Pathology lab 2

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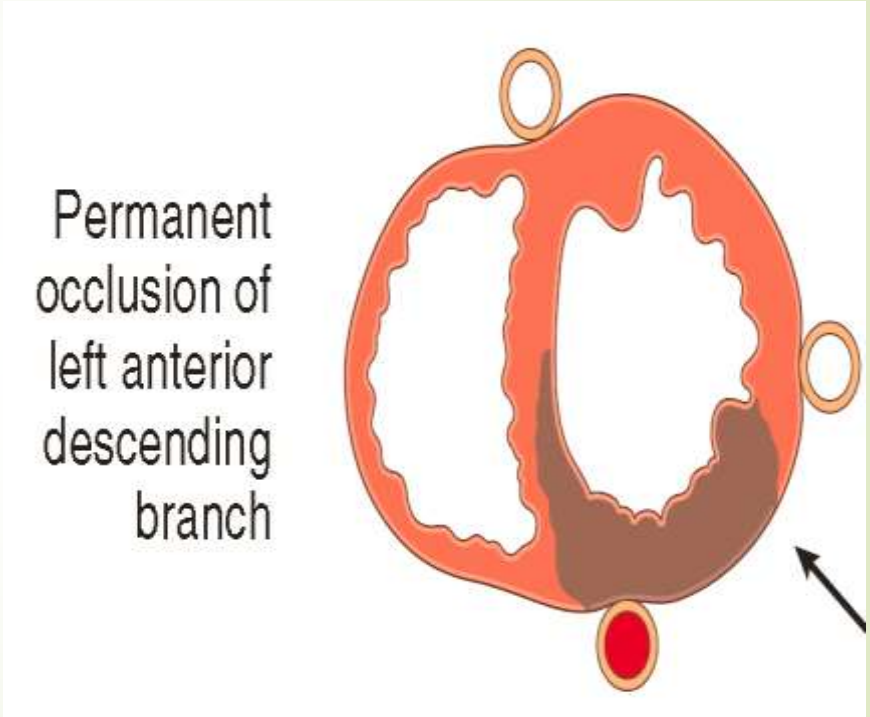
MI - Patterns of Infarction *(distribution)*

Acute occlusion of the proximal left anterior descending (LAD) artery causes 40%-50% of all MIs & typically results in infarction of anterior wall of left ventricle, anterior two thirds of ventricular septum, & most of the heart apex.



MI - Patterns of Infarction (size of vessel & collateral)

- **Transmural infarctions:** involve the full thickness of the ventricle & are caused by epicardial vessel occlusion (without therapeutic intervention).
- typically yield ST segment elevations on (ECG) .
- Called *ST-segment elevated MIs (STEMIs)*.

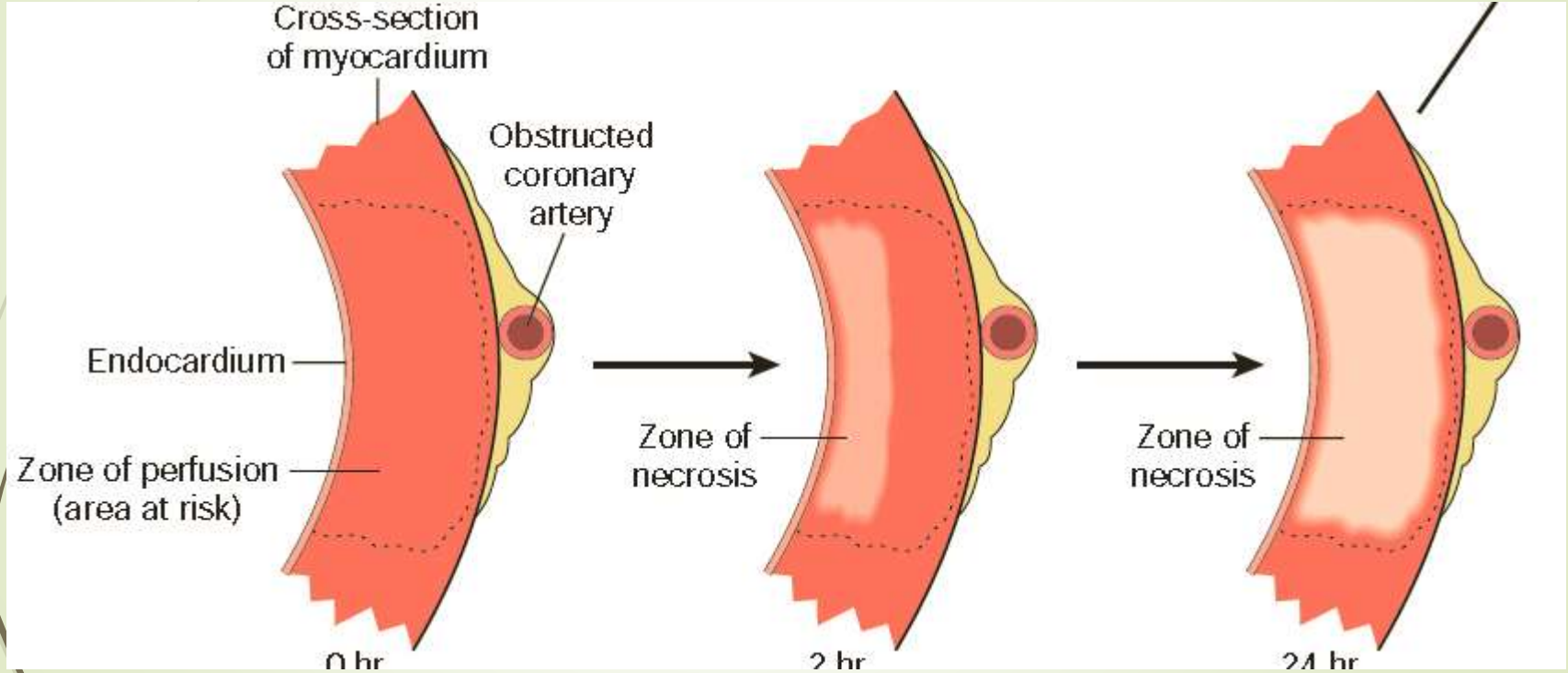
MI - Patterns of Infarction (size of vessel & collateral)

- **Subendocardial infarctions**: limited to the inner third of myocardium.
- No ST segment elevations on ECG "non–ST-segment elevated MIs".
- The most vulnerable region to hypoperfusion & hypoxia→ most distal to the epicardial vessels).

Causes **1.** Transient decreases in oxygen delivery (hypotension, anemia, or pneumonia) or increases in oxygen demand (tachycardia or hypertension) can cause subendocardial ischemic injury in CAD w/o thrombus.

2. Or an occlusive thrombus lyses before a full-thickness infarction.

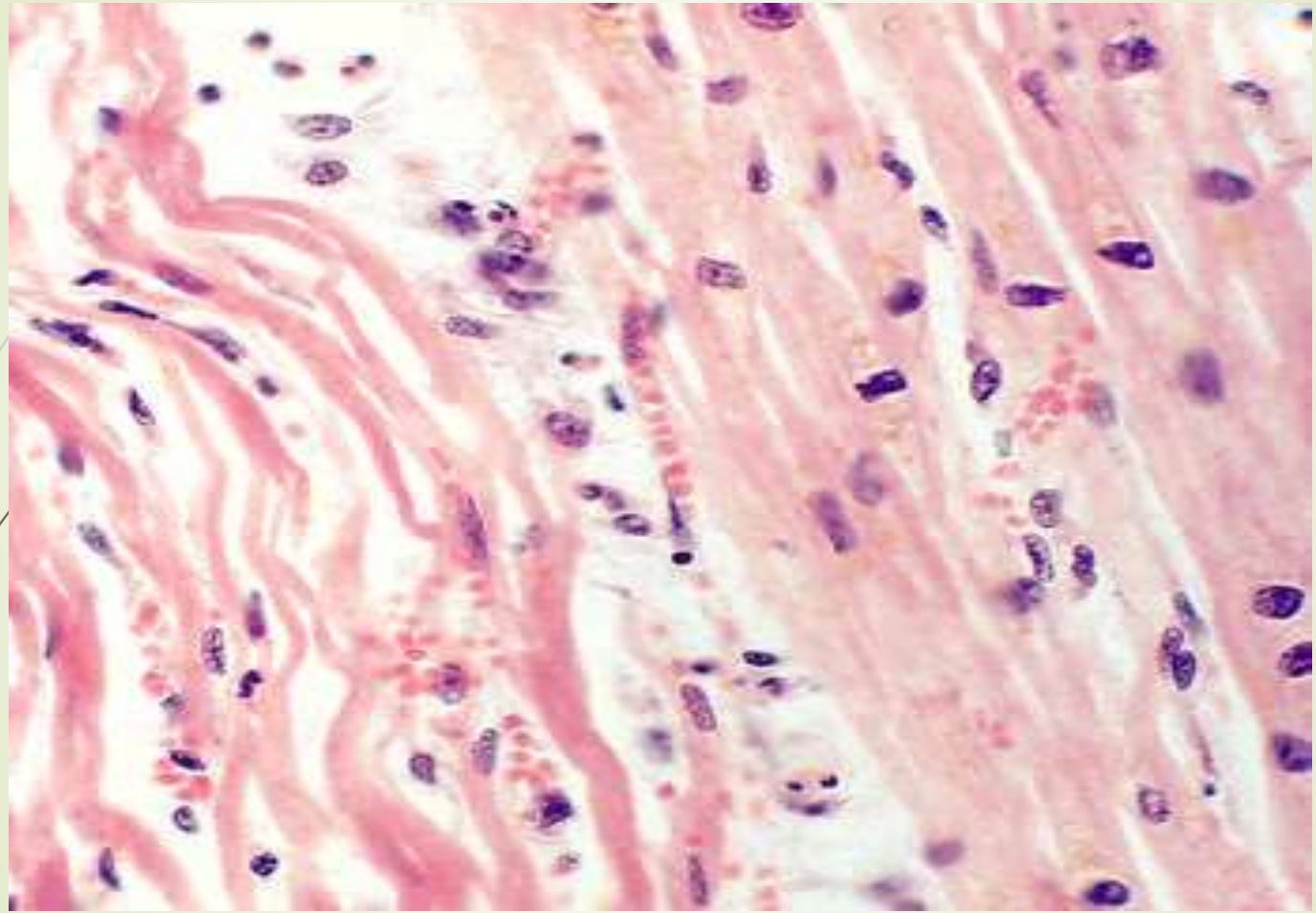
MI - Patterns of Infarction (size of vessel & collateral)



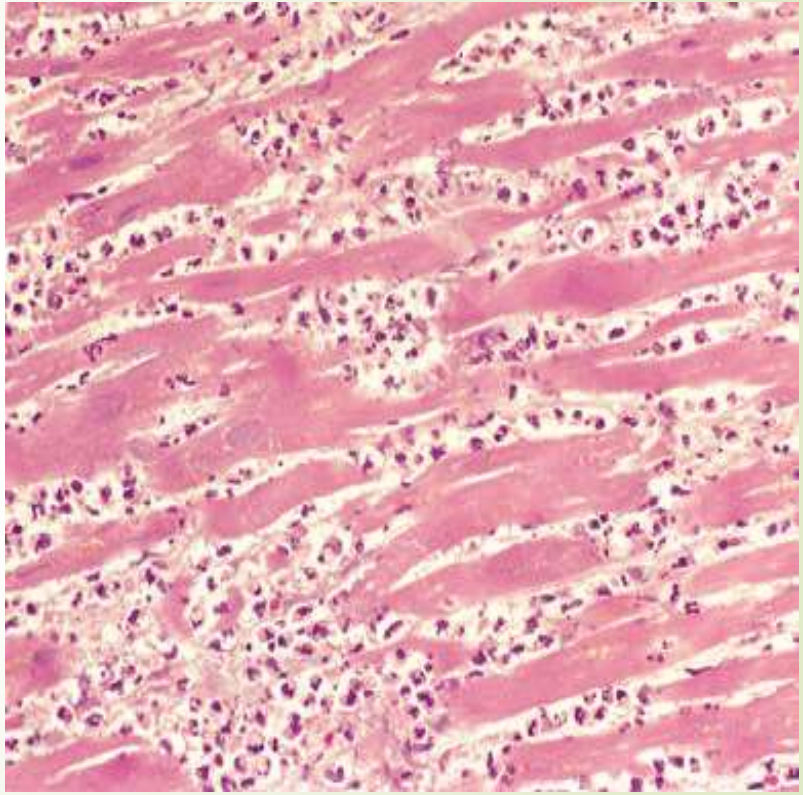
MORPHOLOGY

The gross and microscopic appearance of an MI depends on the age of the injury:

