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Classification Of Inflammation

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Abstract:

This article describes the classification of inflammation, exudate and its types during inflammation, the role of the nervous, endocrine and immune systems during inflammation.

Introduction

Classification of inflammation. Depending on the prevalence of various phenomena in the inflammatory process, alternative, exudative and proliferative inflammation are distinguished.

Alterative inflammation is characterized by a predominance in the tissues of the phenomena of dystrophy, necrosis, necrobiosis, and a very weak exudation and proliferation. It is observed in some of the intoxication and mainly in the parenchymal organs (kidney, liver and heart, less often in the brain).

This type of inflammation can be experimentally caused in rabbits by acting on them with various poisons (cantharidin, bacterial toxins, etc.)

The exudative-infiltration inflammation is characterized by a sharp vascular reaction with the phenomena of exudation and emigration. Depending on the nature of the exudate, inflammations are distinguished: serous, serous-catarrhal, fibrinous, purulent, hemorrhagic and putrid

In serous inflammation, the exudate is a transparent liquid with a density of 1,018-1,020, containing 5-6% protein and a small amount of formed elements; vascular reaction does not reach full development. The process often has a favorable course, since with it the tissue is slightly destroyed, and the exudate quickly resolves; only in some cases (inflammation of the pleura, peritoneum) inflammation takes a protracted nature.

Serous catarrhal inflammation develops on mucous membranes; serous exudate comes to the surface of the tissue with mucus, there are few leukocytes in the exudate.

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In fibrinous inflammation, the exudate contains a large amount of plasma proteins. During exudation, large protein molecules of fibrinogen are released; the latter, behind the vessel wall, coagulates, forming films on the surface of tissues that consist of a fibrin network filled with leukocytes. Inflammation is called croupous if the films easily peel off from the surface of the tissue, and diphtheritic if fibrinous deposits grow deep into the tissues and ulcers form when the films are rejected.

Purulent inflammation is accompanied by accumulation of purulent exudate in the inflammatory focus. It is a thick yellowish liquid with a high content of white blood cells and various tissue elements that are at different stages of destruction. Purulent inflammations are divided into limited and diffuse.

The limited purulent inflammations include: pustules - accumulation of pus in a small area of the malpighian layer of the skin, raising the stratum corneum of the epidermis; abscess (abscess) - accumulation of pus in the cavity formed in the tissues; furuncle - inflammation of the sebaceous glands and hair follicles, limited by the demarcation capsule of fibroblasts; Carbuncle - inflammation of the sebaceous glands and hair follicles.

Spilled purulent inflammation includes: cellulitis - inflammation of the lymphatic vessels and skin glands with uniform placement of pus in the interstitial spaces and lesions of the subcutaneous tissue: empyema - accumulation of pus in the cavities (pleural, pericardial), etc.

In the case of inflammation, red blood cells are mixed with the exudate and it becomes red.

Putrid inflammation is observed when putrefactive bacteria get into the affected focus and putrefactive processes develop in the tissues.

Mostly, there are mixed inflammations - serous-fibrinous, serous-purulent, purulent-fibrinous, etc. Severe forms of inflammation of the mucous membranes - purulent catarrh - are often completed by their atrophy.

Proliferative or productive inflammation is called inflammation, in which the growth of new tissue elements prevails over all other processes. An example of it can serve as a proliferative inflammation in some chronically occurring infections - sapa, actinomycosis, tuberculosis (the so-called infectious granulomas).

Infectious granulomas are proliferations of granulation tissue with the formation of nodules. These nodules, lacking a sufficient supply of nutrients, are usually exposed to necrobiosis and necrosis or calcification.

The cause of proliferative processes is a number of active substances - growth and reproduction stimulants, released during the breakdown of leukocytes and other cellular elements, as well as shifts in osmotic and oncotic pressure in the focus of inflammation. The action of these substances is carried out reflexively, by stimulating the receptors of the affected tissues.

According to the nature of the immunobiological reactivity of the organism, normergic, hyperergic and hypoergic inflammation are distinguished.

Normergic inflammation is a common type of inflammatory reaction that is most common and occurs in the body (non-sensitized) with normal immune properties when it first meets with an inflammatory agent (microbe, toxin, etc.).

