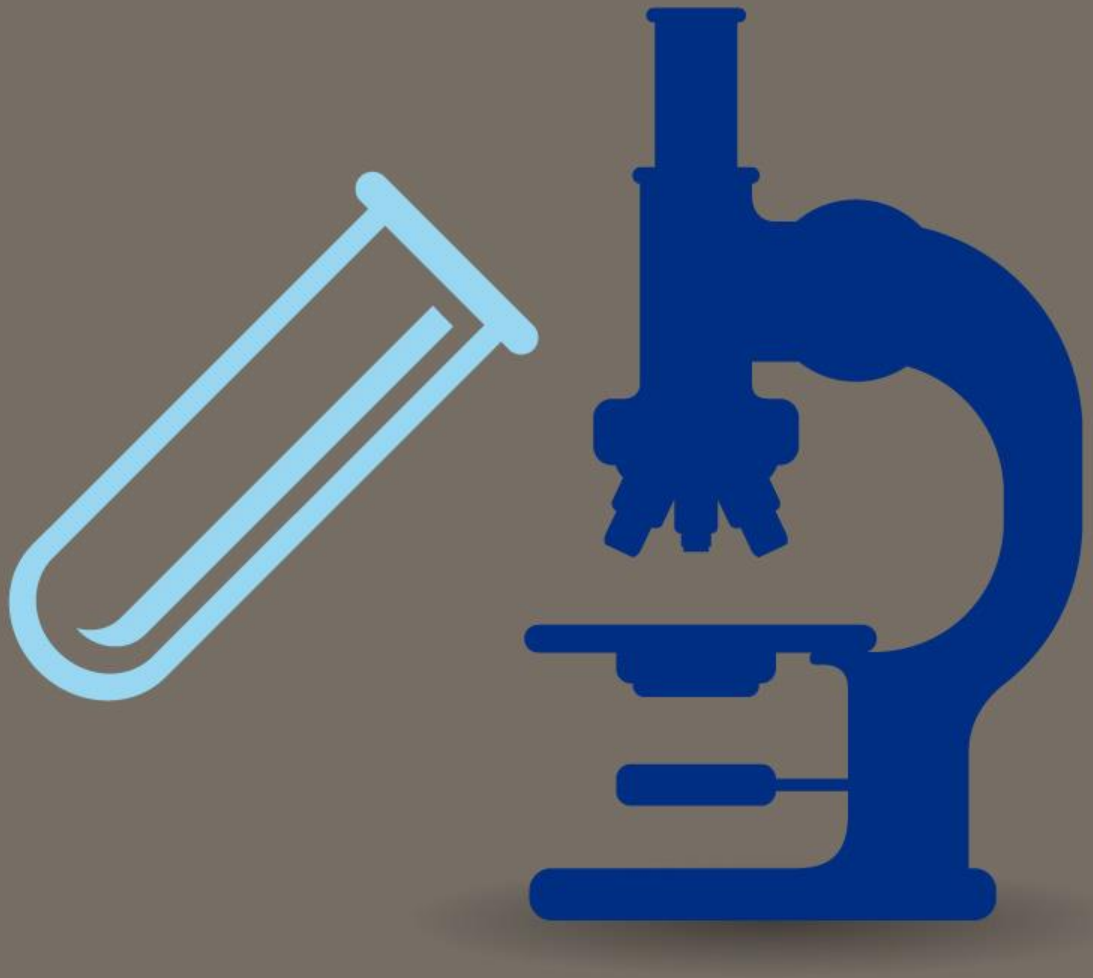


Inflammation

What is pathology?

Pathology is the study of diseases

Pathology is divided broadly into two main branches:

1. General pathology: Basic reaction of cells and tissues to abnormal stimuli that underlie all diseases.

2. Systemic pathology: Specific response of specialized organs or systems to diseases.

Inflammation

DEFINITION

- **Reaction of vascularized tissue to injury**

The aim of inflammation is to:

get rid of injurious stimuli from our body.

❖In doing so there may be destruction of tissues, collection of exudates.

Inflammation

Etiology:

1- Infection like bacteria, viruses and fungi

2- Physical irritation like cold and heat

3- Mechanical irritation like trauma

4- Chemical irritants like acide, alkalies.

5- Exposure to antigens leading to hypersensitivity reactions.

Types of inflammation:

1- Acute inflammation: rapid onset, short duration

2- subacute inflammation: between acute and chronic

3- chronic inflammation: gradual onset, longer duration

Stages of Acute Inflammation

I. Local tissue damage and release of CHEMICAL MEDIATORS.

II. The VASCULAR PHASE of inflammatory response.

III. The CELLULAR PHASE of inflammatory response.

IV. Local reaction of tissue HISTIOCYTES.

I- Local tissue damage and release of chemical mediators

Chemical Mediators

Definition:

Any messenger that acts on blood vessel, inflammatory cells or other cells to contribute to an inflammatory response.

Types:

A. Cell-derived :-

   e.g. histamine, serotonin, prostaglandins.

B. Plasma factors:-

   e.g. Bradykinin, fibrinogen, fibrin.

C. Bacterial products.

I- Local tissue damage and release of chemical mediators

Chemical Mediators

Effects:

- Vasodilatation
- Increased vascular permeability
- Chemotaxis
- Other effects as fever, leukocytosis and pain.

