

Cardiovascular Practical Block

**Done by,
Pahtology Team**

1-Atheroma of aorta



This is severe atherosclerosis of the aorta shows:

- 1. atheromatous plaques**
- 2. ulceration**
- 3. formation of overlying mural thrombus. “thrombus on a cavity”**

Q: what is the organ?

A: Aorta

Q: what is the pathology?

A: Severe Atherosclerosis

Q: what are the risk factors related to this pathology?

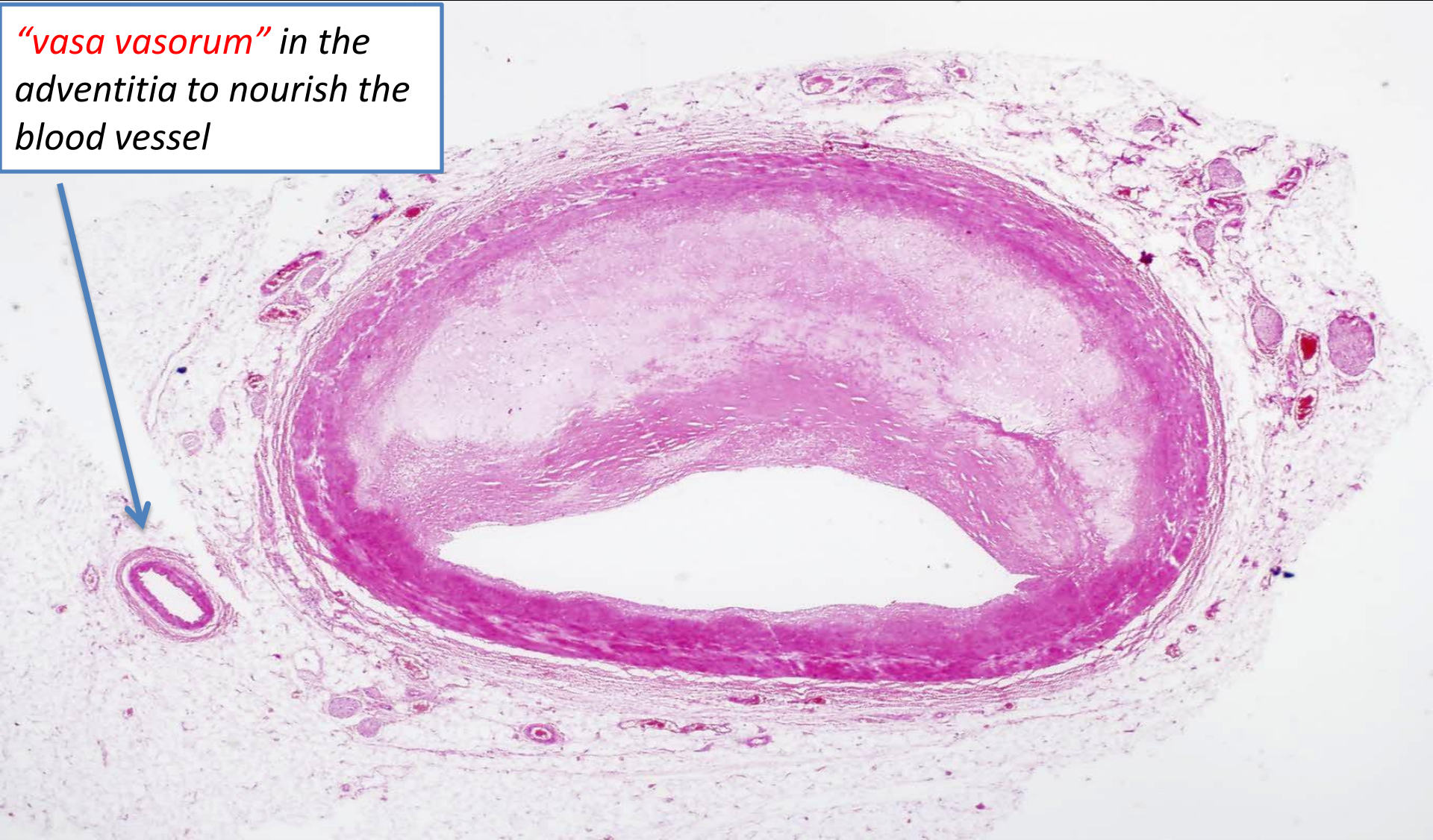
A: 1) increasing age 2) Hyperlipidemia 3) Hypertension 4) Obesity



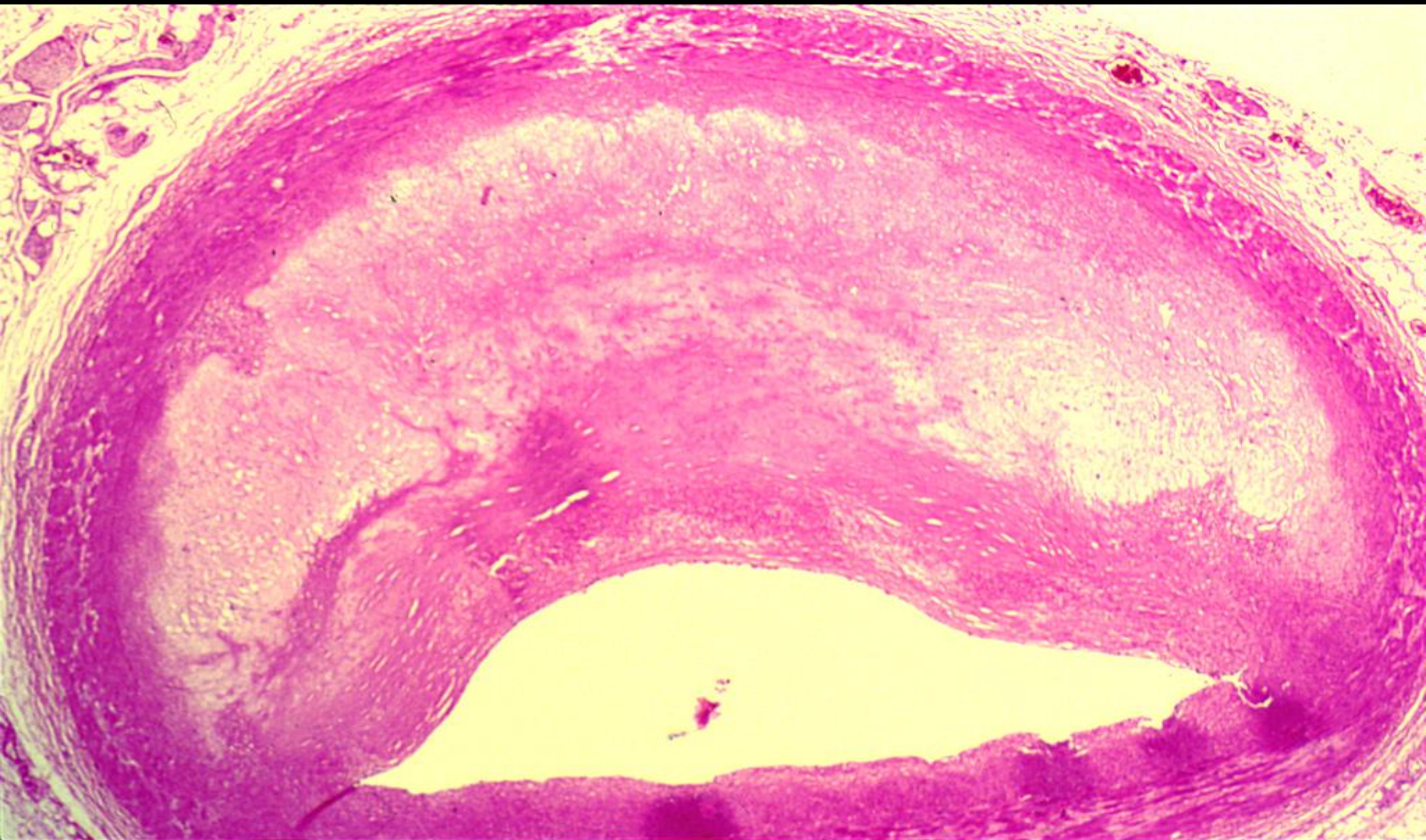
2-Coronary atherosclerosis

CORONARY ARTERY ATHEROSCLEROSIS

***“vasa vasorum”** in the
adventitia to nourish the
blood vessel*







CORONARY ATHEROSCLEROSIS



Coronary atherosclerosis:

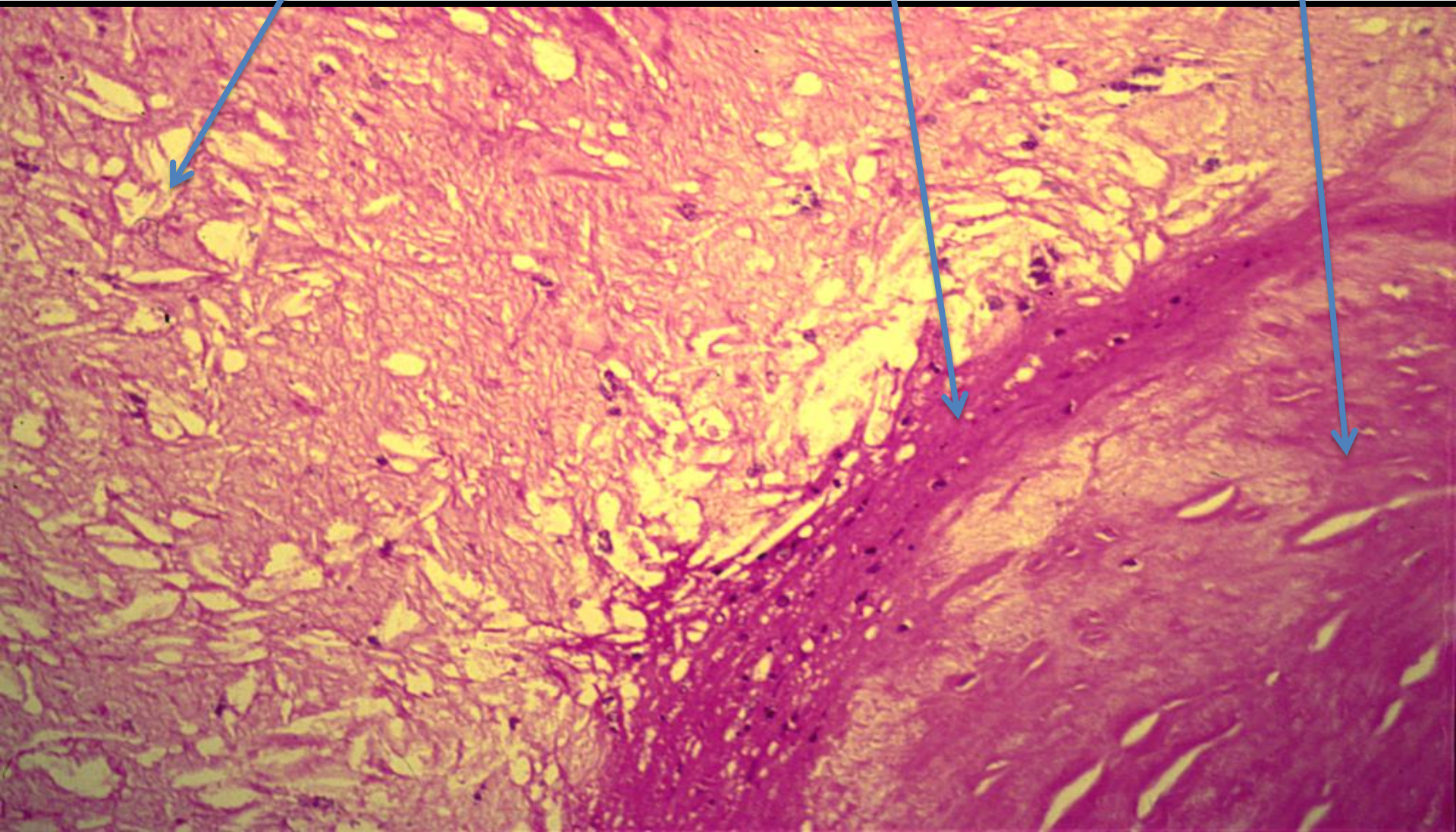
Cross section of a coronary artery shows:

-  **Partial occlusion of the lumen by an atheromatous plaque.**
-  **The plaque consists of dissolved, **cholesterol clefts, hyaline fibrous tissue and some blood capillaries.****
-  **The internal elastic lamina is thin and fragmented.**
-  **Pressure atrophy of the media opposite atheromatous plaque.**

