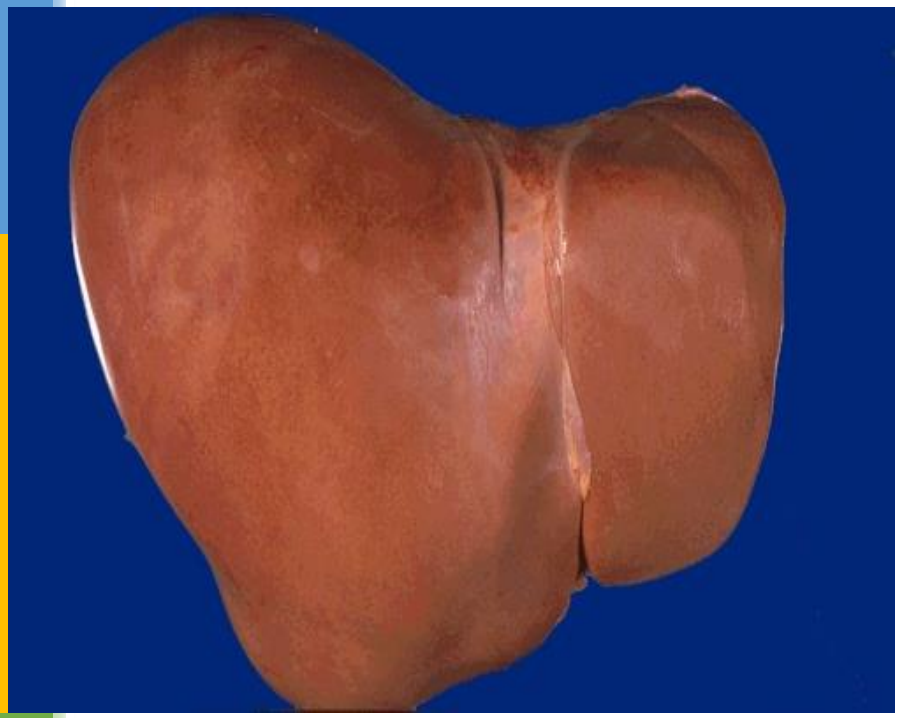
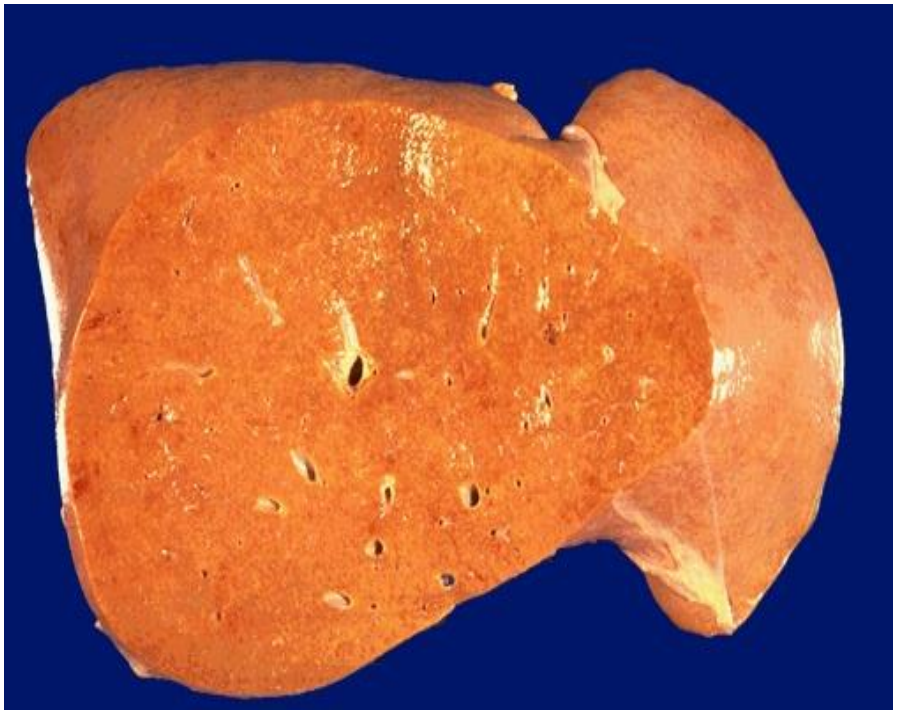
Remaining cell injury Foundation Block Practical

Fatty liver and calcification

1- FATTY LIVER (STEATOSIS)

Normal Liver & Cut Section of Fatty Liver

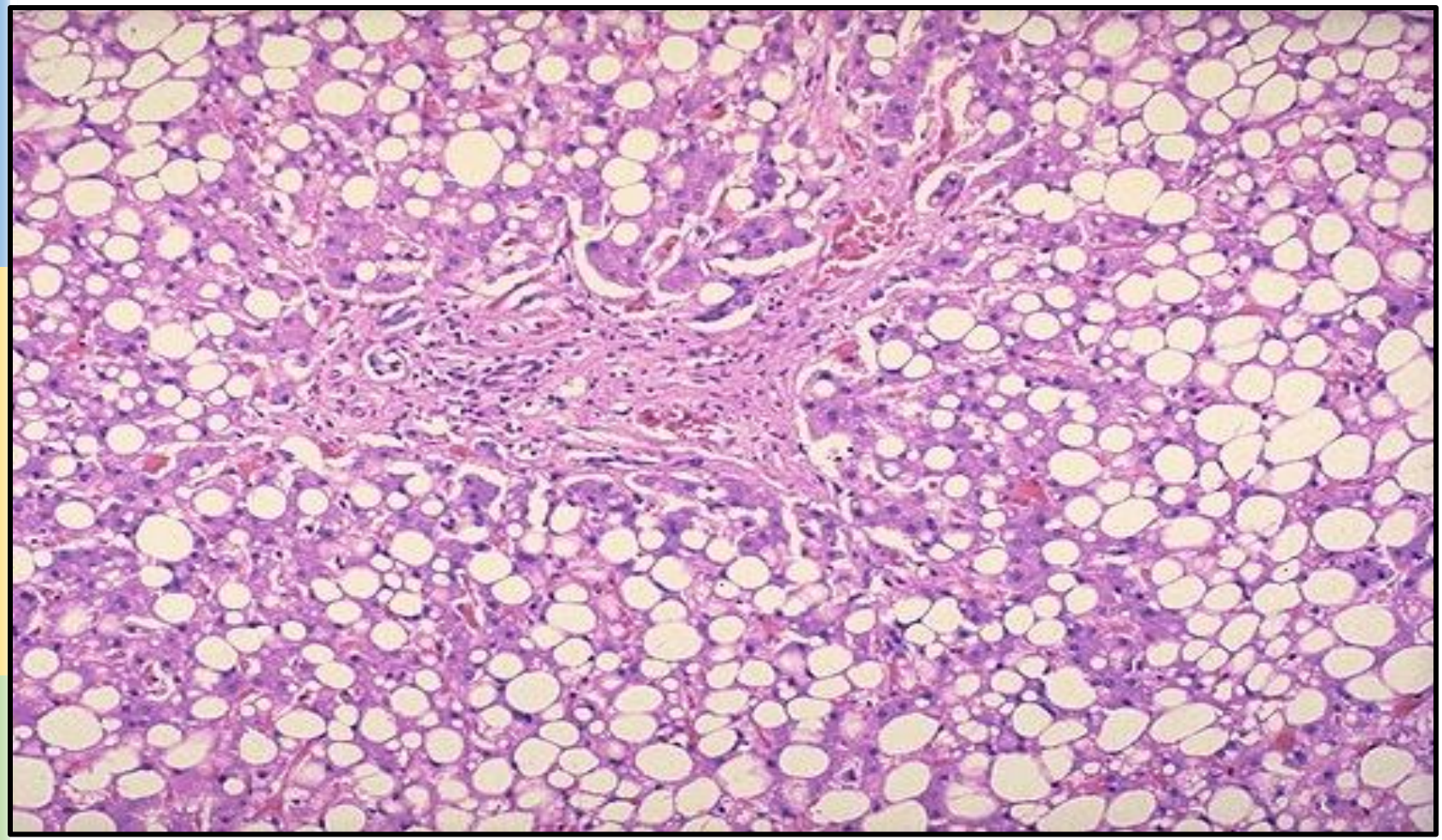




Normal Liver : This is the external surface of a normal liver. The color is brown and the surface is smooth

Steatosis : This liver is slightly enlarged and has a pale yellow appearance, seen both on the capsule and cut surface

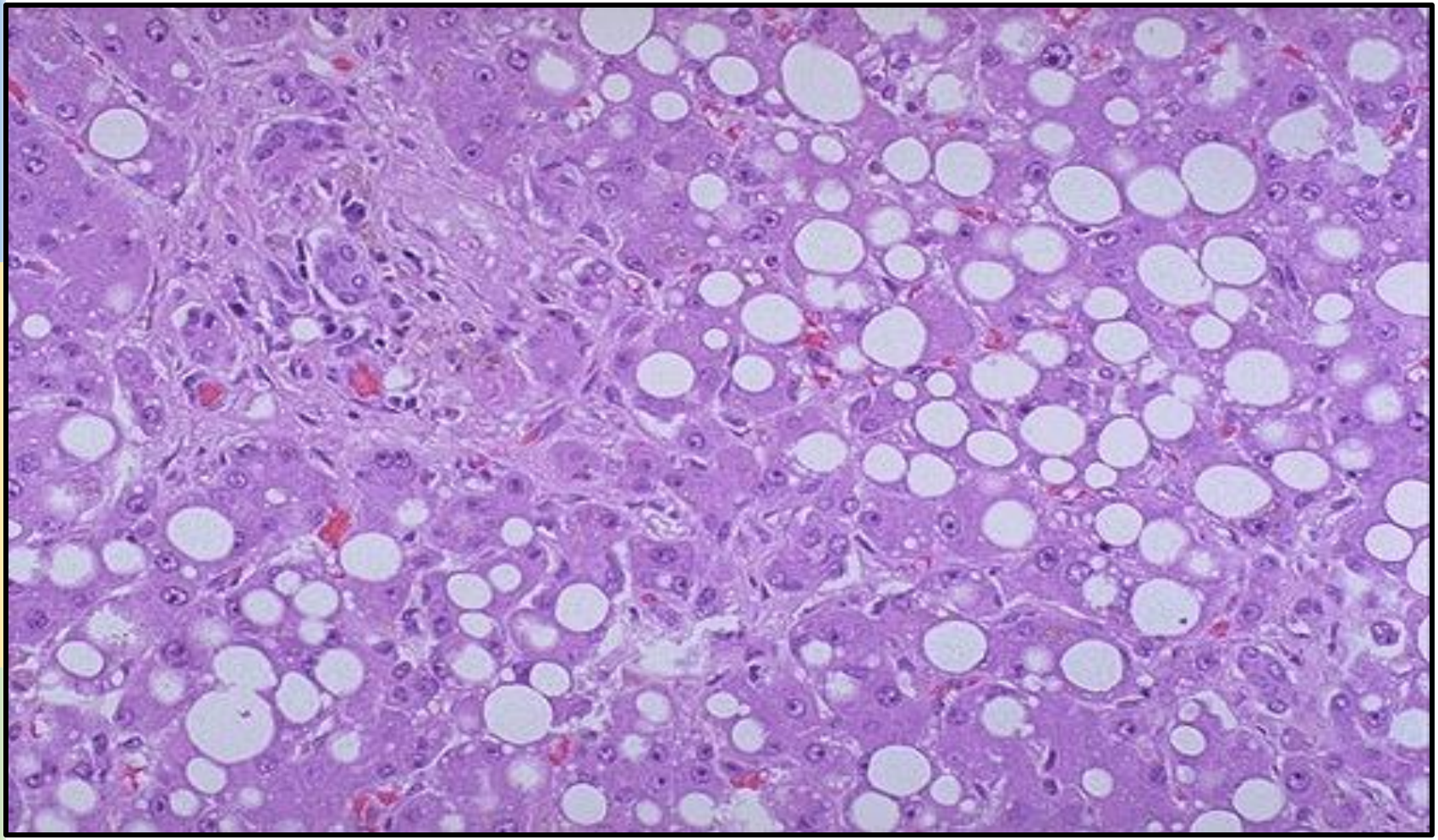
Steatosis – Fatty Liver



This is the histologic appearance of hepatic fatty change. Liver Cells containing fat vacuoles

The most common cause of fatty change in developed nations is alcoholism. Other causes are: Morbid obesity and Hepatitis C

