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

Konstantinos Dean Boudoulas, MD
Associate Professor of Medicine
Section Head, Interventional Cardiology
Director, Cardiac Catheterization Laboratory
The Ohio State University Wexner Medical Center

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.

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

I. Pathophysiologic Mechanisms
II. Diagnosis
III. Management
IV. Prevention
### 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 (ACS)

<table>
<thead>
<tr>
<th>ECG: No ST Elevation</th>
<th>ST Elevation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unstable Angina</td>
<td>NSTEMI (Non-Q wave MI)</td>
</tr>
<tr>
<td></td>
<td>STEMI (Q wave MI)</td>
</tr>
</tbody>
</table>


### Non ST-Elevation Myocardial Infarction

Left Circumflex Artery Occlusion

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

I. Pathophysiologic Mechanisms
II. Diagnosis
III. Management
IV. 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 (most sensitive/specific)
- CK, CK-MB
- Myoglobin
- Other

Guidelines/Level of Evidence

<table>
<thead>
<tr>
<th>Class</th>
<th>Benefit Risk</th>
<th>SHOULD be performed</th>
<th>REASONABLE to perform</th>
<th>MAY BE CONSIDERED</th>
<th>NOT be performed SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Class I</td>
<td>Benefit &gt;&gt;&gt; Risk</td>
<td>SHOULD be performed</td>
<td>REASONABLE to perform</td>
<td>MAY BE CONSIDERED</td>
<td>NOT be performed SINCE IT IS NOT HELPFUL AND MAY BE HARMFUL</td>
</tr>
<tr>
<td>Class II</td>
<td>Benefit &gt;&gt; Risk</td>
<td>REASONABLE to perform</td>
<td>MAY BE CONSIDERED</td>
<td>MAY BE CONSIDERED</td>
<td>MAY BE CONSIDERED</td>
</tr>
<tr>
<td>Class IIb</td>
<td>Benefit ≥ Risk</td>
<td>MAY BE CONSIDERED</td>
<td>MAY BE CONSIDERED</td>
<td>MAY BE CONSIDERED</td>
<td>MAY BE CONSIDERED</td>
</tr>
<tr>
<td>Class III</td>
<td>Risk ≥ Benefit</td>
<td>MAY BE CONSIDERED</td>
<td>MAY BE CONSIDERED</td>
<td>MAY BE CONSIDERED</td>
<td>MAY BE CONSIDERED</td>
</tr>
</tbody>
</table>

Level A: Recommendation based on multiple randomized trials or meta-analyses
Level B: Recommendation based on single randomized trial or non-randomized studies
Level C: Recommendation based on expert opinion, case studies, or standard-of-care

**Electrocardiogram**

- A 12-lead ECG should be performed with a goal of within 10 min of arrival
- Initial ECG is not diagnostic, serial ECGs at 15- to 30-min intervals

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**Cardiac Biomarkers**

- Serial cardiac troponin I or T levels should be obtained at presentation and 3 to 6 hours after symptom onset
- Additional troponin levels should be obtained beyond 6 hours after symptom onset in patients with normal troponin levels on serial examination with suspicion for ACS

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

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

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**Initial Anti-Platelet Therapy**

- Aspirin 162 mg to 325 mg
- Platelet P2Y12 Receptor Antagonists:
  - Clopidogrel 300 or 600 mg or Ticagrelor 180 mg
  - Ticagrelor in preference to Clopidogrel
**Initial Anti-Platelet Therapy**

GP IIb/IIIa inhibitor in patients treated with dual anti-platelet therapy with intermediate/high-risk features (e.g., positive troponin); preferred options are eptifibatide or tirofiban.

**Anti-Coagulation**

- Enoxaparin
  - continued for duration of hospitalization or until PCI performed
- Unfractionated heparin
  - continued for 48 hours or until PCI performed
- Bivalirudin
  - only with early invasive strategy

**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 & transfusion with upstream

**Bleeding Event Before Coronary Angiography and Death In Patients with NSTEMI**

- *More likely to have received:*
  - low-molecular-weight heparin (less likely bivalirudin)
  - upstream P2Y12 or GPIIb/IIIa inhibitors

### 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

Administration of intravenous beta blockers is potentially harmful in patients with NSTEMI who have risk factors for shock.

