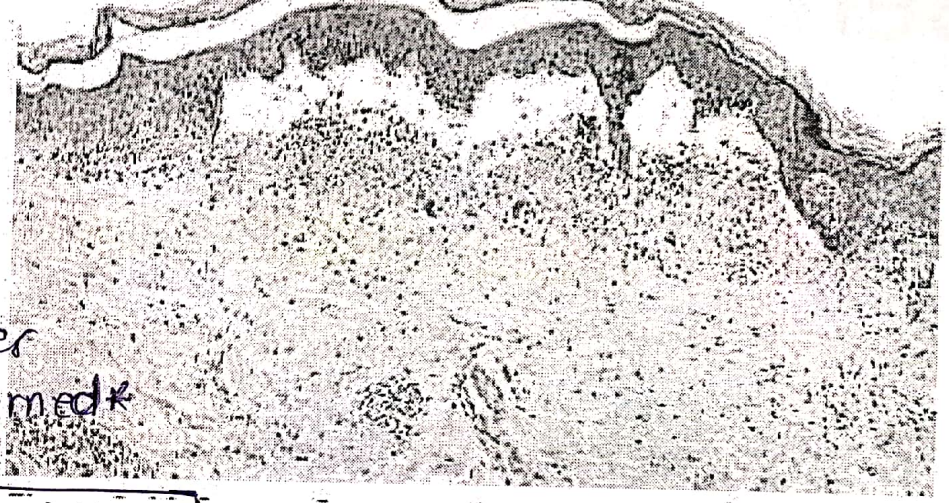
(serous / fluid)

# Serous inflammation → lining of the organ from the outer surface

- Characterized by the outpouring of a watery, relatively protein-poor fluid that, depending on the site of injury, derives either from the plasma or from the secretions of mesothelial cells lining the peritoneal, pleural, and pericardial cavities.
- Fluid in a serous cavity is called an effusion.
- End with respiratory or cardiac impairment.
- The skin blister resulting from a burn or viral infection is a good example of the accumulation of a serous effusion either within or immediately beneath the epidermis of the skin .

← serous

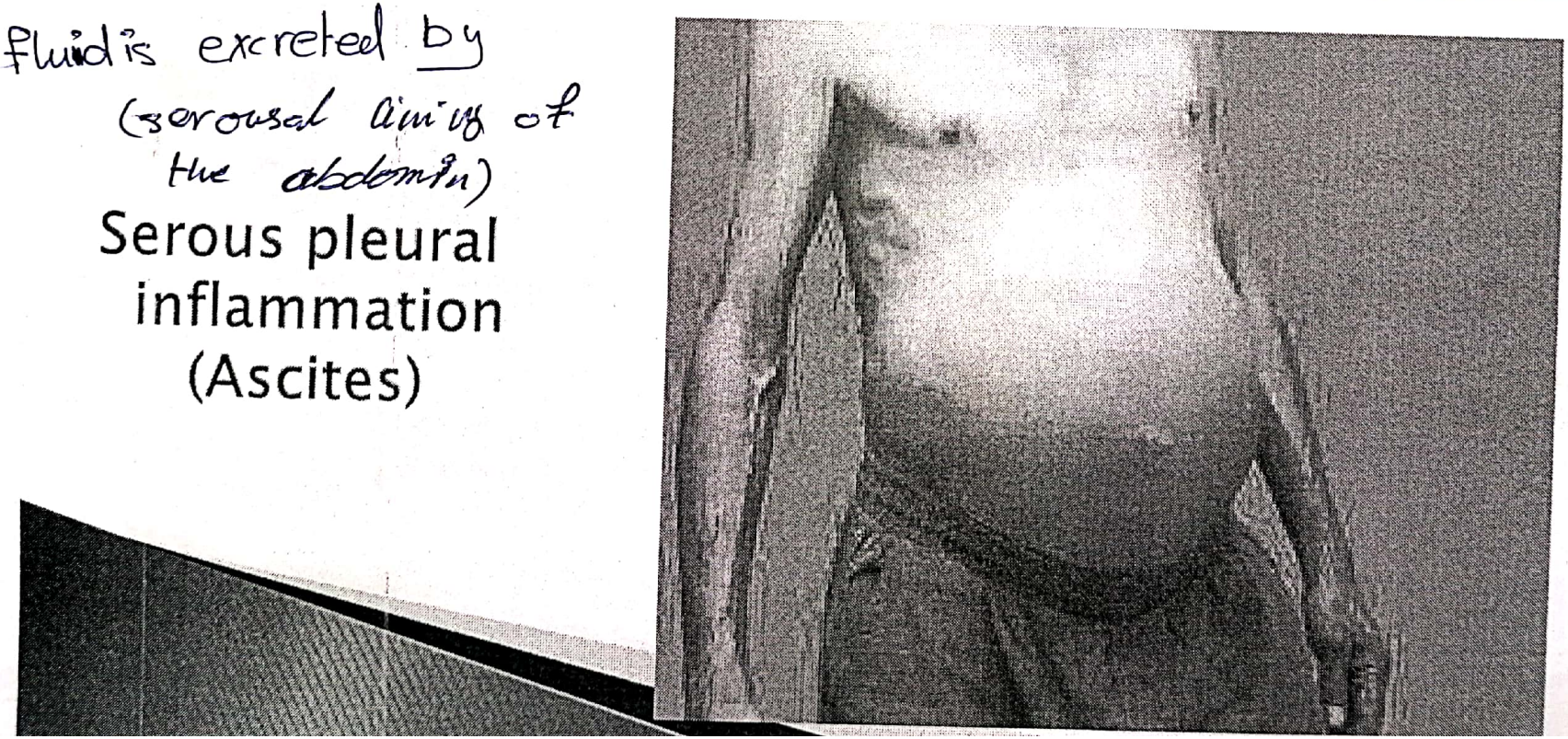
*bubble after this area burned*



Skin blister showing the epidermis separat from the dermis by a focal collection of serous effusion.