Steatosis – Fatty Liver

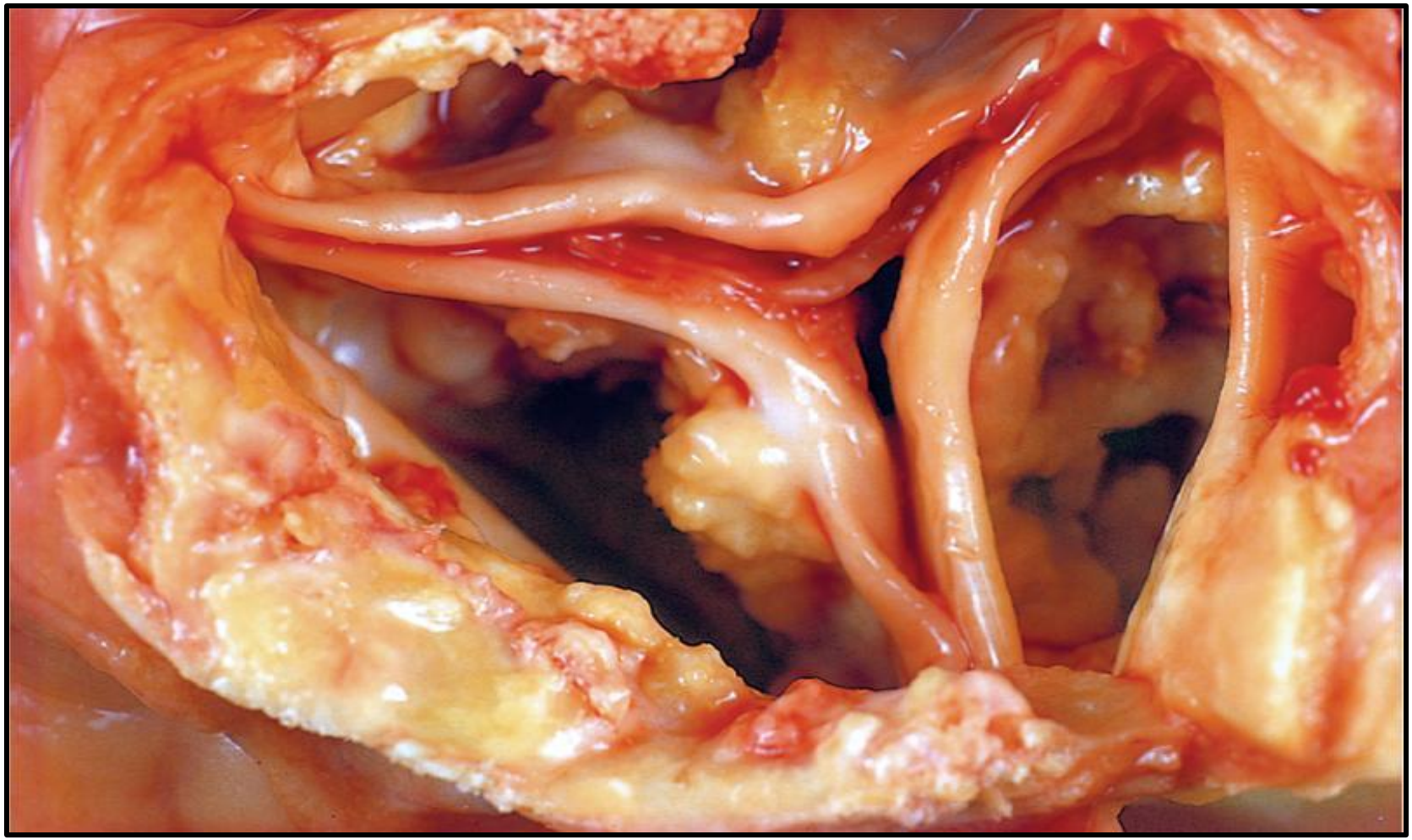


Here are seen the lipid vacuoles within hepatocytes. The lipid accumulates when lipoprotein transport is disrupted and/or when fatty acids accumulate. Alcohol is the most common cause

7 - Dystrophic calcification

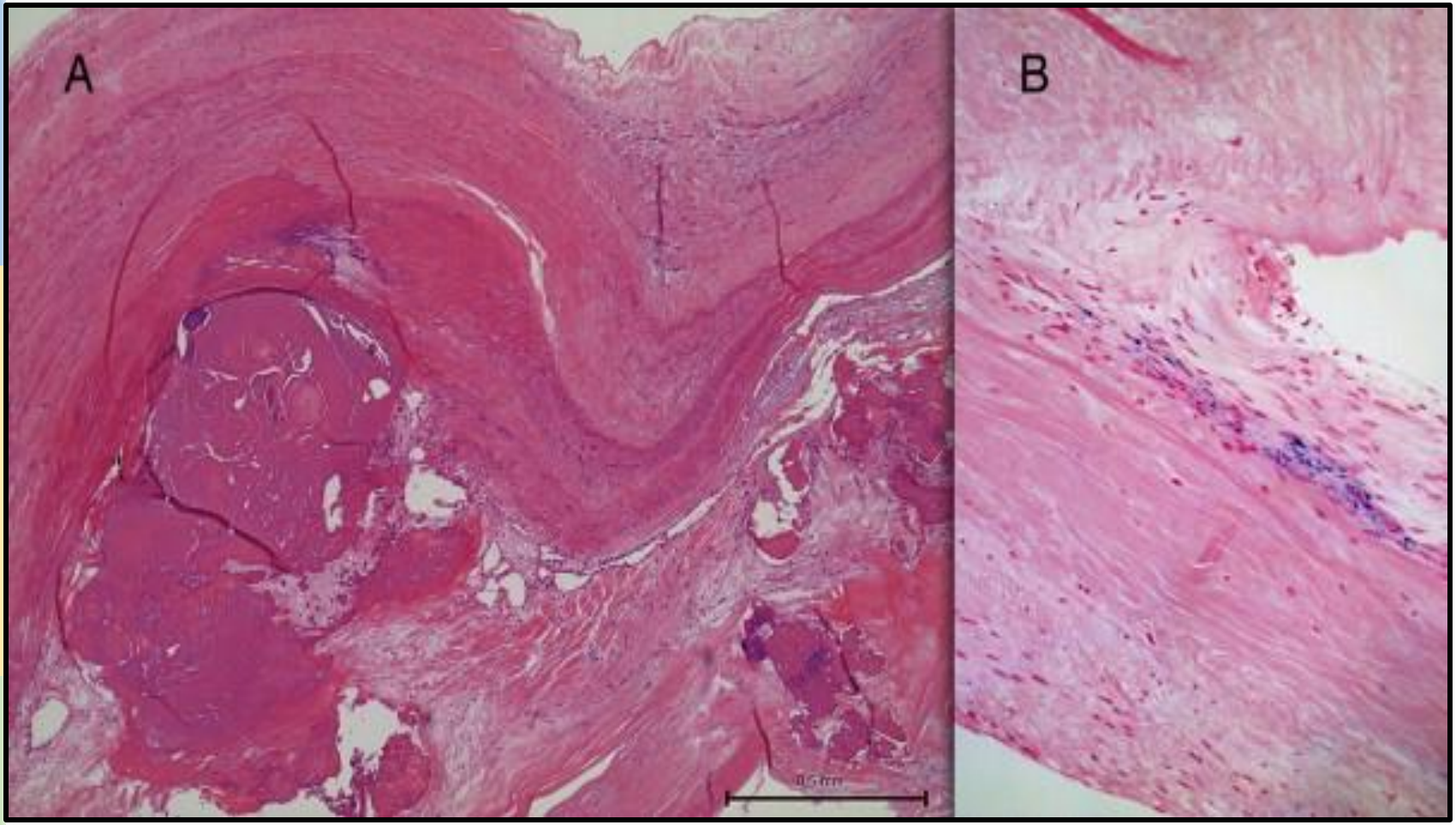
(Aortic valve – Stomach - Skin)

Dystrophic calcification of Aortic Valve



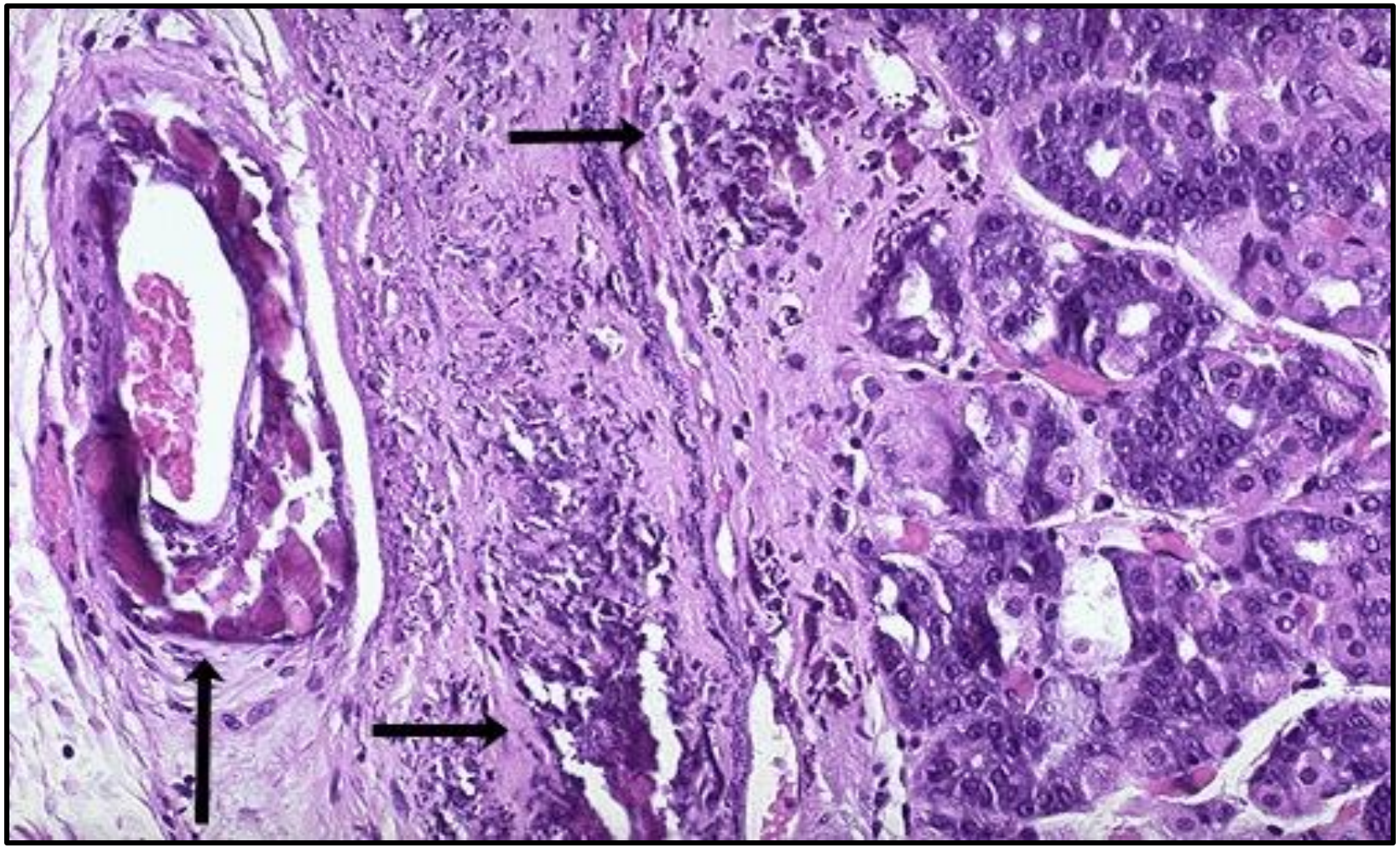
View looking down onto the unopened aortic valve in a heart with calcific aortic stenosis. It is markedly narrowed (stenosis). The semilunar cusps are thickened and fibrotic, and behind each cusp are irregular masses of piled-up dystrophic calcification

