ACS 4: HIGH RISK FEATURES AND COMPLICATIONS

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Risk Stratification

- Early in-hospital identification of patients at increased risk for recurrent ischemic events.
- Identification of patients after AMI who are at increased risk for arrhythmic or nonarrhythmic death.

- 30 day mortality rate in STEMI is reported to be 2.5 – 10%
- Mortality in NSTEMI is about 2%
Risk Factors for Adverse Outcomes in STEMI

Factors present before MI

- # of CHD risk factors
  - Risk of developing CHD increases with # of risk factors
  - Risk of in-hospital mortality decreases with increasing # of risk factors – WEIRD and unexplained!
- Chronic kidney disease = higher risk for MI and a worse outcome after MI
- Peripheral artery disease
  - Presence of intermittent claudication associated with worse outcomes

Heart Failure

- Killip Class - categorizes patients with AMI based on the presence or absence of signs of LV dysfunction
  - Class I: No evidence of heart failure
  - Class II: Mild to moderate HF (S3 gallop, rales < ½ way up posterior lung fields, JVD)
  - Class III: Overt pulmonary edema
  - Class IV: Cardiogenic shock
- The higher the Killip class on presentation, the greater the subsequent mortality.
- Higher Killip class following fibrinolysis or PCI associated with higher mortality.
- **Hypotension** (systolic pressure <100 mmHg)
  - Especially in anterior MI with reduced EF
  - Hypotension in inferior MI may be due to increased vagal activity and is usually transient

- **Tachycardia**
  - Sustained heart rates >90 beats/min associated with larger and more often anterior infarcts, LV dysfunction, and poor prognosis

- **ECG = worse prognosis with:**
  - Anterior compared to inferior infarcts
  - The presence of Q waves
  - A greater number of leads showing ST elevation
  - Lack of ST elevation resolution at 90 to 180 minutes after fibrinolysis

- **Recurrent Ischemia**
  - Higher 30 day reinfarction rate (especially if associated with ST changes or hemodynamic abnormalities)
  - Higher 30 day mortality rate (only if associated with hemodynamic abnormalities)

- **Atrial fib & ventricular arrhythmias**

- **Troponins**
  - Elevated troponin I or T at presentation is associated with higher 30 day and long term mortality
  - 30 day mortality increases with increasing troponin values (the higher the troponin the higher the mortality)
  - Reinfarction rate is higher in patients with troponin elevations in lowest quartile than in those with highest troponins (higher levels associated with completed infarcts?)
Risk Stratification After STEMI

- All patients with ST-elevation myocardial infarction should undergo risk assessment within the first four to six hours of hospitalization.
- Even low-risk patients should undergo primary reperfusion (PCI preferred) in a timely manner.

TIMI Risk Score for STEMI
(sum of 8 independent predictors of mortality)

<table>
<thead>
<tr>
<th>Predictor of Mortality</th>
<th>Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age ≥75 years</td>
<td>3</td>
</tr>
<tr>
<td>Age 65 to 74 years</td>
<td>2</td>
</tr>
<tr>
<td>History of diabetes, hypertension, or angina</td>
<td>1</td>
</tr>
<tr>
<td>Systolic blood pressure &lt;100 mmHg</td>
<td>3</td>
</tr>
<tr>
<td>Heart rate &gt;100/min</td>
<td>2</td>
</tr>
<tr>
<td>Killip class II to IV</td>
<td>2</td>
</tr>
<tr>
<td>Weight &lt;76 kg</td>
<td>1</td>
</tr>
<tr>
<td>Anterior ST elevation or LBBB</td>
<td>1</td>
</tr>
<tr>
<td>Time to reperfusion therapy &gt;4 hours</td>
<td>1</td>
</tr>
</tbody>
</table>
**TIMI Risk Score** (same for PCI & fibrinolysis)

