APPROACH TO CHEST PAIN

BY

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EMERGENCY PHYSICIAN
OUTLINES

• Introduction
• Initial approach
• Diagnosis
• Life-threatening causes of chest pain
• Take home messages
INTRODUCTION

- Approximately 6 million visits per year to ED in US
- 2nd most common complaint in ED
INITIAL APPROACH

- Ensure pt is stable
  - Airway, Breathing, Circulation (ABC’s)
- IV access, oxygen therapy, cardiac & vital signs monitoring
- ECG
- Analgesia
Focused HISTORY

- Chest pain characteristic
  - Quality, location, radiation, size of area or distribution, severity, time of onset, duration, first occurrence, frequency, is it similar to previous cardiac ischemic episodes
- Precipitating or aggravating factors
  - Pleuritic, positional, palpable, exercise, emotional stress, relieving factors
- Associated sx
  - SOB, diaphoresis, vomiting, heart burn
- Past medical hx, past surgical hx, family hx, social hx
PHYSICAL EXAMINATION

• General appearance & vitals
• Neck: distended jugular vein, bruits, crepitus/ s/c emphysema
• Chest exm:
  • Inspection (scars, heaves, tachypnea, work of breathing)
  • Palpation (tenderness, apex beat, crepitus)
  • Percussion (dullness, resonance)
  • Auscultation (murmurs, gallops, breath sounds, air entry, crepitation)
• Extremities: swelling/edema, pulses, tenderness
• Abdomen
# Differential Diagnosis

<table>
<thead>
<tr>
<th>Category</th>
<th>Conditions</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiovascular</td>
<td>AMI, Acute coronary ischemia, Aortic dissection, Cardiac tamponade, UA, Coronary spasm, Prinzmetal’s angina, Cocaine induced, Pericarditis, Myocarditis, Valvular heart disease, Aortic stenosis, Mitral valve prolapse, Hypertrophic cardiomyopathy</td>
</tr>
<tr>
<td>Pulmonary</td>
<td>Pulmonary embolism, Tension pneumothorax, Pneumothorax, Mediastinitis, Pneumonia, Pleuritis, Tumor, Pneumomediastinum</td>
</tr>
<tr>
<td>Gastrointestinal</td>
<td>Esophageal rupture(Boerhaave), Esophageal tear(Mallory-Weiss), Cholecystitis, Pancreatitis, Esophageal spasm, Esophageal reflux, Peptic ulcer, Biliary colic</td>
</tr>
<tr>
<td>Musculoskeletal</td>
<td>Muscle strain, Rib fracture, Arthritis, Tumor, Costochondritis, Nonspecific chest wall pain</td>
</tr>
<tr>
<td>Neurologic</td>
<td>Spinal root compression, Thoracic outlet syndrome, Herpes zoster, Postherpetic neuralgia</td>
</tr>
<tr>
<td>Other</td>
<td>Psychologic, Hyperventilation</td>
</tr>
</tbody>
</table>
INVESTIGATION

- Blood investigation (cardiac enzymes, D-dimer, BNP, blood gas)
- ECG
- CXR
- POCUS
- CT angiogram
LIFE-THREATENING CAUSES OF CHEST PAIN

• Acute Coronary Syndromes
• Pulmonary Embolism
• Aortic Dissection
• Tension Pneumothorax
• Cardiac Tamponade
• Esophageal Rupture (Boerhaave’s syndrome)
ACUTE CORONARY SYNDROMES (ACS)

• 2 million people admitted annually for workup of ACS
• STEMI mortality in Malaysia
  • In-hospital mortality: 10.6%
  • 30-day mortality: 12.3%
  • 1-year mortality: 17.9%
Clinical spectrum of ACS.*

- MI is diagnosed by the rise and/or fall in cardiac troponins, with at least one value above the 99th percentile of the upper reference limits (URL), and accompanied with at least one of the following:
  - Clinical history consistent with chest pain of ischaemic origin.
  - ECG changes
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality.
  - Identification of an intracoronary (IC) thrombus by angiography or autopsy.
Exm:
- skin: diaphoretic, pale, cool
- CV: tachyc/ arrhythmia/ murmur/ JV distension
- Lungs: ± crepitation
- Extr: ± edema