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### 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)

### Anemia and Transfusion

A strategy of routine blood transfusion in hemodynamically stable patients with ACS and hemoglobin levels > 8 g/dL is not recommended.
Unstable Angina (UA) and Non-ST Elevation Myocardial Infarction (NSTEMI)

I. Pathophysiologic Mechanisms
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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

Platelet P2Y12 Receptor Antagonists

<table>
<thead>
<tr>
<th></th>
<th>Plavix (Clopidogrel)</th>
<th>Effient (Prasugrel)</th>
<th>Brilinta (Ticagrelor)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Loading Dose</td>
<td>600 mg</td>
<td>60 mg (peak effect 2-4h)</td>
<td>180 mg (peak effect 2h)</td>
</tr>
<tr>
<td>Maintenance Dose</td>
<td>75 mg daily</td>
<td>10 mg daily</td>
<td>90 mg twice daily</td>
</tr>
<tr>
<td>Max % of Platelet Inhibition</td>
<td>30-50%</td>
<td>75-80%</td>
<td>75-80%</td>
</tr>
<tr>
<td>Time to 50% Inhibition</td>
<td>2-4 hours</td>
<td>Within 1 hour</td>
<td>Within 30-60 mins</td>
</tr>
<tr>
<td>Contraindications</td>
<td>Active bleeding</td>
<td>TIA or stroke</td>
<td>Intracranial hemorrhage</td>
</tr>
</tbody>
</table>

Long-Term Anti-Platelet Therapy at Discharge

UA/NSTEMI Patient Groups at Discharge

Medical Therapy without Stent

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* 90mg/d for up to 1 year (Class I)

Indication for Anticoagulation?

Yes

Add: Warfarin (INR 2.0 to 2.5) (Class IIIb, LOE: C)

No

Continue with dual antiplatelet therapy as above
## Lipid Management

- 2013 ACC/AHA Guideline on Treatment of Blood Cholesterol
  - high intensity statin therapy (atorvastatin 40/80 mg or rosvastatin 20 mg)

## Beta-Blocker Therapy

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

## ACE-Inhibitor

<table>
<thead>
<tr>
<th>Level</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>ACE inhibitors should be given and continued indefinitely for patients with LVEF &lt;40%, hypertension, diabetes mellitus, or stable chronic kidney disease</td>
</tr>
<tr>
<td>B</td>
<td>ACE inhibitors may be reasonable in all other patients with cardiac or other vascular disease</td>
</tr>
</tbody>
</table>

## 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
Aldosterone Blockade

Aldosterone blockade recommended in patients without significant renal dysfunction or hyperkalemia who are receiving therapeutic doses of ACE inhibitor and beta blocker, and have a LVEF ≤ 40%, diabetes mellitus, or heart failure.

Avoid NSAIDS and Estrogen/Progestin Replacement Therapy

- Increase risk of myocardial infarction and death

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.
- Medical therapy should include aspirin, P2Y12 receptor antagonist, β-blocker, ACE inhibitor (especially with LVEF <40%, hypertension, diabetes mellitus, or stable chronic kidney disease) and statin, regardless if revascularization performed.

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

**Conclusion**

- 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
ST Elevation Myocardial Infarction

Cindy Baker, MD, FACC
Clinical Assistant Professor
Division of Cardiovascular Medicine
Director of Peripheral Vascular Intervention
The Ohio State University Wexner Medical Center

Objectives

- Definition
- Statistics
- Reperfusion Strategies
- Drug Therapy
- Complications to Consider

STEMI- Definition

- new ST elevation at the J point in at least 2 contiguous leads of 2 mm in men
- 1.5 mm in women in leads V2–V3 and/or of 1 mm in other contiguous chest leads or the limb leads
- New or presumably new LBBB maybe considered a STEMI equivalent.
- ST depression in 2 precordial leads (V1–V4) may indicate posterior STEMI

The original uploader was Graham Child at English Wikipedia. Later versions were uploaded by Jrockley at en.wikipedia. Image uploaded in Commons by Maderibeyza and translated from English to Portuguese by Mateus Hidalgo. (CC BY-SA 3.0)
**Statistics**

- STEMI comprises 25-40% of myocardial infarction presentations
- In-hospital mortality 5-6%
- One year mortality 7-18% has significantly decreased with appropriate care including primary PCI and GDMT

*Benjamin EJ, et al. Heart Disease and Stroke Statistics 2017 Update, Report From The AHA*

**Statistics**

- Approximately 30% of patients with STEMI are women. Female sex is a strong independent predictor of failure to receive reperfusion therapy.
- Non-whites represent 13.3% of patients with STEMI. Disparities in care of racial and ethnic minorities appears to be improving over time.

