LECTURE COMPLEX

Discipline: Pathological physiology
Discipline code: PF 3214
OP: 6B10107-"Dentistry"
Amount of study hours/credits: 90 hours/3 credits
Course and semester of study: III course, V semester
Volume of lectures: 6 hours

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The lecture complex was developed in accordance with the working curriculum of the discipline "Pathological physiology" (syllabus) OP 6B10103-"Dentistry" and discussed at a meeting of the department

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Head of the Department _____ Sadykova A.Sh.

Lecture No. 1

1. Topic: Subject, tasks and methods of pathological physiology. General nosology. General etiology and pathogenesis.

2. Purpose: to characterize the purpose, objectives and methods of pathophysiology; to define the basic concepts of general nosology; to explain the role of causes and conditions in the occurrence of diseases; to determine causal relationships in pathogenesis.

3. Lecture theses

Pathological physiology is the main fundamental medical and biological science that studies the general features of the onset, development and outcomes of the disease. Pathological physiology studies the causes and mechanisms of functional and biochemical disorders that form the basis of the disease, as well as adaptive mechanisms and restoration of functions disrupted during the disease.

The course of pathological physiology consists of 3 sections.

1. Nosology, or the general doctrine of the disease, provides answers to 2 questions that a doctor faces when analyzing the disease: why the disease arose and what is the mechanism of its development (etiology and pathogenesis).

2. Typical pathological processes – studies the processes underlying many diseases (inflammation, fever, tumors, hypoxia).

3. Private pathological physiology – considers violations of individual organs and systems.

The object of the study of pathophysiology is a disease, the main method of research is a pathophysiological experiment conducted on animals.

The experiment is used by many sciences (normal physiology, pharmacology, etc.). The significance of the experiment in pathological physiology consists in the experimental reproduction of the disease on animals, its study and the use of the data obtained in the clinic.

There are 4 stages of pathophysiological experiment:

- experiment planning;
- reproduction of the model of the pathological process in the experiment and its study;
- development of experimental methods of therapy;
- static processing of the received data and analysis of the study.

The following experimental methods are used to study pathological processes in living objects:

- method of irritation;
- shutdown method;
- activation method;
- the method of parabiosis;
- a method of cell cultivation.

The doctrine of disease, or general nosology, is one of the ancient problems of medicine. Health and disease are the 2 main forms of life. Health and illness can change each other many times during the life of a person and an animal.

Health is, first of all, the state of the body in which the correspondence of structure and function is noted, as well as the ability of regulatory systems to maintain homeostasis. Health is expressed in the fact that in response to the action of everyday stimuli, adequate reactions occur, which in strength, time and duration are characteristic of most people in this population. The conclusion about health is made on the basis of anthropometric, physiological and biochemical studies.

The disease is a qualitatively new condition that arises under the influence of external and internal pathogenic factors, manifested in the limitation of protective and adaptive capabilities to the effect of environmental factors and a decrease in the biological and social capabilities of the whole organism.

In a disease there are always 2 opposite processes, 2 beginnings. During fever, along with high body temperature, headache and other phenomena that reduce a person's ability to work, more active production of antibodies, more energetic phagocytosis, and other "measures against the disease" are observed. "Sex" and "measure against disease" are inextricably linked, if there is no unity between them, there will be no disease. The complete absence of a protective mechanism leads to death. The complete absence of "breakage" means health.

The concept of disease is close to the concepts of pathological reaction, pathological process, pathological condition.

A pathological reaction is an inadequate short–term response of the body to any stimulus. For example, a short-term increase in blood pressure under the influence of negative emotions.

A pathological process is a complex set of pathological reactions. Typical pathological processes include inflammation, fever, etc.

A pathological condition is a slowly developing pathological process or its outcome. For example, scarring of the esophagus developing after a burn, injury, etc.

Etiology is the study of the causes and conditions of the disease.

The cause of the disease is the main etiological factor that causes specific signs of the disease. Most often, the occurrence of the disease is associated with the influence of not one, but several factors. For example, the occurrence of croup pneumonia of the lungs can be influenced by negative emotions, improper nutrition, hypothermia, fatigue. However, without the penetration of pneumococcus into the body, these factors will not cause lung inflammation.

