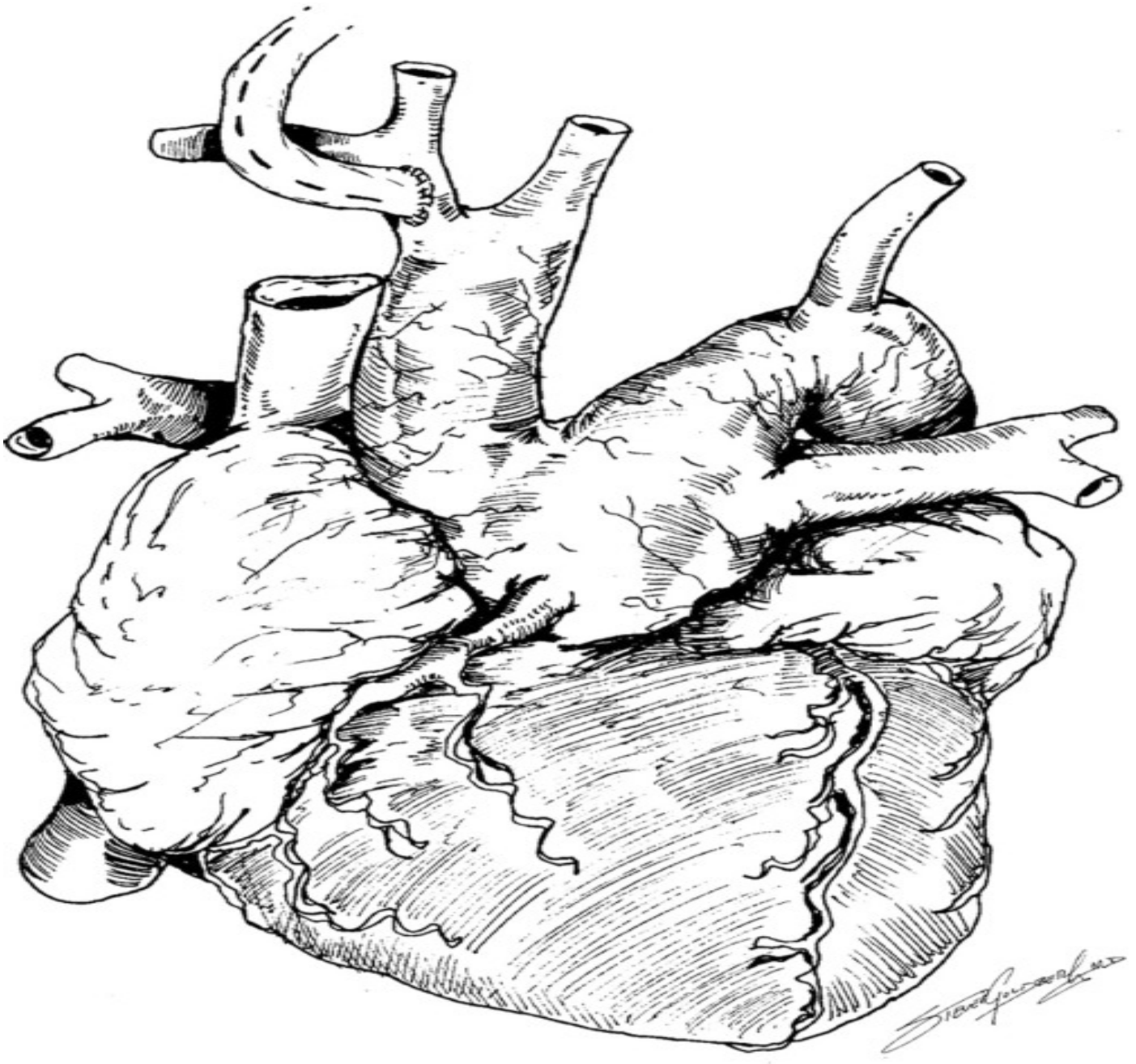
Pathology Practical

Cardiovascular block

Team 435



Color coding:

Dr Marei's notes: Blue

Dr shaesta's notes: Purple

Both doctors notes "most imp": RED

An Extra slide from Dr.Shaesta Zaidi for better understanding.

Arteriosclerosis " Broadest term"

(literally, "hardening of the arteries") is a generic term for thickening and loss of elasticity of arterial walls.

Atherosclerosis,

the most frequent and important pattern.

Two anatomic variations:

- Hyaline -> diabetes mellitus.
- Hyperplastic -> Hypertension

Mönckeberg medial calcific sclerosis characterized by calcific deposits in muscular arteries in persons older than age 50.

Arteriolosclerosis

affects small arteries and arterioles.

Differences between secondary changes and complications:

- Secondary changes:
  What effect can happen
- Complications:
  what will happen after a while, if not treated.

Case #1: Atheroma of the aorta

Theoretical Information's:

- **An atheroma**: an accumulation and swelling in artery walls made up of macrophage cells, or debris, and containing lipids (cholesterol and fatty acids), calcium and a variable amount of fibrous connective tissue.
- **Risk factors:** Male gender, Post menopausal woman, Hyperlipidemia, Hypertension., Diabetes mellitus, Cigarette smoking.
- **Complications:** 1-Vascular thrombosis and distal embolization 2- Aneurysm formation 3- Cardiac ischemia 4- Ischemic encephalopathy 5- Intermittent claudication.

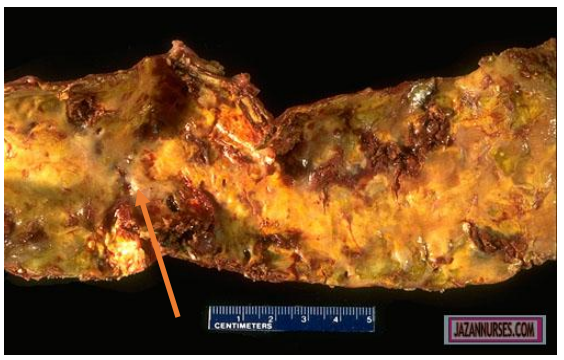
The atheromatous plaques undergoes "secondary changes", "complications":

- 1-Ulceration 2-haemorrhage 3-hrombosis 4-aneurysmal dilatation 5-calcification.

Advanced and complicated atherosclerosis Gross

1-atheromatous plaques "orange arrow".
2-ulceration and haemorrhage " blue arrow"

Atheroma of the Aorta - Gross

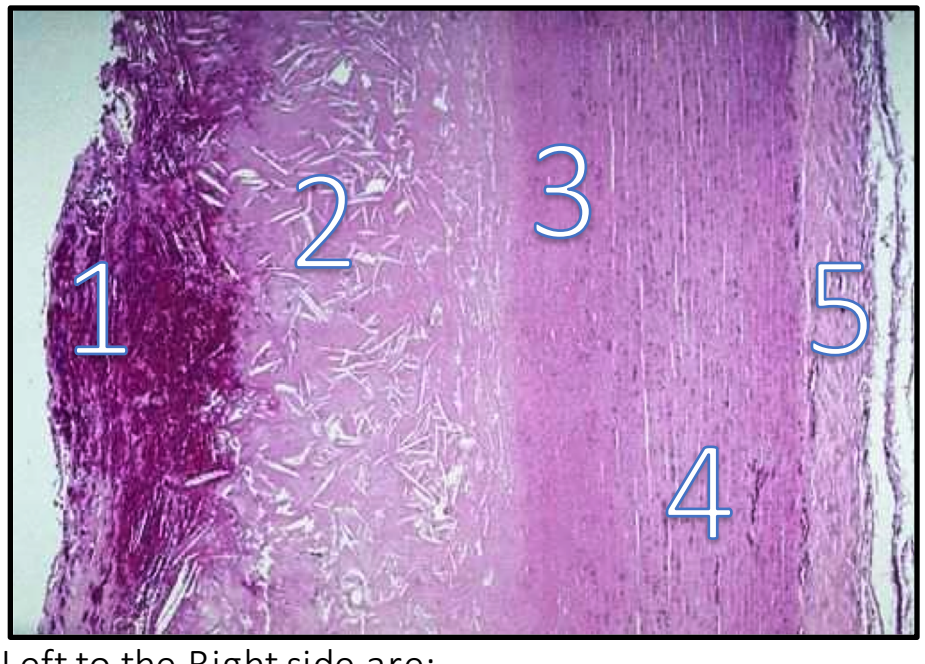


- Severe atherosclerosis of the aorta, ulceration.

Atheroma of the Aorta - Gross

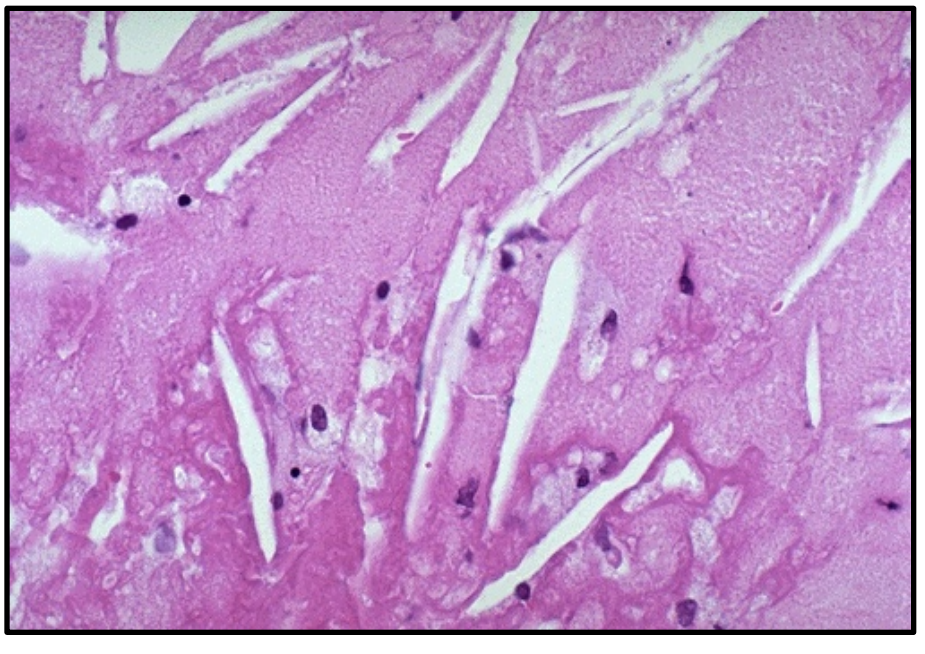
From bottom to top:
1- Mild: shows only scattered lipid plaques. " fatty streaks"
2- Moderate: shows many more larger plaques
3- Sever: shows extensive ulceration in the plaques.

Atheroma of the Aorta - LPF



Left to the Right side are:
1- Haemorrhage
2- Atheromatous plaque containing cholesterol clefts
3- Fibrosis
4- Elastic Media
5- Adventitia

Atheroma of the Aorta – LPF The doctor loves this picture.



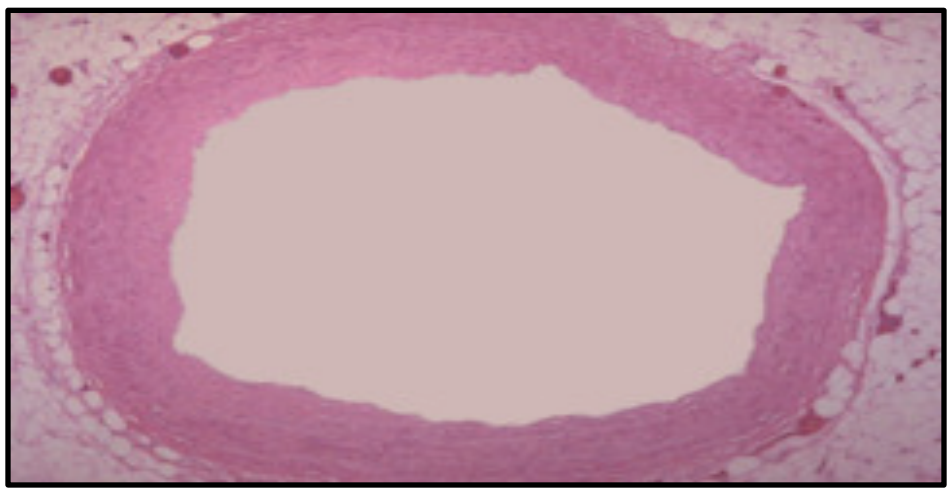
1- Foam cells or Macrophages,
2- Cholesterol clefts
Macrophages function: Engulfing.

