Chest Pain Presentations and ECG Interpretations

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Abstract

Acute coronary syndromes remain a public health concern that has a negative impact on the quality of life of thousands of patients worldwide and represents an important economic burden for health-care systems. To reduce ACS morbidity and mortality in the emergency room (ER), an expedite and accurate diagnosis of acute ischemic heart disease is mandatory. Rapid identification of coronary syndromes leads to prompt and optimal treatment, therefore reducing the burden of chronic disease if patients achieve revascularization within an optimal time frame. Early recognition of ECG changes and use of chest pain scores can reduce delays to treatment and improve clinical outcomes for acute myocardial infarction patients and other medical emergencies. This chapter summarizes ACS pathology, classification, clinical presentation, risk stratification, biomarker use, imaging modalities, as well as the most universally accepted diagnostic and therapeutic strategies.

Keywords: *Acute Coronary Syndromes; Emergency Room (ER); ECG; ACS*

Introduction

Acute coronary syndromes remain a public health concern that has a negative impact on the quality of life of thousands of patients worldwide and represents an important economic burden for health-care systems.

Objectives of the Study

- Establish a differential diagnosis for chest pain.
- Know what clues to obtain on history to rule-in or out MI, PE, pneumothorax and aortic dissection.
- Identify risk factors for MI.
- Know how to do a focused physical exam, identifying features that would distinguish between MI, PE, pneumothorax and aortic dissection.
- Identify investigations required in diagnosing MI.
- Outline management strategy in MI.

Case Series and Discussion

Case 1

A 61-year-old man was on his way to the grocery store when he started feeling chest pressure in the center of his chest. he became diaphoretic and felt short of breath. He notified EMS who brought him to the ED. On arrival to the ED, his blood pressure is 130/70 mm Hg, heart rate 76 beats per minute and oxygen saturation of 98% on room air.
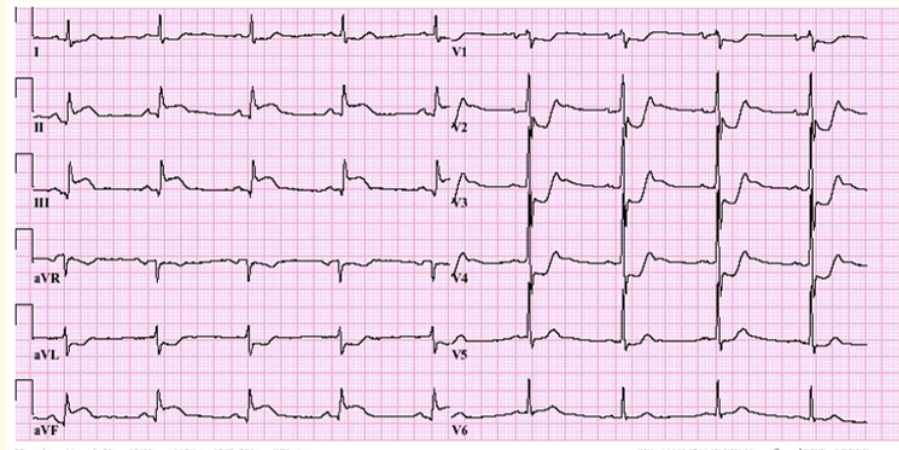
Figure 1

STEMI

A 12-lead ECG should be performed within 10 minutes of ED arrival for all patients with chest discomfort (or anginal equivalent) or other symptoms suggestive of STEMI.

If the initial ECG is not diagnostic of STEMI but the patient remains symptomatic, and there is a high clinical suspicion for STEMI, serial ECGs at 5 to 10 minute intervals or continuous 12-lead ST-segment monitoring should be performed to detect the potential development of ST elevation.

In patients with inferior STEMI, right-sided ECG leads should be obtained to screen for ST elevation suggestive of right ventricular (RV) infarction.

Laboratory examinations should be performed as part of the management of STEMI patients but should not delay the implementation of reperfusion therapy.

For patients with ST elevation on the 12-lead ECG and symptoms of STEMI, reperfusion therapy should be initiated as soon as possible and is not dependent on a biomarker assay.

The delay from patient contact with the healthcare system (typically, arrival at the ED or contact with paramedics) to initiation of fibrinolytic therapy should be less than 30 minutes.

Alternatively, if PCI is chosen, the delay from patient contact with the healthcare system (typically, arrival at the ED or contact with paramedics) to balloon inflation should be less than 90 minutes.

