# Vascular system

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LEC.1

**Normal anatomy & histology:**

Basic components of the wall of blood vessels are including:

1. Endothelial Cells ($EC_s$) & Smooth Muscle Cells ($SMC_s$).
2. Extracellular Matrix (ECM).

These components are arranged into three layers: (from lumen to outside)

1. Intima: consist of single layer of endothelial cells.
2. Media: consist of smooth muscle cells.

**Important notes:**

I. In small vessels & veins, $O_2$ & nutrition of their smooth muscles in media is by diffusion from lumen of vessels.

II. In large & medium size vessels, $O_2$ & nutrition are supplying by small blood vessels within the wall of vessels themselves (Vasa Vasorum) in the outer media & adventitia.

3. Adventitia: consist of connective tissue, Vasa Vasorum & nerve fibers.

**According to their size & structure, arteries can be classified into the followings:**

1. Large, elastic arteries:

Example: AORTA & ITS BANCHES (like subclavian, common carotid, iliac & pulmonary arteries).

Have elastic fiber rich media (expand during systole & recoil during diastole).

2. Medium size arteries (muscular arteries): like coronary, renal arteries.

Their media is composed predominantly of SMC, which regulate regional blood flow & blood pressure by change the size of lumen by vasoconstriction & vasodilatation.

3. Small arteries (less than 2mm in diameter) & arterioles (20 to 100 microns).

Their media consist of smooth muscles cells, which during contraction cause changes in diameter that regulates systemic arterial blood pressure, (as much as small vessel, the higher resistance & pressure).

So the arterioles are the principals points of physiologic resistance (control the blood pressure).

# Vascular diseases:

## I. Congenital anomalies: main two anomalies are

**a. Berry aneurysm (later discussed)**

**b. Arteriovenous fistula:**

## Ateriovenous fistula

They are rare, small abnormal communications between arteries & veins.

Causes:

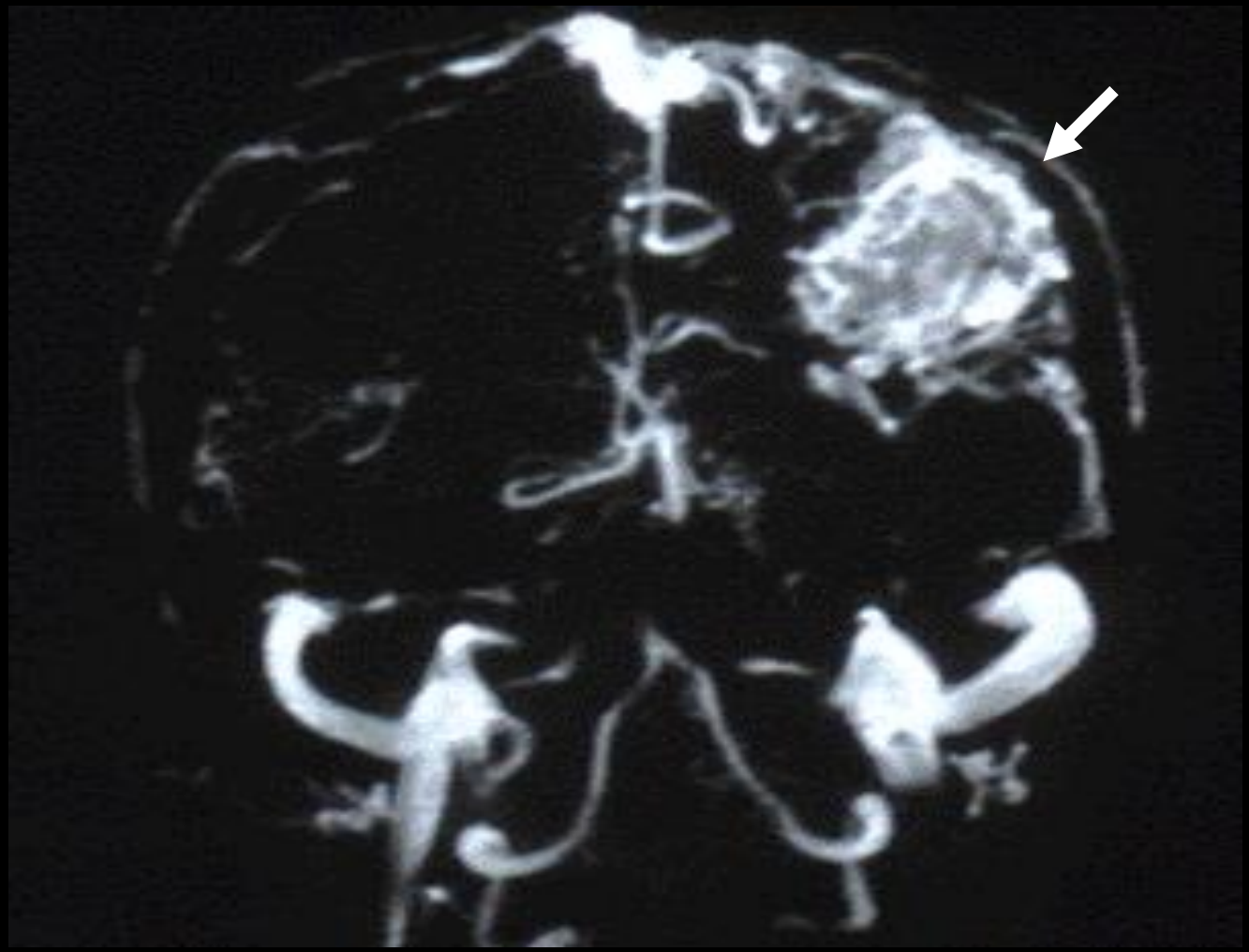
1. Rupture of an arterial aneurysm into adjacent vein.
2. Penetrating injuries through the wall of artery & vein.
3. Inflammatory necrosis of adjacent vessels.
4. Intentionally created arteriovenous fistulas in treatment of chronic renal failure by dialysis.