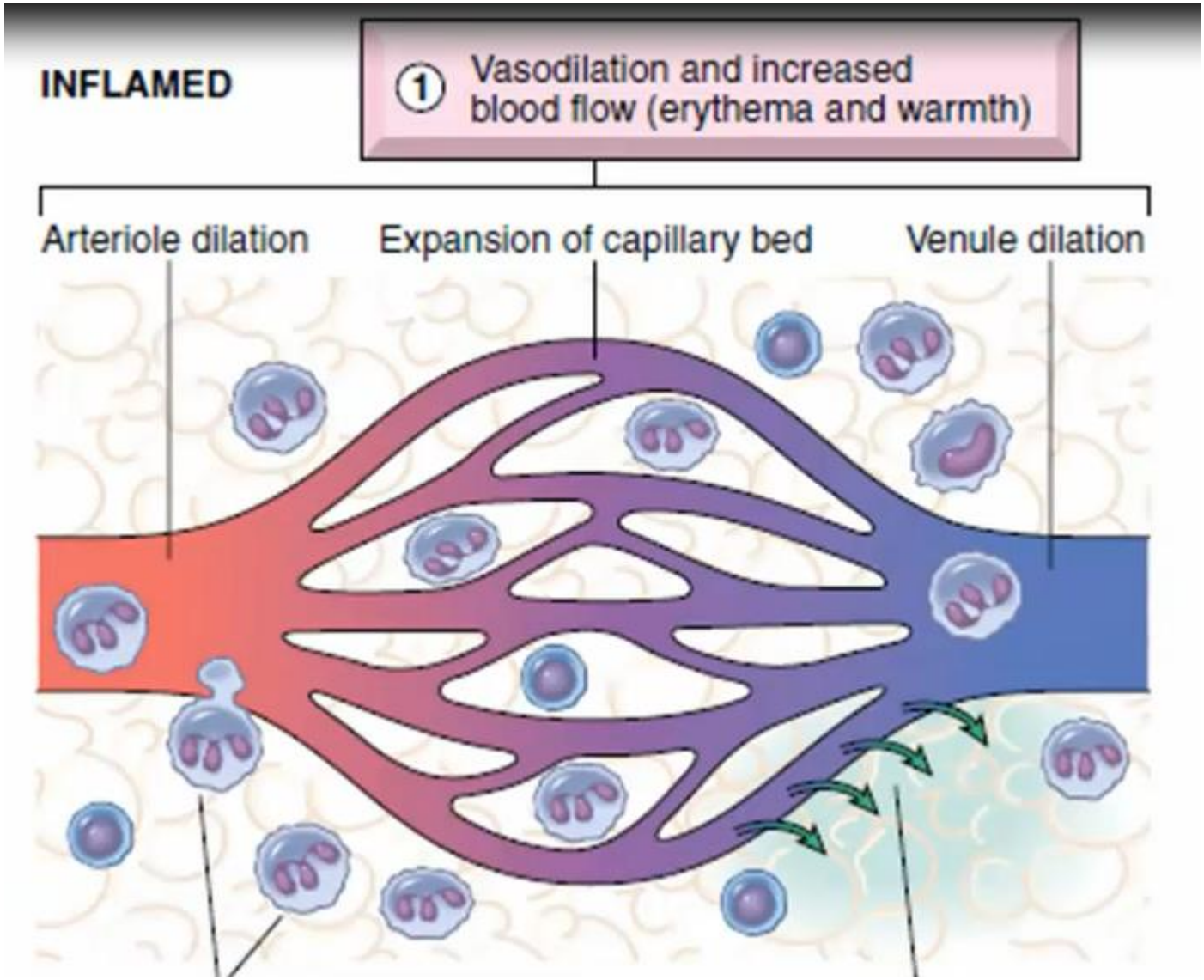
II- The vascular phase

A. **Transient vasoconstriction of the arterioles**

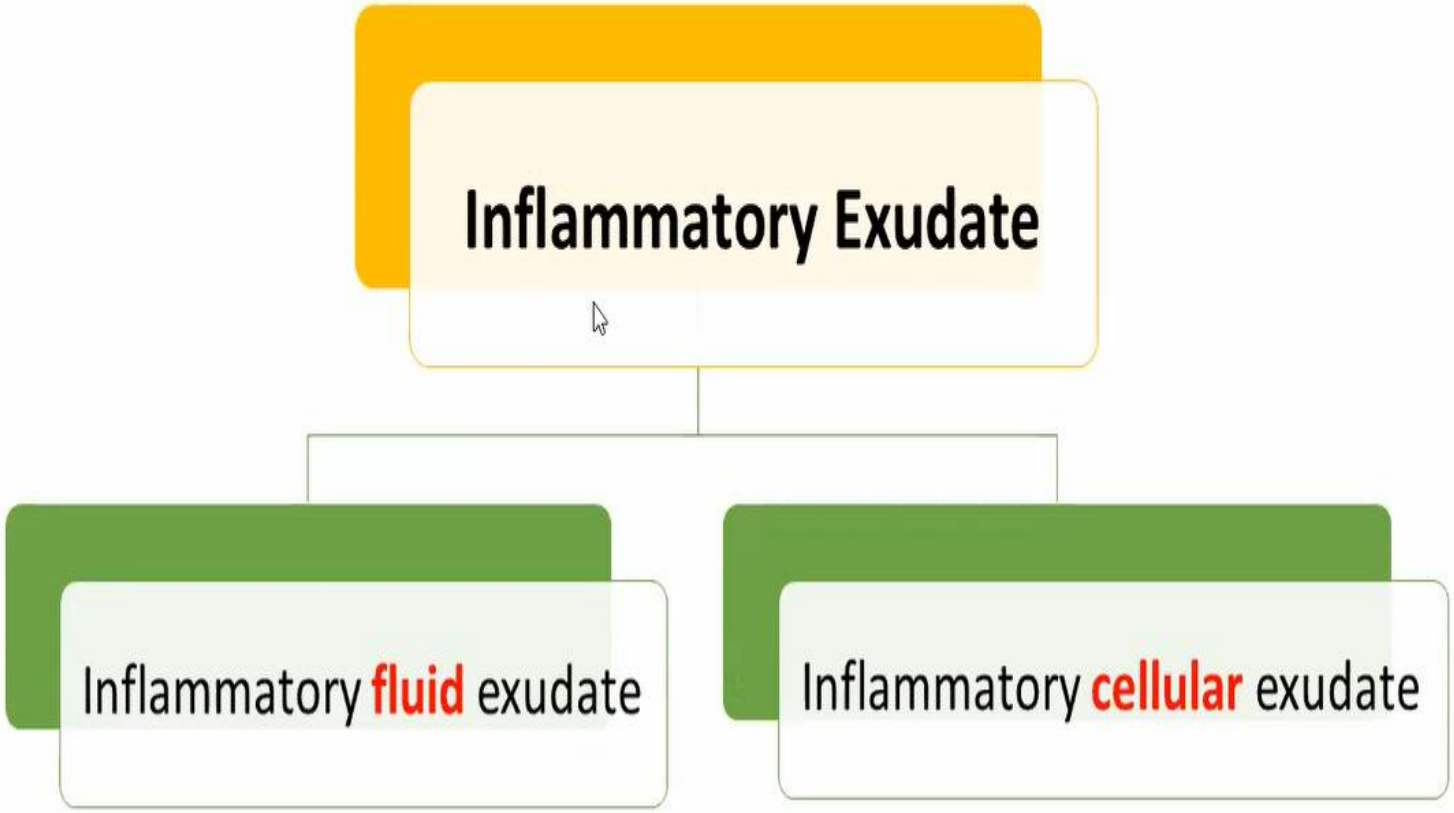
B. **Vasodilatation:-** → ++ blood flow → the inflamed area appears **Red & Hot**.

C. **Increased vascular permeability:** → formation of fluid **EXUDATE** (followed by cellular exudate) → tissue **Edema**.

D. **Vascular slowing** (**STASIS**):- local slowing of the circulation in the area → allowing for leukocyte exudation.


INFLAMED
1 Vasodilation and increased blood flow (erythema and warmth)
Arteriole dilation
Expansion of capillary bed
Venule dilation

The escape of fluid, proteins, and blood cells from the vascular system into the interstitial tissue or body cavities is known as ***Exudation***



Inflammatory Fluid Exudate

Mechanism of formation:

- Increased capillary hydrostatic pressure.
- Decreased intravascular osmotic pressure &
- Increased interstitial osmotic pressure

more fluid exudation & inflammatory tissue edema.

Transudate	Exudate
Low protein content < 3g/dl	High protein content > 3g/dl
Protein content mainly Albumin	Protein content mainly Fibrinogen
Does not clot	Undergoes clotting
Low specific gravity < 1015	High specific gravity > 1015
Poor cellularity	Rich in inflammatory cells

Inflammatory Fluid Exudate

Function:

1) Bringing **A**ntibodies to the inflamed area.
2) Fibrinogen content changes into fi**b**rin which helps to localize the infection and acts as a network for phagocytes and fibroblasts.
3) Carrying the **C**hemical mediators, nutrients & oxygen for inflammatory cells.
4) **D**ilution of bacterial toxins and chemical irritants.
5) **D**raining the products of inflammation.

Inflammatory Fluid Exudate

Fate:

- Fluid exudate is absorbed by lymphatics.
- Existing viable bacterial in the exudate may cause lymphangitis +/- lymphadenitis.

Acute Inflammation

III- The cellular phase

- There is an ***ACTIVATION OF LEUKOCYTES*** through interaction between *leukocyte receptors* and *chemical mediators*.

