Charting New Horizons in Education

Hemodynamics Lab

00

Pathology

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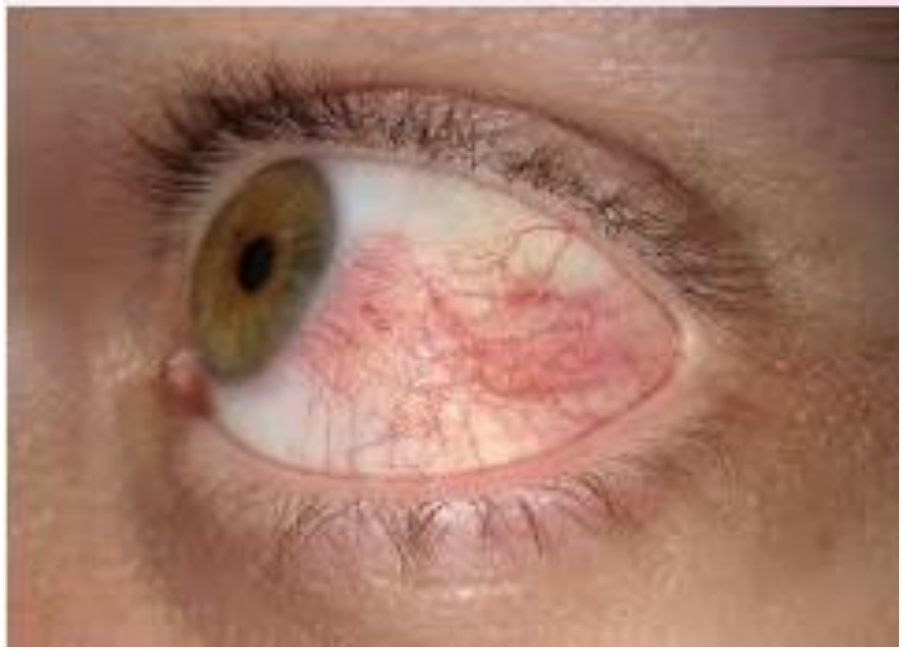
Hyperemia

* ↑ Inflow of Blood (↑ volume)
* Oxegynated (Red)
* Active Process
* Inflammation or Excercising skeletal muscle



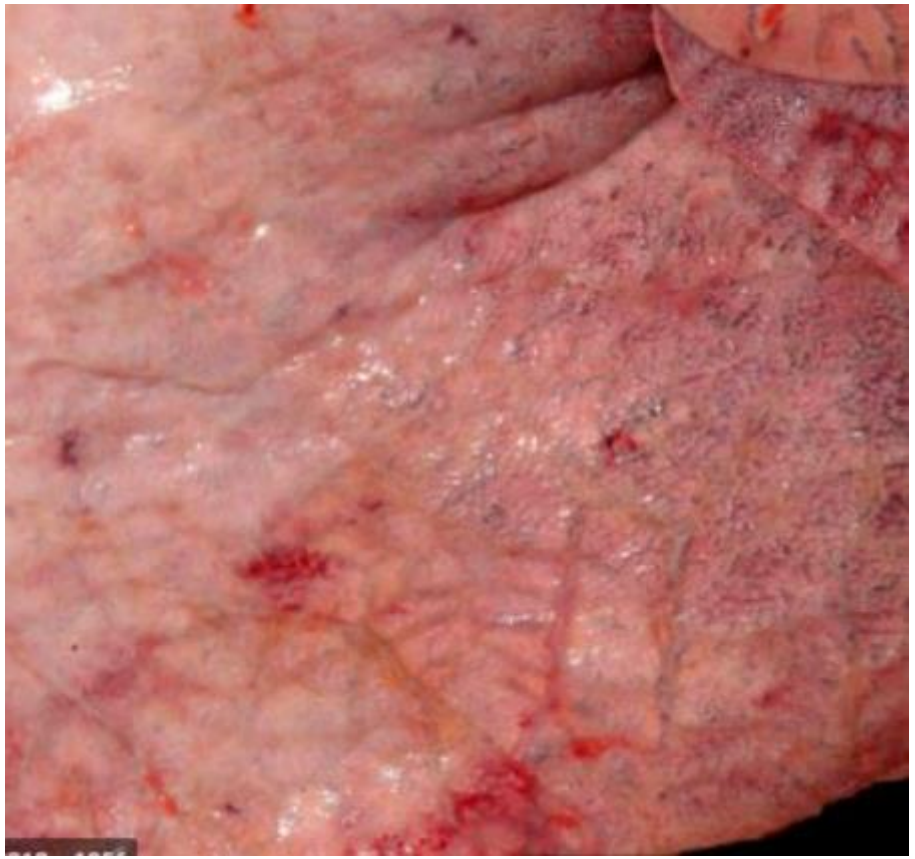
Congestion

* ↓ outflow of Blood (↑ volume)
* Deoxygenated (Cyanosis)
* Passive Process
* Locally → Isolated Venous Obstruction
* Systemic → Congestive heart failure

Lung congestion – Cut surface

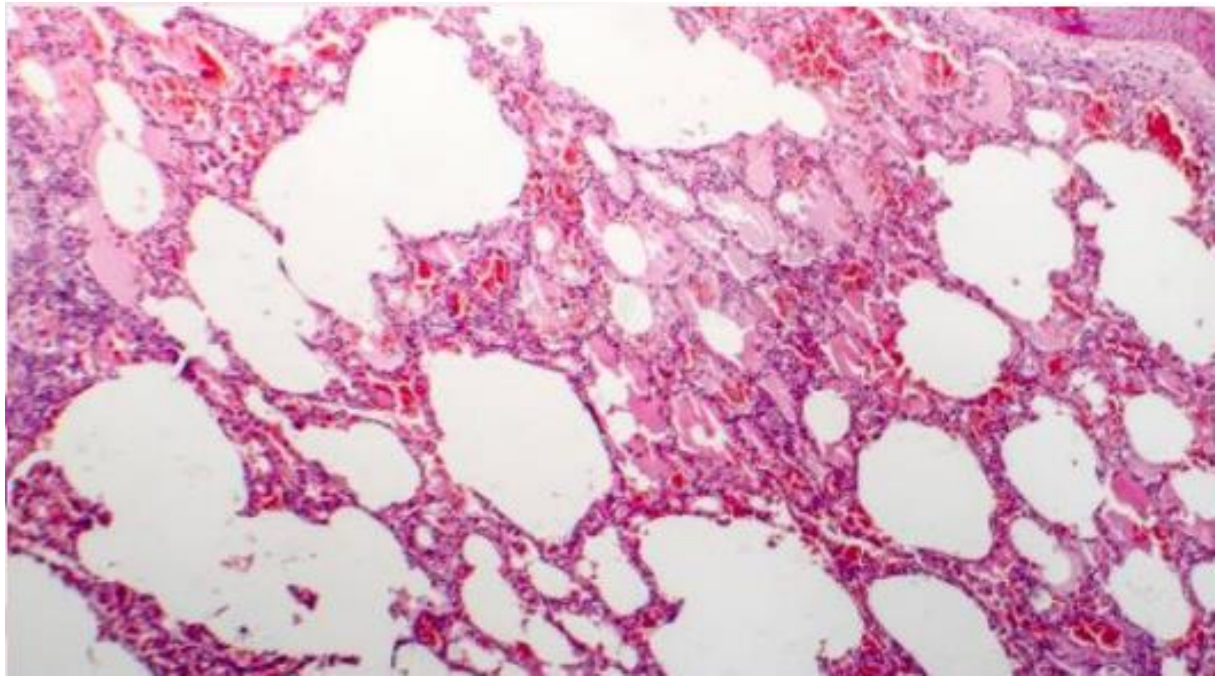
Hyperemic بالحادة او
& congested
tissues