Case #2: Coronary atherosclerosis

Theoretical Information's:.

- More effective than Aorta.
- Complications: Myocardial infarction
- Risk factors: Male gender, Post menopausal woman, Hyperlipidemia, Hypertension., Diabetes mellitus, Cigarette smoking.
- Arteriolosclerosis -> small arteries, atherosclerosis -> Medium to large arteries.

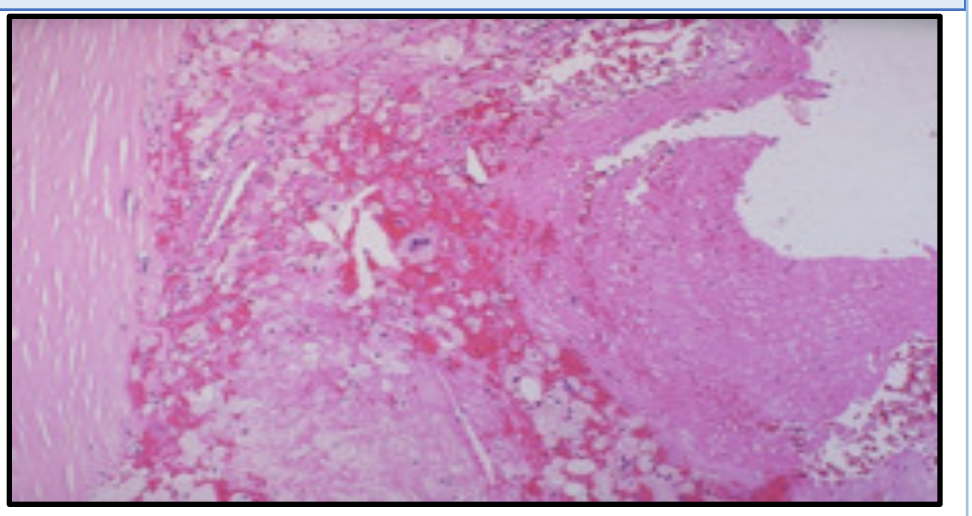
Coronary atherosclerosis - LPF



A normal coronary artery

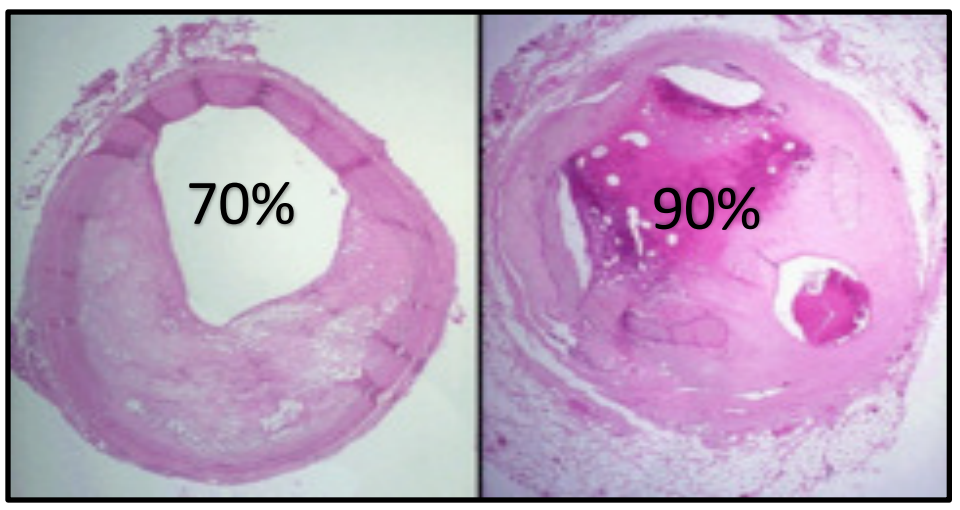
Coronary atherosclerosis - LPF

Coronary atherosclerosis - LPF



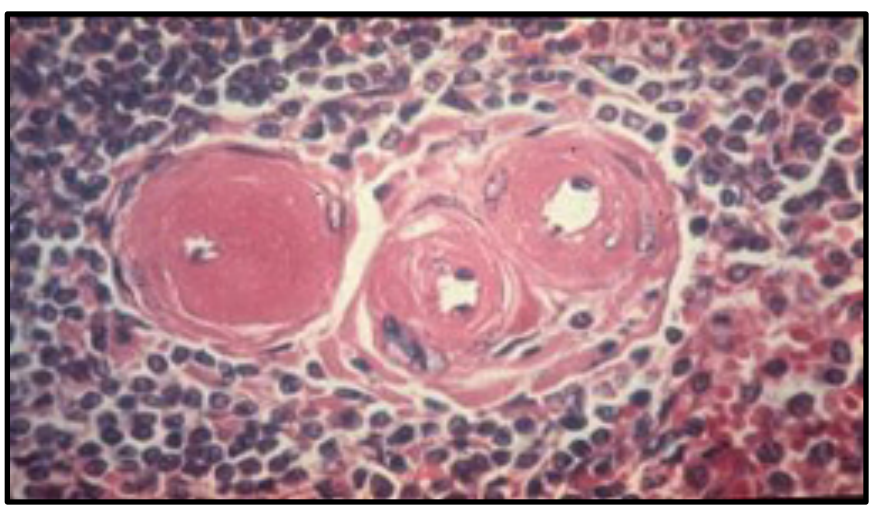
Atheromatous plaque in a coronary artery

Coronary atherosclerosis - MPF



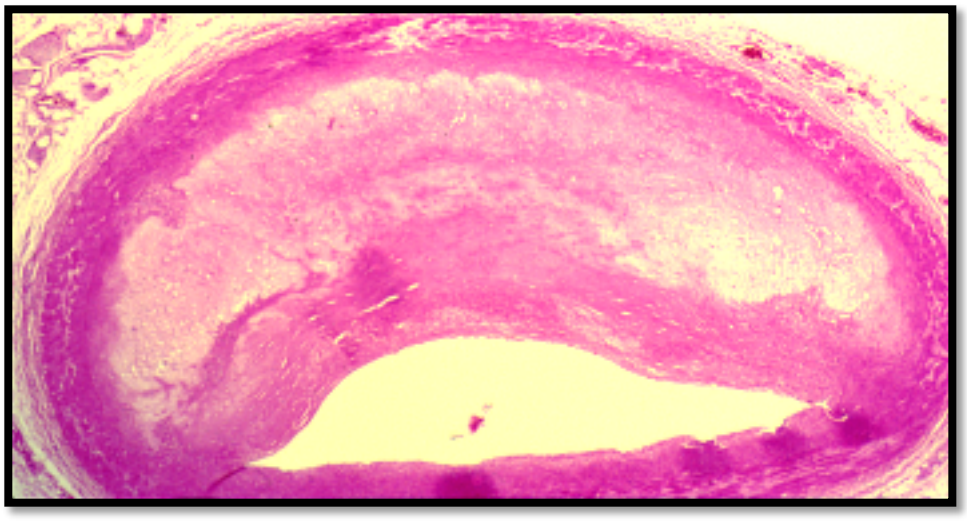
Occlusive coronary atherosclerosis.
Left narrowed by 70%, Right by 90%

Hyaline arteriolosclerosis - HPF



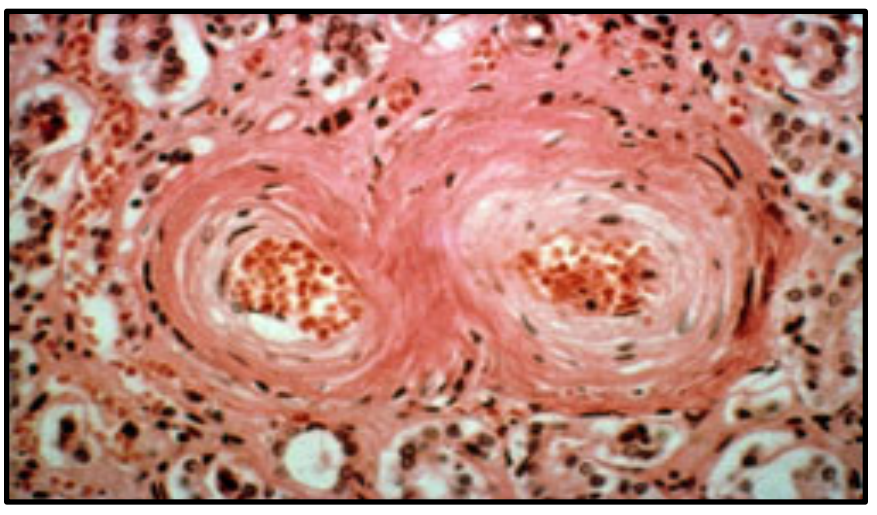
Hyaline arteriolosclerosis -> diabetics, hypertensive

Hyperplastic arteriolosclerosis - HPF



Severe coronary atherosclerosis with narrowing of the lumen

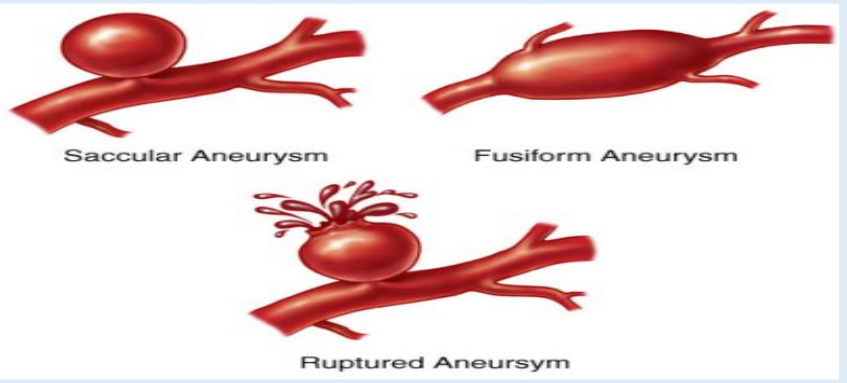
Hyperplastic arteriolosclerosis - HPF



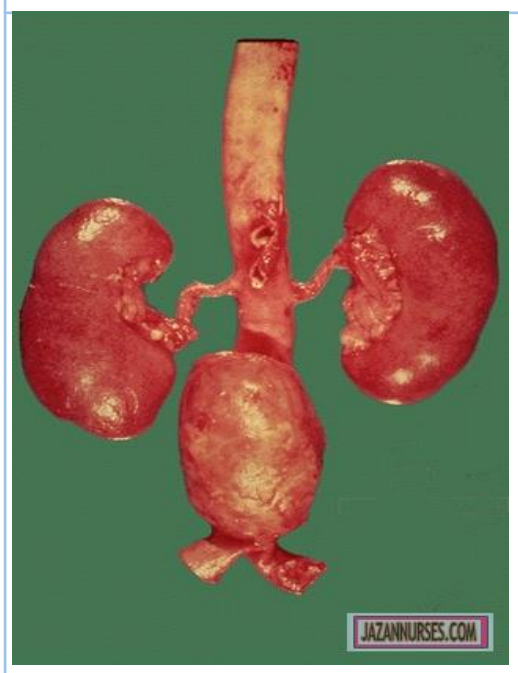
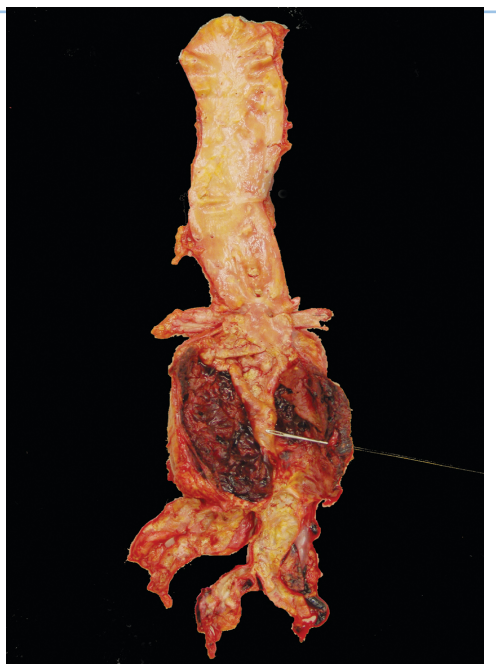
Hyperplastic arteriolosclerosis -> malignant hypertension