**Complications:**

1. Cardiac failure.

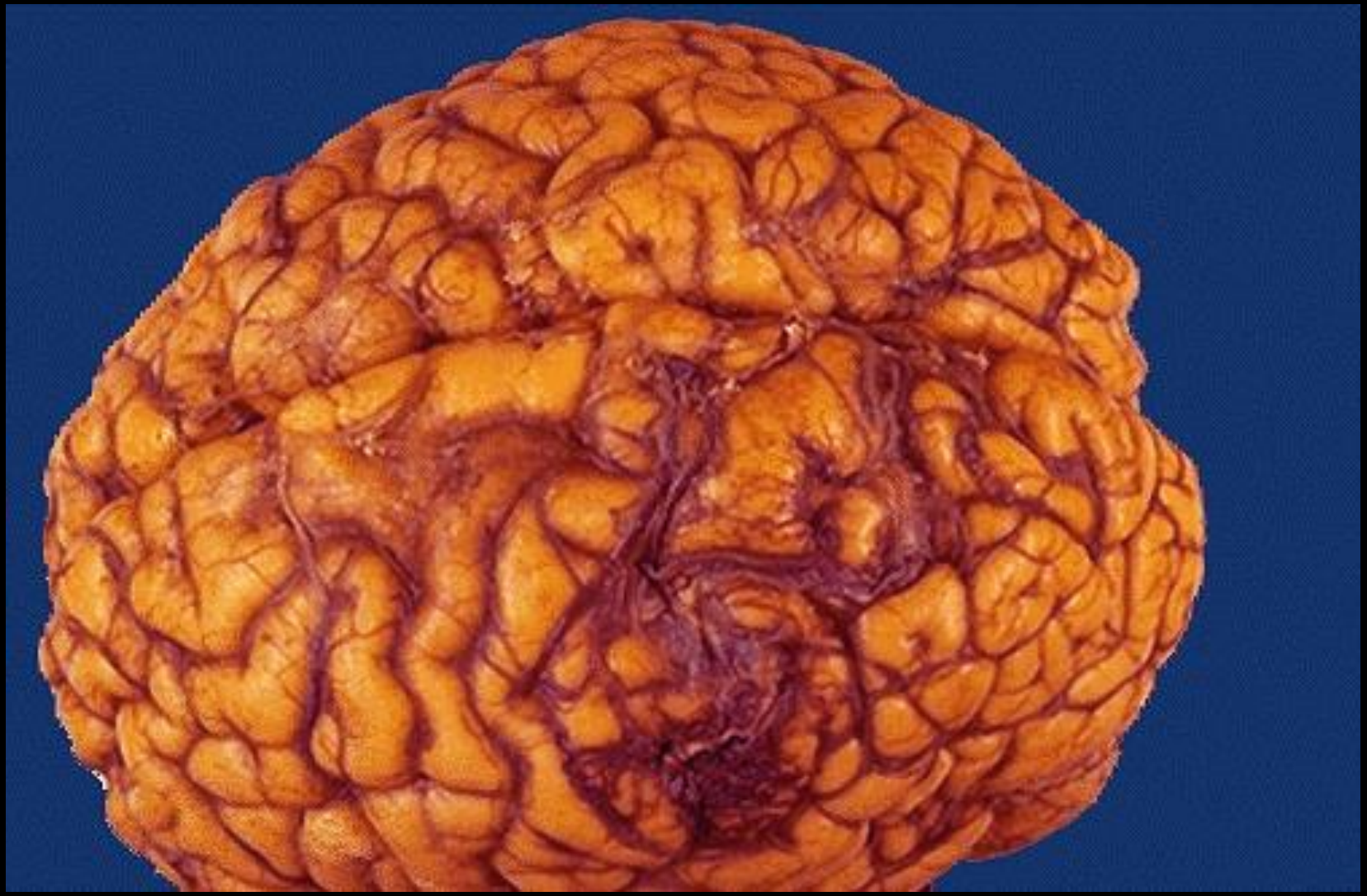
2. Rupture & cause sever hemorrhage especially in brain.

