

# **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?**

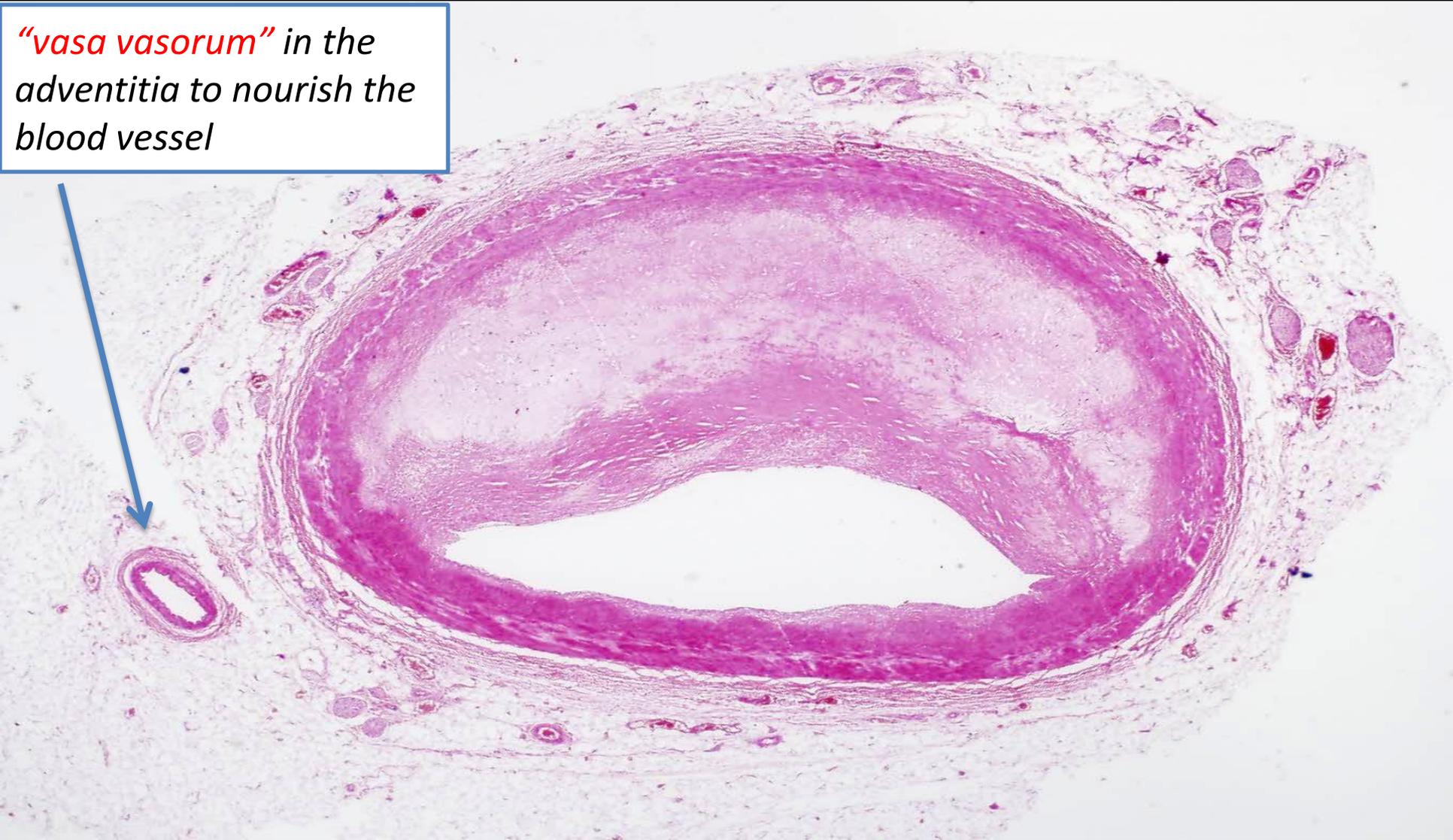
**A: 1) increasing age 2) Hyperlipidemia 3) Hypertension 4) Obesity**



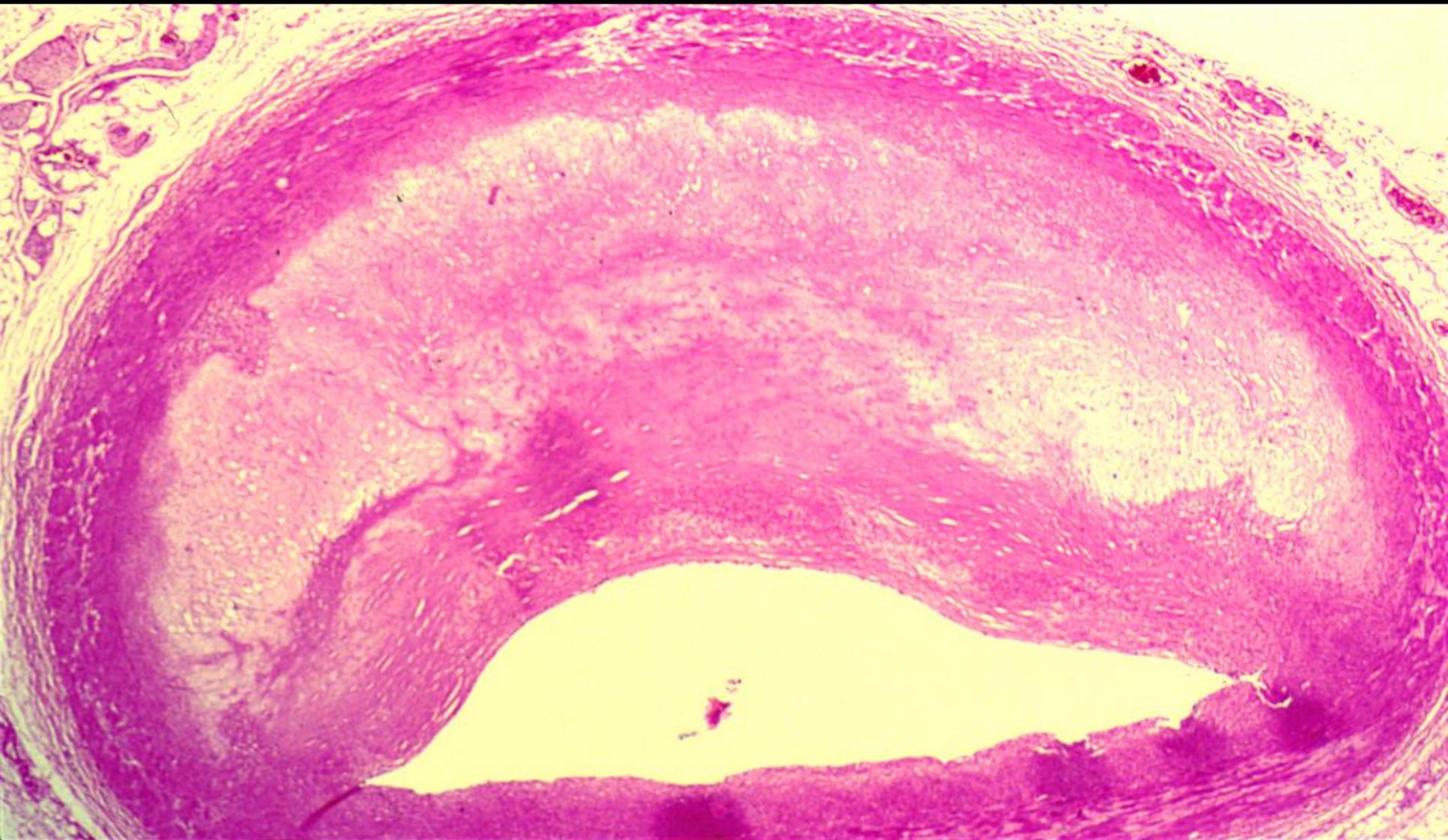
## 2-Coronary atherosclerosis

# CORONARY ARTERY ATHEROSCLEROSIS

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



# CORONARY ATHEROSCLEROSIS



# ***Coronary atherosclerosis:***

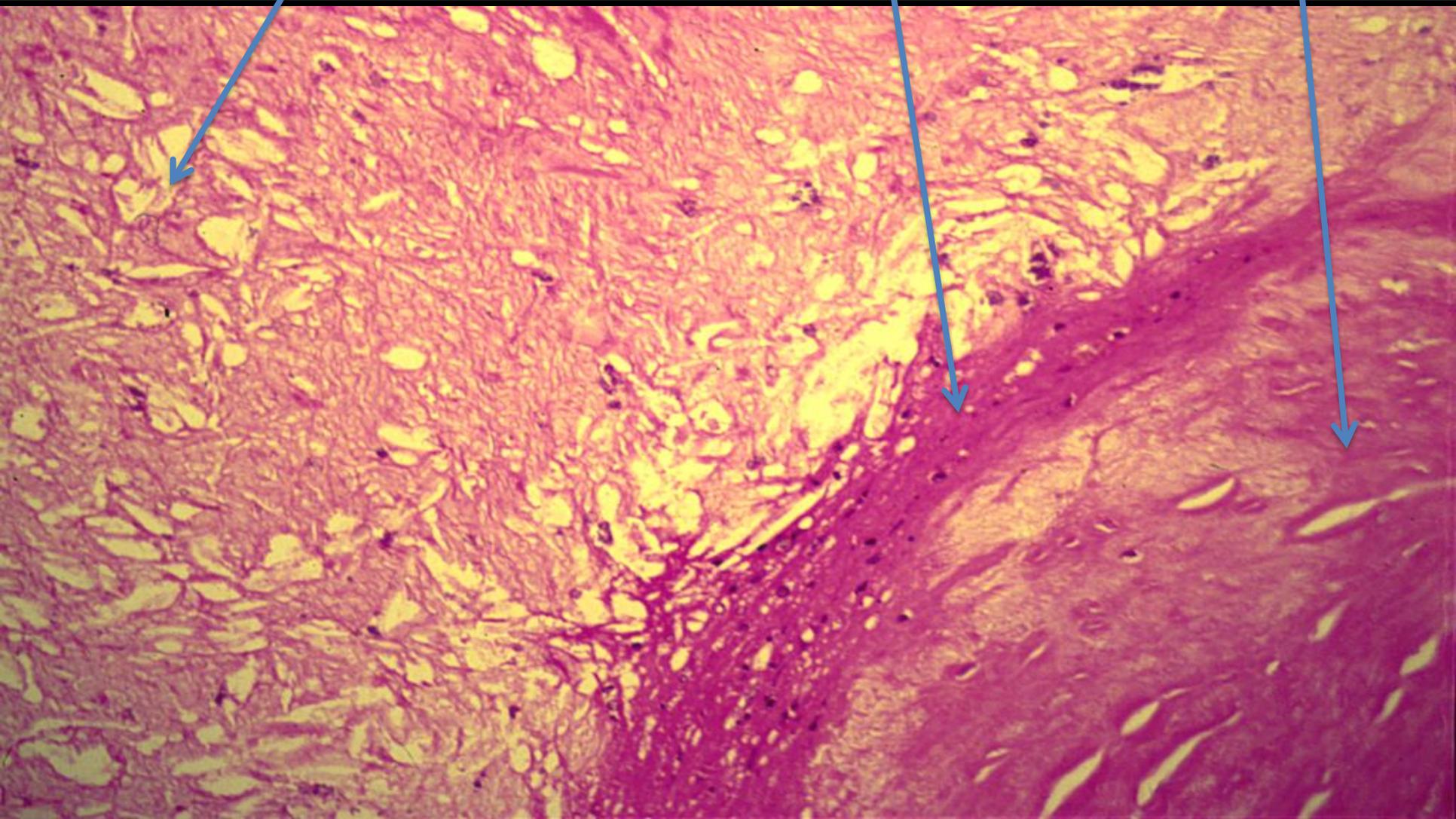
***Cross section of a coronary artery shows:***

