MORPHOLOGIC PATTERNS OF ACUTE INFLAMMATION

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- Special morphologic patterns are often seen in addition to the general features which are characteristic of most acute inflammatory reactions, depending on :

- The severity of the reaction.
- Its specific cause.
- Particular tissue.
- Site involved.

- They can provide valuable clues about the underlying cause.

inflammation with edema & this edema accumulate within closed space

1.SEROUS INFLAMMATION

- Marked by the exudation of cell poor fluid into spaces created by injury to surface epithelial or into body cavities such as peritoneal, pleural, or pericardial cavities.
  GI lung heart
- The fluid in serous inflammation is not infected by destructive organisms and does not contain large numbers of leukocytes
- Accumulation of fluid in these cavities is called an effusion.

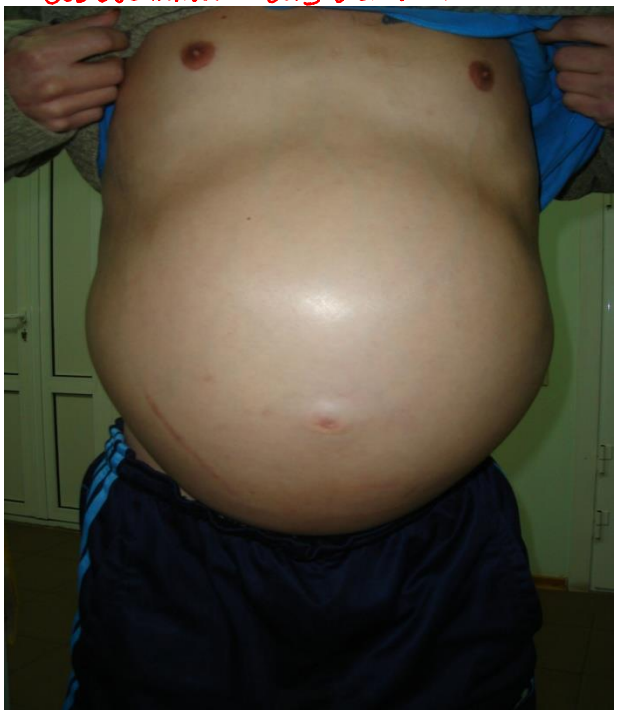
❖Peritoneal effusion = ascites

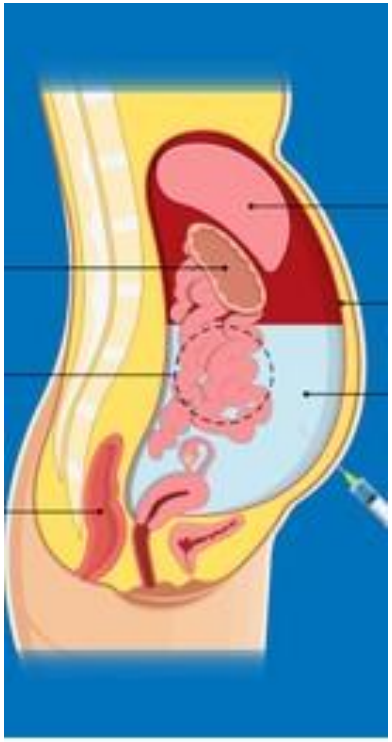
clinical term

excess fluid around the GIT organs

came from inflammation

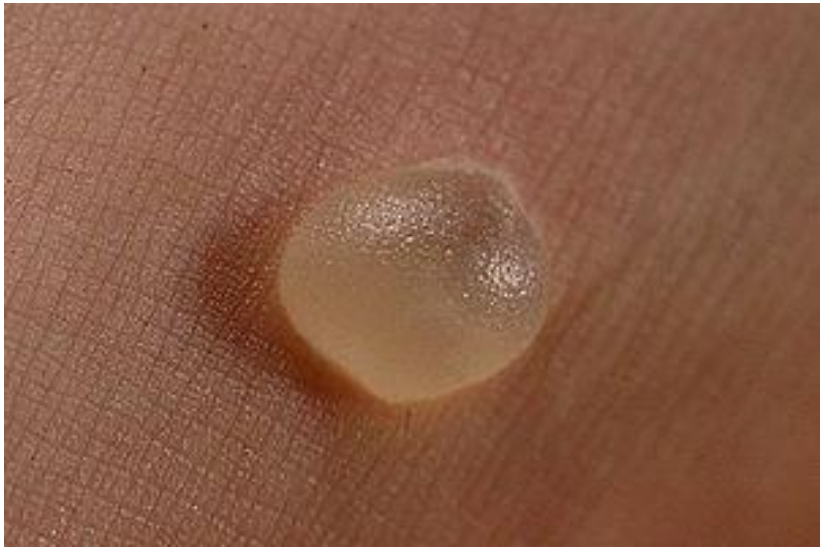
abdominal distention

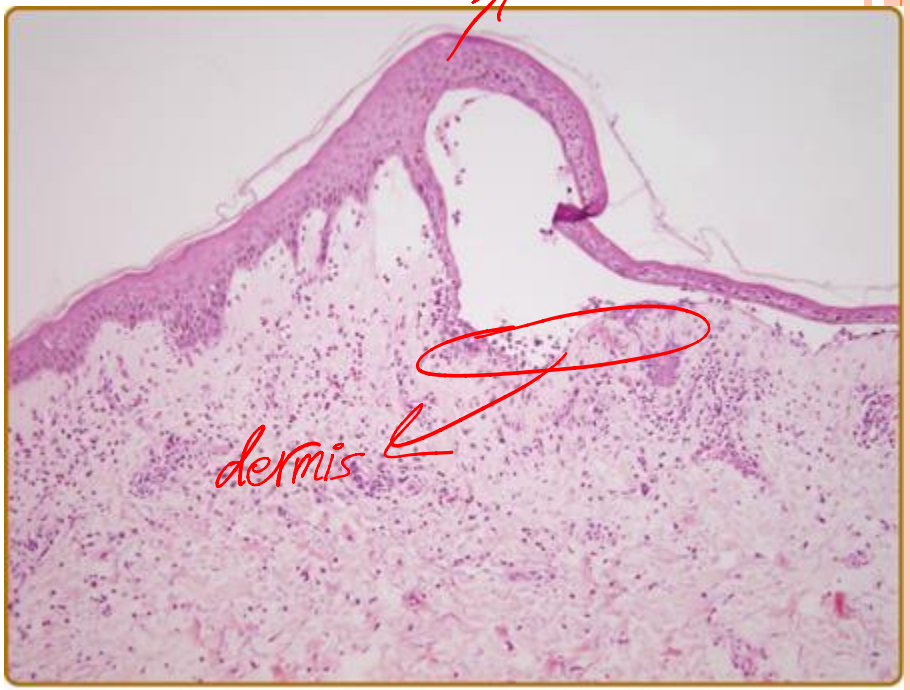




❖SKIN BLISTER—bullous

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





2. Fibrinous Inflammation

- A fibrinous exudate develops when the vascular leaks are large or there is a local procoagulant stimulus.

- A fibrinous exudate is characteristic of inflammation in the lining of body cavities, such as the meninges, pericardium and pleura.

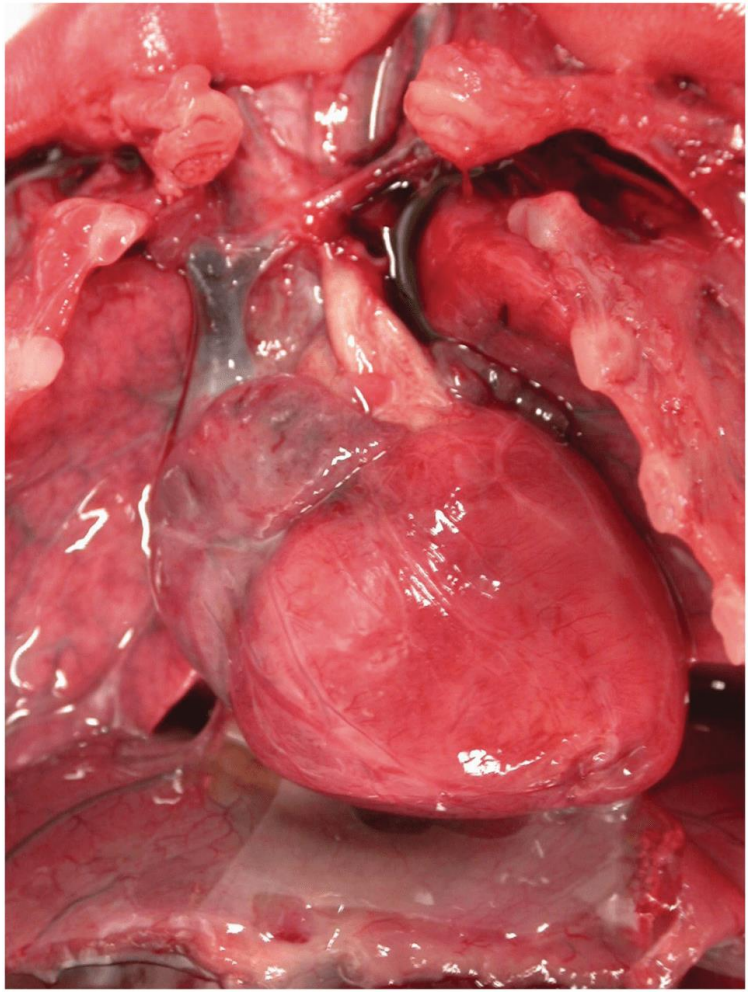
Mechanism of formation

- Large increase in vascular permeability.

- higher-molecular weight proteins such as fibrinogen pass out of the blood.

