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Short Communication

Immunological Reactivity: Pathophysiological Bases of Allergy and Immunodeficiency

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Abstract:

Reactivity is the ability of an organism to react in a certain way to environmental factors. Reactivity is inherent in all living things. The adaptability of the human or animal body to environmental conditions and the maintenance of homeostasis largely depend on reactivity. It depends on the reactivity of the body whether or not the disease will occur when exposed to a pathogenic factor, and how it will proceed. That is why the study of reactivity and its mechanisms is important for understanding the pathogenesis of diseases and their targeted prevention and treatment.

Key words: immunological reactivity; allergy; immunodeficiency

Introduction

Reactivity is the ability of an organism to react in a certain way to environmental factors. Reactivity is inherent in all living things. The adaptability of the human or animal body to environmental conditions and the maintenance of homeostasis largely depend on reactivity. It depends on the reactivity of the body whether or not the disease will occur when exposed to a pathogenic factor, and how it will proceed. That is why the study of reactivity and its mechanisms is important for understanding the pathogenesis of diseases and their targeted prevention and treatment. Reactivity determines the subtle differentiated response of the body to the action of stimuli, determines the quantitative and qualitative characteristics of the response. Reactivity largely determines the ability of a person (or animal) to adapt to changing environmental conditions and maintain homeostasis. Reactivity should be distinguished from the concept of reaction. A reaction is a change in metabolism, structure, and function in response to irritation of the biological system, an expression of reactivity, but not the very property of the body. Reactivity determines the characteristics of the body's response to certain effects. [5] Reactivity as a physiological phenomenon can be divided into species, group, individual, sexual, and immunological. Local reactivity (for example, the peculiarities of carcinogen metabolism in a particular tissue) and general reactivity, which determines the body's holistic response to exposure. [6] Normal and pathological reactivity can also be distinguished. The normal reactivity of an organism is its ability to adequately and differentially, in accordance with the nature of the stimulus, to change its vital activity in response to the effects of external and internal environmental factors.[10] This property is inherent in all living organisms and is an indicator of their normal functioning, allowing them to adapt to environmental conditions. Under the influence of pathogenic factors that cause damage and disruption of homeostasis in the body, pathological reactivity occurs.[1] This reactivity is characterized by a decrease in the adaptability of the ailing organism. It is also called secondary (or painfully altered) reactivity. In fact, the development of the disease is a manifestation of pathological reactivity, which is detected both in individuals and in groups and species of animals. Reactivity can manifest itself in the form of: normal - normergia, increased - hyperergia, decreased - hyperergia (anergy), perverted - dysergia. With hyperergia, arousal processes often prevail. Therefore, inflammation proceeds more rapidly, the symptoms of the disease manifest themselves more intensively with pronounced changes in the activity of organs and systems. For example, pneumonia, tuberculosis, dysentery, etc. occur intensively, violently, with pronounced symptoms, with high fever, a sharp acceleration in the rate of erythrocyte sedimentation, high leukocytosis. With hypergia (decreased reactivity), inhibition processes prevail. Hypergic inflammation is sluggish, unexpressed, the symptoms of the disease are erased, hardly noticeable. In turn, there is a distinction between positive and negative hypergia (anergy). With positive hyperemia (anergy), the external manifestations of the reaction are reduced (or absent), but this is due to the development of active protective reactions, for example, antimicrobial immunity. With negative hypergia (dysergia), the external manifestations of the reaction are also reduced, but this is due to the fact that the mechanisms regulating the body's reactivity are inhibited, depressed, exhausted, and damaged. For example, the slow course of the wound process with sluggish pale