wet يكونوا
& they ooze
Blood



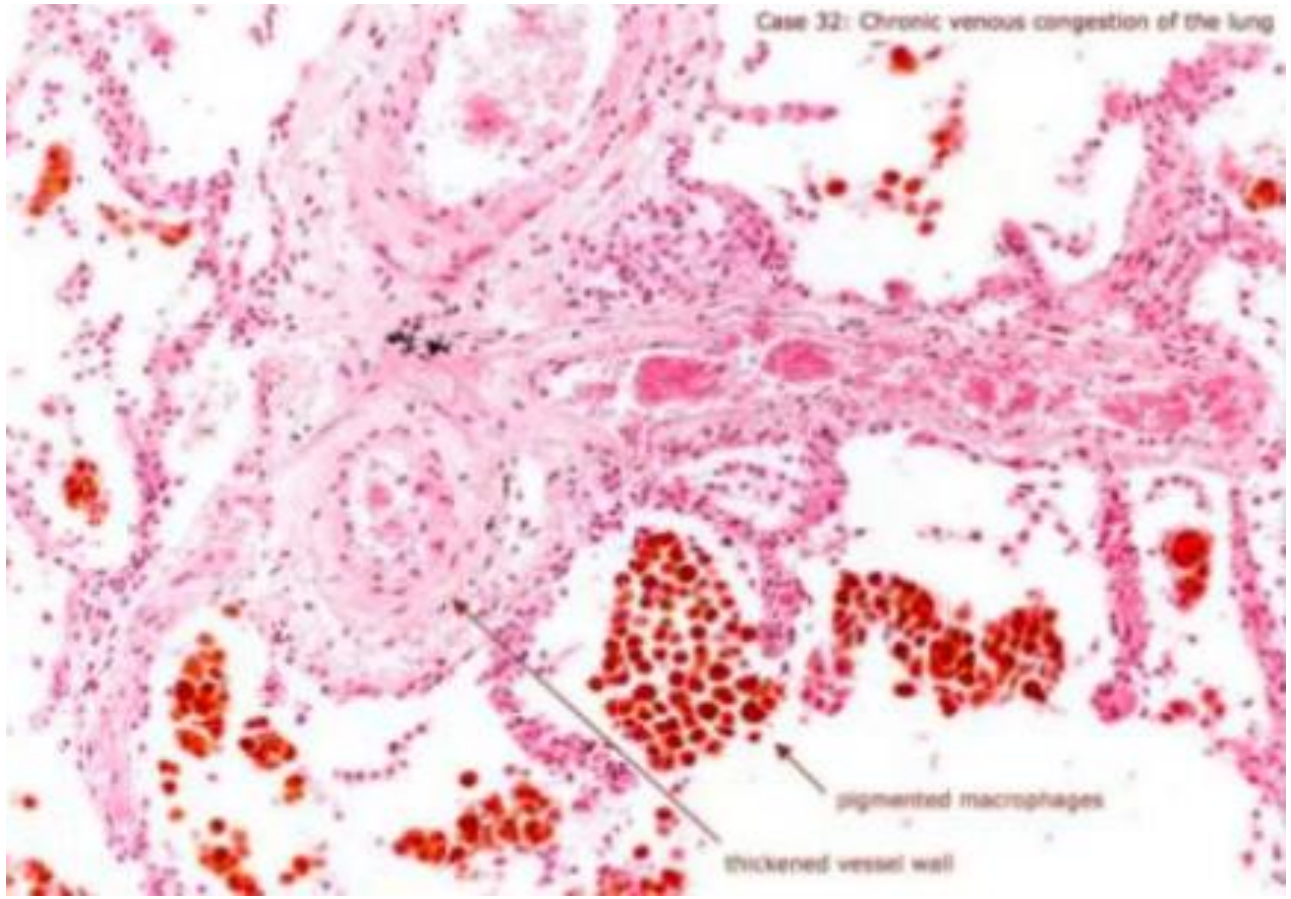
Acute lung congestion - Microscopic

* Blood engorged capillaries
* Alveolar septal edema
* intra-alveolar hemorrhage

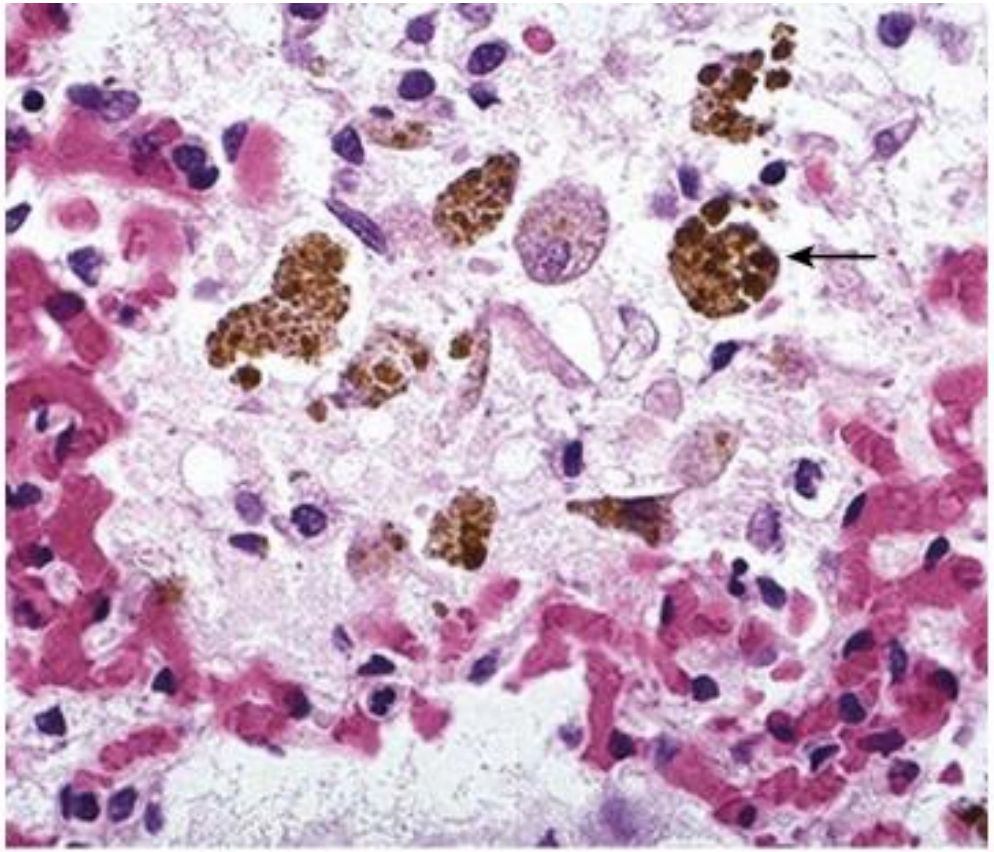


Chronic lung congestion - Microscopic

* Thickened & fibrotic septa
* Heart failure cells



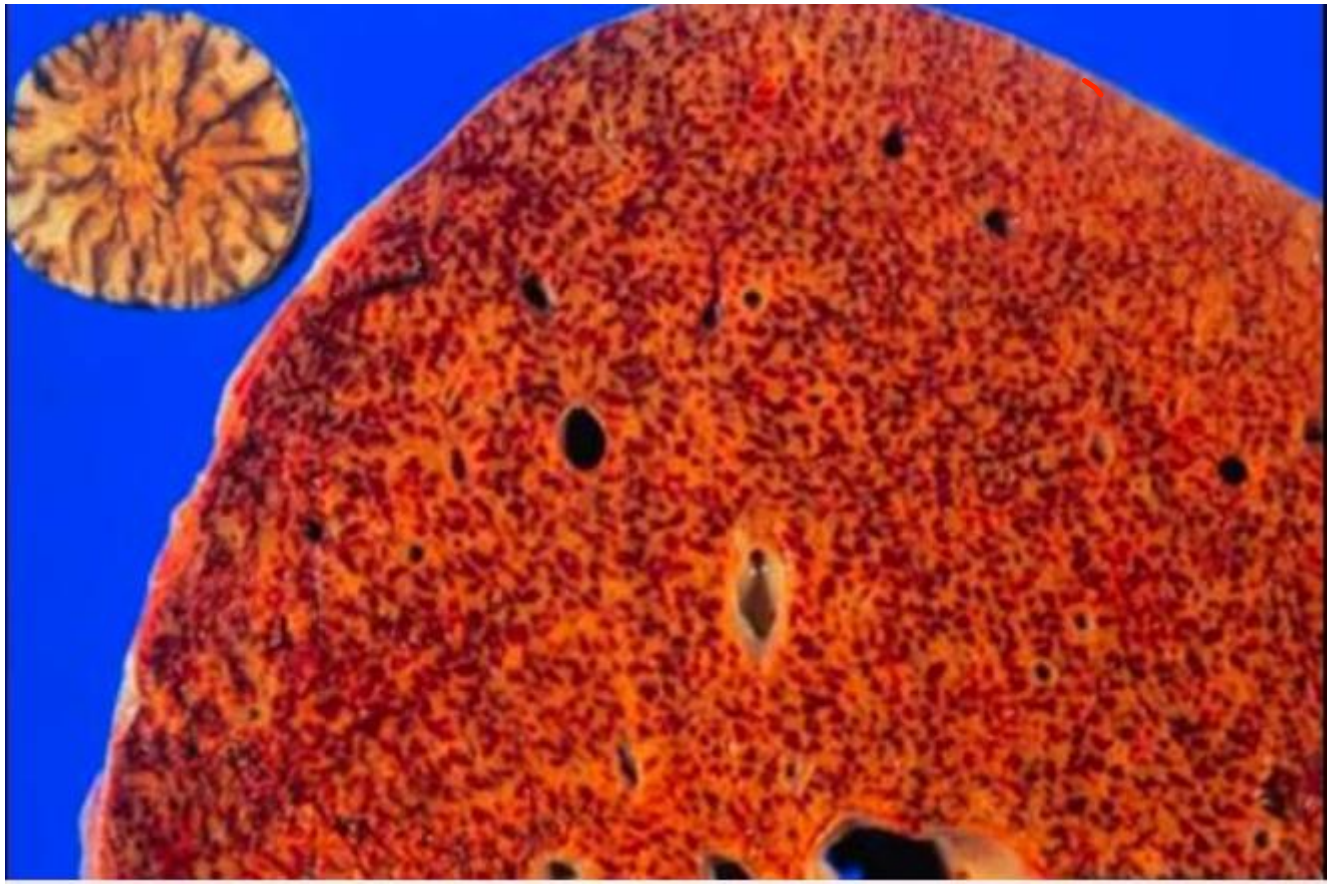
Chronic lung congestion – Microscopic - Robbins



eFIG. 3.1 "Heart failure" cells. The alveolar space contains pinkish edema fluid and "heart failure cells," macrophages with brown hemosiderin pigment *(arrow)* derived from phagocytosed red cells that leaked from congested capillaries. (From Klatt EC: *Robbins and Cotran Atlas of Pathology*, ed 4, Fig. 5.12, Philadelphia, 2021, Elsevier.)

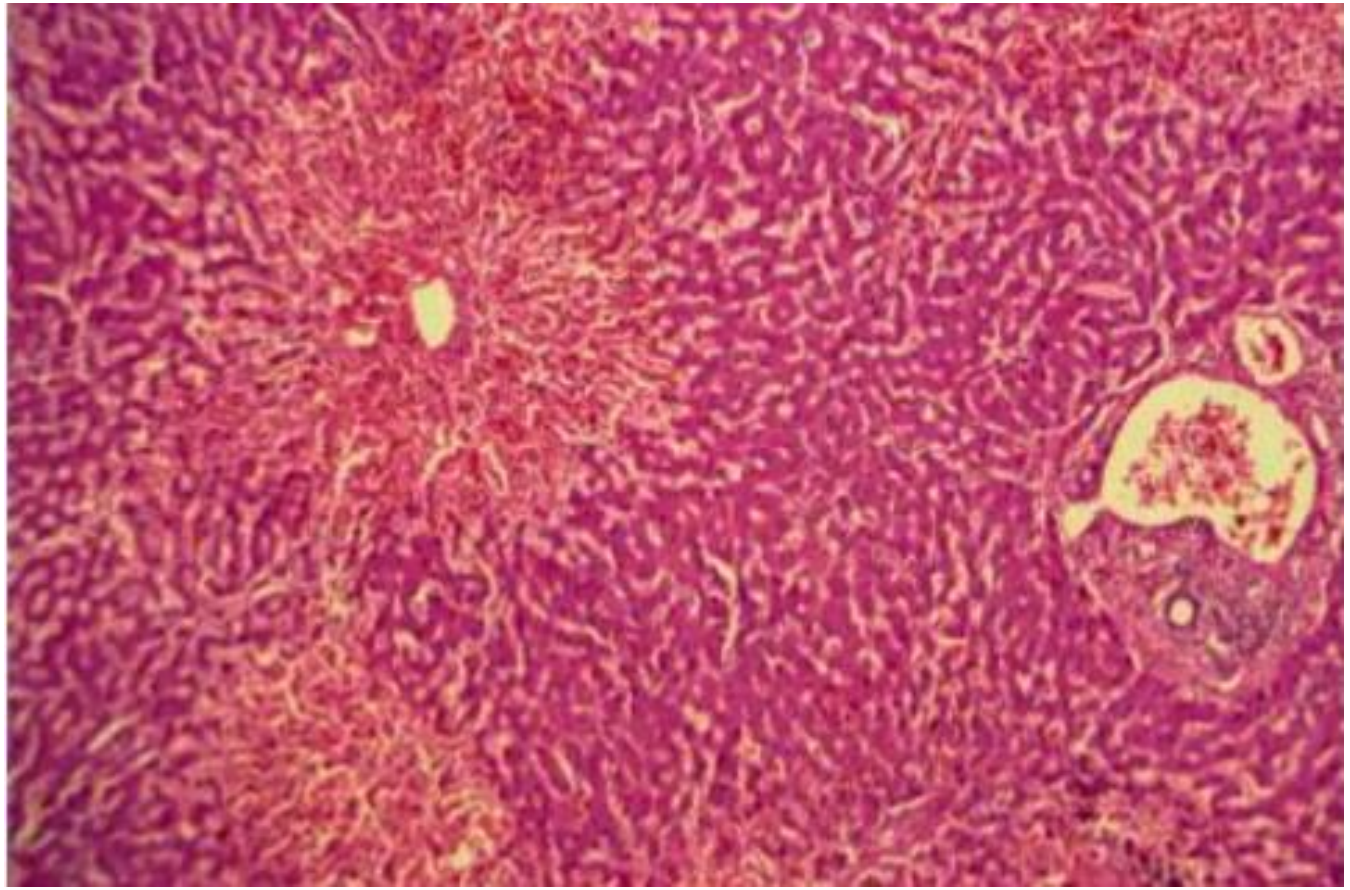
Hepatic congestion – Nutmeg liver

* Necrotic - Red & depressed
* Viable - tan



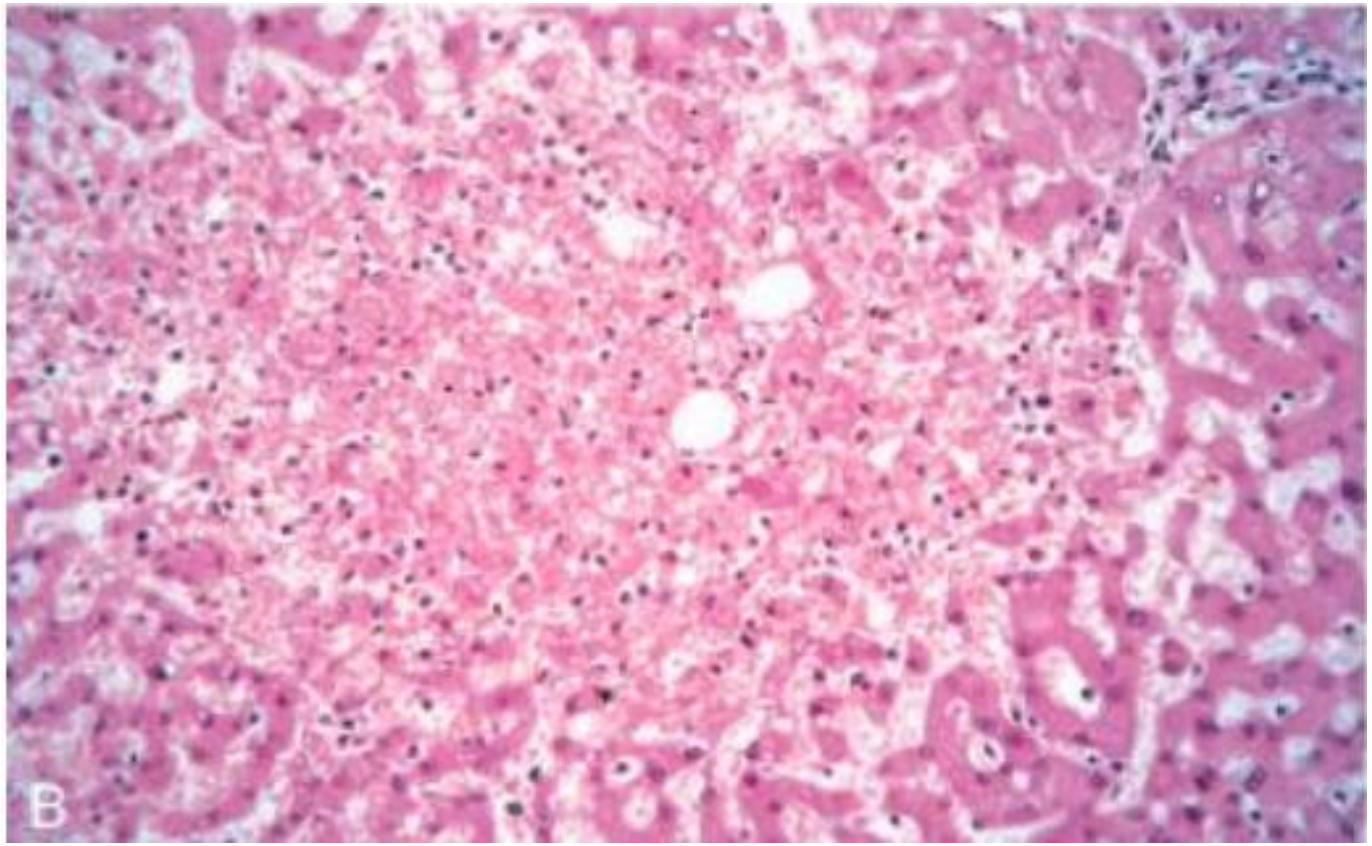
Hepatic congestion – Nutmeg liver

* Centrally located and more prone to necrosis



Hepatic congestion – Nutmeg liver

* Centrilobular hepatocyte necrosis
* Hemorrhage
* Hemosiderin-laden macrophage



Hepatic congestion – Nutmeg liver - Robbins

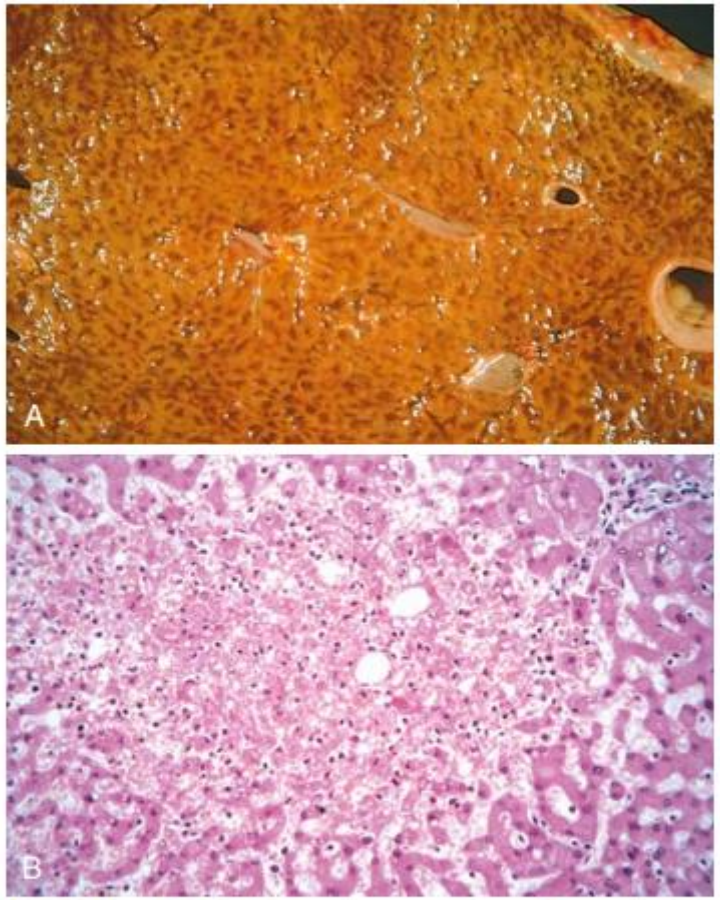
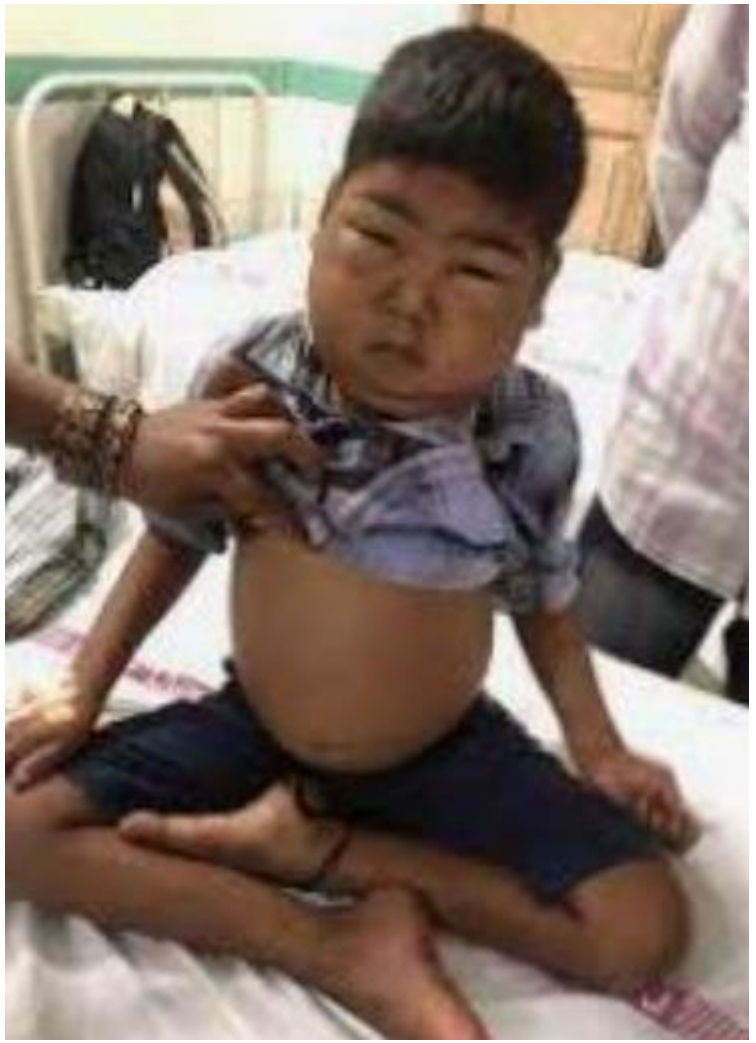


FIG. 3.1 Liver with chronic passive congestion and hemorrhagic necrosis. (A) In this autopsy specimen, centrilobular areas are red and slightly depressed compared with the surrounding tan viable parenchyma, creating "nutmeg liver" (so called because it resembles the cut surface of a nutmeg). (B) Microscopic preparation shows centrilobular hepatic necrosis with hemorrhage and scattered inflammatory cells. (Courtesy of Dr. James Crawford.)

