Inflammation-2

Lecture: 6

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Systemic Pathology
Second Grade

Lecture Outline:

- Morphologic patterns of acute inflammation
- Outcomes of acute inflammation
- Chronic inflammation
- Granulomatous inflammation
- Systemic Effects of Inflammation

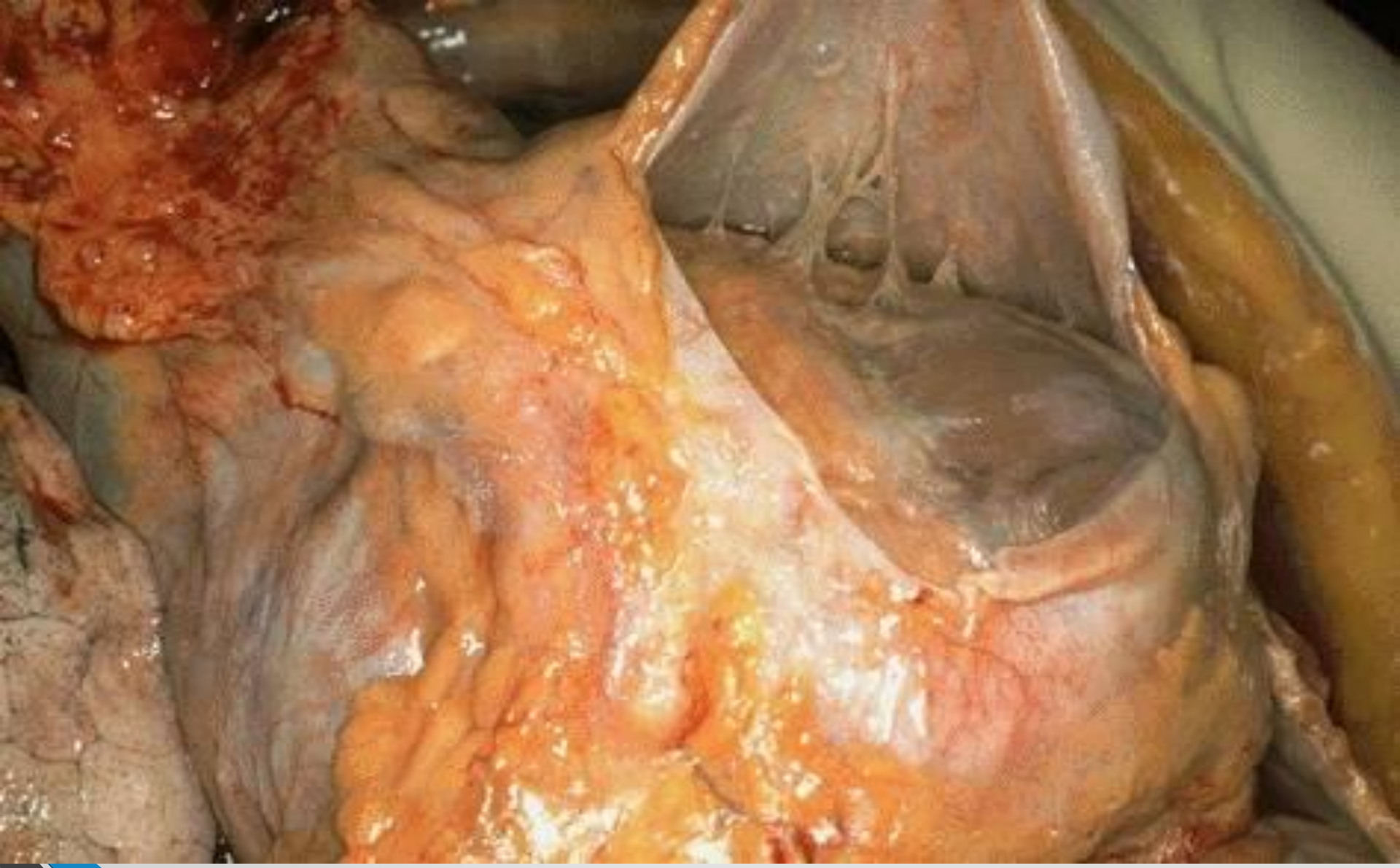
Morphologic patterns of acute inflammation

