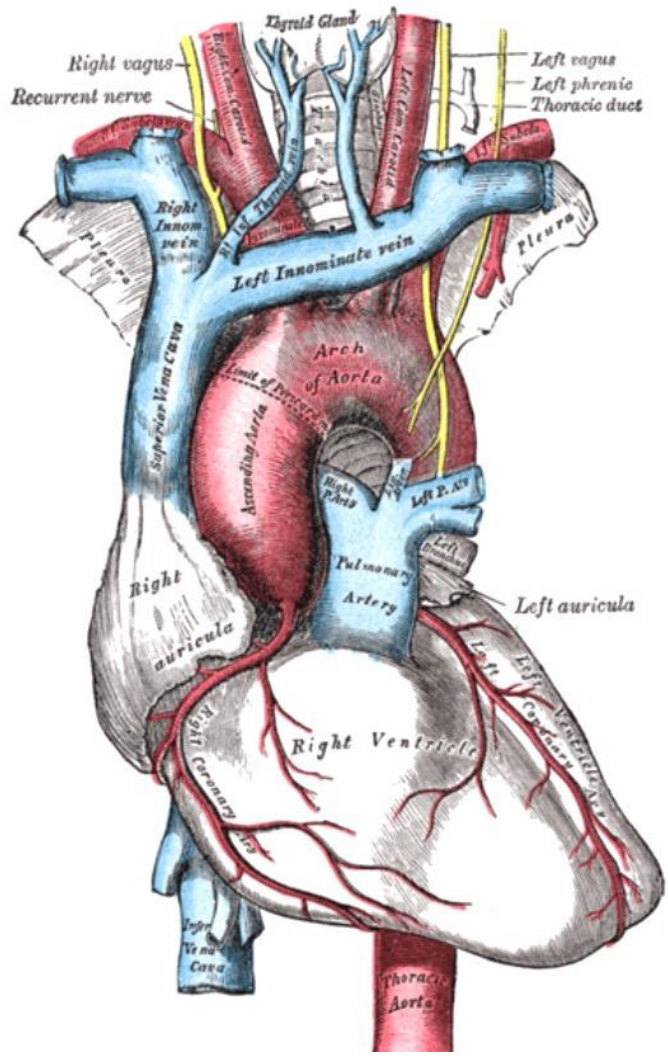
MED437
KING SAUD UNIVERSITY

PATHOLOGY
TEAM 437




# Pathology Practical

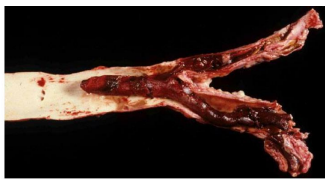
## CVS Block

**Pink: Only in female slides**
**Blue: Only in male slides**
**Grey: Notes**

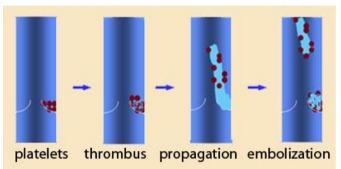
* ترتيب السلايدز في الفايل مطابقة لترتيب سلايدز البنات ...نظرًا لوجود اختلاف بين سلايدز البنات والعيال في الترتيب فقط

# Background information added by Dr. Sufia

## A- Thrombosis

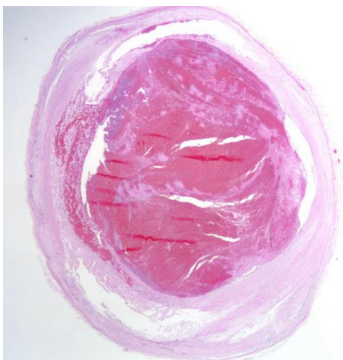


Mural thrombus.



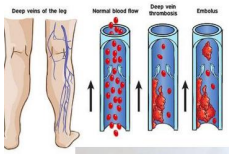

- Thrombosis is a process by which a thrombus is formed.
- A thrombus is a solid mass of blood constituents which develops in artery, vein or capillary.
- It is intravascular coagulation of blood and it can cause significant interruption to blood flow.
- Thrombi may develop anywhere in the cardiovascular 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.**

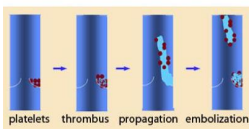
## B- Thrombus



- 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.

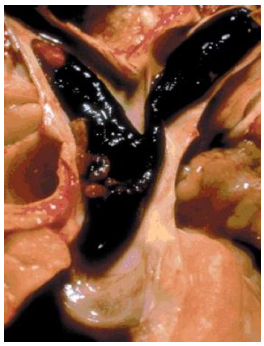
## C- Embolism





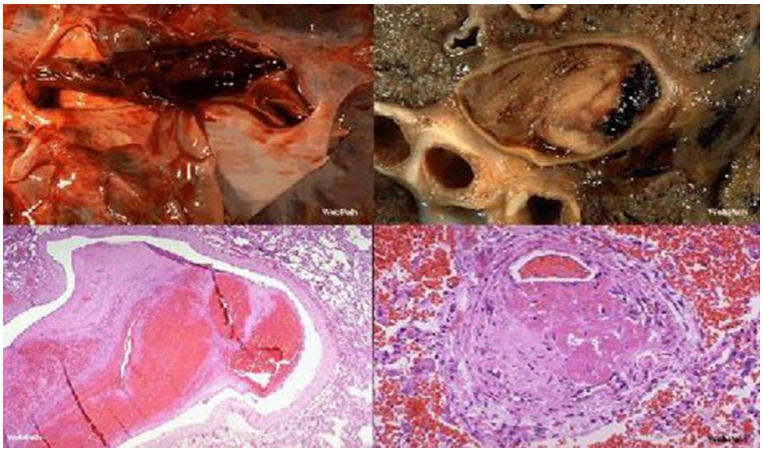

- An embolus is a detached intravascular solid, liquid, or gaseous mass that is carried by the blood to a site distant from its point of origin.
- Almost all emboli represent some part of a dislodged thrombus, hence the commonly used term thromboembolism.
- The emboli ultimately lodged in vessels too 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**.

# D- Pulmonary Thromboembolism

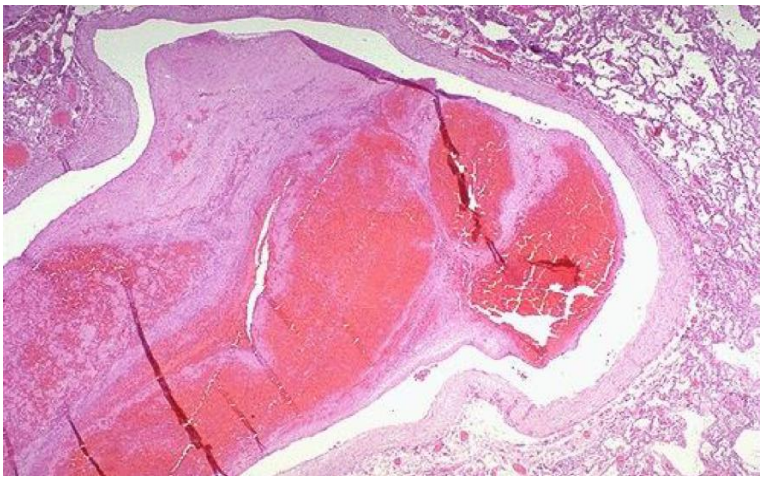


- Here the embolus get lodged in the pulmonary vasculature.
- Depending on size of embolus, it may get stuck and block the main pulmonary artery or block the bifurcation of the pulmonary trunk (saddle embolus) or pass out into the smaller, branching arterioles of the pulmonary circulation.
- Most pulmonary 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.

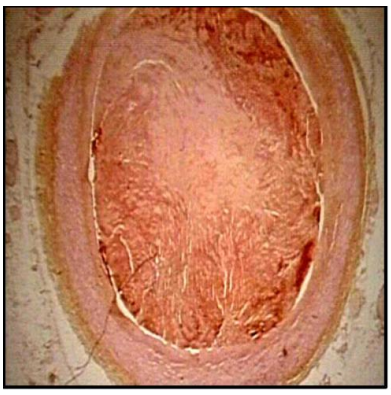
# E- Organizing Thrombus

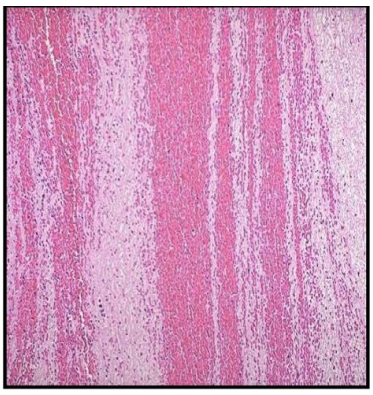


Organizing thrombus in a case of pulmonary embolism

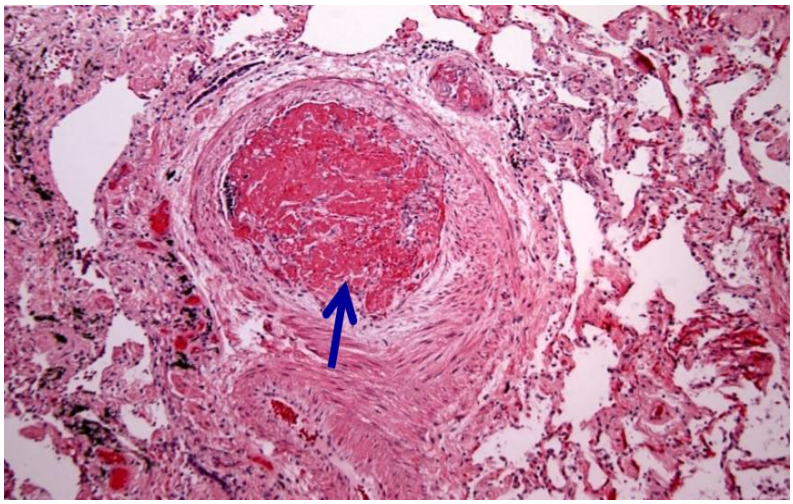


This is the microscopic appearance of a pulmonary thromboembolus in a large pulmonary artery.
There are interdigitating areas of pale pink and red that form the "lines of Zahn" characteristic for a thrombus.
These lines represent layers of red cells, platelets, and fibrin which are laid down in the vessel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



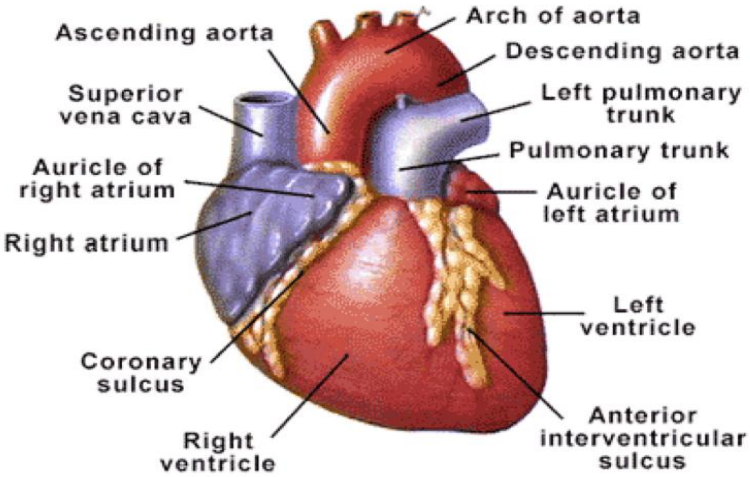


**Lines of Zahn**, gross and microscopic, is evidence to prove a clot is Pre-mortem which is different from the clots appearing like current jelly or chicken fat which are said to be Post-mortem.
These lines represent layers of red cells, platelets, and fibrin



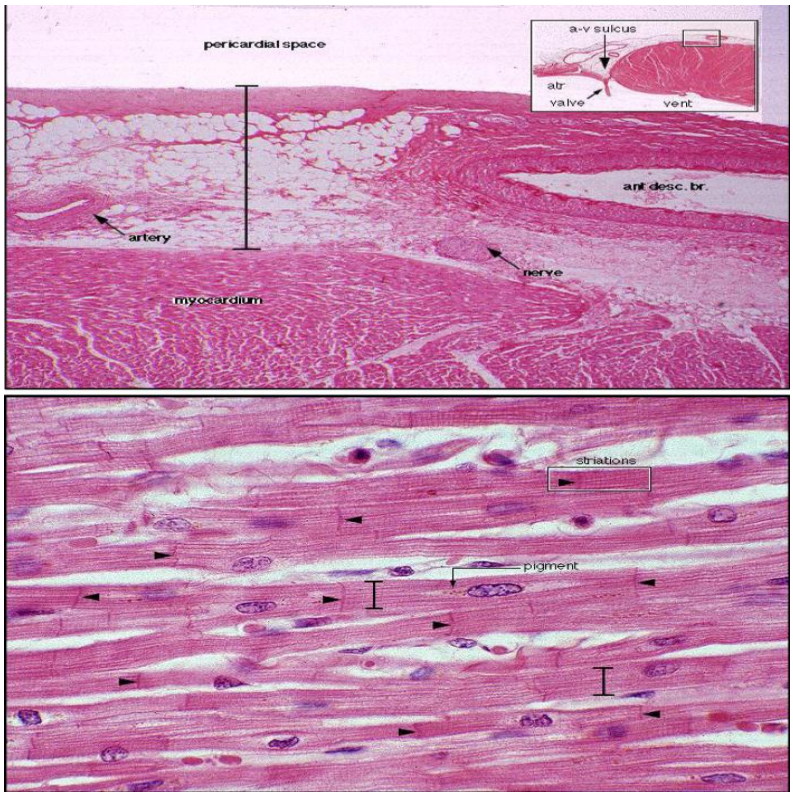
Pulmonary thromboembolus in a small pulmonary artery.
The interdigitating areas of pale pink and red within the organizing embolus form the “lines of Zahn” (arrow) characteristic of a thrombus.
These lines represent layers of red cells, platelets, and fibrin that are laid down in the vessel as the thrombus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

## F- 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

## G- Histology of the Heart



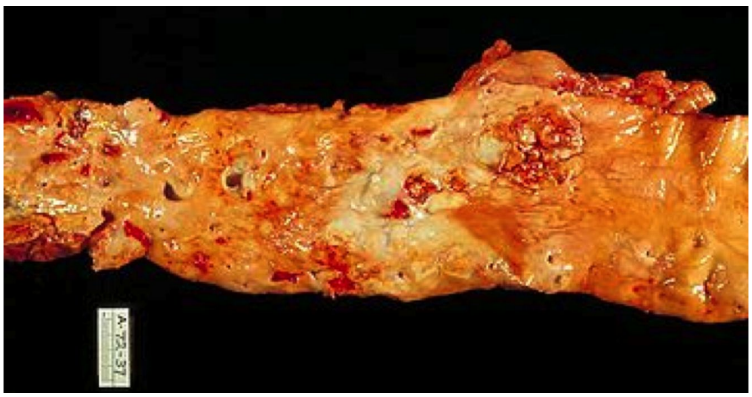

- The heart consists of 3 layers
  - the **Endocardium**
  - the **Myocardium**
  - the **Pericardium**.
- The **Pericardium** consists of arteries, veins, nerves, connective tissue, and variable amounts of fat.
- The **Myocardium** contains **branching, striated muscle cells with centrally located nuclei**. They are connected by **intercalated disks** (arrowheads).

# Case 1 : Atheroma of the aorta

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

- hyperlipidemia
- hypertension
- cigarette smoking
- diabetes

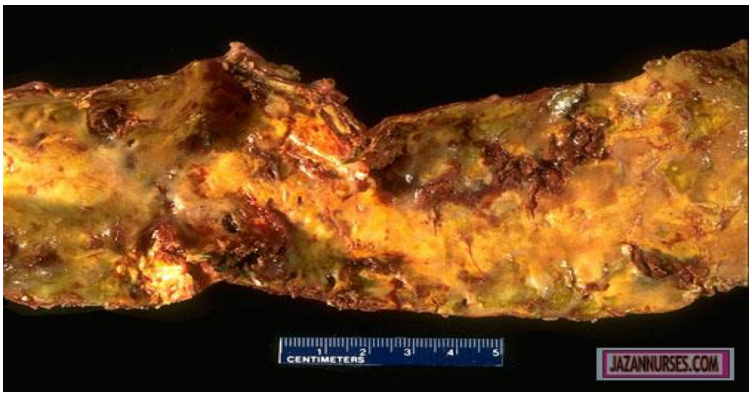
## Gross:



**Atherosclerosis aorta shows**:

- Yellow atheromatous plaque
- Area of ulceration and hemorrhage

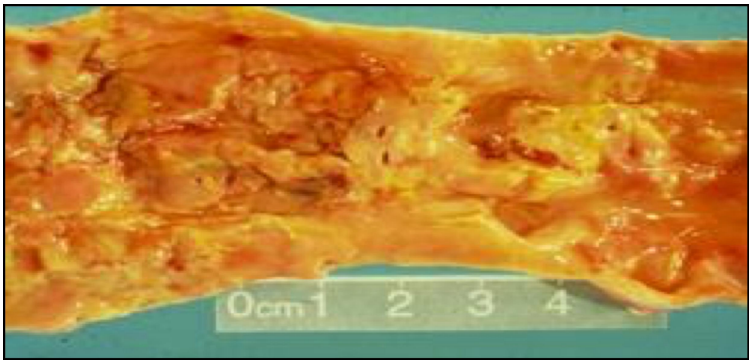
The key processes in atherosclerosis are **intimal thickening** and **lipid accumulation**




**Severe atherosclerosis aorta shows:**

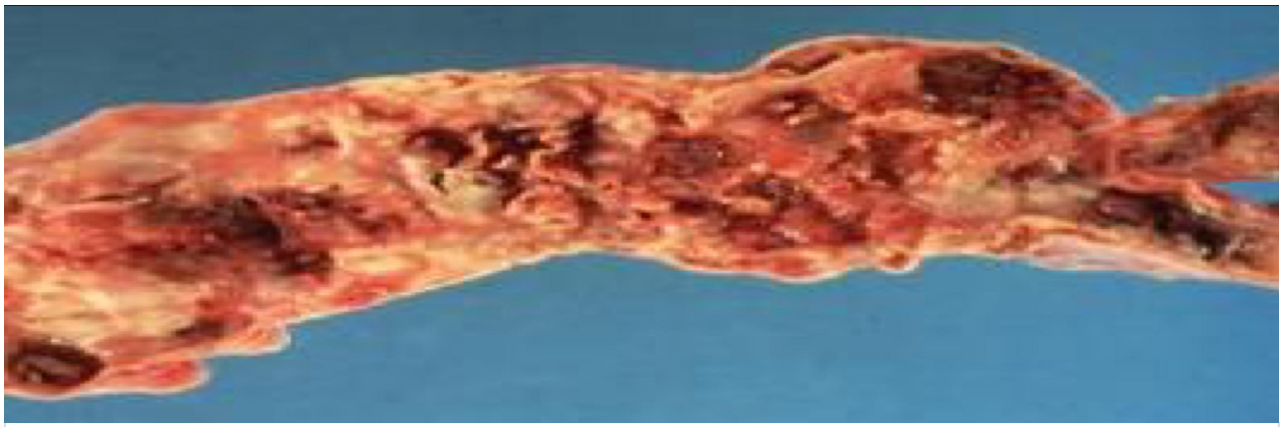
- ulceration in the atheromatous plaques
- formation of underlying mural thrombus

