SNS COLLEGE OF ALLIED HEALTH SCIENCES

SNS Kalvi Nagar, Coimbatore - 35

Affiliated to Dr MGR Medical University, Chennai

DEPARTMENT OF CARDIOPULMONARY PERFUSION CARE TECHNOLOGY

COURSE NAME: PATHOLOGY II

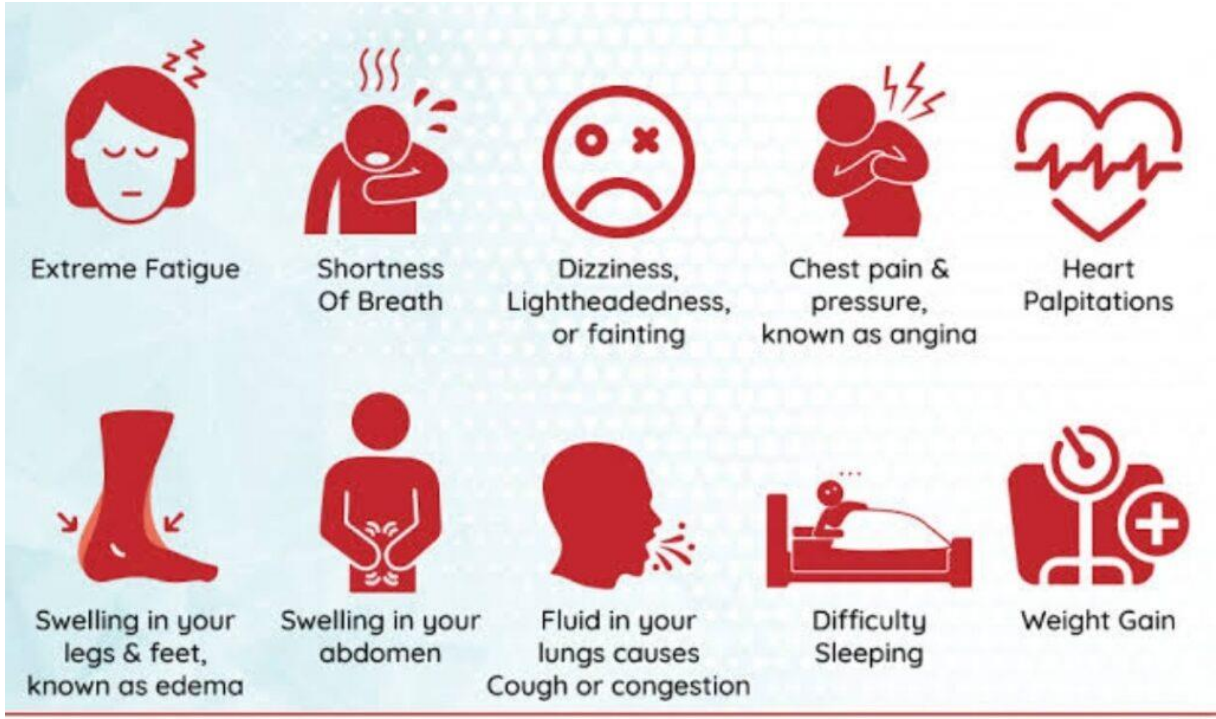
II YEAR

UNIT I: PATHOLOGY OF HEART

TOPIC: ISCHEMIC HEART DISEASES

Case Study

A 50 year old man presents to clinic with a complaint of central chest discomfort of 2 weeks' duration, occurring after walking for more than 5 minutes or climbing more than 1 flight of stairs. The chest discomfort resolves with rest within several minutes. He is obese, has a history of hypertension, and smokes 10 cigarettes a day. His father died from a myocardial infarction at the age of 54 years.

