Definition of Inflammation,Acute Inflammation

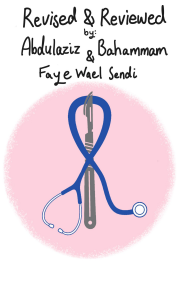
Editing File

Objectives:

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

Color Code:

Female's Notes
Male's Notes
Important
Extra



Inflammation

Definition: Inflammation is a **local** response of the **vascularized living tissue** to infection and tissue damage. that(reponse) brings cells and molecules of host defense from the circulation to the sites where they are needed.

The aim: to localize and eliminate the offending agents,limit tissue injury and restore tissue to normal

Inflammation is part of immunity:
This is a broad protective response (innate immunity)(عام)

Infection: caused by an organism's pathogen such as bacteria, virus, mycoplasma

Injury

Tissue damage

Tissue response

Elimination of the effect of injury (pathogen and necrotic tissue)

Repair

Causes of Inflammation

1)Immunological injury
(Autoimmune disorder e.g. Rheumatoid Arthritis).

2)Trauma.

3)Tissue death
(e.g.Myocardial infarction).

4) Infection

5)Drug reaction

6)Radiation

7) Physical injury (burns & heat or Excess cold, frostbite)

8) Chemical injury (CCl4) .

Steps of inflammation:

1. The offending(الضار) agent is **recognized** by host cells.
2. **Leukocytes and plasma proteins are recruited** (تجنيد)from the circulation to the site of the offending agent.
3. The **leukocytes and proteins are activated** to **destroy** and **eliminate** the offending substance. (After the elimination they should return to the circulation, to not destroy normal cells).
4. The reaction is controlled and **terminated**.
5. The damaged **tissue is repaired**.(some tissue can't repair like myocardial, brain cells so it left scars).

Harmful inflammation

1- acute respiratory distress syndrome (neutrophils)

2- asthma (eosinophils ige antibodies)

3- glomerulonephritis (antibodies and complement neutrophils , monocytes)
4- septic shock (cytokines)

Termination of inflammation

click for short helpful video

how

when

By **activate anti-inflammatory mechanisms** that serve to control the response and prevent it from causing excessive damage to the host.

The **offending agent is eliminated** and the secreted mediators are broken down or dissipated.

Macrophages are phagocytic substances that can eat microbes

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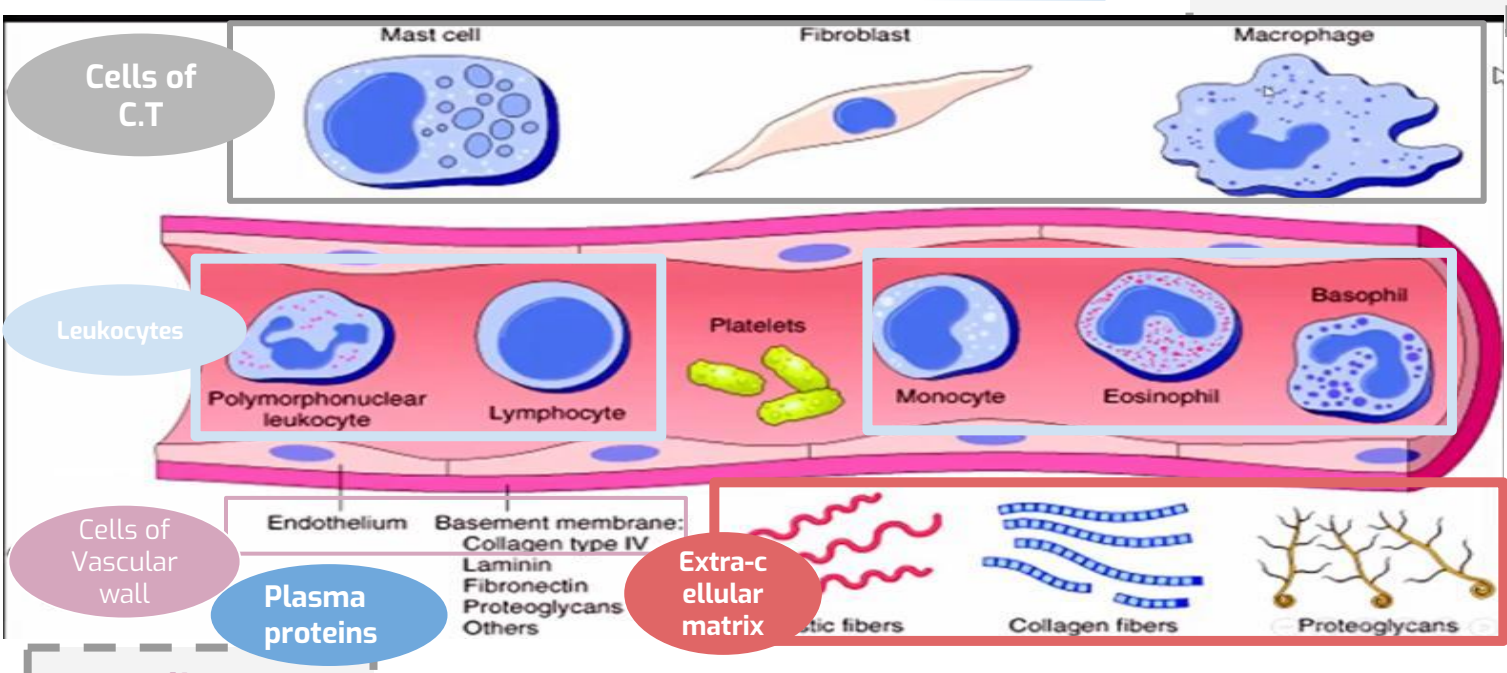
List of cells & molecules that play important roles in inflammation:



Compliments + coagulation factors + fibrinogen + immunoglobulin (Antibody)

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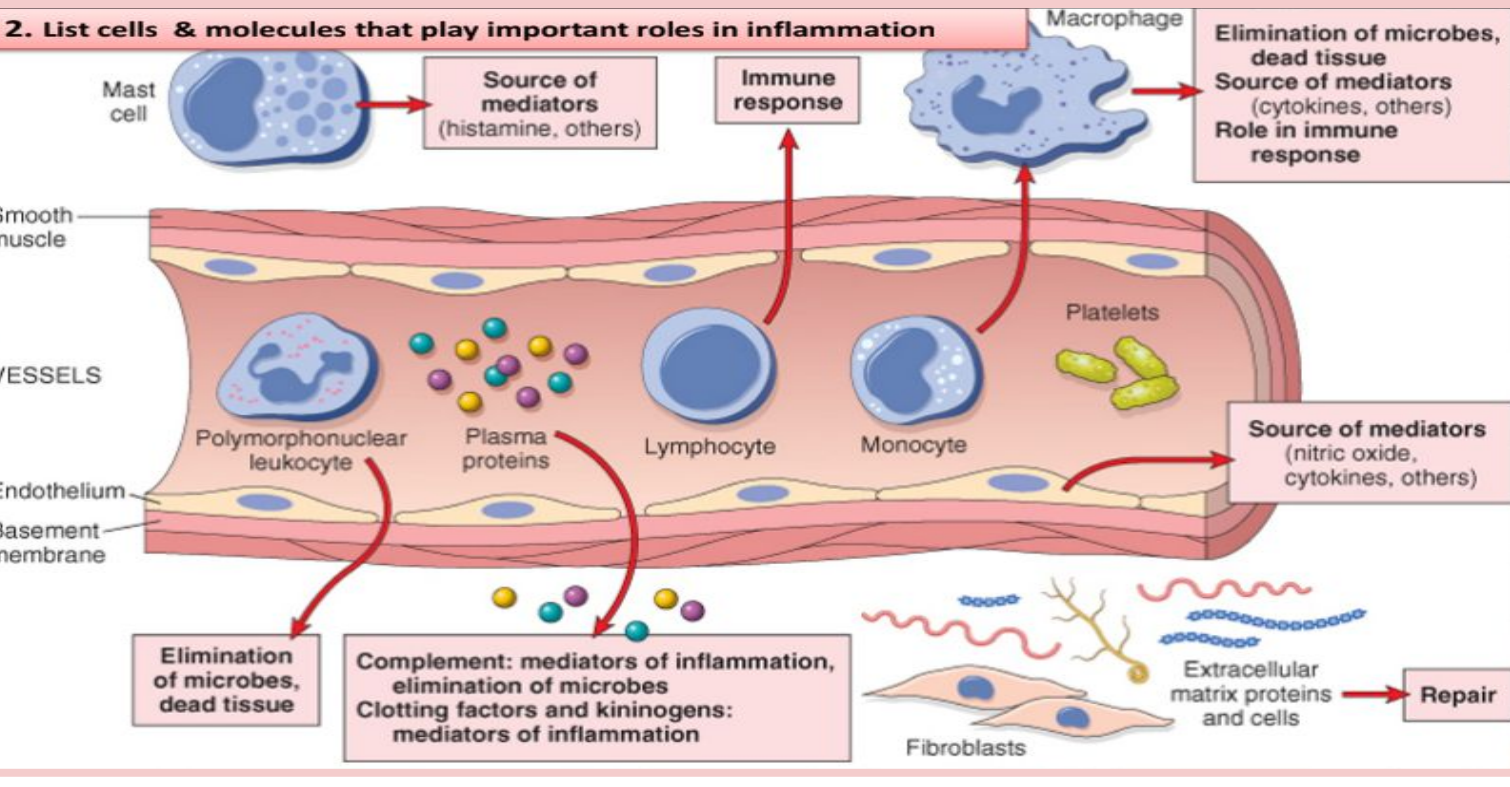
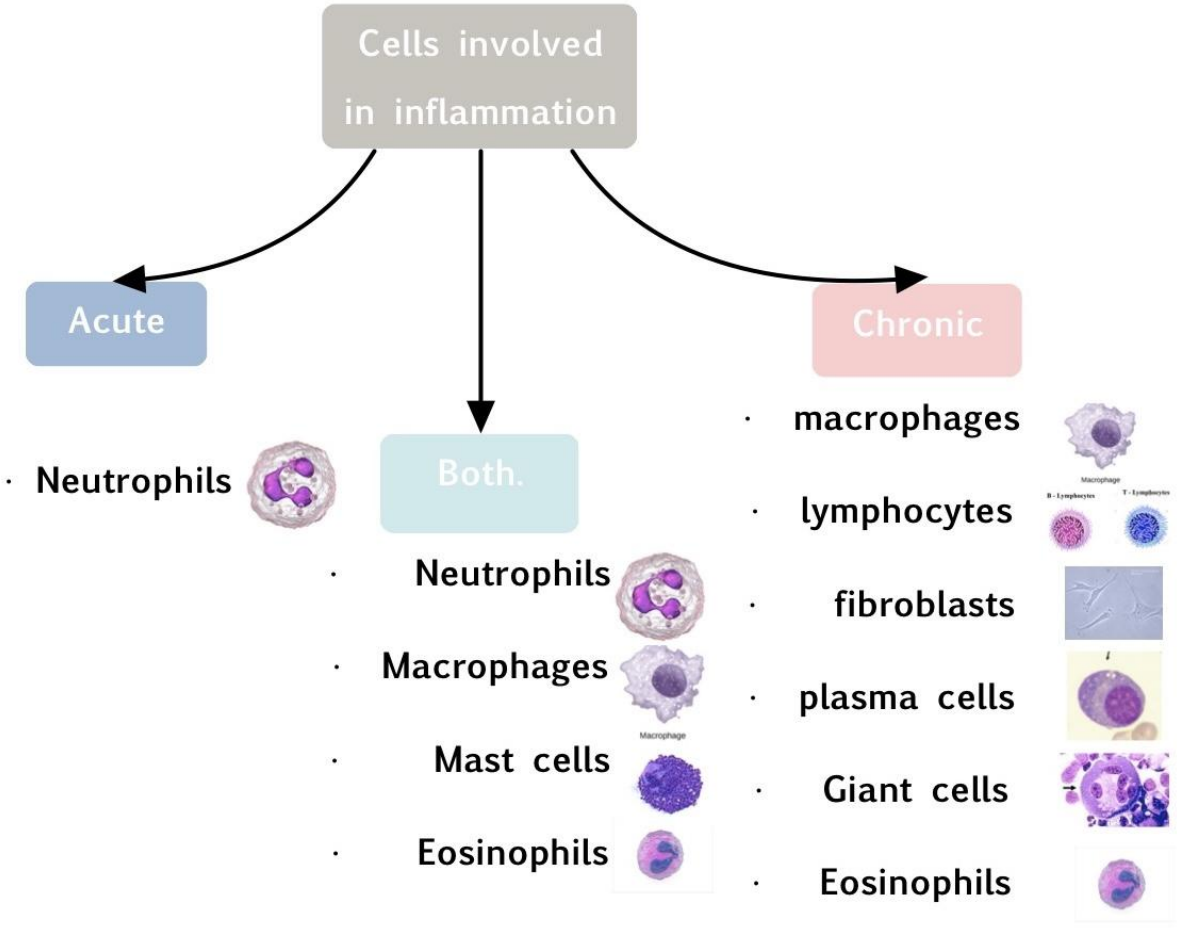