**Complications are:**
- Vascular thrombosis and distal embolization
- Aneurysm formation, hemorrhage , calcifications
- Cardiac ischemia and distal ischemic events



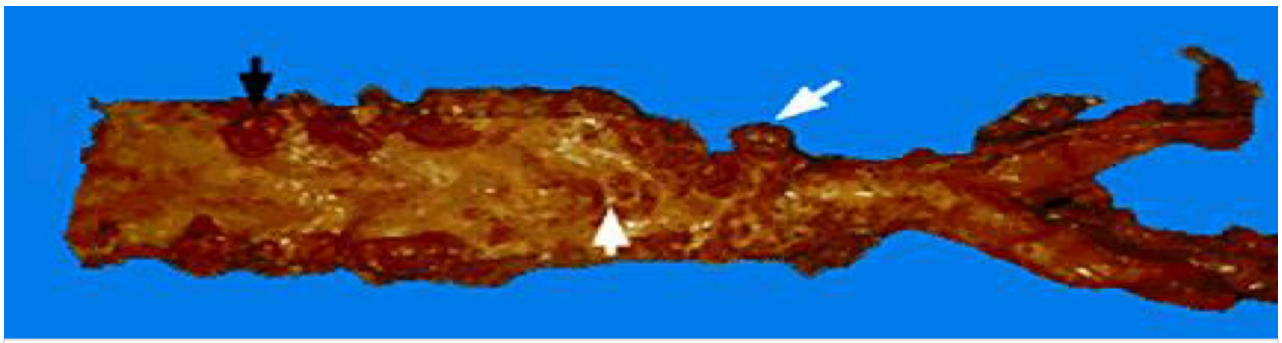

**Complicated atheromatous plaques shows:**

- Raised yellow plaques and the fissures in between the plaques
- Dystrophic calcification



**Complicated atheromatous plaques shows:**

- fissured-appearing endothelial surface
- raised plaque-like structures from the surface
- red clot material is adherent to the plaques in multiple areas
- the clots consist of platelets held together by fibrin strands

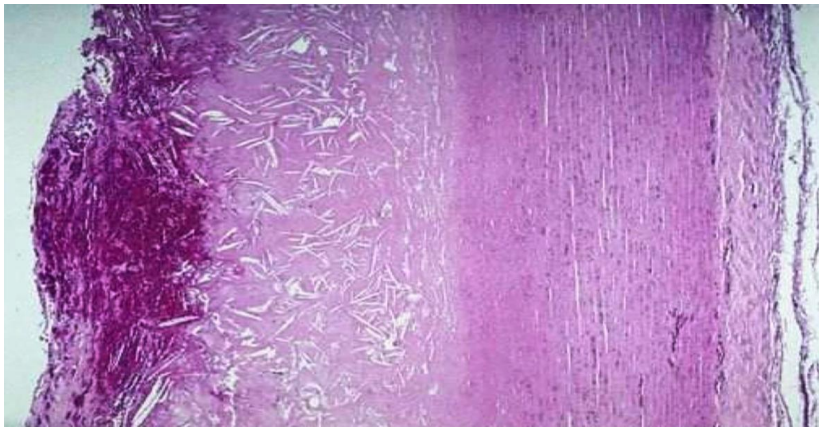


**Inner surface of aorta and bifurcation opened lengthwise along the posterior midline shows:**

- irregular variegated lining due to diffuse disease
- 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

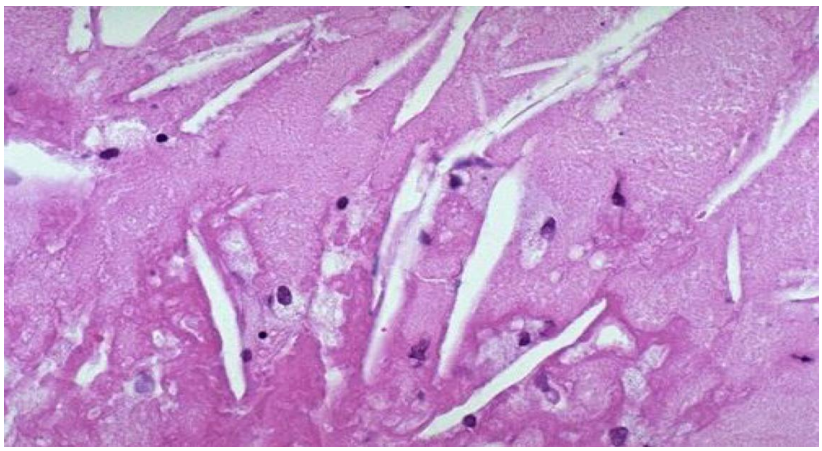

1- **mild atherosclerosis in aorta** shows only scattered lipid plaques. (Fatty streaks)
2- **moderate atherosclerosis in aorta** shows many more larger plaques.
3- **severe atherosclerosis in aorta** shows extensive ulceration in the plaques. (Yellow plaques)

# Histology - LPF



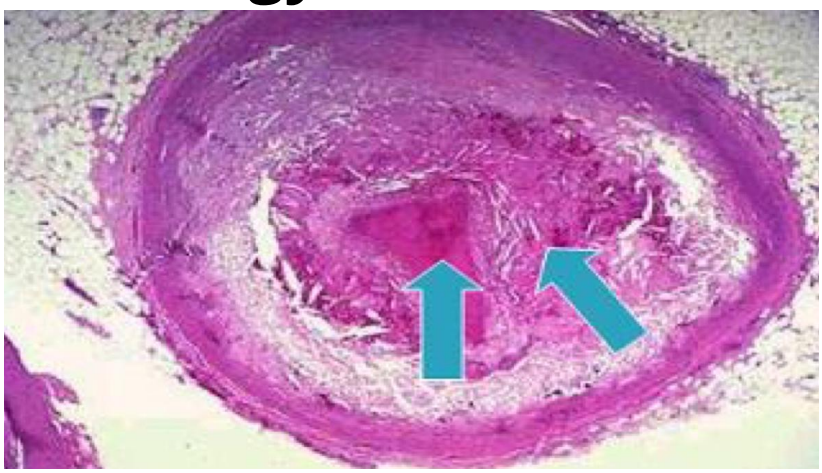
**Cross section of the aorta shows:**
- large overlying atheroma on the **left**
- numerous cholesterol clefts
- ulceration and hemorrhage on the **far left surface**

**Ulceration, atheromatous emboli are rare**



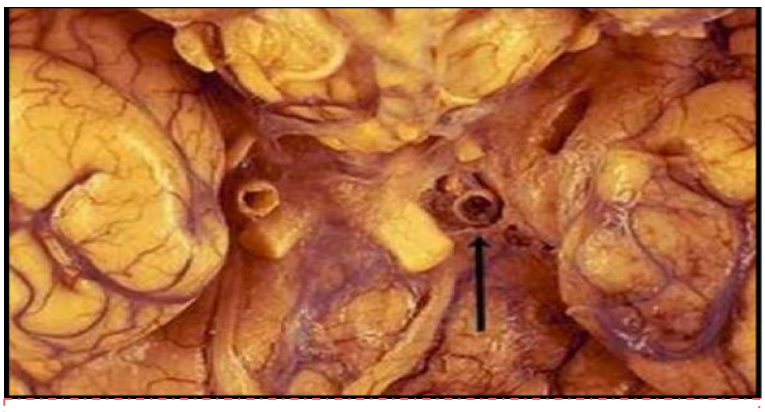
**A high magnification of the aortic atheroma shows:**
- foam cells (macrophages)
- cholesterol clefts

# Histology - MPF

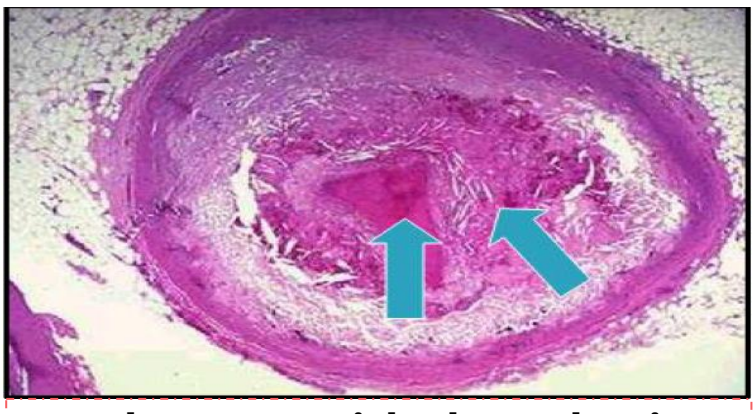


**Aortic Atheroma with Thrombosis**
**(Completely)**

This picture in male slides was with case 4: Thromboangiitis obliterans (Buerger's disease)



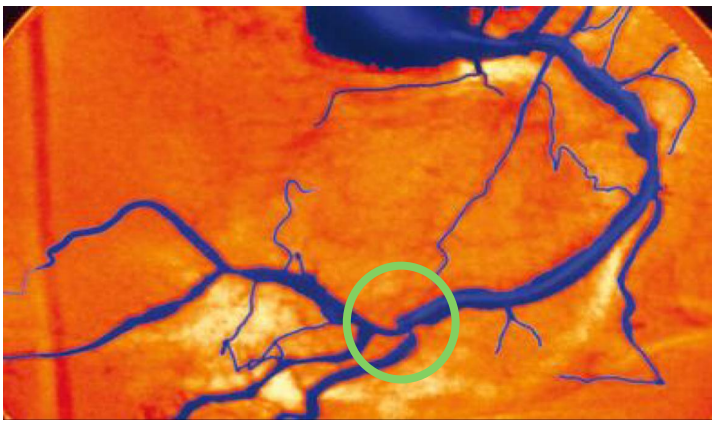
**Thromboembolism**



**Atheroma with thrombosis**

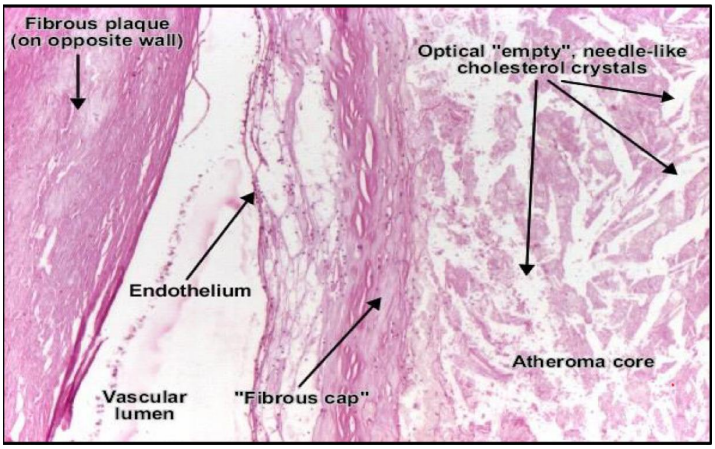
# Case 2 : Coronary Atherosclerosis (lead to MI)

## Gross:



**Coloured angiogram (X- ray) showing:**
- atherosclerosis in a coronary artery seen as the pinching in the blue-coloured artery at bottom centre (narrowed)

## Histology - LPF

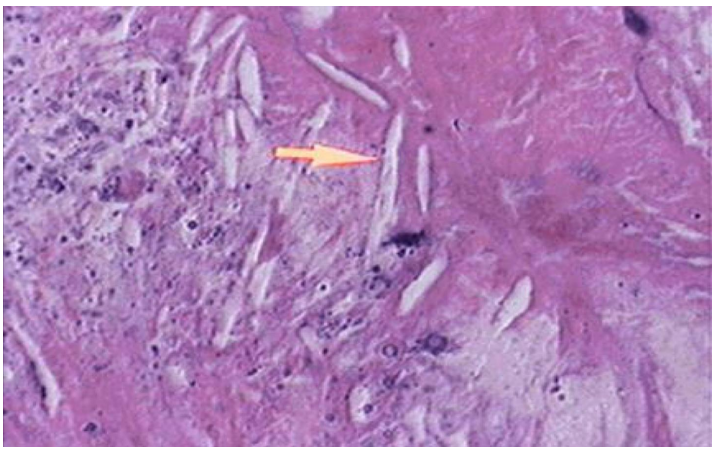



**The atheromatous fibro-fatty plaque is characterized by:**
- accumulation of lipids in the intima of the arteries.
- narrowing of the lumen.
- “fibrous cap” beneath the endothelium which covering the atheromatous “core” of the plaque.

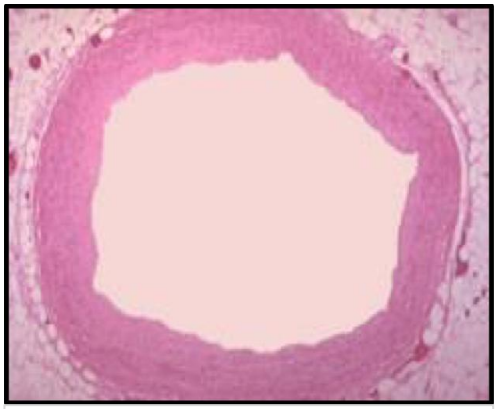
**Atherosclerotic plaques have three principal components:**
1- cells: smooth muscle cells,macrophages, T cells
2- ECM: collagen, elastic fibers, and proteoglycans
3- intracellular and extracellular lipid

## Aortic atherosclerosis -HPF

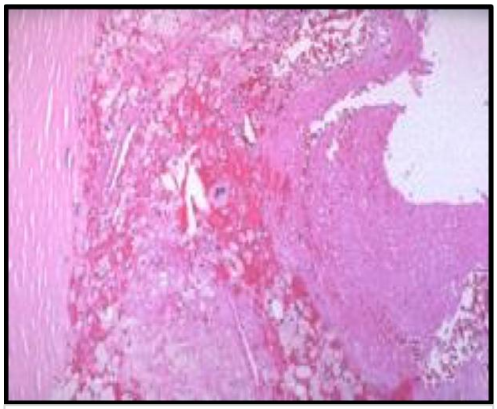
This picture in female slides was with case 1 : Atheroma of the aorta



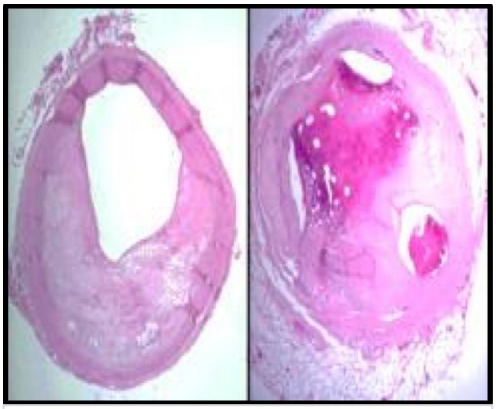
**intimal aspect of atherosclerotic plaque showing:**
- blue calcific spherules
- cholesterol crystal clefts
- fibrous cap



- **A normal coronary artery without atherosclerosis**
- a widely patent lumen that can carry as much blood as the myocardium requires.

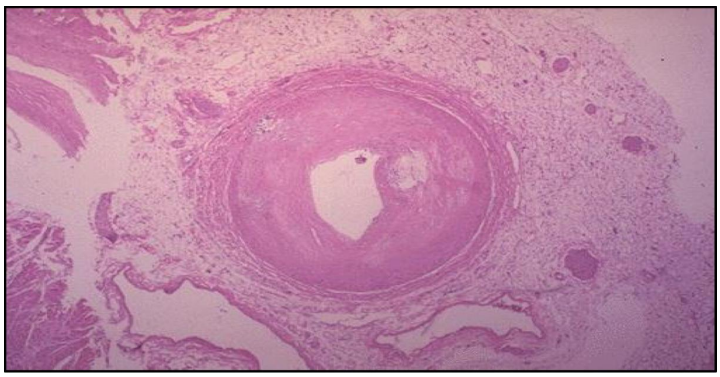


**Atheromatous plaque in a coronary artery shows:**
- endothelial denudation
- disruption and overlying thrombus formation **at the right**
- The arterial media is **at the left**



- **Occlusive coronary atherosclerosis**
- the coronary at the **left** if narrowed by (60-70%)
- The coronary at the **right** is even worse with evidence for previous thrombosis with organization of the thrombus

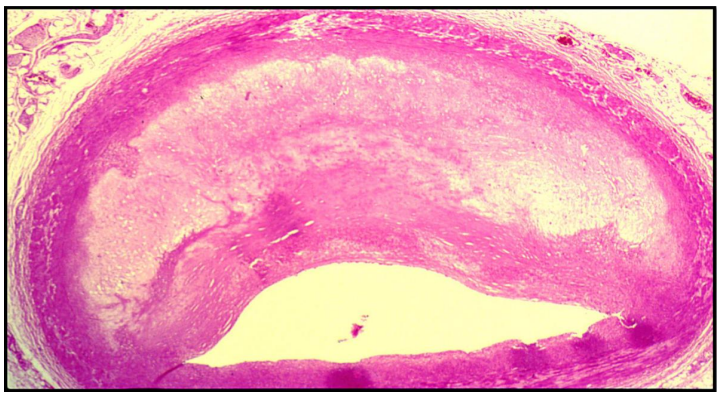
# Histology - MPF



**Distal portion of coronary artery shows:**
- significant narrowing

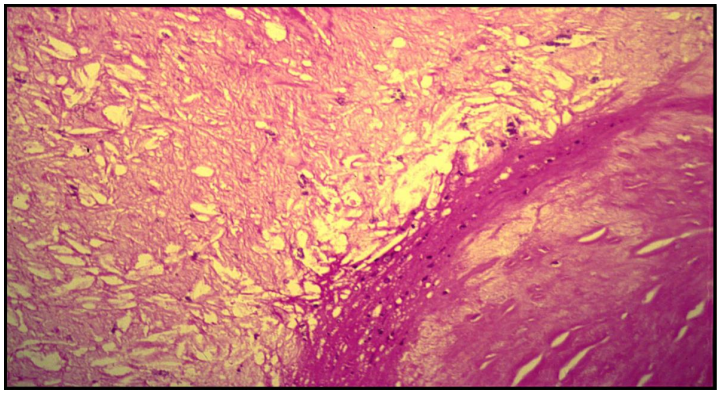
**Distal involvement is typical of severe coronary atherosclerosis.**