Hyperargic inflammation occurs in the body with repeated exposure to a disease agent of antigenic nature. This inflammation is characterized by a violent acute course with pronounced alterative and vascular changes in the tissues; moreover, the inflammatory reaction in terms of

strength and intensity does not correspond to the pathogenicity of the stimuli, but is due to the heightened sensitivity of the organism (its sensitization) to this agent (allergen). Alterative changes in tissues during hyperergic inflammation are often expressed in fibrous swelling or necrosis of collagen fibers and smooth muscle tissue. Exudation is manifested by severe edema, fibrin deposition and suppuration; in exudate are ¬ eatinophils. An example of hyperergic inflammation is allergic reactions (Pirke), the Arthus-Sakharov phenomenon. Elements of hyperergic inflammation are found in acute rheumatism, lobar pneumonia, etc.

Hypoergic inflammation is usually characterized by mild symptoms, it develops in the body, either with immunity to the antigen, or in a weakened, severely depleted, with a sharply reduced reactivity.

Inflammation as a reaction of the whole organism. There is a definite relationship and interaction between the inflammatory focus and the whole organism: on the one hand, the occurrence and development of the inflammatory reaction depends on the reactivity of the organism, the state of its regulatory mechanisms, metabolism, etc .; on the other hand, the resulting focus of inflammation affects the whole body, its metabolism, immune properties, etc.

Neuro-reflex reactions have a great influence on the formation of the inflammatory process. For example, by blocking the receptor apparatus (anesthesia), it is possible to weaken and completely interrupt the course of the inflammatory reaction (process). In denervated tissue, inflammation is asymptomatic, sluggish; stimulation of the sympathetic nerve inhibits the inflammatory process, and parasympathetic intensifies. With chronic stimulation of the gray interdiscipital mound, various inflammatory processes occur in various parts of the body — on the rut, internal organs. In anesthetized animals, as well as during hibernation, when the higher centers (the cortex of the cerebral hemispheres of the brain) are inhibited, disease-causing agents (mustard, lewisite, etc.) do not cause an inflammatory reaction in these animals (under normal conditions a typical inflammation). It is noted that the more complex the organism and the more differentiated its nervous system, the brighter and more fully the inflammatory reaction is, the more pronounced its protective processes in the form of phagocytosis, leukocyte emigration and proliferation occur.

The endocrine system also affects the development of inflammation, for example, when the thyroid gland function is increased, the inflammatory reaction is enhanced, and when it is low, it is weakened, the sex hormones (testosterone, estrogens) have anti-inflammatory properties. The adrenal hormone cortisone inhibits the inflammatory response, and the hormone aldosterone enhances it. The development of inflammation is significantly affected by the hormones of the anterior pituitary: adrenocorticotropic hormone (stimulator of the release of shorticosterone by the adrenal cortex) inhibits the inflammatory process, while growth hormone (growth hormone) stimulates it. Various mediators (histamine, acetylcholine, serotonin, etc.) affect the development of inflammation.

The inflammatory reaction proceeds differently in different ages. For animals of early age is characterized by a flaccid course of inflammation. For example, in rabbits on the 8th – 9th day of life, it is not possible to produce hyperergic inflammation; when exposed to a differential or dysenteric toxin, there is no typical inflammatory reaction (with exudation and emigration of leukocytes, etc.) characteristic of adult rabbits when exposed to them with the same stimuli. In embryos and animals at an early age pneumonia of the lungs sluggishly proceeds, tuberous tubercles are not clearly formed, etc. In these animals during infection, there is a tendency to the spread (generalization) of the pathological process rather than to its localization. This is due to the still weak development of their protective immunological devices, as well as the insufficient activity of their regulatory mechanisms.

The nature of the inflammatory process depends on the type of animal. For example, after the subcutaneous administration of the tubercle bacillus to the guinea pig, a non-healing ulcer is formed at the injection site, and when the same culture is injected into the dog, only the sluggish inflammatory process takes place; in horses, peritonitis (inflammation of the peritoneum) is more common, and the type of inflammation is exudative. In cattle, peritonitis is very rare, and inflammation in most cases is proliferative in nature.

