Ischemic heart disease (IHD)
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The coronaries:

1- Left anterior descending coronary artery:
   - supplies anterior portion of LV, anterior 2/3 of IVS
   - accounts for 40-50% of coronary artery thrombosis

2- Left circumflex coronary artery:
   - supplies the lateral wall of the LV
   - accounts for 15% to 20% of coronary artery thrombosis

3- Right coronary artery:
   - supplies posterior and inferior part of the LV, posterior 1/3 of IVS, the all RV, posteromedial papillary muscle in LV and both atrioventricular and sinoatrial node
   - accounts for 30% to 40% of coronary artery thrombosis

Ischemic Heart Disease (IHD)

- is a group of diseases caused by myocardial ischemia due to imbalance between:
  - the myocardial oxygen demand and
  - supply from the coronary arteries.
- Majority of cases due to atherosclerosis

Epidemiology:
- is the major cause of death in US (500,000 deaths/year)
- is more common in men (peaks in men after age 60 and women after age 70)

**Types:**
- there are four types of IHD:
  1- Angina pectoris (Most common)
  2- Acute Myocardial infarction (AMI)
  3- Chronic IHD
  4- Sudden cardiac death (SCD)

**Pathogenesis:**
- inadequate coronary supply relative to myocardial demand, due to:
  1- pre-existing atherosclerotic occlusion
  2- new superimposed thrombosis (to AS)
  3- vasospasm

- obstruction of 70% to 75% or more causes symptomatic ischemia on exertion
- obstruction of 90% can cause symptomatic ischemia even at rest

**1- Angina pectoris:**
- is an intermittent chest pain caused by transient reversible myocardial ischemia
- the ischemia is insufficient to cause death of myocardium
- three Types:
  1- Stable angina
2- Prinzmetal’s angina (Variant angina)
3- Unstable angina

1- **Stable angina:**
   - most common type
   - characterized by recurrent chest pain due to increased physical activity
   - Pathogenesis:
     - caused by fixed coronary obstruction (>75%)
     - with this narrowing, oxygen supply to heart is sufficient during rest, but becomes insufficient on increased demand (exertion)
   
   - C/F: sudden onset of exercise induced substernal pain lasts 30 seconds to 30 min crushing or squeezing radiated to left arm or to left jaw relieved by rest or nitroglycerin
   - ECG: ST segment depression

2- **Prinzmetal’s angina:**
   - Angina occurring at rest due to coronary artery spasm (thromboxane A2)
   - Stress ECG reveals ST elevation (representing transmural ischemia)

3- **Unstable angina:**
   - characterized by frequent bouts of chest pain at rest or with minimal exertion
   - may progress to acute MI
   - Pathogenesis: associated with plaque disruption with superimposed partial thrombosis
- Stress ECG is unsafe

2- **Myocardial infarction**
   - necrosis of heart muscle resulting from ischemia due to occlusion of one or more of the three main coronary arteries
   - major underlying cause of MI is Atherosclerosis

**Pathogenesis:**
- sudden disruption of an atheromatous plaque
- exposure subendothelial collagen
- platelet adhesion, aggregation, activation
- thrombus formation occlusion ischemia infarction
- thrombosis common in Lt anterior descending coronary artery > Rt coronary artery > Lt circumflex coronary
- MI occurs most commonly in the LV and IVS
- pure right ventricular infarcts are rare

**Coronary artery atherosclerosis:**
- coronary artery is almost completely occluded by atherosclerotic plaque
- thrombus has occluded the tiny lumen that remains

**Acute myocardial infarct:**
- The infarct zone is pale tan
Myocardial Response to Ischemia:
- within seconds: myocyte aerobic glycolysis ceases, switching to Anaerobic glycolysis for ATP
- if ischemia lasts for less than 2 min: loss of contractility
- ischemia lasts between 1 - 10 minutes causes reversible injury to myocytes
- ischemia lasts 20-40 minutes causes irreversible injury to myocytes
- If myocardial blood flow is restored before 20-40 minutes (reperfusion) myocyte viability may be preserved
- reperfusion can cause injury and changes in necrotic myocardium
- it produces:
  1- contraction band necrosis in damaged myocytes
     * are eosinophilic transverse bands
     * composed of hypercontracted sarcomeres
  2- Hyper-contraction of myofibrils in dead cells due to the influx of Ca2+

- reperfusion: can be achieved by:
  1- thrombolytic therapy (e.g tissue plasminogen activator, streptokinase)
  2- Angioplasty

Morphology:
- during 0 to 24 hours:
  - Gross: no changes
  Normal
Necrosis
- Microscopy: coagulative necrosis without neutrophil infiltrate
- **during 1-3 days:**
  - Gross: shows pallor of infarcted myocardium
  - Microscopy: Myocyte nuclei and striations disappear
  - Infiltration by neutrophils (lyse dead myocytes)
Normal 1 – 3
Pallor infarcted area

- **during 4 to 7 days:**
  - red granulation tissue surrounds area of infarction
  - Macrophages begin removal of necrotic debris
  - Period of maximal softness (time for rupture)
- **during 7 to 10 days:**
  - Necrotic area is bright yellow
  - Granulation tissue and collagen formation are well developed
- **during 2 months:**
  - infarcted tissue replaced by white, patchy, noncontractile fibrous tissue