**It can appear with:**
- Diabetes mellitus
- Familial hypercholesterolemia



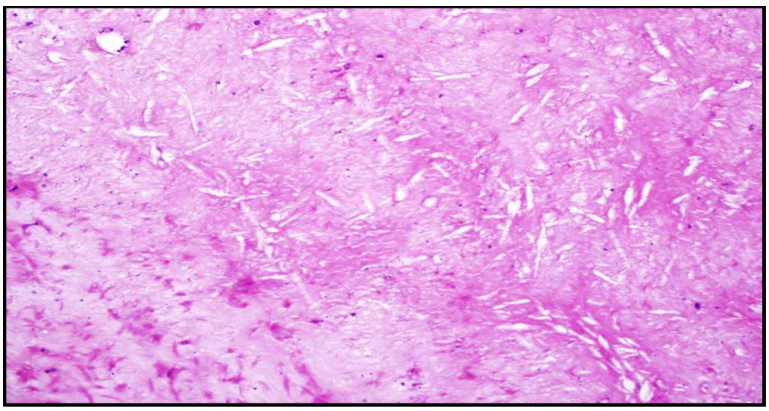
**Severe coronary atherosclerosis with narrowing of the lumen**

# Histology - HPF



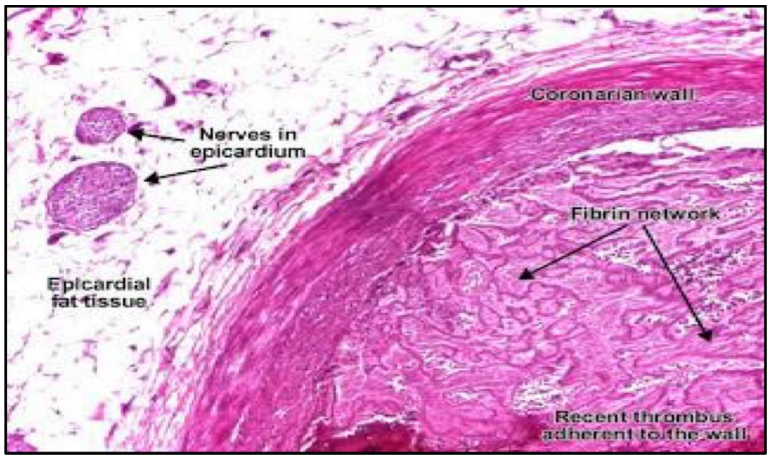
**Partial occlusion of the lumen by an atheromatous plaque.**

**The plaque consists of:**
- cholesterol clefts (extracellular cholesterol)
- hyaline fibrous tissue
- some blood capillaries



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

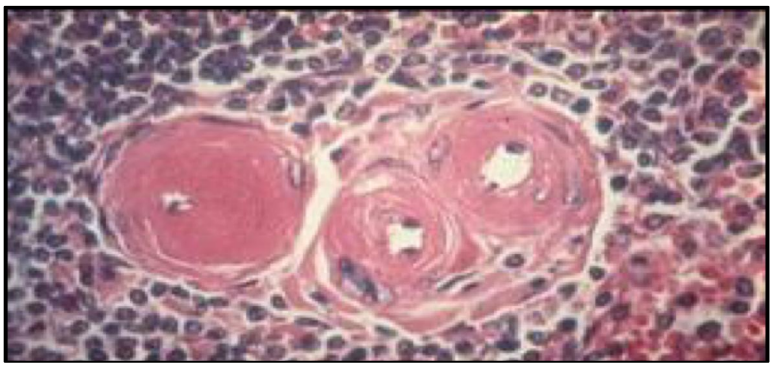
## - Recent thrombus in a Coronary artery




**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"

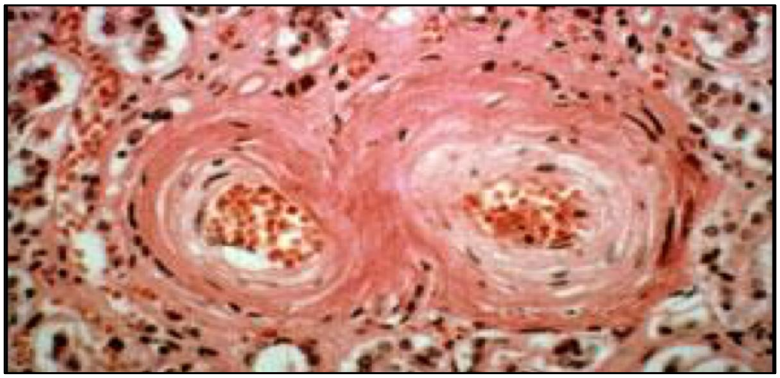


**Arteriosclerosis (hardening of the arteries) involves both small and large vessels shows:**
- homogeneous, pink hyaline thickening
- associated luminal narrowing

**commonly found in diabetics and hypertensive.**

## - Hyperplastic arteriolosclerosis -HPF

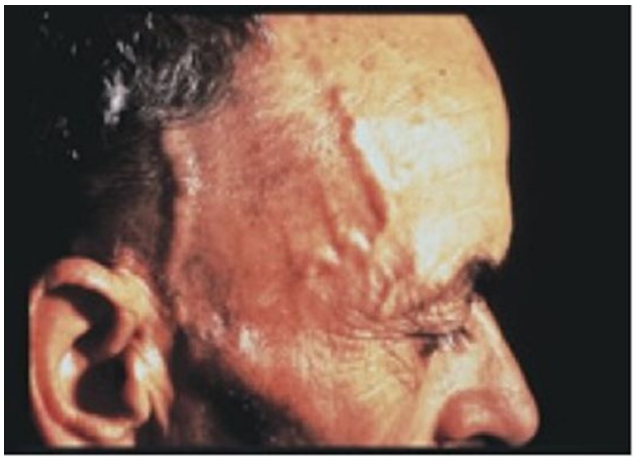
This is the other type of small vessel arteriosclerosis. It is predominantly seen in **malignant hypertension** and **renal disease** associated with **polyarteritis nodosa** and **progressive systemic sclerosis**.



**Hyperplastic arteriolosclerosis, The vessels exhibit "onion-skin lesions" characterized by:**
- concentric, laminated thickening of the walls
- luminal narrowing

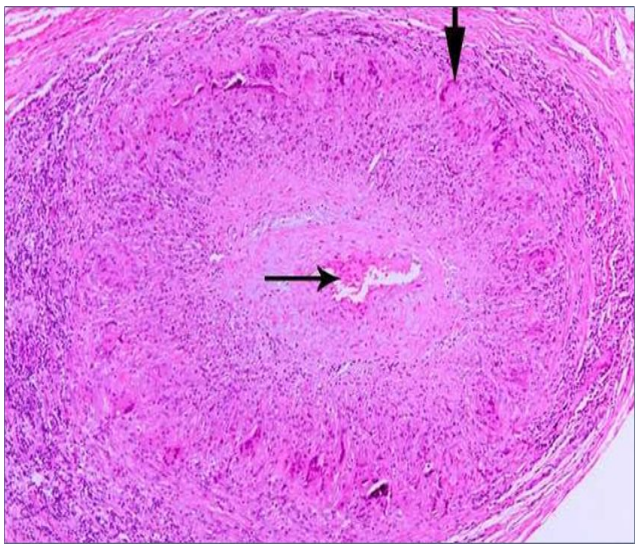
# Case 3: GIANT CELL (TEMPORAL) ARTERITIS

## Gross:



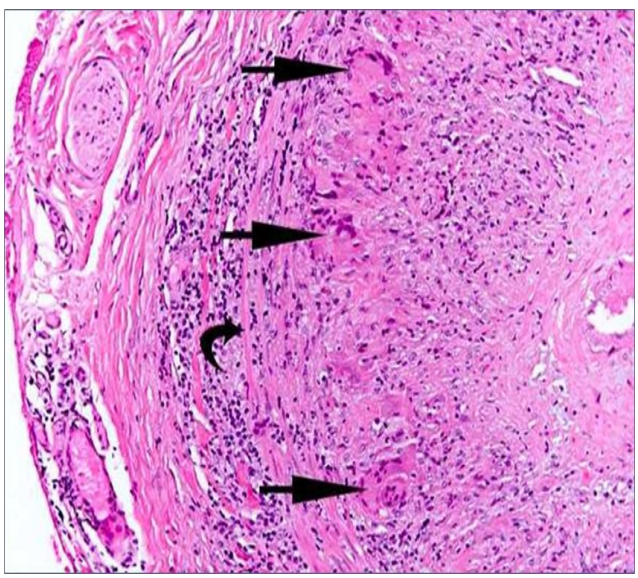
**Tender and thickened temporal artery**

## Histology - LPF



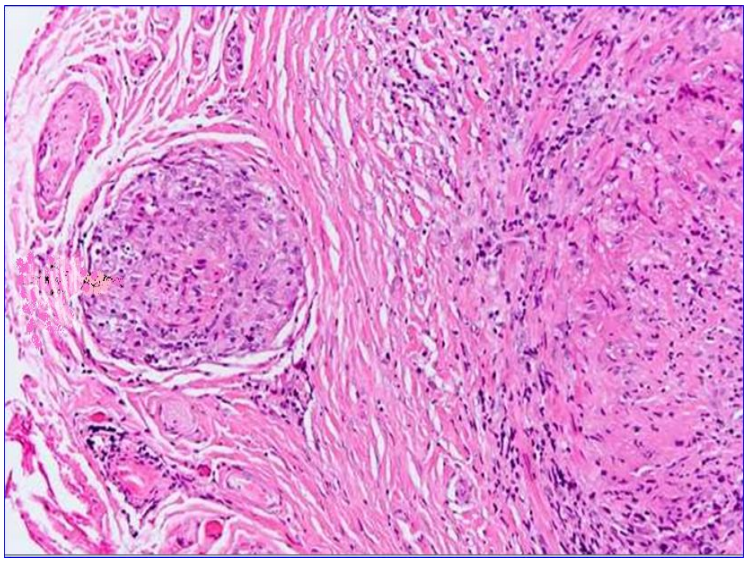
- Circumferential involvement of the vascular media is present (vertical arrow pointing downward).
- Presence of chronic lymphocytic inflammation in the media and adventitia.
- Reactive intimal fibroplasias lead to luminal stenosis with <10% of its original luminal diameter (thin arrow in the center).

## Histology - HPF



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

# Histology - HPF

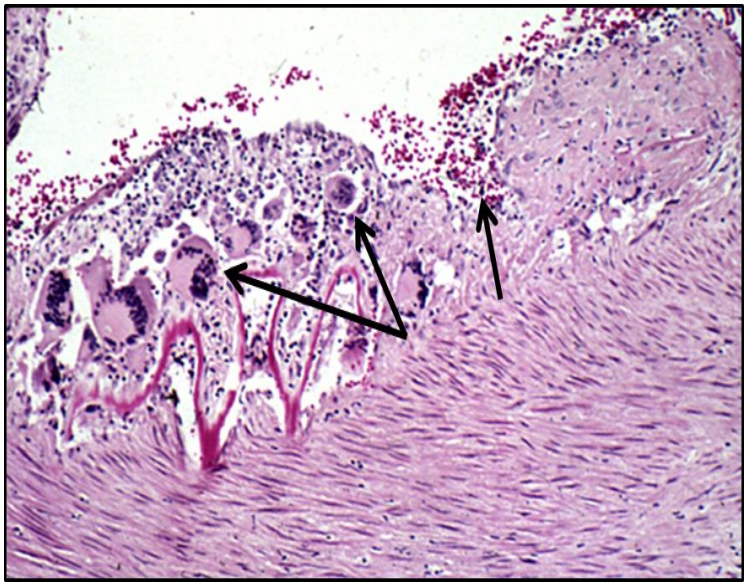


**The inflammation can be granulomatous in addition to both acute and chronic inflammatory cells.**

**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.



**-Disruptions of the elastic lamina with inflammation and giant cells.**

- Segmental inflammatory lesions with intimal thickening .

- medial granulomatous inflammation with :

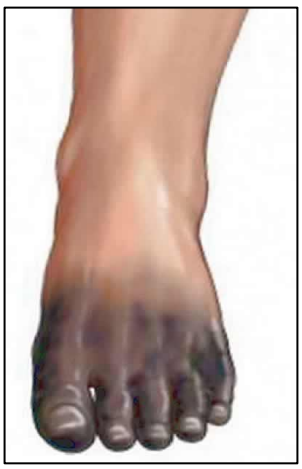
1. giant cells
2. chronic inflammatory cells
3. internal elastic lamina fragmentation

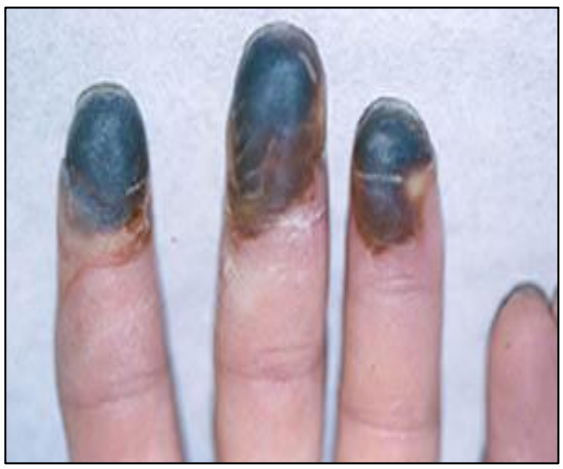
## case 4: Thromboangiitis obliterans (Buerger's disease)

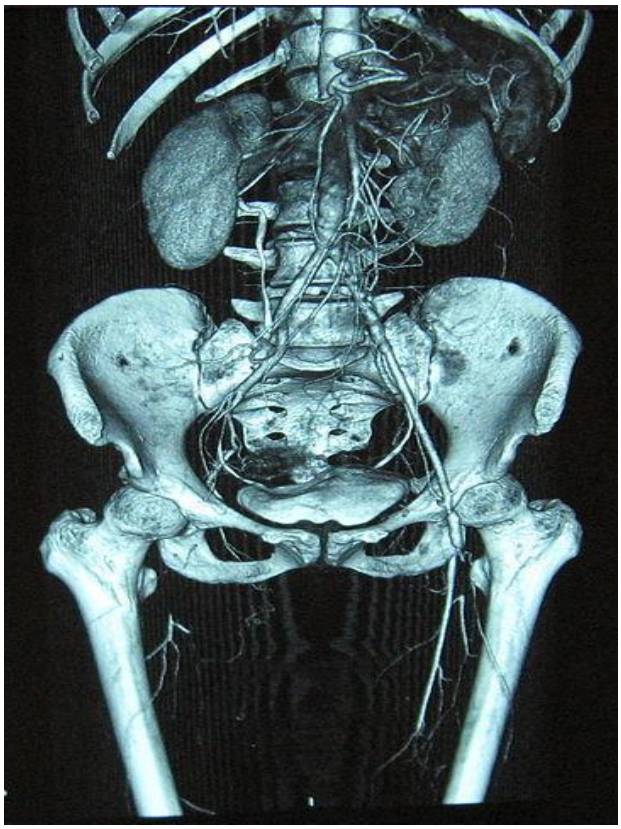
*find extra info on this subject in the practical file, page 48

**Pathologic findings of an acute inflammation** and **thrombosis** (clotting) of arteries and veins of the hands and feet (the **lower limbs** being **more common**)

## Gross:





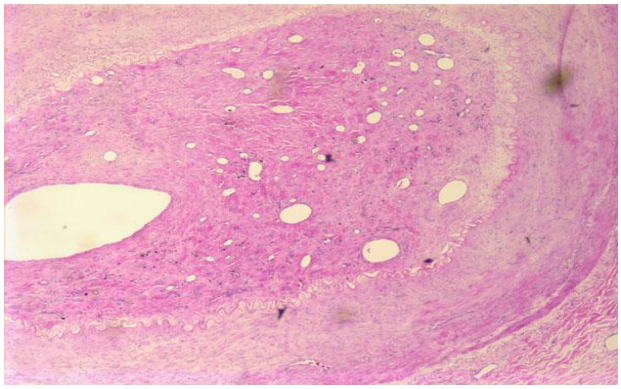


**(Angiogram)**
**Thromboangiitis obliterans , showing:**
-Complete occlusion* of the right femoral artery

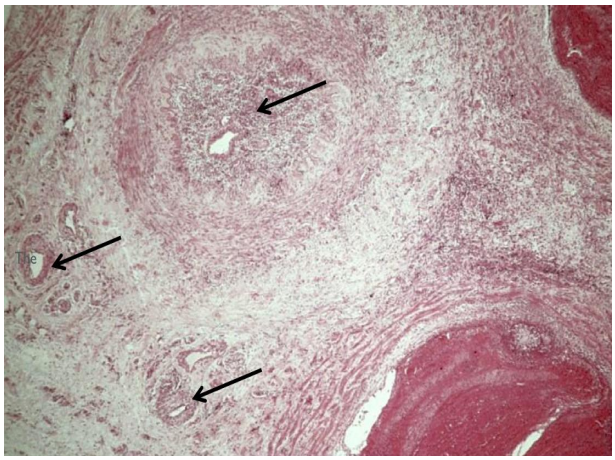
-stenosis of the left femoral artery.

*occlusion : obstruction

# Histology - LPF

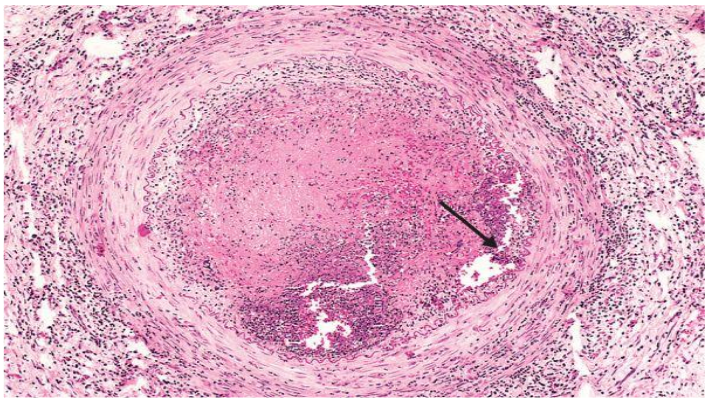


**Thromboangiitis obliterans (Buerger's disease)** :
is a **non atherosclerotic**, segmental, inflammatory, vaso-occlusive disease that affects the **small- and medium-sized arteries and veins** of the upper and lower extremities.



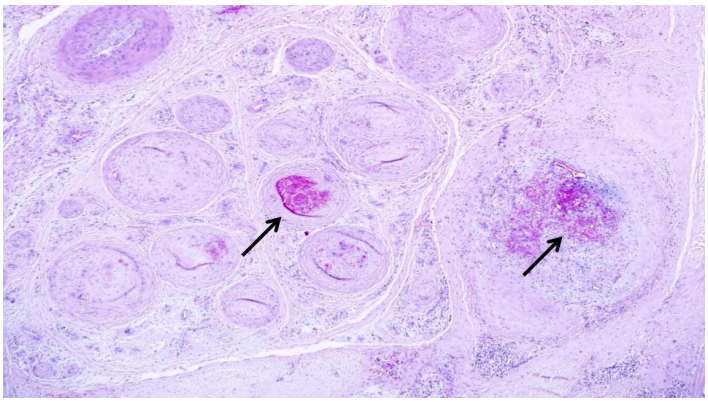
**Large number of small blood vessels in the dermis showing:**
- occlusive organized thrombi
- recanalization and fibrosis around blood vessels.

