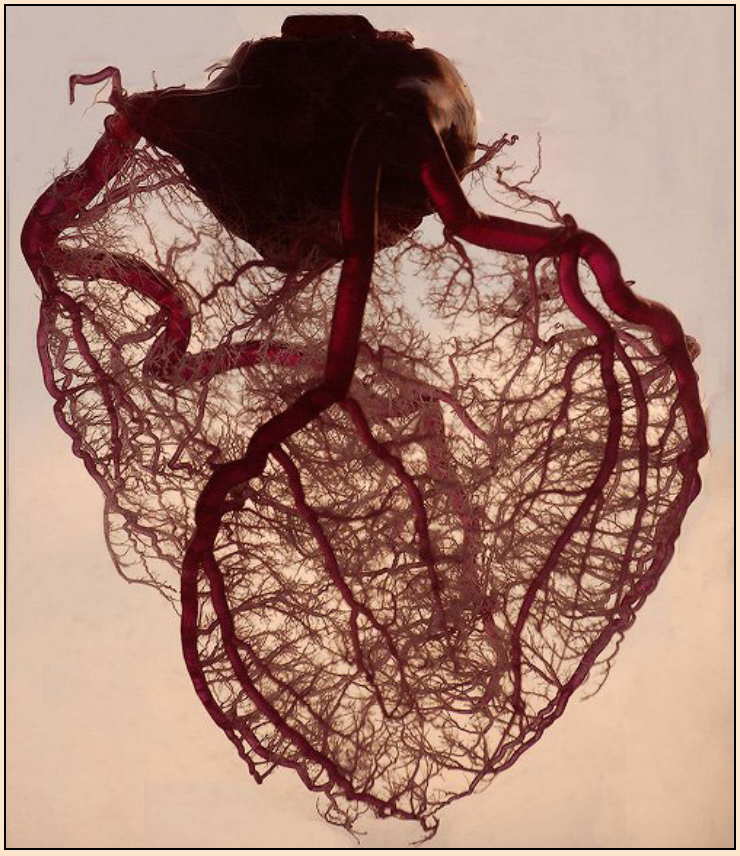
Pathology - CVS Block OSPE



You must know features, Diagnosis and Definition of all cases.

Contents:

1- Atheroma of aorta

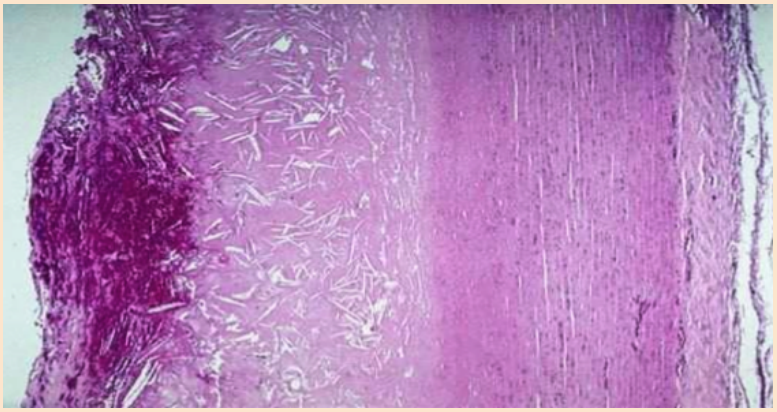
•An atheroma is an accumulation and swelling in artery walls made up of (mostly) macrophage cells, or debris, and containing lipids (cholesterol and fatty acids), calcium and a variable amount of fibrous connective tissue.

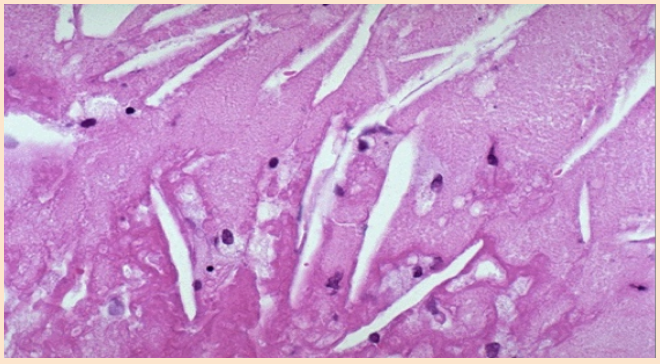
Major Factors:

- Hyperlipidemia
- Hypertension
- Cigarette smoking
- Diabetes

Complications:

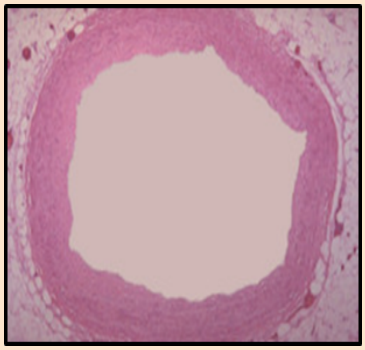
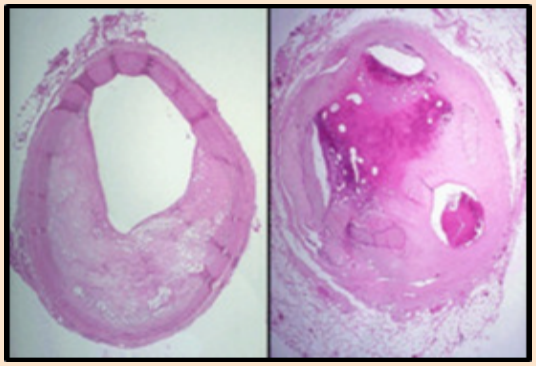
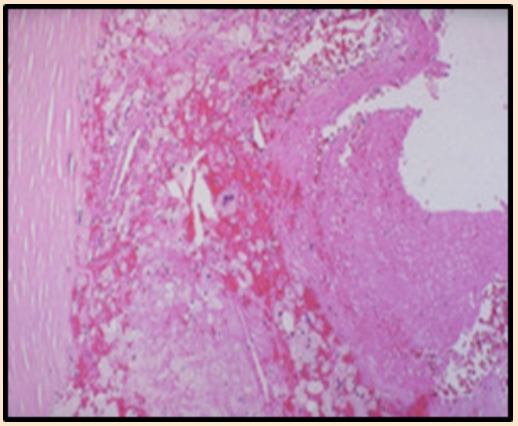
1. Coronary artery disease (angina & MI).
2. Carotid atherosclerotic disease (stroke).
3. Aneurysm formation.
4. Ulceration.
5. calcifications.

