

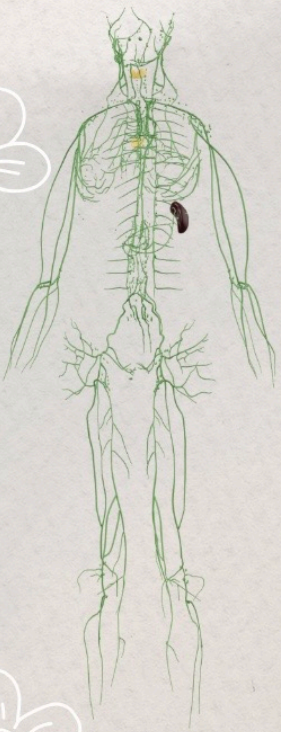
Pathology

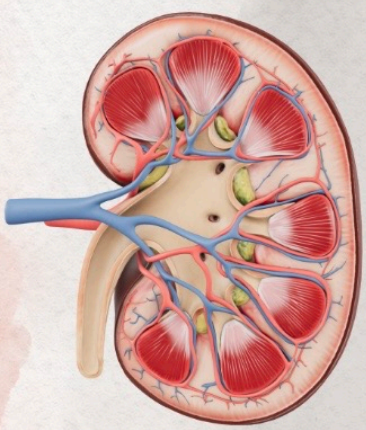
محاضرة 2 د.سامح

Inflammation (Phenomenon of acute inflammation)

Layan







Acute Inflammation

local changes in acute inflammation:

I) Local tissue damage & release of chemical mediators

II)LOCAL VASCULAR CHANGES

III)THE INFLAMMATORY CELLULAR EXUDATE

IV)ACTIVATION OF TISSUE HISTIOCYTES

Systemic (general)change in acute inflammation:

I) leukocytosis

II) fever

I)Local tissue damage & release of chemical mediators:

At the site of injury ,there are some degenerated and necrotic cells. Accompanied by release of chemical substances(chemical mediators) that control the inflammatory response. These mediators may be derived from cells, plasma or bacteria.

Chemical mediators

Types of Chemical mediators:

I- cell derived chemical mediators:

1-vasoactive amines e.g histamine , serotonin

2-arachidonic acid metabolites e.g prostaglandin , leukotrienes

3-cytokines e.g lymphokines , monokines

4-lysosomal enzymes

5-platelet activating factors

II-plasma factors:

1-kinins as bradykinin

2-complement system

3-the clotting system

4-the fibrinolytic system

III- Bacterial products

Effects of chemical mediators:

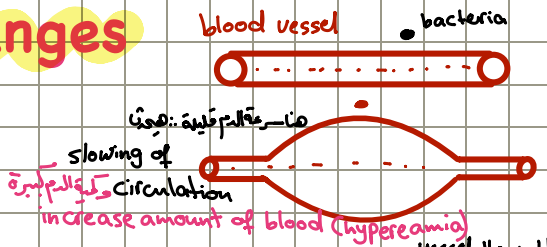
1-Vasodilatation e,g by histamine & prostaglandins

2-Increase vascular permeability e.g by histamine & prostaglandins

3-Chemotaxis

4-Other effect as fever , leukocytosis and pain

II)Local vascular changes



THE VASCULAR phase of the inflammatory response

Including the following sequence:

1-transient vasoconstriction: due to irritation of the vessel wall stimulation of the vasoconstrictor nerves. It lasts for seconds or minutes followed by vasodilation

2-vasodilation: ينشأ عن ← slowing of the blood circulation increase blood flow

Caused by action of chemical mediators as histamine on vessel wall It affects arterioles , capillaries and venules and lead to opening of capillary bed

The increase blood flow (hyperemia) redness & hotness of inflamed area (flare).

3-increase vascular permeability and formation of exudate:

Mechanism

swelling of endothelial cells — widening in gaps in between it

Endothelial cells contraction & retraction (by chemical mediators) widening of the intercellular junction creating gaps between the endothelial cells Endothelial injury, It lead to vascular leakage. ⟶ fluid exudate

4-Vascular slowing(stasis):

this is local slowing of the circulation in the area: It is due to :

-Increase blood viscosity due to vascular leakage. it is most important cause.

