Pathology 2nd class /

Inflammation

Definition: Inflammation is a complex local response of the living vascularized tissues to injury and mainly consists of responses of blood vessels and leukocytes.

Type of inflammation: Inflammation may be divided into **acute** or **chronic**.

	Acute inflammation	*Chronic inflammation*
Onset	Rapid in onset (usually in minutes or hours)	May follow acute inflammation or be slow in onset (days)
Duration	Short duration. Lasts for hours or a few days	Longer duration; may be months
Predominant cells	Neutrophils (also called polymorphonuclear leukocytes)	Lymphocytes, monocytes/macrophages and sometimes plasma cells
Characteristics	Exudation of fluid and plasma proteins (edema) and the emigration of leukocytes	Inflammatory cells associated with the proliferation of blood vessels, tissue destruction and fibroblast proliferation
Injury/damage to tissue and fibrosis	Usually mild and self-limited and can progress to a chronic phase	Usually severe and progressive with fibrosis and scar formation
Signs: Local and systemic	Prominent	Less prominent

Cardinal Signs of Inflammation

Cardinal sign	*Mechanism*
Rubor (redness)	Increased blood flow and stasis
Calor (heat)	Increased blood flow
Tumor (edema/ swelling)	Increased vascular permeability causing escape of a protein-rich fluid from blood vessels
Dolor (pain)	Chemical mediators: Prostaglandins and kinins

A fifth clinical sign, loss of function, was later added by **Rudolf Virchow.**

Causes of acute inflammation

• **Infections** (bacterial, viral, fungal, and parasitic) and microbial toxins

• **Tissue necrosis**.

– **Ischemia**: e.g. myocardial infarction

– **Physical Agents**

◆ Mechanical trauma: e.g. blunt/penetrating/crush injuries

◆ Thermal injury: e.g. burns or frostbite

◆ Radiation

◆ Electric shock

◆ Sudden changes in atmospheric pressure

– **Chemical injury**: e.g. strong acids and alkalies, insecticides, and herbicides

• **Foreign bodies**: e.g. sutures, talc

• **Immune reactions.**

– **Hypersensitivity reactions**

– **Autoimmune diseases.**

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Outcomes of acute inflammation

• **Resolution**: Complete return of tissue architecture to normal following acute inflammation. It occurs:

– When the injury is limited or short-lived

– With no or minimal tissue damage

– When injured tissue is capable of regeneration.

• **Organization/healing by fibrosis**: Process of replacement of dead tissue by living tissue, which matures to form scar tissue is known as organization. It occurs:

– When there is plenty of fibrin exudation in tissue or serous cavities (pleura, peritoneum) which cannot be removed or cleared.

– In presence of with significant tissue destruction.

– With inflammation in tissues incapable of regeneration.

This process involves growing of connective tissue into the area of tissue damage or exudate, and is converted into a mass of fibrous tissue (scar).

– Abscess: Localized collection of pus is called abscess. If the area of acute inflammation is walled off by inflammatory cells and fibrosis, neutrophil products destroy the tissue and form an abscess.

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• Progression to chronic inflammation: Chronic inflammation may follow acute inflammation, or it may be chronic from the beginning itself. Acute progress to chronic when the acute inflammatory response cannot be resolved. This may be due to:

– Persistence of the injurious agent or

– Abnormality in the process of healing.

Examples:

◆ Bacterial infection of the lung may begin as acute inflammation (pneumonia). But when it fails to resolve, it can cause extensive tissue destruction and form a cavity with chronic inflammation known as lung abscess.

◆ Acute osteomyelitis if not treated properly may progress to chronic osteomyelitis.

◆ Chronic inflammation with a persisting stimulus results in peptic ulcer of the duodenum or stomach, which may persist for months or years.

Chronic inflammation

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Definition: Chronic inflammation is defined as inflammation of prolonged duration (weeks or months) in which inflammation, tissue damage, and healing occurs at same time, in varying combinations.

Chronic inflammation may:

1. Follow an acute inflammation, which does not resolve (e.g. chronic osteomyelitis) or

2. Begin as insidious, low-grade, chronic, response without any acute inflammatory reaction.

Causes of Chronic Inflammation

1. Persistent infections: Microbes that are difficult to eradicate elicit delayed-type of hypersensitivity and produce chronic inflammation, e.g. mycobacteria, and certain viruses, fungi, and parasites. Some agents may cause a distinct pattern of chronic inflammation known as **granulomatous reaction**.

2. Immune-mediated inflammatory diseases

• Autoimmune diseases: e.g. rheumatoid arthritis.

• Allergic reactions: e.g. bronchial asthma.

• Unregulated immune response: e.g. inflammatory bowel disease.

3. Prolonged exposure to toxic injurious agents

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• Exogenous: Silica is a non-degradable inanimate exogenous material. If persons are exposed to silica particles for long time, it causes an inflammatory lung disease called silicosis.

• Endogenous: Atherosclerosis is a disease of arterial intima, probably represents a chronic inflammatory process partly due to endogenous toxic plasma lipid components.

Morphologic Features

Chronic inflammation is characterized by:

• Mononuclear cells infiltrate: Macrophages, lymphocytes, and plasma cells.

• Tissue destruction

• Healing by fibrosis.

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