2. List cells & molecules that play important roles in inflammation
Mast cell
Source of mediators (histamine, others)
Immune response
Macrophage
Elimination of microbes, dead tissue
Source of mediators (cytokines, others)
Role in immune response
Smooth muscle
VESSELS
Platelets
Polymorphonuclear leukocyte
Plasma proteins
Lymphocyte
Monocyte
Source of mediators (nitric oxide, cytokines, others)
Endothelium
Basement membrane
Elimination of microbes, dead tissue
Complement: mediators of inflammation, elimination of microbes
Clotting factors and kininogens: mediators of inflammation
Extracellular matrix proteins and cells
Repair
Fibroblasts


Cells involved in inflammation
Acute
Chronic
Both.
· Neutrophils
· Neutrophils
· Macrophages
· Mast cells
· Eosinophils
· macrophages
· lymphocytes
· fibroblasts
· plasma cells
· Giant cells
· Eosinophils

1 Neutrophils (Acute only)

O-loops or banded is when bone marrow give immature cells

Pneumonia: inflammation of lung in respiratory ciliated columnar epithelium inflammatory cell in alveoli of lung become full of WBC neutrophils or (polymorphonuclear) leukocytes
Polymorphism leukocytes because it has 3-5 loops nucleus
It has large number of lysosomes
Function: phagocyte foreign body , secrete enzymes like oxidase & proteases from the cytoplasm they interact with bacteria to form superoxide & hydroxyl radical to help in killing bacteria cells
It's the most important in acute inflammation it increase due to acute inflammation (mostly bacterial infection

2 Lymphocytes (chronic only)

Occur in chronic inflammation specially viral
Lymphocytes could be divided into T Lymph & B lymph

1-T lymph is cell mediated immunity it produce lymphokines
Most of them in the circulating blood
It characterizes chronic inflammation

2-B lymph is Humoral immunity it transferred to plasma when activated Secrete cytokines interferons & interleukins

3 Plasma Cells (chronic only)

- Plasma is modified B lymphocyte
- It secrete immunoglobulins proteins IGG,IGE,IGM (antibodies)
- It increase in chronic inflammation
- It has an eccentric nucleus
- Plasma cells found in tissues

4 Mast Cells

Serotonin:secreted by mast cell & platelets

It has granules organophilic
Seen in :
1-allergic reactions
2-hypersensitivity reaction type 1
IGE antibody bind to receptor on mast cell that lead to release of granules these granules contain vasoactive amine histamine & (5 hydroxytryptamine) Serotonin they make vascular congestion or dilation & increase vascular permeability (the fluid come out of blood vessel to interstitial tissue or edema or tumor in internal organs

5 Macrophages (acute&chronic)

Another name for macrophages is histiocytes when its modified monocyte that comes from bone marrow
It's acute & chronic inflammation active in phagocytosis
Secrete cytokines proteins which is chemical mediator secreted by macrophages & lymphocytes they are `Interleukins & Interferons`

