

Evaluation of Patients with Chest Pain

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Overview

- Chest pain risk stratification using the H and P, ECG, labs, and imaging
- Disease Specific Evaluations:
 - ✓ Acute Coronary Syndrome
 - ✓ Pulmonary Embolism
 - ✓ Aortic Dissection
 - ✓ Pneumothorax
 - ✓ Esophageal Rupture
 - ✓ Valvular Heart Disease
 - ✓ Pericardial/Myocardial Disease

Acknowledgement

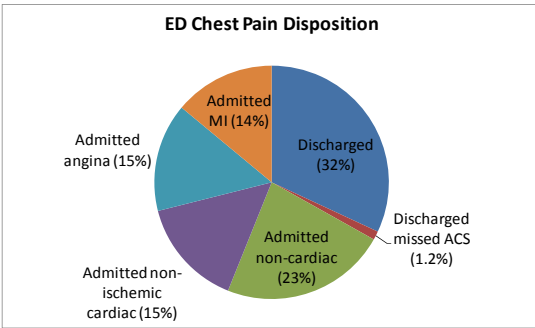
- Mark L. DeBard MD, FACEP
 - ✓ Professor of Emergency Medicine
 - ✓ Attending Emergency Physician, OSU University Hospital East
 - ✓ Co-lecturer with Dr. Moseley for many years on the topic of chest pain evaluation in Emergency Medicine for medical students, residents, and faculty physicians.

The Challenge of Chest Pain

- Whether in the office, ED, or inpatient setting, the patient with chest pain presents a diagnostic dilemma
- You can not treat all patients with chest pain the same due to the broad differential diagnosis that ranges from benign causes to immediately life threatening events
- Must have a strategy that will help identify higher risk patients and differentiate them from lower risk patients

ED Chest Pain Disposition (%)

- 6 million ED Chest Pain Patients (6% of all ED patients)



Chest Pain Evaluation

- Things in common that can be useful for the evaluation of any patient with chest pain regardless of the setting:
 - ✓ History
 - ✓ Physical Exam
 - ✓ ECG
 - ✓ Lab Testing
 - ✓ Imaging

Differential Diagnosis

- Life Threatening
 - ✓ ACS
 - ✓ Pulmonary embolism
 - ✓ Aortic dissection
 - ✓ Tension pneumothorax
 - ✓ Esophageal rupture
 - ✓ Pericardial tamponade
- Non-Life Threatening
 - ✓ Muscular
 - ✓ Skeletal
 - ✓ Pneumonia
 - ✓ Simple pneumothorax
 - ✓ Pericarditis
 - ✓ GERD
 - ✓ Valvular heart disease
 - ✓ Zoster
 - ✓ Radiculopathy

History

- Remains the cornerstone of diagnosis
- The importance of obtaining a thorough chest pain history cannot be over-emphasized

History

- Remains the cornerstone of diagnosis
- Specific characteristics of pain can help with better defining the differential diagnosis:
 - ✓ Chronicity
 - ✓ Onset
 - ✓ Duration
 - ✓ Intensity
 - ✓ Exacerbating factors
 - ✓ Remission/relieving factors
 - ✓ Associated symptoms

History

- Important to ask about associated symptoms that accompany the pain:
 - ✓ Shortness of breath
 - ✓ Nausea and/or vomiting
 - ✓ Diaphoresis
 - ✓ Syncope
 - ✓ Dizziness or weakness
 - ✓ Pain in other locations that might be related

History

- It is helpful to key in on the patient's own description of the pain:
 - ✓ Pain vs. discomfort
 - ✓ Pressure vs. sharp
 - ✓ Intensity at onset
 - ✓ Where is it located
 - ✓ Is the pain positional
 - ✓ What was the patient doing at the time
 - ✓ Worse with exertion
 - ✓ Worse with deep breaths

