INFLAMMATION

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Definition of INFLAMMATION

- Local physiological response of a vascularized tissue to injury

- **Function**: Often beneficial, but sometime harmful:
- 1. Dilute, destroy and wall off injurious agents
- 2. Start process of healing

Causes of Inflammation

1. **Infections:** e.g., bacterial, viral, parasitic, fungal etc.
   - **Viruses** lead to death of individual cells by intracellular multiplication.
   - **Bacteria** release specific exotoxins-chemicals synthesized by them which specifically initiate inflammation-or endotoxins, which are associated with their cell walls.
   - Some organisms cause immunologically-mediated inflammation through hyper-sensitivity reactions such as parasitic infections and tuberculosis
2. **Physical agents**: e.g., trauma, heat, cold, radiation, etc
3. **Chemical agents**: e.g., acid, alkali, drugs, etc.
4. **Hypersensitivity:** e.g., rheumatic fever, SLE, RA….

Cardinal signs of inflammation

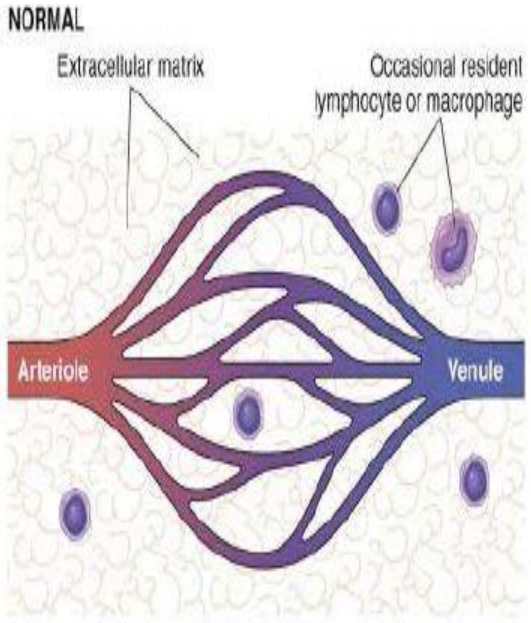
- **Heat** (Calor): vasodilation
- **Swelling** (Tumor): exudate
- **Redness** (Rubor): vasodilation
- **Pain** (Dolor): prstaglandin, bradykinin, nerve compression
- **Loss of function** (Functio laesa): pain & swelling

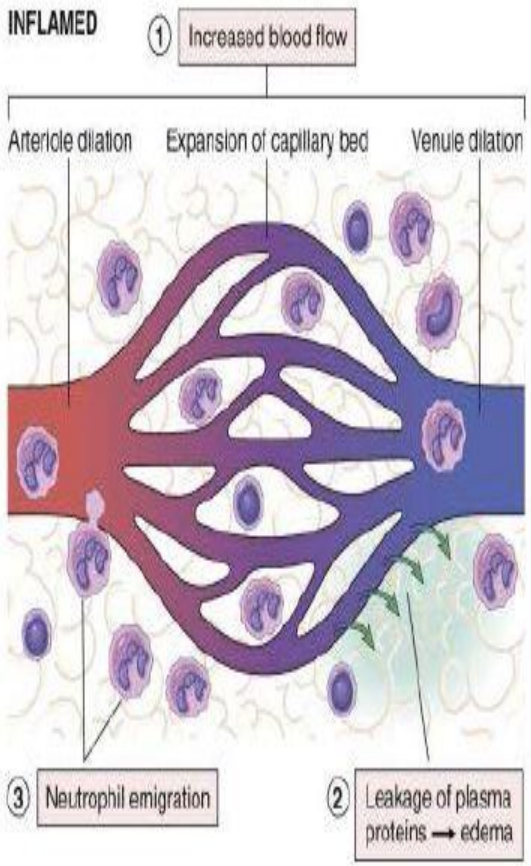
Components involved in inflammation

1. Blood vessels
2. Cells
    1. Circulating N, M, L, E, B, & plasma cells
    2. Connective tissue, mast cells, fibroblasts, M & L
3. Plasma & plasma proteins
4. Extracelluar matrix

    Collagen, elastic tissue, adhesive glycoproteins, protoglycans & basement membrane

Major components of inflammation





- Vascular changes
  - Vasodilation
  - Vascular permeability
  - Increased adhesion of white blood cells
- Cellular events
  - Recruitment and activation of neutrophils (polymorphonuclear leukocytes) and monocytes

Types of inflammation

1. Acute inflammation

- Short duration: hours -days- weeks
- Exudative fluid (protein rich fluid + infl cells + debris)
- Main inflammatory cells
  - N & M

2. Chronic inflammation

- Long duration: months - years
- Fibrosis (indurative)
- Main inflammatory cells
  - L, M, plasma cells + fibroblasts & endothelial cells

