CARDIOVASCULAR SYSTEM

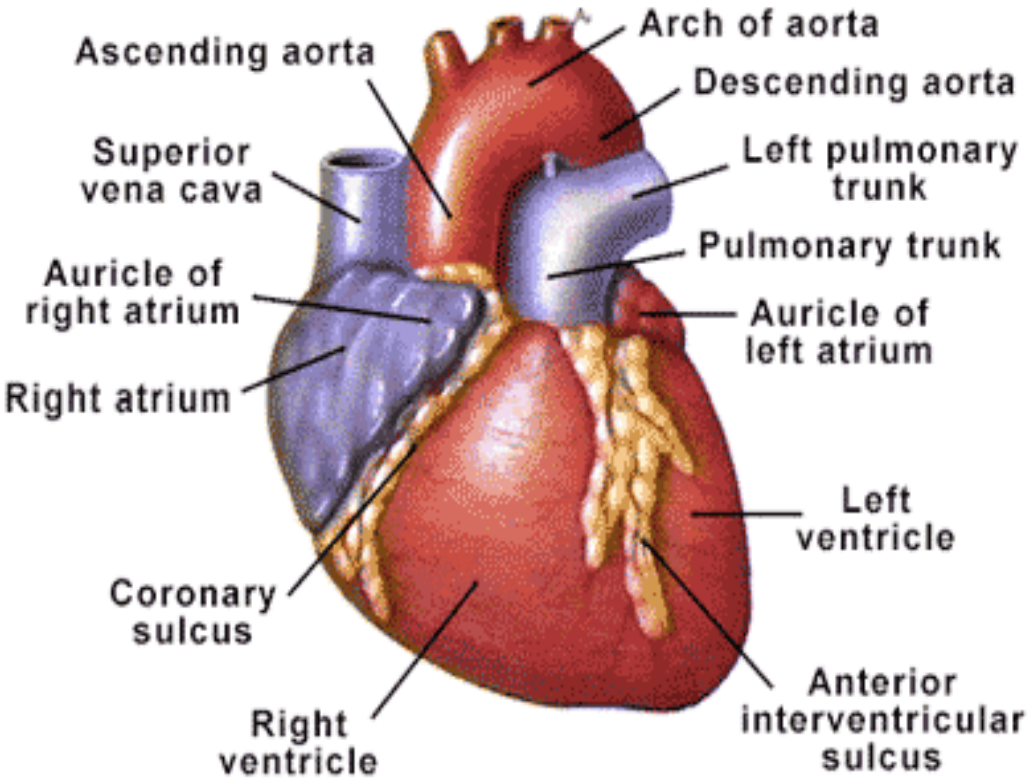
Pathology Practical

Prepared by:

- *Prof. Ammar Al Rikabi*
- *Dr. Sayed Al Esawy*

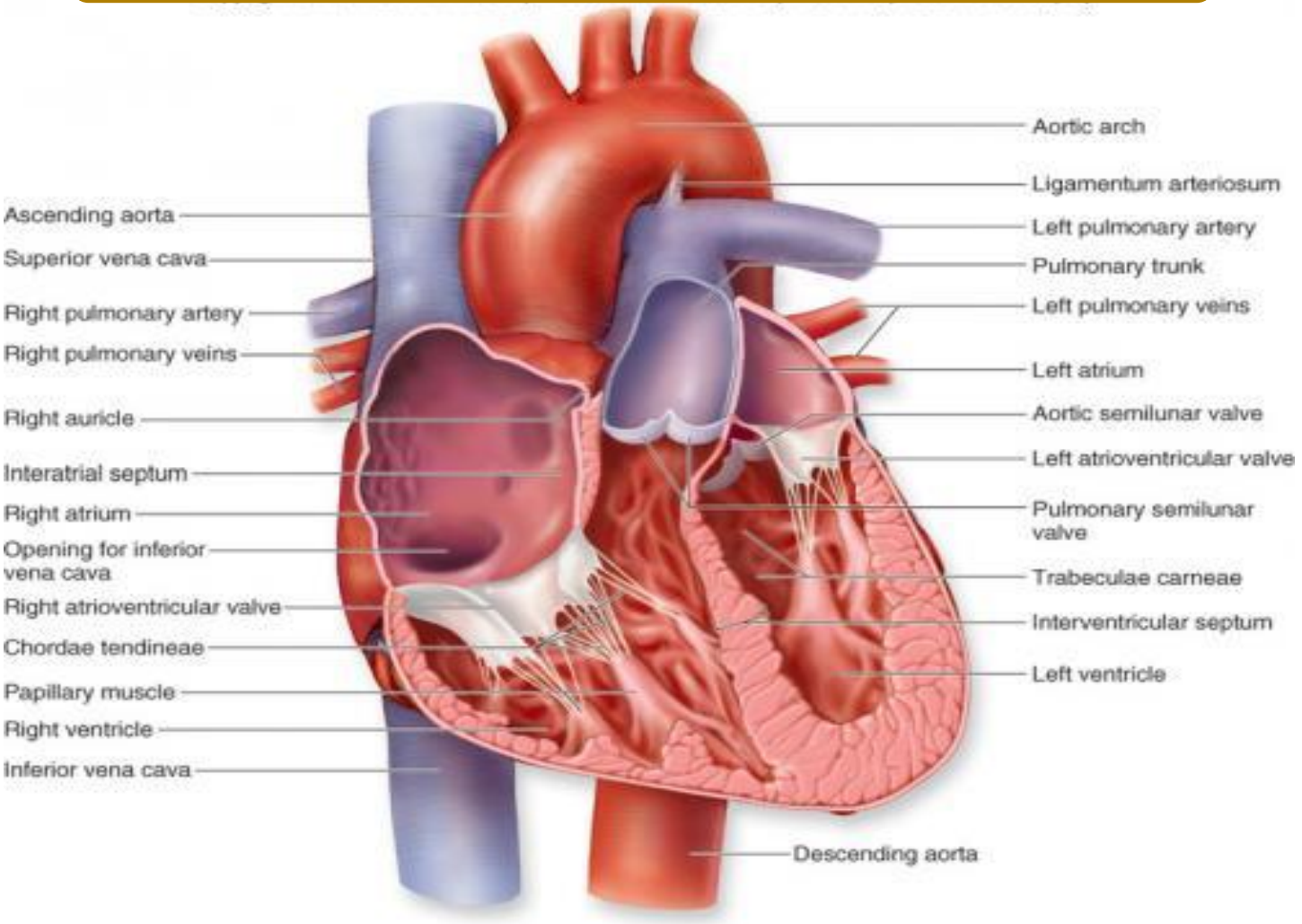
NORMAL ANATOMY AND HISTOLOGY

Anatomy of the Heart



- The heart serves as a **mechanical pump** to supply the entire body with blood, both providing nutrients and facilitate the excretion of 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

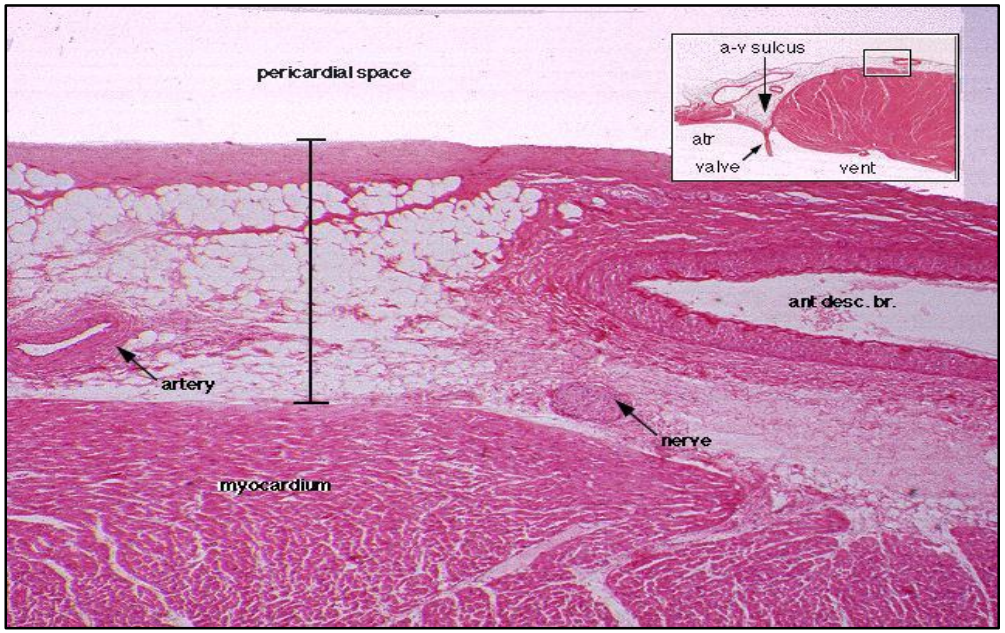


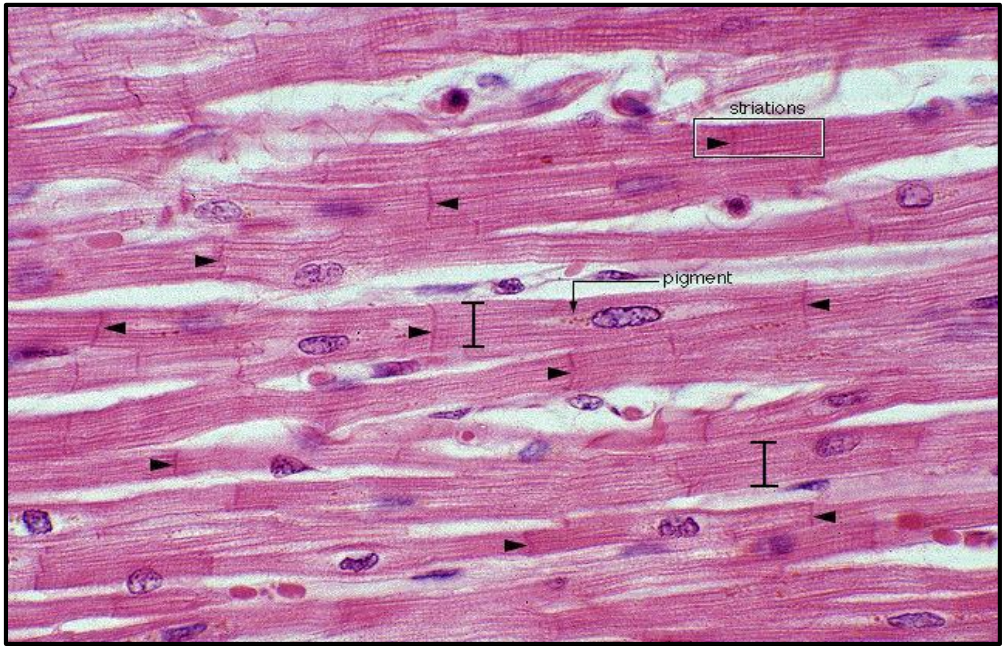
Histology of the Heart

- 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).

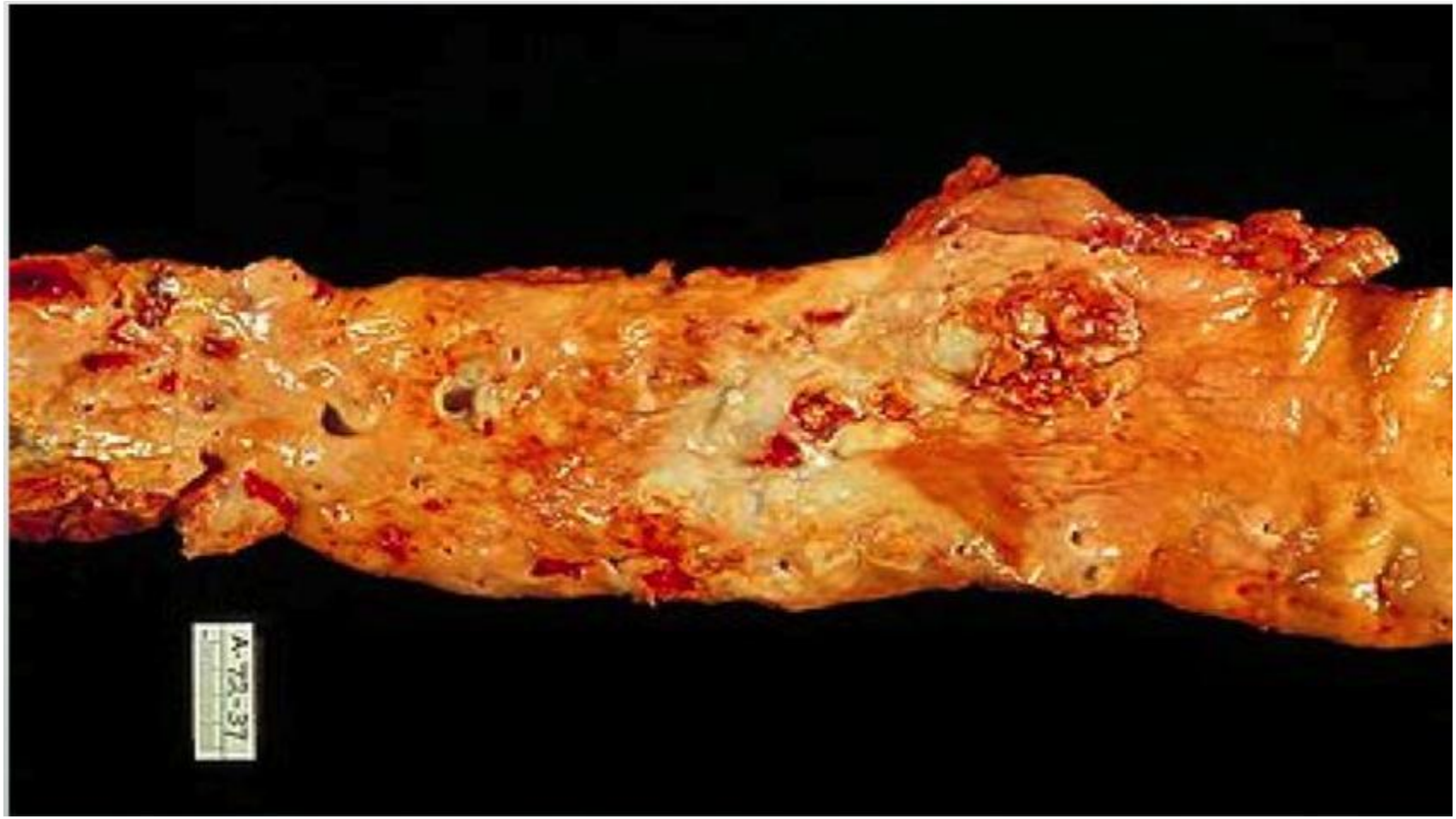




ATHEROMA OF THE AORTA

- *An **atheroma** is reversible accumulation and swelling in the inner 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.*
- *When the atheroma progress, the artery harden and thicken result in atherosclerosis*
- *The four major risk factors are hyperlipidemia, hypertension, cigarette smoking and diabetes .*

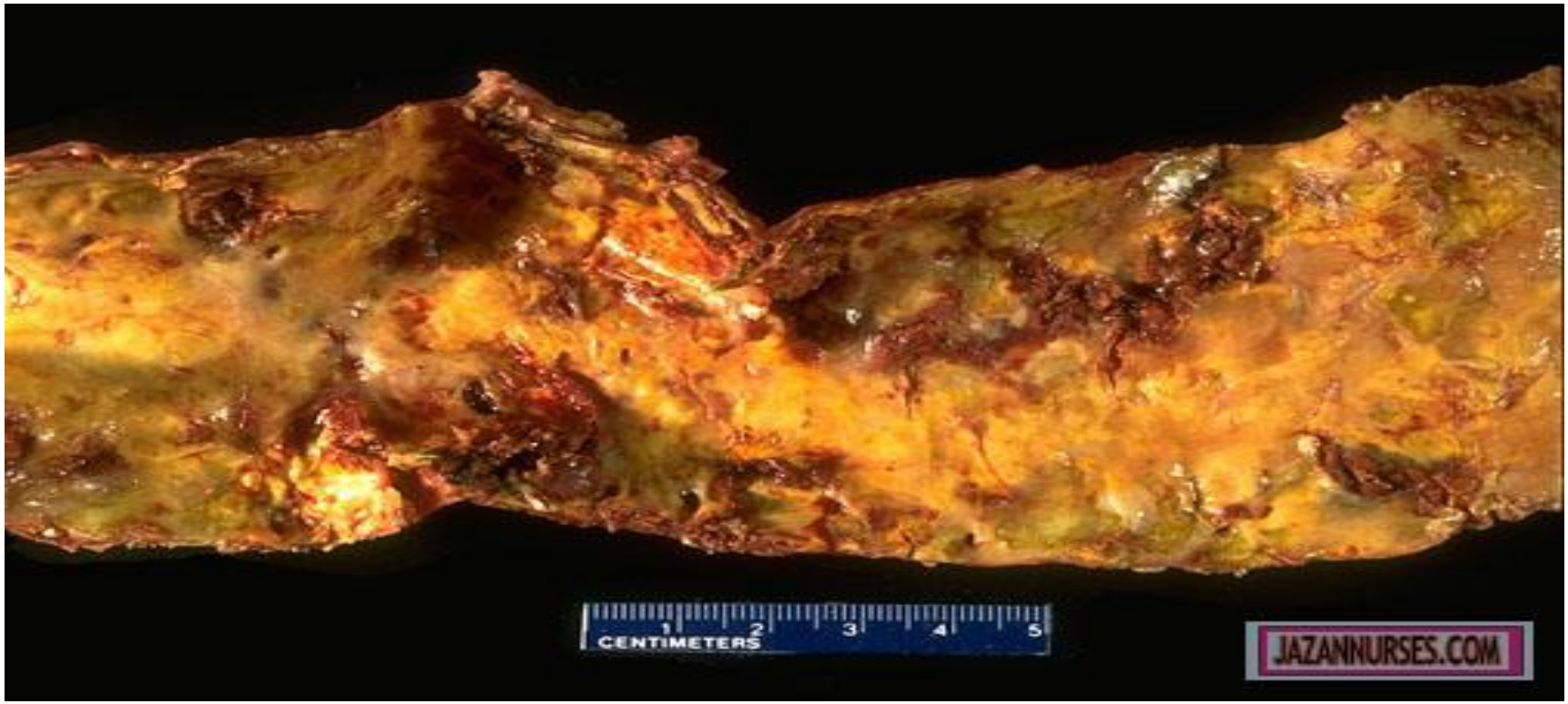
Atheroma of the Aorta - Gross



Yellow atheromatous plaque with area of ulceration and hemorrhage

The key processes in atherosclerosis are intimal thickening and lipid accumulation

Atheroma of the Aorta - Gross

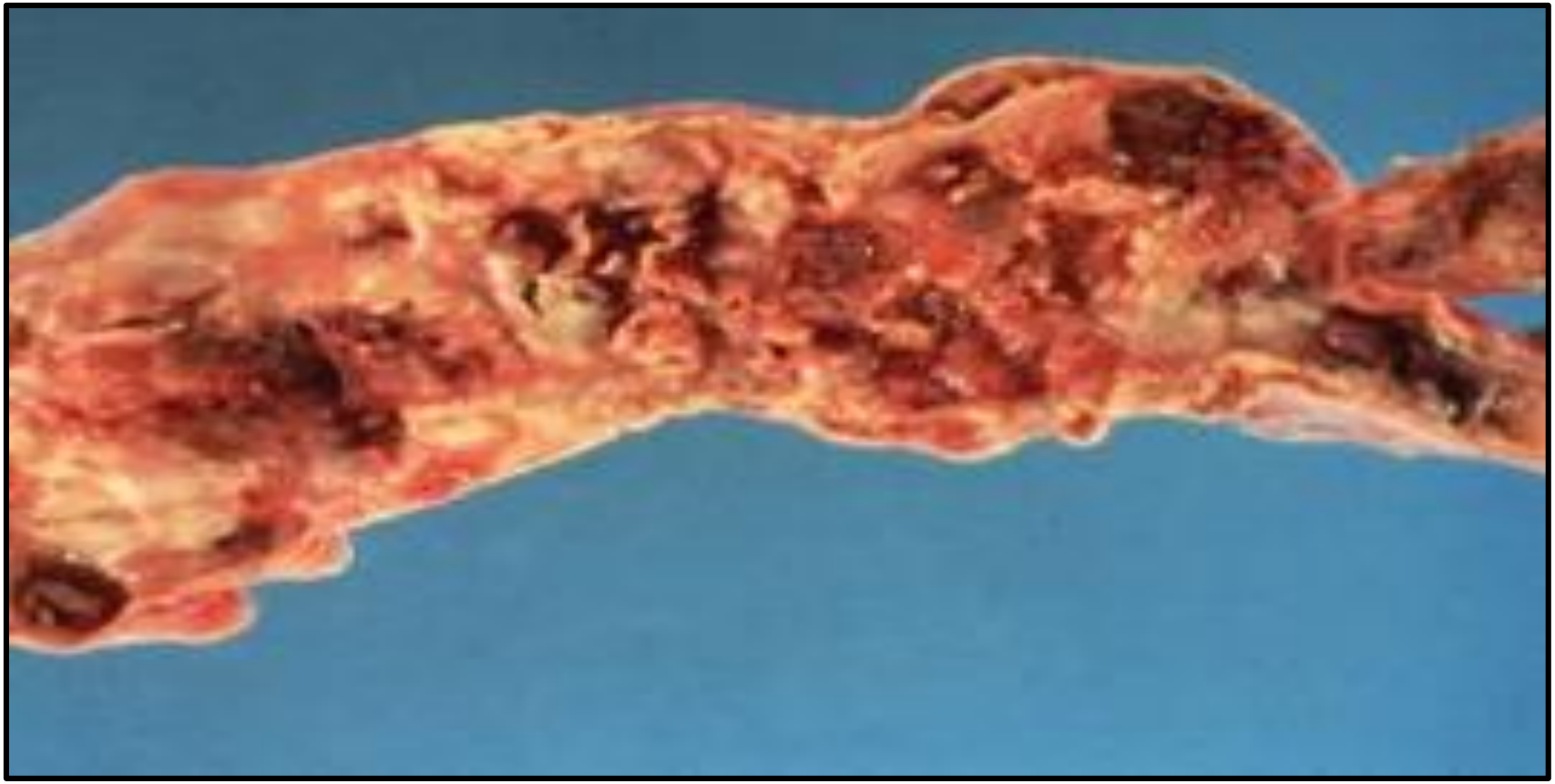


Severe atherosclerosis of the aorta : the atheromatous plaques have undergone ulceration along with formation of overlying mural thrombus.

Complications are

- Vascular thrombosis and distal embolization
- Aneurysm formation
- Cardiac ischemia and distal ischemic events .

Atheroma of the Aorta - Gross

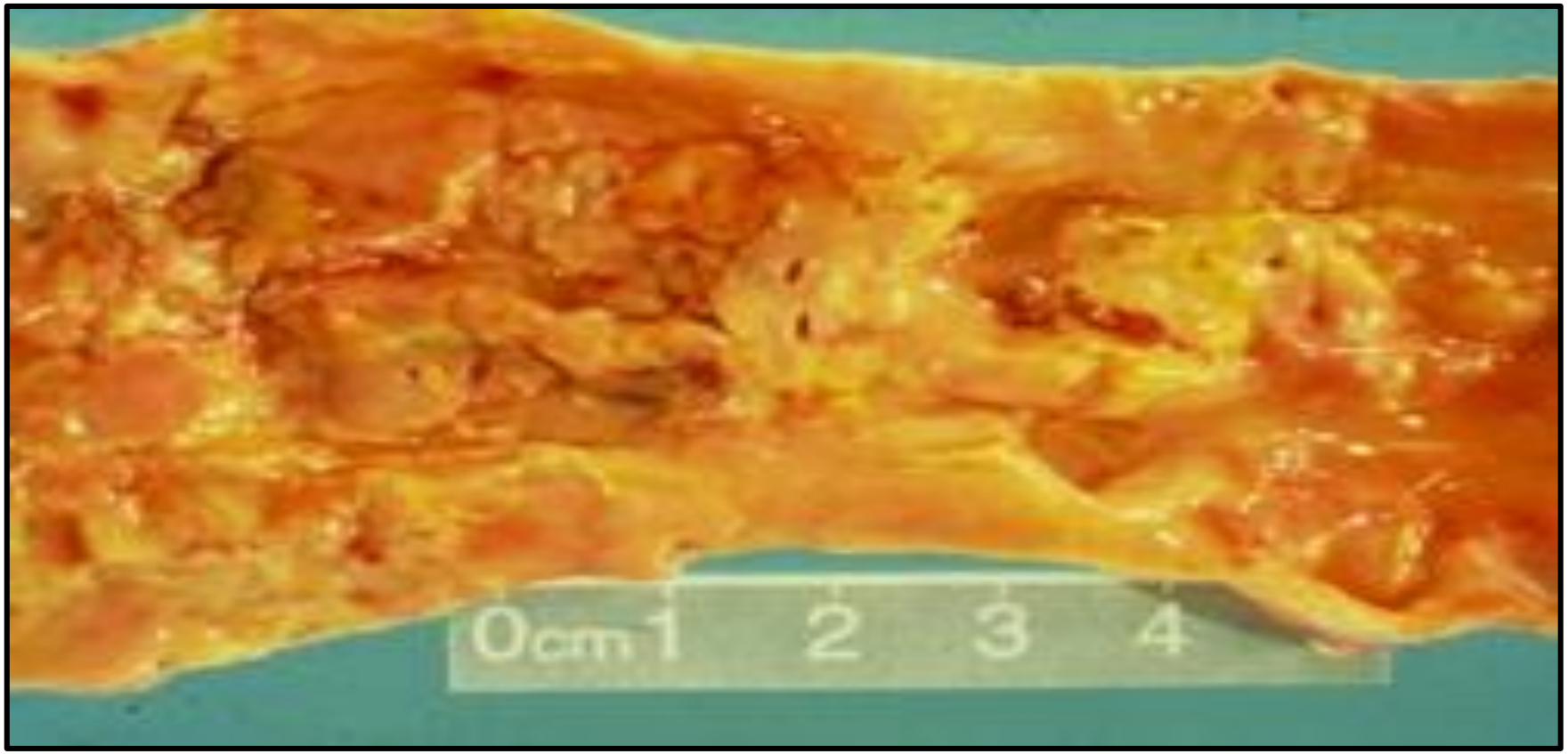


Aorta: complicated atheromatous plaques

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



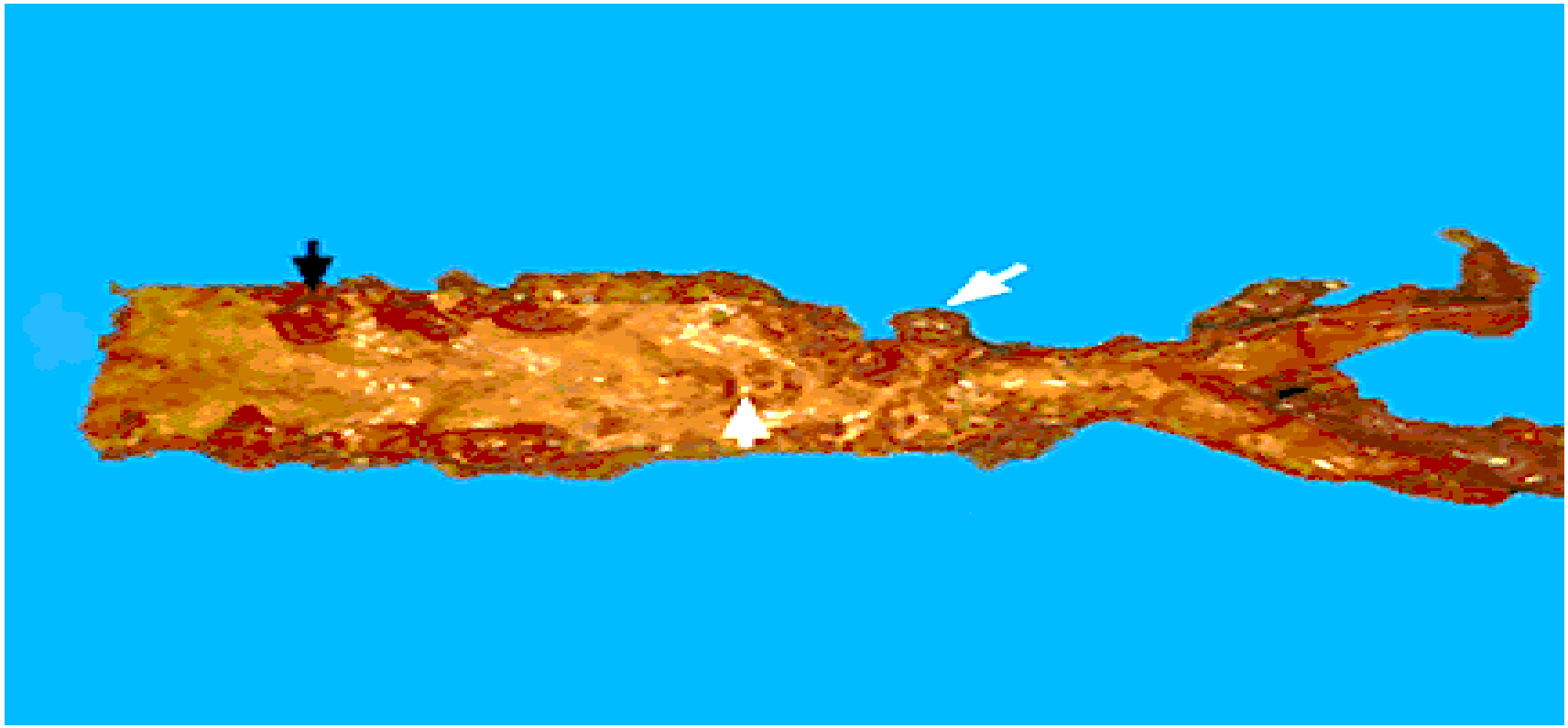
Aorta: complicated atheromatous plaques

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

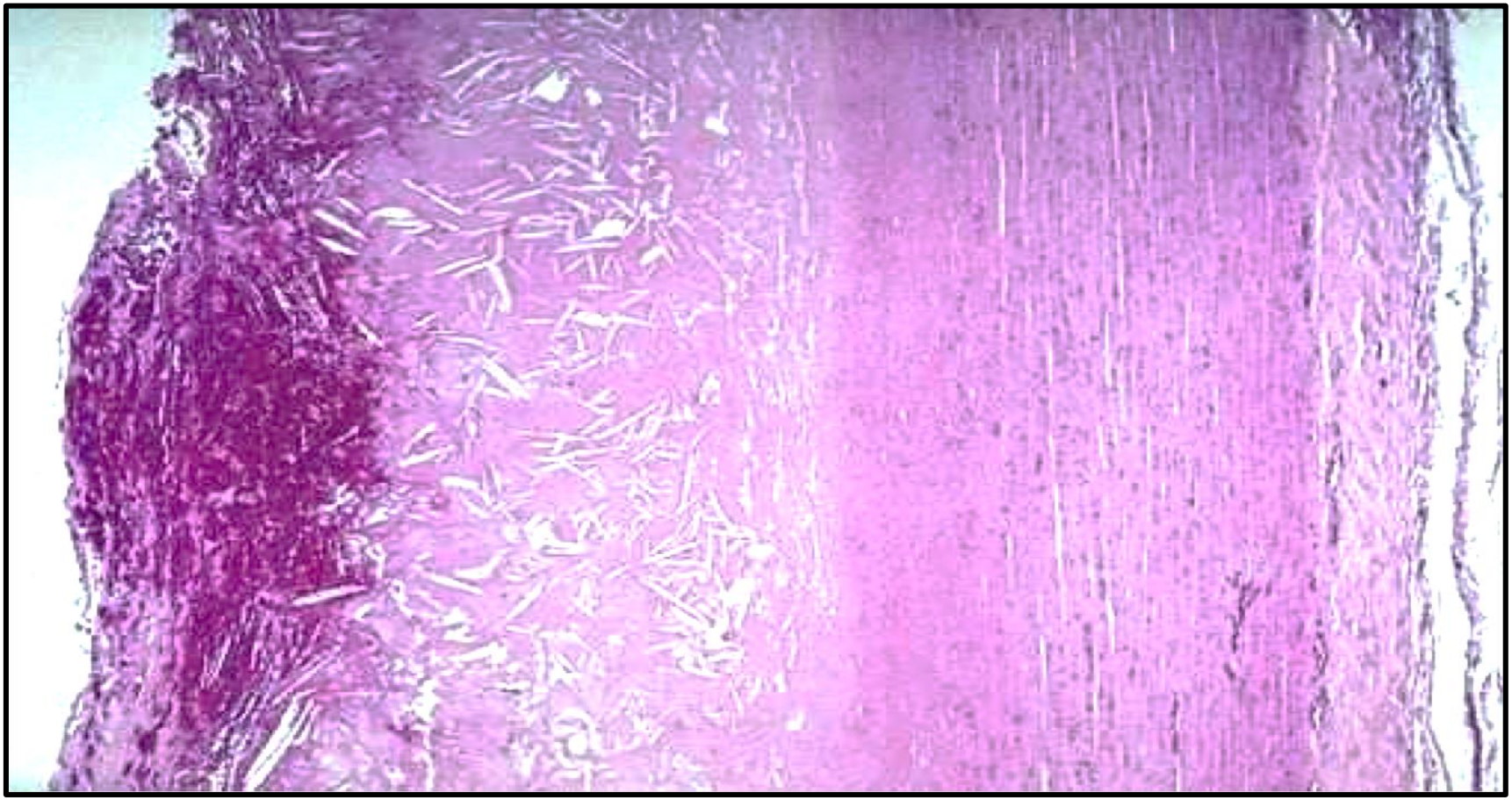


- *Inner surface of aorta and bifurcation,*

opened lengthwise along the posterior midline.

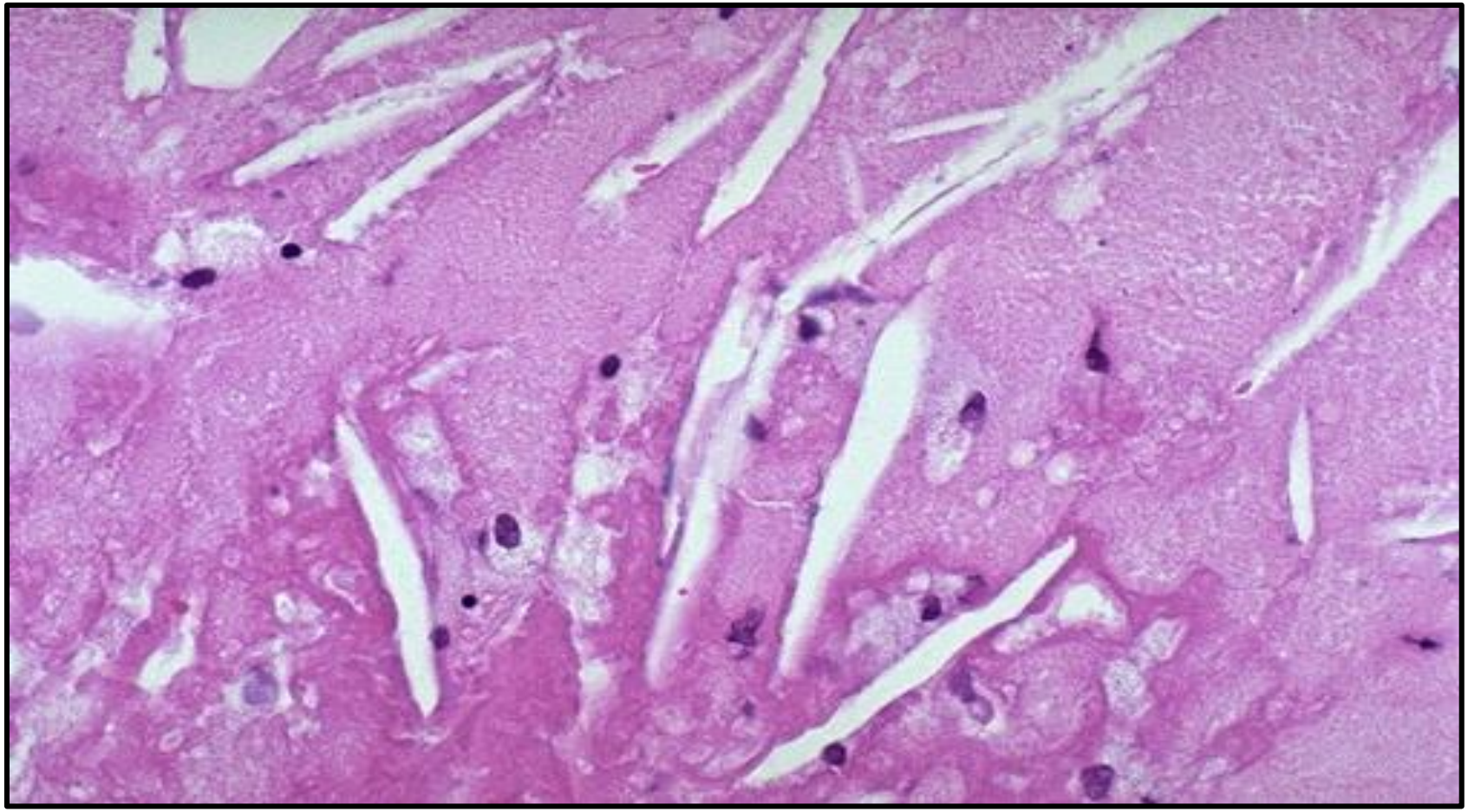
- *Note: irregular variegated lining due to diffuse disease, with red thrombi (black arrow); ostia of celiac and superior mesenteric arteries and right renal artery (white arrows); deceptive narrower caliber of abdominal aorta below celiac artery due to rigidity of calcified atheroma*

Atheroma of the Aorta - LPF



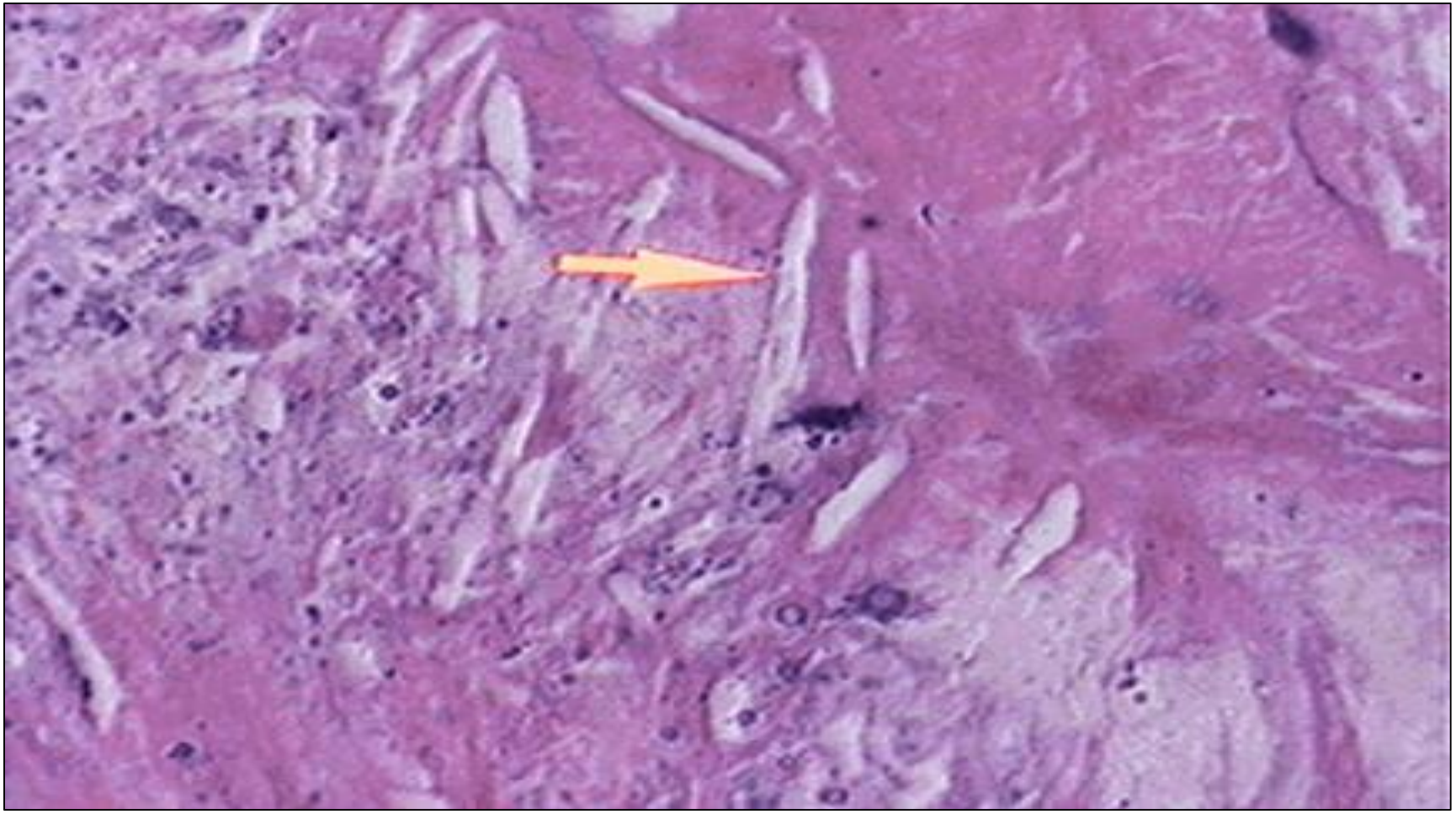
This microscopic cross section of the aorta shows a large overlying atheroma on the left. Cholesterol clefts are numerous in this atheroma. The surface on the far left shows ulceration and hemorrhage. Despite this ulceration, atheromatous emboli are rare

Atheroma of the Aorta - LPF



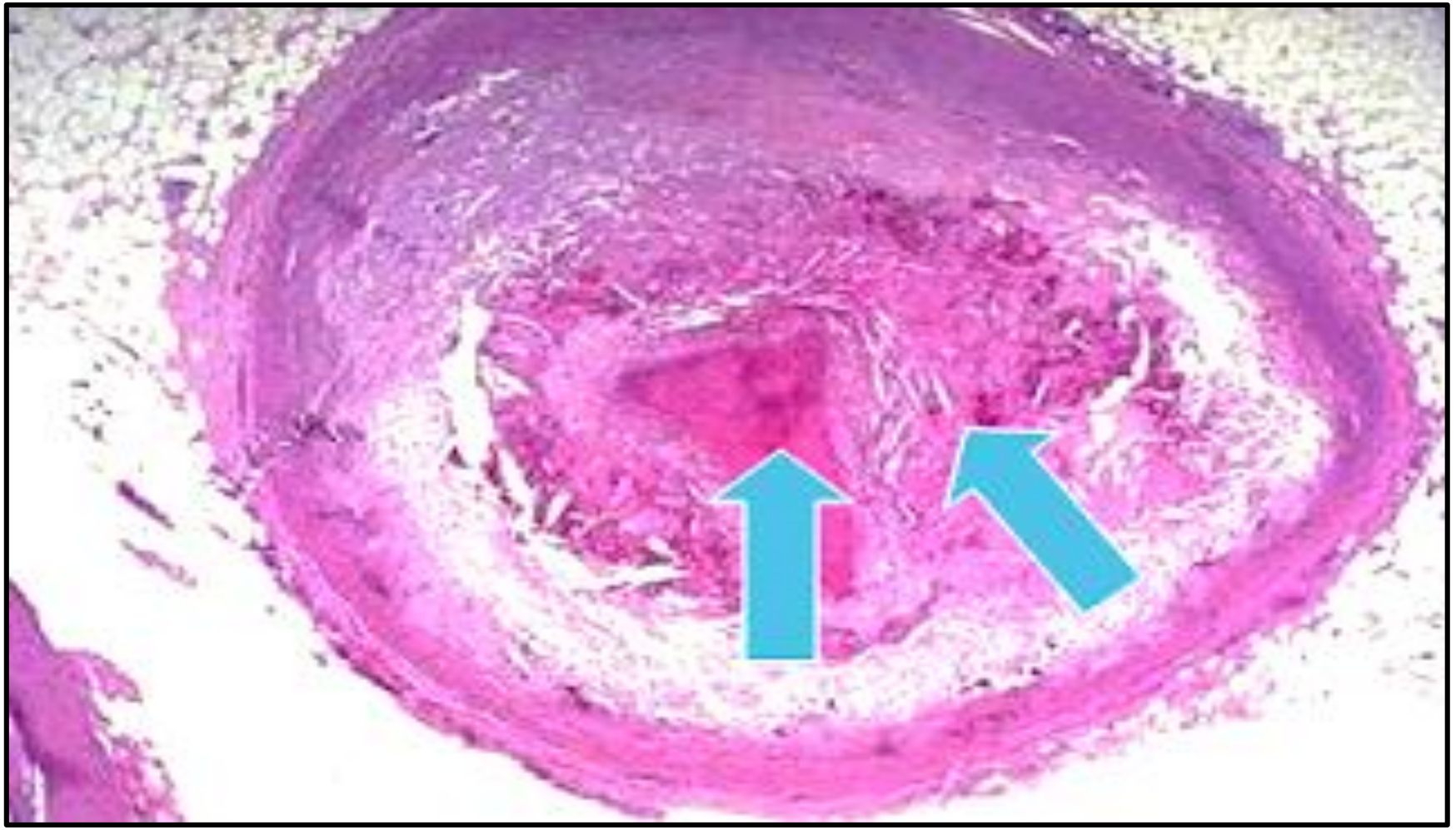
A high magnification of the aortic atheroma with foam cells and cholesterol clefts.

