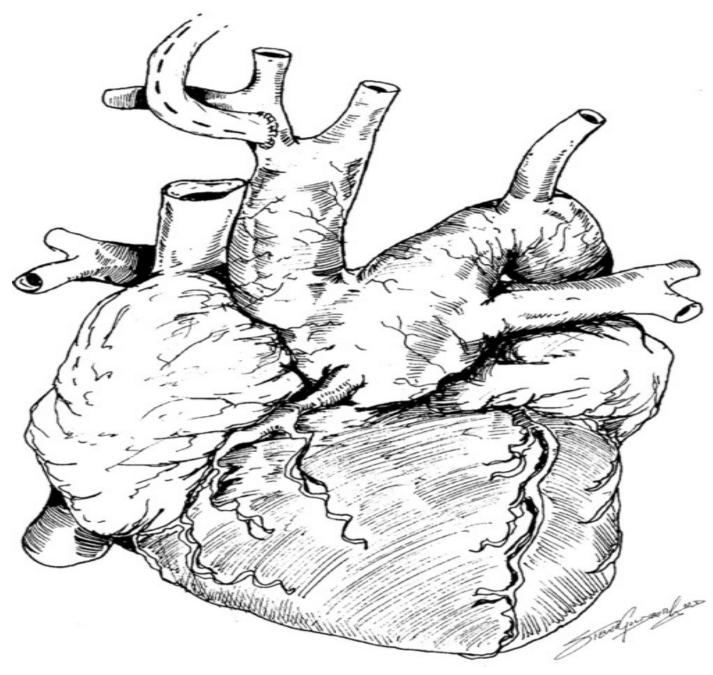


Pathology Practical Cardiovascular block Team 435



Color coding: Dr Marei's notes: Blue Dr shaesta's notes: Purple Both doctors notes "most imp": RED (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:

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

Arteriolosclerosis

affects small arteries and arterioles.

Hyaline -> diabetes mellitus.

Hyperplastic -> Hypertension

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

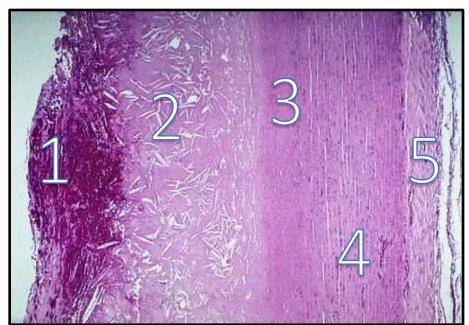
Theoretical Information's: An atheroma: an accumulation and swelling in artery walls made up of macrophage cells, or debris, and containing lipids (cholesterol and fatty acids), calcium and a variable amount of fibrous connective tissue. **Risk factors:** Male gender, Post menopausal woman, Hyperlipidemia, 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 Atheroma of the Aorta - Gross Atheroma of the Aorta - Gross atherosclerosis Gross 1-atheromatous plaques Severe atherosclerosis of the From bottom to top: "orange arrow". aorta, ulceration. 1- Mild: shows only scattered lipid 2-ulceration and haemorrhage

plaques. "fatty streaks" 2- Moderate: shows many more larger plaques 3- Sever: shows extensive ulceration in the plaques.

Atheroma of the Aorta - LPF

" blue arrow"



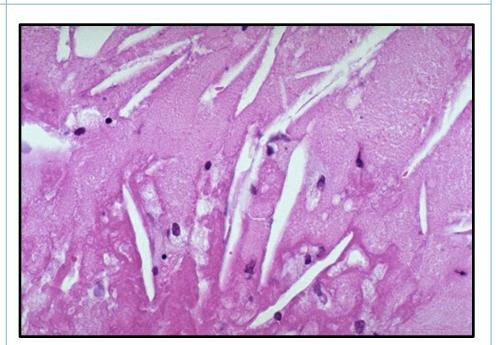


Left to the Right side are:

- 1- Haemorrhage
- 2- Atheromatous plaque containing cholesterol clefts
- 3- Fibrosis

_

- 4- Elastic Media
- 5- Adventitia



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

Case #1: Atheroma of the aorta only in Dr Marie's lecture

Atheroma of the Aorta - Gross

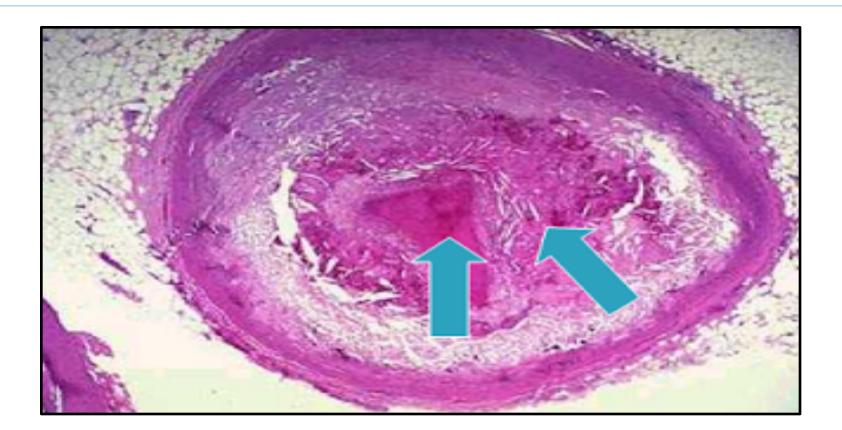
Atheroma of the Aorta - Gross

Atheroma of the Aorta - Gross



These picture are only found in Dr Marie's for more understanding. all have the same description which is Atheroma Plaques. Middle one shows fatty streaks and Dystrophic calcification