Patients with STEMI should have a portable chest X-ray, but this should not delay implementation of reperfusion therapy (unless a potential contraindication, such as aortic dissection, is suspected).

Portable echocardiography is reasonable to clarify the diagnosis of STEMI and allow risk stratification of patients with chest pain on arrival at the ED, especially if the diagnosis of STEMI is confounded by left bundle-branch block (LBBB) or pacing, or there is suspicion of posterior STEMI with anterior ST depressions.

Supplemental oxygen should be administered to patients with arterial oxygen desaturation (SaO$_2$ less than 90%).

Nitrates should not be administered to patients with systolic blood pressure less than 90 mm Hg or greater than or equal to 30 mm Hg below baseline, severe bradycardia (less than 50 bpm), tachycardia (more than 100 bpm), or suspected RV infarction.

Nitrates should not be administered to patients who have received a phosphodiesterase inhibitor for erectile dysfunction within the last 24 hours (48 hours for tadalafil).

Case 2

A 59-year-old woman with a history of prior CABG 8 years ago presents with on- going chest discomfort for 45 minutes that has been unrelieved with 3 sublingual nitroglycerin tablets. Past medical history is notable for diabetes controlled with oral medication, hypertension, and a family history of coronary disease. She has a history of a non–ST-elevation myocardial infarction (NSTEMI) with preserved ejection fraction. In the ambulance, she received additional sublingual nitroglycerin and had resolution of her pain. In the emergency department, an ECG showed 1 mm of ST-segment depression anteriorly. Initial troponin measurements were negative.

NSTE-ACS

In patients with chest pain or other symptoms suggestive of ACS, a 12-lead electrocardiogram (ECG) should be performed and evaluated for ischemic changes within 10 minutes of the patient's arrival at an emergency facility.

If the initial ECG is not diagnostic but the patient remains symptomatic and there is a high clinical suspicion for ACS, serial ECGs (e.g. 15 to 30 minute intervals during the first hour) should be performed to detect ischemic changes.

Serial cardiac troponin I or T levels should be obtained at presentation and 3 to 6 hours after symptom onset in all patients who present with symptoms consistent with ACS to identify a rising and/or falling pattern of values.

Patients with suspected ACS should be risk stratified based on the likelihood of ACS and adverse outcome(s) to decide on the need for hospitalization and assist in the selection of treatment options.

TIMI Score	Yes 1 point	No 0 points
Age ≥65		
≥3 Risk factors for ACS; hypertension, hyperlipidemia, smoking, diabetes, family history		
Use of aspirin in last 7 days		
Prior coronary stenosis >50%		
≥2 angina events in 24 hours or persisting discomfort		
ST-segment deviation of ≥0.05 mV on initial ECG		
Elevated cardiac biomarkers		
Total Score		

Low Risk	0-2
Intermediate Risk	3-4
High Risk	5-7

HEART Score		Points
History	Highly Suspicious	2
	Moderately Suspicious	1
	Slightly Suspicious	0
ECG	Significant ST-depression	2
	Non-specific repolarization abnormality	1
	Normal	0
Age	≥65	2
	45-65	1
	≤45	0
Risk factors	3 or more risk factors	2
	1-2 risk factors	1
	No risk factors	0
Troponin	≥3× normal limit	2
	1-3× normal limit	1
	≤ normal limit	0
Total		

Figure 2

The HEART Score is a scoring system to help emergency departments risk-stratify chest pain patients: who will have a MACE within in the next 6 weeks and who will not?

It involves only a 1-time troponin, at admission.

The rest of the score is based on age, history, risk factors and ECG.

Low risk patients have a score 0-3 and have a less than 2% risk of MACE at 6 weeks.

MACE is defined as: all-cause mortality, myocardial infarction, or coronary revascularization.

All other scores are high risk and require further management and admission.

While HEART is sometimes compared to TIMI and GRACE (older ACS scores), these measure risk of death for patients with ACS, and do not do as well telling who has ACS in the first place:

1. 0 - 3: 0.9 - 1.7% risk of adverse cardiac event. In the HEART Score, these patients were discharged.

2. 4 - 6: 12 - 16.6% risk of adverse cardiac event. In the HEART Score, these patients were admitted to the hospital.