History

- Does the patient have a previous medical history that is relevant?
 - ✓ Known coronary artery disease
 - Previous re-vascularization?
 - Was there chest pain associated with that prior CAD event?
 - Is the current chest pain similar or different than previous chest pain?
 - ✓ Hypertension
 - ✓ Diabetes
 - ✓ CVA/TIA
 - ✓ Peripheral vascular disease

History

- Risk Factors:
 - ✓ Framingham risk factors (population based)
 - ✓ Connective tissue diseases
 - ✓ High potency stimulant use
- Realize that risk factors only convey a lifetime statistical risk for disease processes:
 - ✓ Presentation with chest pain has already selected for a higher risk group
 - ✓ Don't ignore them totally

History

- Pearls:
 - ✓ Abrupt onset think TAD, PE, PTX
 - ✓ Pleuritic pain think pericarditis, PE, pneumonia
 - ✓ Don't rely on response to therapy like NTG or GI cocktails
 - ✓ Radiation to jaw, arms, or neck increases likelihood of ACS
 - ✓ Consider sudden onset of symptoms, associated syncope or near syncope, or CHF like symptoms associated with the chest pain to be high risk

History

- Avoiding Pitfalls:
 - ✓ Don't discount risk solely based on age, gender, or lack of traditional risk factors
 - ✓ Remember that some groups of patients tend to have atypical presentations:
 - Women
 - Elderly
 - Diabetics
 - ✓ Focus the history on life threats first and then broaden to include less concerning causes

Physical Exam

- Realize that most patients with chest pain will have a normal physical examination
- The physical exam should be used to make diagnosis more likely, not totally exclude a diagnosis:
 - The absence of a single physical exam sign or combination of signs lacks the sensitivity and specificity to exclude disease
- Don't fixate on reproducible chest wall pain!
 - 15% of patients having an active AMI report a tender chest wall

Physical Exam

- Focus on vital signs, general appearance, and positive findings:
 - Clinical signs of CHF (S3/S4, JVD, fluid overload)
 - Friction rub (pericarditis)
 - New onset murmur (aortic dissection, AMI)
 - Hamman's crunch (esophageal rupture)
 - Asymmetric pulses (aortic dissection)
 - Swollen extremities (PE, CHF)
 - Diaphoresis (non-specific, but concerning)

ECG

- You can classify the ECG based on ACC/AHA guidelines:
 - ✓ STEMI
 - ✓ NSTEMI/high risk unstable angina
 - ✓ Non-diagnostic
- The ECG is a slice in time only:
 - ✓ <50% of initial ECGs are diagnostic
 - ✓ Serial ECG's recommended, but timing is unclear
 - ✓ Progression with AMI
 - Hyper-acute T-waves
 - STE
 - T-wave inversion
 - Q waves

High Risk Features to the H and P

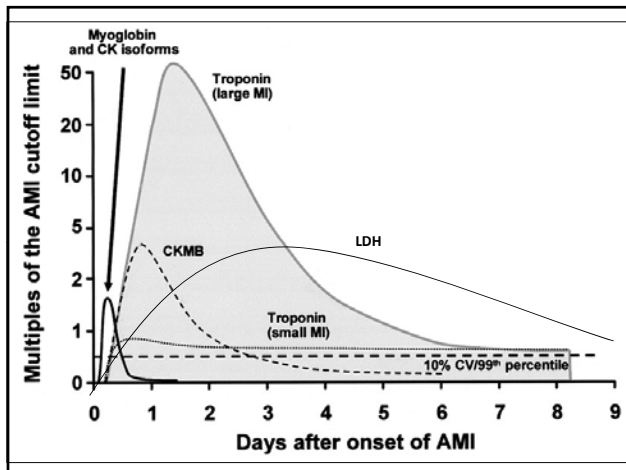
- Sudden onset of pain
- High risk associated symptoms:
 - ✓ SOB, diaphoresis, vomiting, syncope, etc.
- Exertional pain or persistent rest pain
- Abnormal vital signs
- Elderly patients and those with multiple co-morbid conditions