# Histology -HPF



**Thromboangiitis obliterans (Buerger disease),showing:**

-The lumen is occluded by a thrombus containing abscesses (arrow)
-the vessel wall is infiltrated with leukocytes.

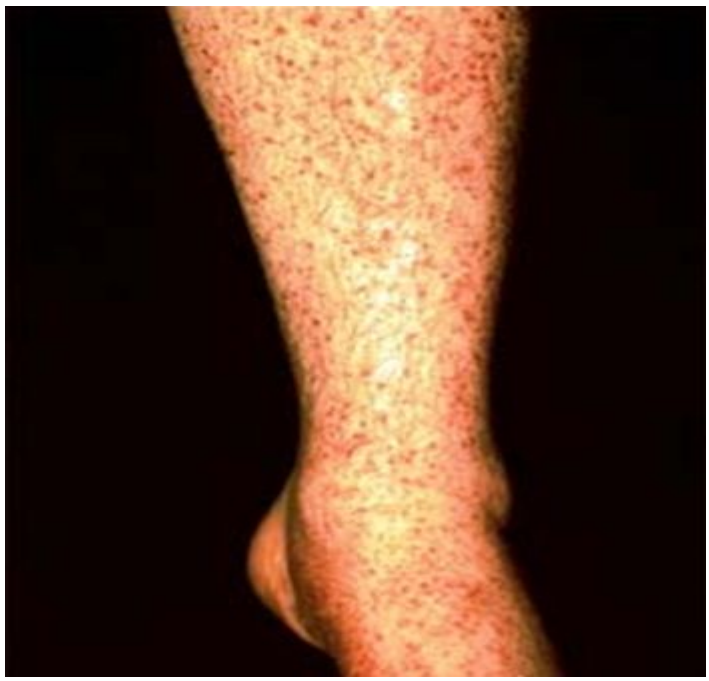


**Some blood vessels showing:**
-recent organizing thrombi.
**while other blood vessels show:**
-infiltration of the wall and surrounding tissue by chronic inflammatory cells

## Case 5: Leukocytoclastic/hypersensitivity vasculitis (microscopic polyangitis):

*find extra info on this subject in the practical file, page 55

## Hypersensitivity vasculitis – Clinical sign (gross) :



- Purple-colored spots and patches on the skin.

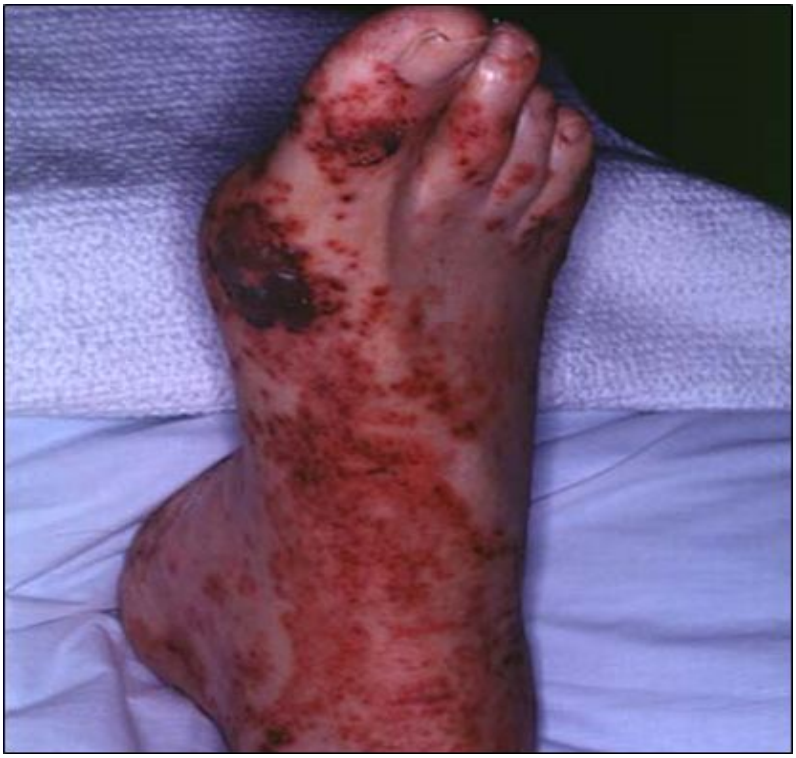
**Hypersensitivity vasculitis might be complicated with :**

1. glomerulonephritis lead to haematouria .
2. hemoptysis due to pulmonary capillaritis .
3. GIT hemorrhage manifested by abdominal pain .

**-test ? >> immunofluorescence for detecting IgA**

Usually affects the lower limbs + trunk

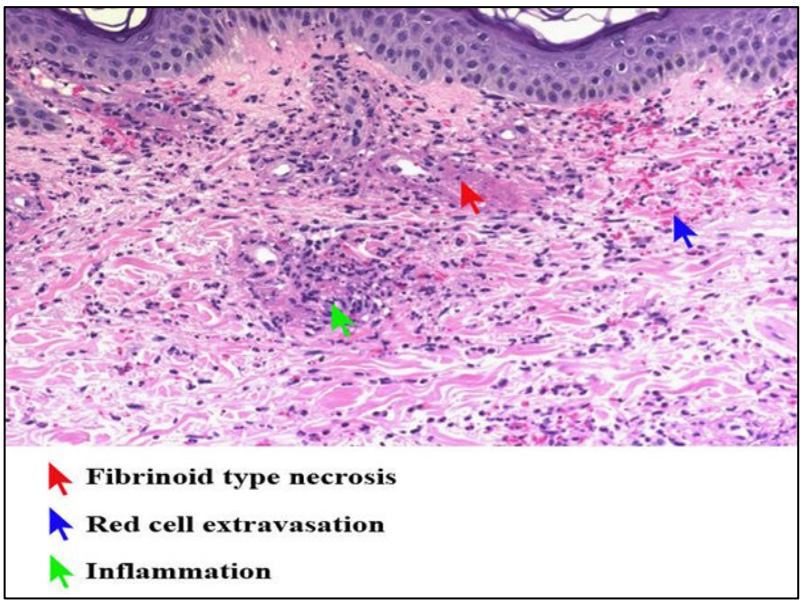
# Leukocytoclastic vasculitis - Clinical sign:



**Leukocytoclastic vasculitis**

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

# Histology - HPF

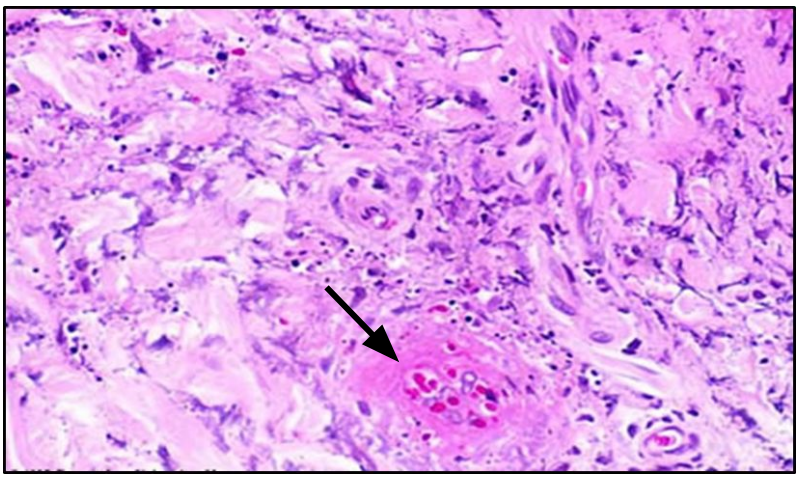



**Vasculitis, leukocytoclasis**

**Section of the skin shows:**

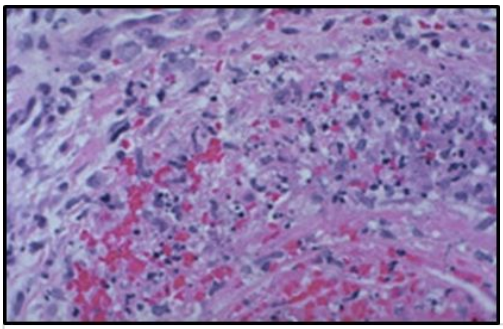
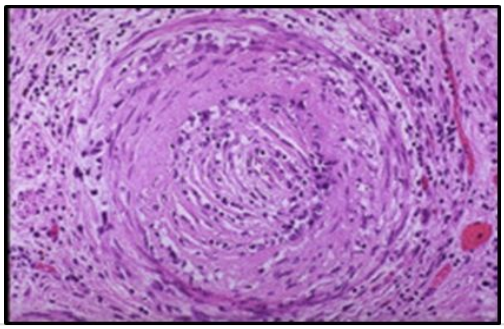
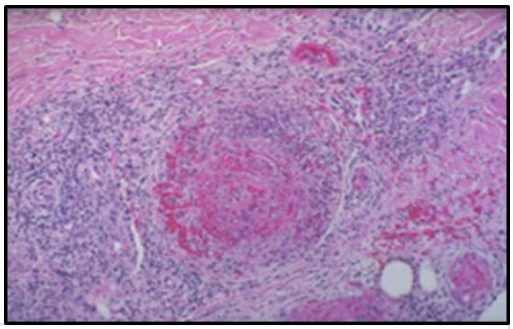
- fibrinoid necrosis of blood vessels (red arrow)
- extravasation of RBCs (blue arrow)
- neutrophilic infiltration with debris (leukocytoclasis /nuclear dust) (green arrow)

**Biopsy? Skin biopsy**



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

# Severe vasculitis – Microscopic views

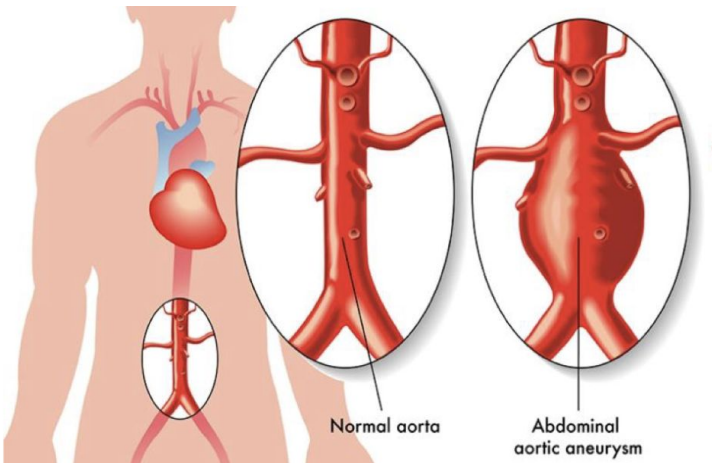
This muscular artery shows a more **severe vasculitis** with:

- acute and chronic inflammatory cell infiltrates
- necrosis of the vascular wall.

# Case 6: Aneurysm of abdominal aorta

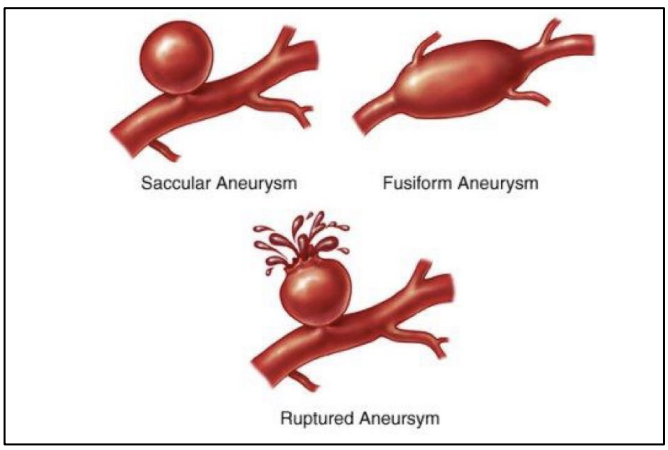
هو تضخم الشريان الأورطي الموجود في البطن مقارنة بالطبيعي

*You can find more extra information at page 61 from the practical file



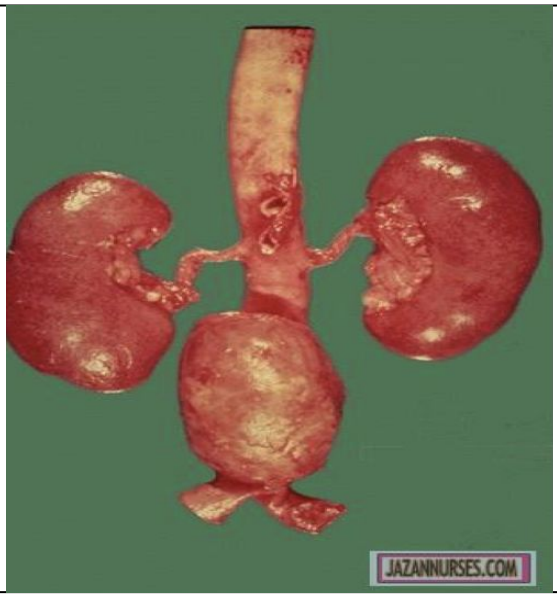

**The most likely causes of aneurysms are:** atherosclerosis , mycotic, syphilitic and congenital

## Types of Aneurysms

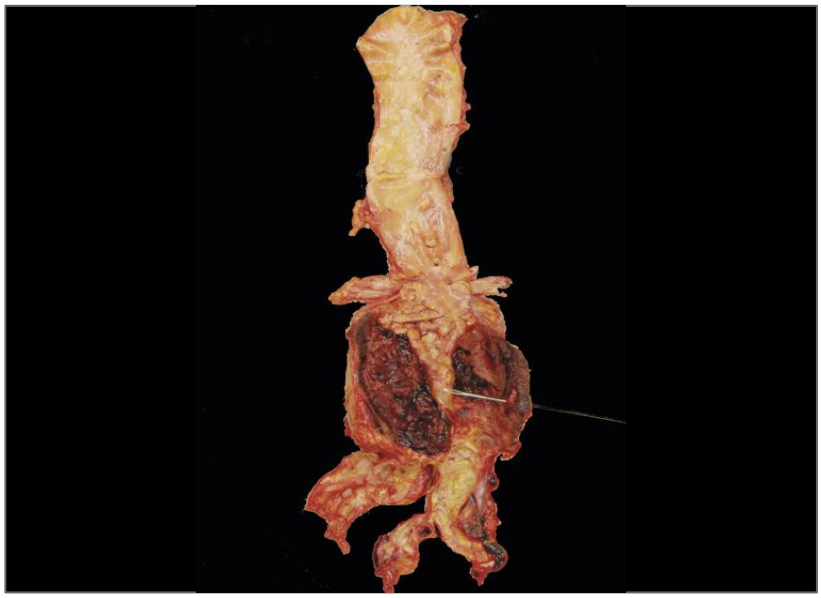



**Other conditions that weaken vessel walls:**
- congenital defects (Circle of Willis: Berry aneurysm)
- Thoracic aorta: Syphilitic (Luetic) aneurysm

# Gross :




**(Abdominal aortic aneurysm)**
**Atherosclerotic aneurysms of the aorta :**
- Large (bulge) above the aortic bifurcation.

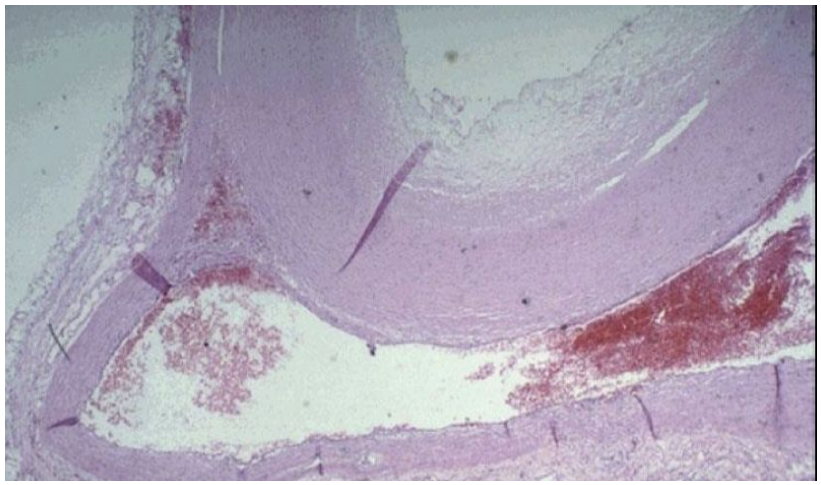


**Origin is abdominal aorta**
- Aneurysmal dilatation of abdominal aorta
- Intraluminal thrombus
- Advanced atherosclerosis

**Here the patient had suddenly developed severe abdominal pain, shocked and collapsed.**

# Dissecting aortic aneurysm LPF :

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.