<table>
<thead>
<tr>
<th>Total Points</th>
<th>30 Day Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 Points</td>
<td>0.8%</td>
</tr>
<tr>
<td>1 Point</td>
<td>1.6%</td>
</tr>
<tr>
<td>2 Points</td>
<td>2.2%</td>
</tr>
<tr>
<td>3 Points</td>
<td>4.4%</td>
</tr>
<tr>
<td>4 Points</td>
<td>7.3%</td>
</tr>
<tr>
<td>5 Points</td>
<td>12%</td>
</tr>
<tr>
<td>6 Points</td>
<td>16%</td>
</tr>
<tr>
<td>7 Points</td>
<td>23%</td>
</tr>
<tr>
<td>8 Points</td>
<td>27%</td>
</tr>
<tr>
<td>9-14 Points</td>
<td>36%</td>
</tr>
</tbody>
</table>


**TIMI Risk Index**

- Heart rate in beats/min x ([age/10]squared) / systolic blood pressure
  - Based on data obtained at presentation

<table>
<thead>
<tr>
<th>Risk index</th>
<th>Risk group</th>
<th>24 hour</th>
<th>Risk of death</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>In-hospital</td>
<td>30 day</td>
</tr>
<tr>
<td>≤ 12.5</td>
<td>1</td>
<td>0.2</td>
<td>0.6</td>
</tr>
<tr>
<td>&gt; 12.5 to 17.5</td>
<td>2</td>
<td>0.4</td>
<td>1.5</td>
</tr>
<tr>
<td>&gt; 17.5 to 22.5</td>
<td>3</td>
<td>1.0</td>
<td>3.1</td>
</tr>
<tr>
<td>&gt; 22.5 to 30</td>
<td>4</td>
<td>2.4</td>
<td>6.5</td>
</tr>
<tr>
<td>&gt; 30</td>
<td>5</td>
<td>6.9</td>
<td>15.8</td>
</tr>
</tbody>
</table>

GRACE Risk Model (Global Registry of Acute Coronary Events)

- Estimates the risk of in-hospital and six-month mortality among all patients with ACS
- In-hospital model uses 8 risk factors and assigns points for each one*
  - Age
  - Killip class
  - Systolic blood pressure
  - Presence of ST segment deviation
  - Cardiac arrest during presentation
  - Serum creatinine concentration
  - Presence of elevated serum cardiac biomarkers
  - Heart rate


GRACE Risk Model (cont.)

- The six-month model uses the following
  - Age
  - Prior history of heart failure
  - Prior history of myocardial infarction
  - Resting heart rate
  - Systolic blood pressure
  - ST-segment depression
  - Initial serum creatinine concentration
  - Elevated serum cardiac biomarkers
  - Performance of in-hospital PCI
GRACE Prediction in STEMI

<table>
<thead>
<tr>
<th>In-hospital Mortality</th>
<th>GRACE Risk Score</th>
<th>Probability of Death In-hospital (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>49-125</td>
<td>&lt;2</td>
</tr>
<tr>
<td>Intermediate</td>
<td>126-154</td>
<td>2-5</td>
</tr>
<tr>
<td>High</td>
<td>155-319</td>
<td>&gt;5</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>6 Month Post-discharge Mortality</th>
<th>GRACE Risk Score</th>
<th>Probability of Death In-hospital (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>27-99</td>
<td>&lt;4.4</td>
</tr>
<tr>
<td>Intermediate</td>
<td>100-127</td>
<td>4.5 - 11</td>
</tr>
<tr>
<td>High</td>
<td>128-263</td>
<td>&gt;11</td>
</tr>
</tbody>
</table>


Late Risk Stratification After STEMI

- Left ventricular ejection fraction should be assessed before discharge (usually by cardiac echo)
  - Patients with LV systolic dysfunction have increased mortality at six months and one year – especially if EF ≤30%
  - Patients with LVEF ≤35 percent are at increased risk for SCD after MI and should be considered candidates for an ICD if EF still low 40 days after discharge
- Stress testing for ischemia should be performed before discharge to assess the presence and extent of inducible ischemia in patients with STEMI who have not had coronary angiography
Risk Factors for Adverse Outcomes in NSTEMI