ECG

- If the ECG is abnormal, its important to compare it to an older ECG if available;
 - ✓ Can be critical for conditions like bundle branch blocks (old vs. new) in the setting of new onset chest pain as it helps determine intervention
- In addition to ACS:
 - ✓ ECG changes in PE
 - ✓ ECG in TAD
 - ✓ ECG in pericarditis
 - ✓ Arrhythmias

Lab Testing

- In patients sick enough to come to the ED or be admitted for chest pain, patients usually require:
 - ✓ Basic chemistries
 - ✓ Complete blood count
- Based on the history and physical examination, utilize a targeted approach to laboratory testing:
 - ✓ Coagulation profile (if on coumadin), cardiac markers, d-dimer, BNP, LFT's, Lipase
 - ✓ Tox screen if concern for high potency stimulant use



Lab Testing

- **Cardiac Markers**
 - ✓ Myoglobin
 - First to peak, non-specific
 - Good NPV
 - ✓ CK-MB
 - Former gold standard; supplanted by troponin
 - Elevation in muscle damage, renal failure, sepsis, CVA
 - ✓ Troponin
 - Prolonged elevation in serum (7-14 days)
 - More sensitive and specific for ACS than CKMB; detects micro infarcts
 - Elevated troponin in UA = 9x increase in death due to MACE

Lab Testing

- **BNP:**
 - ✓ Helpful in the assessment of acute shortness of breath of unclear etiology or for assessment of CHF
 - ✓ Know your lab assay
 - ✓ Level <100 = unlikely CHF
 - ✓ Level >500 = likely CHF
 - ✓ Level between 100 and 500 = uncertain
 - Chronic elevation
 - Chronic renal insufficiency
 - Severe COPD with right sided failure
 - PE
 - Elderly women

Lab Testing

- **D-dimer:**
 - ✓ Helpful for the assessment of PE
 - ✓ ELISA test preferred; know your lab assay
 - ✓ Excellent sensitivity, but poor specificity
 - ✓ Need to combine d-dimer testing with some assessment of pre-test probability
 - ✓ Low or moderate pre-test probability and negative ELISA d-dimer, means you have excluded to the limits of testing
 - ✓ If positive d-dimer, need to follow up with further definitive test like VQ scan or CTPA to exclude disease

Imaging

- For most patients that are being assessed for chest pain, the PA and Lateral Chest X-Ray remains quite useful:
 - ✓ Good screening test for many conditions
 - ✓ Assessment of vasculature, lungs, bones, etc.
 - ✓ Can provide alternative diagnoses in some cases
- Be cautious about sensitivity and specificity and using the CXR to “rule out” diagnoses
- Better to utilize to “rule in” based on findings of the examination

Imaging

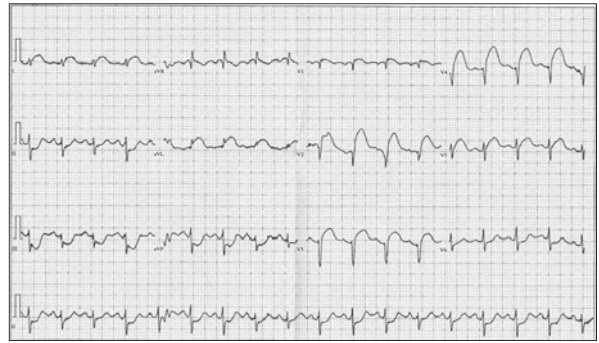
- Let the history and physical examination guide the decision about imaging
- Increasingly many options and difficult to apply the available literature to the patient sitting in front of you:
 - ✓ CT coronary angiogram with calcium scoring
 - ✓ Cardiac MRI with vasodilator stress
 - ✓ 80 lead ECG vest with regional pain mapping