Anasarca

* General swelling of the whole body
* Severe
* Effusion

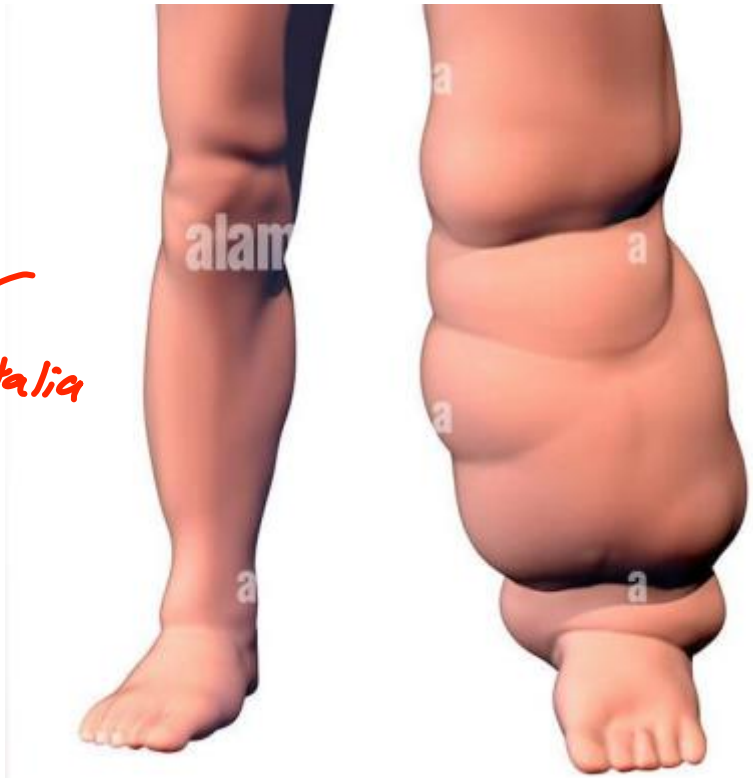


Breast cancer – Peau d' orange

* Infiltration & obstruction of SUPERFICIAL lymphatics by Breast Cancer
* finely pitted Apperance
* Orange Peel

Filiarisis

* Parasitic Infection
filariasis
* Massive edema of lower extremity & External genitalia
و بسمّوها
Elephantiasis

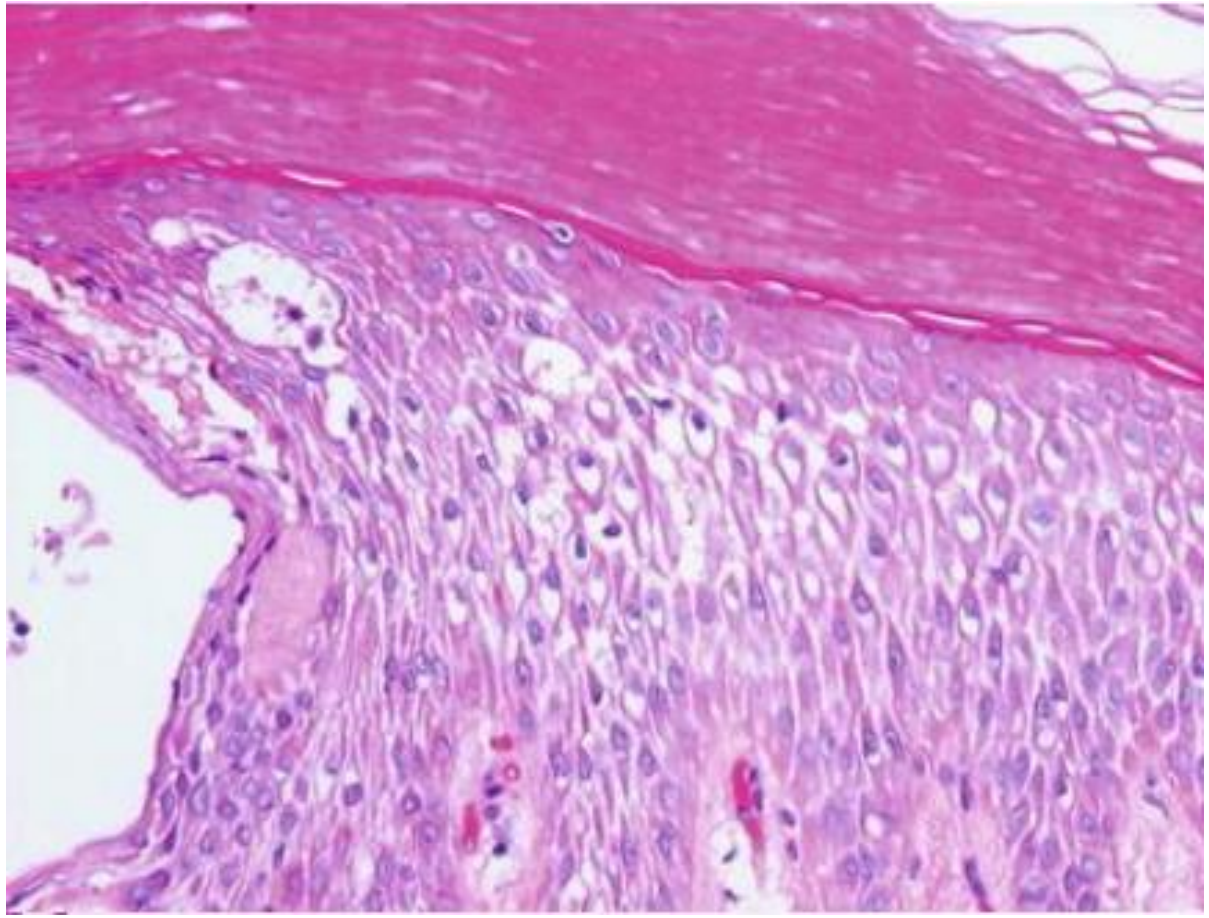


* lymphatic obst

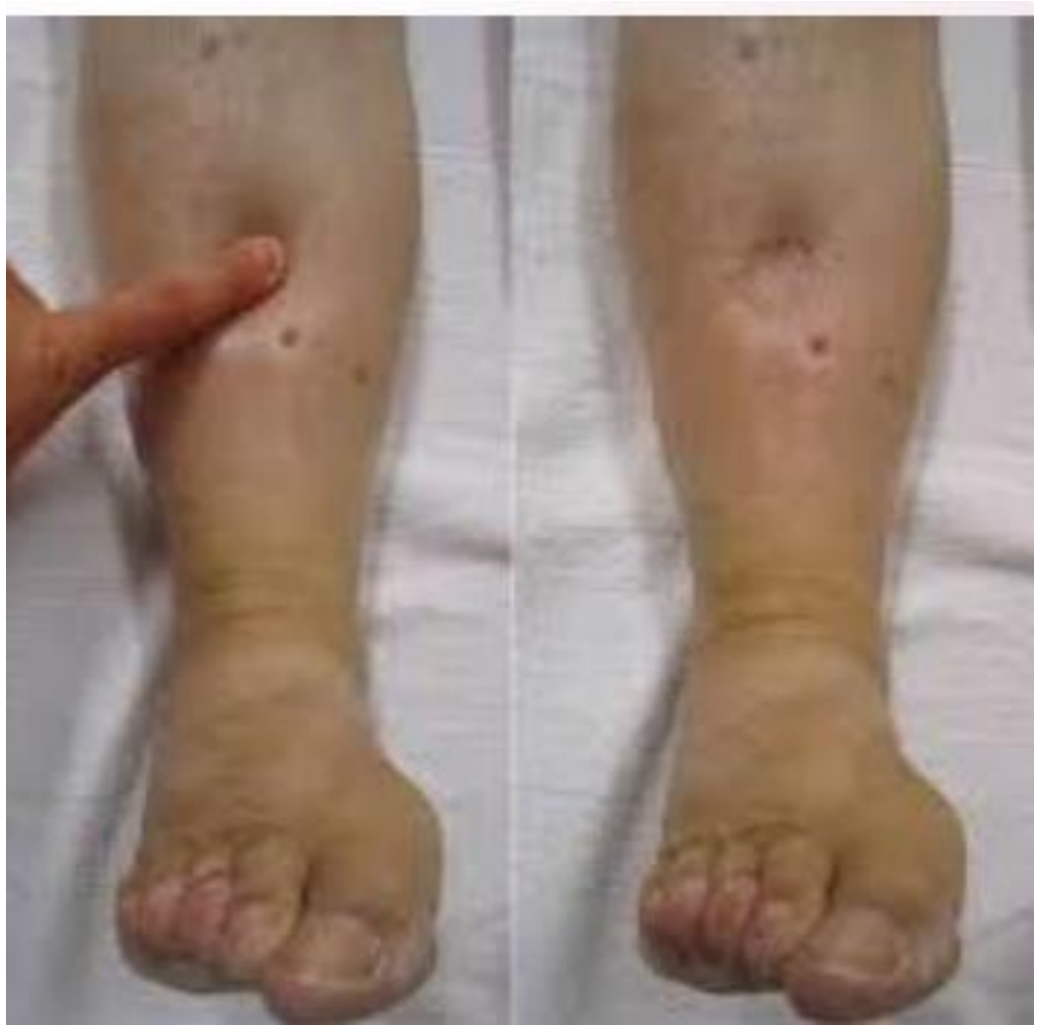
Filariasis - Robbins

eFIG. 3.2 Massive edema and elephantiasis caused by filariasis of the leg. (From Kumar V, Abbas A, Aster JC: *Robbins and Cotran Pathologic Basis of Disease*, ed. 10, Fig. 8.57, Philadelphia, 2020, Elsevier.)