Exudate

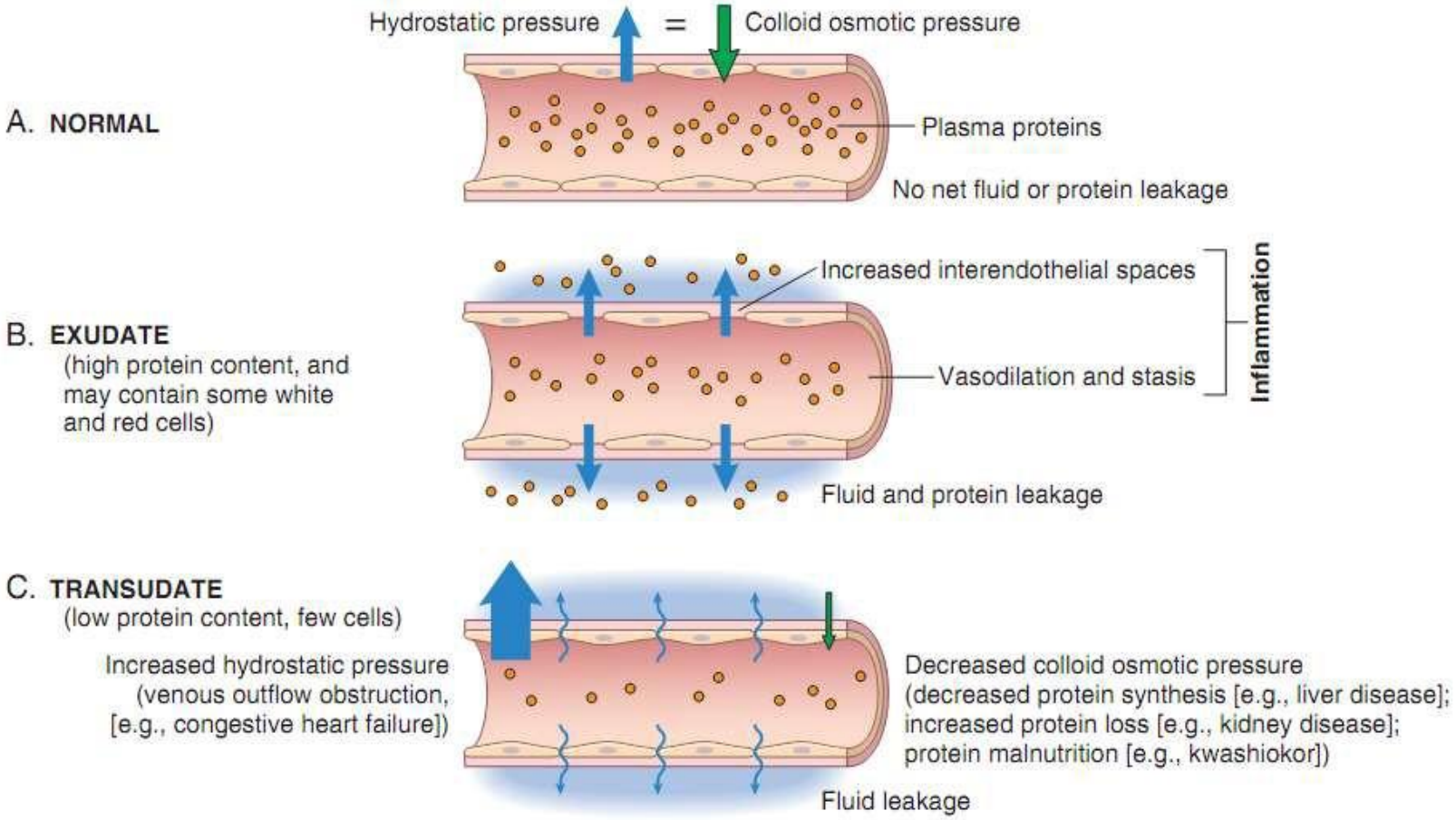


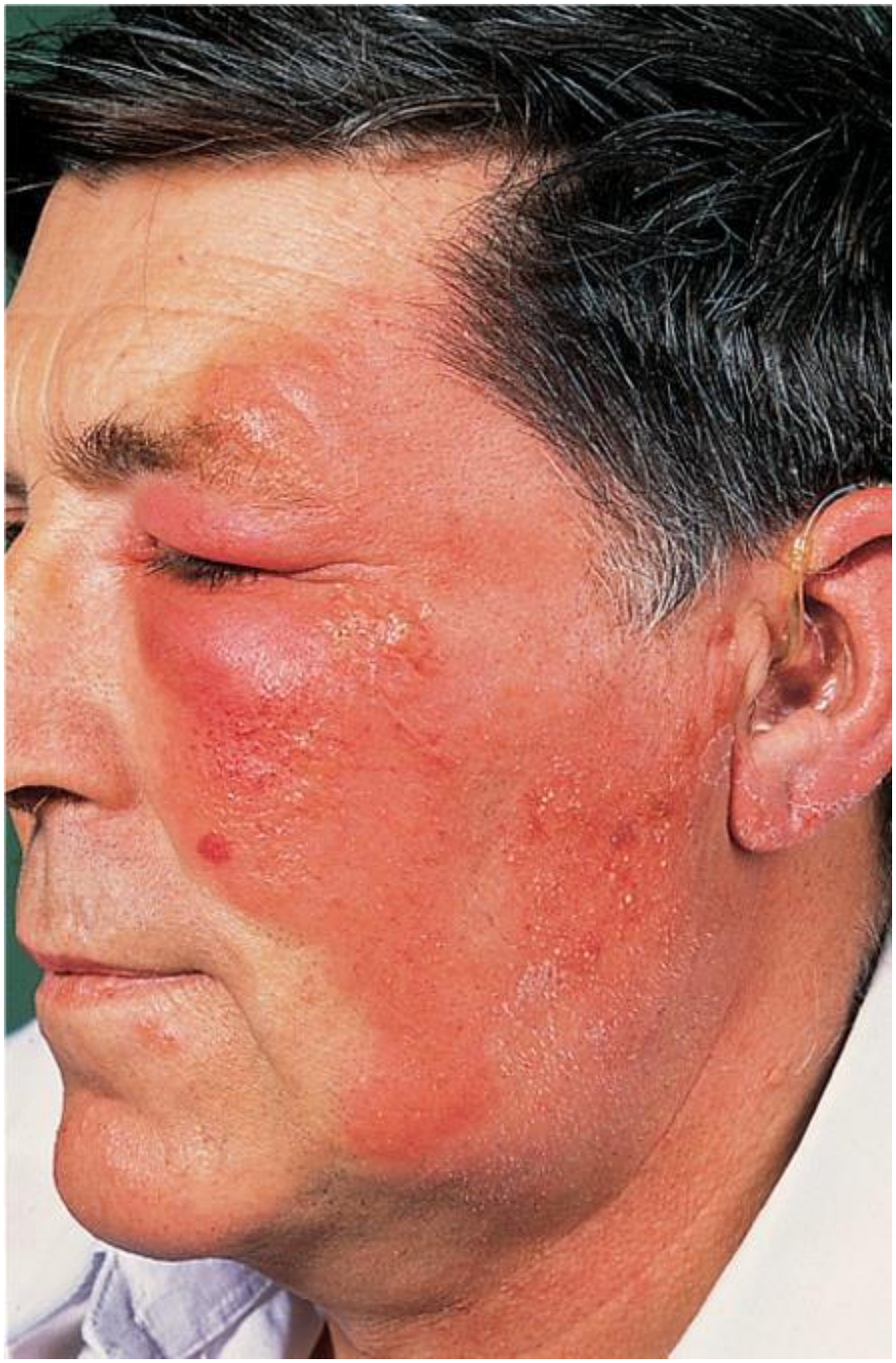
Fig. 3.2 Formation of exudates and transudates. (A) Normal hydrostatic pressure *(blue arrow)* is about 32 mm Hg at the arterial end of a capillary bed and 12 mm Hg at the venous end; the mean colloid osmotic pressure of tissues is approximately 25 mm Hg *(green arrow)*, which is equal to the mean capillary pressure. Therefore, the net flow of fluid across the vascular bed is almost nil. (B) An exudate is formed in inflammation because vascular permeability increases as a result of retraction of endothelial cells, creating spaces through which fluid and proteins can pass. (C) A transudate is formed when fluid leaks out because of increased hydrostatic pressure or decreased osmotic pressure.