Atheroma of the Aorta - MPF



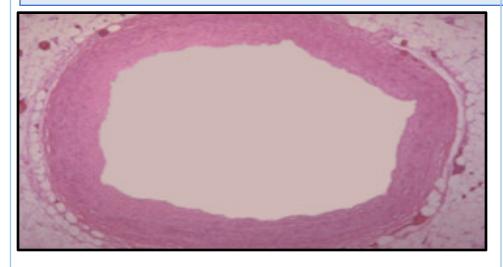
Aortic Atheroma with Thrombosis

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.

Coronary atherosclerosis - LPF Coronary atherosclerosis - LPF Arteriolosclerosis -> small arteries, atherosclerosis -> Medium to large arteries.

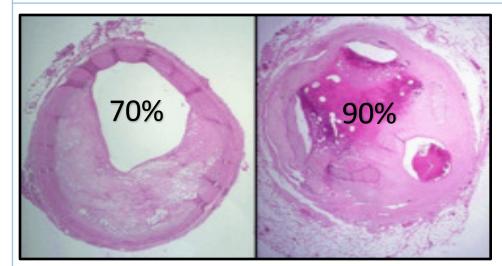


A normal coronary artery

Coronary atherosclerosis - LPF

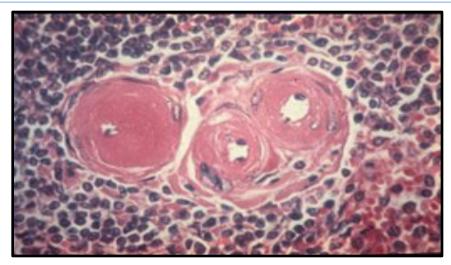
Coronary atherosclerosis - MPF

Atheromatous plaque in a coronary artery



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

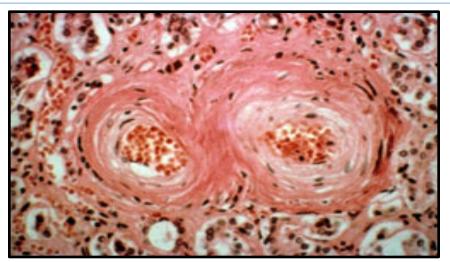
Hyaline arteriolosclerosis - HPF



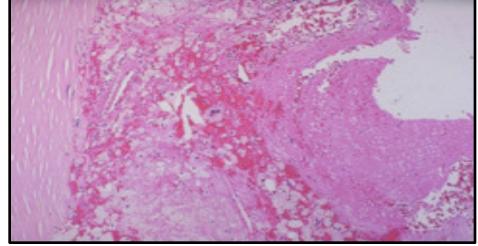
Hyaline arteriolosclerosis -> diabetics, hypertensive

Severe coronary atherosclerosis with narrowing of the lumen

Hyperplastic arteriolosclerosis - HPF

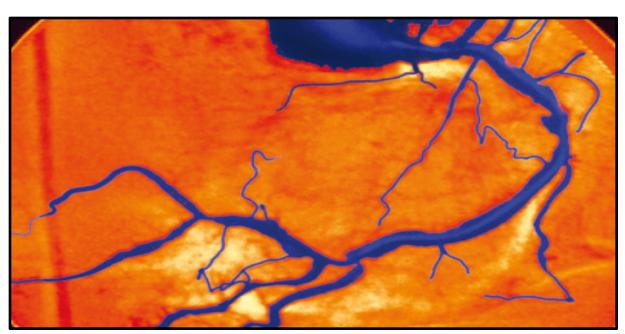


Hyperplastic arteriolosclerosis -> malignant hypertension



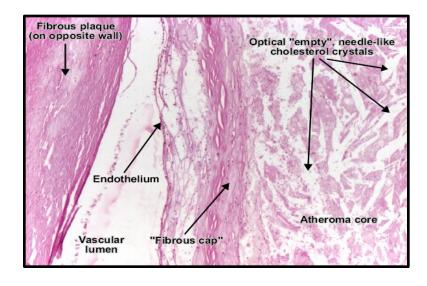
Case #2: Coronary atherosclerosis only in Dr Marie's lecture

