Acute Coronary Syndrome (ACS): Disease Management

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Acute Coronary Syndrome (ACS)

• ST Elevation
  – STEMI

• No ST Elevation
  – NSTEMI
  – Unstable Angina (UA)

The role of Troponin.

ACS refers to any rupture of plaque or thrombotic event that leads to symptomatic ischemia or infarction.
Pathophysiology

- 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
- Vulnerable 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
Hospitalizations in the U.S. Due to ACS

**Acute Coronary Syndromes**

- **1.57 Million** Hospital Admissions - ACS
- **1.24 million** Admissions per year (UA/NSTEMI)
- **0.33 million** Admissions per year (STEMI)

*Primary and secondary diagnoses. †About 0.57 million NSTEMI and 0.67 million UA.

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Ischemic Discomfort
Acute Coronary Syndrome

- **Presentation**
- **Working Dx**
- **ECG**
- **Cardiac Biomarker**
- **Final Dx**

- **No ST Elevation**
  - Non-ST ACS
  - UA
  - Unstable Angina

- **ST Elevation**
  - NSTEMI
  - Myocardial Infarction
  - NQMI
  - Qw MI

Acute Myocardial Infarction

- Development of myocardial necrosis caused by a critical imbalance between the oxygen supply and demand of the myocardium
- 10 seconds of oxygen deprivation: Ischemia
- 1 minute of Ischemia: Myocardial dysfunction affected
- 20 minutes of oxygen deprivation: irreversible cell damage
  - STEMI
  - NSTEMI

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
**NSTEMI**

- Higher mortality and morbidity than STEMI
- 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
  - Treatment focus = antiplatelet therapy
- Differentiated from unstable angina by troponin levels

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**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 vasocostriction) of epicardial and/or microvascular vessels
- Progressive mechanical obstruction to coronary flow
- Coronary arterial inflammation
- Secondary UA
- Coronary artery dissection§

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ACS Symptoms

- Classic Symptoms
  - Stable angina
  - Unstable angina
  - MI

- Symptom Variations
  - Women
  - Elderly
  - Diabetics

Differential Diagnosis of Chest Pain

- Assessment of Pain
- Linking Patient History and Risk factors
- Cardiac Biomarkers
- ECG Findings
Assessment of Angina

- N = Normal
- O = Onset
- P = Precipitation / provoking / palliative factors
- Q = Quality or quantity
- R = Radiation and region
- S = Severity
- T = Time

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 not usually 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

<table>
<thead>
<tr>
<th>Stable</th>
<th>Unstable</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Occurs with physical exertion or emotional stress</td>
<td>• Occurs with minimal exertion or increased dose of nitroglycerin is required to achieve relief.</td>
</tr>
<tr>
<td>• Relieved by rest or sublingual nitroglycerin</td>
<td>• Prolonged rest angina is also considered unstable angina.</td>
</tr>
<tr>
<td>• Predictable pattern</td>
<td>• Angina that increases in severity or is very severe on first presentation</td>
</tr>
<tr>
<td>• Predictable = triggered by the same amount of physical or emotional stress and should be easily relieved by rest or sublingual nitroglycerin.</td>
<td>• Caused by unstable or ruptured plaque that causes abrupt closure of a coronary artery which may spontaneously reperfuse.</td>
</tr>
</tbody>
</table>

### Angina in Women

- Delay presenting with symptoms
- Attribute symptoms to other non-cardiac causes
- Presentation
  - epigastric discomfort
  - less specific complaints: dyspnea or fatigue
  - symptoms of discomfort from nose to navel should be evaluated for presence of CAD
- Less documented stenotic disease of major epicardial coronary arteries
- More likely to have unstable angina than MI
- Older women have higher incidence of complications
## Angina in the Elderly

- Generalized symptoms
  - weakness, dyspnea, and confusion.
- Symptoms often attributed to the aging process
- Cardiac and non cardiac co-morbidities complicate the diagnosis of ACS and increase the risk
- Don’t complain about chest pain
  - 37% of patients > 65
  - 42% of patients > 75 years
  - 75% of those > 85 years

## Angina in Diabetics

- Autonomic dysfunction can affect symptoms experienced with angina
- Less likely to experience pain.
- 25% of all patients presenting with ACS are diabetic
- Have severe multi-vessel disease
- Have higher rates of complications
- Have a greater proportion of ulcerated plaques resulting in intracoronary thrombi
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

