INFLAMMATION AND REPAIR Lecture 1

Definition of inflammation; Acute Inflammation Vascular Events in Inflammation

Lecturer: Dr. Maha Arafah

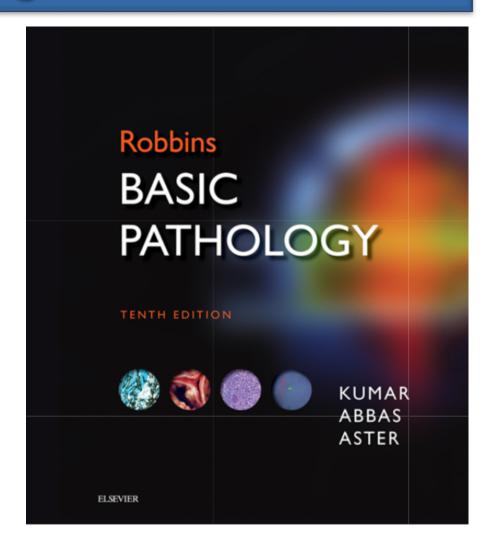
Foundation Block
Pathology
2018

Learning Objectives:

- 1. Define inflammation.
- 2. List cells & molecules that play important roles in inflammation
- 3. Types of inflammation: acute and chronic inflammation
- 4. Recognize the cardinal signs of inflammation.
- 5. Describe the sequence of vascular changes in acute inflammation (vasodilation, increased permeability) and their purpose.
- 6. Compare normal capillary exchanges with exchange during inflammatory response.
- 7. Define the terms edema, transudate, and exudate.

Reference book and the relevant page numbers...

- Robbins Basic Pathology 10th edition
- Page: 57 60



1. Define inflammation: its causes and steps

Injury

Tissue damage

Causes of Inflammation: Infection.

Trauma.

Physical injury (burns).

Chemical injury.

Immunological injury

(AID).

Tissue death (MI).

Tissue response

Eliminates the effect of injury (pathogen and necrotic tissue)

Repair

1. Define inflammation.

What is Inflammation?

Inflammation is a local response of the vascularized living tissue to infection and damaged tissue that brings cells and molecules of host defense from the circulation to the sites where they are needed

The aim: to localize and eliminate the causative agent, limit tissue injury and restore tissue to normality

→ Therefore, Inflammation is part of immunity: This is a broad protective response (innate immunity) 1. Define inflammation.

Steps of inflammation

The offending agent is recognized by host cells and molecules.



Leukocytes and plasma proteins are recruited from the circulation to the site where the offending agent



The leukocytes and proteins are activated to destroy and eliminate the offending substance.



The reaction is controlled and terminated.



The damaged tissue is repaired.

Can inflammation be harmful!?

Disorders	Cells and Molecules Involved in Injury	
Acute		
Acute respiratory distress syndrome	Neutrophils	
Asthma	Eosinophils; IgE antibodies	
Glomerulonephritis	Antibodies and complement; neutrophils, monocytes	
Septic shock	Cytokines	

1. Define inflammation.

Inflammation

 Inflammation is terminated when the offending agent is eliminated and the secreted mediators are broken down or dissipated.

There are active anti-inflammatory mechanisms that serve to control the response and prevent it from causing excessive damage to the host.