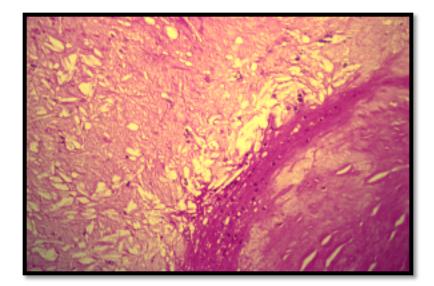
Coronary Atherosclerosis - Gross

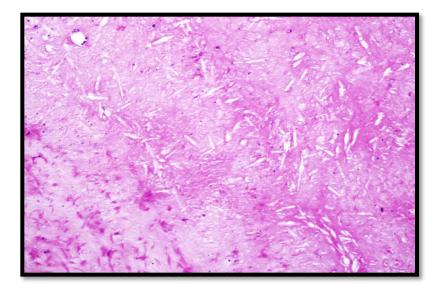


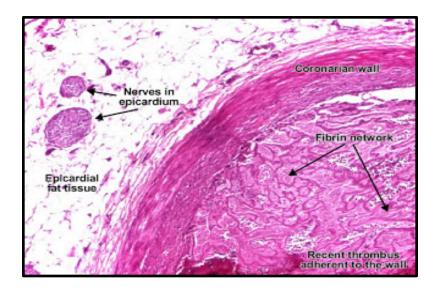
Colored angiogram (X-ray)

atherosclerosis is seen as the pinching in the blue- colored artery at bottom center



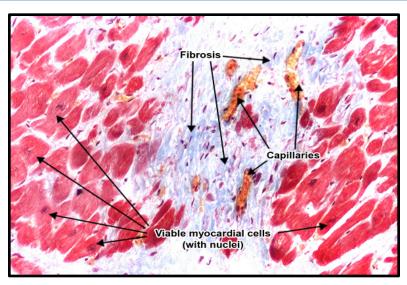






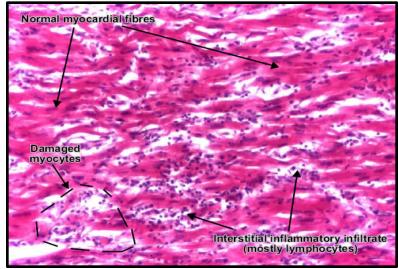
Complications of atheroma only in Dr Marie's lecture

Ischemic fibrosis of myocardium (diffuse ventricular myocardial fibrosis)



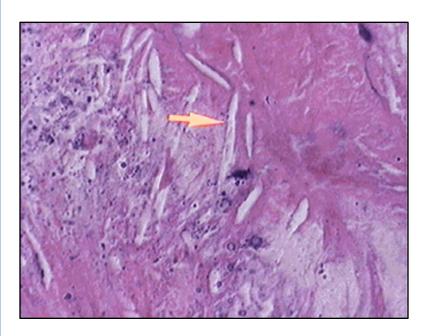
Diffuse myocardial fibrosis (Ischemic fibrosis of the myocardium) Myocardial cells (red) intermingled with collagen-rich fibrosis (blue) which completely replaced the necrotic myocardial cells. Capillaries (with yellow-orange red blood cells) within fibrosis remained from repair by connective tissue process

Acute viral myocarditis - MPF



Myocarditis is an inflammation of the myocardium. Acute viral myocarditis is produced most often by Coxsackie B virus and echoviruses. Myocardial interstitium presents an abundant edema and inflammatory infiltrate, mainly with lymphocytes and macrophages.

Aortic atherosclerosis - HPF



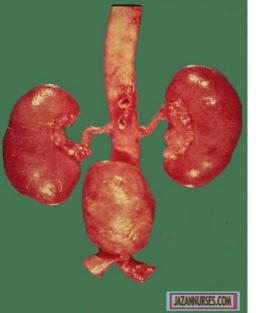
High power view of intimal aspect of atherosclerotic plaque showing stippling by blue calcific spherules, cholesterol crystal clefts, and fibrous cap.

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

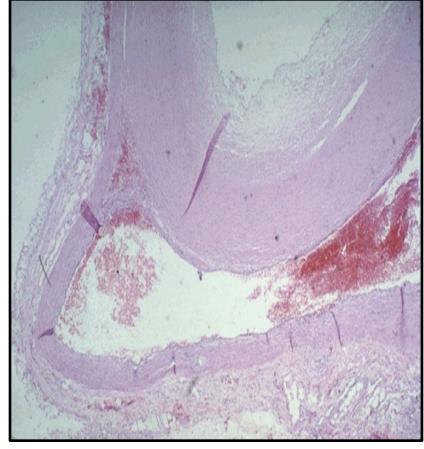


atherosclerotic aneurysm of the aorta <u>above the</u> <u>aortic bifurcation.</u> Abdominal Aortic Aneurysm

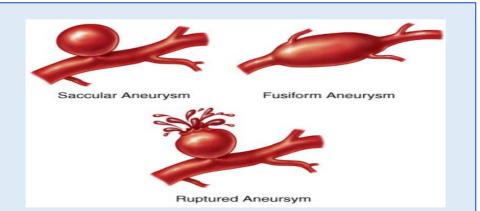


- rupture ,intraluminal thrombus. <u>Se</u>

Dissecting aortic aneurysm – LPF autopsy finding.