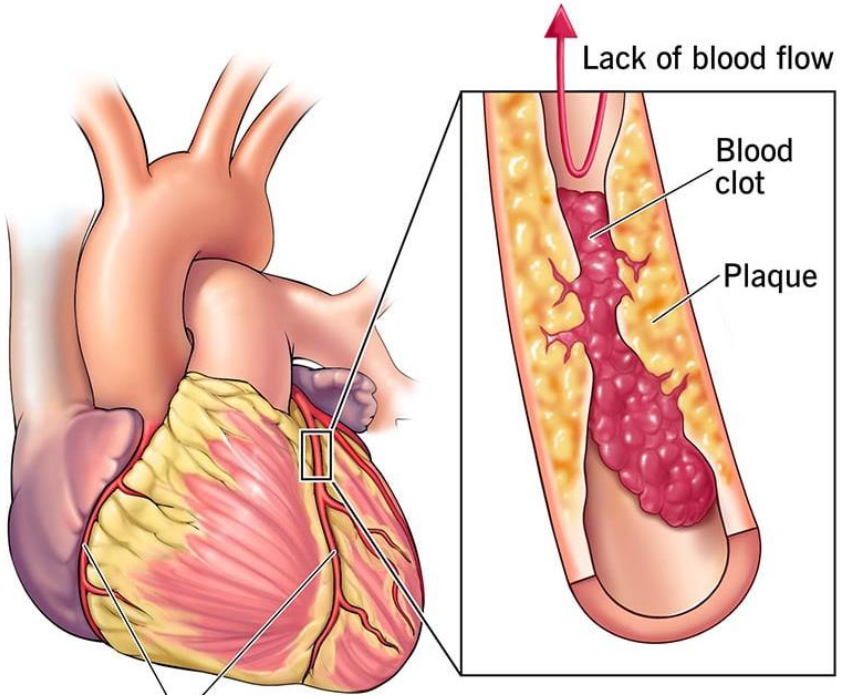


ISCHAEMIC HEART DISEASE

- *Ischaemic heart disease (IHD)* is defined as **acute or chronic form** of cardiac disability arising from imbalance between the myocardial supply and demand for oxygenated blood.

- Narrowing or obstruction of the coronary arterial system is the most common cause of **myocardial anoxia.**

- The alternate term **'coronary artery disease (CAD)'** is used synonymously with IHD



EPIDEMIOLOGY

- IHD or CAD is the leading cause of death in most developed countries (about one-third of all deaths)

- Men develop IHD earlier than women and death rates are also slightly higher for men than for women until the menopause.

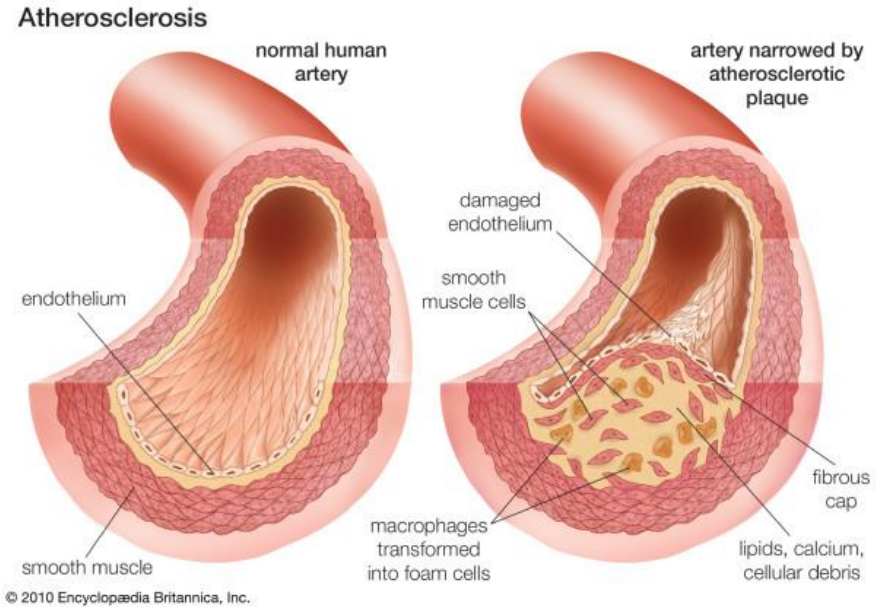
- As per rising trends of IHD worldwide, it is estimated that in the year 2020, the most common cause of death throughout world.

ETIOPATHOGENESIS

- IHD is invariably caused by disease affecting the coronary arteries, the most prevalent being atherosclerosis accounting for more than 90% cases, while other causes are responsible for less than 10% cases of IHD.

