Acute Coronary Syndrome (ACS): Evidence Based Practice Throughout the Continuum

Seton Medical Center
Raising the Bar for Excellence in Cardiac Care

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Acute Coronary Syndrome (ACS)

- No ST Elevation
  - Non STEMI
  - Unstable Angina
- ST Elevation
  - STEMI
Hospitalizations in the U.S. due to Acute Coronary Syndromes

1,190,000 Hospital Discharges with primary or secondary diagnosis of ACS

<table>
<thead>
<tr>
<th>UA/NSTEMI</th>
<th>STEMI</th>
</tr>
</thead>
<tbody>
<tr>
<td>NRMI-4*: 71%</td>
<td>NRMI-4*: 29%</td>
</tr>
<tr>
<td>AHA Get with the Guidelines: 69%</td>
<td>AHA Get with the Guidelines: 31%</td>
</tr>
<tr>
<td>GRACE** Study: 62%</td>
<td>GRACE** Study: 38%</td>
</tr>
</tbody>
</table>

*NRMI-4: National Registry of Myocardial Infarction; **GRACE Study: Global Registry of Acute Coronary Events.
Acute Coronary Syndrome refers to any rupture of plaque or thrombotic event that leads to symptomatic ischemia or infarction.

STEMI

NonSTEMI / Unstable Angina
Pathophysiology of ACS

- Deposit of lipids, calcium, fibrin, and other cellular substances within the lining of the arteries.
- Initiates a progressive *inflammatory* response in an effort to heal the endothelium.
- End result of inflammatory process: the production of a fibrous atherosclerotic plaque.
- Plaque can progress to cause coronary stenosis
- Plaque can also rupture prior to causing significant stenosis
Plaque

- **Stable plaque of stable angina**
  - Thick fibrous caps separate the lipid core from the endothelium
  - Less complicated than vulnerable plaques
  - Tend to have smooth outlines

- **Venerable plaque of ACS**
  - Thin caps
  - Edge of the fibrous cap is a particularly vulnerable area and is commonly the location of ruptured plaque

- Limitations of stress testing and cardiac catheterization
- Intravascular ultrasound
Differential Diagnosis of Chest Pain

Assessment of Pain
Linking Patient History and Risk factors
Cardiac Biomarkers
ECG Findings
ACS Symptoms

Classic Symptoms
- Stable angina
- Unstable angina
- MI

Symptom Variations
- Women
- Elderly
- Diabetics
Stable Angina

- **Typical angina** is defined as angina that meets all three of the following characteristics:
  - Substernal chest discomfort with a characteristic quality and duration
  - Provoked by exertion or emotional stress
  - Relieved by rest or nitroglycerin.

- **Atypical angina** is defined as angina meeting two of the characteristics of typical angina.
  - Non cardiac chest pain is defined as chest pain with none or only one of the characteristics of typical angina.
Unstable Angina

- Occurs with minimal exertion
- OR increased dose of nitroglycerin is required to achieve relief.
- Prolonged rest angina is also considered unstable angina.
- Angina that increases in severity or is very severe on first presentation
- Caused by unstable or ruptured plaque that causes abrupt closure of a coronary artery which may spontaneously reperfuse.
Assessment of Angina

- **Quality:**
  - Use the word “discomfort” or “symptoms” when assessing
  - Many patients with dyspnea or chest pressure deny the presence of pain.

- **Location:**
  - Assessment of location includes radiation of symptoms.

- **Time:**
  - Both the time of onset and duration of symptoms

- **Aggravating and alleviating factors:**
  - Key in differentiating stable from unstable angina.

- **Reproducibility:**
  - Reproducibility of chest pain by applying pressure to the chest wall suggests a musculoskeletal etiology.
  - Does not completely rule out the presence of angina.
Characteristics of Angina

Sensation of pressure, tightness, heaviness, burning, or squeezing.
  - Rarely described as a sharp or stabbing pain.
  - Should not worsen with changes in position or respiration.

Location behind the sternum and in the upper back, shoulder, arm, jaw, or epigastric area.
  - Not usually located in the middle to lower abdomen and does usually not radiate to the lower extremities.

Associated symptoms (or stand alone symptoms) of dyspnea, nausea, palpitations, or diaphoresis.

Duration typically defined in minutes.
  - Not typically defined in seconds or hours.

