CARDIOVASCULAR SYSTEM

Pathology Practical

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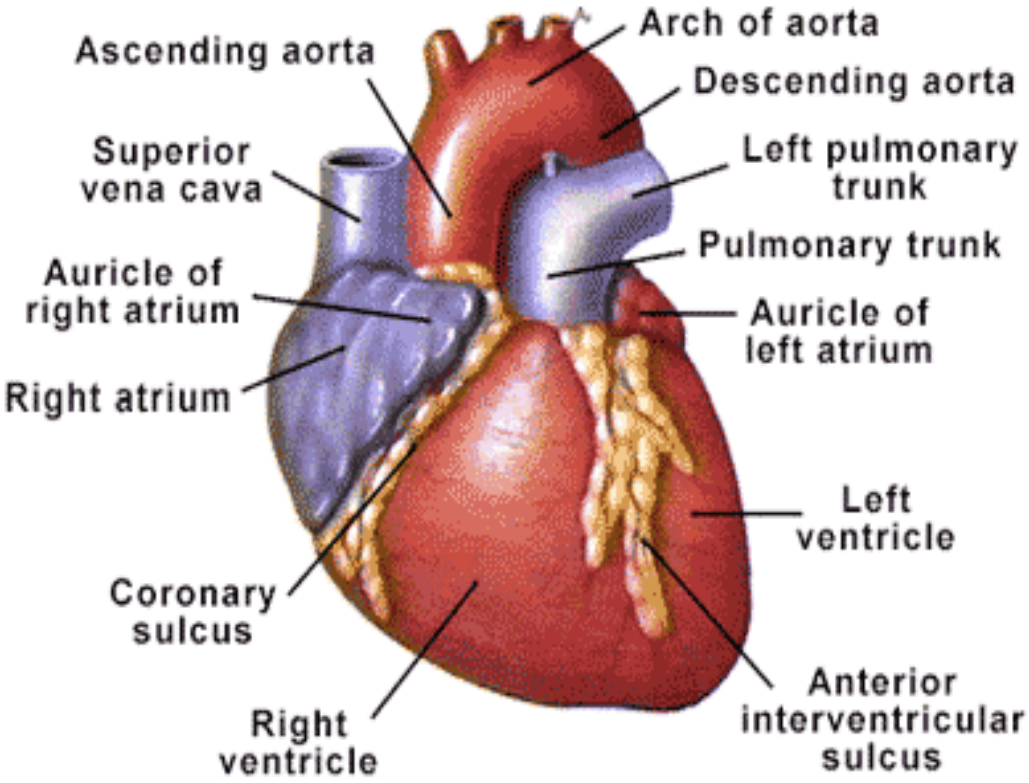
NORMAL ANATOMY AND HISTOLOGY

Objectives:

At the end of the 2nd P2 practical sessions of the cardiovascular block, the medical students will be able to:

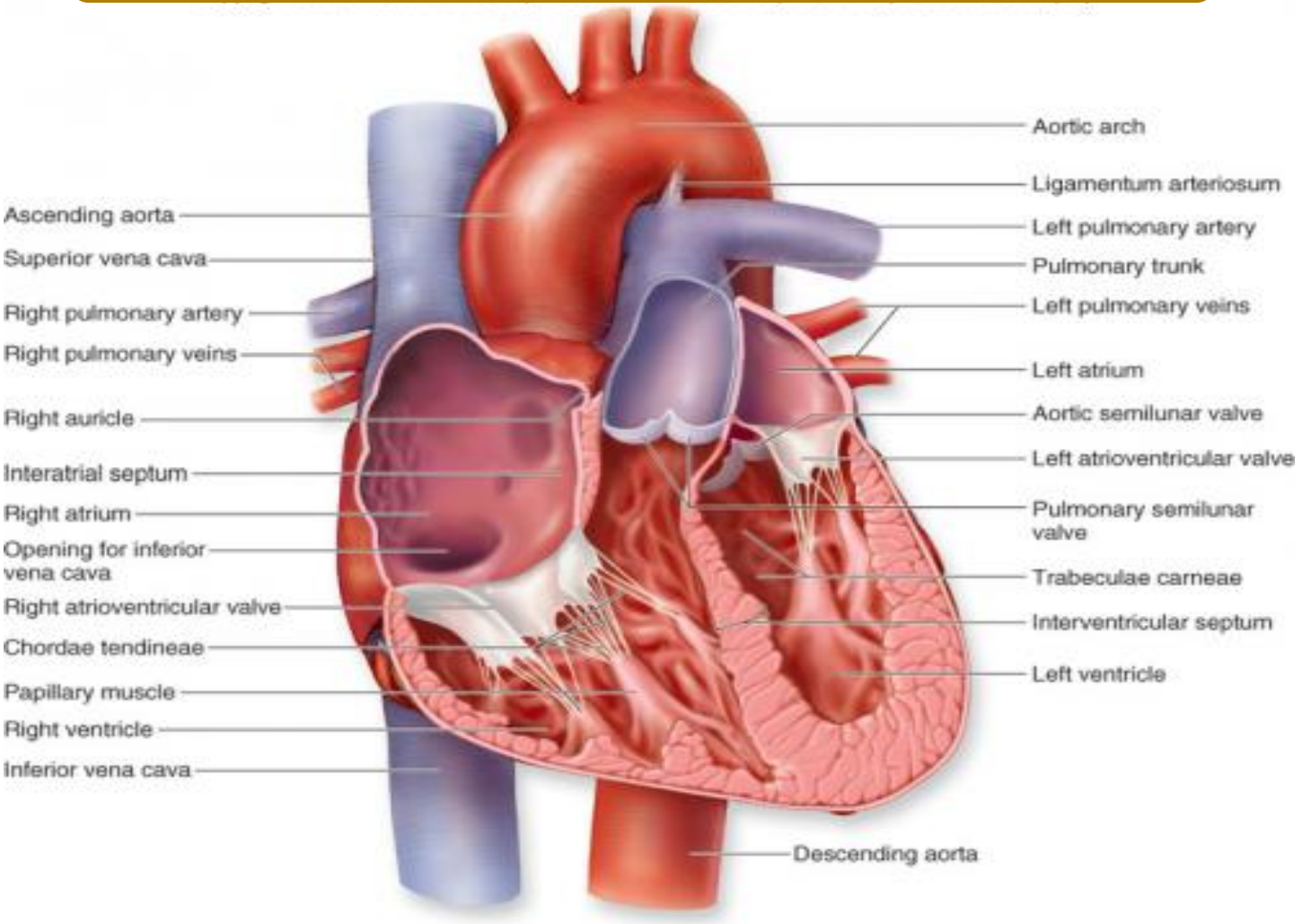
- Identify the morphologic histopathological features of:
  - chronic venous congestion of the liver
  - chronic venous congestion of the lung
  - Hypertensive changes in blood vessels
  - Myocardial hypertrophy
  - Thromboangitis obliterans
  - Giant cell arteritis
  - Leukocytoclastic vasculitis.

Anatomy of the Heart



- The heart serves as a **mechanical pump** to supply the entire body with blood, both providing nutrients and removing waste products.
- The great vessels exit the base of the heart.
- Blood flow: body→ sup & inf venae cava → right atrium→ right ventricle→ lungs → left atrium → left ventricle→ Aorta → body

Anatomy of the Heart – inside view

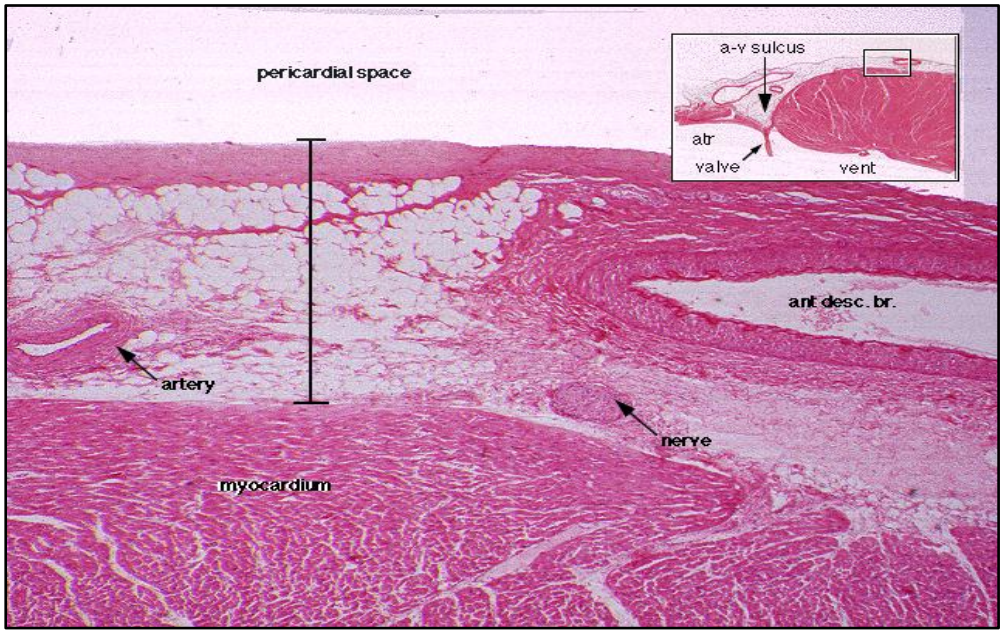


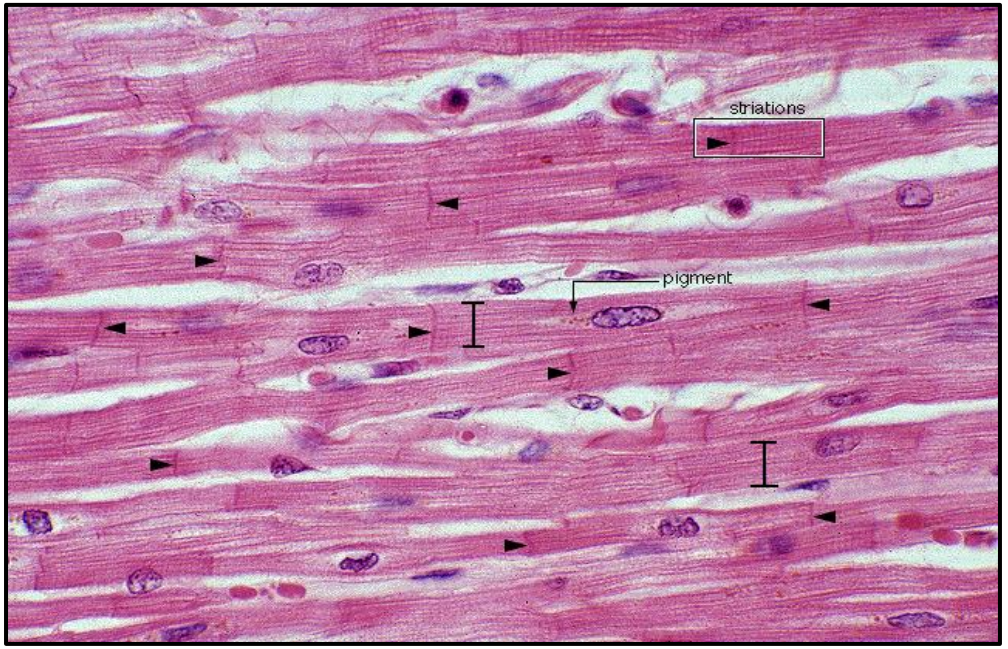
Histology of the Heart

- 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).





PRACTICAL - 2

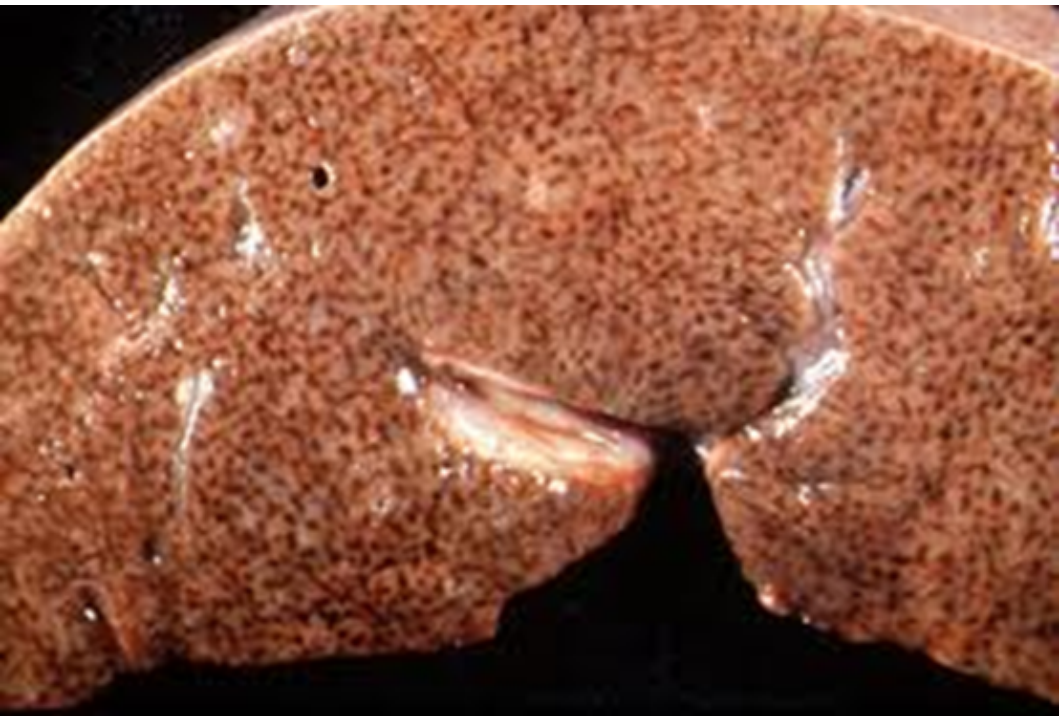
Chronic venous congestion of the liver (seen in right sided heart failure)

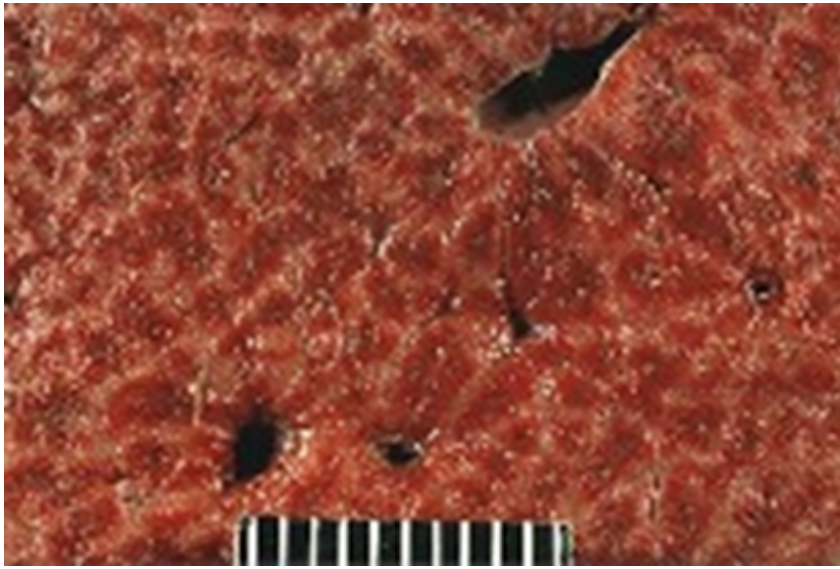
Additional information
Hyperemia & Congestion

- ***Hyperemia*** is an *active process* resulting from tissue inflow because of arteriolar dilation, e.g. skeletal muscle during exercise or at sites of inflammation. The affected tissue is redder because of the engorgement of vessels with oxygenated blood.

- ***Congestion*** is a *passive process* resulting from impaired outflow from a tissue. It may be systemic e.g. cardiac failure, or local e.g. an isolated venous obstruction. The tissue has a blue-red color *(cyanosis)*, due to accumulation of deoxygenated hemoglobin in the affected tissues.