- 1. Serous
  - Watery, protein-poor effusion
  - E.g. blister
- 2. Fibrinous
  - Fibrin accumulation
  - Either entirely removed or becomes fibrotic
  - E.g. fibrinous pericarditis

- **3. Suppurative**
  - Presence of pus
  - Often walled-off if persistent
  - E.g. acute appendicitis
- **4. Ulceration**
  - Necrotic and eroded epithelial surface
  - Underlying acute and chronic inflammation
  - Trauma, toxins, vascular insufficiency
  - E.g. peptic ulcer

Serous inflammation

Purulent inflammation



Fibrinous inflammation

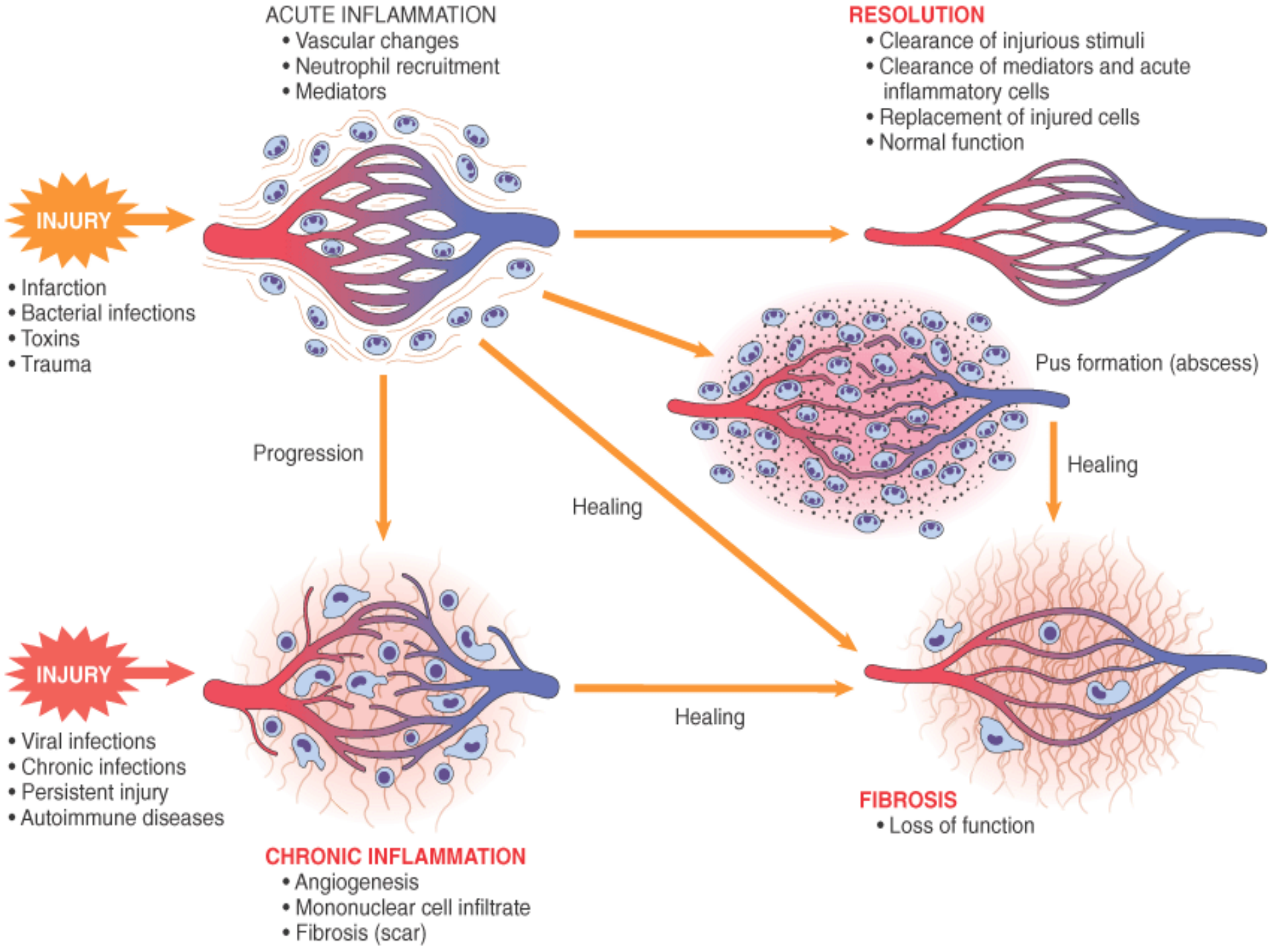
Ulcerative inflammation

Outcomes of acute inflammation

- 1. Complete resolution
  - Little tissue damage
  - Tissue capable of regeneration
- 2. Scarring (fibrosis)
  - In tissues unable to regenerate
  - Excessive fibrin deposition organized into fibrous tissue