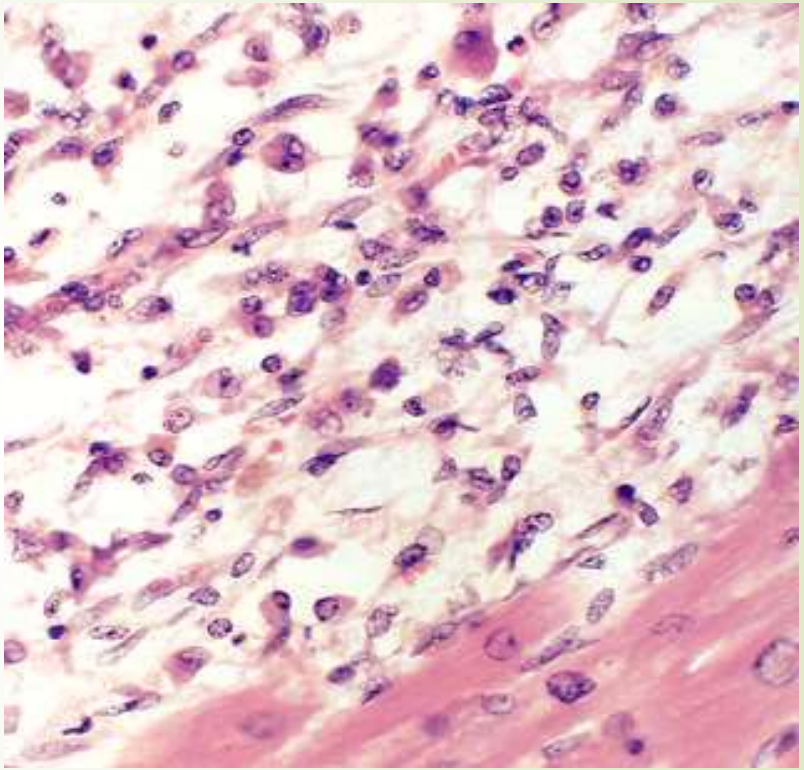
- **After 20-30 minutes**: **irreversible injury** → cell death.
- **4 hours:** only on E.M.: Sarcolemmal disruption; The earliest detectable feature of myocyte necrosis.
- **6-12** hours **L.M.,:** beginning of wavy fibers
- **12 to 24** hours **grossly** : an infarct usually can be identified by a red-blue discoloration caused by stagnated, trapped blood(dark mottling)



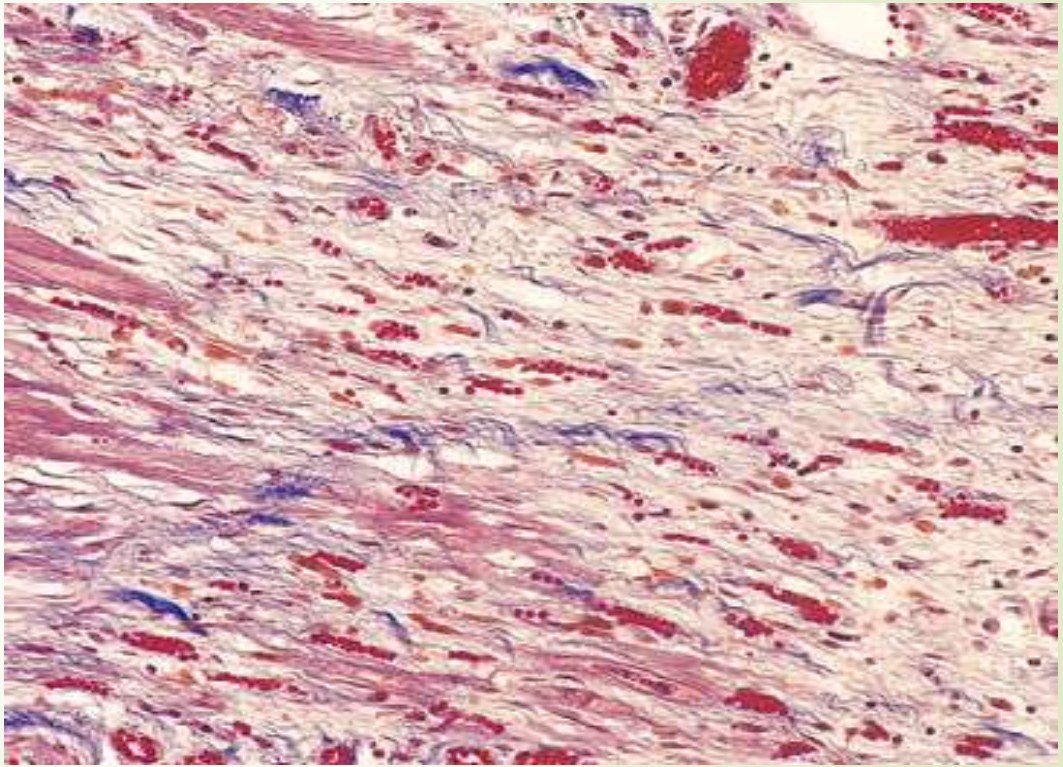
- **1–3 days:**
- yellow-tan infarct center **Grossly**,
- & Coagulation necrosis with loss of nuclei and striations; interstitial infiltrate of neutrophils on **L.M.**



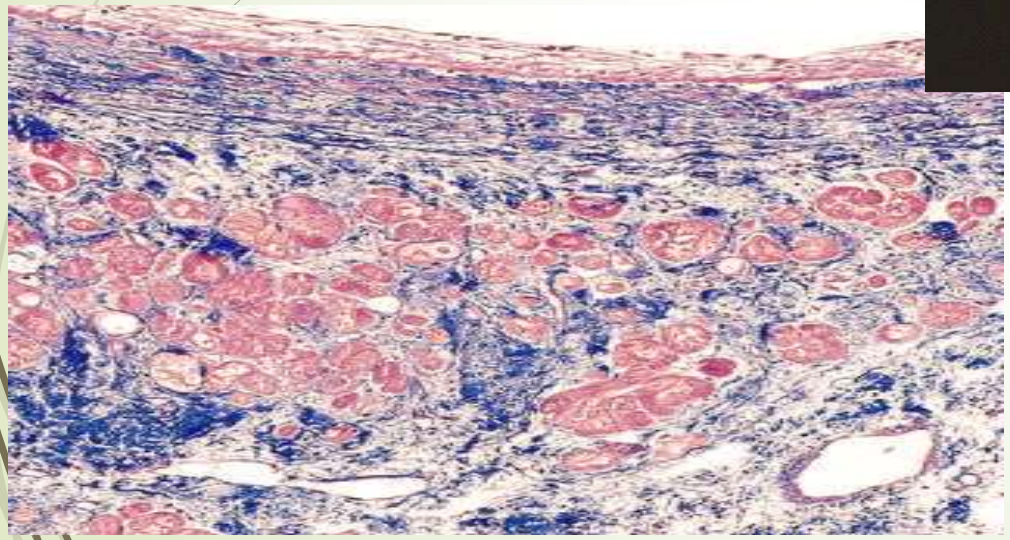
- **L.M.**: Complete removal of necrotic myocytes by phagocytic macrophages **(7 to 10 days).**



- **10-14 days: L.M.**: well established granulation tissue with new blood vessels & collagen deposition.



- **Within 2-8 weeks**; **Grossly** gray white scar progressive from the periphery towards the center of the infarct.



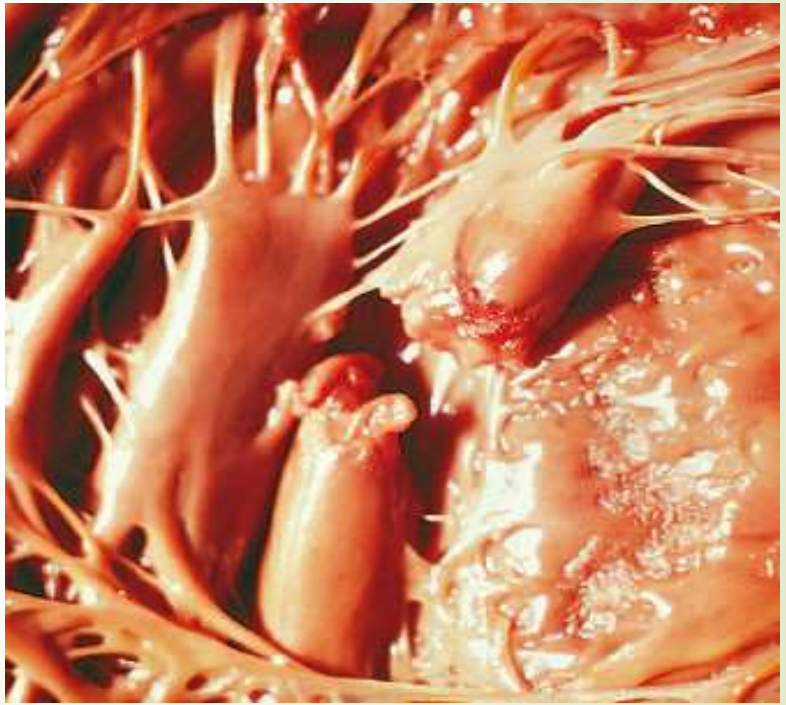
Microscopically:
Healed MI (collagenous scar)

MI – Clinical features

- **Severe retrosternal pain radiate to the neck, jaw, epigastrium, or left arm.**
- Not relieved by rest or vasodilators, may persist for several hours (>20-30 min) .
- Nausea, vomiting sweating & weakness may be accompanying symptoms.

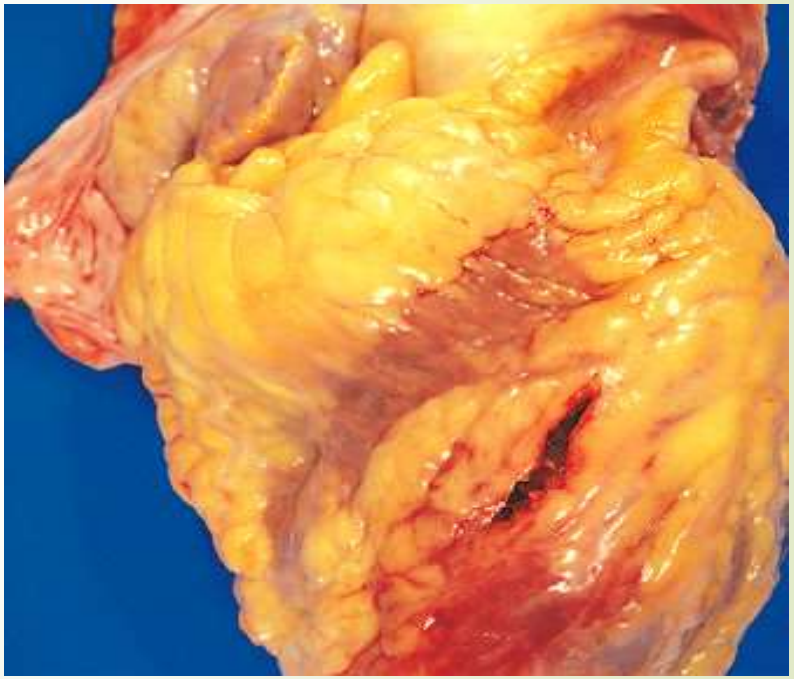
Consequences and Complications of MI

- ***Papillary muscle dysfunction*:**
- ***They*** rupture infrequently after MI..
- but they often are dysfunctional & poorly contractile as a result of ischemia.