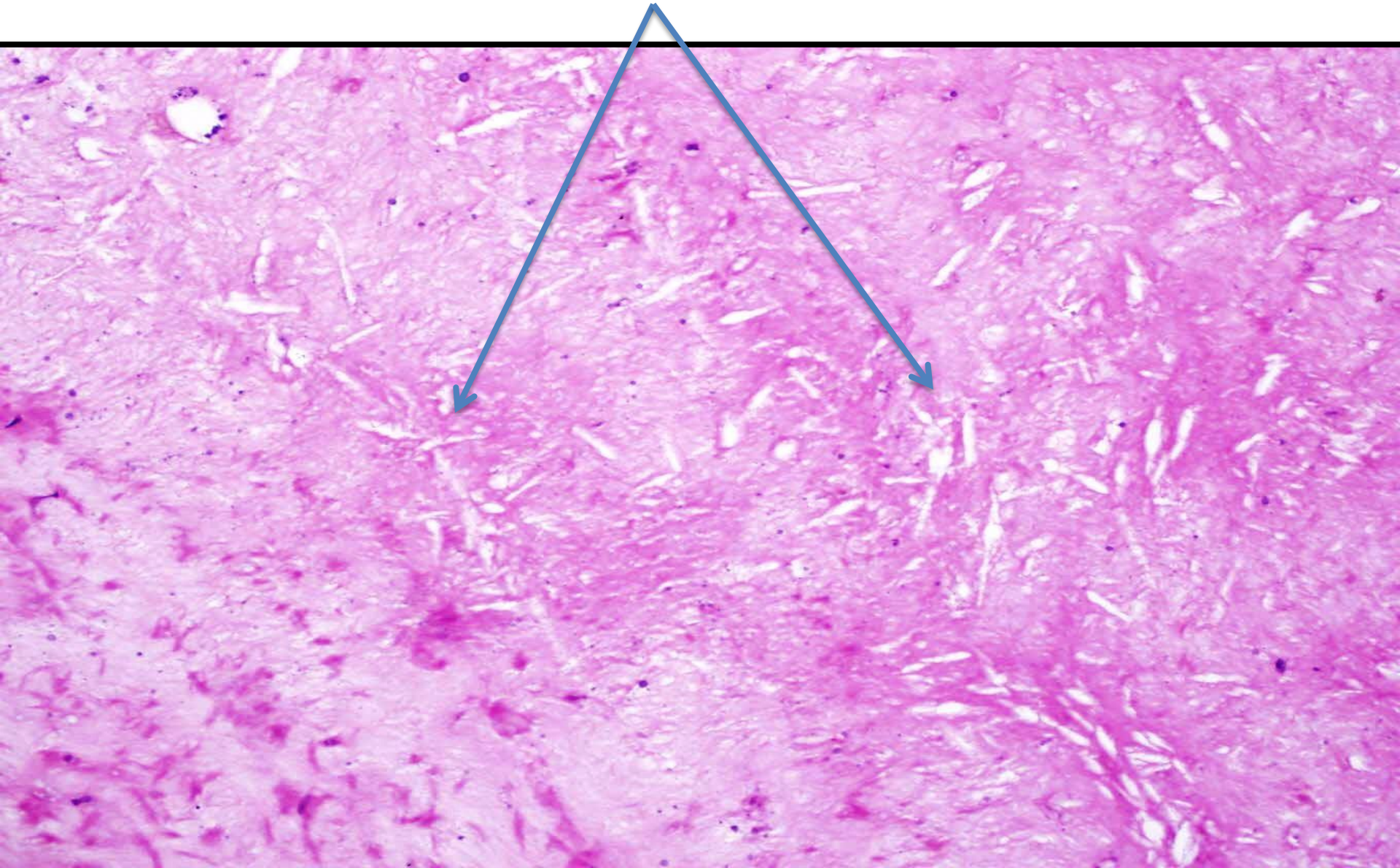
cholesterol clefts

**Internal elastic
lamina**

Intimal thickening

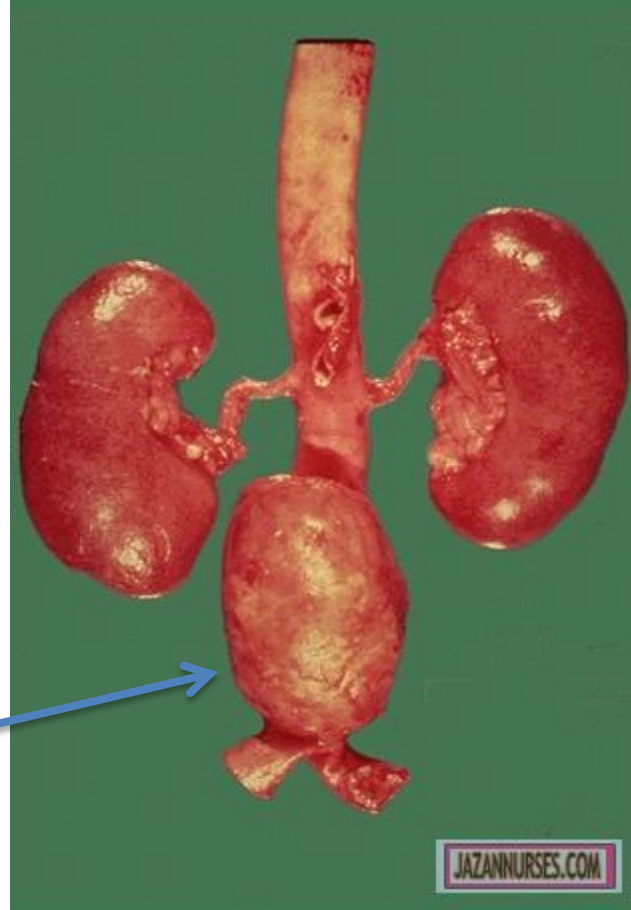


ATHEROMATOUS PLAQUE WITH CHOLESTEROL CLEFTS



3-Aneurysm of abdominal aorta

The Bulge



Here is an example of an **atherosclerotic fusiform aneurysm of the aorta** in which a large "**bulge**" appears just above the aortic bifurcation.

Q: what is the organ?

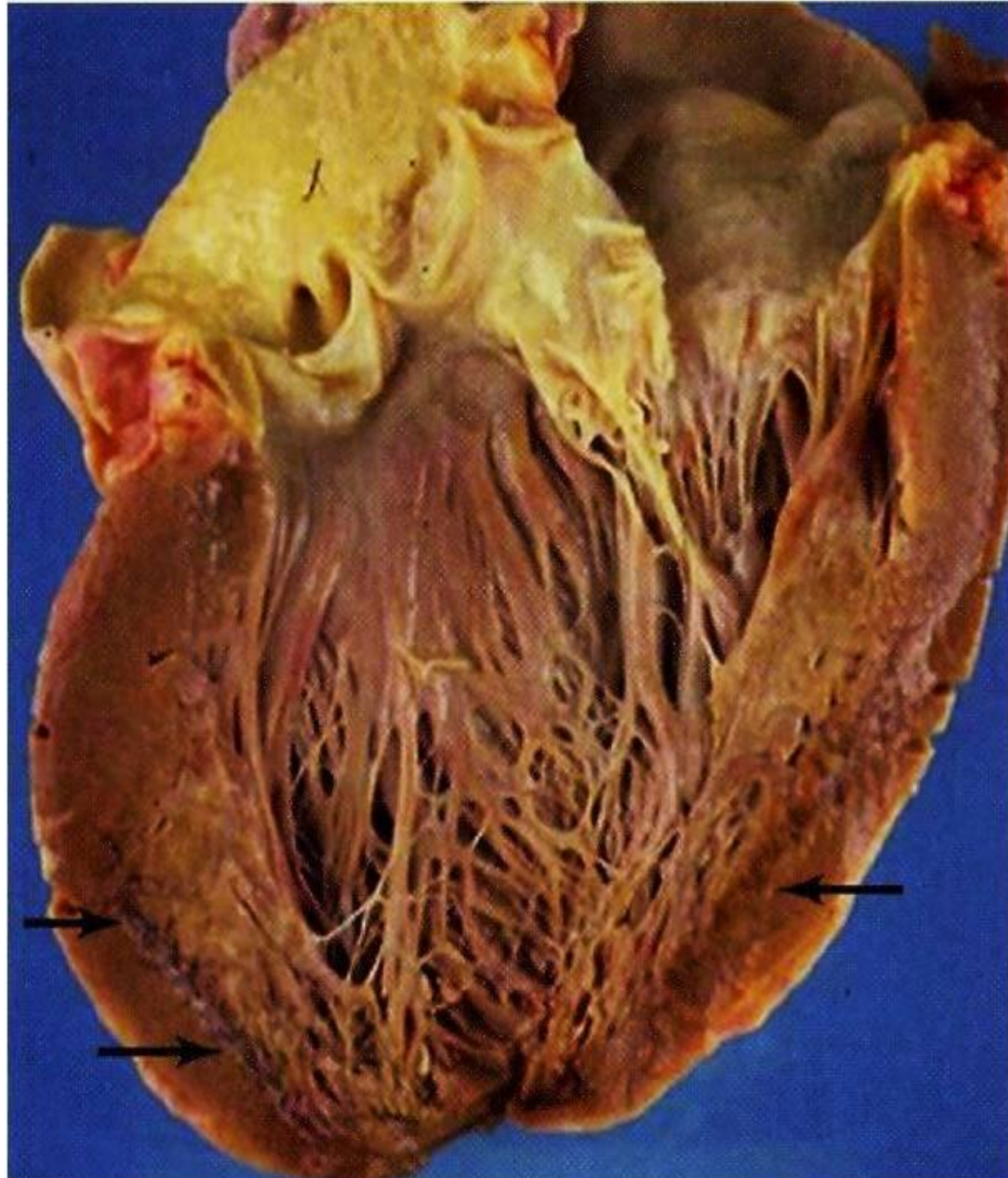
A: Aorta

Q: what is the most important cause of this aneurysm?

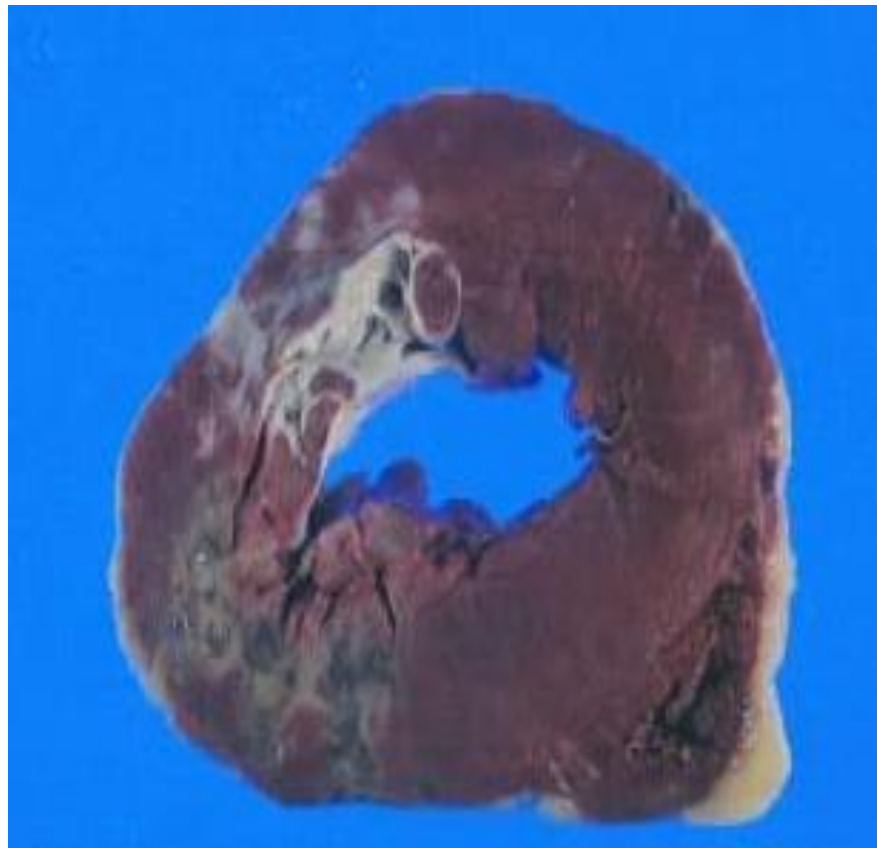
A: Atherosclerosis



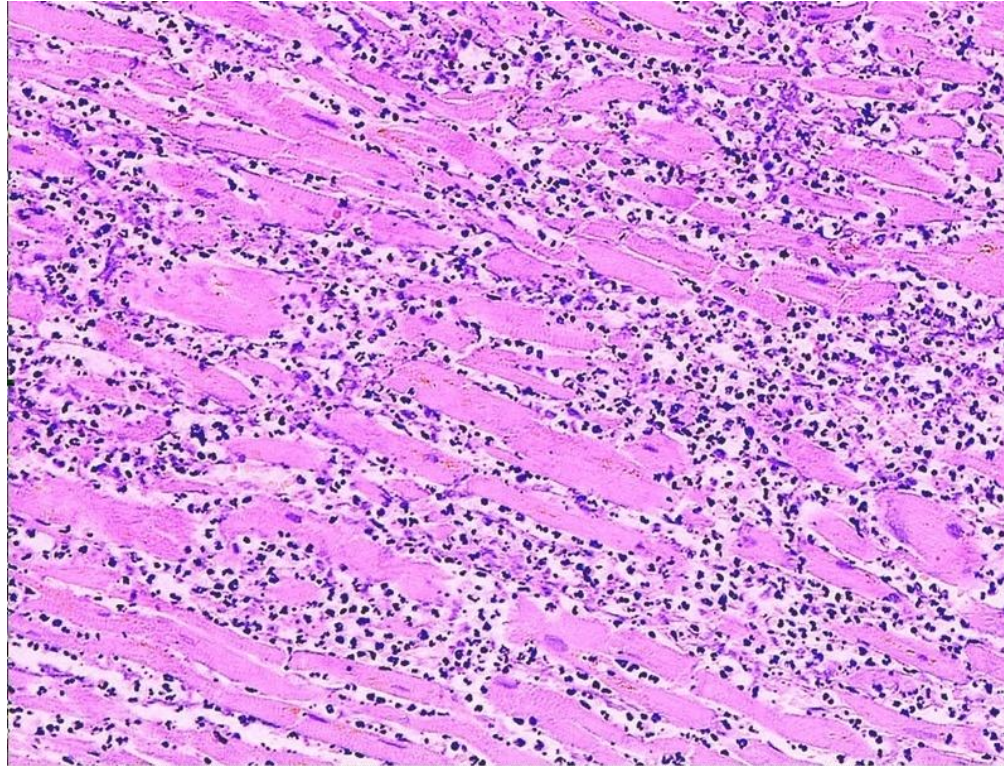
4-Myocardial infarction



Myocardial infarction



Myocardial infarction (recent stage)