Opening of capillary bed.-

It is an important step allowing for leukocyte excudation

It may have an adverse effect since vascular stasis can lead to thrombosis

The vascular changes lead to formation of exudate: Formation of the inflammatory fluid exudate.

(Slowing of circulation & increase amount of blood) vasodilation transient vasoconstriction

(WBCs stick to wall of blood vessels = Margination) vessel WBCs widening of gaps & swelling of endothelial cells

(WBCs pass through gaps in blood vessels = Emigration)

-Acute inflammation steps

1 transient vasoconstriction → 2 vasodilation → 3 margination → 4 emigration → 5 Chemotaxis → 6 Phagocytosis

شو الي بيحدث في ال Acute inflammation ؟

(أول ما ترخى البكتيريا)

(Vascular Changes)

1) blood vessel

أول اشي بيحدث transient vasoconstriction لأنه ال toxin أول ما بيدخل الجسم بيعمل vasoconstriction

2) بعدها بيحدث vasodilation وبيصير increase amount of blood (hyperemia).

slowing of circulation.

3) Swelling of endothelium & widening of gaps.

فبالتالي ال gaps اللي على ال blood vessel بتوسع عشان يصير ↩

Stick to the endothelium = margination بعدها بيجي PMNL بتصير تلتصق

بجدار ال vessel وبعدين بتطلع برا (emigrathion) فبتتوجه على ال bacteria عن طريق ال Chemotaxis

وبتبلعها phagocytosis.

تعريفات مهمة

الخلاصة / شو الفرق بين emigration & margination ؟

- Margination ⟵ White blood Cells (WBCs) Stick to endothelium.
- Emigration ⟵ WBCs leave the blood vessel towards the irritants.

Attraction of white blood Cells to the bacteria (irritant) ؟ Chemotaxis شو هي

(عن طريق ال gaps)

WBCs engulf the bacteria (irritant). ؟ phagcytosis شو هو

inflammatory exudate

definition

Fluids and Cells that pass through walls of blood vessels during process of inflammation.

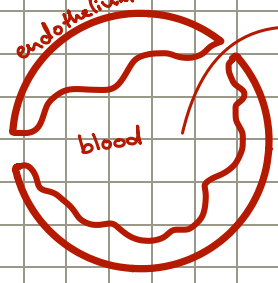
(هو أي حاجة تخرج من ال vessel أثناء inflammation)

hematogenous part — histogenous part

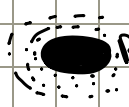
mechanism

(ال vessel بتوسع widening in the poor (gaps) وبصير vasodilation)

(increase permeability) وهاد هيلبي السوائل.



High Osmotic pressure



tissue protien

toxins (بتطلع من البكتيريا) + protien (عبارة عن protien) = protien (بتعمل Osmotic pressure)

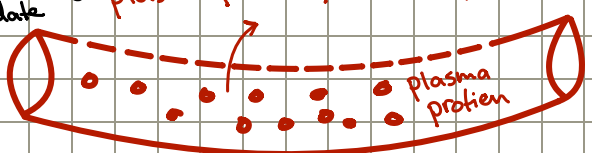
فبالتالي السوائل بتتحرك من الضغط الأسفل للأقل

mechanism هو لل

1) ↑ increase intracapillary hydrostatic pressure (vasodilation بسبب)
2) ↑ increase permeability (swelling of cells بسبب widening of gaps بسبب)
3) ↑ increase tissue osmotic pressure
4) ↑ increase permeability

inflammatory exudate

plasma protien + bacteria + toxins



Characters

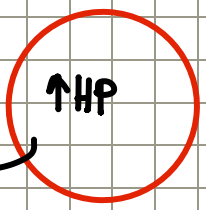
1. high specific gravity > 1018
2. high protien content > 3gm%
3. contain fibronigen (plasma protien) → fibrin network (Clot on standing)
   لو هذا السائل اخذناه برا وعدى عليه تنقية وقت هيتجلط بسبب
4. contain WBCs.
5. Straw Colour.
6. turbid معكّر

*In some Cases:

في حالات مشاكل القلب ← just Hydrostatic pressure increases.