In the history of the development of etiology, different directions were known. According to the direction of monocasualism, any disease occurs due to a single cause, so the action of this cause will necessarily lead to illness. According to the direction called conditionalism, the disease is caused by many different conditions, but none of them can be the cause. Supporters of this trend believed that all the conditions of the disease are equally necessary for the occurrence of the disease, if there is not at least one, then the disease will not arise. Overestimating the significance of conditions, they completely excluded causal factors. At the same time, there was also a trend in etiology called constitutionalism. According to this trend, it is believed that the occurrence of the disease is determined only

by the constitutional features of oragnism. Since constitutional features are related to heredity, the occurrence of the disease is directly

subordinated not to environmental factors, but to the genotype.

There are the following causes of the disease:

1. Mechanical factors (wounds, compression).
2. Physical factors (sound, change in barometric pressure, influence of high or low temperature).
3. Chemical factors (alcohol, acids and alkalis).
4. Biological factors (bacteria, viruses, fungi).

5. Social factors (medical provision, sanitary and hygienic measures).

The interaction of the cause of the disease with the body always occurs under certain conditions. The difference between conditions and the cause is that there is one cause, but there are many conditions and that the latter are not necessary for the occurrence of the disease and do not give it specificity.

Pathogenesis is a branch of pathological physiology that studies the mechanisms of development and outcome of the disease. It is very closely related to the etiology of the disease. The main and most general pattern of pathogenesis is the pattern of self–development and self-maintenance.

The change of causes and effects leads to a vicious circle. Among the links of pathogenesis there are main and secondary. The main thing is the link necessary for everyone else.

4. Illustrative material:
- presentation of lecture material;
- posters on the topic of the lesson;
- tables, diagrams.

5. Literature: see appendix No. 1.

6. Control questions (feedback):

1. What does pathophysiology study?
2. What is the essence of a pathophysiological experiment?
3. What is a disease?
4. What is the difference between a pathological reaction, a pathological process and a pathological condition?
5. What is the etiology?
6. What causes the disease?
7. What is pathogenesis?
8. How does the vicious circle of pathogenesis arise?

Lecture No. 2

1. Subject: General pathology of the cell.

2. Objective: to explain the local and general mechanisms of cell damage.

3. Lecture theses

Cell damage is a typical pathological process. The causes of cell damage may be the following factors:

1) Hypoxia is an extremely important and common cause of cell damage. A decrease in blood circulation, which occurs with atherosclerosis, thrombosis, compression of the arteries, is the main cause of hypoxia.
2) Physical agents – mechanical injury, temperature effects, fluctuations in barometric pressure, ionizing and ultraviolet radiation, electric current.
3) Chemical agents and medicines.
4) Immunological reactions.
5) Genetic damage (for example, hereditary membranopathy, enzymopathy, etc.).
6) Nutritional imbalance.

Cell death is the end result of its damage. There are two main types of cell death – necrosis and apoptosis. To date, there is also a third type of cell death – final differentiation, which, according to most modern scientists, is one of the forms of apoptosis.

Necrosis is a pathological form of cell death due to its irreversible chemical or physical damage (high and low temperature, organic solvents, hypoxia, poisoning, hypotonic shock, ionizing radiation, etc.). Necrosis is a spectrum of morphological changes resulting from the destructive action of enzymes on the damaged cell. 2 competing processes develop: enzymatic digestion of the cell (colliquative, diluting necrosis) and denaturation of proteins (coagulation necrosis). It takes several hours for both of these processes to manifest, so in the case of sudden death, for example, with myocardial infarction, the corresponding morphological changes simply do not have time to develop. This type of cell death is not genetically controlled.

Necrosis may be preceded by periods of paranecrosis and necrobiosis.

Paranecrosis – noticeable but reversible changes in the cell: turbidity of the cytoplasm, vacuolization, the appearance of coarse precipitation, increased penetration of various dyes into the cell.

Necrobiosis is a state "between life and death", changes in the cell preceding its death. With necrobiosis, unlike necrosis, it is possible to return the cell to its original state after eliminating the cause that caused necrobiosis.

If necrosis is considered a pathological form of cell death resulting from excessive (sharp, strong) damaging effects on the cell, then apoptosis is opposed to it as a controlled process of cell self-destruction.

