

Acute Inflammation

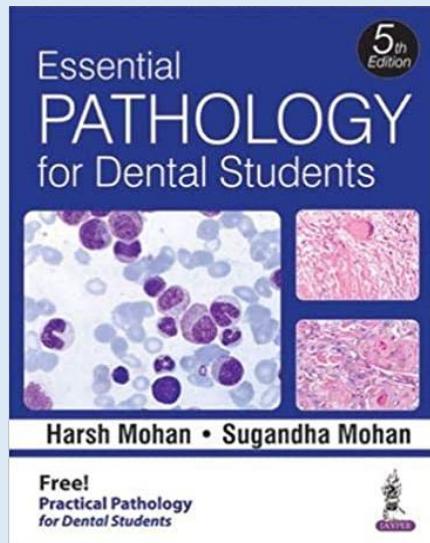


Intended Learning Outcomes:

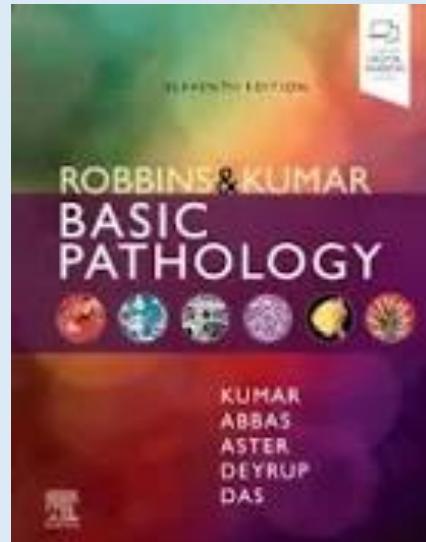
By the end of this lesson, students will be able to:

- 1- Define inflammation and list its local and systemic effects
- 2- Describe the vascular & cellular events of acute inflammation
- 3- Explain the role of chemical mediators (histamine, prostaglandins, cytokines, complement) in the inflammatory response.
- 3- Describe the morphological features of acute inflammation
- 4- Apply knowledge of acute inflammation to interpret its laboratory findings and common clinical conditions as appendicitis, cellulitis and abscess

References



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Inflammation

What is inflammation?

It is protective local response of blood vessels to injury..



Why is it Important?

Inflammation is the first body response aiming to eliminate infections and injurious agents and promotion of immunity and tissue repair.

Is inflammation a disease ?

- No
- Except in autoimmune diseases.

Terminology

Add the Suffix ***-itis*** to the organ: **Appendicitis , Hepatitis , Meningitis**

Exceptions : Pneumonia(lung).



What is the name of inflammation of these parts ?

Oral mucosa , Salivary glands , Cornea , Adipose tissue , Tongue

Stomatitis

Keronitz

Parico

Types of inflammations

- 1- Acute:** rapid onset , short duration *in any place*

2- Subacute : in heart , thyroid , brain, *only*

3- Chronic : Long time , persistent *in any place*



Causes of inflammation

Q) Which of the following cause inflammation?

- 1- • Infections
 - 2- • Chemical agents : smoking
 - 3- • Physical agents : heat, radiation, trauma sun heat
 - 4- • Immune reactions : allergy
 - 5- • Tissue necrosis and foreign bodies

1- Acute Inflammation

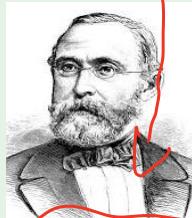
Early and rapid response of body to injury

Local or Cardinal signs of inflammation

- Redness: Ruber
- Hotness: Color
- Swelling : Tumor
- Pain : Dolor
- Loss o function
(Functio laesa)