- 3. Abscess formation occurs with some bacterial or fungal infections.

- 4. Progression to chronic inflammation


ACUTE INFLAMMATION
• Vascular changes
• Neutrophil recruitment
• Mediators
RESOLUTION
• Clearance of injurious stimuli
• Clearance of mediators and acute inflammatory cells
• Replacement of injured cells
• Normal function
INJURY
• Infarction
• Bacterial infections
• Toxins
• Trauma
Pus formation (abscess)
Progression
Healing
Healing
INJURY
• Viral infections
• Chronic infections
• Persistent injury
• Autoimmune diseases
Healing
FIBROSIS
• Loss of function
CHRONIC INFLAMMATION
• Angiogenesis
• Mononuclear cell infiltrate
• Fibrosis (scar)

Chronic inflammation

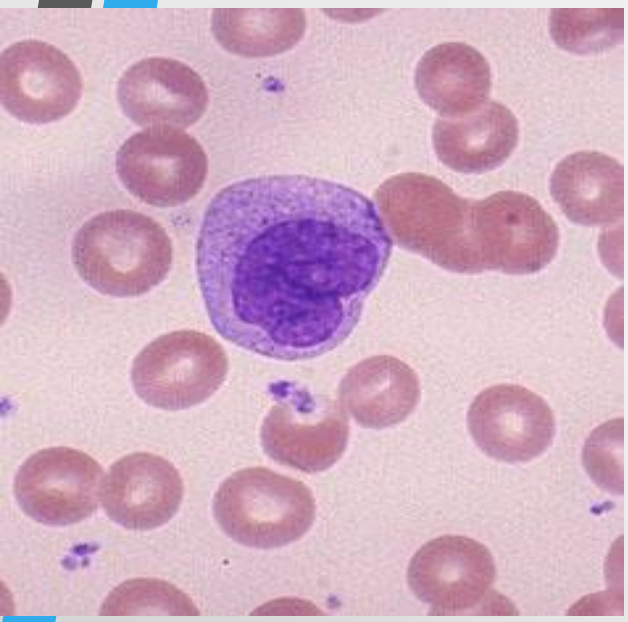
- Chronic inflammation is of longer duration (days to years) and is characterized by:
- mononuclear inflammatory cell infiltration,
- vascular proliferation, and
- scarring (fibrosis).

Chronic inflammation

- Mononuclear cell infiltration e.g Lymphocyte, macrophage, plasma cell
- Tissue destruction by inflammatory cells
- Attempts at repair with fibrosis and angiogenesis (new vessel formation)
- Causes:
  - Persistent injury or infection (ulcer, TB)
  - Prolonged toxic agent exposure (silica)
  - Autoimmune disease states (RA, SLE)