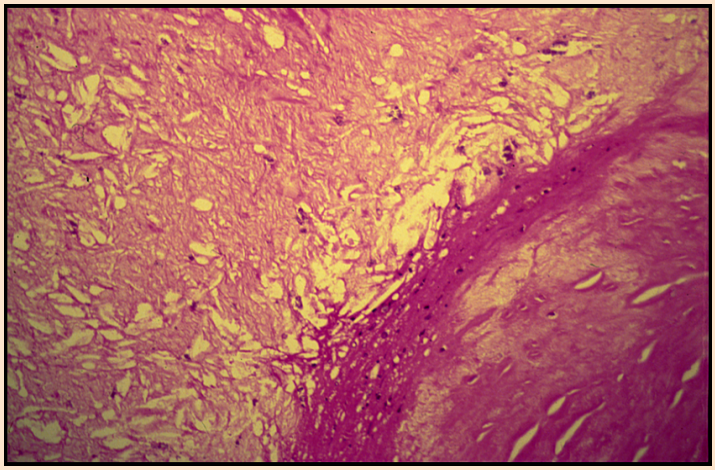
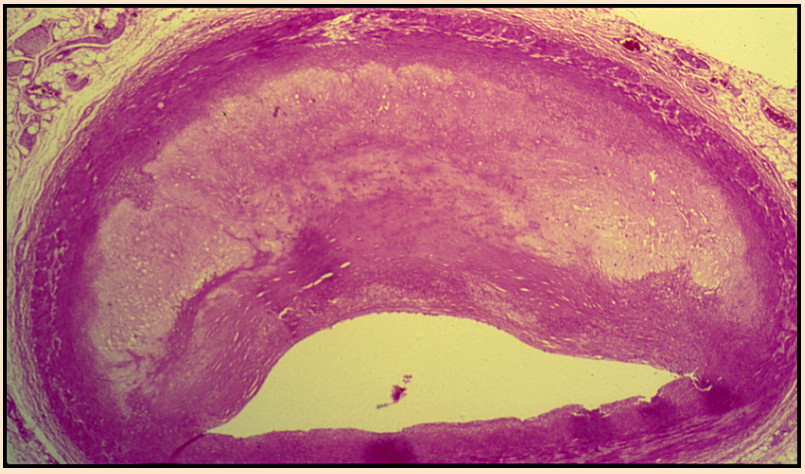
Gross	Microscopic
	
- **Yellow atheromatous plaques.** - **Areas of ulceration and haemorrhage.**	**FIVE layers seen in the aorta's section seen starting from the Left to the Right side are:** **1- Haemorrhage.** **2- Atheromatous plaque containing cholesterol clefts.** **3- Fibrosis.** **4- Elastic Media.** **5- Adventitia.**

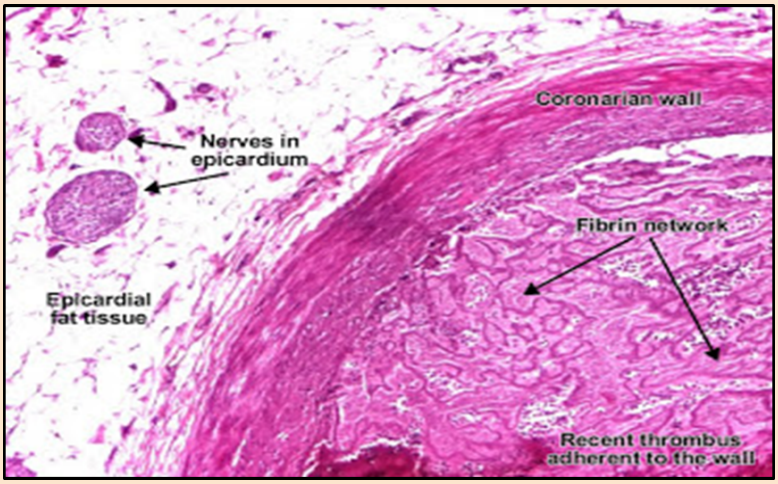


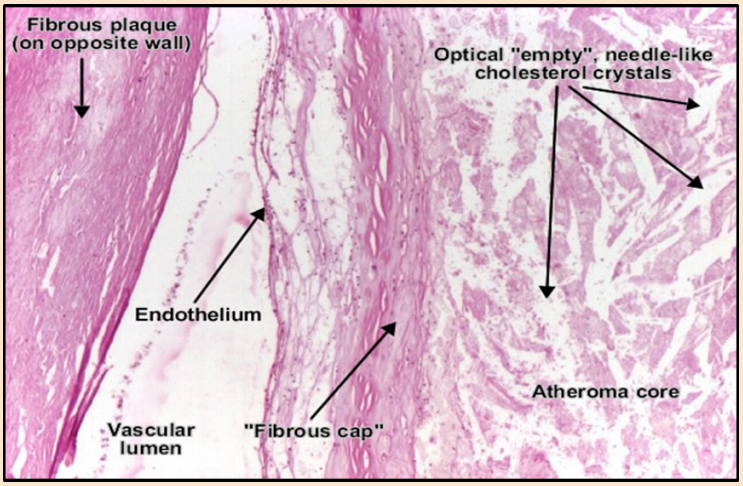
1. **aortic atheroma.**
2. **foam cells.**
3. **cholesterol clefts.**

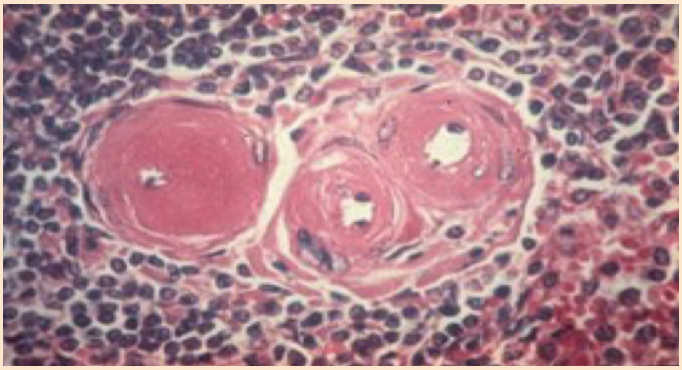
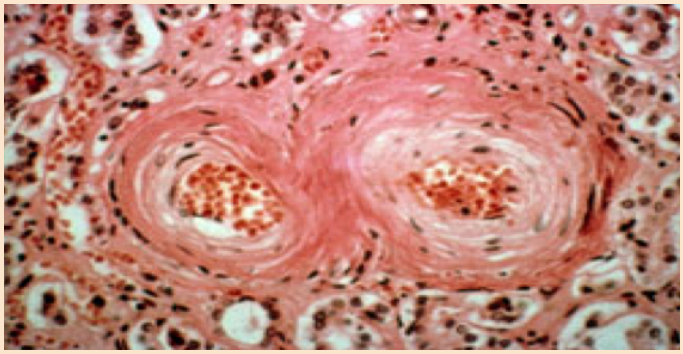
2- Coronary atherosclerosis