IHD under three broad headings

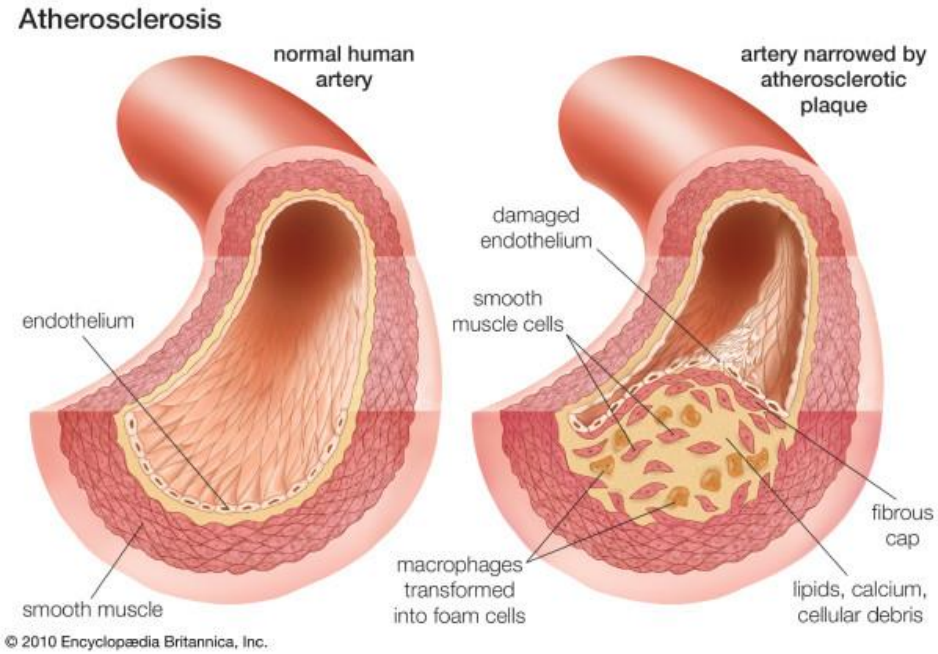
- **coronary atherosclerosis**
- **superadded changes in coronary atherosclerosis**
- **non-atherosclerotic causes**



CORONARY ATHEROSCLEROSIS

- Atherosclerosis is a specific form o arteriosclerosis affecting primarily the **intima** of large and medium-sized muscular arteries and is characterised by fibrofatty plaques or atheromas.

- The term atherosclerosis is derived from *athero-* referring to the **soft lipid-rich material** in the centre of atheroma, and *sclerosis* (scarring) referring to **connective tissue in the plaques.**



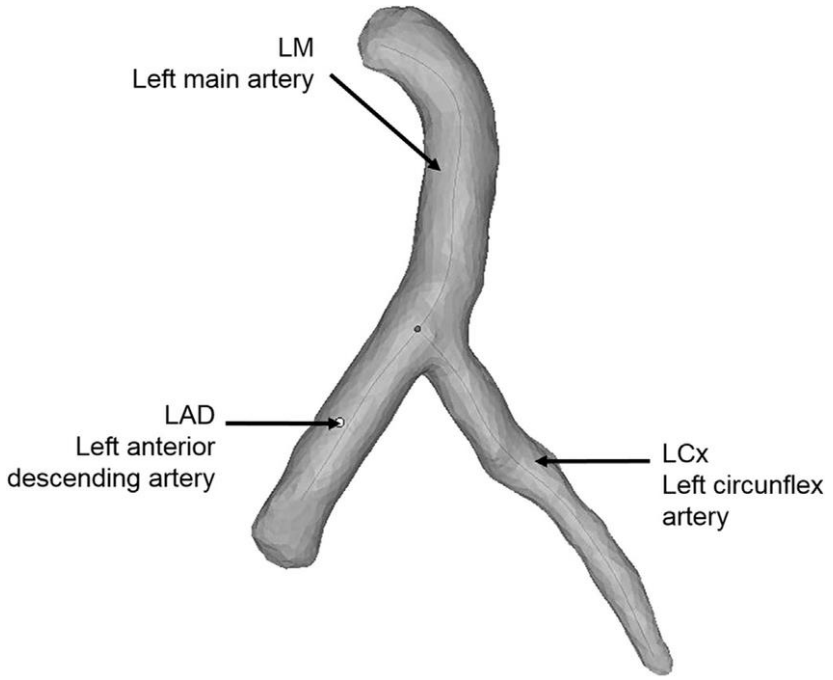
CORONARY ATHEROSCLEROSIS

- **Distribution** - Three major coronary arterial trunks can get affected, the major vessel is **anterio descending branch of the left coronary**, based on vessels it can be,