- + 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.**

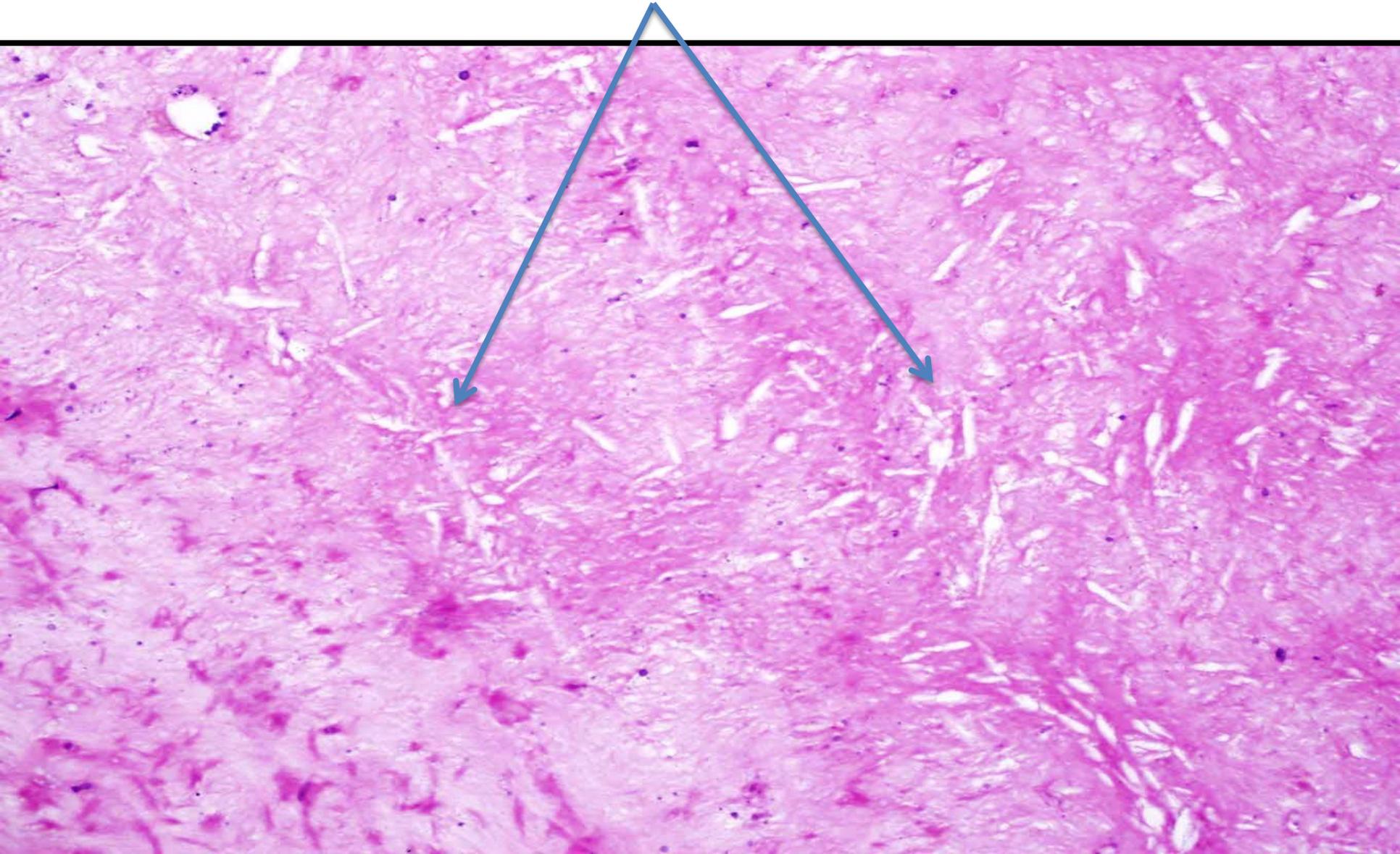
**cholesterol clefts**

**Internal elastic lamina**

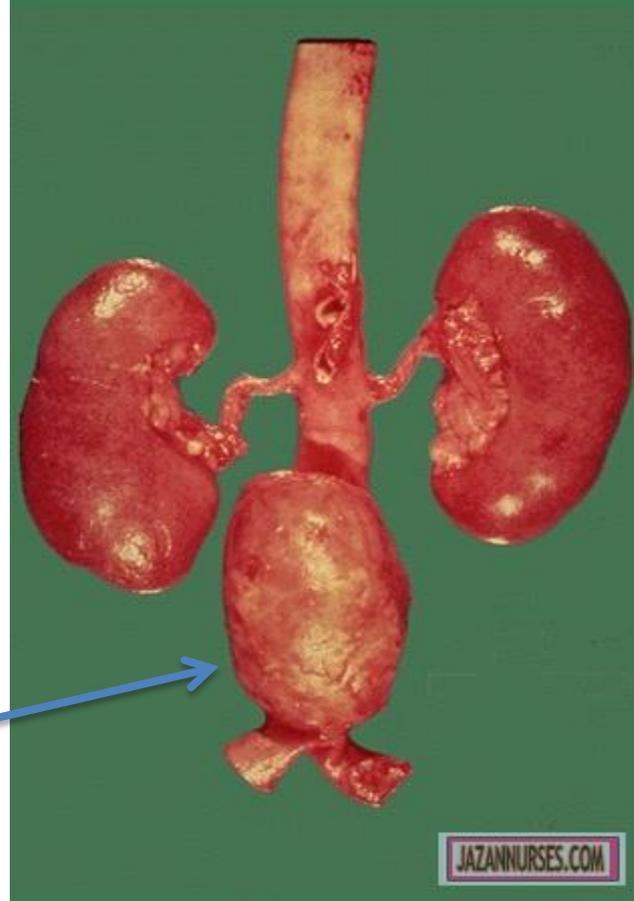
**Intimal thickening**



# 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?

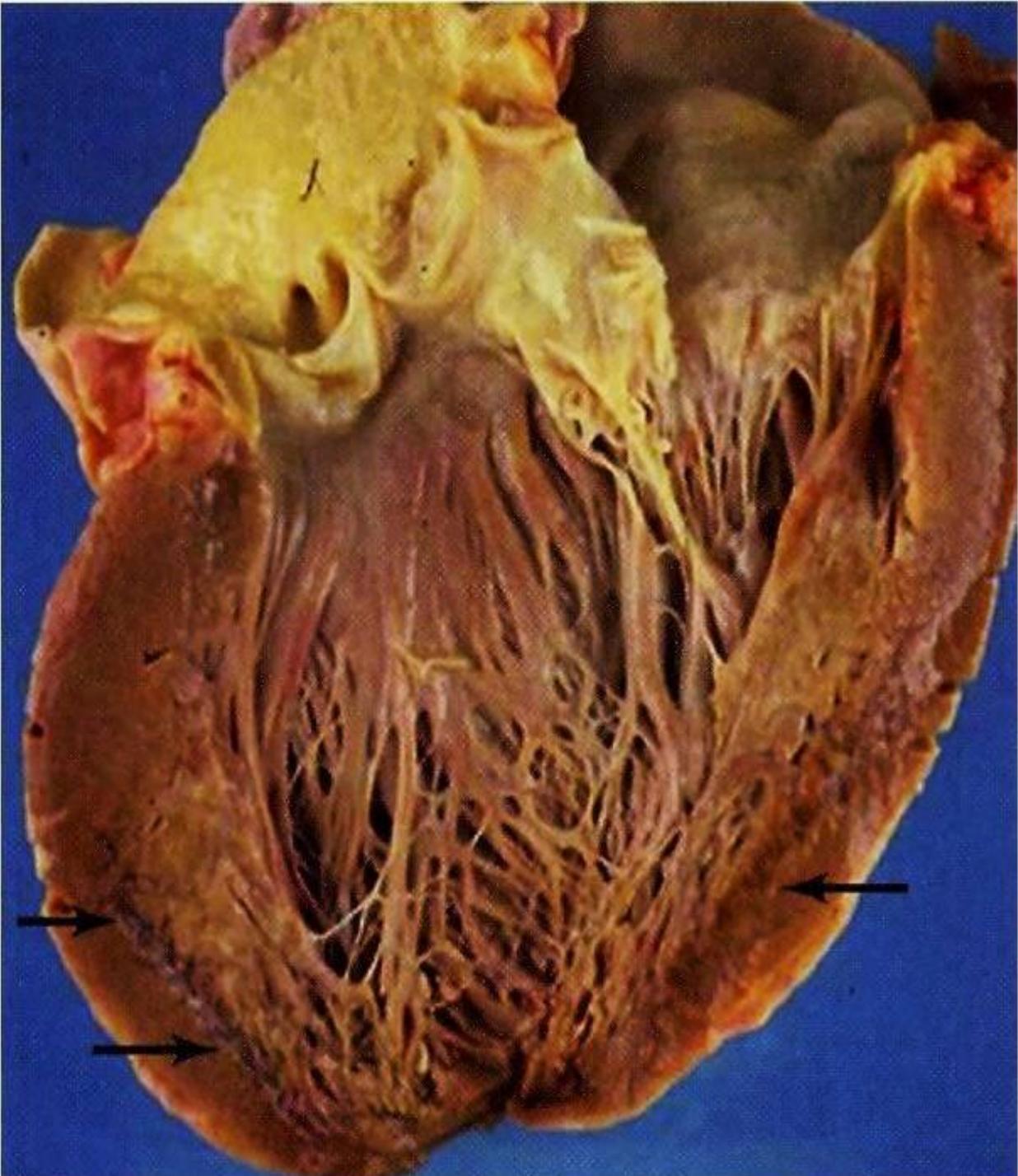
A: Aorta

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

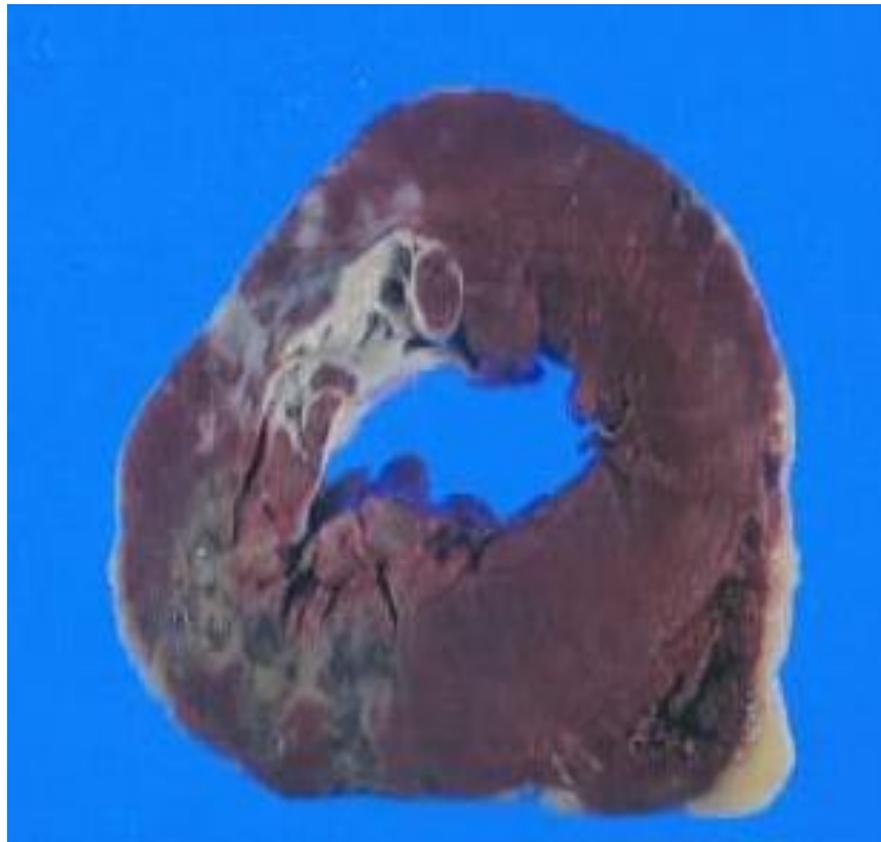
A: Atherosclerosis



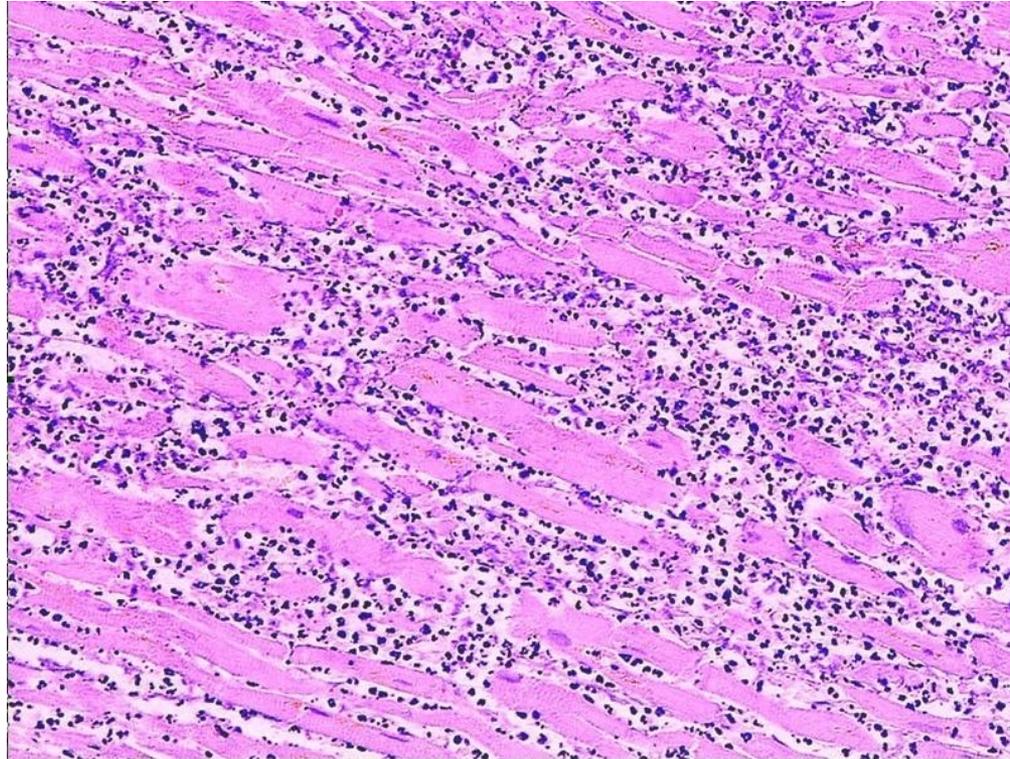