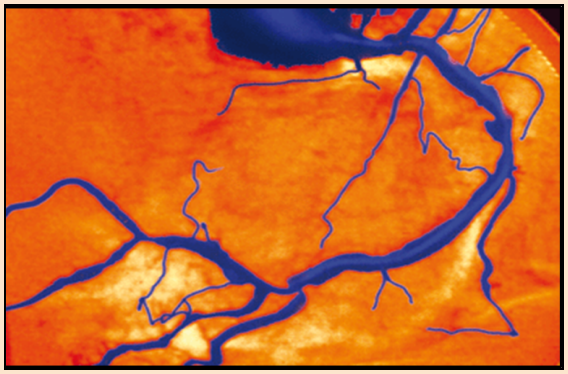
Microscopic		
		
A normal coronary artery with no atherosclerosis	**Occlusive coronary atherosclerosis 70% of lumen with thrombus organization.**	**Atheromatous plaque in a coronary artery with hemorrhage**

	
Partial occlusion of the lumen by an atheromatous plaque. ***Note: The plaque consists of dissolved,cholesterol clefts, hyaline fibrous tissue and some blood capillaries.***	***Severe coronary atherosclerosis with narrowing of the lumen.*** **Note: common in diabetes and hyperlipidemia patients.**





Hyaline arteriolosclerosis	*Hyperplastic arteriolosclerosis*
	
Arteriosclerosis (hardening of the arteries) involves both small and large vessels. It is commonly found in diabetics and hypertensives. ***● fibrinoid necrosis.***	***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.*** ***-looks like onion skin -***

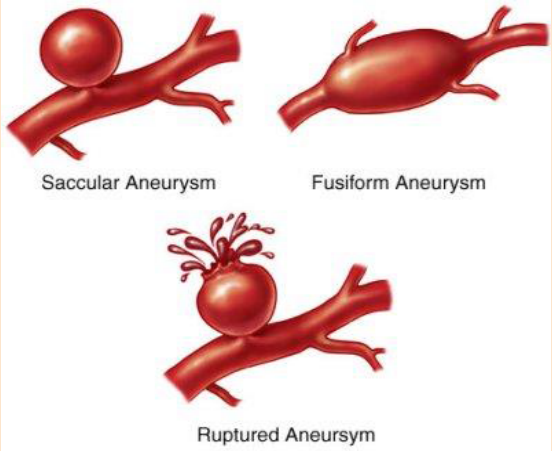


Coloured angiogram (X- ray) showing atherosclerosis in a coronary artery.

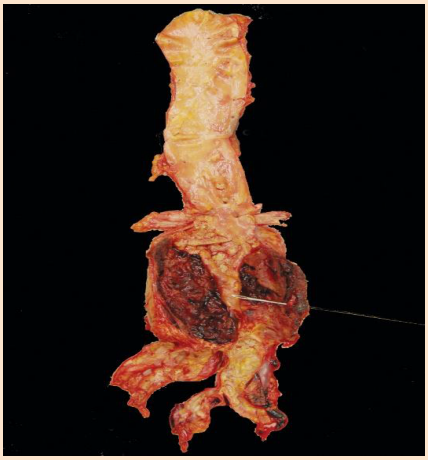
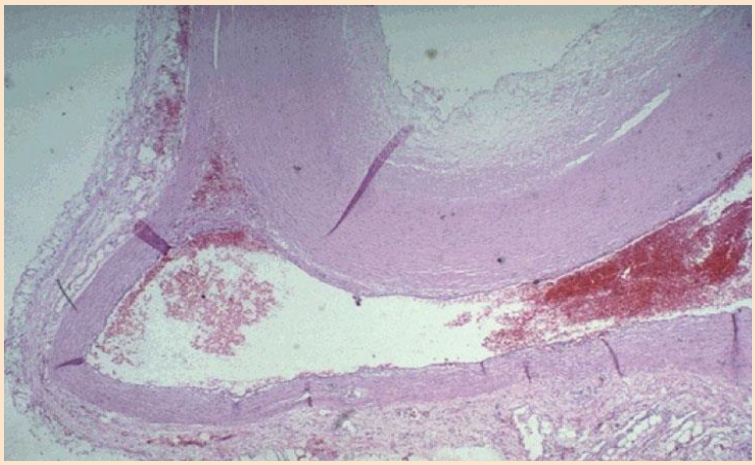
3- Aneurysm of abdominal aorta

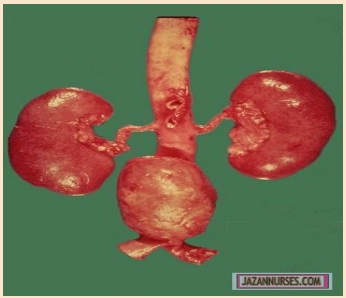
Aneurysm: means abnormal dilation of the blood vessel.

Clinical presentation: sudden development of severe abdominal pain, shock and the patient may collapse.



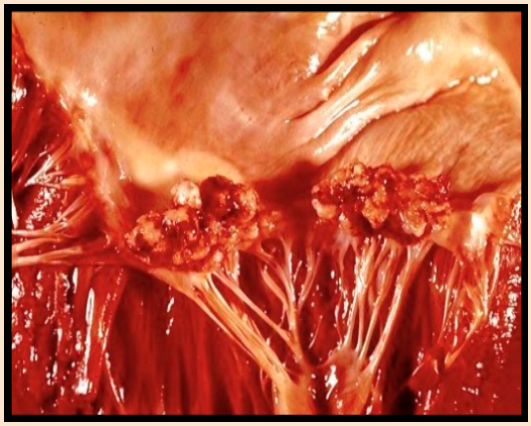
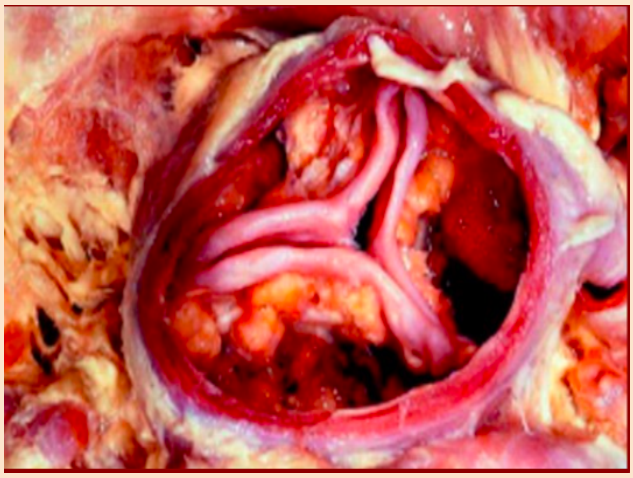
Causes:
1. Advanced atherosclerosis (Usually abdominal aorta).
2. Fungal infection (mycotic).
3. Syphilis (thoracic aorta).
4. Congenital (Berry aneurysm in circle of willis) .
- usually it is asymptomatic unless there is complications

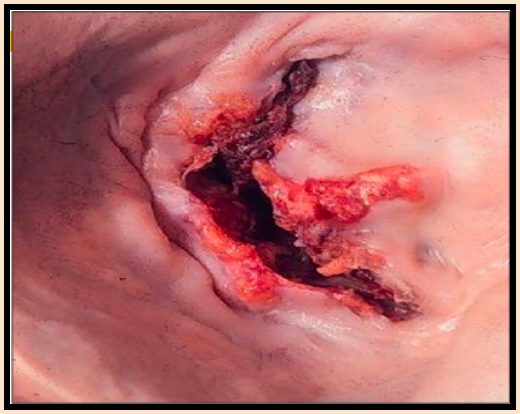
Gross	Microscopic
	
1- Aneurysmal dilatation of the lower aorta with evidence of rupture. 2- There is an intraluminal thrombus with extensive aortic atherosclerosis. (yellowish Areas). Note:The patient had suddenly developed severe abdominal pain, shocked and collapsed.	- Defect in the aortic wall: blood enters accumulates between two layers stripping the inner layer from the outer layer. - Atherosclerosis. - inflammation. - degeneration of the connective tissue of the tunica media.