Acute Coronary Syndrome

Acute Coronary Syndrome

- ECG:
 - ✓ You can classify the ECG based on ACC/AHA guidelines:
 - STEMI
 - NSTEMI/high risk unstable angina
 - Non-diagnostic
 - ✓ The ECG is a slice in time only:
 - Serial ECG's recommended, especially if the patient's symptoms change

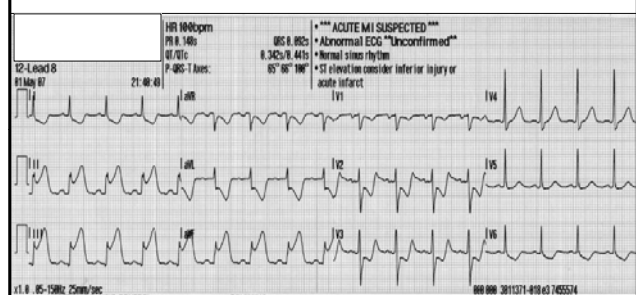
LAD STEMI



Acute Coronary Syndrome

- ECG
 - ✓ Normal ECG does not rule out ACS
 - A circumflex MI may be ECG-"silent"
 - ✓ Review AMI ECGs and anatomic lesions:
 - Anterior = LAD
 - Lateral = Circumflex, diagonal
 - Anterolateral = LAD or Left Main
 - Inferior = RCA (90%), Circumflex (10%)
 - Posterior = Usually RCA; associated with inferior, lateral AMI

Inferior STEMI



Acute Coronary Syndrome

- **LEVEL 1: ST ELEVATION MI (STEMI)**
 - ✓ ST-segment elevation or presumed new LBBB is characterized by ST-segment elevation 1 mm in 2 or more contiguous precordial leads or 2 or more adjacent limb leads.
- **LEVEL 2: HIGH RISK UA/NON-ST ELEVATION MI (NSTEMI)**
 - ✓ Ischemic ST-segment depression 0.5 mm or dynamic T-wave inversion with pain or discomfort. Transient ST-segment elevation 0.5 mm for 20 minutes is also included in this category.
- **LEVEL 3: INTERMEDIATE OR LOW RISK UA**
 - ✓ Normal or non-diagnostic changes in ST segment or T waves are inconclusive and require further risk stratification. This classification includes patients with normal ECGs and those with ST-segment deviation of 0.5 mm or T-wave inversion. Serial cardiac studies (and functional testing) are appropriate.

Acute Coronary Syndrome

- **Cardiac Marker Summary:**
 - ✓ No marker is 100% sensitive all the time
 - ✓ Troponin is the gold standard for AMI diagnosis
 - ✓ Patients with positive markers have distinctly higher all cause morbidity and mortality in the literature
 - ✓ Cardiac marker analysis should not delay coronary intervention in high risk patients (STEMI)

Clinician's Guide to ACS

- Imperative to follow some sort of evidence based strategy for acute coronary syndrome.
- Lots of literature to guide practice, but hard to keep up with.
- Algorithmic approach that standardizes care and minimized practice deviation.



Acute Coronary Syndrome

- **Non-Invasive Testing:**
 - ✓ Numerous studies show the prognostic value of stress testing to help risk stratify chest pain patients
 - Negative stress
 - 2% with MACE at 6 month
 - Positive or inconclusive stress
 - 17% with MACE
 - Combining treadmill or pharmacologic stress with nuclear imaging or echocardiography greatly increases sensitivity.
 - Modality less important than timing of testing
 - Testing is more sensitive if patient actually having symptoms at the time of testing

Acute Coronary Syndrome

- **CT Coronary Angiogram:**
 - ✓ In setting of acute chest pain, pt should be low risk
 - ✓ Non-inferior to stress nuclear imaging in low risk ED chest pain patients:
 - Stress nuclear imaging:
 - Sensitivity=71%, Specificity=90%, NPV=97%
 - Multidetector CT:
 - Sensitivity=86%, Specificity=92%, NPV=99%
 - ✓ What's the physiological significance of lesions it finds?
 - ✓ Radiation exposure and cost for low risk patients?

