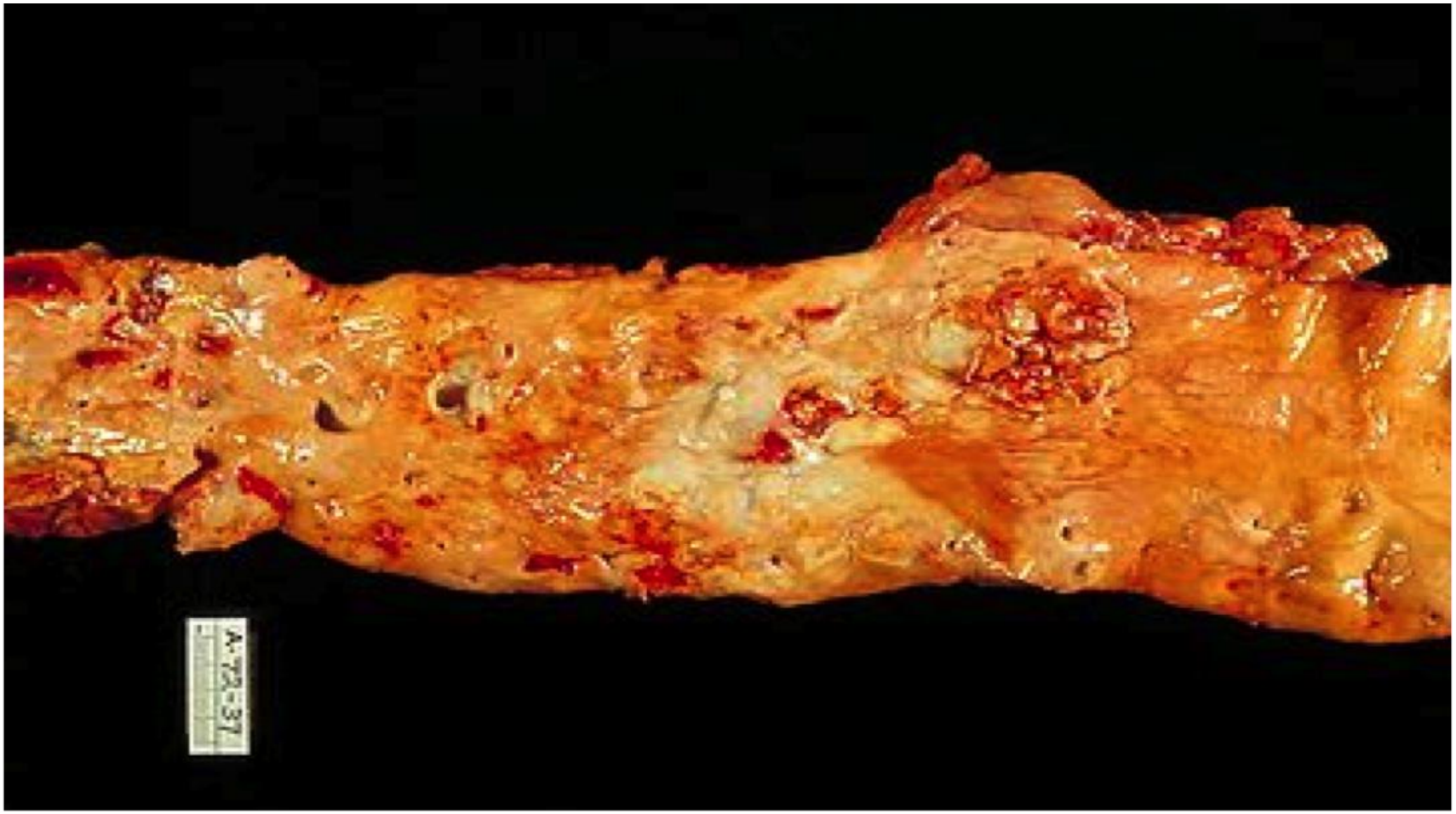
CARDIOVASCULAR SYSTEM

Pathology Practical

ATHEROMA OF THE AORTA

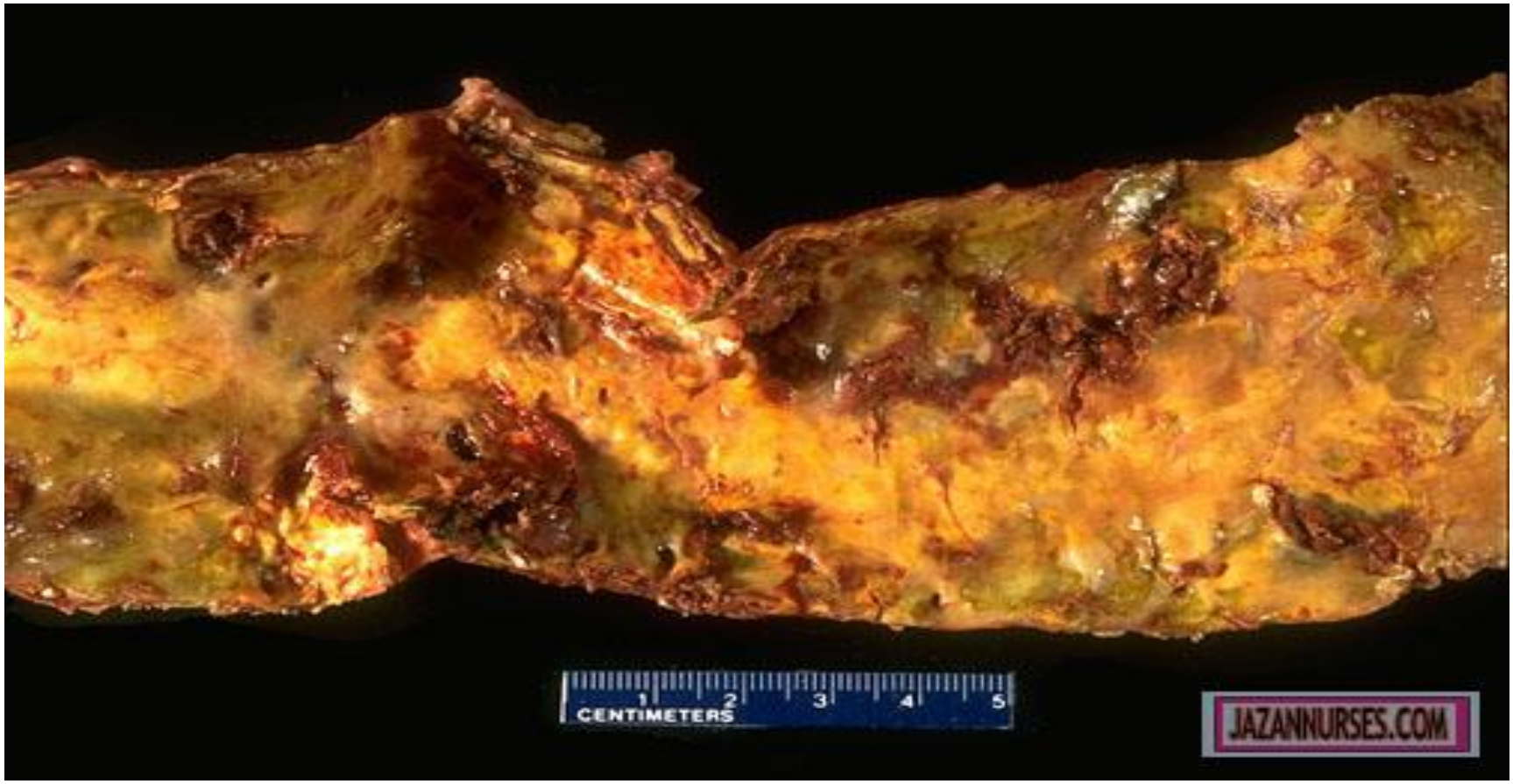
- *An **atheroma** is an accumulation and swelling in artery walls made up of (mostly) macrophage cells, or debris, and containing lipids (cholesterol and fatty acids), calcium and a variable amount of fibrous connective tissue.*
- *The four major risk factors are hyperlipidemia, hypertension, cigarette smoking and diabetes .*

Atheroma of the Aorta - Gross



Atherosclerosis aorta gross pathology shows extensive ulceration in the plaques

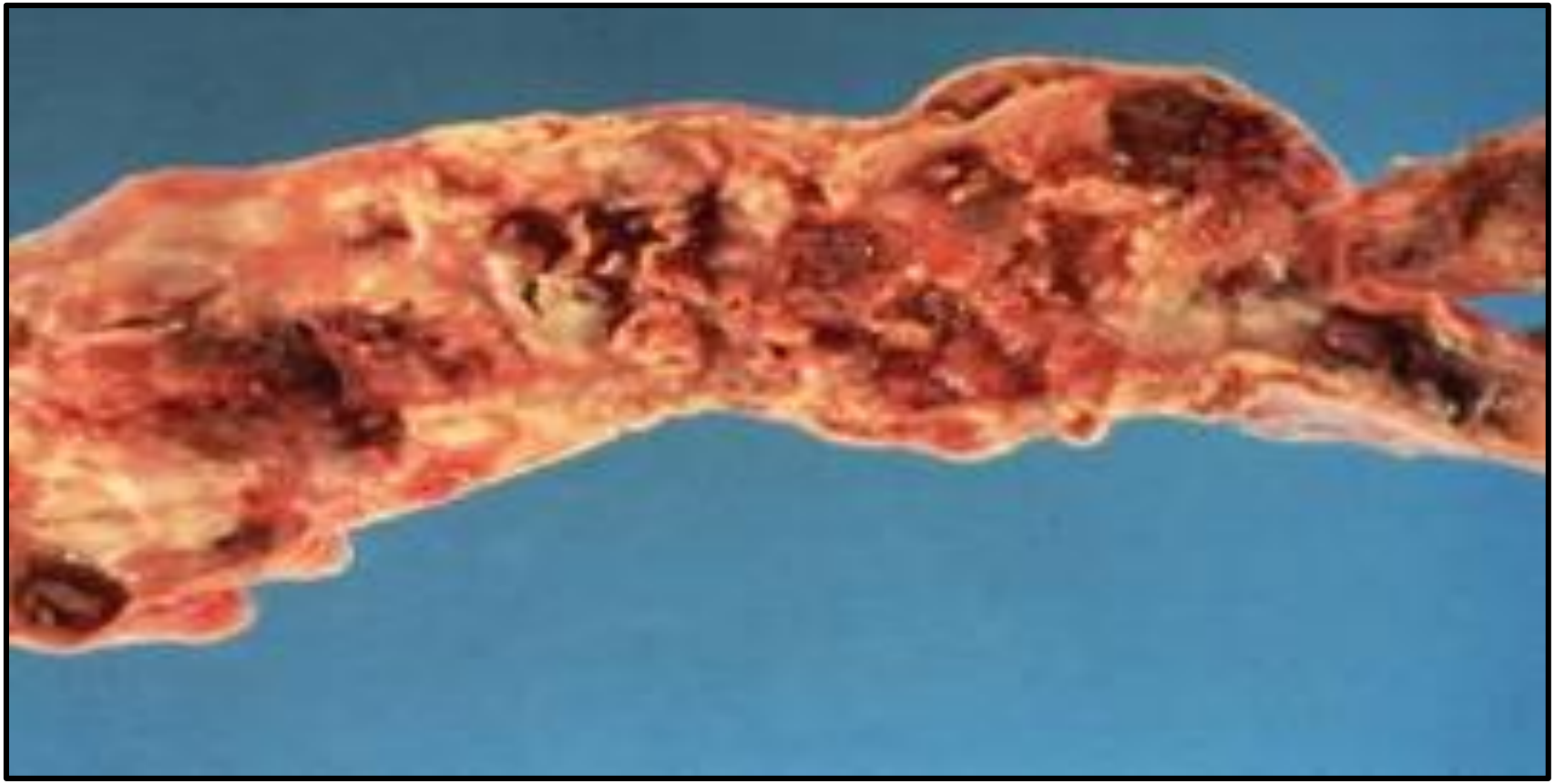
Atheroma of the Aorta - Gross



Severe atherosclerosis of the aorta : *the atheromatous plaques have undergone ulceration along with formation of overlying mural thrombus.*

Complications: *are thrombosis , hemorrhage , calcifications and aneurysmal dilatation with the distal ischemic events .*

Atheroma of the Aorta - Gross

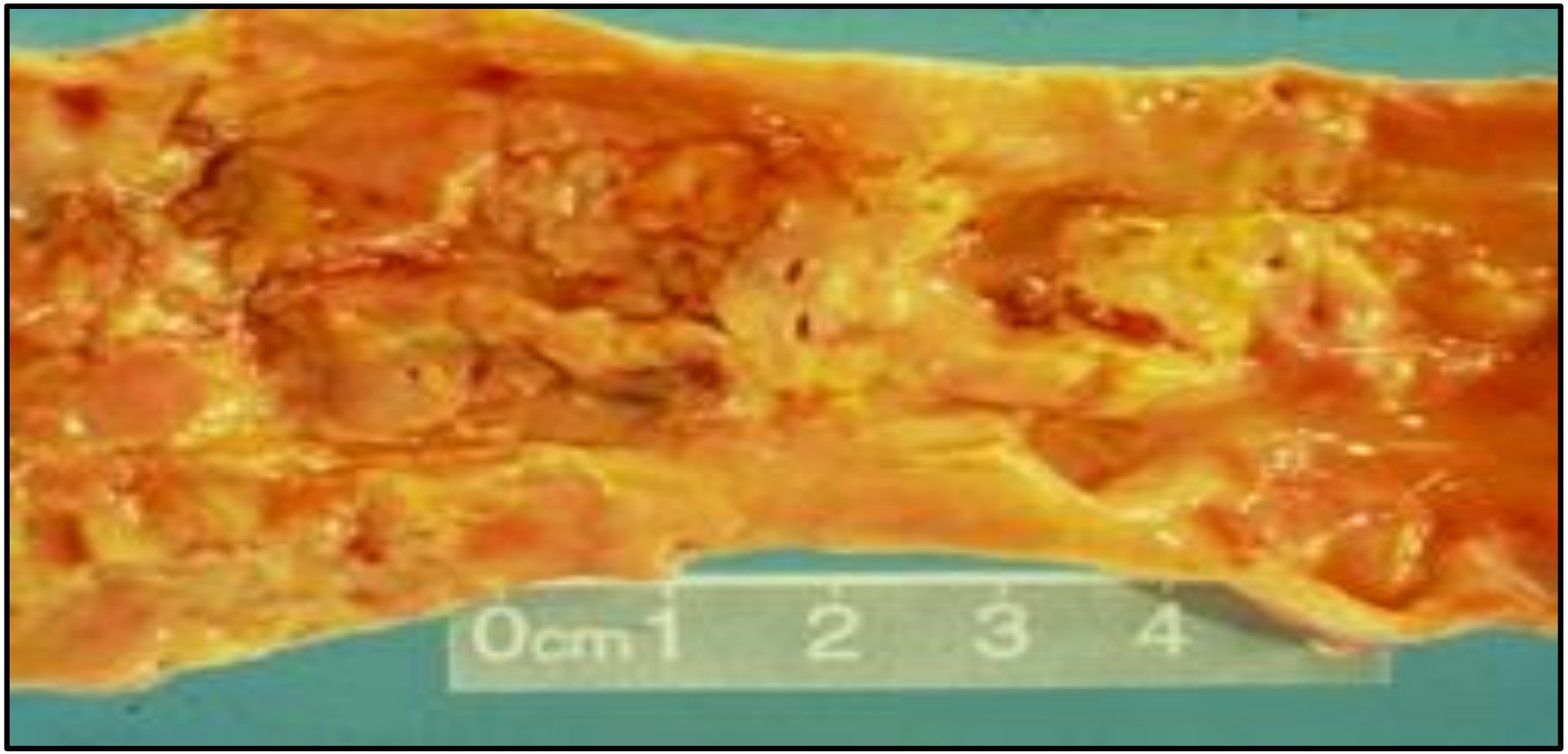


Aorta: complicated atheromatous plaques

Note the fissured-appearing endothelial surface and raised plaque-like structures from the surface.

Red clot material is adherent to the plaques in multiple areas. These clots consist of platelets held together by fibrin strands.

Atheroma of the Aorta - Gross



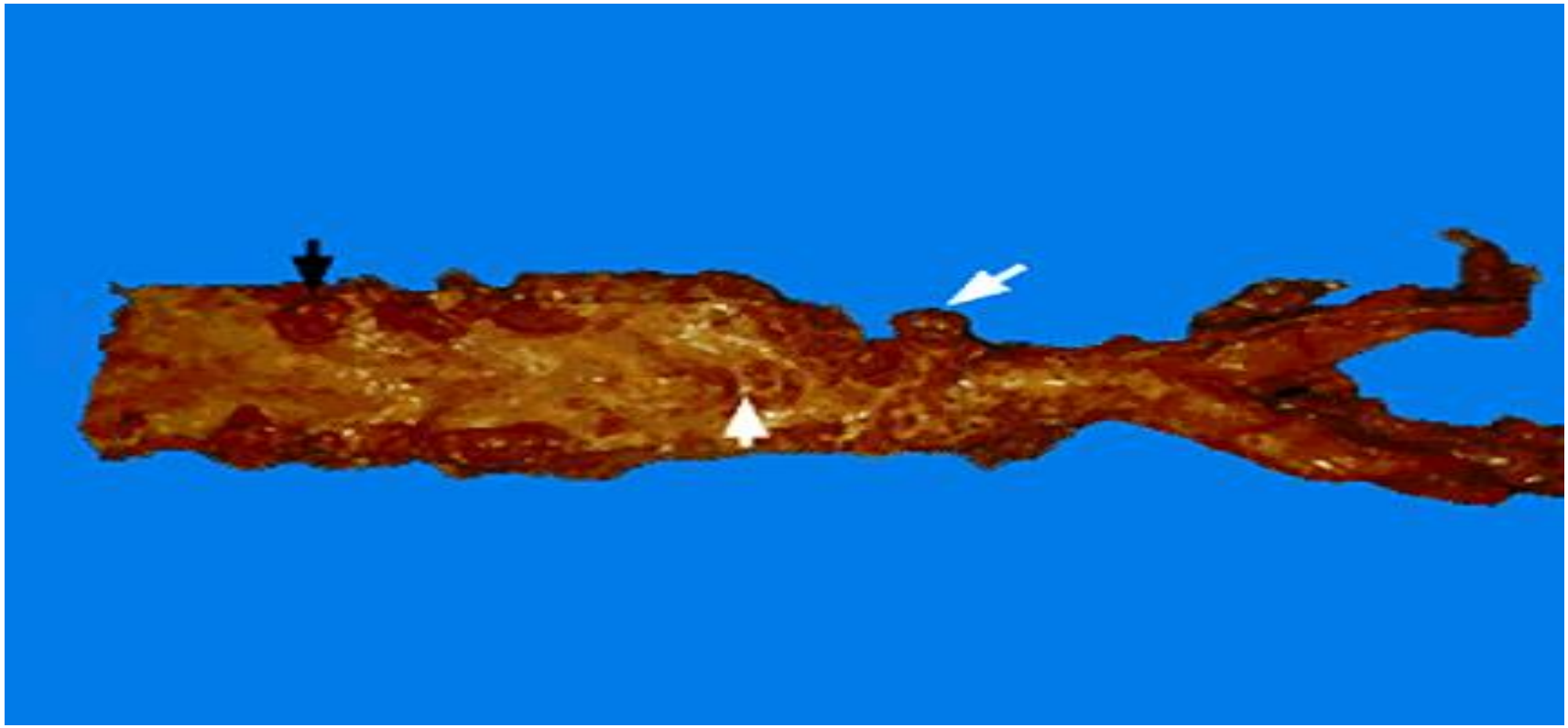
Aorta: complicated atheromatous plaques

Note the raised yellow plaques and the fissures in between the plaques. Dystrophic calcification is likely present as well

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 - Gross

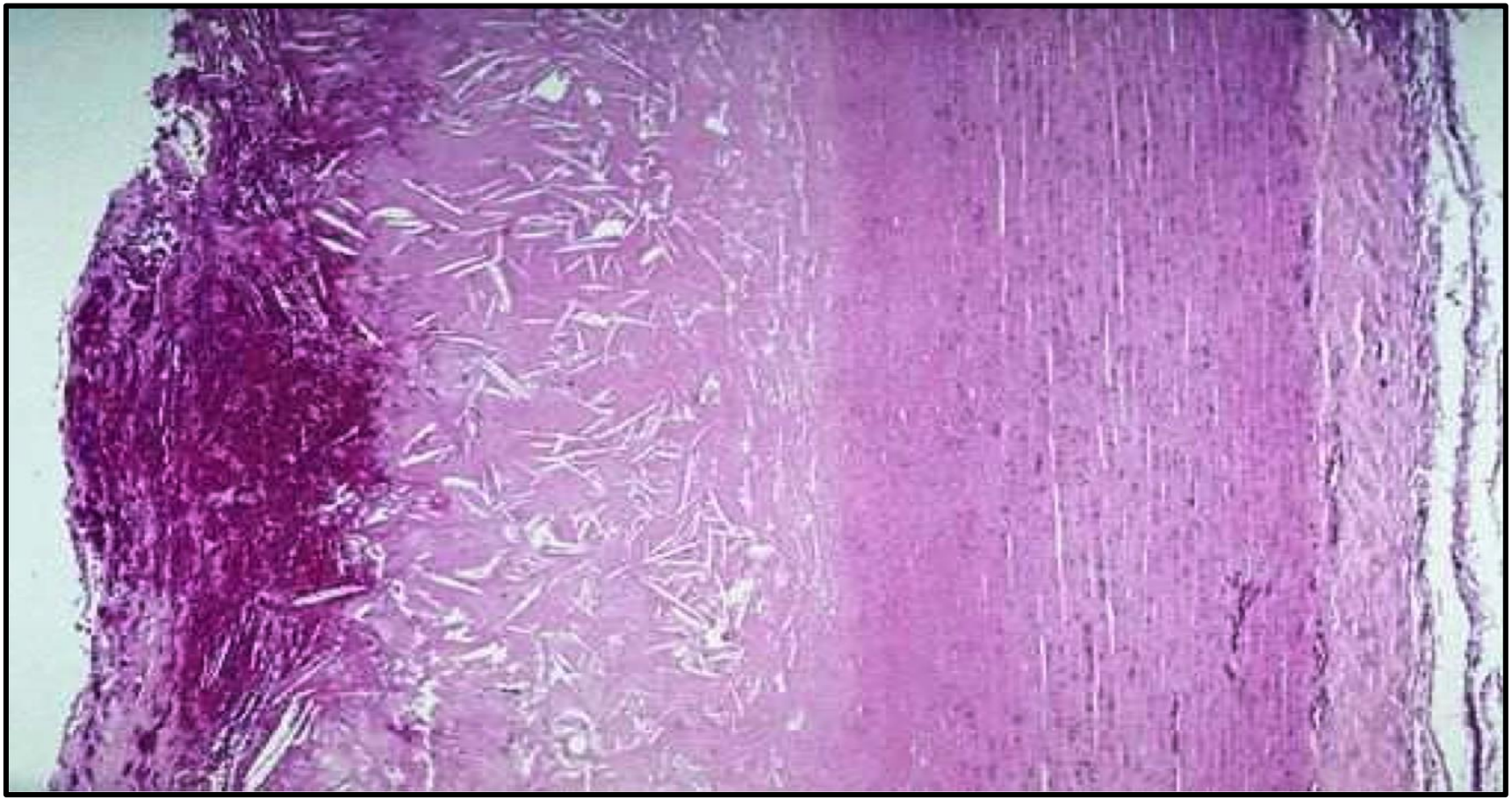


- *Inner surface of aorta and bifurcation,*

opened lengthwise along the posterior midline.

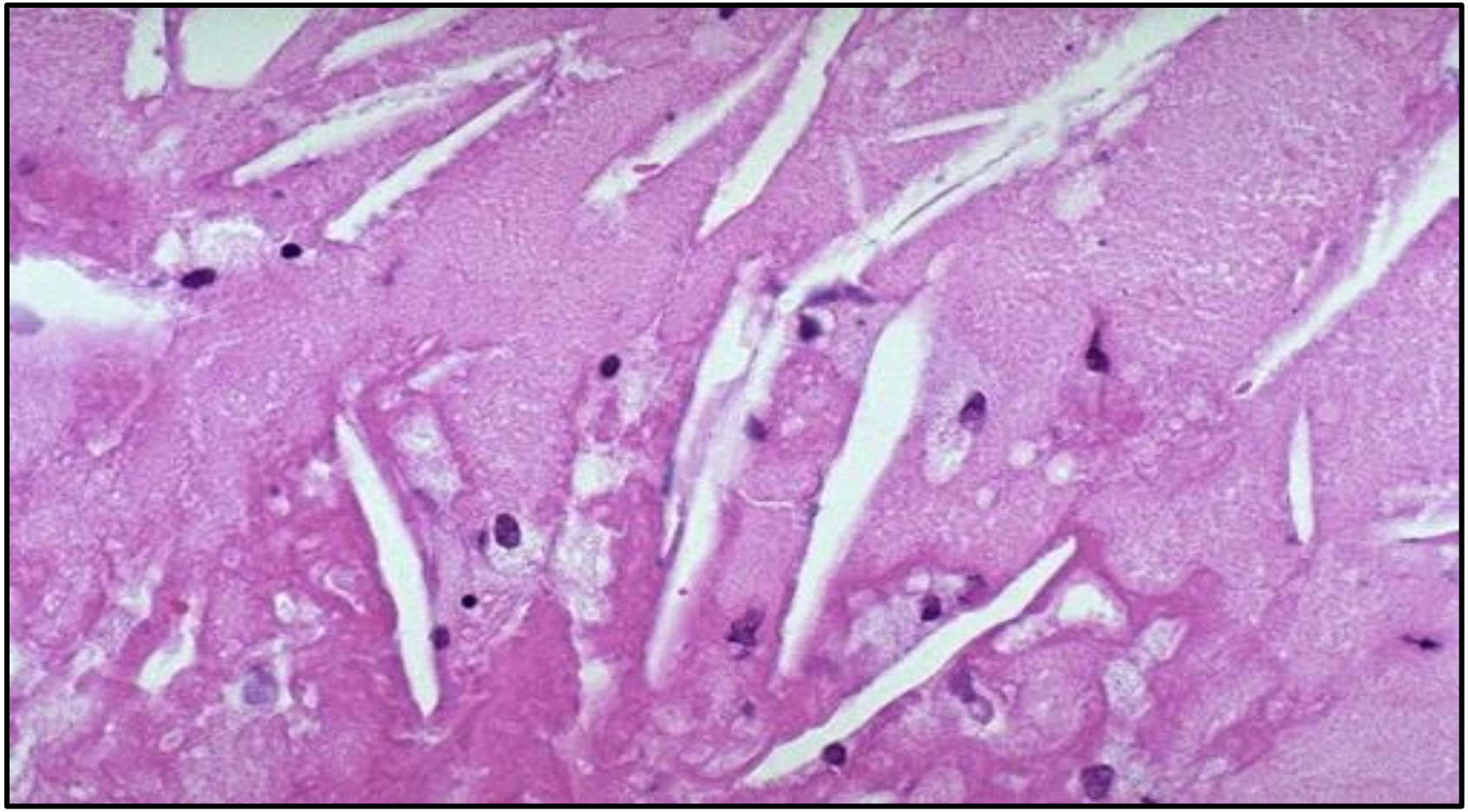
- *Note: irregular variegated lining due to diffuse disease, with red thrombi (black arrow); ostia of celiac and superior mesenteric arteries and right renal artery (white arrows); deceptive narrower caliber of abdominal aorta below celiac artery due to rigidity of calcified atheroma*

Atheroma of the Aorta - LPF



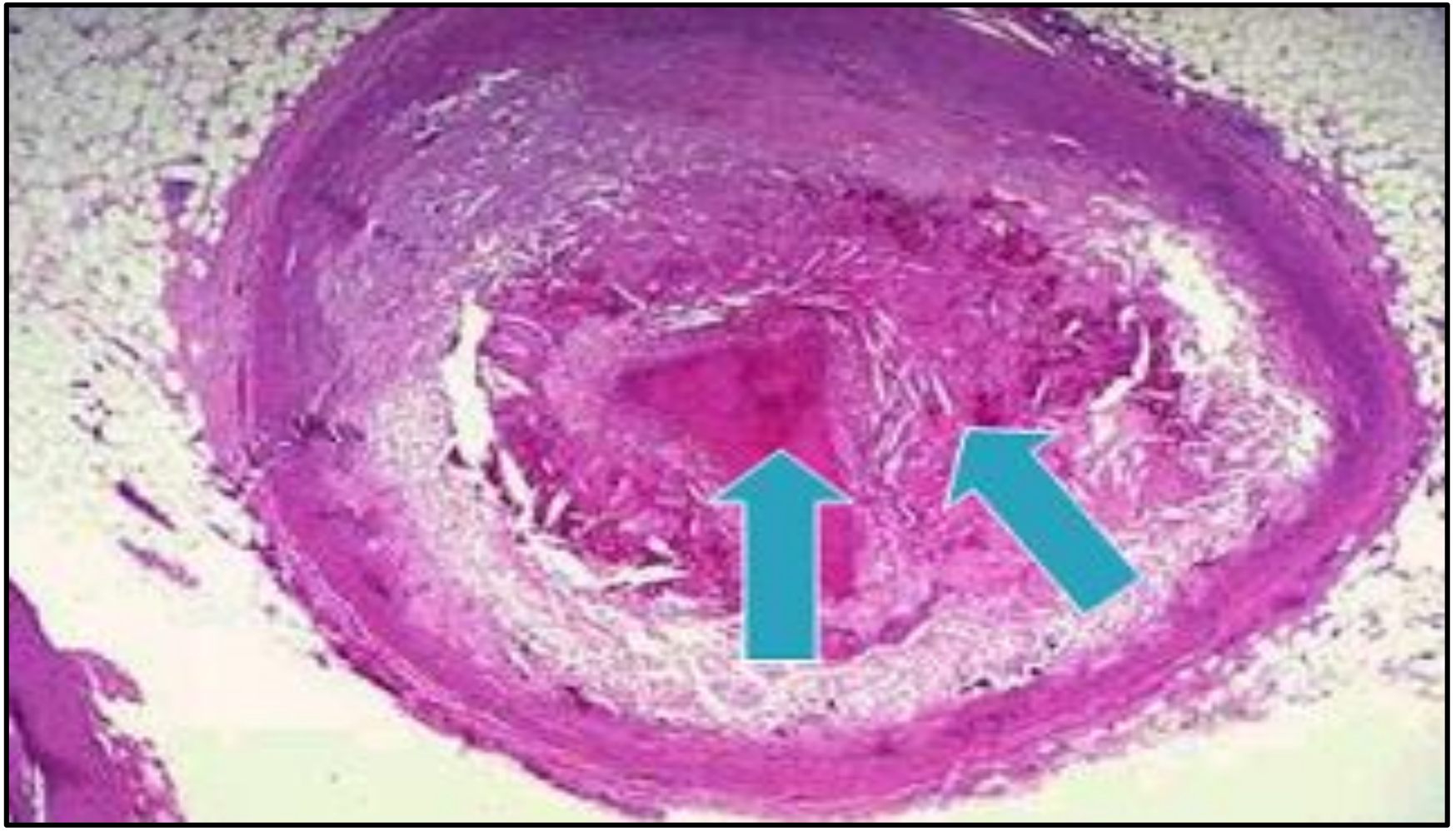
This microscopic cross section of the aorta shows a large overlying atheroma on the left. Cholesterol clefts are numerous in this atheroma. The surface on the far left shows ulceration and hemorrhage. Despite this ulceration, atheromatous emboli are rare