3. 7 - 10: 50 - 65% risk of adverse cardiac event. In the HEART Score, these patients were candidates for early invasive measures.

Case 3

A 47-year-old man with a history of hypertension presents to the ED complaining of continuous left sided chest pain that began while snorting cocaine 1 hour ago. The patient states he never experienced chest pain in the past when using cocaine. His blood pressure is 170/90 mm Hg, heart rate is 101 beats per minute, respiratory rate is 18 breaths per minute, and oxygen saturation is 98% on room air.
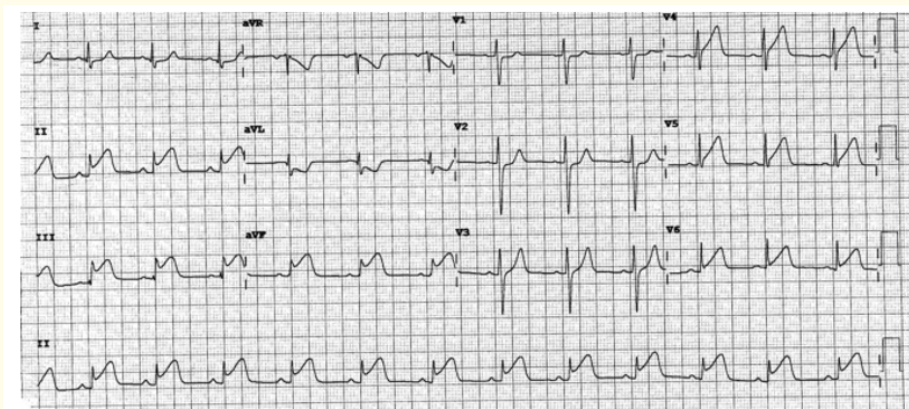
Figure 3

Cocaine induced MI

Aspirin and nitrates continue to be strongly recommended as they are for non–cocaine-associated acute coronary syndrome (ACS), but β-blockers (including agents with mixed α-adrenergic antagonist effects, such as labetolol) are considered contraindicated.

If cocaine intoxication is suspected, benzodiazepines are recommended as the primary treatment for anxiety, tachycardia, and hypertension.

Early percutaneous coronary intervention is particularly preferred over fibrinolysis in patients with cocaine-associated MI because of increased risk for intracranial hemorrhage after administration of fibrinolytic agents in cocaine users.

Case 4

A 21 year old male student from Philippines collapses while playing football. On arrival of paramedics the football doctor and some players are performing CPR, he has been unconscious for 10 minutes. The paramedics shock him once using an AED shock box and there is a return of spontaneous circulation. On arrival in hospital he opens his eyes in response to voice, he utters inappropriate words and localises painful stimuli. His airway is patent and he is spontaneously breathing with saturations of 100% on high flow oxygen. His pupils are equal and reactive.
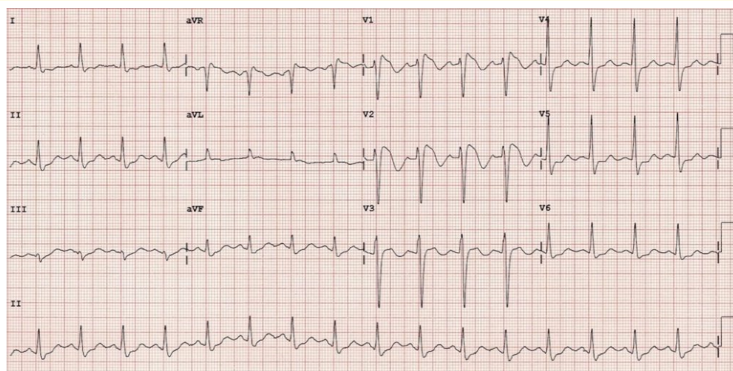

Figure 4

Brugada syndrome

Brugada syndrome is a disorder characterized by sudden death associated with one of several ECG patterns characterized by incomplete right bundle-branch block and ST-segment elevations in the anterior precordial leads.

Signs and symptoms in patients with Brugada syndrome may include the following:

- Syncope and cardiac arrest: Most common clinical manifestations; in many cases, cardiac arrest occurs during sleep or rest.

- Asymptomatic, but routine ECG shows ST-segment elevation in leads V1-V3.

- Associated atrial fibrillation (20%).

- Fever: Often reported to trigger or exacerbate clinical manifestations.

