MACEP Risk Management Course
Module 2: Chest Pain

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Course Objectives

- State the major life threatening diagnoses that are the most common triggers for liability risk in non-traumatic chest pain as chief complaint.
- Indicate the key components of documentation in the evaluation, treatment and disposition of a patient with chest pain as chief complaint.
- Identify specific risk factors that are associated with acute coronary syndrome (ACS), thoracic aortic dissection (TAD) and pulmonary embolism (PE).
- Describe specific systems of care that can potentially minimize chest pain liability risk.
- State other diagnoses that are associated with chest pain liability risk

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Introduction

Evaluation of non-traumatic chest pain remains one of the highest-risk chief complaints in emergency medicine. Based upon most recent data from a review of malpractice claims from 2005-2009, cardiovascular diagnoses accounted for 26% (58/224) of closed claims and 29% (72/247) of open claims as of 10/2010. Prior reviews[1,2] of malpractice claims and prospective research studies have consistently indentified specific chest pain diagnoses that have been the most problematic. The most prevalent diagnoses that are associated with adverse outcomes are **1) acute coronary syndrome (ACS, missed myocardial infarction (MI)) 2) thoracic aortic dissection (TAD); and 3) pulmonary embolism (PE)**. It is also important to note that myocarditis, bacterial endocarditis and other cardiovascular diagnoses are a source of risk management concerns. These are briefly covered in a category for "other" liability concerns. The average payout for chest pain cases closed with payment is approximately $600,000 (average for missed ACS: $560,000; missed dissection: $700,000).

This module presents a summary of the most prevalent triggers of liability and the most important aspects of the clinical evaluation, diagnostic testing, treatment and disposition that are associated with adverse outcomes. A consistent theme remains the failure of clinicians to

diagnose a high risk condition, obtain or interpret routine diagnostic tests or to appreciate the limitations of diagnostic tests such as cardiac biomarkers or plain chest x-ray as screening tests. Each section will briefly review the target condition, key elements of the history and examination that are associated with making or failing to make the diagnosis, the role of diagnostic testing, and specific aspects of disposition and system design from a risk management perspective.

An extensive review of the various diagnostic modalities potentially available or in depth review of treatment considerations is beyond the scope of the module. The availability of diagnostic testing and treatment options is highly dependent on the practice setting. Each section will provide references for further reading about technologies to aide in the diagnosis of ACS or recommendation for the treatment of ACS, PE or TAD based upon consensus guidelines or evidence-based reviews.

Acute Coronary Syndrome

Coronary heart disease remains the most common cause of mortality in the United States. According to the American Heart Association there are an estimated 935,000 cases of myocardial infarction per year in the US and an additional 86,000 cases of unstable angina. About 20% of those are fatal.[3] Our goal in the emergency department (ED) is to identify and appropriately treat patients presenting with acute coronary syndrome (ACS). ACS encompasses both myocardial infarction (including ST-segment elevation MI and non ST-segment elevation MI) and unstable angina. While patients with unstable angina have better outcomes than patients with non-ST segment MI, these syndromes are clinically indistinguishable at presentation.[4]

Determining which patients have ACS can be difficult, resulting in a miss rate of 2-4 %.[5,6] Chest pain is the second most common chief complaint in the emergency department,[7] responsible for an estimated 5.5 million visits to emergency departments in 2008.[8] A minority of patients presenting with chest pain are diagnosed with ACS.[8] A significant percentage of patients with ACS do not have chest pain as their chief complaint.[9,10] Patients with atypical symptoms (i.e. those without chest pain) are at higher risk of misdiagnosis, are less likely to receive standard of care medical treatment, and have a higher mortality.[9,10] Regardless of the presenting complaint, missed ACS results in a much higher mortality and morbidity rate,[1,2,6,11-13] and is a common source of liability for the emergency physician.[1,2,6]

Claims relating to myocardial infarction (MI) accounted for **7.6%** of the emergency medicine closed claims in 2005-2009, and resulted in **23% of the indemnity paid**. **Failure to diagnose** is particularly high risk, with **61.5%** of claims for failure to diagnose MI being *closed with indemnity paid*, compared to **32% of overall claims** against emergency physicians closed with indemnity paid. This is consistent with prior malpractice claims studies showing that *failure to diagnose* makes up the majority of claims for all diagnoses.[1,13,14] Errors in diagnosis most commonly result from: 1) **failure to order a diagnostic test**; 2) **inadequate history and physical**; 3) **incorrect interpretation of test results**; and 4) **failure to order an appropriate consult**.[14] Some of the lawsuits resulting in liability included discharging patients with positive troponin and misinterpretation of ECGs.

Patient characteristics which put them at higher risk of being misdiagnosed include female sex, age < 55, nonwhite race, a normal ECG and shortness of breath as the chief complaint.[5,6] As providers, it is imperative that we *consider ACS in patients with atypical symptoms, interpret*

ECGs carefully, take time to *look at laboratory and diagnostic testing*, and are *not falsely reassured by a negative stress test or even a negative cardiac catheterization in a high-risk patient*.

History

Patients with ACS classically present with chest discomfort, but may also present with anginal equivalents such as shortness of breath, nausea, arm/neck pain, unexplained fatigue, syncope, or abdominal pain.[4,6,9,10] History-taking in patients who are being evaluated for possible ACS should focus on the characterization of the pain or discomfort, timing of symptoms, relationship to exertion, and associated symptoms. **Failure to appropriately characterize the patients' pain is common in missed diagnoses**.[13] The classic chest pain of ACS is usually described as a pressure or burning type pain located in the left chest or substernal area which radiates to one or both arms and is worsened with exertion or worse than the patient's typical angina.[15] Associated symptoms include nausea and/or vomiting, diaphoresis, and shortness of breath.[15] While the presence of each of these symptoms contributes to a higher relative risk of ACS, the absence of these symptoms does not rule the patient out for having ACS.[4,6,15] Additionally, symptoms which are not characteristic of ACS, such as pleuritic or reproducible chest pain, do not rule out the possibility of ACS.[4,15] The patient's history, along with the physician's opinion of the patient's level of likelihood of ACS (high, intermediate or low), should be carefully documented in the chart.