Aortic atherosclerosis - HPF



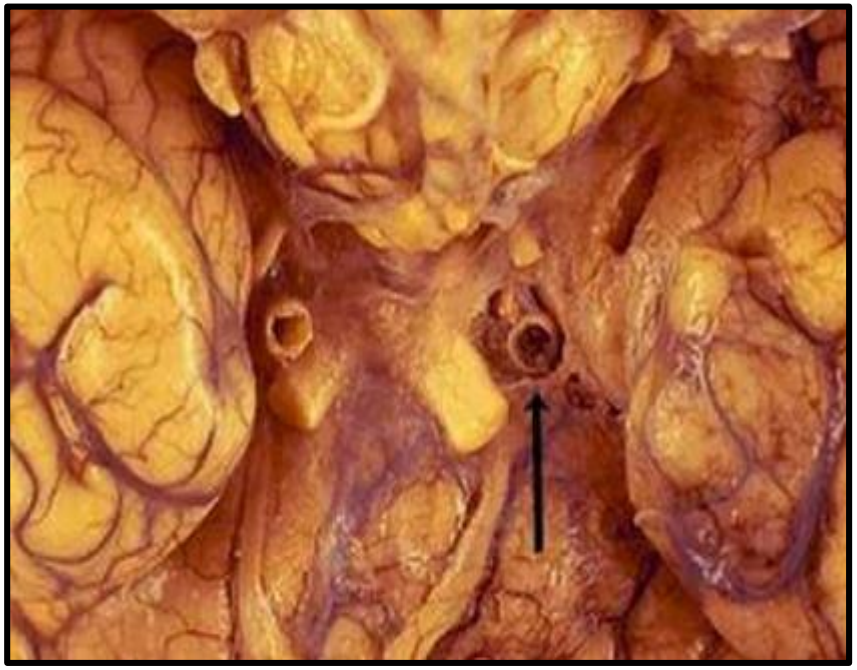
High power view of intimal aspect of atherosclerotic plaque showing stippling by blue calcific spherules, cholesterol crystal clefts, and fibrous cap.

Atheroma of the Aorta - MPF

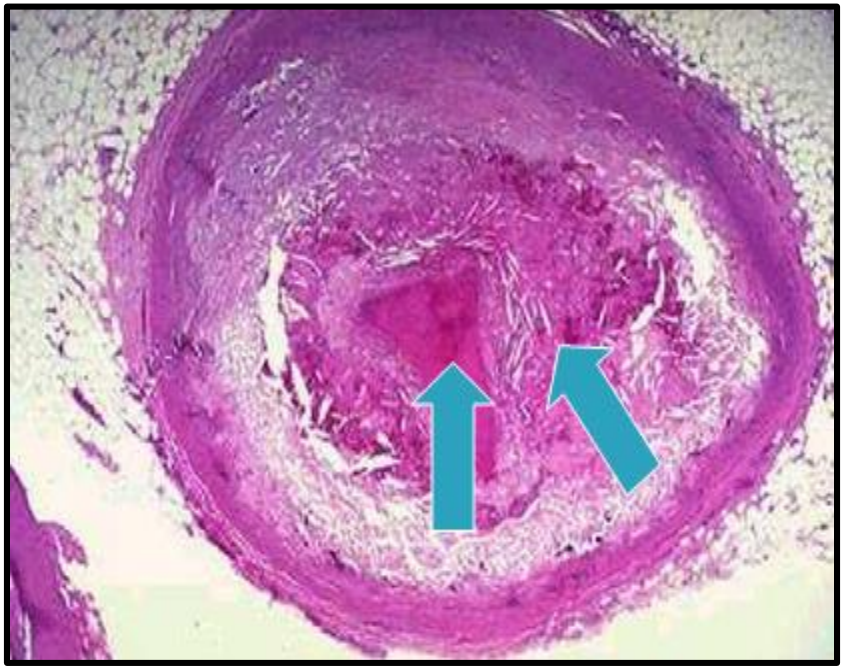


Aortic Atheroma with Thrombosis

Thromboembolism

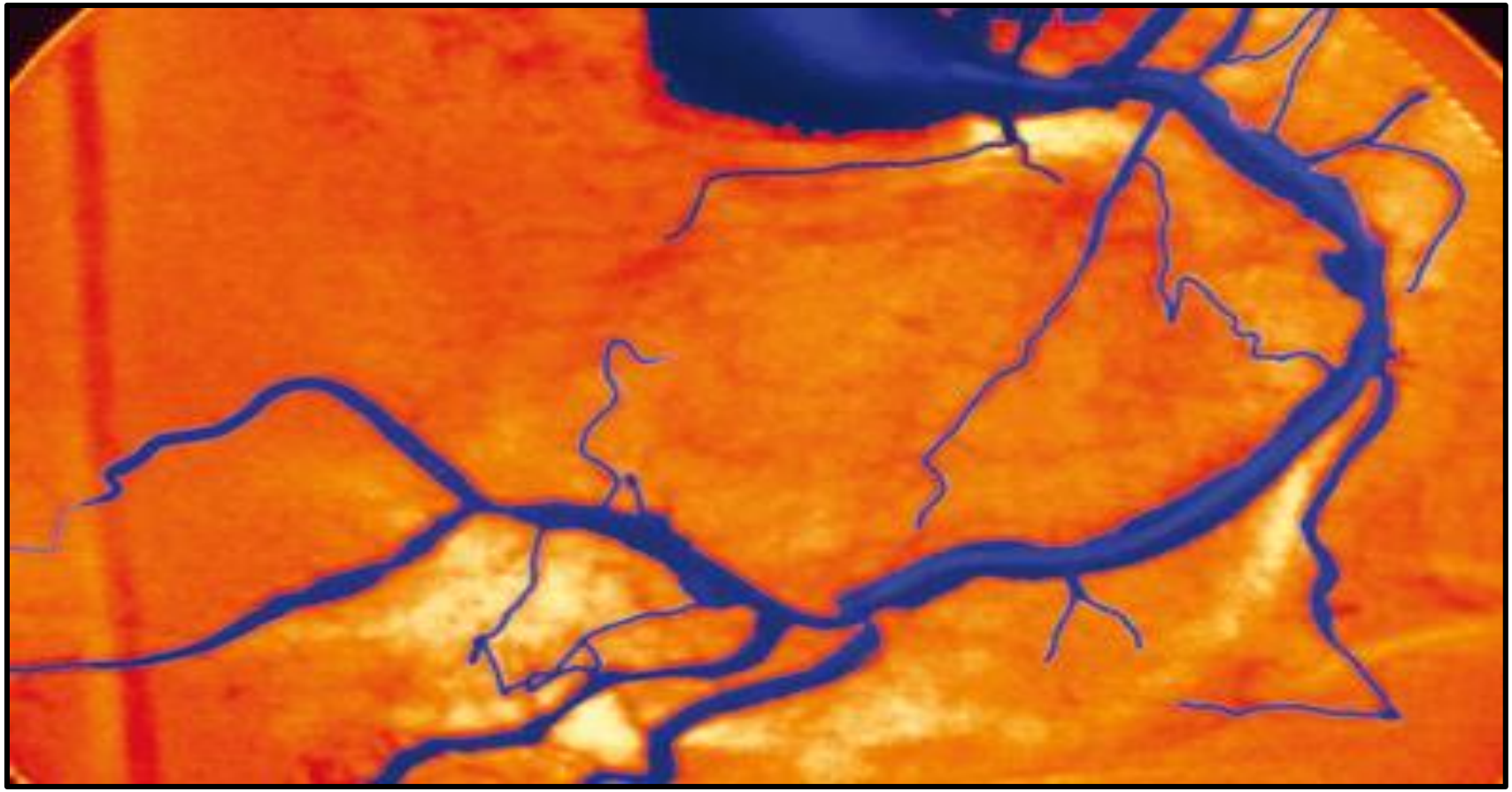


Atheroma with thrombosis



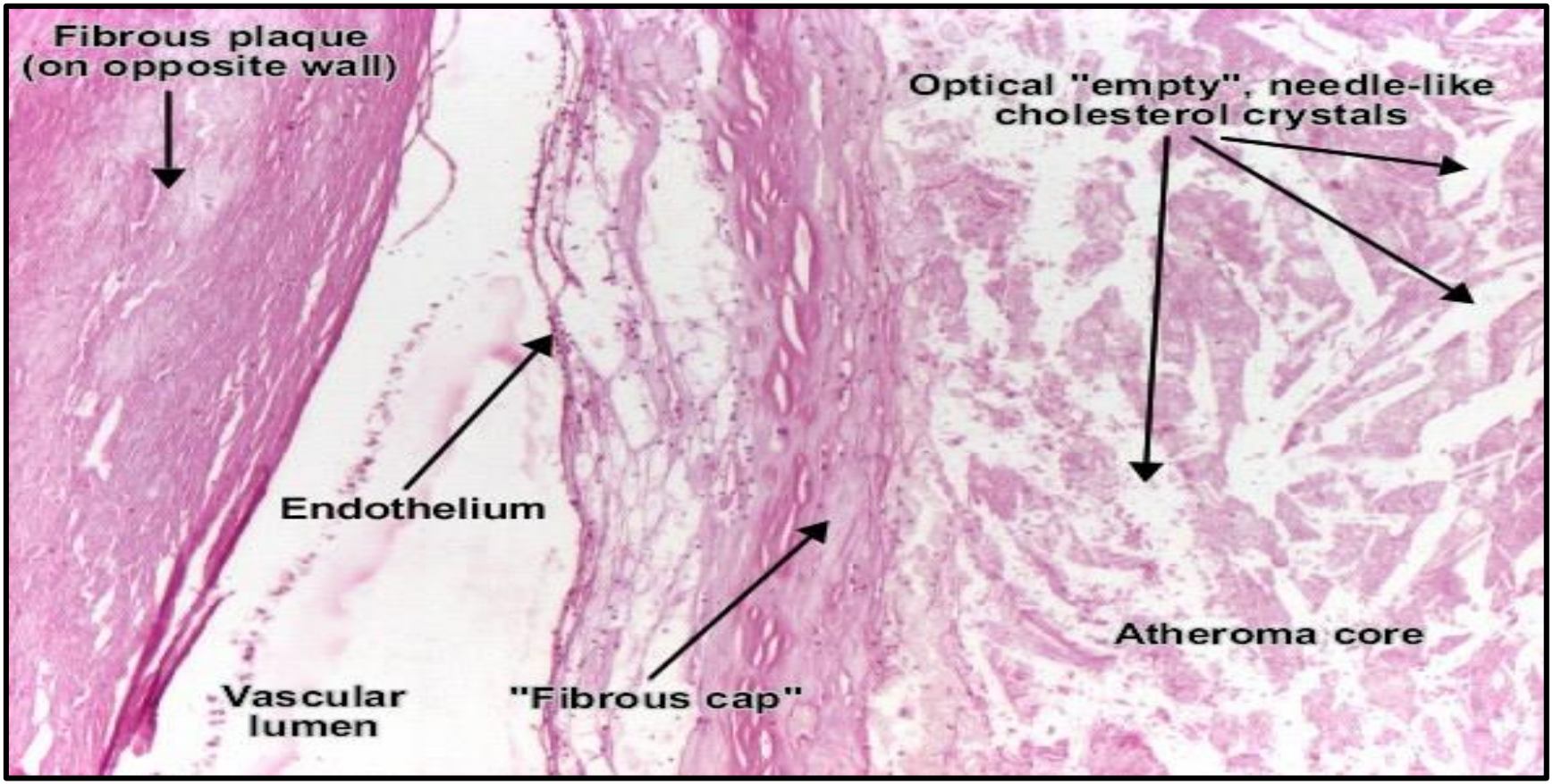
CORONARY ATHEROSCLEROSIS

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*

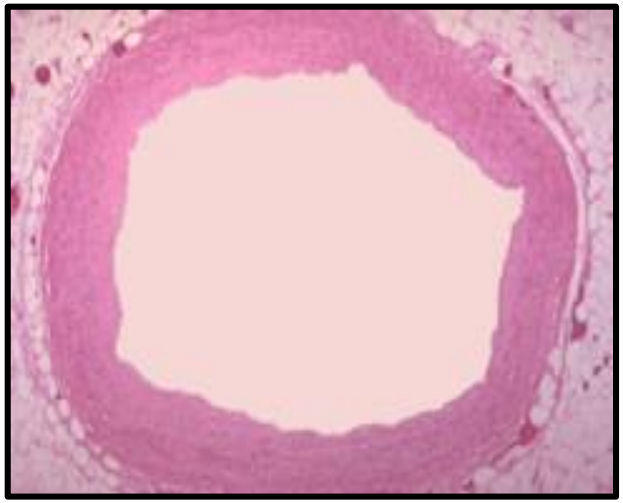
Coronary atherosclerosis - LPF



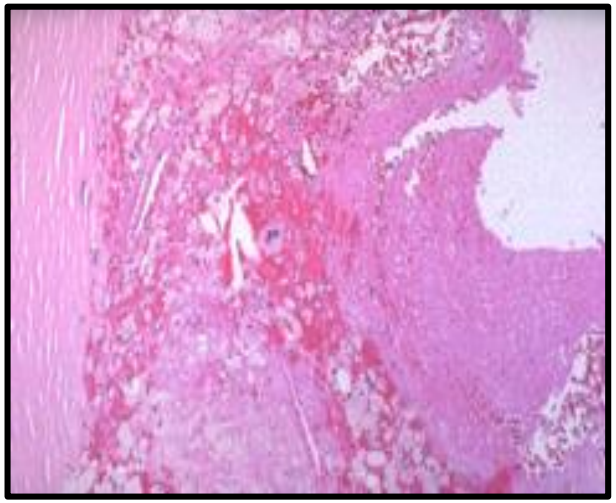
Coronary artery with atherosclerosis (fibro-lipid or fibro-fatty plaque). The atheromatous fibro-fatty plaque is characterized by the accumulation of lipids in the intima of the arteries, narrowing the lumen. Beneath the endothelium it has a "fibrous cap" covering the atheromatous "core" of the plaque.

Atherosclerotic plaques have three principal components: (1) cells: *smooth muscle cells, macrophages,* T cells; (2)ECM: collagen, elastic fibers, and proteoglycans; and (3) intracellular and extracellular lipid

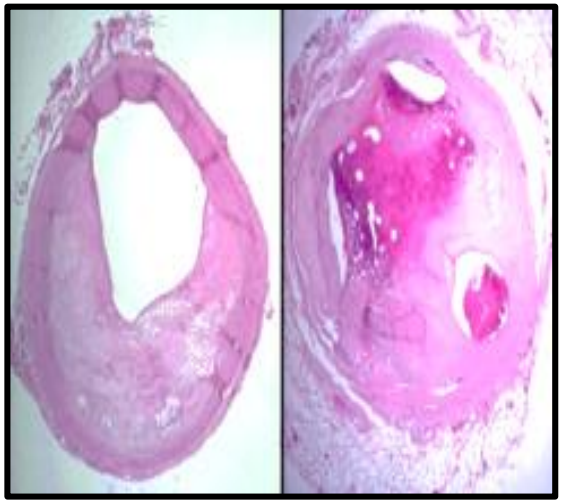
Coronary atherosclerosis - LPF



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

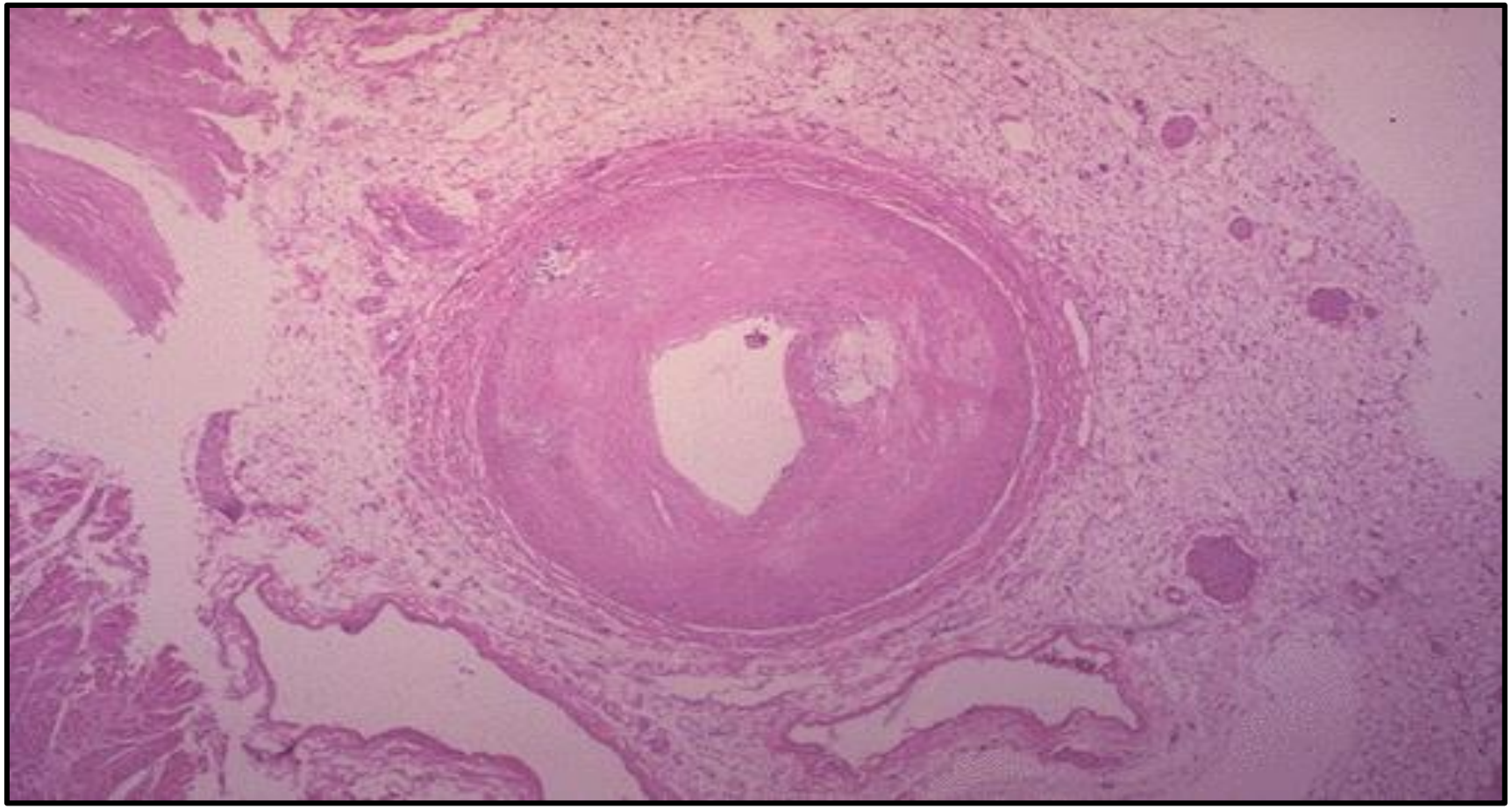


Atheromatous plaque in a coronary artery that shows endothelial denudation with disruption and overlying thrombus formation at the right. The arterial media is at the left



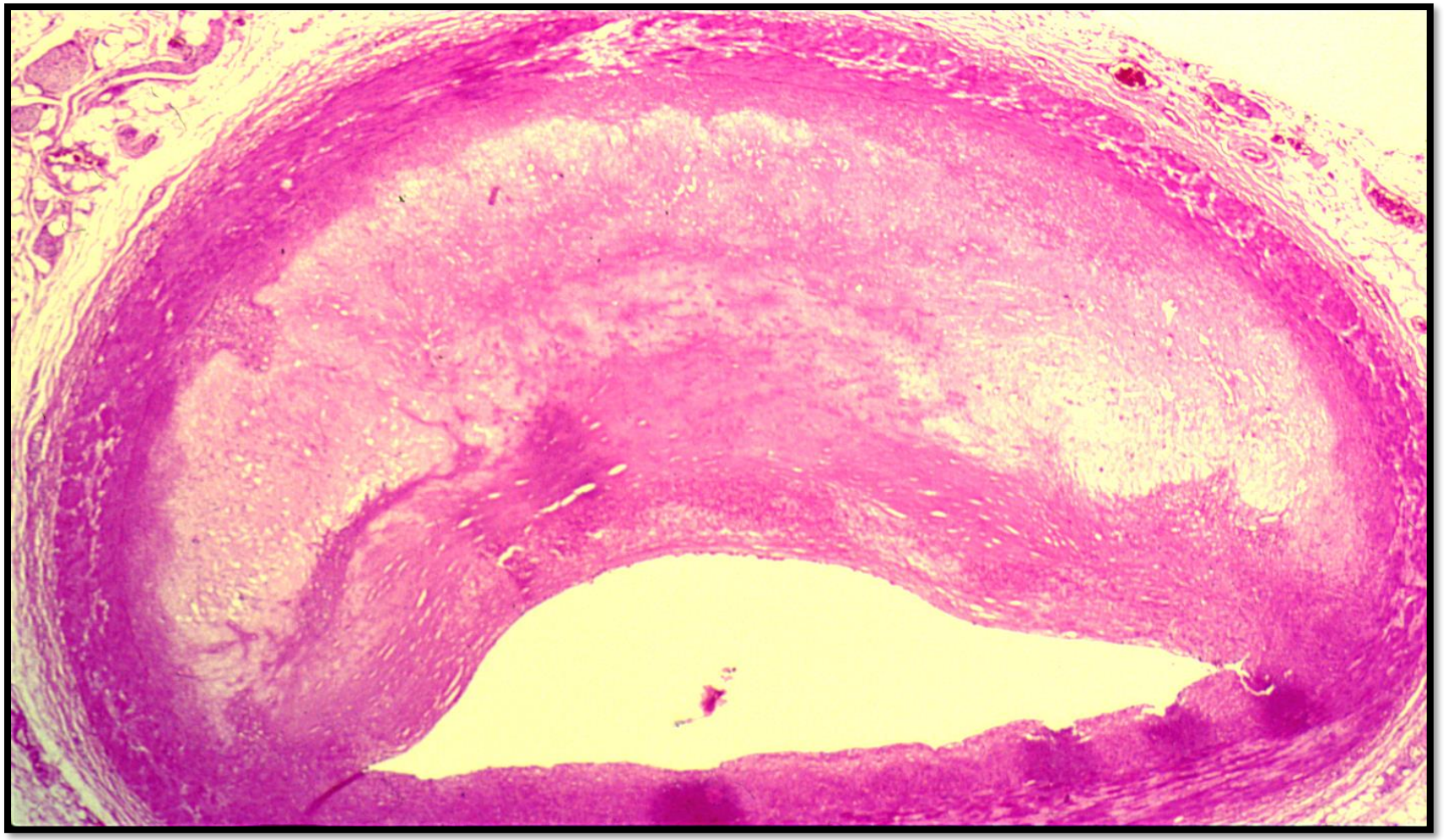
Occlusive coronary atherosclerosis. The coronary at the left is narrowed by 60 to 70%. The coronary at the right is even worse with evidence for previous thrombosis with organization of the thrombus

Coronary atherosclerosis - MPF



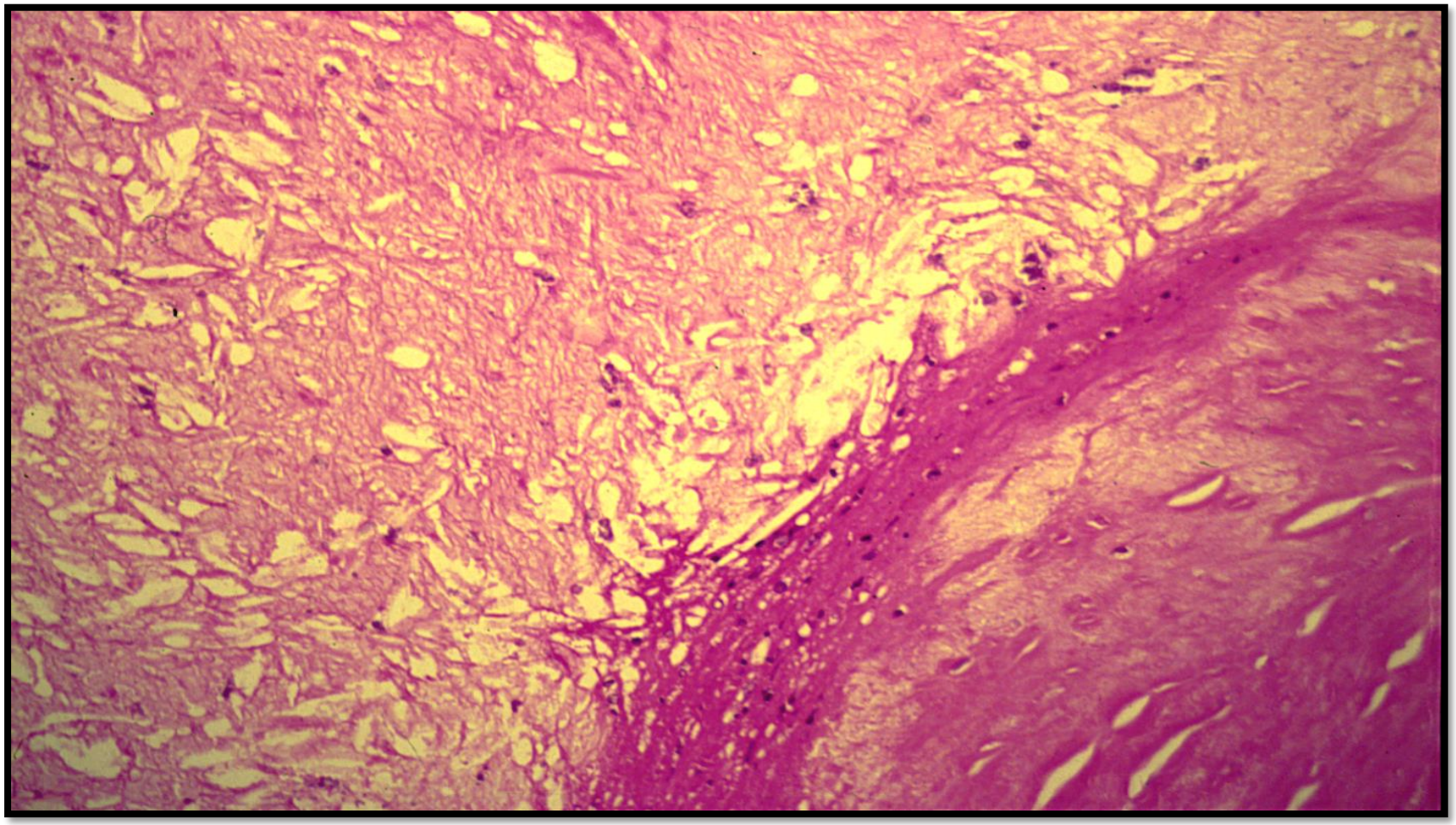
This distal portion of coronary artery shows significant narrowing. Such distal involvement is typical of severe coronary atherosclerosis, such as can appear with diabetes mellitus or familial hypercholesterolemia.

Coronary atherosclerosis - MPF



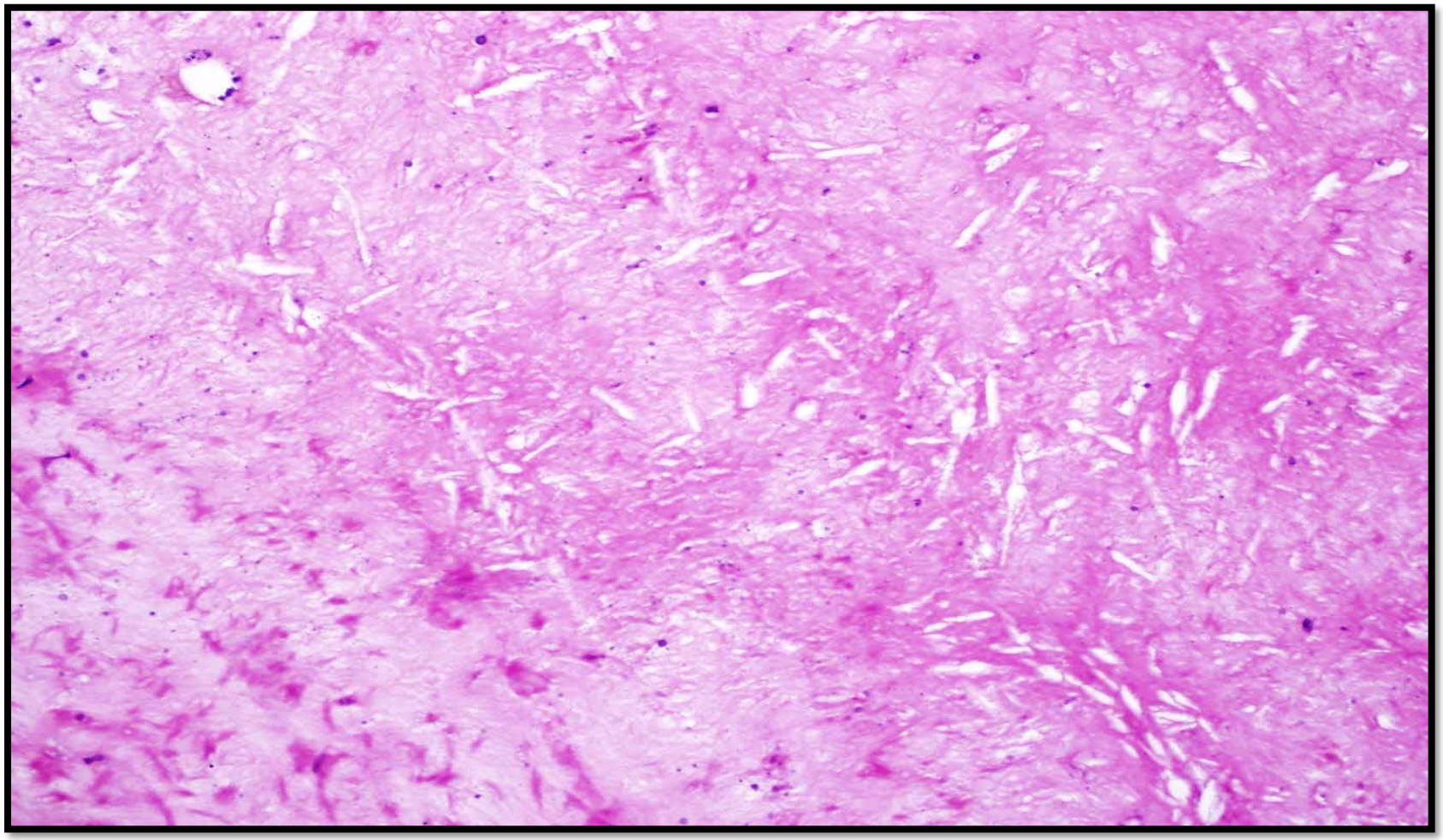
Severe coronary atherosclerosis with narrowing of the lumen

Coronary atherosclerosis - HPF



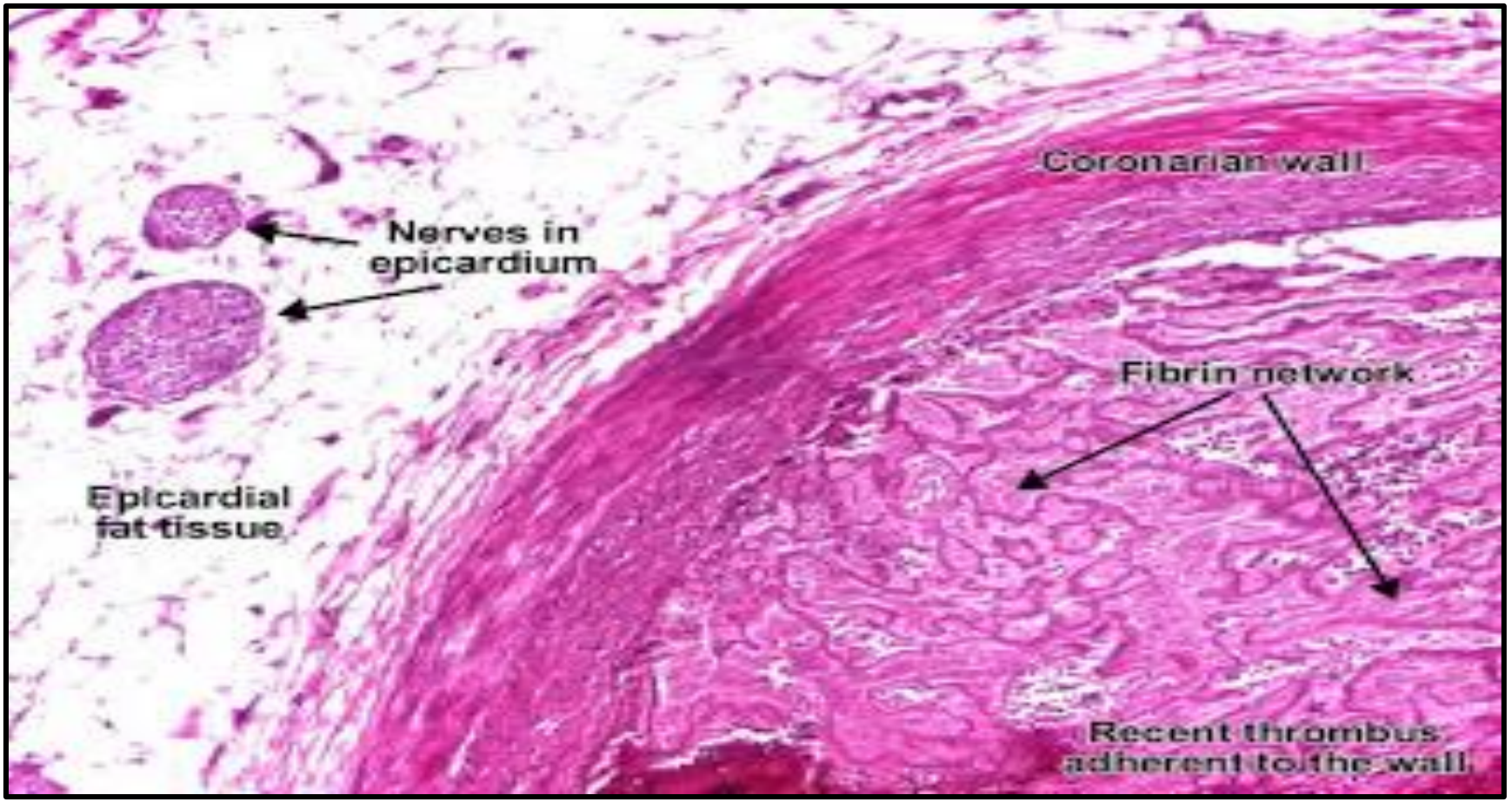
Partial occlusion of the lumen by an atheromatous plaque. The plaque consists of dissolved, cholesterol clefts, hyaline fibrous tissue and some blood capillaries.

Coronary atherosclerosis - HPF



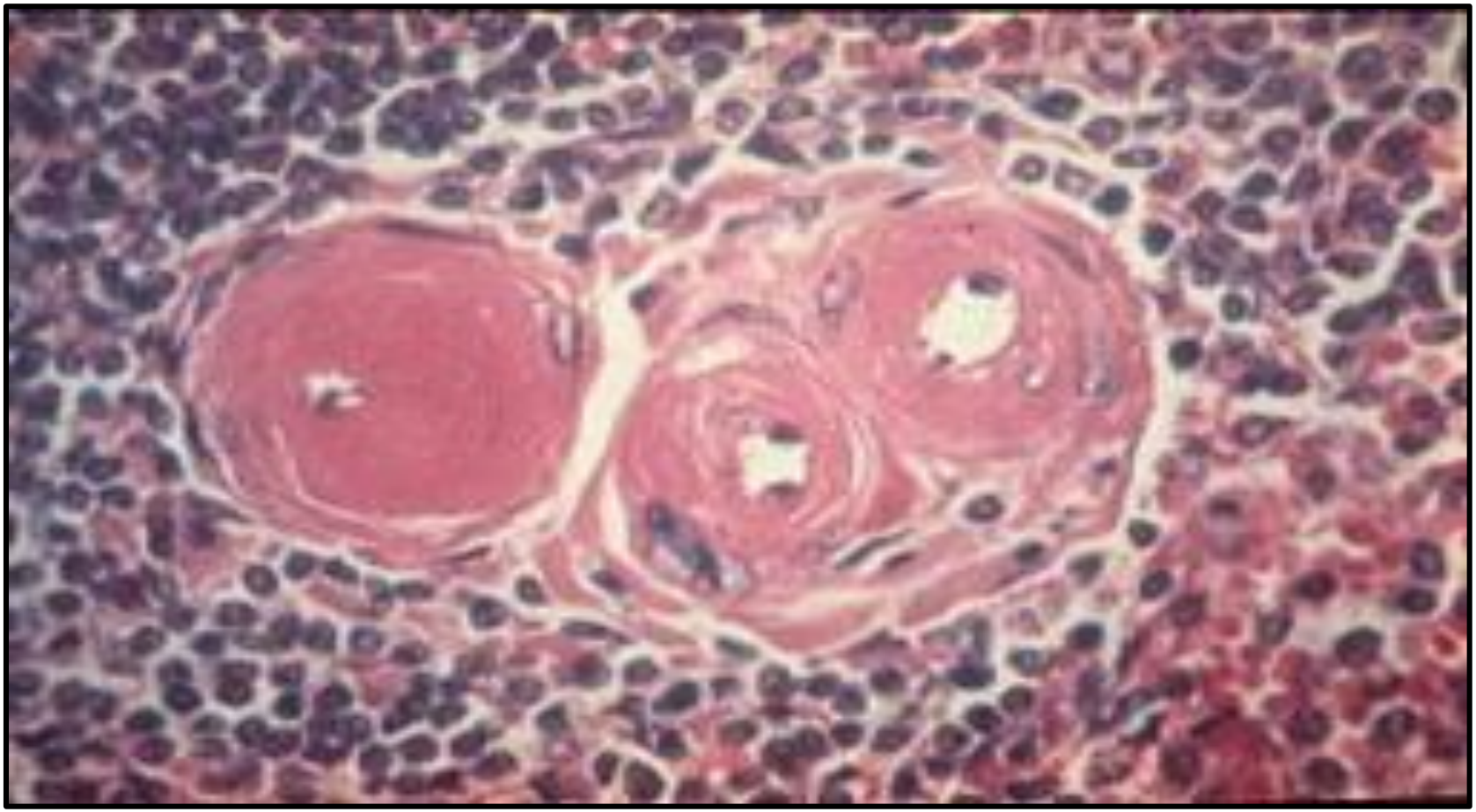
The internal elastic lamina is thin and fragmented.
Pressure atrophy of the media opposition atheromatous plaque consists of cholesterol clefts, hyaline fibrous tissue and some blood capillaries.

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.*

VASCULAR PATHOLOGY IN HYPERTENSION
Hyaline arteriolosclerosis - HPF

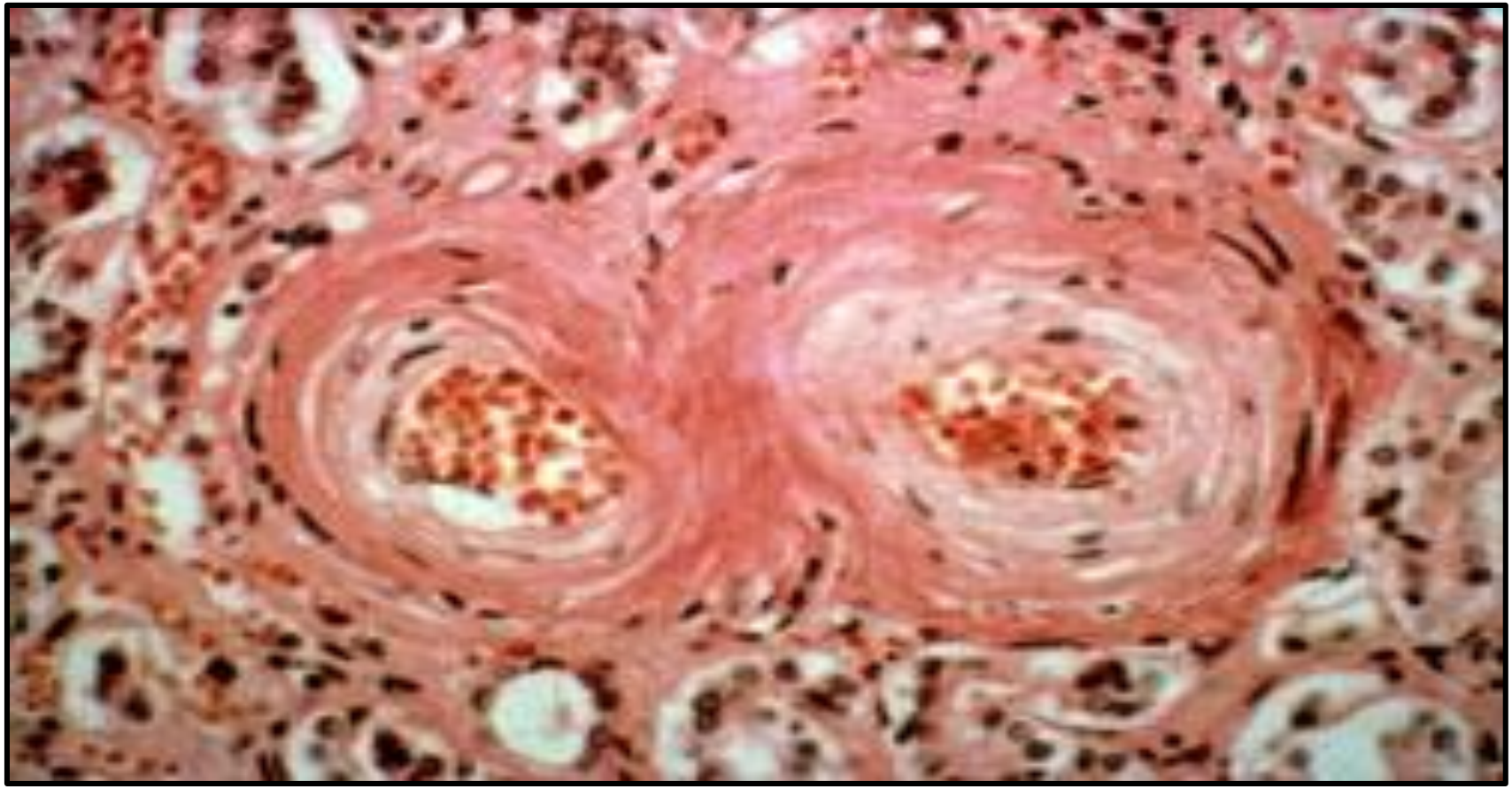


Hyaline arteriolosclerosis

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

Arterioles show homogeneous, pink hyaline thickening with associated luminal narrowing

Hyperplastic arteriolosclerosis - HPF



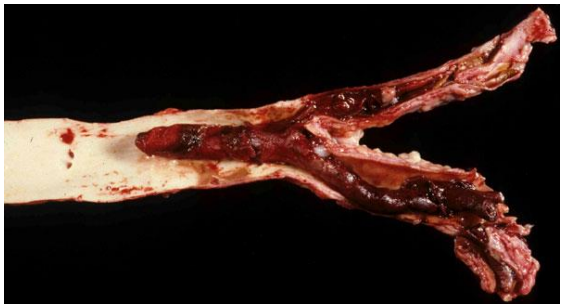
***Hyperplastic arteriolosclerosis**: This is the other type of small vessel arteriosclerosis. It is predominantly seen in malignant hypertension and renal disease associated with polyarteritis nodosa and progressive systemic sclerosis. Vessels exhibit "onion-skin lesions," characterized by concentric, laminated thickening of the walls and luminal narrowing*

THROMBOEMBOLISM

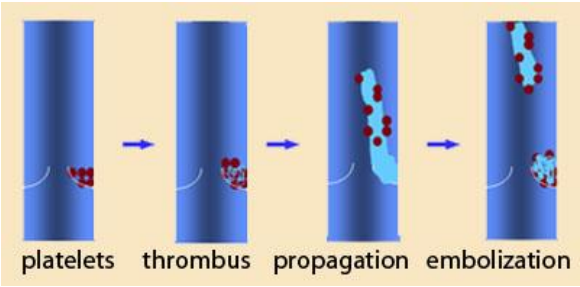
BACKGROUND INFORMATION

Background information: Thrombosis

- Thrombosis is a process by which a thrombus is formed.
- A thrombus is a solid mass of blood constituents which develops in artery, vein or capillary.
- It is intravascular coagulation of blood and it can cause significant interruption to blood flow.
- Thrombi may develop anywhere in the cardiovascular system, the cardiac chambers, valve surface, arteries, veins, or capillaries. They vary in size and shape, depending on the site of origin.
- Thrombi in the vein are called **venous thrombi.** Thrombi in the artery are called **arterial thrombi.** When arterial thrombi arise in heart chambers or in aorta they are termed **mural thrombi.**
- Thrombi can grow. The propagating/growing tail of the thrombi is weak and is prone to fragmentation, creating an **embolus**

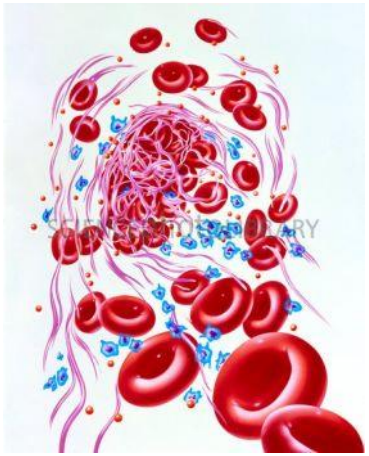


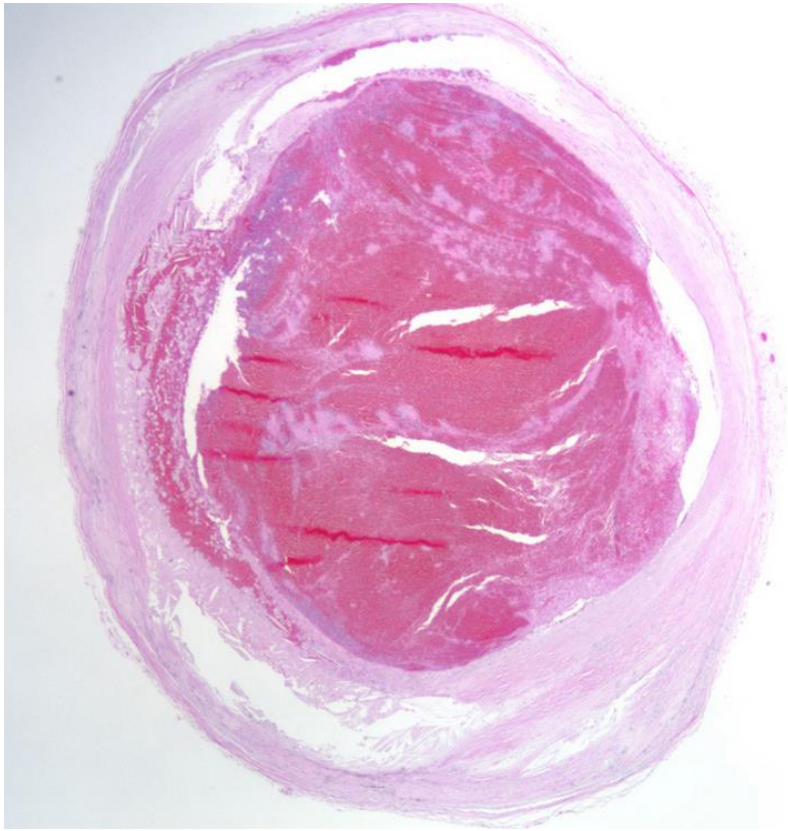
Mural thrombus.