*Benjamin EJ, et al. Heart Disease and Stroke Statistics 2017 Update, Report From The AHA*

**Incidence of STEMI**

133 per 100 000 person-years in 1999

50 per 100 000 person-years in 2008

*Yeh RW et al. N Engl J Med 2010;362:2155-2165*

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**Case Presentation**

55 year old male presents to an OSH with 6 hour history of chest pain. No significant past medical history. The hospital does not have PCI capability.

Vital Signs:
HR 104  BP 95/60  RR 16
Cardiovascular: RRR no murmurs appreciated
Lungs: bibasilar crackles
Extremities: cool with equal pulses
Questions to Consider??

- Reperfusion options
- Medical therapy
- Potential Post-MI complications

Reperfusion Options

<table>
<thead>
<tr>
<th>Patients with STEMI who are candidates for reperfusion therapy</th>
</tr>
</thead>
<tbody>
<tr>
<td>Initially seen at a PCI capable hospital</td>
</tr>
<tr>
<td>Initially seen at a non-PCI capable hospital</td>
</tr>
</tbody>
</table>

Reperfusion Options

- Initially seen at a PCI capable hospital
  - Send to cath lab for primary PCI
  - FMC-device time <= 90 mins
Reperfusion Options

Initially seen at a non-PCI capable hospital
(DIDO time <= 30 mins)

Transfer for primary PCI FMC-device time as soon as possible and <=120 mins

Administer fibrinolytic agent within 30 minutes of arrival when anticipated FMC-device >120 mins

Primary PCI in STEMI

<table>
<thead>
<tr>
<th></th>
<th>COR</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Ischemic symptoms 12 h</td>
<td>I</td>
<td>A</td>
</tr>
<tr>
<td>Ischemic symptoms 12 h and contraindications to fibrinolytic therapy irrespective of time delay from FMC</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Cardiogenic shock or acute severe HF irrespective of time delay from MI onset</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Evidence of ongoing ischemia 12 to 24 h after symptom onset</td>
<td>Ila</td>
<td>B</td>
</tr>
<tr>
<td>PCI of a noninfarct artery at the time of primary PCI in patients without hemodynamic compromise</td>
<td>III: Harm</td>
<td>B</td>
</tr>
</tbody>
</table>


Indications for Transfer for Angiography After Fibrinolytic Therapy

<table>
<thead>
<tr>
<th></th>
<th>COR</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Immediate transfer for cardiogenic shock or severe acute HF irrespective of time delay from MI onset</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Urgent transfer for failed reperfusion or reocclusion</td>
<td>Ila</td>
<td>B</td>
</tr>
<tr>
<td>As part of an invasive strategy in stable* patients with PCI between 3 and 24 h after successful fibrinolysis</td>
<td>Ila</td>
<td>B</td>
</tr>
</tbody>
</table>

*Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.

Indications for Coronary Angiography in Patients Who Were Managed With Fibrinolytic Therapy or Who Did Not Receive Repерfusion Therapy

<table>
<thead>
<tr>
<th></th>
<th>COR</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic shock or acute severe HF that develops after initial presentation</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Intermediate- or high-risk findings on predischarge noninvasive ischemia testing</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Spontaneous or easily provoked myocardial ischemia</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Failed reperfusion or reocclusion after fibrinolytic therapy</td>
<td>Ila</td>
<td>B</td>
</tr>
<tr>
<td>Stable* patients after successful fibrinolysis, before discharge and ideally between 3 and 24 h</td>
<td>Ila</td>
<td>B</td>
</tr>
</tbody>
</table>

### Indications for PCI of an Infarct Artery in Patients Who Were Managed With Fibrinolytic Therapy or Who Did Not Receive Reperfusion Therapy

<table>
<thead>
<tr>
<th>Indication</th>
<th>LOE</th>
<th>COR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiogenic shock or acute severe HF</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Intermediate- or high-risk findings on predischarge noninvasive ischemia</td>
<td>I C</td>
<td></td>
</tr>
<tr>
<td>Spontaneous or easily provoked myocardial ischemia</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>Patients with evidence of failed reperfusion or reocclusion after fibrinolytic therapy (as soon as possible)</td>
<td>IIA B</td>
<td></td>
</tr>
<tr>
<td>Stable* patients after successful fibrinolysis, ideally between 3 and 24 h</td>
<td>IIA B</td>
<td>IIA B</td>
</tr>
<tr>
<td>Stable* patients 24 h after successful fibrinolysis</td>
<td>IIb</td>
<td>B</td>
</tr>
<tr>
<td>Delayed PCI of a totally occluded infarct artery 24 h after STEMI in stable patients</td>
<td>III No Benefit</td>
<td>B</td>
</tr>
</tbody>
</table>

*Although individual circumstances will vary, clinical stability is defined by the absence of low output, hypotension, persistent tachycardia, apparent shock, high-grade ventricular or symptomatic supraventricular tachyarrhythmias, and spontaneous recurrent ischemia.


