Chief Complaint: Chest Pain
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Disclosures
• None

Outline
• Overview of chest pain
• Etiologies
• Obtaining a history
• Physical exam
• Workup
• Testing
Overview

- Chest pain is one of the most common reasons for patients seeking care in the ambulatory and emergency settings
- Accounts for roughly 8 million ER visits annually in the United States
- Typically treated as ACS but only 15-20% of patients actually have ACS, 10% have stable angina
- 1/3-1/2 of patients have musculoskeletal pain, 10-20% have gastrointestinal pain, 5% have respiratory issues
- Diagnosis of ACS estimated to be missed in 2% of patients
- Must be a balance between cost and appropriate workup based on risk and expected results

Meet Our Patients

- Mrs. Jones
  - 51 year old female with diet controlled HTN, positive family history of CAD, not premature
  - Runs 2-3 miles per day
  - Works in an accounting office
- Mr. Smith
  - 41 year old male with no prior history/family history, takes no medications
  - Works out daily including 3 miles of cardio and weight training

Acute Coronary Syndrome (ACS)

- Consists of ST elevation myocardial infarction (STEMI), non-ST elevation myocardial infarction (NSTEMI), and unstable angina (UA)
- Different spectrum from differentiating types of chest pain
Debunking

- Myth #1: positive troponin = NSTEMI/ACS
- Myth #2: chest pain = ACS
- Myth #3: positive troponin = heparin drip

Definitions of Chest Pain/Angina

- Typical chest pain – 1) heavy chest pressure or squeezing, burning feeling or difficulty breathing, 2) increases with exertion or stress, 3) relief with nitroglycerin or rest
- All 3 present to be classified as typical
- 1-2 present for atypical
- 0 present for noncardiac

Anginal Equivalents

- Some patients present with jaw pain, epigastric pain, shoulder pain, nausea, dyspnea
- Women, older patients, and diabetics may have more atypical presentations
Nerves

• Visceral
  – Enter spinal cords at several levels leading to poor localization
  – Includes heart, blood vessels, esophagus, visceral pleura

• Parietal
  – Able to localize stimulus such as pain
  – Includes dermis and parietal pleura

Defining pain

• Pleuritic – sharp pain worsened with breathing movement or coughing
• Epigastric – primary or sole location in the middle or lower abdominal region
• Musculoskeletal – pain reproducible with movement or palpation in specific locations
• Other factors include constant pain (hours to days) and very brief episodes of pain (seconds)

Risk Table

<table>
<thead>
<tr>
<th>Nonanginal</th>
<th>Atypical</th>
<th>Typical</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>Men</td>
<td>Women</td>
</tr>
<tr>
<td>30-39</td>
<td>Very Low</td>
<td>Very Low</td>
</tr>
<tr>
<td>40-49</td>
<td>Intermediate</td>
<td>Very Low</td>
</tr>
<tr>
<td>50-59</td>
<td>Intermediate</td>
<td>Low</td>
</tr>
<tr>
<td>≥60</td>
<td>Intermediate</td>
<td>Intermediate</td>
</tr>
</tbody>
</table>
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Nonischemic Cardiovascular
Aortic dissection*
Myocarditis
Pericarditis

Psychiatric
Affective disorders (ex. Depression)
Anxiety disorders
Thought disorders (ex fixed delusions)

Gastrointestinal
Biliary
Cholangitis
Cholecystitis
Choledocholithiasis
Colic
Esophageal
Esophagitis
Spasm
Reflux
Rupture*
Pancreatitis
Pepitic ulcer disease
Nonperforating or perforating*

Pericarditis

- Visceral and most of parietal pericardium is insensitive to pain
- Pain occurs due to involvement of the pleura
- Pain usually occurs while changing position, breathing (especially deep) and coughing
- Can cause subternal pain mimicking MI
- Central diaphragm involvement manifests as pain in shoulders and neck
- More lateral diaphragm involvement manifests as pain in the upper abdomen and back
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Acute Aortic Dissection