- fibrin is formed and deposited in the extracellular space

❖GROSSLY



the surface not smooth

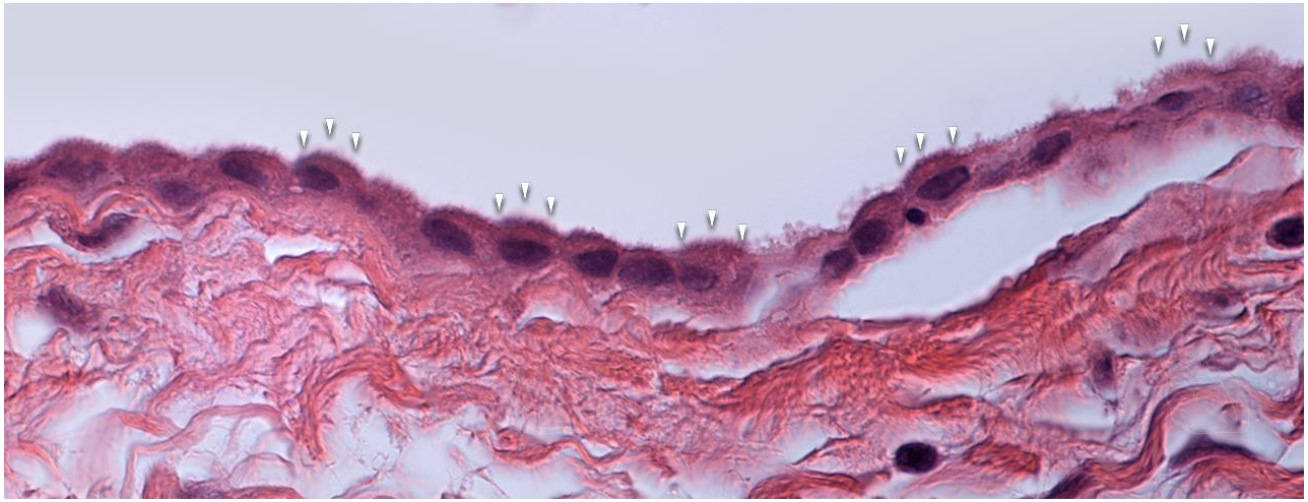
The **pericardial surface** is **dry** with a **coarse granular appearance** caused by **fibrinous exudate**

what are the cells lining the bercardial space?
mesothelial cells

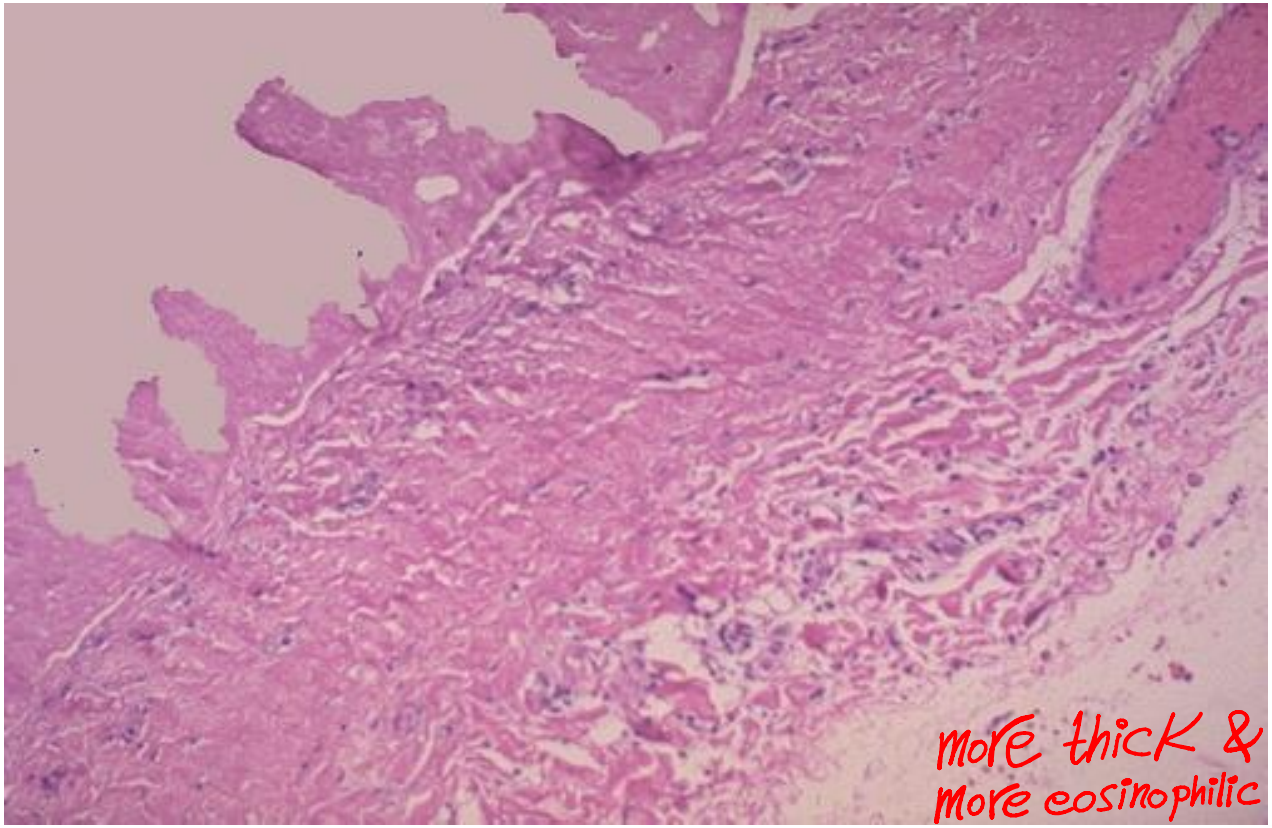
wet

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

Histology



Norml pericrdium composed of thin fibrous wall
Covered by single layer of mesothelial cells



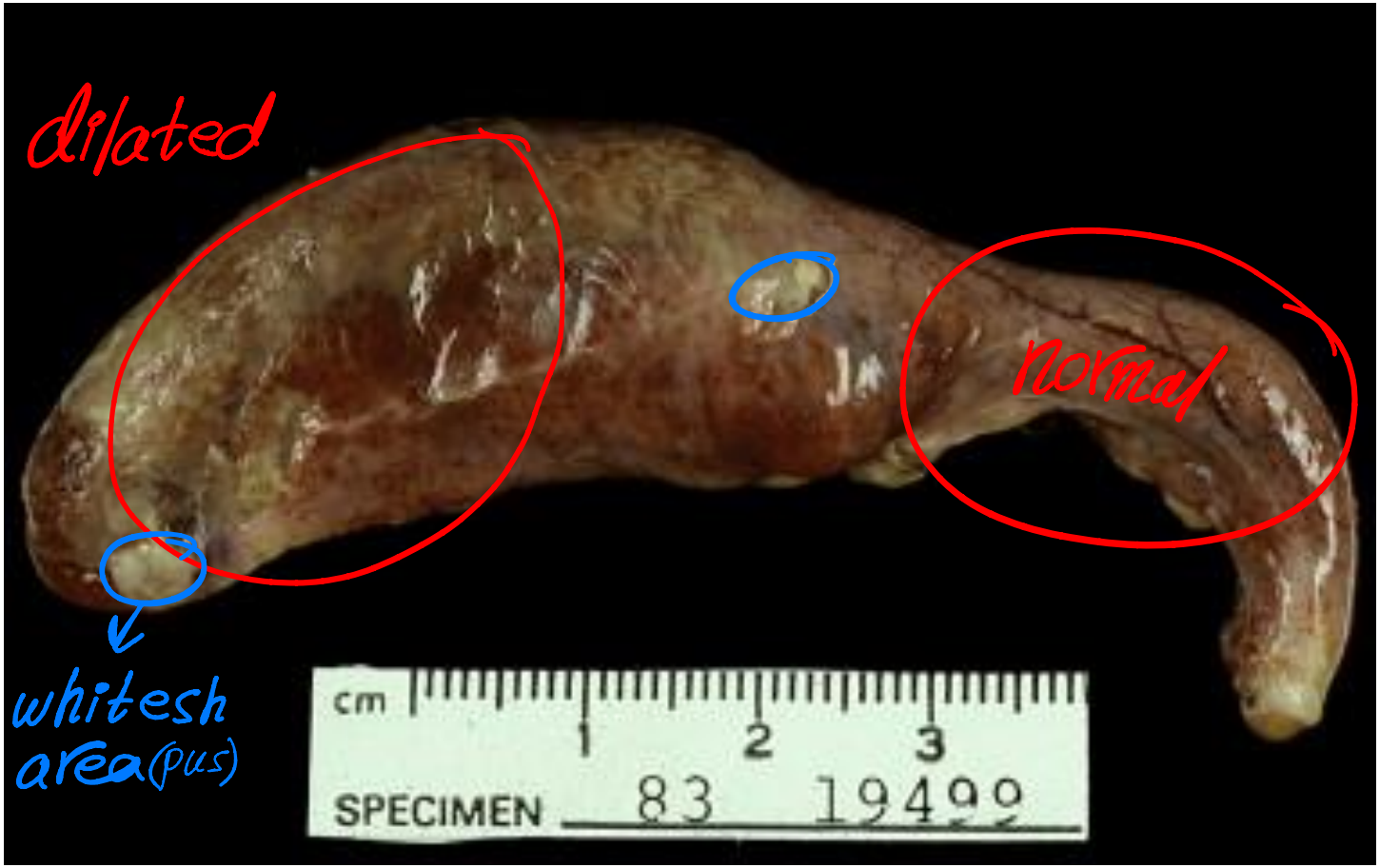
the pericardial surface here shows strands of pink fibrin extending outward. There is underlying inflammation.
fibrin appears as an eosinophilic meshwork of threads