Management of Brugada syndrome

To date, the only treatment that has proven effective in treating ventricular tachycardia and fibrillation and preventing sudden death in patients with Brugada syndrome is implantation of an automatic implantable cardiac defibrillator (ICD).

No pharmacologic therapy has been proven to reduce the occurrence of ventricular arrhythmias or sudden death.

Case 5

37 year old male called the ambulance complaining of intermittent chest pain over the past 3 days.

Episode described as: 7/10 on the pain scale with retrosternal and radiating down left arm diaphoresis, palpitations, dizziness.

Also reported to EMS: Two episodes the previous day lasted about 15 min and were relieved with rest. One episode the previous awoke him from sleep. Most recent episode was immediately prior to call.

Pre hospital assessment and care:

1. O_2 by nasal cannula.
2. Cardiac monitoring including a 12-lead.
    Vital Signs: HR 83 RR 18 BP 125/80 Temp 36.6 O_2 Sats 99%.
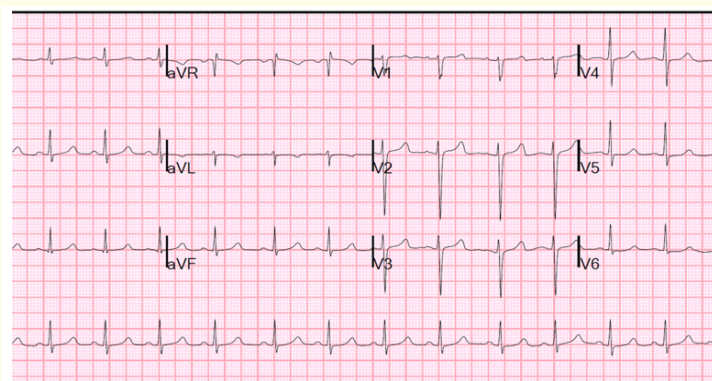3. IV line.
4. GTN.

Pre-hospital ECG - Pain 5/10

Figure 5

Upon arrival at ER - Patient pain free post EMS treatment

Figure 6

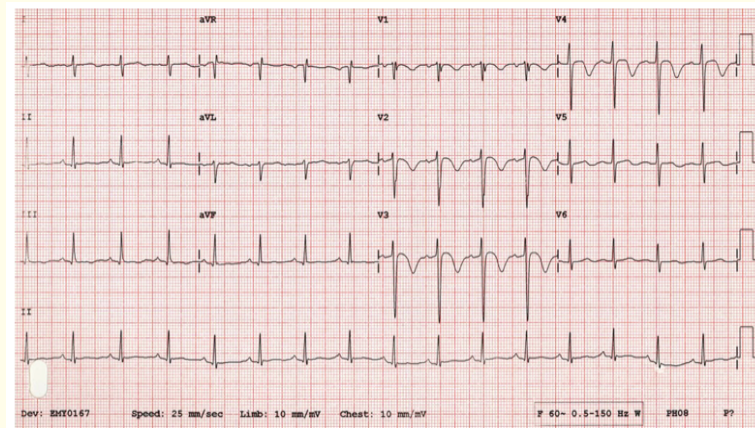## Wellens syndrome

Wellens syndrome was first described in the early 1980s by de Zwaan, Wellens, and colleagues, who identified a subset of patients with unstable angina who had specific precordial T-wave changes and subsequently developed a large anterior wall myocardial infarction (MI).

Wellens syndrome refers to these specific electrocardiographic (ECG) abnormalities in the precordial T-wave segment, which are associated with critical stenosis of the proximal left anterior descending (LAD) coronary artery.

Recognition of this ECG abnormality is of paramount importance because this syndrome represents a preinfarction stage of coronary artery disease (CAD) that often progresses to a devastating anterior wall MI.

Evolution to an anterior wall MI is rapid, with a mean time of 8.5 days from the onset of Wellens syndrome to infarction.

If anterior wall MI occurs, there is the potential for substantial morbidity or mortality. Thus, it is of utmost importance to recognize this pattern early.

Angiography has demonstrated that 100% of patients with Wellens syndrome will have 50% or greater stenosis of the proximal LAD.

Case 6

A 36-year-old woman presents to the ED with sudden onset of left- sided chest pain and mild shortness of breath that began last night. She was able to fall asleep without difficulty but woke up this morning with persistent pain. The pain is worse when she takes a deep breath. She walked up the stairs at home and became very short of breath, which made her come to the ED. Two weeks ago, she took a 7-hour flight from Europe and since then has left-sided calf pain and swelling.