# AVM (MRI of brain)

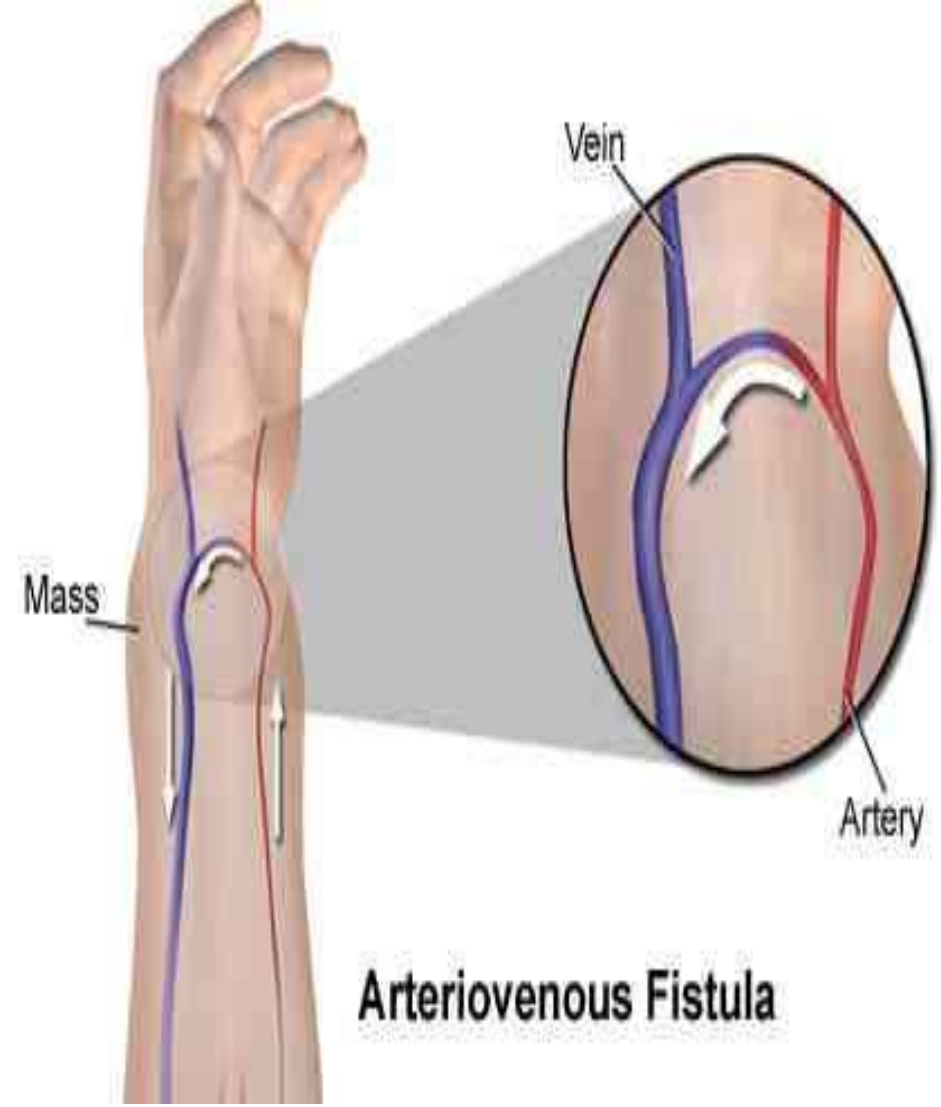


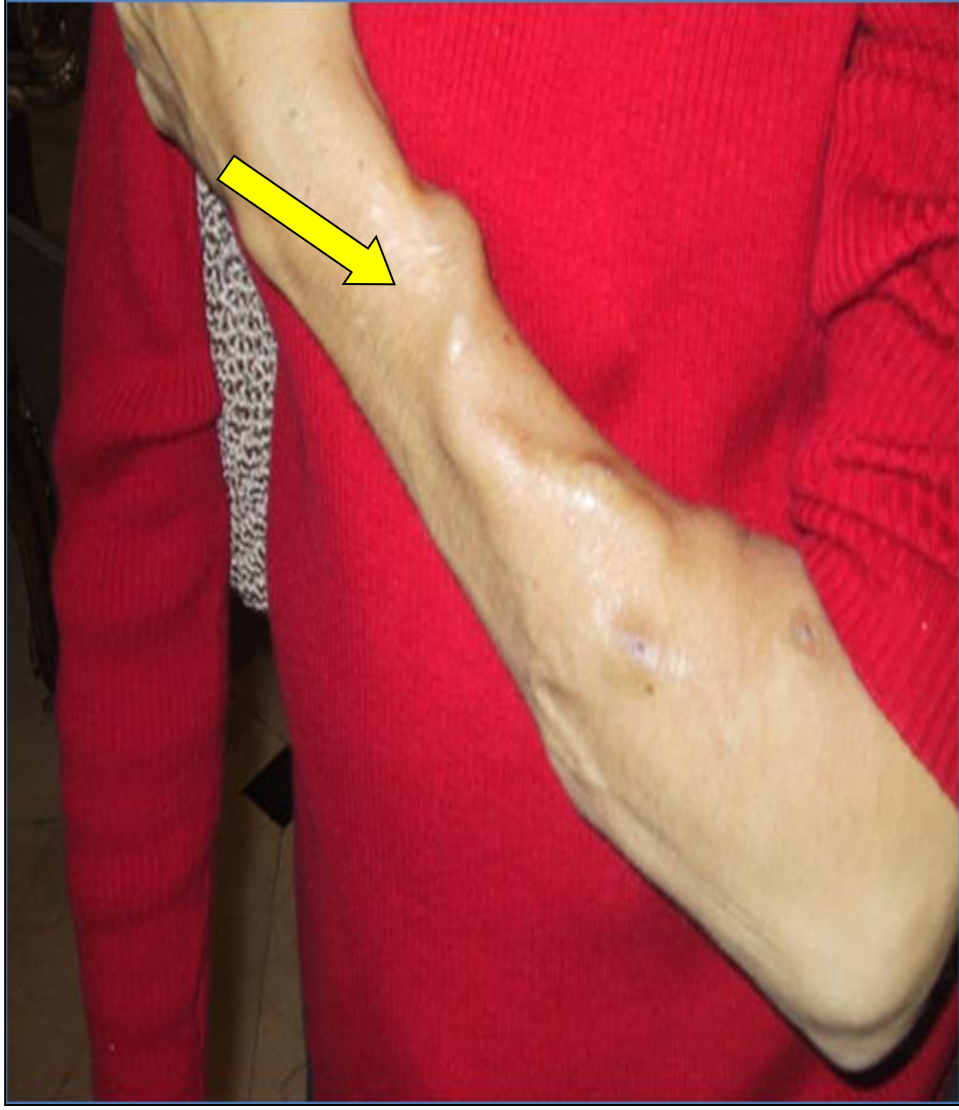
There is a large abnormal mass of vessels in the parietal lobe (arrow). Such abnormal vessels are prone to bleeding

# AVM Brain



**There is a mass of irregular, tortuous vessels over the left posterior parietal region. This is one cause for hemorrhage, particularly in persons aged 10 to 30 years.**



# Arteriovenous fistula

# II. Arteriosclerosis

Is a generic term for thickening & loss of elasticity of arterial walls, it occurs in three forms:

1. Atherosclerosis, the most frequent & important form (later discuss).

2. Monckeberg medial calcific sclerosis, characterized by calcific deposits in muscular arteries in persons older than 50 years, radiological visible & often palpable calcifications.

3. Arteriolosclerosis: (Disease of small arteries & arterioles).

Most often in patient with hypertension & diabetes mellitus (later discuss).

## Atherosclerosis (ATH)

Characterized by intimal lesion called atheromas, or atheromatous or fibrofatty plaques, that protrude & obstruct vascular lumen.

ATH contributes for more than 50% of all death.

## Clinical Significance of ATH

- ✓ ATH primarily affects elastic arteries & medium sized (muscular arteries).
- ✓ *Symptomatic ATH disease most often involves arteries supplying the heart (Myocardial Infarction), arteries of brain (Stroke), arteries of kidney (Renal Infarction), & arteries of lower limbs (Gangrene of legs).*

- ✓ In small arteries, atheroma's can occlude lumina of arteries by super adding thrombosis; result in ischemia of those parts of body.
- ✓ In large & medium sized arteries plaques are destructive, that result in weakening the affected vessel wall causing aneurysm that may rupture & may produce emboli from these friable thrombi.

**Morphology of ATH:** ATH passes in **3** stages:

**1. Fatty streaks:** characterized by intimal thickening & lipid accumulation, this is the precursor of atheromas.

**Components of Fatty streaks:**

These streaks are composed of lipid laden foam cells.

It appear in aorta of children below 10 years, regardless the sex, race, geography, environment except fatty streaks of coronary arteries formed at adolescent which later tend to form the plaques (not all streaks are tend to become plaques).

**Gross:** multiple yellow, fat spots (fatty dots) less than 1mm in diameter that coalesce into large streaks of 1cm or more.

## 2. Atheromatous Plaques:

These are raised focal lesions within the intima, also called (Fibrofatty or Fibrolipid Plaques).