3. Purulent (Suppurative) Inflammation, Abscess

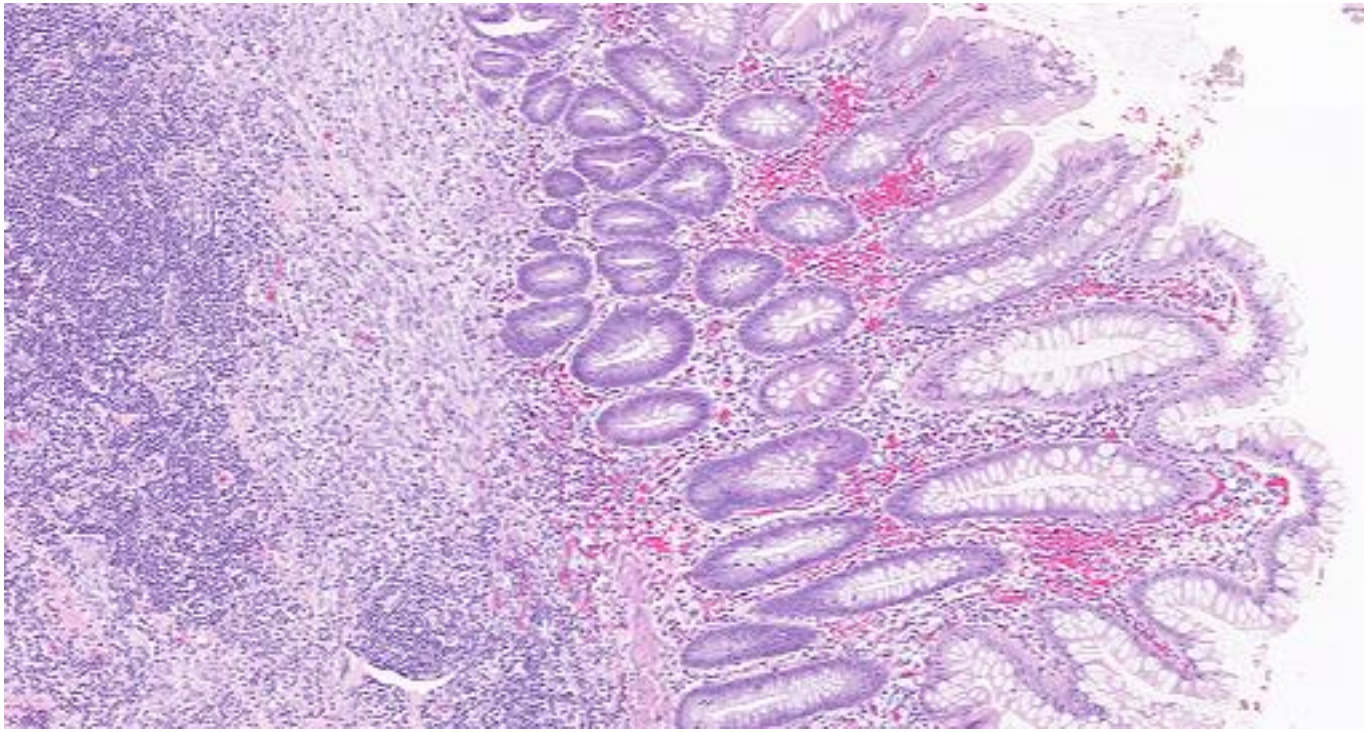
- Purulent inflammation is characterized by the production of pus, an exudate consisting of neutrophils, the liquefied debris of necrotic cells, and edema fluid.

- The most frequent cause is infection with pyogenic (pus-producing) bacteria, such as staphylococci.

A COMMON EXAMPLE OF AN ACUTE SUPPURATIVE INFLAMMATION IS ACUTE APPENDICITIS



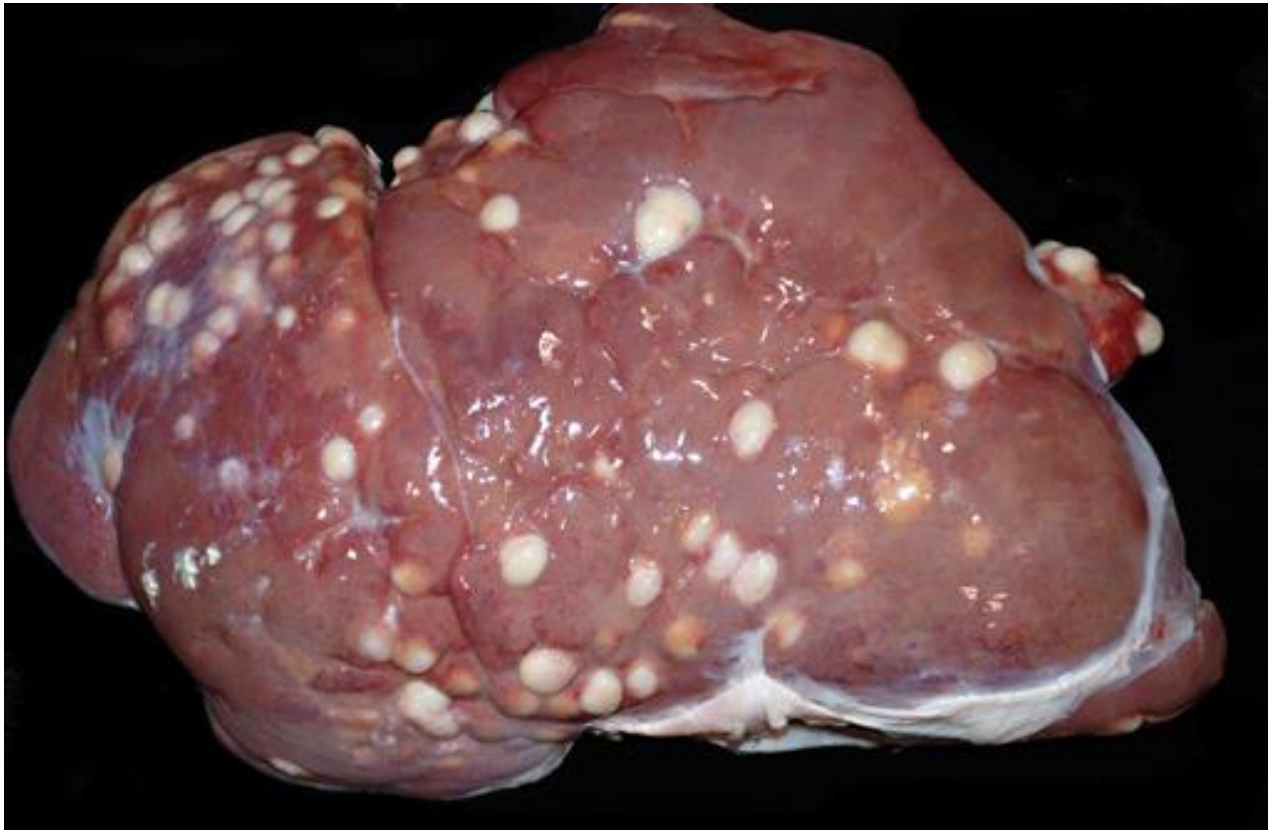
Acute appendicitis



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

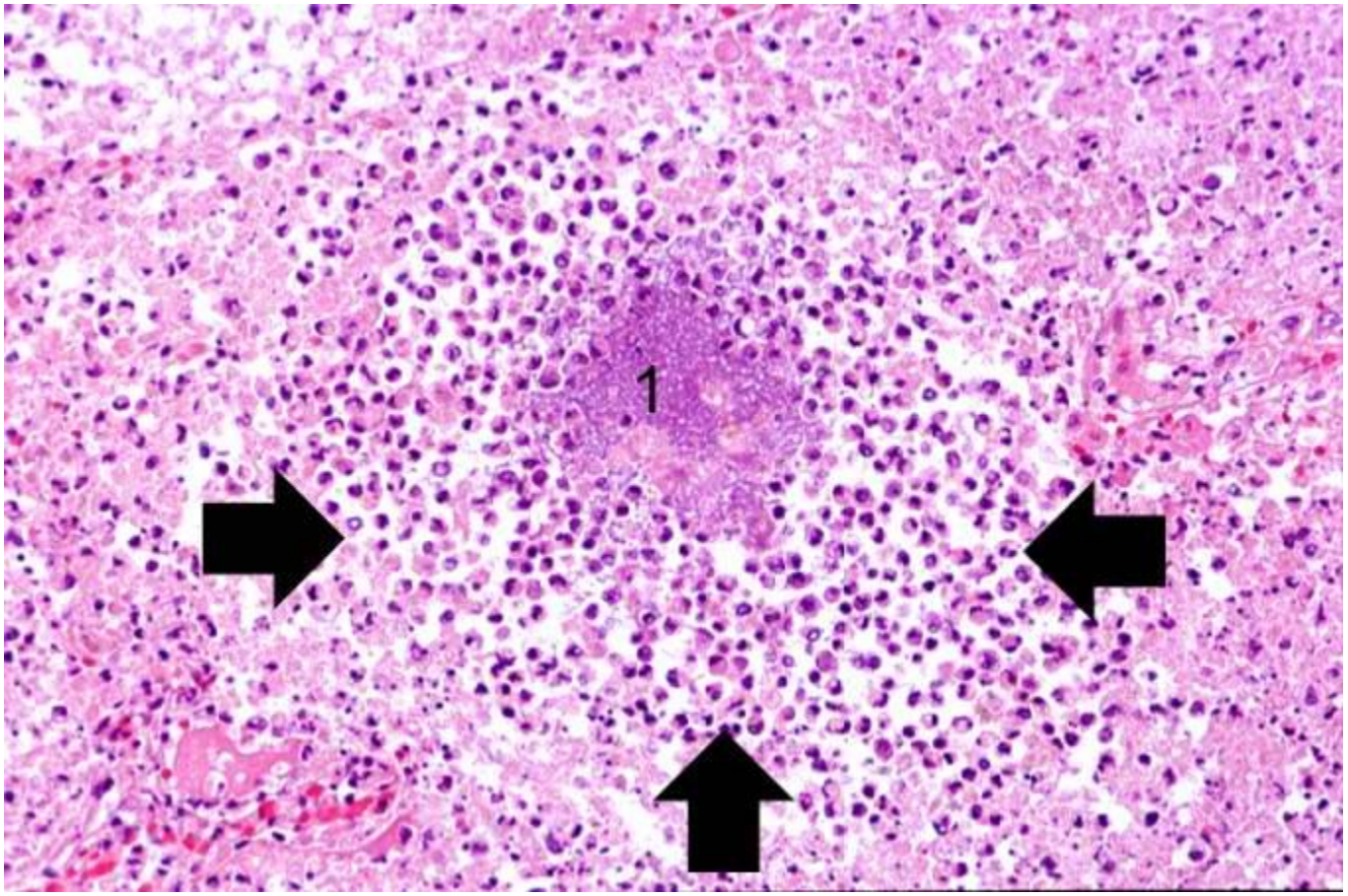
- Abscesses:
- Localized collections of pus caused by suppuration buried in a tissue, an organ, or a confined space.
- They are produced by seeding of pyogenic bacteria into a tissue . In time the abscess may become walled off and ultimately replaced by connective tissue

