

CARDIOVASCULAR SYSTEM

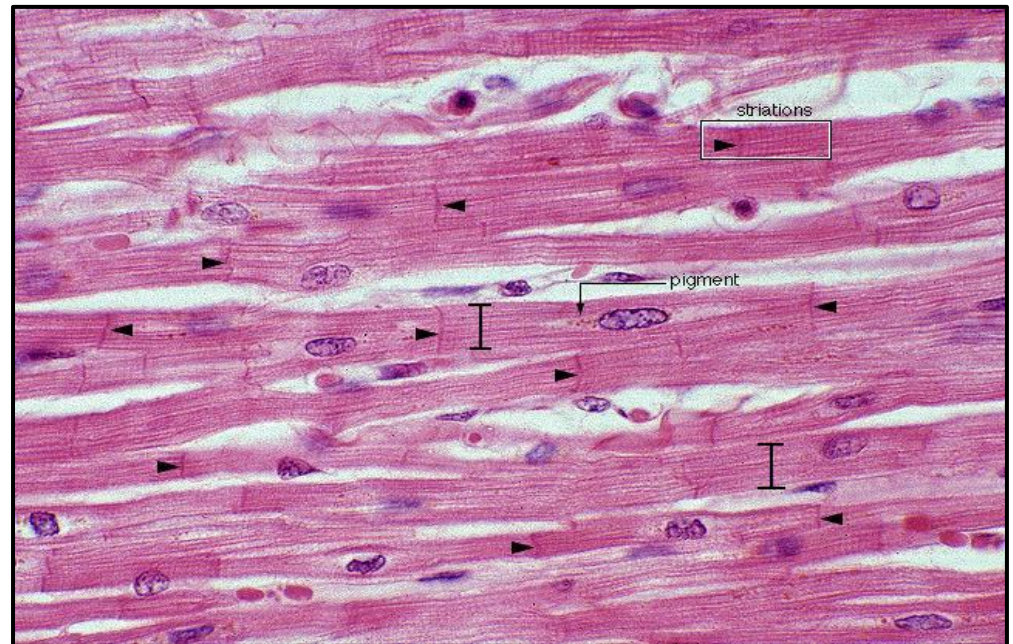
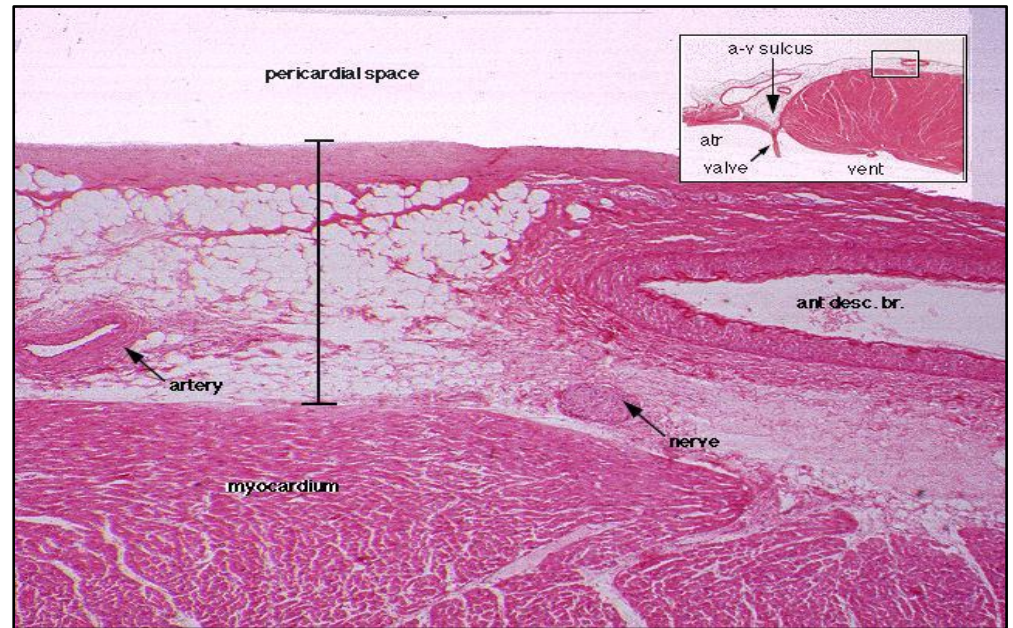
Pathology Practical

Prepared by:

- *Prof. Ammar Al Rikabi*
- *Dr. Sayed Al Esawy*
- *Dr. Shaesta Zaidi*
- *Dr. Marie Mukhashin*

Histology of the Heart

- The heart consists of 3 layers
 - the **Endocardium**,
 - the **Myocardium**, and
 - the **Pericardium**.
- The **Pericardium** consists of arteries, veins, nerves, connective tissue, and variable amounts of fat.
- The **Myocardium** contains **branching, striated muscle cells with centrally located nuclei**. They are connected by **intercalated disks** (arrowheads).



Arteriosclerosis

Arteriosclerosis (literally, "hardening of the arteries") is a generic term for thickening and loss of elasticity of arterial walls.

Three patterns of arteriosclerosis are recognized

- **Atherosclerosis**, the most frequent and important pattern
- **Mönckeberg medial calcific sclerosis** is characterized by calcific deposits in muscular arteries in persons older than age 50.
- **Arteriolosclerosis** affects small arteries and arterioles.

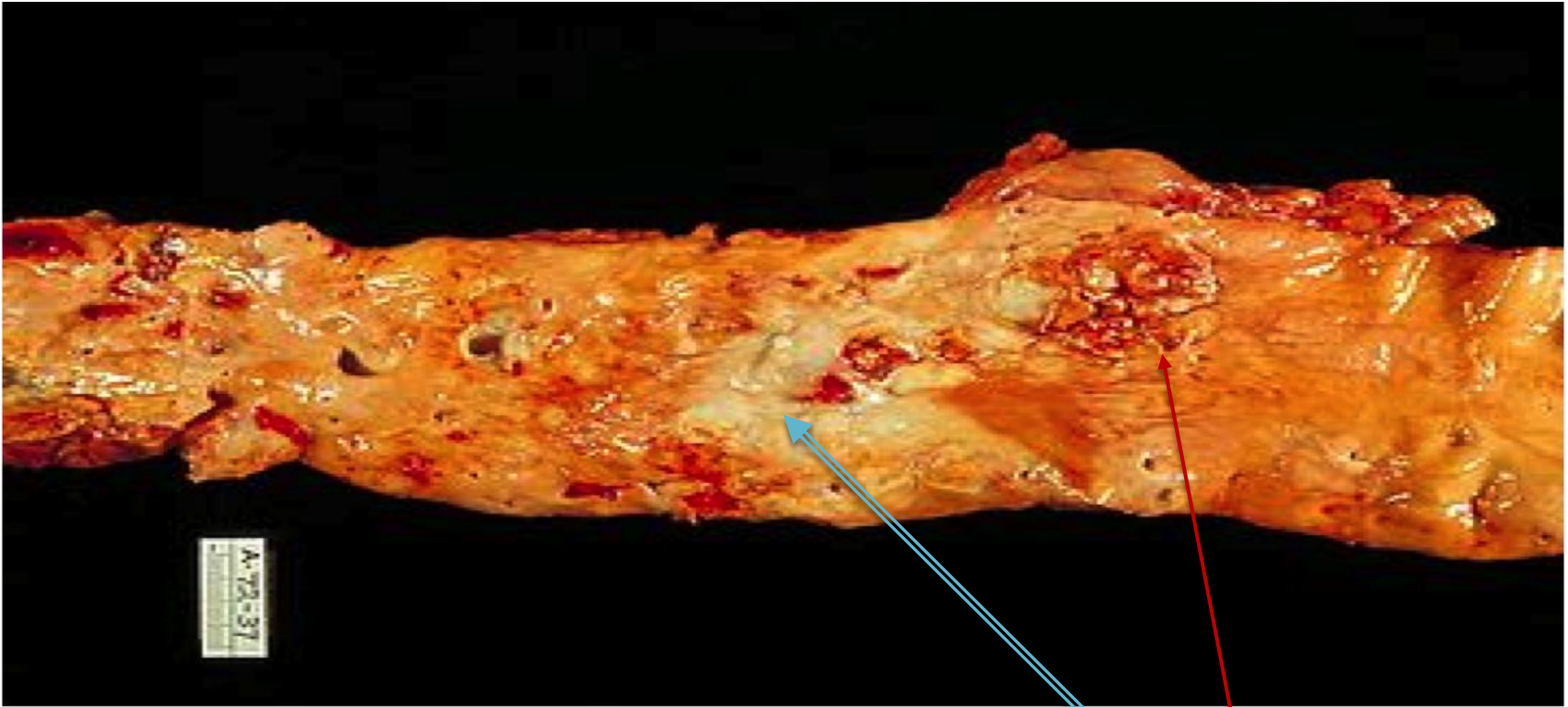
There are two anatomic variants, hyaline and hyperplastic, both associated with thickening of vessel walls with luminal narrowing that may cause downstream ischemic injury.

Most often associated with hypertension and diabetes mellitus.

ATHEROMA OF THE AORTA

- An **atheroma** is an accumulation and swelling in artery walls made up of (mostly) macrophage cells, or debris, and containing lipids (cholesterol and fatty acids), calcium and a variable amount of fibrous connective tissue.

Advanced and complicated atherosclerosis



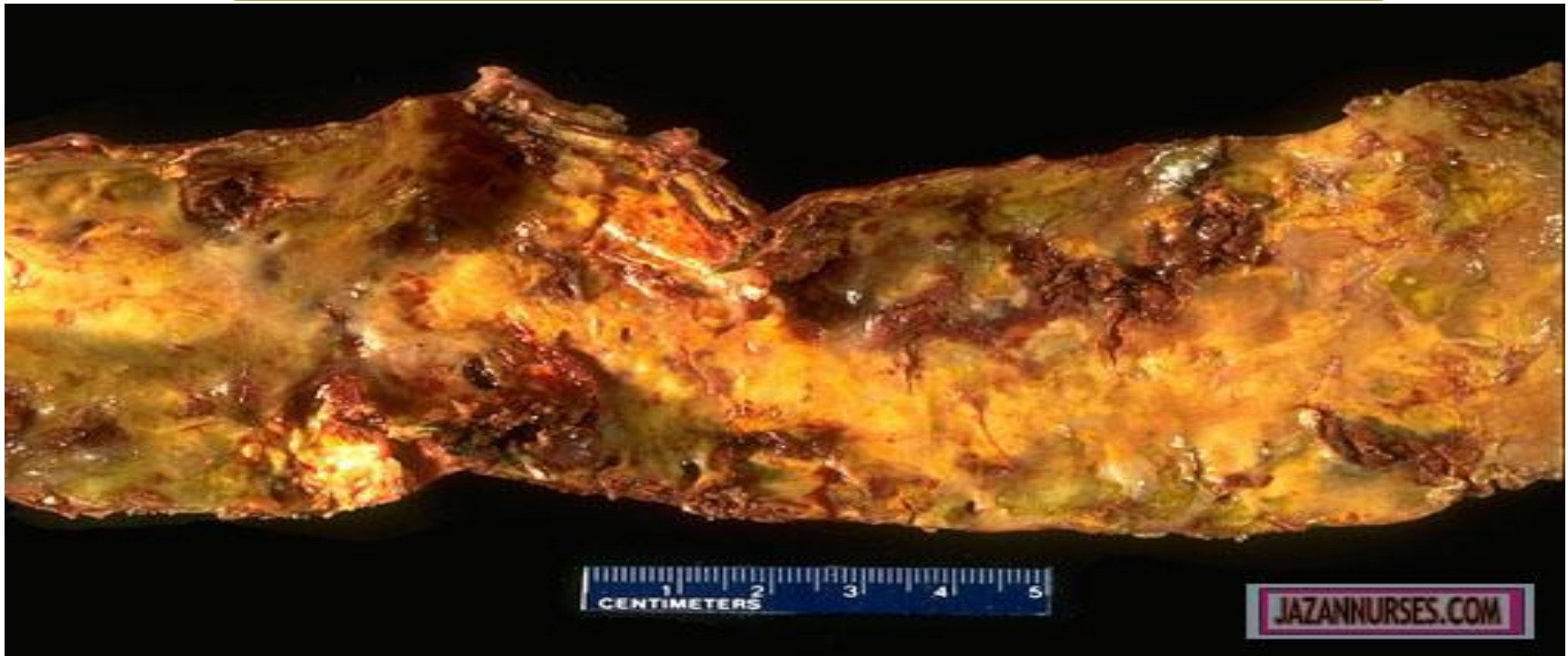
RISK FACTORS:

- Male gender
- Post menopausal woman
- Hyperlipidemia.
- Hypertension.
- Diabetes mellitus.
- Cigarette smoking.

Gross:

- - **Yellow atheromatous plaques.**
- - **Areas of ulceration and haemorrhage**

Severe atherosclerosis of the aorta :



The atheromatous plaques can have secondary changes as:

- ✓ ulceration,
- ✓ haemorrhage,
- ✓ thrombosis,
- ✓ aneurysmal dilatation and
- ✓ calcification.

Complications:

- Vascular thrombosis and distal embolization
- Aneurysm formation
- Cardiac ischaemia
- Ischaemic encephalopathy
- Intermittent claudication

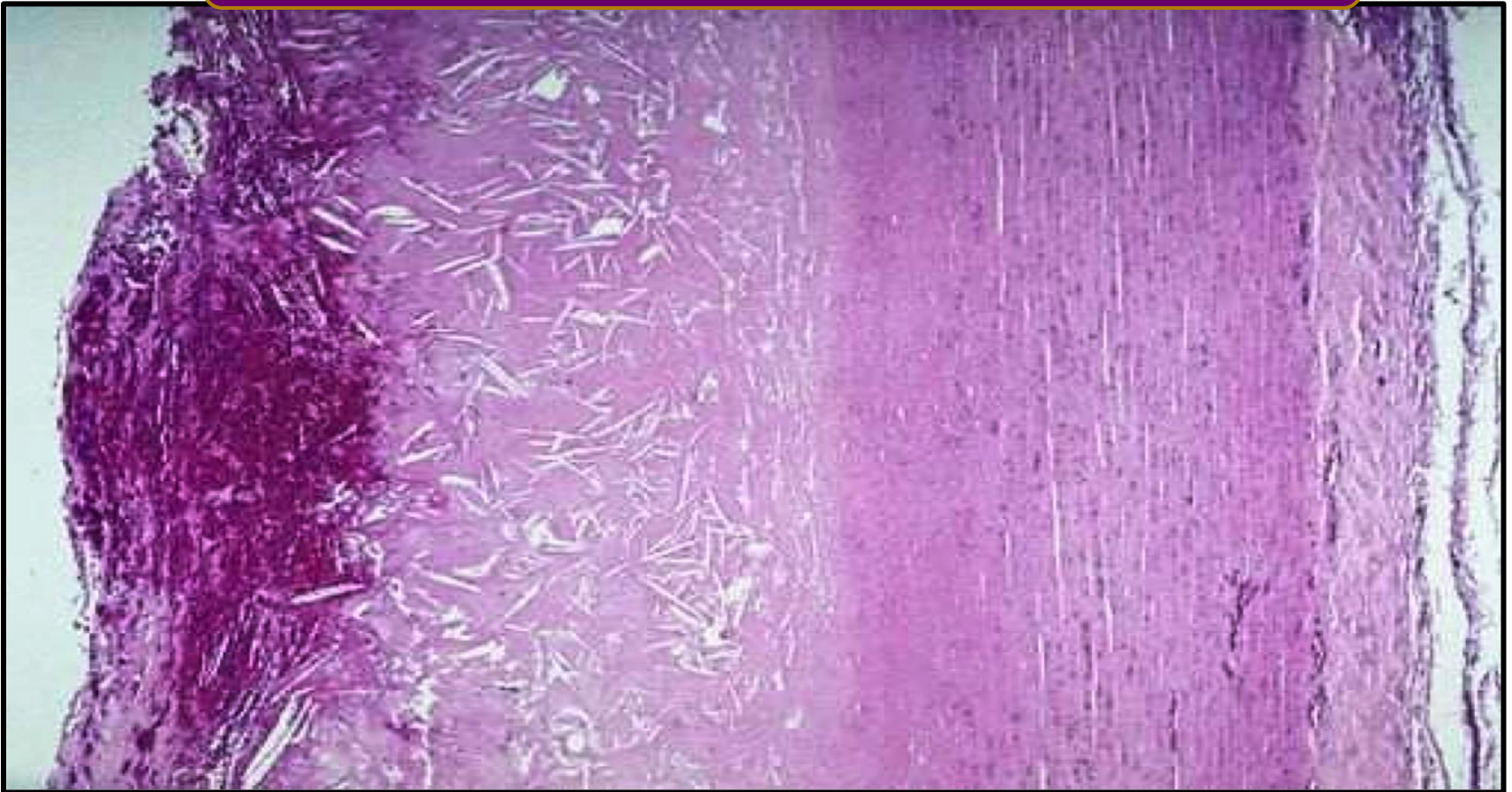
Atheroma of the Aorta - Gross



These three aortas demonstrate mild, moderate, and severe atherosclerosis from bottom to top.