Atheroma of the Aorta - LPF



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

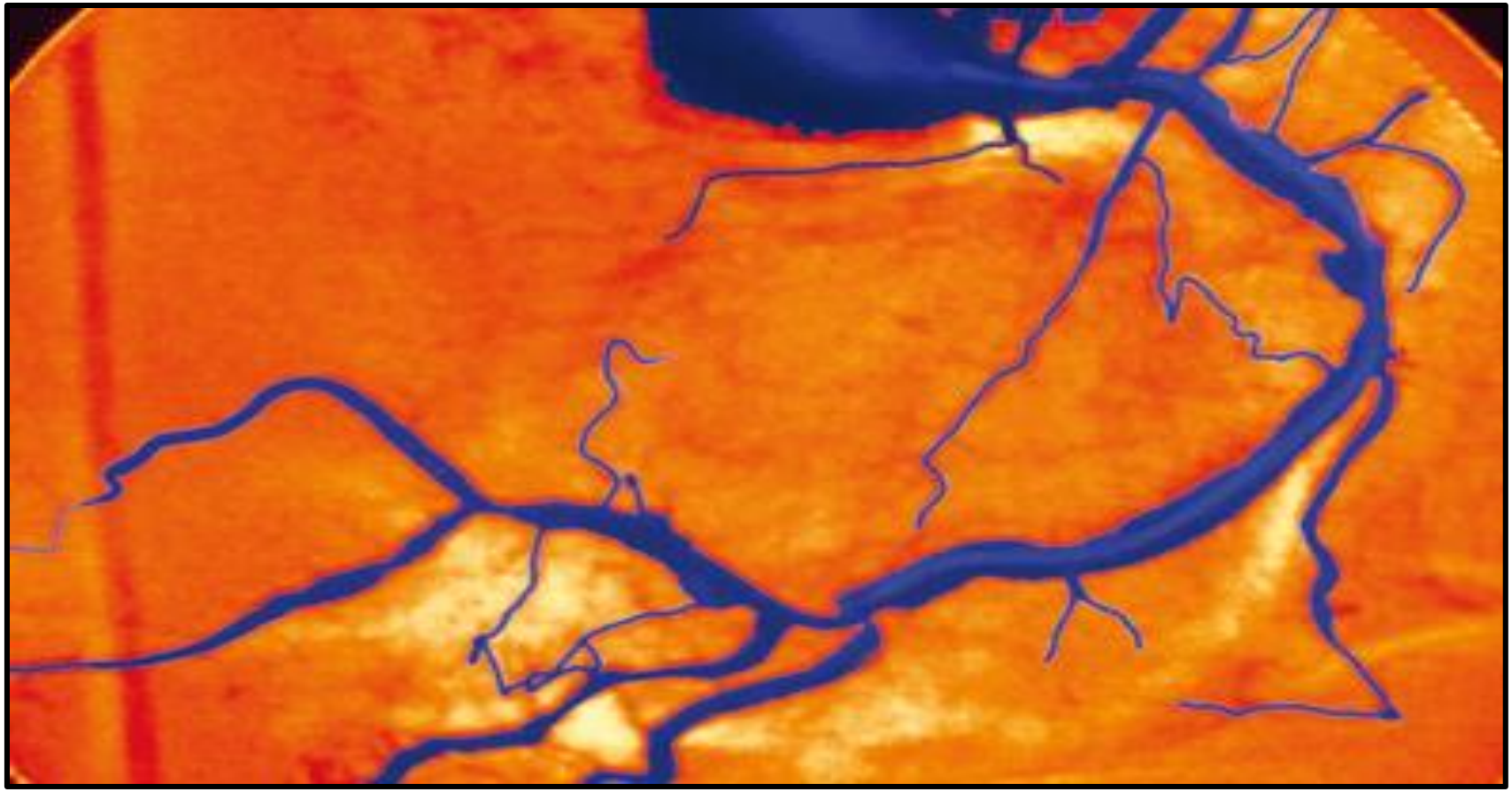
Atheroma of the Aorta - MPF



Aortic Atheroma with Thrombosis

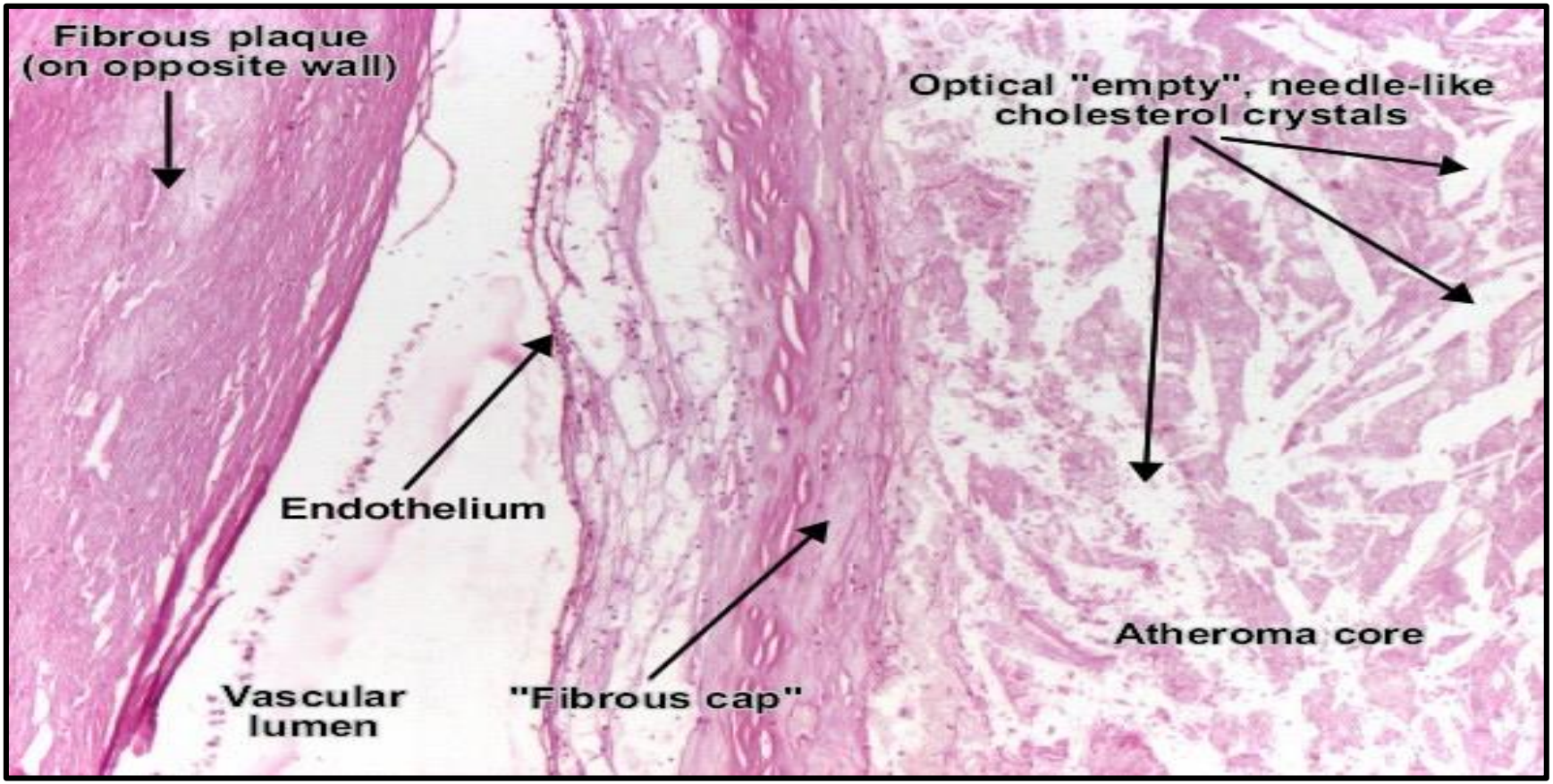
CORONARY ATHEROSCLEROSIS

Coronary Atherosclerosis - Gross



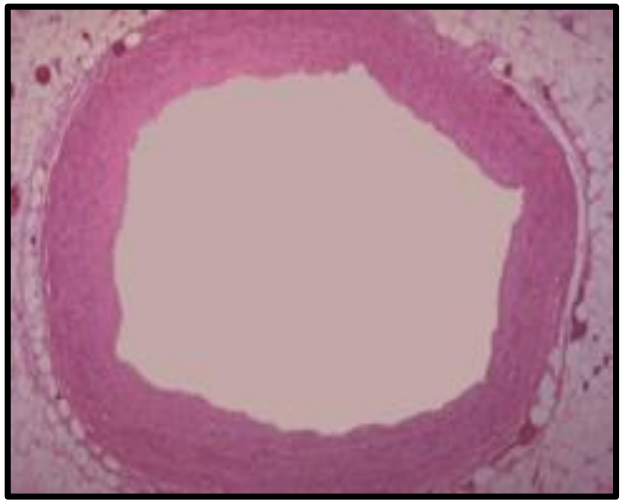
***Coronary atherosclerosis**. Coloured angiogram (X- ray) showing atherosclerosis in a coronary artery. The atherosclerosis is seen as the pinching in the blue- coloured artery at bottom centre*

Coronary atherosclerosis - LPF

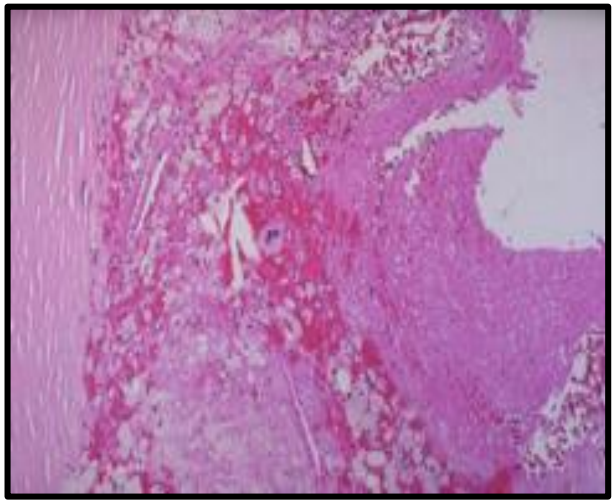


Coronary artery with atherosclerosis (fibro-lipid or fibro-fatty plaque). The atheromatous fibro-fatty plaque is characterized by the accumulation of lipids in the intima of the arteries, narrowing the lumen. Beneath the endothelium it has a "fibrous cap" covering the atheromatous "core" of the plaque, which consists in cholesterol crystals, cholesterol esters, fibrin, macrophages and smooth muscle cells, proteoglycans, collagen, elastin and cellular debris.

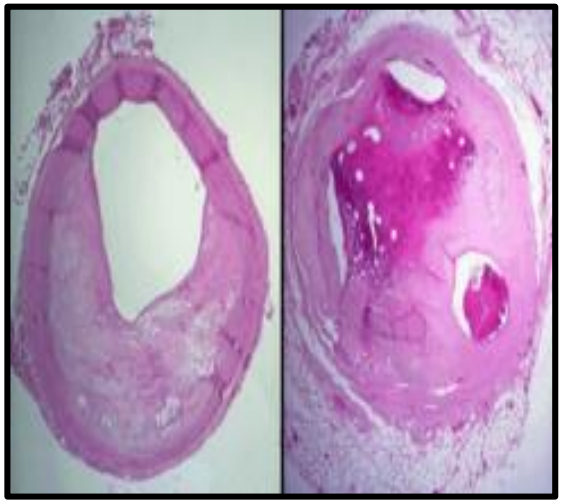
Coronary atherosclerosis - LPF



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

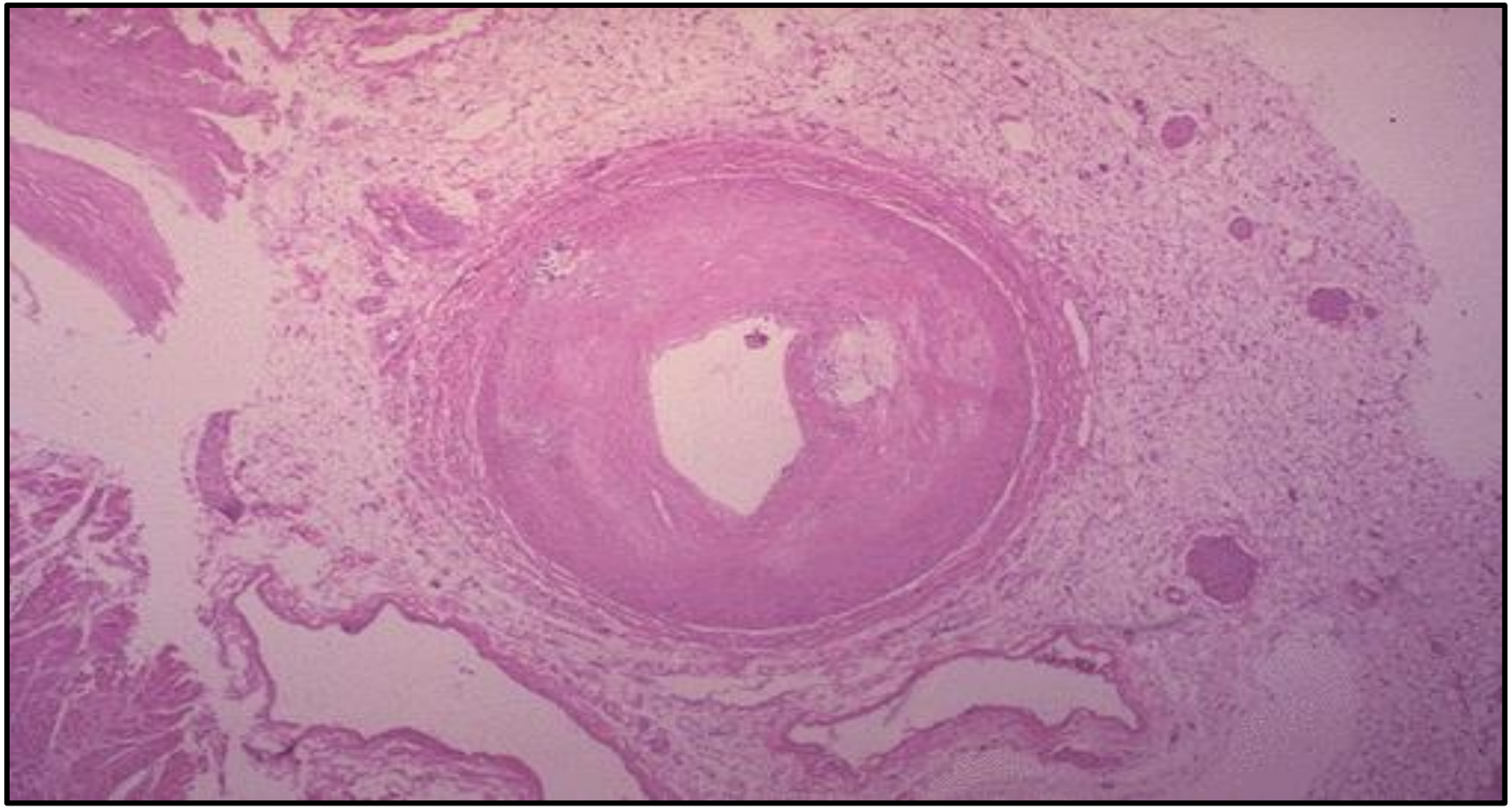


Atheromatous plaque in a coronary artery that shows endothelial denudation with disruption and overlying thrombus formation at the right. The arterial media is at the left



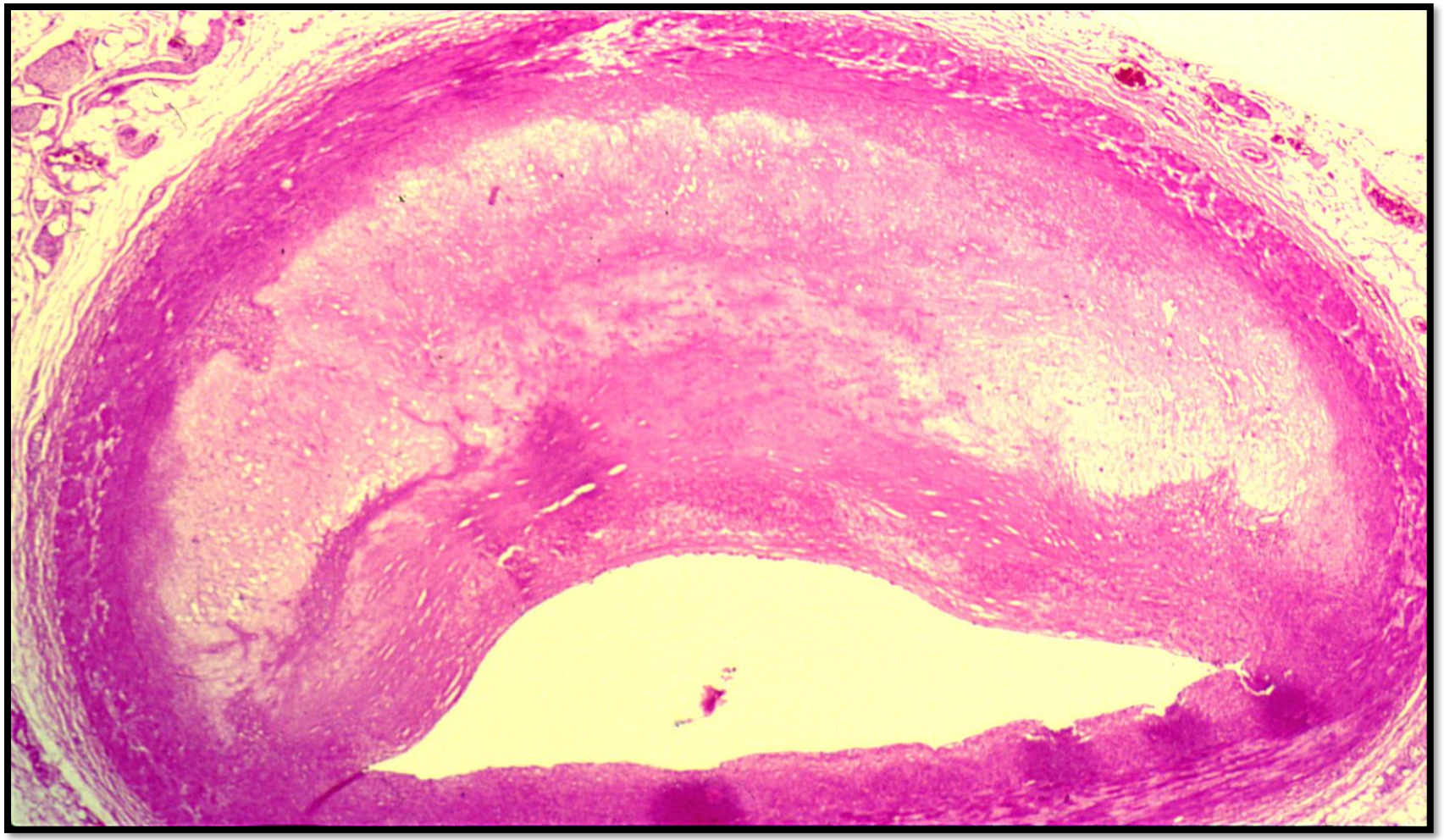
Occlusive coronary atherosclerosis. The coronary at the left is narrowed by 60 to 70%. The coronary at the right is even worse with evidence for previous thrombosis with organization of the thrombus

Coronary atherosclerosis - MPF



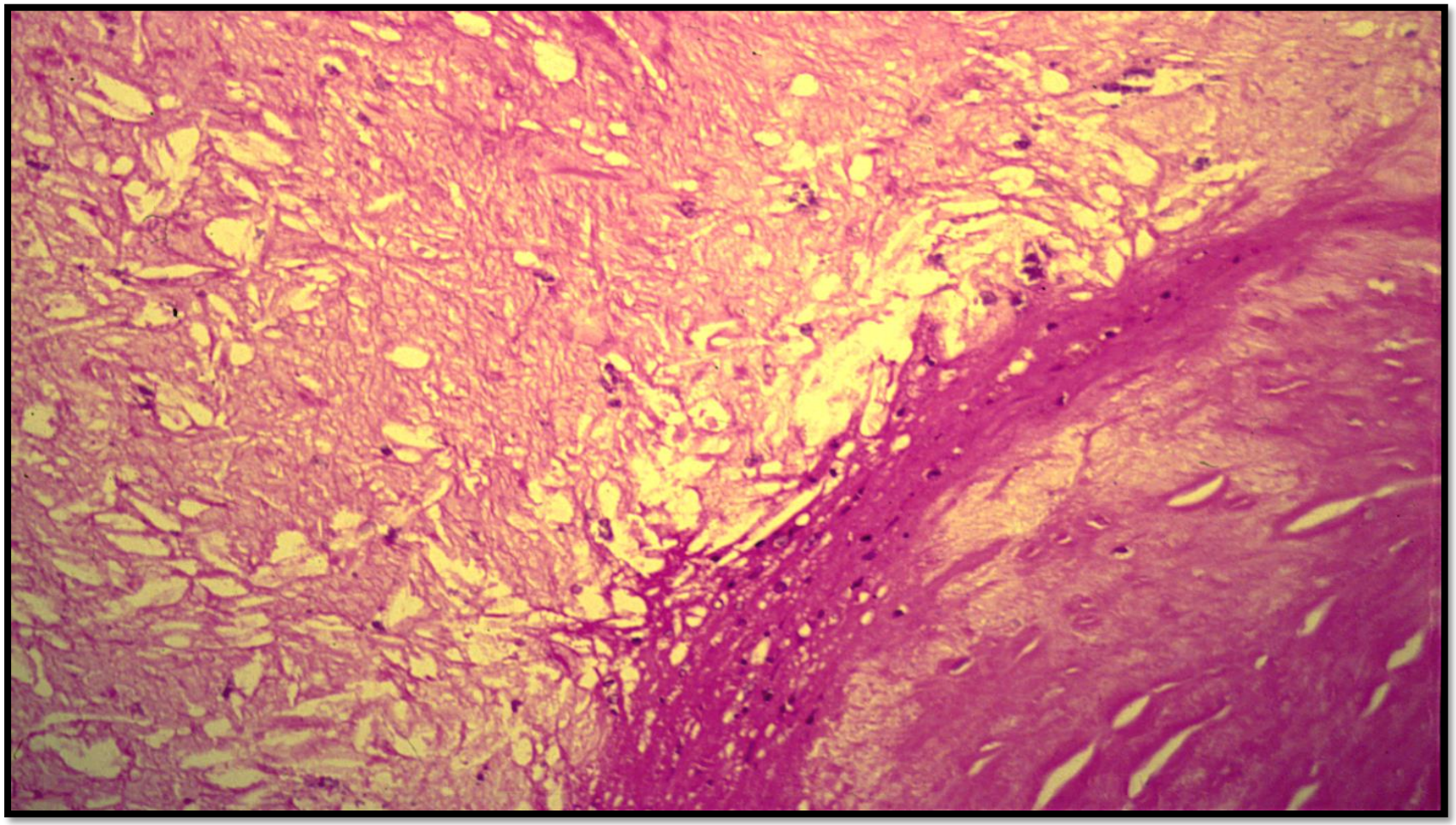
This distal portion of coronary artery shows significant narrowing. Such distal involvement is typical of severe coronary atherosclerosis, such as can appear with diabetes mellitus or familial hypercholesterolemia.

Coronary atherosclerosis - MPF



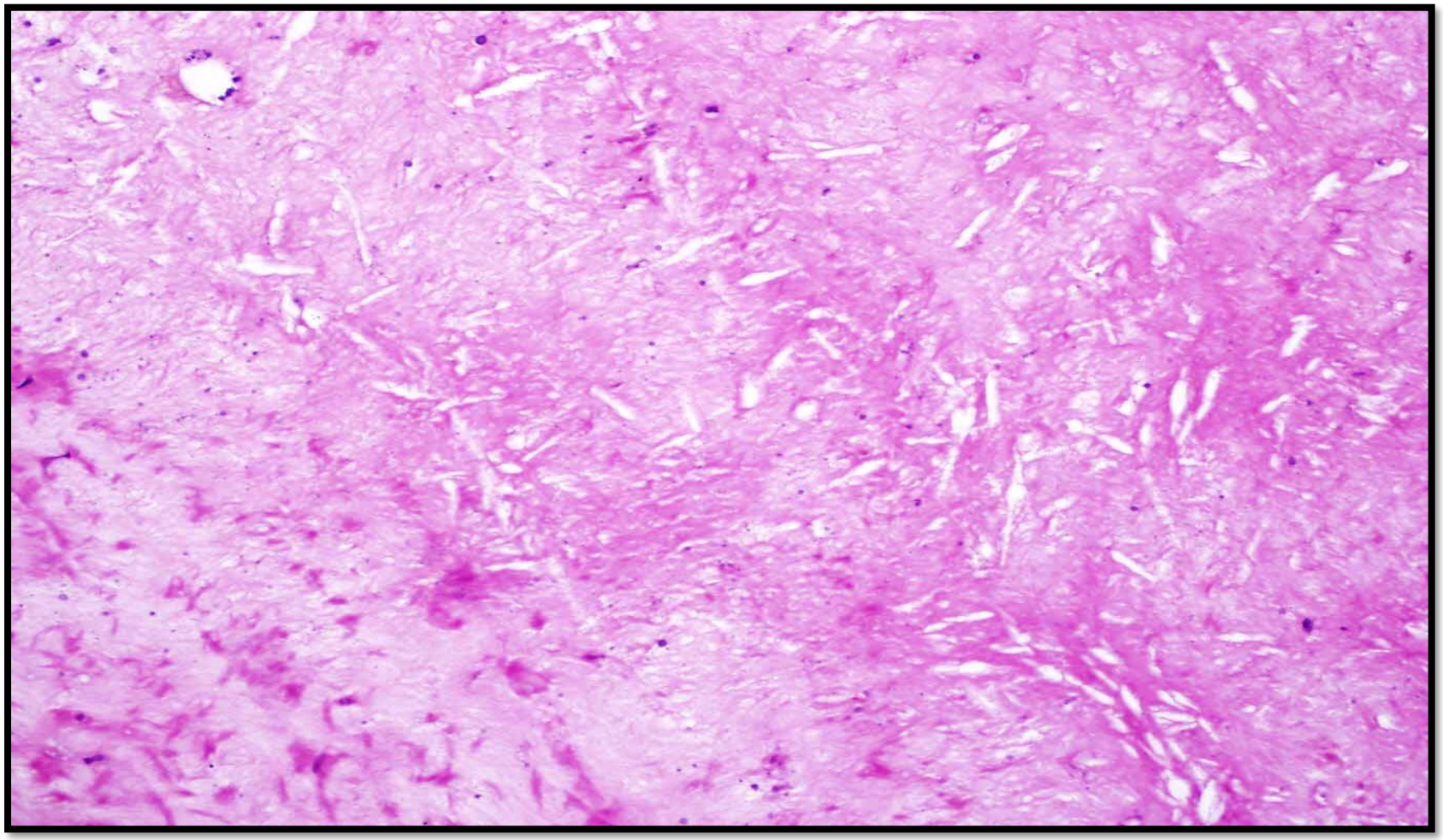
Severe coronary atherosclerosis with narrowing of the lumen

Coronary atherosclerosis - HPF



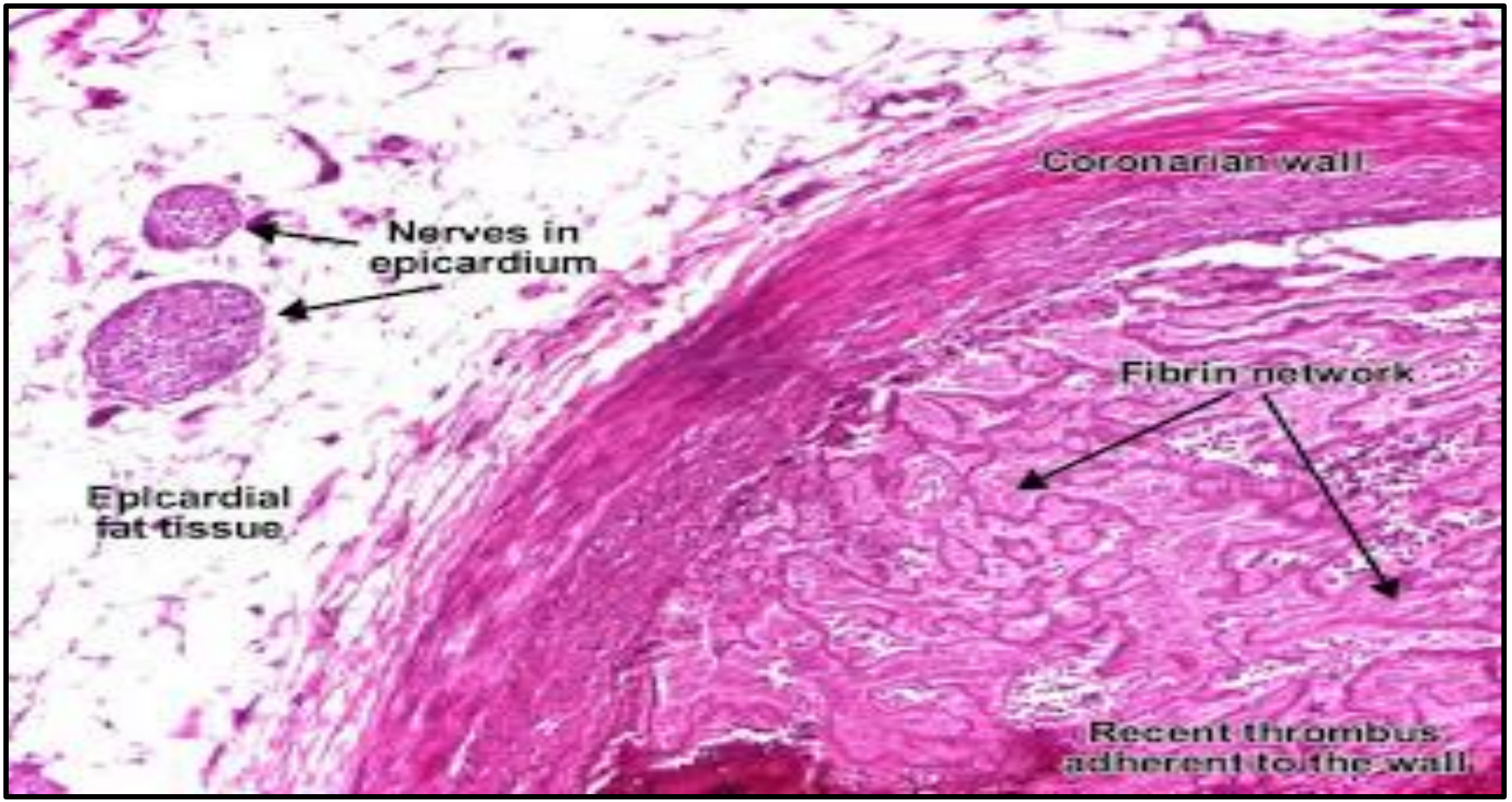
Partial occlusion of the lumen by an atheromatous plaque. The plaque consists of dissolved, cholesterol clefts, hyaline fibrous tissue and some blood capillaries.

Coronary atherosclerosis - HPF



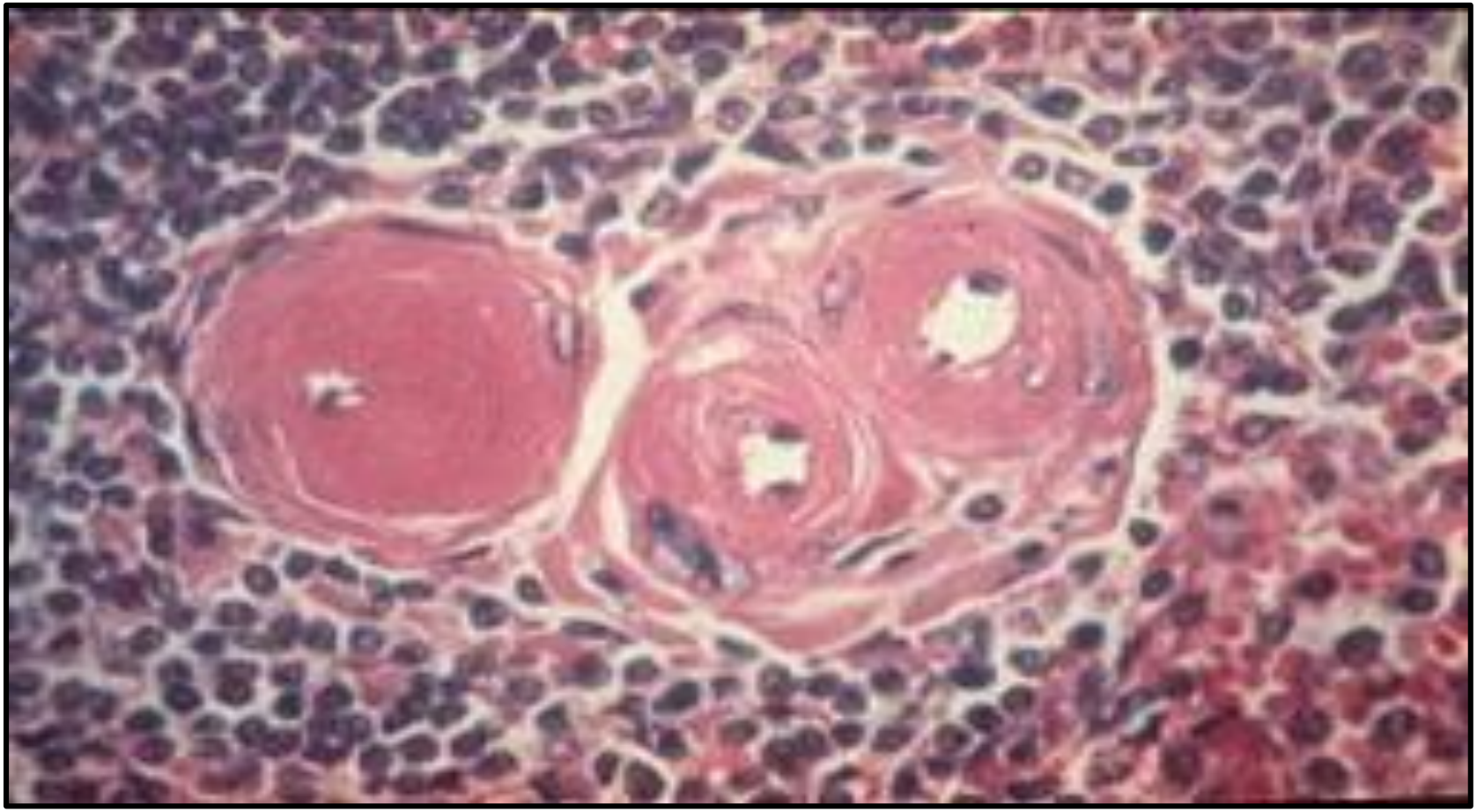
The internal elastic lamina is thin and fragmented.
Pressure atrophy of the media opposition atheromatous plaque consists of cholesterol clefts, hyaline fibrous tissue and some blood capillaries.