Liver

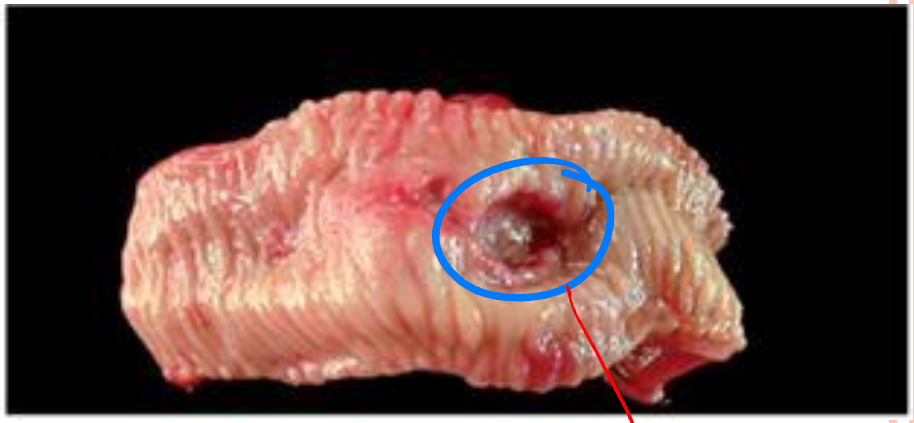


Abscesses have multiple areas:

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



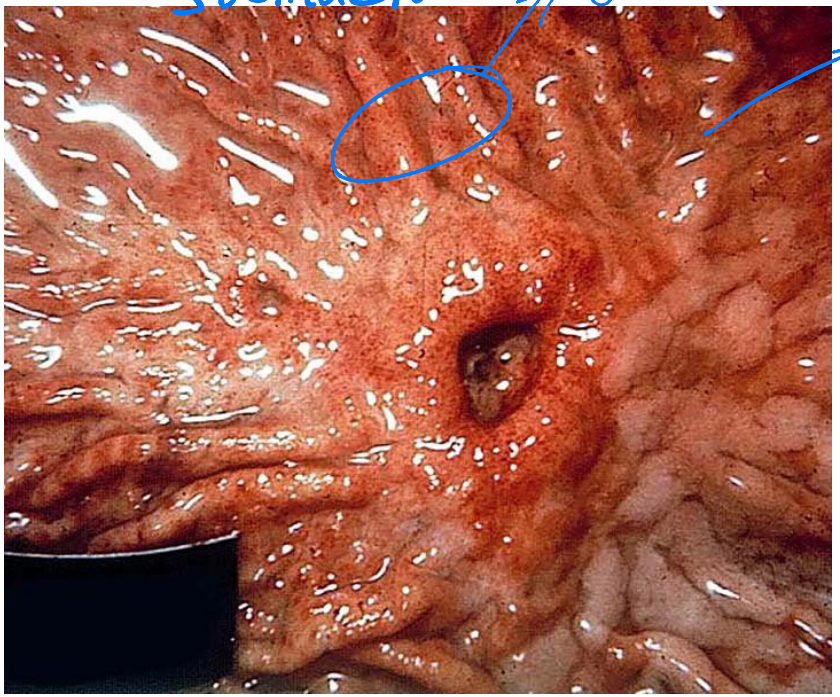
4. Ulcers



- An ulcer is a local defect, or excavation, of the surface of an organ or tissue that is produced by the sloughing (shedding) of inflamed necrotic tissue.

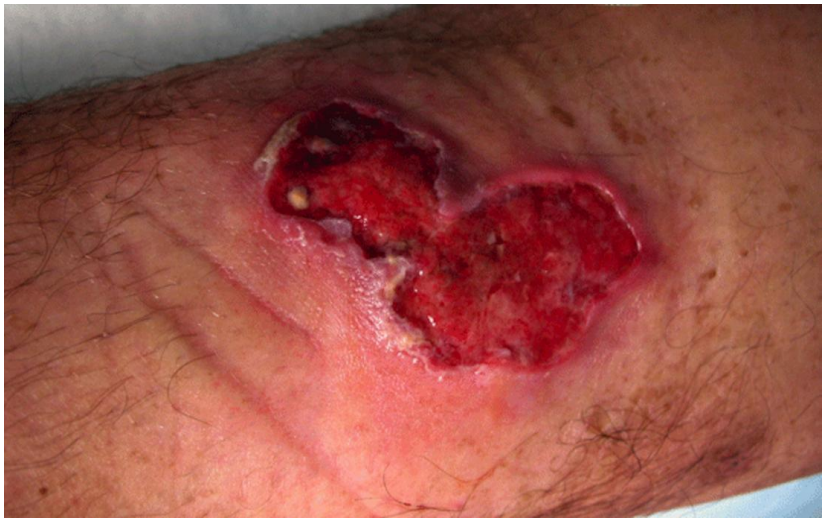
- Ulceration can occur only when tissue necrosis and resultant inflammation exist on or near a surface

- 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



gastric ulcer

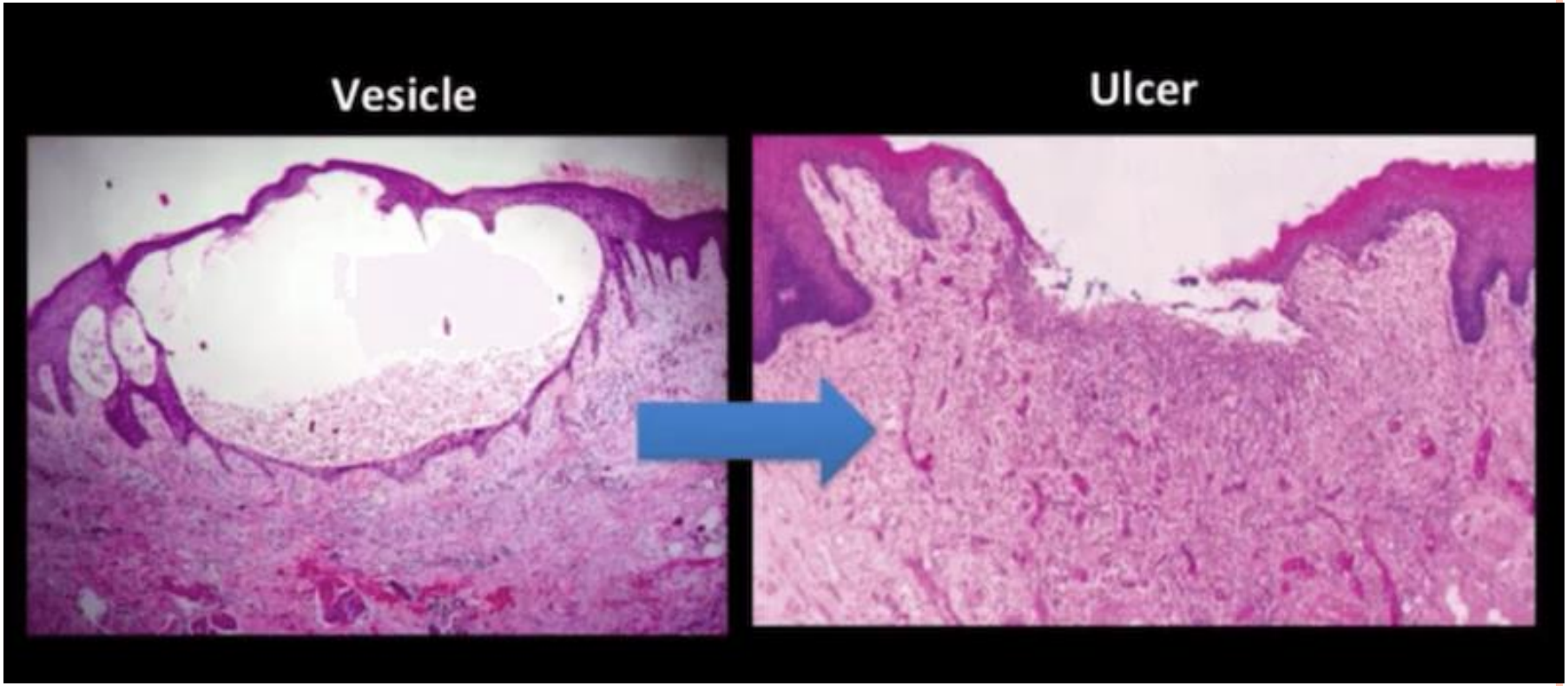
take non-selvetive drugs that inhibit GX-1 & GX-2 with an empty Stomach



