CARDIOVASCULAR SYSTEM

Pathology Practical

Presented by:
Dr. Malak Alzahrani

Prepared by:
- *Prof. Ammar Al Rikabi*
- *Dr. Sayed Al Esawy*

PRACTICAL – 2

A: MYOCARDIAL DISEASE:

- HYPERTROPHY
- MYOCARDIAL INFARCTION

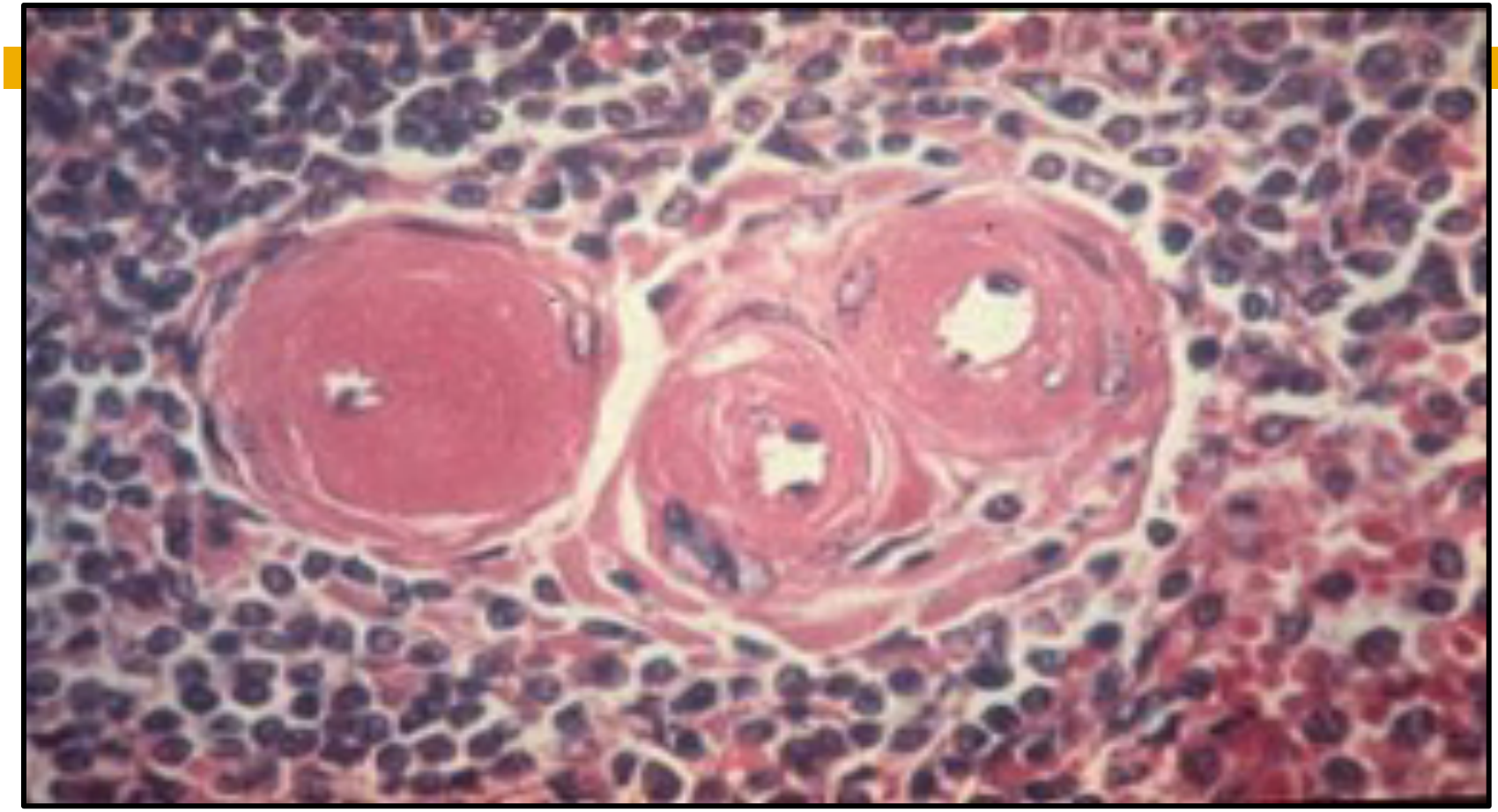
B: VASCULITIS

Morphology of blood vessels in HTN:

In small Blood Vessels (Microangiopathy)

- Arteriolosclerosis
  - Hyaline arteriolosclerosis:
    - Seen in benign hypertension
    - Can also be seen in elderly and diabetic patients even without hypertension.
    - Can cause diffuse renal ischemia.
  - Hyperplastic arteriolosclerosis:
    - Characteristic of malignant hypertension.
    - Can show onion-skinning on histology causing luminal obliteration of vascular lumen

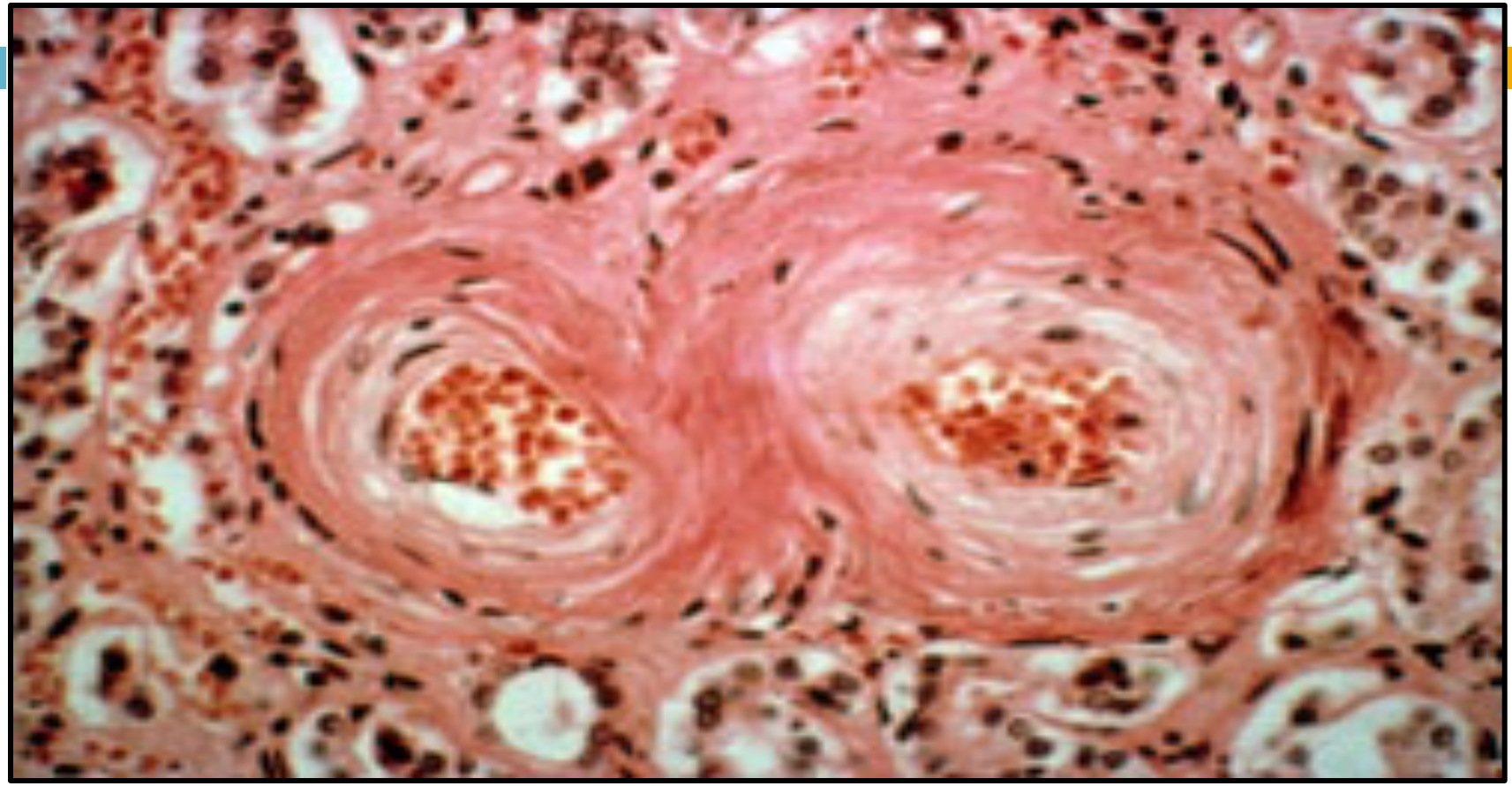
Hyaline arteriolosclerosis - HPF



Hyaline arteriolosclerosis

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

Hyperplastic arteriolosclerosis - HPF



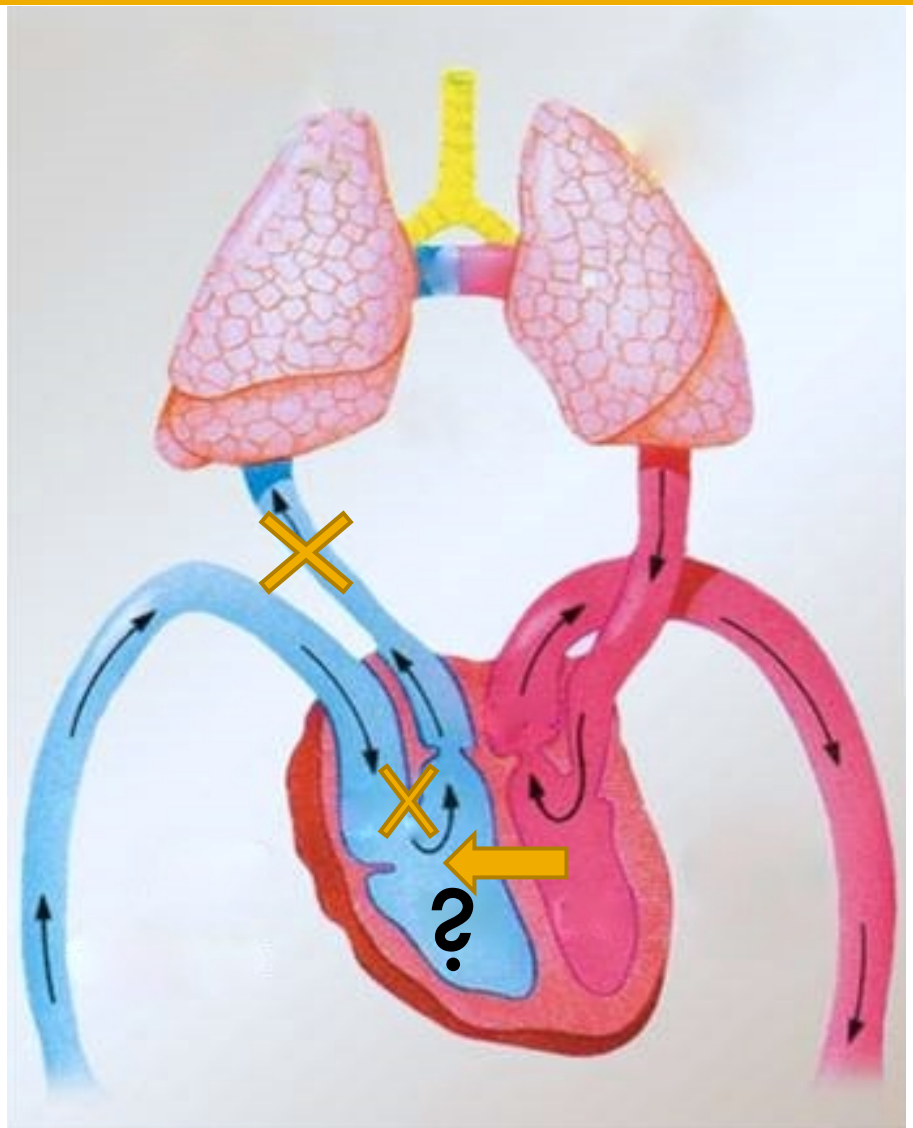
***Hyperplastic arteriolosclerosis**: This is the other type of small vessel arteriosclerosis. It is predominantly seen in **malignant** hypertension and renal disease associated with polyarteritis nodosa and progressive systemic sclerosis.*

MYOCARDIAL HYPERTROPHY

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

Left ventricular hypertrophy :

Right ventricular hypertrophy :