The dominant cellular player in chronic inflammation is the tissue macrophage

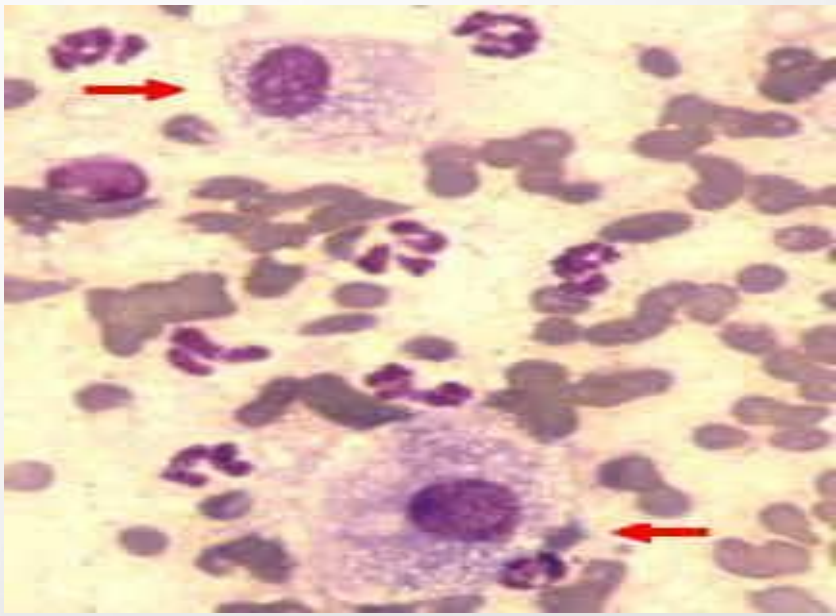
Blood monocyte



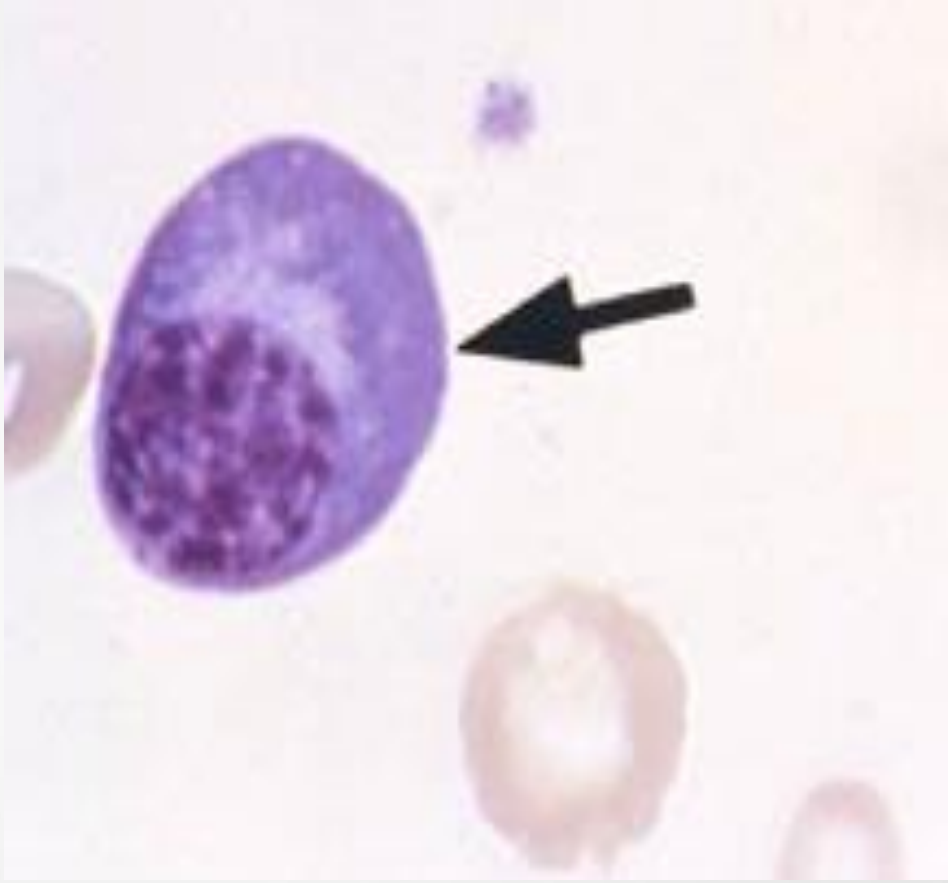
migrate into tissue within 48 hours after injury

and differentiate

Tissue macrophage (RES)



- **Kupffer cell (liver)**
- **Microglia (CNS)**
- **Histiocytes (spleen)**
- **Alveolar macrophages (lung)**