# 4-Myocardial infarction



# Myocardial infarction



# Myocardial infarction (recent stage)

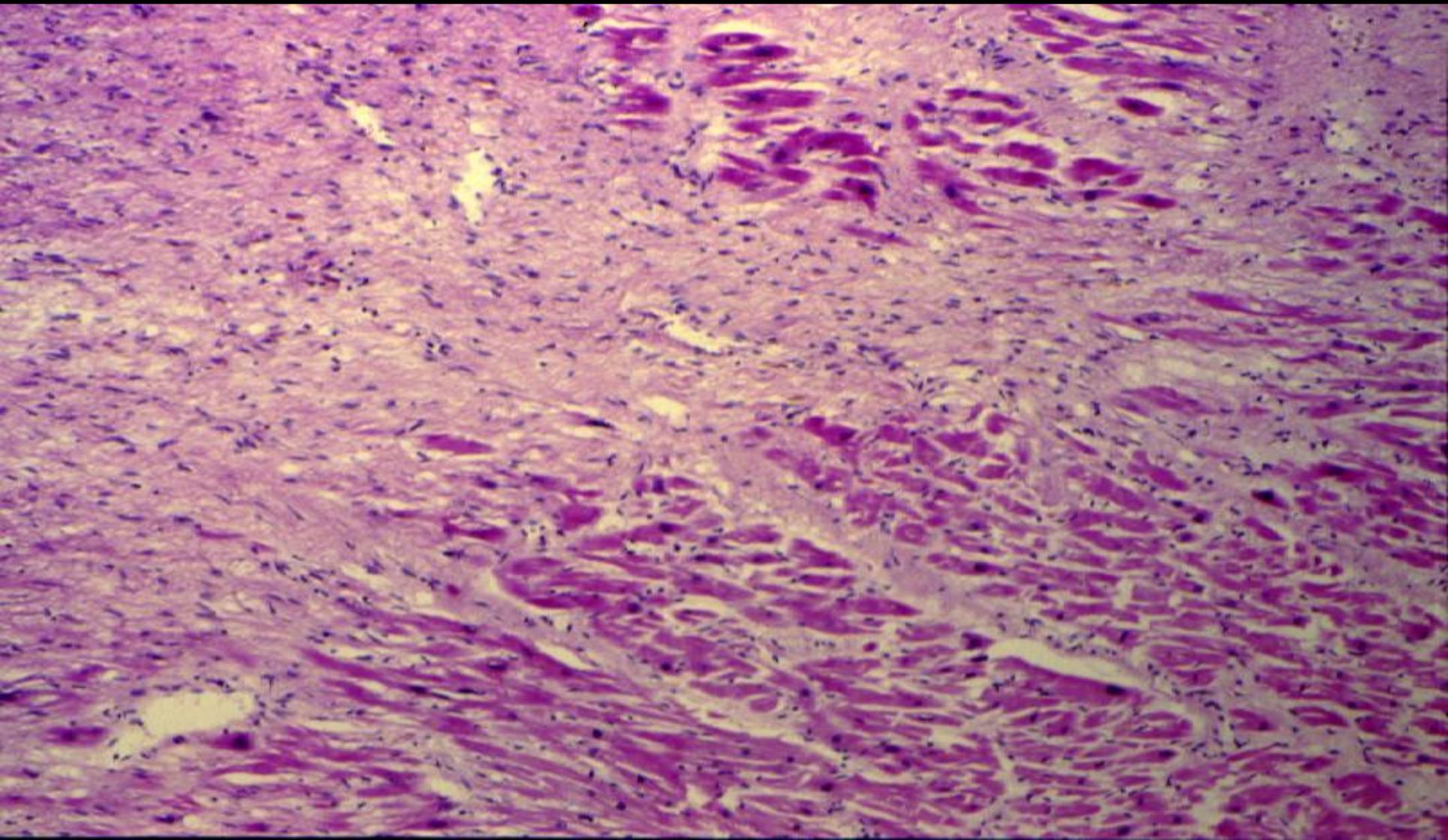


**Infiltration of neutrophils into myocardial muscle fibers**

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:**

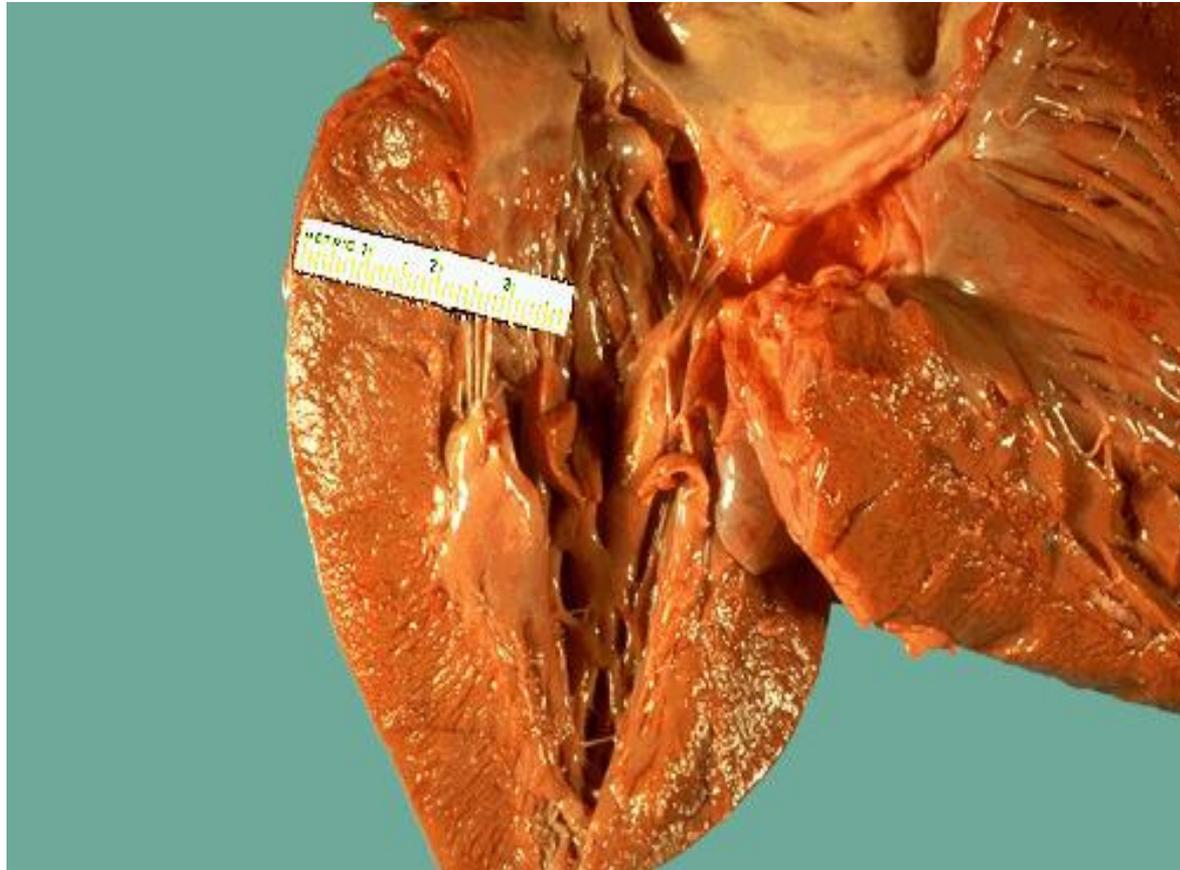
- 1. Inflammatory cells**
- 2. Fibroblasts**
- 3. Neovascularization**

# ***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



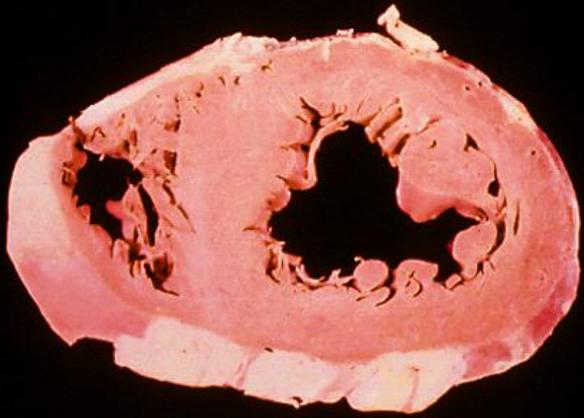
**The normal size of left ventricle is 1.5 cm**