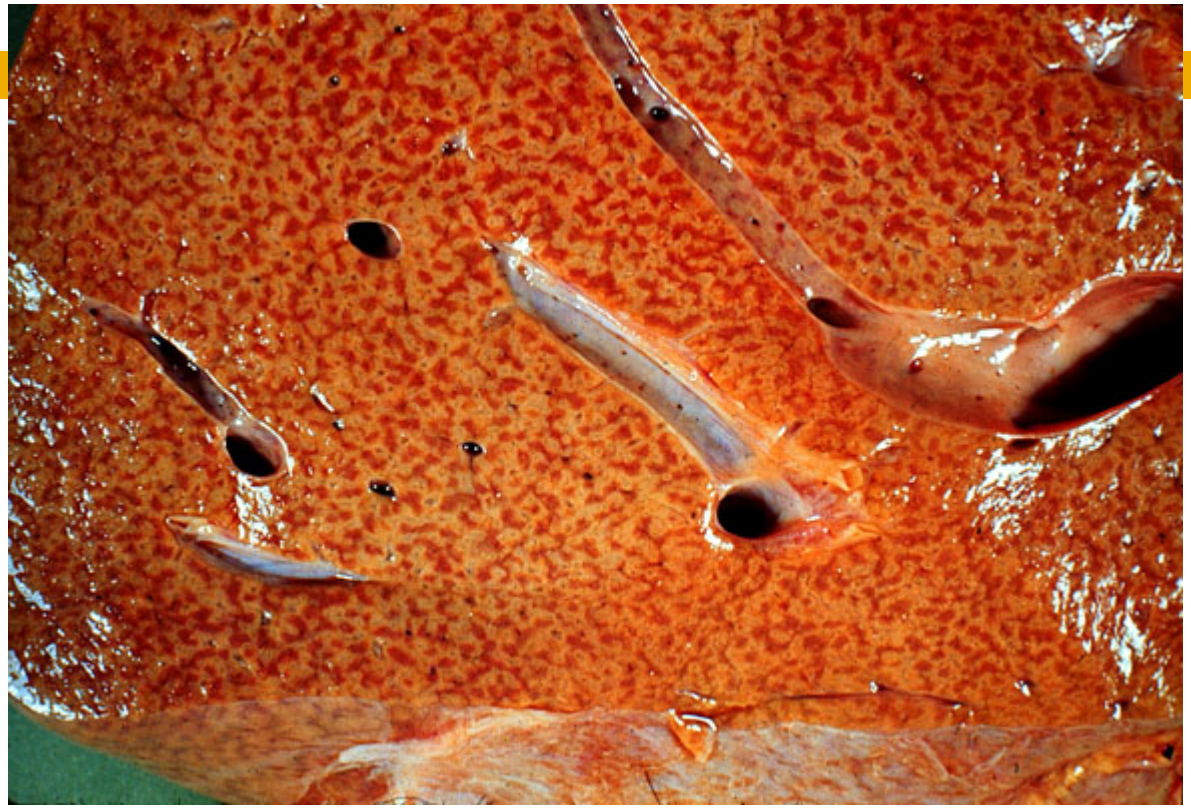
Chronic Congestion of the Liver - HPF





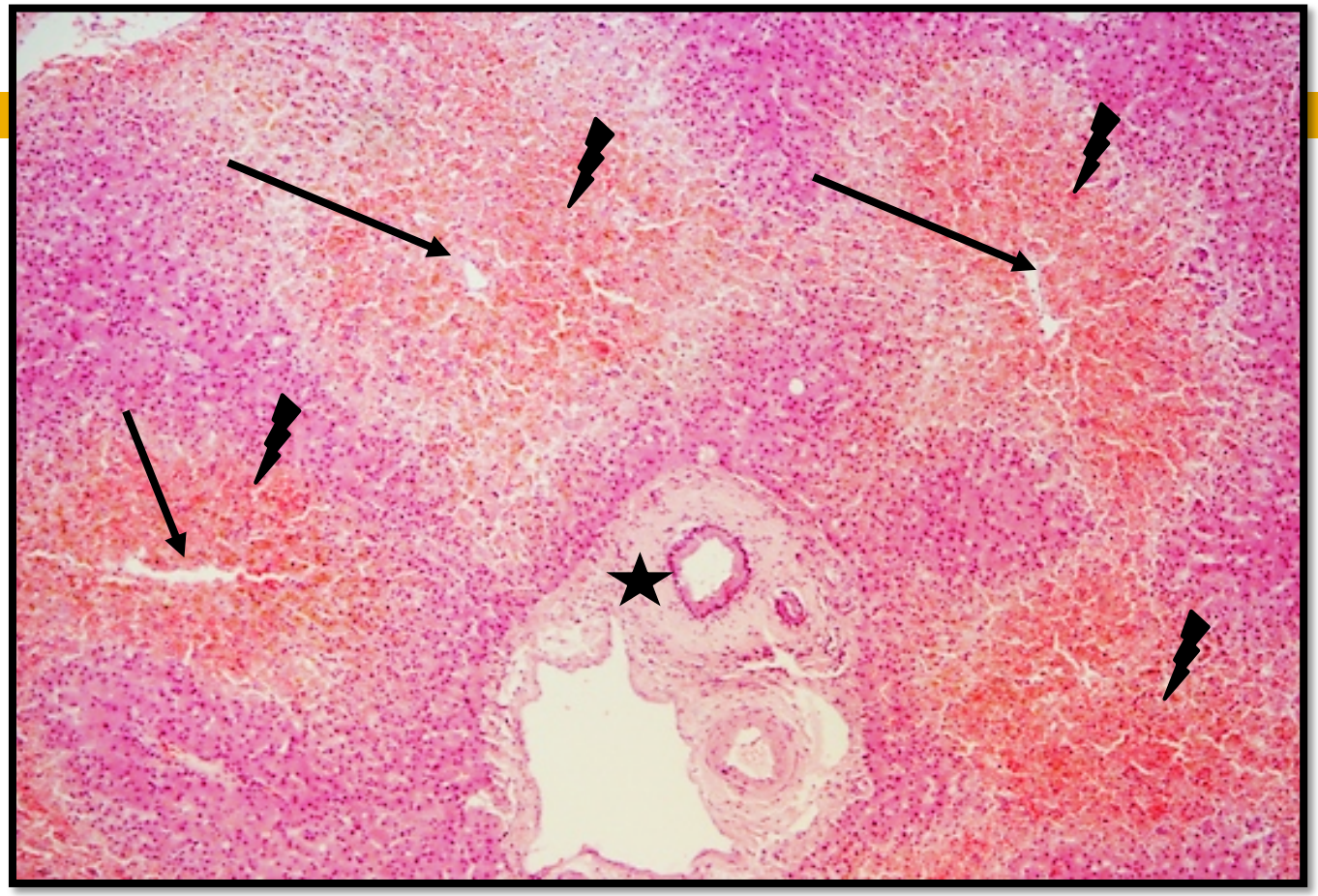
In chronic passive congestion of the liver: on cut surface of liver shows red dots. These red dots represent venous congestion with accumulation of RBC's in centrilobular regions. And they are surrounded by grey/ tan /pale zones of uncongested normal liver. This appearance is described as nutmeg liver. The most common cause is passive congestion secondary to right sided heart failure.

Chronic Congestion of the Liver - CS



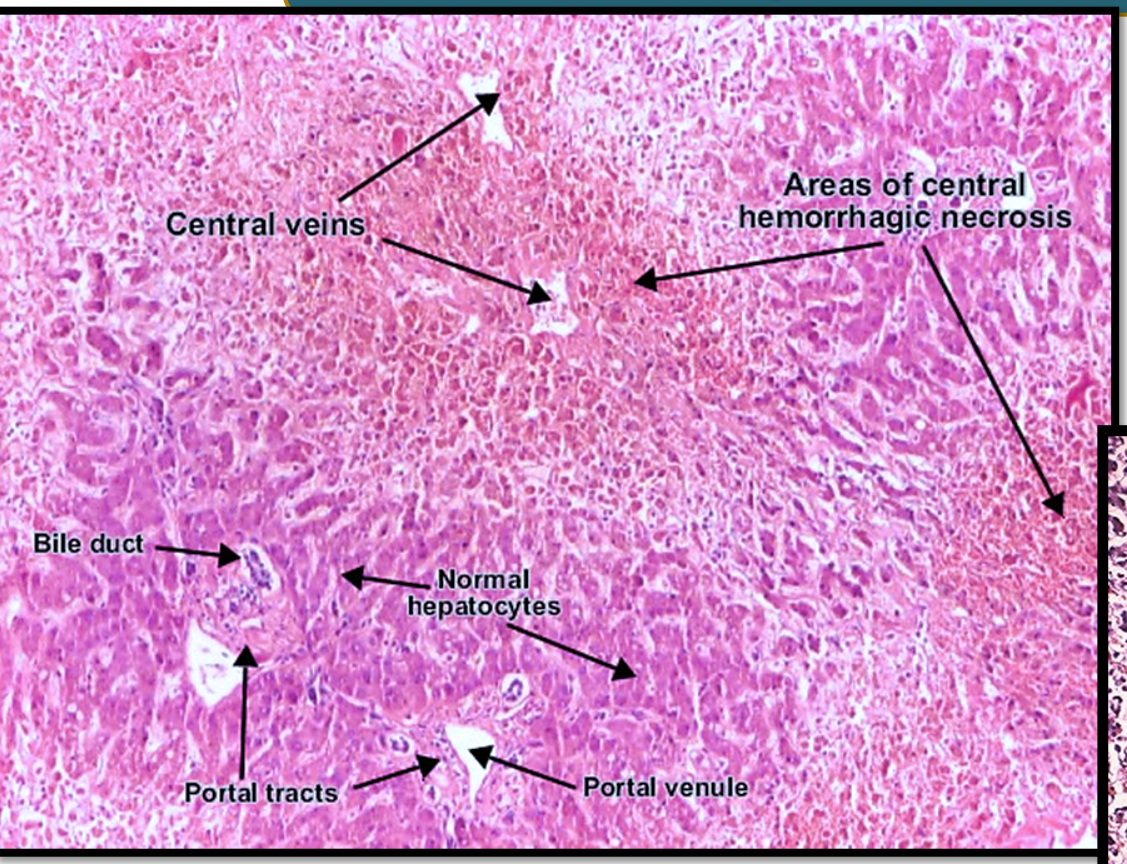
A gross view of nutmeg appearance of liver characteristic of centrolobular necrosis or passive congestion of the liver.

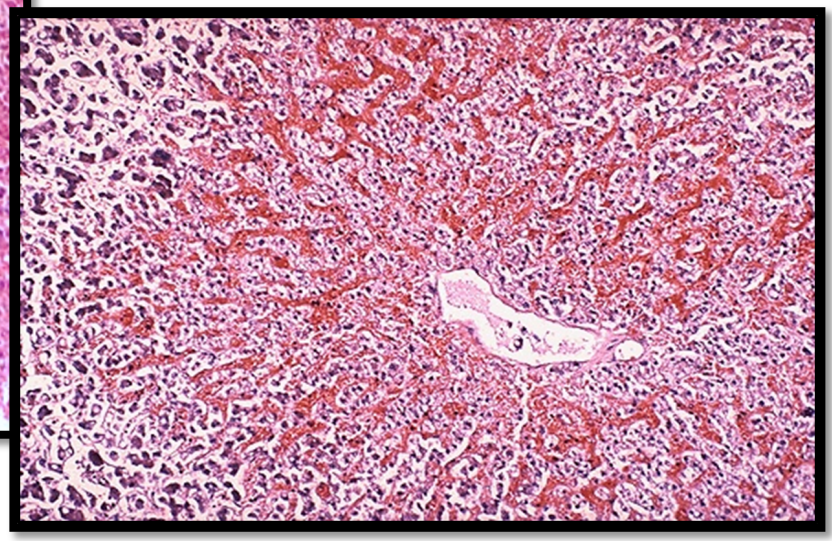
Chronic Congestion of the Liver - CS



Microscopically, there is passive congestion in the central portion of each liver lobule. The central veins (which are present in the center of each liver lobule) become congested and dilated (arrows) → leads to congestion in the surrounding sinusoids → which ultimately leads to necrosis of the hepatocytes around the central vein called centrilobular necrosis (lightning arrows). The portal tracts (star) are unaffected.

Chronic Congestion of the Liver - LPF

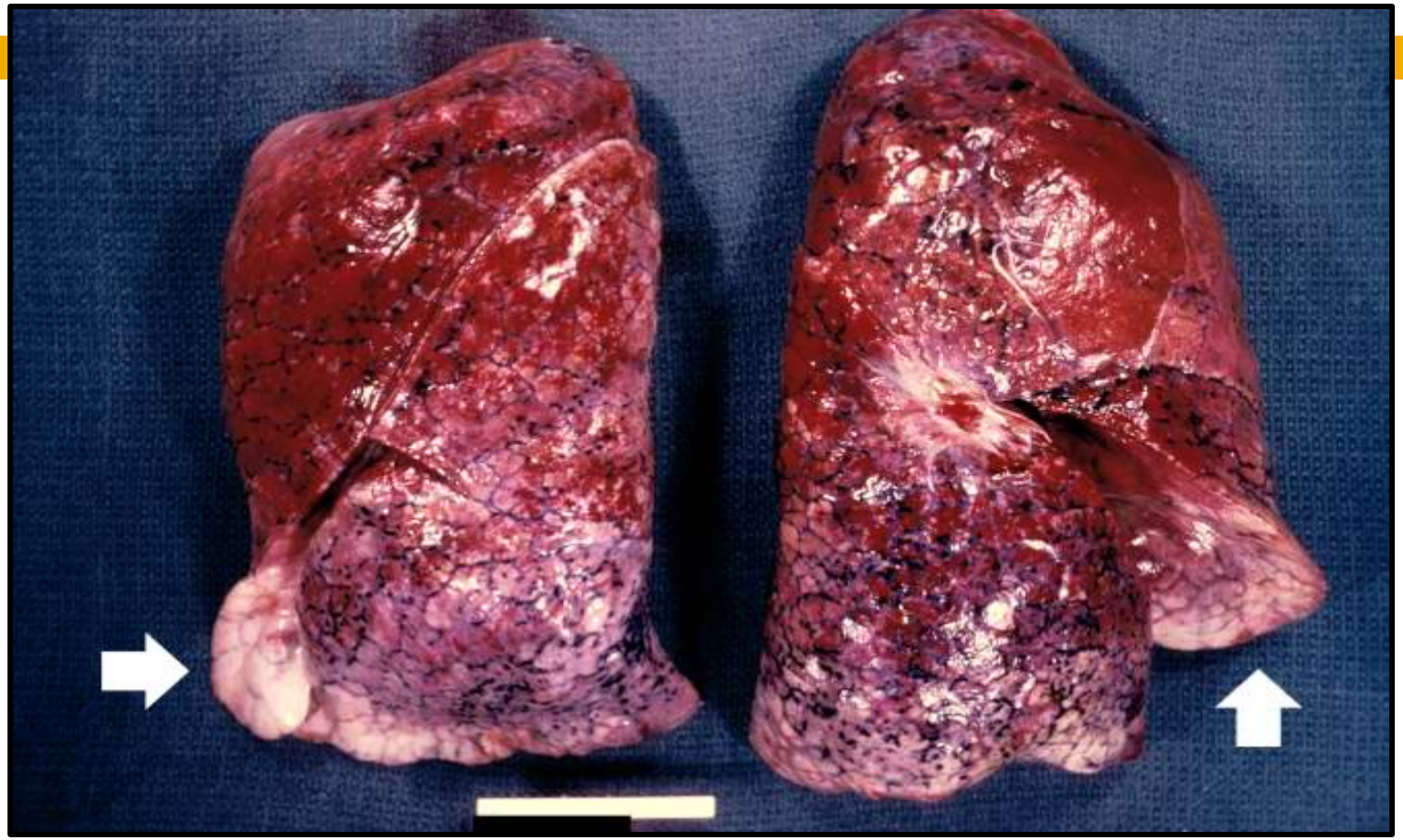




Central veins are dilated and congested surrounded by necrotic hepatocytes (called centrilobular necrosis) with associated hemorrhage and hemosiderin-laden macrophages. In long-standing cases these areas are replaced by fibrosis. This type of fibrosis is called hepatic fibrosis or cardiac cirrhosis.

