Myocardial Infarction

Acute Coronary Syndrome (ACS) is a collection of syndromes associated with acute myocardial ischemia or infarction usually resulting from abrupt reduction in coronary blood flow (Anderson et al, 2013). ACS is typically caused by coronary artery obstruction resulting in a sudden imbalance of myocardial oxygen consumption and demand.

Classification of ACS

ACS is classified based on the presence or absence of ST segment elevation. There are three major classifications of ACS:

- **Non-ST Segment Elevation Acute Coronary Syndrome (NSTE-ACS): Unstable Angina (UA)**
  Clinical symptoms suggestive of ACS with the absence of persistent ST elevation and no elevation in cardiac biomarkers (troponin) [which are elevated with myocardial tissue damage]; with or without electrocardiogram (ECG) changes indicative of ischemia (Aroesty, Simons, & Breall, 2017). Diagnosis may be made by clinical history alone.
- **Non-ST Segment Elevation Acute Coronary Syndrome (NSTE-ACS): Non-ST Segment Elevation Myocardial Infarction (NSTEMI)**
  Clinical symptoms suggestive of ACS with elevated cardiac biomarkers (troponin); with or without ECG changes indicative of cardiac ischemia (Anderson et al, 2013).
  Note: ECG changes suggestive of cardiac ischemia include ST depression, transient ST elevation or prominent T wave inversions.
- **ST-Segment Elevation Myocardial Infarction (STEMI):**
  ACS symptoms with elevated cardiac biomarkers (troponin); ECG shows persistent ST elevation or new left bundle branch block (LBBB) (O'Gara et al, 2013). These patients should be considered for immediate reperfusion therapy (fibrinolysis or percutaneous coronary intervention [PCI]) (Anderson et al, 2013).

Guidelines for the Identification of Patients with ACS in the Emergency Room (Anderson et al, 2013)

Clinical History:

Patients with the following signs and symptoms require immediate assessment by the triage nurse for initiation of the ACS protocol and a STAT ECG:

- Chest pain or severe epigastric pain, nontraumatic in origin, with components typical of myocardial ischemia or myocardial infarction (MI)
  - Central/substernal compression or crushing chest pain
  - Pressure, tightness, heaviness, cramping, burning, aching sensation
  - Unexplained indigestions, belching, epigastric pain
  - Radiating pain in neck, jaw, shoulders, back, or one or both arms
- Severe dyspnea

- Hypotension
- Syncope

Medical History:
Obtaining a medical history must not delay entry into the ACS protocol. The triage nurse should take a brief, targeted, initial history with an assessment of current or past history of:

- CABG, PCI, CAD, angina with exertion, or MI
- NTG use to relieve chest discomfort
- Risk factors, including smoking, hyperlipidemia, hypertension, diabetes mellitus, family history, and cocaine or methamphetamine use
- Regular and recent medication use

Pathogenesis of ACS (Anderson et al, 2013)

- Thrombus or thromboembolism, usually arising on disrupted or eroded plaque:
    - Occlusive thrombus, usually with collateral vessels (UA/NSTEMI)
    - Subtotal occlusive thrombus on pre-existing plaque
    - Distal microvascular thromboembolism from plaque-associated thrombus
- 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

Clinical Presentations of ACS (Anderson et al, 2013; Aroesty et al, 2017)

- Rest angina (angina commencing when the patient is at rest), usually lasting more than 20 minutes in duration.
- New onset (less than 2 months) severe angina that significantly limits physical activity.
- Increasing angina (increasing in intensity, duration, and/or frequency), or occurs with less exertion than previous angina.

Carefully assess women, patients with diabetes mellitus, older patients, those with unexplained dyspnea, history of heart failure or stroke, and patients who complain of chest discomfort but now have a permanent pacemaker that may conceal 12-lead ECG changes.

Diagnosis of Acute Myocardial Infarction

Any one of the following criteria are diagnostic for MI.

