OBJECTIVES

- To effectively assess and identify life threatening causes of chest pain
- To undertake appropriate investigations where required
- To commence appropriate treatment without delay
- To recognize complications and seek experienced help when necessary

Chest Pain

- 5% of ED visits
  - 5 million pts/yr
- Accurate diagnosis remains a challenge

Chest Pain Definitions

- Acute Chest Pain:
  - Acute - sudden or recent onset (usually within minutes to hours), presenting typically <24 hrs
  - Chest - thorax midaxillary to midaxillary line, xiphoid to suprasternum notch
  - Pain - noxious uncomfortable sensation
    - Ache or discomfort

Initial Approach

- Triage
  - Chest pain
  - Significant abnormal pulse
  - Abnormal blood pressure
  - Dyspnea
  - These pts need IV, O2, Monitor, ECG
Initial Approach

• Evaluation:
  – Airway
  – Breathing
  – Circulation
  – Vital Signs
  – Focused exam
    • Cardiac, pulmonary, vascular

Initial Approach

• History:
  – Character of pain
  – Presence of associated symptoms
  – Cardiopulmonary history
  – Pain intensity, 0-10 pain

Initial Approach

• Secondary exam:
  – History
    • Quality, radiation/migration, severity, onset, duration, frequency, progression and provoking or relieving factors of pain
  – Risk factors
  – Physical exam
  – Review old records/ECG

Categorizing Chest Pain

1. Chest Wall Pain
   • Sharp, Precisely localized
   • Reproducible: Palpation, movement

2. Pleuritic or Respiratory CP
   • Somatic pain, Sharp
   • Worse with breathing/coughing

3. Visceral CP
   • Poorly localized, aching, heaviness

Causes

1. Chest wall
   • Costochondritis
   • Precordial catch synd
   • Slipping Rib Synd
   • Radicular Synd
   • Intercostal Nerve
   • Fibromyalgia

2. Pleuritic
   • Pulmonary Embolism
   • Pneumonia
   • Spontaneous pneumo
   • Pericarditis
   • Pleurisy

3. Visceral Pain:
   • Typical Exertional Angina
   • Atypical Angina
   • Unstable Angina
   • Acute Myocardial Infarction (AMI)
   • Aortic Dissection
   • Pericarditis
   • Esophageal Reflux or spasm
   • Esophageal Rupture
   • Mitral Valve Prolapse
Categorizing Chest Pain
Assessment of Risk Factors
• CAD:
  – Cigarette Smoking
  – Diabetes
  – Hypertension
  – Hypercholesterolemia
  – Family History

• Aortic Dissection:
  – Middle Aged
  – Male
  – Hypertension
  – Marfan Syndrome

Categorizing Chest Pain
Assessment of Risk Factors
• Pulmonary Embolism
  – Hypercoagulable Diathesis
  – Malignancy
  – Recent Immobilization
  – Recent Surgery

Chest pain incidentals ACS
• AMI Rare under 30 y/o
  – except with cocaine use
• GI cocktail may cause relief even in AMI
• Nitroglycerin can cause relief of esophagus spasm, biliary colic, and AMI
• NSAIDS can be analgesic for all types of pain

Atypical Chest Pain
• Dyspnea at rest, DOE
• Discomfort: shoulder, jaw, arm
• Nausea, Epigastric pain
• Lightheadedness, Generalized weakness
• MS changes
• Diaphoreosis
• Atypicals usually in
  – DM, females, non-white, elderly, altered MS pts

Differential Dx
Acute Coronary Syndrome (ACS)
Spectrum of clinical presentations due to:
1. atherosclerotic plaque rupture
2. thrombus formation
3. vessel occlusion
4. distal embolism
• ACS = AMI or Unstable Angina
• Visceral chest pain pts
  – AMI – 15%  UA – 25-30%
Differential Dx
Acute Coronary Syndrome (ACS)

1. STEMI
2. NSTEMI
3. UNSTABLE ANGINA/(VARIANT ANGINA)
   - ECG is the most useful test
   - Incidence
     - Significant ST elevation = 80% are AMI
     - ST depression/T wave inversion = 20% are AMI
     - No change <4% are AMI

Myocardial Ischemia:
- Retrosternal, diffuse, heaviness, or pressure
- Radiation to neck or arm
- Usually persistent pain >20 min, severe
- Associated Sx: Dyspnea, Diaphoresis, Nausea
- May even be Reproducible