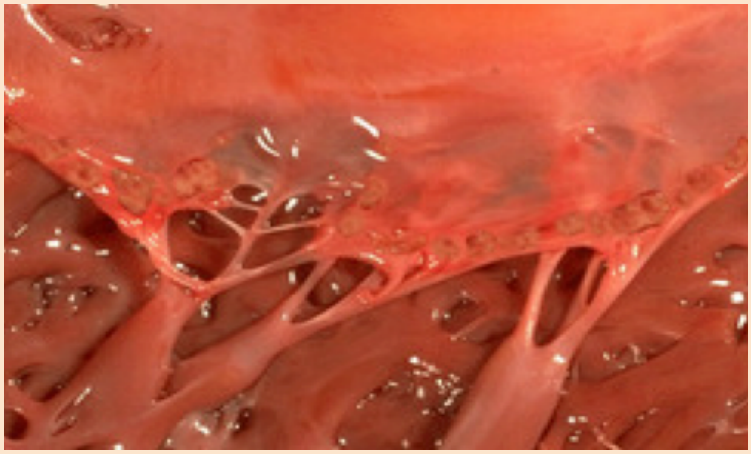
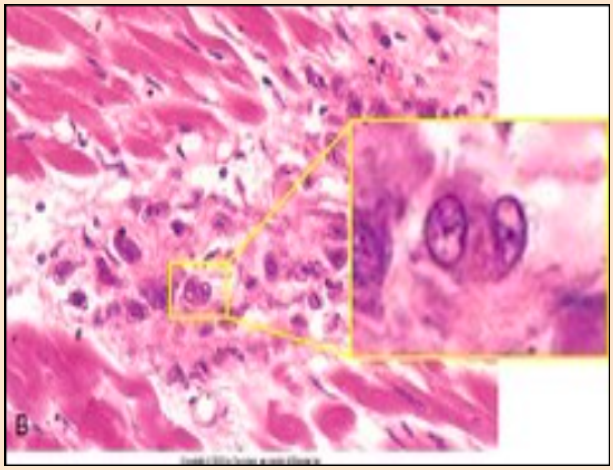
Describe: An atherosclerotic aneurysm of the aorta in which a large swelling is seen just above the aortic bifurcation.

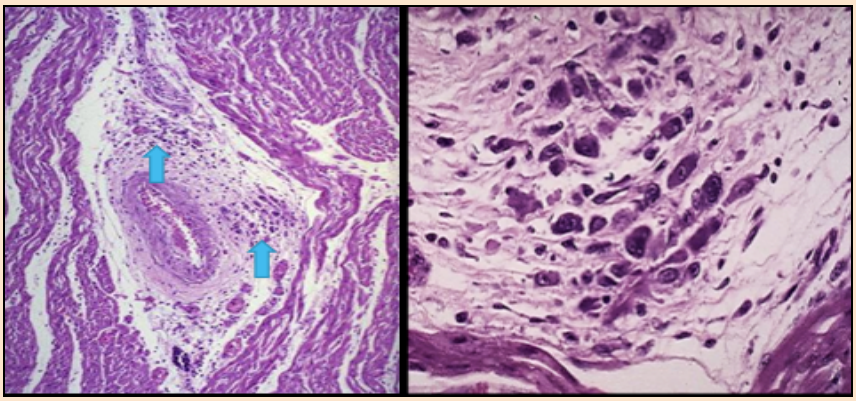
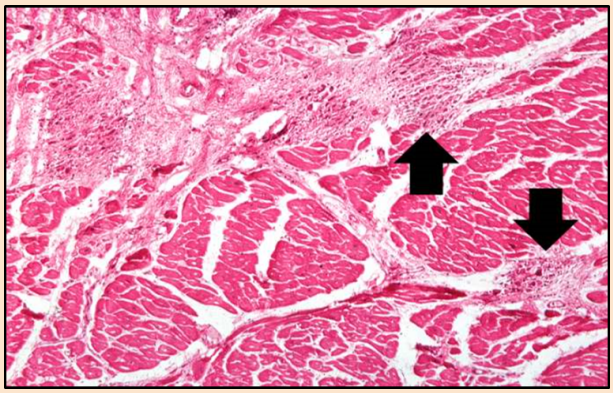
4- Vegetations of rheumatic fever on mitral and aortic valves

Chronic Rheumatic Mitral Valvulitis	**Aortic Stenosis (RHD)**
	
1. ***large vegetations.*** 2. ***hemorrhage along the free margins of the mitral valve.***	**1-Thickened.** **2- fused aortic valve leaflets.**

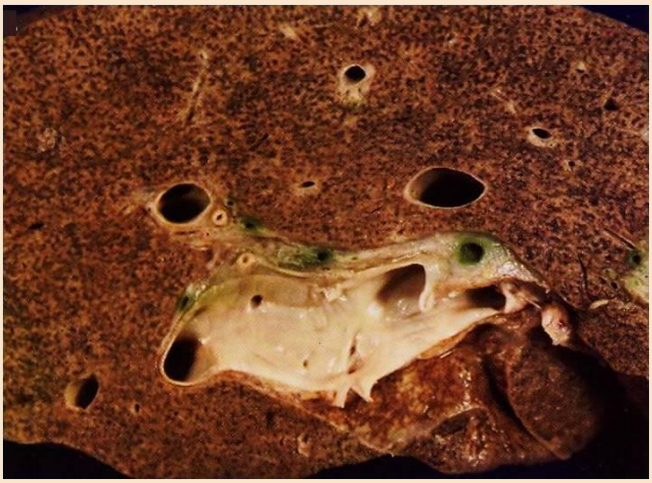
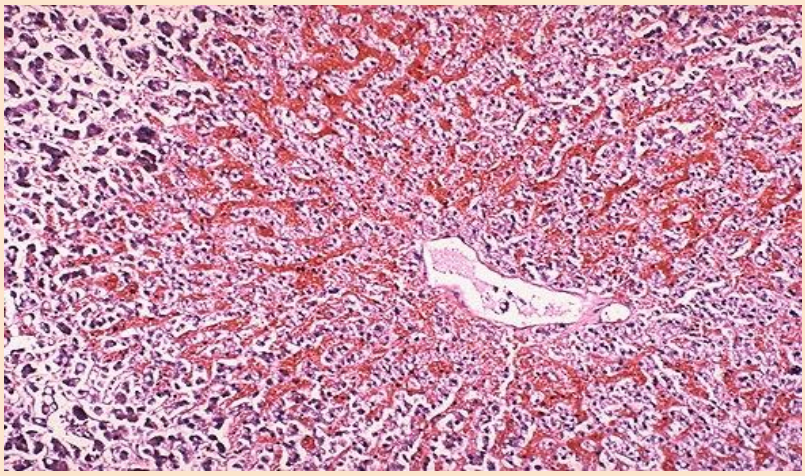
Stenotic mitral valve (fish orifice)