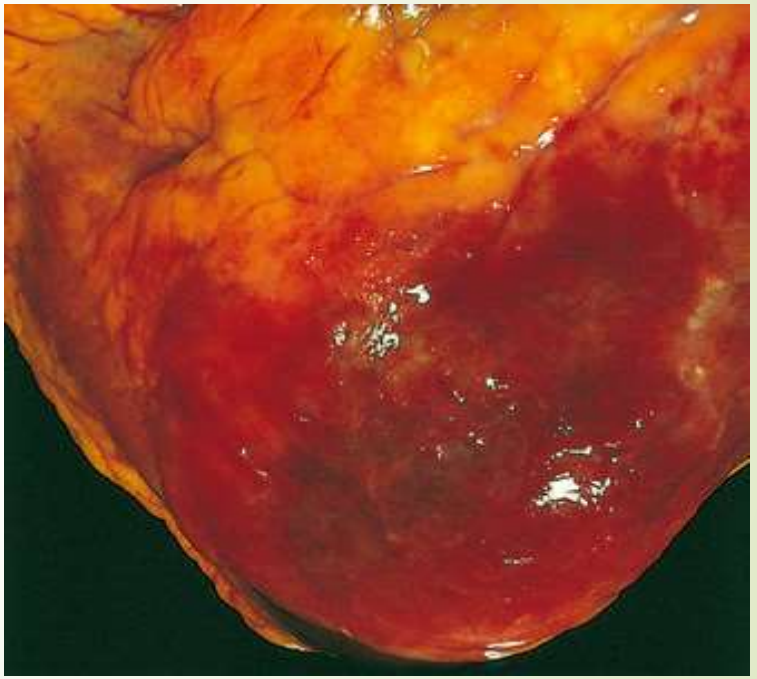
Consequences and Complications of MI

- ***Myocardial rupture***. 1-5% of MIs but is frequently fatal when it occurs.
- Left ventricular free wall rupture is most common.
- Rupture occurs most commonly in 3 to 7 days after infarction → healing process → lysis of necrotic myocardium is maximal & infarct has been converted to soft, friable granulation tissue.



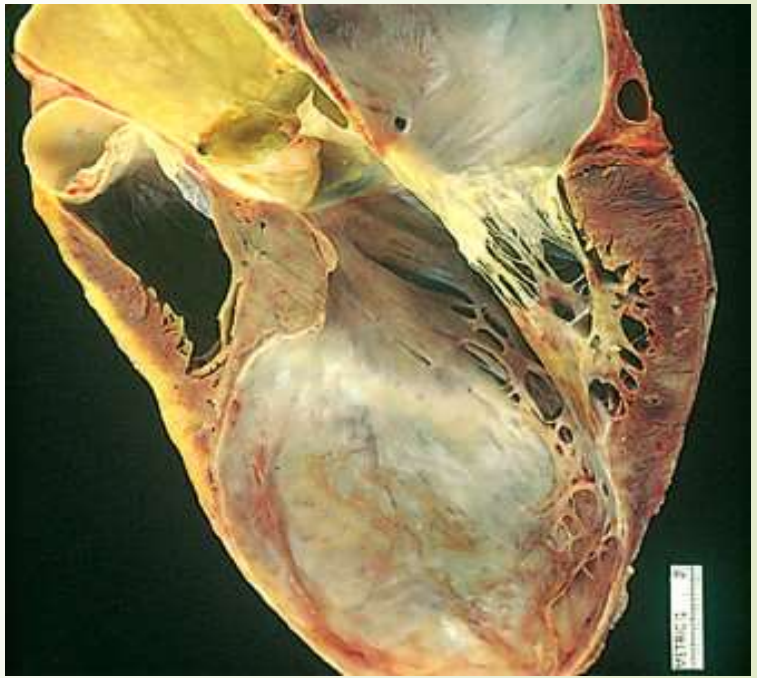
Consequences and Complications of MI

- ***Pericarditis***. Transmural MIs can elicit a fibrinohemorrhagic pericarditis.
- Typically appears 2 to 3 days after infarction and then gradually resolves over the next few days



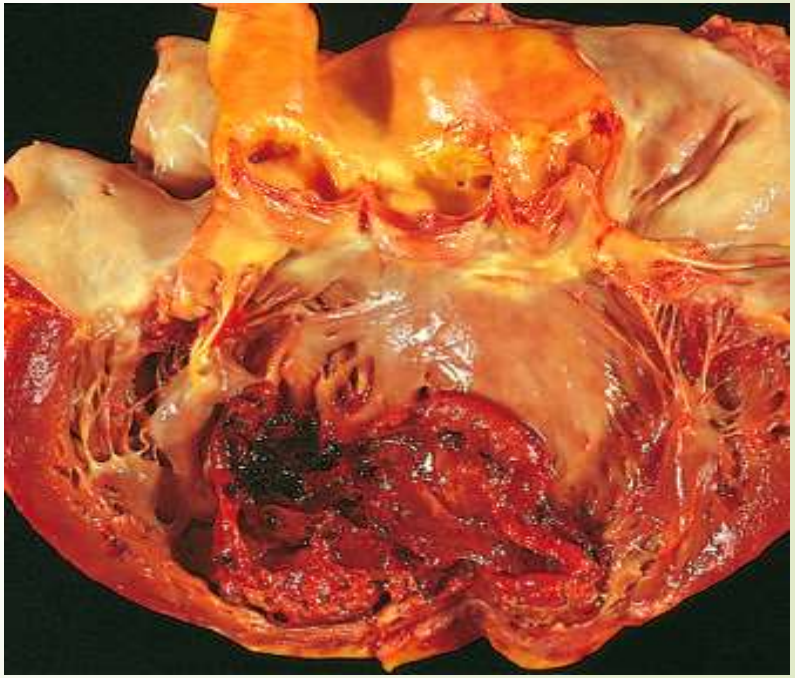
Consequences and Complications of MI

- ***Ventricular aneurysm.*** A late complication, aneurysms of the ventricle most commonly result from a large transmural infarct that heals with the formation of a thinned wall of scar tissue, usually they do not rupture.



Consequences and Complications of MI

- ***Mural thrombus.*** With any infarct, the combination of attenuated myocardial contractility (causing *s*tasis), chamber dilation, & endocardial damage (causing a thrombogenic surface) can foster *mural thrombosis* eventually leading to left-sided *thromboembolism*.



Vasculitis

Classification of Vasculitis

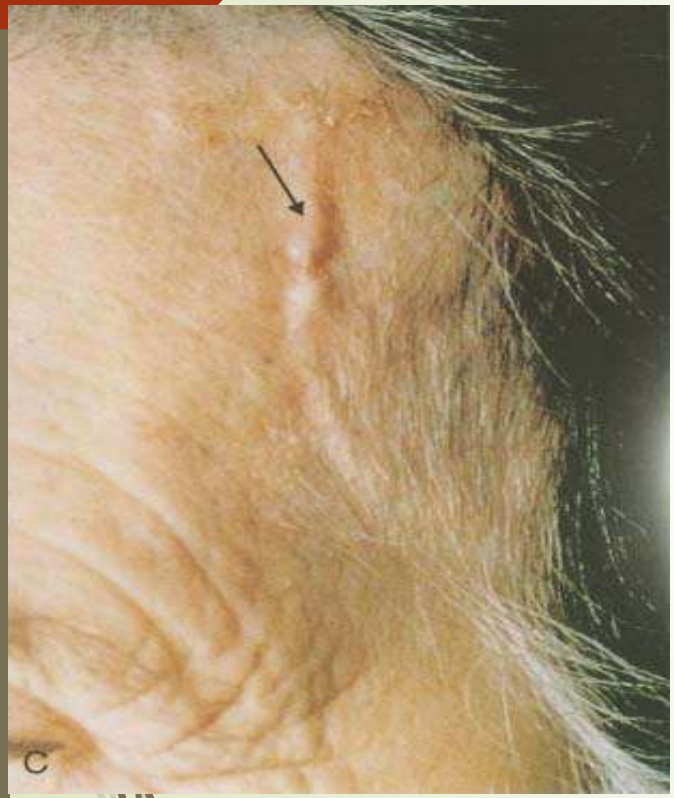
Vessel	Disease	summary
Large	Giant-cell arteritis	F, >50. Arteries of head.
	Takayasu arteritis	F, <40. “Pulseless disease”
Medium	Polyarteritis nodosa	Young adults. Widespread.
	Kawasaki disease	<4. Coronary disease. Lymph nodes.
Small	Wegener granulomatosis	Lung, kidney. c-ANCA.
	Churg-Strauss syndrome	Lung. Eosinophils. Asthma. p-ANCA.
	Microscopic polyangiitis	Lung, kidney. p-ANCA.

Temporal (giant cell, cranial) arteritis

- **Histology**
  - granulomatous inflammation
  - chronic non-specific panarteritis
  - fragmentation of the internal elastic lamina
- **Pathogenesis**
  - T Cell mediated immunity

