Islamic university

Radiology techniques Department

Theoretical Pathology

Third Stage

PATHOLOGY LEC5

Inflammation

By

M.sc.Nadia Alshabanii

M.sc.Ali alruazik

Objectives of this lecture: at the end of this lecture, you will be able to know

- **Definition of inflammation**
- **Causes of inflammation**
- **Signs of inflammation**
- **Types of inflammation**

INFLAMMATION

- is a response of vascularized tissues that delivers leukocytes and molecules of host defense from the circulation to the sites of infection and cell damage in order to eliminate the offending agents.

Nomenclature: has suffix **itis** (after name of tissue) e.g.,

- Appendicitis
- Dermatitis
- Cholecystitis
- Gingivitis

Function of inflammation: 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.

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….

Msc. Nadia Habeeb
Msc. Ali alruazik

CARDINAL SIGNS OF INFLAMMATION

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

TYPES OF INFLAMMATION

1. Acute inflammation

- Short duration: hours -days-weeks
- Exudative fluid (protein rich fluid + infl cells + debris)
- Main inflammatory cells are Neutrophils

2. chronic inflammation

- Long duration: months - years
- Fibrosis (indurative)
- Main inflammatory cells are Lymphocytes, Macrophages and plasma Cells

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.

Msc. Nadia Habeeb
Msc. Ali alruazik

- 3. It carries oxygen & nutrients to the inflammatory cells
- 4. It carries drugs & antibodies against bacteria