Causes of ventricular hypertrophy

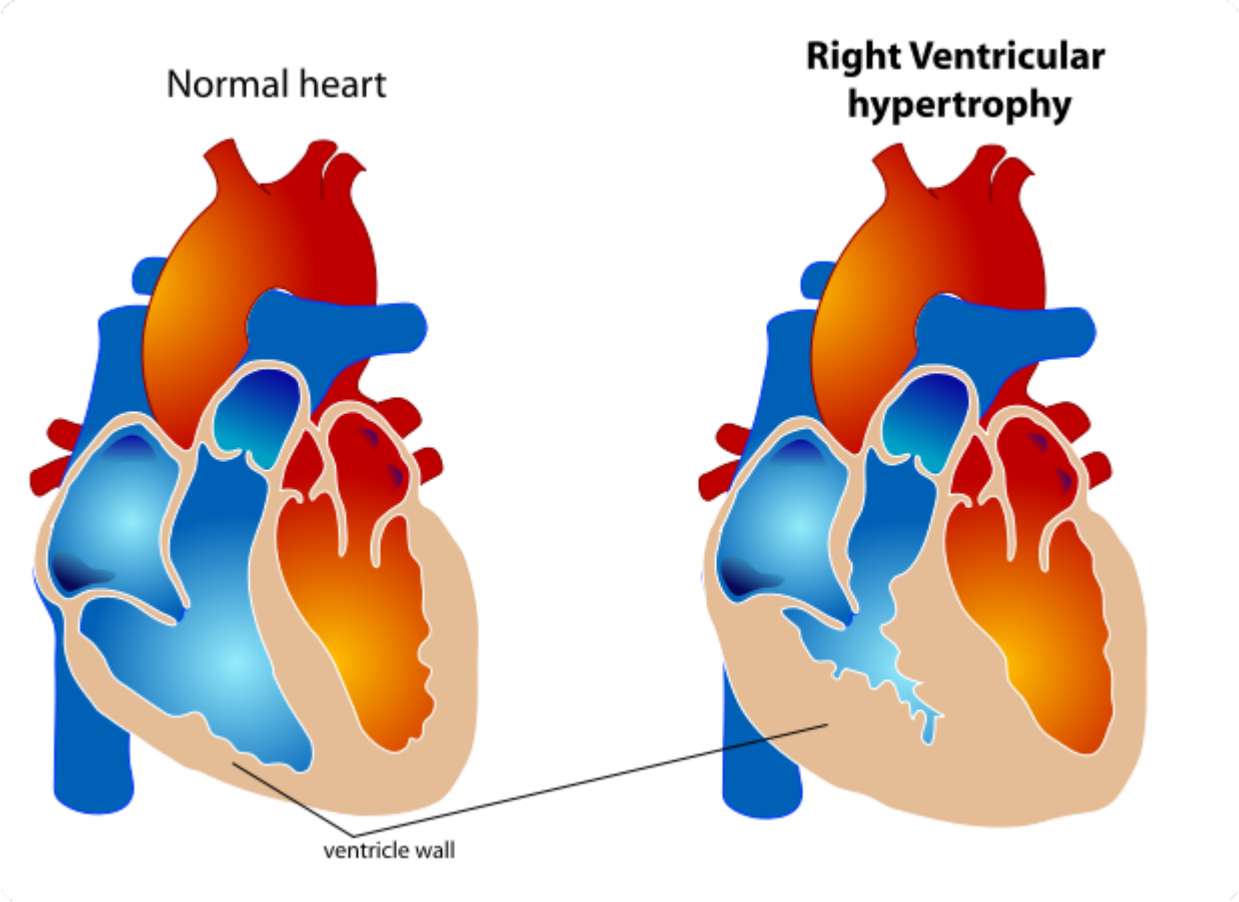
Left ventricular hypertrophy :

- Systemic hypertension
- Aortic valve stenosis

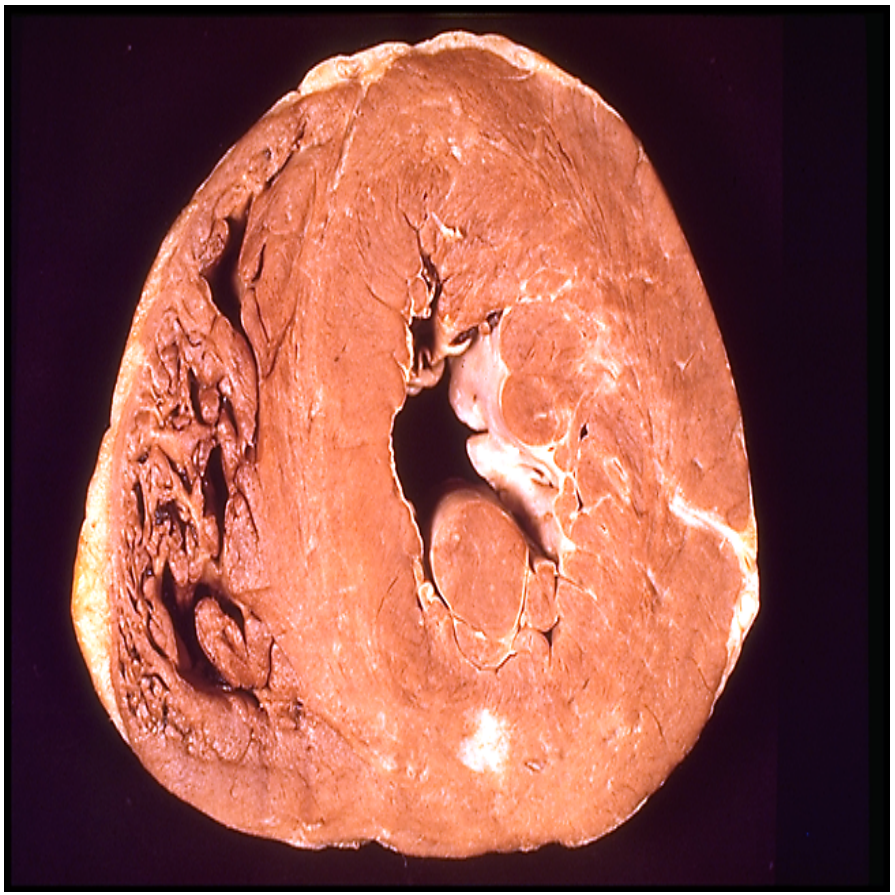
Right ventricular hypertrophy:

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

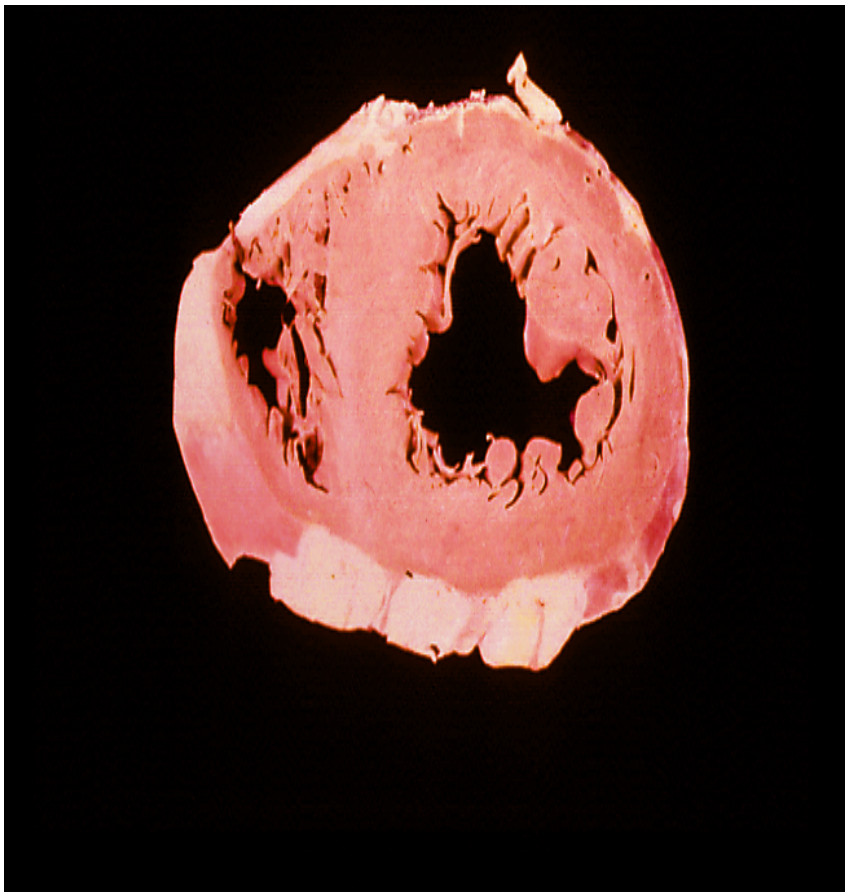
Right ventricular hypertrophy



Normal and hypertrophied left ventricle – cross section

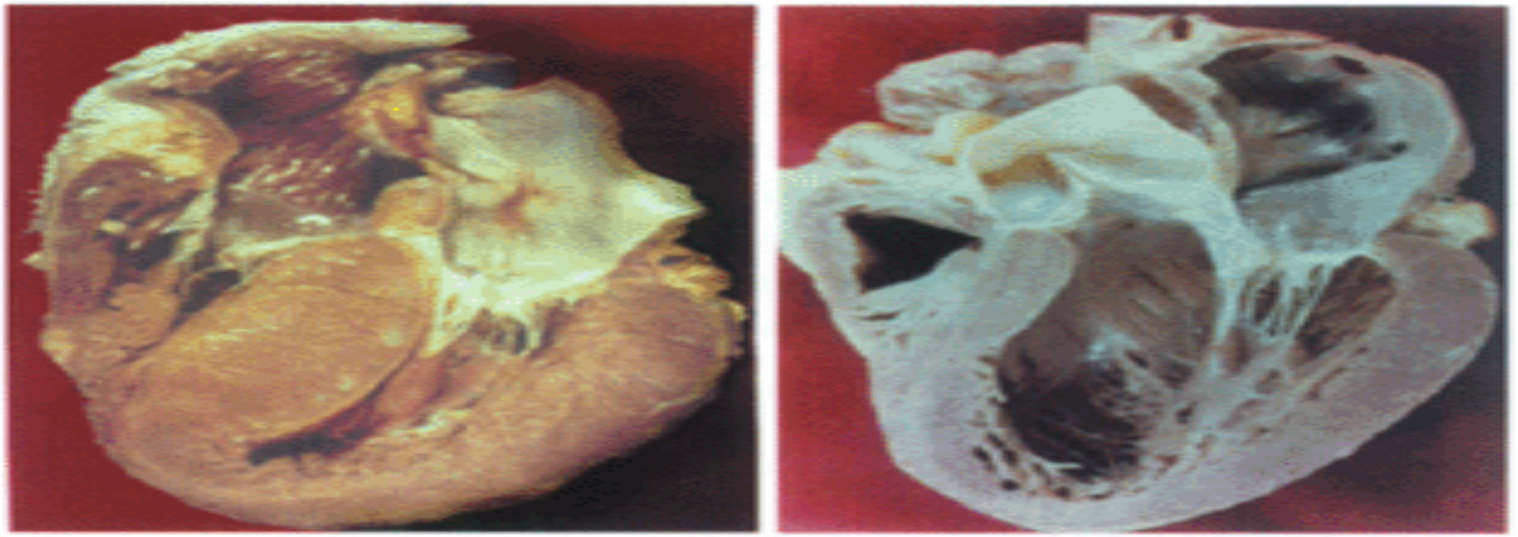


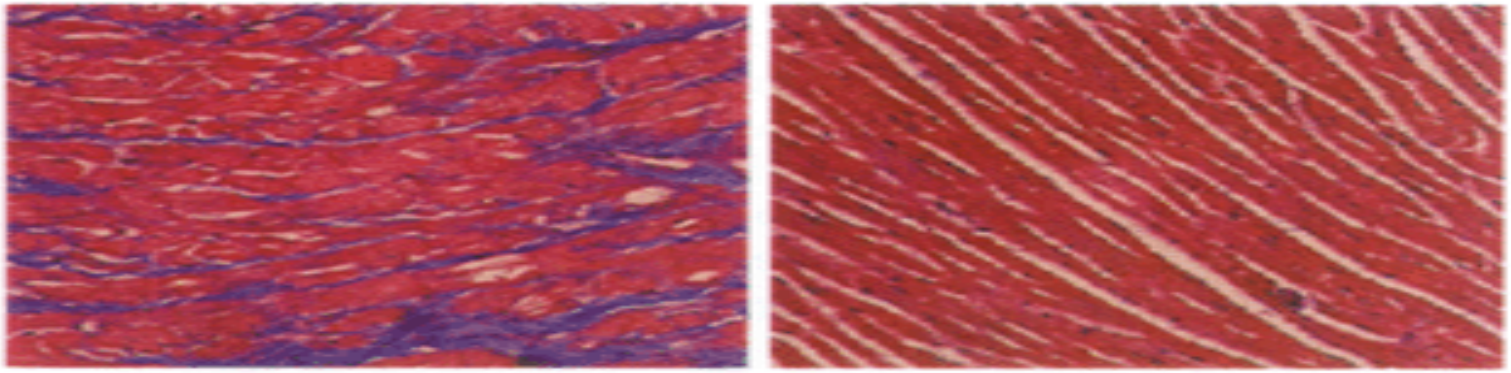
Left ventricular hypertrophy



Normal ventricles

Normal and hypertrophied left ventricle - CS





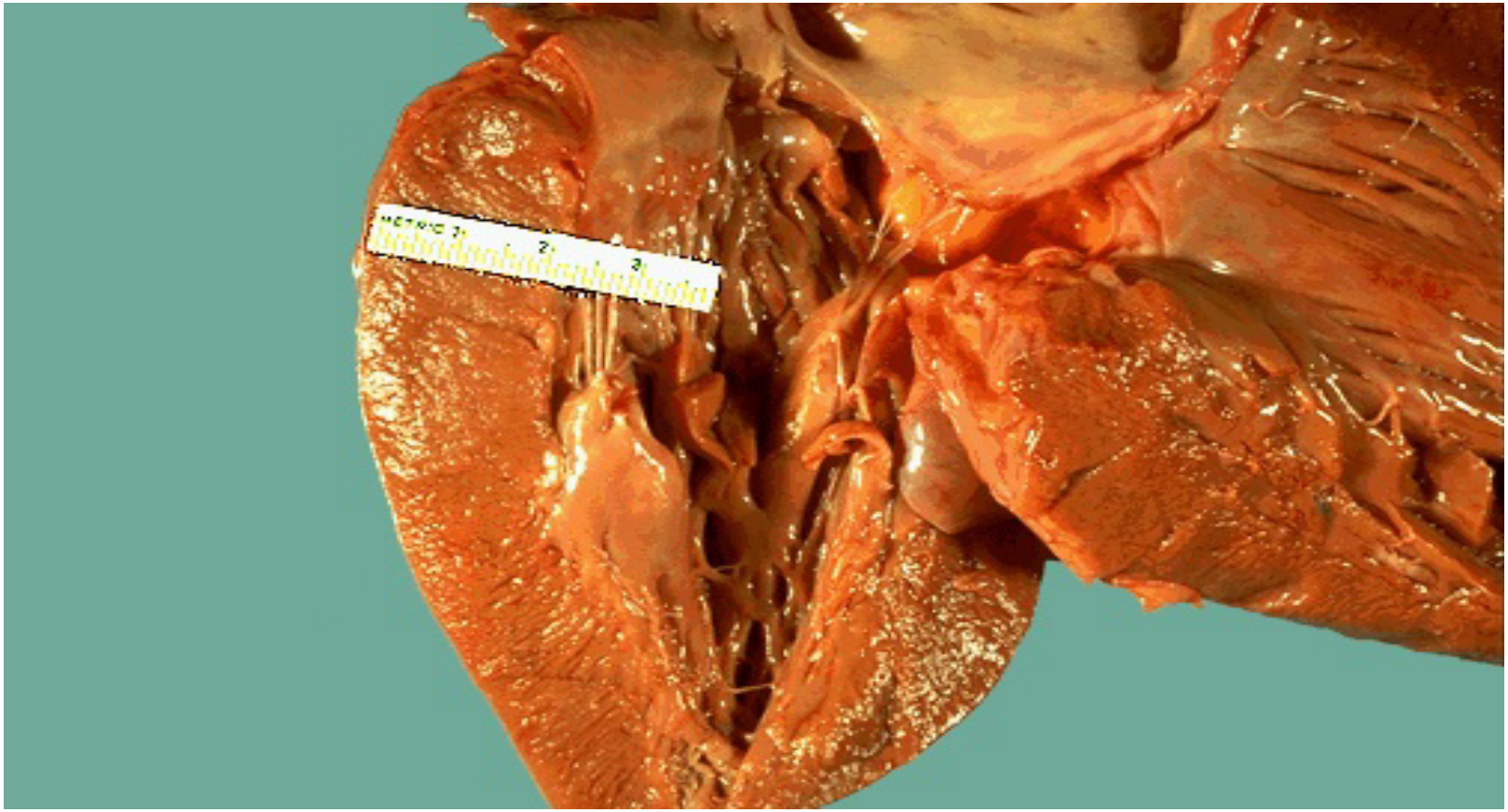
HCM

Normal

Histopathology showing significant myofiber disarray and interstitial fibrosis

Histopathology showing Normal myocytes

Left ventricular hypertrophy - Gross



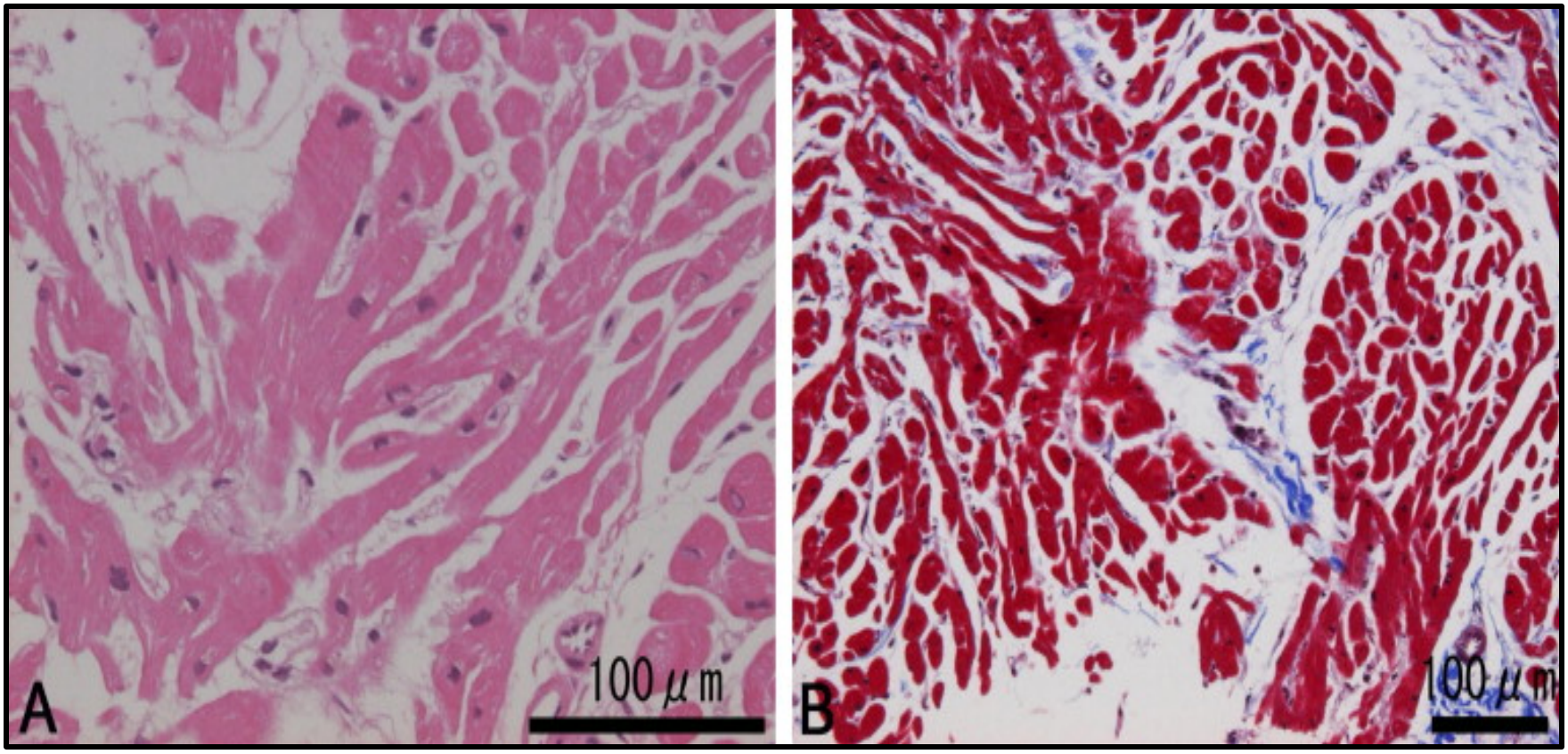
Heart from a hypertensive patient. The left ventricle is very thick (over 2 cm). However the rest of the heart is fairly normal in size as is typical for hypertensive heart disease. The hypertension creates a greater pressure load on the heart to induce the hypertrophy

Left ventricular hypertrophy - Gross

This cross section view of the heart shows the left ventricle in the left of the picture.

The left ventricle is grossly thickened secondary to severe hypertensive. The myocardial fibers have undergone hypertrophy.

Hypertrophic Cardiomyopathy - LPF

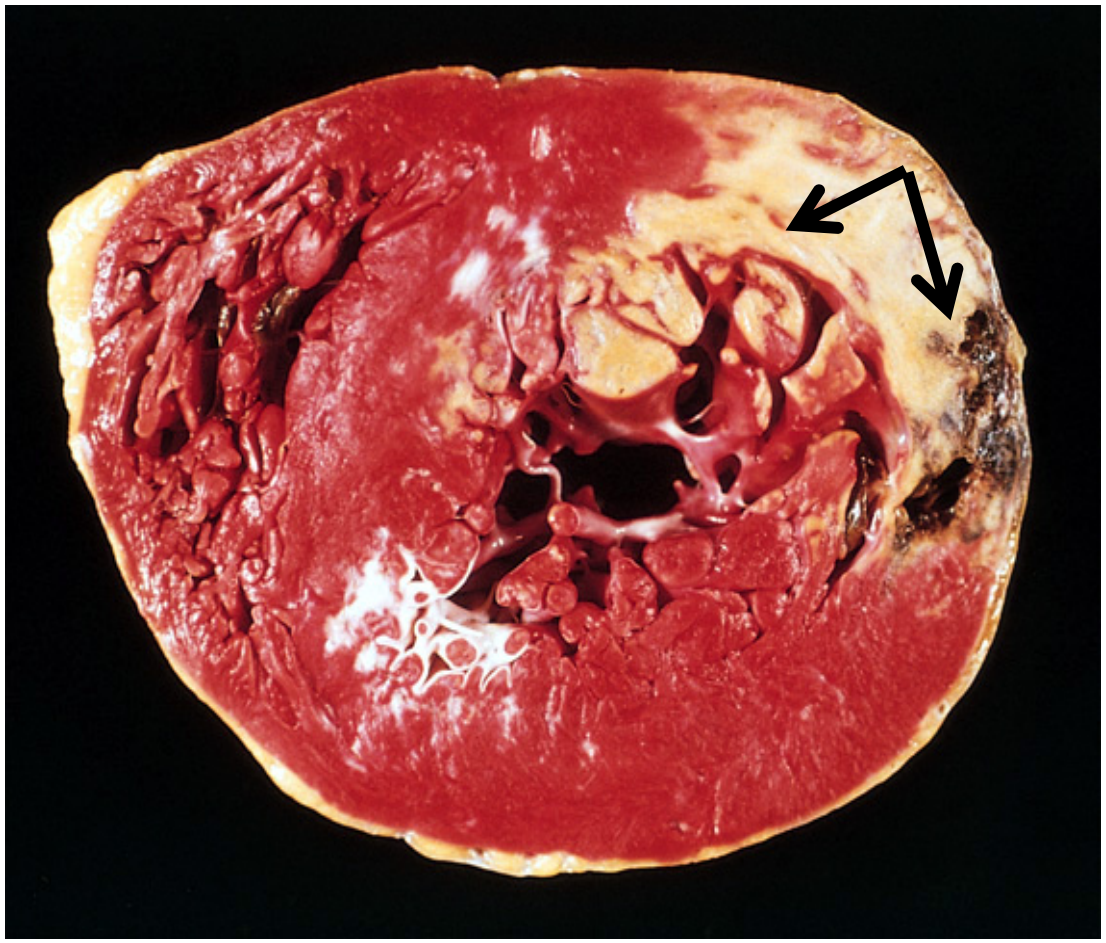


haematoxylin-eosin stain ***Masson's trichrome stain***

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

MYOCARDIAL INFARCTION

Myocardial Infarction - CS

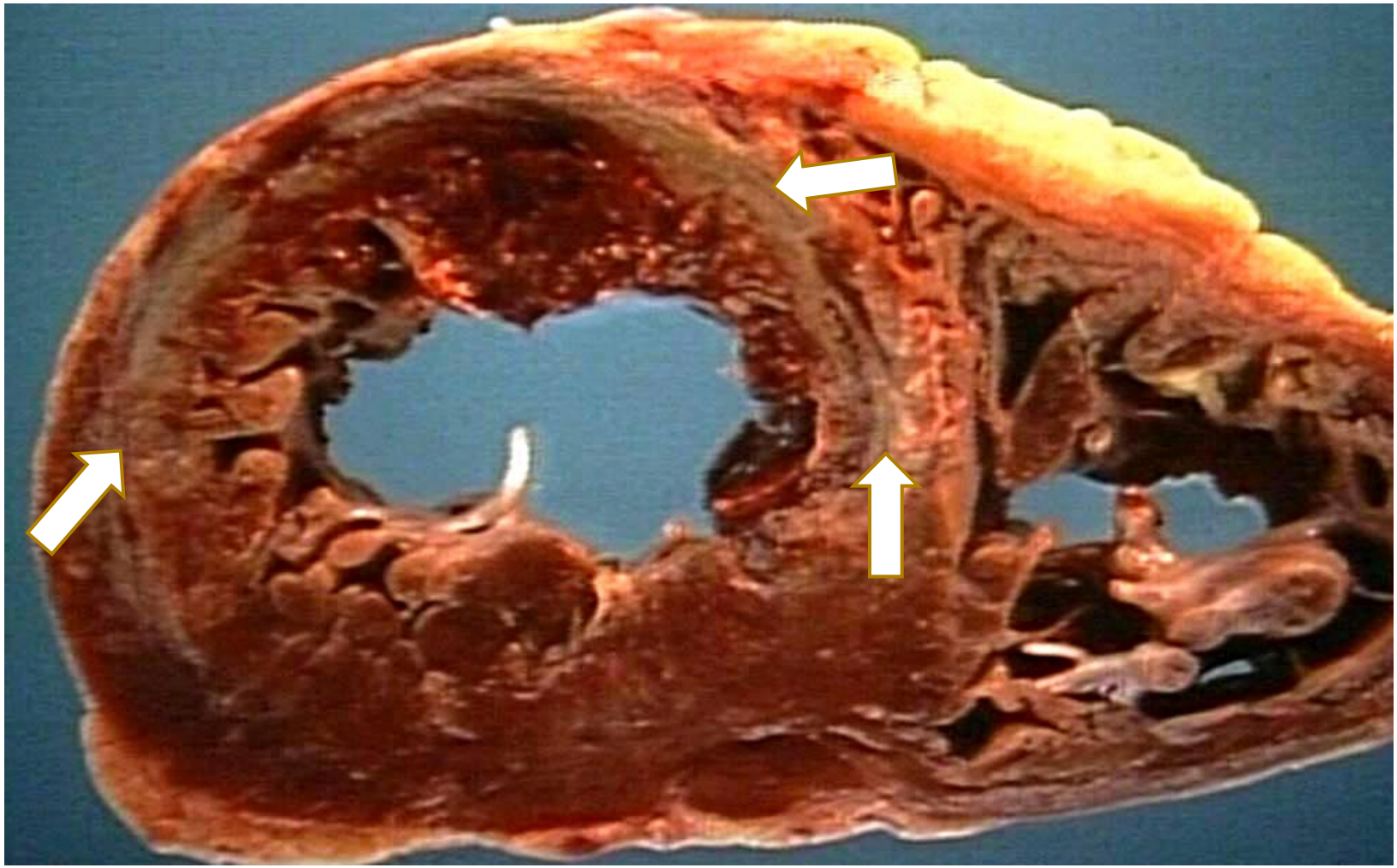


DIAGNOSIS (CAUSE OF DEATH)?
ORGAN?
Complications:

1: Arrhythmias.
2: Cardiac Shock.
3: Pericarditis.
4: Heart Failure.
5: Ventricular Aneurysm.
6: Myocardial Rupture.