Acute Myocardial Infarction

- squeezing/ pressure
  - radiation to shoulders, arms, jaw
- nausea
- diaphoresis
- lightheadedness
- worse with exertion
- improved with rest
<table>
<thead>
<tr>
<th>Location</th>
<th>Leads</th>
<th>ECG findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>Anteroseptal</td>
<td>V1 – V3</td>
<td>• ST elevation in leads V2-3:</td>
</tr>
<tr>
<td></td>
<td></td>
<td>≥ 0.25 mV (in males &lt; 40 years),</td>
</tr>
<tr>
<td></td>
<td></td>
<td>≥ 0.2 mV (in males ≥ 40 years),</td>
</tr>
<tr>
<td></td>
<td></td>
<td>≥ 0.15 mV in females,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Q wave</td>
</tr>
<tr>
<td>Extensive anterior</td>
<td>V1 – V6</td>
<td>• ST elevation of ≥ 0.1 mV in all leads except leads V2-V3. In leads V2-3 :</td>
</tr>
<tr>
<td></td>
<td></td>
<td>≥ 0.25 mV (in males &lt; 40 years),</td>
</tr>
<tr>
<td></td>
<td></td>
<td>≥ 0.2 mV (in males ≥ 40 years),</td>
</tr>
<tr>
<td></td>
<td></td>
<td>≥ 0.15 mV in females,</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Q wave</td>
</tr>
<tr>
<td>Posterior</td>
<td>V7 – V8</td>
<td>• ST elevation ≥ 0.05 mV (≥ 0.1 mV in men &lt; 40 years),</td>
</tr>
<tr>
<td></td>
<td></td>
<td>• Q wave</td>
</tr>
<tr>
<td>Posterior</td>
<td>V1 – V2</td>
<td>• ST depression, Tall R wave</td>
</tr>
<tr>
<td>Anterolateral</td>
<td>I, AVL, V5 – V6</td>
<td>• ST elevation ST elevation of ≥ 0.1 mV, Q wave</td>
</tr>
<tr>
<td>Inferior</td>
<td>II, III, AVF</td>
<td>• ST elevation ST elevation of ≥ 0.1 mV, Q wave</td>
</tr>
<tr>
<td>Right Ventricular</td>
<td>V4R</td>
<td>• ST elevation ≥ 0.5 mm (≥ 1 mm in men &lt; 30 years old).</td>
</tr>
<tr>
<td>(RV)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Short axis (SA)

Anterior
Antero-septal
Infero-septal
Infero-lateral
Inferior
Antero-lateral
Figure 2: Time Course of Elevation of Serum Cardiac Biomarkers after STEMI

**Flowchart 1**

**CHEST PAIN / CHEST PAIN EQUIVALENT**

- Continuous ECG monitoring
- Sublingual glyceryl trinitrate (GTN) (if no contraindication)
- Aspirin + Clopidogrel #
- Analgesia
- Oxygen [if oxygen saturation (SpO₂) < 95%]

**Assessment for reperfusion:**

**Onset of symptoms:**

- **< 3 hours**
  - Primary PCI** or Fibrinolytic Therapy
- **3-12 hours**
  - Primary PCI**
- **> 12 hours**
  - Medical Therapy ± Antithrombotics

**Preferred option:**

- Fibrinolytics
- Primary PCI

**Second option:**

- Consider PCI within 3-24 hours if fibrinolytics are administered as part of the pharmaco-invasive strategy
- PCI if ongoing ischaemia or haemodynamic instability

**Concomitant Therapy:**

- Anti-platelet Therapy (DAPT)
- Statin
- β-blockers
- ACE-Ils/ ARBs
- MRA

*When clinically indicated

**Preferred option in:**
- high-risk patients
- presence of contraindications to fibrinolytic therapy and/or
- if the anticipated time intervals/transport times are within that stated in Flow Chart 2.

