

HISTOPATHOLOGY

1

INFLAMMATION



Sawa University

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Department of Medical Laboratories

Third Stage

جامعة ساوة الاهلية

كلية التقنيات الصحية والطبية

قسم تقنيات المختبرات الطبية

المرحلة الثالثة

محاضرة رقم 4

Lecture No. 4

الجانب النظري

Theoretical

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OBJECTIVE

- ☐ **Define inflammation and distinguish between acute and chronic forms.**
- ☐ **Describe the cellular and molecular mechanisms underlying inflammatory responses.**
- ☐ **Detail the morphological (histological) changes characteristic of inflammation.**

Inflammation:

- ❖ is a fundamental physiological process through which the body responds to injurious stimuli—such as pathogens, damaged cells, or irritants—to eliminate the initial cause of cell injury, clear out necrotic cells and tissues, and initiate tissue repair.
- ❖ The term originates from the Latin **inflammare**, meaning “to set on fire”.
- ❖ While inflammation is protective and necessary, when dysregulated or persistent it may contribute to chronic disease or tissue damage.

Terminology

Inflammatory conditions are termed by adding the suffix (-itis) to the affected organ or tissue. For example:-

Pulpitis : inflammation of pulp.

Gingivitis: inflammation of gingiva

³ **Hepatitis**: inflammation of liver.

Appendicitis: inflammation of appendix

Etiology:

- 1. Physical Agents:** Mechanical trauma , extremes of temperature , radiation.
- 2. Chemical Agents and Drugs:** Industrial and occupational hazards, such as asbestos.
- 3. Infectious Agents.**
- 4. Tissue necrosis.**
- 5. Immune reaction.**
- 6. Foreign body(dirt ,suture).**

Classification of inflammation:

1-Acute.

2-Chronic

Acute inflammation	Chronic inflammation
Early onset (sec. – min)	Later onset (days)
Short duration (min. – days)	Longer duration (wks – yrs)
Fluid exudation (edema)	Inducing B.V. proliferation and scarring
Polymorphonuclear leukocyte emigration. (neutrophils)	Involving lymphocytes and macrophages infiltration

Acute inflammation:

Is a rapid host response that serves to deliver leukocytes and plasma proteins, such as antibodies, to sites of infection or tissue injury.

Macroscopical features of acute inflammation:

Swelling , Redness, Hotness, Pain, Loss of function.



Microscopical changes:

1-Vascular changes:

A-Changes in vascular caliber and blood flow.

B-Changes in vascular structure.

2-Cellular changes:

A-Emigration from circulation.

B-Accumulation at focus of injury.

C-Elimination of the offending agent.

Vascular changes

A. Changes in vascular caliber and blood flow

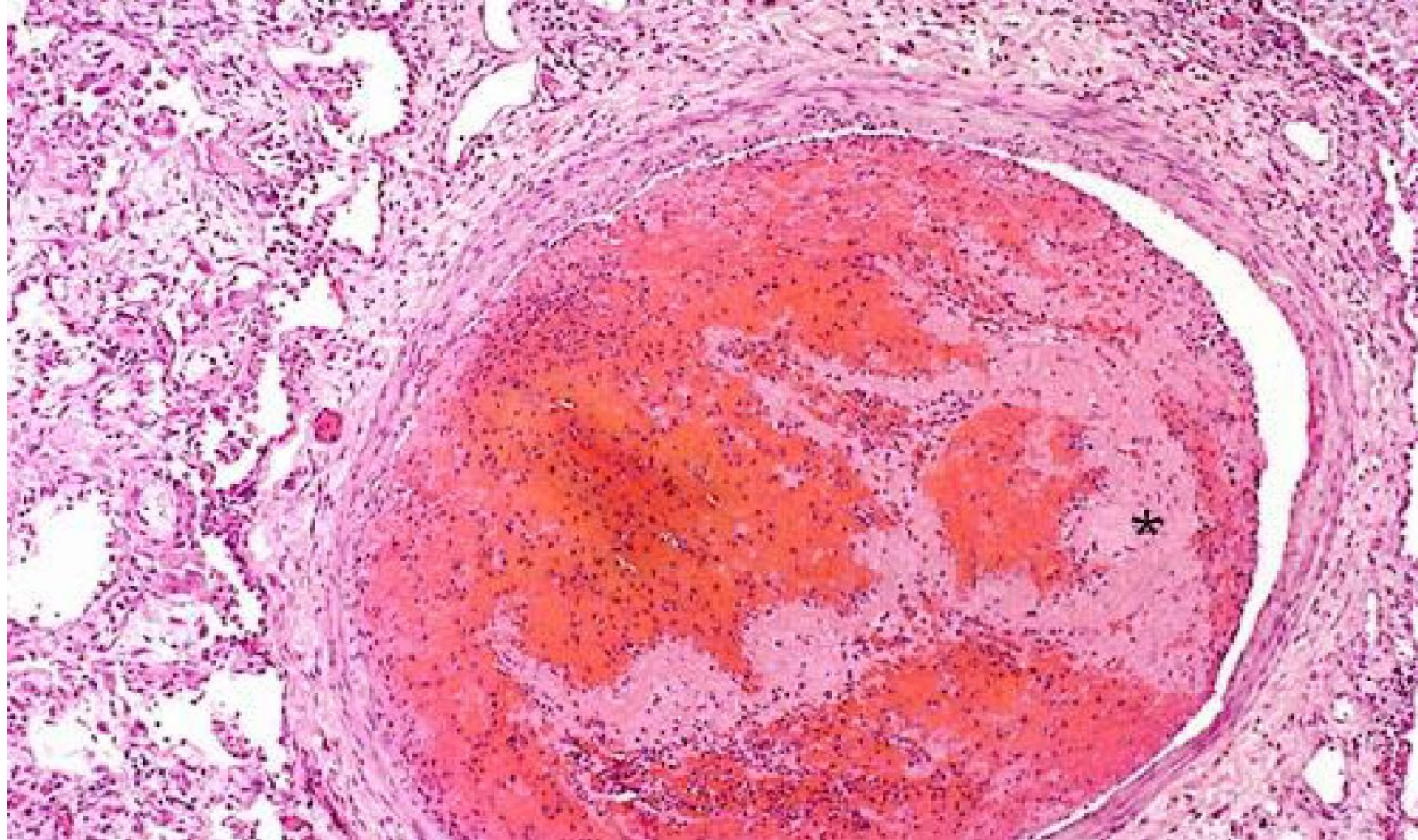
First: transient **constriction** of arterioles.