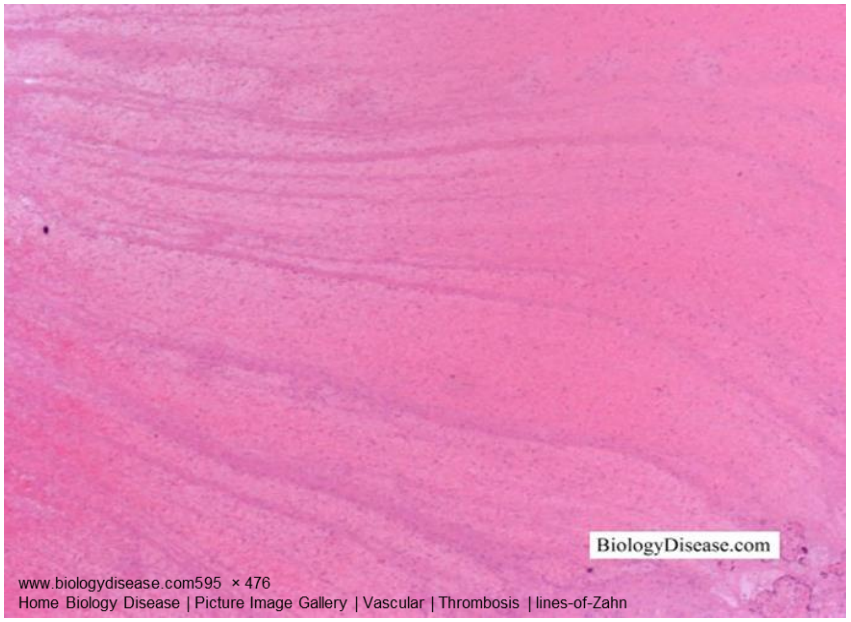
Background information: Morphology of thrombus

- A thrombus is made up of fibrin, platelets & red blood cells and some inflammatory cells.
- When formed in the heart or aorta, thrombi may have laminations produced by alternating of pale and dark layers, called **lines of Zahn**; the pale layers contain platelets mixed with fibrin. The darker layers contain red blood cells.

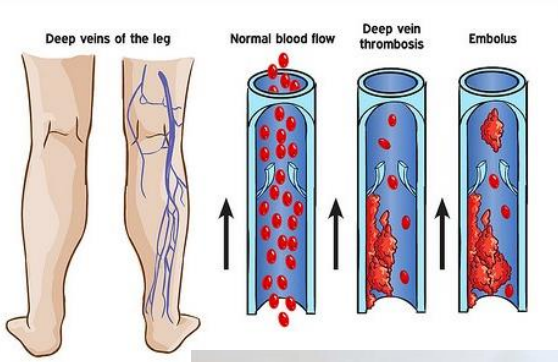


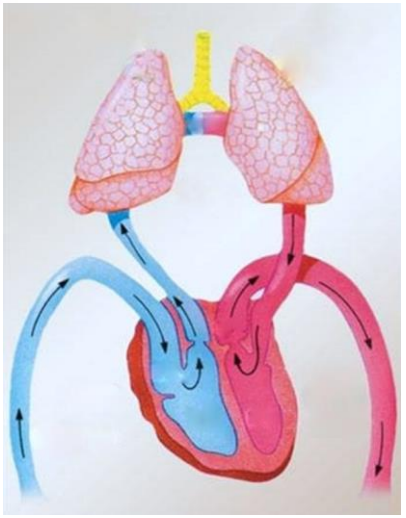


Background information:
Lines of Zahn

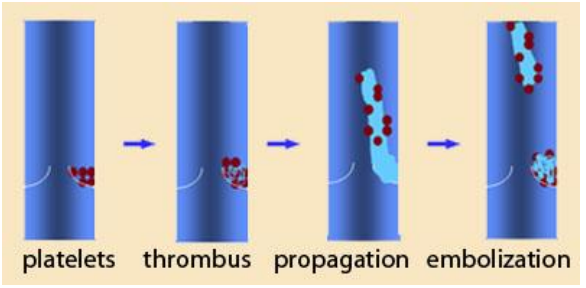


Background information: EMBOLISM



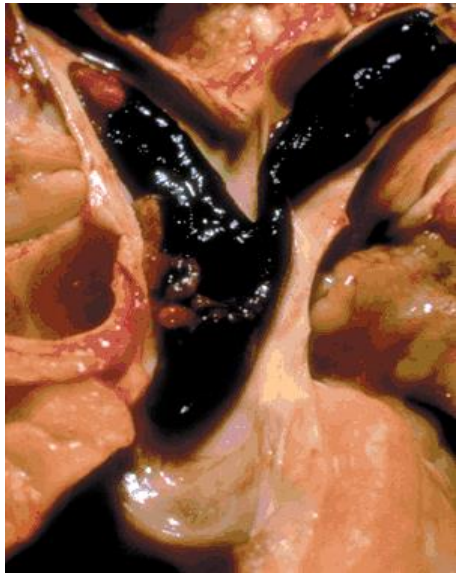


- *An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin.*
- Almost all emboli represent some part of a dislodged thrombus, hence the commonly used term *thromboembolism*.
- The emboli ultimately lodged in vessels too small to permit further passage, resulting in partial or complete vascular occlusion leading to ischemic necrosis of distal tissue (*infarction*).
- Depending on the site of origin, emboli may lodge in the pulmonary or systemic circulations resulting in a **pulmonary embolus** or **systemic embolus**.



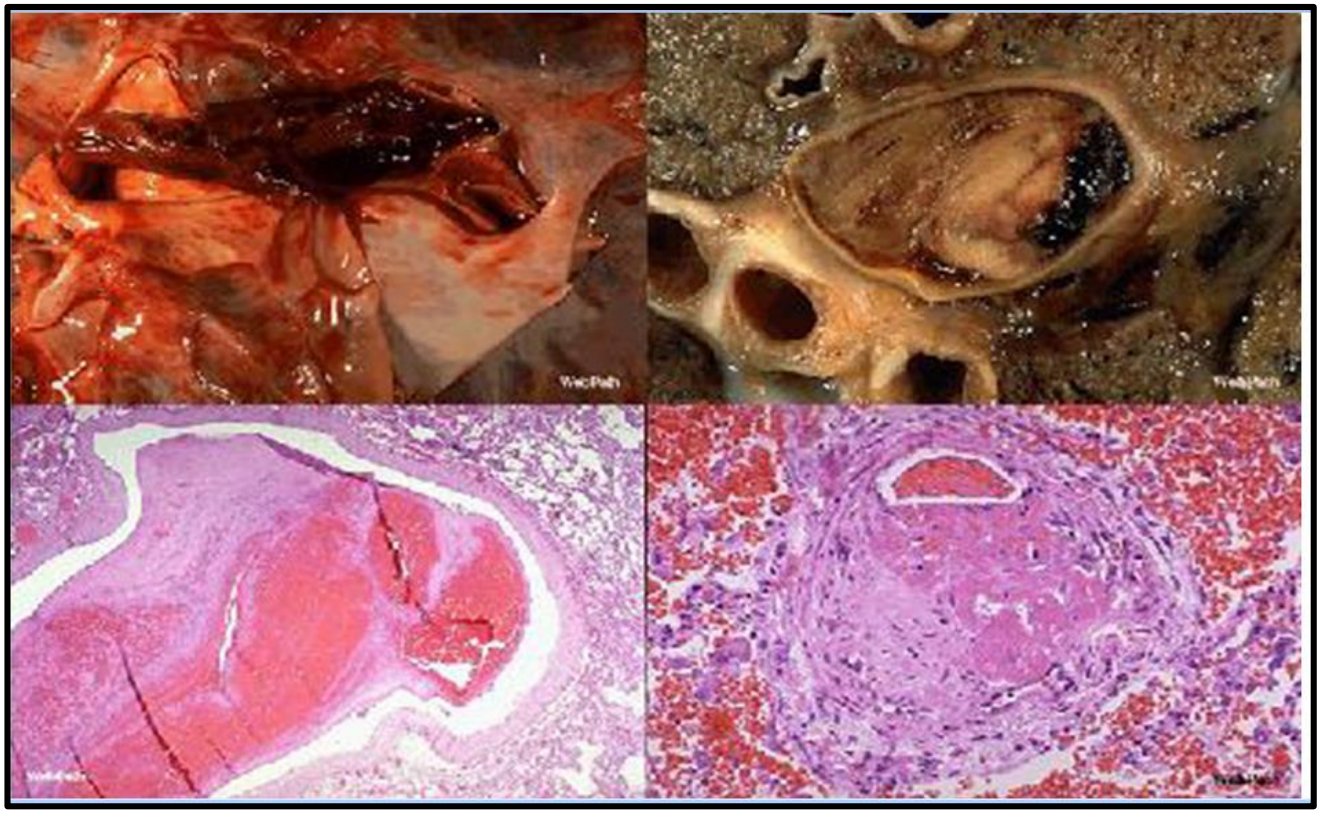
Background information:
PULMONARY THROMBOEMBOLISM

- Here the embolus get lodged in the pulmonary vasculature.
- Depending on size of embolus, it may get stuck and block the main pulmonary artery or block the bifurcation of the pulmonary trunk *(saddle embolus)* or pass out into the smaller, branching arterioles of the pulmonary circulation.
- Most pulmonary emboli (60% to 80%) are clinically silent because they are small.
- Sudden death or cardiovascular problems occurs when 60% or more of the pulmonary circulation is obstructed with emboli.
- Embolic obstruction of small end-arteriolar pulmonary branches may result in infarction.



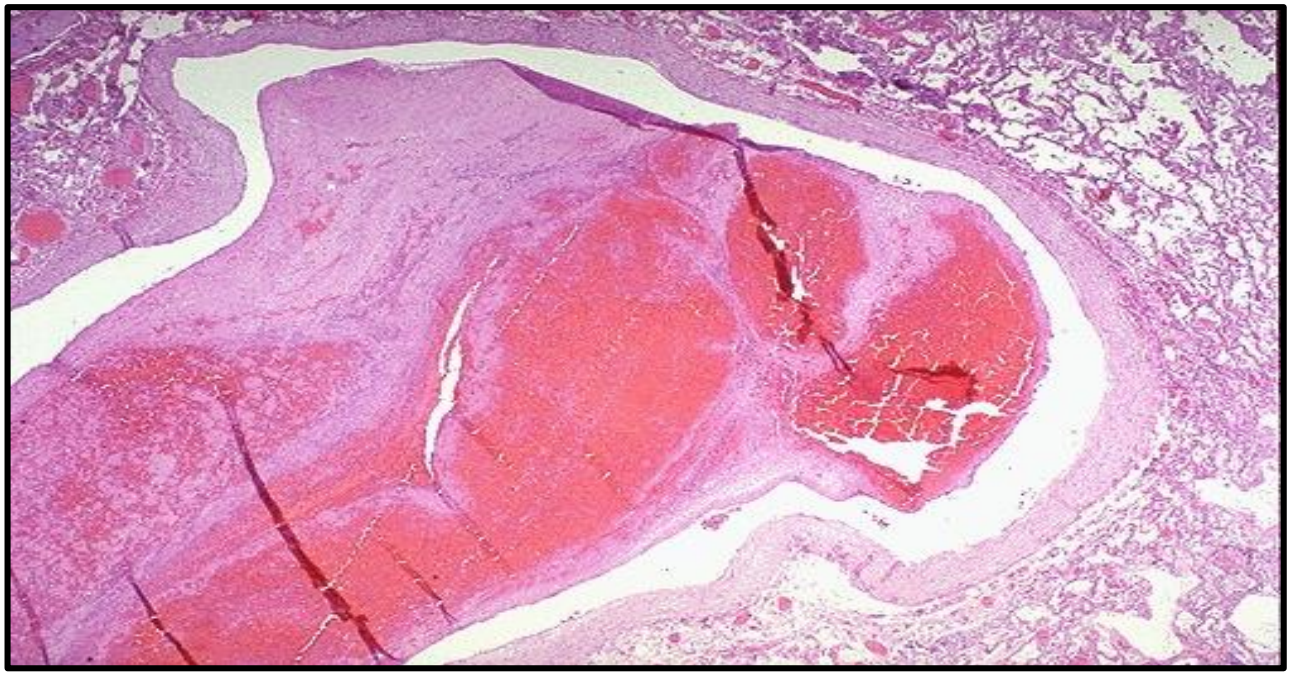
ORGANIZING THROMBUS

Organizing Thrombus



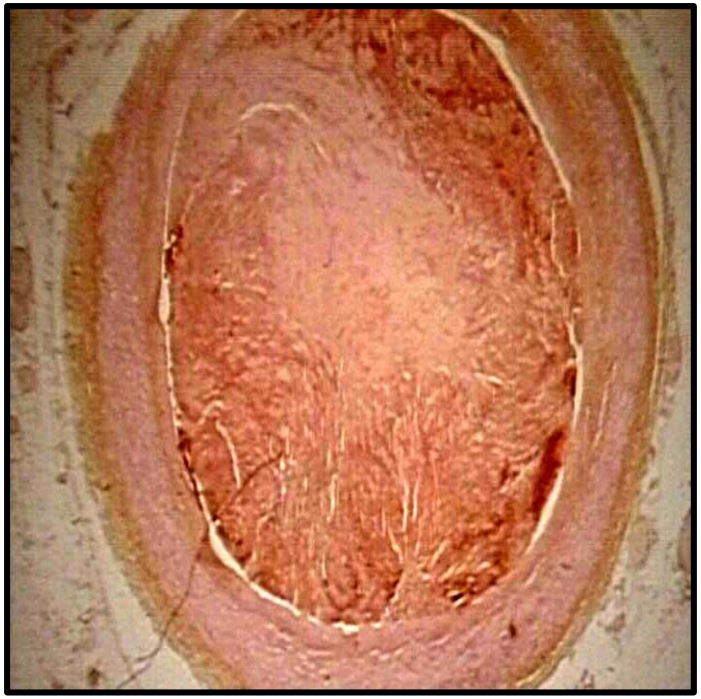
Organizing thrombus in a case of pulmonary embolism

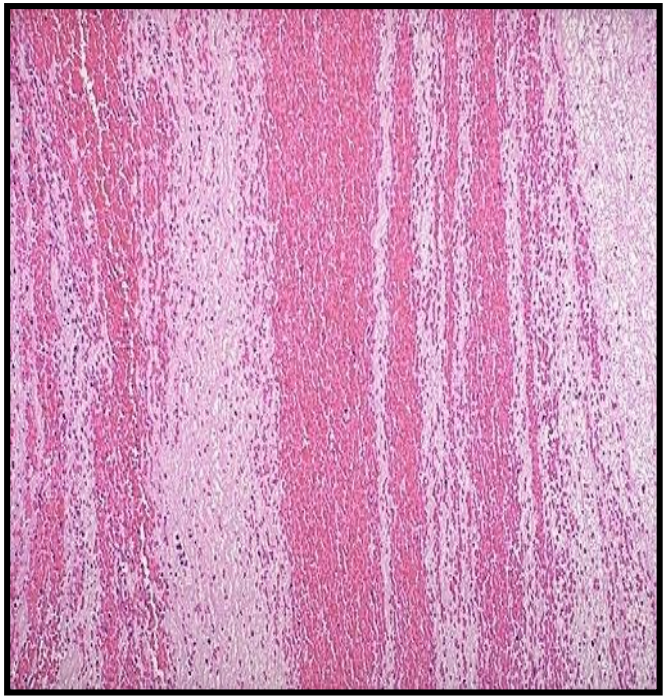
Organizing Thrombus with Lines of Zahn



This is the microscopic appearance of a pulmonary thromboembolus in a large pulmonary artery. There are interdigitating areas of pale pink and red that form the "lines of Zahn" characteristic for a thrombus. These lines represent layers of red cells, platelets, and fibrin which are laid down in the vessel as the thrombus forms.

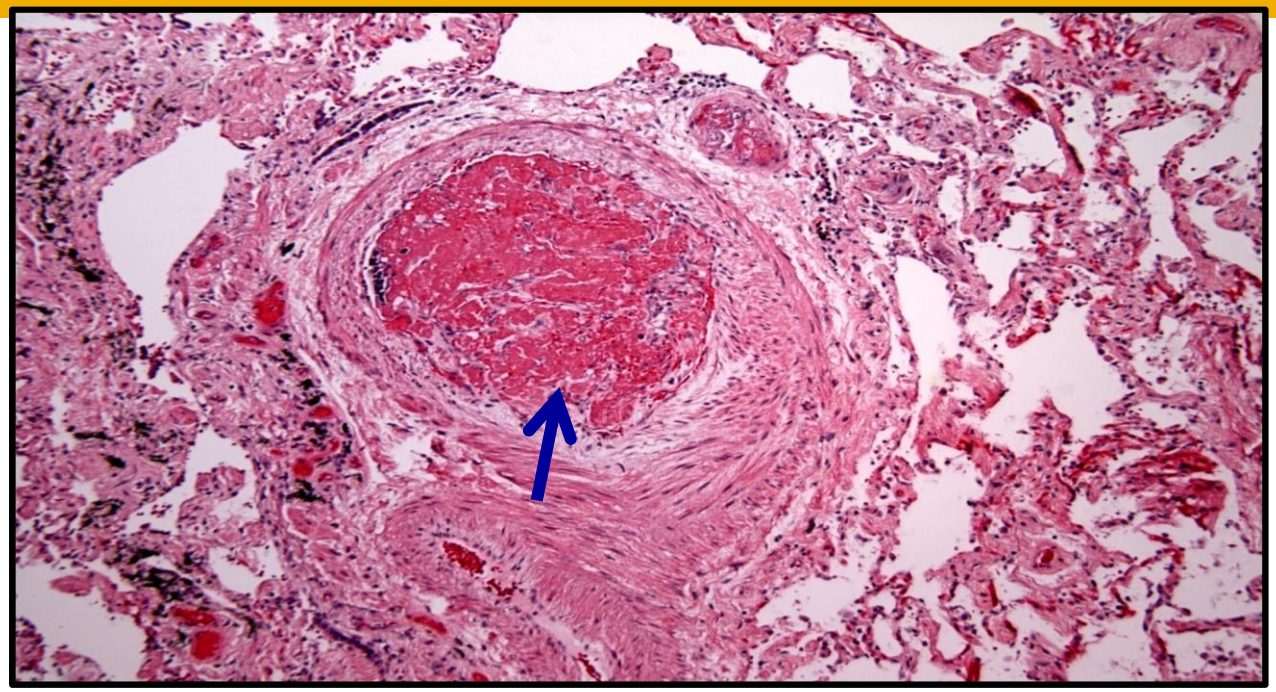
Lines of Zahn





***Lines of Zahn**, gross and microscopic, is evidence to prove a clot is Pre-mortem which is different from the clots appearing like current jelly or chicken fat which are said to be Post-mortem. These lines represent layers of red cells, platelets, and fibrin*

Thromboembolus in Pulmonary Artery



Pulmonary thromboembolus in a small pulmonary artery. The interdigitating areas of pale pink and red within the organizing embolus form the "lines of Zahn" (arrow) characteristic of a thrombus. These lines represent layers of red cells, platelets, and fibrin that are laid down in the vessel as the thrombus forms

Vasculitis

is a general term for vessel wall inflammation

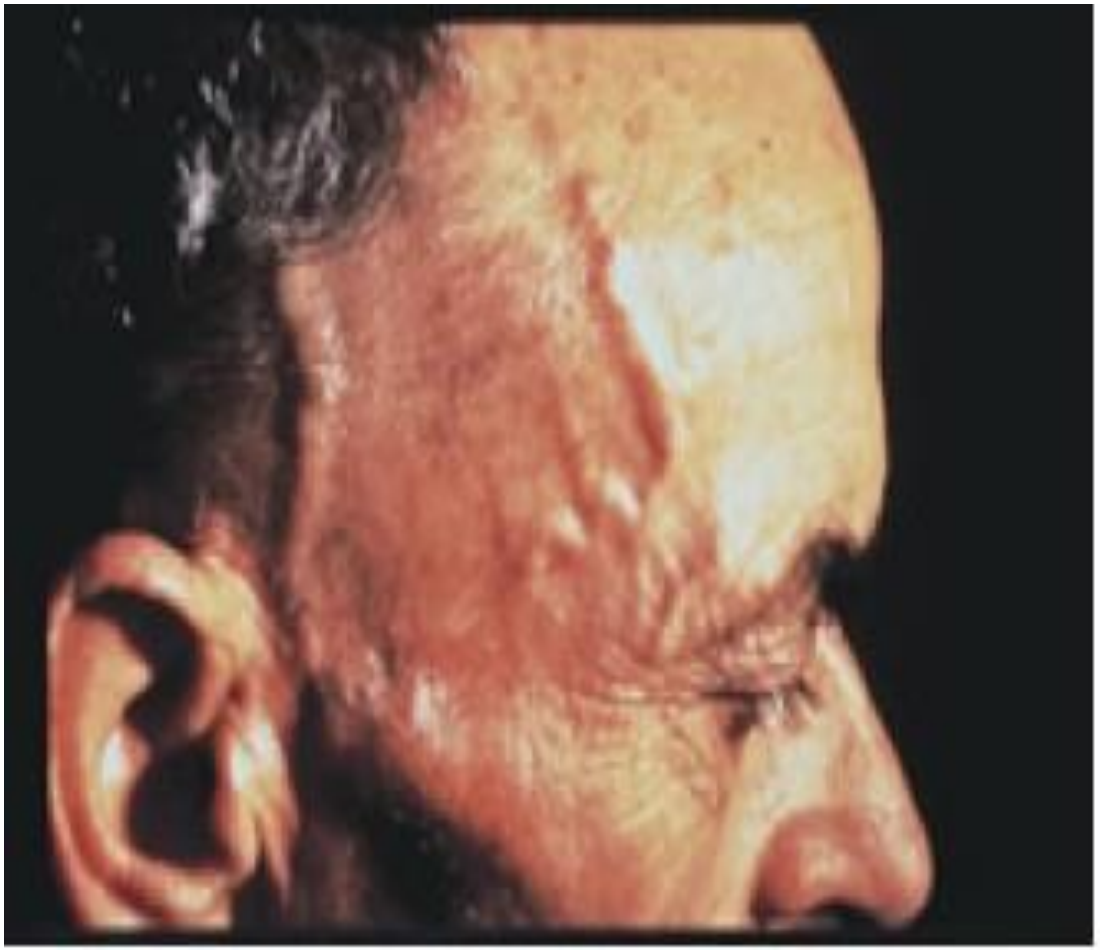
Boehringer Ingelheim

GIANT CELL (TEMPORAL) ARTERITIS

IS THE MOST COMMON FORM OF VASCULITIS AMONG ELDERLY INDIVIDUALS

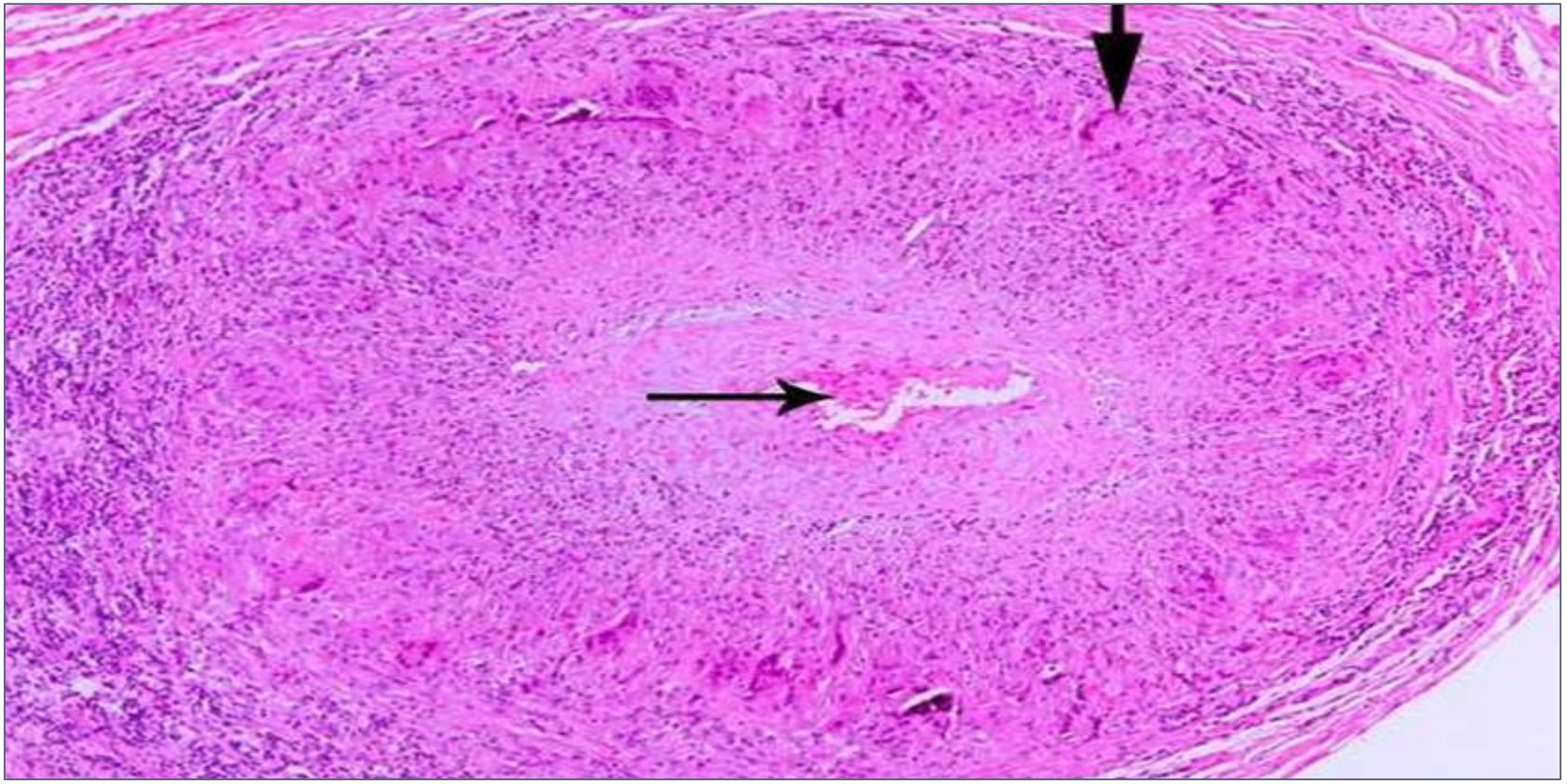
-LARGE TO SMALL-SIZED ARTERIES THAT AFFECTS PRINCIPALLY THE ARTERIES IN THE HEAD—ESPECIALLY THE TEMPORAL ARTERIES

GIANT CELL / TEMPORAL ARTERITIS



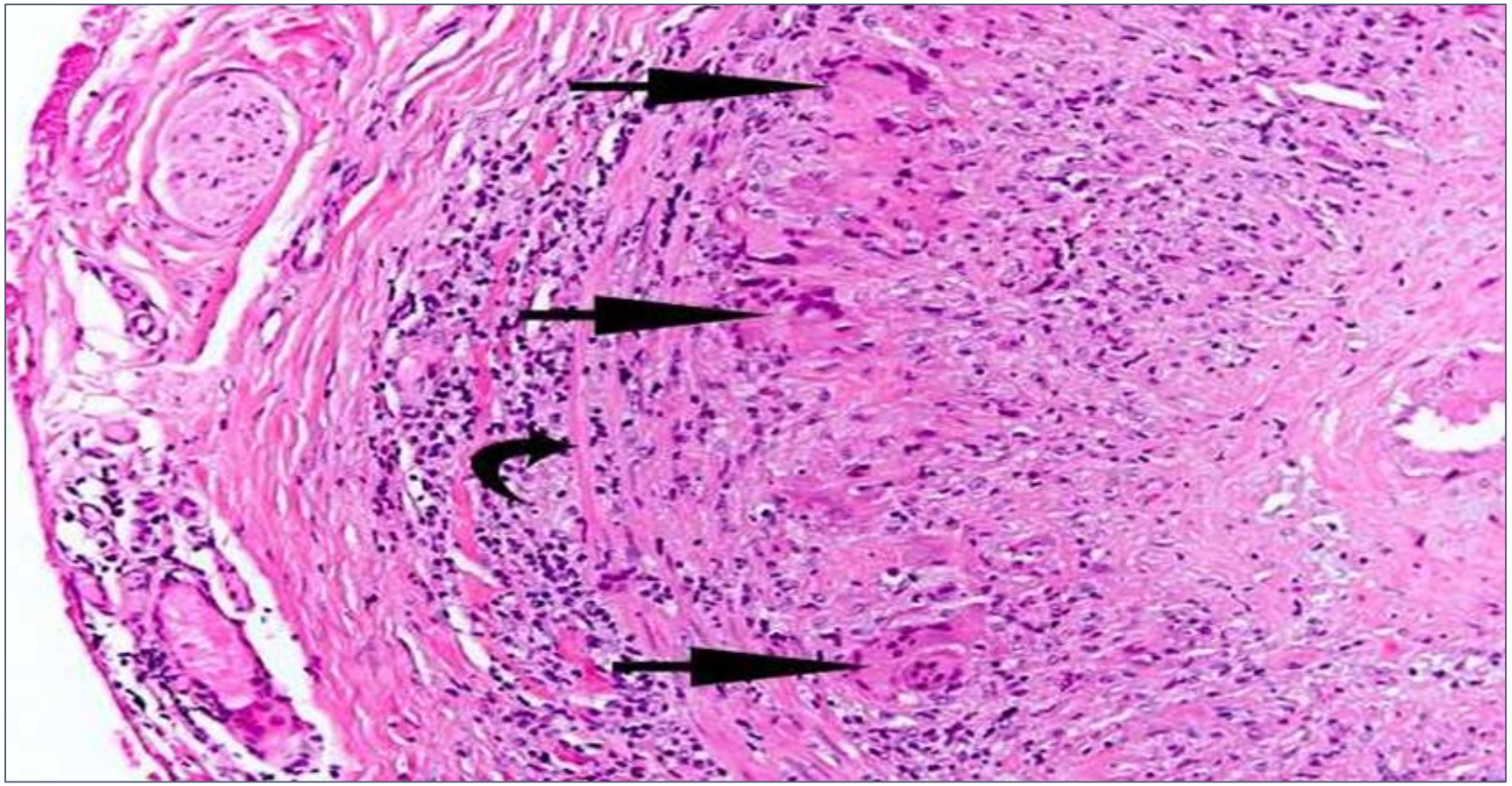
Tender and thickened temporal artery

GIANT CELL / TEMPORAL ARTERITIS - LPF



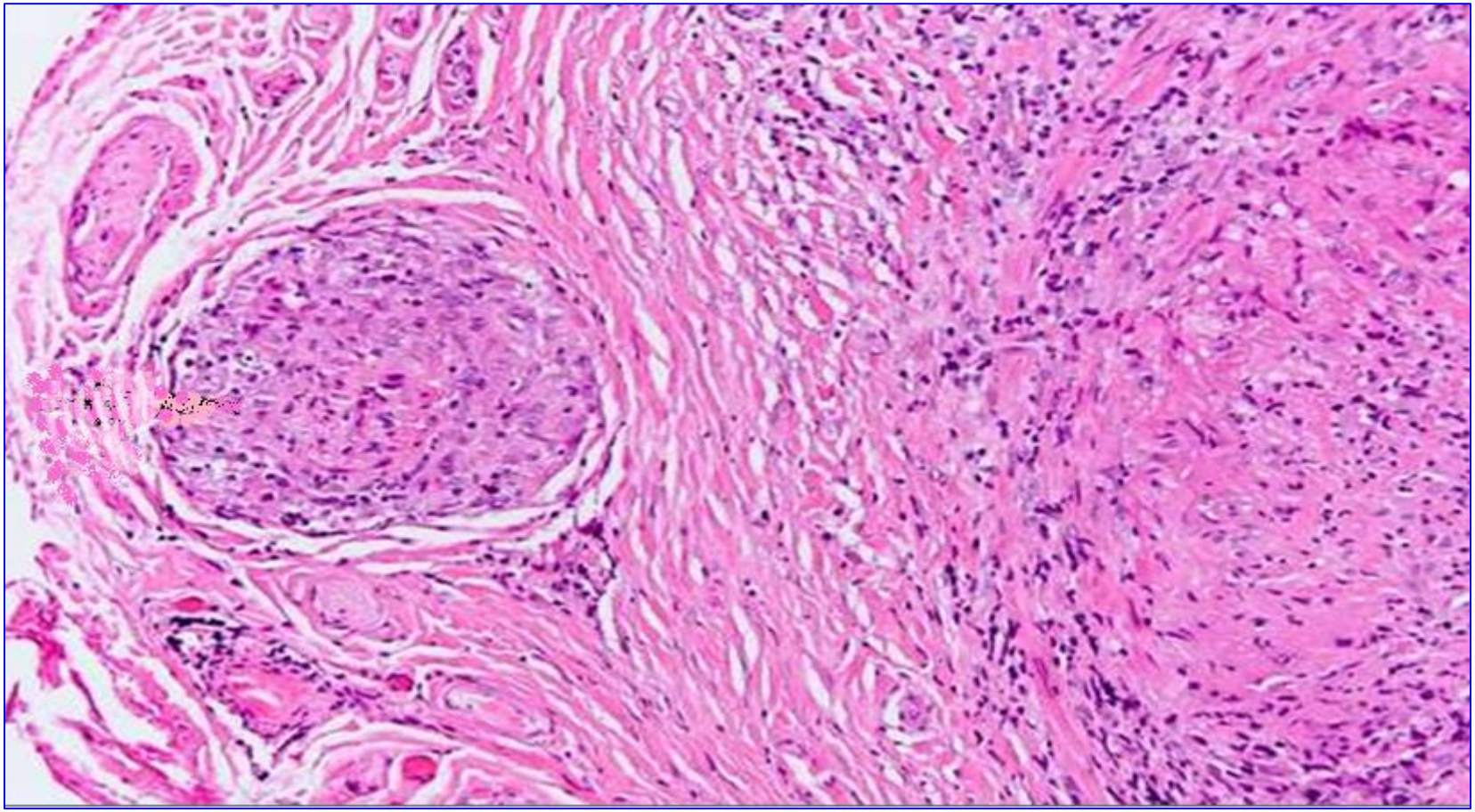
Circumferential involvement of the vascular media is present (vertical arrow pointing downward). Also note the presence of chronic lymphocytic inflammation in the media and adventitia. Reactive intimal fibroplasias lead to luminal stenosis with <10% of its original luminal diameter (thin arrow in the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

GIANT CELL / TEMPORAL ARTERITIS - HPF



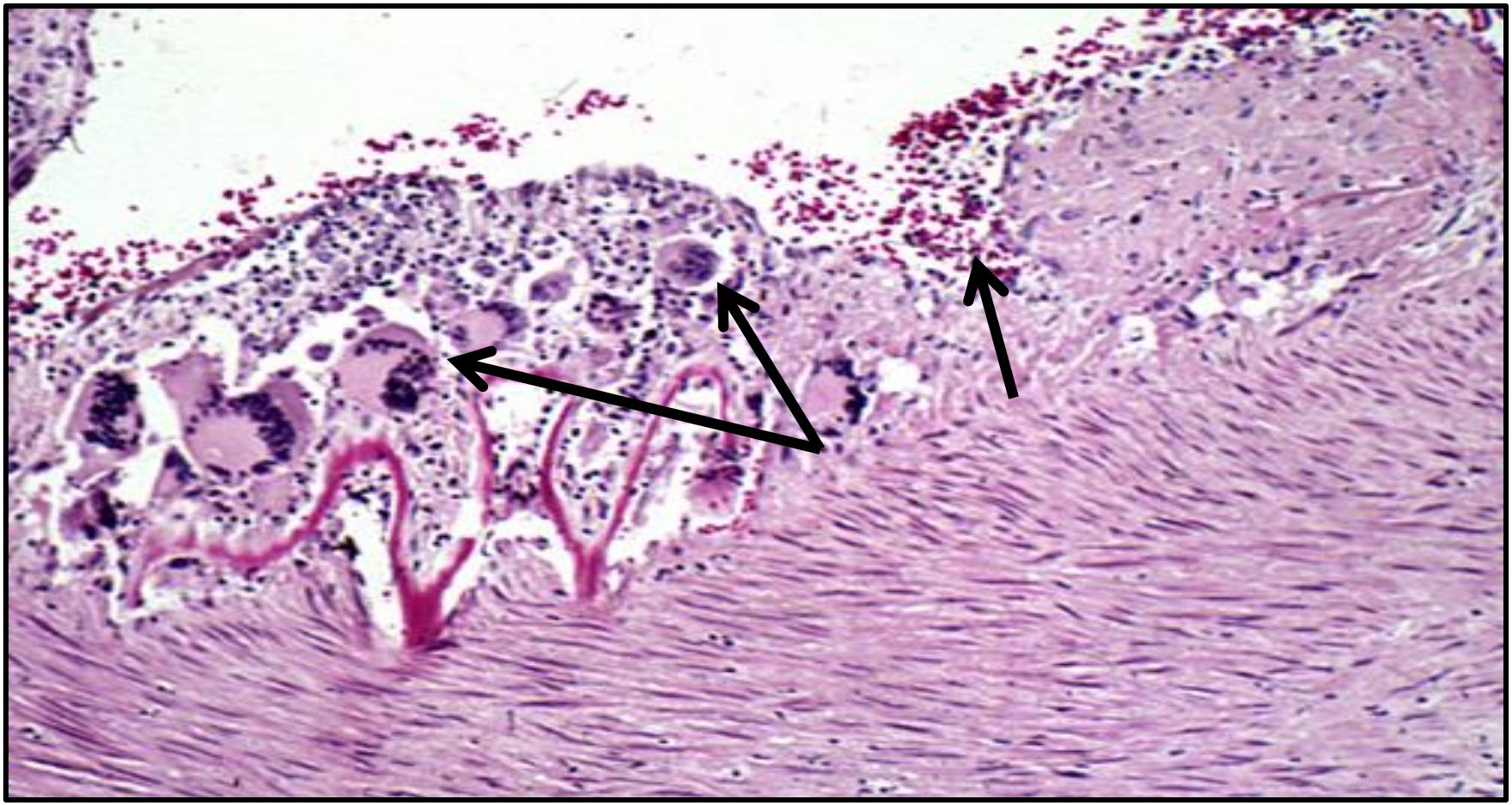
Giant cells can be of Langhans type or foreign-body type (three arrows) and may show fragments of disrupted internal elastic lamina. Note the presence of dense chronic lymphocytic inflammation traversing through circumferential smooth muscle fibers (curved arrow) of vascular media.

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



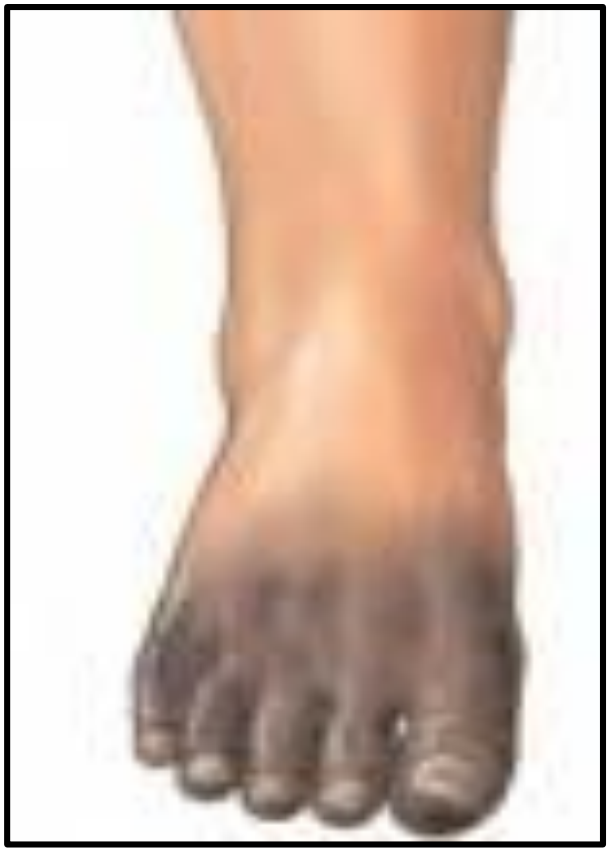
Disruptions of the elastic lamina with inflammation and giant cells.