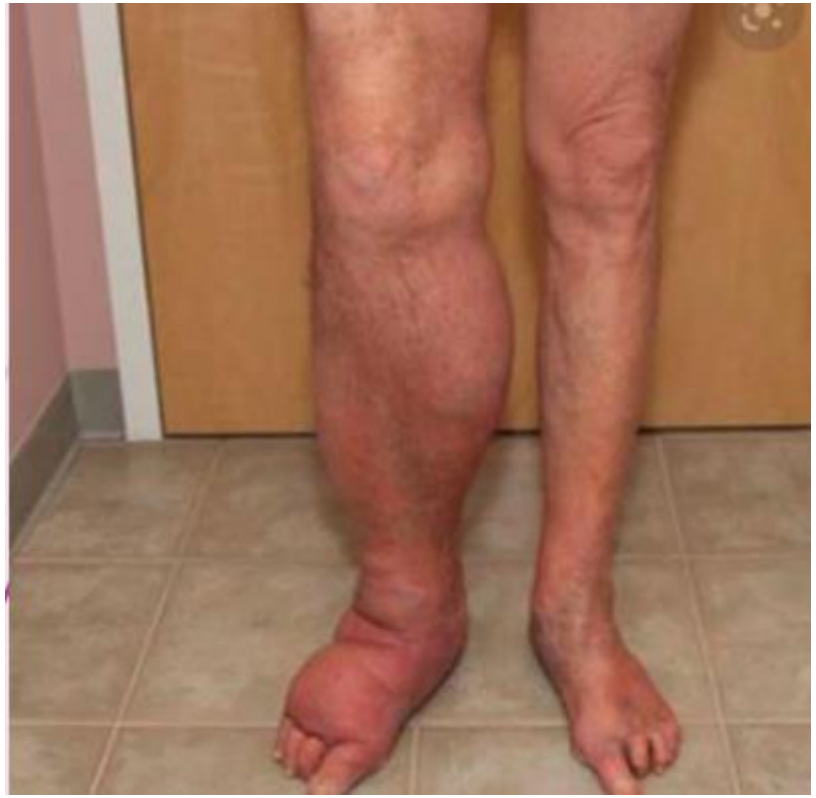
Subcutaneous Edema



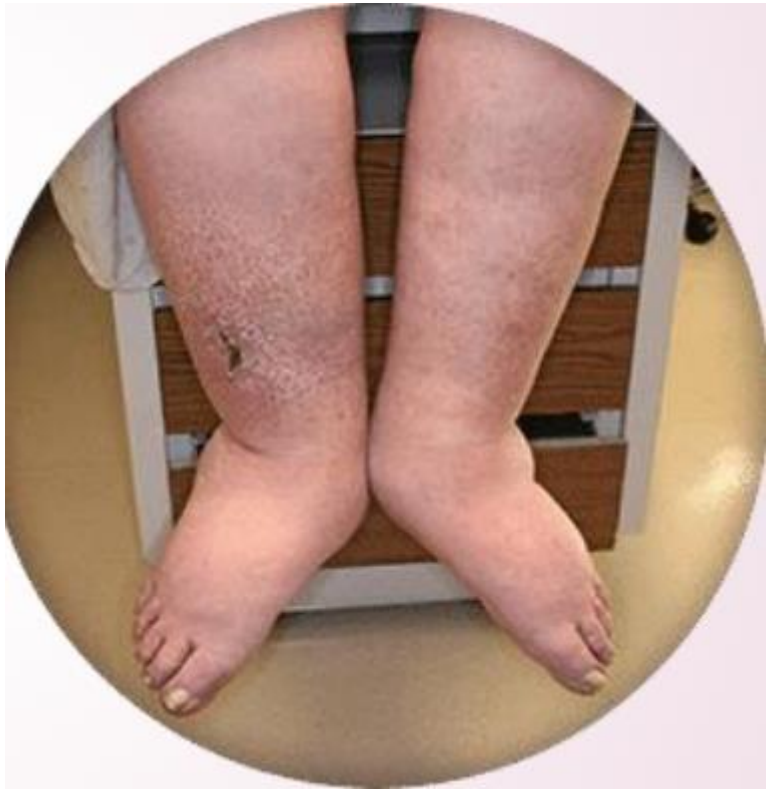
Pitting edema

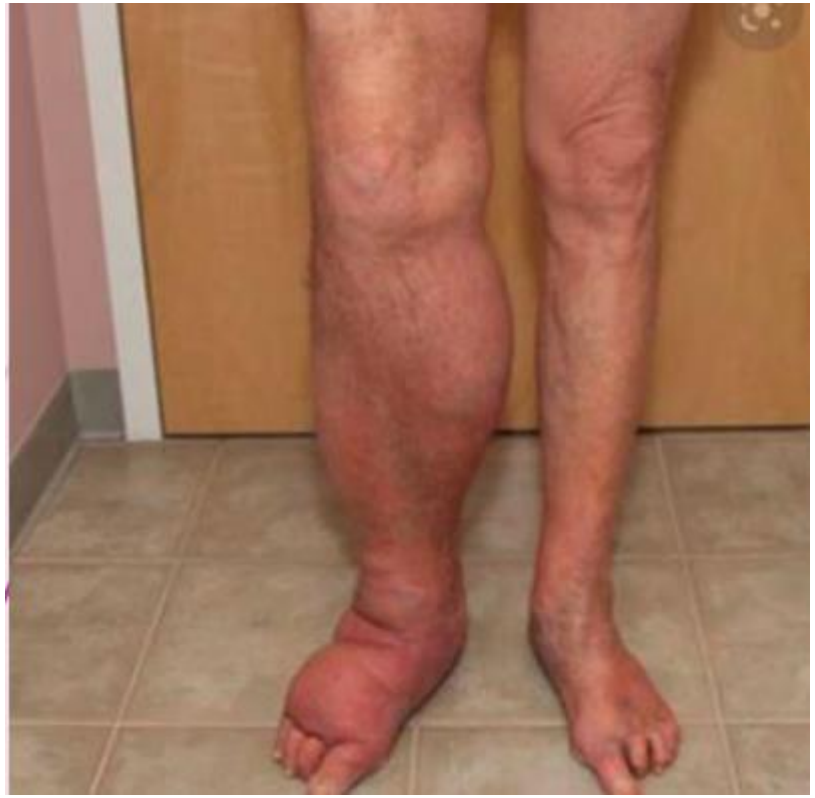


Edema



Edema

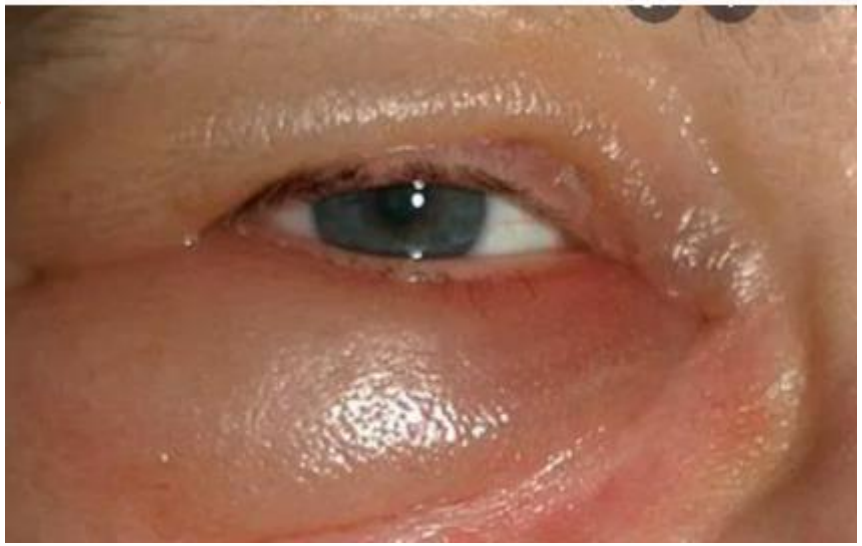




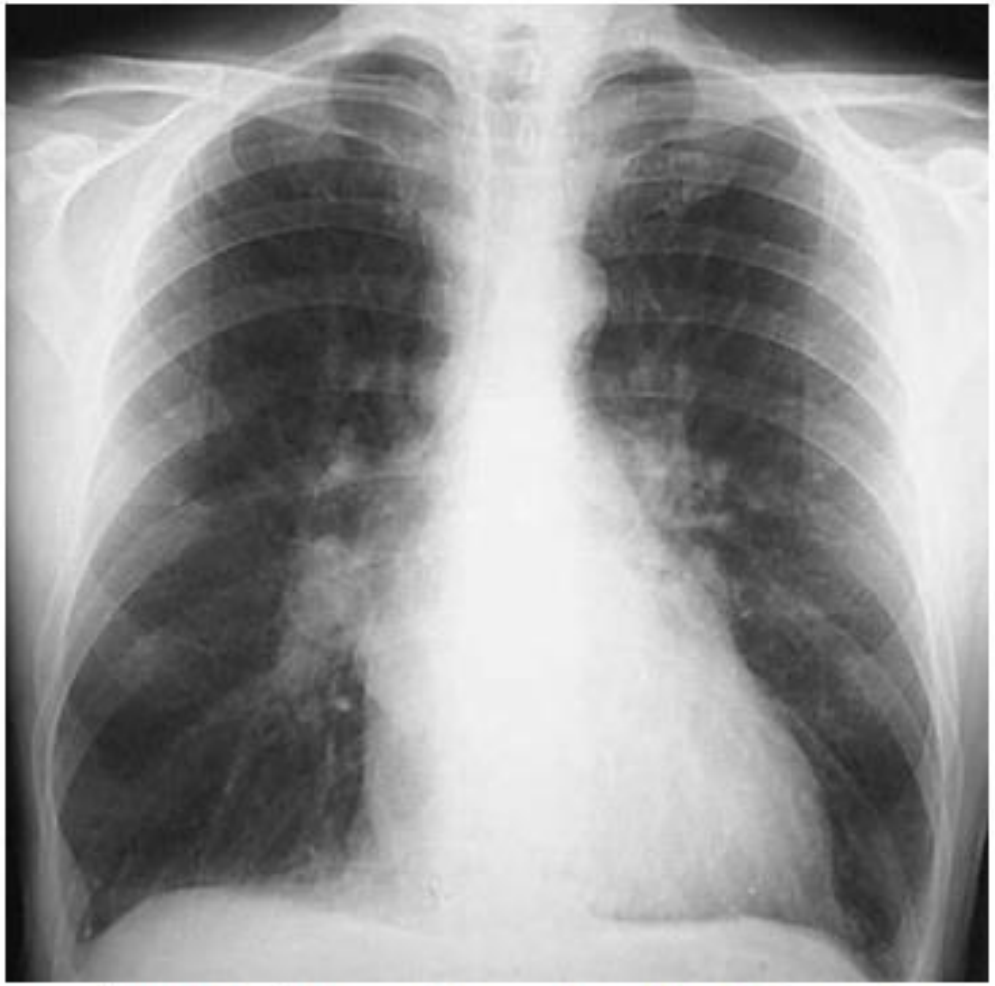
Periorbital edema

Eye 1.01

*Edema resulting from Renal dysfunction or nephrotic syndrome often manifests first in loose C.T.

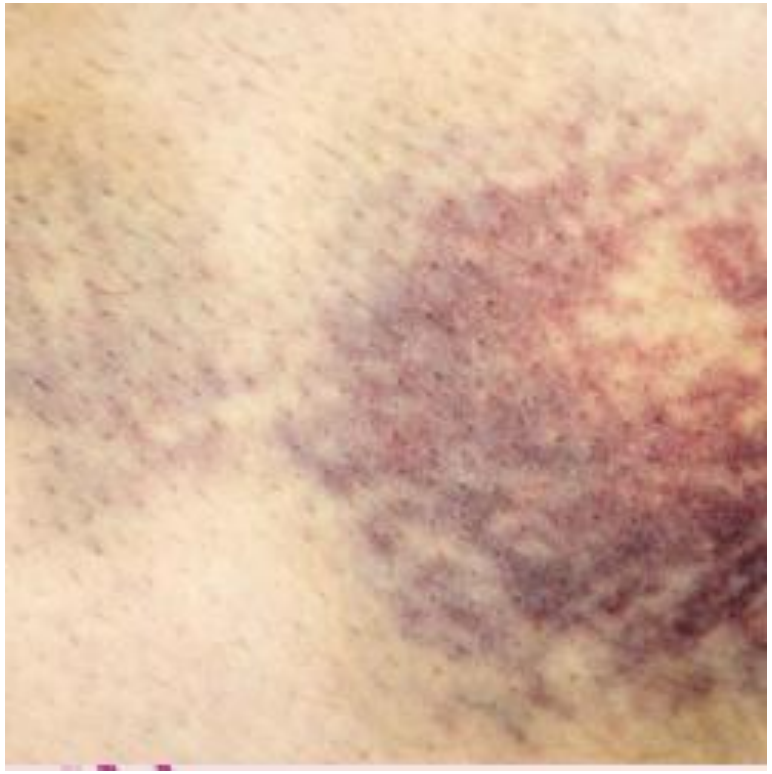


Pulmonary edema - Robbins



eFig. 3.3 Pulmonary edema. This chest x-ray in a patient with mitral stenosis shows increased lung markings, prominent pulmonary veins, and a prominent left heart border due to left atrial dilation. (From Klatt EC: *Robbins and Cotran Atlas of Pathology,* ed 4, Fig. 5.9, Philadelphia, 2021, Elsevier.)

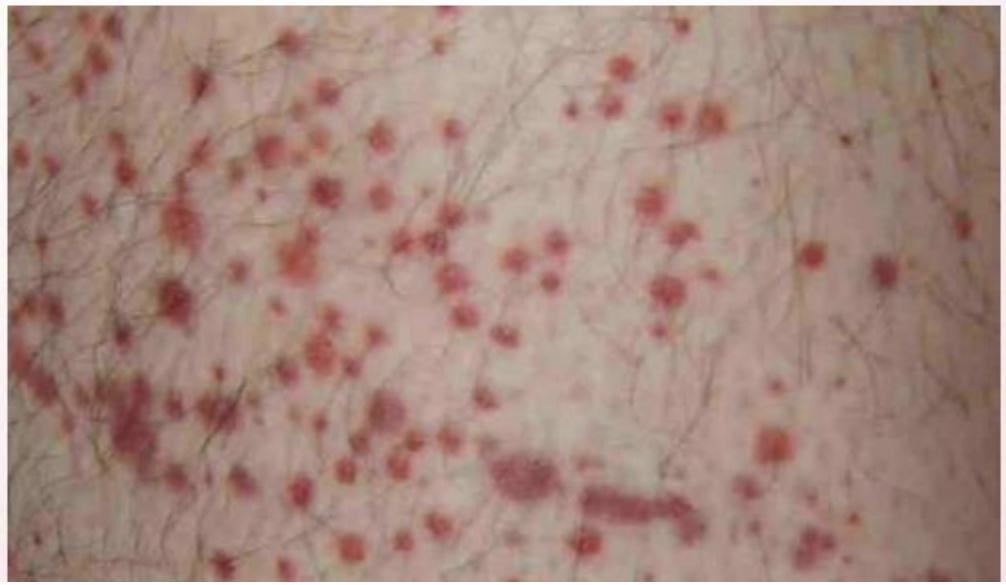
Bruise



Jaundice

*Extensive hemorrhage
→ Jaundice, by
Breakdown of RBCs
& hemoglobin

- **1. Petechiae :**
- are minute (1 to 2 mm in diameter) hemorrhages into skin, mucous membranes, or serosal surfaces
- Causes
- low platelet counts (thrombocytopenia).
- defective platelet function.
- loss of vascular wall support, as in vitamin C deficiency.



2. Purpura

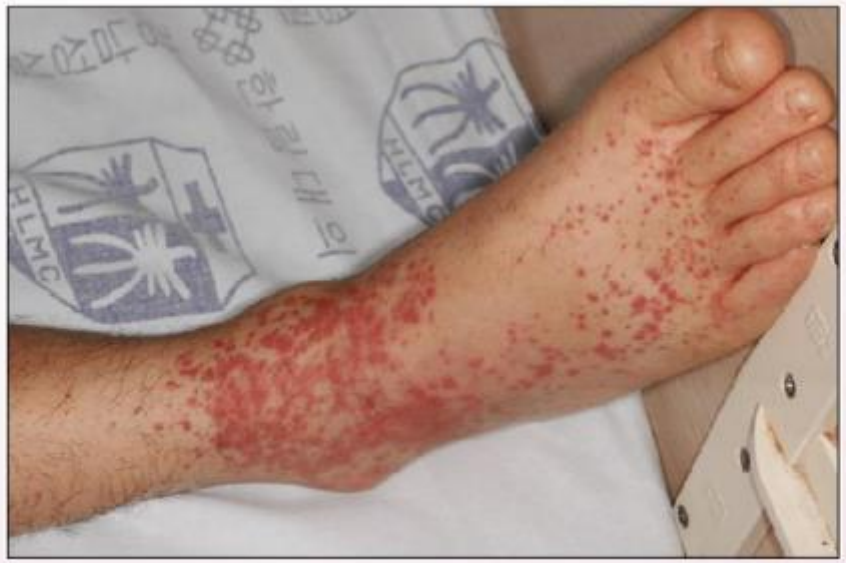
are slightly larger (3 to 5 mm) hemorrhages.

Purpura can result from the same disorders that cause petechiae, as well as:

trauma.

vascular inflammation (vasculitis).

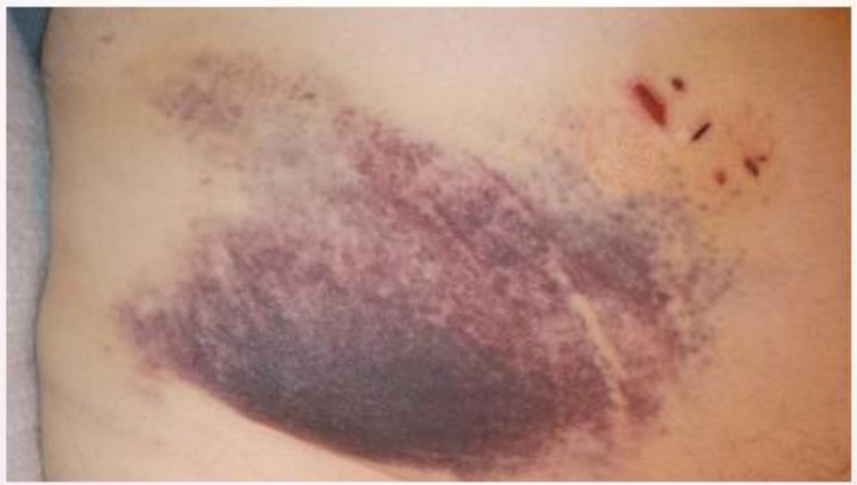
increased vascular fragility.



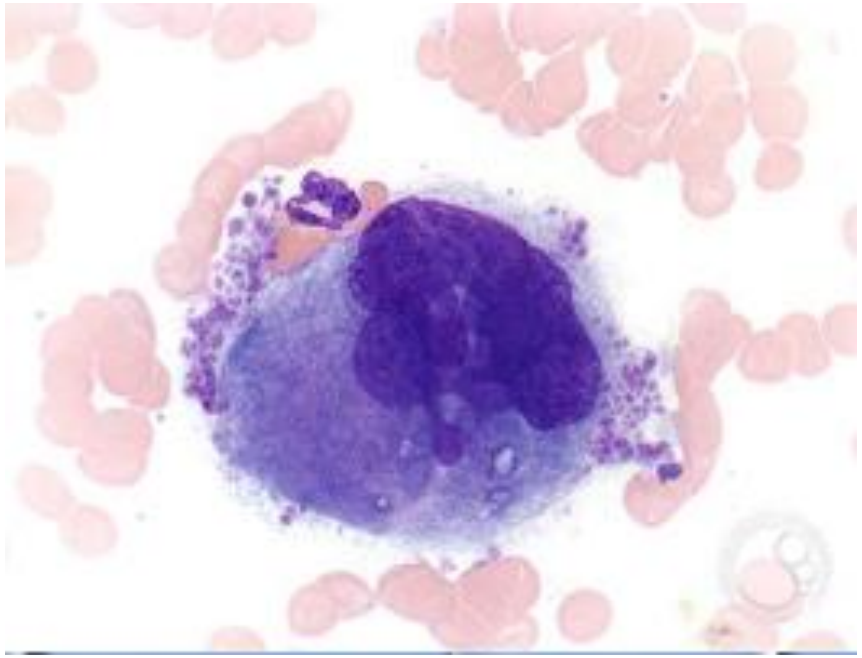
3.Ecchymoses:

are larger (1 to 2 cm) subcutaneous hematomas (also called bruises).

