Acute Coronary Syndrome

Deirdre Walsh, MMSc., PA-C
Outline and Objectives

- Discuss the pathophysiology of coronary artery disease
- Discuss cardiac risk factors for CAD
- Review acute coronary syndromes: unstable angina, NSTEMI, and STEMI
- Discuss cardiac enzymes and EKG changes associated with ACS
- Review non-cardiac etiologies of chest pain
- Analyze case studies related to ACS
Case Study:

- 51 y/o female patient with a PMHx of HTN, DM, GERD, and DVT’s presents to your office c/o intermittent epigastric pain x 2 weeks. She said she remembers feeling “poor” with some GI upset about 2 weeks ago. She says she is now sleeping on 1 pillow and has noticed that her legs are a little “puffy.”
Case Study:

- What is on your differential diagnosis?
  - Acute Coronary Syndrome
  - GERD
  - What else?
<table>
<thead>
<tr>
<th>Differential Diagnoses:</th>
</tr>
</thead>
</table>

<table>
<thead>
<tr>
<th>Ischemia</th>
<th>Non-ischemic CV</th>
<th>Chest wall / Musculoskeletal</th>
<th>Pulmonary</th>
<th>GI</th>
</tr>
</thead>
<tbody>
<tr>
<td>ACS</td>
<td>Aortic Dissection</td>
<td>Costochondritis</td>
<td>Pneumonia</td>
<td>PUD: perforating / non-perforating ulcer</td>
</tr>
<tr>
<td>Myocarditis</td>
<td>Herpes Zoster</td>
<td></td>
<td>Pulmonary Embolus</td>
<td>Cholecystitis</td>
</tr>
<tr>
<td>Pericarditis</td>
<td>Rib fracture</td>
<td></td>
<td>Pleurisy</td>
<td>Esophageal Spasm</td>
</tr>
<tr>
<td>Stress Cardiomyopathy</td>
<td></td>
<td></td>
<td>Pneumothorax</td>
<td></td>
</tr>
</tbody>
</table>
Case Study:

● Physical Exam:
  ○ She appears more lethargic since her last visit 6 months ago. She is warm with 1+ LE edema. You hear a II/VI systolic murmur at the apex with radiation into the axilla. She has some mild crackles bilaterally.

● Vital Signs:
  ○ BP: 140/81, HR 86, O2 sat: 98% on room air

● Labs:
  ○ Notable for a increase in her sCr from her baseline around 0.8 → 1.2
  ○ Troponin 0.8
Case Study:

- **Diagnostics: EKG**

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Value</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vent. rate</td>
<td>71 BPM</td>
<td>Normal sinus rhythm</td>
</tr>
<tr>
<td>PR interval</td>
<td>138 mm</td>
<td>Possible Left atrial enlargement</td>
</tr>
<tr>
<td>QRS duration</td>
<td>82 ms</td>
<td>Septal infarct, age undetermined</td>
</tr>
<tr>
<td>QT/QTc</td>
<td>304/428 ms</td>
<td>Cannot rule out</td>
</tr>
<tr>
<td>P-R-T axes</td>
<td>72 14 67</td>
<td>Abnormal ECG</td>
</tr>
</tbody>
</table>
Case Study

- Diagnostics: CXR
  - Notable for Kerley B lines & venous congestion
Case Study

- Bedside ultrasound: B lines
Case Study:

- What would you like to do next?
- Would you send this lady to the ER or do you think she is stable to undergo outpatient testing?
- What additional diagnostics would you like to order?
Diagnostics

- Echo reveals multiple WMA with a newly reduced LVEF of 35%. She also has moderate MR. What’s next?
Diagnostics

- LHC shows multi-vessel disease, including a 90% left main
Management

● Labs:
  ○ Follow the troponin until it down-trends
  ○ BID BMP’s while diuresing