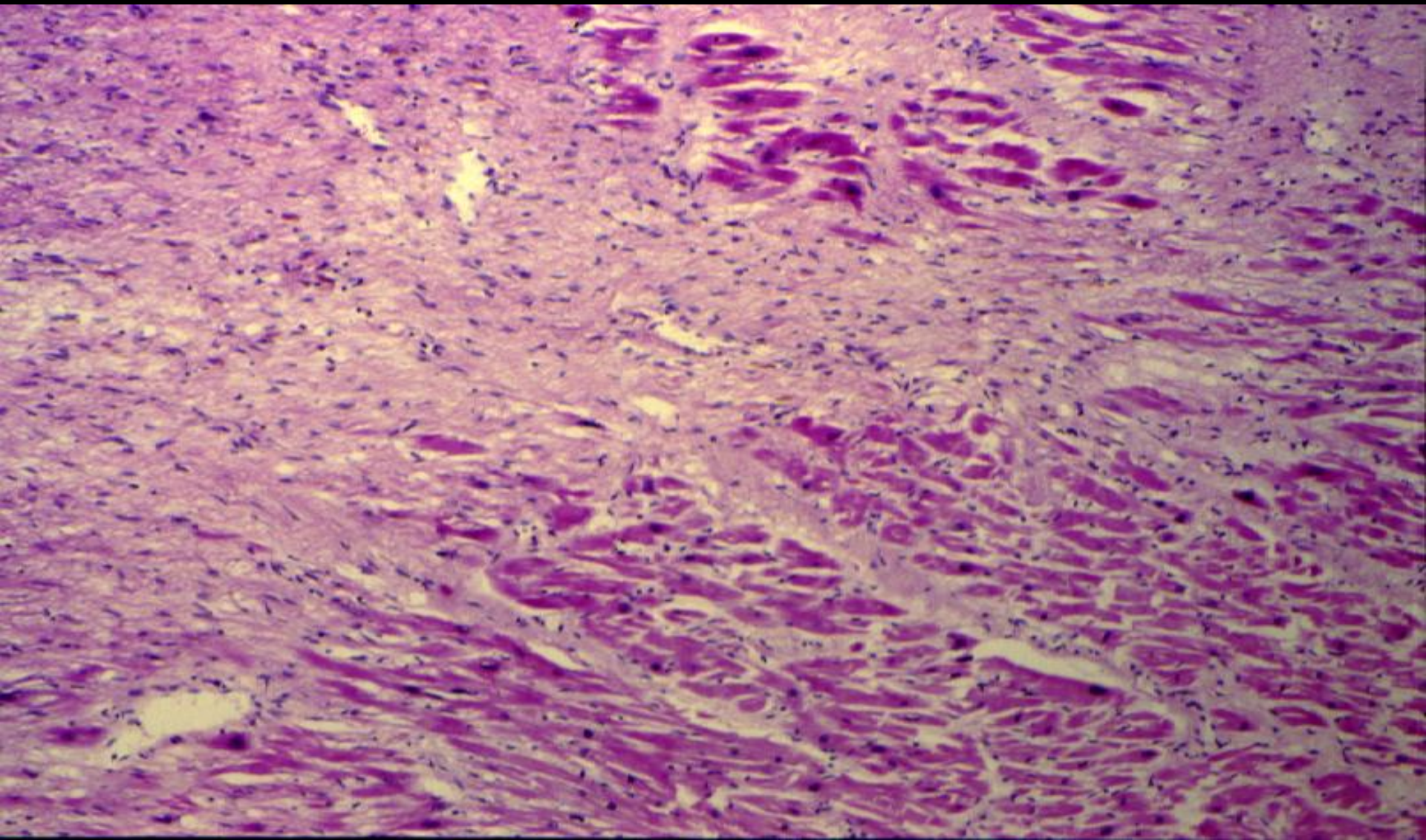


Infiltration of neutrophils into myocardial muscle fibers

The section shows:

Myocardial infarction with **ischemic coagulative necrosis areas of cardiac muscles** which supplied by occluded coronary artery.

MYOCARDIAL INFARCTION (LATE STAGE)



MYOCARDIAL INFARCTION (LATE STAGE)




The description:

In the late stage of myocardial infarction, there is granulation tissue which is composed of:

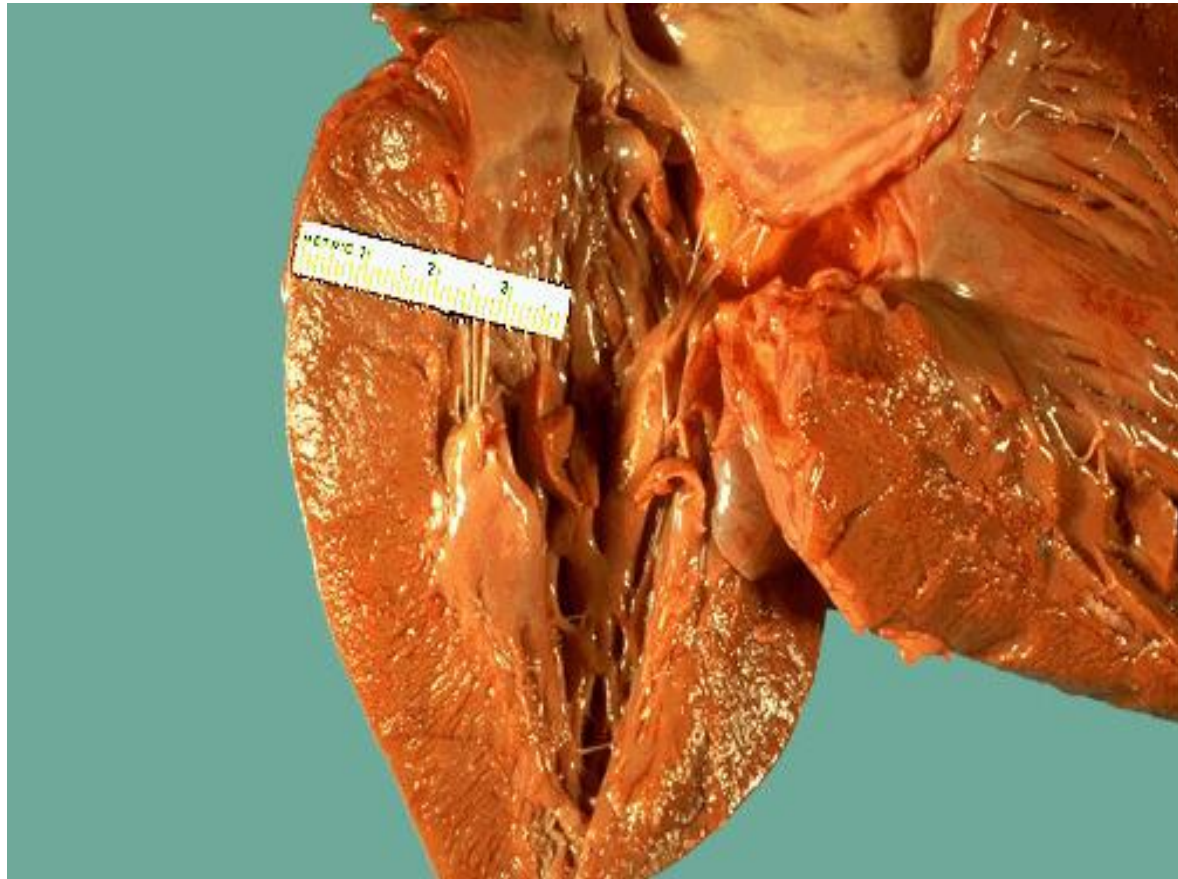
- 1. Inflammatory cells**
- 2. Fibroblasts**
- 3. Neovascularization**

Myocardial infarction:

Section of myocardial shows:

-  **Patchy coagulative necrosis of myocardial fibres.**
The dead muscle fibres are structureless and hyaline.
-  **The necrotic muscle fibres are pale with loss of nuclei and striations. Infiltration of neutrophils in recent stage is seen .**
-  **Later granulation tissue formation and fibrosis.**

5-Left ventricular hypertrophy



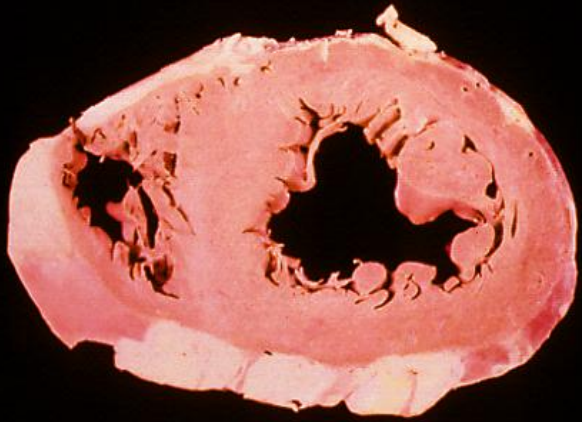
The normal size of left ventricle is 1.5 cm

Here is The left ventricle which is very thick (over 2 cm). "hypertrophy"

The most important cause is hypertension which create a greater pressure load on the heart to induce the hypertrophy then heart failure.



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



Heart, normal

Q: what is the organ?

A: Heart

Q: what is the pathology?

**A: left ventricular
hypertrophy**

**Q: what is the most
important cause of this
pathology?**

A: Hypertension



6-Vegetations of rheumatic fever on mitral and aortic valves



The description:

The small verrucous vegetations seen along the closure line of this mitral valve are associated with acute rheumatic fever “which caused indirectly by *Group A Beta hemolytic streptococcus* . These vegetations form along the line of valve closure over areas of endocardial inflammation. Such verrucae are too small to cause serious cardiac problems.

Q: what is the organ?

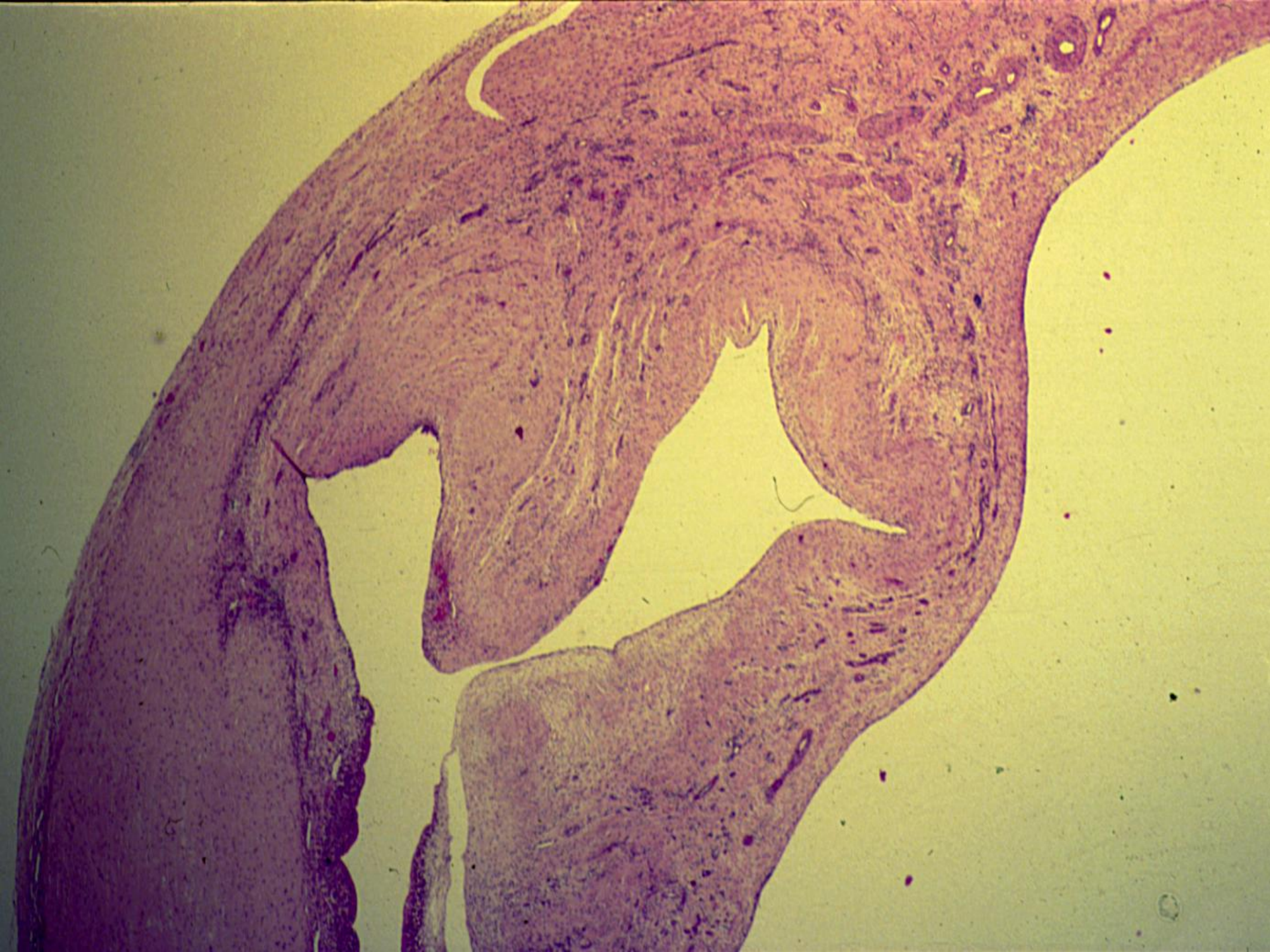
A: Mitral Valve

Q: what is the pathology?