1-Thickening. **2-Calcification of cups.** **3-Inflamed cusps (healed by fibrosis).** **4-Mitral Valve Stenosis.**

5- Acute rheumatic myocarditis

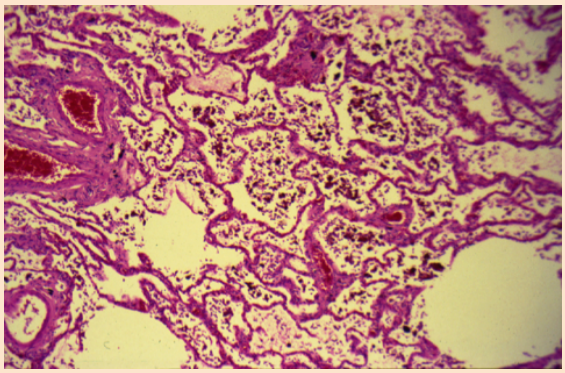
Mitral Valvulitis	Microscopic
	
Small *verrucous vegetations:* *1- They are multiple, firm* *2- adherent, small* *3-form along the line of valve closure over areas of endocardial inflammation.*	1-Aschoff body giant cell 2-activated macrophages 3-chronic inflammation

Aschoff Nodules	
	
1- oval in shape and seen in relation to blood vessels. 2-focus of fibrinoid necrosis 3- Lymphocyte 4-macrophages 5- aschoff giant cells.	*Aschoff bodies with chronic inflammatory cells.*

6- Chronic venous congestion of the liver

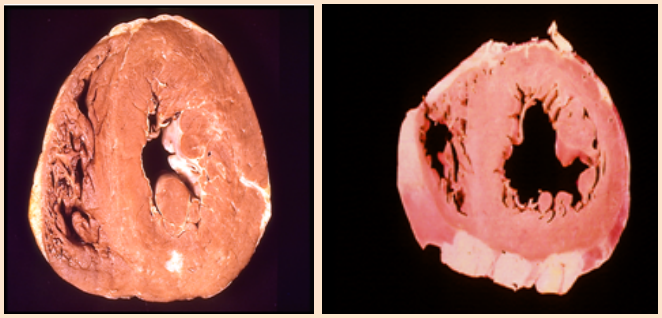
NUTMEG LIVER	(Microscopic) **Liver**
	
(nutmeg=spherical seed of a tropical tree) 1-alternating pale and dark areas 2-nutmeg like appearance. **Why?** possibly due to passive congestion secondary to right sided heart failure.	The central portion of liver lobules shows: (1)congestion and dilatation of central veins and blood sinusoids. (2) atrophy and necrosis of liver cells. Note: Kupffer cells contain few brown hemosiderin pigment granules.(Kupffer cell is a phagocytic cell which forms the lining of the sinusoids of the liver and is involved in the breakdown of red blood cells.)

7- Chronic venous congestion of the lung

Gross	**Microscopic**
	
1-Red due to congestion 2- Dilated lung Arrow shows >> normal pink lung tissue	The alveolar walls are thickened by dilated and engorged capillaries.

8- Myocardial hypertrophy

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



(Right): Normal ventricle. (Left): hypertrophied left ventricle.

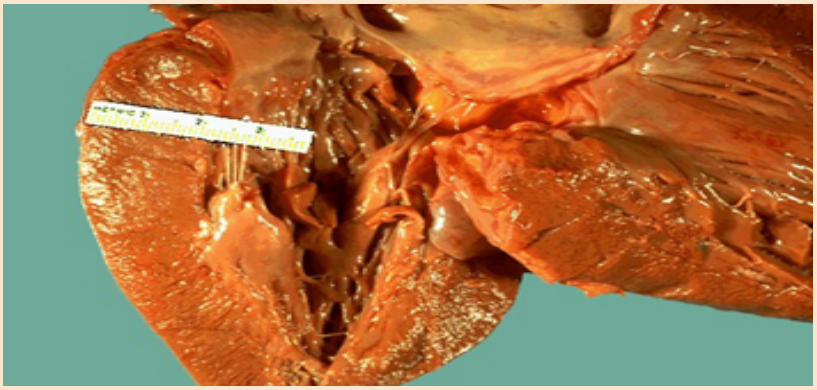
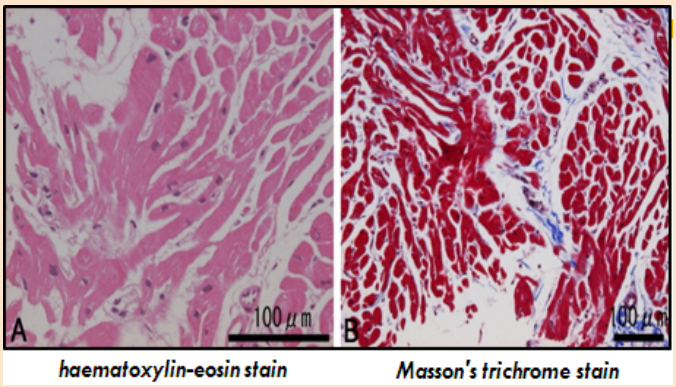
Causes of ventricular hypertrophy:

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)

Gross	***Microscopic***
	
● **The left ventricle is very thick (over 1 cm).** ● **The myocardial fibers have undergone hypertrophy.** ● **The rest of the heart is fairly normal in size as is typical for hypertensive heart disease.** ●	● **1- Unorganized myofibrils.** ● **2- interstitial Fibrosis.**

