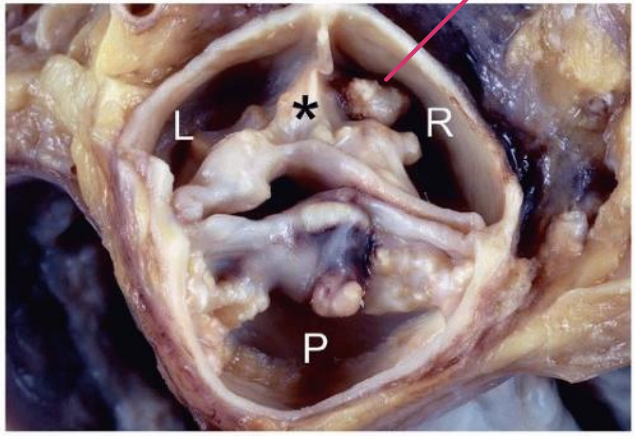
CVS MODULE PATHOLOGY LAB

Calcific Aortic Stenosis

Nodule in cusps



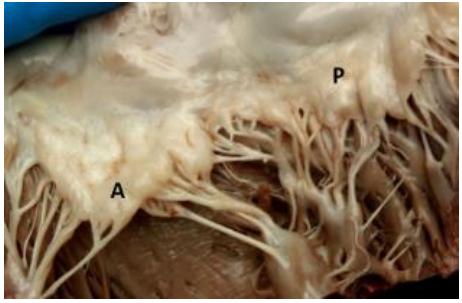
Nodular calcifications fill the cusp pockets

nodular aggregant of pink material (area of calcification)

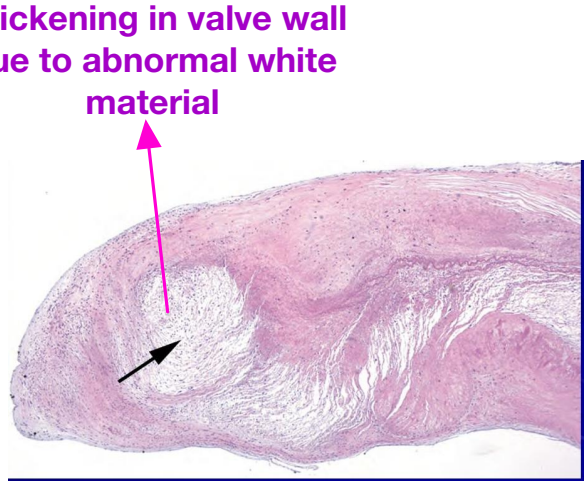
- Normal part of valve

Large nodular calcific deposits in the wall

MYXOMATOUS MITRAL VALVE



voluminous and thickened leaflets

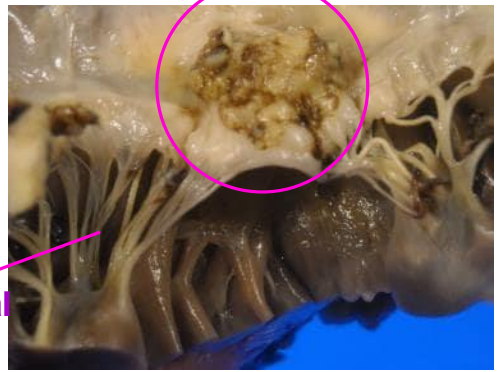


thickening and proliferation of the spongiosa with pooling of glycosaminoglycan that expands to the fibrosa.

MORPHOLOGY OF EI

Chronic associated with fibrosis & lymphocytes

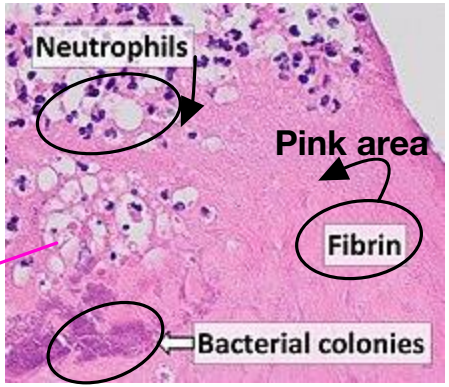
Vegetation



Normal

Large vegetation on atrial aspect of valve

Acute



Micro organisms

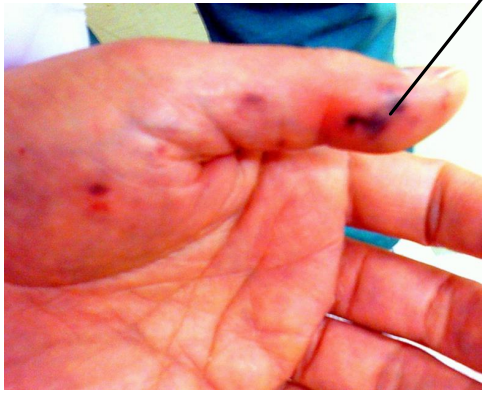
•Fresh vegetations contain platelets and fibrin with a rich infiltrate of neutrophils

* In chronic lesions, vegetations may show varying degrees of organization, vascularization and calcification

Neutrophils are replaced with lymphocytes & macrophages

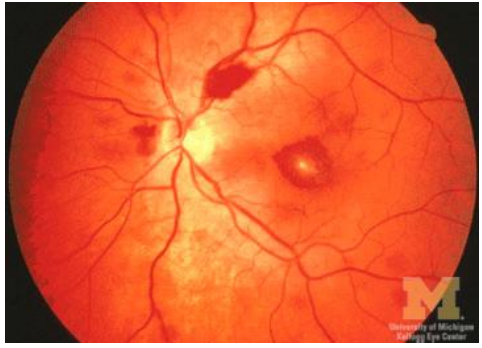
CLINICAL FEATURES OF EI.

Red lesions



Osler nodes: tender lesions found on finger pulps and thenar / hypothenar eminences

The normal is avascular



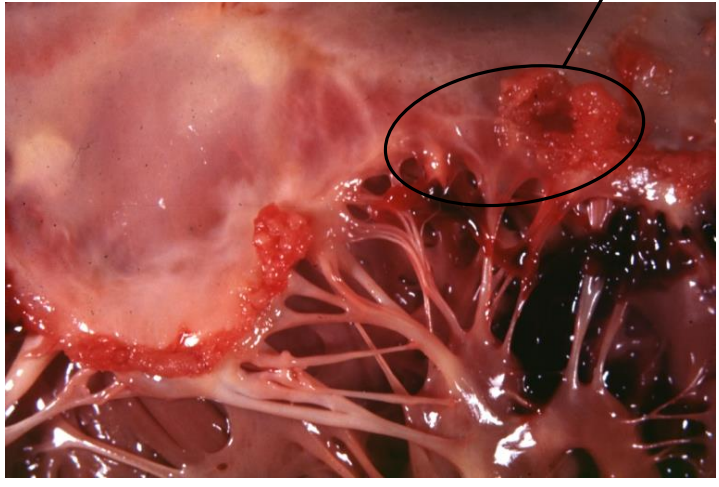
Eyes: Roth spots (boat shaped hemorrhages with pale centers, in retina) .