- ❖ At the bottom, the mild atherosclerosis shows only scattered lipid plaques.***
- ❖ The aorta in the middle shows many more larger plaques.***
- ❖ The severe atherosclerosis in the aorta at the top shows extensive ulceration in the plaques.***

Atheroma of the Aorta - LPF



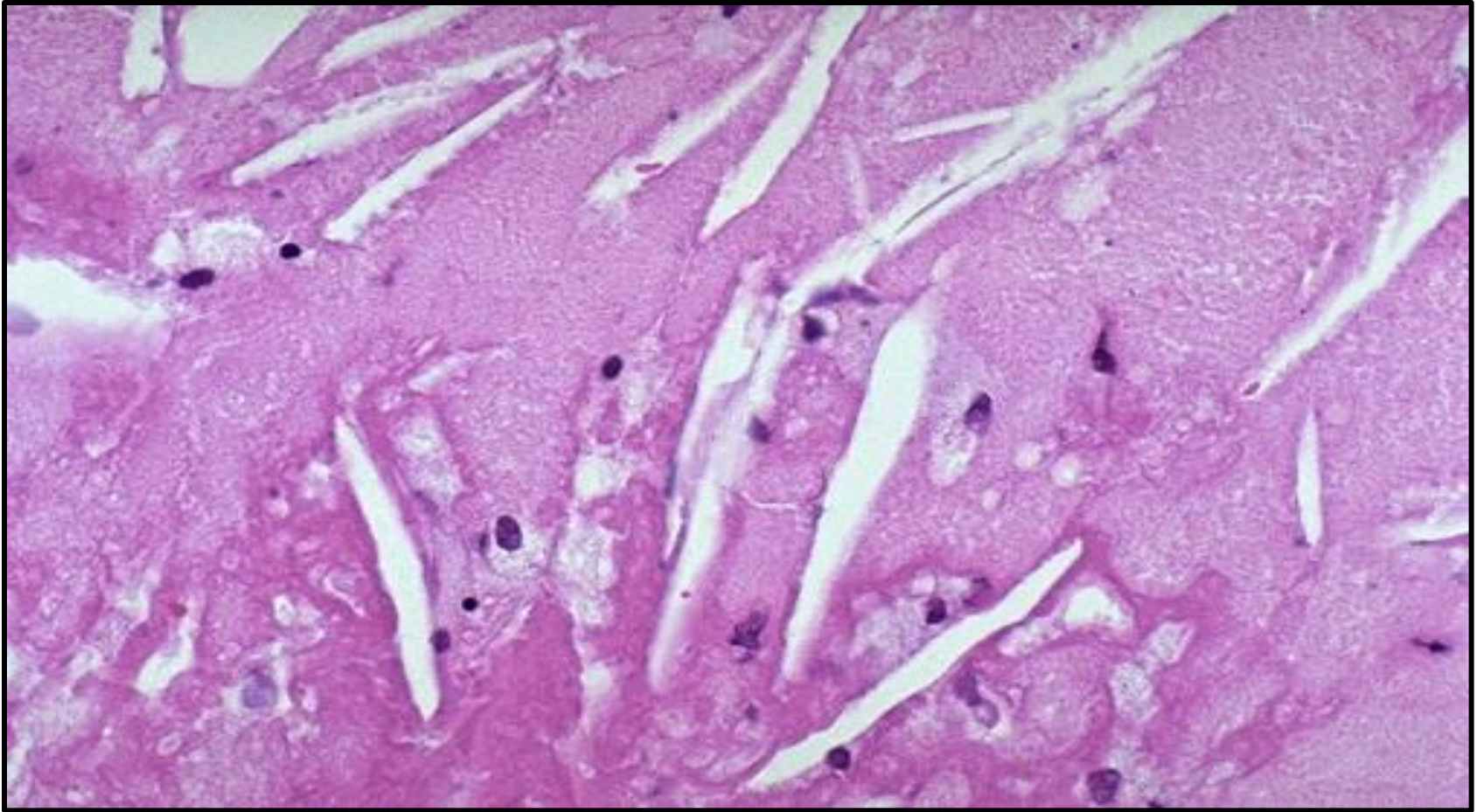
Left

Right

FIVE layers seen in the aorta's section seen starting from the Left to the Right side are:

- 1- Haemorrhage**
- 2- Atheromatous plaque containing cholesterol clefts**
- 3- Fibrosis**
- 4- Elastic Media**
- 5- Adventitia**

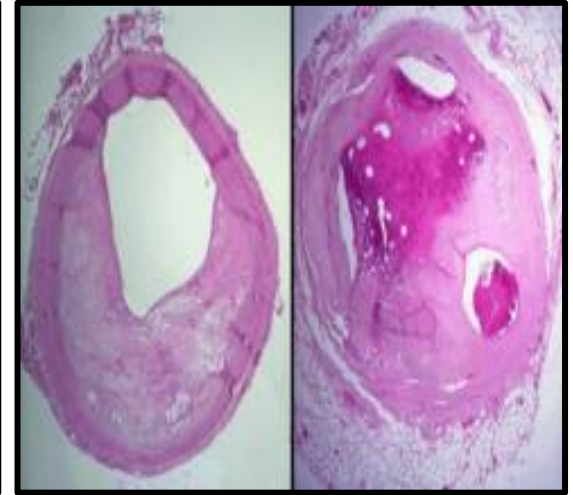
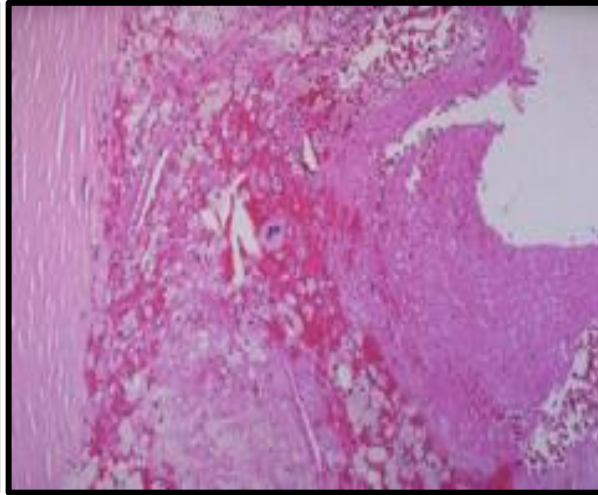
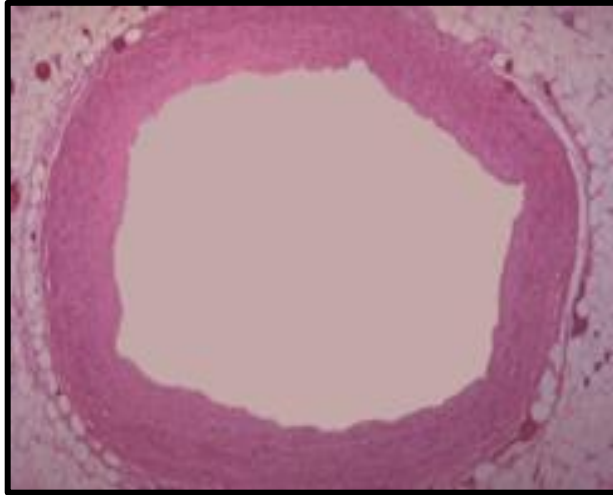
Atheroma of the Aorta - LPF



A high magnification of the **aortic atheroma** with **foam cells** and **cholesterol clefts**.

CORONARY ATHEROSCLEROSIS

Coronary atherosclerosis - LPF

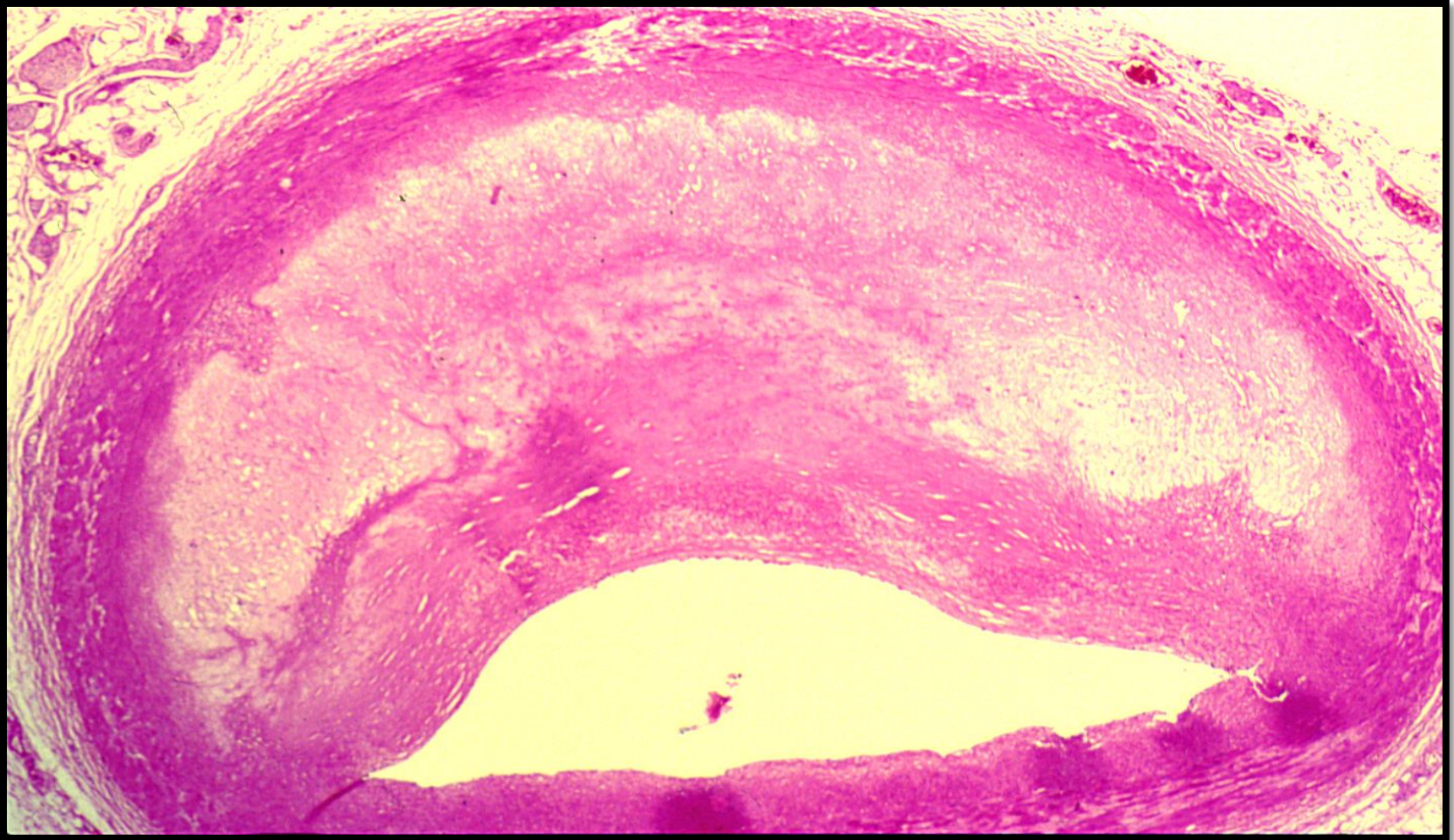


A normal coronary artery with no atherosclerosis and a widely patent lumen that can carry as much blood as the myocardium requires.

Atheromatous plaque in a coronary artery that shows endothelial denudation with disruption and overlying thrombus formation at the right.
The arterial media is at the left

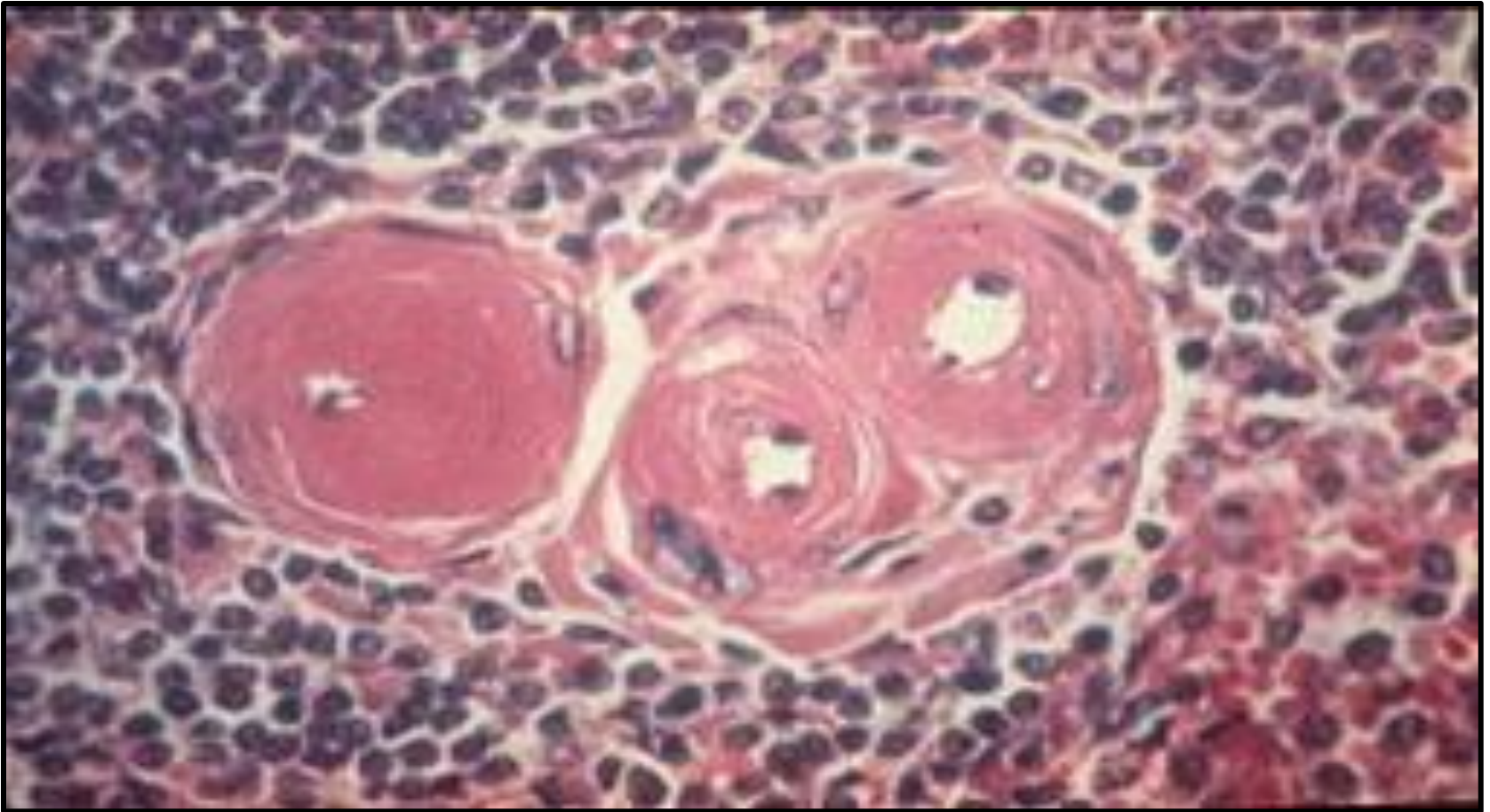
Occlusive coronary atherosclerosis. The coronary at the left is narrowed by 60 to 70%. The coronary at the right is even worse with evidence for previous thrombosis with organization of the thrombus

Coronary atherosclerosis - MPF



***Severe coronary atherosclerosis with narrowing
of the lumen***

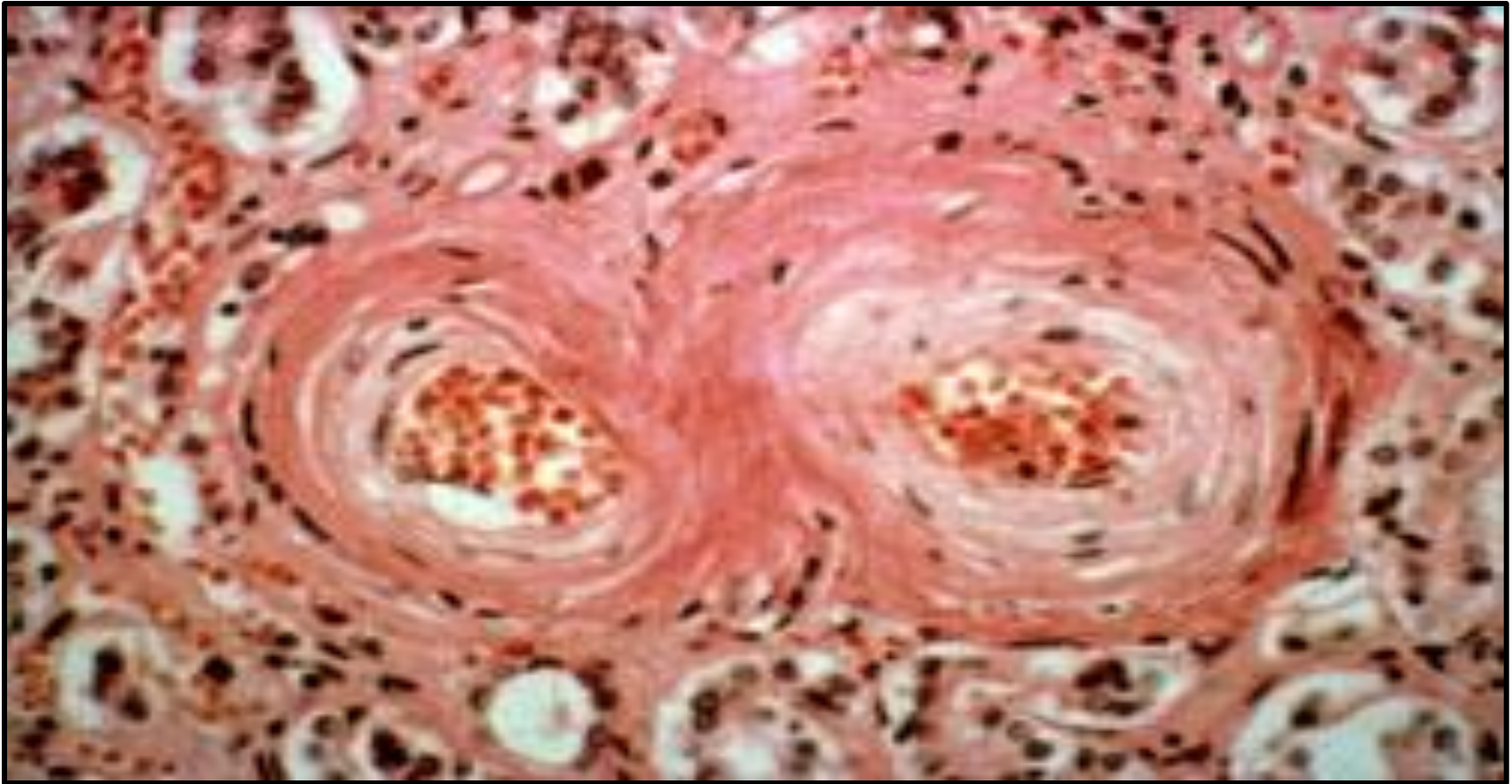
Hyaline arteriolosclerosis - HPF



Hyaline arteriolosclerosis

Arteriosclerosis (hardening of the arteries) involves both small and large vessels. It is commonly found in diabetics and hypertensives.