ACS is commonly misdiagnosed as gastro-esophageal reflux disease.[1] Remember that patients with ACS may present with epigastric pain rather than chest pain. Caution must be taken in these patients, especially in those whose symptoms otherwise seem characteristic for angina (i.e. relationship to exertion, relief with rest, associated shortness of breath or diaphoresis). **Improvement with GI cocktail should not be used as a diagnostic maneuver**.[15]

In conjunction with characterizing the nature of the patient's symptoms, the patient should be asked about risk factors for coronary artery disease (CAD).[4] Important risk factors for CAD include personal history of CAD, family history of CAD, age > 70, male sex, diabetes mellitus, smoking history, hypertension, and hyperlipidemia.[4] Although determining classic Framingham risk factors has not been demonstrated to improve triage decisions because the absence of these risk factors does not reduce the risk of ACS, documentation can support a reasonable medical evaluation. Patients should also be asked about less traditional risk factors such as cocaine or stimulant use, or a history of hypercoagulabilty.[4,16]

A recent negative workup for ACS is important to note in the history, but care must be taken when interpreting these results. Note the type of workup done, whether a stress test was performed and whether the level of stress reached made the test diagnostic or not. Most importantly, consider the patient's pre-test probability and consider their presenting symptoms. If the patient is at high risk by clinical history and therefore has a high pre-test probability, a negative stress test (or other diagnostic test for ACS) is more likely a false negative test result than truly negative, and thus does not rule out ACS.[17,18]

Physical Examination

The primary goals of the physical exam in the patient with suspected ACS is to assess for causes of ACS and to document comorbid conditions which may complicate their course. The physical exam should include assessment of vital signs, a careful cardiac examination including identification of evidence of CHF such as S3 gallop, elevated JVP, pulmonary edema. While a careful physical exam should be performed for all patients with possible ACS to assess for the above findings, it should be recognized that the majority of patients with ACS will have a normal physical exam. In addition, pain which is reproducible on palpation or with movement does not rule out ACS, as approximately 7% of patients with ACS have reproducible chest pain.[4]

ECG

ECGs are a low cost, rapid screening test. As such, they should be performed early, with a low threshold, and should be carefully interpreted and documented. High risk complaints, such as chest pain and unexplained dyspnea, should prompt an ECG within 10 minutes of presentation.[4] While the ECG is at the center of the evaluation of the patient with suspected ACS, it should be recognized that a single ECG has a low sensitivity, and cannot be used to rule out ACS.[4] Rather, the ECG is used to decide the next step in management. Patients with STEMI should be considered candidates for PCI, transfer to a center capable of PCI, or thrombolysis, while those with new ST depressions or T wave inversions should be considered at high risk for unstable angina or non ST-elevation MI.

Serial ECGs should be performed in order to increase sensitivity. Suggestions range from 15-60 minute intervals or at recurrence of symptoms.[4,19] Dynamic ST depressions of 0.5 mm or more which occur with symptoms, are particularly concerning for active ischemia.[4]

ECGs must be interpreted carefully, with specific attention to features which are high risk for ischemia including ST segment elevations, ST segment depressions, and T wave inversions. Best practice includes documenting absence of these findings as pertinent negatives. Misinterpretation of ischemic ECGs in the ED is fairly common, estimated to be approximately 12%,[20] and results in substandard treatment and higher inpatient mortality.[20] While ST elevations were less frequently missed than T wave inversions and ST depressions, they were missed in 8% of ST elevation MIs.[20] Patients with atypical symptoms (i.e. those presenting without chest pain) were at a higher risk of ECG misinterpretation,[20] suggesting that emergency physicians less carefully interpret ECGs when they have a lower suspicion for ACS.

Laboratory Testing

Laboratory testing in patients with ACS is used to identify patients who have had myocardial necrosis. Recent studies have shown increased sensitivity of sensitive troponin assays ("ultrasensitive troponin") within 3 hours of symptom onset. Not all hospitals have access to the more sensitive assays, and each still misses a significant percentage of patients within that time frame. The troponins that most hospitals have access to are only 41-73% sensitive at 4-6 hours after symptom onset.[19] **It is important to recognize that negative cardiac biomarkers cannot be used to exclude unstable angina.** This is a clinical diagnosis that cannot be excluded based on ECG or laboratory evidence.

ACEP recommends using one of the following three approaches to exclude non-ST-elevation MI:[19]

1. A single negative CK-MB, Troponin I or Troponin T measured 8-12 hours after symptom onset.
2. A negative myoglobin in conjunction with a negative CK-MB or negative troponin when measured at baseline and 90 minutes in patients presenting less than 8 hours after symptom onset.
3. A negative 2 hour delta CK-MB in conjunction with a negative 2 hour delta troponin in patients presenting less than 8 hours after symptom onset.

Risk Stratification

There are several risk stratification tools that integrate the above information in order to determine patients' mortality risk. The ACC/AHA recommends their use in the evaluation of patients with suspected ACS.[4] However, their use is controversial,[21,22] is not considered standard of care, and does not allow for safe discharge from the emergency department. Several studies have shown that those with a TIMI risk score of 0 still have a significant risk of ACS.[4,21-24] Clinicians can consider using risk score stratification as part of the clinical evaluation and to help select level of care and therapy for a patient.

