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DYNAMICS OF VASCULAR RESPONSE IN ACUTE INFLAMMATION

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Abstract: One of the most important components of inflammation is the vascular reaction, which manifests itself mainly in terminal vessels, arterioles, precapillaries, capillaries and venules. Changes in microcirculation occur in phases. In the first phase, both a transient narrowing of the arterioles can occur as a result of a pain reaction and local release of norepinephrine, as well as their expansion due to the action of the axon reflex, when acetylcholine is released at the endings of the efferent axon.

Key words: enterochromaffin system, collikrein-kinin system.

Visually, an early manifestation of tissue damage is increased blood flow due to dilation of arterioles, with blood initially flowing faster through the arteriovenous anastomoses. Thus, the first phase of arterial hyperemia has a neurogenic mechanism. The second phase is called arterial inflammatory hyperemia, which is of a myoparalytic nature and is accompanied by dilation of resistance vessels and capillaries. The essence of myopalytic inflammatory arterial hyperemia is that the microcircular vessels, primarily precapillaries, having reached maximum expansion, cease to respond to vascular irritation. This phase lasts from 30 minutes to a day, during which redness, warming of tissues, acceleration of blood flow, and an increase in blood pressure in these vessels are observed. During this period, there is a significant increase in the volume and extent of blood flow through the network of capillary vessels due to the expansion of arterioles. At the same time, changes in the capillary wall itself are still insignificant and therefore the extravascular loss of plasma is insignificant. Gradual damage to the vascular wall, as a rule, occurs in parallel with a decrease in tone and the disappearance of spontaneous myogenic activity, which indicates a common mechanism for these phenomena. At the same time, the vessels and, first of all, the precapillary sphinkers become less sensitive to vasoconstrictor mediators (normally, the threshold concentration of adrenaline that causes sphinter contraction is 1:250,000), and then completely stops responding to them. Thus, it is generally accepted that tissue damage is accompanied by dilation of the arterial part of the terminal vascular bed. Inflammatory hyperemia in the area of action of the pathogenic factor, which has a myoparalytic nature, is limited to the lesion.

There are three main reasons for exudation: - increased permeability of the vascular wall - increased filtration pressure in microvessels - increased colloid-osmotic pressure in tissues. Exudation occurs mainly in capillaries and venules and is one of the earliest phenomena during inflammation. Intravenous administration of colloidal paint, ferric chloride, microbial cells, albumin, I131 leads to their rapid removal from the blood and further to the site of inflammation. The increase in the permeability of the medium is due to the rounding of endothelial cells with the formation of gaps between them, as well as increased transport of fluid through the cells themselves. The leading role in the mechanism of increasing permeability belongs to biologically active substances. These include primarily acetylcholine, histamine, kinins, and prostaglandins. Plasma components that come into the tissue from blood vessels, endothelial

cells, mast cells and other cells take part in the origin of these substances. The increase in vascular permeability is also due to the active substances of leukocytes in the tissue during inflammation. Acetylcholine accumulates in tissues not only during inflammation, but also during other pathological processes accompanied by tissue damage. Simultaneously with the accumulation of acetylcholine, a decrease in cholinesterase activity occurs. _G i st a m i n .. 50% of histamine is released in the first 30 minutes after the onset of acute inflammation. Under the influence of histamine, even in low concentrations, vascular permeability increases, and then the precapillary sphinkers, metarterioles and arterioles expand, and the physicochemical properties of the endothelial colloid also change. Under the influence of histamine, there is an increase in the proteolytic activity of the blood, a decrease in the level of protease inhibitors and activation of the collikrein-kinin system.