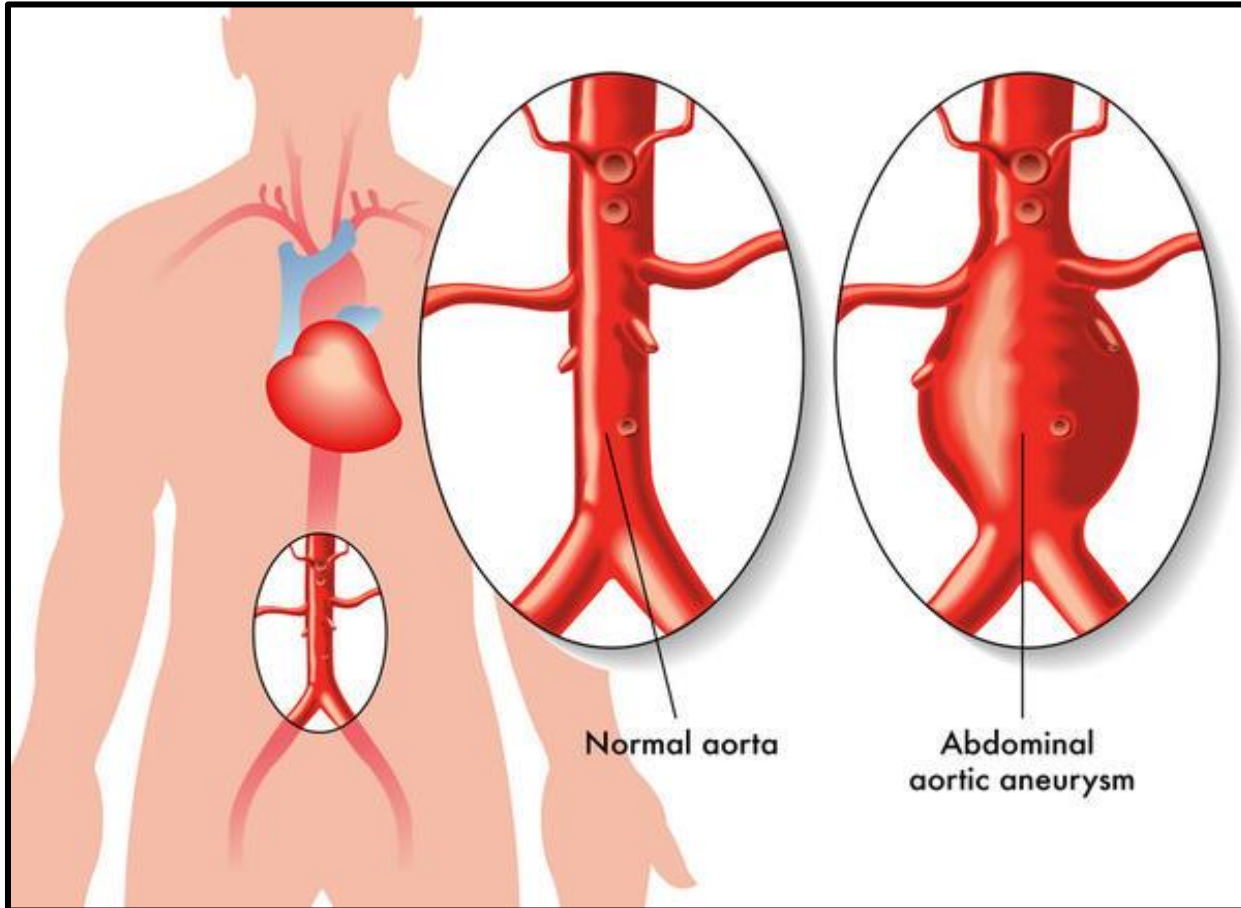
Hyperplastic arteriolosclerosis - HPF



Hyperplastic arteriolosclerosis: This is the other type of small vessel arteriosclerosis. It is predominantly seen in **malignant hypertension** and renal disease associated with **polyarteritis nodosa** and **progressive systemic sclerosis**.

ANEURYSM OF ABDOMINAL AORTA

Abdominal Aortic Aneurysm



Normal aorta

Abdominal
aortic aneurysm

Types of Aneurysms



Saccular Aneurysm



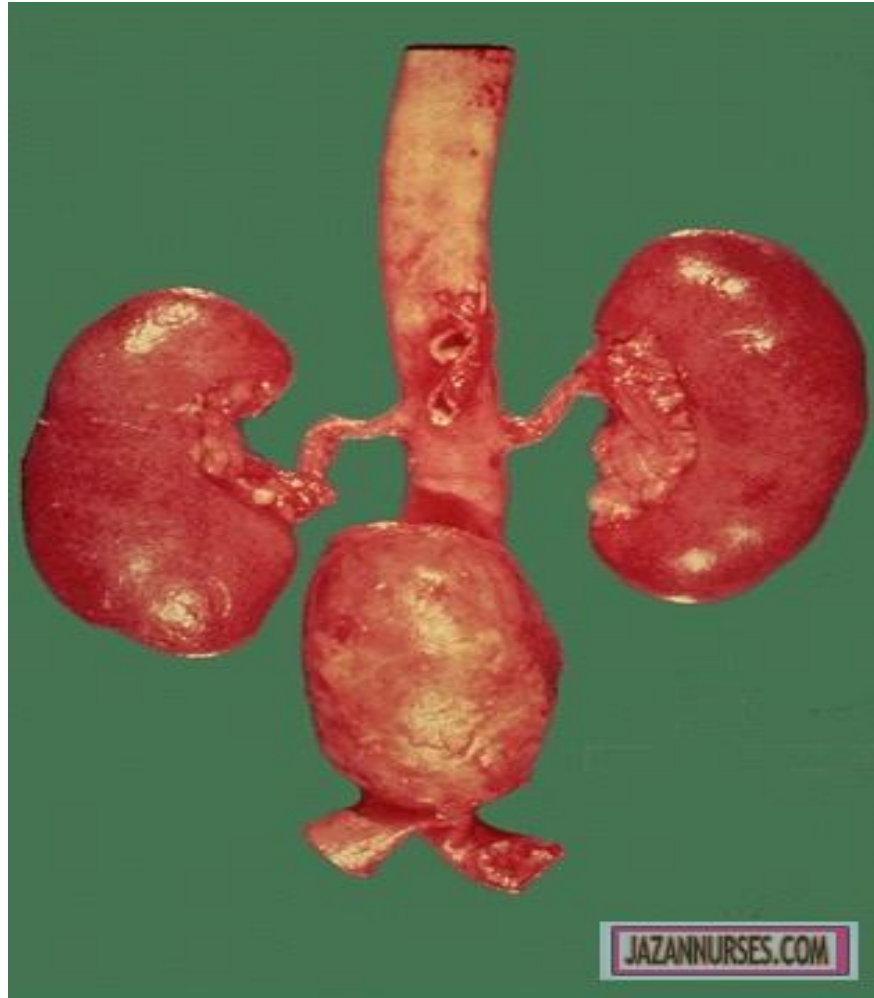
Fusiform Aneurysm



Ruptured Aneurysm

The most likely causes of aneurysms are **atherosclerosis**, **mycotic**, **syphilitic** and **congenital**

Abdominal Aortic Aneurysm



An example of an atherosclerotic aneurysm of the aorta in which a large "bulge" appears just above the aortic bifurcation.

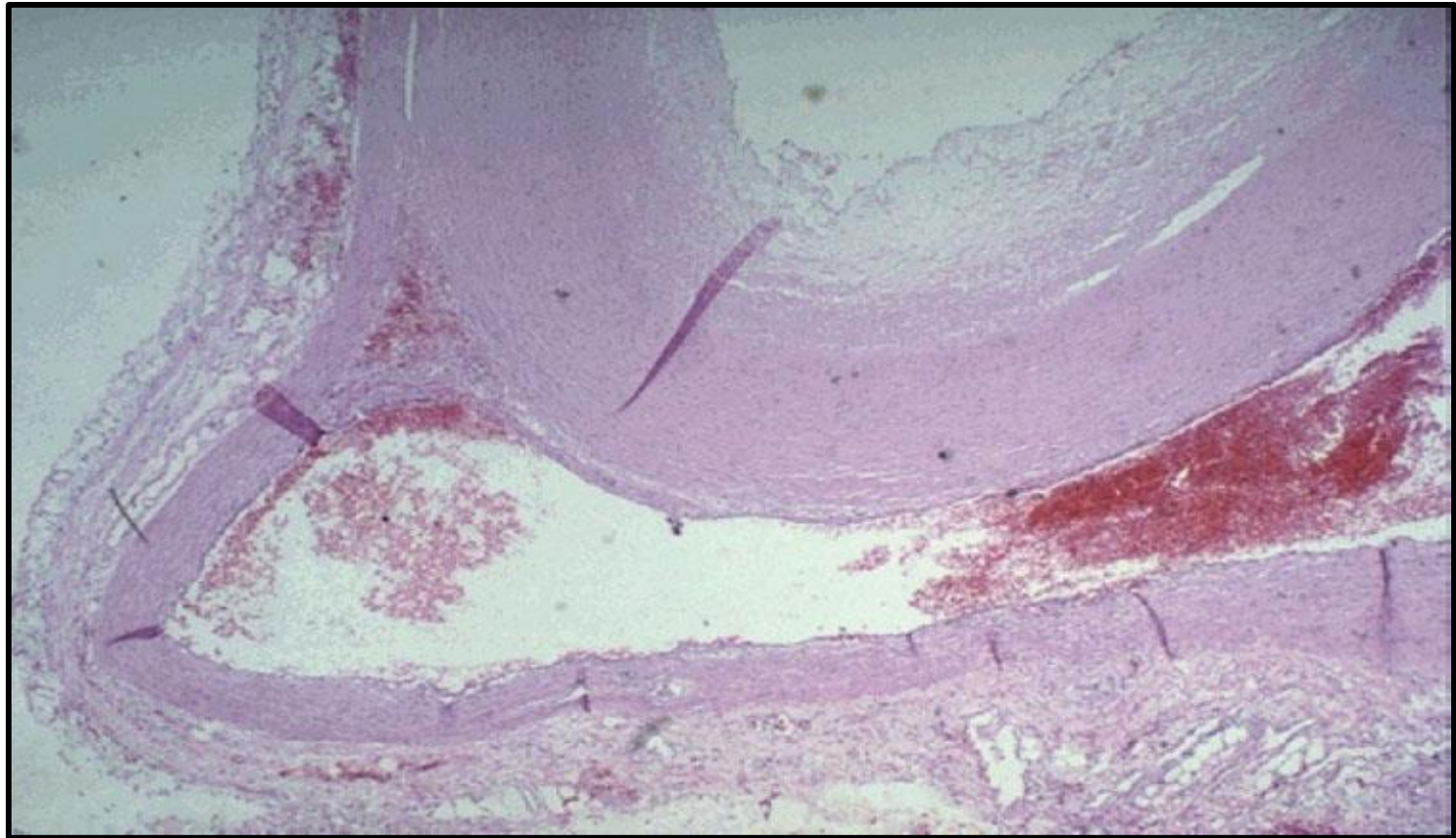
Abdominal Aortic Aneurysm



Aneurysmal dilatation of the abdominal aorta with rupture , intraluminal thrombus and extensive aortic atherosclerosis .

The patient had suddenly developed severe abdominal pain, shocked and collapsed

Dissecting aortic aneurysm - LPF



A dissecting aortic aneurysm occurs when **blood enters the aortic wall** through a defect and moves between two layers of the wall, stripping the inner layer from the outer layer.

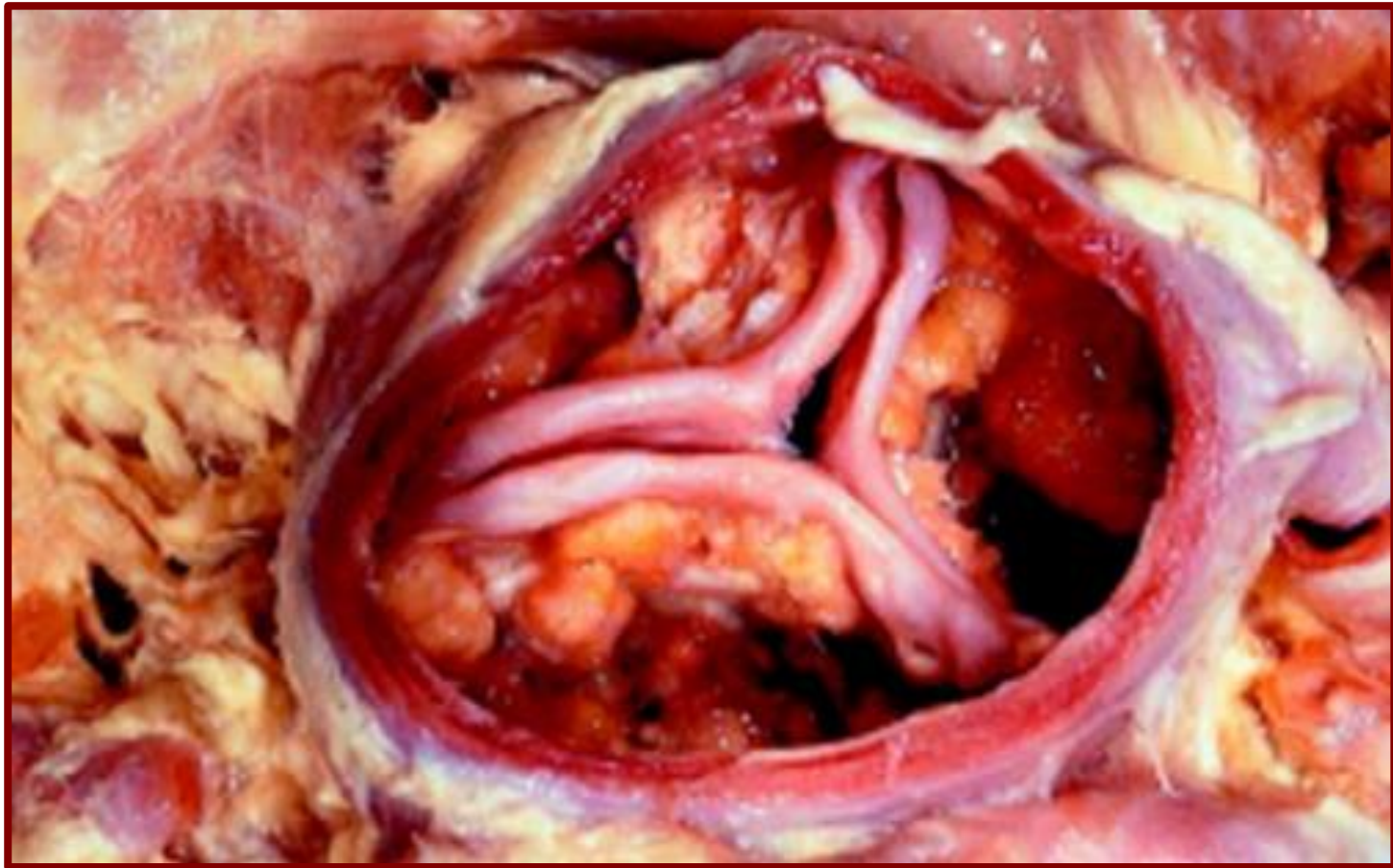
Usually associated with **atherosclerosis, inflammation, and degeneration of the connective tissue of the tunica media**

Vegetations of rheumatic fever on mitral and aortic valves

Chronic Rheumatic Mitral Valvulitis - Gross



large vegetations/hemorrhage along the free margins of the mitral valve.



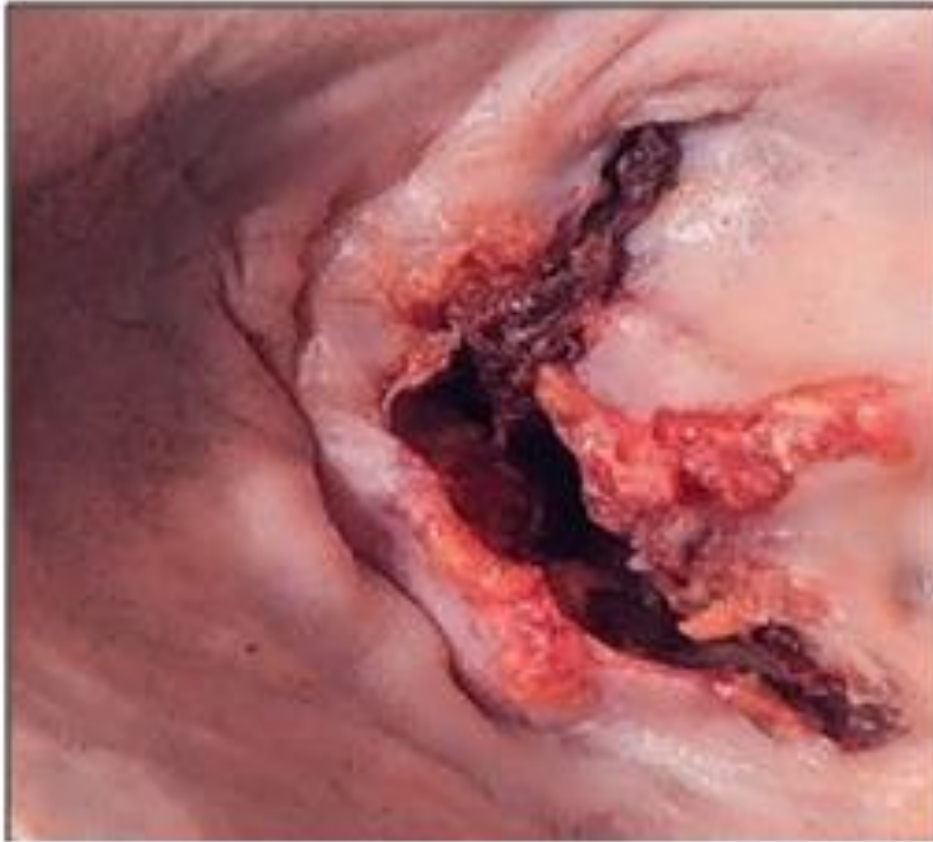
Gross pathology of rheumatic heart disease Aortic stenosis:
Aorta has been removed to show thickened, fused aortic
valve leaflets

Acute Rheumatic Mitral Valvulitis



The small verrucous vegetations are associated with acute rheumatic fever. These warty vegetations are multiple, firm, adherent, small , 1-3 mm in- diameter and form along the line of valve closure over areas of endocardial inflammation.

Mitral stenosis secondary to rheumatic fever.