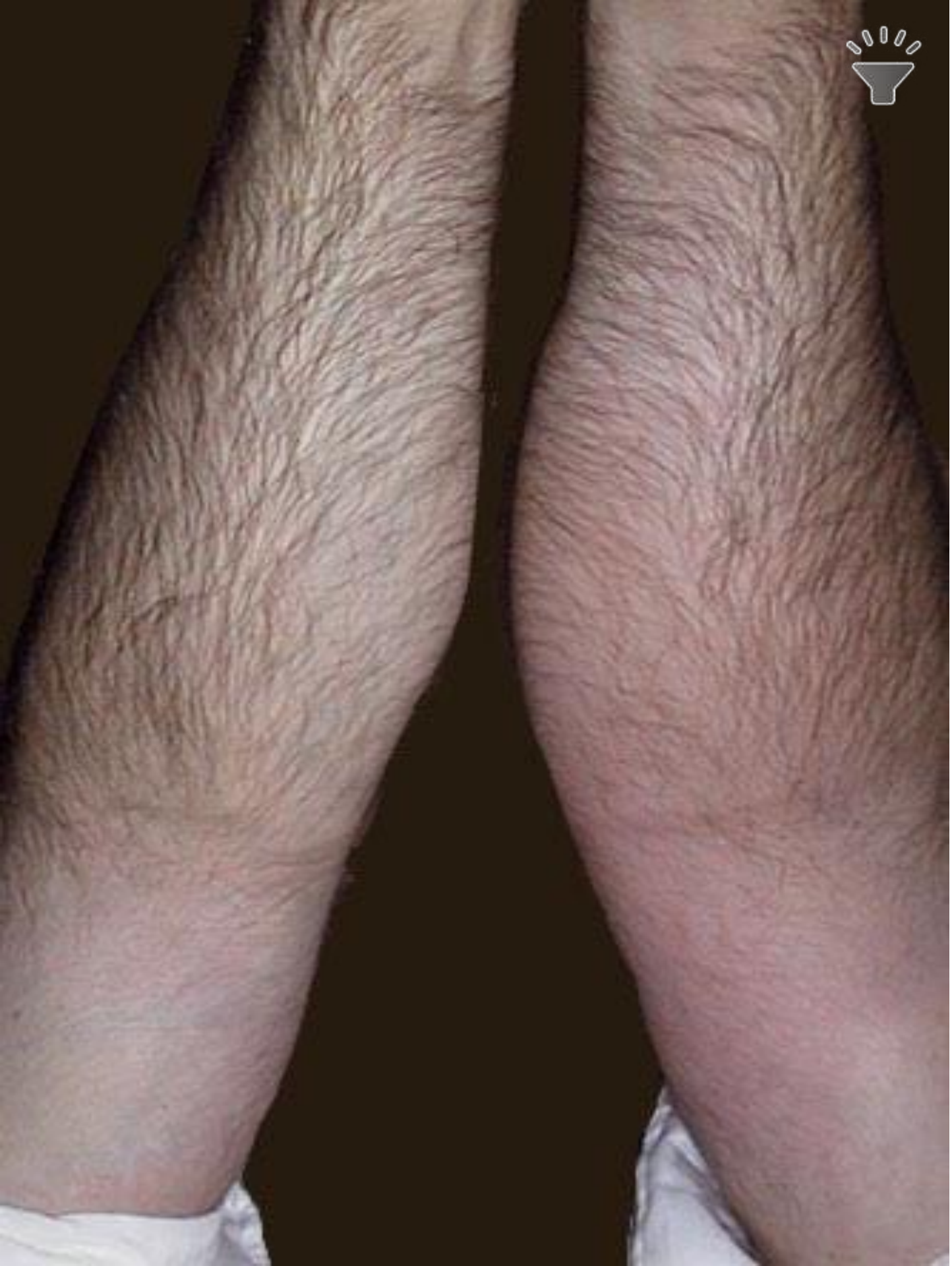
Exudate

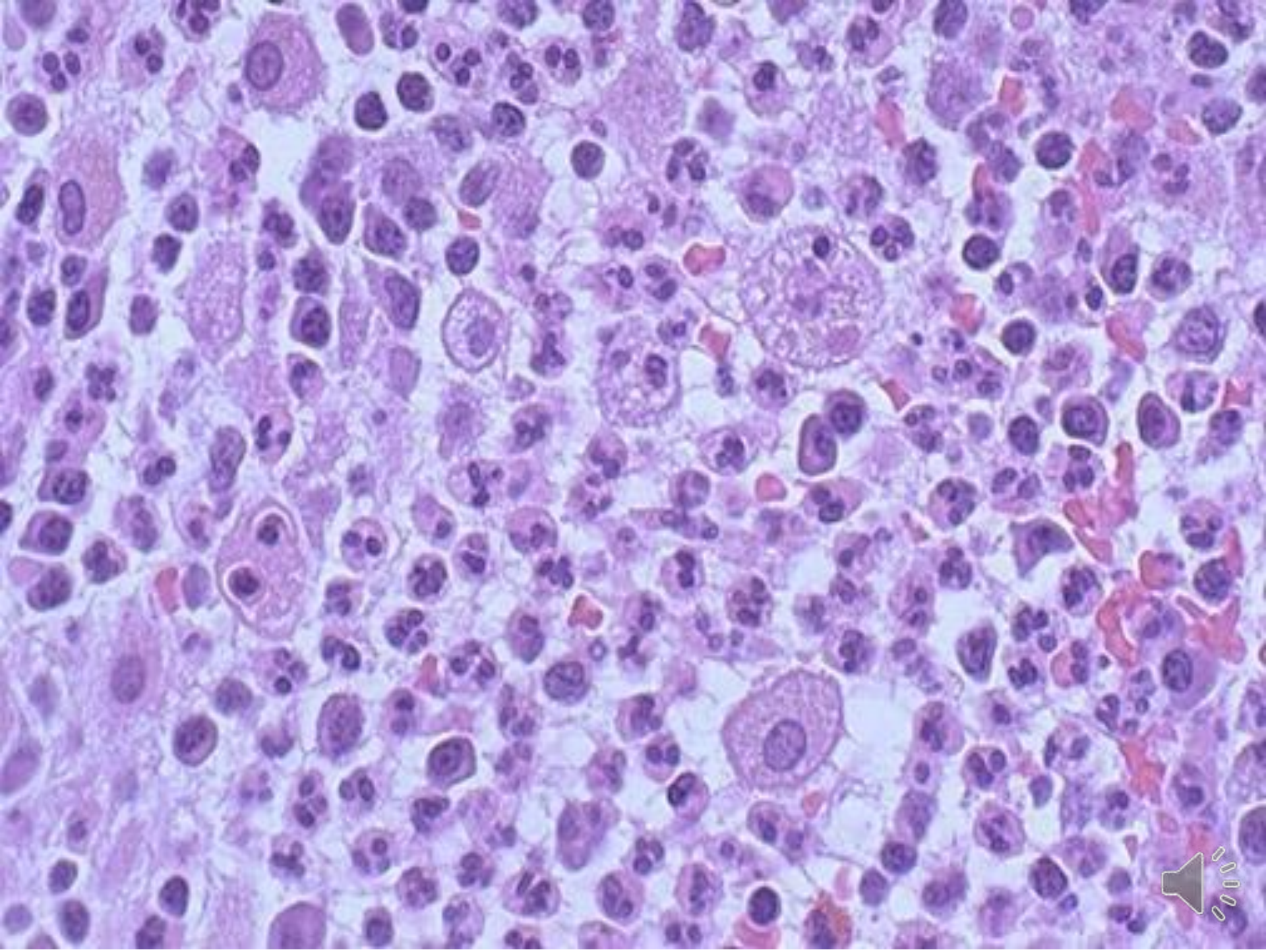
- **Definition**: extracellular fluid rich in proteins & cells. Due to increase vascular permeability induced by chemical mediators and due to the direct damage of the vessels.