Celsus



Virchow



Dermatitis
Skin inflammation
Redness, pain, hotness

↓
Symptom

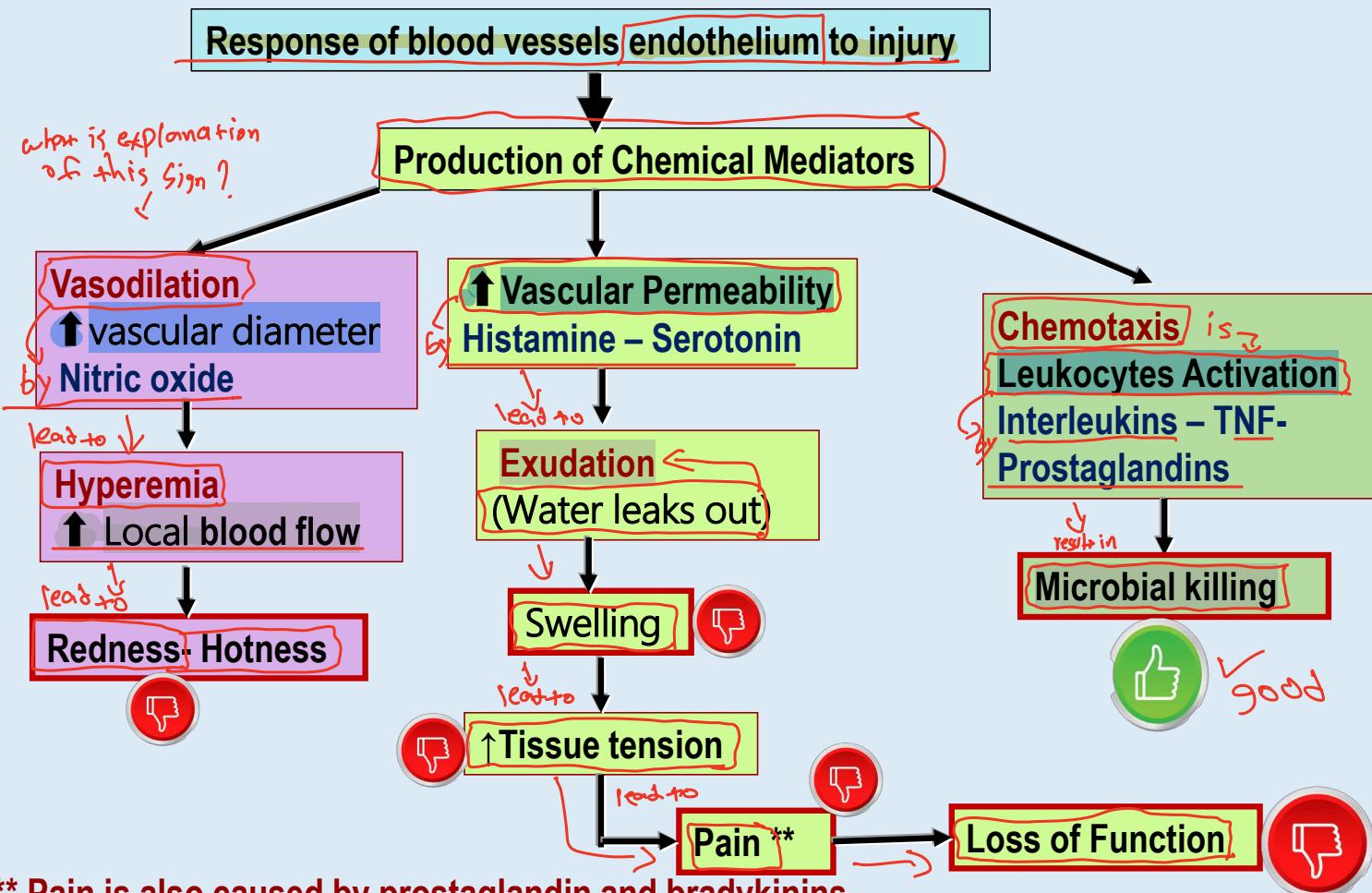


Arthritis
Joint inflammation
Redness , swelling, pain .
Loss of movement

what kind of inflammation?
what sign can be seen?

Mechanism of Acute Inflammation

(Explanation of the cardinal signs)



So we have 3 events in acute inflammatory response

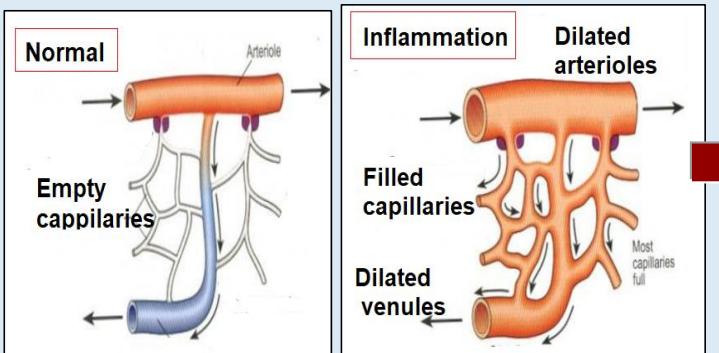
1. Change in vascular diameter → increased blood flow
2. Permeability change → Formation of fluid exudates.
3. Leukocytes migration → Cellular events

