## Demystifying Acute Coronary Syndromes

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## **Disclosure of Financial Relationships**

- I have no affiliation or financial interest to disclose.
- This talk is my opinion and in no way represents the policy or opinions of the White House, US Army, Army Futures Command or the Department of Defense.

#### Before we start: White House PAs



#### President Biden



#### President Trump & First Lady





#### President Obama





#### Case Study (#1)

- A 29-year-old Caucasian male with history of acute lymphoblastic leukemia (ALL) treated with full body radiation, marrow transplant and chemotherapy 12 years ago, presents to the ER with acute chest pain and SOB that began 3.5 hours prior to arrival, while delivering food. He then walked into the local CVS to check his blood pressure, which was "150s/90s and he is diaphoretic.
- What should we do?

## **Terminal Learning Objective**

For a patient with a suspected or known cardiac condition interpret historical data, physical examination findings, laboratory and procedural results, in order to formulate a differential diagnosis, diagnosis and treatment plan in accordance with current medical protocols and standards of care

## **Enabling Learning Objective**

Formulate a differential diagnosis, evaluation plan, and treatment plan for **the acute coronary syndromes** based upon interpretation of patient history, physical exam findings, and diagnostic studies.

# Key Learning Objectives

- Define the various acute coronary syndromes including unstable angina, non-ST elevation myocardial infarction, and ST elevation myocardial infarction
- Describe the pathophysiologic elements underlying acute coronary syndromes including plaque rupture and endothelial dysfunction
- Formulate a diagnostic evaluation plan for patients with acute coronary syndromes
- Formulate a treatment plan for patients with acute coronary syndromes
- Describe who to refer for revascularization in the management of acute coronary syndromes

# Epidemiology

**Chest Pain Statistics** 

- ED Physicians miss 2% of MI's
  - Accounts for 20% of malpractice loss
- Aortic dissection=approx. 1 per 100,000
  - Mortality exceeding 90% if missed
- PE=650,000 deaths annually
  - 400,000 missed
- Tension Pneumothorax=2.5-18 per 100,000
- Esophageal Rupture=12.5 per 100,000
- Pericarditis diagnosis made every 1000 admissions
- Anxiety or Panic Disorder: Common ~ Diagnosis of Exclusion

Define acute coronary syndromes including unstable angina, non-ST elevation myocardial infarction, and ST elevation myocardial infarction

## Terminology

- Ischemic Heart Disease
  - Represents a spectrum from chronic stable angina to acute MI.
- <u>Acute Coronary Syndrome</u>
  - Are life threatening conditions that can occur at any time in patients with coronary artery disease
- Angina
  - Chest pain from decreased blood flow (O2 and nutrients) to cardiac tissue. **Reversible**
- Infarction
  - Chest pain from necrosis of cardiac tissue from complete blockage of blood flow. **Nonreversible**

#### What is Acute Coronary Syndrome?



### **Causes: Coronary Thrombus**

Non atherosclerotic causes:

- Severe anemia
- Severe hypotension
- Aortic stenosis
- Aortic dissection
- Coronary artery spasm (Variant or Prinzmetal's angina)
- Procedural complication (i.e. coronary artery dissection following catheterization)
- Embolic phenomena
- Blunt chest wall trauma

### The Heart and ACS

- Acute Coronary Syndrome
  - Clinically defined
    - UA NSTEMI STEMI
    - Diagnosis based on history and diagnostics
  - Pathologically by amount of damage
    - Transmural- entire thickness of myocardial wall
    - Subendocardial only affects the innermost layer

Formulate a diagnostic evaluation plan for patients with acute coronary syndromes

### **Diagnostic Evaluation**

- The Problem?
  - Heterogeneous, overlapping clinical presentations among ACSs
- Most patients with Chest Pain:
  - OMI, History, PE, EKG, CXR, Markers/labs
  - ACS Key Remember, many patients lack initial laboratory or electrical evidence of ischemia.
  - You MUST rule out suspected ACS with repeat EKGs and Labs over 12 hours!