HISTOLOGY

bullous

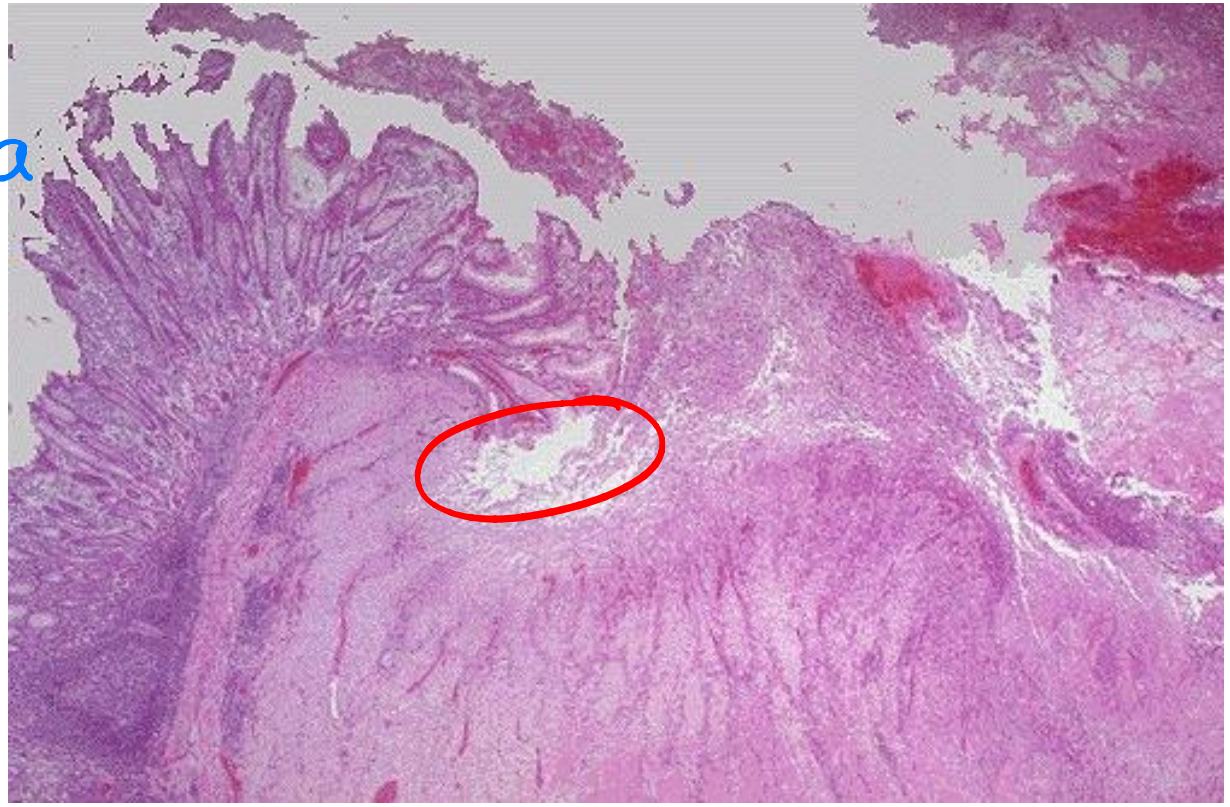
if I loss the surface



sloughing (shedding) of inflamed necrotic tissue

Microscopic features of Ulcers

GI mucosa



Acute stage:

Intense polymorphonuclear infiltration and vascular dilation in the margins of the defect.

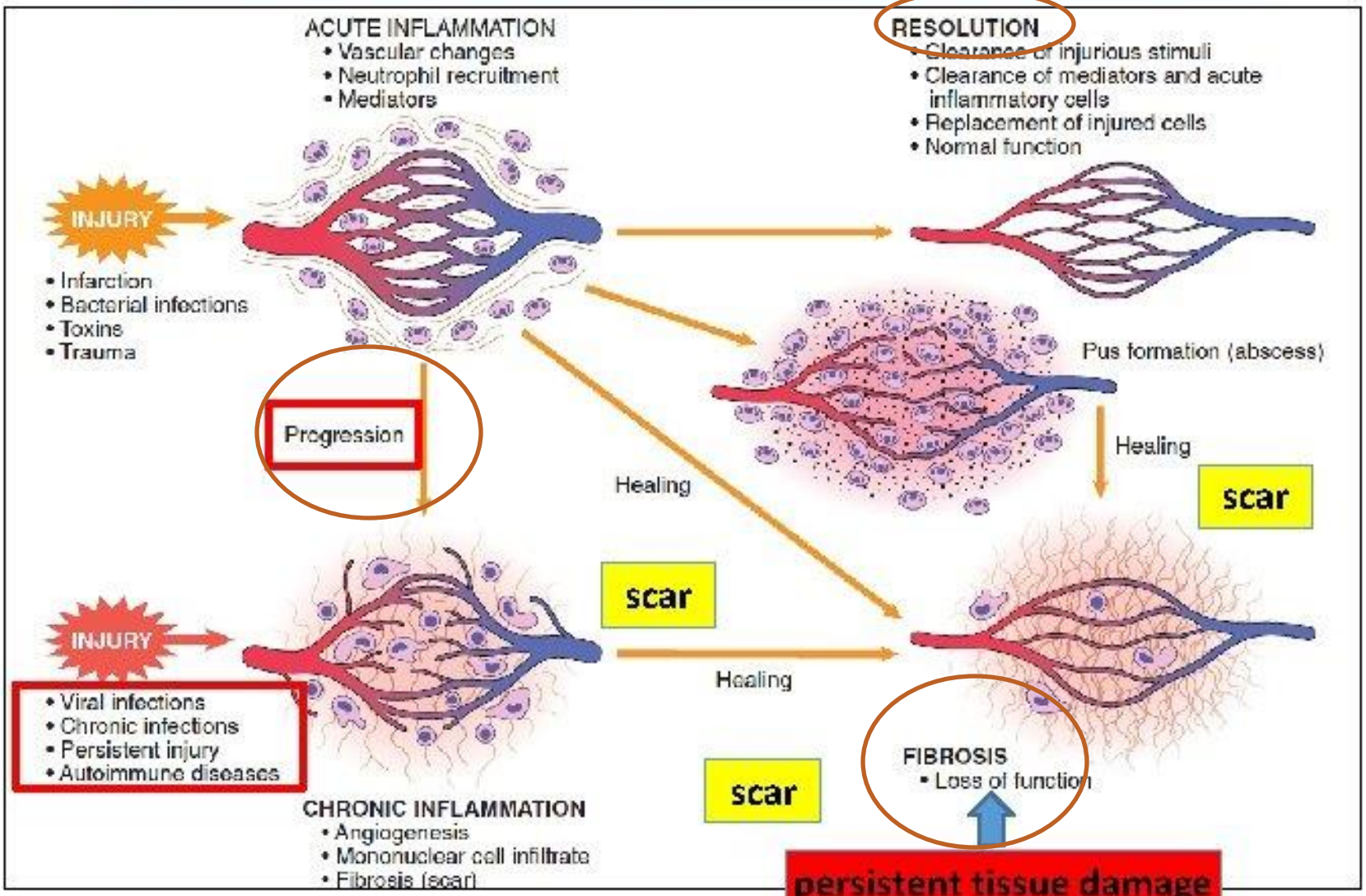
neutrophils

With chronicity:

the margins and base of the ulcer develop fibroblast proliferation, scarring, and the accumulation of lymphocytes, macrophages, and plasma cells.

we have three possible scenarios (outcomes) of termination of acute inflammation

Outcomes of acute inflammation



OUTCOMES OF ACUTE INFLAMMATION

- Acute inflammatory reactions typically have one of three outcomes:

- 1. Complete resolution:
- Occur when the injury is limited or short-lived or when there has been little tissue destruction and the damaged parenchymal cells can regenerate. like liver
- Resolution involves removal of cellular debris and microbes by macrophages, and resorption of edema fluid by lymphatics.

2. Healing by connective tissue replacement (scarring, or fibrosis).

- occurs after substantial tissue destruction, when the inflammatory injury involves tissues that are incapable of regeneration, or when there is abundant fibrin exudation.

- connective tissue grows into the area of damage or exudate, converting it into a mass of fibrous tissue.

from acute to chronic

3. Progression of the response to chronic inflammation.

- occurs when the acute inflammatory response cannot be resolved, as a result of either :
  - the persistence of the injurious agent
  - interference with the normal process of healing

why?? or example

CHRONIC INFLAMMATION

- Chronic inflammation is a response of prolonged duration (weeks or months) in which:

- inflammation.
- tissue injury.
- attempts of repair.

coexist,
in varying combinations.

- It may follow acute inflammation, as described earlier, or may begin insidiously,

