### Outline

- The STEMI Myth: Coronary Occlusion and High-Risk EKGs that Require Reperfusion

  ▼ ResusReview.com
  - Cardiology
  - Critical Care
  - ECMO

  ▼ Beware dichotomies
  - Nothing in biology follows rules like 1 vs 2 mm
  - Dichotomies are false
  - ACC/AHA want you to believe STEMI vs NSTEMI

  ▼ Thrombi are active process
  - forming/dissolving
  - Always changing

  ▼ Occlusion vs Obstruction
  - STEMI = coronary occlusion
  - NSTEMI = MI without occlusion/subendocardial ischemia

  ▼ How sensitive STE for occlusion
  - 70-75%
  - Even worst after arrest

  ▼ Ruled in NSTEMI
  - 25% with occluded infarct, worse outcomes

  ▼ High risk NSTEMI
  - There is a spectrum from NSTEMI to STEMI
  - Especially the NSTEMI with high-risk features
There is a spectrum from NSTEMI to STEMI, especially the NSTEMI with high-risk features. Reperfusion depends not on mm criteria but is there a life-threatening thrombus in vessel?

**Conditions**
- Non diagnostic ECG, +troponin and ongoing pain
- Isolated typical angina, refractory
- Subtle ischemic STE
- Hyperacute T waves

**STEMI Mimics**
- Cardiologists not familiar with

**Dynamic ST segments and T waves**
- Indicates a thrombus that is propagating and lysing
- High risk

**STEMI Equivalent**
- Active cath lab — acute thrombosis in a coronary artery causing persistent ischemia that is refractory to medical management

**Optimal timing for NSTEMI (EUSocCard)**
- Patients at very high risk
  - Refractory angina
  - Severe heart failure
  - Life threatening ventricular arrhythmias
  - Hemodynamic instability
  - “Were not included in RCTs, in order not to withhold potentially life-saving treatment”

  “Such patients may have evolving MI and should be taken for invasive evaluation (<2H) regardless of ECG or biomarker findings”
  - You can do it just on your clinical suspicion
  - ACC/AHA 2014 says the same thing

**AHA/ACC 2013 Guidelines for Reperfusion**
True posterior MI or STE in AVR

Easy to dx low risk NSTEMI by troponin

TIMACS (NEJM 2009)

- NSTEMI randomized to early or routine PCI
- No difference in death/MI/stroke
- Grace >140 better with early PCI
- Early vs 16 vs 52 hours — Need 2 hours

What DO WE Do

Serial ECGs

- Real ischemia always evolves (though may be slowly)

Echocardiogram

- Absence of new wall motion abnormality with high quality contrast echo, read by an expert, essentially rules out transmural ischemia

But

- Absence of WMA in ischemic ST depression

Proportionality

Know your ECGs

Hyperacute Tawws

STEMI Mimics

- LVH

BER

- QTc, STE in V3, and R-wave in V4

LBBB

- New LBBB
  - Downgraded
  - Sgarbossa Criteria
  - Smith-Modifications Sgarbossa Criteria
Isolated Posterior MI

- Posterior wall MI usually occurs along with lateral or inferior wall (RCA or LCX occlusion)
- Posterior wall not directly imaged by standard 12-lead
- But also not electrically silent

Posterior ischemia is seen as reciprocal changes in anterior leads, V1-V4

- Horizontal flat STD, prominent R waves and upright T-waves
- r/S ratio >1 in V2
- These are analogs of anterior STE

Demand ischemia is MUCH more likely than occlusion

- Especially when in V4-V6
- Older patient
- CAD
- Severe supply demand mismatch (tachycardia, anemia, hypoxia, HOTN)

Obtain posterior ECG

- 0.5mm meets STE criteria because of distance to heart

Wellens Syndrome

- Spontaneous reperfusion of LAD occlusion with resolution of chest pain

Two forms

- Anterior biphasic T-waves
- Deeply inverted T-waves with preserved R waves
- Troponins may not be positive depending on time that artery was occluded

Mimics include LVH, severe HTN, benign T-wave inversion

New WMA would have good positive-predictive value

Given the presence of LAD lesion, provocative testing may be disastrous
Anterolateral Wall MI
- Occlusion of branches form the LAD can present with nonclassical noncontiguous STE
- First diagonal (D1) or RI can be a large vessel that perfuses large parts of the anterolateral myocardium
  - Often a target of CAB because of their size
  - Produces STE in aVL and V2 along with upright T-waves and inferior STD with inverted T-waves
  - These are noncontiguous, but mainly due to our simplified visualization of the heart in the thorax
  - This occlusion requires reperfusion

aVR ST Elevation
- aVR lead was developed to evaluate basal segment of septum
- STE seen in two situations
  - Proximal LAD occlusion
  - With diffuse LAD depression
    - LM/LAD insufficiency
    - Severe multi-vessel disease
    - Develop with ACS or supply-demand mismatch (hemorrhage, tachycardia, hypoxia, hypotension)

De Winter
- Total or subtotal occlusion of proximal LAD
  - 2% of cases
- Characterized
  - 1-3mm up-sloping STD after the the J-point in the anterior leads V1-V4
  - Tall positive symmetric T waves
  - Normal QRS
  - Loss of R-wave progression
- 1-2mm STE in aVR
- Probably, but clearly related to K-ATP cardiac channel mutation

▷ Two forms
- Total occlusion, stable ECG features until reperfusion
- Dynamic, seen with subtotal occlusion
- Both require immediate reperfusion

▷ Pseudo-normalization
- Reocclusion of LAD in setting of T-wave inversion results in upright T-waves
  - i.e. Inverted Wellens waves
- These are not normal T-waves, but occluded vessels requiring reperfusion therapy