Dystrophic calcification of Aortic Valve



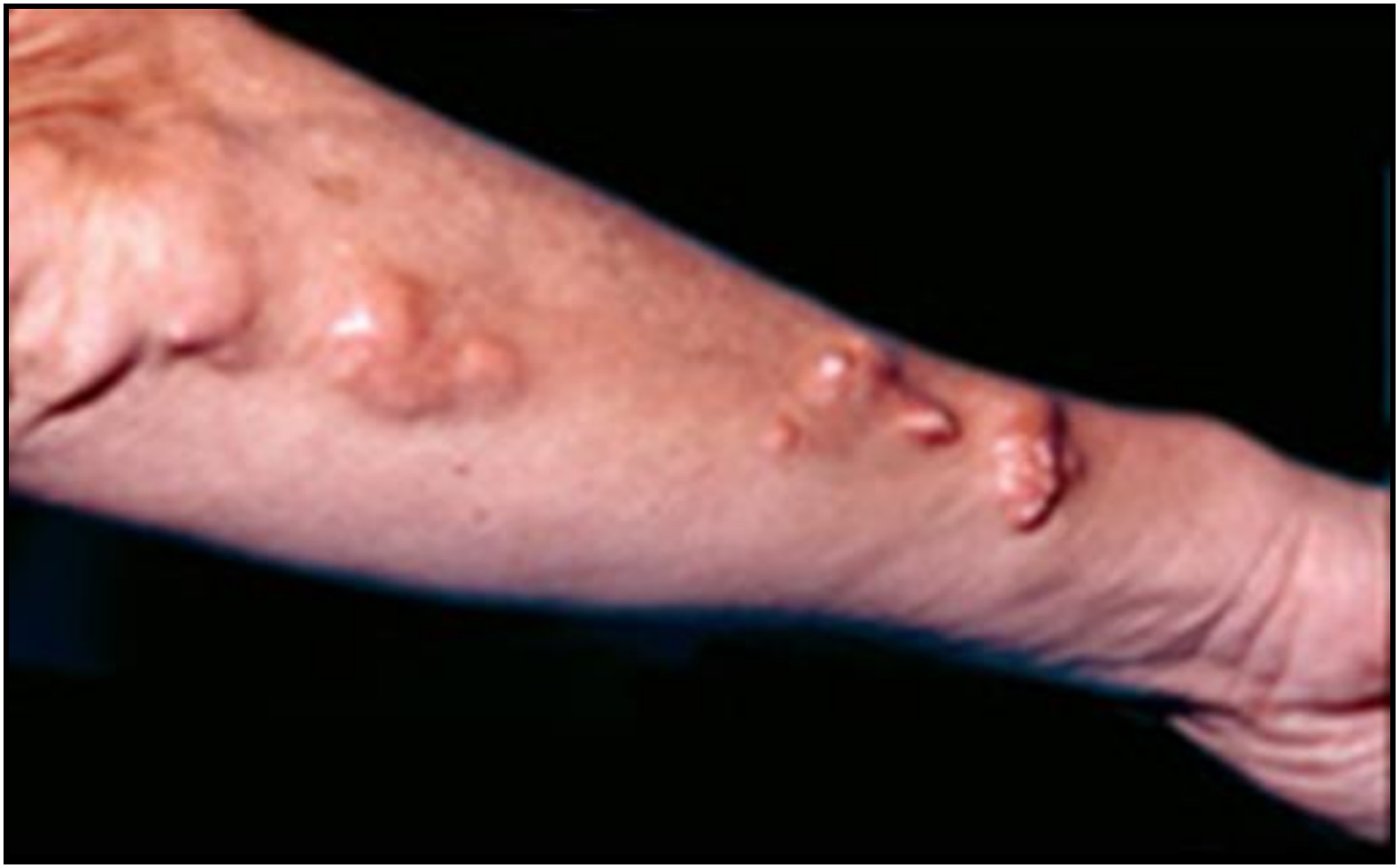
Aortic valve. Fibrosis with some lymphocytes and dystrophic calcification (A) hematoxylin and eosin; 1.25× objective magnification; and siderosis (B) Berlin blue 40× objective magnification

Dystrophic Calcification of Stomach



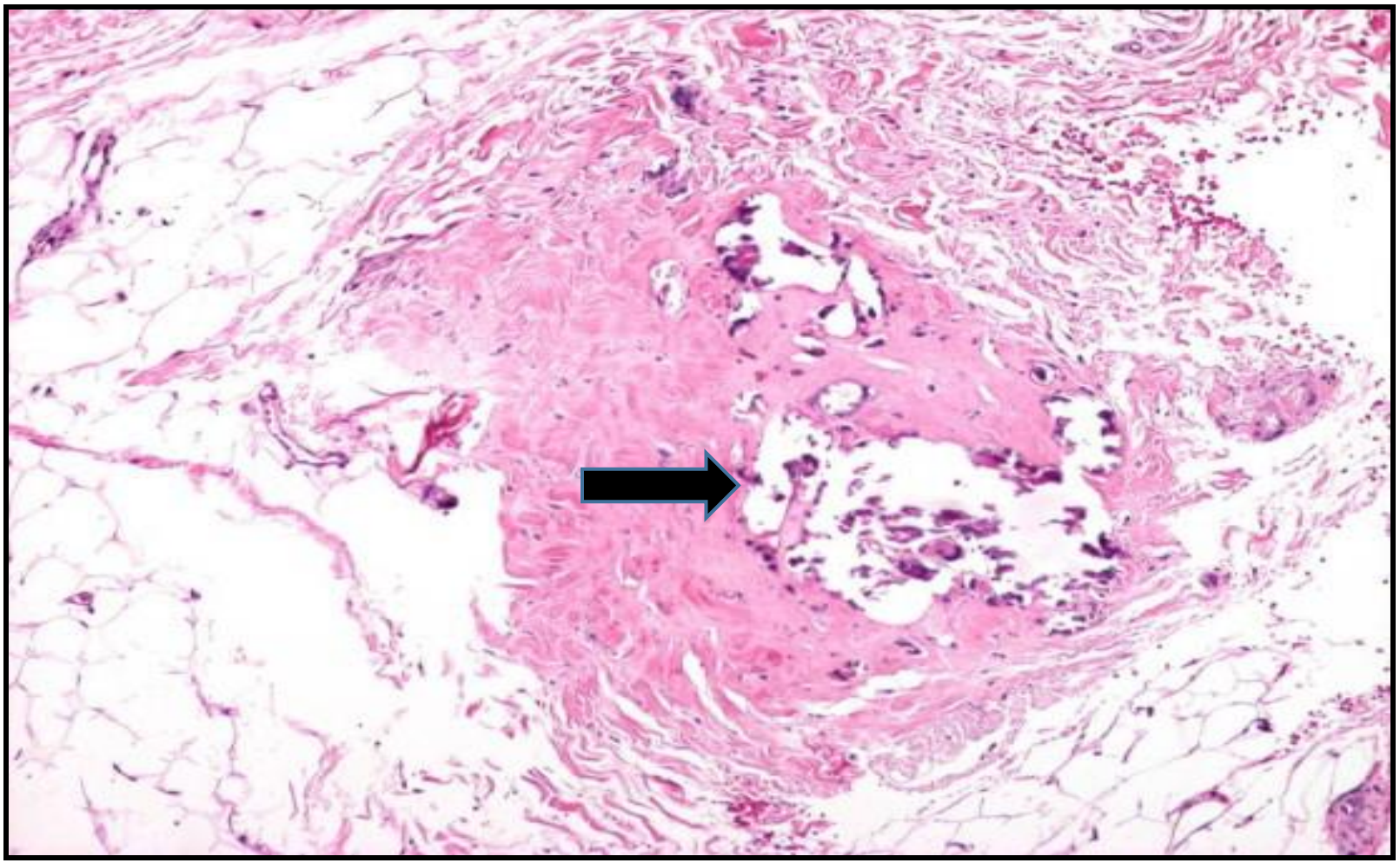
This is a dystrophic calcification in the wall of the stomach. At the far right is an artery with calcification in its wall. There are also irregular bluish-purple deposits of calcium in the submucosa

Dystrophic Calcification of the Skin



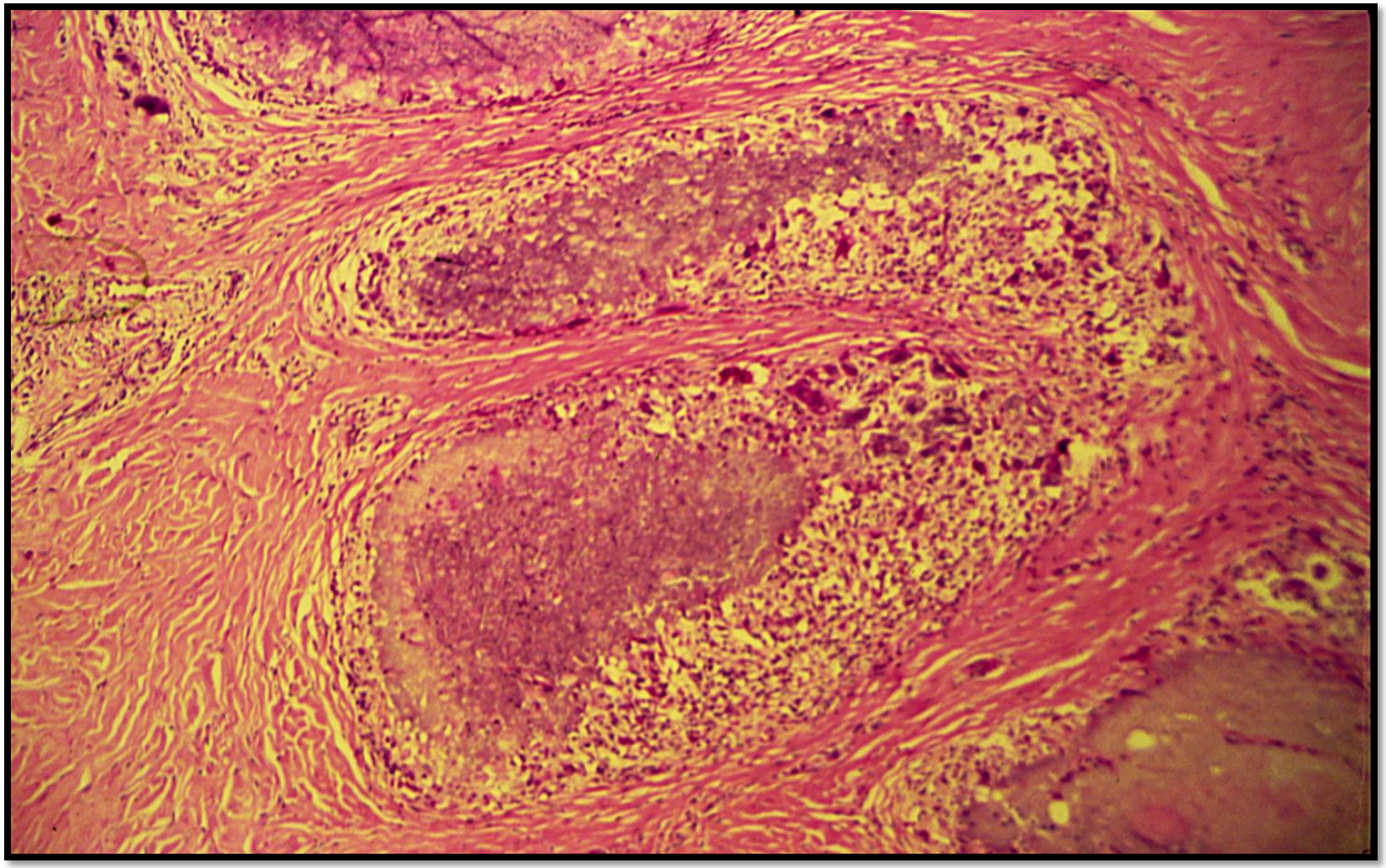
Multiple erythematous hard papules in linear configuration on the extensor aspect of the arm. Within the lesion there were several 2-5 mm white calcifications

Dystrophic Calcification of the Skin



Calcifying panniculitis with fibrosis of the subcutaneous connective tissue septae, adjacent inflammation containing plasmocytes and lymphocytes, and a deposit of calcification (arrow).

Dystrophic Calcification of the Skin



Irregular blue granular deposits of calcium in the dermis surrounded by fibrous tissue and foreign body giant cell reaction

Foundation Block Practical

Thromboembolic disorder

Additional information added by Sufia Husain

1- Organizing Thrombus

ADDITIONAL 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 or vein.
- It is intravascular coagulation of blood and it can cause significant interruption to blood flow.