- Sudden onset of excruciating ripping pain
- Location depends on initiation site and direction of dissection
  - Ascending dissection – anterior chest pain
  - Descending dissection – posterior chest/back pain
- Overall, fairly rare
- Tend to occur with risk factors such as pregnancy (ascending), HTN (descending), collagen vascular disease (CVD; ie Marfan, ED), bicuspid aortic valve

Aortic Dissection

Pulmonary Embolism

- Sudden onset dyspnea and pleuritic chest pain
- Incidence is 1/1000, likely underestimate as some are asymptomatic
- Massive PE can cause severe substernal chest pain due to distension of the PA
- Smaller emboli tend to cause pulmonary infarction and irritation of the pleura
- Hemodynamically significant emboli can cause hypotension, syncope and right heart failure
Pulmonary Etiologies

- Usually produce dyspnea and pleuritic symptoms
- Location typically reflects site of disease
- Pneumonia can cause pain over the involved lung
- Pneumothorax usually sudden onset associated with dyspnea
  - Primary occurs in tall, thin, young men
  - Secondary can occur in COPD, asthma, CF
- Asthma exacerbations can cause chest tightness
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Gastrointestinal Etiologies

- GERD – one of the most common mimickers of chest pain
  - Exacerbated by alcohol, aspirin and foods
  - Usually worse when recumbent
  - Relieved when sitting up or use of H2 blockers/PPIs
- Esophageal spasm – can improve with nitroglycerin
- Mallory-Weiss tears/Boerhaave syndrome – can occur with prolonged vomiting
- PUD – onset usually 60-90 minutes after eating and rapidly responds to acid-reducing therapy; usually epigastric
- Pancreatitis – epigastric with radiation to back
- Cholecystitis – crampy, colicky pain in RUQ

“The Others”

- Musculoskeletal etiologies
  - Costochondritis
  - Herpes zoster (shingles)
  - Heavy exercise or labor
- Psychiatric etiologies
  - Panic or anxiety disorders
  - Brought on by stress or environmental triggers

Meet Our Patients

- Mrs. Jones
  - Has complained of off and on pain in the chest for about 3 weeks
  - No exacerbating or remitting factors
  - Has not gotten worse
- Mr. Smith
  - Has complained of pain since this morning
  - Started as he walked back up the driveway in subzero temperatures after getting the newspaper
  - Went away as he sat down to read the paper
  - Wife made him come in
Evaluation

- Usually begins prior to seeing the physician/extender
- Triage of great importance either over the phone or in ED setting
- ACC/AHA suggests immediate assessment in patients with
  - Chest pain
  - Persistent dyspnea
  - Persistent heartburn
  - Pain radiating to jaw, back, shoulder
  - Syncope

Initial Evaluation

- Clinical stability
- Immediate prognosis
- Safety of triage options

History taking

- Define their chest pain
  - Onset
  - Location
  - Quality
  - Duration
  - Radiation
  - Exacerbating factors
  - Remitting factors
  - Associated symptoms
- Have they ever had anything like this before?
  - History tends to repeat itself
Risk Factors

• Smoking
• Hypertension
• Hyperlipidemia
• Lack of physical activity
• Diets high in fat and salt
• Abdominal obesity
• Family history of premature CAD (prior to age 55 in males and 65 in females)
• Previous diagnosis of CAD or PAD

Physical Exam

• General appearance
• Vital signs
  – Blood pressure
  – Heart rate
  – Respiratory rate
• Heart and lung exam
• Assess for signs of extracardiac vascular disease
  – Carotid bruit
  – Peripheral pulses

Vitals

• Blood pressure
  – Hypertension – may elude to presence of dissection
  – Hypotension – may represent right sided heart failure due to RV infarct from MI or failure from PE
  – Discrepancy – aortic dissection
• Heart rate
  – Tachycardia – may represent presence of shock/underfilled state; includes arrhythmia
  – Bradycardia – seen in inferior MIs
• Respiratory rate
  – Tachypnea – sign of heart failure or due to PE
Heart Exam

