

# **ATHEROSCLEROSIS**

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# Vascular Disorders

## 1. Narrowing / obstruction of lumen

Atherosclerosis

Thrombosis

## 2. Weakening of wall

# Histology

- Arteries --
- a) Large / Elastic
  - b) Medium / Muscular
  - c) Small
  - d) Arterioles
  - e) Capillaries

# Histology

**Tunica Intima** – lined by endothelium

**Internal elastic lamina** – separating intima from media

**Tunica Media** – smooth muscle cells & elastic fibres

**External elastic lamina** - separating media from adventitia

**Tunica adventitia** – layers of connective tissue in which  
nerve fibres & vasa vasorum are dispersed

## Blood vessel Histology

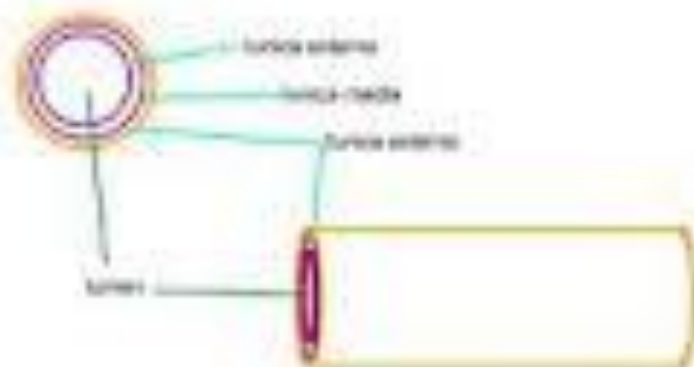
endothelium



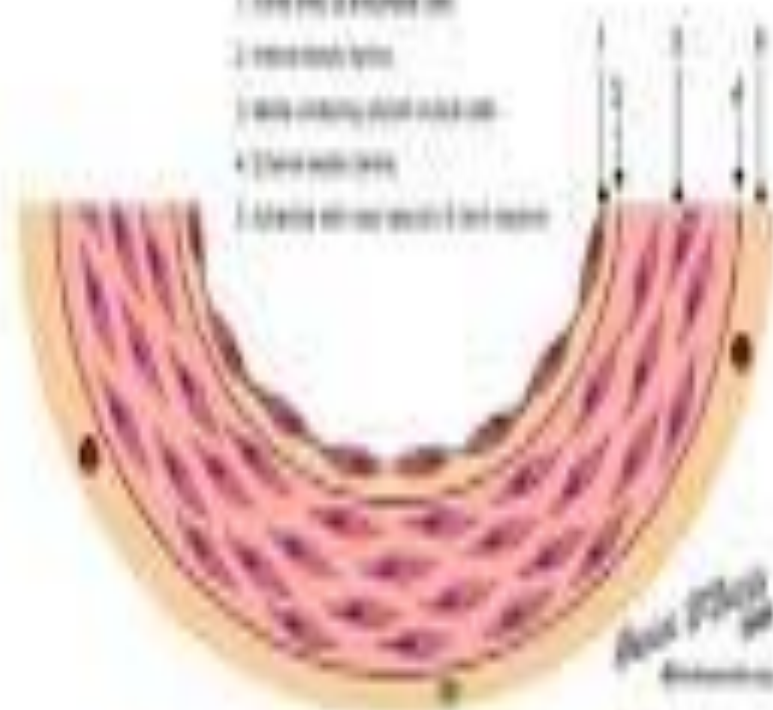
smooth muscle



