VOLGOGRAD STATE
MEDICAL UNIVERSITY
Department of Pathological Anatomy

Immunopathological processes.

Purpose: to study the causes, characteristic signs, significance for the body and mechanisms of immunopathological processes.

Requirements for the level of the student for mastering the discipline - pathological anatomy.
1. What phenomena are covered by the subject of immunopathology.
2. To characterize the central and peripheral organs of the immune system,
3. What refers to cellular immune responses.
4. Describe humoral immune responses.
5. Changes in the thymus to antigenic stimulation.
6. Allergy, its types and characteristics of allergens.
7. The concept of drug allergy.
8. Allergy of immediate type, forms, stages, mechanisms and outcomes.
9. Delayed allergy, forms, stages, mechanisms and outcomes.
10. What does the concept of autoimmunization include?
11.Infection-dependent allergy

Brief inf
1) hypersensitivity reactions, which are mechanisms of immunological tissue damage in a number of diseases;
2) autoimmune diseases, which are immune responses against one's own body;
3) syndromes of immune deficiency arising from a congenital or acquired defect in the normal immune response;
4) amyloidosis.

Regardless of what type of damage is a hypersensitivity reaction (allergic reaction), three stages can be distinguished in its development.
1. Stage of immune reactions. It begins with the first contact with an allergen and consists in the formation of allergic antibodies in the body and their accumulation. As a result, the body becomes sensitized or hypersensitive to a specific allergen. When a specific allergen re-enters the body, the formation of AG-AT complexes occurs, which determine the next stage of the allergic reaction.
2. Stage of biochemical reactions. Its essence consists in the release of ready-made and the formation of new biologically active substances as a result of complex biochemical processes triggered by the AG-AT complexes or the AG-sensitized lymphocyte.
3. The stage of clinical manifestations. It represents the response of cells, organs and tissues to the mediators formed in the previous stage.
Type I hypersensitivity reactions (anaphylactic type) can develop locally and be systemic. The systemic reaction develops in response to intravenous administration of an antigen to which the host's body is previously sensitized. Local reactions depend on the site of penetration of the antigen and have the character of skin edema (skin allergy, urticaria), nasal and conjunctival discharge (allergic rhinitis and conjunctivitis), hay fever, bronchial asthma or allergic gastroenteritis (food allergy).

Type I hypersensitivity reactions occur in two phases in their development. The phase of the initial response develops 5-30 minutes after contact with the allergen and is characterized by vasodilation, increased permeability, as well as spasm of smooth muscles or glandular secretion. The late phase is observed after 2-8 hours without additional contacts with the antigen and lasts for several days. It is characterized by intense tissue infiltration with eosinophils, neutrophils, basophils and monocytes, as well as damage to the epithelial cells of the mucous membranes.
The development of type I hypersensitivity in humans is provided by IgE. Mast cells and basophils sensitized with the Fc-fragment of IgE and basophils activate the complement

components C3a and C5a (anaphylotoxins). The secretion of mast cells is also stimulated by macrophage cytokines (IL-8), some drugs (codeine and morphine) and physical influences (heat, cold, sunlight). Binding of IgE molecules initiates degranulation of mast cells with the release of primary mediators, as well as de novo synthesis and release of secondary mediators, such as arachidonic acid metabolites. The emergence of new symptoms of type I hypersensitivity reaction is associated with these mediators.

Histamine and leukotrienes are rapidly released from sensitized mast cells and basophils, providing an immediate response characterized by mucosal edema, mucus secretion, and smooth muscle spasm. Many other mediators, represented by the platelet activating factor (PAF), TNF□, are included in the late phase of the response, increasing the number of basophils, neutrophils, and eosinophils. Among the cells that appear in the late phase of the reaction, eosinophils are especially important. Their set of mediators is as extensive as in mast cells. In addition, they produce a major basic protein and eosinophilic cationic protein, which are toxic to epithelial cells.