Response to Symptoms

Patients with symptoms of ACS (chest discomfort with or without radiation to the arm[s], back, neck, jaw, or epigastria; shortness of breath; weakness; diaphoresis; nausea; lightheadedness) should be instructed to call 9-1-1 and should be transported to the hospital by ambulance rather than by friends or relatives

- Source: ACC/AHA NSTEMI Guidelines 2007
STAT ECG Indications

- 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
  - Source: ACC / AHA NSTEMI Guidelines 2007

If non diagnostic:
- Repeat q 15 to 30 minutes
- Or use ST segment monitoring
- Perform V7-V9

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

- Smoking
- Hypertension
- Dyslipidemia
- Diabetes
- Obesity
- Metabolic Syndrome
- Inactivity
- Alcohol

9 easily measured and potentially modifiable risk factors account for over 90% of the risk of an initial acute MI
Other Pertinent History

- CAD
- Cerebral vascular disease
- Peripheral vascular disease

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
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.

BNP may also be done to assess risk.

Cardiac Biomarkers

- Troponin I and T
  - Found only in cardiac muscle
  - Most sensitive indicator of myocardial damage
  - Approximately 30% of patients with NSTEMI and normal CKMB levels will test positive
  - Of equal sensitivity and specificity
  - Troponin I remains elevated for a long period, with a gradual return to normal
    - Beneficial indicator in patients presenting late after symptom onset
  - Capable of diagnosing small amounts of myocardial necrosis not measured by rises in CK-MB levels
  - Non CAD causes of troponin elevation (sepsis, pulmonary emboli and chronic kidney disease)
# 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.
Diagnostic Testing

- Non Invasive
  - ECG Evaluation
  - Stress Testing
  - CT / CTA
- Invasive: Cardiac Catheterization
  - IVUS

Stress Testing

- Exercise Stress Test with or without myocardial imaging
  - Nuclear Scanning
  - Echocardiogram
  - Future
- Patient conditions requiring myocardial imaging with stress testing due to lack of reliable ECG interpretation include:
  - Left bundle branch block
  - > 1 mm ST-segment depression at rest
  - Paced ventricular rhythm
  - Wolf-Parkinson-White syndrome
### Exercise Stress Testing

- Treadmills or bicycles
- Able to exercise on a treadmill for 6 to 12 minutes
- While exercising
  - Myocardial oxygen demand increases
  - Coronary arteries dilate in response to increased demand
- If CAD
  - Coronary arteries not able to adequately dilate to meet the needs of the increased myocardial oxygen demand
  - Abnormalities occur on 12-lead ECG or imaging studies
- Consideration with beta-blockers
  - Hold beta-blockers approximately 48 hours prior to testing
  - May not hold if determining effectiveness
- Exercise stress testing is less sensitive in women than in men

### Chemical Stress Testing

- Three pharmacological agents
  - Dobutamine, dipyridamole, and adenosine.
- Done in conjunction with myocardial imaging
  - Dobutamine only with echocardiography
- Dipyridamole and adenosine
  - Causes coronary microvascular dilatation similar to the coronary artery vasodilatation that occurs with exercise
  - Contraindications:
    - Severe lung disease or if wheezing
    - Currently on aminophylline or related products
  - Major side effect: Bronchospasm
  - Antidote: Aminophylline
- Adenosine
  - episodes of heart block due to its ability to slow or stop conduction through the atrioventricular (AV) node
Chemical Stress Testing

• Dobutamine
  – High-dose dobutamine increases contractility and heart rate
  – Increasing myocardial oxygen demand
  – More closely mimics exercise stress testing
  – Side effect: Tachyarrhythmias
  – Antidote: Beta blocker

Contraindications to Stress Testing

• Acute MI < 2 days old
• Acute myocarditis or pericarditis
• Acute pulmonary embolism
• Acute aortic dissection
• Symptomatic heart failure
• Severe aortic stenosis
• Symptomatic arrhythmias
• High-risk unstable angina
Stress Testing in Patients Presenting with Chest Pain

- Indicated when ECG and biomarkers are not diagnostic
- Should be done before discharge or within 72 hours as outpatient
  - Precautionary pharmacotherapy for low risk patients being done on outpatient basis

CT Angiography
“FAST CT”

- 64 slice and beyond
- Detailed 3D Image
- Fast
- Coronary artery calcium scoring
  - Shows calcified plaque
  - Predictor of non-calcified plaque
- Coronary artery anatomy
- Myocardial function
- Need to lower heart rate
- Radiation exposure
- Good negative predictor
Cardiac Catheterization