fluid is excreted by (serousal lining of the abdomin)

Serous pleural inflammation (Ascites)



# Fibrinous inflammation

Occurs in *sever injuries. → (causative agent)

There will be increase vascular permeability lead to exudation of large molecules (such as fibrinogen) passing the endothelial barrier.

specific component of this type

- Histologically, the accumulated extravascular fibrin appears as an eosinophilic meshwork of threads.

- Exudates ► Resolution:

  Degraded by fibrinolysis and the accumulated debris is removed by macrophages.

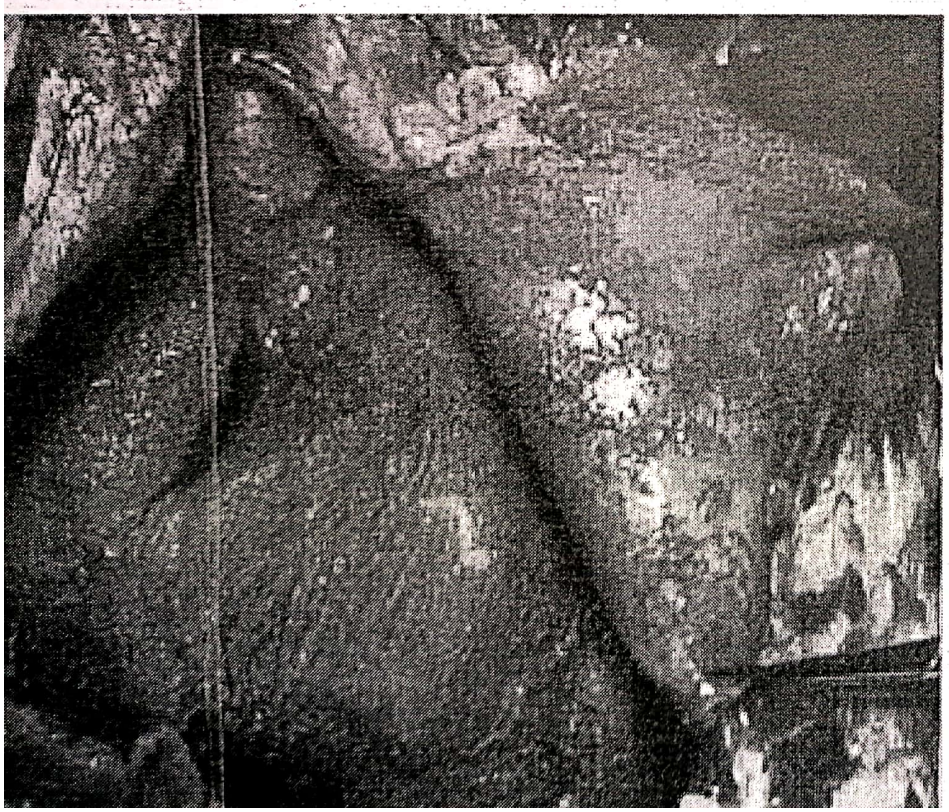
- Exudates ► Organization ► Scarring. (will district the function of the organ)

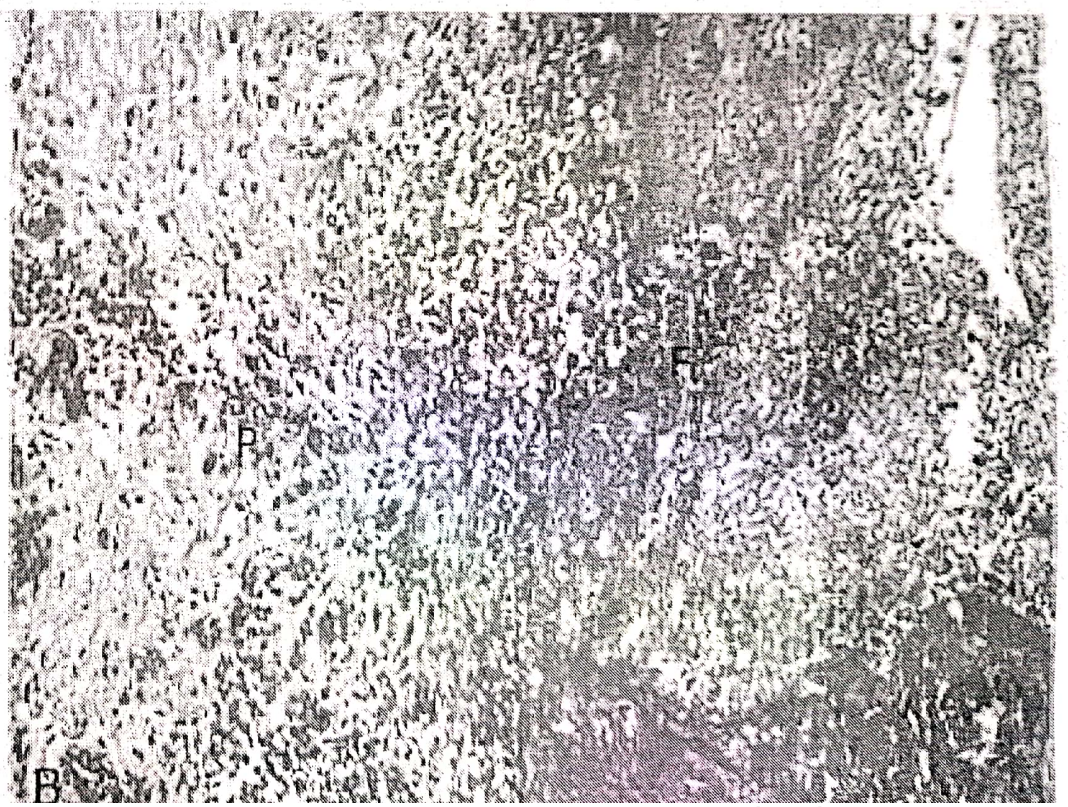
  Failure of removing fibrin completely ► ingrowths of fibroblasts and blood vessels.