Histamine is formed in mast cells from histidine under the influence of histidine decarboxylase and is located in granules. In addition, histamine is released from bonds with proteins in connective tissue when they are denatured. Histamine exists in the body in the following forms: bound, labile and free. It is found in a bound state with connective tissue proteins, in leukocytes and other cells and is released when they are destroyed. Labile histamine is released by factors that promote mast cell degranulation. These include toxic substances, tissue destruction products, antigen-antibody reactions, and lysosomal enzymes. In the Free State, histamine is contained in small quantities. The increase in the concentration of free histamine is contained in small quantities. An increase in the concentration of free histamine in the tissue occurs due to its bound and labile forms, due to the primary alteration of the tissue already in the first minutes of the inflammatory reaction. After 1-2 hours, the histamine content reaches a maximum and then decreases due to an increase in histaminase activity in the tissues. Serotonin. (5-hydroxytryptamine) is formed by decarboxylation of tryptophan. The substance has a pressor effect, especially in relation to venules, and also increases permeability more than histamine. Serotonin plays a role in the early phase of increased permeability in animals (rats and mice), in which this mediator is contained in mast cells. In humans, serotonin is mainly found in cells of the enterochromaffin system, especially in the gastrointestinal tract and brain tissue. During inflammation in humans, serotonin is released mainly from platelets, which are contained in large quantities. In the mechanism of the inflammatory reaction, a large role is played by vasoactive peptides. They are characterized by a wide spectrum of actions, cause contraction or relaxation of smooth muscle preparations, stimulate the activity of the heart, dilate the lumens of blood vessels, lower blood pressure, increase blood flow speed, increase capillary permeability, increase diapides of leukocytes, and cause pain.

The most important kinins in humans and mammals include bradykinin and kallidin. Kinins cause a variety of effects in the microcirculation system (hypotension, increased membrane permeability, contraction of smooth muscles, pain effect). The main physiologically active substance of the kallikrein-kinin system, bradykinin, is a nine-membered polypeptide. Other kinins include kallidin and methionyl lysyl bradykinin. In addition to the kallikrein-kinin system of the blood, there are kallikrein systems of internal organs: kidneys, liver, etc. Bradikinin is formed from an inactive precursor - kininogen, which is synthesized in the liver and is a macromolecular polypeptide. Kininogen is hydrated under the influence of the enzyme kallekrein, which has, in addition to keninogenic, also esterase and protease activity. The activity of kallekrein in the blood is low; it is formed from prekallekrein under the influence of Hageman factor (blood coagulation factor XII) or with the direct participation of plasmin. This process is normally limited by protease inhibitors, the activity of which sharply decreases during inflammation in the tissue, and therefore the Hageman factor “turns on” the mechanism of kinin formation. In addition, activated factor XII stimulates the process of blood coagulation and fibrinolysis. The main reasons that cause activation of the callkrein-kenin system are: tissue damage, the influence of toxic metabolites and nuclei, irradiation. Under their influence, the pH of the environment changes, the release of lysosomal enzymes from intracellular organelles, and the appearance of fields of negatively charged membrane surfaces. In addition, when the

tissue oxygen regime is disrupted, ATP deficiency occurs, which leads to the activation of lysosomal enzymes that stimulate the kallikrein system. A similar effect is achieved under the influence of the antigen-antibody complex. There is an opinion that an increase in vascular permeability during inflammation occurs in two phases: the first is caused by the action of biological amines such as histamine, serotonin, and the second by kinins, prostaglandins and other factors. A significant role in the mechanism of increasing permeability - - 34 - is produced by arachidonic acid, which belongs to prostaglandin-thromboxanoic acid, which belongs to the prostaglandin-thromboxane system. They can be synthesized in all tissues of the body, but prostaglandin is predominantly formed by the endothelium of blood vessels and some other tissues, and thromboxanes are synthesized mainly in platelets. These substances affect the rheological properties of the rock. Prostaglandins are strong permeability factors. In addition, local mediators include substances released from cholinergic (acytylcholine) and adrenergic (norepinephrine) nerve endings. An important role in the mechanism of vascular permeability belongs to the enzyme hyaluronidase. In the connective tissue and especially in the pericapillary layer there is a large amount of hyaluronic acid, which is a polymer compound. Hyaluronidase causes depolymerization of this acid and converts it into a low molecular weight compound.

