Acute inflammation

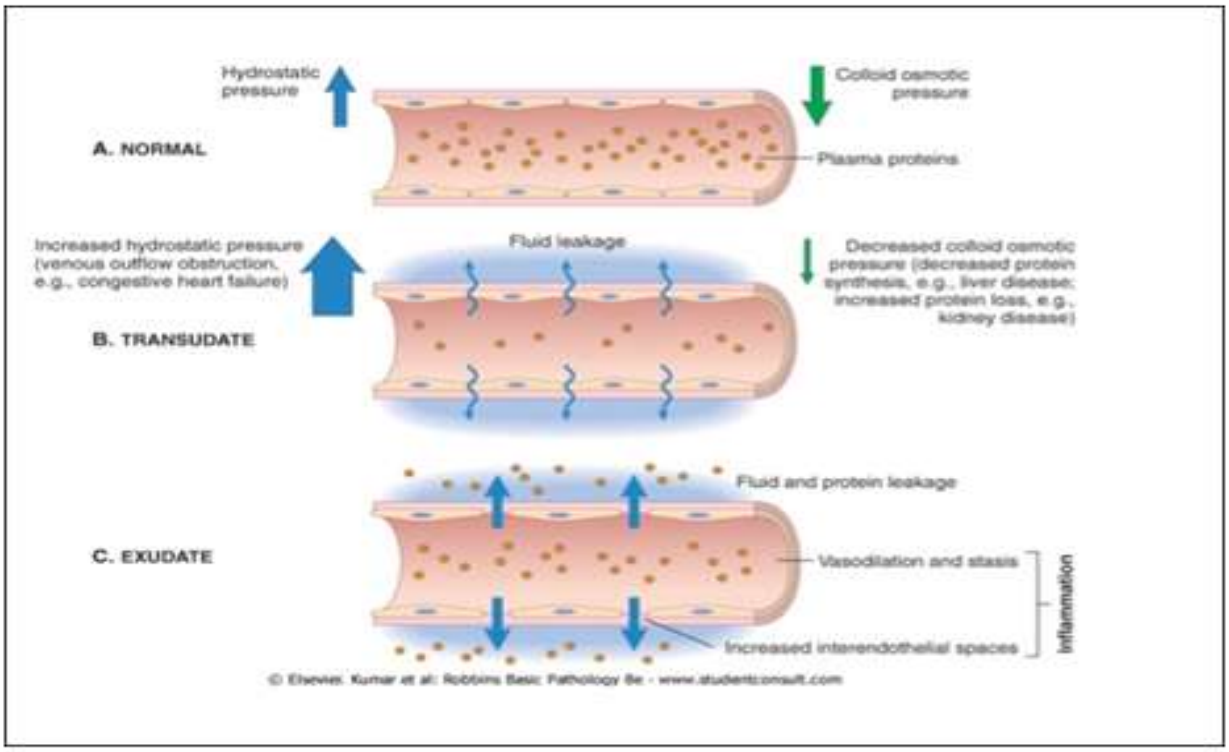
Acute inflammation is a rapid response to cell or tissue injury.

The acute inflammatory response rapidly delivers leukocytes and plasma proteins to sites of injury.
Acute inflammation has two major components:-

• ***Vascular changes:*** alterations in vessel caliber resulting in increased blood flow (vasodilation) and changes in the vessel wall that permit plasma proteins to leave the circulation (increased vascular permeability). In addition, endothelial cells are activated, resulting in increased adhesion of leukocytes and migration of the leukocytes through the vessel wall.

Vasodilation is induced by chemical mediators such as histamine and is the cause of erythema and stasis of blood flow.
• Increased vascular permeability is induced by histamine, kinins, and other mediators that produce gaps between endothelial cells; by direct or leukocyte-induced endothelial injury; and by increased passage of fluids through the endothelium.
This increased permeability allows plasma proteins and leukocytes to enter sites of infection or tissue damage; fluid leak through blood vessels results in edema.

• ***Cellular events:*** emigration of the leukocytes from the circulation and accumulation in the focus of injury (cellular recruitment), followed by activation of the leukocytes, enabling them to eliminate the offending agent.
The principal leukocytes in acute inflammation are neutrophils (polymorphonuclear leukocytes).



Stimuli for Acute Inflammation

Acute inflammatory reactions may be triggered by a variety of stimuli:

- ***Infections*** (bacterial, viral, fungal, parasitic) are among the most common and medically important causes of inflammation.
- ***Trauma*** (blunt and penetrating) and various physical and chemical agents (e.g., thermal injury, such as burns or frostbite; irradiation; toxicity from certain environmental chemicals) injure host cells and elicit inflammatory reactions.
- ***Tissue necrosis*** (from any cause), including ischemia (as in a myocardial infarct) and physical and chemical injury.
- ***Foreign bodies*** (splinters, dirt, sutures, crystal deposits)

Immune reactions (also called *hypersensitivity reactions*) against environmental substances or against "self" tissues. Because the stimuli for these inflammatory responses often cannot be eliminated or avoided, such reactions tend to persist, with features of chronic inflammation.