Case #3: Aneurysm of abdominal aorta

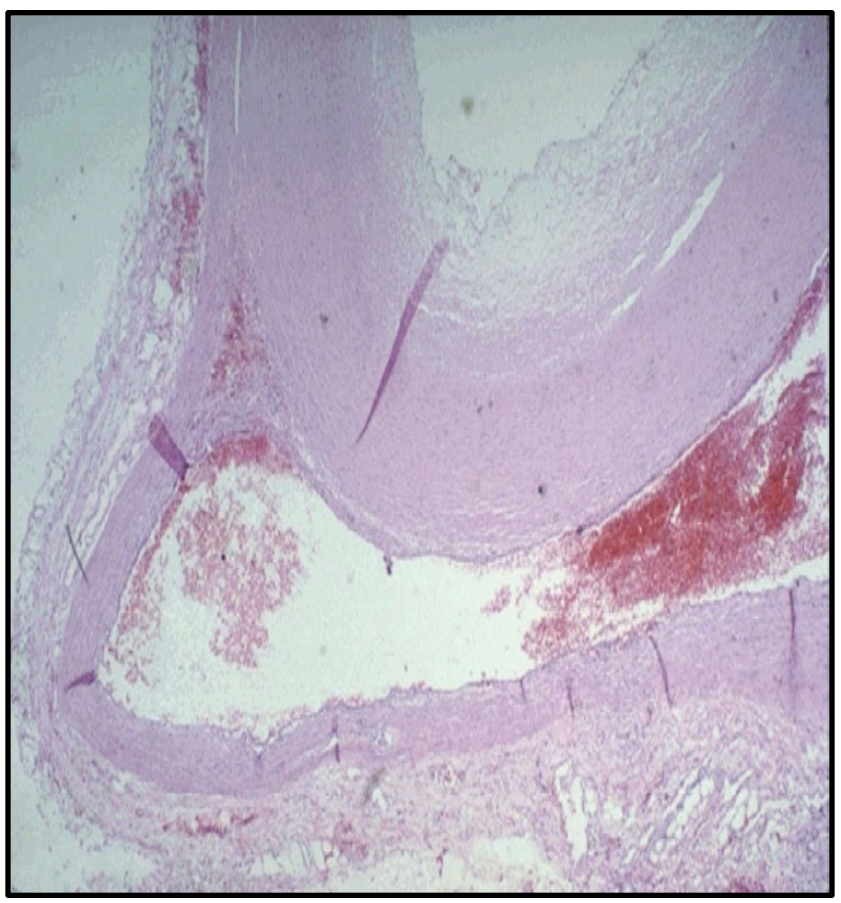
Theoretical Information's:



- Types of Aneurysms:
  1-Fusiform 2-Saccular 3- Raptured
- The most likely causes of aneurysms are:
  1- atherosclerosis -> common cause
  2-mycotic "fungal".
  3-syphilitic
  4-congenital
- Complication: Rapture of the aneurysm
- Secondary changes: Hemorrhage
- The patient suddenly develop severe abdominal pain, shock and collapse
- Dissecting aortic aneurysm : Usually associated with atherosclerosis, inflammation, and degeneration of the connective tissue of the tunica media

Abdominal Aortic Aneurysm	Abdominal Aortic Aneurysm
 atherosclerotic aneurysm of the aorta above the aortic bifurcation.	 - rupture ,intraluminal thrombus. Se

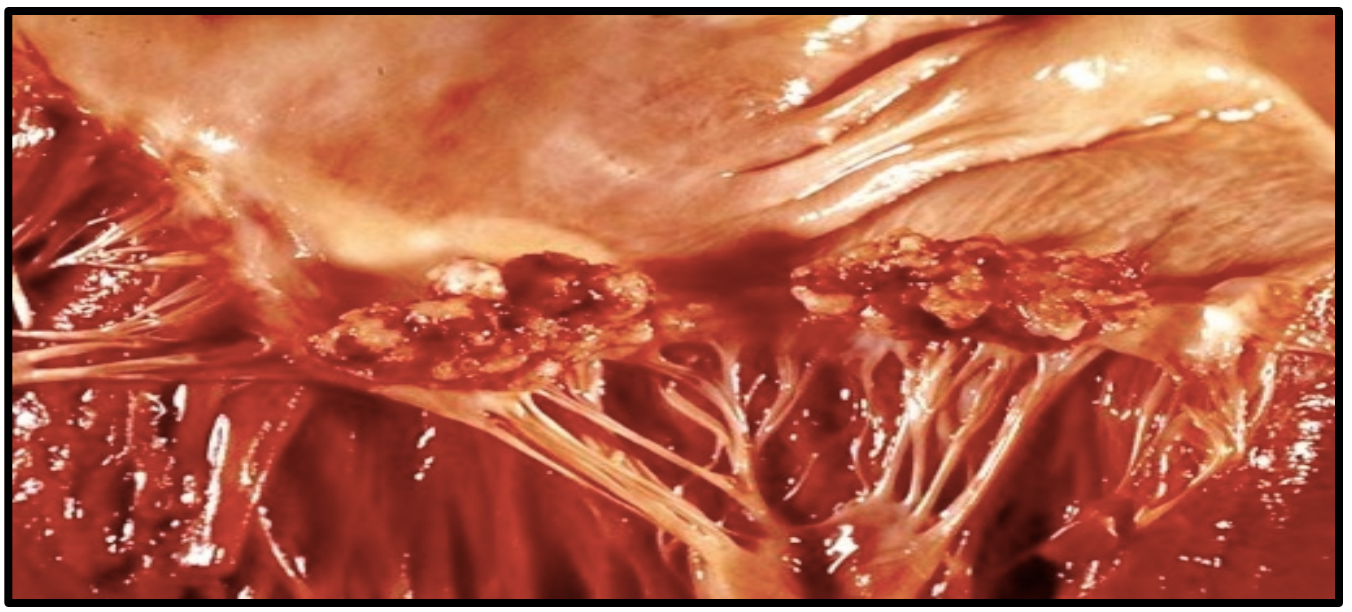
Dissecting aortic aneurysm – LPF autopsy finding.



A dissecting aortic aneurysm occurs when blood enters the aortic wall through a defect and moves between two layers of the wall, stripping the inner layer from the outer layer.

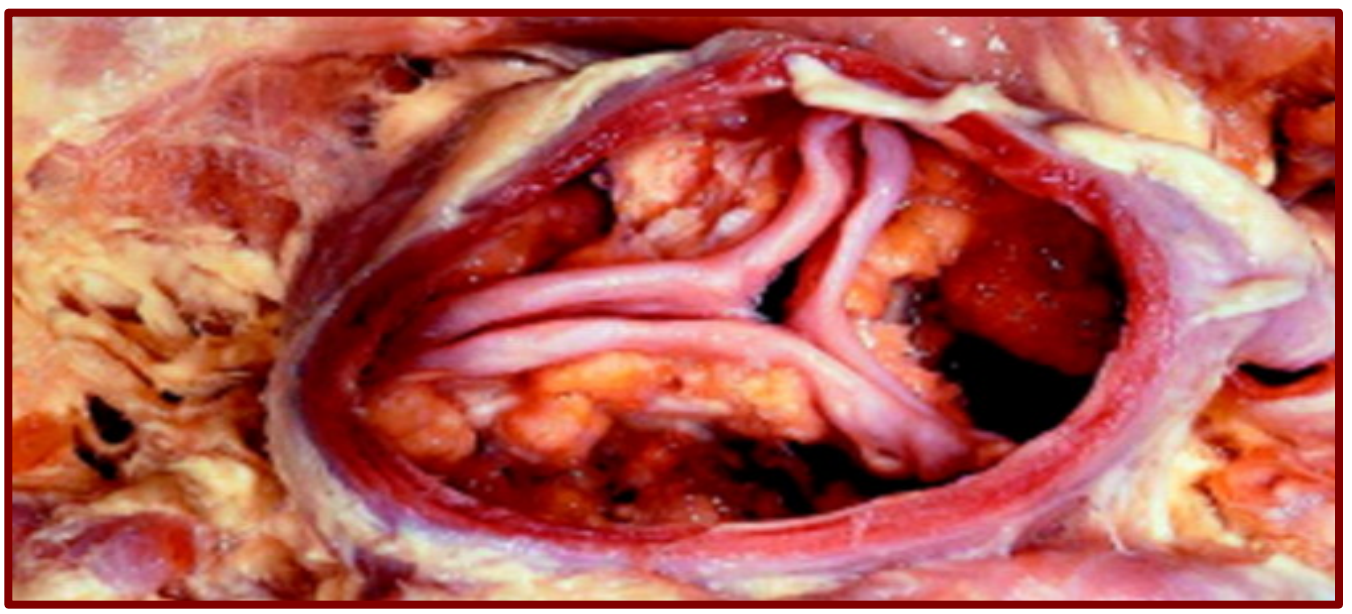
Case #4: Vegetations of rheumatic fever on mitral and aortic valves