Pulmonary embolism

Symptoms of pulmonary embolism include sharp chest pain (may worsen with inspiration, called "pleuritic"), dyspnea, hypoxemia, syncope, or shock. There may be associated cough or hemoptysis.

Patients with pulmonary embolism may be febrile and have leg swelling or pain, and some patients will report chest wall tenderness.

Common physical examination findings include tachypnea, tachycardia, and hypoxemia.

Pulmonary embolism risk factors include recent surgery, trauma, prolonged immobility, active cancer, estrogens from birth control pills or hormone replacement therapy (particularly when combined with smoking), procoagulant syndromes, or a history of prior pulmonary embolism or deep venous thrombosis.

Clinical decision aids, such as the Well`s and Revised Geneva Scores can risk stratify patients with possible pulmonary embolism.

TABLE 56-5 Wells' Score for Pulmonary Embolism	
Factor	Points*
Suspected deep venous thrombosis	3
Alternative diagnosis less likely than PE	3
Heart rate >100 beats/min	1.5
Prior venous thromboembolism	1.5
Immobilization within prior 4 wk	1.5
Active malignancy	1
Hemoptysis	1

*Risk score interpretation (probability of PE): >6 points = high risk (78.4%); 2–6 points = moderate risk (27.8%); and <2 points = low risk (3.4%).

Source: Adapted with permission from Wells PS, Anderson DR, Rodger M, et al: Derivation of a simple clinical model to categorize patients' probability of pulmonary embolism: increasing the model utility with the SimpliRED D-dimer. *Thromb Haemost* 83: 418, 2000.

TABLE 56-7 Revised Geneva Score for Pulmonary Embolism	
Clinical Variable	Points*
Age >65 y	1
Previous venous thromboembolism	3
Surgery requiring anesthesia or fracture of lower limb in past month	2
Active malignancy	2
Unilateral leg pain	3
Hemoptysis	2
Unilateral leg edema	4
Heart rate	
75–94 beats/min	3
>95 beats/min	5

*Total score of 0–3 indicates low probability, score of 4–10 indicates moderate probability, and score of >10 indicates high probability of pulmonary embolism.

Figure 7

The pulmonary embolism rule-out criteria exclude pulmonary embolism in patients with a low pretest probability without further diagnostic testing.

TABLE 56-4 Pulmonary Embolism Rule-Out Criteria Rule (all nine factors must be present to exclude pulmonary embolism)[32]
Clinical low probability (<15% probability of pulmonary embolism based on gestalt assessment)
Age <50 years
Pulse <100 beats/min during entire stay in ED
Pulse oximetry >94% at near sea level (>92% at altitudes near 5000 feet above sea level)
No hemoptysis
No prior venous thromboembolism history
No surgery or trauma requiring endotracheal or epidural anesthesia within the last 4 weeks
No estrogen use
No unilateral leg swelling, defined as asymmetrical calves on visual inspection with patient's heels raised off the bed