Systemic anaphylaxis occurs after administration of heterologous proteins such as antisera, hormones, enzymes, polysaccharides, and some drugs such as penicillin. The severity of the condition depends on the level of prior sensitization. The antigen shock dose, however, can be extremely small. Local anaphylaxis is sometimes called atopic allergy. About 10% of the population suffers from local anaphylaxis, which occurs in response to allergens entering the body: pollen, animal dander, house dust, etc. Diseases based on local anaphylaxis include urticaria, angioedema, allergic rhinitis (hay fever), and some forms of asthma. There is a familial predisposition to this type of allergy.

Type II hypersensitivity reactions. In type II hypersensitivity, antibodies appear in the body directed against the components of its own tissues, which act as antigens. Antigenic determinants can be associated with plasmolemma or are an exogenous antigen adsorbed to the cell surface. In any case, a hypersensitivity reaction occurs as a result of the binding of antibodies to normal or damaged cell structures. There are three antibody-dependent mechanisms for the development of this type of reaction.

1. Complement-dependent reactions. There are two mechanisms by which antibody and complement can induce type II hypersensitivity: direct lysis and opsonization. In the first case, an antibody (IgM or IgG) reacts with an antigen on the cell surface to activate the complement system. It activates the membrane-attacking complex, which disrupts the integrity of the membrane. In the second case, the cells are phagocytosed after fixing the antibody or complement component C3b to the cell surface (opsonization). Clinically, such reactions occur when: blood transfusion of an incompatible donor and reaction with host antibodies; erythroblastosis of the fetus and antigenic differences between the mother and the fetus, when antibodies (IgG) of the mother cross the placenta and cause destruction of the fetal red blood cells; autoimmune hemolytic anemia, agranulocytosis and thrombocytopenia, when antibodies are formed against their own blood cells, which are then destroyed; some drug reactions, when the antibodies formed react with drugs and form complexes with the red blood cell antigen.

2. Antibody-dependent cellular cytotoxicity is not accompanied by complement fixation, but it causes leukocyte cooperation. Target cells coated with low concentrations of IgG antibodies are destroyed by non-sensitized cells that possess Fc receptors. These cells bind target cells using receptors for the IgG Fc fragment, and cell lysis occurs without phagocytosis. This type of cytotoxicity involves monocytes, neutrophils, eosinophils, and NK. This type of cytotoxicity is also important in the reaction of graft rejection.

3. Antibody-mediated cell dysfunction.

In some cases, antibodies directed against receptors on the surface of cells interfere with their function without causing cell damage or inflammation. For example, in myasthenia gravis, antibodies react with acetylcholine receptors in the motor end plates of skeletal muscle, disrupting neuromuscular transmission and thus causing muscle weakness.

Type III hypersensitivity reactions (associated with immune complexes). The development of type III hypersensitivity reactions is caused by antigen-antibody complexes formed as a result of antigen-antibody binding in the bloodstream (circulating immune complexes) or outside the vessels (in situ immune complexes). Circulating immune complexes cause damage when they enter the wall of blood vessels or filter structures (glomerular filter in the kidney). There are two types of immunocomplex damage, which are formed when an exogenous antigen (foreign protein, bacteria, virus) enters the body and when antibodies are formed against its own antigens. Diseases caused by immune complexes can be generalized if immune complexes are formed in the blood and are deposited in many organs, or associated with individual organs such as the kidneys (glomerulonephritis), joints (arthritis) or small blood vessels of the skin (local Arthus reaction).

Systemic immunocomplex disease. One of its varieties is acute serum sickness resulting from repeated administration of large amounts of foreign blood serum used for passive immunization.

The pathogenesis of systemic immunocomplex disease consists of three phases: the formation of antigen-antibody complexes in the blood; deposition of immune complexes in various tissues; inflammatory response. The first phase begins with the entry of the antigen into the bloodstream and the formation of antibodies. In the second phase, these complexes are deposited in various tissues. The further course of the disease is determined by two factors: the size of immune complexes and the state of the system of mononuclear phagocytes (SMF). With a significant excess of antibodies, very large complexes are formed, which are quickly removed from the bloodstream by SMP cells and are relatively harmless. The most pathogenic complexes are small and medium-sized, which are formed with a slight excess of antibodies and remain in the bloodstream for a long time. Once the immune complexes are deposited in the tissues, they initiate an acute inflammatory response. During this phase (approximately 10 days after antigen administration), clinical manifestations of the disease are observed, such as fever, urticaria, arthralgia, swollen lymph nodes and proteinuria. Following the deposition of immune complexes, the complement system is activated with the formation of its biologically active components. Complement activation is accompanied by pro-inflammatory effects: release of C3b-opsonin, which promotes phagocytosis; the formation of chemotactic factors that cause migration of polymorphonuclear leukocytes and monocytes (C5); the release of anaphylotoxins (C3a and C5a), which increase vascular permeability and cause smooth muscle contraction; the formation of a complex (C5b-9), causing the destruction of cell membranes and cytolysis.