- Detection of a rise and/or fall of cardiac biomarker values (preferably cardiac troponin I and T [cTn]) and at least one of the following:
    - Symptoms suggestive of myocardial ischemia
    - Development of pathologic Q waves on ECG
    - New significant ST-segment-T wave (ST-T) changes or new LBBB
    - Identification of intracoronary thrombus by angiography or autopsy

  - Imaging evidence of new loss of viable myocardium or a new regional wall motion abnormality
- Cardiac death with symptoms suggestive of myocardial ischemia and new ischemia- related ECG changes or new LBBB, but death occurred before cardiac biomarkers were obtained, or before cardiac biomarker values would be increased.
- PCI-related MI: defined by elevation of cardiac biomarker values in patients with normal baseline values or a rise of values > 20% if the baseline values are elevated but stable or falling. In addition, one of the following:
  - Symptoms suggestive of myocardial ischemia
  - New ECG changes suggestive of ischemia or new LBBB
  - Angiographic loss of patency of a major coronary artery or a side branch, or persistent slow- or no-flow or embolization
  - Imaging that demonstrates new loss of viable myocardium or new regional wall motion abnormality
- Stent thrombosis associated with MI when detected by coronary angiography or autopsy in the setting of myocardial ischemia and with a rise and/or fall of cardiac biomarkers
- Coronary artery bypass graft surgery (CABG)-associated MI: elevation of cardiac biomarker values in patients with normal baseline cTn values. In addition, one of the following:
  - New pathologic Q waves or new LBBB
  - Angiographic documented new graft or native coronary artery occlusion
  - Imaging evidence of new loss of viable myocardium or new regional wall motion abnormality

Goals of Therapy (Aroesty et al, 2017) and Management Strategies

RECOMMENDATIONS FOR ALL MYOCARDIAL INFARCTIONS (REGARDLESS OF CLASSIFICATION)	
Goals	**Management**
Early identification	• Electrocardiogram (ECG) should be performed within 10 minutes upon arrival to emergency department if not obtained by Emergency Medical System (EMS) prearrival. • If initial ECG not diagnostic and patient remains symptomatic, repeat ECG every 15-30 minutes to detect ischemic changes.
Acute triage	• Assess responsiveness, airway, breathing, and circulation. • Look for evidence of systemic hypoperfusion (hypotension; tachycardia; impaired cognition; cool, clammy, pale skin); cardiogenic shock requires aggressive management. • Left heart failure with hypoxia (dyspnea, hypoxia, pulmonary edema, and/or impending respiratory compromise) requires aggressive oxygenation, airway stabilization, diuretic therapy and afterload reduction.

	• Treat ventricular arrhythmias immediately due to effect on cardiac output and exacerbation of myocardial ischemia.
Initial therapy	• Continuous cardiac monitoring. • Administer oxygen to patients with arterial saturation <90%, patients in respiratory distress including those with heart failure, or those with other high-risk factors for hypoxia. *NOTE: Supplemental oxygen shows no benefit to patients with oxygen saturation ≥ 90%.* • Establish intravenous (IV) access. • Obtain serial cardiac troponin I or T levels at presentation and 3-6 hours after symptom onset.
Relief of ischemic pain	• Administer sublingual NTG every 5 minutes up to 3 times for continuing ischemic pain; administer IV NTG for persistent ischemia, heart failure, or hypertension. Use caution if risk of hypotension, suspicion/confirmed right ventricular failure, or severe aortic stenosis. Contraindicated if phosphodiesterase inhibitor (i.e. Viagra) taken within the previous 24 hours. • IV morphine should be avoided unless patient has an unacceptable level of pain. Initial dose is 2-4 mg, with increments of 2-8 mg at 5- to 15-minute intervals. • Discontinue nonsteroidal anti-inflammatory drugs (NSAIDs), except aspirin, because of increased risk of adverse cardiac events.
Stabilize hemodynamics/prevent and manage arrhythmias	• Atrial fibrillation and flutter can cause symptomatic hypoperfusion; ventricular tachycardia and fibrillation are life-threatening. • Treat with prophylactic IV β-blocker and maintain serum potassium between 3.5 and < 4.5 meq/L and serum magnesium above 2.0 meq/L. • Avoid prophylactic lidocaine. • Treat symptomatic bradycardia and heart block with atropine or temporary pacing.
Estimation of risk	High risk patients require aggressive management. This includes those of advanced age, or those with low blood pressure, tachycardia, heart failure, and an anterior MI. (See TIMI score later).
β-Blocker therapy	• To prevent recurrent ischemia and life-threatening ventricular arrhythmias. • Start IV β-blocker (metoprolol or atenolol) in all patients without contraindications within 24 hours; defer in patients that are hemodynamically unstable. • Contraindications are heart failure, low output state, risk for cardiogenic shock, bradycardia, PR interval > 0.24 seconds,

	second- or third- degree heart block without permanent pacemaker, reactive airway disease/active bronchospasm.

