MINISTRY OF HEALTH PROTECTION OF UKRAINE
ODESSA NATIONAL MEDICAL UNIVERSITY

Faculty Medical number 1

Chair Histology, cytology, embryology and pathological morphology with a course in forensic medicine

I APPROVE
Viee-rector for scientific and pedagogical work
_____ Eduard BURYACHKIVSKY
"__" _____ 2024

METHODOLOGICAL DEVELOPMENT TO INDEPENDENT WORK OF STUDENTS ON PATHOMORPHOLOGY

Faculty, course Medical. III

Academic discipline "Pathomorphology"
(name of academic discipline)

Approved:
Meeting of the department of histology, cytology, embryology and pathological morphology with a course of forensic medicine
Odessa National Medical University

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Plan

1. Topic No. 1: "Elements of cell ultrastructural pathology. Cell-matrix interactions. Cellular and extracellular mechanisms of trophic regulation." Topic #2: "Diseases of the musculoskeletal system. Parathyroid osteodystrophy, osteoporosis, Paget's disease, fibrous dysplasia, osteomyelitis, joint diseases, muscular dystrophies, myasthenia. Osteo- and cartilage tumors"
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3. Topic #3: "Tumors from cambial embryonic tissues. Tumors of childhood, which develop according to the type of tumors of adults.
4. Topic #4: "Cardiomyopathies. Leffler's endocarditis. Idiopathic myocarditis, acquired heart defects. Systemic vasculitis."
5. Topic #5: "Alzheimer's disease. Multiple sclerosis. Amyotrophic lateral sclerosis. Postresuscitation encephalopathy. Diseases of the peripheral nervous system."
6. Topic #6: "Parathyroid osteodystrophy, osteomyelitis, fibrous dysplasia, osteopetrosis, Paget's disease, muscular dystrophies. Myasthenia.
7. Topic #7: "Pathology of pregnancy, postpartum period and placenta. Diseases of the mammary gland."
8. Topic #8: "Asphyxia of newborns. Birth injury."
9. Topic #9: "Tumors of respiratory organs."
10. Topic #10: "Tumors of the gastrointestinal tract."
11. Topic #11: "Diseases of the biliary system and pancreas."
12. Topic #12: "Peritonitis, adhesion disease."
13. Topic #13: "Liver tumors."
14. Topic #14: "Morphological features of epithelial tumors of individual organs." 15. Topic No. 15: "Pathomorphological changes in diseases related to nutrition.
16. Vitamins Radiation sickness. Occupational diseases.."
17. Topic #16: "Quarantine infections."
18. Topic #17: "Rickettsiosis. Prion infections.."
19. Topic #18: "Children's infections."
20. Topic #19: "Diseases caused by protozoa, helminths. Mycoses.." 21. Topic #20: "Preparation for the final control for the year."

Topic No. 1: "Elements of cell ultrastructural pathology. Cell-matrix interactions. Cellular and extracellular mechanisms of trophic regulation."

Goal:as a result of independent study of this topic, students should know
classification and essence of changes associated with ultrastructural pathology, as well as etiology, pathogenesis, pathological anatomy of these pathological conditions. Know the possible consequences and complications of diseases associated with ultrastructural pathology.

Basic concepts:
The student should know:

1. Classification and essence of changes associated with ultrastructural pathology.
2. Etiology, pathogenesis, pathological anatomy of these pathological conditions. 3. Outcomes, complications of diseases associated with ultrastructural pathology.

The student should know: The student should be able to:

1. Classify ultrastructural pathological processes.

2. To characterize the etiology, pathogenesis and morphological essence of core pathology.

Topic content:

Thanks to the works of Rudolph Virchow, a great German scientist, one of the founders of modern pathological anatomy, today there are no diseases whose knowledge is not based on the morphological study of cells - the structural units of living organisms. However, it was possible to see the normal cell structure in all its modifications only in the second half of this century thanks to the application of the ultrastructural method of studying cells using transmission (transparent) and scanning electron microscopy, electronic histo- and immunohistochemistry, and electronic autoradiography.
When studying the course of general histology, you have obtained the necessary information about normal ultrastructures and their functional role in the vital activity of the cell.
Let me remind you that a cell is a highly organized, self-regulating structural and functional unit of a living organism, capable of active exchange with the environment.
The following ultrastructures are distinguished in the cell:
Core:(shell with nuclear pores, karyoplasm, nucleoli and perinuclear space; Cytoplasm: hyaloplasm with various organelles and inclusions:
1) organelles of membrane origin:
• cytoplasmic membrane (including desmosomes);
• mitochondria: (outer smooth membrane, inner folded membrane that forms outgrowths (crista), matrix);
• Golgi apparatus, or Golgi complex;
• endoplasmic reticulum, or endoplasmic reticulum:
• smooth;
• granular (rough);
• lysosomes: primary, secondary: cytolysosomes and phagolysosomes; residual bodies, or telolisosomes;
2) non-membranous organelles*origin:*
• free ribosomes and polysomes;

• centrosome (centriole);
• microtubules and microfilaments;
• specialized structures, or microfilaments (neurofibrils, myofibrils - smooth and striated, tonofibrils, microvilli, cilia and flagella);

*3) inclusion:*trophic, excretory, pigment.

Intracellular structures that appear unchanged under the light and electron microscope are not static during life. During the vital activity of cells, their constant renewal takes place.

Damage to individual ultrastructures and even the death of individual cells from which various human tissues and organs are built can be a manifestation of the "physiological norm." This constant, "programmed" process of cell death in the body, called apoptosis, is very important not only for the normal existence of the body, but also plays one of the key roles in many general pathological processes.

Action of these or other internal and/or external factors leads at the initial stage to damage of elementary cell structures and disruption of their functions, in the future the development of both the pathology of an individual cell and cell cooperations is possible. Cell pathology, or "cellular pathology" is the structural basis of all human pathology.

Numerous studies have shown that any pathological process, regardless of the degree of functional changes it manifests itself, begins at the level of ultrastructures, that is, at the subcellular level. There is not a single striking factor that would not lead to structural changes. A number of diseases were probably first diagnosed only at the ultrastructural level. It is important to note that the initial stages of the pathological process, which are detected only at the level of cell ultrastructures, are usually reversible or can be compensated.

*The main goal of learning is to be able to recognize*quantitative and qualitative morphological changes in cell ultrastructures caused by the influence of various pathogenic factors, and to interpret the functional significance of these changes.

For this you need to be able to:

- *identify*electron micrographs show the characteristic morphological features of cell organelles in pathological conditions;
- *determine*the nature and degree of morphological deviations of the studied organelle from standards accepted as "normal" morphological constants;
- *determine*reversibility and irreversibility of identified structural changes of organelles;
- *identify*stereotyped and specific changes in cell ultrastructures in response to the influence of a disease-causing factor;
- *interpret*morphological changes of ultrastructures and determine their functional significance in the development of general cell reactions in various general pathological processes, such as, for example, cell metabolism disorders, cell death (apoptosis and necrosis), cell hypertrophy and atrophy, cell dysplasia and metaplasia, tumor transformation, etc.

Let me remind you that the normal existence and functioning of the cell depends on:

1. State of the cell environment (homeostasis).
2. Timeliness and sufficiency of the entry of nutrients into the cell (oxygen, glucose, amino acids).
3. The level of metabolic products, especially CO2.

And, since in most cases the influence of any pathogenic (disease-causing) factor is accompanied by a change in homeostasis, the first reception of pathogenic information will be carried out by the cell through its cell membrane.

*Normal cytomembrane permeability is the main condition in cell homeostasis.*The cytomembrane is built both as a barrier and as a passage for all substances that enter or leave the cell. It supports the internal chemical composition of the cell by means of selective permeability and transport. The process of membrane transport involves the transfer of ions and other substrates against a concentration gradient. Transport can be active, then it requires ATP and "mobility" of transport

proteins in the membrane, or passive - with the help of various diffusion and exchange processes. Water and ions cross it by simple diffusion. Molecules such as glucose require a means of transport.

Therefore, we will begin the study of the ultrastructural pathology of the cell by studying the structural changes observed in the cell membrane.

Cytoplasmic membrane

The cell membrane is schematically depicted as a thin line. In a transmission electron microscope, it is represented by a three-layer structure consisting of two dense sheets, each 2 to 3 nm thick, which are separated by a less dense intermediate layer 4 to 5 nm thick. The total thickness of the membrane is from 7.5 to 10 nm. Its outer surface is represented by a thick layer of mucopolysaccharides (glycocalyx). The inner surface is in contact with elements of the cytoskeleton of the cell and is formed by labile proteins that ensure the integrity of microfilaments and microtubules. On the surface of the membrane of some cells there are microvilli, which are filled with actin molecules, as well as desmosomes (cellular junctions), which include microfilaments formed by the protein keratin, which is a histochemical marker of epithelial cells.

Dawson and Danielli in 1935 proposed a model of the cell membrane. The main "highlight" of the model is the nature of the arrangement of lipid molecules. The membrane consists of two rows of phospholipid molecules, located more or less perpendicular to the surface of the membrane so that their non-polar (hydrophobic) ends are in contact with each other, and the polar (hydrophilic) ends are turned to aqueous solutions on one or the other side of the membrane.

In the cytomembrane there are receptors for hormones, such as insulin or adrenaline, and other biologically active substances that affect the function and reactivity of cells; various proteins, mucopolysaccharide molecules and specific proteins (for example, antigenic determinants of histocompatibility) are localized, which determine its permeability and antigenic properties.

The cytomembrane plays a major role in intercellular communication both by forming specialized intercellular contacts and by transmitting signals.

Thus, the cytomembrane plays a critical role in cell growth and proliferation. It is assumed that pathological modifications in the cytomembrane are responsible for tumor transformation of cells.

Violation of the structure and function of the cell membrane

Causes of damage to the cytoplasmic membrane:

A. *Effect of physical and chemical factors*(high and low temperature, chemicals, etc.).

B. *Formation of free radicals*(very unstable particles with an odd number of electrons in the outer orbit), which contain activated oxygen, with the subsequent reaction between them and the lipids of the cell membrane (lipid peroxidation), as a result of which excessive energy is released.

*B. Activation of the complement system.*Complement is a system of plasma proteins (C1-C9) that exist in an inactive form and make up approximately 10% of blood globulins. When activated, its final products, probably C5b, C6, C7, C8 and C9 complexes, exhibit phospholipase activity, that is, they can enzymatically damage the cytomembrane. This phenomenon (fixation of complement and its activation) is an important component of the immune response, which destroys cells recognized as "foreign".

*D. Enzyme lysis.*For example, pancreatic lipases (secreted in excess in acute pancreatitis) and enzymes synthesized by Clostridium perfringens (one of the causative agents of gas gangrene) cause widespread necrosis of cytomembranes.

*D. Lysis by viruses*is carried out both by direct insertion of cytopathic viruses into the cell membrane, and indirectly - through an immune response to viral antigens that are located on the surface of infected cells.

Types of cytoplasmic membrane damage

Ultrastructural pathology of cell membranes can manifest itself in the form of damage to their shape and size and be accompanied by violations of membrane synthesis and exchange, changes in its permeability, violations of membrane transport, alteration of cellular connections, cell communication and their "recognition".

*Damage to the shape and size of the cytoplasmic membrane*morphologically manifests itself in the form of:

- deformation or atrophy of specialized structures;
- increase in the number (thickening of the cell membrane), length and area of membrane structures (pinocytotic and phagocytic vesicles);
- atrophy of the cell membrane with the appearance of cracks or tears;
- formation of special pathological structures (formation of myelin-like or pseudomyelin structures).

Examples of deformation or atrophy of specialized structures can be: atrophy of microvilli of enterocytes in diseases of the small intestine with the development of malabsorption syndrome or deformation of podocyte legs of the epithelium of the inner leaf of Bowman's capsule of the renal glomerulus in some nephropathies.

Most of the listed structural changes of the cell membrane are accompanied by a violation of its permeability. An important role in the implementation of membrane permeability belongs to the glycocalyx and the interaction of membrane proteins with the cytoskeleton, as well as hormones that interact with membrane receptors. Changes in permeability can be severe (irreversible) or superficial.

An increase in cell volume due to the influx of a large amount of water in connection with an anomaly of osmotic pressure is accompanied by the appearance of cracks and even tears in the membrane. If the gaps do not increase, then the gaps close and disappear. Thickening of the cell membrane may be associated with a decrease in the amount of calcium ions in the extracellular fluid. At the same time, the permeability of the membrane for sodium and potassium ions changes and liquid accumulates in the cell.

The action of an aggressive factor on the cell membrane causes corresponding (reactive) morphological changes in the form of an increase in the number, length and area of membrane structures. Cell capture of various foreign substances (liquid and thick) can be carried out using two mechanisms: pinocytosis and phagocytosis.

*Pinocytosis (ripeip - to drink) - invagination (indentation) of the outer cell membrane with the capture of a foreign liquid substance, successive closing of the membrane, its untying and the formation of a pinocytotic bubble.*This process is observed in most cells. Often, pinocytotic vesicles play the role of a vehicle for liquids that sometimes cross the entire cell in this way (for example, in the endothelium).

Phagocytosis (rhagein- eat) is the process of seizing a cell from the outside and pulling in some dense particle by evagination (protrusion) of the cell membrane and the formation of a phagocytic vesicle.

The fate of phago- and pinocytotic vesicles in most cases is the same: fusing in the cell cytoplasm with primary lysosomes, they form multivesicular bodies (in pinocytosis) or phagosomes (in phagocytosis). Those and others are called secondary lysosomes. In secondary lysosomes, the process of digestion of captured particles is carried out with the formation of residual bodies, which are later pushed out of the cell by exocytosis (echo - outside). Amputation of cytoplasmic appendages and release of damaged fragments of intracellular structures is called klasmatosis (slasteip - to damage).

The cellular response to anoxia, antigen-antibody conflict, or metabolic inhibitors is manifested by a peculiar change in the cell membrane in the form of the formation of myelin-like or pseudomyelin structures. They appear as a result of membrane lipid peroxidation and are formed from released phospholipids by twisting elongated cytoplasmic processes or microtubules. Pseudomyelin figures

should not be confused with specific myelin figures, which are associated with myelin. The latter are vacuolated and fragmented in cases of demyelination or damage to neurons.

Results of cytoplasmic membrane damage

Damage to the structural integrity of the cytoplasmic membrane leads to cell necrosis. Limited (local) damage can be restored, but with some loss of the membrane (for example, in erythrocytes, this process leads to the formation of microspherocytes).

Violation of the "barrier" function is accompanied by excess water entering the cell and the development of vacuolar or hydropic dystrophy.

*Alteration of intercellular contacts.*There are different types of contacts in the cell membrane that have been compared to electrical connections. They can be represented by complexes of strong (intermediate) or weak (desmosomes, finger-like contacts) intercellular contacts.

The pathology of intercellular contacts can be manifested in their preservation in those cases when they were obliged to disappear in the process of cell maturation: for example, in the epidermis with parakeratosis (delayed maturation and exfoliation of cells). In other cases, there is a breakdown of those cellular connections that should exist normally. At the same time, cells lose communication with each other. This condition can be caused by a decrease in the amount of calcium ions in the extracellular fluid or by the effect of phospholipases on the cell membrane. Divided cells have a thickened plasma membrane. Alteration of cell contacts is naturally observed in the process of carcinogenesis, is the basis of violation of contact inhibition of tumor cell proliferation, promotes tumor infiltration and metastasis.

*Changes in cell communication and their "recognition".*Communicability of cells and recognition of "own" and "foreign" is a necessary property of cellular cooperation. Cellular "communication" and "recognition" are primarily based on differences in the structure of the outer surfaces of plasma membranes. A special role in these processes is played by the glycocalyx of the membrane with surface antigens - markers of a certain type of cells. Surface antigens can change. Changes in "cellular communication" and "recognition" occur in various pathological processes (inflammation, regeneration, tumor growth). It is indicated that when antigens characteristic of this type of cells disappear, "embryonic" or abnormal (for example, embryonic) antigens may appear. Changes in glycoproteins (glycocalyx) of the membrane make it more accessible to antibodies. The cytoplasmic membrane takes part in immune processes. Antibodies can be fixed on its surface, and then an antigen-antibody conflict will appear. The presence of antigen-antibody complexes can be detected by fluorescence microscopy or in a scanning electron microscope.

Cytoplasm

Cytoplasm in a light microscope after staining with hematoxylin and eosin is acidophilic, looks optically homogeneous or finely granular. In an electron microscope, numerous structures (organelles) that are necessary for cell metabolism are determined. Parts of cells under pathological conditions contain formations that do not participate in metabolic processes, do not have a strictly defined structure and are not structurally homogeneous with the cytoplasm - these are inclusions (fat, glycogen, pigments, etc.).

Mitochondria

*Mitochondria are structures bounded by two membranes - an outer and an inner one, have the shape of a cylinder with a diameter of 0.5-1 nm and a length of 2-5 nm.*The number, shape, and size of mitochondria vary widely in different cells.

Mitochondria are indicators of the functional state of cells, most sensitive to aggression. It is known that one of the first signs of cell autolysis (death) is mitochondrial vacuolization. Although mitochondria are stable structures, they are constantly renewed in cells. The destruction (destruction)

of an excess number of mitochondria is carried out using the processes of autophagy by vacuoles, which play the role of secondary lysosomes.

Mitochondria are "powerhouses" that directly participate in the exchange through the Krebs cycle and the electron transport system of the respiratory chain. The energy they create is converted and accumulated inside ATP molecules in the form of energy-rich phosphate bonds (macroergic bonds). ATP is produced by phosphorylation of ADP; this reaction is associated with the oxidation of reduced substances in the respiratory chain of enzymes. Oxygen is needed for this.

Damage to mitochondria

*Causes of damage (alteration) of mitochondria,*associated with impaired ATP production:

*A. Hypoglycemia: Glucose is the main substrate for energy production in most tissues and the only source of energy in brain cells - neurons.*Therefore, a low level of glucose in the blood (hypoglycemia) leads to insufficient production of ATP, which is most noticeable in the brain. *B. Hypoxia:*A lack of oxygen in the cells (hypoxia) can occur with:

1) the presence of a mechanical barrier to breathing or lung diseases, which is accompanied by a violation of blood oxygenation;

2) ischemia, or disruption of arterial blood flow to tissues as a result of general circulation disorders or the occurrence of a local barrier to blood flow;

3) anemia (that is, with a decrease in the number of erythrocytes and/or the level of hemoglobin in the blood), which leads to a decrease in the transport of oxygen in the blood; 4) violations of the structure of hemoglobin (for example, poisoning with carbon monoxide (CO), in which carboxyhemoglobin is formed, which is unable to transfer oxygen).

*B. Inhibition of enzymes:*for example, potassium cyanide poisoning. Potassium cyanide inhibits cytochrome oxidase - the final enzyme in the respiratory chain - which leads to an acute deficiency of ATP in all cells of the organs and rapid death.

*D. Uncoupling of oxidative phosphorylation:*uncoupling of oxidation and phosphorylation occurs either through chemical reactions or through physical separation of enzymes from the mitochondrial membrane. Mitochondrial swelling, which is a common feature of most types of damage, is the cause of uncoupling of oxidative phosphorylation.

Types of mitochondrial damage

The following structural changes of mitochondria are distinguished:

- increase in number and size;
- formation of megamitochondria;
- change of form;
- *changes in the structure of mitochondrial cristae.*

*An increase in the number and size of mitochondria.*An increase in the number of mitochondria can be observed under an optical microscope. This is manifested by the appearance of oxyphilic tranules in the cytoplasm of cells. Such cells are known as oncocytes or, for example in the thyroid gland, as Hurtl cells. They have abundant cytoplasm, their nucleus is often pushed to the periphery. Oncocytes are often found in the thyroid, parathyroid, salivary, bronchial, and mammary glands. In secretory cells, oncocytic transformation indicates a change in protein synthesis. Cells, the cytoplasm of which is rich in mitochondria, are also found in other pathological conditions (hypertrophy, inflammation, tumors).

*Megamitochondria.*Mitochondria are capable of self-replication as plastids (an analogue of mitochondria) of plant cells. They can grow and divide, reach gigantic sizes, sometimes they are larger than the nucleus - these are megamitochondria. In a light microscope, they can be seen in the form of bright, round, very oxyphilic balls. Megamitochondria are found, for example, in hepatocytes with alcoholism and cirrhosis of the liver, in epithelial cells of kidney tubules with nephrotic syndrome, with riboflavin deficiency, with bromide intoxication, and with some muscle diseases.

However, it is also known that after the elimination of intoxication, giant mitochondria return to normal within a few hours.

*Swelling of mitochondria.*It is connected with the penetration of water into the mitochondrion. Swelling must be differentiated from a true increase in the volume of mitochondria, known as megamitochondria. Swelling of mitochondria is observed in a wide variety of conditions: starvation, hypoxia, intoxications, heartburn, muscle diseases, thyroxine administration, etc. Cloudy swelling, described under the optical microscope as granular cell dystrophy, is also accompanied by mitochondrial swelling.

*IP in vitro*two types of swelling were identified.

*The first type - with*by a small swelling amplitude, in which a change in energy activity entails a reversible alteration of protein structures. This type of swelling is accompanied by the passage of water through the expanded outer space formed by the outer membrane into the inner space formed by cristae and filled with a matrix. At the same time, the mitochondrial matrix is compressed and becomes very dense. After the contraction phase, the mitochondria can return to their normal state.

The second type- with a large swelling amplitude, resulting from an increase in the permeability of the inner membrane. The consequence of this is the smoothing and fragmentation of the crystals. Swelling with a large amplitude can initially be corrected by increasing the concentration of ATP and magnesium, but after damage to the outer membrane, it quickly becomes irreversible, that is, fatal). It is accompanied in vivo by the death of the granules of the mitochondrial matrix, which first become clear, then compact and form flakes in the inner chamber.

The final stage of death is characterized by the fact that both membranes - inner and outer - are torn.

*Changes in the structure of mitochondrial cristae*may refer to their size, shape and number:

- *deformation of crystals and a decrease in their number*(occurs with reduced activity of mitochondria);
- *increase in the number of mitochondrial cristae-* proof of growing functional needs of the cell.

Along with the change of cristae in pathological conditions, there is a change in the structure of dense granules of the mitochondrial matrix. These granules with a diameter of 20 to 50 nm accumulate divalent cations. In addition to calcium, magnesium, phosphorus and other inorganic substances, the matrix of dense granules is formed by proteins and lipids. their increase in volume is observed in cells oversaturated with calcium ions, which can lead to fatal cell damage. Hypertrophy (increase in volume) of these granules was detected in myocardial ischemia, in hepatocytes - in carbon tetrachloride intoxication, in muscle cells - in tetanus.

Endoplasmic reticulum

The endoplasmic reticulum (ER) in the cytoplasm forms numerous plexuses of slits and channels. It participates in the formation of the nuclear membrane and the Golgi apparatus. The function of the membranes that form the reticulum is diverse depending on its connection with ribosomes: the rough endoplasmic reticulum (RHE) is the site of protein synthesis, which forms the basis of cellular protein secretion, while the smooth endoplasmic reticulum (ER) plays a role in the synthesis carbohydrates, steroid metabolism and various toxic substances that need to be neutralized. It is also related to glycogen metabolism. The development of ER is an expression of synthetic activity that can be observed in the exocrine cells of the pancreas or plasma cells, but the accumulation of synthesis products in the ER may be due to the slowing down of their excretion. An example of this is Roussel bodies - round inclusions that are found in old plasma cells. Russel corpuscles are called tombstones for plasma cells. According to most scientists, the lipoproteins that are part of ER membranes are similar to those that are part of the outer cell membrane. Both membranes can connect, and then the ER opens to the outside of the cell, in particular, this happens in the phenomena of cellular secretion. On the other hand, it is possible to consider the moment when substances that enter the cell appear in the ER, while they are usually directed to lysosomes. Therefore, the ER protects the cell from the invasion of foreign substances. which are part of ER membranes, according to most

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Under conditions of pathology, two types of morphological changes can be observed - hyperplasia and atrophy of the endoplasmic reticulum.

ER hyperplasia(smooth or rough), that is, an increase in its number, can be accompanied by the formation of concentric structures, which are often visible under a light microscope as areas of eosinophilic cytoplasm. It has been biochemically proven that the number of enzymes responsible for detoxification increases in the structures formed by ER, thus, this phenomenon indicates the participation of smooth ER in detoxification processes. Such changes are non-specific and are observed under the influence of aflatoxin, carbon tetrachloride, DDT, dimethyl nitrosuede, phosphorus, progesterone, viral infections or tumors (hepatoma).

*ER atrophy,*that is, a decrease in its size, accompanied by a decrease in the protein-synthetic function of the cell (during starvation, liver diseases, aging).

Golgi apparatus

The Golgi apparatus (lamellar complex) is formed by flattened sacs (vacuoles) that contain secretory granules and anastomoses that are interconnected with the endoplasmic reticulum. In them, proteins that are intended for secretion are conjugated with carbohydrate groups. The size of the Golgi apparatus is related to the synthetic activity of the cell and is determined either by the level of external secretion, for example, in the liver or pancreas, or by the intensity of synthesis necessary for the vital activity of the cell itself, for example, in neurons.

Morphological manifestations of violations of the secretory function are expressed either in the form of hyperplasia of the lamellar complex, that is, an increase in the area of its membranes and the number of secretory granules, or in the form of atrophy of the lamellar complex, which is accompanied by a reduction (decrease) of vacuoles and loss of secretory granules.

*Hyperplasia of the Golgi apparatus*usually combined with hyperplasia of the endoplasmic reticulum. If the synthesis of certain substances precedes their secretion and excretion, then these substances accumulate in the Golgi apparatus and can damage it. . For example, accumulation of bile in hepatocytes during cholestasis.

*Atrophy of the Golgi apparatus*indicates a decrease in its functional activity. One of the reasons for such a decrease can be protein starvation, as well as a violation of the interaction of the lamellar complex with the endoplasmic reticulum.

Lysosomes

Lysosomes are found in cells in normal and pathological conditions. They participate in cell nutrition, destruction of cells or their aging parts, thereby facilitating the recovery of cells or contributing to their normal maturation. Lysosomes ensure the integrity of the biological balance disturbed by aggressive agents in numerous processes - inflammation, immune protection, impaired blood coagulation, etc. Lysosomes can be defined as small electron-dense structures that look like

polymorphic granules or vesicles surrounded by a lipoprotein membrane. This definition refers mainly to primary lysosomes, which are derivatives (derivatives) of the endoplasmic reticulum and the Golgi apparatus. They are able to destroy proteins, lipids, polysaccharides and nucleic acids with the help of more than 50 lysosomal enzymes of the hydrolase type.

Primary lysosomes combine with other vacuoles, releasing their contents into them, and thus form secondary lysosomes: pinolysosomes, phagolysosomes and autophago-lysosomes, or cytolysosomes. They are quite polymorphic and rich in acid phosphatase. If the digestion process is not completely carried out, residual (residual) bodies or telolisosomes are formed in them, which have the most diverse appearance. Some of them leave the cell by exocytosis, others by clasmatosis. Some telolisosomes undergo biochemical processing and leave the cell by diffusion through the cell membrane. Others can form brown complexes, such as lipofuscin, liposiderin, hemosiderin, and others, which remain inside or leave the cell. Granules of lipofuscin are considered by some authors as products of breakdown of membrane lipoproteins and are called "pigment of cell wear". They are also called tertiary lysosomes.

Thus, lysosomes belong to the intracellular lytic, or "digestive" system. In some cells, the digestive function can be dominant, as, for example, in polymorphonuclear leukocytes. Unlike most previously studied organelles, lysosomes have a catabolic, not anabolic, function. This function of lysosomes is performed using two mechanisms - endocytosis and autophagy.

Endocytosis. This process is very often observed in the proximal convoluted tubules of the kidneys. Protheses, especially with low molecular weight, after passing through the glomerular filter are reabsorbed and accumulated by lysosomes of the cells of the epithelium of the convoluted tubules of the kidneys. Apparently, it was this phenomenon that Virkhov described under the name "turbid swelling". The presence of granules with a positive reaction to acid phosphatase in the cells of the kidney tubules in many proteinurias indicates their lysosomal origin. Similar accumulation of proteins due to lysosomes can be observed in the liver (Kupffer cells, mononuclear phagocytes).

Autophagy. The ability of lysosomes to capture and destroy the cell's own structures explains how large molecules such as glycogen and ferritin can enter these organelles. The mechanism of autophagy begins with the formation of a system of smooth membranes around a region of the cytoplasm, which surround this region circularly and merge in the form of a vacuole into which primary lysosomes release their enzymes. This phenomenon, which is described under the name "focal cell necrosis", plays the role of an internal regulator of the cytoplasm. It can be assumed that it allows the cell to control the number of its mitochondria, the reproduction of which is carried out more or less autonomously.

Lysosomal diseases

Damage to lysosomal membranes

Destabilization (labilization) of lysosomal membranes in the form of cracks and breaks can be observed under the influence of various aggressive factors; ionizing radiation, anoxia, shock, carbon tetrachloride poisoning, exposure to silicon, vitamin deficiency and hypervitaminosis A, effects of bacterial endotoxins, etc. In these cases, hydrolases diffuse into the cell, which leads to its necrosis or progressive destruction by self-digestion.

However, there are a large number of stabilizers of the lysosomal membrane that protect it. from external influence. These include cholesterol, corticoids, vitamin E in small doses, antihistamine. They increase the resistance of cells in relation to the aggressor. Lysosomes also produce a large number of inactivators of aggressive agents, for example, during inflammation, immune reactions, intoxication. When this function is excessive and exceeds the force of aggression or blocks its nature, lysosomes no longer participate in homeostasis. They become abnormal and stretched.

Insufficiency of lysosomal enzymes

Lysosomes may lack some enzymes necessary for normal cell metabolism. Enzymopathy or dysmetabolic disease is congenital and is inherited in an autosomal recessive manner. Deficiency of enzymes is most often observed in glycogenoses (Pompe's disease, Gierke's disease), lipidoses (adipocyte lipase deficiency), hepatoses (Dabin-Johnson's disease). These conditions are sometimes called "hoarding diseases." In reality, it is not about the excessive formation of various substances, but about slowing down or stopping the destruction of their metabolites during normal synthesis. The term "lysosomal diseases" reflects a genetic deficiency of lysosomal enzymes, not actual damage to lysosomes. Only some states can definitely meet this term. This is a rare ShediakHigachi disease, in which large granules are found in damaged lysosomes of blood polynuclear cells. A similar condition is also observed in Aleutian minks and refers to a violation of the synthesis of various cellular inclusions, in particular, melanin grains, which is accompanied by their excessive accumulation in lysosomes and impaired function. The syndrome includes: albinism, neutropenia, adenopathy, hepatosplenomegaly, recurrent infections.

*The phenomenon of accumulation in lysosomes*underlies Wilson's disease, in which copper accumulates, and hemochromatosis, which is accompanied by the accumulation of ferritin.

Peroxisomes

*Peroxisomes (microbodies) are submicroscopic granules that contain many enzymes, such as D-amino acid oxidase, catalase, and uricase (hence the name -uricosomes).*These organelles are found among ER.

An increase in their number in hepatocytes is described with the use of drugs that reduce the level of lipemia, viral hepatitis, leptospirosis, in cardiomyocytes with long-term exposure to ethanol. A change in the structure of uricosomes was described in Menkes and Wilson's diseases.

*Reduction in the number of peroxisomes*and a decrease in the synthesis of their enzymes is observed in the liver during inflammation, as well as during tumor growth. Destruction of peroxisomes is noted in hyperlipidemia and hypercholesterolemia.

Peroxisomal diseases

To date, three syndromes are known, which are considered as hereditary peroxisomal diseases; acathalasemia, cerebrohepatorenal Zellweger syndrome and systemic carnitine deficiency.

*Acathalasemia is a disease based on a sharp decrease in catalase activity in the liver and other organs.*The main clinical syndrome of this disease is a gangrenous oral cavity covered with ulcers.

*Cerebrohepatorenal syndrome of Zellweger*characterized by:

- lack of peroxisomes in hepatocytes;
- a decrease in catalase activity of the liver to 20% or less;
- reduction of the endoplasmic reticulum;
- atrophy and a decrease in the number of mitochondria;
- an increase in the number of glycogen granules and lipid vacuoles in hepatocytes.

The leading clinical manifestation of peroxisome insufficiency is a violation of the synthesis of bile acids.

Systemic carnitine deficiency is accompanied by oxidation of fatty acids in skeletal muscles, liver, and blood plasma. Myopathy with periodic liver and brain dysfunction is observed in the clinic.

Cytosol (cytoplasmic matrix)

*Cytosol is a component of the cytoplasm that structurally does not belong to organelles and contains proteins that make up organelles, soluble enzymes that participate in the intermediate exchange of the cell.*Its viscosity varies, increasing with the increase in the number of filaments contained in it.

Variations in cytosol density:

*An increase in the density of the cytosol.*This is a non-specific response to various types of factors that damage the cell: anoxia or hypoxia, intoxication, the effect of a virus, cancer intoxication, ionizing radiation, the effect of high temperature, electric current, etc. Cytoplasm is acidophilic under a light microscope and denser under conventional electron microscopic examination as a result of reduced water content or protein denaturation. Alteration is accompanied in some cases by dilatation of the rough ER or compaction of the mitochondrial matrix and nucleoplasm. It is not always reversible. With coagulation necrosis, dense and amorphous fragments of hyaloplasm are visible in the electron microscope, and the cytoplasm is uniformly acidophilic in the light microscope.

*Decrease in cytosol density*may be associated with a decrease or cessation of protein synthesis, as well as with the penetration of water into the cytoplasm. With a local decrease in density, we speak of chromolysis.

PATHOLOGY OF NON-MEMBRANE ORGANELS

Ribosomes

Ribosomes, both free and bound to the membranes of the endoplasmic reticulum, are organoids necessary for recognition of the genetic code of the cell. The localization of ribosomes is related to the type of synthesizing proteins. Free ribosomes, which are found in basophilic erythroblasts and in neurons, ensure the synthesis of cellular proteins. In contrast, ribosomes associated with ER membranes are found in all secreting cells.

In conditions of pathology, ribosomes can build well-defined geometric figures. For example, under the influence of aflatoxin and in tumor cells of Burkitt's lymphoma, they have the appearance of a spiral. Similar changes are observed in cells during hypothermia, during oxygen starvation and protein deficiency in the body.

Pathology of microtubules and microfilaments

Microtubules (macrofilaments) occupy a special place in intercellular contacts. Most cells contain complexes of fibrillar structures that perform supporting, transport, contractile and motor functions. Specialized cells may also contain similar fibrils, but they differ biomechanically.

Some organelles are characterized by the combination of microtubules in groups, mostly of nine, for example, triplets in centrioles, doublets in cilia. Microtubules are very complex structures and contain many proteins, as well as ATP-ase, which is involved in the construction of the ciliated epithelium,

There are genetic abnormalities in the number or location of doublets. For example, the congenital immobile cilia syndrome (Kartagener's syndrome) is characterized by the fact that the cilia of the covering epithelium of the respiratory tract and the mucous membrane of the middle ear are immobile or immobile. Therefore, mucociliary transport is sharply weakened or absent, which leads to chronic inflammation of the respiratory tract and middle ear. In such patients, spermatozoa are also immobile, because their tail is equivalent to eyelashes.

The lack of connection between the peripheral and central doublets in the cilia is accompanied by their immobility. This can be observed in a wide variety of pathologies:

•with infectious bronchitis, which is accompanied by immobilization of cilia and lack of movement in the mucous membrane of the bronchus;

•in smokers, the inviolability of pathologically changed eyelashes, which contain many doublets, is very often noted;

•proliferation of centrioles with the formation of "eyelash cysts" is often observed in the genital tract of women with chronic inflammatory diseases (gonorrhea, chlamydia, ureaplasmosis, etc.).

Various substances, for example, colchicine, periwinkle alkaloids (vinblastine, vincristine), sulfhydryl reactive groups (cocadylate, diamide) can destroy microtubules. All these substances affect mitosis, change the functions of cells associated with microtubules.

*Microfilaments.*Actin filaments and myosin are found in almost all cells, regardless of whether they are muscle or non-muscle.

The pathology of microfilaments is diverse in etiology and pathogenesis.

A sharp increase in microfilaments is found in the epithelium of the bile ducts in primary biliary cirrhosis. It is known that the circulation of bile in the liver is regulated by the microfilament system. However, the question of primary or secondary accumulation of microfilaments in the epithelium of the biliary system has not yet been resolved. An increase in their number is described in cells during wound healing, as well as in tumors, especially in areas of invasion.

Intermediate filaments

Intermediate filaments are quite specialized depending on the cell type. However, intermediate filaments of different types may occur in cells of the same origin. Intermediate filaments include: cytokeratins - in epithelial cells, desmin - in muscle cells, vimentin in mesenchymal cells, neurofilaments - in cells of the central and peripheral nervous system, glial filaments - in glial cells.