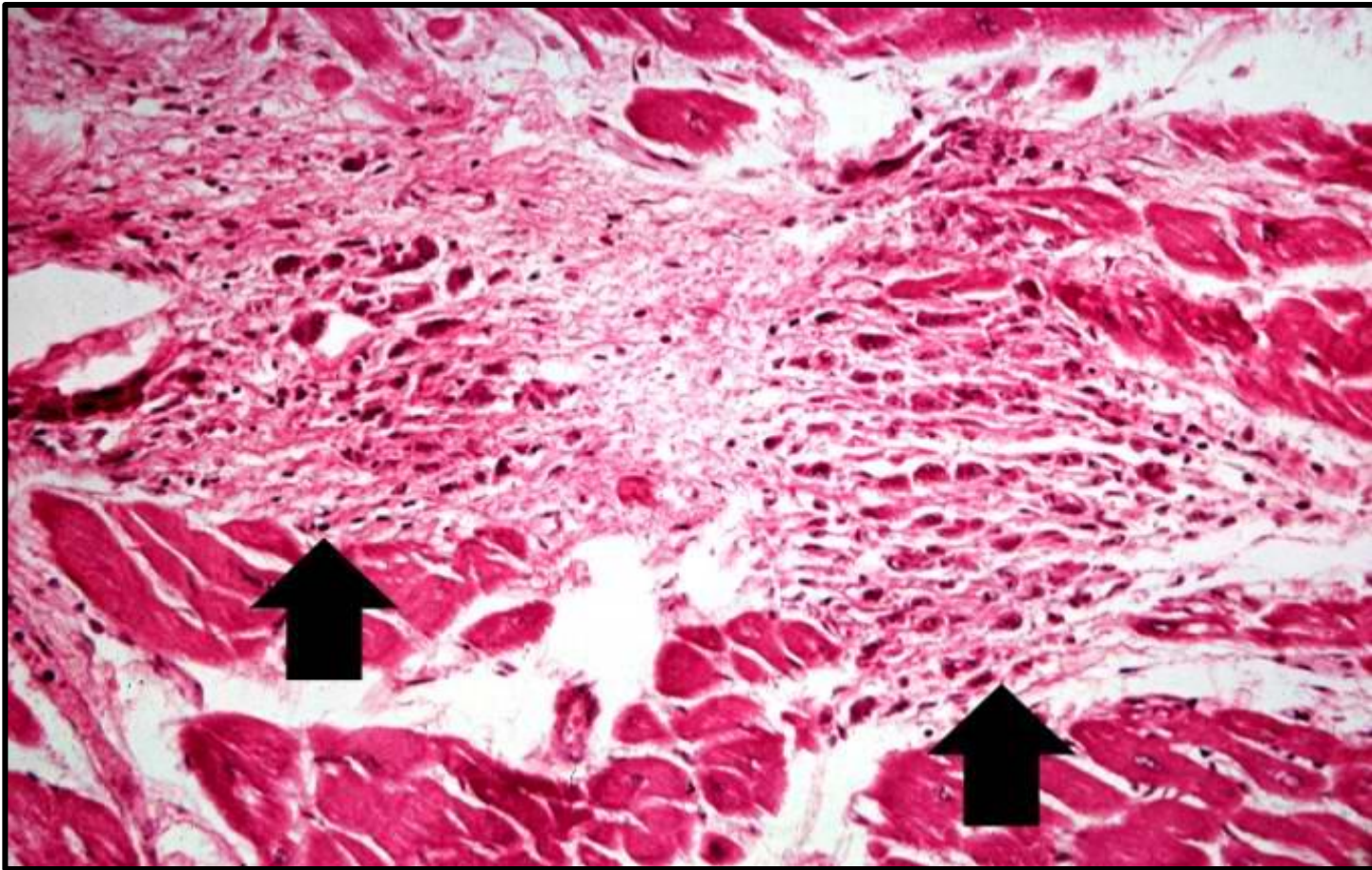
Non-cardiac systemic manifestations rheumatic fever are

- Arthralgia.***
- Arteritis.***
- Sydenham chorea.***
- Erythema marginatum.***

Gross pathologic features of this mitral valve:

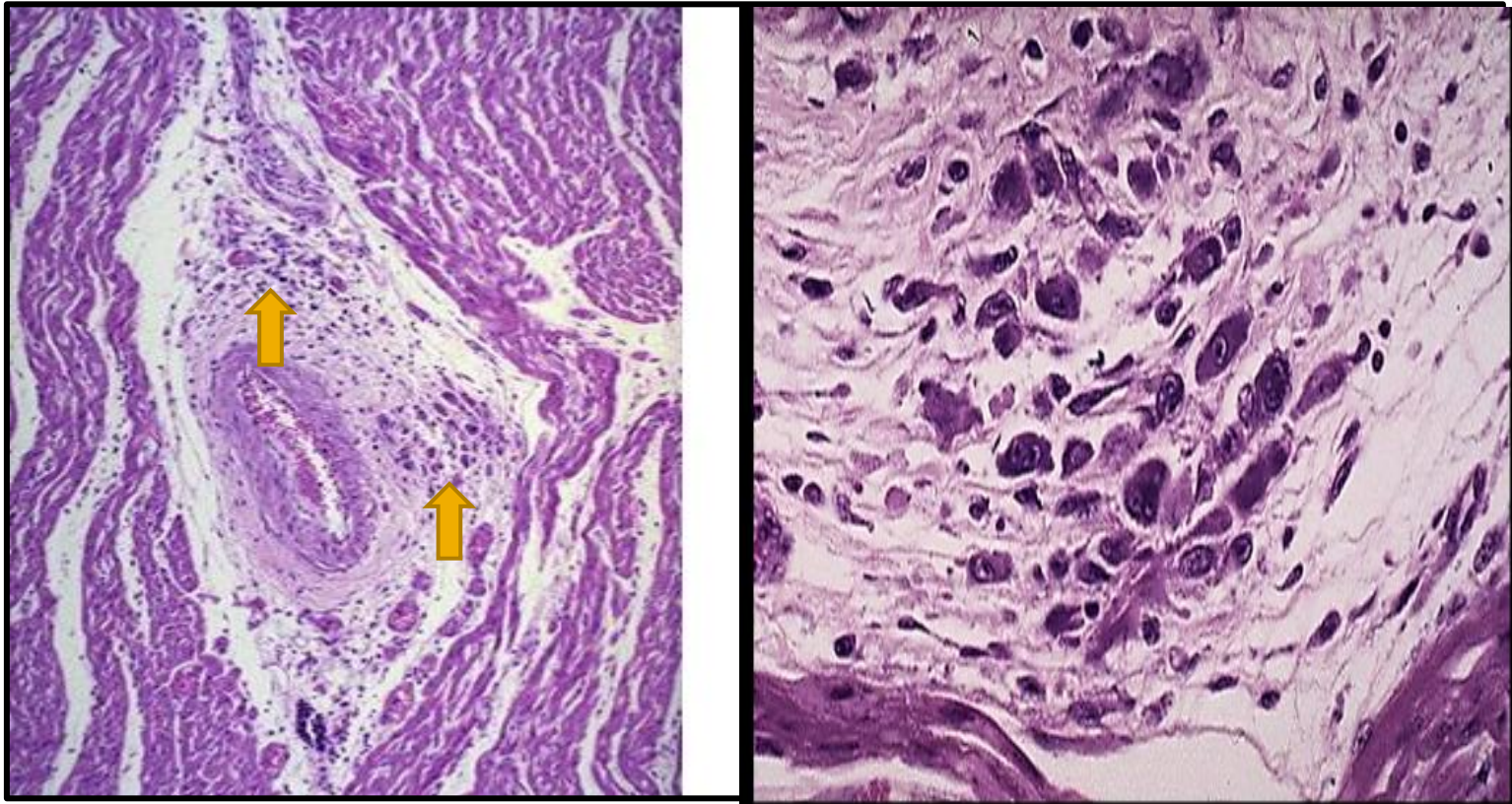
- - Fish mouse deformity.***
- - Fusion of commissures.***
- - Thickening and calcifications of cusps.***
- - Vegetations.***

Acute Rheumatic Carditis



Microscopically, acute rheumatic carditis is marked by a peculiar form of granulomatous inflammation with so-called "Aschoff nodules" seen best in myocardium,

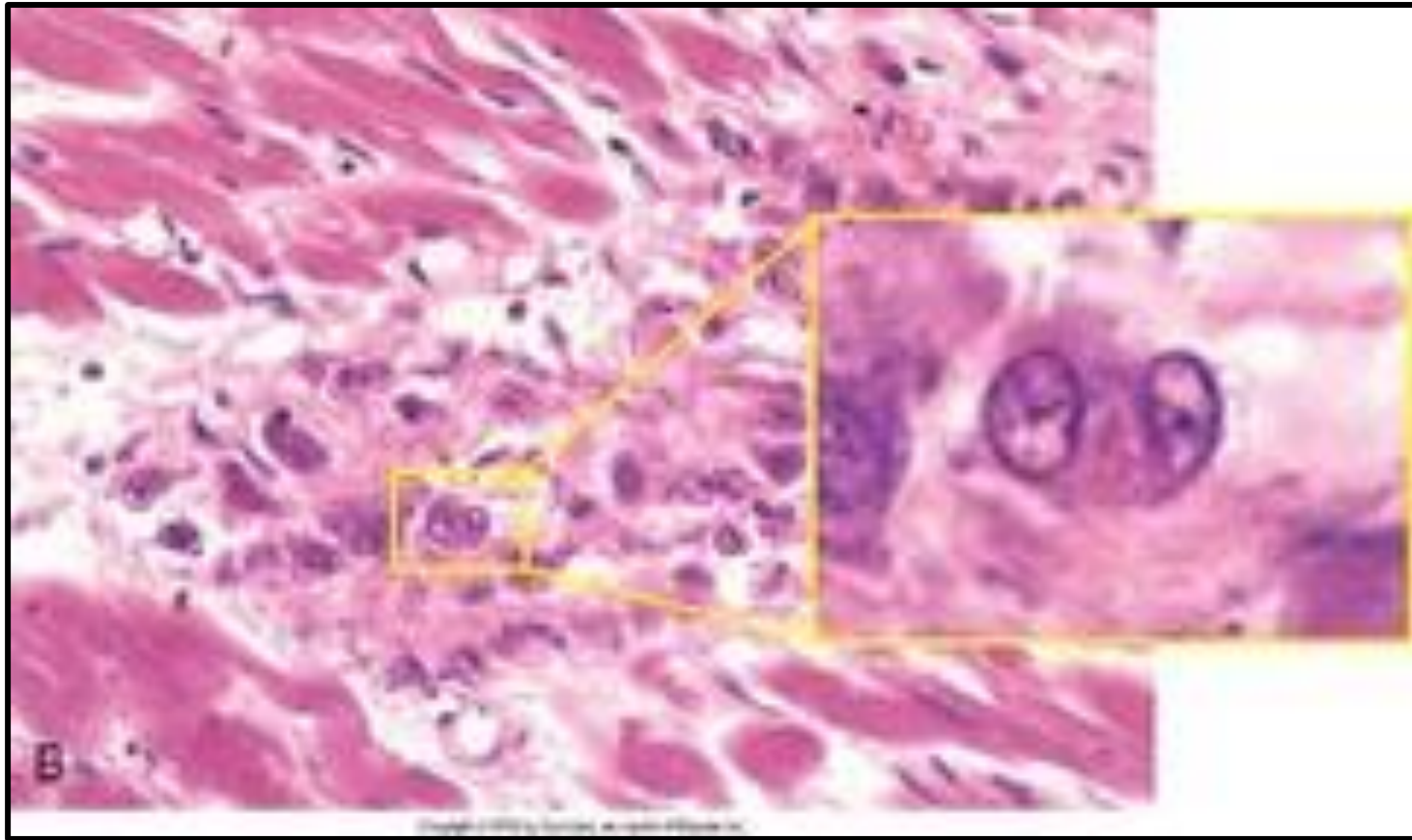
RHEUMATIC MYOCARDIITIS (ASHOFF NODULE)



Aschoff bodies in the intermuscular fibrous septa. They are oval in shape and seen in relation to blood vessels.

Each consists of a focus of fibrinoid necrosis, few lymphocytes, macrophages and few small giant cells with one or several nuclei (Aschoff giant cell).

Ashoff nodule or Anistskow cell.



An **Ashoff nodule** at high magnification.

It affects mainly the **left side of the heart and in particular the posterior wall of the left atrium.**

The most characteristic component is the **Ashoff giant cell.**

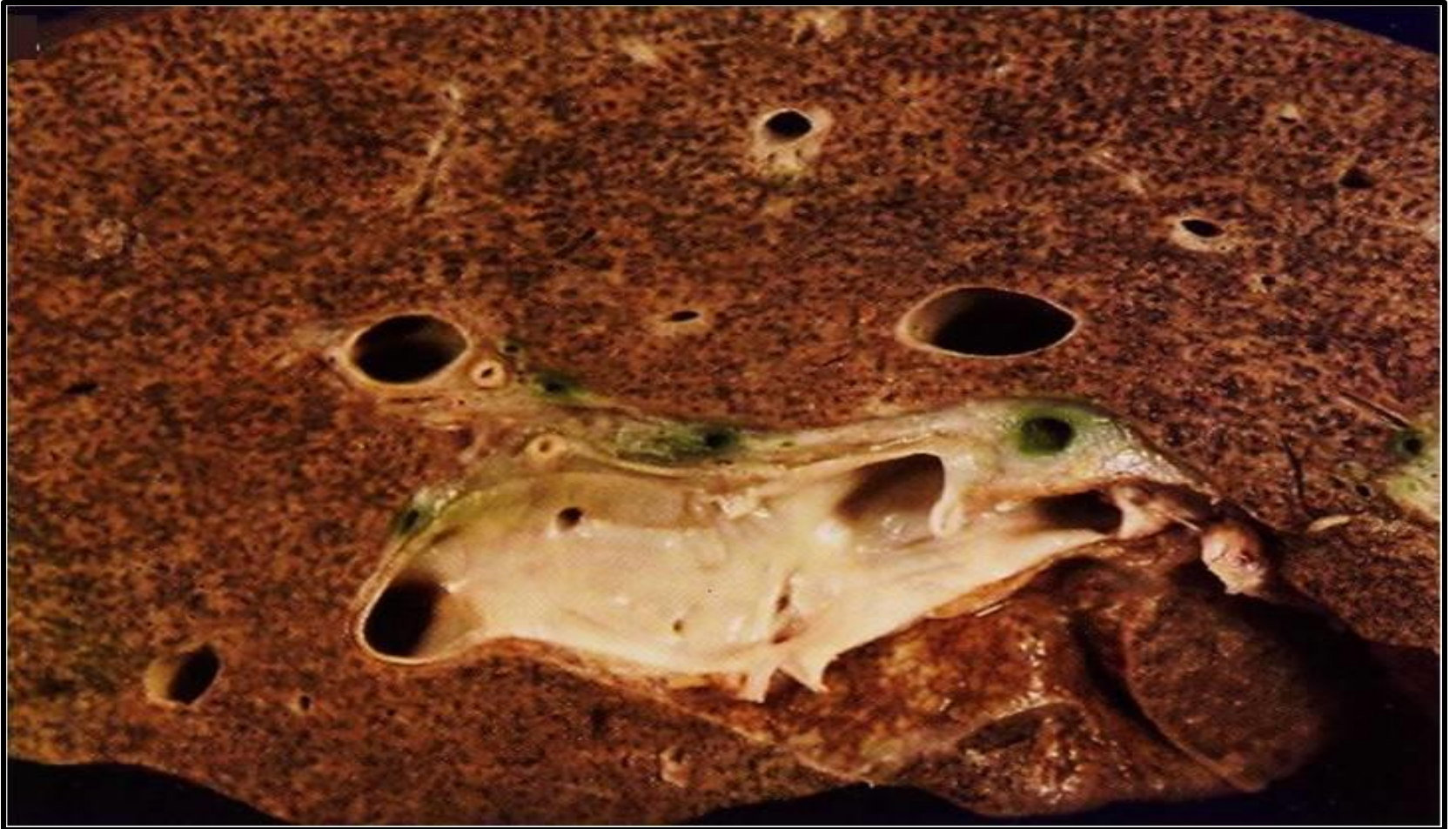
Several appear here as large cells with two or more nuclei that have prominent nucleoli.

HEART FAILURE

Right Sided Heart Failure

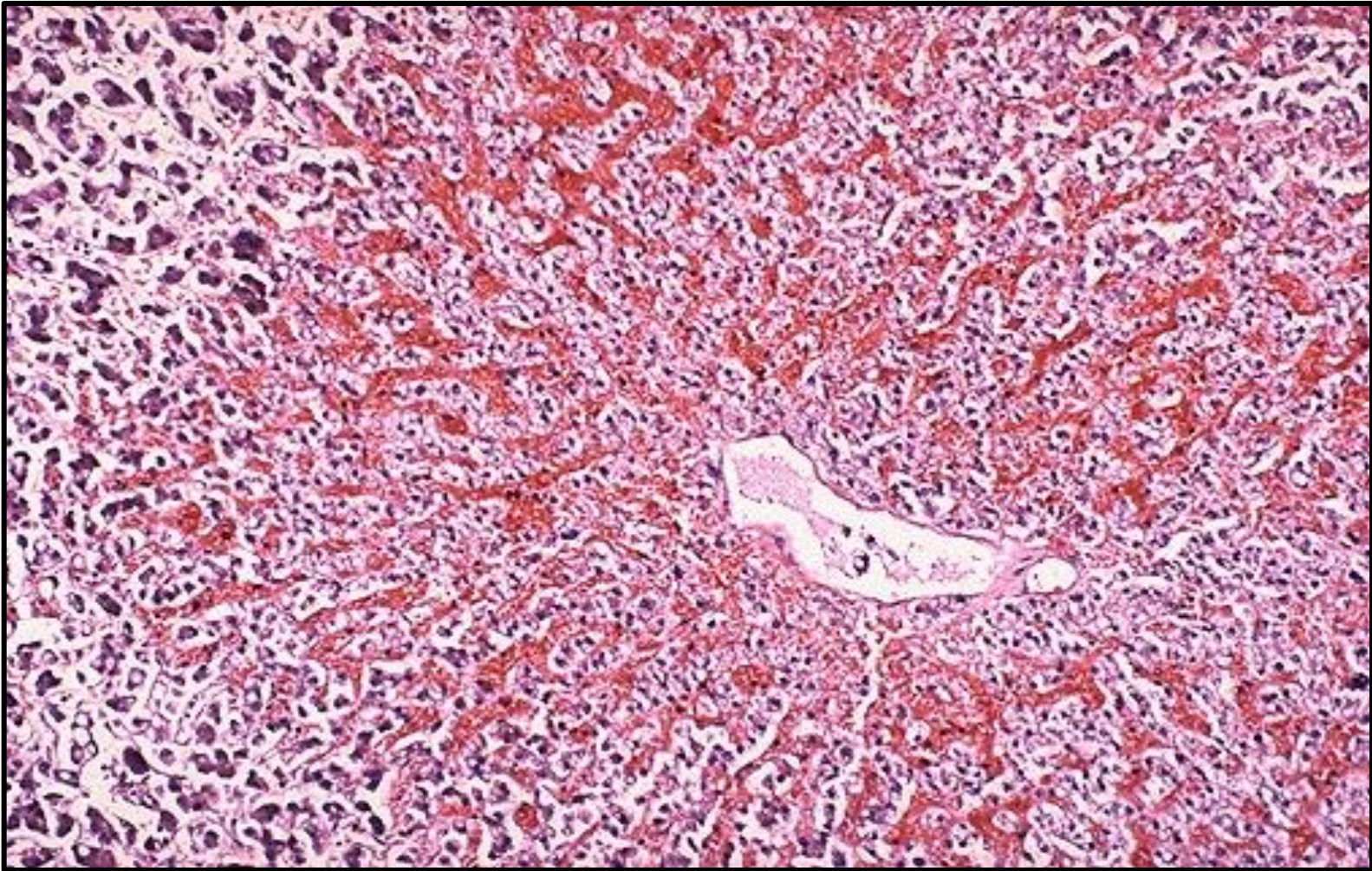
***Chronic venous congestion
of the liver***