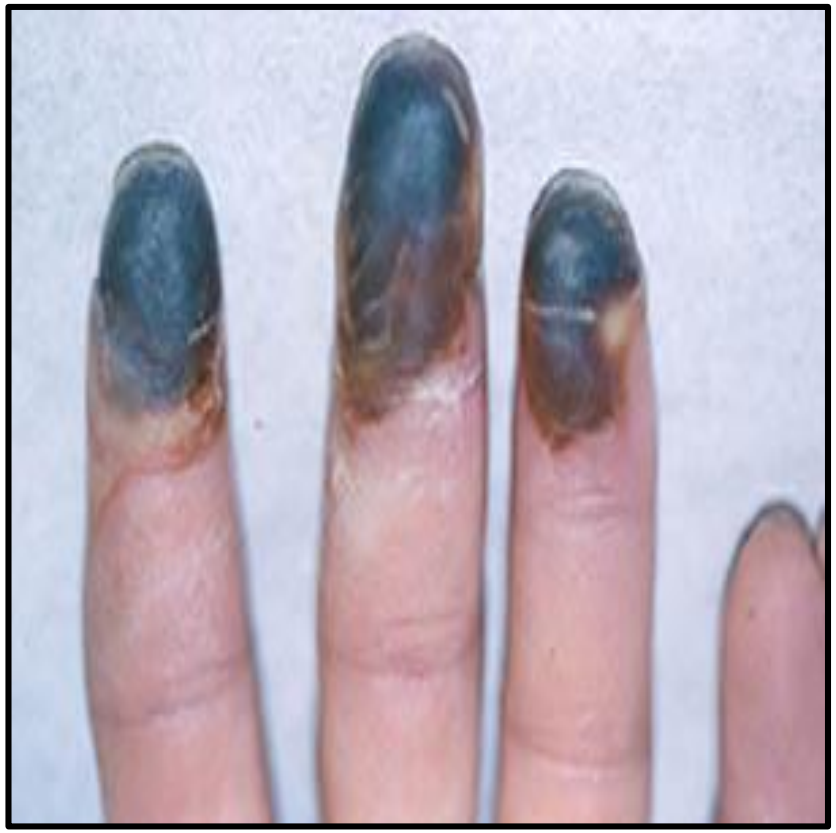
Segmental inflammatory lesions with intimal thickening , medial granulomatous inflammation with giant cells and chronic inflammatory cells and internal elastic lamina fragmentation

Thromboangitis oblitrans (Buerger's disease)

is a distinctive disease that often leads to vascular insufficiency; it is characterized by *segmental, thrombosing, acute and chronic inflammation of medium-sized and small arteries*, principally the tibial and radial arteries

Thromboangitis oblitrans (Buerger`s disease)

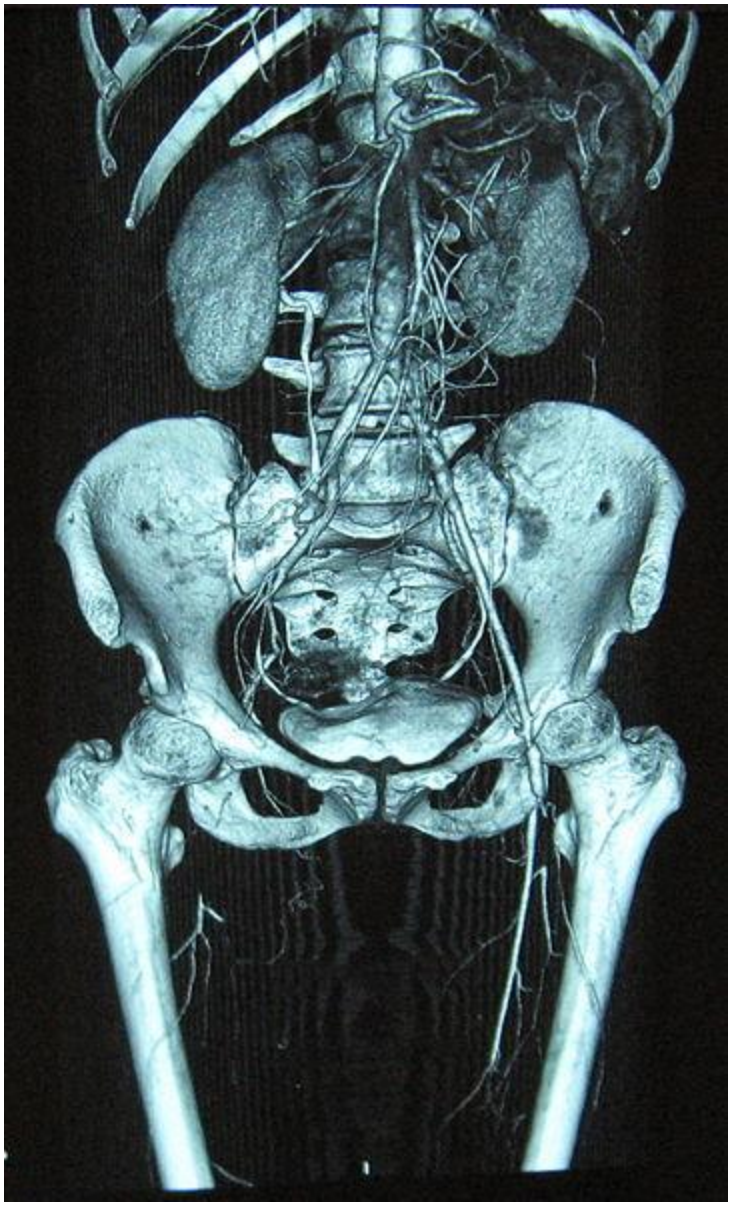




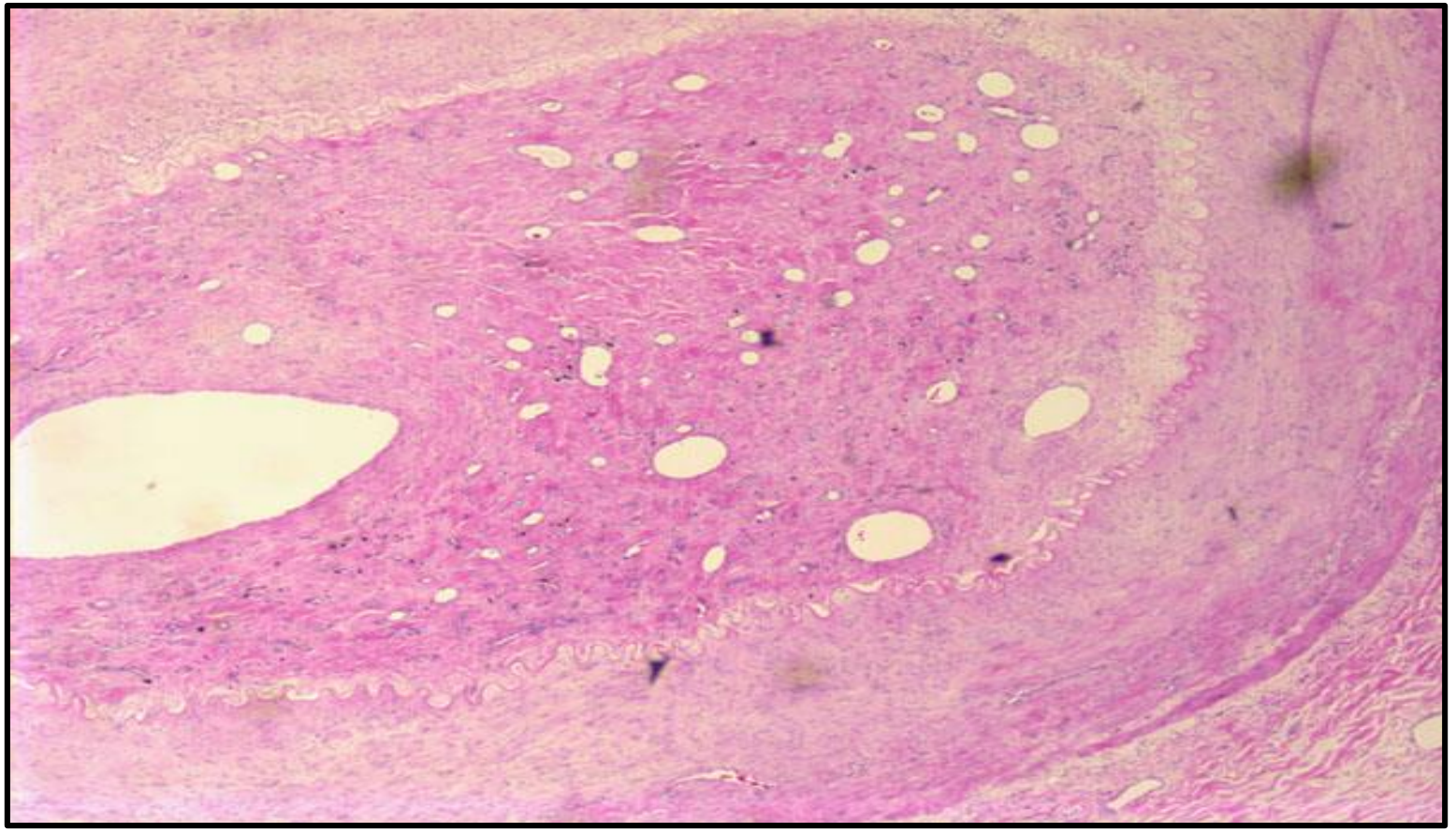
THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE)

- *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*

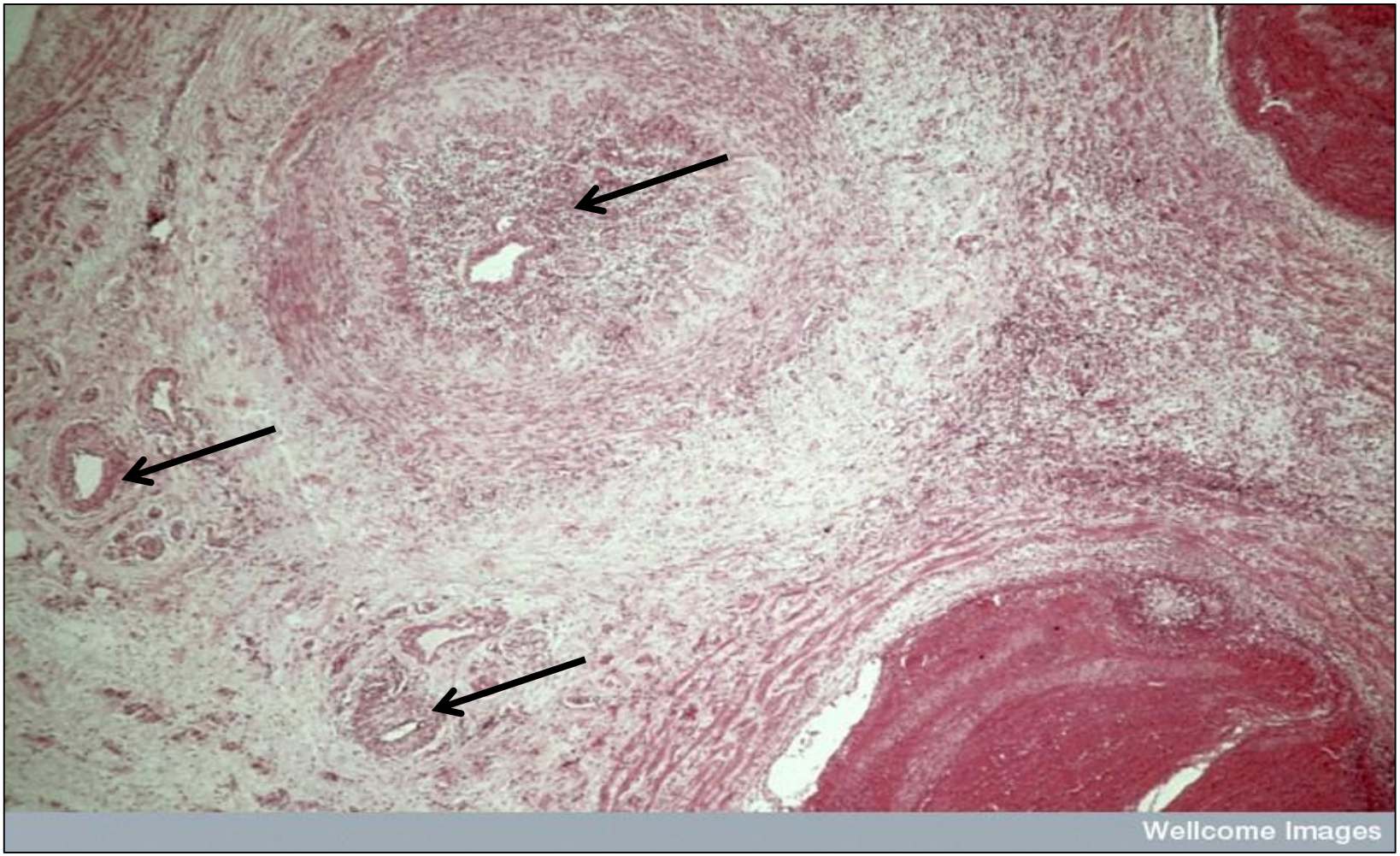


THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE) - LPF



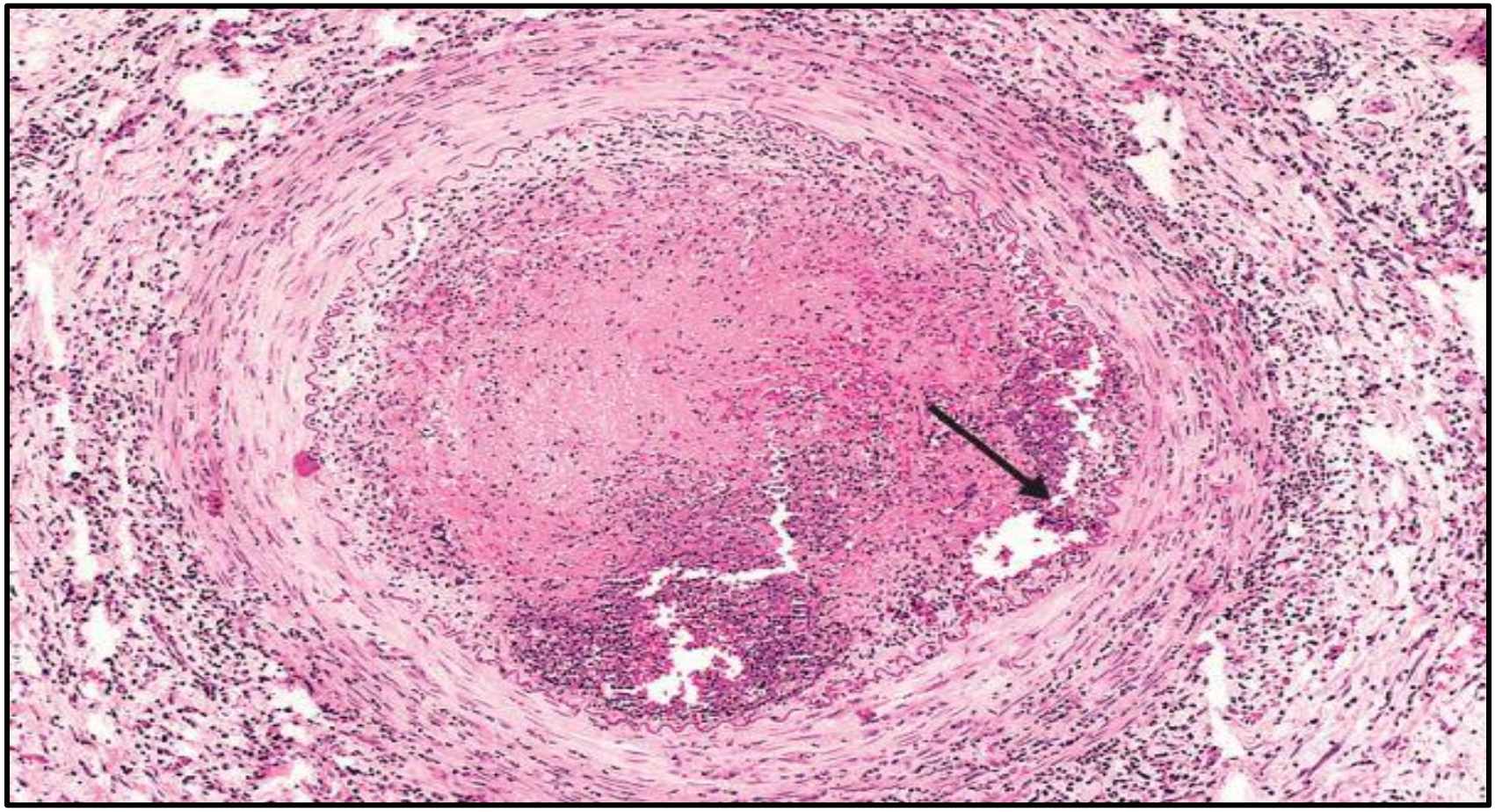
Thromboangiitis obliterans (Buerger's disease) is a non atherosclerotic, segmental, inflammatory, vaso-occlusive disease that affects the small- and medium-sized arteries and veins of the upper and lower extremities.

THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE)- LPF



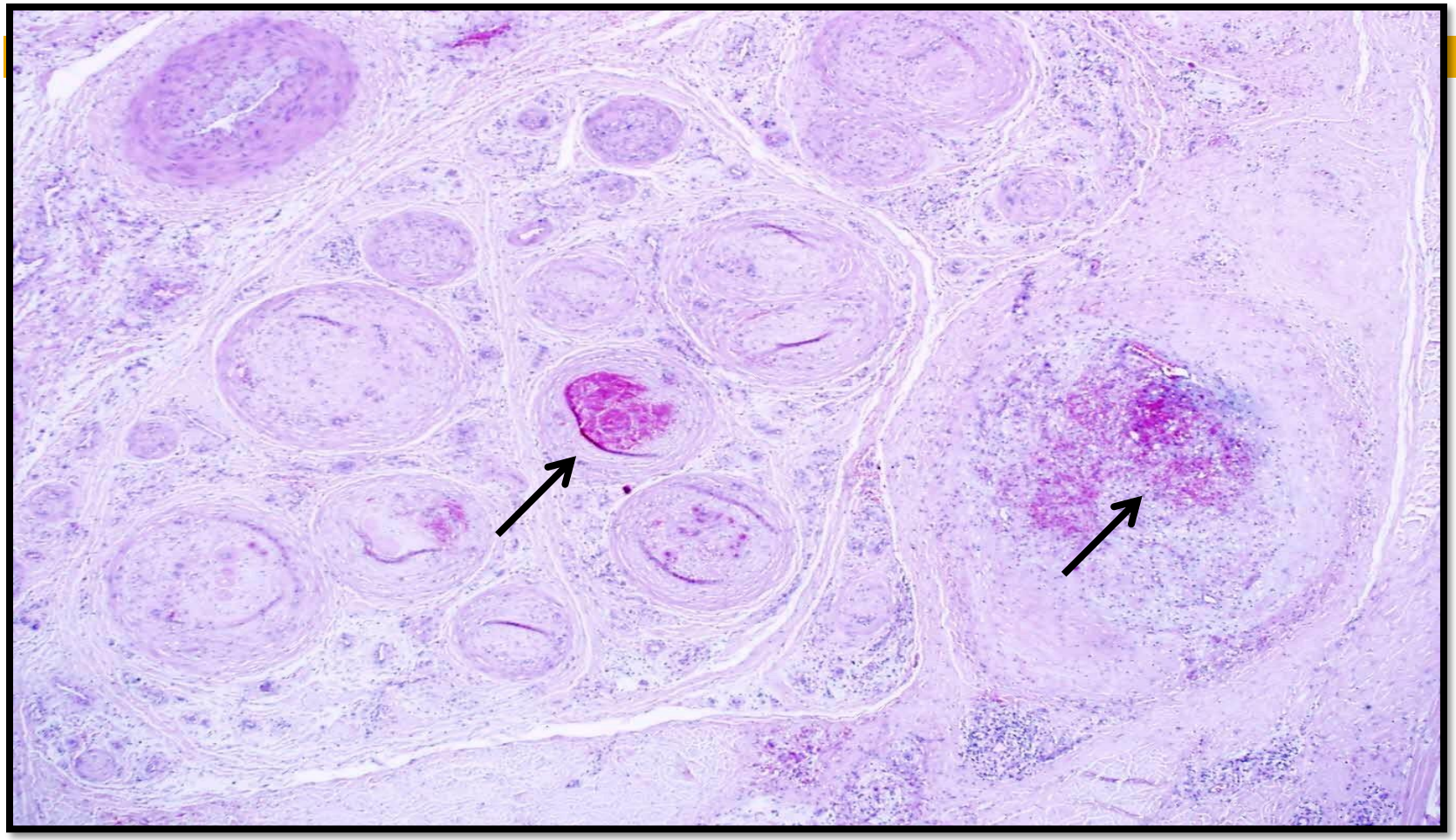
Large number of small blood vessels in the dermis show occlusive organized thrombi with recanalization and fibrosis around blood vessels.

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.

THROMBOANGITIS OBLITERANS (BUERGER'S DISEASE)- HPF

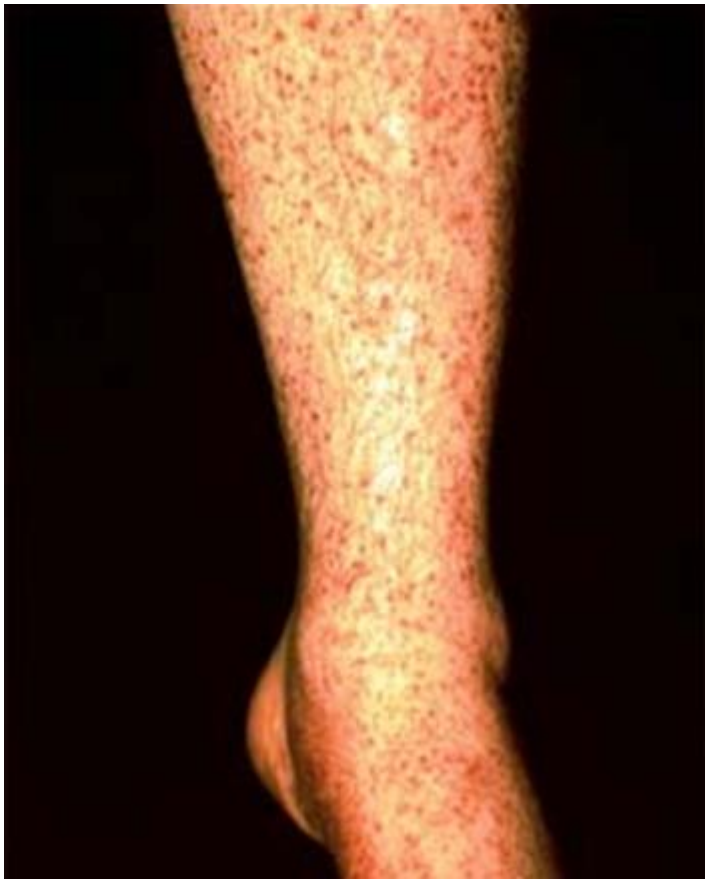


Some blood vessels show recent organizing thrombi while others show infiltration of the wall and surrounding tissue by chronic inflammatory cells

LEUKOCYTOCLASTIC / HYPERSENSITIVITY VASCULITIS
(MICROSCOPIC POLYANGITIS)

is *a necrotizing vasculitis that generally affects capillaries, as well as arterioles and venules*

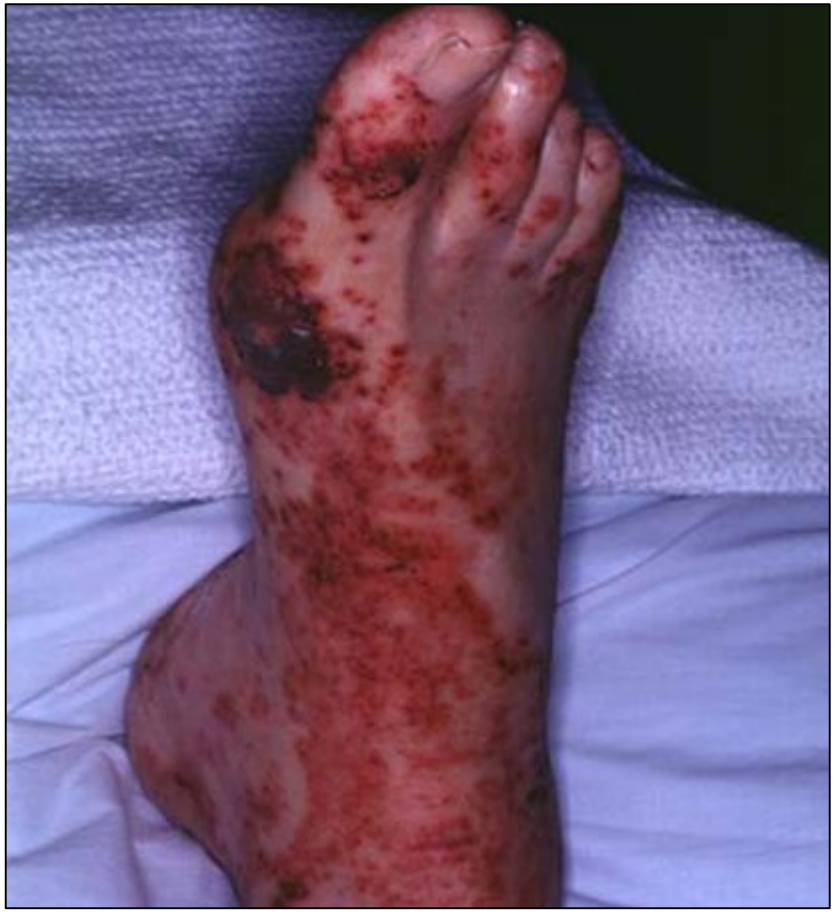
Hypersensitivity vasculitis – Clinical sign



- New rash over large areas
- Purple-colored spots and patches on the skin

Hypersensitivity vasculitis might be complicated with glomerulonephritis lead to haematouria, hemoptysis due to pulmonary capillaritis and GIT hemorrhage manifested by abdominal pain

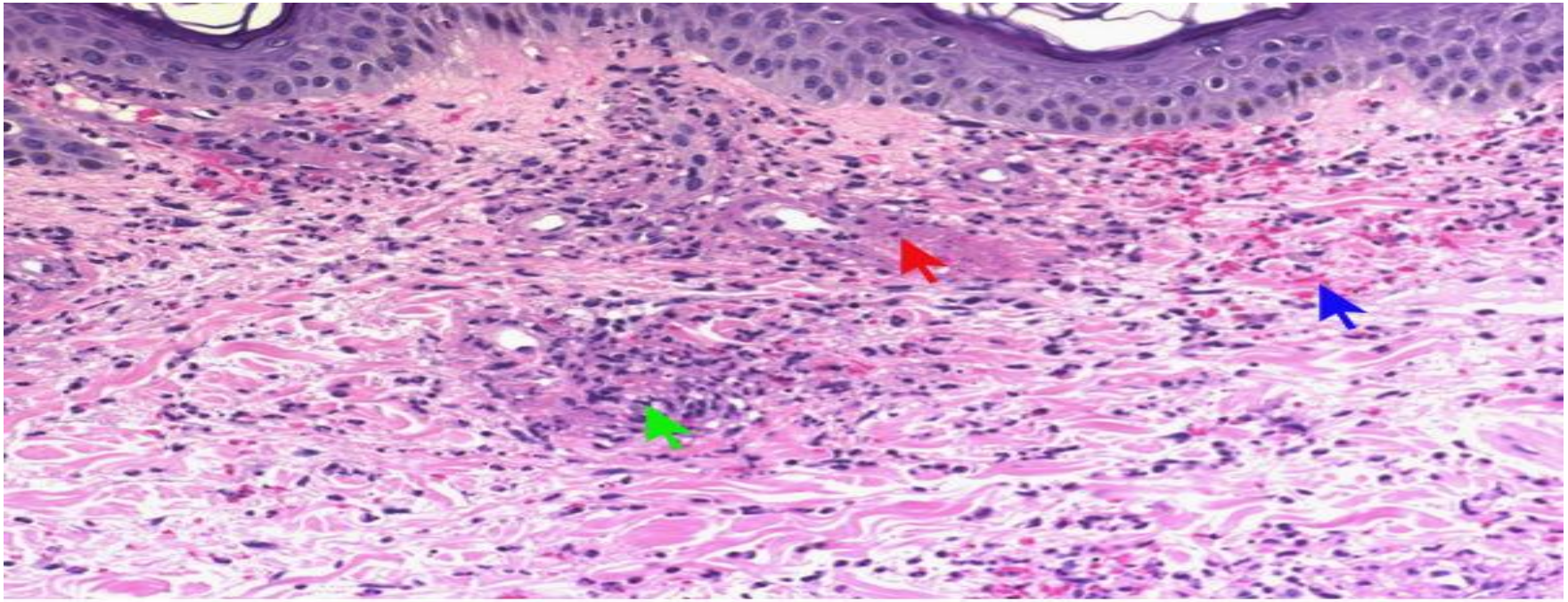
Leukocytoclastic vasculitis - Clinical sign



Leukocytoclastic vasculitis

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

Leukocytoclastic vasculitis - HPF



Fibrinoid type necrosis

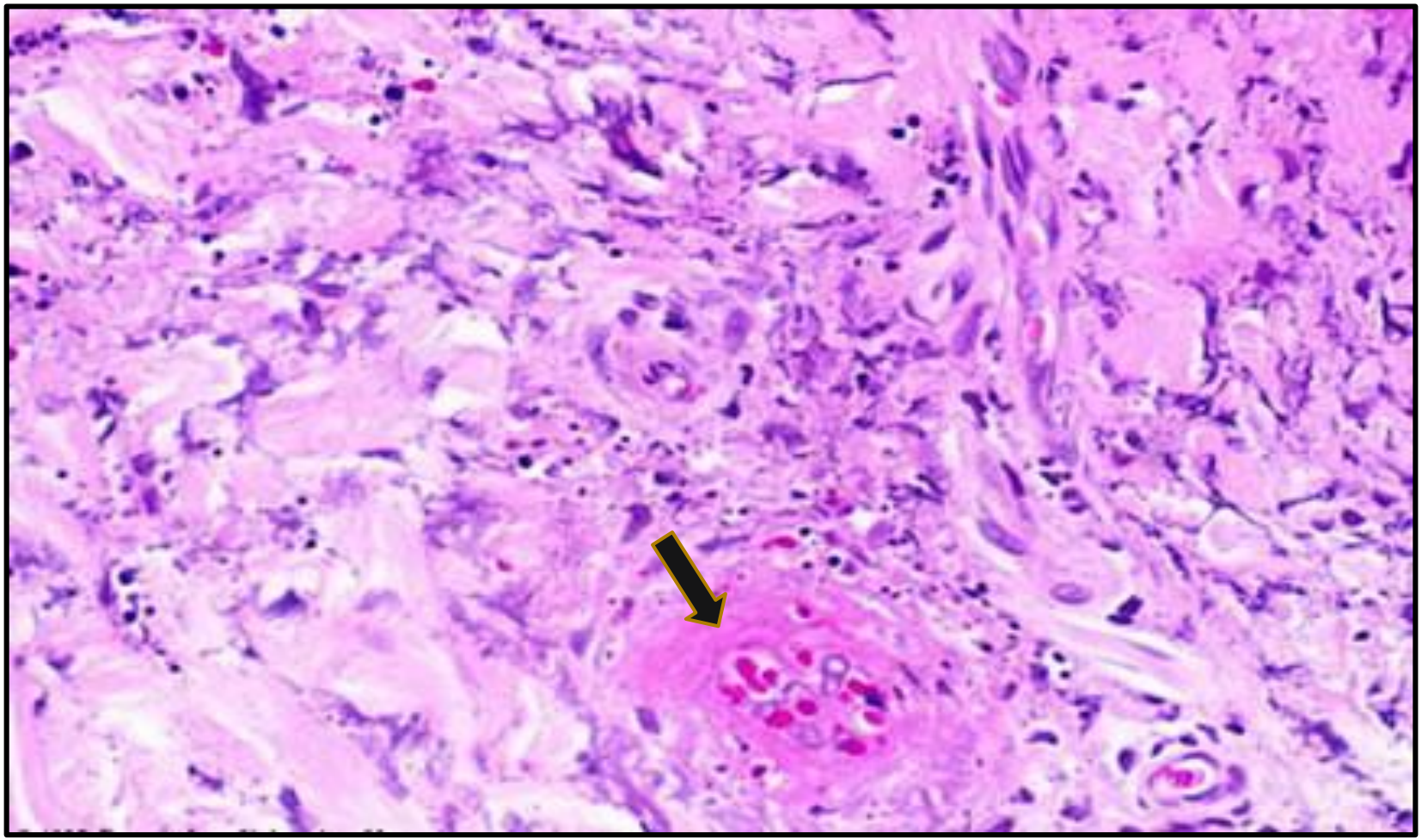
Red cell extravasation

Inflammation

Vasculitis, leukocytoclasis (high power)

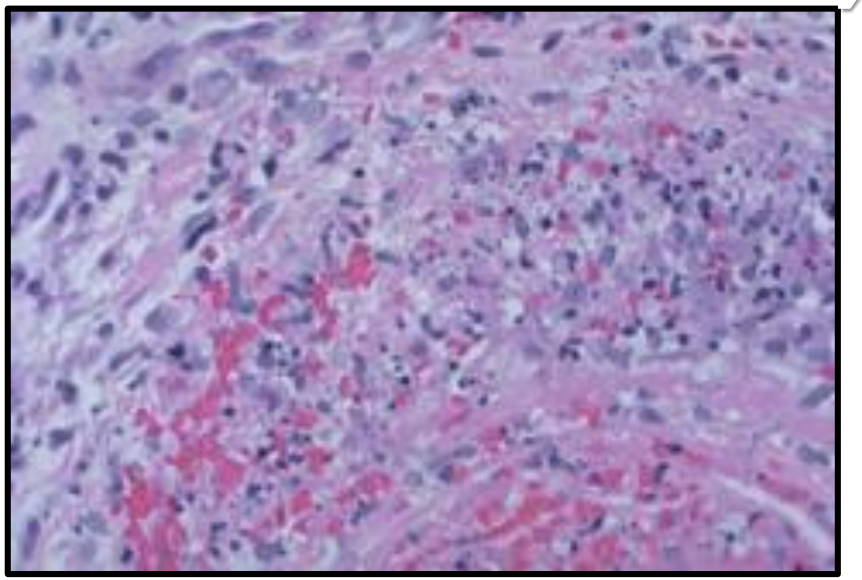
Section of the skin shows fibrinoid necrosis of blood vessels with extravasation of RBCs , neutrophilic infiltration with debris (leukocytoclasis /nuclear dust)

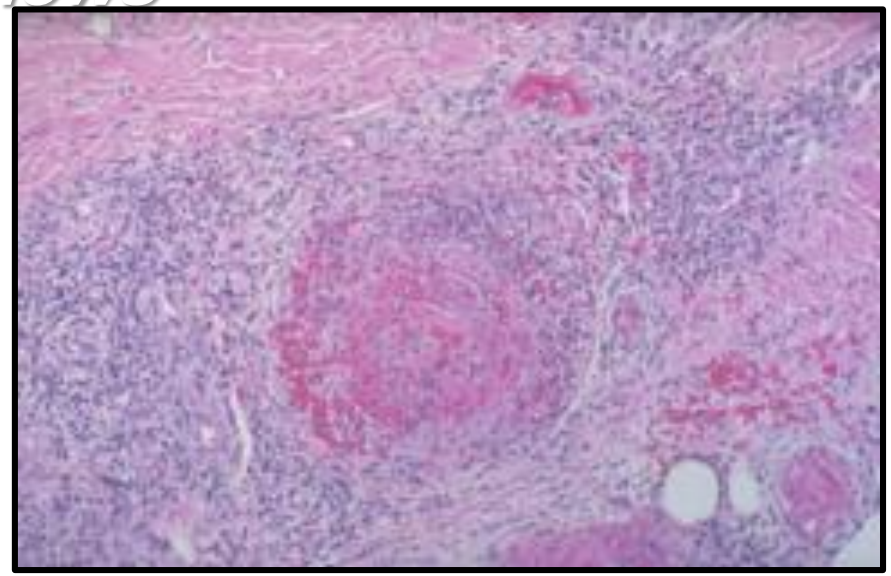
Leukocytoclastic vasculitis - HPF

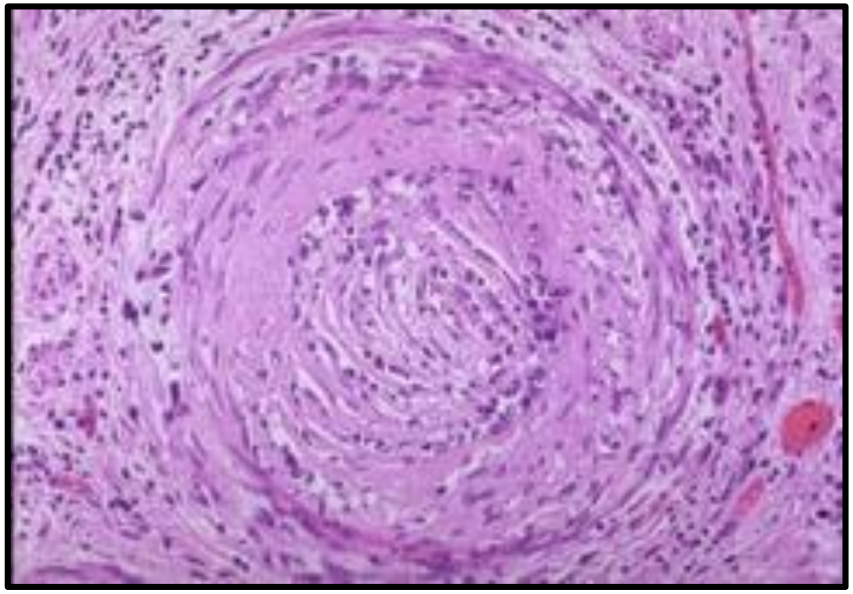


Fibrinoid necrosis of small dermal vessels is present, necessary to establish the diagnosis of leukocytoclastic vasculitis.

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

ANEURYSM OF ABDOMINAL AORTA

An *aneurysm* is a *localized abnormal dilation of a blood vessel or the heart*; it can be congenital or acquired.

The two most important disorders that predispose to aortic aneurysms are atherosclerosis and hypertension.

Types of Aneurysms

Saccular Aneurysm

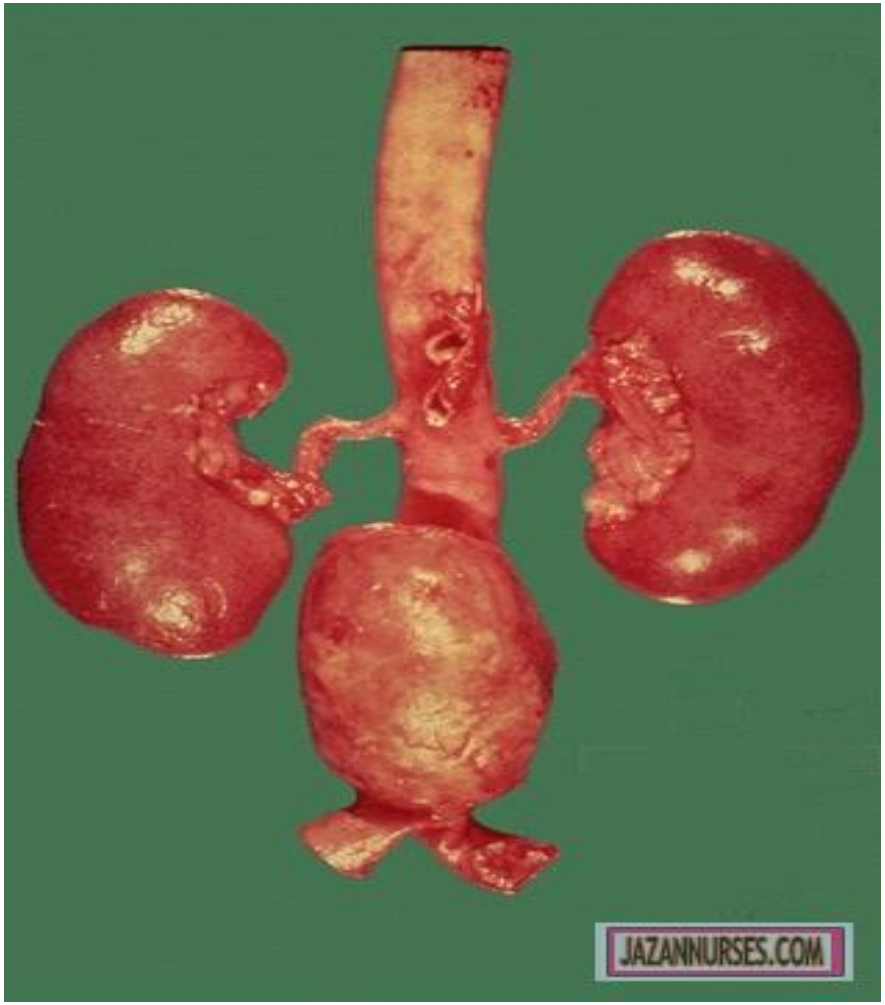
Fusiform Aneurysm

Ruptured Aneursym

Other conditions that weaken vessel walls

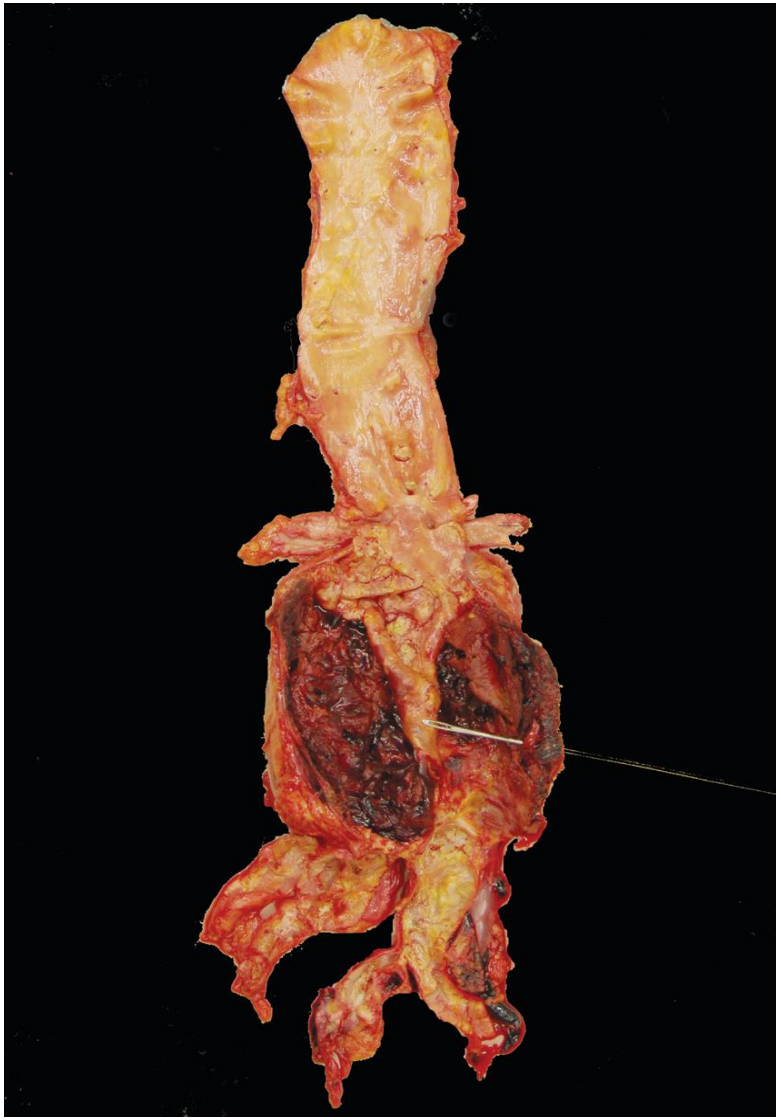
- trauma,
- vasculitis
- congenital defects
  (e.g., *berry aneurysms* typically in the circle of Willis)
- infections *(mycotic aneurysms)*
- Tertiary syphilis is now a rare cause of aortic aneurysms (Thoracic aortic aneurysms)