1-Vascular changes

Vasodilation

Done by :

- Nitric oxide
- Histamine



Hyperemia

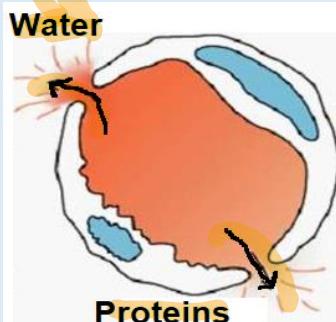
→ Redness

→ Hotness

2-Permeability changes

Done by :

- Early :(histamine, bradykinin)
- Late e (TNF, IL-1,



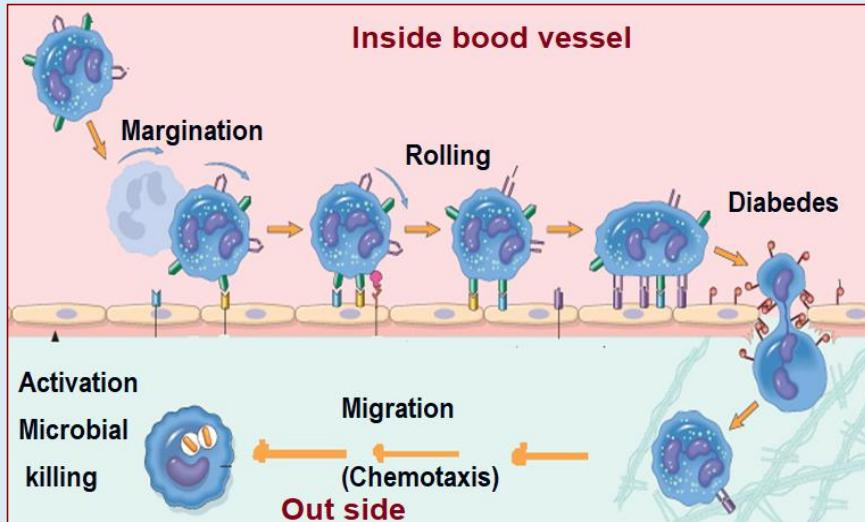
Exudate

→ Swelling

→ Blisters

3- Cellular events

White blood cells migration



↑ WBCs
Leukocytosis



Elimination
of bacteria

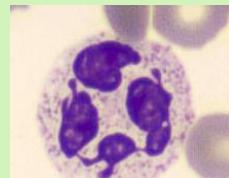
main role

Tissue
damage



what is

Cells in acute
inflammation



The main
cell in
acute
inflammation

Neutrophil: The main cells
Called polymorphonuclear
leukocytes PMN



Monocytes Go to tissue
to become macrophages

main or primary role is kill bacteria

Events	Definition	Mediated by.. <small>Chemical substance</small>
1- Margination and adhesion	WBCs contact and adhere to blood vessel wall	Integrins Cadherins
2- Rolling	Heaping of WBCs on the endothelium	Selectins
3-Diapedesis	Passing of WBCs through pores in vessels wall	CD31
4-Chemotaxis <small>بصوع</small>	Directed movement of WBCs towards area of inflammation	Interleukins C5a, LTB-4
5-Phagocytosis	Eating or engulfment of ^{bacteria} antigen by WBCs	Opsonin (C3b)
6-Microbial killing	Intracellular Lysis of bacterial	Free radicals Lysozymes



In acute inflammation, rolling of leukocytes on endothelium is mediated by which of the following?

- A. Selectins
- B. Integrins
- C. Cadherins
- D. Fibronectin

Inflammatory Mediators

-Chemical substances produced during inflammatory response

- 2 -Sources of mediators: which of the following is a cell derived or plasma derived?
* all is a cell derived except
- a- Cell derived: histamine, prostaglandins, interleukins
 - b- Plasma derived: kinins, complements , from the liver

Mediator	Source	Action
Histamine- serotonin	Mast cells	↑permeability- vasodilation
Interleukins	All white cells	Chemotaxis
Prostaglandins	WBCs and platelets	Fever, pain , vasodilation
Nitric oxide	Endothelial cells	Vasodilation
Bradykinins	Liver	Pain , Muscles contraction
Leukotrienes (LTB-4)	WBCs	Chemotaxis



- Aspirin is an anti-inflammatory drug because it ↓ prostaglandins
- Anti pyretic (fever) , analgesic (pain)

Outcome of acute inflammation

- 1- Resolution or healing
- 2- Progression to chronic inflammation باز ادامه التهاب
- 3- Abscess Formation

Saudi Board style questions:

1- In acute pulpitis, pain is often severe and throbbing. Which of the following mediators is most responsible for pain in acute inflammation?

