LECTURE COMPLEX

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Name and code of the EP: 6B10117 - "Dentistry"
Number of study hours/credits: 90hours/3 credits
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Lecture length: 6 hours

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ОҢТҮСТІК-ҚАЗАҚСТАН MEDISINA AKADEMIASY «Оңтүстік Қазақстан медицина академиясы» АҚ
SOUTH KAZAKHSTAN MEDICAL ACADEMY АО «Южно-Казахстанская медицинская академия»
Department of Pathology and Forensic Medicine
Lecture complex

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

Protocol No. 13 from " 26 " 06 2025 y.

Head of the Department _____ Sadykova A.Sh.

Lecture No 1

1. Topic: Dystrophies

2. Objective: To characterize the goals, objectives, objects, methods and levels of pathological anatomy research; to give the concept of autopsy, biopsy, experiment as methods of studying pathological anatomy, to analyze the issues of their clinical significance in practical medicine.

3. Lecture abstracts

Subject, goals, objectives, objects, methods and levels of research of pathological anatomy. Cell and tissue death: pathology of the cell nucleus, mitosis and cytoplasm *Pathological anatomy is* a medical and biological science that studies the structural foundations of pathological processes, i.e. the material foundations of diseases. Pathological anatomy is an integral part of pathology and a link between theoretical and practical medicine, studies various aspects of disease. with this, is a scientific and applied discipline.

The tasks that modern pathological anatomy solves put it in a special position among medical disciplines. On the one hand, it is the theory of medicine, which, by revealing the material substrate of the disease, serves directly to clinical medicine, on the other hand, it is clinical morphology, which, while actively participating in the fate of the patient, at the same time serves the theory of medicine. Thus, pathological anatomy has a clinical and anatomical direction, therefore it is necessary for the study of other clinical disciplines.

The course of pathological anatomy includes 2 parts: general pathological anatomy and particular pathological anatomy. General pathological anatomy studies pathological processes that are manifestations of various pathological conditions or diseases. For example, cell pathology, dystrophies, necrosis, circulatory disorders, inflammation, regeneration, compensatory-adaptive and immunopathological processes, sclerosis and tumors. Particular pathological anatomy studies specific nosological forms or diseases.

The method of studying pathological anatomy is *the morphological method* - observation with the naked eye and with the help of optical devices.

Its varieties are: autopsy (autopsy, section, duction) - examination of the corpse at the macroscopic level in order to confirm and clarify the clinical diagnosis, identify a diagnostic error, establish the cause of death, the features of the course of the disease, identify the effectiveness of the use of medications, diagnostic manipulations, develop mortality and morbidity statistics; biopsy – intravital examination of organs and tissues for diagnostic purposes, examination of surgical material in order to confirm clinical diagnosis; experiment.

PARENCHIMATOSE DISTROPHIES develop in highly specialized cells – the parenchyma of organs. They are based on the violation of cellular mechanisms of trophic regulation. Enzymeopathy develops and cell function decreases. This determines the clinical manifestations of a particular syndrome, i.e. damage to a specific organ – liver, kidney, heart.

Parenchymal protein dystrophies – dysproteinoses are characterized by impaired metabolism of cytoplasmic proteins: bound (lipoprotein complexes of biomembranes) and free (enzymes). The physico-chemical and morphological properties of cell proteins are changing.

Parenchymal fatty dystrophies are characterized by a violation of the metabolism of lipids – the constituent components of cells. These include lipoproteins- complexes of fats and proteins, which are part of cell membranes, including organelles, as well as neutral fats – esters of glycerol and fatty acids, which are in the cytoplasm of cells.

Parenchymal carbohydrate dystrophies. Carbohydrate dystrophies associated with disorders of glycogen metabolism. Glycogen is a polysaccharide, its depots are in the liver and skeletal muscles. Disorders of

glycogen metabolism manifest themselves in the following ways: 1. A decrease or increase in the amount of glycogen in tissues. 2. The appearance of glycogen in areas where it is normally absent. The most pronounced changes in glycogen metabolism are observed in diabetes mellitus, where there is a defect in the b-cells of the islets of Langerhans and a disruption in insulin synthesis. As a result, glucose utilization is impaired (hyperglycemia, glucosuria). Normally, insulin binds to blood glucose molecules, and this binding is recognized by cell receptors.

