

# Cardiovascular system block

## Pathology team 431



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## Second lecture

### Atherosclerosis

#### Normal vessels:

There are many sizes of blood vessels:

#### Large (elastic) arteries

- **Aorta, common carotid, iliac**
- Lots of elastic fibers
- Can be pulsed
- This kind mostly affected by Atherosclerosis

#### • **Medium (muscular) arteries**

- coronary, renal arteries
- mostly smooth muscle cells
- Also this kind is affected

#### • **Small arteries/arterioles**

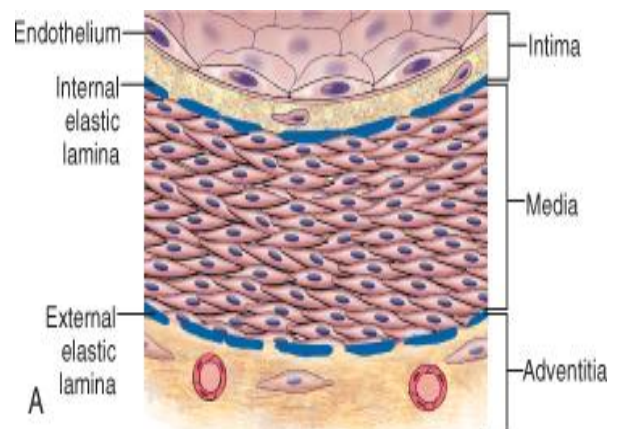
- all smooth muscle cells
- blood pressure controlled here
- This kind affected by hypertension

#### Normal histology: consist of 3 main layers

1-**Intima:** consist of *endothelial monolayer* overlying a thin *extracellular matrix* (collagen, elastin and glycosaminoglycan) demarcated from media by dens elastic membrane called *internal elastic lamina*

2-**Media:** composed mainly by smooth muscle and extracellular matrix (collagen, elastin and glycosaminoglycan) surrounded by connective tissue, nerve fibers and small vessels to supply it from the adventitia .the external elastic lamina define the transition between the media and adventitia.

3-**Adventitia:** termed “vasa vasorum” “vessels of the vessels” supply the outer layer of media.



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Fatty streak: the pattern of underlying

Process which is lipid filling foam (Macrophage)

Fatty streaks can appear in the aortas of infants younger than 1 year and are present in virtually all children older than 10 years.

## ARTERIOSCLEROSIS:

Is hardening of the arteries with loss of elasticity

3 types:

1. atherosclerosis: most common\*\*
2. monckeberg medial sclerosis: CALCIFICATION in the medium sized arteries that supply muscles
3. arteriolosclerosis: small arteries and arterioles (in Hypertension and DM)

This picture show very severe atherosclerosis with the distinguished plaques.

### Atherosclerosis a disease of the intima

- A type of *arteriosclerosis*  
*Some of arteriosclerosis is benign.*
- Chronic inflammatory response in the walls of arteries
- Slowly progressive.
- A build-up of fat (cholesterol) within the artery wall.
- Characterized by **intimal** lesions called: atheromas, atheromatous or fibrofatty plaques.
- Intimal thickening with intra and extra cellular lipid deposition

### Common sites by common to the least.

- *Abdominal aorta*
- *Coronaries*
- *Popliteal artery*
- The internal carotid arteries
- The vessels of the circle of Willis



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## Risk factors

Major		Lesser, Uncertain, or Nonquantitated
<b>Nonmodifiable</b>		
Increasing age		Obesity
Male gender		Physical inactivity
Family history		Stress ("type A" personality)
Genetic abnormalities		Postmenopausal estrogen deficiency
		High carbohydrate intake
<b>Potentially Controllable</b>		
Hyperlipidemia		Alcohol
Hypertension		Lipoprotein Lp(a)
Cigarette smoking		Hardened (trans)unsaturated fat intake
Diabetes		<i>Chlamydia pneumoniae</i>

Some note:

Major means that it has commonly happened.

Non modifiable: cannot be treated.

## Causes of atherosclerosis:

Response to injury initiated by endothelial dysfunction

## LDL vs HDL

- LDL cholesterol : deliver cholesterol to peripheral tissues.
- HDL, "good cholesterol": mobilizes cholesterol from developing and existing atheromas and transports it to the liver for excretion in the bile.

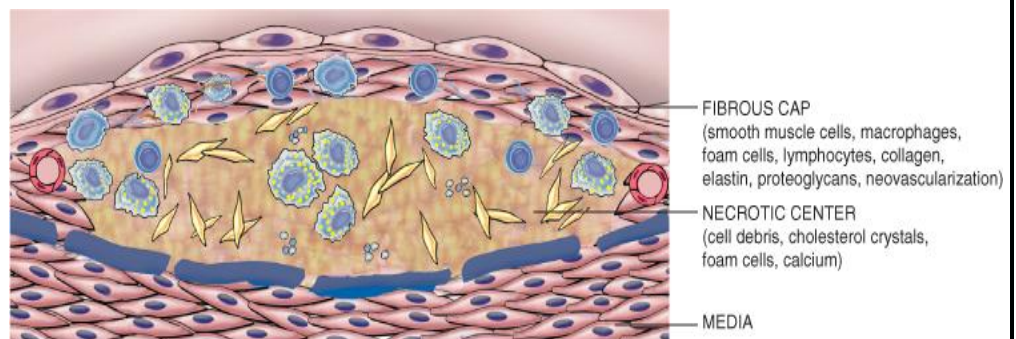
The plaques lesion: consist of

1-fibrous cap: see the pic.