- | | |
|---|-------------------|
| A- Histamine | B- Interleukin- |
| C- Bradykinin | D- Complement C3a |
| E- Tumor necrosis factor (TNF- α) | |



2- A biopsy from gingival tissue in acute gingivitis shows numerous polymorphonuclear leukocytes (PMNs). Which of the following best explains the primary role of neutrophils in acute inflammation?

- A- Antigen presentation macrophage
- B. Collagen synthesis
- C. Phagocytosis and bacterial killing neutrophil
- D. Fibrosis formation
- E. Release of histamine mast cell

What is inflammatory exudate?

Its fluid leaks during inflammation due to increased permeability:

Composed of water, dead cells, inflammatory cells, proteins and bacteria

What is (pus)?

It is exudates rich in neutrophils and proteins.

What is an (abscess)?

It is collection of pus lined by fibrous wall.



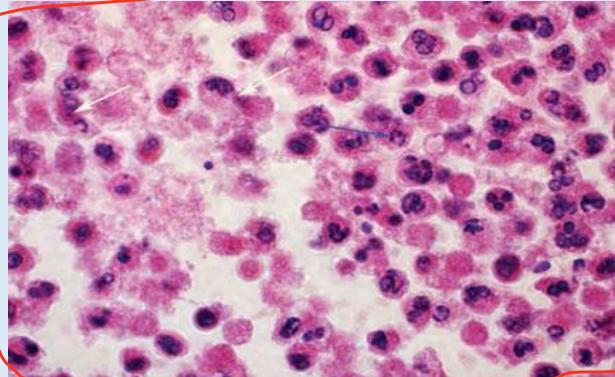
Pus in purulent inflammation



Skin abscess: collection of pus



Dental Abscess : Gross



Dental Abscess : Microscopy

Neutrophils with degenerative features



- A 25-year-old woman presents with a periapical abscess. The pus consists mainly of which of the following ?
- A. Fibrin and plasma proteins
 - B. Dead neutrophils, bacteria, and necrotic debris
 - C. Activated macrophages and lymphocytes
 - D. Collagen and fibroblasts

give a case and ask such as what is the form of acute inflammation?

Morphological forms of acute inflammation

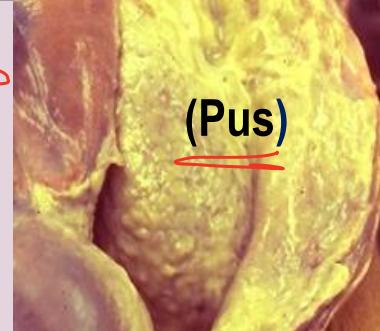
Depending on the exudate contents *5 types*

1- Suppurative (purulent)

- Contains yellow-green pus
- Tissue debris + dead neutrophils

Example

- Pyogenic bacteria infection
- Furuncle



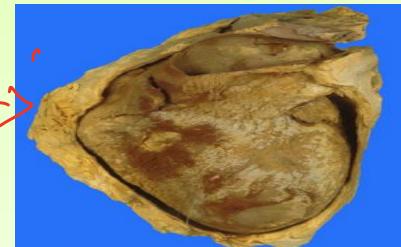
2- Fibrinous

- High Fibrin - No neutrophils

Example

Pericarditis

Severe ulcerative gingivitis



3- Pseudomembranous

Membrane like material- yellow patches

+ fibrin strips

Example

- PM. Colitis (\uparrow antibiotics),
- Acute necrotizing ulcerative gingivitis



PM. Colitis



ANUG

4- Serous

- Thin exudate : ↑ water - ↓ cells and protein
- In body cavities, skin
- Example : Pleural effusion

Skin blisters

Herpes



5- Catarrhal

- ↑ watery discharge with mucin
- In nose, GIT
- Example : Rhinitis (common cold)
Necrotizing ulcerative gingivitis