Acute Coronary Syndrome

- **Treatment:**
 - ✓ Protocol based diagnostic and treatment algorithm:
 - IV, Oxygen, Cardiac Monitor (or call 911)
 - CXR, cardiac markers, other labs
 - ASA (if they have a chest, given them ASA)
 - Chewed, 162-325 mg unless taken
 - Clopidogrel if true ASA allergy

Acute Coronary Syndrome

- **Cardiac Catheterization:**
 - ✓ Considered the gold standard for ACS diagnosis
 - Anatomical and functional assessment
 - Can fix what you find (PCI)
 - Variability in lesion designation (visual assessment)
 - Some centers now using IV ultrasound to look at plaque stability
 - ✓ Complications
 - ATN from IV dye load
 - Bleeding (groin hematoma, RP bleed)
 - Pseudoaneurysm

Acute Coronary Syndrome

- **Treatment**
 - ✓ Nitroglycerin
 - 0.4 mg SL q 5 min X 3
 - IV drip if needed for CP, HTN, CHF
 - Not if BP <90, HR <50, RV MI
 - ✓ Beta Blocker
 - Should be used, but timing?
 - ✓ Morphine
 - Should it be used?
 - ✓ Others
 - ACE, statin important, but not acutely

Acute Coronary Syndrome

- Treatment
 - ✓ Anticoagulation
 - Anticoagulate high risk ACS patients aggressively
 - Don't give high risk therapies to low risk groups
 - Know local practices, but remember that guidelines are increasingly national and expected
 - Positive troponin and ECG changes should get
 - ASA
 - Heparin/LMWH
 - Clopidogrel
 - Glycoprotein IIb/IIIa Inhibitor

Cocaine Associated Chest Pain

- Very common in the ED patient population
- Acute intoxication causes vasospasm, inflammatory mediator release, platelet aggregation
- ACS can result
- Treatment = routine ACS + lorazepam
 - ✓ Exception = concern over use of beta blockers – this may worsen vasoconstriction and hypertension
- Conservative management in most cases

Acute Coronary Syndrome

- ACS Treatment Summary
 - ✓ STEMI
 - ASA, NTG, B-blocker, UFH/LMWH, Clopidogrel, IIb/IIIa Inhibitor
 - Thrombolytics or PCI
 - Disposition = ICU
 - ✓ NSTEMI/High Risk UA
 - ASA, NTG, B-blocker, UFH/LMWH, Clopidogrel, IIb/IIIa Inhibitor
 - Disposition = ICU
 - ✓ Some Risk for UA
 - ASA, NTG, +/- on B-blocker
 - Disposition: Floor telemetry admission vs. short stay unit (depends on local resources)

Pulmonary Embolism

Pulmonary Embolism

- **Diagnostic:**
 - ✓ **Physical exam is unreliable**
 - Hohman's sign
 - ✓ **Symptoms from HPI helpful like dyspnea, rapid heart rate, palpitations, pleurisy, hemoptysis, calf pain, etc.**
 - ✓ **Literature strongly supports some type of structured pre-test probability assessment**
 - Well's Criteria
 - Charlotte Rule

Pulmonary Embolism

- **Diagnostic:**
 - ✓ **DVT**
 - **Duplex Ultrasonography**
 - Non-invasive and first line
 - More sensitive proximally
 - Serial exams necessary?
 - Availability
 - **Venography**
 - Gold standard (does anyone do this?)
 - CTV often combined with CTA

Pulmonary Embolism

- **D-dimer:**
 - ✓ **Helpful for the assessment of PE**
 - ✓ **ELISA test preferred; know your lab assay**
 - ✓ **Excellent sensitivity, but poor specificity**
 - ✓ **Need to combine d-dimer testing with some assessment of pre-test probability**
 - ✓ **Low or moderate pre-test probability and negative ELISA d-dimer, means you have excluded to the limits of testing**
 - ✓ **If positive d-dimer, need to follow up with further definitive test like VQ scan or CTPA to exclude disease**