● Medical Management:
  ○ Aggressive BP control
  ○ BB if compensated & end-organ function is stable
  ○ Nitro gtt if needed for BP/chest pain– caution w/ inferior STEMI
  ○ ASA, statin. Hold ACEi pending LHC. Do not initiate additional anti-platelet until evaluated by surgical team
  ○ IIb/IIIa Inhibitor

● Mechanical Support:
  ○ IABP, Impella, ECMO
Acute Coronary Syndrome

- Refers to any group of clinical symptoms compatible with acute myocardial ischemia
- Includes:
  - Unstable angina (UA)
  - Non-ST segment myocardial infarction (NSTEMI)
  - ST segment elevation myocardial infarction (STEMI)
Why Do We Care?

- Cardiovascular disease is the leading cause of death in the United States; accounting for 1 out of every 3 deaths
- Every 50 seconds, an American will have a myocardial infarction
- CVD accounted for more than 17.6 million deaths in 2016
- It’s expensive! Total cost of CVD in the United States in 2015 was estimated at $351.2 billion; projected to increase to $749 billion in 2035
Etiology

- The **most common cause** of ACS is due to an acute thrombus in an atherosclerotic coronary artery.
- Less commonly caused by coronary artery embolism, coronary artery dissection, or coronary spasm.
Pathophysiology

- Key Stages of Acute Coronary Syndrome:
  - Ischemic cascade
  - Plaque formation and rupture
  - Coronary occlusion
  - Ventricular remodeling
Plaque Formation & Rupture

- Plaque Formation
- Fibrous Cap Rupture
- Myocardial Infarction
- Myocardial Ischemia
- Thrombus Formation
Coronary Artery Occlusion

- The occlusion can occur in minutes and if severe enough, will lead to *myocardial infarction*.
- Blood flow must be restored rapidly, or the muscle dies and scars.
- Partial occlusion can lead to *myocardial ischemia*. 
Myocardial Ischemia

- **Ischemic tissue** has impaired contractility and relaxation
- You’ll see hypokinetic or akinetic wall motion abnormalities on the echo
- The size of the affected area determines effects; ranging from mild heart failure to cardiogenic shock
Myocardial Infarction

- **Myocardial necrosis** is due to abrupt disruption in coronary blood flow
- Infarcted tissue is **permanently** damaged
- Potentially reversible ischemia next to the infarcted tissue
  - Transmural
  - Non-transmural (subendocardial)
Ventricular Remodeling

- Once the myocardium scars, it becomes fibrosed and functionless
- The remaining healthy tissue hypertrophies to compensate for the dead area
Coronary Artery Disease

- **Risk Factors:**
  - Hypertension
  - Hyperlipidemia
  - Diabetes
  - Tobacco use
  - Family hx of premature CAD
  - Male
  - Age
  - Sedentary Lifestyle
## Acute Coronary Syndrome

<table>
<thead>
<tr>
<th>Unstable Angina</th>
<th>NSTEMI</th>
<th>STEMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-occlusive thrombus</td>
<td>Non-occlusive thrombus, but causes tissue damage and mild myocardial necrosis</td>
<td>Complete thrombus occlusion</td>
</tr>
<tr>
<td>Non-specific ST changes</td>
<td>ST depression +/- TWI</td>
<td>ST elevation on 12 lead or new LBBB</td>
</tr>
<tr>
<td>Normal cardiac enzymes</td>
<td>Elevated cardiac enzymes</td>
<td>Elevated cardiac enzymes High risk for cardiac arrest</td>
</tr>
</tbody>
</table>

### Diagrams:
- **Non-occlusive thrombus**: Non-occlusive thrombus, but causes tissue damage and mild myocardial necrosis.
- **Non-specific ST changes**: ST depression +/- TWI.
- **Normal cardiac enzymes**: Elevated cardiac enzymes.
- **Elevated cardiac enzymes**: Elevated cardiac enzymes High risk for cardiac arrest.