**Types of MI:**
1- **Transmural infarction:** (Q wave infarction)
  - involves the full thickness of the myocardium
  - new Q wave develops in an ECG
  - occurs due to complete occlusive thrombus
  - are larger; and have higher mortality
2- **Subendocardial infarction:** (non Q wave infarction):
- involves the inner third of the myocardium
- Q waves are absent.
- occurs due to partial occlusive thrombus
- are smaller; less mortality
- associated with increased risk of reinfarction & sudden cardiac death

**Clinical findings:**
- Sudden onset of severe retrosternal pain: * lasts more than 30 minutes
  * not relieved
  by nitroglycerin
  * radiates
  down the left arm, shoulder, jaw
  * associated
  with sweating, anxiety and hypotension
- Epigastric pain: - mainly due to right coronary artery involvement
  - mistaken for gastroesophageal reflux
  associated pain
- “Silent” Acute MI:
  - may occur in elderly and in individuals with DM
  - due to high pain threshold or problems with nervous system

**Diagnosis:**
1- ECG: inverted T wave, elevated ST segment, new Q wave
2- Cardiac enzymes:
   - Are released when myocytes are damaged
   - Include: 1- Creatine kinase and isoenzyme CK-MB:
     - Appears within 4-8 hours

Peaks in 24 hours

Disappears in 1 - 3 days
   2-Troponin: - Appear within 3-6 hours
     - Peak at 24 hours
     - Disappear within 7-10 days
   3- Lactate dehydrogenase: - Appears within 10 hours
     - Peaks at 2-3 days - disappears within 7 days
   4- Aspartate aminotransferase (AST): not specific, less used

Complications:
1- Arrhythmias
   - Ventricular premature contractions (MC)
   - Most common cause of death is ventricular fibrillation
2- Cardiogenic shock: - Usually occurs within first 24 hours
   - If more than 40% of ventricle is infarcted
3- Congestive heart failure (CHF)
4- Rupture:
   - most common on 3rd to 7th day
     i- Anterior wall rupture: - associated with thrombosis of the LAD
        - hemopericardium, compression of heart (cardiac tamponade)
     ii- Papillary muscle rupture: - associated with RCA thrombosis
        - leads to acute onset of mitral valve regurgitation
     iii- Interventricular septum rupture: - associated with thrombosis of LAD
        - produces left to right shunt causing Rt-sided HF

Anterior wall rupture  IVS rupture
Papillary muscle rupture
Anterior wall rupture  Interventricular septum
Rupture papillary muscle

5- Mural thrombus:
   - adjacent to noncontractile area
   - risk of embolism

6- Ventricular aneurysm:
- clinically recognized within 4 to 8 weeks:
  ** Precordial bulge during systole
  Blood enters the aneurysm causing
  anterior chest wall movement

7- Fibrinous pericarditis with or without effusion:
   - days 1-7 of transmural acute MI
   - Substernal chest pain relieved by leaning forward
   - Precordial friction rub is present
   - due to increased vessel permeability in the pericardium.
8- Autoimmune pericarditis: (Dressler’s syndrome)
   - develops 6 to 8 weeks after an MI
   - Autoantibodies are directed against pericardial tissue (antigen)
   - Fever. Joint pain and pericardial friction rub

**Treatment:**
- aims of treatment:
  - relief of pain (Morphine)
  - thrombolysis (streptokinase)
  - prophylaxis for arrhythmias (lidocaine)
  - low flow oxygen
  - aspirin (reduce risk of thrombosis)
  - reduce afterload (beta blockers)
  - reduce preload (diuretics)

**3- Chronic Ischemic Heart disease**
- progressive heart failure as a consequence of ischemic myocardial damage
- In most cases there is a history of MI
- causes: 1- usually results from postinfarction cardiac decompensation
- 2- in other cases severe obstructive CAD may be present without prior infarction, but with diffuse myocardial dysfunction
- is seen typically in elderly patients who insidiously develop CHF
- C/F: CHF
- diagnosis depends on exclusion of other CHF causes
- death can result from: 1- slowly progressive CHF
- 2- superimposed acute MI
- 3- arrhythmia

4- Sudden cardiac death
- defined as unexpected death from cardiac causes either without symptoms or within 1 to 24 hours of symptom onset
- in many adults SCD is the first clinical manifestation of IHD
- Pathogenesis: - severe atherosclerosis with superimposed partial or complete occlusive thrombosis
- Ultimate mechanisms: - lethal arrhythmia (ventricular arrhythmia) triggered by acute ischemia without infarction
- in younger victims other nonatherosclerotic causes are more common:
1- Congenital coronary arterial abnormalities  
2- Aortic valve stenosis  
3- Mitral valve prolapse  
4- Myocarditis  
5- Dilated or hypertrophic cardiomyopathy  
6- Pulmonary hypertension  
- some young individuals who die suddenly (including athletes) have unsuspected hypertrophic cardiomyopathy, myocarditis, or congenital abnormalities of coronary arteries.