Pulmonary Embolism

- **Diagnostic:**
 - ✓ **PE**
 - **VQ Scan** – have to do structured PTP assessment
 - PLOPED I
 - Low PTP and normal or very low prob = essentially rules out PE
 - High PTP and high prob = rule in PE
 - Anything else needs further testing
 - **CT scan** – high quality scanner and radiologist
 - PLOPED II
 - Study of choice; often gives alternative diagnosis
 - Literature unclear what to do with negative CTA when you have a high PTP

Pulmonary Embolism

- **Diagnostic:**
 - ✓ PE
 - CT Scan
 - Can increase sensitivity if combining CTA with CT venography
 - Helps define pelvic vein VTE, and can catch proximal femoral DVT
 - Pulmonary Angiography
 - Gold standard
 - Not available
 - Not trivial morbidity

Aortic Dissection

Pulmonary Embolism

- **Treatment:**
 - ✓ Anticoagulate with UFH or LMWH
 - LMWH superior for treatment of DVT
 - Unclear about PE; safe to use, but many still start with UFH
 - ✓ IVC filter to prevent further clot in those with contra-indications to anticoagulation
 - ✓ Thrombolytics or surgical embolectomy for massive PE and hemodynamic instability
 - Immediate consultation with ICU and cardiothoracic surgery if available

Aortic Dissection

- **Path:**
 - ✓ Sheer stress leads to intimal tear
 - ✓ **Risk factors:**
 - Hypertension (most common)
 - Trauma
 - Pregnancy
 - Coarctation
 - Bicuspid AV
 - High potency stimulants
 - Syphilis

Aortic Dissection

Stanford

- Type A
 - ✓ Any involvement of ascending aorta
- Type B
 - ✓ Limited to descending aorta

DeBakey

- Type I
 - ✓ Ascending and descending aorta
- Type II
 - ✓ Ascending only
- Type III
 - ✓ Descending only

Aortic Dissection

• Diagnostic:

- ✓ ECG
 - Beware AMI mimic; beware anticoagulation
- ✓ CXR
 - More helpful in trauma; normal CXR not sensitive enough to rule out the diagnosis
 - Review classic findings in trauma
- ✓ CT scan with IV contrast
 - Study of choice in stable patient

Aortic Dissection

• Clinical:

- ✓ Sudden onset of intense, ripping, tearing pain that often is in the chest radiating to the back or abdomen (suspect when pain above and below diaphragm)
- ✓ Absent pulses, discrepancy in UE/LE BP, ischemic limb
- ✓ New murmur of AR
- ✓ Cardiac tamponade
- ✓ Neuro symptoms like CVA, syncope, cauda equina syndrome
- ✓ Mesenteric ischemia

Aortic Dissection

• Diagnostic:

- ✓ Conventional angiography
 - Gold standard, but not readily available
 - Being supplanted by CTA
- ✓ Echocardiography
 - TTE helpful to rule out complications but not great sensitivity
 - TEE is the test of choice for unstable patient; often done perioperatively
 - Dubious availability even with cardiology back up

Aortic Dissection

- **Treatment:**
 - ✓ Close HD monitoring; arterial line
 - ✓ Control BP (<120 SBP) and HR (<60)
 - Beta blockers are mainstay
 - Prevent sheer stress and further tear
 - Classic combination is esmolol and nitroprusside
 - ✓ Classic dictum of Stanford A managed surgically and Stanford B managed medically
 - Blurred with new stent grafts and PCI?