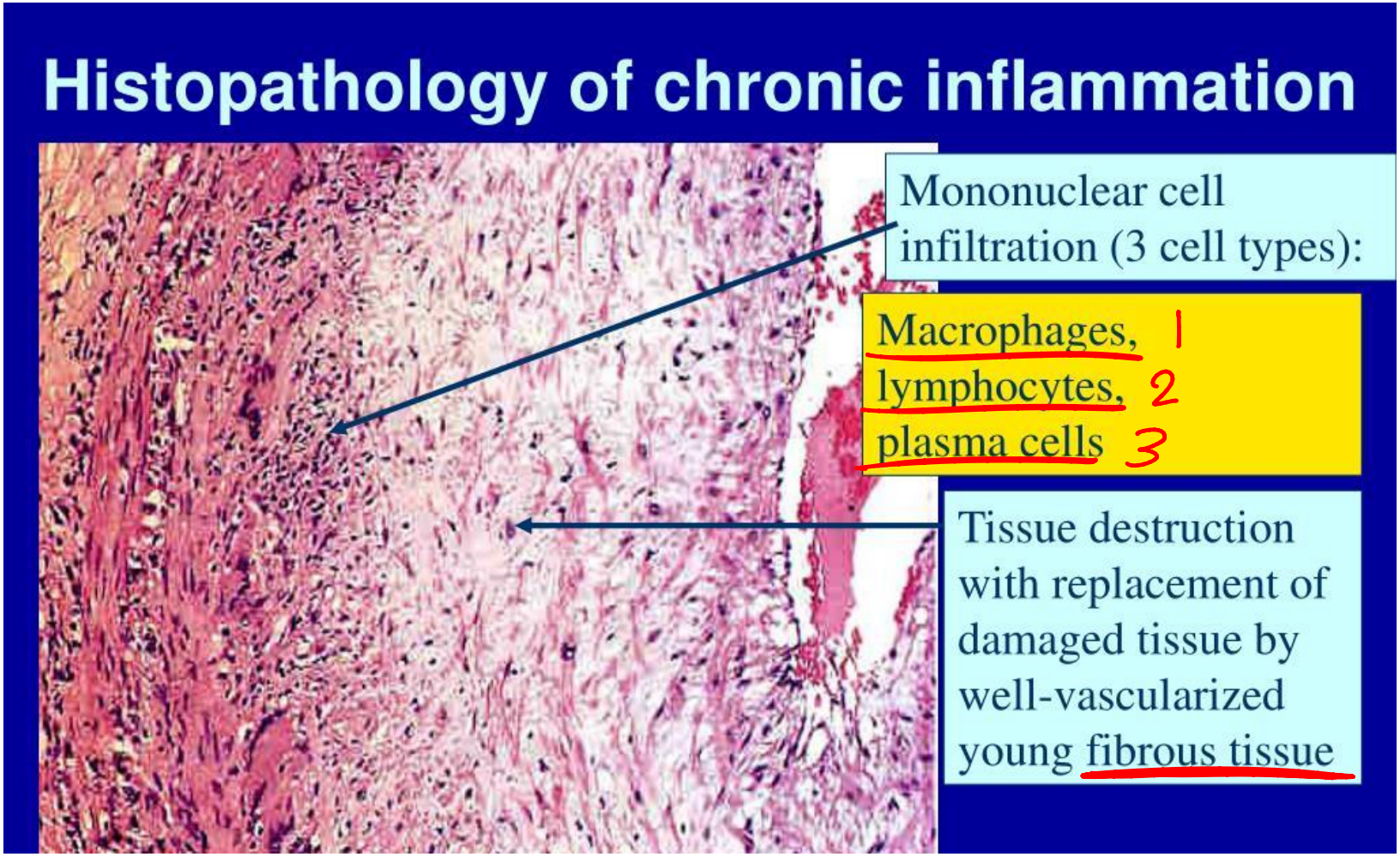
من البداية بلش وهو chronic

Causes of Chronic Inflammation

- Persistent infections, e.g??. Like ⇒ TB, fungal infection parasitic infection & HIV
- Hypersensitivity diseases.
- Autoimmune disease.
- Allergic diseases.
- Prolonged exposure to potentially toxic agents, e.g Silica.

People who work in mines, the silica will enter there airway and then enter inside macrophage, the macrophage can't destroy the silica

Histopathology of chronic inflammation



- healing by connective tissue replacement of damaged tissue,

Cells and Mediators of Chronic Inflammation

- Macrophages
- Lymphocytes

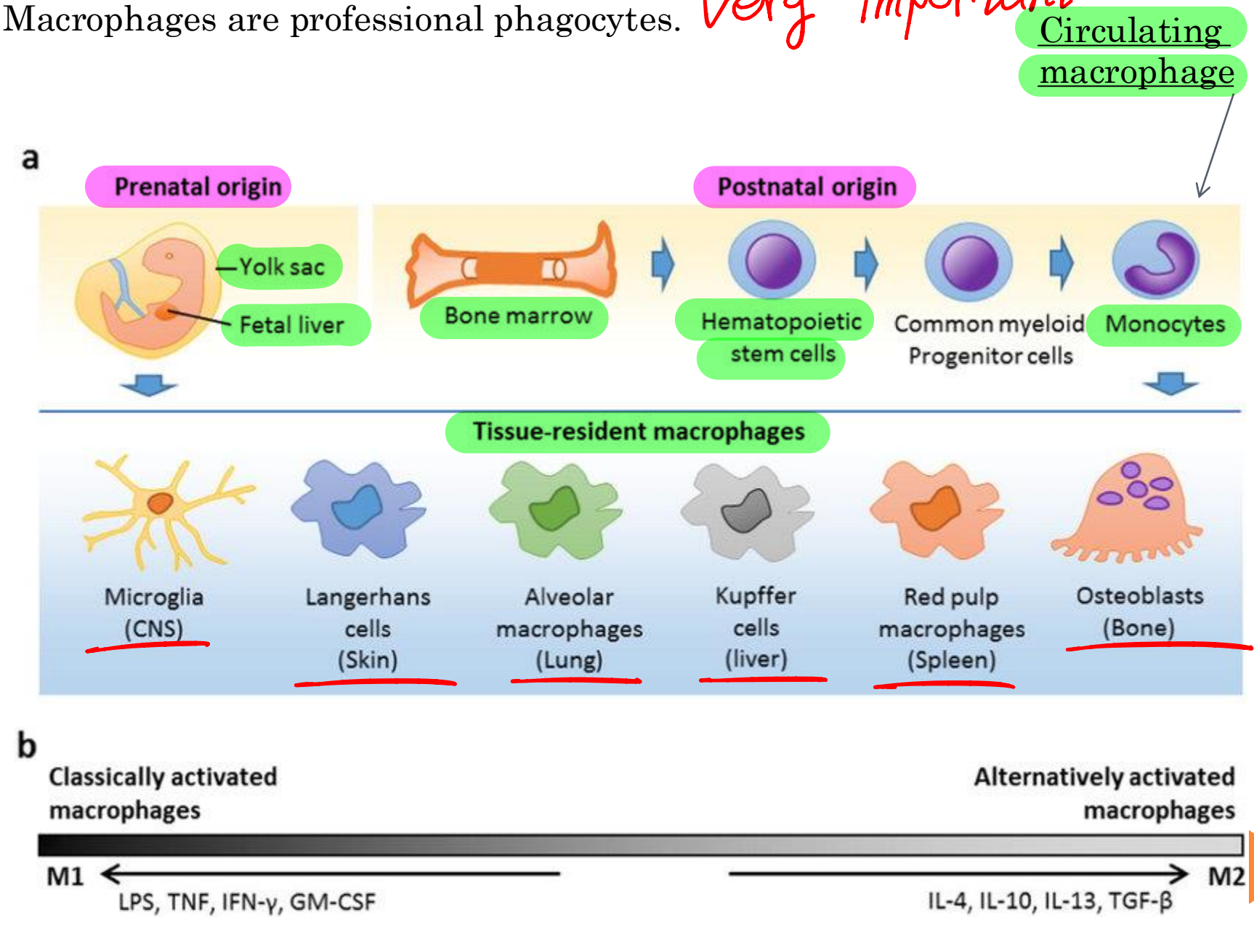
1. Macrophages

- The dominant cells in most chronic inflammatory reactions .
- Become the dominant cell population in inflammatory reactions within 48 hours of onset.
- There are two major pathways of macrophage activation, (depends on the nature of the activating signals):

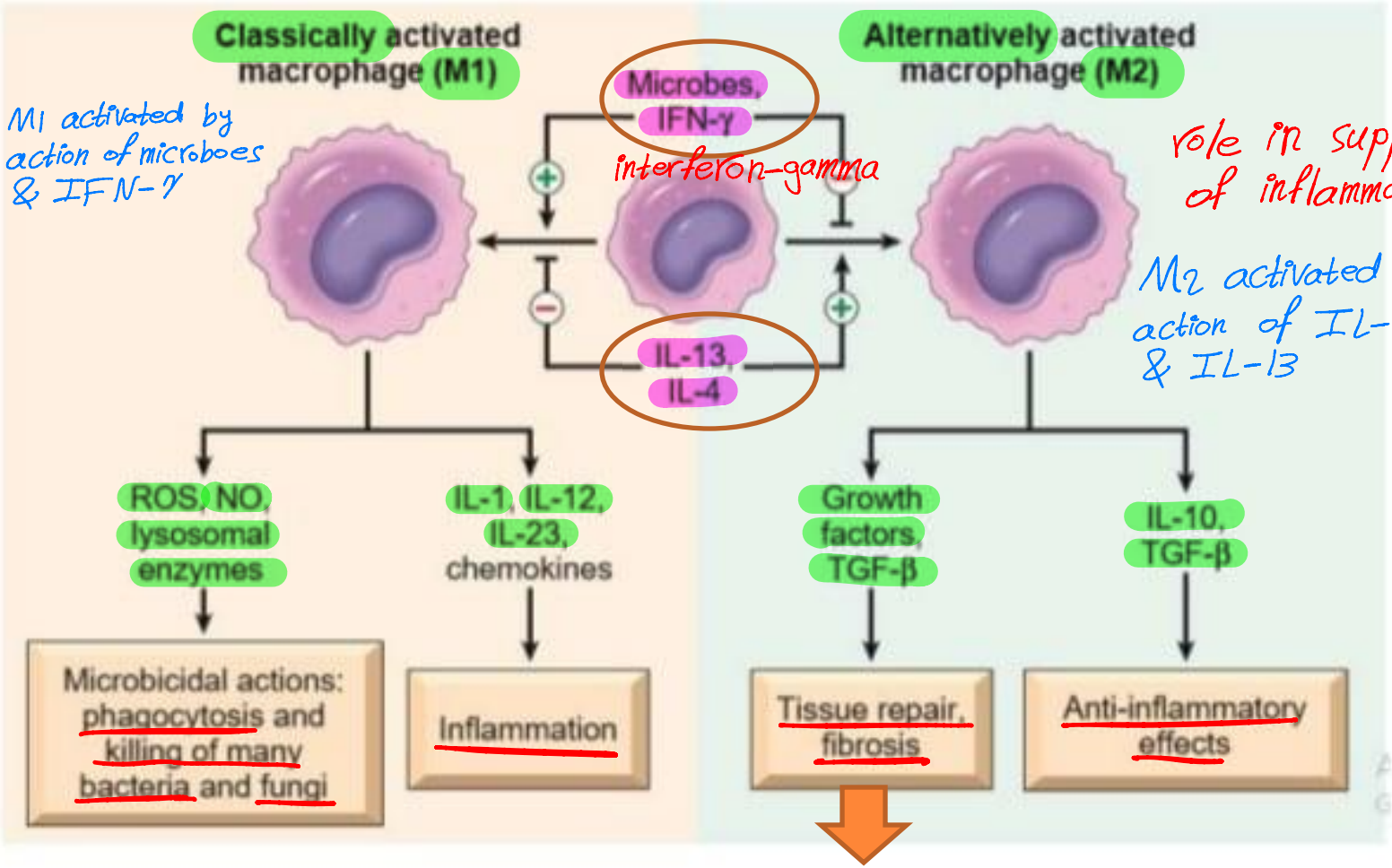
- Classical:
- designed to destroy the offending agents.