# or ticagrelor or prasugrel (after angiogram)
PULMONARY EMBOLISM

• Epidemiology
  • VTE
    • 3rd most frequent cardiovascular disease
    • Incidence: 100-200 per 100,000 general population annually
Increased RV afterload

RV dilatation
TV insufficiency
RV wall tension
Neurohormonal activation
Myocardial inflammation
RV O₂ demand
RV ischaemia

Cardiogenic shock
Death

RV O₂ delivery
RV coronary perfusion
Systemic BP
Low CO
LV pre-load
RV output
RV contractility

BP = blood pressure; CO = cardiac output; LV = left ventricular; RV = right ventricular; TV = tricuspid valve.
Pulmonary Embolism

Exm:
- V/S: HTN/ hypotension, tachyc, hypoxic
- CV: JV distension, tachyc
- Lungs: Normal lungs finding
- Extr: unilateral leg swelling

- Dyspnea
- Pleuritic chest pain
- cough
- Substernal chest pain
- Fever
- Hemoptysis
- syncope
- Unilateral leg pain/swelling
<table>
<thead>
<tr>
<th>Predisposing factors for VTE</th>
<th>Strong risk factors (odd ratio &gt; 10)</th>
<th>Moderate risk factors (odd ratio 2-9)</th>
<th>Weak risk factors (odd ratio &lt; 2)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>- # of lower limb</td>
<td>- Arthroscopic knee surgery</td>
<td>- Bed rest &gt; 3/7</td>
</tr>
<tr>
<td></td>
<td>- Hospitalization for HF or AF/flutter (within previous 3/12)</td>
<td>- Auto-immune diseases</td>
<td>- HTN/ DM</td>
</tr>
<tr>
<td></td>
<td>- Hip/knee replacement</td>
<td>- Blood transfusion</td>
<td>- Immobility dt sitting (eg: prolonged car or air travel)</td>
</tr>
<tr>
<td></td>
<td>- Major trauma</td>
<td>- Central venous lines</td>
<td>- Increasing age</td>
</tr>
<tr>
<td></td>
<td>- MI (within previous 3/12)</td>
<td>- Chemotherapy</td>
<td>- Laparoscopic surgery</td>
</tr>
<tr>
<td></td>
<td>- Previous VTE</td>
<td>- CHF/ resp failure</td>
<td>- Obesity</td>
</tr>
<tr>
<td></td>
<td>- Spinal cord injury</td>
<td>- Erythropoiesis-stimulating agents</td>
<td>- Pregnancy</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- HRT</td>
<td>- Varicose veins</td>
</tr>
<tr>
<td></td>
<td></td>
<td>- In vitro fertilization</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- Infection</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>- IBD, cancer, postpartum period, thrombophilia</td>
<td></td>
</tr>
</tbody>
</table>
ECG:
- Non-specific ST segment & T wave changes (50%)
- Sinus tachycardia (44%)
- RV strain pattern (34%)
- RBBB (18%)
- RAD (16%)
- Atrial tachyarrhythmias (8%)
CXR:
- Hampton’s hump (20%)
- Westermark sign: regional oligaemia (10%)
- Fleishner sign: Enlarged pulmonary artery (20%)
- Pleural effusion (35%)
- Normal
CT = computed tomographic; PE = pulmonary embolism.

a Two alternative classification schemes may be used for clinical probability assessment, i.e. a three-level scheme (clinical probability defined as low, intermediate, or high) or a two-level scheme (PE unlikely or PE likely). When using a moderately sensitive assay, D-dimer measurement should be restricted to patients with low clinical probability or a PE-unlikely classification, while highly sensitive assays may also be used in patients with intermediate clinical probability of PE. Note that plasma D-dimer measurement is of limited use in suspected PE in hospitalized patients.

b Treatment refers to anticoagulation treatment for PE.

c CT angiogram is considered diagnostic of PE if it shows PE at the segmental or more proximal level.

d In case of a negative CT angiogram in patients with high clinical probability, further investigation may be considered before withholding PE-specific treatment.
### Clinical prediction rules for pulmonary embolism