STROMAL-VASCULAR DISTROPHIES. Mucoid swelling is a superficial and reversible disorganization of the connective tissue. Fibrinoid swelling (fibrinoid) is a deep and irreversible disorganization of the connective tissue with the formation of fibrinoid. Hyalinosis (hyaline dystrophy) is a deep disorder of protein metabolism with the formation and deposition of protein hyalin in the connective tissue. Amyloidosis is a stromal-vascular disproteinosis that is accompanied by a profound disruption of protein metabolism and the formation of an abnormal protein called amyloid, which is deposited in the stroma of organs and the walls of blood vessels.

MIXED DISTROPHIES. Hemosiderosis is an excessive formation of hemosiderin. In pathological conditions, hemosiderosis can be local or general. General hemosiderosis develops in pathological conditions associated with increased hemolysis. Impaired formation and excretion of bilirubin is accompanied by jaundice. In this case, the sclerae, skin, mucous membranes, and serous membranes of the body turn yellow. There are three types of jaundice, depending on the mechanism of its development: Hemolytic (suprahepatic) jaundice; Parenchymal (hepatic) jaundice; and Mechanical (subhepatic) jaundice. Hemolytic (suprahepatic) jaundice is associated with increased hemolysis. Melanin metabolism disorders can be widespread or localized, congenital or acquired.

MINERAL DISTROPHIES. There are three forms of calcification: 1. Metastatic (calcareous metastases) 2. Dystrophic metastases (petrificates) 3. Metabolic calcification (calcareous gout, interstitial calcification).Wilson-Konovalov disease (hepato-cerebral dystrophy) is an hereditary disease associated with insufficient production of alpha-globulin ceruloplasmin in the liver, which binds copper ions in the blood. As a result, copper is released from its weak bonds with plasma proteins and accumulates in various tissues, including the liver, brain, kidneys, pancreas, testicles, cornea, and others. Copper deposition in the cornea is described as a pathognomonic symptom of Kayser-Fleischer disease in the form of a greenish-brown ring around the cornea. Disorders of iron metabolism. Iron is contained in hemoglobin, and disorders of its metabolism are morphologically manifested by disorders of the metabolism of hemoglobinogenic pigments.

4 .Illustrative material: Electronic content.Topic№1

5. Literature: see Appendix No 1.(1.3.4.5.6.7)

6. Control questions (feedback)

What does pathological anatomy study?

1. The goals of pathological anatomy?

2. Methods, objects of research of pathological anatomy?

3. Pathology of the cell nucleus?

4. Cytoplasmic pathology?

5. Definition, classification of parenchymal, stromal-vascular, mixed dystrophies, mineral dystrophies.

6. The importance and consequences of the formation of stones, dental plaque, tartar.

Lecture No 2

1. Topic: Circulatory disorders

2. Objective: to explain the main causes, pathogenesis, pathological anatomy, outcomes, and clinical significance of arterial and venous plethora.

3. Lecture abstracts

: Arterial congestion (hyperemia) is an increase in blood supply to the organ and tissue due to increased arterial blood flow.

ν Arterial plethora can be: ν General, ν Local.

Venous plethora (venous stasis) is an increased blood supply to an organ or tissue due to a decrease or obstruction of blood outflow. Blood flow is not changed or decreased. ν Stagnation of venous blood leads to dilation of veins and capillaries, slowing down blood flow in them, and the development of hypoxia. ν Hypoxia is the main pathogenetic factor that determines changes in organs in venous plethora.

4 .Illustrative material: Electronic content.Topic№3

5. Literature: see Appendix No 1(1.3.4.5.6.7)

6.Control questions (feedback)

1. Classification of disorders of the fluid circulation system in the body.
2. Arterial hyperemia or congestion, definition, types of general and local arterial hyperemia.
3. Definition of venous hyperemia, types.
1. General venous plethora as a morphological manifestation of heart failure syndrome, definition of heart failure.
2. General acute venous plethora, causes, pathological anatomy (macro- and microscopic signs).
3. General chronic venous plethora, causes, pathological anatomy (macro- and microscopic signs).

Lecture No 3

1. Topic: Inflammation

2. Objective: To give a definition of inflammation, explain the pathoanatomical features, outcome, and clinical significance of various types of inflammation, and discuss their clinical significance in practical dentistry.