Recent thrombus in a Coronary artery



***Recent thrombus in a coronary artery**: The arterial lumen is completely obstructed by a recent thrombus - fibrin network (pink) containing red blood cells and platelets. The thrombus is developed on an ulcerated atherosclerotic (fibrous) plaque and is adherent to the arterial wall.*

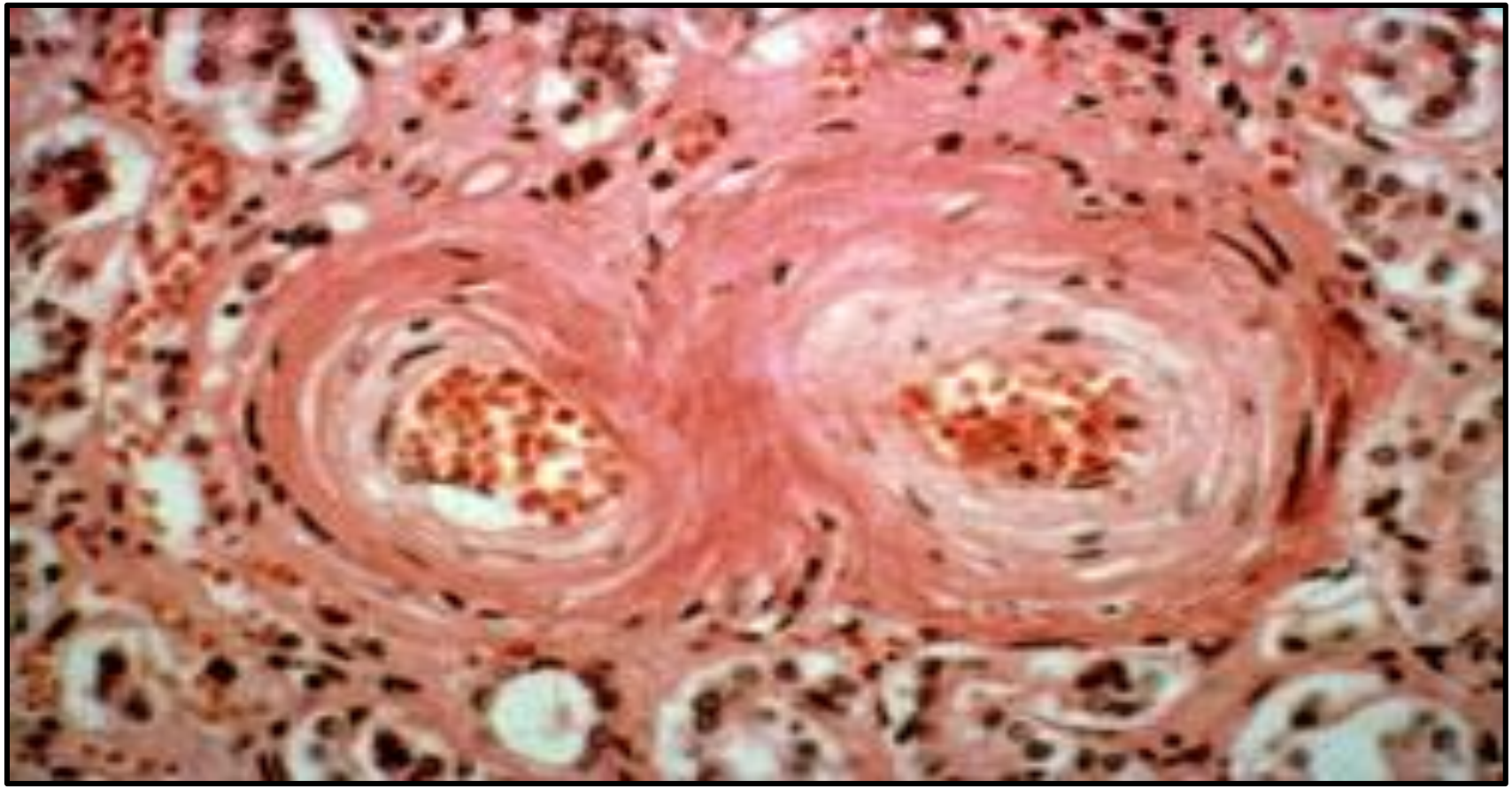
Hyaline arteriolosclerosis - HPF



Hyaline arteriolosclerosis

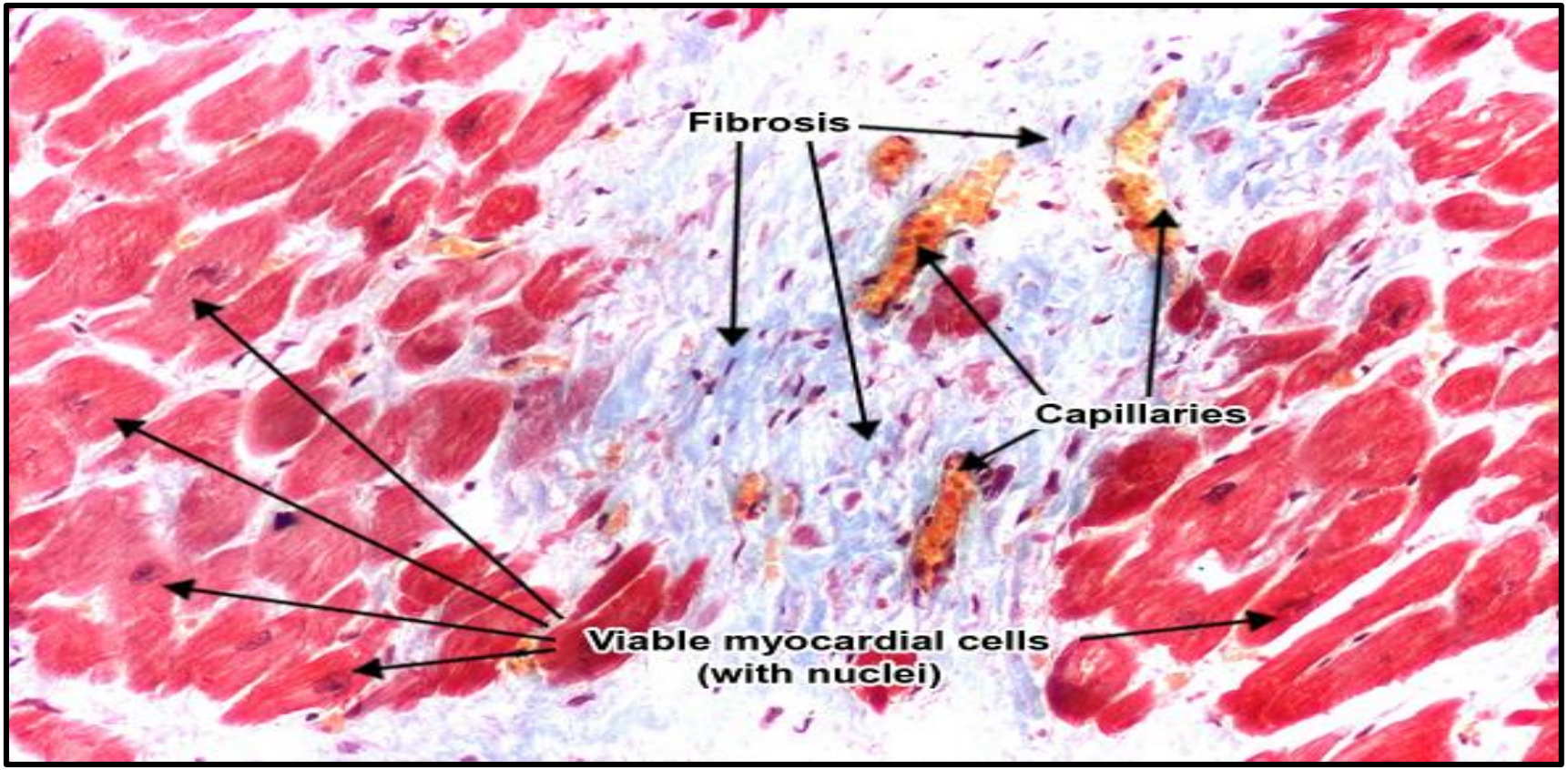
Arteriosclerosis (hardening of the arteries) involves both small and large vessels. It is commonly found in diabetics and hypertensives.

Hyperplastic arteriolosclerosis - HPF



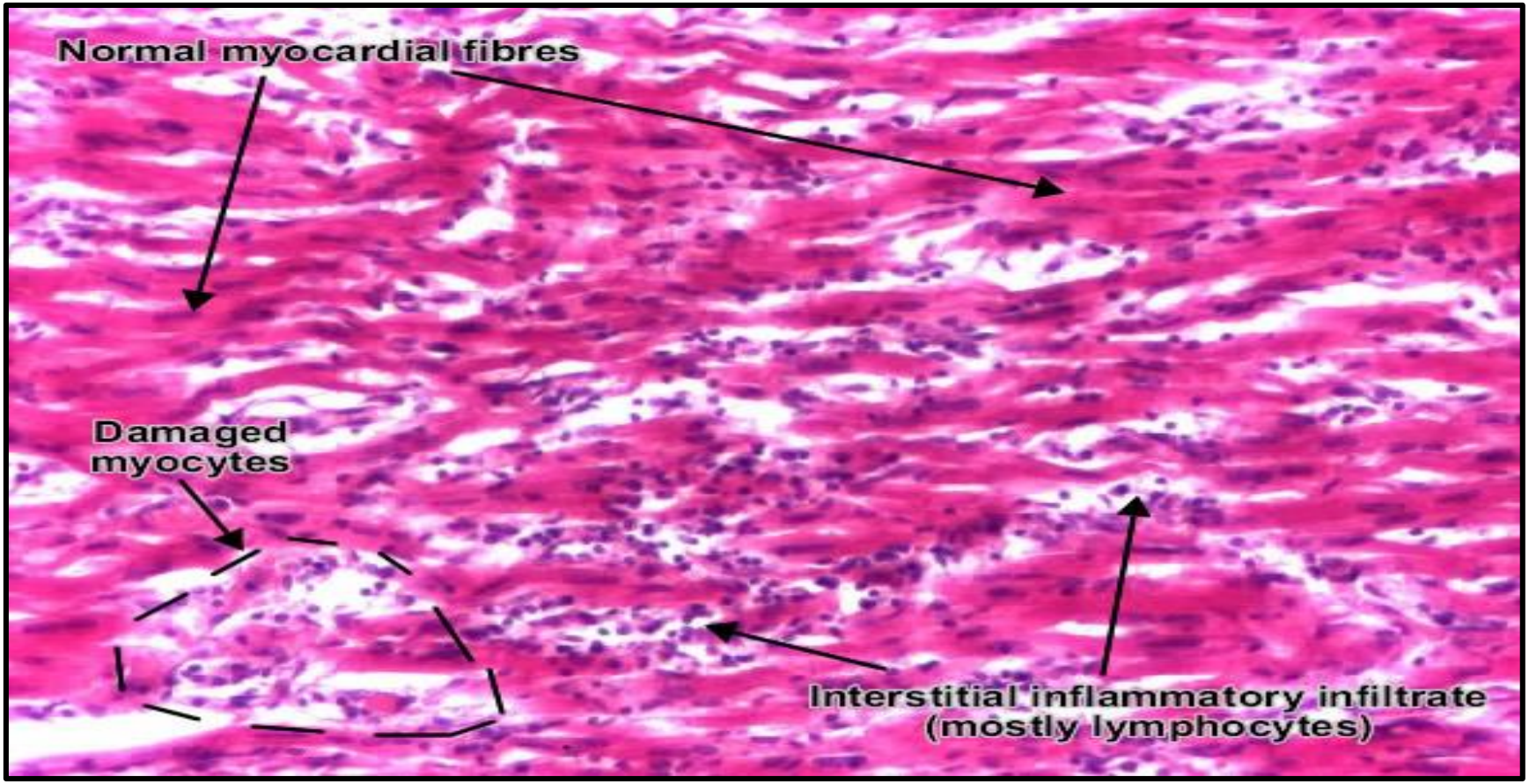
***Hyperplastic arteriolosclerosis**: This is the other type of small vessel arteriosclerosis. It is predominantly seen in malignant hypertension and renal disease associated with polyarteritis nodosa and progressive systemic sclerosis.*

Ischemic fibrosis of myocardium (diffuse ventricular myocardial fibrosis)



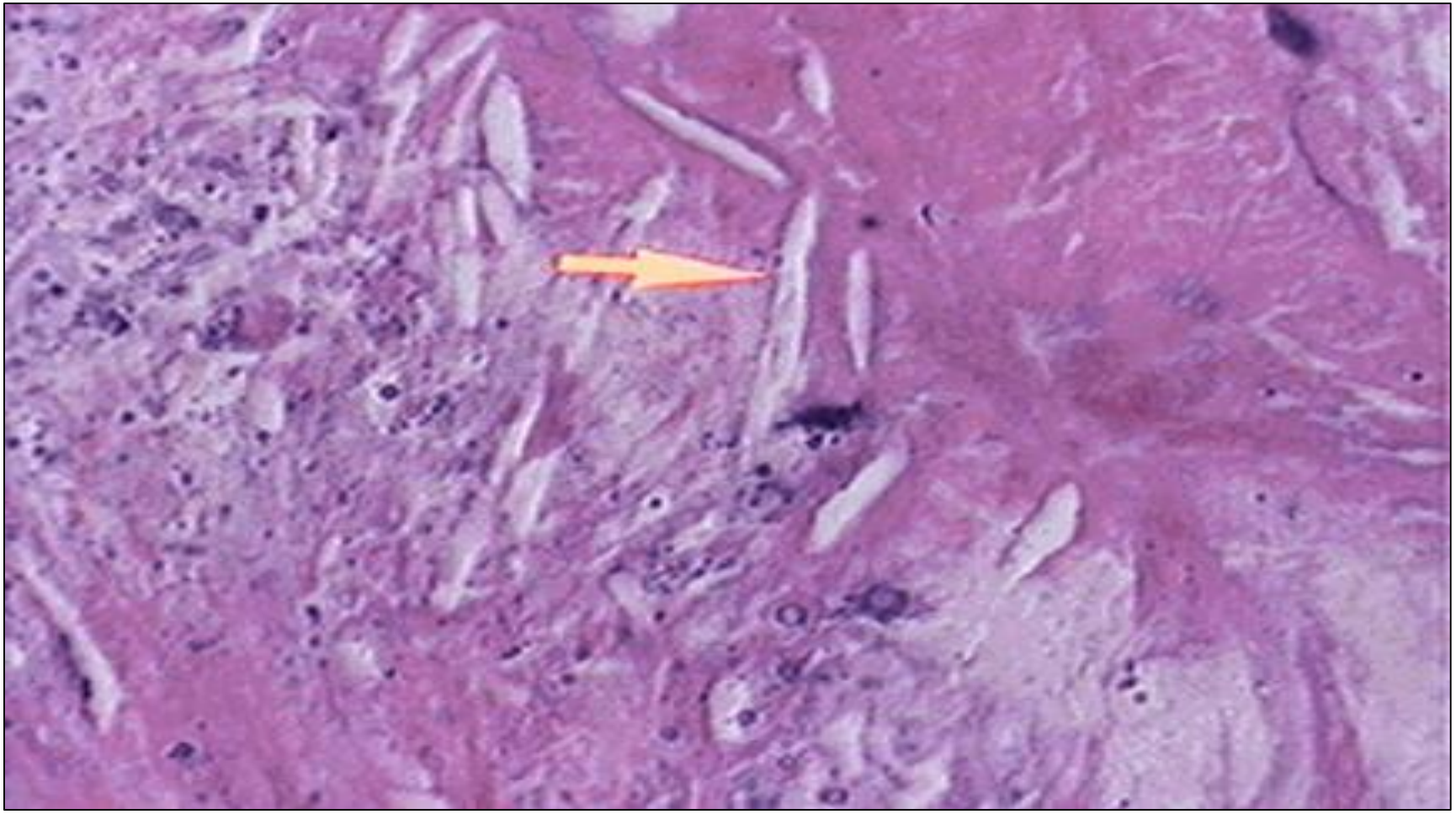
Diffuse myocardial fibrosis *(Ischemic fibrosis of the myocardium) Myocardial cells (red) intermingled with collagen-rich fibrosis (blue) which completely replaced the necrotic myocardial cells. Capillaries (with yellow-orange red blood cells) within fibrosis remained from repair by connective tissue process.*

Acute viral myocarditis - MPF



***Myocarditis** is an inflammation of the myocardium. Acute viral myocarditis is produced most often by Coxsackie B virus and echoviruses. Myocardial interstitium presents an abundant edema and inflammatory infiltrate, mainly with lymphocytes and macrophages.*

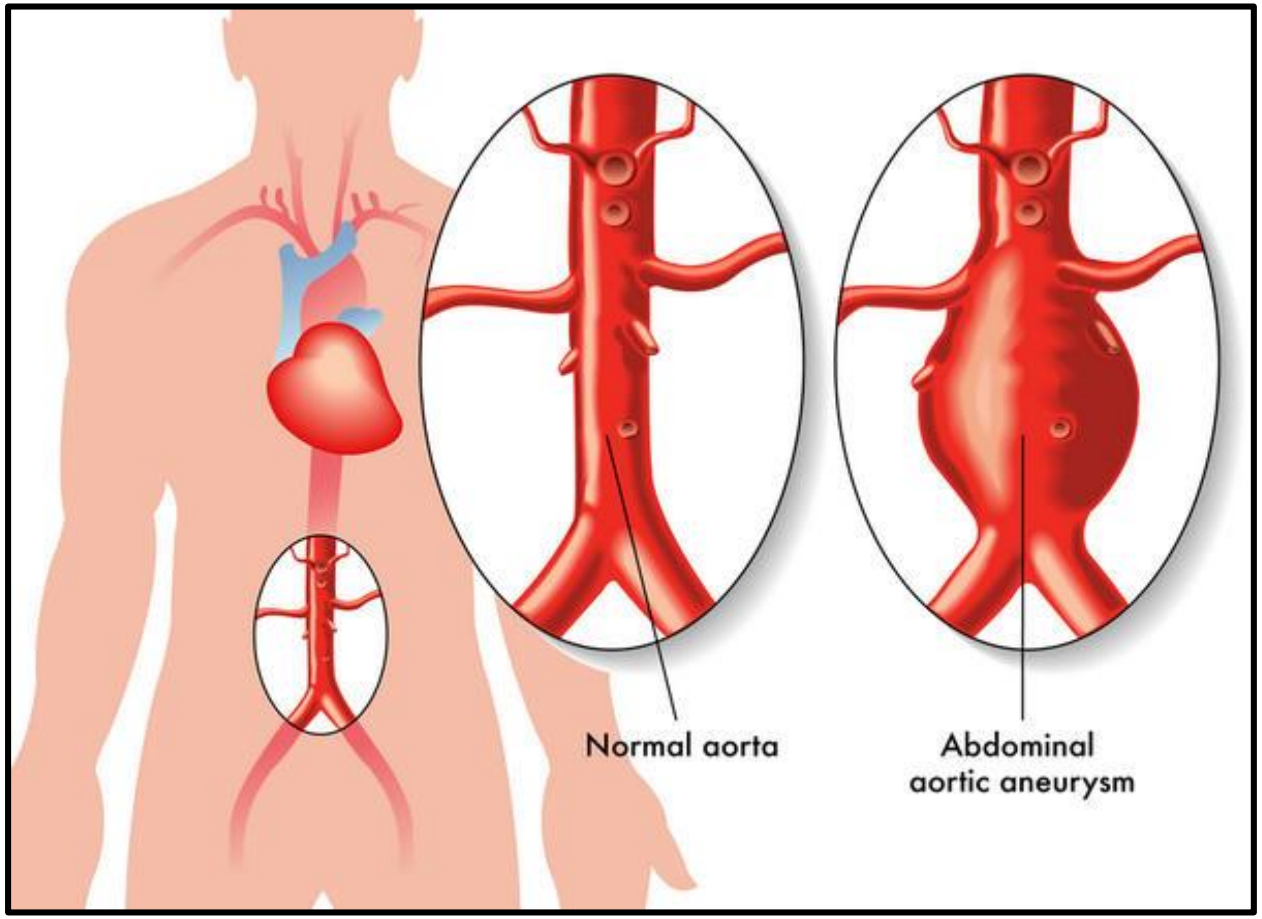
Aortic atherosclerosis - HPF



High power view of intimal aspect of atherosclerotic plaque showing stippling by blue calcific spherules, cholesterol crystal clefts, and fibrous cap.

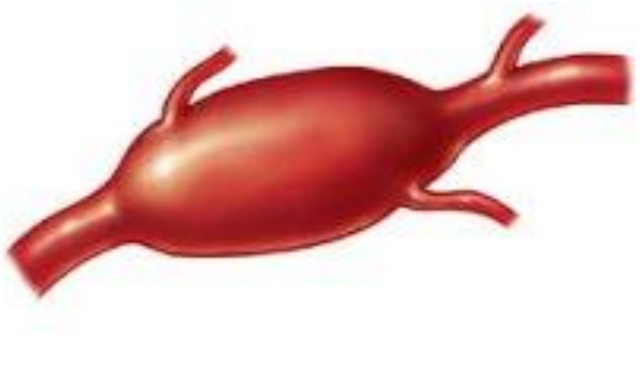
ANEURYSM OF ABDOMINAL AORTA

Abdominal Aortic Aneurysm



Types of Aneurysms

Saccular Aneurysm

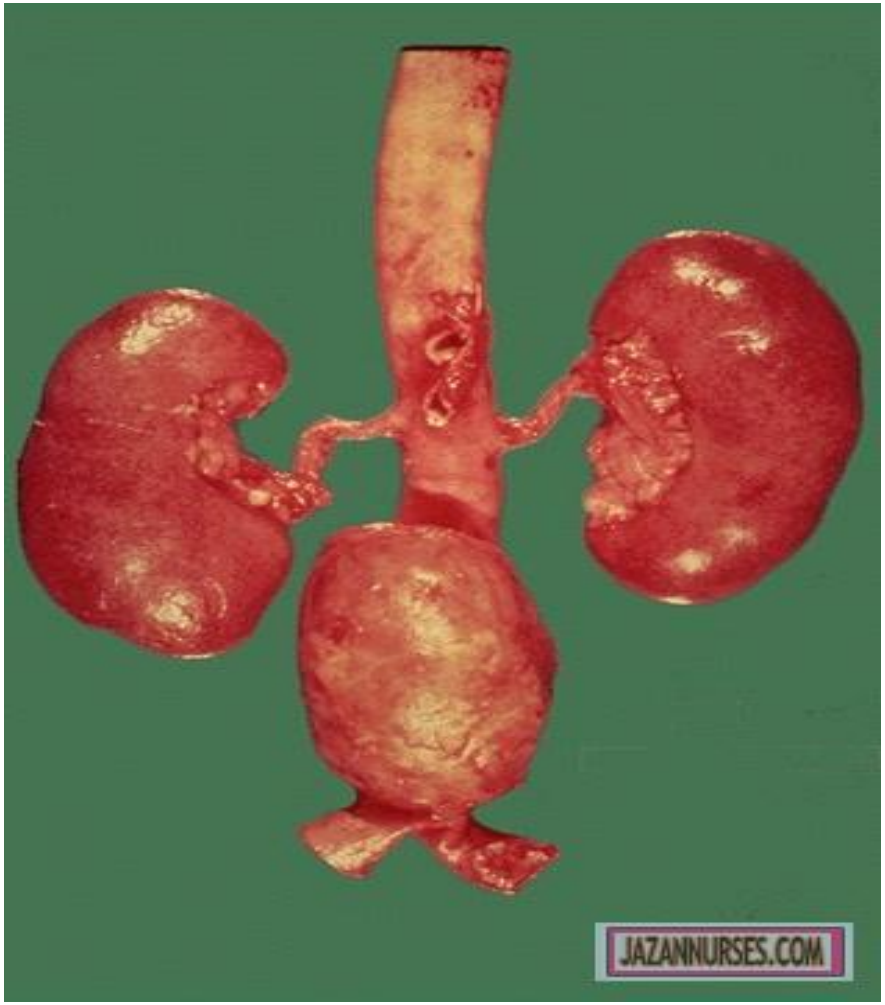


Fusiform Aneurysm

Ruptured Aneursym

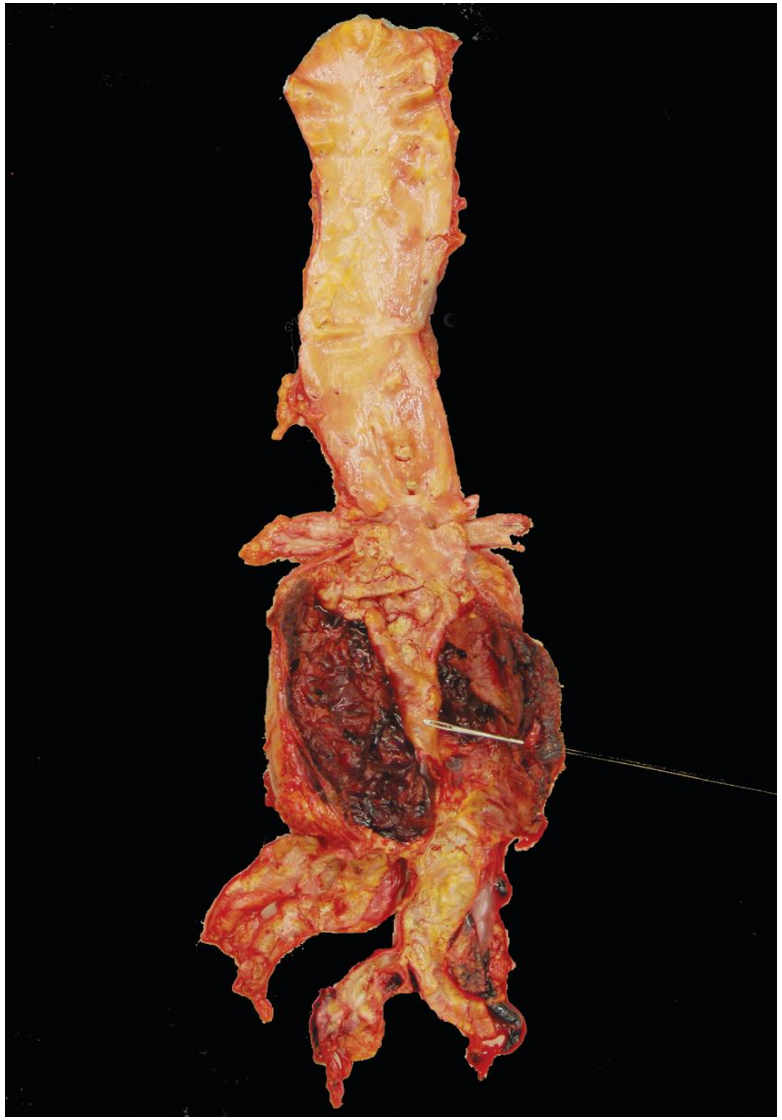
The most likely causes of aneurysms are atherosclerosis , mycotic, syphilitic and congenital