**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?**

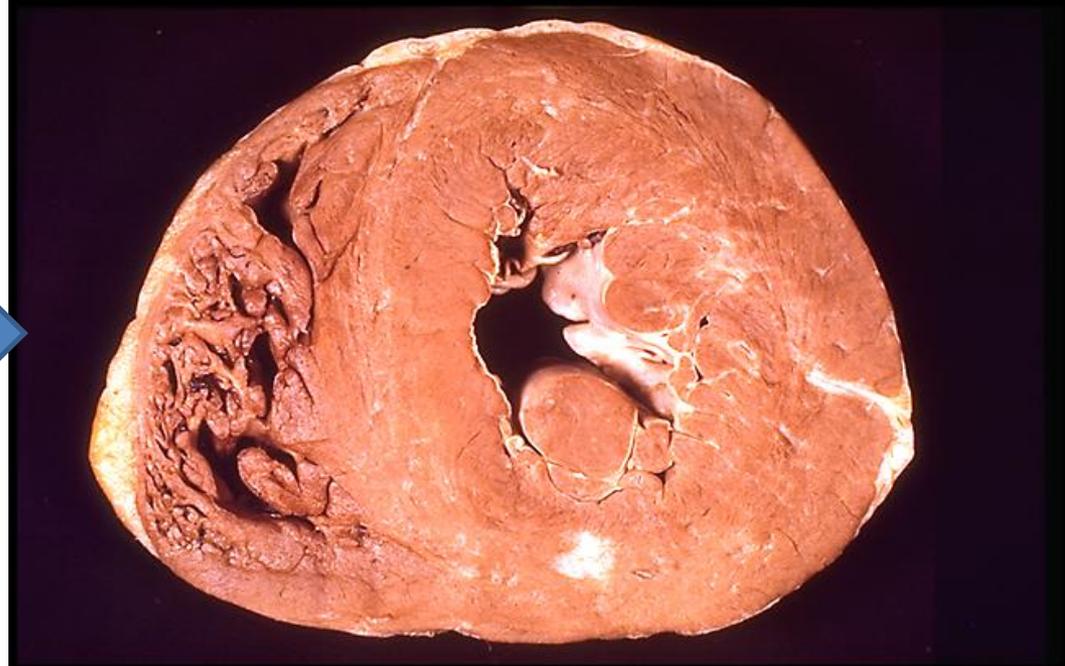
**A: Heart**

**Q: what is the pathology?**

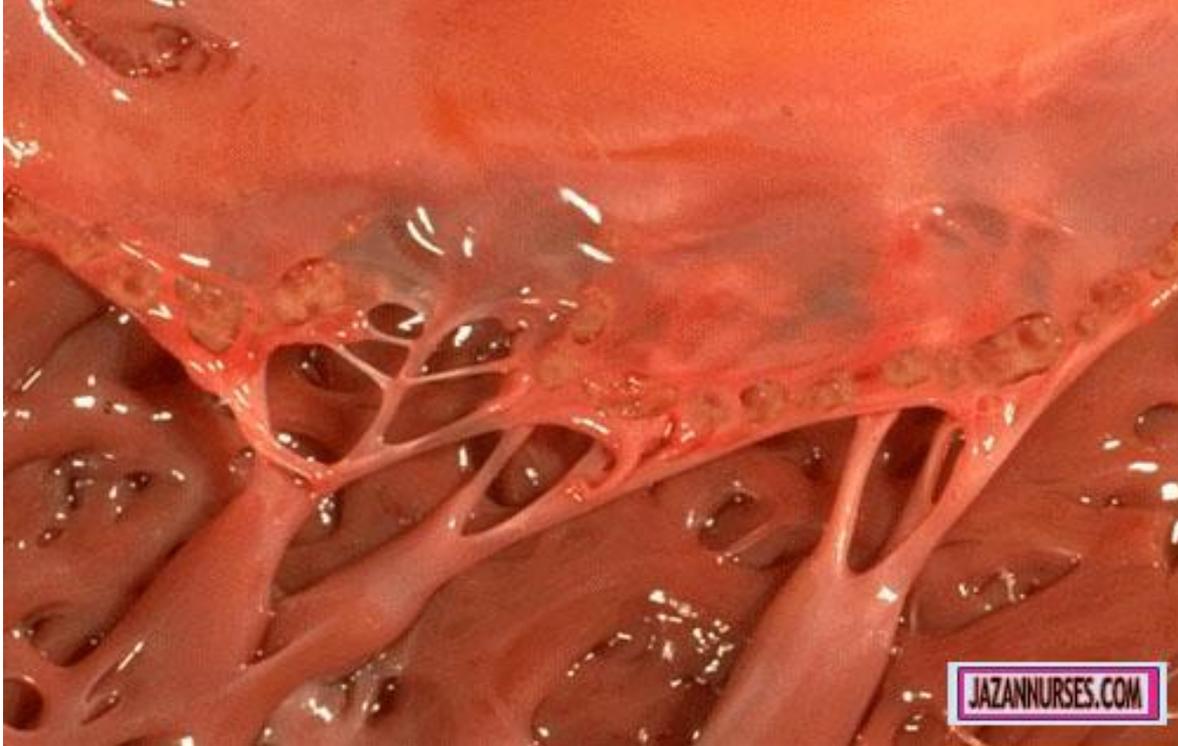
**A: left ventricular hypertrophy**

**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?**

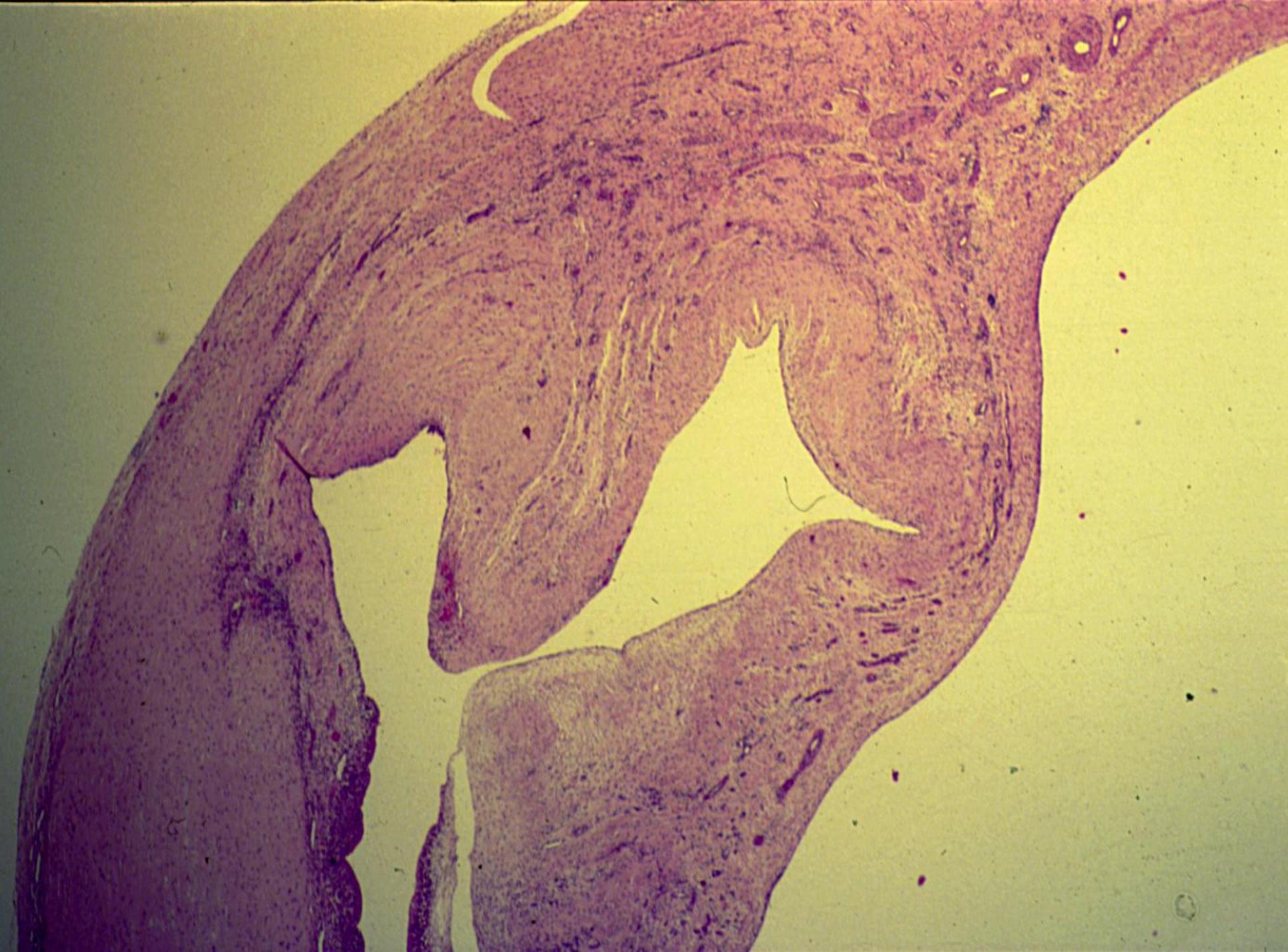
**A: Mitral Valve**

**Q: what is the pathology?**

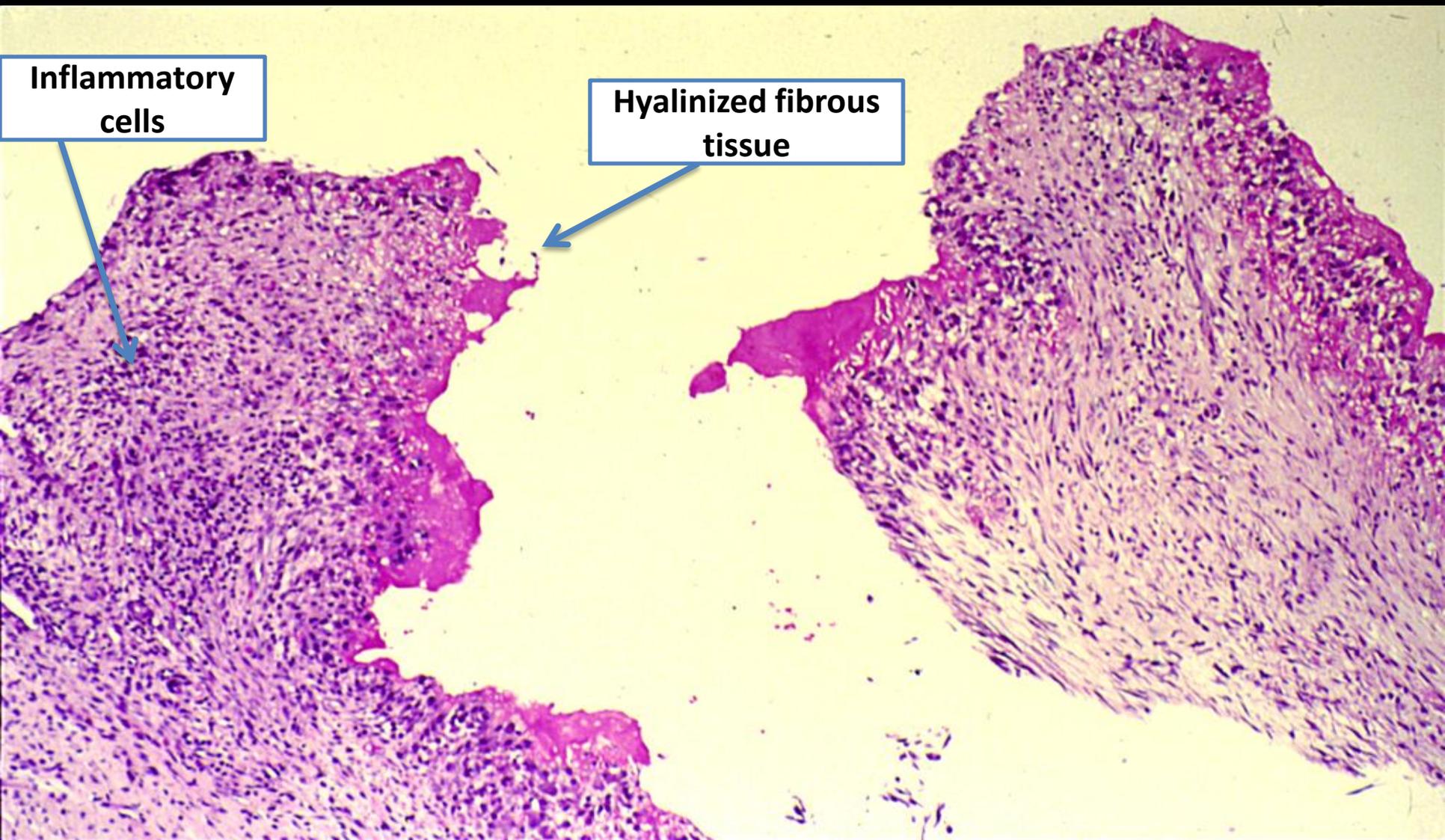
**A: Rheumatic vegetations**

## Vegetations on the Aortic valve

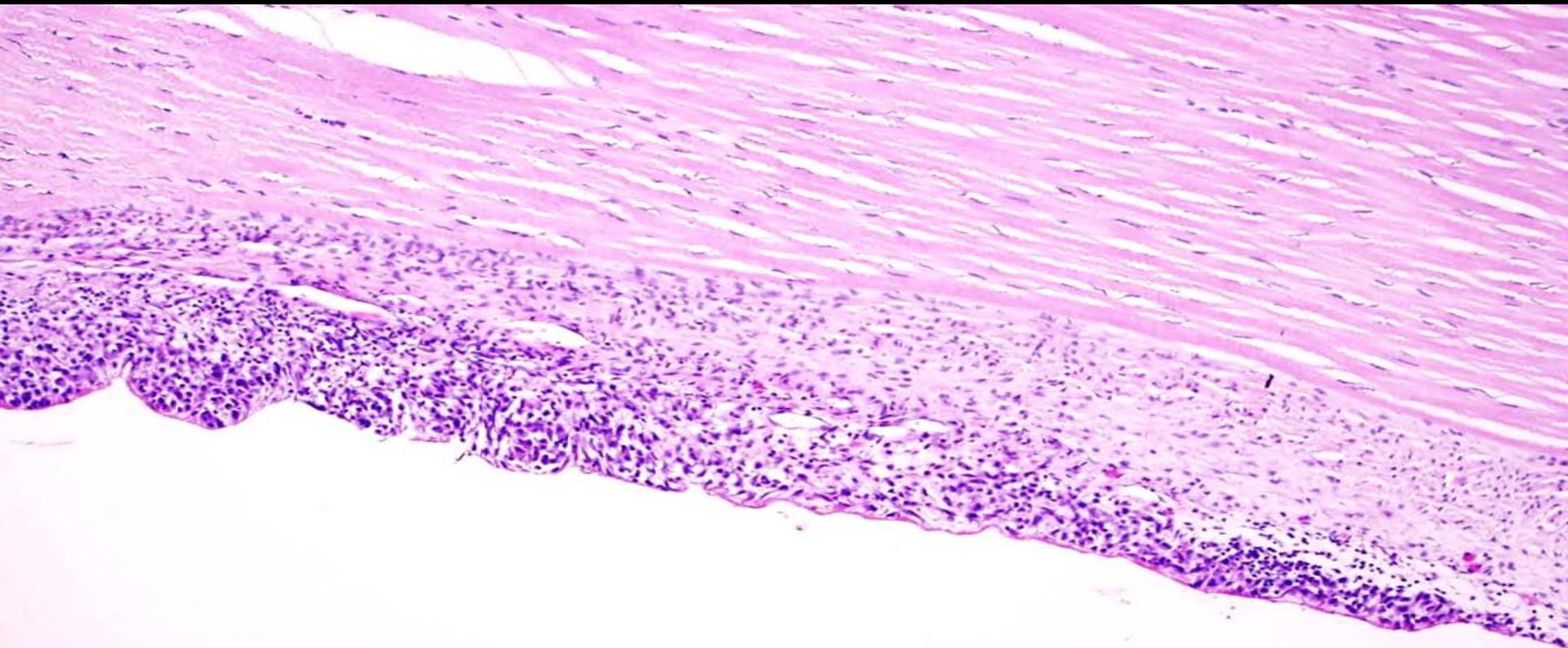