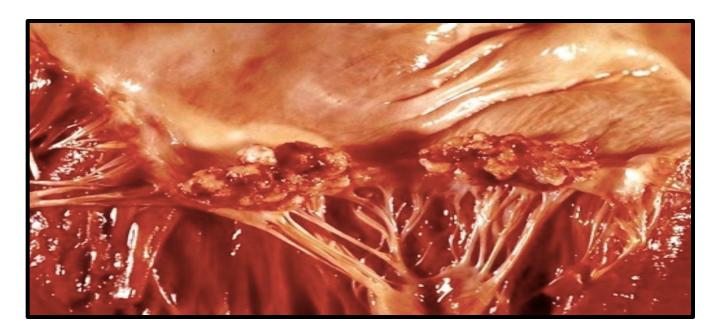


<u>A dissecting aortic aneurysm</u> occurs when blood enters the aortic wall through a defect and moves between two layers of the wall, stripping the inner layer from the outer layer.



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.

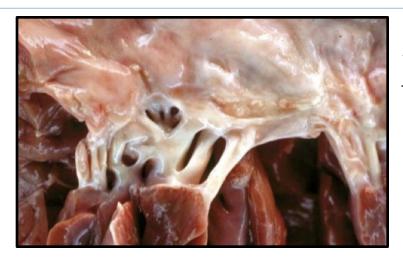
Rheumatic Aortic Valvulitis - Gross



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

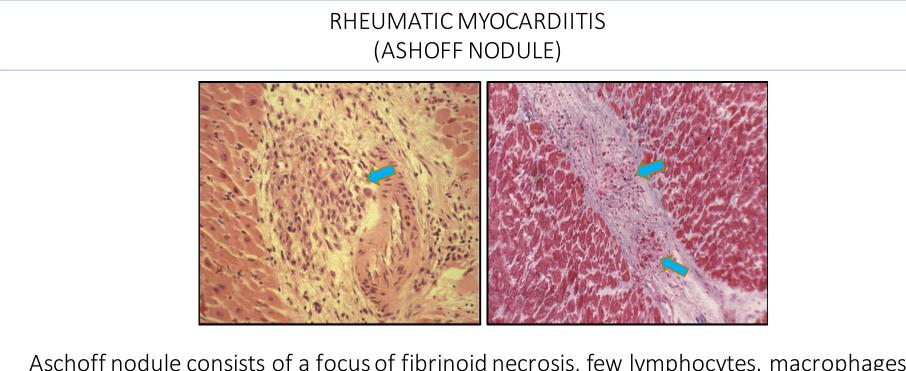
Case #4: Vegetations of rheumatic fever on mitral and aortic valves **only in Dr Marie's lecture**

Chronic Rheumatic Mitral Valvulitis - Gross



Chronic rheumatic mitral valvulitis the valve leaflets are thick, fibrotic, fused. Short, thickened, fused chordae tendinae stenosis and / or incompetence

Case #5: Acute rheumatic myocarditis



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).

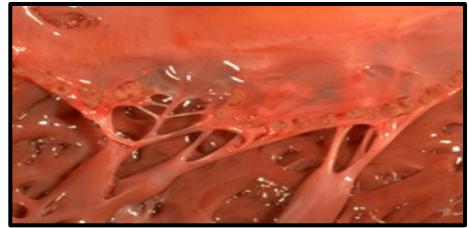
Case #5: Acute rheumatic myocarditis

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



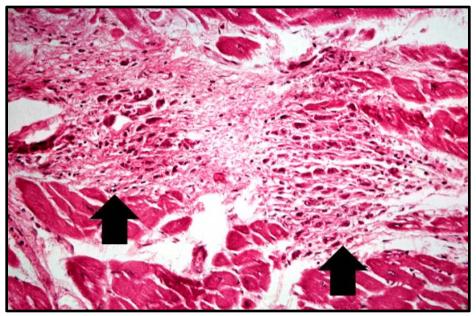
These warty vegetations are multiple, firm, adherent, small, 1-3 mm in- diameter and form along the line of valve closure over areas of endocardial inflammation



- Fish mouse deformity.

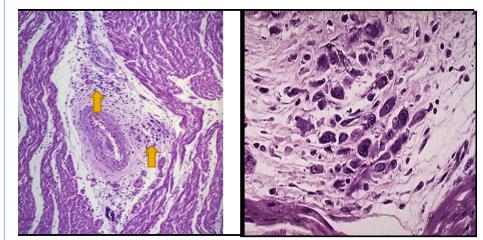
- Fusion of commissures.
- Thickening and calcifications of cusps.
- Vegetations.