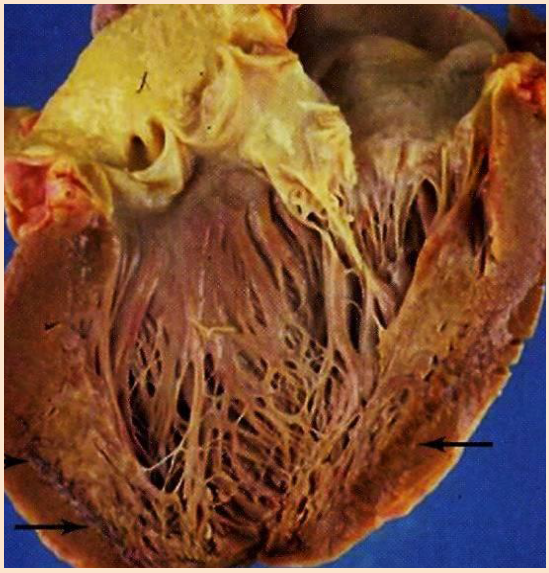
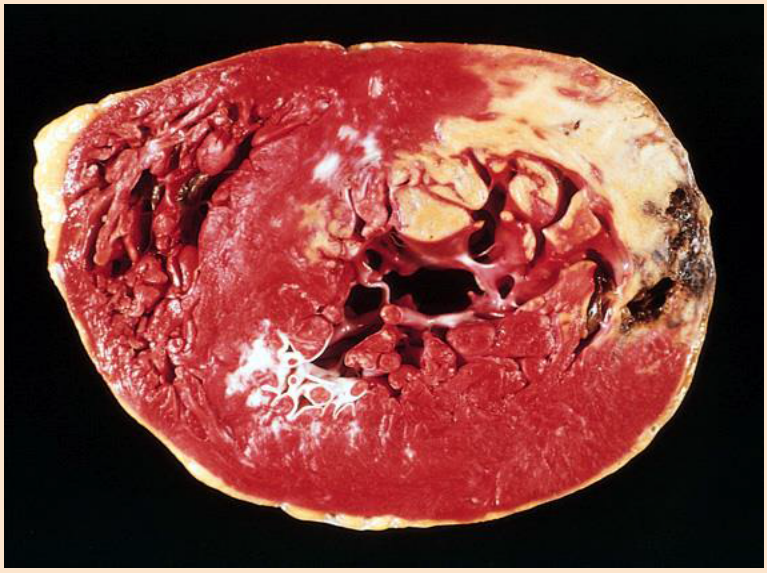
9-Myocardial infarction

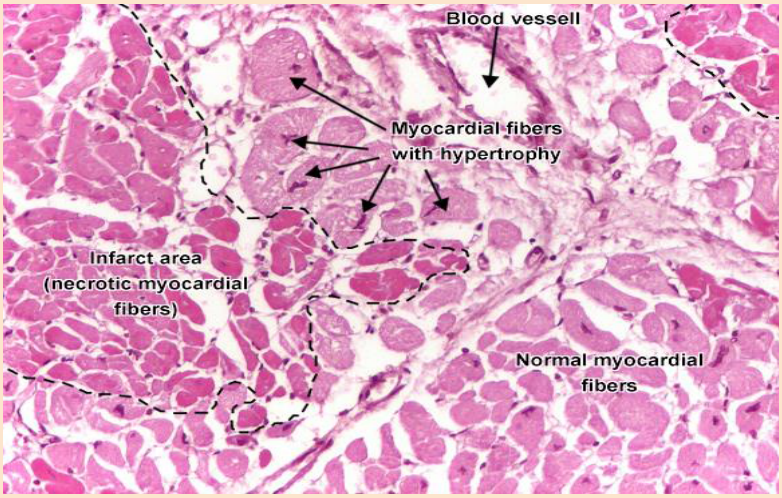
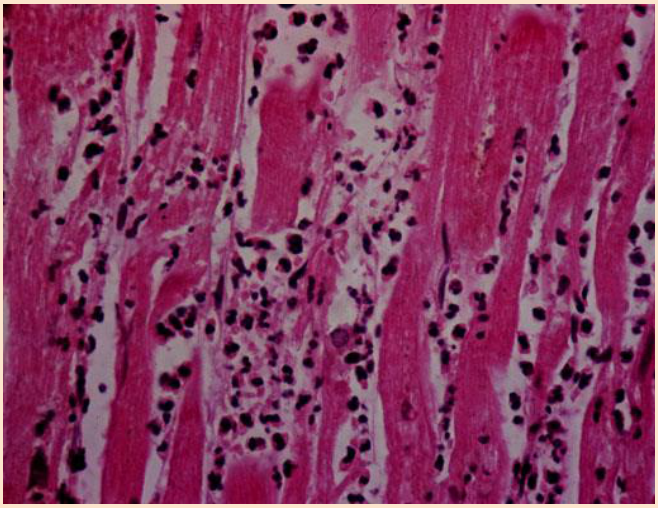
Complications: arrhythmias, ventricular aneurysm, rupture of myocardium, cardiac tamponade and others.

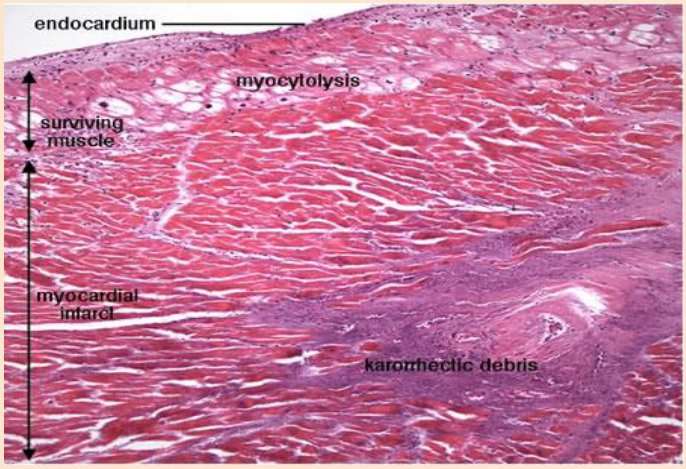
The enzymes which are usually elevated in case of MI: CKMB, Troponin I (Best marker) and LDH.

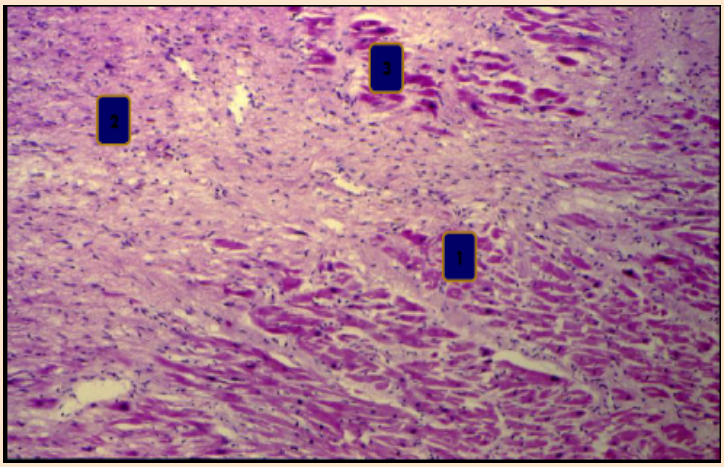
Time	Gross changes	Microscopic changes
0-4 h	None	None
4-12h	Mottling	Coagulation necrosis
12-24h	Mottling	More coagulation necrosis; neutrophils come in
1-7 d	Yellow infarct center	Neutrophils die, macrophages come to eat dead cells
1-2 w	Yellow center, red borders	Granulation tissue
2-8 w	Scar	Collagen

You dont have to memorize this table.

