INFLAMMATION

- **INFLAMMATION**
  Definition: Inflammation is the local vascular, lymphatic and cellular reactions of living tissue against an irritant for localization and removal of the irritant. Inflammation is designated by adding the suffix “itis” to the name of the organ affected e.g. tonsillitis, appendicitis, gastritis ... etc.

- **CAUSES OF INFAMMATION**
  Inflammation is caused by irritants. Irritants are of different types:
  (1) Living Irritants**:** Bacteria, viruses, parasites and fungi.
  (2) Non Living Irritants: include:
  (a) *Physical irritants:* e.g. excess heat, excess cold and radiations.
  (b) *Chemical irritants:* e.g. acids, alkalis, poisons.
  (c) *Mechanical irritants:* e.g. trauma, mechanical friction.
  (3) Antigens: Cause allergic inflammation.

- **CLASSIFICATION OF INFLAMMATION**
- **Acute inflammation:** Irritant of short duration. The tissue response is rapid. Inflammation lasts for days to weeks. Characterized by the presence of fluid exudate and cellular exudate, polymorphonuclear leucocytes. And classified as:
  I. Suppurative inflammation.
  (1) Localized (abscess)
  (2) diffuse (cellulitis)
  II. Non suppurative inflammation: Includes:
  (1) Catarrhal inflammation.
  (2) Membranous inflammation.
  (3) Sero-fibrinous inflammation.
  (4) Fibrinous inflammation.
  (5) Serous inflammation.
  (6) Hemorrhagic inflammation.
  (7) Necrotizing inflammation.
  (8) Allergic inflammation

- **Chronic inflammation:** Irritant of prolonged action. The tissue response is slow. Inflammation lasts for months to years. Characterized by the presence of lymphocytes, plasma cells, macrophages and fibrosis. And classified as:
  1. **Chronic non-specific inflammation**
  11. **Chronic specific inflammation**

- **ACUTE INFLAMMATION**
  The acute inflammatory reaction consists of:
  I. Local tissue damage.
  II. Local vascular reactions.
  Ill. Local reaction of tissue histiocytes.

I. LOCAL TISSUE DAMAGE

Occurs at the center of the inflamed area and trigger the release of ***chemical mediators*** as histamine, serotonin, prostaglandins and others. These chemical mediators play an important rules for vascular and cellular reaction.

II. LOCAL VASCULAR REACTIONS

(I) Transient Constriction of the Blood Vessels
(2) Dilatation of the Blood Vessels
(3) Slowing of the Blood Stream (Stasis)
(4) Formation of the Inflammatory Exudate **(fluid and cellular)**
(5) Dilatation of lymphatic's

- **The Inflammatory Fluid Exudate**

  Composition: High protein content, 4-8 gm% (the normal interstitial tissue fluid contains 1 gm% protein). The fibrinogen is specially increased (clots on standing). The specific gravity is above 1018. And high cellular content (polymorph and macrophages)

  Functions:

  (1) It dilutes toxins, so minimizes their effects.

  (2) Brings antibodies from the blood to the site of inflammation.

  (3) Supplies nutrition for the cells and carries away waste products.

  (4) Supplies fibrinogen which helps in repair.

  Fate and complication:

  (1) Drained by lymphatic.

  (2) Carry bacteria to lymph nodes causing lymphadenitis

  (3) carry bacteria to blood causing bacteremia, toxemia, and septicemia:

- **The Inflammatory cellular Exudate**
  1- Polymorphnuclear leucocytes (PML) and monocytes become adherent to the endothelial cell of blood vesseles.
  2- Then they pass throw the inter- endothelial spaces outside the vessels by psudobode.
  3- chemotaxis which is the directional movement of PML and macrophages towards the irritant.
  4- Phagocytosis which is the ingestion and destruction of bacteria by phagocytic cells (PML and macrophages).

- **III. LOCAL REACTION OF TISSUE HISTIOCYTES**
  Late in acute inflammation the macrophages replace the polymorphonuclear leucocytes. Macrophages are derived from tissue histiocytes and blood monocytes. They have a longer life span than the polymorphonuclear leucocytes. They phagocytose dead bacteria, necrotic debris, pus cells and fibrin threads cleaning the area of inflammation and preparing the tissue for the repair process.

- **GENERAL CHANGES IN ACUTE INFLAMMATION**
  (1) Leucocytosis: Increase in the number of polymorphonuclear leucocytes in the blood above l0000/cmm.
  (2) Fever (Pyrexia)
  (3) Toxic effect: anorexia, headache, and degeneration of parenchymatous organs.

- **CARDINAL SIGNS AND SYMPTOMS OF ACUTE INFLAMMATION**
  (1) Redness: Caused by vascular dilatation and opening of all the collapsed capillaries.
  (2) Hotness: Caused by arteriolar dilatation and increased blood flow.
  (3) Swelling: Caused by the vascular dilatation and the accumulation of the inflammatory fluid and cellular exudate.
  (4) Pain: Caused by: (a) Irritation of the nerve endings by the chemical mediators. (b) Pressure of the inflammatory exudate on the sensory nerves.
  (5) Loss of function**:** Due to: (a) Pain. (b) Tissue damage.