Apoptosis is a genetically controlled physiological form of cell death. The biological significance of apoptosis is to maintain the internal homeostasis of the body at the cellular, tissue and systemic levels. Apoptosis is responsible for programmed cell destruction at the stage of embryogenesis (autonomous apoptosis).

4. Illustrative material:

- presentation of lecture material;
- posters on the topic of the lesson;
- tables, diagrams.

5. Literature: see appendix No. 1.

6. Control questions (feedback)

1. What is cell damage?
2. What are the causes of cell damage?
3. What are the general mechanisms of cell damage?
4. What is apoptosis?

Lecture No. 3

1. Topic: Violations of water-electrolyte metabolism.

2. Objective: to explain the main mechanisms of disorders of water and electrolyte metabolism.

3. Lecture theses

Dehydration of the body can develop with the excretion of large volumes of water, insufficient electrolytes. At this time, intracellular and extracellular water decreases.

There are 2 types of dehydration: 1) when there is a shortage of water in the body; 2) when there is a shortage of mineral salts (electrolytes) in the body.

Dehydration from water loss occurs in various pathological conditions: with difficulty swallowing; in weakened and seriously ill; in premature or seriously ill children; hyperventilation of the lungs; with the release of a very large amount of urine.

Dehydration can develop with insufficient electrolytes, since, even if other useful properties of electrolytes are not taken into account, they have properties to bind and retain water. Such properties are possessed by sodium, potassium, and chlorine ions.

Hypoosmotic dehydration develops with greater excretion of salts from the body than water. Increased excretion of electrolytes from oragnism can occur through the gastrointestinal tract, kidneys and skin.

Isoosmotic dehydration develops with an equal loss of water and salts. This condition is possible with polyuria, dyspepsia and blood loss. At the same time, the extracellular fluid is mainly reduced.

Hyperosmotic dehydration develops with greater excretion of water from the body than salts. This condition can develop with a large discharge of saliva, increased deep breathing and diabetes insipidus.

With dehydration, after deterioration of blood circulation in the renal parenchyma, the ability of the kidneys to urinate decreases. This leads to azotemia, then to uremia.

Dehydration from lack of electrolytes cannot be restored only by the introduction of water. The composition of the water must necessarily contain electrolytes. Electrolytes are lost together with water through the digestive system: with vomiting, diarrhea, etc. In addition, the loss of electrolytes and water is observed in some types of nephritis, Adisson's disease. Water and electrolytes are released in large quantities and with abundant

sweating. Dehydration affects the functioning of many body systems. On the part of the cardiovascular system, there is a decrease in blood pressure, a decrease in the volume of circulating blood, and blood thickening.

Water retention in the body occurs with a large intake of water or a decrease in excretion processes. Hypoosmotic hyperhydration develops when a large amount of water is injected into the organism. Isoosmotic hyperhydration can be observed for a short time with excessive administration of

isotonic fluids for therapeutic purposes. Hyperosmotic hyperhydration is observed with the forced use of seawater, the introduction of a large number of hypertonic solutions. Since there is more osmotic pressure outside the cell, more liquid leaves the cell. Because of this, dehydration of the cell develops.

The retention and accumulation of fluid in tissues due to a violation of the water exchange between blood and tissues is called edema.

Pathological accumulation of fluid in the serous cavities of the body is called dropsy. The accumulation of fluid in the abdominal cavity is called ascites, in the pleural cavity – hydrothorax. There are cardiac, renal, hepatic edema.

With the development of edema, the tissues are mechanically compressed, and blood circulation in them is disrupted. A large amount of fluid in the tissues makes it difficult to exchange substances between the cell and the blood. On the other hand, edema has a protective and adaptive property. Edema lowers the concentration of toxic substances entering the body, their absorption, distribution through the body.

4. Illustrative material:

- presentation of lecture material;
- posters on the topic of the lesson;
- tables, diagrams.

5. Literature: see appendix No. 1.

6. Control questions (feedback):

1. What are the types of dehydration?
2. What is dehydration?
3. What is hyperhydration?
4. Why does edema develop?

Lecture No. 4

1. Topic: Hypoxia. Peripheral circulatory disorders.

2. Purpose: to explain the etiology and pathogenesis of hypoxic conditions, to give an idea of the importance of hypoxia in the pathogenesis of structural and functional changes in cells and tissues in pathology; to explain the main causes and mechanisms of fever development.