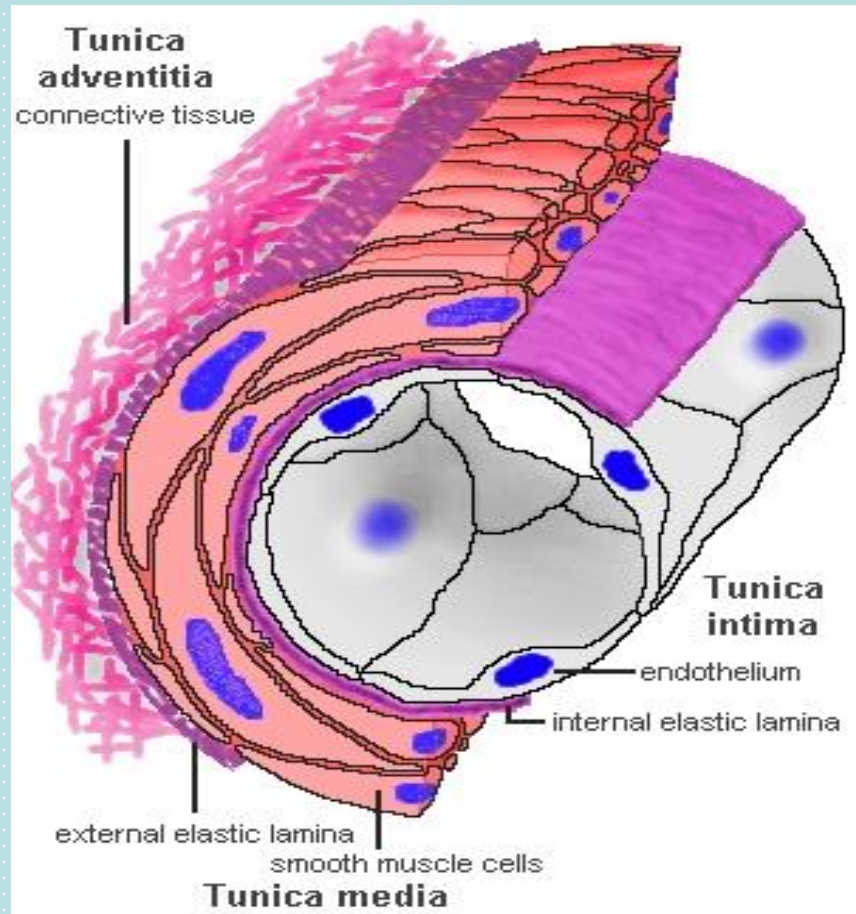
elastic connective tissue



1. Tunica interna
2. Tunica media
3. Tunica externa
4. Elastic connective tissue
5. Elastic lamina



1  
2  
3

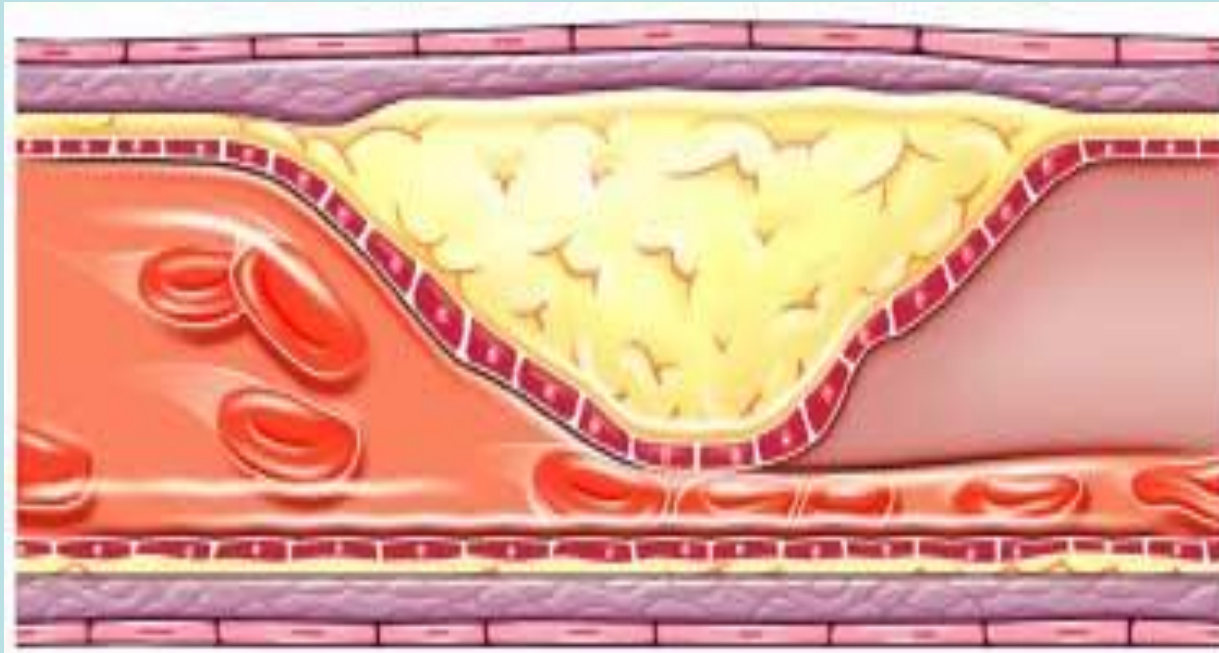


# Arteriosclerosis

- Thickening & hardening of arteries
  - Hypertensive arteriosclerosis / Arteriolosclerosis
  - Monckeberg's arteriosclerosis
  - Atherosclerosis

# Atherosclerosis

- ❖ **Definition** – Intimal lesions,
  - protrude into lumen
  - Obstruction
  - Weaken media & undergo complications





# Atherosclerosis

Athero – soft lipid rich material in the centre

Sclerosis – connective tissue

Elastic & large & medium or muscular arteries

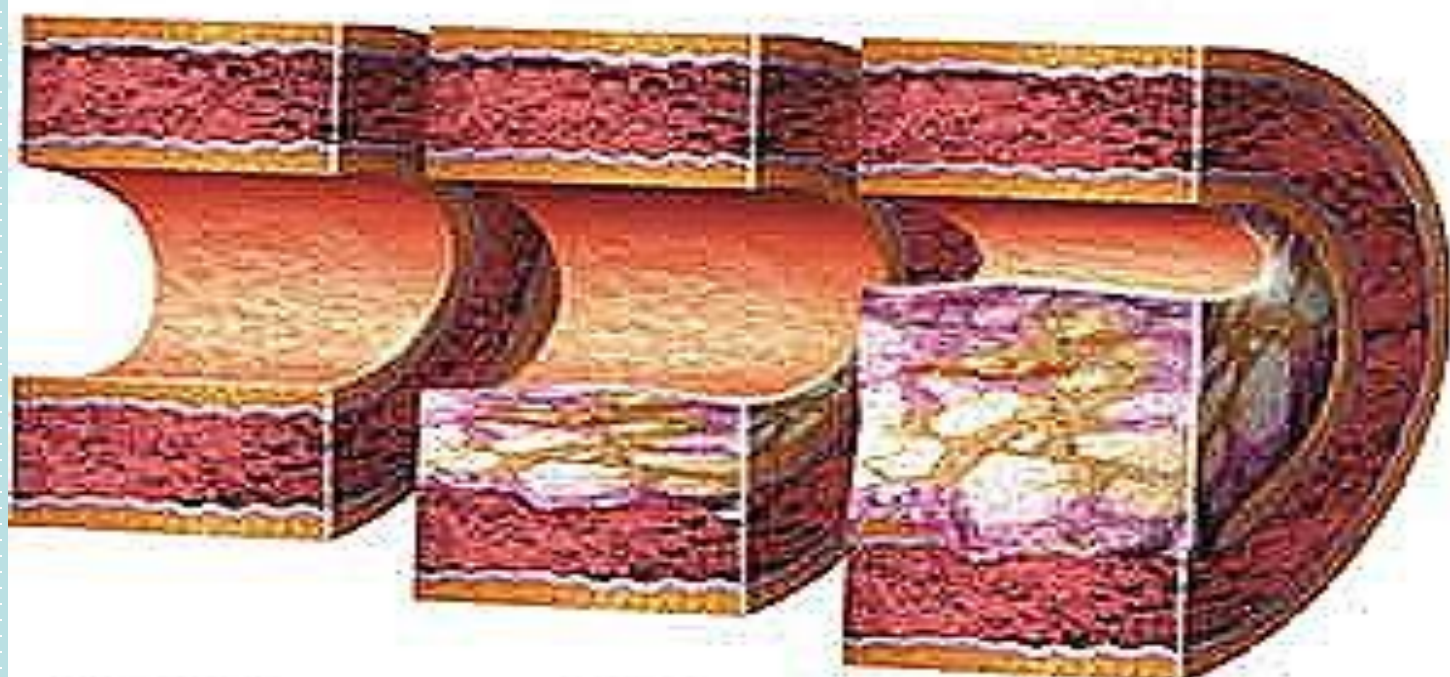
Begins in childhood but symptoms not evident till middle age or later when the lesions precipitate organ injury.