such as → severe MI

**ization of fibrinous pericardial exudate forms dense fibrous tissue that bridges or obliterates the pericardial space and restricts myocardial function.**

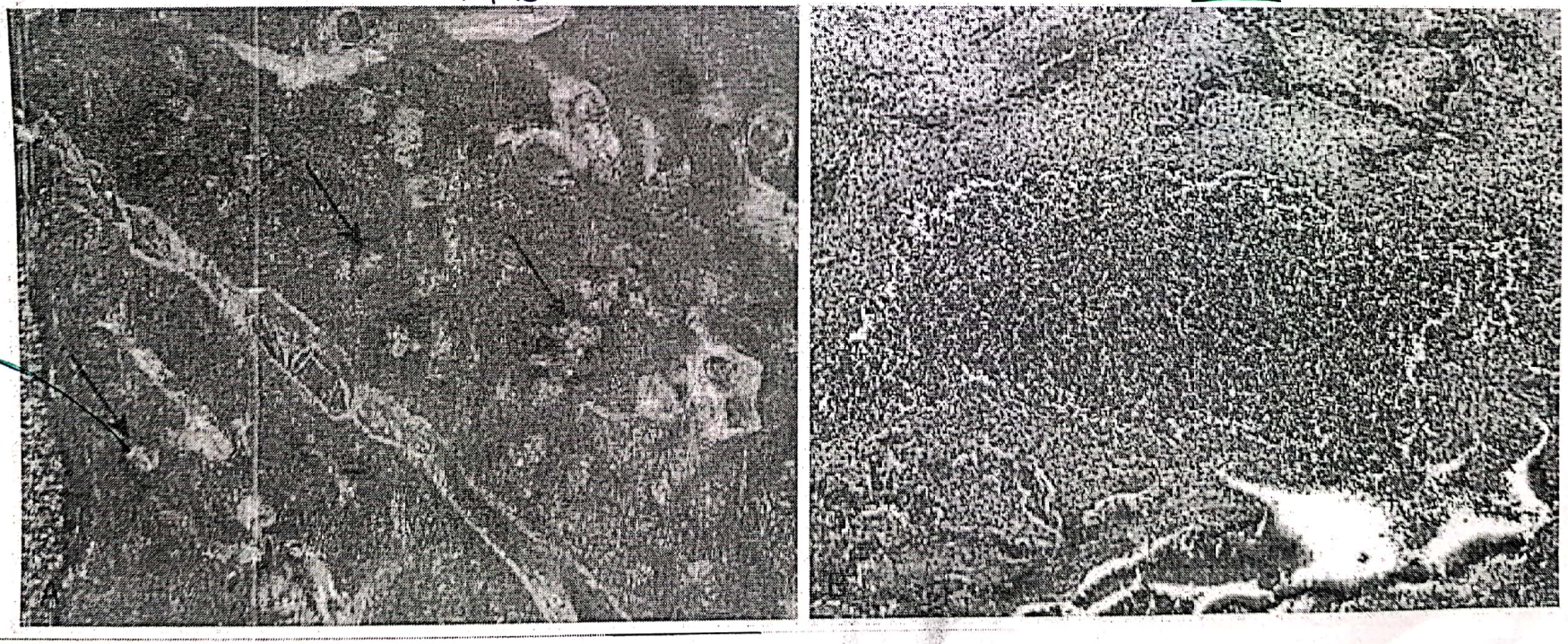
scarring distict the function of the organ





- **Abscess** :Focal collection of pus caused by pyogenic organisms or by secondary infections of necrotic foci.
- Composed of: Large amounts of purulent exudate (pus) consisting of neutrophils, necrotic cells and edema fluid.

