MYOCARDIAL INFARCTION
Acute Coronary Syndrome

No ST Elevation

NSTEMI

Unstable Angina

ST Elevation

Myocardial Infarction

NQMI

QwMI
Unstable Angina

- In patients with unstable angina, anticoagulant and antiplatelet drugs play a major role in therapy. Aggressive therapy with antilipid drugs, heparin, and antiplatelet agents is recommended.

- In addition, therapy with nitroglycerin and β-blockers should be considered; calcium channel blockers should be added in refractory cases.
Myocardial Infarction is the rapid development of myocardial necrosis by a critical imbalance between oxygen supply and demand to the myocardium.
Pathophysiology of Myocardial Infarction: Disrupted Plaque

- Thin cap subject to enzymatic degradation
- High macrophage content and activation

Mature Plaque
- Large lipid core
- Extracellular matrix proteins in fibrous cap

In case of complete coronary occlusion:
- Plaque rupture
- Platelet aggregation

- Acute myocardial infarction
- Spontaneous lysis, repair, and wall remodeling

In case of incomplete coronary occlusion:
- Future high-risk lesion
- Unstable angina or non-Q-wave myocardial infarction

Mechanisms of Myocardial damage

The severity of an MI is dependent of three factors:

- The level of the occlusion in the coronary
- The length of time of the occlusion
- The presence or absence of collateral circulation
History

- Chest Pain- anterior precordium tightness
- Pain may radiate to jaw, neck and epigastrium
- Dyspnea-
- Anxiety
- Nausea with and without vomiting
- Diaphoresis or sweating
- Syncope or near syncope
- As many as half of MI are clinically silent
Physical Exam.

- The physical exam can often be unremarkable
- Hypertension
- Hypotension
- Acute valvular dysfunction may be present
- Rales
- Neck vein distention
- Third heart sound may be present
- A fourth heart sound poor LV compliance
- Dysrhythmias
Diagnosis: Cardiac Biomarkers

- Cardiac biomarkers are protein molecules released into the blood stream from damaged heart muscle.
Electrocardiogram

- A normal ECG does not exclude ACS
- High probability include ST segment elevation in two contiguous leads or presence of q waves
- Intermediate probability ST depression
- T wave inversions are less specific
Diagnosis
Evolution of ECG changes in Myocardial Infarction

Hours

0

4

6

12

24

96

2 wks.

6 wks.

1 yr.

Hyper acute phase
The goals of therapy in AMI are the expedient restoration of normal coronary flow and the maximum salvage of functional myocardium.
Antiplatelet Agents

- **Aspirin** at least 160mg immediately
- Interferes with function of cyclooxygenase and inhibits the formation of thromboxane
- ASA alone has one of the greatest impact on the reduction of MI mortality.
- **Clopidogrel, ticlopidine**, have not been shown to be superior to Aspirin in acute MI
Supplemental Oxygen

- Because MI impairs the circulatory function of the heart, oxygen extraction by the heart and other tissues may be diminished
Nitrates

- IV nitrates to all patients with MI and congestive heart failure, persistent ischemia, hypertension, or large anterior wall MI
- Primary benefit: vasodilator effect
- Vasodilatation reduces myocardial oxygen demand and preload and afterload
When administered sublingually or intravenously, nitroglycerin has a rapid onset of action.

Clinical trial data have supported the initial use of nitroglycerin for up to 48 hours in MI.
Pain Control

- Pain from MI is often intense and requires prompt and adequate analgesia. The agent of choice is morphine sulfate, given initially IV at 5 to 15 minute intervals at typical doses of 2 to 4 mg.
- Reduction in myocardial ischemia also serves to reduce pain, so oxygen therapy, nitrates, and beta blockers remain the mainstay of therapy.
Thrombolytics

- Indicated with MI and ST segment elevation who present less than 12 hours but not more than 24 hours after symptom onset
- The most critical variable in achieving successful fibrinolysis is time form symptom onset to drug administration
Thrombolytics...

- As a class the plasminogen activators have been shown to restore coronary blood flow in 50-80% of patients
- Contraindication active intracranial bleeding, CVA 2months, CNS neoplasm, HTN, coagulopathy
- Intracranial bleed risk major drawback
- Eg. Streptokinase 15lakh units in 100ml NS infused IV over 1hr
Beta-blockers

- Recommended within 12 hours of MI symptoms and continued indefinitely
- Reduces Myocardial mortality by decreasing death due to arrhythmias
- Decrease the rate and force of myocardial contraction and decreases overall oxygen demand
Beta Blocker Therapy

- **Metoprolol**
  - 15 mg IV × 1 then 200 mg/day PO in divided doses

- **Atenolol**
  - 5-10 mg IV × 1, then 100 mg/day PO

- **Carvedilol**
  - 6.25 mg bid titrated to 25 mg BID
Angiotensin-Converting Enzyme Inhibitors

- should be used in all patients with a STEMI without contraindications.
- Also recommended in patients with NSTEMI who have diabetes, heart failure, hypertension, or an ejection fraction less than 40%. In such patients, an ACE inhibitor should be administered within 24 hours of admission and continued indefinitely.
- Benefit of ACE inhibitor therapy can likely be extended to all patients with an MI.
Contraindications to ACE inhibitor use include hypotension and declining renal function. ACE inhibitors:

- **Captopril**: 6.25 mg bid titrated to 50 mg bid started within 24 hr of MI
- **Lisinopril**: 5 mg/day titrated to 10 mg/day : started within 24 hr of MI
- **Ramipril**: 1.25 mg bid titrated to 5 mg bid, 3-10 days post-MI with symptoms of heart failure
Unfractionated heparin

-Forms a chemical complex with antithrombin III inactivates both free thrombin and factor Xa

-Recommended in patients with MI who undergo PTCA or fibrinolytic therapy with alteplase
Unfractionated Heparin Dosing

Loading Dose

- 60 U/kg IV bolus
- Max 5000 U if >65 kg or 4000 U if <65 kg

Maintenance Dose

- 12 U/kg/hr IV
- Max 1000 U/hr if >65 kg or 800 U/hr if <65 kg

Titration Goal

- PTT 50-70 sec
Low-molecular weight heparin

- Direct activity against factors Xa and IIa
- Proven to be effective in treating ACS that are characterized by unstable angina or non ST-elevation MI
- Their fixed doses are easy to administer and laboratory testing to measure their therapeutic effect is not necessary makes them attractive alternative of un-fractioned heparin
Low-Molecular-Weight Heparin

- **Dalteparin**
  - $t_1/2$: 3-5 hr
  - 120 U/kg SC bid
  - Prevention of ischemic complications in UA and NSTEMI

- **Enoxaparin**
  - 4.5 hr
  - 100 U/kg (1 mg/kg) SC q12h
  - Prophylaxis of ischemic complications of UA and NSTEMI when administered with aspirin

UA, unstable angina; NSTEMI, non–ST segment elevation myocardial infarction.
A statin should be started in all patients with a myocardial infarction without known intolerance or adverse reaction prior to hospital discharge. Preferably, a statin would be started as soon as a patient is stabilized after presentation.

- *Eg.* Simvastatin 20-80mg, Pravastatin,

- Trials show benefit of starting patients on high-dose therapy from the start (e.g., atorvastatin 80 mg/day).
Long term Medications

- Most oral medications instituted in the hospital at the time of MI are continued long term
- Aspirin, beta blockers and statin are continued indefinitely
- ACEI indefinitely in patients with CHF, ejection fraction < .40, hypertension, or diabetes