Consultation

Interventional cardiology consultation should be undertaken immediately in patients with ECG findings concerning for ST segment elevation MI. This should be part of the standard protocol for STEMI. A formal protocol for reperfusion or transfer protocol for patients eligible for reperfusion is an important component of medical care and risk management.

Consider cardiology consultation for patients with other high risk features on ECG, especially those with deep T wave inversions or biphasic T waves in the anterior leads concerning for Wellen's syndrome. These patients are at risk for high grade LAD stenosis, and should be considered candidates for inpatient cardiac catheterization. Other high risk patients include those with a history of CABG or coronary stenting, prior myocardial infarction or known CAD. One should consider a cardiology consultation if unsure about the ECG interpretation and ensure that there is a system for prompt ECG consultation.

Disposition

Patients with high risk of ACS based on history, ECG and biomarkers should be admitted to the hospital for further management. Intermediate and low risk patients with suspected ACS can be admitted or observed in an ED chest pain unit with serial measurements of cardiac biomarkers and serial ECGs performed.

Further Testing

The ACC recommends stress testing for patients with low or intermediate risk of ACS after two sets of negative cardiac biomarkers, with coronary CT angiography (CCTA) as a reasonable alternative in those who cannot undergo provocative testing.[4] As with any diagnostic test, it is important to consider the pre-test probability of a patient prior to further testing. Given their low

pre-test probability for CAD, patients who are at very low risk of ACS are likely to have a high false positive rate of stress test and coronary CTA.[25,26] This may result in unnecessary downstream testing, perhaps exposing the patient and the health care system to unnecessary costs and risk. Part of the clinical evaluation should include discussing the clinical decision making and risks and benefits of diagnostic testing with the patient and family.

Management

The management of ACS is well known to most emergency physicians. As the choice of pharmacologic agents and the type of reperfusion therapy available vary by hospital, each hospital should have standardized treatment protocols for patients with UA/NSTEMI and those with STEMI. Each practicing emergency physician should be aware of the recommendations of the ACC/AHA and their institutional treatment algorithm.[4,27]

Discharge Instructions

Patients who are thought not to have ACS or another serious cause of chest pain based upon the clinical evaluation can be discharged from the ED. Because the evaluation of chest pain remains imperfect, indicating the limitations of the evaluation and need for follow up is important for patient care and risk management. Several standardized chest pain instructions are available. Most importantly, patients should be advised to follow up within a short time frame with their physician or cardiologist and to return to the nearest emergency department should their symptoms recur.

Thoracic Aortic Dissection

Background

Claims relating to thoracic aortic dissection represented only 2/72 closed claims for cardiovascular conditions in Massachusetts from 2005-2009; both resulted in death. Claims included not obtaining a family history of dissection and not ordering a diagnostic study (chest x-ray). Cost per claim was $750,000 and $450,000. Prior ProMutual reviews indicated 4/41 (10%) of the chest pain cases closed with payment were from TAD. Present open claims indicate an increasing liability risk related to TAD. 11/72 (15%) of open cardiac claims are for TAD. A 2008 study by Elefteriades analyzed patterns in 33 nontraumatic thoracic aorta related legal cases in which 23 patients (69.7%) had aortic dissection. All but one of the dissection cases resulted in death with the lone survivor suffering a severe stroke with neurologic deficits. The most common malpractice claim was failure to diagnose.[28] Because missed TAD can lead to unexpected and preventable death and can affect young patients and pregnant women, TAD must be considered as a possible diagnosis in all patients who present with chest pain as a chief complaint.

According to the CDC the broad category “disease of the Aorta” accounts for 43,000- 47,000 deaths annually in the United States.[29] Whereas the incidence of myocardial infarction in the United States has been estimated at 4,400 per 1 million people per year, the incidence of aortic

dissection has been estimated from 5-30 per 1 million people per year.[30] (These data represent aortic dissection ranging from 100 to 1,000 times less prevalent than myocardial infarction.)

Autopsy studies conducted prior to the modern era estimated 48 hour mortality from a proximal dissection to be 40-50%. Modern estimates of over-all in hospital 30 day mortality were reported as 27.4%. Highest mortality occurred in patients with type A dissection not receiving surgery (58.0%), while patients receiving surgery had an improved mortality (26%). Patients with type B dissection treated medically have a lower mortality (10.7%).[30]

It has been estimated that in the setting of acute chest pain, back pain, or both, acute coronary syndromes outweigh the frequency of acute aortic dissection by 80:1, with diagnostic separation failing in 0.01%.[31] Additional studies have shown that **the diagnosis of aortic dissection is frequently missed** on initial evaluation and **not made until postmortem examination in 27-55%** of patients.[28] It has been reported that **38% of dissections are missed on initial evaluation.** There are no validated decision rules for the clinical diagnosis of aortic dissection.[32] Modern aggressive treatment of ACS presents another challenge to the clinician. The consequence of rapid ACS treatment with antiplatelet, antithrombin, and fibrinolytic agents showed a higher rate of the primary end point of mortality or major bleeding (composite frequency of 54% vs. 23%).[33] Due to the severity of outcome, its time dependent nature, and similarity of presentation to acute coronary syndrome, aortic dissection continues to be a quagmire of medical malpractice litigation.