Pathogenesis: 3 primary factors that predispose to thrombus formation, Called as *Virchow triad*:

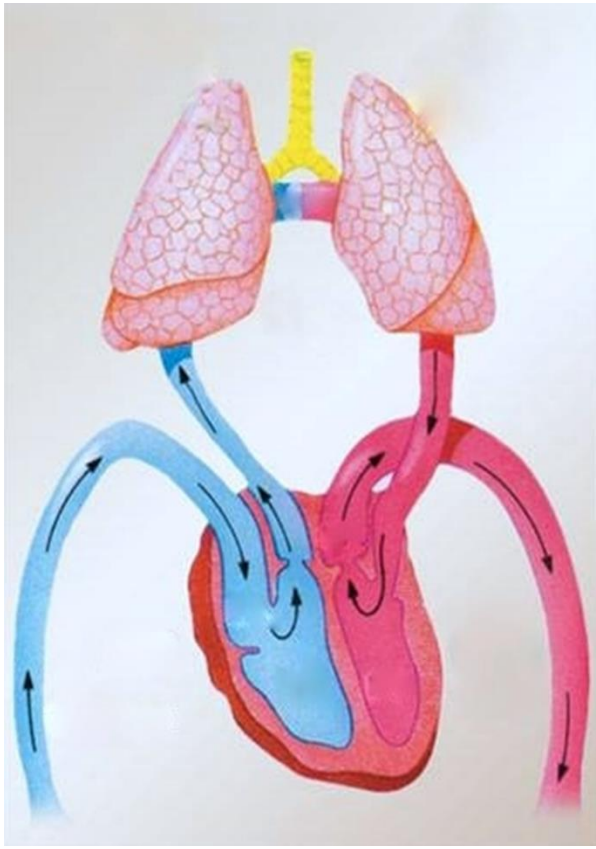
(1) endothelial injury

(2) stasis or turbulence of blood flow

(3) blood hypercoagulability

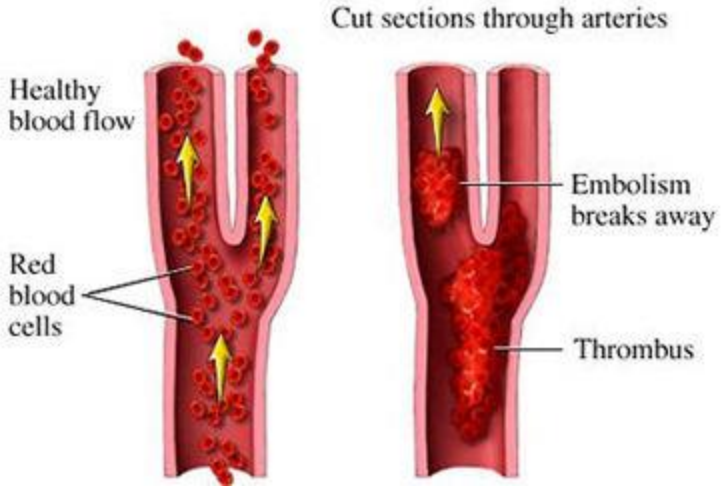
ADDITIONAL INFORMATION:

- Thrombi may develop anywhere in the cardiovascular system, the cardiac chambers, valve cusps, arteries, veins, or capillaries. They vary in size and shape, depending on the site of origin.
- Thrombi in the artery or heart usually begin at a site of endothelial injury (e.g., atherosclerotic plaque) or turbulence (vessel bifurcation). Thrombi in vein occur in sites of stasis.
- A thrombus is made up of fibrin, platelets and red blood cell. Inflammatory cells can also be present.
- When formed in the heart or aorta, thrombi may have laminations, called **lines of Zahn**; these are produced by alternating pale layers of platelets admixed with some fibrin and darker layers containing more red cells.



ADDITIONAL INFORMATION: EMBOLISM

- The growing end of thrombi may not be well attached (particularly in veins) is prone to detachment and fragmentation, creating an **embolus**.
- *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 lodge 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.



ADDITIONAL INFORMATION: EMBOLISM

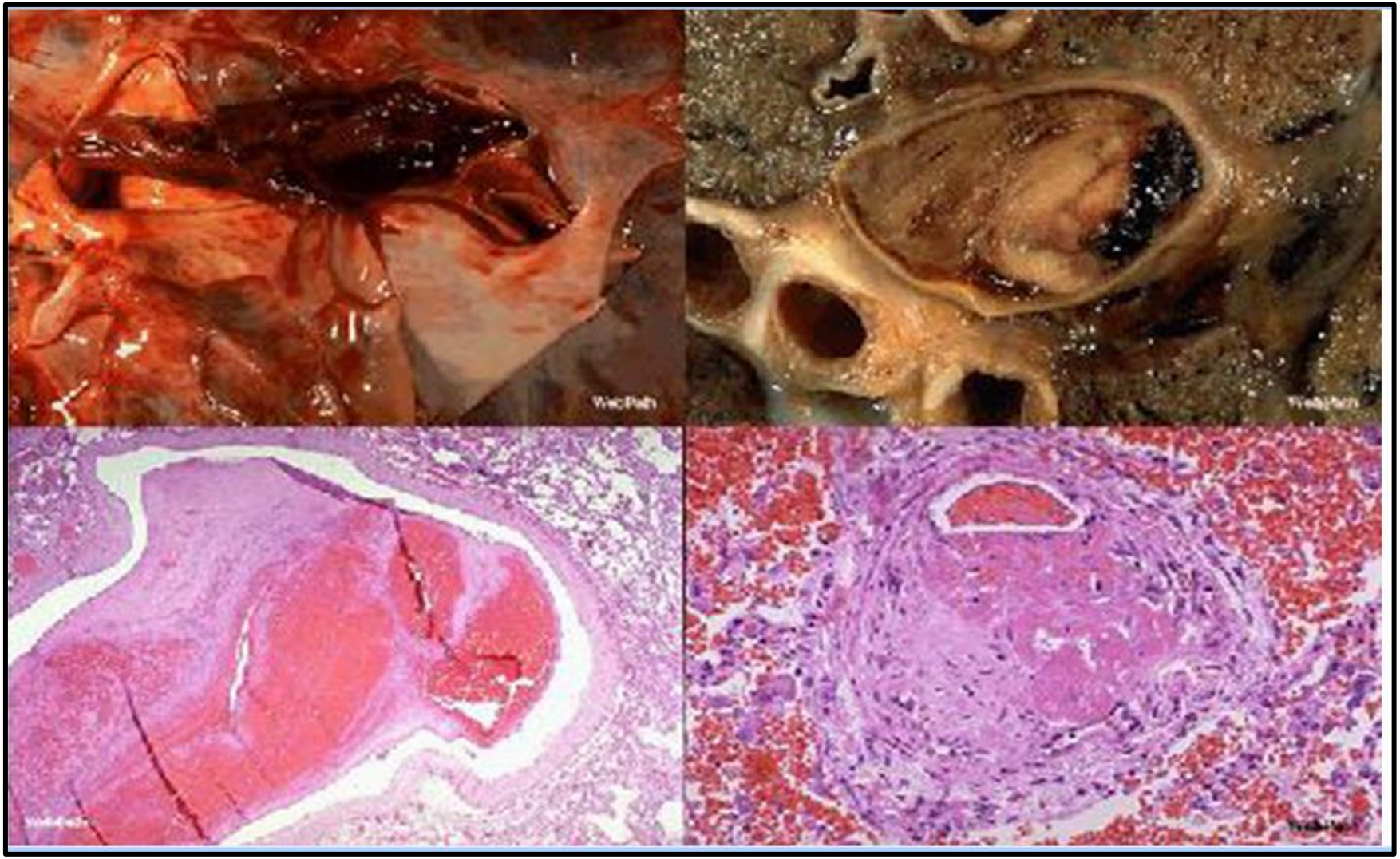
PULMONARY THROMBOEMBOLISM

- Depending on size of embolus, it may occlude main pulmonary artery, or impact across the bifurcation *(saddle embolus)*, or pass out into the smaller, branching arterioles
- Most pulmonary emboli (60% to 80%) are clinically silent because they are small. The large pulmonary emboli e.g saddle emboli can cause sudden death.

SYSTEMIC THROMBOEMBOLISM

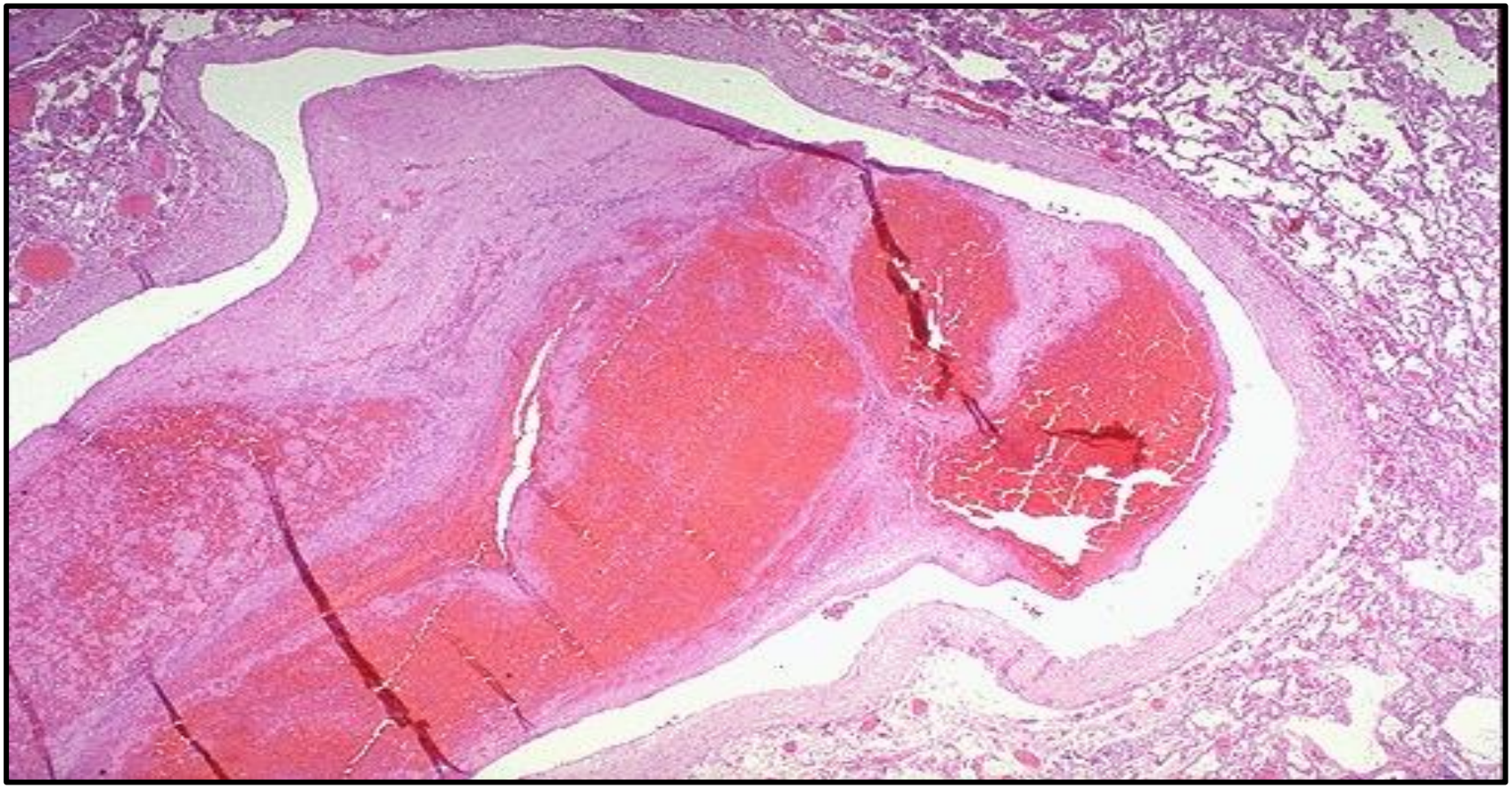
- refers to emboli traveling within the arterial circulation.

Organizing Thrombus



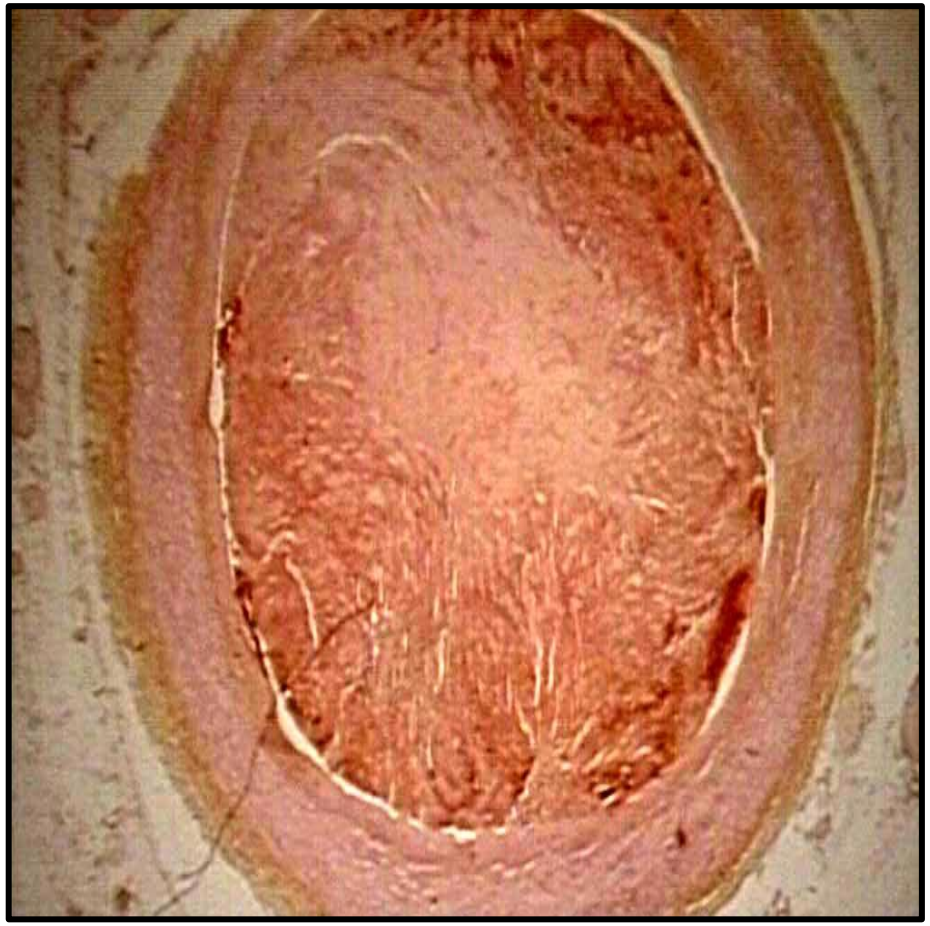
Organizing thrombus in a case of pulmonary embolism