Exertional Angina:
- Episodic pain, <10 min
- Onset with exertion
- Resolves with rest, sublingual NTG
- Response to exertion and rest follows same pattern

Atypical Angina:
- Occurs at rest
- Coronary spasm
- Pattern of episodes same

Unstable Angina (UA):
- Change in the pattern of angina
  - New Onset
  - More frequent, severe, easily provoked
  - More difficult to relieve
  - Occurs at rest, lasting >20 min
  - High risk of AMI
Immediate Management and Treatment of all Acute Coronary Syndromes

- A,B,C,D,E approach
- "MONA"
  - Morphine
  - Oxygen
  - Nitrates
  - Aspirin (300 mg orally)

Differential Dx ACS

- Pulmonary Embolism:
  - Atypical, presenting with any combination of:
    - Chest Pain, Dyspnea, Syncope, Shock, Hypoxia
    - Fever, cough, hemoptosis
  - Pain is often pleural
    - Reproducible with breathing, palpation
  - Classic presentation:
    - Sharp pain, Dyspnea
    - Tachypnea, tachycardia, hypoxemia

- Aortic Dissection:
  - Risk Factors – Atherosclerosis, HTN (uncontrolled), Coarctation of Aorta, Bicuspid Aortic Valve, Aortic Stenosis, Marfan Syn, Ehlers-Danlos Syn, Pregnancy
  - Pain – midline Substernal CP, tearing, ripping, searing, radiating to interscapular area
  - Pain Above AND Below Diaphragm
  - Often assoc. with stroke, AMI, limb ischemia

- Spontaneous Pneumothorax:
  - Risks:
    - Sudden Change in barometric pressure
    - Smokers, COPD, Idiopathic Bleb DZ
  - Pain:
    - sudden, sharp, pleuritic chest pain, and dyspnea
  - Dx:
    - Absence of breath sounds ipsilaterally
    - Hyper resonance to percussion
    - CXR – Dx simple pneumo

- Esophageal Rupture (Boerhaave Syn):
  - Life-threatening
  - Substernal, sharp CP
  - Sudden onset after forceful vomiting
  - Dyspneic, diaphoretic, and ill-appearing
  - CXR: Normal, SQ air, Pleural Effusions, Pneumothorax, pneumoperitoneum, pneumomediastinum
  - Water Soluble Contrast Study
Differential Dx ACS

- **Acute Pericarditis:**
  - Acute, sharp, severe, constant, substernal CP
  - Radiation to back, neck, shoulders
  - **Worse** with lying down and inspiration
  - **Relief** with leaning forward
  - **FRICION RUB**
  - ECG: ST segment elev., T wave inversion, or PR depression

Differential Dx ACS

- **Pneumonia:**
  - Sharp and Pleuritic
  - Fever, cough, hypoxia
  - Rales, decreased breath sounds, etc.
  - CXR

Differential Dx ACS

- **Mitral Valve Prolapse:**
  - Women > Men
  - Discomfort at rest
  - Assoc. Sx:
    - Dizziness, Hyperventilation, Anxiety, Depression, Palpitations, Fatigue, SVT, Ventricular Dysrhythmia
  - Tx: Beta-Adrenergic Blockers
  - Dx: Echo

Differential Dx ACS

- **Musculoskeletal/Chest Wall Disorders:**
  - LOCALIZED, Sharp, positional CP
  - Reproducible
  - Types –
    - Costochondritis, Tietze Syndrome
    - Xiphodynia

Differential Dx ACS

- **GI Disorders: GERD/dyspepsia**
  - burning, gnawing low CP
  - Acidic taste
  - Recumbent position increases pain
  - Relief per antacids
    - CAREFUL, can also help in ACS

Differential Dx ACS

- **Esophageal Spasm:**
  - Sudden onset, dull, tight, gripping
  - Hot or cold liquids
  - Large food bolus
  - Responds to NTG
Differential Dx ACS

• Peptic Ulcer Disease:
  – Gastric:
    • Postprandial, dull, boring pain
    • Midepigastic, may awake pt.
  – Duodenal Ulcer:
    • Relieved after eating
    • Symptomatic Tx: antacids
    • DDx: Pancreatitis and Biliary tract Dz

• Panic Disorder:
  – Recurrent, Unexpected panic
  – Including at least 4 SX:
    • Palpitations, diaphoresis, tremor, dyspnea,
      choking, CP, nausea, dizziness,
      derealization, or depersonalization, fear of
      losing control or dying, paresthesias, chills,
      hot flashes
  – Rule out substance abuse