(Abscess is surrounded by thin wall)



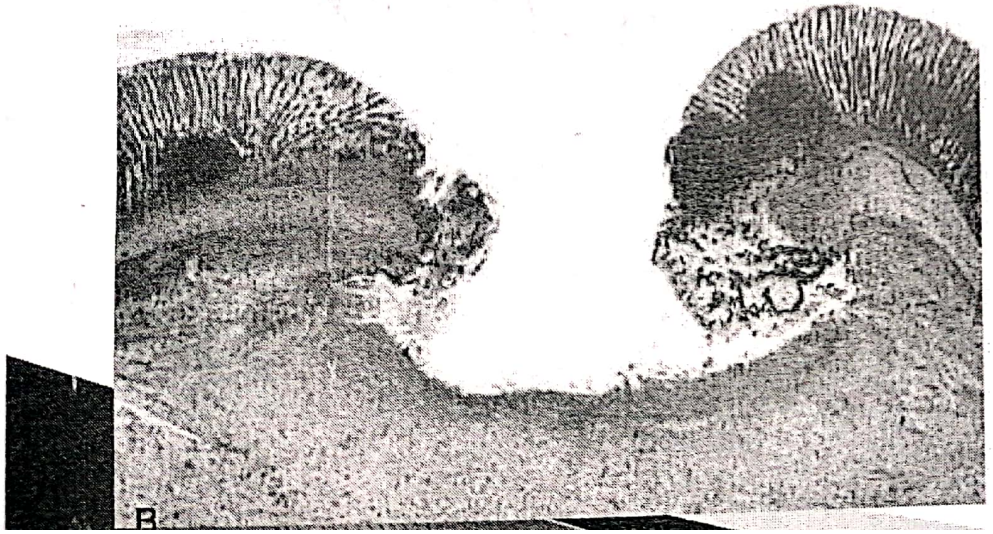
neutrophils (sheets)

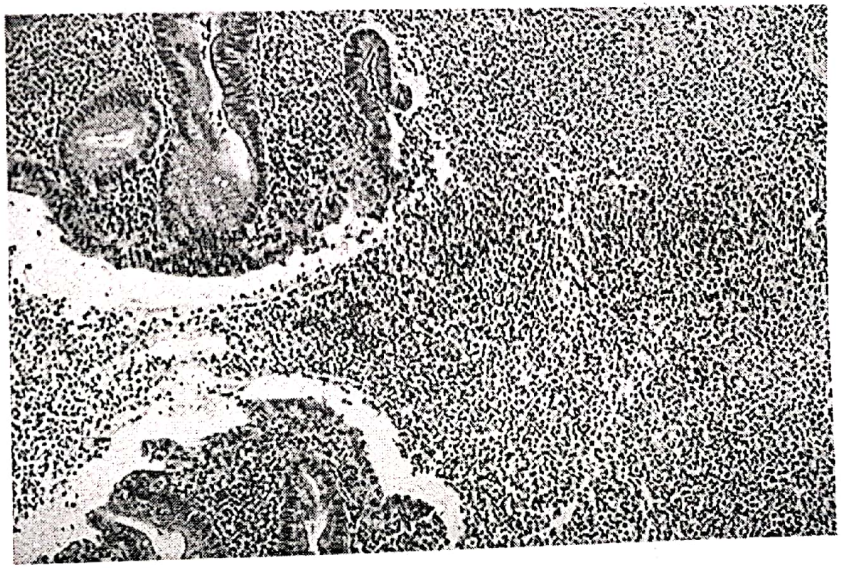
- **Ulcer**: It is local defect, or excavation, of the surface epithelium produced by sloughing (shedding) of inflammatory necrotic tissue.
- Ulcers are most commonly encountered in:

(1) The gastrointestinalor genitourinarytract

(2) The subcutaneous tissue.

* Components of ulcer
  1. sloughing of the epithelium
  2. sheets of neutrophils





# Categories of Chronic Inflammation

- Chronic inflammation is inflammation of prolonged duration (weeks to years) in which continuing inflammation, tissue injury, and healing, often by fibrosis, proceed simultaneously.

- It is either:-

1- Progression of acute inflammation to chronic.

Occurs when the acute response cannot be resolved due to:-

A. Persistence of the injurious agent. / problem in the Immune-system

B. Interference of normal healing process.

2- Chronic inflammation from the onset.

(e.g. Some viral infections) T.B

# Causative agents of chronic inflam

**1- Low-grade, persistent irritant agents** that are u penetrate deeply or spread rapidly.

**2- Moderate to low virulent microbes** like viruses, bacteria, fungi, and larger parasites that are difficu eradicate like:-

imp

- Mycobacteria → Tuberculosis T.B
- Treponema pallidum → Syphilis
- Actinomyces → Actinomycosis

**3- Foreign bodies** such as talc, silica, asbestos, and suture materials.

# Features of chronic inflammation

1. **Long duration:** Persists for weeks, months or years. (Days = ~~acute~~ in acute)
2. **Tissue destruction:** Greater than in acute inflammation (Induced by products of the inflammatory cells). * because it causes Fibrosis & scarring
3. **The inflammatory infiltrate** is a* mixture of mononuclear cells: Macrophages, lymphocytes and plasma cells.
4. **Productive rather than exudative:** Production of fibrous tissue through formation of granulation tissue.
5. Healing will occur by proliferation of large number of fibroblasts end with fibrosis. (Scar)

# Microscopical features of chronic inflammation:

**1. Infiltration by mononuclear cells:**

- Lymphocytes: B-lymphocytes → plasma cell → Ab.
  T-lymphocytes → sensitized → cytokines.
- Plasma cells : Antibody-producing cells.
- Macrophages: Blood monocytes when extravasated it became activated macrophages.
- Eosinophiles and mast cell: Specially presents in allergic reaction.

**2. Tissue destruction induced by infl. Cells.** ~~displacement of tissue type~~ Change in tissue type

**3. Healing by granulation tissue composed of macrophages, new blood vessels & fibroblasts.**

① ② ③

How we will know the type? → (If these found in the organ it will be chronic inflammation)

# 2. Granulomatous inflammation

**Granulomatous inflammation** is a distinctive pattern of chronic inflammation characterized by aggregates of activated macrophages with scattered lymphocytes.