# RHEUMATIC VALVULITIS (HEART)



# RHEUMATIC VALVULITIS (HEART)

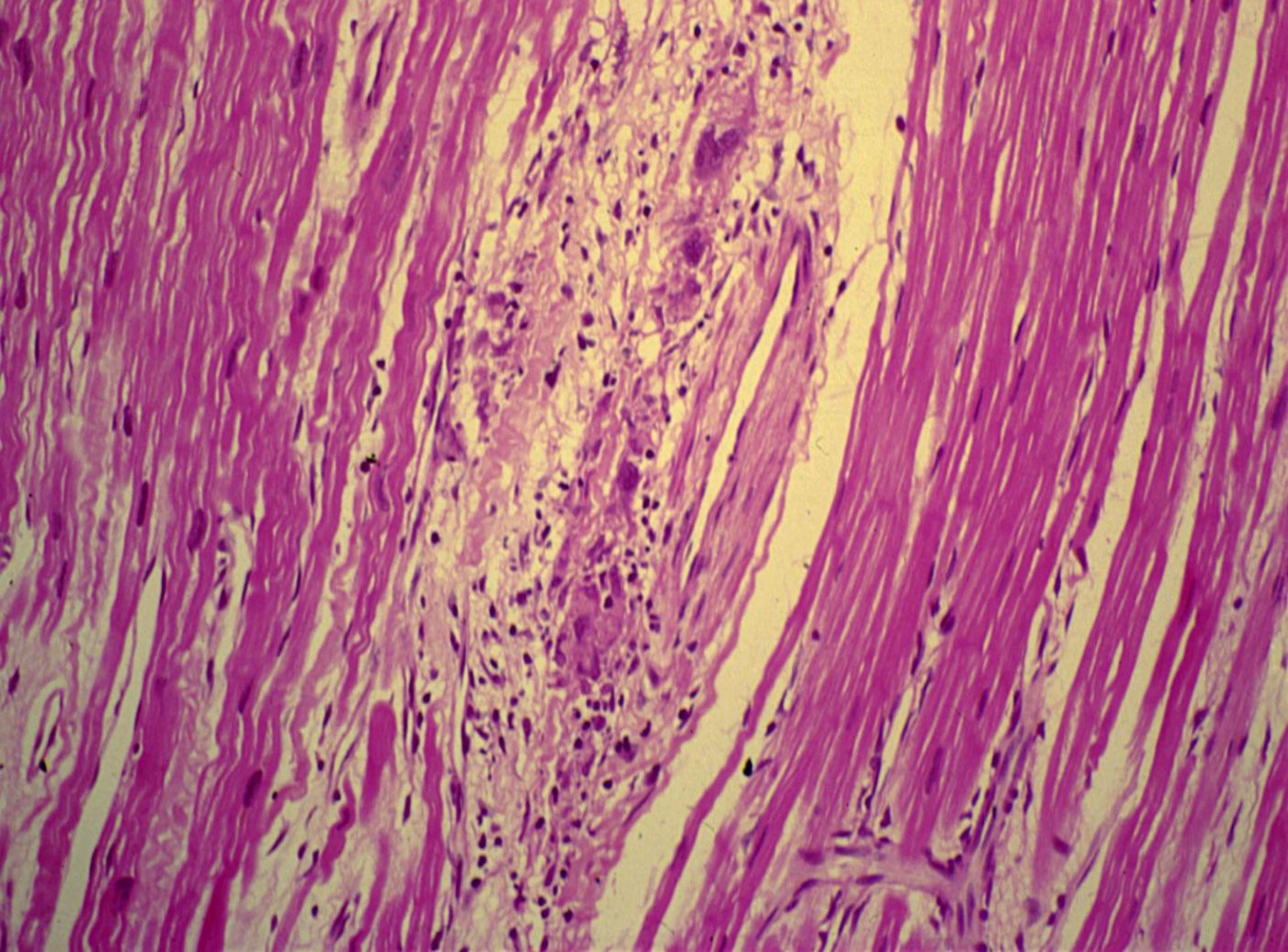


# ***Rheumatic valvulitis:***

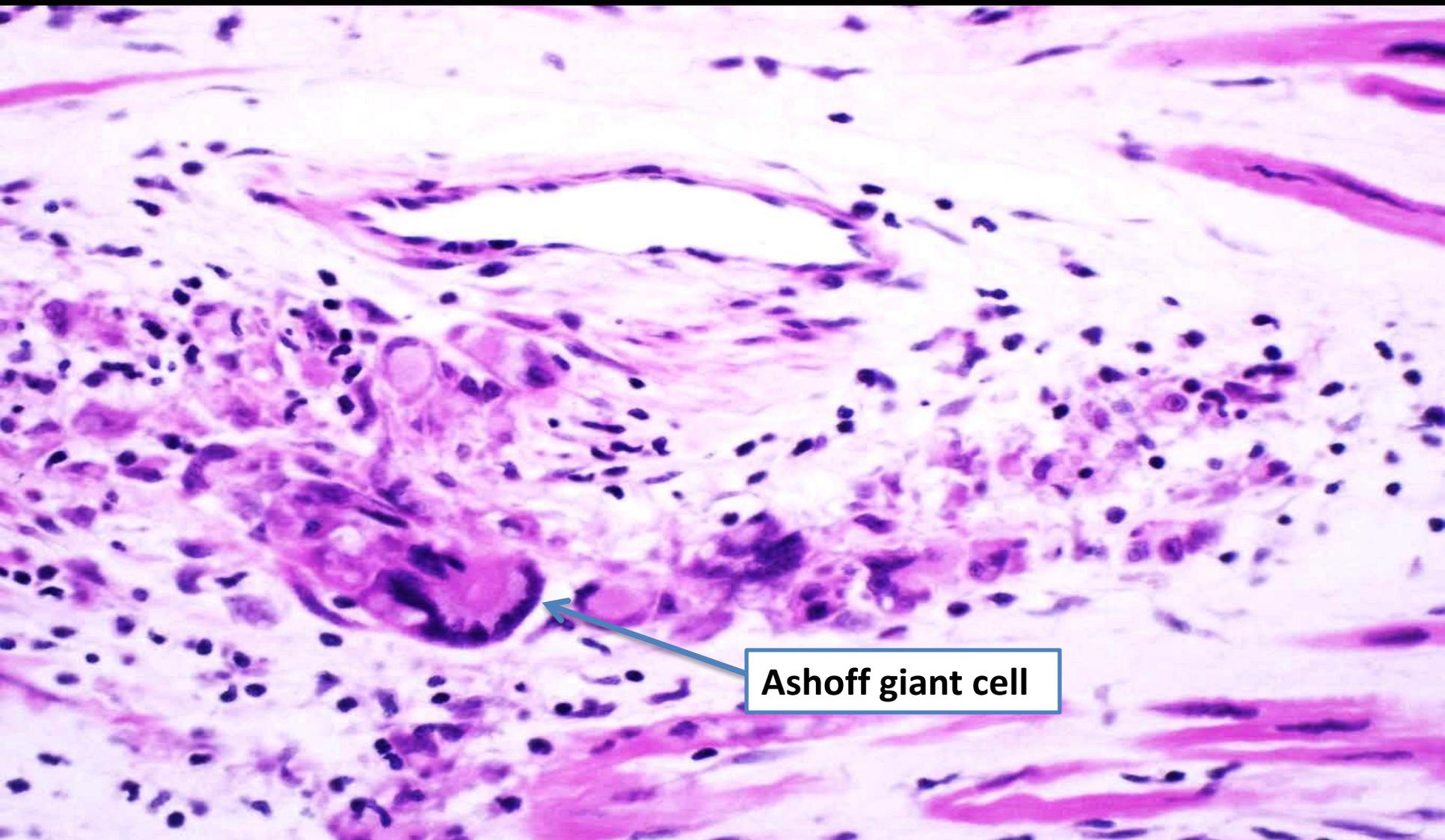
***Section of fragments of endocardial valve shows:***

- + 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

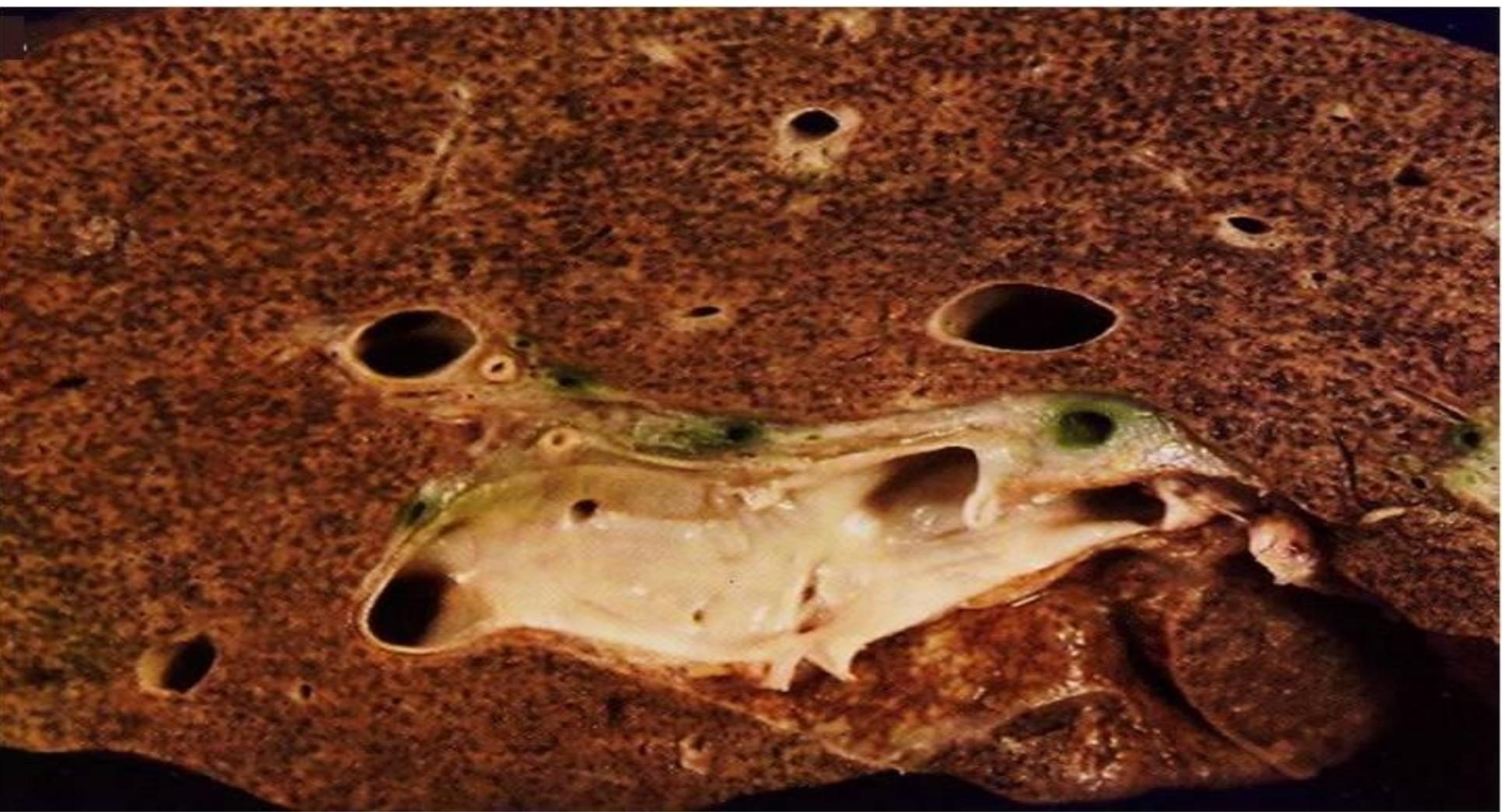
# ***Acute rheumatic myocarditis:***

## ***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”

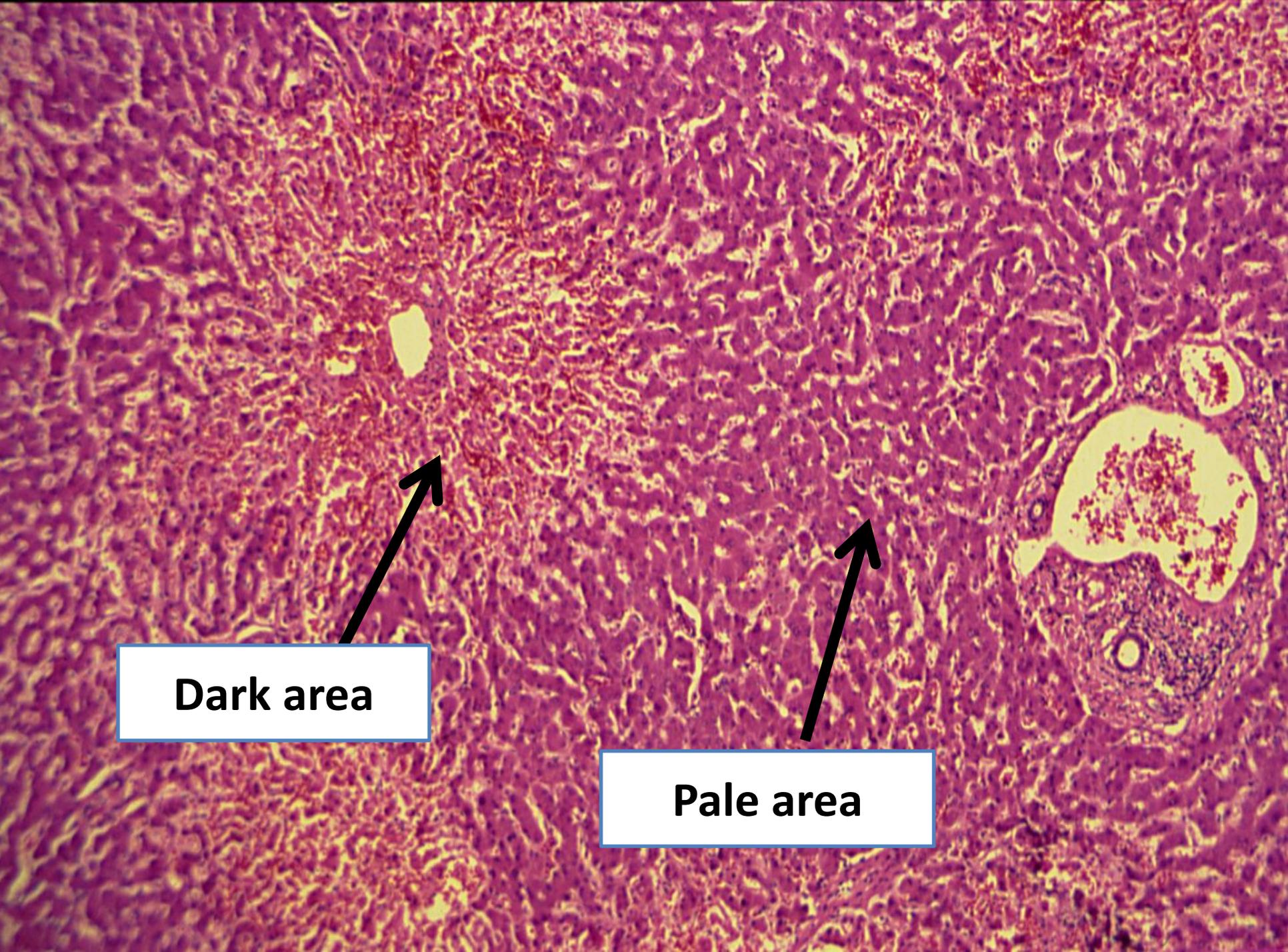


Gross name : **NUTMEG LIVER**

Two areas :