Arteries supplying heart, brain, kidneys intestine & lower extremities.

Clinical syndromes –

Myocardial infarct, cerebral infarct, peripheral vascular disease, aneurysmal dilatation, mesenteric occlusion, chronic IHD

# Atherosclerosis

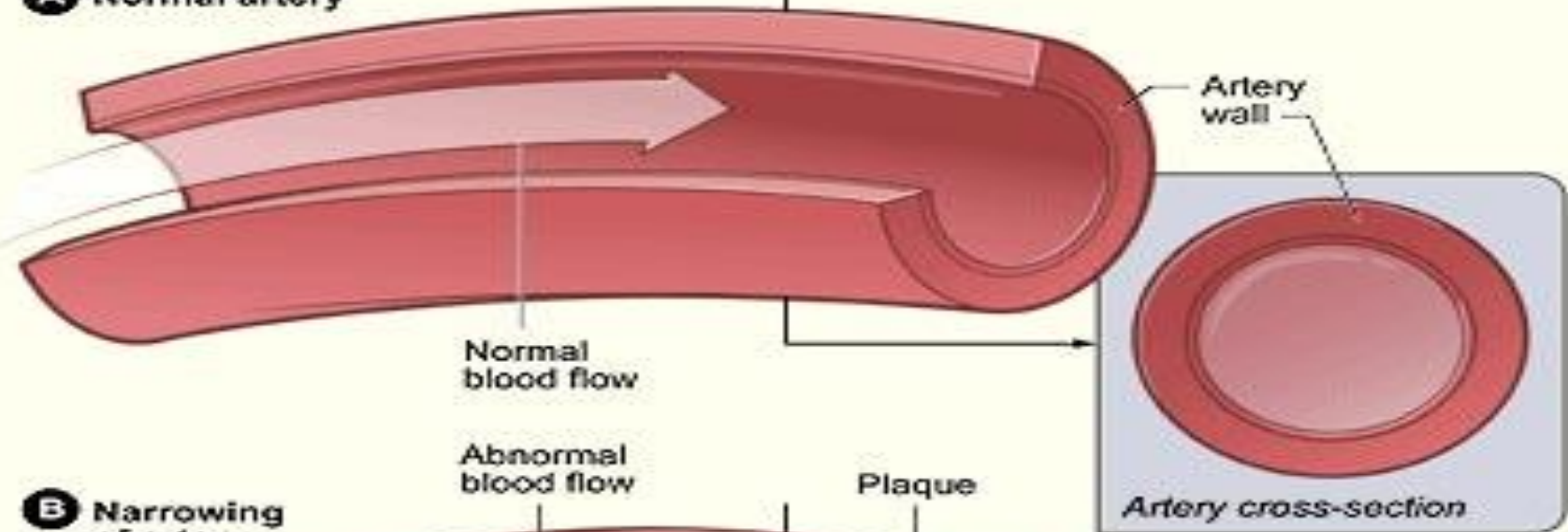


**Normal  
Artery**

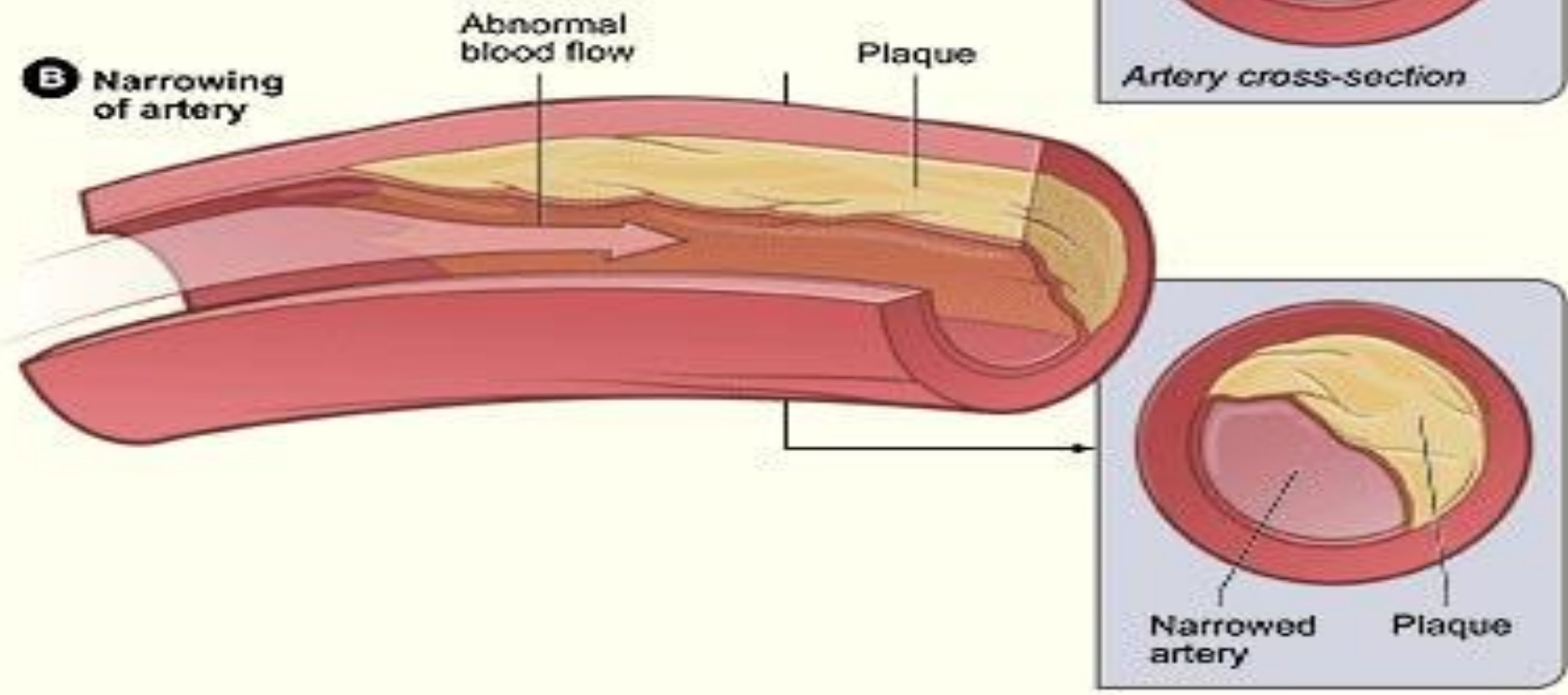
**Mild  
Atherosclerosis**

**Severe  
Atherosclerosis**