- ***Consist of:***
  1. Fluid rich in plasma proteins
  2. Fibrin
  3. Cells: Neutrophils, macrophages, eosinophils, few lymphocytes & red blood cells
  4. Debris

- **Function:**
  1. Dilute toxins.
  2. It contain fibrin which localize infection.
  3. It carries oxygen & nutrients to the inflammatory cells
  4. It carries drugs & antibodies against bacteria



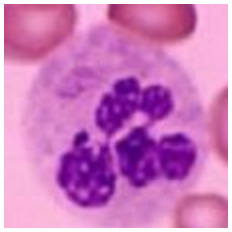




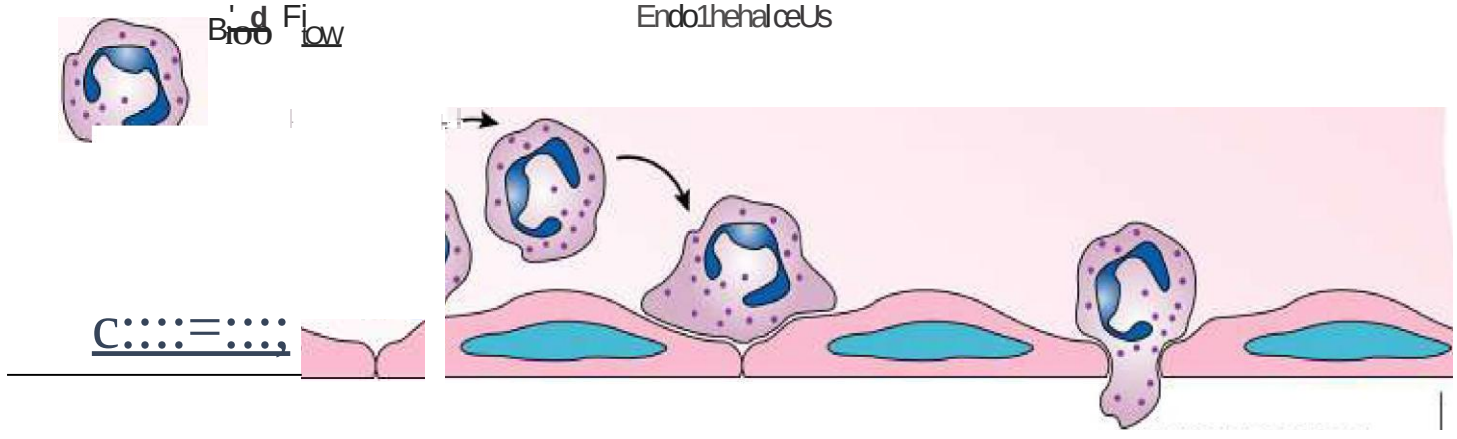
ACUTE INFLAMMATION

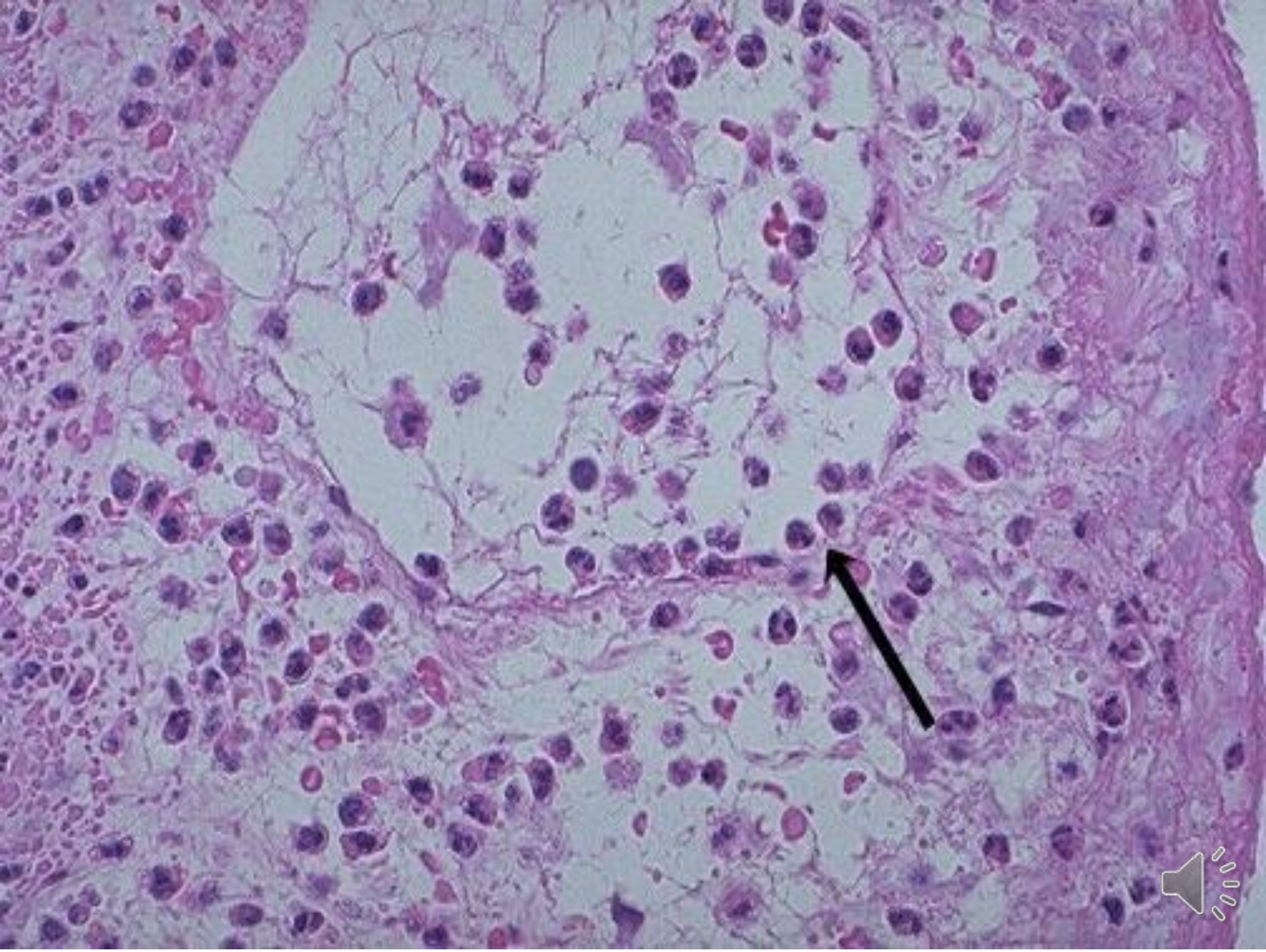
Acute inflammation

- Initial reaction of vascularized tissue to injury
- Neutrophils are predominant inflammatory cells in early stages (6-24 hours)



- Monocytes (macrophages) predominate in later stages (24-48 hours)


Blood Flow
Endothelial Cells
ROLLING
(Selectins)
FIRM ADHESION
(1, 2 integrins)
TRANSMIGRATION
(PCAM-1, etc.)