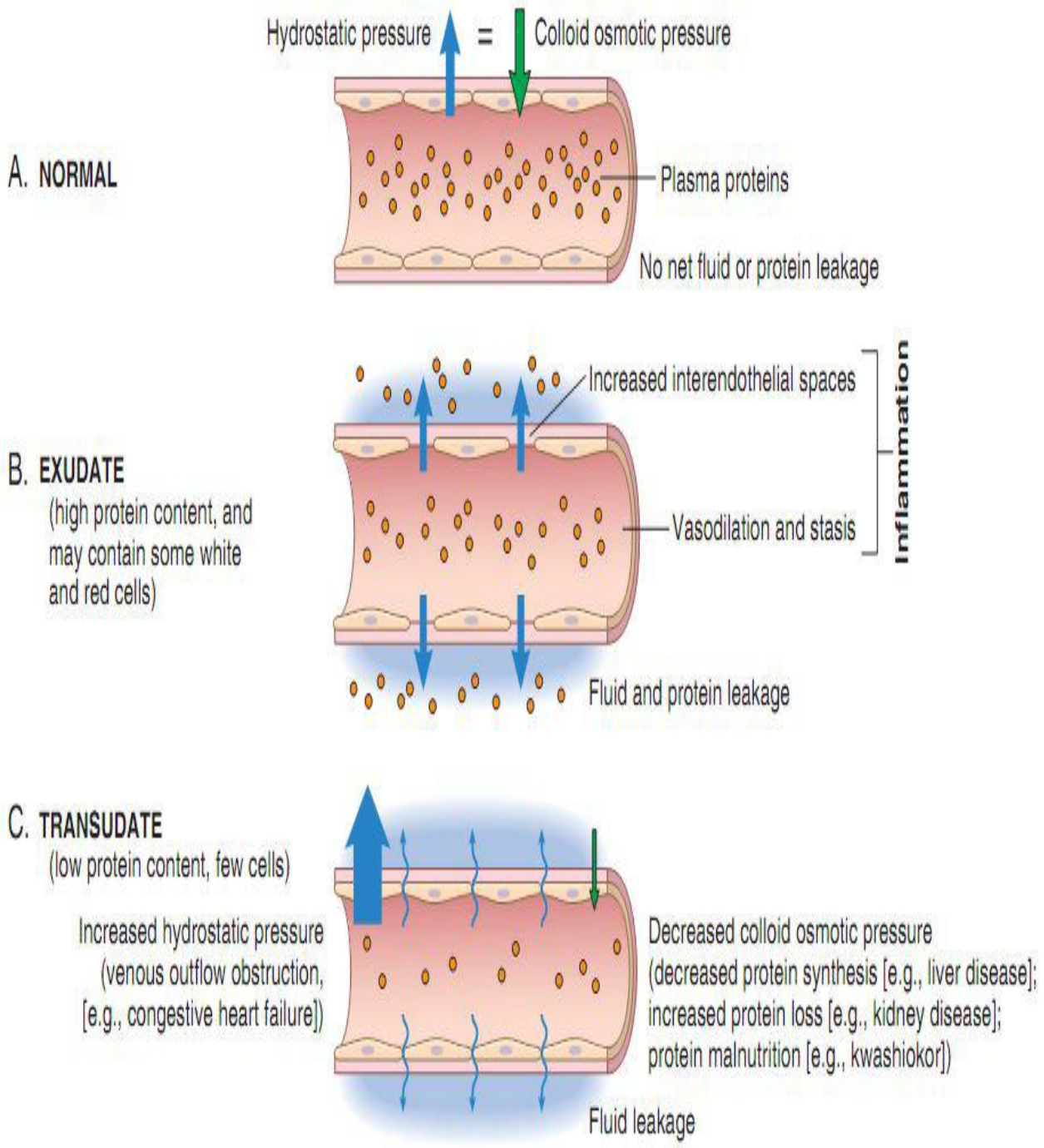