## Your Differential Diagnosis (DDX)

- Unstable angina versus NSTEMI
- STEMI
  - Although the presentation of a true STEMI is more likely to be "classic" in nature (i.e., diaphoretic older male performing Levine's sign while stating that it feels like an "elephant is on my chest")
- "Silent" MIs do occur, especially among elderly, diabetics, and woman.
- How do we diagnose ACS?
  - 1. Presenting symptoms
  - 2. Acute EKG abnormalities
  - 3. Detection of specific serum markers of myocardial necrosis

#### **Diagnostic Evaluation**

#### Distinguishing Features of Acute Coronary Syndromes

		Myocardial Infarction	
Feature	Unstable Angina	NSTEMI	STEMI
Typical symptoms	Crescendo, rest, or new-onset severe angina	Prolonged "crushing" chest pain, more severe and wider radiation than usual angina	
Serum biomarkers	No	Yes	Yes
Electrocardiogram initial findings	ST depression and/or T-wave inversion	ST depression and/or T-wave inversion	ST elevation (and Q waves later)

### Diagnostic Evaluation: H&P

#### **Concerning characteristics**

- Severe, persistent typically substernal/retrosternal pain
- Chest pain described as "pressure" or "heaviness" "squeezing"
- Radiation to the jaw, neck or left arm
- Nausea and/or vomiting (parasympathetic response)
- Profuse sweating (diaphoresis) (sympathetic response)
  - Nausea, fatigue, weakness, vague discomfort or generally "just not feeling well"

## Diagnostic Evaluation: H&P

Concerning characteristics

\*\*\*\*Atypical presentations frequently missed are in **women**, the **elderly** and **diabetics**\*\*\*

- Physical exam may or may not be helpful, watch out for those that look "ok" or deceptively well
- Pulse rate may be bradycardic (inferior MI), tachy or irregular
- New systolic murmur....very bad sign... Indicates papillary muscle involvement, mitral valve regurgitation or a Ventricular Septal Defect
- Signs of CHF
- S1 S2 diminished due to poor contractility
- JVD

Approach to any Chest Pain Patient:

• Triage-High level, GOAL Assess < 10 minutes

#### OML Your initial intervention

- •O2 (Esp. if O2 sat less than 90%)
- Monitor (EKG, vitals/SPO2)
- IV access, Obtain labs/push Morphine if needed

- NKDA Aspirin upon arrival (160-325mg chewed)
- Door to ECG: < 5 minutes , if initial not diagnostic, repeat at 5–10-minute intervals
- Door to PCI < 90 minutes (Early Warning System, EMS Notification, STEMI Alert and IF AVAILABLE)

#### OR

 Door to fibrinolytics <30 minutes if PCI not available early intervention/medical management

History: Focused history - OPQRST:

"Any history of heart problems?" If yes

"How you feel now, is this the same or worse?

"Have you ever been diagnosed with angina/heart attack?"

"Is this your typical angina/heart attack pain (or is there a change from the baseline)?"

• If all yes....the patient just told you what they hav!e

History

- Any Red Flags?
- Previous MI, Altered Mental Status, known CAD, or taking drugs?
- Cardiac Risk Factors
- Drug Use (illicit, prescribed, OTC)
- Medications

Beta Blockers, Calcium Channel Blockers, Digoxin Erectile Dysfunction meds prescribed or recreational

• Allergies?

### Calculate a TIMI Score – UA/NSTEMI

Thrombolysis in Myocardial Infarction (TIMI risk score)

- 1. Age greater than 65
- ≥ 3 risk factors for CAD Age, Sex, Family history, Smoking, High blood pressure, High blood cholesterol levels, Diabetes, Overweight or obesity
- 3. Known coronary artery stenosis of  $\geq$  50% by prior angiography
- 4. ST segment deviations on the ECG at presentations
- 5. At least 2 angina episodes in the last 24 hours
- 6. Use of aspirin in the prior 7 days
- 7. Elevated serum troponin or CK-MB

History

- ROS: Review of Systems
- Up to 40% of ACS patients do NOT have chest pain as chief complaint
- May lead to delayed or inadequate treatment
- Dyspnea, N/V, lightheadedness, diaphoresis
- Shoulder, arm, or neck/jaw discomfort
- Mental status change, syncope, near-syncope
- Epigastric or upper abdominal discomfort