These plaques consist of two parts:

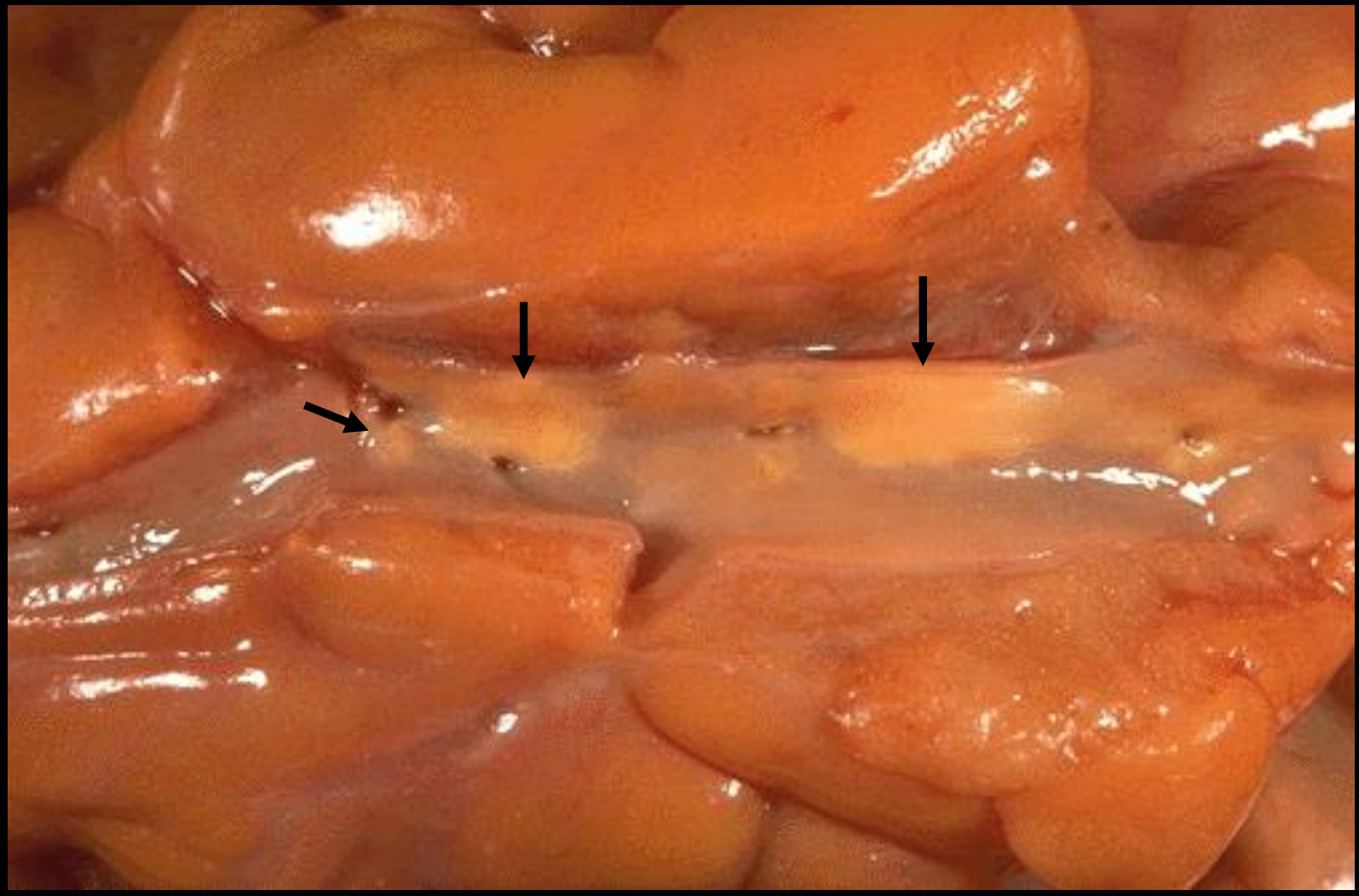
I. Core: soft, yellow core.

Consist of cholesterol & cholesterol esters.