The anatomical and physiological features of the organism also affect the course of inflammation: the more developed the vascular network is in this area, the brighter the picture of inflammation (develops according to the type of exudative inflammation), on the contrary, in the area poor in blood vessels, the picture of inflammation is more obscured.

In the development of inflammation, nutritional conditions and metabolism also play a certain role - lack of protein in the diet reduces the body's resistance to the action of the inflammatory agent; with avita-minosis, inflammation of the eyes and respiratory tract is easily developed. For various avitaminosis is characterized by a sluggish course of the inflammatory process.

Effect of inflammation on the body. The inflammatory process, manifesting itself as a local vascular-tissue reaction, has a very significant effect on the general condition of the body, on its metabolism, immunobiological reactivity, blood composition, thermoregulation and, finally, on the activity of healthy organs.

Local inflammation can cause metabolic disturbances in the body, which is manifested by increased glycolysis and an increase in blood sugar, a change in the albumin-globulin plasma index in the direction of an increase in the globulin fraction; increase in the blood of residual nitrogen, albumin, peptones, histamine, choline substances, nucleic acid metabolism products and acetone bodies. In the blood, the number of leukocytes increases, many immature forms appear, and the erythrocyte sedimentation rate accelerates. Sometimes, when inflammation occurs, an increase in the overall body temperature of the animal occurs.

A change in the immunobiological reactivity of the organism during inflammation is manifested either by an increase in the sensitivity of the organism to the inflammatory agent, or, conversely, by an increase in resistance to it. For example, after suffering inflammation of the lungs, the body's sensitivity to the disease agent often increases, and this disease may occur more often. In some cases, proteins of the inflamed tissue are made by autoantigens and cause the production of autoantibodies, which, in turn, is the cause of the emergence of a new inflammation in the affected organ.

The inflammatory process affects nearby tissue, as well as more distant organs and tissues (intact). An example of the effect of inflammation on intact organs can be the occurrence (sometimes) of peptic ulcer in appendicitis or cardiac arrhythmias in inflammation of the peritoneum. Violation of the activity of intact organs caused by impulses coming from the source of inflammation is often protracted and persists even after the disappearance of the clinical symptoms of the underlying disease.

The effect of inflammation on the entire body is associated with the accumulation in it (as a result of impaired metabolism) of decay products of tissues, microorganisms and their metabolic products, various toxins, metabolites, active and polypeptide substances of nucleic nature, etc. These substances are absorbed into the blood or affecting the receptor apparatus, have a general effect on the entire body, its metabolism, reactivity, the functions of intact organs and systems, etc. They affect the body and pain impulses coming from the source of inflammation. As for the general increase in

body temperature, it is due to the influence of these substances of the inflammatory focus on the thermoregulation centers of the diencephalon. Thus, the influence of the source of inflammation on the entire body is carried out neuro-reflex and neuro-humoral.

The development of views on the pathogenesis of inflammation. Inflammation is a complex reaction of the body to irritation and damage. This reaction has long been observed, but the study of the mechanisms of the emergence and development of the inflammatory process began to take shape only in the middle of the 19th century.

Nutritive, or nutritional, theory of inflammation R. Virchow (1858). According to this theory, the essence of inflammation is to increase the vital functions of cellular elements, in which, under the influence of an inflammatory agent, the metabolism is accelerated and they multiply vigorously; other processes, such as vascular exudative, are secondary and play a secondary role in the development of the inflammatory reaction.

Virchow's views on the pathogenesis of inflammation are one-sided and mechanical, they characterize only individual moments of the inflammatory reaction, and then only at the cell level. In fact, during inflammation in the affected area, phenomena of alterative, vascular exudative and proliferative nature are noted; they coexist simultaneously and mutually each other, although in various stages and types of inflammation one or the other processes may prevail. In addition, advancing the exceptional role of the cellular reaction in the development of inflammation, R. Virchow ignores the importance of the whole organism, its neuro-regulatory mechanisms for the pathogenesis of inflammation.