- In addition to same things mentioned for STEMI:
  - Older age
    - Patients over the age of 75 with NSTEMI had more diffuse and severe coronary disease and more adverse outcomes both in hospital and within the first six weeks after discharge
  - Sex
    - Most studies show higher in-hospital & 30 day mortality in women, especially those <55 years
    - Long term mortality appears to be equal between men and women
  - Prior MI
  - Prior stroke
  - COPD
  - Obstructive sleep apnea
  - Aspirin use (signifies more extensive disease)

- ECG
  - ST depression has worse prognosis than T wave inversion
  - LBBB associated with increased rate of reinfarction and long term mortality
  - Anterior NSTEMI worse than inferior

- Silent ischemia = increased risk of death or reinfarction
- BNP – elevated levels associated with increased risk of death, new or recurrent MI, and new or worsening heart failure
- Diabetics have worse long term outcomes after UA/NSTEMI than non-diabetics.
- MI without chest pain
  - About 33% of patients have atypical presentations without pain or present with cardiac arrest
  - In one study, patients who presented without pain had higher 30 day and 1 year mortality rate than those who presented with chest pain
    - Less likely to receive evidence-based care

**Risk Stratification After NSTEMI**

- Very High Risk Patients (these patients should go directly for urgent coronary angiography)
  - Cardiogenic shock
  - Overt heart failure (HF) or severe left ventricular dysfunction
  - Recurrent or persistent rest angina despite intensive medical therapy
  - Hemodynamic instability due to mechanical complications (acute mitral regurgitation, ventricular septal defect)
  - Unstable ventricular arrhythmias
TIMI Risk Score in NSTEMI
(most widely used and validated predictive instrument in patients with NSTEMI)

- One point for each of the following criteria
  - Age ≥65 years
  - Presence of at least three risk factors for coronary heart disease (CHD)
  - Prior coronary stenosis of ≥50 percent
  - Presence of ST segment depression on admission ECG
  - At least two anginal episodes in prior 24 hours
  - Elevated serum cardiac biomarkers
  - Use of aspirin in prior seven days


TIMI Risk Score (cont.)

- A higher TIMI risk score correlated significantly with increased numbers of events (all-cause mortality, new or recurrent MI, or severe recurrent ischemia requiring revascularization) at 14 days

<table>
<thead>
<tr>
<th>TIMI Score</th>
<th>Percent Mortality</th>
<th>TIMI Score</th>
<th>Percent Mortality</th>
</tr>
</thead>
<tbody>
<tr>
<td>0-1</td>
<td>4.7</td>
<td>4</td>
<td>19.9</td>
</tr>
<tr>
<td>2</td>
<td>8.3</td>
<td>5</td>
<td>26.2</td>
</tr>
<tr>
<td>3</td>
<td>13.2</td>
<td>6/7</td>
<td>40.9</td>
</tr>
</tbody>
</table>

- Only patients with score ≥3 benefited from an early invasive strategy
  - Degree of troponin elevation and magnitude of ST segment deviation were independent predictors of an adverse outcome and of benefit from an early invasive strategy
### GRACE Prediction in NSTEMI

#### In-hospital Mortality

<table>
<thead>
<tr>
<th>Risk Category (tertiles)</th>
<th>GRACE Risk Score</th>
<th>Probability of Death In-hospital (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>1-108</td>
<td>&lt;1</td>
</tr>
<tr>
<td>Intermediate</td>
<td>109-140</td>
<td>1-3</td>
</tr>
<tr>
<td>High</td>
<td>141-372</td>
<td>&gt;3</td>
</tr>
</tbody>
</table>

#### 6 Month Post-discharge Mortality

<table>
<thead>
<tr>
<th>Risk Category (tertiles)</th>
<th>GRACE Risk Score</th>
<th>Probability of Death in 6 months (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low</td>
<td>1-88</td>
<td>&lt;3</td>
</tr>
<tr>
<td>Intermediate</td>
<td>89-118</td>
<td>3-8</td>
</tr>
<tr>
<td>High</td>
<td>119-263</td>
<td>&gt;8</td>
</tr>
</tbody>
</table>


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### COMPLICATIONS IN ACUTE CORONARY SYNDROMES
Arrhythmias

❖ Sinus Bradycardia
- Occurs in 15-25% of AMI – usually inferior wall or RV
- Usually transient and resolves within 24 hours
- Caused by increased vagal tone, SA node ischemia, drugs (beta blockers), reperfusion after fibrinolysis
- Treatment: atropine 0.5 mg IV, temporary pacing

❖ Sinus Tachycardia
- Occurs in 30-40% of acute MI
- Persistent tachycardia more common with larger MI and anterior MI
- Associated with higher morbidity and mortality
- Treat the cause!