History

The classic presentation of aortic dissection is often described as chest pain with "a ripping or tearing sensation" radiating to the back. It is most often considered in middle aged men with a history of hypertension. In fact, emergency physicians suspected aortic dissection most commonly when both chest and back pain were the presenting complaints, considering the diagnosis in 86% of cases.[34] Unfortunately there can be significant variation in presentation. The same study by Sullivan et al. found that emergency physicians suspected the diagnosis only 45% of the time when chest pain was the sole presenting complaint and only 8% of the time when patients presented with epigastric, abdominal, or flank pain.[34] It has been estimated that up to 20% of affected patients present without typical signs and symptoms of chest pain or neurological dysfunction.[35]

The data surrounding presentation of aortic dissection are quite variable. A meta-analysis of 21 studies (1848 patients) by Klompas et al. found that most patients with thoracic aortic dissection present with a history of **severe pain** (pooled sensitivity 90%) **with sudden onset** (sensitivity 84%). They also found that the absence of sudden pain onset lowers the likelihood of dissection by a negative likelihood ratio of 0.3.[36] Interestingly, a history of hypertension only showed sensitivity of 64%, "ripping or tearing" quality showed only 39%, migrating pain only 31%, syncope only 9%, and Marfan's a mere 5% sensitivity. The specific location of pain (anterior, posterior, back, or abdomen) showed limited sensitivity with ranges from 57% to 23% respectively.[36] Additionally, neurological symptoms can be part of the presentation. They have been reported as dramatic, varied, and may dominate the clinical picture. In a large case series

and review article, Gaul et al. report that the frequency of neurological symptoms varied from 17- 40%.[37]

The most common risk factor has traditionally been reported as hypertension with sensitivity as high as 72%.[38] A large meta-analysis found that history of hypertension had a pooled sensitivity of 64% with an increased likelihood ratio of 1.6 (95% CI, 1.2-2.0).[36] Traditionally aortic dissection has been described as a disease affecting men in the later decades of life. It has been reported that 95% of patients present at age >40 with a mean age of 65 years.[39] A recent IRAD study of 464 aortic dissection patients noted that two thirds were male and that the mean age for all patients was 63 years.[30]

Clinicians are, however, frequently presented with patients who are younger than 40 complaining of chest pain. A 2004 IRAD study of 1078 AD patients found that 6.4% were younger than age 40. They also found that traditional risk factors such as hypertension were less common in this group while Marfan's syndrome, bicuspid aortic valve, and prior aortic valve surgery were significantly higher.[40] The International Registry of Aortic Dissection (IRAD) lists the following risk factors: Long standing arterial hypertension: smoking, dyslipidemia, cocaine/crack; Connective tissue disorders: Hereditary fibrillinopathies (Marfan's and Ehlers Danlos); Hereditary vascular disease (Bicuspid aortic valve, Coarctation); Vascular inflammation (Giant cell arteritis, Takayasu arteritis, Behcet's disease, Syphillis, Ormond's disease).[41] The 2010 ACCS/AHA Guidelines for the Diagnosis and Management of Patients With Thoracic Aortic Disease: Executive Summary lists similar categories and adds pheochromocytoma, weight lifting/valsalva maneuver, Turner's syndrome, Loeys-Deitz syndrome, pregnancy, polycystic kidney disease, chronic corticosteroid use or immunosuppresion agent administration, and infections involving the aortic wall.

Despite the varied presentation and risk factors, it is important to **take a history** that minimally inquires about **quality, radiation, and intensity at onset**. One investigation of 83 patients with subsequent confirmed aortic dissection noted that only 42% of conscious patients were asked all three questions. The study went on to show that if all 3 questions were asked diagnostic accuracy improved to 91%. If one or more of the three questions were omitted, suspicion fell to 49%.[42] (Of course the retrospective nature of this study is limiting. It is possible that physicians were more likely to ask questions if they had an initially high degree of clinical suspicion from other data: chest xray, physical exam, etc. Nonetheless, history matters.)

Physical Exam

Although a history of hypertension is considered a significant risk factor for the development of aortic dissection it is less commonly seen on initial presentation. An IRAD review noted that elevated blood pressure was present in 36% of type A dissection and 70% of type B 56.[30] Pooled sensitivity from a large meta-analysis shows acute hypertension to be present in 64% of cases.[36]

Impaired blood flow can present as a **pulse deficit** in any of the major associated vessels and is often a much more subtle finding. It was found to be present in the carotid, brachial, or femoral arteries infrequently (31%). However, when present in the setting of chest pain or back pain the positive likelihood ratio increased to 5.7 (95% CI 1.4-23).[36]

A diastolic heart murmur has often been discussed as one possible manifestation of a type A dissection however it has little value in predicting dissection. One third of patients with aortic dissection will have a diastolic murmur (pooled sensitivity 28%). However the positive likelihood ratio for this finding was found to be only 1.4 and the negative likelihood ratio was found to be 0.9. Whereas a new murmur might be helpful in making the diagnosis, it is unlikely that an emergency physician would be able to make such an observation given limited familiarity with most patients.[36]

Focal neurological deficits, although rare (17% of cases), can be very helpful in diagnosing dissection. The high specificity of focal neurological deficits yielded a positive likelihood ratio ranging from (6.6-33).[31,43] Unfortunately the absence of focal neurological deficits does not rule out dissection.[36] Neurological deficits can be isolated. In one study, up to 10% of dissection patients presented with neurological symptoms, and no chest pain.[37]

Evaluation

ECG on its own is of limited value in diagnosing aortic dissection. One study of 464 patients found the ECG to be normal 31% of the time. Nonspecific ST and T wave changes were identified in 42% of patients. Ischemic changes were seen in 15% of patients and those with type A (ascending) dissection showed evidence of infarction 5% of the time.[30] Meta-analysis showed new Q waves or ST segment elevation in 7% of admission ECGs. Yet, in the same analysis normal ECGs were documented an average of 22% of the time.[36]

Chest x-ray also has limited utility as the sole diagnostic study, however, when used in combination with key history and physical exam findings it can be quite helpful.[31] In a study of 216 patients chest radiography had a sensitivity of 64% and a specificity of 86%. Sensitivity was lower for pathology involving the proximal aorta (47%) and better for disease involving distal aortic segments (77%).[31] A review of 464 patients found mediastinal widening present in 63 percent with type A dissections and 56 percent of type B dissections. The same study unfortunately reported **no abnormality whatsoever in 11% of type A and 16% of type B patients**.[30] Because chest radiography can yield variable or indeterminate results, sensitivity and specificity are somewhat limited. Almost all patients with suspected aortic disease receive more definitive testing.