**We can see:**
-Dissecting aortic aneurysm
- Blood between two layers of the aortic wall
- The inner layer stripped from the out layer

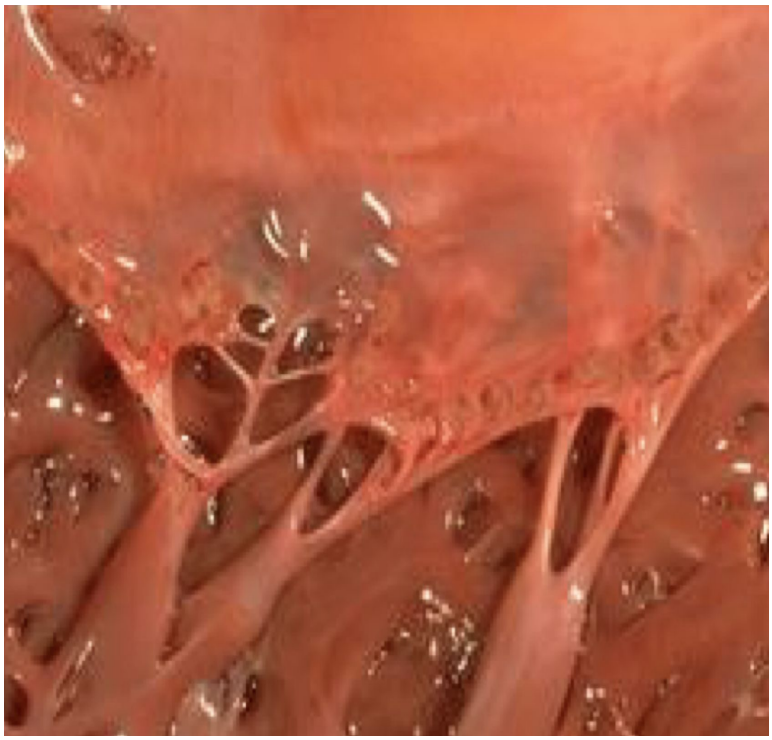
**usually associated with:**
1- atherosclerosis
2- inflammation
3- degeneration of C.T of tunica media

# Case 7: Valvular Heart Disease "rheumatic fever"

*You can find more extra information at page 66 from the practical file

## A- Acute Rheumatic Mitral Valvulitis

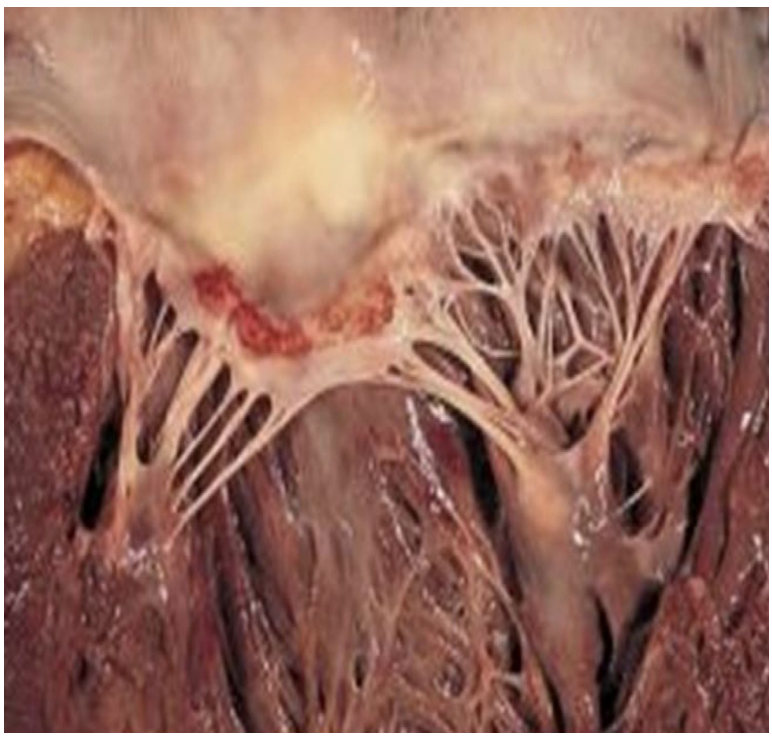
## Gross:



**Rheumatic mitral valves shows:**
Multiple small warty verrucous vegetations* which are:

- firm.
- adherent.
- small, about 1-3 mm in diameter.
- form along the line of valve closure over areas of endocardial inflammation.

**Affects mainly Aortic & Mitral valves**



**Rheumatic mitral valves shows:**
- severe thickening and retraction of the cusps
- chordae tendineae are shortened and fused into short thick cords.

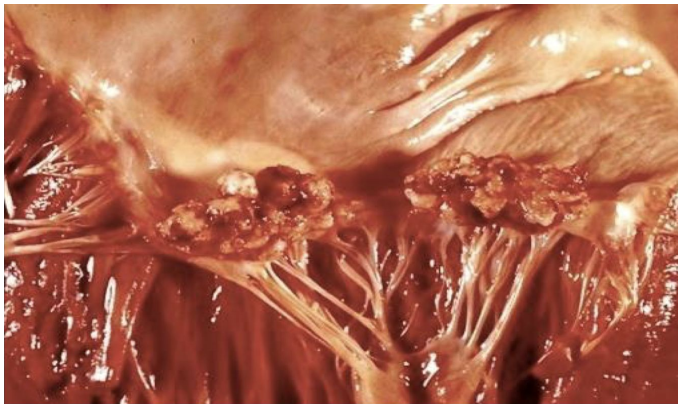
**This rigid valve would have been stenosed**

*vegetations: abnormal growth in or on the body

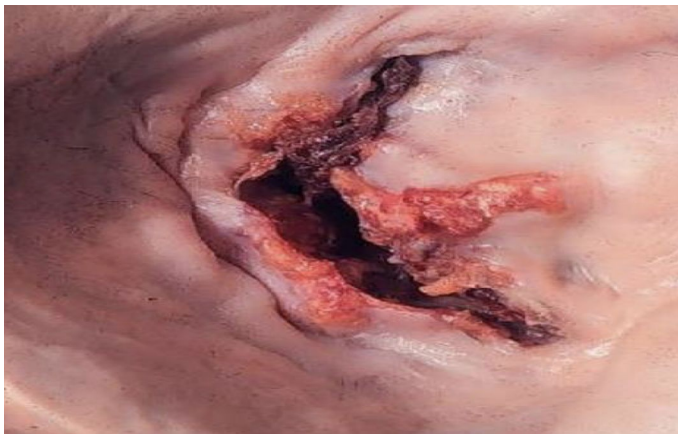
Treatment: surgery, replacement of the affected valve

# B- Chronic Rheumatic Mitral Valvulitis

## Gross:

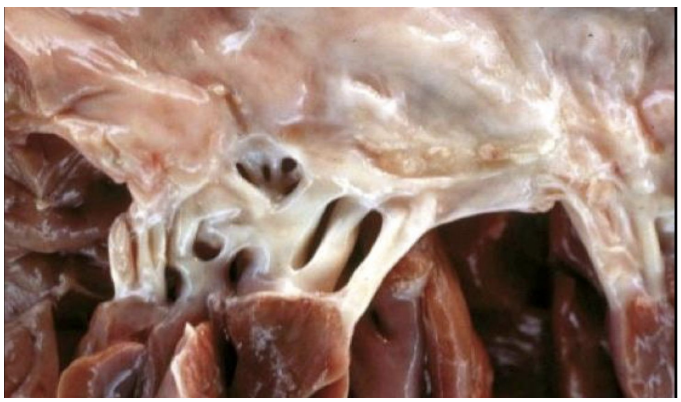


**large vegetations/hemorrhage along the free margins of the mitral valve.**



**Mitral stenosis secondary to rheumatic valvulitis:**

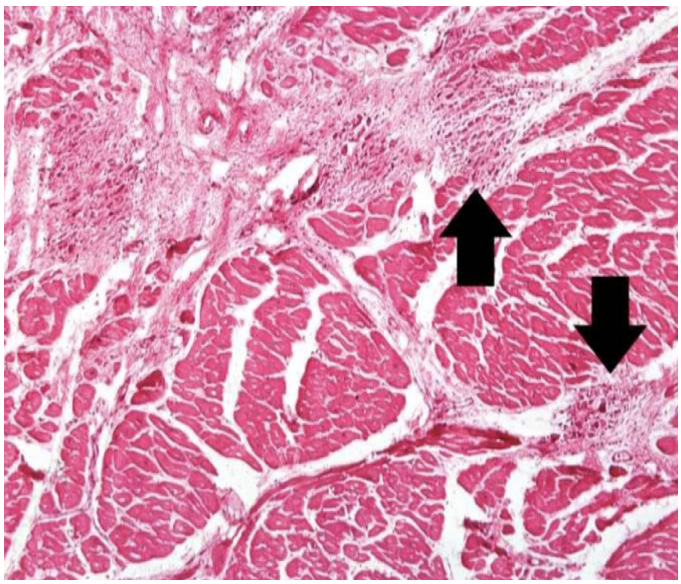
- **Stenotic valve with "fish mouth" appearance.**
- **Fusion of valve commissures.**
- **Thickened and calcified cusps.**
- **Vegetations.**



**Mitral valves shows:**

-The valve leaflets are: thick, fibrotic, fused.

-Chordae tendineae are: short, thickened, fused, stenosis and / or incompetence.



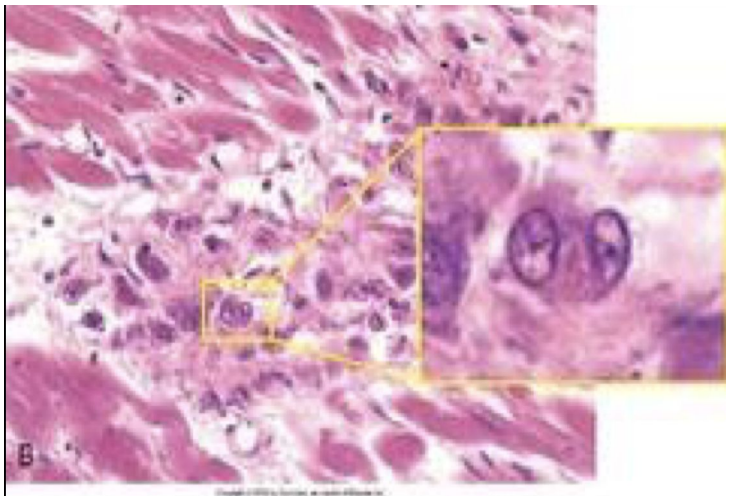
**The valve is thickened by:**

- dense hyalinized fibrous tissue
- vascularization
- chronic inflammatory cell infiltrate

**interstitium of myocardium shows:**

- *Arrows*= cellular accumulations (Aschoff bodies)

# An Aschoff nodule at high magnification



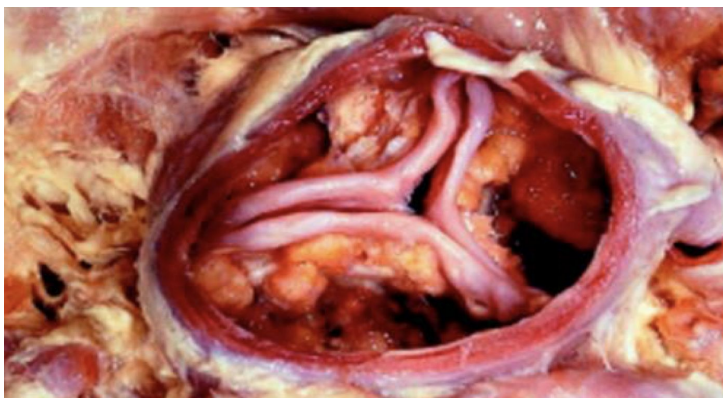
**It affects mainly the left side of the heart and in particular the posterior wall of the left atrium.**

The most characteristic component is the **Aschoff giant cell**. (Originate from macrophages)

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

**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).
**Jones criteria (Major ):** (1) carditis; (2) migratory polyarthritis of large joints; (3) subcutaneous nodules; (4) erythema marginatum skin rashes; and (5) Sydenham chorea, a neurologic disorder characterized by involuntary purposeless, rapid movements (also called St. Vitus dance).
**Minor criteria** such as fever, arthralgias, ECG changes, or elevated acute phase reactants also can help support the diagnosis.

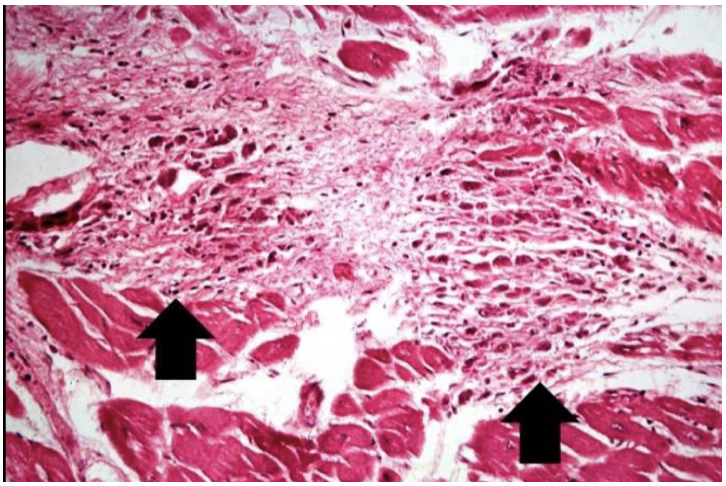
## C- Rheumatic Aortic Valvulitis



**Aorta has been removed to show:**

- Aortic stenosis.
- thickened, fused aortic valve leaflets.

## Acute Rheumatic Carditis -HPF

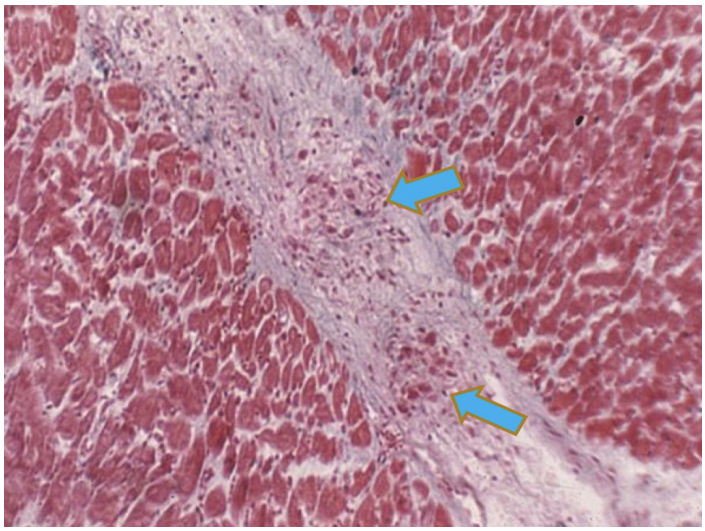


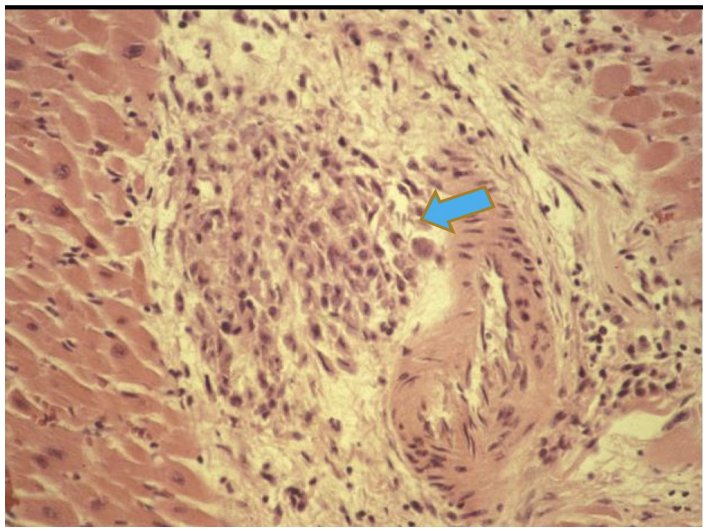
**Acute rheumatic carditis is marked by:**

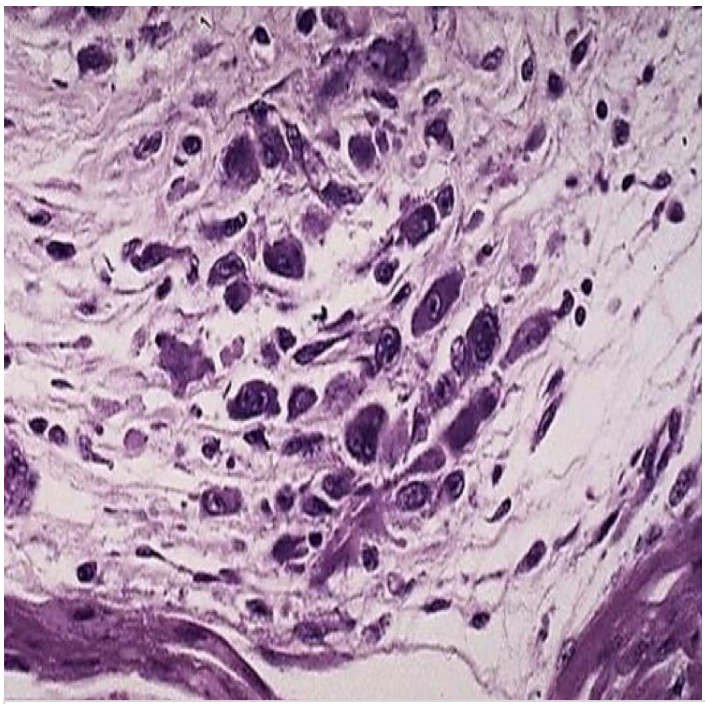
a peculiar form of granulomatous inflammation with so-called "Aschoff nodules".

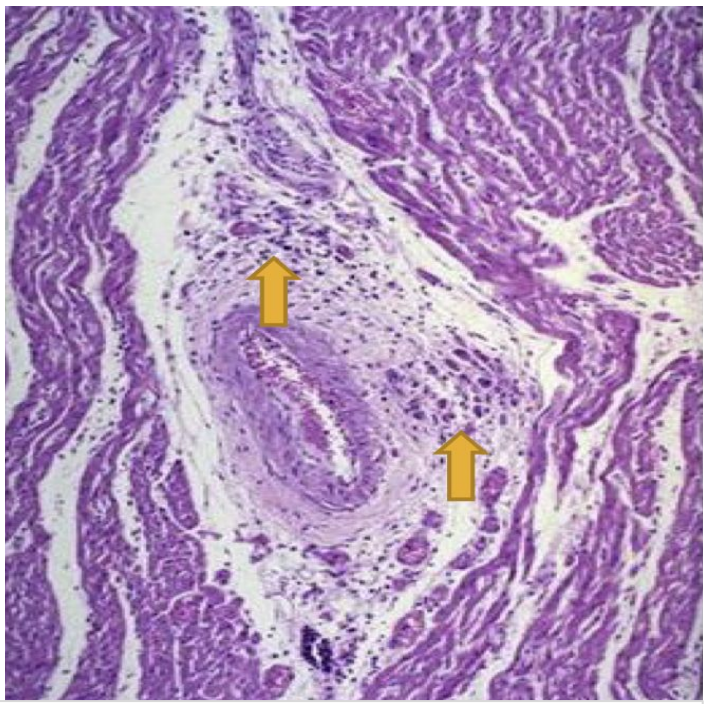
**Aschoff nodules seen best in myocardium (seen in acute more than chronic)**

# Aschoff Nodules









**Intermuscular fibrous septa Aschoff bodies, they are:**

- oval in shape.
- Seen in relation to blood vessels.