**A** Normal artery



**B** Narrowing of artery



## ❖ **Epidemiology & Risk factors**

### **I] Major Constitutional factors**

- a. Age
- b. Sex
- c. Genetic factors
- d. Familial & Racial factors

### **II] Major acquired/ reversible factors**

- a. Hyperlipidaemia 140-200mg/dl
- b. Hypertension 169/95 mm of Hg
- c. Smoking
- d. Diabetic mellitus

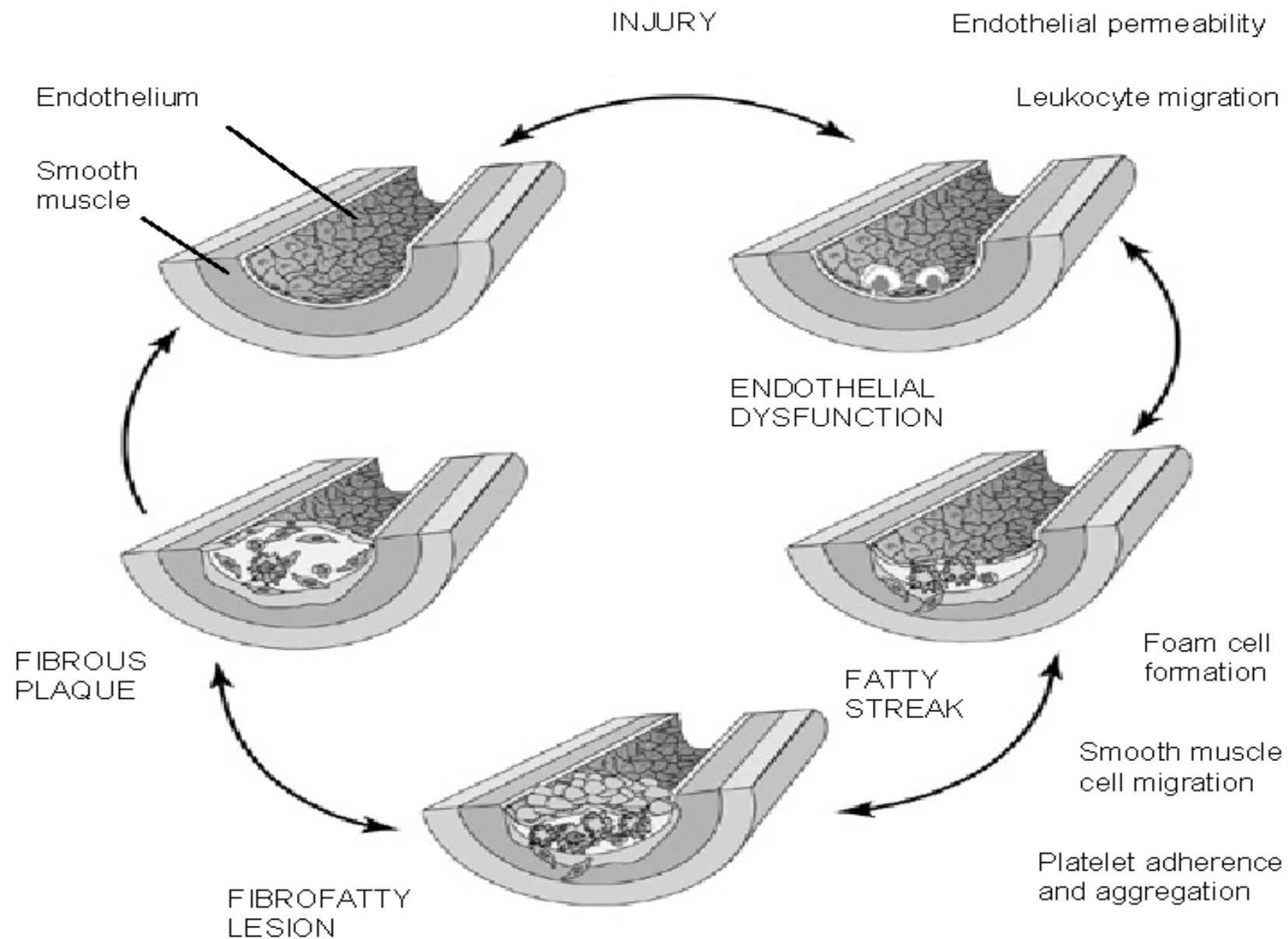
### **III] Minor risk factor**

- a. Obesity
- b. Physical inactivity
- c. Stress (type A personality)
- d. Alcohol
- e. Homocysteinuria