3. Lecture theses

Hypoxia is a typical pathological process that occurs as a result of insufficient biological oxidation and the resulting energy insecurity of vital processes.

Classification of hypoxic conditions

1. Exogenous:

A) hypobaric; B) normobaric.

2. Respiratory (respiratory).
3. Circulatory (cardiovascular).
4. Hemic (blood).
5. Tissue (primary tissue).
6. Mixed.

According to the criteria for the prevalence of hypoxic condition , there are: a) local; b) general hypoxia.

According to the rate of development and duration: a) lightning-fast; b) acute; c) subacute; d) chronic.

By severity: a) mild; b) moderate; c) severe; d) critical. Hypobaric hypoxia develops with a decrease in atmospheric pressure.

It is most often observed during high-altitude ascents. Hypoxemia is also the leading pathogenetic factor of its occurrence, but unlike normobaric hypoxia, hypocapnia serves as an additional negative factor. Normobaric hypoxia occurs when the oxygen content in the inhaled air drops at normal atmospheric pressure. A similar situation may occur during prolonged stay in unventilated

spaces of small volume, when working in wells, mines.

Respiratory hypoxia occurs as a result of insufficient gas exchange in the lungs due to alveolar hypoventilation, violations of ventilation-perfusion relations, excessive extracellular and intracellular shunting of venous blood or with difficulty in the diffusion of oxygen in the lungs. The pathogenetic basis of respiratory hypoxia, as well as exogenous, is also arterial hypoxemia, in most cases combined with hypercapnia.

Circulatory develops with circulatory disorders, leading to insufficient blood supply to organs and tissues. The main reason for the development of this type of hypoxia is circulatory disorders: general and local.

During hemic hypoxia, due to quantitative and qualitative changes in hemoglobin, the function of oxygen transport by blood is disrupted. Quantitative changes in hemoglobin are associated with a decrease in the number of red blood cells. And a decrease in the number of red blood cells can occur with anemia (anemia) and with acute or chronic blood loss.

Tissue hypoxia develops due to a violation of the ability of cells to absorb oxygen.

Mixed gtpoxia is a combination of 2 or more of its main types. Emergency adaptation of the body to hypoxia

Urgent compensatory reactions occur reflexively and manifest themselves in deepening and quickening of breathing, an increase in the minute volume of breathing, the inclusion of reserve alveoli.

Long-term adaptation of the body to hypoxia

Long-term compensatory reactions occur with chronic hypoxia. This is manifested in the respiratory system by an increase in the diffusion volumes of the lungs, in the cardiovascular and blood systems by myocardial hypertrophy, due to the activation of erythropoiesis processes in the bone marrow by an increase in the number of red blood cells and the concentration of hemoglobin.

Fever is a typical pathological process characterized by both damaging and protective-adaptive reactions of the body. At the same time, a change in the activity of the thermoregulation center under the influence of pyrogens leads to an increase in body temperature. With fever, the mechanisms of thermoregulation are not disrupted, but rise to a higher level.

For reasons of occurrence, fever is divided into infectious and non-infectious.

Infectious fever occurs when exposed to bacteria, viruses, protozoa. Non-infectious fever occurs under the influence of external and internal factors leading to tissue damage. These include: burn, wound, heart attack, blood transfusion, internal hemorrhage, allergy, tumor growth, cirrhosis, etc.

Pyrogenic substances are of great importance in the development of fever. They are divided into exogenous and endogenous (leukocyte). Exogenous pyrogens include substances formed as a result of the activity or destruction of microbes.

Fever occurs in 3 stages:

1. the stage of temperature increase (stadium incrementum);
2. the stage of standing temperature at a high level (stadium decrementum);
3. the stage of lowering the temperature.

There are the following types of fever:

1. subfebrile – temperature rise to 37.90 C;
2. moderate – temperature rise to 38-39.50 C;
3. high – temperature rise to 39.6-40.90C;
4. hyperpyretic – temperature rise to 410C and above.

4. Illustrative material:

- presentation of lecture material;
- posters on the topic of the lesson;
- tables, diagrams.