Characters of this fluid in this case:-

- specific gravity (< 1018).
- protien (< 3gm%).
- does'nt clot on standing.
- pale yellow - Clear.



↑HP → water ← transudate بنسميها

mechanism of transudate يعني في هذه الحالة

increase intracapillary hydrostatic pressure. (only)

Mechanism of formation & composition:

1. Increase in capillary hydrostatic pressure (due to increased blood flow)
2. Increased vascular permeability leakage of fluid rich in plasma proteins ① , particular fibrinogen ② , which has a protein content 3gm/dl ③ and specific gravity ④ 1015 . This protein rich fluid is called exudate .it differs from the normal tissue fluid or transudate which has protein 1g/dl & lower specific gravity 1015.

هذه ال characters

3. Reduced intravascular osmotic pressure (OP) & increased interstitial OP; both are due to loss of plasma proteins in the exudate

The amount of fluid exudate :is influenced by many factors such as : Type of tissue,Type of irritant

Functions of inflammatory fluid exudate:

1-dilutes bacterial toxins & chemical irritant

2-brings antibodies to the area as opsonine ,antitoxins , bacteriolysin and other……

3-brings chemical mediators derived from plasma e.g complement

4-contains fibrinogen fibrin (by activation of the clotting system)

نظام التجلّط

Function of fibrin :

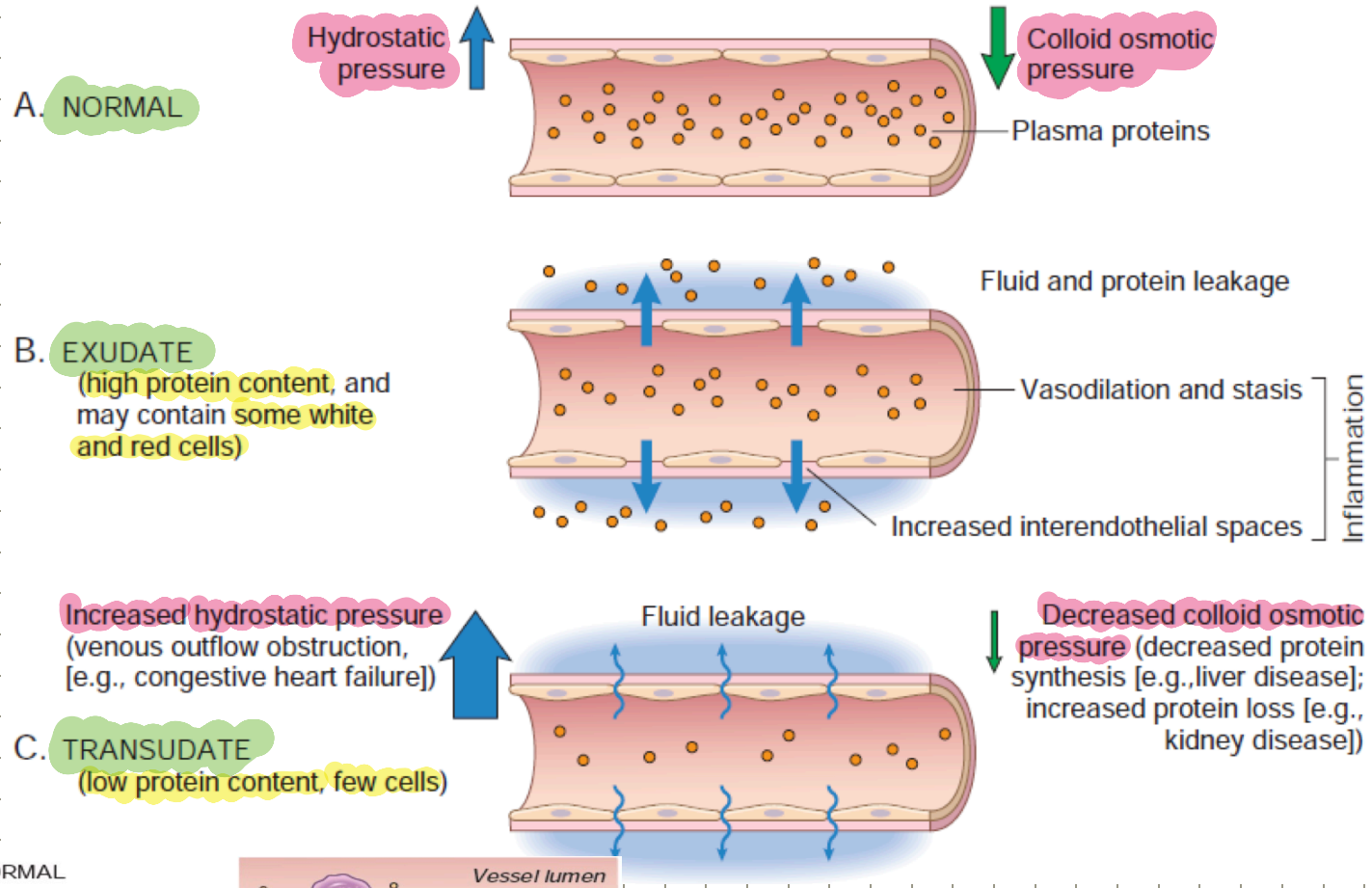
-helps localizing infection by surrounding the inflamed area and block some lymphatics