- Indications
  - Patients with disabling angina despite medical treatment
  - Patients with high-risk criteria for coronary heart disease (CHD) on noninvasive testing
  - Patients who have survived sudden cardiac death
  - Patients with angina and clinical signs of CHD
  - Patients with low ejection fraction and ischemia on noninvasive testing
  - Patients with inadequate information obtained from noninvasive testing

STEMI Management

- 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.
Options for Transport of Patients With STEMI and Initial Reperfusion Treatment

Onset of symptoms of STEMI
9-1-1 EMS Dispatch
EMS on-scene
- Encourage 12-lead ECGs.
- Consider prehospital fibrinolytic if capable and EMS-to-needle within 30 min.

GOALS
EMS Transport
- Door-to-Needle within 30 min.
- EMS transport EMS-to-balloon within 90 min.
- Patient self-transport Hospital door-to-balloon within 90 min.

EMS capable
Not PCI capable
Inter-Hospital Transfer
EMS Triage Plan
GOALS
PCI capable

Golden Hour = first 60 min.
Total ischemic time: within 120 min.

Reperfusion Therapy
Fibrinolytic Therapy
Primary PCI

Fibrinolytic therapy criteria for administration:
- Symptom onset within 12 hours of administration (ideally within 3 hours)
- ST-segment elevation of greater than 1 mm in two leads evaluating the same wall of the myocardium or the presence of a new left bundle branch block
- ECG and other findings consistent with a true isolated posterior wall MI.

Fibrinolytics

**Contraindications to the administration of fibrinolytics:**
- Prior intracranial hemorrhage
- Known structural cerebral vascular lesion
- Malignant intracranial neoplasm
- Significant closed head injury within last 3 months
- Ischemic stroke within last 3 months (unless within last 3 hours)
- Suspected aortic dissection
- Active bleeding or bleeding diathesis (excluding menses)
- Symptoms greater than 24 hours old
- ST-segment depression (unless indicative of a true posterior wall MI)

**Successful reperfusion with Fibrinolytics:**
- Relief of presenting symptoms
- Reduction of at least 50% of initial ST-segment segment elevation on repeat ECG
- Hemodynamic and electrical stability
- Reperfusion arrhythmias such as accelerated idioventricular rhythm
- Early peaking of the CKMB

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**STEMI Anticoagulation**

Fibrinolytics
- Minimum of 48 hours – up to 8 days
- Agents other than Unfractionated Heparin (UFH) if > 48 hours to reduce risk of Heparin Induced Thromocytopenia (HIT)
- Options for anticoagulation
  - UFH
  - Enoxaparin (low molecular weight heparin (LMWH))
  - Fondaparinux (indirect factor Xa inhibitor)

PCI
- UFH
- Enoxaparin
- Bivalrudin (direct thrombin inhibitor)
- Fondaparinux cannot be used as sole anticoagulant
  - Higher risk for catheter thrombosis
  - Need agent with anti IIa activity
Reasons for Delayed or Missed Reperfusion Therapy

- Missed performance of ECG due to atypical symptoms
- Unrecognized unequivocal ECG
- Delay in diagnosis of subtle ECG
- Failure to perform serial ECGs
- Delay in administration of therapy
- Abortion of treatment
  - Resolution of pain alone is not indication for aborting therapy
Medical Management of STEMI

- ASA
- Clopidogrel (with or without reperfusion)
- Oxygen
- NTG
- MS (Class I)
- D/C NSAIDS
- Beta-blockers (within 24 hours)
- ACE Inhibitors (within 24 hours with impaired EF, HTN, diabetes or chronic kidney disease)
- Anticoagulants (related to reperfusion strategy)
- Intravenous insulin may be indicated in first 24 to 48 hours after STEMI to tightly control blood sugars.

Reperfusion is primary management strategy.
Treatment of NSTEMI / UA: New Guidelines

- **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)
- **DC – NSAIDS**
- **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

Early invasive strategy versus early conservative strategy

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Treatment of NSTEMI / UA: New Guidelines

- **Antiplatelet Therapy**
  - Clopidogrel
  - Glycoprotein (GP) IIb/IIIa inhibitors
  - Upstream administration for invasive strategies
  - Also used in conservative strategies

- **Anticoagulation Options:**
  - Unfractionated heparin
  - LMWH (enoxaparin)
  - Direct thrombin inhibitor (bivalrudin)
  - Indirect factor Xa inhibitor (fondaparinux)
Early Invasive Option in UA / NSTEMI

• Not waiting for failed medical treatment
• Not waiting for + noninvasive test
• Angiography with intent of revascularization
• Better outcomes with GP IIb/IIIa inhibitors
• Excluded: very frail elderly, severe hepatic, pulmonary or renal failure, active or inoperable cancer

Overall reduction in mortality and increased quality of life.