Abdominal Aortic Aneurysm



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

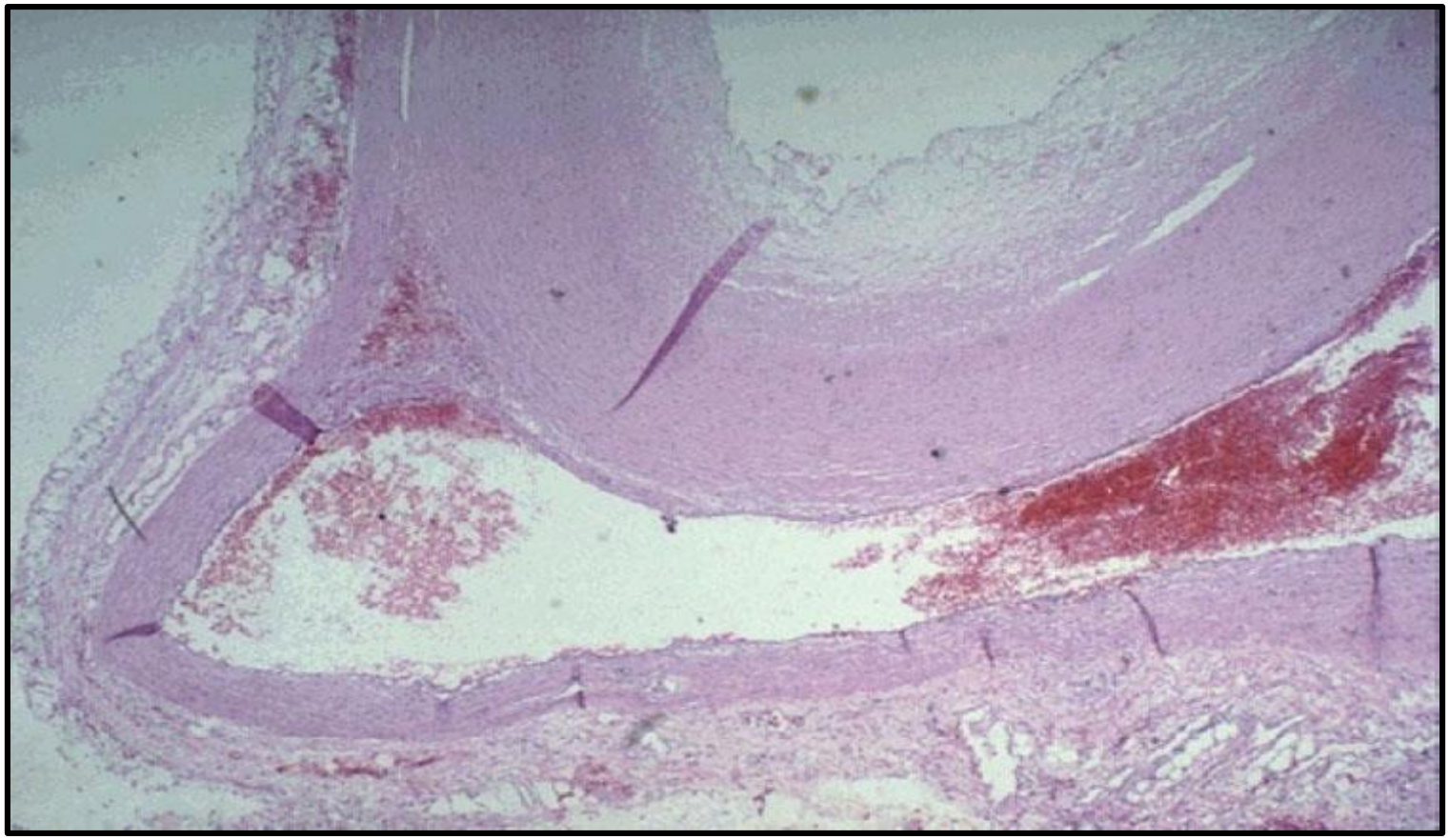
Abdominal Aortic Aneurysm



- *Aneurysmal dilatation* of the abdominal aorta with rupture
- *intraluminal thrombus*
- *extensive aortic atherosclerosis*

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

Dissecting aortic aneurysm - LPF



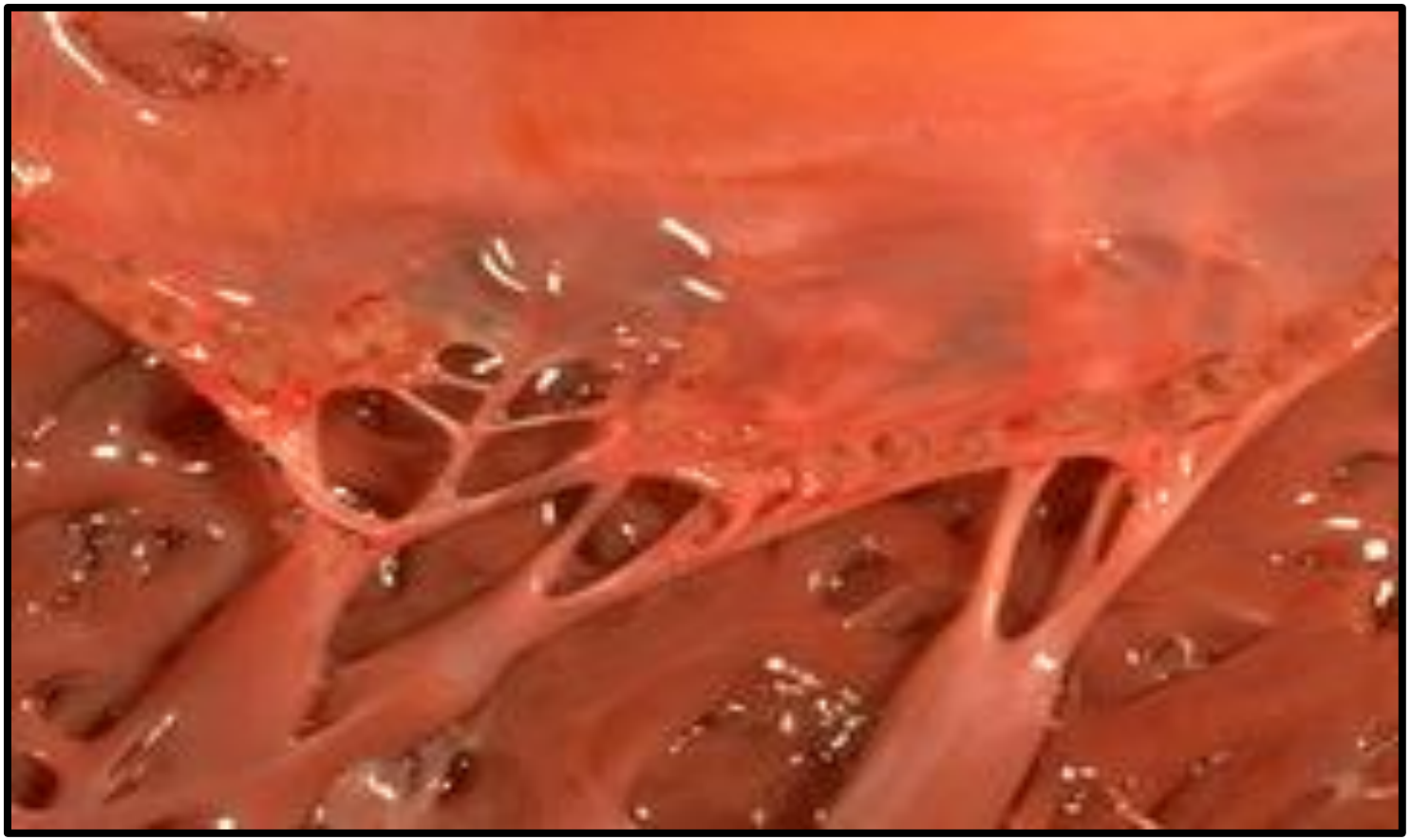
***A dissecting aortic aneurysm** occurs when blood enters the aortic wall through a defect and moves between two layers of the wall, stripping the inner layer from the outer layer. Usually associated with atherosclerosis, inflammation, and degeneration of the connective tissue of the tunica media*

Valvular Heart Disease

Vegetations of rheumatic fever on mitral and aortic valves

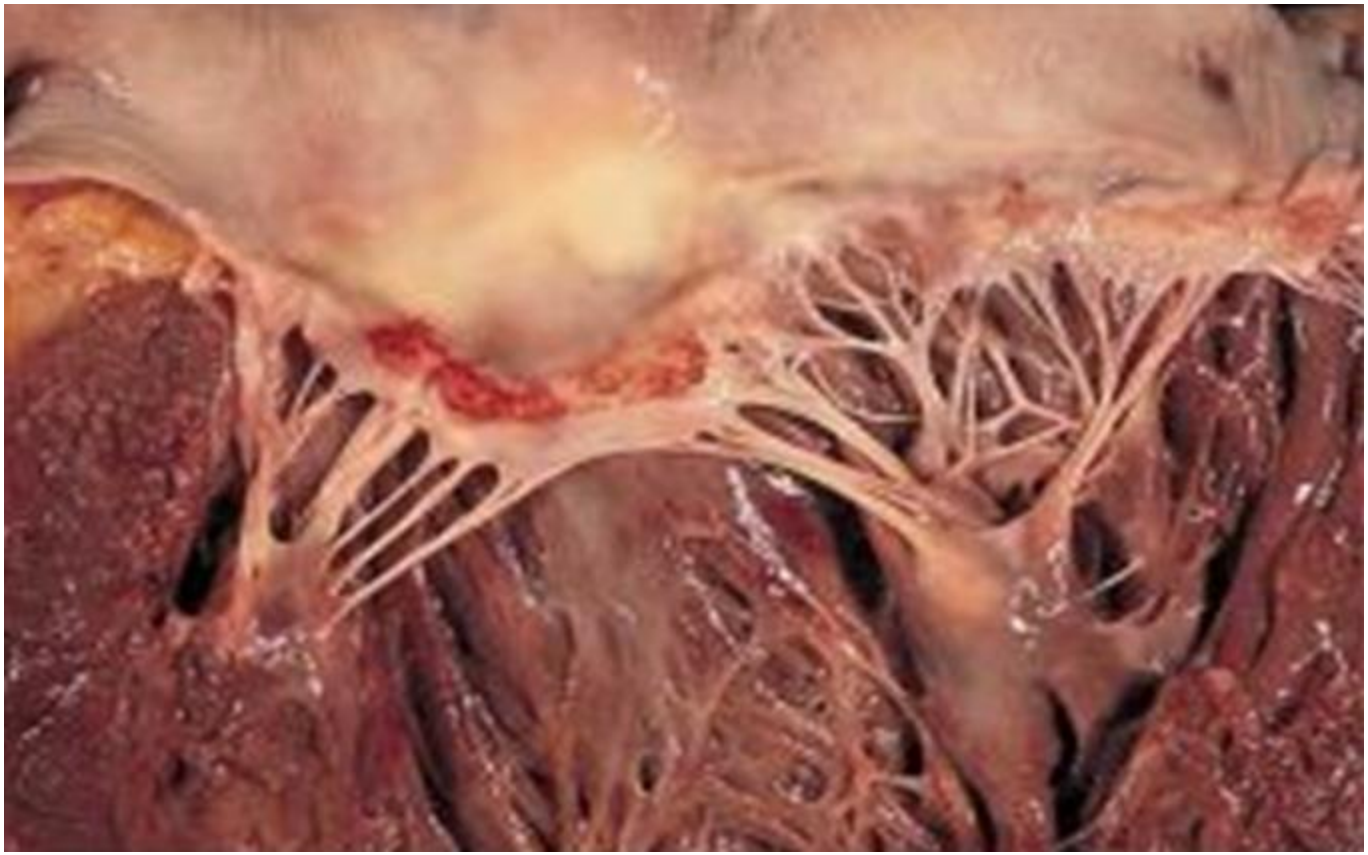
- Rheumatic fever (RF) is an acute, immunologically mediated, multisystem inflammatory disease that occurs a few weeks after an episode of group A streptococcal pharyngitis
- Acute rheumatic carditis is a frequent manifestation during the active phase of RF and may progress over time to chronic rheumatic heart disease (RHD), of which valvular abnormalities are key manifestations.

Acute Rheumatic Mitral Valvulitis - Gross



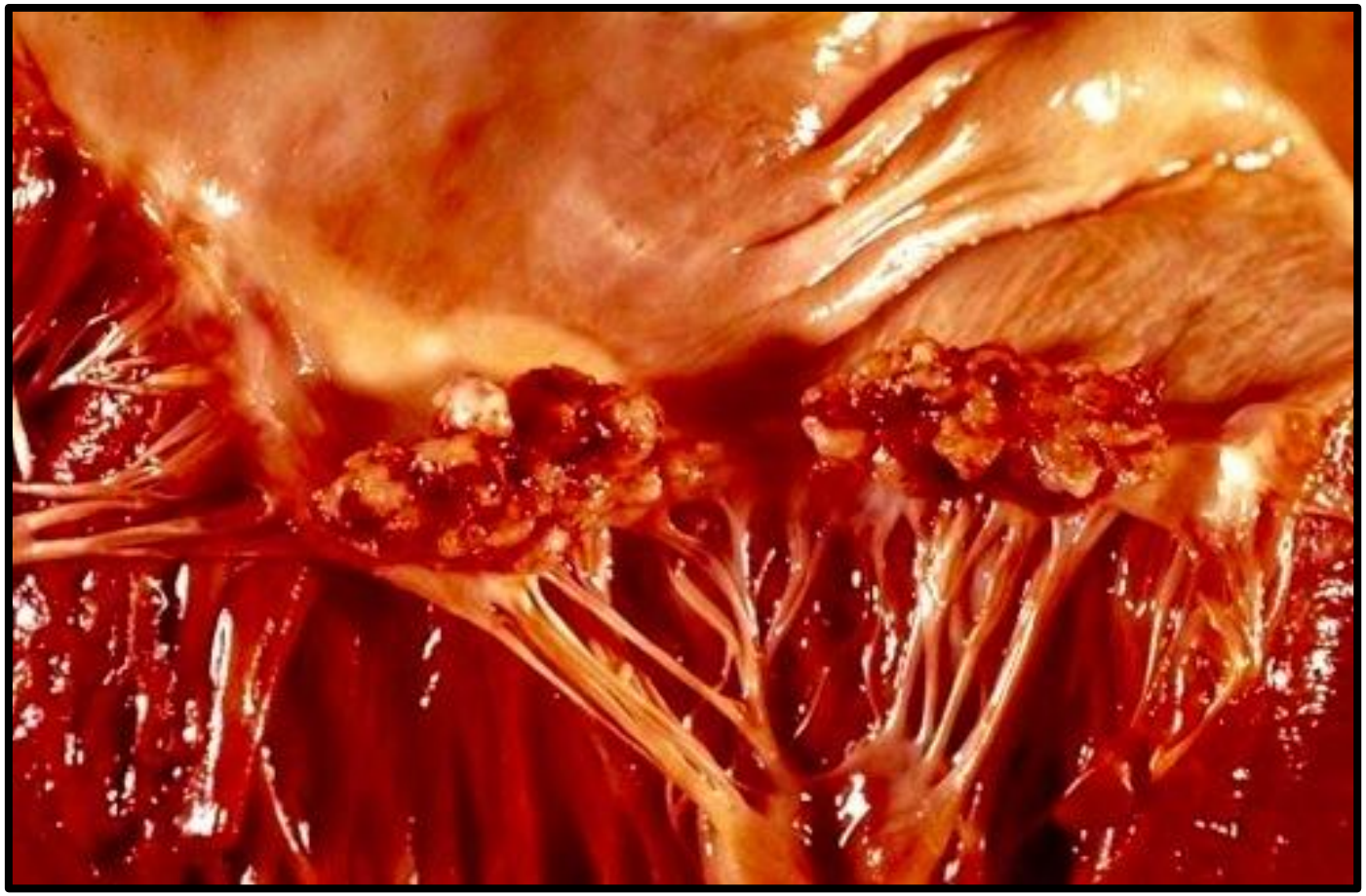
The small verrucous vegetations are associated with acute rheumatic fever. These warty vegetations are multiple, firm, adherent, small , 1-3 mm in- diameter and form along the line of valve closure over areas of endocardial inflammation. Affects mainly Aortic & Mitral valves

Acute Rheumatic Mitral Valvulitis - Gross



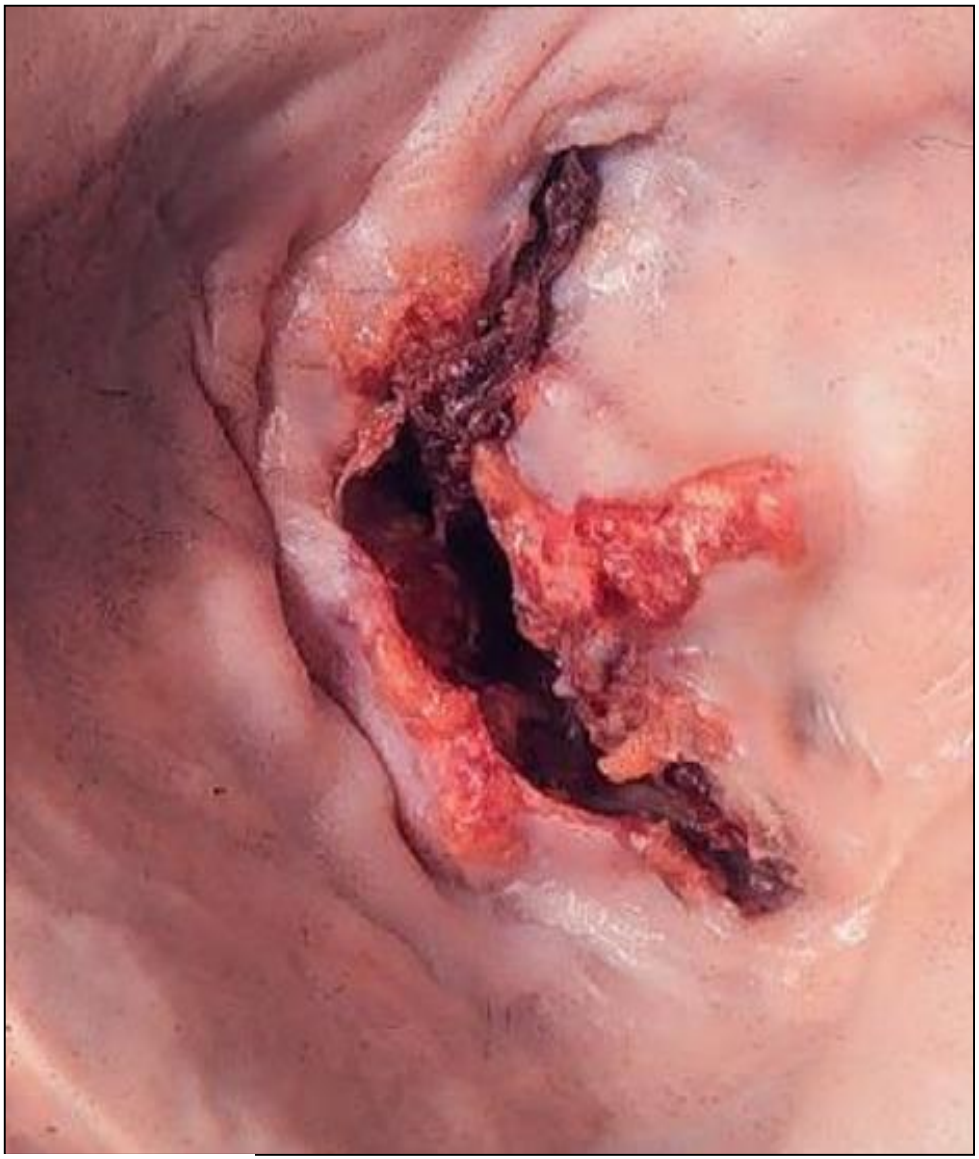
Close-up view of an opened-out rheumatic mitral valve showing severe thickening and retraction of the cusps. The chordae tendineae are shortened and fused into short thick cords. This rigid valve would have been stenosed

Chronic Rheumatic Mitral Valvulitis - Gross



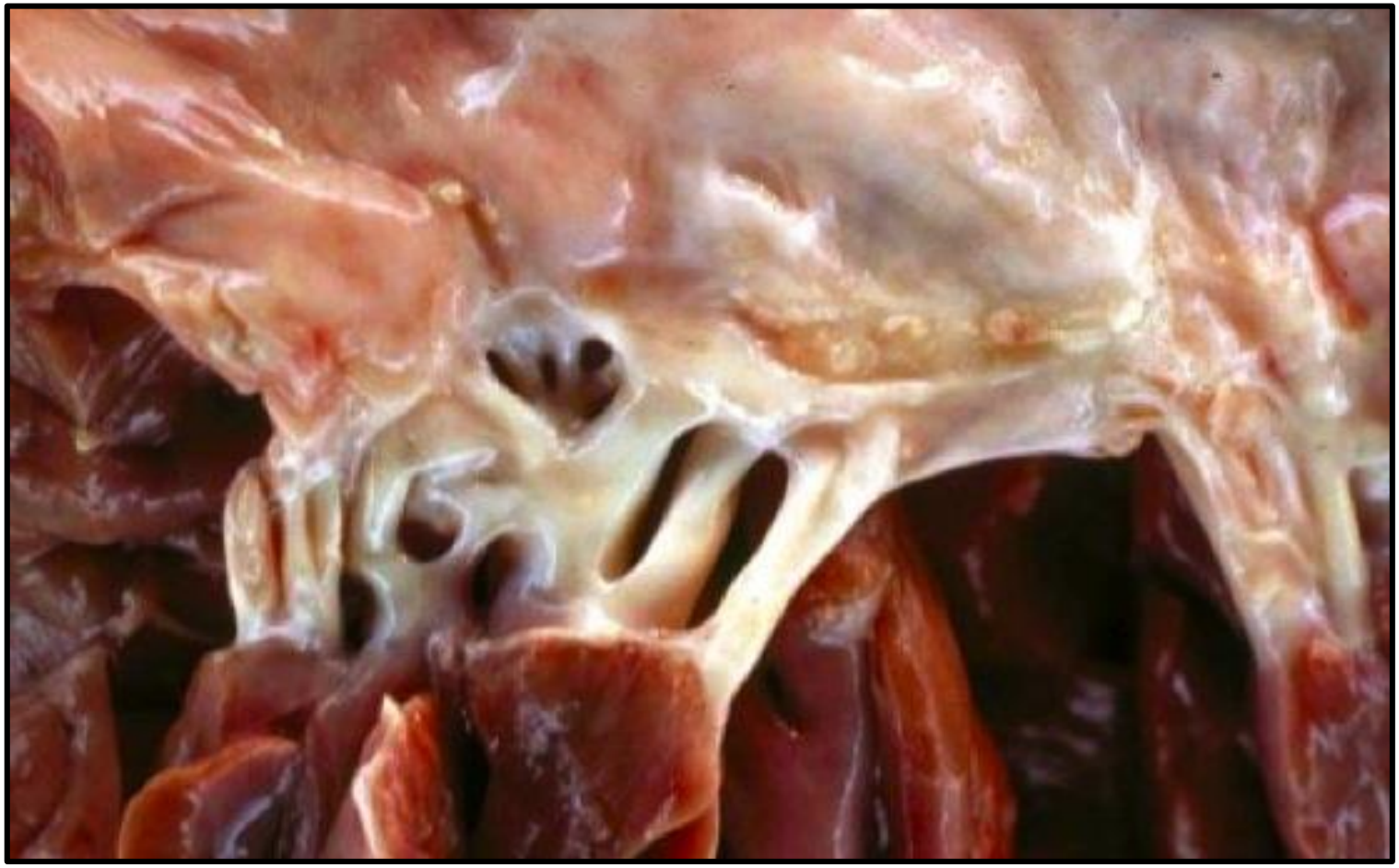
large vegetations/hemorrhage along the free margins of the mitral valve.

Chronic Rheumatic Mitral Valvulitis - Gross



- *Stenotic mitral valve seen from left atrium (Fish-Mouth)*
- *fusion of valve commissures,*
- *thickening and calcification of the cusps,*
- *The vegetations*

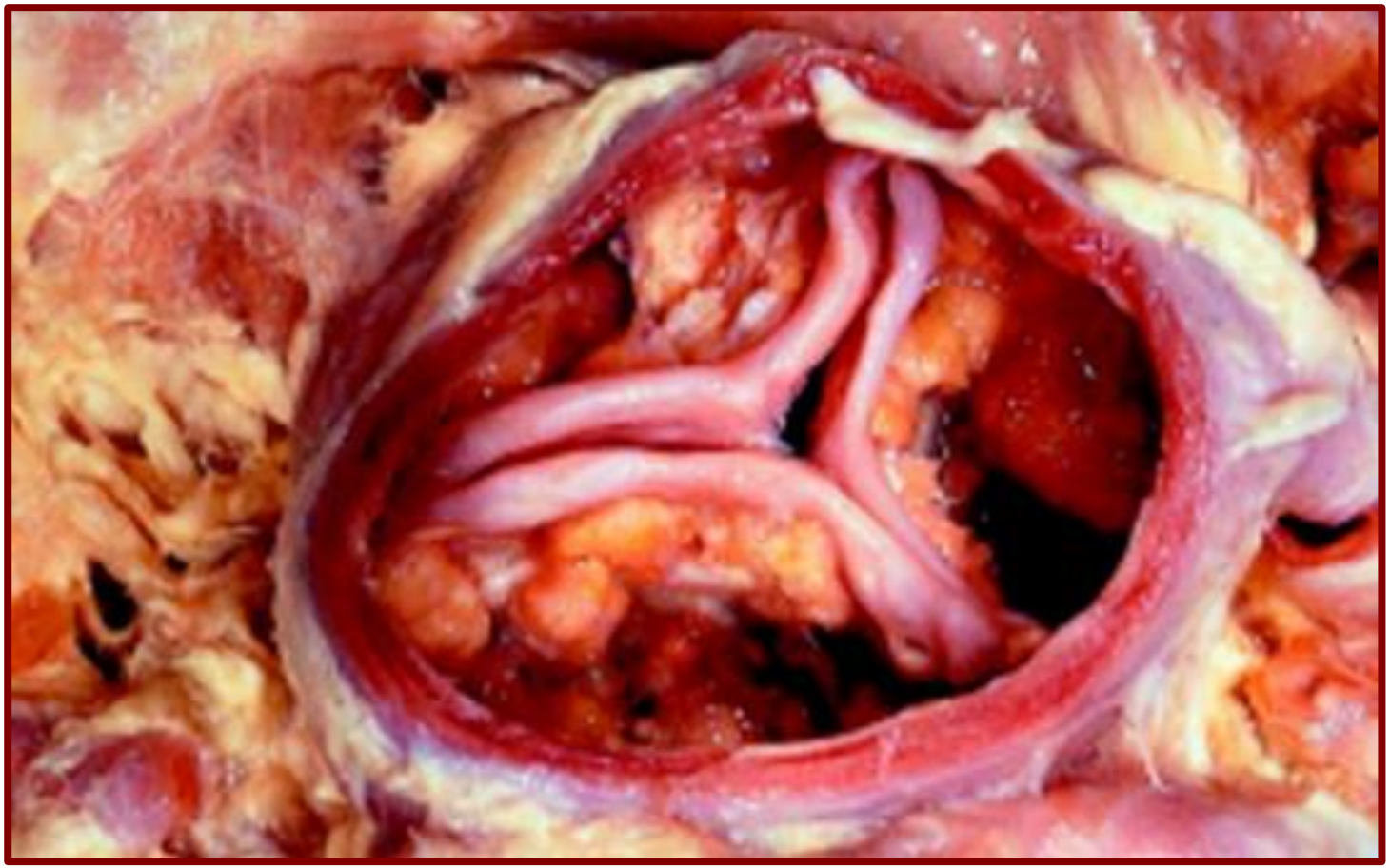
Chronic Rheumatic Mitral Valvulitis - Gross



Chronic rheumatic mitral valvulitis

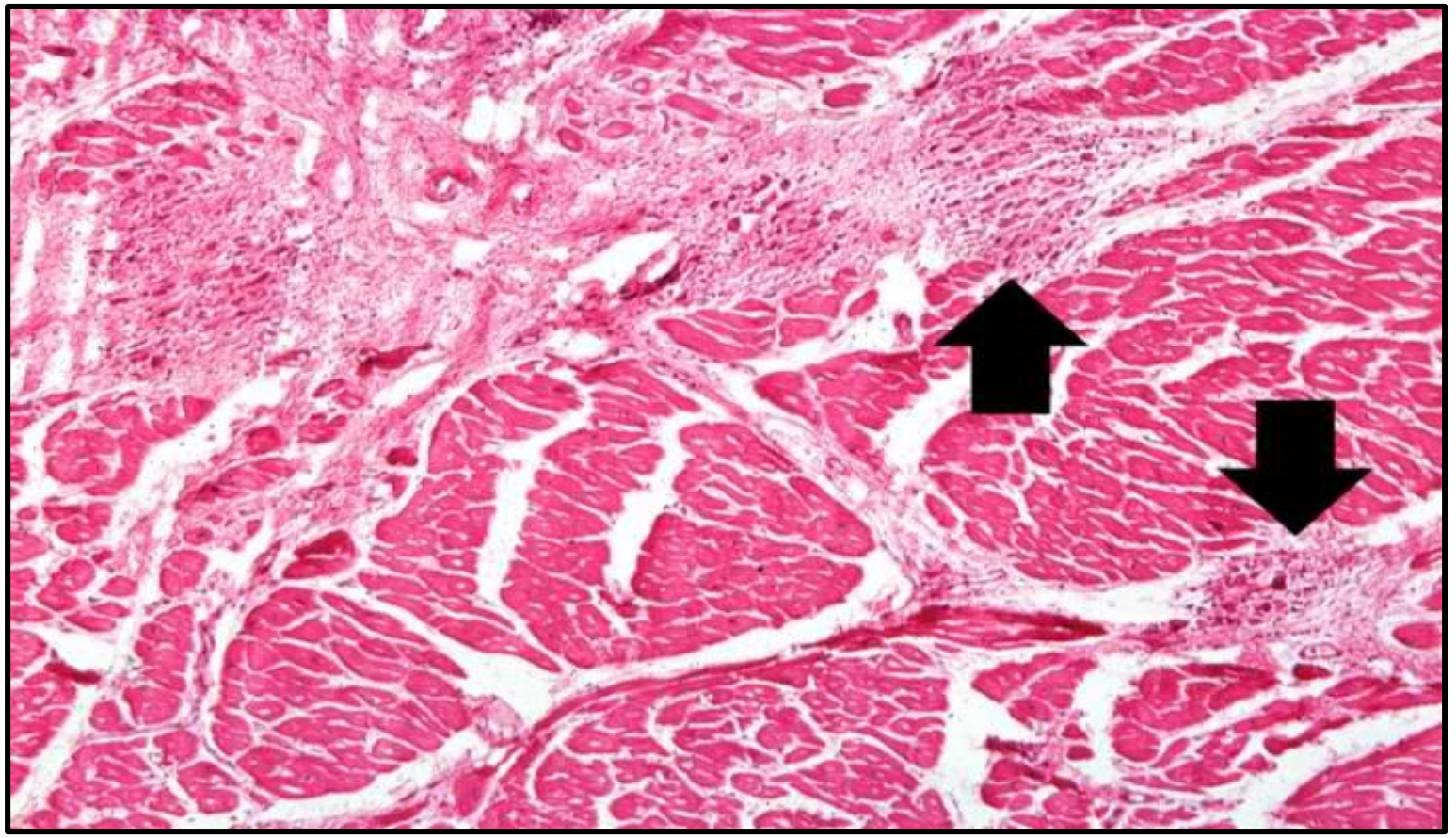
the valve leaflets are thick, fibrotic, fused. Short, thickened, fused chordae tendinae ➡ stenosis and / or incompetence

Rheumatic Aortic Valvulitis - Gross



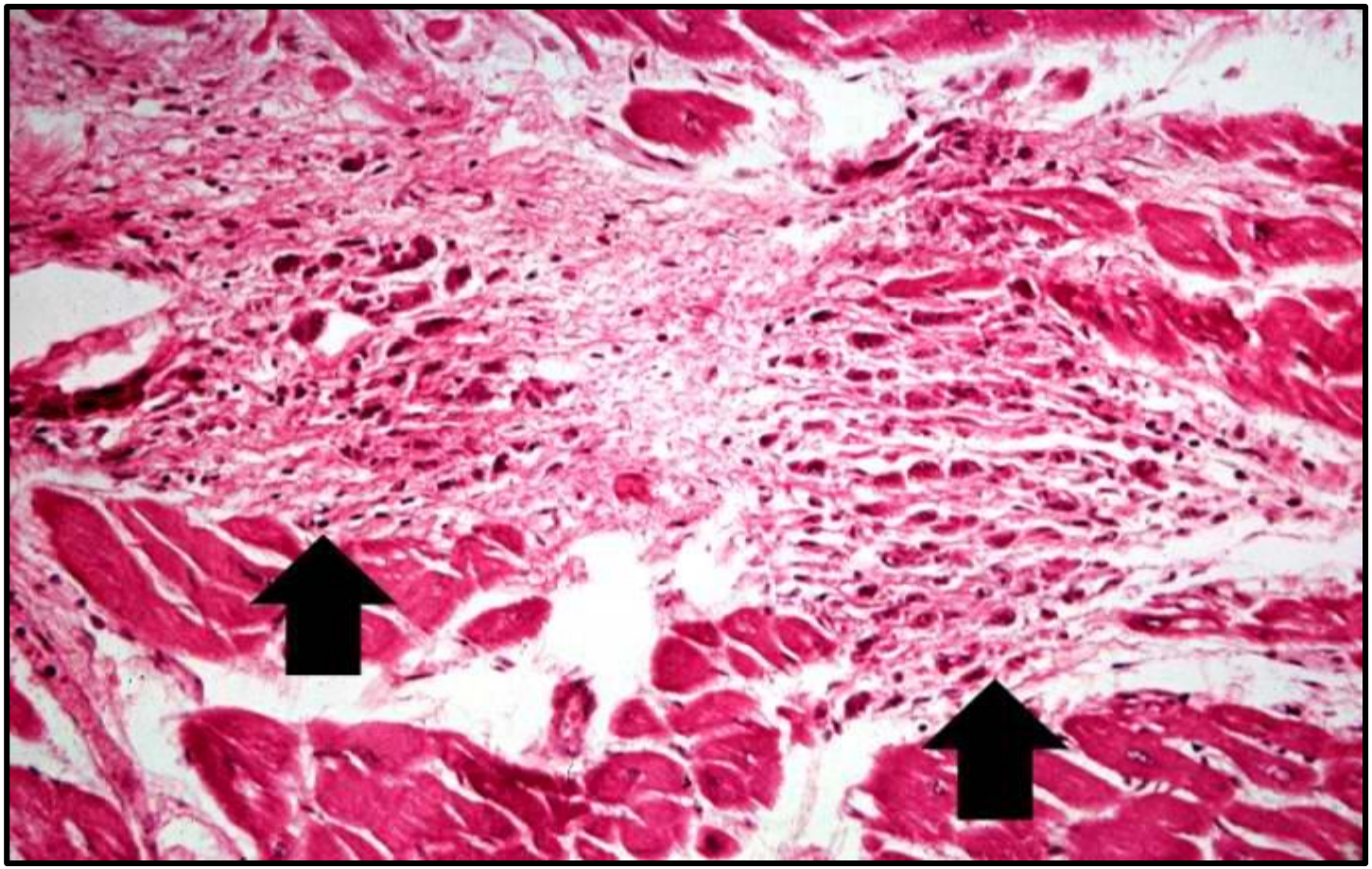
Gross pathology of rheumatic heart disease Aortic stenosis:
Aorta has been removed to show thickened, fused aortic valve leaflets

Rheumatic Valvulitis - LPF



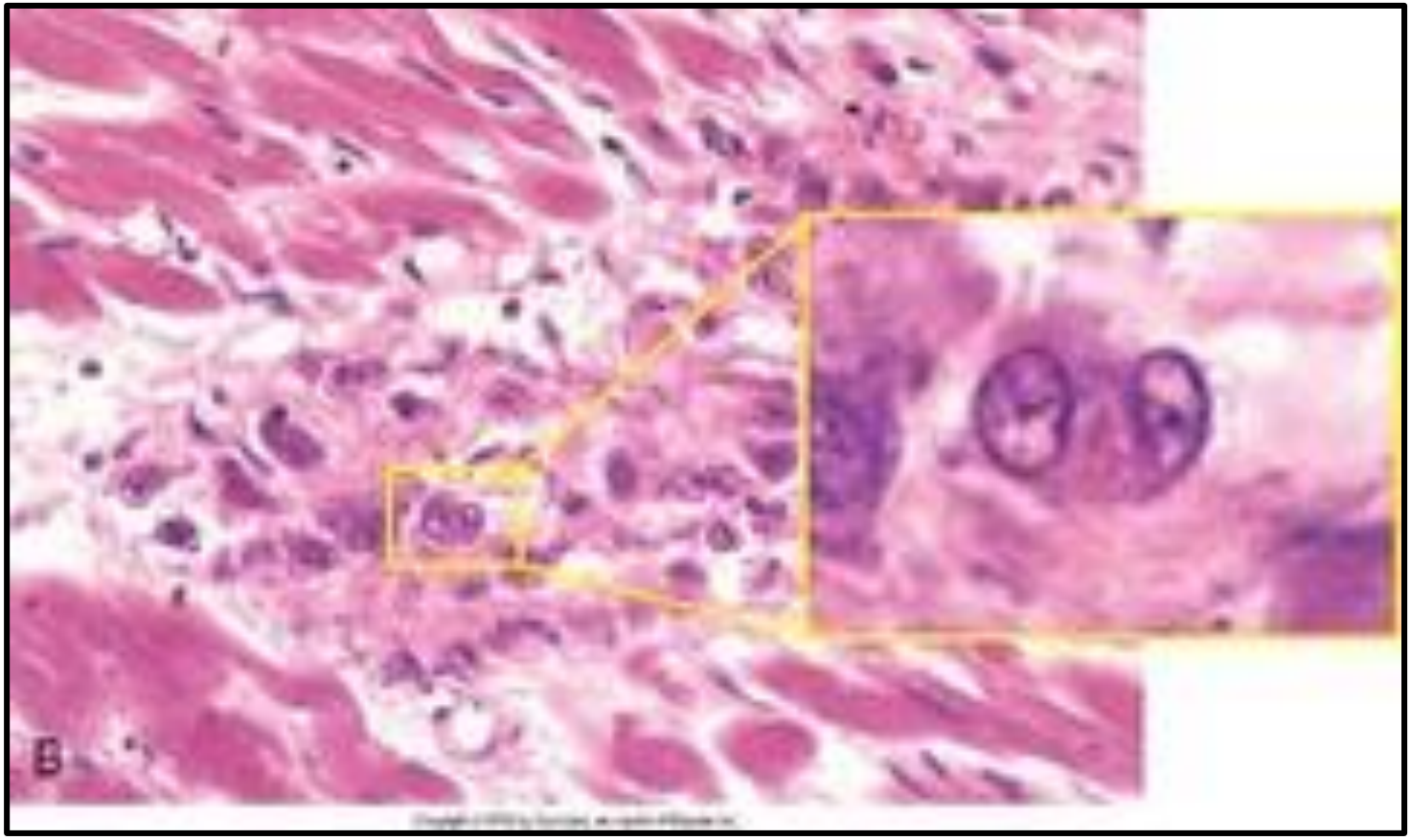
The valve is thickened by dense hyalinized fibrous tissue with vascularization and chronic inflammatory cell infiltrate. The myocardium showing cellular accumulations - Aschoff bodies (arrows) -within the interstitium of the myocardium.

Acute Rheumatic Carditis - HPF



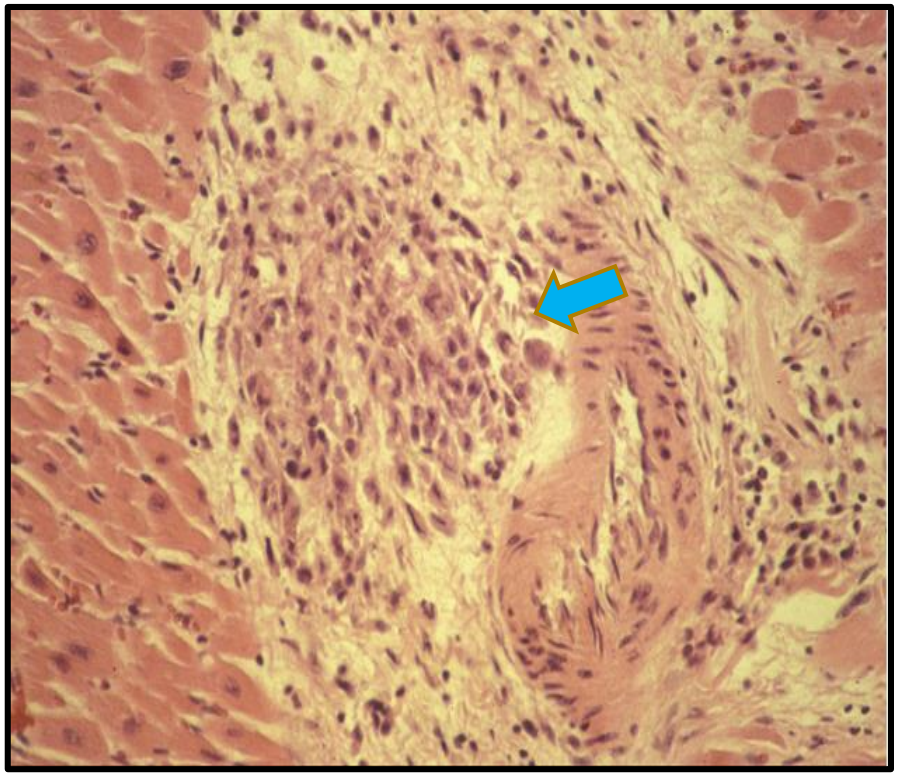
Microscopically, acute rheumatic carditis is marked by a peculiar form of granulomatous inflammation with so-called "Aschoff nodules" seen best in myocardium,

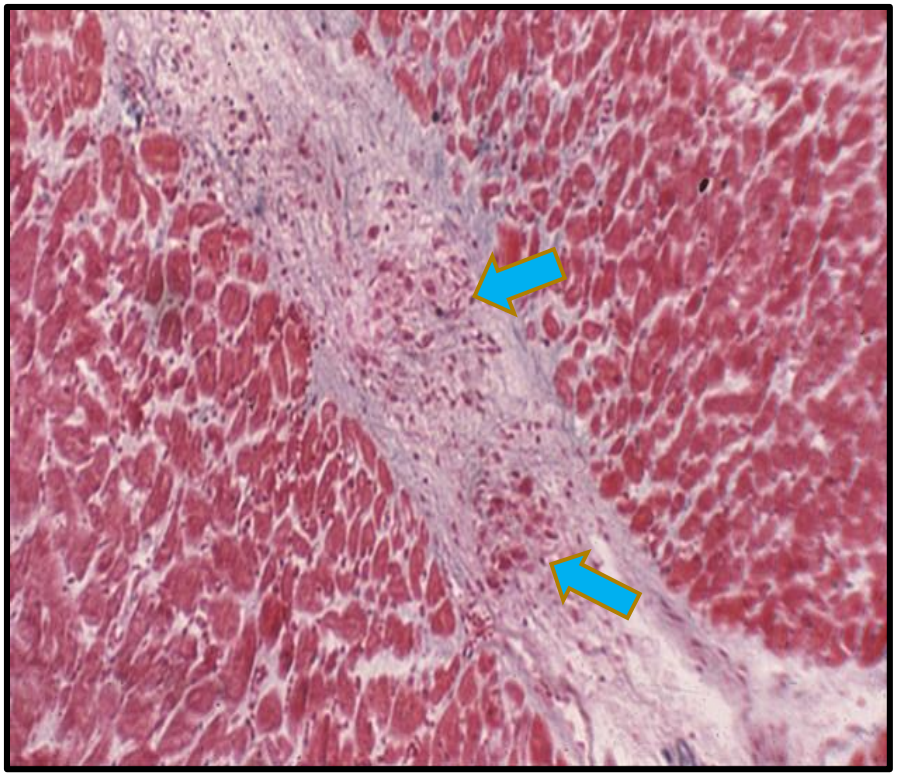
Acute Rheumatic Myocarditis - HPF



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

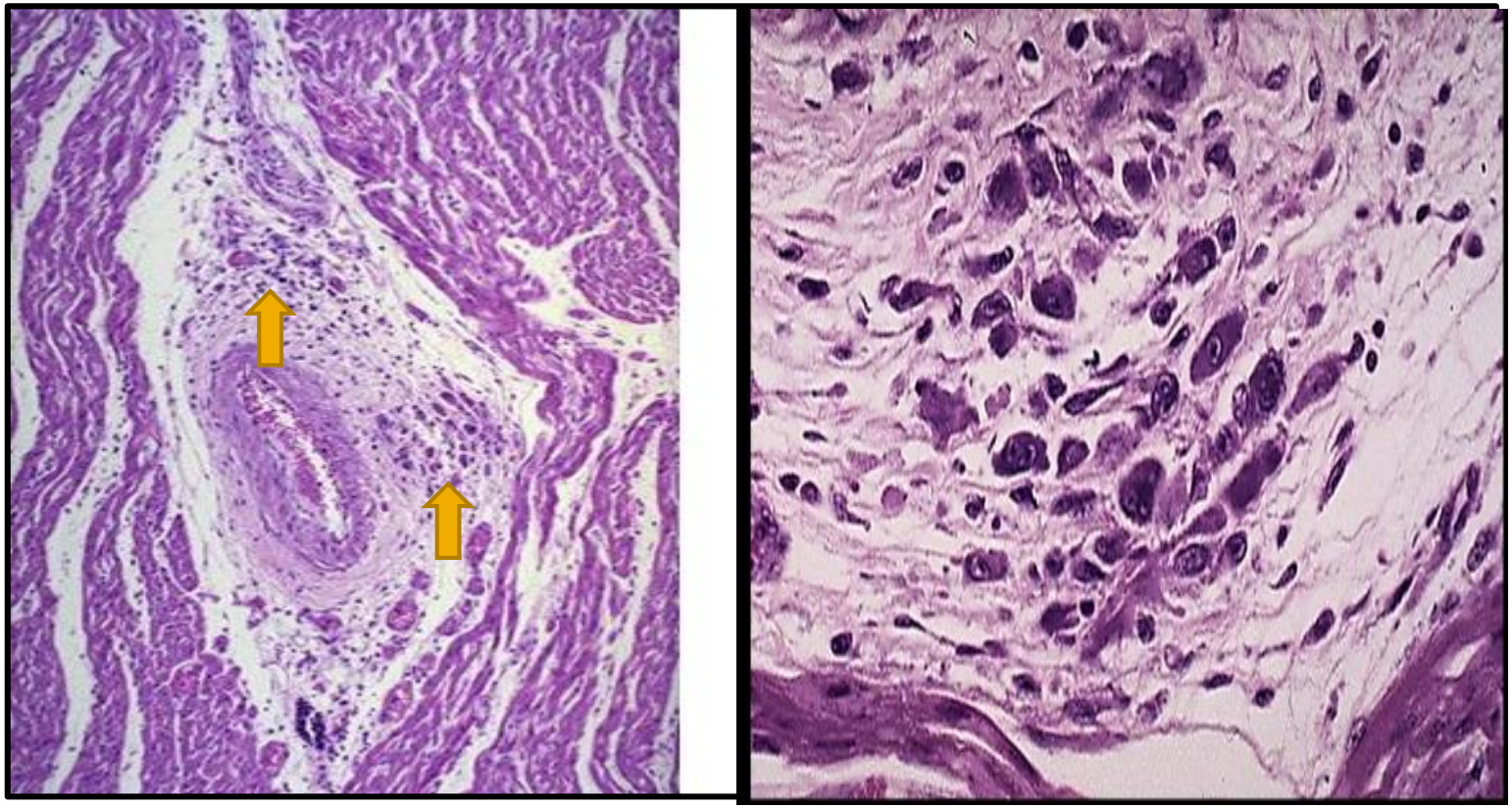
RHEUMATIC MYOCARDIITIS (ASCHOFF NODULE)





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

RHEUMATIC MYOCARDIITIS (ASCHOFF 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).