3. Lecture abstracts:

Inflammation is a complex, multifaceted, local vascular-tissue (mesenchymal) protective and adaptive response of the body to the action of a pathogenic irritant. Inflammation manifests itself in the form of changes in blood circulation, primarily in the microcirculatory system, at the site of tissue or organ damage, accompanied by increased vascular permeability, tissue dystrophy, and cell proliferation. The purpose of inflammation is to eliminate the pathogenic agent and restore the damaged tissue. Causes of inflammation 1) biological: - microorganisms: bacteria, viruses, pathogenic fungi, animal parasites; - antibodies, immune complexes. 2) physical and chemical: - exogenous: radioactive lesions, thermal factors, injuries, chemicals (acids, alkalis), poisons, etc.; - endogenous: uremic toxins, toxins of foreign bodies, necrotic tissues. Classification of inflammation: by the nature of the course: - acute; - subacute; - chronic; according to the predominance of the inflammatory reaction: - exudative; - proliferative. Outcome of inflammation: 1) resolution; 2) restoration of tissue in a small area; 3) scarring.

Proliferative (productive) inflammation is characterized by the predominance of reproduction (proliferation) of cellular elements of the affected tissue. Alteration and exudation are poorly expressed, difficult to recognize, segmented granulocytes are single (neutrophilic leukocytes). The basis of proliferative inflammation is the reproduction of young cells of connective tissue, as well as the cambial cells of blood capillaries, during differentiation, forming new capillaries. In this case, a cellular infiltrate is formed, consisting of cells. There are two types of proliferative inflammation: non-specific and specific.

In case of non-specific proliferative inflammation, the cells are diffusely distributed, and there is no specific morphological pattern characteristic of the pathogen that caused the inflammation. In case of specific proliferative inflammation, the cellular composition of the infiltrate, the grouping of cells, and the cycle of the process are characteristic of the pathogen that caused the inflammation. Specific inflammation is mostly characterized by the formation of granulomas, which are nodules composed of granulation tissue. There are two types of proliferative inflammation: 1. Interstitial inflammation. 2. Granulomatous inflammation.3. Inflammation with the formation of polyps and genital warts. 4. Inflammation around animal parasites

4 .Illustrative material: Electronic content.Topic№4

5. Literature see Appendix No 1.(1.3.4.5.6.7)

6. Control questions (feedback)

1. Definition, phases of inflammation.
2. Exudative inflammation. Types, morphological manifestations. Outcome and significance.
3. Types of proliferative inflammation. Morphogenesis. Outcome and significance.
4. Morphological structure of tuberculous, syphilitic granuloma, granuloma in actinomycosis.
5. Clinical significance of inflammation in dentistry.

Lecture No 4

1.Topic: Necrosis

2.Objective: explain the main causes, pathogenesis, pathological anatomy, outcomes, and clinical significance of necrosis

3.Lecture abstracts

Necrosis (from the Ancient Greek νέκρωσις, nekrosis, meaning "death") is the damage to a cell that leads to its premature death in living tissue through autolysis. Necrosis is the result of external factors affecting a cell or tissue, such as infection or trauma, which can disrupt the cell's functioning.

There are six distinctive morphological patterns of necrosis:

1. **Coagulative necrosis** is characterized by the formation of a gelatinous (jelly-like) substance in dead tissues that retain their tissue architecture and can be observed using light microscopy. Coagulation occurs as a result of protein denaturation, which causes albumin to transform into a solid and opaque state. This pattern of necrosis is commonly observed in hypoxic (low oxygen) environments, such as in cases of heart attack. Coagulative necrosis primarily occurs in tissues such as the kidneys, heart, and adrenal glands. Severe ischemia most often causes necrosis of this form.

2. **Colliquative necrosis** (or liquefactive necrosis), in contrast to coagulative necrosis, is characterized by the decomposition of dead cells to form a viscous liquid mass. This is typical of bacterial and sometimes fungal infections due to their ability to stimulate an inflammatory response. The necrotic liquid mass is often creamy yellow due to the presence of dead white blood cells and is commonly known as pus. Hypoxic infarctions in the brain are a type of necrosis because the brain contains little connective tissue but a large amount of digestive enzymes and lipids, so the cells can be easily digested by their own enzymes.

3. **Gangrenous necrosis** can be considered a type of coagulative necrosis that resembles mummified tissue. This is characteristic of ischemia in the lower extremities and the gastrointestinal tract. If an infection is superimposed on dead tissue, it can lead to liquefactive necrosis (wet gangrene).