*Pathology of intermediate filaments*associated with their accumulation in the cell and is observed in the formation of alcoholic hyaline (Mallory corpuscles), Alzheimer's disease and some forms of cardiomyopathies.

*1) Mallory's hyaline (alcohol hyaline).*At the beginning of the century, the famous American pathologist Mallory described hyaline inclusions of an irregular shape in liver cells in alcoholism, which bear his name. The question of their specificity was discussed for a long time. Hyaline Mallory can appear in many cases, but most often - with alcoholic cirrhosis. It was experimentally induced in animals with the help of griseofulvin (it is used in the clinic as an antifungal agent). Today, the accumulation of intermediate filaments is a morphological marker of chronic alcoholism.

*2) Alzheimer's disease,*or "presenile" dementia, is accompanied by the formation of fibrillary masses in the neurons of the cerebral cortex in the elderly. These fibrillar masses stain like Congo red amyloid substances and give birefringence in polarized light. But they are always found intracellularly, unlike amyloid, which is always located extracellularly. In the clinic, such patients develop dementia.

*3) Cardiomyopathies,*associated with a violation of desmin metabolism, are clinically manifested by progressive myocardial insufficiency and are characterized by massive deposits in cardiomyocytes of SHIK-negative material, which consists of intermediate filaments.

Cytoplasmic inclusions

*Secretory granules.*They are represented in cells in three varieties - these are exo-, endo- or neurosecretory granules. An important place in pathology is the secretion of abnormal (large in volume) secretory granules in Shediak-Higachi syndrome.

*Melanin and melanosomes.*Melanin is secreted by melanocytes of the skin, the specific function of which is the synthesis of melanin pigment and the formation of melanosomes. Both processes are independent, since melanocytes can contain melanosomes without melanin. Such melanocytes are found in albinos and with local depigmentation of the skin. During ultraviolet irradiation, melanosomes accumulate in the basal keratinocytes above the apical part of the nucleus, forming a kind of screen that protects the genetic apparatus of the cell from damaging radiation. In albinism, melanin synthesis is impossible due to insufficient polymerization of derivatives of aromatic acidic amines.

Two types of protein granules have been described.

An example of the first type can be protein granules, usually acidophilic in a light microscope, SHIK-positive (glycoproteins), the presence of which is caused by a deficiency of α1 antitrypsin. They can be found in the cells of the liver, kidneys, in neurons, in benign or malignant tumors, α1antitrypsin is produced in the liver and inhibits collagenase, and in most tissues - elastase. With a deficiency of α1antitrypsin, elastase damages the lung tissue, which leads to the development of emphysema.

An example of the second type is acidophilic protein granules, or Lewy bodies, which are observed in sympathetic neurons. They are elongated eosinophilic and SHIK-negative formations and are typical for idiopathic Parkinson's disease.

Tubulareticular inclusions are located in the endoplasmic reticulum and form cells from anastomosing irregular tubules. They were first discovered in the glomerular capillaries of the kidneys in an autoimmune disease -diseased red sheep. These inclusions are similar to some viral inclusions, such as myxoviruses. There is a hypothesis that tubuloreticular inclusions have a viral origin. They are found in the skin, in the kidneys, in lymphocytes with various injuries - scleroderma, idiopathic purpura, Goodpasture's syndrome, with malignant lymphomas.

They can be reproduced experimentally with the help of 5-bromo-deoxyuridine in lymphocyte culture. This drug is used in antiviral therapy and can unmask the latent virus.

THE NUCLEUS AND ITS ANOMALIES

Ultrastructural study of the nucleus allows one to objectively judge its morphofunctional state, and therefore, the cell as a whole. The nucleus plays a dominant role in cell division. The structure and size of the nucleus, which is in the interphase (intermitotic) state, depend on its ploidy (DNA content, number of chromosomes) and functional state. Most cells contain diploid nuclei. Tetraploid nuclei naturally have a larger diameter than diploid ones. Polyploidy is a multiple increase in the number of sets of chromosomes in cell nuclei. An increase in the number of polyploid cells is very common in all organs, for example, with old age, with reparative regeneration in the liver, with myocardial hypertrophy, with tumor growth. Aneuploidy - a condition in which there is an incomplete set of chromosomes in the nuclei, caused by chromosomal mutations,

Nuclei of a wide variety of specialized cells differ in size and shape, are similar in internal structure and mainly contain granules or clumps of chromatin. Chromatin is a complex substance contained in chromosomes. Basophilic staining of chromatin, caused mainly by the content of DNA in it. Chromatin, which is visible under a light microscope, is called condensed, and those parts of the chromosomal thread, which are visible only in an electron microscope, are called decondensed chromatin. Only decondensed chromatin DNA is involved in the transmission of information directing protein synthesis in a non-dividing cell. In other words, paradoxically, all that chromatin that can be seen in the nuclei of functioning cells under a light microscope does not perform any functions. Decondensed chromatin was called euchromatin, i.e. "good" chromatin, because it "works", and condensed chromatin is called heterochromatin (from the Greek. hetero -other), that is, chromatin of a different kind. The distribution of hetero- and euchromatin is a reflection of the functional state of the nucleus. Since the nucleus can go from a state of relative functional rest to a state of high functional activity and vice versa, the ratio of hetero- and euchromatin is not always constant. In addition, it is necessary to know that the interpretation of the character of chromatin distribution is not always unambiguous. For example, the margination of chromatin, that is, its predominant distribution under the nuclear envelope, can be interpreted both as a sign of nuclear activity and as a manifestation of its damage. Since the nucleus can go from a state of relative functional rest to a state of high functional activity and vice versa, the ratio of hetero- and euchromatin is not always constant. In addition, it is necessary to know that the interpretation of the character of chromatin distribution is not always unambiguous. For example, the margination of chromatin, that is, its predominant distribution under the nuclear envelope, can be interpreted both as a sign of nuclear activity and as a manifestation of its damage. Since the nucleus can go from a state of relative

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*Damage (alteration) of nuclei*can be reversible (sublethal alterations) and irreversible (lethal or fatal).

Sublethal alterations (reverse)

Condensation and margination of chromatin- accumulation of chromatin under the nuclear membrane in the form of a regular ribbon or small lumps. At the same time, the core is somewhat reduced in size. A chromatin conglomerate appears as a result of a decrease in the pH of cells with increased glycolysis. This process is a direct response to a variety of aggression and, probably, its first manifestation.

Change of the nuclear membrane. Vacuoles and pseudovacuoles

It is known that the nuclear membrane consists of two lipoprotein leaflets (plates), which have pores or round holes. The inner plate is smooth, the outer one is covered with ribosomes and is in contact with the endoplasmic reticulum,

Under conditions of pathology, true vacuoles and pseudovacuoles may appear in the nuclei.

Under the influence of a number of disease-causing factors, the nuclear membrane can become discontinuous, for example, during the dilation of perinuclear cisterns, or form local blisters by invagination of the inner leaf of the nuclear membrane, for example, in response to radiation. These are true intranuclear vacuoles.

Pseudovacuoles are formed by intranuclear invagination of the cytoplasm, they are surrounded by two membrane plates and contain various particles, organelles, in particular, ribosomes. They are characteristic of some types of cells, such as meningeal, Schwann, non-oral, and others, and are also found in tumor cells. Pseudovacuoles are verified in hepatocytes in various metabolic disorders.

Intranuclear inclusions

There are true inclusions and pseudo-inclusions.

*true*inclusions are represented by some viruses.

*Pseudoinclusions are glycogen particles (in the nuclei of hepatocytes in diabetes), as well as spherical, linear, fibrillar structures, the nature of which is not always known. In glial cells, fibrillar structures are detected after exposure to aluminum hydroxide Al(OH)3*The appearance of spherical bodies is associated with increased protein synthesis and accumulation of fibrillar structures. Complex structures appear in hepatocytes and epithelial cells of kidney tubules after exposure to heavy metals (Pb s Vi).

Lethal damage (irreversible)

There are three types of irreversible morphological changes of the nucleus: pyknosis, karyorrhexis and karyolysis.

*Pycnosis*An unfavorable result of the reverse condensation and marginalization of chromatin under the nuclear envelope can be its irreversible total condensation over the entire area of the nucleus. Then the nucleus becomes homogeneous, intensely basophilic stained and wrinkled - this is pyknosis. It is obvious that when the nucleus is pyknotic - it is dead, the chromatin threads condense as a result of the action of DNAse and lysosomal cathepsins and their destruction occurs more or less quickly.

Karyorrhexis(rexis - gap). This is the splitting of condensed chromatin, mainly into small, irregularly shaped fragments that can be inside the nuclear membrane, if it is preserved, or be located in the cytoplasm when it is destroyed.

Karyolysis {lysis- dissolution, melting) is a type of nuclear death in which the chromatin is more or less totally disintegrated and is not stained. One gets the impression that the nucleus is devoid of chromatin, which disappears due to the absorption of the surrounding cytoplasm.

It is believed that karyopIknosis, karyorhexis and karyolysis exist as successive stages of nuclear death. In fact, very often, but not always, karyorrhexis can be observed without pyknosis and karyolysis may not occur if the cell dies immediately after pyknosis or karyorrhexis, and chromatin fragments are eliminated to the outside.

Damage to mitosis Abnormalities of the mitotic rhythm

The mitotic rhythm, which is mainly adequate to the need to restore aging, desquamated, dead cells, can be changed under pathological conditions. A slowing of the rhythm is observed in aging or poorly vascularized tissues, an increase in the rhythm - in tissues with various types of inflammation, hormonal influence, in tumors, etc.

Anomalies of the development of mitoses

Some aggressive agents, acting on the S phase, slow down the synthesis and duplication of DNA. These include ionizing radiation, various antimetabolites (metatrexate, mercapto-6-purine, fluoro-5-uracil, procarbozine). they are used for antitumor chemotherapy.

Other aggressive agents act on the M phase and prevent the formation of an achromatic spindle. They change the viscosity of the plasma without splitting the strands of chromosomes. Such a cytophysiological change can cause the blockade of mitosis in metaphase, and then acute cell death, or mitonecrosis. Mitonecrosis is often observed, in particular, in tumor tissue, in the foci of some inflammation with necrosis. they can be caused by podophyllin, which is used in the treatment of malignant neoplasms.

Anomalies of the morphology of mitoses

Morphological abnormalities of mitoses are revealed during inflammation, action of ionizing radiation, chemical agents, and especially in malignant tumors. They are associated with severe metabolic changes in cells and can be labeled as "abortive mitoses." An example of such an anomaly is mitosis with an abnormal number and shape of chromosomes; three-, four- and multipolar mitoses.

Multinucleated cells

Cells that contain many nuclei are also found in a normal state, for example: osteoclasts, megakaryocytes, syncytiotrophoblasts. But they are often found in pathological conditions, for example: Langhans cells in tuberculosis, giant cells of foreign bodies, many tumor cells. The cytoplasm of such cells contains granules or vacuoles, the number of nuclei can vary from a few units to several hundreds, and the volume is reflected in the name: giant cells. their origin is variable: epithelial, mesenchymal, histocytic. The mechanism of formation of giant multinucleated cells is different. In some cases, their formation is due to the fusion of mononuclear cells, in others it is carried out due to the division of nuclei without division of the cytoplasm. It is also believed that their formation may be a consequence of some abnormalities of mitosis after irradiation or administration of cytostatics,

Changes in nucleoli

Under normal conditions, the size and structure of nucleoli in most cases are adequate for the intensity of cellular protein synthesis.

In conditions of pathology (for example, in tumor cells), high functional (secretory) activity of cells is often accompanied by an increase in the volume, and sometimes the number of nucleoli with their vacuolization. In these cases, they speak of nucleolar hydroopia (or hydropic nucleolus).

Disintegration (separation) of nucleolar structures into RNA granules and fibrils reflects a violation of the functional state of both the nucleolus and the cell and occurs under the action of various agents, such as actinomycin, aflatoxin, ionizing radiation. Accompanied by a change in RNA synthesis.

Thus, various structural changes can occur in the cell in response to the influence of many factors. In some cases, we are talking about general stereotypical submicroscopic reactions, or reversible (reversible), adaptive to changed living conditions, or irreversible dystrophic damage that leads to the death of the entire cell (apoptosis or necrosis). In other cases, specific submicroscopic changes may occur in the cell, which are manifested at the macroscopic level in the form of a certain nosological form. In such cases, they talk about chromosomal, lysosomal, peroxisomal and other diseases.

Almost all general pathological processes have their own ultrastructural characteristics that allow them to be differentiated. Therefore, the knowledge of ultrastructural pathology will allow a deeper understanding of the basics of general pathological processes and to learn the secrets of not yet studied human diseases.

1. Theoretical questions Questions

for self-control:

1. Cell pathology as an integrative concept. Pathology of the cell nucleus.
2. Pathology of mitosis, chromosomal aberrations and chromosomal diseases. Stereotypic damage of ultrastructures in response to various influences.
3. Pathological changes in cell membranes and changes in cells with damaged plasmolemma.

Pathological changes of the endoplasmic reticulum.

4. Pathological changes of the Golgi complex.
5. Pathological changes of mitochondria.
6. Pathological changes in lysosomes. Pathological changes of peroxisomes.
7. Pathological changes in the cytoskeleton (microfilaments, microtubules). Cell movement and its role in pathology.
8. Specific changes in ultrastructures: "diseases" of receptors, lysosomal, mitochondrial, peroxisomal "diseases".

2 Practical tasks

1. Prepare an essay on the topic: "Damage of mitosis and related diseases"

2. Make a graph of the logical structure "Ultrastructural pathology".

3. **Test tasks for self-control:**

1. An electron micrograph of the nerve cells of the spinal cord revealed organelles consisting of cisterns, flattened in the central part and expanded on the periphery, and small vesicles. What are these organelles called?

A*Golgi complex
B Centrioles
Lysosomes
DPeroxisomes Mitochondria

2. Maximum spiraled chromosomes were formed in the cell. They are located at the equator of the somatic cell. Which phase of mitosis does this correspond to?

A* Metaphase and
BTelophase and
CProphase and

Anaphase and
EPrometaphase and
3. Intensive aerobic accumulation of energy takes place in the muscles in the form of macroergic bonds of ATP. This process takes place with the participation of organelles:
A* M itochondrion
BGlad enkoy EPS
CL isosomes
D Granular EPS
ECl emergency center
4. Mucopolysaccharidosis refers to accumulation diseases. Due to the lack of enzymes, the breakdown of polysaccharides is disrupted. In patients, there is an increase in their excretion in urine and their accumulation. In which organelles is the accumulation of mucopolysaccharides?
A* Lysosomes
B Golgi complex
Endoplasmic reticulum
D Mitochondria
ECellular center
5. The parents of a sick 5-year-old girl turned to medical and genetic counseling. After the karyotype study, 46 chromosomes were found. One of the chromosomes of the 15th pair was longer than normal because a chromosome from the 21st pair joined it. What type of mutation does this girl have?
A* Translocation
B Deletion
C Inversion
D shortage EDuplication
6. As a result of radiation exposure, a section of the DNA chain turned 180 degrees. Which of the following types of mutations occurred in the DNA chain?
A*Inversion
B Deletion
CDuplication
DTranslocation
EReplication

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

1. Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
2. The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
3. Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

1. http://moz.gov.ua- Ministry of Health of Ukraine
2. www.ama-assn.org– American Medical Association /American Medical Association
3. www.who.int- World Health Organization
4. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
5. http://bma.org.uk– British Medical Association
6. www.gmc-uk.org- General Medical Council (GMC)
7. www.bundesaerztekammer.de– German Medical Association
8. http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory 9. http://www.webpathology.com/- Web Pathology

Topic #2: "Diseases of the musculoskeletal system. Parathyroid osteodystrophy, osteoporosis, Paget's disease, fibrous dysplasia, osteomyelitis, joint diseases, muscular dystrophies, myasthenia. Osteo- and cartilage tumors"

Purpose: as a result of independent study of this topic, students should know the classification and essence of changes associated with diseases of the musculoskeletal system, as well as the etiology, pathogenesis, pathological anatomy of these pathological conditions. Outcomes, complications of diseases associated with diseases of the musculoskeletal system.

Basic concepts:

The student should know:

4. Classification and essence of changes associated with diseases of the musculoskeletal system.
5. Etiology, pathogenesis, pathological anatomy of these pathological conditions.
6. Outcomes, complications of diseases associated with diseases of the musculoskeletal system.

The student should be able to:

- Classify diseases of the musculoskeletal system.
- To characterize the etiology, pathogenesis and morphological essence of these diseases

Topic content:

Diseases of the bone system The disease of this system can be caused by:

1. Dystrophic: toxic (Urov disease), alimentary (rickets), endocrine, nephrogenic. A significant place belongs to parathyroid osteodystrophy.
2. Incendiary
3. Dysplastic: fibrous dysplasia of bones, osteopetrosis, Paget's disease.
4. Neoplastic - often develop against the background of dysplastic. **Parathyroid osteodystrophy**

Parathyroid osteodystrophy (Recklinghausen's disease, generalized osteodystrophy) is a disease caused by hyperfunction of the parathyroid glands and accompanied by generalized damage to the skeleton. It occurs mainly in women aged 40-50.

Etiology. Parathyroid osteodystrophy is a consequence of primary hyperparathyroidism caused by adenoma of parathyroid glands or hyperplasia of gland cells.

Pathogenesis. Increased parathyroid hormone synthesis causes hypercalcemia with progressive demineralization of the entire skeleton. In the bone tissue, osteoclasts are activated, diffuse fibroosteoclasy increases - bone tissue is replaced by fibrous connective tissue. Bone deformation, osteoporosis, pathological fractures are possible. Formations resembling giant cell tumors appear in the changed cells. They are reactive structures that are built by giant cell granulomas.

Hypercalcemia leads to the development of calcareous metastases. Nephrocalcinosis often develops. Pathological anatomy. Adenoma, rarely cell hyperplasia, is often found in the parathyroid glands. Morphological changes of the skeleton depend on the stage and course of the disease. In the initial stage, they are completely absent, then they find deformation of the bones, especially the limbs, spine, ribs. They become soft, easily cut with a knife.

During microscopic examination, foci of lacunar resorption, neoplasms of fibrous tissue are found in bone tissue, giant cell granulomas, accumulation of erythrocytes and hemosiderin are possible in tumor-like formations.

The death of patients occurs from cachexia or uremia due to shrinkage of the kidneys. **Osteomyelitis**

Osteomyelitis is an inflammation of the bone marrow, which spreads to the spongy and compact substance of the bone and to the periosteum. According to the course, acute and chronic osteomyelitis are distinguished, according to the mechanism of infection - primary and secondary.

Primary hematogenous osteomyelitis

Acute hematogenous osteomyelitis is most common in young people. Chronic osteomyelitis is a consequence of acute.

Etiology. The causative agents of acute osteomyelitis are mostly purulent microbes: hemolytic staphylococcus, streptococcus, coliform bacilli, pneumococci, gonococci. It is most likely that patients with osteomyelitis have bacteremia with minor intestinal trauma, dental disease, and upper respiratory tract infection.

Pathogenesis. The purulent inflammatory process begins in the bone-marrow crevices of the metaphyses, where there is slowed blood circulation. Further, the process spreads to the bone marrow, where necrosis appears, and passes to the cortical layer of the bone, periosteum, and adjacent soft tissues.

Pathological anatomy. In acute hematogenous osteomyelitis, the inflammation has a phlegmonous nature. Resorption of bone tissue near the epiphyseal cartilage can end with the separation of the metaphysis from the epiphysis (epiphyolysis). Tissue infiltration by neutrophils appears around necroses; thrombi are found in the vessels of the compact plate. Abscesses often develop under the periosteum.

Chronic hematogenous osteomyelitis, as a result of acute, is accompanied by the formation of sequestrations, around which granulation tissue and a capsule are formed. From the sequestrations, fistulas go to the surface of the skin or to the body cavity. Along with the destruction of the bone in the periosteum and bone marrow canal, bone formation occurs - the bones become thick and deformed. Scars form in soft tissues.

Complications of primary hematogenous osteomyelitis: bleeding from fistulas, spontaneous fractures, formation of false joints, development of sepsis, secondary amyloidosis in chronic osteomyelitis.

Fibrous dysplasia

Fibrous dysplasia (fibrous osteodysplasia, Lichenschein-Breitsev disease) is a disease in which bone tissue is replaced by fibrous tissue, which leads to bone deformation.

Etiology and pathogenesis. The reasons for the development of the disease are unknown, perhaps hereditary factors are of some importance. It is believed that the tumor process is at the root of the disease. The disease begins in childhood, but can also develop in adults.

Classification. Depending on the spread of the pathological process, two forms of fibrous dysplasia are distinguished:

10. Monoosseous - pathological changes occur in only one bone. It can develop at any age.
11. Polyosseous - several bones are affected, mostly on one side of the body. Sometimes it is combined with melanosis of the skin. It develops in childhood.

Pathological anatomy. With the monoaxial form, pathological changes most often develop in the ribs, long tubular bones, shoulder blades, skull bones; with poliomyelitis - more than 50% of the bones of the skeleton, mainly on one side of the body. The damaged bone at the beginning of the disease retains its shape and size. In the future, "swelling", deformations of the bone, its lengthening or shortening appear. Femurs acquire the shape of a "shepherd's staff". On the cutting, clearly limited areas of whitish color with black-brown inclusions are determined. The bone marrow canal is expanded or filled with newly formed tissue. Upon microscopic examination, the centers of fibrous dysplasia are represented by fibrous fibrous tissue, which in some areas consists of randomly arranged bundles of mature collagen fibers and spindle-shaped cells, and in others - from thin collagen fibers and stellate cells. If fibrous dysplasia affects the bones of the face, then the dense component in the cells may be represented by cement-type tissue (cement-like formations). Complications are represented by pathological bone fractures, especially often in children, the femur is broken. A sarcoma may develop.

Osteopetrosis

Osteopetrosis (marble disease, congenital osteosclerosis, Albers-Schönberg disease) is a rare hereditary disease in which generalized excessive bone formation is observed, which leads to bone thickening, narrowing, and even complete disappearance of bone-marrow cavities. Osteopetrosis is characterized by a triad: increased bone density, bone fragility, and anemia.

Etiology and pathogenesis. Undoubted participation of hereditary factors, which are associated with a violation of the development of bone and hematopoietic tissue. The development of anemia, thrombocytopenia, the appearance of extraosseous hematopoietic centers in the liver, spleen, and lymph nodes is associated with the growing squeezing of bone marrow by the bone.

Classification. There are two forms of osteopetrosis:

4. Early (autosomal recessive) - appears at an early age, proceeds malignantly, often ends fatally.
5. Late (autosomal dominant) - a more benign course.

Pathological anatomy. The whole skeleton can be affected, but especially tubular bones, bones of the base of the skull, pelvis, spine, ribs. In the early form, the face acquires a characteristic appearance: it is wide, with widely spaced eyes, the root of the nose is depressed, and the lips are thick. With this form, hydrocephalus, increased hair growth, hemorrhagic diathesis, and multiple bone lesions are noted.

Characteristic column-shaped expansion of the lower femurs. On cuts in long bones, the medullary canal is filled with bone tissue and is often not defined. The spongy substance resembles polished marble.

The microscopic picture is peculiar: pathological ossification occurs throughout the entire bone, the bone substance is randomly accumulated in the internal parts of the bones. Osteoclasts are single, signs of bone resorption are insignificant. Bone architecture loses its functional characteristics. At the base of the cartilage, peculiar round islands of bone beams are formed.

Complications: bone fractures, especially femoral fractures, purulent osteomyelitis.

Causes of death. Patients often die in early childhood from anemia, pneumonia, sepsis.

Paget's disease

Paget's disease (deforming ostosis, deforming osteodystrophy) is a disease characterized by increased pathological remodeling of bone tissue, continuous changes in the processes of bone resorption and new formation, while the bone tissue acquires a peculiar mosaic structure. It is observed more often among men older than 40 years, progresses slowly, becomes noticeable only in old age. The lesion may involve a single bone (mono-osseous form) or several often paired or regional bones (poly-osseous form), but is never generalized.

Etiology. The reasons are unknown, the family nature of the disease is emphasized. Patho- and morphogenesis. Bone tissue reconstruction processes are continuous, there is no connection with functional load. There are three phases of the disease:

Initial (osteolytic) - the processes of bone resorption with the participation of osteoclasts prevail, deep lacunae are formed in the bone tissue.

Active (combination of osteolysis and osteogenesis) - osteoblasts appear, lacunae are filled with newly formed bone substance. The bone beams are built from small fragments forming a characteristic mosaic.

Inactive - the process of osteosclerosis prevails.

Pathological anatomy. Long tubular bones, especially the femur and tibia, are covered, sometimes spiral-shaped, which is due to the growth of the bone during its reconstruction. A narrow medullary canal is revealed on cuttings. When the periosteum is removed, there are numerous small openings of vascular channels on the surface of the cortical layer. On cutting, the cortical layer loses its compact structure and becomes almost spongy.

When the bones of the skull are damaged, only the bones of the brain skull are involved in the pathological process. The entire bone mass has an uneven spongy structure with pockets of rarefaction and compaction.

In the spine, the process involves one or more vertebrae in different parts of it, but never affects the entire spine. The vertebrae increase in volume or, on the contrary, flatten, depending on the stage of the disease. Focal points of osteoporosis and osteosclerosis are found on bone cuts.

Microscopic examination: determine small fragments of bone structures with uneven contours, with wide, well-defined basophilic adhesion lines. The areas of the bone fragments of the mosaic are usually calcified, their structure is irregular, thin-fibrous or lamellar. A large number of osteoblasts, axillary resorption cavities are found in the deep lacunae of bone structures. Signs of a bone neoplasm are noted: expanded bone cavities are filled with delicate fibrous tissue.

Complications: hemodynamic disorders (related to the expansion of blood vessels in the affected bone tissue), pathological fractures (develop in the active phase), osteogenic sarcoma (in 1-10% of patients, is localized in the thigh, lower leg, pelvic bones, in the scapula).

Diseases of the joints

Joint diseases can be associated with dystrophic processes of the structural elements of the joints (arthrosis) or their inflammation (arthritis). Among arthrosis, osteoarthrosis occupies a significant place, and among arthritis - rheumatoid arthritis.

Osteoarthritis

Osteoarthritis is one of the most frequent joint diseases of a dystrophic nature. Elderly women suffer more often. Osteoarthritis is divided into primary (idiopathic) and secondary (in other diseases). The pathological process develops in the joints of the lower limbs - pelvic-femoral, tibiofoot.

Etiology and pathogenesis. Hereditary (genetically determined disturbance of metabolism in articular cartilage) and acquired (mechanical trauma) factors are important.

Classification. There are three stages of osteoarthritis:

1. Pain in the joints during exercise, narrowing of the joint space and osteophytes (radiologically) are noted.
2. Pain in the joints becomes constant, the narrowing of the joint space and the development of osteophytes are more pronounced.
3. Along with constant pain, functional insufficiency of the joints due to the development of subchondral sclerosis is noted.

Pathological anatomy. Macroscopic changes depend on the stage of the disease. In the early stage, the edges of the articular cartilage appear fibrous, fibrous tissue. In the second stage, patterns and humps are found on the articular surface of the cartilage, bone growths - osteophytes - are formed.

In the third (late) stage, the articular cartilage disappears, depressions appear on the bones of the joints, and the joints themselves are deformed. The amount of synovial fluid decreases sharply. Microscopic changes: in the first stage, the cartilage retains its structure, the amount of glycosaminoglycans decreases in its surface and intermediate zones. In the second stage, shallow patterns appear in the surface zone of the cartilage, on the crowns of which chondrocytes accumulate. The pathological process also develops in the subchondral part of the bone. In the third stage, the surface zone and part of the intermediate zone of cartilage die, in the deep zone the number of glycosaminoglycans is sharply reduced, and the number of chondrocytes with pyknotic nuclei is increased.

Diseases of skeletal muscles

Among skeletal muscle diseases, the most widespread are striated muscle diseases of dystrophic (myopathy) and inflammatory (myositis) origin. Progressive muscular dystrophy and myopathy in myasthenia occupy a significant place among myopathies.

Progressive muscular dystrophy

Progressive muscular dystrophy (progressive myopathy) is a variety of primary hereditary chronic diseases of striated muscles. The disease is characterized by growing, often symmetrical, muscle atrophy, accompanied by progressive muscle weakness, almost to complete immobility.

Etiology and pathogenesis are little studied. The significance of abnormalities in structural proteins, sarcoplasmic reticulum, innervation, and enzymatic activity of muscle cells is discussed.

Classification. There are three main forms of progressive muscular dystrophy:

1. Duchenne (early form). The recessive type of inheritance associated with the Xchromosome occurs mainly in children aged 3-5 years. First, the muscles of the pelvic girdle, thighs and lower legs are affected, then the shoulder girdle and trunk.
2. Erba (youth form). Autosomal dominant type of inheritance, develops during puberty. Changes develop first in the muscles of the chest and shoulder girdle, sometimes in the face (smooth forehead, insufficient closing of the eyes, thick lips).
3. Leyden Autosomal recessive type of inheritance, begins in childhood or during puberty. It begins in the muscles of the pelvic girdle and hips, gradually covering the muscles of the trunk and limbs.

Pathological anatomy. Muscles are atrophic, thin, depleted of myoglobin, resemble fish meat at autopsy.

Upon microscopic examination, muscle fibers are different in size: along with atrophic ones, there are sharply enlarged (thickened) ones. Pronounced dystrophic changes of muscle fibers, their necrosis and phagocytosis. Adipose tissue accumulates between damaged muscle fibers.

Ultrastructural changes in muscle fibers in Duchenne muscular dystrophy: at the beginning of the disease, expansion of the sarcoplasmic reticulum, foci of destruction of myofibrils, and movement of nuclei to the center of the fiber are found. In the late stage, myofibrils are subject to fragmentation and disorganization, mitochondria swell. In the final stage of the disease, muscle fibers are compacted and surrounded by a hyaline-like substance.

The death of patients with a severe course of progressive muscular dystrophy occurs from a pulmonary infection.

Myasthenia

Myasthenia gravis is a chronic disease, the main symptom of which is weakness and pathological fatigue of the striated muscles. Normal contraction of muscles after their active activity decreases in strength and volume and may stop completely. Muscle rest time becomes longer in the late stage of the disease. Eye muscles (ptosis), masticatory, speech and swallowing muscles are most often affected. The disease occurs at any age, in women 3 times more often than in men.

Etiology and pathogenesis. The etiology is unknown. Correlation between thymus abnormalities and myasthenia occupies a significant place in pathogenesis. The development of the disease is

associated with a decrease of up to 90% in the number of acetylcholine receptors per unit of muscle plate, which is associated with autosomal reactions.
Pathological anatomy. In patients, follicular hyperplasia or thymoma is often found in the thymus. Skeletal muscles are slightly changed or in a state of dystrophy, sometimes accumulation of lymphocytes among muscle cells is revealed. IgG and C3 are also detected in postsynaptic membranes. Lymphoid infiltrates are found in the liver, thyroid gland, and other organs.
Complications arise when the respiratory muscles are damaged. An inadequate response of the lungs leads to the development of pneumonia and asphyxia, which, as a rule, become the cause of death.

1. Theoretical questions

Questions for self-control:

1. Describe the macro- and microscopic changes in the human body during parathyroid dystrophy,
2. Describe macro- and microscopic changes in the human body during primary hematogenous osteomyelitis,
3. Describe the macro- and microscopic changes in the human body during fibrous dysplasia,
4. Describe the macro- and microscopic changes in the human body during osteopetrosis,
5. Describe the macro- and microscopic changes in the human body during Paget's disease,
6. Describe the macro- and microscopic changes in the human body during osteoarthritis,
7. Describe the macro- and microscopic changes in the human body during progressive muscular dystrophy,
8. Describe the macro- and microscopic changes in the human body during myasthenia gravis.

2 Practical tasks

1. Prepare an abstract on the topic: "Pathological-anatomical features of ostermyelitis"

2. Make a graph of the logical structure "Diseases and pathological processes of bone tissue".

3. **Test tasks for self-control:**

1. In older people, there is an excessive loss of bone mass, which reflects the development of osteoporosis. Activation of which cells of bone tissue determines the development of this disease?
Osteoblasts.
Tissue basophils.
Osteocytes.
*Osteoclasts.
Macrophages.
Answer

2. In the histogenesis of bone tissue, two ways of its development are possible. What stages are not characteristic of membranous osteogenesis?
Replacement of coarse-fiber bone tissue with lamellar.
Formation of the mesenchyme of the osteogenic embryo.
Osteoid stage.
*Formation of the epiphyseal center of ossification.
Formation of coarse-grained bone.

3. A boy with a traumatic injury of the upper limb was brought to the hospital. A fracture of the humerus was detected during X-ray examination. At the expense of which structure will the reparative regeneration of the bone take place?
Diaphysis
* Oblast
A layer of external general plates.
A layer of internal general plates.
Epiphysis

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: All-Ukrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

3. http://moz.gov.ua- Ministry of Health of Ukraine
4. www.ama-assn.org– American Medical Association /American Medical Association
5. www.who.int- World Health Organization
6. www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
7. http://bma.org.uk– British Medical Association
8. www.gmc-uk.org- General Medical Council (GMC)
9. www.bundesaerztekammer.de– German Medical Association
10. http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
11. http://www.webpathology.com/- Web Pathology

Topic #3: "Tumors from cambial embryonic tissues. Tumors of children's age, which develop according to the type of tumors of adults.»

Purpose: as a result of independent study of this topic, students should know the topic for studying the characteristics of tumor growth in children at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:

The student should know:

12. - modern classification of childhood tumors;

13. - morphological manifestations of childhood tumors that develop according to the type of adult tumors;

14. -morphological manifestations of children's tumors developing from cambial embryonic tissues.

The student should know: The student should be able to:

12. interpret the modern classification of tumors in children;
13. interpret the peculiarities of the structure of tumors in children, which develop according to the type of tumors in adults;
14. to characterize the specifics of the development and ways of metastasis of malignant tumors in children, which develop according to the type of tumors in adults;
15. to explain the morphological features of the structure of children's tumors that develop from cambial embryonic tissues

Topic content:

Tumors from cambial embryonic tissues

*Medulloblastoma*is a malignant tumor from neuroectodermal embryonic stem cells - medulloblasts. It consists of oval rounded cells with scanty, almost indistinct cytoplasm. Cells are arranged in rosettes (forming ring-like structures), in the center of which cell processes are found. The formation of rhythmic structures is typical - in the form of cell rows or columns. It is believed that in the process of ontogenesis, medulloblasts differentiate into neuroblasts and spongioblasts. It is found mainly in children, it is localized in the cerebellum along the middle line - along the line of medullary tube closure. The tumor has a soft consistency, is grayish-pink in color, sprouts brain tissue and soft meninges. It metastasizes through the cerebrospinal fluid within the central nervous system. Very rarely gives hematogenous metastases in the lungs.

Retinoblastoma- a malignant tumor from embryonic undifferentiated cells of the retina. Some believe that medulloblasts are also the source of development. Tumor masses of grayish-yellow color, medullary, soft consistency. The tumor consists of round and oval cells, forms rosettes, is prone to necrosis, often has the appearance of cuffs located around vessels. Necrosis easily becomes calcareous. Retinoblastomas are more common in children under the age of 2, are congenital, and are bilateral. The tumor grows into the surrounding tissues, causes the eye to protrude, distorts the face, and grows into the area of the base of the skull. It metastasizes to the bones, liver, less often to the lungs, and also to the lymph nodes.

Neuroblastoma- malignant tumor from stem cells of sympathetic ganglia and medulla of adrenal glands. It is localized in the area of the adrenal glands or sympathetic ganglia of the neck, chest cavity, and abdominal space. It can have a multicentric origin - in both adrenal glands and in the ganglia of the chest cavity, etc. It has the appearance of a node in a thin capsule, which is often germinated, destroys the adrenal gland; on the section, the tumor is pinkish-white in color, with numerous necrosis and hemorrhages. Histologically, the tumor consists of round lymphocyte-like cells with a hyperchromic nucleus and barely visible cytoplasm - sympathogonium, hence the name of this undifferentiated type of tumor - sympathogonium. Cells form rosettes, in the center of which, during silvering, nerve processes are revealed. In the tumor, there are extensive fields of necrosis with karyorrhexis and hemorrhage. In more differentiated tumors - sympathoblastomas, the cells are larger, their cytoplasm is wider, the nuclei are lighter, there are giant cells, and the neurofibrillary network is more pronounced. An even more differentiated type of tumor - ganglioneuroblastoma, is characterized by the presence of atypical ganglion cells. Cases of transition of undifferentiated neuroblastoma into mature benign ganglioneuromas or ganglioneurofibromas both spontaneously and under the influence of treatment have been described.