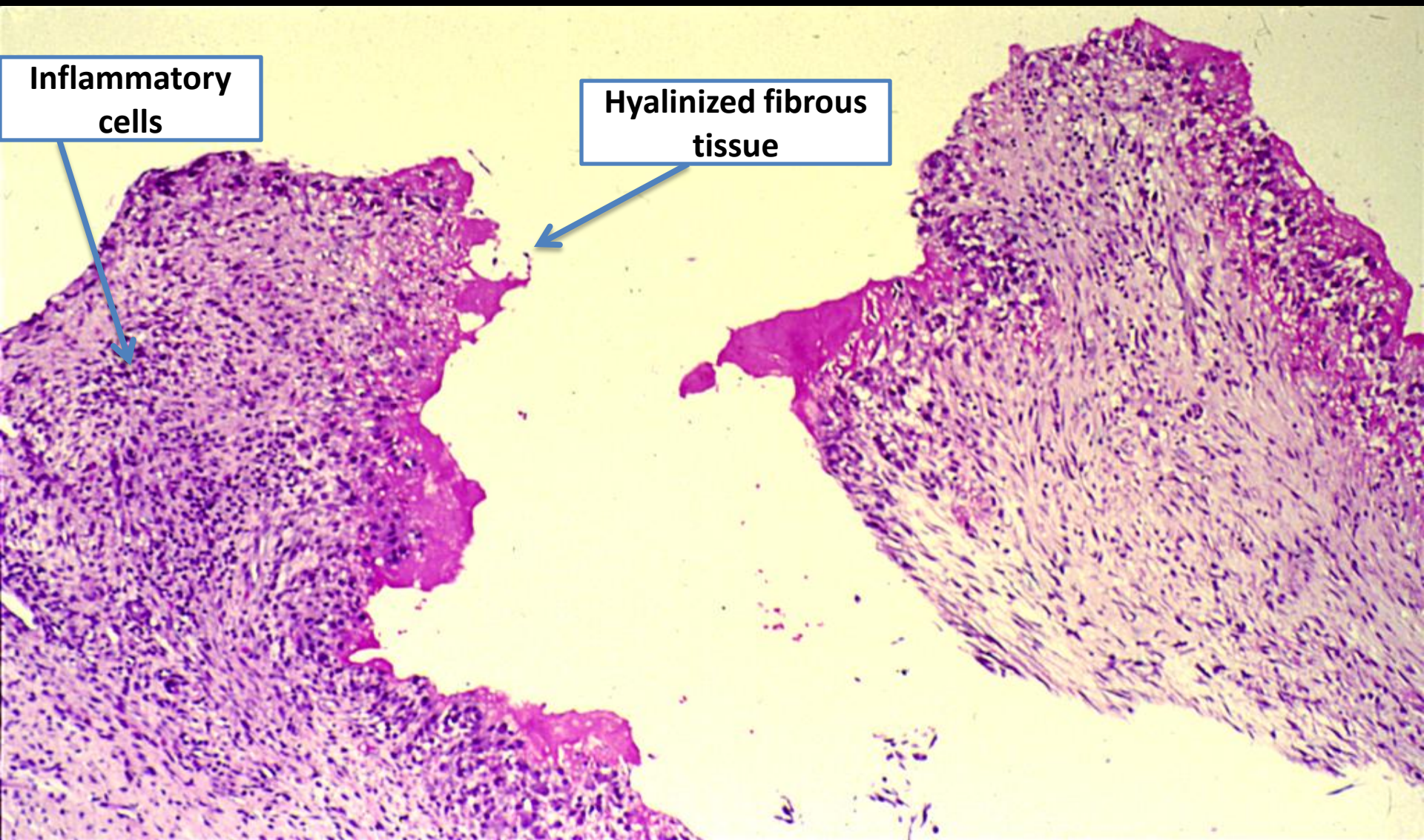
A: Rheumatic vegetations

Vegetations on the Aortic valve

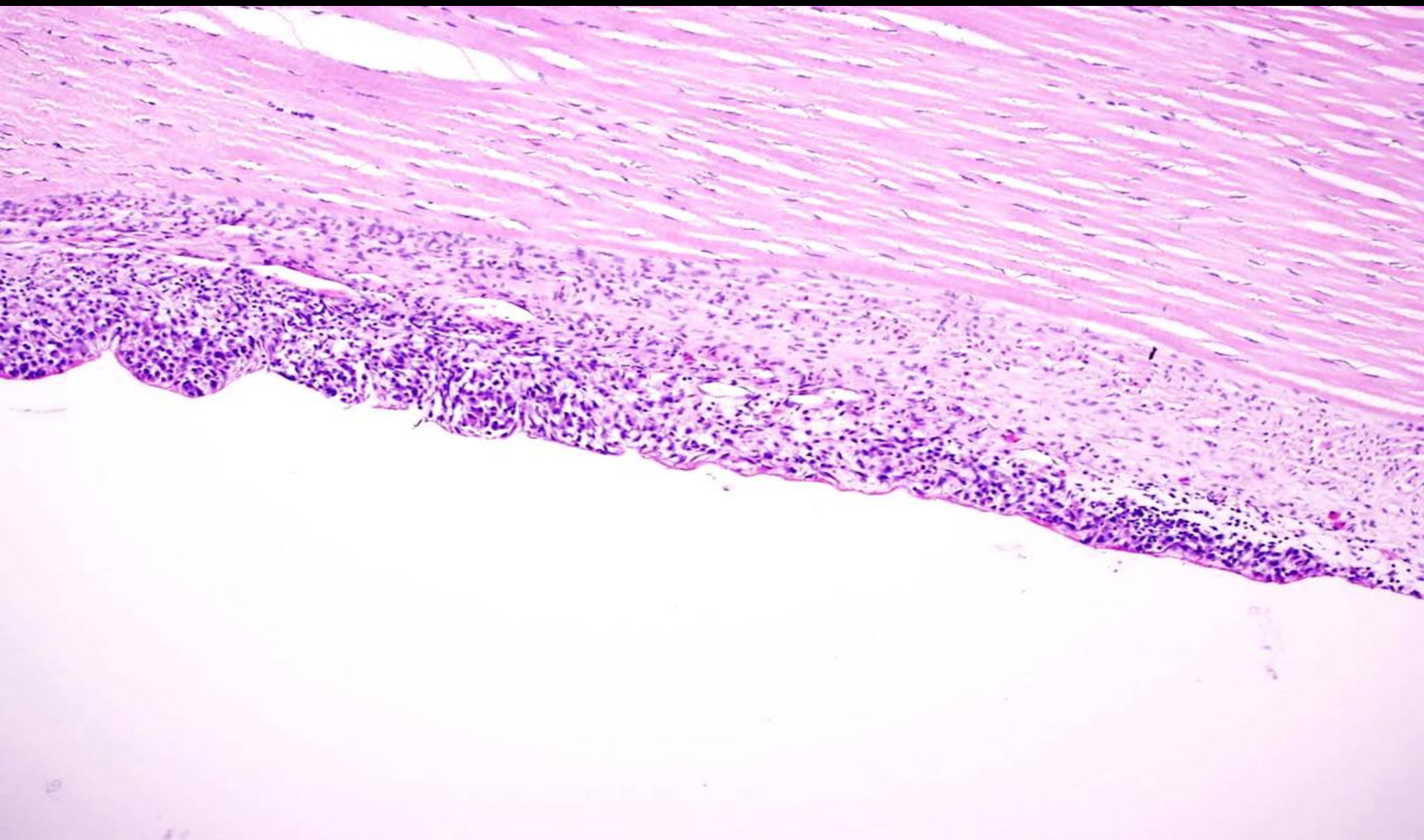




RHEUMATIC VALVULITIS (HEART)



RHEUMATIC VALVULITIS (HEART)

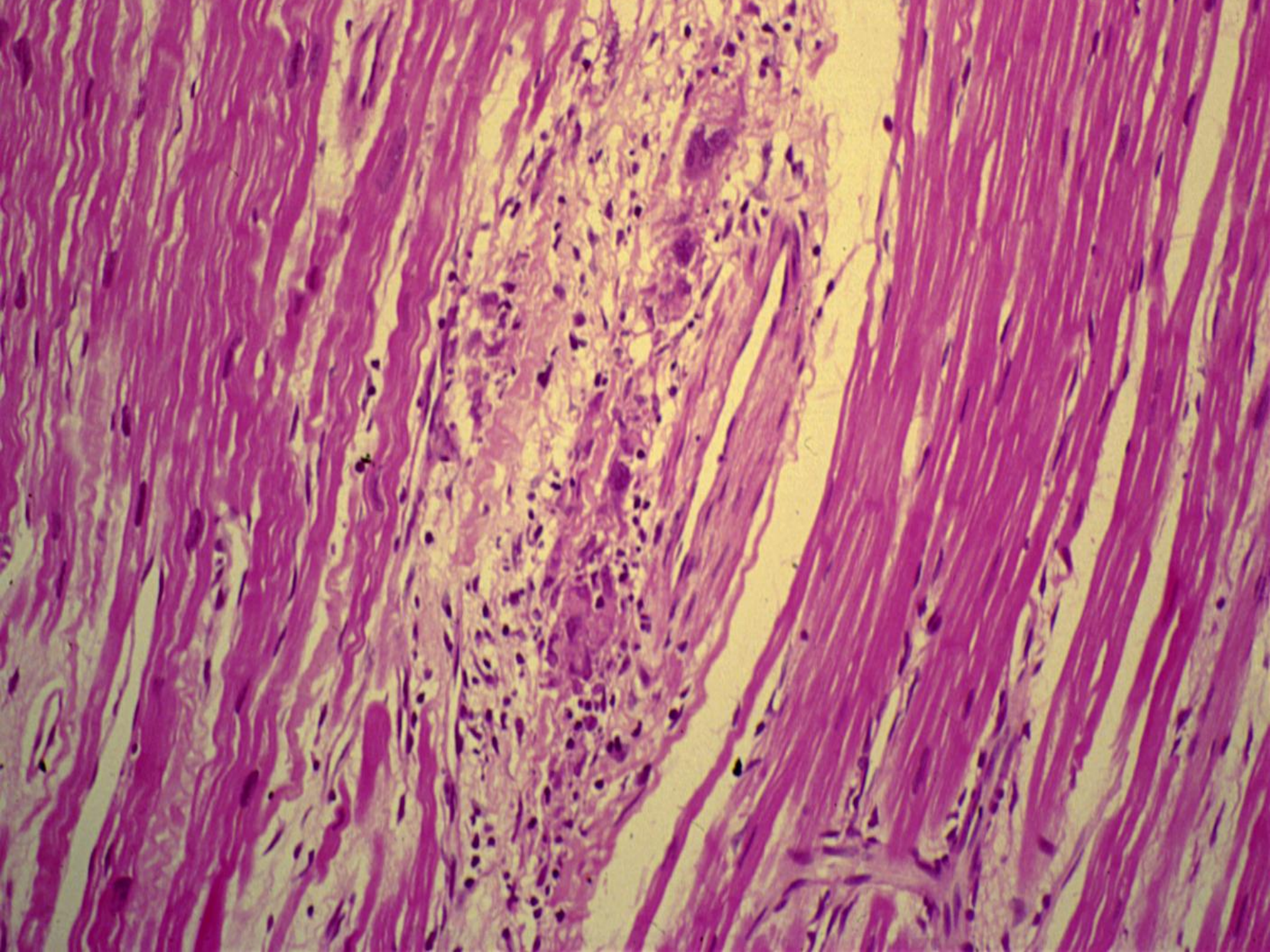


Rheumatic valvulitis:

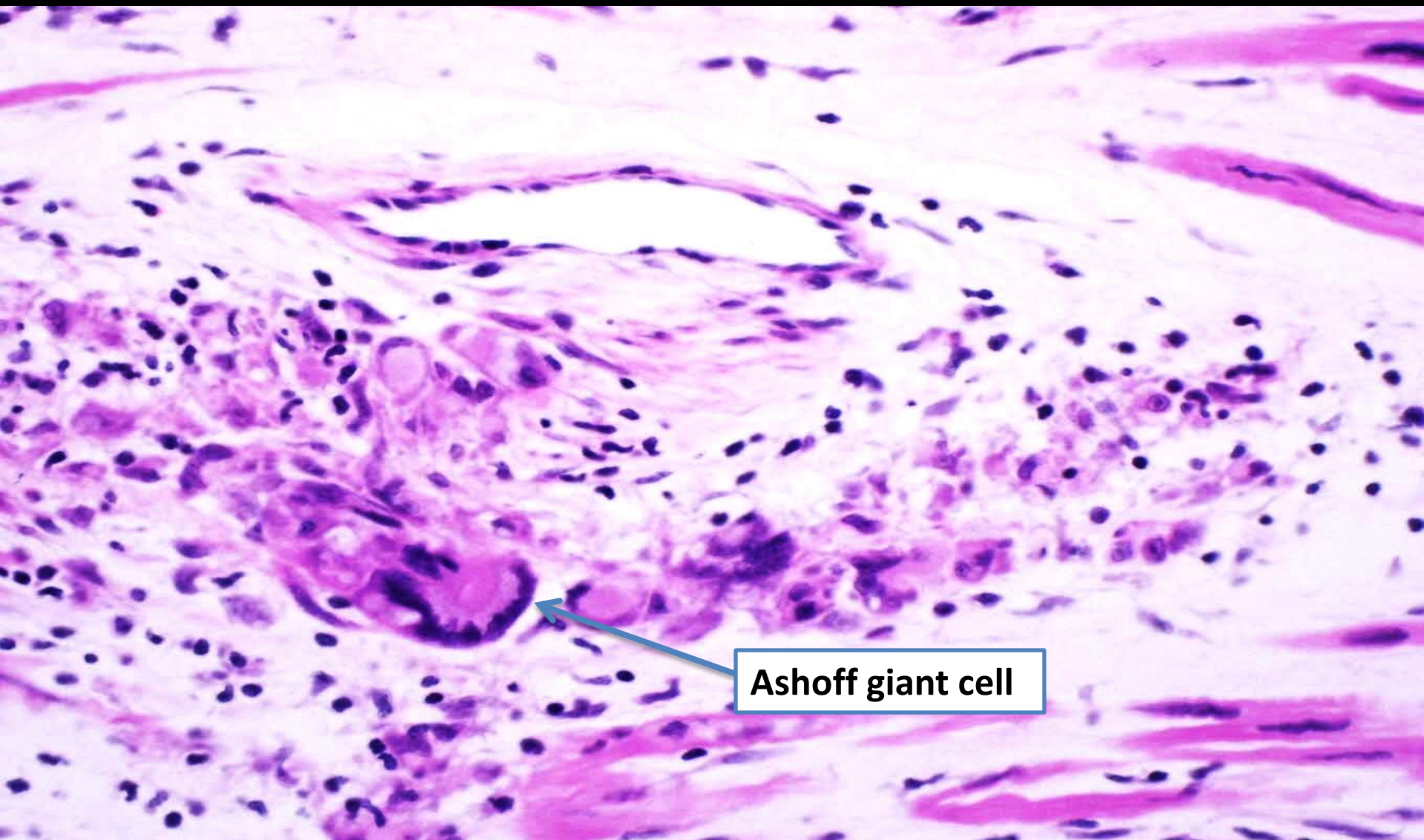
Section of fragments of endocardial valve shows:

- + Irregular endocardial surface, no endocardial lining and focal fibrin deposits.**
- + The valve is thickened by dense hyalinized fibrous tissue with vascularization and chronic inflammatory cell infiltrate.**

7-Acute rheumatic myocarditis





RHEUMATIC MYOCARDIITIS (ASHOFF NODULE)



Ashoff giant cell

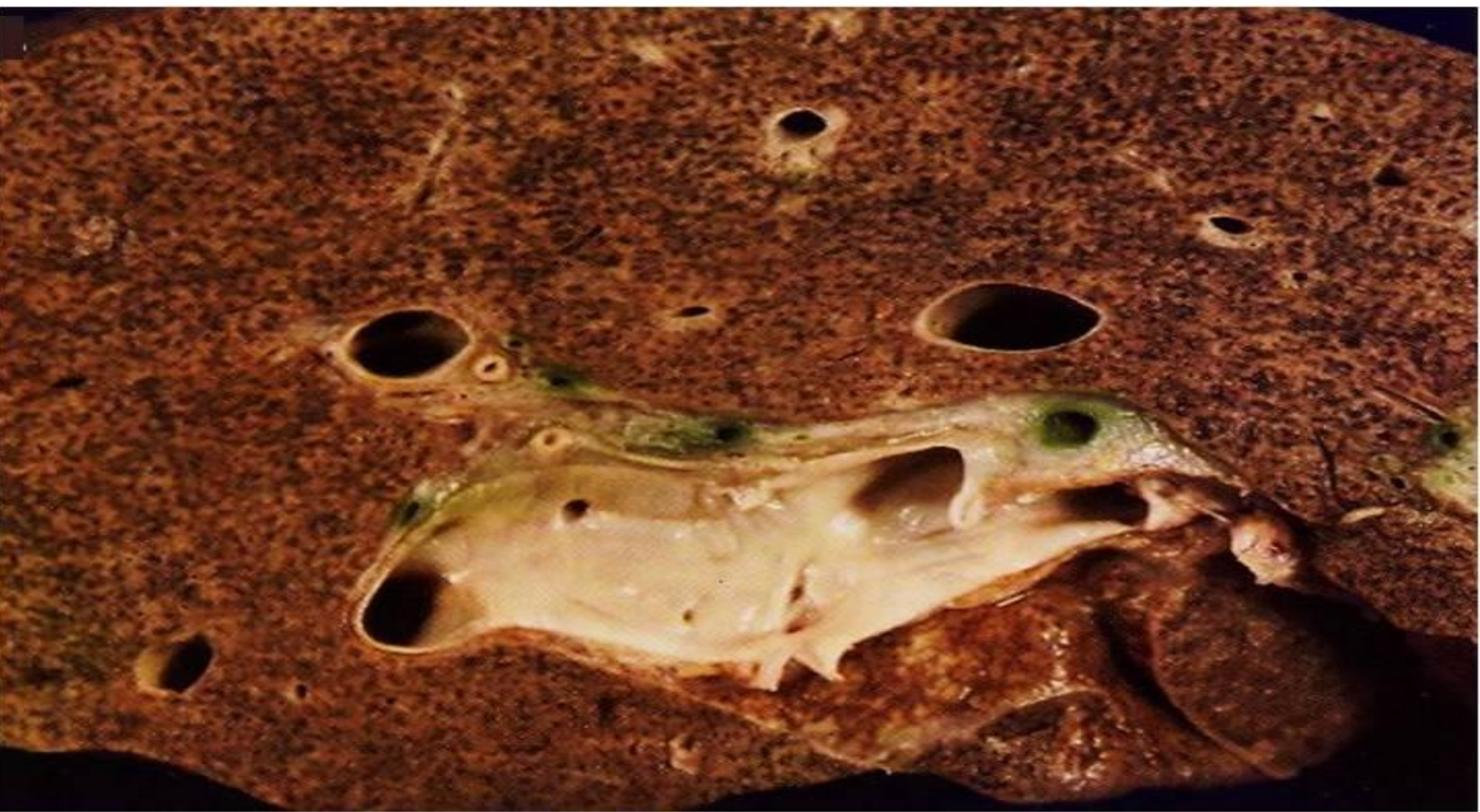
Acute rheumatic myocarditis:

Section of cardiac muscle shows:

-  **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).**

8-Chronic venous congestion of the
liver

“secondary to **RIGHT** heart failure”

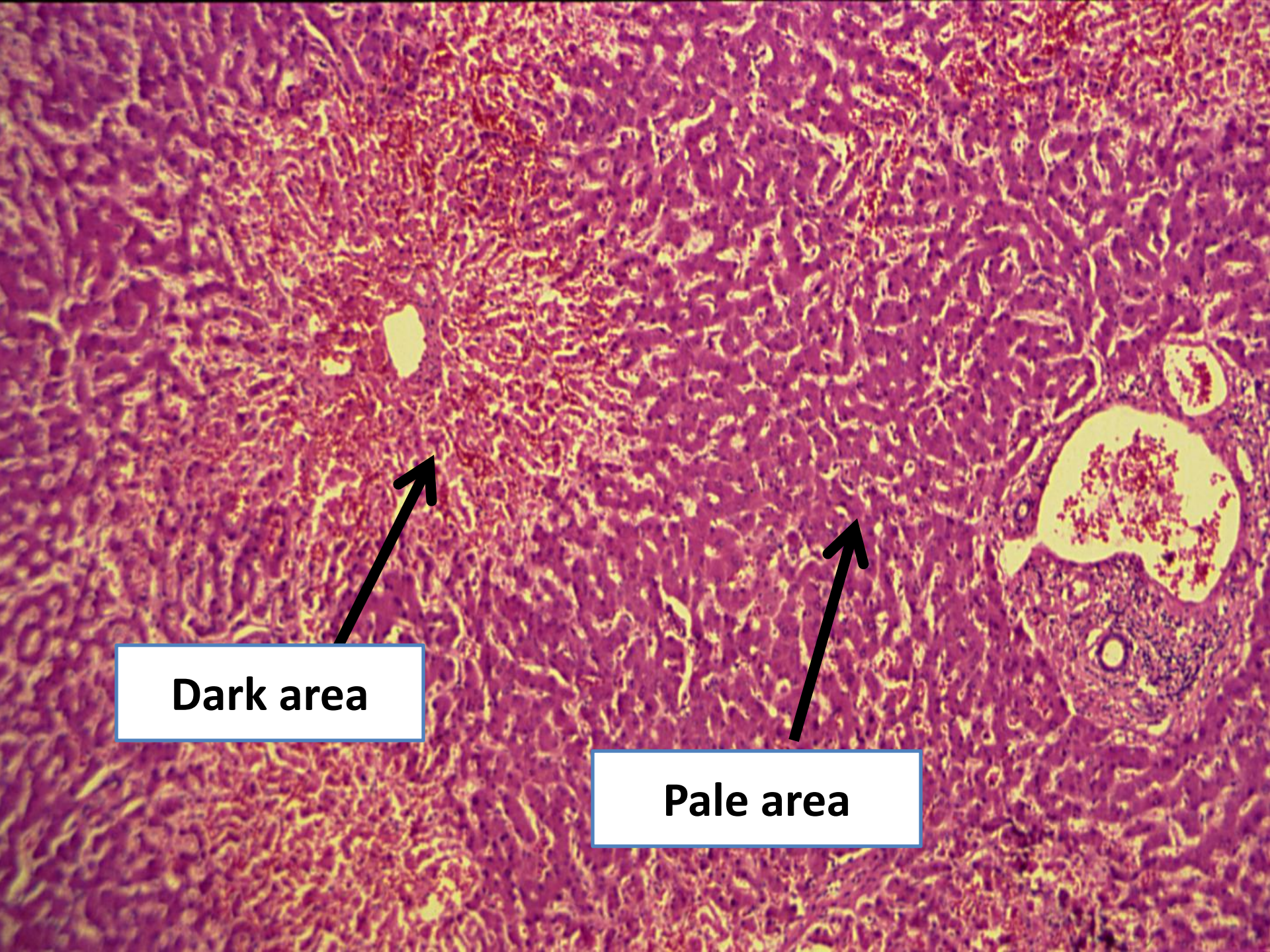


Gross name : **NUTMEG LIVER**

Two areas :