RHEUMATIC HEART DISEASE

MITRAL VALVULITIS

Area of inflammation + hemorrhage

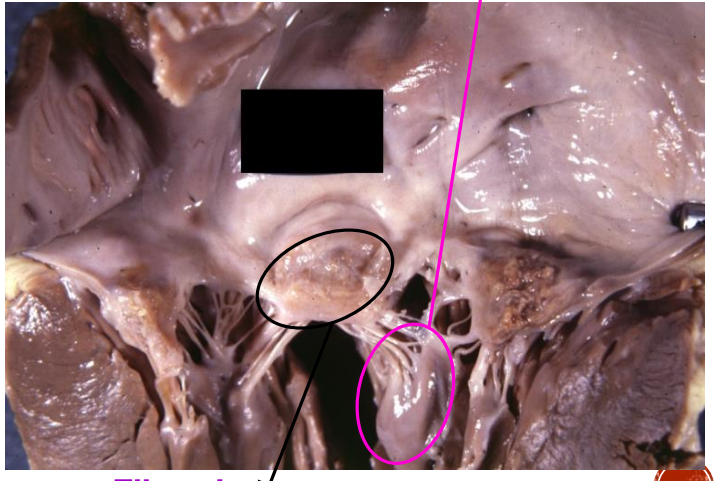
Acute (no fibrosis)



MITRAL SCARRING

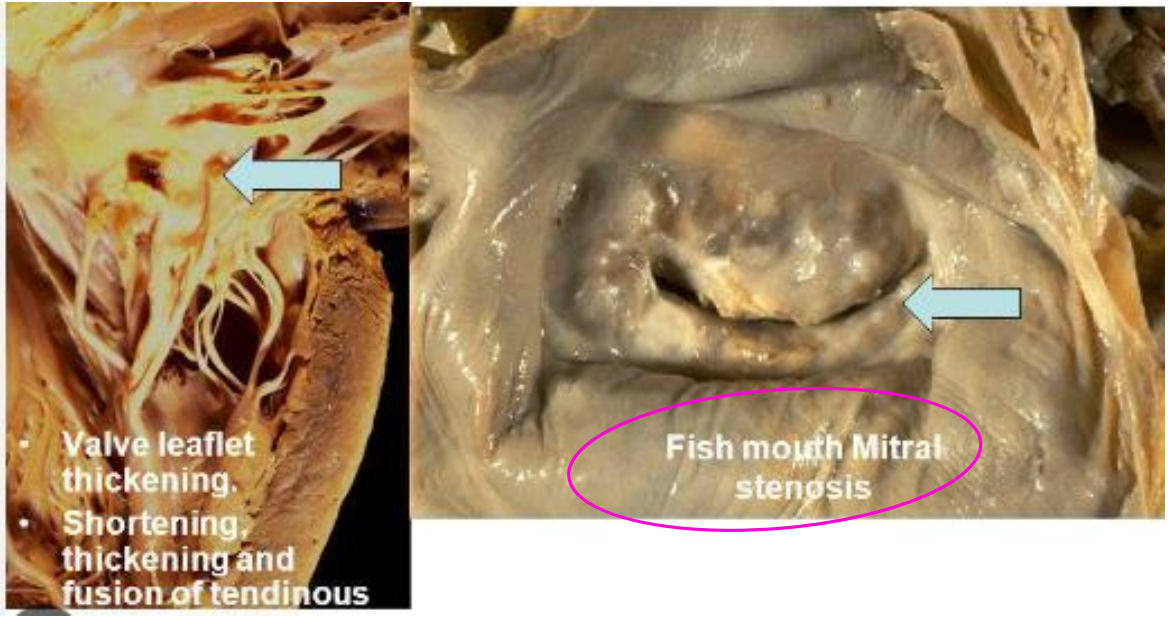
Papillary muscle

Distorted structure of mitral valve



Fibrosis

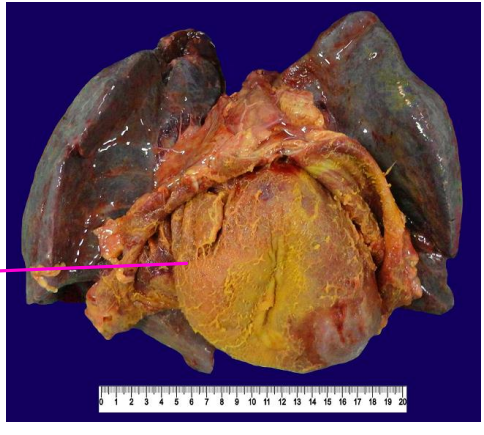
RHEUMATIC HEART DISEASE



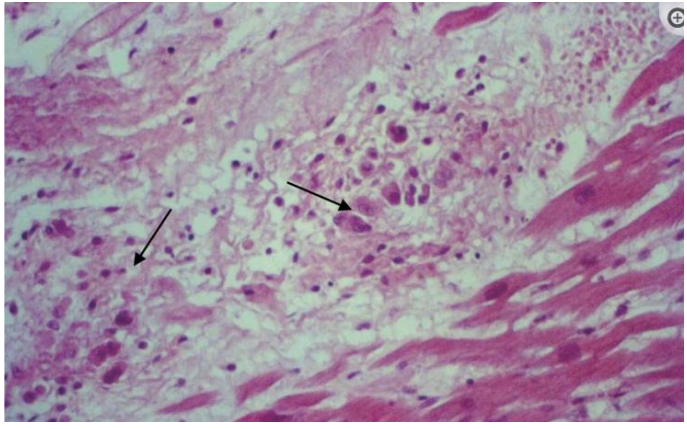
The pericardium with fibrinous exudate, seen in RHEUMATIC HEART DISEASE

Lead to restrictions pericarditis

Abnormal yellow material
No smooth muscle surface
pericardum



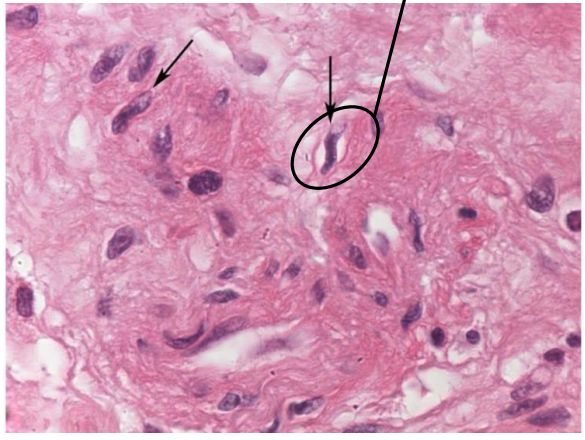
Rheumatic heart disease



Aschoff nodules

At first neutrophils then lemphocyte

Modified macrophage

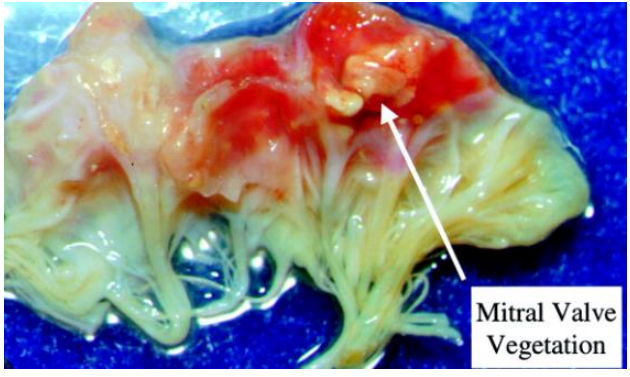


Anitschkow cells.