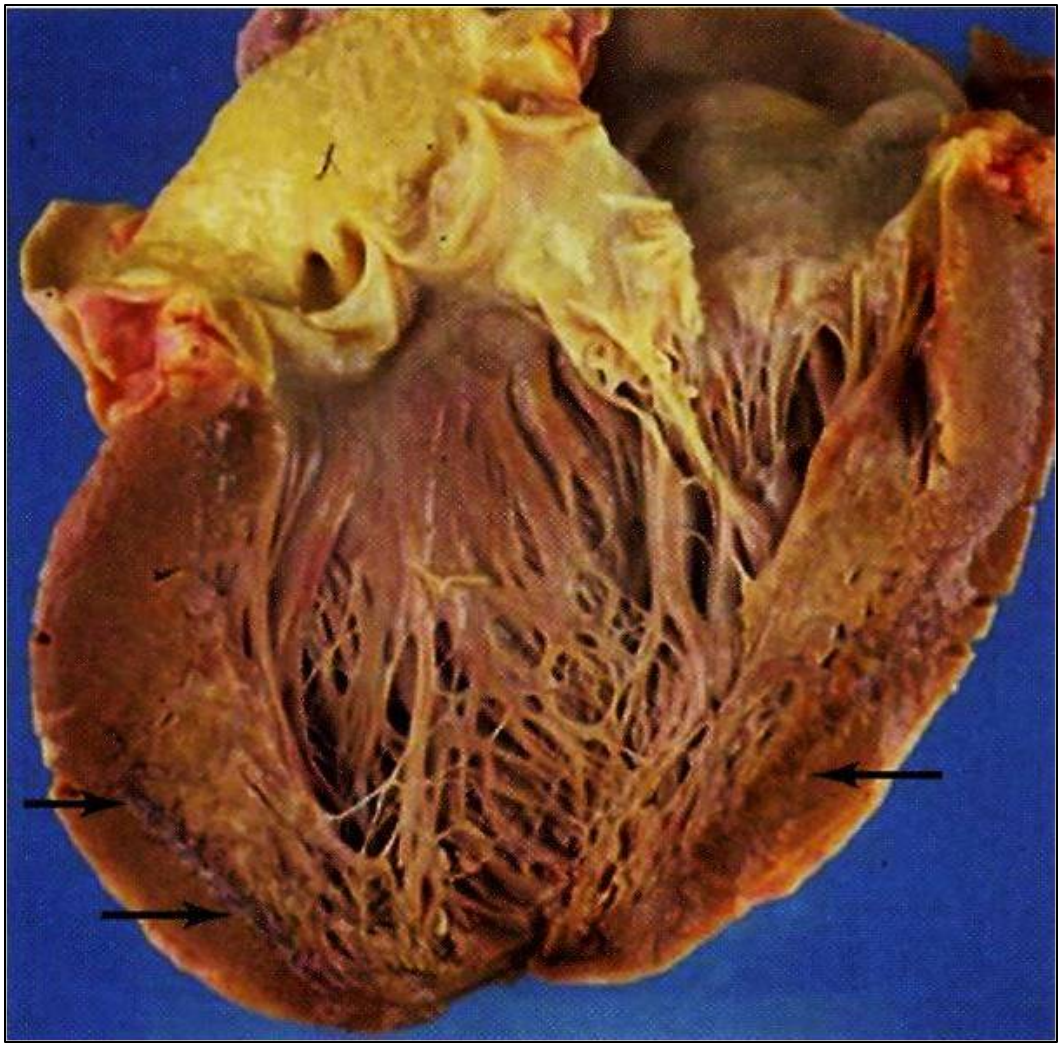
Cross section of the left and right ventricles shows a pale and irregular focal fibrosis in the left ventricular wall with increased thickness .

Myocardial Infarction



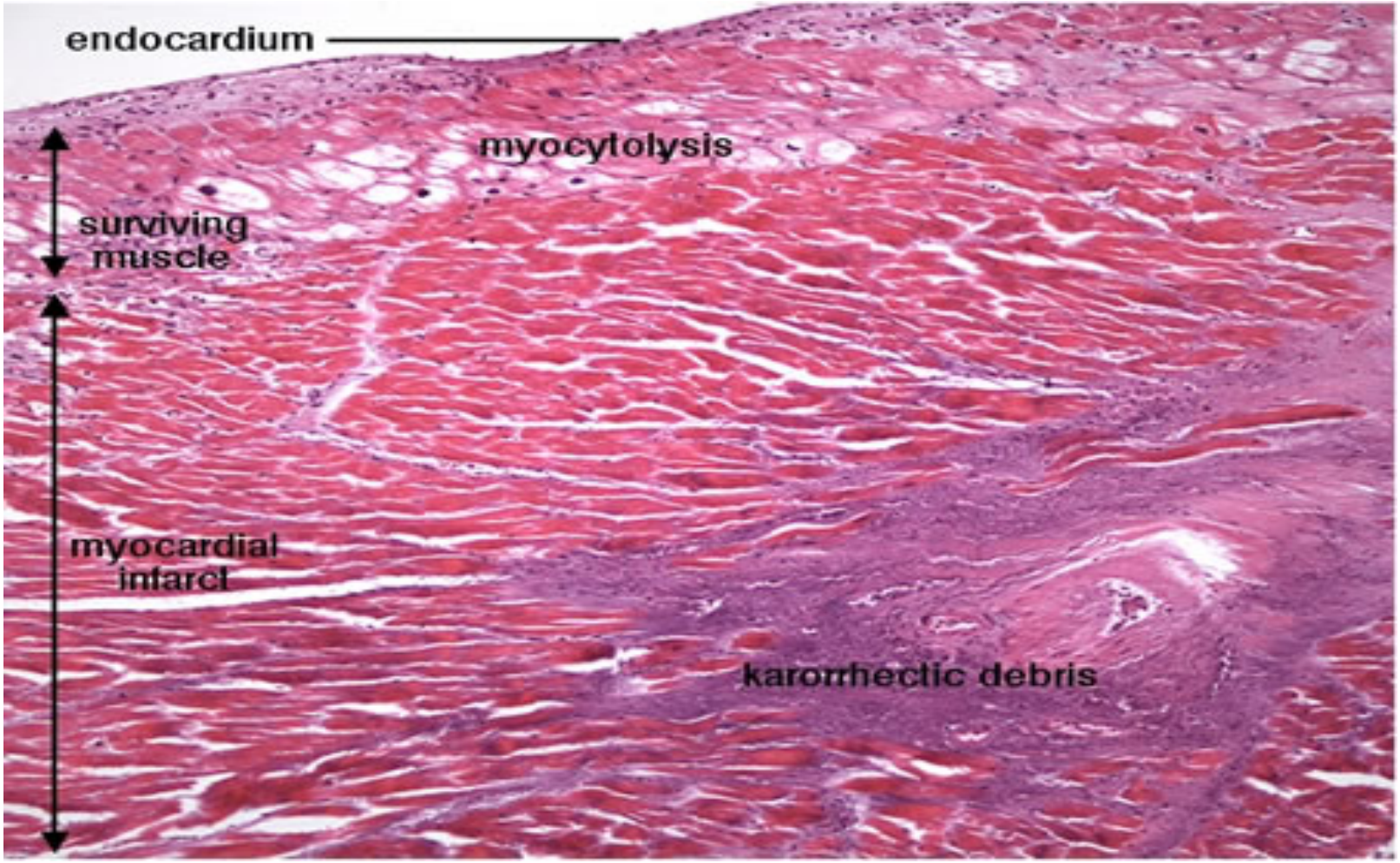
Cross section of the left and right ventricles shows a pale and irregular focal fibrosis in the left ventricular wall with increased thickness .

Myocardial Infarction - CS



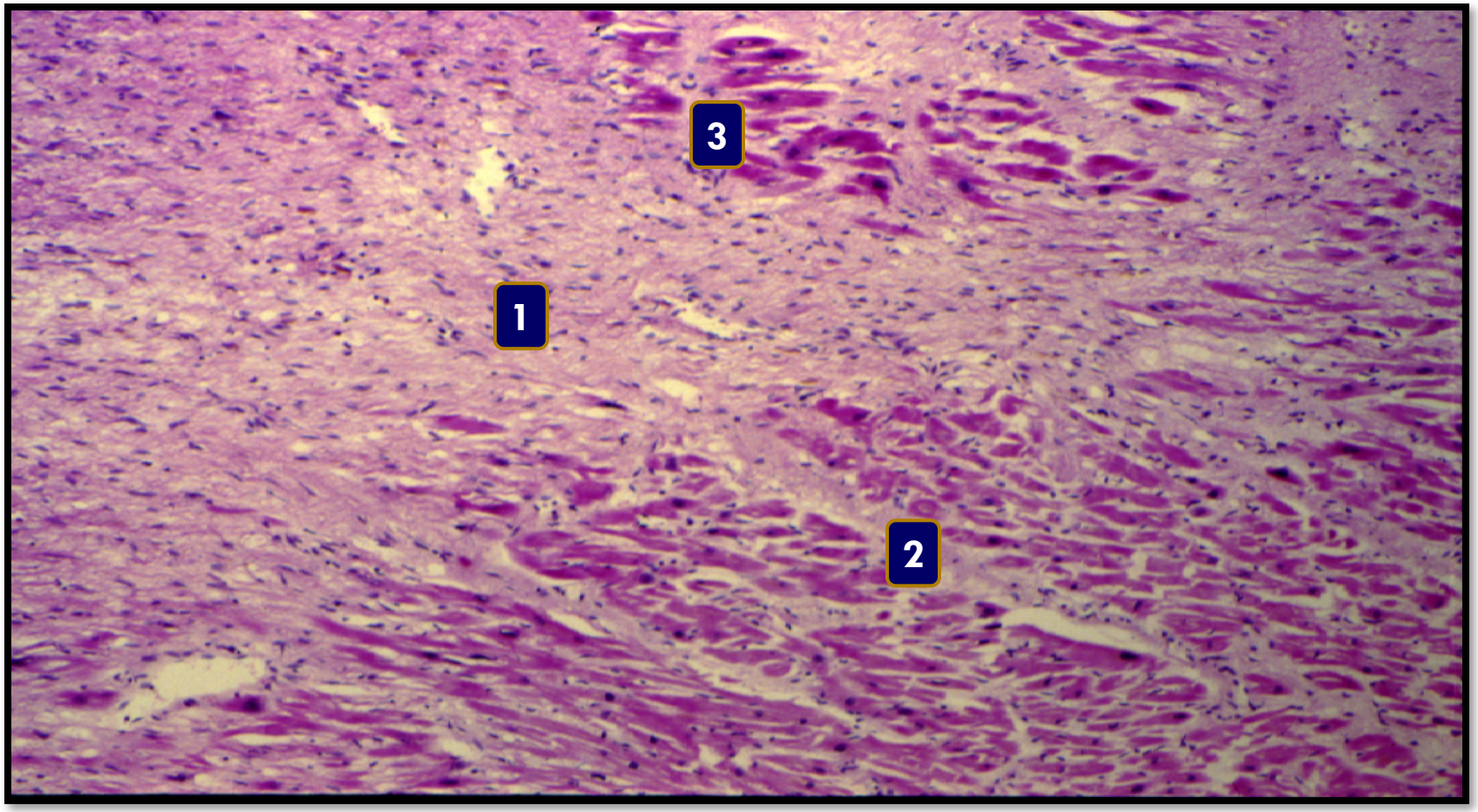
Complications *that might occur : arrhythmias , ventricular aneurysm, rupture of myocardium, cardiac tamponade and others .*

