General pathology lab cell injury and inflammation.

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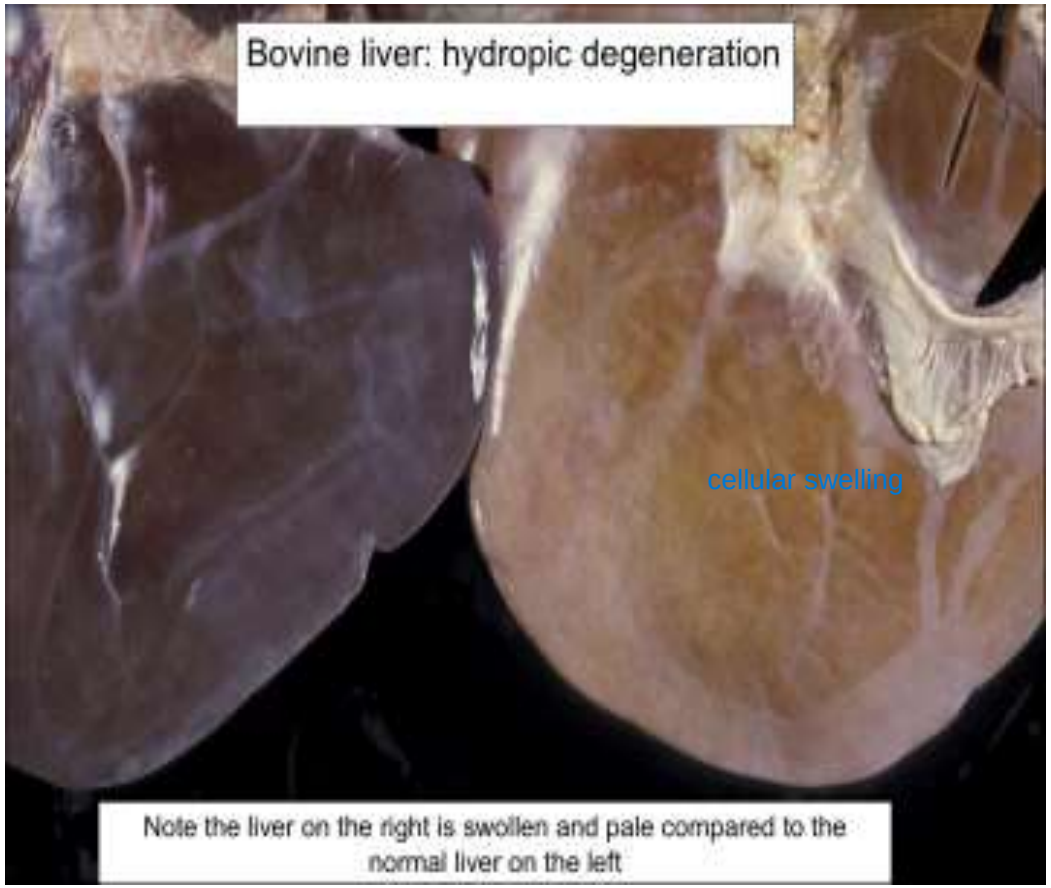
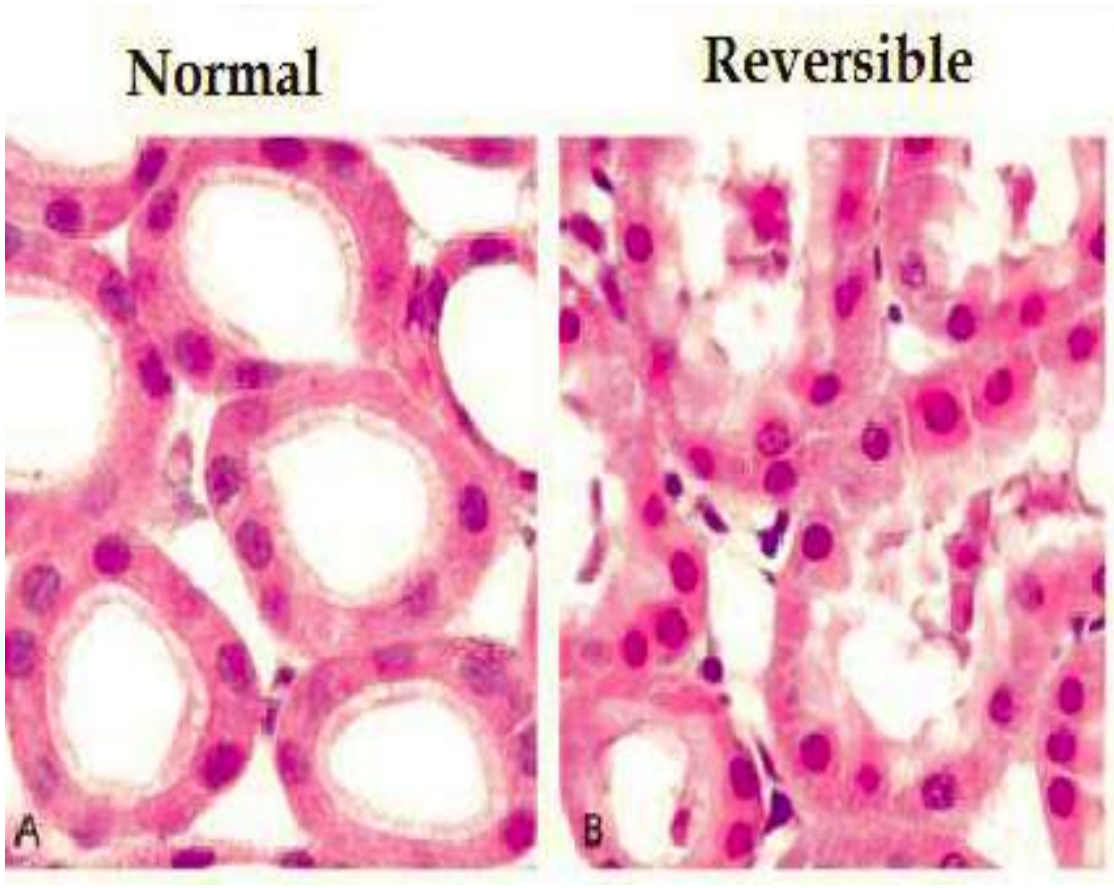
Morphological changes of reversible cell injury:
1. Cellular Swelling

cytoplasm passed its normal boundaries

renal tubules

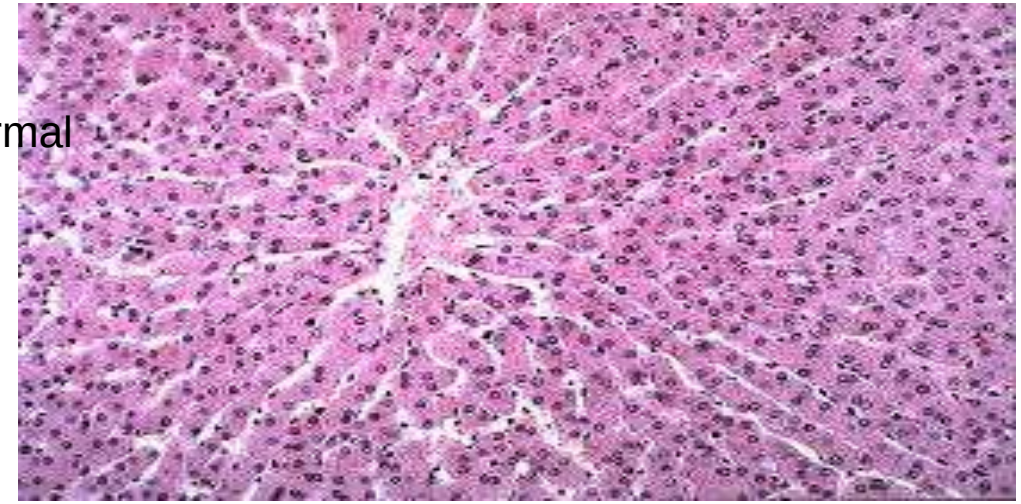


cellular swelling

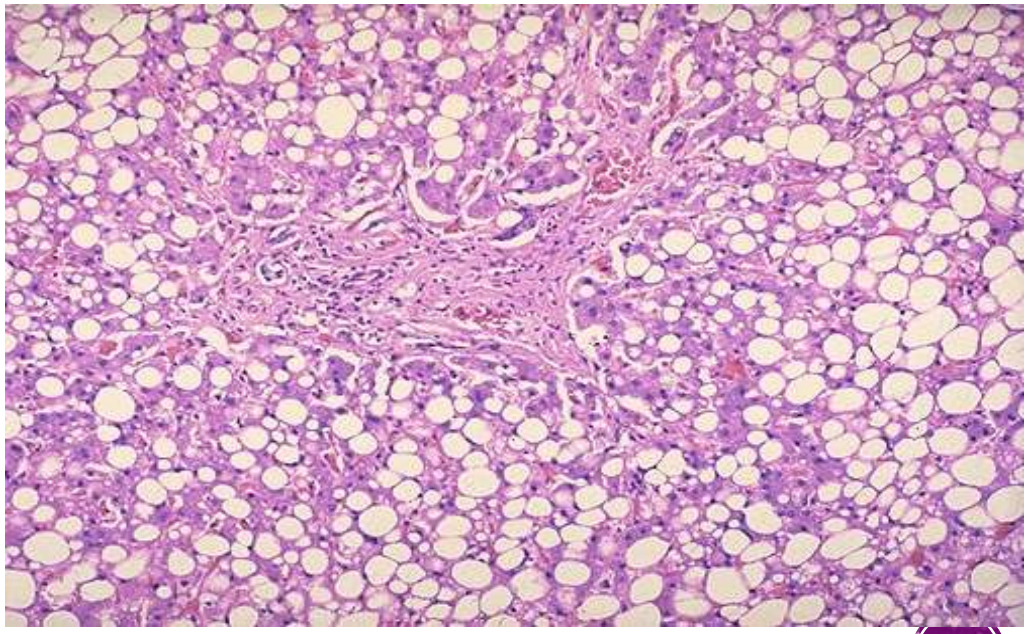


2. Fatty change

normal



cells contain fatty globules

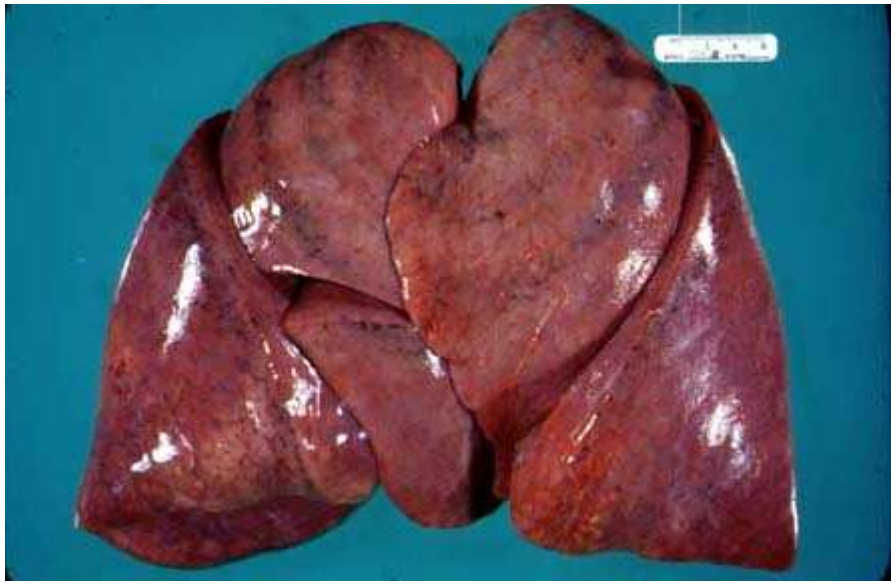


irreversible

Morphological features of necrosis:

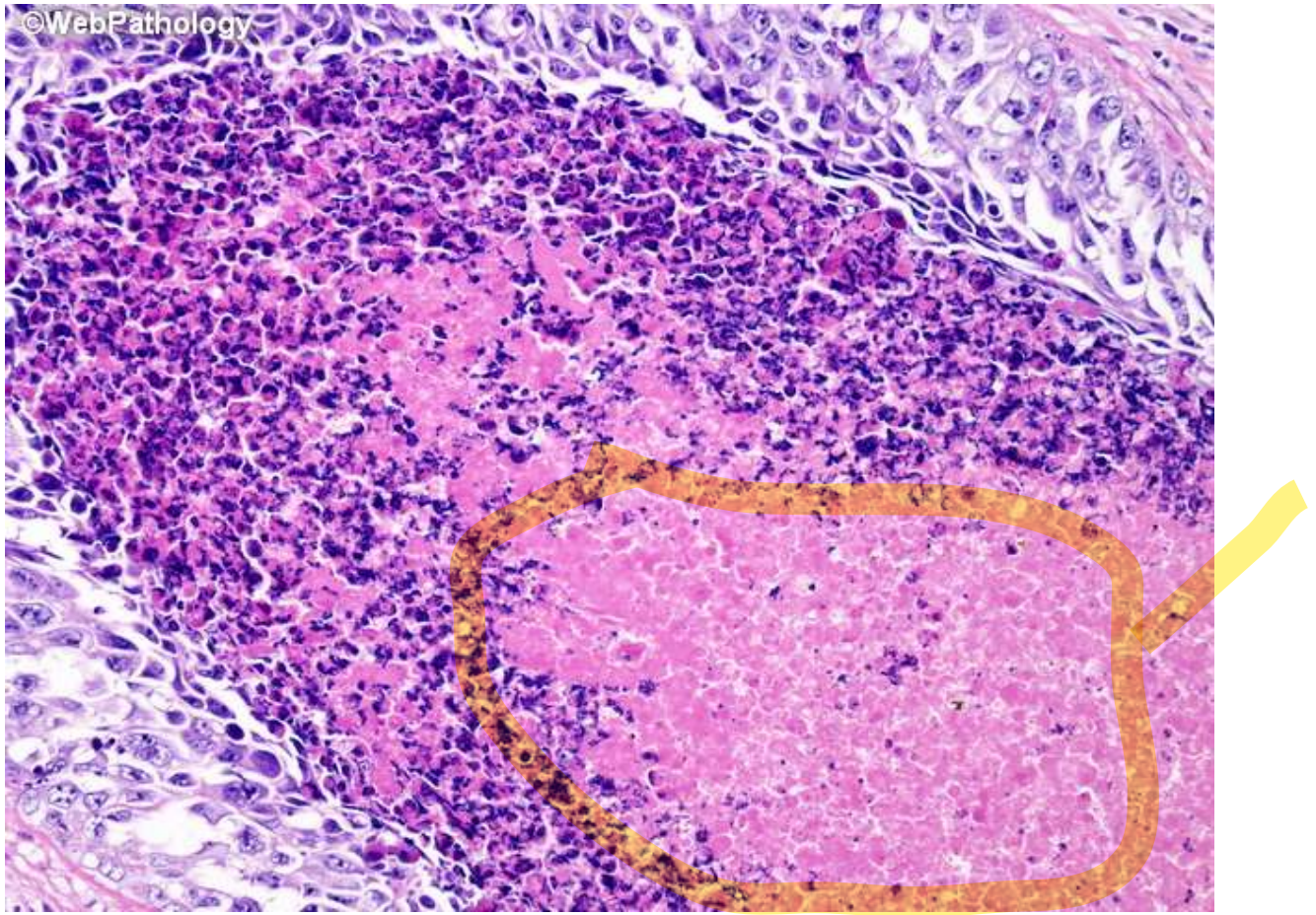
I. Grossly:

undefined whitish area that differs from the surrounding lung and it is inhomogeneous and rough