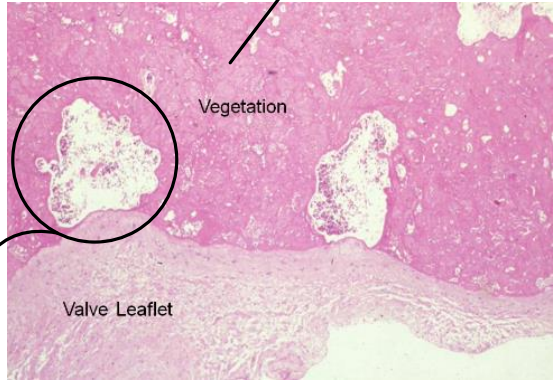
RHEUMATIC HEART DISEASE

• Valve involvement results in fibrinoid necrosis and fibrin deposition along the lines of closure forming 1- to 2-mm vegetations—verrucae—that cause little disturbance in cardiac function.

Acute preserved anatomy



Fibrin



Necrotic debris

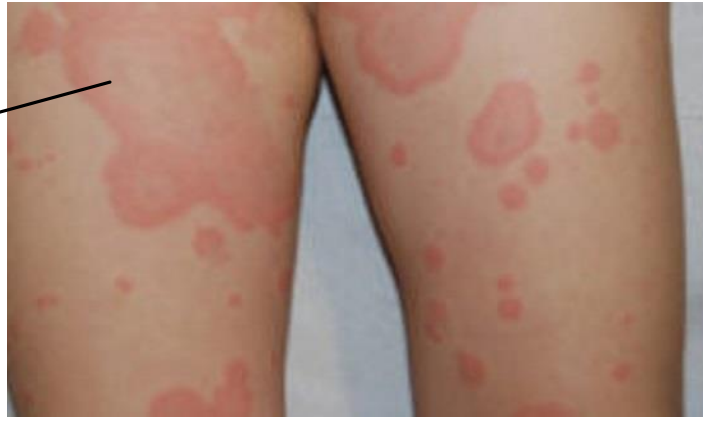
Rheumatic heart disease

The center differe than the periphery

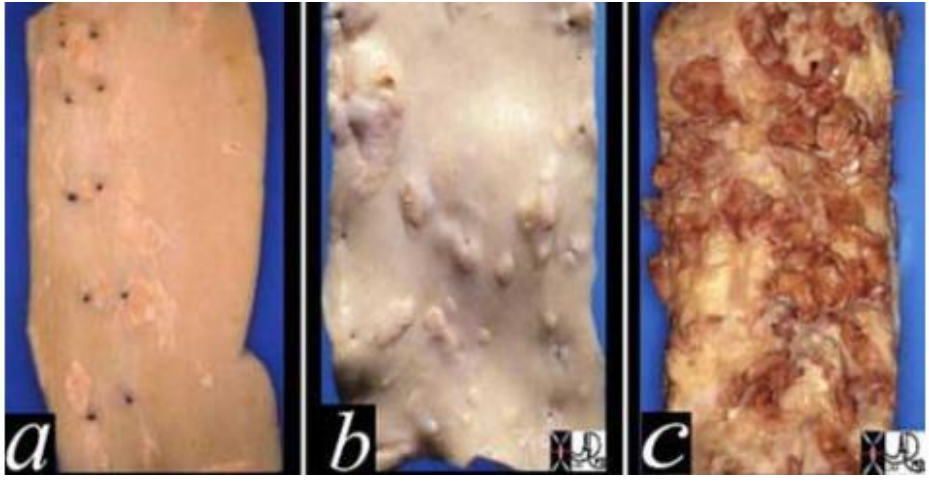
Erythema Marginatum

Caint than boundary

Well demarcated



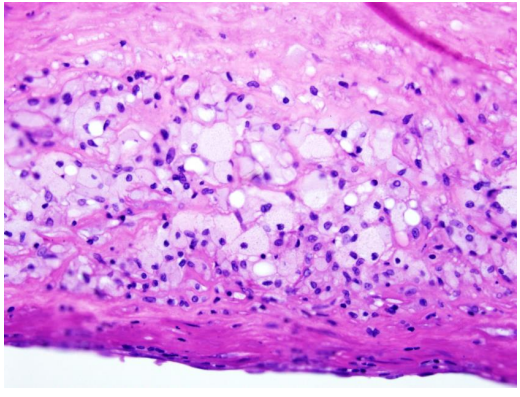
ATHEROSCLEROSIS



a. raised fatty streaks.
b. raised fibrofatty nodules
c. Rupture plaque

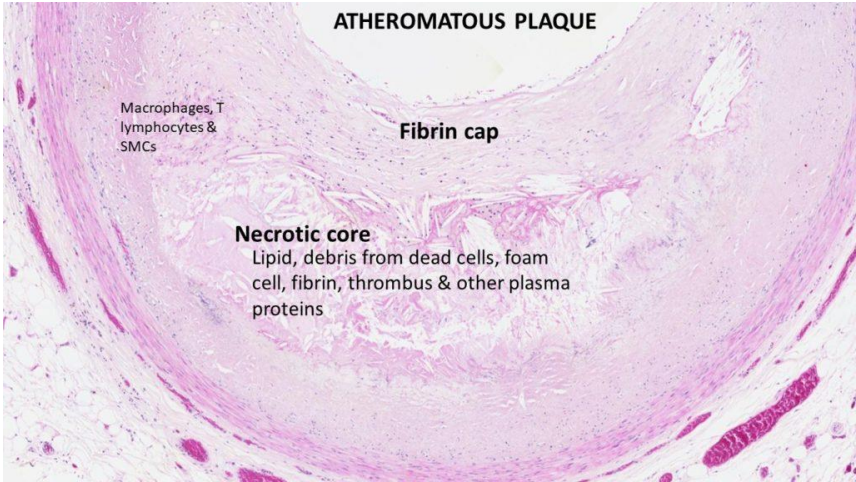
ATHEROSCLEROSIS HISTOLOGY

* The earliest lesion is the fatty streak:
They consist of many lipid-laden foam cells that contain cholesteryl esters and a variable amount of extracellular lipid



ATHEROSCLEROSIS HISTOLOGY

- Atherosclerotic plaques: have three principal components:
  - ❖cells, including SMCs, macrophages, and T cells.
  - ❖ECM, including collagen, elastic fibers, and proteoglycans.
  - ❖intracellular and extracellular lipid.



