



PATHOLOGY PRACTICAL CARDIOVSCULAR BLOCK



CVS – Pathology practical

#Case1 ATHEROMA OF THE AORTA

*An **atheroma** is an accumulation and swelling in artery walls made up of (mostly) macrophage cells, or debris, and 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:





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- extensive ulceration in the plaques
- formation of overlying mural thrombus.
- fissured-appearing endothelial surface.
- raised plaque-like structures from the surface.
- Red clot material adherent to the plaques in multiple areas.
- These clots consist of platelets held together by fibrin strands.
- raised yellow plagues and the fissures in between the plaques.
 - **Dystrophic calcification**

1/aorta with mild atherosclerosis: scattered lipid plaques. 2/aorta with moderate atherosclerosis: many more larger plaques. 3/aorta with severe atherosclerosis: extensive ulceration in the plaques.

*Inner surface of aorta and bifurcation, opened lengthwise along the posterior midline.



- irregular variegated lining due to diffuse disease
- black arrow: red thrombi .
- white arrows : ostia of celiac and superior mesenteric arteries and right renal artery .
- deceptive narrower caliber of abdominal aorta below celiac artery due to rigidity of calcified atheroma.

#Microscopic :



- atheromatous emboli are rare.
- large overlying atheroma the left.
- The far left shows ulceration and hemorrhage.
- Cholesterol clefts are numerous.



- cholesterol clefts.
- foam cells .
- Arrows : Aortic Atheroma with Thrombosis



- cholesterol clefts.
- foam cells .
- Arrows : Aortic Atheroma with Thrombosis

#Case2 CORONARY ATHEROSCLEROSIS

#Gross:



as the pinching in the blue- coloured artery at bottom center.

#Microscopic :



Coronary artery with atherosclerosis (fibro-lipid or fibro-fatty plaque). The atheromatous fibro-fatty plaque is characterized by the accumulation of lipids in the intima of the arteries, narrowing the lumen. Beneath the endothelium it has a "fibrous cap" covering the atheromatous "core" of the plaque, which consists in cholesterol crystals, cholesterol esters, fibrin, macrophages and smooth muscle cells, proteoglycans, collagen, elastin and cellular debris.

#Microscopic :



A normal coronary artery with no atherosclerosis and a widely patent lumen that can carry as much blood as the myocardium requires



Atheromatous plaque in a coronary artery that shows endothelial denudation with disruption and overlying thrombus formation at the right. The arterial media is at the left



Occlusive coronary atherosclerosis. The coronary at the left is narrowed by 60 to 70%. The coronary at the right is even worse with evidence for previous thrombosis with organization of the thrombus



This distal portion of coronary artery shows significant narrowing. Such distal involvement is typical of severe coronary atherosclerosis, such as can appear with diabetes mellitus or familial hypercholesterolemia.



Severe coronary atherosclerosis with narrowing of the lumen

*Coronary Atherosclerosis:



Partial occlusion of the lumen by an atheromatous plaque. The plaque consists of dissolved, cholesterol clefts, hyaline fibrous tissue and some blood capillaries.



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

*Recent Thrombus in coronary Artery :



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.

*Hyaline Arteriosclerosis:



Arteriosclerosis (hardening of the arteries) involves both small and large vessels. It is commonly found in diabetics and hypertensives.

*Hyperplastic arteriolosclerosis - HPF



-Hyperplastic arteriolosclerosis: 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.

Ischemic fibrosis of myocardium (diffuse ventricular myocardial fibrosis)



Acute viral myocarditis - MPF



Aortic atherosclerosis - HPF

-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.

-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.



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

#Case3 ANEURYSM OF ABDOMINAL AORTA



*Types of Aneurysms:1-Fusiform 2-Saccular 3- Raptured*Complication: Rapture of the aneurysm

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

*Abdominal Aortic Aneurysm:



atherosclerotic aneurysm of the aorta in which a large "bulge" appears just above the aortic bifurcation.



Aneurysmal dilatation of the abdominal aorta with rupture , intraluminal thrombus and extensive aortic atherosclerosis . The patient had suddenly developed severe abdominal pain, shocked and collapsed.

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#Dissecting aortic aneurysm LPF



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 laye, Usually associated with atherosclerosis, inflammation, and degeneration of the connective tissue of the tunica media

#Case4 VEGETATIONS OF RHEUMATIC FEVER ON MITRAL AND AORTIC VALVES

#Gross :

Acute Rheumatic <u>Mitral</u> valvulitis



- Multiple warty vegetations are firm and adherent.
- small (1-3 mm) in diameter .
- Form along the line of valve closure over areas of endocardial inflammation.

Rheumatic Aortic valvulitis



Aorta has been removed to show thickened, fused aortic valve leaflets .

Chronic Rheumatic Mitral valvulitis



- Large vegetations
- Hemorrhage along the free margins of mitral valve.



- Fusion of the commissures.
- Calcification of the cusps
- Vegetations.
- "fish mouth" deformity.



- The valve leaflets are thick, fibrotic and fused.
- Short, thickened, fused chordae tendinae.
- Stenosis and \ or incompetence.

#Case5 RHEUMATIC MYOCARDITIS

#Microscopic :



The myocardium showing cellular accumulations of Aschoff bodies (arrow) within the interstitium of the myocardium



An Aschoff nodule at high magnification



- Aschoff bodies in the intermuscular fibrous septa.
- They are oval in shape and seen n relation to blood vessels.
- fibrinoid necrosis .
- Few lymphocytes and macrophages.
- small giant cells with one or several nuclei (Aschoff giant cell).