Steps of cellular phase:-

1- **Margination:** sticking of neutrophils, monocytes to endothelium

2- **Diapedesis:** Squeezing cells through endothelial pores

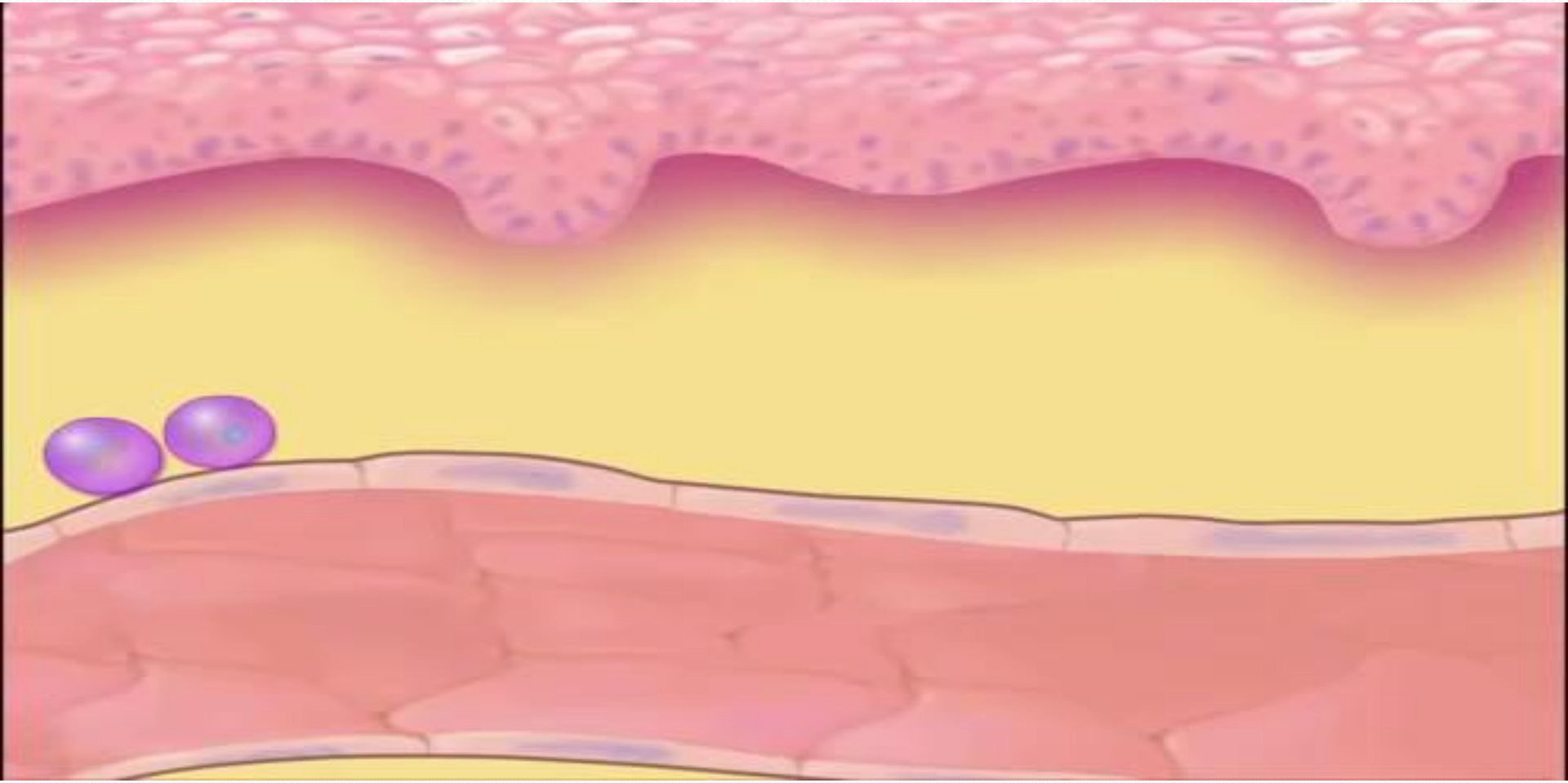
3- **Chemotaxis:** Breakdown of inflammed tissue attract phagocytes to the site of inflammation.

4- **Opsonization:** IgG and complement coat bacteria to make them tasty to phagocytes

5- **Phagocytosis:** coated bacteria then bind to receptors on phagocytic cells to be engulfed

- Steps:

Margination – diapedesis – chemotaxis – opsonization- phagocytosis



Phagocytosis involves three sequential steps

1. *Recognition* and *attachment* of the particle
2. Its *engulfment*, with subsequent formation of a phagocytic vacuole
3. *Killing* or *degradation* of the ingested material

Types of phagocytic cells in acute inflammation

1. *Neutrophils (polymorphonuclear neutrophils, PMNs):*

   Pus cells → release proteolytic enzymes → liquefaction of necrotic tissue and fibrin→ facilitating their drainage.

2. *Macrophages:*

   These include emigrating monocytes & tissue histiocytes.

Acute Inflammation

Systemic (general) changes

1. Leukocytosis: *Leukocyte count usually > 10000/mm³.*
2. Fever

Local cardinal signs & symptoms

1. Redness
2. Hotness
3. Swelling
4. Pain
5. Loss of function

local CARDINAL SIGNS OF INFLAMMATION:

There are five cardinal signs of inflammation:

1- redness due to increased blood flow and vasodilatation. Vasodilatation is produced by chemical mediators like histamine and kinin.