CLINICAL EXAMPLES

- Marfan syndrome result from defective synthesis of the scaffolding protein fibrillin and progressive loss of elastic tissue leading to dilation.

- Ehlers Danlos syndrome result from defective in type III collagen synthesis leading to aneurysm formation.

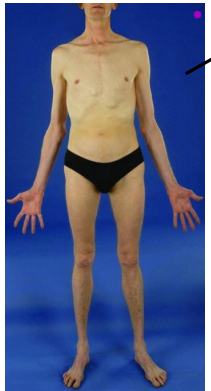
Aortic stenosis

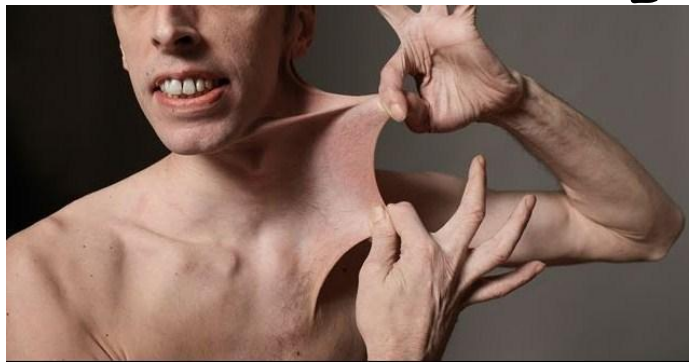
-Hyperflexibility joint

• lens dislocation

• Hyper stretchable skin

Marfan

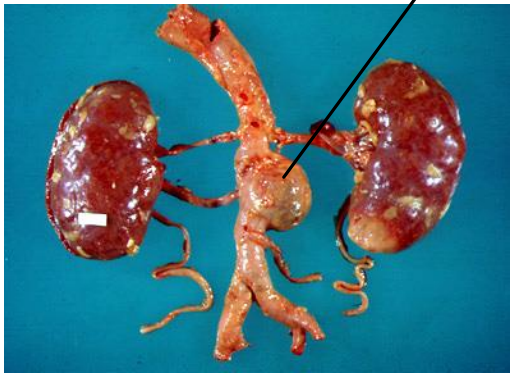




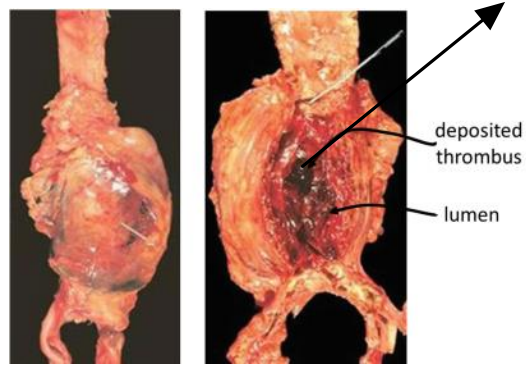
ANEURYSMS MORPHOLOGY

- The aneurysm sac usually contains bland, laminated, poorly organized mural thrombus, which can fill much of the dilated segment, usually extensive atherosclerosis is present.

- Localized dilation

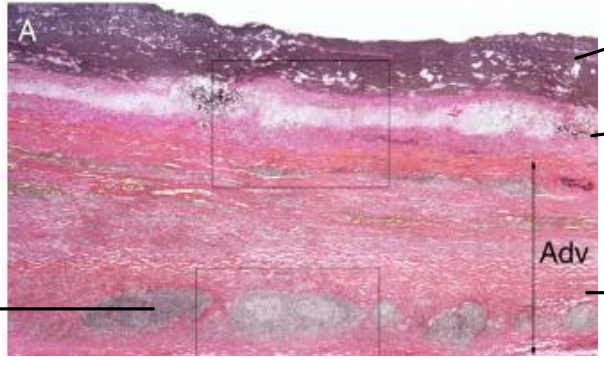


- Dence hemorrhage
- Mass thrombus



ANEURYSMS , HISTOLOGY

- Destruction of the tunica media which is thinned or no longer and replaced by hyalinized connective tissue.
- The inflammatory reaction consists primarily of lymphocytes and macrophages.
- Thrombus formation is invariably present on the luminal surface of coronary aneurysms



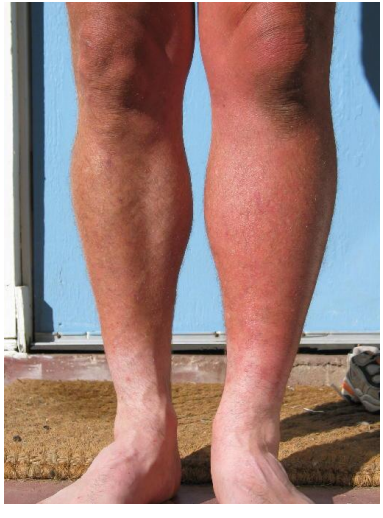
Present intima

Weak or disappeared media

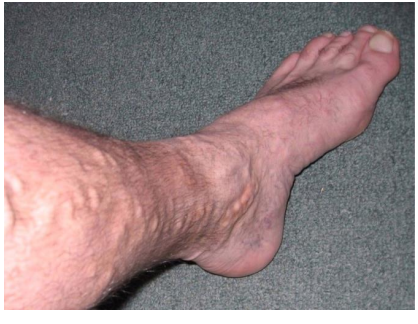
Expanded due to pooling with inflammatory cells

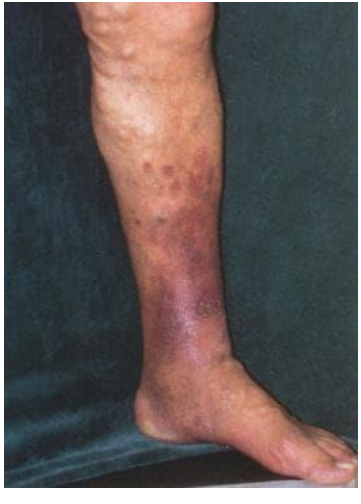
- Inflammation

DVT



VARICOSE VEIN



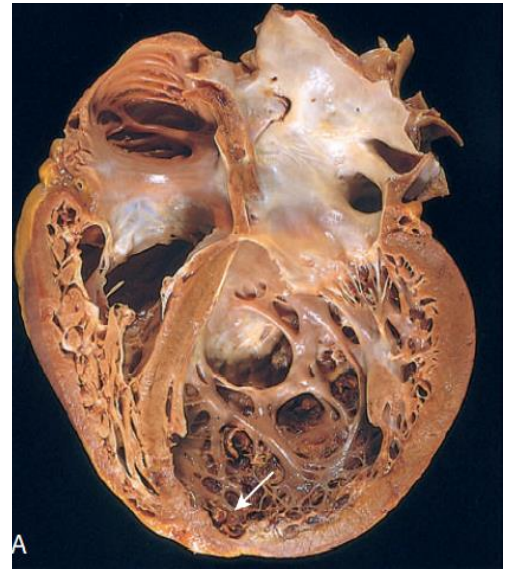


DCM

- The heart assumes a globular shape.
- ventricular chamber dilatation.
- atrial enlargement.
- Mural thrombi are often present

and may be a source of thromboemboli.