- Presence of systolic murmur
  - Can represent new mitral regurgitation in MI involving the posterior descending branch of the dominant coronary artery
    - Single blood supply makes it more prone to rupture during infarction
  - Murmur can also represent ventricular septal rupture
  - Echocardiography and right heart catheterization can differentiate

Heart Exam

- Diastolic murmur – aortic insufficiency due to aortic dissection
- Accentuated P2 – PE due to increased pulmonary artery pressure
- Friction rub – pericarditis
  - Post MI rub typically occurs >2 weeks post MI
- Muffled heart sounds
  - Pericardial effusion – primary or due to dissection

Lung Exam

- Rales
  - Bilateral may represent heart failure
  - Unilateral may represent severe mitral regurgitation
- Absent breath sounds – pneumothorax
- Deep breaths – evaluate for pleuritic pain
ECG

- Fastest test to rule out overt ACS (STEMI)
- Those with unstable angina may have a normal ECG
- 1-5% of patients may have a normal ECG upon presentation which may progress during the ER workup
- Recommended to obtain within 10 minutes of hospital arrival
- Pre-hospital ECG very beneficial

Value of ECG Findings

<table>
<thead>
<tr>
<th>ECG Finding</th>
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<tbody>
<tr>
<td>New ST elevation ≥1 mm</td>
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<tr>
<td>New Q wave</td>
</tr>
<tr>
<td>Any ST elevation</td>
</tr>
<tr>
<td>New conduction defect</td>
</tr>
<tr>
<td>New ST depression</td>
</tr>
<tr>
<td>Any Q wave</td>
</tr>
<tr>
<td>Any ST depression</td>
</tr>
<tr>
<td>T wave peaking and/or</td>
</tr>
<tr>
<td>Inversion ≥1 mm</td>
</tr>
<tr>
<td>New T wave inversion</td>
</tr>
<tr>
<td>Any conduction defect</td>
</tr>
</tbody>
</table>

ECG

- ST elevation
  - 1 mm in all leads except V₂ and V₃
  - V₁ and V₃: 2 mm in men ≥40, 2.5 mm in men <40, 1.5 mm in women
- ST depression/T wave changes
  - ≥0.5 mm ST depression in two contiguous leads
  - T wave inversion of 1 mm or more in two contiguous leads
ECG

- Dynamic ST changes
  - Changes occurring during active episodes of chest pain but resolving when pain abates have high predictive value
- Tachycardia
- S1 Q3 T3 – most commonly cited ECG manifestation in PE
  - RBBB, rightward axis, T wave inversions V1-4

ECG

- Diffuse ST elevation – pericarditis
  - Usually has some degree of PR depression
- LBBB no longer considered a STEMI equivalent
  - Studies show that less than ½ of patients with suspected MI and LBBB actually have an MI
  - 2004 guidelines updated in 2013
- Compare to prior!!
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Chest X-ray

- Can show pneumothorax
- Presence of pulmonary edema – heart failure
- Look for widened mediastinum – aortic dissection
- Hampton hump/Westermark sign - PE
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Biomarkers

- Troponin
  - T or I
- Cardiac specific troponin
  - cTnT or cTnI
- CK-MB
- Myoglobin
- CRP
- D-dimer

<table>
<thead>
<tr>
<th>Marker</th>
<th>Initial Rise</th>
<th>Peak</th>
<th>Return to Normal</th>
</tr>
</thead>
<tbody>
<tr>
<td>Troponin</td>
<td>2-4 hours</td>
<td>10-24 hours</td>
<td>5-10 days</td>
</tr>
<tr>
<td>CK-MB</td>
<td>3-4 hours</td>
<td>10-24 hours</td>
<td>2-4 days</td>
</tr>
<tr>
<td>LDH</td>
<td>10 hours</td>
<td>24-72 hours</td>
<td>14 days</td>
</tr>
<tr>
<td>Myoglobin</td>
<td>1-2 hours</td>
<td>4-8 hours</td>
<td>24 hours</td>
</tr>
</tbody>
</table>