Phagocytosis of antigen-antibody complexes by leukocytes results in the release or formation of various additional pro-inflammatory substances, including prostaglandins, vasodilating proteins, and chemotactic substances.

Tissue damage is also mediated by free oxygen radicals produced by activated neutrophils. Immune complexes cause platelet aggregation and Hageman factor activation, which leads to an increase in the inflammatory process and the formation of microthrombi. As a result, vasculitis, glomerulonephritis, arthritis, etc. develop.

The morphological picture of immunocomplex damage is dominated by acute necrotizing vasculitis. For example, damage to the glomeruli of the kidney is accompanied by hypercellularity due to swelling and proliferation of endothelial and mesangial cells, as well as infiltration by neutrophils and monocytes.

Chronic serum sickness develops with repeated or prolonged contact (exposure) with the antigen. Constant antigenemia is necessary for the development of a chronic immune complex disease, since immune complexes most often settle in the vascular bed. For example, systemic lupus erythematosus is associated with long-term preservation (persistence) of autoantigens. Often, however, despite the presence of characteristic morphological changes and other signs indicating the development of an immune complex disease, the antigen remains unknown, for example, in rheumatoid arthritis, periarteritis nodosa, membranous nephropathy and some vasculitis. Local immunocomplex disease (Arthus reaction) is expressed in local tissue necrosis resulting from acute immunocomplex vasculitis. The Arthus reaction develops within a few hours and reaches a

peak 4-10 hours after injection, when a zone of visible edema with hemorrhages appears. Fibrinoid vascular necrosis is described in light-optical examination. Vascular rupture leads to the development of local hemorrhages, but thrombosis is more often observed, contributing to the development of local ischemic damage.

IV type of hypersensitivity (cell-mediated).

Type IV hypersensitivity is caused by specifically sensitized T-lymphocytes. It includes the classic delayed hypersensitivity reactions caused by CD4 + T-lymphocytes and direct cellular cytotoxicity mediated by CD8 + T-lymphocytes. This is the main type of immune response to various intracellular microbiological agents, especially mycobacterium tuberculosis, as well as to many viruses, fungi, protozoa and parasites. Two variants of type IV hypersensitivity reactions are described.

Delayed type hypersensitivity (HRT).

An example is the reaction to intradermally injected tuberculin - a component from the walls of mycobacterium tuberculosis. In a sensitized patient, after 8-12 hours, redness and induration occurs at the injection site, and the peak of the reaction occurs after 24-72 hours. In highly sensitized patients, necrosis develops in the injection area. HRT is characterized by the accumulation of mononuclear cells in the subcutaneous tissue and dermis, mainly around small veins and venules with the formation of characteristic perivascular cuffs. The release of plasma proteins outside the vascular bed increases the edema of the dermis and is accompanied by the deposition of fibrin in the interstitium. CD4 + T-lymphocytes predominate in the damaged areas.

With persistence of the antigen, macrophages are transformed into epithelioid cells surrounded by a shaft of lymphocytes - a granuloma is formed. This type of inflammation is characteristic of type IV hypersensitivity and is called granulomatous inflammation. IFN - is one of the most important mediators of HRT and a strong activator of macrophages. Activated macrophages capable of phagocytosis destroy microorganisms. At the same time, macrophages produce some polypeptide growth factors - platelet growth factor (TCGF) and transforming growth factor (TGF), which stimulate the proliferation of fibroblasts and enhance their synthesis of collagen. Thus, activated macrophages provide antigen elimination, and if activation continues, they contribute to the development of fibrosis. Cytokines TNF and TNF act on endothelial cells, causing an increase in prostacyclin secretion, which leads to an increase in blood flow as a result of vasodilation, and an increase in the expression of the adhesive molecule E-selectin (ELAM-1), which promotes the attachment of alien lymphocytes and monocytes. At the same time, there is an increase in the secretion of low molecular weight chemotactic factors, for example, IL-8. All these changes in the endothelium contribute to the release of lymphocytes and monocytes outside the vascular bed into the zone of HRT development.