❖ Atrial fibrillation
- Most common atrial tachyarrhythmia following AMI – incidence 5-18% during hospitalization
- Usually associated with comorbidities: heart failure, kidney disease, hypertension, diabetes, pulmonary disease
- Most commonly due to atrial stretch secondary to HF; can be due to disrupted blood supply to SA node or atrial ischemia
- Rate control with beta blockers is first priority
- Amiodarone for recurrent AF or AF not responding to cardioversion
Heart Block in MI

- **Inferior wall MI**
  - 1\(^{st}\) degree and Wenckebach occur in the AV node and usually due to RCA occlusion
    - Usually resolves within 5-7 days and requires no treatment
  - 3\(^{rd}\) degree block usually occurs in the AV node, has a narrow QRS, occurs gradually (1\(^{st}\) → 2\(^{nd}\) → 3\(^{rd}\))
    - Usually results in asymptomatic bradycardia with junctional escape rhythm at 40 to 60 beats/min
    - Usually resolves within 5-7 days
  - 3\(^{rd}\) degree block more common with inferior than anterior MI

- **Anterior wall MI**
  - More serious blocks that occur below the AV node with wide QRS
  - Second degree type II and 3\(^{rd}\) degree blocks more common
    - Likely to occur suddenly and result in symptoms
    - Often preceded by bifascicular block
    - Slow, wide, unstable ventricular escape rhythm
    - High mortality rate – 80%
Ventricular Arrhythmias

- **Pre-fibrinolytic Era**
  - PVCs – 10 to 93%
  - VT – 3 to 39%
  - VF – 4 to 20%

- **Fibrinolytic Era (STEMI)**
  - Overall incidence of sustained VT or VF 10.2%
    - VT = 3.5%
    - VF = 4.1%
    - Both VT and VF = 2.7%
    - 80 to 85% occurred in the first 48 hours

- **PCI (STEMI)**
  - VT or VF = 5.7%

- **NSTEMI**
  - Overall incidence of sustained VT or VF = 2.1%

Ventricular arrhythmias in STEMI (all locations)

- **AIVR (rate 50-100)**
  - Occurs in up to 50% of acute MI
  - Not a sensitive or specific marker of reperfusion
  - No treatment needed

- **Nonsustained VT (< 30 seconds)**
  - No treatment unless frequent and symptomatic: beta blockers, amiodarone, procainamide
- Sustained monomorphic VT (> 30 seconds) with hemodynamic symptoms
  - Usually transient and due to ischemia in first 48 hours of AMI
  - Associated with higher in-hospital mortality
  - In any other setting is considered a marker of permanent arrhythmic substrate and an increased long-term risk of SCD
  - Cardioversion, amiodarone, procainamide, lidocaine

- Polymorphic VT
  - Uncommon in AMI but associated with ischemia or reperfusion when it occurs (0.3% incidence)
  - Defibrillate if sustained, beta blockers, early reperfusion

- VF
  - Usually occurs in 48-72 hours after AMI
  - Thought to be due to ischemia and lack of reperfusion of infarct artery
  - Primary VF refers to VF that occurs early (usually <48 hours post myocardial infarction) and is not associated with recurrent ischemia or heart failure
    - The presence of ST elevation is the most powerful predictor of primary VF
    - Other predictors: early repolarization, hypokalemia, hypotension, higher troponins
    - Associated with higher in-hospital mortality
  - Defibrillate, amiodarone, reperfusion
Cardiogenic shock (pump failure)

- **Persistent hypotension** (systolic blood pressure <80 to 90 mmHg or mean arterial pressure 30 mmHg lower than baseline) with **severe reduction in the cardiac index** (<1.8 L/min/m² without support or <2.2 L/min/m² with support) with **adequate or increased LV filling pressure** (PWP ≥15).
  - 15% have CS on presentation
  - 85% develop CS during hospitalization
- **Mortality rate used to be 80-90% prior to reperfusion era, now around 42-48%**

Cardiogenic Shock (cont.)