Abdominal Aortic Aneurysm



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

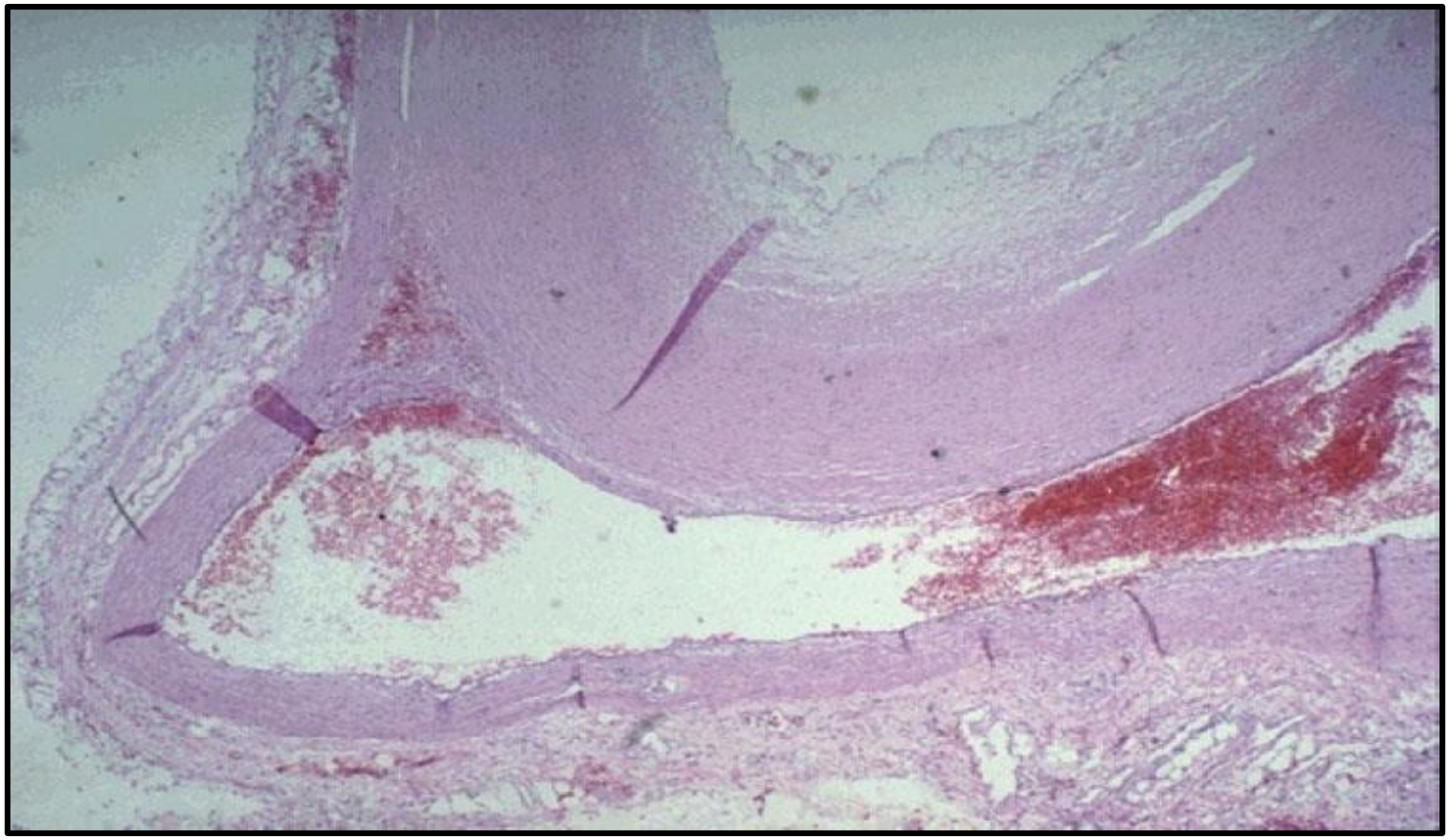
Abdominal Aortic Aneurysm



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

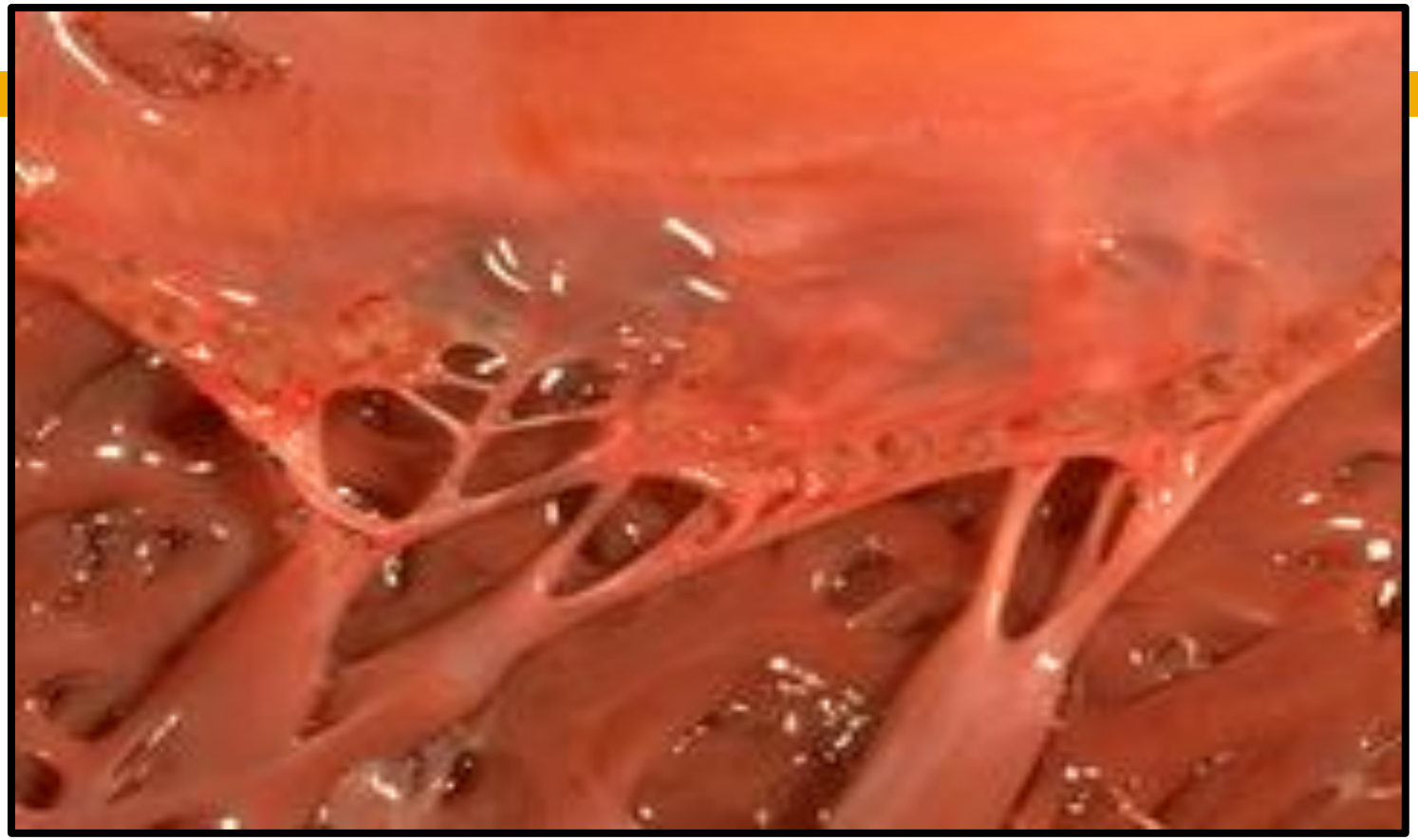
Dissecting aortic aneurysm - LPF



***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*

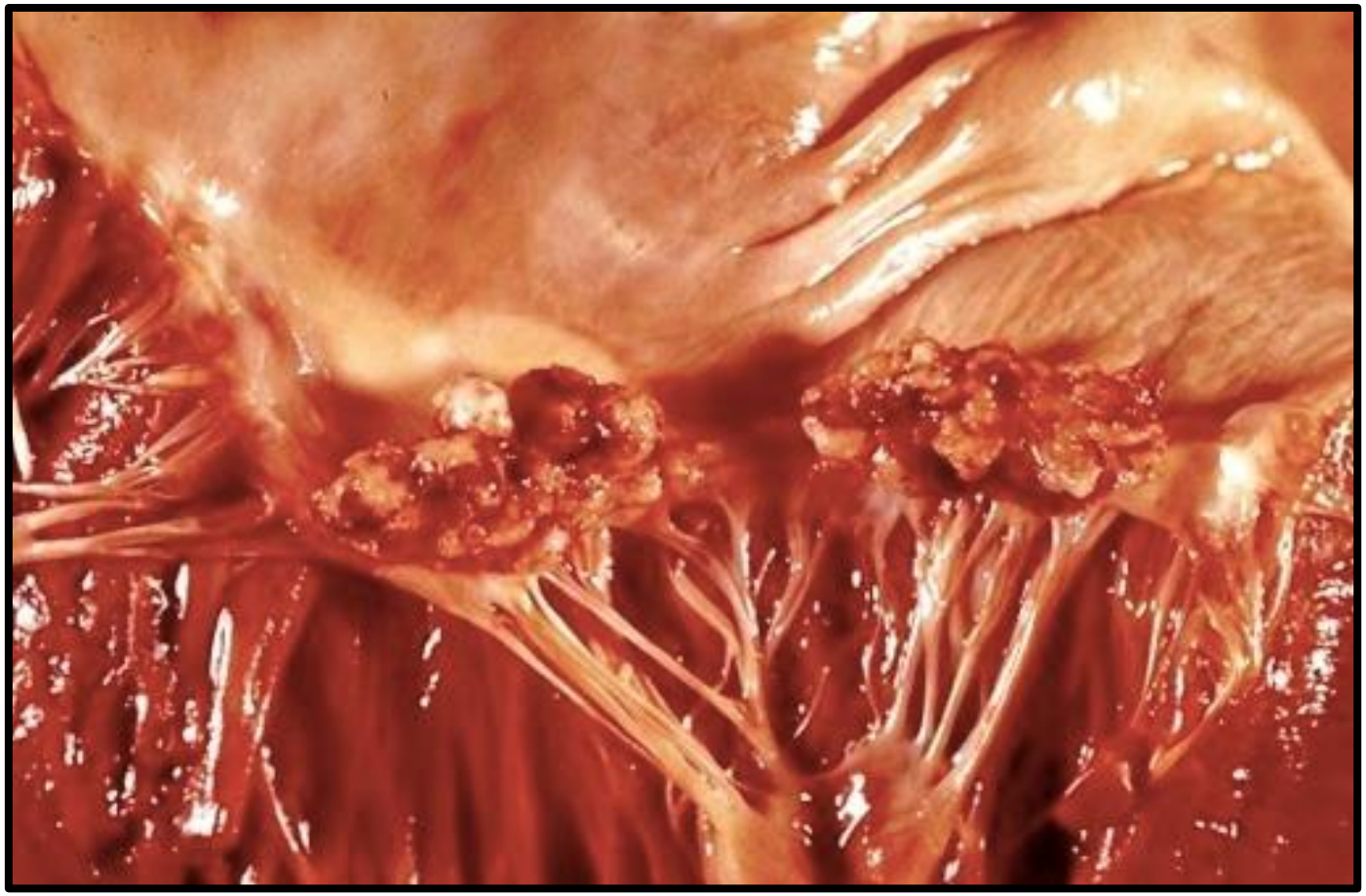
Vegetations of rheumatic fever on mitral and aortic valves and myocarditis

Acute Rheumatic Mitral Valvulitis - Gross



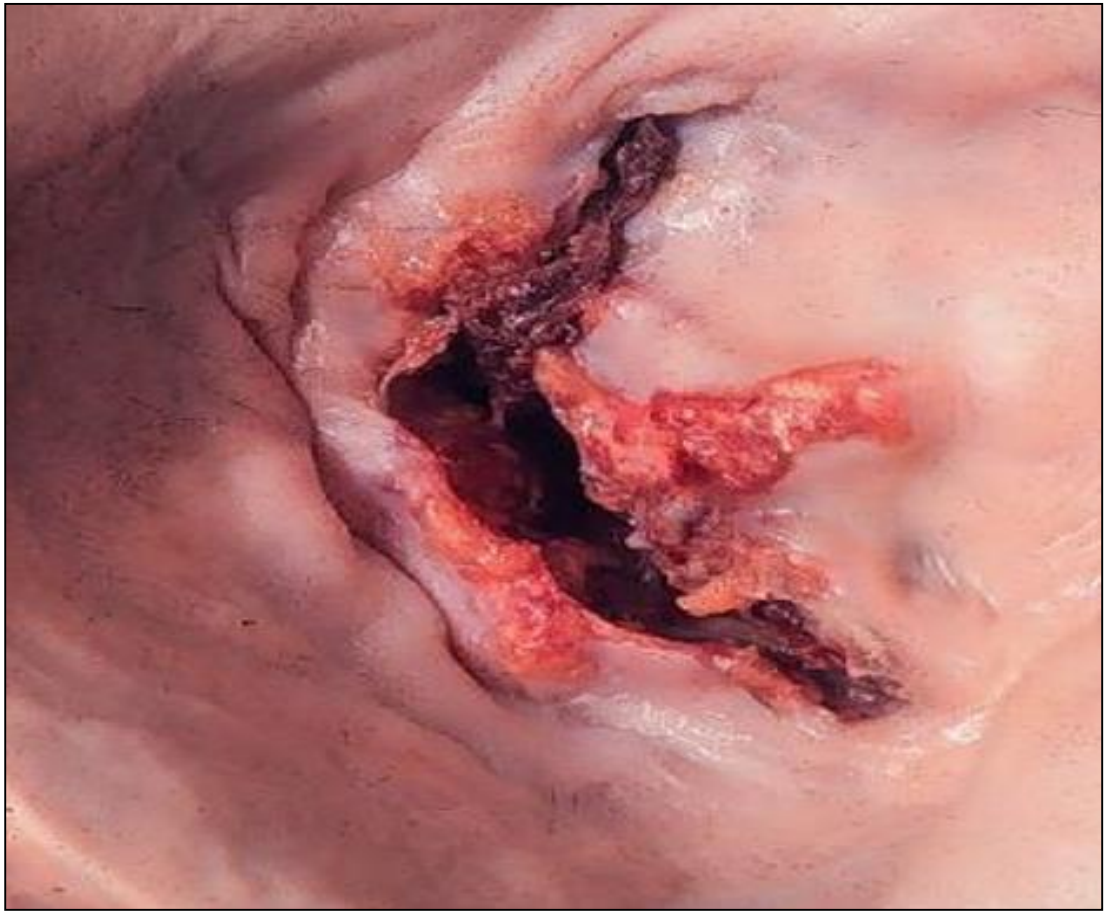
The small verrucous vegetations are associated with acute rheumatic fever. These warty vegetations are multiple, firm, adherent, small , 1-3 mm in- diameter and form along the line of valve closure over areas of endocardial inflammation. Affects mainly Aortic & Mitral valves

Chronic Rheumatic Mitral Valvulitis - Gross



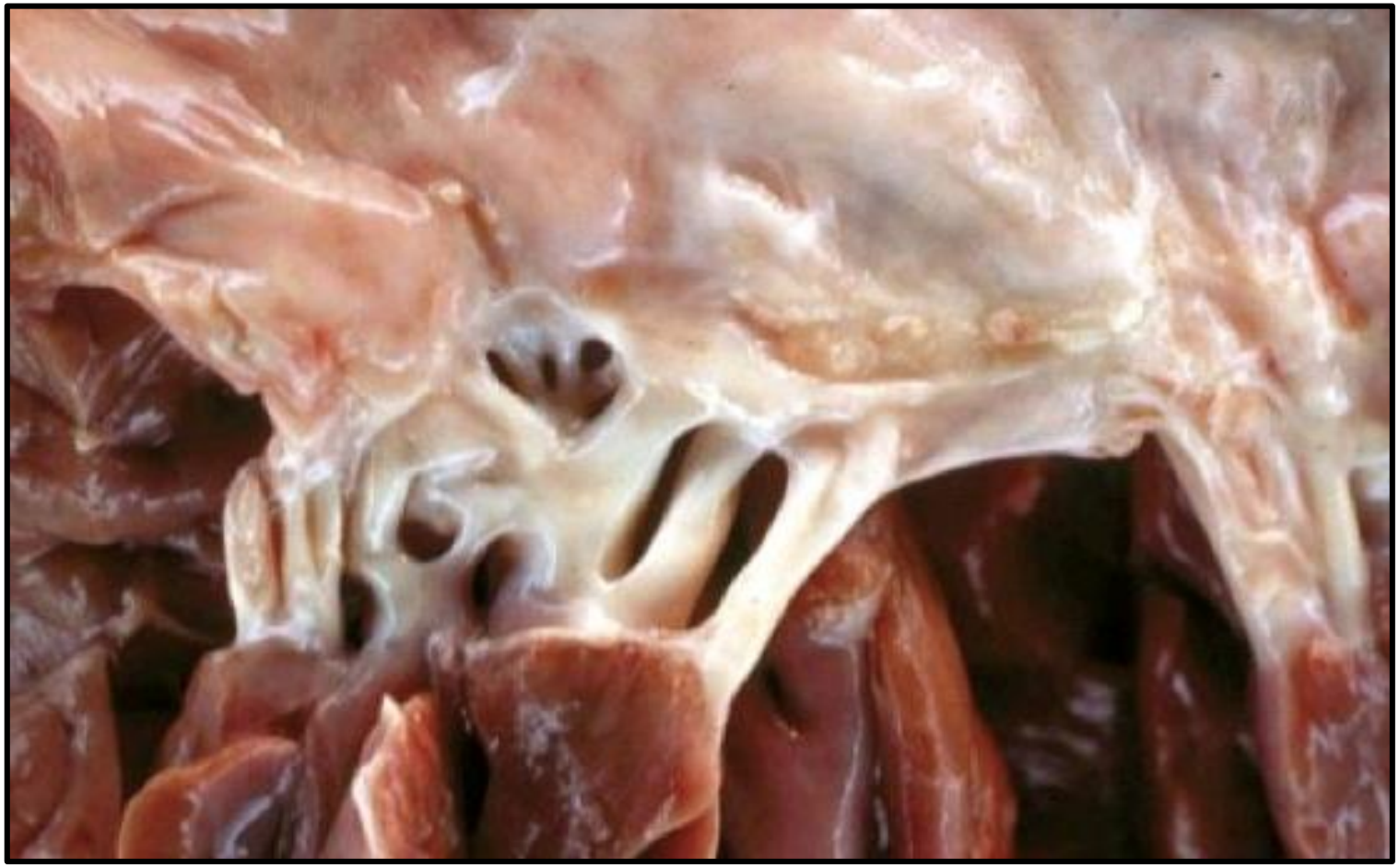
large vegetations/hemorrhage along the free margins of the mitral valve.

Chronic Rheumatic Mitral Valvulitis - Gross



- Fusion of the commissures.
- Calcification of the cusps.
- Vegetations.
- **"Fish-mouth"** deformity of the aortic valve.

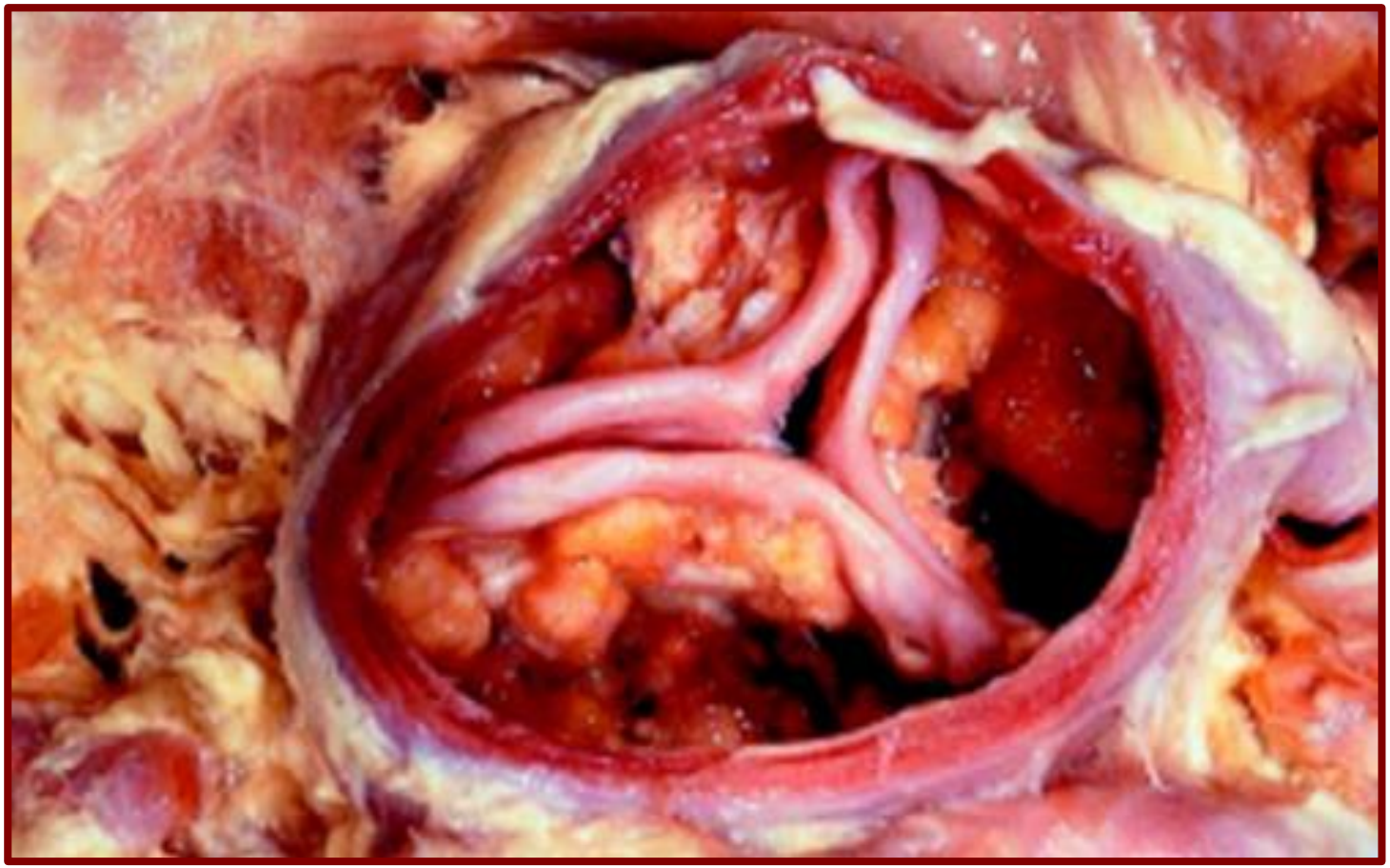
Chronic Rheumatic Mitral Valvulitis - Gross



Chronic rheumatic mitral valvulitis

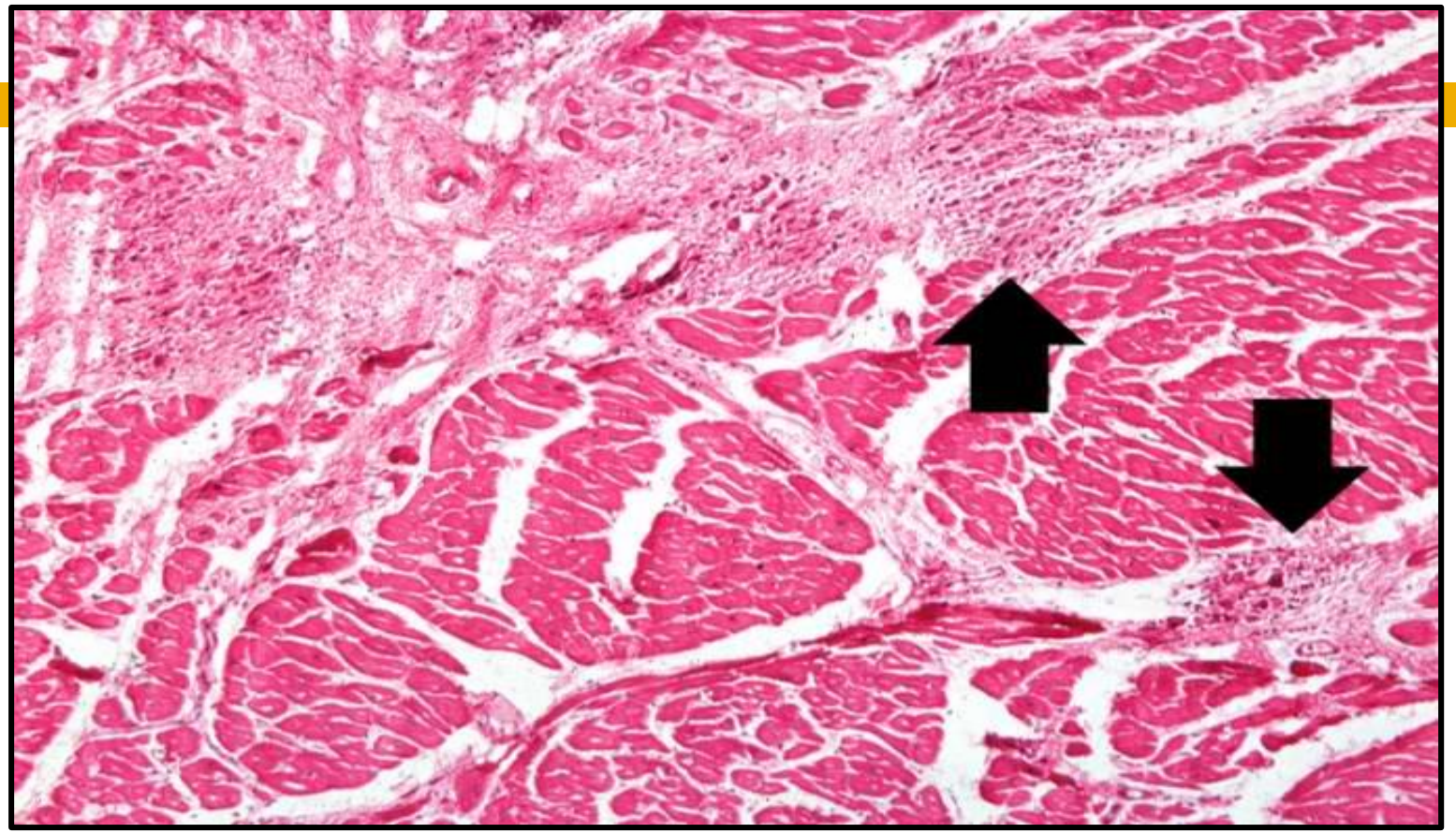
the valve leaflets are thick, fibrotic, fused. Short, thickened, fused chordae tendinae ➡ stenosis and / or incompetence

Rheumatic Aortic Valvulitis - Gross



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

Rheumatic myocarditis - LPF

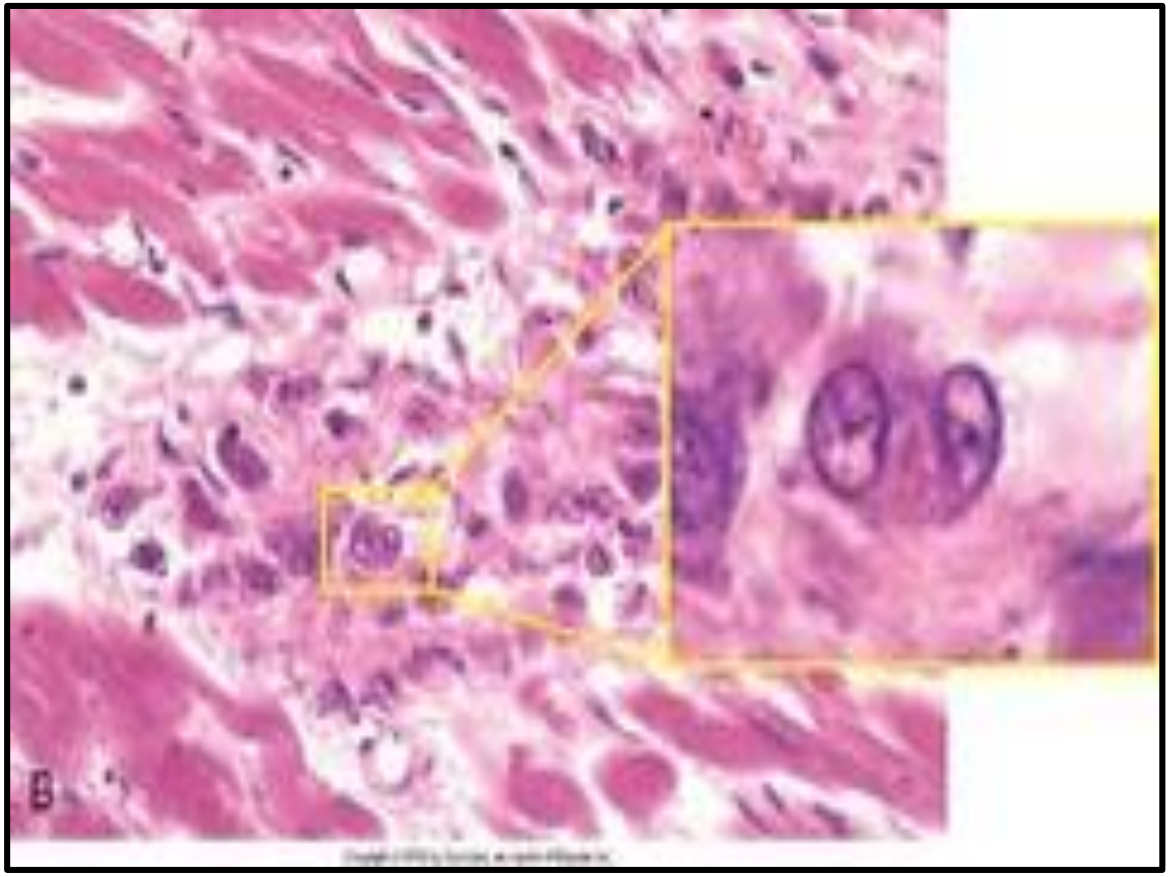


The myocardium showing cellular accumulations - Aschoff bodies (arrows) - within the interstitium of the myocardium.

Major Jones criteria:

- *Carditis.*
- *Polyarthritis.*
- *Chorea.*
- *Erythema marginatum.*
- *Subcutaneous nodules*

Acute Rheumatic Carditis - HPF



- Anti-streptolysin Antibodies *is the* serological test that can help to establish the aetiology and ***indicates previous streptococcal infection.***

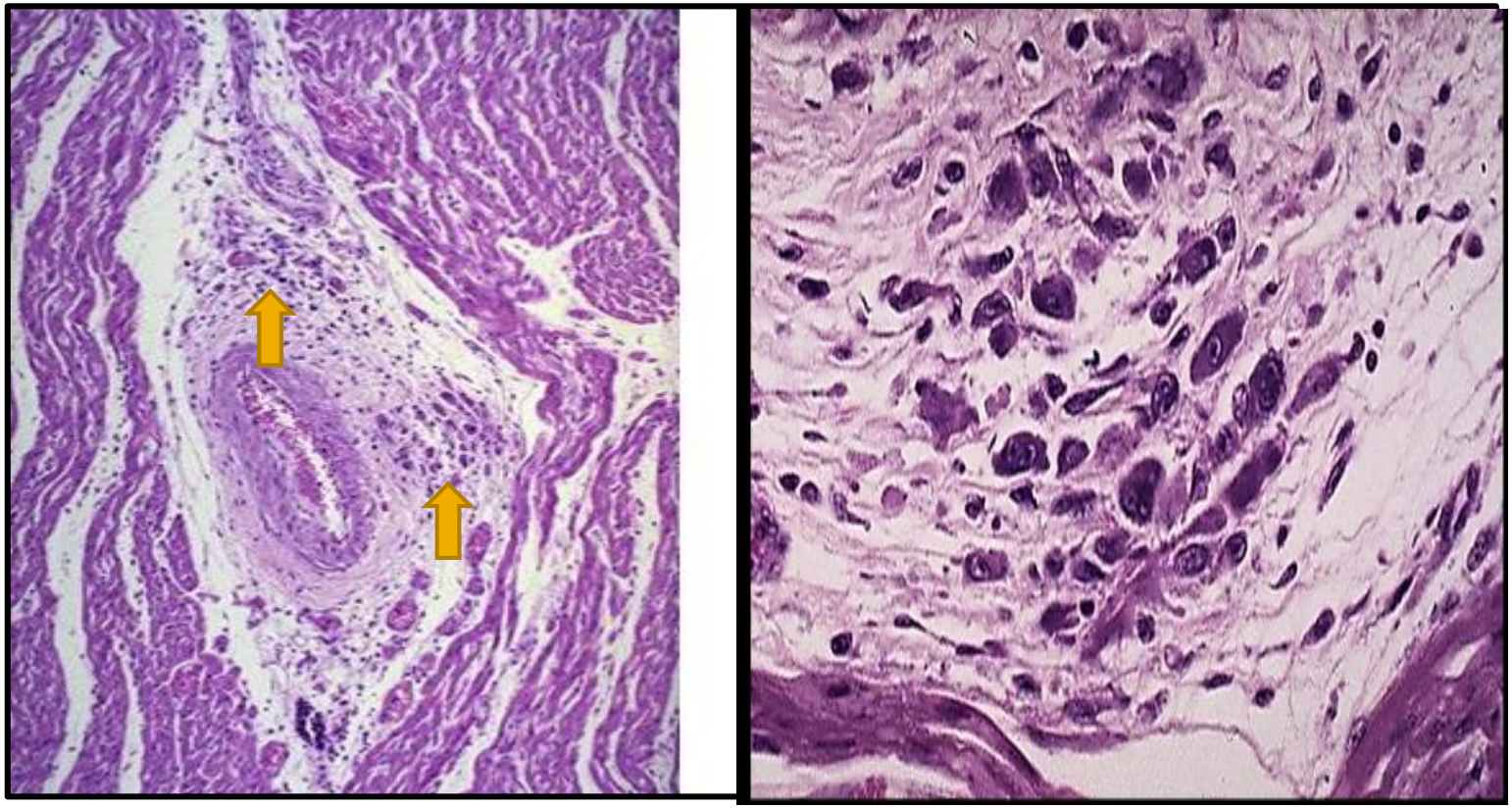
***An Aschoff nodule at high magnification*.**

It is formed by (Main component):

-Macrophages including Ashoff giant cells.

- Collagen necrosis

RHEUMATIC MYOCARDIITIS (ASHOFF NODULE)



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

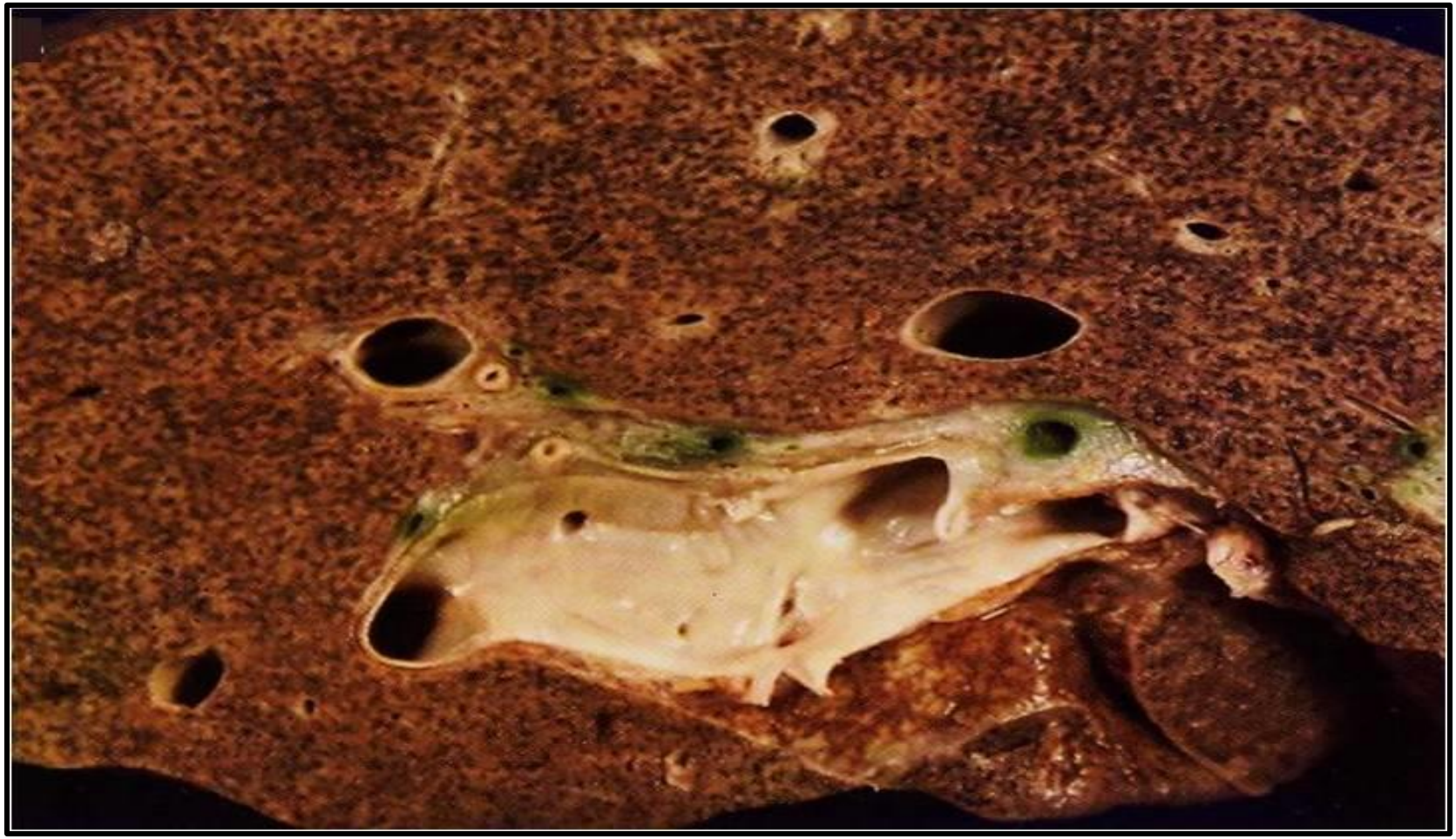
Each consists of a focus of fibrinoid necrosis, few lymphocytes, macrophages and few small giant cells with one or several nuclei (Aschoff giant cell).