CAUTION WHEN ASKING THE PATIENT ABOUT “PAIN”!
Angina in Women

- Delay presenting with symptoms
- Attribute symptoms to other non-cardiac causes
- Presentation
  - More epigastric discomfort
  - Less specific complaints: dyspnea or fatigue
  - More atypical (sharp) chest pain
    - WISE Study: 65% of women presented with atypical symptoms
- Symptoms of discomfort from nose to navel should be evaluated for presence of CAD
- Less documented stenotic disease of major epicardial coronary arteries
  - Altered microvascular and endothelial function
  - Downstream microembolization

WISE study- Women’s Ischemic Syndrome Evaluation
More on Women and Heart Disease

- Stable angina is often initial presentation
- Women with Non-STEMI and unstable angina are older than men and have more co-morbid conditions (diabetes and HTN)
- The average age for first MI is 64.7 years for men and 72.2 years for women (Go et al., 2013)
- Female sex is a risk factor for mortality in STEMI
- Women receive less evidence based therapies including reperfusion
1st ECG on arrival to ED @ 10:15am
53 y/o female smoker
Vague C/O chest pain, nausea
Next ECG on Arrival to CCU @ 4PM
3 Days Later
Angina in the Elderly

- Generalized symptoms
  - Dyspnea, diaphoresis, N&V, and syncope
  - Confusion
- Symptoms often attributed to the aging process
  - Importance of assessment with activity tolerance
- Don’t complain about chest pain
  - 37% of patients > 65
  - 42% of patients > 75 years
  - 75% of those > 85 years
- Silent MIs account for 60% of MIs in those > 85 years of age
  - STEMI
    - < 65 years = 90% pain
    - > 85 years = 57% pain
LBBB complicates the recognition of STEMI
- LBBB is present 33.8% of STEMI patients ≥ 85 years.
- STEMI recognized only 70% of time in patients > 85 years.

Often co-existing heart failure with the ACS diagnosis
- NSTEMI
  - 44% not diagnosed on admission
- HF with STEMI
  - < 65 years = 12%
  - > 85 years = 45%

Diagnosis of “Other”
- < 65 years = 5%
- > 85 years = 24%
Clinical Features in the Older Adult

- Age related changes that affect the cardiovascular system:
  - Decreased arterial compliance
  - Increased cardiac afterload
  - Diastolic dysfunction of the left ventricle.
  - Inflammatory dysregulation

- Other changes:
  - Declining lean body mass
  - Functional and cognitive changes
Markers of Risk: Specific to Elderly

- Mobility and function
  - Activities of daily living
  - Strength
- Frailty: state of declining reserves in physical strength and functional status
- Poor Nutrition Status
  - Albumin
  - Weight loss
- More co-morbid conditions
- Altered renal and hepatic function
- Poly pharmacy
- Cognitive Impairment
- Hearing Alterations
- Vision Alterations
- Isolation
- Resources / Education
- Socioeconomic
Age Related Risk for Mortality & Morbidity

**Non-STEMI**

- ≥ 75 years of age: high risk for short term death or non-fatal MI.
- In hospital death:
  - < 65 years: 1 in 100

1 year mortality rate:
- > 75 years: 1 in 5
- > 85 years: 1 in 4

**STEMI**

- Morbidity and mortality after STEMI also increased due to electrical and mechanical complications.
- Heart failure and pulmonary edema occur in more than half of patients ≥ 75 years
- Shock occurs in > 10% of patients ≥ 75 years.
Angina and CAD in Diabetics

- Autonomic dysfunction can affect symptoms experienced with angina
- Less likely to experience pain.
- Approximately 20 - 25% of all patients presenting with ACS have diabetes
- More severe multi-vessel disease
- Greater proportion of ulcerated plaques resulting in intracoronary thrombi
- Higher rates of complications from ACS, higher mortality, and high rates of sudden death
Acute MI Symptoms

Symptoms occur spontaneously and are not relieved by rest or nitroglycerin

Chest pressure or discomfort may be accompanied by nausea, vomiting, or diaphoresis

Patient may have hemodynamic instability or cardiac arrest from ventricular fibrillation

Acute MI patients have positive biomarkers and are classified as STEMI or NSTEMI based on ECG presentation
STEMI

< 25% of ACS patients
Complete occlusion of a vessel by a thrombus
Fibrin stable clot (red clot)
Classified more specifically by the portion of the left ventricle suffering injury.
Mortality is greatest within the first 24 to 48 hours of symptom onset

TREATMENT FOCUS = REPERFUSION
Nationally under treated according to evidence based practice guidelines (Crusade Registry)

Pathophysiology often involves a platelet plug or white clot

Less stable clot

Opportunity for spontaneous reperfusion

Differentiated from unstable angina by troponin levels

TREATMENT FOCUS = ANTIPLATELET THERAPY
Causes of UA/NSTEMI*

- Thrombus or thromboembolism, usually arising on disrupted or eroded plaque
  - Occlusive thrombus, usually with collateral vessels
  - Subtotally occlusive thrombus on pre-existing plaque
  - Distal microvascular thromboembolism from plaque-associated thrombus
  - Thromboembolism from plaque erosion
- Non–plaque-associated coronary thromboembolism
- Dynamic obstruction (coronary spasm or vasoconstriction) of epicardial and/or microvascular vessels
- Progressive mechanical obstruction to coronary flow
- Coronary arterial inflammation
- Secondary UA
- Coronary artery dissection

Evaluation of Oxygen Supply and Demand

- Increase myocardial oxygen demand:
  - Hyperthermia
  - Hypertension
  - Tachycardia
  - Conditions producing over stimulation of the sympathetic nervous system (cocaine use, hyperthyroidism)

- Decrease myocardial oxygen delivery:
  - Anemia
  - Pulmonary disease.