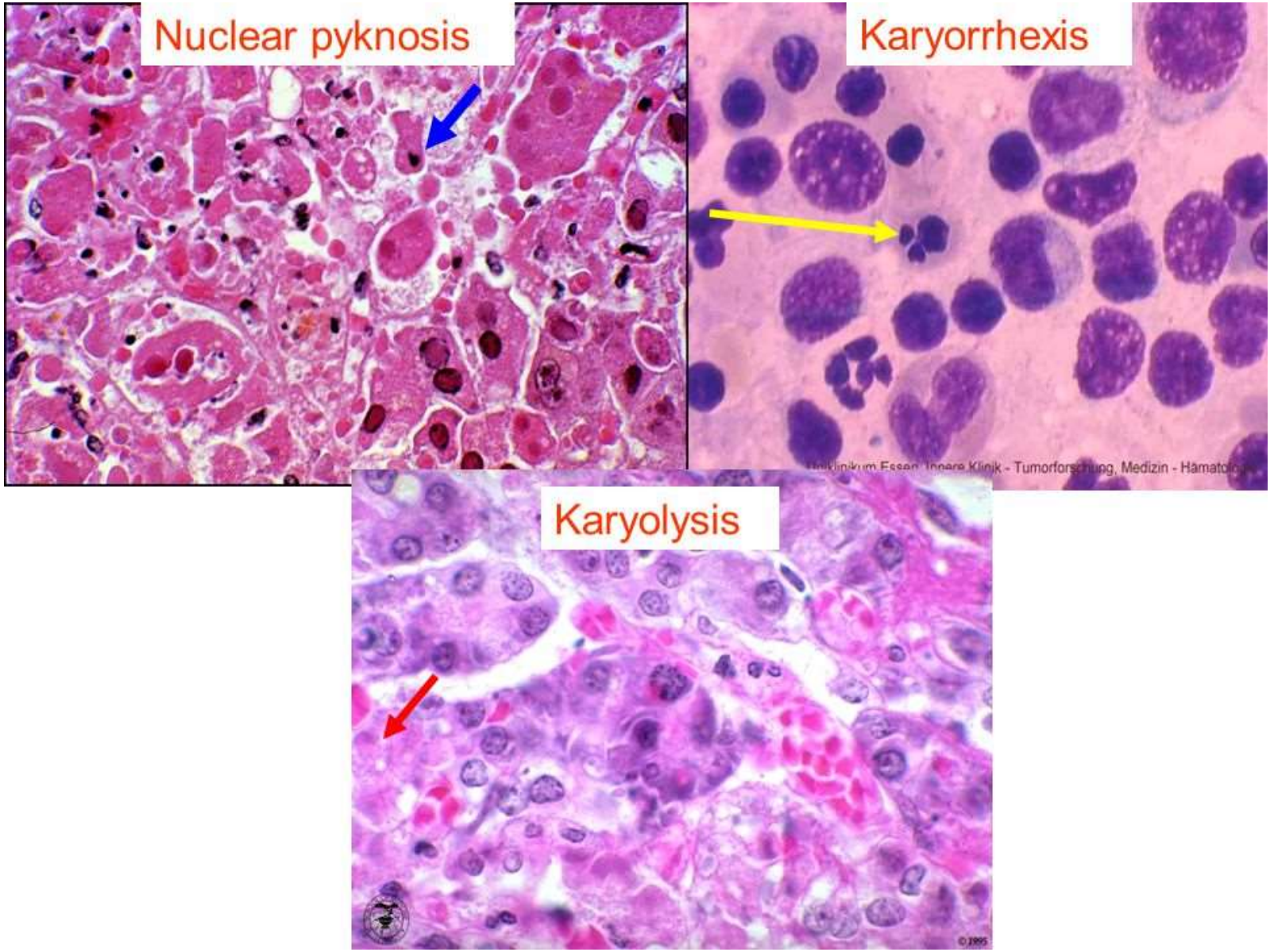


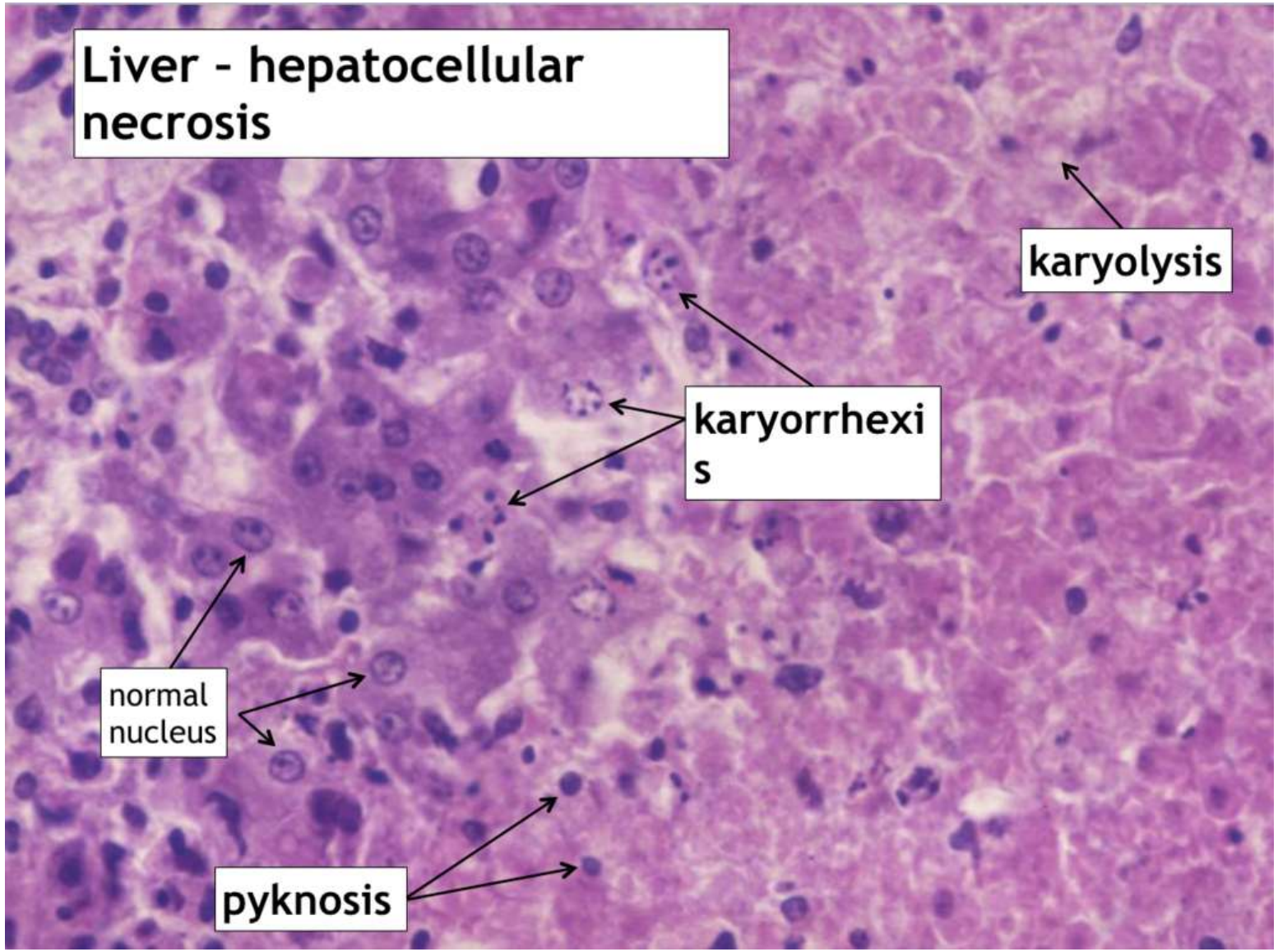
Microscopic appearance of Necrotic dead cells:

changes affecting cytoplasm:
1-pink in color eosinophilic due to loss of cytoplasmic Rna, increased binding of eosin to denatured cytoplasmic proteins and loss of glycogen particles.