  single vessel disease

  two vessel disease

  triple vessel disease



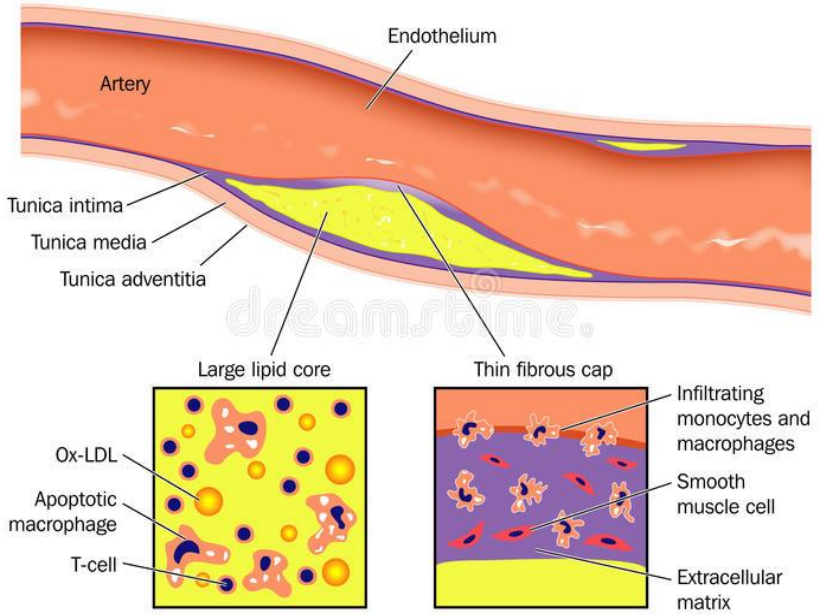
- **Location -** The area of severest involvement is about 3 to 4 cm from the coronary ostia, more often at or near the **bifurcation of the arteries**

CORONARY ATHEROSCLEROSIS

- **Fixed atherosclerotic plaques** – the atherosclerosis will be bulging into the lumen from one side

The complications like,

- calcification,
- coronary thrombosis
- ulceration
- haemorrhage
- rupture and
- aneurysm formation.

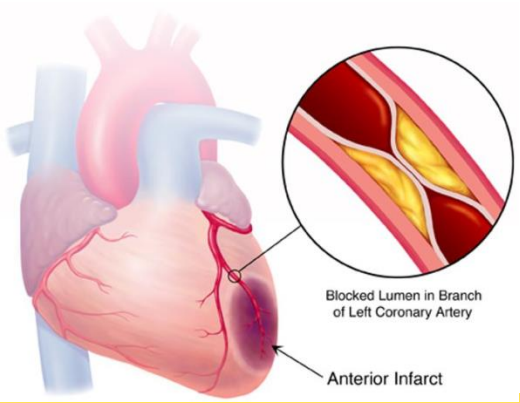


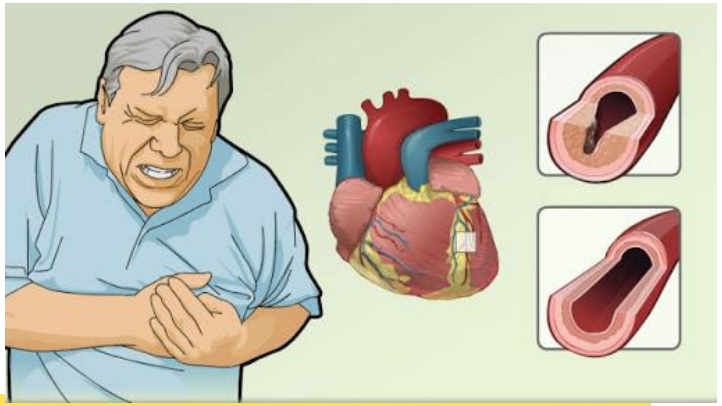
SUPERADDED CHANGES IN CORONARY ATHEROSCLEROSIS

The attacks of ***acute coronary syndromes,*** which include,

- **acute myocardial infarction**
- **unstable angina**
- **sudden ischaemic death**

These are precipitated by certain changes superimposed on a pre-existing fixed coronary atheromatous plaque