In T-lymphocyte-mediated cytotoxicity, sensitized CD8 + T-lymphocytes destroy target cells that are carriers of the antigen (cytotoxic lymphocytes - CTL). T-lymphocytes directed against histocompatibility antigens fixed on the cell surface play an important role in transplant rejection. They are also involved in protecting against viral infections. In cells infected with a virus, viral peptides bind to MHC class I molecules and are transported in the form of complexes to the cell surface. This complex is recognized by cytotoxic CD8 + T lymphocytes. Lysis of the infected cells is completed before the virus replicates, which leads to the destruction of the viruses. It is believed that many tumor antigens are present on the cell surface, and CTLs are involved in anti-tumor immunity.

When studying immunopathological processes, one cannot fail to mention the state of autoimmunization. Autoimmunization is a condition characterized by the appearance of a reaction of the immune system to normal antigens of its own tissues. Autoimmunization can be based on three different mechanisms: a violation of the physiological isolation of organs and tissues in relation to which there is no immunological tolerance, primary disorders in the immunocompetent system, which ceases to distinguish between "own" and "foreign" antigens and the appearance in the body of new "foreign" antigens ... Guided by the mechanism of development of autoimmunization, there are three groups of autoimmune diseases.

The first group of autoimmune diseases includes: Hashimoto's struma, encephalomyelitis, polyneuritis, multiple sclerosis, idiopathic Addison's disease, aspermatogenia, sympathetic ophthalmia. These are true autoimmune diseases, the occurrence of which is provoked by an infection, especially viral, chronic inflammation, etc. Autoimmunization develops in connection with damage to the physiological barriers of immunologically isolated organs, which allows the immune system to respond to their unchanged antigens by producing autoantibodies and sensitized lymphocytes. At the same time, morphological changes develop in the organs, characteristic mainly for delayed-type hypersensitivity. Organ tissue is infiltrated by lymphocytes, parenchymal elements die, and sclerosis develops in the final. The second group of autoimmune diseases includes: rheumatism, systemic lupus erythematosus, rheumatoid arthritis, systemic scleroderma, dermatomyositis, secondary hemolytic anemia, and thrombocytopenic purpura. Leading in these diseases is a violation of the control of the immunological hemostasis of the lymphoid system, which can occur in connection with genetic factors, viral or bacterial infection, radiation. In organs and tissues with these diseases, morphological changes develop, characteristic of the reaction of both delayed and especially immediate hypersensitivity. The third group of autoimmune diseases includes: certain forms of glomerulonephritis, hepatitis, chronic gastritis and enteritis, cirrhosis of the liver, burn disease, allergic anemias, agranulocytosis, drug disease. The formation of autoantigens in these diseases is associated with a change in the antigenic properties of tissues and organs - denaturation of tissue proteins in burns, radiation, trauma, chronic inflammation.

Immunodeficiency

All immunodeficiencies are divided into primary, which are almost always genetically determined, and secondary, associated with complications of infectious diseases, impaired absorption, aging, side effects of immunosuppression, radiation, chemotherapy, and other autoimmune diseases.

Primary immunodeficiencies - genetically determined diseases affect specific immunity (humoral and cellular) or nonspecific host defense mechanisms caused by complement and cells (phagocytes or natural killer cells). Although most immunodeficiencies are rare, some, such as lgA deficiency, are fairly common (especially in children). Usually, primary immunodeficiencies are manifested in children in the age interval between 6 months and 2 years of hypersensitivity to infectious diseases. The main manifestations of congenital immunodeficiencies: 1. Infectious diseases, mainly of organs and systems in contact with the external environment. 2. tumor diseases, predominantly of the lymphoid system. 3. Autoimmune disorders such as systemic lupus erythematosus, rheumatoid arthritis, hemolytic autoimmune anemia. One of the main criteria of congenital immunodeficiency is a decrease in thymus mass - a decrease in it by 5 times or an increase by 3-4 times.