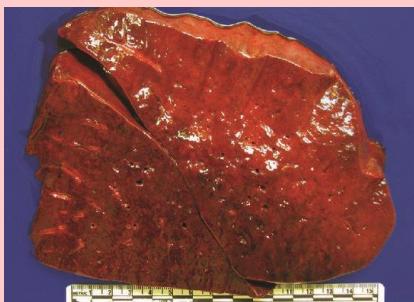
5- Hemorrhagic

- Microvascular injury- Bleeding - ↑ RBCs exudate

Example

H. pneumonia

H. Pancreatitis



Hemorrhagic Pneumonia

Morphological Forms of Acute Inflammation in Dentistry

SEROUS	FIBRINOUS	PURULENT (SUPPURATIVE)	CATARRHAL
			
vater	fibrin		mucin + water
Mild swelling, clear fluid	Yellowish-white coating	Swelling, redness, <u>pus formation</u>	Mucosal swelling, <u>mucus</u> discharge
Early pulpitis herpetic	Severe pericon gngivostas	Dental abscess periapical or periodontal	Necrotizing ulcerative gingivitis

Saudi Board style question:

A patient develops swelling with clear fluid accumulation in the gingiva after mild trauma. Which of the following best describes this type of inflammation?

- A. Purulent or suppurative
- B. Pseudomembranous
- C. Serous
- D. Catarrhal



Q) What are the systemic effects of acute inflammation?

Effect	Pathophysiology
1- Fever	Pyrogens produce by WBCs: prostaglandins(PGE2) and TNF acting on thermostatic center in the brain <i>how fever happen</i>
2-Leukocytosis	Interleukins acting on bone marrow to produce high WBC
3-Anorexia	TNF acting on appetite center <i>ingest</i>
4-Acute phase proteins	Interleukins and TNF acting on liver to produce plasma proteins as C-reactive protein (CRP), ferritin <i>like</i>

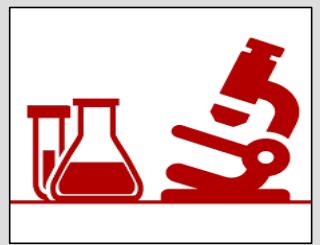
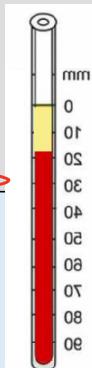
What are the laboratory findings in acute inflammation?

- ↑ WBCs: normal 4000-11000 / cmm
- ↓ Hemoglobin & albumin
- ↑ CRP (normal : 1-3 mg/L)
- ↑ ESR (normal : 10-15 mm/hr)

WBCs : white blood cells count

CRP: C- reactive protein

ESR: Erythrocytes sedimentation rate



ESR tube



1-Which of the following lab test results support (go with) the diagnosis of acute inflammation?

- a-White blood cell count 17000/ cmm (normal 4000- 1100/ cmm) ✓
- b-Serum albumin 5.1 g/dl (normal 3.5- 5.0 g/dl) ✗
- c-C- reactive protein 0.4 mg/dl (normal ; 0.3 – 1.0 mg/dl) ✗
- d-ESR 65 mm/ hour (normal 10- 15 mm/hour) ✓
- e- Neutropenia (low WBC) ✗

2. A 22-year-old dental patient presents with pain, swelling, and redness around the lower right third molar. On examination, the area is hot and tender. Which of the following is the earliest vascular event in acute inflammation . What is the explanation of the redness?

- A- Fluid exudate due to action of histamine
- B- Hyperemia and vasodilatation due to action of Nitric oxide 1
- C- Leukocytes migration
- D- Red blood cells extravasation

**THANK
YOU**



THANK
↑

Questions

- 1- What is the likely diagnosis ? Acute Putulent bacterial tonsilitis
- 2- Explain the pathogenesis of the clinical and lab findings in this case ?
 - a- Fever Prostaglandine acts in the center of the brain
 - b- Pain Prostaglandine, bradikinins, tissue tension
 - c- Tonsillar congestion (redness) and patches hyperemia from vasodilation.
 - d- Tonsillar swelling exudation from vascular permeability
 - e- High WBCs Interleukines act on bone marrow
 - f- High CRP Interleukins act on liver..
- 3- Describe the gross and microscopical feature of acutely inflamed spot
Gross: tissue swelling , hyperemia , with pus collection (abscess)
Microscopy: neutrophils infiltration, tissue edema