Second : **vasodilation** which involves the arterioles

Is the cause of heat and redness(erythema).

Third : **slowing** of blood flow (stasis)

Loss of fluid >>>Increased vessel diameter >>>Concentration of red cells in small vessels >>Increased viscosity of the blood.



B. Changes in the vascular structure (increased vascular permeability)

— Protein-rich exudate, edema

- ◆ Several mechanisms are responsible for the increased vascular permeability:
 - **Contraction** of endothelial cells : resulting in increased interendothelial spaces is the most common mechanism of vascular leakage.
 - **Endothelial injury** : resulting in endothelial cell necrosis and detachment.
 - **Increased transport** of fluids and proteins through the endothelial cell (called transcytosis).

Cellular changes:

❖ **Emigration of the leukocytes from the microcirculation:**

Leukocyte Adhesion to Endothelium(Margination>Rolling>Adhesion)

Leukocyte Migration through Endothelium.

❖ **Accumulation of the leukocytes in the focus of injury:**

Chemotaxis of Leukocytes.

❖ **Activation of the leukocytes to eliminate the offending agent:**

Recognition of Microbes and Dead Tissues

11 Removal of the Microbes and Dead Tissues

Phagocytosis

- ❖ Is the engulfment and degradation of the bacteria and cellular debris **by neutrophils and macrophages.**

Phagocytosis involves three distinct but interrelated steps:-

- (1) Recognition and attachment of the particle to be ingested by the leukocyte (adherence and opsonization).
- (2) Its engulfment, with subsequent formation of a phagocytic vacuole.
- (3) Killing or degradation of the ingested material. *(reactive oxygen and nitrogen species and lysosomal enzyme)*

Outcomes of Acute Inflammation depends on:-

- 1-The nature and intensity of the injury.
- 2-The site and tissue affected.
- 3-The responsiveness of the host.

Outcomes of acute inflammation :

1-Resolution : Regaining normal histological & functional tissue. It occurs when there is:

- a- Limited or short lived injury.
- b. Minimal or no tissue damage.
- c. Tissue which is capable of replacing any irreversibly injured cells.

2- Progression to chronic inflammation: Occur if

- a. The offending agent is not removed.
 - b. There is continuing tissues injury.
 - c. There is decreased capacity of the affected tissue to regrow.
- ❖ Chronic inflammation may be followed by restoration of the normal structure and function or may lead to scarring.

3-Suppuration : means pus formation

- ❖ **Pus:** A purulent inflammatory exudate caused by pyogenic bacteria

Pus is a mixture of:-

1. Living & dead neutrophils.
2. Bacteria.
3. Cellular debris.
4. edema fluid

❖ **Abscesses** are focal collections of pus, typically have a central, largely necrotic region rimmed by a layer of preserved neutrophils with a surrounding zone of dilated vessels and fibroblast

4- Scarring and fibrosis

It means replacement of the injured tissue by fibrous connective tissue. This occurs if we have large tissue destruction as in abscess or if the inflammation occur in tissue that do not regenerate.