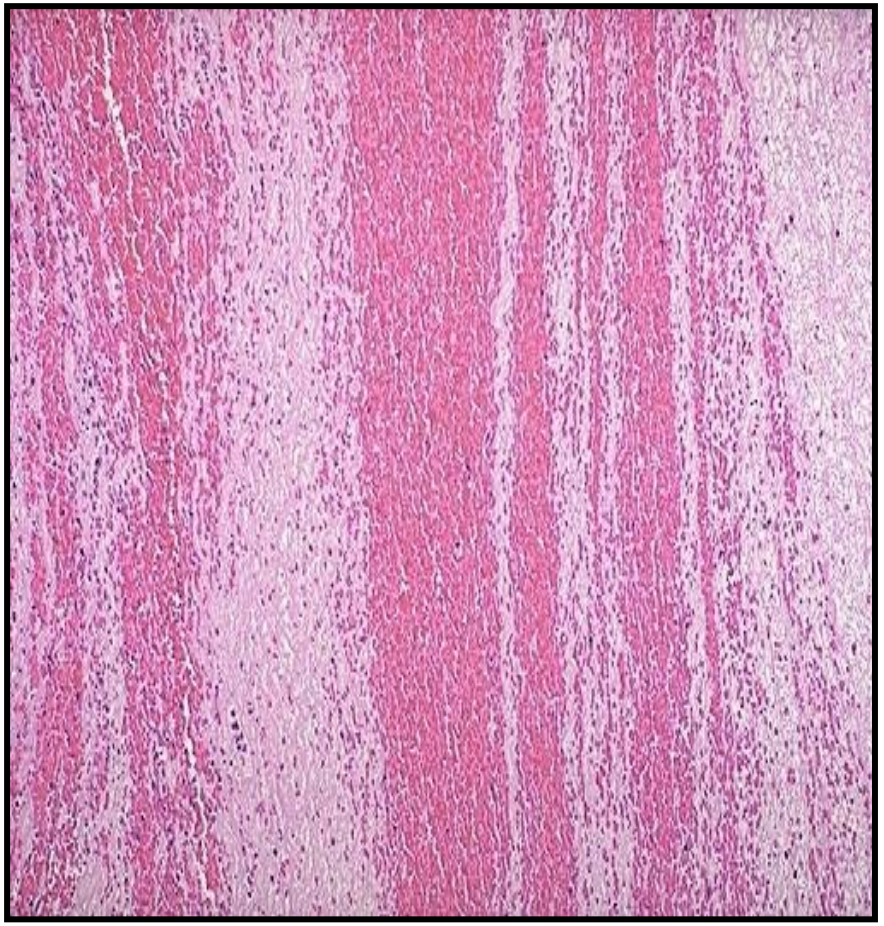
Organizing Thrombus with Lines of Zahn



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



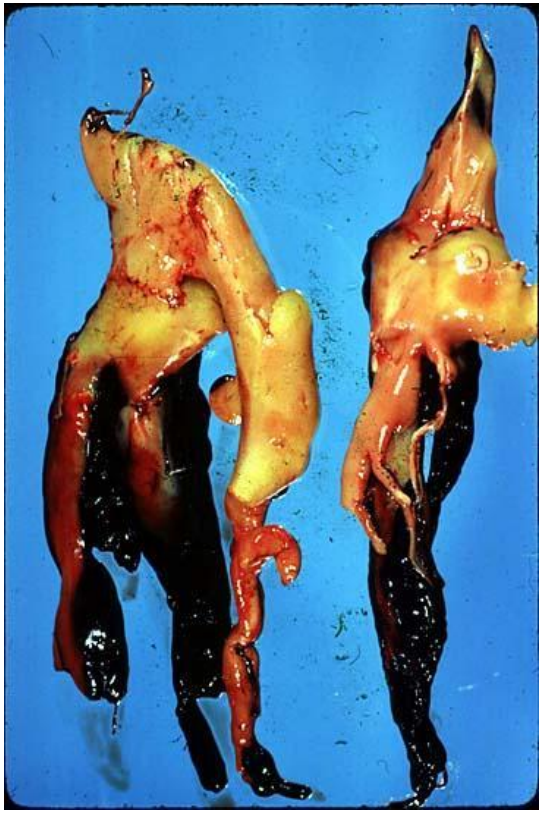


***Lines of Zahn**, gross and microscopic, is evidence to prove a clot is Pre-mortem which is different from the clots appearing like current jelly or chicken fat which are said to be Post-mortem. These lines represent layers of red cells, platelets, and fibrin*

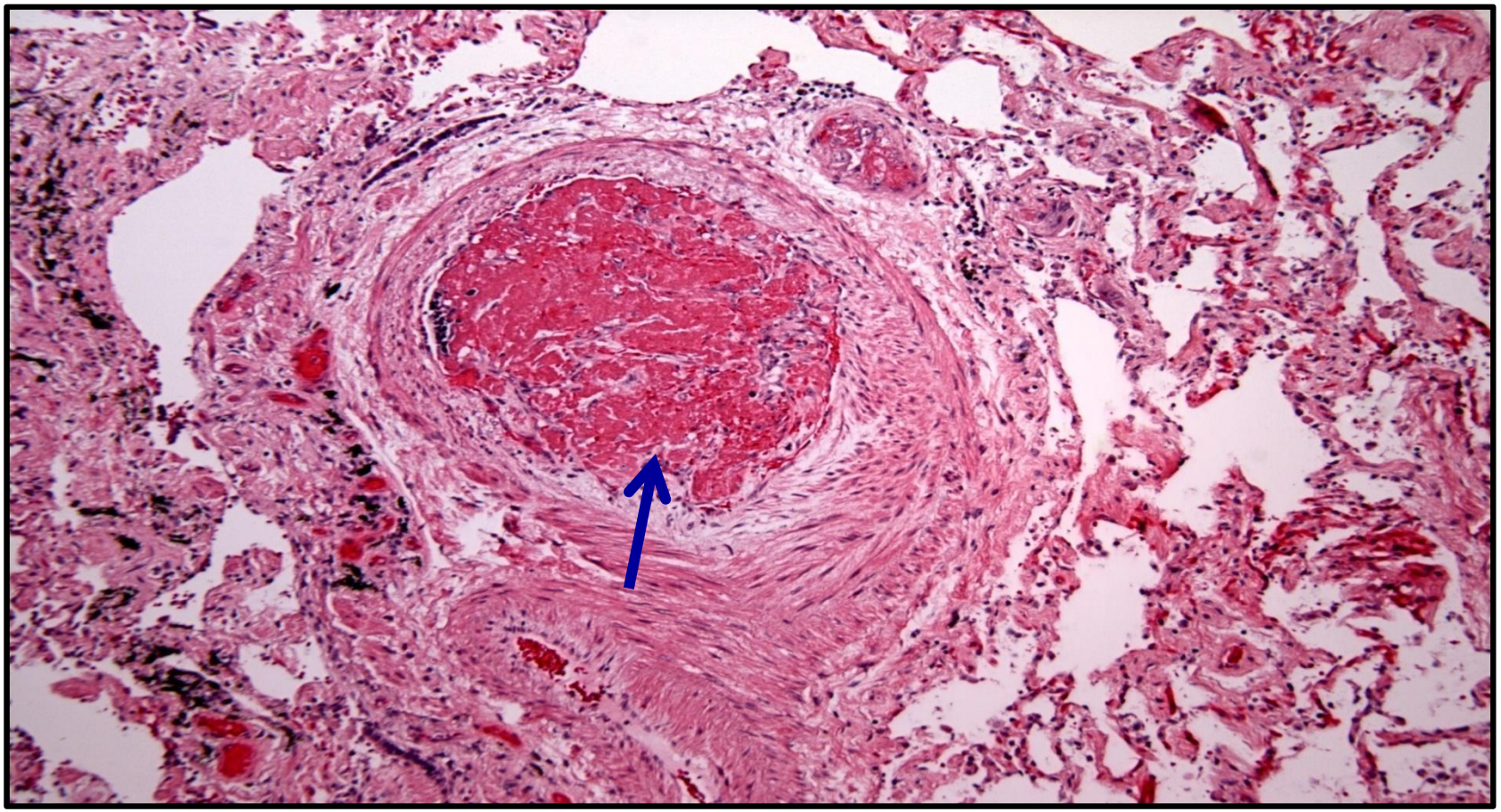
ADDITIONAL INFORMATION:

Postmortem clots: is a clot formed in the heart or in a blood vessel after death.

- At autopsy, postmortem clots may be confused for thrombi.
- Postmortem clots are gelatinous with a dark red dependent portion where red cells have settled by gravity at the bottom and an upper layer of yellow *chicken fat* like supernatant.
- Post mortem clots are not attached to the underlying blood vessel wall.
- Thrombi are firmer, have a point of attachment to blood vessel, and show vague lines of pale gray fibrin.