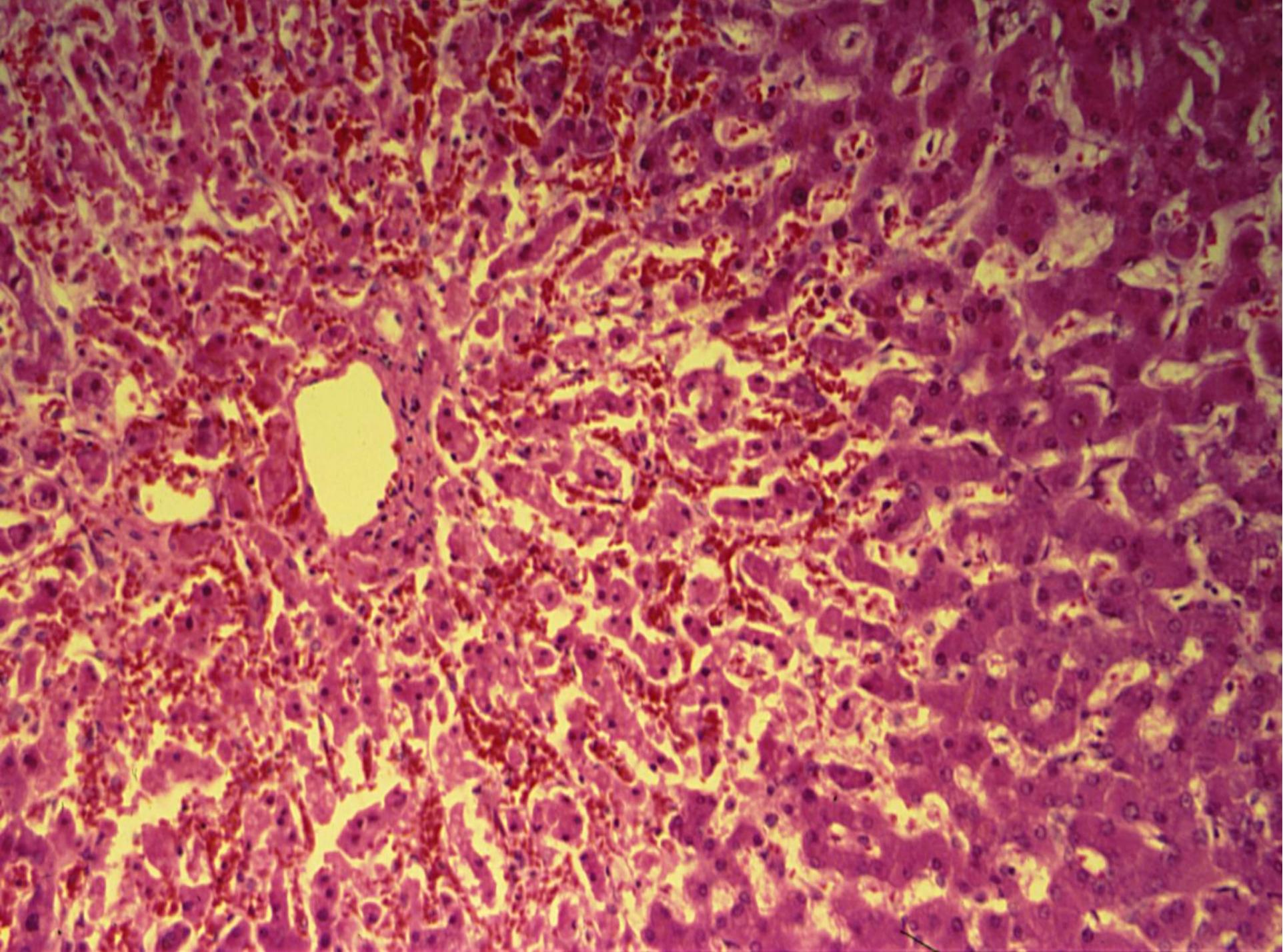
Dark : congested blood

Pale : non-congested blood



**Dark area**

**Pale area**



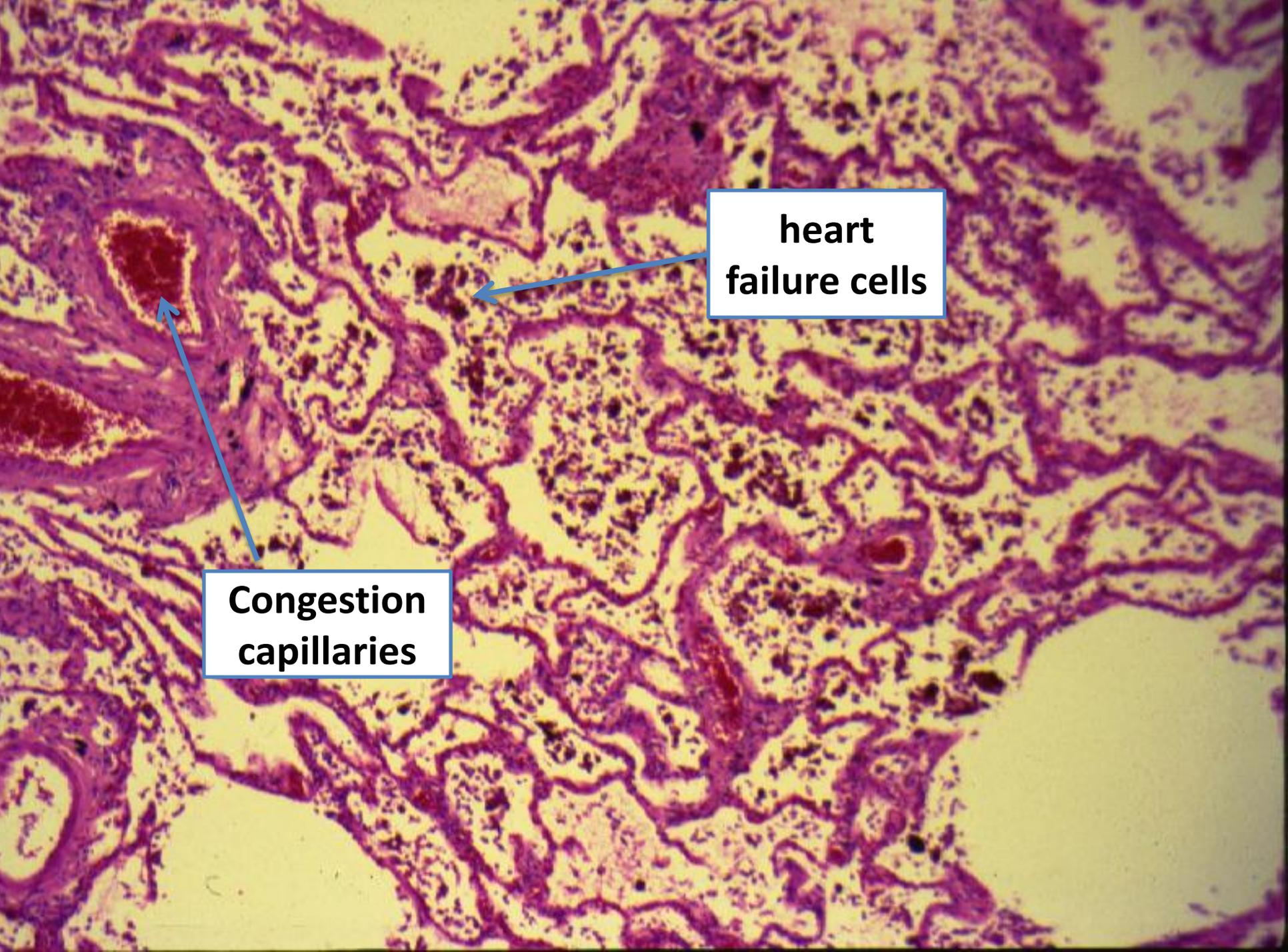
# ***Chronic venous congestion of the liver:***

## ***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.**

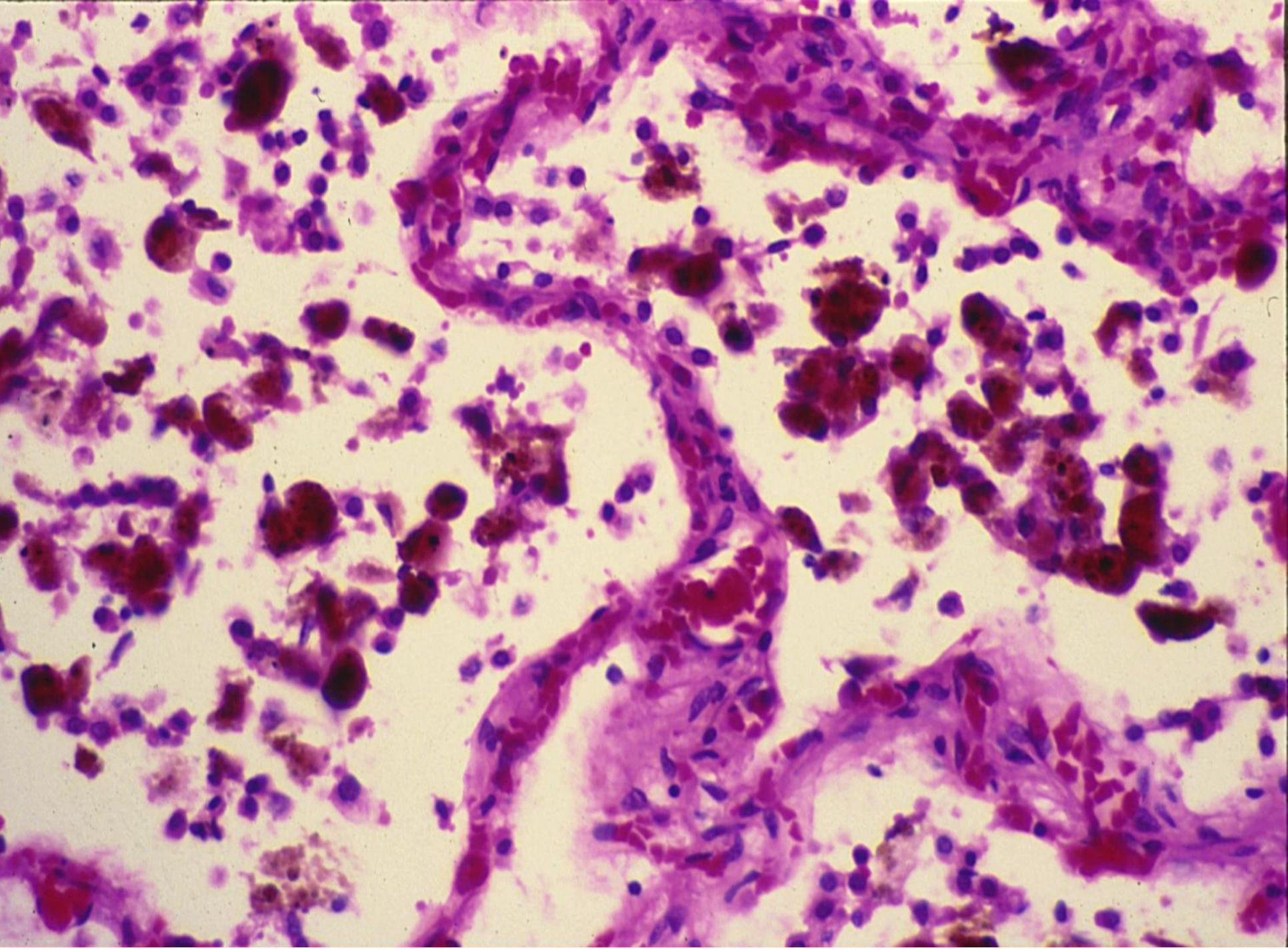
9-Chronic venous congestion of the  
lung