- Increase myocardial oxygen demand and decrease myocardial oxygen supply:
  - Aortic stenosis
  - Hypertrophic cardiomyopathy

Elderly are at risk for secondary coronary events related to supply and demand imbalance.
Cardiac Risk Factors

- Non-Modifiable Risk Factors
  - Previous history
  - Family history
    - 1st degree relative (parents, siblings)
    - Men < 55; Women < 65
  - Age
  - Gender
  - Socioeconomic Factors and Ethnicity

- 9 easily measured and potentially modifiable risk factors account for over 90% of the risk of an initial acute MI
  - Smoking
  - Hypertension
  - Dyslipidemia
  - Diabetes
  - Obesity
  - Metabolic Syndrome
  - Inactivity
  - Alcohol

Mortality Rate Age > 40 years:
- 1 year: F - 23%, M - 18%
- 5 year: F - 43%, M - 33%
Other Pertinent History

- CAD
- Cerebral Vascular Disease
- Peripheral Vascular Disease
Cardiac Biomarkers

- Released into the blood when necrosis occurs as a result of membrane rupture of the myocytes
- Used in the evaluation of ACS
- Myoglobin
  - Rises the earliest
  - Within 2 hours after damage
  - Very sensitive, not specific
- CK (creatine kinase)
  - Enzyme present in the heart, brain, and skeletal muscle
  - Elevations are not specific to myocardial damage.
- CK-MB
  - More specific to the heart
  - Helpful in identifying more than minor amounts of myocardial damage
  - Rapidly rises in the presence of myocardial damage.
Cardiac Biomarkers
Troponin I and T (cardiac troponins)

- Found only in cardiac muscle
- Most sensitive indicator of myocardial damage
  - Capable of diagnosing small amounts of myocardial necrosis not measured by rises in CK-MB levels
- Approximately 30% of patients with non-ST elevation and normal CKMB levels will test positive for Non-STEMI
- Of equal sensitivity and specificity
- Troponin remains elevated for a long period
  - Beneficial for late presentation
  - Challenging for re-infarction
- Positive troponin + ECG changes of injury / ischemia or ACS symptoms = infarct
Non infarct cardiac causes of elevated troponin: heart failure, left ventricular hypertrophy, tachyarrhythmias, pericarditis, cardiac trauma

Non CAD causes of troponin elevation (sepsis, pulmonary emboli, chronic kidney disease, chemotherapy, respiratory failure, burns, neurological disease)

Troponin I more specific in renal dysfunction
- Patients with ESRD commonly have elevated troponin T
  - Not a false positive - relates to overall dysfunction of the cardiorenal system
- < 10% of patients with ESRD have elevated troponin I in absence of ACS

Elevated troponin levels are marker of risk and associated with an increased mortality – even when diagnosis is not myocardial infarction

Degree of troponin elevation correlates with risk of death
# Cardiac Biomarker Summary

<table>
<thead>
<tr>
<th>Cardiac Biomarker</th>
<th>Specificity / Sensitivity</th>
<th>Rise</th>
<th>Peak</th>
<th>Duration</th>
</tr>
</thead>
<tbody>
<tr>
<td>Myoglobin</td>
<td>Sensitive but not specific</td>
<td>Within 2 hours</td>
<td>4 to 10 hours</td>
<td>&lt; 24 hours</td>
</tr>
<tr>
<td>CK-MB</td>
<td>Highly specific</td>
<td>4 to 6 hours</td>
<td>18 to 24 hours</td>
<td>2 to 3 days</td>
</tr>
<tr>
<td>Troponin I or T</td>
<td>Highly specific and sensitive</td>
<td>4 to 6 hours</td>
<td>18 to 24 hours</td>
<td>10 or more days</td>
</tr>
</tbody>
</table>
Timing of Release of Various Biomarkers After Acute Myocardial Infarction


Anderson JL, et al. J Am Coll Cardiol 2007;50:e1–e157, Figure 5.
Chest pain or severe epigastric pain, non traumatic in origin, with components typical of myocardial ischemia or MI:
- Central/substernal compression or crushing chest pain
- Pressure, tightness, heaviness, cramping, burning, aching sensation
- Unexplained indigestion, belching, epigastric pain
- Radiating pain in neck, jaw, shoulders, back, or 1 or both arms

Associated dyspnea
Associated nausea/vomiting
Associated diaphoresis

If non diagnostic:
- Repeat q 15 to 30 minutes
- Use ST segment monitoring
- Perform V7-V9
ECG Criteria for STEMI

