Disclosures

• I have no financial or commercial interests that impact this presentation.
• There are no specific proprietary products discussed.
• I will not be addressing and specific scientific research.
• I will not be making any specific therapeutic recommendations.
Goals and Objectives

Upon completion of this lecture, the audience will be able to:

- Describe the inclusion and exclusion criteria for thrombolytic therapy in the STEMI patient
- Recognize the opportunity at their entity for use of Thrombolytics
- Explain the risk benefit ratio of using thrombolytics in the STEMI patient
- Explain the care differences in STEMI vs. NSTEMI
- Differentiate between the different types of cardiac disease that cause chest pain
- Explain the usefulness of applying Mission: Lifeline System of Care model to NSTEMI patients
Gold Standard for Treatment of ACS
Gold Standard for Treatment of ACS
Overview of ACS

Acute Coronary Syndromes*

1.57 Million Hospital Admissions - ACS

- UA/NSTEMI†: >1 Million Admissions per year
- STEMI: 235,000 Admissions per year

5/2/2016 American Heart Association; Mission: Lifeline
Acute Coronary Syndrome (ACS)

The spectrum of acute ischemia related syndromes ranging from UA to MI with or without ST elevation that are secondary to acute plaque rupture or plaque erosion.
Types of Angina

- **Stable**: There is a stable pattern of onset, duration and intensity of symptoms, pain is triggered by a predictable degree of exertion or emotion.

- **Variant Angina (Prinzmetal's)**: Cyclical, may occur at rest. Ventricular arrhythmia, brady arrhythmia and conduction disturbances occur. Syncope associated with arrhythmia may occur.

- **Nocturnal Angina** only at night. Possible associated with REM sleep.

- **Unstable Angina** AKA Pre-infarction angina; Pain is more intense, lasts longer
Assessment

- History
- Physical Exam
- 12-Lead ECG
- Stress Testing
- Thallium Scan
- Coronary Angiography
- Cardiac Enzymes
Medications for Angina

Nitrates decrease myocardial 02 demand via peripheral vasodilation and reverse coronary artery spasm thus increase 02 supply to myocardial tissue.

Understanding how Nitrates Work
Peripheral vasodilation results in:
- decreased 02 demand
- decreased venous return to heart
- decreased ventricular filling results in decreased wall tension and thus lower 02 demand
NTG Forms:

- SL (Nitrostat)
- Lingual Sprays - similar to SL in use (Nitrolingual)
- Sustained release capsules/tablets (Nitrobid)
- Ointments 2% (Nitrobid)- wear gloves when applying
- Transdermal Patch (Nitro-Dur)
- IV (Tridil) For attacks unresponsive to other tx
Side/Adverse Effects

- Vascular HA (may be severe)
- Hypotension (may be marked)
- Tachycardia
- Palpitations
Acute Angina Treatment

Goal: Enhance 02 supply to myocardium, relieve acute attacks and prevent further attacks.

- Fentanyl or Morphine for pain
- Oxygen 4-6L as ordered
- NTG sublingual, repeat q5 minutes x3
- Aspirin to prevent platelet aggregation

**Activity/exercise tolerance** - a regular exercise prescription is established after stress testing and/or cardiac cath.

- Establish a baseline
- Gradual increase in activity
- Avoid stressor (emotional, environmental)
- Alternate activity and rest periods
- ADLS – clump cares
- NTG before exercise
Patient education

- Lifestyle modifications for controllable risk factors.
- Support groups are helpful
- Identify precipitating factors for Angina pain
- Medication compliance
What’s the difference?