CT is considered the most frequently used definitive diagnostic test. Its sensitivity exceeds 95% with specificities ranging from 87-100%.[38]

Ultrasound: Transthoracic echocardiography (TTE) has limited value in imaging the entire aorta but can help with identifying involvement of the proximal aorta and with assessing some of the complications of dissection (aortic insufficiency, pericardial tamponade, and regional LV dysfunction). An appropriately trained EP can identify aortic widening or an intimal flap using TTE-findings with high specificity for TAD in a patient with chest pain. Transesophageal echocardiography (TEE) requires experienced staff and is often not obtainable on an emergent basis. However, it can be performed at the bedside in an unstable patient and its availability is institution dependent. It has been reported to reach a sensitivity of 99% with a specificity of 89%. MRI is less commonly used in the acute evaluation, but may be used for preoperative staging.[38]

D-dimer has been discussed as a potential biomarker to screen for TAD and much hope has been placed in finding a future biomarker that will help "rule out" this disease. Initial investigations were promising; however, a recent meta-analysis of studies using D-dimer as the "sole screening tool" for acute dissection found that there is a subset of patients who develop a thrombosed false lumen, which may be less likely to stimulate the clotting cascade than those with luminal extension, resulting in negative D-dimer results. Additionally none of the studies defined a specific patient population eligible for D-dimer screening and many studies had wide confidence intervals because of low patient numbers. Despite high sensitivity, D-dimer could not be recommended as the sole screening tool for acute aortic dissection.[44] In the future, biomarkers along with other key clinical variables and ancillary studies may play a role in the formation of algorithms and decision rules to better identify aortic dissection.

Summary of History, Physical Exam, and Evaluation: An analysis of 250 patients with acute chest and/or back pain (128 with a dissection) found that 96 percent of acute aortic dissections could be identified based upon some combination of the following three clinical features.[31]

- **Abrupt onset of thoracic or abdominal pain with a sharp, tearing and/or ripping quality**
- **Variation in pulse (absence of a proximal extremity or carotid pulse) and/or blood pressure** (>20 mmHg difference between the right and left arm)
- **Mediastinal and/or aortic widening on chest radiograph**

(Probability of dissection was low with absence of all 3 variables: 7%.)

"Aortic pain" alone (sudden, tearing, ripping) has a positive likelihood ratio of 2.6. The presence of both aortic pain and pulse or blood pressure differentials increased the likelihood ratio to 10.5%. The addition of abnormal widening of the mediastinum on chest radiograph essentially seals the diagnosis with a likelihood ratio of 66.0. Unfortunately only 27% of all dissection patients presented with this triad.[36] Although many patients suspected to have dissection turn out not to have acute aortic disease, anywhere 50-75% are diagnosed with alternative serious disease. This should encourage a thorough work-up in any patient suspected of having aortic pathology.[31,43]

Treatment and Management

Treatment and management of aortic dissection is somewhat controversial and somewhat dependent upon the resources available to the clinician. The 2010 ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM Guidelines for Diagnosis and Management of Patients With Thoracic Aortic Disease: Executive Summary provides detailed recommendations on how to manage aortic disease.[45] All patients with suspected dissection should undergo further diagnostic evaluation immediately under close monitoring. Patients should be admitted to an intensive care unit or transferred (with appropriate staff and monitoring) to a facility with cardiothoracic surgery capabilities. Pain can be controlled with opiates, and a systolic blood pressure of 110 mm Hg should be the goal of therapy. Beta-blockers are generally the antihypertensive of choice and usually sufficient, however, additional vasodilating agents may be required. If beta blockers are contraindicated, intravenous verapamil or diltiazem may also be used. **Surgical services must be consulted early** in all cases of suspected aortic

dissection and transfer to an appropriate facility with appropriate interfacility care at sites without thoracic surgery.

Documentation

From a risk management perspective, documentation of the history of pain, clinical evaluation and clinical reasoning are important. Some risk managers have suggested that symmetrical blood pressures and symmetrical pulses should be documented in **all chest pain patients** because of the liability risk with missed dissection. Consider documenting these findings in patients with presentation suggestive of TAD.

Pulmonary Embolism

Background

Over 500,000 cases of pulmonary embolism (PE) events occur each year in the United States. Of these cases, over 50% are fatal.[46] *Failure to diagnose pulmonary embolism* remains a liability concern for emergency physicians **(8% of open cardiovascular claims as of 2010)**. The diagnosis of pulmonary embolism must be considered in all patients with acute cardiovascular complaints including chest pain, dyspnea, palpitations and syncope. Infectious diagnoses (pneumonia, pleurisy) or respiratory diagnoses (COPD or asthma) are often sources of diagnostic error. The clinical presentation of PE can vary from mild dyspnea on exertion or unilateral leg swelling to severe respiratory distress and cardiac arrest.[47] Although historically mortality rates for untreated or missed PE were 26-30%, a recent study shows that with new technologies to diagnose PE, mortality and recurrence rates are now less than 5%.[48] However, because anticoagulation therapy has been shown to decrease mortality rates,[49] failure to diagnose PE can be associated with what is considered a preventable adverse outcome.[48] The availability of validated risk stratification tools and rapid access to diagnostic imaging has advanced the ability of emergency physicians to rapidly diagnose PE. However, **failure to consider the diagnosis** and **errors in diagnosis** remain liability concerns.

Risk Factors

In patients involved in the PIOPED II study**, fewer than 20% of patients** with PE present with the classic triad of hemoptysis, dyspnea, and chest pain. However, **94% had at least 1 identified risk factor**.[47] Reviewing a patient's risk factors is a key component of assessing the likelihood of PE. Risk factors can be either hereditary or acquired.