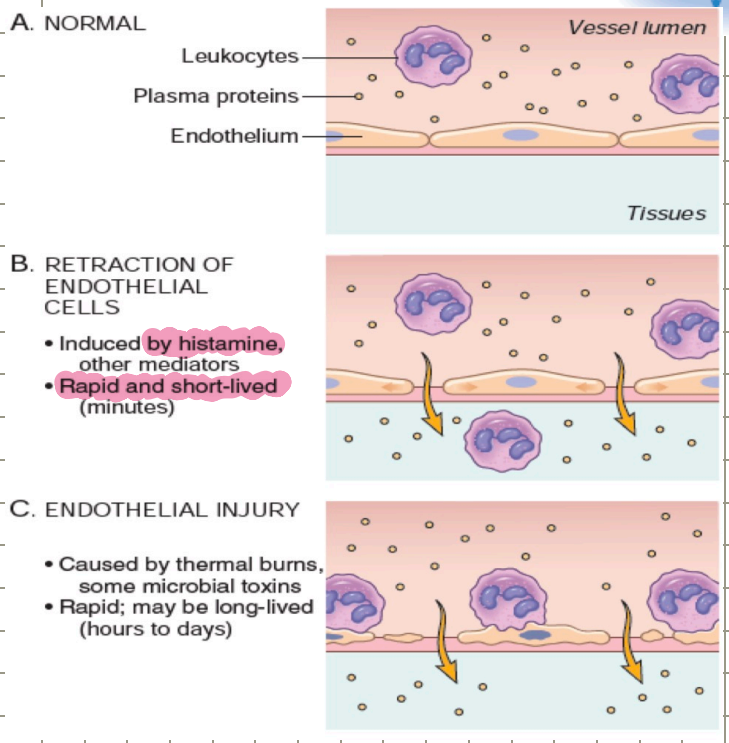
-forms a network upon which phagocytic cells can move towards their target

-fibrin also allows movement of proliferating fibroblast during the process of repair

-supplies nutrients of inflammatory cells

-if finally drains the products of inflammation





Item	Exudate	Transudate
Specific gravity	>1018	<1018
Colour	Straw	Pale yellow
Aspect	turbid	Clear
protiens	>3gm%	<3gm%
Fibrin	rich	no fibrin
Coagulation	present	absent
Cells	rich in inflammatory cells.	minimal or absent.

• Function of inflammatory exudate ?!