Hyaluronidase is activated by a decrease in pH and, probably, other conditions that occur in the tissue during inflammation. In changes in permeability, a significant role is played by the disturbance of the ionic composition, especially the predominance of potassium in the tissue fluid. Acidosis that develops during acute inflammation causes tissue colloids to swell and become more permeable. Exudation, together with disruption of tissue colloids and restriction of outflow, is the main cause of inflammatory outflow. The meaning of exudation: - exudate reduces the concentration of toxins and thereby weakens their effect on the tissue. - The exudate contains enzymes that destroy toxic substances and lyse necrotic tissue. immunoglobulins are released into the tissue by exudate, which have an antitoxic effect (and antimicrobial), and also have a general protective effect due to the presence of nonspecific protective factors: lysozyme, complement, interferon, beta-lysines, etc. - with exudate in the tissue secretes a large amount of fibrinogen, which turns into fibrin and thus has a protective effect, preventing the spread of the pathogenic factor, mainly through the intercellular spaces. The process of exudation, i.e. The release of the liquid part of the blood beyond the vascular bed occurs simultaneously with the emigration of leukocytes, i.e. the vascular wall becomes permeable not only to high-molecular substances, but also to the formed elements of blood. During this period, in the center of small vessels there is a continuous movement of red blood cells, while the parietal (plasma) layer is filled with leukocytes, which first move along the wall (marginal location) and then seem to stick to the wall, after which, after 2-4 hours, sometimes later, their emigration begins. Externally, in preparations of the mesentery of a frog or rat, it looks like this. On the outer surface of the vessel, small colorless processes (pseudopodia) protrude, which stretch, thicken, form new processes and, finally, the leukocyte separates from the vessel and passes into the tissue. The mechanisms leading to the marginal position of leukocytes were one of the first to study by the Russian pathologist A.S. Shklyarevsky. He set up a model experiment, on the basis of which he expressed the hypothesis that leukocytes, which have a lower specific gravity compared to erythrocytes, when their movement slows down, deviate to the side from the axial layer into the plasmatic layer of Poiseuille. However, subsequent studies showed that the mechanical factor in the migration of leukocytes during inflammation, if at all, is of secondary importance. Attention was also paid to the electrostatic component in the mechanism of leukocyte emigration. Normally, the formed elements of blood and the vessel wall are expressed negatively, which prevents their marginal standing. During inflammation, the charge on the vessel wall decreases, allowing the cell to move closer and then closer to it. The decrease in the electrostatic potential of endothelial cells is due to a decrease in pH in the tissue and the presence of heparin, which is released from mast cells, where it is found in granules in association with histamine. It is possible that calcium and fine fibrin play a significant role in the connection of leukocytes with endothelial

cells. Following the marginal standing, the movement of leukocytes into the tissue beyond the vascular wall begins. Leukocytes move towards the site of inflammation along a broken curve. The speed of movement depends on the type of leukocyte. The number of leukocytes released depends on the form of inflammation: with serrous inflammation there are few of them, with purulent inflammation there are a huge number. The increased influx of leukocytes is supported by an increase in their production in the bone marrow. Most of the leukocytes die, purulent bodies are found in the pus smear, i.e. dead leukocytes are at different stages of their disintegration, but some of the cells slip into the lymphatic vessels and into the lymph draining from the inflammatory focus. The modern concept of leukocyte movement, which explains this phenomenon by chemotaxis, is essentially a development of the theory formulated by I.I. Mechnikov. According to Mechnikov's theory, the movement of leukocytes towards an object is due to the manifestation of their vital properties, i.e. primitive sensitivity chymotakis is one of the forms of manifestation of the vital reaction of leukocytes, and movement is a specific form of excitation of leukocytes. Emigration of leukocytes occurs already in the first minutes of inflammation, then this process intensifies and reaches a maximum after 3-4 hours. In this case, neutrophils come out first, then monocytes and lymphocytes.

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