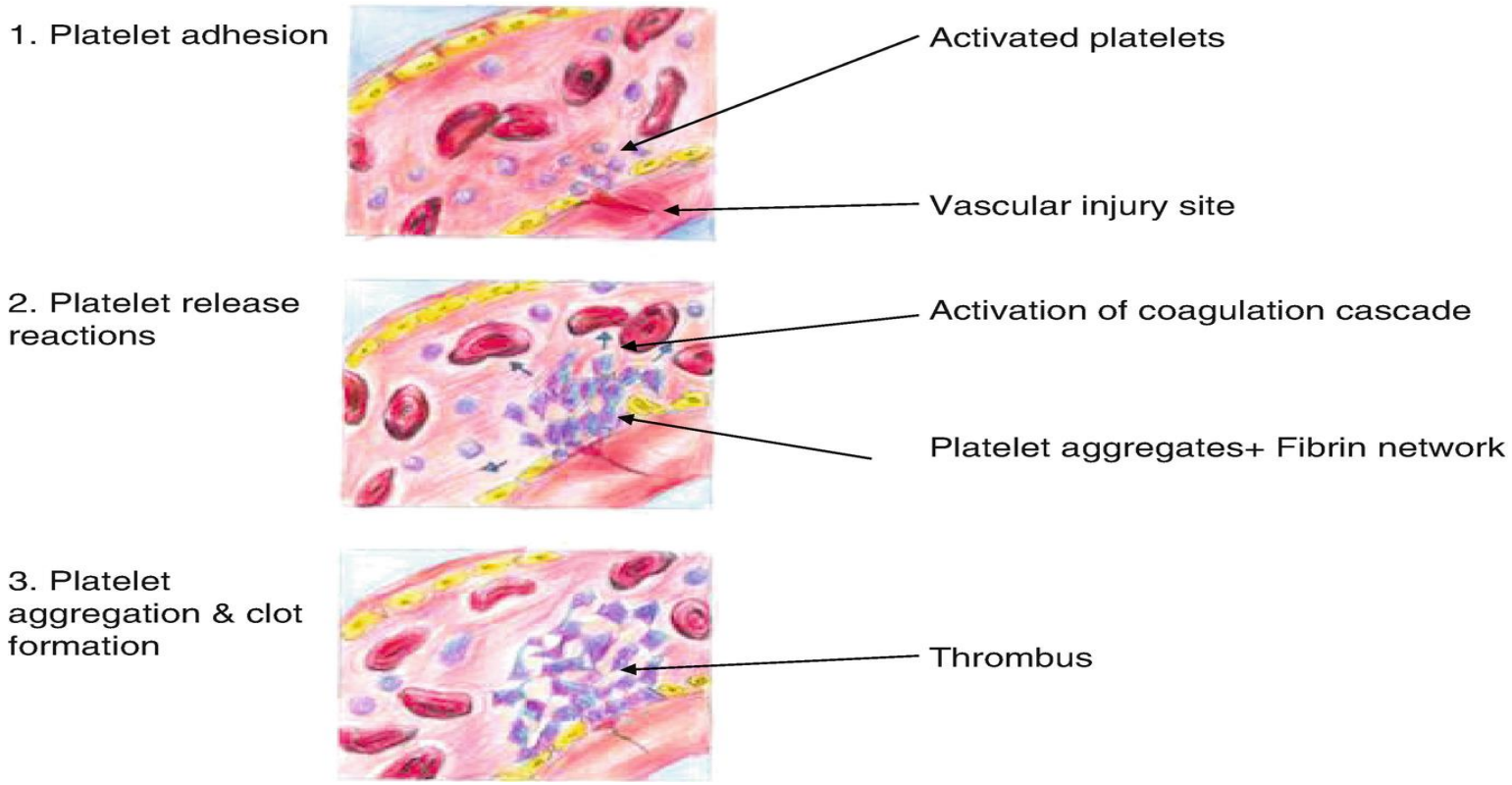
SUPERADDED CHANGES IN CORONARY ATHEROSCLEROSIS

- **Acute changes in chronic atheromatous plaque -** plaque haemorrhage,fissuring, or ulceration that results in thrombosis and embolisation of atheromatous debris.

- **Coronary artery thrombosis -** The initiation of thrombus occurs due to surface ulceration of fixed chronic atheromatous plaque, ultimately causing complete luminal occlusion.
- The lipid core of plaque, in particular, is highly **thrombogenic.**

SUPERADDED CHANGES IN CORONARY ATHEROSCLEROSIS

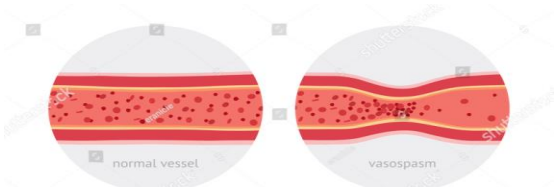
Local platelet aggregation and coronary artery spasm - The aggregated platelets release vasospasmic mediators such as thromboxane A2 which may probably be responsible for coronary vasospasm