II. Cap: firm, white, fibrous cap.

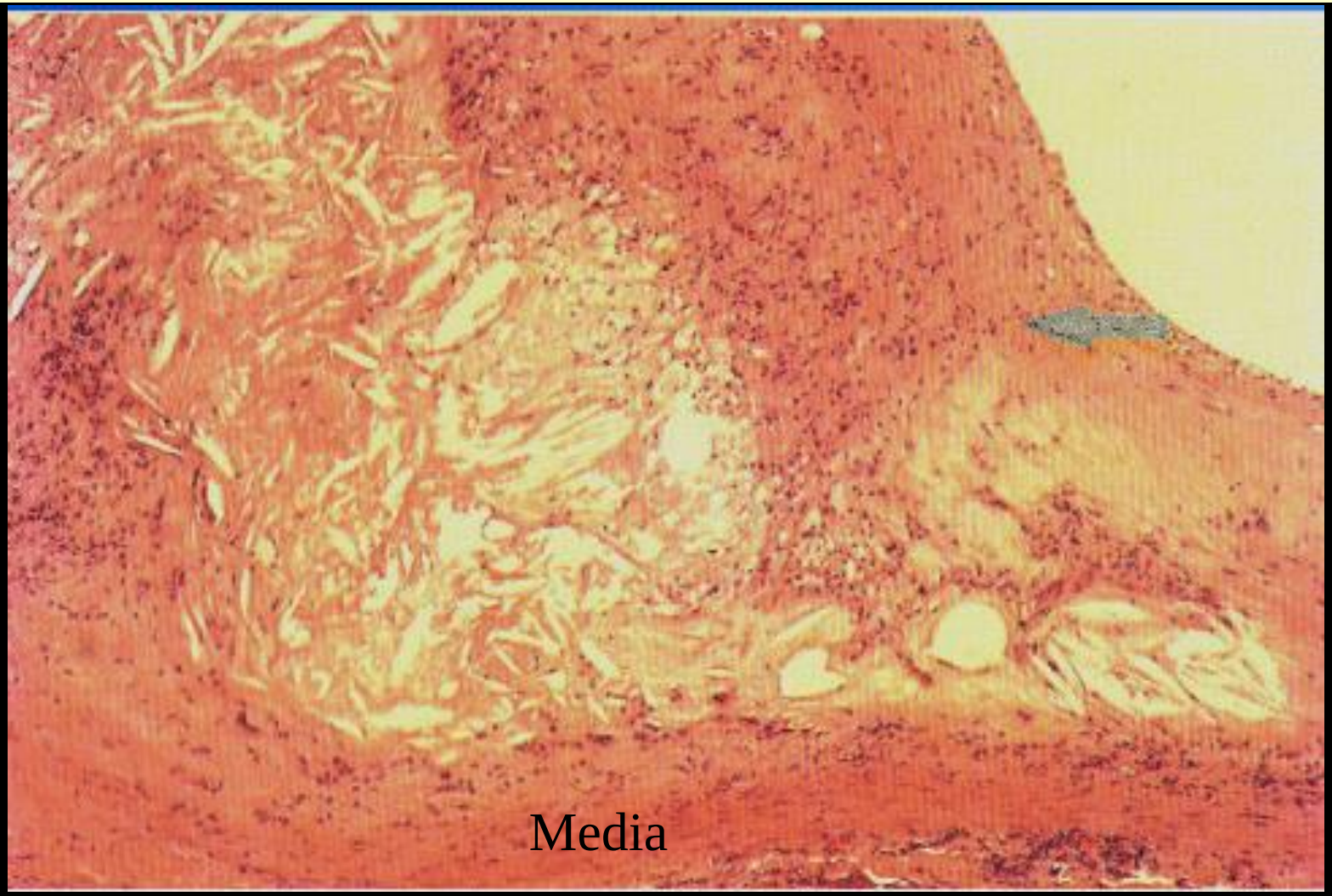
These plaques are varied in their size from 0.3 to 1.5cm in diameter, sometimes they are coalesces to form large masses protrude into lumen.

# Mild degree of coronary athersclerosis



**A coronary artery has been opened longitudinally. The coronary extends from left to right across the middle of the picture and is surrounded by epicardial fat. This coronary shows only mild atherosclerosis, with only an occasional yellow-tan lipid plaques (arrows) and no narrowing.**

# Coronary artery atheromatous plaque: (HE) medium power




**The lumen of the artery is at the top right corner, and the band of smooth muscle at the bottom is the atrophic media. The intima is enormously thickened, by the presence deep in it (centre and left) of amorphous material containing large numbers of cholesterol crystals (the unstained clefts). There are many foamy (lipid-filled) macrophages and chronic inflammatory cells in this zone and also in the thick layer of dense fibrous tissue layer (arrow) which separates it from the lumen.**

## 3. Advanced atheromatous lesions:

These lesions are called eccentric lesions (partial, patchy lesions), as disease become advanced, these lesions become diffusely involved the arterial wall.

**Components of atheromatous plaques:**

I. Cells: Smooth muscle cells (SMC), macrophages & other WBC.

II. Extracellular Matrix (ECM): Collagen, elastic fibers, proteoglycans.

III. Intracellular & extracellular lipid.

**Caps:** composed of SMC & dense collagen fibers, beneath & the sides of cap, there are cells include (T-lymphocytes, Macrophages & SMC).