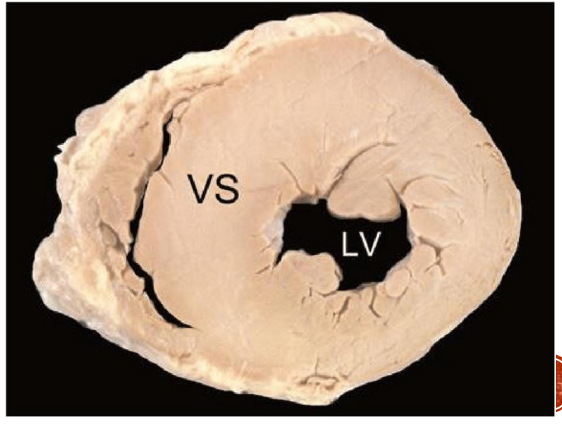
- **We found thrombus**



HCM

- Hypertrophic cardiomyopathy is marked by massive myocardial hypertrophy without ventricular dilation.

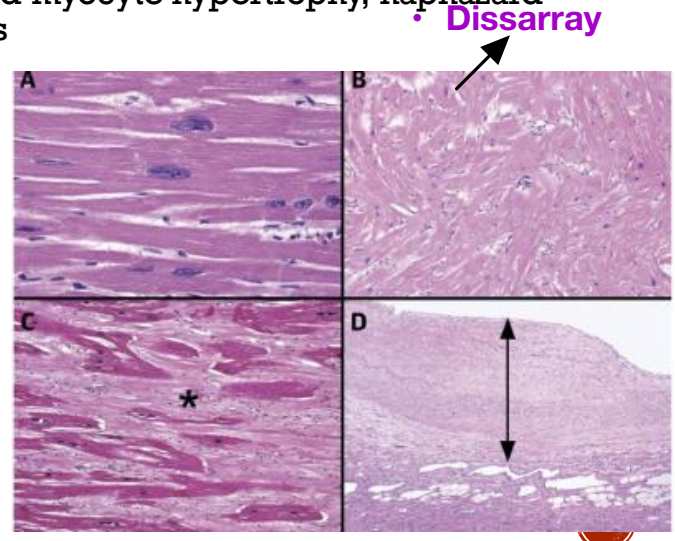
Hypertrophy may be complication of HTN



HISTOLOGICAL FEATURES OF HCM

- The characteristic histologic features in HCM are marked myocyte hypertrophy, haphazard myocyte (and myofiber) disarray, and interstitial fibrosis

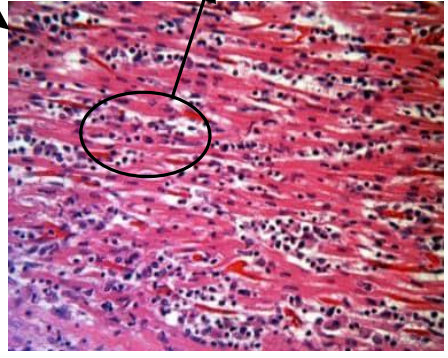
- A) Myocyte hypertrophy.
- (B) myocyte disarray.
- (C) interstitial (pericellular-type) fibrosis (asterisk).
- (D) endocardial fibrosis (double-headed arrow).

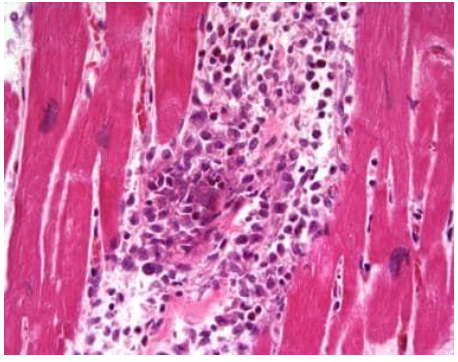


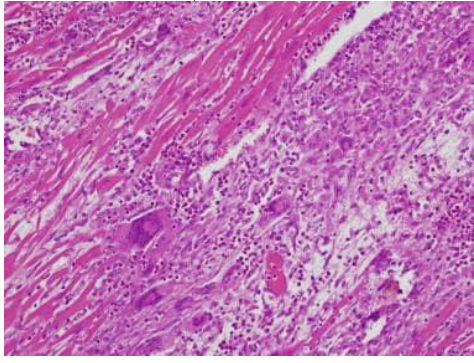
HISTOLOGICAL FEATURES OF MYOCARDITIS

- edema and myocyte injury.
- interstitial inflammatory infiltrates:
- Lymphocytic type: numerous lymphocytes.
- hypersensitivity myocarditis: abundant eosinophils.
- Giant cell myocarditis: containing multinucleate giant cells

- **Lemphocyte**



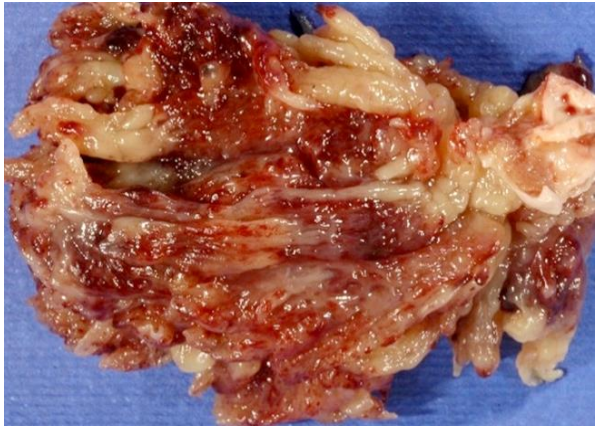




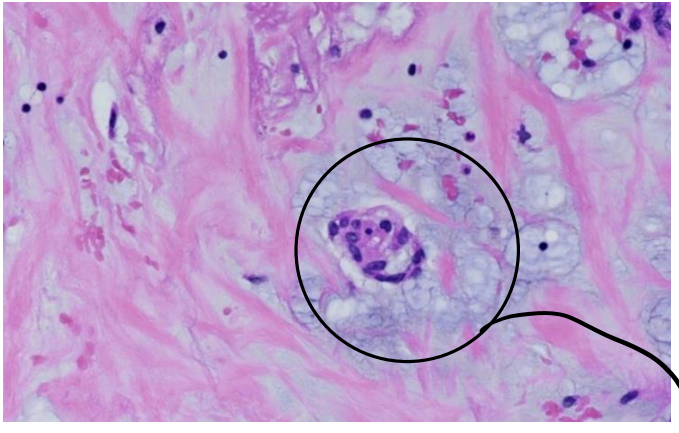
Most common benign tumor of heart—> mitral valve obstruction as fatal complication

