CHAPTER-I

MYOCARDIAL INFARCTION
**Definition**

- A myocardial infarction, more commonly known as MI or acute myocardial infarction (AMI) or heart attack is a condition where there is interruption of blood supply to a part of the heart.
Plaque build up in the coronary artery blocking blood flow and oxygen to the heart

Damage and death to heart tissue shown in purple
PATHOPHYSIOLOGY
Causes:
- Coronary Atherosclerotic Heart Disease
- Coronary Thrombosis/Embolism
- Decreased Blood Flow with Shock and/or Hemorrhage

Myocardial Ischemia → ↓ Myocardial Oxygen Supply → ↑ Cellular Hypoxia

↓ Cardiac Output

↓ Arterial Pressure

↓ Myocardial Contractility

Stimulation of Baroreceptors

↑ Peripheral Vasoconstriction → ↑ Afterload

↑ Myocardial Contractility → ↑ Heart Rate

↓ Diastolic Filling

Decreased Myocardial Tissue Per.

Myocardial Oxygen Demand
Predisposing Factors

Premature, Accelerated Atherosclerosis

Progressive narrowing of Blood Vessels

Ischemia of the heart muscles

Risk for excessive blood clot formation

Thromboembolism

Hypoxia

necrosis

aerobic to anaerobic metabolism

Lactic Acid Formation

Chest pain/muscle spasm

Release of lysozymal enzyme

Altered depolarization

Altered repolarization

Myocardial contractility

Cardiac output

Renal Ischemia/Oligurina

Myocardial Infarction

www.nusingdepartment.blogspot.com
Pathophysiology of Coronary Heart Disease

Risk Factors

- Modifiable
  - Smoking (29 yrs)
  - HPN (2 yrs)
- Non-Modifiable
  - Age: 49
  - Gender: Male
  - Heredity: Family History of HPN (Maternal)

Vasoconstriction

- Contributes to
  - Goes to blood vessels of mouth and near by structures
  - Decrease Blood Supply
  - Decrease Tissue Perfusion
  - Numbness of Tongue
  - Choking Sensation

Modifiable

Non-Modifiable

Non-specific injury to arterial wall (endothelial lining)

- Desquamation of Endothelial lining
  - Increase permeability/Adhesion molecules
  - Lipids and Platelets assimilate into the area
  - Plaque Formation

Atherosclerosis

- Part of plaque formation can be dislodged because of HPN
  - Embolus
  - Goes to Blood Vessels of Brain
    - Clogs the Blood Vessels
    - Decrease Blood Supply
    - Decrease Oxygen Supply
  - In Myocardium
    - Infarct
    - Chest Pain
    - Remaining functional tissues will composite to maintain normal cardiac output especially the left ventricle
    - Hypertrophy
  - In Brain
    - Infection
    - Mild Stroke
COAGULATION CASCADE (making a clot)

The activation of Factor X is caused by the coagulation cascade regulators upstream (not discussed here).

Antithrombin

Low-molecular-weight heparin

Factor Xa

Prothrombin

Fibrinogen

ASAP

Platelets

Factor XIIIa

Fibrin

Cross-linked fibrin

Fibrin split products, D-dimers

FIBRINOLYTIC PATHWAY (breaking a clot)

t-PA (tissue plasminogen activator)

Plasmin

Plasminogen

Urokinase, streptokinase
The three pathways that makeup the classical blood coagulation pathway

**Intrinsic**
- surface contact
  - XII → XII$_a$
  - XI → XI$_a$
  - IX → IX$_a$
  - (VIII, PL, Ca++)

**Extrinsic**
- tissue damage
  - TF:VII$_a$

**Common**
- prothrombin → thrombin (serine protease)
- fibrinogen → fibrin → stable fibrin clot
- thrombin
- Ca$^{++}$
- PL
- VI
- XII
- XI
- IX
- VIII
- VII
- V
- PL
- Ca$^{++}$

- Hageman factor, a serine protease
- Plasma thromboplastin, antecedent serine protease
- Christmas factor, serine protease
- Stable factor, serine protease
- Fibrin stabilising factor, a transglutaminase
- Platelet membrane phospholipid
- Calcium ions
- Tissue Factor (a = active form)
Clinical Presentation

- __________, __________, __________
- ____________________
- __________, _________
- ____________ and/or ____________
- ________ (more
- ______
**DIAGNOSIS**

**Physical Examination:**

Pallor, diaphoresis, tachycardia, S4, dyskinetic cardiac impulse may be present. If CHF exists, rales and S3 are present. Jugular venous distention is common in right ventricular infarction.

**ECG:** ST elevation, followed by T-wave inversion, then Q-wave development over several hours.

Non-ST Elevation MI, or NSTEMI ST depression followed by persistent ST-T-wave changes without Q-wave development.