These macrophages will be activated and modified
o be similar to epithelial cells so called:
Epithelioid cells.

* Granuloma is aggegation of ① + ② + ③

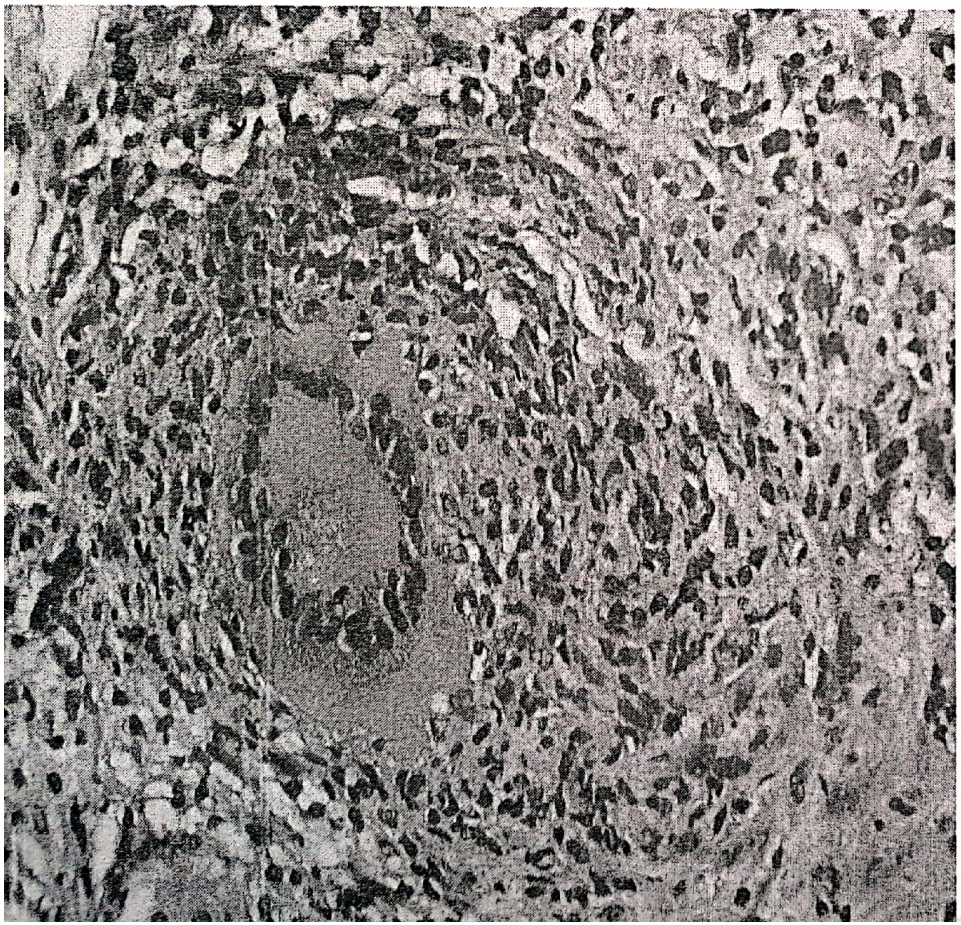
ı Many epithelioid cell united together forming:
Giant cell.

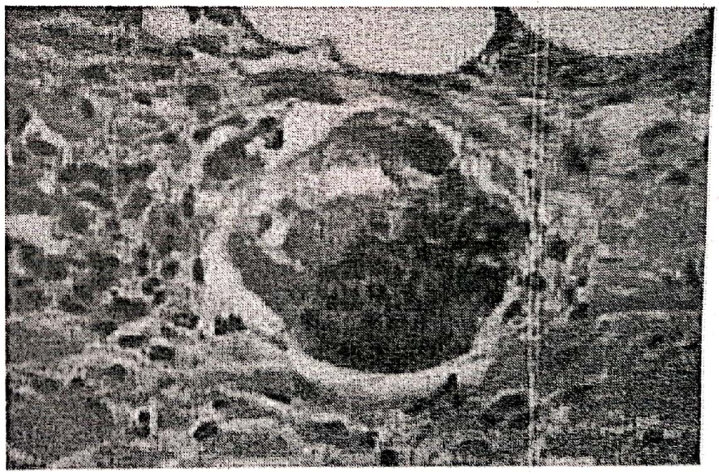
ı A dense membrane of connective tissue encapsulates
he lesion and isolates it.

# Giant cells

'he epithelioid cells coalesce, forming a large, multinucleated giant ell that attempts to surround the foreign Agent.

↳ upto 100 nucleu





# Examples of granulomatous inflammation:

imp → for specific diagnosis.