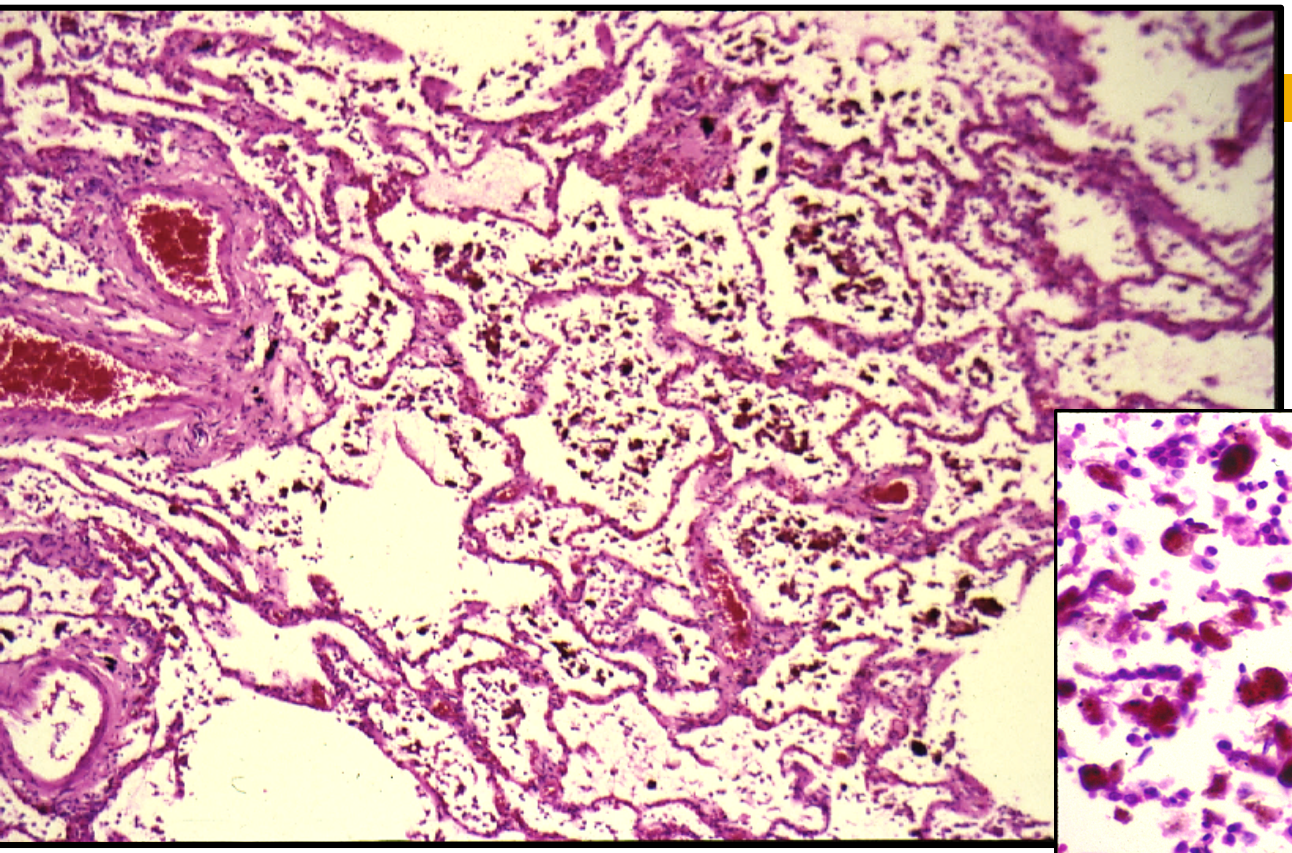
Chronic venous congestion of the lung (seen in left sided heart failure)

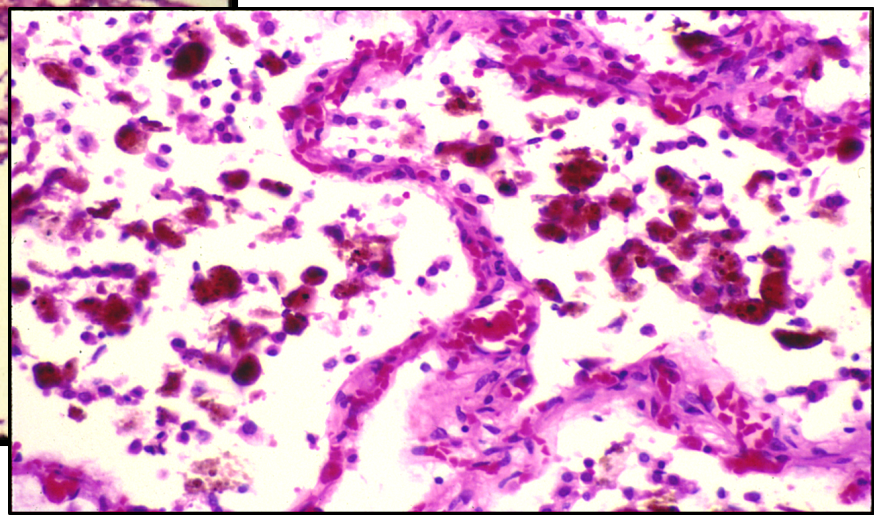
Chronic venous congestion of the lung - Gross



The lungs are enlarged, heavy, 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) The cut surface is hemorrhagic and wet and frothy blood oozes on squeezing.

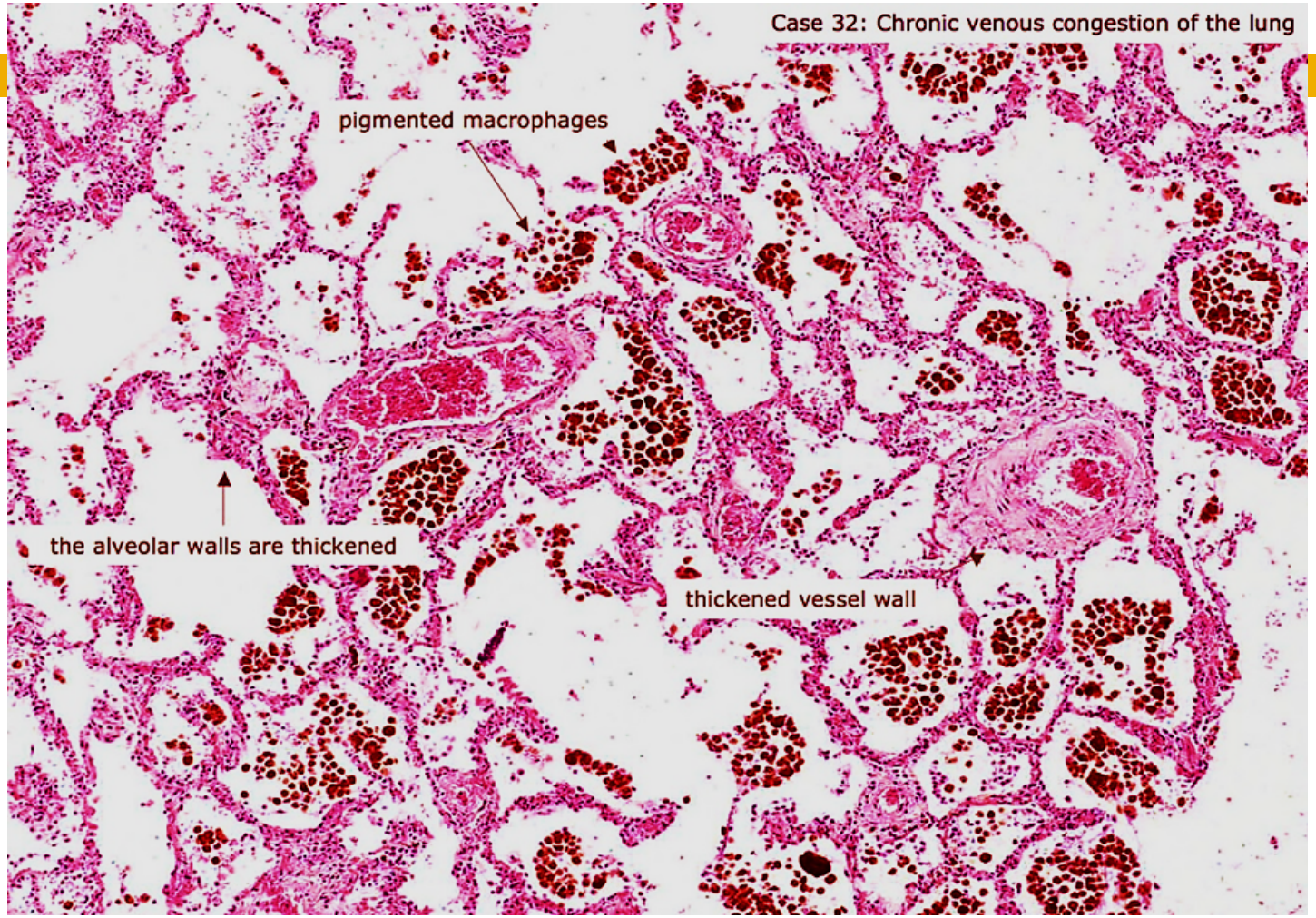
Chronic venous congestion of the lung - LPF



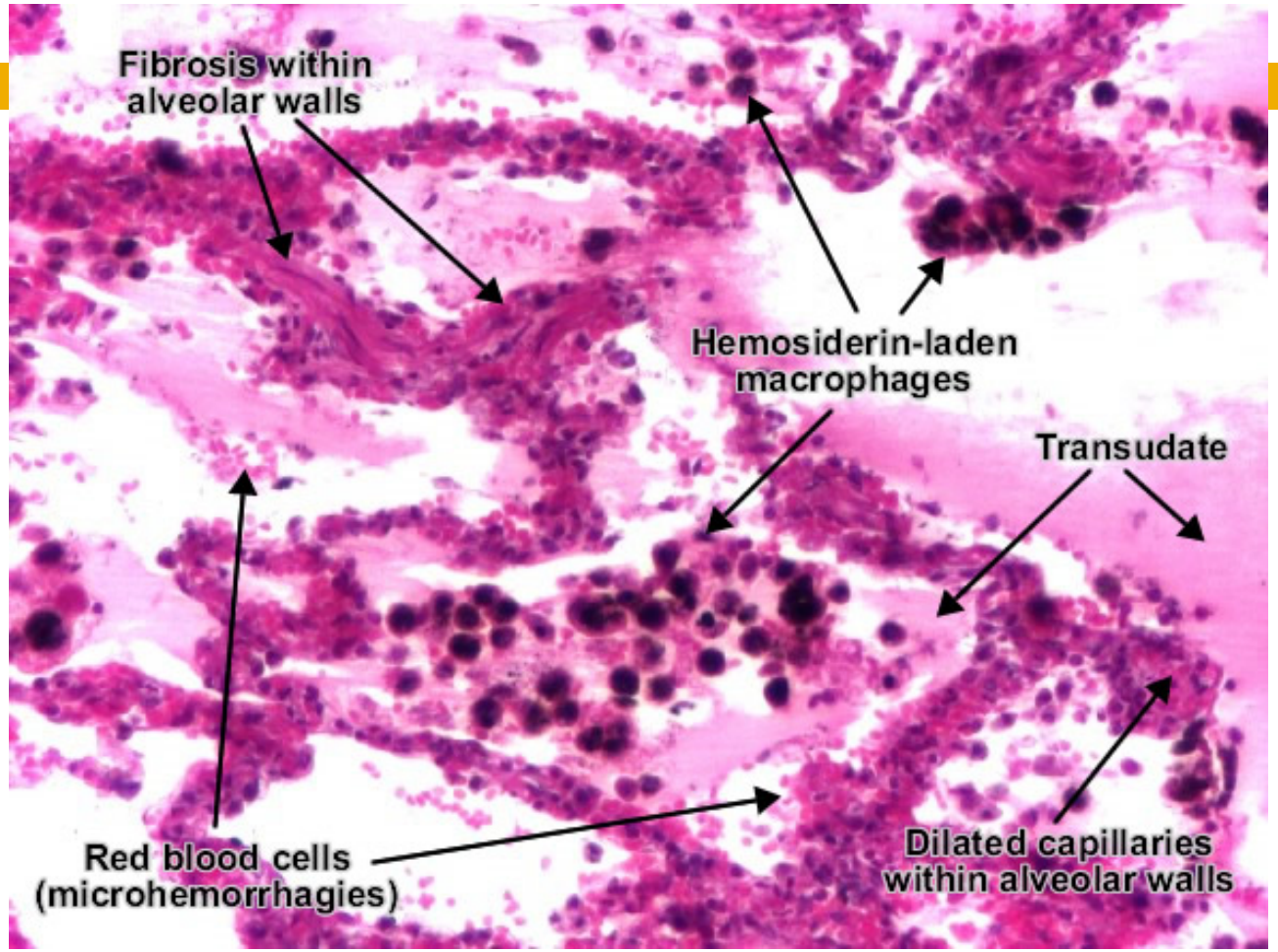


The alveolar walls are thickened by dilated and engorged capillaries and edema fluid. The alveoli are filled with edematous fluid, red blood cells and many alveolar macrophages which are filled with hemosiderin pigment derived from red cells breakdown. These alveolar macrophages are called heart failure cells. With time the alveolar septa become fibrosed.