HEART FAILURE

Right Sided Heart Failure

Chronic venous congestion of the liver

NUTMEG LIVER – Cut surface

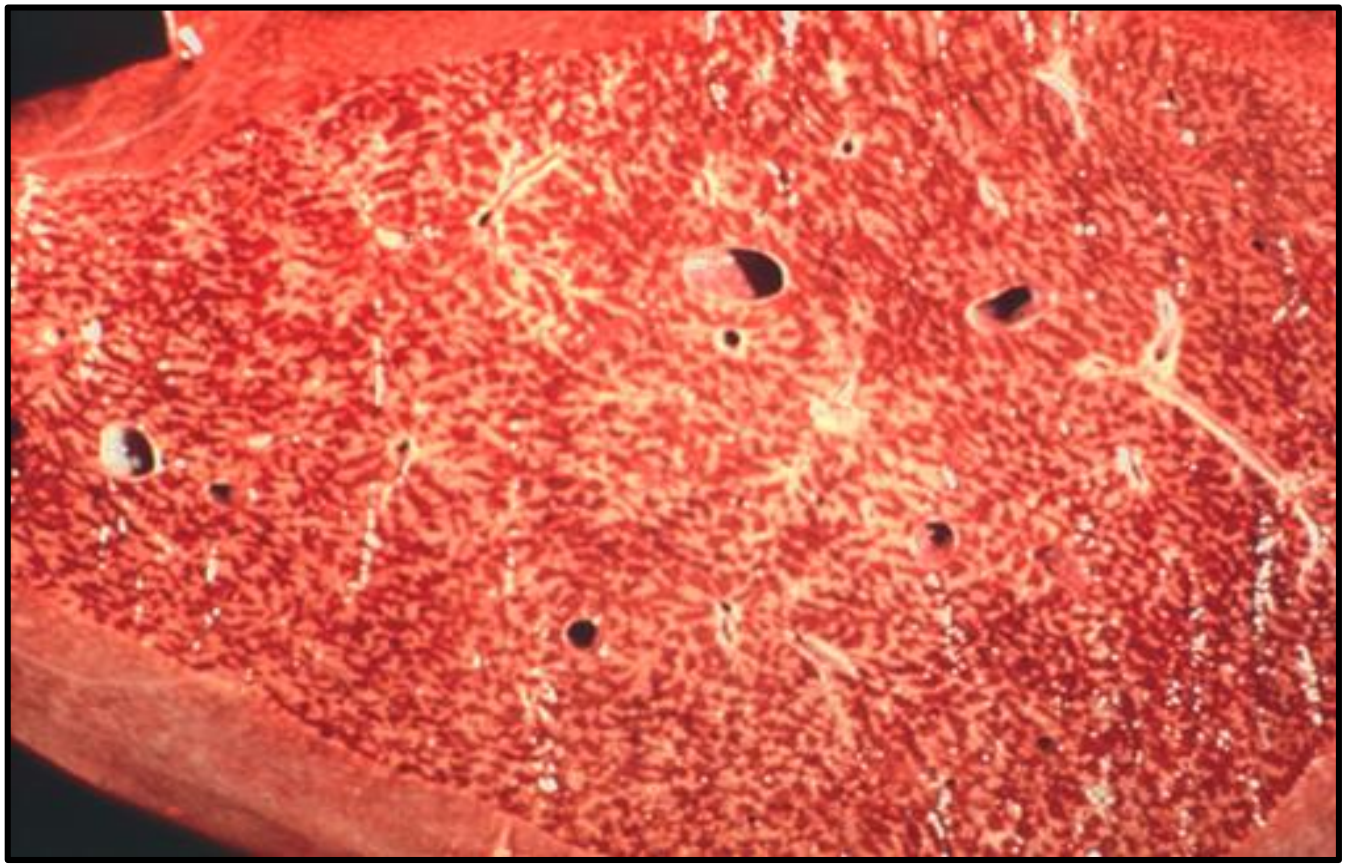


Section of liver showing alternating pale and dark areas with a nutmeg like appearance possibly due to passive congestion secondary to right sided heart failure.

NUTMEG LIVER – Cut surface

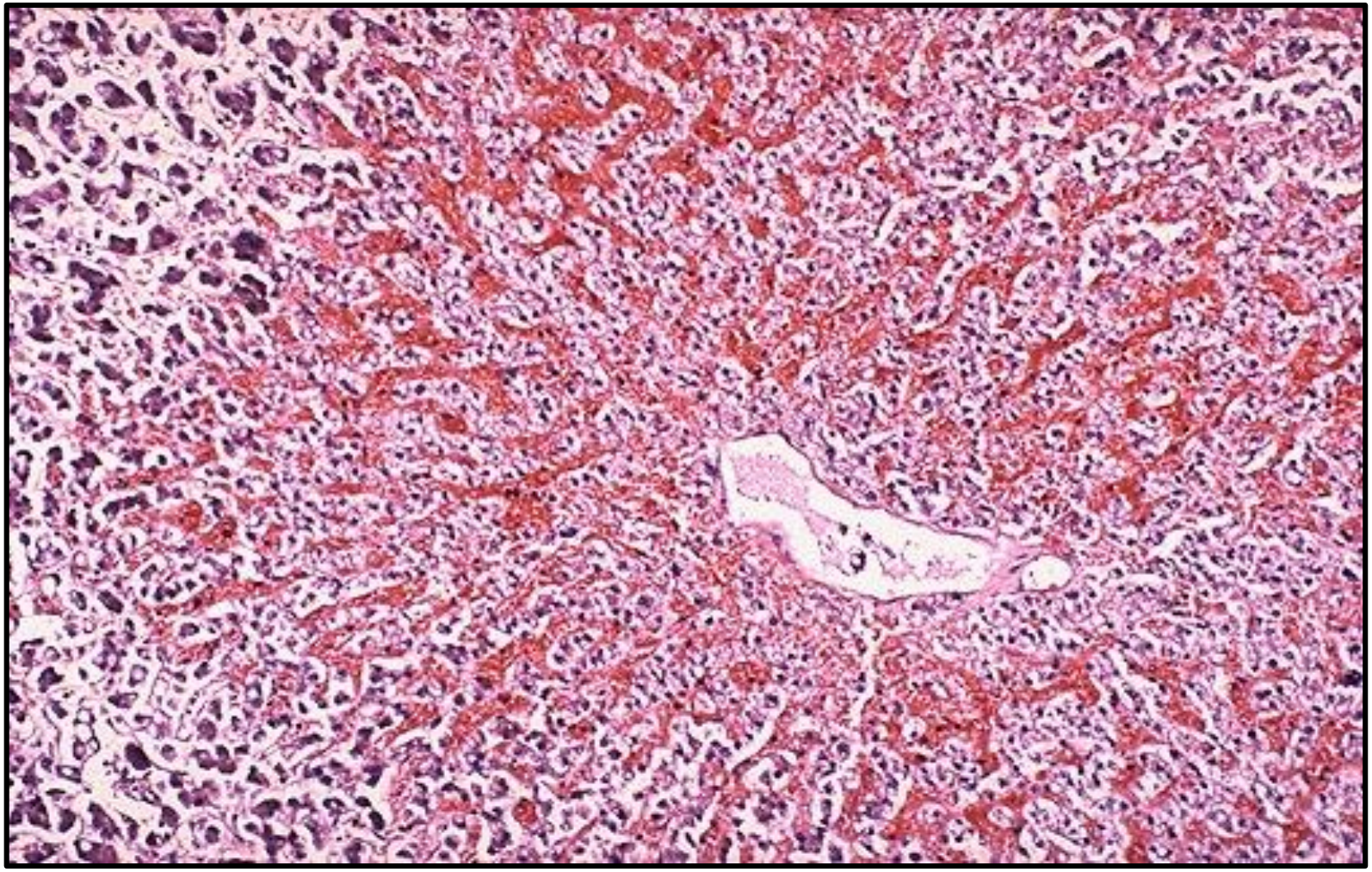
The hepatic parenchyma contains a faintly nodular pattern and nutmeg staining due to chronic passive congestion due to Right sided heart failure.

Chronic Congestion of the Liver - CS



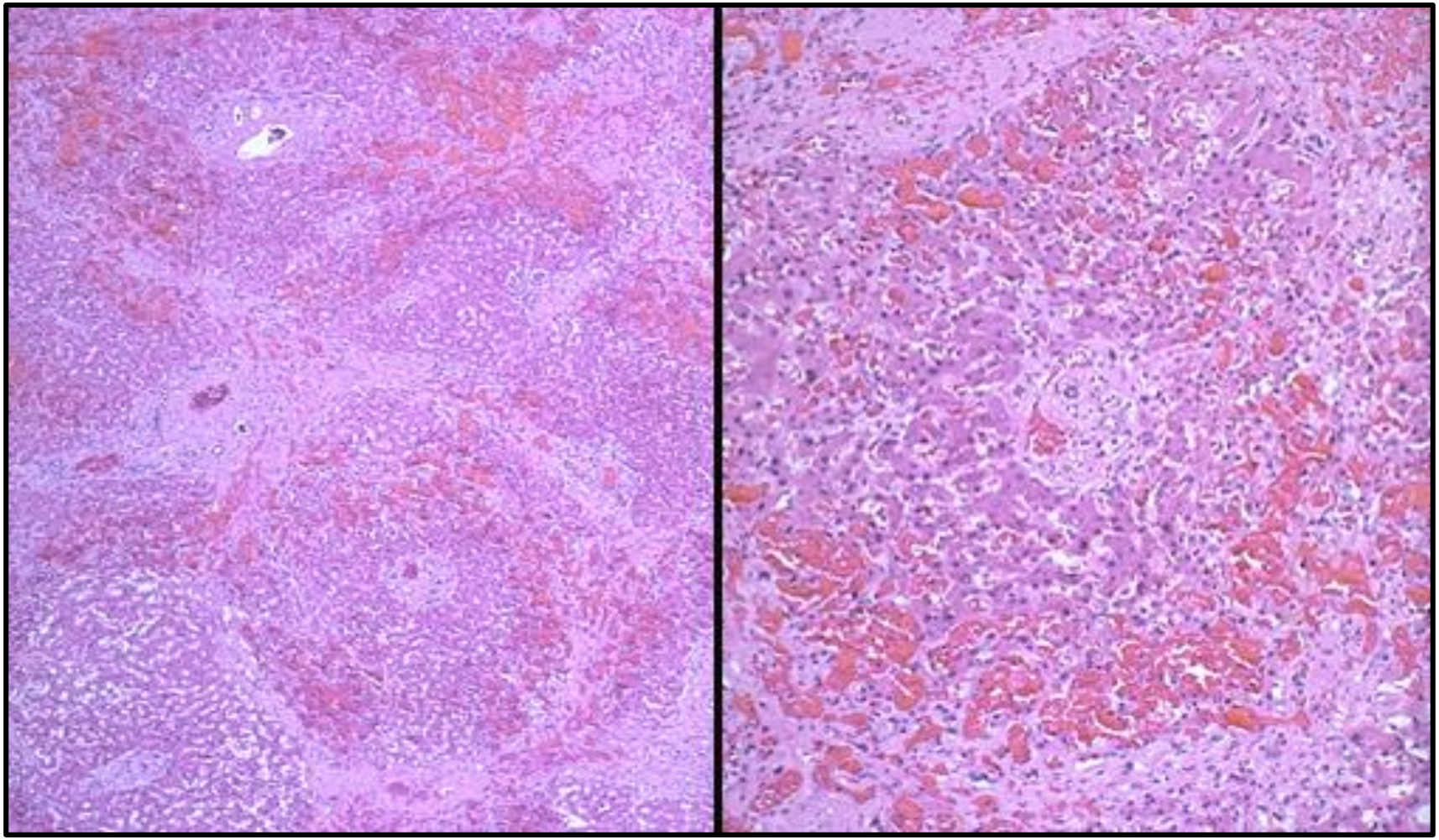
A gross view of nutmeg appearance of liver characteristic of centrolobular or necrosis or passive congestion of the liver. The central areas of the liver are congested and take on a sort of dusky appearance. They are soft in consistency and they are surrounded by the paler areas of fatty liver that are more normal in appearance microscopically.

Chronic Congestion of the Liver - LPF



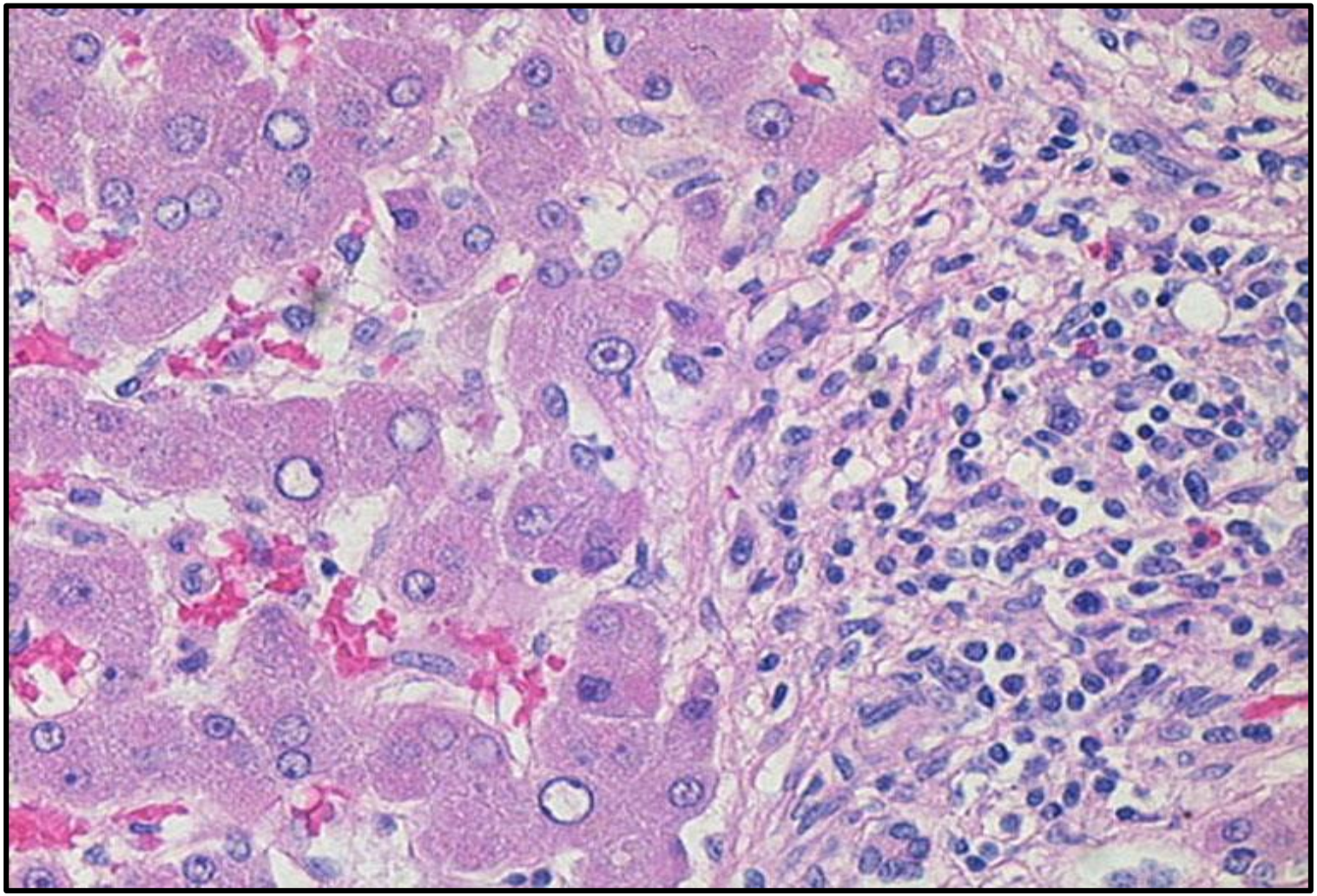
The central portion of liver lobules shows congestion and dilatation of central veins and blood sinusoids, with atrophy and necrosis of liver cells.

Chronic Congestion of the Liver - LPF



Central veins dilated and congested , necrotic hepatocytes , kupffer cells and steatosis

Chronic Congestion of the Liver - HPF

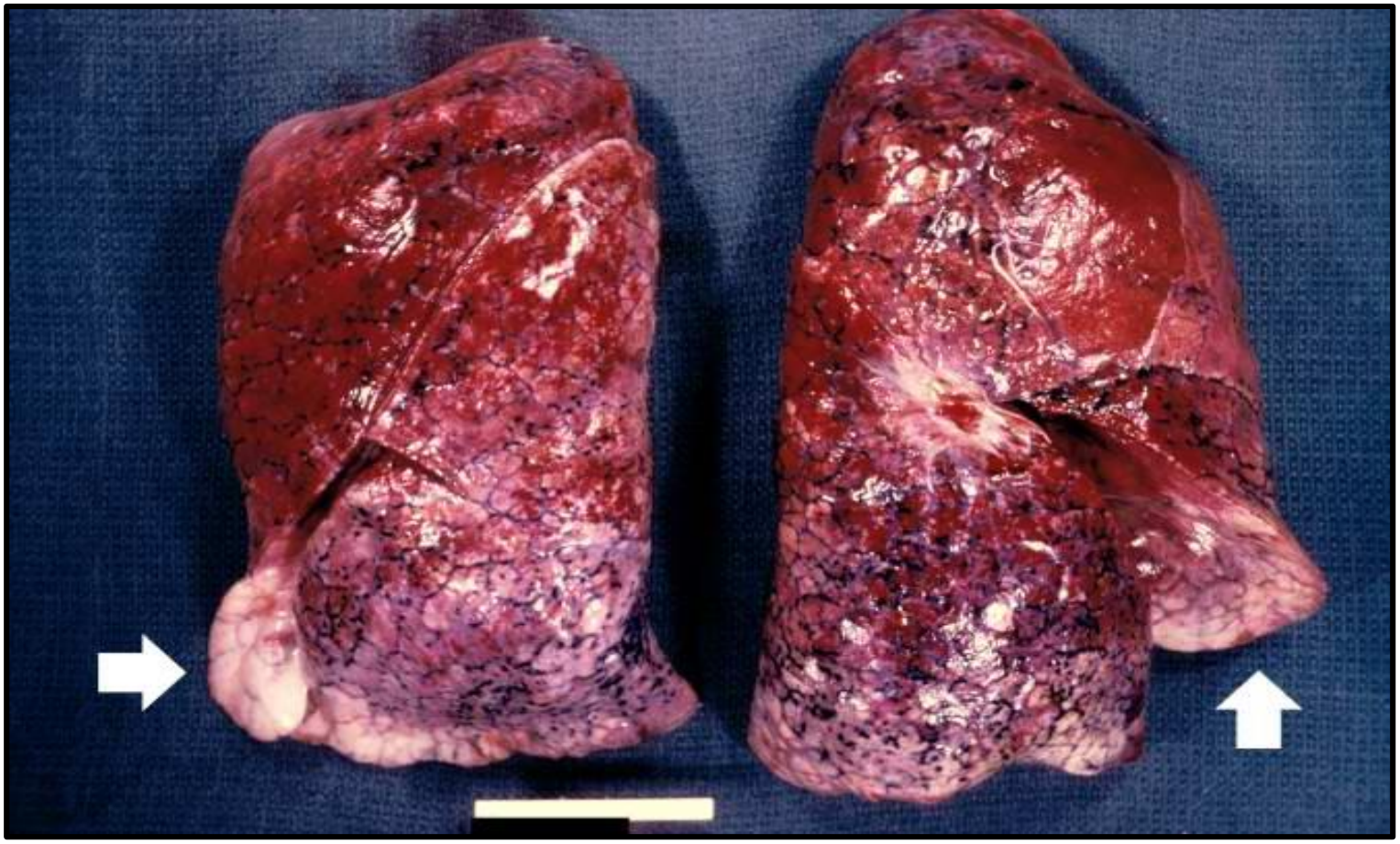


The central portion of liver lobules shows congestion and dilatation of central veins and blood sinusoids, with atrophy and necrosis of liver cells.

Left Sided Heart Failure

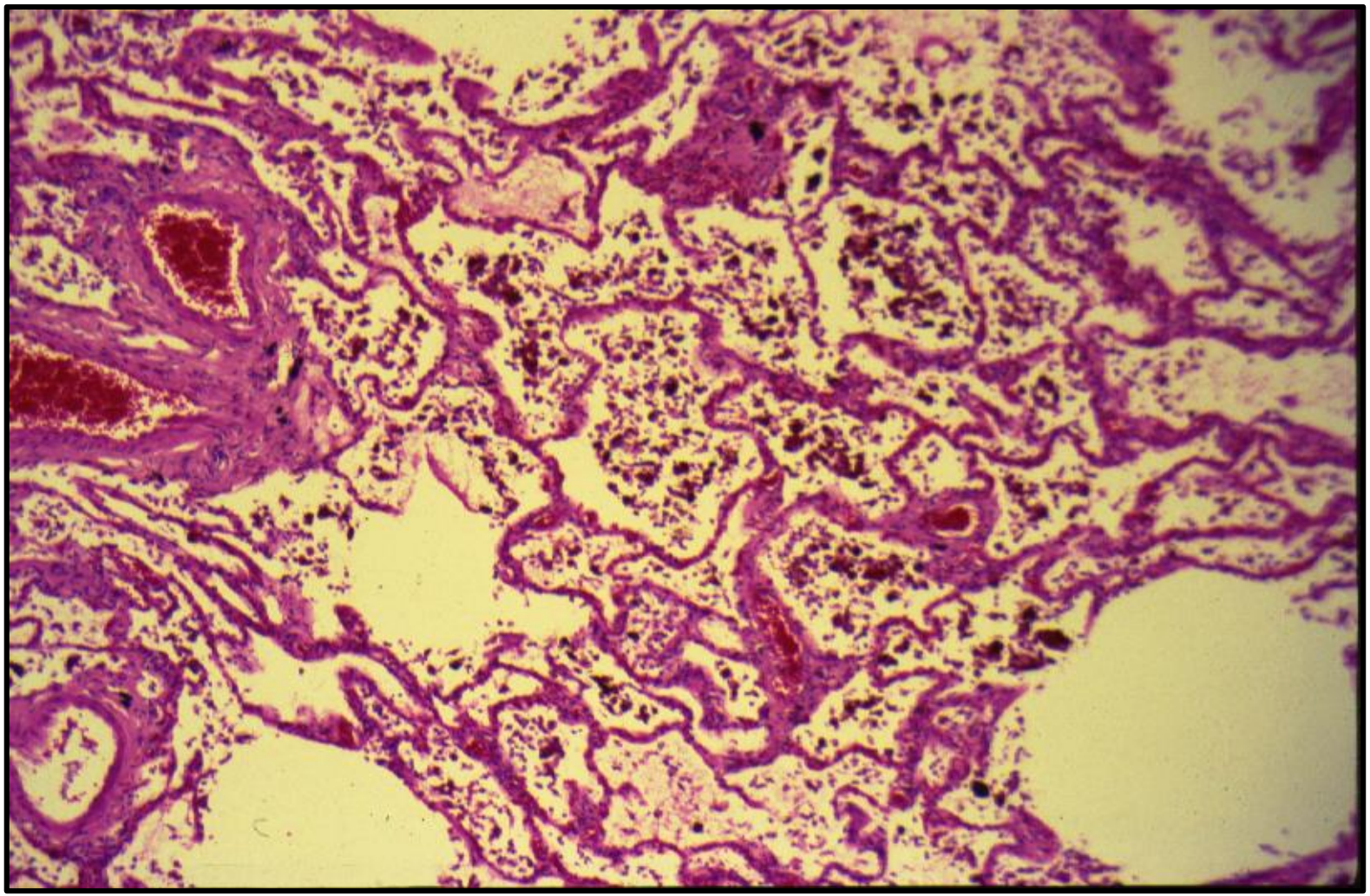
Chronic venous congestion of the lung

Chronic venous congestion of the lung - Gross



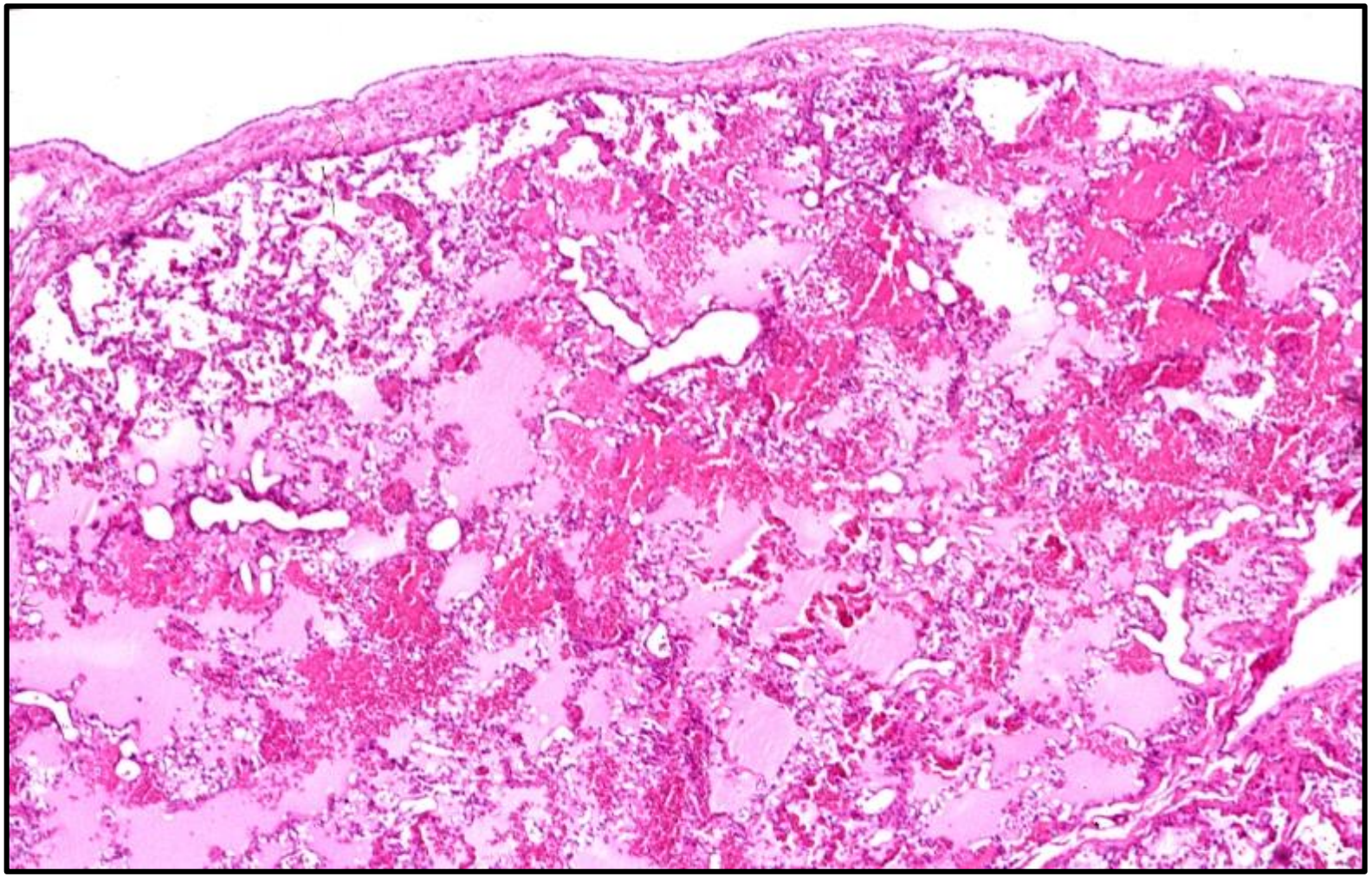
This is a gross photograph of lungs that are distended and red. The reddish coloration of the tissue is due to congestion. Some normal pink lung tissue is seen at the edges of the lungs (arrows).

Chronic venous congestion of the lung - LPF



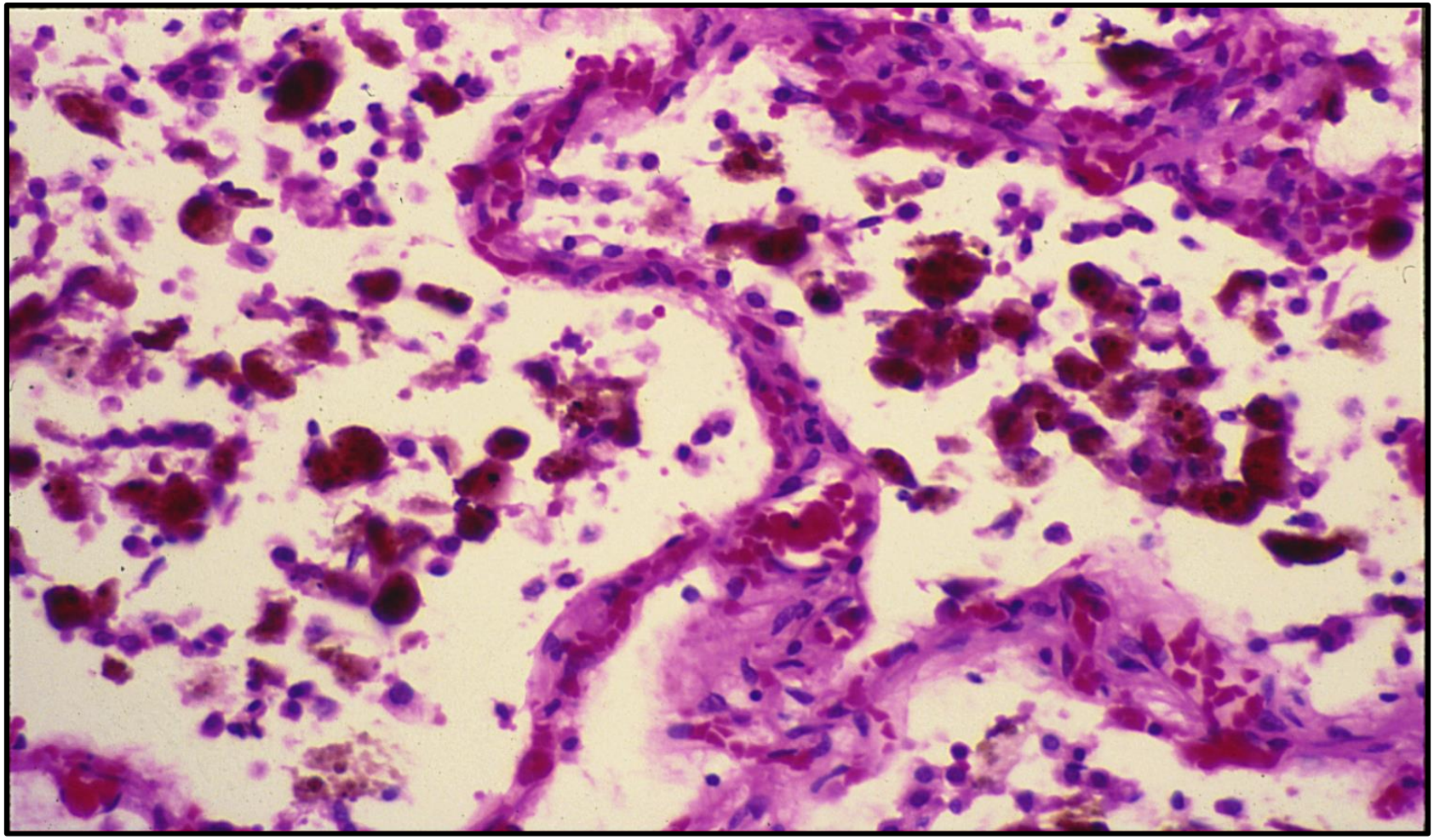
The alveolar walls are thickened by dilated and engorged capillaries.

Chronic venous congestion of the lung - LPF



Lung, pulmonary edema in patient with congestive heart failure due to heart transplant rejection

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.