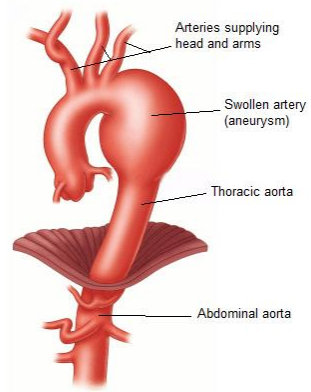


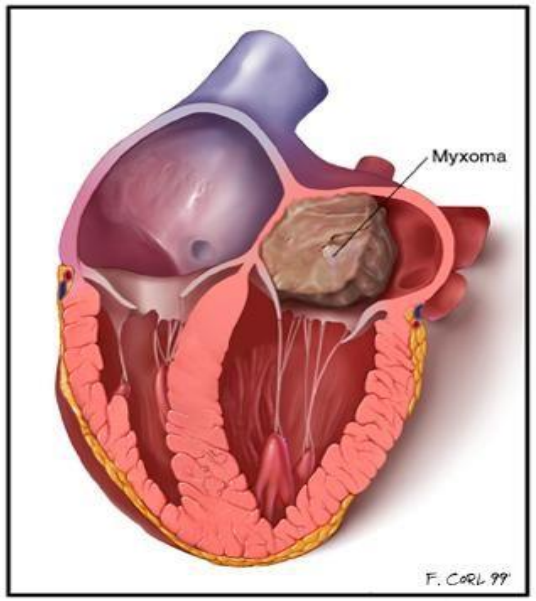
NON-ATHEROSCLEROTIC CAUSES

Several other coronary lesions may cause IHD in less than 10% of cases

- **Vasospasm**
- **Stenosis of coronary ostia -** syphilitic aortitis
- **Arteritis**
- **Embolism**
- **Thrombotic diseases -** hypercoagulability of the blood such as in shock, polycythaemia vera, sickle cell anaemia
- **Trauma**
- **Aneurysms -** congenital, mycotic and syphilitic aneurysms
- **Compression – through** tumour





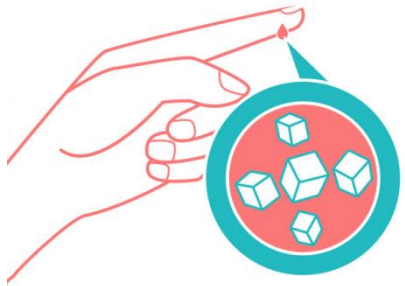


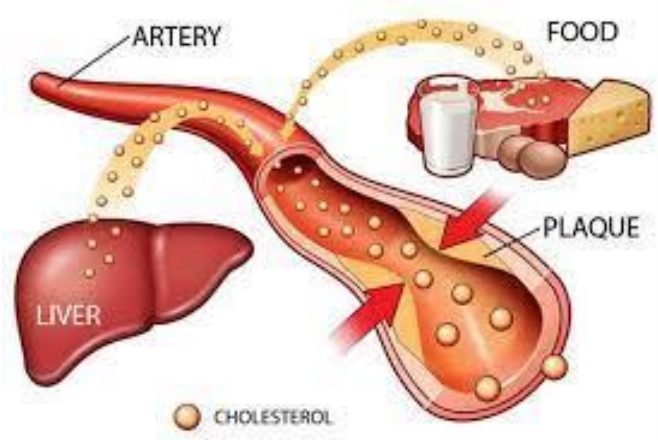
MAJOR RISK FACTORS

Major risk factors modifiable by life style and/or therapy:

This includes major risk factors which can be controlled by modifying life style and/or by pharmacotherapy and includes:

- **Dyslipidaemias**
- **Hypertension**
- **diabetes mellitus and smoking.**





MAJOR RISK FACTORS

Dyslipidaemias

- Hypercholesterolaemia has directly proportionate relationship with atherosclerosis and IHD.
- The major classes of lipoprotein particles are *chylomicrons, very-low density lipoproteins (VLDL), low-density lipoproteins (LDL)*, and *high-density lipoproteins (HDL).*

LIPID PROFILE TheNoMansLand.Net			
	DESIRABLE	BORDERLINE	HIGH RISK
CHOLESTEROL	<200mg/dl	200-239 mg/dl	240 mg/dl
TRIGLYCERIDES	<150mg/dl	150-199 mg/dl	200-499 mg/dl
HDL	60mg/dl	35-45 mg/dl	<35 mg/dl
LDL	60-130mg/dl	130-159 mg/dl	160-189 mg/dl
Cholesterol/HDL Ratio	4.0	5.0	6.0

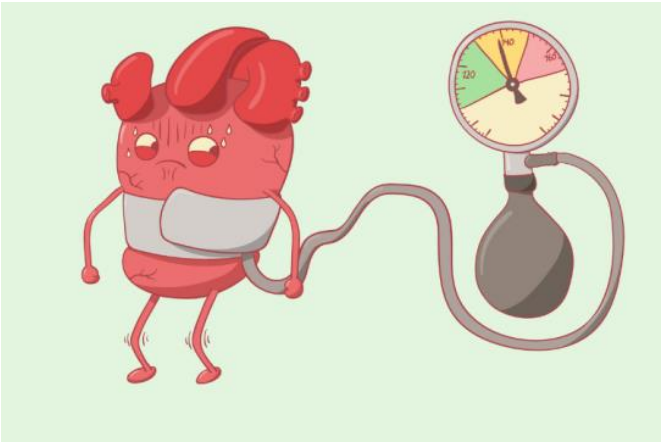
MAJOR RISK FACTORS

Hypertension

- Hypertension doubles the risk of all forms of cardiovascular disease.