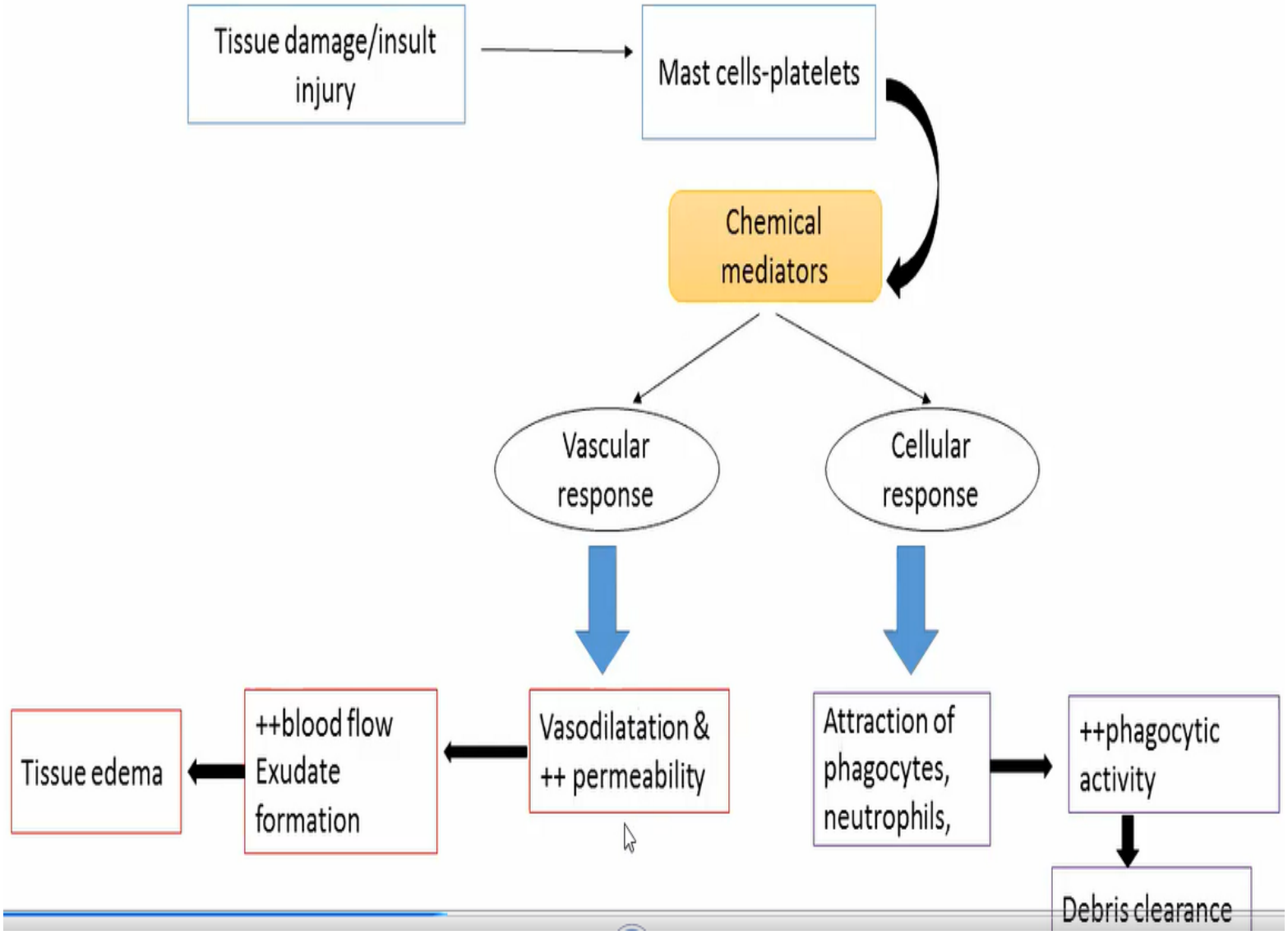
2- Hotness due to increased blood flow and vasodilatation.

3- pain due to synthesis of cytokine bradykinin and prostaglandin.

4- swelling due to accumulation of protein-rich fluid or exudate

5- loss of function due to combination of any of the above.

ACUTE INFLAMMATION



Types Of Acute Inflammation

I. Suppurative inflammation

Def.:- Acute inflammation with pus formation

Types:

1- Localized form

2- Diffuse form

II. Non-Suppurative inflammation

Def.:- Acute inflammation without pus formation.

Types:

1-Mild

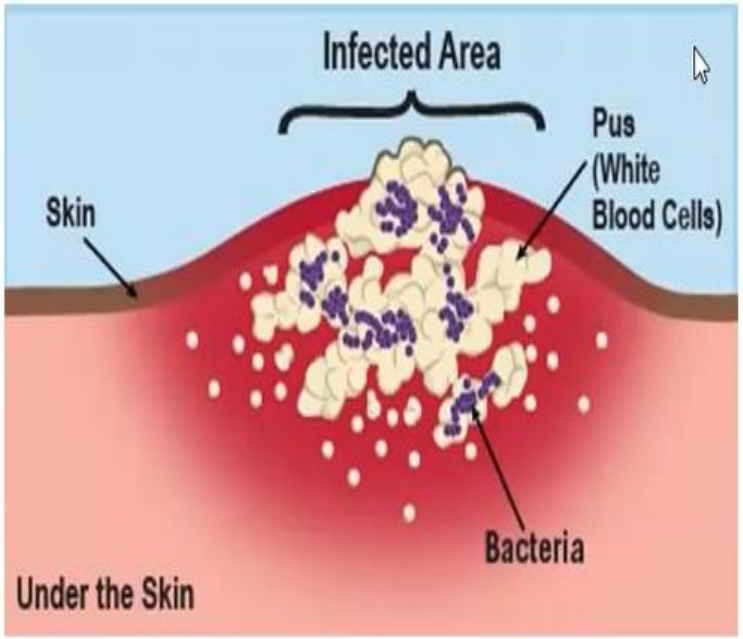
2- Severe

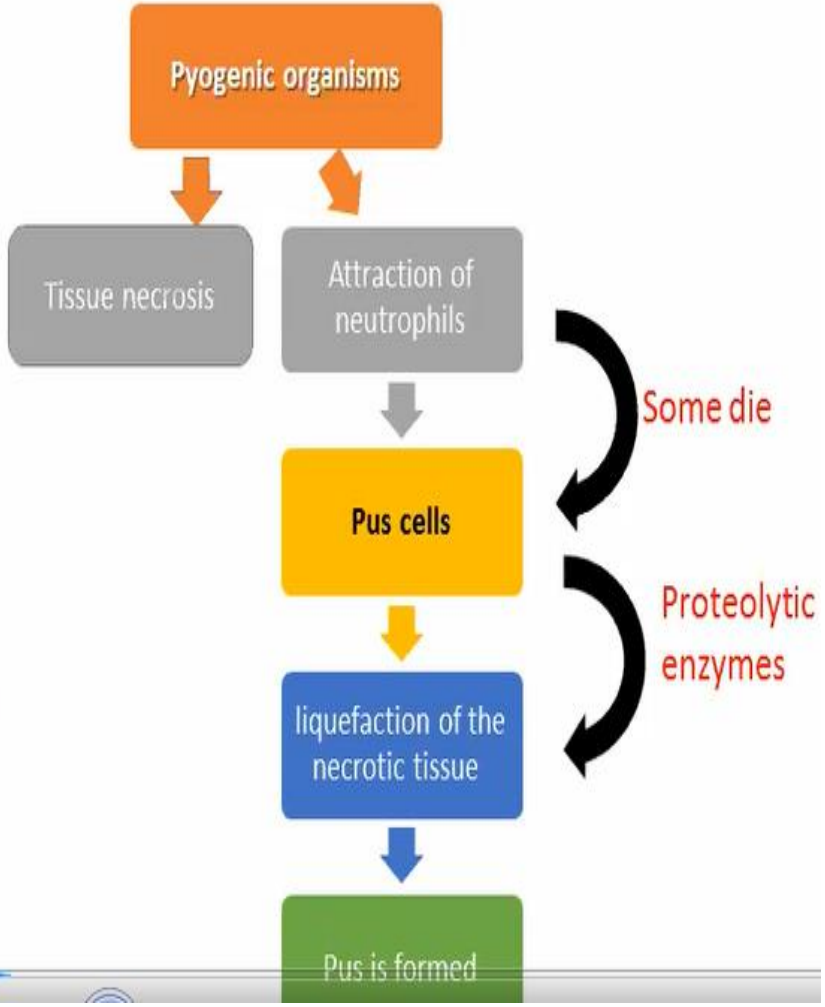
I. Suppurative inflammation

- It is acute inflammation characterized by pus formation.
- **Caused by**: pyogenic organisms such as: staphylococci, streptococci, gonococci, meningococci, and. E-coli.

I. Suppurative inflammation

Mechanism of pus formation:





I. Suppurative inflammation

Composition of pus:

1. Liquefied necrotic tissue.
2. Living and dead organisms.
3. Pus cells (dead neutrophils).
4. Inflammatory fluid exudate