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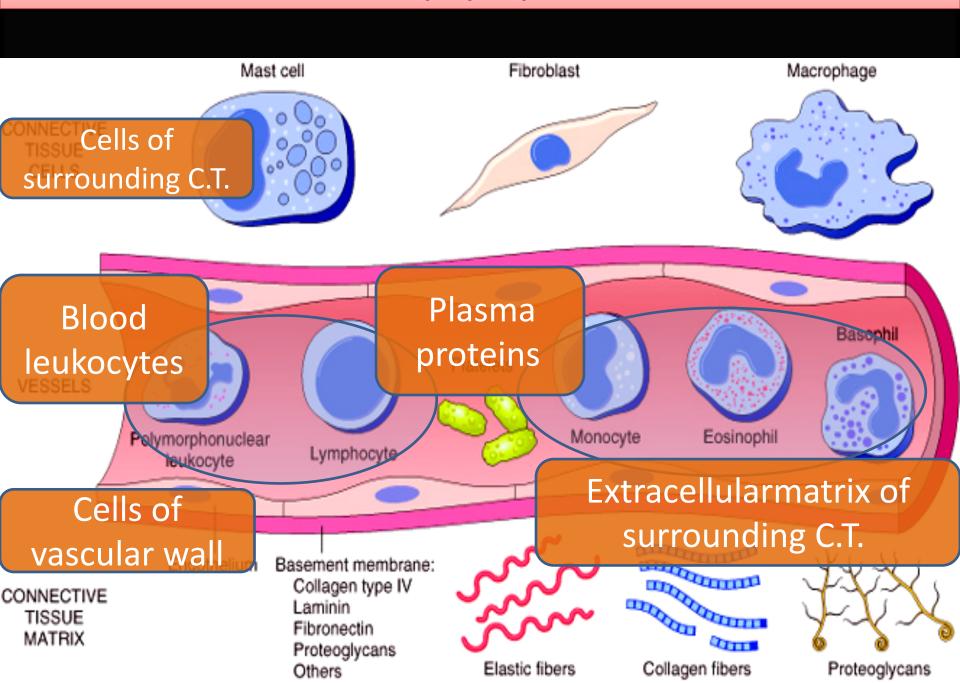
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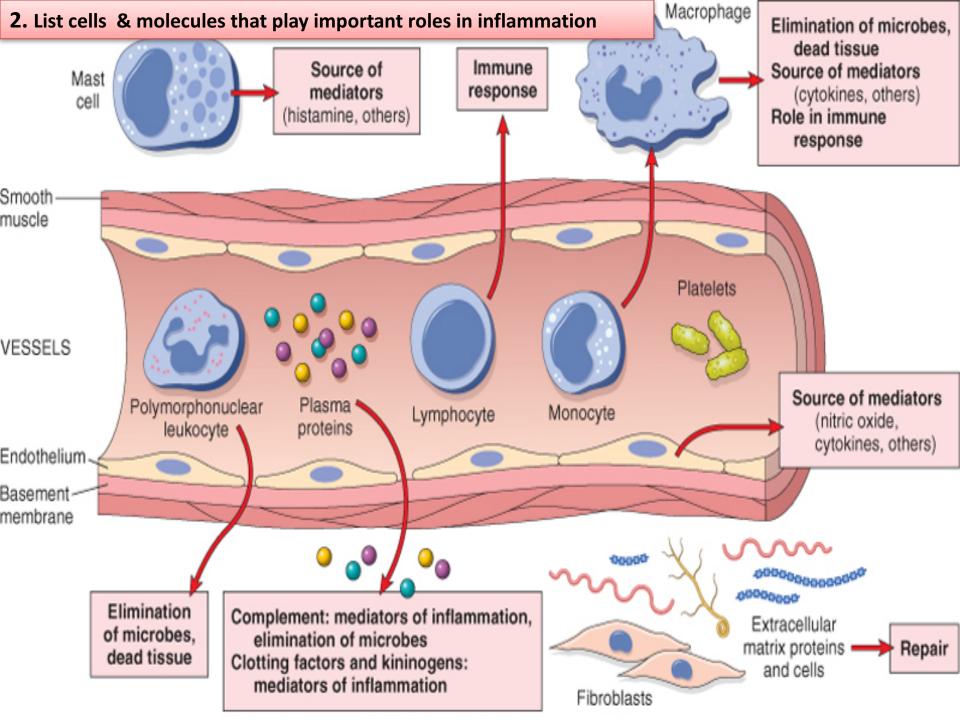
Inflammation is mediated by chemical substances called

CHEMICAL MEDIATORS

What is the source of these chemical mediators?

Phagocytes and other host cells
 Leukocyte
 Endothelium
 Mast cell

2. Plasma proteins



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TYPES OF INFLAMMATION

Acute inflammation

Chronic Inflammation

3. Compare between acute and chronic inflammation

Features of acute and chronic inflammation

Feature	Acute	Chronic
Onset		
Cellular infiltrate		
Tissue injury, fibrosis		
Local & systemic signs		

Acute inflammation

 A rapid response to an injurious agent that serves to deliver mediators of host defense-leukocytes and plasma proteins-to the site of injury.