Chronic Rheumatic Mitral Valvulitis - Gross



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

Rheumatic Aortic Valvulitis - Gross



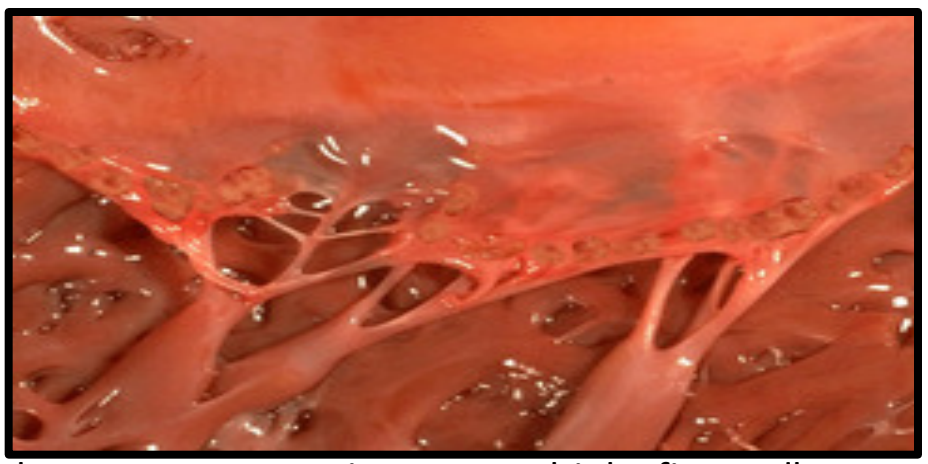
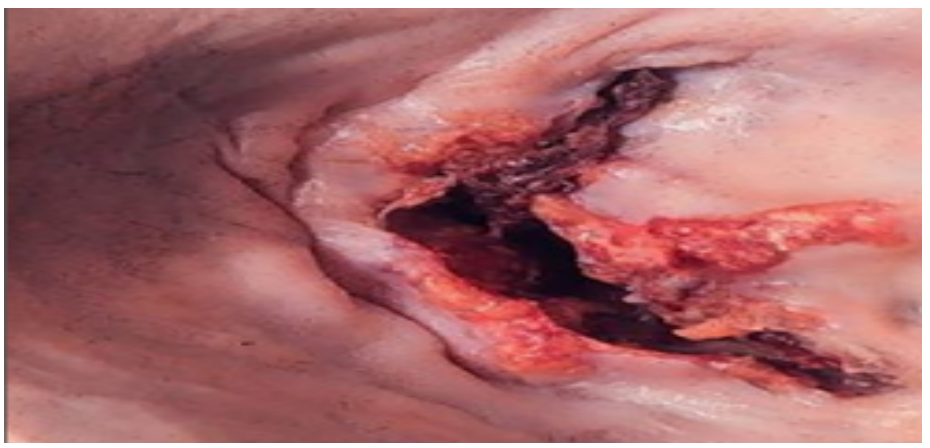
Aortic stenosis:
Aorta has been removed to show thickened, fused aortic valve leaflets

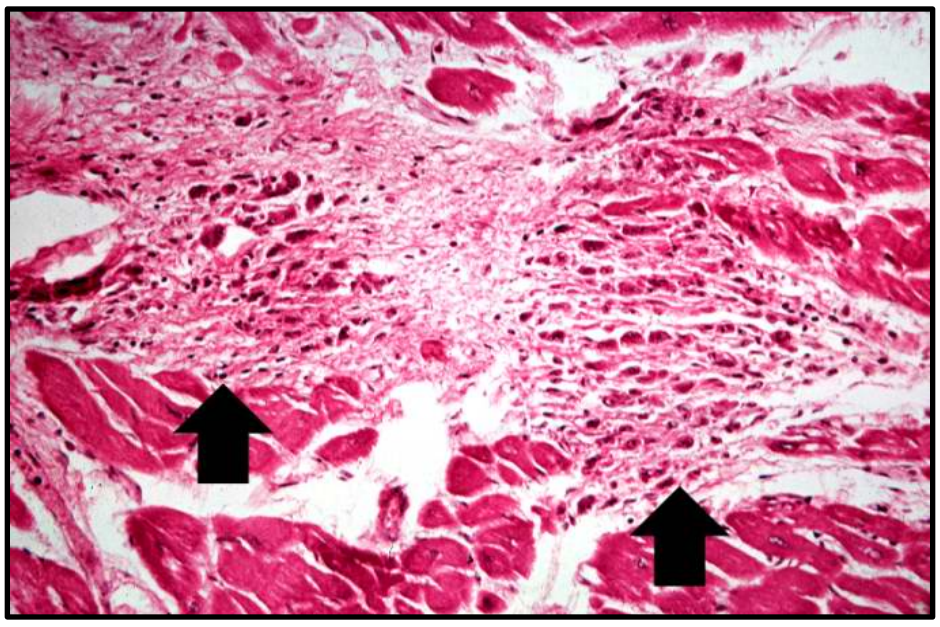
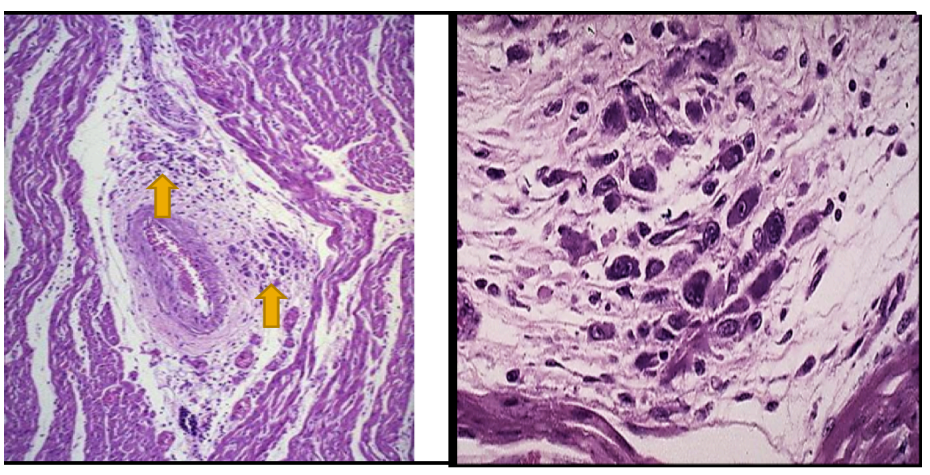
Case #5: Acute rheumatic myocarditis

Theoretical Information's:

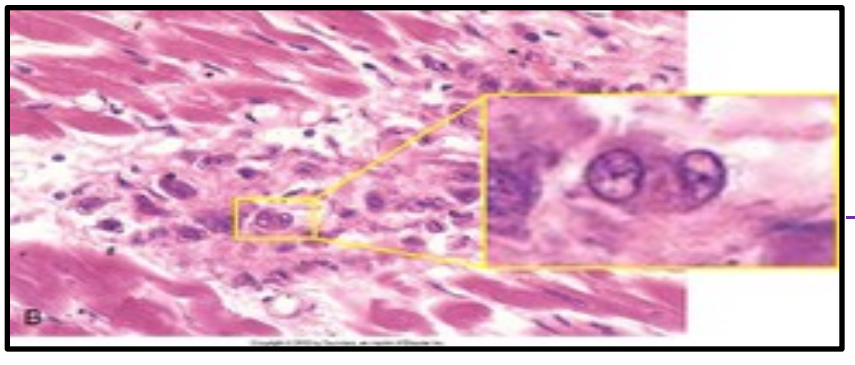
- Mitral stenosis secondary to rheumatic fever.
- Non-cardiac systemic manifestations rheumatic fever are:

-Arthralgia, Arteritis, Sydenham chorea, Erythema marginatum

Acute Rheumatic Mitral Valvulitis	mitral valve
 These warty vegetations are multiple, firm, adherent, small , 1-3 mm in- diameter and form along the line of valve closure over areas of endocardial inflammation	 - Fish mouse deformity. - Fusion of commissures. - Thickening and calcifications of cusps. - Vegetations.

Acute Rheumatic Carditis	(ASHOFF NODULE)
 "Aschoff nodules" seen best in myocardium	 Aschoff bodies in the intermuscular fibrous septa. They are oval in shape and seen in relation to blood vessels. - fibrinoid necrosis - few lymphocytes - anitschkow (Aschoff giant cell).

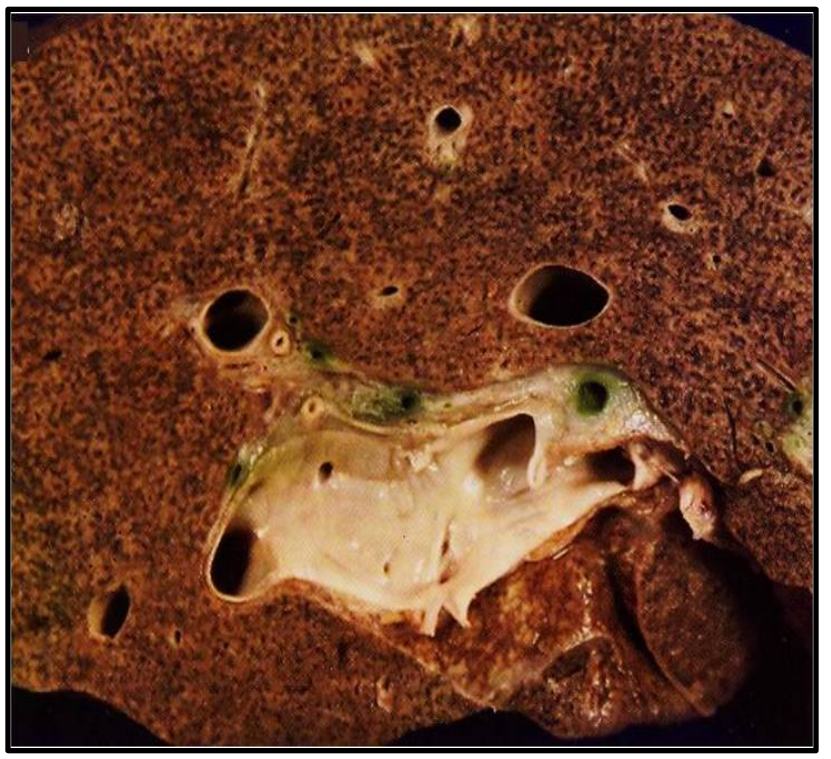
Ashoff nodule or Anistskow cell.



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

Case #6: Right Sided Heart Failure
Chronic venous congestion of the liver

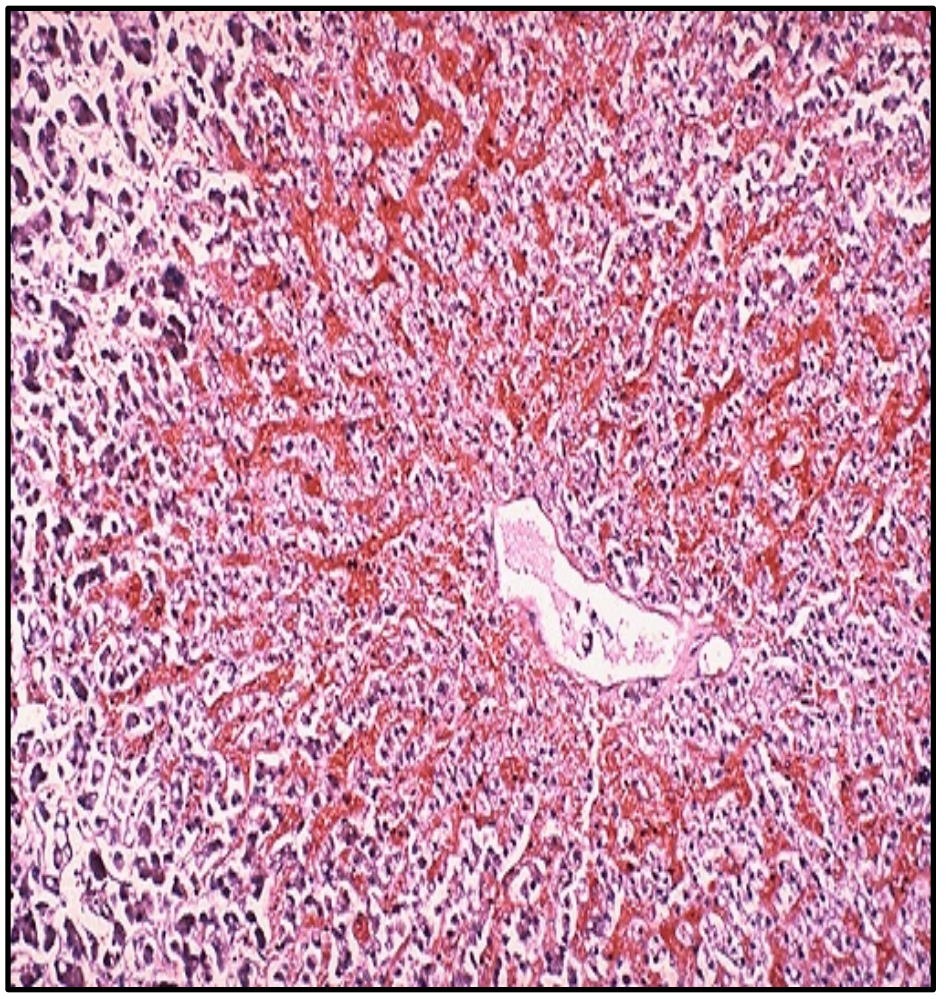
NUTMEG LIVER – Cut surface



alternating pale and dark areas with a **nutmeg like appearance** ,due to passive **congestion secondary to right sided heart failure.**

