ST – Elevation Myocardial Infarction

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Outline

- Case Presentation
- STEMI Incidence and Mortality
- STEMI Diagnosis
- STEMI Pathogenesis and Therapy
- Conclusion of Case

Case Presentation

- 46 year old male with no significant past medical history
- Family history of early myocardial infarction – Brother at 35 years old
- Sudden onset chest pain, nausea and emesis
- EKG
Case Presentation

- 325 mg aspirin, 600 mg clopidogrel and heparin
- Transferred for emergent angiography

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**STEMI Incidence and Mortality**

62% reduction in STEMI between 1999 and 2008

Yeh et al. 2010 NEJM 362(23): 2155-2165.

**Outline**

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**STEMI Diagnosis**

- Symptoms Concerning for Myocardial Ischemia
  - Chest pain, shortness of breath, anginal equivalent
- Persistent ST Elevation
  - ≥ 1 mm ST elevation in ≥ 2 continuous leads
    - V2-V3; ≥ 2 mm in men, ≥ 1.5 mm in women
  - LBBB
  - ST depression in V1-V4
  - Question? Consider urgent echocardiogram
- Subsequent Release of Biomarkers

**STEMI Incidence and Mortality**

United States in-hospital mortality is 5-6%, 1 year mortality 7 to 18%


*O’Gara et al. 2013 ACC/AHA STEMI Guideline.

O’Gara et al. 2013 ACC/AHA STEMI Guideline.
STEMI Diagnosis

Acute Coronary Syndrome

- Electrocardiogram
- ST-elevation
- No ST-elevation
- Cardiac markers
- Unstable angina
- Myocardial infarction

Inferior Myocardial Infarction

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Adapted from Alpert et al. 2000, JACC 36(3): 959-69
STEMI Therapy

“The importance of absolute rest in bed for several days is clear”

James B Herrick
1861 – 1954

The National Library of Medicine believes this image to be in the public domain.

STEMI Pathogenesis


2013 ACCF/AHA Guideline for the Management of ST-Elevation Myocardial Infarction

Developed in Collaboration with American College of Emergency Physicians and Society for Cardiovascular Angiography and Interventions

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STEMI Therapy in 2013

- Medical Antiplatelet
  - Aspirin
    - 325 mg load before procedure
    - 100-325 mg daily maintenance dose (optional)
  - Thrombus aspiration
  - Beta blockers

STEMI Therapy

- Medical Anticoagulant therapy

O’Gara et al. 2013 ACC/AHA STEMI Guideline.
STEMI Therapy

• Medical Therapy

A note about beta-blockers

Oral beta blockers should be initiated in the first 24 hours in patients with STEMI who do not have any of the following: signs of HF, evidence of a low output state, increased risk for cardiogenic shock,* or other contraindications to use of oral beta blockers (PR interval >0.24 seconds, second- or third-degree heart block, active asthma, or reactive airways disease).

It is reasonable to administer intravenous beta blockers at the time of presentation to patients with STEMI and no contraindications to their use who are hypertensive or have ongoing ischemia.

O’Gara et al. 2013 ACC/AHA STEMI Guideline.

• Revascularization

Markis et al. NEJM 1981 305: 777-82
Rentrop et al., Circulation 1981 63: 307-317

STEMI Therapy

Medical Therapy

Revascularization

Systems of Care

O’Gara et al. 2013 ACC/AHA STEMI Guideline.
**STEMI Therapy**

- Greater than 120 minute delay to primary PCI?
  
  *Consider Fibrinolysis*

<table>
<thead>
<tr>
<th>COR</th>
<th>LOE</th>
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</thead>
<tbody>
<tr>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>IIa</td>
<td>C</td>
</tr>
<tr>
<td>III</td>
<td>B</td>
</tr>
</tbody>
</table>

- Ischemic symptoms < 12 h
- Evidence of ongoing ischemia 12 to 24 h after symptom onset and a large area of myocardium at risk or hemodynamic instability
- ST depression, except if true posterior ( Inferobasal) MI is suspected or when associated with ST elevation in lead III

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**STEMI Therapy**

- Systems of Care

  All communities should create and maintain a regional system of STEMI care that includes assessment and continuous quality improvement of EMS and hospital-based activities. Performance can be facilitated by participating in programs such as Mission: Lifeline and the D2B Alliance.

