Inflammation

Inflammation:

Inflammation is the body's response to tissue damage. Physiologically, it has a defensive function, but it can also have a damaging effect on the body (development of a chronic inflammatory process). It can be caused by **infectious microorganisms**, **chemical** and **physical** influences or by tissue **ischemia**.

The inflammatory response first occurs **locally** (activation of the coagulation, kinin, complement and fibrinolytic system). This is followed by a **systemic** response (fever, leucocytosis, tachycardia, increased acute phase reactants production, release of glucocorticoids). The goal of these processes is to limit the damaged lesion, eliminate the spread of the pathogenic agent, stimulate both nonspecific immunity and specific immunity, induce the lost homeostasis and repair the damaged tissue. Inflammation has an alternative, exudative, proliferative and immune component.

Clinical picture

- **rubor, redness** – is a manifestation of hyperemia of the inflammatory deposit, in which both the amount of blood in its vascular network and the number of capillaries through which blood flows increases,
- **calor, heat** – is due to increased blood flow through the lesion (hyperemia), further increased intensity of catabolic processes and the release of pyrogens,
- **dolor, pain** – caused by biochemical, physico-chemical and mechanical changes in the inflammatory lesion. These are mainly the accumulation of acidic metabolic waste products (tissue acidosis), the formation and release of eicosanoids, increased osmotic pressure and oncotic pressure in the tissue, increased concentration of potassium and hydrogen cations, as well as mechanical pressure of the tissue affecting the nerve endings in the lesion,
- **tumor, swelling** – is related to the increased volume of blood in the lesion and the subsequent outflow of fluid and blood cells from the blood to the tissues (a process called exudation and infiltration),
- **functio laesa, dysfunction** – is caused by tissue damage, disorders of blood and lymphatic circulation and reflexive attenuation of the affected organ.

Local response

There are changes in the microcirculation – **vasodilation**, increased vascular permeability, **migration** of leukocytes from capillaries to tissues. Everything takes place under the influence of adhesive molecules and chemotactic substances (histamine, serotonin, PAF, IL-8, bradykinin, complement, ...) and cytokines. Damage to the blood vessels activates the **coagulation system** and thrombin is produced, which means insoluble fibrin can be produced, bleeding stops and the spread of the infection slows down. Tissue damage also activates the **Hageman factor** (factor XII), which activates prekallikrein to **kallikrein**, which activates the C5 component of **complement**. The products induce mast cell degranulation, and release of histamines and bradykinin, which is a vasodilator that is used to feel pain. Upon activation of the complement system, individual proenzymes are converted into enzymes and their products are effector molecules of inflammation.

Systemic manifestations of inflammation

Leukocytosis is an increase in the concentration of leukocytes in blood above 10,000. It is due to increased production and migration of leukocytes into the blood, demargination and increased rate of transfer from tissues.

In the liver, cytokines stimulate the production of **acute-phase reactants**, which have various functions - they neutralize inflammatory agents, minimize tissue damage, and help repair and regenerate tissue. These include, for example, CRP, α_1-antitrypsin or fibrinogen.

Fever is caused by stimulation of the hypothalamic thermoregulatory center by certain cytokines (TNF, IL-1, IL-6). Tissue metabolism is activated. This leads to the expression of **HSP** (heat shock proteins). These proteins are activated at elevated temperatures or when exposed to stress. They serve as chaperones, bind to newly synthesized proteins and help them assemble into the right shape.

Glucocorticoids are involved in the regulation of acute inflammation. This is important negative feedback.

Repair phase of inflammation

Reparation is activated together with the activation of inflammatory mechanisms. It eliminates damaged cells and activates fibroplastic mechanisms, angiogenesis, tissue regeneration and remodelling. This can lead to organ fibrosis.

Inflammatory cells

- **Neutrophilic granulocytes** are involved in the development of an immune response to damage. Their main function is phagocytosis, which they are unable to repeat and therefore die by apoptosis.
- **Bazophilic granulocytes** are a source of inflammatory mediators and cytokines. Furthermore, they are effector cells in IgE-mediated reactions.
- **Eosinophilic granulocytes** are the main effector cells in allergic inflammation and are involved in the destruction of parasites.
- **Monocytes** are circulating cells that are precursors to tissue macrophages. Macrophages have the ability to phagocytose, they are also antigen-presenting cells, producers of a number of cytokines, growth factors for fibroblasts and endothelial cells. They initiate a whole cascade of immune responses.
- **Endothelial cells** are stimulated by IL-1 and TNFα. They have a procoagulant activity, produce prostaglandins, PAF, induce the expression of adhesive molecules, nitric oxide. Upon activation, the endothelial cell changes its morphology and becomes more rounds, allowing leukocyte diapedesis.
- **Platelets** form the primary hemostatic plug and release a number of inflammatory mediators. They also activate the internal coagulation system.
- **T-lymphocytes and B-lymphocytes** are responsible for developing a specific immune response, producing a number of cytokines.
- **Plasmocytes** produce antibodies.

Septic shock can occur if microorganisms enter the bloodstream massively. **Anaphylactic shock** occurs when inflammatory elements are activated by an intravascular non-infectious stimulus. In both conditions, there is an excessive release of inflammatory mediators, which causes significant vasodilation, which can lead to circulatory failure.

References

Related articles

- Exudative interstitial inflammation
- Exudative superficial inflammation
- Granulomatous inflammation
- Macroscopic manifestations of inflammation
- Microscopic manifestations of inflammation

External links

- Zánět

Literature

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