---

**STEMI**

- STEMI: Complete thrombus occlusion

**Non-specific ST changes**

- Non-specific ST changes: ST depression +/- TWI

**Normal cardiac enzymes**

- Normal cardiac enzymes: Elevated cardiac enzymes

**Elevated cardiac enzymes**

- Elevated cardiac enzymes: Elevated cardiac enzymes High risk for cardiac arrest
Stable Angina

- **Presentation:**
  - Substernal chest pain often described as pressure or squeezing
    - Can radiate to left neck, arm, or jaw
  - Associated symptoms: SOB, nausea, HA, dizziness, palpitations, diaphoresis
  - Symptoms may improve with rest & nitroglycerin
Stable Angina

● **Diagnosis:**
  ○ Detailed history and physical exam
  ○ No significant electrocardiogram changes
  ○ Normal cardiac enzymes

● **Treatment:**
  ○ Modification of risk factors:
  ○ BB if hypertensive, statin if DLD, blood sugar control in DM, etc
  ○ ASA recommended for all adults >50 for primary prevention
  ○ Consider stress test or echo
Unstable Angina

● **Presentation:**
  ○ Angina that is increasing in severity and/or frequency
  ○ Exacerbated by less exertion or not relieved by pharmacologic treatment or rest
  ○ Usually lasts >20 minutes

● **Evaluation:**
  ○ Cardiac biomarkers are **negative**
  ○ EKG: non-specific ST segment and T wave changes
Quick EKG Review:
ST-segment Depression
ST-segment Elevation (STEMI)

- STE is defined as elevation at the J point, or the point where the ST takes off from the QRS.
Unstable Angina

● **Treatment:**
  ○ Maximize medical therapy
    ■ ASA, BB, nitrates, opiates, oxygen, heparin gtt
  ○ If pain persists, consider Ranexa, long acting oral nitrates (imdur), and CCB

● **Additional Diagnostics:**
  ○ Stress test or MPI to evaluate severity of CAD, then may proceed with LHC if indicated
NSTEMI

- Caused by sub-endocardial MI
- **Evaluation:**
  - **Elevated** cardiac biomarkers: troponin, CK, CK-MB
    - Indicates myocardial ischemia is present with cell death
  - EKG:
    - ST depressions and T wave changes:
      - NEW horizontal or down-sloping ST depression >0.05 mV in two contiguous leads and/or
      - TWI >0.1 mV in two continuous leads with prominent R/S ratio >1
Cardiac Markers

- Serum markers of myocardial cell injury that are released into the bloodstream after myocardial cell necrosis
- **Cardiac enzymes**: CK-MB
- **Cell contents**: troponin 1, troponin T, myoglobin
Cardiac Biomarkers

● Troponin:
  ○ Preferred biomarker for cardiac necrosis: most sensitive & specific
  ○ Acute rise 3-12 hours after necrosis begins & can remain elevated for days depending on the size of the infarct
  ○ Peaks at 20 hours
  ○ Troponin can also be elevated in acute heart failure, pericarditis, and AKI

● Creatinine kinase (CK): peaks in 12-24 hours, non-specific for cardiac event, returns to baseline in 3—48 hours

● CK-MB: peaks in 10-18 hours, more specific than CK, less than troponin
Release of Biomarkers after MI

- Myoglobin and CK isoforms
- Troponin (large MI)
- CKMB
- Troponin (small MI)
- 10% CV/99th percentile

Days after onset of AMI

Multiples of the upper limit of normal
# Cardiac Markers

<table>
<thead>
<tr>
<th>Marker</th>
<th>Initial Elevation</th>
<th>Peak Elevation</th>
<th>Return to Baseline</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myoglobin</td>
<td>1-4 h</td>
<td>6-7 h</td>
<td>18-24 h</td>
</tr>
<tr>
<td>CK-MB</td>
<td>4-12 h</td>
<td>10-24 h</td>
<td>48-72 h</td>
</tr>
<tr>
<td>Cardiac Trop I</td>
<td>3-12 h</td>
<td>10-24 h</td>
<td>3-10 d</td>
</tr>
<tr>
<td>Cardiac Trop T</td>
<td>3-12 h</td>
<td>12-48 h</td>
<td>5-14 d</td>
</tr>
</tbody>
</table>
Troponin and Mortality in ACS