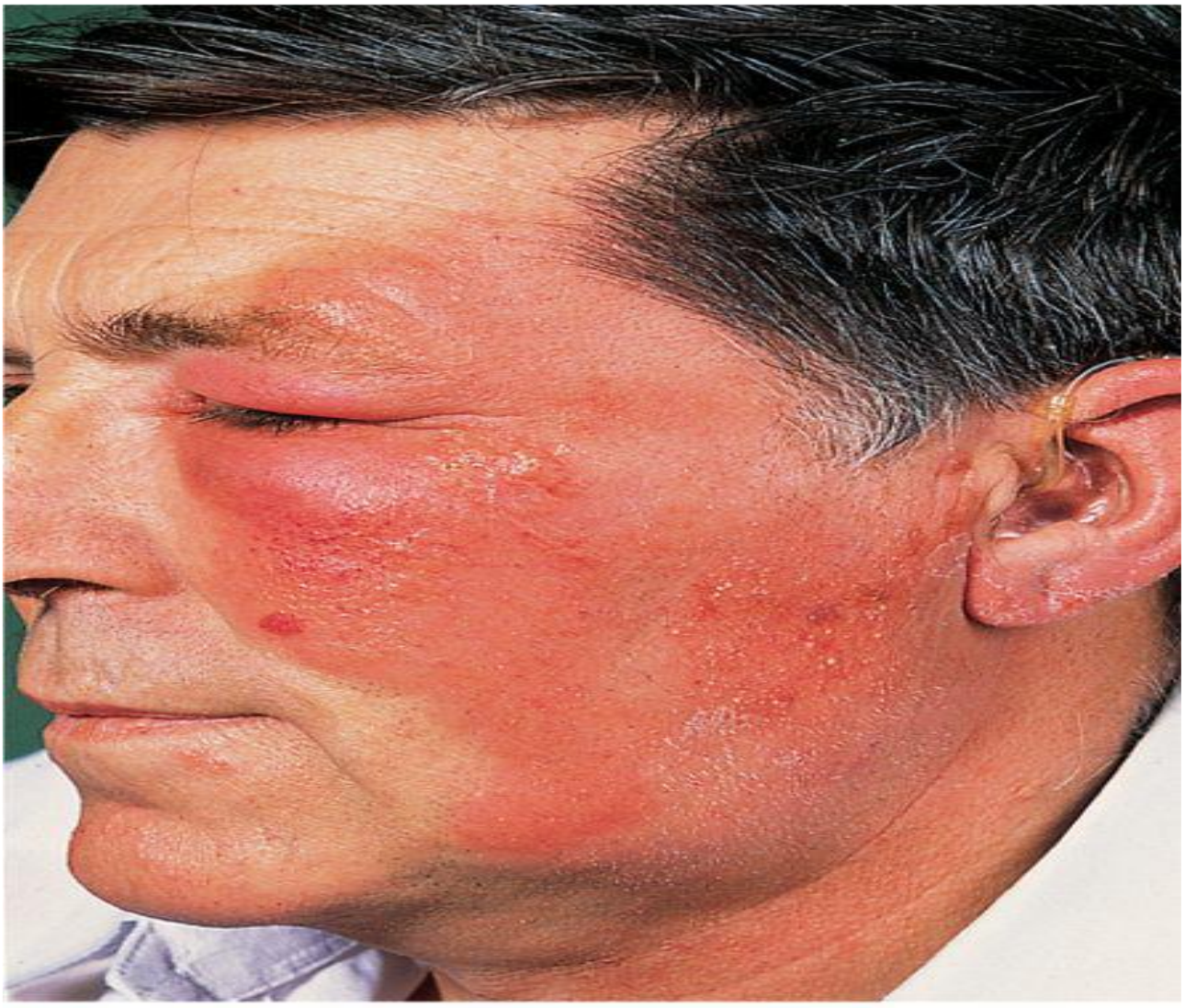