Myocardial Infarction - LPF



Transmural myocardial infarct at 2 weeks

Myocardial Infarction - LPF

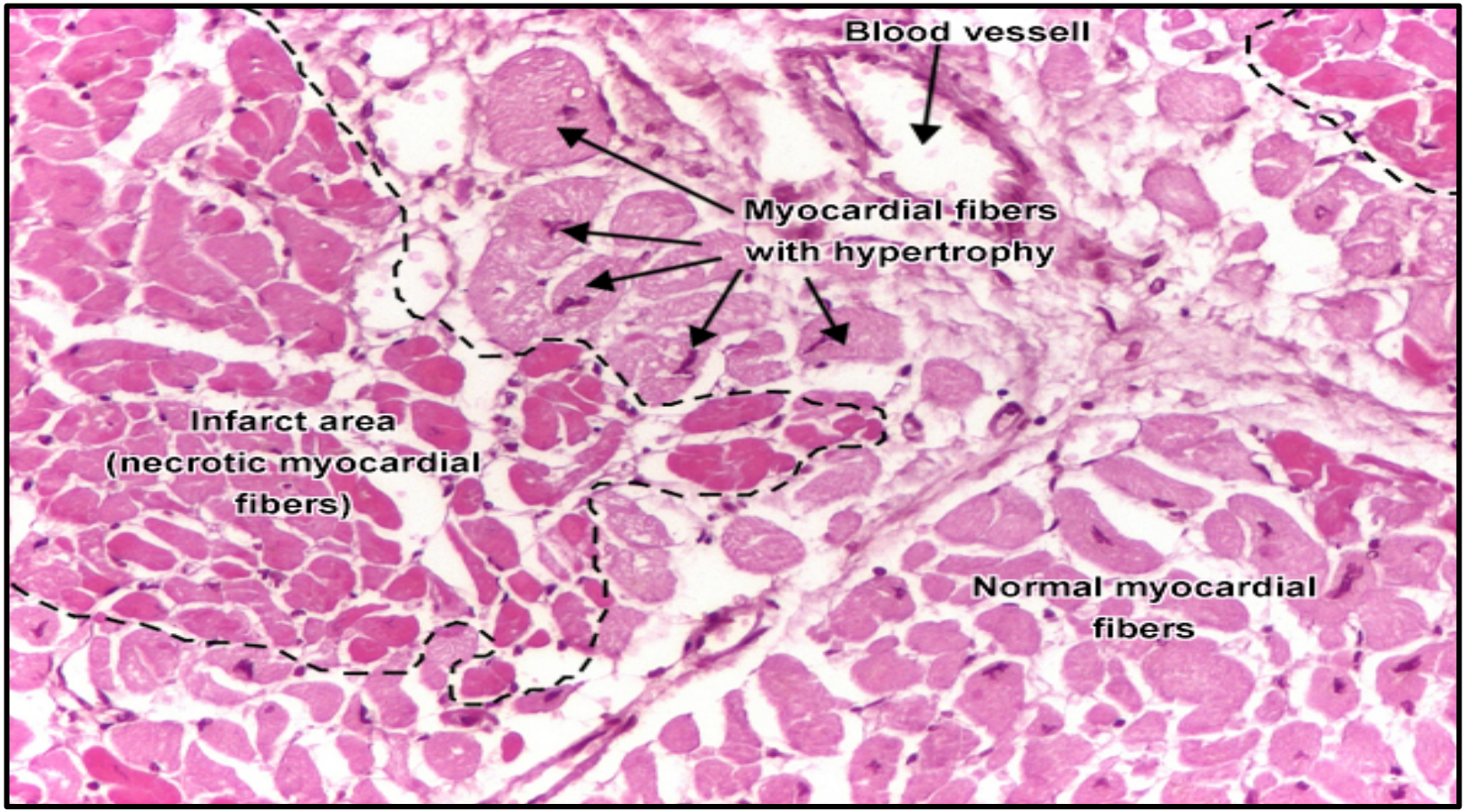


1- ***Patchy coagulative necrosis of myocardial fibers.*** *The dead muscle fibers are structureless and hyaline with loss of nuclei and striations.*

2- ***Chronic ischemic fibrous scar*** *replacing dead myocardial fibers .*

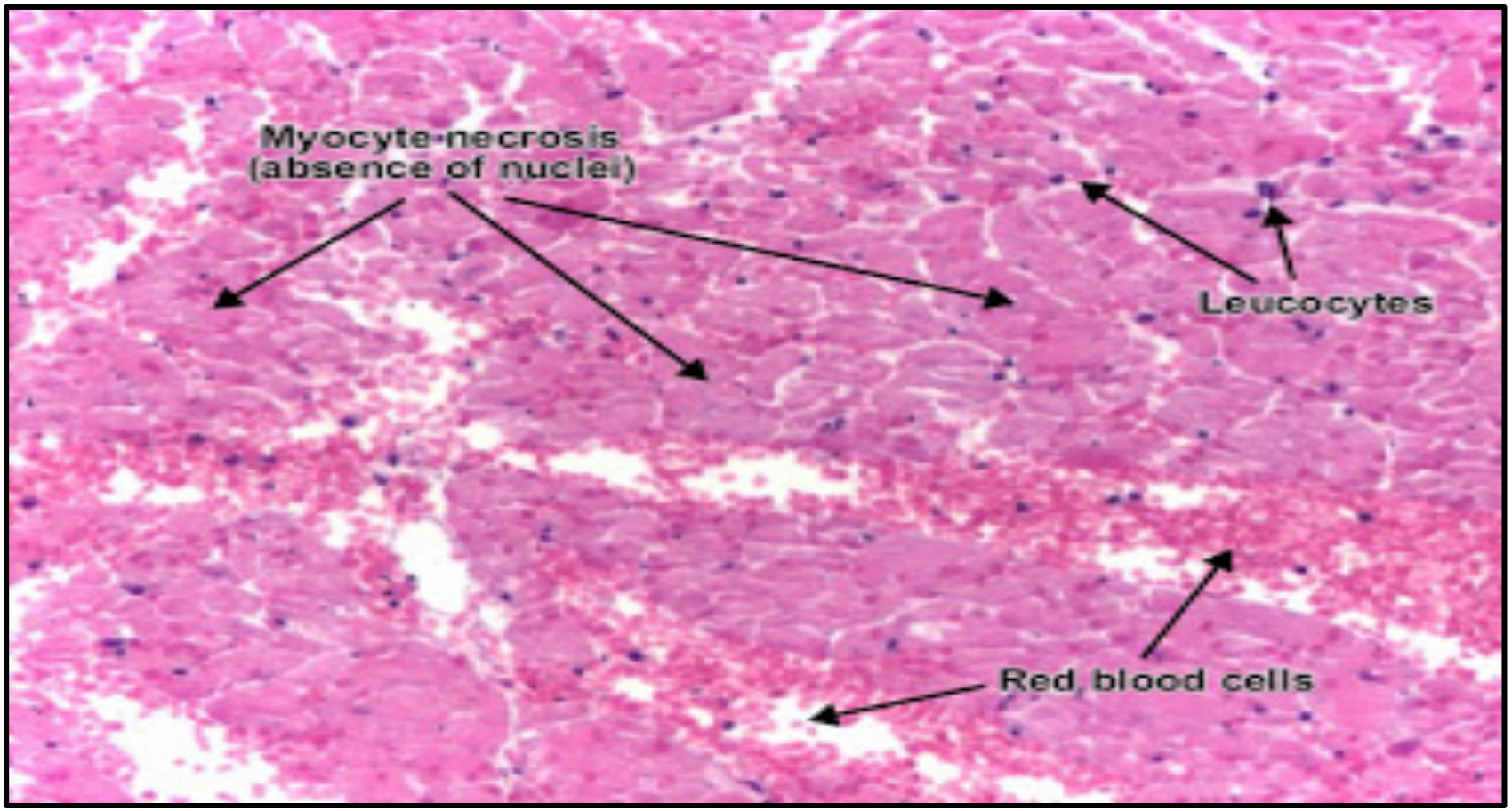
3- ***The remaining myocardial fibers*** *show enlarged nuclei due to ventricular hypertrophy .*

Myocardial Infarction - LPF



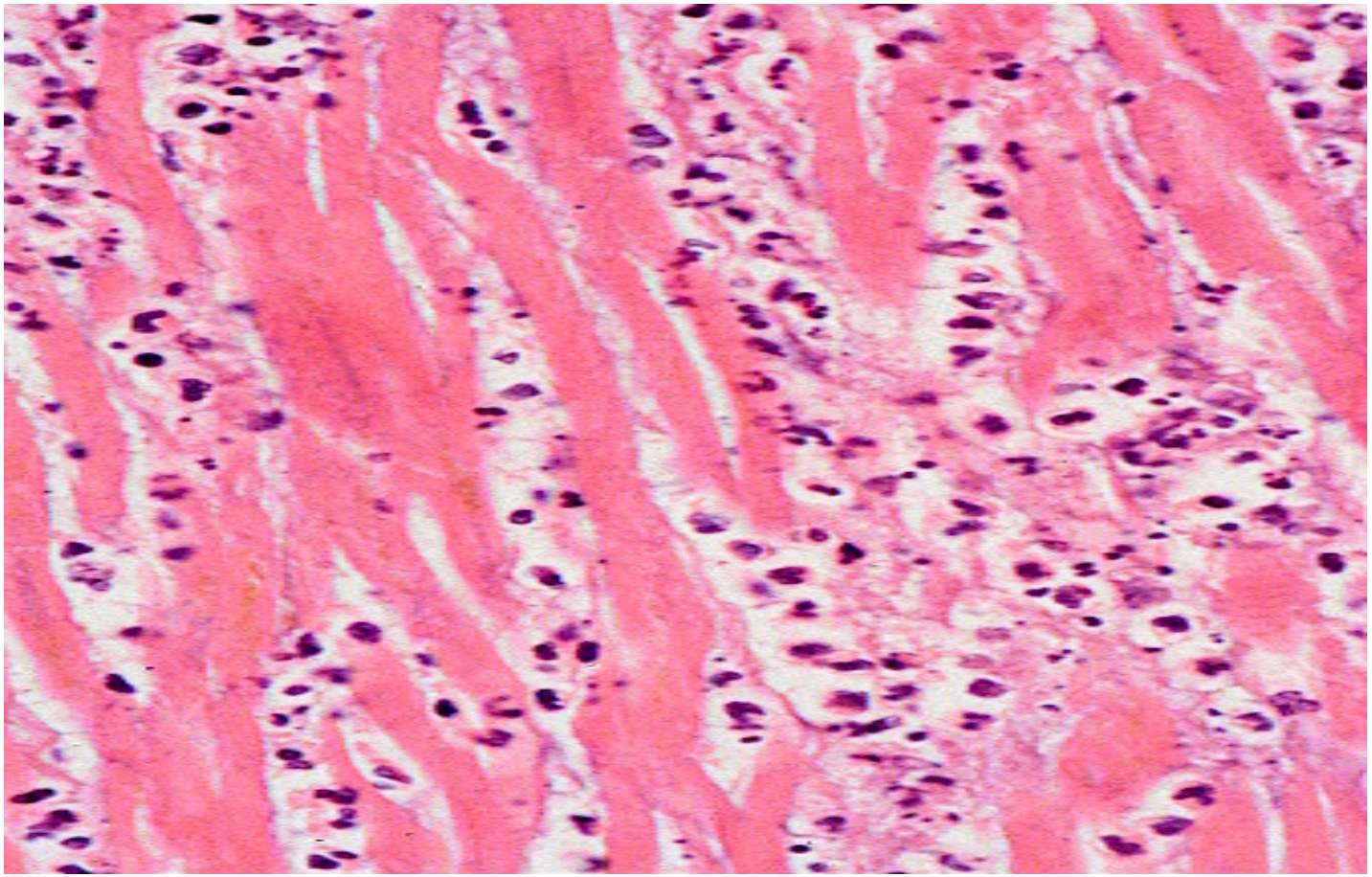
Myocardial infarct *- circumscribed area of ischemic necrosis - coagulative necrosis. In the first 12 - 24 hours, myocardial fibers are still well delineated, with intense eosinophilic (pink) cytoplasm, but lost their transversal striations and the nucleus (left side of the picture). Notice a few myocardial fibers showing hypertrophy (increased size of the fiber, irregular shape of the nuclei)*

Myocardial Infarction - LPF



***Recent myocardial infarct** (in the first 12 - 24 hours): myocardial fibers are still well delineated, with intense eosinophilic (pink) cytoplasm, but lost their transversal striations and the nucleus. The interstitial space may be infiltrated with red blood cells.*

Myocardial Infarction - HPF



***Acute myocardial infarct**, histology. This 3-4 day old infarct shows necrosis of myocardial cells and is infiltrated with **polymorphnuclear** leukocytes.*

Myocardial Infarction - HPF

Microscopic image of Myocardial Infarction

THROMBOEMBOLISM / VASCULITIS

Thromboangitis oblitrans (Buerger's disease)