<table>
<thead>
<tr>
<th>Wells rule</th>
<th>Clinical decision rule points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Previous PE or DVT</td>
<td>Original version: 1.5</td>
</tr>
<tr>
<td></td>
<td>Simplified version: 1</td>
</tr>
<tr>
<td>Heart rate ≥ 100 b.p.m.</td>
<td>Original version: 1.5</td>
</tr>
<tr>
<td></td>
<td>Simplified version: 1</td>
</tr>
<tr>
<td>Surgery or immobilization within the past 4 weeks</td>
<td>Original version: 1.5</td>
</tr>
<tr>
<td></td>
<td>Simplified version: 1</td>
</tr>
<tr>
<td>Haemoptysis</td>
<td>1</td>
</tr>
<tr>
<td>Active cancer</td>
<td>1</td>
</tr>
<tr>
<td>Clinical signs of DVT</td>
<td>Original version: 3</td>
</tr>
<tr>
<td></td>
<td>Simplified version: 1</td>
</tr>
<tr>
<td>Alternative diagnosis less likely than PE</td>
<td>Original version: 3</td>
</tr>
<tr>
<td></td>
<td>Simplified version: 1</td>
</tr>
</tbody>
</table>

### Clinical probability

**Three-level score**

<table>
<thead>
<tr>
<th>Low</th>
<th>0–1</th>
<th>N/A</th>
</tr>
</thead>
<tbody>
<tr>
<td>Intermediate</td>
<td>2–6</td>
<td>N/A</td>
</tr>
<tr>
<td>High</td>
<td>≥ 7</td>
<td>N/A</td>
</tr>
</tbody>
</table>

**Two-level score**

<table>
<thead>
<tr>
<th>PE unlikely</th>
<th>0–4</th>
<th>0–1</th>
</tr>
</thead>
<tbody>
<tr>
<td>PE likely</td>
<td>≥ 5</td>
<td>≥ 2</td>
</tr>
<tr>
<td>Revised Geneva score</td>
<td>0</td>
<td>1</td>
</tr>
<tr>
<td>-------------------------------------------------</td>
<td>-----</td>
<td>-----</td>
</tr>
<tr>
<td>Previous DVT or PE</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Heart rate 75–94 b.p.m., ≥95 b.p.m.</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Surgery or fracture within the past month</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Haemoptysis</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Active cancer</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>Unilateral lower limb pain</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>Pain on lower limb deep venous palpation and unilateral oedema</td>
<td>4</td>
<td>1</td>
</tr>
<tr>
<td>Age &gt; 65 years</td>
<td>1</td>
<td>1</td>
</tr>
</tbody>
</table>

**Clinical probability**

**Three-level score**

<table>
<thead>
<tr>
<th></th>
<th>Low</th>
<th>Intermediate</th>
<th>High</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0–3</td>
<td>4–10</td>
<td>≥11</td>
</tr>
</tbody>
</table>

**Two-level score**

<table>
<thead>
<tr>
<th></th>
<th>PE unlikely</th>
<th>PE likely</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0–5</td>
<td>≥6</td>
</tr>
</tbody>
</table>

b.p.m. = beats per minute; DVT = deep vein thrombosis; N/A = not available; PE = pulmonary embolism.
Suspected PE with shock or hypotension

CT angiography immediately available

No

Echocardiography

RV overload

No

Search for other causes of haemodynamic instability

No other test available or patient unstable

CT angiography available and patient stabilized

Positive

PE-specific treatment: primary reperfusion

Search for other causes of haemodynamic instability

CT angiography

Yes

CT angiography

CT = computed tomographic (pulmonary angiography);
PE = pulmonary embolism; RV = right ventricular.

a Includes the cases in which the patient's condition is so critical that it only allows bedside diagnostic tests.
b Apart from the diagnosis of RV dysfunction, bedside transthoracic echocardiography may, in some cases, directly confirm PE by visualizing mobile thrombi in the right heart chambers. Ancillary bedside imaging tests include transoesophageal echocardiography which may detect emboli in the pulmonary artery and its main branches, and bilateral compression venous ultrasonography which may confirm deep vein thrombosis and thus be of help in emergency management decisions.
c Thrombolysis; alternatively, surgical embolectomy or catheter-directed treatment.
Aortic Dissection

- Mortality rate: 1-2% per hour
- Classic tearing interscapular pain: 50% of pt
- 5-15% no pain (present with syncope, stroke, CCF, elderly)