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

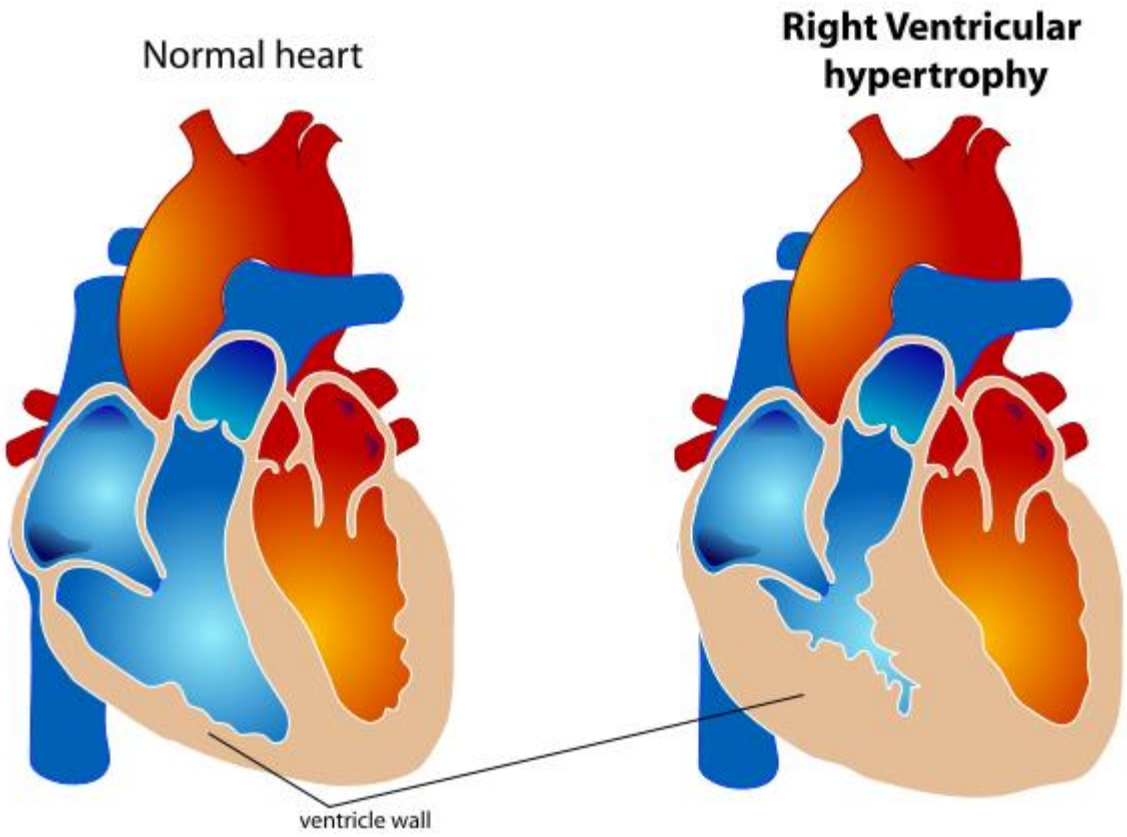
Left ventricular hypertrophy :

- Systemic hypertension
- Aortic valve stenosis

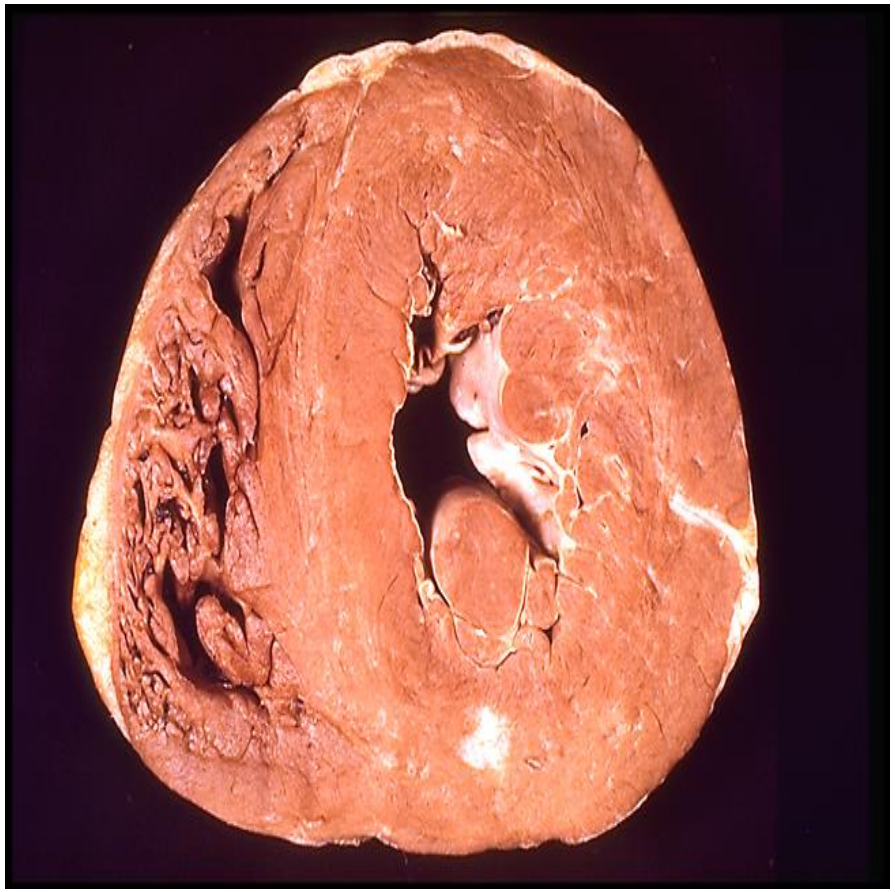
Right ventricular hypertrophy:

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

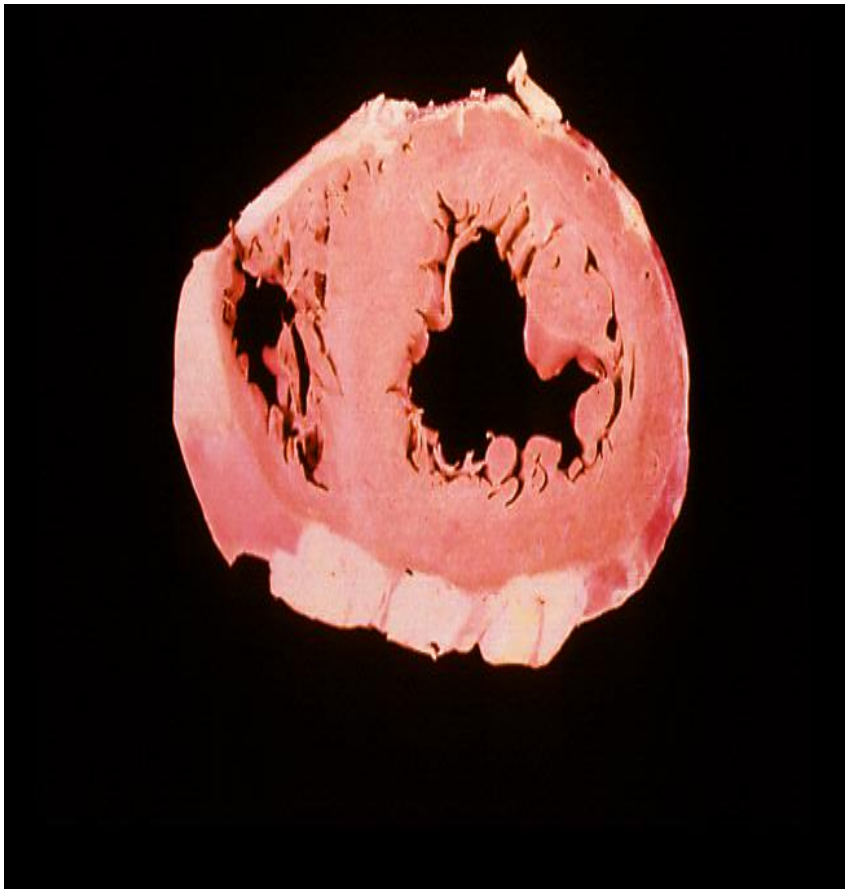
Right ventricular hypertrophy



Normal and hypertrophied left ventricle – cross section

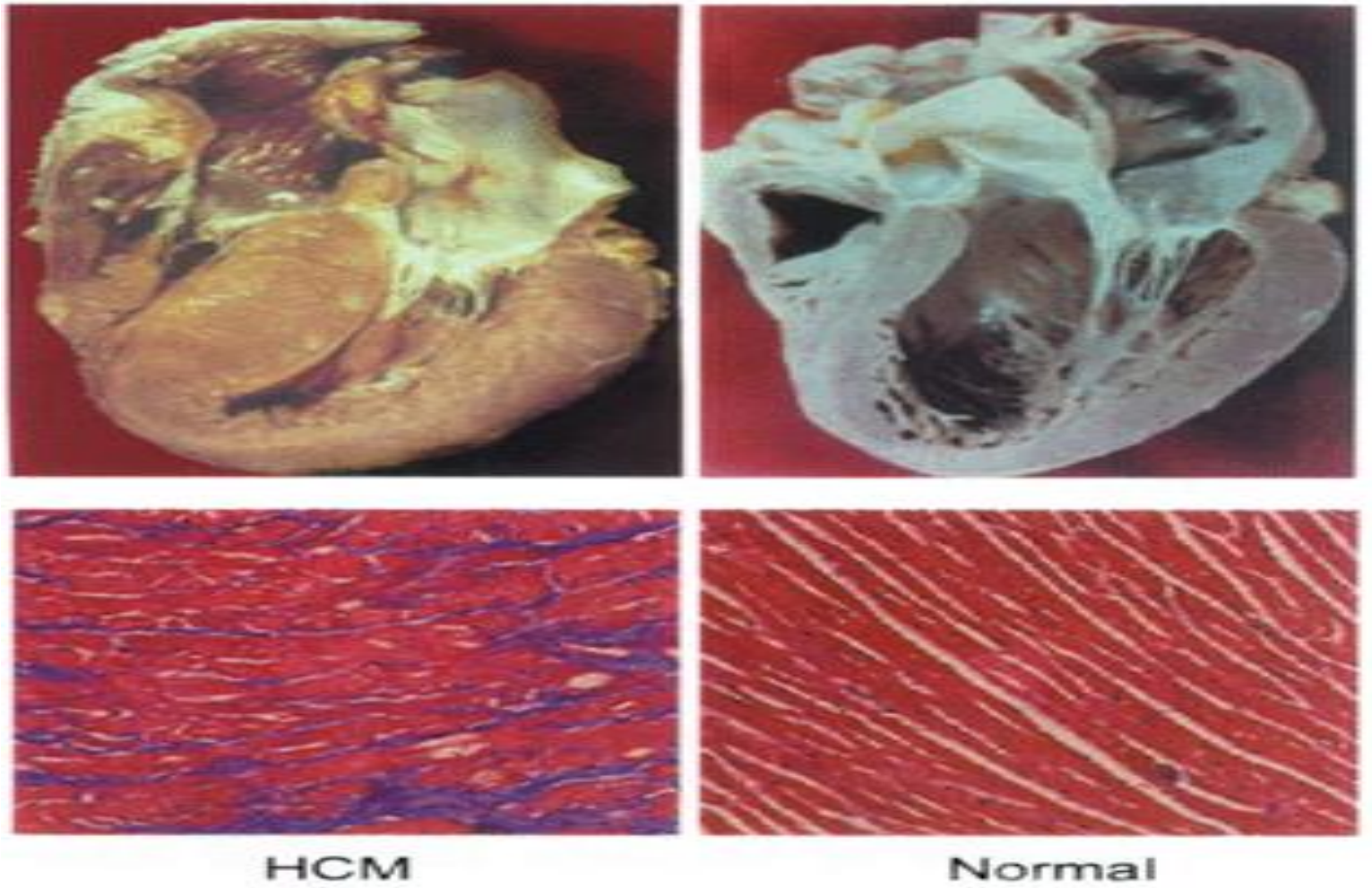


Left ventricular hypertrophy



Normal ventricles

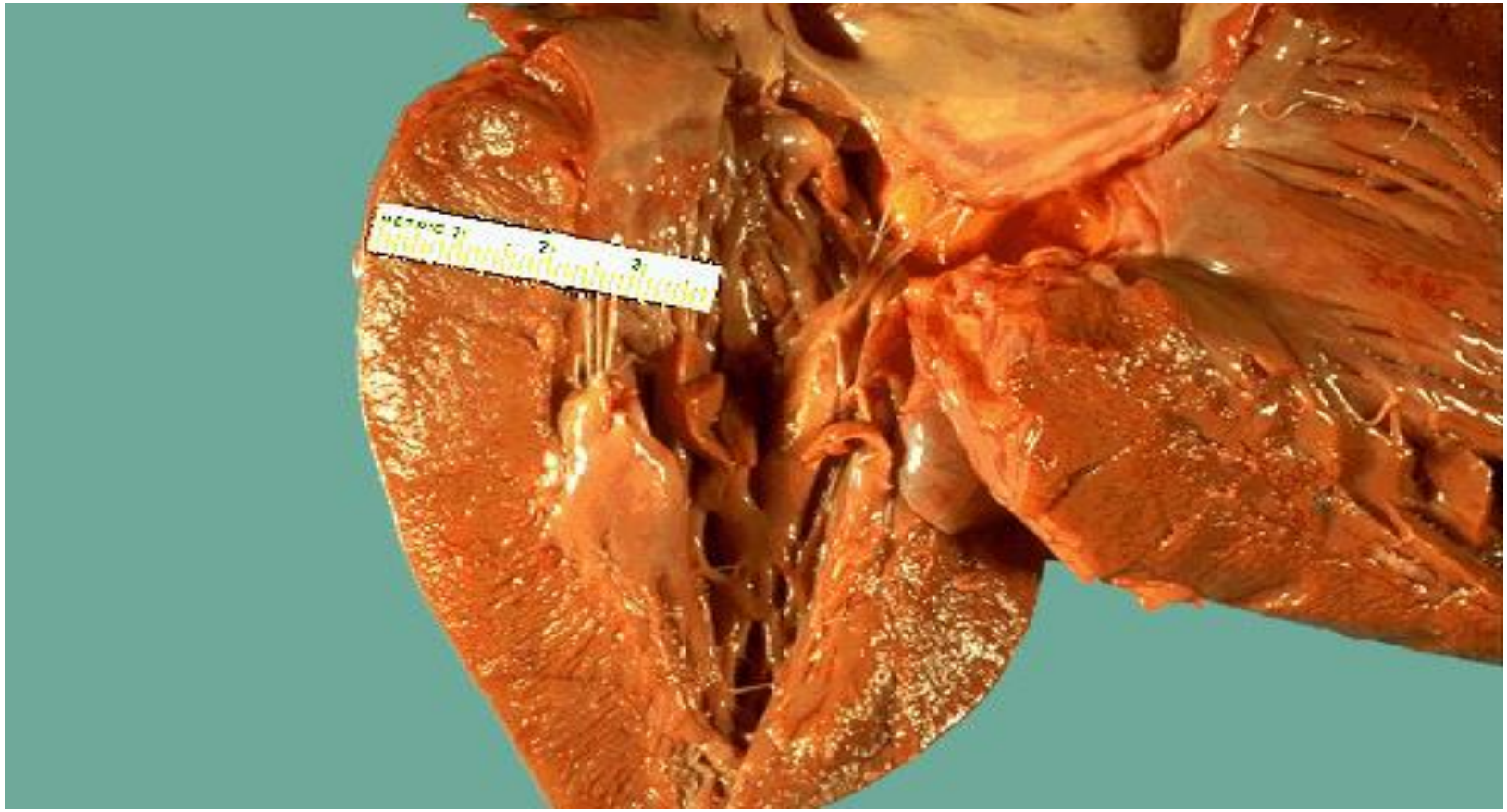
Normal and hypertrophied left ventricle - CS



Histopathology showing significant myofiber disarray and interstitial fibrosis

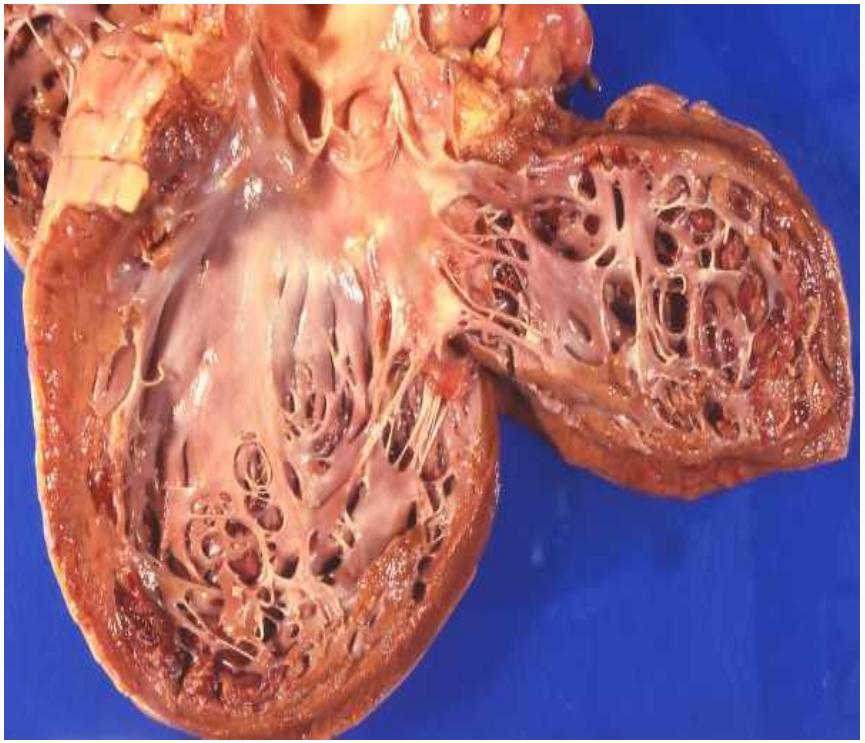
Histopathology showing Normal myocytes

Left ventricular hypertrophy - Gross



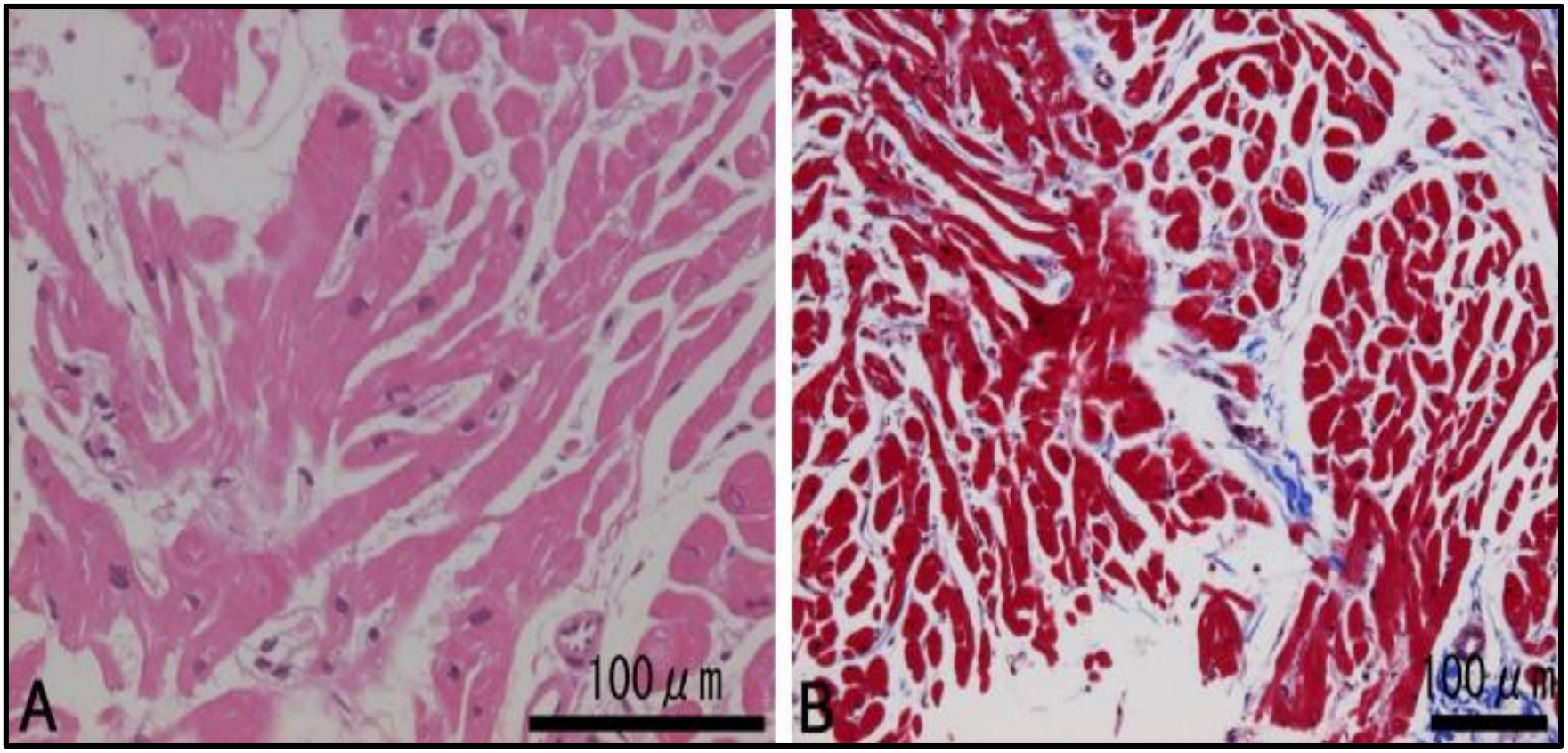
Heart from a hypertensive patient. The left ventricle is very thick (over 2 cm). However the rest of the heart is fairly normal in size as is typical for hypertensive heart disease. The hypertension creates a greater pressure load on the heart to induce the hypertrophy

Left ventricular hypertrophy - Gross



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

Hypertrophic Cardiomyopathy - LPF

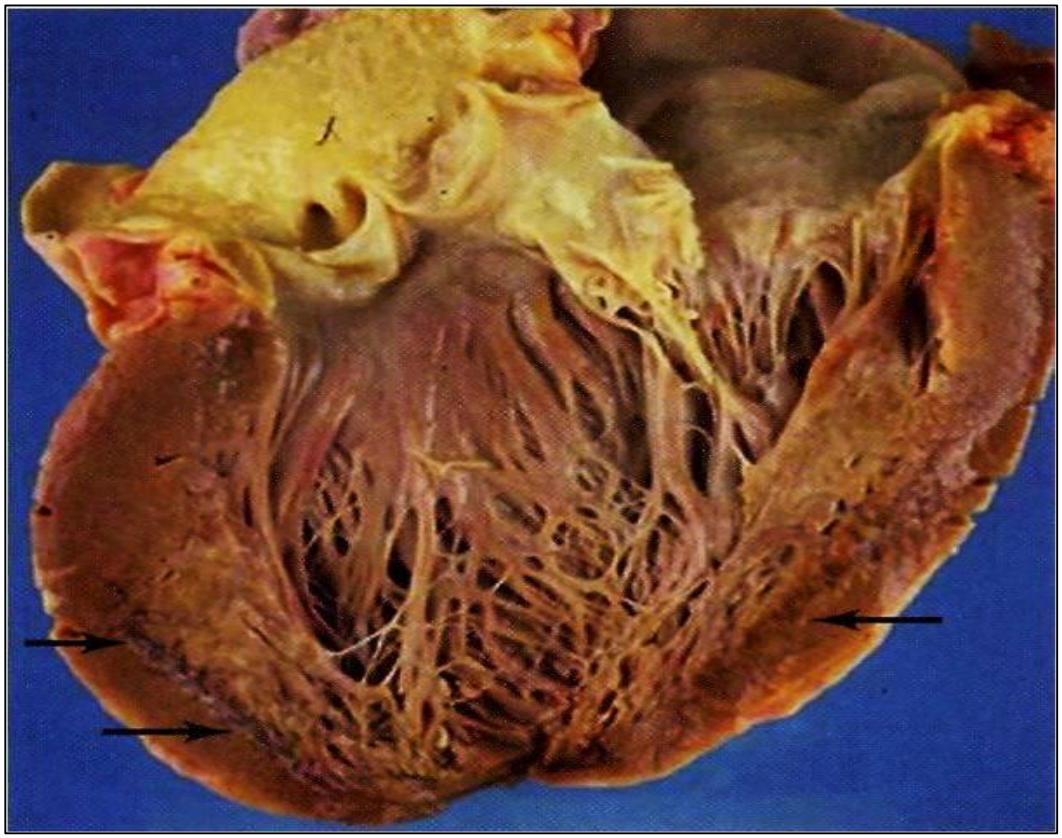


haematoxylin-eosin stain ***Masson's trichrome stain***

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

MYOCARDIAL INFARCTION

Myocardial Infarction - CS

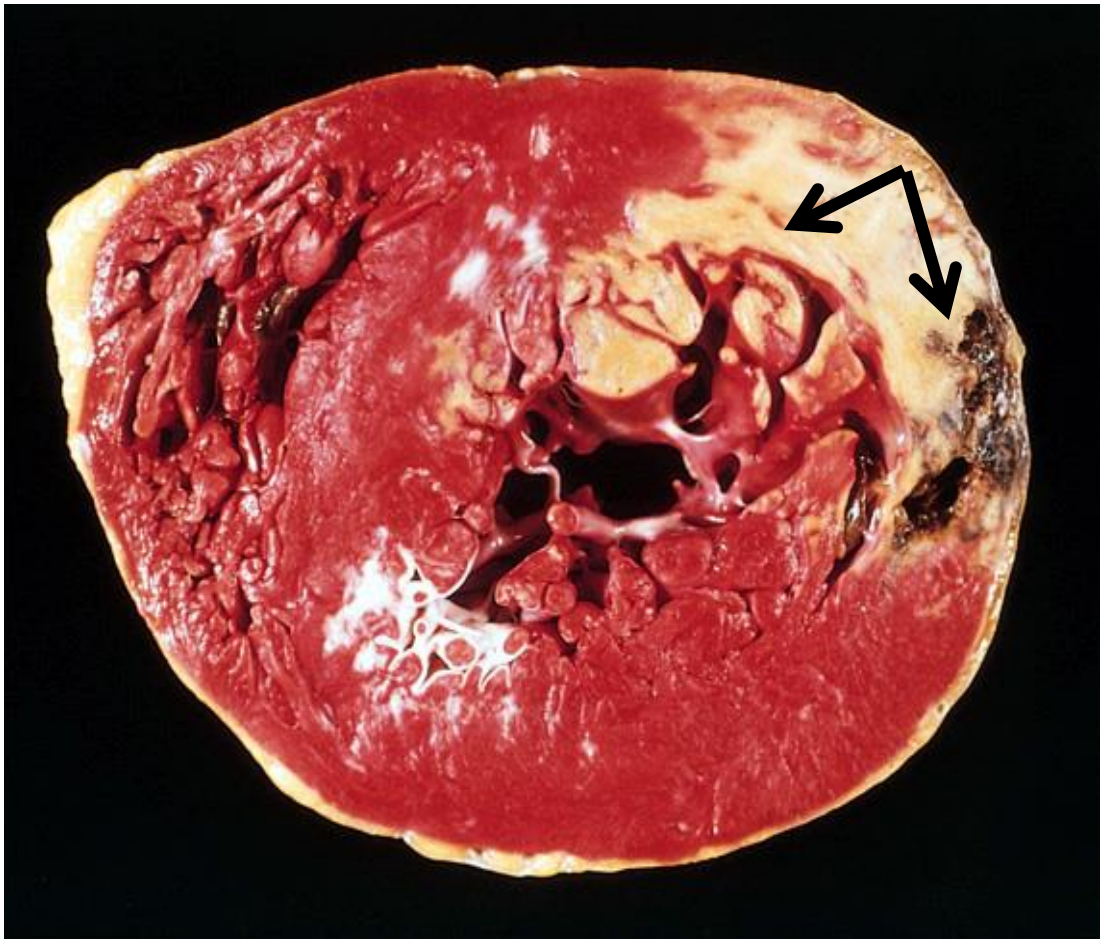


- Black arrows show soft hemorrhagic areas, consistent with MI.
- Part of the heart which is affected: Left ventricle.

Complications that might occur in MI:

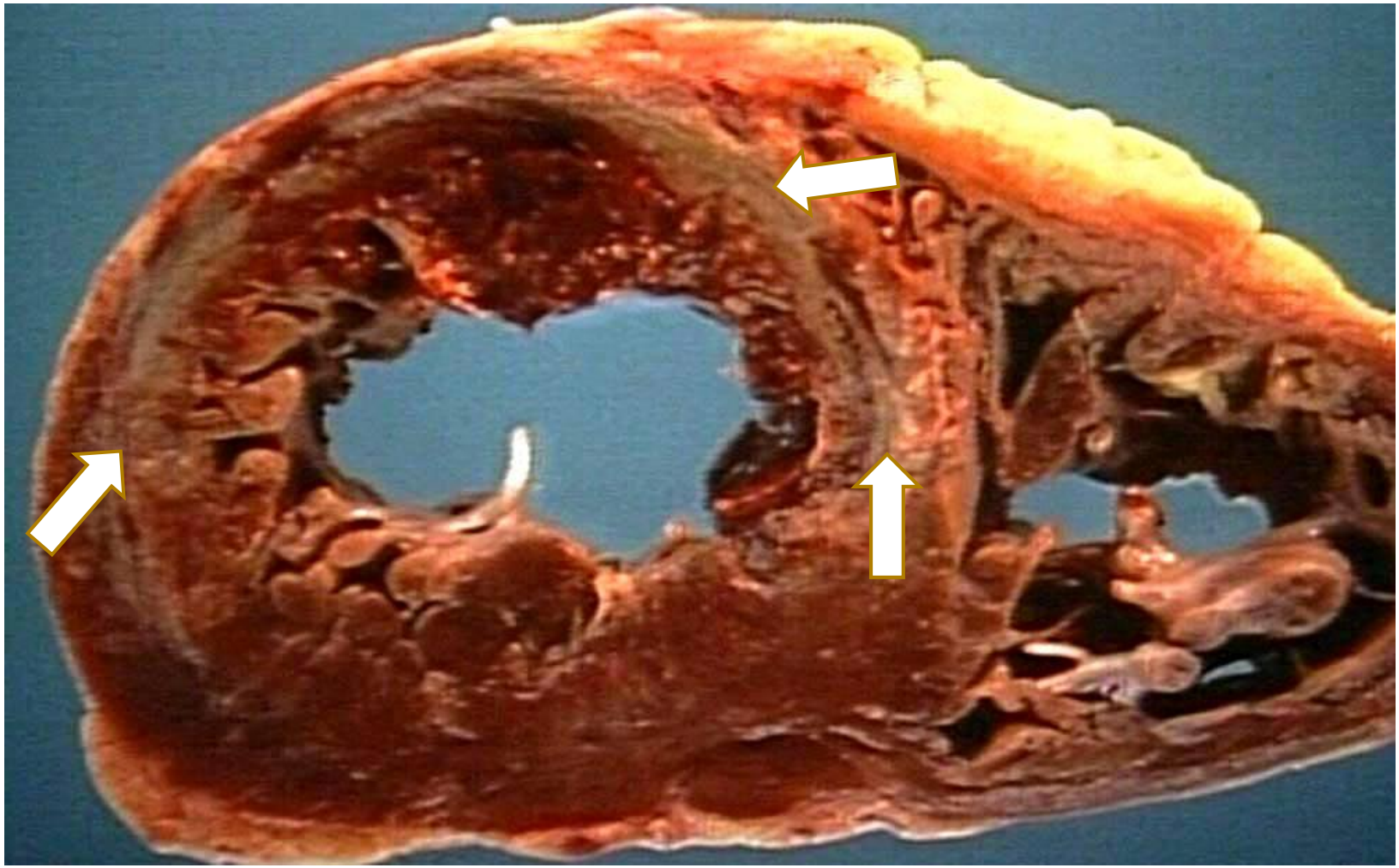
- Heart Failure.
- Myocardial rupture (3 days).
- Ventricular aneurysm (7 days).

Myocardial Infarction - CS



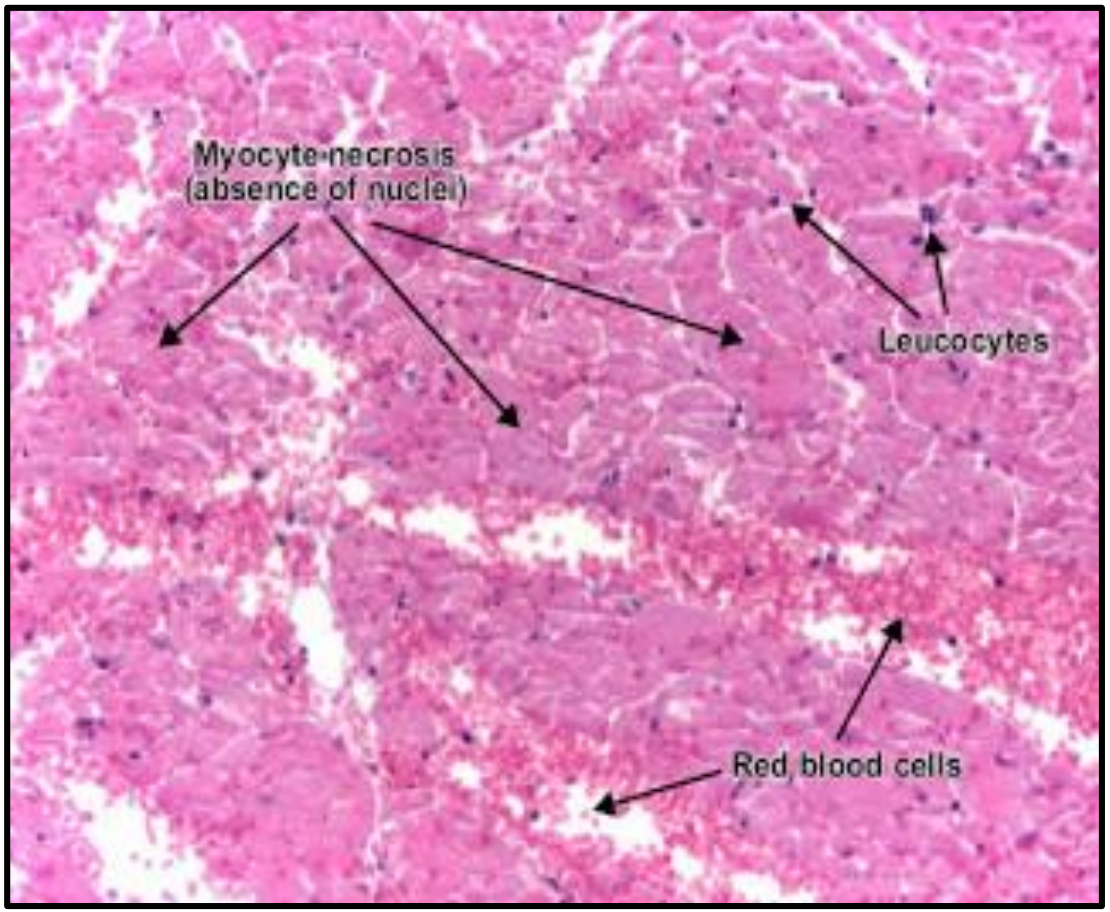
Cross section of the left and right ventricles shows a pale and irregular focal fibrosis in the left ventricular wall with increased thickness .