Acute Rheumatic Carditis



"Aschoff nodules" seen best in myocardium

(ASHOFF NODULE)



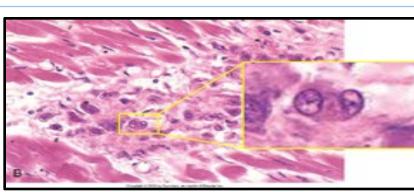
Aschoff bodies in the intermuscular fibrous septa. They are oval in shape and seen in relation to blood vessels.

- 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. <u>The most characteristic component is the Aschoff giant cell</u> (anitschkow)

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



Case #6: <u>Right Sided Heart Failure</u> Chronic venous congestion of the live<u>r</u>

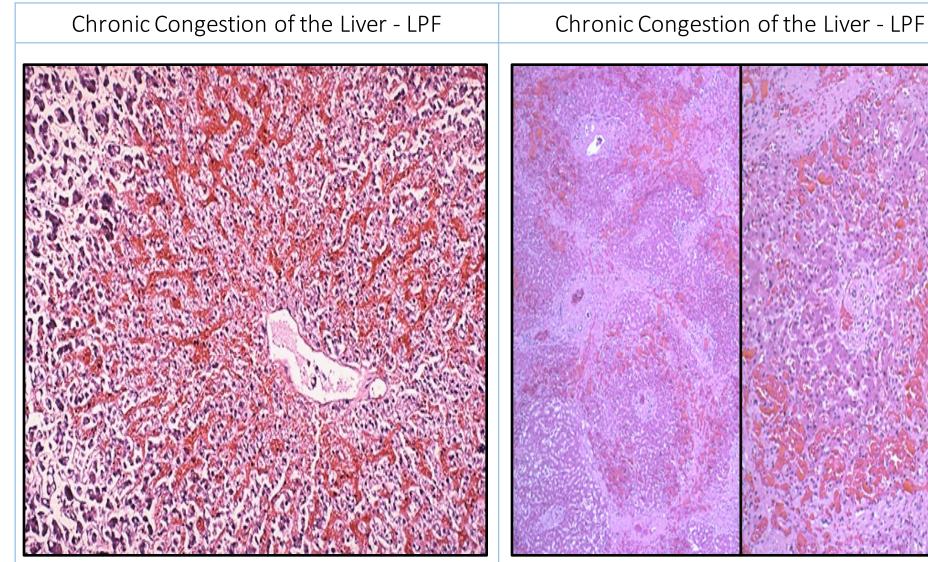
NUTMEG LIVER – Cut surface



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*





The central portion of liver lobules shows:

- Congestion and dilatation of central veins and blood sinusoids.
- atrophy and necrosis of liver cells.
- Central veins dilated and congestednecrotic hepatocytes

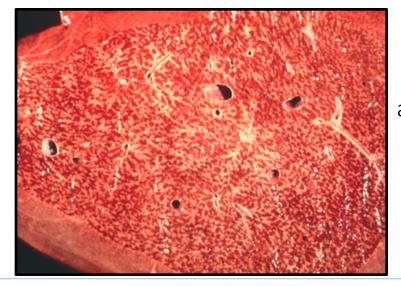
Case #6: <u>Right Sided Heart Failure</u> Chronic venous congestion of the live<u>r</u> **Only in Dr Marie's lecture**

NUTMEG LIVER – Cut surface



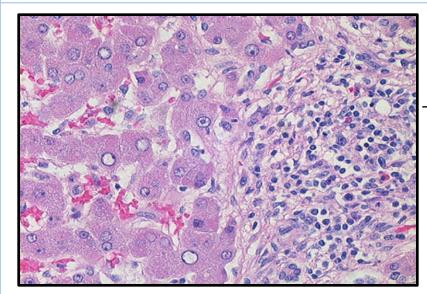
he hepatic parenchyma contains a faintly nodular pattern and nutmeg staining due to chronic passive congestion due to Right sided heart failure.

Chronic Congestion of the Liver - CS



A gross view of nutmeg appearance of liver characteristic of centrolobular or necrosis or passive congestion of the liver. The central areas of the liver are congested and take on a sort of dusky appearance. They are soft in consistency and they are surrounded by the paler areas of fatty liver that are more normal in appearance microscopically

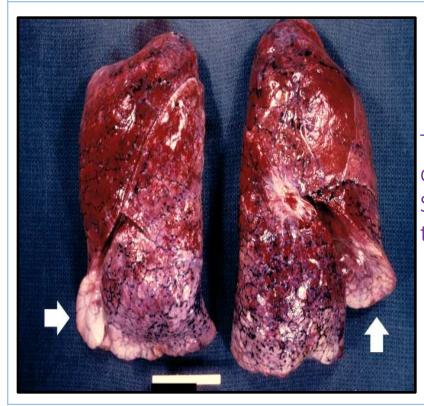
Chronic Congestion of the Liver - HPF



The central portion of liver lobules shows congestion and dilatation of central veins and blood sinusoids, with atrophy and necrosis of liver cells.