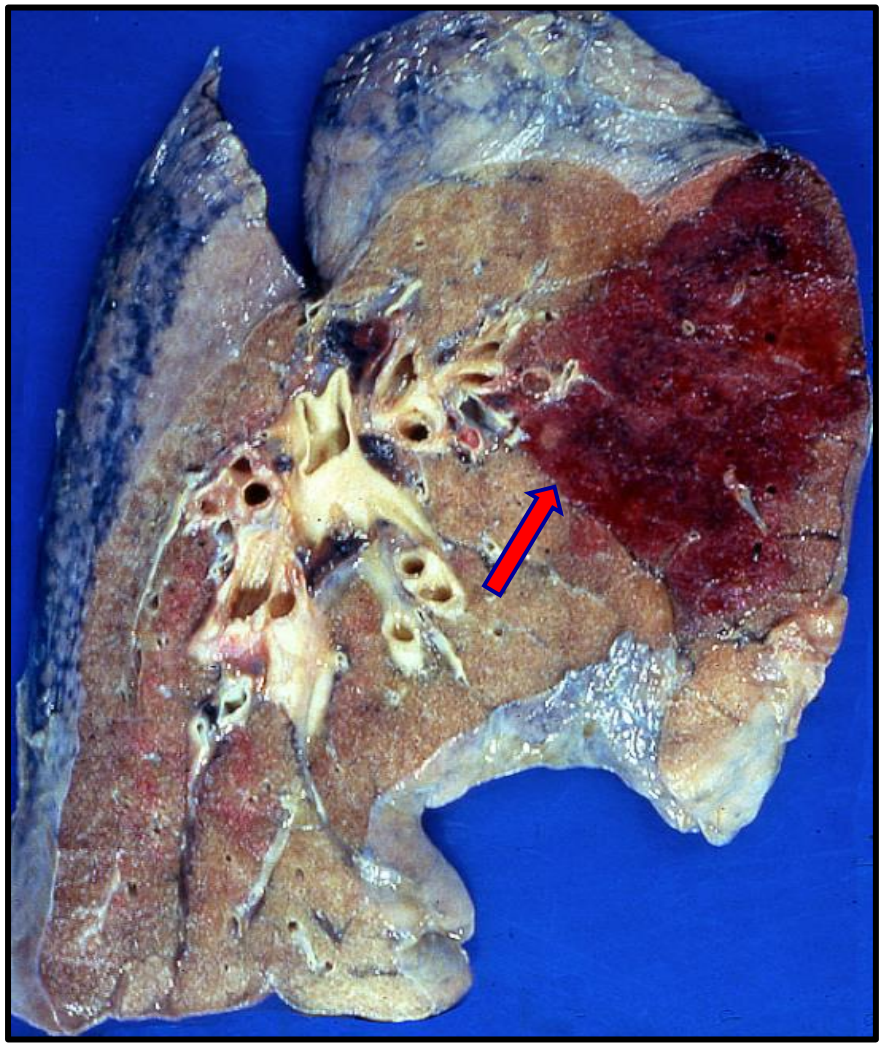
Thromboembolus in Pulmonary Artery

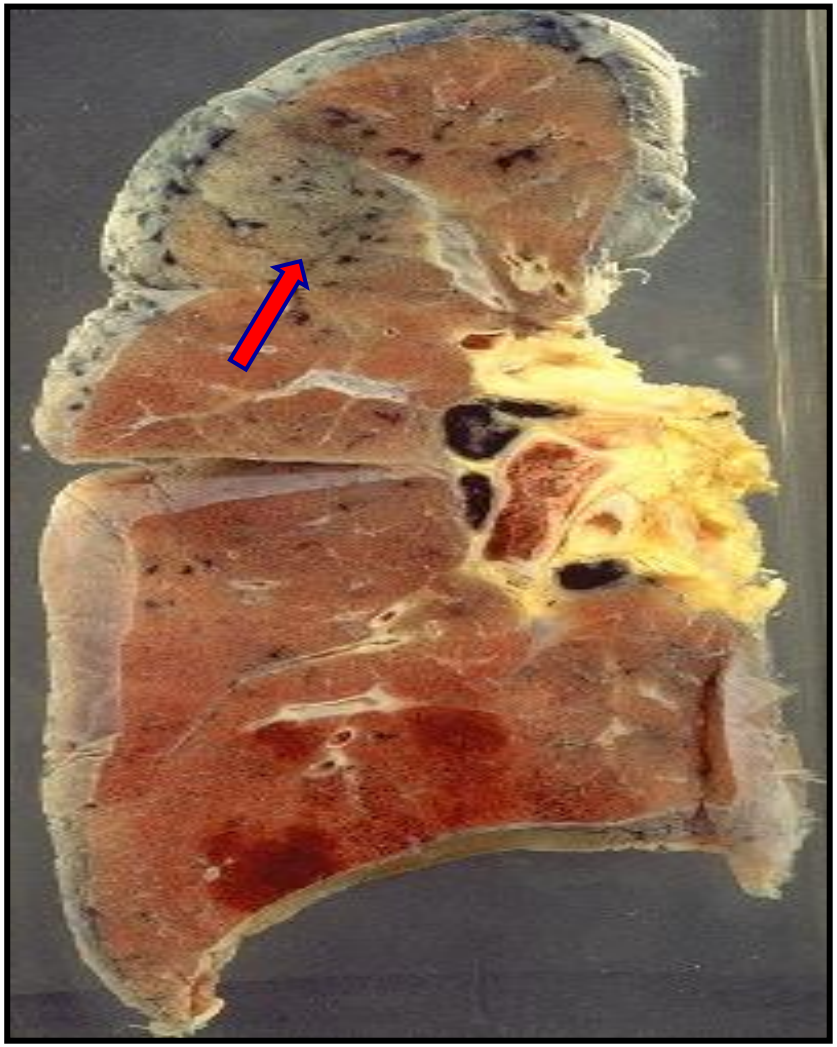


Pulmonary thromboembolus in a small pulmonary artery. The interdigitating areas of pale pink and red within the organizing embolus form the "lines of Zahn" (arrow) characteristic of a thrombus. These lines represent layers of red cells, platelets, and fibrin that are laid down in the vessel as the thrombus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

2- Pulmonary Embolus with Infarction

Pulmonary Embolus with Infarction





This specimen shows an area of dead lung tissue ("infarction") due to blockage of one of the major arteries to the lung by an embolus ("blood clot") originating from the deep veins of the leg.

Pulmonary Embolus with Infarction

A large pulmonary thromboembolus is seen in the pulmonary artery of the left lung. Such thromboemboli typically originate in the leg veins or pelvic veins of persons who are immobilized

3- Myocardial Infarction

ADDITIONAL INFORMATION: INFARCTION

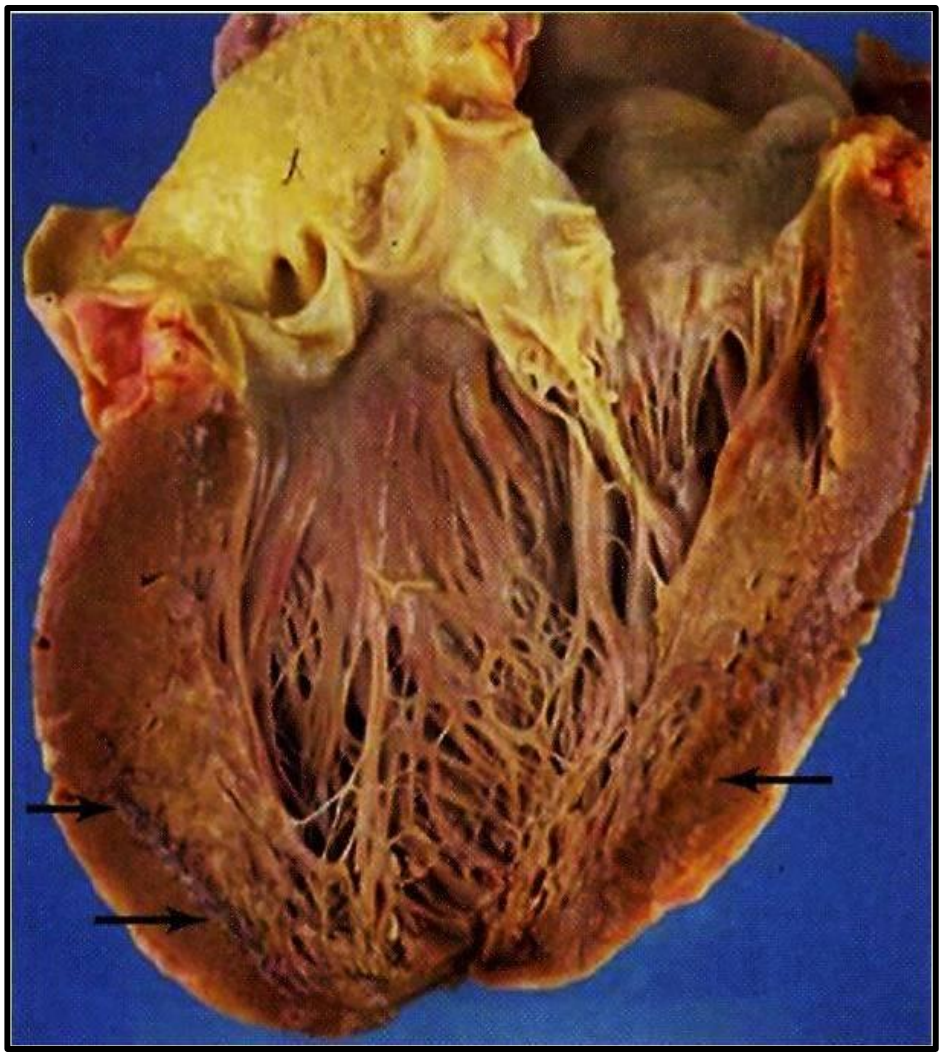
- *An infarct is an area of ischemic necrosis caused by occlusion of either the arterial supply or the venous drainage in a particular tissue e.g.* myocardial, cerebral, pulmonary and bowel infarction.
- Most infarcts result from thrombotic or embolic events, and almost all result from arterial occlusion.
- Infarcts are classified on the basis of their color as **red (hemorrhagic)** and **white (anemic).**

1)Red (hemorrhagic) infarcts occur

- with venous occlusions (such as in ovarian torsion)
- in loose tissues (such as lung),
- and in tissues with dual circulations (e.g., lung and small intestine), permitting flow of blood from the unobstructed vessel into the affected zone

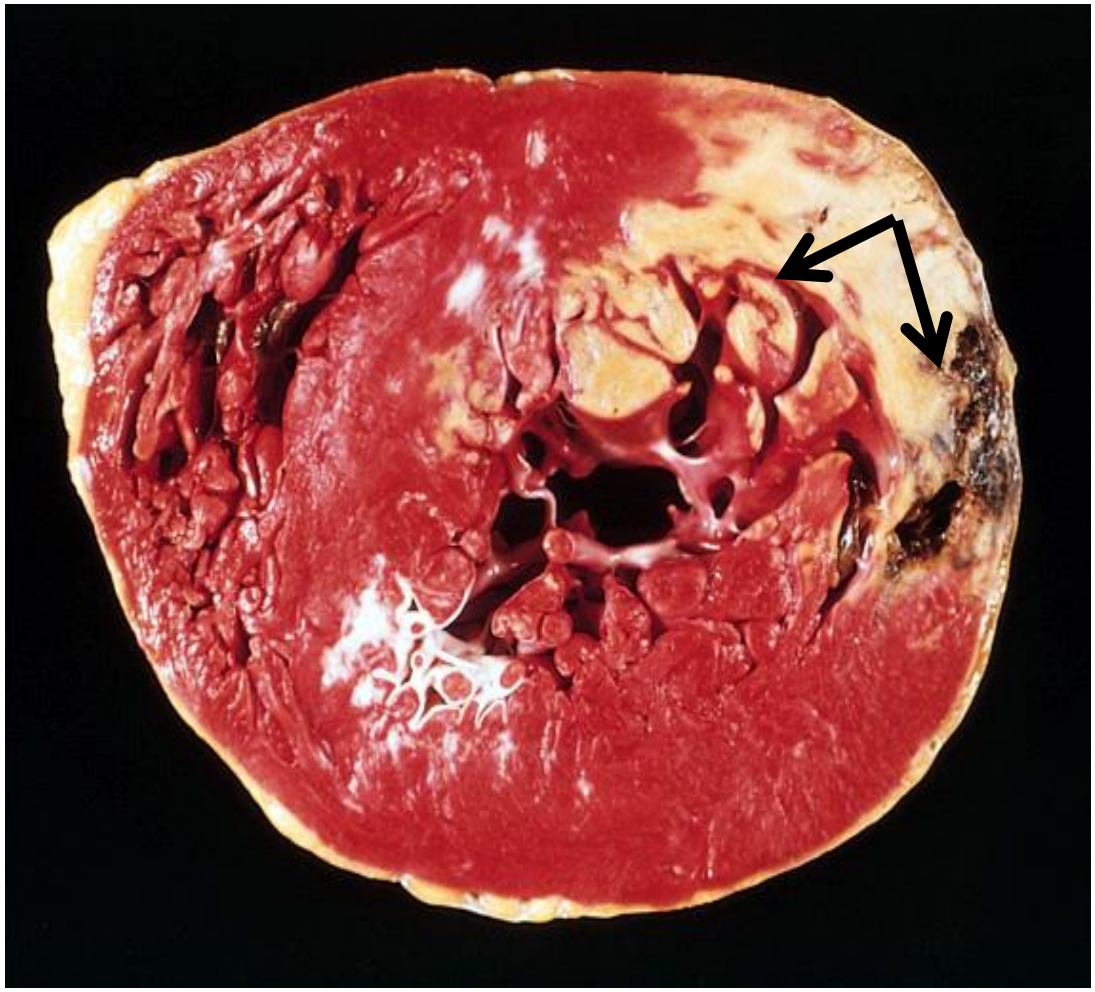
2) White (anemic) infarcts occur with arterial occlusions in solid organs with end-arterial circulation such as heart, spleen, liver kidney, etc.

Myocardial Infarction



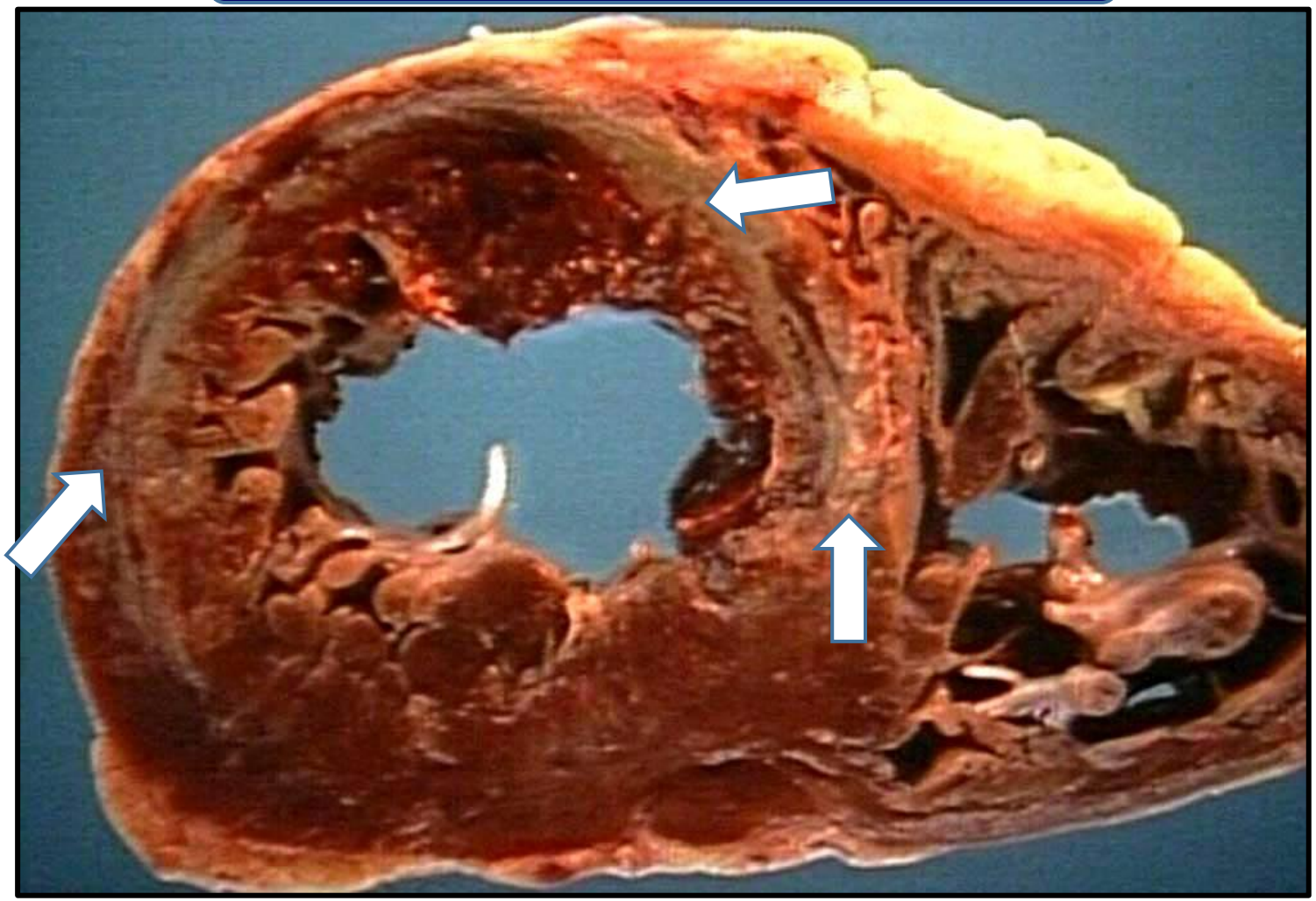
Complications that might occur : arrhythmias , ventricular aneurysm, rupture of myocardium, cardiac tamponade and others .