Testing for ACS

• ECGs
• Serum Markers
• Imaging studies
  – AHA Guidlines:
    • Any pt with Ischemic type pain is to have
      an ECG done within 10 minutes of
      arrival.
    • This is to be handed directly to the
      physician

Testing for ACS - EKG

• AMI PT ECGs:
  – 50% = ST elevation > 1mm in 2 contiguous
    leads
  – 20-30% = new ST seg. changes or T wave
    inversion
  – 10-20% = ST depression and T wave
    inversions Similar to previous ECGs
  – 10% nonspecific changes
  – 1-5% will have NORMAL initial ECG

Testing for ACS - Serum Markers

• CK
  – Elevates 4-8 hours after coronary Art. Occlusion
  – Peaks = 12 to 24 hours
  – Nml = 3 to 4 days
• CK-MB
  – Detectable 4-8 hrs
  – Peak = before 24 hrs
  – Nml = in 48hrs
• CK-MB normally can be 5% of total CK (Rapid
  Index)
Testing for ACS - Serum Markers

- Common Causes of CK-MB Elevation:
  - UA, ACS
  - Inflammatory Heart Dz
  - Cardiomyopathies
  - Shock
  - Cardiac Surgery/ Trauma
  - Trauma
  - Dermatomyositis
  - Myopathic Disorders

Testing for ACS - Serum Markers

Myoglobin: Abnormal in 80 – 100% AMI pts

- Small protein in striated and cardiac muscle, released in cell disruption
  - In AMI
    - Rises within 3 hours
    - Peak at 4 to 9 hours
    - Baseline at 24 hours
  - Except in trauma pts, renal pts, and cocaine users myoglobin can be as sensitive as CK-MB and Troponins

Testing for ACS - Troponins

- AMI: Cardiac Troponin I (cTnI) and cTnT
  - Elevates in 6 hrs
  - peaks in 12 h
  - Remain elevated for 7 to 10 days
  - Higher specificity than CK-MB
  - Controversy = Troponins are found to be elevated in Renal Failure pts without proof of ACS/AMI

Testing for ACS - Serum Markers

- Using Myoglobin, CK-MB, and cTnI initially and at 3 hours = 90% of AMI pts diagnosed
- New Bedside cardiac marker tests are now available with results in less than 20 minutes
- Overall value of this remains to be determined

Testing for ACS

Prognosis Categorization Strategy

1. AMI = Immediate Revascularization candidate
2. Probable acute Ischemia: High risk
   (Any of the following)
   Clinical Instability
   Ongoing pain
   Pain at rest with ischemic ECG changes
   Positive cardiac marker(s)
   Positive perfusion imaging study
Testing for ACS
Prognosis Categorization Strategy

3. Possible acute Ischemia: Intermed. Risk:
   
   * **Hx suggestive of ischemia with…**
     * Rest pain, now resolved
     * New onset of pain
     * Crescendo pattern of pain
     * Ischemic pattern on EKG without CP

4. A. Probably NOT Ischemia: low risk
   
   * **Requires all of following**
     * Hx not strong for ischemia
     * ECG normal, unchanged from previous, or nonspecific changes
     * Negative markers

4.B. Stable Angina Pectoris: low risk Pt
   
   * **Requires all the following**
     * > 2wk unchanged Sx pattern,
     * Longstanding Sx with only mild change in exertional pain threshold
     * ECG normal, unchanged, nonspecific changes
     * Negative initial myocardial markers

5. Definitely not ischemia: very low risk for adverse events
   
   * **Requires All**
     * Clear objective evidence of nonischemic Sx etiology
     * ECG normal, unchanged, nonspecific
     * Negative Initial Markers

Testing for ACS - Echo

- Noninvasive, dynamic nature
- Can assess cardiac function, aortic dissection, pericardial pathology, valvular dz, possibly PE
- Normal Echo during CP theoretically excludes ischemia, however false positives and false negatives make it unreliable to rule out ACS

Testing for ACS
Prognosis Categorization Strategy

4. B. Stable Angina Pectoris: low risk Pt
   
   * **Requires all the following**
     * > 2wk unchanged Sx pattern,
     * Longstanding Sx with only mild change in exertional pain threshold
     * ECG normal, unchanged, nonspecific changes
     * Negative initial myocardial markers

5. Definitely not ischemia: very low risk for adverse events
   
   * **Requires All**
     * Clear objective evidence of nonischemic Sx etiology
     * ECG normal, unchanged, nonspecific
     * Negative Initial Markers
Testing for ACS

- Perfusion Imaging allows us to see the uptake and function of the cardiac muscle as the isotope is taken up by functioning muscle and not by damaged muscle.