**Core:** consist of

I. disorganized mass of lipid (cholesterol & cholesterol esters).

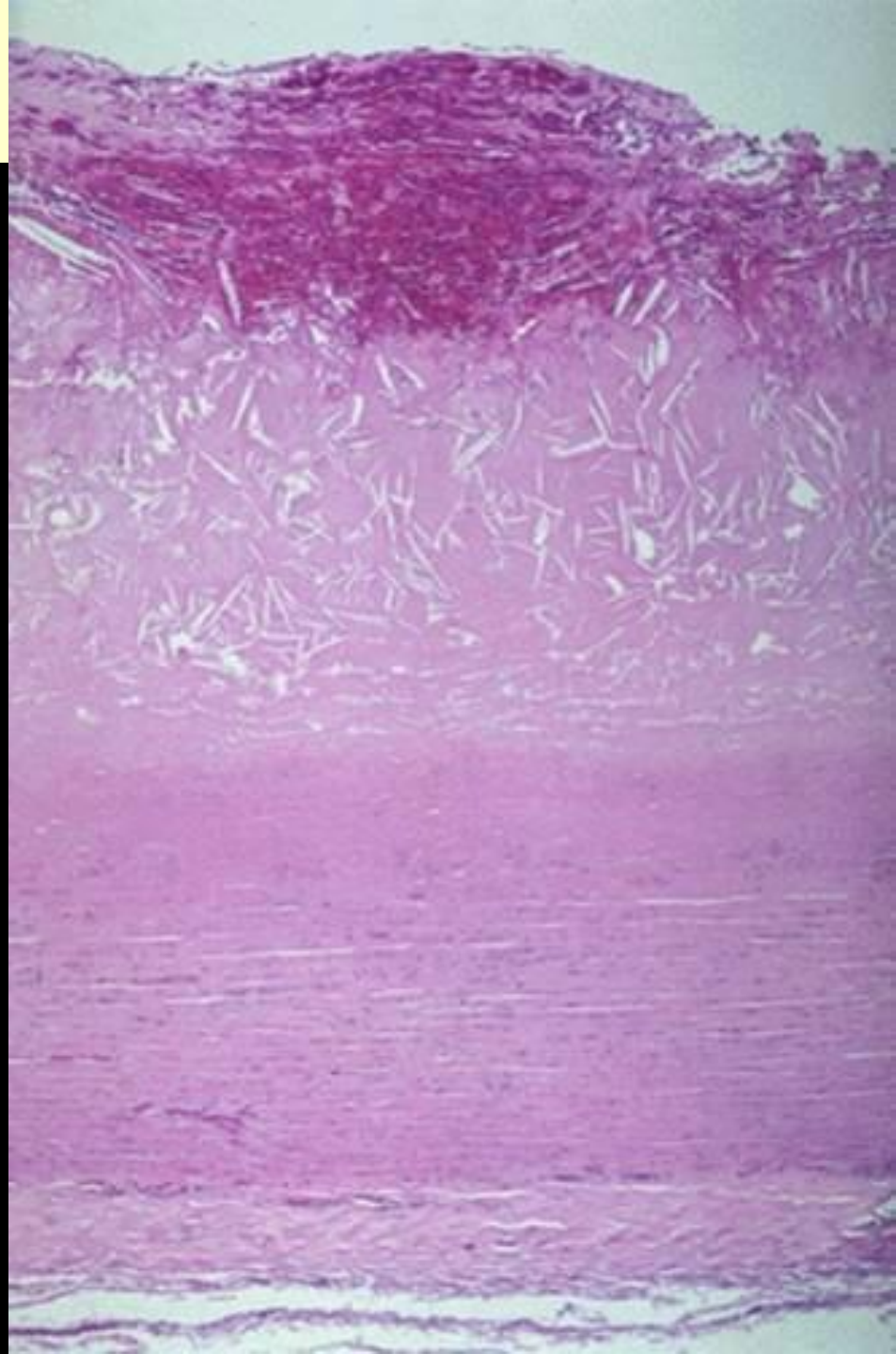
II. Cholesterol clefts.

III. Foam cells, they are tissue macrophages filled with lipid (called lipid laden cells), sometime, SMC are also form lipid laden cells.