**Diagnostic Criteria**

- New ST elevation at the J point in at least 2 contiguous leads
  - > 2 mm in men and > 1.5 mm in women for leads V\textsubscript{2} and V\textsubscript{3}
  - > 1 mm for other chest leads and limb leads on standard 12 lead
  - > 0.5 mm in leads V\textsubscript{7}-V\textsubscript{9}

**Special Considerations**

- True Posterior MI
  - No ST segment elevation on ECG.
  - Look for ST segment depression ≥ 2 mm in leads V\textsubscript{1}-V\textsubscript{4}
  - Hyperacute T wave changes
    - May be observed early in a before the J point elevates & the ST segment significantly lifts off baseline.
- Special criteria exist for interpreting the ECG in the presence of LBBB.
- Baseline ECG abnormalities such as left ventricular hypertrophy, ventricular paced rhythm, and Brugada syndrome make the interpretation of the ECG challenging.
Reperfusion is number one treatment strategy

Primary Coronary Intervention (PCI) preferred treatment strategy if within 90 minutes
  - Goal: 90 minutes from 1st medical contact

Fibrinolytics within 30 minutes of hospital presentation (or 30 minutes from EMS to fibrinolytics)

Facilitated PCI with full dose fibrinolytics is not recommended.

Rescue PCI may be done after failed fibrinolytics
Reperfusion Therapy

Fibrinolytic Therapy

Primary PCI

However:
Timely reperfusion is the priority over method of reperfusion.
Primary PCI

Benefits

• Higher infarct artery patency
• Lower rates of recurrent ischemia or infarction
• Lower rates of intracranial bleeding
• Lower rates of death

Considerations

• No-reflow phenomenon
• Procedure site bleeding
• Use of DES
  • Considered safe
  • Cobalt chromium everolimus stents
  • Dual antiplatelet therapy
• PCI of non-infarct artery
Fibrinolytic Therapy

Fibrin Specific Agents

- Tenecteplase (TNK-tPA)
- Reteplase (rPA)
- Alteplase (tPA)

Successful Reperfusion:

- **BEST INDICATOR:** Relatively sudden and complete relief of chest pain combined with > 70% ST segment resolution in the lead where there was the greatest ST elevation.
- Other useful indicator: Complete or nearly compete ST segment resolution 60 to 90 minutes after therapy
Fibrinolytics

**Absolute Contraindications:**

- Prior intracranial hemorrhage
- Known structural cerebral vascular lesion
- Malignant intracranial neoplasm
- Significant closed-head injury or facial trauma within last 3 months
- Intracranial or intraspinal surgery within 2 months
- Ischemic stroke within last 3 months (unless acute stroke within last 4.5 hours)
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Severe uncontrolled hypertension (unresponsive to treatment)
Fibrinolytic Complications

- Intracranial hemorrhage:
  - Any neurological change within 24 hours should be considered and intracranial hemorrhage until proven otherwise
  - All therapy including anticoagulants and antiplatelets discontinued
  - Treat with: Fresh frozen plasma and cryoprecipitate for clotting factors, platelets, and protamine
  - Other treatment to decrease intracranial HTN
  - May require surgical intervention for evacuation
Fibrinolytics

**Issues specific to elderly:**
- Poorly controlled HTN
- Prior CVA
- Dementia
- Chronic Anticoagulation
- Patient Preference
Reasons for Delayed or Missed Reperfusion Therapy

- Missed diagnosis of unequivocal ECG due to atypical symptoms
- Unrecognized unequivocal ECGs
- Delay in diagnosis of subtle ECGs or failure to perform serial ECGs
- Delay in administration of therapy or inappropriate abortion of treatment.
  - Resolution of pain alone is not an indication for aborting therapy. Look for 50-100% resolution of ST-segment elevation before considering suspending reperfusion therapy based on further assessment.
Each community should develop a STEMI system of care consistent with minimum standards of AHA’s Mission Lifeline

Door to device time alone is not sufficient to further reduce mortality

The average time of presentation after symptom onset is 1.5 to 2.0 hours.

Patient populations with the longest delays are women, blacks, and the elderly.

Nurses can make an impact through patient and community education and awareness campaigns.

60 minutes is the golden hour: Survival rates improve significantly.
Medical Management of STEMI

- ASA: 325 mg (non enteric coated)
  - If fibrinolytic therapy – 162-325 mg
- P2Y12 inhibitor (loading dose before or at time of PCI)
  - If fibrinolytic therapy - clopidogrel only
- Anticoagulants (related to reperfusion strategy)
  - If fibrinolytic – weight based heparin x 48 hours
- Oral beta blockers ASAP
  - IV if hypertensive or tachycardic
- NTG – Sublingual vs IV
- Morphine Sulfate (Class I)
- Oxygen if hypoxemic (arterial oxygen saturation < 90%)
- High intensity statin therapy
- D/C NSAIDS
- ACE Inhibitors (within 24 hours)
  - Greatest benefit in anterior wall MI, LVEF < 40%, HTN, diabetes or chronic kidney disease
- Aldosterone Antagonists
  - Initiate within 7 days in those with LVEF <40% , HF , or diabetes