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 - LPF



Enzymes elevated:

a. Troponin

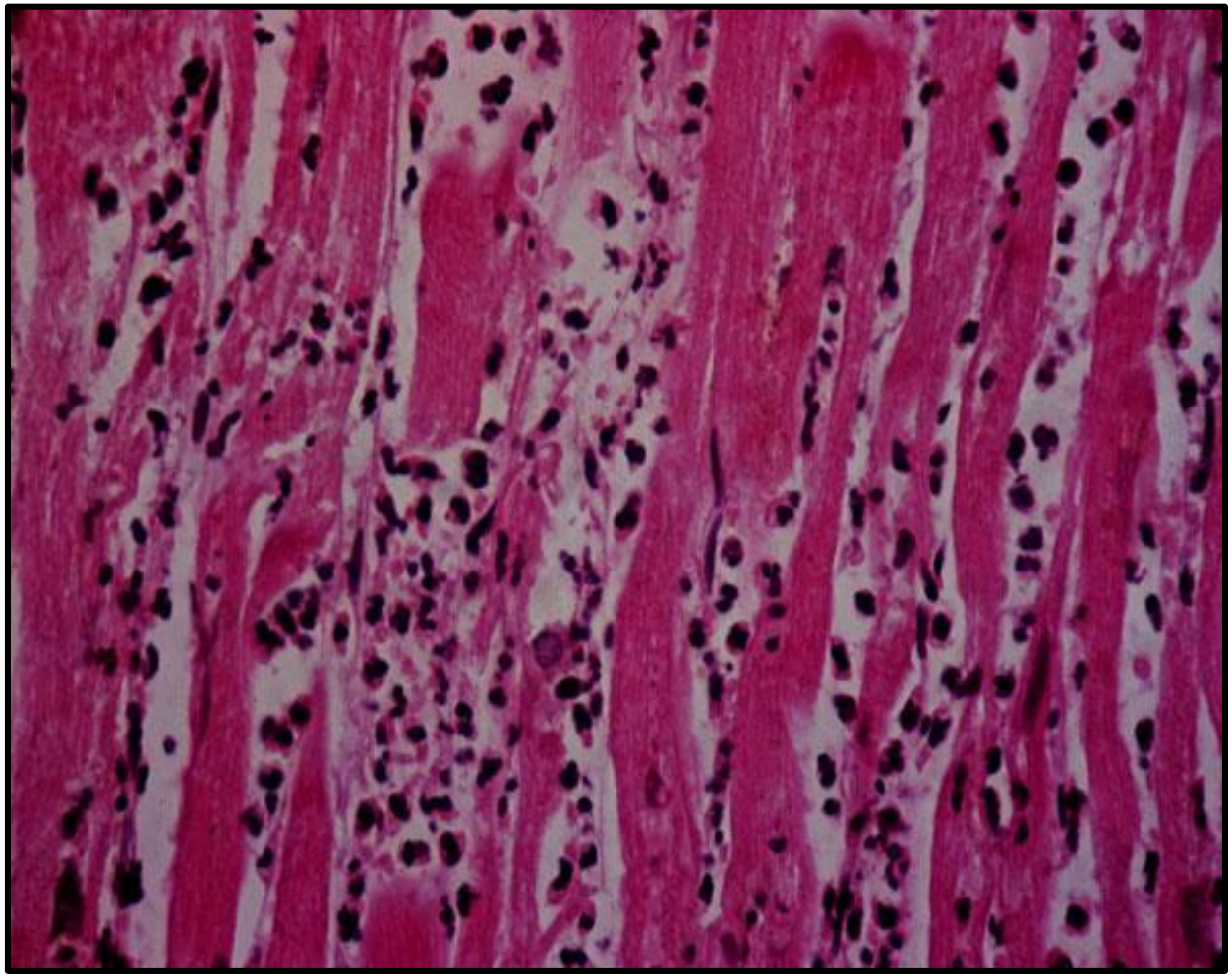
b. CK-MB (Creatine Kinase).

c. LDH (Lactic dehydrogenase).

d. Myoglobin.

***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.*

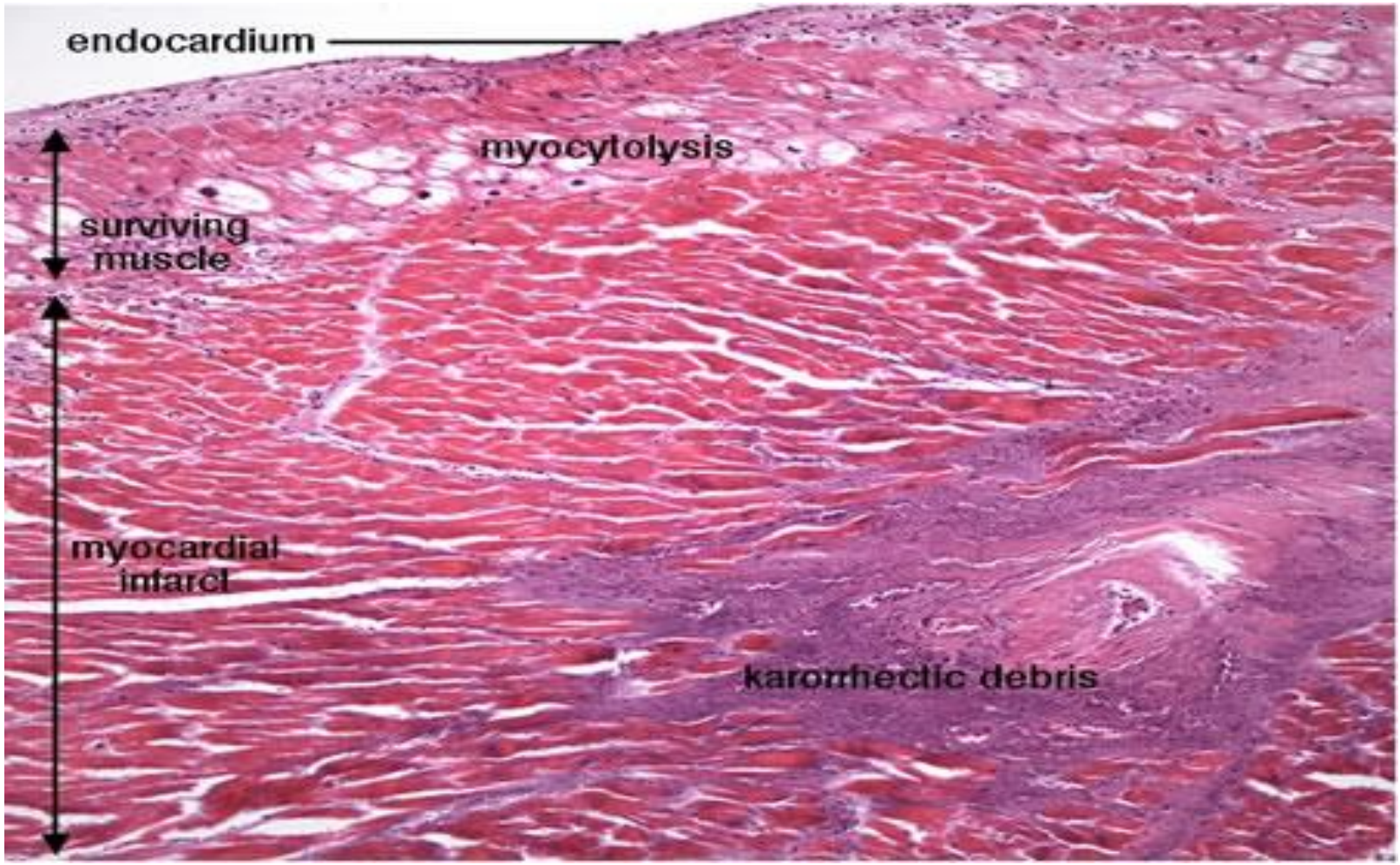
Myocardial Infarction 2-3 days - HPF



Occlusion of *Left anterior descending coronary artery* is responsible for 40-50 % of MI.

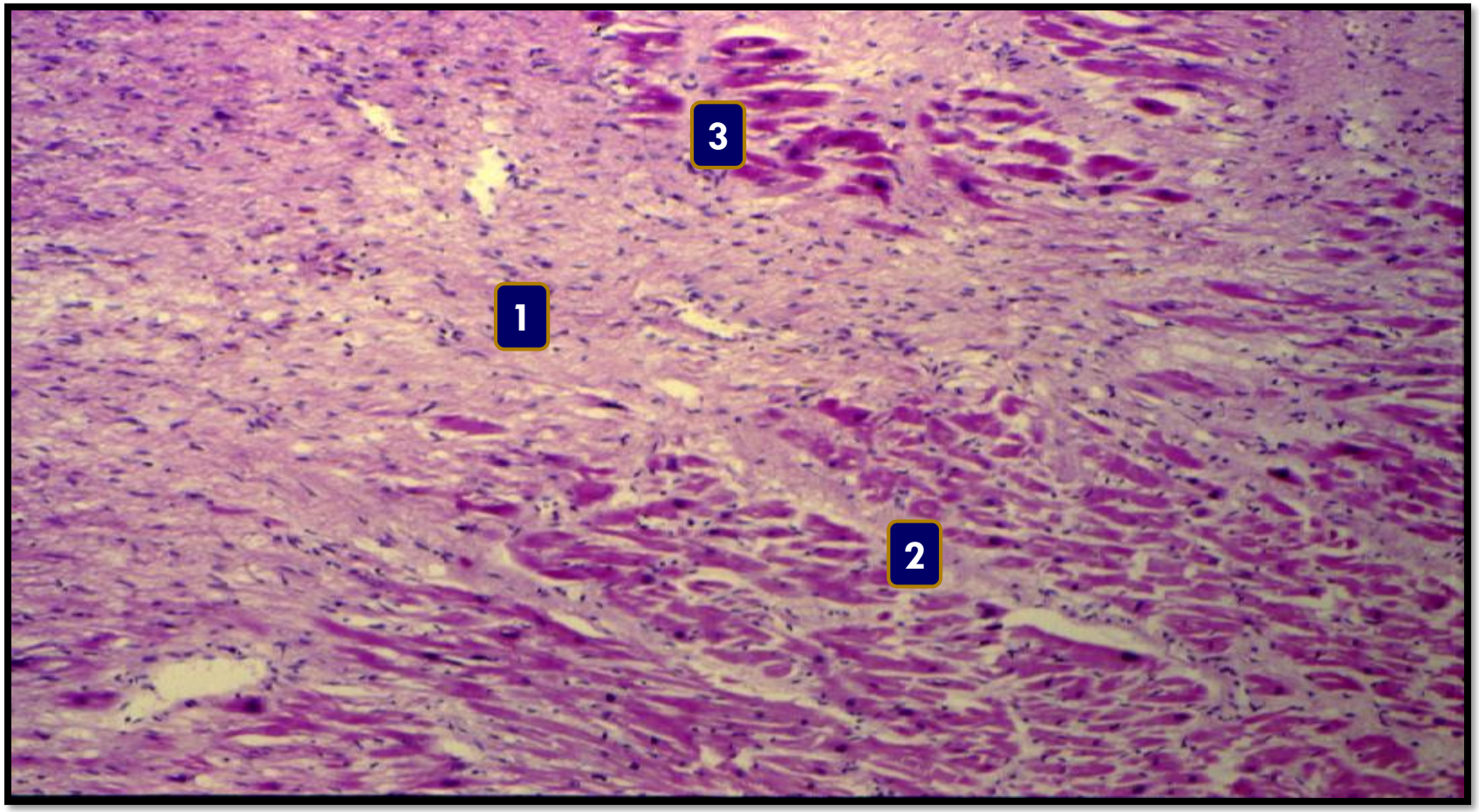
Acute myocardial infarct*, histology. This *3-4 day* old infarct shows *necrosis of myocardial cells* and is infiltrated with *polymorphnuclear leukocytes.

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 .*

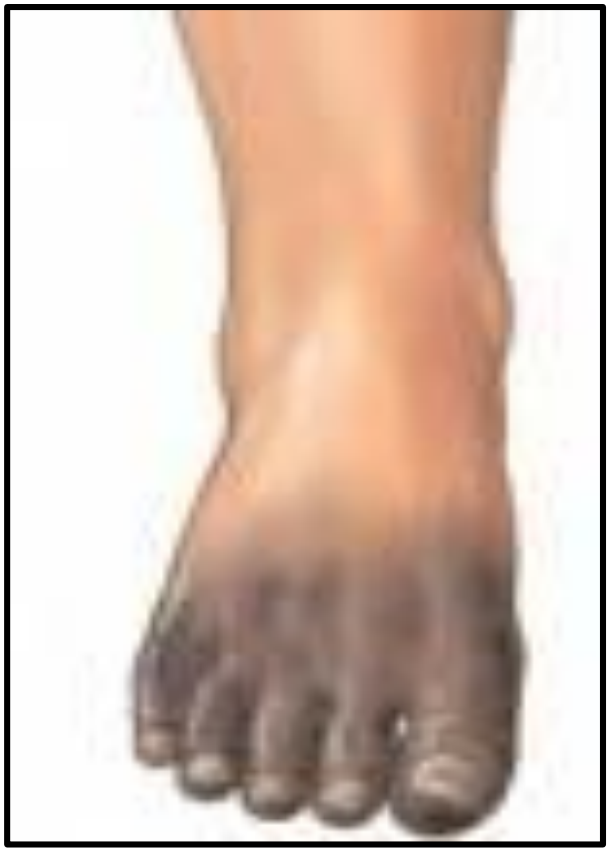
3- ***The remaining myocardial fibers*** *show enlarged nuclei due to ventricular hypertrophy .*

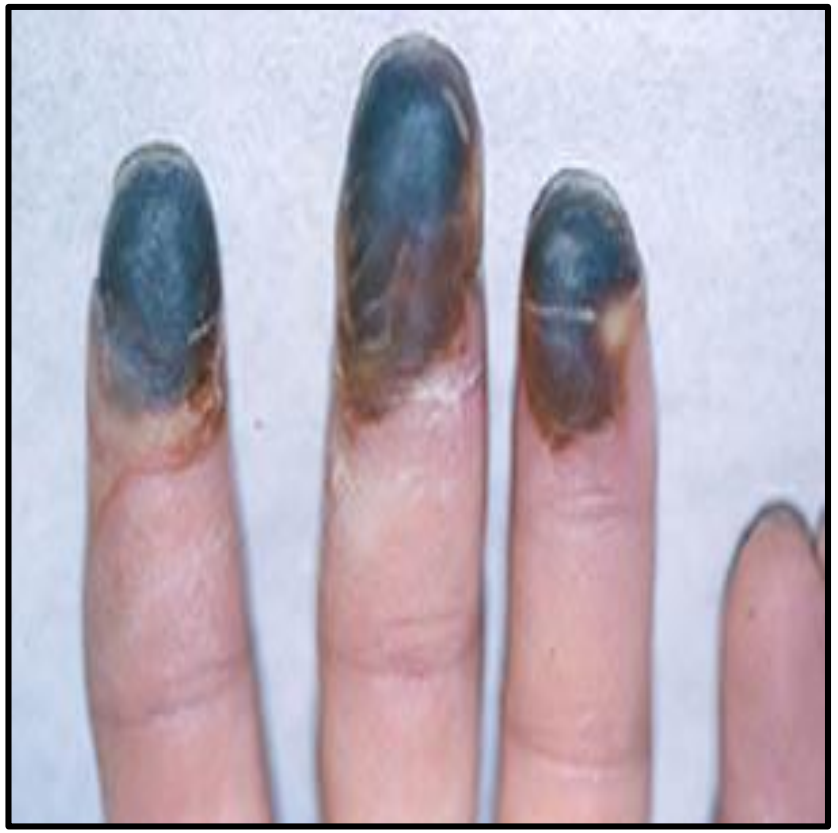
THROMBOEMBOLISM / VASCULITIS

Thromboangitis oblitrans (Buerger's disease)

Boehringer Ingelheim

Thromboangitis oblitrans (Buerger`s disease)



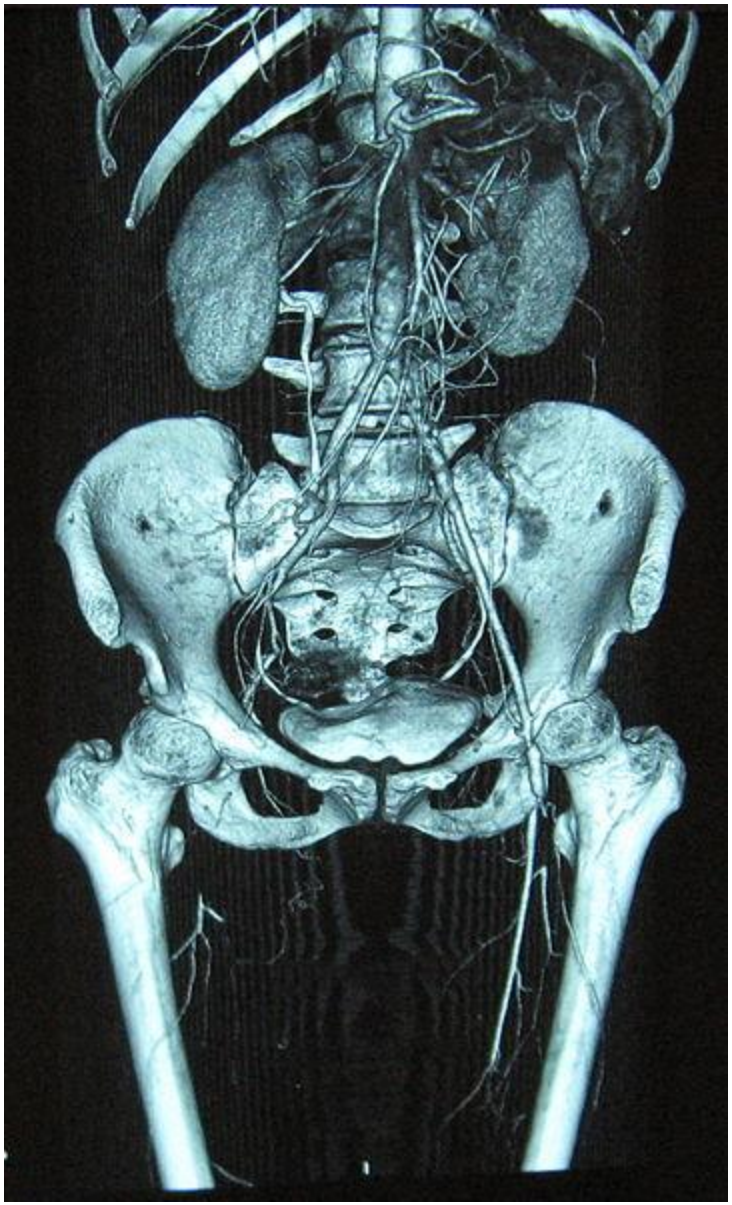


- Black discoloration of the skin possibly caused by ischemia.
- Etiology is hypersensitivity to tobacco products.

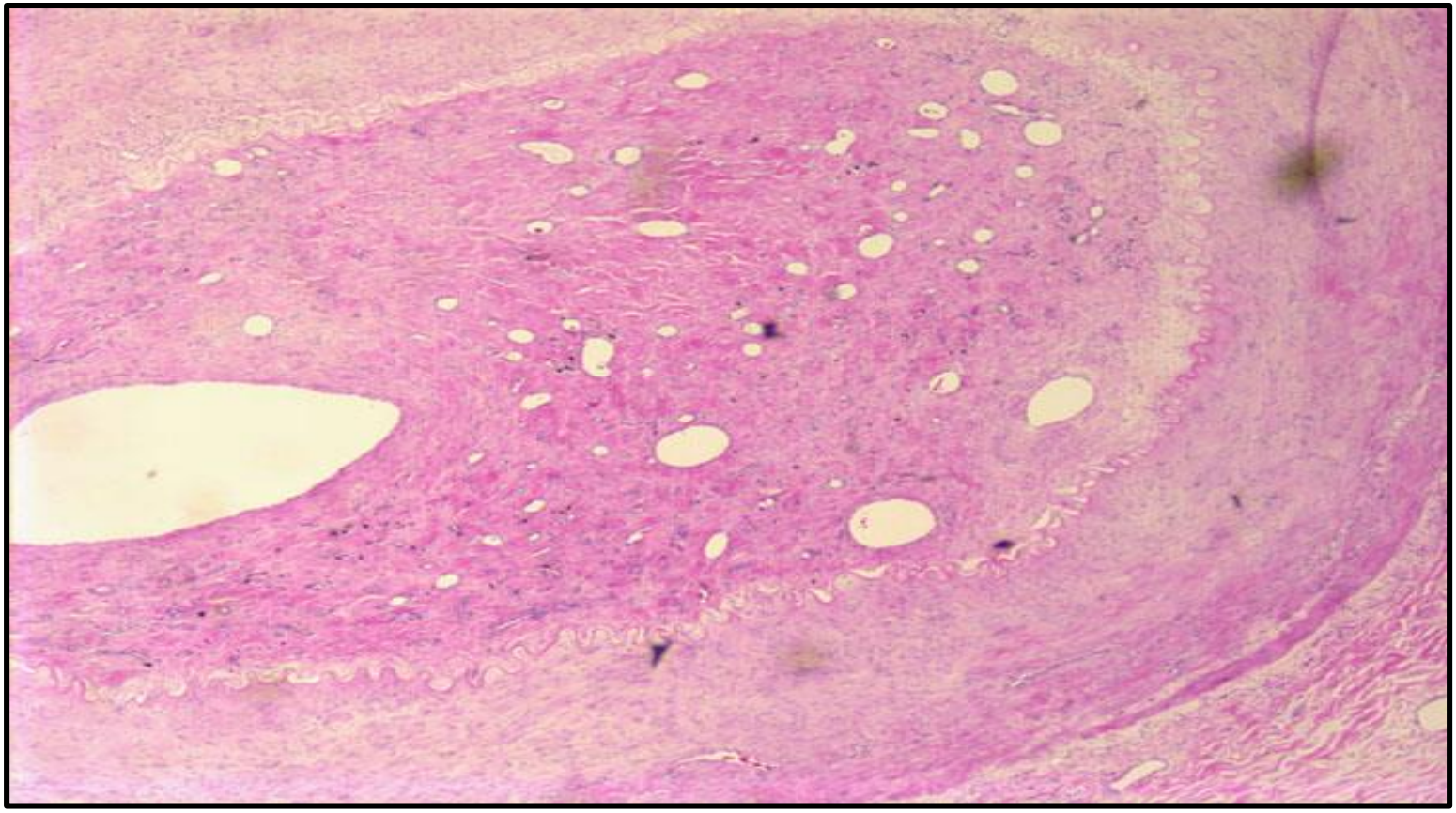
THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE)

- *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*



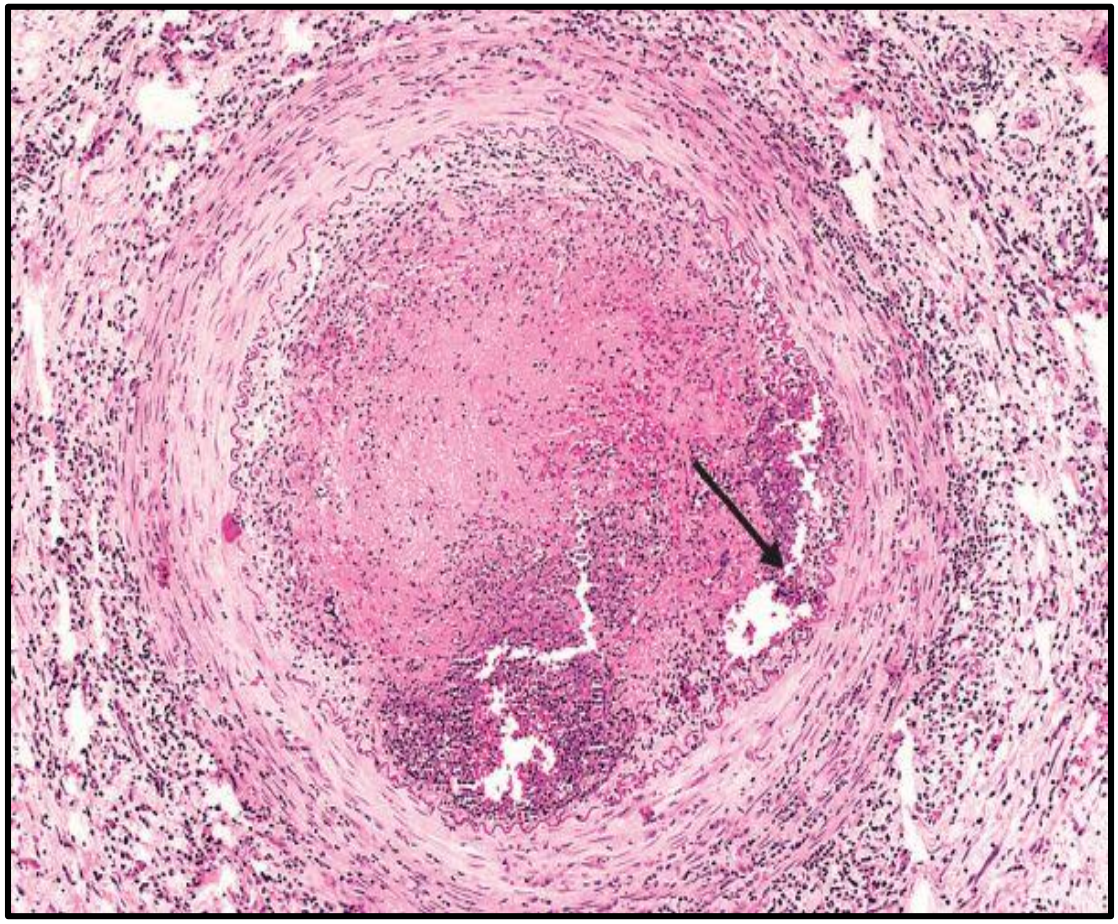
THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE) - LPF



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.

Wegener's granulomatosis (Granulomatous polyangitis) is a **destructive vasculitis characterized by the formation of ill-defined granulomas in vessel wall together with common renal and lung involvement.**

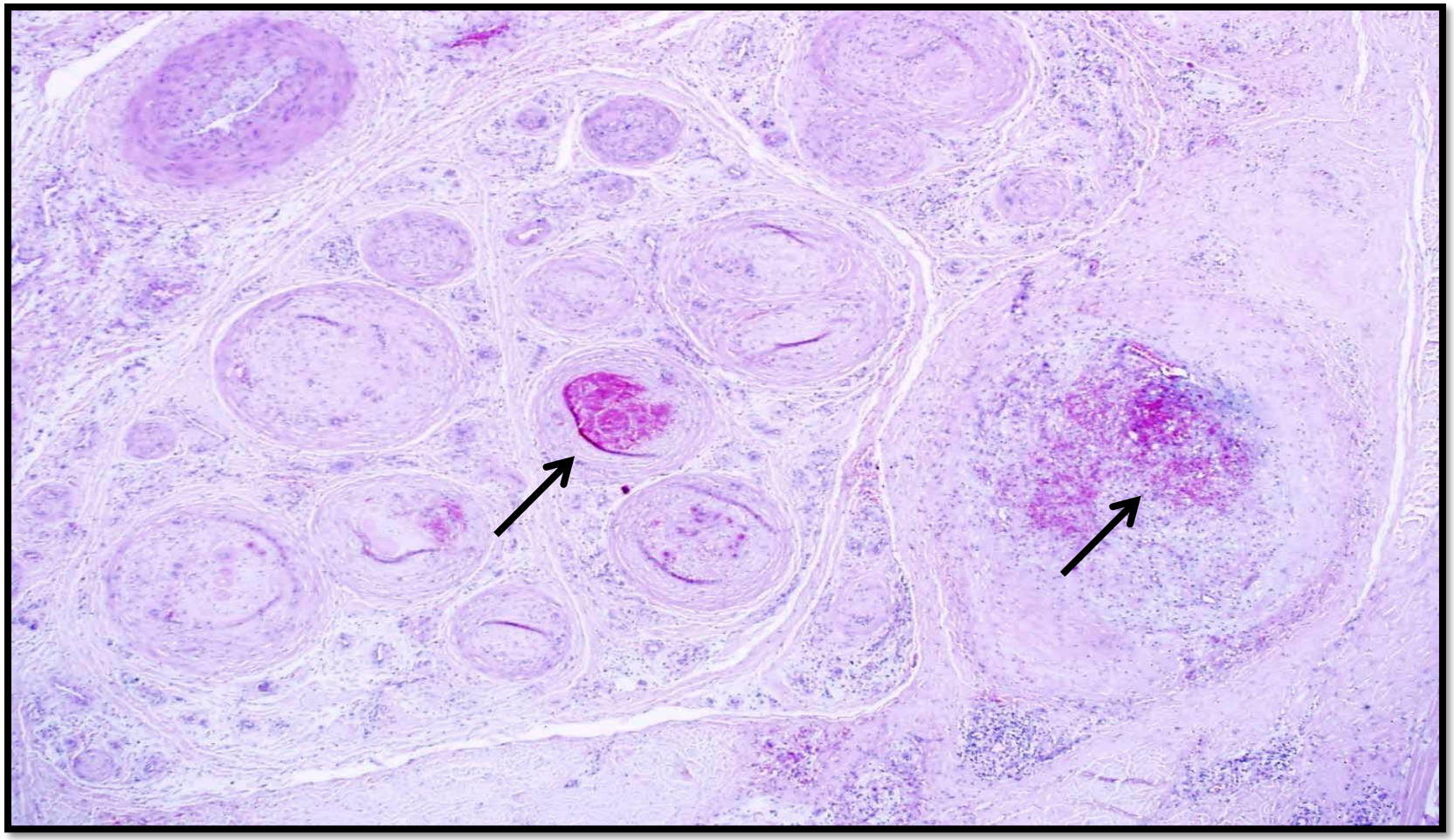
THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE) - HPF



a. *Thrombus.*
b. *Microabscesses within the thrombus.*
c. *Vasculitis (infiltration of the vessel wall by neutrophils).*

Sometimes nerves are also involved giving rise to pain in affected areas.