Acquired risk factors for PE are more common than inherited thrombophilias. Acute medical illness or reduced mobility lead to the majority of episodes of PE: examples include hospital or nursing home confinement, recent surgery, trauma, malignancy, paresis (stroke), etc.[47,50,51] Extended travel times have also been associated with PE.[52] For female patients, oral contraceptive use or hormone replacement therapy are additional risk factors for PE.[53]

Hypercoagulable states, whether inherited or acquired, are associated with increased PE events. Inherited thrombophilias may be suspected in patients who present with PE at a young age (see Table 1). Acquired hypercoagulable states that elevate risk of PE include polycythemia vera and antiphospholipid syndrome (e.g. lupus) and active inflammatory bowel disease.[54] In addition, a personal or family history of prior venous thromboembolism elevates risk for PE.[50] Patients with a prior history of VTE are 8 times more likely to have recurrent VTE compared to patients without a history of DVT or PE.[55] Therefore noting patient level factors or a family history of DVT/PE is an important part of risk stratification.

Clinical Evaluation

A **minority** of patients present with the classic symptoms associated with PE: dyspnea, pleuritic chest pain and hemoptysis. Clinical findings associated with PE can include the following: history-dyspnea, chest pain, unilateral calf pain or swelling, physical exam-tachypnea, hypoxemia, tachycardia, and exam findings suggestive of deep venous thrombosis (DVT).[50] Of note orthopnea, which is not historically associated with PE, was a moderately frequent complaint (38%) in patients with PE in the PIOPED II study.[47] Hemoptysis does not have any predictive value for diagnosing PE.[47,50]

No single physical exam finding is sensitive (rules out) or specific (rules in) for PE. Half of the patients in the PIOPED II study with PE did not have tachypnea; three quarters of PE patients were not tachycardiac. The presence of fever does not exclude PE nor make an infectious process such as pneumonia more likely in a patient with respiratory symptoms. Only 30% of PE patients have abnormal lung exam findings.[47] Lower leg swelling or tenderness may suggest DVT. However these findings were neither sensitive nor specific for PE.[47,54] From a risk management perspective, **one can not use a normal heart rate, respiratory rate or oxygen saturation to exclude the diagnosis of PE**.

Atypical Presentations

"Atypical presentations" of PE include syncope or cardiac arrest.[56] Syncope is present in 8-13% of all patients with PE and is more frequent in patients with massive PEs (20%) compared to those with smaller PEs (4%). Pulseless electrical activity is the primary presenting rhythm in patients presenting with cardiac arrest secondary to PE.

PE can be challenging to diagnose in certain populations. The frequency of PE events increases with age; thus the likelihood that a patient with PE will have multiple comorbidities is raised as well. Patients with cardiopulmonary diseases such as congestive heart failure (CHF), coronary artery disease (CAD), or chronic obstructive pulmonary disease (COPD) may have similar complaints of chest pain or shortness of breath that obscure the process of diagnosing PE. In one study, 19.9% of patients hospitalized for acute COPD exacerbations were later found to have objective evidence for PE requiring therapeutic anticoagulation.[57] Clinicians should have a low threshold to evaluate patients for COPD if they are being admitted for acute COPD exacerbations, especially if there is a history of prior VTE or active malignancy. The risk of missed PE is higher in these populations as overall mortality for PE is higher in COPD (12%) and CHF (17%) patients compared to those without either diagnosis (10%).[58]

In addition, clinicians may overlook PE in atypical population groups, such as children or pregnant women, leading to cases of missed PEs. Pulmonary embolism is rare in children[56] and is due to an inherited or acquired hypercoagulable state. The most common acquired risk factor in children is central venous access devices. Other factors include infection, renal disease, autoimmune diseases, vasculitis, severe inflammatory bowel disease, malignancy, surgery and trauma.

The incidence of PE **is 5 times higher in pregnant women** compared to non-pregnant women. The postpartum period is associated with an even higher risk of PE. However, PE is especially difficult to diagnose as dyspnea is a common complaint during pregnancy. Physiologic dyspnea of pregnancy is usually mild, with no symptoms present at rest. Symptoms tend to remain stable as the fetus matures. Syncope, hemoptysis, chest pain, shortness of breath at rest, or rapid onset of symptoms should raise red flags for further workup and not be attributed to physiologic dyspnea.

Pretest Probability

If PE is on the differential diagnosis, further evaluation is based on risk factors, history and physical exam. An objective clinical assessment tool should be used to determine the pretest probability prior to instigating further workup[54,59] (See Appendix, Tables 2-4 for prediction tools). Comparison between the Canadian (Wells) scoring system with either the revised or the original Geneva criteria has shown similar accuracy in predicting the likelihood of PE in emergency department patients.[60,61] However, the Geneva score has not been as well tested as the revised Wells criteria and revisions of the original Geneva criteria have not been tested for clinical usefulness in outcome studies.

Preliminary Workup

Based on clinical assessment (formed from history, physical exam, risk factor profile, etc.), standard workup for chest pain or shortness of breath should be considered. ECG findings are nonspecific, but may be consistent with acute right heart strain such as right axis deviation, right bundle branch block or the "classic" PE ECG finding of the S1Q3T3 pattern. Nonspecific t-wave changes often trigger an admission for possible ACS in a patient with "atypical chest" pain leading to a failure to consider and diagnose PE. The CXR is often non-diagnostic for PE, and findings (Hampton's hump, Westermark's sign) are rare but could identify another cause of chest pain and dyspnea (e.g. pneumothorax etc). ABG results have not been shown to be of diagnostic value in clinical studies.[62] In conjunction with pre-test probability assessment, an enzyme-linked immunosorbent assay (ELISA)-based D-dimer tests (sensitivity 97-100% and negative-predictive value 99.6%) enables use of a negative result to rule out PE in patients with low pretest probability.[63] The primary drawback to D-dimer assays is that they can be positive in any patients with inflammatory states, such as pregnancy, infection, cancer, trauma, etc., and therefore cannot be used uniformly for all patients.[64] Kline et al. developed the pulmonary embolism rule-out criteria (PERC), an eight variable decision rule to validate not ordering further diagnostic testing in patients determined to be of low clinical suspicion for PE.[65] A prospective multicenter study later showed that a combination of low clinical suspicion for PE with a negative PERC result reduced the probability of VTE to less than 2%. There is controversy about whether the PERC rule can be used in clinical practice or requires further testing.