6 Eosinophils (acute&chronic)

Usually has 2 loops
Can do phagocytosis but very weak
Occur because of parasites or allergic reaction

Case: 8yo boy has abdominal pain so we did urine test we discover parasites in abdomen bilobed
Eosinophilia increase in eosinophil granules

Increase in these cases : bronchial asthma ,hypersensitivity reaction, allergic reaction,parasitic infection

Cell type	Function	Appearance
Neutrophils	Phagocytosis & Protease & Oxidase	**Acute**
Lymphocyte	Lymphokines	**Chronic**
Plasma cells	Formation of Antibodies	**Chronic**
Mast cell	Histamine & Serotonin	**Both**
Macrophages	Phagocytosis & presenting Antigens	**Acute & Chronic**
Eosinophils	Parasite & allergy reaction	**Acute & Chronic**

Phagocytosis : cell engulf other particles or microbes as defense mechanism against any damaging effect

Clinical features of inflammation

1 Systemic signs of inflammation

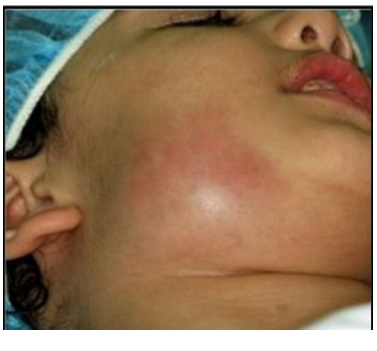
1. **Fever** also known as **(Pyrexia) caused by chemical mediators**
2. **Malaise** (fatigue)
3. **Vomiting** cause of otitis in young kids only.
4. **Headache**
5. **Loss of sensation**
6. **Increased erythrocyte sedimentation rate ESR** or RBC
7. Nonspecific findings, **increase of C-reactive protein** [بايوكمستري لاب] **secreted by the liver** it tells you that the patient has inflammatory disease

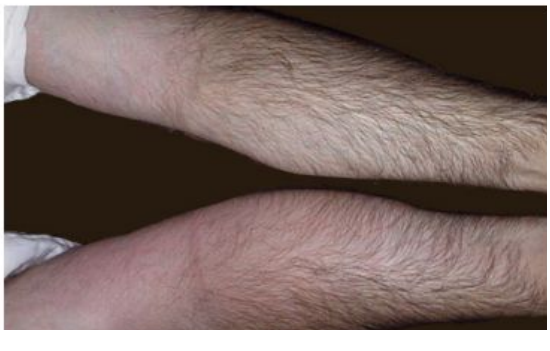
2 Cardinal signs of inflammation

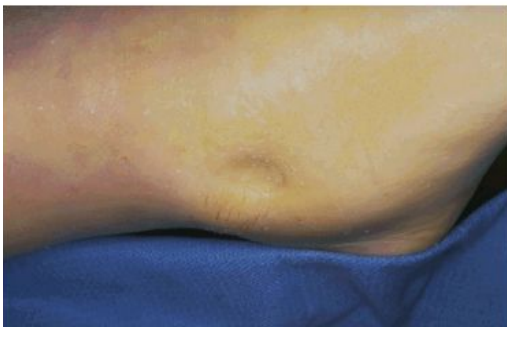
latin (important)	Meaning
Tumor	Swelling (histamine increase permeability of venules
Rubor	Redness (histamine vasodilation)
Calor	Warmth (histamine vasodilation)
Dolor	Pain (PGE2/ bradykinin)
Functio laesa	Loss of function

Increase caliber+blood supply to the area

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







Types of inflammation

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

Outcomes of inflammation:

1. **Elimination of the noxious stimulus**, followed by decline of the reaction and repair of the damaged tissue
2. **Persistent injury resulting in chronic inflammation**

	Acute (Immediately after injury)	Chronic
Onset	Fast : minutes or hour	Slow : days or weeks
Cellular infiltrate	Neutrophils	Lymphocytes and macrophages
Tissue injury, fibrosis	Mild , self limited	Often severe and progressive
Local & systemic signs	Prominents (more clear+painful)	Less prominent may be subtle (minimal change), (less clear+ less pain)
Examples	Hepatitis A & Pneumonia	Hepatitis B & Tuberculosis

Events of acute inflammation

Vascular

Alterations in vascular caliber (internal diameter) that lead to an increase in blood flow (Vasodilation).

Structural changes in the microvasculature (capillaries) that permit plasma proteins and leukocytes to leave the circulation (Increase in the escape of the fluids and cells from circulation to the tissue).