**Each nodule is consists of a focus of:**

- fibrinoid necrosis.
- few lymphocytes.
- macrophages.
- few small giant cells with one or several nuclei **(Aschoff giant cell).**

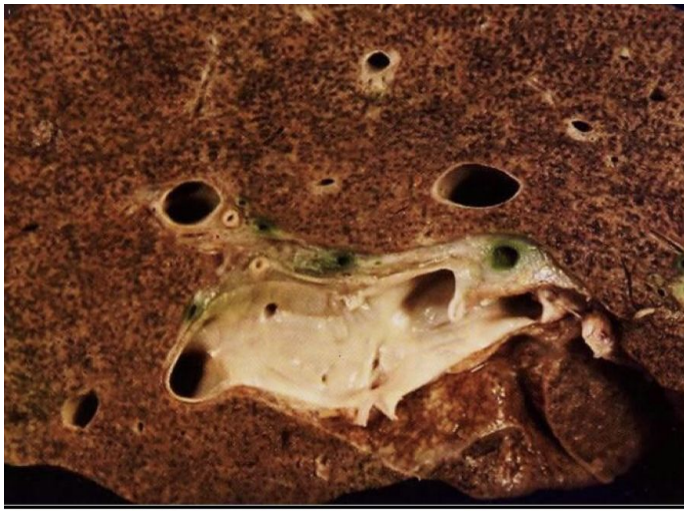
# Case 8: Right Sided Heart Failure

## Chronic venous congestion of the liver

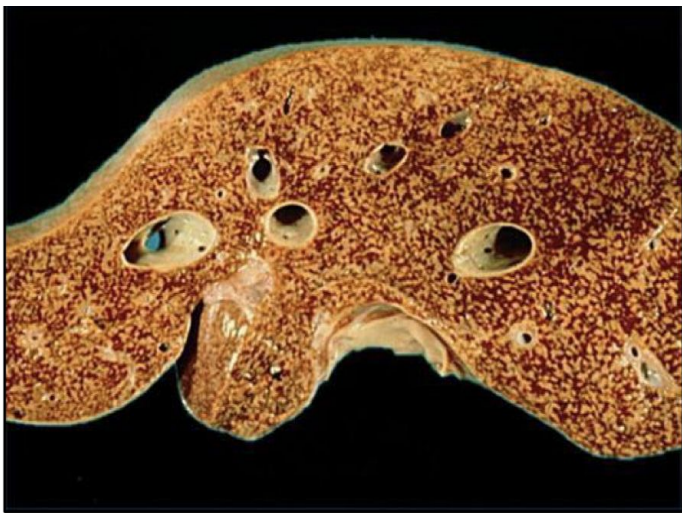
*You can find more extra information at page 79 from the practical file

# Gross:

## Nutmeg liver - cut surface

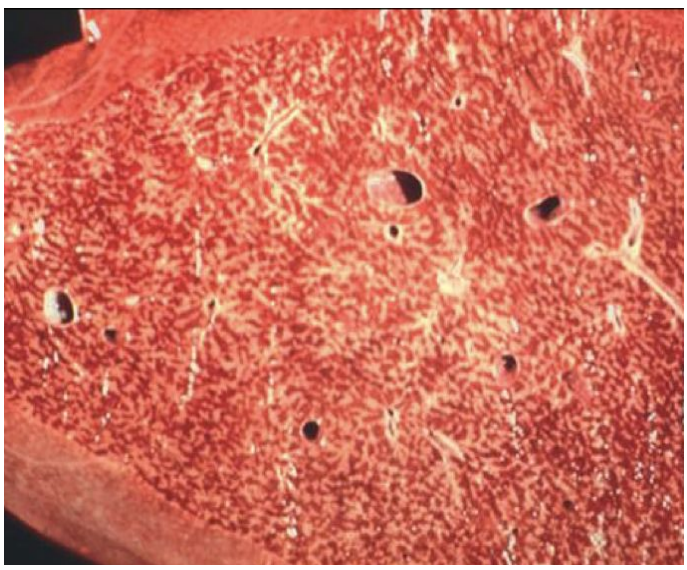


**Section of liver showing:**
- Alternating pale and dark areas
- nutmeg like appearance possibly due to passive congestion secondary to right sided heart failure



**Hepatic parenchyma contain:**
- faintly nodular pattern
- nutmeg staining due to chronic passive congestion due to right sided heart failure

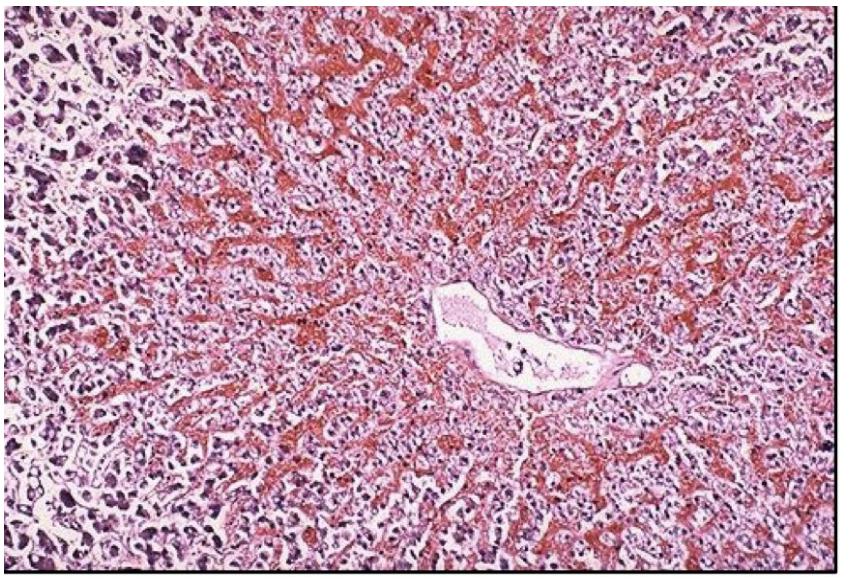
## Chronic Congestion of the Liver -CS



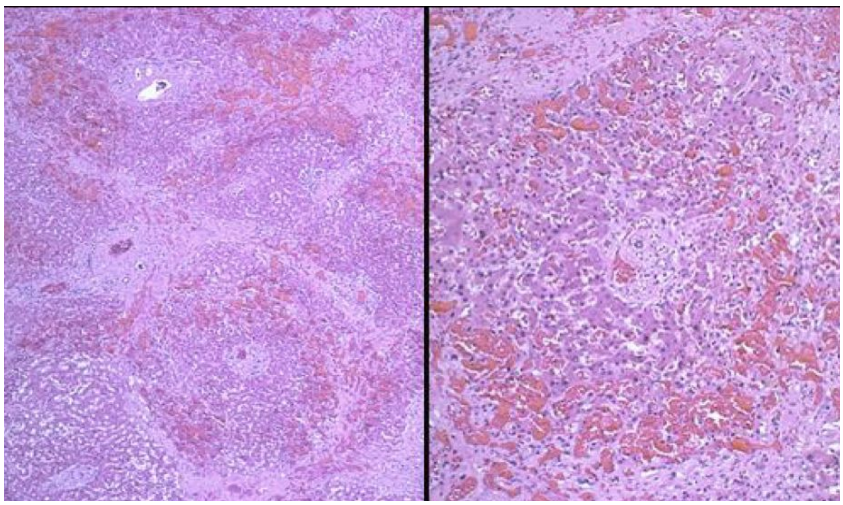
**Gross view of nutmeg appearance of liver characteristics of :**
- centrolobular or necrosis or passive congestion of the liver

**The Central areas are:**
- congested
- take on a sort of dusky appearance
- soft in consistency
- Surrounded by polar areas of fatty liver that are more normal in appearance microscopically.

# Histology - LPF

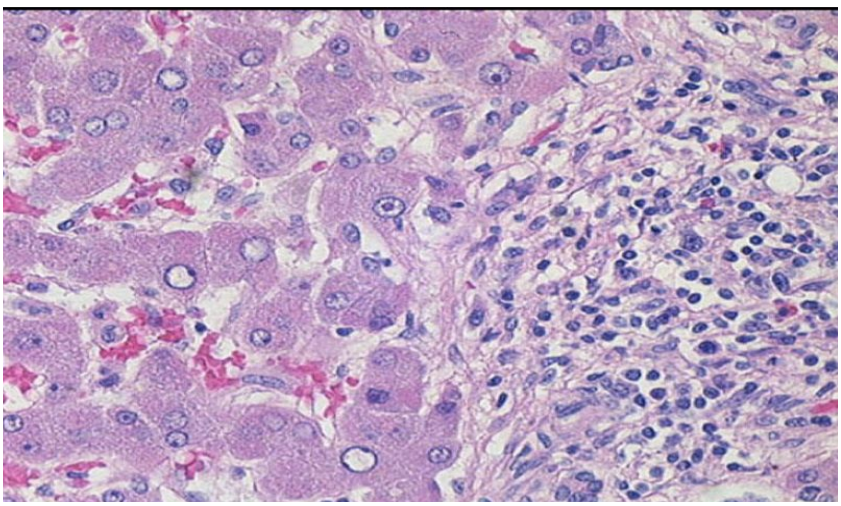


**The central portion of liver lobules shows:**
- congestion and dilation of central veins
- blood sinusoids
- atrophy and necrosis of liver cells



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

# Histology - HPF



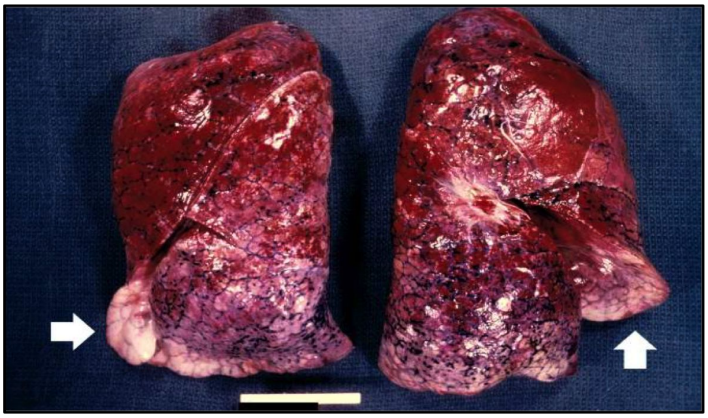
**The central portion of liver lobules shows:**
- congestion and dilation of central veins
- blood sinusoids
- atrophy and necrosis of liver cells

# case 9: left Side Heart Failure

## Chronic venous congestion of the lung

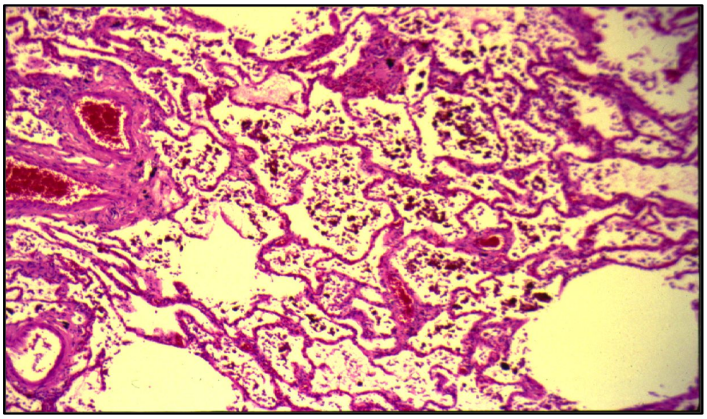
*You can find more extra information at page 86 from the practical file

## Gross

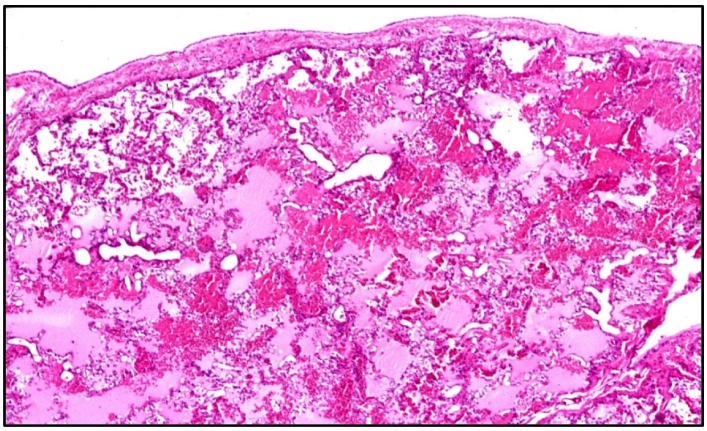


**gross photograph of lungs shows:**
- lungs are distended and red
- the redness of the tissue is due to venous congestion
- some normal pink lung tissue at the edges of the lungs (arrows)

## Histology - LPF



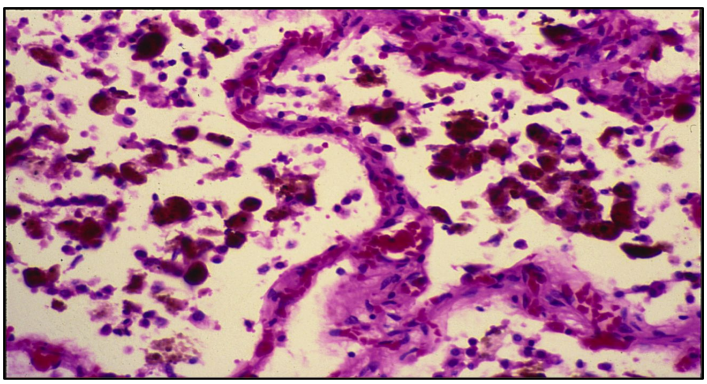
**The alveolar walls are thickened by dilated and engorged capillaries.**



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

## Histology - HPF

*you see them in the lung not the heart



**The alveoli contain:**
- edematous fluid
- RBC
- large alveolar macrophages (Heart Failure cell)* **Which are filled with hemosiderin pigment derived from red cells breakdown.**

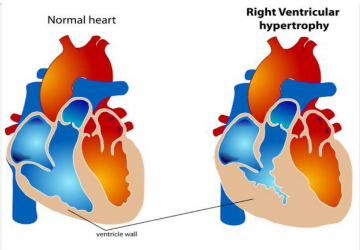
# case 10: Myocardial Hypertrophy

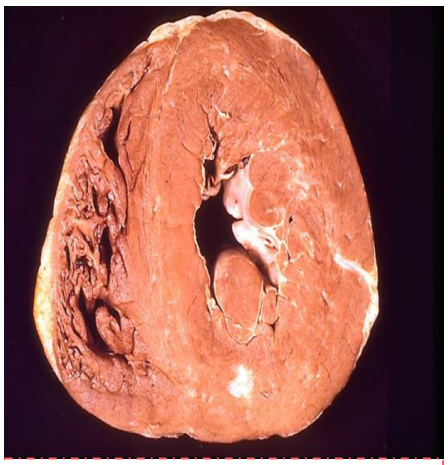
*You can find more extra information at page 86 from the practical file

**Causes of Left ventricular hypertrophy:**
- Systemic hypertension
- Aortic valve stenosis

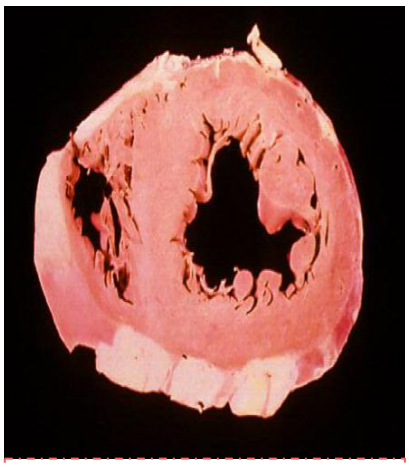
**Causes of Right ventricular hypertrophy:**
- Pulmonary hypertension

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

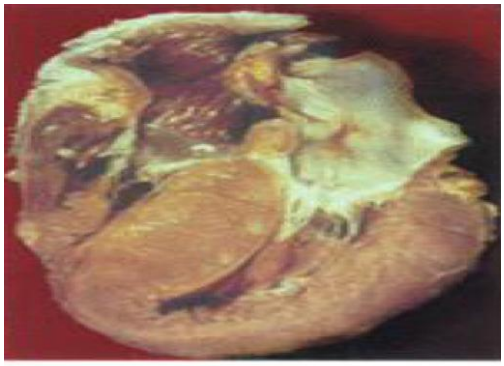


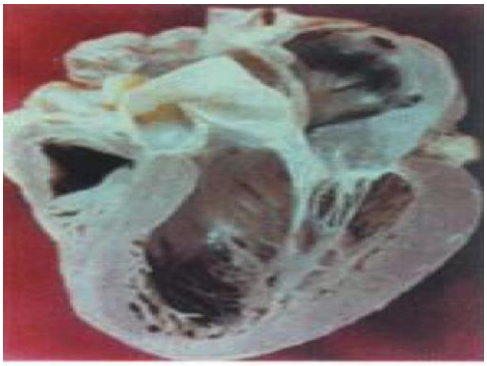



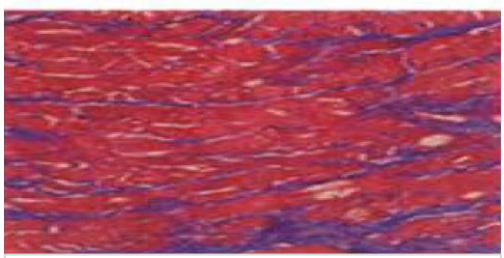
**Left ventricular hypertrophy**



**Normal ventricles**





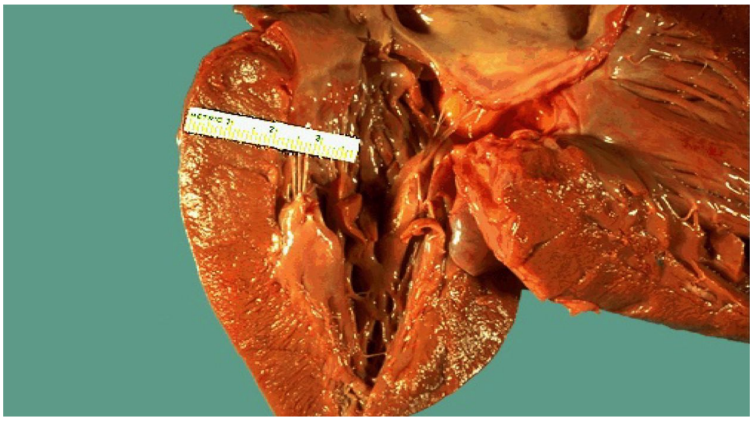


**Histopathology showing:**
- myofiber disarray
- interstitial fibrosis

**Histopathology showing:**
- Normal myocytes

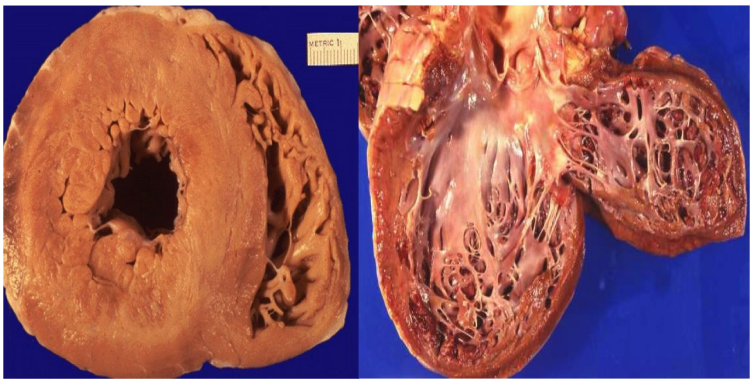
# Left ventricular hypertrophy

## Gross:



**Heart from a hypertensive patient shows:**
- left ventricle is very thick **(more than 2 cm)**
- the rest of the heart is normal in size as is typical for hypertensive heart disease

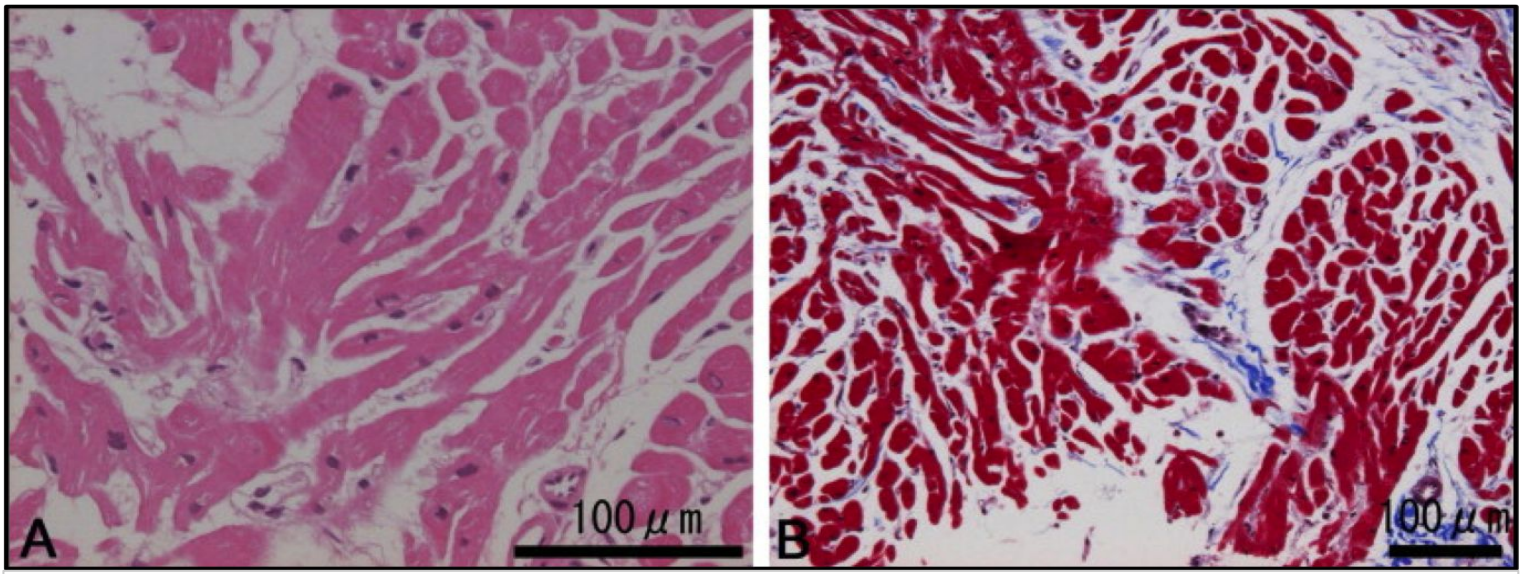
**More info:**
The hypertension creates a greater pressure load on the heart to induce the hypertrophy



**This cross section view of the heart shows:**
- The left ventricle is grossly thickened
- The myocardial fibers have undergone hypertrophy.

