Inflammation

Lecture: 5

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Objectives:

1. To define inflammation and explain types and causes of inflammation

2. To explain nomenclature of inflammation

3. To study mechanism of inflammation

Introduction

- **Inflammation:**

  Is the response of vascularized living tissue to injury.

- **Functions to:**
  - **Dilute toxins**
  - **Destroy and Isolate offending agent.**
  - **Initiate repair of tissue**

Inflammation is divided into two patterns:

1. Acute inflammation

(is an immediate response to injury)

Short duration minutes, hours to 2 days characterized by exudation of fluid & plasma protein, edema and emigration of leukocytes predominately neutrophils.

2. **Chronic inflammation :**

Longer duration with presence of lymphocytes, macrophages, proliferation of blood vessels and connective tissue.

Causes of Acute inflammation:

- Infections (bacterial, viral, parasitic)
- Trauma
- Physical and chemical agents (thermal injury, e.g., burns or frostbite; irradiation; some environmental chemicals)
- Tissue necrosis (from any cause)
- Foreign bodies (splinter, dirt, sutures)
- Immune reactions (also called hypersensitivity reactions)

Cardinal signs of acute inflammation

- Rubor = redness
- Tumor = swelling
- Calor = heat
- Dolor = pain
- Functio laesa = loss of function

Nomenclature

The nomenclature used to describe inflammation in different tissues employs

the tissue name and the suffix "-*itis*"

e.g.