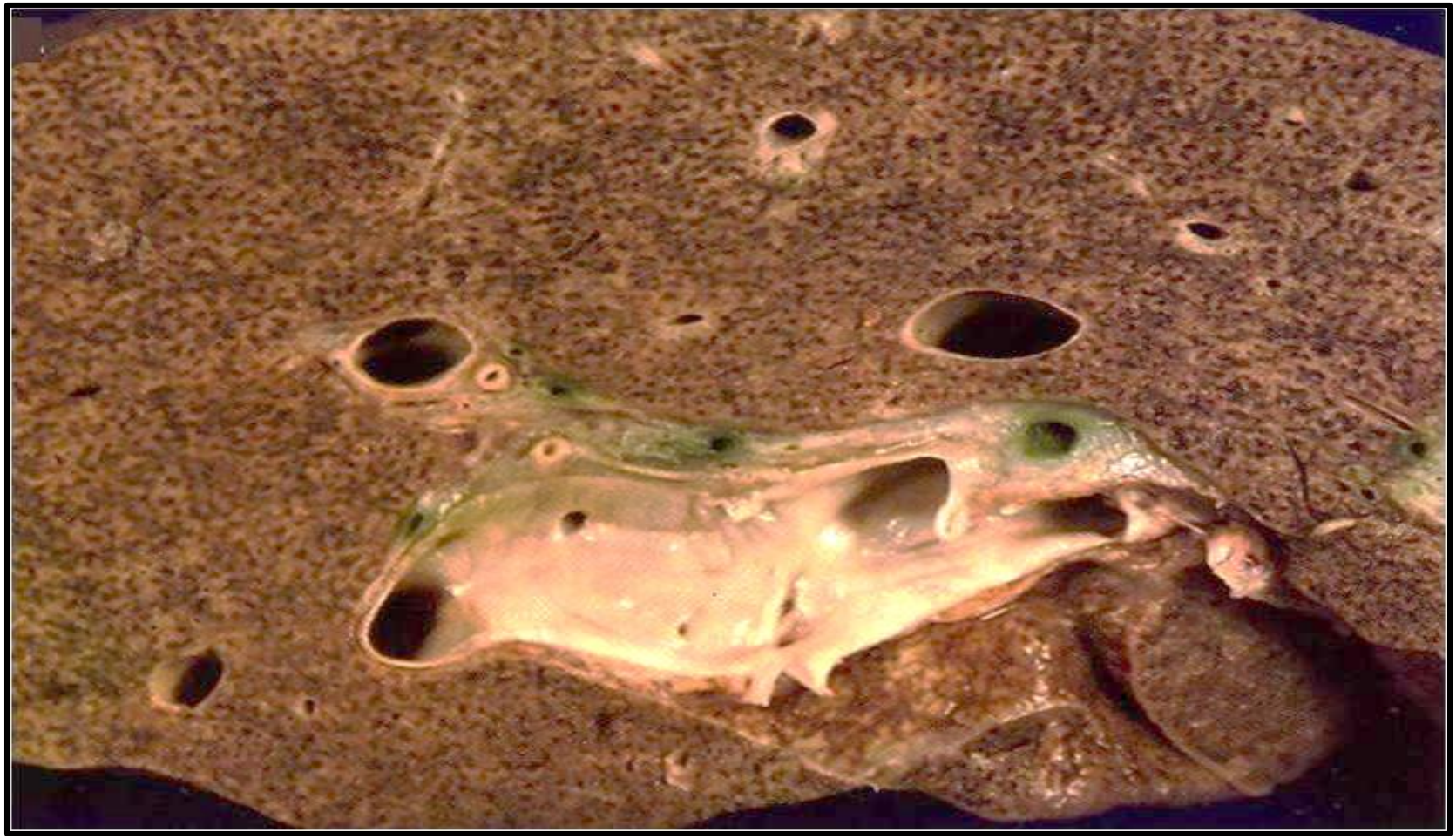
HEART FAILURE

Right Sided Heart Failure

- *Most commonly, right-sided heart failure is caused by left-sided heart failure*, as any increase in pressure in the pulmonary circulation incidental to left-sided failure inevitably burdens the right side of the heart.
- Pure right-sided heart failure is infrequent and usually occurs in patients with any one of a variety of disorders affecting the lungs; hence, it is often referred to as cor *pulmonale*.
- The clinical features of isolated right-sided heart failure are those related to systemic (and portal) venous congestion.

Chronic venous congestion of the liver

NUTMEG LIVER – Cut surface

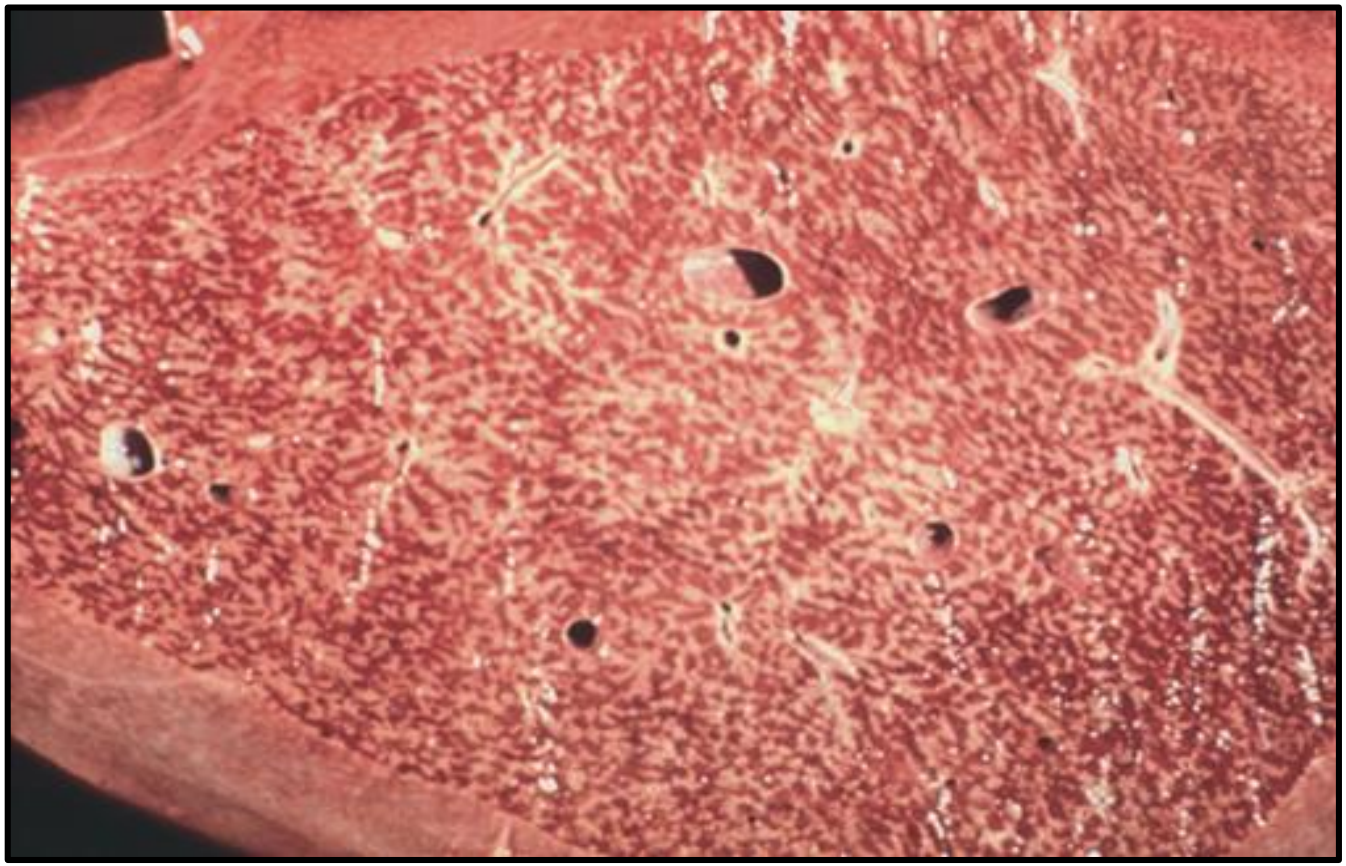


Section of liver showing alternating pale and dark areas with a nutmeg like appearance possibly due to passive congestion secondary to right sided heart failure.

NUTMEG LIVER – Cut surface

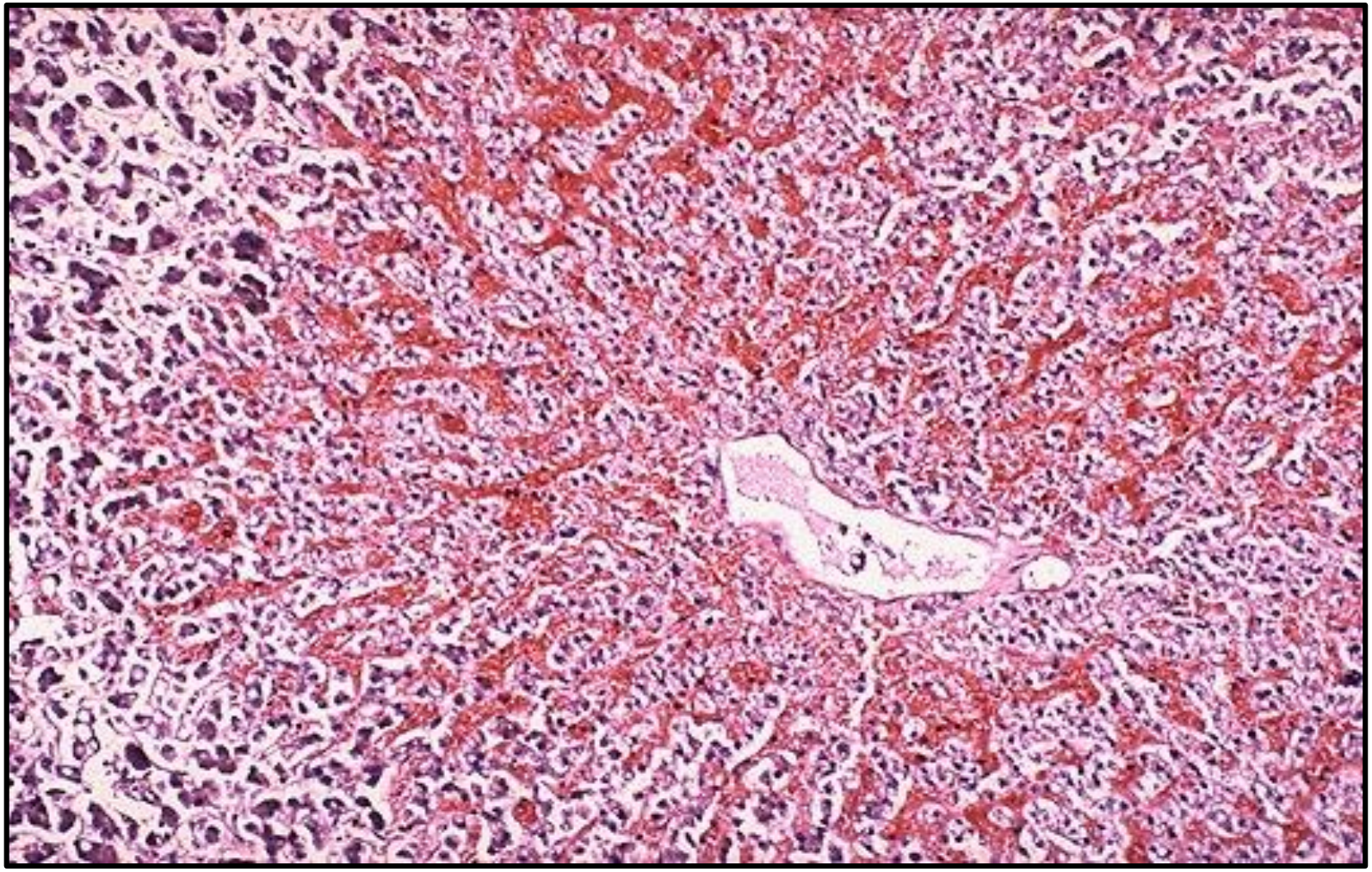
The hepatic parenchyma contains a faintly nodular pattern and nutmeg staining due to chronic passive congestion due to Right sided heart failure.

Chronic Congestion of the Liver - CS



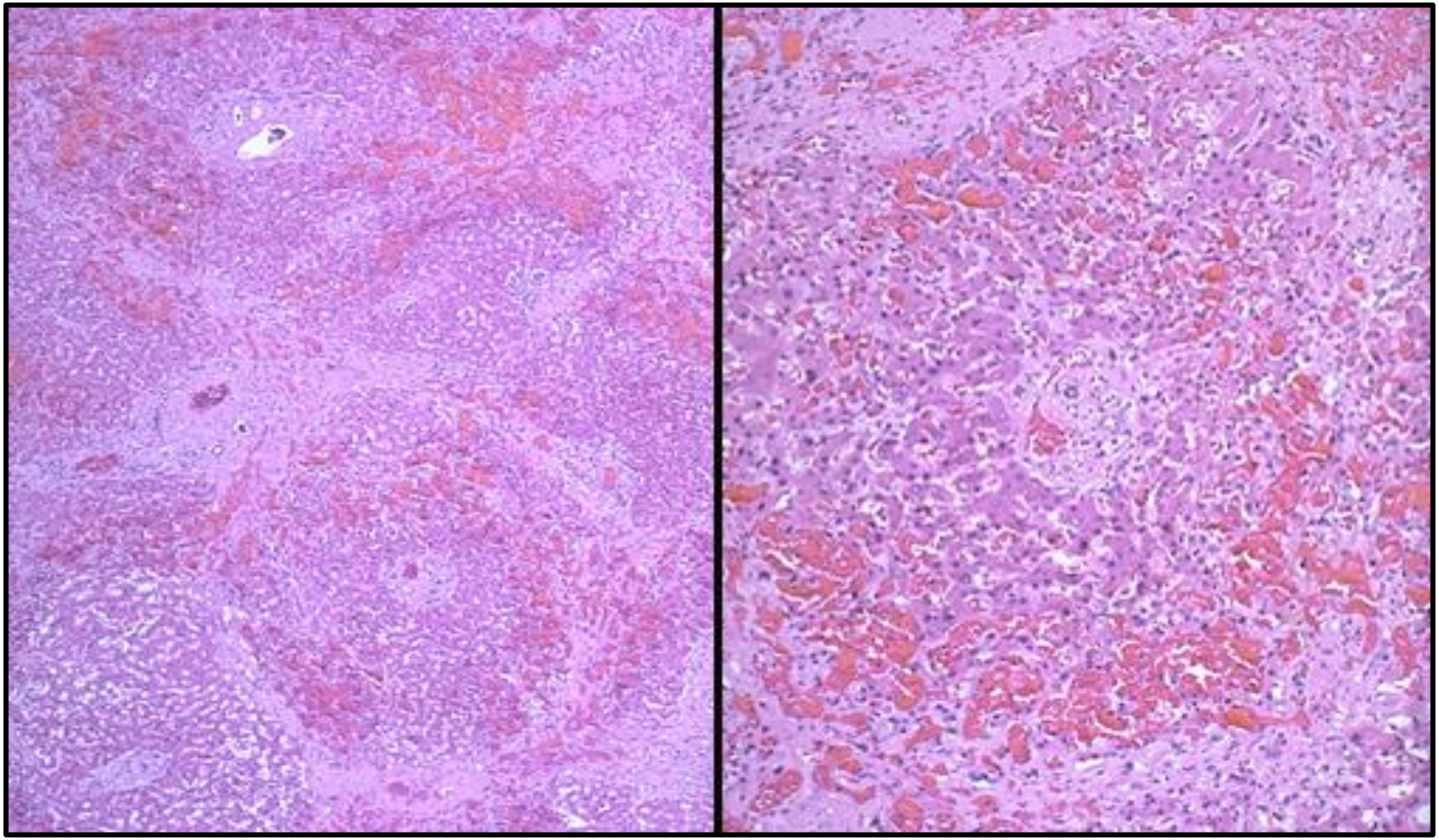
A gross view of nutmeg appearance of liver characteristic of centrolobular or necrosis or passive congestion of the liver. The central areas of the liver are congested and take on a sort of dusky appearance. They are soft in consistency and they are surrounded by the paler areas of fatty liver that are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

Chronic Congestion of the Liver - LPF



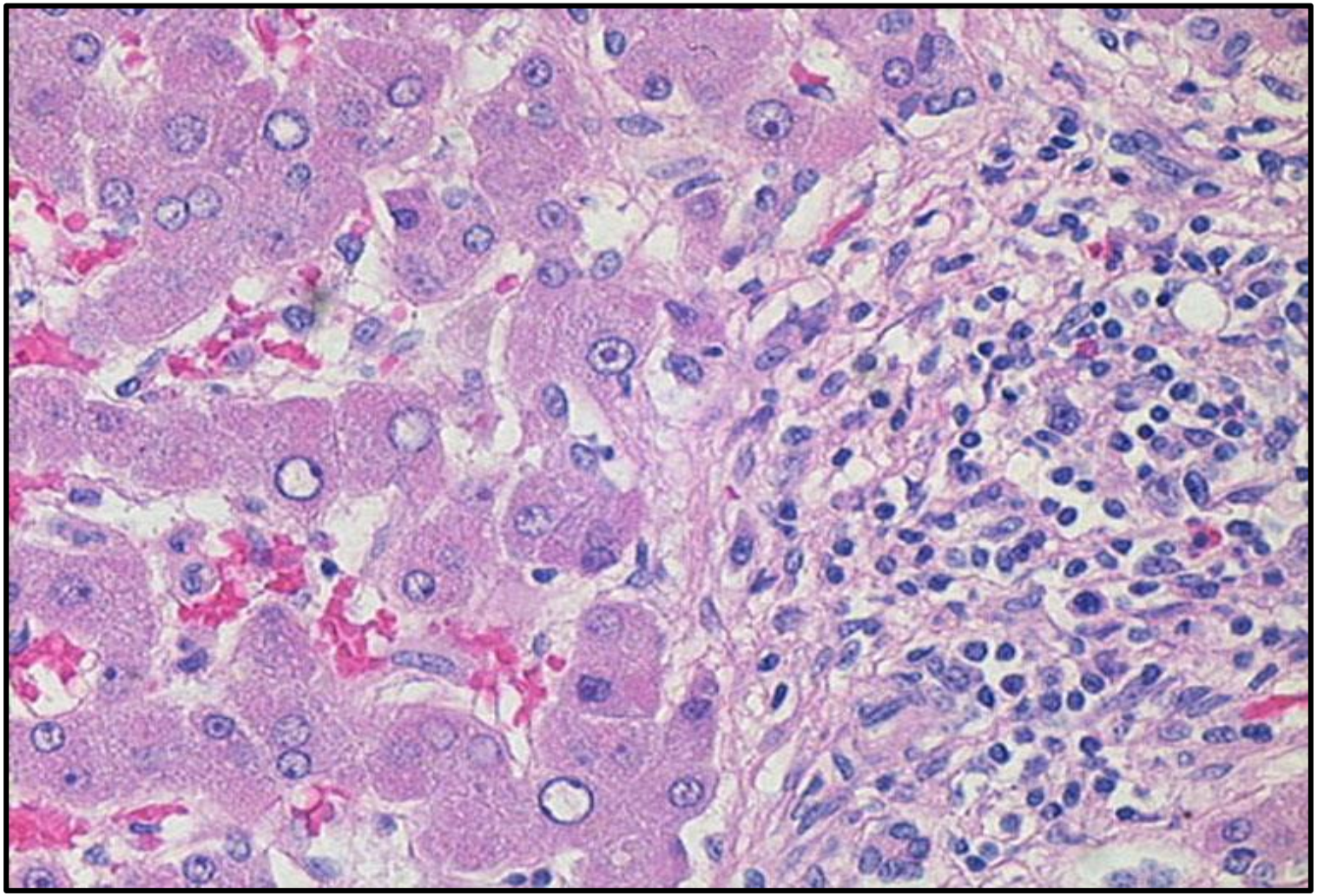
The central portion of liver lobules shows congestion and dilatation of central veins and blood sinusoids, with atrophy and necrosis of liver cells.

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

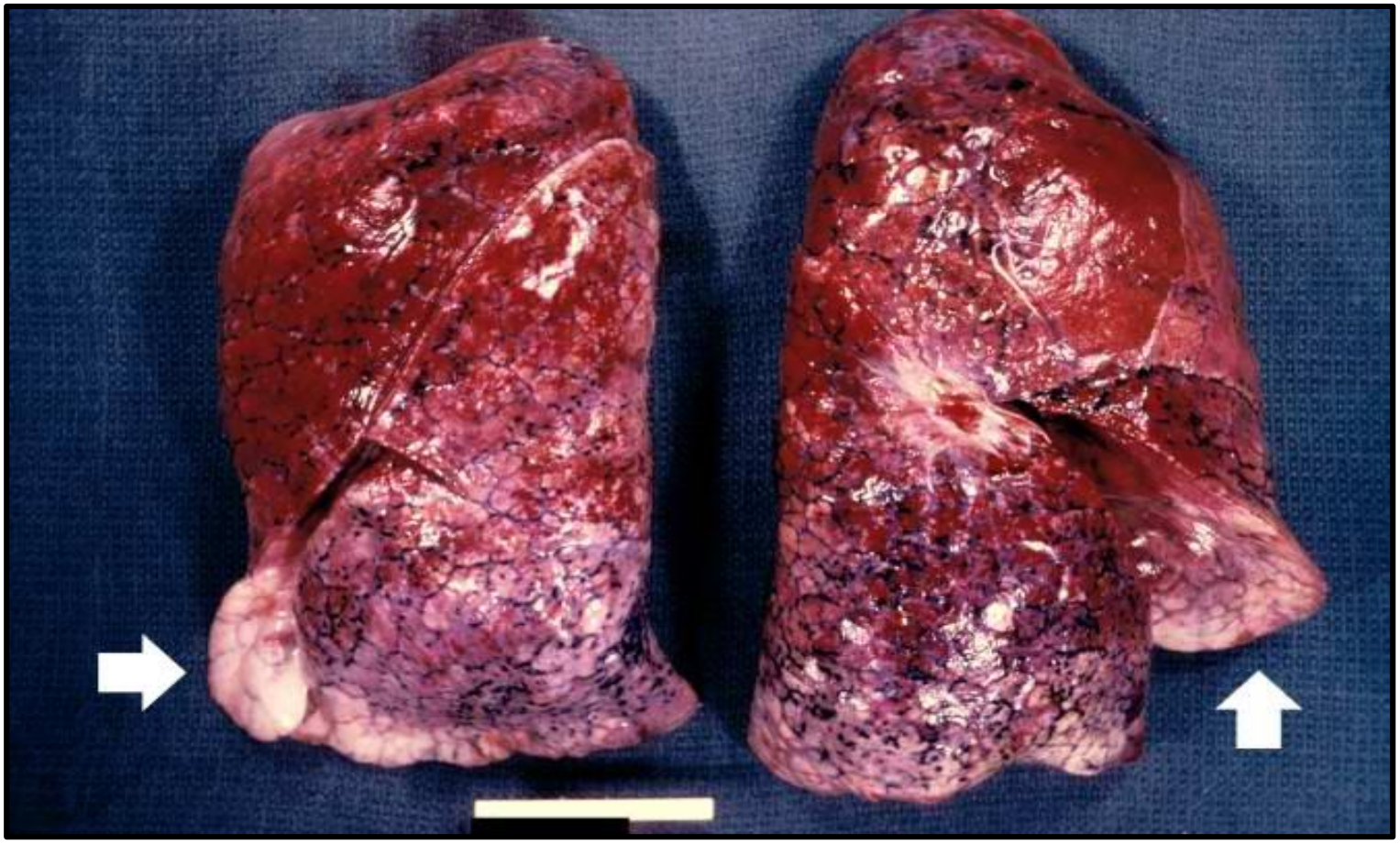
- Left-sided heart failure is most often caused by

(1) ischemic heart disease,
(2) hypertension,
(3) aortic and mitral valvular diseases, and
(4) myocardial diseases.

- The morphologic and clinical effects of left-sided CHF primarily result from congestion of the pulmonary circulation, stasis of blood in the left-sided chambers, and hypoperfusion of tissues leading to organ dysfunction.

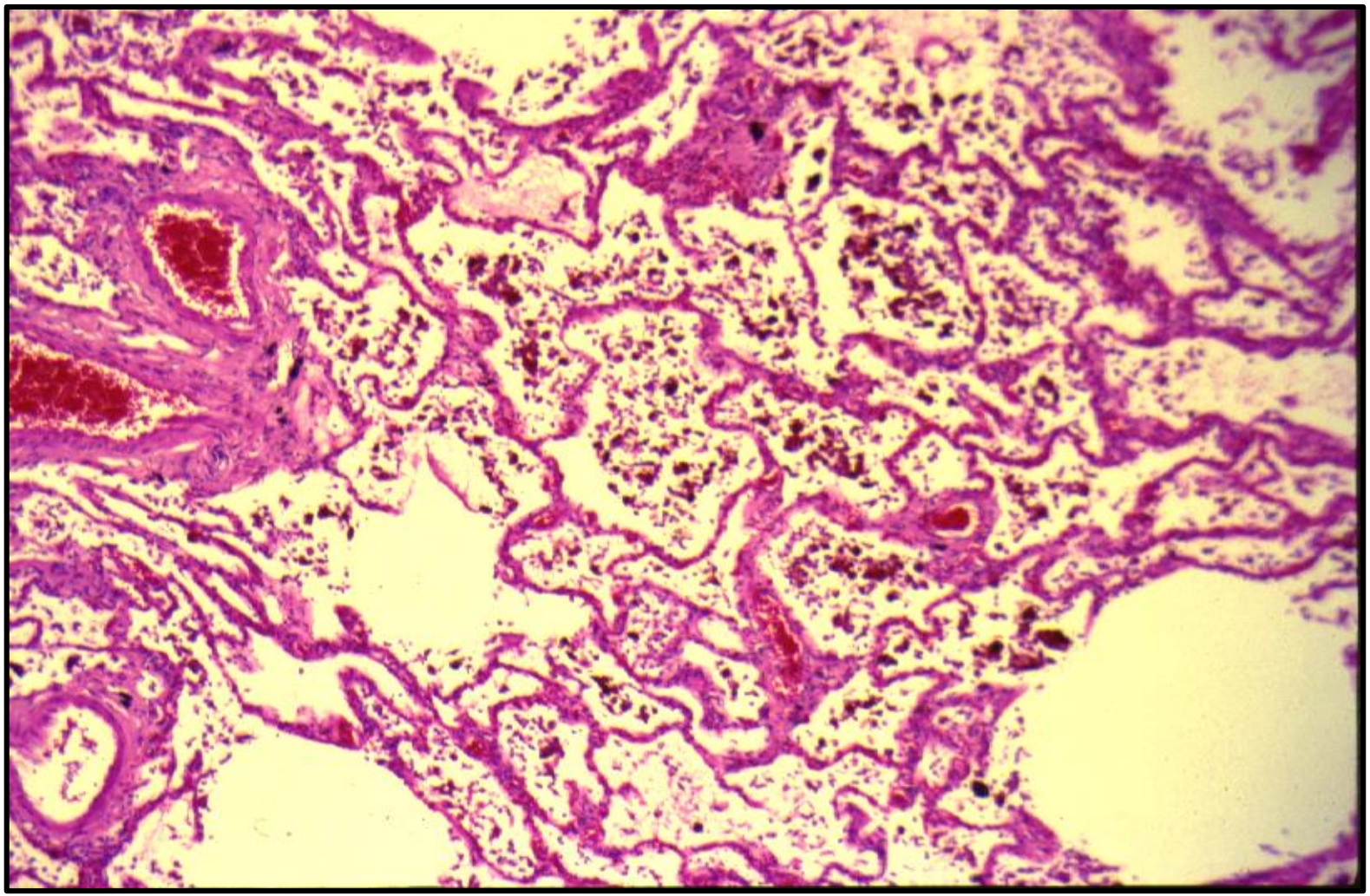
Chronic venous congestion of the lung

Chronic venous congestion of the lung - Gross



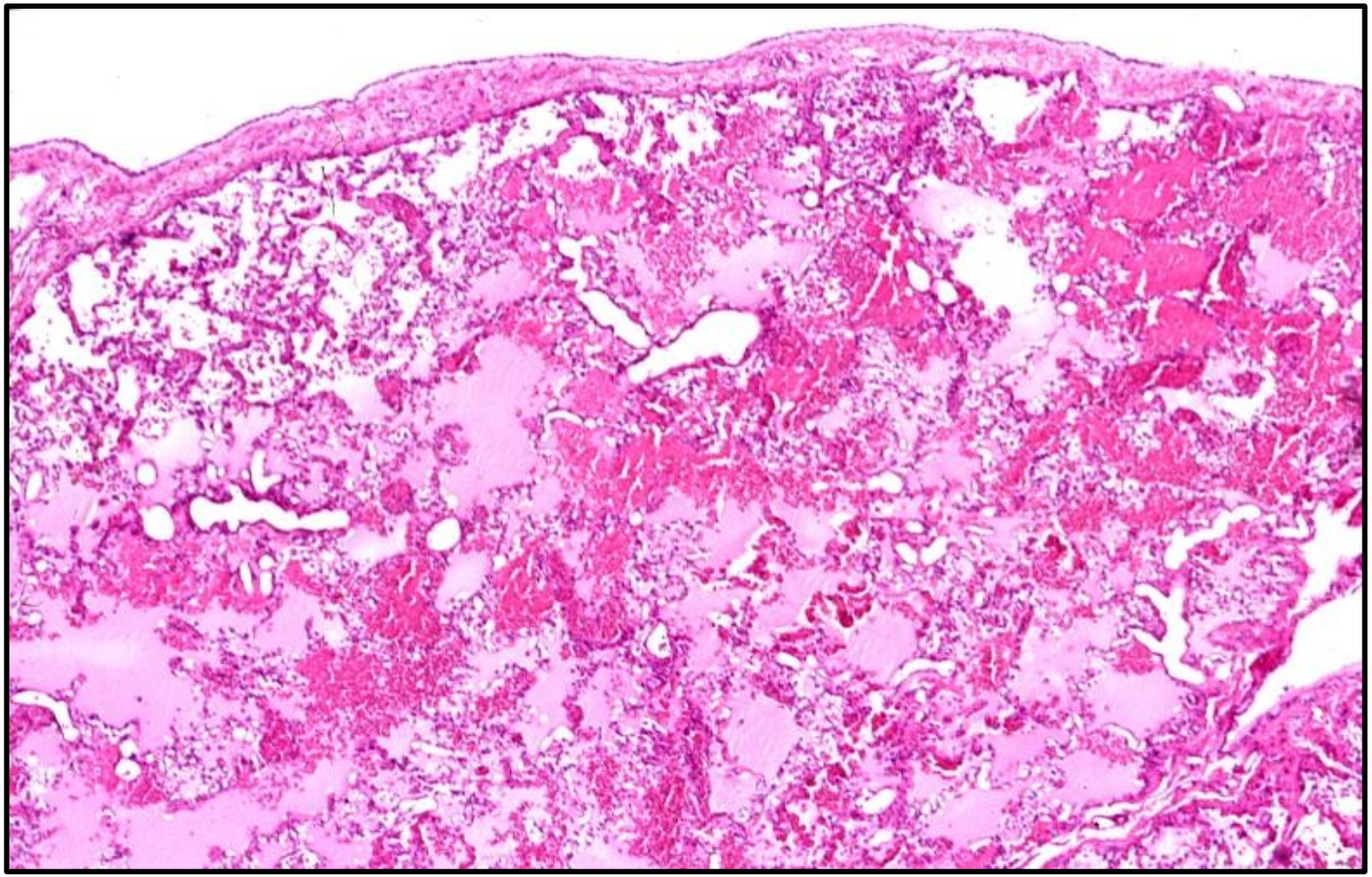
This is a gross photograph of lungs that are distended and red. The reddish coloration of the tissue is due to congestion. Some normal pink lung tissue is seen at the edges of the lungs (arrows).

Chronic venous congestion of the lung - LPF



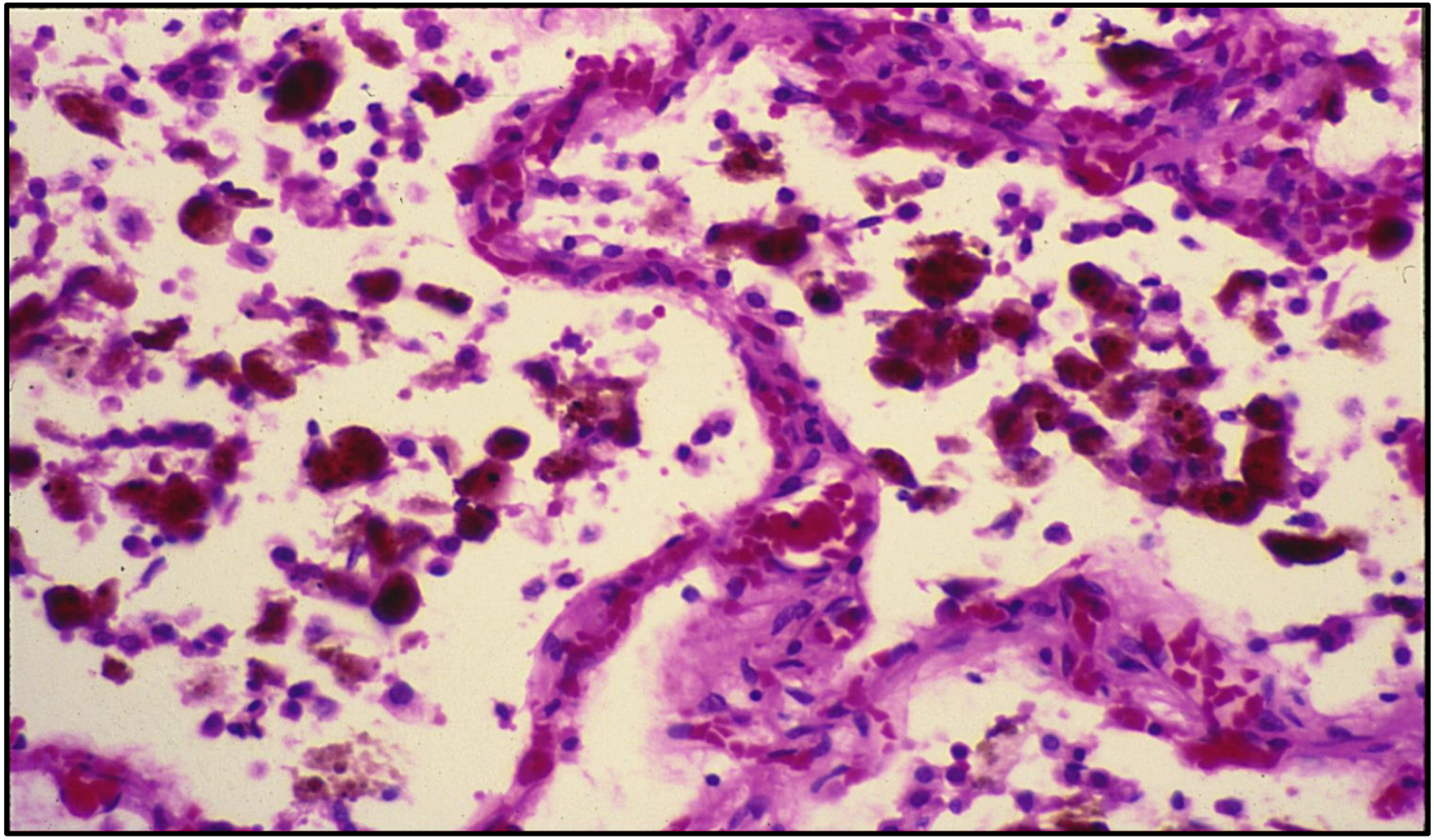
The alveolar walls are thickened by dilated and engorged capillaries.

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.

MYOCARDIAL HYPERTROPHY

- thickening of the ventricle walls of the heart.
- The ventricle is working against high pressure, or "pumping" higher than normal volume leading to myocardial hypertrophy.

Causes of ventricular hypertrophy

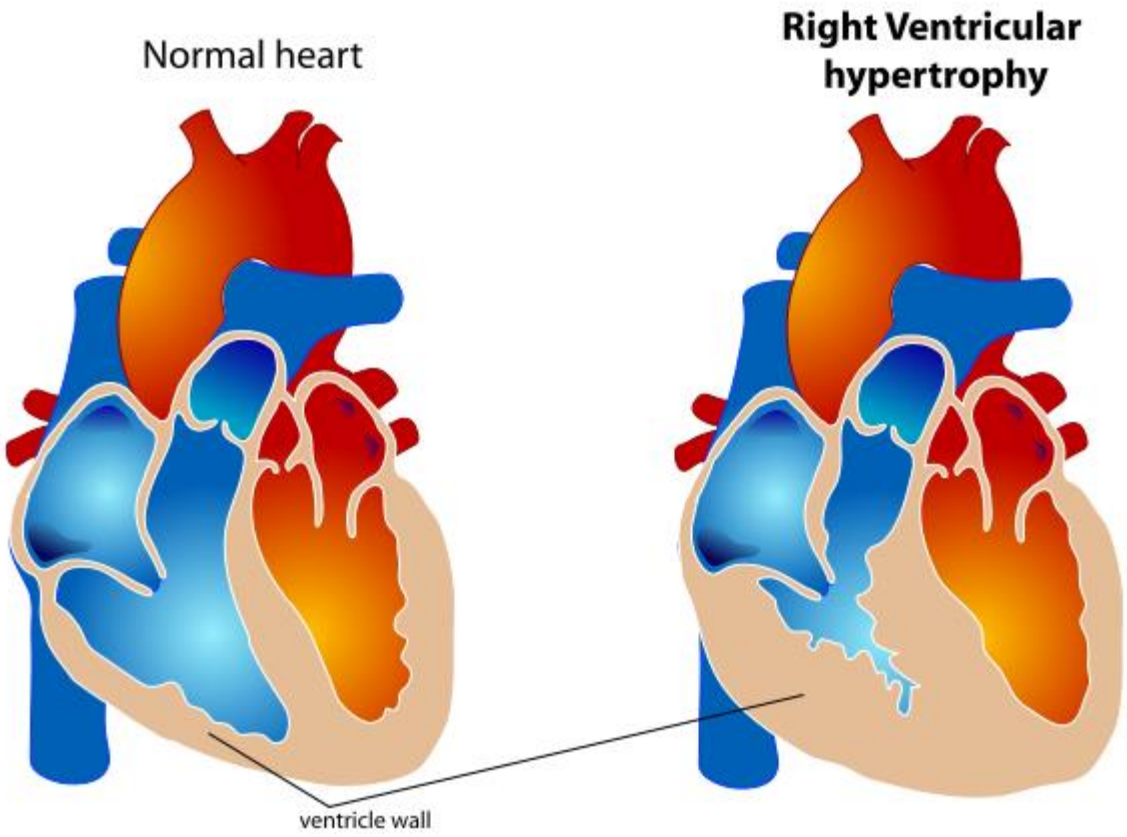
Left ventricular hypertrophy :

- Systemic hypertension
- Aortic valve stenosis

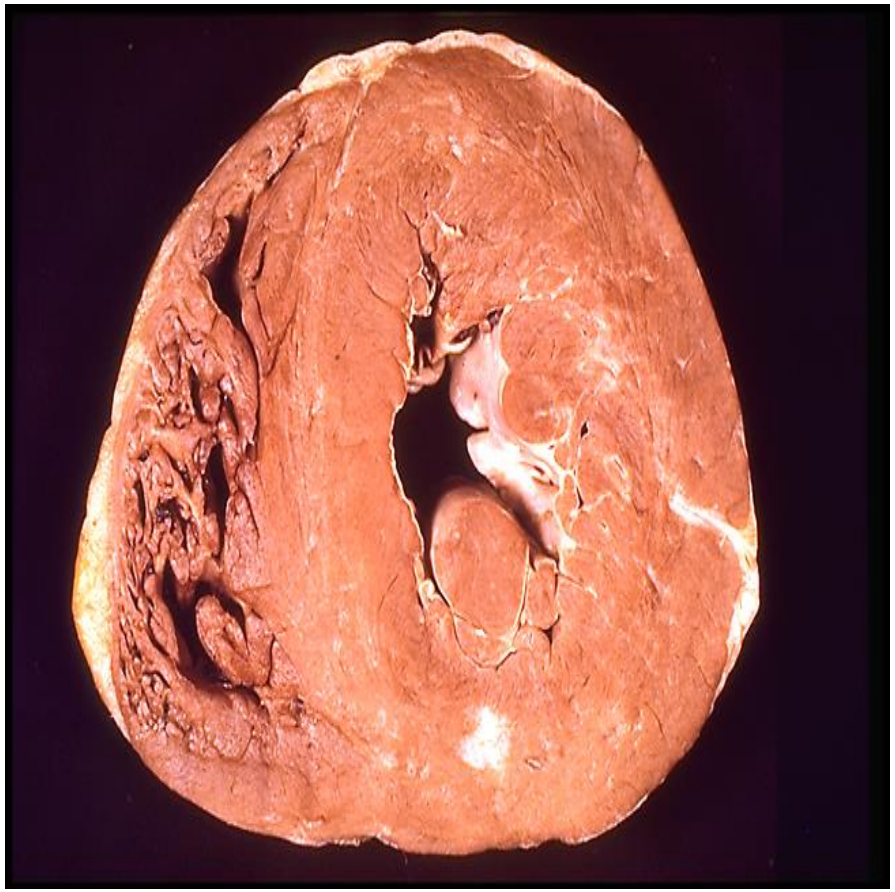
Right ventricular hypertrophy:

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

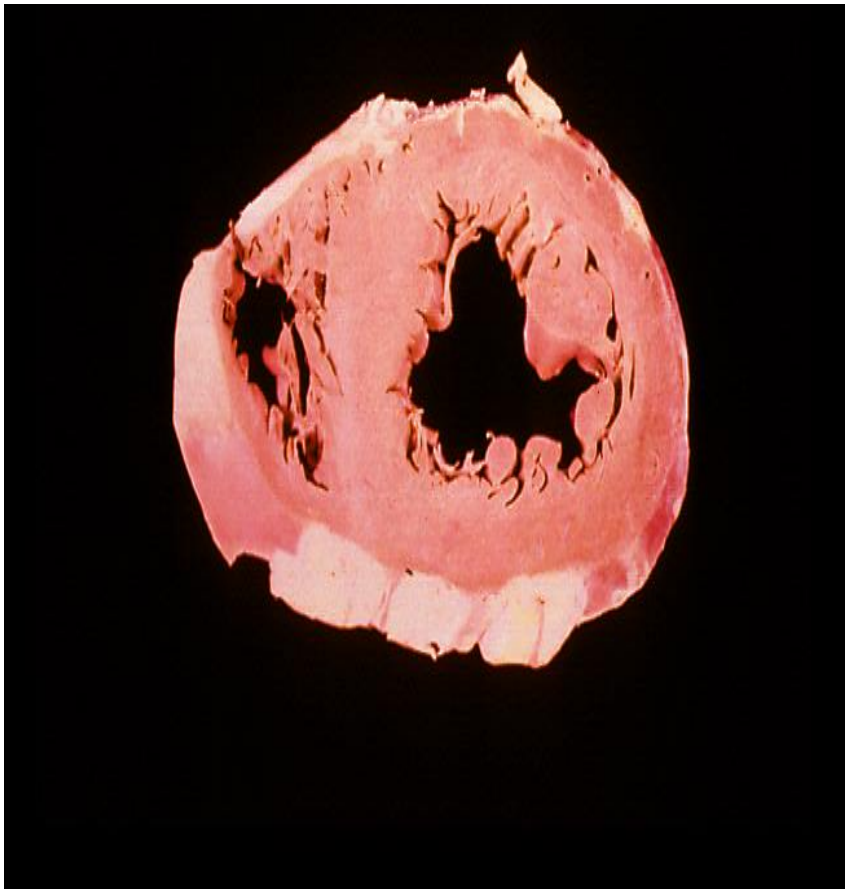
Right ventricular hypertrophy



Normal and hypertrophied left ventricle – cross section

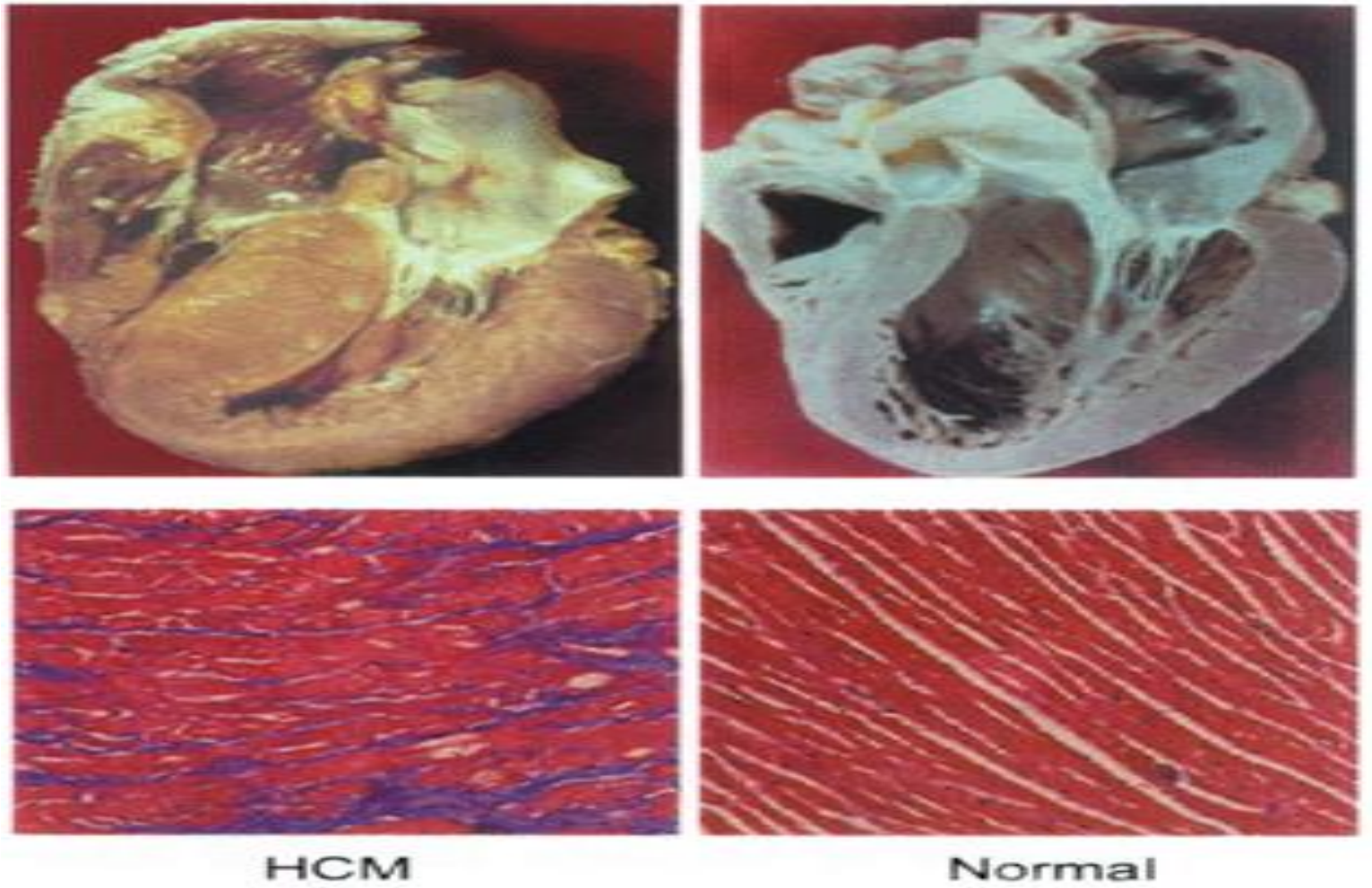


Left ventricular hypertrophy



Normal ventricles

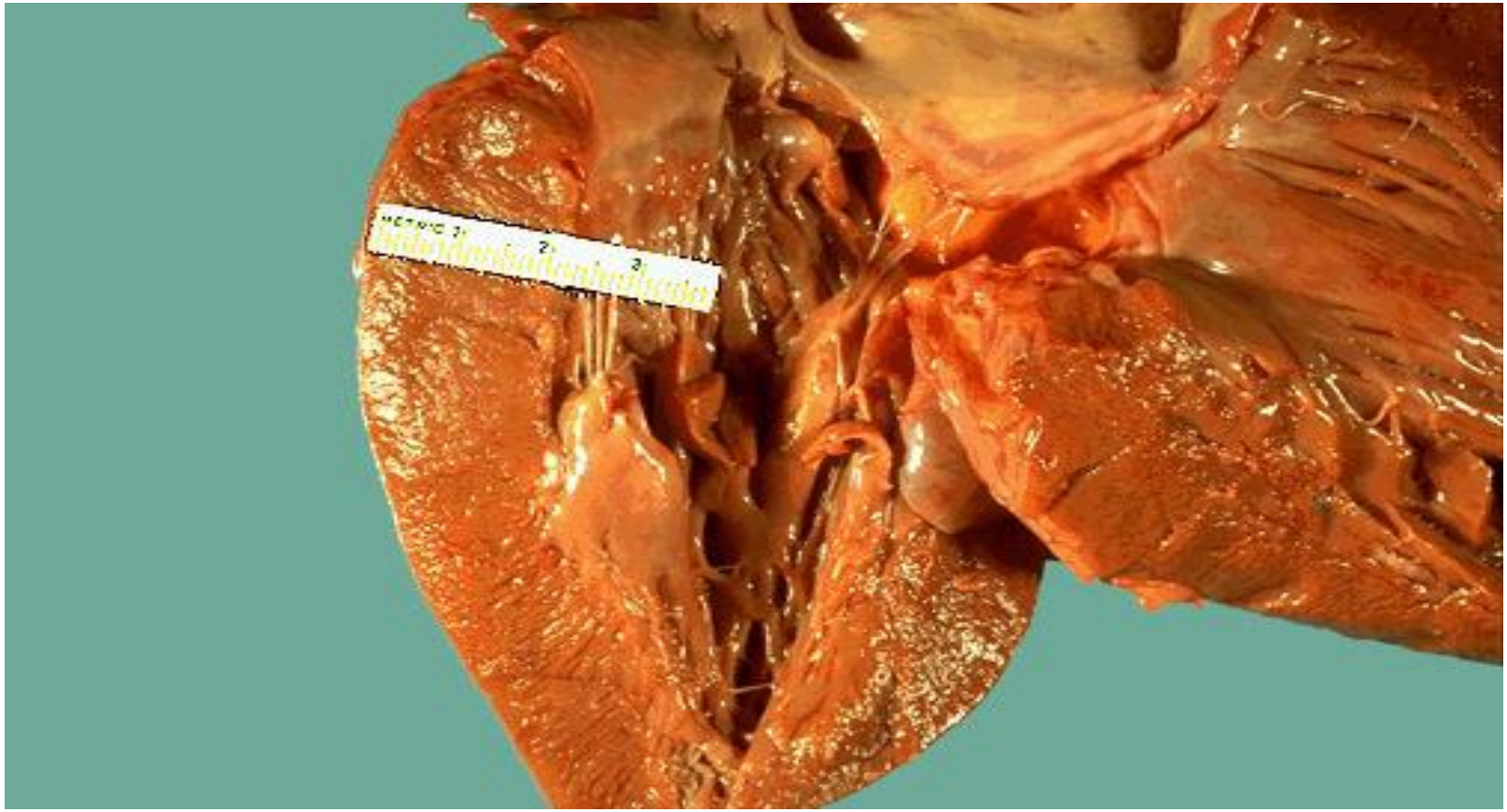
Normal and hypertrophied left ventricle - CS



Histopathology showing significant myofiber disarray and interstitial fibrosis

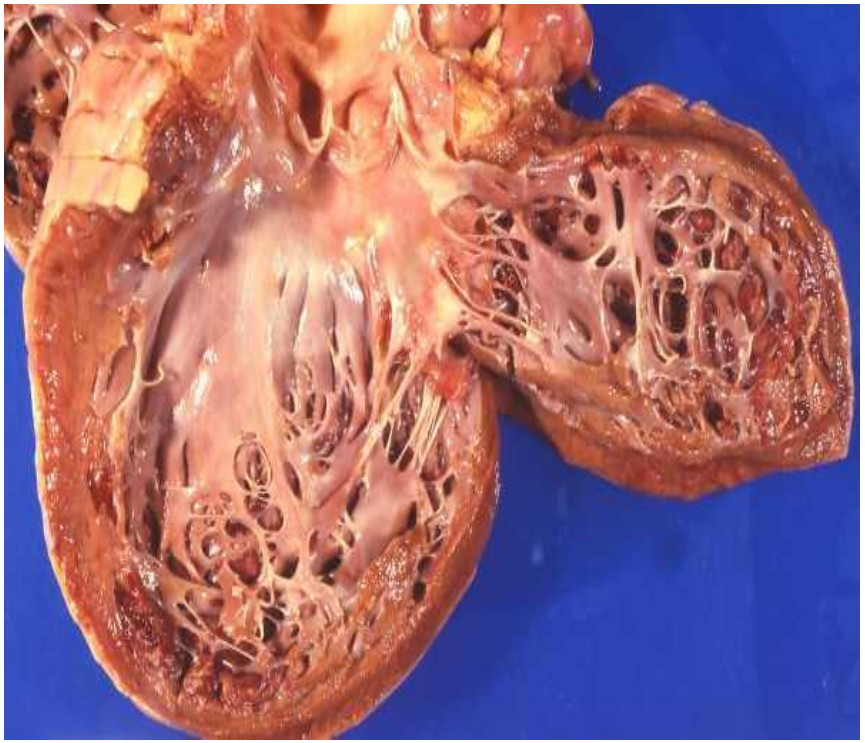
Histopathology showing Normal myocytes

Left ventricular hypertrophy - Gross



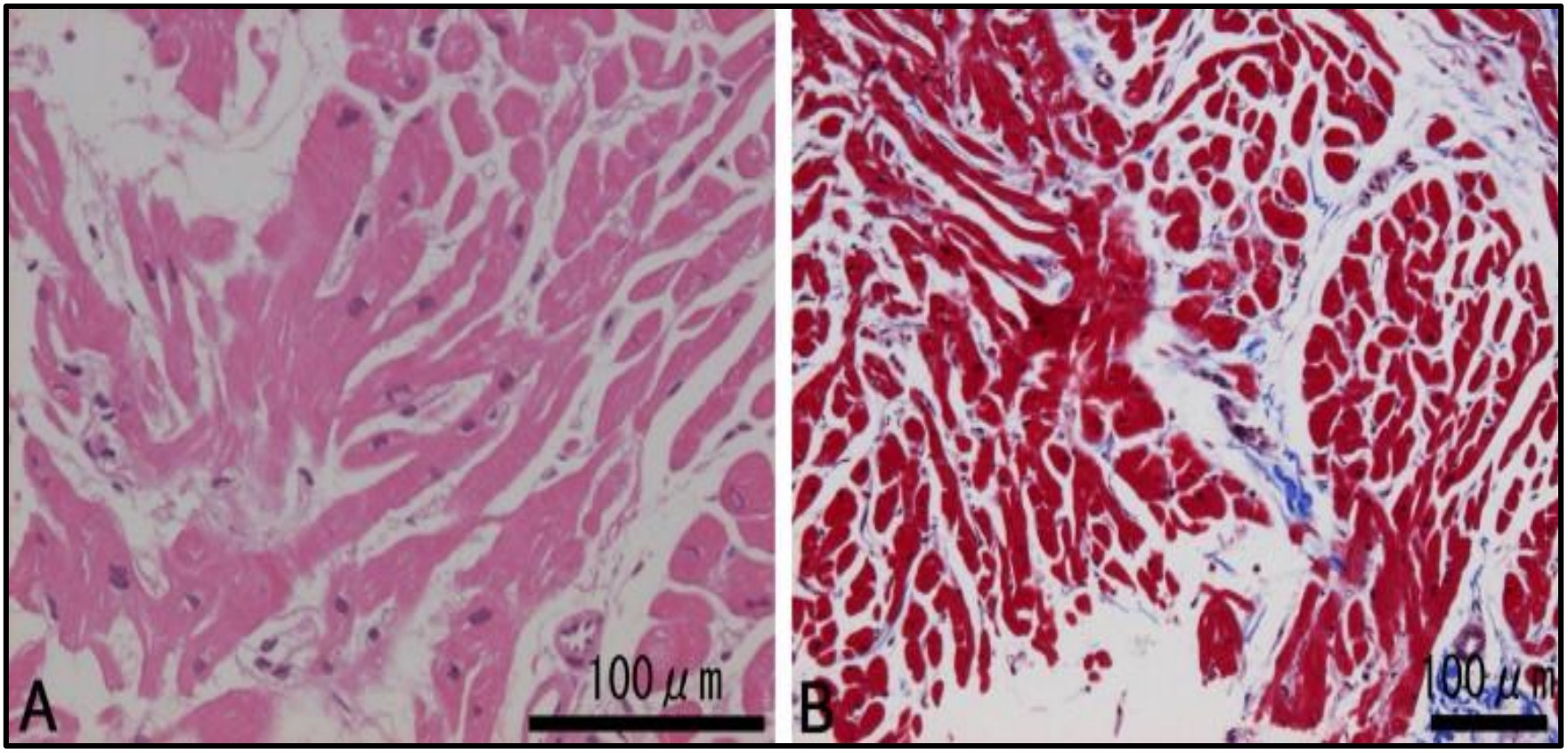
Heart from a hypertensive patient. The left ventricle is very thick (over 2 cm). However the rest of the heart is fairly normal in size as is typical for hypertensive heart disease. The hypertension creates a greater pressure load on the heart to induce the hypertrophy

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

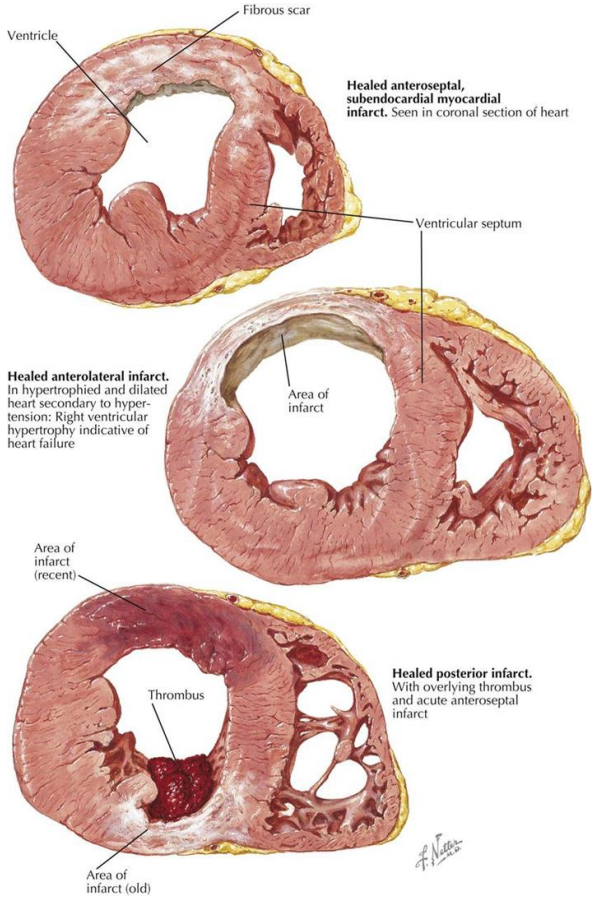
- *MI, also known as "heart attack," is the death of cardiac muscle due to prolonged severe ischemia.*
- It is by far the most important form of IHD
- **Risk Factors**

•Left anterior descending coronary artery (40% to 50%): infarcts involving the anterior wall of left ventricle near the apex; the anterior portion of ventricular septum; and the apex circumferentially
•Right coronary artery (30% to 40%): infarcts involving the inferior/posterior wall of left ventricle; posterior portion of ventricular septum; and the inferior/posterior right ventricular free wall in some cases
•Left circumflex coronary artery (15% to 20%): infarcts involving the lateral wall of left ventricle except at the apex

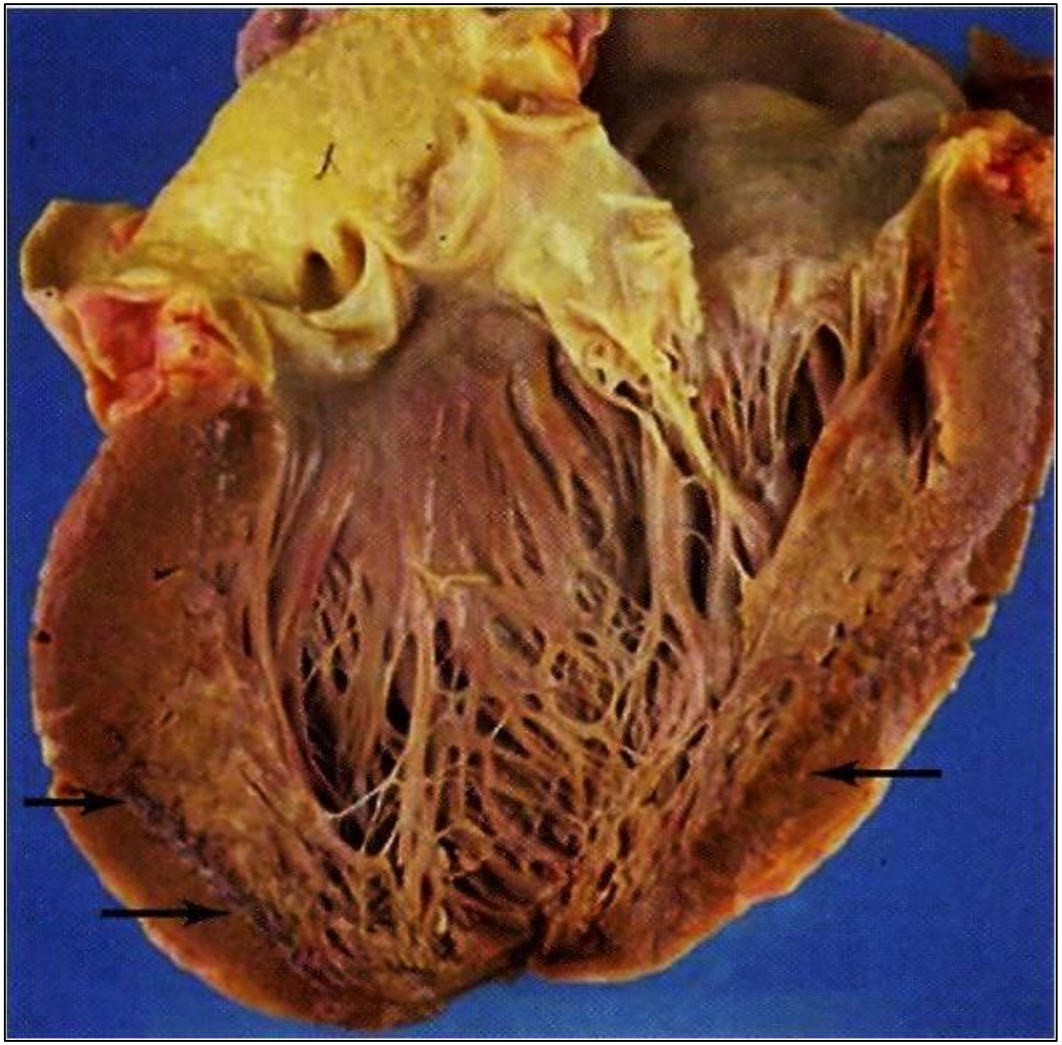
Background information

Changes in myocardial Infarction

Time	Gross changes	Microscopic changes
0-4 hours	None	None
4-12 hours	Mild Mottling (hemorrhagic look)	Coagulation necrosis
12-24 hours	Dark Mottling	More coagulation necrosis; neutrophils come in
1-7 days	Yellow infarct center with surrounding red borders	Neutrophils die, macrophages come to eat dead cells
1-2 weeks	Yellow infarct center with red gray borders	Granulation tissue
2-8 weeks	Scar	Collagen and fibrosis



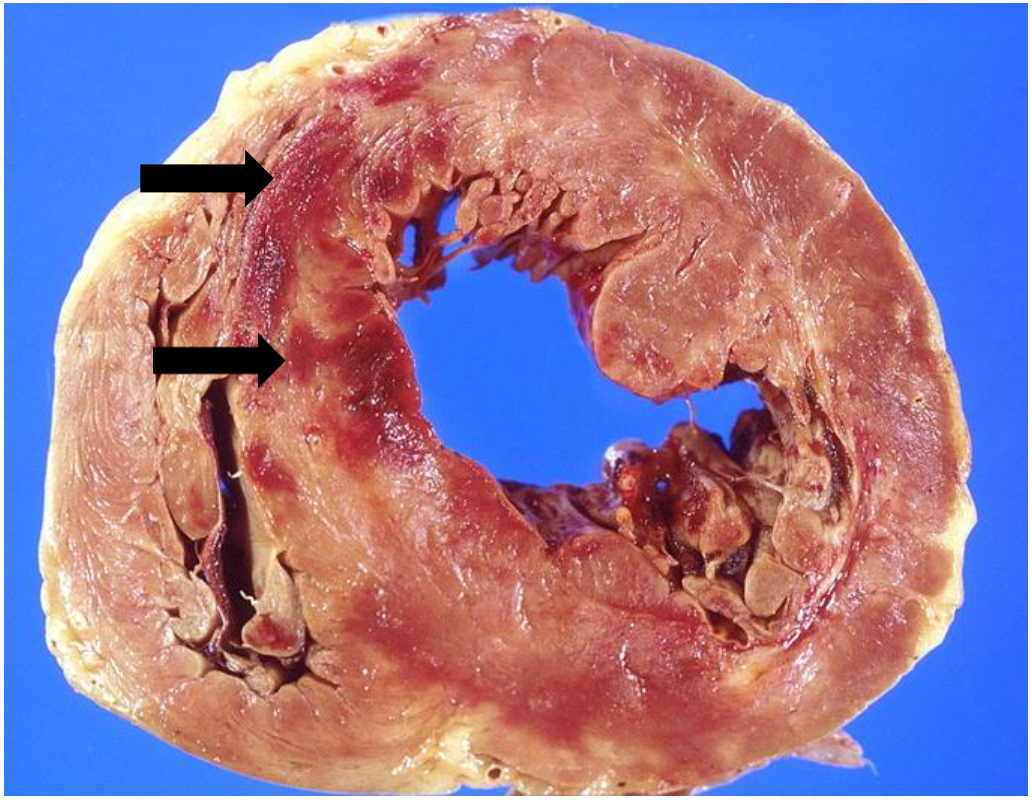
Myocardial Infarction - CS

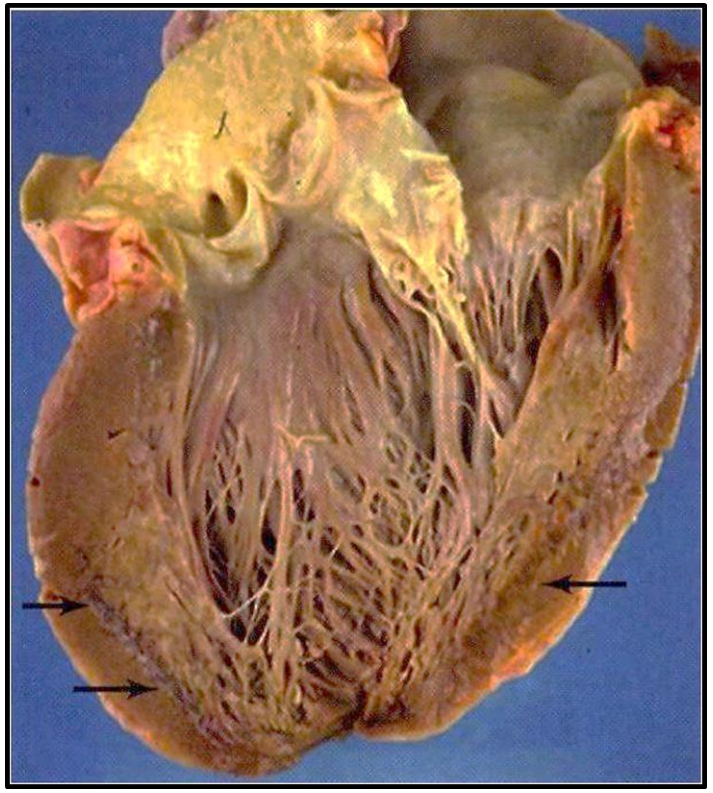


Complications that might occur :

- ***arrhythmias ,***
- ***HF***
- ***ventricular aneurysm,***
- ***rupture of myocardium,***
- ***cardiac tamponade***

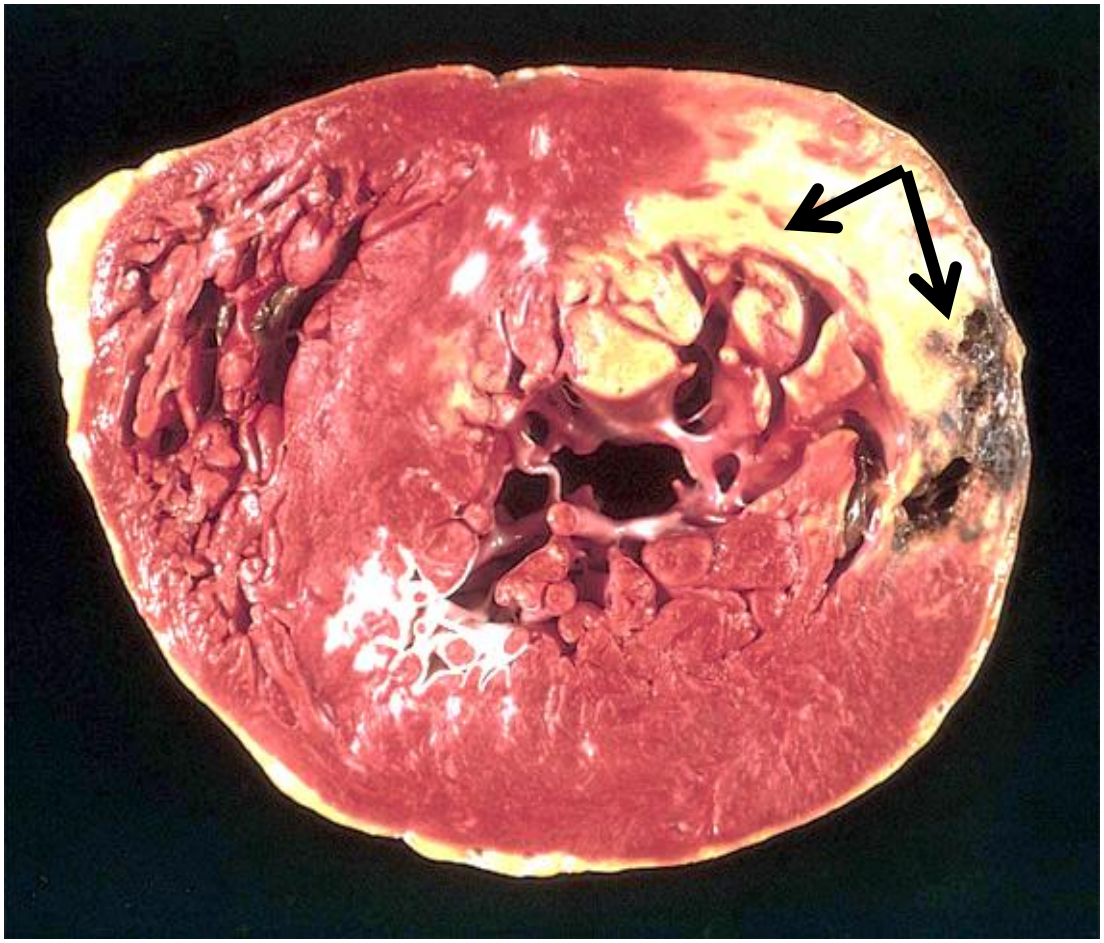
Myocardial Infarction





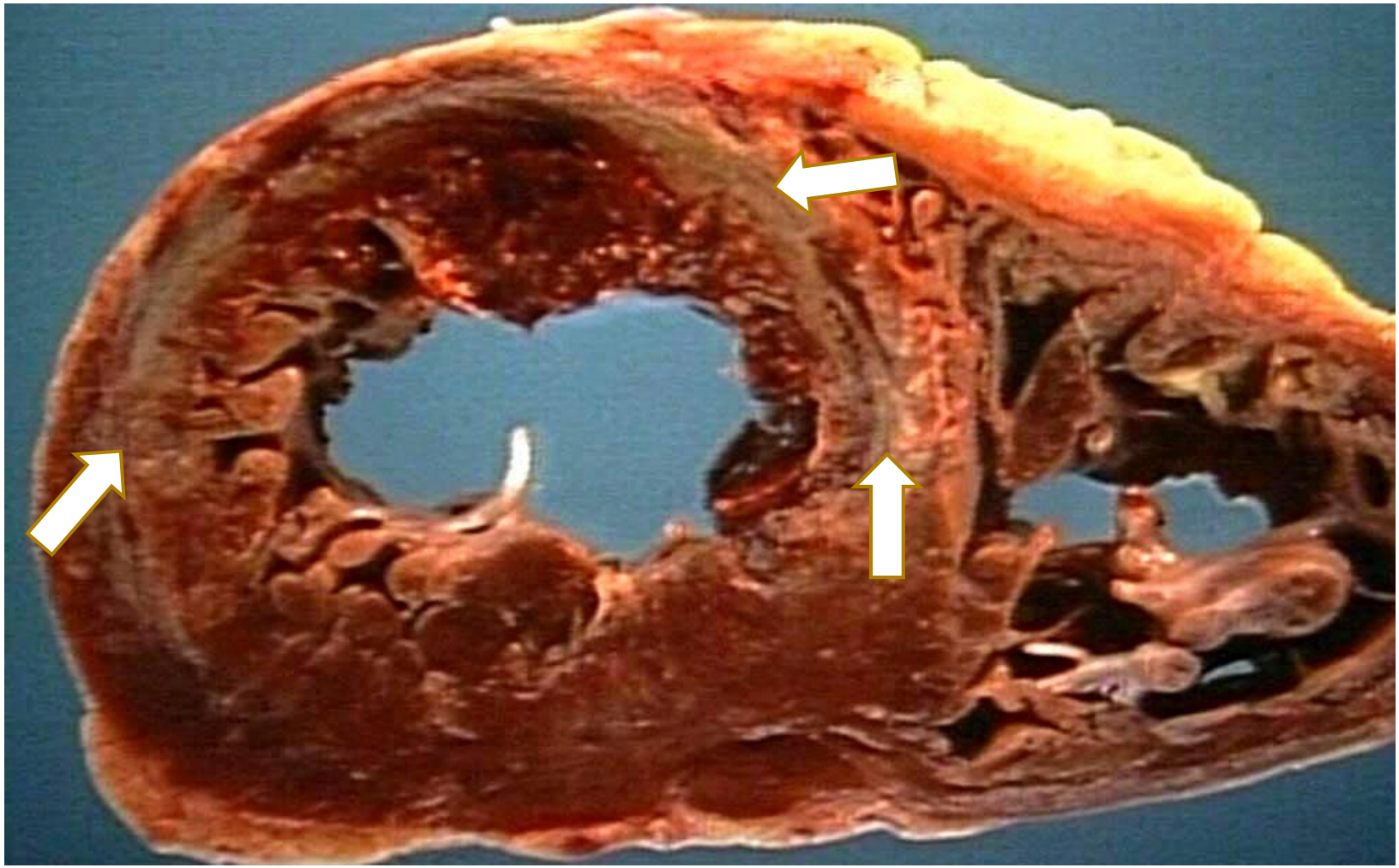
Acute MI: area of fresh myocardial infarction (arrows) in the left ventricle. Initially the area of fresh infarct appears red. The area of infarct becomes well defined by 2 to 3 days with a central area of yellow discoloration surrounded by a thin rim of hemorrhage.

Myocardial Infarction - CS



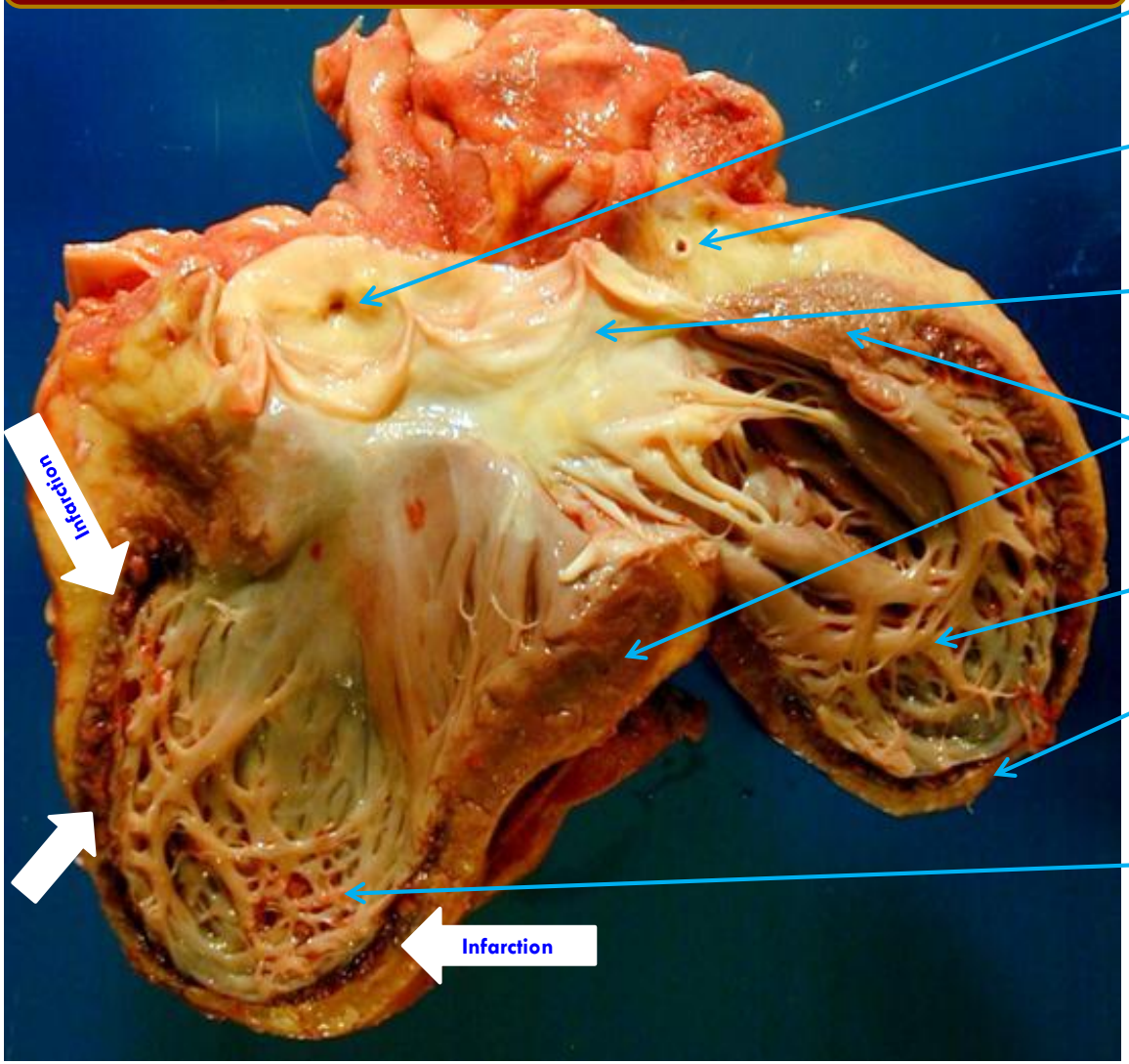
Cross section of the left and right ventricles shows a pale and irregular focal fibrosis in the left ventricular wall with increased thickness .

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



Right coronary ostium, patent

Left circumflex artery, patent

Aortic valve

Residual normal myocardium

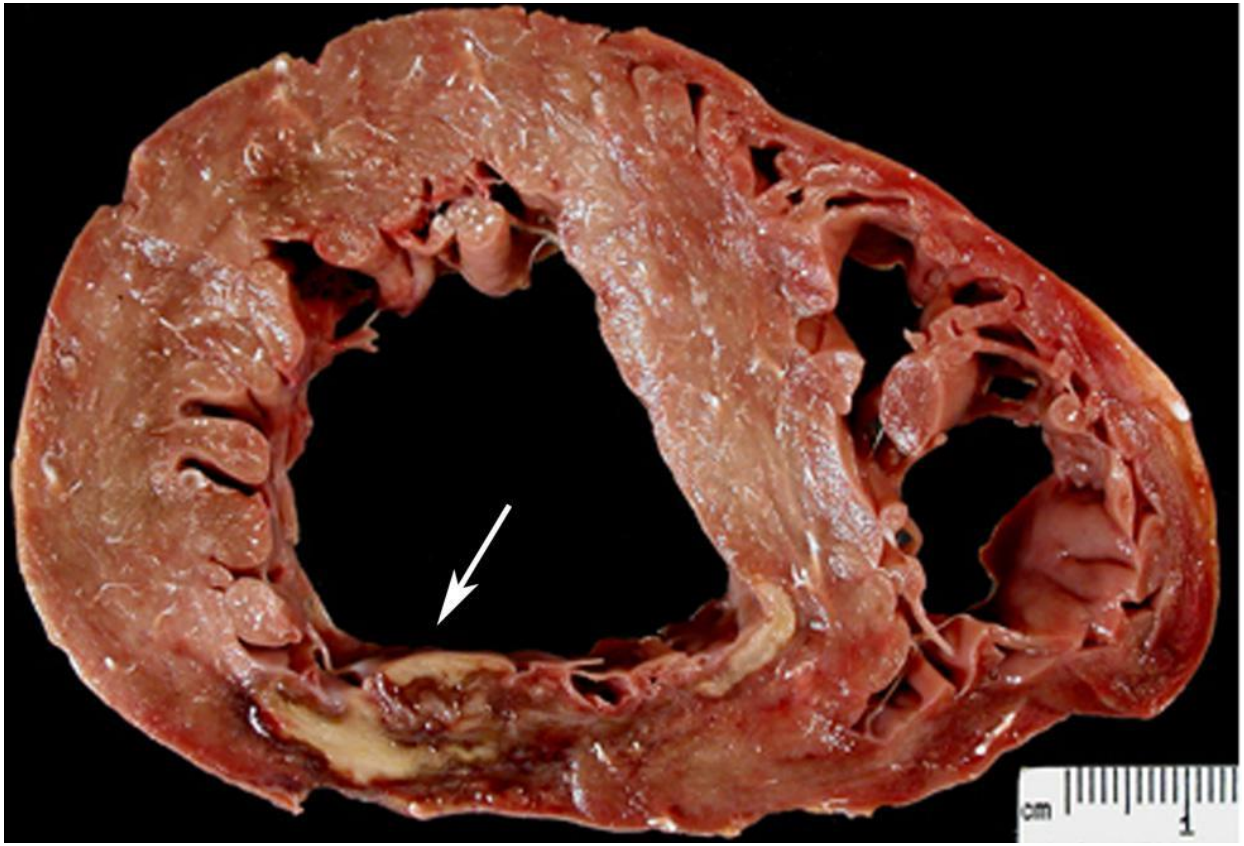
Free wall of left ventricle

Markedly thinned left ventricular apex

Septal wall

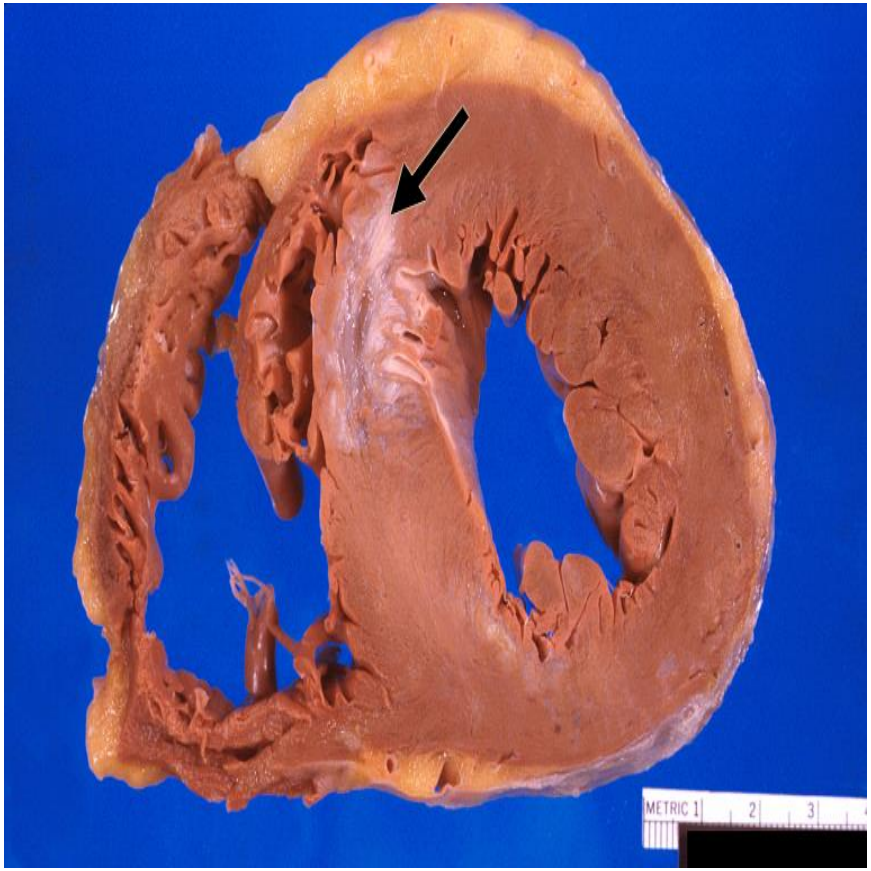
The heart is opened showing the left ventricle. There is a Massive Transmural Infarction extending around the entire wall between the white arrows.

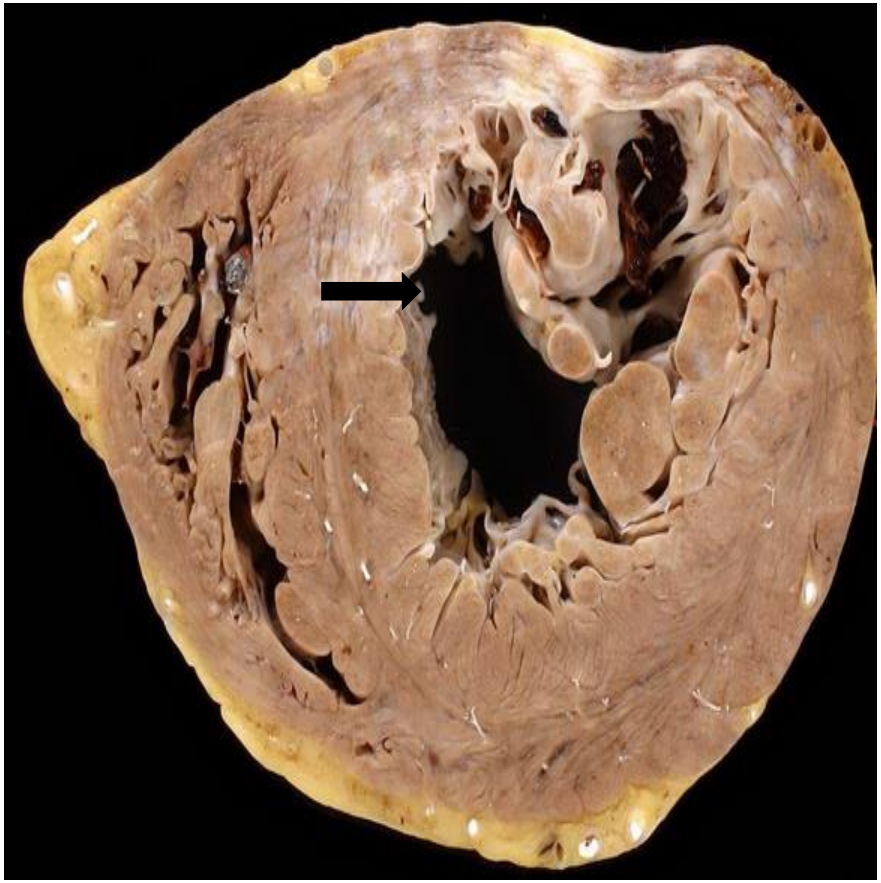
Myocardial Infarction



Acute myocardial infarct. At 3 days, there is a zone of yellow necrosis surrounded by darker hyperemic borders. The arrow points to an infarct in the wall of the left ventricle.