Troponin I Levels Predict the Risk of Mortality in ACS

NSTEMI

- Additional Diagnostics:
  - Echo: evaluate LV function and look for WMA
  - Myocardial Perfusion Imaging
  - CT Coronary Angiography
  - LHC
NSTE MI

- **Treatment**
  - Focused on stabilizing the patients condition & relieving ischemic pain
    - Nitrates: SL, topical, or IV
    - Beta Blockers: indicated in all patients unless contraindicated (hypotensive, acute decompensated CHF, bradycardia)

- **Antithrombotic therapy:**
  - ASA
  - Heparin or LMWH
  - P2Y12 inhibitors: Clopidogrel, Prasurgel, Ticagrelor
  - Glycoprotein IIb/IIIa receptor antagonists (tirofiban)
  - May be dependent on TIMI score (risk stratification)
NSTE MI

- **Additional Treatment**
  - Percutaneous coronary intervention
  - Coronary artery bypass and grafting for severe multi-vessel disease
## Risk Stratification: TIMI Score

<table>
<thead>
<tr>
<th>TIMI Risk Score Calculator</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Age ≥65 years?</td>
<td>Yes (+1)</td>
</tr>
<tr>
<td>≥3 Risk Factors for CAD?</td>
<td>Yes (+1)</td>
</tr>
<tr>
<td>Known CAD (stenosis ≥50%)?</td>
<td></td>
</tr>
<tr>
<td>ASA Use in Past 7d</td>
<td></td>
</tr>
<tr>
<td>Severe angina (≥2 episodes w/in 24 hrs)?</td>
<td>Yes (+1)</td>
</tr>
<tr>
<td>ST changes ≥0.5 mm?</td>
<td></td>
</tr>
<tr>
<td>+ Cardiac Marker?</td>
<td>Yes (+1)</td>
</tr>
<tr>
<td>Total Score</td>
<td>pts</td>
</tr>
</tbody>
</table>

TIMI Score Interpretation

N = 3687  P_{(trend)} < 0.0001

Mortality at 30 Days (%)

<table>
<thead>
<tr>
<th>TIMI Risk Score for STEMI</th>
<th>% at risk: 6%</th>
<th>19%</th>
<th>22%</th>
<th>16%</th>
<th>15%</th>
<th>10%</th>
<th>6%</th>
<th>3%</th>
<th>2%</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>0.9</td>
<td>2.0</td>
<td>2.4</td>
<td>4.9</td>
<td>9.2</td>
<td>10.7</td>
<td>16.0</td>
<td>18.0</td>
<td>32.2</td>
</tr>
</tbody>
</table>
NSTE MI
Case Study

● 71 y/o female presents from a nursing home with cough and nausea x 4 days.
● Vital Signs:
  ○ Temp 100.6, BP: 97/50, HR: 110
● Pertinent labs:
  ○ WBC 14, troponin 3.5, lactic 2.4
Case Study Cont.

- **CXR:**
Case Study Cont.

- EKG:
More of your lab work comes back...
- Troponin is now 6
- CRP, d-dimer, and ferritin are all mildly elevated

What’s your most likely diagnosis?
How are we handling ACS in COVID patients?
UA/NSTEMI PRESENTATION WITH CONFIRMED COVID-19 OR SUSPECTED AND UNDER INVESTIGATION FOR COVID-19

Personal Protective Equipment (PPE):
- Airborne (Respirator: PAPR/N95)
- Contact: Gown/gloves/Boot covers
- Eye Shield: Goggles, Face/Eye Shield

CAREFUL DOFFING OF GOWNS
SEE VIDEO INSTRUCTIONS:
https://youtu.be/bG6zrSnenPg

Early angiography with PPE
Treatment based on findings:
- Medical management or Revascularization (PCI w CABG)

NSTEMI/UA

Confirmed COVID-19 or PUI?