homogenous appearance

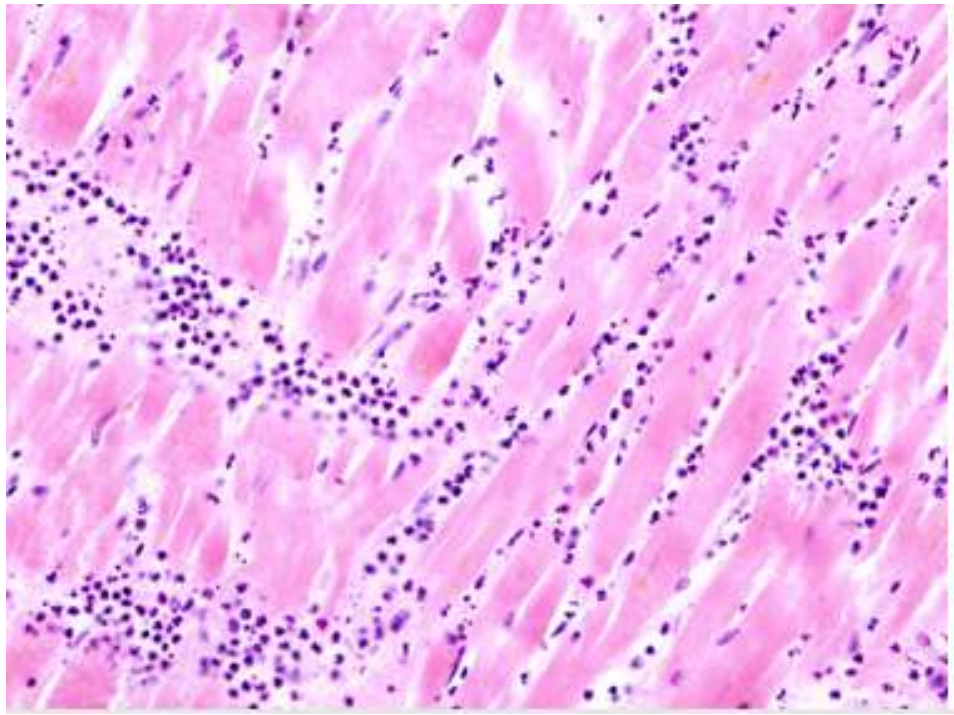

Nuclear pyknosis
Karyorrhexis
Karyolysis

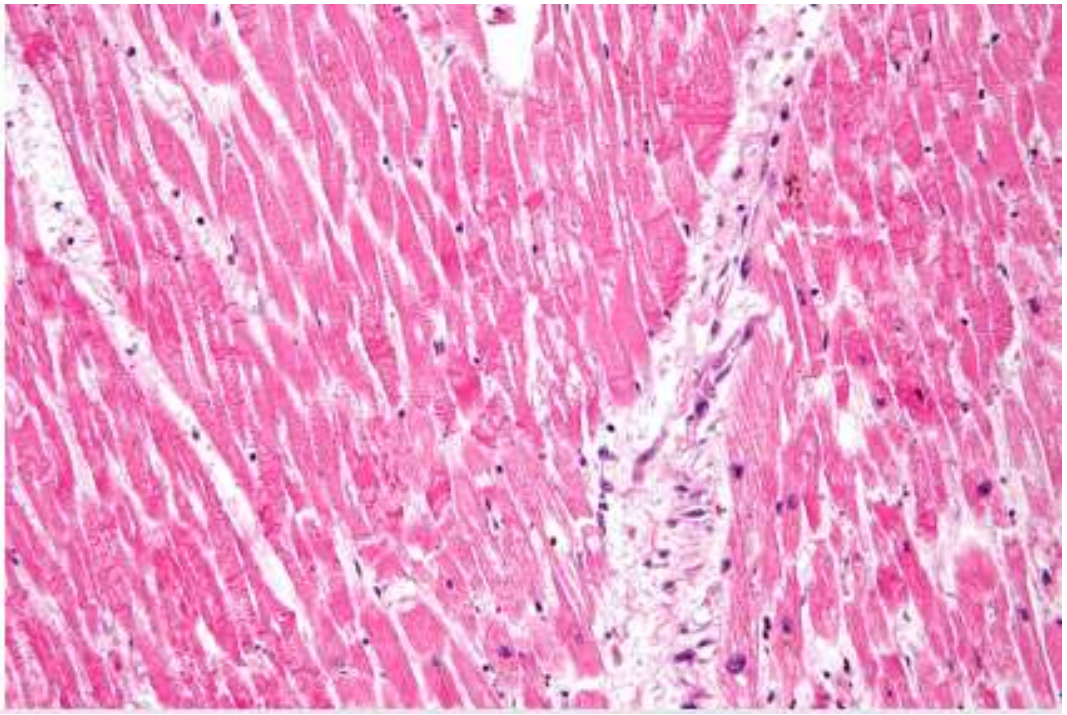


decrease basophilia in karyolysis

coagulative necrosis n the myocardium after infarction

hypoxia or ischemia



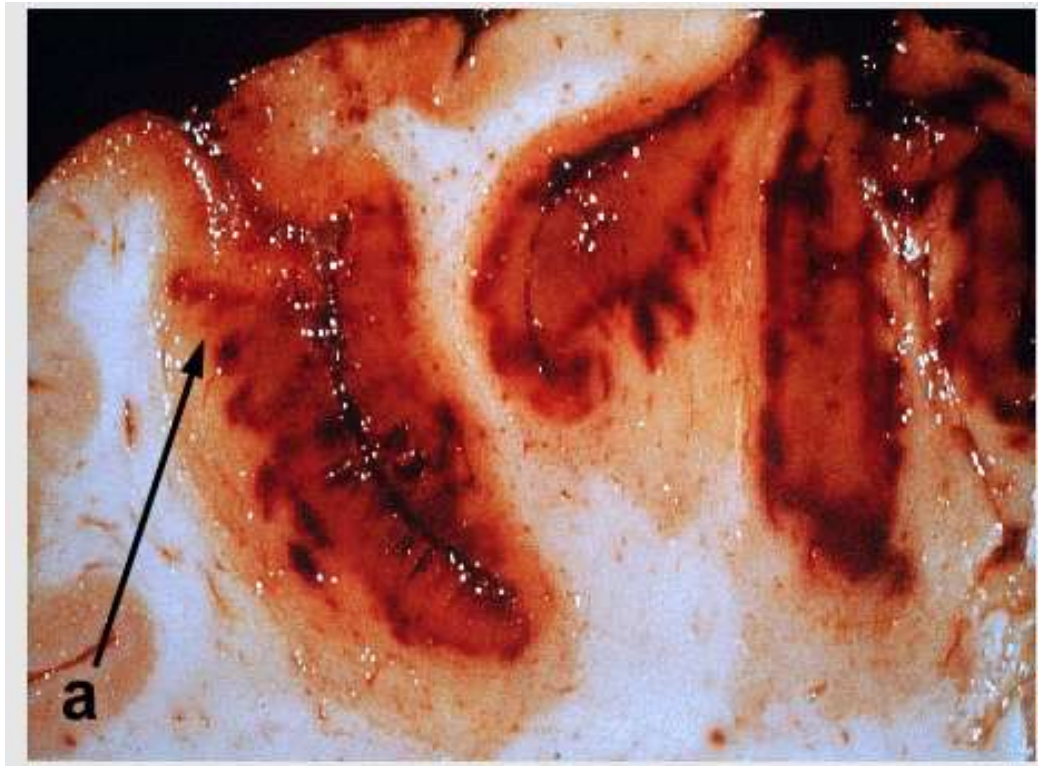


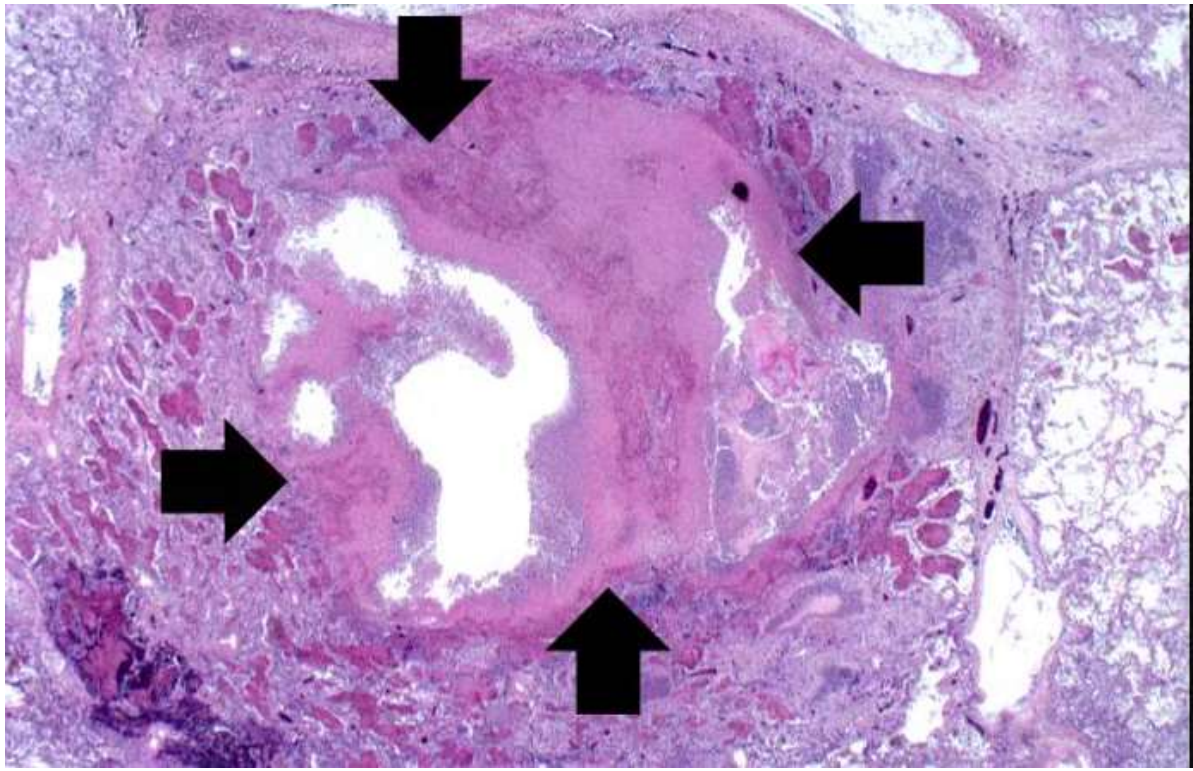
ghost cells looks like normal but more eosinophilic and nonviable (dead) anucleus

Liquefactive necrosis

seen in brain and other fatty tissue due to presence of lipid rich tissue

white and wet appearance due to accumulation of neutrophils that degrade lipid tissue

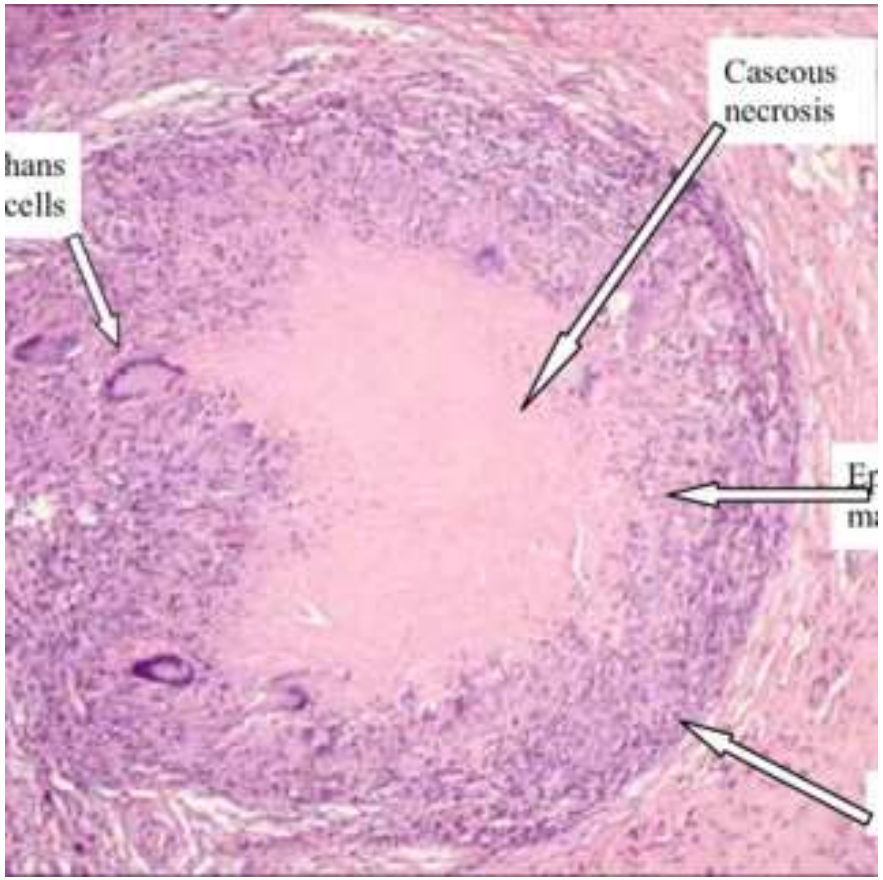




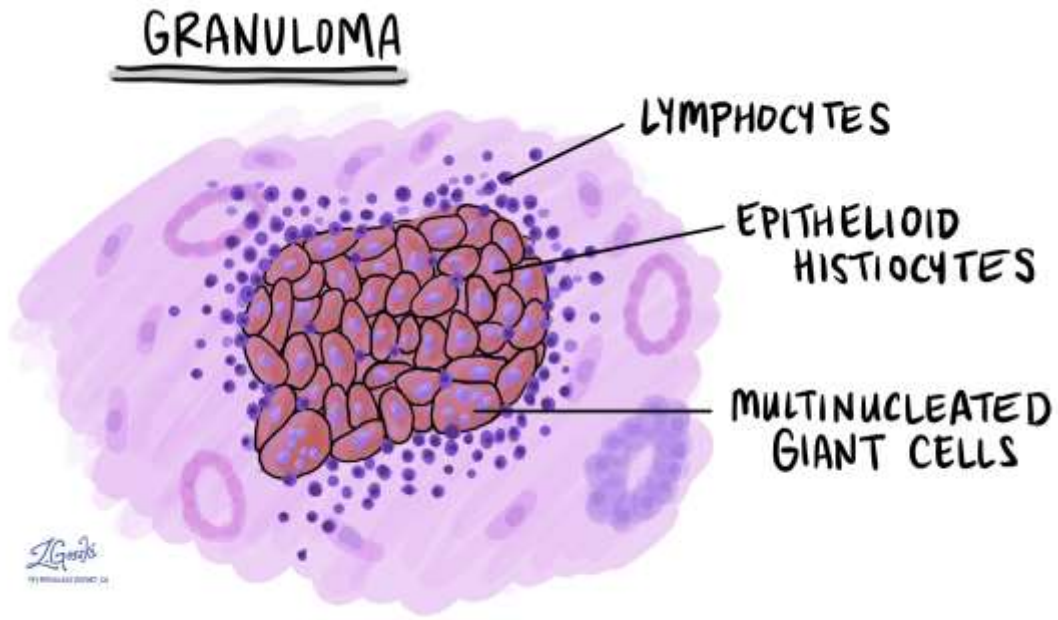
Caseous necrosis

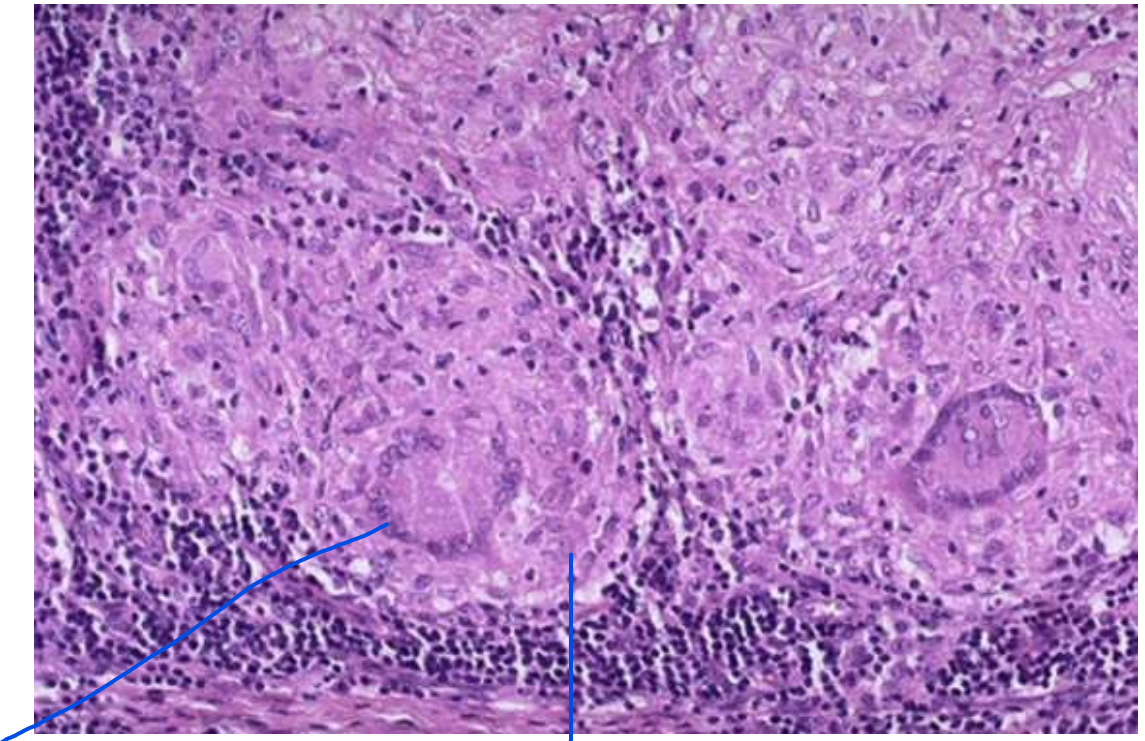
Chessy like appearence

caseating granuloma

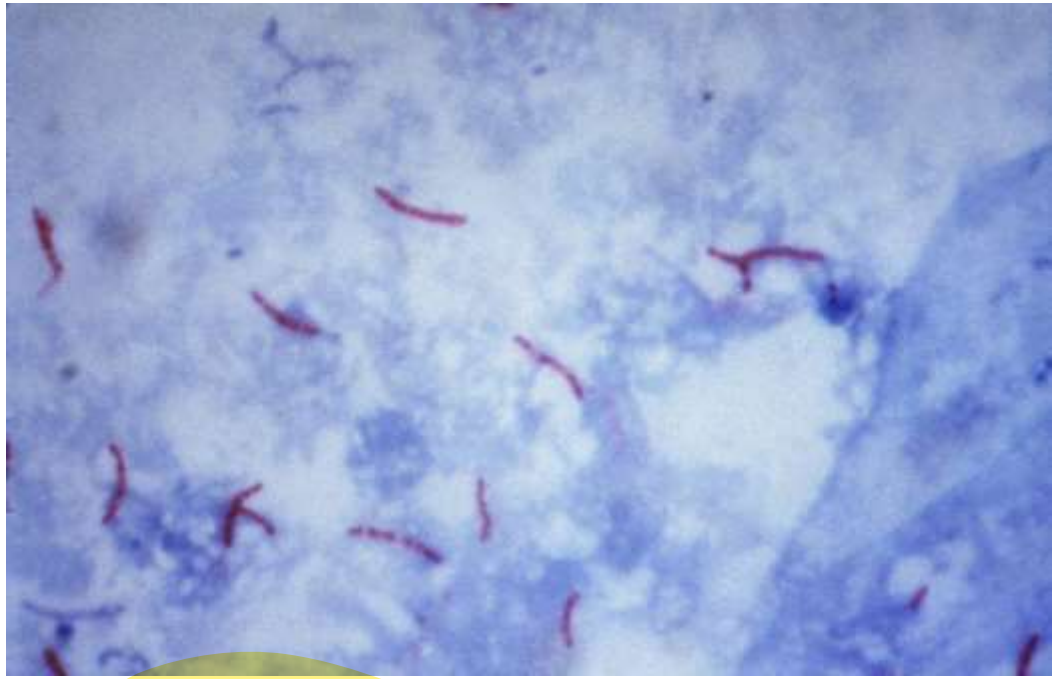


Granuloma structure





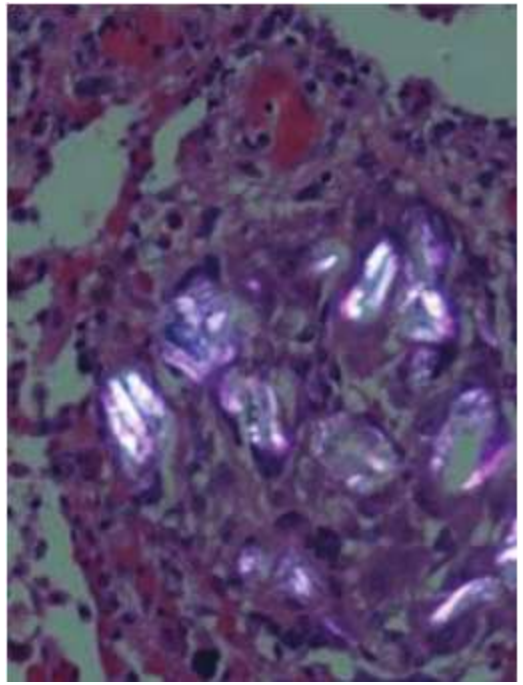
main cause of granuloma

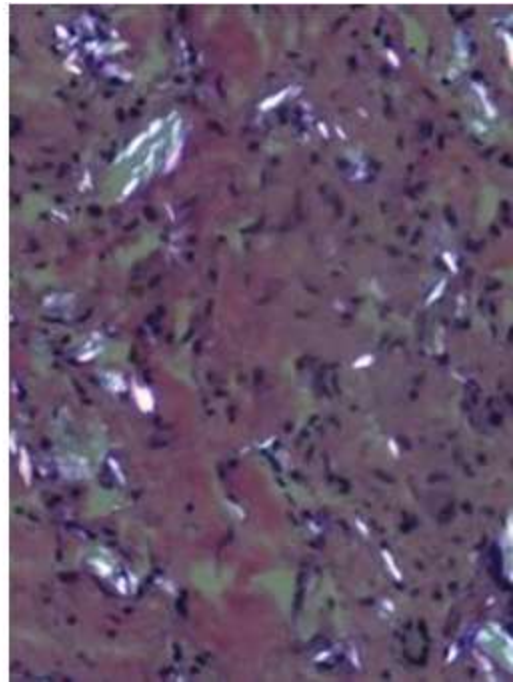


Ziehl neelsen stain

M.tuberculosis

viewed by polarized microscope





foreign bodies

Fat necrosis

seen in pancreas
acute pancreatitis (acinar cells die leads to lipase release)
trauma to fatty tissue