pancreatitis

meningitis

pericarditis

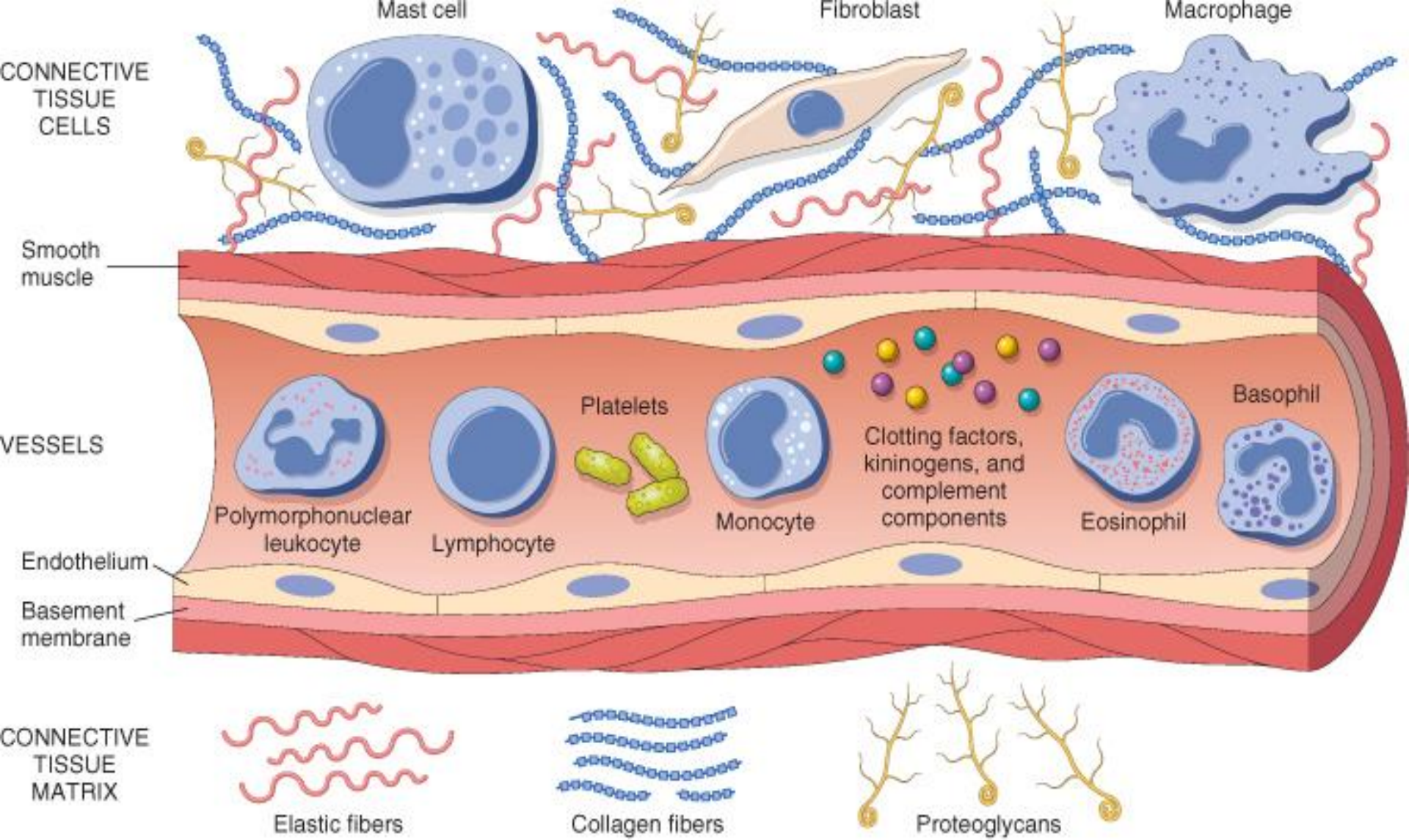
arthritis

Acute inflammation involves:

- **Alteration of vascular caliber**
  (vasodilation leads to increased blood flow)

- **Changes of microvasculature**
  (increased permeability for plasma proteins and cells)

- **Emigration of leukocytes from microcirculation**
  (leukocyte activation leads to elimination of offending agent)



The components of acute and chronic inflammatory responses: circulating cells and proteins, cells of blood vessels, and cells and proteins of the extracellular matrix.

Vascular changes and fluid leakage during acute inflammation lead to Edema in a process called Exudation

Edema : excess fluid in interstitial tissue or body cavities, either:

1. **Exudate:**

   - result of inflammation
   - high protein and cell debris
   - specific gravity >1.020

2. Transudate:

- result of hydrostatic or osmotic imbalance

- ultrafiltrate of plasma,

- no increased vascular permeability

- low protein content

- specific gravity < 1.012

Pus: **inflammatory exudate rich in neutrophils, debris of dead cells and microbes.**

Two Major Changes in Acute Inflammation:

1. Vascular changes.
2. Cellular changes.

These two changes result in three of five classic local sign (heat, redness & swelling)

The other two cardinal features of acute inflammation (pain & loss of function) occur as a consequence of mediator elaboration.

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Vascular Changes:

1. Transient momentary vasoconstriction (5 sec.)

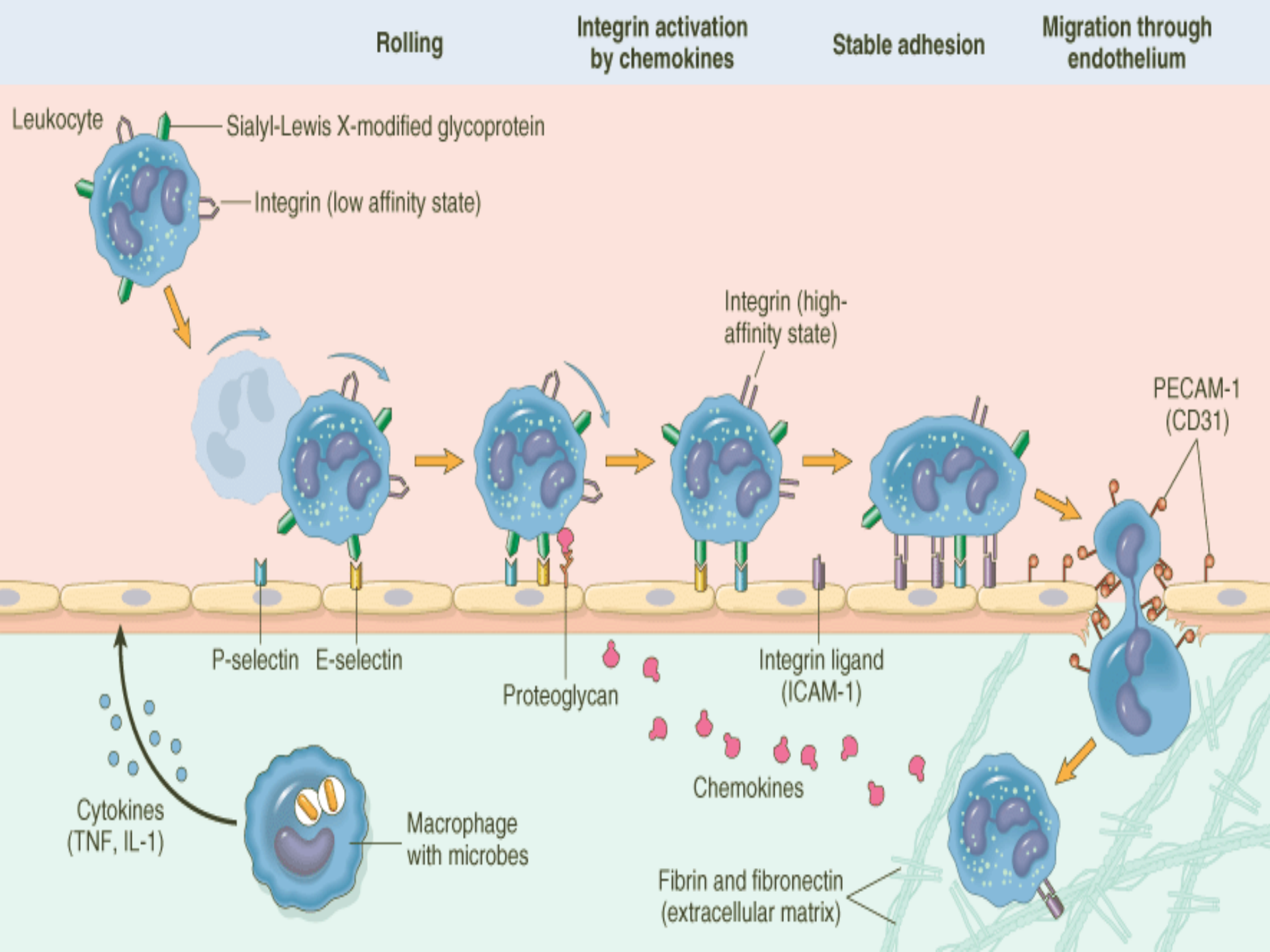
2. Followed by vasodilatation result in increase blood flow & engorgement of blood vessels.

3. Increase Vessel permeability & movement of protein rich fluid to extravascular system.

Increased vessel permeability manifest clinically as edema which occur in microcirculation (small arterioles, capillary & venules)

Cellular Changes

- Leukocytes leave the vasculature routinely through the following sequence of events:
  - Margination and rolling
  - Adhesion and transmigration
  - Chemotaxis and activation
- They are then free to participate in:
  - Phagocytosis and degranulation
  - Leukocyte-induced tissue injury


Rolling
Integrin activation by chemokines
Stable adhesion
Migration through endothelium
Leukocyte
Sialyl-Lewis X-modified glycoprotein
Integrin (low affinity state)
Integrin (high-affinity state)
PECAM-1 (CD31)
P-selectin
E-selectin
Proteoglycan
Integrin ligand (ICAM-1)
Chemokines
Cytokines (TNF, IL-1)
Macrophage with microbes
Fibrin and fibronectin (extracellular matrix)

Phagocytosis and Degranulation

- Once at site of injury, leukocytes:
  - Recognize and attach to the particle.
  - Engulfment (form phagocytic vacuole)
  - Killing (degradation) of ingested material.

THANK YOU