Chronic inflammation

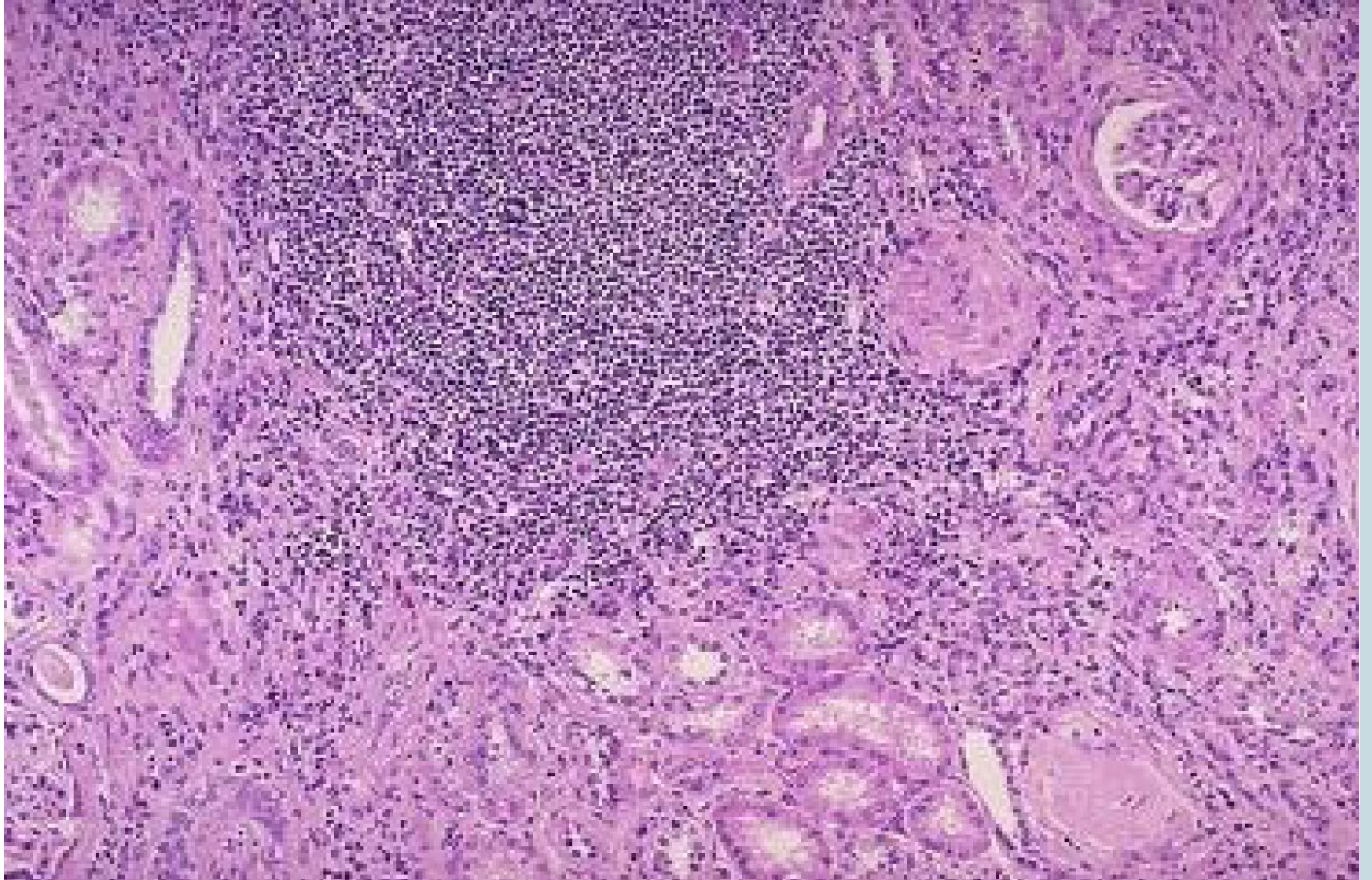
- ❖ is inflammation of prolonged duration (weeks to years) in which
 - continuing inflammation, tissue injury, and healing, often by fibrosis characterized by :

1-Prolonged host response to persistent stimulus

2- Caused by microbes that resist elimination, immune responses against self and environmental antigens, and some toxic substances (e.g., silica); underlies many important diseases

3- Characterized by persistent inflammation, tissue injury, attempted repair by scarring, and immune response

4- Cellular infiltrate consisting of activated macrophages, lymphocytes, and plasma cells, often with prominent fibrosis which Mediated by cytokines produced by macrophages and lymphocytes .



References

1. Kumar, V., Abbas, A. K., & Aster, J. C. (2023). Robbins & Cotran Pathologic Basis of Disease (11th ed.). Elsevier.
2. Mitchell, R. N. (2023). Basic Pathology. Elsevier.

Any questions?

*Thank
You*