Secondary immunodeficiencies are not associated with a genetically mediated block of any link of immunity; they develop under the influence of a wide variety of pathological processes that initially act on the unchanged immune system.

Acquired Immunodeficiency Syndrome (AIDS). The causative agent of AIDS is the human immunodeficiency virus (HIV), a retrovirus belonging to the lentivirus family. There are two main targets for HIV: the immune system and the central nervous system. The immunopathogenesis of AIDS is characterized by the development of profound immunosuppression, which is mainly associated with a pronounced decrease in the number of CD4 + T cells.

In addition to the death of infected CD4 + T cells, there are other indirect mechanisms for the development of the disease. First, the number of immature precursors of CD4 + T cells decreases, which is associated with their direct infection in the thymus, as well as infection of cells secreting cytokines necessary for the differentiation of CD4 + T cells. Secondly, infected and uninfected cells fuse to form syncytium (giant cells), autoimmune destruction of both

infected and uninfected CD4 + T cells occurs. Since many patients have circulating antibodies to gpl20, cells carrying gpl20 on the surface can be destroyed. A decrease in the number of CD4 + T cells causes a change in the CD4 / CD8 ratio in peripheral blood.

Infection of monocytes and macrophages is extremely important for the pathogenesis of AIDS. Like T-lymphocytes, most macrophages infected with HIV are formed in tissues, not in peripheral blood. Many macrophages express small amounts of CD4, and the virus can infect these cells. In addition, the virus can enter macrophages by phagocytosis or endocytosis of its particles coated with antibodies, or by means of Fc receptors. Infected macrophages bud off relatively small amounts of virus, but these cells contain many viral particles located exclusively in the intracellular vacuoles. Despite the fact that viral replication is possible in macrophages, unlike CD4 + T cells, they are completely resistant to the cytoplasmic action of the virus.

Infection with macrophages leads to the fact that monocytes and macrophages turn into a real "factory" for the production of viruses and a reservoir for their storage. In addition, macrophages are able to transport the virus throughout the body, especially to the nervous system. Unlike tissue macrophages, the number of monocytes in the bloodstream decreases. At the same time, antimicrobial activity, chemotaxis, secretion of IL-1 and TNF-a, the ability to present antigens to T-lymphoiitis decrease. Dendritic cells of lymph nodes are also an important reservoir of the virus.

Thus, CD4 + T cells, macrophages and dendritic cells, not blood cells, are the main reservoirs of the virus. In AIDS patients, the function of B-lymphocytes is significantly impaired. Thus, hypergammaglobulinemia and circulating immune complexes associated with polyclonal activation of B cells arise. Among the reasons for this phenomenon is the infection of B-lymphocytes with cytomegalovirus and Epstein-Barr virus, each of which is a polyclonal activator of B cells. Gpl20 itself can induce the growth and differentiation of B-lymphocytes, and HIV-infected macrophages produce increased amounts of IL-6, which promotes B-lymphocyte activation. Despite the presence of spontaneously activated B cells, the antibody response to the new antigen cannot be sustained in AIDS patients, therefore such patients are susceptible to disseminated infections caused by encapsulated bacteria.

Macrophages and microglia (cells related to monocytes and macrophages) are the main types of brain cells that are infected with HIV. Infected macrophages produce soluble factors (eg, IL-1 and gpl2O) that can be toxic to neurons or impair their function without direct toxicity.

The course of the acquired immunodeficiency syndrome consists of three phases, reflecting the dynamics of the interaction of the virus with the host: early acute phase; middle chronic phase; the final crisis phase. In the early phase, the initial response of the immunocompetent organism to the virus develops. This phase is characterized by a high level of virus formation, viremia and widespread seeding of lymphoid tissue. During this period, however, the infection is controlled by an anti-viral immune response. The chronic phase is a period of relative containment of the multiplication of the virus. The immune system is intact, but there is little viral replication (mainly in lymphoid tissue). This phase can last for several years. The final phase is characterized by disruption of the host's defense mechanisms and rampant viral replication. The content of CD4 + T cells decreases. After the "unstable" period, severe opportunistic infections, secondary tumors, and signs of neurological disease develop.