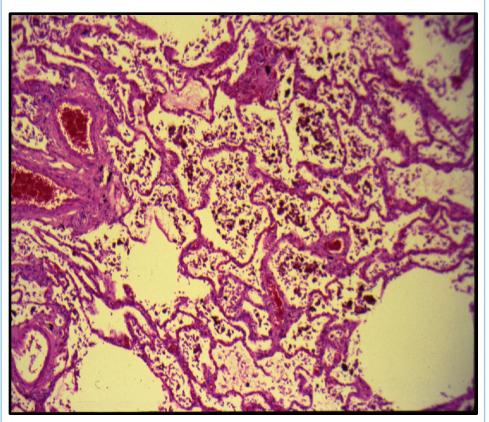
Case #7: Left Sided Heart Failure Chronic venous congestion of the Lung

Chronic venous congestion of the lung - Gross

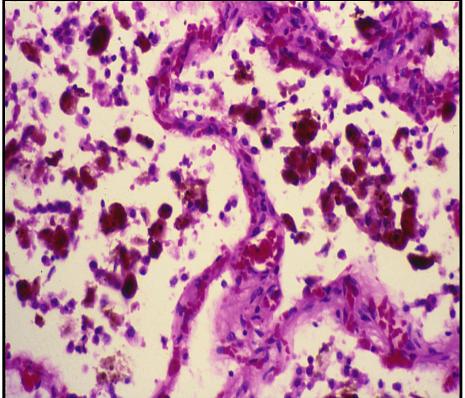


The reddish coloration of the tissue is due to congestion. Some normal pink lung tissue is seen at the edges of the lungs (arrows).

chronic vehous congestion of the fung in a chronic vehous congestion of the fung in t	Chronic venous congestion of the lung - LPF	Chronic venous congestion of the lung - HPF
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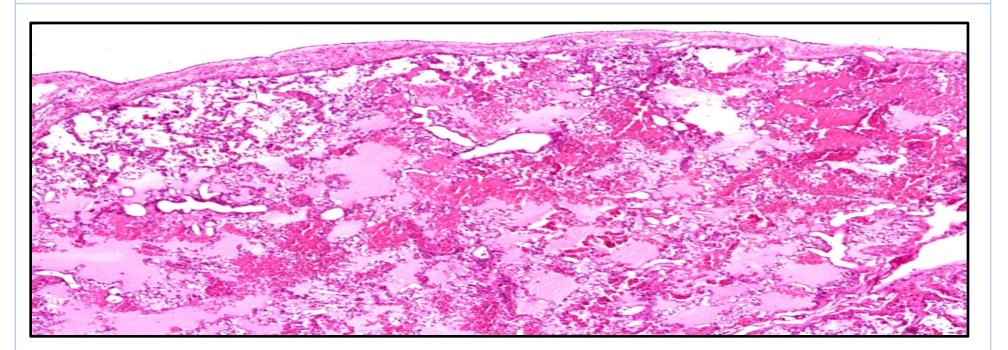
The alveolar walls are thickened by dilated and engorged capillaries. Engorged= full of RBC.



-Heart failure cells: Macrophages - Congested RBC

Case #7: Left Sided Heart Failure Chronic venous congestion of the Lung **Only in Dr Marie's lecture**

Chronic venous congestion of the lung - LPF



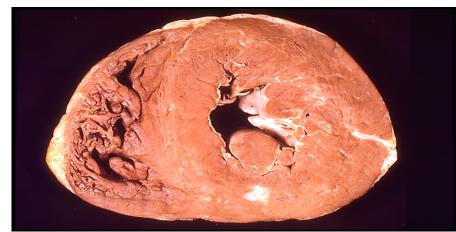
Lung, pulmonary edema in patient with congestive heart failure due to heart transplant rejection

Case #8: MYOCARDIAL HYPERTROPHY

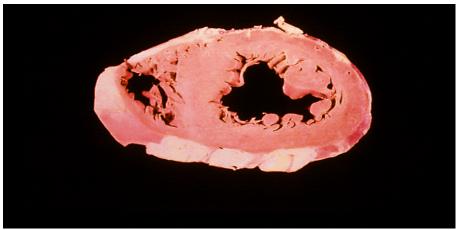
Theoretical Information's:

- 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

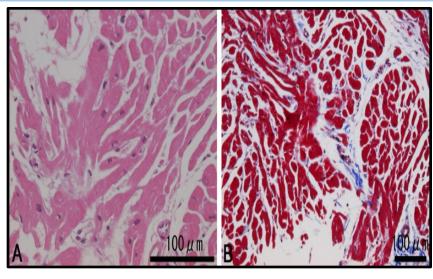


Normal ventricles

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

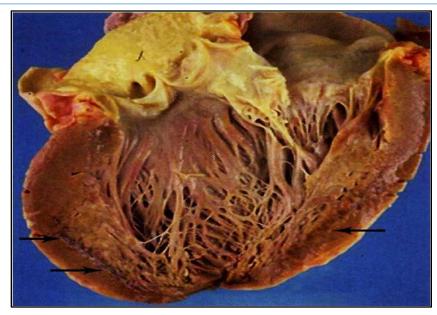
Theoretical Information's:

- Acute myocardial infarction can be complicated by:
 - <u>Cardiac Arrhythmias, MOST COMMON CAUSE.</u>
 - 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.