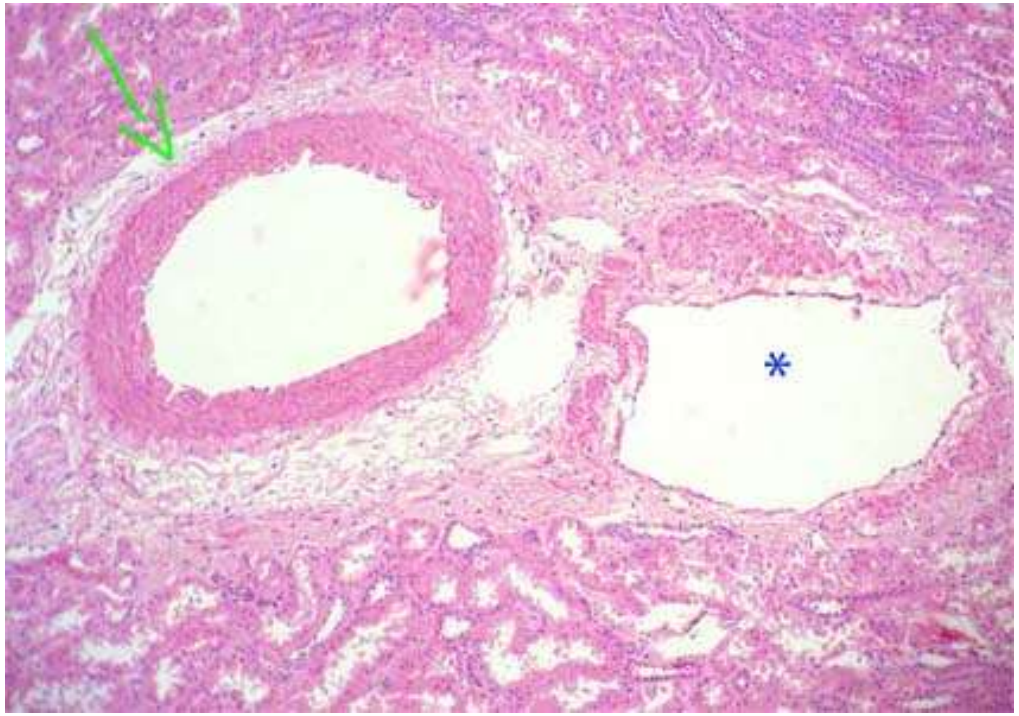
* fatty acids bind and precipitate calcium ions, forming insoluble salts.

chalky deposits!

* foamy macrophages adjacent to adipose tissue

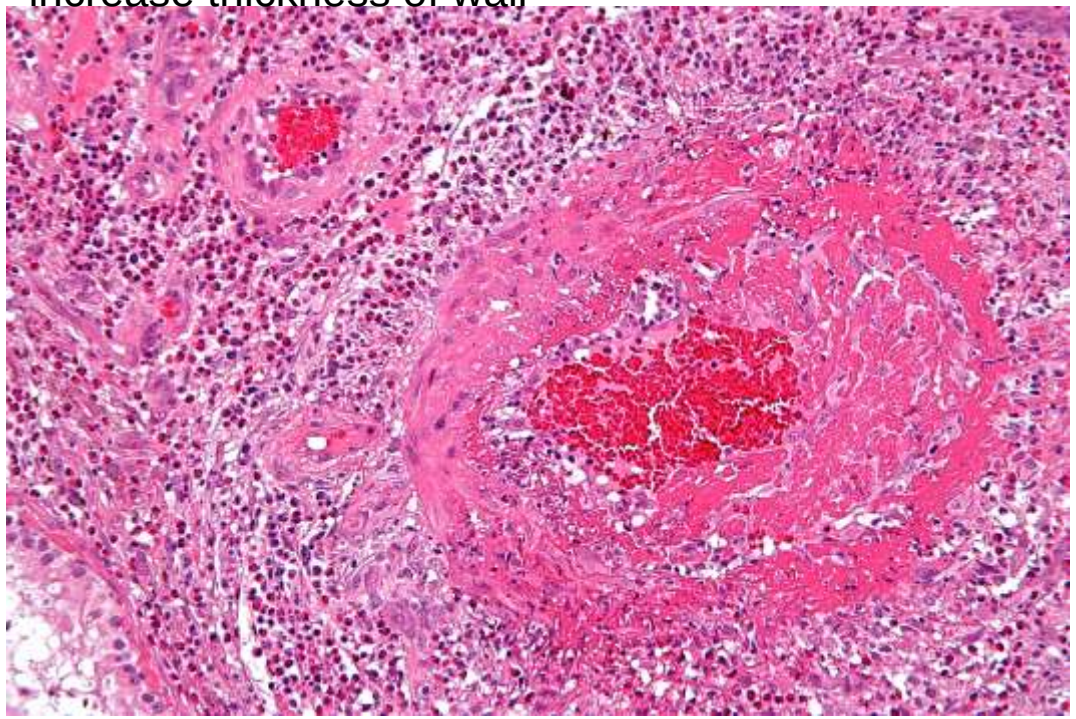
Fibrinoid necrosis

happens in severe hypertension



Normal B.V

Antigen antibody complex with fibrin (has pink color)
increase thickness of wall



amorphous (structureless appearance

Fibrinoid necrosis

Reactions of Blood Vessels in Acute Inflammation

- Vasodilation:

- induced by histamine, acting on vascular smooth muscle
- first involves the arterioles and then leads to the opening of new capillary beds in the area.
- The result is increased blood flow, which is the cause of heat and redness (erythema) at the site of inflammation.

- Edema
- Edema denotes an excess of fluid in the interstitial tissue or serous cavities.

k

either from exudate or transudate
1-exudate
happens from increased vascular permeability
2-transudate
happens due to increase in hydrostatic pressure or decrease in osmotic pressure

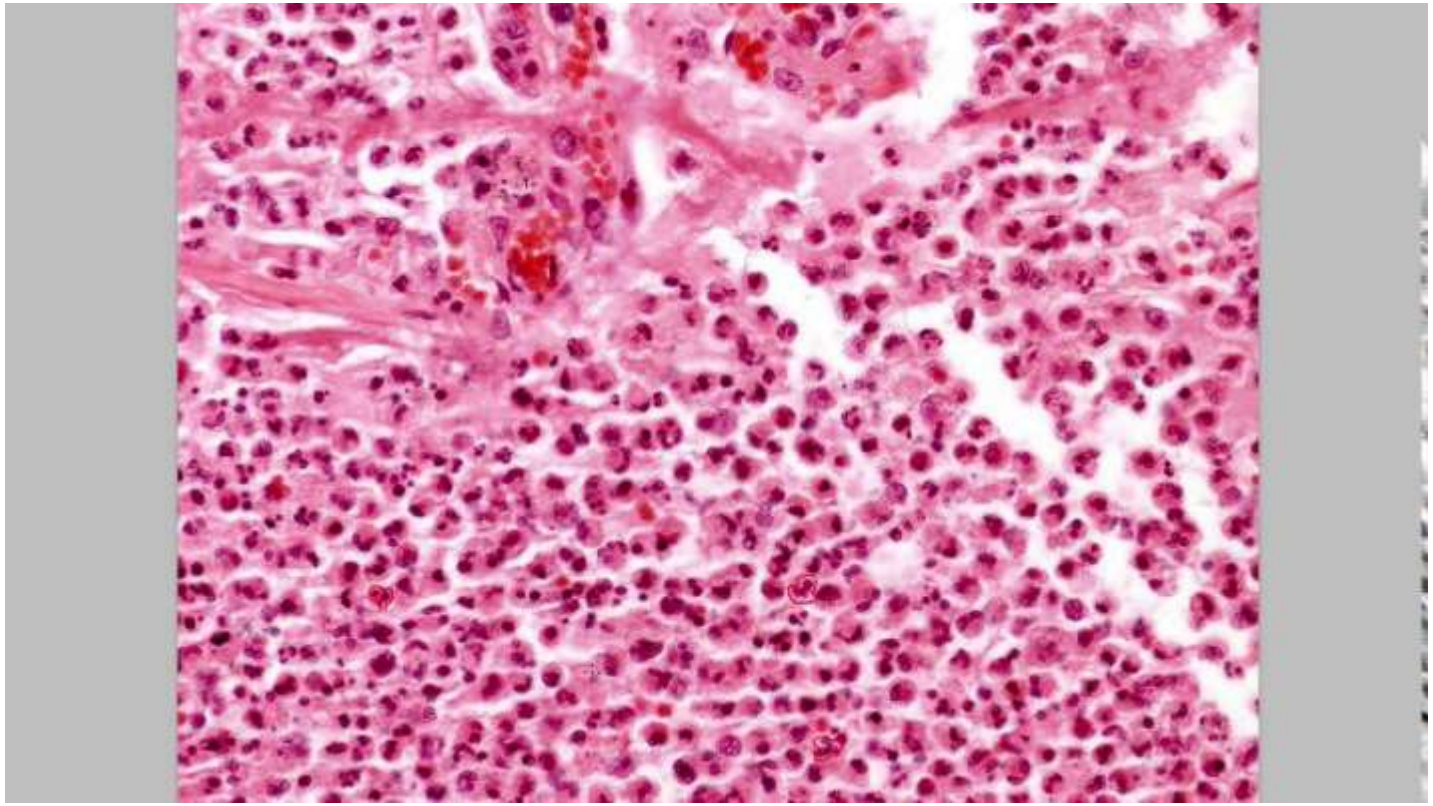
Lymphangitis and lymphadenitis.

- This streaking follows the course of the lymphatic channels and indicates the presence of lymphangitis

- painful enlargement of the draining lymph nodes, indicating lymphadenitis.

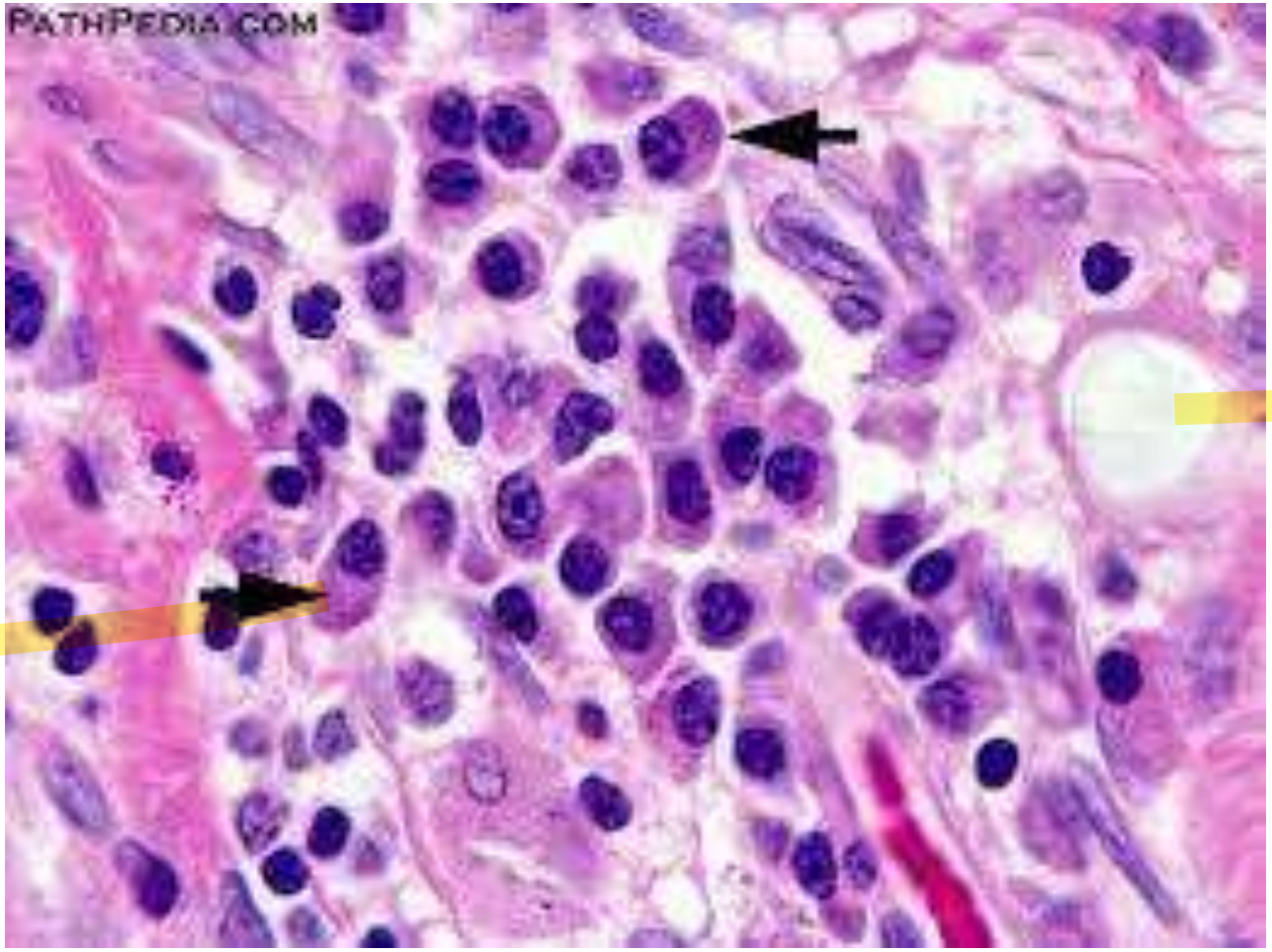
Due to inflammation lymphatics try to drain the excess edema fluid which contains microbes or leukocytes and cell debris that cause inflammation in lymph vessels or lymph nodes