Chronic venous congestion of the lung - LPF

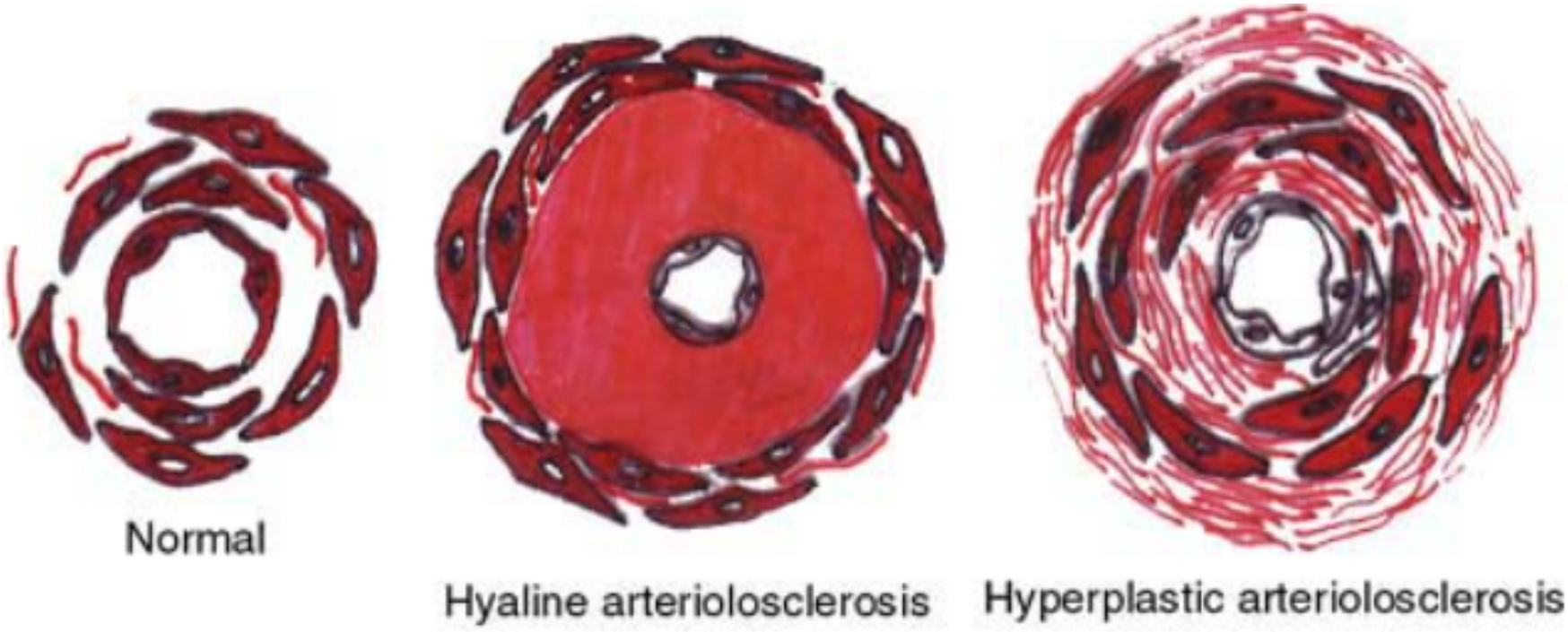


Chronic venous congestion of the lung - HPF

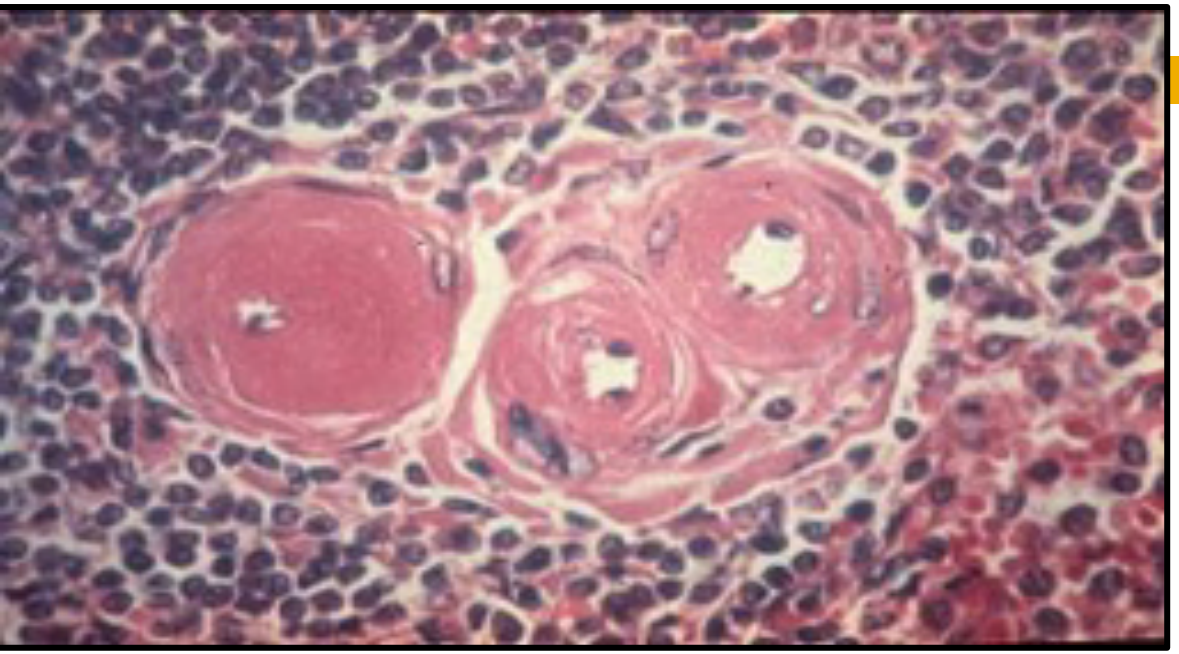


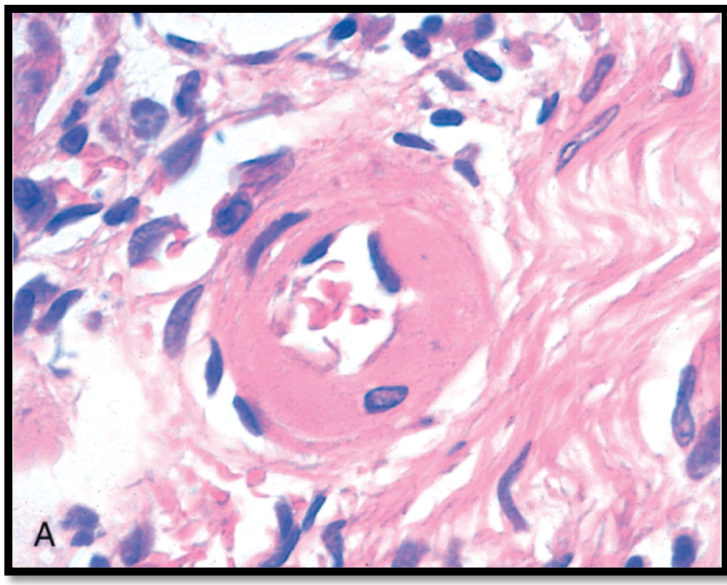
The alveoli contain edematous fluid, red blood cells and large alveolar macrophages (heart failure cells), which are filled with haemosiderin pigment derived from red cells breakdown.

BLOOD VESSELS IN HYPERTENSION


Normal
Hyaline arteriolosclerosis
Hyperplastic arteriolosclerosis

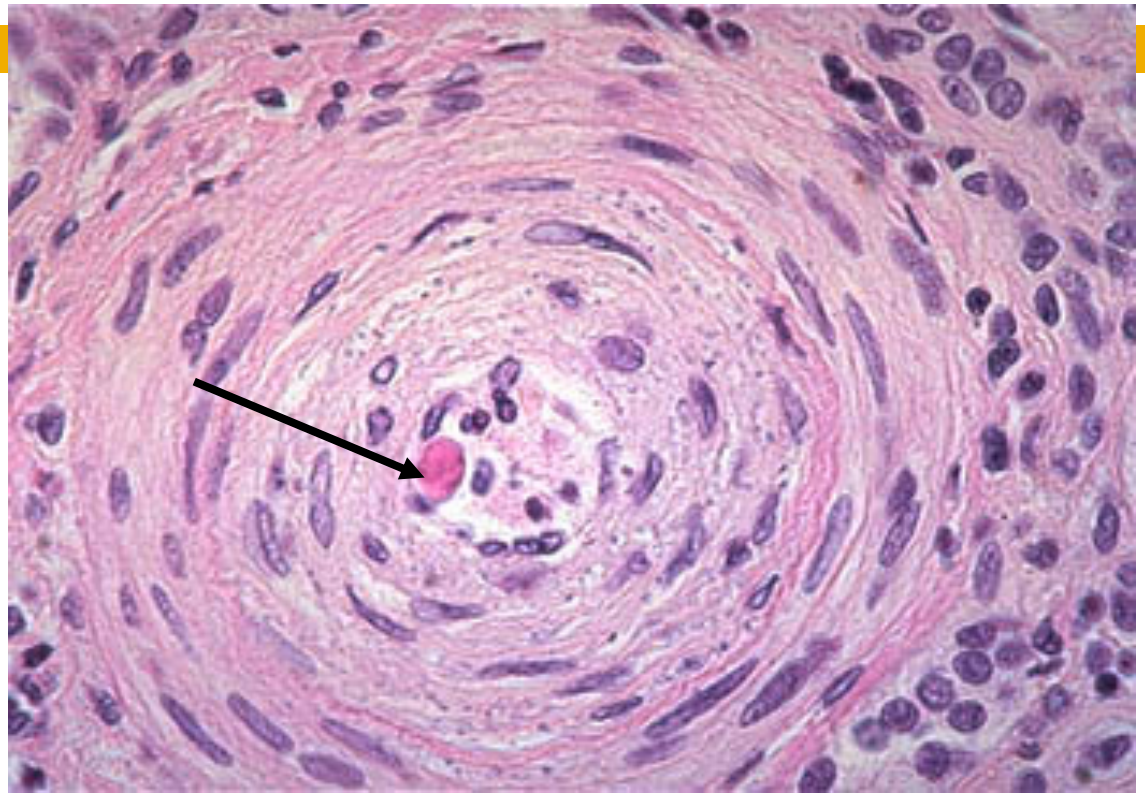
Hyaline arteriolosclerosis - HPF





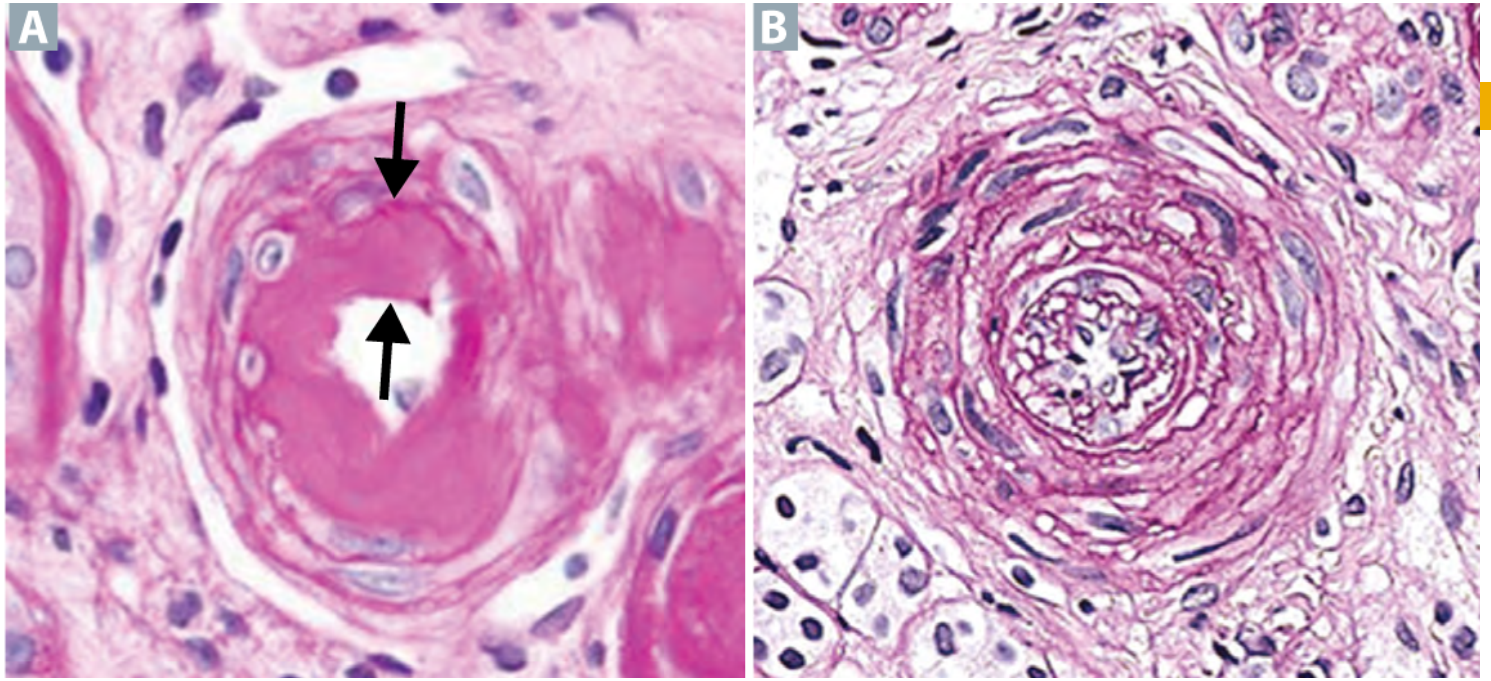
Hyaline arteriolosclerosis (arterial hyalinosis or arteriolar hyalinosis): *it is the thickening of the walls of arterioles by the deposits that appear as homogeneous pink hyaline material. It is commonly found in diabetics, hypertensives and elderly.*

Hyperplastic arteriolosclerosis - HPF



***Hyperplastic arteriolosclerosis**: It is characterized by thickening of the arteriolar wall due to the concentric proliferation of smooth muscle cells, giving the arterioles an "onion skin" appearance. These changes represent an adaptive response of arterioles to severe ("malignant") hypertension. The onion-skinning causing luminal obliteration of the arteriole (arrow). May be associated with necrotizing arteriolitis and fibrinoid necrosis of the blood vessel.*

Hyaline arteriolosclerosis - HPF



A. Hyaline arteriolosclerosis

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

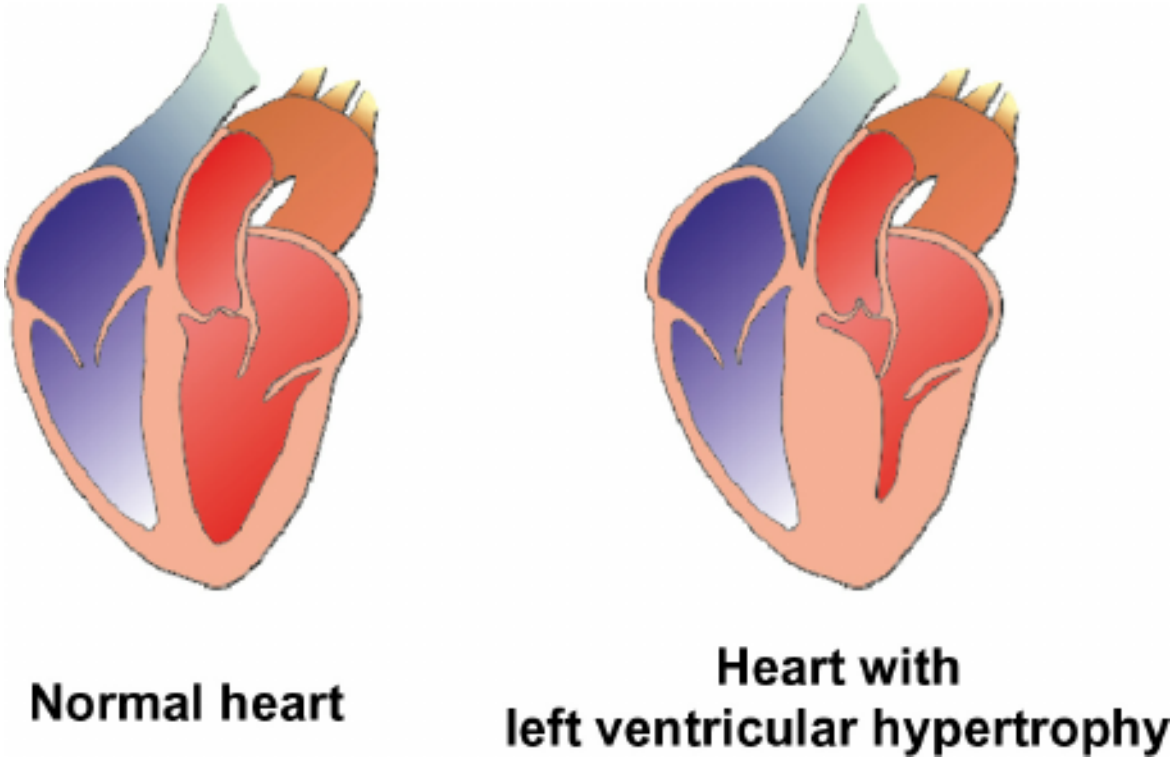
B. Hyperplastic arteriolosclerosis: ***is a type of small vessel arteriosclerosis, it is characteristic of malignant hypertension. The "onionskinning" is causing luminal obliteration. Hyperplastic arteriolosclerosis is often accompanied by fibrinoid necrosis of the arterial intima and media (most prominent in the kidney and can lead to acute kidney failure).***

LEFT VENTRICULAR HYPERTROPHY

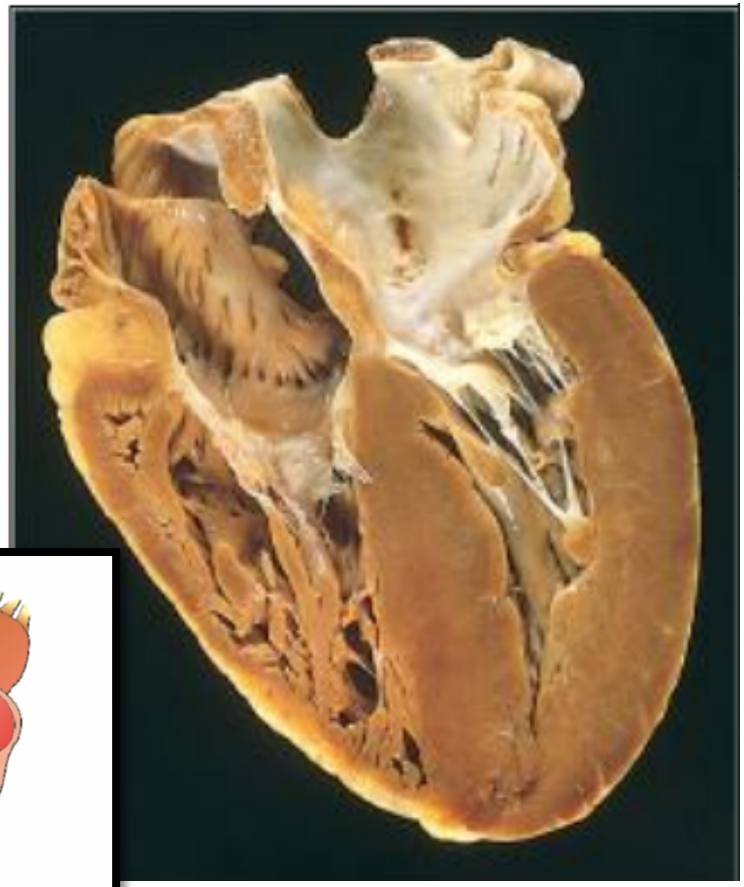
Causes of ventricular hypertrophy

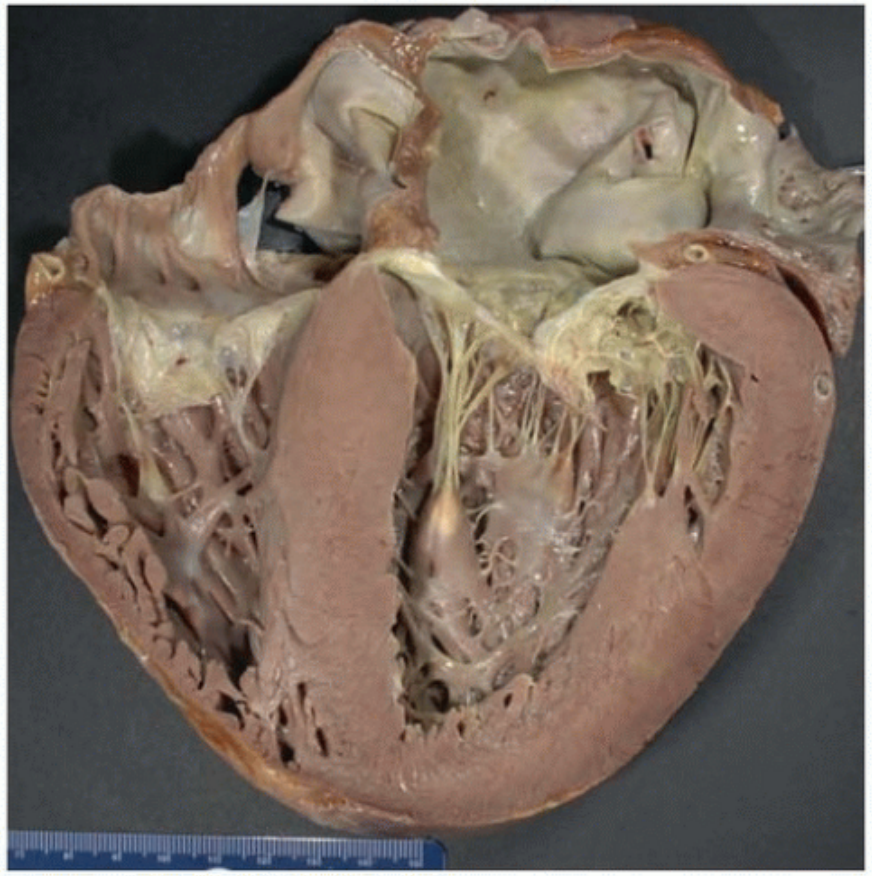
Common causes of left ventricular hypertrophy :