Neuroblastoma grows quickly and metastasizes widely. There are two types of metastases: 1) in the regional lymph nodes and in the liver; 2) in the bones of the skeleton - ribs, spine, bones of the pelvis and skull. Sometimes there are multiple metastases in the skin. The clinical course in children can be accompanied by an increase in blood pressure, sweating due to the secretion of catecholamines by tumor cells, which can be detected in the patient's blood and urine. Neuroblastoma is more common in children under the age of 1, but can occur up to 11 years of age.

Cases in fetuses and newborns are described. In adults, it is described as a case study.

Tumors in children that develop according to the type of tumors in adults

Juvenile fibroma of the nasopharynx should be noted among benign soft tissue tumors. It occurs in children aged 8 years and older and in young subjects up to 18 years old. It has a dense consistency and the appearance of polypous growths covered with a mucous membrane, is localized in the nasopharynx, grows very quickly, fills the nasal cavity, grows into the bones of the facial skeleton, the base of the skull. It often turns out to be bleeding, infected. Histologically, it is a fibroma with the presence of juicy fibroblasts and a large number of thin-walled vessels. It is difficult to radically remove the tumor, it often recurs after surgery. Sometimes it undergoes spontaneous regression after puberty. Although the structure of the tumor is benign and metastases are not observed, the clinical course and prognosis are unfavorable.

Common tumors in children are bone tumors: benign - osteomas and chondromas, osteoblastoclastomas and malignant - osteosarcomas, Ewing's sarcomas. Benign chondromas or socalled cartilaginous exostoses are often found in school-aged children in the extremities.

Osteosarcomas (including Ewing's sarcoma) account for 18% of all sarcomas in children, chondrosarcomas in children are rare. In half of the cases, osteosarcoma is localized in the lower metaphysis of the thigh, it occurs mainly in children aged 11 to 14 years. It has a very fast progressive growth and widely metastasizes (see the morphology in more detail in the textbook of A.I. Strukov "Pathological Anatomy", "Tumor").

Ewing's sarcoma is observed in children aged 10 to 18 years, is more often localized in long tubular bones (in diaphyses). It has the appearance of whitish-yellowish nodes with areas of necrosis and hemorrhages. It consists of small cells with a rounded nucleus, creating continuous fields, with the presence of thin-walled vessels. The histogenesis of the tumor has not been definitively studied. It grows relatively slowly, gives late hematogenous metastases in the lungs, as well as in the bones of the skeleton.

Astrocytoma is a tumor that develops from astrocytic glia, there are juvenile and adult types. The adult form in children is less common than the juvenile form. It is localized in the hemispheres and brain stem. Can grow quickly. It consists of polymorphic stellate and spindle-shaped cells, there are giant forms, figures of mitoses. There may be polygonal figures around the foci of necrosis. Acute lymphoblastic leukemia is the predominant form of acute leukemia in children, while acute myeloblastic leukemia predominates in adults. Acute lymphoblastic leukemia is the most common form of leukemia in children. Thrombocytopenia and anemia are emphasized. Hemorrhagic diathesis is expressed moderately. Leukemic infiltration of lymph nodes, spleen, liver, bone marrow, kidneys, endocrine and exocrine glands is observed, with a particularly significant increase in organ mass. The bone marrow when dissected is crimson-red, juicy. Leukemic infiltration of the thymus leads to a significant increase in the mass of the organ with a complete loss of its structure.

Acute myeloblastic leukemia is characterized by a predominant lesion of the bone marrow, which usually has a grayish, sometimes even greenish color, lymph nodes and tonsils can be enlarged in the same color. Leukemic infiltration of the liver in the form of growths along the portal tracts and sinusoids is especially pronounced. Tumor infiltrates occur less often than in lymphoblastic leukemia, and are localized mainly in the bone marrow, sometimes in the stomach, kidneys and liver in the form of separate nodules - myeloma. In the bone marrow, they sprout periosteum and surrounding soft tissues. Sometimes such tumor nodes are located in the bones of the skull, in the ribs, vertebrae. Hemorrhagic diathesis can be expressed significantly, which gives the organs a variegated appearance due to the presence of blackish foci of hemorrhages. More often than with lymphoblastic leukemia,

Acute monoblastic leukemia is characterized by a moderate number of blast forms in the peripheral blood, is characterized by a nested lesion of the bone marrow and sometimes unusual localization of leukemic infiltrates (esophagus, ureters), pronounced necrotic changes in tissues and organs.

Adult-type chronic myeloid leukemia is characterized by the presence of the Philadelphia chromosome in leukemic cells, very high leukocytosis, mainly observed in girls aged 11-13 years, pronounced hepato- and splenomegaly, hemorrhagic diathesis and generalization of the process in the terminal period of the disease.

Malignant non-Hodgkin's lymphomas (NHL) in children are observed mainly with a high degree of malignancy. Undifferentiated lymphoblastic CKD - the process begins in the lymph nodes (cervical, axillary, mediastinal, abdominal cavity) and outside the nodes - in the stomach, intestines, nasopharynx, tonsils. The growth can be infiltrating, creeping, as well as destructive with germination outside the organs. The uniformity of the general appearance of the tumor is characteristic, with loss of the l/node pattern, sprouting of its capsule. Follicular-like structures are never present, as tumor growth is diffuse. Tumor cells are small and medium-sized, the nucleus is rounded, the cytoplasm is in the form of a narrow basophilic rim, and it contains SHIK - positive granules. Many mitoses, single macrophages. The process ends in such a way that it is generalized in most cases and leukemia.

Burkitt's sarcoma endemic to Equatorial America, which is consistent with the idea of the viral nature of this sarcoma, belongs to the HCVL with B - differentiation. In addition to the typical localization in the upper jaw, the primary node can be located in the abdominal cavity, retroperitoneally. The localization of the primary node outside the lymphoid organs and the picture of the so-called starry sky are characteristic. The latter is due to the presence of a large number of light-colored macrophages with a wide rim of cytoplasm. The tumor grows quickly, the process ends in such a way that it becomes hematogenous and generalizes with damage to many organs - bone marrow, liver, kidneys, gonads.

A lymphoma with a twisted nucleus (Sternberg's sarcoma) belongs to lymphoblastic HCV with T - differentiation. It occurs mainly in children aged 10-15 years. The primary node is localized in the thymus, the bone marrow is involved only in the late stage of the disease. Leukemia is often observed. The tumor consists of small and medium-sized lymphoid cells with a thin rim of basophilic cytoplasm. The nucleus of some cells is twisted, many cells have mitoses. Like thymus lymphocytes, the cells give a focal positive reaction to acid phosphatase.

Lymphogranulomatosis is less common in children than in adults. Of the 4 histological options, the first option with a predominance of lymphoid proliferation and mixed - cellular, as options with a more favorable prognosis, are more common in children. In children, forms with damage to peripheral lymph nodes without typical general symptoms of the disease prevail. L/nodes of the neck are enlarged in 80% of cases, while the mediastinal lymph nodes of the abdominal cavity are also very often involved. Death occurs from the fact that the process is generalized, intercurrent infectious diseases, in some cases from general amyloidosis.

1. Theoretical questions Questions for self-control:

6. Morphological characteristics of tumors in children developing from cambial embryonic tissues.
7. Morphological characteristics of children's tumors that develop according to the type of tumors in adults.

2 Practical tasks

1. Prepare an essay on the topic: "Features of childhood tumors"

2. Make a graph of the logical structure "Classification of teratoma".

3. **Test tasks for self-control:**

1). A 6-month-old child has a flat red nodule on the skin of the neck, the nodule turns pale when pressed with a glass. What is the most likely diagnosis? - Leiomyoma + Hemangioma - Pigment nevus - Melanoma - Lymphangioma

2). During the histological examination of the cystic formation of the skin, it was found that the structural component of the cyst wall is the appendages of the skin. What type of pathology corresponds to the specified changes?- Epidermal cyst- Tricholemoma cyst+ Dermoid cyst- Steatocystoma- Hydroadenoma

3). In a 6-year-old child with a tumor of the diaphysis of the femur, the presence of several metastatic foci of other bone localization was noted during the examination. In the histological description, it is indicated that the primary tumor mainly consists of rounded cells with poor cytoplasm, which have a slight tendency to form pseudorosettes, and manifest themselves by single mitoses. The specified changes are characteristic of:- Plasmacytomas- Chondromas- Osteosarcomas- Fibrosarcomas+ Ewing's sarcomas

4) In a 14-year-old boy, an increase in the volume of the lower third of the thigh, local hyperemia, and an increase in the venous pattern in the area of the pathological process were found. Radiologically, the cortical layer of the femur is destroyed, the structures of the knee joint are preserved. Examination of the biopsy revealed a cluster of atypical osteoblasts with multiple mitoses and areas of abnormal bone beams, signs of invasion into adjacent tissues. Diagnose the disease.+ Osteogenic sarcoma- Chondrosarcoma- Fibrous dysplasia- Osteoid-osteoma- Osteoblastoclastoma

5) Hair and teeth were found in the tumor removed from the abdominal cavity. To which tumors is this formation attributed by histogenesis?+ Teratomas- Mesenchymal- Embryonic- Epithelial- Organ-specific

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization

- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory

 http://www.webpathology.com/- Web Pathology

Topic #4: "Cardiomyopathies. Leffler's endocarditis. Idiopathic myocarditis, acquired heart defects. Systemic vasculitis.»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:

The student should know:

15. The meaning, etiology of the pathogenesis of acquired defects, their results and consequences.
16. Morphological substrate of acquired heart defects.
17. Clinical and anatomical forms of acquired defects;
18. Types of cardiomyopathies;
19. Morphofunctional changes in Leffler's fibrous parietal endocarditis;
20. Morphofunctional changes in alcoholic cardiomyopathy; The student should know: The student should be able to:

16. Distinguish morphological manifestations of acquired heart defects;
17. Master the skills of interpreting the results of a macro- and microscopic study based on the study of macropreparations: "alcoholic cardiomyopathy", "mitral valve stenosis" and micropreparations: "alcoholic cardiomyopathy".
18. Differentiate different types of defects among themselves;
19. Study and explain morphological changes, describe the macropreparation;

Topic content:

Acquired heart defects are characterized by damage to the valvular apparatus of the heart and main vessels. These are stable deviations in the structure of the heart, disrupting its function. Occur as a result of heart diseases after birth. Among these diseases, rheumatism is of great importance, atherosclerosis, syphilis, bacterial endocarditis, brucellosis, and trauma are less important. The formation mechanism is closely related to the evolution of endocarditis, the organization of thrombotic masses, which culminates in scarring, petrification, and deformation of valves and fibrous rings.

Clinical and anatomical forms of organic heart disease:

1) Stenosis of the valve
2) Insufficiency of the valve
3) Combined defect

Stenosis of the mitral valve - develops at the level of the fibrous ring, the opening has the appearance of a narrow slit that resembles a "button loop". There is an obstruction of the current in the small circle, the left atrium expands, its wall is thickened, the endocardium is sclerosed. As a result of hypertension in the lesser circle, the walls of the right ventricle undergo hypertrophy with expansion of the cavity.

Insufficiency of the mitral valve - the progression of sclerosis, and therefore defects are often caused by repeated attacks of rheumatism (endocarditis), as well as hyperplastic changes of the valve. Vessels appear in the leaflets of the valve, the connective tissue thickens, turns into scarred, sometimes calcified, fused formations. Chords are sclerosed, shortened. As a result of the reverse flow of blood during diastole, the left heart overflows with blood, compensatory hypertrophy of the left ventricle develops.

Aortic valve defect - develops more often against the background of rheumatism, in connection with the same processes that form a mitral valve defect. Valves grow together, thicken, become sclerosed and calcified, which in some cases leads to predominance of stenosis, in others to insufficiency of valves.

In atherosclerosis, calcification and sclerosis are combined with lipoidosis and liposclerosis. With septic endocarditis and brucellosis - sharp destruction (patterns, perforation holes, aneurysms) of valves and their deformation due to pronounced petrification.

Syphilitic blight is combined with mesoorthitis. With aortic defects, the heart undergoes significant working hypertrophy, mainly due to the left ventricle.

In addition to isolated defects, combined defects are often observed: mitral-aortic, mitral-tricuspid, mitral-aortic-tricuspid.

Acquired heart defects are compensated and decompensated.

Compensated defect - occurs without circulatory disorders, often prolonged, latent.

Decompensated heart disease - leads to cardiovascular failure. The cause of decompensation is exacerbation of the rheumatic process, accidental infection, excessive physical exertion, mental trauma. The heart becomes sluggish, cavities expand, blood clots form in the ears. Protein and fatty dystrophy of muscle fibers is revealed, in the stroma of foci of inflammatory infiltrate.

Causes of death:

1. Cardiovascular insufficiency;
2. Thromboembolic syndrome;
3. Paralysis of the hypertrophied heart;
4. Pneumonia, etc.;

CLASSIFICATION OF CARDIOMYOPATHIES:

1. Hypertrophic obstructive cardiomyopathy:

1) Family

2) Non-family;

2. Congestive cardiomyopathy:

1) idiopathic;

2) family;

3) postpartum;

4) tropical;

5) endomyocardial fibrosis (fibrous parietal endocarditis of Leffler).

3. Alcoholic cardiomyopathy;

4. Cobalt (beer) cardiomyopathy;

5. Medicinal cardiomyopathy;

6. Cardiomyopathy associated with hereditary diseases;

7. Cardiomyopathy with endocrine disorders:

1) thyrotoxicosis;

2) hypothyroidism;

3) acromegaly.

8. Cardiomyopathy with amyloidosis;

9. Cardiomyopathy in hemochromatosis.

Hypertrophic obstructive cardiomyopathy is a special disease of the heart caused by hypertrophy of the myocardium and obstruction of the outflow tract of the left ventricle. It is observed in the age range from 2.5 to 70 years.

Macroscopically, it is characterized by two main features:

1) sharply expressed symmetric or asymmetric hypertrophy of the left ventricle and septum, sometimes in combination with hypertrophy of the right ventricle.

2) Reduction of the cavity of the left ventricle with obstruction of the outflow tract.

Microscopic changes consist of the following signs:

1. hypertrophy of muscle fibers;

2. decrease in their length due to fibrosis;

3. the presence of ugly hyperchromic nuclei of cardiac myocytes;

4. uneven focal and interstitial fibrosis;

5. the presence of areas of the myocardium with unusual microarchitectonics.

The disease can be asymptomatic for many years. After the appearance of clinical symptoms (shortness of breath, heart pain, arrhythmia), they usually die quickly, often suddenly.

Congestive cardiomyopathy is characterized by expansion and hypertrophy of the entire heart and moderate thickening of the endocardium, which is often based on mural thrombosis. The age of

patients is more often 40-50 years. The main manifestation of congestive cardiomyopathy is left ventricular failure.

A provoking factor in the development of heart failure often becomes a respiratory infection.

Macroscopically, the heart has a spherical shape, weighing up to 1000 g or more. The cavities of the atria and ventricles are expanded. There are extensive areas of fibrosis of the parietal endocardium. In the cavities of the atria and ventricles, wall thrombi. The perimeter of the atrioventricular openings is significantly increased, the leaflets and tendinous threads are elongated. Papillary muscles are hypertrophied.

Microscopic changes consist of uneven hypertrophy of muscle fibers, myocytolysis, collapse of the stroma, replacement and interstitial fibrosis, focal replacement fatty dystrophy of muscle fibers, foci of fresh necrosis and fuchsinophilia of cardiac myocytes with pyknosis of their nuclei.

The cause of death is heart failure, heart rhythm disorders, thromboembolism.

Tropical cardiomyopathy includes endomyocardial fibrosis (fibrous parietal endocarditis of Leffler) - it is observed at the age of 10 to 30 years. Cases of this disease have been described in Uganda, Sudan, Tanzania, Zambia, Kenya, Nigeria, West African countries, South India, and Sri Lanka. Certain diseases were observed among Europeans who lived for a long time in Africa and among black Americans. The etiology remains unclear, but judging by the limited geographic distribution, it is associated with:

1) environmental factors;
2) a genetic factor;
3) febrile state of unclear etiology (100% - filariasis);
4) autoimmune nature (antimyocardial antibodies were found); 5) manifestation of a hyperergic reaction.

Macroscopic picture:

-When the right or left half of the heart is damaged, the endocardium, papillary muscles, and chordae are replaced by fibrous tissue, sclerosis spreads to adjacent parts of the myocardium; - thrombotic deposits on the surface of the sclerosed endocardium.

Microscopic picture:

-In the endocardium, areas of disorganization and necrosis of collagen fibers, foci of calcification, fibrin deposition with phenomena of its organization;

• In the myocardium, foci of young connective tissue, rich in capillaries and infiltrated with lymphocytes, histiocytic and plasma cells with an admixture of eosinophils. Focal accumulation of glycosaminoglycans and areas of disorganization and necrosis of collagen fibers. Fibrous tissue of varying degrees of maturity penetrates into the subendocardial layers of the myocardium. Muscle

fibers are atrophic, with pyknotic nuclei; there are foci of hyaline necrosis, there may be focal hypertrophy of myocytes. Small perivascular lymphocytic infiltrates are found in the myocardium.

ALCOHOLIC CARDIOMYOPATHY - currently identified as an independent form of cardiomyopathy found in alcoholics. The pathogenesis of this type of cardiomyopathy is primarily related to the biological properties of ethanol - its direct toxic effect on cardiomyocytes, as well as the effect of ethanol's metabolite - acetaldehyde. Vascular disorders and associated hypoxia, as well as the damaging effect of catecholamines on the myocardium, are of absolute importance.

Macroscopically: the heart is enlarged (500-600g). The cavities are enlarged, the wall of the left ventricle is thickened, the myocardium is flaccid, clay-like. Atrial endocardium with foci of fibrosis (organized atrial thrombi) and fresh thrombotic overlays. Sometimes there is a "tiger heart" - yellow stripes corresponding to areas of fatty dystrophy, focal sclerosis of the left ventricle.

Microscopic picture:

- uneven hypertrophy with foci of atrophy;
- vacuolar dystrophy and homogenization with loss of transverse markings;
- foci of necrosis of varying degrees of antiquity with phenomena of organization;
- lymphoid infiltrates in the stroma;
-accumulation of neutral lipids in cardiomyocytes.

Vasculitis

Vasculitis is a disease characterized by inflammation and necrosis of the vessel wall; the process can be local or systemic. Local vasculitis occurs in the foci of inflammation as a result of the transition of the process to the vessel wall from the adjacent tissues (eg, purulent-necrotic vasculitis in phlegmon). Systemic vasculitis, which can be the basis of independent manifestations (primary vasculitis) or a manifestation of any other disease (secondary vasculitis), is characterized by widespread swelling of vessels.

Depending on the type of inflammatory reaction, alterative-exudative or productive changes, wax is divided into necrotic (destructive), destructive-inductive, and productive, distinguishing separately the lematous branch. \ hiding the depth of damage to the blood vessels: i.e. involvement in the inflammatory process of the inner, middle and outer sheath, endovasculitis, mesovasculitis, perivasculitis, and mesovasculitis and panvasculitis are distinguished in the case of a combined lesion of the membranes. The vast majority of systemic vasculitis is characterized by damage to all membranes of the vessel wall with the transition to sclerosis and calcinosis, which leads in some cases to sharp stenosis and even obliteration of vessels, in others to the development of an aneurysm. The classification system of systemic vasculitis takes into account the following criteria (V.V. Syerov, O.E. Kogan, 1982): etiology, pathogenesis, nosological affiliation, predominant nature and

prevalence of the inflammatory reaction, morphological type of affected vessels, cervical localization, which determines the interest of certain organs (organopathology), clinical manifestations of the disease. At the same time, the nosological principle should be followed, based on which vasculitis is divided into primary and secondary.

Classification of systemic vasculitis A.

Primary vasculitis.

I. With predominant damage to the aorta and its large branches and a giant cell granulomatous reaction: nonspecific aortoarteritis (Takayasu's disease), temporal arteritis (Horton's disease). II. With a predominant lesion of medium- and small-caliber arteries and a destructive-productive reaction: 1) nodular periarteritis; 2).allergic granulomatosis; 3) systemic necrotizing vasculitis; 4) Wegener's granulomatosis; 5) lymphatic syndrome with damage to the skin and mucous membranes.

III. With predominant damage to small-caliber arteries, vascular microcirculatory bed and veins: obliterative thromboangiitis (Buerger's disease).

IV. With damage to arteries of different calibers — a mixed (unclassified) form.

B. Secondary vasculitis.

V. With infectious diseases: 1) syphilitic; 2) tuberculosis; 3) rickettsial, including typhoid fever; 4) septic; 5) others.

In addition to the respiratory tract, granulomas can also be found in the kidneys, skin, joint tissues, liver, heart, and other organs. As a result, granulomatous lesions lead to sclerosis and deformation of organs.

Gromeluronephritis is a rather characteristic feature of Wegener's granulomatosis. Most often, it is a manifestation of interangioproliferative or mesangiocapillary form - with fibrinoid necrosis of capillary loops and glomerular arterioles and extracapillary reaction (formation of characteristic "crescents"). In most cases, simultaneous damage to the respiratory tract, lungs, and kidneys is observed.

Obliterating thromboangiitis

Obliterating thromboangitis (Winiwarter-Buerger's disease) is a systemic vasculitis in which mainly small arteries and veins of the lower extremities are affected, which leads to occlusion of these vessels.

The causes of the disease and the mechanisms of its development are unknown. But smoking is of utmost importance. Men under the age of 40 are more often ill. In this disease, damage to the veins of the lower extremities prevails, primarily productive ENDO-, meso- and periphlebitis develops. In the arteries of the lower extremities, which are less affected than the veins, similar processes develop — productive endo-, meso- and periarteritis. The vessels take on the appearance of thick fibrous cords with segmental thickening of the walls.

Acute, subacute and chronic stages of the disease are distinguished. The development of alterativeproliferative thrombovasculitis is characteristic of the advanced stage. Alterative changes are joined by infiltration of the vessel wall and perivascular tissue by polymorphonuclear leukocytes, which cause the destruction of the internal elastic membrane, and sometimes even the formation of microabscesses. In the subacute stage, a productive tissue reaction prevails. Lymphohistocytic infiltrates, signs of excessive vascularization and early formation of thrombi are found in the vessel wall. The formation of granulomas, which are found mostly in the middle shell and around necrotized fragments of the internal elastic membrane, as well as in thrombotic masses, is typical. Granulomas resemble either oleogranulomas or tuberculous granulomas. In the chronic stage, signs of the organization of blood clots dominate, which leads to complete obliteration of the vessel. The organization of blood clots can be accompanied by their drainage and calcification.

1. Theoretical questions Questions for self-control:

1. Definition of acquired heart defects, their classification.
2. Morphological criteria and dynamics of morphological changes in acquired heart defects;
3. Etiology. Pathogenesis of acquired heart defects;
4. Clinical and anatomical forms of heart defects;
6. Definition of cardiomyopathies, their classification;
7. Etiology, pathogenesis of cardiomyopathies;
8. Morphological characteristics of various types of cardiomyopathies;
9. Leffler's endocarditis;
10. Consequences, complications, causes of death in cardiomyopathies and heart defects
11. Define and classify the concept of vasculitis.
12. Criteria for morphological assessment of systemic vasculitis, types of inflammatory reactions in vasculitis.
13. Classification of vasculitis according to the depth of damage to the vascular wall and their localization.

14. Changes in organs and tissues with vasculitis.
15. Classification of systemic vasculitis.
16. Pathomorphology of nonspecific aortoarteritis.
17. Pathomorphology of Wegener's granulomatosis.
18. Pathomorphology of obliterating thrombogenitis.

2 Practical tasks

1. Prepare an essay on the topic: "Clinical and anatomical forms of heart defects"
2. Make a graph of the logical structure "Classification of systemic vasculitis.".

3. **Test tasks for self-control:**

1. In which of the listed defects is the left atrium most often significantly enlarged?
8. valvular stenosis of the pulmonary artery
9. mitral valve insufficiency
10. mitral valve stenosis
11. coarctation of the aorta

2. Mitral stenosis in combination with aortic insufficiency is characterized by a shift in the boundaries of cardiac dullness: up and to the right up and to the left up, left and down not changed

3. In a patient with pronounced mitral stenosis on the ECG: answer options
4. wide jagged P and deviation of the axis to the right
5. wide jagged P and hypertrophy of the right ventricle
6. wide jagged R
7. all of the above

4. With rheumatism, the following are most often affected:
answer options
4. mitral and tricuspid valves
5. tricuspid valve and pulmonary artery valves
6. valves of the pulmonary artery and aorta
7. mitral and aortic valves

5. A 42-year-old patient applied to the reception room of the cardiology center with complaints of severe shortness of breath at rest, palpitations, lethargy, aching and stabbing pains in the heart, which are partly associated with physical exertion, severe swelling of the legs. He considers himself sick during the year, when for the first time, for no apparent reason, pain in the heart, shortness of breath, and a feeling of interruptions appeared. He was treated in an inpatient hospital and on an outpatient basis with partial improvement in his condition. However, 3 months after physical exertion, he began to feel much worse, shortness of breath began to progress, palpitations became frequent, and swelling appeared on his legs. Objectively: the general condition is severe, orthopnea, tachypnea, acrocyanosis. The pulse is arrhythmic, 98 in 1 minute, BP 150/80 mmHg. Limits of relative dullness of the heart: right - 1 cm outward from the right edge of the sternum, upper - second intercostal space, left - 2, 5 cm outward from the left

midclavicular line in the fifth intercostal space. Auscultatively - the rhythm of cardiac activity is irregular, extrasystole, tones are muffled, systolic murmur and protodiastolic murmur over the apex and at Botkin's point. In the lungs, breathing is vesicular. The abdomen is soft, the liver protrudes 3 cm from under the edge of the costal arch. Swelling of the lower legs and feet. ECG - ventricular extrasystole of the bigimen type. Diagnosis: dilated cardiomyopathy. NC II B. dilated cardiomyopathy. NC II B. dilated cardiomyopathy. NC II B.

Which of the following is the most favorable screening test?

9. X-ray of chest organs with contrast esophagus
10. echocardioscopy
11. electrocardiography
12. phonocardiography

6. Woman S., 25 years old, turned to a cardiologist with complaints of long-lasting, intense pain in the area of the heart of an aching, stabbing nature with radiation to the left upper arm, shoulder blade, shoulder, which does not increase with physical exertion, rapid fatigue, marked lethargy, which intensifies in the afternoon. Lethargy bothers more than cardialgia, a feeling of dissatisfaction with breathing that occurs at rest, palpitations. Sick for a month. The listed complaints appeared after a lacunar sore throat. Suffers from chronic tonsillitis. On objective examination - tachycardia, displacement of all limits of relative dullness of the heart by 1 cm. Heart sounds are sonorous, systolic murmur. In the lungs, breathing is vesicular. The abdomen is soft, the liver is not enlarged, there is no edema. On the ECG – migration of the supraventricular pacemaker, atrial extrasystoles,
   - rheumatic tests
   - bicycle ergometry
   - echocardioscopy
   - Obzydan test

7. What diseases are most often complicated by heart defects?
   - rheumatoid arthritis
   - myocardial infarction
   - rheumatism
   - angina pectoris

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
- http://www.webpathology.com/- Web Pathology

Topic #5: "Alzheimer's disease. Multiple sclerosis. Amyotrophic lateral sclerosis. Postresuscitation encephalopathy. Diseases of the peripheral nervous system.»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:

The student should know:

21. Classification and essence of CNS diseases.

22. Etiology, pathogenesis, pathological anatomy of these diseases.

23. Outcomes and complications of these CNS diseases.

The student should know: The student should be able to:

20. Classify CNS diseases;
21. To characterize the etiology, pathogenesis and morphological essence of dystrophic diseases of the central nervous system.
22. To characterize the etiology, pathogenesis and morphological essence of demyelinating diseases of the central nervous system.
23. To characterize the etiology, pathogenesis and morphological essence of inflammatory diseases of the central nervous system.
24. To characterize the etiology, pathogenesis and morphological essence of CNS tumors.

Topic content:

CNS diseases are divided into:

- I dystrophic
- II demyelinating
- III incendiary
- IV tumors

In dystrophic (degenerative) diseases, damage to neurons prevails in any localization of the process: cerebral cortex in Alzheimer's disease, basal ganglia and midbrain in Hutchinson's chorea, parkinsonism, motor neurons in amyotrophic lateral sclerosis. Dystrophic diseases are diseases with a deficiency of substances (thiamine, vitamin B12), metabolic disorders (primary encephalopathy, exposure to toxic (alcohol) or physical (irradiation) factors).

Demyelinating diseases - in which the myelin sheaths are primarily damaged - primary demyelinating diseases; secondary demyelination is associated with axonal damage (for example, multiple sclerosis).

Inflammatory diseases are divided into meningitis in childhood infections and encephalitis.

CNS tumors

Alzheimer's disease

It develops presenile (presenile dementia or dementia). A number of authors include senile (senile) dementia, as well as Pick's disease. Senile dementia includes progressive dementia in people aged 40-65 years. When the disease appears after the age of 65, it is called senile dementia, and when a speech disorder is added, it is called Pick's disease.

Alzheimer's disease progresses with pronounced intellectual disorders and emotional lability, while focal neurological symptoms are absent. The cause of the disease is not clear enough. Previously, a deficiency of acetylcholine was assumed. Recently, a connection with senile demented cerebral amyloidosis has been established. Deposition pathology of intracellular fibrillar structures - proteins of the cytoskeleton. The pathology of amyloidosis is found in senile plaques, vessels of the brain and membranes, and in vascular plexuses. Along with the synthesis of extracellularly located amyloid fibrils, which are the basis of the senile plaque, in Alzheimer's disease, the cytoskeleton is expressed in the accumulation of actin microfilaments - Hirano bodies - in the proximal dendrites.

At autopsy, atrophy of the cerebral cortex is found in the frontal, temporal, and occipital lobes, hydrocephalus often develops. Microscopic examination reveals senile plaques, neurofibrillary tangles (tangles), Hirano bodies in the cortex of atrophied brain lobes, the hippocampus, and amyloids. There are no changes in motor and sensitive areas. The cause of death is respiratory infection, bronchopneumonia.

Amyotrophic lateral sclerosis

Charcot disease is a progressive disease of the nervous system associated with simultaneous damage to the motor neurons of the anterior and lateral columns of the spinal cord and peripheral neurons. Characteristic: slow development of spastic paresis, mainly of hand muscles, muscle atrophy, increase of tendon and suprabony reflexes. Men get sick 2 times more often, mostly middleaged, it ends in death after 2-6 years. The cause and mechanism of disease development are unknown. It is assumed that the disease is associated with a chronic viral infection (polio).

Selective atrophy of anterior motor roots of the spinal cord, posterior-normal. Lateral corticospinal tracts are compacted, white in color. Sometimes there is atrophy of the precerebral gyrus of the cerebrum, as well as VIII – X and XII pairs of cranial nerves. In all cases, skeletal muscle atrophy was pronounced. Microscopic examination reveals pronounced changes in nerve cells: they are wrinkled or in the form of shadows, large fields of neuronal loss. In nerve fibers - demyelination, uneven swelling, then disintegration and death of axial cylinders. The reactivity of glial proliferation is observed.

Cause of death: cachexia or aspiration pneumonia.

Multiple sclerosis

- (multiple sclerosis) is a chronic disease that is characterized by the formation of scattered sclerosis cells - plaques - in the brain and spinal cord (white matter). The frequency of the disease is at the age of 20-40 years, more often in men. The course is wavy. Multiple localization of damage centers determines the variety of clinical manifestations of the disease: nystagmus, slurred speech, sharp increase in tendon reflexes, spastic paralysis, visual disturbances. An acute and severe course with the rapid development of blindness and cerebellar disorders and a mild course with minor dysfunction of the central nervous system and its rapid recovery are possible.

The reasons are unclear, the probable viral origin of the disease: a virus tropic to oligodendroglia cells.

Well-studied morphogenesis of sclerotic plaques in multiple sclerosis. Fresh foci of demyelination appear around the veins, which are combined with remyelination. The vessels expand and are surrounded by infiltrates of lymphoid and plasma cells. In response to destruction, the proliferation of glia occurs, myelin breakdown products are phagocytosed by macrophages, then sclerosis develops.

Externally, the brain and spinal cord have changed little. On its sections, a large number of gray plaques are found, which are scattered in the white matter with a diameter of up to several centimeters. There are many plaques, especially around the ventricles of the brain, in the spinal cord and medulla oblongata, brain stem, optic tubercles, white matter of the cerebellum, the chiasm, optic nerves, optic pathways are often affected.

Under microscopy, at an early stage, foci of demyelination are found around vessels, especially veins and venules, perivenous demyelination. Vessels are surrounded by lymphocytes, mononuclear cells; axons are relatively preserved. Myelin sheaths swell, their tinctorial properties change, contours are uneven, globular thickening along the fibers. Then there is fragmentation and disintegration of myelin sheaths, the products of which are phagocytized by microglia, turning into granular balls.

During the progression of the disease (late stage), small perivascular cells are demyelinated, merge, proliferates from microglial cells, cells loaded with lipids appear. At the end, typical plaques are formed in which oligodendrites are absent. The cause of death is pneumonia.

Brain damage in cardiac arrest

(post-resuscitation encephalopathy)

Many patients with severe diffuse cerebral lesions resulting from cardiac arrest die within a few days. Brain damage is usually limited to selective neuronal necrosis (a necrotic process affecting only neurons), while most patients do not have an overt infarction. In people who survive within 12 hours after a cardiac arrest, widespread and pronounced necrosis of neurons is determined under a

microscope. As a result of the selective sensitivity of groups of neurons to hypoxia, necrosis is most pronounced in the hippocampus, the third, fifth and sixth layers of the cerebral cortex (in particular, in the furrows of the posterior halves of both hemispheres), some basal nuclei of pear-shaped neurocytes of the cerebellum (Purkinje cells) [according to MacSween RNM, Whaley K., 1994]. After a few days, the dead neurons disappear and an intense reaction is observed on the part of astrocytes, microglia and capillaries. Similar changes occur with carbon monoxide poisoning, severe forms of epilepsy, and hypoglycemia.

Diseases of peripheral nerves and paraganglia

Degenerative changes in peripheral nerves. There are two types of reactive changes in peripheral nerves in response to damage: axonal degeneration, which includes Wallerian degeneration and develops segmental demyelination as a result of crossing the axon. Often, both types of changes are combined in one patient, but one of them, as a rule, prevails. The basis for their differentiation is a completely different forecast. If the axon remains intact when the pathological process subsides, the restoration of nerve conduction occurs much faster, because lemocytes are capable of rapid remyelination of fibers. However, if the axon is interrupted or crossed, Wallerian degeneration develops. In this case, the axon can grow, adding fibers approximately 1 mm in length from the proximal stump.