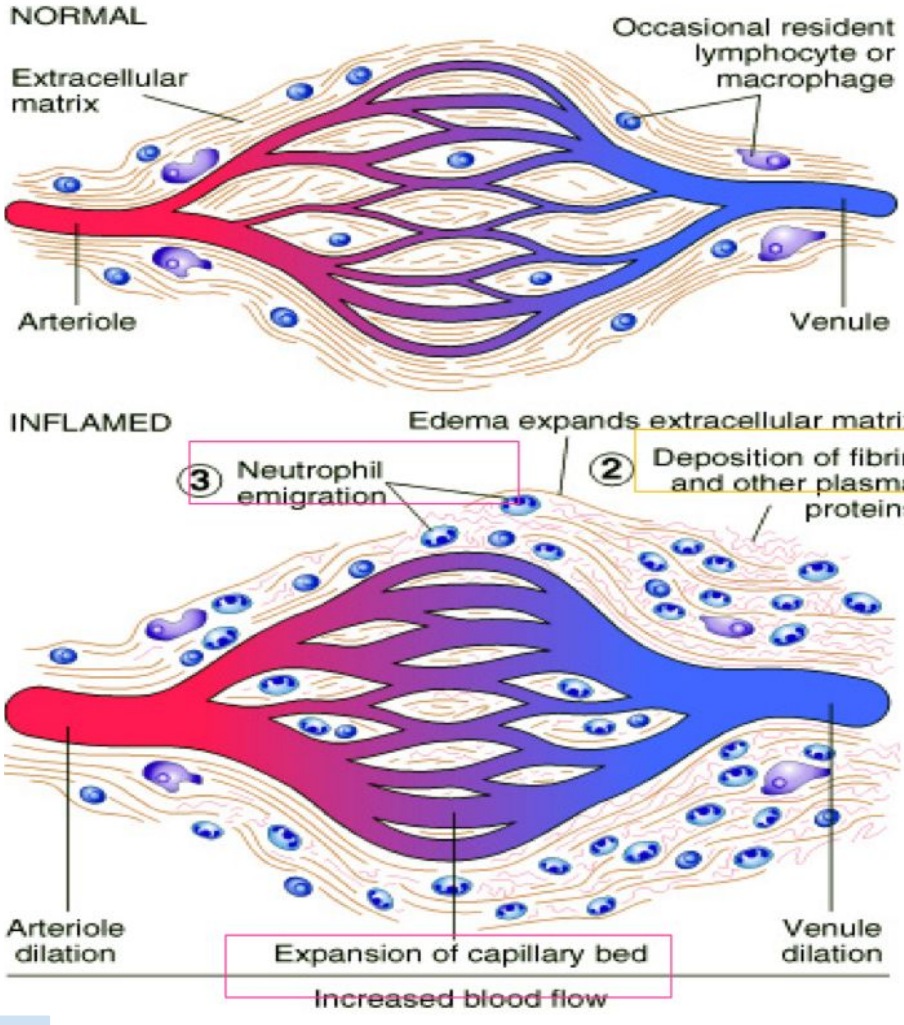
Cellular

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

Events of acute inflammation

Sequence of vascular events in acute inflammation:

1. Reflex vasoconstriction(يعني بالبداية تنصدم وتطلع ردة فعل ثم بعد ثواني تتصرف صح في الخطوة 2)
2. Vasodilatation/ Vasodilation(in response to histamine)
3. Increased capillary permeability



Hydrostatic pressure:القوة الدافعة اللي تجي من القلب-الشرايين- تدفع الدم إلى الخلايا

Oncotic pressure: القوة الساحبة تسحب الدم الى الوريد

Normal state:

Hydrostatic=Oncotic

But in inflammation caliber increases so does permeability.

↑ Diameter of the capillaries

↓

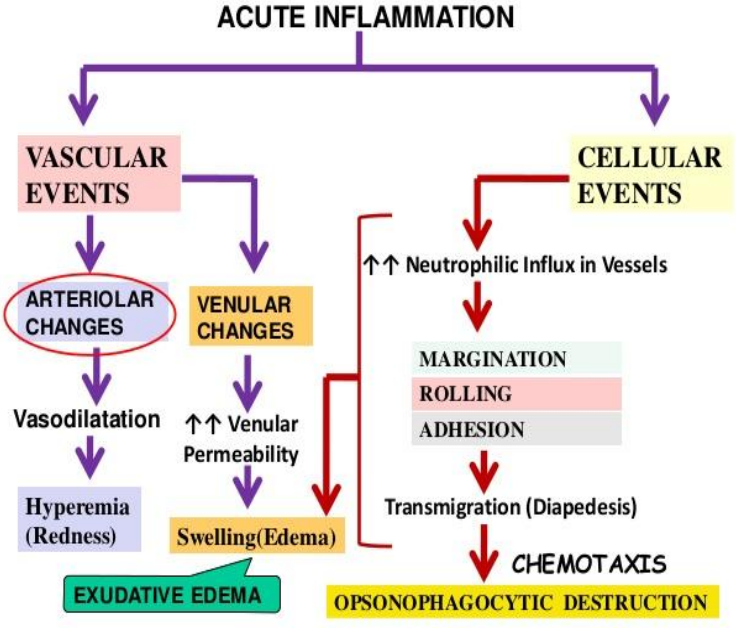
↑ Fluid coming to the area

↓

↑ Hydrostatic pressure (The pressure exerted by a fluid at equilibrium at a given point within the fluid due to the force of gravity)

↓

↑Permeability =↑Gaps between endothelial cells =Accumulation of fluid,cells ,and protein in the tissue =swelling