Acute inflammation



dominant cells are neutrophils

Chronic inflammation



dominant cells are macrophages , plasma , lymphocytes

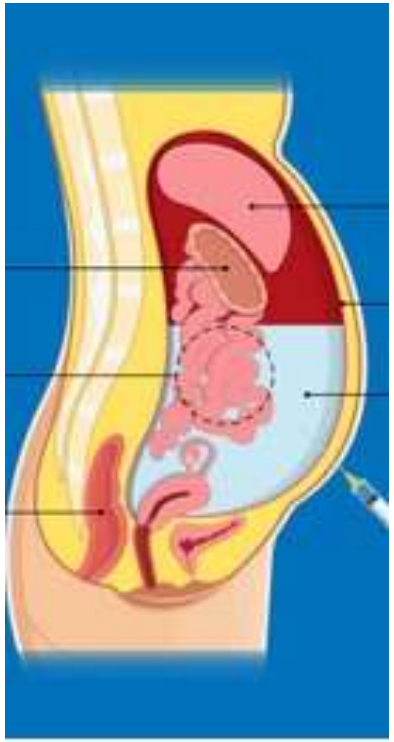
B cells

cachexia

- Pathologic state characterized by weight loss, muscle atrophy, and anorexia that accompanies some chronic infections and cancers. Explained by sustained production of TNF. TNF controls metabolism of protein tissue or body fluids

❖Peritoneal effusion an example of serous inflammation

Exudation of cell poor fluid into body cavities
Doesn't contain destructive organism nor large number of leukocytes



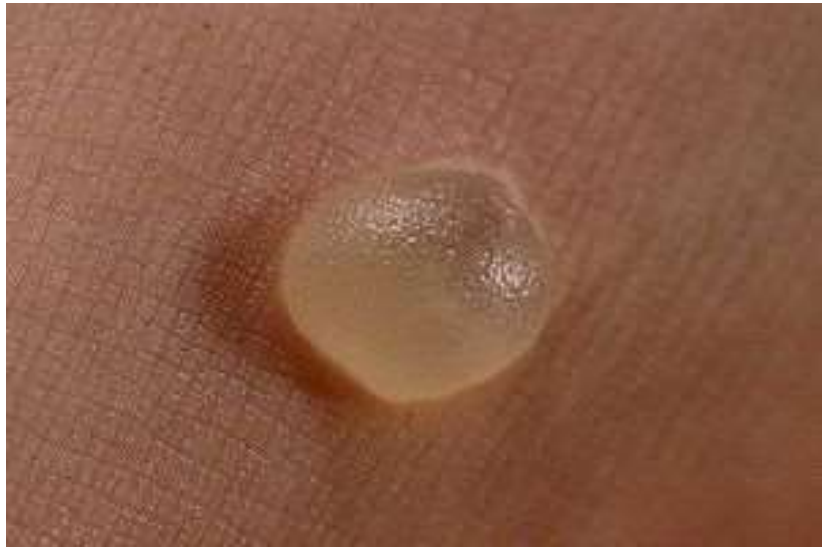
doesn't contain high weight protein molecule like fibrionous

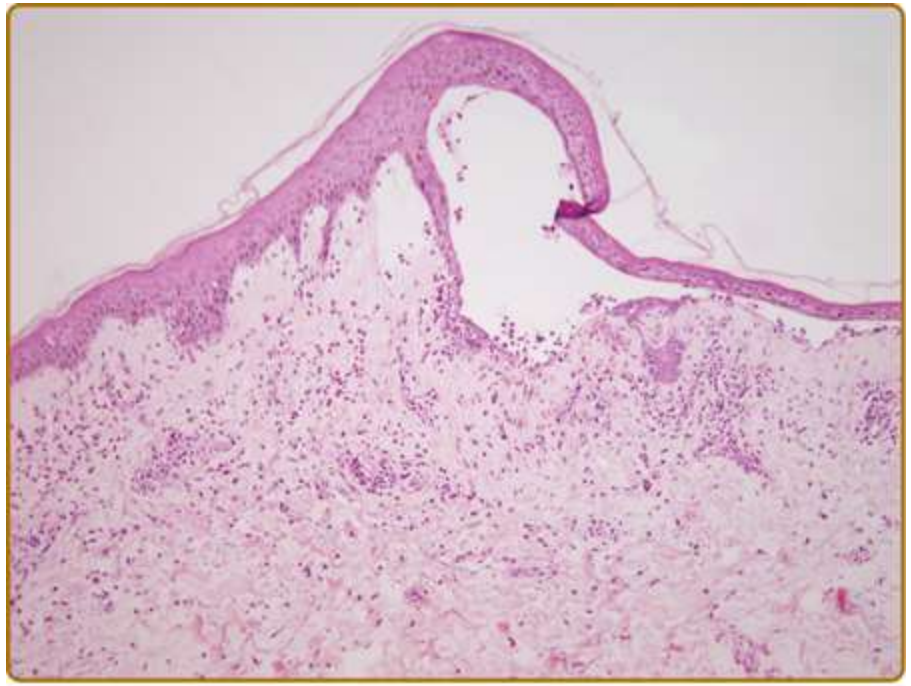
in peritoneal cavity it is called ascites

❖skin blister

Serous

- Resulting from a burn or viral infection.
- Represents accumulation of serous fluid within or immediately beneath the damaged epidermis of the skin





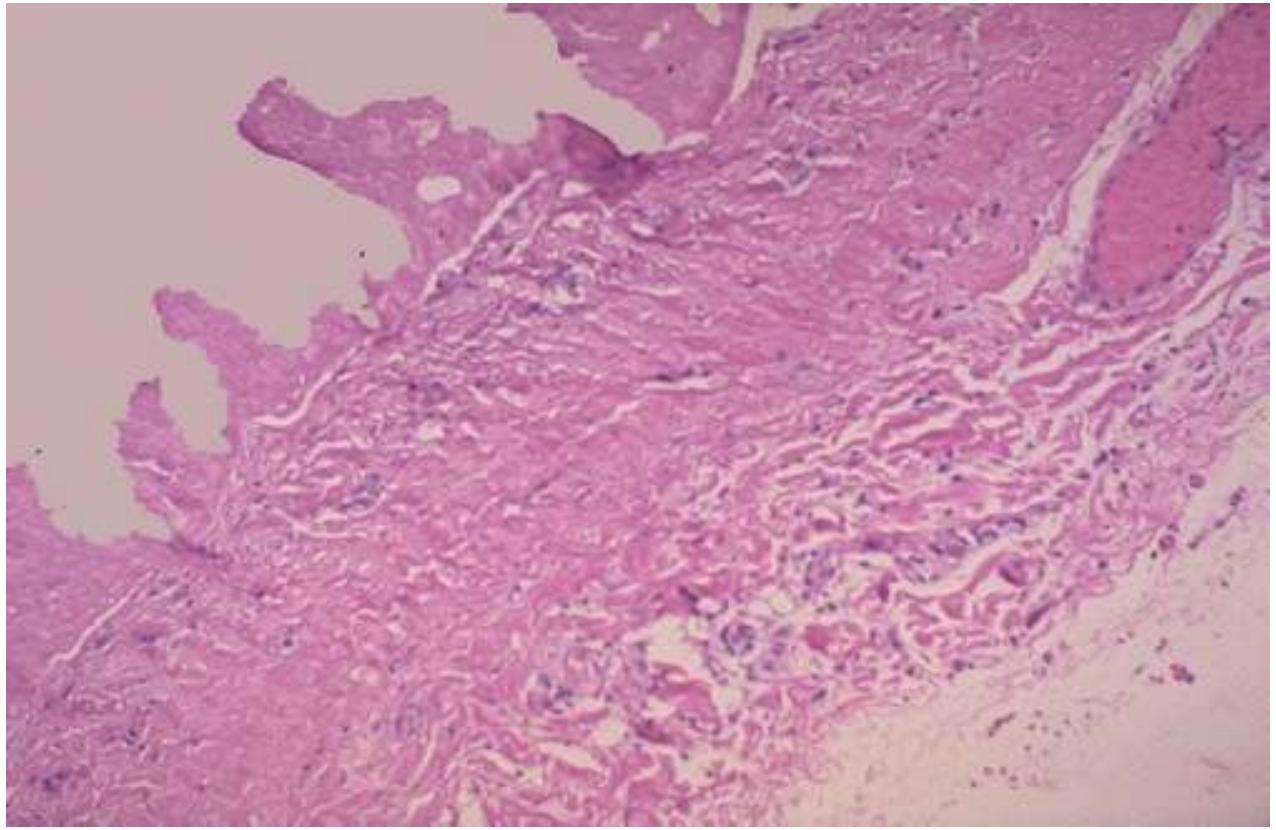
❖Fibrinous inflammation: Grossly

perforation of high molecular weight molecule like fibrinogen which transform to fibrin

Normally, the visceral **pericardium** is **translucent**

The **pericardial surface** is **dry** with a **coarse granular appearance** caused by **fibrinous exudate**

abnormal



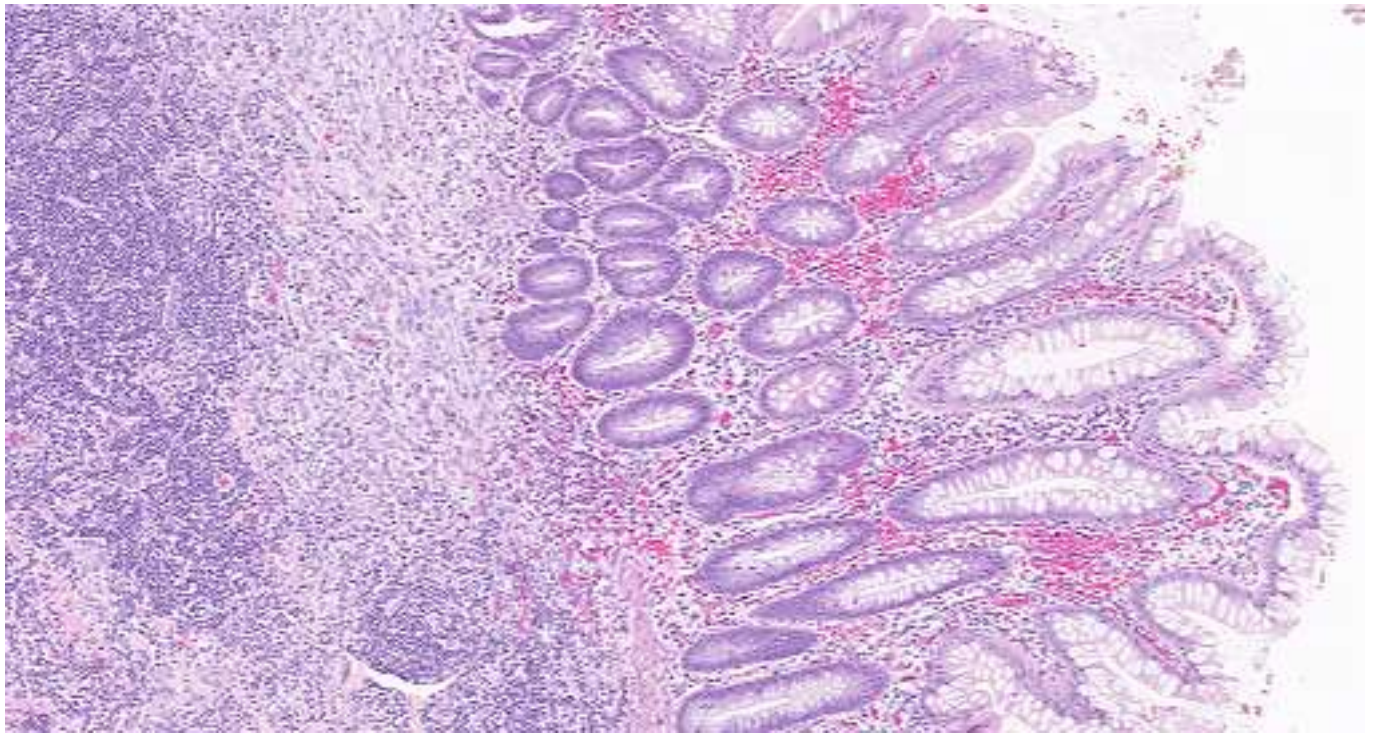
the pericardial surface here shows strands of pink fibrin extending outward. There is underlying inflammation.
fibrin appears as an eosinophilic meshwork of threads

A common example of an acute suppurative inflammation is acute appendicitis

purulent contains pus

normal

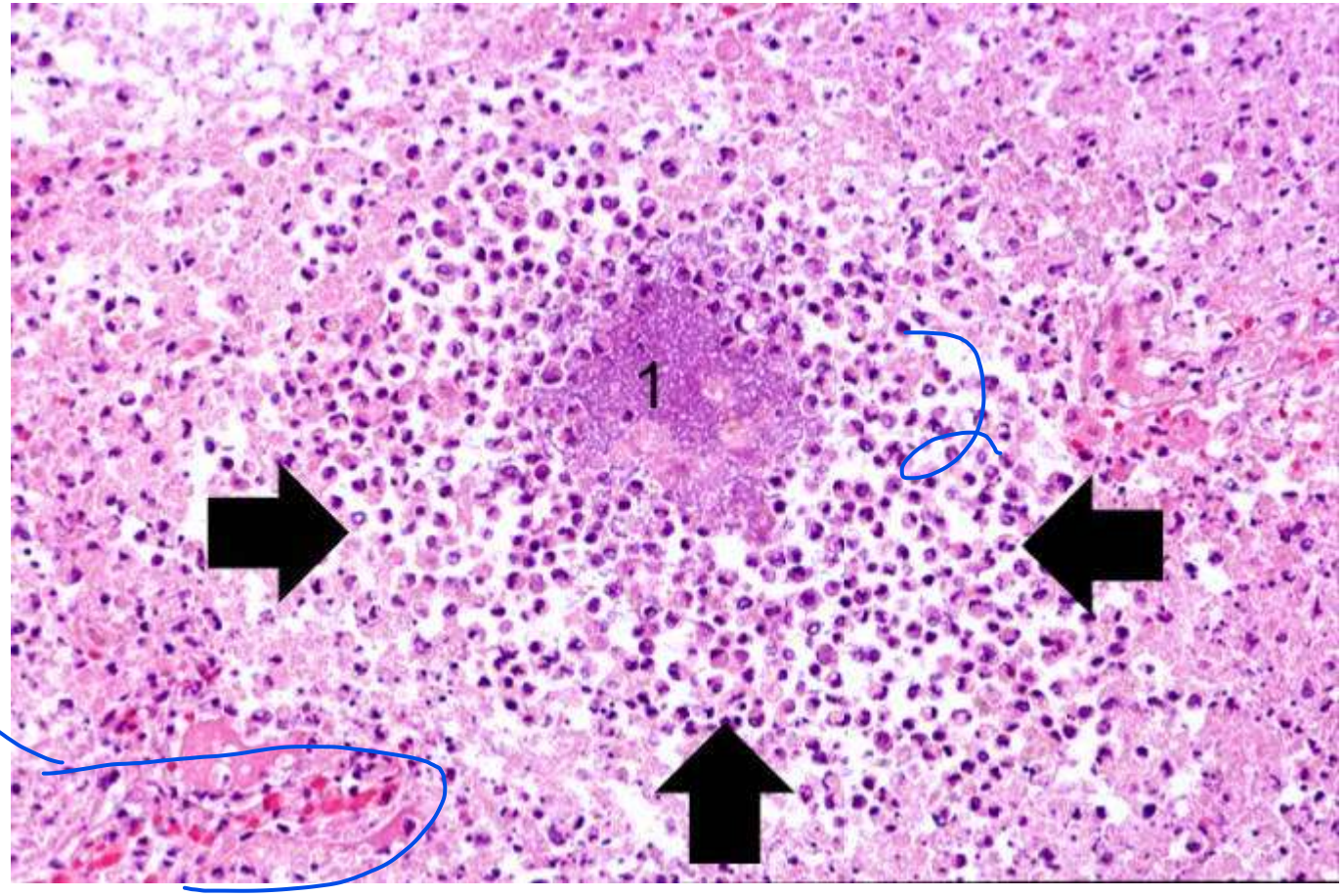
Acute appendicitis



Acute inflammation with predominance of neutrophils; involves some or all layers of the appendiceal wall.