Characteristics of pus

Thick, opaque, yellowish white fluid

Cream-like consistency

I. Suppurative inflammation

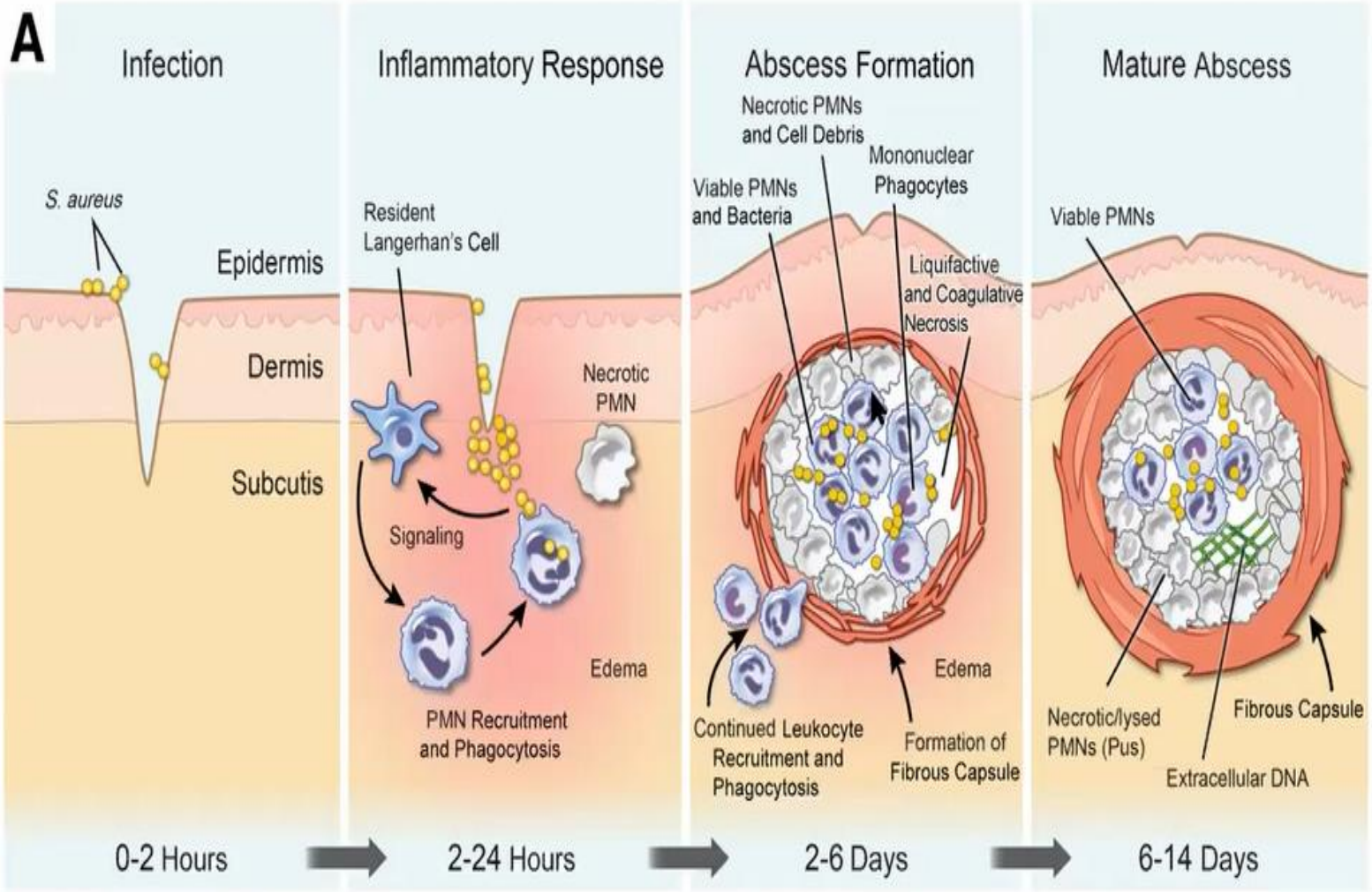
- **Types:**

	Localized suppurative inflammation	Diffuse suppurative inflammation
Etiology	commonly caused by Staphylococcus aureus bacteria	caused by Streptococcus hemolyticus bacteria
Mechanism	produce Coagulase Enzyme which helps in localization of the inflammation	produce Hyaluronidase Enzyme with spreading of the inflammatory process by liquefying the ground substance of the fibrous tissue.
Examples	Abscess, Carbuncle, Furuncle (boil)	cellulitis, diffuse septic peritonitis

Abscess

- **Definition:** it is a localized collection of pus in a cavity
- **Sites:** it can occur on the skin surface or in any internal organ such as lung, brain, kidney, or tonsils.

Steps Of Abscess Formation



Abscess

- **Fate:**
  - Small abscess **Absorption, healing**
  - Large abscess (if not surgically incised) → pointing and rupture, followed by healing.
- **Complications:**
  - Spread of infection:
  - Complications of healing **eg,. Ulcer, sinus**
  - Chronicity
  - Compression effects

II. Non Suppurative Inflammation

1. Serous inflammation — **Excess serous fluid poor in fibrin as in burns**
2. Fibrinous inflammation — **Excess fluid rich in fibrin as in pneumonia**
3. Catarrhal inflammation — **Inflammation of mucus membranes characterized by mucus secretion as in common cold**
4. Allergic inflammation — **Due to hypersensitivity characterized by esinophils as in bronchial asthma**
5. Hemorrhagic inflammation — **Excess RBCs due to vascular damage**
6. Necrotizing inflammation — **Excess necrosis associated with inflammation**

Chronic Inflammation

- **Features of chronic inflammation:**

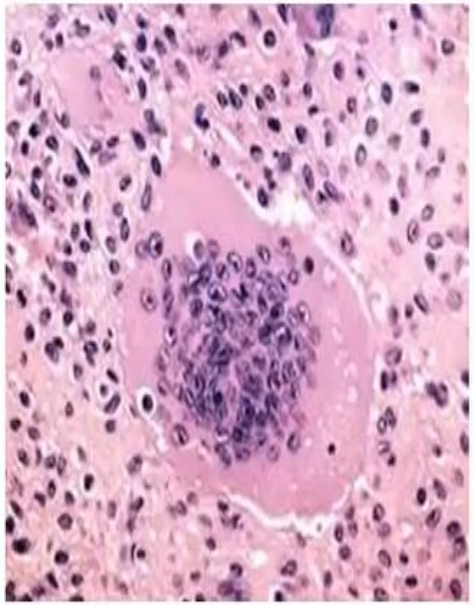
1. It is of gradual onset and prolonged duration (weeks or months) due to persistent aggressive stimuli.
2. It can follow acute inflammation that fail to eliminate the injurious agent or can be chronic right from the beginning.

Chronic Inflammation

In contrast to acute inflammation chronic inflammation is characterized by::

- Absence cardinal signs
- Absence of neutrophil emigration
- Usually scanty fluid exudate
- Milder vasodilatation
- **Tissue destruction**
- Attempts at repair by **fibrosis**.

Chronic inflammation



Inflammatory Cells of Chronic Inflammation are:

- **Macrophages:** are the dominant cells
- **Giant cells**: they are fused macrophages.
- **Lymphocytes**: T-lymphocytes, B- lymphocytes give
- **Plasma cells**
- **Eosinophils**

Types of chronic inflammation

I. Chronic **NON SPECIFIC** inflammation

- *Different irritants produce inflammatory reaction of the same microscopic picture.*
- *Usually follow acute inflammation*

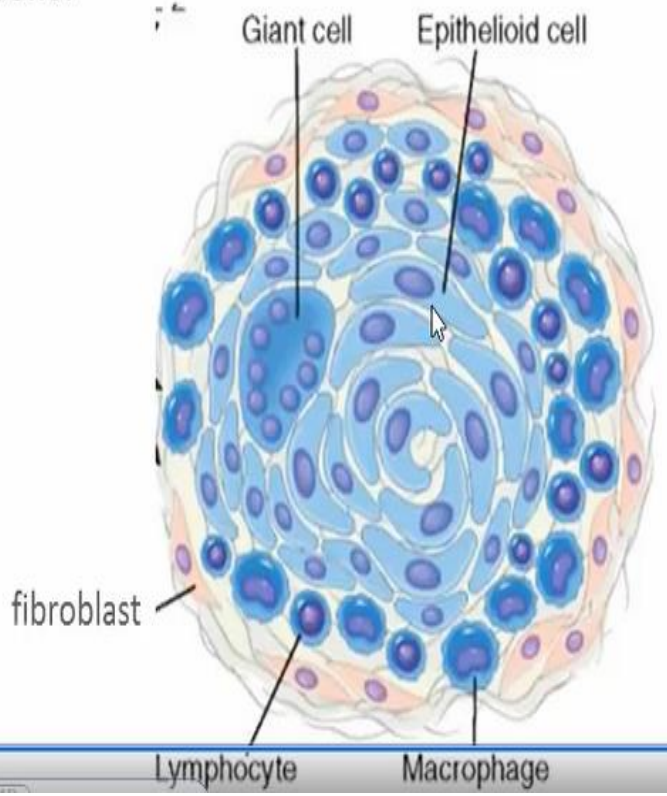
II. Chronic **SPECIFIC** inflammation (**Granulomatous**)