Figure 8

Normal d-dimer testing, in a hemodynamically stable low- to intermediate-risk patient (with a Revised Geneva Criteria Score of 0 to 10) makes pulmonary embolism exceptionally unlikely; in those with higher risk assessment, a negative d-dimer has limited value.
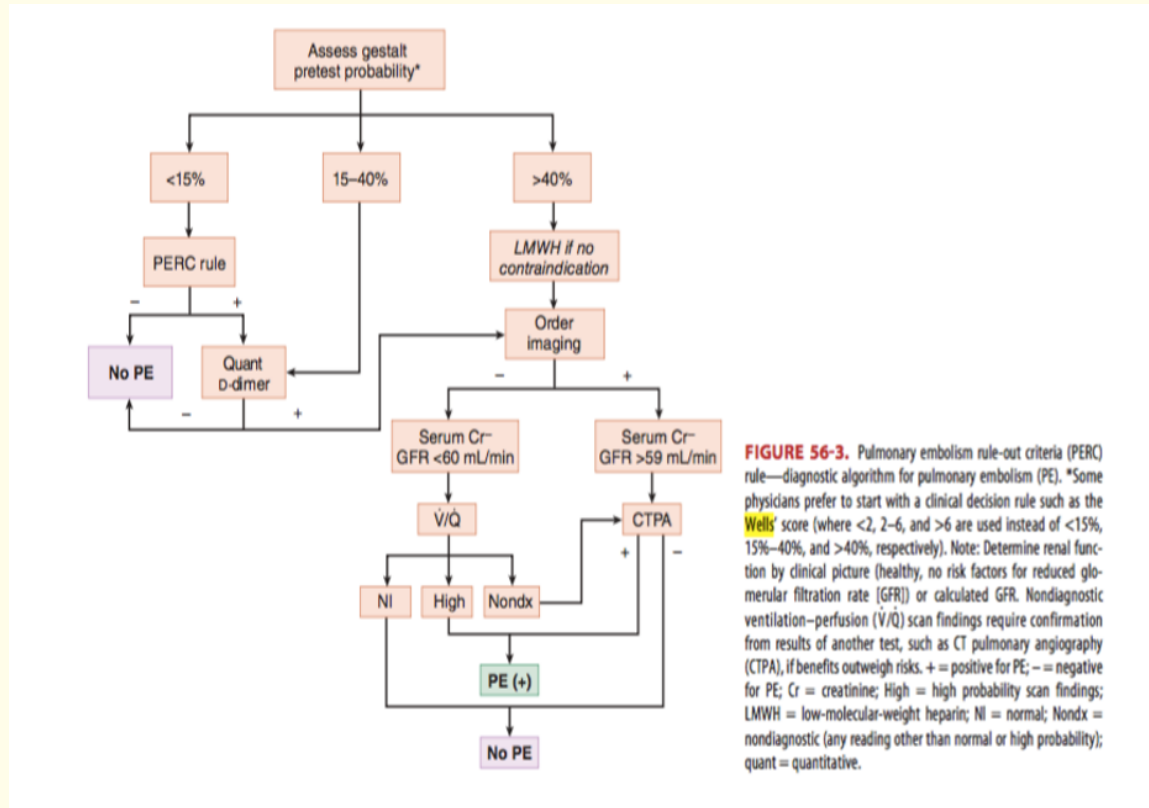

FIGURE 56-3. Pulmonary embolism rule-out criteria (PERC) rule—diagnostic algorithm for pulmonary embolism (PE). *Some physicians prefer to start with a clinical decision rule such as the Wells' score (where <2, 2–6, and >6 are used instead of <15%, 15%–40%, and >40%, respectively). Note: Determine renal function by clinical picture (healthy, no risk factors for reduced glomerular filtration rate [GFR]) or calculated GFR. Nondiagnostic ventilation–perfusion (V̇/Q̇) scan findings require confirmation from results of another test, such as CT pulmonary angiography (CTPA), if benefits outweigh risks. + = positive for PE; − = negative for PE; Cr = creatinine; High = high probability scan findings; LMWH = low-molecular-weight heparin; Nl = normal; Nondx = nondiagnostic (any reading other than normal or high probability); quant = quantitative.

Figure 9

In patients with pulmonary embolism, elevated cardiac troponin (cTn) indicates ventricular dysfunction and identifies patients with an elevated risk of death and complications.

In pulmonary embolism, ECG findings are nonspecific, with the most common finding being sinus tachycardia.

Chest radiographs are usually normal, but in rare cases may show signs of pulmonary infarction.

CT pulmonary angiography is the test of choice and is highly sensitive for the detection of large to medium-sized pulmonary emboli.

Case 7

A 65-year-old man with a history of diabetes and uncontrolled hypertension presents to your ED complaining of crushing chest pain radiating to his back that was worst at onset but continuing until arrival in the ED. An ECG is nondiagnostic and chest x-ray is normal. On physical exam of the patient's pulses you note significant radiofemoral delay.

Aortic dissection

Pain from aortic dissection is classically described as a ripping or tearing sensation radiating to the interscapular area of the back.

The pain is often sudden in onset, maximal at the time of symptom onset, and may migrate or be noted above and below the diaphragm. Lack of sudden-onset pain decreases the probability of aortic dissection but cannot exclude it.

Secondary symptoms of aortic dissection result from arterial branch occlusions and include stroke, acute myocardial infarction, or limb ischemia.

Risk factors include male sex, age over 50 years, poorly controlled hypertension, cocaine or amphetamine use, a bicuspid aortic valve or prior aortic valve replacement, connective tissue disorders (Marfan's syndrome and Ehlers-Danlos syndrome) and pregnancy.

