ISCHAEMIC HEART DISEASE

BY KIMINA

- Also known as **CORONARY ARTERY DISEASE(CAD)**
- **IHD** is a condition in which there is insufficient blood supply to the myocardium to meet functional demand.
- Imbalance between **myocardial oxygen supply and demand.**
- Caused mainly by **ATHEROSCLEROSIS OF CORONARY ARTERY.**
- There are 4 basic clinical syndrome of IHD

Clinical syndrome of IHD

- Angina pectoris ;in this case ischaemia causes pain but is insufficient to cause myocardial death.3 types of Angina .

  - stable angina;occurs on exertion

  -variant angina (prinzmetal);due to coronary artery spasm

  -unstable Angina;occurs progressively or even at rest

- Acute myocardial infaction;the severity or duration of ischaemia ,is enough to cause cardiac myocytes death.
- Chronic Ischaemic heart disease;it refers to progressive cardiac decompensation (heart failure)following myocardiac infaction
- Sudden cardiac death;this result from lethal arrhythmia

RISK FACTORS

Modifiable

- Cigarette smoking
- Hyperlipidemia
- Hypertension
- Diabetes
- High density lipoprotein levels
- obesity

Non -modifiable

- Age
- Male sex
- Family history

Other risk factors

- Physical inactivity
- Diet
- Alcohol consumption
- Oral contraceptives

Pathogenesis of IHD

- The underlying pathogenesis of IHD is **ATHEROSCLEROSIS.**
- Can affect any artery in the body ,tend to occur at sites of altered arterial shear stress such as bifurcation.
- Starts with any abnormal endothelial function
- Inflammatory cells ,predomantly monocytes,bind to receptor expressed by endothelial cells
- Migrate to intima
- Take up oxidized low density lipoprotein particles
- Become lipid laden macrophages or foam cells

Cont

- As form cell dies it releases its lipid pool in intimal space with cytokines and growth factor
- In response smooth muscle cells migrate from the media of the arterial wall into the intima
- Lipid core will be covered by smooth muscle cells matrix
- Form stable atherosclerotic plaque that will remain asymptomatic until it becomes large enough to obstruct arterial flow.

Cont

- IHD occurs because of inadequate coronary perfussion relative to myocardial demand
- Can result from a combination of ;
  - pre existing (fixed) atherosclerotic occlusion of coronary artery
  -new superimposed thrombosis and or vasospasms
- A lesion obstructing 70-75% or more of a vessel lumen is referred to as CRITICAL STENOSIS and it causes asymptomatic ischaemia (Angina) only in the setting of increased demand.
- A fixed steinosis of 90% can lead to inadequate coronary blood flow even at rest (unstable angina)
- If a coronary artery develops an atherosclerotic occlusion at a slow rate it stimulates colateral blood flow from other major epicardial vessels.
- Such colateral perfussion can then protect against myocardial infarction even in setting of a complete vascular occlusion
- Acute coronary occlusions cannot spontaneously recruit colateral flow and will result in infaction

Role of acute plaque change

- In most patients ,unstable angina ,infarction and sudden cardiac death all occur because of abrupt plaque change followed by thrombosis .
- The initiating events is typically disruption of a plaque due to the following;

-rapture or ulceration of plaques this exposes highly thrombogenic plaques constituents or the underlying subendothelial basement membrane

-hemorrhage into the core of plaque this results in expansion of plaque volume and worsening of luminal occlusion

Role of thrombus

- Thrombus associated with a disrupted plaque is critical to pathogenesis of IHD .
- Partial vascular occlusion by a newly formed thrombus on a disrupted atherosclerotic plaque can worsen with time and lead to unstable angina or sudden death.
- Partial luminal occlusion can compromise blood flow sufficiently to cause a small infarction of the innermost zone of the myocardium .

ANGINA

- A type of chest pain ,not a disease but a symptom of an underlying hesrt problem especially IHD
- Described as heavy,tight or gripping .
- Types of Angina;

-stable angina; this is pain that occurs on exertion when blood supply to myocardium is inadequate ,usually due to severe significant narrowing of coronary arteries .relieved by rest

-variant(prinzmetal's)angina ;occurs without provocation usually at rest as aresult of coronary artery spasm,more frequently in women.

-unstable angina ;clinically characterized by transient episodic myocardial

MYOCARDIAL INFACTION

- Is defind as death of myocardial tissue caused by ischaemia .
- Popularly known as HEART ATTACK.
- An MI occurs when there is diminished blood supply to heart which leads to myocardial cell damage
- Contractile function stop in the necrotic area of heart
- Ischaemia results from blockage of coronary vessels
- This blockage is often as a result of thrombus tht is superimposed on an ulcerated or unstable atherosclerotic plaque formation in coronary artery

Pathophysiology

- Ischaemia develops when there is an increased demand for oxygen or decresed supply of oxygen .
- It can develop within 10 seconds and if it last longer than 20 minutes irreversible cell and tissue death occurs
- Myocardial cell death begins at the endorcardium.the area most distal to the arterial blood suppy
- As vessel occlusion continues cell death spreads to the myocardium and eventually to the epicadium

cont

- Sevirity of an MI depends depends on three factors;
  - -level of occlusion
  - -length of time of occlusion
  - -presence or absence of collateral circulation

Clinical feature

- Severe crushing substernal chest pain or discomfort tht can radiate to the neck,jaw,epigastric or left arm
- In constrast to the pain of Angina pectoris,the pain of MI typically lasts for 20 mins to several hours and its not relieved by rest or nitrate administration.
- Pulse is generally rapid and weak and patients can be diaphoretic .
- Dyspnoea is common and is caused by impaired myocardial contractility and dysfunction
- With massive MI (>40% of left ventricle)cardiogenic shock develops

ECG ABNORMALITIES

- They include changes such as Q-waves ;indicating transmural infacts
- S-T segment abnormalities and T-wave inversion;represents abnormalities in MI

LAB Evaluation

- Based on measuring the blood levels of intracellular macromolecules that leak out of injured myocardial cells through damaged cell membrane.
- They include;

  -Myoglobulin

  -Cardiac troponins I and T.

  -Creatinine kinase-myocardial band

  -lactate dehydrogenase
- Troponins and CK-MB have high specificy and severity to myocardial damage .
- TnI and TnT are not normally detected in the circulation but after 2-4 hrs and reaches peak at 48 hrs ,their levels remain elavated for 7-10 days.