Troponin

- Evolved over the years
- Prior troponin assays were not specific for cardiac muscle
- Led to false positive results which, in turn, led to excess use of resources
- Differentiation between T and I
Cardiac Troponin

- Most sensitive and specific biomarker, therefore, preferred for cardiac muscle injury
- False positive findings are rare
- Always signifies some sort of myocardial injury
  - ACS, defibrillation, myocarditis, myocardial contusion, tachyarrhythmia, LV/RV strain, HTN emergency, extreme exercise, transplant rejection, sepsis
- Cleared more slowly in renal dysfunction
  - TnI may be elevated chronically in stage IV/V CKD

Cardiac Troponin

- Studies based on 12 hour sampling
  - >95% sensitive and 90% specific
  - Single sample – 70% sensitive & 75% specific
- Further studies have used a second troponin 3 hours after the initial troponin
  - 96% NPV initial, 99% NPV at 3 hours
- JACC Nov, 2018
  - Generation 5 cardiac troponin T (cTnT)
  - Median time from symptom onset – 10.2 hrs
High Sensitivity cTnT (hs-cTnT)

- JACC Nov. 2018
- Sub-analysis of PROMISE trial
  - Primarily looked at coronary CTA in low to intermediate risk patients with chest pain
- ≥800 symptomatic ambulatory patients
- Found that higher levels of hs-cTnT correlated with higher likelihood of coronary calcification as well as more diffuse and obstructive CAD

High Sensitivity cTnT (hs-cTnT)

- Earlier study investigated hs-cTnT levels in patients who ruled in for NSTEMI/UA
  - NSTEMI: 72% had baseline levels above the 99th percentile, 28% had levels above the limit of detection at baseline
  - UA: 44% had baseline levels above the 99th percentile, 52% had levels above the limit of detection at baseline
- Can correlate better with burden of ischemia

ED Setting

- ¼ of patients had undetectable levels with 100% NPV
- High-sensitivity assays are more quantifiable and may allow for better triage of chest pain patients
- Significance of lower levels will need to be defined for future use
- Mainly used in Europe at this time
Cardiac Troponin Elevation in Patients Without a Specific Diagnosis

- Retrospective sub-analysis of TOTAL-AMI
  - Goal is to study the mechanisms and implications of different subtypes of MI and comorbidities
- Excluded patients with planned admissions, MI within 8 weeks, missing information, and patients who had coronary intervention

- If your troponin was elevated, your risk for future events was higher
- As troponin rises, risk also rises
- Comorbidities added to risk of MAE
  - Lower BMI, diabetes, renal dysfunction, COPD, previous CV disease, malignancies
- Don’t write non-ACS patient’s off
- Plan for close follow up from the ER
Creatine Kinase (CK)

- CK is not specific to cardiac muscle
  - Found in skeletal muscle, tongue, diaphragm, small intestine, uterus and prostate
- CK-MB was the biomarker of choice prior to troponin assays
- Used as a ratio of CK-MB to CK
  - Factors in the skeletal muscle component of CK
  - Disadvantage - CK-MB present in skeletal muscle in conditions such as muscular dystrophy, high performance athletics, rhabdomyolysis

Creatine Kinase (CK)

- Elevations common in ED patients due to higher use of alcohol and trauma
- Shorter half-life in circulation
  - Allows for gauging timing of MI, new or recurrence
- Used less frequently at this point

Other Markers

- Myoglobin
  - Smaller size molecule allowing for more rapid clearing
  - Non-specific to cardiac tissue
- C-reactive protein (CRP)
  - Also non-specific and elevated in a variety of medical conditions
  - May be some implication for high sensitivity CRP (hsCRP) in the future
Other Markers

- Ischemia-modified albumin (IMA)
  - Reduced cobalt binding in the setting of ischemia
  - JACC 2013 – study examined use of IMA prior to angiography to gauge severity of CAD
  - Use still remains unclear

- D-dimer
  - >99% NPV for PE in low risk patient; high risk patient should consider imaging
  - 96% NPV for aortic dissection