Congeim's vascular theory (1885) is that the author, in the pathogenesis of inflammation, puts forward vascular disorders (an increase in the permeability of the vascular walls), causing the occurrence of hyperemia, and then exudation and emigration; tissue changes (alteration and proliferation) are secondary and minor. The one-sidedness of this theory is also obvious, because with inflammation, both vascular and tissue disorders are observed simultaneously. In addition, Congeym does not disclose the mechanism of the occurrence of the vascular disorders themselves, the value of reflex influences on the development of the inflammatory vascular reaction, etc.

Ricker's Vasomotor Theory. The author in the development of inflammation plays a major role in the primary dysfunction of the vasomotor nerves. Depending on the degree of irritation of vasomotors, a vascular reaction of varying intensity occurs, which causes the occurrence of hyperemia and stasis, changes in nutrition and metabolism in the focus of inflammation. Although this theory takes into account the role of vascular reflexes in the pathogenesis of the inflammatory process, it is not possible to reduce the complex inflammatory response only to irritation of vasomotors, while ignoring the significance of the disturbance of the trophic function of the entire nervous system, which is typical of inflammation.

Biological, phagocytic theory of inflammation I. Mechnikov (1892). He considers inflammation as a kind of protective reaction of the body to the effects of a pathogenic stimulus, developed in the course of a long evolution. Leukocytes of the blood that have emigrated from the vessels to the inflammatory focus, as well as other local mesenchymal cells that absorb and digest (phagocytosis) the intracellular harmful agent (foreign substances), play a protective role. In highly organized animals with a developed nervous system, the reaction of phagocytosis enters into complex relationships with various functional systems of the body, and especially with the central nervous system, which influence the course and development of this body reaction, the action of a harmful agent. The biological theory of I. I. Mechnikov, although he cannot fully explain the whole complex

pathogenesis of the inflammatory reaction, differs favorably from other theories in that it is based on the principle of evolution; it emphasizes an active adaptive character, bringing it closer to the immune system, showing the role and significance of the whole body (its reactivity) in the development of the local inflammatory process.

Schade's Physico-Chemical Theory of Inflammation (1923). According to this theory, at the basis of the development of inflammation is the primary violation of tissue metabolism. This leads to acidosis, an increase in osmotic and oncotic pressure in the focus of inflammation, which, in turn, causes a local circulatory disorder and various tissue disorders. Representatives of the physico-chemical field have accumulated a number of interesting facts regarding the development of certain aspects of the inflammatory response; However, it is doubtful that the physicochemical changes observed in the inflamed focus would be primary, usually they are recorded at the height of the inflammatory process and are secondary. In addition, supporters of this theory interpret the phenomenon of inflammation as a purely local process, while ignoring the significance of the whole organism, its reactivity, the role of regulatory mechanisms in the genesis of inflammation.

The role of the nervous system in the pathogenesis of inflammation. The question of the role of the nerve system in the development of inflammation was raised at the end of the XIX century. Samuel hypothesized the neurotrophic nature of inflammation. He recognized the possibility of action of the stimulus through the nerves on the cell (except for direct damage to the cell). V. Ya. Danilevskiy drew attention to the development of rapid inflammation in the tissues after transection of the sympathetic nerve that innervates them. Ricker inflammation explained as a result of vasomotor disorders. All these hypotheses and theories reflected a predominantly organolocalistic understanding of the inflammatory process.

Only with the development of Pavlovsky nervism, his views on the role of the nerve system in trophism (nutrition and metabolism) of tissues, the study of the pathogenesis of inflammation took a fundamentally new direction. I.Pav¬lov discovered the appearance of ulcers on the skin and mucous membranes in dogs with fistulas; they arose under the influence of chronic irritation coming from the site of adhesions formed by improper imposition of a fistula on the intestine or stomach. N.I. Simanovskaya observed inflammatory and degenerative processes on the heart muscle after chronic irritation of the gallbladder in a dog. A. D. Speransky caused extensive inflammatory processes and the emergence of trophic ulcers on the skin, mucous membranes and internal organs by prolonged stimulation of the submandibular region of the diencephalon with a foreign body. The inflammatory process arose not only in the organ innervated by the affected nerve, but also outside the segment. For example, inflammation of the cornea occurred during irritation of the sciatic nerve, the cervical sympathetic node, and several centers of the gray bump.

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