Extravasated red cells are phagocytosed and degraded by macrophages; the characteristic color changes of a bruise result from the enzymatic conversion of hemoglobin (red-blue color) to bilirubin (blue-green color) and eventually hemosiderin (golden-brown)



Megakaryocyte

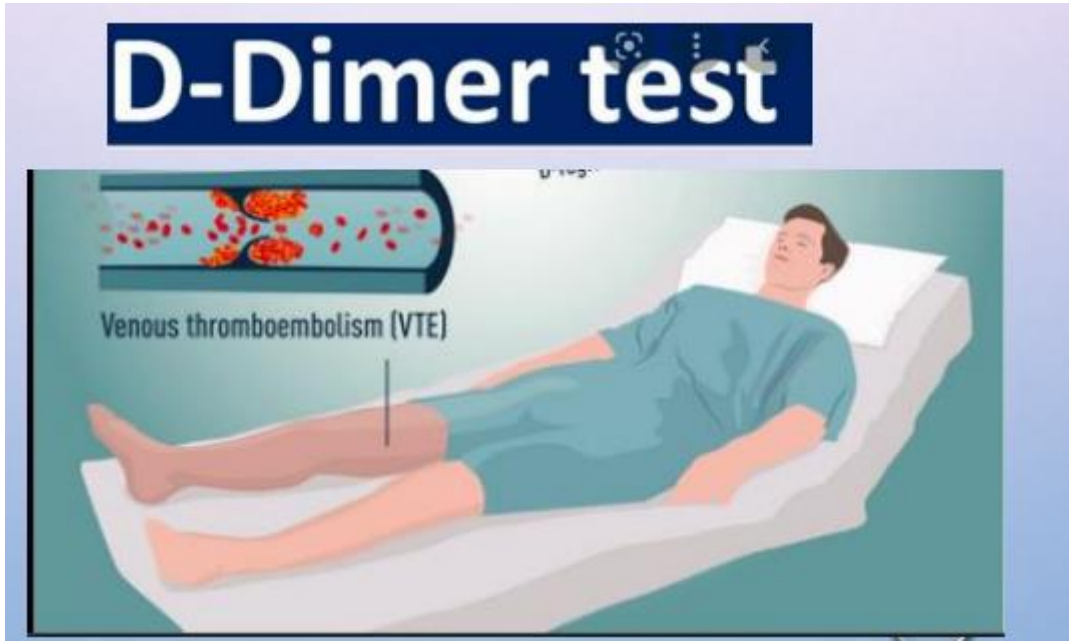


* Vitamin K - antagonist

* Vitamin K - dependent Coag. factors 10, 9, 7, 2

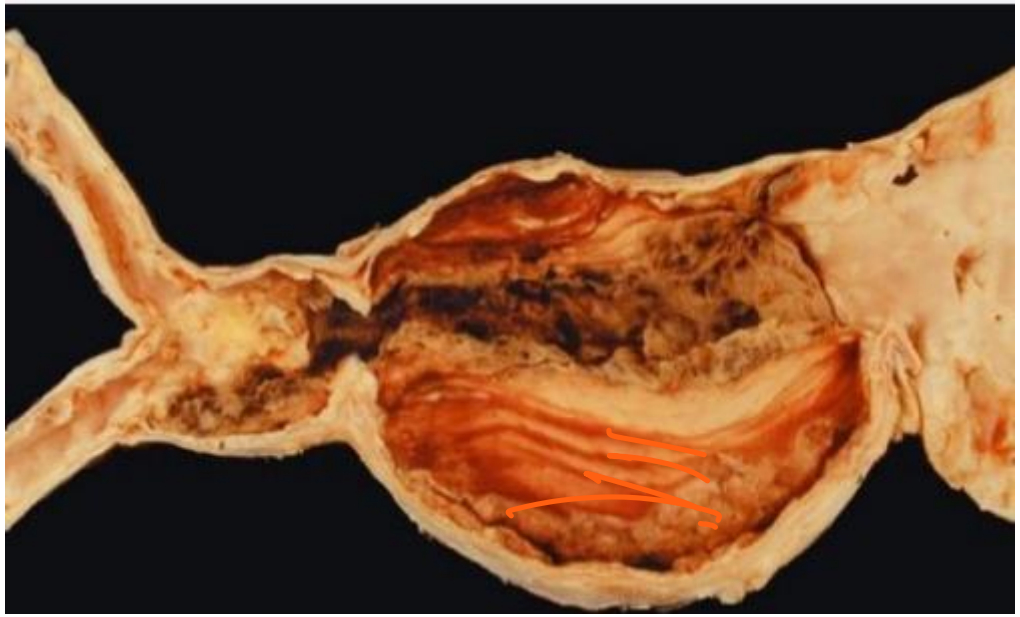
- DVT

* D-Dimer is a fibrin degradation product

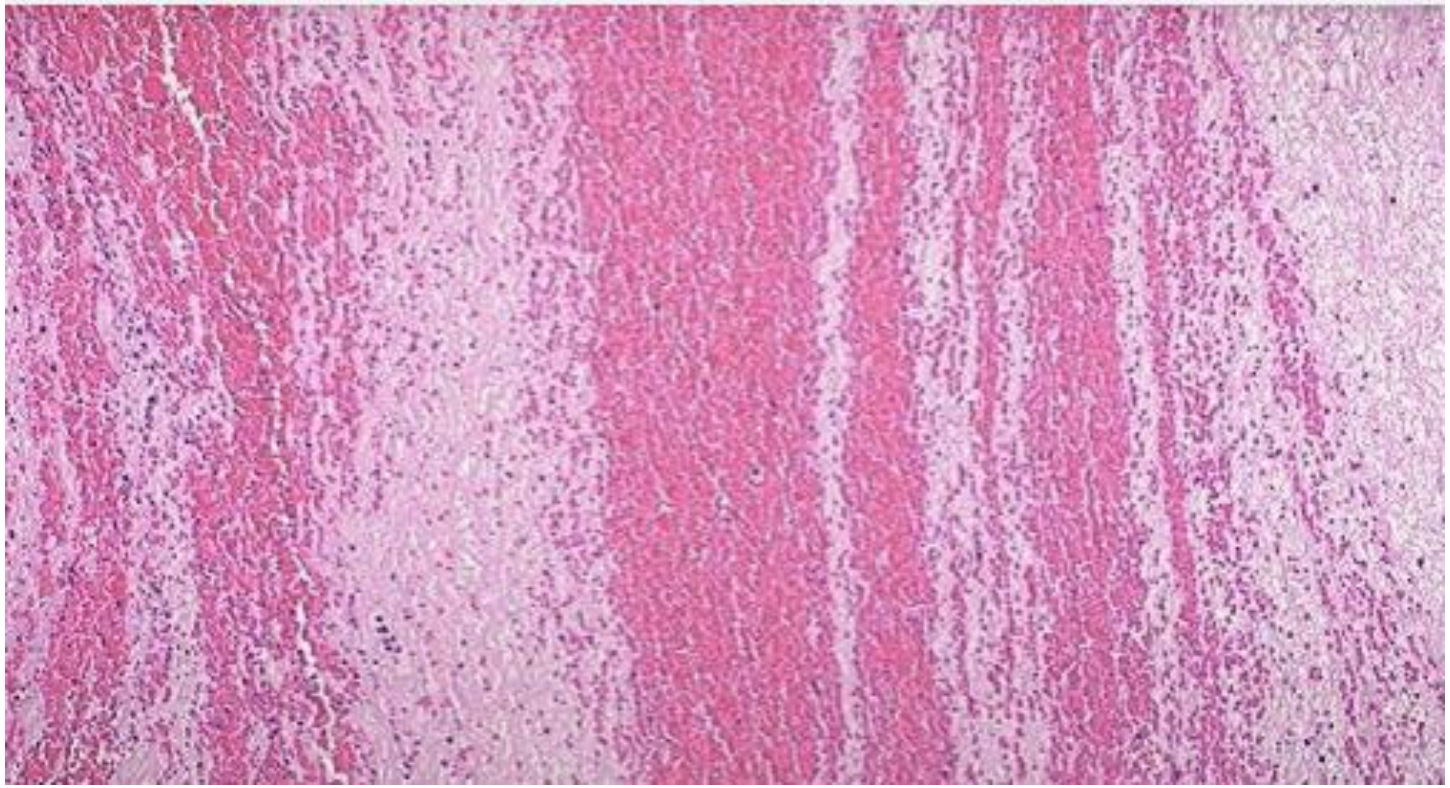


Lines of Zahn

* Abdominal Aortic Aneurysm



Lines of Zahn



Mural Thrombi

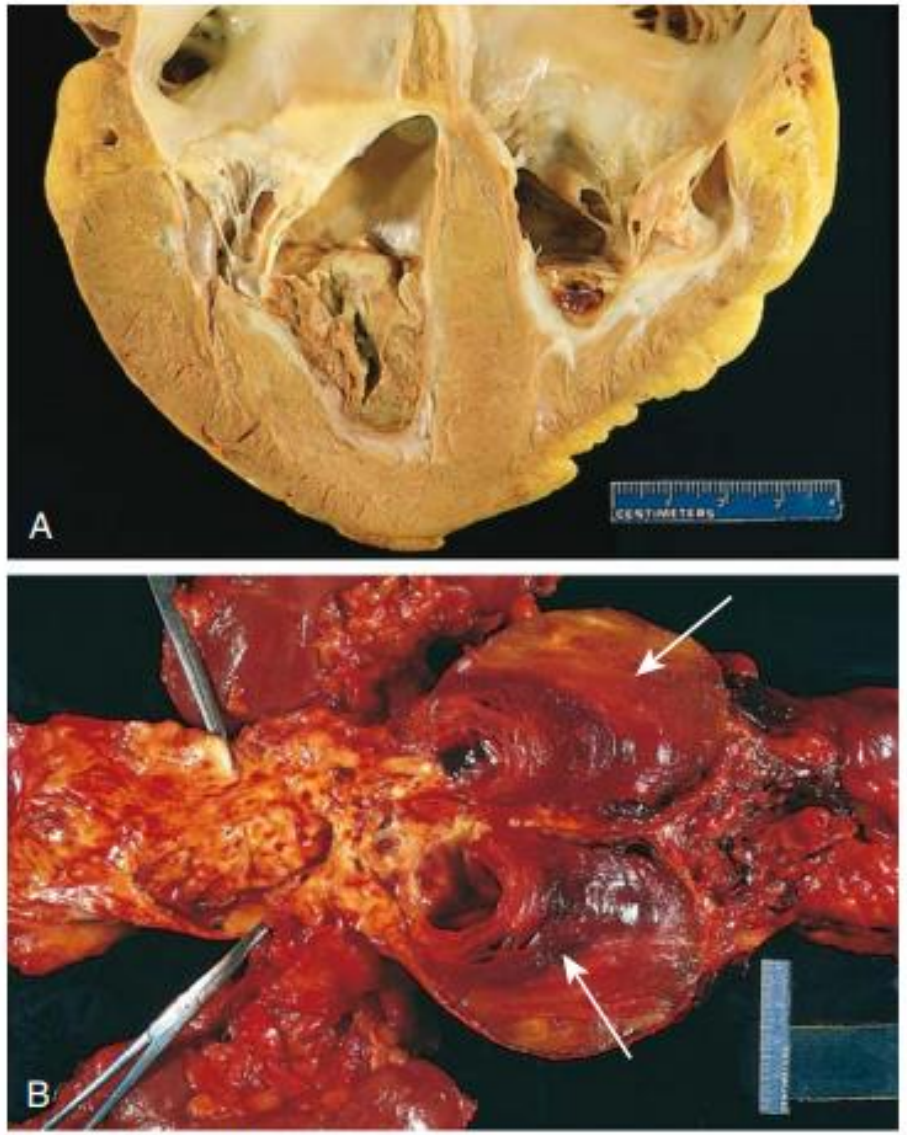
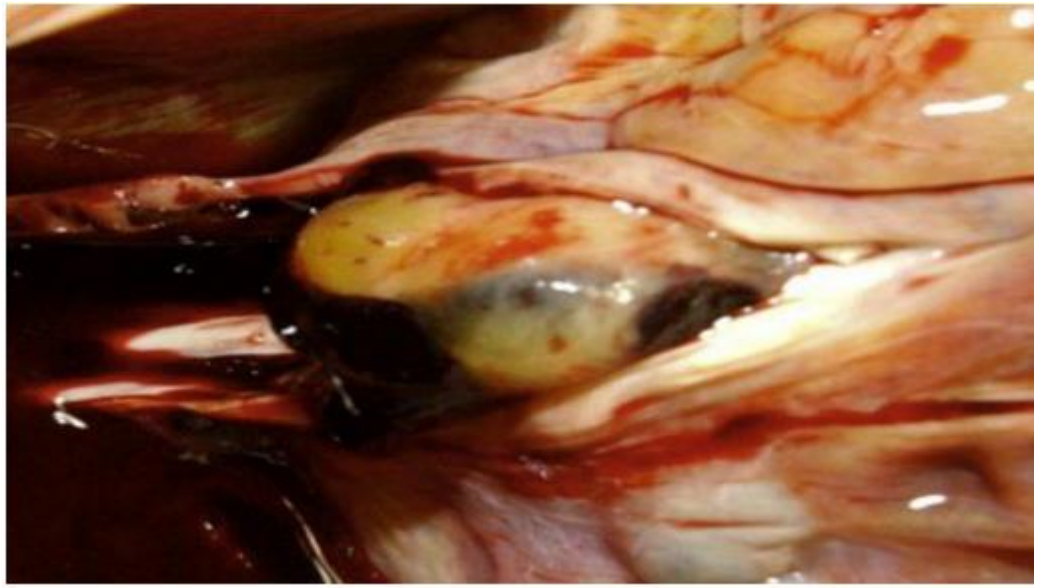


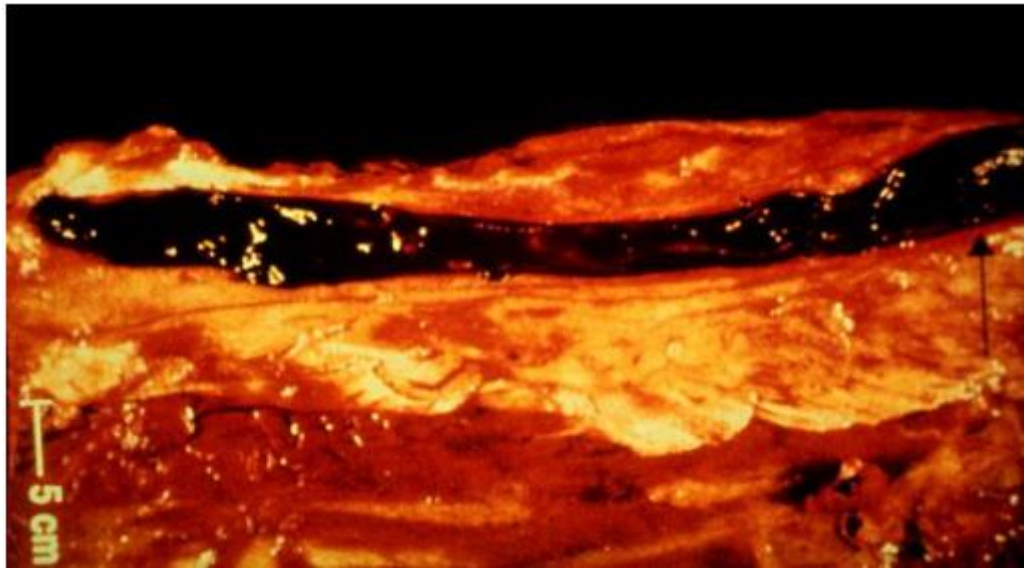
FIG. 3.13 Mural thrombi. (A) Thrombus in the left and right ventricular apices, overlying white fibrous scar. (B) Laminated thrombus *(arrows)* in a dilated abdominal aortic aneurysm. Numerous friable mural thrombi are also superimposed on advanced atherosclerotic lesions of the more proximal aorta *(left side of photograph)*.