Reperfusion is primary management strategy.
Treatment of Non STEMI / Unstable Angina

- Attacking Platelet is number one treatment strategy

- There are 3 Types of Antiplatelet Agents
  - Aspirin
  - P2Y12 Receptor Antagonists
  - Intravenous GP IIb/IIIa Inhibitors

- Anticoagulation is another indirect way to cool off platelet activity
Dual antiplatelet therapy for invasive strategies in medium to high risk patients
- ASA (and one of following)
- Clopidogrel (600 mg loading)
- Prasugrel (60 mg loading)
- Ticagrelor (180 mg loading)
- GP IIb/IIIa Inhibitors (*eptifibatide, *tirofiban, abciximab)
  - * preferred agents

Antiplatelet therapy also in conservative treatment
- Prasugrel not unless PCI is planned
- Abciximab not unless PCI is planned
P2Y$_{12}$ Receptor Inhibitors

Thienopyridines

• Clopidogrel (Plavix)
• Prasugrel (Effient)

Non thienopyridine

• Ticagrelor (Brilinta)
Medications and Safety

- Use of warfarin in conjunction with aspirin and/or P2Y$_{12}$ receptor inhibitor therapy is associated with an increased risk of bleeding, and patients and clinicians should watch for bleeding, especially gastrointestinal.

- Extreme caution with triple therapy.

- Particular increased risk for GI bleeding.

- Creatinine clearance should be estimated in UA/NSTEMI patients and the doses of renally cleared medications should be adjusted according to the pharmacokinetic data for specific medications.
It is reasonable to use an insulin-based regimen to achieve and maintain glucose levels less than 180 mg/dL while avoiding hypoglycemia* for patients with STEMI with either a complicated or uncomplicated course.
Treatment of NSTEMI / UA

- ASA
- Oxygen (1st 6 hours)
- NTG
  - IV in first 48 hours for persistent ischemia, HTN, HF
  - Should not interfere with mortality reducing beta blockers or ace inhibitors
- MS (if NTG unsuccessful and other anti ischemic drugs on board)
- Beta Blockers (within 24 hours)
  - Start PO when hemodynamically stable
  - May use IV if hypertensive
- ACE Inhibitors (within 24 hours)
  - In select patients – pulmonary congestion or LVEF ≤ 40% – may also be used in other patients

Medical Supportive Therapy: Similar to STEMI

Mortality Benefit vs. Symptom Relief
Early Invasive Option in UA / NSTEMI

- What is it?
  - Not waiting for failed medical treatment
  - Not waiting for + noninvasive test
  - Angiography with intent of revascularization
  - Done within 12 to 24 hours

Overall reduction in mortality and increased quality of life.
Early Invasive Option in UA / NSTEMI

- When to do it?
  - Refractory angina
  - Hemodynamic instability
  - Electrical instability
  - Initially stable patients with a high risk for clinical events

- Excluded: very frail elderly, severe hepatic, renal or pulmonary disease / active or inoperable cancer

- Early invasive therapy is not recommended in patients with acute chest pain with a low likelihood of ACS

- Early invasive therapy is not recommended in patients who do not want to consent to revascularization.
High Risk Features in UA / NSTEMI

- Recurrent angina / ischemia
  - Rest or low level activity with medical treatment
- Troponin +
- New or presumed new ST depression
- S&S HF or worsening mitral regurgitation
- High risk findings on noninvasive testing
  - EF < 35%, large anterior perfusion defect, multiple perfusion defects
- Hemodynamic instability
- Sustained VT
- PCI within 6 months
- Prior CABG
- Reduced LV Function
- High risk TIMI or GRACE Score

Population > 75 years: 80% are high risk

Elderly:
cancer, renal insufficiency, lung disease, anemia, and heart failure are common co morbid conditions
Risk Assessment in UA / NSTEMI

- **TIMI Risk Score**
  - Age > 65
  - 3 or > risk factors for CAD
  - Prior 50% or > stenosis
  - ST deviation on ECG
  - 2 or > anginal events in previous 24 hours
  - Use of ASA in prior 7 days
  - Elevated cardiac biomarkers