Boehringer Ingelheim

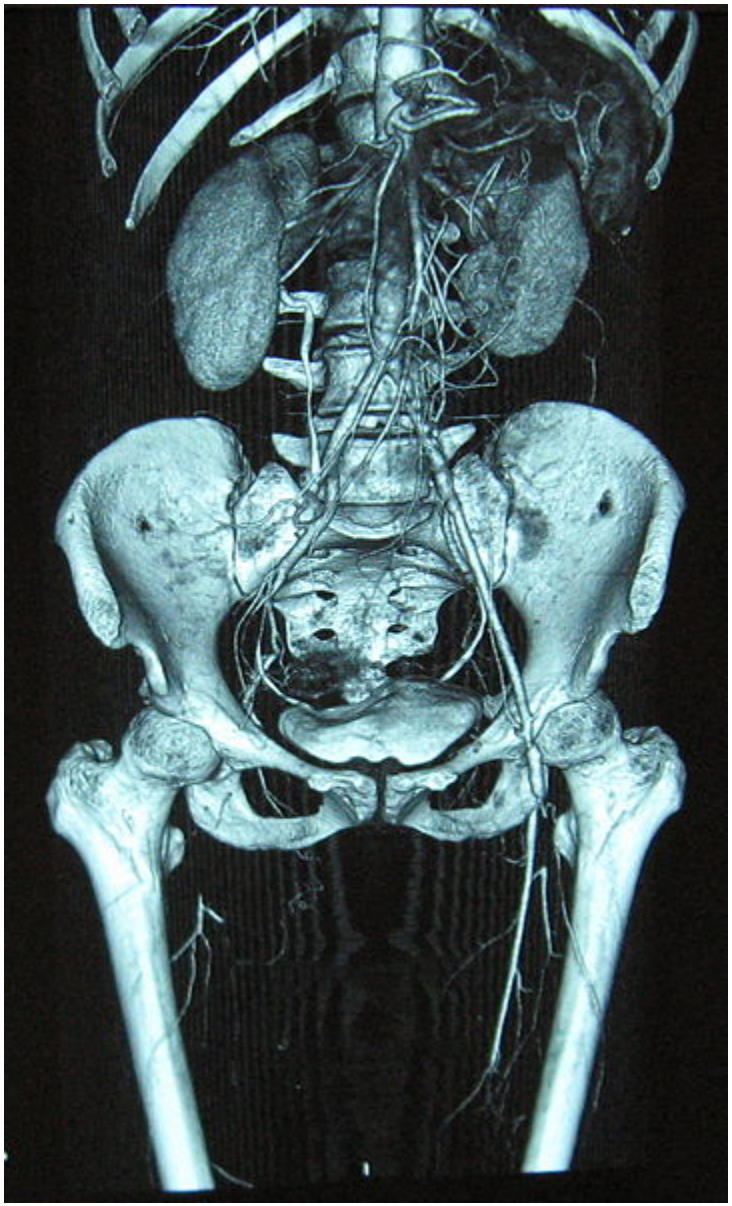
Thromboangitis oblitrans (Buerger`s disease)

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

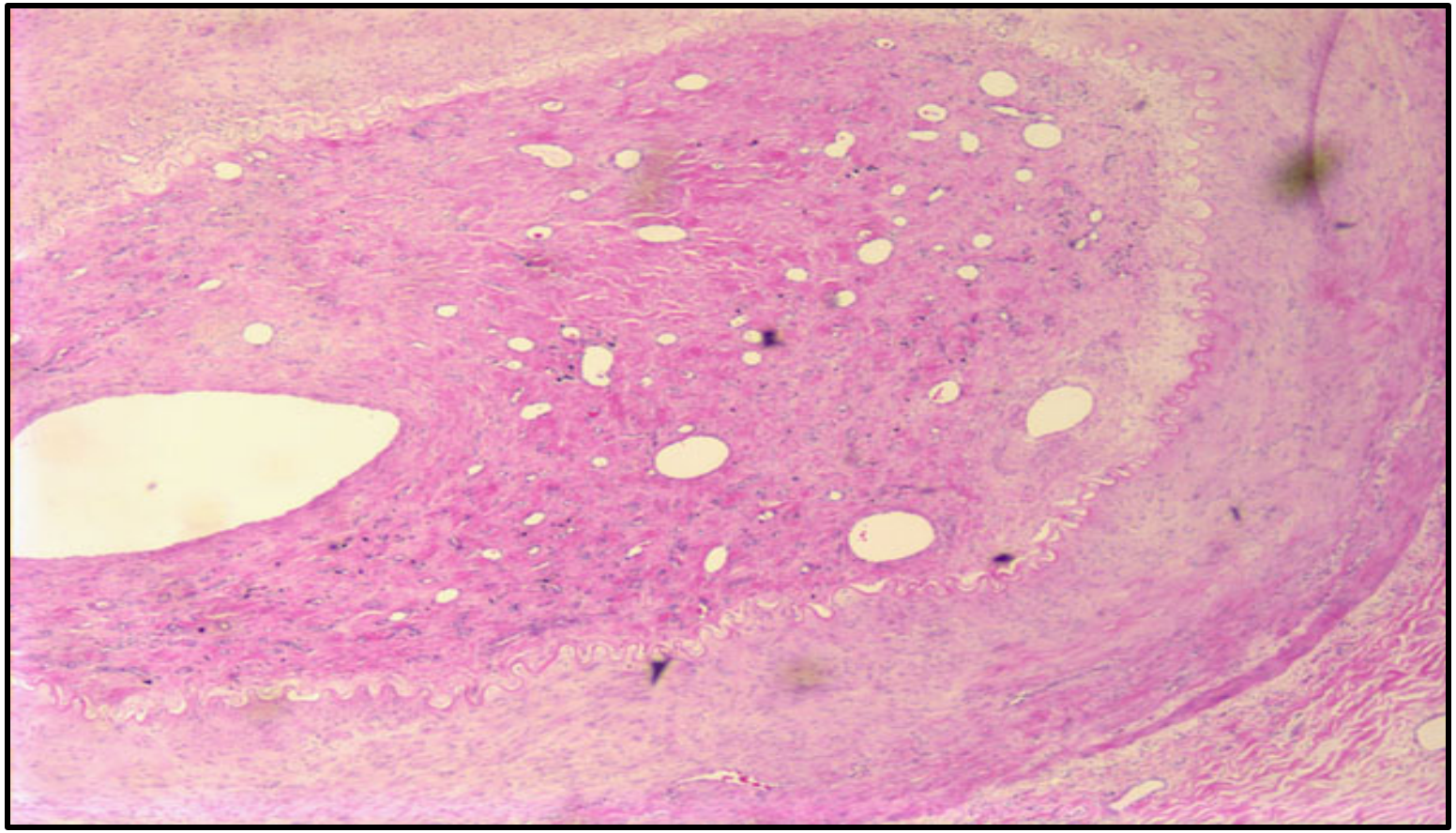
THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE)

- *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*

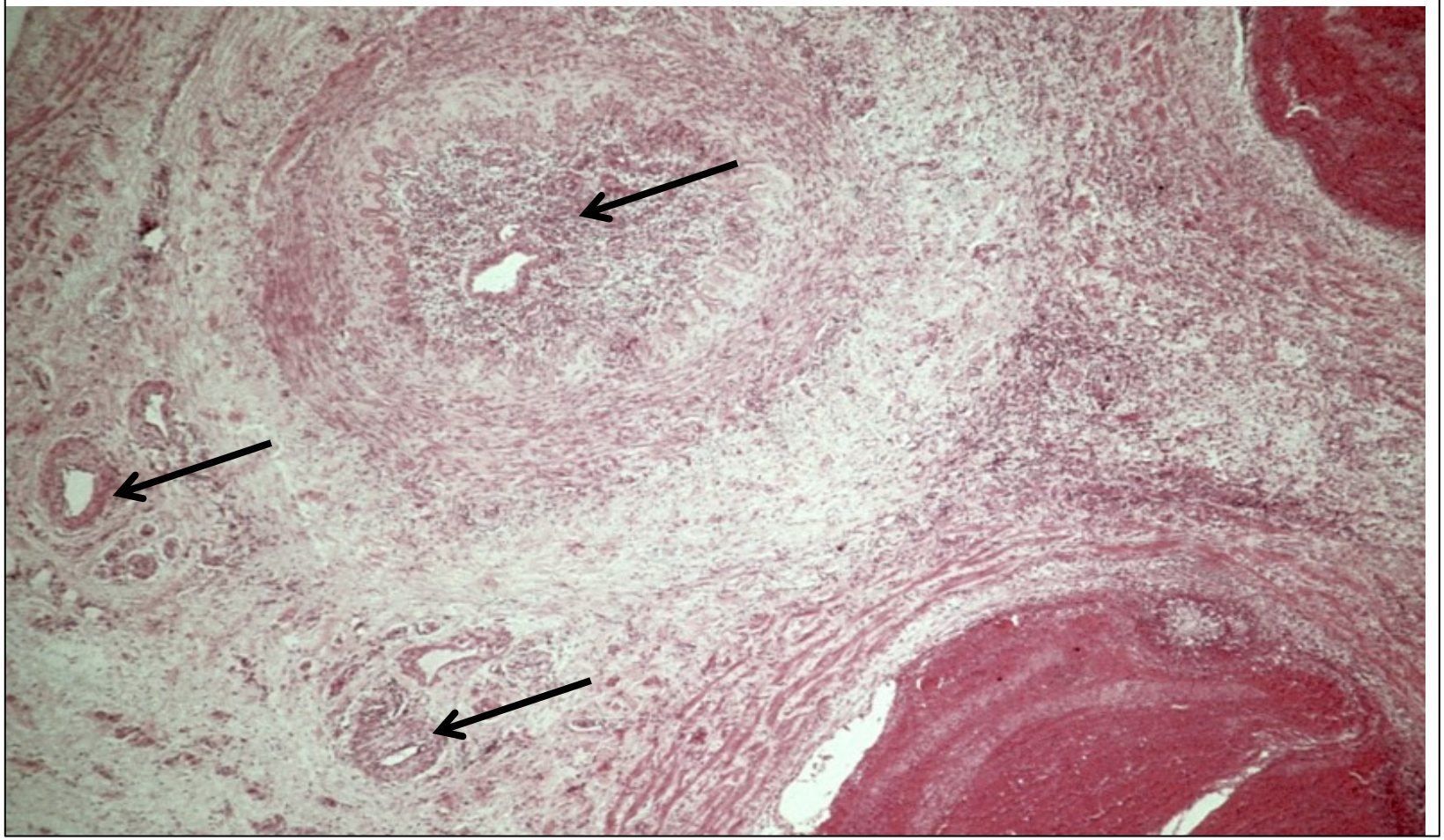


THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE) - LPF



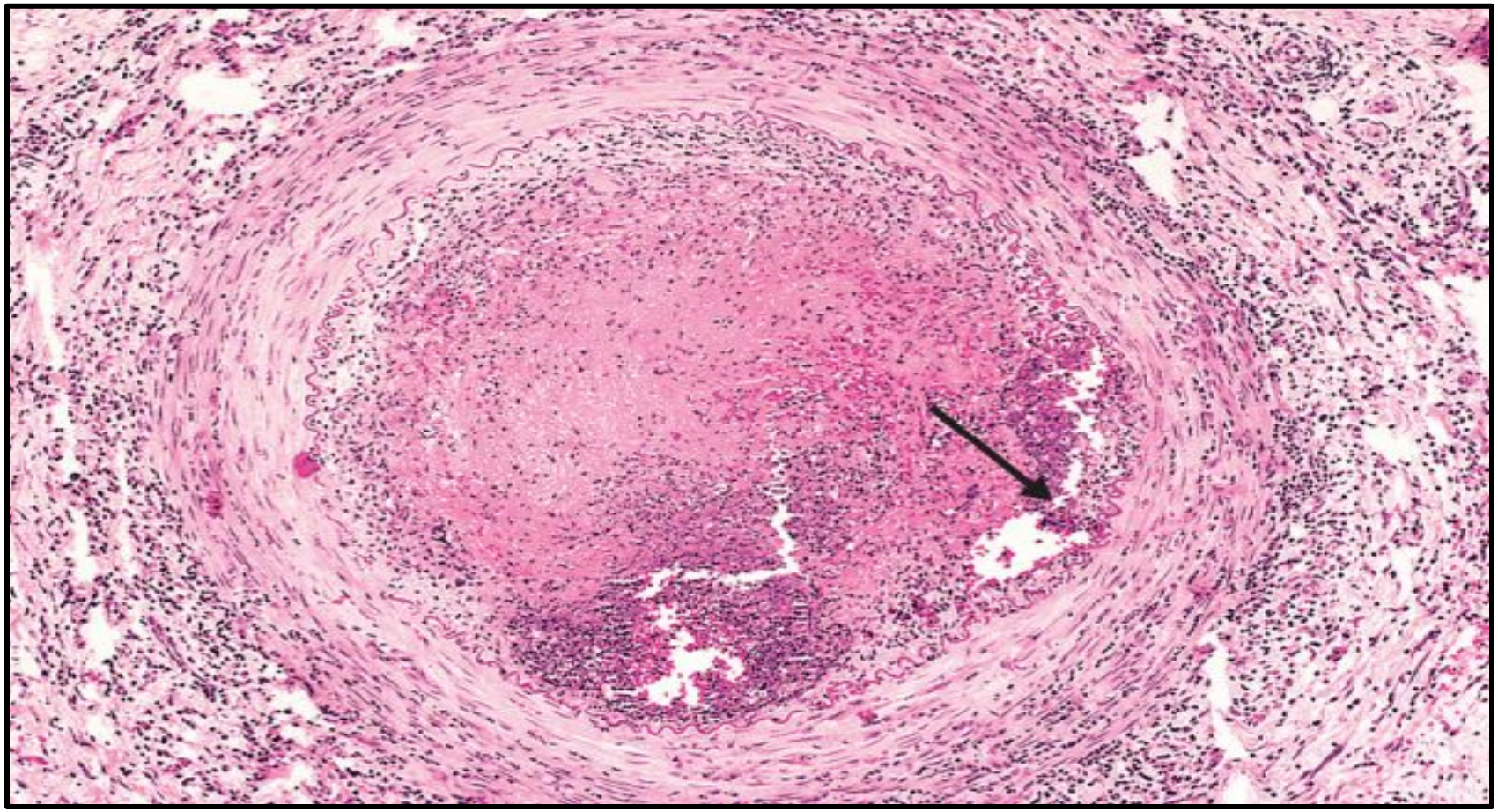
Thromboangiitis obliterans (Buerger's disease) is a non atherosclerotic, segmental, inflammatory, vaso-occlusive disease that affects the small- and medium-sized arteries and veins of the upper and lower extremities.

THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE)



Large number of small blood vessels in the dermis show occlusive organized thrombi with recanalization and fibrosis around blood vessels.

THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE) - HPF



Thromboangitis obliterans (Buerger disease). The lumen is occluded by a thrombus containing abscesses (arrow), and the vessel wall is infiltrated with leukocytes.

Thromboembolism

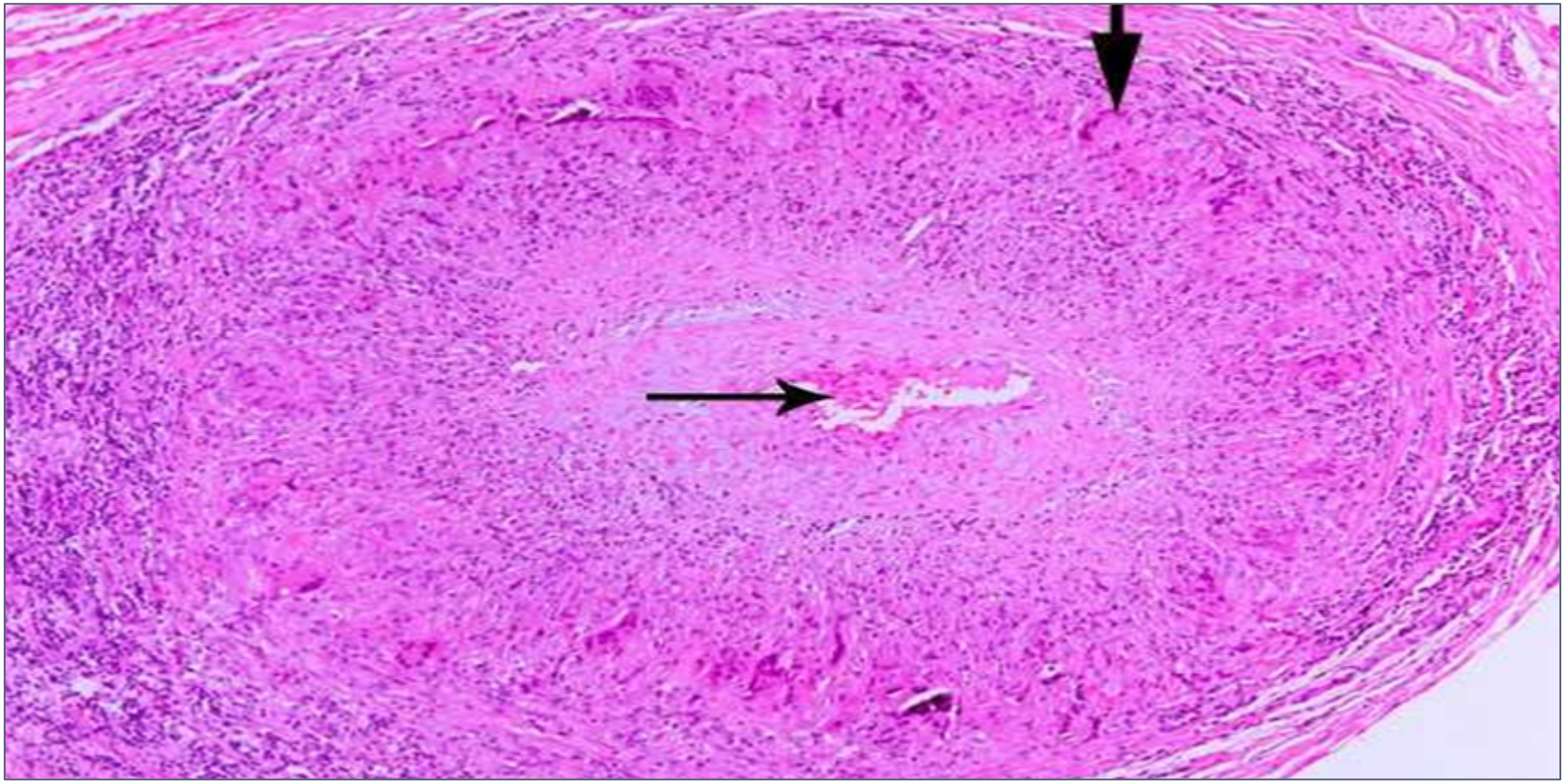
Atheroma with thrombosis

GIANT CELL (TEMPORAL) ARTERITIS

GIANT CELL / TEMPORAL ARTERITIS

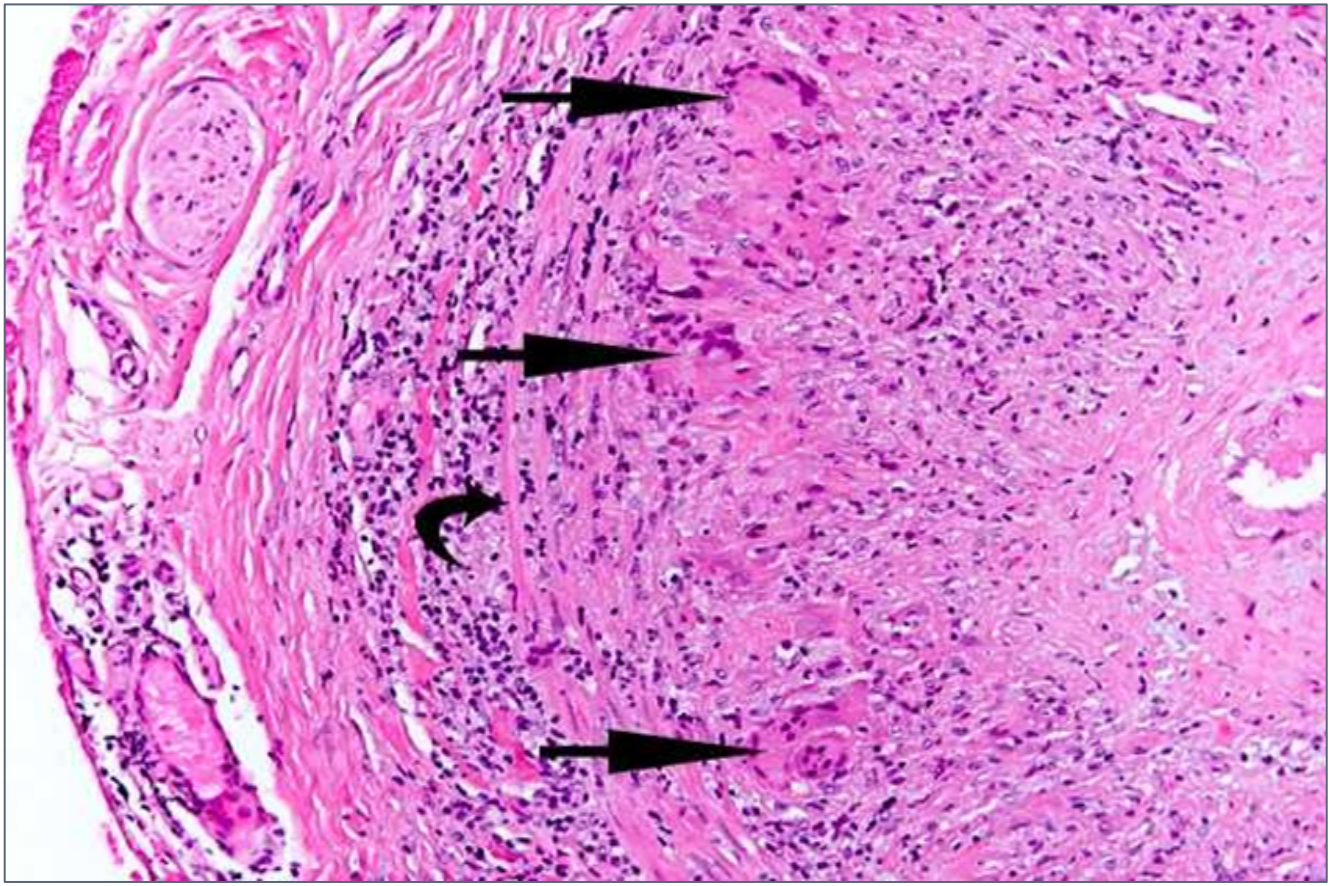
Tender and thickened scalp veins

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).

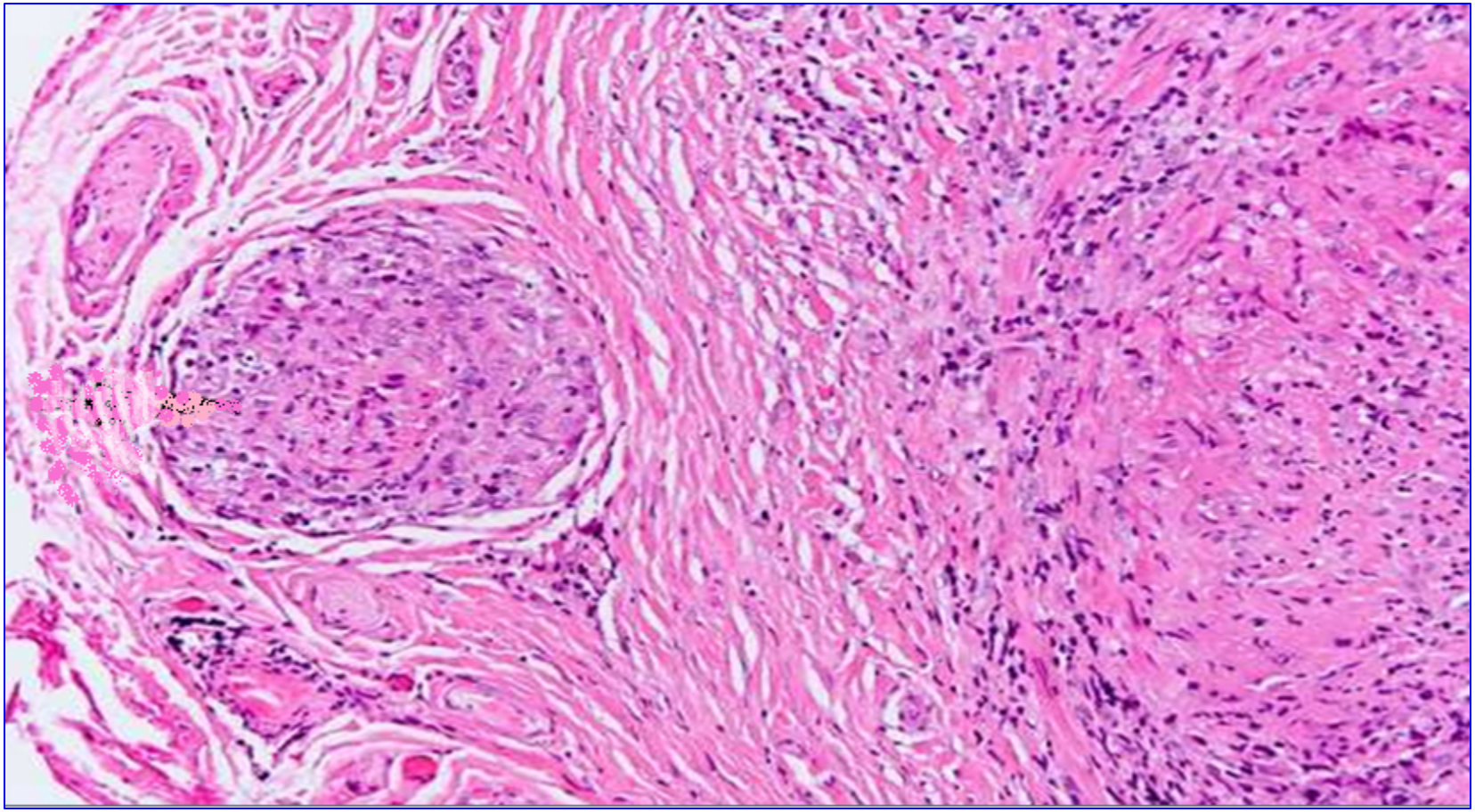
GIANT CELL / TEMPORAL ARTERITIS - HPF



- Name of the cells?
- Diagnosis?
- Vacuities + HSP?

Giant cells can be of Langhans type or foreign-body type (three arrows) and may show fragments of disrupted internal elastic lamina. Note the presence of dense chronic lymphocytic inflammation traversing through circumferential smooth muscle fibers (curved arrow) of vascular media.

GIANT CELL (TEMPORAL) ARTERITIS - HPF



The inflammation can be granulomatous in addition to both acute and chronic inflammatory cells. This photomicrograph shows a single granuloma in the adventitia of the artery. Acute inflammation when present is generally mild and represents an early stage of the disease.