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*O’Gara et al. 2013 ACC/AHA STEMI Guideline.*


Case Conclusion

Outline

- Case Presentation
- STEMI Incidence and Mortality
- STEMI Pathogenesis
- STEMI Diagnosis
- STEMI Therapy
- Conclusion of Case
Case Conclusion

• 46 year old male chest pain, anterior ST Elevations
• Underwent emergent angiography
• Drug Eluting Stent placed to LAD
• Uncontrolled Diabetes discovered during admission
• Discharged 3 days later
• Ejection Fraction 30% at 3-months follow up

Summary

• STEMI: Decreasing Incidence, High Mortality
• Plaque Rupture or Erosion
• Interpreting the EKG and Early Diagnosis is Key!!
• We can Reduce Mortality
• Morbidity Remains an Issue
• New Guidelines: Minimize Treatment Delay!

Unstable Angina and Non-ST Elevation Myocardial Infarction:
Diagnostic and Therapeutic Management Based on Current Knowledge and Clinical Judgment

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Section of Interventional Cardiology
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I. Pathophysiologic Mechanisms

II. Diagnosis

III. Prognosis

IV. Management

V. Prevention

Unstable Angina (UA) and Non-ST Elevation Myocardial Infarction (NSTEMI)

Hospitalizations in the U.S.A. due to Acute Coronary Syndromes

1.57 Million Hospital Admissions

UA/NSTEMI 1.24 million Admissions per year

STEMI 0.33 million Admissions per year


Common Pathophysiologic Mechanisms

- UA and NSTEMI are acute coronary syndromes (ACS) characterized as a general rule by a significant decrease in blood supply to the myocardium.

- Most common cause for the decrease in myocardial perfusion is by a non-occlusive thrombus (with potential distal embolization) that has developed on a disrupted atherosclerotic plaque resulting in luminal narrowing.

- UA and NSTEMI pathogenesis and clinical presentations are similar differing in severity with NSTEMI resulting in myocardial damage releasing detectable quantities of a marker of myocardial injury.
**Less Common Causes of UA/NSTEMI**

- Occlusive thrombus with collateral vessels
- Non–plaque thromboembolism (atrial fibrillation; LV thrombus)
- Dynamic obstruction (coronary spasm; vasoconstriction)
- Coronary arterial inflammation
- Coronary artery dissection
- Mechanical obstruction to coronary flow
- Hypotension, tachycardia, anemia, other

**Acute Coronary Syndromes**

**ECG:**

- No ST Elevation
- ST Elevation
  - Unstable Angina
  - NSTEMI (Non-Q wave MI)
  - STEMI (Q wave MI)

**Non ST-Elevation Myocardial Infarction**

Left Circumflex Artery Stenosis

**ST-Elevation Myocardial Infarction**

Left Anterior Descending Artery Acute Total Occlusion

Unstable Angina (UA) and Non-ST Elevation Myocardial Infarction (NSTEMI)

I. Pathophysiologic Mechanisms
II. Diagnosis
III. Prognosis
IV. Management
V. Prevention

Clinical Presentation

- Chest pain or severe epigastric pain typical of myocardial ischemia or infarction:
  - Chest pressure, tightness, heaviness, cramping, burning, aching sensation
  - Unexplained indigestion, belching, epigastric pain
  - Radiating pain in neck, jaw, shoulders, back, or arm(s)
- Associated dyspnea, nausea/vomiting or diaphoresis

Electrocardiogram

- ST segment depression
  - 1 mm ≥ 2 contiguous leads
- T-wave inversion

Cardiac Biomarkers

- Troponin I or T
- CK, CK-MB
- Myoglobin
- Other
Unstable Angina (UA) and Non-ST Elevation Myocardial Infarction (NSTEMI)

I. Pathophysiologic Mechanisms
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TIMI Risk Score for UA/NSTEMI
Assessing Death, Myocardial Infarction or Urgent Revascularization

<table>
<thead>
<tr>
<th>HISTORICAL POINTS</th>
<th>RISK OF CARDIAC EVENTS (%) BY 14 DAYS IN TIMI 11B</th>
</tr>
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<tbody>
<tr>
<td>Age ≥ 65</td>
<td>1</td>
</tr>
<tr>
<td>≥ 3 CAD risk factors</td>
<td>1</td>
</tr>
<tr>
<td>(FHx, HTN, ↑ chol, DM, active smoker)</td>
<td>1</td>
</tr>
<tr>
<td>Known CAD (stenosis ≥ 50%)</td>
<td>1</td>
</tr>
<tr>
<td>ASA use in past 7 days</td>
<td>1</td>
</tr>
<tr>
<td>PRESENTATION</td>
<td></td>
</tr>
<tr>
<td>Recent (≤24h) severe angina</td>
<td>1</td>
</tr>
<tr>
<td>↑ cardiac markers</td>
<td>1</td>
</tr>
<tr>
<td>ST deviation ≥ 0.5 mm</td>
<td>1</td>
</tr>
</tbody>
</table>