Physical exam findings for aortic dissection lack sensitivity and specificity.

A unilateral pulse deficit of the carotid, radial, or femoral arteries is suggestive of aortic dissection (likelihood ratio 5.7; 95% confidence interval, 1.4 - 23).

Focal neurologic deficits are rare, occurring in only 17% of patients with aortic dissection, but the combination of chest pain and a focal neurologic deficit greatly increase the likelihood of aortic dissection.

While a completely normal chest radiograph lowers the likelihood of aortic dissection being present, it does not exclude dissection.

A negative d-dimer lowers the probability of aortic dissection, but it also cannot exclude the disease.

ECG changes are common among patients with aortic dissection, with up to 40% to 50% presenting with ST-segment or T-wave changes.

Elevated cTn among patients with aortic dissection is associated with increased mortality.

If aortic dissection is suspected, obtain a CT aortogram or transesophageal echocardiogram.
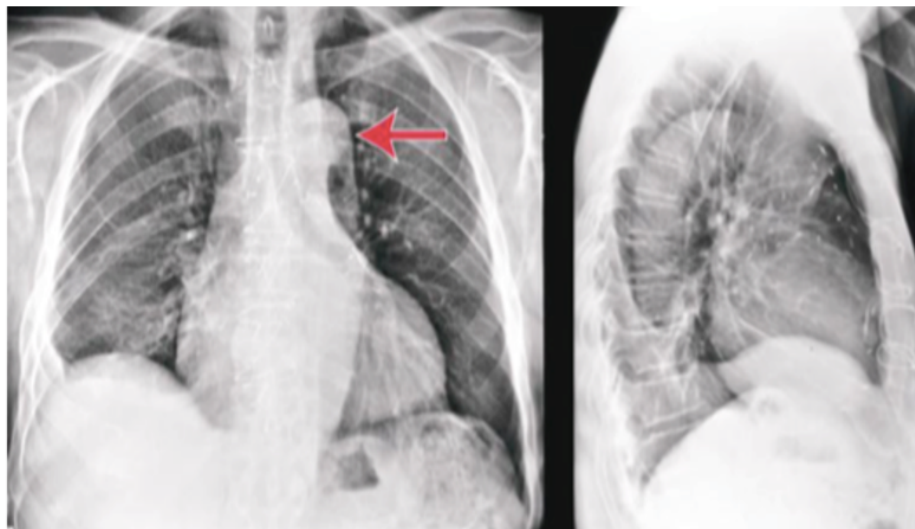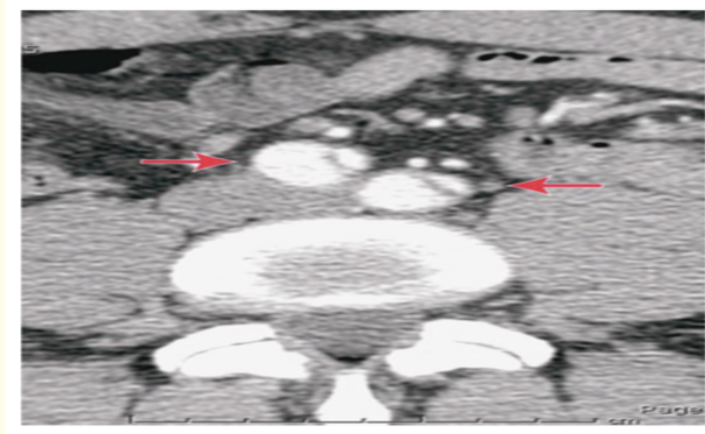
Figure 10

Case 8

A 38-year-old woman with a history of sickle cell disease presents to the ED complaining of 2 days of right-sided chest pain that is worse on inspiration. She had a fever yesterday to 38.5. She feels short of breath and has a cough productive of yellow sputum. She denies leg swelling, hemoptysis, or vomiting. Her vital signs are blood pressure 120/65 mm Hg, pulse 96 beats per minute, respiratory rate 18 breaths per minute, and oxy- gen saturation 94% on room air. Physical exam is remarkable for crackles in her lower right lung field. There is no jugular venous distension, calf swelling, or lower extremity edema. Chest x-ray reveals a new infiltrate in the right lower lobe.

Acute chest syndrome (ACS)

Potentially life-threatening complication of sickle-cell disease can lead to respiratory failure ACS is the leading cause of death among patients with sickle-cell disease.