Pneumothoracic

- Sudden onset of sharp unilateral chest pain
- Traumatic usually obvious by history and part of standard trauma evaluation
- Spontaneous 1:13,000; 6x more common in young males
- Treatment depends on size:
 - ✓ Observation
 - ✓ Small lumen catheters
 - ✓ Heimlich valves
 - ✓ Traditional tube thoracostomy for all larger PTX

Pneumothorax

Esophageal Rupture

Esophageal Rupture (Boerhaave's Syndrome)

- History is usually protracted vomiting, followed by severe chest pain and dysphagia:
 - ✓ Constant pain; usually much worse with swallowing
 - ✓ Dyspnea also common
 - ✓ If late presentation, patients are critically ill
 - ✓ Commonly described in alcoholics
 - ✓ 92% iatrogenic or trauma, 8% spontaneous
- Hammond's Crunch:
 - ✓ cellophane sound of mediastinum due to SQ emphysema
- Pneumomediastinum, Left pleural effusion
- Gastrografin swallow, CT, antibiotics, OR, ICU

Mitral Valve Prolapse

- Path
 - ✓ Familial incidence
 - ✓ Increased risk of dysrhythmia, endocarditis, sudden death
- Clinical
 - ✓ Atypical chest pain and palpitations; advanced disease leads to MVR
- Treatment
 - ✓ Beta blockers help for atypical chest pain and palpitations

Valvular Heart Disease

Aortic Stenosis

- Path
 - ✓ <65 = rheumatic HD and congenital bicuspid
 - ✓ >65 = calcifications
 - ✓ Obstruction of LV outflow track lead to CHF and sudden death
- Clinical
 - ✓ Classic triad of angina, dyspnea, and syncope
 - ✓ Harsh murmur with radiation to carotids
- Treatment
 - ✓ Extreme caution with preload/afterload reduction (get help with acutely decompensated patients)
 - ✓ Valvuloplasty stabilizing, replacement definitive

Pericarditis

Pericarditis

- **Clinical**
 - ✓ Sharp, stabbing, pleuritic CP
 - ✓ Worse with supine, better when sitting up
 - ✓ Friction rub (classic) but usual transient and difficult to auscultate
 - ✓ Viral symptoms (low grade fever, malaise, URI symptoms)
 - ✓ Usually tachycardic

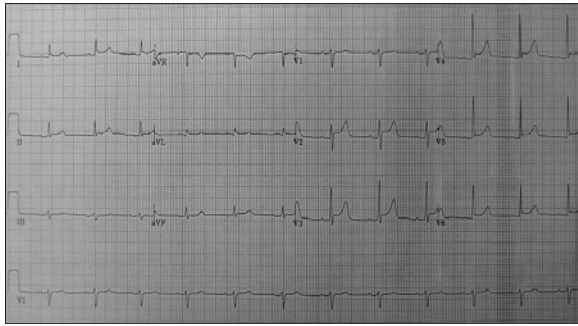
Pericarditis

- **Path**
 - ✓ Idiopathic (most common)
 - ✓ Infectious (viral, TB was classic cause)
 - ✓ Malignancy (lung and breast)
 - ✓ Drug induced (procainamide, hydralazine)
 - ✓ Post radiation
 - ✓ Post MI (Dressler's syndrome)
 - ✓ Uremia
 - ✓ Connective tissues (RA, lupus)

Pericarditis

- **ECG**
 - ✓ Electrical alternans with big effusions
 - ✓ First stage = ST elevation, PR depression
 - ✓ Second stage = ST isoelectric
 - ✓ Third stage = T wave inversion
 - ✓ Fourth stage = resolution
 - ✓ BEWARE OF MIMIC TO AMI!
- **CXR**
 - ✓ Limited value (alternative diagnosis)
 - ✓ With acute effusion can have normal silhouette

Pericarditis



<http://en.wikipedia.org/wiki/File:PericarditisECG.JPG>

Pericarditis

- Treatment
 - ✓ NSAIDs unless contra-indicated
 - ✓ Not steroids in most cases
 - ✓ Recent studies show colchicine helpful
 - ✓ Pericardiocentesis or pericardial window for tamponade
- Disposition
 - ✓ Depends
 - ✓ Classically outpatient management