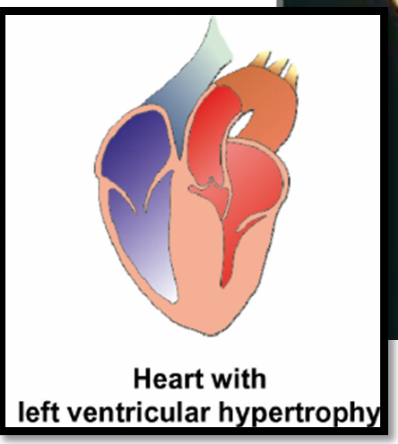
- **Long standing poorly controlled systemic hypertension**
- **Aortic valve stenosis**



Left ventricular hypertrophy - Gross



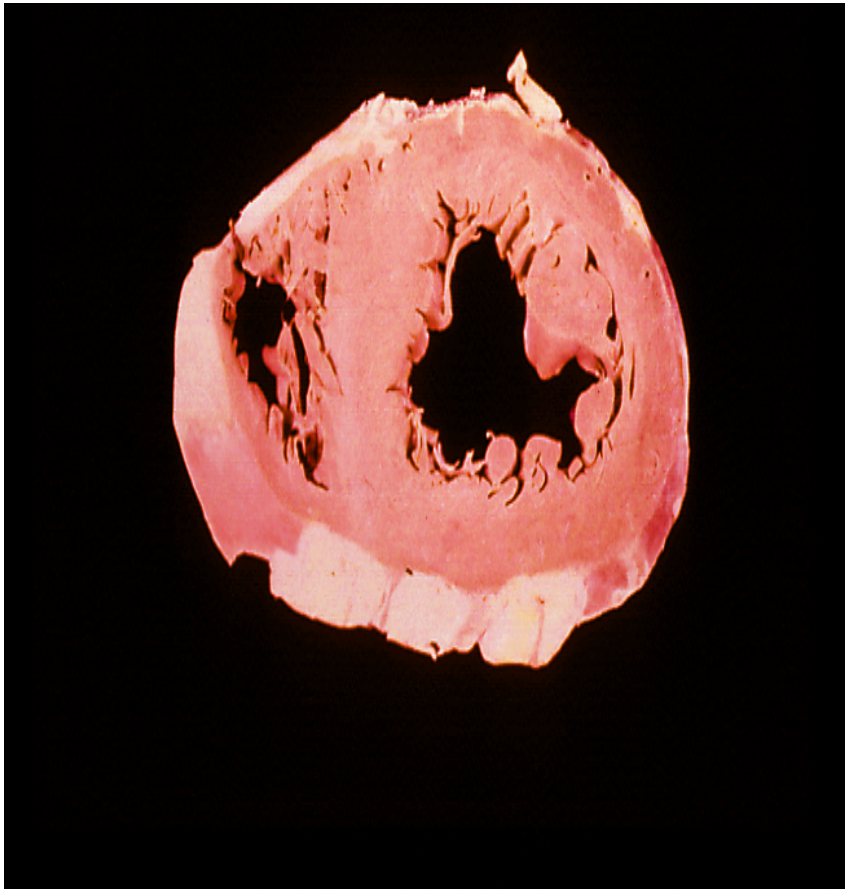




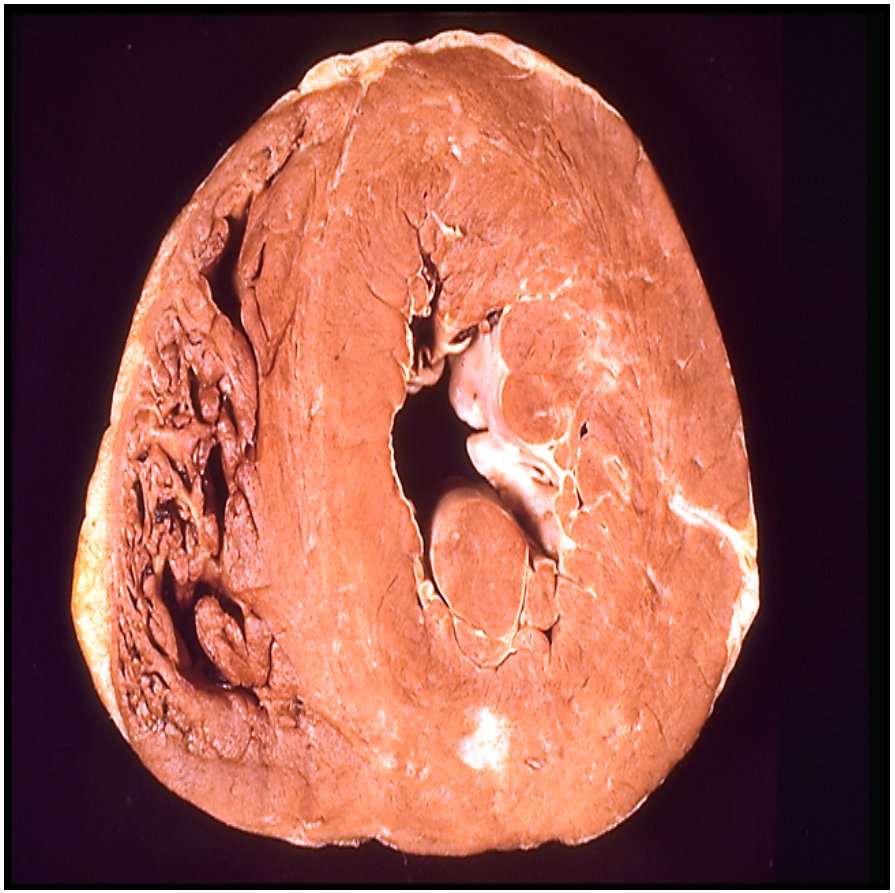
Heart with
left ventricular hypertrophy

Heart from a hypertensive patient. The left ventricle wall is very thick. 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 left side of the heart to induce the hypertrophy

Normal and hypertrophied left ventricle – cross section

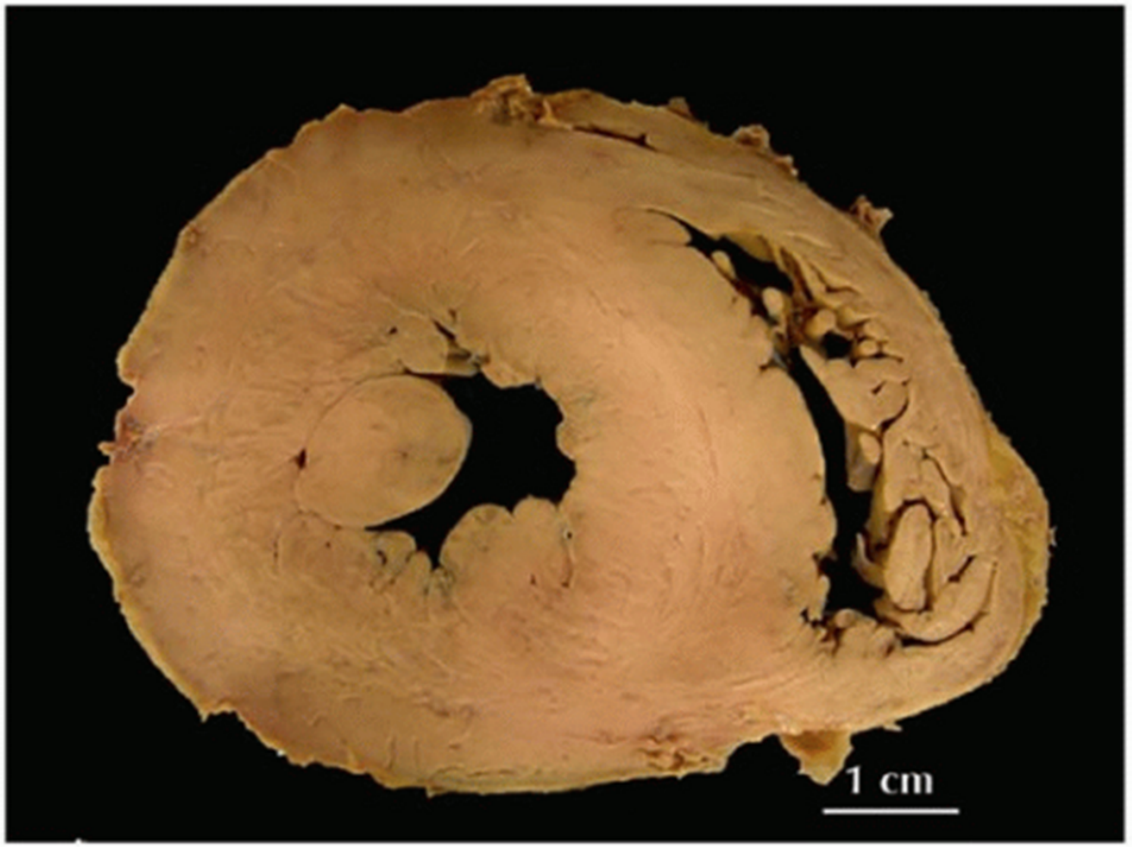


Normal ventricles



Left ventricular hypertrophy

Left ventricular hypertrophy - Gross

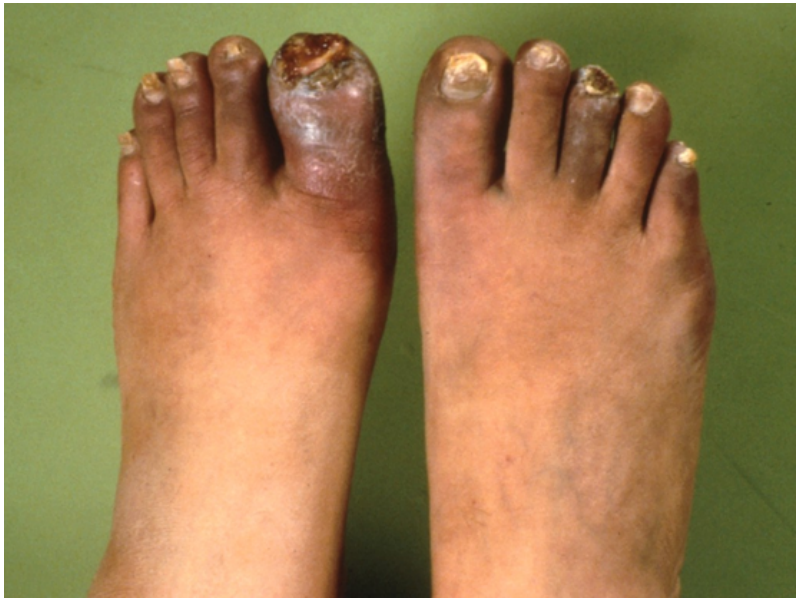


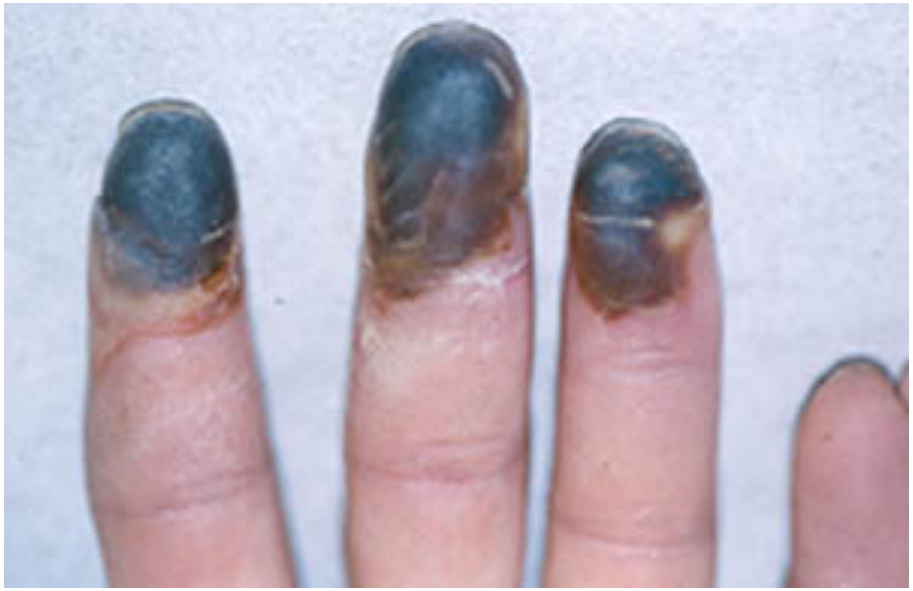
This cross section view of the heart shows the left ventricle in the left of the picture. The heart is from a severe hypertensive. The left ventricle is grossly thickened. The myocardial fibers have undergone hypertrophy.

Thromboangitis oblitrans (Buerger's disease)

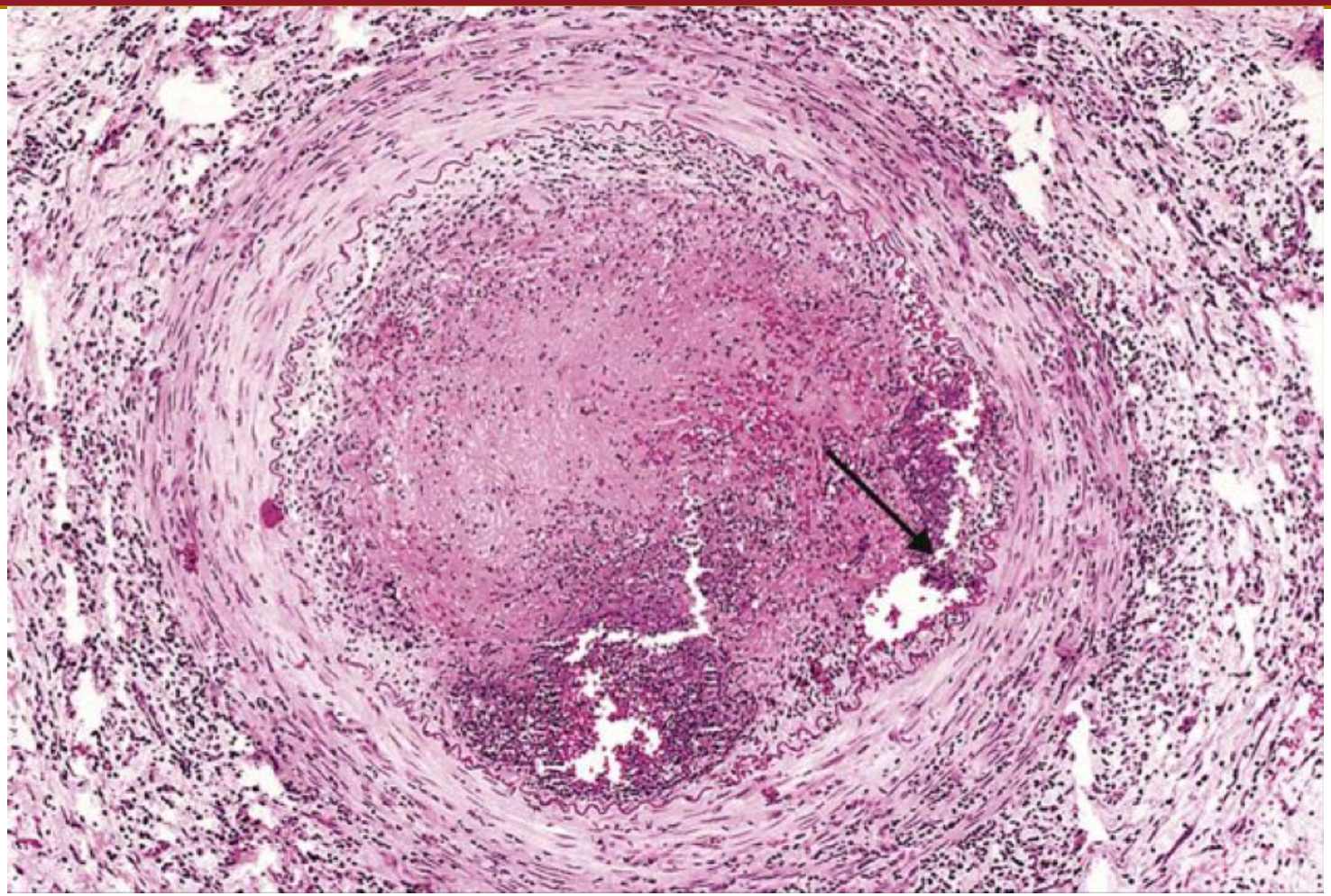
Boehringer Ingelheim

Thromboangitis oblitrans (Buerger`s disease)