- **GRACE**
  - Older age
  - Killip class
  - Systolic BP
  - Cardiac arrest during presentation
  - Serum creatinine
  - Positive initial cardiac markers
  - HR
<table>
<thead>
<tr>
<th>Feature</th>
<th>High Risk</th>
<th>Intermediate Risk</th>
<th>Low Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>≥ 1 of the features below must be present:</td>
<td>No high-risk features, but must have 1 of the following:</td>
<td>No high- or intermediate-risk features but may have any features below:</td>
</tr>
<tr>
<td>History</td>
<td>Accelerating tempo of ischemic sx in preceding 48 h</td>
<td>Prior MI, peripheral or cerebrovascular disease, or CABG; prior ASA use</td>
<td></td>
</tr>
</tbody>
</table>
| Character of pain | Prolonged ongoing (> 20 min) rest pain | • Prolonged (> 20 min) rest angina, now resolved, w/ moderate/high likelihood of CAD  
• Rest angina (> 20 min) or relieved with rest or sublingual NTG  
• Nocturnal angina  
• New-onset or progressive CCS class III/IV angina in past 2 wks w/o prolonged (> 20 min) rest pain but with intermediate/high likelihood of CAD  
 • ↑ Angina frequency, severity or duration  
 • Angina provoked at lower threshold  
 • New onset angina with onset 2 wks to 2 mos prior to presentation |
### Short-Term Risk of Death/Nonfatal MI in Patients With UA/NSTEMI, Continued

<table>
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<tr>
<th>Feature</th>
<th>High risk</th>
<th>Intermediate risk</th>
<th>Low risk</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical findings</strong></td>
<td>• Pulmonary edema, most likely due to ischemia</td>
<td>Age &gt; 70 y</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• New/worsening MR murmur</td>
<td></td>
<td></td>
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<tr>
<td></td>
<td>• S₃ or new/worsening rales</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Hypotension, bradycardia, tachycardia</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Age &gt; 75 y</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>ECG</strong></td>
<td>• Angina @ rest with transient ST-segment changes &gt; 0.5 mm</td>
<td>• T-wave changes</td>
<td>Normal or unchanged ECG</td>
</tr>
<tr>
<td></td>
<td>• BBB, new/presumed new</td>
<td>• Pathological Q-waves/resting ST-</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Sustained VT</td>
<td>depression &lt; 1 mm in multiple lead</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>groups (anterior, inferior, lateral)</td>
<td></td>
</tr>
<tr>
<td><strong>Cardiac markers</strong></td>
<td>↑ Cardiac TnT, TnI, or CK-MB (e.g., TnT/TnI &gt; 0.1 ng/mL)</td>
<td>Slightly ↑ cardiac TnT, TnI, or CK-MB</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(e.g., TnT &gt; 0.01, but &lt; 0.1 ng/mL)</td>
<td></td>
</tr>
</tbody>
</table>

Estimation of the short-term risk of death and nonfatal cardiac ischemic events in UA/NSTEMI is a complex multivariable problem that cannot be fully specified in a table such as this; this table is meant to offer general guidance & illustration rather than rigid algorithms. Braunwald E, et al. AHCPR Publication No. 94-0602:1–154. Anderson JL, et al. *J Am Coll Cardiol* 2007;50:e1–e157, Table 7.
Other Medication Considerations

- Hold ace inhibitors for BP < 100 mm Hg systolic or < 30 mm Hg below baseline

- No IV ace inhibitor within 24 hours due to risk of hypotension

- Start beta blocker prior ACE-inhibitor

- No immediate release dihydropyridine calcium channel blockers without beta blockade on board – otherwise reflex tachycardia will occur
Add ASA 81 mg and PPI to patients at increased risk of thrombotic events *

- Acetaminophen, ASA, tramadol, narcotic analgesics (short term)
- Nonacetylated salicylates
- Non COX-2 selective NSAIDs
- NSAIDs with some COX-2 activity
- COX-2 Selective NSAIDs

Regular monitoring for sustained hypertension or worsening of prior blood pressure control, edema, worsening renal function, or gastrointestinal bleeding.

If these events occur, consider reduction of the dose or discontinuation of the offending drug, a different drug, or alternative therapeutic modalities, as dictated by clinical circumstances.

NSAIDS (except for ASA), whether nonselective or COX-2–selective agents increase risk of mortality, reinfarction, hypertension, HF, and myocardial rupture

Medications to improve prognosis

- **Aspirin**
  - ASA benefits > in those ≥ 65 years
  - Long term benefit with 81 mg

- **Clopidogrel / Prasugrel / Ticagrelor**
  - Dual antiplatelet therapy in conservative management for up to 12 months
    - Clopidogrel or Ticagrelor
  - Higher risk of bleeding with dual antiplatelet therapy
    - No elderly sub group data for clopidogrel

* Beta blockers and ACE inhibitors impact long term ventricular remodeling
Medications to improve prognosis

- Beta-blockers
- ACE inhibitors (definite in select patients / reasonable in all)
  - ARBs if ACE-I intolerant
- Aldosterone antagonists (EF ≤ 40 with HF or diabetes)
- Lipid-lowering drugs (statins)
  - Have greater benefit in elderly for reduction of future MI and death than in younger patient populations
Secondary Prevention: ACS and Stable CAD

- Smoking cessation
- Reduction of hyperlipidemia
  - LDL < 100 mg/dL or < 70 mg/dL (optimal)
- Hypertension control
  - <130/80 for kidney disease or diabetes
- Diabetes control Hb A1c < 7
- Physical activity minimum of 5 days / per week
  - 7 days recommended
- BMI 18.5 – 24.9 kg/mm²
- Phase II Cardiac Rehab
- Influenza Vaccine / Pneumonia Vaccine
SL NTG Instruction

- No more than 1 dose of SL NTG
  - If chest discomfort is unimproved or is worsening 5 min after 1 NTG call 9-1-1 immediately before taking additional NTG.
  - May take additional NTG while waiting EMS.
  - Chew ASA while waiting EMS.