Angina vs Myocardial Infarction

Pathologic Basis Of Disease.
Pathophysiology of Stable Angina and ACS

**Pathophysiology**

Decreased \( O_2 \) Supply
- Flow-limiting stenosis
- Anemia
- Plaque rupture/clot

Increased \( O_2 \) Demand

\[ O_2 \text{ supply/demand mismatch} \rightarrow \text{Ischemia} \]

\[ \text{Myocardial ischemia} \rightarrow \text{necrosis} \]

**ACS**

Asymptomatic

Angina

Myocardial Infarction

5/2/2016  American Heart Association; Mission: Lifeline
Collateral Circulation

- A network of blood vessels present at birth that can dilate and become functional due to age and long term coronary artery occlusion and ischemia. “collateral circulation”

- Natural “bypass” mechanism helps decrease the size of the MI
Unstable Angina
- Non Occlusive thrombus
- Non Specific on 12-Lead ECG
- Normal Cardiac Enzymes

NSTEMI
- Non-occlusive thrombus sufficient to cause tissue damage & mild myocardial necrosis
- ST depression +/- T wave inversion on 12-Lead ECG
- Elevated cardiac enzymes

STEMI
- Complete thrombus occlusion
- ST elevation on 12-Lead ECG or new LBBB
- Elevated cardiac enzymes
- More severe symptoms
- High risk for cardiac arrest

9/2/2016 American Heart Association; Mission: Lifeline
Complications of MI

- CHF
- Mitral Valve Insufficiency
- Dysrhythmias
- Pericarditis
- Post Infarction MI
- Thromboembolic Complications
- Rupture of Ventricular Wall
Goals

- Limit size of infarct/prevent further damage
- Increase O2 supply and decrease O2 demand
- Prevent and/or recognize complications early
- Reduce pain
- Get to appropriate treatment as safely and quickly as possible
- Primary PCI in STEMI’s
Name 3 situations in which you cannot diagnose STEMI
Name 3 situations in which you cannot diagnose STEMI

- Left Ventricular Hypertrophy
- Chronic or Rate Dependent LBBB
- Paced Rhythm
Cardiac Catheterization

Name 3 situations that demand emergent cardiac catheterization.
Cardiac Catheterization

- Name 3 situations that demand emergent cardiac catheterization.
  - STEMI or new LBBB
  - ACS with hemodynamic or electrical instability despite optimal medical management
  - Uncontrolled CP despite optimal medical management
Diagnosis of ACS

- **At least 2 of the following**
  - History (angina or angina equivalent)
  - Acute ischemic ECG changes
  - Typical rise and fall of cardiac markers
  - Absence of another identifiable etiology
# 12-Lead ECG Interpretation

<table>
<thead>
<tr>
<th>Lead Name &amp; Standard Color</th>
<th>I Lateral</th>
<th>aVR</th>
<th>V1 Septal</th>
<th>V4 Anterior</th>
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<tbody>
<tr>
<td>II Inferior</td>
<td>aVL Lateral</td>
<td>V2 Septal</td>
<td>V5 Lateral</td>
<td></td>
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<tr>
<td>III Inferior</td>
<td>aVF Inferior</td>
<td>V3 Anterior</td>
<td>V6 Lateral</td>
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</tbody>
</table>
12-Lead ECG Sensitivity

**Likelihood of MI**

- **WNL**
- **ST ↓**
- **ST ↑**
- **Q**
- **ST ↑** (Reciprocal Δ’s or ST ↑ Q Waves)
- **ST ↑** (Reciprocal Δ’s Q Waves)

<table>
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<tr>
<th>0-20%</th>
<th>50%</th>
<th>60%</th>
<th>75%</th>
<th>90%</th>
<th>90% - 95%</th>
</tr>
</thead>
</table>
Response to NTG

Usefulness as a Predictor of Ischemic C.P. in the ED

- 223 ED pts. with chief complaint of C.P.
- Ultimately 1/3 had Ischemic C.P. and 2/3 did not
- Used ECG, Enzymes, Stress Testing (1/2) and Cath (29%)
- 90% of all pts. responded at NTG (88 vs. 92%)
- Complete relief seen in 72% (70 vs. 73%) of all pts. whether they had ACS or no ACS