- It acts probably by mechanical injury to the arterial wall due to increased blood pressure.

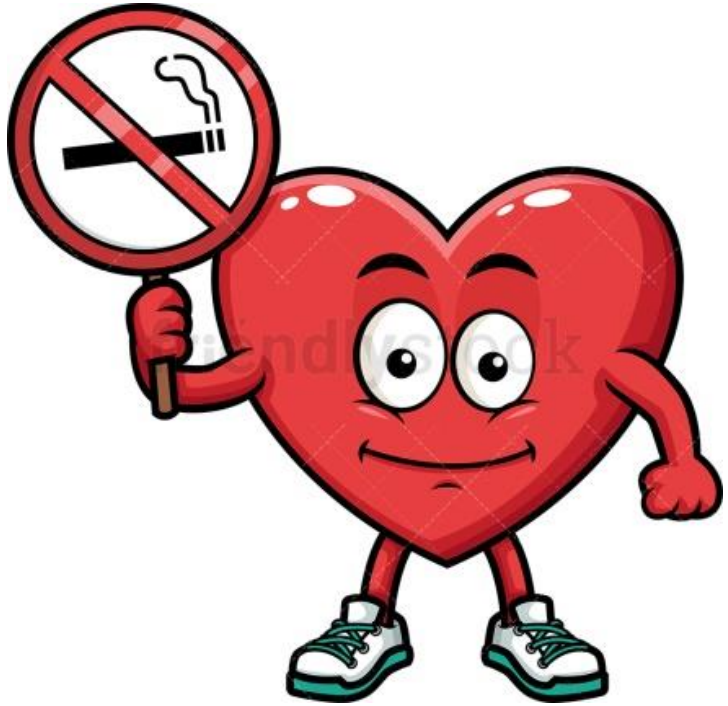
- Elevation of systolic pressure of over 160 mmHg or a diastolic pressure of over 95 mmHg is associated with five times higher risk of developing IHD



MAJOR RISK FACTORS

Smoking:

- The extent and severity of atherosclerosism are much greater in smokers than in non-smokers.
- Cigarette smoking is associated with higher risk of atherosclerotic IHD and sudden cardiac death.
- Men who smoke a pack of cigarettes a day are 3-5 times more likely to die of IHD than non-smokers.



MAJOR RISK FACTORS

Diabetes mellitus:

Type 2 diabetes mellitus characterised by metabolic (insulin resistance) syndrome and abnormal lipid profile termed **'diabetic dyslipidaemia'** is common and heightens the risk of cardiovascular disease

CONSTITUTIONAL RISK FACTORS

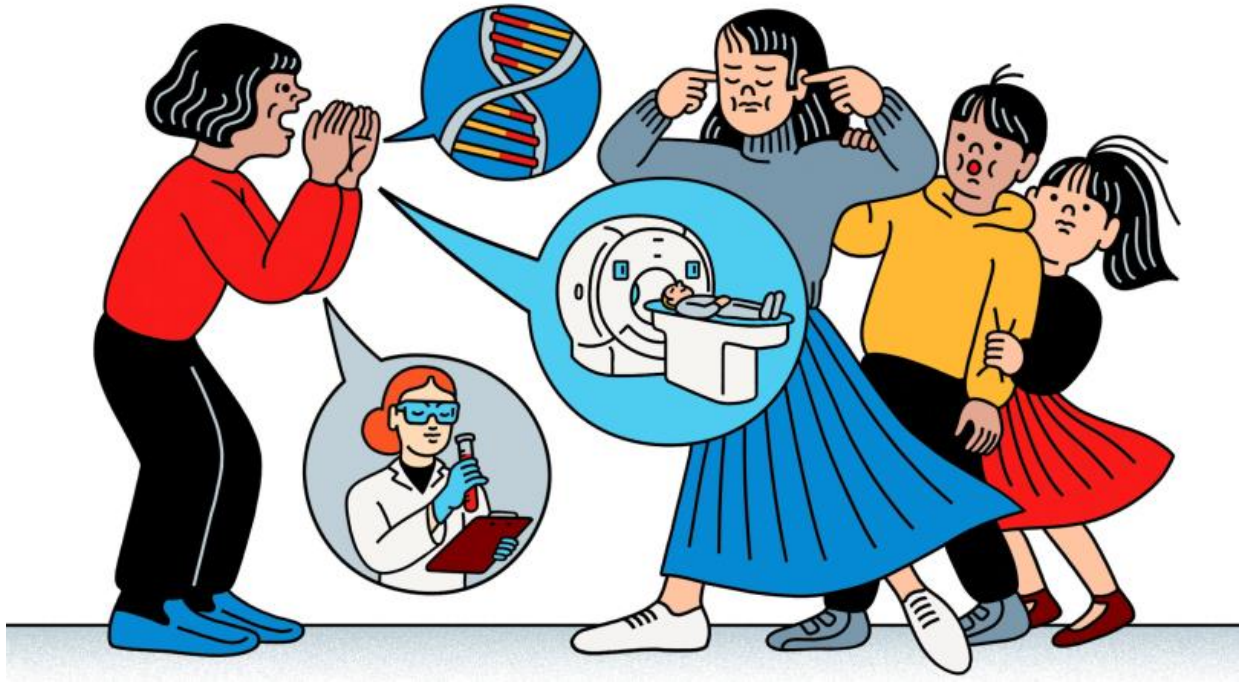
These are non-modifiable major risk factors that include:

- **Increasing age -** Fully-developed atheromatous plaques usually appear in the 4th decade and beyond

- **Male sex -** The lower incidence of IHD in women, especially in premenopausal age, is probably due to high levels of oestrogen and high-density lipoproteins, both of which have **anti-atherogenic influence**

CONSTITUTIONAL RISK FACTORS

- **Genetic abnormalities -** Hereditary genetic derangements of lipoprotein
- **Familial predisposition –** DM, HTN, hyperlipoproteinaemia
- **Racial predisposition -** Racial differences too exist; Blacks have generally less severe atherosclerosis than Whites.



NON-TRADITIONAL EMERGING RISK FACTORS

This includes a host of factors whose role in atherosclerosis is minimal, and in some cases, even uncertain.

- *environmental influences* - Higher incidence of atherosclerosis in developed countries
- *Obesity*
- Use of *exogenous hormones* - oral contraceptives
- *Physical inactivity*
- *Stressful life style* - 'type A' behaviour pattern
- consumption of *alcohol*
- *Prothrombotic factors*
- *elevated C reactive protein* - an acute phase reactant
- Role of *infections* - viruses such as herpesvirus and cytomegalovirus

EFFECTS OF MYOCARDIAL ISCHEMIA

The range of changes and clinical features may vary from an asymptomatic state at one extreme to immediate mortality at another

- **Asymptomatic state**
- **Angina pectoris (AP)**
- **Acute myocardial infarction (MI)**
- **Chronic ischaemic heart disease (CIHD)/ Ischaemic**
- **cardiomyopathy/ Myocardial fibrosis**
- **Sudden cardiac death**

PROGRESSION OF DISEASE

Nomenclature and main histology	Sequences in progression	Main growth mechanism	Earliest onset	Clinical correlation
Type I (initial) lesion Isolated macrophage foam cells	I	Growth mainly by lipid accumulation	From first decade	Clinically silent
Type II (fatty streak) lesion Mainly intracellular lipid accumulation	II			
Type III (intermediate) lesion Type II changes and small extracellular lipid pools	III		From third decade	
Type IV (atheroma) lesion Type II changes and core of extracellular lipid	IV			Clinically silent or overt
Type V (fibroatheroma) lesion Lipid core and fibrotic layer, or multiple lipid cores and fibrotic layers, or mainly calcific, or mainly fibrotic	V	Accelerated smooth muscle and collagen increase	From fourth decade	
Type VI (complicated) lesion Surface defect, haematoma-haemorrhage, thrombus	VI	Thrombosis, haematoma		

PRIMARY PREVENTION

- Strategies taken before onset of disease in high risk individual.
- Two complementary strategies
- **Population strategies**
  - modify the risk factors of the whole population
  - through diet and lifestyle advice
  - For ex: Public restricting of smoking

- **Targeted strategies**
  - identify and treat high risk individuals who usually have a combination of risk factors

SECONDARY PREVENTION

- Already have evidence of atheromatous vascular disease are at high risk of future cardiovascular events.
- Various secondary measures in this case
  - energetic correction of modifiable risk factors,
    Smoking
    Hypertension
    Hypercholesterolaemia,
  - Statin therapy irrespective of their serum cholesterol concentration
  - Target BP of ≤ 140/85 mmHg
  - Aspirin and ACE inhibitors
  - Beta-blockers: h/o MI or heart failure.

THANK YOU

Reference:

Text book of Pathology, Harsh Mohan