Definitive Diagnosis and Management

Computed thoracic (CT) pulmonary angiography has become the test of choice for definitive diagnosis of PE.[66] Treatment of segmental or subsegmental PEs seen on CT, is controversial and the investigators of the PIOPED II study recommend reassessing the certainty of the CT diagnosis as the cause of the patient's symptoms.[59,66] Patients who have contraindications to CT pulmonary angiography should undergo other diagnostic imaging studies (usually a V/Q scan). In patients with a high pretest probability for PE but for whom the initial imaging scan is negative, consideration for further diagnostic testing is warranted. Alternatively, imaging studies may be ordered to detect DVT. In unstable patients, bedside transthoracic echocardiography (TTE) may be suggestive of either PE or DVT and can be performed by an appropriately trained EP. In one study assessing massive PE in hemodynamically unstable patients, if any two of three variables were positive (high clinical probability of PE, bedside TTE, or lower extremity ultrasonography), the sensitivity was 97% and the negative predictive value was 98%.[67]

In patients who have moderate-to-high pretest probability for PE, consider initiating treatment with antithrombin therapy prior to obtaining diagnostic testing results.[59] Patients with contraindications to anticoagulation or reoccurrence of VTEs despite therapeutic anticoagulation may require a venous filter.[59] Thrombolytics should be used in patients with shock secondary to a massive PE; however, in patients who are hemodynamically stable, thrombolytics have not been shown to reduce mortality or recurrent PE.[68] In patients with massive PE, surgical thrombectomy and percutaneous catheter fragmentation can also be considered.[54,69] An institutional pathway for DVT/PE (including those with massive PEs) can encourage a consistent management strategy based upon an institution's resources and a patient's clinical condition.

Other Chest Pain Diagnoses

Although the diagnoses responsible for medical legal risk for emergency physicians are ACS, TAD and PE, it is important to note that there are other diagnoses that are associated with liability exposure. Among these are myocarditis (4 of the open claims pending as of 2010), bacterial endocarditis and heart failure.

Myocarditis can be a difficult diagnosis, present with nonspecific symptoms especially during a time when viral illnesses associated with myocarditis, such as influenza, may be prevalent. Exercise caution when a patient, especially child or young adult with either a suspected viral syndrome or recent viral syndrome, reports cardiovascular complaints such as dyspnea, has low oxygen saturation or either unexplained or persistent tachycardia. Cardiomegaly on CxR or depressed ejection function on transthoracic ECHO (can be done by an appropriately trained emergency physician) can support the diagnosis of myocarditis. Because of the risk of death from ventricular dysrhythmia or heart failure and the young age of patients whose adverse outcomes have often triggered malpractice suites, emergency physicians should be aware of myocarditis as a liability risk.

Bacterial endocarditis (BE): Exercise caution when a patient with chest pain has fever, murmur or is at increased risk for BE (injection drug use, abnormal or prosthetic valve). If

blood cultures are sent in a patient at low risk for BE, ensure a follow up system to contact the patient if positive.

Heart failure (CHF): Heart failure or cardiogenic pulmonary edema is identified in 7 of the open malpractice claims.

SUMMARY POINTS

History:
- Pain: timing, location, quality, radiation, mitigating and exacerbating factors, severity
- Associated symptoms
- Previous similar symptoms
- Previous cardiac history
- Previous cardiac studies: ECG, stress test, catheterization
- Risk factors
- Family history of ACS, TAD, PE

Physical examination:
- Look for complications of ischemia (rales, edema, S3 gallop, rales, or a new systolic murmur).
- May identify an alternate diagnosis.
- Beware of chest wall tenderness.
- Consider documenting symmetric BP, presence of pulses.

Evaluation:
- ECG: have a low threshold for ordering it.
- A normal electrocardiogram does not exclude cardiac ischemia.
- Compare with previous ECGs.
- Use as a supplement to your clinical judgment.
- Cardiac biomarkers: most useful when performed serially.
- A single enzyme determination cannot rule out cardiac ischemia. Interpret in clinical context.
- If a cardiac troponin is obtained, discharge with an elevated level is a high risk decision.
- Obtain and interpret other tests (chest x-ray, d-dimer) in clinical context.

Treatment:
- Have a protocol in place for reperfusion therapy.
- Clinical pathways (low risk chest pain, other chest pain diagnoses) can standardize care and reduce liability exposure

Risk Management:
- Documentation is crucial
- Document history, risk factors, past cardiac history, physical examination.

- Write your ECG findings on the chart and document comparison with previous ECGs if available.
- Document discussions with consultants and the patient's physician.

Discharge Instructions:
- Ensure the instructions include specific symptoms for which they should return for recheck.

