Evaluation of Patients with Chest Pain

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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.
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

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)

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
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

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

- 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

• 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

• 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

**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
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

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)

## 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

- 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

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

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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
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

- 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
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

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

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
LAD STEMI

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.

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

- **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)

### 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

• 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:
  ✓ 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

• 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

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)
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

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

• 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:
  ✓ 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

• Diagnostic:
  ✓ PE
  • VQ Scan – have to do structured PTP assessment
    – PIOPED 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
    – PIOPED 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

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

<table>
<thead>
<tr>
<th>Stanford</th>
<th>DeBakey</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Type A</td>
<td>• Type I</td>
</tr>
<tr>
<td>✓ Any involvement of ascending aorta</td>
<td>✓ Ascending and descending aorta</td>
</tr>
<tr>
<td>• Type B</td>
<td>✓ Ascending only</td>
</tr>
<tr>
<td>✓ Limited to descending aorta</td>
<td>✓ Descending only</td>
</tr>
</tbody>
</table>

### 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:**
- 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

- 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:**
  - Closed HD monitoring; arterial line
  - Control BP (<120 SBP) and HR (<60)
    - Beta blockers are mainstay
    - Prevent shear 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?

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# Pneumothorax
Pneumothoraxic

- 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

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
- Gastrograffin swallow, CT, antibiotics, OR, ICU

Valvular Heart Disease
### 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

### 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

- **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

**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

**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

• 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
### Pericarditis

<table>
<thead>
<tr>
<th>• Treatment</th>
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<tbody>
<tr>
<td>✓ NSAIDs unless contra-indicated</td>
</tr>
<tr>
<td>✓ <strong>Not</strong> steroids in most cases</td>
</tr>
<tr>
<td>✓ Recent studies show colchicine helpful</td>
</tr>
<tr>
<td>✓ Pericardiocentesis or pericardial window for tamponade</td>
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<table>
<thead>
<tr>
<th>• Disposition</th>
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<tr>
<td>✓ Depends</td>
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<tr>
<td>✓ Classically outpatient management</td>
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### Myocarditis

<table>
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<tr>
<th>• Path</th>
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<tr>
<td>✓ Inflammation of heart muscle</td>
</tr>
<tr>
<td>✓ Broad range of etiologies</td>
</tr>
<tr>
<td>✓ Most common etiology in U.S. is viral (enteroviruses and echoviruses)</td>
</tr>
<tr>
<td>✓ Most common worldwide is parasitic</td>
</tr>
<tr>
<td>✓ Often mixed pericarditis and myocarditis picture</td>
</tr>
</tbody>
</table>
### 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

#### 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
### 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

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### 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

- 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
**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

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**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