Amyloidosis

Amyloid is a protein that is deposited between cells in various tissues and organs. Protein recognition in the clinic depends solely on its detection in biopsies. In light-optical examination using traditional stains, amyloid looks like an amorphous, eosinophilic, hyaline-like intercellular substance, as a result of the progressive accumulation and pressure of which, cell atrophy develops. In order to distinguish amyloid from other deposits (collagen, fibrin), a number of histochemical methods are used, for example, staining with Congo red. In a polarizing microscope, the amyloid is greenish in color, giving birefringence.

Despite the fact that all deposits have the same appearance and tinctorial properties, amyloid is chemically heterogeneous. There are two main and several minor biochemical forms. They are

formed with the participation of various pathogenetic mechanisms. Apparently, amyloidosis is a group of diseases, the main symptom of which is the deposition of similar protein substances.
In an electron microscope, amyloid consists of unbranched fibrils about 7.5-10.0 nm long (F-component). This structure is the same for all types of amyloidosis. Crystallography and infrared spectroscopy revealed a characteristic folded structure of the shell. This structural feature explains the appearance of birefringence. In addition, the second component (P), which has a pentagonal structure, was revealed in smaller amounts.
Approximately 95% of amyloid consists of fibrillar protein, the remaining 5% is from the glycoprotein P-component.
Among 15 different biochemical variants of amyloid protein, two main ones are distinguished: amyloid from light chains (AL), which is produced by plasma cells (immunocytes), it contains immunoglobulin light chains; bound amyloid (AA) is a unique non-immunoglobulin protein synthesized in the liver.
Other proteins are also found in amyloid deposits. Trans-thyretin is a normal serum protein that binds and transports thyroxine and retinol. The mutant form of transthyretin (and its fragments) are detected in genetically determined diseases called familial amyloid polyneuropathy. Amyloid transthyretin (ATTR) differs from normal transthyretin by a single amino acid residue in the molecule.
P2-Amyloid is a peptide that makes up the nucleus of brain plaques in Alzheimer's disease. It is formed from the largest transmembrane glycoproteins, the so-called amyloid precursor protein (APP). There are also amyloid deposits formed from various precursors such as hormones (procalychitonin) and keratin.
The P-component differs from amyloid fibrils, but is closely associated with them in all forms of amyloidosis. It has structural homology to C-reactive protein. The serum P-component has an affinity for amyloid fibrils and is necessary for the formation of deposits in tissues.
The classification of amyloidosis is based on the chemical structure of the amyloid molecule (AL, AA, ATTR) and clinical syndromes. Amyloidosis can be systemic (generalized) with damage to several organ systems or local, when deposits are found in only one organ. Systemic (generalized) amyloidosis is primary, if it is associated with dyscrasia of immunocytes, or secondary, when it occurs as a complication of chronic inflammation or destructive processes in tissues. Congenital (familial) amyloidosis is a separate heterogeneous group.

Lesson plan.

Explore and describe:

1. . To study the reaction of type I hypersensitivity according to the microscopic picture. Describe the micropreparation "Bronchial biopsy in bronchial asthma" (staining with hematoxylin and eosin). Pay attention to the blood filling and permeability of the vessels of the mucous membrane, the basement membrane of the epithelium, the mucous glands, the composition and localization of the cellular infiltrate, the contents of the bronchus.

2. To study the reaction of hypersensitivity II mupa according to the macroscopic picture. Describe the macro-preparation "Large variegated kidney". Pay attention to the size, consistency, color of the kidney surface and on the cut.

3. To study the reaction of type III hypersensitivity in the microscopic picture. Describe the micropreparation "Glomerulonephritis" (staining with hematoxylin and eosin). Pay attention to the necrosis of the vascular loops of the glomeruli, the thickness of the basement membranes of the capillaries, blood clots in the lumen of some capillaries, changes in cell nuclei.

4. To study the reaction of hypersensitivity of the delayed type on the microscopic picture. Describe the micropreparation "Tuberculous productive tubercles in the intestine." Pay attention to the focus of caseous necrosis and the composition of the cellular infiltrate.