- In chronic stable angina if symptoms are significantly improved by 1 dose of NTG may repeat NTG every 5 min for a maximum of 3 doses and call 9-1-1 if symptoms have not resolved completely.
Medical Therapy Issues in the Elderly

- Altered responses and vulnerability to drugs with:
  - Hypotensive action (nitrates, calcium blockers)
  - Cerebral effects (beta blockers)
  - Caution with renally cleared drugs

START LOW and GO SLOW!!
Treatment of Stable Angina

- Medical Treatment
  - Lipid-lowering therapy
  - Antiplatelet therapy
  - Antianginal therapy
- Revascularization (* if criteria are met)
  - Primary Coronary Intervention
  - Coronary Artery Bypass Graft
- Aggressive risk factor modification
Key Nursing Care Considerations

- Assess response to beta-blocker therapy.
  - HR / BP
  - Arrhythmia control
  - Need for higher / lower dose
- Reassess oxygen saturation after 6 hours and discontinue $O_2$ if saturation is more than 90%.
- Assess for complications related to specific type of MI
  - Assess heart sounds for new holosystolic murmurs
    - Risk for myocardial rupture
  - Observe for signs of left ventricular dysfunction, including hypotension or clinical signs of heart failure.
  - Monitor ECG for conduction disturbances and arrhythmias
  - Assess for presence of RV infarct
Key Nursing Care Considerations

- Restrict activity for at least the first 12 hours, and then begin Phase I Cardiac Rehabilitation
  - Referral to Phase II Cardiac Rehabilitation

- Utilize cardiac monitoring
  - ST-segment monitoring
  - Uninterrupted monitoring for first 24-48 hours

- Focus on holistic approach to anxiety reduction
  - Include the family. Family visits do not have a negative impact on vital signs or cardiac rhythm

- Address addiction to nicotine
  - Consideration for nicotine withdrawal
  - Specific smoking cessation plan
Complications of MI
Anterior MI

- Proximal LAD
  - Proximal to first diagonal
    - Anterolateral
  - Proximal to first septal perforator
    - Anteroseptal
- Mid LAD
  - Anterior MI

LAD: Anterior Wall, High Lateral Wall, Septum
Complications of Anterior MI

- Myocardium at risk
- Mortality and morbidity
- Post Infarction ejection fraction

Urgency for Reperfusion!
Complications of Anterior MI

- Tachycardia
  - Sinus tachycardia
  - Atrial tachycardia
  - Ventricular tachycardia
- Right BBB and left anterior hemiblock
- Complete heart block
- Ventricular septal defect
  - New loud systolic murmur
- Cardiogenic shock
- Long term ventricular modeling and heart failure
Inferior MI

- RCA occlusion 80% to 85% of time
  - Marginal branch: Right ventricle
  - Posterior descending artery = Posterior wall of LV
    - Concept of right versus left dominant

Clinical application: Assess right sided leads in patients with inferior MI.
Inferior MI

- Variations
  - Inferior posterior
  - Inferior with RV
  - Inferior posterior and RV
  - Inferolateral (often with circumflex)

- Complications
  - Sinus Bradycardia, 1st degree and 2nd Degree HB Type I
  - Increased parasympathetic activity
  - Papillary muscle rupture with posterior wall involvement
  - RV failure with RV involvement
Lateral Wall MI

- Lateral wall MIs are frequently associated with anterior, inferior, or posterior wall MIs
- However – when isolated are frequently missed
  - ST elevation may be < 1 mm
  - ST elevation may only be in aVL

Myocardium at Risk and Mortality Benefit Warrant Reperfusion Therapy
Cellular edema produces an inflammatory response.
Recruitment of some stem cells leads to some tissue regeneration.
Damaged tissue is bruised and cyanotic.
Catecholamines are released from myocardial cells, thus increasing the risk of arrhythmias.
Cardiac biomarkers are released.
White blood cells invade the necrotic tissue within 2 to 3 days.
Scavenger cells release enzymes to break down necrotic tissue.
The necrotic wall can become very thin during this phase, and the patient is at risk for cardiac rupture.