Gross	
	
Arrows point at Congested, hemorrhagic and soft area in the left ventricular wall	**1. pale and irregular focal fibrosis.** **2. hemorrhage.** **3. coagulative necrosis in the left ventricular wall.** **4. increased thickness.**

In the first 12 - 24 hours	3-4 day old infarct
	
● Myocardial fibers are: (in the left side of the picture). 1- still well delineated, 2- intense eosinophilic (pink) cytoplasm, 3- lost transversal striations and the nucleus ● Notice a few myocardial fibers showing hypertrophy: a. increased size of the fiber ● The interstitial space may be infiltrated with red blood cells.	1- coagulative necrosis of myocardial cells 2- infiltrated with polymorphonuclear leukocytes.





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

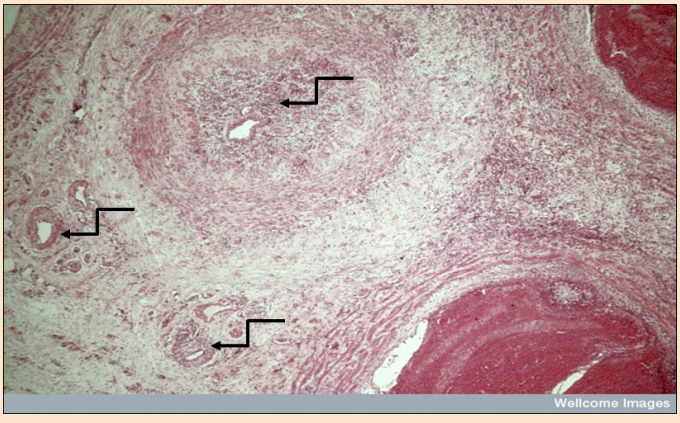
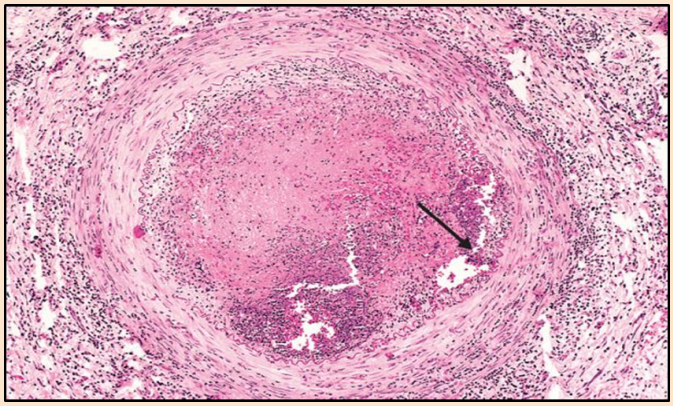
10- Thromboangiitis obliterans (Buerger disease)

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.

Main predisposing factors:
1- Smoking habits
2- Various HLA haplotypes (Genetic predisposition).
Features: *(the lower limbs being more common)*

- *acute inflammation and thrombosis (clotting) of arteries and veins of the hands and feet*
- It 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.

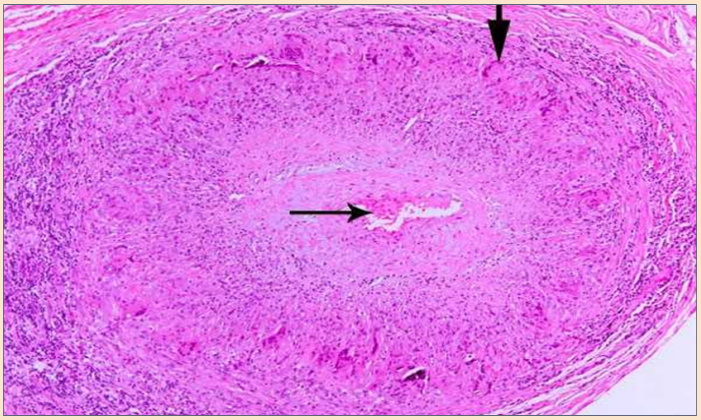
Black discoloration of fingers and toes caused by ischemia.

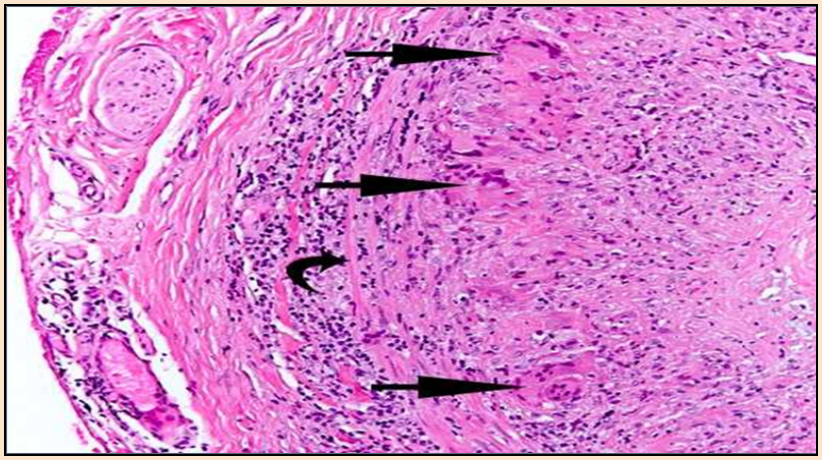
Microscopic	
	
1-Large number of small blood vessels in the dermis show occlusive organized thrombi with recanalization ***2-fibrosis around blood vessels.***	***The lumen is occluded by a thrombus containing abscesses (arrow), and the vessel wall is infiltrated with leukocytes.***

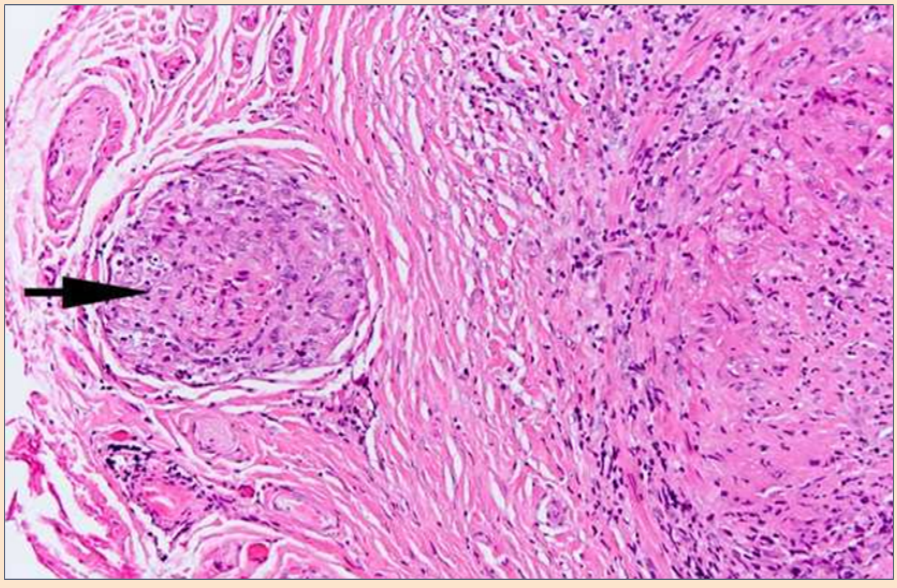
11- Giant cell (temporal) arteritis

Clinical Manifestations:

1. Fever.
2. facial pain or headache often most intense along the course of the superficial temporal artery.
3. Thickened and painful temporal artery.
4. Jaw pain.
5. Visual problems and acute vision loss.
    - Treatment: corticosteroids.