CARDIAC MYXOMA MORPHOLOGY

- Left atrium



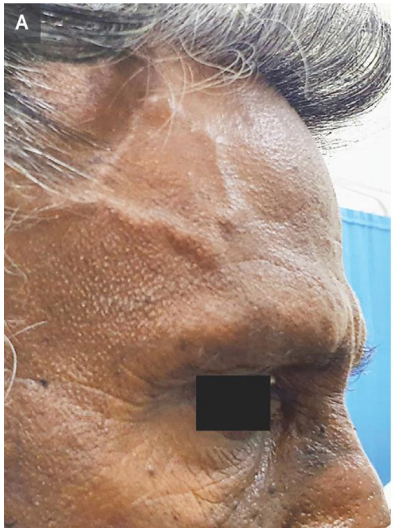
Grossly : appear as sessile or pedunculated mass.

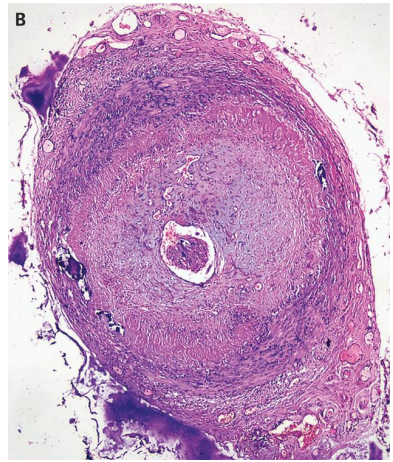


Microscopic: neoplastic cells within myxoid stroma

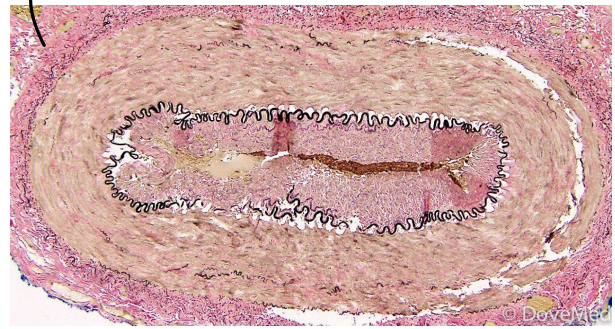
GIANT CELL (TEMPORAL) ARTERITIS

- Transmural inflammation pattern with lymphocytes, giant cells and macrophages arranged in concentric rings, surrounding the external and internal elastic lamina, the later is disrupted as viewed by- an elastic stain.



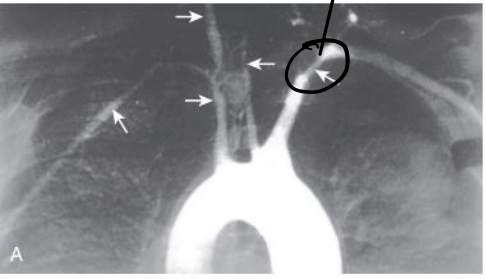


Internal elastic lamina defect

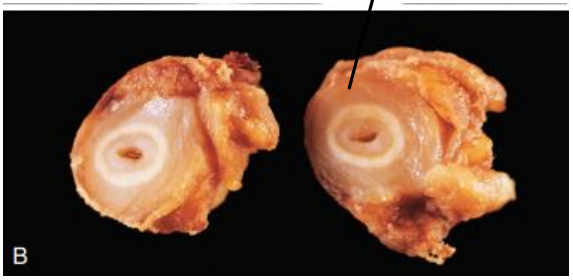


TAKAYASU ARTERITIS (PULSELESS DISEASE).

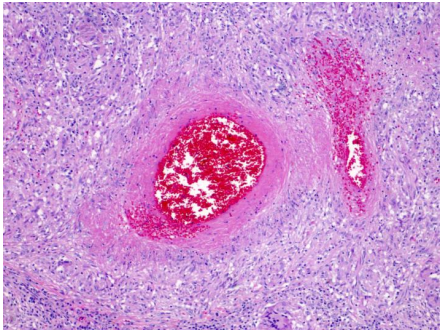
Area of stenosis



Thickening of wall (grossly)