5. Examine Hashimoto's goiter in a macroscopic picture. Describe the Hashimoto's Goiter macro-preparation. Pay attention to the size, surface of the gland, consistency, type of tissue in the section.

6. Examine Hashimoto's goiter by a microscopic picture. Describe the micropreparation "Hashimoto's Goiter" (staining with hematoxylin and eosin). Pay attention to the localization and cellular composition of the infiltrate, the state of the parenchyma.

7. To study the micropreparation "Fibro-edematous polyp of the nose". Pay attention to dilated, full-blooded vessels, edema of connective tissue, inflammatory infiltration with a predominance of eosinophils.

8. To study the micropreparation "Kidney with periarteritis nodosa". Pay attention to the fibrinoid necrosis of the inner and middle membranes of the arterial wall, the cellular reaction in the adventitia and the surrounding connective tissue.

9. To study the spleen amyloidosis according to the macroscopic picture. Describe the spleen amyloidosis macro-preparation. Pay attention to organ size, consistency, color and sectional view.

10. To study the micropreparation "Amyloidosis of the spleen" on the microscopic picture. Describe the micropreparation "Spleen amyloidosis" (staining with hematoxylin and eosin, Congo red). Pay attention to the localization, structure and color of amyloid, the state of the cellular elements of the pulp. Mark the amyloid color when using Congo red.

11. To study the micropreparation "Pneumocystis pneumonia in HIV". To study Pneumocystis pneumonia in HIV, to describe the micropreparation "Pneumocystis pneumonia in HIV", to pay attention to mixed cell infiltration.

12. To study the micropreparation "Kidney amyloidosis". To study renal amyloidosis by microscopic picture. Describe the micropreparation "Kidney amyloidosis" (staining with hematoxylin and eosin, Congo red). Pay attention to the localization, structure and color of the amyloid. Mark the amyloid color when dyeing Congo red.

Questions

1. Macrophages secrete substances
a) IL-1
b) TNF
c) plasminogen activator
d) kappa chains of immunoglobulins
e) derivatives of arachidonic acid

2. Characteristics of cd4 + lymphocytes
a) secrete IgE
b) secrete IL-2
c) have cytotoxicity
d) perform suppressive functions

3. The reaction of hypersensitivity associated with the formation of IgE, refers to
a) type I (anaphylactic)
b) type II (cytotoxic)
c) type III (immunocomplex)
d) IV type (delayed type hypersensitivity)

4. The mechanism of development of autoimmune hemolytic anemia
a) immunocomplex
b) cell-associated cytotoxicity
c) antibody-associated cytotoxicity
d) immediate hypersensitivity
e) delayed-type hypersensitivity

5. Select the mechanisms underlying allergic reactions:
a) anaphylaxis
b) cytotoxic reactions
c) the reaction of immune complexes
d) blast transformation reaction
e) activation of T-killers
f) activation of T-suppressors

6. Immunopathological processes include:
a) disorders of immunogenesis
b) allergic reactions
c) reactions during transplantation
d) infectious immunity
e) autoimmune reactions
f) immunodeficiencies

7. The accidental transformation of the thymus is characterized by:
a) loss of lymphocytes
b) an increase in lymphocytes
c) collapse of the stroma
d) overgrowth of the stroma
e) fatty tissue degeneration
f) fatty tissue atrophy

8. Immunocompetent cells include:
a) lymphocytes
b) macrophages
c) mast cells
d) plasma cells
e) neutrophils
f) eosinophils

9. Allergens emit:
a) household
b) production
c) idiopathic
d) infectious

e) haptens
f) endogenous

10. Allergies of immediate type include:
a) Quincke's edema
b) bronchial asthma
c) allergic rhinitis
d) tuberculin reaction
e) atopic dermatitis
f) graft rejection

11. The most typical penetration of the allergen:
a) through the skin
b) respiratory
c) with food
d) with water
e) during sexual intercourse
f) parenterally

12. Stages of an allergic reaction:
a) sensitization
b) immunological
c) pathochemical
d) pathomorphological
e) pathophysiological
f) restorative