NUTMEG LIVER – Cut surface



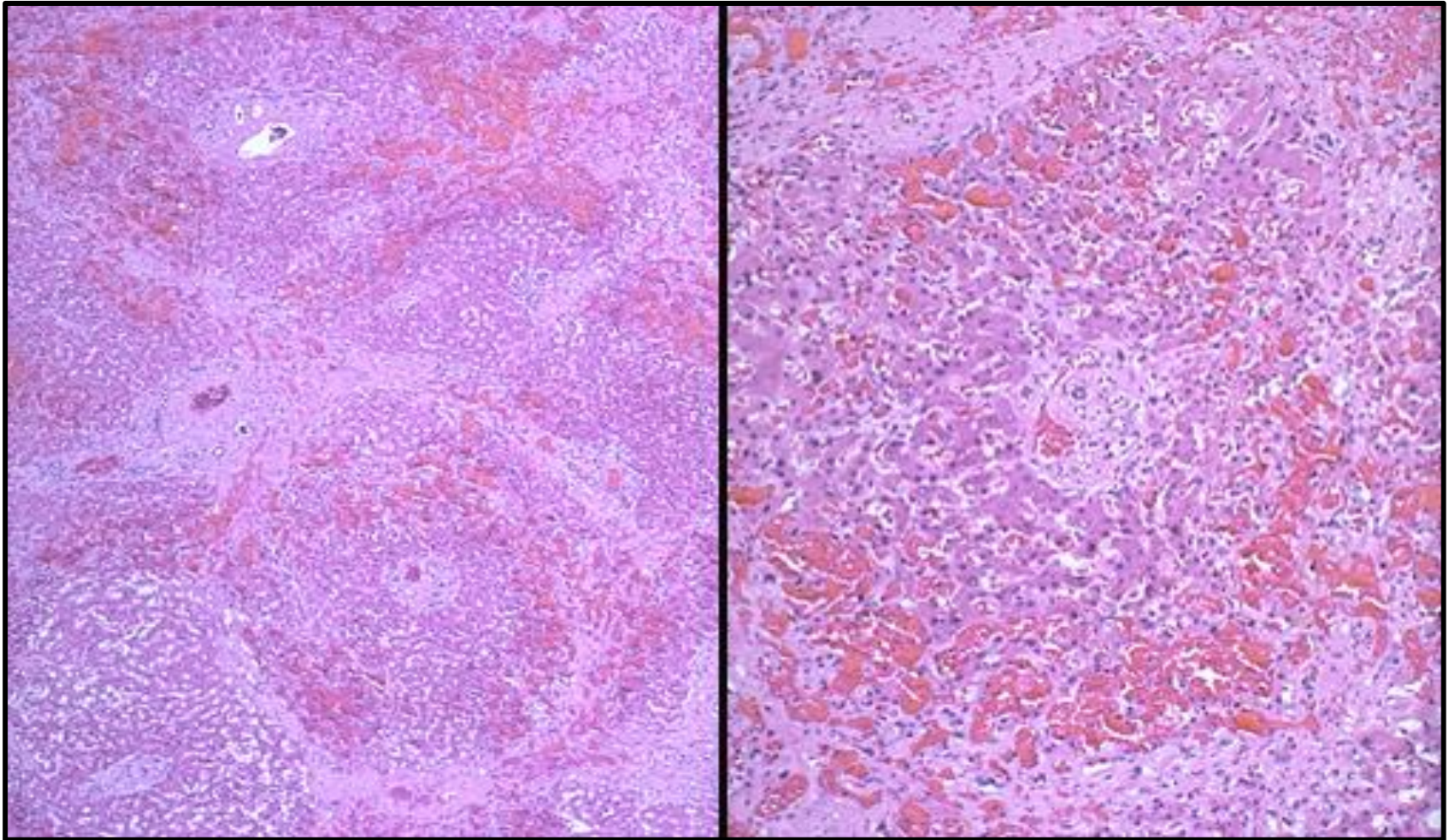
Section of liver showing alternating pale and dark areas with a nutmeg like appearance possibly due to passive congestion secondary to right sided heart failure.

Chronic Congestion of the Liver - LPF



The central portion of liver lobules shows congestion and dilatation of central veins and blood sinusoids, with atrophy and necrosis of liver cells.

Chronic Congestion of the Liver - LPF

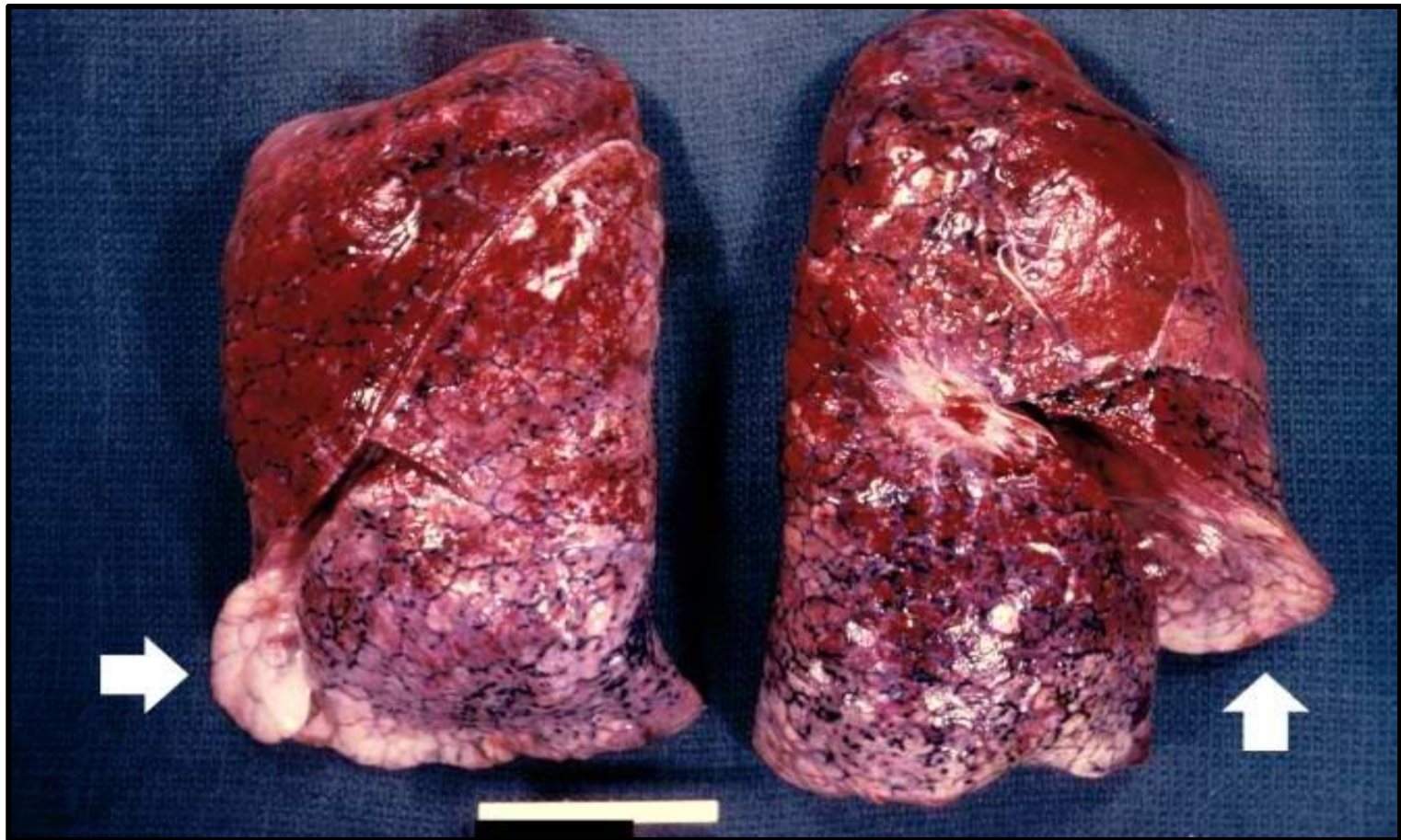


Central veins dilated and congested , necrotic hepatocytes

Left Sided Heart Failure

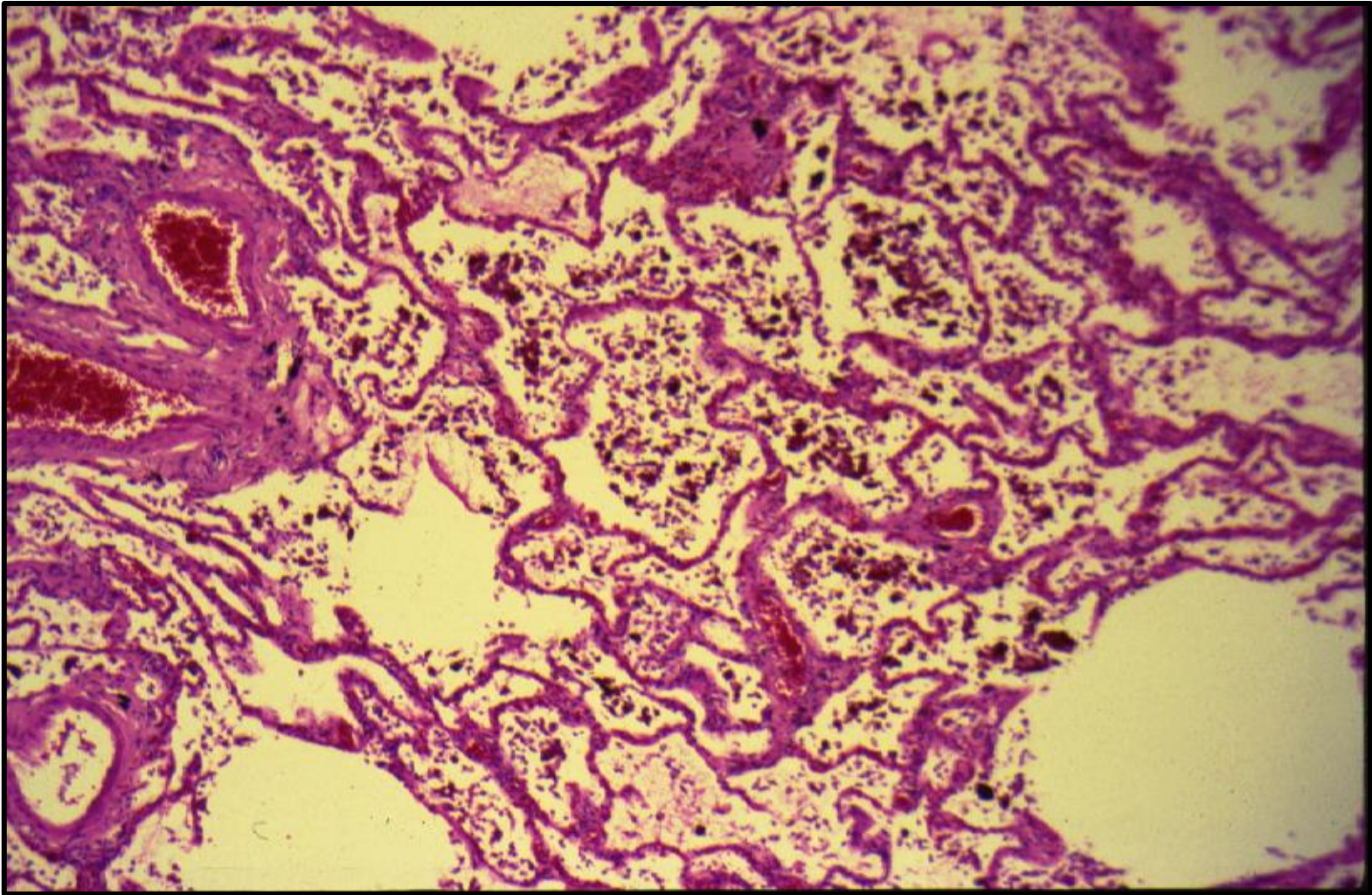
Chronic venous congestion of the lung

Chronic venous congestion of the lung - Gross



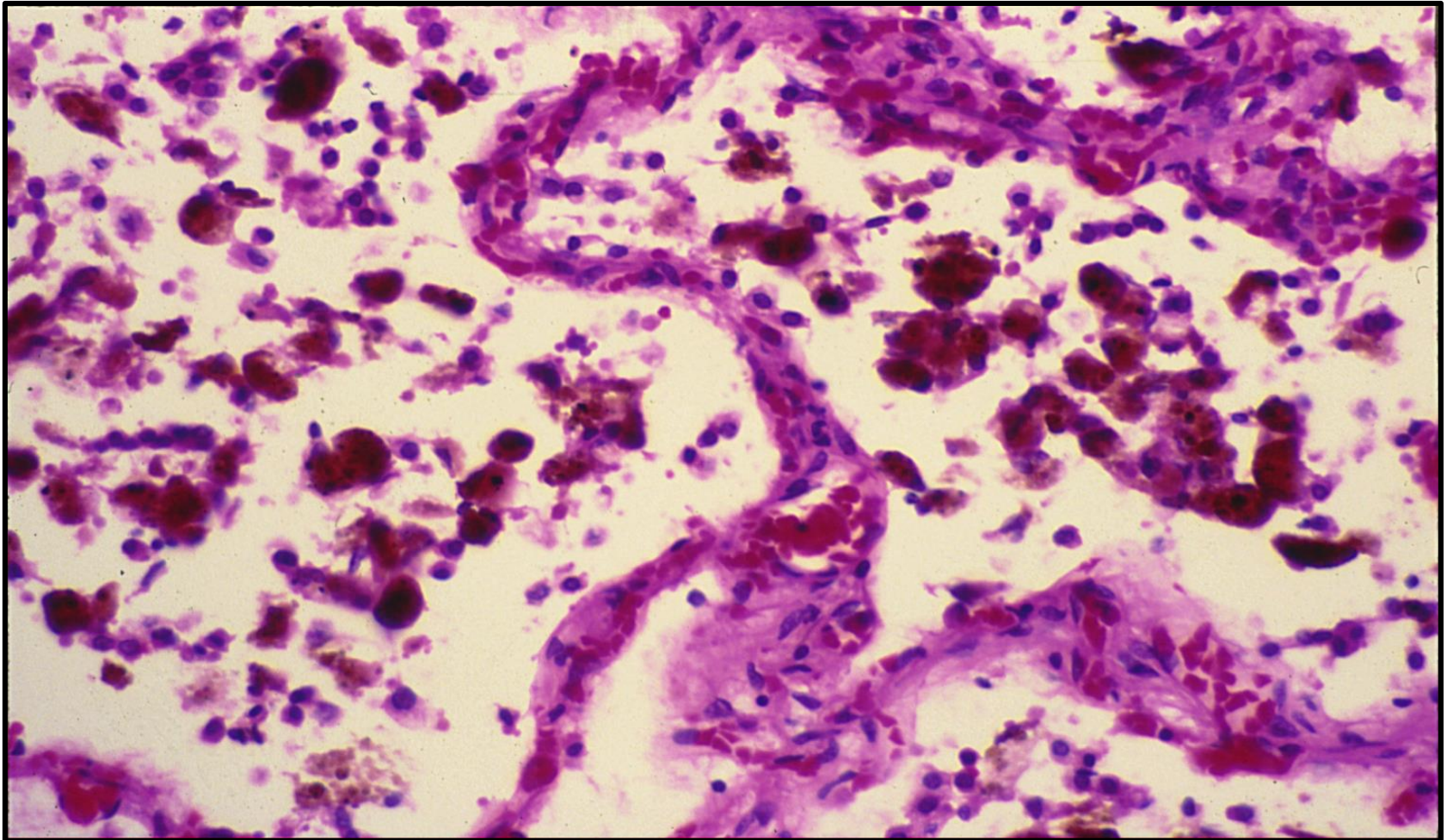
This is a gross photograph of lungs that are distended and red. The reddish coloration of the tissue is due to congestion. Some normal pink lung tissue is seen at the edges of the lungs (arrows).

Chronic venous congestion of the lung - LPF



The alveolar walls are thickened by dilated and engorged capillaries.

Chronic venous congestion of the lung - HPF



The alveoli contain edematous fluid, red blood cells and large alveolar macrophages (heart failure cells), which are filled with hemosiderin pigment derived from red cells breakdown.

PRACTICAL - 2

MYOCARDIAL HYPERTROPHY

The ventricle is working against high pressure, or “pumping” higher than normal volume leading to myocardial hypertrophy.