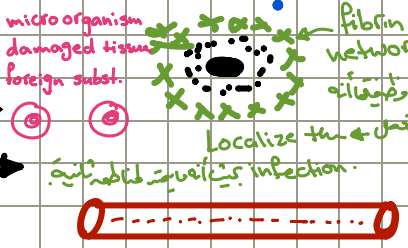
hematogenous part
- Dilute toxins. (by the fluids & protiens of exudate).
- Bring Antibodies to the area of inflammation.
- Localize the infection.
- phagocytosis (prepare the Area for healing process).

histogenous part

healing & repair for the infection

Phagocytosis

Localization



The inflammatory cellular exudate

(THE CELLULAR PHASE OF THE IMFLAMMATORY RESPONSE)

The cellular phase comprises the following steps:

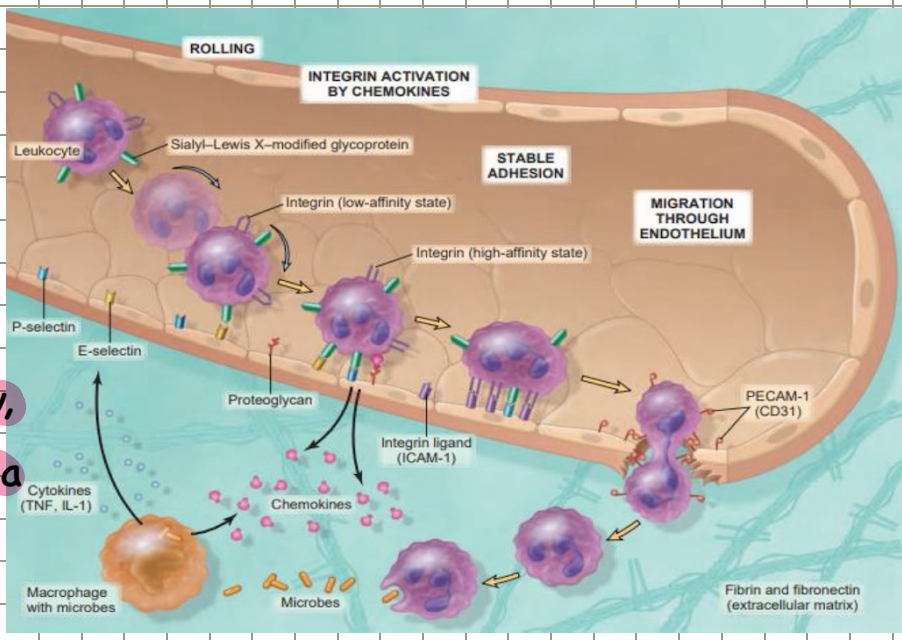
1- Margination and pavementing of leucocytes:

•In normal flowing blood, erythrocytes and leucocytes are confined to the central (axial) column surrounded by plasma.

•When blood slowing occurs, some leukocytes (mainly neutrophils &monocytes fall out of the central column and begin to line the endothelium (margination)

•Adhesion (pavementing) of the marginating leukocytes to the endothelial surface occurs. Adhesion is achieved by complementary adhesive molecules (receptors) on the surfaces of leukocytes and endothelial cells in a way like a key and lock.

These receptors include selectins, immunoglobulins and integrins. Adhesion is very important for leukocyte emigration



2-Emigration of leucocytes (neutrophils &monocytes).

•Once the leukocytes adhere to the endothelium, they gain stability, allowing them to insert pseudopodia into the junction between the endothelial cells, eventually succeeding in traversing the basement membrane and escape into the extravascular space (emigration).

•In most types of acute inflammation, neutrophils (also called polymorphnuclear neutrophils, PMNs or microphages) emigrate first, followed 24 hours later by monocytes (these together with tissue histiocytes are called macrophages). It has to be noted that monocytes emigrate after neutrophils, partly because neutrophils release chemotactic (attracting) factors for monocytes.

3- Passive escape of red cells (diapedesis) :

Occasional passive emigration of red cells may occur, being mechanically pushed by the emigrating leukocytes (diapedesis). No significance is known for this process.

4-Chemotaxis:

•It is the directed movement of emigrating leukocytes to the site of injury. The aim of this movement is to reach the organisms or particles to be phagocytized by these leukocytes