“The practice of supporting or excluding the diagnosis of myocardial ischemia should not be based on a patient’s response to nitroglycerin”

“Our data...strongly suggests that the response of chest pain to nitroglycerin although therapeutically beneficial, has little diagnostic or prognostic value”

Changes in the Numeric Scale for Pain after SL NTG
Do not Predict Cardiac Etiology
Biomarkers in ACS
Risk Stratification by Troponin levels

Mortality at 42 Days

Cardiac troponin I (ng/ml)

0 to <0.4: 831, 1.0%
0.4 to <1.0: 174, 1.7%
1.0 to <2.0: 148, 3.4%
2.0 to <5.0: 134, 3.7%
5.0 to <9.0: 50, 6.0%
≥9.0: 67, 7.5%
Other causes for Troponin Increase

- Trauma
- Congestive heart failure (acute and chronic)
- Aortic valve disease and HOCM with significant LVH
- Hypertension
- Hypotension, often with arrhythmias
- Non-cardiac surgery
- Renal failure
- Critically ill patients, especially with diabetes, respiratory failure
- Drug toxicity
- Hypothyroidism
- Coronary vasospasm, including apical ballooning syndrome
- Inflammatory diseases (eg, myocarditis, Kawasaki disease, smallpox vaccination, Post-PCI
- Pulmonary embolism, severe pulmonary hypertension
- Sepsis
- Burns, especially if TBSA greater than 30%
- Infiltrative diseases: amyloidosis, hemachromatosis, sarcoidosis, and scleroderma
- Acute neurologic disease, including CVA, subarchnoid bleeds
- Rhabdomyolysis with cardiac injury
- Transplant vasculopathy
- Vital exhaustion
Treatment of Acute Coronary Syndrome

Initial Treatment of ACS

**STEMI**
- antiplatelet, anti-ischemic, or anticoagulant therapy
  - Thrombolytics
  - PCI or CABG

**UA/NSTEMI**
- antiplatelet, anti-ischemic, or anticoagulant therapy
  - PCI or CABG

Long-term medical management

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*Also known as Q-wave MI.
†Also known as non-Q-wave MI.
Management of Unstable angina/NSTEMI

Evaluate for **conservative vs. invasive** strategy based upon:

- Likelihood of actual ACS
- TIMI risk score
- ACS risk categories per AHA guidelines
TIMI Risk Scoring

Predicts risk of death, new/recurrent MI, need for urgent revascularization within 14 days

<table>
<thead>
<tr>
<th>Points</th>
<th>1) Age ≥ 65</th>
<th>2) ≥ 3 CAD Risk Factors</th>
<th>3) Known CAD (stenosis ≥50%)</th>
<th>4) ASA use in past 7 days</th>
<th>5) Recent (&lt;24H) severe angina</th>
<th>6) ST deviation ≥ 0.5 mm</th>
<th>7) ↑ Cardiac Markers</th>
</tr>
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RISK SCORE = Total Points (0 - 7)

Risk Score

4.7 | 8.3 | 13.2 | 19.9 | 26.2 | 40.9

Antman et al JAMA 2000; 284:835
Importance of a SYSTEM OF CARE

It is vital to the STEMI patient that every region has a coordinated and collaborative STEMI Team!

Have a plan A, plan B and plan C for how you will get the STEMI patient to primary PCI in under 120 minutes.

If your region can’t make FMC to device in 120 minutes, always screen the patient for fibrinolytic therapy and start immediately if qualified....then transport the patient right away.
ACS includes UA, NSTEMI, and STEMI

Management guideline focus

- Immediate assessment/intervention Risk stratification (UA/NSTEMI vs. STEMI)
- RAPID reperfusion for STEMI (PCI vs. Thrombolytics)
- Conservative vs Invasive therapy for UA/NSTEMI

Aggressive attention to secondary prevention initiatives for ACS patients

- Beta blocker, ASA, ACE-I, Statin
Questions