Temporal (giant cell, cranial) arteritis

- **Clinical features**
  - Females > males (~X4)
  - > 50 years (rare before 50 years of age)
  - non-specific constitutional symptoms
  - Headache, local facial pain & tenderness
  - ocular symptoms, 50 % (range from diplopia to blindness)
- **Diagnosis**
  - Biopsy (at least 1cm)→ Could be negative (very focal involvement)
  - Rx: corticosteroids are effective.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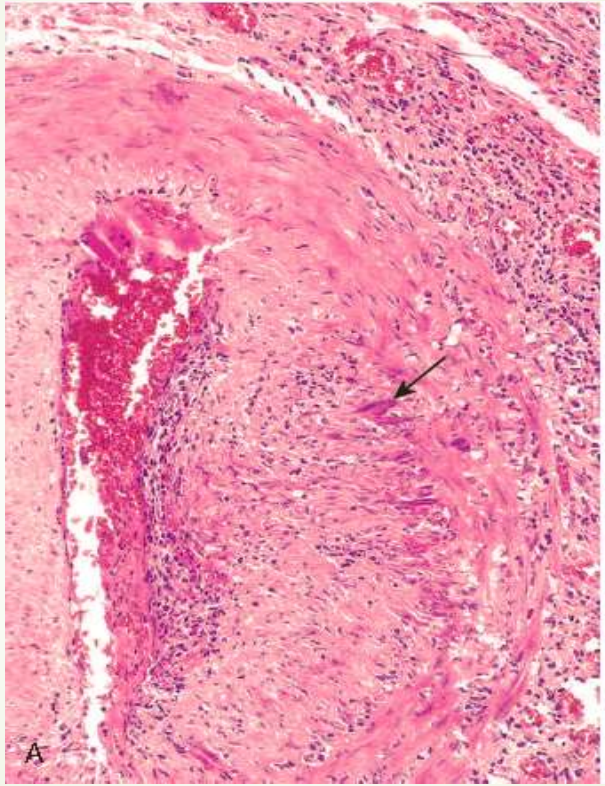
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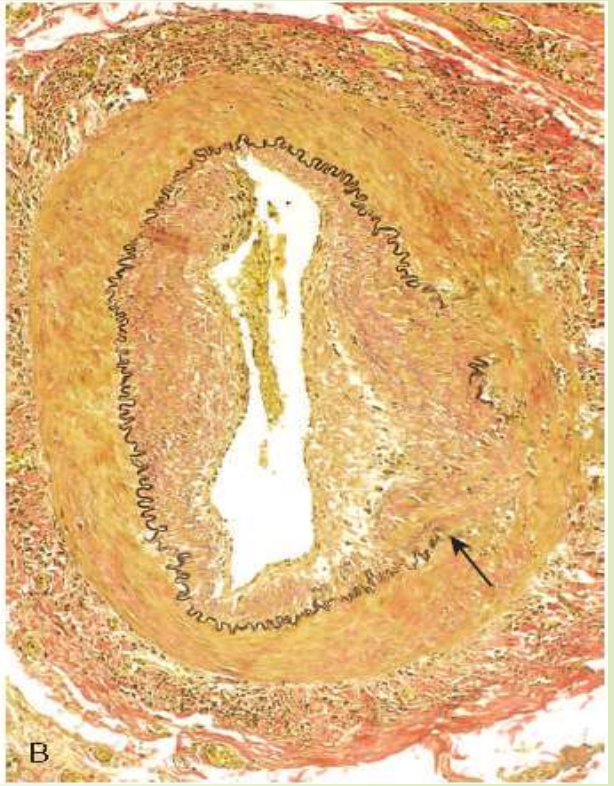
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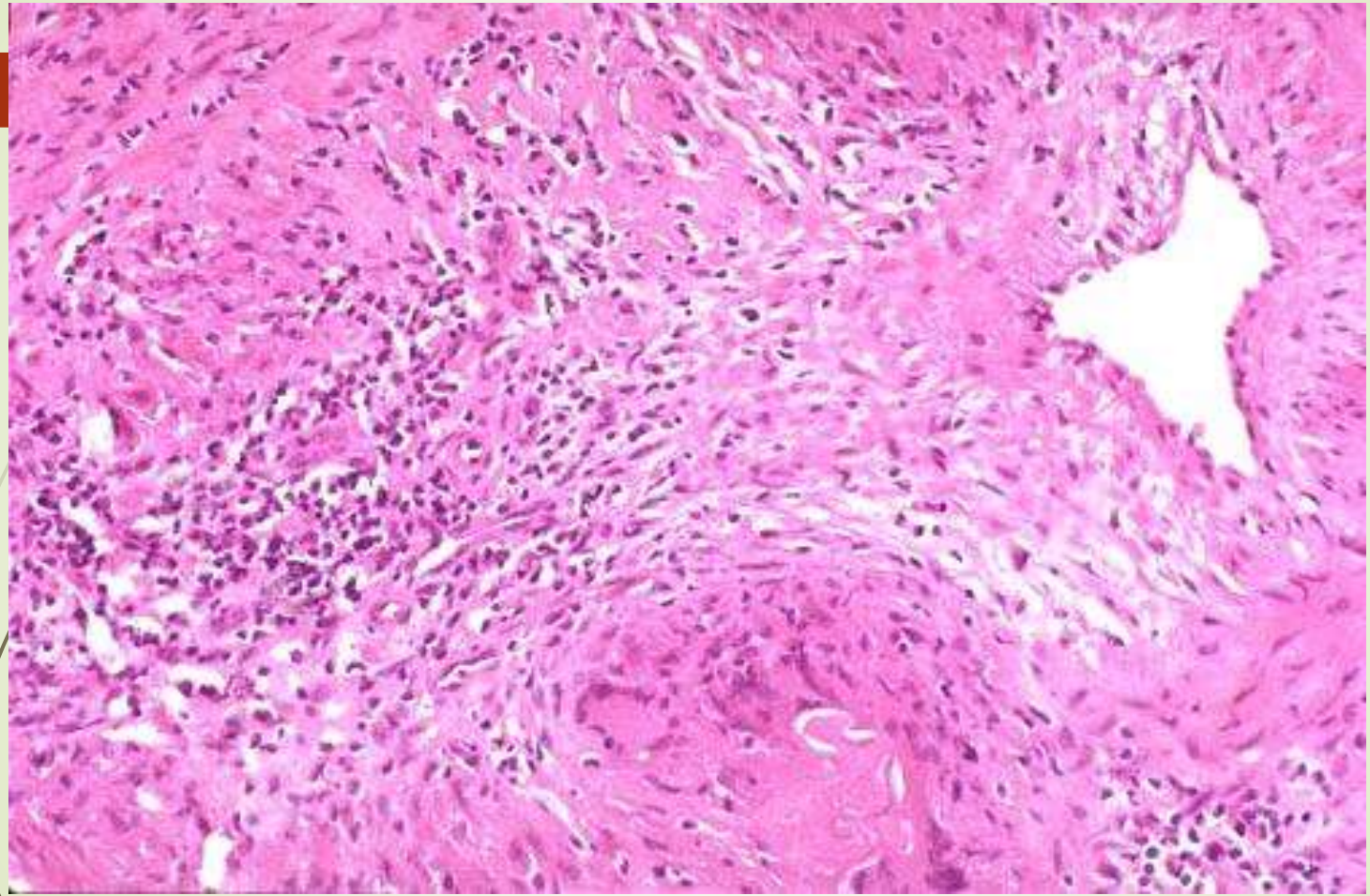
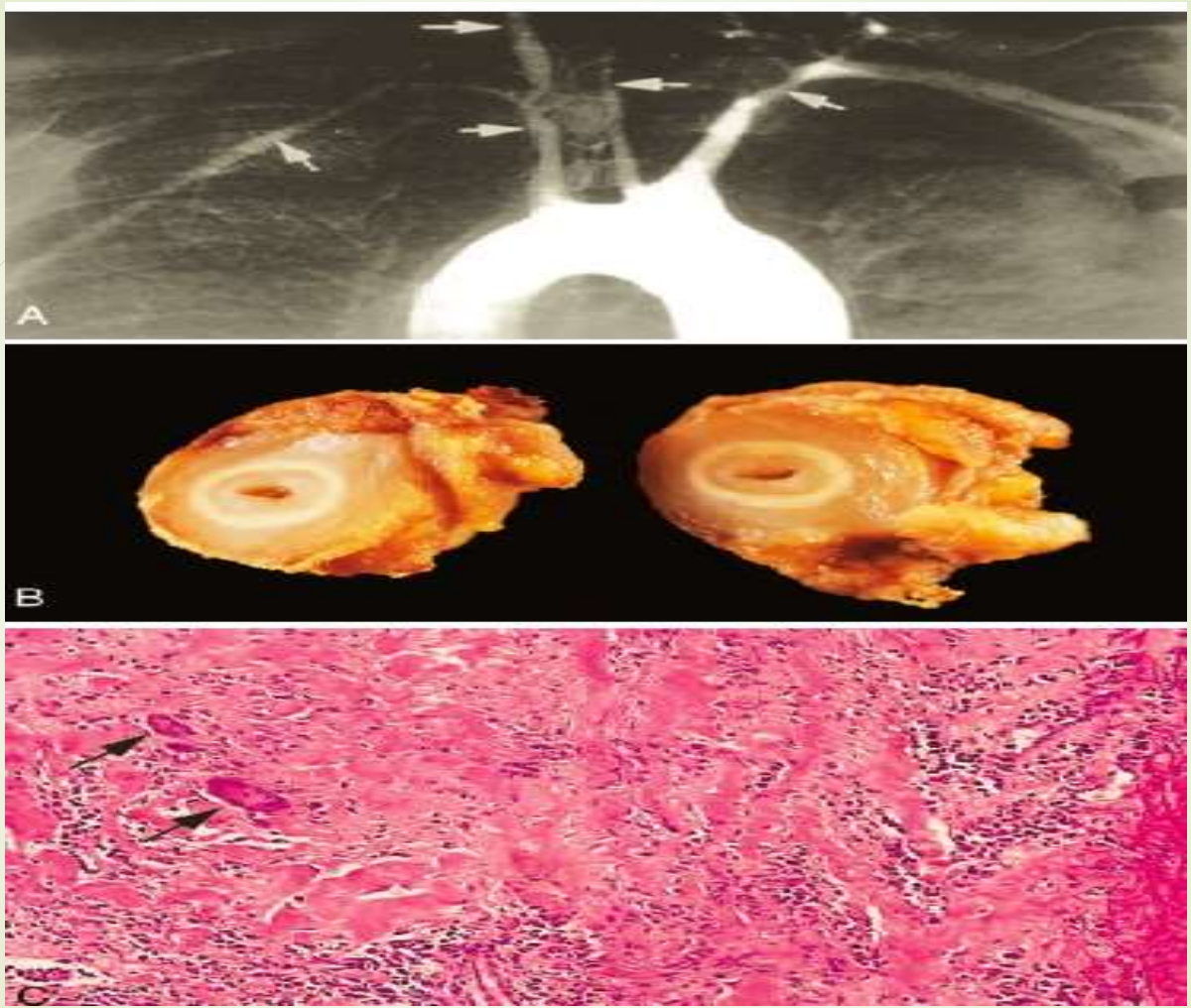


Takayasu Arteritis (pulseless disease)

- A granulomatous vasculitis of **medium & larger** arteries affects mainly arch of aorta.
- Characterized by transmural fibrous thickening & obliteration of aortic arch & great vessels → luminal narrowing of the major branch vessels → the origin of great vessels.
- Symptoms are secondary to luminal narrowing:

ocular disturbances & marked weakening of the pulses in the upper extremities →(pulseless disease).

- More common in females younger than 40 years and more frequently in Asian countries.
- Course of disease is variable ,may enter in quiescent stage.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Polyarteritis nodosa (PAN)

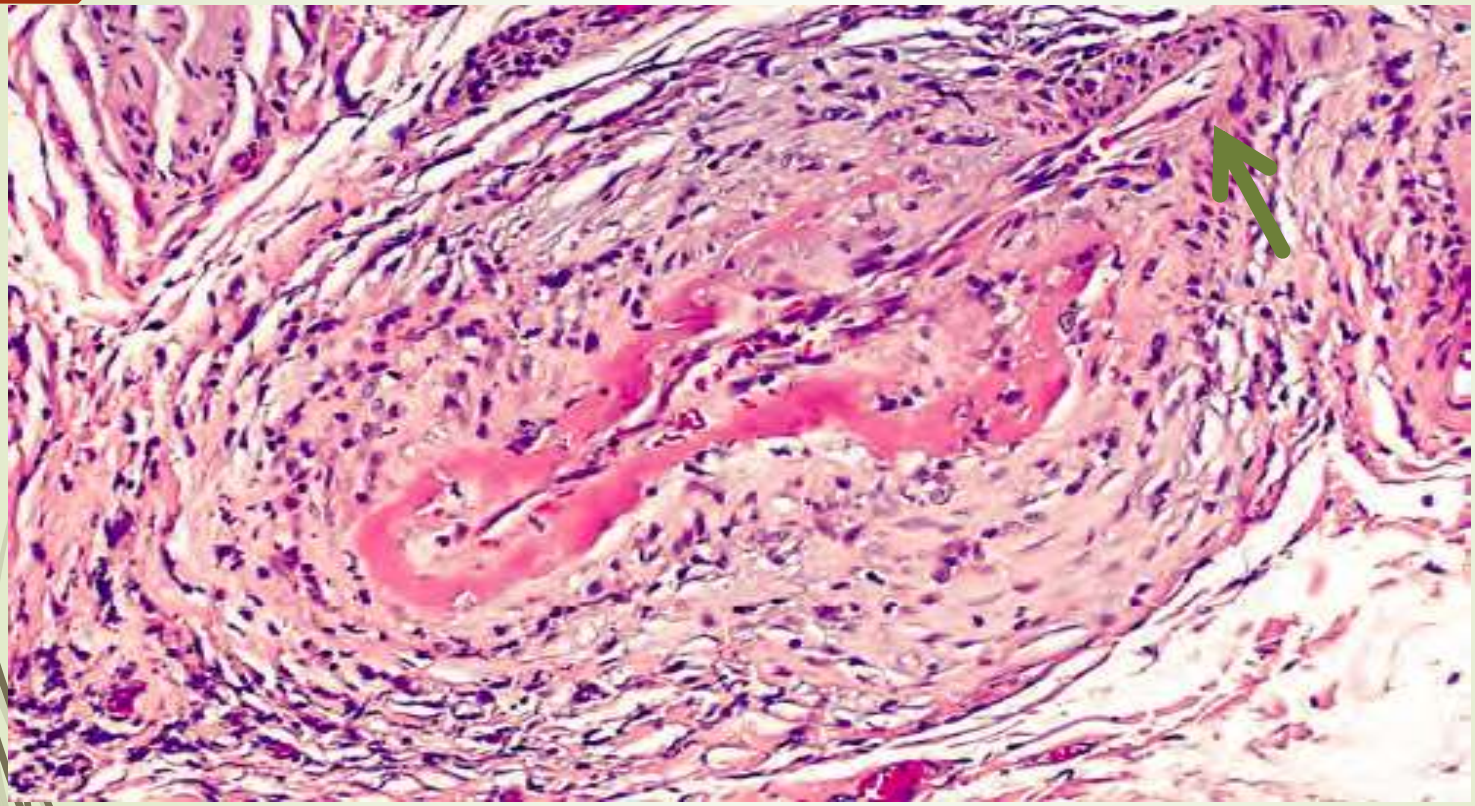
- A systemic disease characterized by necrotizing inflammation of **small - to medium - sized arteries** throughout the body (renal and visceral), sparing the pulmonary circulation.
- The involvement of the vessels is **focal, random & episodic.**
- It often produces irregular aneurysmal **dilatation (coz it weakens the arterial wall)** nodularity, and vascular **obstruction leading to infarctions.**
- Acute lesions show **segmental transmural necrotizing inflammation** extending around the vessel.