**“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

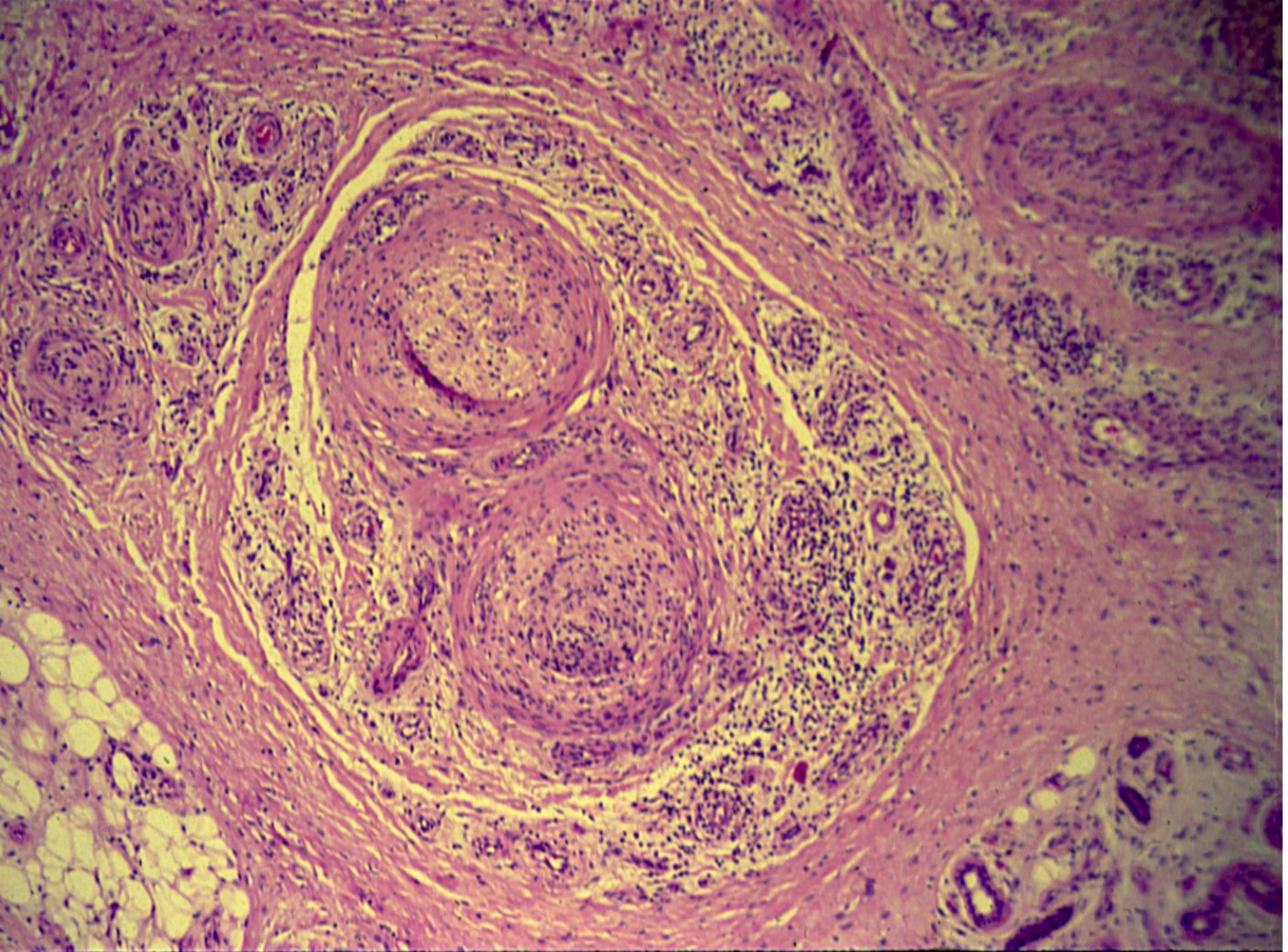
### 2- infectious

# 10-Thromboangitis obliterans (Buerger disease)

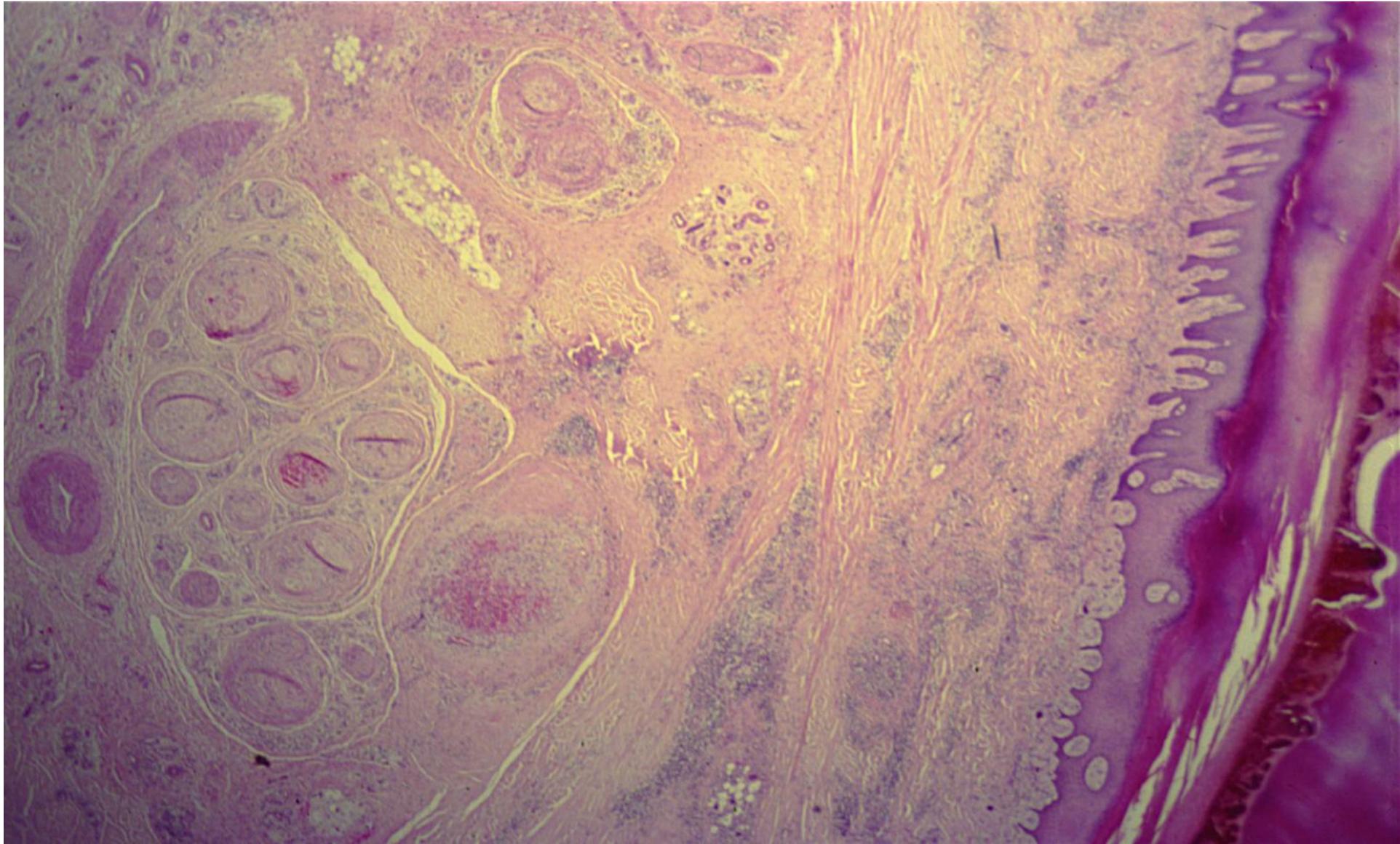


## **Thromboangiitis obliterans (Buerger disease)**

- **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

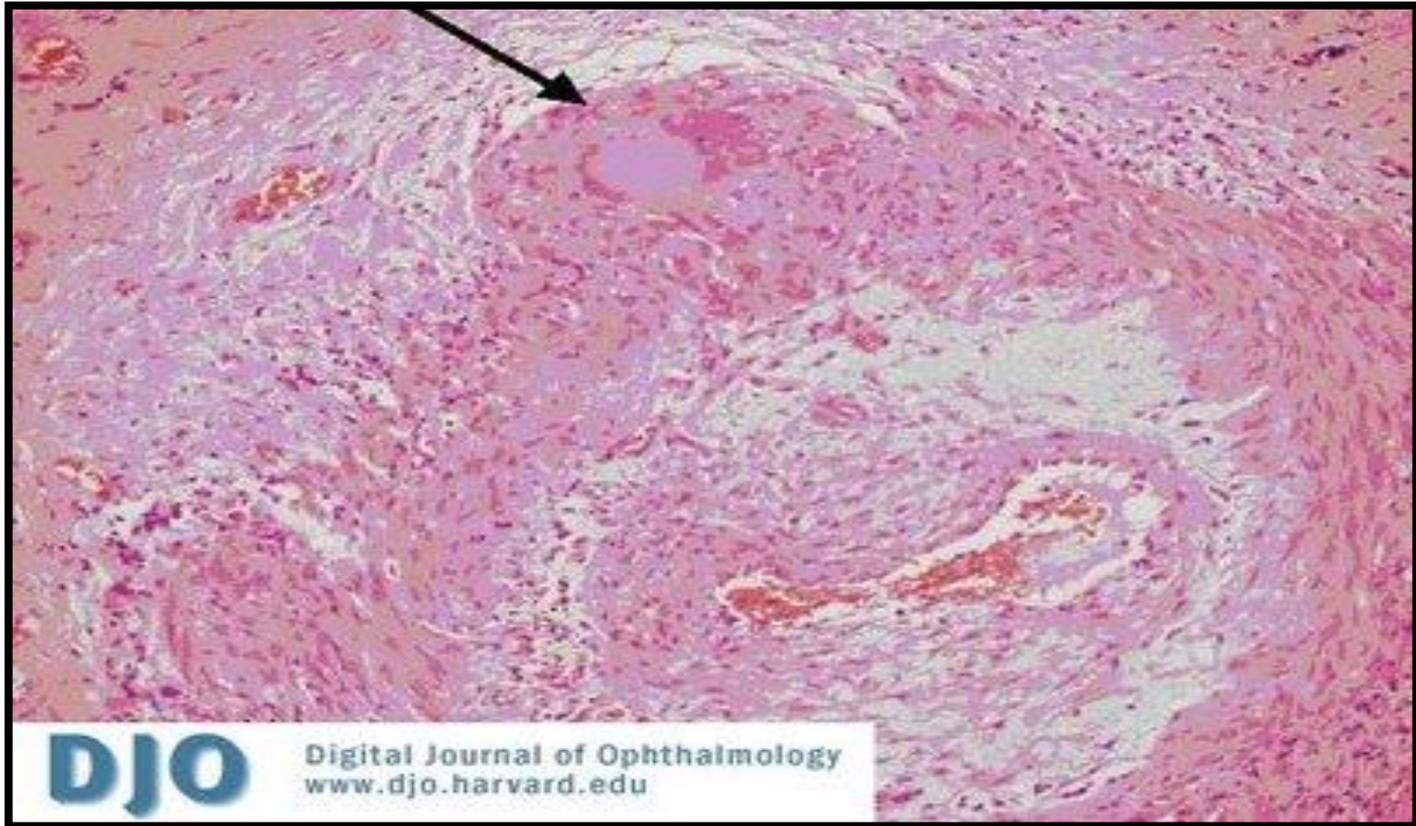
# Giant cell arteritis

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

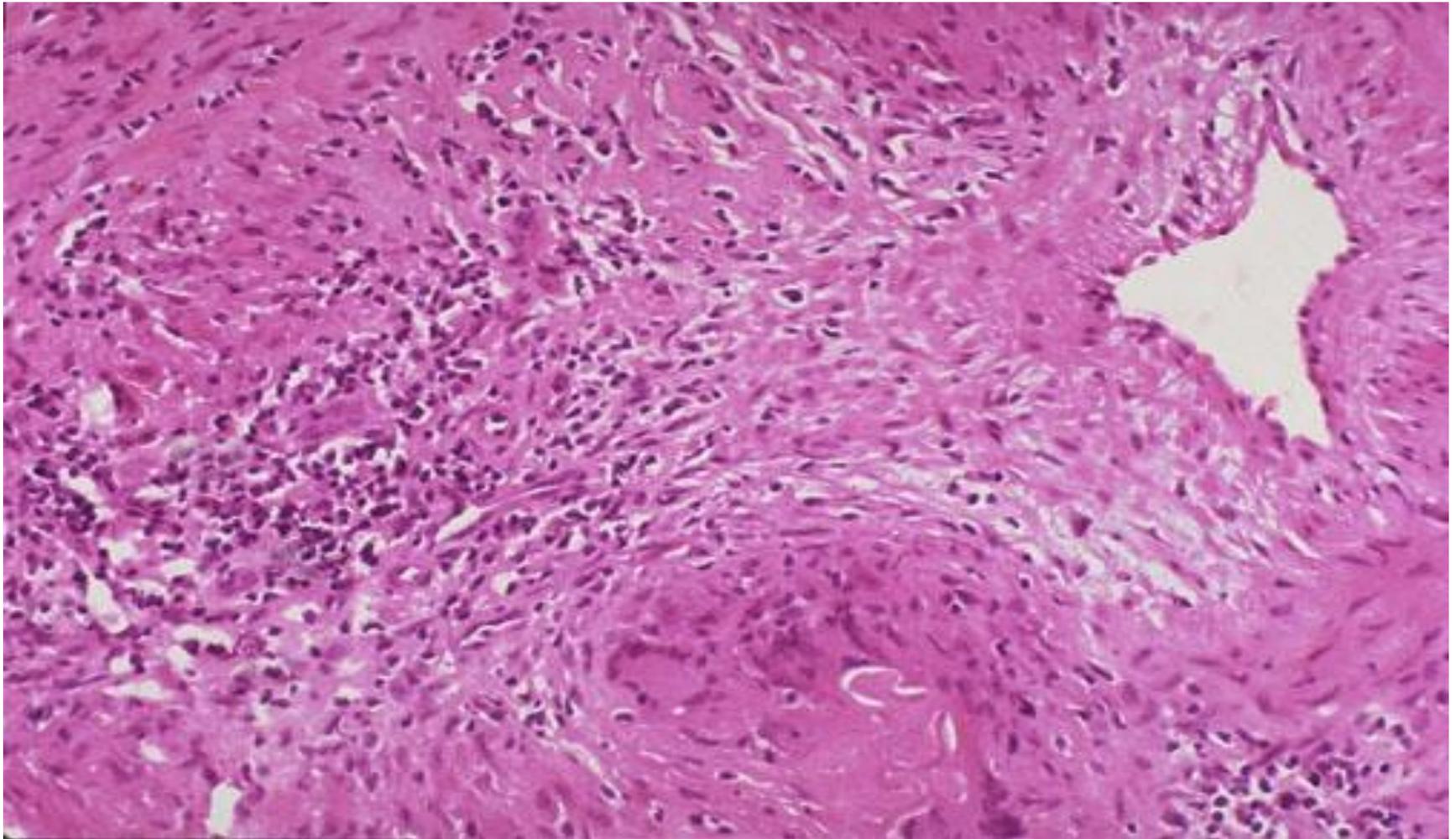


Tender and thickened scalp veins

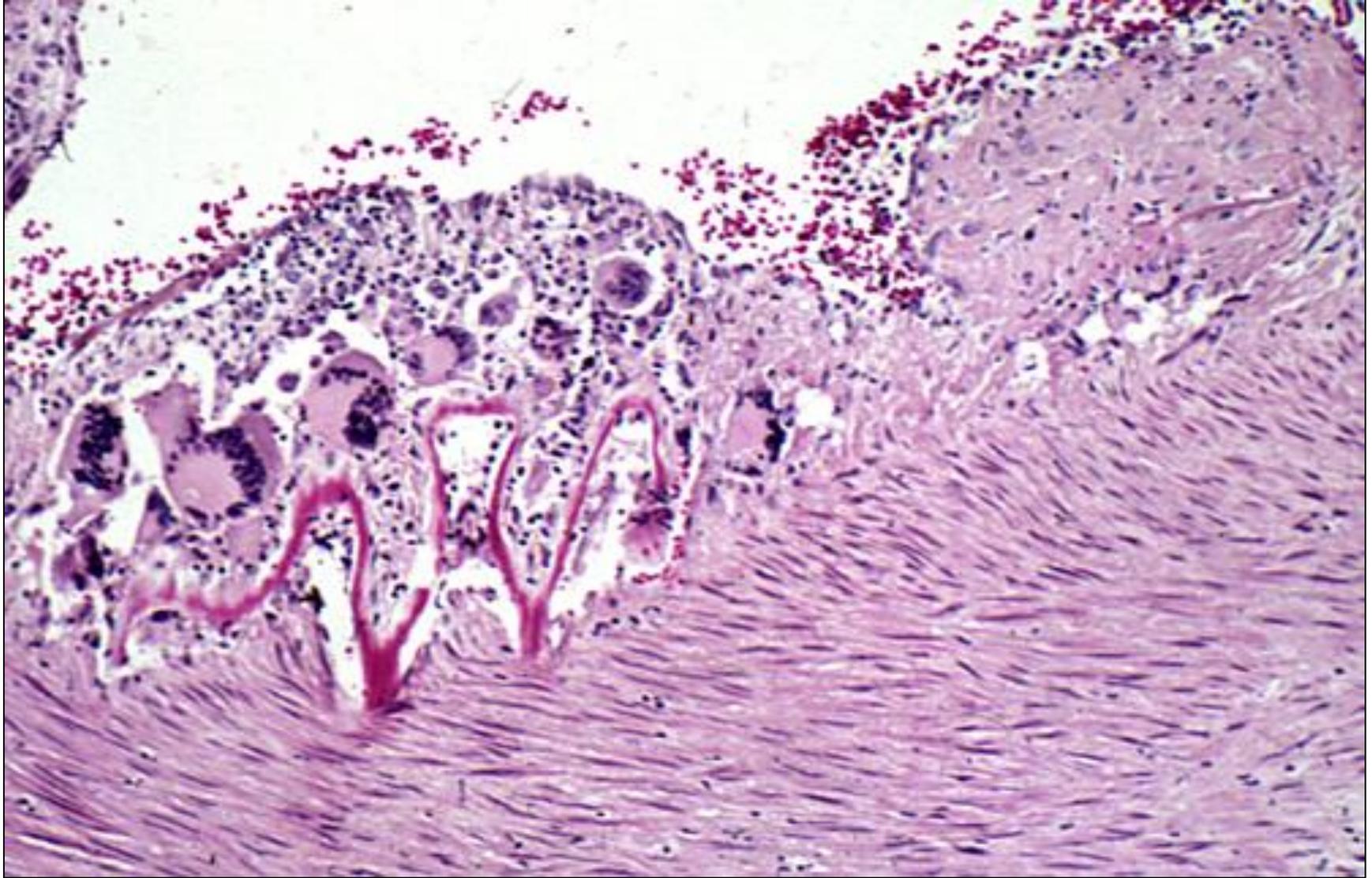
# Giant cell arteritis



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.**