granulations, weak epithelialization after prolonged and severe infection. Dysergia is manifested by an atypical reaction of the patient to any medicine, the effect of cold is vasodilation and increased sweating. Another important concept, which also reflects the basic properties of a living organism, is closely related to the concept of "reactivity" - "resistance". Resistance is the body's ability to resist the effects of pathogenic factors. The body's resistance to pathogenic influences is expressed in various forms. Specific and non-specific resistance can be distinguished. Nonspecific resistance of the body is an innate, nonspecific reaction of the immune system to the effects of foreign agents, such as the barrier function of the skin and mucous membranes, cellular mechanisms such as phagocytosis, the action of leukocytes, and humoral factors, such as lysozyme, interferon, which prevent the development of infection or disease, regardless of their nature. The condition of increased perverse reactivity to substances with or without antigenic properties is called allergy. Allergies are caused by allergens. Allergens are mainly substances of an antigenic nature that can stimulate the pathological reaction of the immune system in an organism sensitive to them. The basis of any allergic reaction is sensitization. Sensitization is an increase in the body's sensitivity to a foreign agent.[15] In turn, an allergic reaction can develop in several stages. The first stage is immunological. This stage is based on the process of active antibody formation during the first two weeks after ingestion of the antigen. This process is called active sensitization. The second stage of an allergic reaction is pathochemical. [4]At this stage, repeated contact with the allergen leads to the activation of immune cells and the release of biologically active substances - inflammatory mediators. Immediate type allergy mediators include serotonin, histamine, bradykinin, heparin, and others. These mediators cause allergy symptoms by destroying or disrupting the functionality of body cells and tissues. The manifestation of clinical symptoms of allergy, in turn, already corresponds to the third, pathophysiological stage. [3] In immunology, there are four types of allergic reactions. The first type is anaphylactic, or reaginic. The reagins, with their end Fs (constant fragment), are fixed on the corresponding receptors of mast cells and basophils, nerve receptors of blood vessels, smooth muscles of intestinal bronchi and shaped blood elements.[12] IgE is synthesized in the lymphatic tissue of the mucous membranes and lymph nodes, therefore, with the reagin type of damage, the respiratory organs, intestines, and conjunctiva are shock organs. This leads to the development of an atypical form of bronchial asthma, hay fever, urticaria, food and drug allergies. The most dangerous consequence of developing this type of allergic reaction is anaphylactic shock. Anaphylaxis manifests itself in different ways for different animals. In turn, in humans, anaphylactic shock involves almost all organ systems. [11] The second type of allergic reaction is cytotoxic. The cause of cytotoxic reactions is the appearance of cells in the body with altered components of the cytoplasmic membrane. The cytotoxic type of immune response plays an important role in the immune response when microbes, protozoa, tumor cells, or expired cells of the body act as antigens. However, in conditions when normal cells of the body acquire autoantigenicity under the influence of damaging effects, this protective mechanism becomes pathogenic and the reaction turns from immune to allergic. In this type of allergic reaction, the antibodies formed to the antigen attach to the cells and cause their damage or even lysis, since the body's cells acquire auto-allergenic properties under the influence of various causes, such as chemicals.[9] The third type of allergic reaction is immunocomplex. This type develops when circulating immune complexes of antibodies and antigens are formed in the body, which settle on the walls of blood vessels or in tissues, causing inflammation, thrombosis and organ damage. This reaction occurs several hours or days after exposure to the allergen and leads to conditions such as systemic lupus erythematosus, rheumatoid arthritis, serum sickness, and allergic vasculitis. The third type of allergic reactions is the leading one in the development of serum sickness, exogenous allergic alveolitis, some cases of drug and food allergies, autoimmune diseases (systemic lupus erythematosus, etc.). With significant complement activation, systemic anaphylaxis develops in the form of shock. [2] A cell-mediated type of allergic reaction, or delayed-type hypersensitivity, the fourth type, occurs due to the activation of sensitized T-lymphocytes, rather than antibodies, as in other types of allergies. This type of reaction is not associated with the formation of immune complexes and does not manifest itself immediately, but several hours or days after contact with the allergen, causing inflammation and tissue damage. This form of reactivity was formed in the late stages of evolution on the basis of immunological reactions and inflammation. It is aimed at recognizing and limiting the action of the allergen. Type IV immune damage underlies many allergic and infectious diseases, autoimmune diseases, transplant rejection, contact dermatitis (contact allergy), and antitumor immunity. The prototype of this form of response is a tuberculin test used in the diagnosis of tuberculosis. With the gradual introduction of the allergen and its constant contact with the body of a person sensitive to this allergen, desensitization may occur. Desensitization in immunology, also known as immunotherapy, is a procedure aimed at reducing or completely eliminating an allergic reaction in humans. It is based on the gradual habituation of the body to the allergen that causes the reaction. This process involves the introduction of increasing doses of the allergen into the body, which eventually leads to a decrease in sensitivity to it. Desensitization can be divided into specific and nonspecific. Specific desensitization is the introduction of allergens to which there is hypersensitivity. Non–specific desensitization is an approach to the treatment of allergic reactions, which, unlike specific desensitization, is not aimed at affecting a specific allergen. Instead, non-specific desensitization methods seek to reduce the body's overall sensitivity to allergens, as well as reduce the severity of allergy symptoms. This approach focuses on strengthening the immune system, reducing inflammation, and controlling allergic reactions in general. Immunodeficiency is a condition in which the immune system does not function properly. They can be primary (genetic, congenital) or secondary (acquired). With immunodeficiency, the immune system's reactivity to pathogens (viruses, bacteria, fungi, parasites) and other antigens is reduced. This means that the immune system cannot effectively recognize, attack, and destroy threats, leading to increased susceptibility to infections, autoimmune diseases, and even cancer. Thus, immunodeficiency is a typical violation of immune reactivity. As mentioned earlier, immunodeficiency is divided into primary and secondary. Primary immunodeficiency is quite rare. They are caused by defects in a single gene or entire clusters of genes responsible for the synthesis of protein molecules of the immune system. Manifest forms of primary immunodeficiency conditions are manifested in the first or second year of life by pustular lesions of the skin and mucous membranes, recurrent infections of the respiratory system, urinary tract, and intestines, which in some cases (severe forms), in the absence of adequate therapy, can lead to death. However, if we consider the problem more broadly, taking into account poorly diagnosed minor forms, and take into account the imbalance of the population sets of HLA and the most common pathogens in the habitat of this population (on which the strength of the immune response depends), we can state that for the most part all