- Alternative :
- initiates tissue repair.

Macrophages are professional phagocytes. very important



Activation pathways



M1 activated by action of microboes & IFN-γ

interferon-gamma

role in suppression of inflammation

M2 activated by action of IL-4 & IL-13

They secrete growth factors that promote

1 ➢ Angiogenesis. formation of new blood vessels

2 ➢ activate fibroblasts.

3 ➢ stimulate collagen synthesis.

The products of activated macrophages

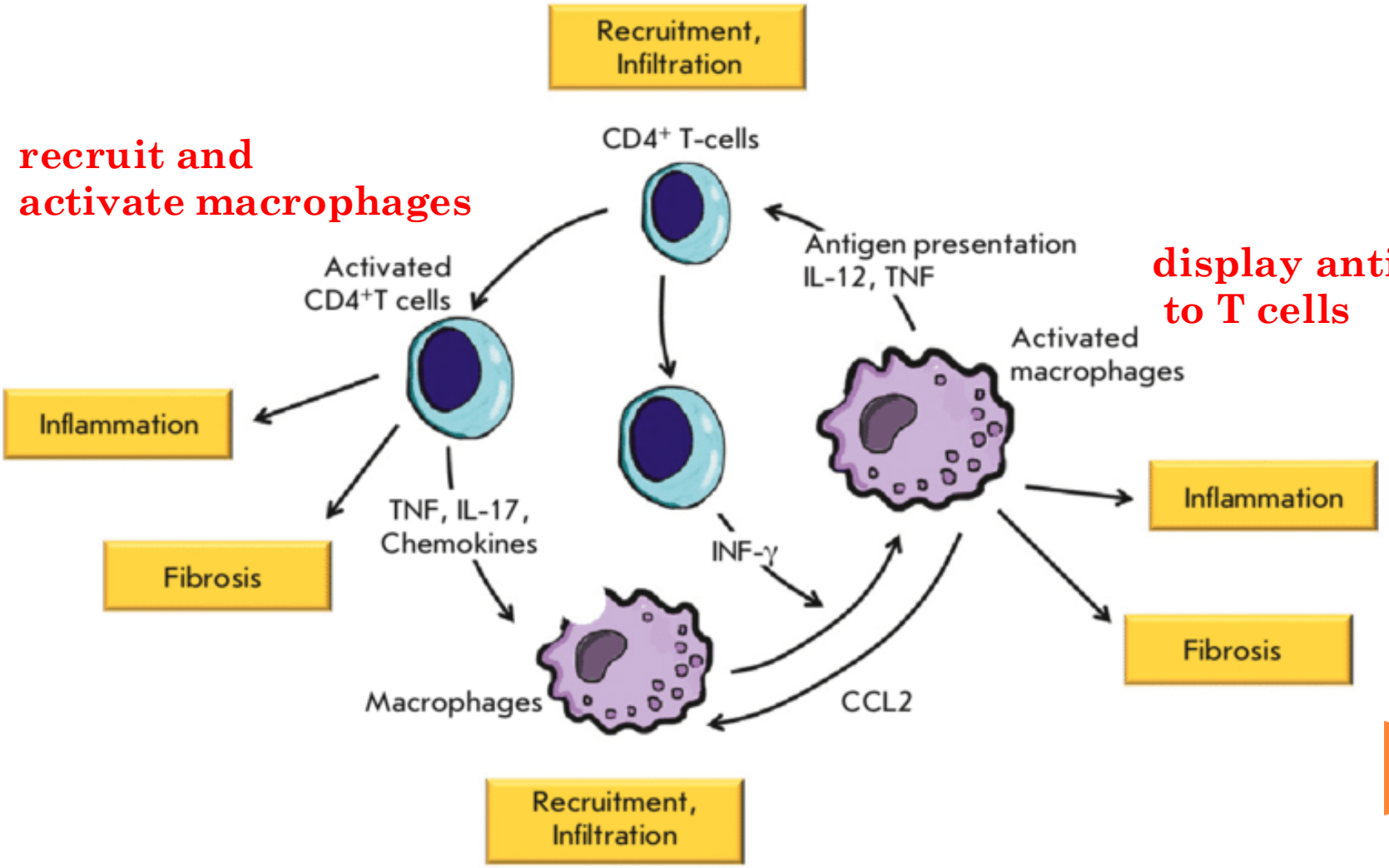
- **Eliminate injurious agents such as microbes.**
- **Initiate the process of repair.**
- **Responsible for much of the tissue injury in chronic inflammation**

2. Lymphocytes

- Microbes and other environmental antigen activate T and B lymphocytes, which amplify and propagate chronic inflammation.

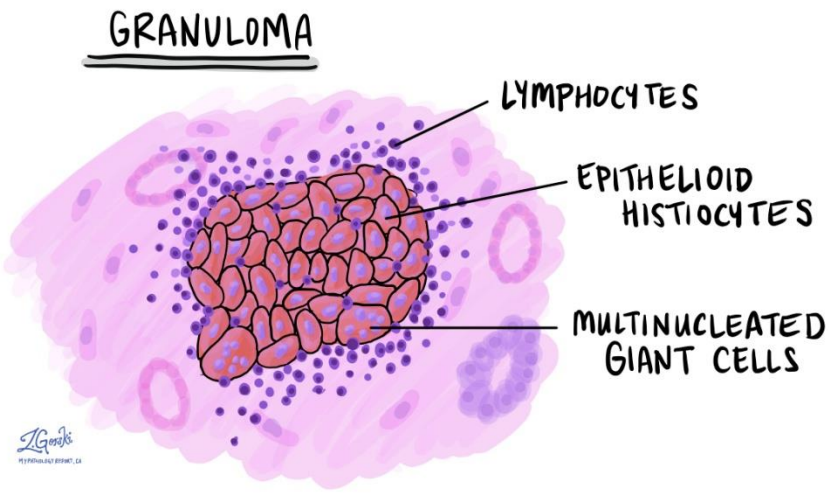
- Some of the strongest chronic inflammatory reactions, such as granulomatous inflammation, are dependent on lymphocyte responses.

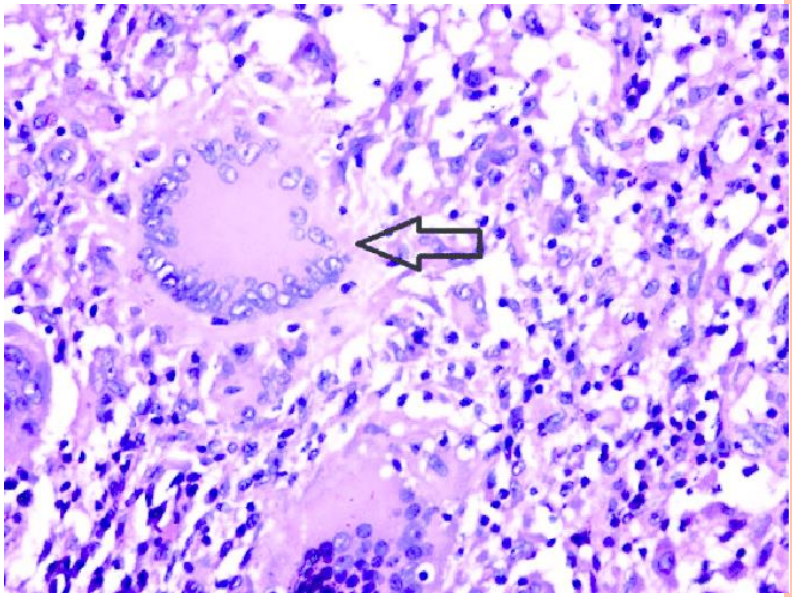
Lymphocytes and macrophages interact in a bidirectional way.