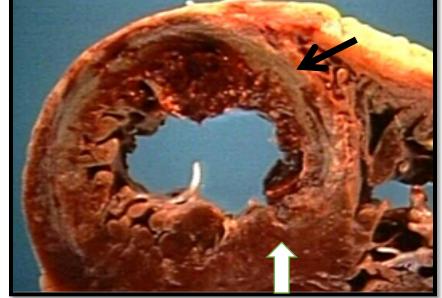
Death in these patient can be due to complications secondary to acute myocardial infarction

Myocardial Infarction - CS



arrows pointing at infracted areas

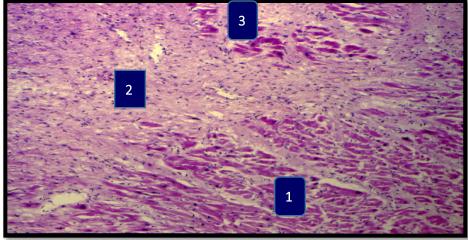
Myocardial Infarction - CS



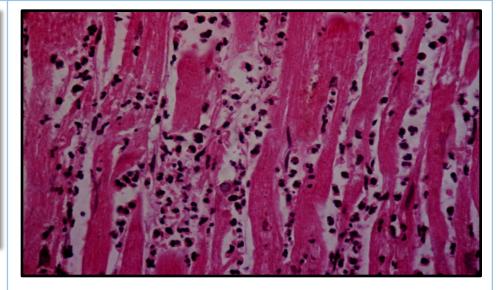
a- Pale and irregular myocardial fibrosis(Black arrow)
Caused by :<u>atherosclerosis</u>
b- Thick left ventricular wall. (white arrow) Caused by: <u>Hypertension</u>

Myocardial Infarction – late stage

Acute Myocardial Infarction



- 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.

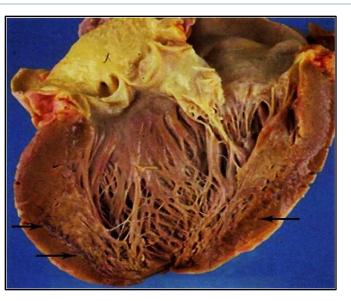


This 3-4 day old infarct showing:

- Necrotic myocardial fibers.
- Infiltration by polymorphonuclear leukocytes.

Case #9: Myocardial Infarction only in Dr Marie's lecture

Myocardial Infarction - CS



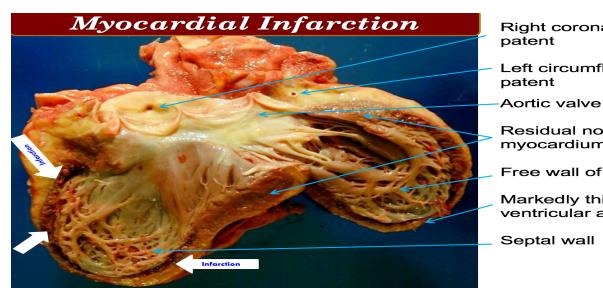
Complications that might occur : arrhythmias , ventricular aneurysm, rupture of myocardium, cardiac tamponade and others.

Myocardial Infarction - CS



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

Myocardial Infarction



Right coronary ostium,

eft circumflex artery,

Residual normal myocardium

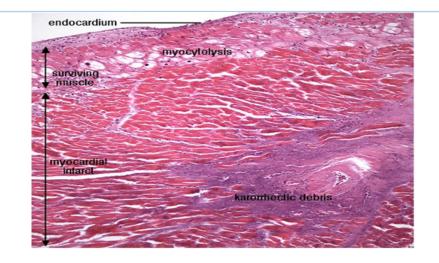
Free wall of left ventricle

Markedly thinned left ventricular apex

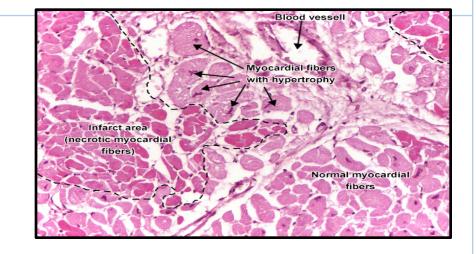
The heart is opened showing the left ventricle. There is a Massive Transmural Infarction extending around the entire wall between the white arrows.

Case #9: Myocardial Infarction only in Dr Marie's lecture

Myocardial Infarction - LPF

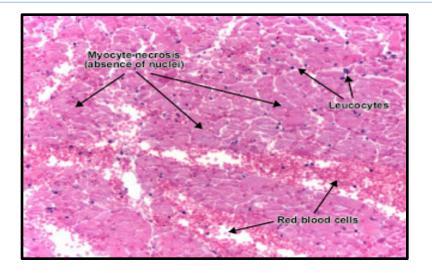


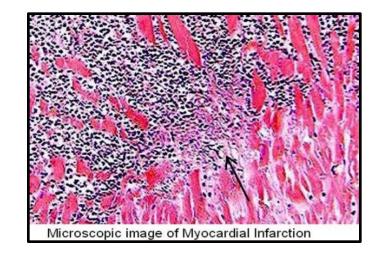
Transmural myocardial infarct at 2 weeks.