- Cuffing around vasa vasourum

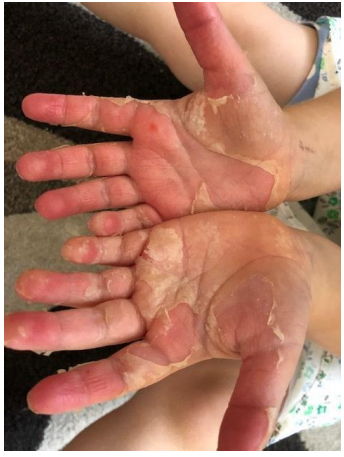


transmural (including the adventitia) mononuclear inflammation

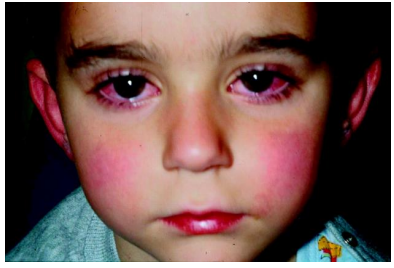
- Lemphocyte

KAWASAKI DISEASE

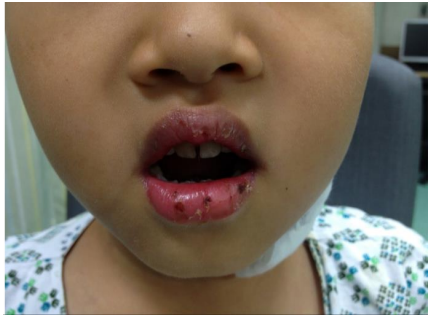
- Peeling of skin



- Red eye

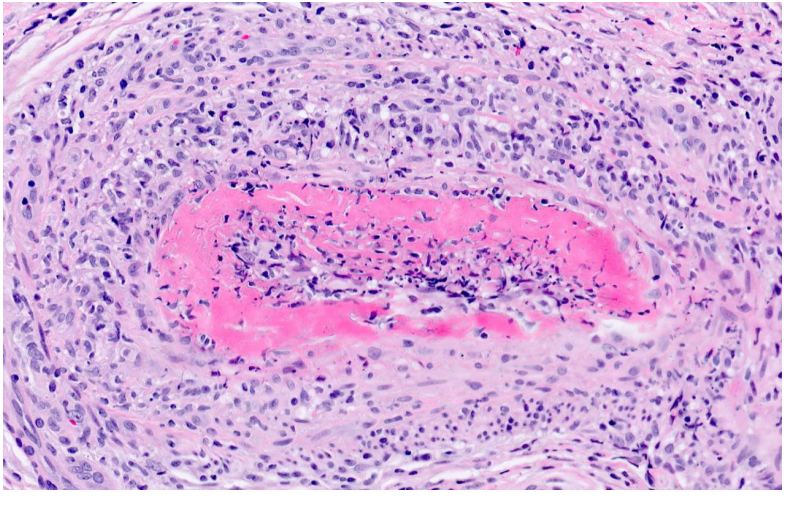


- Cracked lips



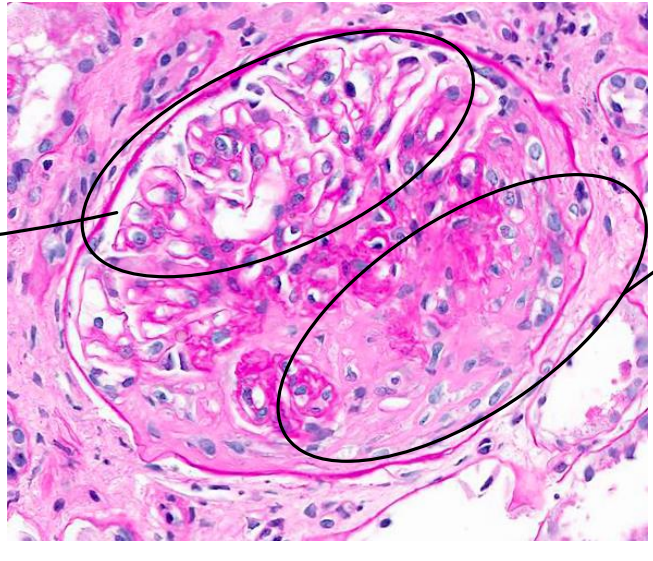
LEUKOCYTOCLASTIC VASCULITIS

- Nuclear necrosis
- Nuclear debri



GRANULOMATOSIS WITH POLYANGIITIS CRESCENTIC GLOMERULONEPHRITIS.

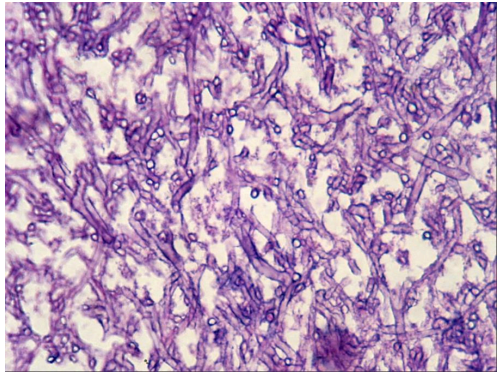
- Normal capillary



- No capillary loop
- Crecentric shape

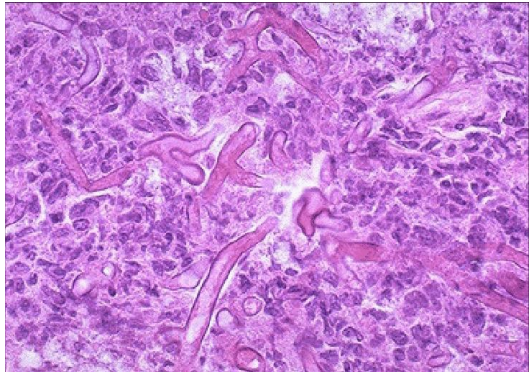
INFECTIOUS VASCULITIS

- Thin hype
- Sepatated
- Branching in acute angle



Aspergillus

- Broad hype
- Non sepataed
- Branching 90 degree



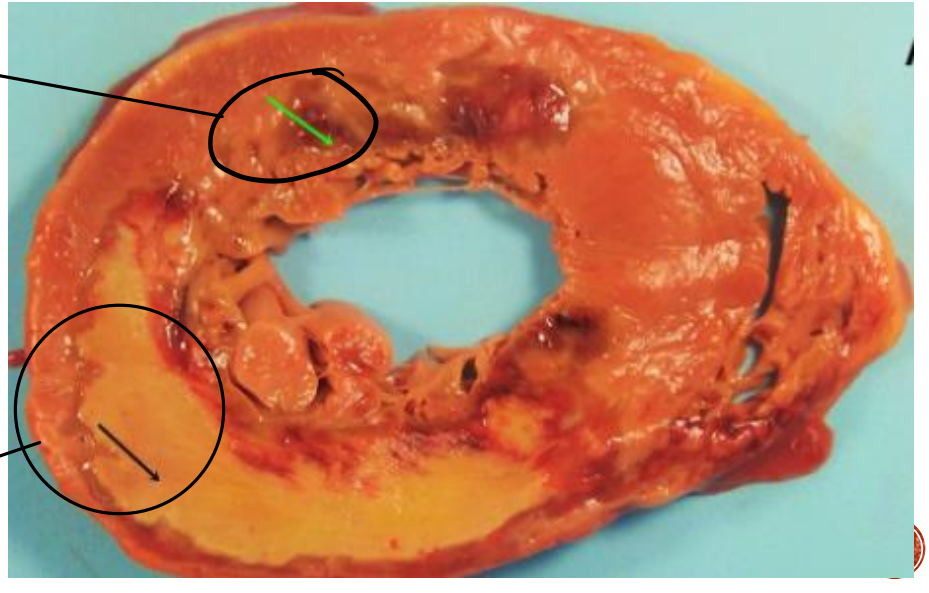
Mucor mycosis

GROSS MORPHOLOGY OF MI

- **Hemorraghe**

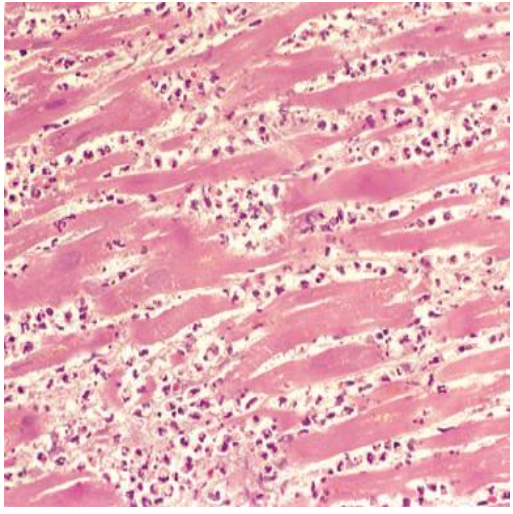
postmortem autopsy revealed areas of Coagulative necrosis.