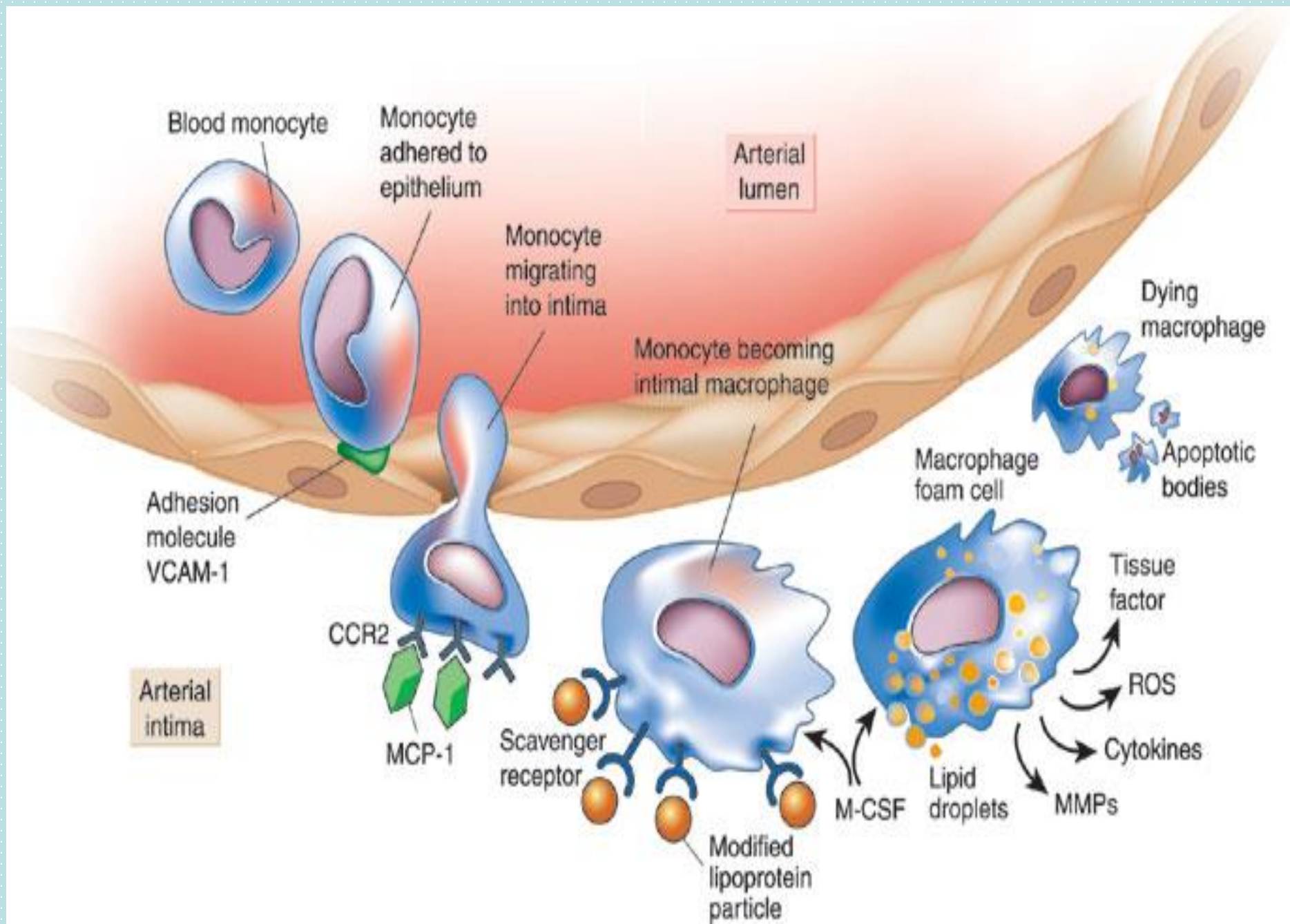
## ❖ Pathogenesis:

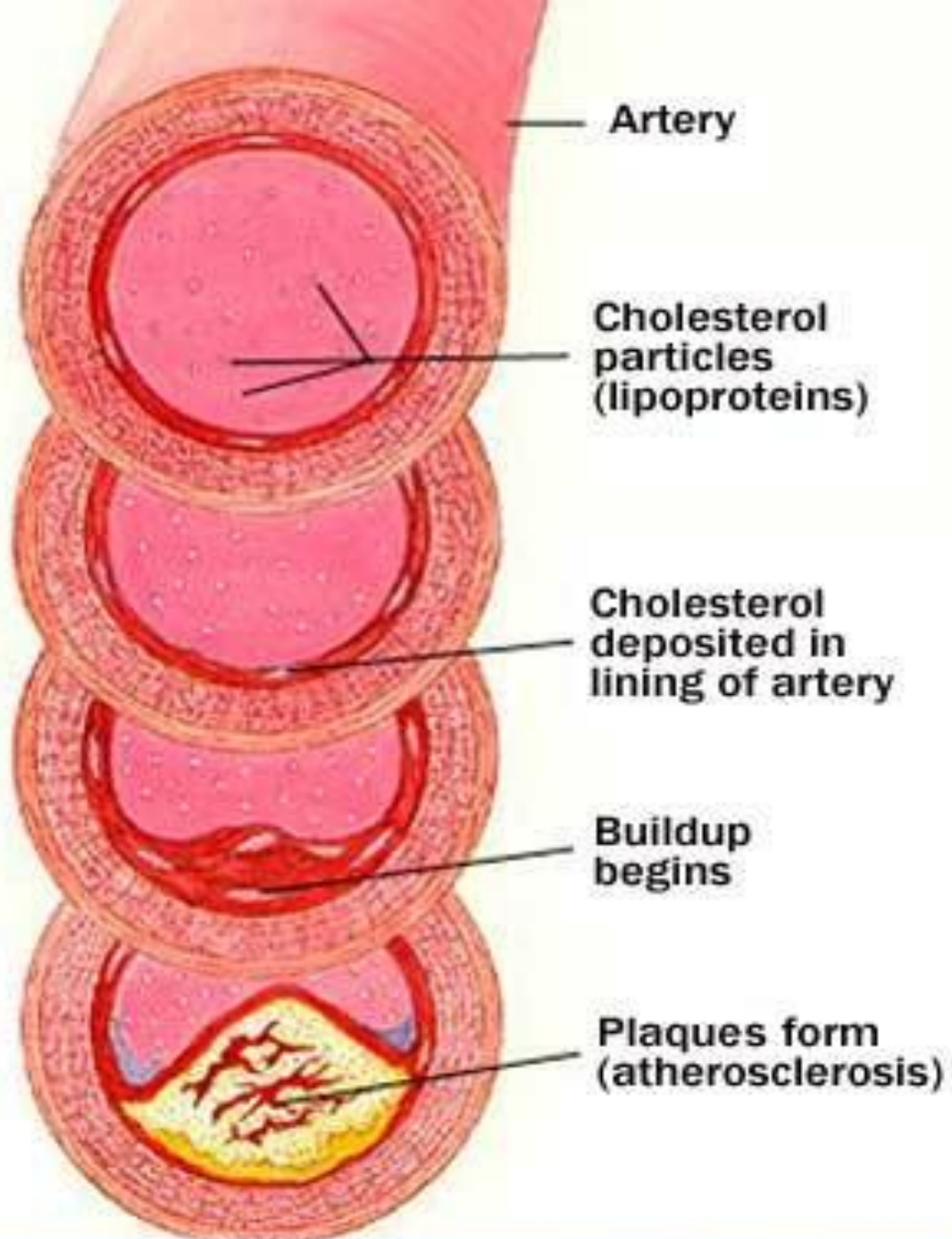
### **Response to injury hypothesis – Chronic inflammatory response of arterial wall initiated by injury to endothelium**

- Development of focal region of endothelial injury
- Insudation of lipoproteins into the vessel wall
- Adhesion of blood monocytes to the endothelium (foam cells)
- Adhesion of platelets
- Release of factors from activated platelets
- Proliferation of smooth muscle cells & elaboration of extra cellular matrix & migration of SMC into intima
- Enhanced accumulation of Lipids both intra & extra cellularly











❖ **Pathological changes:**

❖ **Fatty streaks** – Multiple, yellow, flat spots (<1 mm)

❖ **M/E** – Lipid containing foam cells with lymphocytes & extracellular lipid

❖ **Atheromatous plaque [Atheroma]**

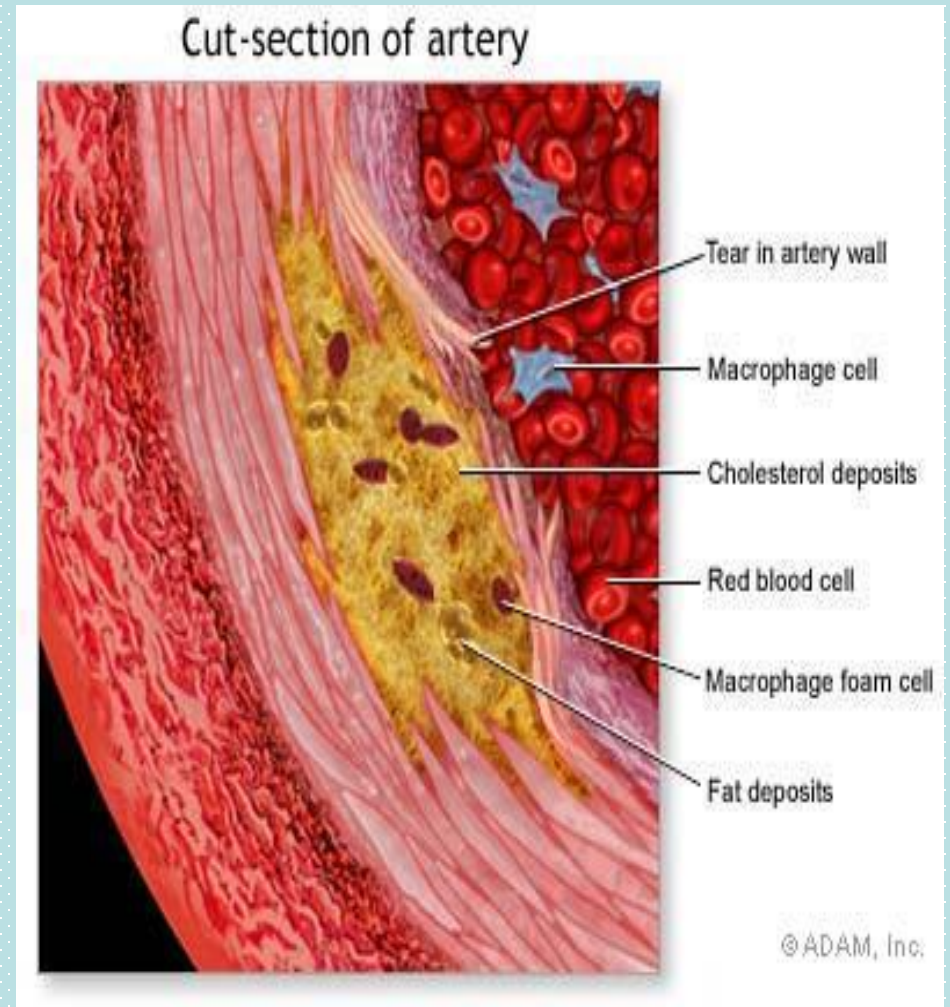
❖ **Gross** – White to whitish yellow 0.3 – 1.5 cm

❖ **C/S** – fibrous cup

– firm & white & yellow & soft in the centre

• **Sites** – Abdominal aorta, coronaries, popliteal arteries, descending thoracic aorta, internal carotid & vessels of circle of willis, around ostia of major branches.