Polyarteritis nodosa (PAN)

- Healed lesions show marked fibrotic thickening of the arterial wall ,with associated elastic lamina fragmentation.
- All stages of activity may coexist in different vessels or even in the same vessel and this is so characteristic of PAN → ongoing and recurrent insults.
- Clinical features

young adults, males > females , vascular involvement is widely scattered → clinical picture can be varied & puzzling.

- Diagnosis .... Biopsy
- Rx … immunosuppression



Kawasaki disease (mucocutaneous lymph node syndrome)

- Acute febrile illness, **large- to medium-sized vasculitis** of infants & children (< 4 years) characterized by

  - Fever,
  - Lymphadenopathy,
  - Skin rash
  - Oral / conjunctival erythema.

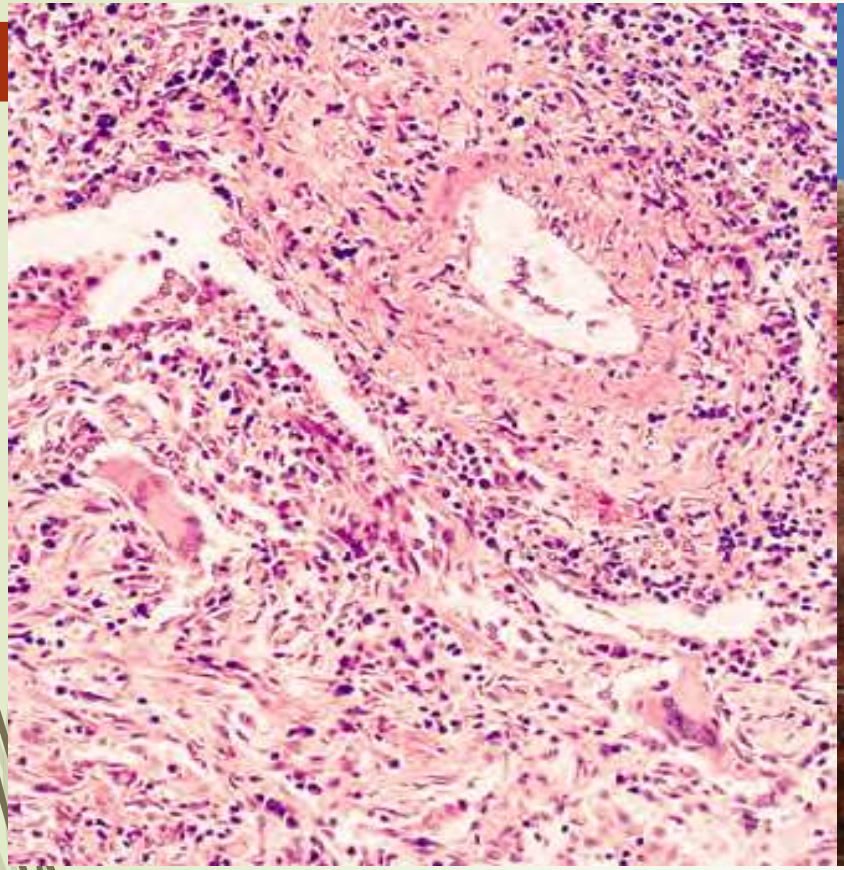
- 20% have coronary vasculitis ,often with aneurysm.
- Histology like PAN
- Etiology ; unknown (auto-antibodies to ECs)
- Self-limited disease, rarely fatal(1%) → complications of coronary involvement.

Granulomatosis With Polyangiitis

- Previously called *Wegener granulomatosis*.
- Classical cases consist of a triad:
  1. Granulomatous or necrotizing vasculitis mainly in the **lung** & upper respiratory tract.
  2. Necrotizing granulomas of upper &/or lower respiratory tract
  3. Renal involvement
     - focal necrotizing glomerulonephritis
     - rapidlly progressive glomerulonephritis
- Clinical picture
  - overlaps with PAN
  - Males > females, peak at 5th decade

Granulomatosis With Polyangiitis

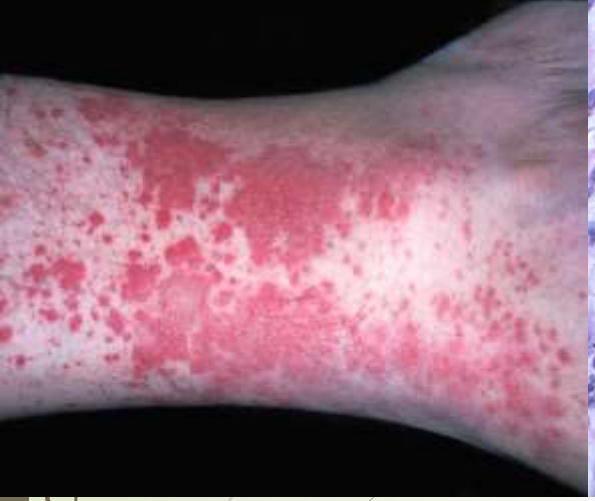
- Diagnosis
  - Lung biopsy, kidney biopsy, or nasal biopsy,
- Prognosis
  - 80% die within a year (if not treated)
  - 90% respond to treatment
- Pathogenesis
  - T Cell Mediated.
  - > 95% PR3-ANC positive (Mirrors the clinical coarse)

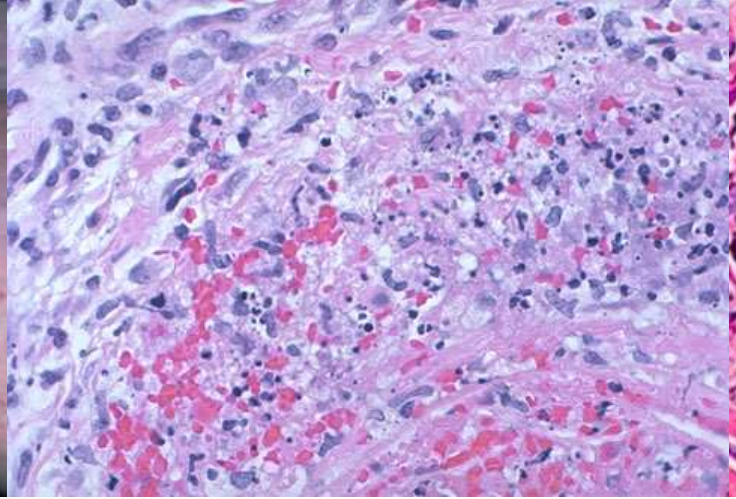


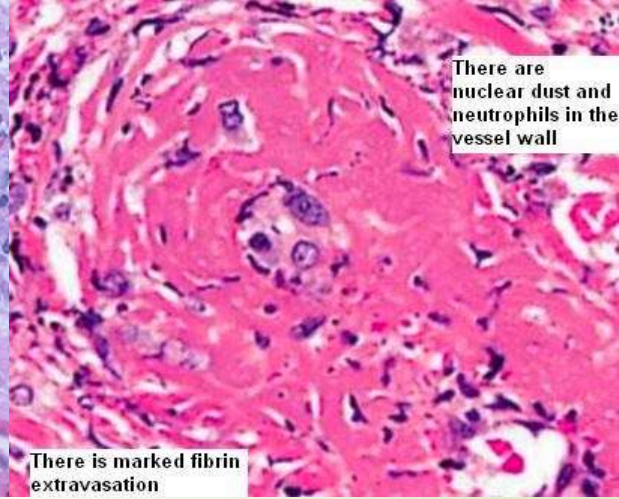

cm
1
2
3
HÔPITAL
SAINTE-JUSTINE

Microscopic polyangiitis (microscopic polyarteritis)
or
HYPERSENSITIVITY vasculitis
OR
LEUKOCYTOCLASTIC vasculitis

Most cases of microscopic polyangiitis are associated with MPO-ANCA , Although immunoglobulins and complement components can be demonstrated in early skin lesions, most lesions are “pauci-immune”



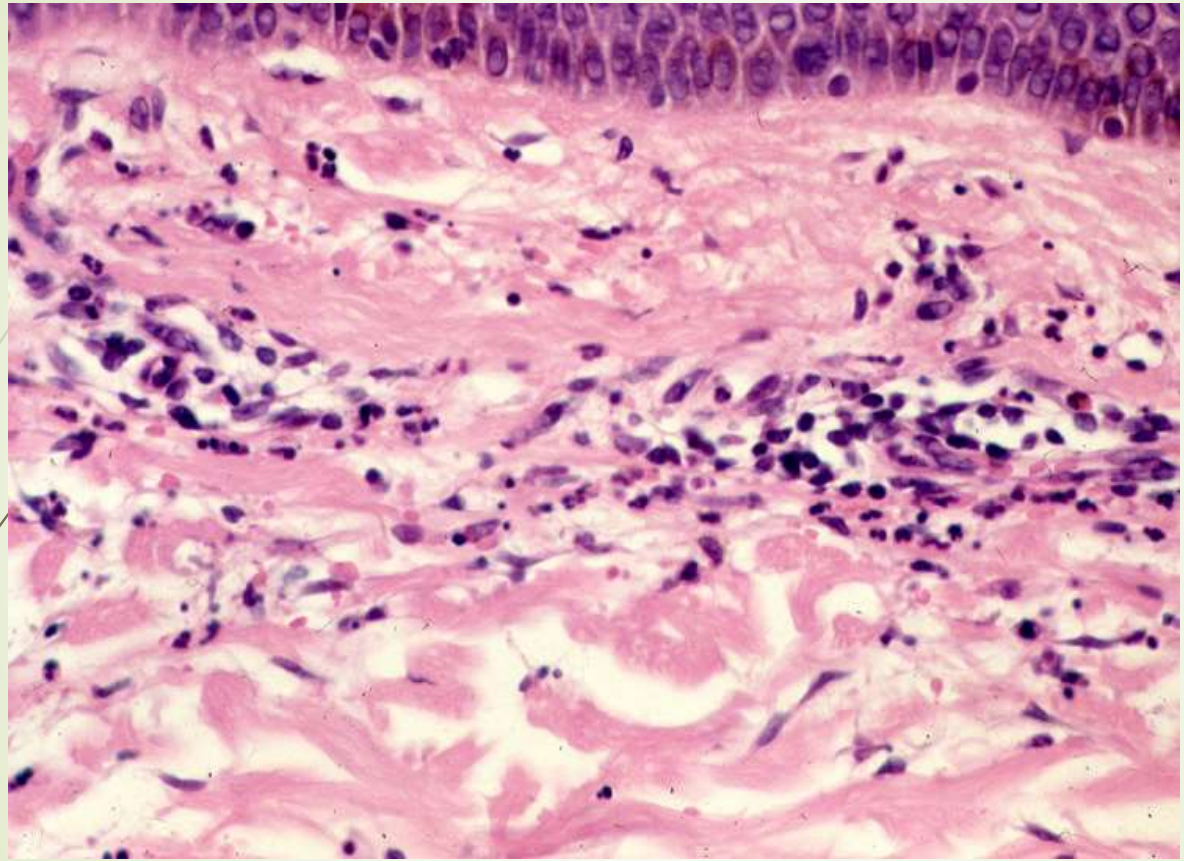




- Involvement of small vessels (arterioles, capillaries, & venules).
- all lesions of the same stage/age.
- Skin, mucous membranes, lungs, brain, heart, GIT, kidney & muscles.
- In contrast to PAN, necrotizing glomerulonephritis (90%) & pulmonary capillaritis are common

