with inspiration) and Kussmaul's sign (the paradoxical rise in jugular venous pressure with inspiration).

Patients who have cardiac tamponade require prompt diagnosis and definitive treatment. Mortality for cardiac tamponade directly correlates with the time to definitive treatment. Although cardiac tamponade is a clinical diagnosis, the most useful noninvasive diagnostic test remains echocardiography. Echocardiography can successfully differentiate cardiac tamponade from other conditions associated with low cardiac output (eg, systolic dysfunction). Echocardiographic features suggestive of cardiac tamponade include pericardial effusion, early diastolic right ventricular collapse, late diastolic compression or collapse of the right atrium, and impaired right-sided blood flow during inspiration. Diagnostic confirmation, based on intracardiac and intrapericardial pressure measurement, and treatment (therapeutic pericardiocentesis) often occur in the cardiac catheterization lab.

Summary

Chest pain is one of the most common and potentially life-threatening complaints in the geriatric population. The incidence and mortality of ACS, AD, and PE increase with advanced age. Malignancy, the most common cause of cardiac tamponade in the United States, is also common among older individuals. Elderly patients who have chest pain are more likely to present with atypical symptoms and nonspecific physical findings, putting them at increased risk for misdiagnosis. To ensure the best possible patient outcome, physicians must conduct a thorough interview, including assessment of known risk factors; perform a detailed physical examination; and maintain a high level of suspicion when evaluating elderly patients with chest pain.

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