(elastin+collagen+macrophage

+lymphocyte+lipid)

2-necrotic center: see the pic.



## Pathogenesis

### Phase one:

- ***Chronic Endothelial injury: most initial hypothesis.***
  - Not completely understood
  - Nevertheless, the two most important causes of endothelial dysfunction are:
    - Hemodynamic disturbances: Due to turbulent movement of blood at the point of bifurcation
    - Hypercholesterolemia
  - Inflammation is also an important contributor.
- Smooth muscle cell proliferation

### Phase two:

Endothelial dysfunction: increase permeability then leukocytes adhesion and monocytes adhesion then emigration. (CHRONIC INFLAMMATORY RESPONSE)

### Phase three:

Smooth muscle recruitment from the media to the intima and macrophage activation.

### Phase four:

Macrophage and smooth muscle engulf the lipid. Lead to appearance of fatty *streak*

### Phase five:

Smooth muscle proliferation, collagen and other extracellular matrix deposition and extracellular lipid accumulation

### Role of lipid:

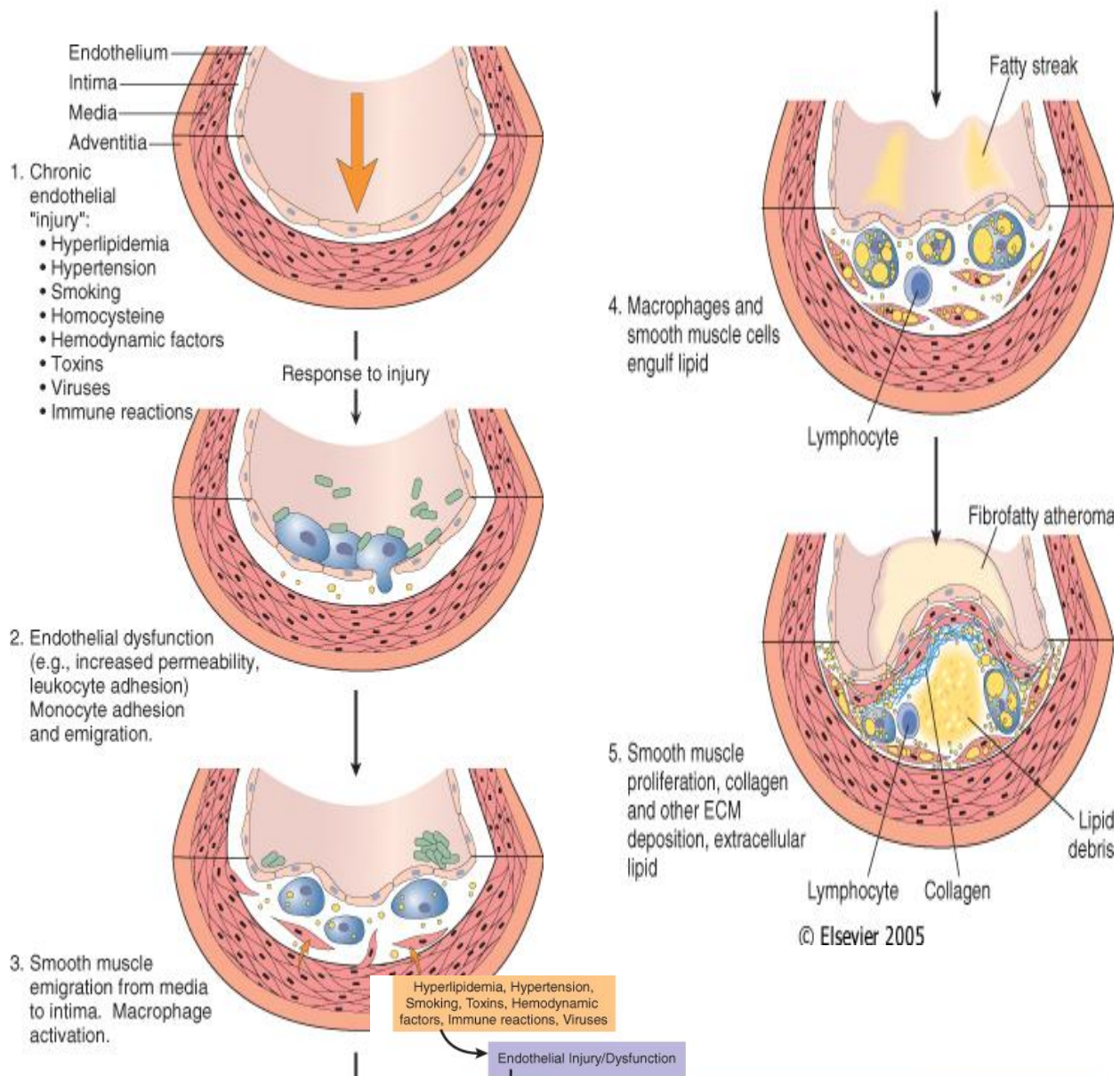
Lipid come to this lesion because it s normally circulating in the blood. But there are three main factors cause its deposition.