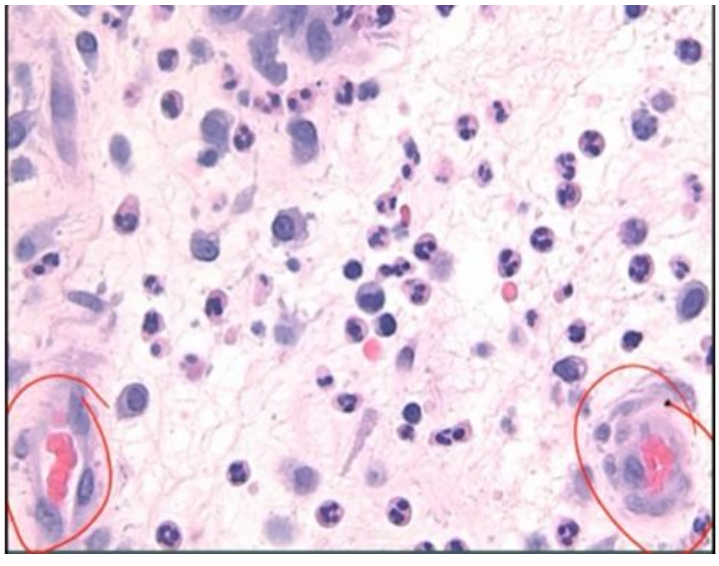


oncotic pressure Colloid osmotic pressure Physiology The colloid pressure in solutions produced by proteins; in plasma, OP counterbalances the egress of fluid from capillaries due to hydrostatic pressure. Is caused by Albumin

1. Hemodynamic changes

Click here

Hemodynamic changes:
Phases of changes in Vascular Caliber and Flow:
1. **Transient vasoconstriction** of arterioles
2. **Vasodilatation**
3. **Slowing of the circulation** (لأن السوائل تطلع من الأوعية للخارج فما يبقى إلا جزيئات لذلك تكون الحركة أقل)
4. **Stasis** (Benefit for move WBC to the injury site)



1

Transient vasoconstriction of arterioles

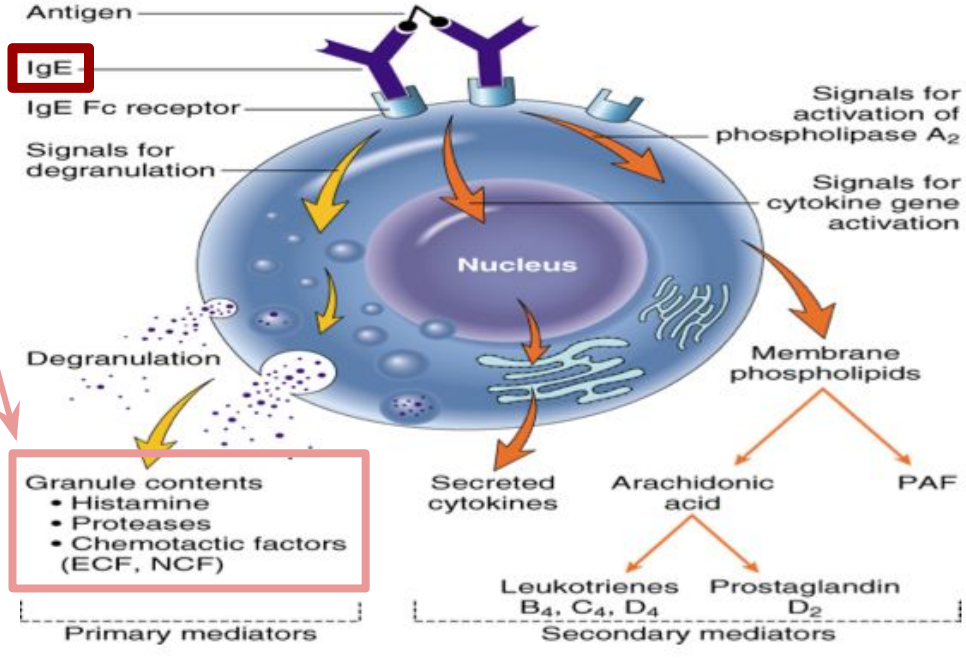
It **disappears within 3-5 seconds in mild injuries**
(Due to a neurogenic reflex that lasts only a few seconds)

2

Vasodilatation

Involves the arterioles results in opening of new microvasculature beds in the area leading to increasing blood flow and cause of redness (rubor) and hotness (calor) in acute inflammation (**Due to Histamine effect released from mast cells** located in interstitial tissue around the small vessels)

Once the injury occurs the granules will open up (Degranulate) and release its contents



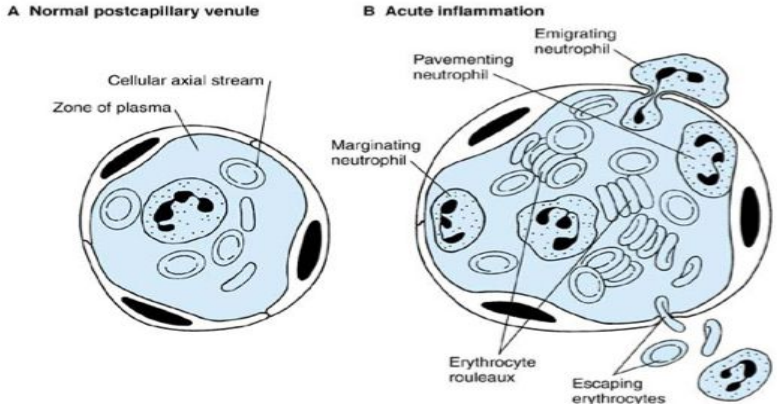
1. Hemodynamic Changes (Cont.)

3

Slowing of the circulation
Due to increased permeability of the microvasculature, this leads to outpouring of protein-rich fluid in the extravascular tissues.
⬆Fluid concentration in the tissue.
⬆Cells concentration in the blood vessels.