- Abscesses:
- Localized collections of pus caused by suppuration buried in a tissue, an organ, or a confined space.
- They are produced by seeding of pyogenic bacteria into a tissue . In time the abscess may become walled off and ultimately replaced by connective tissue

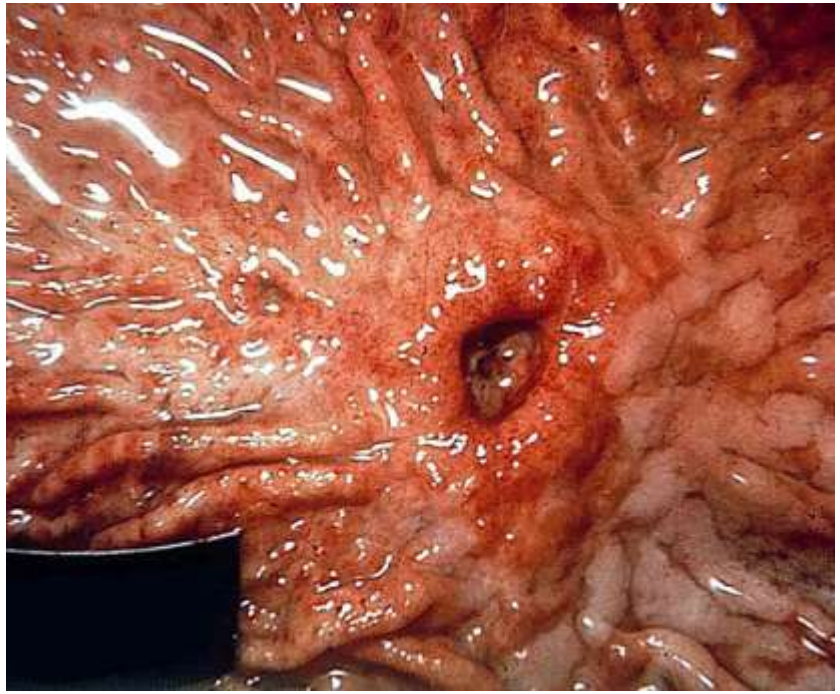
Abscesses have multiple areas:
* central region with necrotic leukocytes and tissue cells.
 * zone of preserved neutrophils around this necrotic focus.
 *vascular dilation, parenchymal and fibroblastic proliferation.



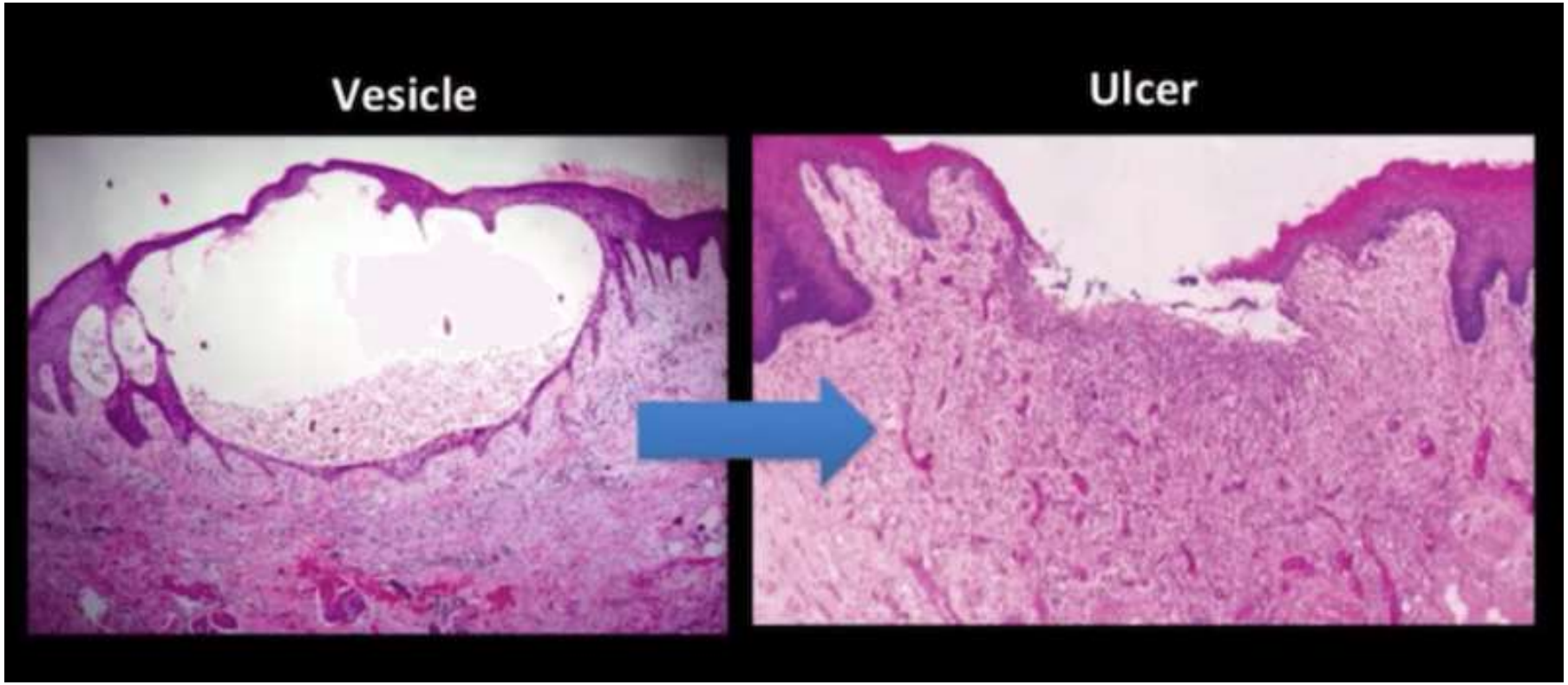
ulcer

Shedding of inflamed necrotic tissue

- It is most commonly encountered in:
- (1) the mucosa of the mouth, stomach, intestines, or genitourinary tract.
- (2) the skin and subcutaneous tissue of the lower extremities in older persons



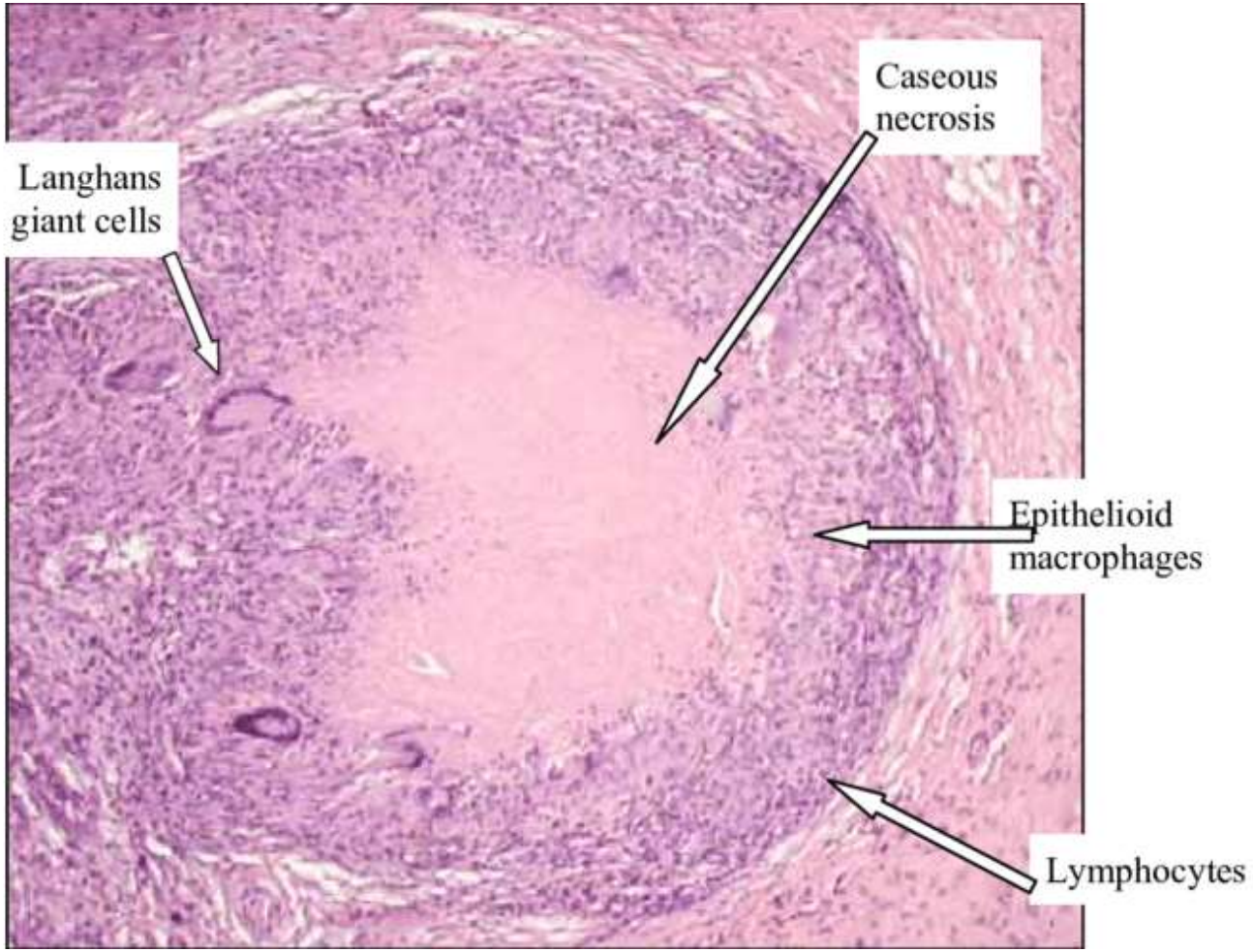
histology



sloughing (shedding) of inflamed necrotic tissue

Microscopic features of Ulcers

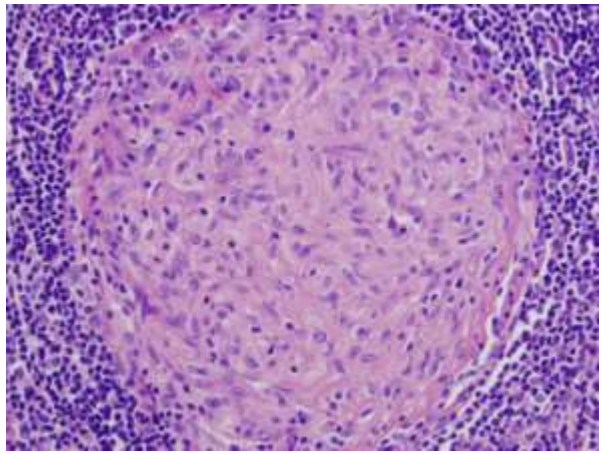
Acute stage:
Intense polymorphonuclear infiltration and vascular dilation in the margins of the defect.
With chronicity:
the margins and base of the ulcer develop fibroblast proliferation, scarring, and the accumulation of lymphocytes, macrophages, and plasma cells.



in TB

What? Where??

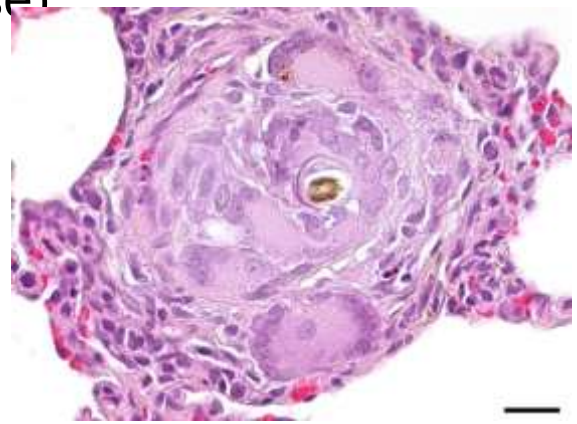
Types of granulomas:

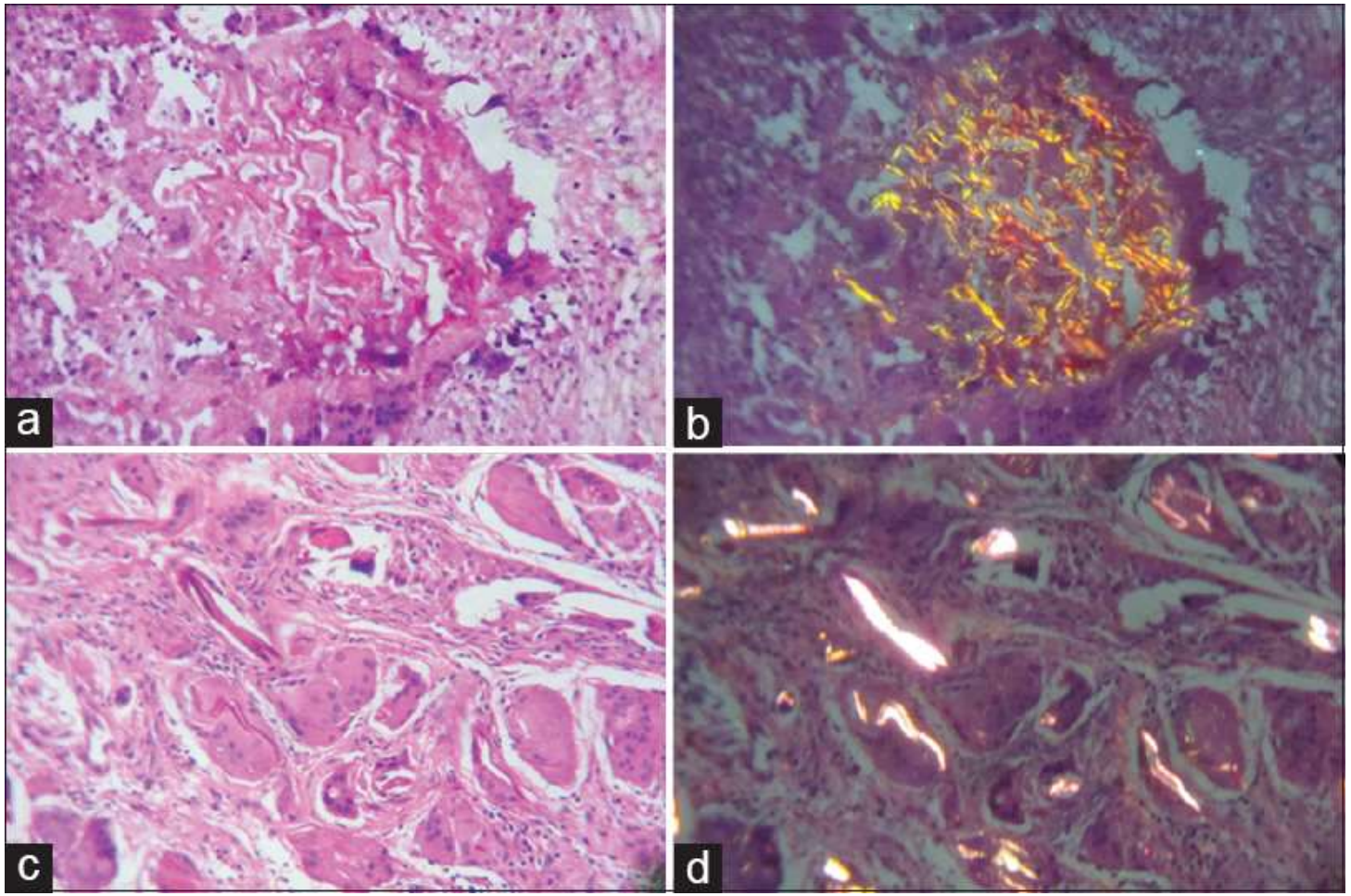


- 1.Immune granulomas:
- caused by persistent T cell–mediated immune response.
- when the inciting agent cannot be readily eliminated.

- **2.Foreign body granulomas:**
- seen in response to inert foreign bodies, in the absence of T cell– mediated immune responses.
- May form around materials such as talc (associated with intravenous drug abuse), sutures, or other fibers

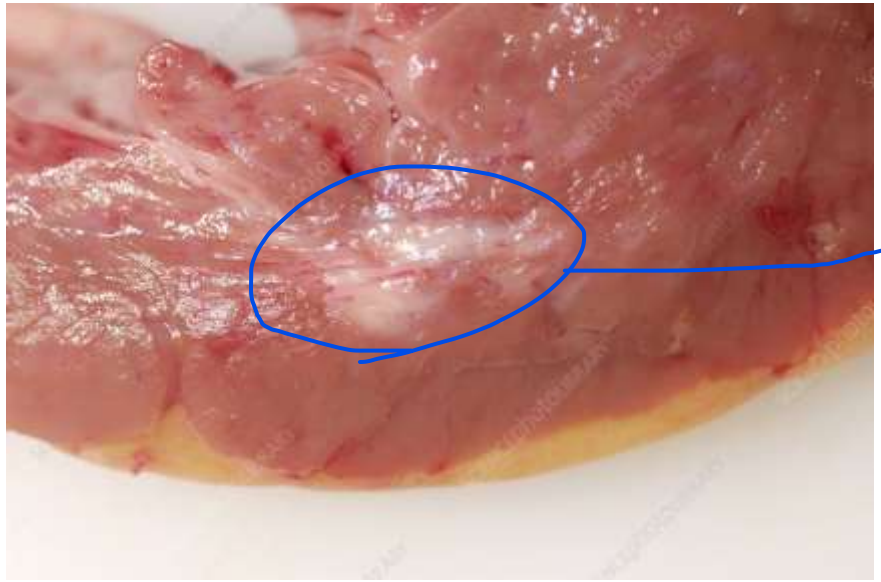
Talc is substance added to drugs to decrease the amount of active substance used





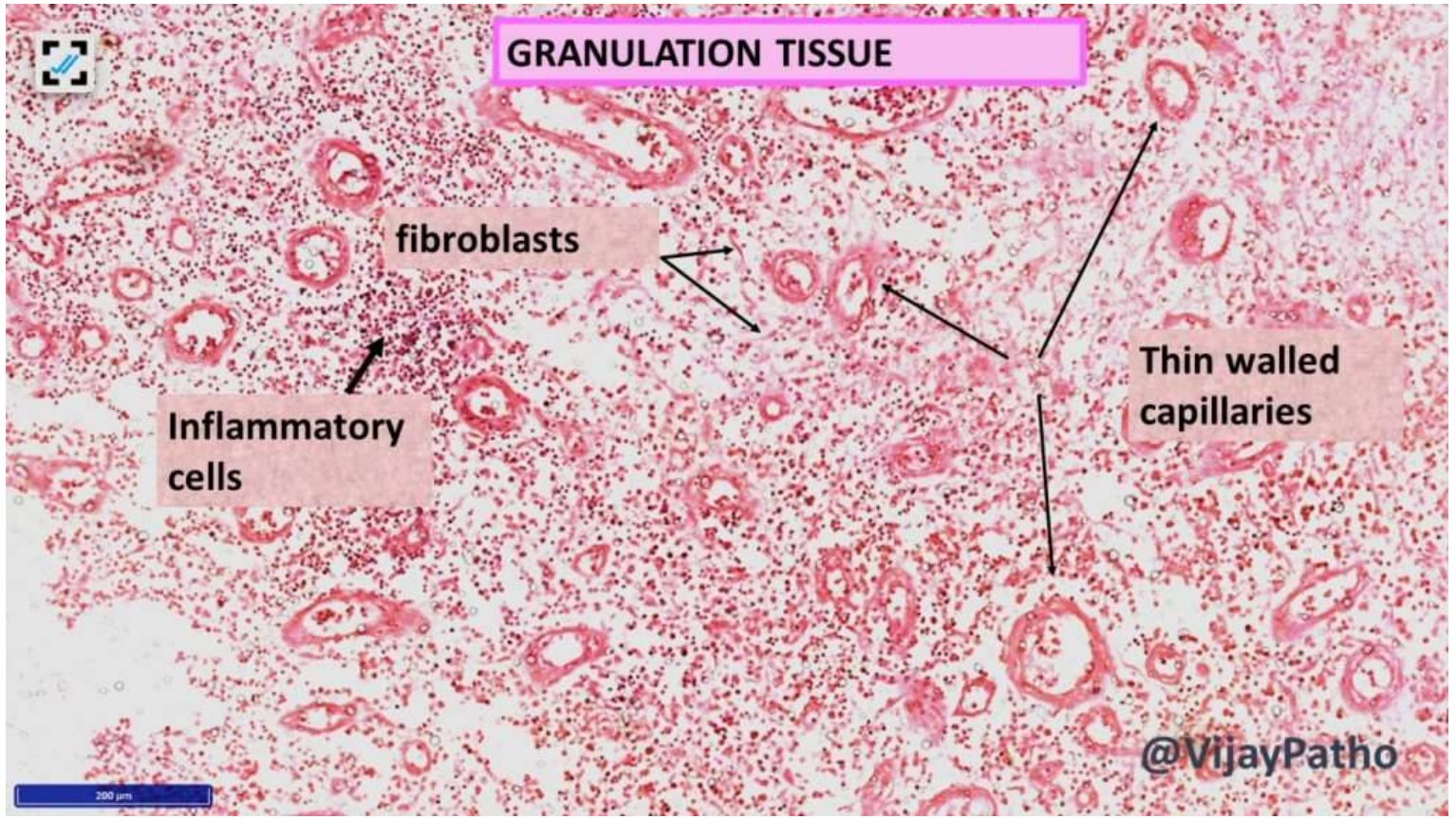
The foreign material can usually be identified in the center of the granuloma, particularly if viewed with polarized light, in which it may appear refractile.

The term scar is most used in connection to wound healing in the skin.



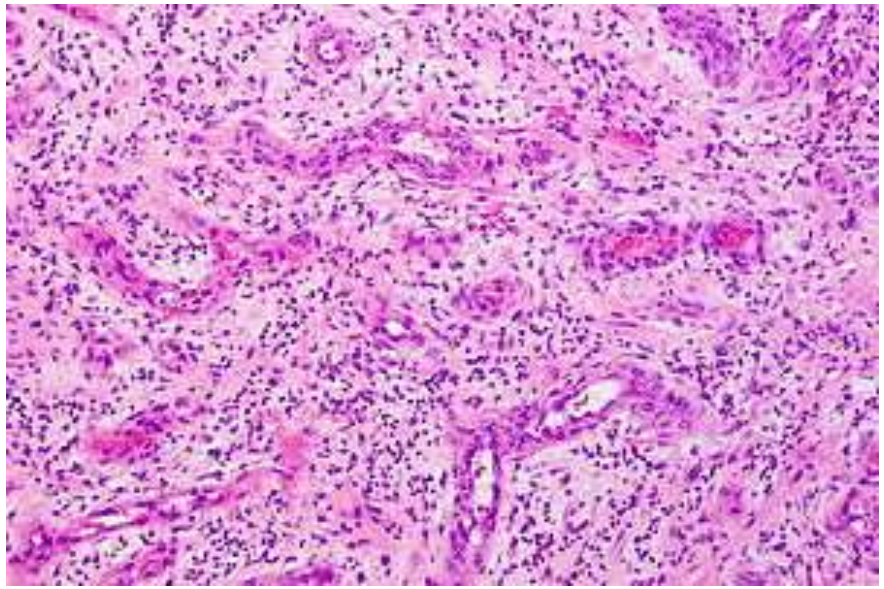
Replacement of parenchymal cells in any tissue by collagen, as in the heart after myocardial infarction.

- The combination of proliferating fibroblasts, loose connective tissue, new blood vessels and scattered chronic inflammatory cells, forms a **granulation tissue.**



Granulation tissue.

pink, soft, granular gross appearance, such as that seen beneath the scab of a skin wound.



proliferating fibroblasts, loose connective tissue, new blood vessels and scattered chronic inflammatory cells

- 1.Venous leg ulcers:

- Seen in elderly people as a result of chronic venous hypertension, which may be caused by severe varicose veins or congestive heart failure.

- These ulcers fail to heal because of poor delivery of oxygen to the site of the ulcer.

2.Arterial ulcers:

- develop in individuals with atherosclerosis of peripheral arteries, especially associated with diabetes.

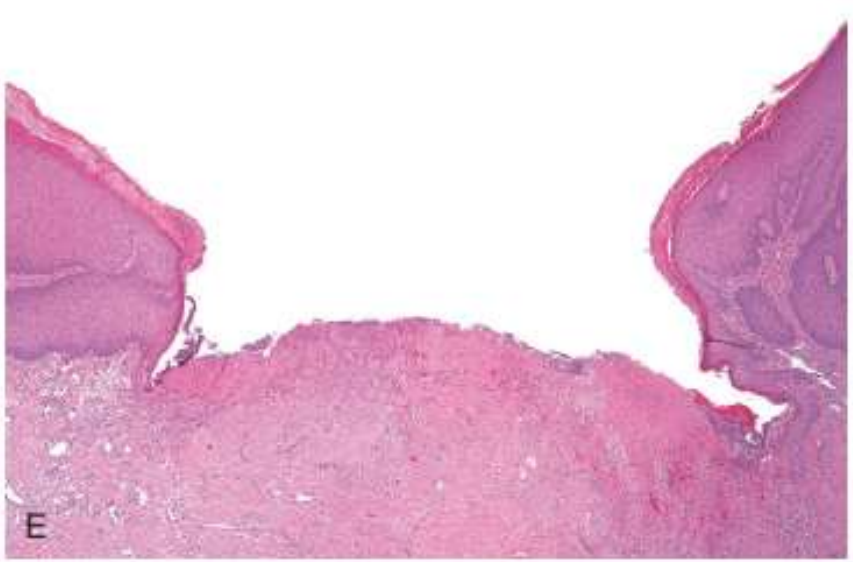
3. Pressure sores : in sacrum due to stasis in circulation that leads to ulcer (no repair due to low perfusion)

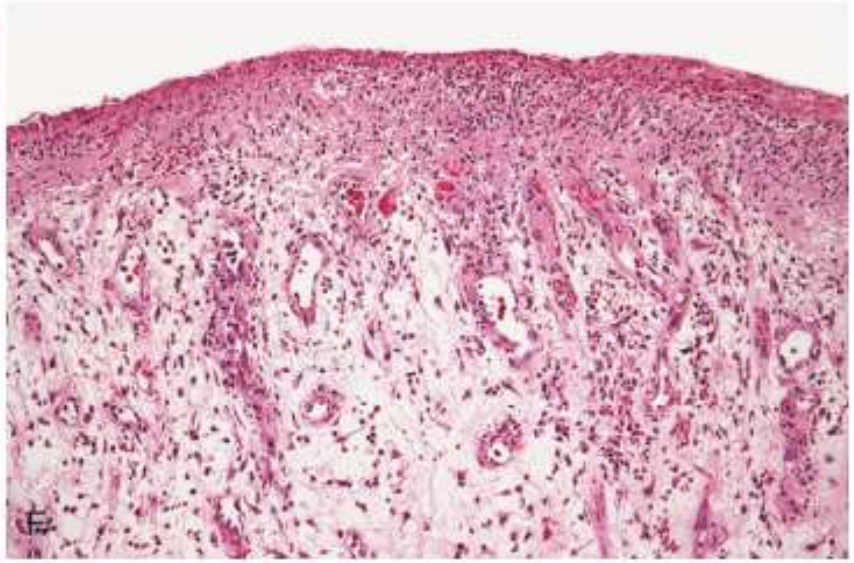
- are areas of skin ulceration and necrosis of underlying tissues.
- caused by prolonged compression of tissues against a bone, for example, in bedridden. The lesions are caused by mechanical pressure and local ischemia.

Bedridden: have to stay in bed due to injury

4.Diabetic ulcers;

- affect the lower extremities, particularly the feet. Tissue necrosis and failure to heal are the result of small vessel disease causing ischemia, neuropathy, systemic metabolic abnormalities, and secondary infections.





epithelial ulceration and extensive granulation tissue in the underlying dermis

5. wound rupture (dehiscence):

- occurs most frequently after abdominal surgery and is a result of increased abdominal pressure, such as may occur with vomiting or coughing. and constipation

they tend to regress over several month

✓hypertrophic scar.