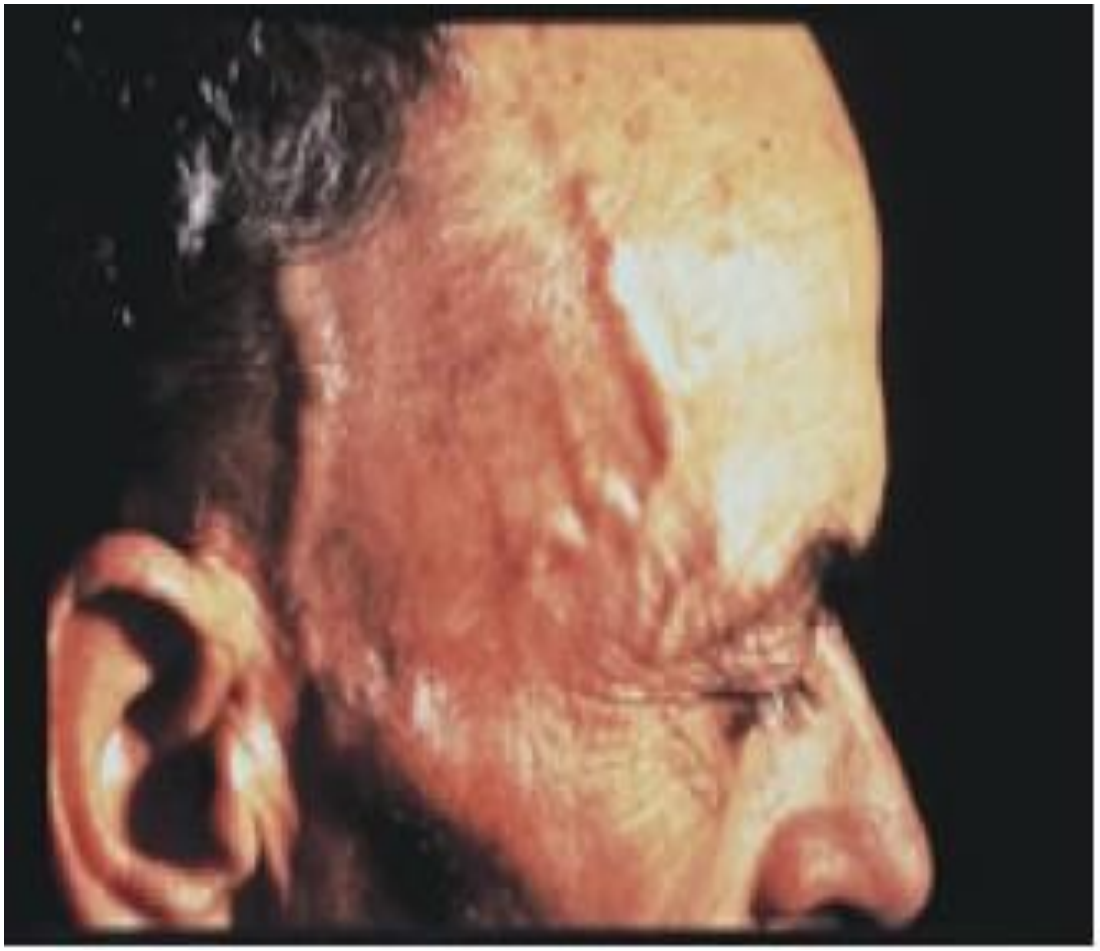
THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE)- HPF



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

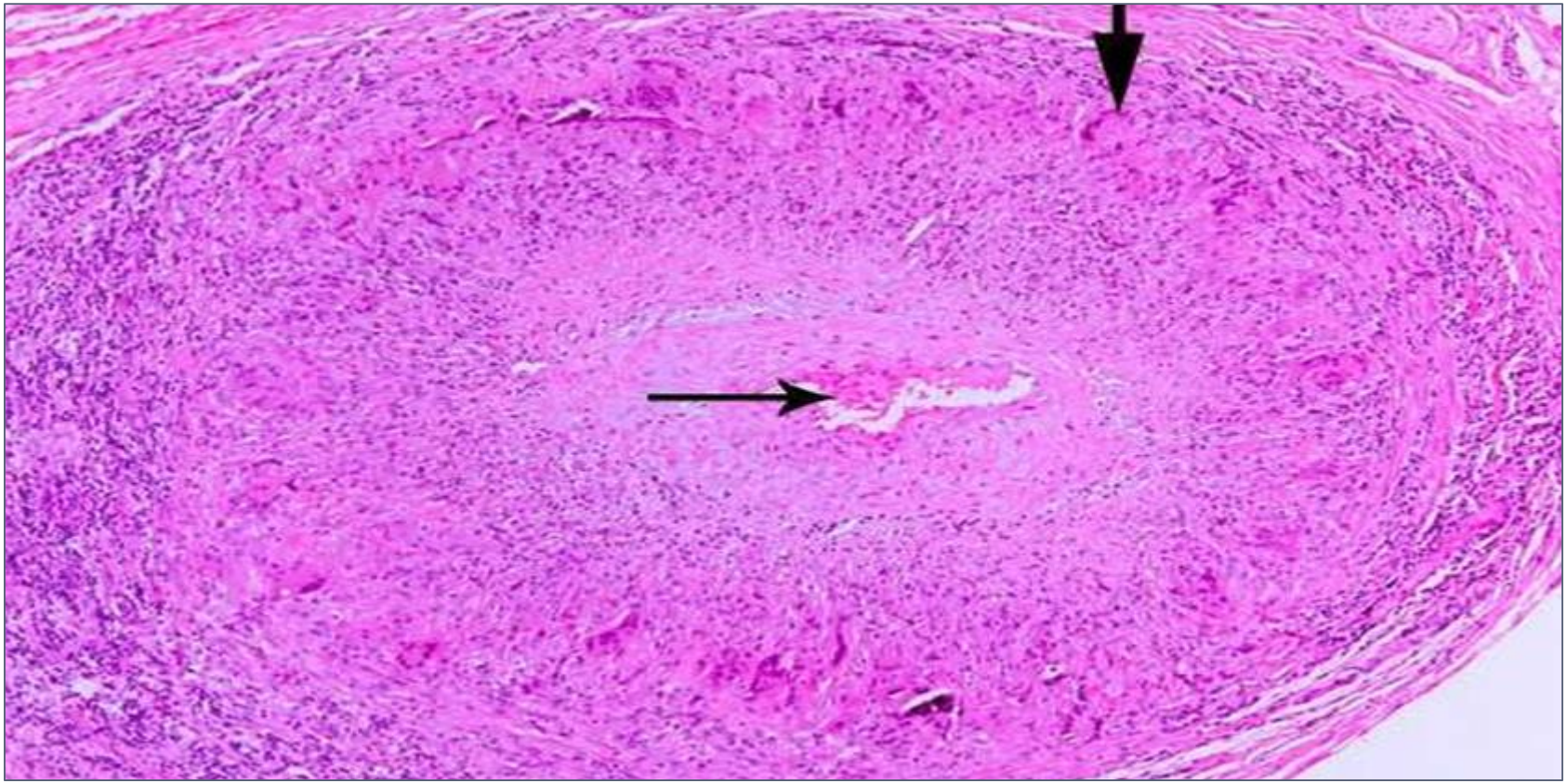
GIANT CELL (TEMPORAL) ARTERITIS

GIANT CELL / TEMPORAL ARTERITIS



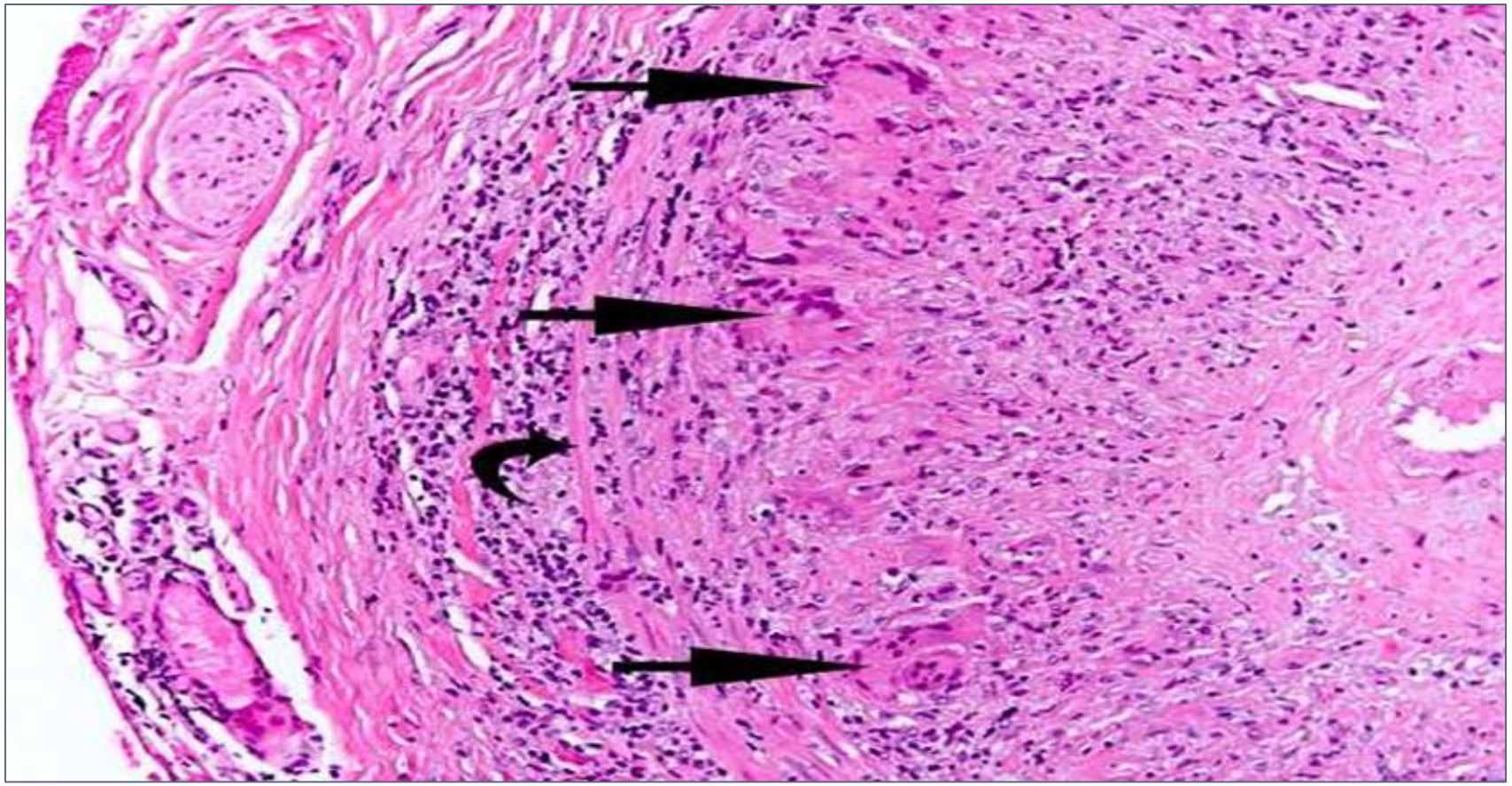
Tender and thickened scalp veins

GIANT CELL / TEMPORAL ARTERITIS - LPF



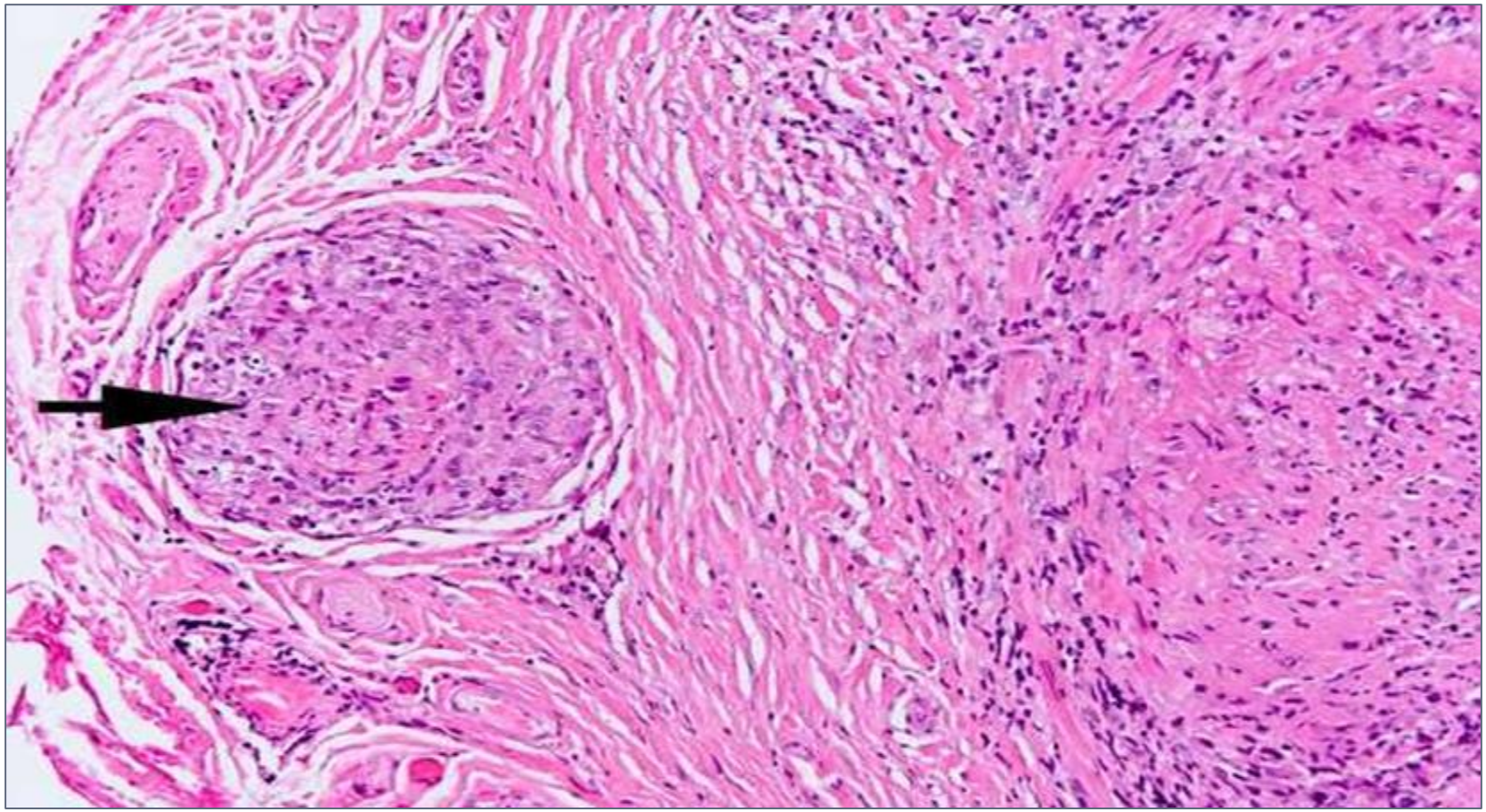
Circumferential involvement of the vascular media is present (vertical arrow pointing downward). Also note the presence of chronic lymphocytic inflammation in the media and adventitia. Reactive intimal fibroplasias lead to luminal stenosis with <10% of its original luminal diameter (thin arrow in the center).

GIANT CELL / TEMPORAL ARTERITIS - HPF



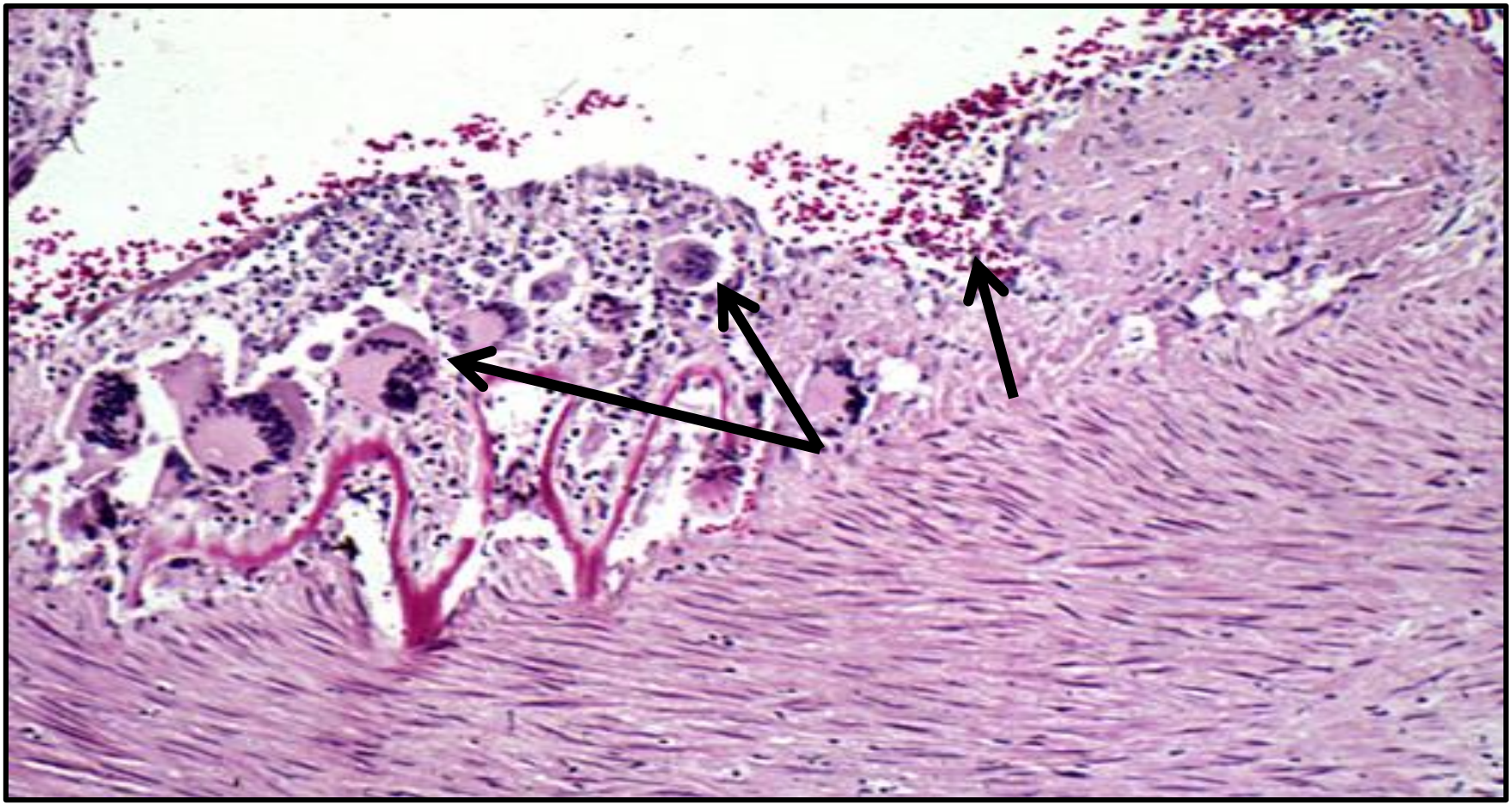
Giant cells can be of Langhans type or foreign-body type (three arrows) and may show fragments of disrupted internal elastic lamina. Note the presence of dense chronic lymphocytic inflammation traversing through circumferential smooth muscle fibers (curved arrow) of vascular media.

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



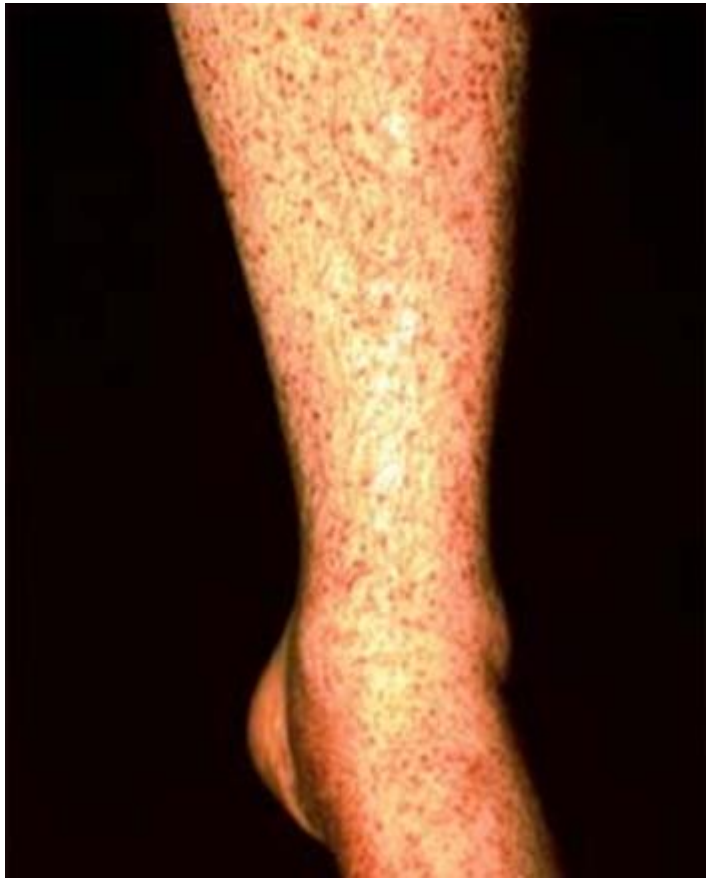
Disruptions of the elastic lamina with inflammation and giant cells.

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

LEUKOCYTOCLASTIC / HYPERSENSITIVITY VASCULITIS

(MICROSCOPIC POLYANGITIS)

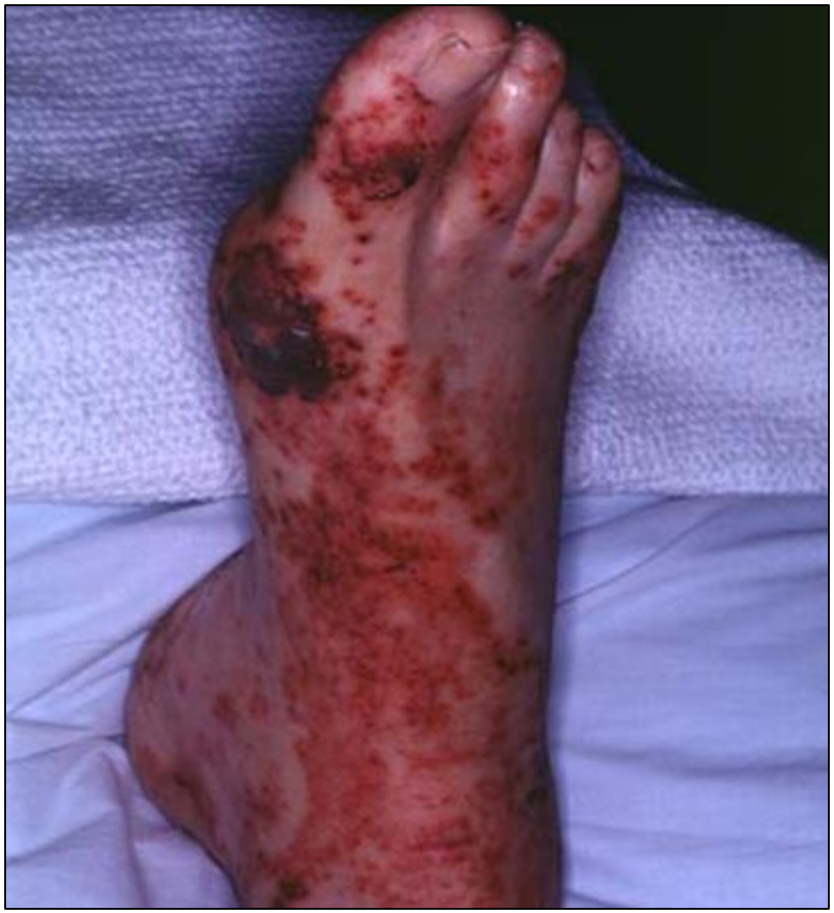
Hypersensitivity vasculitis – Clinical sign



Vasculitis is secondary to deposition of immunoglobulins and complement in the vessel wall.

Hypersensitivity vasculitis might be complicated with necrotizing glomerulonephritis (Hematuria) and hemoptysis due to pulmonary capillaritis

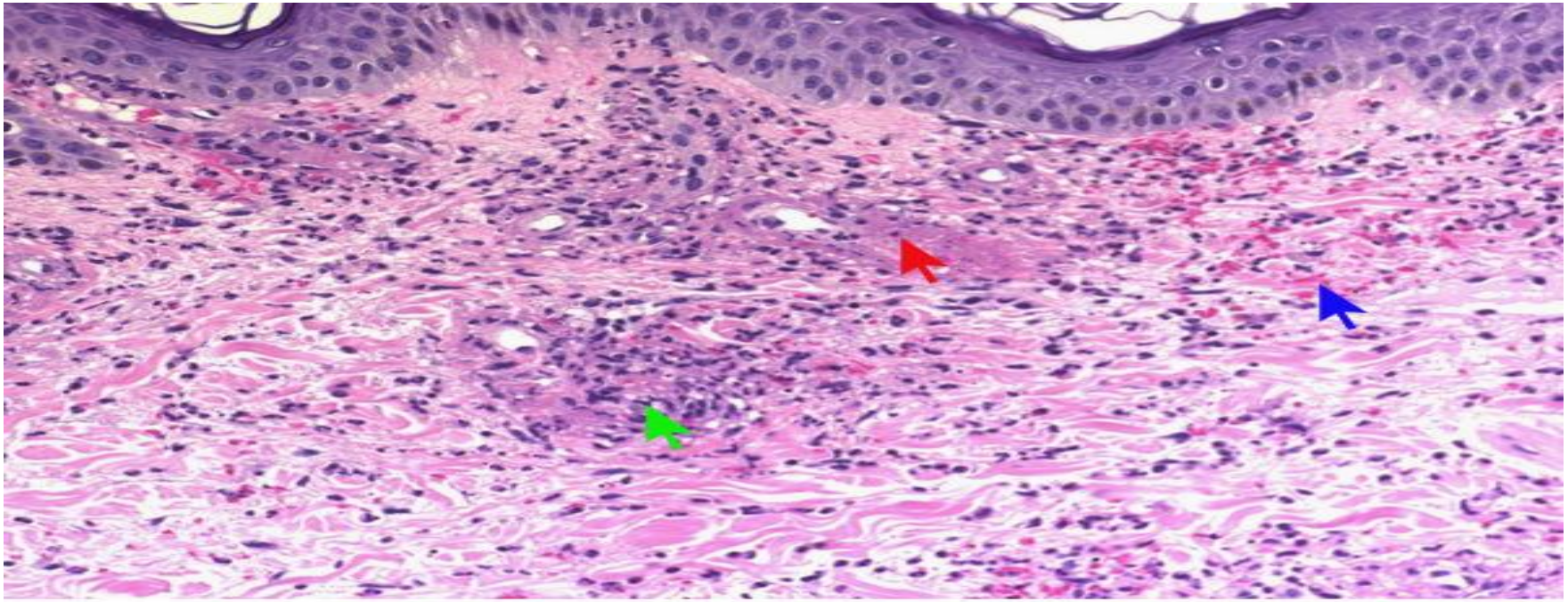
Leukocytoclastic vasculitis - Clinical sign



The purpuric patches because of Subcutaneous bleeding

Most pronounced in dependent areas.

Leukocytoclastic vasculitis - HPF

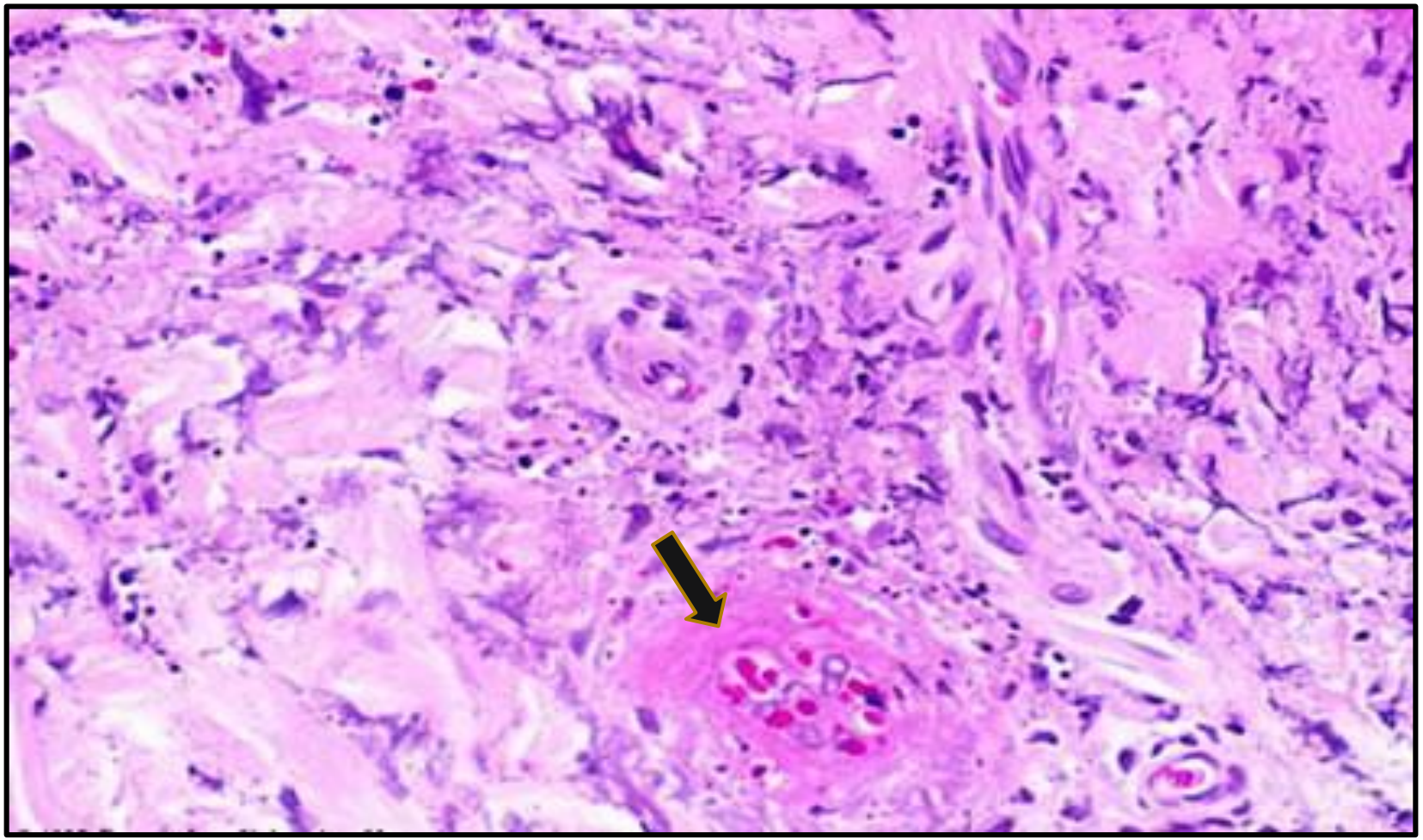


Fibrinoid type necrosis

Red cell extravasation

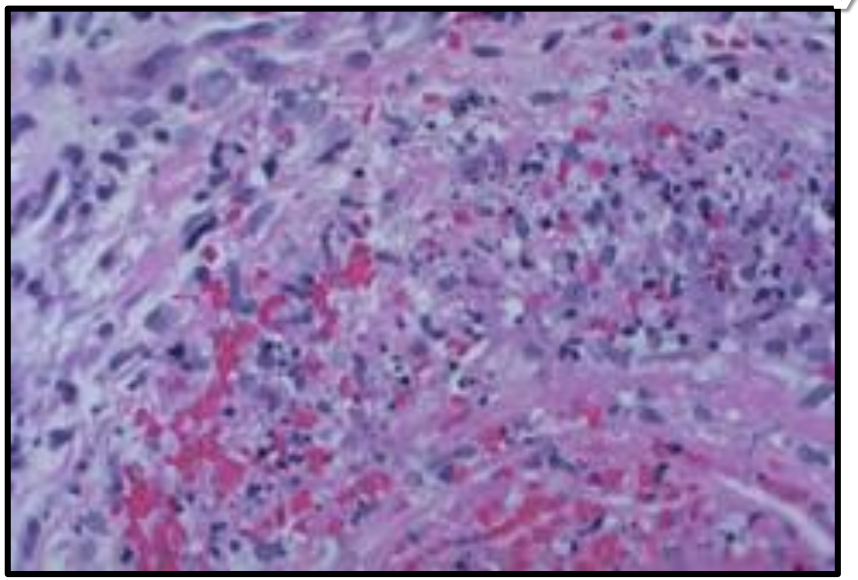
Inflammation

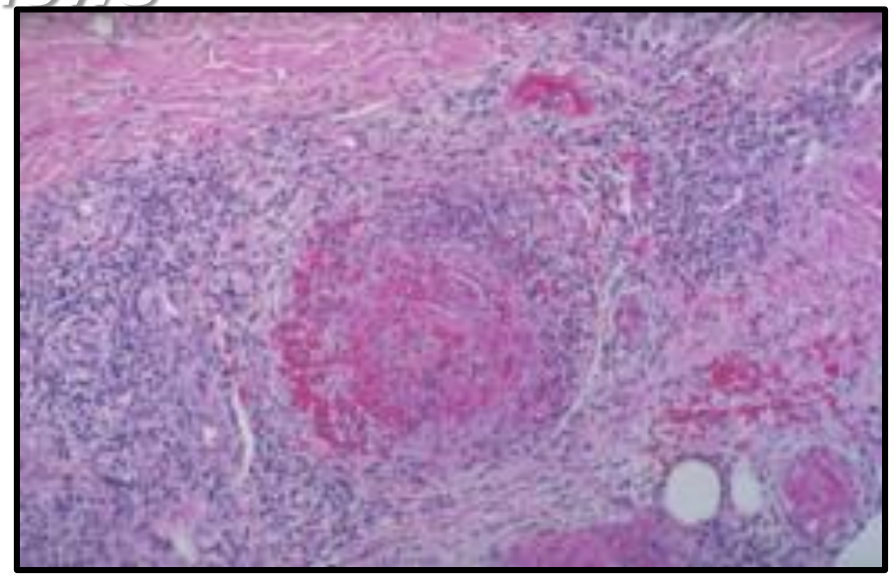
Leukocytoclastic vasculitis - HPF

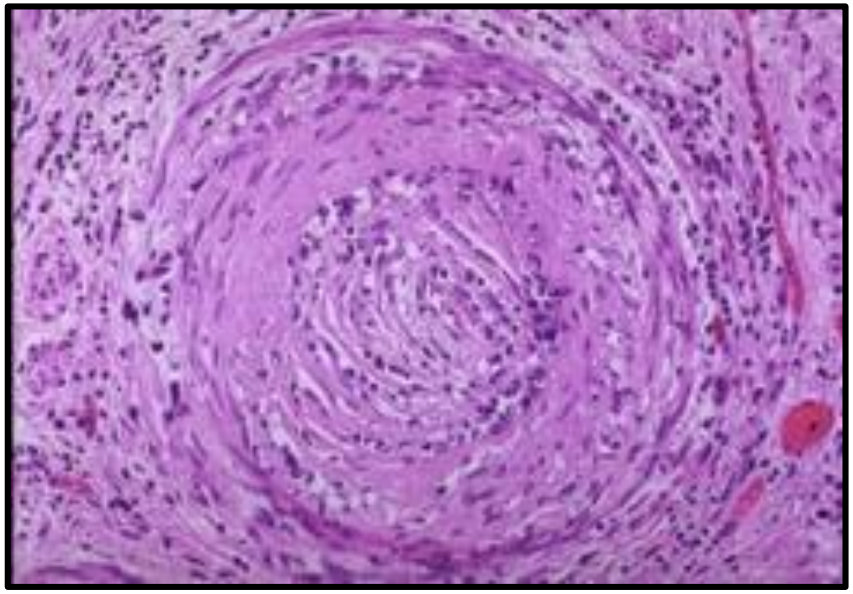


- Fibrinoid necrosis of small dermal vessels is present, necessary to establish the diagnosis of leukocytoclastic vasculitis (Black arrow).
- Surrounding tissues is showing nuclear debris and neutrophils.

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