**Table 2–8** 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 histiocytes; plasma cell infiltrate; central cells are necrotic without loss of cellular outline |
| Cat-scratch disease | Gram-negative bacillus | Rounded or stellate granuloma containing central granular debris and neutrophils; giant cells uncommon |
| Sarcoidosis | Unknown etiology | Noncaseating granulomas with abundant activated macrophages |
| Crohn disease | Immune reaction against intestinal bacteria, self antigens | Occasional noncaseating granulomas in the wall of the intestine, with dense chronic inflammatory infiltrate |

*necrosis happens to some cells

# Macrophages

*inactive form of Macrophages

**It is the dominant cells of chronic inflammation derived from circulating blood monocyte after their emigration into the tissue forming tissue macrophage that scattered in all connective tissues.**

1- Liver: Kupffer cells
2- Spleen and lymph nodes: Sinus histiocytes
3- Central nervous system: Microglial cells
4- Lungs : Alveolar macrophages

# Differences between Activated macrophage and blood monocyte:

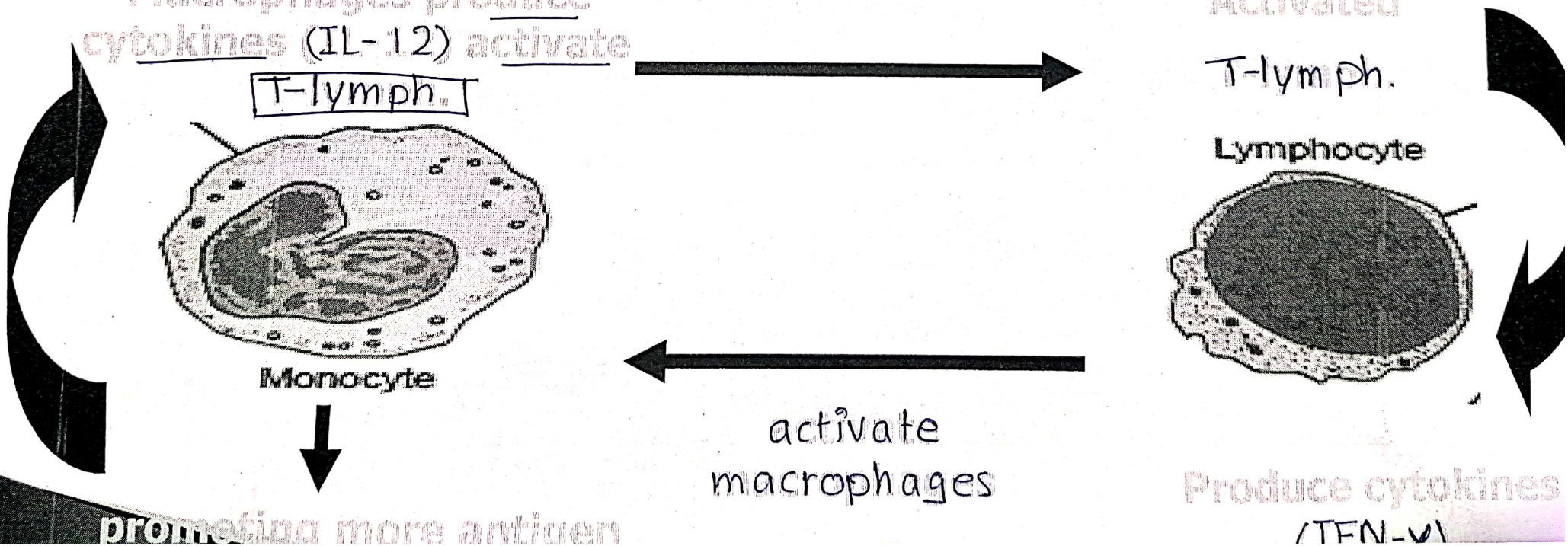
↳ The prepared type which is resposible for inflammation, & it's ready to engulf micro organisms.

- Increased cell size.
- Increased content of lysosomal enzymes.
- More active metabolism.
- Greater ability to kill ingested organisms.
- Longer half-lives.
- Greater capacity for phagocytosis than monocytes.

# Macrophage Lymphocytes interaction

**Lymphocytes & macrophages interact in a bidirectional way resulting in a cycle of cellular reactions that play an important role in chronic inflammation.**

* This cycle can be stopped by Anti-inflammatory mediators.



# Plasma cells

- Plasma cells produce antibodies directed against:
  1- Persistent antigens in the inflammatory site.
  2- Altered tissue components.

- In some strong chronic inflammatory reactions the accumulation of :-

  Lymphocytes
  Macrophages
  Plasma cells

- Have morphologic features of lymphoid organs, so appear as lymph nodes.

(Joint) in Rheumatoid Arthritis disease.
↳ contain large amount of chronic inflammatory cells.