13. Examples of hapten allergy:
a) hay fever
b) reaction to penicillin
c) reaction to tuberculin
d) reaction to sulfonamides
e) transfusion of incompatible blood
f) photosensitivity

14. Mediators of reagin allergy:
a) histamine
b) bradykinin
c) adrenaline
d) serotonin
e) heparin
f) leukotrienes

15. Mediators of cytotoxic allergy:
a) lysozyme
b) interferon
c) kallikrein
d) complement
e) prostaglandins
f) superoxide anion radical

16. Mediators of immune complex type allergy:

a) leukotrienes
b) interleukins
c) complement
d) histamine
e) superoxide anion radical
f) interferon

17. Mediators of delayed allergy:
a) prostaglandins
b) leukotrienes
c) interleukins
d) histamine
e) superoxide anion radical
f) heparin

18. Delayed allergies include:
a) anaphylactic shock
b) serum sickness
c) multiple sclerosis
d) urticaria
e) systemic lupus erythematosus
f) HIV infection

19. A reaction like urticaria in response to exposure to sunlight is more correctly called:
a) incomplete allergy
b) photoallergy
c) pseudo-allergy
d) photosensitization
e) idiosyncrasy
f) allergoid reaction

20. On the surface of the mucous membranes, immunoglobulins of the class predominate.
a) A
b) D
c) E
d) G
e) M

Situational tasks

Problem No. 1 Sasha S., 6 years old, fell ill with diphtheria. Of the three children in contact with him in the apartment, two fell ill after 10 days. The third child did not fall ill; it is known from the anamnesis that he had already suffered diphtheria at the age of 3 years.
Questions: 1. Why didn't the child get sick? 2.What is immunity? 3. What types of immunity exist and which one does the child have?
Problem number 2 Patient I., 48 years old, was admitted to a surgical clinic about the carbuncle of the thigh. In the upper part of the right thigh on the inner surface there is a sharply painful dense infiltration of crimson-red color with a focus of necrosis in the center. The tissues around the infiltrate are swollen and tense. The lymph nodes in the right groin area are sharply enlarged and painful. Body temperature 38.5.
Questions: 1. How to explain the increase in lymph nodes? 2. What are “barriers” and what is their role in the mechanisms of immunity? 3. What are the main mechanisms of immunity?

Problem No. 3 Patient S., 32 years old, was admitted to the emergency room of the hospital in a serious condition: consciousness is lost, skin and mucous membranes are cyanotic, breathing is shallow. After several convulsive movements, the patient died with symptoms of asphyxia. It turned out that the patient suffered from hypersensitivity to aspirin, which caused her attacks of bronchial asthma. The doctor, not knowing about it, prescribed her powders containing this drug. After 10-15 minutes. after taking this powder, the patient felt unwell. Death came after 1 hour.
Questions: 1. What allergic reaction did the patient develop? 2. How is this reaction characterized?
Problem No. 4 Olya S., 9 years old, complains of general malaise, weakness, loss of appetite. In the evenings, the body temperature rises to 37.5. Physical examination did not reveal any changes in the internal organs. The child underwent a Pirquet reaction, which turned out to be sharply positive: after 24 hours, a papule (nodule) 15 mm in diameter with a zone of hyperemia was found at the site of tuberculin application. From the anamnesis it is known that 3 months ago Pirquet's reaction was negative.
Questions: 1. What is the mechanism for the development of a positive Pirquet reaction? 2. What is the difference between GZT and GNT?
Problem No. 5 Patient V., 50 years old, was treated with chloramphenicol for chronic bronchitis. The treatment proved to be effective; he was prescribed a maintenance dose of chloramphenicol, which he took for 6 months. By the end of this period, he developed severe anemia. The examination revealed the presence of antibodies in the patient's blood in relation to erythrocytes.
Questions: 1. What is the mechanism of anemia development in a patient? 2. What are autoaggressive diseases? 3. What diseases can develop as autoaggressive ones?

List of recommended literature:
Basic literature:

1. “Basic pathology” Vinay Kumar, Ramzi S. Cotran, Stanley L. Robbins, 1997.

Additional literature:

1. “Pathology. Quick Review and MCQs” Harsh Mohan, 2004.
2. “Textbook of Pathology ” Harsh Mohan, 2002.
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