Myocardial Infarction



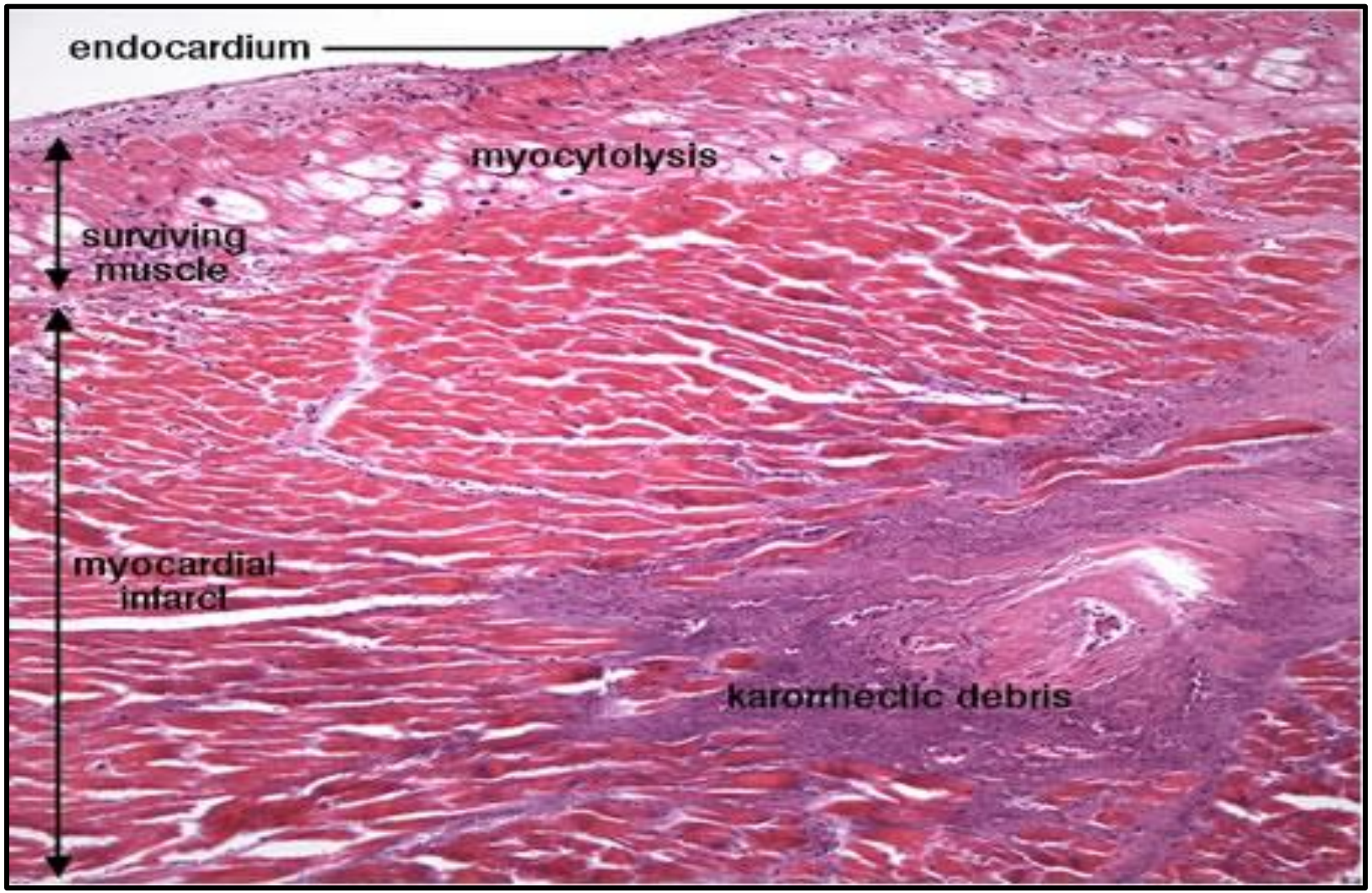
Cross section of the left and right ventricles shows a pale and irregular focal fibrosis in the left ventricular wall with increased thickness .

Myocardial Infarction



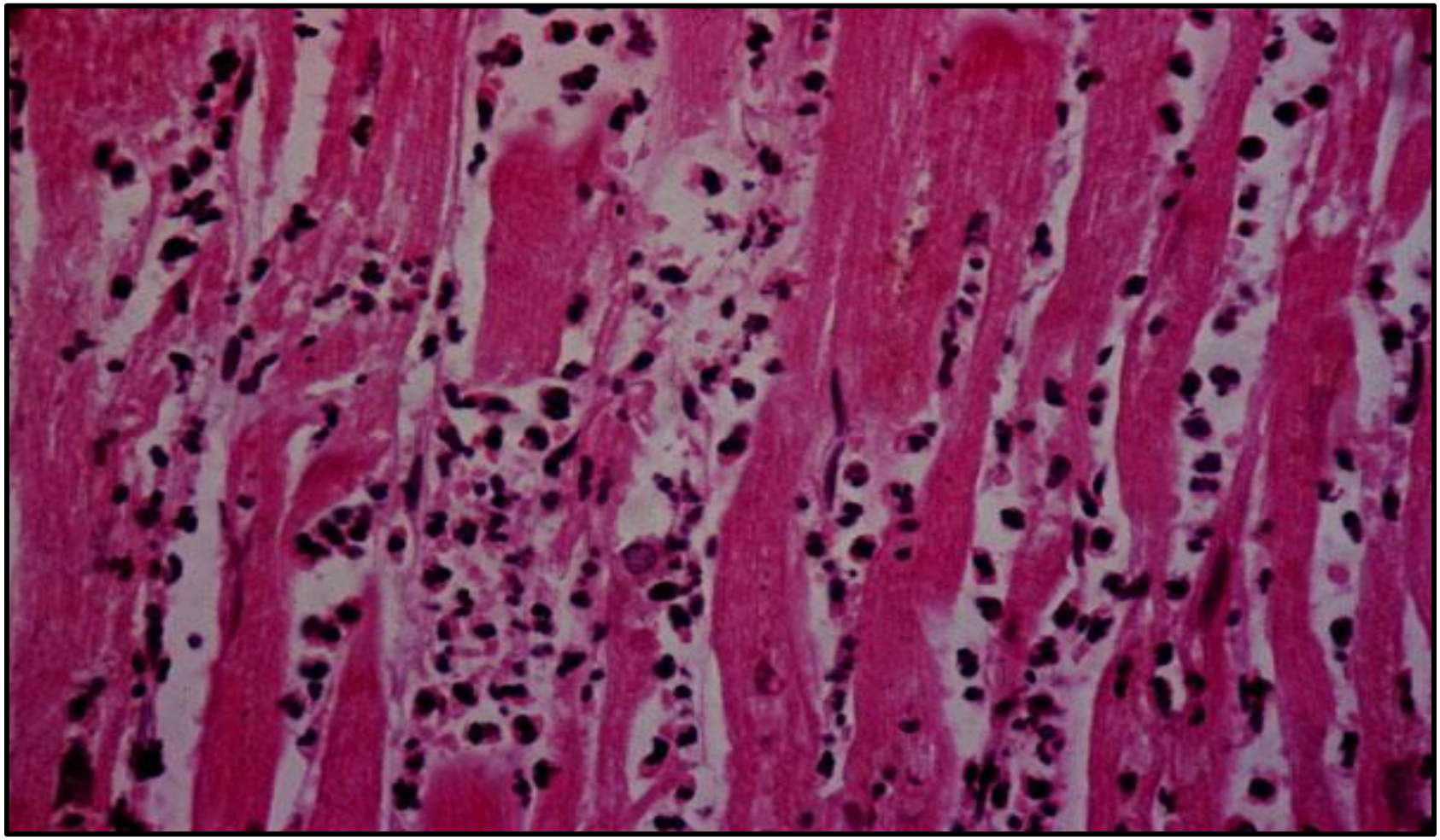
Cross section of the left and right ventricles shows a pale and irregular focal fibrosis in the left ventricular wall with increased thickness .

Myocardial Infarction



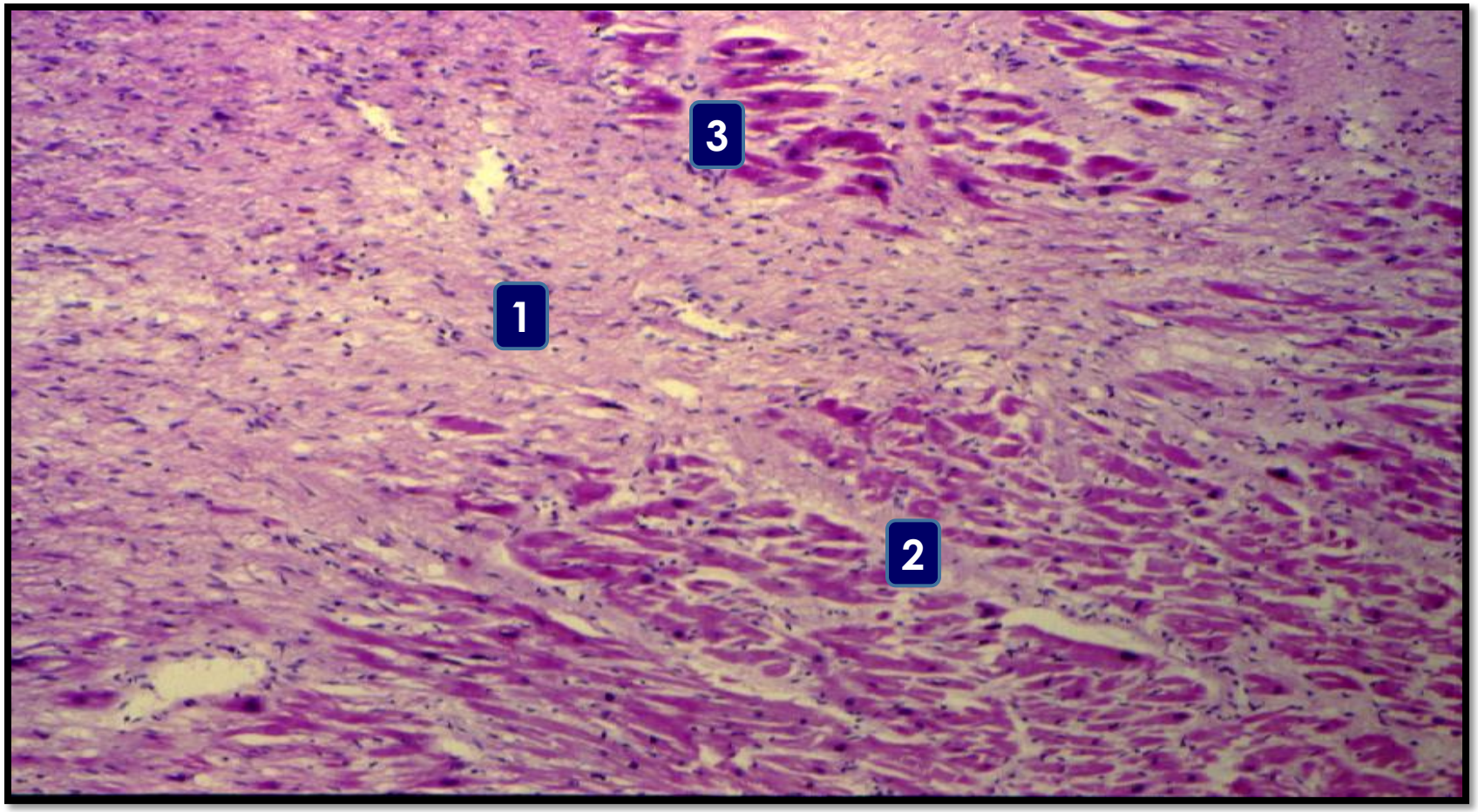
Transmural myocardial infarct at 2 weeks