The term "immune-mediated inflammatory disease" is sometimes used to refer to this group of disorders.

Although each of these stimuli may induce reactions with some distinctive characteristics, in general, all inflammatory reactions have the same basic features.

Table 2–1 Features of Acute and Chronic Inflammation

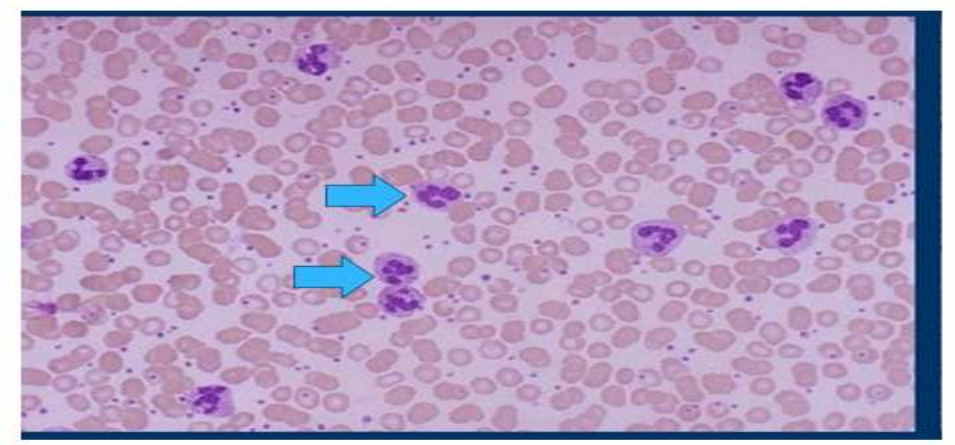
Feature	Acute	Chronic
Onset	Fast: minutes or hours	Slow: days
Cellular infiltrate	Mainly neutrophils	Monocytes/macrophages and lymphocytes
Tissue injury, fibrosis	Usually mild and self-limited	Often severe and progressive
Local and systemic signs	Prominent	Less prominent; may be subtle

Acute inflammatory cells

Polymorphonuclear cells

1) Neutrophil or pus cell (Hetrophil in rabbit and poultry).

The neutrophil (granulocyte) contain a nucleus divided into 2–5 lobes, the cytoplasm have a granules .The granules rich with proteolytic enzymes , this cells active, motile present in large number in the blood circulation the origin of these cell from myeloid tissue of bone marrow and mature inside the bone marrow. It's the best cell in acute inflammation act as a phagocytic cell and attract macrophage. They form part of the polymorphonuclear cell family (PMNs) together with basophils and eosinophils.



2) Eosinophil

Larger than neutrophil has a lobulated nucleus ,eosinophilc cytoplasm, motile cell, less phagocytic than neutrophil ,originated from bone marrow , not proliferate in blood circulation and inflamed area. This cell appear in the parasitic infection and hypersensitivity reaction with basophil because they have enzymes that destroy the histamine to decrease the allergic reaction.

3) Basophil

A granular leukocyte with an irregularly shaped, relatively pale-staining nucleus that is partially constricted into two lobes, and with cytoplasm containing coarse bluish-black granules of variable size, not phagocytic, present very few in blood circulation.

Outcomes of Acute Inflammation

The consequences of acute inflammation are modified by the nature and intensity of the injury, the site and tissue affected, and the ability of the host to mount a response.

Acute inflammation generally has one of three outcomes :-

1) ***Resolution: Regeneration and repair***.

When the injury is limited or short-lived

Before the process of resolution can start, the acute inflammatory response has to be terminated.

This involves neutralization, decay, or enzymatic degradation of the various chemical mediators; normalization of vascular permeability; and cessation of leukocyte emigration, with subsequent death (by apoptosis) of extravasated neutrophils.

Furthermore, leukocytes begin to produce mediators that inhibit inflammation, thereby limiting the reaction.

The necrotic debris, edema fluid, and inflammatory cells are cleared by phagocytes and lymphatic drainage, eliminating the detritus from the battlefield.

Leukocytes secrete cytokines that initiate the subsequent repair process, in which new blood vessels grow into the injured tissue to provide nutrients, growth factors stimulate the proliferation of fibroblasts and laying down of collagen to fill defects, and residual tissue cells proliferate to restore structural integrity.

2) *Chronic inflammation* may follow acute inflammation if the offending agent is not removed, or it may be present from the onset of injury (e.g., in viral infections or immune responses to self-antigens).

Depending on the extent of the initial and continuing tissue injury, as well as the capacity of the affected tissues to regrow, chronic inflammation may be followed by restoration of normal structure and function or may lead to scarring.

3)*Scarring* is a type of repair after substantial tissue destruction (as in abscess formation) or when inflammation occurs in tissues that do not regenerate, in which the injured tissue is filled in by connective tissue.