- **Causes**
  - Extensive LV infarction (usually anterior) with LV failure
  - Mechanical complications
    - Papillary muscle rupture
    - Ventricular septal rupture
    - Free-wall rupture with tamponade
    - RV infarction
  - The larger the infarct the more pump failure occurs.
Cardiogenic Shock (cont.)

- Predictors of Mortality
  - More common in STEMI than NSTEMI but mortality the same
  - Mortality higher in patients with LV failure than in those with mechanical complications
  - Culprit lesion location
    - 78.6% mortality in left main, 69.7% in saphenous vein graft, 42.4% in circumflex, 42.3% in LAD, and 37.4% in RCA
  - Higher with EF <28% and with moderate to severe mitral regurgitation
  - Higher with longer time from symptom onset to reperfusion

Management of Cardiogenic Shock
(ACC/AHA Guidelines)

- Emergency revascularization with either PCI or CABG for cardiogenic shock due to pump failure after STEMI irrespective of the time delay from MI onset (Class Ib).
- Immediate transfer to a PCI-capable facility with on-site cardiac surgical backup is indicated for patients with STEMI and CS.
- Fibrinolytic therapy for patients without contraindications within 24 hours of MI if revascularization is not feasible.
Management of CS (cont.)

- Usual medical treatment of STEMI except for no beta blockers or other negative inotropes
- Inotropic and vasopressor support
  - Norepinephrine better than dopamine (higher 28 day mortality and more arrhythmias with dopamine)
  - Dobutamine or milrinone not recommended if hypotension but OK if BP can tolerate
- IABP (class IIa)
- Volume (especially with RVMI) or diuretics to keep PWP optimal (usually around 18 mmHg)

Right Ventricular Infarction

- 40% incidence with inferior MI
  - Most often proximal RCA occlusion
  - Higher mortality when RV infarcted
- Pathophysiology in RV MI
  - Decreased right ventricular compliance → reduced RV filling → decreased RV stroke volume → decreased LV filling → decreased LV stroke volume → peripheral hypoperfusion: hypotension, tachycardia.
RV Infarction (cont.)

- Diagnosis: clinical triad of **hypotension**, **elevated jugular venous pressure** (↑ CVP), **clear lung fields** (↓ PWP)
  - Get right sided chest leads (V4R-V6R) with all inferior wall MIs.
    - 1-mm ST elevation in lead V1 and in right precordial lead V4R is the most sensitive ECG marker
- Treatment:
  - Fluids to increase LV filling
  - Avoid preload reduction (NTG, diuretics)
  - Inotropes (dobutamine, dopamine)
  - Maintain atrial kick (cardioversion of AF may be needed)
  - AV sequential pacing if bradycardia

Mechanical Complications

- Mitral Regurgitation
- Ventricular Septal Rupture
- Ventricular Free Wall Rupture
- Cardiac Tamponade
- Ventricular Aneurysm
**Myocardial Rupture**

- **Free Wall Rupture**
- **Papillary Muscle Rupture**
- **Septal Rupture**

- Life-threatening complication that causes acute mitral regurgitation
- More common in inferior MI
  - Posterior papillary muscle more common due to single blood supply from posterior descending artery
- Occurs 2-7 days after MI
- Presents with hypotension, acute dyspnea, heart failure → pulmonary edema, new systolic murmur
- Diagnosis by cardiac echo (TTE or TEE)
- Management:
  - Afterload reduction: nitroprusside, IABP
  - Diuretics
  - Emergent surgery for mitral valve repair (if no papillary muscle necrosis) or replacement