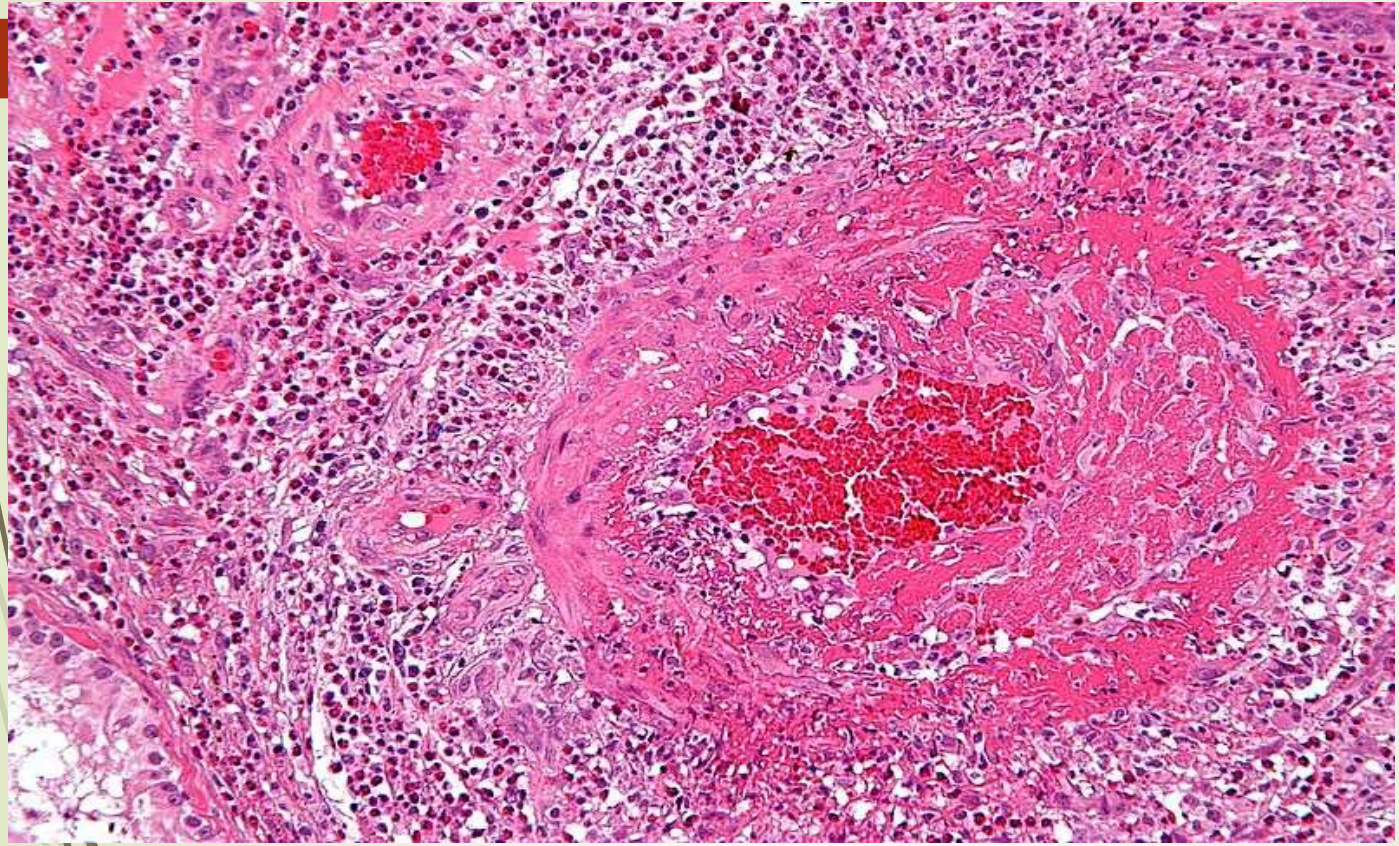
- Segmental fibrinoid necrosis of media.
- No granulomatous inflammation
- Sometimes limited to infiltration of vessel wall by neutrophils with **nuclear fragmentation (leucocytoclasia), leukocytoclasatic vasculitis.**
- A reaction to an Ag such as drugs, microorganisms or hetrologous protein in a previously sensitized patients.

- Clinical: hemoptysis, arthralgia, abdominal pain, hematuria, proteinuria, hemorrhage, & muscle pain or weakness.
- Except in brain or renal involvement most patients respond to removal of offending antigens & immunosuppression.
- Ass : Henoch -schonlein purpura, essential mixed cryoglobulinemia, vasculitis with malignancy



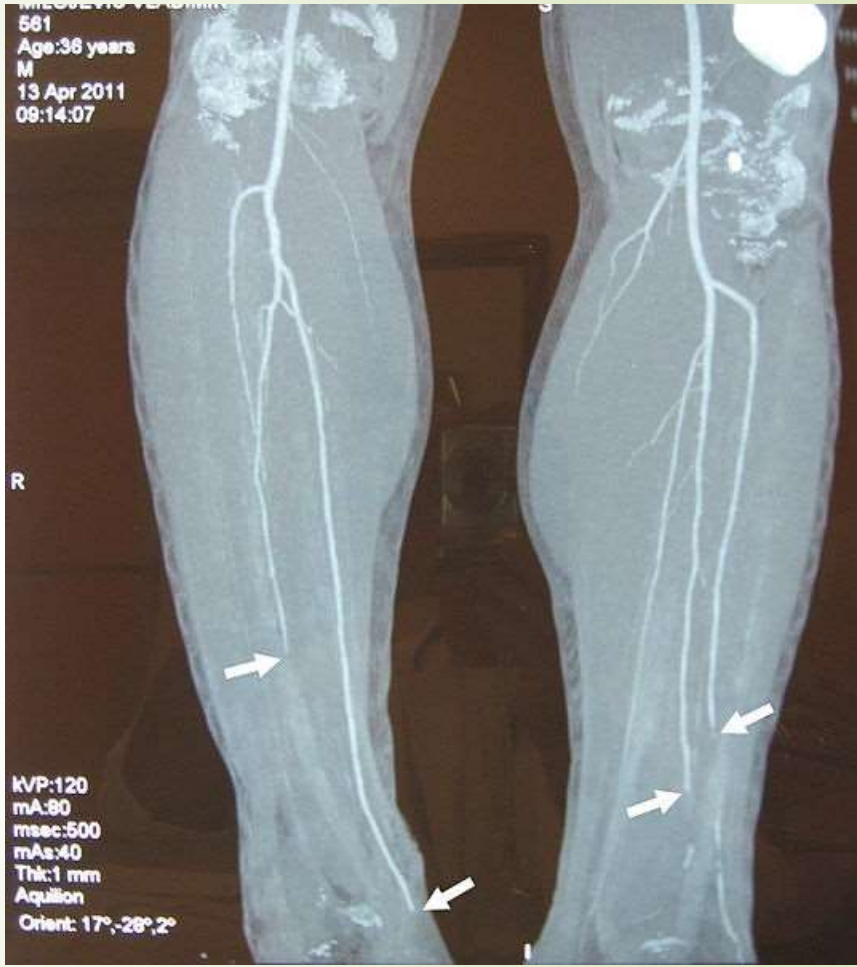
In allergic granulomatosis and angiitis (Churg-Strauss syndrome)

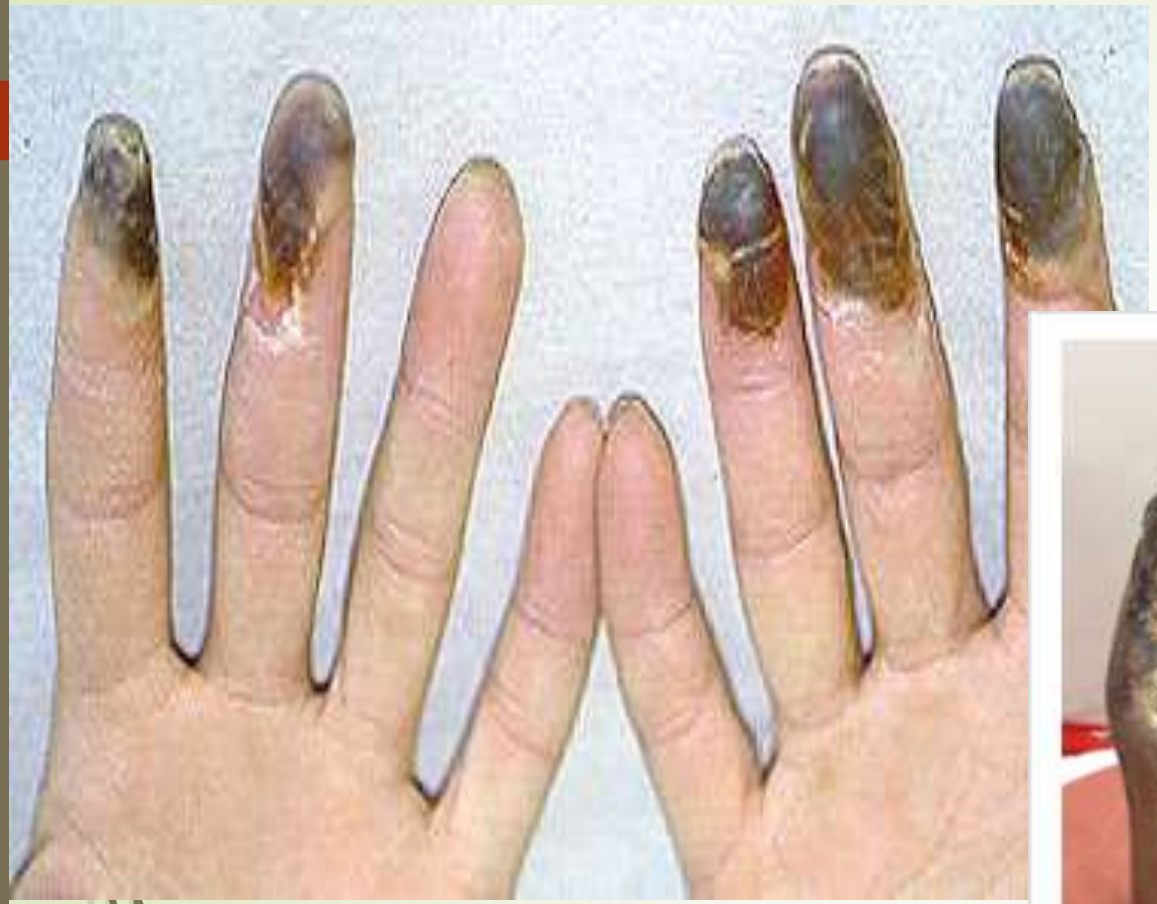
- Rare disease characterized by
  - necrotizing vasculitis accompanied by granulomas with **eosinophilic** necrosis.
  - p-ANCAs are present in A minority of patients.
  - There is a strong association with allergic rhinitis, bronchial asthma, and peripheral eosinophilia.
  - Coronary arteritis & myocarditis are the principal causes of morbidity and mortality.