NO

Admit Early angiography < 24 hours

YES

Risk Assessment

Conservative Management
- Antplatelet therapy
- Anticoagulation 48 hours
- High intensity statin therapy
- Beta blockers/ACE inhibitors

Conservative Management

REASSESSMENT
Consider Angiography >> 14 days
If confirmed negative (x2)
AND Clinically Indicated

Admit to Isolation room with cardiology F/U

VERY HIGH CV RISK:
- Refractory angina
- Hemodynamic Instability
- Electrical Instability

HIGH or LOW CV RISK

Piedmont HEART
STEMI PRESENTATION WITH CONFIRMED COVID-19 OR SUSPECTED AND UNDER INVESTIGATION FOR COVID-19

Person Under Investigation (PUI)
- Fever
- Respiratory Symptoms
- Recent Foreign Travel to a Country with Level 3 Travel Health Notice

Confirmed COVID-19 or PUI?
YES

Call Code STEMI and proceed with Primary PCI

Consider Limited Transesophageal Bedside Echo*
- If NO NEW RV/MA Consider Medical Rx only
- Consider CTA in certain cases if clinically indicated

CV Risk Assessment

LOW CV RISK
Consider fibrinolytic therapy

HIGH CV RISK
Consider primary PCI

Contraindicated

No Contraindications for Fibrinolytics:
- Active internal bleeding
- History of CVA
- Intracranial or intraspinal surgery or trauma ≤ 3 months
- Intracranial neoplasm, AV malformation, or aneurysm
- Known bleeding diathesis
- Severe uncontrolled hypertension

Administer tenecteplase (Thrombus) weight-based boluses over 5 seconds

Contraindications for PCI:
- Weigh risks/benefits to patient vs the risk of viral exposure to PCI staff

PCI with Full PPE*

Reperfusion Assessed by:
Symptom relief
STEMI resolution > 50%

Reperfusion?
NO
Tranfer to inpatient rooms for recovery with cardiology F/U

YES

* Personal Protective Equipment (PPE):
- Airborne/Respirator: FAPR/N95
- Contact: Gown/gloves/Boot covers
- Eye Shield: Goggles, Face/Eye Shield

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Piedmont HEART
A Word on COVID & Myocardial Injury

- Acute COVID cardiovascular syndrome – myocarditis like syndrome involving myocardial injury w/ reduced LV fxn in the absence of obstructive CAD
- Acute myocardial injury (elevated troponin) occurs in 20-30% of patients with COVID – associated with increase in risk of all-cause death
- ACovCS may be due to ACS, demand ischemia, microvascular ischemic injury, injury related to cytokine dysregulation, or myocarditis
A Word on COVID & Myocardial Injury

- Mechanisms unclear at this point
- Endomyocardial biopsy of patient with CGSs showed low-grade myocardial inflammation – localization of SARS-COV-2 within macrophages, but not cardiomyocytes
- Manage conservatively if possible
**STEMI**

- ST elevation MI: persistent complete occlusion of an artery supplying a significant area of the myocardium without adequate collateral circulation
- **80% of the heart damage occurs within the first 2 hours**
- Goal is immediate intervention either pharmacologically or percutaneously
- EMS-transported patients have improved symptom onset to reperfusion time
STEMI

- EKG Criteria:
  - >/2mm of STE in 2 contiguous precordial leads in men (1.5 mm for women)
  - >1mm in other leads
  - Reciprocal ST depressions
  - New LBBB on EKG
**STEMI**

