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

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  - 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)

Differential Diagnosis

<table>
<thead>
<tr>
<th>Life Threatening</th>
<th>Non-Life Threatening</th>
</tr>
</thead>
<tbody>
<tr>
<td>✓ ACS</td>
<td>✓ Muscular</td>
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<tr>
<td>✓ Pulmonary embolism</td>
<td>✓ Skeletal</td>
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<tr>
<td>✓ Aortic dissection</td>
<td>✓ Pneumonia</td>
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<tr>
<td>✓ Tension pneumothorax</td>
<td>✓ Simple pneumothorax</td>
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<tr>
<td>✓ Esophageal rupture</td>
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<tr>
<td>✓ Pericardial tamponade</td>
<td>✓ GERD</td>
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<tr>
<td></td>
<td>✓ Valvular heart disease</td>
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<tr>
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<td>✓ Zoster</td>
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<td>✓ Radiculopathy</td>
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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

- 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

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**LAD STEMI**

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**Inferior STEMI**

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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
**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.

**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)

**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
  - 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
    • Thrombolitics 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

- 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

- 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

- **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

**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

**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

**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

• 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?

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 (Boerhaave’s Syndrome)

• History is usually protracted vomiting, followed by severe chest pain and dysphagia:
  ✓ Constaraft pain; usually much worse with swallowing
  ✓ Dsypnea 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

**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

**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)

**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

- **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

### 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

**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

**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
### 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