Myocardial infarct - circumscribed area of ischemic necrosis - coagulative necrosis. In the first 12 - 24 hours, myocardial fibers are still well delineated, with intense eosinophilic (pink) cytoplasm, but lost their transversal striations and the nucleus (left side of the picture). Notice a few myocardial fibers showing hypertrophy (increased size of the fiber, irregular shape of the nuclei)

Myocardial Infarction LPF





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)



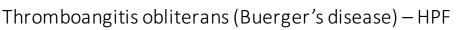


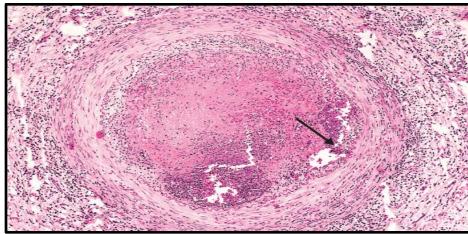
Black discoloration of the patient's finger and toes Thromboangitis obliterans (Buerger's disease) – HPF



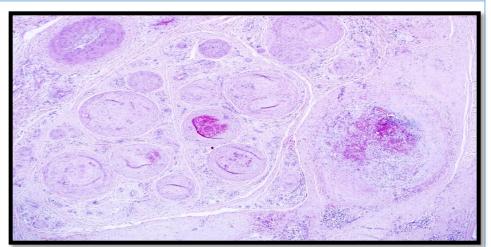
Thromboangitis obliterans (Buerger's disease)

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



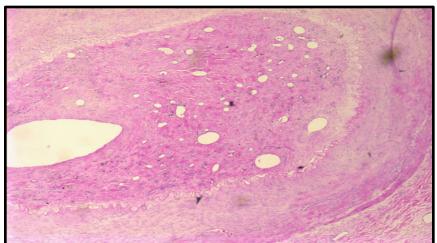


lumen is occluded by a thrombus in lumen (arrow)
vessel wall is infiltrated with leukocytes.



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

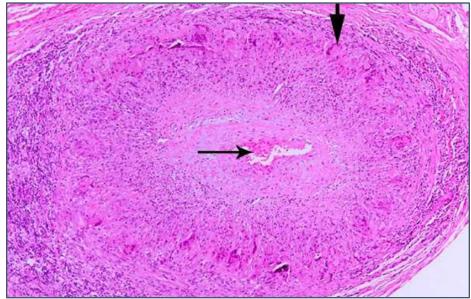
Theoretical Information's:

- 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).



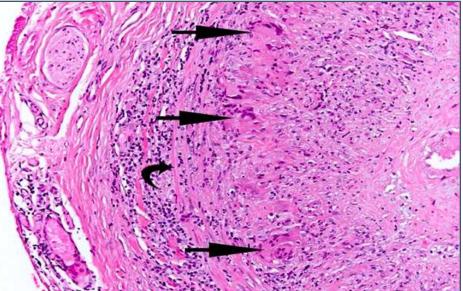
Tender and thickened temporal artery

GIANT CELL / TEMPORAL ARTERITIS - LPF

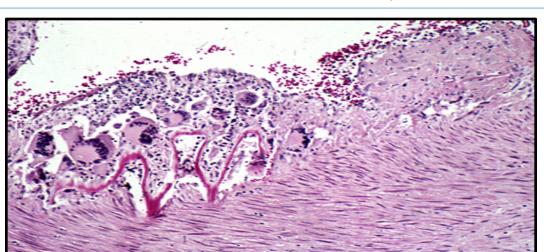


- Circumferential involvement of the vascular media is present (vertical arrow pointing downward).
- chronic lymphocytic inflammation in the media and adventitia.

GIANT CELL / TEMPORAL ARTERITIS - HPF



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



GIANT CELL / TEMPORAL ARTERITIS – HPF

- Chronic inflammation.
- Giant cells.
- <u>Fragmentation of the vascular internal</u> <u>elastic lamina.</u>
- Granulomatous inflammation.

Case #12: <u>Leukocytoclastic vasculitis</u>/ 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 Erythematous and purpuric skin rash affecting the glomerulonephritis and hemoptysis due to pulmonary right foot (Purpura:- Subcutaneous bleeding.) capillaritis Blood vessels: small vessels " capillaries " Leukocytoclastic vasculitis - HPF Leukocytoclastic vasculitis - HPF Fibrinoid type necrosis

Red cell extravasation

A Inflammation

-Fibrinoid vascular necrosis.- Nuclear debris.- Neutrophilic (polymorphonuclear) infiltration.



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 Xray 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: <u>PathologyP435@gmail.com</u>