**Papillary Muscle Rupture**

- Life-threatening complication that causes acute mitral regurgitation
- More common in inferior MI
  - Posterior papillary muscle more common due to single blood supply from posterior descending artery
- Occurs 2-7 days after MI
- Presents with hypotension, acute dyspnea, heart failure → pulmonary edema, new systolic murmur
- Diagnosis by cardiac echo (TTE or TEE)
- Management:
  - Afterload reduction: nitroprusside, IABP
  - Diuretics
  - Emergent surgery for mitral valve repair (if no papillary muscle necrosis) or replacement
Ventricular Septal Rupture

- Occurs equally in anterior and inferior MI
  - Anterior MI – rupture usually in apical septum
  - Inferior MI – usually at the base of the heart.
  - Usually occurs within 3-5 days after MI (sometimes in first 24 hours)
- Risk factors: “wrap around” LAD (ST elevation in anterior and inferior leads), large infarcts, RV infarction
- Presents with sudden onset of hypotension, biventricular failure (mostly right-sided due to left-to-right shunt), and a new harsh holosystolic murmur.

- Diagnosed by doppler echo or right heart cath showing left-to-right shunt through septum
- Treatment: afterload reduction (nipride, IABP), diuretics, inotropes if cardiogenic shock, and surgical repair.
  - Surgery is urgent if shock present but can be delayed for weeks until infarct heals if patient stable enough.
LV Free Wall Rupture

- Present in up to 26% of patients who died with acute MI but occurs in < 1% of all MIs
- Occurs within 5 days in half of cases & within 2 weeks in 90% of cases
- Risk factors for rupture:
  - Fibrinolytic therapy (higher incidence than PCI)
  - No history of angina or previous MI (less collateral circulation)
  - ST elevation or Q waves on initial ECG
  - Large infarcts, higher biomarkers
  - Anterior MI
  - Age > 70
  - Female

- Complete rupture is tear through entire wall and is often fatal due to tamponade
  - Presents with acute right heart failure → shock → PEA → death
- Incomplete/subacute rupture: organized thrombus and the pericardium seal the ventricular perforation
  - Persistent or recurrent chest pain (pericardial); nausea, agitation; sudden, transient hypotension; and/or ECG signs of pericarditis
- Diagnose with echocardiogram → pericardiocentesis if fluid present → emergency surgery if fluid is blood
- Management: fluids, inotropes, vasopressors, IABP, surgical repair.
Ventricular Aneurysm

- Thin, scarred or fibrotic area in LV wall with no muscle or necrotic muscle resulting from healed transmural MI
- Occurs in 8-15% of Q-wave MI
  - 70-85% occur in anterior or apical wall – from occlusion of LAD with no collaterals
  - 10-15% in inferior wall from RCA occlusion
  - Filled with clot in about 50% of cases
- Diagnosis
  - Often prolonged ST elevation following anterior wall MI
  - Cardiac enlargement and dyskinetic area on echo
  - 3rd or 4th heart sound, systolic murmur of mitral regurg
- Complications with LV aneurysm
  - Heart failure - bulging of aneurysm during systole steals part of stroke volume so ↓ CO and ↑ volume load
  - Ventricular arrhythmias – ischemia, stretch, and heterogeneous tissue at border zone of aneurysm are set-up for ventricular arrhythmias → SCD
  - Thromboembolism – systemic embolization and stroke due to clots in aneurysm
  - Ventricular rupture
- Therapy: afterload reduction (usually with an ACEI), anti-ischemic medications for angina, anticoagulation, surgical aneurysmectomy (if intractable ventricular arrhythmias and/or heart failure unresponsive to medical and catheter-based therapy)
**Thromboembolism**
- Mural thrombi at site of infarction (especially large anterior MI)
- In atria during atrial fib
- Treated with anticoagulation

**Cardiac Tamponade**
- Can occur due to rupture at site of infarction or rupture during PA catheter or pacer insertion
- Presents with hypotension, JVD, muffled heart sounds
- Treatment
  - Pericardiocentesis – needle or pigtail drain
  - Surgery if blood in pericardium
  - Emergent return to OR to control bleeding in post surgical patient