**LINKING KNOWLEDGE TO PRACTICE:** Beta blockers are particularly important in suppressing cardiac arrhythmias in ischemic tissue during the acute remodeling phase because they suppress catecholamine release.
Long Term Ventricular Remodeling

- A weak collagen matrix forms by second week, myocardium is still vulnerable to re-injury.
- Scar formation has started by third week.
- Necrotic area is completely replaced with scar tissue by week 6. Scar tissue does not contribute to the contractile function of the myocardium.
- Myocardial necrosis (transmural and non transmural) and stunned or hibernating viable myocardium adversely affect the synergy of left ventricular contraction.
- Surviving myocytes hypertrophy in an attempt to compensate for damaged tissue.
- Excessive non-contractile collagen is present in the newly hypertrophied myocardium, leading to a ventricle that is stiff and noncompliant.
- Regional wall motion dysfunction may improve due to recovery of post-ischemic viable myocardium. Does not necessarily correlate with an improvement in overall left ventricular ejection fraction.
- Non-uniform left ventricular dilatation occurs. Occurs even in patients with recovery of regional wall motion abnormalities.
Left Ventricular Remodeling Following Myocardial Infarction

Acute Infarction, hours

Infarct Expansion, hours to days

Global Remodeling, days to months
Progressive adverse LV remodeling

Acute MI  At one year

Alteration in Myocyte architecture, Size, Shape and Contractility

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Hemodynamic Alterations

**Pre Shock Hypoperfusion**
- Cold extremities
- Cyanosis
- Oligurua
- Decreased mentation

**Shock**
- Large LV infarction (> 40% myocardium)
- Right ventricular infarct
- Mechanical complication

Heart Failure:
- **Cause:** Ischemic, stunned, hibernating, or injured myocardium.
- HF after a STEMI is a predictor of mortality.
- Functional mitral valve regurgitation can co-exist.
Ventricular Arrhythmias

No treatment needed: AIVR, PVCs, & non-sustained VT.

Early
- V-fib preventable cause of death
- 90% of sustained arrhythmias occur in first 48 hours

Treatment:
- Reperfusion
- Beta blocker therapy
- Correction of electrolytes
- Treatment of hemodynamic alterations

> 48 hours post MI
- Higher mortality than arrhythmias early in course
- ICD consultation if no reversible cause
12 Lead ECG Post Inferior STEMI on Arrival to CCU

Vital Signs Stable

12 lead ECG Computer Interpretation:

- Atrial Fibrillation
- RBBB with Left Anterior Hemiblock
High Grade AV Block with RCA Occlusion
RBBB with Wrap Around LAD Occlusion
Alternating LBBB with Wrap Around LAD Occlusion
Pericarditis

- Transmural infarct extending to the epicardium and causing an inflammatory response
- Decreased incidence with reperfusion
- Persistent pain > one week: Dressler’s syndrome
  - Accompanied by fever and malaise

- High dose (650 mg) enteric coated aspirin every 4-6 hours can be used
  - If pain is not controlled with aspirin, then colchicine, acetaminophen, or narcotic agents can be used
  - Non-steroidal anti-inflammatory medication and glucocorticoids should not be used due the increased risk of myocardial scar thinning
True Ventricular Aneurysm

- Occurs in approximately 5% of STEMI patients
- If no reperfusion, the incidence of ventricular aneurysm is as high as 10% to 30%
- More common with transmural anterior wall MI patients
- Localized myocardial wall thinning and bulging of the left ventricle at the site of infarction
- Stretched portion of the myocardium contains three layers and is connected to a ventricle by a wide neck
- Expands during systole during the acute phase and thus contributes to mechanical dysfunction of the left ventricle
  - Can contain thrombus
  - Site of junction can be source of ventricular arrhythmias
True Ventricular Aneurysm

- Persistent ST elevation after AMI (anterior) often indicates true aneurysm
  - Often accompanied by deep QS waves and T wave inversion
  - Persistent ST elevation may be associated with systolic dyskinesis, akinesis, or a large area of necrosis, even in the absence of anatomic aneurysm

- Considered chronic if persist for > 6 weeks
- ACE inhibitors can reduce true aneurysm development
- NSAIDs can increase development of aneurysms
- Left-ventricular aneurysmectomy
  - Heart failure
  - Ventricular arrhythmias
  - Thrombus on anticoagulation
Mechanical Rupture

- Cardiac tamponade from free wall rupture
- Formation of left ventricular diverticulum or pseudoaneurysm from free wall rupture
- Left to right shunt from septal rupture
- Acute mitral regurgitation from papillary muscle rupture.

- 10% of MIs
- 15% of in hospital deaths after MI
- Without surgical intervention, the mortality rate for rupture is > 80% at two weeks.
- Two high risk periods
  - 1\textsuperscript{st} 24 hours
  - Within 1\textsuperscript{st} week (3 to 5 days)
- Associated with delayed fibrinolytics and late presentation
A DILIGENT ASSESSMENT BY A NURSE MAY UNCOVER A FINDING THAT WILL MAKE A DIFFERENCE IN A LIFE.

Maintain your diligence!
BE THE BEST THAT YOU CAN BE EVERY DAY. YOUR PATIENTS ARE COUNTING ON IT!

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