Postmortem clot

* Chicken fat clot
* Forensic medicine
* Not a cause of death



Venous clot



Organized thrombus

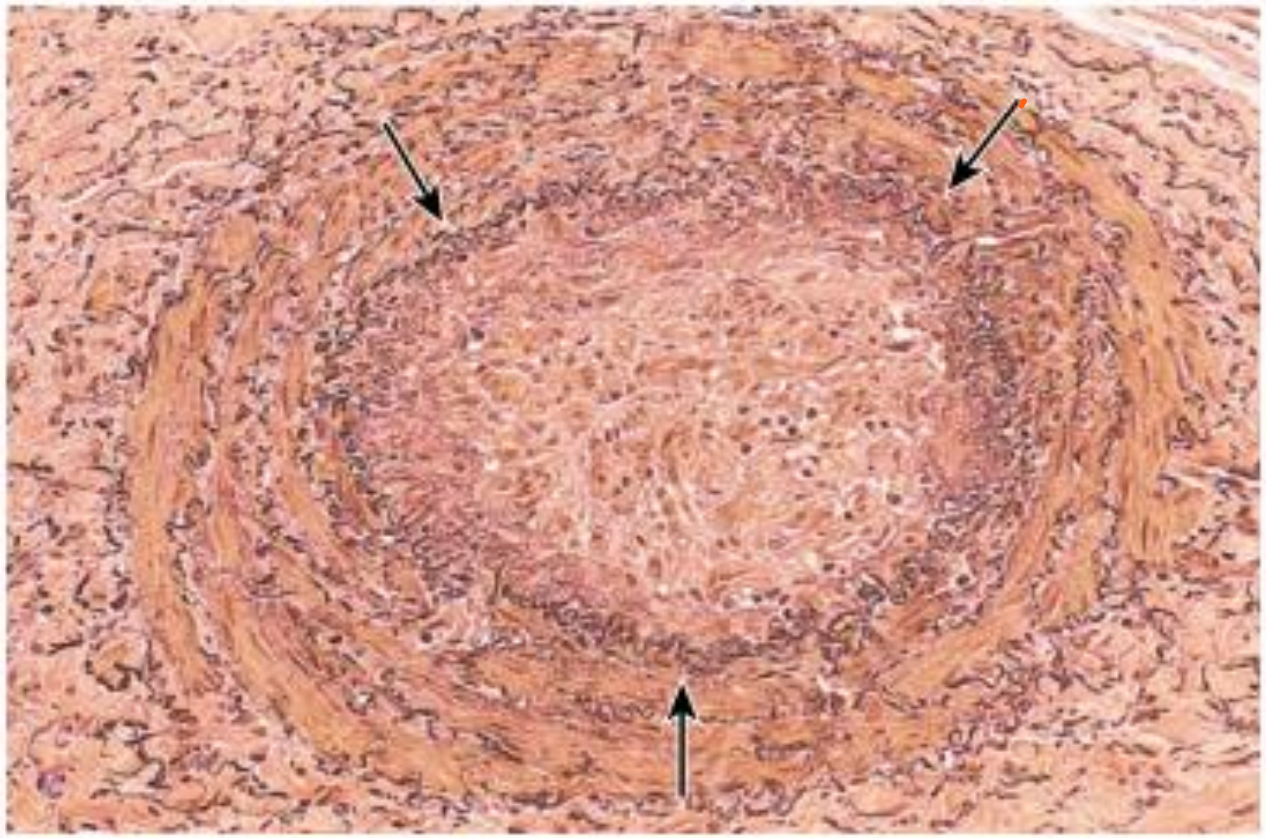
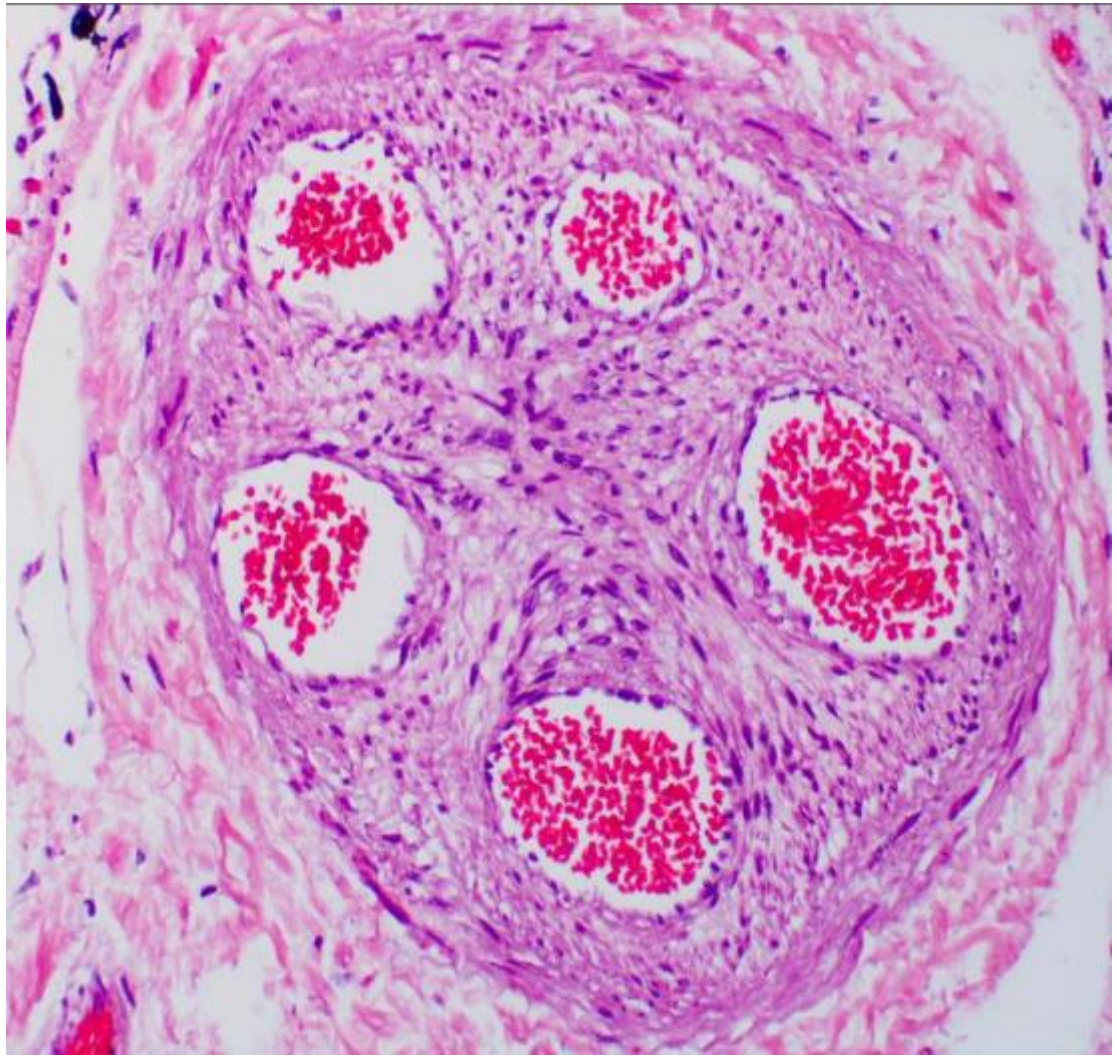
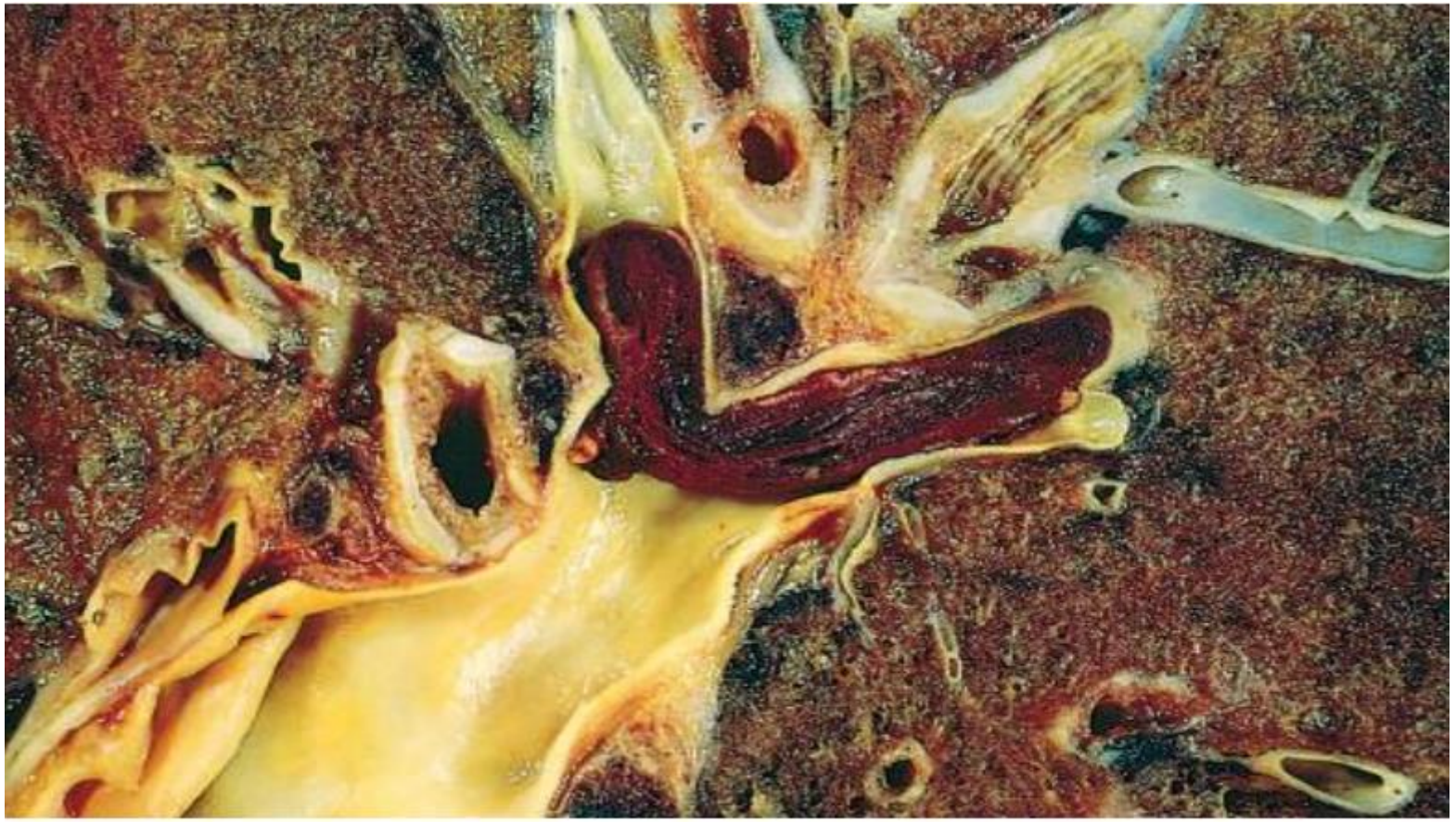


FIG. 3.14 An organized thrombus. Low-power view of a thrombosed artery stained for elastin. The original lumen is delineated by the internal elastic lamina *(arrows)* and is completely filled with organized thrombus.