- **New LBBB:** Delay in conduction of the electrical system to the left ventricle
  - QRS > 120 ms
  - Tall R waves in lateral leads (I, V5-V6) and deep S waves in right precordial leads (V1-V3)
- Associated with higher in hospital mortality rates (up to 25%)
- Less likely to have early recognition than patients with recognizable STE
STEMI

- STE due to an acute MI demonstrates a regional or territorial pattern
  - **Anterior leads:** V1-V6
  - **Lateral leads:** I, aVL, V5, V6
  - **Inferior leads:** II, III, aVF
## Regions, expected EKG leads, culprit vessels

<table>
<thead>
<tr>
<th>Vessel Involved</th>
<th>Anterior</th>
<th>Anteroseptal</th>
<th>Anterolateral</th>
<th>Lateral</th>
<th>Inferior</th>
<th>Posterior</th>
</tr>
</thead>
<tbody>
<tr>
<td>LAD</td>
<td>LAD</td>
<td>Circumflex, LAD, Diagonal</td>
<td>Circumflex</td>
<td>RCA, LCx</td>
<td>RCA, LCx</td>
<td></td>
</tr>
<tr>
<td>EKG correlation</td>
<td>V2-V6</td>
<td>I, aVL, V1-V4</td>
<td>I, aVL, V4-V6</td>
<td>I, aVL, V5-V6</td>
<td>III, III, aVF</td>
<td>V1-V3 (ST depressions)</td>
</tr>
<tr>
<td>EKG Changes</td>
<td>Poor R wave progression, STE &amp; TWI</td>
<td>R wave disappears ST segment rises &amp; T wave inverts</td>
<td>STE</td>
<td>STE, TWI</td>
<td>STE, TWI</td>
<td>Tall R waves, ST depression, upright T waves</td>
</tr>
</tbody>
</table>
STEMI Management

- Morphine
- O2
- Nitro (SL, IV)
  - Caution use with inferior MI
  - Beta blocker: ideally decrease HR & decrease oxygen demand, but must be used cautiously
    - Anterior MI: is your patient compensated? Are they warm & perfused or cold & hypotensive?
    - Inferior MI: bradycardia & CHB more common
- ASA 325 mg
- BB if no contraindication
- Additional tx: heparin, clopidogrel, IIb/IIIa inhibitors (CABG candidate?)
STEMI Management

● Emergent Revascularization:
  ○ PCI with goal door to balloon time 90 minutes
  ○ If facility unable to do PCI, fibrinolytic therapy with goal door-to needle time 30 minutes if onset of symptoms <12 hours
  ○ Gather family history for contraindications to fibrinolytic therapy
## STEMI

- **Thrombolytic Therapy:**

<table>
<thead>
<tr>
<th>Absolute Contraindication</th>
<th>Relative Contraindication</th>
</tr>
</thead>
<tbody>
<tr>
<td>Active bleeding or known bleeding disorder</td>
<td>Severe/uncontrolled HTN</td>
</tr>
<tr>
<td>Prior hemorrhagic stroke/ other stroke within 1 year</td>
<td>Abnormal coagulation studies (elevated INR)</td>
</tr>
<tr>
<td>Intracranial or spinal cord cancer</td>
<td>Old ischemic stroke</td>
</tr>
<tr>
<td>Suspected/known aortic dissection</td>
<td>Recent major surgery/trauma</td>
</tr>
<tr>
<td>Pregnancy</td>
<td></td>
</tr>
</tbody>
</table>
Post-STEMI Revascularization

- DAPT (ASA, Plavix) for 12 months, then ASA indefinitely
  - Ticagrelor was found to be superior to Plavix (PLATO trial)
  - Prasugrel was found to be superior to Plavix (Triton-TIMI 38 trial)
- Continue O2 as needed
- High-intensity statin
- Beta-blockers
- ACEi for patients with LV dysfunction (ARB if ACEi not tolerated)
- Glycemic and BP optimization
- Modify lifestyle as necessary
- Follow up is important: may have other critical or noncritical disease that needs to be staged
- Secondary prevention: prevent further MI’s, CHF, and strokes
STEMI Complications