Causes of ventricular hypertrophy

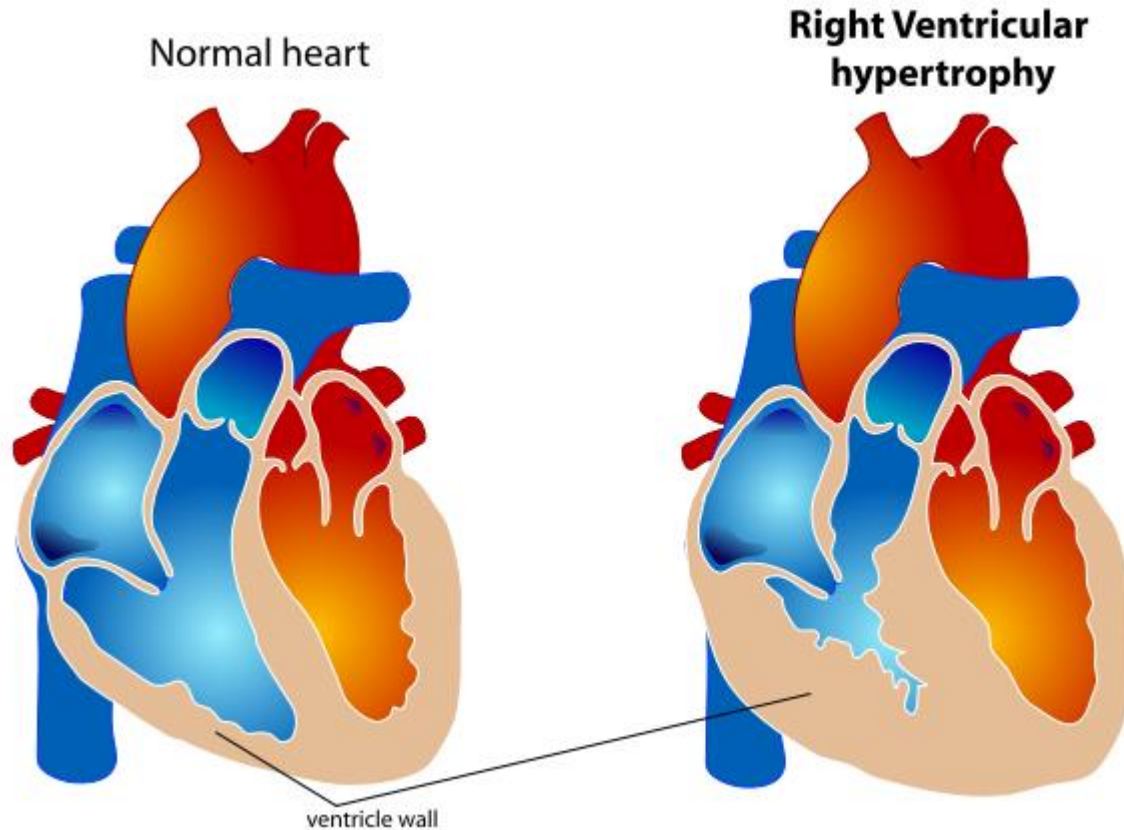
Left ventricular hypertrophy :

- **Systemic hypertension**
- **Aortic valve stenosis**

Right ventricular hypertrophy:

- **Pulmonary hypertension**
 - **asthma, COPD**
 - **pulmonary thromboembolic disease**
 - **primary pulmonary hypertension**
- **Pulmonary valve stenosis**
- **Left-to-right shunts (volume overload)**

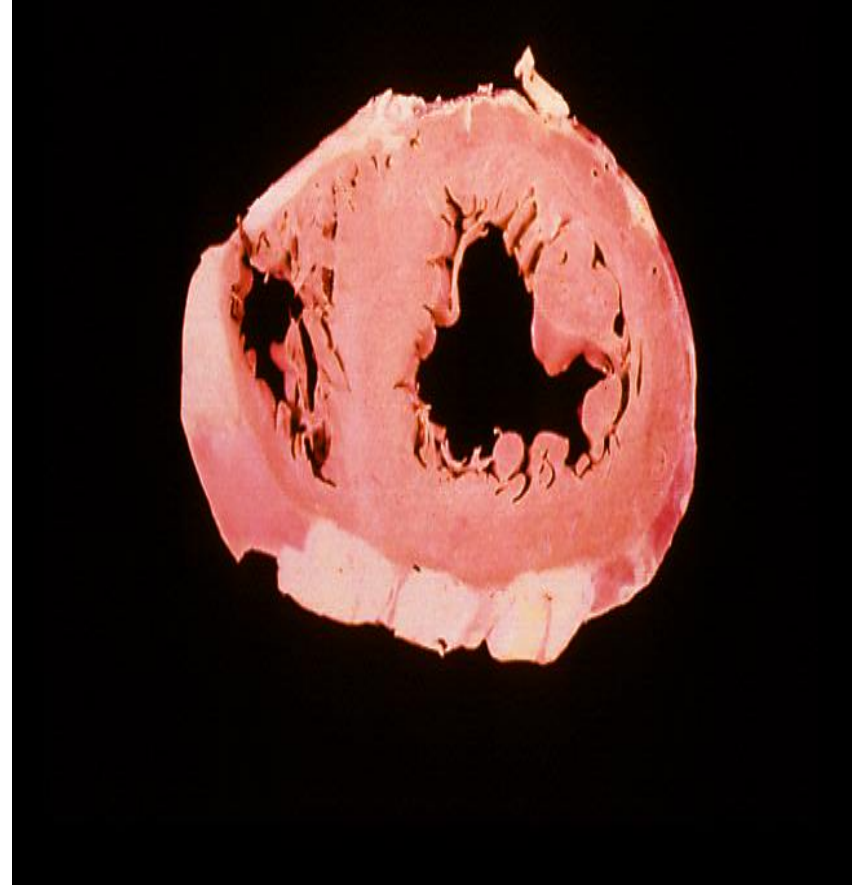
Right ventricular hypertrophy



Normal and hypertrophied left ventricle – cross section



Left ventricular hypertrophy



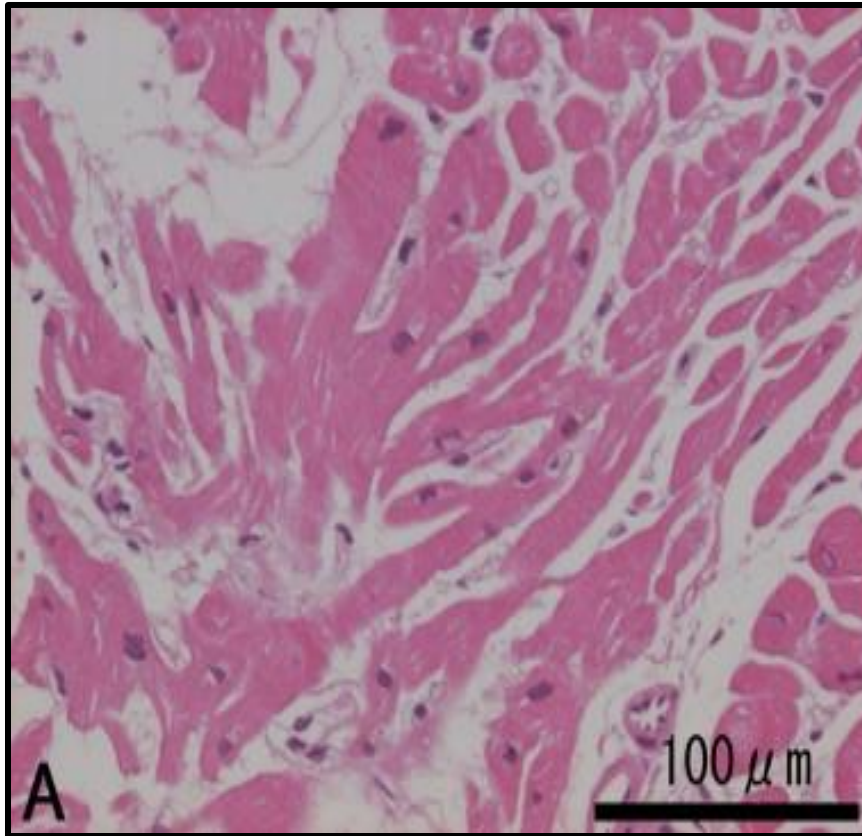
Normal ventricles

Left ventricular hypertrophy - Gross

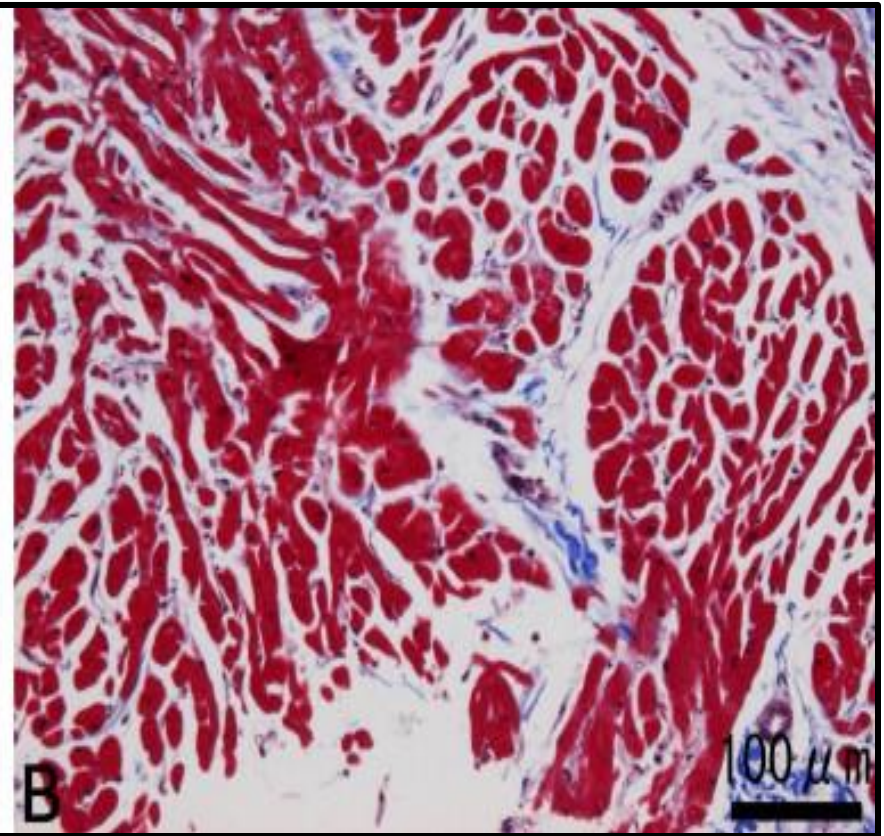


Heart from a hypertensive patient. The left ventricle is very thick (over 2 cm). However the rest of the heart is fairly normal in size as is typical for hypertensive heart disease. The hypertension creates a greater pressure load on the heart to induce the hypertrophy

Hypertrophic Cardiomyopathy - LPF



haematoxylin-eosin stain

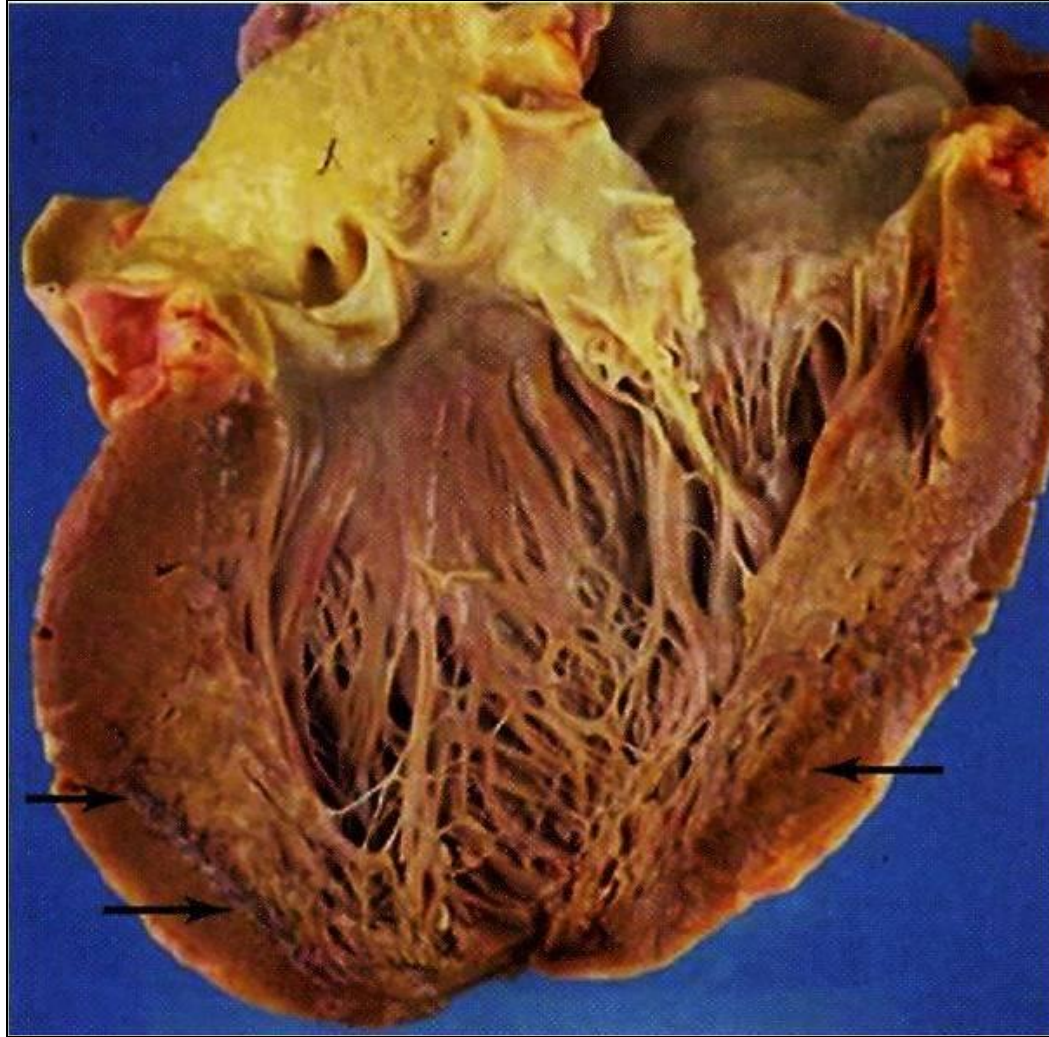


Masson's trichrome stain

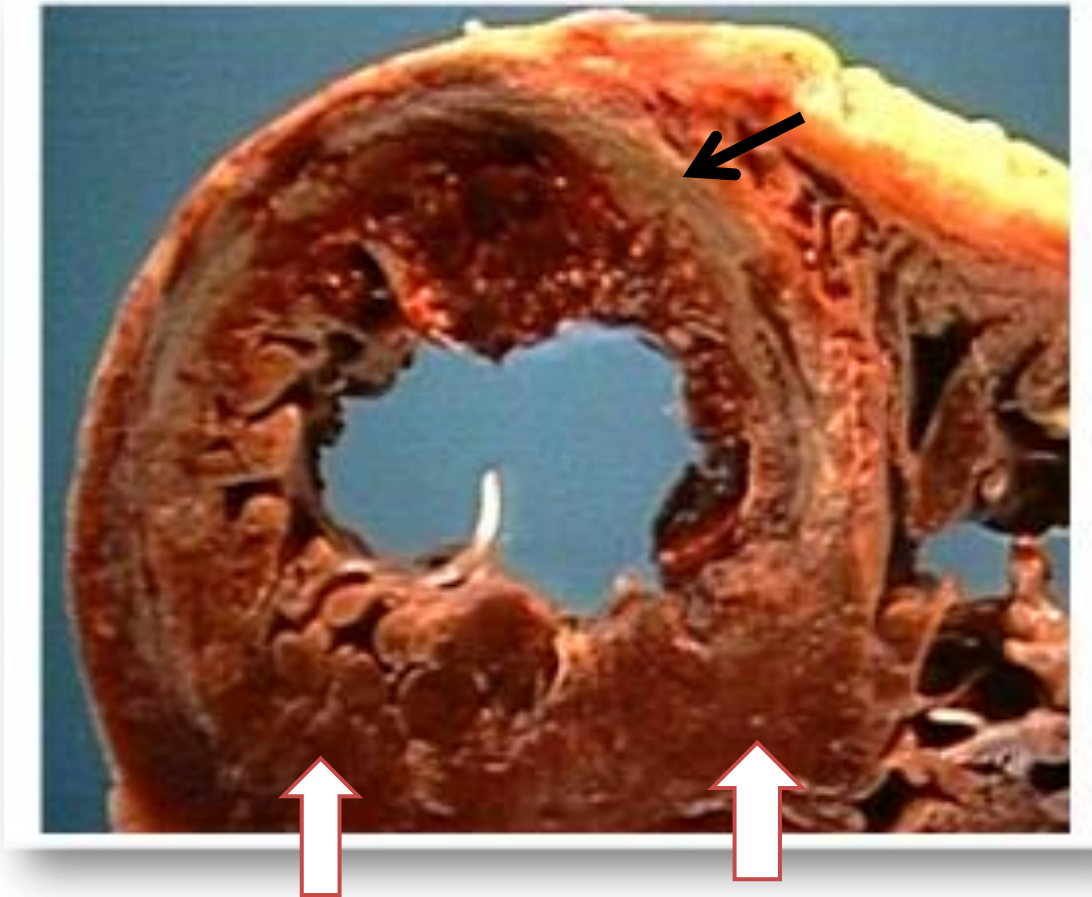
Histopathology of heart sections of ventricular septum showing significant *myofiber disarray* and slight interstitial fibrosis indicating hypertrophic cardiomyopathy (HCM).