ACS defined

- Acute complication
- New pulmonary infiltrate on chest X-ray
- Accompanied by at least one other new sign or symptom: fever, chest pain, coughing, wheezing, tachypnea.

Possible causes of ACS

- Fat embolism - More common in adults, diagnosis confirmed with bronchoscopy, can progress to ARDS.
- Infection - Chlamydia pneumoniae, *Mycoplasma pneumoniae*.
- Atelectasis - Secondary to hypoventilation and poor respiratory effort with opioid use.

Diagnostics

- Chest X-ray is cornerstone of diagnosis.
- Hemoglobin levels.
- White blood cell count.
- SpO_2.

Caring for patients with ACS

- Improving oxygenation is first priority; supplemental oxygen may be given (incentive spirometry, nebulizer treatments).
- Administer opioids as ordered for pain; be careful of hindering respiratory effort.
- Continue to assess respiratory, neurologic, and oversedation status.
- Administer antibiotics as ordered.
- Administer I.V. fluids to reverse dehydration and decrease blood viscosity.
- Monitor intake and output to prevent fluid overload, which can worsen pulmonary status.

Case 9

   A 31-year-old kindergarten teacher presents to the ED complaining of acute onset substernal chest pain that is sharp in nature and radiates to her back. The pain is worse when she is lying down on the stretcher and improves when she sits up. She smokes cigarettes occasionally and was told she has borderline diabetes. She denies any recent surgeries or long travel. Her blood pressure is 145/85 mm Hg, heart rate is 99 beats per minute, respiratory rate is 18 breaths per minute and temperature is 38.5. Examination of her chest reveals clear lungs and a friction rub. Her abdomen is soft and her legs are not swollen. Chest radiography and echocardiography are unremarkable. Her ECG is shown below. Which of the following is the most appropriate next step in management?
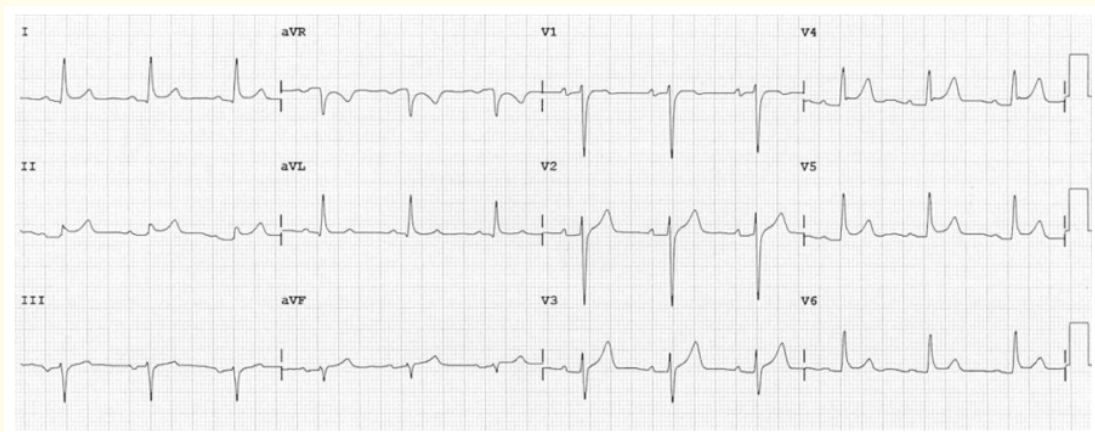

Figure 11

Acute pericarditis

- Pain from acute pericarditis is classically described as a sharp, severe, constant pain with a substernal location.
- The pain may radiate to the back, neck, or shoulders; worsens by lying flat and by inspiration; and is relieved by sitting up and leaning forward.
- A pericardial friction rub is the most specific physical exam finding but is not always evident. The classic ECG findings are diffuse ST-segment elevation with PR depression [1-32].

Conclusion

1. Early recognition of the ECG changes has great impact on the survival rate.
2. Using HEART score help emergency departments risk-stratify chest pain patients: who will have a MACE within in the next 6 weeks and who will not?

3. Well`s and Revised Geneva Scores can risk stratify patients with possible pulmonary embolism

4. Chest X-ray is cornerstone in the diagnosis of ACS.

5. ECG changes are common among patients with aortic dissection, with up to 40% to 50% presenting with ST-segment or T-wave changes.

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