**this heart from patient with severe hypertensive**

## Histology - LPF (Hypertrophic cardiomyopathy)



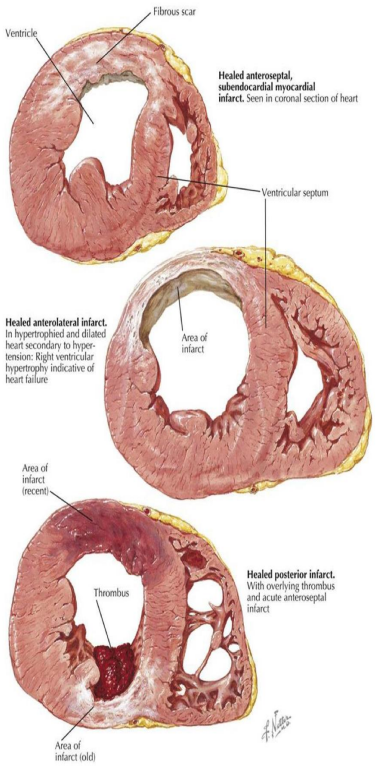

**A- haematoxylin-eosin stain**
**B- Masson's trichrome stain**
**Histopathology of heart sections of ventricular septum showing:**
- interstitial fibrosis indicating hypertrophic cardiomyopathy (HCM).
- myofiber disarray

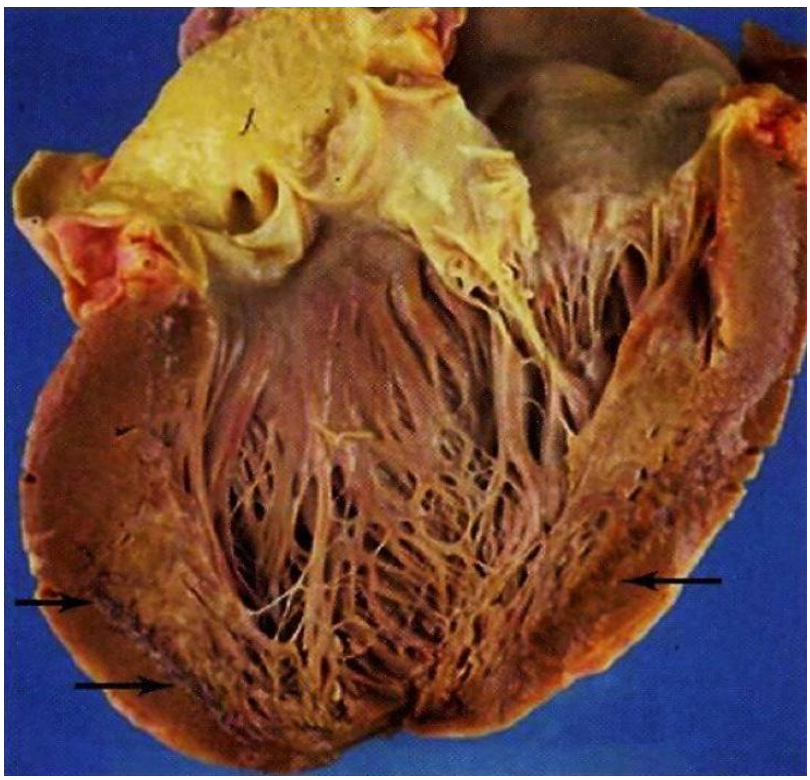
# Case 11 : myocardial infarction

*find extra info on this subject in the practical file, pages 99

## Background information important

| Time Frame | Gross Features | Light Microscopic Findings | Electron Microscopic Findings |
|---|---|---|---|
| Reversible Injury | | | |
| 0–1½ hours | None | None | Relaxation of myofibrils; glycogen loss; mitochondrial swelling |
| Irreversible Injury | | | |
| ½–4 hours | None | Usually none; variable waviness of fibers at border | Sarcolemmal disruption; mitochondrial amorphous densities |
| 4–12 hours | Occasionally dark mottling | Beginning coagulation necrosis; edema; hemorrhage | |
| 12–24 hours | Dark mottling | Ongoing coagulation necrosis; pyknosis of nuclei; hypereosinophilic appearance of myocytes; marginal contraction band necrosis; beginning neutrophilic infiltrate | |
| 1–3 days | Mottling with yellow-tan infarct center | Coagulation necrosis with loss of nuclei and striations; interstitial infiltrate of neutrophils | |
| 3–7 days | Hyperemic border; central yellow-tan softening | Beginning disintegration of dead myofibers, with dying neutrophils; early phagocytosis of dead cells by macrophages at infarct border | |
| 7–10 days | Maximally yellow-tan and soft, with depressed red-tan margins | Well-developed phagocytosis of dead cells; early formation of fibrovascular granulation tissue at margins | |
| 10–14 days | Red-gray depressed infarct borders | Well-established granulation tissue with new blood vessels and collagen deposition | |
| 2–8 weeks | Gray-white scar, progressive from border toward core of infarct | Increased collagen deposition, with decreased cellularity | |
| >2 months | Scarring complete | Dense collagenous scar | |



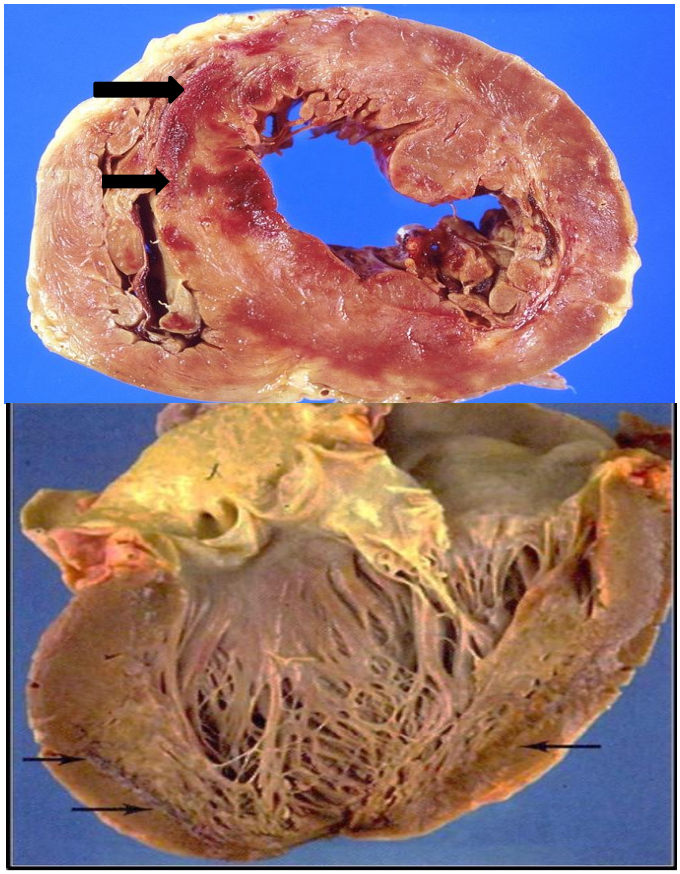



**Complications that might occur:**

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

-Dark red infarction
-Occlusive coronary artery

# Gross:

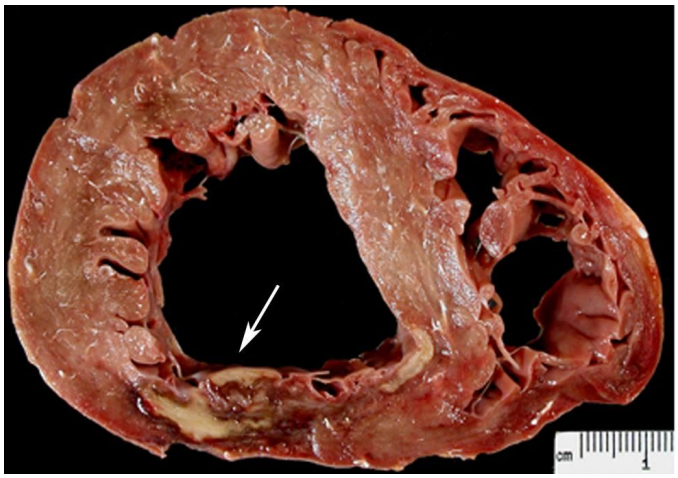


**acute myocardial infarction shows**:

- area of fresh myocardial infarction (arrows) in the **left ventricle.**

Initially the area of fresh infarct appears **red**.

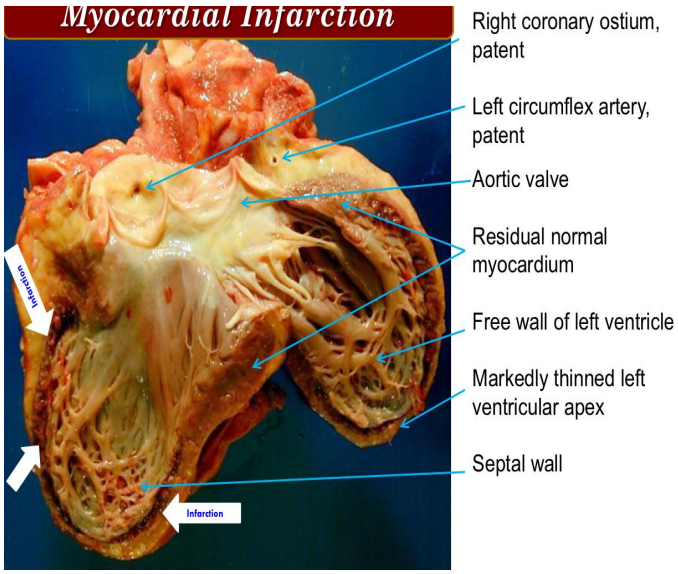
The area of infarct becomes well defined by 2 to 3 days with a central area of yellow discoloration surrounded by a thin rim of hemorrhage.



**Acute myocardial infarct (At 3 days), shows:**

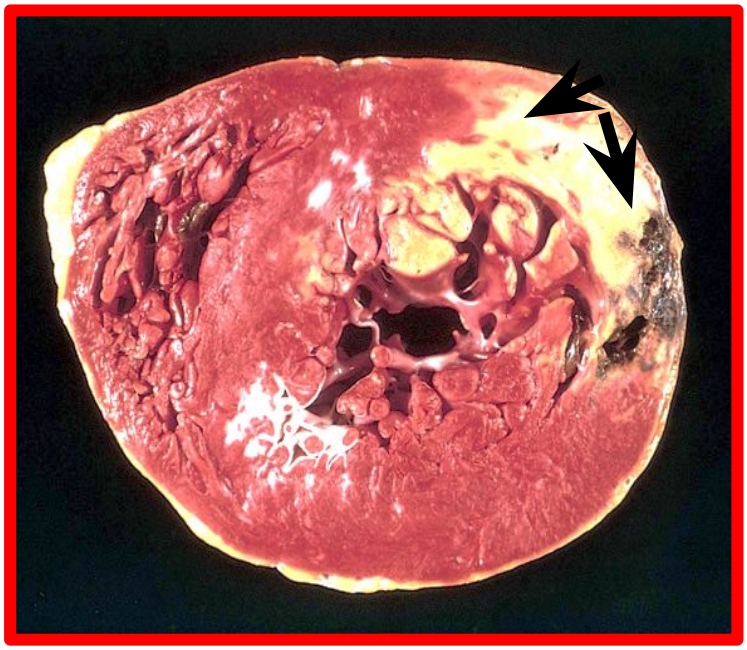
a zone of yellow necrosis surrounded by darker hyperemic borders.

**The arrow points to an infarct in the wall of the left ventricle.**



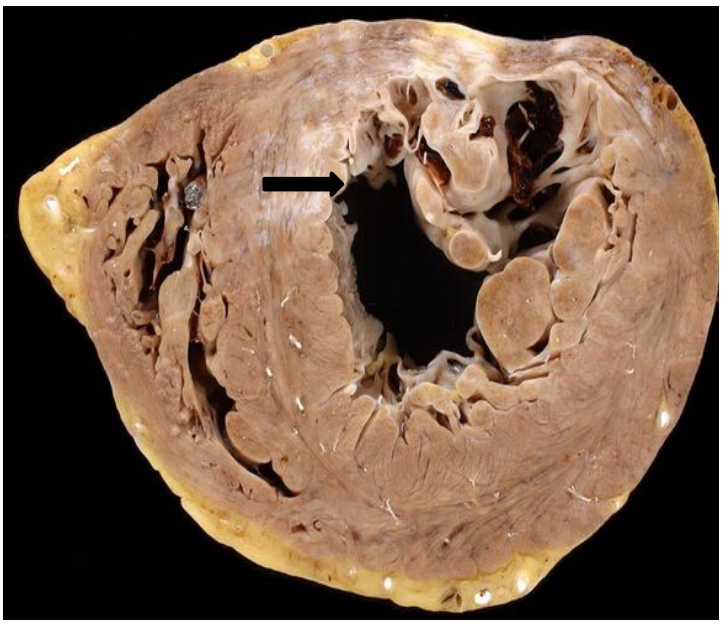

**The heart is opened showing the left ventricle:**

There is a **Massive Transmural Infarction** extending around the entire wall between the white arrows.

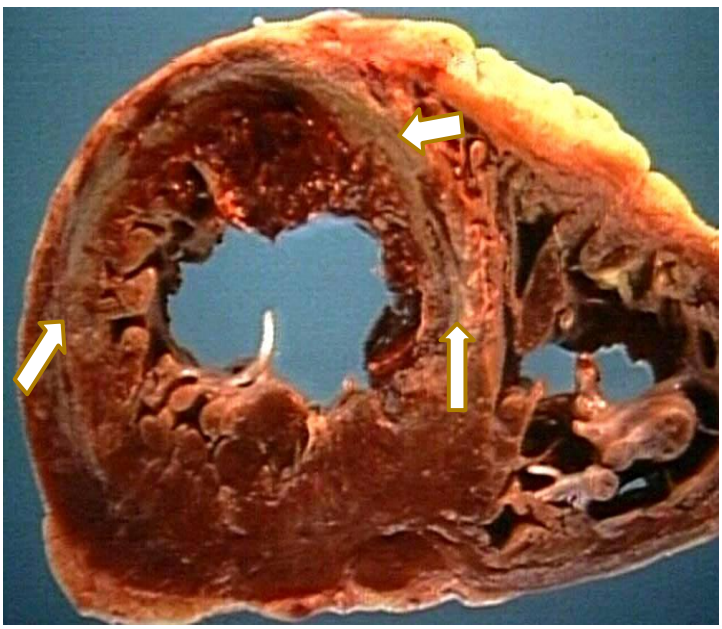


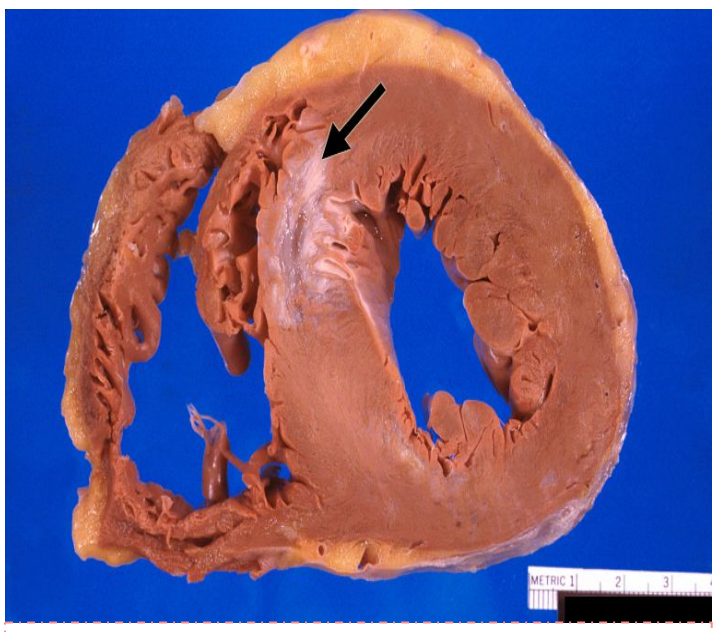
**Cross section of the left and right ventricles shows:**

- pale
- irregular focal fibrosis in the left ventricular wall.
- increased thickness of the left ventricular wall (**hypertrophy**)