The outcome of acute inflammation

is either

 elimination of the noxious stimulus, followed by decline of the reaction and repair of the damaged tissue

or

persistent injury resulting in chronic inflammation

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4. Recognize the cardinal signs of inflammation

Clinical Features

The 5 ancient cardinal signs of inflammation are



Tumor:-swelling

Rubor :- redness

Calor: - warmth

Dolor :- pain

Functio Laesa :- loss of function

The suffix "its" is added to the base word to state the condition as in appendix/appendicitis

4. Recognize the cardinal signs of inflammation







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Events of acute Inflammation

- Acute inflammation has three main events:
 - (1) Hemodynamic changes

(alterations in vascular caliber that lead to an increase in blood flow)

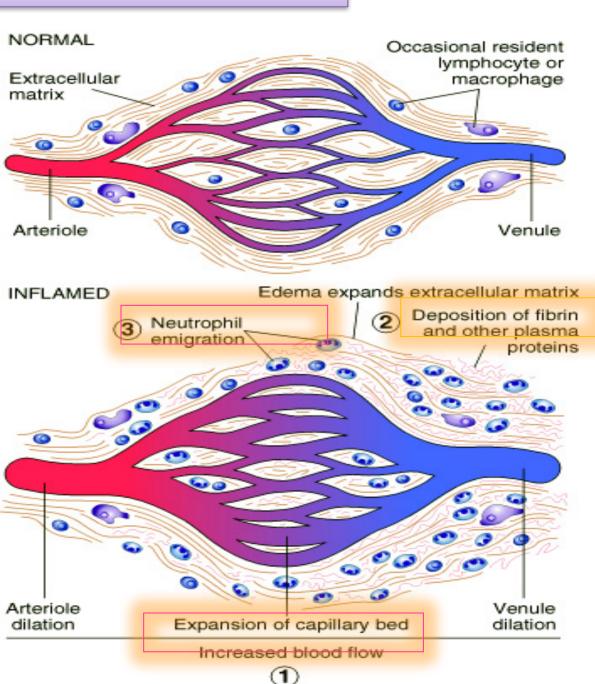
(2) Increased vascular permeability

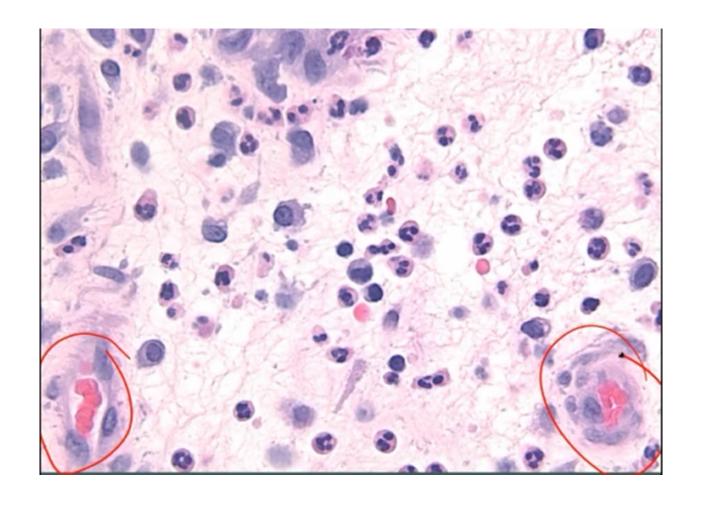
(structural changes in the microvasculature that permit plasma proteins and leukocytes to leave the circulation)

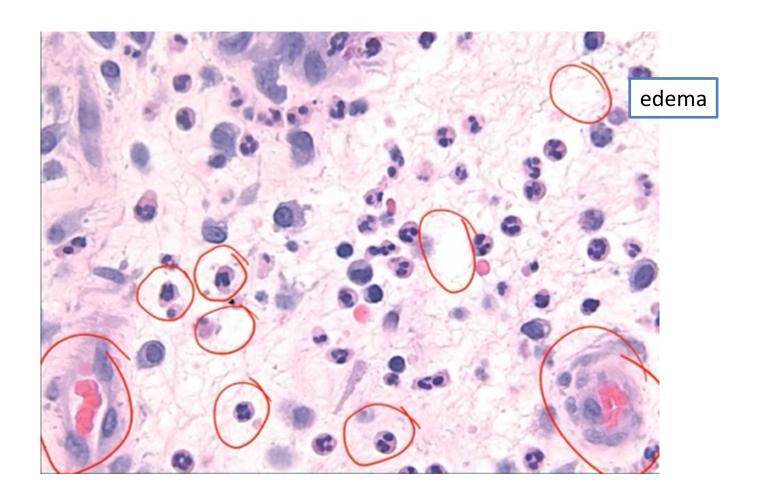
(3) Emigration of the leukocytes from the microcirculation

(their accumulation in the focus of injury, and their activation to eliminate the offending agent)