- **Necrosis**



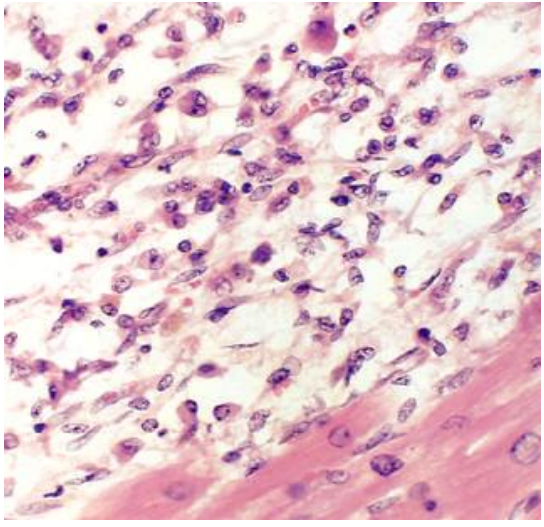
HISTOPATHOLOGICAL FEATURES OF MI

- 1-3 days



Coagulation necrosis with loss of nuclei and striations; interstitial infiltrate of neutrophils

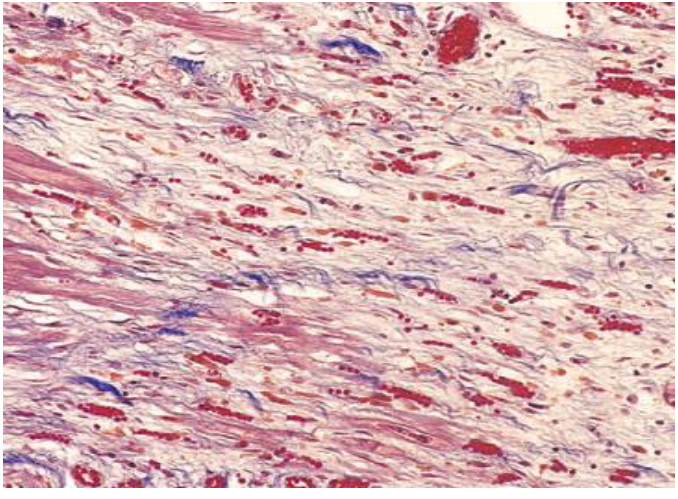
- 7-10 days



Complete removal of necrotic myocytes by phagocytic macrophages

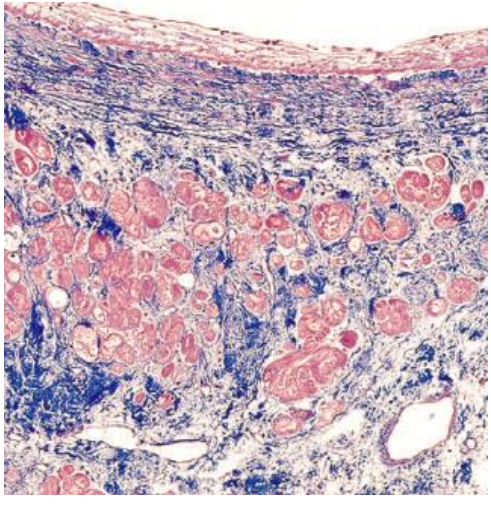
Histopathological features cont.

- 10-14 days



well established granulation tissue with new blood vessels & collagen deposition.

- More than 2 month

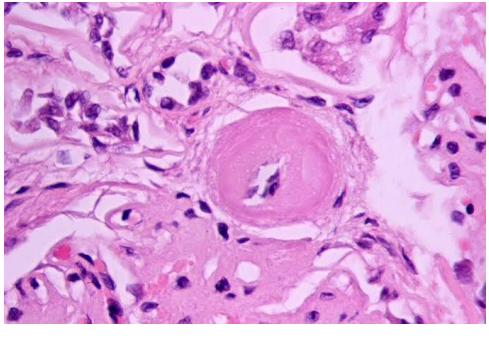


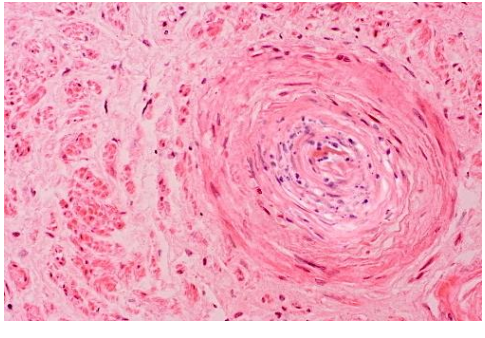
Dense collagenous scar

HYPERTENSION-RELATED SMALL BLOOD VESSEL DISEASE

- 1-Hyaline arteriolosclerosis: associated with benign hypertension.
- It is marked by homogeneous, pink hyaline thickening of the arteriolar walls, with loss of underlying structural detail, and luminal narrowing.

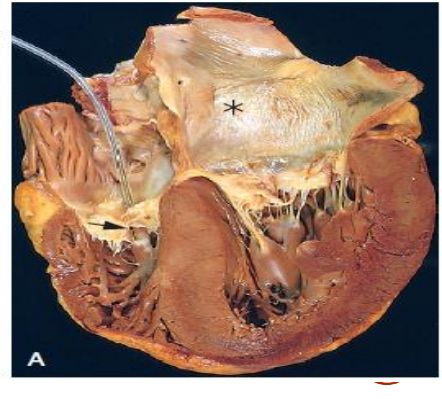
- 2. Hyperplastic arteriolosclerosis: Associated with severe hypertension.
  - Vessels exhibit "onionskin," concentric, laminated thickening of arteriolar walls and luminal narrowing.
- The laminations consist of smooth muscle cells and thickened, reduplicated basement membrane.





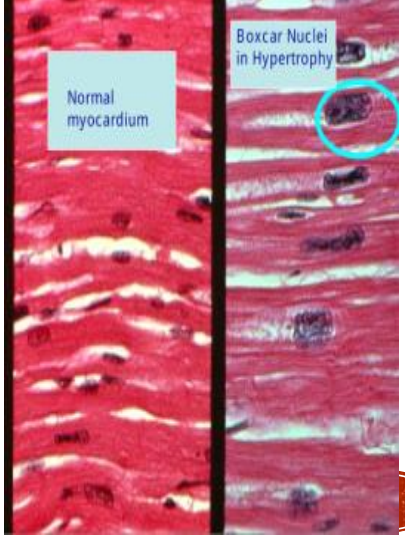
CARDIAC MORPHOLOGY IN SYSTEMIC HTN

- left ventricular hypertrophy:
- heart weight can exceed 500 g, left ventricular wall thickness can exceed 2.0 cm.
- left atrial dilation : due to increased left ventricular wall thickness that impairs diastolic filling .



MICROSCOPICALLY

- The transverse diameter of myocytes is increased with prominent nuclear enlargement and hyperchromasia (“boxcar nuclei”).
- Intercellular Fibrosis.



MORPHOLOGY OF COR PULMONALE

- In acute cor pulmonale, the right ventricle usually shows only dilation; if an embolism causes sudden death, the heart may even be of normal size.
- Chronic cor pulmonale is characterized by right ventricular (and often right atrial) hypertrophy.
  - The right ventricle is markedly dilated and hypertrophied with a thickened free wall and hypertrophied trabeculae.