### Adjunctive Antithrombotic Therapy to Support Reperfusion With Primary PCI

#### Antithrombotic Therapy

<table>
<thead>
<tr>
<th>Therapy</th>
<th>LOE</th>
<th>COR</th>
</tr>
</thead>
<tbody>
<tr>
<td>Aspirin</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>- 162- to 325-mg load before procedure</td>
<td></td>
<td></td>
</tr>
<tr>
<td>- 81- to 325-mg daily maintenance dose (indeterminate)</td>
<td>I A</td>
<td></td>
</tr>
<tr>
<td>- 81 mg daily is the preferred maintenance dose</td>
<td>IIA B</td>
<td>IIA B</td>
</tr>
</tbody>
</table>

#### P2Y12 Inhibitors

<table>
<thead>
<tr>
<th>Loading doses</th>
<th>LOE</th>
<th>COR</th>
</tr>
</thead>
<tbody>
<tr>
<td>- Clopidogrel: 600 mg as early as possible or at time of PCI</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>- Prasugrel: 60 mg as early as possible or at time of PCI</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>- Ticagrelor: 180 mg as early as possible or at time of PCI</td>
<td>I</td>
<td>B</td>
</tr>
</tbody>
</table>

*The recommended maintenance dose of aspirin to be used with ticagrelor is 81 mg daily.


### A Platelet Inhibition and Patient Outcomes (PLATO) Trial

- Randomized controlled trial comparing ticagrelor to clopidogrel
- STEMI substudy showed ticagrelor had significant reduction in myocardial infarction, total mortality and definite stent thrombosis over clopidogrel.
- There was a low stroke rate but it was significantly higher in the ticagrelor group (1.7% vs 1%) p<0.02


### Anticoagulant Therapy

<table>
<thead>
<tr>
<th>Therapy</th>
<th>LOE</th>
<th>COR</th>
</tr>
</thead>
<tbody>
<tr>
<td>- UFH</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>- With GP IIb/IIIa receptor antagonist planned: 50- to 70-U/kg IV bolus to achieve therapeutic ACT‡</td>
<td>I C</td>
<td></td>
</tr>
<tr>
<td>- With no GP IIb/IIIa receptor antagonist planned: 70- to 100-U/kg bolus to achieve therapeutic ACT§</td>
<td>I</td>
<td>C</td>
</tr>
<tr>
<td>- Bivalirudin: 0.75-mg/kg IV bolus, then 1.75–mg/kg/h infusion with or without prior treatment with UFH. An additional bolus of 0.5 mg/kg may be given if needed.</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>- Reduced infusion to 1 mg/kg/h with estimated CrCl 30 mL/min</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>- Fondaparinux: not recommended as sole anticoagulant for primary PCI</td>
<td>III: Harm</td>
<td>B</td>
</tr>
</tbody>
</table>

*The recommended ACT with planned GP IIb/IIIa receptor antagonist treatment is 200 to 250 s.
‡ The recommended ACT with no planned GP IIb/IIIa receptor antagonist treatment is 200 to 250 s (HemoTec device) or 250 to 300 s (Hemocon device).
§ The recommended ACT with no planned GP IIb/IIIa receptor antagonist treatment is 300 to 350 s (Hemocon device).

Philippe Gabriel Steg et al. Journal of the American College of Cardiology Jan 2013, 61 (4) e78-e140; DOI: 10.1016/j.jacc.2012.11.019
Adjunctive Antithrombotic Therapy to Support Reperfusion With Primary PCI (cont.)