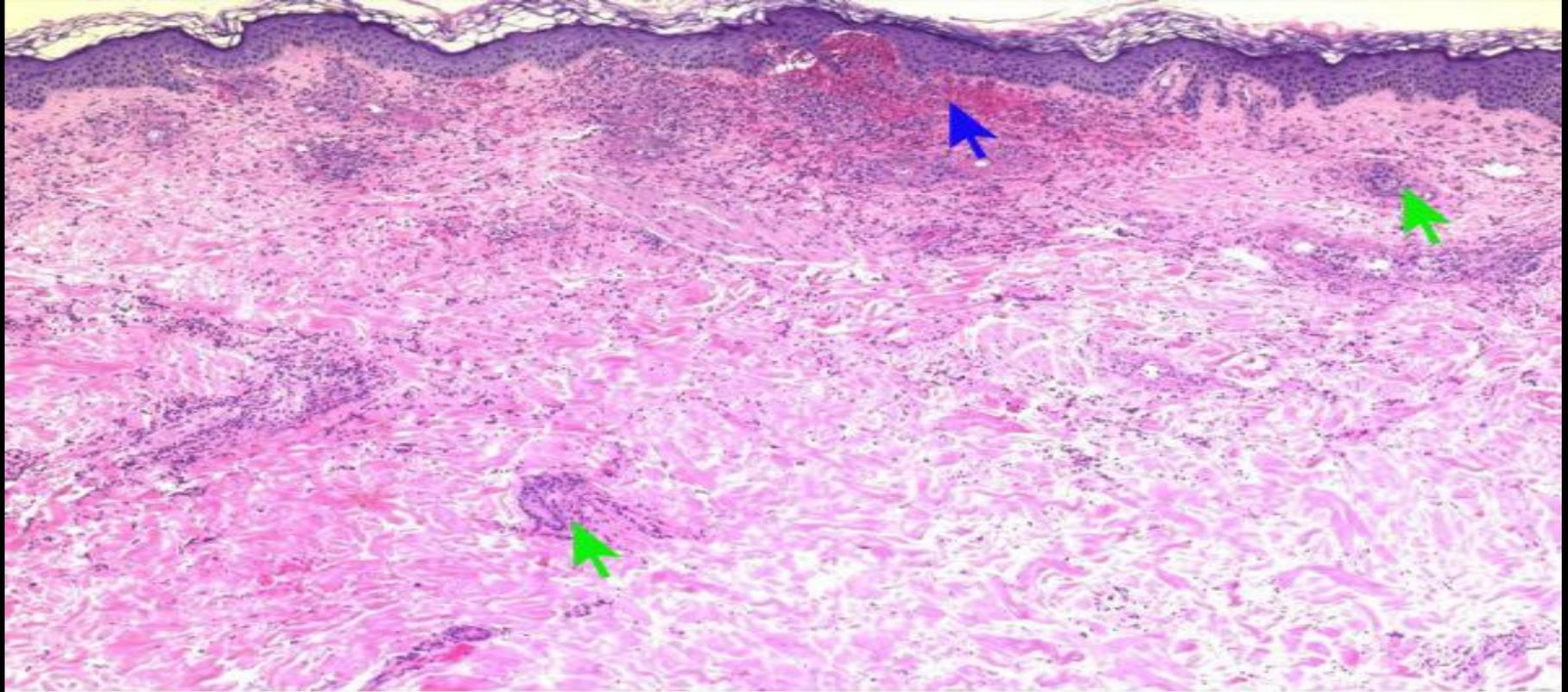


**Segmental inflammatory lesions with intimal thickening , medial granulomatous inflammation with giant cells and chronic inflammatory cells and internal elastic lamina fragmentation .**

# 12-Leukocytoclastic vasculitis

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

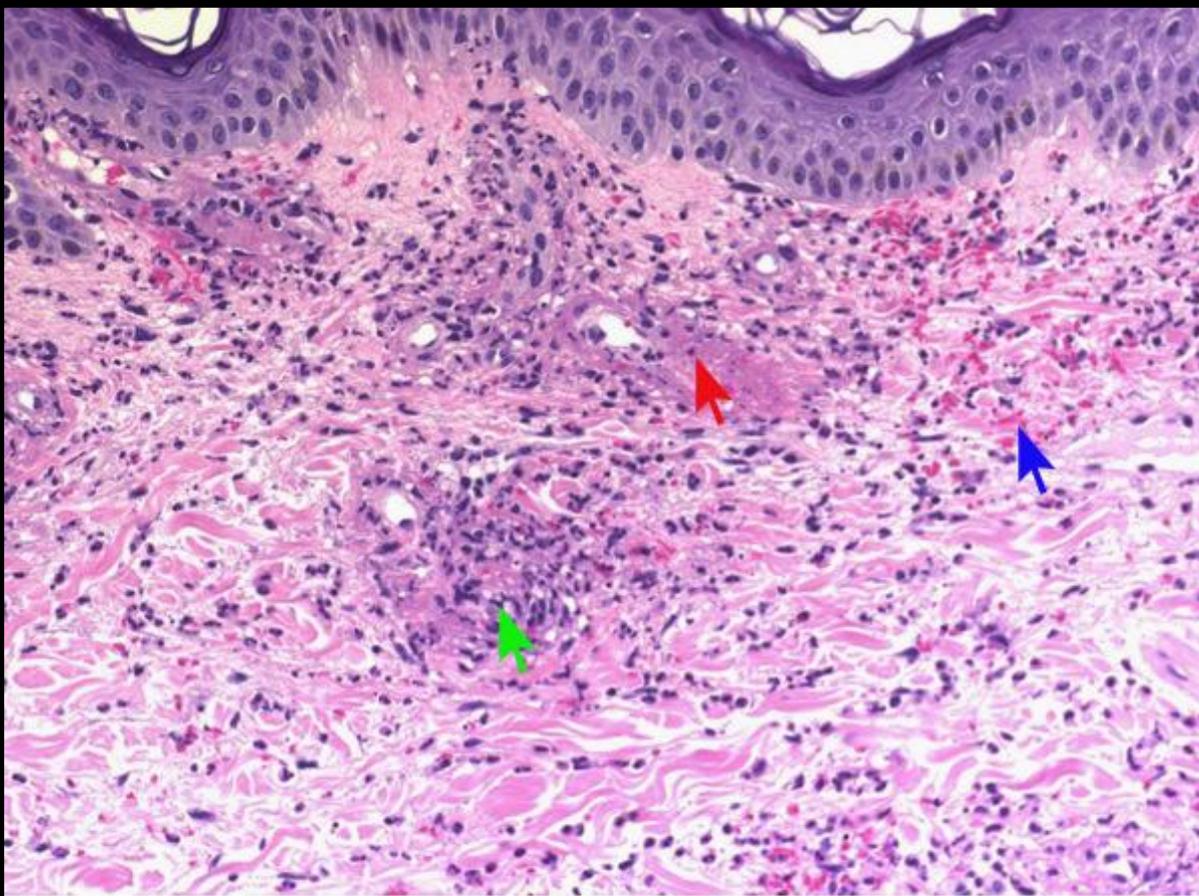




 **Red cell extravasation**

 **Inflammation**

**Histology: vasculitis, leukocytoclastic, low power**



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

**Histology: vasculitis, leukocytoclastic, high power**