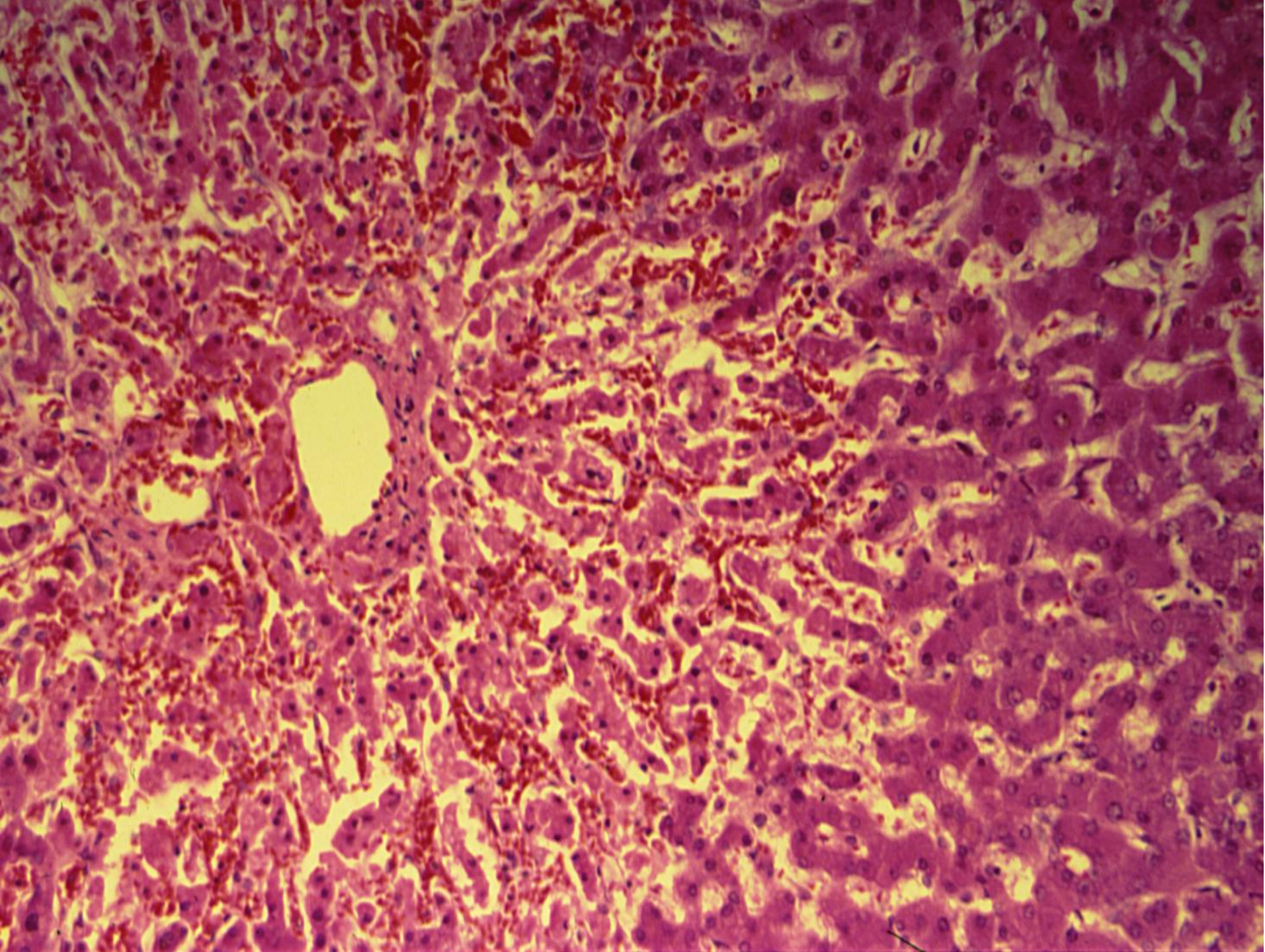
Dark : congested blood

Pale : non-congested blood





Dark area

Pale area



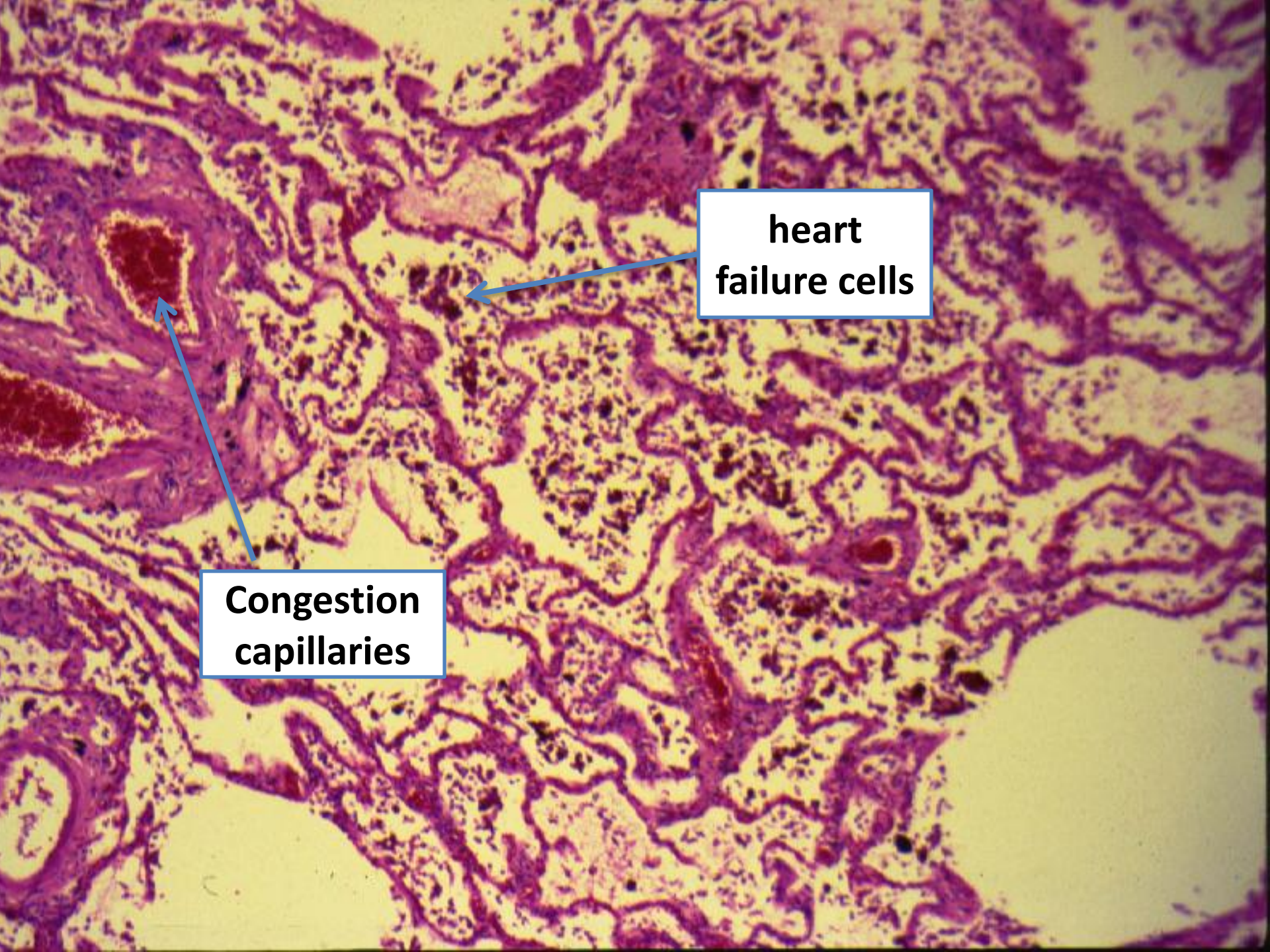
Chronic venous congestion of the liver:

Section of liver shows:

-  **The central portion of liver lobules shows congestion and dilatation of central veins and blood sinusoids, with atrophy and necrosis of liver cells.**
-  **Kupffer cells (macrophage of liver) contain few brown haemosiderin pigment granules.**

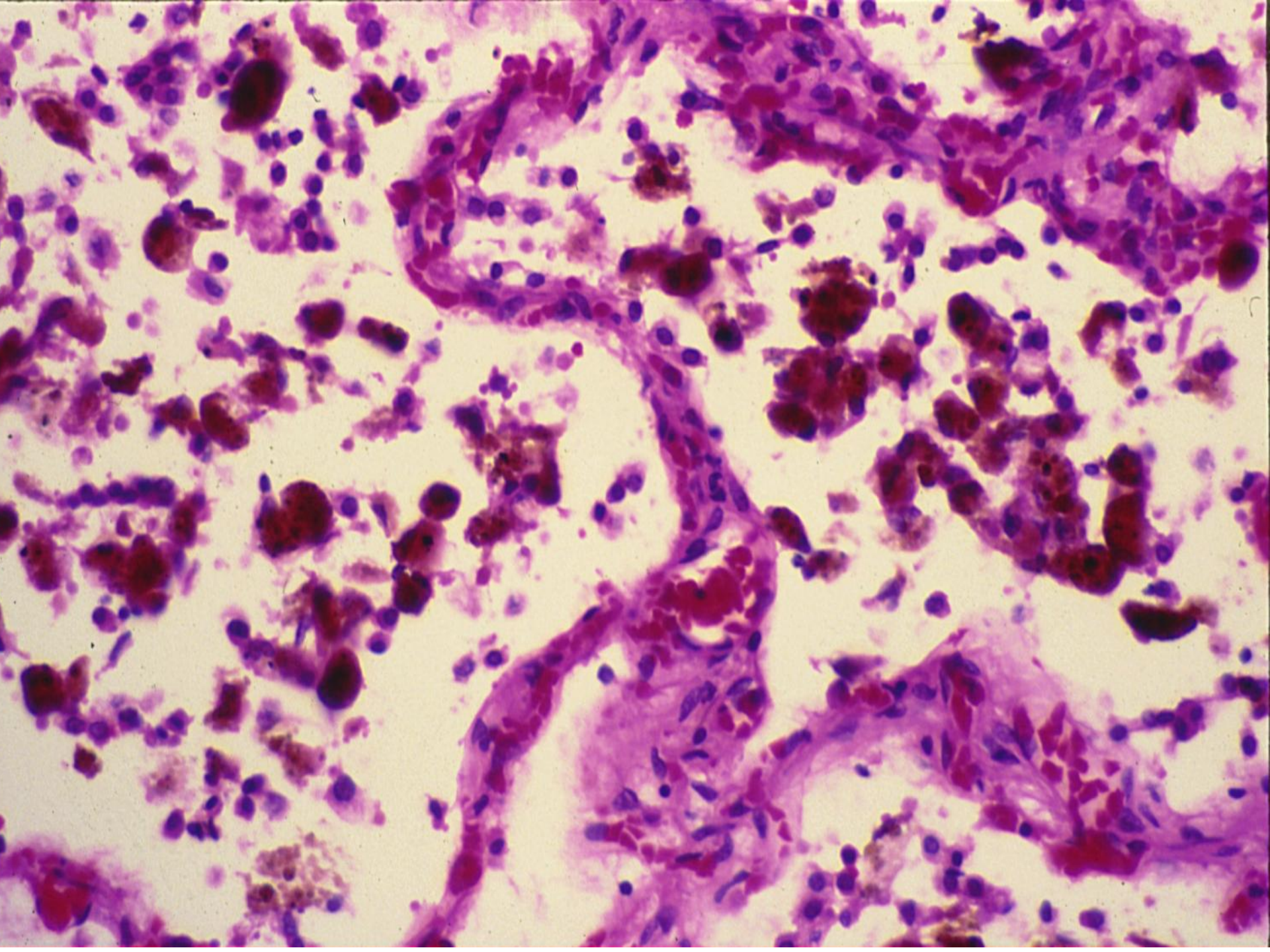
9-Chronic venous congestion of the
lung

“secondary to LEFT heart failure”






**heart
failure cells**

**Congestion
capillaries**



Chronic venous congestion of the lung:

Section of lung shows:

-  **The alveolar walls are thickened by dilated and engorged capillaries.**
-  **The alveoli contain edema fluid, **red blood cells and large alveolar macrophages (heart failure cells), which are filled with haemosiderin pigment** derived from red cells breakdown.**
-  **In the late stage some fibrous tissue may also be seen.**

Vasculitis

Inflammation of vessel walls.