Gross	Microscopic
	
Tender and thickened scalp veins effect old people >50 yrs	1- chronic lymphocytic inflammation (in the media and adventitia) 2- Reactive intimal fibroplasias lead to luminal stenosis

Microscopic	
	
1-Disruptions of the elastic lamina with inflammation and giant cells. ***2-smooth muscle fibers (curved arrow) of vascular media.***	**1-Disruptions of the elastic lamina with inflammation and giant cells.** **2-Medial Granulom.**



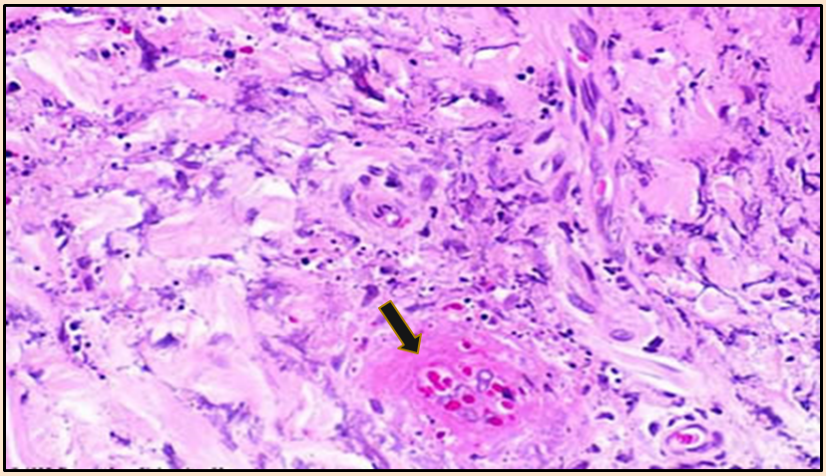
1-Single Granuloma in Adventitia.
2-both acute and chronic inflammatory cells.

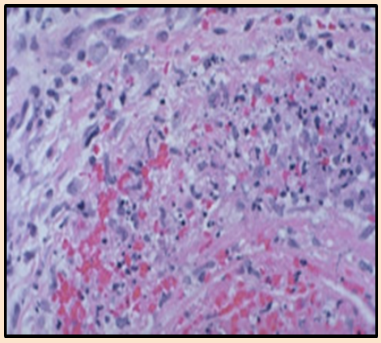
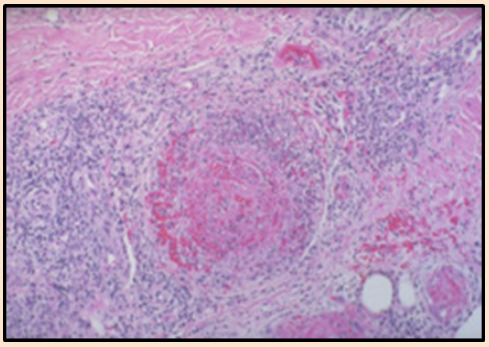
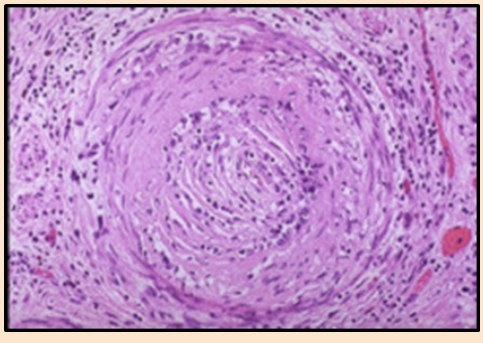
12- Microscopic Polyangiitis (Leukocytoclastic, hypersensitivity vasculitis)

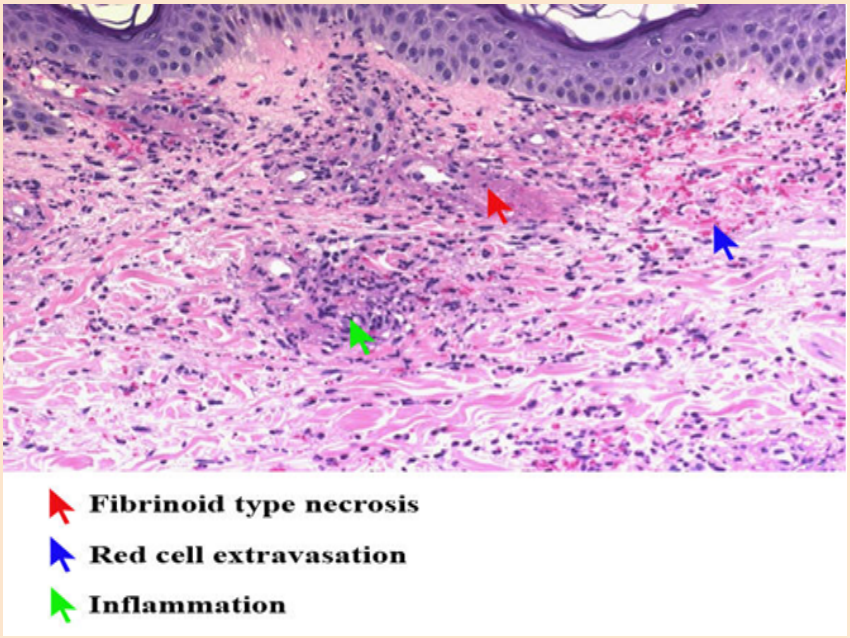
- **It is characterized by acute inflammation of small blood vessels (usually venules in the dermis).**
- **P-ANCA.**

Complications that might occur as a result of this condition.

- ***Necrotizing Glomerulonephritis.***
- ***Pulmonary capillaritis.***
- ***Gastrointestinal vasculitis.***
- ***CNS and muscle involvement.***

Gross	Microscopic
	
This erythematous and purpuric skin rash (Subcutaneous bleeding patches)	● ***Fibrinoid vascular necrosis.*** ● ***Nuclear debris.*** ● ***Neutrophilic (polymorphonuclear) infiltration.***

Microscopic		
		
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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Good Luck!

Done by:

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