Pericarditis

- Diagnostic
 - ✓ Echocardiogram
 - Everyone with pericarditis eventually needs this to rule out significant effusion
 - Tamponade is associated with right heart diastolic collapse
 - Remember that tamponade is a clinical diagnosis (muffled heart sounds, hypotension, JVD)
 - ✓ Lab tests
 - Somewhat dependent on suspicion of etiology
 - Cardiac markers? Can have small troponin leak
 - ESR and CRP are non-specific but usually elevated

Myocarditis

- Path
 - ✓ Inflammation of heart muscle
 - ✓ Broad range of etiologies
 - ✓ Most common etiology in U.S. is viral (enteroviruses and echoviruses)
 - ✓ Most common worldwide is parasitic
 - ✓ Often mixed pericarditis and myocarditis picture

Myocarditis

- **Clinical**
 - ✓ Cardiac symptoms 7-14 days following viral illness (URI or GE)
 - ✓ CP reflects pericardial irritation
 - ✓ Fatigue, DOE
 - ✓ Fever, tachycardia out of proportion to fever
 - ✓ CHF and pulmonary edema in severe cases

Myocarditis

- **Treatment**
 - ✓ Primarily supportive and monitoring for complications
 - ✓ Antimicrobials if appropriate for infectious etiology
 - ✓ Steroids, IVIG, and antiviral agents are all controversial
 - ✓ Treatment of heart failure if present
 - ✓ LVAD and cardiac transplantation in severe cases

Myocarditis

- **Diagnostic**
 - ✓ ECG
 - Sinus tach, dysrhythmias, acute LVH
 - ✓ CXR
 - cardiomegaly
 - ✓ Echo
 - Best single test; also evaluates for complications like pericardial effusion
 - Many centers now using cardiac MRI
 - ✓ Lab tests
 - Often elevated cardiac markers
 - ESR and CRP usually elevated, but non-specific

Other Etiologies of Chest Pain

Hypertension

- Significant hypertension in conjunction with hypertensive heart disease can manifest at chest pain
- This is alleviated with reduction of blood pressure
- It is important to exclude co-existing coronary artery disease in these patients as presentation may be very similar
- Patients with HTN and diastolic dysfunction are prone to chest pain when blood pressure is uncontrolled

GERD / Esophagitis

- Things to remember:
 - ✓ Do NOT assume chest pain is from GERD in a patient with a history of GI complaints
 - ✓ Urgent / life-threatening causes MUST be excluded first
 - ✓ Do NOT assume pain relief from Maalox, GI cocktail, etc, correlates to a GI cause of the pain
 - This is similar to reproducible chest pain – it may still be from coronary ischemia / angina

GERD / Esophagitis

- A not entirely uncommon manifestation of GERD is atypical chest pain
- Depending on the person, rather than classic “heartburn,” “chest pain” may be the presenting symptom
- Something to consider once more worrisome chest pain causes are excluded and to clinically correlate with overall presentation
- Consideration for empiric therapy and EGD/GI evaluation

Esophageal Spasm

- This is a somewhat controversial entity in regards to correlation with chest pain
- Occasional correlation between evaluation with manometry and chest pain
- Occasional relief with calcium channel blockers
- This is a difficult diagnosis to evaluate and should be done in conjunction with gastroenterology in the appropriately selected patient population

Costochondritis

- Inflammation of the cartilage at the rib/sternum junction
- More common in women than men
- “Tietze’s syndrome” if significant inflammation at the rib/sternum cartilaginous region with possible overlying erythema
- Diagnosis is clinical with tenderness to palpation seen
- It is important to know that angina can have reproducible chest wall pain and distinguishing these two conditions is imperative
- Treatment is supportive and with NSAIDs