1. Cells including smooth muscles cells, macrophages & other leucocytes.
2. connective tissue, extra cellular matrix including collagen, elastic fibers & proteoglycans
3. intracellular & extra cellular lipid deposits



## **Superficial fibrous cap:-**

Smooth muscle cells with a few leucocytes & relatively dense connective tissue.

## **Cellular area beneath to the side of cap (shoulder)**

Mixture of macrophages, smooth muscle cells & T Lymphocytes

## **Deeper necrotic core:-**

Disorganized mass of lipid material, cholesterol clefts,

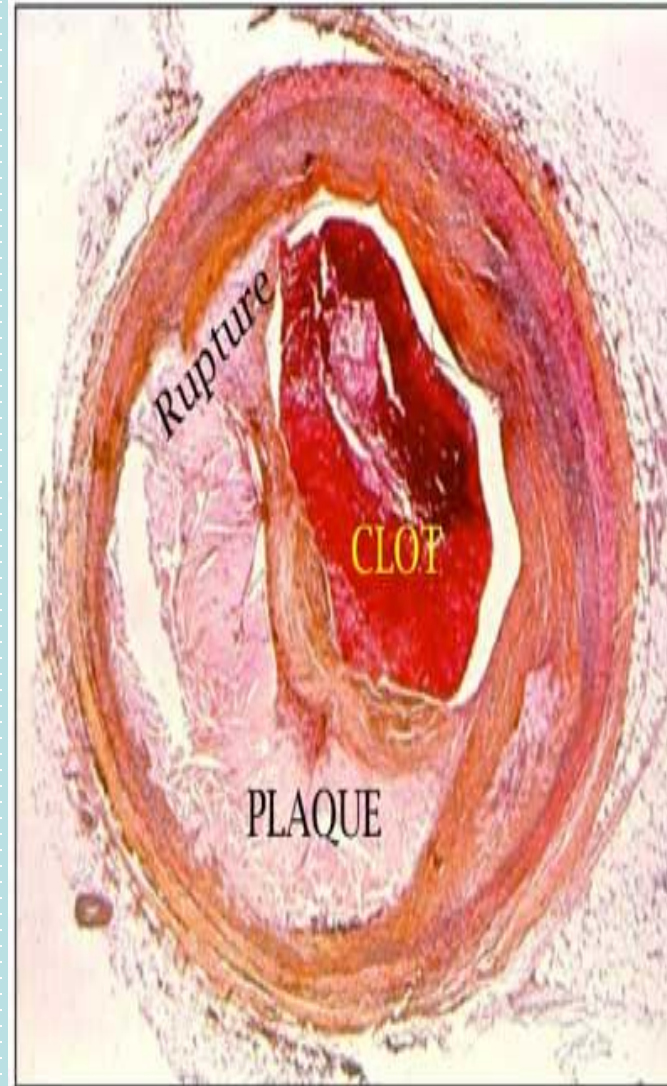
Cellular debris, lipid laden, foam cells.

**Periphery:** - e/o neovascularization

## Complicated plaque

- a. Calcification
- b. Ulceration
- c. Haemorrhage
- d. Superimposed thrombosis
- e. Aneurismal dilatation

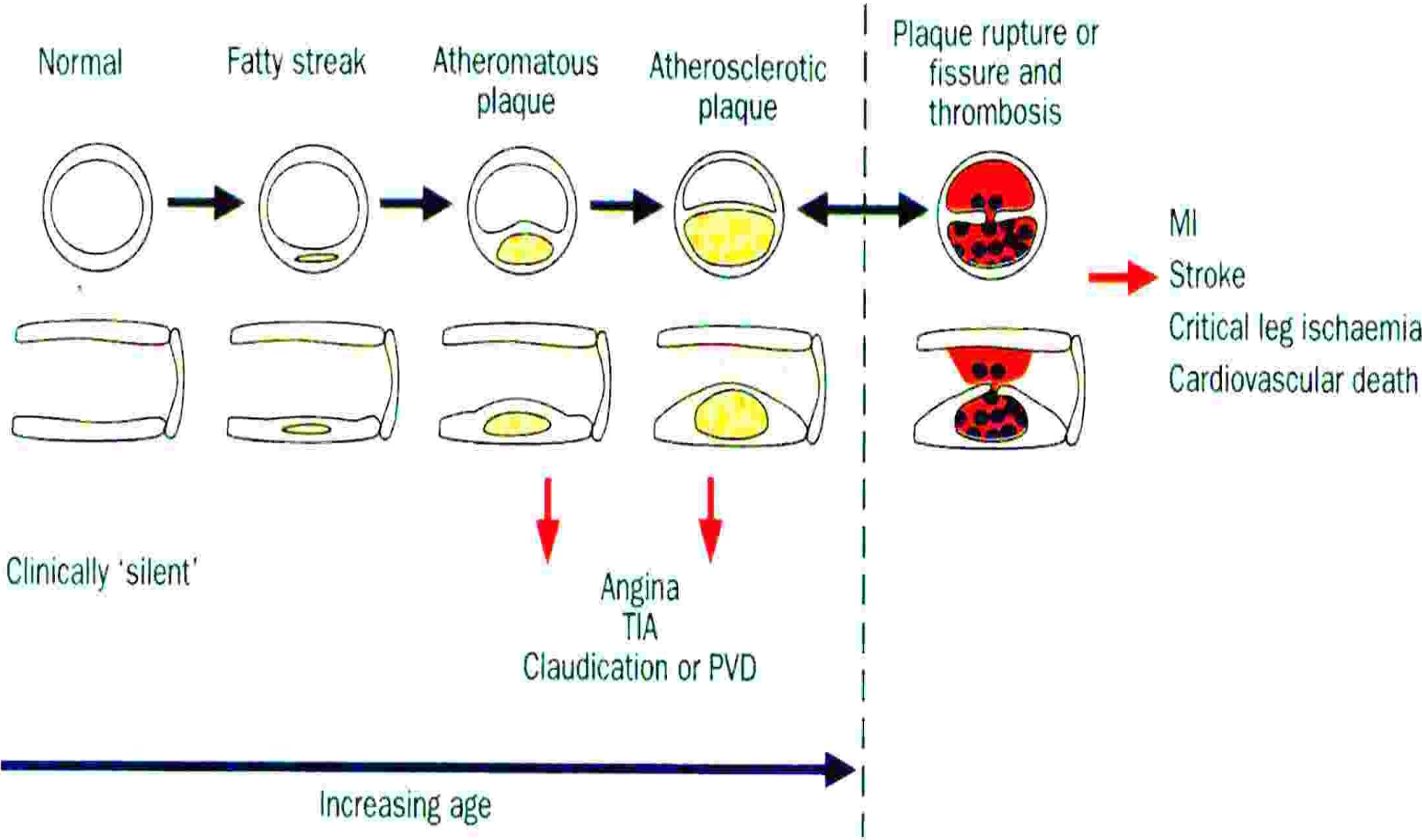
## The vulnerable atherosclerotic plaque