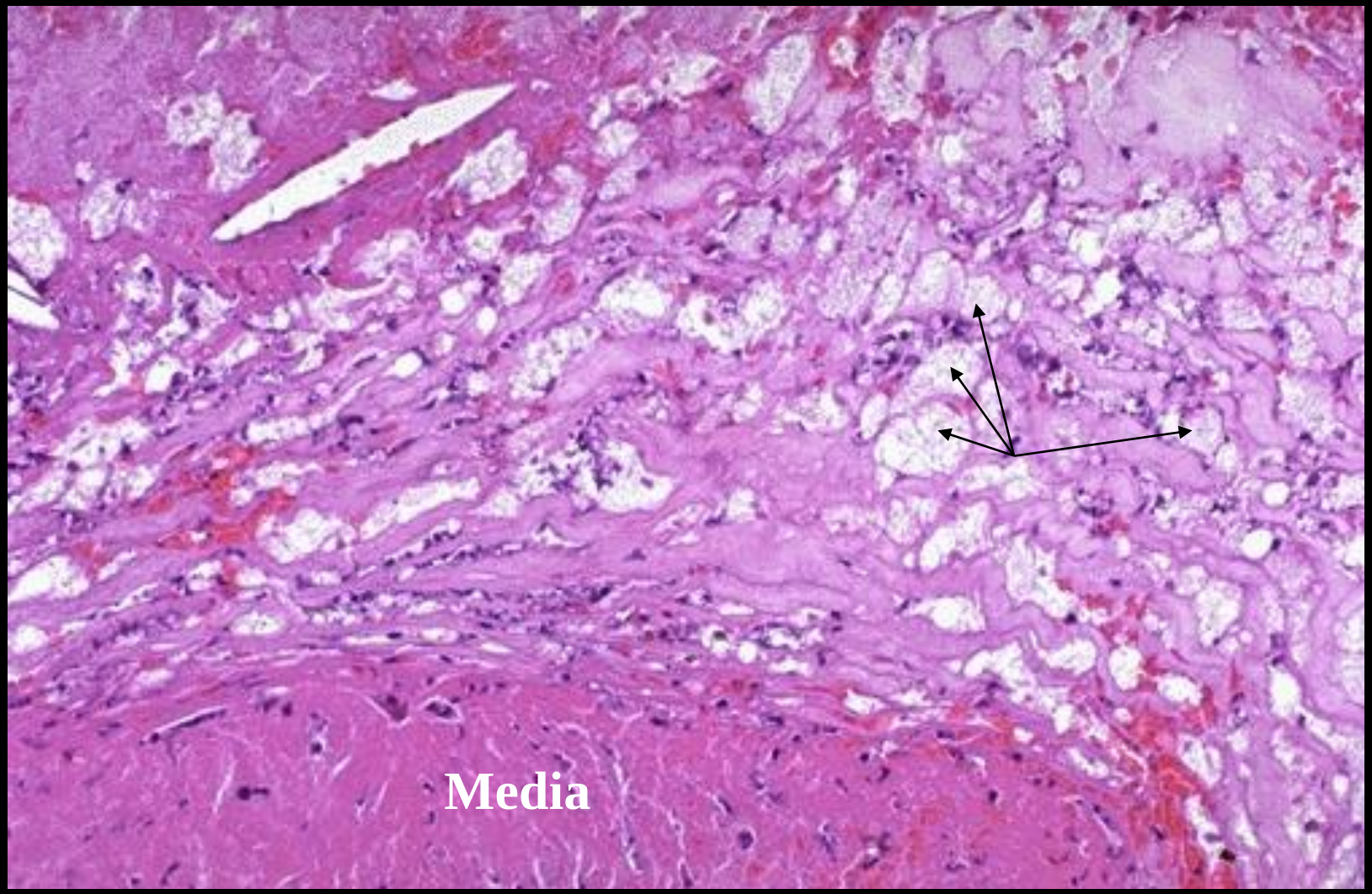
IV. Neovascularization: formation of new blood vessels at the periphery of atheromatous lesions

# Aorta: atheromatous plaque with hemorrhage. (HE) low power

This microscopic cross section of the aorta shows a large luminal atheroma. Cholesterol clefts are numerous in this atheroma. The surface shows intraplaque hemorrhage.



# Atheromatous plaque. (HE) High power




This high magnification of an atheroma shows numerous foam cells (arrows) and an occasional cholesterol cleft. A few dark blue inflammatory cells are scattered within the atheroma.

## Distribution of ATH (Sites)

- **Lower abdominal aorta:** most common site of involvement, mainly around the Ostia of its major branches.
- Then coronary arteries, popliteal, internal carotid artery, braches of circle of Willis.

**Important notes:**

Vessels of upper limbs are usually spared also renal & mesenteric arteries, except at their Ostia.

## Pathological changes in ATH lesions (copmplications):

1. Focal rupture, ulceration & erosion of the luminal surface of atheromatous plaques.

These changes induce thrombus formation, or result in discharge of contents of atheromatous lesion into the circulation, called (cholesterol emboli).

2. Hemorrhage:

Hemorrhage into the plaques (mainly in coronary arteries), may result in formation of hematoma within the plaques that increase the risk of rupture.

3. Superimposed thrombosis:

This is a most feared complication; usually follow the rupture, ulceration & erosion of luminal surface of plaque.

4. Embolization: it is a complication of superimposed thrombosis.

5. Aneurysmal dilatation:

Weakening in the wall of involved vessels, result in abnormal dilatation in the wall of blood vessel.

## Risk Factors of ATH:

Major risk factors: this include

### Unmodified risk factors

**1. Age:**

ATH is not usually clinically evident until middle age or later, death rate from Ischemic Heart Diseases (IHD) due to ATH is raised with each decade.

**2. Sex:** Male more prone to ATH than the female (due to hormonal causes), **estrogen.**

**This is proved by following evidences:-**

- ✓ Estrogen is protective factor against ATH in premenopausal female unless the female develop hypertension, diabetes mellitus & hyperlipidemia.
- ✓ After menopause, the risk of ATH is increased due to decrease the level of estrogen.
- ✓ Postmenopausal treatment with estrogen gives some protection against ATH.

**3. Familial predisposition:**

Familial predisposition to ATH & IHD is most likely polygenic, may be related to other risk factors (e.g. hypertension, diabetes mellitus & familial hyperlipidemia).

**4. Genetic abnormalities.**