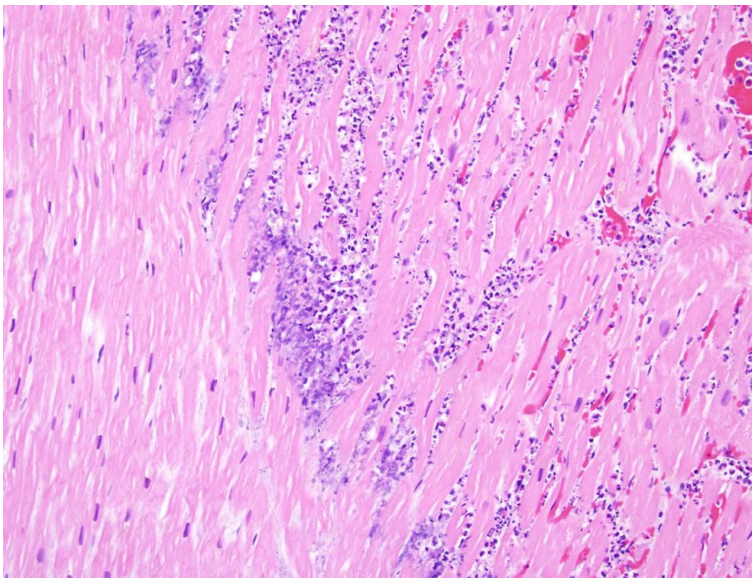
These 3 pictures has the same comment of the previous picture

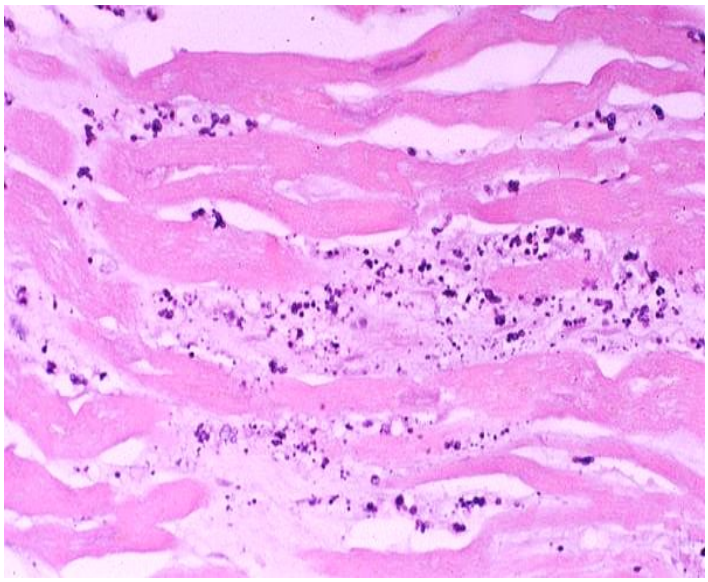




**healed myocardial infarct**

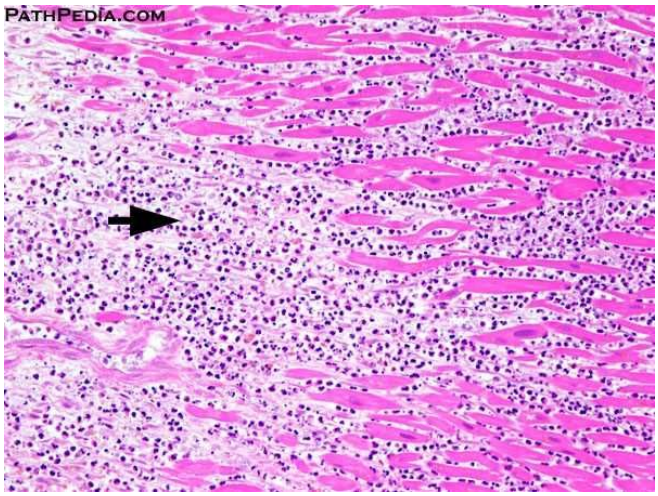
# Histology - LPF

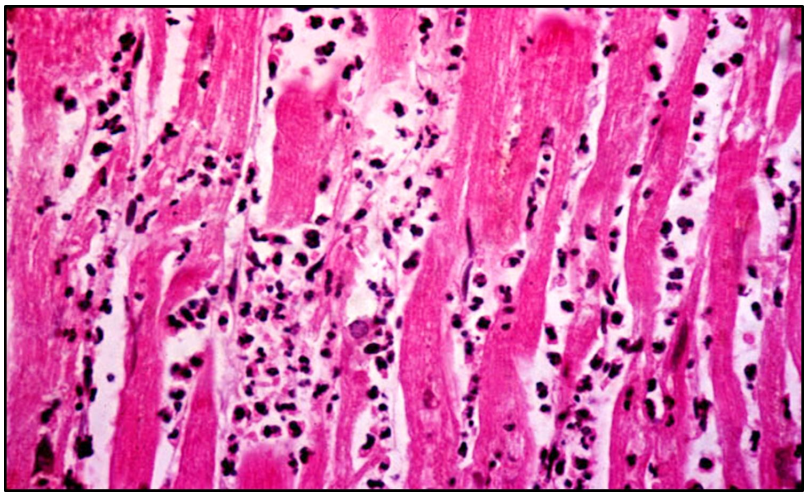




**Acute myocardial infarct (after 24 hours) , shows:**

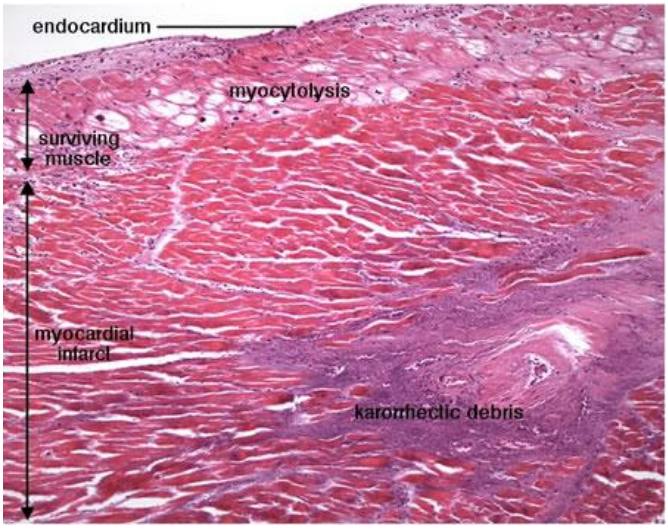
- neutrophilic infiltrate at the border of the infarct.
-neutrophils are seen infiltrating the necrotic muscle.
-Viable myocardium at the left.
-the nuclei are not clearly visible in most of the necrotic cells.



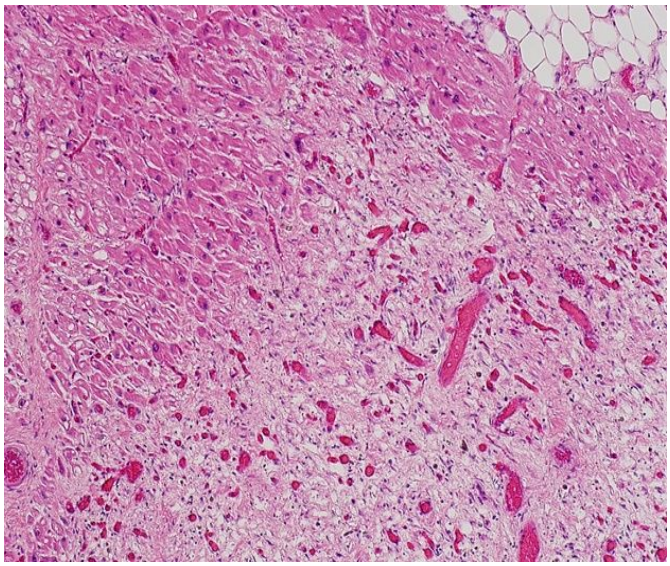



**Acute myocardial infarction (a 3-day old acute infarct ), showing;**

**-** 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.



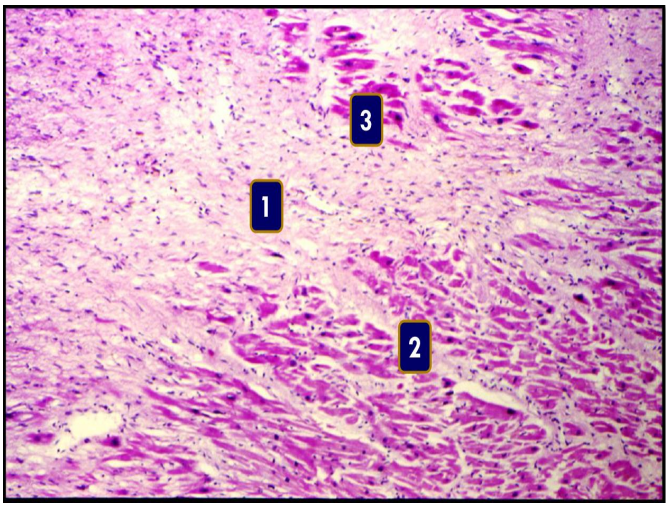

**Transmural myocardial infarct at 2 weeks**



**Recent MI with early healing changes (3 weeks post MI) , showing:**

- granulation tissue (growth of capillaries and fibroblasts)
- collagen is being laid down to form a scar.

**The non-infarcted myocardium is present on the left and upper part of the picture.**




**Healed myocardial infarct (This stage is reached about 2 months post MI), showing:**

-replacement of the necrotic cells by a dense collagenous scar.

-The myocardium shows fibrosis with collagenization (scar)

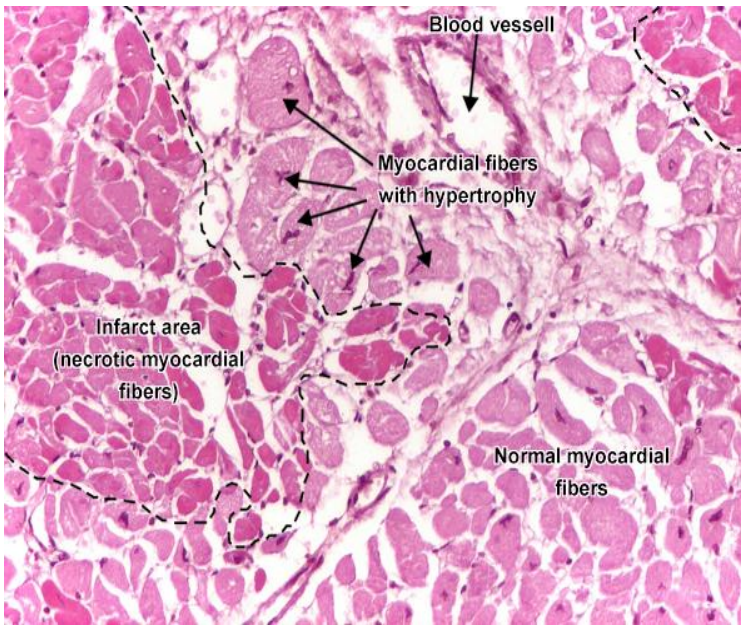
-Residual viable red myocardial fibers are present.

**the numbers on the picture show:**

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 .

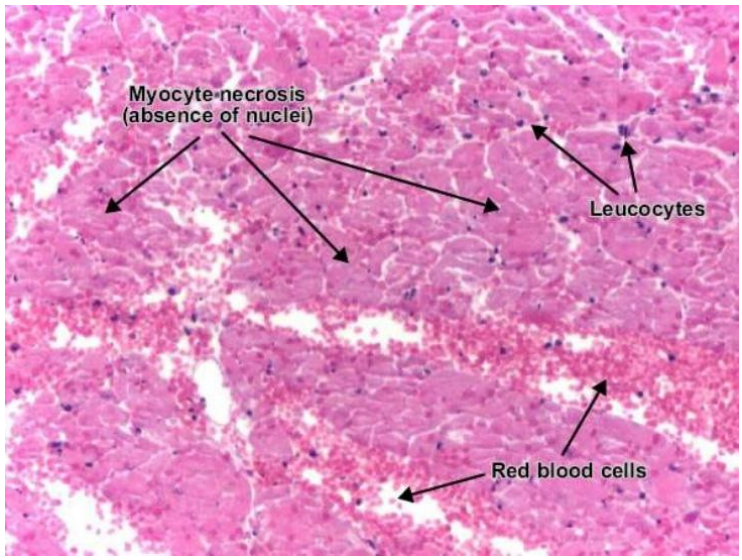
3- The remaining myocardial fibers show enlarged nuclei due to ventricular hypertrophy .




**Myocardial infarct - circumscribed area of ischemic necrosis - coagulative necrosis.( In the first 12 - 24 hours) showing:**

- myocardial fibers are still well delineated
- intense eosinophilic (pink) cytoplasm
- myocardial cells lost their transversal striations and the nucleus **(left side of the picture)**.

-a few myocardial fibers showing hypertrophy (increased size of the fiber, irregular shape of the nuclei).



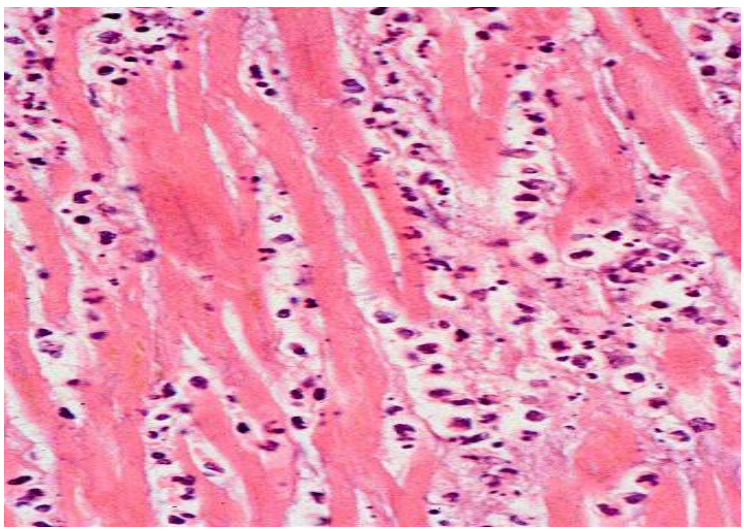

**Recent myocardial infarct (in the first 12 - 24 hours) showing:**

- myocardial fibers are still well delineated
- intense eosinophilic (pink) cytoplasm

-myocardial cells lost their transversal striations and the nucleus.

-The interstitial space may be infiltrated with red blood cells.

# Histology -HPF



**Acute myocardial infarct, (1-3 / 3-4 day infarct) showing:**

- coagulation necrosis of myocardial cells
- Neutrophilic (polymorphonuclear) infiltration..
- **tests? Troponin (protein) / CK-MB (enzyme)**

**Cause: 1/ Narrowing of lumen caused by atherosclerosis. 2/ Haemorrhage and thrombosis over atheromatous plaques.**

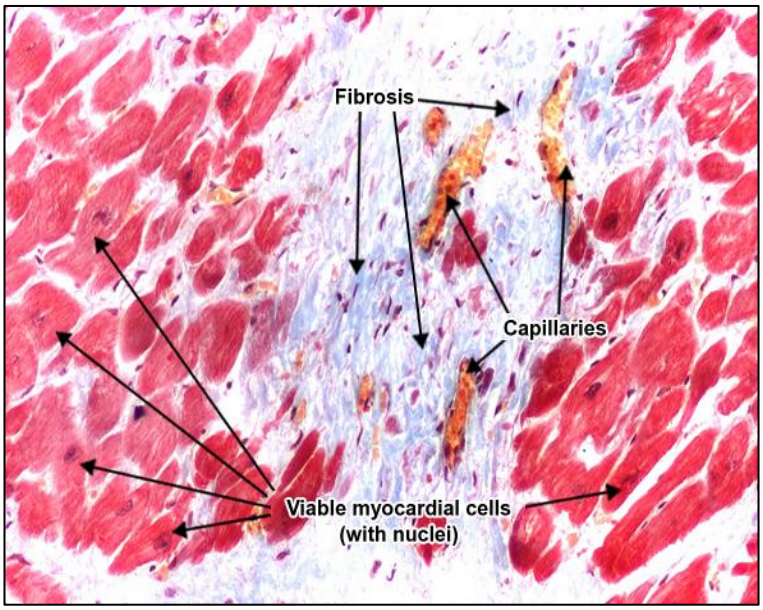
**Complications: • Heart failure • Arrythmias • Ventricular aneurysm • Myocardial rupture • Ruptured papillary muscle**

Microscopic image of Myocardial Infarction

**no comments were written on this picture.**

# Ischemic fibrosis of myocardium
## (diffuse ventricular myocardial fibrosis)

This picture in male slides was with case 2 : coronary atherosclerosis

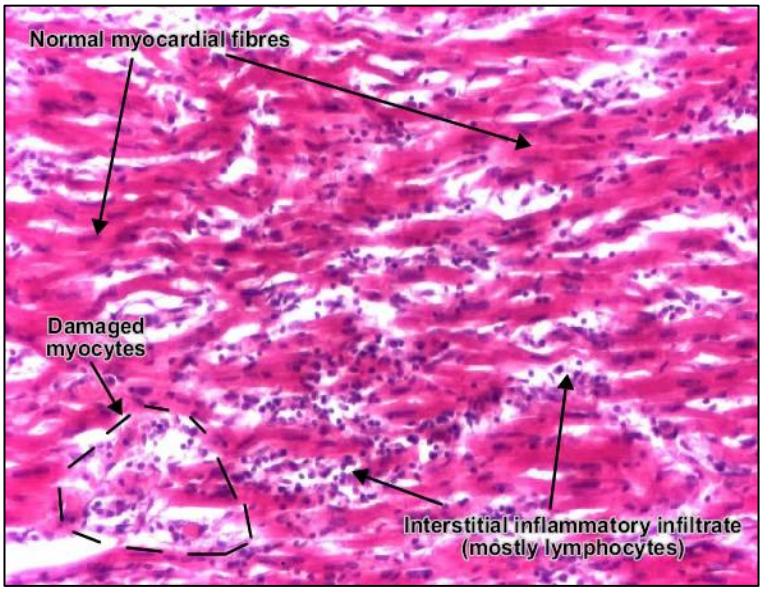



**Diffuse myocardial fibrosis (Ischemic fibrosis of the myocardium) , showing:**

- 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

This picture in male slides was with case 2 : coronary atherosclerosis




**Acute viral myocarditis, showing:**

-an abundant edema in the Myocardial interstitium

-inflammatory infiltrate, mainly with lymphocytes and macrophages.

**Myocarditis** is an inflammation of the myocardium**. Acute viral myocarditis** is produced most often by **Coxsackie B virus** and **echoviruses.**

MED437
KING SAUD UNIVERSITY

PATHOLOGY
TEAM 437

# Team Leaders:

Dimah Alaraifi  Mansour Alobrah

---

# Team Members:

- Shirin Hammadi
- Laila Alsabbagh
- Marwah Alkhalil
- Ghada E.Almuhanna
- Lujain Alzaid