Chest Pain That Can Kill

- Acute Coronary Syndromes
- Pulmonary Embolism
- Aortic Dissection
- Esophageal Rupture
- Pneumothorax
- Pneumonia

Various others: Pulmonary HTN, Myocarditis, Tamponade

Benign Causes

- Musculoskeletal
- Esophagitis
- Bronchitis (Chest Pain secondary to cough)
- Recently placed nipple rings
- “Non-Specific Chest Pain” *

*Most common – means we don’t know, but it is not going to hurt you.

History matters!

- Location: Central, left, or right
- Associated symptoms: SOB, sweating, nausea
- Timing: Gradual or sudden onset
- Provocation: What makes worse or better?
- Quality: Visceral vs somatic
- Radiation: Back, neck, arm
- Severity: Scale of 1-10

The Rest of the History

What can you get out of the pt in 4 min

- **PMH** – Duh
- **Meds** – Cardiac meds? Nitro? ASA? Plavix? Coumadin?
- **Allergies** – Always important!
- **Social** – Smoker? Alcoholic? Cocaine?
- **Family** – Sudden Death? Early MI? DVT? PE?

Key Emergency Physical

What can you exam in only 2 min.

- **General Appearance**
- **Vital Signs**
- **Heart** (Muffled? Regular? Fast?)
- **Lungs** (Equal? Wet? Tympanic?)
- **Neck** (JVD?)
- **Abdomen** (Distention?)
- **LE** (Edema? calf tenderness?)
This guy is rushed back by EMS, what do you do?

Approach to Chest Pain

INITIAL GOAL in ED is to identify life threats
  – MI, PE, aortic dissection

Remember ABCs always first

First 60 seconds

• How does the pt look?
• What are the pt’s vital signs?
• EMS story?

Next 5 Minutes

• Brief History
• Brief Physical (ABCs)
• What are 2 bedside tests that can be done to help stratify the pt?
  – ECG
  – Portable CXR
• What is an important and cheap medication you should consider?
  – ASA (More on this later)

Next 10 Minutes

• Patient already stabilized, initial data gathered, and initial orders submitted
• Secondary survey: More detailed history and physical exam
• Address patient’s pain
• Goal now is to categorize patient
  1) Chest wall pain- Musculoskeletal
  2) Pleuritic chest pain- Respiratory
  3) Visceral chest pain- Cardiac

Case 1

• 46 yo M with DM, HTN, CAD and MI 1 year ago says “I think I am having a heart attack.”
Case 1 - ACS

- ECG – This will differentiate what you must do now. (Specific but not sensitive)
  - ST elevation in 2 contiguous leads: STEMI
  - New LBBB
  - Ischemia/strain: ST depressions, new T wave inversions, Q waves
  - Nonspecific: T wave flattening/inversions or Q waves without old EKG

Case 1 - ACS

- CXR – To look for failure and evaluate for other cause of chest pain
- Cardiac Enzymes

Cardiac Markers

Commonly measured markers of myocardial damage include:
- Troponin T or I
- Creatine kinase (CK)
- Lactate dehydrogenase (LDH)
- Aspartate transaminase (AST)

Cardiac markers are of no value in making a decision regarding thrombolysis, as even the “negative” markers may be undetectable for the first 6-12 hours after the infarction.

PE Diagnosis

- Symptoms
  - SOB or dyspnea- Present in 90%
  - Chest pain (pleuritic)- 66% of patients with PE
  - Cough
  - Sudden onset
- Signs
  - Tachycardia > 100 beats per minute
  - Tachypnea > 20 breaths per minute
  - Hypoxia < 95% on RA (no other cause)
  - Lower extremity swelling

Case 2

- 30 yo M had an ORIF of ankle fx 2 weeks ago, c/o sudden onset of chest pain.
Pulmonary Embolus Risk Factors

- Hypercoaguability
  - Malignancy, pregnancy, estrogen use, factor V Leiden, protein C/S deficiency
- Venous stasis
  - Bedrest > 48 hours, recent hospitalization, long distance travel
- Venous injury
  - Recent trauma or surgery

PE Treatment

- IV fluid to maintain blood pressure
- Heparin (Will limit propagation but does not dissolve clot)
  - Unfractionated: 80 u/kg bolus, 18 h/kg/hr
  - Fractionated (Lovenox): 1 mg/kg SC BID
- Fibrinolytics
  - Consider with large if pt is unstable
  - No study has shown survival benefit, but very difficult to study.
  - Alteplase 50–100 mg infused over 2–6 hrs, (bolus in severe shock)

Case 3

- 35 yo M with sudden ripping pain radiating to back.