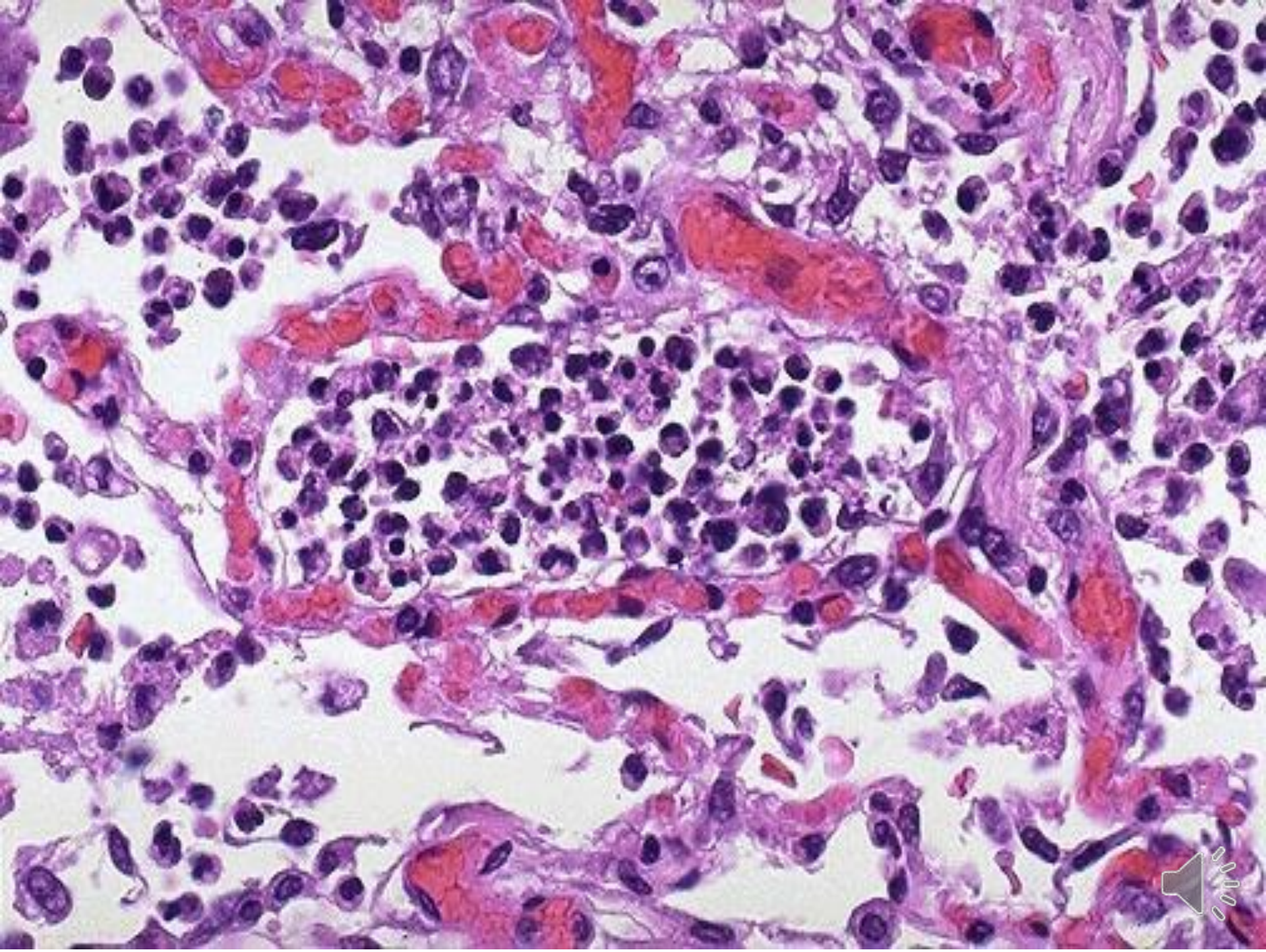
Chemical Mediators of Inflammation

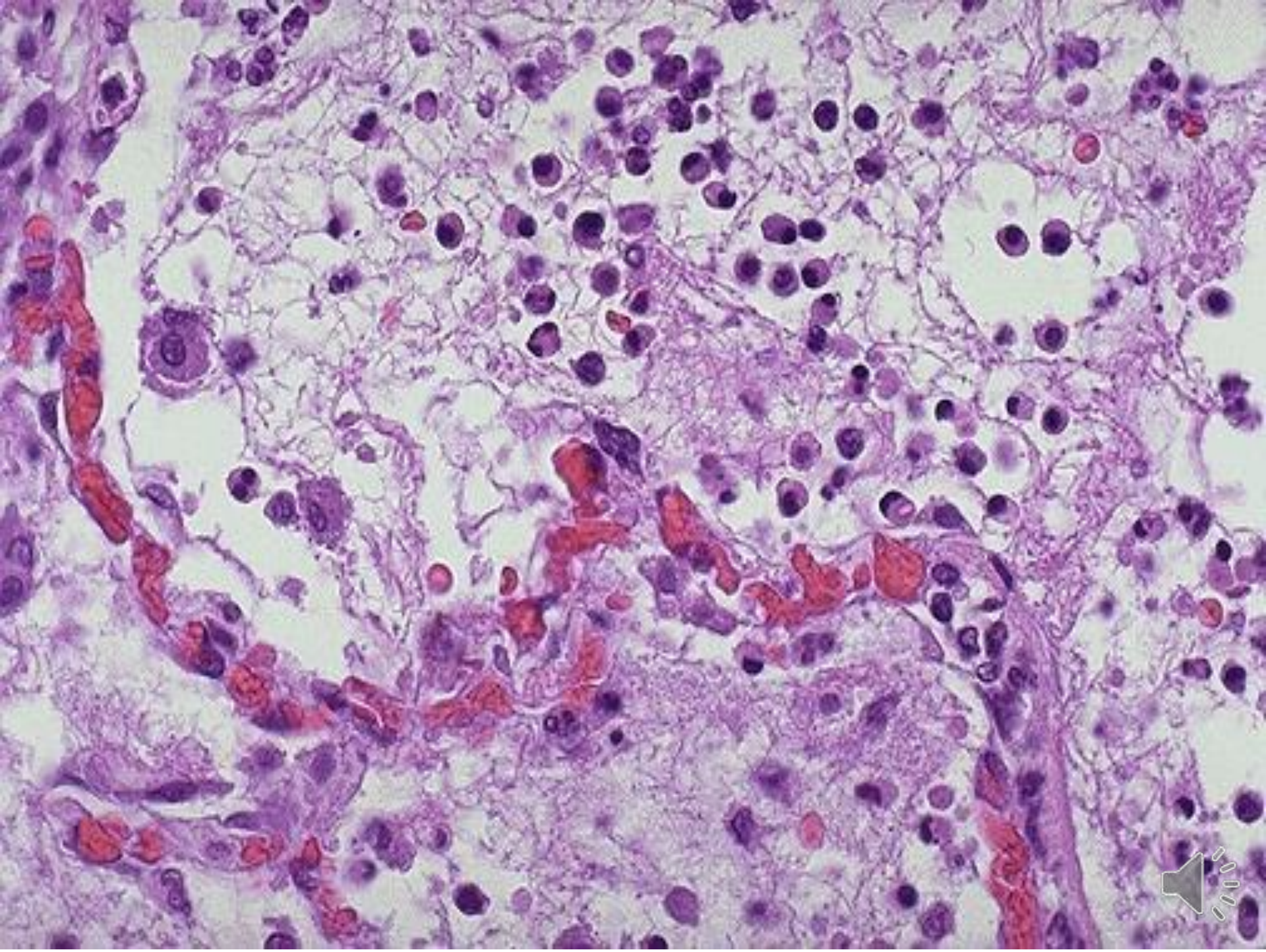
- A substances which *play a role in genesis and modulation of inflammatory reaction*

- ***They are responsible for:***
  - 1. Vasodilatation
  - 2. Increased permeability
  - 3. Emigration of WBC (Chemotactic agent).

Microscopic appearance of acute inflammation

- Congestion of blood vessels
- Exudation of fluid
- Exudation of inflammatory cells mainly neutrophils





Special macroscopic appearances of acute inflammation

- **1. Serous inflammation:**
- There is abundant protein-rich fluid exudate with a relatively low cellular content. Examples include inflammation of the serous cavities, such as peritonitis, and inflammation of a synovial joint, acute synovitis.

- **2. Catarrhal inflammation:**
- When mucus hypersecretion accompanies acute inflammation of a mucous membrane.The common cold is a good example.

- **3. Fibrinous inflammation :**
- When the inflammatory exudate contains plentiful fibrinogen, this polymerises into a thick fibrin coating.This is often seen in acute pericarditis and gives the parietal and visceral pericardium a 'bread and butter' appearance.

Effects of Acute Inflammation

BENIFITIAL EFFECTS

- Dilution of toxins
- Entry of antibodies
- Drug transport
- Fibrin formation
- Delivery of nutrient & O2
- Stimulation of immune system

HARMFUL EFFECTS

- Digestion of normal tissue
- Swelling & pain
- Inappropriate inflammatory response

Outcome of acute inflammation

1- Regeneration and repair. When the injury is limited
2- tissue destruction and persistent acute inflammation
3- ***Chronic inflammation*** may follow acute inflammation if the offending agent is not removed, or it may be present from the onset of injury (e.g., in viral infections or immune responses to self-antigens). This is marked by the replacement of neutrophils and monocyte with lymphocyte plasma cells and macrophage.It often include proliferation of fibroblast and new vessels with resultant scarring and distortion of architecture
4-Scar: this is the final result of tissue destruction with resultant distortion of structure and in some cases altered function

Abscess; this is a cavity filled with pus(neutrophils ,monocytes and liquefied cellular debris),Its often walled off by fibrous tissue and is relatively inaccessible to the circulation),Its usually caused by bacterial infections ,often by staphylococci

Ulcer :this is loss of surface epithelium ,this can be caused by acute inflammation of epithelial surface (e.g peptic ulcer and ulcer of the skin)

Fistula: this is an abnormal communication between two organ or between an organ and a surface

THANKS