GIANT CELL (TEMPORAL) ARTERITIS - HPF

Disruptions of the elastic lamina with inflammation and giant cells.

***Segmental** inflammatory lesions with **intimal thickening** ,*
granulomatous inflammation with giant cells and chronic
*inflammatory cells and **internal elastic lamina** fragmentation*

LEUKOCYTOCLASTIC / HYPERSENSITIVITY VASCULITIS
(MICROSCOPIC POLYANGITIS)

Hypersensitivity vasculitis – Clinical sign

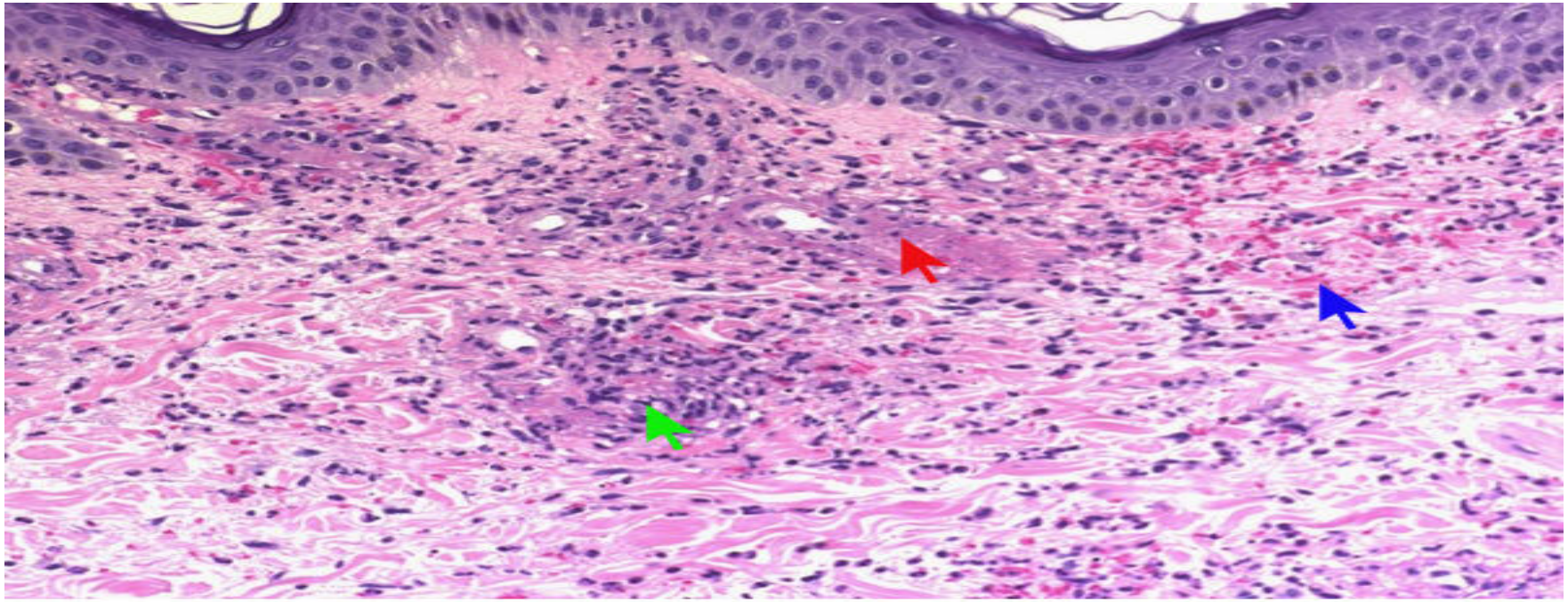
Hypersensitivity vasculitis might be complicated with glomerulonephritis and hemoptysis due to pulmonary capillaritis

Leukocytoclastic vasculitis - Clinical sign

Leukocytoclastic vasculitis

The purpuric eruption (Subcutaneous bleeding patches) of the foot tends to be most pronounced on dependent areas.

Leukocytoclastic vasculitis - HPF



Fibrinoid type necrosis

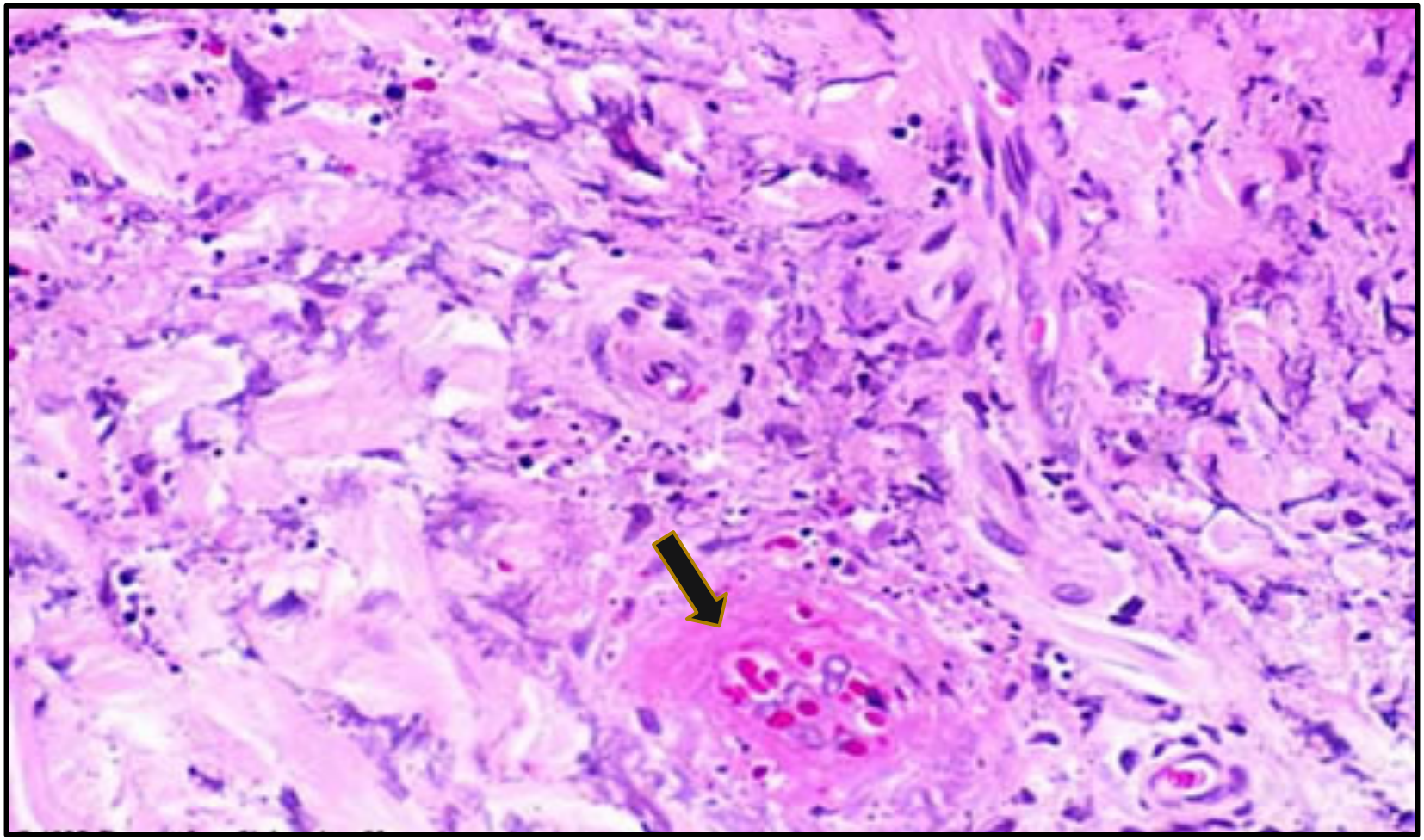
Red cell extravasation

Inflammation

Vasculitis, leukocytoclasis (high power)

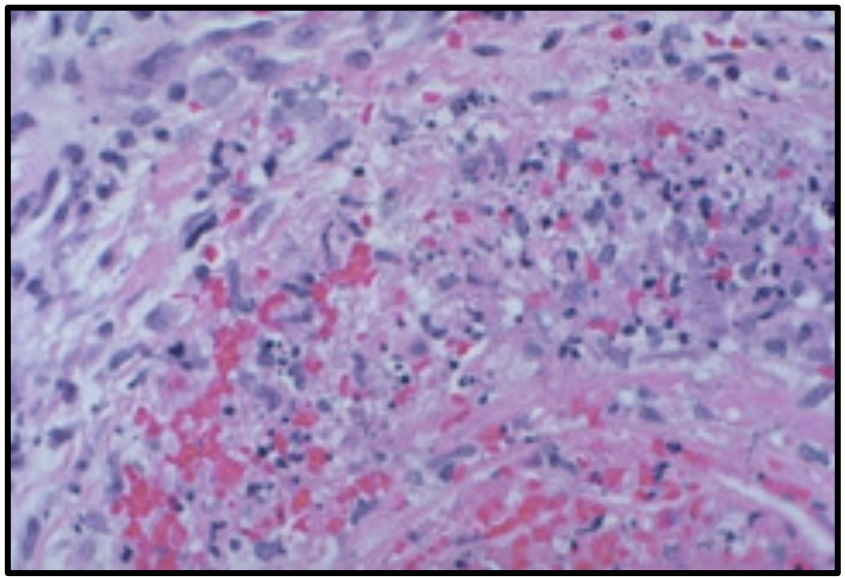
Section of the skin shows fibrinoid necrosis of blood vessels with extravasation of RBCs , neutrophilic infiltration with debris (leukocytoclasis /nuclear dust)

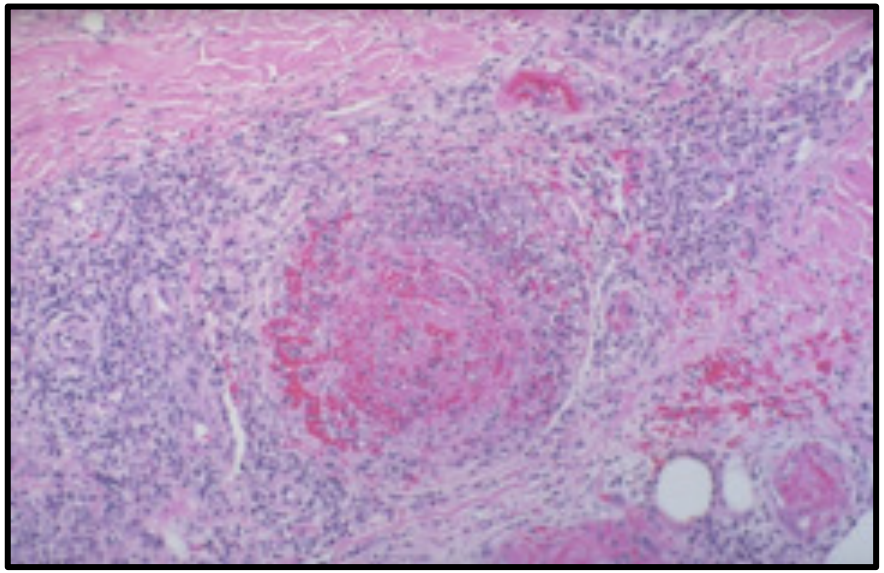
Leukocytoclastic vasculitis - HPF

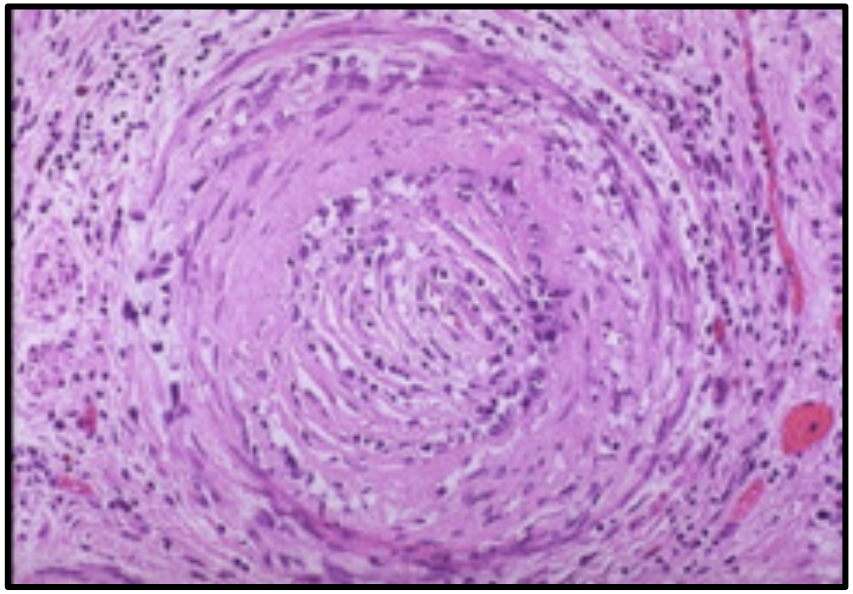


Fibrinoid necrosis of small dermal vessels is present, necessary to establish the diagnosis of leukocytoclastic vasculitis.

Severe vasculitis – Microscopic views







This muscular artery shows a more severe vasculitis with acute and chronic inflammatory cell infiltrates, along with necrosis of the vascular wall


THE END