RISK SCORE = Total Points (0 - 7)


Anti-Platelet Therapy
Aspirin 162 mg to 325 mg
PLUS:

- Before PCI:
  - Clopidogrel 600 mg (LOE: B) or
  - Ticagrelor 180 mg (LOE: B) or
  - GP IIb/IIIa inhibitor: eptifibatide or tirofiban (LOE: A)

- At the time of PCI (if not initiated):
  - Clopidogrel 600 mg (LOE: A) or
  - Ticagrelor 180 mg (LOE: B) or
  - Prasugrel 60 mg (LOE: B) or
  - GP IIb/IIIa inhibitor: including abciximab (LOE: A)

*Do not give if:
- <60 kg
- >75 years old
- h/o TIA/CVA

GP IIb/IIIa Inhibitor
Upstream vs. Time of Angiogram

- ACUITY Timing Trial\(^1\) (n=9207)
  - No difference in ischemia end-points
  - 30-day major bleeding in upstream (6.1%) vs. deferred (4.9%)

- EARLY ACS\(^2\) (n=9492)
  - No difference in ischemia end-points
  - 5 day non-life-threatening bleeding and transfusion with upstream


Beta-Blocker Therapy

Oral beta-blocker therapy should be initiated within the first 24 h for patients who do not have 1 or more of the following:

1. signs of heart failure
2. evidence of a low-output state
3. increased risk for cardiogenic shock*
4. other relative contraindications (PR interval >0.24 s, 2nd or 3rd degree AV block, active asthma/reactive airway disease)

* > 70 years, SBP < 120 mmHg, heart rate >100 or < 60 bpm

Anti-Coagulation

Initiate as soon as possible after presentation with one of the following:

- Unfractionated Heparin
- Enoxaparin
- Bivalirudin


Beta-Blocker Therapy

Reasonable to administer IV beta blockers at the time of presentation for hypertension who do not have 1 or more of the following:

1. signs of heart failure
2. evidence of a low-output state
3. increased risk for cardiogenic shock*
4. other relative contraindications (PR interval >0.24 s, 2nd or 3rd degree AV block, active asthma/reactive airway disease)

* > 70 years, SBP < 120 mmHg, heart rate >100 or < 60 bpm

Initial Invasive (Coronary Angiogram) Versus Conservative Strategy

Invasive
- Recurrent angina/ischemia at rest despite medical therapy
- Elevated cardiac biomarkers (TnT or TnI)
- New ST-segment depression
- Heart failure or new/worsening mitral regurgitation
- High-risk findings from noninvasive testing
- Hemodynamic instability
- Sustained ventricular tachycardia
- PCI within 6 months
- Prior CABG
- High risk score (e.g., TIMI, GRACE)
- Reduced left ventricular function (LVEF < 40%)

Conservative
- Low risk score (e.g., TIMI, GRACE)
- Patient/physician preference in the absence of high-risk features

All-Cause Mortality for Initial Invasive Versus Conservative Therapy 2 Year Follow-up

<table>
<thead>
<tr>
<th>Study</th>
<th>Invasive Deaths</th>
<th>Conservative Deaths</th>
<th>Follow-up Months</th>
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<tbody>
<tr>
<td>FAMOS-II</td>
<td>15</td>
<td>9</td>
<td>24</td>
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<tr>
<td>TRICE</td>
<td>3</td>
<td>0</td>
<td>12</td>
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<tr>
<td>TIMI-10</td>
<td>17</td>
<td>9</td>
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<td>6</td>
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<tr>
<td>MITA-II</td>
<td>102</td>
<td>132</td>
<td>80</td>
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<tr>
<td>ISAR-CDRULIC</td>
<td>0</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>ICTUS</td>
<td>15</td>
<td>0</td>
<td>12</td>
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</table>

After Coronary Angiogram Management Options

- **Medical therapy**
- **Coronary revascularization**
  - Percutaneous coronary intervention (PCI)
  - Coronary artery bypass surgery
  - Hybrid procedure (LIMA to LAD and PCI to all other vessels)
Anti-Platelet and Anti-Coagulation Therapy After Angiography


- Aspirin (Class I)
- Clopidogrel, Ticagrelor or Prasugrel (Class I)
- GP IIb/IIIa ≥ 12 h if started pre angi (Class I)
- D/C anti-coagulant after PCI (Class I)

No significant obstructive CAD

Physician discretion

• Aspirin (Class I)
• Clopidogrel or ticagrelor (Class I)
• Cont IV UFH ≥ 48 h, or enoxaparin or fondaparinux for duration of hospitalization (Class I)
• D/C GP IIb/IIIa (Class I)