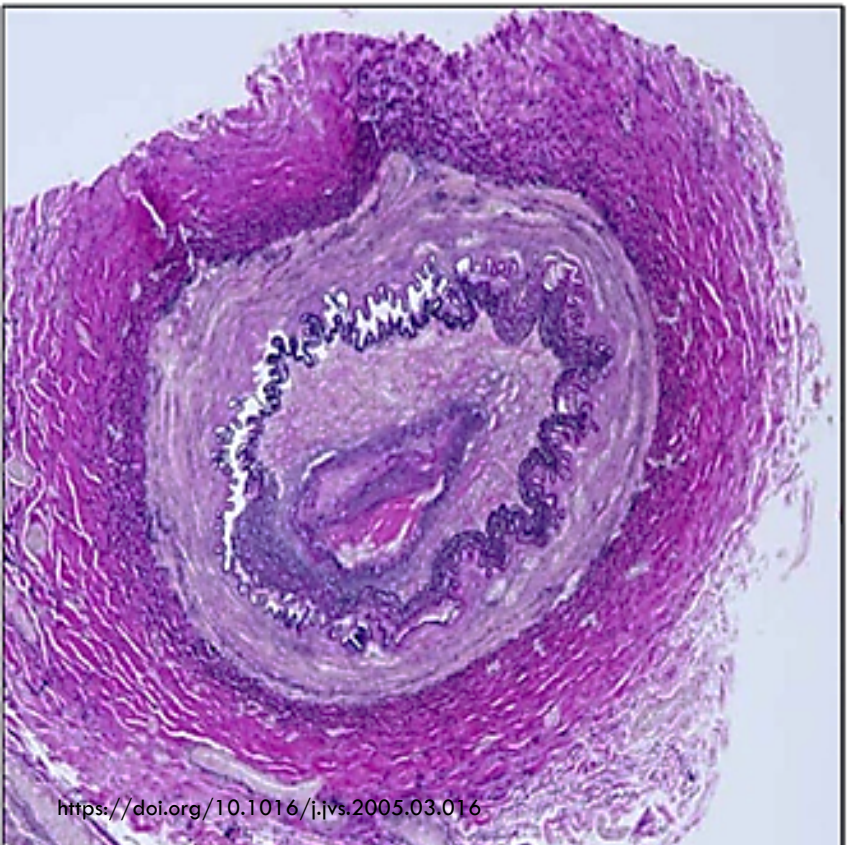


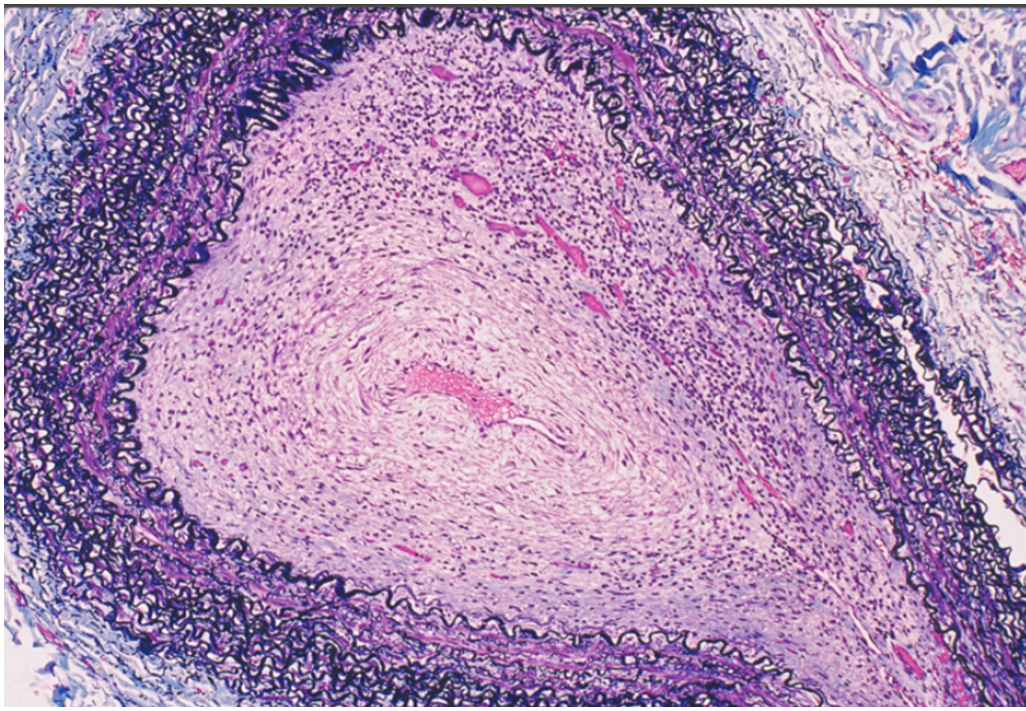
ACTIVE STAGE OF THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE)



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

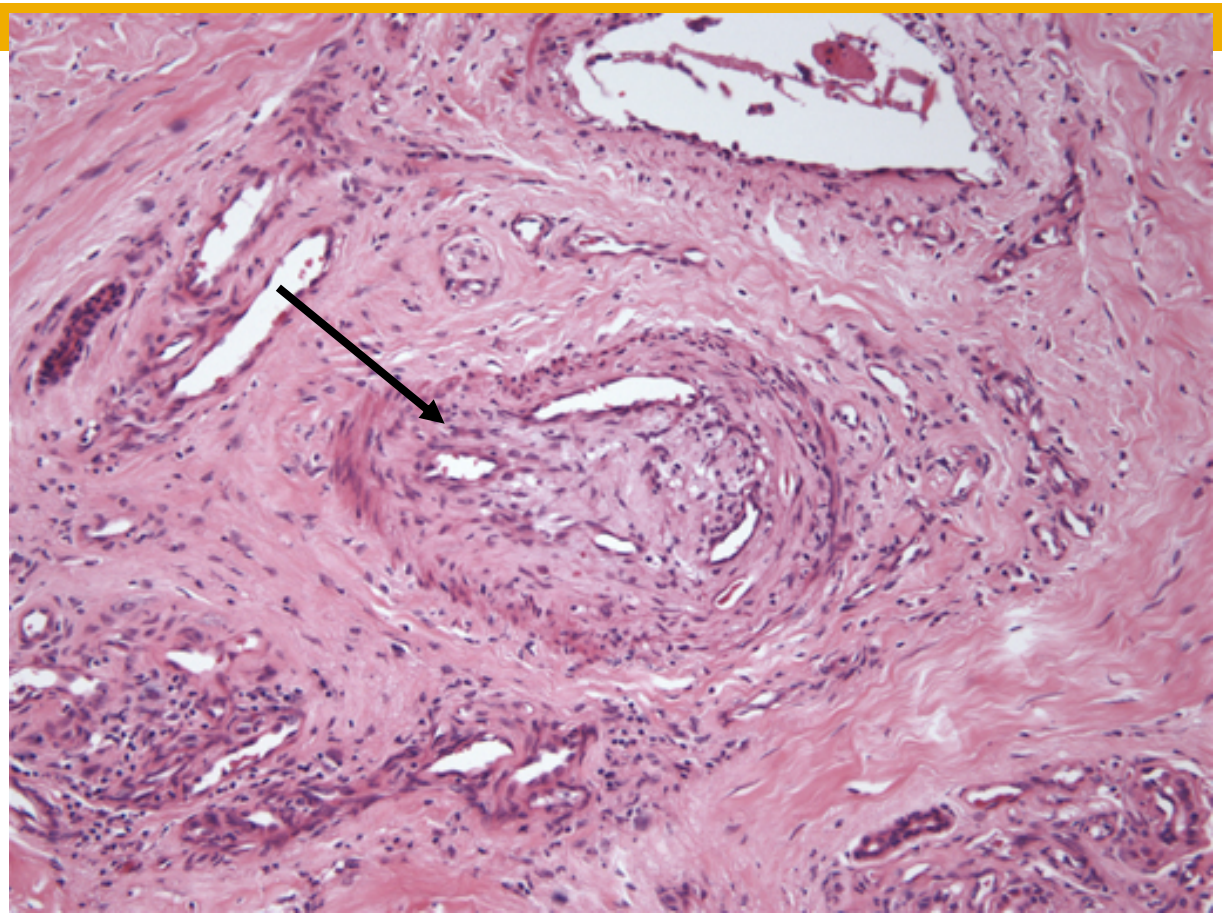
Chronic stage Thromboangitis obliterans (Buerger's disease)





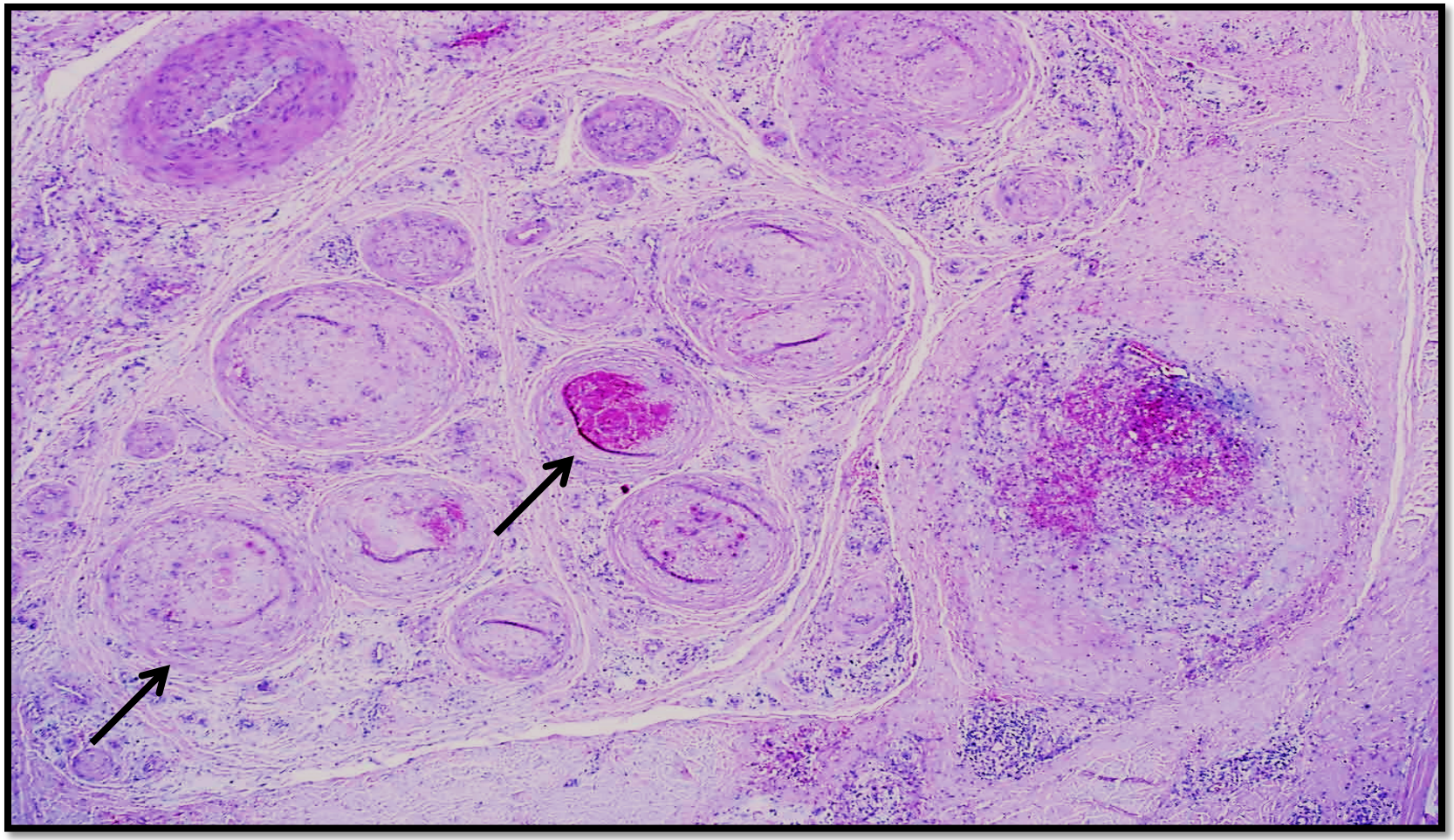
Thromboangiitis obliterans (Buerger's disease) is a non atherosclerotic, segmental, inflammatory, vaso-occlusive disease that affects the small- and medium-sized arteries and veins of the upper and lower extremities. Both pictures show cut-section of artery with near total occlusion of the lumen by a fibrotic thrombus. There are inflammatory cells in the fibrotic occlusive thrombus in the picture on the right (PAS stain & trichrome elastic stain).

Chronic stage Thromboangitis obliterans (Buerger's disease)



Blood vessel shows chronic stage of TAO with occlusive organized thrombi (arrow) with recanalization and fibrosis around the blood vessel.

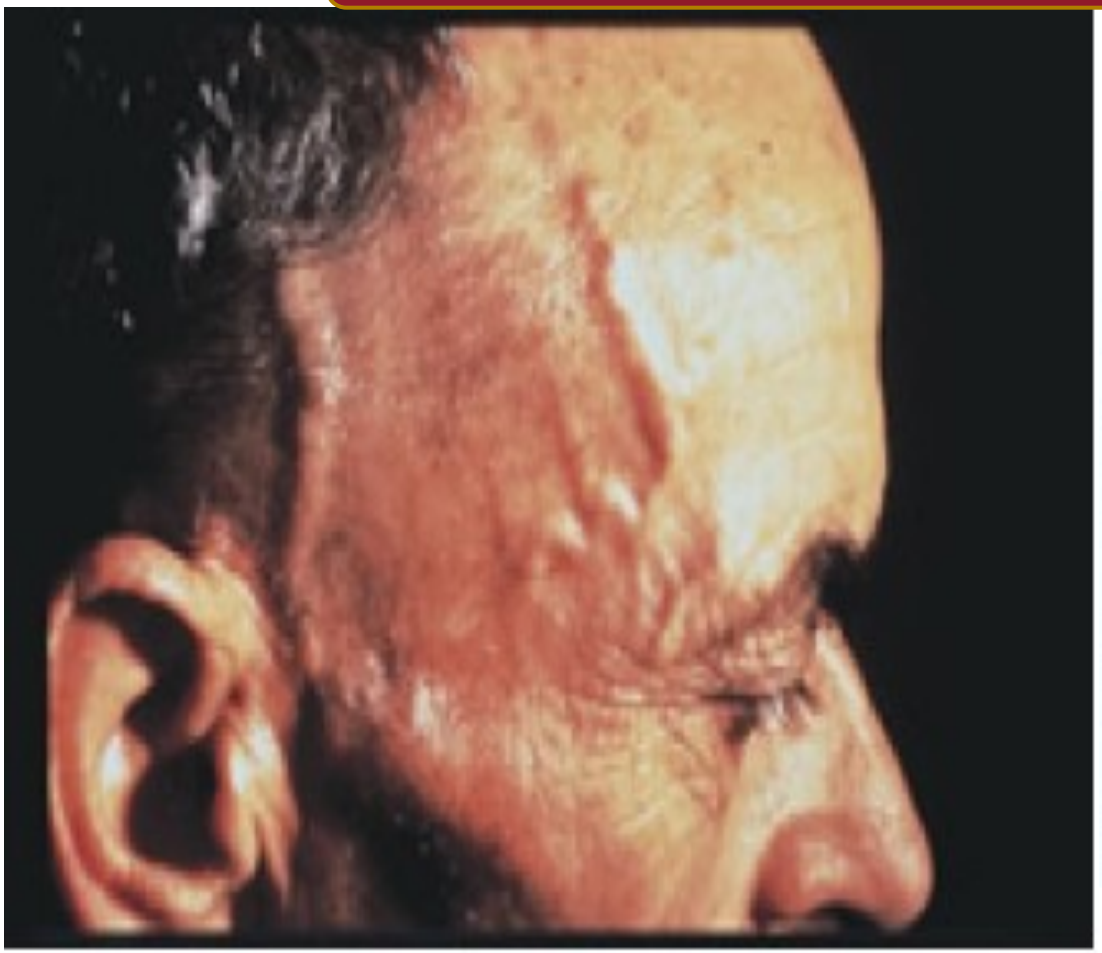
THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE)