• Initial conservative (selective invasive) is an alternative option
• Initial conservative strategy in low risk women

Early Invasive Indications

• Refractory angina or hemodynamic or electrical instability
  – Without serious co-morbidities or contraindications to such procedures
  – May be reasonable in patients with chronic renal insufficiency

• Initially stabilized with high risk for clinical events
  – Initial conservative is also an option
Algorithm for Patients with UA/NSTEMI Managed by an Initial Invasive Strategy

- **Diagnosis of UA/NSTEMI is Likely or Definite**
  - ASA (Class I, LOE: A)
  - Clopidogrel if ASA intolerant (Class I, LOE: A)

- **Select Management Strategy**
  - Proceed to Invasive Strategy
    - Proceed to Diagnostic Angiography

- **Invasive Strategy**
  - Init. ACT (Class I, LOE: A)
    - Acceptable options: enoxaparin or UFH (Class I, LOE: A), bivalirudin or fondaparinux (Class I, LOE: B)

- **Prior to Angiography**
  - Init at least one (Class I, LOE: A) or both (Class IIa, LOE: B) of the following:
    - Clopidogrel
    - IV GP IIb/IIIa inhibitor

Factors favoring admin of both clopidogrel and GP IIb/IIIa inhibitor include:
- Delay to Angiography
- High Risk Features
- Early recurrent ischemic discomfort

- Proceed to Diagnostic Angiography

Anderson JL, et al. J Am Coll Cardiol. 2007;50:e1-e157, Figure 7. ACT = anticoagulation therapy; LOE = level of evidence.

Algorithm for Patients with UA/NSTEMI Managed by an Initial Conservative Strategy

- **Diagnosis of UA/NSTEMI is Likely or Definite**
  - ASA (Class I, LOE: A)
  - Clopidogrel if ASA intolerant (Class I, LOE: A)

- **Select Management Strategy**
  - Proceed with Invasive Strategy
    - Conservative Strategy
  
- **Conservative Strategy**
  - Acceptable options: enoxaparin or UFH (Class I, LOE: A), bivalirudin or fondaparinux (Class I, LOE: B), but enoxaparin or fondaparinux are preferable (Class IIa, LOE: B)

- Init. clopidogrel (Class I, LOE: A)
  - Consider adding IV eptifibatide or tirofiban (Class IIb, LOE: B)

- **(Continued)**

Algorithm for Patients with UA/NSTEMI Managed by an Initial Conservative Strategy

(Continued)

Any subsequent events necessitating angiography?

Yes

No

Evaluate LVEF

EF greater than 40%

Low Risk

Cont ASA (Class I, LOE A)
Cont clopidogrel (Class I, LOE A) and ideally up to 1 yr (Class I, LOE B)
DC IV GP IIb/IIIa if started previously (Class I, LOE A)
* DC ACT (Class I, LOE A)

No

EF 40% or less

Not Low Risk

Low Risk

Stress Test

Proceed to Dx Angiography

Management after Diagnostic Angiography in Patients with UA/NSTEMI

Dx Angiography

Select Post Angiography Management Strategy

CABG

PCI

Medical therapy

CAD on angiography

Cont ASA (Class I, LOE A)
Cont clopidogrel if not given pre angiography (Class I, LOE A)
DC IV GP IIb/IIIa if not started pre angiography (Class I, LOE A)
DC ACT after PCI for uncomplicated cases (Class I, LOE A)

No significant obstructive CAD on angiography

Cont ASA (Class I, LOE A)
Cont clopidogrel if not given pre angiography (Class I, LOE A)
DC IV GP IIb/IIIa after at least 12 h if started pre angiography (Class I, LOE A)
IV UFH for at least 48 h if started after angiography (Class I, LOE A)

Antiplatelet and ACT at physician's discretion (Class I, LOE C)

2009
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

Early Conservative:
- Low Risk Score
- Patient / Physician Preference
### 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