MYOCARDIAL INFARCTION

Myocardial Infarction - CS



Myocardial Infarction - CS



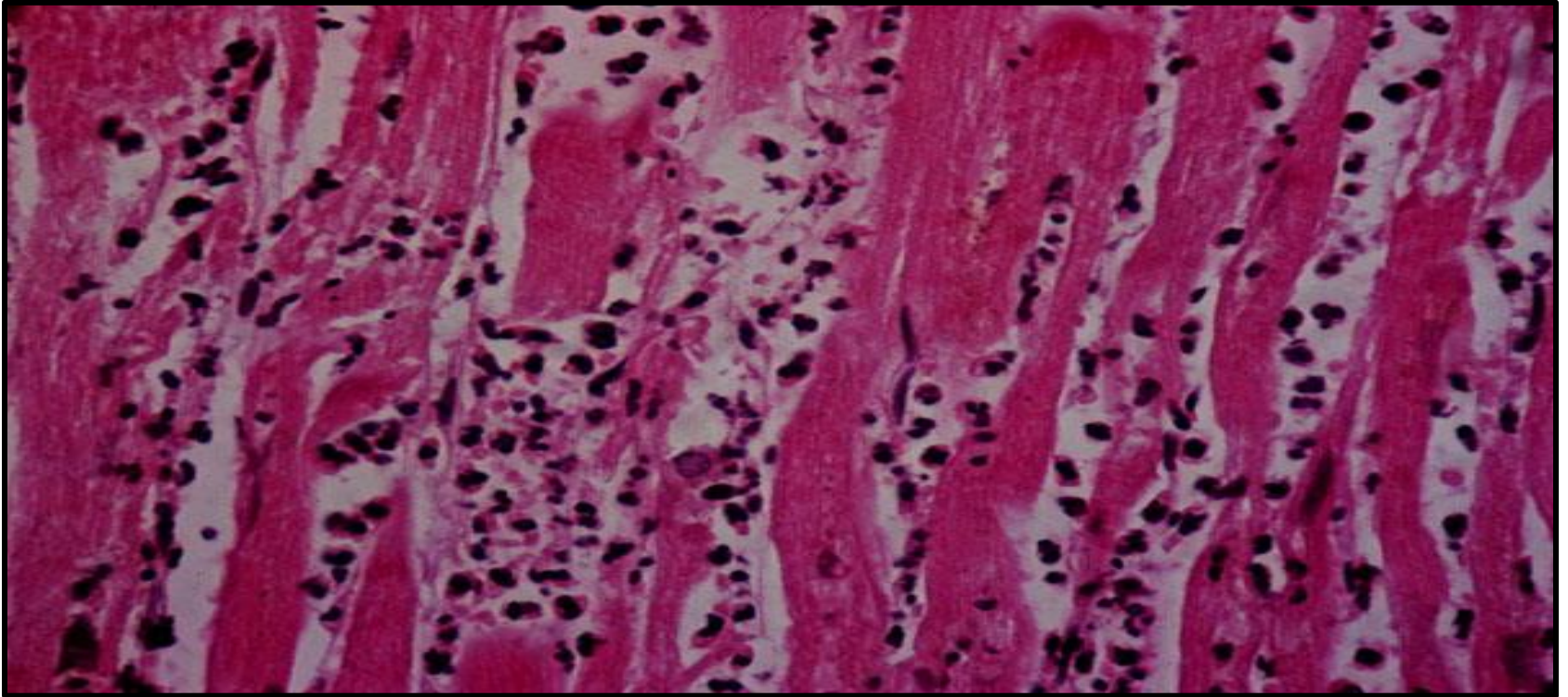
a- Pale and irregular myocardial fibrosis(Black arrow).

b- Thick left ventricular wall. (white arrow)

The cause of these pathology can be:

- Chronic ischemic heart disease.***
- Long standing hypertension and/or left ventricular failure.***

Acute Myocardial Infarction



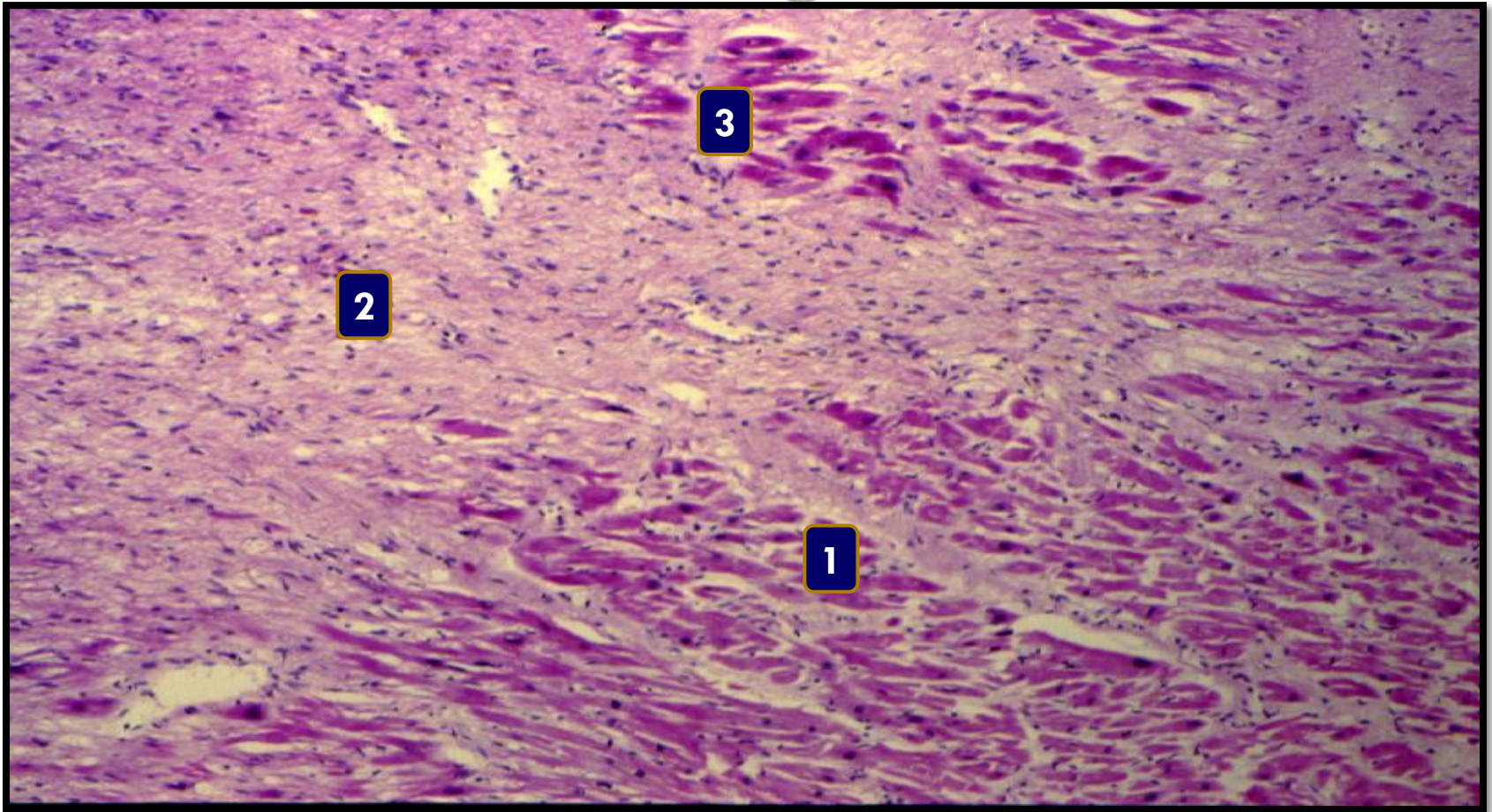
This 3-4 day old infarct showing:

a- Necrotic myocardial fibers.

b- Infiltration by polymorphonuclear leukocytes.

Death in this patient can be due to **complications secondary to acute myocardial infarction.**

Myocardial Infarction – late stage



- 1- **Patchy coagulative necrosis of myocardial fibers.** The dead muscle fibers are structureless and hyaline with loss of nuclei and striations.
- 2- **Chronic ischemic fibrous scar** replacing dead myocardial fibers .
- 3- **The remaining myocardial fibers** show enlarged nuclei due to ventricular hypertrophy .

THROMBOEMBOLISM / VASCULITIS

***Thromboangitis obliterans
(Buerger's disease)***

Thromboangitis obliterans (Buerger's disease)



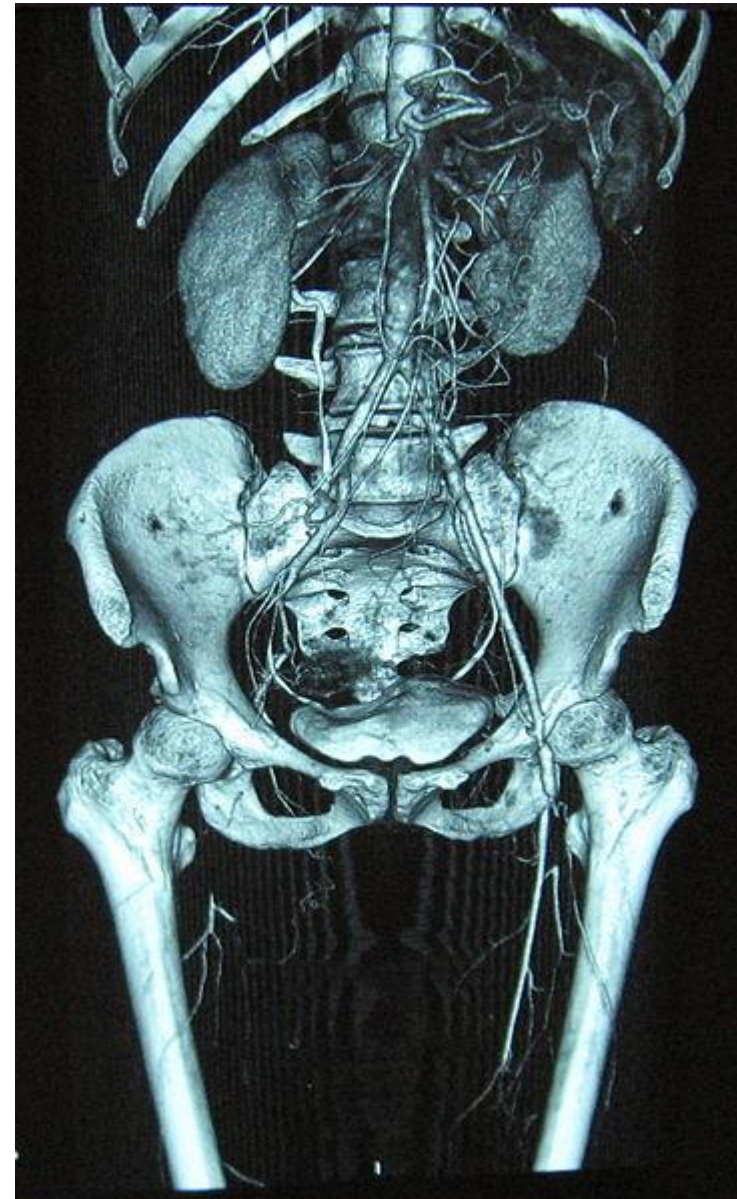
Black discoloration of the patient's finger and toes caused by ischaemia

The main **predisposing factors** for this condition are:

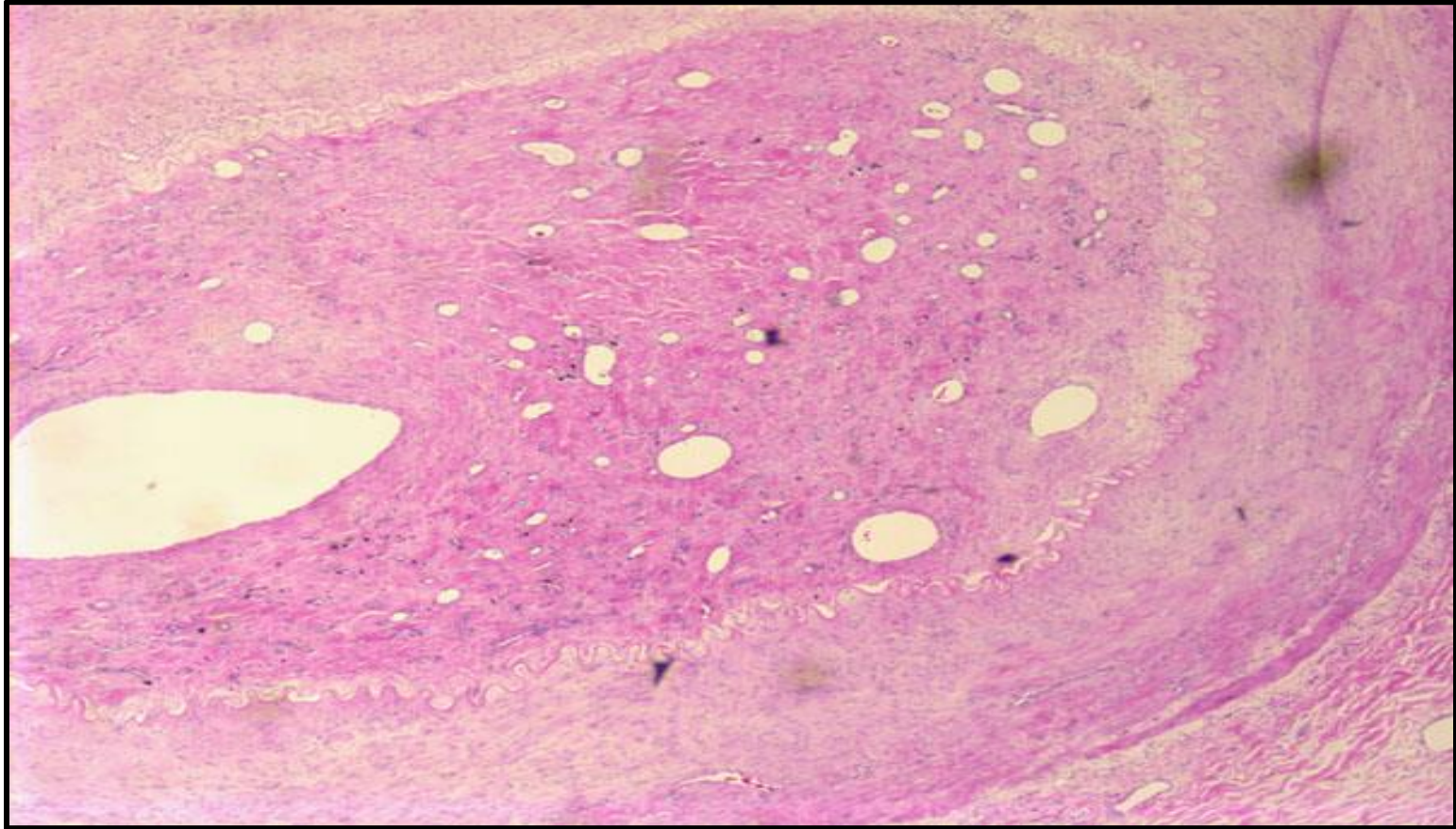
- **Smoking habits.**
- **Certain HLA haplotypes (Genetic predisposition).**

THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE)

- **Pathologic findings of an acute inflammation and thrombosis (clotting) of arteries and veins of the hands and feet (the lower limbs being more common)**
- **Complete occlusion of the right and stenosis of the left femoral artery**

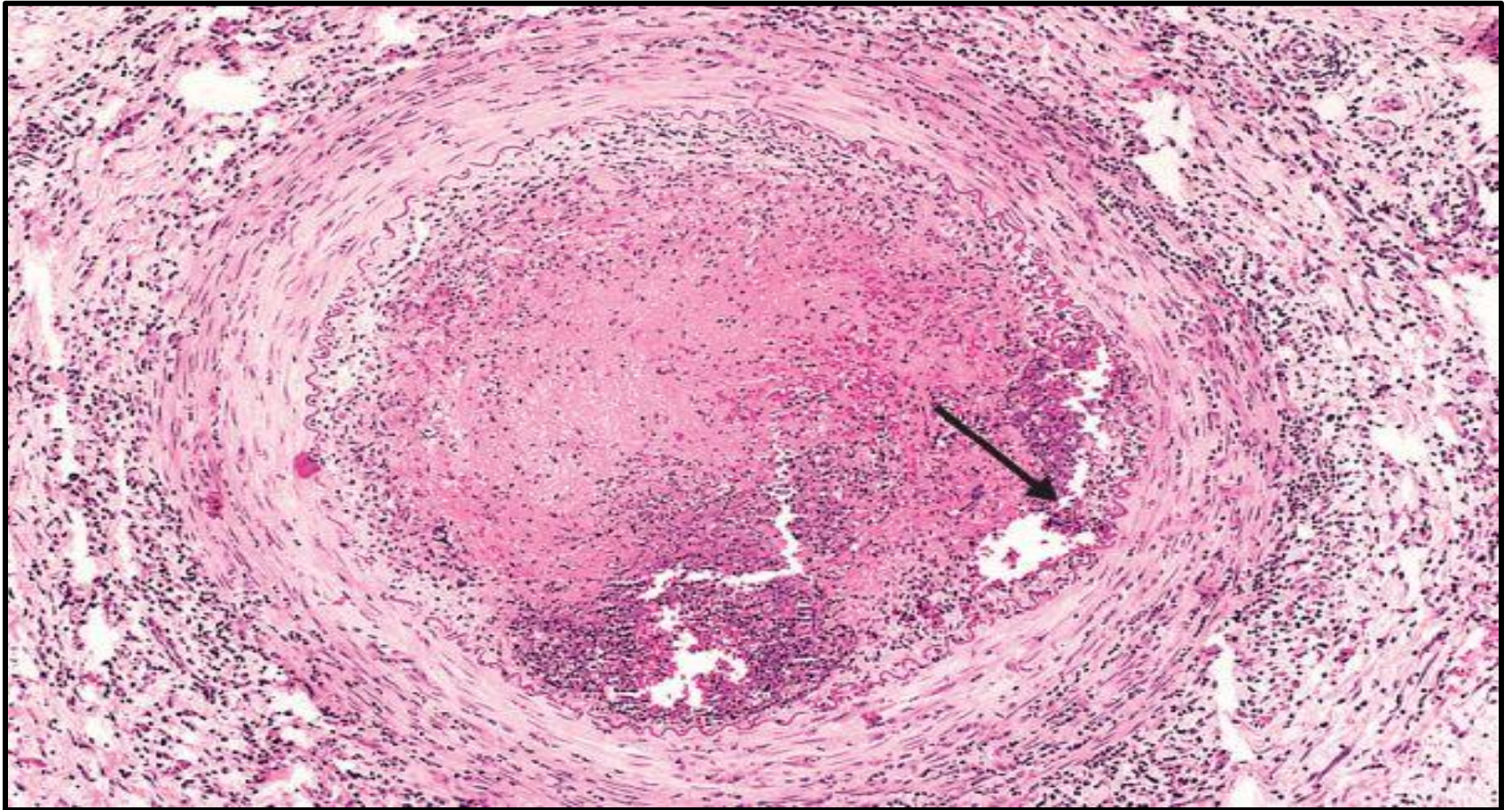


THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE) - LPF



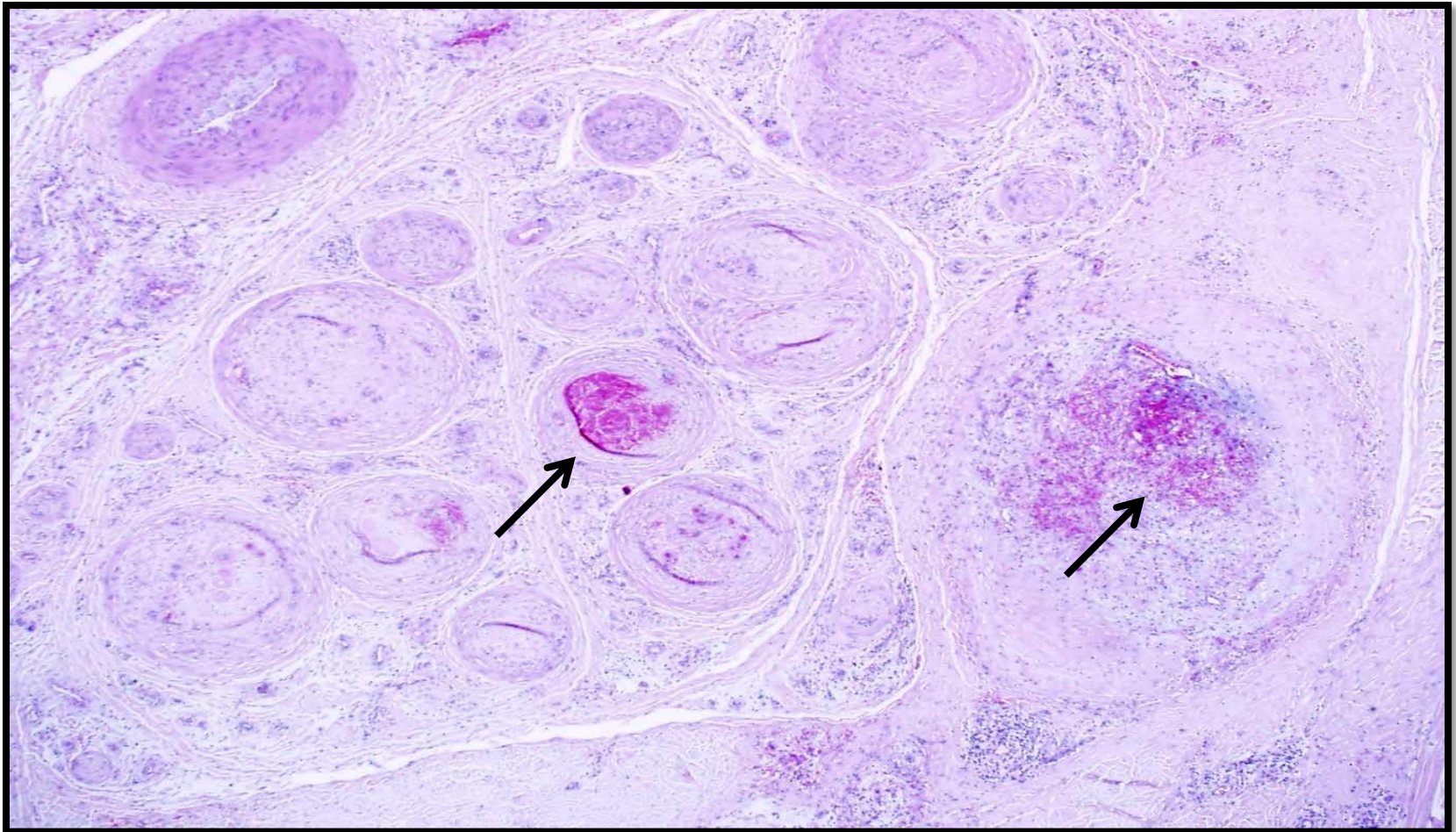
*Thromboangiitis obliterans (Buerger's disease) is a **non atherosclerotic**, **segmental**, **inflammatory**, **vaso-occlusive** disease that affects the **small- and medium-sized arteries and veins** of the upper and lower extremities.*

THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE) - HPF



- The lumen is occluded by a thrombus in lumen (arrow)*
- The vessel wall is infiltrated with leukocytes.*

THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE) - HPF



Some blood vessels show recent organizing thrombi while others show infiltration of the wall and surrounding tissue by chronic inflammatory cells

GIANT CELL (TEMPORAL) ARTERITIS

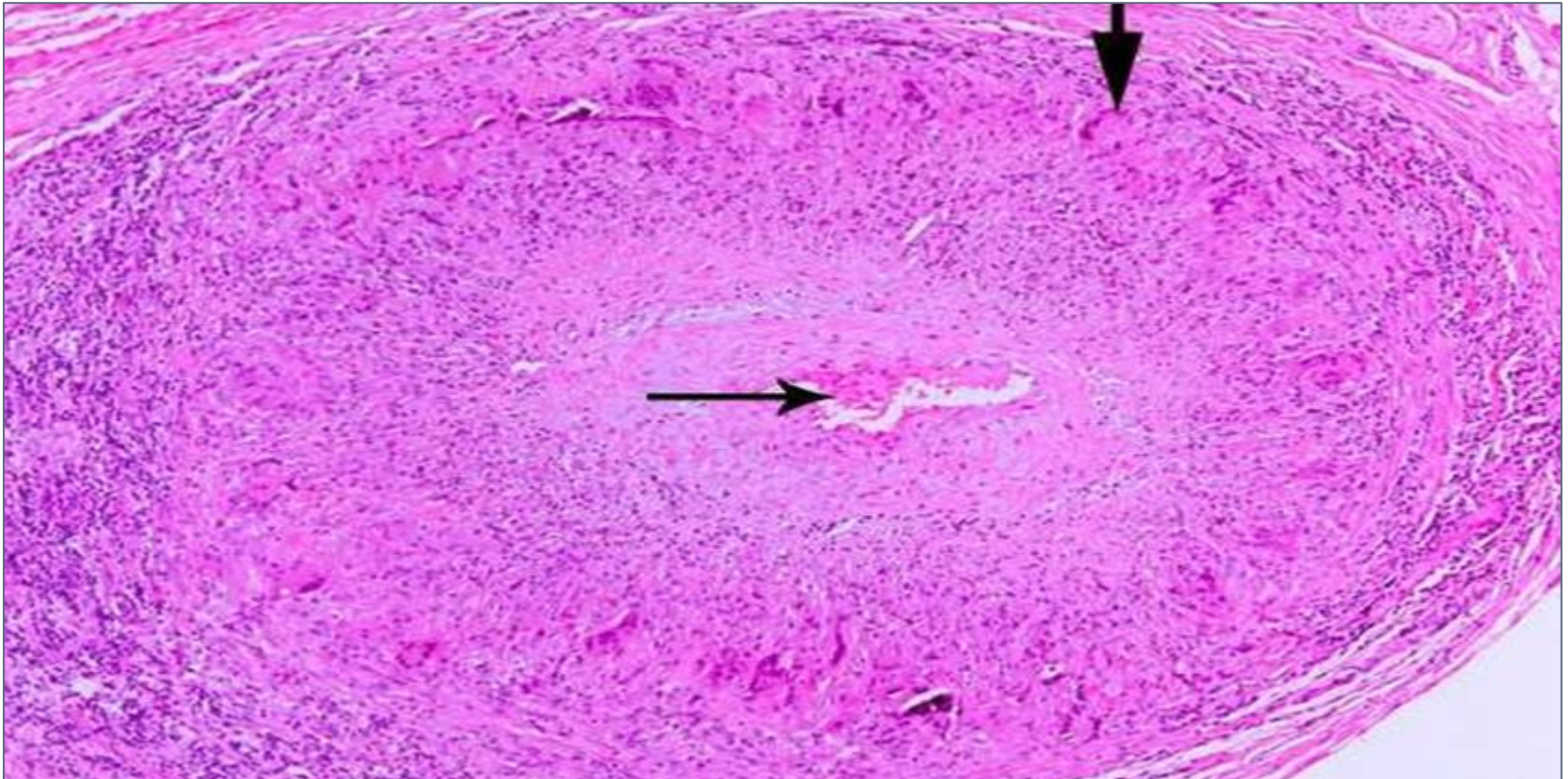
GIANT CELL / TEMPORAL ARTERITIS



Tender and thickened temporal artery

- ***Elevated erythrocytes sedimentation rate (ESR) is raised in these patients.***
- ***Complication: Blindness because of involvement of ophthalmic artery.***

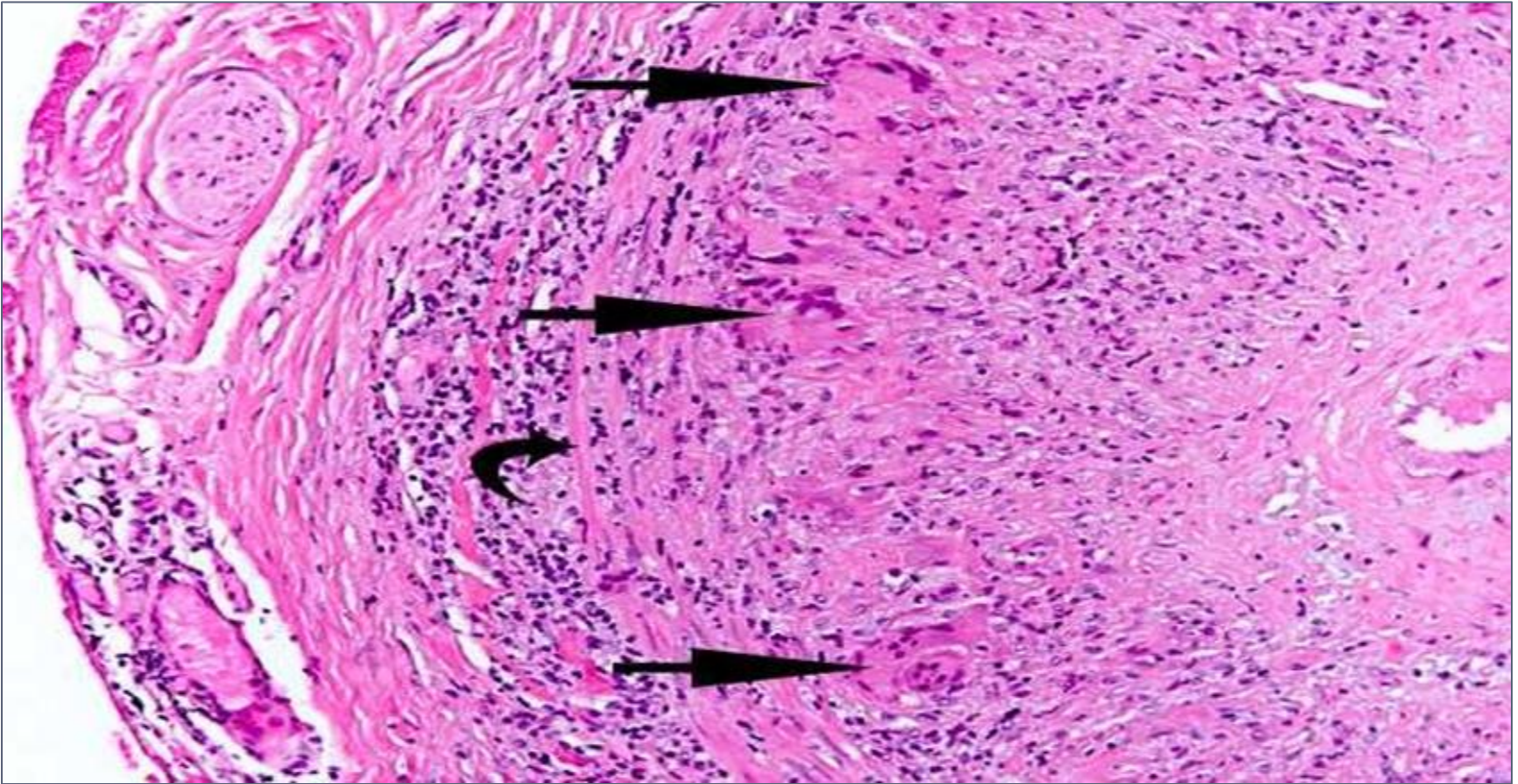
GIANT CELL / TEMPORAL ARTERITIS - LPF



*Circumferential involvement of the **vascular media** is present (vertical arrow pointing downward). Also note the presence of chronic lymphocytic inflammation in the media and adventitia.*

Reactive intimal fibroplasias lead to luminal stenosis with $<10\%$ of its original luminal diameter (thin arrow in the center).

GIANT CELL / TEMPORAL ARTERITIS - HPF



Giant cells can be of Langhans type or foreign-body type (three arrows) and may show **fragments of disrupted internal elastic lamina**..

GIANT CELL (TEMPORAL) ARTERITIS - HPF



- **Chronic inflammation.**
- **Giant cells.**
- **Fragmentation of the vascular internal elastic lamina.**
- **Granulomatous inflammation.**

***LEUKOCYTOCLASTIC VASCULITIS /
HYPERSENSITIVITY VASCULITIS /
MICROSCOPIC POLYANGITIS /
HENOCH- SCHÖNLEIN PURPURA.***

Hypersensitivity vasculitis – Clinical sign



Hypersensitivity vasculitis might be complicated with glomerulonephritis and hemoptysis due to pulmonary capillaritis

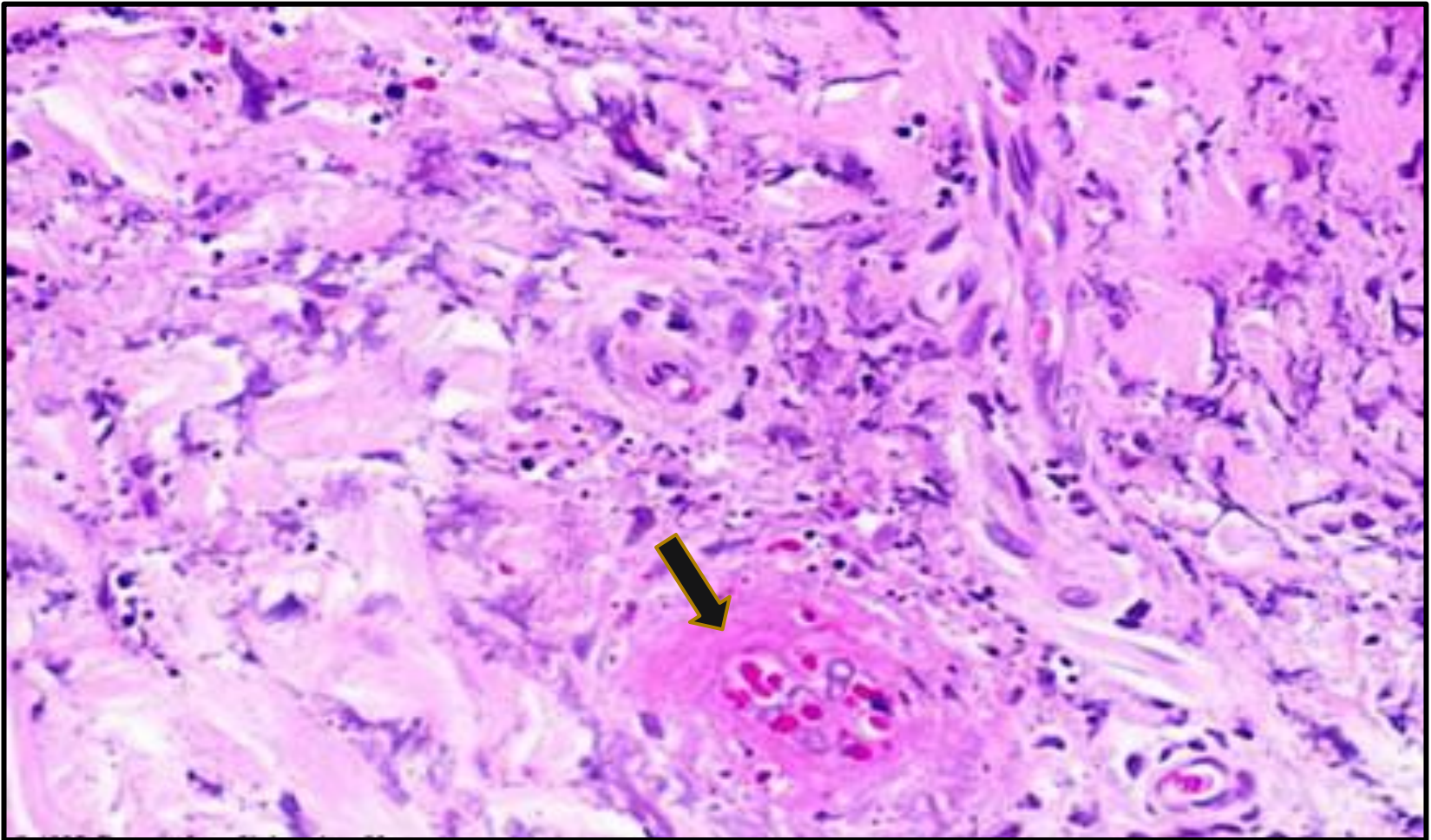
Leukocytoclastic vasculitis - Clinical sign



Leukocytoclastic vasculitis

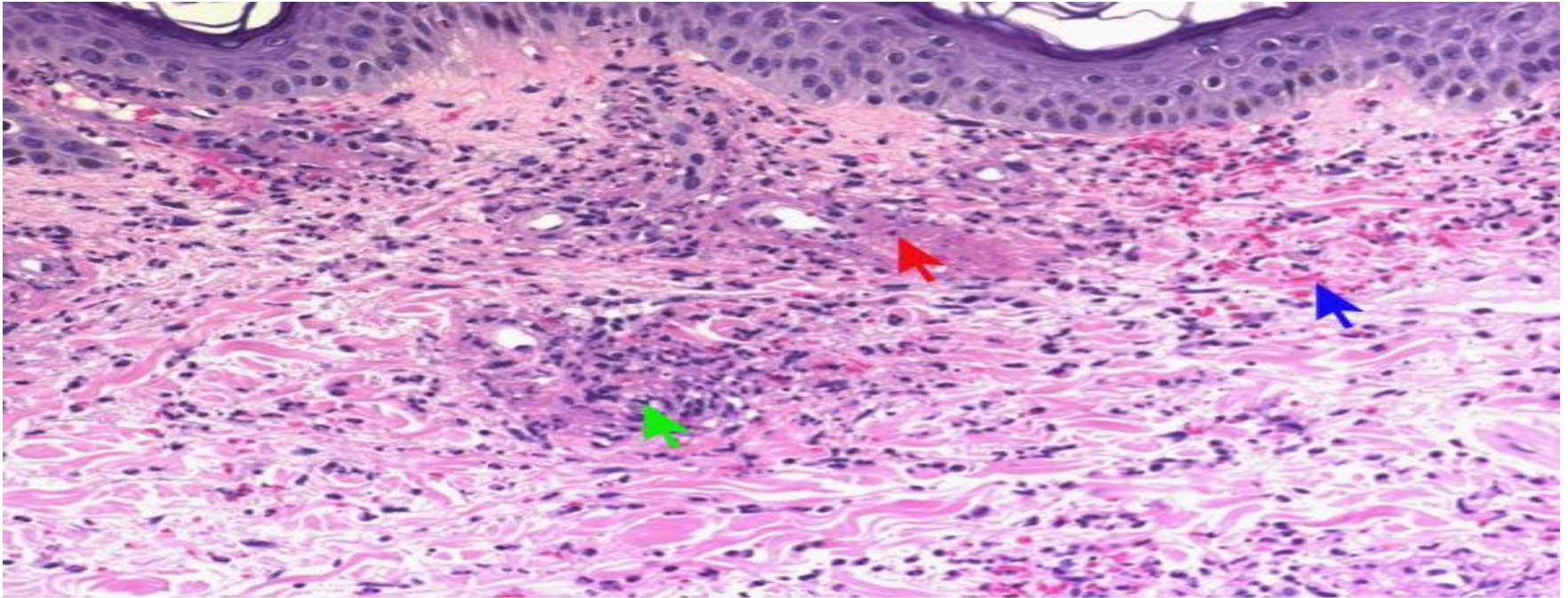
- ***Erythematous and purpuric skin rash affecting the right foot (Purpura:- Subcutaneous bleeding.)***




Leukocytoclastic vasculitis - HPF



- ***-Fibrinoid vascular necrosis.***
- ***- Nuclear debris.***
- ***- Neutrophilic (polymorphonuclear) infiltration.***

Leukocytoclastic vasculitis - HPF



-  **Fibrinoid type necrosis**
-  **Red cell extravasation**
-  **Inflammation**

Complications that might occur as a result of this condition.

- *Necrotizing Glomerulonephritis.*
- *Pulmonary capillaritis.*
- *Gastrointestinal vasculitis.*
- *CNS and muscle involvement.*