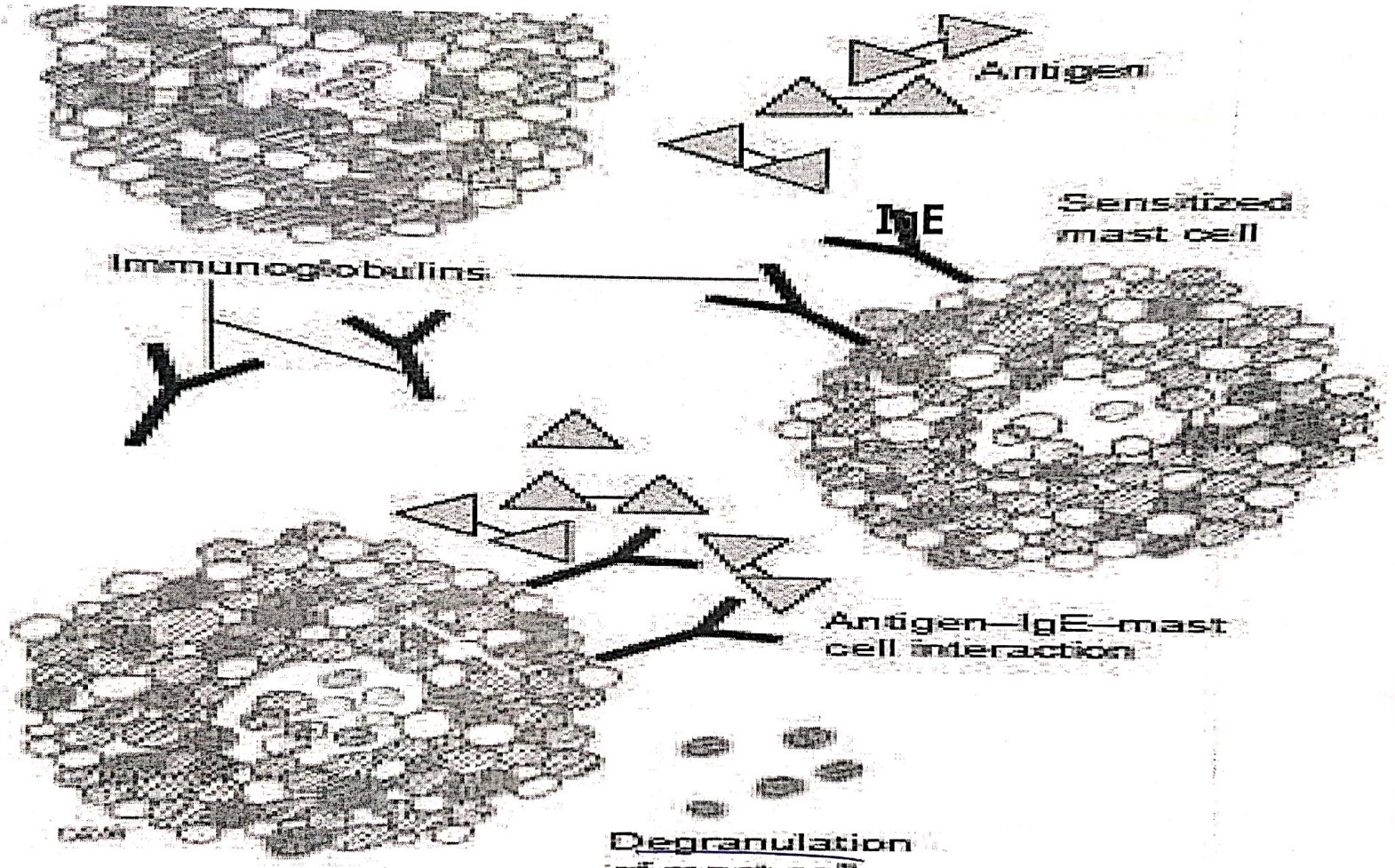
# Mast cells

* play role in acute & chronic inflammations

**Widely distributed in the connective tissues throughe the body. Seen in acute & chronic inflammation.**

- **<u>In individuals prone to allergic reactions:</u>**
- **Mast cells are bounded with IgE antibody specific fo certain antigens ⟹ IgE-armed mast cells ⟹ Re infl. Mediators ⟹ Early vascular changes of inflammation.**

- **IgE-armed mast cells are central players in <u>allergic reactions</u>, including <u>anaphylactic shock.</u>**




Degranulation of mast cell

Liberation of histamine, bradykinin leukotrienes, hepann, enzymes, chemotactic factors for neutrophils and eosinophils → each one have a specific role in (acute / chronic) inflammations.

- Vascular permeability
- Chemotaxis
- Smooth muscle contraction

**Chronic inflammation** is prolonged inflammation (weeks/months/years) that occurs when ongoing tissue destruction is present at the same time as ongoing tissue repair from:

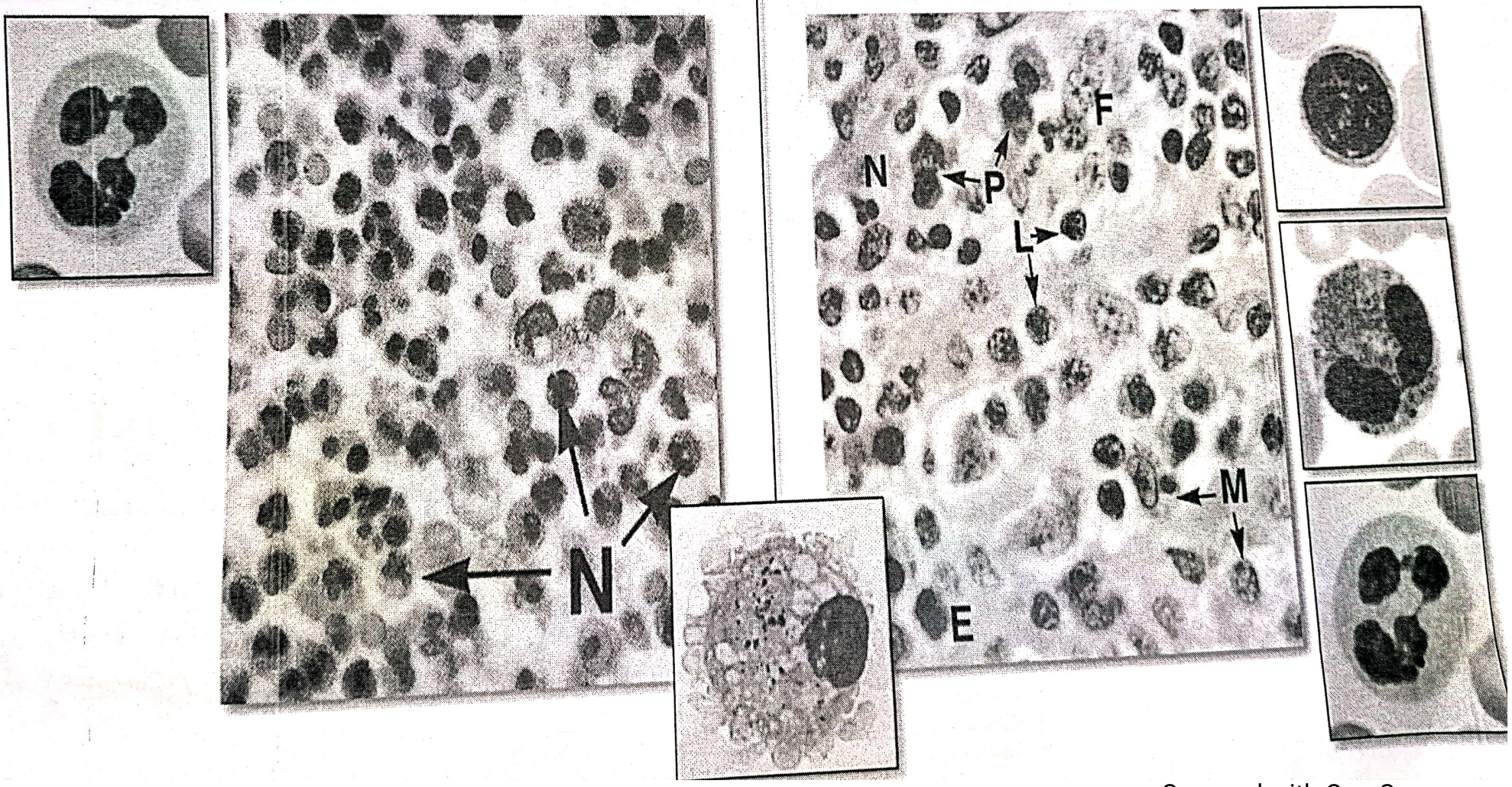
- Persistent infections
- Continuous exposure to toxins/irritants
- Autoimmune diseases

plasma cells criterea → eosinophilic cytoplasm with Extrinsic cartwheel nucleus

H&E stain

Acute inflammatory exudate

Chronic inflammatory exudate

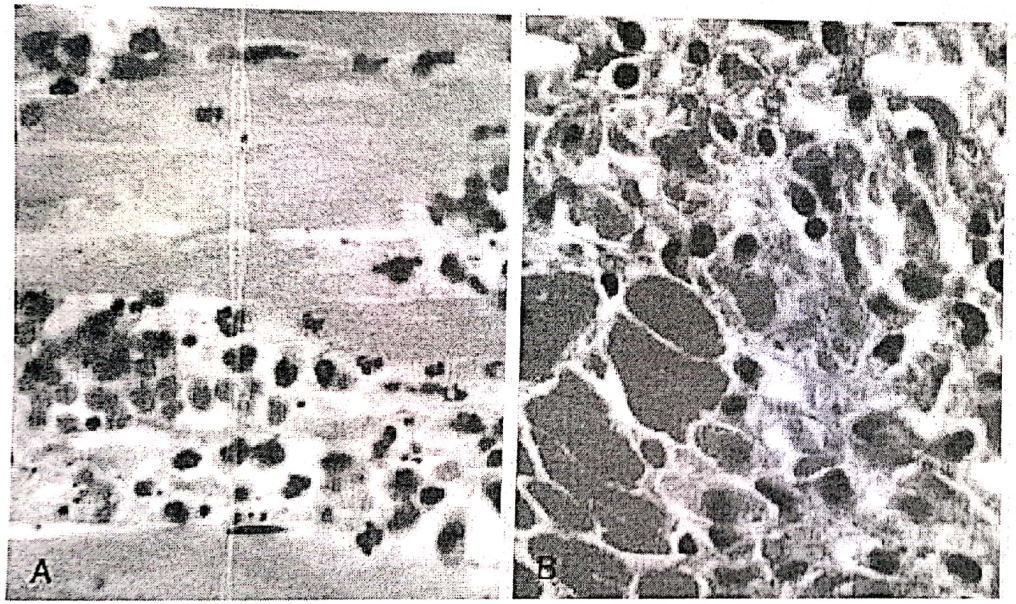


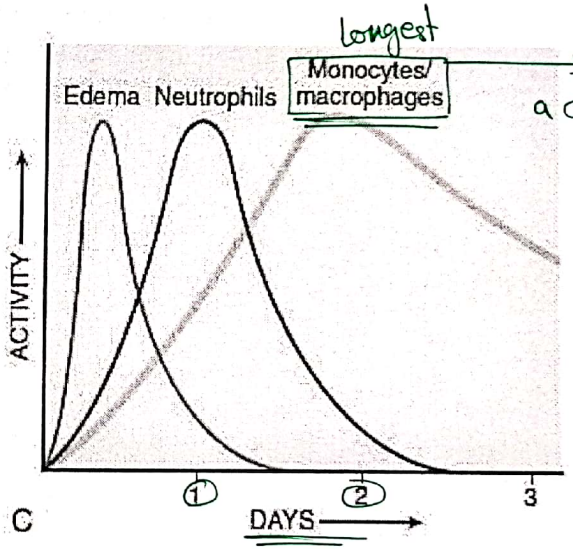
*muscles are parallel

*random (distruction of the normal tissue)

## Acute

## chronic




# Thank You