immunodeficiencies are primary. Many of the primary immunodeficiencies are X-linked, i.e. they are inherited mainly by boys from their mothers. Primary immunodeficiency can include specific and nonspecific immunodeficiency. [8] Non-specific ones include deficiency of the phagocytosis system and defects in the complement system proteins. Specific primary immunodeficiencies include selective immunoglobulin deficiencies. IgM and IgG deficiency is associated with a high sensitivity of animals to pyogenic microflora. IgA plays an important role in protecting the mucous surfaces (respiratory and gastrointestinal tracts), inhibits the adhesion and colonization of viruses and bacteria, and limits the absorption of pathogens from the gastrointestinal tract. IgA deficiency predisposes to bacterial and viral infections. Secondary immunodeficiency is caused by quantitative and qualitative starvation (lack of proteins, vitamins, trace elements Fe, Zn, Cu, etc.), endocrinopathies (diabetes mellitus, Itsenko-Cushing's disease), loss of immunocompetent cells and molecules during injuries, bleeding, surgery, burn disease, loss of organs of the immune system, exposure to ionizing radiation, polychemotherapy, hypertoxic infections, and infections to which the human immune system has proven to be evolutionarily unprepared, such as HIV, etc..[14] Acquired immunodeficiency syndrome is a classic example of this kind of pathology. The causative agent of the disease, HIV, has a selective affinity of one of its shell proteins (gp120) to the CD4 molecule, which is expressed by helper T lymphocytes. [13] With AIDS, T-cell immunity is sequentially turned off, which leads to the activation of opportunistic infections and other disorders of the immune system, including tumor diseases (Kaposi's sarcoma). AIDS (acquired immunodeficiency syndrome) is the final stage of HIV infection in which the immune system is severely weakened, making the body extremely vulnerable to opportunistic infections and certain types of cancer. [7] This is not a single disease, but a complex of signs and symptoms resulting from the devastating effects of the human immunodeficiency virus (HIV) on the immune system. The prospects for the treatment of immunodeficiency states are associated with the replacement of missing components (administration of immunoglobulins, bone marrow transplantation, etc.), the appointment of immunotropic drugs (in minor forms), and gene therapy. Studying the interrelationships between various pathological reactions of the body to an irritant, as well as studying the pathogenesis of these reactions, is of paramount importance for understanding the complex mechanisms of the immune system and developing effective strategies for the prevention and treatment of diseases.

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