Axon degeneration. As a rule, it is the result of severe damage or death of a neuron or its axon and develops rapidly, starting with the breakdown of myelin. Macrophages then "clean" the affected area from particles of myelin and other structures. Lemocytes proliferate, being ready for the formation of a new myelin sheath. The most common cause of such damage is a peripheral nerve injury, and all of the above changes develop distal to the site of damage. Proximal to this place, nerve cell bodies undergo temporary swelling and destruction of the endoplasmic reticulum (chromatolysis). They then repair and support the regeneration of the damaged axon. If the continuity of the endoneural tubes is preserved, the prognosis in terms of the possibility of recovery is not bad, despite even the slow pace of axon regeneration. Many axonal sprouts may not reach the distal stump, but will continue the proliferative process in the dense scar tissue. As a result, thickenings are formed, which were named amputation (traumatic) neuromas. With some lesions of peripheral nerves, there is a tendency to primary damage to the axon itself, and it is more severe at the distal end. In this case, axonal degeneration occurs in the opposite direction in relation to the neuron. All this is called retrograde axon death. Such a process is observed in retrograde neuropathy or distal axonopathy, manifested in glove-stocking anesthesia (loss of sensitivity in the areas of the extremities, conditionally limited by the tissues of gloves and stockings). Finally, axonal degeneration is also secondary to degeneration of the neuron body. but will continue the proliferative process in dense

scar tissue. As a result, thickenings are formed, which were named amputation (traumatic) neuromas. With some lesions of peripheral nerves, there is a tendency to primary damage to the axon itself, and it is more severe at the distal end. In this case, axonal degeneration occurs in the opposite direction in relation to the neuron. All this is called retrograde axon death. Such a process is observed in retrograde neuropathy or distal axonopathy, manifested in glove-stocking anesthesia (loss of sensitivity in the areas of the extremities, conditionally limited by the tissues of gloves and stockings). Finally, axonal degeneration is also secondary to degeneration of the neuron body. but will continue the proliferative process in dense scar tissue. As a result, thickenings are formed, which were named amputation (traumatic) neuromas. With some lesions of peripheral nerves, there is a tendency to primary damage to the axon itself, and it is more severe at the distal end. In this case, axonal degeneration occurs in the opposite direction in relation to the neuron. All this is called retrograde axon death. Such a process is observed in retrograde neuropathy or distal axonopathy, manifested in glovestocking anesthesia (loss of sensitivity in the areas of the extremities, conditionally limited by the tissues of gloves and stockings). Finally, axonal degeneration is also secondary to degeneration of the neuron body. which received the name of amputation (traumatic) neuromas. With some lesions of peripheral nerves, there is a tendency to primary damage to the axon itself, and it is more severe at the distal end. In this case, axonal degeneration occurs in the opposite direction in relation to the neuron. All this is called retrograde axon death. Such a process is observed in retrograde neuropathy or distal axonopathy, manifested in glove-stocking anesthesia (loss of sensitivity in the areas of the extremities, conditionally limited by the tissues of gloves and stockings). Finally, axonal degeneration is also secondary to degeneration of the neuron body. which received the name of amputation (traumatic) neuromas. With some lesions of peripheral nerves, there is a tendency to primary damage to the axon itself, and it is more severe at the distal end. In this case, axonal degeneration occurs in the opposite direction in relation to the neuron. All this is called retrograde axon death. Such a process is observed in retrograde neuropathy or distal axonopathy, manifested in glove-stocking anesthesia (loss of sensitivity in the areas of the extremities, conditionally limited by the tissues of gloves and stockings). Finally, axonal degeneration is also secondary to degeneration of the neuron body. In this case, axonal degeneration occurs in the opposite direction in relation to the neuron. All this is called retrograde axon death. Such a process is observed in retrograde neuropathy or distal axonopathy, manifested in glove-stocking anesthesia (loss of sensitivity in the areas of the extremities, conditionally limited by the tissues of gloves and stockings). Finally, axonal degeneration is also secondary to degeneration of the neuron body. In this case, axonal degeneration occurs in the opposite direction in relation to the neuron. All this is called retrograde axon death. Such a process is observed in retrograde neuropathy or distal axonopathy, manifested in glove-stocking anesthesia (loss of

sensitivity in the areas of the extremities, conditionally limited by the tissues of gloves and stockings). Finally, axonal degeneration is also secondary to degeneration of the neuron body.

Segmental demyelination. It occurs when lemmocytes and myelin sheaths are damaged, but with an intact (undamaged) axon. As a rule, such a process is focal. Since lemocytes can divide, remyelination develops. Demyelinated areas of nerve fibers are replaced by one or more lemocytes, which is accompanied by some shortening of the affected segments of the fibers (between the two nearest interceptions of Ranvier). If the injury is restored or repeated (for example, in hereditary neuropathies), then the impaired repair process turns into lemocyte hyperplasia. Concentric layers of cytoplasmic processes of lemocytes form along the nerve fibers the so-called "onion spheres", which remotely resemble the sensitive nerve cells of Pacini in the dermis.

Peripheral neuropathies. They are accompanied by muscle weakness and atrophy, loss or change of sensitivity, as well as vegetative disorders. During the development of axonal degeneration, fasciculation (involuntary contraction of individual bundles of muscle fibers in the area innervated by the affected nerves) and exhaustion occur. However, in the case of demyelination, these signs are absent, since there is no denervation, but only insufficient conduction. Diseases that cause peripheral neuropathies are acute, developing over several days, subacute (progressing over several weeks), and chronic, progressing over several months or years. A single nerve (mononeuropathy) or several separate nerves (complex mononeuritis) or several symmetrically located nerves (polyneuropathy) can be affected. At the same time, one or both types of degenerative changes occur. To recognize peripheral neuropathies, electroresearch of nerve conduction and morphological study of biopsies are used.

Acute idiopathic inflammatory polyneuropathy (acute ascending polyradiculoneuritis, GuillainBarre syndrome; G.Guillain, MJBarre). This is the most common form of demyelinating peripheral neuropathy, which usually begins 1-2 weeks after an acute stroke. Such polyneuropathy can also occur after immunization, surgery, or as a result of mycoplasma infection, HIV infection, or a malignant process. The disease is characterized by a rapid onset. There is numbness in the areas innervated by the affected nerves, paresthesias (false sensations of tingling, burning, crawling of ants, etc.) and ascending paralysis (which starts from the legs and quickly spreads to the muscles of the trunk, arms, neck, face, tongue , pharynx and larynx). Due to the associated respiratory failure, the patient may urgently need artificial ventilation. Cranial nerves (especially oculomotor) can be involved in the process, and functional disorders of sphincters and cardiac arrhythmias are noted when the autonomic nervous system is affected. Changes in the cerebrospinal fluid, in which increased protein concentrations and a very small number of lymphocytes are found, are quite characteristic. The high content of protein in cerebrospinal fluid is explained by its exudation from inflamed blood vessels located in the area of the spinal roots. Regardless of the localization of the

lesion in the peripheral nervous system, in Guillain-Barre syndrome, there is segmental demyelination with relative preservation of axons, as well as the appearance of endoneural lympho-macrophage infiltrates. Macrophages ensure exfoliation and phagocytosis of myelin surface plates. and when the autonomic nervous system is affected, functional disorders of the sphincters and cardiac arrhythmias are noted. Changes in the cerebrospinal fluid, in which increased protein concentrations and a very small number of lymphocytes are found, are quite characteristic. The high content of protein in cerebrospinal fluid is explained by its exudation from inflamed blood vessels located in the area of the spinal roots. Regardless of the localization of the lesion in the peripheral nervous system, in Guillain-Barre syndrome, there is segmental demyelination with relative preservation of axons, as well as the appearance of endoneural lympho-macrophage infiltrates. Macrophages ensure exfoliation and phagocytosis of myelin surface plates. and when the autonomic nervous system is affected, functional disorders of the sphincters and cardiac arrhythmias are noted. Changes in the cerebrospinal fluid, in which increased protein concentrations and a very small number of lymphocytes are found, are quite characteristic. The high content of protein in cerebrospinal fluid is explained by its exudation from inflamed blood vessels located in the area of the spinal roots. Regardless of the localization of the lesion in the peripheral nervous system, in Guillain-Barre syndrome, there is segmental demyelination with relative preservation of axons, as well as the appearance of endoneural lympho-macrophage infiltrates. Macrophages ensure exfoliation and phagocytosis of myelin surface plates. The high content of protein in cerebrospinal fluid is explained by its exudation from inflamed blood vessels located in the area of the spinal roots. Regardless of the localization of the lesion in the peripheral nervous system, in Guillain-Barre syndrome, there is segmental demyelination with relative preservation of axons, as well as the appearance of endoneural lympho-macrophage infiltrates. Macrophages ensure exfoliation and phagocytosis of myelin surface plates. The high content of protein in cerebrospinal fluid is explained by its exudation from inflamed blood vessels located in the area of the spinal roots. Regardless of the localization of the lesion in the peripheral nervous system, in Guillain-Barre syndrome, there is segmental demyelination with relative preservation of axons, as well as the appearance of endoneural lympho-macrophage infiltrates. Macrophages ensure exfoliation and phagocytosis of myelin surface plates.

After a few weeks (sometimes after 1 week), recovery begins. It can become complete, but sometimes the disease acquires a chronic relapsing or wave-like character. In these cases, "onion balls" can be found in the affected nerves. The causes of the development of acute idiopathic inflammatory polyneuropathy are associated with immunological disorders. In favor of this, the fact that in the early stages of the disease a good therapeutic effect is achieved with the help of plasmaphoresis (taking blood plasma with the return of formed elements into the bloodstream).

Other forms of neuropathies. Diabetic neuropathy (diabetic polyneuropathy) is damage to the nervous system in diabetes, mainly peripheral nerves. Fibers of various thicknesses are affected, including unmyelinated fibers. In the peri- and endoneural capillaries, there is a noticeable narrowing of the lumen, thickening and bifurcation of the basal membrane, which indirectly indicates the possible pathogenetic role of chronic ischemia.

Uremic neuropathy - changes in the central nervous system, caused by the accumulation of toxic products of nitrogen metabolism, prevail. Peripheral nerve damage is transient and secondary (related to changes in the central nervous system) in nature.

Paraneoplastic neuropathy is one of the complications caused by neoplasms. Sometimes the clinical manifestations of the underlying disease begin with it.

Paraproteinemic neuropathy is the content in the blood of monoclonal proteins (immunoglobulins) of any isotype, but more often IgG. Specific IgM-antibodies to myelin-bound glycoprotein were found in affected peripheral nerves [according to MacSween RNM, Whaley K., 1994]. It is shown that their presence is associated with demyelination.

Toxic neuropathy. Many drugs and environmental factors have a harmful effect on nerve tissue, causing peripheral neuropathies. These include furadonin, diaphenylsulfone, dierenyl hydantogen, etc. A frequent cause of toxic neuropathy is chronic alcoholism. Alcoholic neuropathy can occur with severe degeneration of myelin sheaths. Among the chemical and non-medicinal agents, it is worth mentioning lead, arsenic and hexacarbons.

Hereditary motor (motor) and sensitive (sensory) neuropathies. These are rare diseases in which there is selective damage to lower motor neurons (Werdnig-Hoffmann atrophy; G. Werdnig, J. Hoffmann; infantile spinal muscular atrophy) or primary sensory neurons (hereditary sensory neuropathy). There are two more diseases in this group. In Charcot-Marie-Tooth disease (JMCharcot, P.Marie, HHTooth) demyelination with "onion balls" is often found. Dejerine-Sotta disease (JJDejerine, J. Sotta) is characterized by axonal degeneration.

1. Theoretical questions Questions for self-control:

1. CNS diseases, classification.
2. To characterize the etiology, pathogenesis and morphological essence of dystrophic diseases of the central nervous system.
3. To characterize the etiology, pathogenesis and morphological essence of demyelinating diseases of the central nervous system.
4. To characterize the etiology, pathogenesis and morphological essence of inflammatory diseases of the central nervous system.
5. To characterize the etiology, pathogenesis and morphological essence of CNS tumors.
6. The main causes of death in the above-mentioned diseases.

2 Practical tasks

12. Prepare an essay on the topic: "Cellular mechanisms of damage to nervous tissue"

13. Make a graphological structure of "Diseases of the CNS".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory http://www.webpathology.com/- Web Pathology

Topic #6: "Parathyroid osteodystrophy, osteomyelitis, fibrous dysplasia, osteopetrosis, Paget's disease, muscular dystrophies. Myasthenia.»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:

The student should know:

24. Classification and essence of changes associated with diseases of the musculoskeletal system.

25. Etiology, pathogenesis, pathological anatomy of these pathological conditions.

26. Outcomes, complications of diseases associated with diseases of the musculoskeletal system.

The student should know: The student should be able to:

25. Classify diseases of the musculoskeletal system.
26. To characterize the etiology, pathogenesis and morphological essence of these diseases

Topic content:

Diseases of the musculoskeletal system

Diseases of the bone system

The disease of this system can be caused by:

1. Dystrophic: toxic (Urov disease), alimentary (rickets), endocrine, nephrogenic. A significant place belongs to parathyroid osteodystrophy.
2. Incendiary
3. Dysplastic: fibrous dysplasia of bones, osteopetrosis, Paget's disease.
4. Neoplastic - often develop against the background of dysplastic.

Parathyroid osteodystrophy

Parathyroid osteodystrophy (Recklinghausen's disease, generalized osteodystrophy) is a disease caused by hyperfunction of the parathyroid glands and accompanied by generalized damage to the skeleton. It occurs mainly in women aged 40-50.

Etiology. Parathyroid osteodystrophy is a consequence of primary hyperparathyroidism caused by adenoma of parathyroid glands or hyperplasia of gland cells.

Pathogenesis. Increased parathyroid hormone synthesis causes hypercalcemia with progressive demineralization of the entire skeleton. In the bone tissue, osteoclasts are activated, diffuse fibroosteoclasy increases - bone tissue is replaced by fibrous connective tissue. Bone deformation, osteoporosis, pathological fractures are possible. Formations resembling giant cell tumors appear in the changed cells. They are reactive structures that are built by giant cell granulomas.

Hypercalcemia leads to the development of calcareous metastases. Nephrocalcinosis often develops.

Pathological anatomy. Adenoma is often found in the parathyroid glands, cell hyperplasia is less common. Morphological changes of the skeleton depend on the stage and course of the disease. In the initial stage, they are completely absent, then they find deformation of the bones, especially the limbs, spine, ribs. They become soft, easily cut with a knife.

During microscopic examination, foci of lacunar resorption, neoplasms of fibrous tissue are found in bone tissue, giant cell granulomas, accumulation of erythrocytes and hemosiderin are possible in tumor-like formations.

The death of patients occurs from cachexia or uremia due to shrinkage of the kidneys.

Osteomyelitis

Osteomyelitis is an inflammation of the bone marrow, which spreads to the spongy and compact substance of the bone and to the periosteum. According to the course, acute and chronic osteomyelitis are distinguished, according to the mechanism of infection - primary and secondary.

Primary hematogenous osteomyelitis

Acute hematogenous osteomyelitis is most common in young people. Chronic osteomyelitis is a consequence of acute.

Etiology. The causative agents of acute osteomyelitis are mostly purulent microbes: hemolytic staphylococcus, streptococcus, coliform bacilli, pneumococci, gonococci. Most often, patients with osteomyelitis have bacteremia with minor intestinal trauma, dental disease, and upper respiratory tract infection.

Pathogenesis. The purulent inflammatory process begins in the bone-marrow crevices of the metaphyses, where there is slowed blood circulation. Further, the process spreads to the bone marrow, where necrosis appears, and passes to the cortical layer of the bone, periosteum, and adjacent soft tissues.

Pathological anatomy. In acute hematogenous osteomyelitis, the inflammation has a phlegmonous nature. Resorption of bone tissue near the epiphyseal cartilage can end with the separation of the metaphysis from the epiphysis (epiphyolysis). Tissue infiltration by neutrophils appears around necroses; thrombi are found in the vessels of the compact plate. Abscesses often develop under the periosteum.

Chronic hematogenous osteomyelitis, as a result of acute, is accompanied by the formation of sequestrations, around which granulation tissue and a capsule are formed. From the sequestrations, fistulas go to the surface of the skin or to the body cavity. Along with the destruction of the bone in the periosteum and bone marrow canal, bone formation occurs - the bones become thick and deformed. Scars form in soft tissues.

Complications of primary hematogenous osteomyelitis: bleeding from fistulas, spontaneous fractures, formation of false joints, development of sepsis, secondary amyloidosis in chronic osteomyelitis.

Fibrous dysplasia

Fibrous dysplasia (fibrous osteodysplasia, Lichenschein-Breitsev disease) is a disease in which bone tissue is replaced by fibrous tissue, which leads to bone deformation.

Etiology and pathogenesis. The reasons for the development of the disease are unknown, perhaps hereditary factors are of some importance. It is believed that the tumor process is at the root of the disease. The disease begins in childhood, but can also develop in adults.

Classification. Depending on the prevalence of the pathological process, two forms of fibrous dysplasia are distinguished:

1. Monoosseous - pathological changes occur in only one bone. It can develop at any age.

2. Polyosseous - several bones are affected, mostly on one side of the body. Sometimes it is combined with melanosis of the skin. It develops in childhood.

Pathological anatomy. With the monoaxial form, pathological changes most often develop in the ribs, long tubular bones, shoulder blades, skull bones; with poliomyelitis - more than 50% of the bones of the skeleton, mainly on one side of the body. The damaged bone at the beginning of the disease retains its shape and size. In the future, "swelling", deformations of the bone, its lengthening or shortening appear. Femurs acquire the shape of a "shepherd's staff". On the cutting, clearly limited areas of whitish color with black and brown inclusions are determined. The bone marrow canal is expanded or filled with newly formed tissue.

Under microscopic examination, the centers of fibrous dysplasia are represented by fibrous fibrous tissue, which in some areas consists of randomly arranged bundles of mature collagen fibers and spindle-shaped cells, and in others - of thin collagen fibers and stellate cells. If fibrous dysplasia affects the bones of the face, then the dense component in the cells may be represented by cementtype tissue (cementicle-like formations).

Complications are represented by pathological bone fractures, especially often in children, the femur is broken. A sarcoma may develop.

Osteopetrosis

Osteopetrosis (marble disease, congenital osteosclerosis, Albers-Schönberg disease) is a rare hereditary disease in which generalized excessive bone formation is observed, which leads to bone thickening, narrowing, and even complete disappearance of bone-marrow cavities. Osteopetrosis is characterized by a triad: increased bone density, bone fragility, and anemia.

Etiology and pathogenesis. Undoubted participation of hereditary factors, which are associated with a violation of the development of bone and hematopoietic tissue. The development of anemia, thrombocytopenia, the appearance of extraosseous hematopoietic centers in the liver, spleen, and lymph nodes is associated with the growing squeezing of bone marrow by the bone.

Classification. There are two forms of osteopetrosis:

1. Early (autosomal recessive) - appears at an early age, proceeds malignantly, often ends fatally.

2. Late (autosomal dominant) - a more benign course.

Pathological anatomy. The whole skeleton can be affected, but especially tubular bones, bones of the base of the skull, pelvis, spine, ribs. In the early form, the face acquires a characteristic appearance: it is wide, with widely spaced eyes, the root of the nose is depressed, and the lips are thick. With this form, hydrocephalus, increased hair growth, hemorrhagic diathesis, and multiple bone lesions are noted.

Characteristic column-shaped expansion of the lower femurs. On cuts in long bones, the medullary canal is filled with bone tissue and is often not identified. The spongy substance resembles polished marble.

The microscopic picture is peculiar: pathological ossification occurs throughout the entire bone, the bone substance is randomly accumulated in the internal parts of the bones. Osteoclasts are single, signs of bone resorption are insignificant. Bone architecture loses its functional characteristics. At the base of the cartilage, peculiar round islands of bone beams are formed.

Complications: bone fractures, especially femoral fractures, purulent osteomyelitis.

Causes of death. Patients often die in early childhood from anemia, pneumonia, sepsis.

Paget's disease

Paget's disease (deforming ostosis, deforming osteodystrophy) is a disease characterized by increased pathological remodeling of bone tissue, continuous changes in the processes of bone resorption and new formation, while the bone tissue acquires a peculiar mosaic structure. It is observed more often among men older than 40 years, progresses slowly, becomes noticeable only in old age. The lesion may involve a single bone (mono-osseous form) or several often paired or regional bones (poly-osseous form), but is never generalized.

Etiology. The reasons are unknown, the family nature of the disease is emphasized.

Patho- and morphogenesis. Bone tissue reconstruction processes are continuous, there is no connection with functional load. There are three phases of the disease:

1. Initial (osteolytic) - the processes of bone resorption with the participation of osteoclasts prevail, deep gaps are formed in the bone tissue.
2. Active (combination of osteolysis and osteogenesis) - osteoblasts appear, lacunae are filled with newly formed bone substance. The bone beams are built from small fragments forming a characteristic mosaic.
3. Inactive - the process of osteosclerosis prevails.

Pathological anatomy. Long tubular bones, especially the femur and tibia, are covered, sometimes spiral-shaped, which is due to the growth of the bone during its reconstruction. A narrow medullary channel is revealed on the cuttings. When the periosteum is removed, there are numerous small openings of vascular channels on the surface of the cortical layer. On cutting, the cortical layer loses its compact structure and becomes almost spongy.

When the bones of the skull are damaged, only the bones of the brain skull are involved in the pathological process. The entire bone mass has an uneven spongy structure with pockets of rarefaction and compaction.

In the spine, the process involves one or more vertebrae in different parts of it, but never affects the entire spine. The vertebrae increase in volume or, on the contrary, flatten, depending on the stage of the disease. Focal points of osteoporosis and osteosclerosis are found on bone cuts.

Microscopic examination: determine small fragments of bone structures with uneven contours, with wide, well-defined basophilic adhesion lines. The areas of the bone fragments of the mosaic are usually calcified, their structure is irregular, thin-fibrous or lamellar. A large number of osteoblasts, axillary resorption cavities are found in the deep lacunae of bone structures. Signs of a bone neoplasm are noted: expanded bone cavities are filled with delicate fibrous tissue.

Complications: hemodynamic disorders (related to the expansion of blood vessels in the affected bone tissue), pathological fractures (develop in the active phase), osteogenic sarcoma (in 1-10% of patients, is localized in the thigh, lower leg, pelvic bones, in the scapula).

Diseases of the joints

Joint diseases can be associated with dystrophic processes of the structural elements of the joints (arthrosis) or their inflammation (arthritis). Among arthrosis, osteoarthrosis occupies a significant place, and among arthritis - rheumatoid arthritis.

Osteoarthritis

Osteoarthrosis is one of the most frequent diseases of joints of a dystrophic nature. Elderly women suffer more often. Osteoarthritis is divided into primary (idiopathic) and secondary (in other diseases). The pathological process develops in the joints of the lower limbs - hip, ankle.

Etiology and pathogenesis. Hereditary (genetically determined disturbance of metabolism in articular cartilage) and acquired (mechanical trauma) factors are important.

Classification. There are three stages of osteoarthritis:

1. Pain in the joints during exercise, narrowing of the joint space and osteophytes are noted (radiologically).
2. Pain in the joints becomes constant, the narrowing of the joint space and the development of osteophytes are more pronounced.
3. Along with constant pain, functional insufficiency of the joints due to the development of subchondral sclerosis is noted.

Pathological anatomy. Macroscopic changes depend on the stage of the disease. In the early stage, the edges of the articular cartilage appear fibrous, fibrous tissue. In the second stage, patterns and humps are found on the articular surface of the cartilage, bone growths-osteophytes are formed. In the third (late) stage, the articular cartilage disappears, depressions appear on the bones of the joints, and the joints themselves are deformed. The amount of synovial fluid decreases sharply.

Microscopic changes: in the first stage, the cartilage retains its structure, the amount of glycosaminoglycans decreases in its surface and intermediate zones. In the second stage, shallow patterns appear in the surface zone of the cartilage, on the crowns of which chondrocytes accumulate. The pathological process also develops in the subchondral part of the bone. In the third stage, the surface zone and part of the intermediate zone of cartilage die, in the deep zone the number of glycosaminoglycans is sharply reduced, and the number of chondrocytes with pyknotic nuclei is increased.

Diseases of skeletal muscles

Among skeletal muscle diseases, the most widespread are striated muscle diseases of dystrophic (myopathy) and inflammatory (myositis) origin. Progressive muscular dystrophy and myopathy in myasthenia occupy a significant place among myopathies.

Progressive muscular dystrophy

Progressive muscular dystrophy (progressive myopathy) is a variety of primary hereditary chronic diseases of striated muscles. The disease is characterized by growing, often symmetrical, muscle atrophy, accompanied by progressive muscle weakness, almost to complete immobility.

Etiology and pathogenesis are little studied. The significance of abnormalities in structural proteins, sarcoplasmic reticulum, innervation, and enzymatic activity of muscle cells is discussed.

Classification. There are three main forms of progressive muscular dystrophy:

1. Duchenne (early form). The recessive type of inheritance associated with the X-chromosome occurs mainly in children aged 3-5 years. First, the muscles of the pelvic girdle, thighs and lower legs are affected, then the shoulder girdle and trunk.

2. Erba (youthful form). Autosomal dominant type of inheritance, develops during puberty. Changes develop first in the muscles of the chest and shoulder girdle, sometimes in the face (smooth forehead, insufficient closing of the eyes, thick lips).

3. Leiden. Autosomal recessive type of inheritance, begins in childhood or during puberty. It begins in the muscles of the pelvic girdle and hips, gradually covering the muscles of the trunk and limbs.

Pathological anatomy. Muscles are atrophic, thin, depleted of myoglobin, resemble fish meat at autopsy.

Upon microscopic examination, muscle fibers are different in size: along with atrophic ones, there are sharply enlarged (thickened) ones. Pronounced dystrophic changes of muscle fibers, their necrosis and phagocytosis. Adipose tissue accumulates between damaged muscle fibers.

Ultrastructural changes in muscle fibers in Duchenne muscular dystrophy: at the beginning of the disease, expansion of the sarcoplasmic reticulum, foci of destruction of myofibrils, and movement of nuclei to the center of the fiber are found. In the late stage, myofibrils are subject to fragmentation and disorganization, mitochondria swell. At the end of the disease, muscle fibers are compacted, surrounded by a hyaline-like substance.

The death of patients with a severe course of progressive muscular dystrophy occurs from a pulmonary infection.

Myasthenia

Myasthenia gravis is a chronic disease, the main symptom of which is weakness and pathological fatigue of the striated muscles. Normal contraction of muscles after their active activity decreases in strength and volume and may stop completely. Muscle rest time becomes longer in the late stage of the disease. Eye muscles (ptosis), masticatory, speech and swallowing muscles are most often affected. The disease occurs at any age, in women 3 times more often than in men.

Etiology and pathogenesis. The etiology is unknown. Correlation between thymus abnormalities and myasthenia occupies a significant place in pathogenesis. The development of the disease is associated with a decrease of up to 90% in the number of acetylcholine receptors per unit of muscle plate, which is associated with autosomal reactions.

Pathological anatomy. In patients, follicular hyperplasia or thymoma is often found in the thymus. Skeletal muscles are slightly changed or in a state of dystrophy, sometimes accumulation of lymphocytes among muscle cells is revealed. IgG and C3 are also detected in postsynaptic membranes. Lymphoid infiltrates are found in the liver, thyroid gland, and other organs.

Complications arise when the respiratory muscles are damaged. An inadequate response of the lungs leads to the development of pneumonia and asphyxia, which, as a rule, become the cause of death.

1. Theoretical questions Questions for self-control:

14. Describe the macro- and microscopic changes in the human body during parathyroid dystrophy,
15. Describe the macro- and microscopic changes in the human body during primary hematogenous osteomyelitis,
16. Describe the macro- and microscopic changes in the human body during fibrous dysplasia,
17. Describe the macro- and microscopic changes in the human body during osteopetrosis,
18. Describe the macro- and microscopic changes in the human body during Paget's disease,
19. Describe the macro- and microscopic changes in the human body during osteoarthritis,
20. Describe the macro- and microscopic changes in the human body during progressive muscular dystrophy,

21. Describe the macro- and microscopic changes in the human body during myasthenia.

2 Practical tasks

On the basis of autopsy protocols, to investigate the frequency of occurrence of diseases of the musculoskeletal system over the past 5 years according to archival data of OOKL

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
- http://www.webpathology.com/- Web Pathology

Topic #7: "Pathology of pregnancy, postpartum period and placenta. Breast disease.»

Purpose: as a result of independent study of this topic, students should know the topic for studying the characteristics of tumor growth in children at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations.

Basic concepts:
The student should know:

27. classification of diseases of pregnancy, postpartum period;

28. classification of litter pathology;

29. principles of diagnosis of pathology of pregnancy, postpartum period and litter;

30. characteristic morphological features of any of the forms of the aforementioned pathology.

The student should know: The student should be able to:

27. to recognize certain types of the aforementioned pathology during macro- and microscopic examination;
28. evaluate the results of the conducted research and describe them in the study album;
29. predict possible complications. **Topic content:**

DISEASES OF PREGNANCY AND THE POSTPARTUM PERIOD

Neurohumoral changes occurring during pregnancy can lead to a violation of its normal development, which creates prerequisites for the occurrence of pregnancy pathology. Pregnancy pathology includes: 1) gestosis (pregnant toxicosis), 2) ectopic pregnancy, 3) spontaneous abortion; 4) premature birth; 5) chorioadenoma. After childbirth or abortion, placental polyps, chorionepithelioma, and congenital infection of the uterus develop.
Gestosis (from the Latin gesto - to carry, to be pregnant), or toxicosis of pregnant women - a group concept that unites dropsy of pregnant women, nephropathy, preeclampsia and eclampsia. E t i pro l pro g i ia and p a t pro g e n e z. The causes of preeclampsia have not been established. Among the numerous theories of pathogenesis (renal, hormonal, coagulation, neurogenic, etc.), the most evident is the immune theory, which is based on the weakening of the mother's immune recognition of fetal antigens in the event of a violation of the bar specific properties of the placenta. Insufficient immune recognition of fetal antigens by the mother, as well as insufficient production of suppressive factors (T-suppressors, blocking antibodies, etc.), are associated with the relative homozygosity of the pregnant woman, husband and fetus for D-antigens of histocompatibility. The lack of suppressive factors leads to the development of immune cell and immune complex reactions. Immune complexes appear not only in the blood of pregnant women, but also in the vessels of the placenta, the changes of which resemble the reaction of transplant rejection. Damage to a number of internal organs, in particular the kidneys (nephropathy of pregnant women), is associated with immunocomplex reactions in preeclampsia. The sensitivity to angiotensin increases sharply, which leads to widespread angiospasm and arterial hypertension.
A major role in the pathogenesis of preeclampsia is played by blood coagulation disorders, which are largely associated with the release of thromboplastin by the placenta. The syndrome of disseminated intravascular coagulation (DVS syndrome) develops, which is especially pronounced in eclampsia.

Among the manifestations of toxicosis of pregnant women, eclampsia is the most clinically significant and dangerous, it develops in the second half of pregnancy (late toxicosis of pregnant women), less often - during childbirth and the postpartum period.

Pathological anatomy of eclampsia. The changes are represented by disseminated thrombosis of small vessels, numerous small necrosis and hemorrhages in internal organs. At autopsy, edema, jaundice, pronounced changes in the brain, lungs, heart, liver, and kidneys are found. In g pro l pro v n pro mm pro z g e find edema, thrombi in small vessels, hemorrhage, more frequent in subcortical nuclei, in l e g to i x- edema and draining hemorrhagic pneumonia, in z e r d c e - blood clots in blood vessels, focal necrosis of the myocardium and hemorrhage. The liver is enlarged, variegated, with numerous hemorrhages. Microscopic examination reveals thrombi in small vessels, hemorrhage, and foci of necrosis. P pro ch to and are enlarged, flaccid, their cortical layer is swollen, variegated, medulla - sharply full-blooded. Sometimes they find - symmetric necrosis of the cortical substance of the kidneys.

Death occurs from liver or kidney failure, as well as from DIC and hemorrhage to vital organs.

Ectopic pregnancy is the development of the fetus outside the uterine cavity: in the fallopian tube (tubal pregnancy), in the uterus (ovarian pregnancy) or in the abdominal cavity (peritoneal pregnancy). Tubal pregnancy is the most common. The development of an ectopic pregnancy is associated with those changes in the fallopian tubes that prevent the progress of a fertilized egg through them (chronic inflammation, congenital anomalies, tumors, etc.).

Tubal pregnancy, as a rule, is observed in one tube. If the egg is attached and develops in the ventral end of the tube, it is called an ampullary and tubal pregnancy, if it is in the uterine end of the tube (isthmus area), it is an intrauterine pregnancy. about tubal pregnancy. As the embryo grows, the egg can break the tube and become embedded between the leaves of the broad ligament, then an ectopic interligamentary pregnancy occurs.

During a tubal pregnancy, a decidual reaction develops in the mucous membrane of the tube, where the egg is attached and formed, which is characterized by the appearance of large and light-colored decidual cells both in the mucous membrane and in the wall of the tube. In the mucous membrane, a perforation also appears, and the villi of the chorion penetrate the muscle layer and its vessels, destroying the tissue elements of the tube (Fig. 239). In connection with this, in the first months of tubal pregnancy, bleeding into the tubal cavity and the release of the fetus into the tubal cavity are possible - incomplete tubal abortion. The dead fetus and its blood-soaked membranes are ejected through the fimbrial end into the abdominal cavity - a complete tubal abortion.

A rupture of the tube wall and bleeding into the abdominal cavity are possible, which can lead to the death of a woman. When the tube ruptures, the dead fetus can end up in the abdominal cavity, where it dies and mummifies ("paper fetus") or calcifies (lithopedion); secondary abdominal pregnancy rarely develops.

During the operation to remove the tube with the fetal egg, the basis for the diagnosis of ectopic pregnancy is the detection of chorionic villi and decidual cells, not to mention the elements of the fetus. A decidual reaction is also found in the mucous membrane of the uterus (nipples).

Spontaneous abortion and premature birth. They are termination of pregnancy; which happens at different times. Termination of pregnancy and expulsion of the fetus from the uterus earlier than 14 weeks from the moment of conception is marked as an abortion (miscarriage), from 14 to 28 weeks - as a late abortion, from 28 to 29 weeks - premature birth.

In an involuntary abortion, the entire fetal egg (fetus and membranes) is thrown out of the uterus, which can be stored or damaged, with blood clotting. With premature birth, the fetus is first born, and then the shell with a baby pestle. During the histological examination of fragments of the fetal egg, which were released independently or removed with an abrasive (scraping of the uterine cavity), the membranes of the fetus, chorionic villi and decidual tissue are revealed. Abortion often occurs when the fetus dies as a result of incomplete introduction of the fetal egg into the mucous membrane of the uterus, failure of the mucous membrane itself, in the presence of hemorrhoids; tumors, etc.

An artificial abortion is performed according to medical indications in a medical institution. Abortion carried out in unsanitary conditions, outside a medical hospital, may cause infection of the uterus, development of sepsis; it may be subject to legal proceedings (criminal abortion).
Trophoblastic disease. Trophoblastic disease is a group concept. It includes cystic fibrosis, invasive cystic fibrosis, choriocarcinoma, and trophoblastic tumor of the placental bed. Placenta tissue is the source of the disease. Trophoblastic disease is relatively rare. Thus, for every 1,000 births there is 1 case of cyst insertion, for every 100,000 births or abortions - 2 cases of choriocarcinoma. Compared to Europe, the frequency of trophoblastic disease is much higher in Asian and African countries. Differences in morbidity may be of a racial nature, or may be due to a large number of deliveries and the age of pregnant women (it has been established that the frequency of trophoblastic disease is increased in pregnant women younger than 16 and older than 35 years). The share of choriocarcinoma among malignant neoplasms of female genital organs is only 2.1%.
Chorioadenoma is manifested by vaginal bleeding in the 1st trimester, which may be accompanied by the discharge of vesicular villi. and at the same time, an increase in the size of the uterus and an unusually high level of chorionic gonadotropin are observed. With vesicular drift, cluster-like clusters consisting of numerous bubbles filled with a clear liquid are microscopically visible. Bubbles can be freely located in the uterine cavity and be released from the vagina.
Microscopically, a sharp swelling of the villi is revealed, often with the formation of cavities (cisterns) filled with liquid in the center of the villi. The degree of trophoblast proliferation can be different. With complete cystic drift, the entire placenta is affected; the fruit is usually absent. With partial cystic drift, as a rule, there is no noticeable increase in the volume of the placenta, vesicular villi are distributed among morphologically normal placental tissue. There is usually fruit, but it dies early. Complete vesicular drift. With this type of insertion, there is a diploid set of chromosomes, all of parental origin. It is assumed that the chromosomal pressure of the sperm doubles, and the nucleus of the egg is inactivated or dies. Dispermic fertilization is less often observed. In partial cystic drift, the karyotype is triploid, and the additional, third set of chromosomes is of parental origin. If an additional set of chromosomes is of maternal origin, hydropic transformation of the villi does not develop. Thus, the cystic transformation of the placental villi with the formation of a vesicular drift is caused by the predominance of parental chromosomes in the karyotype of the embryo.
After the removal of cystic tissue, a woman recovers most often, but the possibility of progression of the disease is quite high. The risk of developing choriocarcinoma after a complete cystic drift is about 5%. The frequency of choriocarcinoma after partial entrainment has not been established, but it is known to be significantly lower than in complete entrainment.
Invasive vesicular drift is characterized by the growth of villi in the myometrium. Clinically, it is manifested by bleeding, which occurs several weeks after the removal of the drift. Hemorrhagic foci of various sizes are determined microscopically in the myometrium. The liquid tissue of the trophoblast penetrates the entire wall of the uterus and spreads to the adjacent organs.
Microscopically, swollen villi are found in the myometrium, more often in vessels. The degree of trophoblast proliferation can be different. The invasive nature of the drift is not considered a sign of true neoplasia. A normal trophoblast has the capacity for invasive growth, and the villi of a normal placenta can penetrate deep into the myometrium. However, with invasive cystic drift, metastases can be observed, more often in the lungs and vagina. These metastases regress spontaneously or after a single course of chemotherapy.
Choriocarcinoma. This is a malignant tumor of the trophoblastic epithelium. About 50% of such neoplasms develop after a pregnancy complicated by insertion of a bladder, 25% - after an abortion, 2.5% - after an ectopic pregnancy, and 22.5% - after a clinically normal pregnancy.
Choriocarcinoma can occur immediately after termination of pregnancy, after a few weeks and even after 15-20 years. The most characteristic symptom is vaginal bleeding. Relatively often, the disease is manifested by signs caused by metastases. The development of pulmonary hypertension associated with the growth of metastatic nodes in the pulmonary arteries is possible.
Choriocarcinoma is hormonally active because the trophoblast synthesizes chorionic ronadotropin.

in connection therefore, regardless of the size of the primary cyst, an increase in the uterus and thickening of its mucous membrane with a pronounced decidual reaction are always noted. Choriocarcinoma is one of the most malignant tumors, but it is well treated with a combination of hysterectomy and chemotherapy. Exceptions are cases when the tumor develops after a normal pregnancy. In these cases, the prognosis is extremely unfavorable.
Choriocarcinoma has the appearance of a juicy yellowish-white or variegated spongy node on a wide base. When located under the mucous or serous membranes, the node can shine through in the form of a dark cherry formation. Microscopically, choriocarcinoma consists of cytotrophoblast cells and polymorphic giant syncytiotrophoblast elements. There are never true villi in the tumor. The degree of atypism and mitotic activity in tumor cells varies significantly. Using immunohistochemical methods, chorionic gonadotropin can be found in these cells. Stroma and vessels" are absent in the tumor. The rapid growth of the tumor is accompanied by multiple foci of necrosis and hemorrhages. Choriocarcinoma is characterized by extensive early hematogenous metastases in the lungs (80%), vagina (30%), brain, liver, and kidneys.
Trophoblastic tumor of the placental bed is rare. Usually, this neoplasm develops after a normal pregnancy, however, in the anamnesis of sick women, a high incidence of cyst insertion is noted. The uterus is enlarged, whitish-yellow or yellowish-brown masses are visible in the myometrium, exploding into the cavity in the form of polyps. Microscopically, the tumor consists mainly of mononuclear cells of the intermediate trophoblast with an admixture of multinucleated cells that resemble multinucleated cells of the placental bed. Cells form islands and rods penetrating between muscle fibers. Hemorrhage and necrosis are not characteristic. Tumor cells secrete placental lactogen, chorionic gonadotropin is found in only a small part of them. The result of the disease is more often favorable. A malignant course with metastases is observed in 10-20% of patients. Unlike choriocarcinoma, the cells of the trophoblastic tumor of the placental bed are insensitive to chemotherapy. The main treatment is surgical.
A placental polyp is formed in the mucous membrane of the uterus at the site of parts of the droppings that have remained in it after childbirth or abortion. A polyp consists of villi, coils of fibrin, decidual tissue, which are subject to organization; a connective tissue area appears in the uterus. Placental polyp interferes with the postpartum involution of the uterus, supports inflammation in the mucous membrane and is the cause of bleeding.
Obstetric infection of the uterus is a very dangerous complication of the postpartum period, and streptococcus, staphylococcus, and Escherichia coli are the most important pathogens. Infection of the uterus leads to purulent endometritis, which can occur during or after childbirth. Obstetric infection occurs exogenously (non-compliance with the rules of asepsis) or endogenously (an outbreak of an earlier infection during childbirth). In the most severe cases, endometritis can become septic. The inner surface of the uterus becomes dirty gray, covered with purulent plaque. The infection spreads along the course of lymphatic vessels and veins (lymphogenic and hematogenous), lymphangitis, phlebitis and thrombophlebitis develop. Endometritis is joined by metritis and perimetritis, which leads to peritonitis. As a result of this, the uterus turns into a septic focus, which determines the one that generalizes infections.