Vascular Events 1. Vasodilatation Hemodynamic changes







Phases of changes in Vascular Caliber and Flow

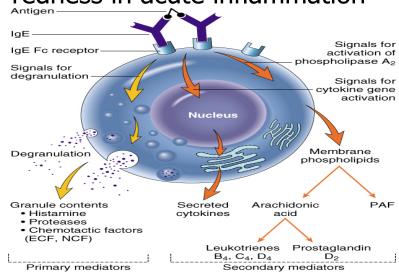
1. Transient vasoconstriction of arterioles

It disappears within 3-5 seconds in mild injuries

2. Vasodilatation: It involves the arterioles results in opening of new microvasculature beds in the area leading to increasing blood flow – Histamine effect

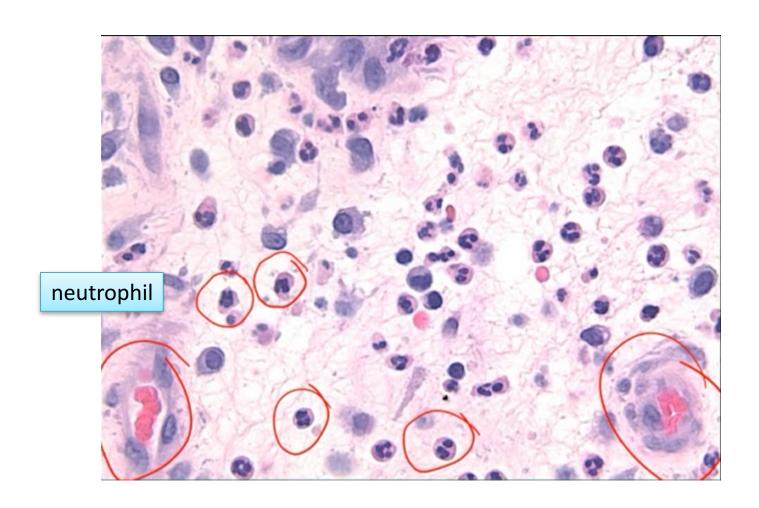
Dilatation of blood vessel is the cause of redness in acute inflammation

- 3. Slowing of the circulation due to increased permeability of the microvasculature, this leads to outpouring of protein-rich fluid in the extravascular tissues.
- 4. Stasis: slow circulation due to dilated small vessels packed with red cells



Slowing of the circulation

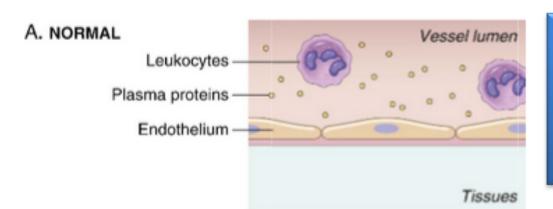
A Normal postcapillary venule B Acute inflammation Emigrating neutrophil Pavementing neutrophil Cellular axial stream Zone of plasma, Marginating neutrophil Erythrocyte rouleaux Escaping erythrocytes



Vascular Events

2. Increased Vascular Permeability

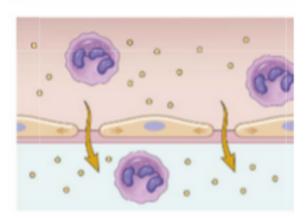
- A hallmark of acute inflammation (escape of a protein-rich fluid).
 - induced by histamine, kinins, and other mediators
- It affects small & medium size venules, through gaps between endothelial cells
- It result in swelling (tumour) which occurs as a cardinal sign of inflammation



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

B. RETRACTION OF ENDOTHELIAL CELLS

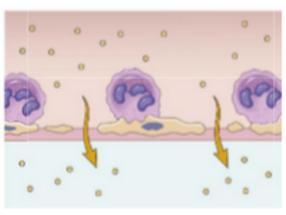
- Induced by histamine, other mediators
- Rapid and short-lived (minutes)



venules

C. ENDOTHELIAL INJURY

- Caused by burns, some microbial toxins
- Rapid; may be long-lived (hours to days)



Arteriols, capillaries and venules

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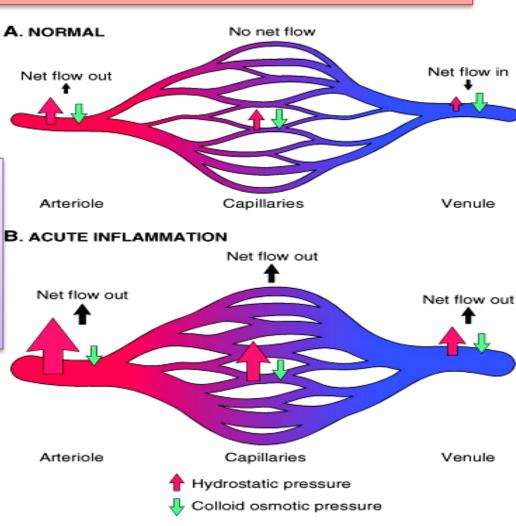
Increased blood volume lead to increased local hydrostatic pressure leading to transudation of protein-poor fluid into the extravascular space.