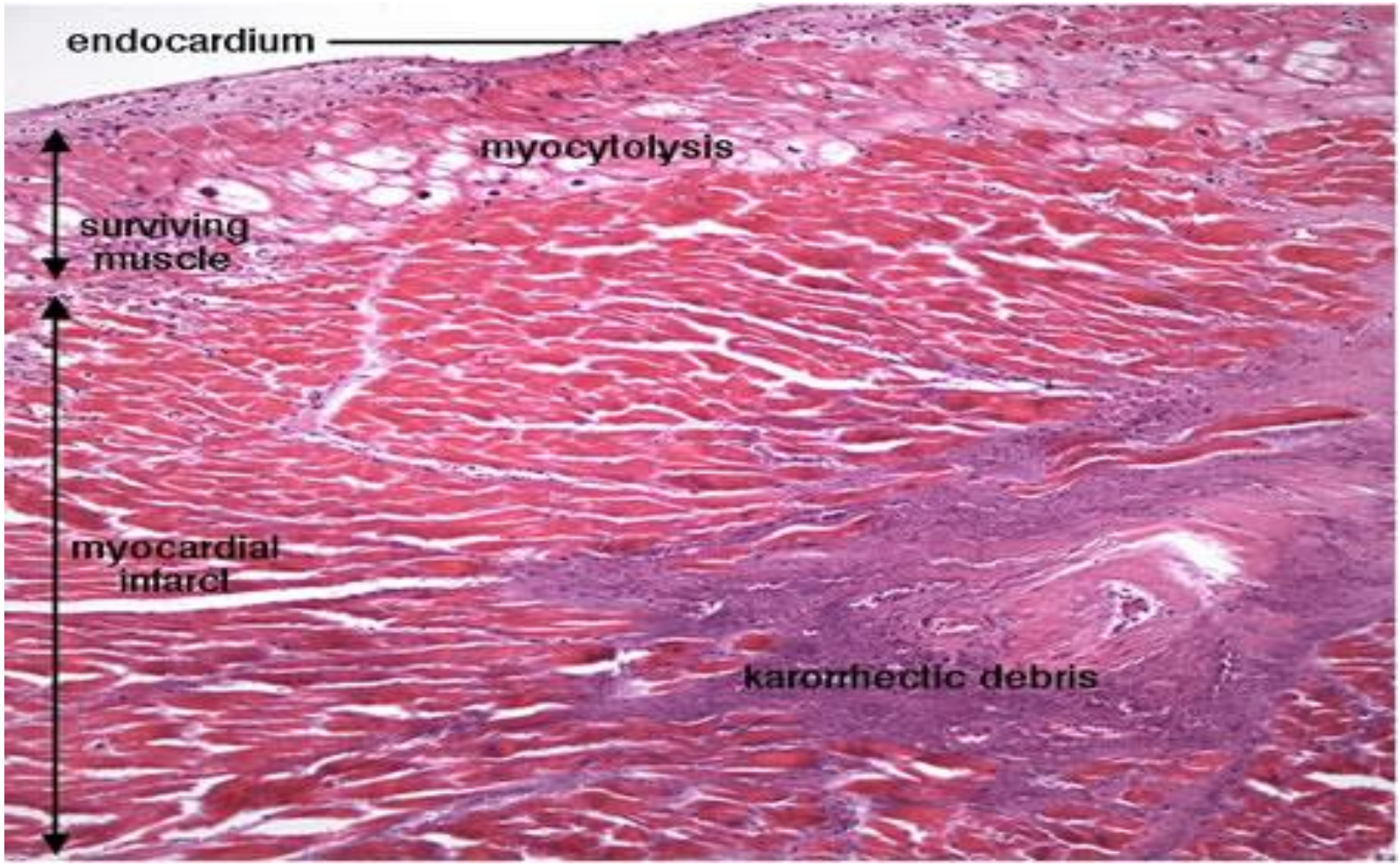
Myocardial Infarction





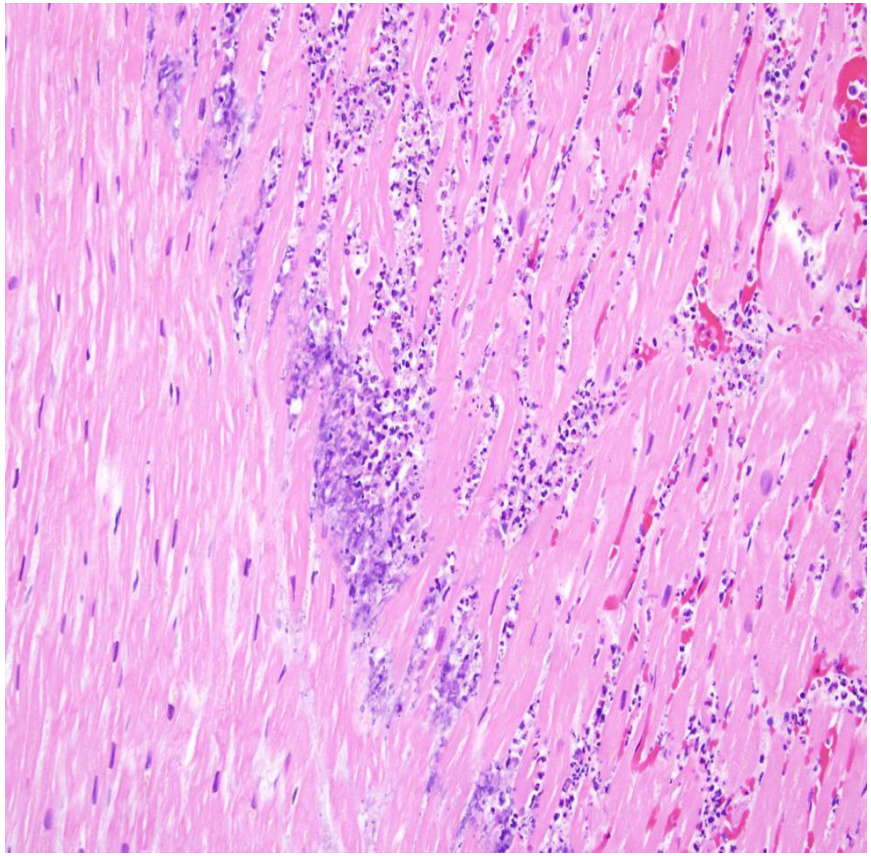
Healed myocardial infarct: cross section of the left and right ventricles shows a pale and irregular area of fibrosis (arrow) in the left ventricular wall. There is also increased thickness of the left ventricular wall (left ventricular hypertrophy).

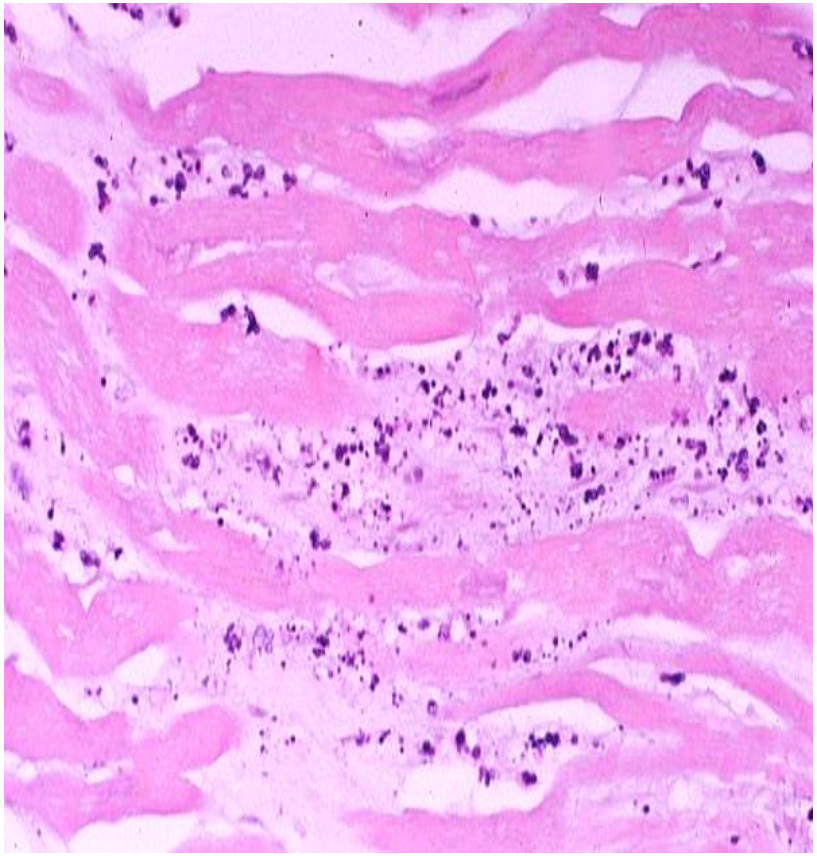
Myocardial Infarction - LPF



Transmural myocardial infarct at 2 weeks

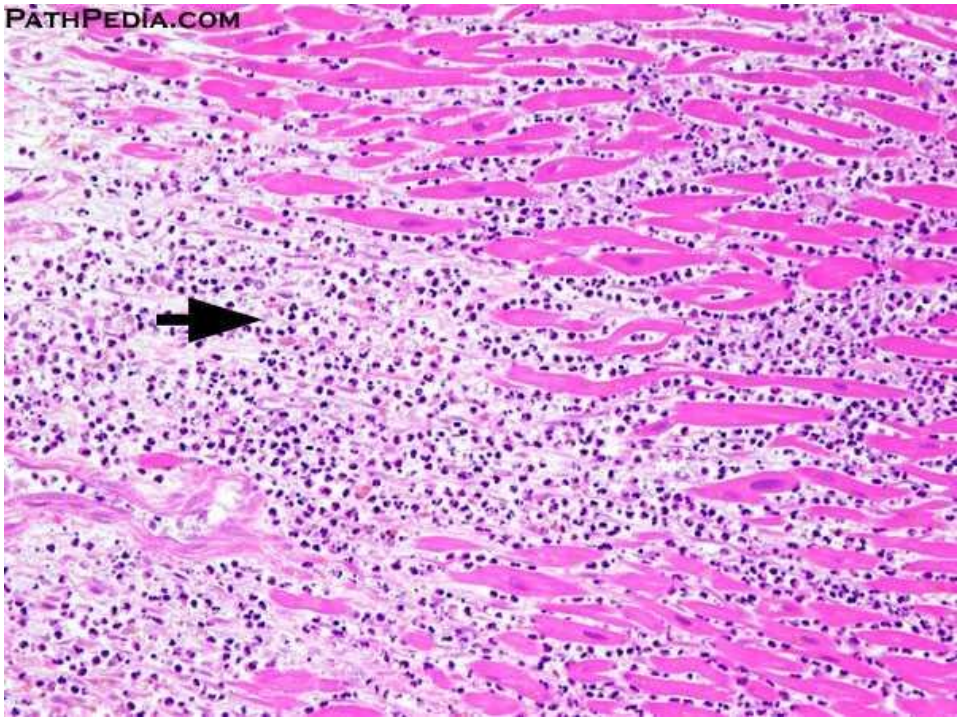
Myocardial Infarction

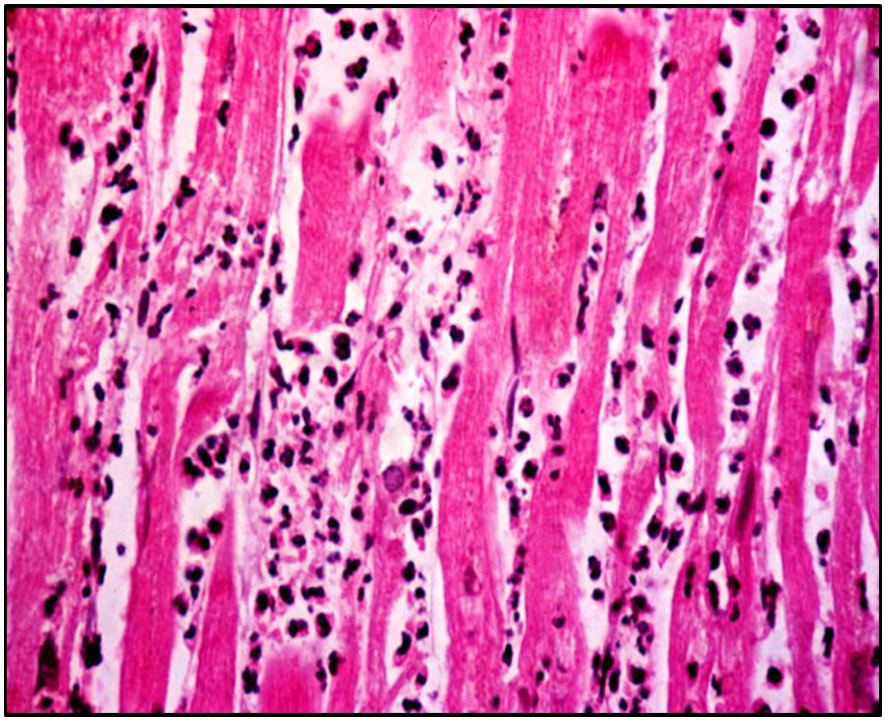




Acute myocardial infarct (after 24 hours) there is a neutrophilic infiltrate at the border of the infarct. Viable myocardium is at the left, and neutrophils are seen infiltrating the necrotic muscle. Note: the nuclei are not clearly visible in most of the necrotic cells.

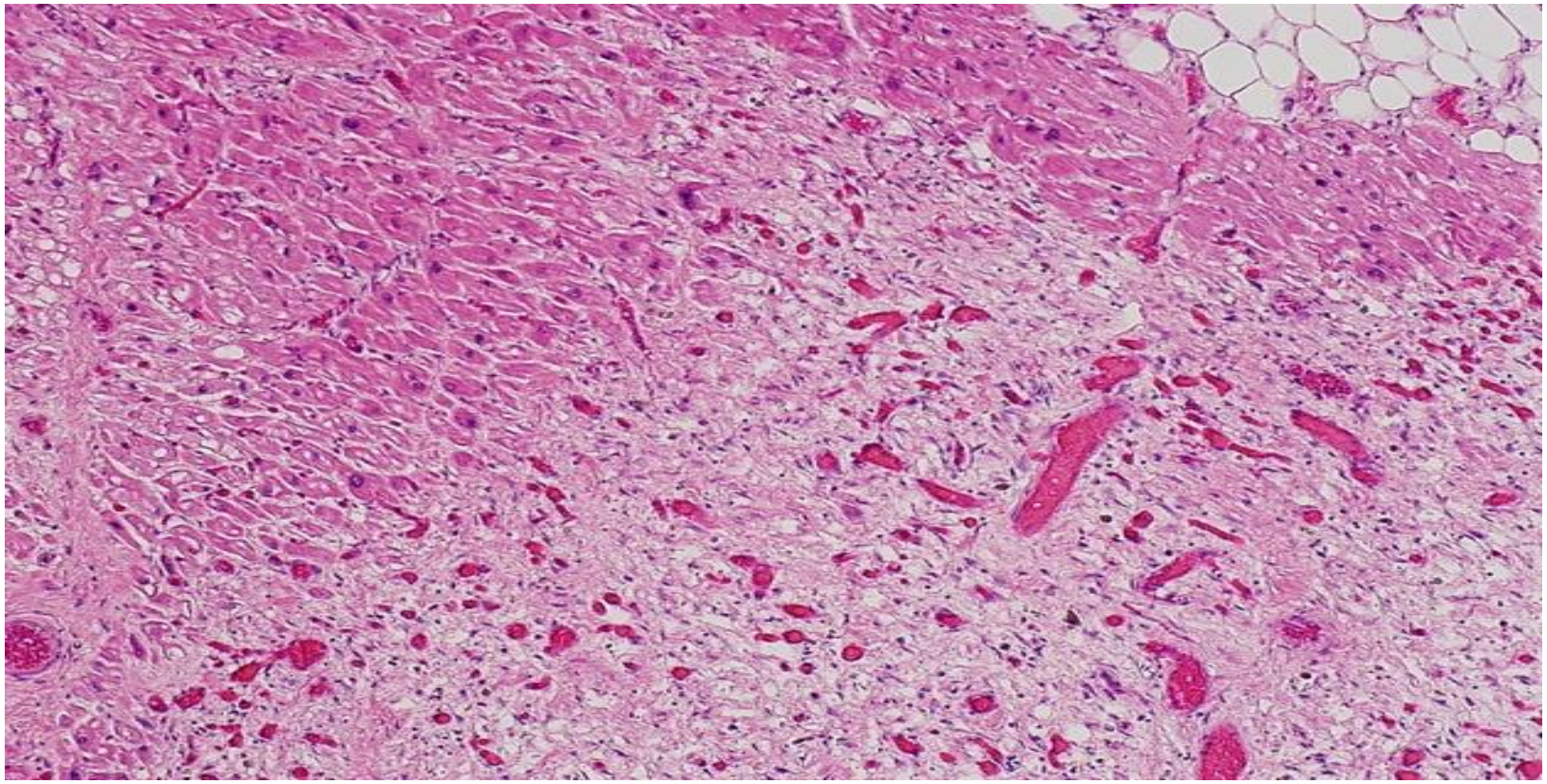
Myocardial Infarction





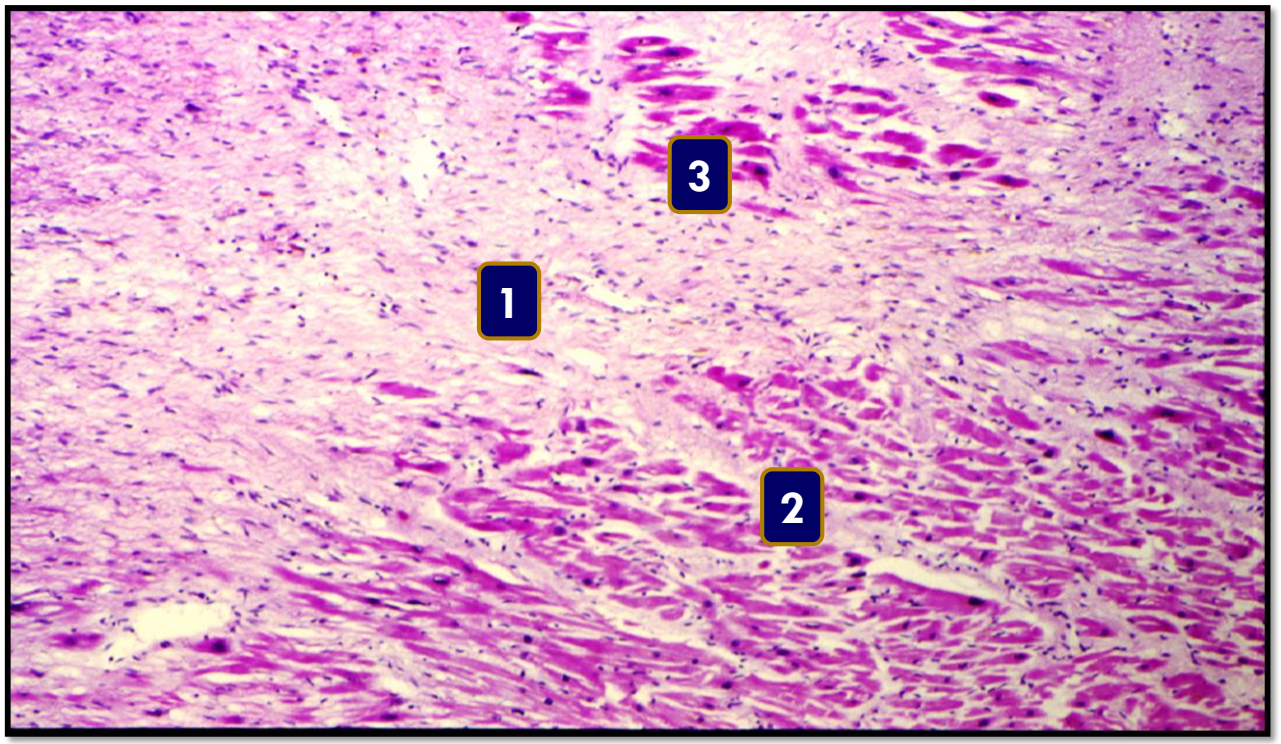
***ACUTE MYOCARDIAL INFARCTION:** a 3-day old acute infarct showing necrosis of myocardial cells (cardiomyocytes) infiltrated by a heavy neutrophilic infiltrate (arrow). The neutrophils release enzymes that help dissolve dead cell bodies which will be phagocytized by macrophages. With time the neutrophils begin to die and replaced by an influx of macrophages.*

Myocardial Infarction



Recent MI with early healing changes (3 weeks post MI) → shows granulation tissue (growth of capillaries and fibroblasts) and the collagen is being laid down to form a scar. The non-infarcted myocardium is present on the left and upper part of the picture.

Myocardial Infarction



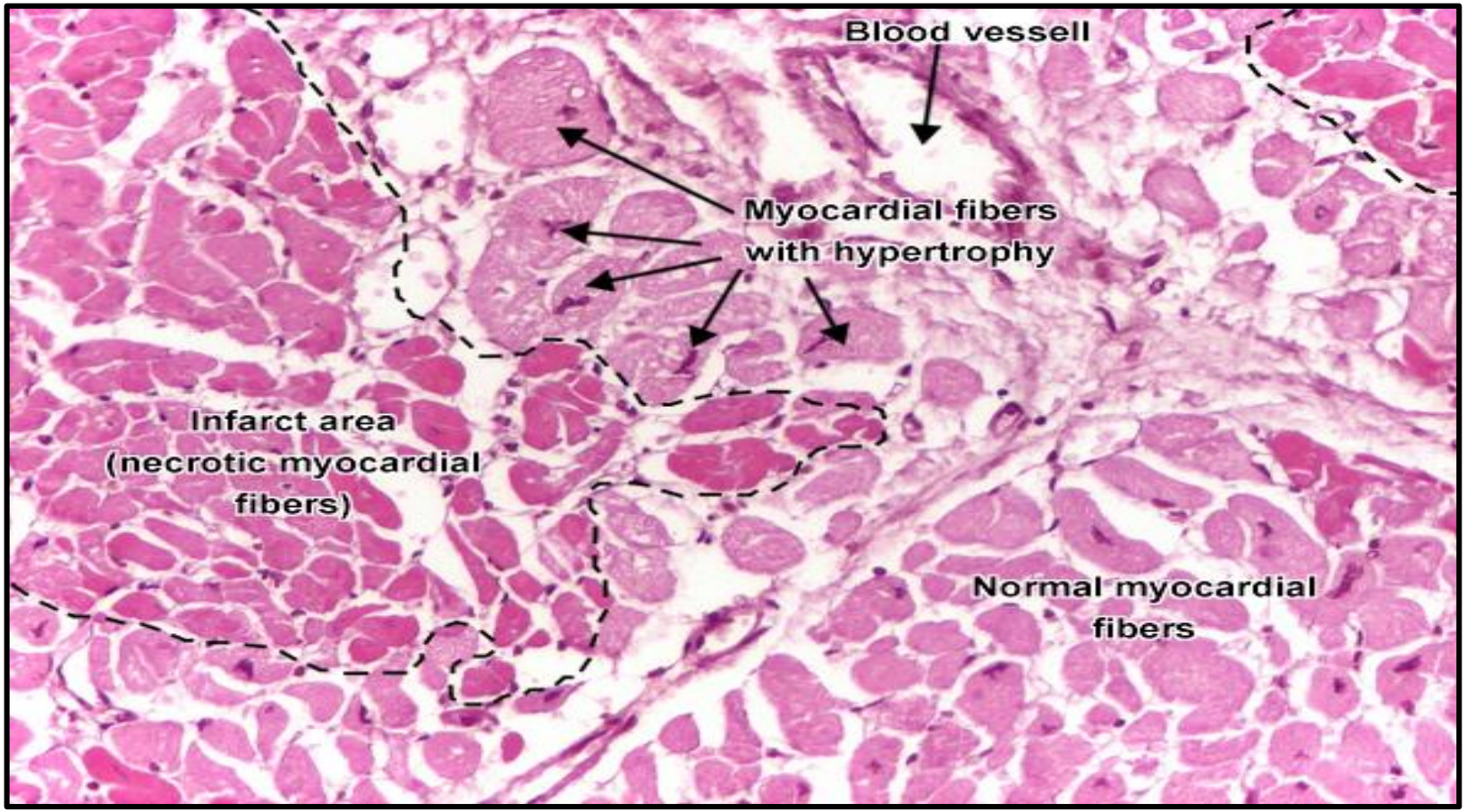
Healed myocardial infarct: in it there is replacement of the necrotic cells by a dense collagenous scar. The myocardium shows fibrosis with collagenization (scar) following healing of a myocardial infarction. Residual viable red myocardial fibers are present. This stage is reached about 2 months post MI.

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

2- Chronic ischemic fibrous scar replacing dead myocardial fibers .

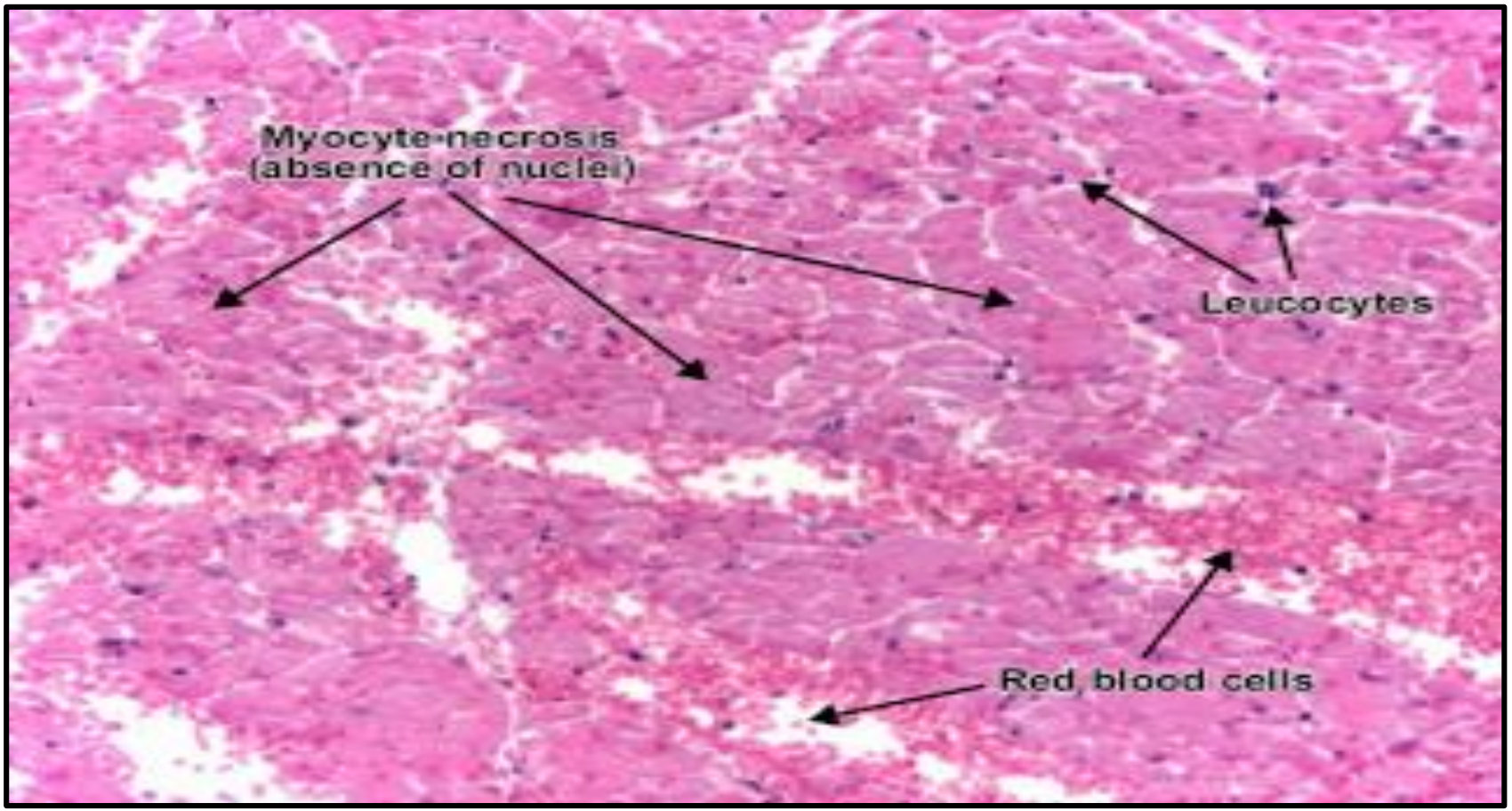
3- The remaining myocardial fibers show enlarged nuclei due to ventricular hypertrophy .

Myocardial Infarction - LPF



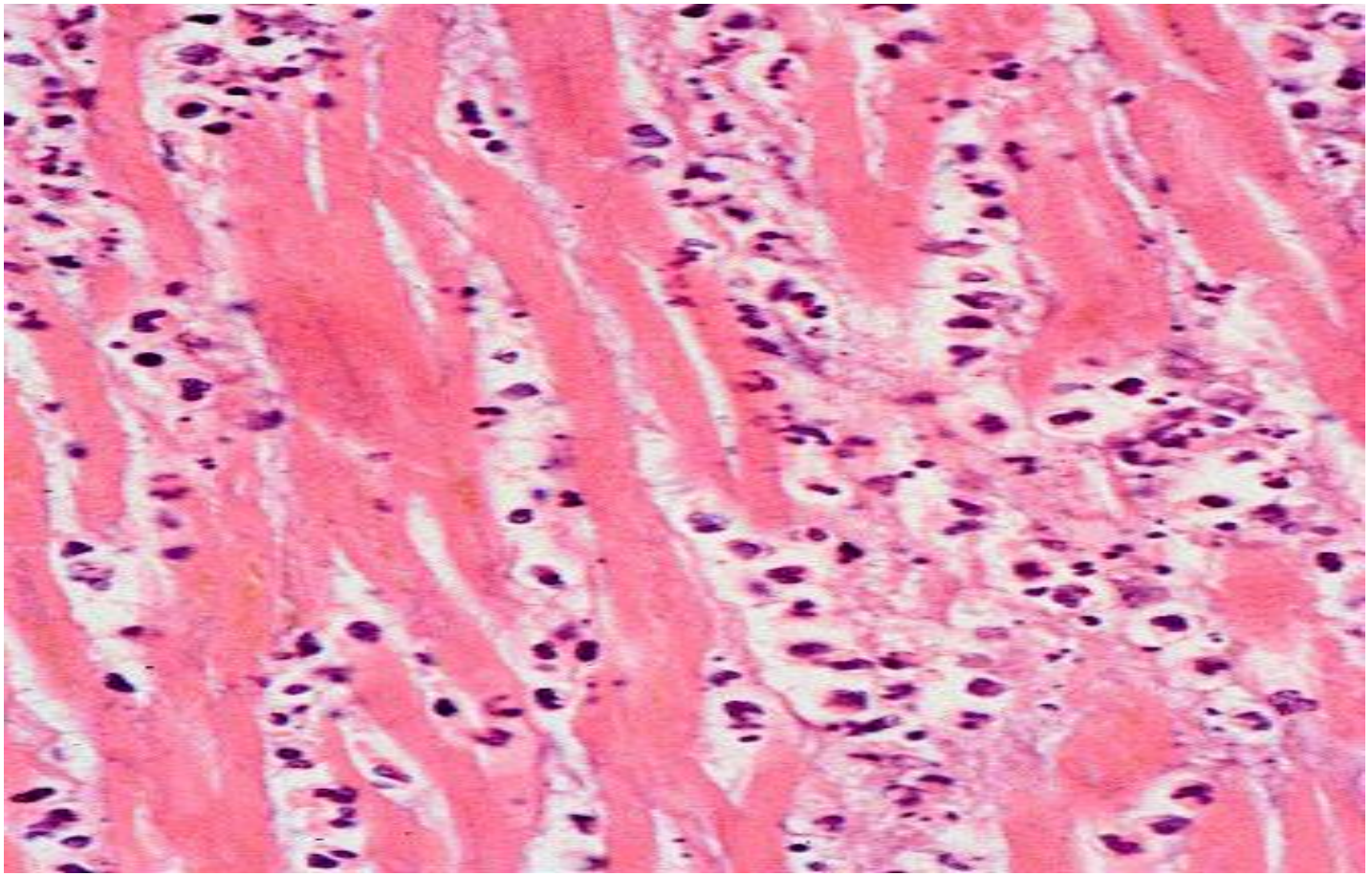
Myocardial infarct *- circumscribed area of ischemic necrosis - coagulative necrosis. In the first 12 - 24 hours, myocardial fibers are still well delineated, with intense eosinophilic (pink) cytoplasm, but lost their transversal striations and the nucleus (left side of the picture). Notice a few myocardial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

Myocardial Infarction - LPF



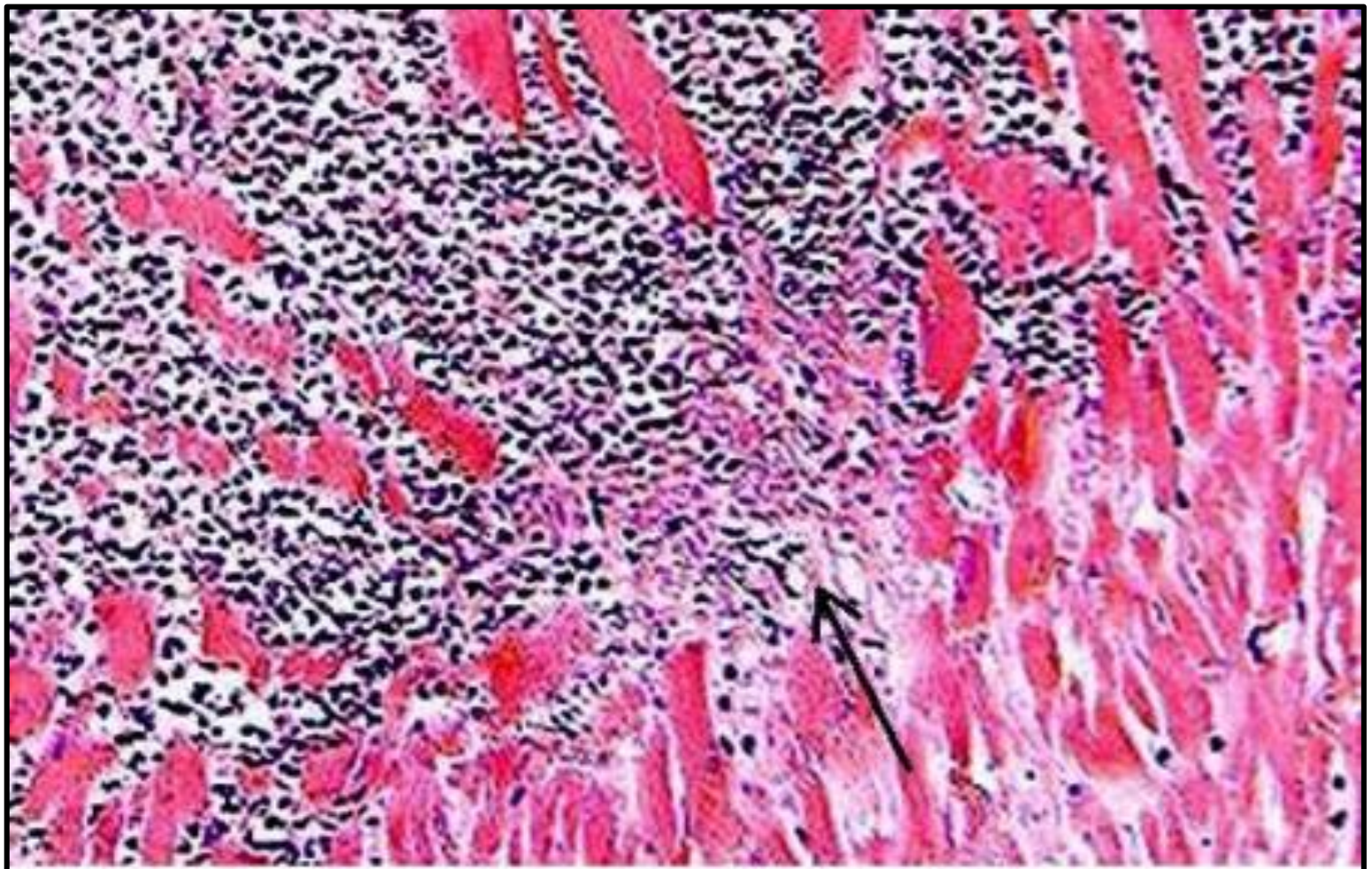
***Recent myocardial infarct** (in the first 12 - 24 hours): myocardial fibers are still well delineated, with intense eosinophilic (pink) cytoplasm, but lost their transversal striations and the nucleus. The interstitial space may be infiltrated with red blood cells.*

Myocardial Infarction - HPF



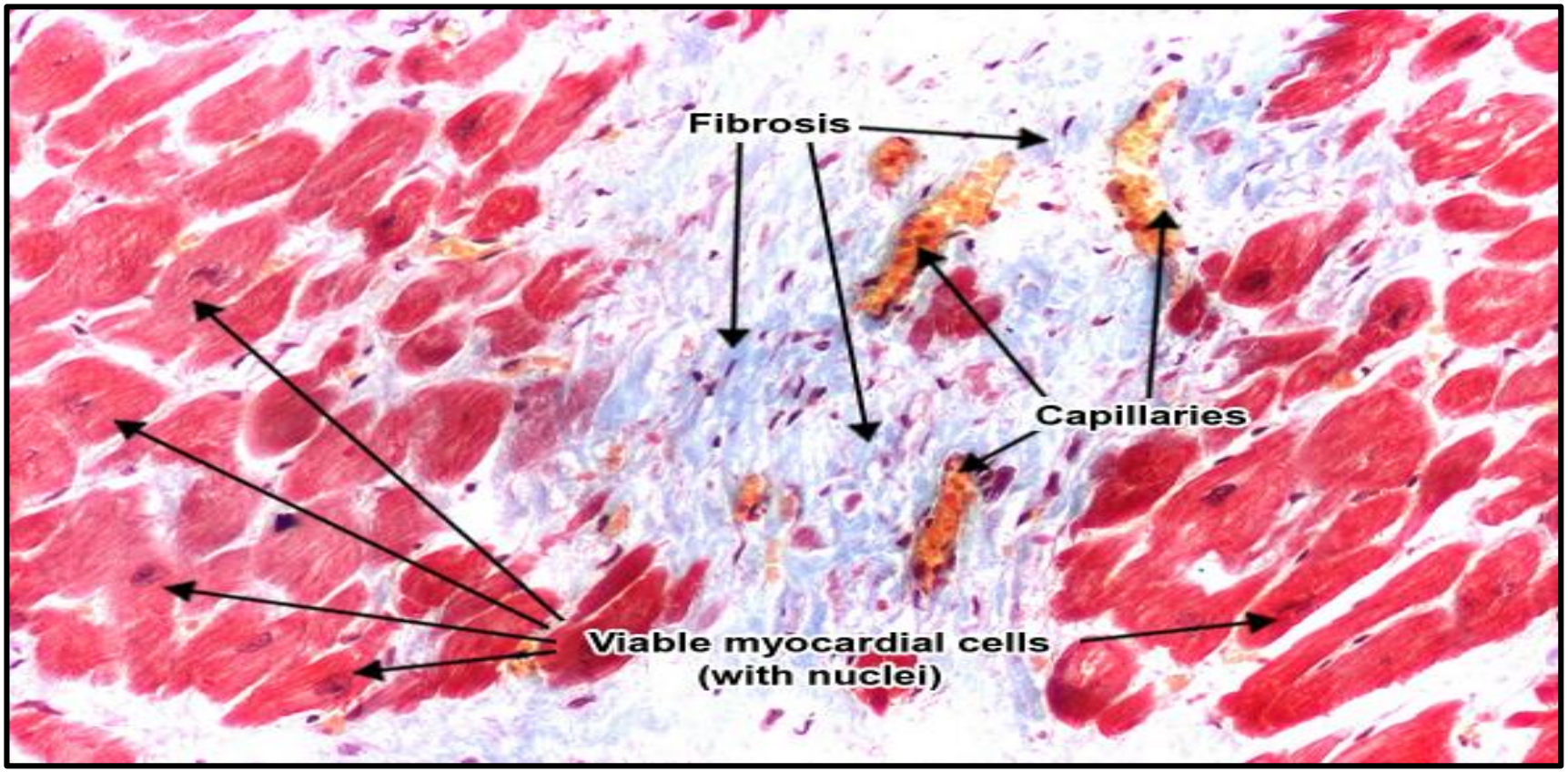
***Acute myocardial infarct**, histology. This 1-3 day infarct shows coagulation necrosis of myocardial cells and is infiltrated with polymorphnuclear leukocytes.*

Myocardial Infarction - HPF



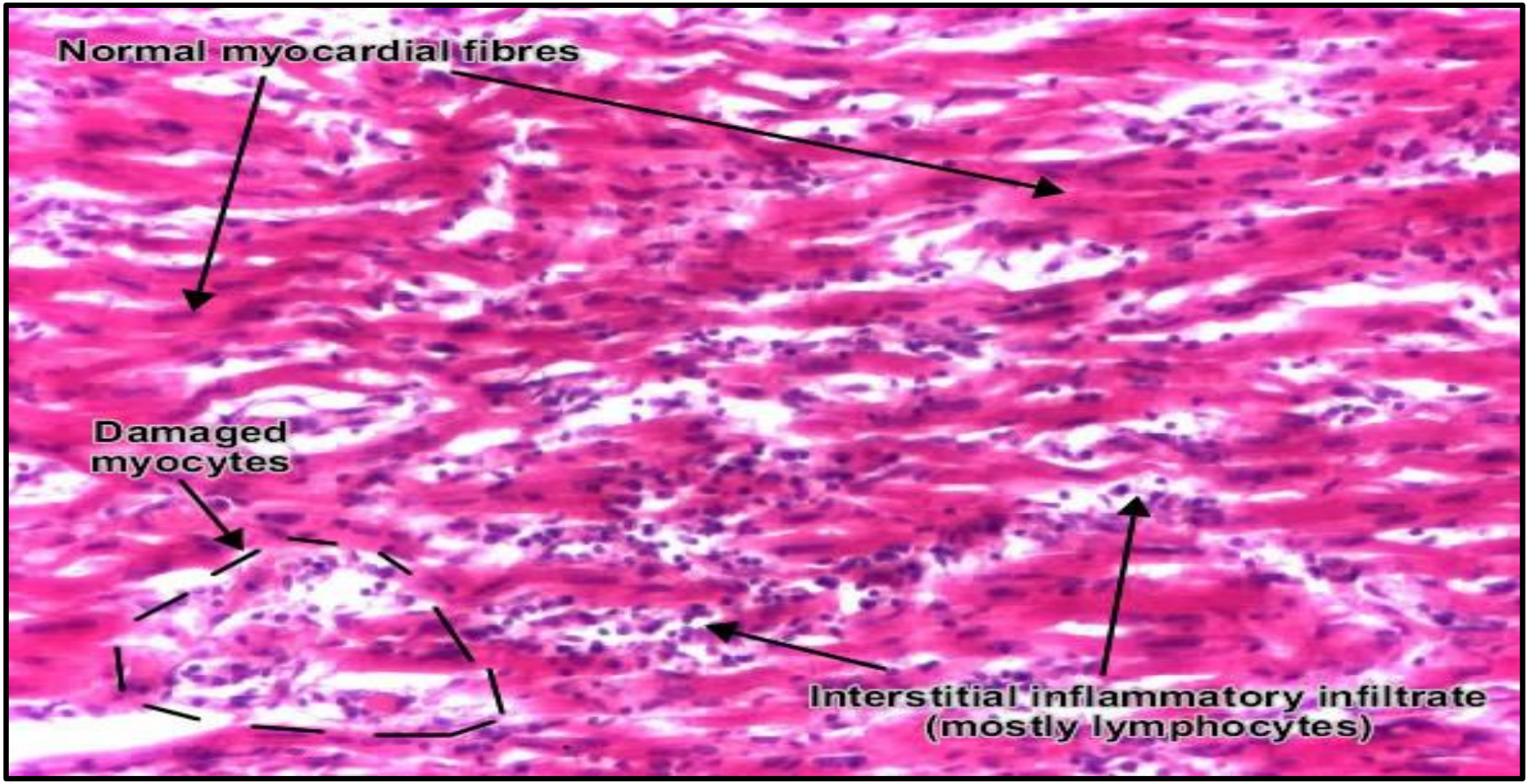
Microscopic image of Myocardial Infarction

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.*


THE END