Dual antiplatelet therapy (O'Gara et al., 2013)	• Aspirin: loading dose 162-325 mg uncoated aspirin; maintenance dose 81-325 mg/day (81 mg/day is preferred dose and is the **only** dose option when used concomitantly with ticagelor.)
	• $P2Y_{12}$ inhibitors for 12 months, regardless if treated with primary-PCI or ischemia-guided strategy. Loading and maintenance doses are the same for both indications, however prasugrel is an option only in primary PCI, not in ischemia-guided strategy.
	  • Clopidogrel: Loading dose 300-600 mg; maintenance dose 75 mg/day
	  • Ticagelor: Loading dose 180 mg; maintenance 90 mg every 12 hours (must only be given with aspirin 81 mg/day)
	• Prasugrel (primary PCI only): Loading dose 60 mg; maintenance 10 mg/day (contraindicated with history of stroke or TIA, age ≥ 75 years, and weight < 60 kg.)

Cholesterol therapy	• High-intensity statin therapy should be initiated as early as possible; obtain fasting lipid panel within 24 hours. • Atorvastatin 80 mg daily or rosuvastatin 20 or 40 mg daily
Long-term management	• Antiplatelet therapy to reduce the risk of recurrent coronary artery thrombosis or, with PCI, coronary artery stent thrombosis • Statins • Oral anticoagulation in the presence of left ventricular thrombus or chronic atrial fibrillation to prevent embolization • Possible use of angiotensin converting enzyme (ACE) inhibitor in patients at increased risk • β-blockers, if no contraindications

RECOMMENDATIONS BASED ON CLASSIFICATION		
Goals	**Unstable Angina/NSTEMI**	**STEMI**
Invasive interventions (O'Gara et al, 2013)	Urgent/immediate diagnostic angiography with intent to re-vascularize (within 2 hours) in NSTE-ACS patients with: • Hemodynamic instability or cardiogenic shock • Severe left ventricular dysfunction or heart failure • Recurrent or persistent rest angina despite intensive medical therapy	Reperfusion therapy should be administered to all eligible patients with STEMI with symptom onset within the prior 12 hours: • PCI-capable hospitals – door-to-balloon time within 90 minutes upon arrival. • Non-PCI-capable hospitals – transfer to PCI hospital for door-to-balloon time of 120 minutes. If arrive

	• New or worsening mitral regurgitation or new ventricular septal defect • Sustained ventricular arrhythmias Within 24 hours of admission for initially stabilized high-risk patients. Not recommended in those with extensive co-morbidities, for whom the risks are likely to outweigh the benefits of revascularization OR in those with acute chest pain and low likelihood of ACS who are troponin negative (especially women).	within 2 hours of onset of symptoms, administer lytic therapy then transfer. Fibrinolysis is recommended for patients with symptom onset within 12 hours who cannot receive primary PCI within 120 minutes of first medical contact. Time from hospital arrival to initiation of fibrinolytic drug infusion (door-to-needle time) should <30 minutes. High risk of bleeding with fibrinolysis.
Anticoagulant therapy	Choice of agent (enoxaparin, bivalirudin, fondaparinux or unfractionated heparin [UFH]) depends on intervention strategy planned.	• UFH to maintain therapeutic activated clotting time (ACT). If given with GP IIb/IIIa receptor antagonist planned: 50-70 U/kg IV bolus; if no GP IIb/IIIa receptor antagonist planned: 70-100 U/kg bolus • Bivalirudin 0.75 mg/kg IV bolus, then 1.75 mg/kg/h infusion with or without prior treatment with UFH. (Preferred if high risk of bleeding). • Fondaparinux: not recommended as sole anticoagulant for primary PCI.

Risk Assessment

- **Early Risk Stratification (UA/NSTEMI)**: identify patients at highest risk for future cardiac events.
    - Presence and extent of ST segment depression
    - Elevated cardiac biomarkers

- Evidence of hemodynamic instability
- Persistent chest pain despite appropriate medical therapy

- **Thrombolysis in Myocardial Infarction (TIMI) Risk Score (Antman, Cohen, & Bernink, 2000)** Seven variables at presentation were independently predictive of outcome in patients with unstable angina or an acute non-ST elevation MI (1 = present, 0 = absent)
  - Age ≥ 65 years
  - Presence of at least 3 risk factors for coronary heart disease (hypertension, diabetes, dyslipidemia, smoking, or positive family history of early MI)
  - Prior coronary stenosis ≥ 50%
  - Presence of ST segment deviation on admission electrocardiogram
  - At least 2 anginal episodes in prior 24 hours
  - Elevated serum cardiac biomarkers
  - Use of aspirin in prior 7 days (possible marker of more severe coronary disease)
  - **TIMI Scoring**:
    - Low risk score = 0 to 2
    - Intermediate risk score = 3 to 4
    - High risk score = 5 to 7

References:

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