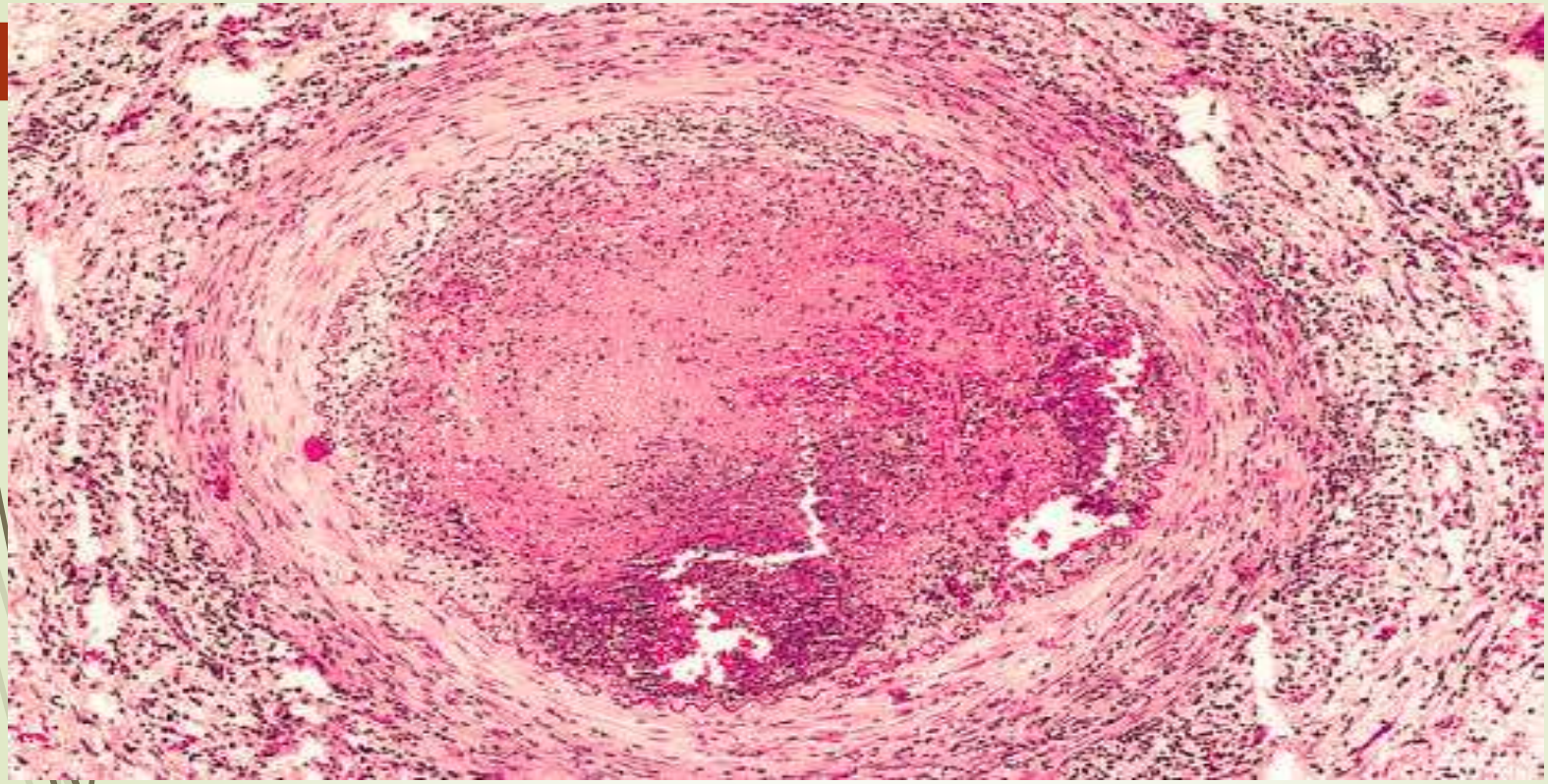


Thromboangiitis obliterans (Buerger's Disease)

- A condition marked by segmental, **thrombosing**, acute & chronic inflammation of intermediate & small arteries & veins in the limbs with extension to accompanying nerves.
- Exclusively seen in heavy smokers males before the age of 35.
- Instep claudication followed by pain at rest, might end in gangrene.
- Etiology ? Endothelial cell injury by toxins in tobacco.







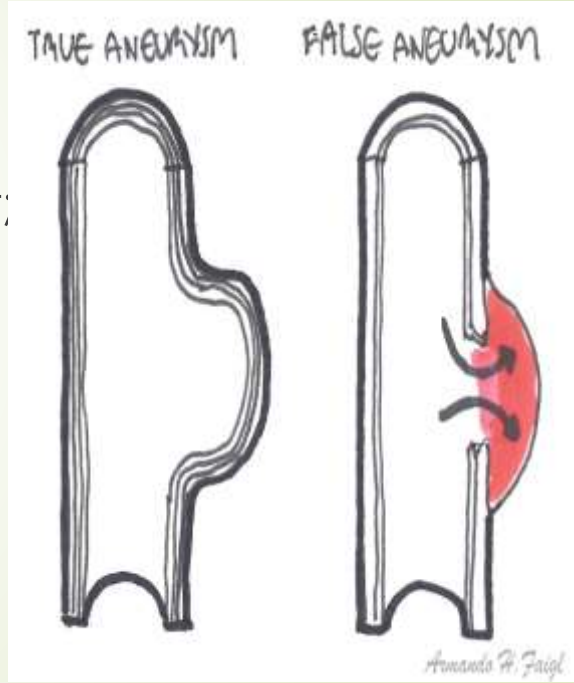
Aneurysms & Dissections

Aneurysms

- **A congenital or acquired dilations of blood vessels or the heart,** could be:

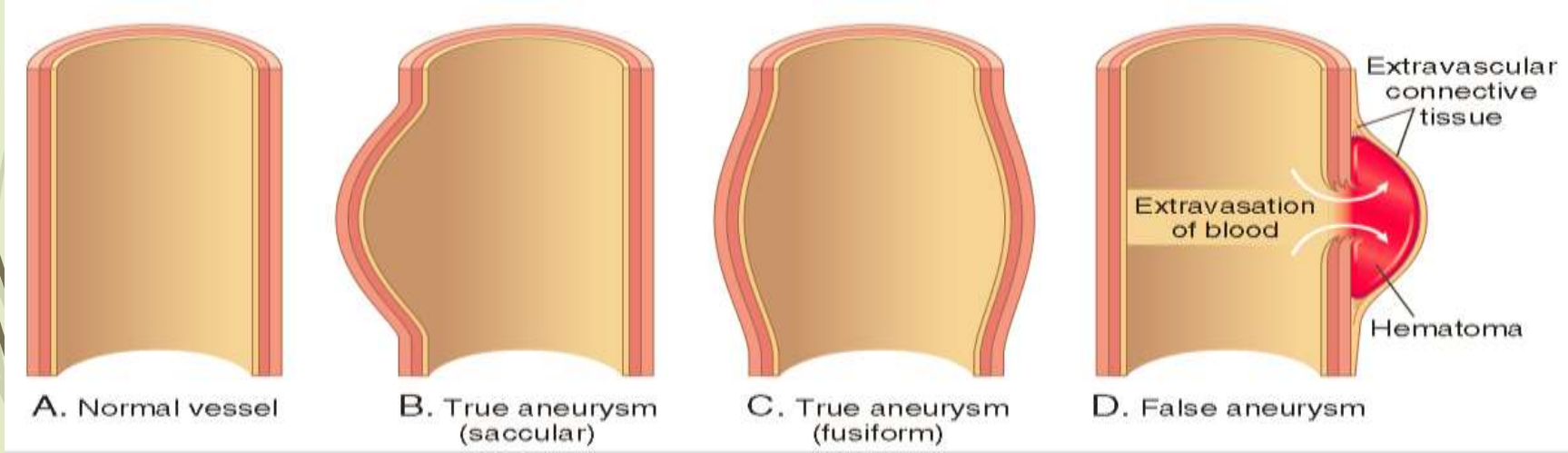
1. "**True**" : all three layers of the artery (intima, media, & adventitia) or the wall of the heart; e.g. atherosclerotic, congenital vascular aneurysms, ventricular aneurysms after MI

1. "**false**" : a wall defect leads to the formation of an extravascular hematoma that communicates with the intravascular space ("pulsating hematoma")



Aneurysms – Types by shape

- **Saccular aneurysms**: discrete outpouchings ranging (5-20 cm) in diameter, often with a contained thrombus.
- **Fusiform aneurysms:** circumferential dilations up to 20 cm in diameter, most commonly involve aortic arch, abdominal aorta, or iliac arteries.

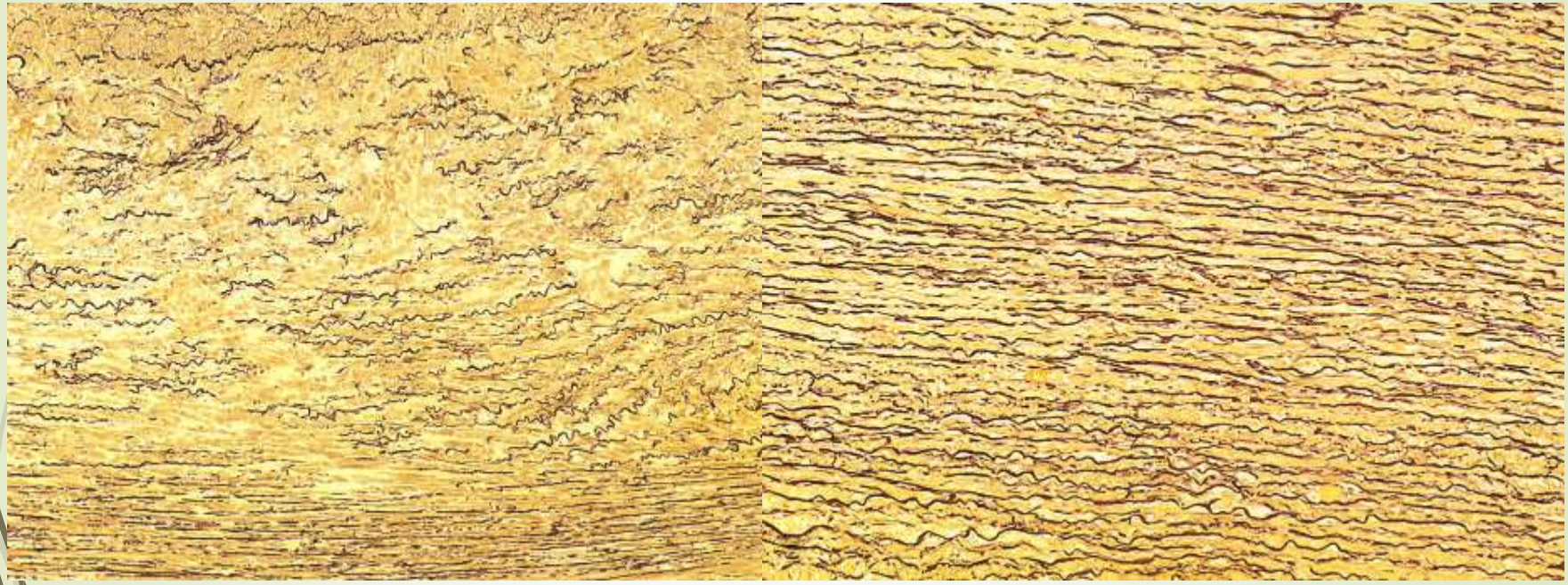


Aneurysms – Pathogenesis

- **Medial ischemia may lead to "degenerative changes" of the aorta**; Ischemia → smooth muscle cell loss → scarring and loss of elastic fibers → inadequate extracellular matrix synthesis → production of increasing amounts of amorphous ground substance (glycosaminoglycan).