1-Increased LDL levels

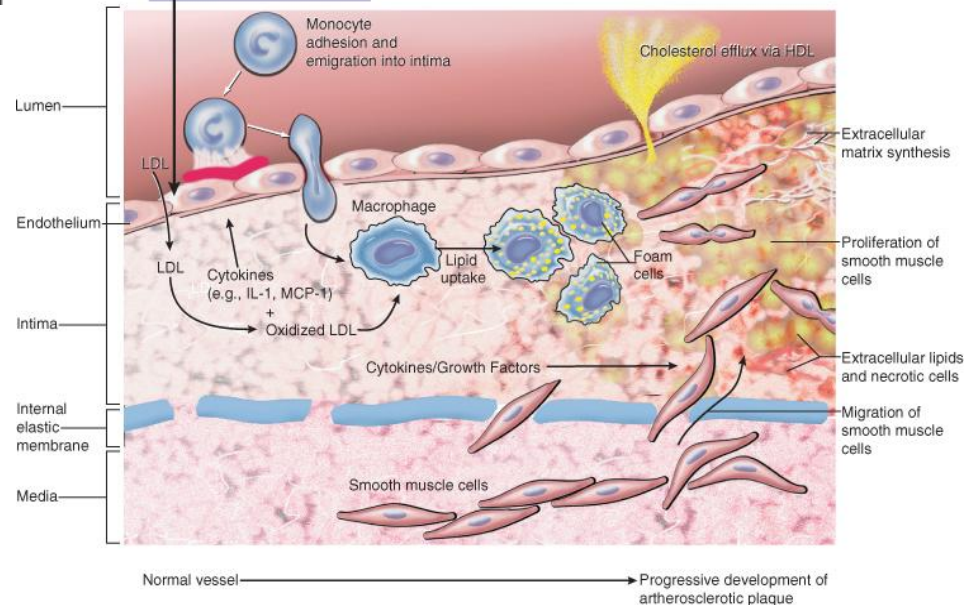
2-Decreased HDL levels

3-Increased levels of the abnormal lipoproteins.

## Some pictures will help



Note the LDL and HDL



### Consequences:

1-The plaques overgrow till it occludes 70% of the lumen which lead to **critical stenosis**.

2-If the endothelial cells affected, the intima become weak and the smooth muscle of the media stretched and this cause weakness also. This weakness causes an **aneurysm** (انبعاث) which easily can be **ruptured**.

3-**Occlusion by thrombus**: whenever there are no endothelial cells the blood clots and cause thrombus. Even if the plaques become eroded or rupture or there is hemorrhage insideit, can cause thrombus.

If the thrombus small it can move easily and go to small vessels and cause what we call **embolism**.

This consequence is clinical appearance that can be seen.

### Complication

- Myocardial infarction (heart attack)
- Cerebral infarction (stroke)
- Aortic aneurysms
- Mesentric occlusion (visceral)
- Peripheral vascular disease (gangrene of the legs)

### Summary of morphological changes:

- Neovascularization (new blood vessels)
- Calcification
- Hemorrhage
- Fissure
- Ulcer
- Thrombosis
- Medial thinning
- Cholestrolmicroemboli
- Aneurismal dilation

#### Summary

- Atherosclerosis is an inflammatory disease
- The lesion localized in the intima.
- Plaques composed of: 1-fibrous cap 2-necrotic center
- The main initial cause is endothelial dysfunction.
- The disease growth is slowly and only can be present when the consequences happened.
- Risk factor recognition can reduce the incidence and the severity.

## Questions

**What is the most likely type of vessels affected in atherosclerosis?**

- A. Elastic
- B. muscular
- C. Arterioles
- D. Capillaries

**What is the layer that affected in case of atherosclerosis?**

- A. Intima
- B. Media
- C. Adventitia
- D. External elastic lamina

**The characteristic lesion in atherosclerosis is:**

- A. Fibrofatty Plaques
- B. Nodules
- C. Granuloma
- D. Liquefactive necrosis

**Section of lung tissue obtained during an autopsy of 66-year-old women reveal numerous hemosiderin laden macrophages within the alveoli. Which of the following is the basic pathologic cause of this abnormality?**

- A. Bacterial infection.
- B. Diabetes mellitus.
- C. Heart failure.
- D. Pulmonary hypertension.

**Answers:**

- 1-B
- 2-A
- 3-A
- 4-C