Aortic Dissection

- Blood violates aortic intimal and adventitial layers
- False lumen is created
- Dissection may extend proximally, distally, or in both directions

PULMONARY EMBOLUS

- Examination/ECG/CXR/ABG often unhelpful unless large
- D-dimer
- Analgesia/Oxygen/Heparin
- Refer for V/Q scan or CT angiography
- Consider bolus thrombolytic if suspected and patient is hypotensive or arrests

Aortic Dissection

- Bimodal distribution
  - Young: Connective tissue (Marfan) or pregnancy
  - Older: Most commonly > 50 (mean age 63)
- Risk factors
  - Male: 66% of patients
  - Hypertension: 72% of patients
  - Connective tissue disease
    - 30% of Marfan’s patients get dissections
  - Cocaine Use
  - Syphilis
Aortic Dissection

- Presentation (Difficult clinical diagnosis)
  - 85% have chest or back pain
  - “Ripping” or “tearing” in 50%
  - Neurologic symptoms in 20%
  - Hematuria
  - Asymmetric pulses

Aortic Dissection Diagnosis

- CXR - Widened mediastinum, abnormal aortic knob, pleural effusions
  - Not sensitive (25% have wide mediastinums)
- Chest CT - Very sensitive and specific
  - Quickly obtained
  - Must think about kidney + contrast
- Angiography - Gold standard
  - Most reliable anatomy of dissection

AORTIC DISSECTION

Aortic Management

- Involve CT surgery early
- Blood pressure control
  - Goal SBP 120-130 mmHg
  - Beta blockers are first line (Labetalol and Esmolol)
  - Can add vasodilators i.e. nitroprusside
- Admission to ICU
  - Ascending dissections will need surgery
  - If dissection is only descending, management is only medical

Case 4

- 55 yo alcoholic with persistant vomiting presents with sudden onset of CP followed by hematemesis.
Case 5

- 18 yo healthy male was lifting weights when he had sudden onset of sharp CP + SOB.
- HR 122, RR 34, BP 70/P, Sat 88%
- Decreased breath sounds on left.

Needle Decompression

Case 6

PERICARDITIS

- DIAGNOSED ON HISTORY/EXAMINATION/ECG
- MOST COMMONLY VIRAL OR POST MI
- PAIN RELIEF WITH SIMPLE ANALGESIA/NSAID
- ADMIT FOR ECHOCARDIOGRAPHY/MONITORING

MANAGEMENT OF CHEST PAIN

- Focussed Assessment
- Early Diagnosis
- Simultaneous Treatment
- Necessary Investigations Only
- MULTIDISCIPLINARY EXPERTISE AVAILABLE!
Questions

1. T or F 100% of AMI pts will have a change on EKG.
2. How long after coronary artery occlusion does the CK-MB become detectable?
   a. 2-4 hrs
   b. 4-8 hrs
   c. 8-12 hrs
   d. 24 hrs

3. Common Causes of CK-MB elevation include all the following except:
   a) Acute Coronary Syndrome
   b) Muscular Dystrophy
   c) Cardiomyopathies
   d) Delirium Tremens
   e) Speaking in front of Dr. XYZ

4. On day number 5 following coronary ischemia, which serum marker(s) will still be elevated?
   a. Myoglobin
   b. CK-MB
   c. Troponin I
   d. CK

• 5. Chest pain units have shown no real value in eliminating missed MIs or unnecessary admissions to rule out ACS.
  – TRUE OR FALSE

Answers

1. False, 1-5% are normal
2. B. 4-8 hrs
3. E
4. C - CK takes 3-4 days to return to normal, Trop. I 7-10 days, CK-MB 48 hrs, Myoglobin 24 hrs
5. False – actually CP units help avoid missed MIs, yet are able to discharge 82% after a set obs period and serial markers and EKG’s

Thank You!

Questions?