ACS Summary:

1. Carefully document a complete history and physical exam as well as the clinical reasoning behind testing and management decisions
2. Consider the diagnosis of ACS in patients presenting with atypical complaints such as shortness of breath, nausea or weakness
3. A "GI" presentation for ACS is high risk. Consider diagnoses such as "GERD" or "esophageal spasm" to be high risk diagnoses in patients with chest pain or epigastric pain and possible ACS.
4. Have a low threshold to obtain an ECG, and spend time carefully interpreting and comparing the ECG to an old ECG
5. Do not rely on a single set of cardiac enzymes in patients presenting within 12 hours of symptom onset or if the onset is unable to be reliably determined
6. Consider unstable angina and further testing in patients who have ruled out for MI.
7. Give clear discharge instructions to patients regarding follow up and when to return to the emergency department

TAD Summary:

1. TAD has a low incidence but high rate of malpractice claims
2. TAD is easily mistaken for ACS and both diagnoses can occur simultaneously.
3. Document that these essential chest pain history questions were asked:
   - quality
   - radiation
   - intensity at onset
4. Pursue the diagnosis of TAD in a patient with these essential findings:
   - **severe pain with sudden onset**
   - **pulse deficit in any of the major associated vessels**
   - **chest x-ray showing mediastinal widening**
5. Do not let a negative chest x-ray rule out dissection is a patient with high suspicion.
6. Manage pain and blood pressure appropriately
7. Consult surgery early or stabilize and transfer to an appropriate facility.
8. Be aware of common TAD presentations
   i. -Chest pain with neurologic symptoms
   b. Chest pain with limb ischemia
   c. Chest pain with syncope
9. Consider TAD in young patients, pregnant women and patients with risk factors for TAD

PE Summary:

1. Consider PE in differential of acute cardiovascular complaints especially chest pain, dyspnea and syncope, any patient with tachypnea.
2. PE should be considered in patients with chest pain and t-wave abnormalities. Diagnosing ACS and missing PE is a noted error.
3. Consider important aspects of the history (risk factors, family history) and exam to risk stratify. Documentation of this evaluation can indicate that medical decision-making was reasonable.
4. Consider the use of a validated decision rule to risk stratify for diagnostic testing
5. Admission and disposition may be dependent on patient stability and availability of testing.
6. In patients who are discharged home, provide appropriate instructions to return for worsening of symptoms

Appendix

TIMI Risk Score:

The TIMI score assigns a one point value to each of the following predictor variables: age greater than 65, three or more traditional risk factors for CAD, known CAD, two or more episodes of angina in the preceding 24 hours, aspirin in the 7 days prior to presentation, ST segment deviation of 0.5 mm or more, and elevated cardiac markers.

TIMI Risk Score Predictor Variables for Patients with NSTEMI or UA

- Age ≥ 65 years
- ≥3 risk factors for CAD
    - hypertension
    - dyslipidemia
    - diabetes mellitus
    - cigarette smoking
    - family history of CAD
- Prior coronary stenosis ≥50%
- ST-segment deviation on ECG at presentation
- ≥2 anginal events in the prior 24 hours
- Use of aspirin in the prior 7 days
- Elevated serum cardiac markers

CAD = coronary artery disease; ECG = electrocardiogram; NSTEMI = non-ST-segment elevation myocardial infarction; TIMI = Thrombolysis in Myocardial Infarction; UA = unstable angina

Patient risk might be classified as low for a TIMI risk score of 1 or 2, moderate for a score of 3 or 4, and high based on a score of 5 or 6.

Table 1. Risk Factors for Pulmonary Embolism[51,54]	
Inherited	Acquired
Antithrombin III deficiency Protein C deficiency Protein S deficiency Factor V Leiden Activated protein C resistance (most common) without factor V Leiden Prothrombin gene mutation Dysfibrinogenemia Plaminogen deficiency *Prior patient or family history of venous thromboembolism	Immobilization - Travel - Paralysis/Spinal Cord Injury - Bedridden state - Immobilizer or cast Surgery Trauma Acute medical illness Malignancy (active) Hypercoagulability state - Polycythemia vera - Antiphospholipid antibody syndrome Central venous access devices Pregnancy and the puerperium Oral contraceptives/hormone replacement therapy Advanced age Obesity Inflammatory bowel disease

Table 2. Revised Canadian (Wells) Prediction Score[70]	
Variable	Score
DVT symptoms and signs	3.0
PE as likely as or more likely than alternative diagnosis	3.0
Heart rate >100 beats/min	1.5
Immobilization or surgery in previous 4 weeks	1.5
Previous DVT or PE	1.5
Hemoptysis	1.0
Cancer	1.0
Total Score	
Score	Pretest Probability
<2.0	Low
2.0 – 6.0	Moderate
>6.0	High

Table 3. Original Geneva (Wicki) Score[71]	
Variable	Score
Age - 60-79 years - ≥ 80 years	 1 2
Previous DVT or PE	2
Recent surgery (in previous 4 weeks)	3
Heart Rate > 100 beats/min	1
PaCO2 - <36.2 mmHg (<4.8 kPa) - 36.2-38.9 mmHg (4.8-5.19 kPa)	 2 1
PaO2 - <48.8 mmHg (<6.5 kPa) - 48.8-59.9 mmHg (6.5-7.99 kPa) - 60-71.2 mmHg (8.0-9.49 kPa) - 71.3-82.4 mmHg (9.5-10.99 kPa)	 4 3 2 1
Chest radiograph - platelike atelectasis - elevation of hemidiaphragm	 1 1
Total Score	
Score	Pretest Probability
0-4	Low
5-8	Moderate
9-12	High

Table 4. Revised Geneva Score[72]	
Variable	Score
Age > 65 years	1
Previous DVT or PE	3
Surgery or lower limb fracture in previous week	2
Active cancer	2
Unilateral lower limb pain	3
Hemoptysis	2
Heart rage - 75-94 beats/min - ≥ 95 beats/min	 3 5
Pain on leg palpation or unilateral edema	4
Total Score	
Score	Pretest Probability
0-3	Low
4-10	Moderate
≥ 11	High

Module 2: Chest Pain References

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