Nutmeg: Type of spice
Extra

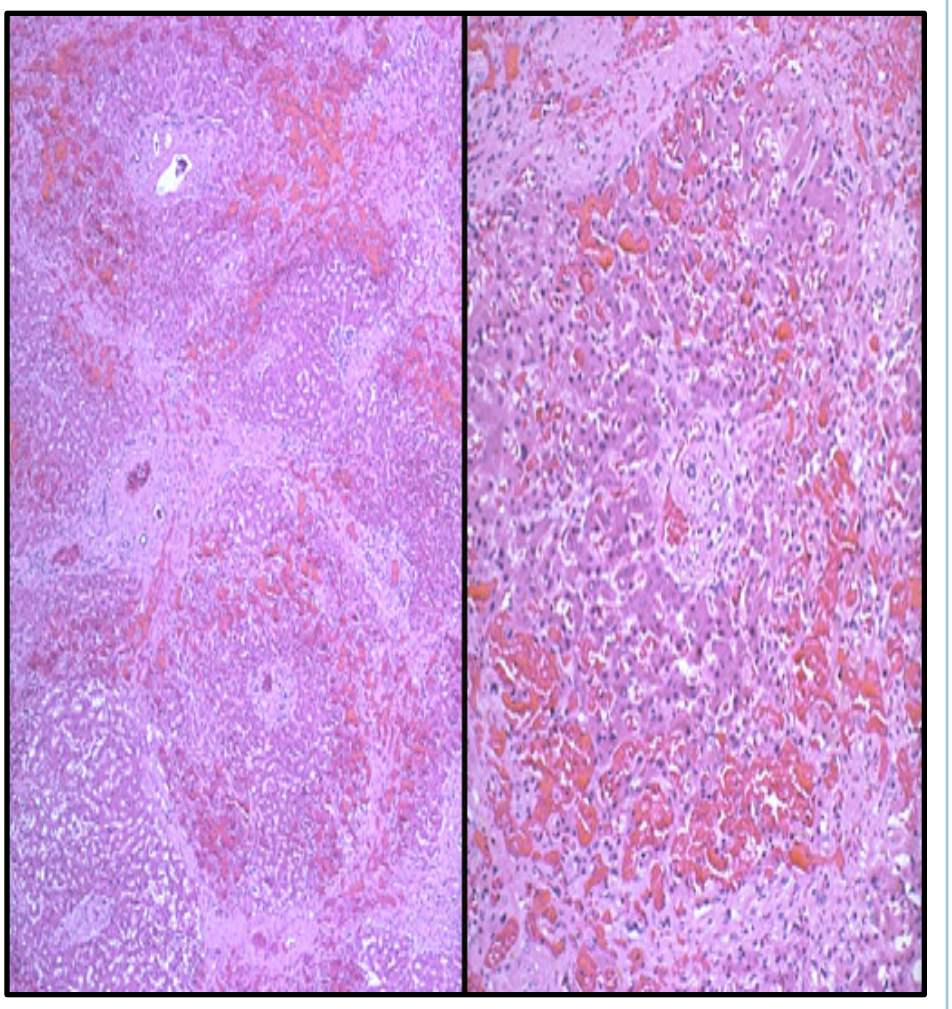
Chronic Congestion of the Liver - LPF



The central portion of liver lobules shows:

- Congestion and dilatation of central veins and blood sinusoids.
- atrophy and necrosis of liver cells.

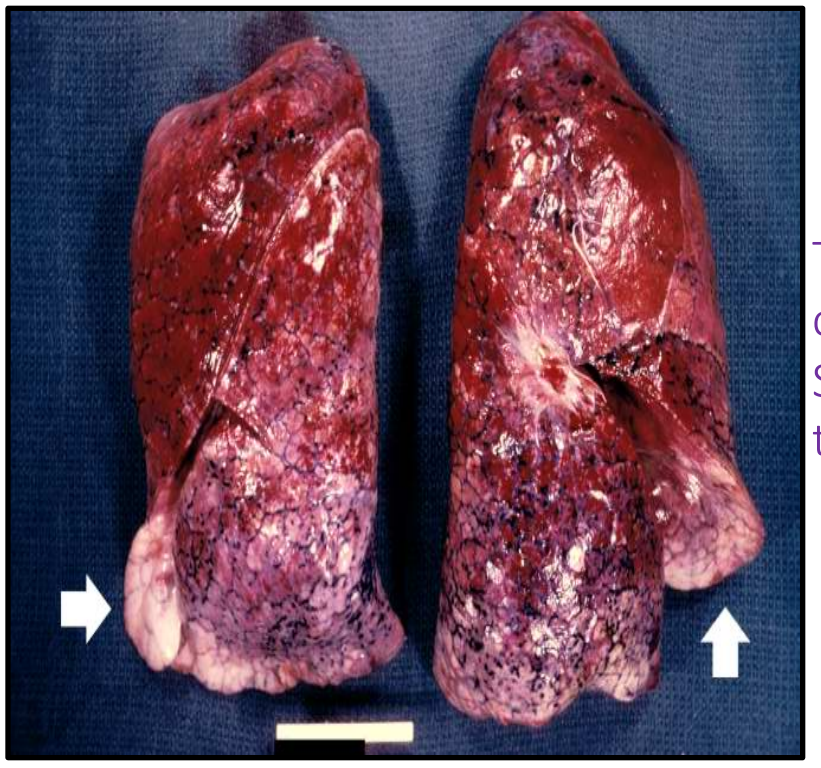
Chronic Congestion of the Liver - LPF



- Central veins dilated and congested
- necrotic hepatocytes

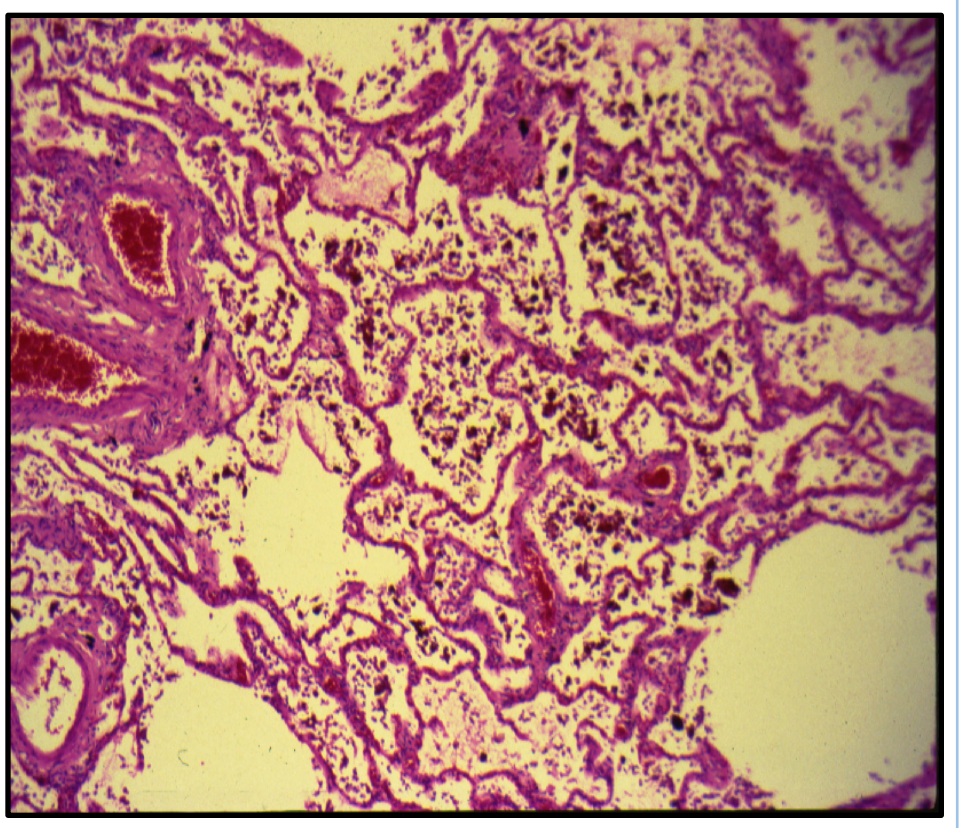
Case #7: Left Sided Heart Failure
Chronic venous congestion of the Lung

Chronic venous congestion of the lung - Gross



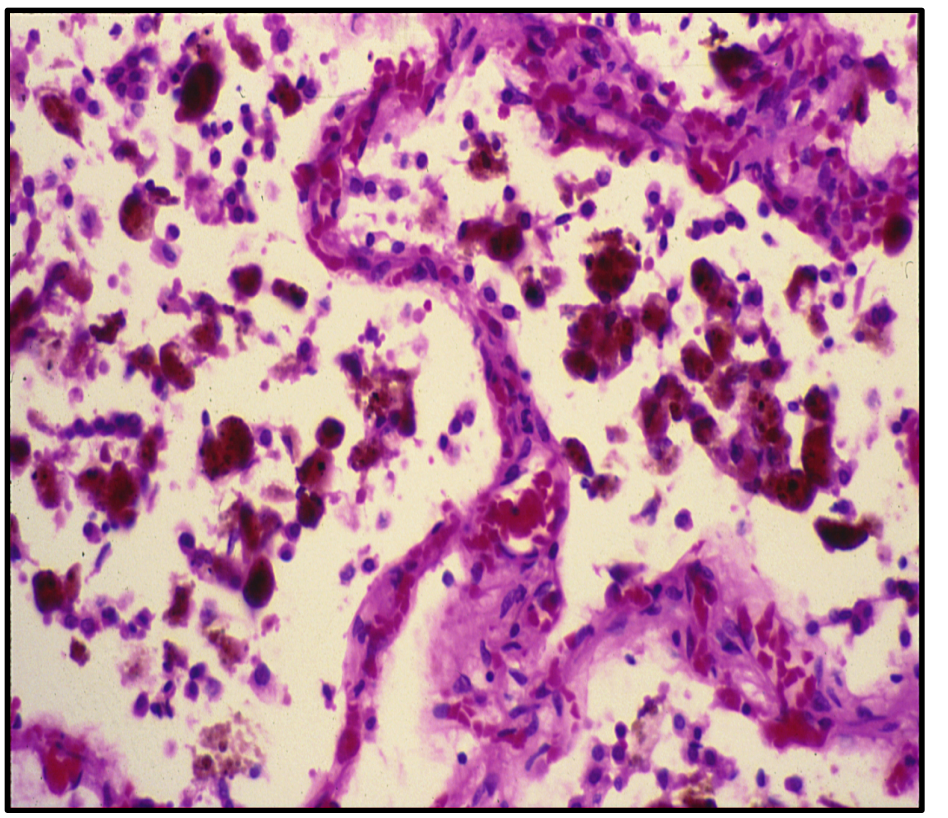
The reddish coloration of the tissue is due to congestion.
Some normal pink lung tissue is seen at the edges of the lungs (arrows).

Chronic venous congestion of the lung - LPF



The alveolar walls are thickened by dilated and engorged capillaries.
Engorged= full of RBC.