4

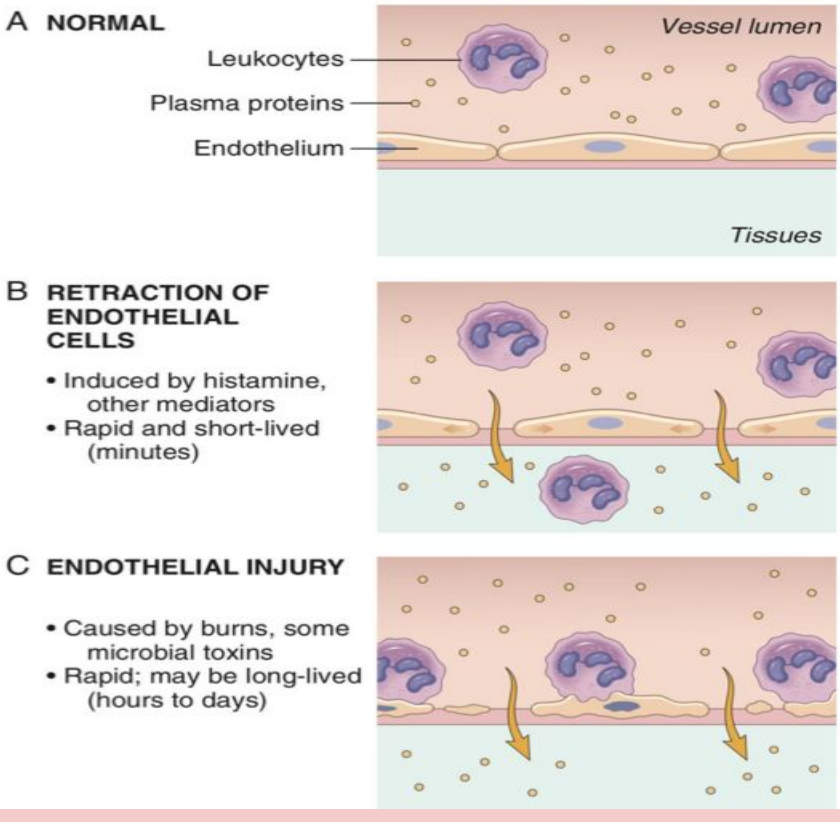
Stasis:
Slow circulation due to dilated small vessels packed with red cells.the viscosity will increase which stops the blood movement and makes it easier for the neutrophils in the middle of the blood vessels to move to the periphery and leave the blood vessels.



2. Increased Vascular Permeability

- **A hallmark of acute inflammation** (escape of a protein-rich fluid) usually the escaped molecules are small molecules but in inflammation they are macromolecules e.g. protein
- **Induced by histamine** (produced in mast cells), kinins (A factor in the blood), and other mediators
- **It affects small & medium size venules**, through gaps between endothelial cells. (mostly in medium size venules)
- It **results in swelling (tumor)** which occurs as a cardinal sign of inflammation

Increased Vascular Permeability (Cont.)



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

Venules

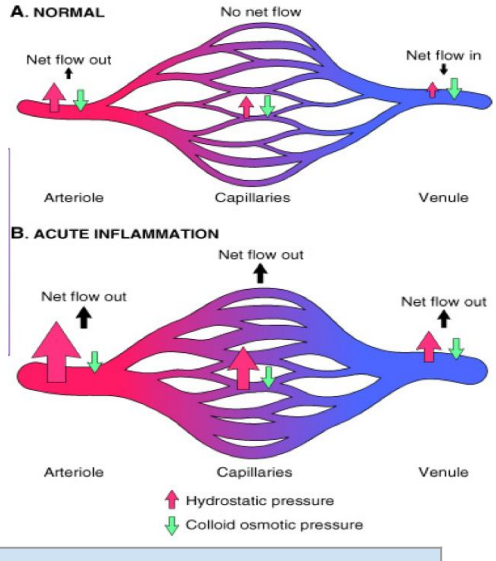
Arterioles, capillaries and venules

Longer lived than the ones affected by histamine

Edema

What is the edema?