Anti-Platelet and Anti-Coagulation Therapy After Angiography


- Con’t Aspirin (Class I)
- Cont UFH (Class I)
- D/C Clopidogrel or Ticagrelor ≥ 5 days and Prasugrel ≥ 7 days prior to CABG (Class I)
- D/C IV GP IIb/IIIa 4 h prior to CABG (Class I)
- D/C enoxaparin 12 to 24 h prior to CABG (Class I)
- D/C fondaparinux 24 hours before CABG (Class I)
- D/C bivalirudin 3 hours before CABG
I. Pathophysiologic Mechanisms

II. Diagnosis

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### Prevention

- Medical therapy
  - Anti-platelet
  - Statin
  - Beta-blocker
  - ACE inhibitor
- Management of other diseases (HTN, DM, etc)
- Exercise and Diet
- Tobacco cessation
- Other

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### Altered Clopidogrel Metabolism

- Clopidogrel conversion to active form via CYP 2C19; mutations in CYP 2C19 may result in lower active form of the drug
- Tests available to identify CYP2C19 genotype; however, insufficient evidence to recommend routine testing
- Consider higher clopidogrel dose regimen (150 mg daily) in poor metabolizers; however, appropriate dose not established
- Consider other anti-platelet medications
- Proton pump inhibitor – clopidogrel interaction?

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### Long-Term Anti-Platelet Therapy at Discharge

- **Medical Therapy without Stent**
  - Aspirin 81 mg indefinitely (Class IIa)
  - AND
  - Clopidogrel 75 mg/d or Ticagrelor* 90 mg q12h for up to 1 year (Class I)

- **Drug Eluting Stent OR Bare Metal Stent**
  - Aspirin 81 mg indefinitely (Class Iia)
  - AND
  - Clopidogrel 75 mg/d or Prasugrel 10 mg/d or Ticagrelor* 90 mg q12h for up to 1 year (Class I)

- **Indication for Anticoagulation?**
  - Yes
  - Add: Warfarin (INR 2.0 to 3.0) (Class IIb)
  - Continue with dual antiplatelet therapy as above (Class I)

  - No

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**Wright RS, et al. JACC. 2011;57:1920-1959.**

**Modified from Anderson JL, et al. JACC. 2007;50:e1-e157.**
**Lipid Management**

Achieve an LDL-C <100 mg/dL

- Further titration to < 70 mg/dL is reasonable (Class IIa, Level of Evidence: A)


**ACE-Inhibitor**

ACE inhibitors should be given and continued indefinitely for patients with HF, LVEF <40%, hypertension, or diabetes mellitus.

ACE inhibitors are reasonable for patients recovering from UA/NSTEMI in the absence of LV dysfunction, hypertension, or diabetes mellitus.


**Beta-Blocker Therapy**

Beta blockers are indicated for all patients recovering from UA/NSTEMI especially with LV systolic dysfunction unless contraindicated.


**Heart Outcomes Prevention Evaluation (HOPE Trial)**

- Patients with CAD or high-risk of developing CAD (n=9,297)
  - 52% prior MI, 25% UA
- No LV dysfunction or heart failure
- Ramipril 10 mg/day vs placebo
- Primary end point (myocardial infarction, stroke, or CV death):
  - 14.0% ramipril vs 17.8% placebo (p<0.001)
  - statistically lower for all individual endpoints

Blood Pressure Control

Blood pressure control according to JNC 7 guidelines is recommended (i.e., BP <140/90 mm Hg or <130/80 mm Hg if the patient has diabetes mellitus or chronic kidney disease).

Avoid NSAIDS and Estrogen/Progestin Replacement Therapy

- Increase risk of myocardial infarction and death.

Diabetes Mellitus

Diabetes management should include lifestyle and pharmacotherapy measures to achieve a near-normal HbA1c level of <7%.

Unstable Angina (UA) and Non-ST Elevation Myocardial Infarction (NSTEMI) Conclusion

- Most commonly caused by a decrease in myocardial perfusion by a non-occlusive thrombus that has developed on a disrupted atherosclerotic plaque resulting in luminal narrowing.
- Coronary angiogram should be performed to define coronary anatomy and need for coronary artery revascularization.
### Unstable Angina (UA) and Non-ST Elevation Myocardial Infarction (NSTEMI)

#### Conclusion

- Medical therapy should include aspirin, thienopyridine, β-blocker, ACE inhibitor and statin, regardless if revascularization performed.

- Coronary artery disease is progressive requiring close follow-up with particular attention to modifying risk factors:
  - smoking cessation, obesity, hypertension, dyslipidemia, diabetes mellitus, avoidance of NSAID and hormone replacement therapy, other