5. Literature: see appendix No. 1.

6. Control questions (feedback):

1. What is hypoxia?
2. What are the types of hypoxia?
3. What factors cause fever?
4. What are the stages of fever?

Lecture No. 5

1. Topic: Inflammation.

2. Objective: to explain the main causes and mechanisms of inflammation.

3. Lecture theses

Inflammation is a typical process based on the influence of a damaging (phlogogenic) factor. With inflammation in the damaged tissue or organ, cell structure disorders, changes in blood circulation, increased vascular permeability and tissue proliferation are observed. Phlogogenic factors are divided into 2 groups – exogenous and endogenous.

Exogenous include microorganisms (bacteria, viruses, fungi); animal organisms (protozoa, worms, insects); chemicals (acids, alkalis); mechanical effects (foreign body, pressure); thermal effects (cold, heat); radiation energy (X-rays, radioactive, ultraviolet rays).

Endogenous factors include: accumulation of salts in the joints, thrombosis, embolism. For example, an inflammatory process develops at the site of a heart attack associated with a violation of microcirculation.

The inflammatory process consists of 3 stages: stage 1 – alteration;

Stage 2 – exudation with emigration of leukocytes; stage 3 – proliferation.

Vascular changes occur in 4 phases: phase 1 – vascular spasm;

Phase 2 – arterial hyperemia; phase 3 – venous hyperemia;

Phase 4 – stasis.

Inflammatory mediators:

a) mediators of humoral origin (kinins, complement system);

b) mediators of cellular origin, ready or pre-existing (mast cell mediators, serotonin, heparin, lysosomal enzymes);

c) mediators of cellular origin, newly formed (eicosanoids, lymphokines, monokines, free radicals).

Alterations can be primary and secondary. Primary alteration occurs when a damaging agent is directly exposed. Secondary alterations are the body's response to primary alterations.

Exudation is the exudation of the protein–containing liquid part of the blood through the vascular wall into the inflamed tissue. The fluid that comes out of the vessels during inflammation into the tissue is called exudate. Depending on the qualitative composition, the following types of exudates are distinguished: serous, fibrinous, purulent, putrefactive, hemorrhagic, mixed.

According to the mechanism of development, the process of exudation is associated with the influence of inflammatory mediators. The leading factor of exudation is considered to be an increase in vascular permeability.

Emigration is the release of leukocytes outside the vessels. Polymorphonuclear leukocytes are the first to be found in the focus of inflammation. The main function of leukocytes in the focus of inflammation is the absorption of foreign bodies (phagocytosis).

Proliferation is the 3rd stage of inflammation. Leukocytes die after several hours of phagocytic function. Macrophages purify the focus of inflammation from microorganisms. Dead cells secrete substances that stimulate proliferation.

4. Illustrative material:

- presentation of lecture material;
- posters on the topic of the lesson;
- tables, diagrams.

5. Literature: see appendix No. 1.

6. Control questions (feedback):

1. What is inflammation?

2. What factors cause inflammation?
3. What are the stages of inflammation?
4. What vascular changes occur in the focus of inflammation?

Lecture No. 6

1. Topic: Immunopathological processes.

2. Purpose: to explain the main causes, mechanisms of development and manifestations of allergies; to explain the mechanisms of development of the main types of allergic reactions.

3. Lecture theses

Allergy is an altered immune response to foreign substances characterized by damage to one's own tissues.

Many substances have antigenic properties to cause allergic reactions.

They are called allergens. Classification of allergens

Allergens can be exogenous and endogenous. Exogenous allergens enter the body from the environment, endogenous allergens are formed in the body itself. Exoallergens are divided into two types: infectious and non-infectious. Infectious allergens include bacteria, viruses, fungi and helminths. Non-infectious allergens include household (household dust, cosmetics), epidermal (wool, fluff and animal hair), vegetable (pollen, fruits), food (fish, chocolate, nuts, eggs), medicinal (antibiotics, sulfonamides, chloramine). These allergens enter the body from the outside through the respiratory, digestive tract, skin.

Classification of allergic reactions

Regarding the classification of allergic reactions, there are several views. R. Cook (1930) divided all allergic reactions into 2 types: allergic reactions of the immediate type and allergic reactions of the delayed type. Allergic reactions of the immediate type are observed a few minutes after re-entry of the allergen into the body.