- *There are additional features specific for each irritant.*
- *The majority of chronic specific inflammation occur in the form of Granulomas.*

Granulomatous inflammation

Granuloma is a distinctive pattern of chronic inflammation

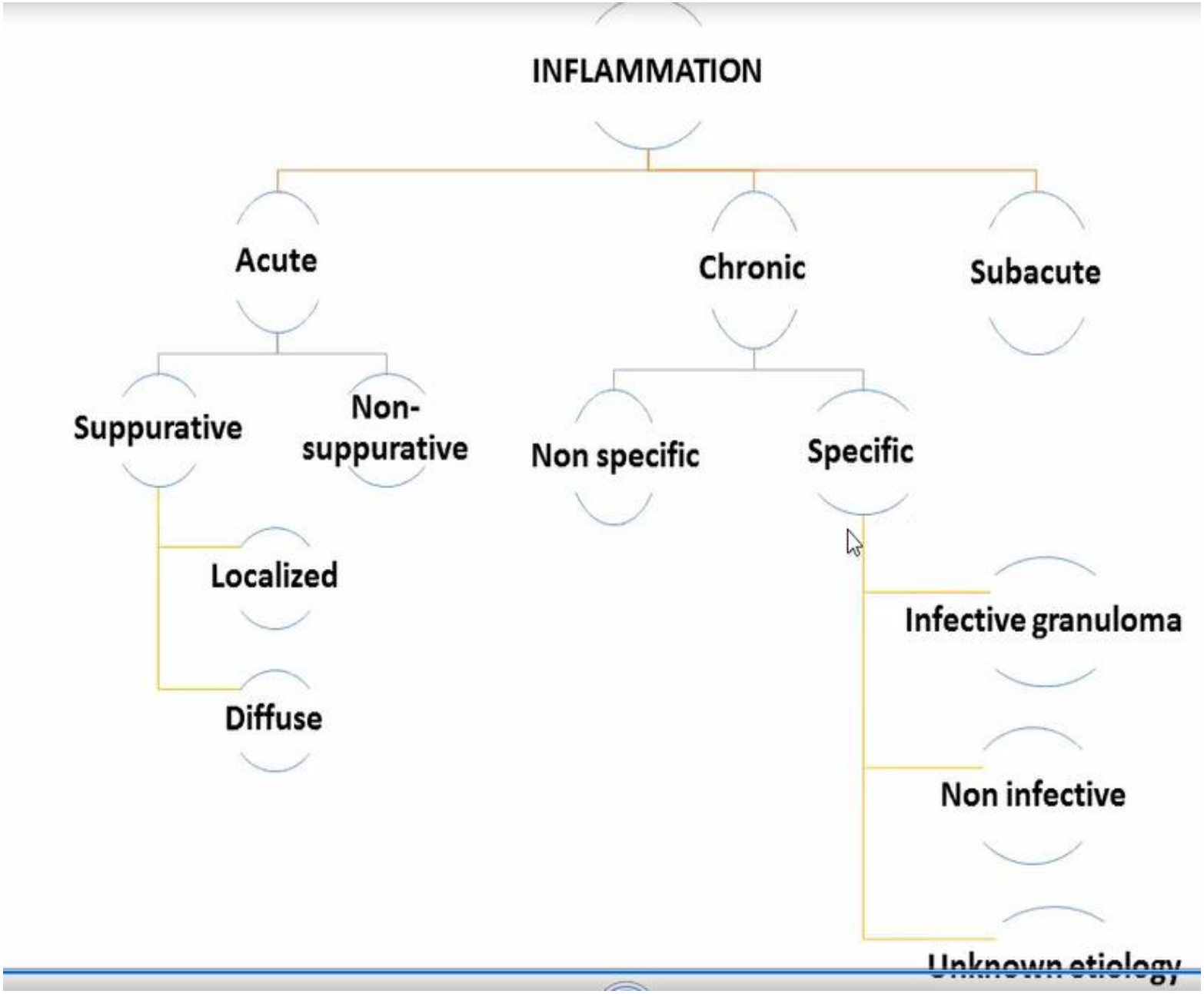
Microscopically: it is a circumscribed collection of **epithelioid cells** , multinucleated giant cells and rimmed at the periphery by lymphoid cells.



Granulomatous inflammation

Classification of granulomatous inflammation, according to the etiology:

Infectious granuloma	Non infective granuloma	Unknown etiology
Bacterial : Tuberculosis, Leprosy, Syphilis, Actinomycosis.	Silicosis, asbestosis Foreign body granuloma	Sarcoidosis Crohn's disease
Parasitic: Toxoplasmosis, Bilharziasis		
Fungi: Candidiasis		


INFLAMMATION
Acute
Chronic
Subacute
Suppurative
Non-suppurative
Non specific
Specific
Localized
Diffuse
Infective granuloma
Non infective
Unknown etiology


Thank you!