PATHOLOGY OF THE FEED

The litter, consisting of the placenta, fetal membranes and umbilical cord, is an important intermediate element of the mother-fetus functional system. Its main role consists in timely and adequate supply of constantly growing needs of the fetus.

Violation of implantation and placentation processes

Malformations of the shape of the placenta. The main changes in shape, which negatively affect the fetus, the course of pregnancy and childbirth, include the placenta, surrounded by a shaft and surrounded by a rim. The process in terms of the nature of the changes is unambiguous, but it is expressed more sharply in the case of a roller-shaped placenta. It is a consequence of detachment

and twisting of the edges of the placenta in the early stages of pregnancy. Microscopically, the shaft consists of necrotized villi and decidual tissue impregnated with fibrinoid, which gradually undergo hyalinosis. With a roller-shaped placenta during pregnancy, bleeding is observed, premature births and the birth of a dead fetus are more common. Terminal placenta, two-lobed, multipartite placenta and with additional lobes do not have serious thanatogenetic significance, but are indirect signs of a violation at the stage of implantation and placentation.

II years of development of placental localization. These defects include marginal or central placenta previa in relation to the internal uterine cavity. Placenta previa occurs as a result of implantation of the blastocyst in the lower segment of the uterus. The reasons for such implantation are unclear, it is more common in multiple pregnancies and in multiparous women. It is registered in approximately 0.25-0.5% of all births, accompanied by a high level of fetal and newborn mortality (17-19%). The main danger is premature detachment of such a placenta, massive bleeding and death of the fetus, or severe intracranial trauma of the newborn during emergency extraction through an insufficiently dilated cervix. The placenta often has an irregular shape, flattened, rounded or with additional lobes. Defects of detachment of the placenta. Placenta growth is manifested by ingrowth of chorionic villi into the myometrium, difficulty in its separation and/or massive uterine bleeding, which sometimes requires extirpation of the uterus. The defect occurs as a result of insufficient development of the basal layer of the shell in the area of implantation of the ovum. Insufficient development of decidual tissue can be associated with endometritis, repeated scrapings of the uterine cavity, etc. Premature exfoliation. Detachment of the placenta, which occurs before the birth of the fetus, is called premature. Premature detachment can occur with defects in the development of the location of the placenta and a normally located placenta. It can also be a consequence of nephritis, hypertensive disease of pregnant women or abdominal trauma, short umbilical cord, late opening of the amniotic sac, rapid outflow of amniotic fluid with polyhydramnios.

Blood circulation disorders of the placenta

Diffuse ischemia of the placenta is observed in hemolytic disease in combination with edema, in posthemorrhagic conditions, as postmortem changes in connection with intrauterine death of the fetus. The decline of the capillaries of the terminal villi, the formation of syncytial kidneys is revealed. Diffuse hyperemia is observed in hypoxic conditions of the mother (diseases of the cardiovascular system), in the case of difficulties in the outflow of blood through the umbilical vein - wrapped umbilical cord, true nodes of the umbilical cord, etc. Bleeding can be from the maternal part of the placenta in case of presentation or premature separation of the placenta and from the fetal part in the form of hemorrhaging in the stroma of the villi in nephropathy, infectious diseases of the mother, and in the amniotic fluid - hemamnion in the case of rupture of the vessels of the umbilical cord.

Edema of the placenta is observed in hemolytic disease, infectious processes, diabetes and nephropathy of the mother. The maternal surface of the placenta is pale, its mass is increased. Swelling of the stroma of the villi is accompanied by an increase in their volume by 2-3 times, in all such cases, the combination with immaturity of the villous tree is emphasized, therefore, the swelling of the villi should be differentiated from the presence of embryonic and intermediate immature villi with characteristic stromal channels and Kashchenko-Hoffbauer cells.

Thrombosis of the intervillous space occurs with physiological aging of the placenta, with toxicosis of pregnant women, with infectious diseases of the mother. It is important to determine the age of occurrence of blood clots: fresh or old, with hemolysis of erythrocytes, fibrin deposition. Microscopically, this is the so-called red infarction of the placenta.

A heart attack is a focus of necrosis of the villi, arising as a result of a violation of nutrition in honest disorders of the maternal blood flow, in particular in the spiral arteries of the uterus. Infarcts in the form of whitish-yellowish foci occur in small numbers with physiological aging of the placenta, large in volume - with diseases of the mother, which leads to vascular spasms, thrombosis (hypertensive disease, severe toxicosis of pregnancy, diabetes, etc.). Microscopically, complexes of necrotized villi surrounded by coagulated blood can be seen. A diagnostic sign of a heart attack that

has developed a long time ago can be considered the presence of syncytial kidney clusters, calcifications, and fibrinoid on the periphery of the necrotic zone. The volume of distribution of white infarcts is of great importance in the assessment of placental insufficiency. If it occupies more than 20-30% of the placental parenchyma,

Classification of placental insufficiency

The concept of "placental insufficiency (PN) or dysfunction" is interpreted in the literature inconsistently. Thus, E. Hovorka (1970) proposed to distinguish three types of placental insufficiency, depending on the pathogenesis of hypotrophy of newborns:

1) the placenta in the case of primary deficiency of the mass of the newborn, in cases of disorders of the utero-placental blood circulation that are detected early, in chronic diseases of the mother (hypertensive disease, nephritis, etc.) with characteristic chronic heart attacks, intervillous thrombi in the hypoplastic organ;
2) placenta with secondary deficiency of body weight of newborns - with late-onset blood supply disorders, most often in cases of carrying pregnancy;
3) placenta in undifferentiated forms of newborn body weight deficiency, when signs of primary and secondary newborn body weight deficiency are detected at the same time in the absence of hypertension, nephropathy and ongoing pregnancy.

K1oos, M. Voge1 (1974), and then W. Hopker, B. Ohlendorf (1979) defined placental insufficiency as the inability of the placenta to maintain adequate exchange between the mother and the fetus and distinguished acute, subacute, chronic respiratory and chronic metabolic forms.

Acute placental insufficiency is characterized by placental dysfunction that develops over several hours as a result of extensive hemorrhage or partial detachment. Histologically, retroplacental hematoma with collapse of the intervillous space, reactive hyperemia of fetal vessels, destruction of the epithelial covering of the villi against the background of immaturity of the villous tree, often of the type of chorioangiomatosis are determined. Intrauterine death or acute asphyxia of the fetus occurs most often.

Subacute placental insufficiency develops over several days, causing intermittent placental dysfunction. By the nature of the lesion, this form is close to the previous one, but the areas of hemorrhage are small, they are characterized by thrombi in the intervillous spaces of various ages. Violations of uteroplacental blood circulation are emphasized in the presence of immaturity of the villi, but detachment of the placenta does not occur. Intrauterine hypoxia and hypotrophy of the fetus develops.

Chronic respiratory placental insufficiency is characterized by disturbances in the diffusion of gases at the level of the placental barrier for weeks as a result, mainly, of the pathological immaturity of the villi, without pronounced disturbances of blood circulation in the placenta. Microscopically, small foci of necrosis, immature villi without syncytiocapillary membranes and syncytial kidneys are visible. A latent form of hypoxia develops in the fetus.

Chronic metabolic placental insufficiency is a long-term (months) impairment of placental function with a compensatory increase in its mass, pathological immaturity of its villi, diffuse sclerosis of their stroma, hemorrhages, and extensive heart attacks. Depending on the volume of the placenta lesion, intrauterine hypotrophy and hypoxia develop, or fetal death occurs.

In our country, E. P. Kalashnikova (1981, 1986) singles out primary and secondary placental insufficiency, taking into account the duration of action of pathological factors during pregnancy. Primary placental insufficiency occurs during the period of egg implantation, placentation and early embryogenesis. Disturbances in the development of the mass, the shape, location, maturation, and vascularization, which are revealed in this case, lead to placental insufficiency, threatening termination of pregnancy, and death of the fetus during the first half of pregnancy.

Secondary placental insufficiency develops when the placenta has already formed as an organ. There are two forms of this deficiency: acute (disruption of maternal-placental blood circulation, hemorrhage, extensive heart attacks, etc.) and chronic, which occurs in late toxicosis, foci of latent infection, cardiovascular and renal diseases of the mother, etc. Under the influence of pathogenic

factors on the immature placenta, the imperfection of compensatory reactions causes absolute placental insufficiency and intrauterine death of the fetus.

Pathology of the placenta in various diseases of the mother

Placenta in late toxicosis of pregnant women. The complex pathogenesis of toxicosis in pregnant women causes a variety of changes in the placenta. Among them, it is advisable to distinguish villus maturation disorders, common hemorrhagic heart attacks, immune disorders and compensatory and adaptive processes.

According to Z. P. Zhemkova, Pro. I. Topchieva (1973), out of 138 placentas of full-term newborns from mothers with late toxicosis of pregnancy (without other pathology), in 11.3 cases placental pathology of the type of maturita retardata and dissociated maturation disorder was found. In 43.9% of full-term fetuses that died in the antenatal period, similar ripening disorders were observed. A constant sign in all forms and degrees of pathological immaturity of the placenta is the insufficient development of villi vessels, which indicates the early manifestations of the disease, which later manifest as late toxicosis of pregnant women. The same characteristic signs of toxicosis are multiple and widespread placental infarcts of various ages: the earlier the toxicosis develops and the more severe it is, the greater the number of chronic infarcts in the placenta

Placenta in hypertensive disease and chronic nephritis of pregnant women. The commonality of pathogenetic mechanisms of ethical diseases with toxicosis of pregnant women also explains the undeniable similarity of histological changes in the placenta. Therefore, some authors consider it impossible to differentiate the pathology of the placenta in these diseases and combine it into one group - the so-called toxemic placentas. They are also similar in the presence of typical complications: widespread heart attacks and premature detachment of a normally located placenta, which are based on changes in the spiral arteries of the uterus, which are easily damaged due to the lack of a sufficiently developed elastic framework in their wall. Changes in the form of plasmorrhagia, secondary lipoidosis and thrombosis, as well as fibrinoid lesion of the vessels of the fetal villi, destruction of the capillary endothelium dominate in the hypertensive disease of the mother.

Placenta with anemia in pregnant women. Iron-deficiency anemia in pregnant women is a frequent and common pathology that leads to many complications: with a mild degree of anemia, complications during childbirth make up 10%, with a severe degree - 70%. The placenta undergoes changes, mainly as a result of the deterioration of the oxygen supply of the mother's erythrocytes. With moderate and severe anemia in pregnant women, dyscirculatory, alternative and compensatory reactions are emphasized in the placenta. Typical accumulations of maternal erythrocytes in the intervillous space, hemorrhage or white infarcts. In many terminal villi, dystrophy and desquamation of the syncytial cover, sclerosis of the stroma, a large number of immature villi with a two-layer structure of the syncytium, and a central arrangement of capillaries are observed. Compensatory and adaptive mechanisms are found, mainly, angiomatosis of immature villi, an increase in the number of terminal villi with SCM, the presence of syncytial kidneys. At the same time, as the severity of anemia increases, the area of the syncytial cover decreases. It is important to emphasize that newborns from mothers suffering from iron-deficiency anemia are less adaptive in the first hours and days of extrauterine life.

Placenta in pregnant women with diabetes. With diabetes in pregnancy, significant variability in the mass and histological structure of the placenta is emphasized, which is mainly explained by the degree of severity of the mother's main disease and the term of pregnancy. At the same time, E. Govorka singles out three variants of the placenta in terms of mass: excessively large, medium, very small.

An excessively large placenta (550-800 g) is observed during full-term pregnancy in mothers whose diabetes began around the age of 20, lasted no more than 10 years, and was not accompanied by vascular complications (micro- and macroangiopathy, etc.). Histologically, such a placenta most often corresponds to the variant of pathological immaturity - the type of embryonic villi. The

placental tissue is dominated by large, multilobed villi with a two-layer syncytium, a loose stroma with characteristic channels containing Kashchenko-Hoffbauer cells, and narrow, centrally located capillaries. Nuclear forms of fetal erythrocytes are sometimes visible in their lumen. Thickening of the walls of arterioles in the supporting villi and in the composition of the chorionic plate is also common. They also describe severe changes in the spiral arteries of the uterus with expansion of their subendothelial zone as a result of the formation of fibroblasts and fibrin deposits, similar to diabetic angiopathy of other localizations. The body weight of the child reaches, as a rule, 50006000 g.

The average placenta (400-500g) is found during full-term pregnancy in mothers suffering from diabetes in compensated forms. The structure of such a placenta corresponds to a dissociated variant of pathological immaturity with a characteristic alternation of mature and immature cotyledons. Along with the observations described above, there are also terminal villi with numerous SCM. The prognosis for the newborn is good, the body weight of the newborn does not exceed 3700-4500 g. A very small placenta (less than 300 g) is observed in premature pregnancies of 28-30 weeks. in mothers who suffered from juvenile diabetes with a disease history of more than 20 years. A very characteristic combination with late toxicosis of pregnant women, and the histological picture of small placentas resembles that of toxicosis. Changes in the wall of the arterial vessels of the choral plate and umbilical cord, as well as the spiral arteries of the uterus in the form of plasmatic impregnation, sclerosis, fibrinoid necrosis, damage to the endothelium, proliferation of myofibroblasts in the subintimal layering prevail.

Placenta in isoimmune conflict between mother and fetus. In this pathology, the placenta has large dimensions, its weight is 450-600 g. There are cotyledons of various sizes, separated by deep furrows; the surface of the fruit is pale yellow, the parenchyma is loose, swollen, and poorly drained. Swelling and yellowish color are also found in the fruit membranes and in the thickened umbilical cord. The histological picture of such a placenta corresponds to that of large placentas with maternal diabetes.

Litter inflammation

Infectious damage to the litter is important in perinatal pathology, as it can lead to the death of the fetus or to the disease of the newborn. There are inflammations of: intervillous space - intervillusitis; villi - villousite; basal plate - basal deciduit; chorionic plate - chorioamnionitis. Occasionally, the entire placenta is affected - spilled placentitis. Accordingly, inflammation of the umbilical cord - funiculitis, fetal membranes - parietal amnionitis.

The etiology of litter inflammation is associated with viruses, plasmas, protozoa, fungi, bacteria, as well as chemical irritants - meconium, its proteolytic enzymes, changes in the pH of the amniotic fluid, etc. However, not every inflammation of the litter is accompanied by infection of the fetus, and, in addition, infection of the fetus, for example, with some viral infections, can occur without inflammation of the litter. The ways of spreading the infection can be different. Most often, the ascending route of infection is observed with early drainage of water and a long dry period; hematogenous infection from the mother's blood through the spiral arteries into the intervillous space or during the transition of the process to the chorionic villi is less common.

Morphological diagnosis of placentitis differs in a number of features.

First, the inflammatory reaction is expressed moderately, in particular, leukocyte infiltration. Leukocytes can come from the blood of the mother - in the basal plate, intervillous space, or from the blood of the fetus - in the capillaries of the villi, umbilical vessels, or be of mixed origin. The assessment of leukocyte infiltration requires some caution, since aseptic accumulations of leukocytes in the umbilical cord and chorionic plate (of fetal origin) are formed during prolonged labor accompanied by intrauterine hypoxia of the fetus. They are found in the droppings of dead fetuses that suffered from oxygen starvation for a long time, as well as in live children born asphyxiated. In fact, this is a peculiar reaction of rejection of the placenta by the maternal tissue - the uterus. In controversial cases, the decisive argument in the diagnosis is a virological or bacteriological examination of placenta tissue,

Secondly, the accompanying signs of delayed villous tree development can be used to judge the early action of an infectious agent, for example, in toxoplasmosis, listeriosis, syphilis, etc. B-third, with an infectious defeat of the droppings, blood circulation disorders, alteration and productive changes of the epithelium of the villi and fruit membranes often dominate.

B-fourthly, the use of appropriate methods: immunofluorescence, bacterioscopy, detection of viral inclusions provides significant help in the etiological diagnosis of litter lesions.

The most common type of placentitis are viral and mynoplasmic lesions, which are clinically manifested as SARS during pregnancy. In the placenta, changes similar to those found in the respiratory organs of a fetus or newborn are emphasized.

Microplasma infection is characterized by hypertrophy of syncytiotrophoblast villi with vacuolization of their cytoplasm and the presence of inclusions in vacuoles; by the immunofluorescent method, mycoplasmas are also detected in the cells of the stroma of the villi, in the basal plate. In the intervillous space and the basal lamina, lymphoid infiltrates with an admixture of leukocytes are constantly observed, while their presence is rarely noted in the chorionic plate and stroma of the villi. The prognosis for the life of the fetus depends on the prevalence of inflammatory and alternative changes in the tissue of the placenta.

With herpetic and adenovirus infection, cells with large, hyperchromic nuclei appear in the placenta tissue in the chorionic plate, villous epithelium, basal plate, and in the septal cells. With cytomegaly in the placenta, typical cytomegaloviruses are found in the stroma of the villi; foci of inflammation do not have clear boundaries, are more often located under the syncytial cover of villi, larger ones occupy the entire stroma of individual villi.

When the placenta is damaged by RNA viruses, in addition to the proliferation of syncytiotrophoblast villi in para-influenza and MS infection, the formation of papillary structures in the epithelium of the amnion and fetal membranes is characteristic. Small foci of acidophilic necrosis, areas of disorganization of the stroma of villi and vessel walls are observed during influenza. In smears - nipples from the amnion, villous chorion and basal plate, fuchsinophilic inclusions are constantly detected, more often cytoplasmic, liquid intranuclear ones. Lymphoid infiltrates with an admixture of leukocytes in the composition of the chorionic plate are constantly detected, as well as swelling of the endothelium, proliferation of cells of all layers and narrowing of the lumen of fetal vessels, although such pathology of the endothelium is hardly specific. The most favorable prognostic factors for the fetus and the newborn are combined viralmycoplasma-bacterial lesions of the placenta, proceeding according to the type of basal deciduit or spilled placentitis.

A purulent bacterial infection is characterized by serous-purulent, purulent inflammation, sometimes with the development of phlegmon or abscesses.

In case of listeriosis, yellowish-gray foci of necrosis with histioleukocyte infiltration on the periphery are found, granulomas are emphasized in the composition of the chorionic plate, in Warton's cord and blood vessels. Listerella are clearly visible on semi-thin sections of the villi and in the basal plate.

In tuberculosis, foci of caseosis, nodules with epithelioid and giant cells are observed, the basal plate is more often affected. Changes in the placenta in congenital syphilis are characterized by swelling or fibrosis of the stroma in the terminal and trunk villi, focal polymorphic cellular infiltrates with or without necrosis inside the villi. Mesenchymal cells and Kashchenko-Hofbauer cells are part of the infiltrates. The diagnosis is clarified when spirochetes are detected in the tissue of the placenta and with the help of serological tests of the mother and fetus.

With toxoplasmosis, cysts, pseudocysts and free parasites are found in the area of extensive necrosis with calcifications of the placenta tissue.

In case of candidiasis, inflammatory infiltrates consist of polymorphonuclear leukocytes and mononuclear cells. Many hyphae of the fungus are usually found, more often in the chorionic plate, fruit membranes.

In malaria, the causative agent is detected in large quantities in the intervillous space and in the vessels of the decidual membrane, as well as in the erythrocytes of the mother, and in the tissues - deposition of malarial pigment.

Tumors of the placenta

True tumors of the placenta are represented by hemangiomas, angiofibromas and occasionally teratomas. Placental hemangiomas occur relatively often, approximately 1 case per 100 births. Their sizes vary from microscopic nodules to large foci resembling hematomas or heart attacks.

Cavernous or capillary forms of hemangiomas are usually diagnosed histologically. They should be differentiated from the option of pathological immaturity - chorioangiomatosis of the placenta and compensatory angiomatosis of the villi. Often, angiomas have the character of angiomyxoma or angiofibroma. Large chorioangiomas are often combined with polyhydramnios, disorders of fetal development.

Quite large teratomas of the placenta with various tissue components of all three germ layers are occasionally observed. It is assumed that such teratomas are the so-called amorphous fetus in multiple pregnancy. Sometimes metastatic nodes are found in the placenta: melanoblastoma of the mother, various forms of cancer. There were cases of congenital leukemia with marked leukemic infiltration of the stroma of the villi, but without the transition of leukemic cells into the maternal vascular bed.

Pathology of the umbilical cord and fetal membranes

Anomalies of the length of the umbilical cord. In perinatal pathology, both shortening and excessive lengthening of the umbilical cord are important. At 34-42 weeks of pregnancy, the length of the umbilical cord increases from 53 to 57-60 cm, this parameter is closely correlated with the length of the fetus.

The umbilical cord is considered short if it is 40 cm or less in length. A rare syndrome of umbilical cord insufficiency is known - aplasia or rare shortening of the cord up to 8 cm. Such a case is characterized by a combination with underdevelopment

Front abdominal wall and internal organs, therefore this syndrome is often called "eventration", "umbilical-fetal dysplasia". The development of the spine, limbs, lungs, heart, and genitourinary system of the fetus is repeatedly disrupted. Although the time of fetal damage is established (3rd week of pregnancy), the cause of umbilical cord aplasia is unknown. Fetuses die around 15-25 weeks of pregnancy. Shortening of umbilical cords from 10 to 20 cm in 60 cases is accompanied by premature birth, in 36% - the birth of dead fetuses, with the length of the umbilical cord of 25-35 cm, such complications are less - 32% and 14%, respectively.

Excessive elongation of the umbilical cord (more than 62 cm) sometimes occurs during pregnancy, but it has no serious thanatogenetic significance.

Umbilical cord cysts. There are false cysts in Warton's cold with sizes up to 1-1.5 cm; most often, they are emphasized in cords with twists in dead fetuses, but they also occur in full-term newborns. True cysts are formed from the remnants of the yolk or allantoic ducts. Cysts of the yolk duct have a typical localization - in the triangle between the vessels of the umbilical cord. They usually have microscopic dimensions and are lined with cuboidal epithelium. Cysts of the allantoic duct consist of a flat epithelium, a connective tissue membrane and a concentric layer of Warton jelly.

Occasionally, tumors are found in the cord: teratoblastoma, etc.

Forms of compression of the umbilical cord. There are prolapses, entanglements, entanglements, knots and compression of the umbilical cord.

Prolapse of the umbilical cord is closely related to premature rupture of the fetal membranes and occurs most often before or during childbirth. Tachycardia and bradycardia of the fetus develops. If this formidable complication is not diagnosed in time, the fetus dies intranatally as a result of asphyxiation.

Wrapped or true knots of the umbilical cord are emphasized in the presence of a small fetus, a long umbilical cord and polyhydramnios. Such complications occur in 0.4-0.5% of all births. The timing of the formation of umbilical cord knots is difficult to determine, since during pregnancy the knots usually do not tighten due to blood pressure and pulsation of the umbilical cord vessels. The entwined umbilical cord and the formation of its true knots represent a danger in childbirth, when

their tightening leads to the death of the fetus. Serious difficulties arise when assessing the thanatogenetic role of cord twists in stillbirth. It is believed that signs of intrauterine acute torsion of the umbilical cord are compression or obliteration of the umbilical vein and the presence of strangulations on the body of the fetus.

Symonart's compression of the umbilical cord by amniotic cords is known as amniotic cord syndrome. S. Heifetz, analyzing 6 of his own observations and 57 cases described in the literature, singled out a triad of signs: separation of the amnion from the placenta, adhesions between the fetus and the remains of the amnion, as well as deformations or severe defects in the development of the fetus. In 58 observations, the fetuses died in the antenatal period, in 3 - in the intranatal period, and 2 newborns died during the 1st week.

Abnormalities of attachment of the umbilical cord. The most clinical significance is the marginal and membrane attachment of the umbilical cord. It should be emphasized that these placentation disorders often accompany variants of placental insufficiency. Based on the analysis of 1000 placentas in singleton pregnancies, P. Uyanwah-Akrot, H. Fox (1979) concluded that marginal and membrane attachment of the umbilical cord has a pathogenetic connection with an increase in the frequency of miscarriages, malformations, fetal hypoxia, intrauterine death, prematurity and etc.

Aplasia of one of the umbilical arteries. This disorder refers to rare, but serious malformations of the umbilical cord; diagnosed by the absence or obliteration of one of the two arteries on the section of the cord. Its attachment to the thicket is atypical - edge or membrane. The placenta is lobular: in 21% of cases it is very small (100 g less than the gestational norm), in 18.6% it is surrounded by a shaft, in 32.6% it has heart attacks. B 80-90% of observations emphasize severe defects: fetuses without a heart, Down's disease, malformations of the genitourinary organs, etc. With aplasia of the umbilical artery, the number of premature babies increases, perinatal mortality increases to 16.5%, chromosomal disorders are not uncommon, in particular, trisomy of the 18th pair of chromosomes.

Pathology of fruit membranes. Premature rupture of the fetal membranes, which can occur starting from the 28th week of pregnancy, has the greatest clinical significance. Early rupture of membranes increases the frequency of pre- and neonatal infections. Pathology of the fetus is caused most often by the accompanying loss of the umbilical cord. Polyhydramnios is a frequent symptom of late toxicosis of pregnant women, placental transfusion in multiple pregnancies. At the 37th week of pregnancy, the volume of amniotic fluid is 450-500 ml, before childbirth - 600 ml. An increase in the amount of water up to 2 liters is more often combined with fetopathy - hemolytic disease, diabetic fetopathy, sometimes with embryopathy.

Anemic - a decrease in the amount of amniotic fluid to 500 ml or less - is combined with hypoplasia of the fetus and placenta and with embryopathies. The etiology and pathogenesis of polyhydramnios and hypohydramnios have not been established.

Amniotic adhesions (Simonart cords) are dense connective tissue hyalinized cords or threads that go from the amnion to the surface of the fetus. In full-term fetuses, they cause the formation of furrows or amputation of fingers, toes, forearms, lower legs, shoulders, and thighs. They are less often attached to the body. Embryos are allowed to have a teratogenic influence on the development of hypoplasia or malformations of the limbs. They are especially common in hypohydramnios. Rare defects include an incomplete amnion, resulting in an embryo it is partially located outside the amniotic cavity, which is accompanied by its fusion with the chorion and severe developmental defects. Benign dysplasia of the mammary gland (synonyms: mastopathy, fibrocystic disease) is characterized by a violation of the differentiation of the epithelium, its atypia, a change in histostructure, but without penetration through the basement membrane and the possibility of reversible development. Its development is associated with a violation of the balance of estrogens.

There are two main forms of mastopathy - non-proliferative and proliferative.

The non-proliferative form is characterized by the spread of dense connective tissue with areas of hyalinosis, in which atrophic lobes and cystic-dilated ducts are located. Ducts and cysts are lined with atrophic or high (apocrinized) epithelium, which forms papillary growths. This form of dysplasia can be in the form of a single dense node (nodes) - this is fibrous mastopathy; or a whitish dense node with cysts in it (fibrocystic mastopathy) more often in one mammary gland.

The proliferative form is characterized by the growth of the epithelium and myoepithelium or the joint growth of the epithelium and connective tissue. Varieties of this form of mastopathy include adenosis (masoplasia) - proliferation of intraductal or lobular epithelium. Adenosis (masoplasia) is characterized by an increase in the size of the particles due to the proliferation of glandular epithelium. The growth of ductal or lobular epithelium leads to the formation of structures of solid, adenomatous and cribriform type, and at the same time connective tissue grows. In sclerosing (fibrosing) adenosis, the proliferation of myoepithelium prevails. At the same time, foci formed by myoepithelial cells and epithelial tubules appear; later sclerosis and hyalinosis of the entire gland join. Against the background of benign breast dysplasia, cancer often develops, Mastitis is an inflammation of the mammary gland, depending on the course, it can be both acute and chronic.

Acute purulent (phlegmous) mastitis is quite common in women in the postpartum period; more often, its causative agent is staphylococcus. In most cases, chronic mastitis is a consequence of acute and purulent inflammation.

1. Theoretical questions Questions for self-control:

22. Give the classification of diseases of pregnancy and the postpartum period.
23. Etiology, pathogenesis and pathology of eclampsia.
24. Ectopic pregnancy, types, course and complications.
25. Morphology of spontaneous abortion.
26. Trophoblastic disease: morphology and invasive invasion of the bladder.
27. Trophoblastic disease: morphology of choroid carcinoma.
28. Pathology of the litter: age-related changes and disorders of placenta implantation processes.
29. Morphology of placental blood circulation disorders.
30. Classification of placental insufficiency.
31. The morphology of litter inflammation.
32. Tumors of the placenta.
33. Pathology of the umbilical cord and membranes.
34. Types of mastitis

2 Practical tasks

1. Prepare an essay on the topic: "Ectopic pregnancy, types, course and complications."
2. Make a graph of the logical structure "Classification of placental insufficiency.".
3. **Test tasks for self-control:**

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

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Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory http://www.webpathology.com/- Web Pathology

Topic #8: "Asphyxia of newborns. Birth trauma.»

Purpose: as a result of independent study of this topic, students should know the topic as the diseases of newborn children are the main problem of neonatology at the modern level, because the reason for the mortality of children in the first year of life is most often such diseases. Modern doctors are obliged to know about diseases of newborns in order to prevent their occurrence and various complications that may result from these diseases

Basic concepts:

The student should know:

31. causes of newborn asphyxia

32. classification of asphyxia

33. definition of birth trauma

34. principles of diagnosis of the pathology of the postpartum period

35. characteristic morphological signs of asphyxia of newborns

36. characteristic morphological signs of birth trauma The student should be able to:

30. distinguish morphological signs of asphyxia
31. master the skills of interpreting the results of macro- and microscopic research based on the study of anatomical preparations
32. study and explain morphological changes, describe the macropreparation

33. evaluate the results of the conducted research and describe them in the study album 34. predict possible complications

Topic content:

Asphyxia

Asphyxia of the newborn is a syndrome characterized by the absence of breathing or individual irregular and ineffective respiratory movements at birth in a child with cardiac activity. Intrauterine hypoxia refers to oxygen starvation of the fetus. The birth of a child in asphyxia can be a consequence of hypoxia. Causes of chronic intrauterine hypoxia: - maternal diseases
(decompensated heart defects, diseases of the broncho-pulmonary system, anemia, infections and intoxication); - occupational hazards of a pregnant woman; - bad habits of a pregnant woman; - violation of utero-placental blood circulation due to long-term toxicosis of pregnant women, placenta previa, carriage, changes in the placenta in case of somatic diseases of the mother (hypertensive disease, nephropathy, etc.) and endocrine diseases of the mother (decompensated or poorly compensated diabetes); - fetal diseases (severe forms of hemolytic disease, generalized intrauterine infections, brain malformations, etc.). Not only the presence of one or another maternal pathology determines the development of hypoxia of the fetus, but also its severity and duration, as well as the combination with other concomitant conditions and diseases that sharply increase dystrophic and reduce compensatory and adaptive changes in the placenta.

The causes of acute hypoxia of the fetus, which lead to the birth of a child in asphyxia, are as follows: - acute hypoxia during childbirth in the mother (posthemorrhagic, etc. shock in the mother, carbon monoxide poisoning, etc.); - acute violation of utero-fetal (umbilical) blood circulation as a result of tight entanglement with the umbilical cord, its own knots of the umbilical cord, stretching of the short umbilical cord, loss of the umbilical cord loops, pressing the head of the umbilical cord loops against the walls of the birth canal; - acute disturbance of utero-placental blood circulation in the case of uterine rupture, premature detachment of the placenta, weakness or excessive labor activity, compression of the vena cava by the uterus; - damage to the brain of the fetus with suppression of the respiratory center (medicinal, narcotic, infectious,

Pathogenesis of asphyxia. Each fetus that develops normally in the mother's womb experiences physiological asphyxia due to the peculiarities of intrauterine development. A number of compensatory and adaptive mechanisms (fetal hemoglobin, polycythemia, relative tachycardia) allow the fetus to develop normally. Under the action of etiological factors, a short-term moderate hypoxia first develops, which is accompanied by the tension of compensatory mechanisms aimed at maintaining adequate oxygenation of the fetus. The release of glucocorticoids by the adrenal cortex, the number of circulating erythrocytes, BCC increases. Tachycardia and some increase in systolic pressure occur. Continued hypoxia leads to activation of anaerobic glycolysis, redistribution of blood circulation with predominant blood supply to vital organs. Severe and prolonged hypoxia leads to the breakdown of compensatory mechanisms: the adrenal cortex is exhausted, bradycardia, arterial hypotension, collapse, shock develop. Pathological acidosis increases the permeability of the vascular wall and cell membranes, which leads to hemoconcentration, the formation of intravascular blood clots, and hypovolemia. The central link in the pathogenesis of newborn asphyxia is metabolic acidosis, the more severe it is, the more severe the asphyxia. A blood pH of less than 7.2 is considered pathological for a newborn. With long-term hypoxia, the reticular formation is suppressed, and therefore, during birth, the flow of afferent impulses is unable to lead it to the excitation necessary for the first inhalation of air. This explains the main clinical symptom of asphyxia - lack of breathing in a newborn. The most pronounced changes occur with asphyxia in the central nervous system. Disorders of cerebral blood circulation can be transient, in the form of edema and swelling of the brain, and focal (hemorrhages, ischemic heart attacks).