Chronic venous congestion of the lung - HPF



-Heart failure cells: Macrophages
- Congested RBC

Case #8: MYOCARDIAL HYPERTROPHY

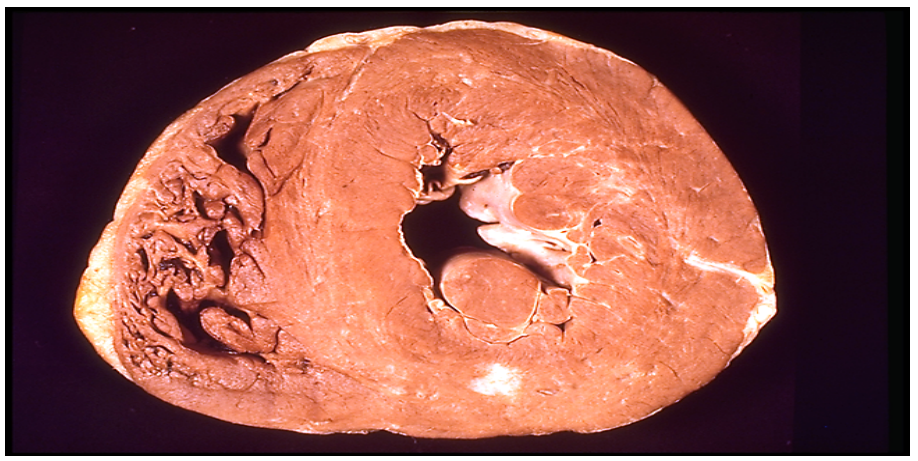
Theoretical Information's:

- The ventricle is working against high pressure, or "pumping" higher than normal volume leading to myocardial hypertrophy.
- Causes of Hypertrophy:

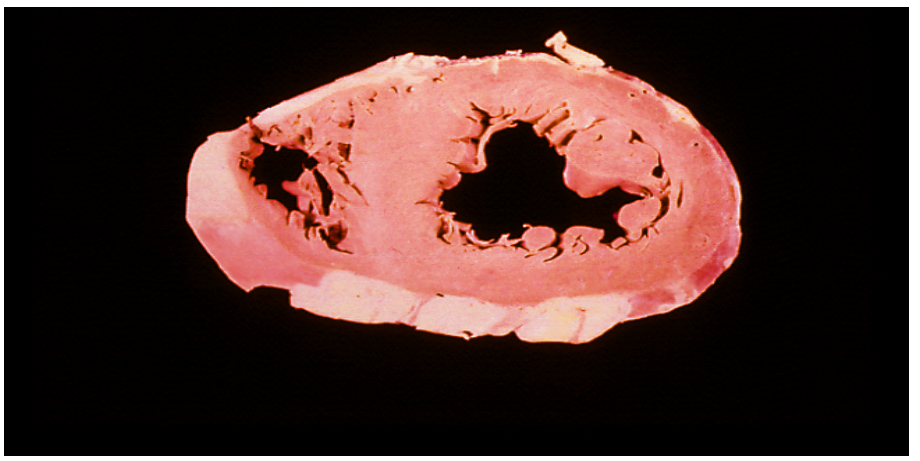
  Left ventricular hypertrophy: Systemic hypertension, Aortic valve stenosis.

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

Normal and hypertrophied left ventricle – cross section

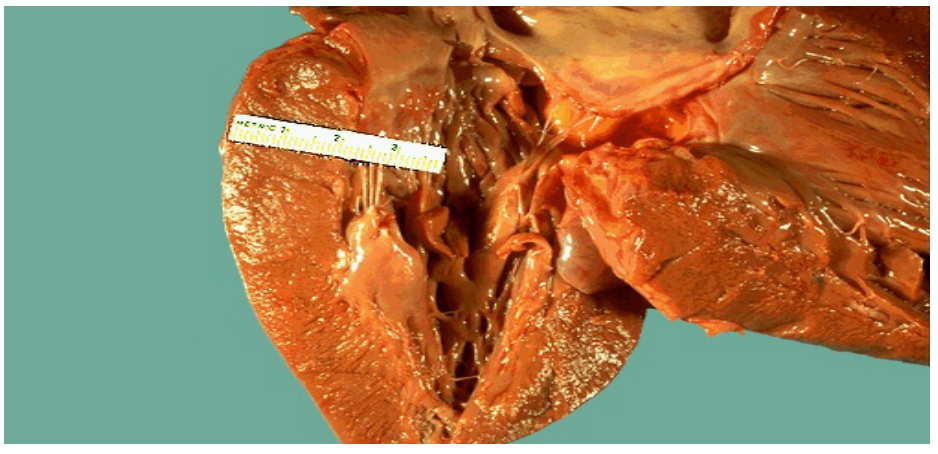


Left ventricular hypertrophy



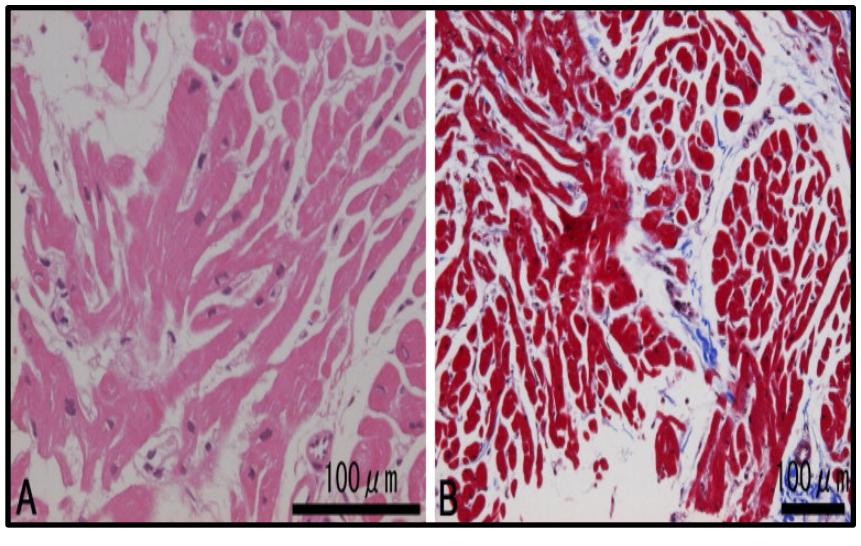
Normal ventricles

Left ventricular hypertrophy - Gross



Heart from a hypertensive patient showing
More than 2cm thickening.

Hypertrophic Cardiomyopathy - LPF



haematoxylin-eosin stain *Masson's trichrome stain*

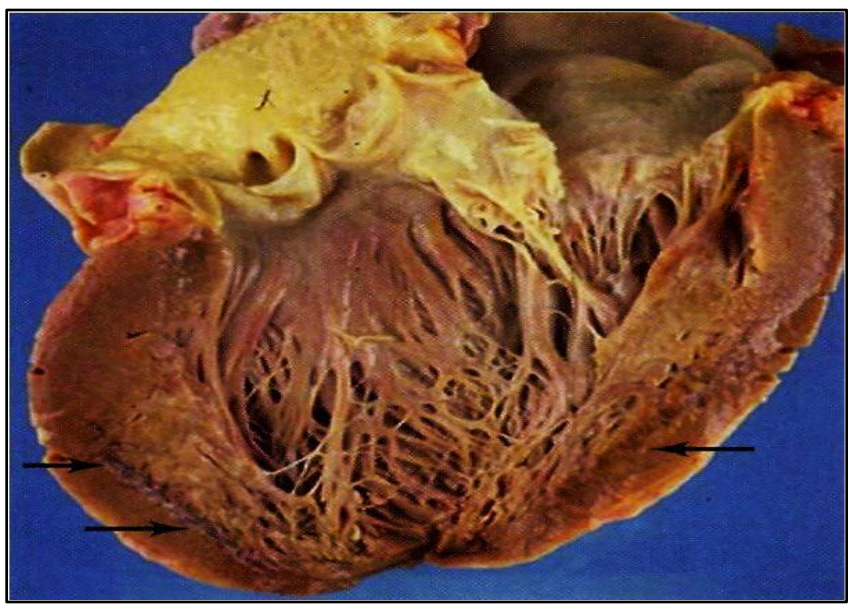
- significant myofiber disarray
- slight interstitial fibrosis indicating hypertrophic cardiomyopathy (HCM).

Case #9: Myocardial Infarction

Theoretical Information's:

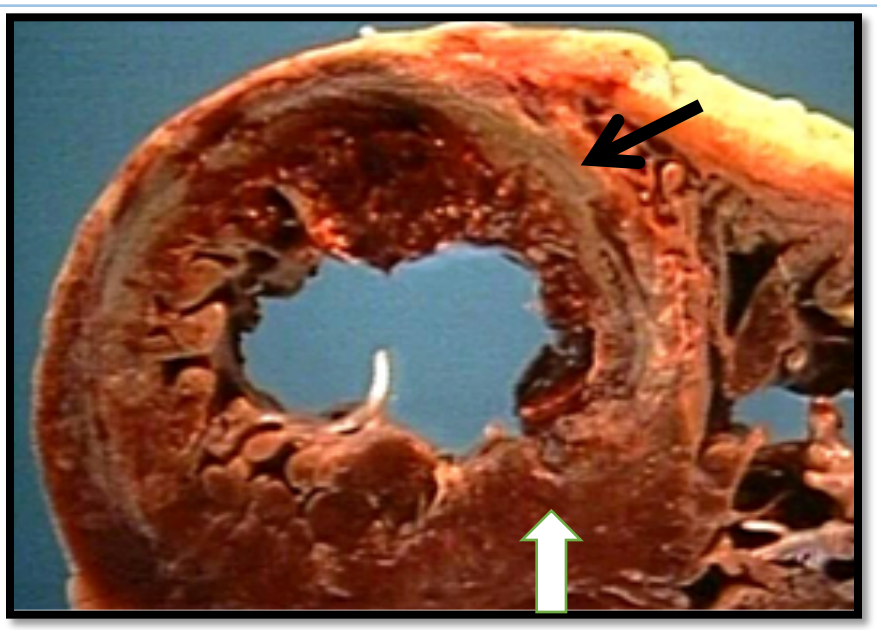
- Acute myocardial infarction can be complicated by:
  - Cardiac Arrhythmias, MOST COMMON CAUSE.
  - Myocardial rupture and haemopericardium.
  - Ventricular aneurysm.
  - Heart failure.
  - Mural thrombosis.
- Serum enzyme or protein that is elevated 24 hours after the patient's admission to hospital:
  - CK-MB - Troponin I
- The cause of these pathology can be:
  -Chronic ischemic heart disease, Long standing hypertension and/or left ventricular failure.
- Death in these patient can be due to complications secondary to acute myocardial infarction