### Short-Term Risk of Death/Nonfatal MI in Patients With UA/NSTEMI

<table>
<thead>
<tr>
<th>Feature</th>
<th>High Risk</th>
<th>Intermediate Risk</th>
<th>Low Risk</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>History</strong></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><strong>Character of pain</strong></td>
<td>Prolonged ongoing (&gt; 20 min) rest</td>
<td>Prolonged (&gt; 20 min) rest angina, now resolved, w/ moderate/high likelihood of CAD</td>
<td>✪ Angina frequency, severity or duration</td>
</tr>
<tr>
<td></td>
<td>pain</td>
<td>Rest angina (&gt; 20 min) or relieved with rest or sublingual NTG</td>
<td>Angina provoked at lower threshold</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Nocturnal angina</td>
<td>New onset angina with onset 2 wks to 2 mos prior to presentation</td>
</tr>
<tr>
<td></td>
<td></td>
<td>New-onset or progressive CCS class III/IV angina in past 2 wks w/o prolonged (&gt; 20 min) rest pain but with intermediate/high likelihood of CAD</td>
<td></td>
</tr>
</tbody>
</table>
Short-Term Risk of Death/Nonfatal MI in Patients With UA/NSTEMI, Continued

<table>
<thead>
<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-depression &lt; 1 mm in multiple lead groups (anterior, inferior, lateral)</td>
<td></td>
</tr>
<tr>
<td></td>
<td>- Sustained VT</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Cardiac markers</strong></td>
<td>↑ Cardiac TnT, Tnl, or CK-MB (e.g., TnT/Tnl &gt; 0.1 ng/mL)</td>
<td>Slightly ↑ cardiac TnT, Tnl, or CK-MB (e.g., TnT &gt; 0.01, but &lt; 0.1 ng/mL)</td>
<td>Normal</td>
</tr>
<tr>
<td></td>
<td>↑ Cardiac markers</td>
<td></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.

Noninvasive Test Results That Predict High Risk for Adverse Outcomes

<table>
<thead>
<tr>
<th>Stress Radionuclide Ventriculography</th>
<th>Stress Echocardiography</th>
<th>Stress Radionuclide Myocardial Perfusion Imaging</th>
</tr>
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<tbody>
<tr>
<td>Exercise EF ≤ 50 %</td>
<td>Rest EF ≤ 35%</td>
<td>Abnormal myocardial tracer distribution in &gt; 1 coronary artery region</td>
</tr>
<tr>
<td>Rest EF ≤ 35%</td>
<td>Wall-motion score &gt; 1</td>
<td>Abnormal myocardial distribution with ↑ lung intake</td>
</tr>
<tr>
<td>Fall in EF ≥ 10%</td>
<td></td>
<td>Cardiac enlargement</td>
</tr>
</tbody>
</table>

### Long-Term Antithrombotic Therapy at Hospital Discharge after UA/NSTEMI

**UA/NSTEMI Patient Groups at Discharge**

- **Medical Therapy without Stent**
  - **ASA 162 to 325 mg/d for at least 1 month, then 75 to 162 mg/d indefinitely (Class I, LOE: A)**
  - **& Clopidogrel 75 mg/d for at least 1 month and up to 1 year (Class I, LOE: A)**

- **Bare Metal Stent Group**
  - **ASA 162 to 325 mg/d for at least 1 month, then 75 to 162 mg/d indefinitely (Class I, LOE: A)**
  - **& Clopidogrel 75 mg/d for at least 1 month and up to 1 year (Class I, LOE: A)**

- **Drug Eluting Stent Group**
  - **ASA 162 to 325 mg/d for at least 3 to 6 months, then 75 to 162 mg/d indefinitely (Class I, LOE: A)**
  - **& Clopidogrel 75 mg/d for at least 1 year (Class I, LOE: B)**

**Indication for Anticoagulation?**

- **Yes**
  - Add: Warfarin (INR 2.0 to 2.5) (Class IIb, LOE: B)

- **No**
  - Continue with dual antiplatelet therapy as above

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### Beta Blockers Considerations

- **Oral Beta Blockers**
  - Within 24 hours

- **IV Beta Blockers**
  - Reasonable in patients who are hypertensive
  - May be harmful in patients with high risk for cardiogenic shock

**Contraindications**

- Signs of HF
- Low cardiac output state
- Increased risk for cardiogenic shock

**Relative contraindications**

- **PR > .24 seconds**
- **2nd or 3rd degree block**
- Active asthma
- Reactive airway disease

**Nondihydropyridine calcium channel blocker if beta blocker contraindicated and no significant LV dysfunction.**
Nitrate Contraindications