The classification of asphyxia is based on the evaluation of the condition of the newborn according to the Apgar scale. A healthy newborn has a score of 8-10 points. In accordance with the international classification of diseases (Geneva, 1980), the following are distinguished: - moderate or moderate asphyxia - assessment on the Apgar scale for 1 minute. 4-6 points, for 5 minutes. – 810 points, for 1 minute. – 7 points, for 5 minutes. - 7 points; - severe asphyxia - assessment on the Apgar scale for 1 minute. – up to 3 points or 4 points, which in dynamics do not reach 7 points. Clinic of asphyxia: Mild degree of asphyxia (6-7 points) – the newborn is in a depressed state. Then follows a period of hyperexcitability, which lasts for 1-2 days. Of course, there are no signs of focal changes. The prognosis is favorable. Moderate asphyxia (4-5 points) is manifested by a greater degree of depression of the central nervous system. This is followed by a long period of excessive excitement. After 12-24 hours. convulsions may develop. The forecast is different. Severe asphyxia (0.3 points) manifests itself in the form of a coma, constant convulsions caused by cerebral edema and intracranial hemorrhages. After severe and prolonged asphyxia, hypoxic cardiomyopathy may develop.

According to foreign and domestic authors, a component of severe asphyxia of newborns is multiple organ failure syndrome /SPN/, which is characterized by the failure of two or more systems in a critical state of the body. Therefore, to establish a diagnosis of "asphyxia" in a newborn, it is necessary to take into account the peculiarities of antenatal development, the course of childbirth, support of the main vital systems of the body from the first seconds of the child's life, the Apgar score, the degree and nature of acidosis, the results of biochemical studies and other indicators.

Asphyxia - hypoxic, which is combined with hypercapnia, the condition of the fetus or newborn, which can develop before, during and after delivery. Causes of asphyxia: Causes of asphyxia in the antenatal period:

- anoxic state of the mother;
- acute disorders of utero-placental or placental-fetal blood circulation;

Causes of asphyxia in the intranatal period:

- premature detachment of the placenta;
- violation of utero-placental blood circulation;
- placenta previa;
- violation of the blood flow along the umbilical cord as a result of: squeezing the head of the fetus, falling out of the birth canal of the mother, overstretching of the umbilical cord, tight wrapping around the neck of the fetus, true knots of the umbilical cord.

Types of asphyxia:

- blue asphyxia, characteristic of chronic intrauterine asphyxia; • white asphyxia as a result of an acute hemodynamic disorder of the collapse type.

Morphological signs of asphyxia:

- dark liquid blood in the cavities of the heart and large blood vessels; • cyanosis and acrocyanosis;
- edema of the feet, scrotum and labia;

•hemorrhages on the serous membranes.;

- lungs of a fleshy consistency, do not fill the chest, airless pieces sink in water; • aspiration of amniotic fluid elements;
- consumption coagulopathy.

Birth trauma

Childbirth trauma is local damage to the tissues of the fetus during the birth act, which occurred as a result of the action of mechanical force directly on the fetus, and not on the placenta or umbilical cord, and is manifested by tears, fractures, dislocations, crushing of tissues.

Pathogenesis. Mechanical damage to the brain leads to a violation of hemo- and fluid dynamics, brain edema, and intracranial hemorrhage. In the first hours and days of life, brain damage has mainly an ischemic-thrombotic genesis. Hemorrhage into the brain most often occurs from terminal veins with damage to the periventricular areas of the white matter of the cerebral hemispheres and subcortical nodes. Intracranial hemorrhages can be epidural, subdural, subarachnoid, intraventricular, intracerebral and mixed.

In relation to the tent of the cerebellum, hemorrhages are divided into supra- and subtentorial.

Classification of intracranial hemorrhages:

1). Extracerebral (epidural, subdural, subarachnoid); 2). Intracerebral (peri-, intraventricular, parenchymal, hemorrhages in the thalamus and basal nuclei, intracerebellar).

The degree of damage to the fruit depends on

- the degree of prematurity or prematurity of the fetus;
- the degree of formation and size of the skull;
- the degree of formation of the sickle-shaped process and the tent of the cerebellum;
- rigidity of the tissues of the birth canal;
- the shape and size of the pelvis;
- disruption of tissue displacement of the birth canal during premature rupture of the fetal bladder;
- dynamics of childbirth (rapid childbirth);
- duration of standing of the fetal head in the cervical canal of the uterus.

Morphological signs of birth trauma

- birth tumor;
- hemorrhages;
- cephalohematoma;
- hemorrhages in the cranial cavity;
- hemorrhages in the ventricles of the brain;
- damage to the bones of the skull.

Mechanical injuries during childbirth are accompanied by hemodynamic disturbances, tissue swelling, and hemorrhages. In order to understand the processes that take place in this case, knowledge of the anatomical and physiological features of the central nervous system in newborns is necessary. Pathological symptoms of birth trauma of the central nervous system appear immediately after birth or after the so-called "light interval" - after a few hours, and sometimes days of the postnatal age. Typical in the acute period are: violation of thermoregulation; disorders of muscle tone; change in the nature of the cry; abnormal movements of the eyeballs, convulsions, tension and bulging of the parietal lobe, vegetative-visceral disorders, peripheral blood circulation disorders, progressive anemia and metabolic disorders. Neurosonography and a thorough medical examination of the baby with the participation of a neurologist are necessary to recognize a birth injury.

1. Theoretical questions Questions for self-control:

35. Define asphyxia.
36. Classification of asphyxia
37. Types of asphyxia
38. Etiology and pathogenesis of asphyxia of newborns.
39. Morphological signs of asphyxia.

40. Etiology and pathogenesis of birth trauma.

41. Morphological signs of birth trauma.

2 Practical tasks

1. Prepare an essay on the topic: "Morphological signs of birth trauma."

2. Make a graph of the logical structure "Types of asphyxia".

3. **Test tasks for self-control:**

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

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Electronic information resources

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- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory http://www.webpathology.com/- Web Pathology

Topic #9: "Tumors of respiratory organs.»

Purpose: as a result of independent study of this topic, students should know the topic, as knowledge of the topic is necessary in the practical activity of a doctor to understand the mechanisms of the occurrence and development of diseases of the respiratory organs, as well as knowledge of the topic is necessary for the interpretation of morphological data obtained when using biopsy and autopsy research methods.

Basic concepts:

The student should know:

37. Etiology and pathogenesis of tumors, methods of experimental reproduction of tumors, mechanisms of infiltrative growth of malignant tumors and the formation of metastases, anti-transformation materials, the relationship between the tumor and the organism.

38. Morphological classification, morphological characteristics of tumors of the respiratory organs.

The student should be able to:

35. explain the main etiological and pathogenetic mechanisms of the occurrence and development of respiratory diseases;
36. interpret pathomorphological changes in the respiratory organs with subsequent diagnosis of a specific disease.
37. determine ways of diagnosing malignant tumors

Topic content:

The cause of a tumor is the action of factors that lead to irreversible changes in the genetic apparatus of the cell, as a result of which the process of its distribution is disturbed. In affected cells, uncontrolled stimulation of the partition occurs or its inhibition is lost (becomes ineffective). The generally accepted theory of tumor growth has not yet been defined, but the polyetiological theory and the concept of carcinogenesis of neoplasms, as a staged process of transformation of a normal somatic cell into a tumor, have received the widest recognition. Changes in cells can be caused by the direct or indirect action of various endogenous (5% of all carcinogens) and exogenous factors (95%) - chemical, biological and physical.

Chemical carcinogens are the largest group, their number is constantly increasing as a result of industrial development, urbanization, oil production and processing, etc. They can be of biological origin and formed endogenously in the human body as a result of metabolic processes. Physical carcinogens are ionizing and ultraviolet radiation, elevated temperature, highintensity ultrasound, and galvanic currents, which directly damage DNA and activate free radical oxidation with damage to the genetic apparatus. Long-term mechanical action (chronic trauma, long-term compression of tissues) also has a carcinogenic effect.

Biological carcinogens are various DNA- and RNA-containing viruses that penetrate the cell and integrate into its genome. They either directly contain the gene responsible for tumor growth (oncogene), or activate the corresponding genes in the human genome.

Although carcinogens are extremely diverse in nature, they all share certain characteristics:

1. Carcinogens are able to cause irreversible changes in the genome of a cell that do not lead to its death.
2. They can penetrate through biological barriers, including the cell membrane (which is a condition for the interaction of carcinogenesis with the DNA of chromosomes). 3. Have the ability to cumulate and summation of the carcinogenic effect.
4. Subthreshold concentrations/doses of a carcinogen do not exist, even its minimal concentration can lead to the appearance of a tumor. 5. Most carcinogens suppress the body's immune reactions.

Mechanisms of tumor cell transformation and subsequent tumor development. The mechanism of cell division is simplified in the Hughes hypothesis, according to which the regulation of cell division is determined by a system of initiator genes that trigger mitosis and suppressor genes that block the initiator gene. During reparative regeneration, inflammation, compensatory hyperplasia, cell division occurs under the influence of external paracrine regulatory factors (growth factor, etc.). Cell division is controlled and regulated by several mechanisms: - inhibition of the synthesis of growth factors by a feedback mechanism;

- contact inhibition (a cell stops dividing after its membrane comes into contact with the membrane of a neighboring cell);
- Hayflick's limit - the cell divides a certain number of times, then dies.

Phases of carcinogenesis:

1. Initiations - when irreversible violations of the genotype of a normal somatic cell occur and it becomes prone to transformation into a tumor cell. When genotoxic carcinogens interact with DNA, translocation occurs and the action of the proto-oncogene, a gene that controls cell growth and differentiation and gives it its inherent qualities, is enhanced. In the initiation stage, about 30 doublings occur, that is, 30 generations (about 1 billion) of latent cells are formed and the production of oncoprotein increases, with the appearance and increase of its production, the second phase of carcinogenesis begins - promotion. 2. Promotion - the beginning of tumor growth (promotion), as a rule, begins under the influence of a provoking factor - trauma, inflammation, chemical agents, hormonal stimuli. Tumor cells, which were in a latent state, begin to actively divide, forming a tumor node. believe

Tumor growth is accompanied by a change in the properties of tumor cells aimed at increasing the autonomy of the neoplasm (tumor progression). Mainly, they develop in the direction of malignization and increase in malignancy of the tumor.

The following changes are most characteristic: - an increase in the number of spontaneous and induced mutations in tumor cells with the appearance of poorly differentiated, aggressive forms; - "clonal evolution" - under the action of the body's protective mechanisms and drug treatment (chemotherapy), only the most resistant tumor cells survive. This explains the ability of benign tumors to undergo malignant transformation and the appearance of x-ray and chemoresistance in malignant tumors during treatment. The influence of a carcinogenic factor and the inclusion of the mechanisms of carcinogenesis is not enough for the emergence of a tumor, since there are various mechanisms of antitumor resistance in the body.

Theories of the occurrence of cancer: - "immunological surveillance" (Bernet, 1970) - active T-lymphocytes are unable to recognize and destroy tumor cells from the moment of their appearance. - mutational theory - the basis of the disease lies in changes in the cell genome (accumulation of mutations in specific areas of cellular DNA, which lead to the formation of defective proteins). Malignant tumors develop from one cell, that is, they are of monoclonal origin. However, Lawrence A. Loeb (1974) believes that the basis of carcinogenesis is the occurrence of a huge number of mutations. - early instability (K. Lingaur, 1997) – early chromosomal instability causes the appearance of mutations in oncogenes and suppressor genes. - aneuploidy (P. Duesberg, 1999) – cancer is a consequence of aneuploidy – shortening and lengthening of chromosomes, moving their large sections (translocations).

Biological characteristics of tumor tissue:

1. Tissue and cellular anaplasia or metaplasia. Anaplasia refers to the loss of specific functions of the original tissue and the approach of tumor cells to the embryonic state. Metaplasia is the acquisition by a tumor of the properties of another tissue.
2. Uncontrolled and often limitless growth.
3. Autonomy of tumor tissue.
4. A change in the metabolism of tumor cells is more characteristic of malignant tumors. It consists in the activation of glycolysis with a simultaneous change (inhibition) of oxidative phosphorylation and simplification of the biochemical structure.
5. Change in regulation mechanisms. Tumor tissue does not respond or responds inadequately to normal mechanisms of neurohumoral regulation. At the same time, the tumor tissue has its own regulatory mechanisms associated with intensive release of paracrine regulators and growth factors.

6. Change in antigenic properties of a tumor cell. Tumor tannin differs in its antigenic properties from body tissues, but the tumor effectively avoids mechanisms of immunological control.

Tumors of the respiratory system

The respiratory system is divided into the airways and the respiratory department. The airways include the nasal cavity, pharynx, larynx, trachea, bronchi of various calibers, including bronchioles. Here the air is heated (cooled), cleaned of various particles and moistened. The respiratory department consists of alveolar ducts and alveoli that form acini. Gas exchange takes place in them.

Lung neoplasms represent a large group of diseases that are distinguished by their morphological structure, type of growth, clinical course, treatment tactics, and prognosis. Below is the International histological classification of lung and pleural tumors (1999). First published in 1967, it facilitates comparability of data for professionals from all over the world.

1. Epithelial tumors

1.1. Benign

1.1.1. Papillomas

1.1.1.1. Squamous cell papilloma:

1.1.1.1.1. Exophytic

1.1.1.1.2. Inverted

1.1.1.2. Glandular papilloma

1.1.1.3. Mixed squamous-glandular papilloma

1.1.2. Adenomas

1.1.2.1. Alveolar adenoma

1.1.2.2. Papillary adenoma

1.1.2.3. Adenoma of the salivary gland type

1.1.2.3.1. Adenoma of mucous glands

1.1.2.3.2. Pleomorphic adenoma

1.1.2.4. Mucinous cystadenoma

1.2. Preinvasive lesions

1.2.1. Squamous cell dysplasia/carcinoma in situ

1.2.2. Atypical glandular hyperplasia

1.2.3. Diffuse idiopathic pulmonary neuroendocrine hyperplasia

1.3. Invasive malignant

1.3.1. Squamous cell carcinoma

1.3.1.1. Papillary

1.3.1.2. Light cell

1.3.1.3. Small cell

1.3.1.4. Basalioid

1.3.2. Small cell carcinoma

1.3.2.1. Combined

1.3.3. Adenocarcinoma

1.3.3.1. Acinar

1.3.3.2. Papillary

1.3.3.3. Bronchioloalveolar

1.3.3.3.1. Non-mucinous

1.3.3.3.2. Mucinous

1.3.3.3.3. Mixed non-mucinous and mucinous or with an intermediate type of cells

1.3.3.4. Solid with mucus production

1.3.3.5. mixed

1.3.3.6. Other options

1.3.3.6.1. Differentiated fetal adenocarcinoma

1.3.3.6.2. Mucinous (colloid) adenocarcinoma

1.3.3.6.3. Mucinous cystadenocarcinoma

1.3.3.6.4. Ring-shaped adenocarcinoma

1.3.3.6.5. Clear cell adenocarcinoma

1.3.4. Large cell carcinoma

1.3.4.1. Large cell neuroendocrine carcinoma

1.3.4.1.1. Combined large cell neuroendocrine carcinoma

1.3.4.2. Basalioid

1.3.4.3. Lymphoepithelioma-like

1.3.4.4. Light cell

1.3.4.5. Large cell carcinoma with rhabdoid phenotype

1.3.5. Adenosquamous carcinoma (glandular squamous cell)

1.3.6. Carcinomas with pleomorphic, sarcomatous or sarcomatous elements

1.3.6.1. Carcinomas with spindle-shaped and/or giant cells

1.3.6.1.1. Pleomorphic carcinoma

1.3.6.1.2. Spindle cell carcinoma

1.3.6.1.3. Giant cell carcinoma

1.3.6.2. Carcinosarcoma

1.3.6.3. Lung blastoma

1.3.6.4. Others

1.3.7. Carcinoid tumors

1.3.7.1. A typical carcinoid

1.3.7.2. Atypical carcinoid

1.3.8. Carcinoma of bronchial glands

1.3.8.1. Mucoepidermoid carcinoma

1.3.8.2. Adenocystic carcinoma

1.3.8.3. Others

1.3.9. Unclassified carcinomas

2. Tumors of soft tissues

2.1. Focal fibrous tumors

2.2. Epithelioid hemangioendothelioma

2.3. Pleuropulmonary blastoma

2.4. Chondroma

2.5. Calcifying fibrous pseudotumor of the pleura

2.6. Congenital peribronchial myofibroblastic tumor

2.7. Diffuse pulmonary lymphangiomatosis

2.8. Desmoplastic small round cell tumor

2.9. Others

3. Mesothelial tumors

3.1. Benign

3.2. Malignant

3.2.1. Epithelioid mesothelioma

3.2.2. Sarcomatous mesothelioma

3.2.2.1. Desmoplastic mesothelioma

3.2.3. Biphasic mesothelioma

3.3. Others

4. Other types of tumors

4.1. Hamartoma

4.2. Sclerosing hemangioma

4.3. Clear cell tumor

4.4. Germinogenic tumors

4.4.1. The teratoma is mature

4.4.2. Teratoma is immature

4.4.3. Other germinogenic tumors

4.5. Timoma

4.6. Melanoma

4.7. Others

5. Lymphoproliferative diseases

5.1. Lymphoid interstitial pneumonia

5.2. Nodular lymphoid hyperplasia

5.3. Indolent B-cell lymphoma

5.4. Lymphomatoid granulomatosis

6. Secondary tumors

7. Unclassified tumors

8. Tumor-like lesions

8.1. Tumorlet

8.2. Multiple meningothelioid nodules

8.3. Histiocytosis from Langerhans cells

8.4. Inflammatory pseudotumor (inflammatory myofibroblastic tumor)

8.5. Organizing pneumonia

8.6. Myeloid tumor

8.7. Hyalinizing granuloma

8.8. Lymphangioleiomyomatosis

8.9. Multifocal micronodular pneumocytic hyperplasia

8.10. Endometriosis

8.11. Bronchial inflammatory polyp 8.12. Others

BENIGN TUMORS OF THE LUNGS

Benign lung tumors are a heterogeneous group of neoplasms that originate from pulmonary structural elements. They account for 2–5% of all lung tumors, but the exact incidence is unknown because most are asymptomatic.

The nomenclature of benign tumors is based on the origin and histological features. Therefore, epithelial (adenoma, papilloma), mesodermal (fibroma, lipoma, leiomyoma, chondroma, sclerosing hemangioma), germinogenic tumors (teratoma) and others (hamartoma) are distinguished. Etiology and pathogenesis are unknown.

In addition to morphological classification, clinical classification is used, which divides tumors into central (endobronchial) and peripheral (parenchymal), single and multiple. Most often, hamartomas and bronchial adenomas are detected.

Hamartoma is the most common benign tumor of the lung, found mainly in adults. Located most often peripherally.

The shape of the hamartoma is usually rounded. The diameter of the tumor at the time of surgery is in most cases 2–3 cm, and sometimes reaches 10–12 cm or even more. The surface of the hamartoma

is smooth or, more often, finely bumpy, the consistency is dense or elastic. The tumor is clearly separated from the surrounding tissue, does not have a capsule, and is surrounded by lung tissue.

Adenomas are epithelial tumors that develop mainly from the glands of the mucous membrane of the bronchus and grow into the lumen of the bronchus - endobronchially, in the thickness of the bronchus - intramurally, as well as extrabronchially. However, in most cases, a combination of all the listed types of adenoma growth is noted.

The widespread use of the term "adenoma" in relation to carcinoid tumors, mucoepidermoid and adenocystic carcinomas, which are in fact malignant tumors, should be avoided.

The form of adenoma with endobronchial growth is more often polypoid, less often lobular, hilly. Externally, the adenoma is covered with a mucous membrane, which can sometimes be eroded.

Rare benign lung tumors include fibroids, vascular tumors (hemangioendothelioma, hemangiopericytoma, capillary hemangioma), teratomas, neurogenic tumors (neurinoma, schwannoma, neurofibroma, chemodectoma, pheochromocytoma), papilloma (fibroepithelioma), leiomyoma, histiocytoma, xanthoma.

CLINIC

Symptoms and clinical features of central benign tumors are primarily determined by the degree of impaired bronchial patency. The first clinical stage corresponds to partial, the second to the socalled valve or valve bronchostenosis, and the third to occlusion of the bronchus.

When bronchial stenosis occurs, the clinical picture of the disease depends on changes in the lung tissue in the area of impaired ventilation. As a rule, a secondary inflammatory process develops, caused by periodic obturation of the bronchus by a tumor, with the accumulation of bronchial secretions, blood, and swelling of the mucous membrane. There are atelectasis, obstructive pneumonitis, poststenotic retention bronchiectasis.

The analysis of clinical symptoms provides a reason to introduce into practice the concept of the syndrome of a central benign tumor of the lung. This syndrome is characterized by: 1) young and middle age of patients; 2) equal incidence of men and women; 3) long history; 4) wavy course; 5) cough; 6) hemoptysis of light blood, which occurs without precursors, "among full health"; 7) chronic pneumonia (Perelman M.I. et al., 1981).

With regard to peripheral tumors, two stages of the clinical course are distinguished: preclinical (asymptomatic) and the stage of the appearance of clinical symptoms associated with impaired function of the lungs and other chest organs.

At the preclinical stage, tumors are detected only during X-ray examination. In the future, the symptomatology and clinic of peripheral benign tumors are determined by the size of the tumor, the depth of its location in the lung tissue, and the relationship to the adjacent bronchi, vessels, and

organs. Peripheral benign tumors of the lungs, as a rule, do not have pathognomonic clinical symptoms and, therefore, a typical clinical picture.

A significant increase in a benign tumor can cause compression of the bronchi, pulmonary vessels and the development of inflammation in the lungs.

DIAGNOSTICS

X-ray examination is a very valuable diagnostic method for detecting benign lung tumors: X-ray and X-ray, tomography, CT. X-ray examination allows to correctly recognize benign lung tumors in 2/3 of patients and to plan the use of other research methods.

Bronchoscopy is the most important method of diagnosing central benign lung tumors. This is the only reliable method of tumor recognition before the appearance of X-ray signs of impaired bronchial patency. Bronchoscopy, when a tumor is detected, should end with a biopsy.

The X-ray picture during the period of bronchial obturation depends on the caliber of the affected bronchus. For example, with a tumor of the main bronchus, it is characterized by a complete darkening and reduction of the area of the lung field, a shift of the mediastinal organs to the diseased side regardless of the breathing phase, a high standing and restriction of the mobility of the dome of the diaphragm, a narrowing of the intercostal spaces, that is, a picture of lung atelectasis.

Benign peripheral tumors are characterized by the following symptoms:

1. The shape of the shadow is almost always rounded.
2. Uniform, as a rule, structure of the shadow.
3. Even and clear contours.
4. Very slow increase in the size of the pathological shadow during X-ray examination.
5. Absence of a decay cavity in the area of the pathological shadow, the surrounding lung tissue is unchanged, there is no path to the root.
6. Calcinates.

Differential diagnosis of benign lung tumors should be carried out with malignant lung tumors, lung metastases, tuberculosis, pneumonia, bronchial foreign bodies, echinococcosis.

MALIGNANT TUMORS OF THE LUNGS

Lung cancer ranks first among lung cancers, the share of other tumors — sarcoma, carcinosarcoma, carcinoids, lymphoma — is 0.4–5%. Treatment of sarcoma and lymphoma is carried out according to its own standards and is outlined in the relevant sections.

LUNG CANCER

Lung cancer remains the leading cause of cancer-related death in the world, accounting for ~1.2 million cases per year, and late diagnosis is a major obstacle to improving treatment outcomes. Only 15% of patients survive 5 years after diagnosis, 7% — 10 years.

In the structure of oncological morbidity in Ukraine, lung cancer ranks 2nd after skin cancer: the incidence is 37.9 per 100,000 population, the mortality rate is 30.1 per 100,000 population, the mortality rate up to one year is 63.9% (2008 r). Almost 90% of cases are over the age of 50.

Coal combustion products, exhaust gases from internal combustion engines, emissions from energy, chemical, and metallurgical enterprises, and ionizing radiation are also important in the etiology of lung cancer. Background diseases contributing to the development of lung cancer are chronic bronchitis, chronic pneumonia, pneumosclerosis, tuberculosis, anthracosis, silicosis, asbestosis.

Lung carcinogenesis is a multistage process. Squamous cell carcinoma and adenocarcinoma have defined precancerous lesions. The lung epithelium undergoes morphologic changes that include hyperplasia, metaplasia, dysplasia, and carcinoma in situ. Dysplasia and carcinoma in situ are obligate (obligatory) precancerous lesions because they are highly likely to transform into invasive cancer and have a low probability of spontaneous regression. In addition, after surgical treatment, the risk of a second lung cancer is 1–2% per year per patient. Cancer occurs more often in the right lung (56%) than in the left (44%).

CLASSIFICATION

1. Clinical and anatomical classification of lung cancer:

I. Central cancer — (endobronchial, peribronchial, branched). The tumor is localized in the main, partial or segmental (subsegmental) bronchus.

II. Peripheral cancer develops from small bronchi, bronchioles (spheroidal, pneumonia-like, Penkost cancer — the apex of the lung or cancer of the upper sulcus).

III. Atypical forms (mediastinal cancer, miliary carcinomatosis, brain, bone, liver forms) are due to the peculiarities of growth and metastasis.

Clinical and anatomical classification allows for a deeper understanding of the clinical course of the disease, interpretation of data from diagnostic procedures, and treatment planning. It is most common in the CIS countries, but the division into central and peripheral tumors and the separation of Pancoast cancer (Pancoast Tumor) is also accepted in the world.

2. Morphological classification is given above (see International histological classification of lung and pleural tumors).

In clinical practice, the terms "non-small cell lung cancer" (NSCLC) and "small cell lung cancer" (SCLC) are widely used, due to the differences in the origin, course, treatment methods and prognosis of these tumors. The division of squamous cell carcinoma and adenocarcinoma into highly differentiated, moderately differentiated, and poorly differentiated types is also widespread. Squamous cell lung cancer is noted in 40–50% of cases, adenocarcinoma in 20–30%, and small cell cancer in 20–25% of cases.

3. Classification according to the TNM system (7th revision — August 2009).

There are direct and indirect, anatomical and functional signs of lung cancer.

Direct anatomical signs: 1) bumpy, papillomatous, tumor growths of various sizes and colors; 2) various types of mucosal infiltrates in the form of elevations with a smooth, bumpy, uneven surface; 3) narrowing of the lumen of the bronchus of an eccentric or concentric character with rigidity of the walls; 4) Ikeda's triad: infiltrates with expansion of blood vessels, pathological change of the mucous membrane, blurred cartilage pattern.

Indirect anatomical signs of lung cancer are symptoms caused by tumor pressure on the bronchus from the outside, its growth into the bronchus or metastatic lymph nodes: 1) a saddle-shaped, flattened spur of the bifurcation of the trachea; 2) densification of bronchial walls, during instrumental palpation; 3) dislocation of the beginnings of segmental bronchi; 4) deformation and destruction of the crest of the intersegmental and subsegmental spur; 5) indistinctness of the pattern of cartilaginous rings; 6) loose, swollen, bleeding mucous membrane with local hyperemia. Indirect functional signs of cancer: immobility of the walls of the trachea and bronchi, local protrusion of the membranous part of the bronchi with simultaneous restriction of respiratory mobility, absence of visible vascular pulsation.

Direct bronchoscopic signs are characteristic of central cancer with endobronchial growth and peripheral cancer with bronchial sprouting. Indirect — for central cancer with peribronchial growth, peripheral cancer spreading to the walls of the bronchi, metastases in the bronchopulmonary and mediastinal lymph nodes.

The introduction of direct catheterization of small bronchi under x-ray control with aspiration of bronchial contents for cytological examination and obtaining a scraping of tumor tissue (brush biopsy) made it possible to significantly increase the diagnostic effectiveness of the method. Transthoracic puncture under x-ray control with morphological examination of the punctate is an informative method of differential diagnosis, which allows to verify the diagnosis in the absolute majority of patients with peripheral cancer.

When the totality of the results of the patient's examination does not allow both an absolutely accurate diagnosis and the exclusion of the diagnosis of lung cancer, the final stage and the only method of morphological diagnosis is thoracotomy (which can be both diagnostic and therapeutic).

In order to choose the most rational method of treatment for a patient with lung cancer, it is necessary to know the degree of spread of the tumor process to the mediastinum and to determine the state of the lymph nodes. This is determined by X-ray examination, CT scan, angiography, radionuclide diagnostic methods, bronchoscopic examination using transtracheobronchial puncture biopsy, as well as surgical methods of investigation - mediastinoscopy and parasternal (anterior) mediastinotomy.

Detection of distant metastases, especially in patients with resectable forms of lung cancer, is one of the important tasks of diagnostics. Lung cancer metastases are most often localized in the liver,

adrenal gland, kidneys, bones and brain, less often in other organs. Widespread metastasis is most characteristic of undifferentiated forms of lung cancer. Ultrasound echolocation, puncture biopsy of the liver (including under the control of a laparoscope), CT, as well as laparoscopy and diagnostic laparotomy are used to detect metastases of lung cancer in the organs of the abdominal cavity and retroperitoneal space.

For the diagnosis of lung cancer metastases to the brain, MRI and CT are the most informative. When detecting bone metastases, radionuclide methods or MRI are the most effective, since radiological signs are usually noted at a later stage.

MALIGNANT NEUROENDOCRINE TUMORS OF THE LUNGS

Among all neuroendocrine tumors, ~25% are localized in the respiratory tract. Neuroendocrine tumors of the lung include typical carcinoid, atypical carcinoid, as well as large cell neuroendocrine carcinoma and small cell carcinoma and account for 20–25% of all lung cancers. The most common are small cell lung cancer (15–20%), large cell carcinoma (~3%) and carcinoids (1–2%). About 70% of all carcinoids are localized in the large bronchi, the rest — on the periphery of the lungs. They occur most often in the right lung — 61%, especially in the middle lobe.

A typical carcinoid is a slow-growing tumor that rarely recurs. Metastases after adequate surgery develop in 7% of cases.

A special feature of carcinoid diagnosis is the possibility of scanning with octreotide to determine the spread of the disease. The use of biochemical markers (chromogranin A) contributes both to clarifying the diagnosis and further monitoring of the disease.

1. Theoretical questions Questions for self-control:

42. Definition of the concepts "tumor" and "tumorous growth".
43. Infiltrative and expansive growth, their features.
44. Metastasis, its mechanisms.
45. Tumor progression.
46. Classification of respiratory tumors
47. Characteristics of benign lung tumors
48. Characteristics of malignant lung tumors
49. Methods of diagnosing lung tumors

2 Practical tasks

1. Prepare an abstract on the topic: "Characteristics of malignant lung tumors"
2. Make a graph of the logical structure "Classification of tumors of the respiratory organs".

3. **Test tasks for self-control:**

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory http://www.webpathology.com/- Web Pathology

Topic #10: "Tumors of the gastrointestinal tract.»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:

The student should know:

39. the place of diseases of the digestive organs in the structure of general morbidity;

40. morphological classification, morphological characteristics of the most important diseases of the digestive organs.

The student should be able to:

38. to explain the main etiological and pathogenetic mechanisms of the occurrence and development of diseases of the digestive organs;

39. interpret pathomorphological changes in the digestive organs with subsequent diagnosis of a specific disease.

40. The student should have an idea of the general pathological processes that develop in the digestive organs with various pathologies.

Topic content:

Tumors of the gastrointestinal tract

general information

The cause of a tumor is the action of factors that lead to irreversible changes in the genetic apparatus of the cell, as a result of which the process of its distribution is disturbed.In affected cells, uncontrolled stimulation of division occurs or its inhibition is lost (becomes ineffective). The generally accepted theory of tumor growth has not yet been defined, but the polyetiological theory and the concept of carcinogenesis of neoplasms, as a staged process of transformation of a normal somatic cell into a tumor, have received the widest recognition. Changes in cells can be caused by the direct or indirect action of various endogenous (5% of all carcinogens) and exogenous factors (95%) - chemical, biological and physical.

Tumor growth is accompanied by a change in the properties of tumor cells aimed at increasing the autonomy of the neoplasm (tumor progression). Mainly, they develop in the direction of malignization and increase in malignancy of the tumor.

The following changes are most characteristic: - an increase in the number of spontaneous and induced mutations in tumor cells with the appearance of poorly differentiated, aggressive forms; - "clonal evolution" - under the action of the body's protective mechanisms and drug treatment (chemotherapy), only the most resistant tumor cells survive. This explains the ability of benign tumors to undergo malignant transformation and the appearance of x-ray and chemoresistance in malignant tumors during treatment. The influence of a carcinogenic factor and the inclusion of the mechanisms of carcinogenesis is not enough for the emergence of a tumor, since there are various mechanisms of antitumor resistance in the body.

Biological characteristics of tumor tissue:

1. Tissue and cellular anaplasia or metaplasia. Anaplasia refers to the loss of specific functions of the original tissue and the approach of tumor cells to the embryonic state. Metaplasia is the acquisition by a tumor of the properties of another tissue.
2. Uncontrolled and often limitless growth.
3. Autonomy of tumor tissue.
4. A change in the metabolism of tumor cells is more characteristic of malignant tumors. It consists in the activation of glycolysis with a simultaneous change (inhibition) of oxidative phosphorylation and simplification of the biochemical structure.
5. Change in regulation mechanisms. Tumor tissue does not respond or responds inadequately to normal mechanisms of neurohumoral regulation. At the same time, the tumor tissue has its own regulatory mechanisms associated with intensive release of paracrine regulators and growth factors.
6. Change in antigenic properties of a tumor cell. Tumor tannin differs in its antigenic properties from body tissues, but the tumor effectively avoids mechanisms of immunological control.

Canceresophagus most often occurs at the level of the middle and lower third of it, which corresponds to the level of bifurcation of the trachea. Much less often, it is found in the initial part of the esophagus and its transition into the stomach. Esophageal cancer accounts for 2-5% of all malignant tumors.

E t i o l o g y. The development of esophageal cancer is facilitated by chronic irritation of its mucous membrane (hot coarse food, alcohol, smoking), cicatricial changes after burns, chronic gastrointestinal infections, anatomical disorders (diverticula, ectopy of the cylindrical epithelium and gastric glands, etc.). Among the precancerous changes, leukoplakia and severe dysplasia of the epithelium of the mucous membrane are the most important.

*P a t h o l o g i c a l anatomy.*The following macroscopic forms of esophageal cancer are distinguished: annular dense, papillary and ulcerative.

*Ring-shaped dense cancer*is a tumor formation that circularly thickens the wall of the esophagus in a certain area and narrows its lumen; with disintegration (destruction) and ulceration of the tumor, the patency of the esophagus is restored.

*Papillary cancer*Esophagus-like mushroom-like cancer of the stomach. It easily disintegrates, as a result of which ulcers are formed, which penetrate into neighboring organs and tissues.

*Ulcerative cancer*is a cancerous ulcer that has an oval shape and is elongated along the esophagus.

Among the microscopic forms of esophageal cancer, carcinoma in situ, squamous cell carcinoma, adenocarcinoma, glandular-squamous, glandular-cystic, mucoepidermal, and undifferentiated cancer are distinguished.

M e t a s t a s e s these tumors are mainly lymphogenic.

C o m p l i c a t i o n associated with tumor growth in neighboring organs — trachea, mediastinum, stomach, pleura. At the same time, esophageal-tracheal fistulas are formed, aspiration pneumonia, lung abscess and gangrene, pleural empyema, purulent mediastinitis occur; cachexia develops early.

Stomach cancer

Gastric cancer (RS) ranks second in the world in the structure of malignant neoplasms, second only to lung cancer.

Approximately 95% of cases are adenocarcinoma. From the point of view of anatomical localization, a distinction is made between cancer of the cardiac part of the stomach (according to the classification of cancers of the esophageal-gastric junction according to Siewert, cancer of the cardiac department is diagnosed if the epicenter of the tumor is within 1 cm above and up to 2 cm below the upper limit of the gastric folds) and cancer subcardiac department, and according to the histopathological picture (classification according to Laurén) — intestinal and diffuse forms of adenocarcinoma. The intestinal form of cancer of the subcardiac department is the most common, which develops against the background of chronic atrophic H. pylori-associated gastritis. Cancer of the esophageal-gastric junction, in particular of the cardiac department, most often occurs against the background of gastroesophageal reflux with a long course. Diffuse cancer is characterized by an aggressive course and diffuse spread of neoplastic cells in the wall of the stomach (most often in the body of the stomach); often develops in young people against the background of genetically determined syndromes, such as hereditary diffuse gastric cancer.

Early stomach canceris a neoplasm that does not infiltrate beyond the submucosal layer of the stomach wall, regardless of the presence of metastases in the lymph nodes (very rare in early cancer).

Etiology. Clear etiological factors of RS were not identified. One of the most important etiological factors is alimentary, in particular, it is affected by carbohydrate food, a lack of vitamins A, E in food, a high content of nitrates and herbicides in water, nitrates and amines form compounds of nitrosamines, which are carcinogens, which are especially active at low acidity of gastric secretions. Smoking tobacco increases the incidence of CKD by 4 times. Salty products - increase the risk of the disease several times. Eating fresh vegetables and fruits reduces the likelihood of developing CKD by 30%. In the development of the disease, a lack of cobalt magnesium plays its role, an excess of zinc, copper in the soil. Liquid food with overeating also contributes to the development of the disease. These factors contribute to a deep restructuring of the structure of the gastric mucosa with its metaplasia and enterization.