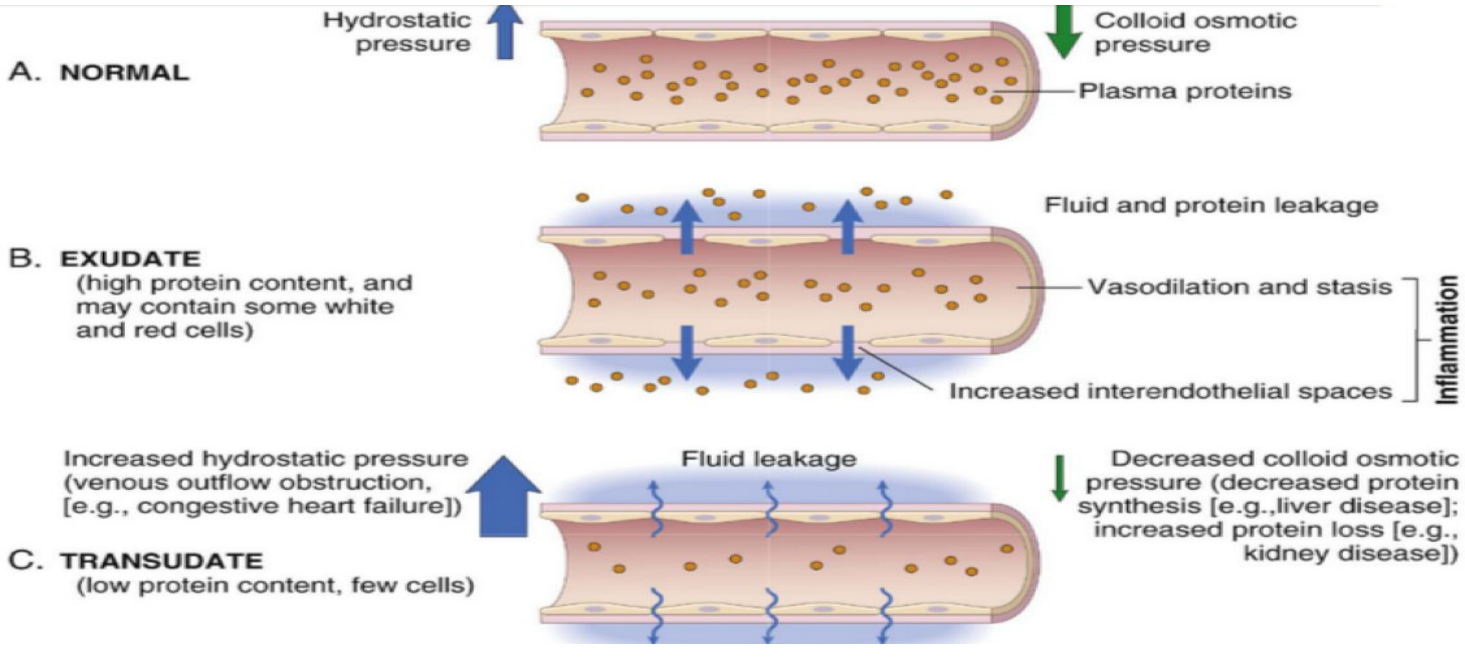
- Denotes an **excess of fluid in the interstitial or serous cavities.**
  Accumulation of fluid in the tissue
- Increased blood volume lead to increased local hydrostatic pressure leading to transudation of protein-poor fluid into the extravascular space.



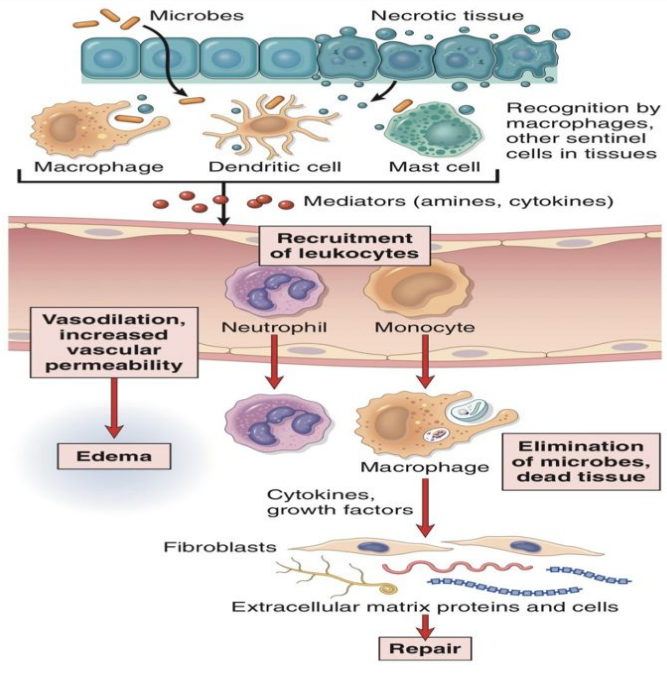
Inflammation can be either:

Exudative	Transudative
An Inflammatory reaction, **rich in protein and cells**.	• **Imbalance** in hydrostatic pressure or in colloid osmotic pressure. • **No protein content**. • Common in pregnant females in the lower limb

Edema (Cont.)



Exudate	Transudate
An **inflammatory extravascular fluid** that has a **high protein** concentration, **cellular debris**, and a specific **gravity above 1.020**	Is a **fluid with low protein** content and a specific **gravity of less than 1.012**
It implies **significant alteration in the normal permeability** of small blood vessels in the area of injury	It is essentially an **ultrafiltrate of blood** plasma that **results from osmotic or hydrostatic imbalance** across the vessel wall
Increased vascular permeability.	**without** an increase in vascular permeability



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★ MCQs

Q1: Which of the following is not a clinical feature of inflammation?			
A. Redness	B. Swilling	C. Itching	D. Warmth
Q2: acute inflammation causes?			
A. Chronic inflammation	B. elimination	C. Edema	D. Don't end
Q3: Which cell secretes Histamine?			
A. Mast cell	B. leukocytes	C. A&B	D. Non
Q4:Increased vascular permeability will results in			
A. Calor	B. Tumor	C. Dolor	D. Functio laesa
Q5: Rich protein fluid in the interstitial cavities is called			
A. Transudate	B. Rubor	C. Exudate	D. Dolor
Q6: Which of the following events involves the arterioles resulting in redness and hotness in acute inflammation			
A. Transient vasoconstriction of the arterioles	B. Vasodilation	C. Slowing in the circulation	D. Stasis

★ SAQ

1.enmarate cells that play a role in inflammation
Slide 3
2.explain the role of stasis in cellular events
Slide 8
3. How does the edema occur
Slide 9

Q1:C Q2:C Q3:A Q4 :B Q5:C Q6:B

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