MECHANISM OF ACTION AND TYPES OF MEDIATORS THAT OCCUR IN INFLAMMATION.

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Abstract: Identifying the etiology of inflammation, the types of mediators that occur during inflammation, the mechanism of the effect of mediators on the body

Key words: inflammation, tissue, mediator, histamine, alteration, exudate.

Inflammation (Latin "inflammation", Greek "phlogosis") is a typical pathological process that develops as a result of tissue damage and is manifested in the form of alterative, exudative and proliferative changes in microcirculation, blood and connective tissue. ladi Inflammation develops in the area of the histion or "organ functional element". Alteration is characterized by tissue damage; Exudation is a violation of blood circulation and the release of fluid, electrolytes and protein from the vessels into the surrounding tissue. Emigration of leukocytes takes a special place in this. Proliferation is the increase of connective tissue cell elements. The external signs of inflammation are known as Celsus - Galen's classic pentad: redness (rubor), swelling (tumor), temperature (calor), pain (dolor), dysfunction (functio laesa). These are local signs of inflammation. Common signs of inflammation include fever, leukocytosis, increased ECHT. Phases of inflammation:

1 - damage (alteration) of the tissue and its constituent cells;

2 - release of biologically active substances (known as inflammatory mediators) - they play the main role in the mechanism of inflammation;

3 - changes in microcirculation (increased permeability of the walls of capillaries and venules);

4 - reaction of the blood system to injury, including changes in the rheological properties of blood;

5 - proliferation - the reparative stage of inflammation (healing of the defect).

Causes of inflammation:

- exogenous factors (physical, chemical, mechanical, biological, etc.). For example, exposure to high and low temperatures, acids, bases, trauma, microorganisms (bacteria, viruses, fungi, etc.);

- endogenous effects. For example, inflammation of the gall bladder, urinary bladder, inflammation of the joint due to the accumulation of salts in them, inflammation of the infarct, hemorrhage, inflammation around tumors. ignition and others.

Pathogenesis of inflammation. The initial or initial link of inflammation is tissue damage - alteration. Primary and secondary alteration are distinguished. Primary alteration occurs at the initial stage of inflammation under the influence of the phlogogenic factor. As the secondary alteration α-inflammation develops, it occurs as a result of the release and activation of lysosomal enzymes, which lead to the damage of macromolecules and the formation of mediators, as well as blood circulation disorders.

In the process of alteration, structural changes and metabolic disorders occur in cells and cell organelles (mitochondria, endoplasmic reticulum, lysosome). According to Shade (1923), "metabolic fire" develops: metabolism is not only increased, but also qualitatively changed, incompletely burned products accumulate, acidosis occurs, biologically active substances - inflammation is formed mediatorially. Changes in metabolism. Its intensity increases, especially in the center of the inflammation. This is due to cell damage and the release of hydrolytic enzymes from damaged lysosomes. During inflammation, incompletely decomposed carbohydrate products (milk, pyruvic acid), incompletely burned fats (fatty acids, ketone bodies) and protein breakdown products (polypeptides, amino acids) accumulate. Mitochondria damage - the morphological substrate of Krebs cycle enzymes - causes disruption of aerobic oxidation and oxidation with phosphorylation. The formation of carbon dioxide decreases, the respiratory rate decreases. Physicochemical changes at the site of inflammation: - due to the accumulation of incompletely burned products, acidosis and H* hyperionia develop; - hyperosmia - the concentration of molecules and ions (NaT, K% Ca2+, etc.) increases as a result of tissue damage at the site of inflammation. In particular, potassium concentration increases, hyperkalemia occurs. All this leads to an increase in osmotic pressure, in the center of inflammation it is 19 atm. enough. A decrease in tissue freezing point is also characteristic. It is normally 0.62, and in inflammation it reaches 1.4; - hyperonicia - increase in oncotic pressure. The release of O protein from the blood vessels and the breakdown of large protein molecules into small molecules lead to an increase in the concentration of protein molecules at the site of inflammation, as a result of which hyperonxia develops; - changes in cell surface tension - associated with the accumulation of fatty acids and other compounds that reduce surface tension. Inflammatory mediators are biologically active substances and are considered the leading pathogenetic factors determining the development of inflammation. Histamine and serotonin produced as a result of degranulation of mast cells (tissue basophils) and the breakdown of platelets, as well as lysosomal enzymes, cationic proteins, cyclic nucleotides, RNA, DNA degradation products, hyaluronidase, lymphokines, E, and E2 prostaglandins, kallidin and bradykinin, complement fragments C2a, C 3a, C5a, etc. enters. Later it was found that bradykinin is the most powerful mediator of inflammation. It is formed from plasma 6-globulin under the

action of the activation product of kallikreinogen - kallikrein enzyme with the participation of Hagemann's factor (blood coagulation factor XII). Depending on their biological nature, mediators can be proteins (for example, permeability factor or globulin, plasmin), polypeptides (for example, bradykinin), biogenic amines (for example, histomin, serotonin). According to the mechanism of action, mediators are vasoactive substances: they change vascular permeability and tone, cause swelling, pain. Emigration of leukocytes, rheological properties of blood, etc. affects. Vascular changes in the focus of inflammation. A. M. Chemukh (1979) and A. I. Strukov (1982) distinguish the following stages of vascular reactions: 1 - short-term vascular spasm and subsequent formation of arterial hyperemia; 2 - venous hyperemia; 3 - stasis. Exudation is the release of the liquid part of blood, electrolytes, proteins and cells from the tissues into the tissue. First, the liquid part of the blood (acellular phase of exudation), then the shaped elements come out (cellular phase of exudation). Causes of exudation: increase in hydrostatic pressure in capillaries, increase in permeability of vessel wall, increase in oncotic and osmotic pressure in tissue. In exudation, fluid escapes through the spaces between endothelial cells. Cytopempsis phenomenon (from the Greek "pem psis" - "transfer") is also important in exudation - active capture of liquid droplets and their transport through the cytoplasm of the endothelium (ultrapinocytosis).

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