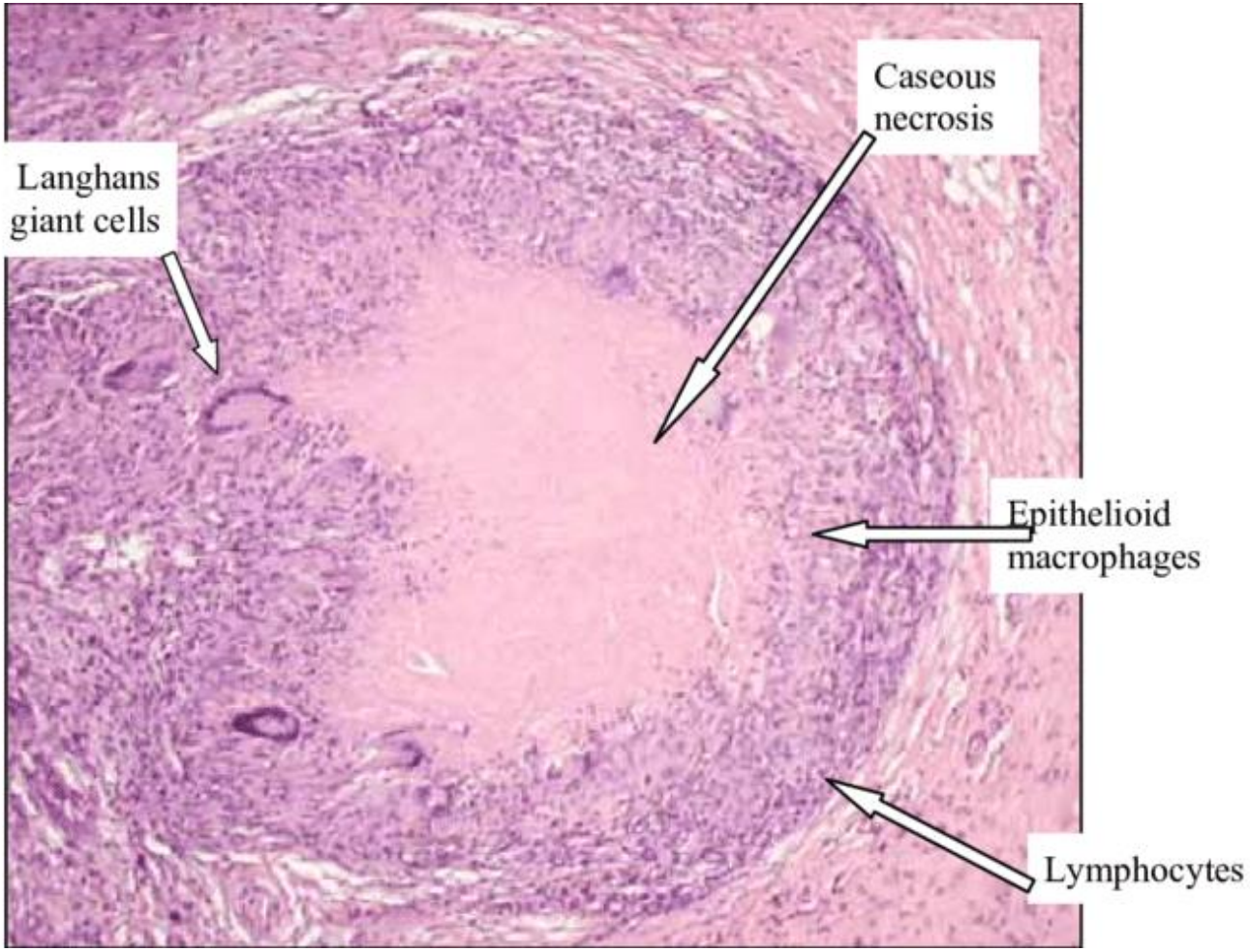


Granulomatous Inflammation

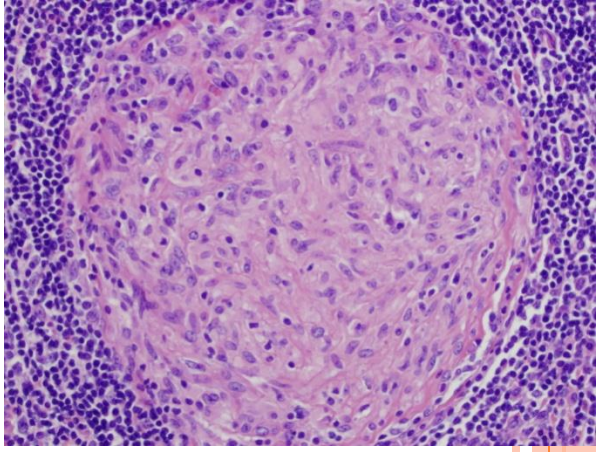
- Granulomatous inflammation is a form of chronic inflammation characterized by collections of activated macrophages, often with T lymphocytes.
- Granuloma formation is a cellular attempt to contain an offending agent that is difficult to eradicate






Caseous necrosis
Langhans giant cells
Epithelioid macrophages
Lymphocytes

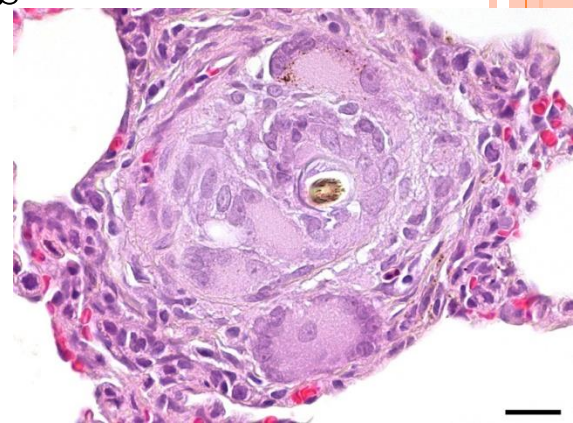
Where??

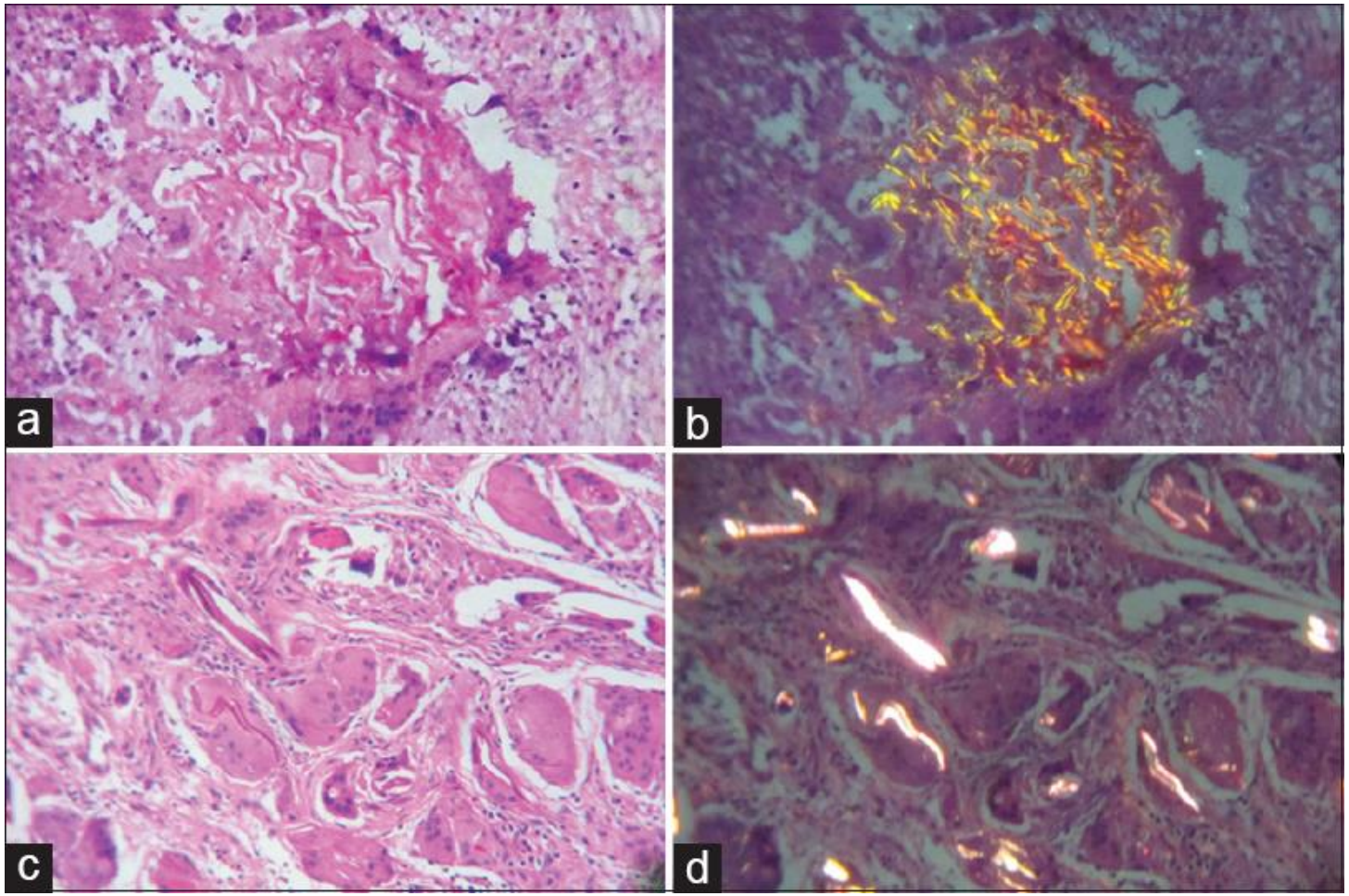


TYPES OF GRANULOMAS;

- 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





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.

Table 3.9 Examples of Diseases With Granulomatous Inflammation

Disease	Cause	Tissue Reaction
Tuberculosis	*Mycobacterium tuberculosis*	Caseating granuloma (tubercle): focus of activated macrophages (epithelioid cells), rimmed by fibroblasts, lymphocytes, histiocytes, occasional Langhans giant cells; central necrosis with amorphous granular debris; acid-fast bacilli
Leprosy	*Mycobacterium leprae*	Acid-fast bacilli in macrophages; noncaseating granulomas
Syphilis	*Treponema pallidum*	Gumma: microscopic to grossly visible lesion, enclosing wall of macrophages; plasma cell infiltrate; central cells are necrotic without loss of cellular outline; organisms difficult to identify in tissue
Cat-scratch disease	Gram-negative bacillus	Rounded or stellate granuloma containing central granular debris and recognizable neutrophils; giant cells uncommon
Sarcoidosis	Unknown etiology	Noncaseating granulomas with abundant activated macrophages
Crohn disease (inflammatory bowel disease)	Immune reaction against undefined gut microbes and, possibly, self antigens	Occasional noncaseating granulomas in the wall of the intestine, with dense chronic inflammatory infiltrate

SYSTEMIC EFFECTS OF INFLAMMATION

- Inflammation is associated with cytokine-induced systemic reactions that are collectively called the acute-phase response.

- The cytokines TNF, IL-1, and IL-6 are important mediators of the acute phase reaction.

The acute-phase response consists of several clinical and pathologic changes:

- 1.Fever:
- Substances that induce fever are called pyrogens.
- caused by prostaglandins especially **PGE2** that are produced in the vascular and perivascular cells of the hypothalamus.

2.Acute-phase proteins

3.Leukocytosis