#Theoretical information:

> Aschoff nodules consists of:

- 1- macrophages including Aschoff giant cells.
- 2- Collagen necrosis.
- Major Jones criteria:
 - 1-Carditis. 2- Polyarthritis. 3- Chorea.
 - 4- Erythema marginatum. 5- Subcutaneous nodules.
- Serological Test for Rheumatic carditis: Anti streptolysin Antibodies.
- > The most likely organism: group A beta hemolytic streptococci. #

#Case6 HEART FAILURE

*Right-sided heart failure :

(Chronic venous congestion of the liver)

NUTMEG LIVER – Cut surface Section of liver showing alternating pale and dark areas with a nutmeg (نبتة جوزة الطيب) like appearance possibly due to passive congestion secondary to right sided heart failure.
NUTMEG LIVER – Cut surface The 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

*Right-sided heart failure :

(Chronic venous congestion of the liver)

#Microscopic: LPF/HPF



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



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



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

*Left-sided heart failure : (Chronic venous congestion of the lung)

#Gross :



This is a gross photograph of lungs that are distended and red. The reddish coloration of the tissue is due to congestion. Some normal pink lung tissue is seen at the edges of the lungs (arrows).

#Microscopic :

	(LPF)
	The alveolar walls are thickened by dilated and engorged capillaries. *RBCs present in the alveolar spaces
	(LPF)
	Lung, pulmonary edema in patient with congestive heart failure due to heart transplant rejection
and a property of	(HPF)
	The alveoli contain edematous fluid, red blood cells and large alveolar macrophages (heart failure cells), which are filled with haemosiderin pigment derived from red cells breakdown.

#Case7 MYOCARDIAL HYPERTROPHY





Heart from a hypertensive patient. The left ventricle is very thick (over 2 cm). However the rest of the heart is fairly normal in size as is typical for hypertensive heart disease. The hypertension creates a greater pressure load on the heart to induce the hypertrophy.



#Microscopic:



#Case8 MYOCARDIAL INFARCTION

<u>*Definition:</u> (MI), also commonly referred to as "heart attack," is necrosis of heart muscle resulting from ischemia

*Complications that might occur in MI:

- Heart Failure.
- Myocardial rupture (3 days).
- Ventricular aneurysm (7 days).

*Enzymes elevated:

- a. Troponin
- b. CK-MB (Creatine Kinase).
- C. LDH (Lactic dehydrogenase). d. Myoglobin.

#Gross:



 Black arrows show soft hemorrhagic areas, consistent with MI.

 Part of the heart which is affected: Left ventricle.



In both pictures we see: Cross section of the left and right ventricles showing a pale and irregular focal fibrosis in the left ventricular wall with increased thickness

#Microscopic:





Acute myocardial infarct, histology. This 3-4 day old infarct shows necrosis of myocardial cells and is infiltrated with polymorpohnuclear leukocytes. Occlusion of Left anterior descending coronary artery is responsible for 40-50 % of MI.



 Patchy coagulative necrosis of myocardial fibers. The dead muscle fibers are structure-less and hyaline with loss of nuclei and striations.
 Chronic ischemic fibrous scar replacing dead myocardial fibers .
 The remaining myocardial fibers show enlarged nuclei due to ventricular hypertrophy .



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#Case9 THROMBOANGITIS OBLITRANS (BUERGER`S DISEASE)

#Gross:



*Black discoloration of the skin possibility caused by ischemia .

*Etiology is hypersensitivity to tobacco products .



*pathologic finding of an acute inflammation and thrombosis (clotting) of arteries and veins of the and feet (the lower limb being more common)

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

#Microscopic 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.

Wegener's granulomatosis (Granulomatous polyangitis) is a destructive vasculitis characterized by the formation of ill-defined granulomas in vessel wall together with common renal and lung involvement.

#Microscopic LPF:



- a. Thrombus.
- b. Microabscesses within the thrombus.
- c. Vasculitis (infiltration of the vessel wall by neutrophils).

*Sometimes <u>nerves are also involved</u> giving rise to pain in affected areas.

#Microscopic HPF:



Some blood vessels show recent organizing thrombi while others show infiltration of the wall and surrounding tissue by chronic inflammatory cells

#Case10 GIANT CELL / TEMPORAL ARTERITIS

#Gross :



#Microscopic:



(LPF)

 ①Circumferential involvement of the vascular media is present .
 ②Chronic lymphocytic inflammation in the media and adventitia , Reactive intimal fibroplasias lead to luminal stenosis with <10% of its original luminal diameter .

(HPF)

③ Giant cells can be of Langhans type or foreign-body type , and may show fragments of disrupted

internal elastic lamina .

④ Dense chronic lymphocytic inflammation traversing through circumferential smooth muscle fibers of vascular media.

#Microscopic HPF:



* single granuloma in the adventitia of the artery.
* granulomatous in addition to both acute and chronic inflammatory cells , 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 giant cells and chronic inflammatory cells and internal elastic lamina fragmentation.

#Case11 LEUKOCTYOCLASTIC / HYPERSENSITIVITY VASCULITIS (MICROSCOPIC POLYANGITIS)

#Gross:



*Clinical sign: vasculitis is secondary to deposition of <u>immunoglobulin and complement</u> in vessel wall . Hypersensitivity vasculitis might be complicated with necrotizing glomerulonephritis (Hematuria) and hemoptysis due to pulmonary capillaritis .

(The purpuric patches because of Subcutaneous

Most pronounced in dependent areas.



bleeding)

#Microscopic:





1- Fibrinoid necrosis of small dermal vessels is present , necessary to establish the diagnosis of leukocytoclastic vasculitis (black arrow)

2-surrounding tissue is showing Nuclear debris and neutrophils .



This muscular artery shows a more severe vasculitis with acute and chronic inflammatory cell infiltrates, along with necrosis of the vascular wall





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