#### History

- Prior History
- Family history
- Smoking history
- Co-morbidities: HTN, Dyslipidemia, DM, COPD, Vascular DZ
- Meds/Allergies/Herbals/Street Drugs
- Specific risk factors for Differential Diagnosis

#### IF THEY ARE UNSTABLE DON'T MESS AROUND!!! TREAT THEM!!!

### **Diagnostic Evaluation: Physical Exam**

**Possible Findings:** 

- S4 gallop (Pressure Overload)
- •S3 heart sound (if CHF present) (Volume Overload)
- Systolic Murmur (new MR?)
- Crackles/rales (if CHF present)
- JVD (CHF or right vent MI)
- Vitals, ALWAYS re-evaluate after each intervention
- EKG: what do you see?
- Labs...are the results in?

### **Diagnostic Evaluation: EKG**

#### • <u>ECG</u>

- Acute or chronic findings?
- Do you have a previous ECG to compare with?
- Normal versus nonspecific ST/T-wave changes versus ST depression versus ST elevation
- Telemetry (continuous ECG monitoring)
- Daily 12-lead ECGs after admission
- REMEMBER a few patients will have a <u>completely normal</u> <u>ECG</u>

### **Diagnostic Evaluation: EKG**

- ECG -- MUST know which leads correspond to the ischemic/infarcted area of the heart
  - ST-segment depression >> suggest ischemia
  - ST-segment elevations >>> suggest acute injury/necrosis
  - Q-waves suggest >>> myocardium has died
  - ALL inferior wall acute myocardial infarctions need a rightsided lead V4 (V4R) prior to using nitroglycerin

#### Localizing areas of myocardial ischemia or infarction



ı	aVR	v,	V <sub>4</sub>
Lateral		Septal	Anterior
"	aVL	v <sub>2</sub>	v <sub>5</sub>
Inferior	High Lateral	Septal	Lateral
III	aVF	V3	v <sub>6</sub>
Inferior	Inferior	Anterior	Lateral

#### The 12-Lead ECG: Anatomic Locations and Supplying Coronary Arteries



#### Diagnostic Evaluation: EKG







#### Diagnostic Evaluation: EKG



Anteroseptal MI
#### Diagnostic Evaluation: EKG



#### Anterior MI

#### Diagnostic Evaluation: EKG



#### Serum markers

Are usually obtained serially (i.e. every 3-4 hours) with pattern observation

- A patient with three negative "sets" of cardiac enzymes is often said to have been "ruled out" for MI
  - General Sensitivity of CK-MB and Troponins
    - 2hr 20%
    - 4hr 40%
    - 6hr 60%
    - 8hr 80%
    - 10hr 95%
    - 12hr 100% (rule out MI)

• Serum markers

UA patients often will have normal or minimally abnormal serum markers

Most places are consistent with ACC/AHA guidelines and have observed patients for approximately 8-12 hours

- Serum Cardiac markers
  - Troponin I and T
    - Very specific and sensitive-especially Troponin I
    - Rises in 3-4 hours
    - Peaks at 18-36 hours
    - Returns to baseline in 7-10 days
      - Very helpful if presentation is delayed
    - A rise in either Troponin I or T is diagnostic for AMI

- Serum Cardiac markers
  - CK
    - Neither sensitive nor specific
  - CK-MB isoenzyme
    - A rise 5 times above baseline is diagnostic
    - Rises in 3-8 hours
    - Peaks at 24 hours
    - Returns to baseline in 2-3 days
  - Myoglobin
    - Neither sensitive nor specific
    - First to rise and first to fall (24 hours)

## Diagnostic Evaluation: other tests

#### Diagnostics

Additional Testing

- Serial ECGs
- Serial labs
- Chest X-Ray (2 view)
- Echocardiogram (stunning, wall motion abnormalities, acute valve disorders)
- CT angiogram (CTA)/VQ (Considering Pulmonary Embolism?)
  - (CT Angiogram PE) If pregnant or renal failure –Example Creatinine >1.5 – VQ Scan should be ordered to rule out Pulmonary Embolism).
- Stress Test
- Nuclear Testing