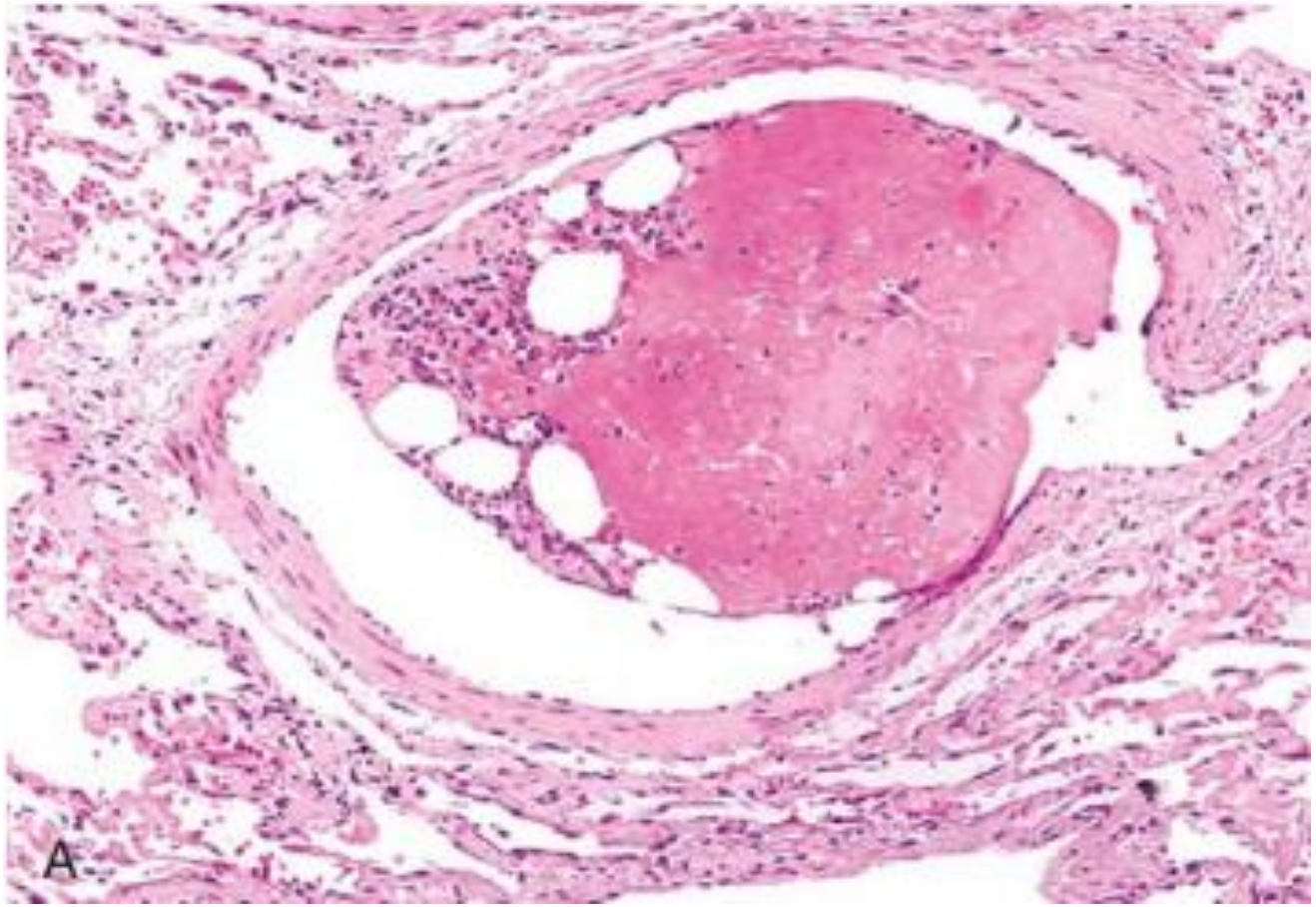
Recanalization



Saddle embolus

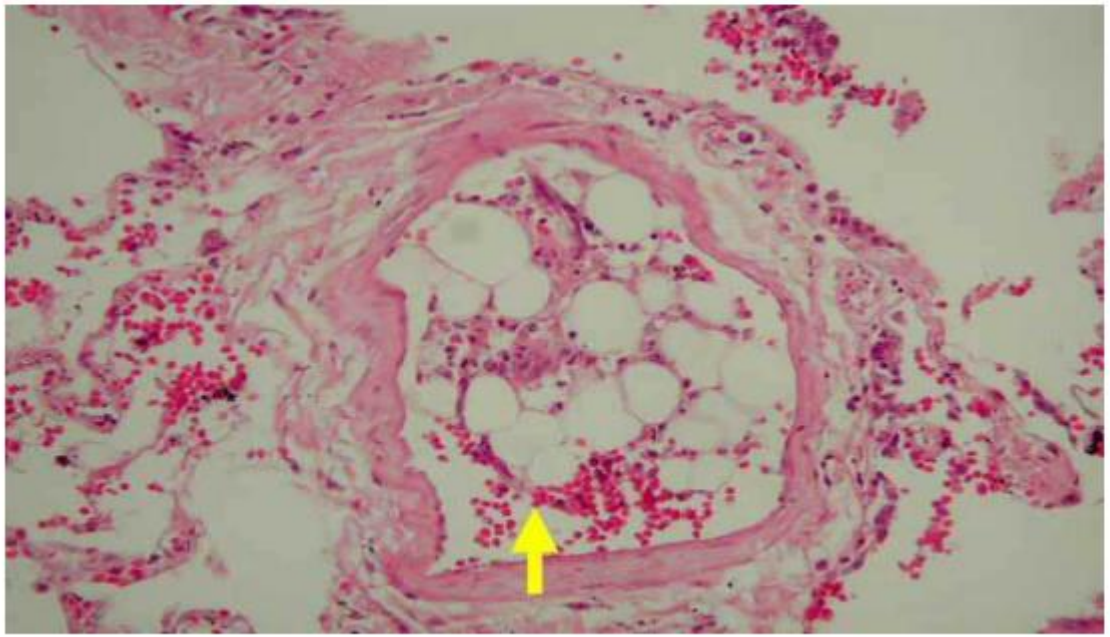


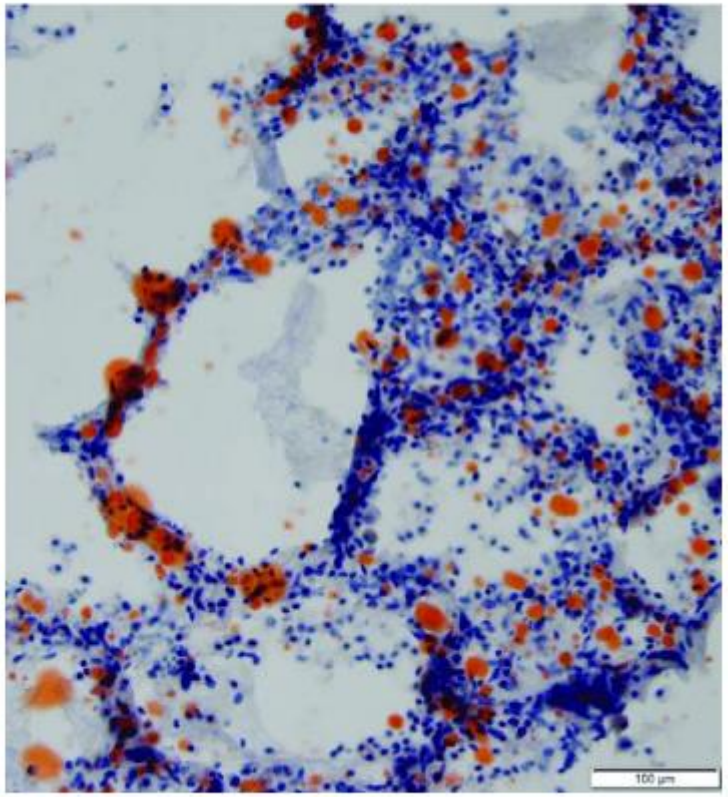
Fat embolus



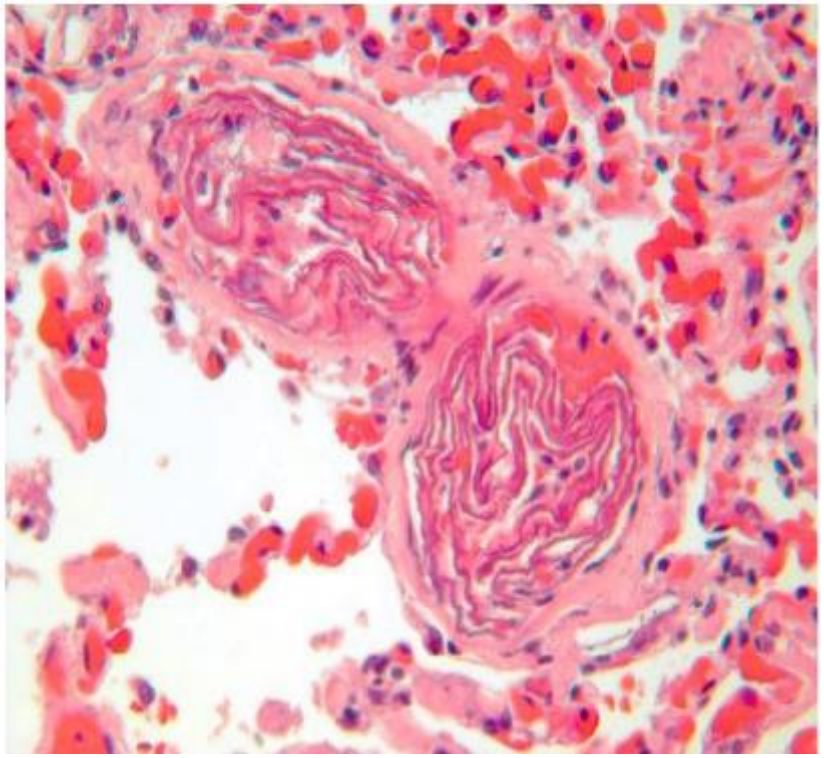
Fat embolus

Lipids are dissolved by the solvents during tissue processing → so microscopic demonstration of fat microglobules (especially in the absence of accompanying marrow elements) requires specialized techniques (frozen sections & fat stains).



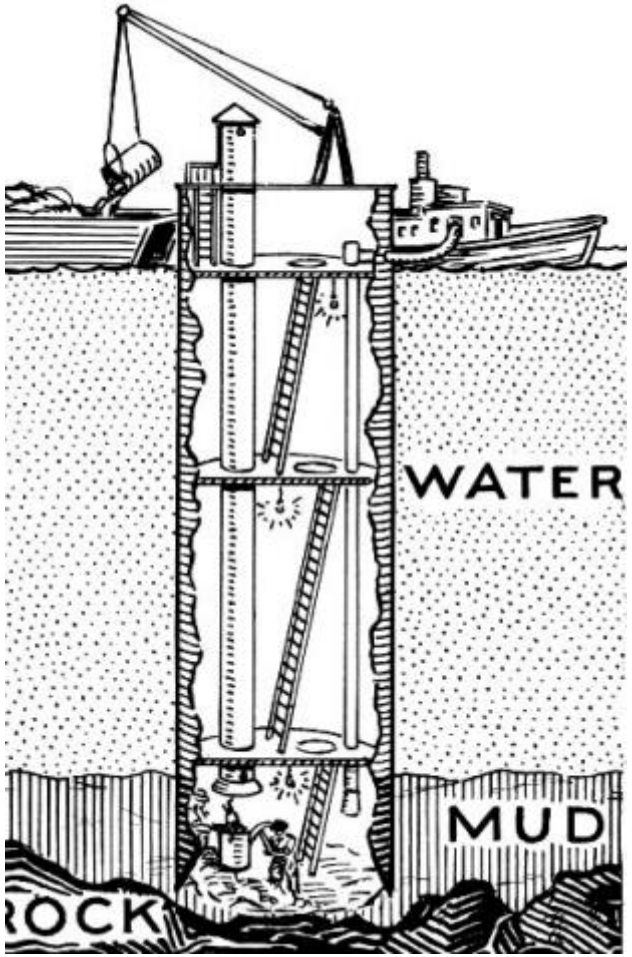


Amniotic fluid embolus

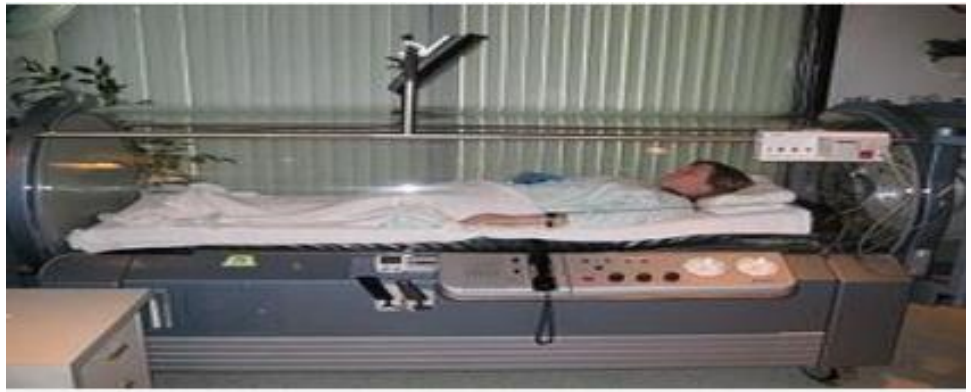


Caisson

* Chronic decompressio Sickness
* multifocal ischemic necrosis
* Fumur, tibiae humeri



High pressure chamber



Red infarcts

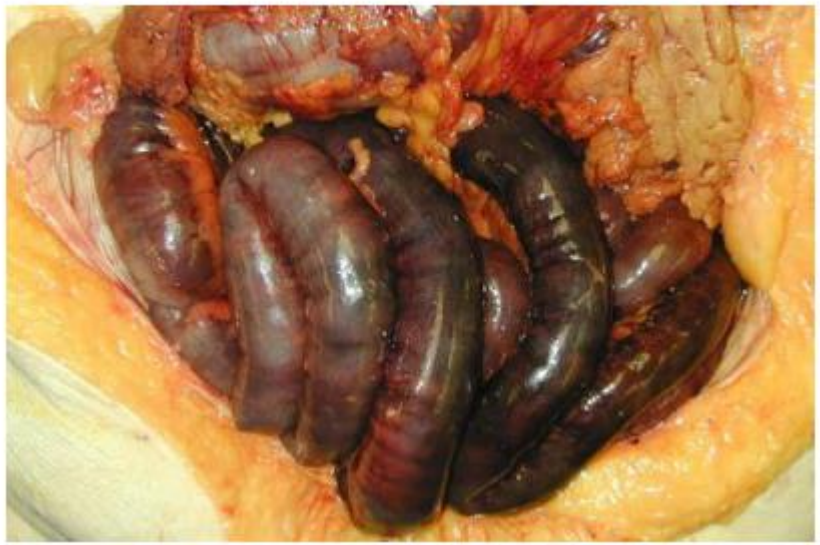
(1) **Venous** occlusions (ovarian torsion).

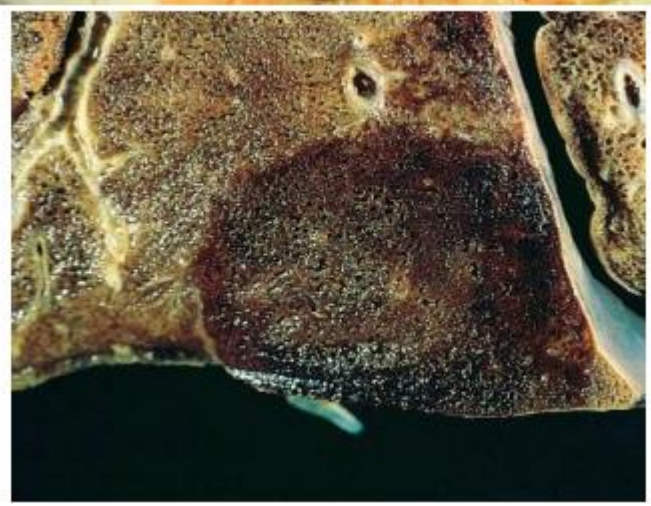
(2) **In loose tissues** (e.g., lung) where blood can collect in infarcted zones.

(3) Tissue with **dual circulations** (lung & small intestine); it allow blood to flow from an unobstructed (collateral)into a necrotic zone.

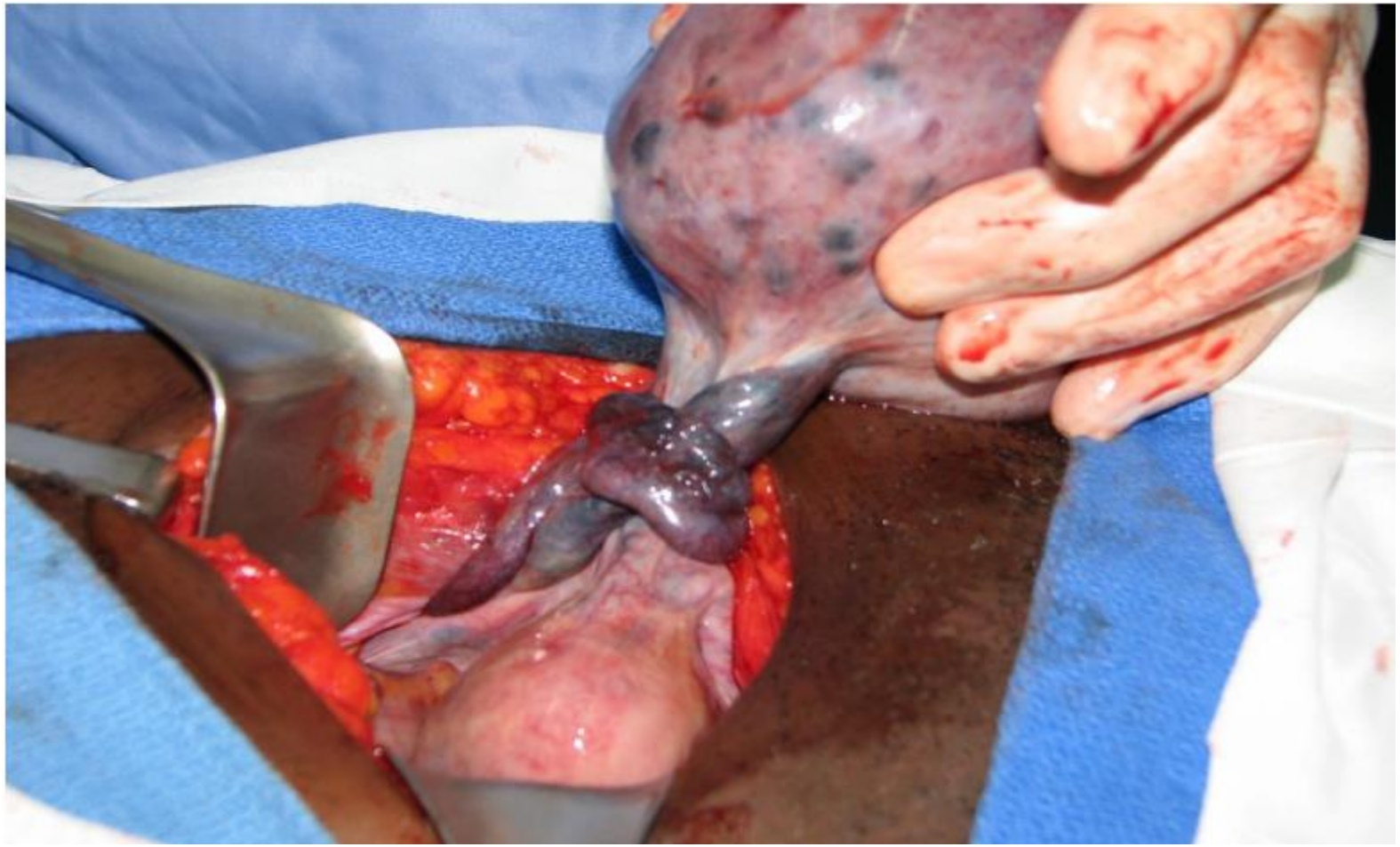
(4) previously **congested tissues** (as a consequence of sluggish venous outflow).

(5) when **flow is reestablished** after infarction has occurred (e.g., after angioplasty of an arterial obstruction).





Adnexal torsion



White Infarction

- In arterial occlusions in **solid** organs **with end-arterial circulations** (heart, spleen, & kidney), **tissue density limits the seepage** of blood from adjoining patent capillary beds.
- **wedge-shaped**, with the occluded vessel at the apex & the organ periphery forming the base.
- If the base is a serosal surface, there is an overlying fibrinous exudate.
- Lateral margins may be irregular, reflecting flow from adjacent vessels.
- Fresh infarcts are poorly defined & slightly hemorrhagic.
- over a few days the margins tend to become better defined by a narrow rim of congestion (inflammation).