Pathogenesis. The pathogenesis of RS is complex and largely unstudied. Most researchers agree that the histogenesis of RS can develop in two directions. The first path can be schematically presented as follows: the long-term effect (more than 20 years) on the normal mucosa of environmental factors, nutrition, and above all Helicobacter pylori, leads to the development of atrophic gastritis. Atrophic gastritis either due to intestinal metaplasia, dysplasia / adenoma, differentiated carcinoma, or due to non-metaplastic atrophy of the mucosa and poorly differentiated adenocarcinoma leads to the development of invasive cancer and metastasis. This type of histogenesis is more often observed in the elderly and is not related to a hereditary factor. The second type of histogenesis suggests the presence of a multipotent proliferative cell of the cervical zone, which develops either into a carcinoid, or through differentiated adenocarcinoma into a number of malignant neoplasms: mucinous ("mucous") adenocarcinoma, poorly differentiated adenocarcinoma, signet-ring cell carcinoma, endocrine cell carcinoma, AFP (afetoproducing) cancer. This type of histogenesis often develops without previous gastritis in young patients.

Pathological anatomy and morphology of stomach cancer

There are three main variants of stomach cancer based on the type of growth

I. Exophytic form: (expansive growth) a) plaque-like, b) polyp-like, c) saucer-like, cup-like.

II. Infiltrative form (endophytic growth) 1. Ulcerative-infiltrative form; 2. Diffuse-infiltrative form (skin, submucosal, plane infiltrative).

III. Mixed form A. Cancer from a polyp. B. Cancer from an ulcer.

The exophytic form is observed in 50-60%.

Endophytic form - 40-50%.

Morphological forms:

- adenocarcinoma;
- glandular squamous cell cancer;
- squamous cell cancer; - undifferentiated cancer; - unclassified.

Clinically, there are:

- early cancer, which develops against the background of the above-mentioned precancerous conditions;
- manifest cancer — advanced cancer (II-III stages).

Precancerous changes

1. Intestinal metaplasia

Intestinal metaplasia is a frequent pathological phenomenon. With atrophic gastritis and stomach cancer, it is found in almost 100% of cases, with stomach ulcers - in 80%. Intestinal metaplasia can be diagnosed not only during histological examination, but also macroscopically during endoscopic examination. Intestinal metaplasia of the stomach is divided into complete (small intestinal) and incomplete (large intestinal). The first variant is characterized by the presence of all the cells characteristic of the small intestine, "goblet" cells bordering enterocytes that do not secrete mucus. The deep parts of pits are similar to intestinal crypts. They are lined with basophilic epithelium, contain Paneth cells, which in their structure resemble acinar cells of the pancreas. Paneth cells are the most important feature of complete intestinal metaplasia. In incomplete intestinal metaplasia, goblet cells are located among tall prismatic cells, similar to colonocytes. Paneth cells are not detected. Polymorphism of the nuclei, an increase in the nuclear-cytoplasmic ratio is noted in the epithelium. In contrast to complete metaplasia, with incomplete metaplasia, the superficial parts of the stomach are almost indistinguishable from the deep ones, which indicates a violation of cell maturation. The volume of intestinal metaplasia is also evaluated: weak occupies up to 5% of the stomach surface, moderate up to 20%, pronounced - more than 20%. which indicates a violation of

cell maturation. The volume of intestinal metaplasia is also evaluated: weak occupies up to 5% of the stomach surface, moderate up to 20%, pronounced - more than 20%. which indicates a violation of cell maturation. The volume of intestinal metaplasia is also evaluated: weak occupies up to 5% of the stomach surface, moderate up to 20%, pronounced - more than 20%.

2. Dysplasia

Dysplasia of the gastric mucosa can be confirmed in the presence of cellular atypia, signs of epithelial dysdifferentiation and structural disorders. However, the main difference between dysplasia and cancer is the lack of own plasticity of the mucous membrane. Depending on the expressiveness of structural changes, 3 degrees of dysplasia are distinguished: I - mild, II - moderate, III - pronounced.

Dysplasia of the first degree is characterized by hyperplastic and inflammatory changes and increased cellular renewal. Such dysplasia is characterized by elongation of pits, hyperchromatosis of nuclei, increase in their diameter and nuclear-cytoplasmic ratio. With dysplasia of the II degree, the severity of the above-described changes increases. With dysplasia of the III degree, its areas have the appearance of thickening of the mucous membrane, in which two zones are distinguished. External - formed by closely spaced glands lined with basophilic columnar cells with elongated nuclei, sometimes goblet cells are found. Characteristic papillomatous growths of the epithelium. The inner zone is represented by pyloric glands or intestinal crypts. Signs of intestinal metaplasia are always observed perifocally.

3. Early stomach cancer

This stomach cancer is found within the mucosa and submucosa without signs of damage to the muscle layer, the size of the tumor may exceed 2 cm in diameter. M-type of early cancer is distinguished, when the tumor is limited only to the mucous membrane, and SM-type when the tumor spreads to the submucosa.

According to the classification of the Japanese Society of Gastrointestinal Endoscopy, three types of early gastric cancer are distinguished: type I, which rises above the mucous membrane, type II - superficial, which is divided into three subtypes: IIa - superficially elevated, IIb - superficially flat, IIc - superficially depressed , III type - recessed.

Type I and type II early gastric cancer develop from an adenomatous polyp, or may be a manifestation of exophytic growth of a tumor arising from proliferation centers of the cervical or pit epithelium of the mucous membrane in atrophic gastritis. Type I can develop against the background of an ulcer that has scarred or has a tendency to scar. Type III develops against the background of chronic deep gastric ulcer.

Early forms of cancer, as a rule, develop against the background of existing pathological changes of other diseases of the stomach and do not have their own specific picture. Most patients complain of pain in the epigastrium, weakness, nausea, less often - loss of appetite and weight loss. Early stomach cancer can be manifested by gastrointestinal bleeding.

In early cancer, when the pathological process is limited only to the mucous membrane, micrometastasis to regional lymph nodes is noted in 4% of patients, and 5-year survival with timely treatment is almost 100%. When the mucosa and submucosa are affected by a tumor process, the 5-year survival rate is 90%, and micrometastases occur in 50% of patients.

Stomach cancer screening program

Screening is a system of using tests and procedures to identify the degree of risk and development of the disease among people who have not consulted a doctor about the symptoms of this disease.

Regarding stomach cancer, it is necessary to screen people over 40 years of age in order to identify groups at risk of stomach cancer and further dynamic endoscopic monitoring of them, including

gastrobiopsy and laboratory tumor marker blood tests. Cooperation between endoscopists, morphologists and clinicians is an important condition for improving early gastric cancer diagnosis results.

Before the introduction of endoscopic methods of diagnosis into wide practice, stomach roentgenoscopy was almost the only method of diagnosing stomach cancer. Today, this method is inferior to endoscopy in the diagnosis of epithelial tumors of the stomach and retains its significance in the diagnosis of epithelial tumors. Therefore, this method also has the right to life. The disadvantages of the method are the radiation load on the patient and the impossibility of morphological verification of the detected changes.

TUMORS OF THE INTESTINE

Epithelial tumors are important among intestinal tumors - benign and malignant.

Among benign epithelial tumors, adenomas (in the form of adenomatous polyps) are most common. They are localized in the rectum, followed by frequency in the sigmoid, transverse colon, cecum and small intestine. According to the macroscopic appearance and histological structure, tubular, tubulovillous and villous are distinguished. Villous adenoma is a pink-red soft tissue with a villous surface (villous tumor), histologically has a glandular papillary structure; sometimes it becomes malignant. With multiple adenomatous polyps, we are talking about polyposis of the intestines, which can be familial.

Cancer can occur both in the colon and in the small intestine. Colon cancer has been occurring more and more recently; the mortality rate increases at the same time. Most often, cancer develops in the rectum, less often in the sigmoid, cecum, hepatic and splenic nodes of the transverse colon.

*Rectal cancer*develops against the background of chronic ulcerative colitis, polyposis, villous tumor or chronic fistulas, which are considered precancerous diseases.

*Cancer of the small intestine*it occurs less often, mostly in the duodenum in the area of its large (fater) nipple. The tumor does not reach significant sizes, causes difficulty in the outflow of bile, which is the cause of subhepatic jaundice and is complicated by inflammation of the bile ducts.

Depending on the nature of growth, exophytic, endophytic and transitional forms of cancer are distinguished.

Exophytic cancers include plaque-like, polypous and large-nodular; to endophytic - ulcerative and diffuse-infiltrative, often narrowing the lumen of the intestine; to transitional ones - saucershaped cancer.

According to the histological structure, adenocarcinoma, mucinous adenocarcinoma, ring-shaped cell, squamous cell, glandular-squamous, undifferentiated, unclassified cancer are distinguished. Exophytic forms of cancer are built according to the type of adenocarcinoma; endophytic - ringshaped cell or undifferentiated cancer.

Cancers of the anus are distinguished separately: squamous cell, cloacogenic, mucoepidermal, adenocarcinoma.

I t m e t a s t a s i z e s rectal cancer to regional lymph nodes and liver.

1. Theoretical questions Questions

for self-control:

1. General characteristics of tumor growth.
2. Tumors of the esophagus.
3. Types of stomach tumors.
4. Clinical and morphological characteristics of stomach tumors.
5. Tumors of the small and large intestine, morphological characteristics.

2 Practical tasks

50. Prepare an essay on the topic: "Tumors of the esophagus."

51. Make a graphological structure "Types of stomach tumors.".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

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- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
- http://www.webpathology.com/- Web Pathology

Topic #11: "Diseases of the biliary system and pancreas.»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:

The student should know:

41. the place of diseases of the organs of the hepatobiliary system and the pancreas in the structure of the general morbidity;

42. morphological classification, morphological characteristics of the most important diseases of the biliary system and pancreas.

The student should be able to:

41. explain the main etiological and pathogenetic mechanisms of the occurrence and development of diseases of the biliary system and pancreas;

42. interpret pathomorphological changes in the biliary system and pancreas with subsequent diagnosis of a specific disease.

43. The student should have an idea of general pathological processes that develop in the biliary system and pancreas in various pathologies.

Topic content:

DISEASES OF THE GALL BLADDER

Inflammation, formation of stones and tumors are observed in the gallbladder.

Acute cholecystitis (AC) - acute nonspecific inflammation of the gallbladder - is one of the most common urgent diseases of the gastrointestinal tract. In emergency surgery, GC ranks second in frequency after acute appendicitis and accounts for about 10-25% of all acute diseases of the abdominal cavity. The clinical course of GC is multifaceted and depends on a number of reasons, among which the degree of disruption of the bile passage by the cystic duct and choledochoma, the virulence of the infection, and the presence or absence of the pancreato-vesical reflex are the most important. Previous anatomical and functional changes in the gallbladder and adjacent organs, as well as the state of the patient's protective and regulatory mechanisms should be added to this course. In recent years, the proportion of elderly patients among patients with acute cholecystitis has significantly increased. They are characterized by a high frequency of development of destructive forms of cholecystitis and their complications with peritonitis. The atypical course of these patients is manifested mainly by the inconsistency of the clinical picture of the disease with the pathomorphological changes present in the gallbladder. In the clinical picture, symptoms of intoxication often appear in patients at the forefront, while pain and signs of peritonitis may not be expressed sharply. It is the significant frequency of GC, which has a tendency to grow, a significant percentage of its complicated forms, high mortality, that determine the relevance and significance of the GC problem at the current stage. In the clinical picture, symptoms of intoxication often appear in patients at the forefront, while pain and signs of peritonitis may not be expressed sharply. It is the significant frequency of GC, which has a tendency to grow, a significant percentage of its complicated forms, high mortality, that determine the relevance and significance of the GC problem at the current stage. In the clinical picture, symptoms of intoxication often appear in patients at the forefront, while pain and signs of peritonitis may not be expressed sharply. It is the significant

frequency of GC, which has a tendency to grow, a significant percentage of its complicated forms, high mortality, that determine the relevance and significance of the GC problem at the current stage.

Classification of acute cholecystitis

I. Depending on the presence of concretions
1. calculous
2. non-calculous (stone-free).

II. According to the depth of morphological changes (O.O. Shalimov, V.T. Zaitsev):
1. catarrhal
2. phlegmonous
3. gangrenous

III. According to the presence of complications:
1. not complicated
2. complicated (drops and empyema of the gallbladder, perforation of the gallbladder, perivesical infiltrate or abscess, mechanical jaundice, cholangitis, acute pancreatitis, hepatitis, peritonitis - local, diffuse or general, hepatorenal failure, liver abscess, pylephlebitis, biliary 4 sepsis, internal fistulas, acute obturation (stone) intestinal obstruction)

At*acute cholecystitis*inflammation is catarrhal, fibrinous, purulent (phlegmonous). Complication of acute cholecystitis – perforation of the bladder wall with subsequent development of peritonitis; in cases of closure of the bladder duct and accumulation of pus in the bladder cavity - empyema; purulent cholangitis and cholangiolitis, pericholecystitis with the formation of adhesions. *Chronic cholecystitis*is mostly a consequence of acute; at the same time there is atrophy of the mucous membrane, histiolymphocytic infiltration, sclerosis; often petrification of the bladder wall. Chronic cholecystitis: primary-chronic; chronic relapsing uncomplicated; chronic-recurrent complicated (stenosis of the bile ducts, septic cholangitis, pancreatitis, hepatitis, biliary cirrhosis, dropsy of the gallbladder, chronic peribladder abscess, chronic empyema of the gallbladder, sclerosis of the gallbladder, bilio-digestive fistulae, choledocho-bladder fistulas).

*Stones of the bile ducts and gall bladder*is a fairly common cause of gallstone disease, calculous cholecystitis.

There are three factors in the formation of gallstones: stagnation of bile, violation of the ratio of bile components and the presence of inflammation in the bile ducts. It is known that for the development of housing and communal services, the following are necessary: oversaturation of bile with cholesterol, biliary stasis and imbalance of substances that participate in the formation of cholesterol crystals. It has been established that when the ratio of cholesterol to phospholipids is below 1:13 (N=1:20), conditions arise under which cholesterol settles and bile becomes lithogenic.

As shown in numerous studies, the composition of gallstones includes a significant amount of organic and inorganic substances: cholesterol, bilirubin, bile acids, proteins, calcium and phosphorus salts, trace elements. Most gallstones are mixed in composition.

For practical purposes, all gallstones are divided into 4 groups: - Cholesterol, in which the amount of cholesterol in relation to other substances is 70-80%. Pure cholesterol stones, as a rule, are single, egg-shaped, 3 to 30 mm in size.

-Pigment, cholesterol is less than 30%, and the combination of calcium with bilirubin and calcium salts is 50-60%. They are black and brown.

-Calcium stones. They mainly include calcium salts. These stones are radiopaque.

-Mixed stones are stones that consist of cholesterol, pigments, and calcium salts in almost equal proportions. Most of them have a radiopaque coating.

Perforation of the wall of the bladder with a subsequent development of peritonitis is possible. In cases where a stone from the gall bladder descends into the hepatic or common bile duct and closes

the lumen, there is*subhepatic jaundice*. Sometimes gallstones are not the cause of inflammation or biliary colic, they are found accidentally during the autopsy of the deceased.

*Gallbladder cancer*mostly occurs against the background of calculous cholecystitis with localization in the neck or its bottom; by histological structure, it is an adenocarcinoma.

Stenosis of the region of the large duodenal papilla (VDS)

The development of VDS stenosis is facilitated by the removal of sand and small gallstones from the bile duct in the DPC. In accordance with clinical data and the results of operative studies, VV Vinogradov distinguished 3 degrees of stenosis of the VDS:

1) characterized by a compensated violation of bile duct patency and the absence of jaundice;
2) is accompanied by the expansion of the bile duct and the presence of a narrowing in its distal part, through which a probe with a diameter of 3 mm cannot be passed. Jaundice with this degree of stenosis has an unstable character;
3) is characterized by complete blockage of the bile duct and decompensation phenomena, which is clinically manifested by persistent mechanical jaundice.

Intraoperative damage to the extrahepatic bile ducts (EPB)

Damage to the bile ducts is very different both in nature and in consequences, which can vary from minor bile leakage to incurable strictures of the intrahepatic ducts. Large and small damages are distinguished. "Major damage" is a complete transection of the common bile duct, the common hepatic duct, or transection of the duct by more than 50% of its diameter. "Minor damage" - marginal damage to the PPZP (no more than 50% of the diameter), failure of the stump of the cystic duct, damage to small bile ducts (for example, Lyushka's ducts). Damage to the PPZP manifests itself in the form of biliary leakage, biliary hypertension, and their combination.

DISEASES OF THE PANTACRIC GLAND

Inflammatory or tumor processes most often occur in the pancreas.

Pancreatitis- inflammation of the pancreas - the course is acute or chronic.

*Acute pancreatitis*develops in case of violation of the outflow of pancreatic secretion (ductal dyskinesia), penetration of bile into the excretory ducts of the gland (biliopancreatic reflux), alcohol poisoning, nutritional disorders (overeating), etc. Morphological manifestations of changes in the gland are edema, the appearance of white-yellow areas of necrosis (fat necrosis), hemorrhages, suppuration, false cysts, sequestrations. With the predominance of hemorrhagic changes that become diffuse, we are talking about hemorrhagic pancreatitis; purulent inflammation - about acute purulent pancreatitis; necrotic changes - pancreatic necrosis.

*Chronic pancreatitis*may be a consequence of acute relapses. Its causes are also infectious diseases and intoxications, metabolic disorders, poor nutrition, diseases of the liver, gall bladder, stomach and duodenum. In chronic pancreatitis, as distinguished from acute, not destructiveinflammatory, but sclerotic and atrophic processes in combination with the regeneration of acinous cells and the formation of regenerative adenomas prevail. Sclerotic changes lead to impaired duct patency and the formation of cysts. Cicatricial deformation of the gland is associated with its calcification; at the same time, the gland decreases, acquires cartilaginous density. With chronic pancreatitis, manifestations of diabetes mellitus are possible. Death of patients with acute pancreatitis occurs from shock or peritonitis.

Pancreatic cancercan occur in any part of it (head, body, tail), but is more often observed in the head, where it looks like a dense gray-white knot; the latter compresses and then sprouts the ducts of the gland and the common bile duct. Such tumor growth causes disorders of the function of both the pancreas (pancreatitis) and the liver (cholangitis, jaundice). Tumors of the body and tail of the

pancreas often reach significant sizes, since they do not cause dysfunction of both the gland itself and the liver for a long time.

Pancreatic cancer develops from the epithelium of the ducts (adenocarcinoma) or from the epithelium of the parenchyma acini (acinar or alveolar).*It metastasizes*the tumor is lymphogenic in the lymph nodes located near the head of the gland, or hematogenous in the liver, lungs, and other organs. Patients die from exhaustion, tumor metastases or pneumonia that joins.

1. Theoretical questions Questions for self-control:

1. Acute and chronic cholecystitis: morphological characteristics, complications, causes of death.
2. Gallstone disease: types of stones.
3. Pancreatitis: general data, morphological changes of the pancreas.
4. Complication of acute pancreatitis
5. Pancreatic cancer: morphological characteristics.

2 Practical tasks

52. Prepare an abstract on the topic: "Pancreatitis: general data, morphological changes of the pancreas."

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
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Topic #12: "Peritonitis, adhesion disease.»

Purpose: as a result of independent study of this topic, students should know the topic, as knowledge of the topic is necessary in the practical activity of a doctor to understand the mechanisms of the occurrence and development of peritonitis, as well as knowledge of the topic is necessary for the interpretation of morphological data obtained when using biopsy and autopsy research methods.

Basic concepts:

The student should know:

43. Anatomical and physiological information about the peritoneum

44. General data on the etiology and pathogenesis of peritonitis.

45. Morphological classification of peritonitis

46. Complications and completion of peritonitis The student should be able to:

44. Explain the main etiological and pathogenetic mechanisms of the occurrence and development of peritonitis;
45. Interpret pathomorphological changes in the peritoneum and organs of the abdominal cavity with subsequent diagnosis of a specific disease.
46. To have an idea about the general pathological processes that develop in the organs of the abdominal cavity during inflammatory processes of the peritoneum.

Topic content:

Anatomical and physiological information about the peritoneum

Peritoneum is a serous membrane that lines the walls of the abdominal cavity (parietal peritoneum) and covers its organs (visceral peritoneum). Regarding the visceral peritoneum, the following organs are distinguished: intraperitoneal (stomach, small intestine, transverse colon and sigmoid colon); mesoperitoneal (liver, ascending and descending intestines); extraperitoneal (duodenum, pancreas, kidneys, main vessels). The area of the peritoneum is equal to the area of human skin, that is, from 1.5 to 2 m2. There are 6 layers of the peritoneum: 1) mesothelium – single-layer flat cells; 2) basement membrane; 3) superficial wavy collagen layer; 4) surface diffuse elastic network; 5) deep longitudinal elastic network; 6) deep lattice collagen elastic layer.

Functions of the peritoneum as a barrier or protective (mechanical) membrane. Protection mechanism: a) mechanical protection; b) cellular mechanism (macrophages); c) humoral mechanism (immunoglobulins).

Phagocytic cells of the peritoneum, together with T- and B-lymphocytes, phagocytize and destroy all foreign matter that has entered the abdominal cavity (microbes). The main "guard" of the

abdominal cavity is the large omentum. It has a large number of T-lymphocytes. The omentum covers the wound, perforation site, organ inflammation, etc. Exudative In one hour, the peritoneum secretes about 5% of the body weight, and in 1 day - 5-6 liters, which is antimicrobial in its activity. Resorptive (absorbing). The same amount of fluid is absorbed through the lymphatic and blood vessels together with bacteria, products of necrosis and protein lysis. As inflammation increases, the absorptive function of the peritoneum increases. These properties are most pronounced in the peritoneum of the diaphragm and cecum. Interoreception (pathological impulses). The parietal peritoneum is innervated by branches of the intercostal nerves (somatic innervation). There is a visceromotor reflex - defense, clearly localized somatic pains. This is important for self-diagnosis and diagnosis. The visceral peritoneum is innervated by parasympathetic and sympathetic nerves. Therefore, visceral pains are not localized, but diffuse. It is important to remember that with inflammation of the pelvic peritoneum there will be no defense, since there is no somatic innervation in this department. Plastic. Immediately after an injury (mechanical, chemical), fibrin appears on the peritoneum, it sticks together, in particular the loops of the intestine and the omentum, thanks to which the focus of inflammation is delimited. The visceral peritoneum is innervated by parasympathetic and sympathetic nerves. Therefore, visceral pains are not localized, but diffuse. It is important to remember that with inflammation of the pelvic peritoneum there will be no defense, since there is no somatic innervation in this department. Plastic. Immediately after an injury (mechanical, chemical), fibrin appears on the peritoneum, it sticks together, in particular the loops of the intestine and the omentum, thanks to which the focus of inflammation is delimited. The visceral peritoneum is innervated by parasympathetic and sympathetic nerves. Therefore, visceral pains are not localized, but diffuse. It is important to remember that with inflammation of the pelvic peritoneum there will be no defense, since there is no somatic innervation in this department. Plastic. Immediately after an injury (mechanical, chemical), fibrin appears on the peritoneum, it sticks together, in particular the loops of the intestine and the omentum, thanks to which the focus of inflammation is delimited.

Peritonitis

Peritonitis is an inflammatory lesion of the peritoneum that has a phase course, covers individual areas or the entire surface of the peritoneum, is accompanied by intestinal paresis, endogenous intoxication and disorders of homeostasis, against the background of which there are disorders of systemic and regional blood supply, pulmonary gas exchange, liver and kidney function. Peritonitis is a complication, or rather, an inevitable consequence or stage of development of various acute surgical diseases and injuries of the abdominal organs. In the modern classification according to the etiological structure, it is customary to distinguish primary, secondary and tertiary peritonitis.

Primary peritonitis is quite rare - in about 1% of all cases of peritonitis. It usually has a serious underlying disease (cirrhosis of the liver, chronic kidney failure, etc.).

Secondary peritonitis is the most common form of abdominal surgical infection, occurring in 80-90% of cases. The source of secondary peritonitis is destructive diseases or injuries of abdominal organs. Approximately 20% are complications after intra-abdominal operations. The problem of tertiary (or "reverse") peritonitis is separate. This form occurs mostly in severe, weakened patients who have undergone, as a rule, more than one operation on the organs of the abdominal cavity. As the main risk factors for tertiary peritonitis, it is customary to consider: digestive disorders (exhaustion) of the patient, a decrease in the concentration of plasma albumin, the presence of problematic pathogens resistant to most antibiotics (hospital strains), the development of organ failure. Tertiary peritonitis occurs as a result of the inability of the body's defense forces (both at the systemic and local levels) to form an adequate response to the infectious process. Clinically, these patients do not show bright peritoneal symptoms, but have signs of severe sepsis (multiorgan failure).

According to the clinical course, acute and chronic peritonitis are distinguished. The latter in most cases have a specific character: tuberculous, parasitic, cancerous, ascites-peritonitis, etc. In emergency surgery, acute peritonitis is often encountered as a complication of the destructive process in the abdominal cavity.

According to the nature of the exudate, serous, fibrinous, purulent peritonitis is distinguished, as well as intermediate forms - serous-fibrinous, fibrinous-purulent. Separately, biliary and fecal peritonitis are distinguished.

The greatest inconsistency among surgeons occurs when considering the classification of peritonitis by prevalence. In foreign literature, there are only two forms of peritonitis - local and total. In domestic literature, the prevalence of peritonitis is considered in more detail.

According to V.D. Fedorov (1974), the following forms are distinguished:

1. Local peritonitis: a). limited (inflammatory infiltrate, abscess); b). unlimited (there are no limiting adhesions, but the process is localized in one of the areas of the abdominal cavity). 2. Common: a). diffuse (the peritoneum is affected over a considerable length, but the process covers less than two floors of the abdominal cavity); b). spilled (affected peritoneum of more than two floors of the abdominal cavity); in). general (total inflammation of the visceral and parietal peritoneum).

Etiology. Secondary peritonitis is always a complication of diseases or injuries of abdominal organs. Currently, peritonitis is not considered as an independent nosological form. The main reason for its development is the entry of microflora into the abdominal cavity from various parts of the gastrointestinal tract. It is known that the microflora of hollow organs contains many aerobic and anaerobic species. Some of them die as soon as they get into the unusual environment in the abdominal cavity. However, when the exudate is properly examined for the presence of anaerobes and aerobes, as a rule, several types of bacteria from each group are isolated. Therefore, a priori

widespread purulent peritonitis should be considered polymicrobial, caused by anaerobic and aerobic flora.

Pathogenesis. Anatomical and functional features of the peritoneum explain the speed of development of the inflammatory process in the abdominal cavity, the severity of its course, and rapidly increasing intoxication. The greatest absorption capacity is inherent in the diaphragmatic surface of the peritoneum (absorption occurs through the stomata under the influence of negative pressure in the chest cavity). Therefore, intoxication with localization of the purulent process in the upper floor of the abdominal cavity is more pronounced than with a similar process in the cavity of the pelvis. The pathogenesis of peritonitis is extremely complex. It is known that the reactive phase of peritonitis in the absence of adequate treatment quickly turns into toxic and terminal. The reactive phase can last a very short time in case of sudden perforation of a hollow organ and is a normal reaction to severe painful irritation.

Pathomorphology. Leukocytes and macrophages, which phagocytose 80% of bacteria, play the main role in the fight against the microflora of the peritoneum. The result of the fight against the infectious environment that has entered the abdominal cavity depends on the speed of migration of these formed blood elements to it. They are viable for about two days, and then they disintegrate. One leukocyte, depending on phagocytic activity, as well as the number and virulence of bacteria, absorbs about 50 microbial bodies. After a few minutes, leukocytes form a leukocyte shaft around the source of infection. This is the first barrier. A rather sticky exudate appears, due to its bactericidal effect, about 5% of bacteria die. At first, it is bright yellow, without fibrin - as a result, serous local peritonitis occurs. The focus of inflammation is delimited by fibrin films. The granulation shaft is the second barrier. In the next 2 hours, adhesion (sticking) of the peritoneum occurs with the adjacent loop of the intestine and the large omentum, and after 18 hours, a loose infiltrate forms around the focus - the third most reliable barrier. For example, a periappendiceal infiltrate is formed on the 3rd–4th day, and thanks to adequate therapy, it can be resolved. With sufficient reactivity of the body, delimited peritonitis develops. However, when the barrier is insufficient, the body's defenses are exhausted, the exudates, and with it the microflora, spread throughout the abdominal cavity, occupying other areas (between 20 intestinal loops, in the pelvis, under the diaphragm). Therefore, the formed barriers are local protective factors that prevent the spread of infection. and after 18 hours, a loose infiltrate forms around the focus - the third most reliable barrier. For example, a periappendiceal infiltrate is formed on the 3rd–4th day, and thanks to adequate therapy, it can be resolved. With sufficient reactivity of the body, delimited peritonitis develops. However, when the barrier is insufficient, the body's defenses are exhausted, the exudates, and with it the microflora, spread throughout the abdominal cavity, occupying other areas (between 20 intestinal loops, in the pelvis, under the diaphragm). Therefore, the formed barriers are local protective factors that prevent the spread of infection. and

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With an unfavorable course, tissue necrosis occurs in the center of the focus, followed by their purulent melting and the formation of a purulent cavity. In case of inadequate treatment and destruction of the abscess wall, peritonitis occurs, which is even more malignant in nature than the one against which the abscess arose.

Adhesion disease

Adhesion disease can occur due to abdominal injuries, inflammation, as a result of taking certain drugs or congenital anomalies of development. But most often adhesions are formed due to surgical intervention. According to statistics, adhesion disease is the most common complication after cavity operations on the abdominal cavity. Disrupting natural mobility, adhesions interfere with the normal functioning of internal organs, cause spasms of ligaments and muscles. In gynecology, adhesions can block the lumen of the fallopian tube, causing infertility. Sometimes the patient practically does not feel the discomfort caused by the fusion, but often after operations on internal organs, acute intestinal obstruction occurs. Acute intestinal obstruction is the most severe manifestation of adhesion disease. This is a condition that requires immediate medical attention, and often emergency surgery.

1. Theoretical questions Questions for self-control:

53. Anatomical and functional information about the parietal and visceral peritoneum.
54. Etiology and pathogenesis of acute peritonitis.
55. Classification of acute peritonitis.
56. Pathomorphological picture of local acute peritonitis.
57. Pathomorphological picture of widespread peritonitis.
58. Complication of acute peritonitis.

59. Postoperative complications in acute peritonitis, their diagnosis, prevention and treatment. 60. Adhesion disease

2 Practical tasks

1. Prepare an essay on the topic: "Adhesion disease"
2. Make a graph of the logical structure "Classification of acute peritonitis.".
3. **Test tasks for self-control:**

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
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Topic #13: "Liver tumors.»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:

The student should know:

47. the place of diseases of the organs of the hepatobiliary system in the structure of general morbidity;

48. morphological classification, morphological characteristics of liver tumors.

The student should be able to:

47. explain the main etiological and pathogenetic mechanisms of the occurrence and development of liver diseases;

48. interpret pathomorphological changes in the liver with subsequent diagnosis of a specific disease.

49. The student should have an idea of general pathological processes developing in the liver.

Topic content:

LIVER CANCER

Liver cancer —relatively rare tumor. It usually occurs against the background of cirrhosis of the liver, which is considered a precancerous condition; Among the precancerous changes of the liver, dysplasia of hepatocytes is of great importance. In Asia and Africa—regions of the globe with a high incidence of liver cancer—the tumor often arises in the intact liver; regions with a low incidence of liver cancer are Europe and North America, where cancer develops in a cirrhotic liver. The following factors play a significant role in the development of primary liver cancer:

49. chronic viral hepatitis B(80% of patients with hepatoma). The risk of hepatocellular carcinoma in carriers of the virus increases 200 times. It is 50% higher in male carriers;
50. cirrhosis(especially the large-nodular form) is found in approximately 60-90% of patients with hepatoma;
51. hemochromatosis(excess retention of iron in the body); **52.** schistosomiasisand other parasitic diseases;
53. carcinogens:
61. industrial products - polychlorinated diphenyls, chlorinated hydrocarbon solvents (for example, carbon tetrachloride, nitrosamines); organic pesticides containing chlorine;
62. organic compounds (for example, aflatoxins contained in food products, for example, peanuts).

Pathological anatomy

Histologically distinguish:

Hepatocellular cancer(malignant hepatoma) - arises from liver cells. It occurs most often among liver tumors. It develops in the form of one, less often several tumor-like formations. Local invasive growth is characteristic, especially often the tumor grows into the diaphragm. Distant metastases are most often found in the lungs (up to 45% of cases).

Cholangiocellular cancer- arises from the cells of the epithelium of the bile ducts, accounts for 5 to 30% of all primary malignant liver tumors. Dense grayish tumor. Most often, it develops at the age of 60 to 70 years. It metastasizes to regional lymph nodes or to other parts of the liver.

Angiosarcoma(malignant hemangioendothelioma) arises from spindle-shaped cells lining the lumen of intrahepatic vessels. A rare vascular tumor (accounts for 2% of malignant liver neoplasms), one of the most malignant liver tumors. Most often, angiosarcoma occurs in men (in 85% of cases).

Angiosarcoma is characterized by spread to the spleen (in 80% of cases) and distant metastases in the lungs (in 60% of cases).

Morphological classification of liver cancer involves macroscopic form, nature and features of tumor growth, histogenesis and histological types.

According to the macroscopic appearance of the tumor, the following forms of liver cancer are distinguished: nodular - the tumor is in the form of one or several nodes; massive - the tumor occupies a massive part of the liver; diffuse cancer — the entire liver is occupied by numerous tumor nodes that merge with each other. Special forms include small and peduncular cancer.

Liver with cancer is dramatically enlarged (sometimes 10 times or more), its mass can reach several kilograms. With a nodular tumor, it is lumpy, moderately dense; with diffuse - even stony.

According to the nature of growth, liver cancer can be expansive, infiltrating and mixed (expansive-infiltrating). Features of liver cancer growth include sinusoidal growth and replacement growth.

Depending on the features of histogenesis, liver cancer is divided into:

1) hepatocellular (hepatocellular);
2) from the bile duct epithelium (cholangiocellular); 3) mixed (hepatocholangiocellular); 4) hepatoblastoma.

Among the histological types of liver cancer, the following are distinguished: trabecular, tubular, acinous, solid, clear cell. Each histological type has a different degree of differentiation.

Liver cancer metastasizes both immunogenically (portal lymph nodes, peritoneum) and hematogenously (lungs, bones).

Complicationand causes of death — hepatargia, hemorrhages in the abdominal cavity from disintegrating tumor nodes, cachexia.

1. Theoretical questions Questions for self-control:

1. Risk factors for the development of liver tumors
2. Classification of liver tumors
3. Pathomorphology of liver cancer: histological form, macroscopic forms
4. Ways of metastasis
5. Histological structure of metastatic liver cancer
6. Complications and causes of death in malignant liver tumors

2 Practical tasks

63. Prepare an essay on the topic: "Pathomorphology of liver cancer: histological forms, macroscopic forms."

64. Make a graphological structure "Paths of metastasis".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory http://www.webpathology.com/- Web Pathology

Topic #14: "Morphological features of epithelial tumors of individual organs.»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:

The student should know:

54. definition of the concept of "tumor";

55. morphological characteristics of tumors, signs of morphological atypism;

56. principles of tumor classification;

57. types of epithelial tumors according to existing principles of classification;

58. the biological essence of tumor growth.

The student should be able to:

50. to determine the morphological features of various types of atypism;

51. determine the type of tumor growth;

52. distinguish between mature and immature tumors on the basis of tissue and cellular atypism, the nature of tumor growth, in relation to the surrounding tissues, explain the probable cause and mechanism of development;

53. on the basis of morphological features, be able to distinguish benign and malignant tumors from flat and iron epithelium;

54. explain the features of metastasis of malignant tumors from the epithelium;

Topic content:

General doctrine about tumors

A tumor, neoplasm, blastoma is a pathological process characterized by uncontrollable cell proliferation. The growth and reproduction of cells in a tumor differs from the growth and reproduction of cells in other processes (inflammation, regeneration, hyperplasia, organization). Tumor cells are characterized by a state of anaplasia or cataplasia - partial loss of cell differentiation factors (due to biochemical rearrangement of DNA).