Myocardial Infarction - CS



arrows pointing at infracted areas

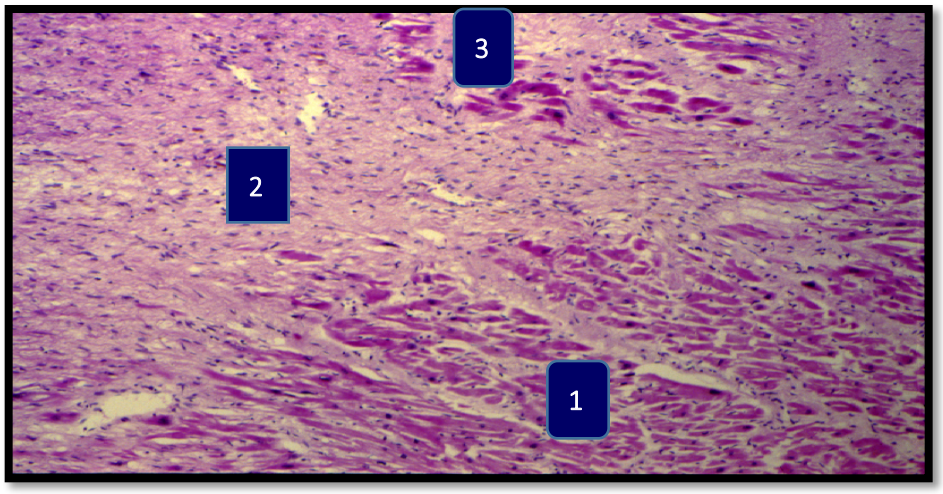
Myocardial Infarction - CS



a- Pale and irregular myocardial fibrosis(Black arrow) Caused by :atherosclerosis

b- Thick left ventricular wall. (white arrow) Caused by: Hypertension

Myocardial Infarction – late stage

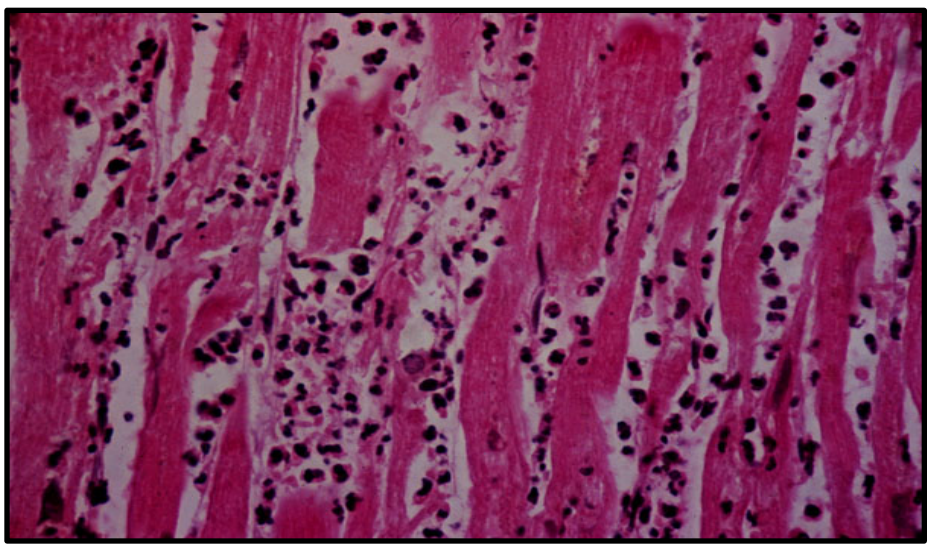


1- Patchy coagulative necrosis of myocardial fibers.
The dead muscle fibers are structureless and hyaline with loss of nuclei and striations.

2- Chronic ischemic fibrous scar replacing dead myocardial fibers .

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

Acute Myocardial Infarction



This 3-4 day old infarct showing:

- Necrotic myocardial fibers.
- Infiltration by polymorphonuclear leukocytes.

Case #10: Thromboangitis oblitrans (Buerger's disease)

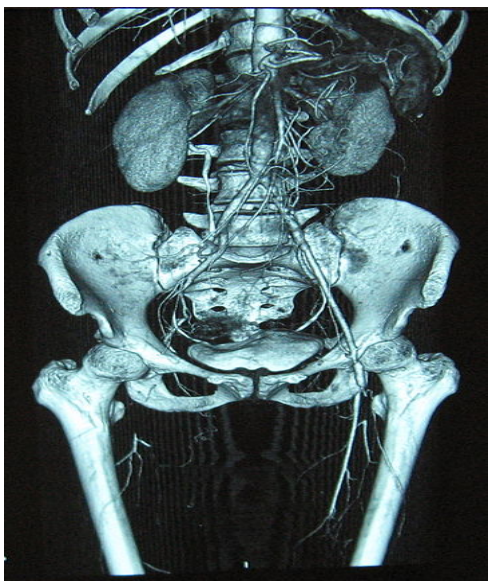
Theoretical Information's:

- Black discoloration of the patient's finger and toes caused by: ischemia
- The main predisposing factors for this condition are:
  - Smoking habits.
  - Certain HLA haplotypes (Genetic predisposition).
- Pathologic findings of an acute inflammation and thrombosis (clotting) of arteries and veins of the hands and feet (the lower limbs being more common)

Thromboangitis oblitrans (Buerger`s disease)

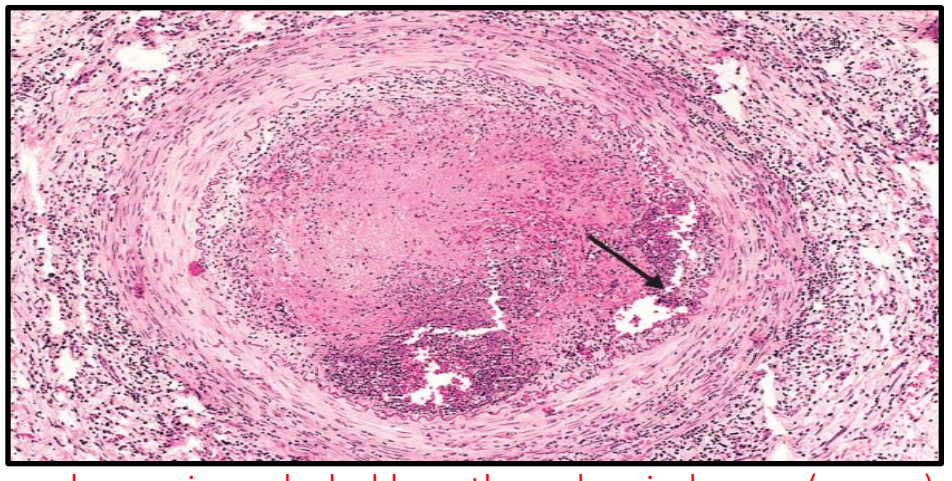
Black discoloration of the patient's finger and toes

Thromboangitis obliterans (Buerger's disease)



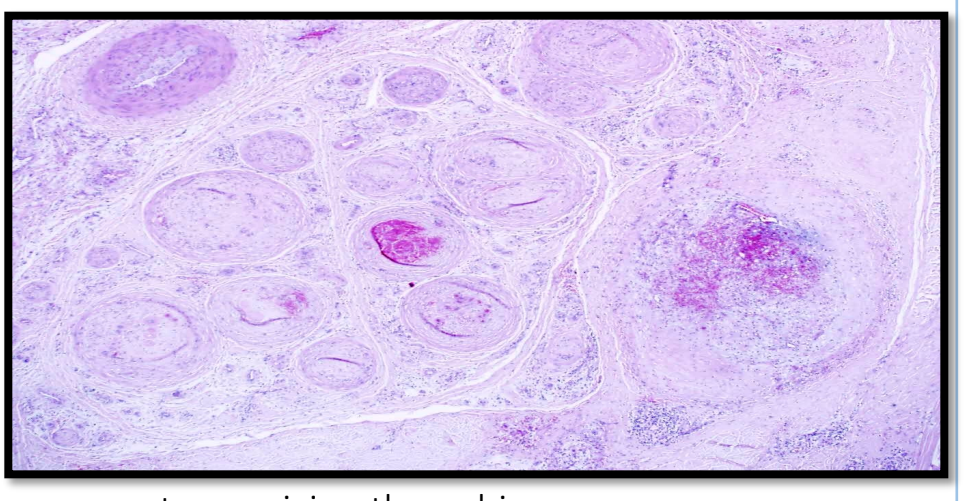
Complete occlusion of the right and stenosis of the left femoral artery

Thromboangitis obliterans (Buerger's disease) – HPF



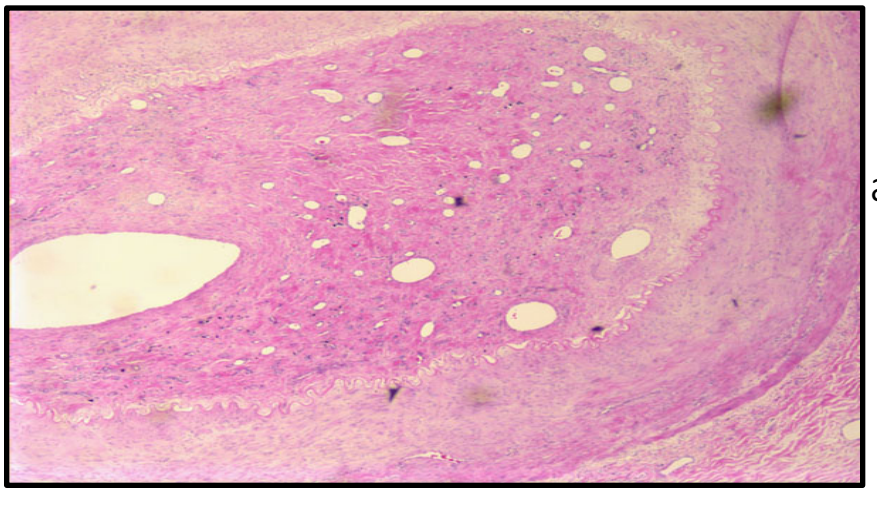
- lumen is occluded by a thrombus in lumen (arrow)
- vessel wall is infiltrated with leukocytes.

Thromboangitis obliterans (Buerger's disease) – HPF



- recent organizing thrombi
- infiltration of the wall and surrounding tissue by chronic inflammatory cells

Thromboangitis obliterans (Buerger's disease) - LPF



- (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

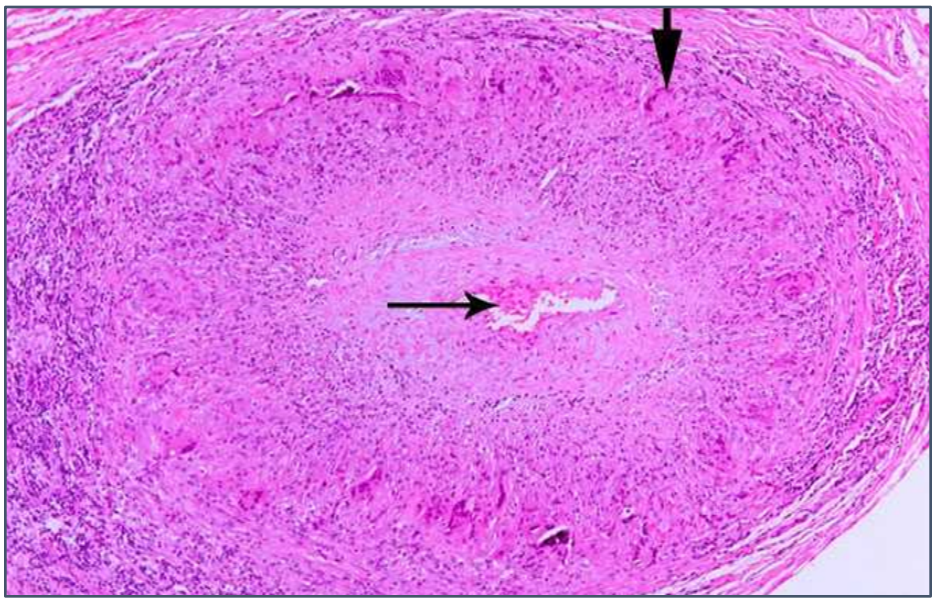
Case #11: Giant cell (temporal) arteritis

Theoretical Information's:

- Elevated erythrocytes sedimentation rate (ESR) is raised in these patients.
- Complication: Blindness because of involvement of ophthalmic artery.
- Reactive intimal fibroplasias lead to luminal stenosis with <10% of its original luminal diameter (thin arrow in the center).