Lymphocyte

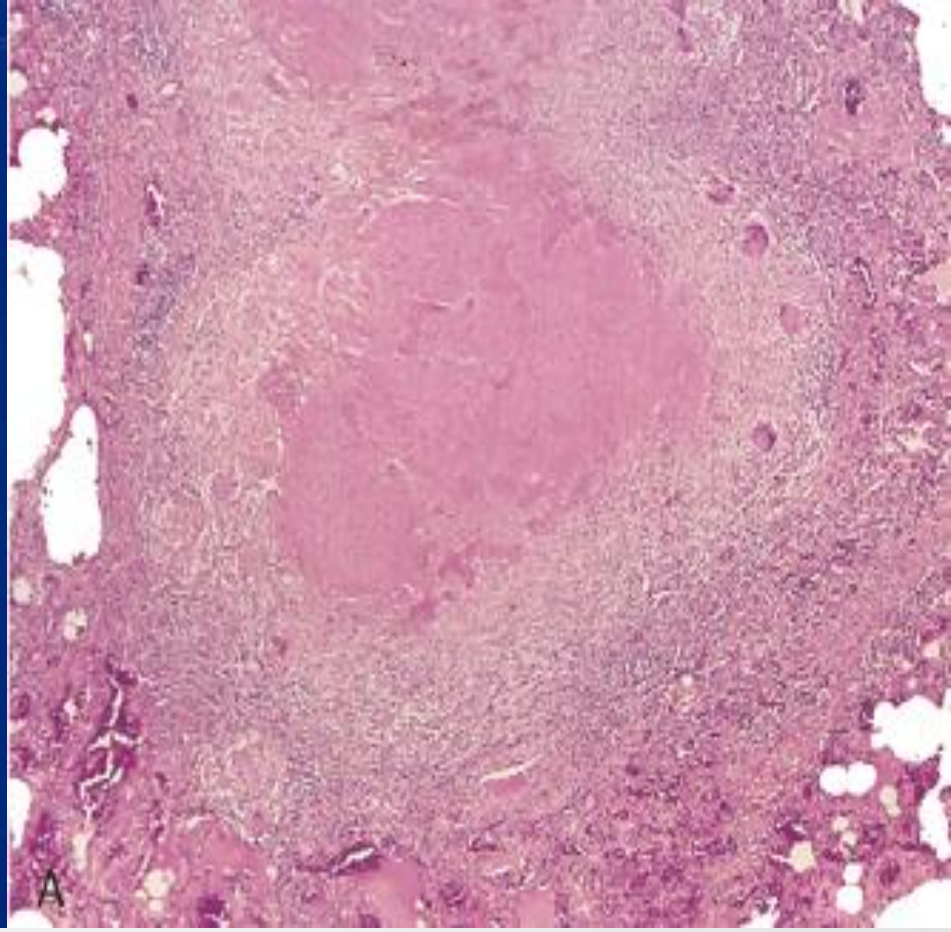
Plasma cell

*** however mast cells and eosinophils are as well involved in chronic allergic diseases**

Granulomatous Inflammation

It is a distinctive form of chronic inflammation characterized by granuloma formation which is nodular collection of epithelioid cells (activated macrophage) surrounded by a collar of lymphocytes. Epithelioid macrophage may fuse to form multinucleate giant cells and central necrosis may be present e.g. in tuberculosis.

Gross Appearance Lung



caseating necrosis

Tuberculosis

Systemic Effects of Inflammation

It is called acute phase response or systemic inflammatory response syndrome (SIRS) which represent responses to cytokines produced either by bacterial products or by other inflammatory stimuli and include the following clinical and pathological changes:

1. **Fever.**
2. **Acute Phase Protein: these are plasma protein synthesized in liver and increases in response to inflammatory stimuli e.g C-reactive protein.**
3. **Leukocytosis**
   - **Elevated white blood cell count:**
     Bacterial infection (neutrophilia).

     Parasitic infection (eosinophilia).

     Viral infection (lymphocytosis).

4. Other manifestations include:

- increased pulse and blood pressure.
- decreased sweating.
- rigors, chills.
- anorexia.
- malaise.

5. In severe bacterial infections may lead to septic shock.

Features	Acute Inflammation	Chronic Inflammation
Duration	Short (hours - days)	Long (weeks-months)
Onset	Acute	Insidious
Cardinal clinical signs	Present	Absent
Fluid exudation & edema	Present	Absent

Vascular changes	**Active vasodilation Increased permeability**	**New vessel formation (granulation tissue)**
Inflamma-tory cells	**Neutrophils**	**macrophages, Lymphocytes, plasma cells, fibroblasts .**
Fibrosis	**Absent**	**Present**

THANK YOU