Delayed allergic reactions are observed 24-48 hours after the allergen re-enters the body.

In 1969, Jell and Coombs divided allergic reactions into 4 types:

- allergic reactions of the reagin or anaphylactic type – Type I (atopic bronchial asthma, pollinosis);
- allergic reactions of cytotoxic type – type II (hemolytic anemia, agranulocytosis);
- allergic reactions of immunocomplex type – type III (serum sickness);
- allergic reactions of cytotoxic type – type IV (contact dermatitis); The mechanism of development of allergic reactions consists of 3 stages:

I. Immune stage. Antibodies or sensitized T-lymphocytes to a certain allergen are formed in the body. This stage is called sensitization. Sensitization is a gradual increase in sensitivity after ingestion of an allergen.

II. Pathochemical stage. At this stage, allergy mediators are released as a result of the interaction of the allergen and a specific antibody or sensitized T-lymphocyte.

III. Pathophysiological stage. Under the influence of mediators, violations of specific functions of organs and systems occur: increased blood pressure, increased permeability of the vascular wall, edema, bronchospasm.

Allergic reactions of type I (reagin)

In the immune stage, T cells, under the influence of allergens and macrophages, produce interleukin-4, stimulate B cells. After that, they turn into plasma cells and produce IgE. IgE are attached to blood labrocytes or basophils. Upon repeated ingestion of the allergen, it binds to IgE. After that, intracellular granules are released (degranulation). In the 2nd stage of allergic reactions, allergens interact with antibodies. This will lead to the release of mediators. In allergic reactions of the immediate type, histamine, serotonin, and bradykinin are released. The 3rd stage of allergic reactions is considered to be a set of functional, biochemical and structural changes. At this stage, disorders of the cardiovascular, respiratory, digestive, endocrine and nervous systems may develop. These include microcirculation disorders (capillary dilation, increased permeability, changes in rheological properties of blood), bronchospasm, an increase in glucocorticoids, changes

in the processes of excitation and inhibition at different levels of the nervous system.

Type II allergic reactions (cytotoxic)

At the stage of immune reactions, autoallergen is recognized with the participation of macrophages, T- and B-lymphocytes, B-lymphocytes, turning into plasma cells, produce IgG1 and IdM. These antibodies attach to cells with autoallergens. Then the stage of pathochemical changes develops, allergy mediators are formed. These mediators include complement components, lysosomal enzymes,

and oxygen free radicals. At the stage of pathophysiological disorders, the destruction of cells with allergens is observed. According to the II cytotoxic type

allergic reactions develop hemolytic anemia, thrombocytopenia, autoimmune thyroiditis, myocarditis, hepatitis, etc. autoimmune diseases.

Allergic reactions of type III (immunocomplex)

Allergens are medicines, therapeutic serums, food products, mushrooms, etc. Plasma cells form IgG1, IgG4 instead of IdM. These antibodies bind to allergens in biological fluids and form allergen-antibody immune complexes. If this complex is with a small excess of antigen, then it sticks to the capillary wall. Due to the formation of the allergen-antibody complex, a certain number of allergy mediators (complement, lysosomal enzymes, oxygen free radicals, histamine, serotonin) are released. Complement components increase the permeability of the walls of blood vessels. Immune complexes, attaching to platelets, destroy them.

Type IV allergic reactions (cell-mediated)

Allergens are proteins, glycoproteins and chemicals that bind to proteins. These reactions develop on proteins of low molecular weight and weak ability to antibody formation. The cellular immune response is carried out with the help of T cells. Allergens trapped from the outside or formed in the body bind to macrophages and develop. After the allergen re-enters the body, T-cells are very important in the formation of a rapid immune response. They bind to allergens. Sensitized T-cells attached to cells with allergens on the surface form cytokine mediators. From their exposure, inflammation develops in a few hours at the location of the allergen.

4. Illustrative material:

- presentation of lecture material;
- posters on the topic of the lesson;
- tables, diagrams.

5. Literature: see appendix No. 1.

6. Control questions (feedback)

1. What is an allergy?
2. What factors cause allergies?
3. How can allergic reactions be classified?
4. What is the general pathogenesis of allergic reactions?

Literature:

Приложение № 1

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