Most common pathogenic mechanisms are :

1- noninfectious (immune-mediated)

- Buerger disease
- Giant cell
- Leukocytoclastic vasculitis

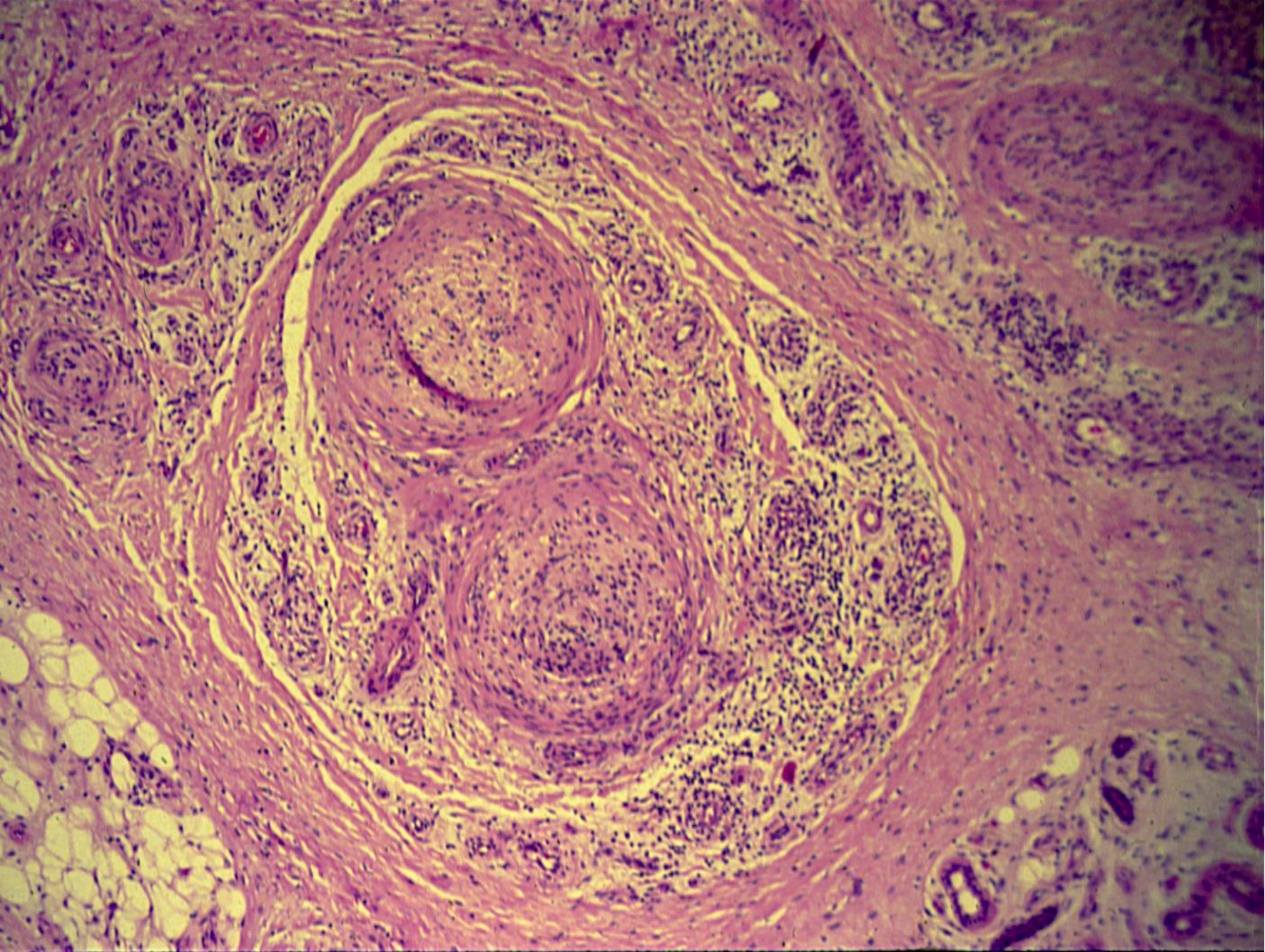
2- infectious

10-Thromboangitis obliterans (Buerger disease)

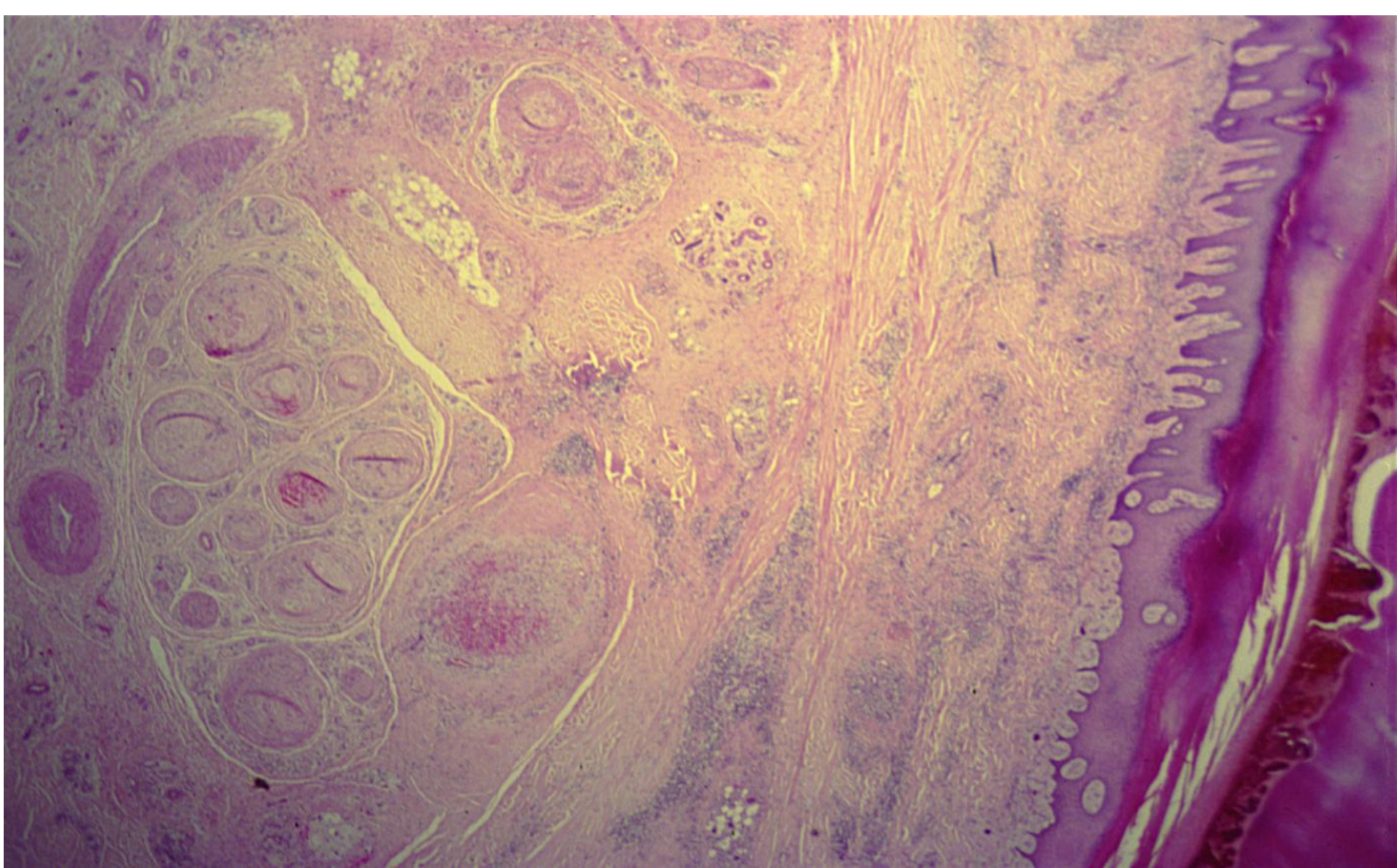


Thromboangitis obliterans (Buerger disease)

- **It is related to smoking**
- **It affects small & medium size arteries**
- **Examples: fingers, toes, tibial and radial artery**



THROMBOANGITIS OBLITERANS



Thromboangitis obliterans (Buerger's disease):

Section of the skin and subcutaneous tissue shows marked hyperkeratosis with inflammatory exudate in epidermis:

- ✚ Section : Skin**
- ✚ Large number of small blood vessels in the dermis show occlusive organized thrombi with recanalization and fibrosis around blood vessels.**
- ✚ Some blood vessels show recent organizing thrombi while others show infiltration of the wall and surrounding tissue by chronic inflammatory cells.**
- ✚ When reach nerves , pain occur**

11-Giant cell (temporal) arteritis

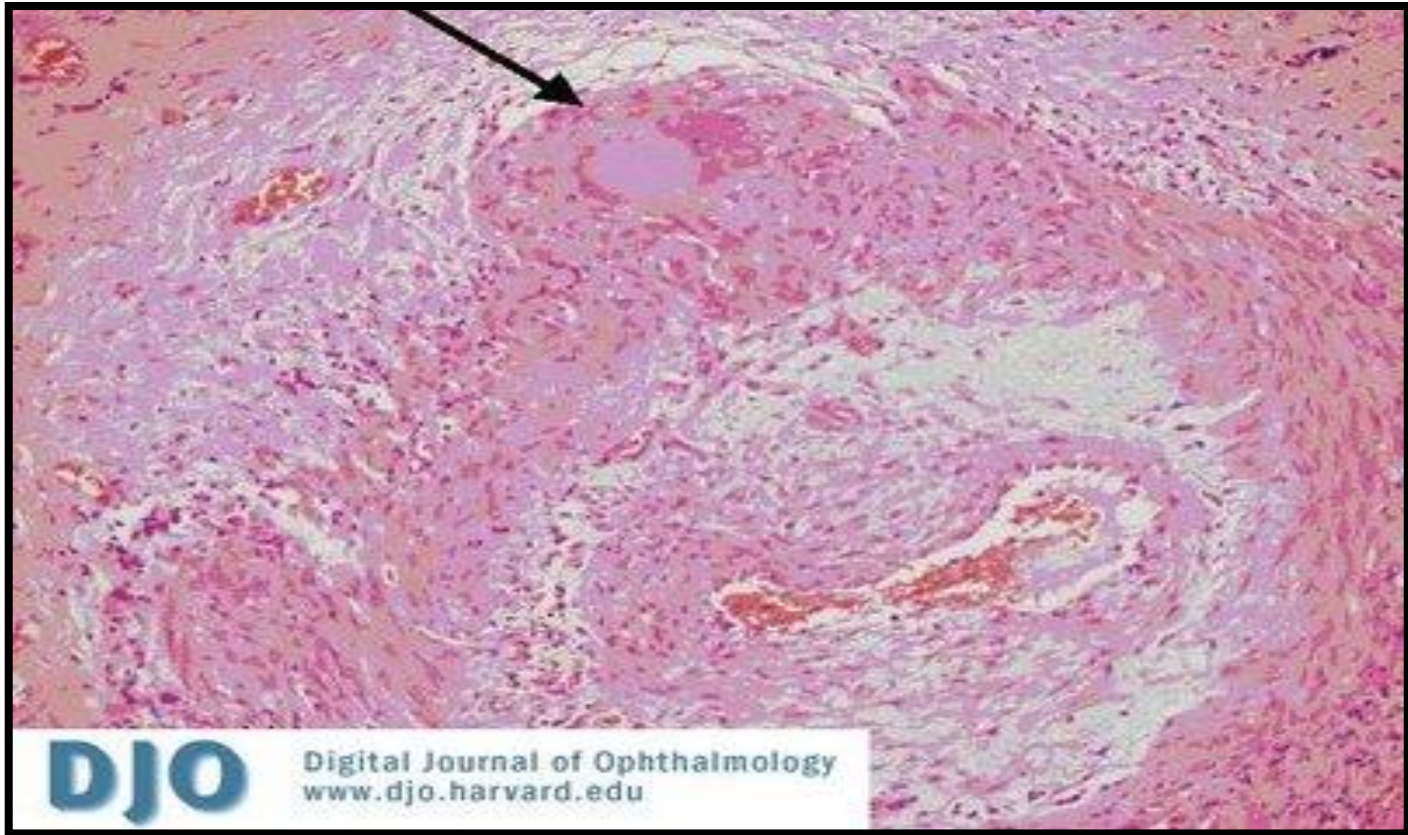
Giant cell arteritis

- Chronic granulomatous inflammation “segmental” of the temporal artery
- lead to media damage and intimal thickening
- affect medium & large vessels (aorta)