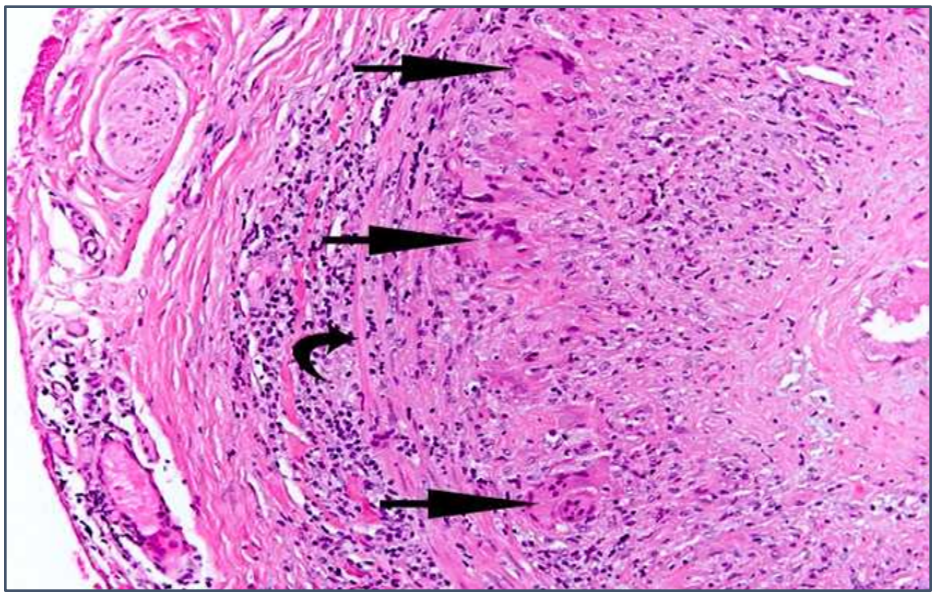
Tender and thickened temporal artery

GIANT CELL / TEMPORAL ARTERITIS - LPF



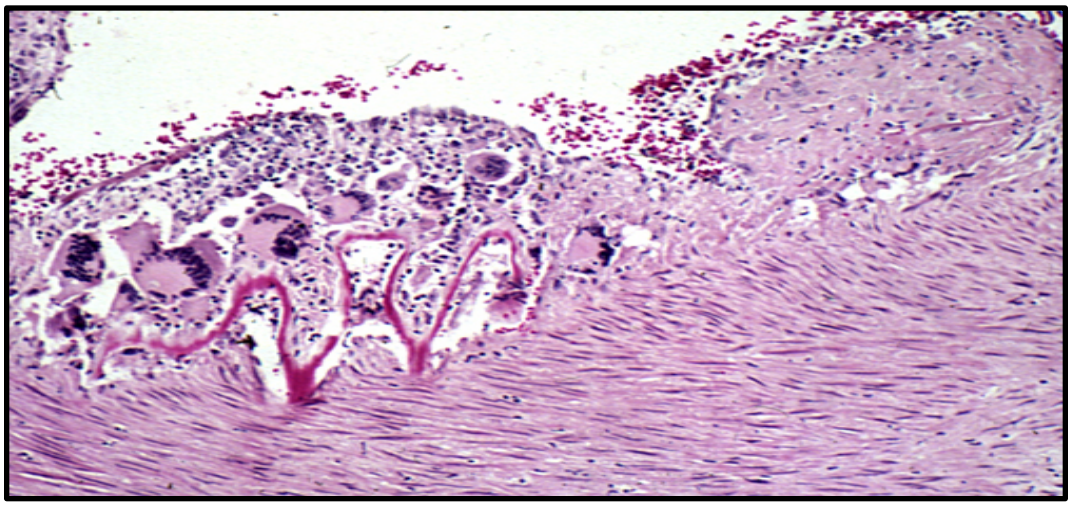
- Circumferential involvement of the vascular media is present (vertical arrow pointing downward).
- chronic lymphocytic inflammation in the media and adventitia.

GIANT CELL / TEMPORAL ARTERITIS - HPF



Giant cells can be of Langhans type or foreign-body type (three arrows) and may show fragments of disrupted internal elastic lamina..

GIANT CELL / TEMPORAL ARTERITIS – HPF

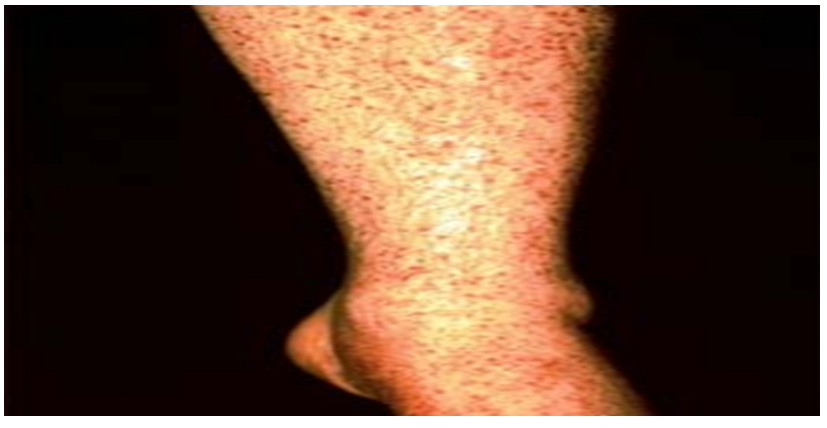


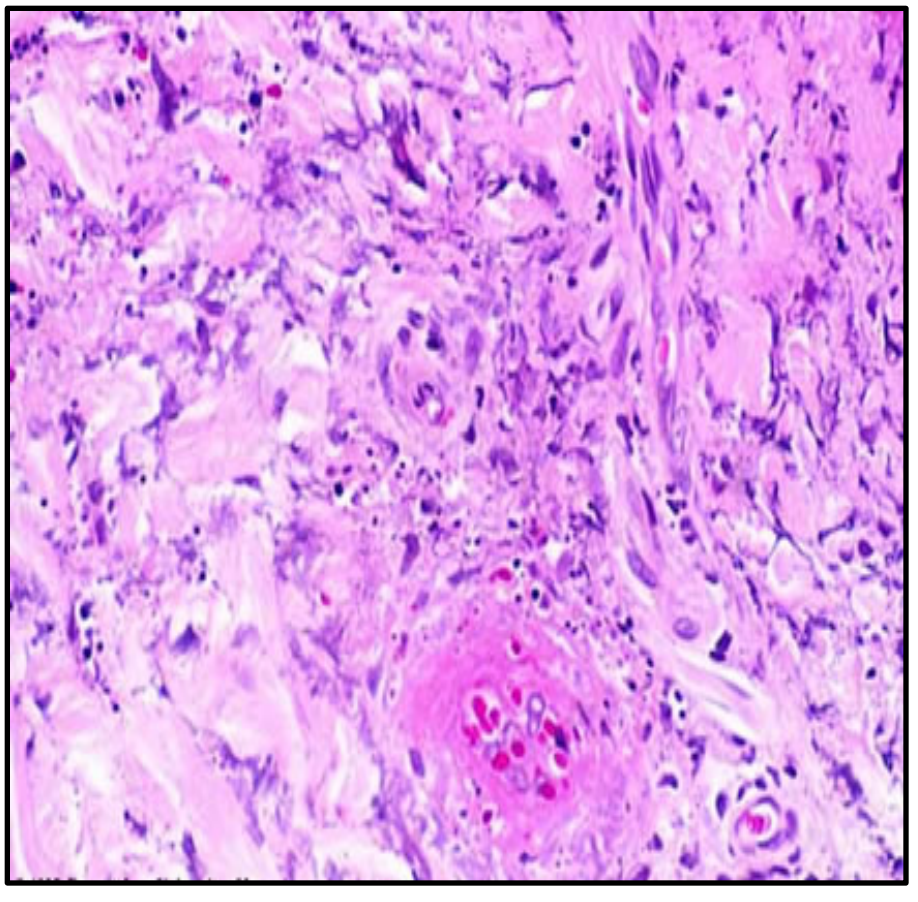
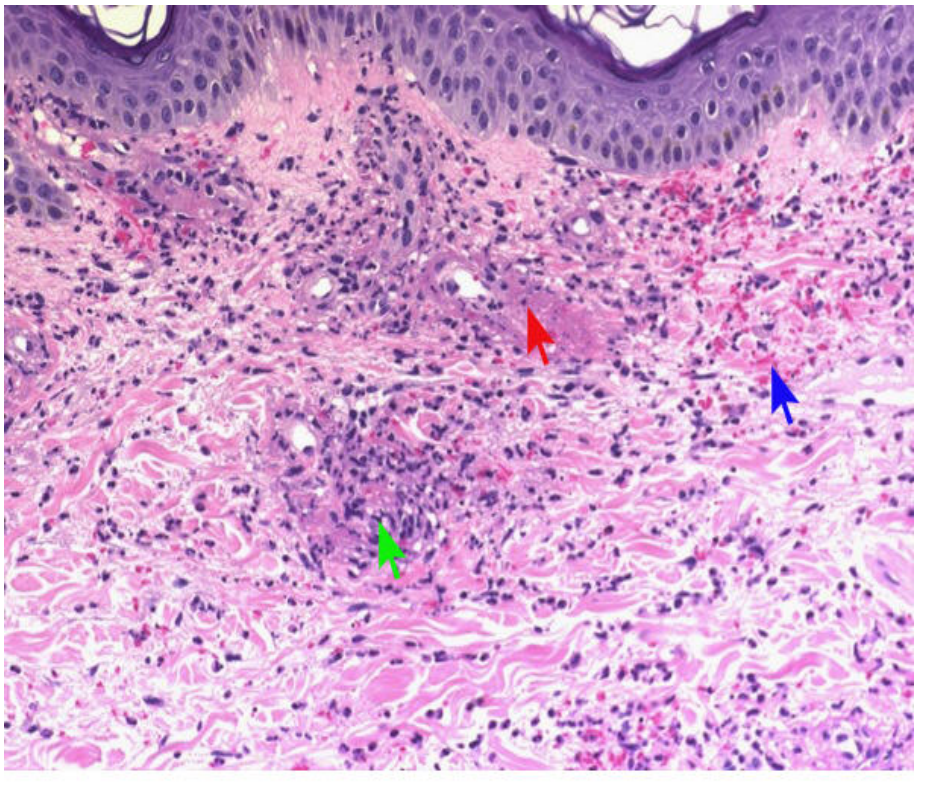
- Chronic inflammation.
- Giant cells.
- Fragmentation of the vascular internal elastic lamina.
- Granulomatous inflammation.

Case #12: Leukocytoclastic vasculitis/ hypersensitivity vasculitis / microscopic polyangitis / Henoch- Schönlein purpura "All of the four names are the same disease".

Theoretical Information's:

- Complications that might occur as a result of this condition.
  - Necrotizing Glomerulonephritis.
  - Pulmonary capillaritis.
  - Gastrointestinal vasculitis.
  - CNS and muscle involvement.

Hypersensitivity vasculitis – Clinical sign	Leukocytoclastic vasculitis - Clinical sign
	
Hypersensitivity vasculitis might be complicated with glomerulonephritis and hemoptysis due to pulmonary capillaritis Blood vessels: small vessels " capillaries "	Erythematous and purpuric skin rash affecting the right foot (Purpura:- Subcutaneous bleeding.)

Leukocytoclastic vasculitis - HPF	Leukocytoclastic vasculitis - HPF
	
-Fibrinoid vascular necrosis. - Nuclear debris. - Neutrophilic (polymorphonuclear) infiltration.	Fibrinoid type necrosis Red cell extravasation Inflammation

Important notes:

- Red notes are covering 95% of what's coming in the exam.
- 4 cases each with a scenario, histopathology description, one theoretical based question " You can find them in the blue text box", Gross or X-ray pictures depends on the case

Thank you for checking our work.

This work was done by:

- Munerah alOmari
- Shamma alSohaily
- Amjad alDuhaish
- Malak alShareef

- For any corrections or suggestion contact us on:

PathologyP435@gmail.com