Risk factors:
- HTN, CTD, chest trauma, vasculitis, iatrogenic

Pathophysiology:
The aortic dissections originate with an intimal tear in:

1. Ascending aorta (65%)
2. Aortic arch (10%)
3. Descending thoracic aorta (20%)
4. No evidence of tear (13%)

Blood penetrates the intima and enters the media layer.
Aortic Dissection

Exam:
- V/S: HTN, tachyC
- Skin: diaphoretic, pale, cool
- CV: tachyC, murmur (AR), ± cardiac tamponade, MI
- Abd: pulsatile mass, bruising, distension
- Extr: unequal pulses, weakness
Investigation

- ECG: nonspecific changes
- Erect CXR:
- TTE/TOE
- CTA
### Figure 13 Classification of acute aortic syndromes (AAS)

<table>
<thead>
<tr>
<th>De Bakey</th>
<th>Type I</th>
<th>Type II</th>
<th>Type III</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stanford</td>
<td>Type A</td>
<td>Type A</td>
<td>Type B</td>
</tr>
</tbody>
</table>

Diagram showing different types of acute aortic syndromes (AAS) with arrows indicating blood flow directions.
Diagnostic approach for Acute Aortic Syndrome

- STEMI can be associated with AAS in rare cases.
- Pending local availability, patient characteristics, and physician experience.
- Proof of type-A AD by the presence of flap, aortic regurgitation, and/or pericardial effusion.
- Preferably point-of-care, otherwise classical.
- Also troponin to detect non-ST-segment elevation myocardial infarction.
<table>
<thead>
<tr>
<th>High-risk conditions</th>
<th>High-risk pain features</th>
<th>High-risk examination features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Marfan syndrome (or other connective tissue diseases)</td>
<td>Chest, back, or abdominal pain described as any of the following:</td>
<td>Evidence of perfusion deficit:</td>
</tr>
<tr>
<td>Family history of aortic disease</td>
<td>- abrupt onset</td>
<td>- pulse deficit</td>
</tr>
<tr>
<td>Known aortic valve disease</td>
<td>- severe intensity</td>
<td>- systolic blood pressure difference</td>
</tr>
<tr>
<td>Known thoracic aortic aneurysm</td>
<td>- ripping or tearing</td>
<td>- focal neurological deficit (in conjunction with pain)</td>
</tr>
<tr>
<td>Previous aortic manipulation (including cardiac surgery)</td>
<td></td>
<td>Aortic diastolic murmur (new and with pain)</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Hypotension or shock</td>
</tr>
</tbody>
</table>

Table modified from Rogers AM et al Circulation 2011;123:2213-8.
Risk score varies from 0–3 according the number of positive categories (1 point per column).
Management

- Supportive mx:
  - Pain mx: analgesia
  - BP & HR control: beta-blocker (e.g., labetolol)

- Definitive treatment depends upon the type of dissection
  - Type A: open surgery
  - Type B: medically

Assessed the presence of cx of dissection process:
- Persistent intractable pain
- Rapidly expanding aortic diameter
- Development of a periaortic or mediastinal hematoma
- Malperfusion of a branch vessel organs
- Malperfusion due to aortic lumen compression


Tension pneumothorax

Risk factors:
- Spontaneous (tall, thin man),
- sec to underlying ds (COPD, asthma),
- penetrating trauma
Tension Pneumothorax

Exm:
- V/S: hypotension, tachycardia, hypoxia
- Skin: diaphoretic, pale, cool
- CV: JV distension, tachycardia
- Lungs: unequal/ decreased BS, hyperresonance
CXR
Pneumothorax US algorithm

- Lung sliding → NO
- B-lines → NO
- YES → YES
- Lung point → YES
- NO → No pneumothorax
- YES → Lung pulse
- No pneumothorax → NO
- YES → Pneumothorax
• Rx
  • Supportive: O2 supplement
  • Definitive:
    • Needle thoracocentesis
    • Chest tube insertion
Cardiac Tamponade

Causes:
- Chest trauma, pericarditis, myocardial rupture, cancer, kidney failure, CTD, hypothyroidism, Cx of cardiac surgery
Cardiac Tamponade