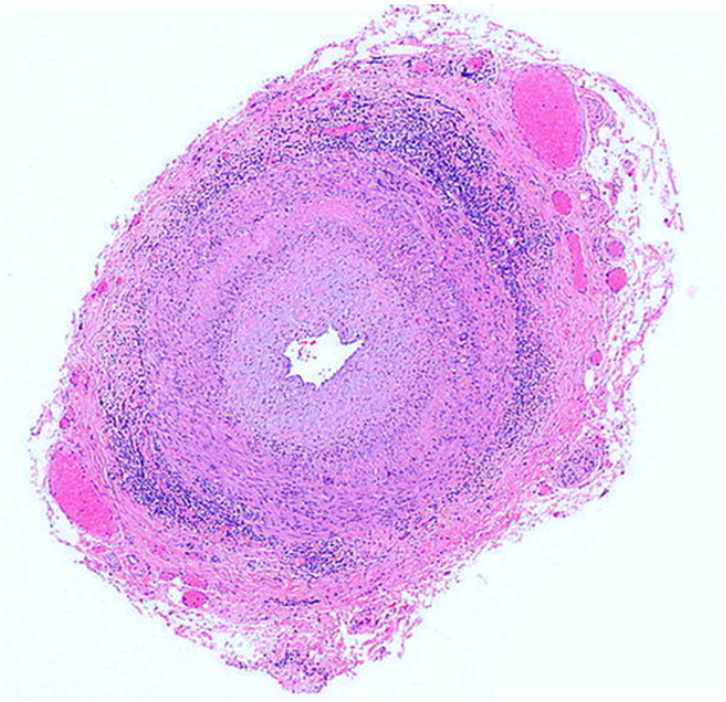


Several blood vessels showing occlusive organized thrombi (arrow). Some vessels show chronic inflammatory cell infiltration in their wall. There is fibrosis in the surrounding tissue.

GIANT CELL (TEMPORAL) ARTERITIS

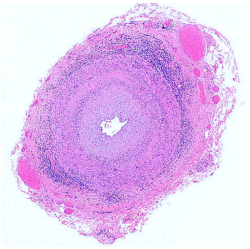
GIANT CELL / TEMPORAL ARTERITIS



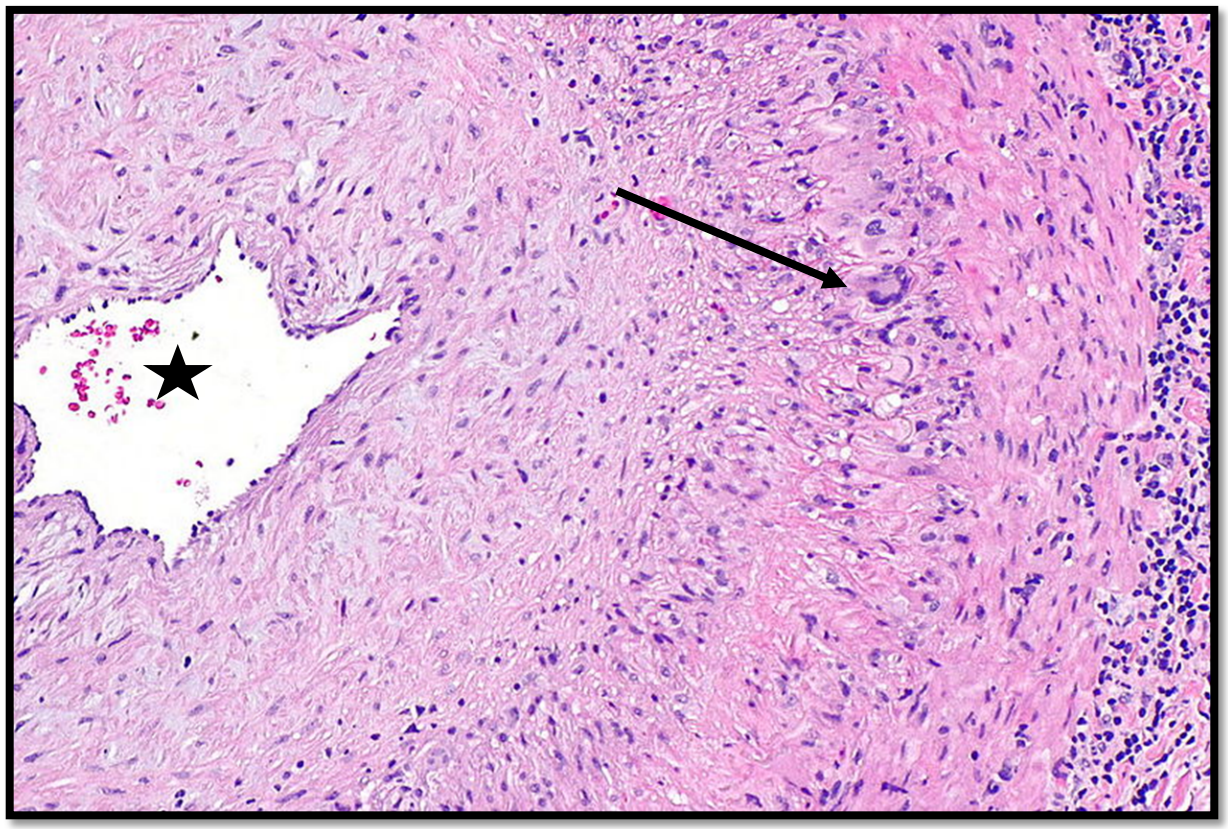


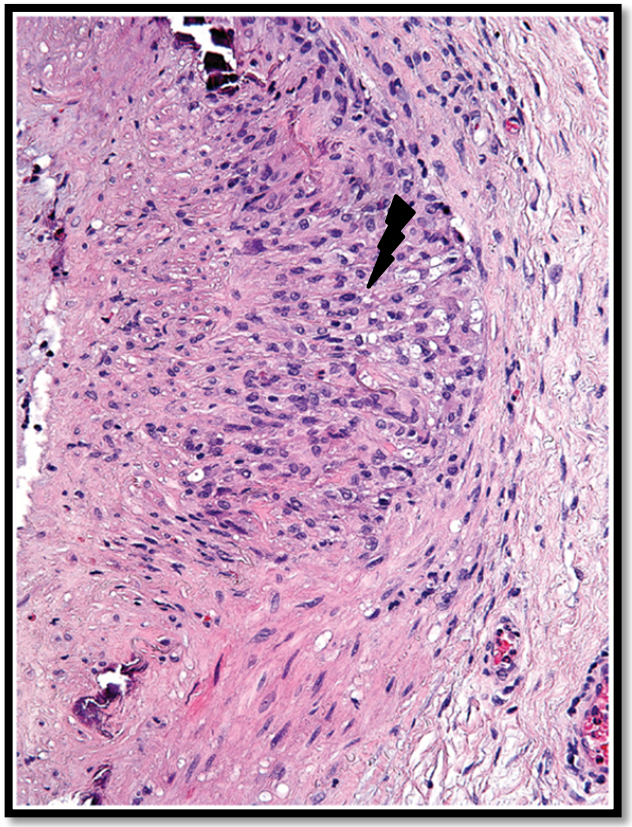
A. Tender and thickened scalp veins

B. Cut section of temporal artery showing circumferential chronic lymphocytic inflammation/ arteritis



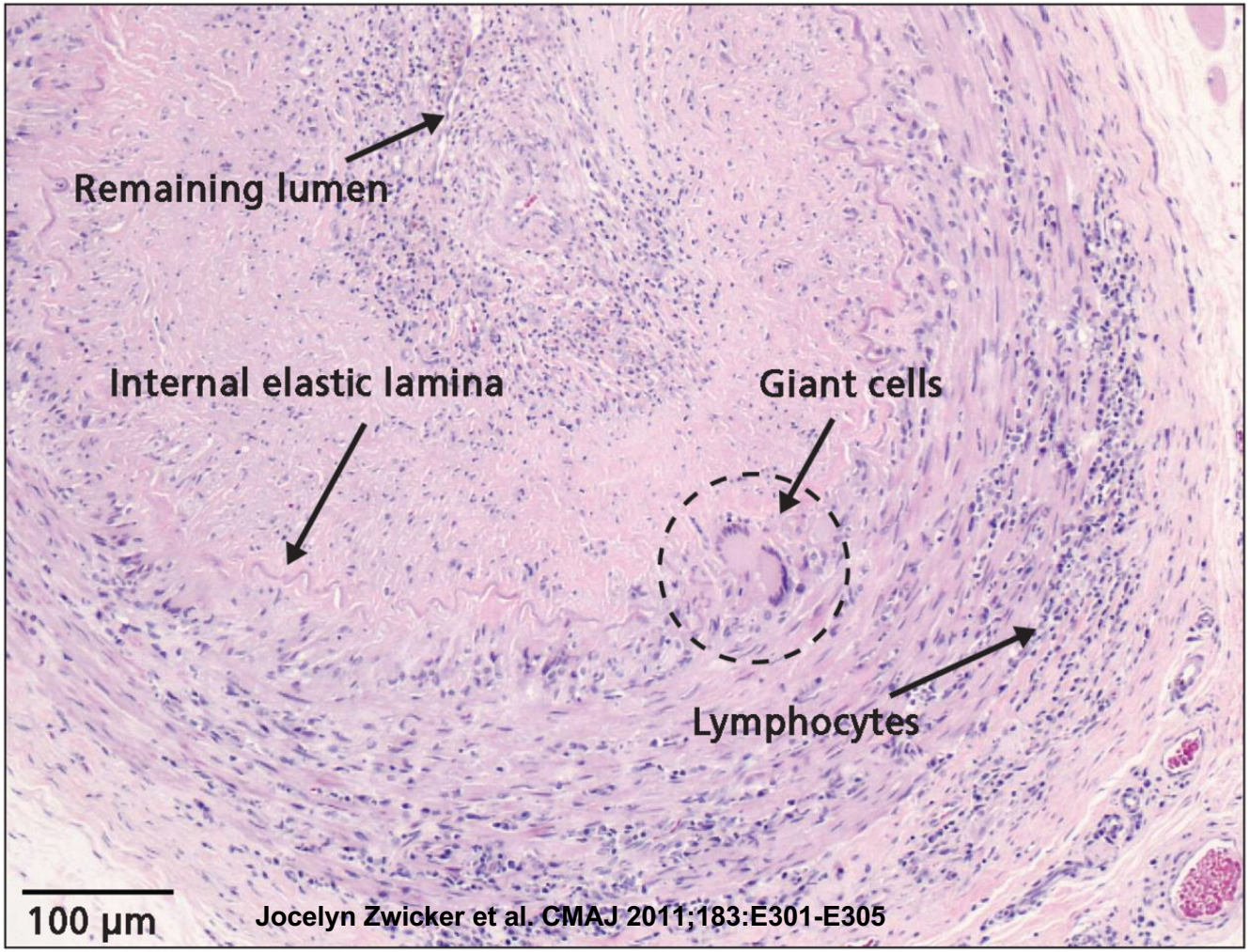
GIANT CELL (TEMPORAL) ARTERITIS - HPF





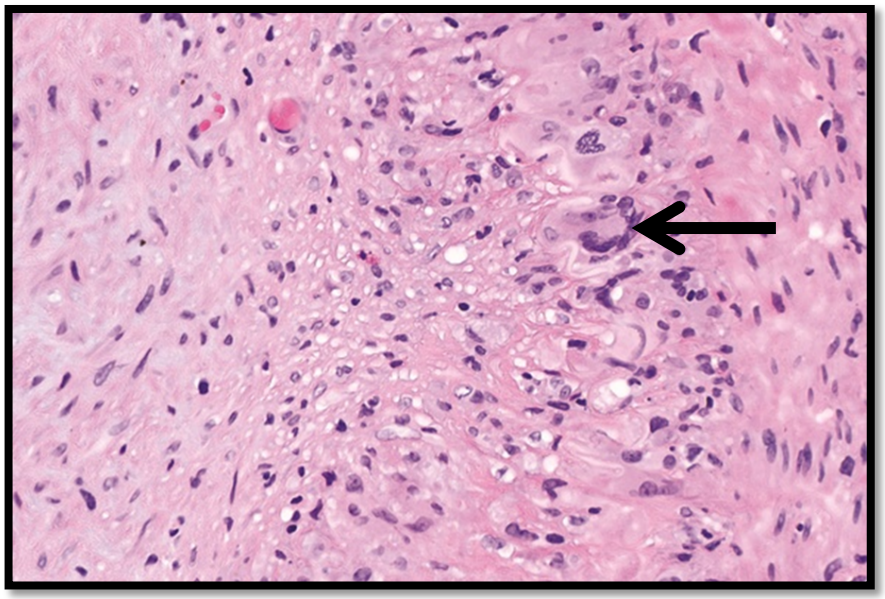
Temporal artery showing lymphocytic inflammation, granuloma (lightning arrow) and multinucleated giant cells (arrow). The lumen (star) of the artery shows narrowing because reactive intimal fibrosis.

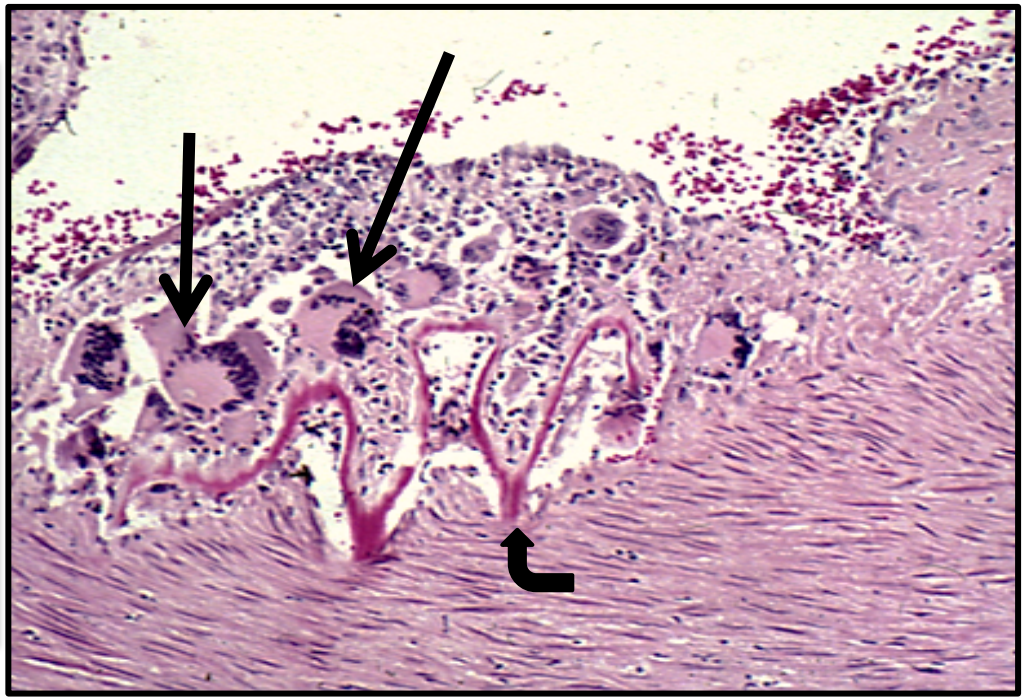
GIANT CELL (TEMPORAL) ARTERITIS



Inflammation of temporal artery showing multinucleated giant cells, fragmentation of the internal elastic lamina and chronic lymphocytic infiltrate consistent with giant cell arteritis. The lumen of the artery is narrowed by intimal fibrosis.