4. **Cicatricial necrosis** can be considered as a combination of coagulative and liquefactive necrosis, usually caused by mycobacteria (e.g., tuberculosis), fungi, and certain foreign substances. The necrotic tissue appears white and loose, resembling a lumpy cheese. The dead cells disintegrate but do not completely decompose, leaving behind granular particles.] Microscopic examination reveals amorphous granular remnants enclosed in a characteristic inflammatory sac. Some granulomas contain this pattern of necrosis.

5. **Fat necrosis** is the necrosis of exclusively fatty tissue,[9] resulting from the action of activated lipases on fatty tissues such as the pancreas. In the pancreas, this leads to acute pancreatitis, a condition in which pancreatic enzymes leak into the abdominal cavity and liquefy the membrane, breaking down triglyceride esters into fatty acids through the process of fat saponification. Calcium, magnesium, or sodium can bind to these lesions, forming a chalky white substance. Calcium deposits are microscopic and may be large enough to be visible on X-ray examination. To the naked eye, calcium deposits appear as granular white spots.

6**. Fibrinoid necrosis** is a specific form of necrosis, usually caused by immune-mediated vascular damage. It is marked by antigen-antibody complexes called immune complexes, which are deposited in the arterial walls along with fibrin.

4 .Illustrative material: Electronic content.Topic№3

5.Literature: see Appendix No 1(1.3.4.5.6.7)

6.Control questions (feedback)

1. Macroscopic signs of necrosis.
2. Microscopic signs of necrosis.
3. Clinical and morphological signs of necrosis.
4. Pathological manifestations of necrosis.
5. Pathological manifestations of necrosis.

Lecture No 5

1.Topic Cardiovascular pathology

2.Objective: Explain the main causes, pathogenesis, pathological anatomy, outcomes, and clinical significance of atherosclerosis and arterial hypertension.

3.Lecture abstracts Atherosclerosis is a chronic disease that occurs as a result of impaired fat and protein metabolism, characterized by damage to elastic and muscular-elastic arteries in the form of focal deposition of lipids and proteins in the intima and reactive growth of connective tissue.

The following factors are of the greatest importance in the development of atherosclerosis: metabolic (exogenous and endogenous); hormonal; hemodynamic; nervous; vascular; hereditary and ethnic. Metabolic disorders in atherosclerosis are based on dyslipoproteidemia with a predominance of VLDL (VLDL) and LDL (LDL), which leads to unregulated cellular metabolism of cholesterol (the receptor theory of atherosclerosis by Goldstein and Brown), the appearance of so-called foam cells in the intima of the arteries, which are associated with the formation of atherosclerotic plaques.

In macroscopic examination, the following types of atherosclerotic changes are distinguished, reflecting the dynamics of the process: fatty spots or stripes; fibrous plaques; complicated lesions, represented by fibrous plaques with ulceration, hemorrhages and deposits of thrombotic masses; calcification, or aterocalcinosis. Microscopic stages of morphogenesis of atherosclerosis: pre-lipid; lipoidosis; liposclerosis; atheromatosis; ulceration; aterocalcinosis.

Arterial hypertension (AH) is a persistent increase in blood pressure. It can be primary, idiopathic, or secondary (symptomatic hypertension). At any stage of hypertension and in any clinical or morphological form, a hypertensive crisis can develop, which is often observed in the malignant course of the disease.

Morphological signs of a crisis: 1) spasm; 2) crinkling and tearing of the basal membrane of the intima of small arteries; 3) loosening of the vascular wall due to plasma infiltration; 4) fibrinoid necrosis of the vascular wall; 5) thrombosis. 6) diapedetic hemorrhages, manifested by the extravascular location of blood cells. In the case of malignant hypertension, frequent crises lead to fibrinoid necrosis and thrombosis of the arterioles of various organs, resulting in multiple infarctions and hemorrhages.

4.Illustrative material: Electronic content.Topic№4

5.Literature: see Appendix No 1(1.3.4.5.6.7)

1.Control questions (feedback)

2. Macroscopic signs of atherosclerosis.
3. Microscopic signs of atherosclerosis.
4. Clinical and morphological signs of atherosclerosis.
5. Pathological and anatomical manifestations of arterial hypertension.
6. Pathological and anatomical manifestations of hypertensive crisis.