- Histologically, these changes recognized as ***cystic medial degeneration***

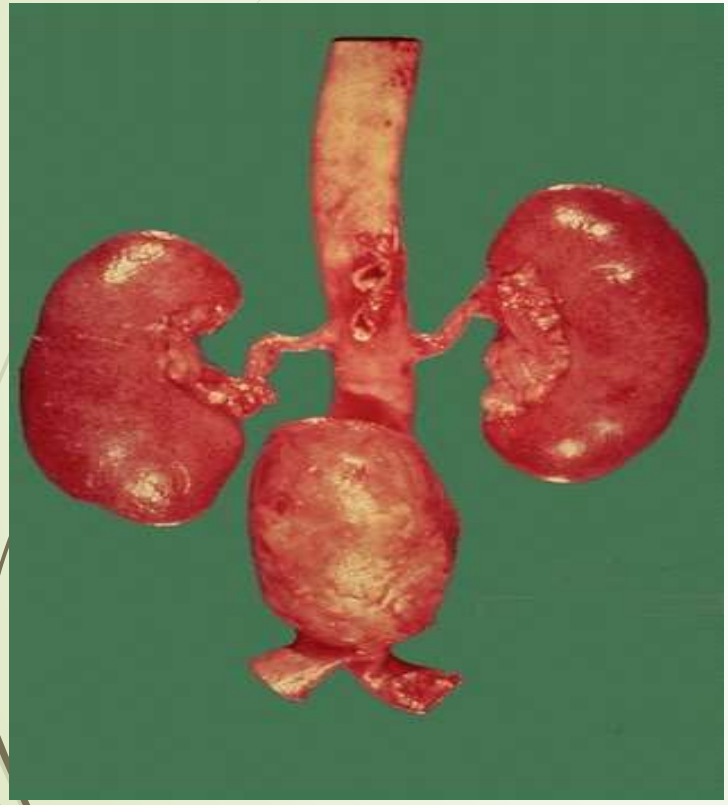
Aneurysms – *cystic medial degeneration*

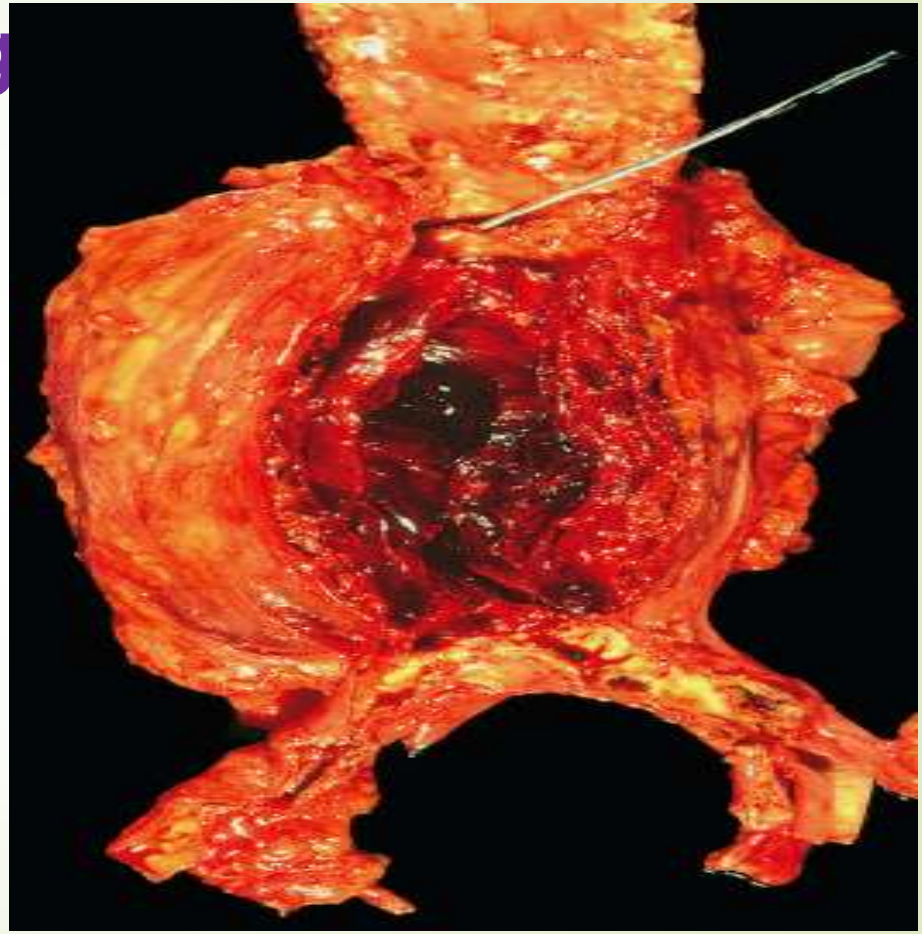


AAA- Morphology

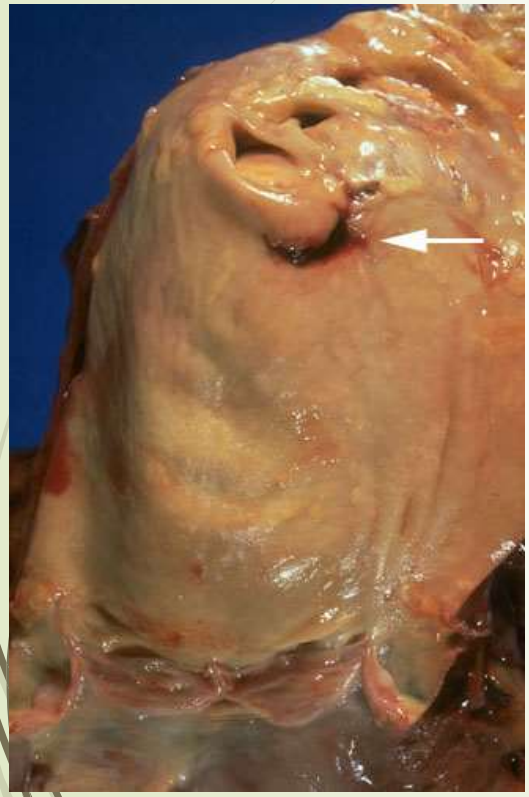
- AAAs typically occur between the renal arteries & the aortic bifurcation; can be saccular or fusiform & up to 15 cm in diameter and 25 cm in length.
- In the vast majority extensive atherosclerosis is present, with thinning & focal destruction of the underlying media.
- The aneurysm sac usually contains bland, **laminated**, poorly organized mural thrombus …can fill much of the dilated segment.
- Not infrequently, AAAs are accompanied by smaller iliac artery aneurysms.

AAA- Morpholog

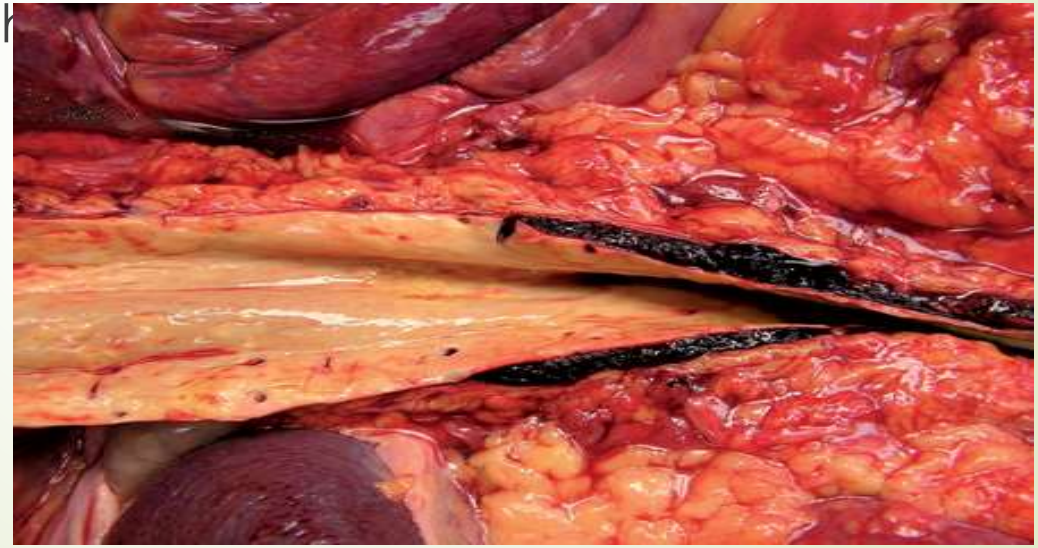




Aortic Dissection - Morphology

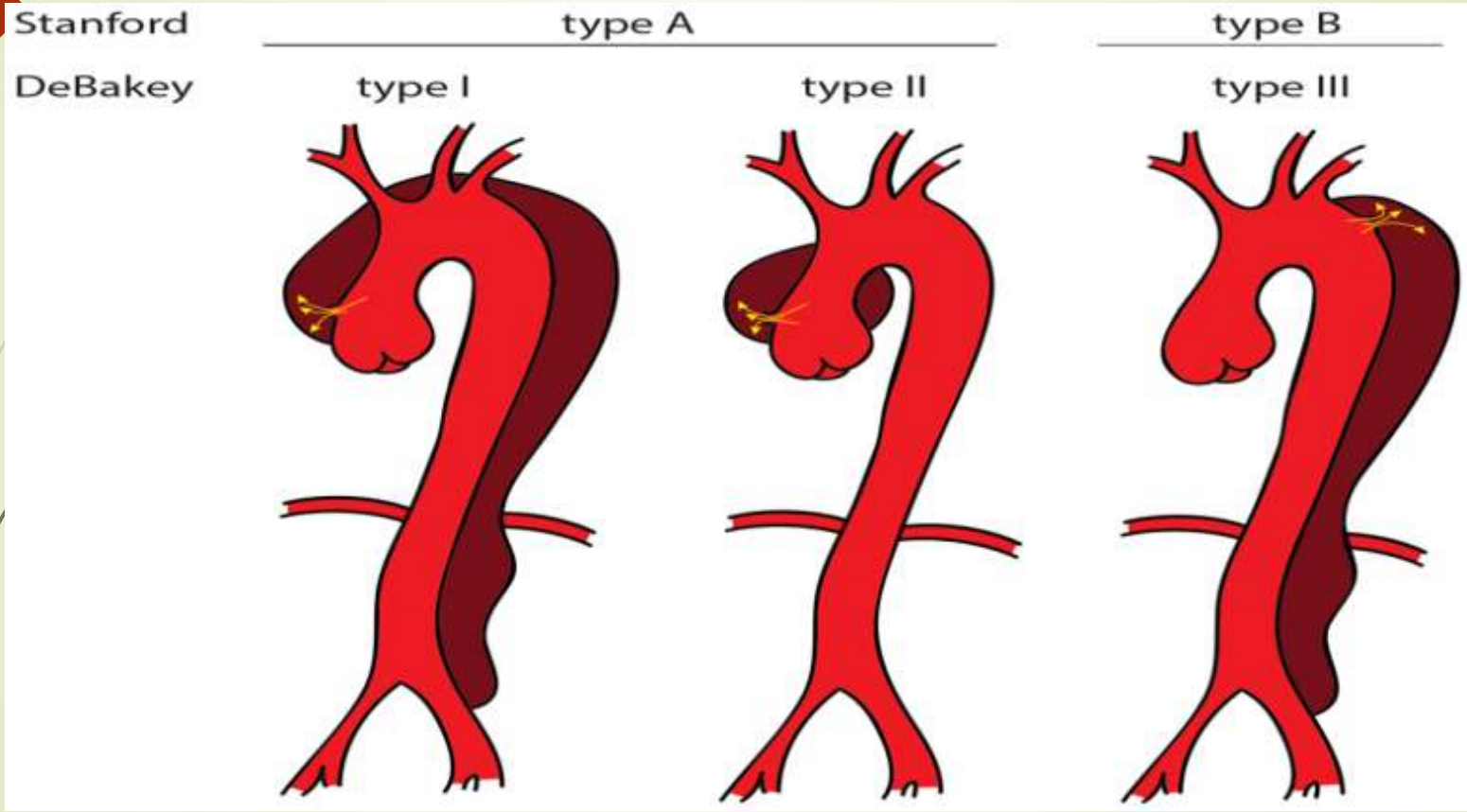


mostly the intimal tear marking origin point is found in the ascending aorta within 10 cm of the valve. Dissection plane can extend retrograde toward the h



Aortic Dissection - Morphology

Dissection plan usually lies between the middle and outer thirds of the media.


Stanford
type A
type B
DeBakey
type I
type II
type III

Veins and Lymphatics

90% of clinical venous disease caused by Varicose veins and phlebothrombosis/thrombophlebitis

Varicose veins

- **Abnormally dilated tortuous veins produced by chronically (1)increased intraluminal pressures & (2)weakened vessel wall support.**
- Venous valves incompetent → lower-extremity stasis, congestion, edema, pain, & thrombosis.
- **Locations:** typically, superficial veins of the upper & lower leg.
- **Risk factors:** Obesity, female sex, pregnancy, & familial tendency.
- **Clinical features:** persistent edema & secondary ischemic, skin changes, including stasis dermatitis and ulcerations.

Varicose veins– Clinical presentation