GIANT CELL (TEMPORAL) ARTERITIS - HPF





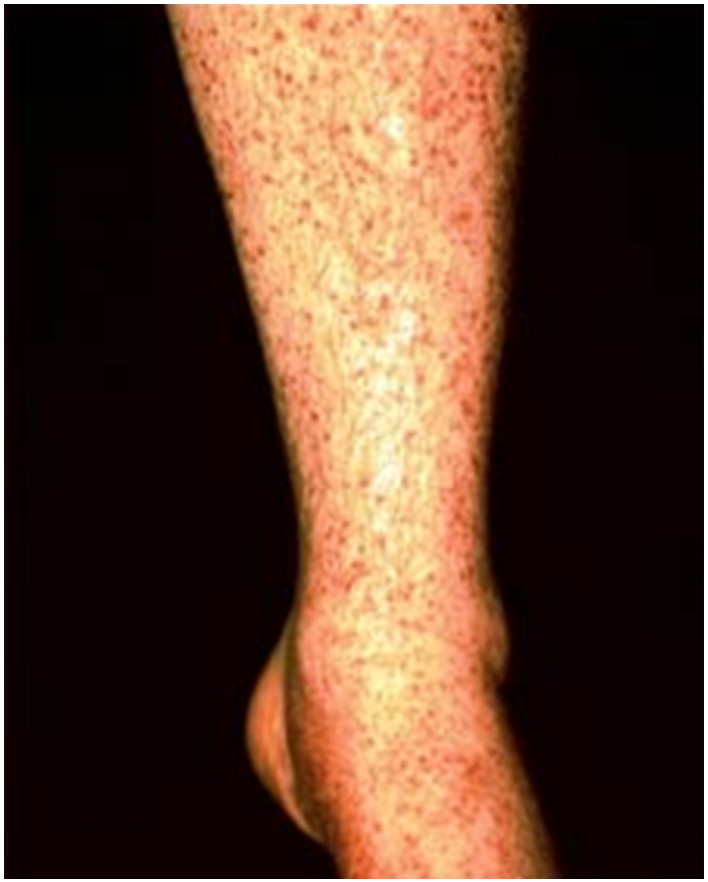
Disruptions of the elastic lamina with inflammation and giant cells.

Segmental inflammatory lesions with intimal thickening , medial granulomatous inflammation with giant cells (arrows) and chronic inflammatory cells and there is fragmentation of the(pink) internal elastic lamina (curved arrow)

LEUKOCYTOCLASTIC / HYPERSENSITIVITY VASCULITIS

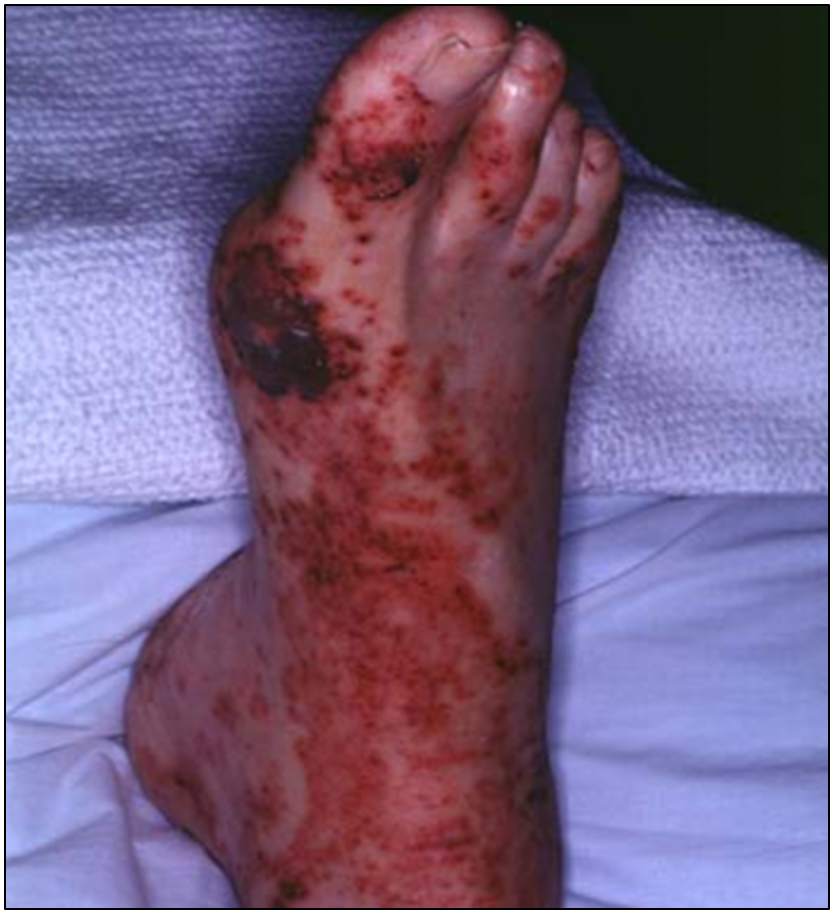
(MICROSCOPIC POLYANGITIS)

Hypersensitivity vasculitis – Clinical sign



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

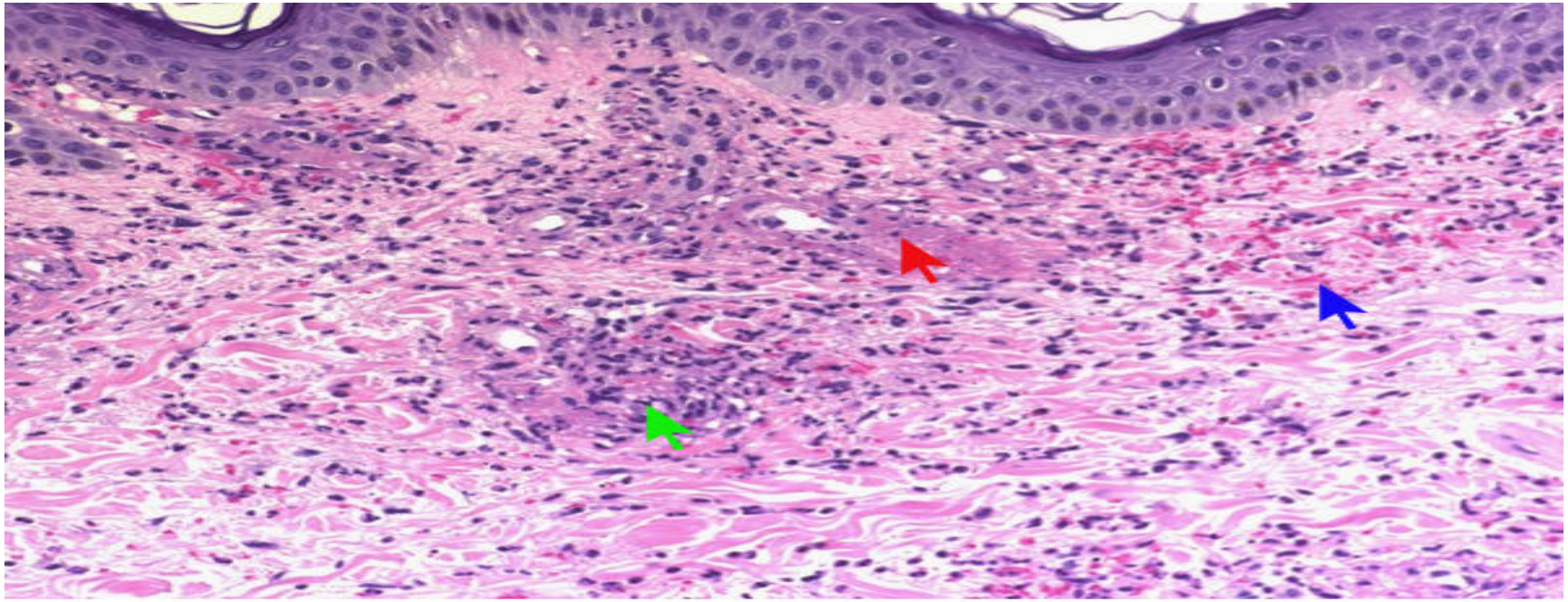
Leukocytoclastic vasculitis - Clinical sign



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

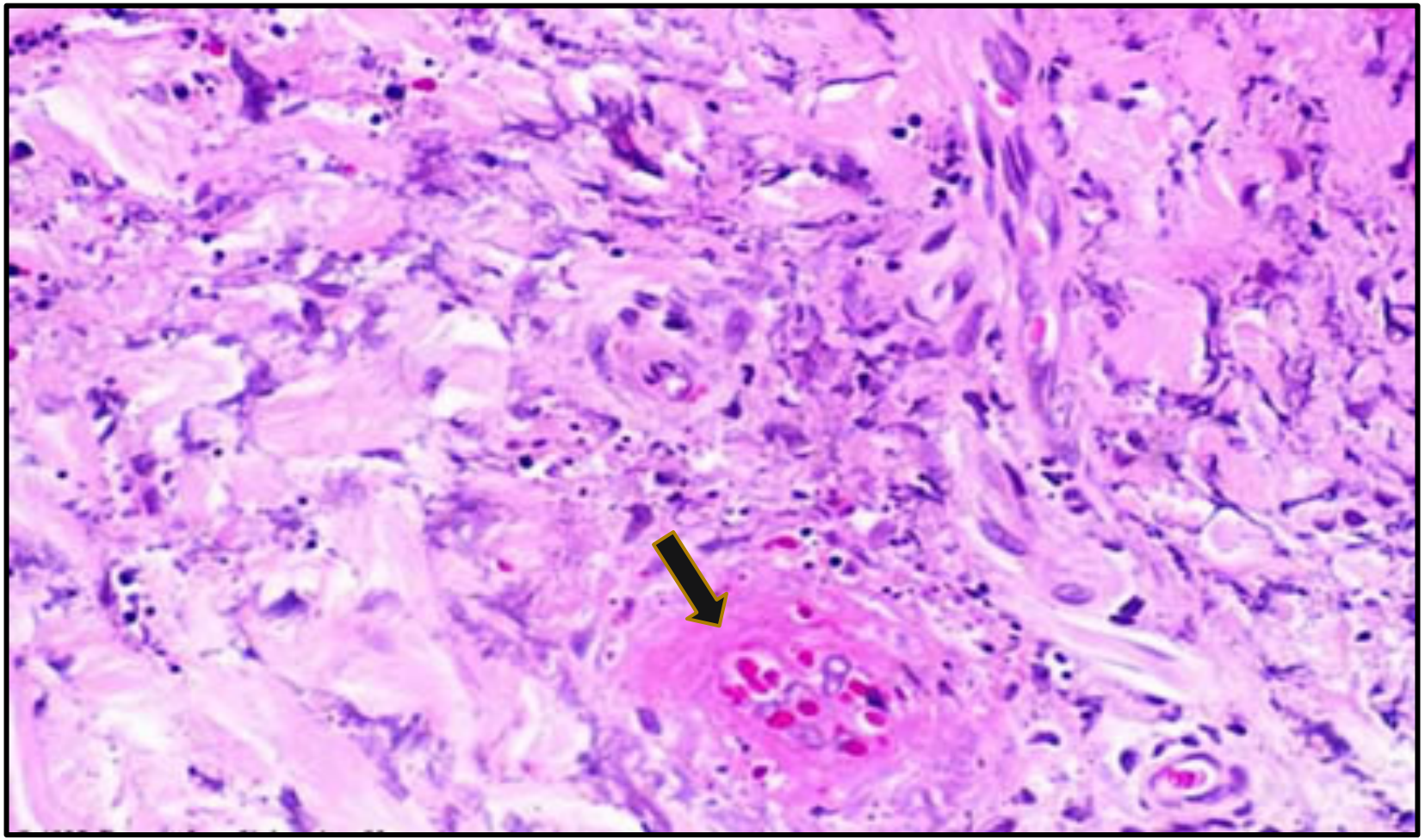
Red cell extravasation

Inflammation

Vasculitis, leukocytoclasis (high power)

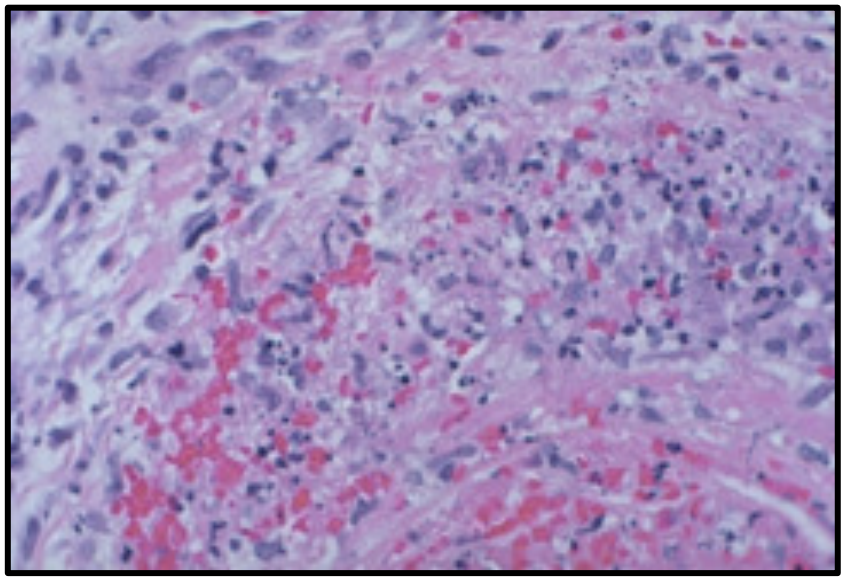
Section of the skin shows fibrinoid necrosis of blood vessels with extravasation of RBCs , neutrophilic infiltration with debris (leukocytoclasis /nuclear dust)

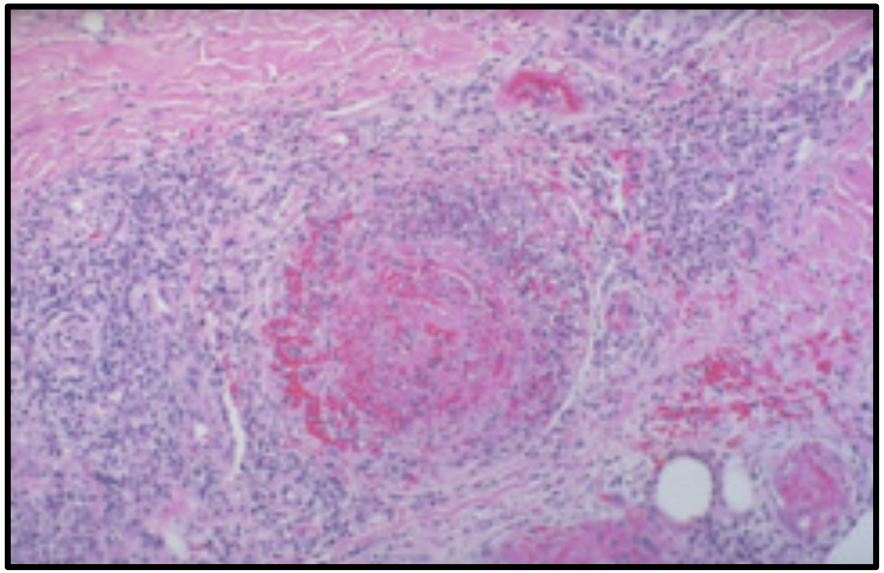
Leukocytoclastic vasculitis - HPF

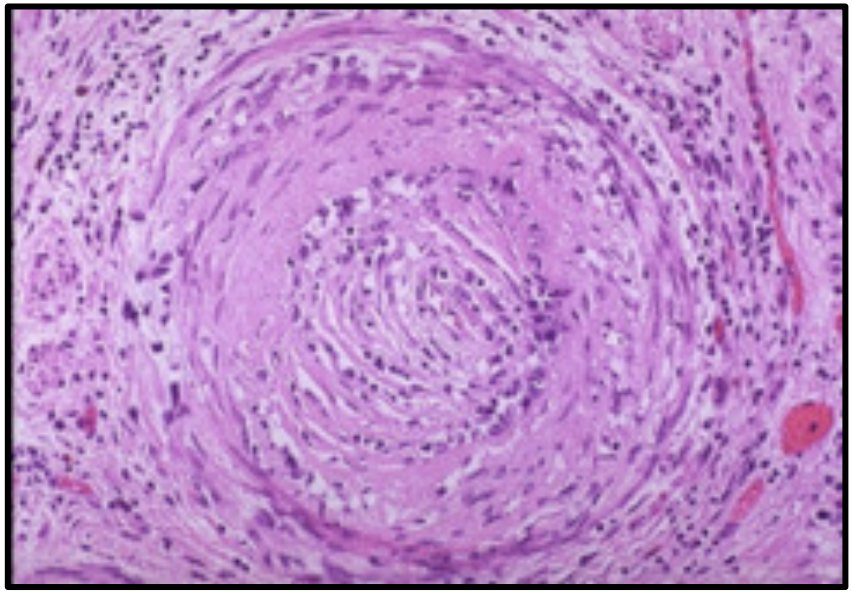


Fibrinoid necrosis of small dermal vessels is present, necessary to establish the diagnosis of leukocytoclastic vasculitis.

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