Lecture No 6

1.Topic Pathology of inflammatory diseases of the respiratory system

2.Objective: to explain the main causes, mechanisms of development, morphogenesis, pathological anatomy, outcome, and clinical significance of inflammatory diseases of the respiratory system.

3.Lecture abstracts Pathological anatomy of bronchitis. In acute bronchitis, the mucous membrane of the bronchi becomes full of blood and swollen, and small hemorrhages and ulcerations may occur. In most cases, there is a lot of mucus in the bronchial lumen. Various forms of exudative inflammation develop in the mucous membrane of the bronchi, with the accumulation of serous, mucous, purulent, or mixed exudate. Fibrinous or fibrinous-hemorrhagic inflammation may develop in the bronchi; bronchial wall destruction, sometimes with ulceration of its mucous membrane, is possible, in this case, destructive-ulcerative bronchitis is said. Complications of acute bronchitis are often associated with impaired bronchial drainage function, which contributes to the aspiration of infected mucus into the distal parts of the bronchial tree and the development of inflammation of the lung tissue (bronchopneumonia).

In panbronchitis and panbronchiolitis, inflammation can spread not only to the peribronchial tissue, but also to the interstitial tissue of the lung (peribronchial interstitial pneumonia). The outcome of acute bronchitis depends on the severity of the damage to the bronchial wall. Serous and mucous bronchial catarrh are easily reversible. Destruction of the bronchial wall (purulent catarrh, destructive bronchitis, and bronchiolitis) contributes to the development of pneumonia. Long-term exposure to a pathogenic factor can lead to the development of chronic bronchitis. Pneumonias are a group of inflammatory diseases that differ in etiology, pathogenesis, and clinical and morphological manifestations, and are characterized by the predominant involvement of the distal airways, especially the alveoli. According to the topographic-anatomical feature (localization), there are three main types of pneumonia:— parenchymal pneumonia; interstitial (interstitial) pneumonia; mixed. According to the extent of inflammation: focal: miliary pneumonia, or alveolitis; acinar; lobular, confluent lobular; segmental, polysegmental. lobular pneumonia; total pneumonia. According to the nature of the inflammatory process, pneumonia can be: serous (serous-desquamative, serous-hemorrhagic); purulent; fibrinous; hemorrhagic; mixed. Croupous pneumonia is an acute infectious-allergic disease in which one or more lobes of the lung are affected (lobular pneumonia), fibrinous exudate appears in the alveoli (fibrinous or croupous pneumonia), and fibrinous deposits appear on the pleura (pleuropneumonia). All of the above-mentioned names of the disease are synonyms and reflect one of the features of the disease. Croupous pneumonia is considered an independent disease. It mainly affects adults and rarely affects children. Complications. There are pulmonary and extrapulmonary complications of croupous pneumonia. Pulmonary complications develop due to impaired fibrinolytic function of neutrophils. When this function is insufficient, the fibrin masses in the alveoli undergo organization, i.e. they germinate into granulation tissue, which, as it matures, turns into mature fibrous connective tissue. This process of organization is called carnification (from Latin. sagpo — meat) The lung turns into a dense, fleshy, airless tissue. With excessive neutrophil activity, lung abscess and gangrene may develop. The attachment of pus to fibrinous pleurisy leads to empyema of the pleura. Interstitial pneumonia is characterized by the development of an inflammatory process in the interstitial tissue (stroma) of the lung. It can be either a morphological manifestation of certain diseases (for example, respiratory viral infections), or a complication of inflammatory processes in the lungs. Bronchiectasis is characterized by a stable expansion of a bronchus or bronchiolus. Bronchiectasis can be congenital or acquired.. The lungs show dilatation of the bronchi or bronchioles, with inflammatory infiltration, especially by polymorphonuclear leukocytes. The inflammation and fibrosis extend to the surrounding lung tissue. Bronchiectasis can be cylindrical, saccular, or fusiform; its shape does not have any prognostic or etiological significance.

4.Illustrative material: Electronic content.Topic№6

5.Literature: see Appendix No 1(1.3.4.5.6.7)

1.Control questions (feedback)

1. Classification of diseases of the respiratory system
2. Bronchitis, pneumonia, bronchiectasis, pathoanatomical characteristics.
3. Complications, outcome, causes of death in bronchitis, pneumonia, bronchiectasis. Clinical significance

"Appendix No1".

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