Fig. 3.2 Formation of exudates and transudates. (A) Normal hydrostatic pressure *(blue arrow)* is about 32 mm Hg at the arterial end of a capillary bed and 12 mm Hg at the venous end; the mean colloid osmotic pressure of tissues is approximately 25 mm Hg *(green arrow)*, which is equal to the mean capillary 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.

Msc. Nadia Habeeb
Msc. Ali alruazik



Msc. Nadia Habeeb
Msc. Ali alruazik

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)

PROCESSES OF ACUTE INFLAMMATORY RESPONSE

1- **Vascular changes**
2- **Leukocyte cellular events**

MECHANISMS OF INCREASED VASCULAR PERMEABILITY

1. **Endothelial cell contraction (venules)**
2. **Direct endothelial injury (any vessel)**
3. **Increased transcytosis (venules)**
4. **Leakage from new blood vessels**

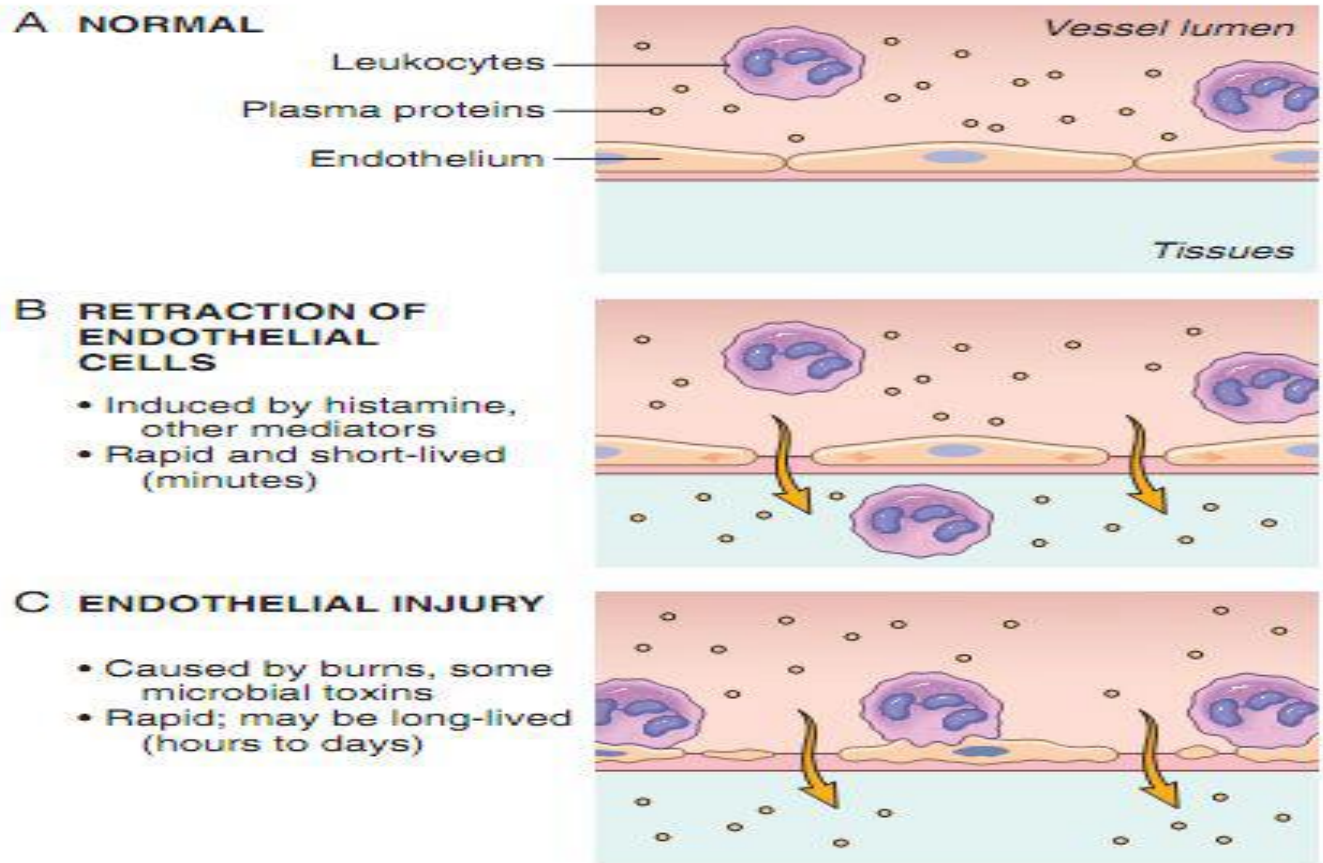
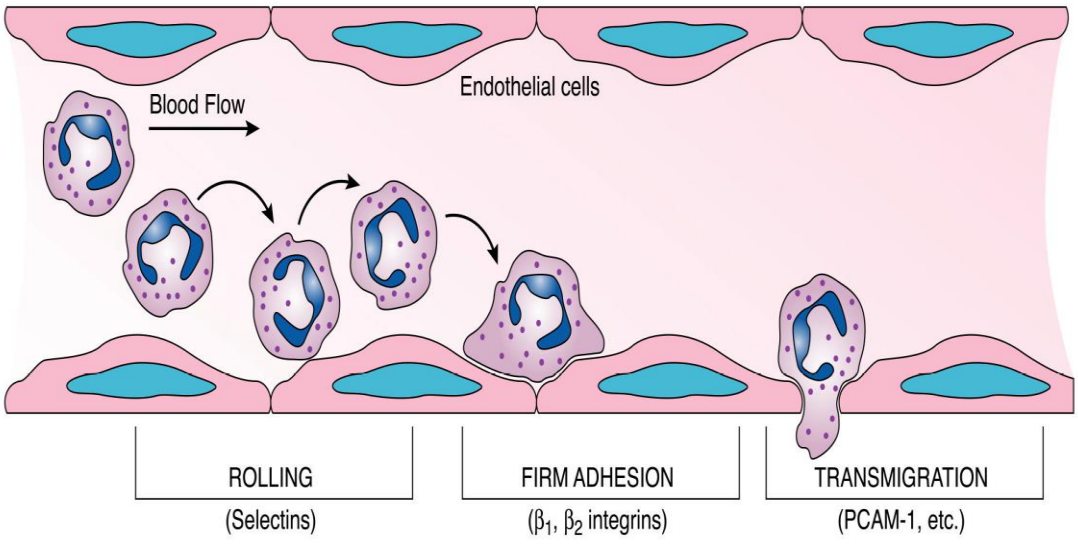


Fig. 3.3 Principal mechanisms of increased vascular permeability in inflammation and their features and underlying causes.

Msc. Nadia Habeeb
Msc. Ali alruazik

LEUKOCYTE CELLULAR EVENTS IN ACUTE INFLAMMATION

1. In the lumen: *margination, rolling, and adhesion to endothelium*. Vascular endothelium in its normal state does not bind circulating cells or allow their passage. In inflammation the endothelium is activated and can bind leukocytes as a prelude to their exit from blood vessels.
2. *Migration across the endothelium* and vessel wall.
3. *Migration in the tissues* toward a chemotactic stimulus.



Msc. Nadia Habeeb
Msc. Ali alruazik