- large lipid core
- thin fibrous cap
- rich in macrophages
- increased MMPs
- poor in smooth muscle cells
- low-grade stenosis

❖ C/F -

1. **Slow luminal narrowing causing ischemia of atrophy**
2. **Sudden luminal occlusion causing infarcts of necrosis**
3. **Propagation of plaque by formation of thrombi & emboli**
4. **Formation of aneurismal dilatation & eventual rupture**





↓ ENDOTHELIAL DYSFUNCTION ↓

**NOMANCLATURE AND MAIN HISTOLOGY**

**SEQUENCES IN PROGRESSION OF ATHEROSCLEROSIS**

**EARLIEST ONSET**

**MAIN GROWTH MECHANISM**

**CLINICAL COLLERLATION**

**Initial lesion**

- histologically "normal"
- macrophage infiltration
- isolated foam cells

**Fatty streak**

mainly intracellular lipid accumulation

**Intermediate lesion**

- intracellular lipid accumulation
- small extracellular lipid pools

**Atheroma**

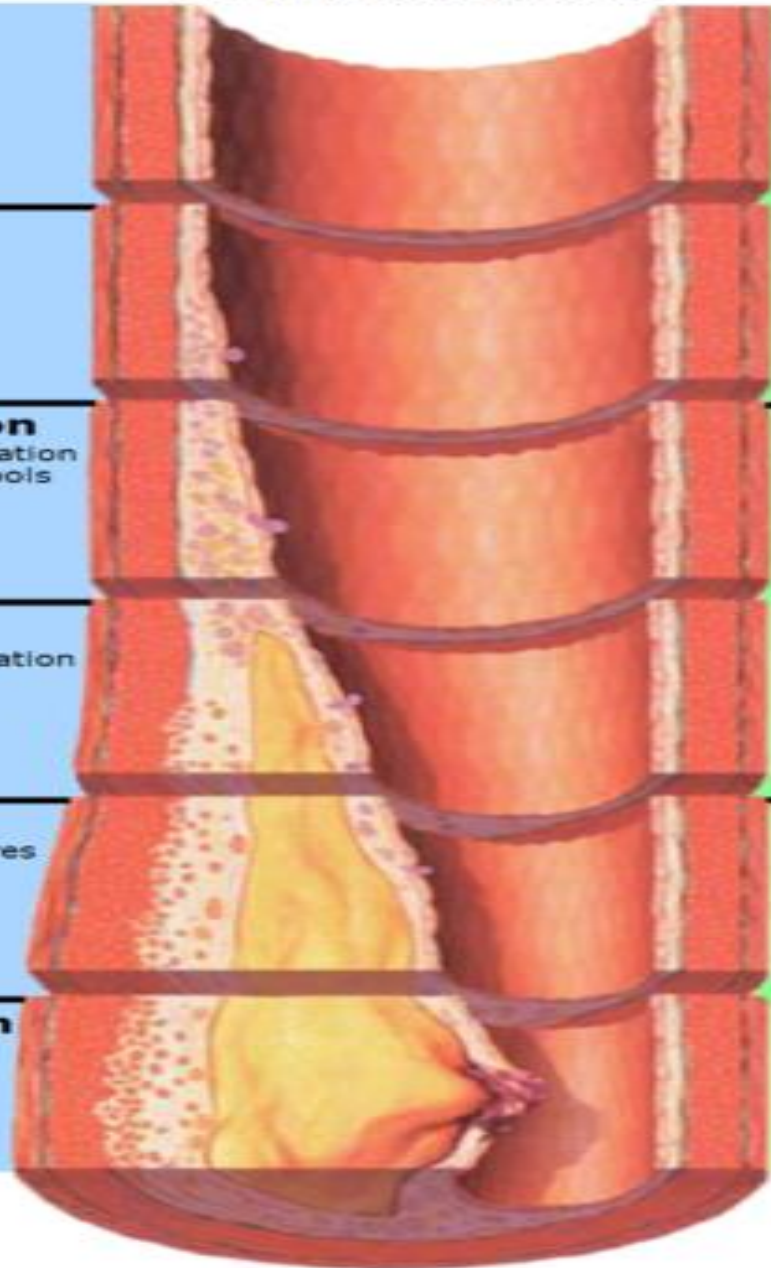
- intracellular lipid accumulation
- core of extracellular lipid

**Fibroatheroma**

- single or multiple lipid cores
- fibrotic/calcific layers

**Complicated lesion**

- surface defect
- hematoma-hemorrhage
- thrombosis



from first decade

from third decade

from fourth decade

growth mainly by lipid addition

increased smooth muscle and collagen increase

thrombosis and/or hematoma

clinically silent

clinically silent or overt

