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 Ca^{2+}

- 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:
  ** Precardial 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

اللهمّ انفعني بما علمتني وعلمني ما ينفعني وزدني علما.

الحمد لله على كل حال وأعوذ بالله من حال أهل النار