Acute and Chronic Inflammation
OVERVIEW OF INFLAMMATION

- The survival of all organisms requires that they eliminate foreign invaders, such as infectious pathogens, and damaged tissues. These functions are mediated by a complex host response called inflammation.

- Inflammation is a protective response intended to eliminate the initial cause of cell injury as well as the necrotic cells and tissues.
Inflammation accomplishes its protective mission by diluting, destroying, or otherwise neutralizing harmful agents (e.g., microbes and toxins).

Although inflammation helps clear infections and other noxious stimuli and initiates repair, the inflammatory reaction and the subsequent repair process can cause considerable harm.
The components of the inflammatory reaction that destroy and eliminate microbes and dead tissues are capable of also injuring normal tissues.
The cells and molecules of host defense normally circulate in the blood, and the goal of the inflammatory reaction is to bring them to the site of infection or tissue damage.

Several types of cells and molecules play important roles in inflammation. These include:
- Blood leukocytes.
- Plasma proteins.
- Cells of vascular walls.
- Cells and extracellular matrix (ECM) of the surrounding connective tissue.
Inflammation can be acute or chronic. Acute inflammation is rapid in onset and of short duration, lasting from a few minutes to as long as a few days, and is characterized by fluid and plasma protein exudation and a predominantly neutrophilic leukocyte accumulation.

Chronic inflammation may be of longer duration (days to years), and is typified by influx of lymphocytes and macrophages. However, as we will see later, these basic forms of inflammation can overlap.
All acute inflammatory reactions follow a fairly sequence in which blood vessels and leukocytes are the main participants. When a host encounters an injurious agent (e.g., a microbe) or dead cells, phagocytes that reside in tissues try to eliminate these agents.

At the same time, phagocytes and other host cells react to the presence of the foreign or abnormal substance by liberating several protein and lipid molecules that function as chemical mediators of inflammation.
Mediators are also produced from plasma proteins that react with the microbes or to injured tissues. Some of these mediators act on small blood vessels and promote the efflux of plasma and the recruitment of circulating leukocytes to the site where the offending agent is located. The recruited leukocytes are activated by the injurious agent and by locally produced mediators, and the activated leukocytes try to remove the offending agent by phagocytosis.
The external manifestations of inflammation, often called its cardinal signs, result from the vascular changes and cell recruitment:

- Heat (calor).
- Redness (rubor).
- Swelling (tumor).
- Pain (dolor).
- Loss of function (functio laesa).
The cardinal signs occur as consequences of mediator elaboration and leukocyte-mediated damage. As the injurious agent is eliminated and anti-inflammatory mechanisms become active, the process subsides and the host returns to a normal state of health. If the injurious agent cannot be quickly eliminated, the result may be chronic inflammation.
General Features of Inflammation: Inflammation is a beneficial host response to foreign invaders and necrotic tissue, but it is itself capable of causing tissue damage. The main components of inflammation are a vascular reaction and a cellular response; both are activated by mediators that are derived from plasma proteins and various cells. The steps of the inflammatory response can be remembered as the five Rs:
(1) Recognition of the injurious agent.
(2) Recruitment of leukocytes.
(3) Removal of the agent.
(4) Regulation (control) of the response.
(5) Resolution (repair).

The outcome of acute inflammation is either elimination of the noxious stimulus followed by decline of the reaction and repair of the damaged tissue, or persistent injury resulting in chronic inflammation.
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