- Arrhythmias: VT or VF (most common cause of death), a.fib, heart block
- Heart failure:
  - New LV dysfunction / Cardiomyopathy
  - Papillary muscle rupture causing new MR
- LV free wall rupture or aneurysm
- RV failure
- Cardiogenic Shock
- Cardiac Arrest
Post-STEMI Sequelae

- Cardiac arrests can occur at any time with STEMI
- Pt should be considered for coronary angiography once stabilized (may mean mechanical support +/- ECMO)
- Typically caused by lethal ventricular arrhythmias (VT/VF)
- Targeted Temperature Management:
  - Lower body temperature to 33 degrees Celsius for 24-28 hours; only intervention proven to improve brain function post arrest
Post-MI Cardiogenic Shock

- Mechanical Support:
  - IABP
  - Impella
  - ECMO

- Inotropes:
  - Milrinone, Dobutamine, Epinephrine

- Pressors:
  - Levophed, vasopressin

- Invasive hemodynamic monitoring
IABP & Impella

Diastole

Systole
**STEMI Prognosis**

- Varies depending on baseline health, extent of heart damage, and treatment
- 5-6% die before leaving the hospital, 7-18% die within 1 year
- **Risk factors:**
  - Age, hemodynamic variables (hypotension), DM, AKI
  - Heart failure:
    - Symptoms: pulmonary edema, S3 gallop, elevated JVD
    - 5 year survival for newly diagnosed CHF – 50%
    - Mechanical complication: papillary muscle rupture, VSD, free wall rupture
What is this?
Acute Anterolateral STEMI

- Progressive STE and Q wave formation in V2-V5
- STE now present in 1 and aVL
- Reciprocal ST depression in lead III
- What artery do you expect to be the culprit lesion on LHC
  - LAD, Cx, Diagonal
Case Studies

- You have a 68 y/o male with a prior hx of CABG in 2015, PCI to RCA in 2018, DM type II, and obesity who presented to your office c/o increasing SOB with activity.
- You get an EKG which reveals....
Case Study

- You’re concerned with a posterior STEMI, how can you confirm?
Posterior STEMI

- Lead: V2
Posterior STEMI

- Inverted V2
- Consider posterior STEMI when EKG show significant precordial ST depressions in leads V1-V3
- Correlate with history & clinical findings
Posterior EKG

Figure 1 - Posterior ECG lead placement

V7, V8, V9

Left paraspinal region: in the same horizontal plane as V4-V6

V1-V3
Should remain unchanged from standard 12-lead ECG
Case Study Continued

- EKG confirms posterior STEMI, what now?
Case Study Continued

- Emergent LHC
- While waiting... ASA, heparin bolus +/- nitro
Case Study

- You have a 52 y/o female with a PMHx of SLE, HTN, and obesity who presents to your office c/o chest pain. She describes the pain as sharp without radiation into her neck or back.
Diagnostics & Lab Results

- BP: 131/80
- O2 sat: 98%
- HR: 68 bpm
- Temp: 98.1
- EKG reveals diffuse STE
- Troponin: 0.08
What’s next?
Case Study cont.

- Upon further questioning, she states she had a fever lasting about 3 days a week ago.
- Her pain is worse with deep inspiration and also worse when she lays flat.
- Does this change your most likely diagnosis?
Pericarditis

- Etiology: usually viral or idiopathic
- Chest pain is sharp/pleuritic in nature
- Pericardial friction rub
- EKG changes: new ST elevation or PR depression
- Pericardial effusion
- Treatment:
  - ASA or NSAID
  - Colchicine
  - Refractory pain – add steroids
What else can cause an elevated troponin?
Questions?
Resources