# Formulate a treatment plan for patients with acute coronary syndromes

## **ACS: Treatment**



## Summary of treatment



## Treatment

#### Basic considerations for STEMI

- Acute Treatment:
  - Relieve ischemic pain (nitro, morphine, O2)
  - Assess hemodynamic state and correct abnormalities
    - Fluid, Inotropes, pressors
  - Correct dysrhythmias
  - Antithrombotic therapy
  - Initiate reperfusion
    - PCI
    - Fibrinolytics
    - CONSULT cardiology early!!!



46 yo presents with 2 wk history of intermittent pain. You order this EKG when the patient is not having chest pain. What are you concerned about?



# Wellen's Syndrome

#### Clinical Significance

- Wellen's syndrome is a pattern of **deeply inverted or biphasic T waves in V2-3**, which is highly specific for a **critical stenosis of the left anterior descending artery** (LAD).
- Patients may be pain free by the time the ECG is taken and have normally or minimally elevated cardiac enzymes; however, they are at extremely high risk for extensive anterior wall MI within the next few days to weeks.
- Due to the critical LAD stenosis, these patients usually require invasive therapy; do poorly with medical management; and may suffer MI or cardiac arrest if inappropriately stress tested.

#### Treatment: Disposition

#### ACS

#### **Disposition**

- Although dispositioning patients with ACSs cannot be "cook booked," certain general conclusions are reasonable based on a given patient's ACS likelihood and short-term risks
- 1. Low likelihood & low risk  $\rightarrow$  outpatient evaluation
- 2. Intermediate likelihood & low risk → outpatient evaluation versus inpatient admission w/telemetry (i.e., 24-hour chest pain unit)
- 3. Intermediate likelihood & intermediate risk  $\rightarrow$  inpatient admission with telemetry
- Intermediate or high likelihood & high risk → Cardiac Care Unit (CCU)

## **Treatment: Disposition**

#### ACS

<u>Disposition</u>

- Clinical factors which *almost always* require admission in spite of above assessments
  - Continuous chest pain
  - Positive serum markers
  - Significant, new ST segment abnormalities
  - New, deep T-wave inversions ("Wellens" T-waves)
  - Significant hemodynamic abnormalities, especially hypotension
  - "High risk" stress test results (+/-)

## **Treatment: Disposition**

## Disposition in ACS

- STEMI
  - Cardiology especially if interventional
  - Internal Medicine if no Cardiology
- NSTEMI
  - Internal Medicine-may end up in Cardiology
- Angina
  - Internal Medicine

\*\*\* Don't send anyone home without a rule out! \*\*\*\*

# **Review the key points**

# **Basic considerations for STEMI**

## - Standard of Care

- Aspirin upon arrival?
- Aspirin prescribed at discharge?
- Beta-blocker upon arrival?
- Beta-blocker prescribed at discharge?
- LDL-measurement as inpatient?
- Lipid-lowering therapy at discharge?
- ACE or ARB prescribed at discharge for patients with LV dysfunction?
- Time to fibrinolytics (if appropriate)?
- Time to PCI (if appropriate)?
- Overall reperfusion therapy time?
- Smoking cessation advice or counseling?

## Complications of acute coronary syndromes

- Prognosis
  - 5-15% of hospitalized patients will die!
    - Risk factors:
      - Infarct size and severity
      - Age
      - Co-morbid conditions
      - Development of heart failure or hypotension

### Complications of acute coronary syndromes

#### Prognosis

- Post discharge mortality:
  - 6-8% within the first year
    - <sup>1</sup>/<sub>2</sub> within the first three months
  - 4% per year following the first year
    - Risk factors:
      - LV dysfunction
      - Residual cardiac ischemia
      - Ventricular arrhythmias
      - History of prior MI
      - Resting LVEF is most useful prognostic indicator

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#### THANK YOU

• Stay Safe and I look forward to seeing you at AAPA next year!