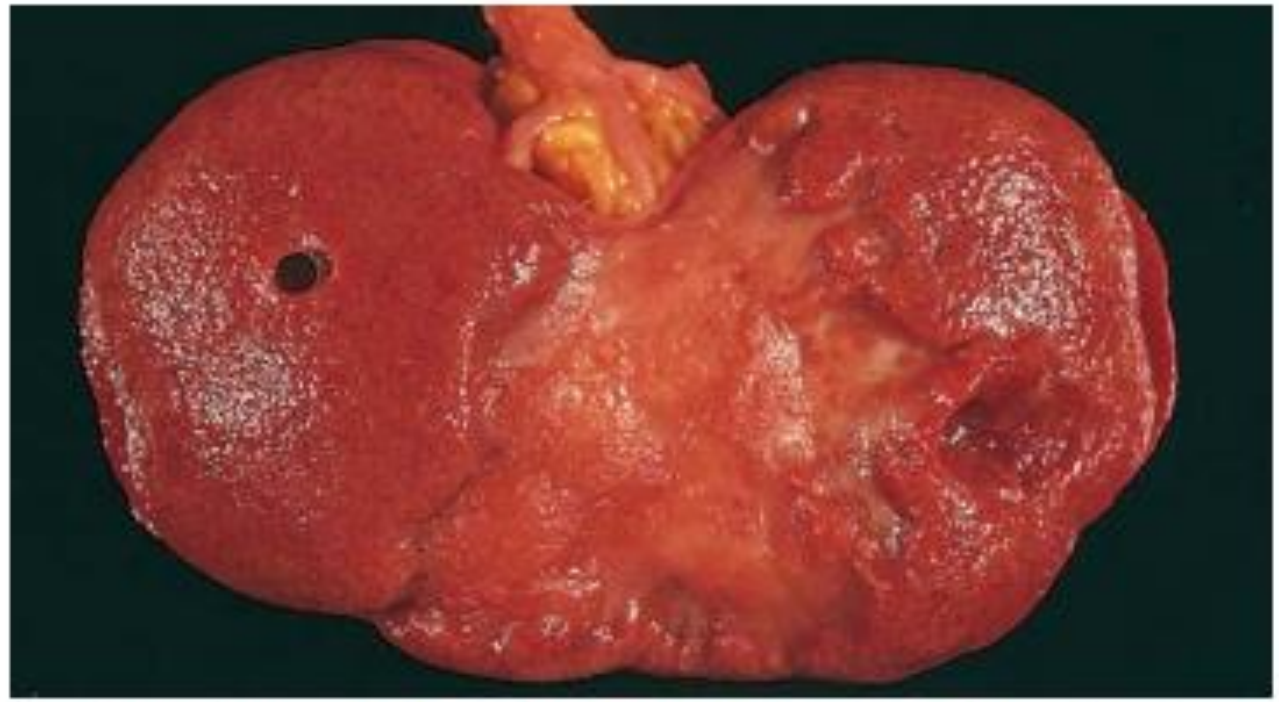
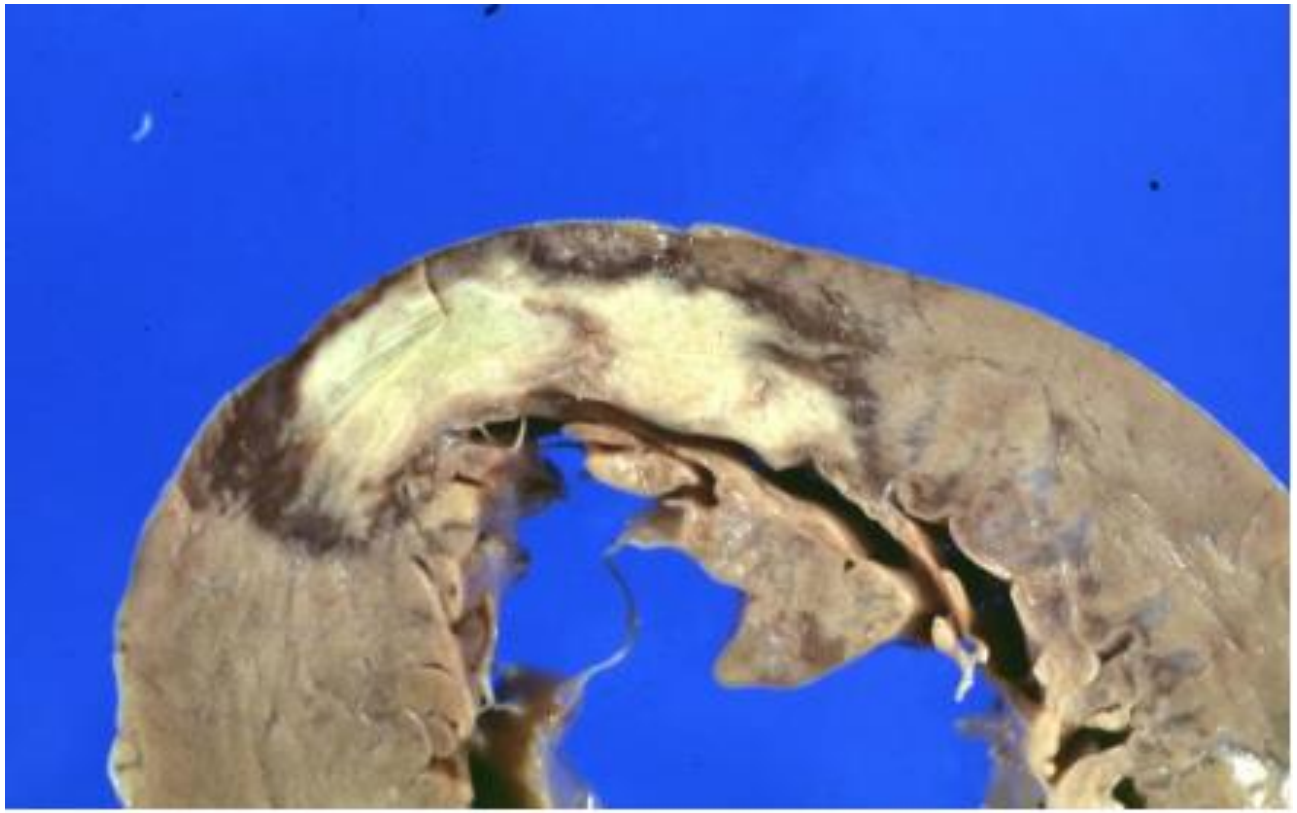


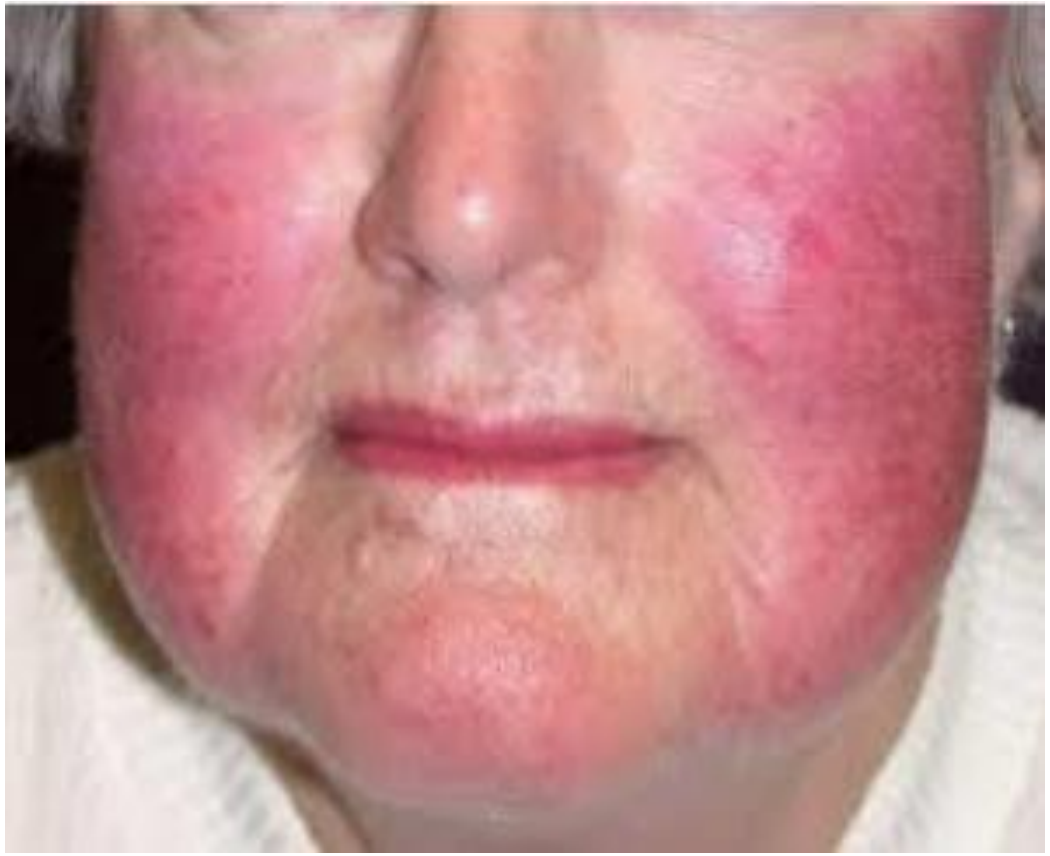
FIG. 3.18 Remote kidney infarct, now replaced by a large fibrotic scar.

Myocardial infarction



Septic shock

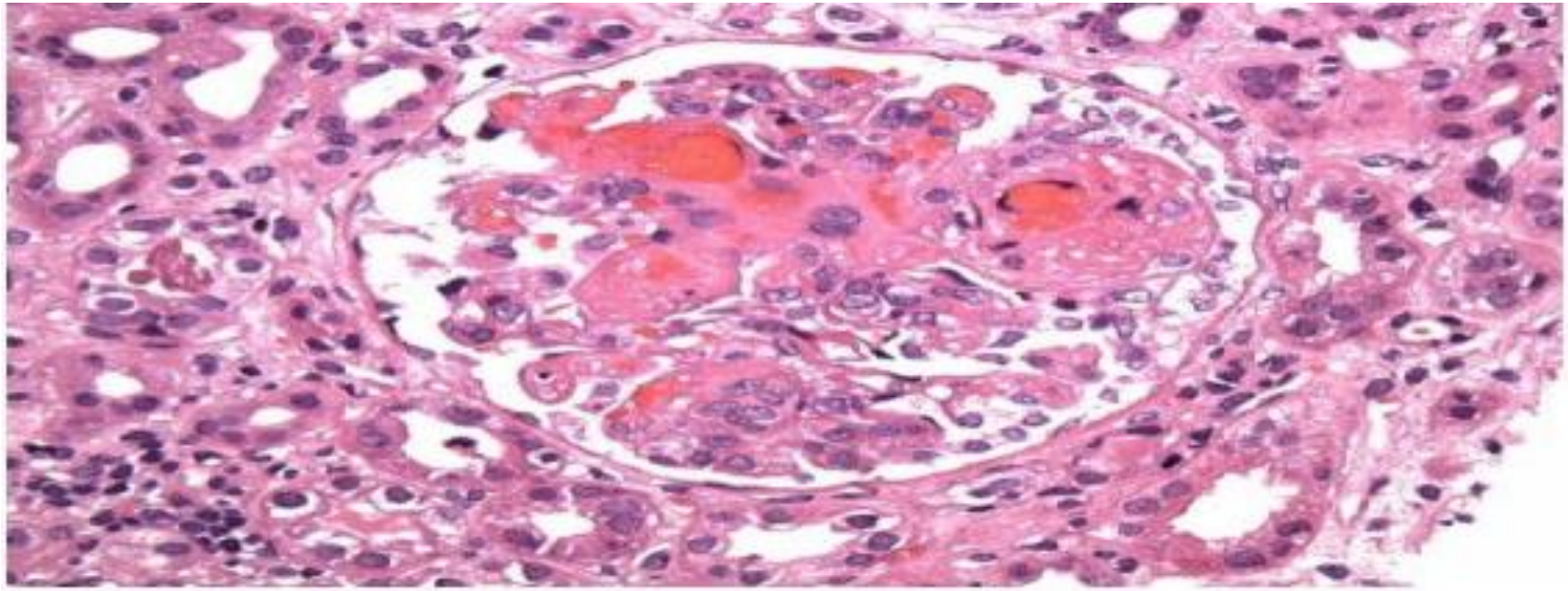
* Redness
* hetness
* vasodilation



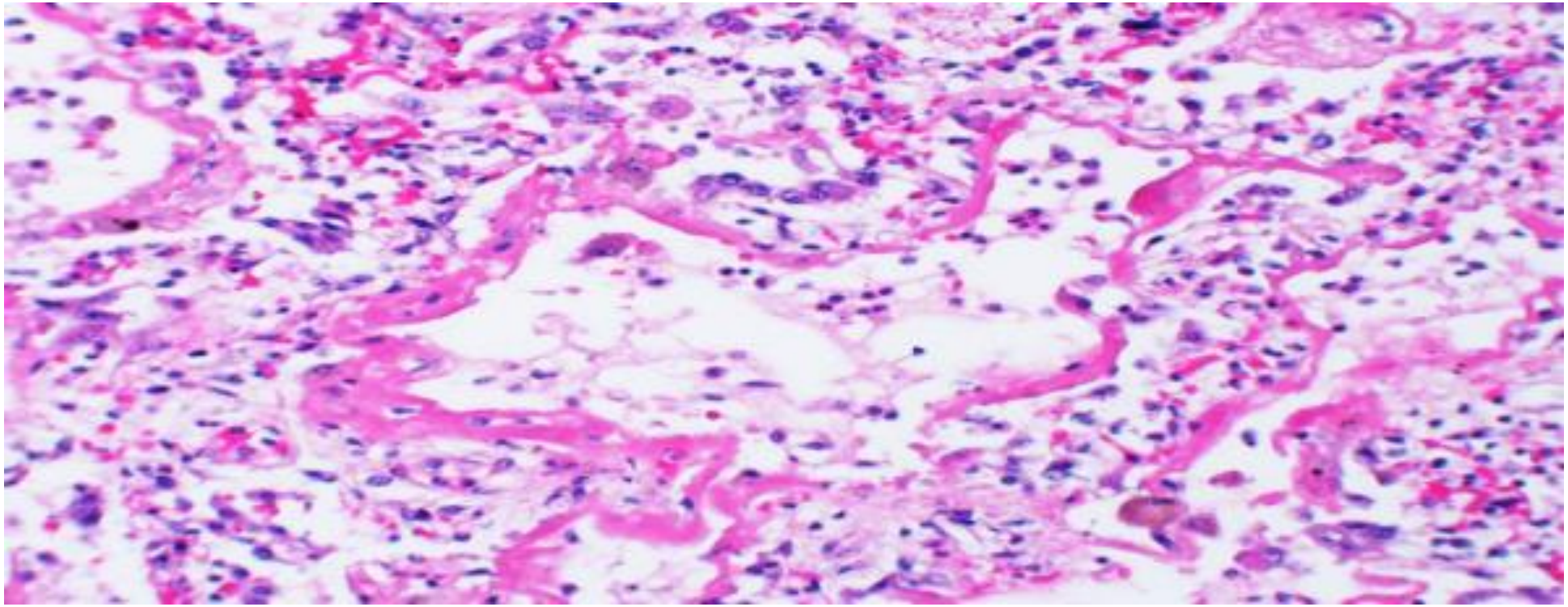
Hypovolemic shock

✗ pallor ✓

* Glomeruli → DIC
* Small thrombi
* happens in very small circulation → Kidney, Brain, Adrenals



* Acute Respiratory distres syndrome (Diffuse Alveolar damage)
* Fibrin deposition
* Inflamatory cells



Alveolar space ↩

«Wherever the art of medicine is loved,
there is also a love of humanity.»

- Hippocrates-

Charting New Horizons in Education