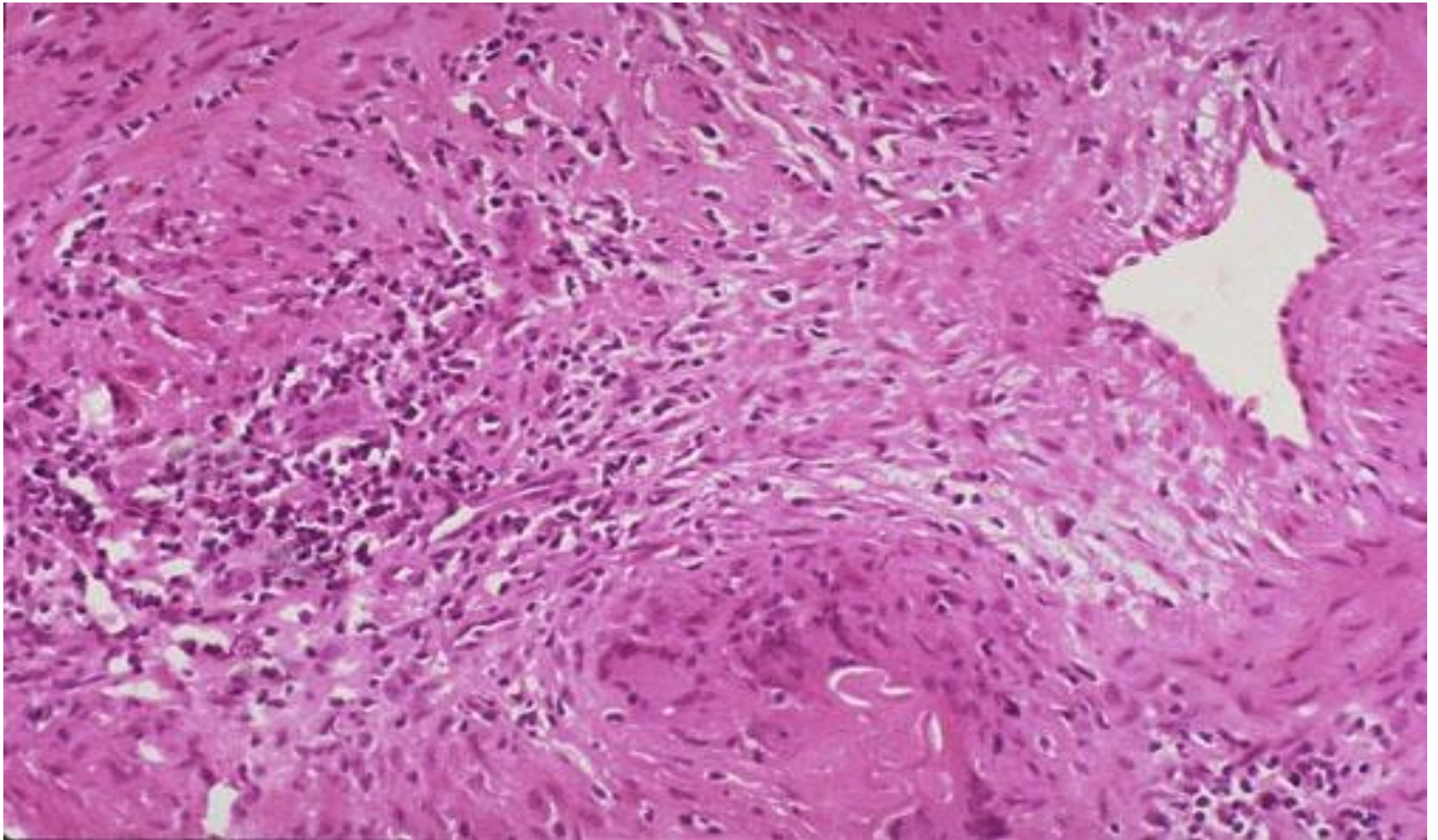


Tender and thickened scalp veins

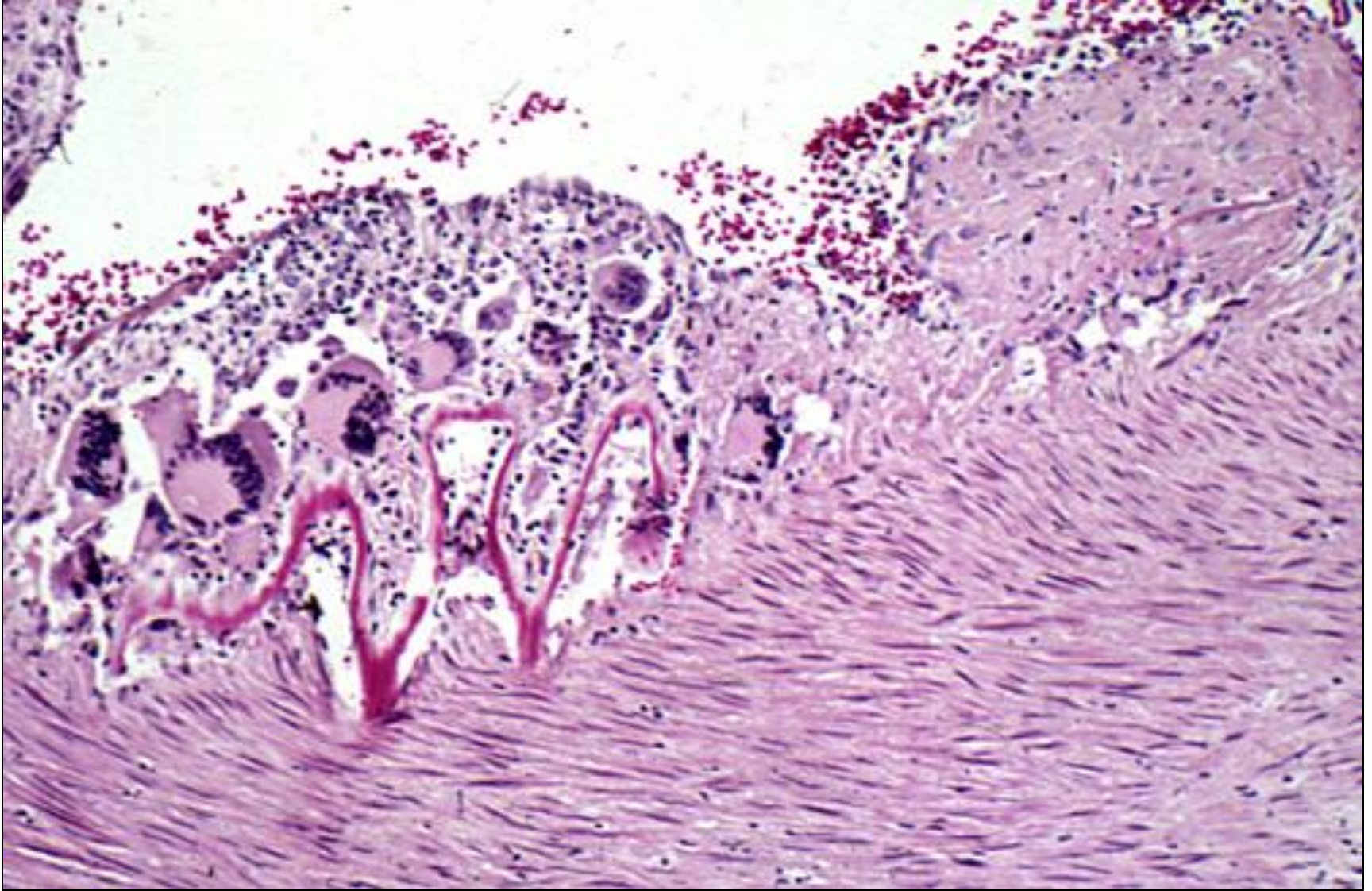
Giant cell arteritis



Superficial temporal artery biopsy - **intimal thickening and medial damage**, giant cells with inflammatory cell infiltration in the internal elastic lamina



This temporal artery at medium power shows features of giant cell arteritis, **with giant cells in the media.**

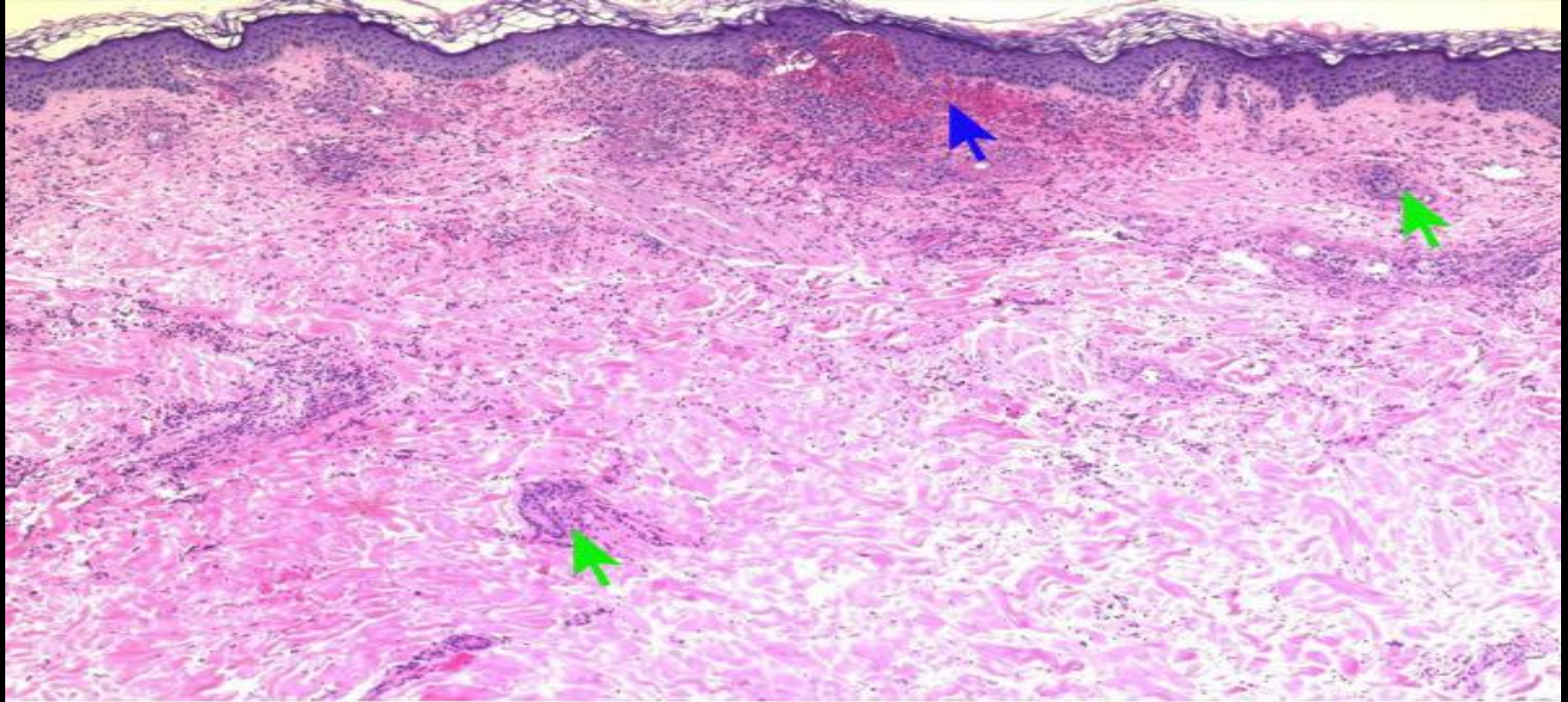


Segmental inflammatory lesions with intimal thickening , medial granulomatous inflammation with giant cells and chronic inflammatory cells and internal elastic lamina fragmentation .

12-Leukocytoclastic vasculitis

- It is hypersensitivity reaction
- Can affect venules, arteriols and capillaries

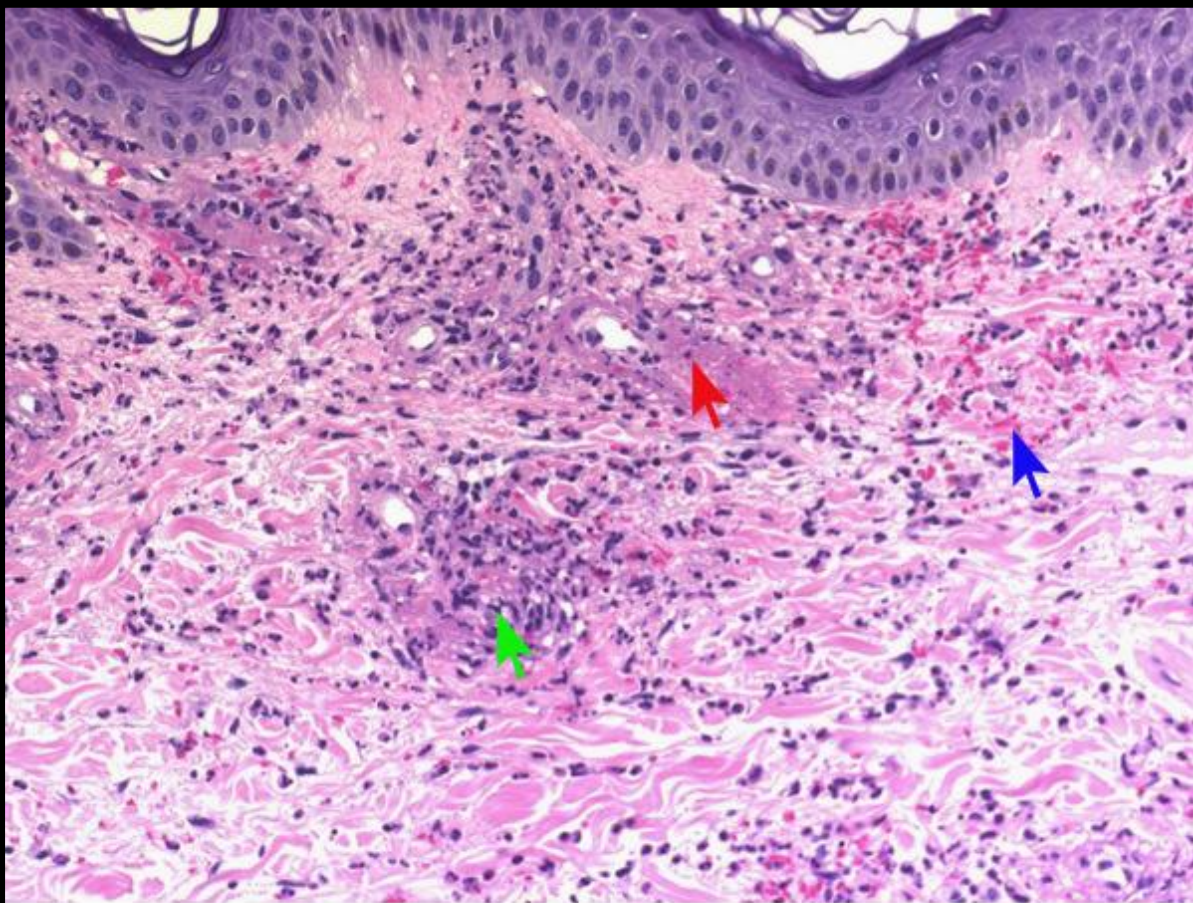







 **Red cell extravasation**

 **Inflammation**

Histology: vasculitis, leukocytoclastic, low power



-  **Fibrinoid type necrosis**
-  **Red cell extravasation**
-  **Inflammation**

Histology: vasculitis, leukocytoclastic, high power