What is the edema?

denotes an excess of fluid in the interstitial or serous cavities

It can be either an

exudate or a transudate



6. Compare normal capillary exchanges with exchange during inflammatory response

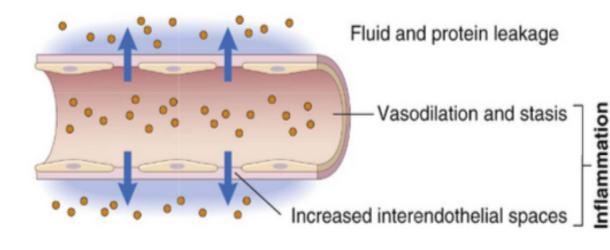
Hydrostatic pressure Colloid osmotic pressure

Plasma proteins

A. NORMAL

B. EXUDATE

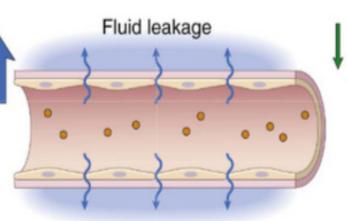
(high protein content, and may contain some white and red cells)



Increased hydrostatic pressure (venous outflow obstruction, [e.g., congestive heart failure])

C. TRANSUDATE

(low protein content, few cells)



Decreased colloid osmotic pressure (decreased protein synthesis [e.g.,liver disease]; increased protein loss [e.g., kidney disease])

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Edema is defined as an excess of fluid in the interstitial space. What is the difference between transudates and exudates?

Transudate

is a fluid with low protein content and a specific gravity of less than 1.012

It is essentially an ultrafiltrate of blood plasma that results from osmotic or hydrostatic imbalance across the vessel wall

without an increase in vascular permeability

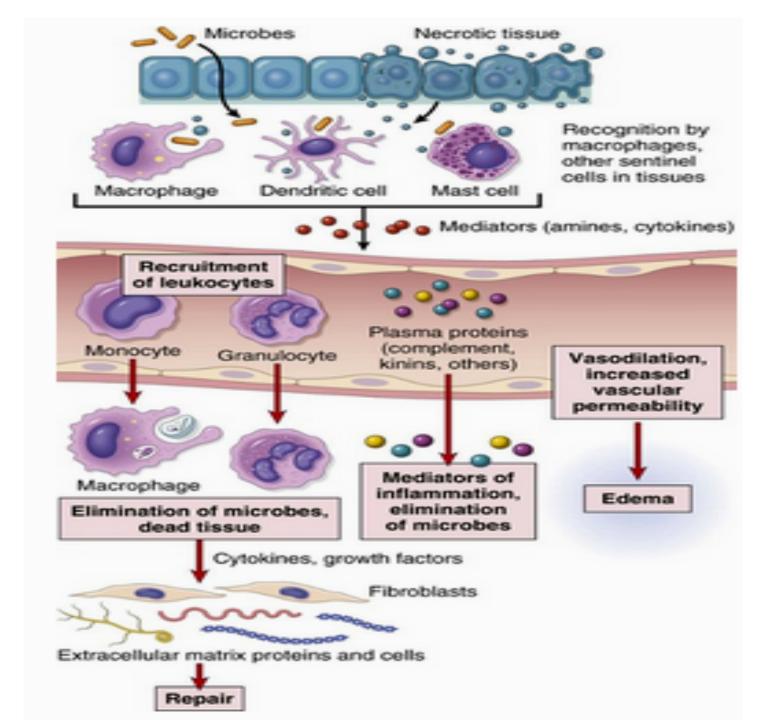
Exudate

An inflammatory extravascular fluid that has a high protein concentration, cellular debris, and a specific gravity above 1.020

It implies significant alteration in the normal permeability of small blood vessels in the area of injury

TAKE HOME MESSAGES

- Inflammation, the local response of the vascularised living tissue to injury.
- Could be acute or chronic.
- Several cells & molecules that play important roles in inflammation.
- Inflammation has vascular and cellular events to eliminate the cause.
- Vascular events include vasodilation and increased permeability to deliver a protein rich fluid to the site of inflammation.



Rolling → Activation → Adhesion → Transmigration