<table>
<thead>
<tr>
<th>PT/IIb/III Inhibitors</th>
<th>CON</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Maintenance doses and duration of therapy</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>DES placed: Continue therapy for 1 y with:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Lixiopidogrel: 75 mg daily</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>• Prasugrel: 10 mg daily</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>• Ticagrelor: 90 mg twice a day*</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>EES placed: Continue therapy for 1 y with:</td>
<td></td>
<td></td>
</tr>
<tr>
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<td>I</td>
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<td>I</td>
<td>B</td>
</tr>
<tr>
<td>DES placed:</td>
<td></td>
<td></td>
</tr>
<tr>
<td>• Lixiopidogrel, prasugrel, or ticagrelor* continued beyond 1 y</td>
<td>IIB</td>
<td>C</td>
</tr>
<tr>
<td>• Patients with STEMI with prior stroke or TIA: prasugrel</td>
<td>III</td>
<td>Harm</td>
</tr>
</tbody>
</table>

*The recommended maintenance dose of aspirin to be used with ticagrelor is 81 mg daily.
†Balloon angioplasty without stent placement may be used in selected patients. It might be reasonable to provide P2Y12 inhibitor therapy to patients with STEMI undergoing balloon angioplasty alone according to the recommendations listed for BMS. (LOE: C).


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).

Beta blockers should be continued during and after hospitalization for all patients with STEMI and with no contraindications to their use.

*Risk factors for cardiogenic shock (the greater the number of risk factors present, the higher the risk of developing cardiogenic shock) are age >70 years, systolic BP <120 mm Hg, sinus tachycardia >110 bpm or heart rate <60 bpm, and increased time since onset of symptoms of STEMI.


Renin-Angiotensin-Aldosterone System Inhibitors

An ACE inhibitor should be administered within the first 24 hours to all patients with STEMI with anterior location, HF, or EF less than or equal to 0.40, unless contraindicated.

An ARB should be given to patients with STEMI who have indications for but are intolerant of ACE inhibitors.

**Renin-Angiotensin-Aldosterone System Inhibitors**

- An aldosterone antagonist should be given to patients with STEMI and no contraindications who are already receiving an ACE inhibitor and beta blocker and who have an EF less than or equal to 0.40 and either symptomatic HF or diabetes mellitus.

- ACE inhibitors are reasonable for all patients with STEMI and no contraindications to their use.

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**Lipid Management**

- High-intensity statin therapy should be initiated or continued in all patients with STEMI and no contraindications to its use.

- It is reasonable to obtain a fasting lipid profile in patients with STEMI, preferably within 24 hours of presentation.

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**Post STEMI Complications**

- Cardiogenic shock
- Congestive heart failure
- Infarct expansion/ recurrent ischemia
- Arrhythmias- tachy and brady
- Pericarditis (Dressler’s syndrome)
- LV aneurysm
- Mechanical complications

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**Mechanical Complications**

- Rupture of LV free wall 0.5%
- Rupture of the interventricular septum 0.17%
- Acute mitral regurgitation 0.25%

*Significantly fewer mechanical complications since Primary PCIs performed*
Left Ventricular Free Wall Rupture

- Most die suddenly.
- Those presenting with contained rupture or pseudoaneurysm are typically hypotensive with cardiogenic shock. Echo is the diagnostic tool of choice to assess pericardial effusion and site of rupture.
- Emergent surgical repair is recommended for those surviving initial rupture.
- Pericardiocentesis is controversial.

Rupture Of The Interventricular Septum

- Typically occurs in large anterior or inferior MIs.
- Physical exam: pulmonary edema, harsh pansystolic murmur at left sternal border or thrill palpated
- Echo is diagnostic tool of choice to assess
- Right heart cath maybe necessary if echo not adequate. Evaluate for O2 step-up in PA to signify left-right shunt.
- Prompt surgical patch repair recommended. Occasionally percutaneous closure device can bridge patient to surgery.

Ventricular Septal Defect

- Typically occurs in large anterior or inferior MIs.
- Physical exam: pulmonary edema, harsh pansystolic murmur at left sternal border or thrill palpated.
- Echo is diagnostic tool of choice to assess.
- Right heart cath maybe necessary if echo not adequate. Evaluate for O2 step-up in PA to signify left-right shunt.
- Prompt surgical patch repair recommended. Occasionally percutaneous closure device can bridge patient to surgery.
Acute Mitral Regurgitation

- Typically from papillary muscle rupture or severe ischemia to the posterior papillary muscle.
- Physical exam: patient’s with severe pulmonary edema, pansystolic murmur at apex radiating to axilla.
- Echo is gold standard to diagnose but may need TEE to fully assess valve.
- Initial management is afterload reduction to stabilize patient followed by mitral valve repair and revascularization.

Conclusions

- Incidence of STEMI is decreasing but mortality remains elevated.
- Early reperfusion is primary goal to treatment.
- Goal directed medical therapy important prior to discharge
- Monitor for post MI complications – rare but high mortality