**Cardiac Biomarkers** Cardiac-specific troponins T and I are highly specific for myocardial injury and are the preferred biochemical markers for diagnosis of MI.
- Heart failure
- Ventricular fibrillation
- High Blood Cholesterol (Hyperlipidemia).
- Diabetes (Diabetes Mellitus).
- Male Gender
- Family History of Heart Disease.
- High Blood Pressure (Hypertension).
- Smoking or tobacco use
TREATMENT
Nonpharmacological treatment

- Keep your blood pressure, blood sugar, and cholesterol under control.
- Don't smoke.
- Eat a heart-healthy diet rich in fruits, vegetables, and whole grains, and low in animal fat.
- Get plenty of exercise, at least 30 minutes a day, at least 5 days a week.
- Get checked and treated for depression.
- Limit yourself to no more than one drink a day for women, and no more than two drinks a day for men.
- Stay at a healthy weight. Aim for a body mass index (BMI) of between 18.5 and 24.9.
Antiplatelet and antithrombotic therapy

- Aspirin
- Clopidogrel
- Ticlopidine
- Unfractioned heparin
- Low molecular weight heparin
  - (e.g. enoxaparin, dalteparin, tinzaparin)
- GPIIb/IIIa inhibitors
Antiplatelet drugs

- ADP antagonists (Thienopyridines)
  - Tirodipine
  - Clopidogrel
  - Prasugrel

- COX inhibitors
  - Aspirin

- Phosphodiesterase inhibitors
  - Diprydarnole

- GP IIb/IIIa Inhibitors
  - Tirofiban
  - Eptifibatide
  - Abciximab
Fibrinolytic therapy

- Streptokinase
- Alteplase
- Retepase
- Tenecteplase
Analgesics

- Morphine sulfate IV
- Nitroglycerine
Beta blockers

- Propranolol
- Metaprolol
- Atenolol
Other therapies

- ACE inhibitors
- Calcium channel blockers
- Amiodarone
- Lidocaine
Antiplatelets

Aspirin has an antiplatelet effect which inhibits formation of further thrombi (blood clots) that clog arteries. An antiplatelet drug (antiaggregant) is a member of a class of pharmaceuticals that decrease platelet aggregation and inhibit thrombus formation.
Active GP IIb/IIla receptors

Ca^{2+}

ADP

Damaged endothelial cells

Ticlopidine and clopidogrel inhibit ADP-mediated platelet aggregation.
Heparin

- Heparin binds to the enzyme inhibitor antithrombin III (AT). The activated AT then inactivates thrombin and other proteases involved in blood clotting, most notably factor Xa. The rate of inactivation of these proteases by AT can increase by up to 1000-fold due to the binding of heparin.

- It produces its major anticoagulant effect by inactivating thrombin and activated factor X (factor Xa) through an antithrombin (AT)-dependent mechanism. Heparin binds to AT through a high-affinity pentasaccharide, which is present on about a third of heparin molecules. For inhibition of thrombin, heparin must bind to both the coagulation enzyme and AT, whereas binding to the enzyme is not required for inhibition of factor Xa.
GPIIb/IIIa antagonists

- GPIIb/IIIa antagonists.
  - Platelet GPIIb/IIIa inhibitors block the final pathway of platelet aggregation
Fibrinolytic agent

- Improve myocardial oxygen supply by dissolving coronary thrombus, reestablishing blood flow to ischemic myocardium. Prompt therapy limits infarct size and improves survival
Morphine sulphate

- Reduce myocardial oxygen demand by causing peripheral arterial dilatation that decreases systemic vascular resistance and afterload, circulating concentration of catecholamine.
Nitroglycerin under the tongue or buccally can be given is useful to alleviate chest pain. The Glyceryl Trinitrate acts as a nitric oxide donor to smooth muscles cells adjacent to the coronary artery endothelium resulting in increased vasodilation and increased coronary blood flow.
Beta blockers

\(\beta\)-blockers decrease the effect of the sympathetic nervous system on the heart. Since it is known that the sympathetic nervous system increases the heart rate and blood pressure in order to increase the cardiac output. Hence its blockage spares the heart the extra work load.
EMERGENCY TREATMENTS

- **Angioplasty** is a procedure to open narrowed or blocked blood vessels that supply blood to the heart. Usually a small, metal mesh tube called a stent is placed at the same time.

- Angioplasty is often the first choice of treatment. It should be done within 90 minutes after you get to the hospital, and no later than 12 hours after a heart attack.
A stent is a small, metal mesh tube that opens up (expands) inside a coronary artery. A stent is often placed after angioplasty. It helps prevent the artery from closing up again.

Some patients may also have heart bypass surgery to open narrowed or blocked blood vessels that supply blood to the heart. This procedure is also called open heart surgery.