- Systolic BP < 90 mm Hg or ≤ 30 mm Hg below baseline
- Bradycardia < 50 BPM
- Tachycardia > 100 BPM (in absence of clinical HF)
- Right ventricular infarct
- Within 24 hours of sildenafil
- Within 48 hours of taldalafil

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
- No immediate release dihydropyridine calcium channel blockers without beta blockade on board
- NSAIDS (except for ASA), whether nonselective or COX-2-selective agents increase risk of mortality, reinfarction, hypertension, HF, and myocardial rupture
- Proton Pump Inhibitors should be prescribed to patients at risk for GI bleed
Stepped Care Approach To Pharmacologic Therapy for Musculoskeletal Symptoms with Known Cardiovascular Disease or Risk Factors for Ischemic Heart Disease

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

Select patients at low risk of thrombotic events
Prescribe lowest dose required to control symptoms
Add ASA 81 mg and PPI to patients at increased risk of thrombotic events *

* Addition of ASA may not be sufficient to reduce risk of thrombotic events


Long Term Management of ACS

Medications to improve prognosis
- Aspirin
- Clopidogrel
- *Beta-blockers
- *ACE inhibitors (in select patients)
  - ARBs (may be used with ACE-I in systolic dysfunction)
  - Aldactone (EF ≤ 40 with HF or diabetes)
- Lipid-lowering drugs (statins)
- * Beta blockers and ACE inhibitors impact long term ventricular remodeling

Medications to control ischemia
- Increased dose of beta-blockers
- Nitrates (all patients should be given sublingual nitroglycerin @ DC)
- Calcium channel blockers
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.

Secondary Prevention

- 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
### 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
  - 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

• Hemodynamic Alterations
• Ventricular Arrhythmias
• Atrial Arrhythmias
• Pericarditis
• Ventricular Aneurysms
• Mechanical Complications
  – Myocardial Rupture (free wall or VSD)
  – Papillary Muscle Rupture
• Long Term: Ventricular Remodeling
Ventricular Aneurysm

- Persistent ST elevation after AMI (anterior)
- Anatomic LV aneurysm
  - Myocardial thinning and bulging
- Use of echo in the reperfusion decision
- Risk of fibrinolytics with ventricular aneurysm
  - Embolization of thrombus

ST Elevation of Ventricular Aneurysm

- Most common in V1-V3
- Usually less than 3 mm elevation
- Relatively unchanged from previous ECGs
- Q waves are deep and well formed
  - QS pattern in V1-V3 or very minimal r
  - QR pattern common in inferior aneurysm / RBBB
Myocardial Rupture

- **Incidence**
  - 10% MI deaths
- **Definition**
  - Myocardial leakage – hemipericardium – tamponade
  - Perceived sudden; often slow tear
- **Associated Factors**
  - Late fibrinolytics
  - Delayed hospital admission
- **Septal involvement = VSD**
Myocardial Rupture

- Post-infarction regional pericarditis precedes rupture
- Confirmation of rupture

T Wave Patterns in Post-infarction Regional Pericarditis

- Persistently positive T waves 48 hours after an MI
- Premature reversal of T wave inversion to positive ST segment reelevation

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Acute Mitral Regurgitation

- Acute event causing mitral valve regurgitation
  - Papillary muscle rupture
  - Once free the attached valve leaflet will not close
  - Gaping hole left for blood to eject through

Pathophysiology

- Impairment or rupture of a papillary muscle
- Damaged to myocardial wall → damage to attachment of the papillary muscle to that ventricular wall
- Papillary muscle continues to contract with each cardiac cycle
- Attachment of papillary muscle to ventricular wall becomes weaker with each contraction
- With enough damage to the myocardial wall or papillary muscle the papillary muscle will actually disconnect from the ventricular wall
- Acute mitral regurgitation state
- Emergency measures are necessary to preserve the patient’s life
Acute Mitral Regurgitation

Acute decrease in cardiac output ➔ ↑ SVR

➔ blood flow to area of least resistance (non-functional MV)

➔ decrease in cardiac output ➔ ↑ SVR

➔ blood flow to area of least resistance (non-functional MV)

➔ decrease in cardiac output ➔ ETC.!!!!!
Diagnosis and Treatment

- Murmur
- STAT Echocardiogram
- Afterload reduction
- Emergent Surgery

*Note: Antibiotics for acute endocarditis*

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Final Quote:
Our grand business in life
is not to see what lies
dimly at a distance,
but to do what lies clearly at hand.

*Thomas Carlyle (1795-1881)*
Thanks for Attending
Cardiovascular Boot Camp

You may contact us at
www.cardionursing.com