Exm:
- V/S: Hypotension, tachyC, narrow PP, pulsus paradoxus
- CV: tachyC, muffled heart sound, JV distension

Beck’s triad of cardiac tamponade:
- Hypotension
- JV distension
- Muffled HS

- sharp
- L sided
- pleuritic
- positional
- associated SOB
- recent fever/illness

Myocarditis / pericarditis / pericardial effusion
Treatment

- Pericardiocentesis

Other approach:
- Apical
- Parasternal
Esophageal Rupture (Boerhaave’s syndrome)

- Spontaneous perforation of the esophagus after rapid intraesophageal pressure increase (e.g., severe vomiting/retching)
- > 90% cases, rupture occurs in the distal 3rd of esophagus
- High mortality
- a/w gastroduodenal ulcers & alcoholism
Esophageal Rupture

**Exm:**
- V/S: hypotension, tachycardia
- Lungs: ± ronchi/crepitus
- Abd: epigastric pain, peritoneal signs

Mackler’s triad:
- Vomiting
- Retrosternal chest pain
- s/c emphysema

- sharp
- radiation to abdomen
- associated SOB
- after vomiting
- bloody emesis
Investigation:
- CXR
- Esophagram
- CT scan

Classic CXR:
- s/c emphysema
- Widened mediastinum
- Pneumomediastinum
- Pneumothorax
- pneumoperitoneum
- Pleural effusion (usually left side)
• RX:
  • Surgical repair or endoscopic stenting
  • Broad-spectrum antibiotics (e.g.: gentamicin plus metronidazole)
  • Fluid resuscitation

Complications:
- Acute mediastinitis
- Esophagopleural fistula
- Pneumonia
- Empyema
- Sepsis
Approach to chest pain algorithm

- Assess vital signs
  - Cardiac monitor, IV access, O2
  - Focused history and physical exam

Stable patient

Obtain 12 lead ECG and CXR
- Administer ASA (if patient low risk for aortic dissection)

ECG diagnostic or suggestive of ACS
- Yes
  - ECG diagnostic or suggestive of ACS AND Patient low risk for aortic dissection
  - Assess vital signs
  - Cardiac monitor, IV access, O2
  - Stabilize airway, breathing and circulation
  - Treat arrhythmias according to ACLS protocols
  - Check for presence of life-threatening chest pain diagnosis: AMI, Massive PE, Tension pneumothorax, Pericardial tamponade

- No
  - CXR diagnostic
    - Yes
      - CXR shows pneumothorax
        - Treatment based on symptoms and size of pneumothorax
        - Emergent thoracic surgery consultation
        - Blood pressure control
        - Confirmatory imaging (CT, MRI, echocardiography)
        - NT-proBNP
        - Cardiology consultation
    - No
      - CXR shows infiltrate or evidence of HF
      - History and exam consistent with CXR
        - Appropriate management
        - Use scoring system to assess pretest probability for pulmonary embolism
        - Obtain cardiac markers
          - Risk stratify for ACS
          - Cardiac markers positive
            - Anticoagulation and appropriate ACS medications
            - Confirm alternate diagnosis OR Age <40 yrs and low cardiac risk
            - History, exam, and ECG diagnostic or suggestive of pericarditis or pericardial tamponade
              - Bedside ultrasound
                - Appropriate treatment
          - Cardiac markers negative
            - No
              - Further testing based upon patient risk
        - Cardiac markers negative
          - No
            - Outpatient evaluation
              - Admit telemetry for rule out myocardial infarction
              - Further testing based upon patient risk
      - CXR nondiagnostic
        - Obtain cardiac markers
          - Risk stratify for ACS
          - Cardiac markers positive
            - Anticoagulation and appropriate ACS medications
            - Confirm alternate diagnosis OR Age <40 yrs and low cardiac risk
            - History, exam, and ECG diagnostic or suggestive of pericarditis or pericardial tamponade
              - Bedside ultrasound
                - Appropriate treatment
          - Cardiac markers negative
            - Yes
              - Treat alternate diagnosis
              - Outpatient evaluation
            - No
              - Further testing based upon patient risk
TAKE HOME MESSAGES

• ABC’s first
• Think multi systems involvement
• Always rule out life-threatening chest pain
THANK YOU