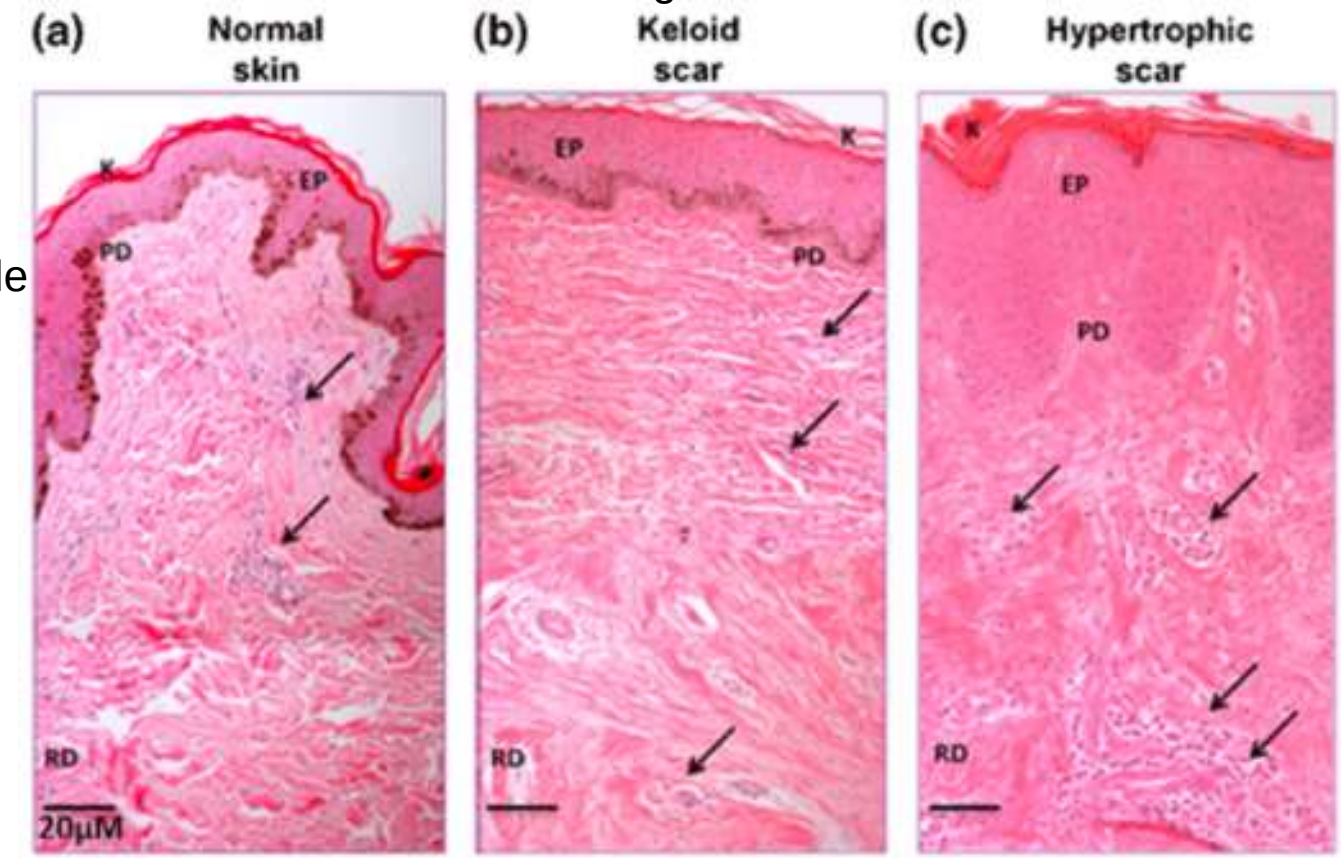
by myofibroblast due to excess contraction
but does not extend out of the boundaries of
the injury

- **keloid:**

- It is a hypertrophic scar that grows beyond the boundaries of the original wound and does not regress.

thick collagen bundles

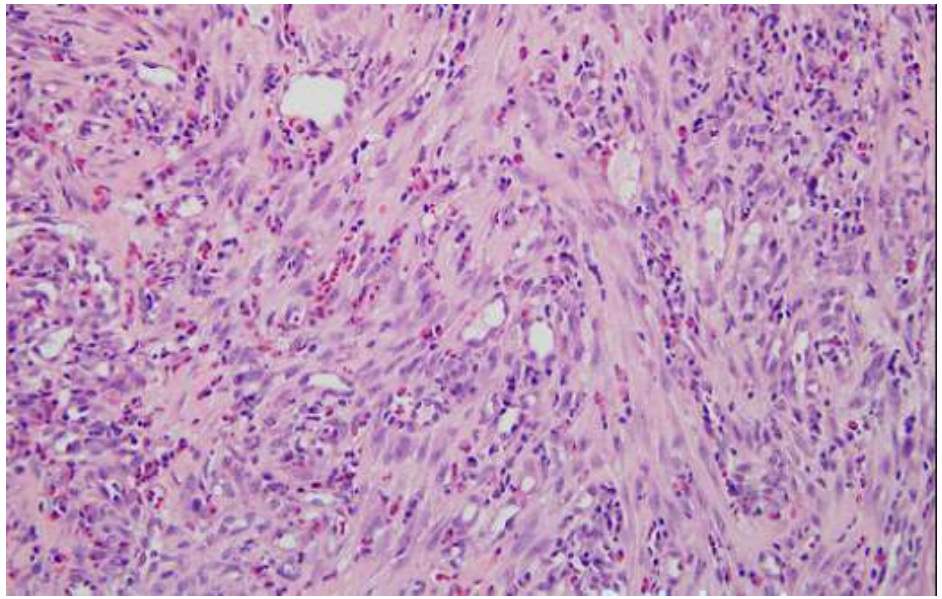
fine collagen bundle



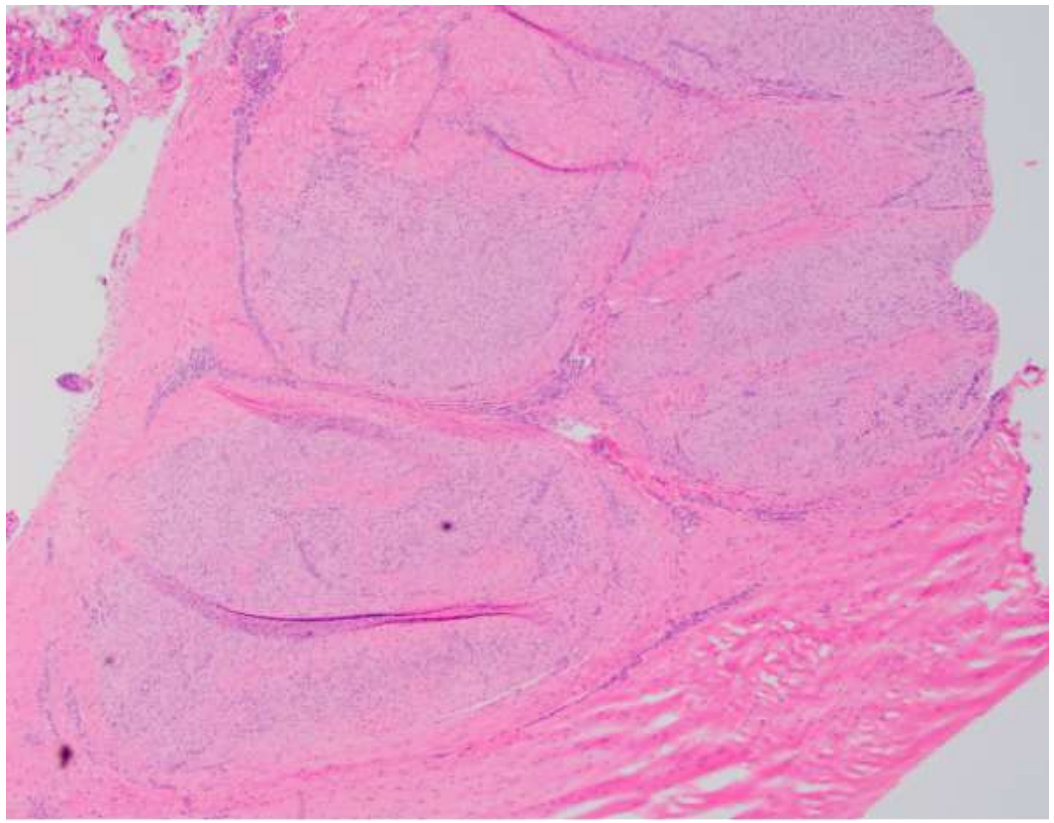
A. In normal skin, the characteristic random orientation and bundle formation of collagen fibres
B. increased number of thick collagen fibres arranged in bundles
C. The collagen fibres were arranged randomly and showed highly cellular zones

Exuberant granulation

- formation of excessive amounts of granulation tissue, which protrudes above the level of the surrounding skin and blocks reepithelialization .



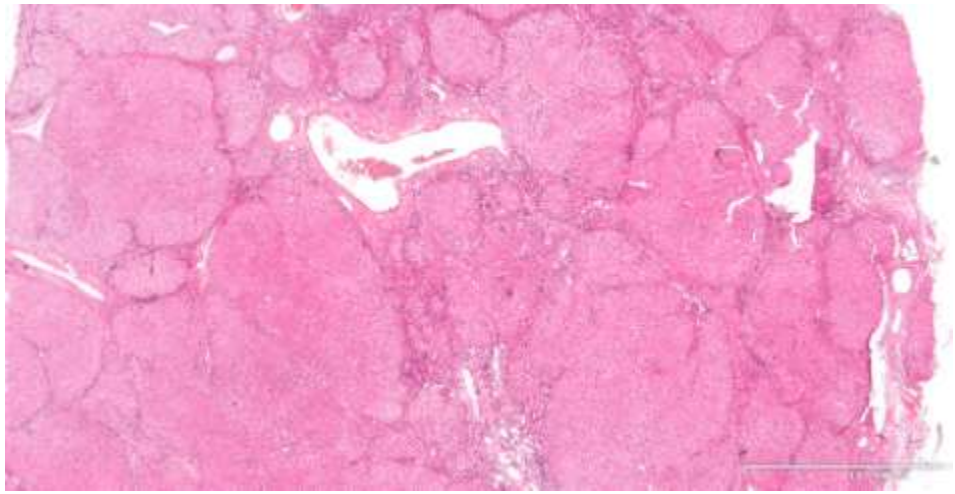
contracture



Nodule formation:
Composed of spindle cells (myofibroblasts and fibroblasts) with dense collagen. replacing inelastic tissue with elastic tissue

Examples of Fibrotic parenchymal disorders

- 1. liver cirrhosis. loss of hepatocytes



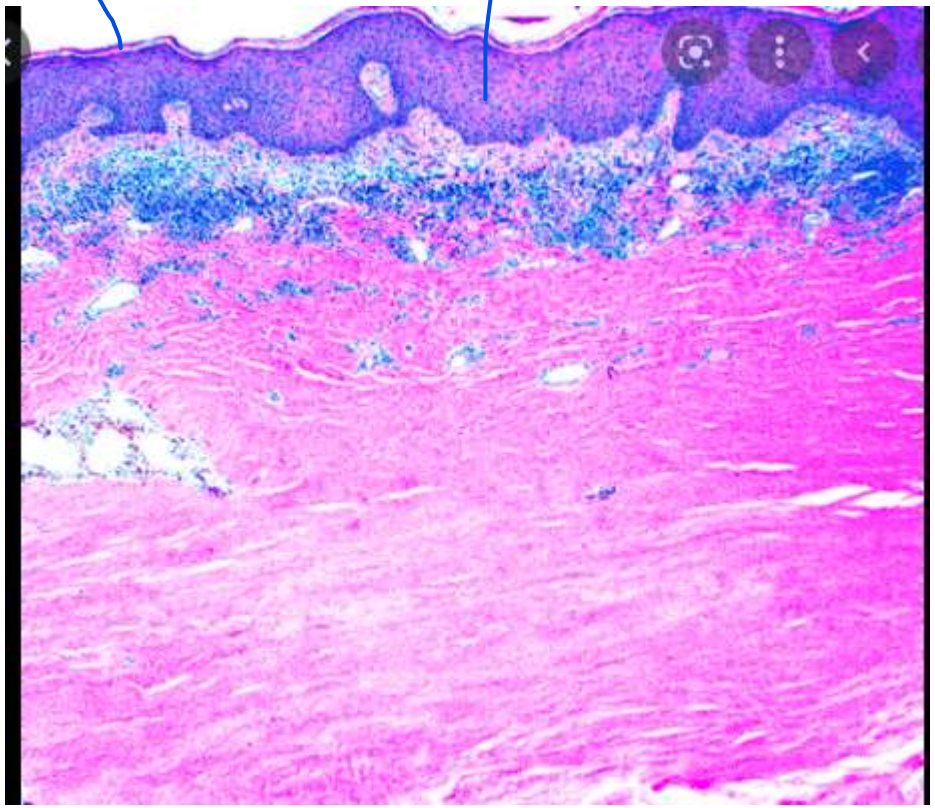
2.systemic sclerosis (scleroderma).

autoimmune disease characterised by overproduction of collagen

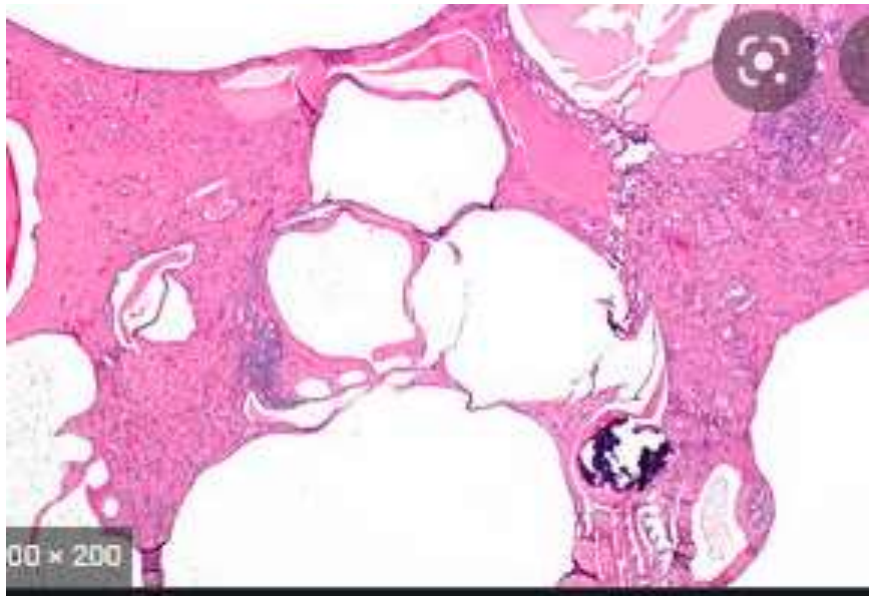
epidermis is lighter

loss of hair follicles

thickening of dermis doesn't contain elastic tissue



3. end-stage kidney disease.



❖fibrosing diseases of the lung.

Grossly: Honeycomb, Cystic spaces with fibrotic wall
Histology: cystic spaces lined by bronchiolar epithelium and fibrotic wall