Other Markers

- B-type natriuretic peptides (BNP and N-terminal pro-BNP)
  - Released in the setting of increased ventricular wall stress
  - Can rise in setting of transient myocardial ischemia as well
  - Increased levels during ACS correlates with worse prognosis

Emerging Biomarkers

- Growth differentiation factor 15 (GDF-15)
- TGF-β1 cytokine released from cardiomyocytes after ischemia and reperfusion injury

- Heart type fatty acid binding protein
- Cytoprotective protein involved in intracellular uptake and buffering of free fatty acids in myocytes

- Myoperoxidase
- Hemeprotein released during degranulation of neutrophils and some monocytes

- Pregnancy-associated plasma protein A
- Matrix metalloproteinase abundantly expressed in eroded and ruptured plaque but absent in stable plaque

- Phosphatidyl ethanolamine
- VESG marker that is strongly upregulated in plaque primary inflammatory instigator of plaque instability

- Secretory phospholipase A2
- Mediates phospholipid hydrolysis to generate lysophospholipids and fatty acids

- Interleukin 6
- Stimulates synthesis of CRP

- Chemokine ligand 5 and ligand 18
- Mediators of monocyte recruitment induced by ischemia
Next step

• Do our patients need to be managed in the outpatient/ED/inpatient settings?
Developed a decade ago in the Netherlands

Proven to be a safe way to triage chest pain in the ER setting

Outpatient management

- Echocardiography
- Stress testing
  - Exercise only testing
  - Exercise or pharmacologic testing with echo or nuclear imaging
- Computed tomography (CT)
  - Calcium score
  - Cardiac CTA

Ischemic Cascade
Echocardiography

- Assess for systolic and diastolic dysfunction
- Assess for pericardial effusion/enhancement
- Assess valvular function
- Ancillary findings such as pleural effusion/aortic dilatation

Stress Testing Modalities

Stress testing

- Non-imaging
  - Exercise

- Imaging
  - Echo
  - CT-PET
  - Nuclear
    - Regadenoson
    - Adenosine
    - Dobutamine

Stress Echo

- Very good specificity
- Exercise or dobutamine
- Almost always approved by insurance
- No radiation
Nuclear Stress

- Exercise
  - Coronary dilatation at 2-3x normal
  - Allows assessment of functional capacity, heart rate and blood pressure response and electrical changes

- Pharmacological
  - Allows for maximum coronary dilatation at 4-6x normal
  - Side effects from vasodilator agents
Coronary Calcium Score

- Typically used in asymptomatic, lower risk patients
- Detects stable plaques which contain diffuse amounts of calcium
- Interpretation based on age, sex, ethnicity, and standard cardiac risk factors
- Increased risk for CAD
  - 2-fold for scores up to 100
  - 10-fold for scores over 1000
- Appropriate use criteria support the use in patients with:
  - Intermediate level of CHD risk (10% to 20% over 10 years)
  - Young patients with a low to intermediate risk (6% to 10% over 10 years)
  - Low-risk patients with a family history of premature CHD

Cardiac CTA

- Can be used to rapidly assess for ACS or unstable plaque at risk for rupture
  - Positive vessel remodeling
  - Low-attenuation plaque with high lipid content
- Sensitivity of 87% to 99% and specificity of 93% to 96%
- Improved since initial trials with use of dual-sources and retrospective gating
Cardiac CTA

- PROMISE trial – CTA (anatomical testing) equivocal to functional testing in low to intermediate risk patients
- Optimal test for low to intermediate risk patients
  - Low calcium burden
- Assessment for patency of bypass grafts
  - Minimal motion
  - Large size

What do we do with our patients??

- Mrs. Jones
- Mr. Smith

Teasing It All Out

- Sudden onset and severe
  - ACS, PE, aortic dissection, pneumothorax
- Pleuritic pain
  - PE, pericarditis, pneumothorax, MSK
- Improved with nitroglycerin
  - ACS, esophageal spasm
- Patients with low probability of ACS should have as little workup as safely possible to avoid unnecessary tests, hospitalizations, procedures, and complications
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