Myocardial Infarction



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

Myocardial Infarction



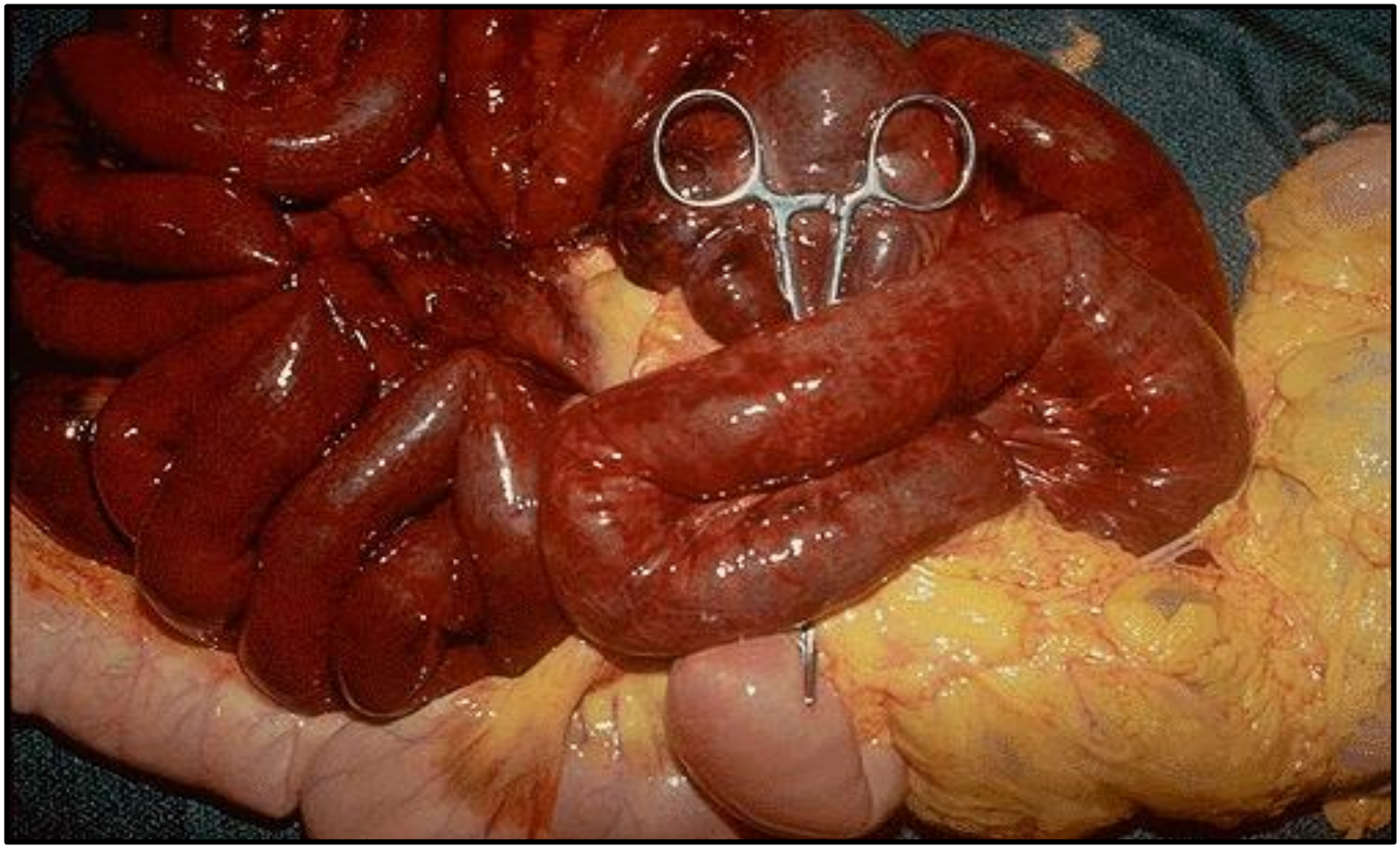
1- Patchy coagulative necrosis of myocardial fibers. The dead muscle fibers are structureless and hyaline with loss of nuclei & striations.

2- Chronic ischemic fibrous scar replacing dead myocardial fibers .

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

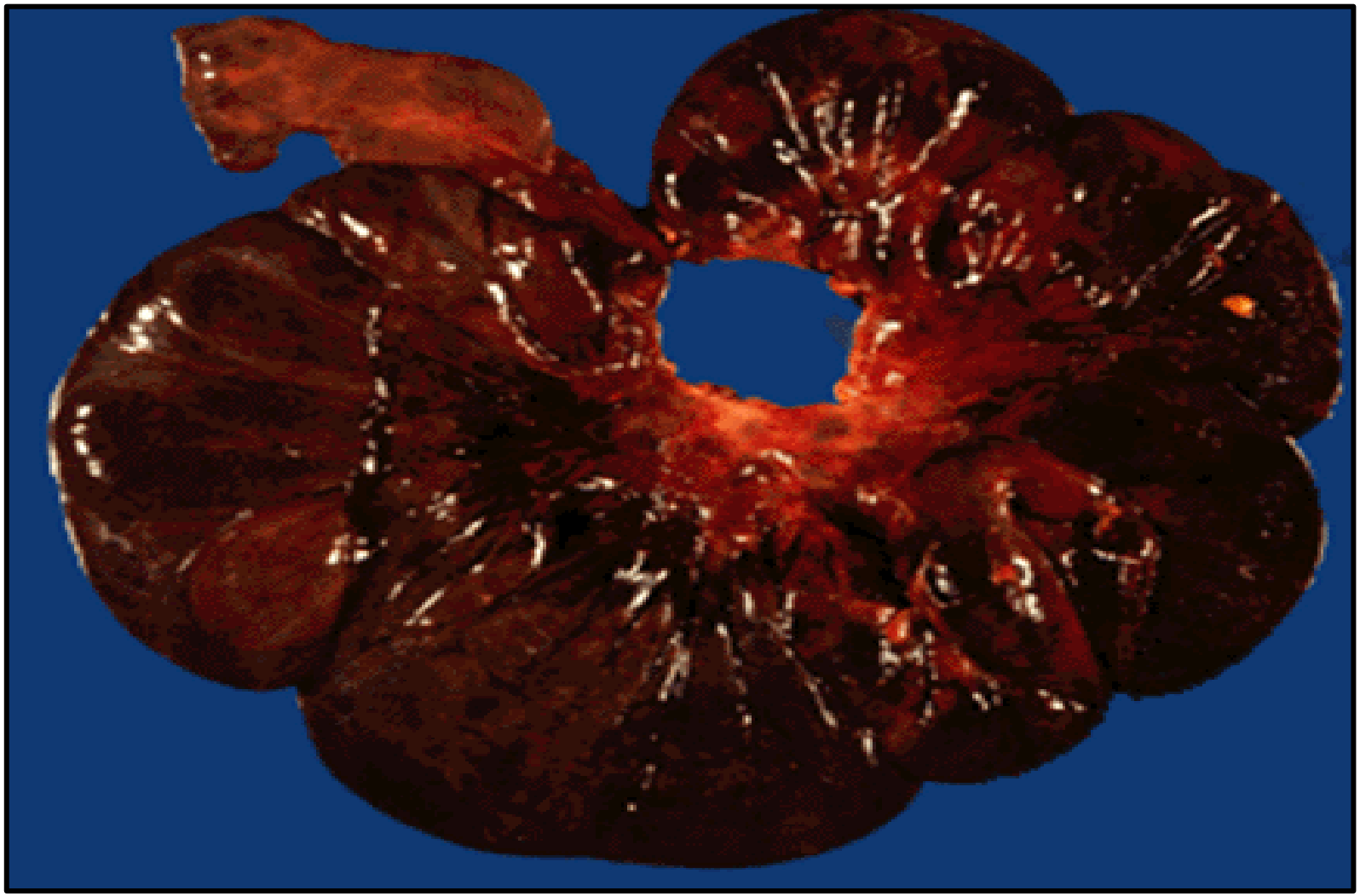
4- Infarction of the Small Intestine

Infarction of the Small Intestine



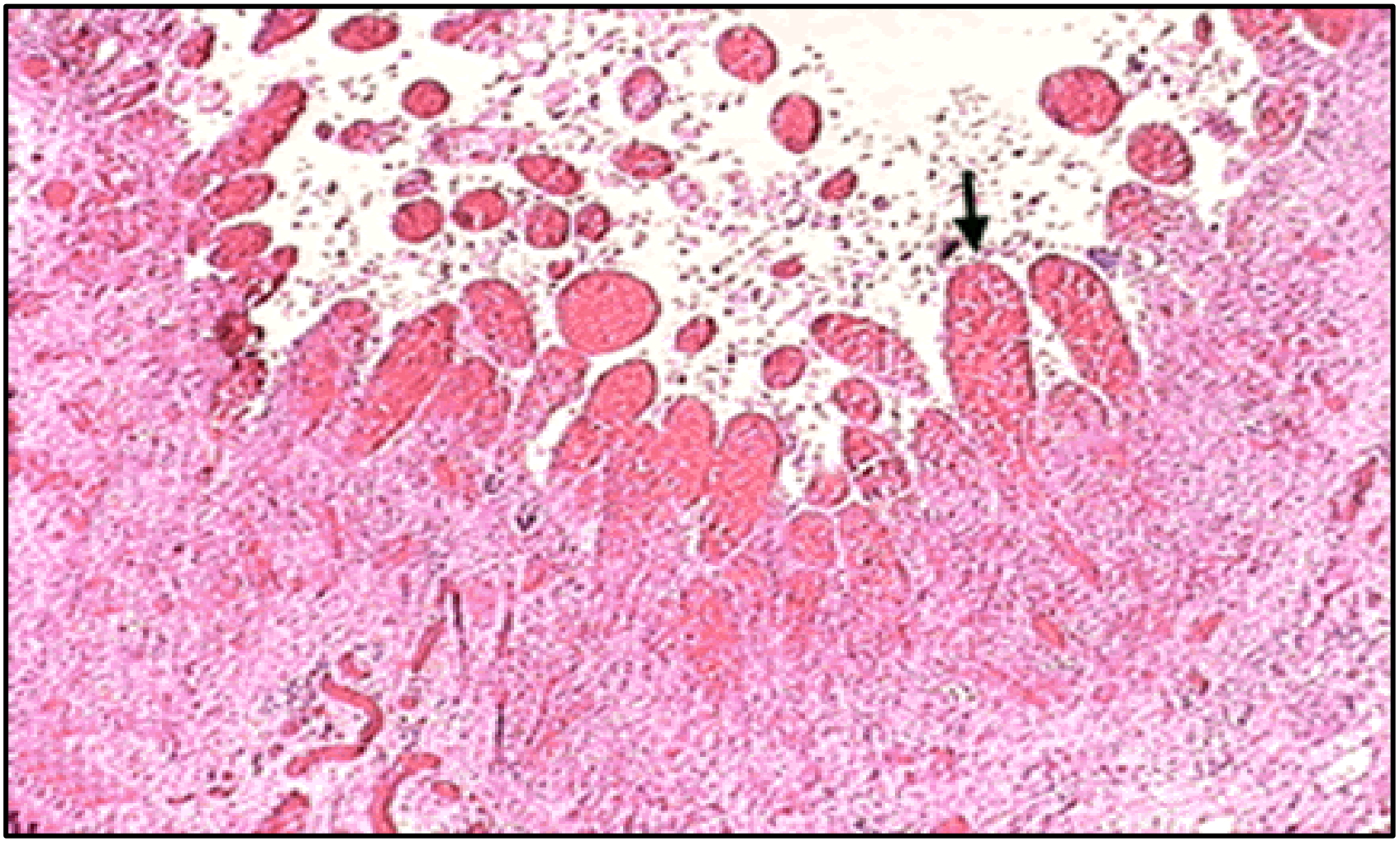
The dark red infarcted small intestine contrasts with the light pink viable bowel. The forceps extend through an internal hernia in which a loop of bowel and mesentery has been caught. This is one complication of adhesions from previous surgery. The trapped bowel has lost its blood supply

Infarction of the Small Intestine



Diffuse violacious red appearance is characteristic of transmural hemorrhagic intestinal infarction

Infarction of the Small Intestine



Intestinal infarction typically begins in the villi, which are end vasculature without anastomoses. There is complete loss of the mucosal epithelium. Broad areas of hemorrhage with moderate inflammatory infiltrate is present

THE END