Most tumors have an organoid structure and consist of parenchyma and stroma. Tumors that consist of one tissue are called histoid. Parenchyma for them is specifically functioning tumor cells, and stroma - fibrous structures, amorphous substance, lymphatic and blood vessels, nerves. If the cells and parenchyma of the tumor are sufficiently differentiated and resemble the parent tissue, the tumor is called homologous, mature, benign. If the cells and parenchyma of the tumor are poorly differentiated and do not resemble the parent tissue, then such a tumor is called heterotypic, immature, malignant.

The morphological structure of tumors differs from normal tissues. The set of features that distinguish them is called atypical. Allocate tissue and cellular atypism: biochemical; antigenic atypism.

Tissue atypism consists of: 1. incorrect quantitative ratio of stroma and tumor parenchyma; 2. the presence in the tumor of various sizes and number of vessels; 3. incorrect, chaotic fiber direction; 4. weakening of the collagenization process; 5. discomplexation and formation of irregular structures.

Cellular atypism consists of: 1. cellular polymorphism, violation of the nuclear cytoplasmic index; 2. increase in the number and size of nuclei; 3. the presence of various cellular inclusions (protein grains, glycogen); 4. the presence of pathological figures of mitoses (multipolar, asymmetric, hyper- and hypochromic, abortive); 5. unusual number of chromosomes, chromosomal aberration; 6. presence of multinucleated giant cells; 7. close topographic contacts between the nuclear membrane and the mitochondrial membrane, between the membrane and the endoplasmic reticulum; 8. loss of membrane-forming and covering properties of the epithelium.

The atypicality of ultrastructures is manifested in an increase in the number of ribosomes, which can lie freely in the form of rosettes and chains, the shape, size and location of mitochondria are changed. Abnormal mitochondria appear. The cytoplasm is poor, the nucleus is large with a diffuse or marginal arrangement of chromatin. Membrane contacts of the nucleus, mitochondria and endoplasmic reticulum appear in large numbers. Hybrid cells appear.

According to the nature of growth, tumors are divided into expansive and infiltrating tumors. Expansive growing tumors preserve syncytial connections between tumor sites. With such growth, the tumor has a clear border that separates it from the tissues of the body.

Expansive growth is characteristic of benign, mature tumors. Appositional growth of a tumor occurs through neoplastic transformation of normal cells into tumor cells.

Infiltratively growing tumors (invasive growth) are characterized by deep penetration of tumor cells into the underlying tissue. This growth is a consequence of weak syncytial connections between tumor cells and is characteristic of malignant, immature tumors.

Exophytic growth of tumors - external growth or into the cavity of the organ (characteristic of mature tumors). Endophytic growth - the growth of a tumor deep into an organ or into its wall (more typical for immature tumors).

Clinical tumorsdivided into benign, malignant and tumors with local destructive growth.

Signs of immature, malignant tumors: 1. sharply expressed anaplasia and atypism; 2. a lower degree of differentiation compared to mature tumors; 3. weak syncytial connections between tumor cells; 4. rapid growth, infiltrating, invasive, endophytic; 5. tumors can recur and metastasize.

Metastasis of a tumor is manifested in the fact that tumor cells enter other places through embolism and begin to multiply there, forming daughter nodes (metastases). There are hematogenous (characteristic for sarcoma), lymphogenic (characteristic for cancer), and mixed metastases. Most often, in a metastasis, the tumor has the same structure as in the main node. However, tumor cells in metastases can change, and then it is difficult to establish the nature and localization of the primary node based on the histological structure of the metastasis. Metastases, as a rule, grow faster than the primary node. The time of development of metastasis can be different.

Tumor recurrence - the appearance of a tumor in the same place where it was removed surgically or by radiation. It is possible from cancer cells that remained in the wound, as well as from nearby metastases.

In addition, it should be taken into account that when removing a tumor, not the cause is eliminated, but the result.

The effect of a tumor on the body can be local and general. Local influence: compression or destruction of an organ or its part. The general effect is characteristic of malignant tumors:
metabolic disorders, the development of cachexia, some tumors are hormonally active.

Epithelial tumors

Epithelial tumors originate from the covering or glandular epithelium.

A papilloma is a mature benign tumor of the covering epithelium.

Adenoma is a mature benign tumor of the glandular epithelium.

Carcinoma, or cancer, is an immature malignant tumor of the covering or glandular epithelium.

Tumors of this type develop from flat or glandular epithelium, which does not perform any specific function.

Papilloma- epithelial tumor from flat or transitional epithelium. Tissue atypism is manifested in uneven development of epithelium and stroma with excessive formation of small blood vessels. Macroscopically, a papilloma looks like a node with a papillary surface. The consistency of the node can be dense or soft, depending on the ratio of stroma and parenchyma. Microscopically, the tumor consists of papillae. Papillomas, as benign formations, are characterized by pronounced tissue atypism with weakly expressed cellular atypism. Papillomas of the larynx, skin, and bladder have the greatest clinical significance.

Adenoma- a tumor of glandular organs and mucous membranes lined with prismatic epithelium. Adenoma has the appearance of a well-separated node of soft-elastic consistency. Adenomas have an organoid structure, consist of cells of prismatic or cubic epithelium, which forms iron formations, sometimes with papillary growths. If the stroma predominates, it is called a fibroadenoma. Epithelium preserves complexity and polarity, located on its own membrane.
Adenomas are found on mucous membranes and endocrine glands. Often, adenomas have hormoneproducing properties. The microscopic structure of an adenoma is represented by a glandular component surrounded by stroma.

According to the ratio of these components, adenomas are divided into:

1. Simple adenoma
2. Fibroadenoma
3. Adenofibroma

Depending on the histological structure, adenomas are divided into:

4. Alveolar
5. Tubular
6. Trabecular
7. solid
8. Cystic

Malignant, immature tumors from the epithelium - cancer.

Cancerusually has the form of a node of a soft or dense consistency with indistinct borders. A cloudy liquid - cancer juice - separates from the cut surface of the tumor. Sharply expressed tissue and cellular atypism is determined microscopically.

Histological variants of cancer:

Microscopic forms of cancer:

1. "cancer in place";
2. squamous cell carcinoma with and without keratinization;
3. adenocarcinoma;
4. mucous (colloid);
5. solid;
6. small cell;
7. fibrous (skirr);
8. medullary (adenogenic).

1. "Cancer in place" - the initial form of cancer without invasive growth, but with pronounced atypism. This is only the initial stage of cancer growth. Then it becomes infiltrating.

2. *Squamous cell cancer*consists of cords of atypical epithelial cells that grow into the underlying tissue. Tumor cells can retain the ability to keratinize and then "cancer pearls" appear.

3. Adenocarcinoma (glandular cancer) develops from the prismatic epithelium of mucous membranes and glandular epithelium. Its histological structure resembles an adenoma, but in contrast to it, there is a sharply expressed tissue and cellular activity. Tumor cells form iron formations that grow into the surrounding tissue, destroying it.

4. Mucous (colloid cancer). The tumor has the appearance of a colloidal mass. Consists of atypical cells.

5. Solid cancer grows in the form of trabeculae separated by layers of connective tissue. It grows quickly and metastasizes.

6. Small cell cancer is a form of undifferentiated cancer that consists of monomorphic lymphocyte-like cells that do not form any structures. There is little stroma. Growth is fast. Early metastases.

7. Fibrous cancer (skirr) - a form of undifferentiated cancer, which consists of atypical hyperchromic cells located among the layers and masses of the stroma, which prevails over the parenchyma. The tumor is highly malignant. There are often early metastases.

8. Medullary - undifferentiated cancer. The main quality is the superiority of parenchyma over stroma. The tumor is soft. It consists of atypical cells, has a large number of metastases, grows quickly, gives early and numerous metastases.

Tumors of exo- and endocrine glands and epithelial coverings (organ-specific) are characterized by the fact that they develop from cells of a certain organ and retain morphological and sometimes functional features.

Liver. Benign organ-specific tumor from hepatocytes - adenoma from liver cells (hepatoadenoma, hepatoma). Malignant hepatocellular (hepatocellular) cancer. The tumor consists of atypical hepatocytes that form irregularly arranged trabeculae of an irregular shape. The stroma is weakly expressed.

KidneysBenign organ-specific tumors - various adenomas, malignant - renal cell cancer.

Skin. Benign tumors: syringoadenoma (from the epithelium of sweat gland ducts); hydroadenoma (from the epithelium of the secretory departments of sweat glands); trichoepithelioma (from hair follicles). Malignant tumors: basal cell cancer, sweat gland cancer, sebaceous gland cancer, hair follicle cancer.

Mammary gland. Benign tumors: fibroadenoma (pericanalicular, intracanalicular). Organspecific breast cancer includes: non-infiltrating intralobular and intraductal cancer: Paget's disease (nipple and areola cancer).

Uterus.Organ-specific epithelial tumors of the uterus are de-structuring (malignant) insertion of the bladder and chorion epithelioma (chorion carcinoma). Insertion of the bladder is manifested by the ingrowth of chorionic villi into the veins of the uterus and pelvis. Chorionepithelioma develops from the epithelium of the villi of the chorion and consists of light Langhans cells and syncytium cells. There is no stroma in the tumor. The function of blood vessels is performed by cavities formed by tumor cells. Gives hematogenous metastases.

Tumors of salivary glands and organs of the oral cavity. Adenomas of the salivary glands are quite common. They consist of epithelial structures in combination with fibrous, lacrimal, cartilaginous, and bone-like structures. They are benign, can recur, turn into cancer.

Odontoma develops from the enamel organ, consists of dense tooth tissues (mainly dentine). There are soft and hard odontomas. Benign flow. Relapses are possible.

Ameloblastoma develops from the epithelium of the enamel organ. A tumor of a soft consistency, consisting of delicate connective tissue and ducts lined with cubical and prismatic epithelium, may have a cystic structure. It proceeds benignly, tends to give relapses. Localization - jaw bones.

1. Theoretical questions Questions for self-control:

General doctrine about tumors.
Nomenclature of epithelial tumors. Histological variants of cancer.
Morphological features of benign (papilloma, adenoma) tumors from the epithelium without specific localization: and malignant (cancer).
Morphological features of malignant (cancer) tumors from the epithelium without specific localization
Tumors of exo- and endocrine glands and epithelial covers.

2 Practical tasks

65. Prepare an abstract on the topic: "Morphological features of malignant (cancer) tumors from the epithelium without specific localization"

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory http://www.webpathology.com/- Web Pathology

Topic No. 15: "Pathomorphological changes in diseases related to nutrition. Vitamins Radiation sickness. Occupational diseases..»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:

The student should know:

59. principles of classification of occupational diseases;

60. principles of diagnosis of occupational diseases;

61. peculiarities of the development of occupational diseases depending on the cause;

62. characteristic morphological signs of occupational diseases.

63. peculiarities of vitamin metabolism, pathomorphology of vitamin deficiency.

64. causes and features of the development of radiation sickness and its morphological features.

The student should be able to:

55. recognize the characteristic morphology of a number of occupational diseases during microscopic examination;

56. evaluate the research results and describe them in the study album;

57. make drawings after studying micropreparations in accordance with the instructions in the study album;

58. predict possible complications.

Topic content:

Avitaminosis

Vitamins are part of food products and are important for the normal functioning of the body. Insufficiency or lack of vitamins of both exogenous and endogenous origin lead to the development of a number of pathological processes and diseases (hypo- and vitamin deficiency). As a result of insufficiency or absence of vitamins, the following develop most often: rickets, scurvy, xerophthalmia, pellagra, deficiency of vitamin B^ and folic acid.

RICKETS

Rickets —a consequence of hypo- or vitamin D deficiency.

There are several forms of rickets:

1) classical form in children of different ages (from 3 months to 1 year — early rickets; from 3 to 6 years — late rickets);

2) vitamin-0-dependent rickets — a hereditary disease with an autosomal recessive type of transmission;

3) vitamin D-resistant rickets — a hereditary sex-linked (X-chromosome) disease; 4) rickets in adults, or osteomalacia.

The classic form deserves the most attentionrickets in childhood and rickets in adults.

Etiology. The cause of rickets is caused by a deficiency of vitamin D. The origin of this deficiency can be: 1) hereditary; 2) as a result of insufficient ultraviolet radiation, necessary for the formation of vitamin D3 in the body; 3) in connection with the insignificant intake of vitamin D with food; 4)

impaired absorption of vitamin D in the intestines; 5) increased need for the vitamin with its normal intake into the body; 6) chronic diseases of the kidneys and liver, in which the formation of the active metabolite of vitamin D3 — 1.25 $(OH)_2O_3$ — is disturbed. In D-avitaminosis in adults, a violation of vitamin absorption due to diseases of the gastrointestinal tract and an excessive need for vitamin D, for example, during pregnancy, hyperthyroidism, renal acidosis, etc., is of great importance.

Pathogenesis. At the heart of the disease are deep disturbances in the metabolism of calcium and phosphorus, which leads to a violation of the calcification of the osteoid tissue, which loses the ability to accumulate calcium phosphate. This is explained, first of all, by the fact that with rickets, the content of inorganic phosphorus in the blood decreases (hypophosphatemia), the intensity of oxidative processes in tissues decreases with the subsequent development of acidosis. With rickets, protein and fat metabolism is also disturbed, while fatty acids have a ricketsstimulating effect.

Pathological anatomy. In children with early rickets, morphological changes are most pronounced in the bones of the skull, at the junctions of the cartilage and bone parts of the ribs, and in the metaesphyseal sections of long tubular bones, that is, in places with the most intensive growth of the skeleton. Round or oval softenings (craniotabes) appear in the bones of the skull, primarily in the occipital-parietal regions, and periosteal growths (osteophytes) appear in the area of the frontal and parietal humps. At the same time, the child's head acquires a quadrangular shape (sarii; diaotaiit). The size of the fountains increases sharply, they close late. At the joints of the cartilage and bone parts of the ribs, thickenings appear (especially noticeable on the inner surface of the VI, VII and VIII ribs), which have received the name "rickets". Epiphyses of long tubular bones become thickened - "rachitic bracelets".

In the places of enchondral ossification, the germinal zone expands sharply, and it turns into a "rachitic zone", the width of which is proportional to the severity of rickets. In the area of enchondral ossification, an excess of cartilage and osteoid tissue is formed, and calcification does not occur in the latter. Cartilage cells are arranged randomly. Osteoid tissue accumulates not only enchondrally, but also endo- and periosteally, which leads to the development of osteophytes. The cortical layer of the diaphyses thins due to lacunar resorption of the bone; it becomes less elastic and bends easily. Due to the excessive formation of osteoid tissue, which is not capable of calcification, the formation of a full-fledged bone is delayed. Sometimes microfractures of individual bone beams are possible,

In late-onset rickets in children, disorders of endosteal ossification prevail, not enchondral. Bones, especially of the lower limbs and pelvis,subject to deformation, the shape of the chest and spine changes.

In early and late rickets, anemia, enlargement of the spleen and lymph nodes, muscle atony, especially of the abdominal wall and intestines ("frog's belly") are observed.

With rickets in adults (osteomalacia), bone changes are the result of impaired calcification of new bone structures and excessive formation of osteoid tissue.

Complicationin children with rickets, pneumonia, eating disorders anddigestion, as well as purulent infections.

SCURVY

Scurvy(synonyms: scurvy, Barlow's disease) — vitamin deficiency S.

Etiology and pathogenesis. The disease occurs in the absence of vitamin C (ascorbic acid) in food or insufficient absorption. The disease manifests itself most clearly when, along with vitamin C, vitamin P is excluded from food. Insufficient intake of vitamin C in the body disrupts the function of redox enzymes and leads to significant changes in carbohydrate and protein metabolism. The increased formation of melanin and excessive pigmentation of the skin is associated with the disorder of the oxidation of aromatic amino acids (tyrosine and phenylalanine). With an insufficient amount of vitamin C, the condition of the main substance of connective tissue, collagen synthesis, fibrillogenesis, maturation of connective tissue is disturbed, which is associated with an increase in vascular and tissue penetration. It increases especially sharply with a combination of vitamin C and P deficiency. In such cases, the hemorrhagic syndrome is most pronounced. Disturbances and delays

in collagen formation also explain the changes in bone tissue in scurvy, which are manifested by inhibition of proliferative processes in the areas of the most intensivebone growth and remodeling.

Pathological anatomy. Morphological changes in scurvy consist of manifestations of hemorrhagic syndrome, bone changes and complications associated with secondary infection.

*Hemorrhagic syndrome*manifests itself equally in both children and adults. Hemorrhages appear on the skin, mucous membranes, internal organs,bone marrow, under the periosteum, in the joint cavity (hemarthrosis). Ulcers appear on the skin and mucous membranes.

Bone changes in children and adults have the same manifestation. In children, they become leaders in the picture of the disease and are expressed in depressionbone formation In the germinal zone of tubular bones, the replacement of cartilaginous structures by bone slows down, the compact layer of diaphyses becomes thin, fractures easily occur. Hemorrhages in the region of the germinal growth zone lead to the separation of the epiphysis from the diaphysis (epiphysiolysis). Bone marrow is replaced by fibrous tissue. In adults, bone changes appear mainly at the border with the cartilaginous part of the ribs, where chondroplastic bone growth continues until 40-45 years of age. Here, the bone beams become thinner, the bone marrow is replaced by fibrous tissue, fibrin and blood accumulate, then the cartilaginous part of the rib can separate from the bone, the sternum in such cases sinks.

The skin with scurvy becomes dark due to the accumulation of melanin in it.

Complicationassociated mainly with the attachment of a secondary infection that develops in areas of hemorrhage. Stomatitis and gingivitis appear, teeth loosen and fall out easily; ulcerative and necrotic processes occur on the tongue and tonsils (ulcerative glossitis, phlegmonous and gangrenous angina). As a result of possible aspiration, pneumonia, abscesses or gangrene of the lungs develop; sometimes joins tuberculosis. Enteritis and colitis are possible.

XEROPHTHALMIA

Xerophthalmia— a disease that is a consequence of vitamin A deficiency.

Etiology and pathogenesis. Avitaminosis A can be of exogenous and endogenous origin and is caused by a number of reasons: its insufficient amount in food, impaired absorption of both vitamin A and fats in the intestines, excessive use of this vitamin in some pathological processes and diseases. It is known that vitamin A determines the condition of the epithelium and the synthesis of rhodopsin. With a deficiency of vitamin A, metaplasia of the prismatic and transitional epithelium takes place into a keratinized, multi-layered flat one. When the synthesis of rhodopsin is disturbed, hemeralopia (chicken blindness) appears. Metaplasia of the prismatic epithelium of the respiratory tract, especially the trachea and bronchi, is often observed in measles and influenza, which is largely associated with endogenous vitamin A deficiency. Manifestations of endogenous vitamin A deficiency can also be observed in other infectious diseases (for example,

Pathological anatomy. Changes in xerophthalmia are characterized by epithelial metaplasia and secondary inflammation of mucous membranes. Epithelial metaplasia into stratified stratum corneum is particularly evident in the conjunctiva of the eye and the cornea. At the same time, tear glands atrophy and their secretion decreases. There is dryness of the cornea and conjunctiva, which become whitish. The transparency of the cornea decreases sharply, dystrophic and necrotic changes occur in its tissue (keratomalacia). Metaplasia of the epithelium is also observed in the mucous membranes of the respiratory (nasal passages, trachea, bronchi) and urinary tracts, in the vagina, uterus, prostate and pancreas. Inflammatory and ulcerative processes easily occur on mucous membranes changed in this way. Healing of ulcers and wounds in patients withVitamin A is significantly delayed.

PELLAGRA

Pellagra— a chronic disease that occurs when the body lacks nicotinic acid (vitamin PP) and other B vitamins.

Etiology and pathogenesis. Pellagra develops when there is a deficiency in the bodyonly nicotinic acid and other vitamins, but also tryptophan. A significant loss of nicotinic acid by the body is

observed when there is insufficient protein in food products. Deficiency of nicotinic acid becomes the cause of disruption of redox processes, which is accompanied by the development of both dystrophic and atrophic changes.

Pathological anatomy. Morphological changes develop mainly in the skin, nervous system and intestines. Erythema with swelling appears on the skin of exposed parts of the body, which are gradually replaced by hyperkeratosis and atrophy, the skin becomes dry and acquires a brown color. During histological examination, in addition to atrophy and hyperkeratosis, cellular infiltrates around the vessels of the dermis, dystrophic changes in sweat glands and nerve fibers are found. Excessive production of melanin is found in the basal layer of the skin. Dystrophic changes develop in the nervous system, primarily in various areas of the brain (motor cortex, midbrain, cerebellum), spinal cord, and peripheral nerves. With a long course, dystrophic changes develop mainly in the conduction system of the spinal cord. Atrophy of the mucous membrane, cystic expansion of the glands are found in both the small and large intestines, ulceration of follicles with successive epithelization of ulcers. Atrophicchanges also develop in the stomach, liver and pancreas.

VITAMIN B12 AND FOLIC ACID DEFICIENCY

With a deficiency of vitamin B12 and folic acid, various forms of anemia develop.

PNEUMOCONIOSIS

Pneumoconiosis - dust diseases of the lungs. The term "pneumoconiosis" was proposed by Zenker in 1867. Industrial dust refers to the smallest particles of a solid substance that are formed during an industrial process, which, falling into the air, are suspended in it for a more or less long time. A distinction is made between inorganic and organic dust. Inorganic dust includes quartz (which consists of 97-99% free silicon dioxide), silicate, and metal dust. Organic includes plant (flour, wood, cotton, tobacco, etc.) and animal (cotton, hair, etc.) products. There is mixed dust, for example, containing coal, quartz and silicate dust in various proportions, or iron ore dust consisting of iron and quartz dust. Particles less than 5 μm in size that penetrate deep into the lung parenchyma pose the greatest danger. The shape and consistency of dust particles and their solubility in tissue fluids are of great importance. Dust particles with sharp edges injure the mucous membrane of the respiratory tract. Fibrous dusts of animal and plant origin cause chronic rhinitis, laryngitis, tracheitis, bronchitis, pneumonia. During the dissolution of dust particles, chemical compounds are formed that have an irritating, toxic, and histopathogenic effect. They have the ability to cause the development of connective tissue in the lungs, that is, pneumosclerosis.

When dust of various composition enters the lungs, lung tissue can react differently.

The reaction of lung tissue can be:

- inert, for example, with ordinary pneumoconiosis - anthracosis of coal miners;
- fibrosing, for example, with massive progressive fibrosis, asbestos with silicosis;
- allergic, with exogenous allergic pneumonitis;

- non-plastic, for example, mesothelioma and lung cancer with asbestosis. Localization of the process in the lungs depends on the physical properties of the dust. Particles less than 2-3 μm in diameter can reach the alveoli, larger particles are retained in the lungs and nasopharynx, from where they can be removed from the lungs by mucociliary transport. An exception to this rule is asbestos, whose particles of 100 microns can settle in the terminal parts of the respiratory tract. This is because the asbestos particles are very thin (about 0.5 microns in diameter). Dust particles are phagocytosed by alveolar macrophages, which then migrate to the lymphatic vessels and go to the basal lymph nodes.

Classification. Anthracosis, silicosis, metalloconiosis, carboconiosis, pneumoconiosis from mixed dust, pneumoconiosis from organic dust are distinguished among pneumoconiosis.

ANTHRACOS

Inhalation of coal dust is accompanied by its local accumulations, which are invisible until massive pulmonary fibrosis is formed. The accumulation of coal in the lungs, which is referred to as "pulmonary anthracosis", is typical for residents of industrial cities. It can be observed practically in

- the amount of inhalable silicon and quartz, as well as the type of coal (bituminous coal is more dangerous than wood coal);
- co-infection with a tubercle bacillus or atypical mycobacteria;
- the development of a hypersensitivity reaction due to the death of macrophages and the release of antigens;
- the development of fibrosis associated with the deposition of immune complexes.

But none of the theories have been proven, and some researchers believe that the determining factor is only the amount of absorbed dust. At the end of the disease, the lungs have the appearance of honeycombs, the formation of the pulmonary heart is observed. Patients die either from pulmonary and heart failure, or from joining intercurrent diseases.

Silicosis

Silicosis is a disease that develops as a result of long-term inhalation of dust containing free silicon dioxide. Most of the earth's crust contains silica and its oxides. Silicon dioxide is present in nature in three different crystalline forms: quartz, cristobalite and tridymite. Uncombined forms of silicon dioxide are called "free silicon", and combined forms containing cations make up various silicates. Silica dust is found in most industrial productions, in particular in gold, tin and copper mines, during cutting and grinding of stones, during the production of glass , in metal smelting, in the production of pottery and porcelain. In all these industries, the size of the particles matters. Sand usually contains 60% silicon oxide. But its particles are so large that they can reach the periphery of the lungs. Only small particles, entering the bronchioles and alveoli, can cause their damage. Silicon, especially its particles with a size of 2-3 nm, is a powerful stimulator of the development of fibrosis. The amount and duration of exposure to silicon also play a large role in the development of silicosis. Approximately 10-15 years of work in industrial dust conditions without respirators can cause silicosis.

If the concentration of dust is significant, then its acute form may occur in 1-2 years, "acute" silicosis. In some cases, the disease may appear several years after the end of exposure to industrial dust (late silicosis). The risk group for the given disease includes the workers mentioned above professions The amount and duration of exposure to silicon also play a large role in the development of silicosis. Approximately 10-15 years of work in industrial dust conditions without respirators can cause silicosis.

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Pathogenesis. Now the development of silicosis is associated with chemical, physical and immune processes that occur when dust particles interact with tissue. At the same time, the value of the mechanical factor is not excluded.

According to modern ideas, the pathogenesis of silicosis includes the following stages:

- inhalation of silicon particles with a diameter of less than 2 μm with their penetration into the terminal parts of the airways;
- absorption (phagocytosis) of these silicon particles by alveolar macrophages;
- death of macrophages;
- release of the contents of dead cells, including silicon particles;
- repeated phagocytosis of silicon particles by other macrophages and their death; appearance of fibrous hyalinized connective tissue;
- possible development of further complications.

The exact nature of the factor or factors causing fibrosis is still unknown. Unlike coal dust, silicates are toxic to macrophages and lead to their death with the release of proteolytic enzymes and unchanged silicate particles. Enzymes cause local tissue damage with subsequent fibrosis; silicate particles are again absorbed by macrophages and the cycle repeats endlessly. According to this theory, the leading role in the pathogenesis of silicotic fibrosis is the death of koniophages with the subsequent stimulation of fibroblasts by macrophage decay products. It is believed that hydrogen bonds between the released silicic acid formed when it is absorbed by macrophage lysosomes and the phospholipids of the phagosome membrane lead to membrane rupture. Rupture of the phagosome membrane leads to the death of macrophages. All macrophage derivatives formed able to stimulate fibroblastic proliferation and activation of fibrillogenesis. Since plasma cells and immunoglobulins are found in the places of impression, participation in fibrillogenesis and immune reactions is assumed, but the mechanism of their development in silicosis has not yet been clarified. According to the immunological theory, during the influence of silicon dioxide on tissues and cells, during their disintegration, auto antigens appear, which leads to auto immunization. The immune complex arising from the interaction of an antigen with an antibody exerts a pathogenic influence on the connective tissue of the lungs, resulting in the formation of a nodule. But no specific antibodies were detected. but the mechanism of their development in silicosis has not yet been clarified. According to the immunological theory, during the influence of silicon dioxide on tissues and cells, during their disintegration, auto antigens appear, which leads to auto immunization. The immune complex arising from the interaction of an antigen with an antibody exerts a pathogenic influence on the connective tissue of the lungs, resulting in the formation of a nodule. But no specific antibodies were detected. but the mechanism of their development in silicosis has not yet been clarified. According to the immunological theory, during the influence of silicon dioxide on tissues and cells, during their disintegration, auto antigens appear, which leads to auto immunization. The immune complex arising from the interaction of an antigen with an antibody exerts a pathogenic influence on the connective tissue of the lungs, resulting in the formation of a nodule. But no specific antibodies were detected.

Pathological anatomy. With chronic silicosis, atrophy and sclerosis are found in the mucous membrane and submucosal layer of the nasal cavity, larynx, and trachea. In humans, the histological evolution of silicosis lesions is not well known, since an advanced form of the disease is detected at autopsy. According to the study of silicosis in animals and in the case of an acute disease, the following has been established. The first response to the appearance of silicon in the acinus is the accumulation of macrophages. If the pollination is massive, then macrophages fill the lumen of the bronchioles and the surrounding alveoli. It is possible to develop a serous inflammatory reaction, similar to what can be observed in alveolar proteinosis. In some cases, the described picture is similar to gray hepatization of the lungs in case of croup pneumonia. With the slow development of the process in the early stages, multiple small nodules are found in the lung tissue, mainly in the upper parts and in the portal area, which give the lung parenchyma a fine-grained appearance, as if the tissue

is covered with sand. During this period, granulomas are formed, which are mainly represented by macrophages surrounded by lymphocytes and plasmocides. These granulomas are found around bronchioles and arterioles, as well as in paraseptal and subpleural tissues. In the process of evolution, the size of the nodules increases, some of them merge and then they are visible to the naked eye. The nodules become larger and denser, and then large areas of the lungs turn into scar layers, separated from each other by areas of mixed emphysema. Pleural leaves grow together with dense scar moorings.

In the lungs, silicosis manifests itself in the form of two main forms: nodular and diffuse-sclerotic (or interstitial).

With the nodular form, a significant number of zygotic nodes are found in the lungs, which are small paired or larger sclerotic areas of round, oval or irregular shape, gray or gray-black in color (coal miners). With severe silicosis, the nodes merge into large silicotic nodes, occupying a large part of the fate or even the entire fate. In such cases, they speak of a tumorous form of pulmonary silicosis. The nodular form occurs with a high content of free silicon dioxide dust and with prolonged exposure to dust.

With the diffuse-sclerotic form, typical silicotic nodes in the lungs are absent or there are very few of them, they are often found in their bifurcation lymph nodes. This form is observed when inhaling industrial dust with a small content of free silicon dioxide. In this form, the connective tissue in the lungs grows in the alveolar membranes, peribronchially and perivascularly. Diffuse emphysema, bronchial deformation, various forms of bronchioles and bronchitis (more often catarrhal-desquamative, less often purulent) develop. Sometimes a mixed form of pulmonary silicosis is found. Silicotic nodes can be typical and atypical. The structure of typical silicone nodes is twofold: some are formed from concentrically arranged hyalinized bundles of connective tissue and therefore have a rounded shape, others are not rounded and consist of bundles of connective tissue, that go like a vortex in different directions. Atypical silicotic nodes have irregular shapes, they lack concentric and vortex-like arrangement of bundles of connective tissue. In all nodes, there are many dust particles lying freely or in macrophages, which are called dust marks or coniophages. Silicotic nodes develop in the lumen of the alveolar passages, as well as in the place of lymphatic vessels. Alveolar histiocytes phagocytize dust particles and turn into coniophages. During long-term and strong pollination, all dust cells are removed, so their accumulations are formed in the lumens of the alveoli and alveolar passages. Collagen fibers appear between the cells, a cell-fibrous nodule is formed. Gradually, dust cells die, the number of fibers increases, resulting in the formation of a typical fibrous node. Similarly, a silicotic nodule is formed at the site of a lymphatic vessel. In silicosis, in the center of large silicotic nodules, connective tissue disintegrates with the formation of silicotic caverns. Disintegration occurs as a result of changes in blood vessels and the nervous system of the lungs, as well as as a result of the instability of the connective tissue of silicotic nodules and nodes that differ in biochemical composition from normal connective tissue. Silicone connective tissue is less resistant to the action of collagenase compared to normal. A lot of quartz dust, diffuse sclerosis and silicotic nodules are found in lymph nodes (bifurcations, basal, less often in paratracheal, cervical, supraclavicular). Occasionally, silicotic nodules are found in the spleen, liver, and bone marrow. In silicosis, in the center of large silicotic nodules, connective tissue disintegrates with the formation of silicotic caverns. Disintegration occurs as a result of changes in blood vessels and the nervous system of the lungs, as well as as a result of the instability of the connective tissue of silicotic nodules and nodes that differ in biochemical composition from normal connective tissue. Silicone connective tissue is less resistant to the action of collagenase compared to normal. A lot of quartz dust, diffuse sclerosis and silicotic nodules are found in lymph nodes (bifurcations, basal, less often in paratracheal, cervical, supraclavicular). Occasionally, silicotic nodules are found in the spleen, liver, and bone marrow. In silicosis, in the center of large silicotic nodules, connective tissue disintegrates with the formation of silicotic caverns. Disintegration occurs as a result of changes in blood vessels and the nervous system of the lungs, as well as as a result of the instability of the connective tissue of silicotic nodules and

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ASBESTOS

The word "asbestos" comes from the Greek word "indestructible". About 6 million tons of this mineral are extracted annually in the world. Asbestos contains many fibrous minerals consisting of hydrated silicates. Asbestos fibers give a double refraction of the beam in polarized light, which can be used in microscopic diagnosis. They are often found in combination with silicates. In these cases, they contain calcium, iron, magnesium and sodium. According to most authors, the carcinogenicity of asbestos depends not only on its type, but on the length of the fibers. Thus, fibers larger than 5 microns do not have carcinogenic properties, while fibers smaller than 3 microns have a pronounced carcinogenic effect. The risk of lung cancer in patients with asbestosis increases approximately 10 times, and in the case of smokers, then 90 times. Patients with asbestosis are twice as likely to have cancer of the esophagus, stomach, and colon. It is now proven that asbestos potentiates the action of other carcinogens. The onset of pneumoconiosis is different. It happens that pulmonary manifestations appear after 1-2 years of contact with asbestos, but most often - after 10-20 years. The pathogenesis of pulmonary fibrosis is unknown. Asbestos fibers have a small thickness (0.25-0.5 μm, so they penetrate deeply into the alveoli in the basal parts of the lungs. The fibers are found not only in the lungs, but also in the peritoneum and other organs. The fibers damage the walls of the bronchioles and alveoli, which is accompanied by shallow hemorrhages, which are the basis for the formation of hemosideria inside macrophages. Sets consisting of asbestos fibers are sometimes covered with protein, but most often with glycosaminoglycans, on which iron-containing grains of hemosiderin are deposited, were named "asbestos bodies". Under an optical microscope, they are reddish or elongated yellowish structures in the form of rings or strung pearls, resembling the appearance of "elegant dumbbells". In the electron microscope, their appearance is even more specific; their outer contours are represented by roughness resembling the steps of a ladder, and their axis contains parallel lines. These bodies (length 10-100 and width 5-10 μm) are found in sputum and help to differentiate asbestos from fibrosing alveolitis. Histologically, interstitial fibrosis is observed in the lungs. Macroscopically, the lungs in the late stages look like honeycombs. Fibrosis and emphysema of the lungs are found mainly in the basal parts of the lungs. Patients die from pulmonary

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Vibration diseaseoccurs in workers who use vibrating equipment in the course of their work: pneumatic hammers, machines for grinding and polishing metal and wooden products, for compacting concrete, asphalt road surfaces, driving\piles, and others. The disease is chronic. Workers develop a clinical and morphological picture of obliterating endarteritis. Vascular changes are accompanied by a violation of tissue trophism of the upper and lower extremities. Contractures of the fingers, deforming arthrosis, develop at the final stage of gangrene of the fingers, hands, and feet. Dystrophic changes up to the complete death of neurons are noted in the spinal cord. In the heads of the bones of the wrist, in the epiphyses of the radial and ulnar cysts, cystic foci of rarefaction and sclerosis are observed.

Diseases caused by exposure to radio frequency electromagnetic waves. Electromagnetic waves of radio frequencies are widely used in the field of radio (radio location, radio navigation, radio astronomy, radio linear communications, radio telephony, etc.), television, and during physiotherapeutic procedures. They distinguish:—microwaves (MKH), or ultrahighfrequency (UHF) with a wavelength from 1 mm to 1 m;—ultrashort waves (VHF), or ultrahighfrequency waves (UHF) with a wavelength of

distance up to 10 m;

— short waves (HF) or high frequency waves (HF) with a wavelength from 10 to 1000 m and more.

There have been no reports of acute deaths among lairds exposed to massive exposure to radio frequency electromagnetic waves.

Chronic exposurelow intensities of electromagnetic waves of radio frequencies of various ranges occurs in industry, workers of radio-television and radio relay stations, and residents of adjacent areas. The victims have damage to the function of the nervous, cardiovascular systems and gonads.

Morphological changes are detected in synapses and sensory nerve fibers of the receptor zones of the skin of internal organs. In the brain, the neurosecretory function of the neurons of the hypothalamic region is disturbed, which is accompanied by a steady drop in blood pressure. Fatty dystrophy of cardiomyocytes occurs in the myocardium. Dystrophic changes in the germinal epithelium up to its necrosis occur in testes. The most pronounced clinical and morphological changes are noted as a result of exposure to microwaves (MKH).

Diseases caused by exposure to industrial noise (noise disease)

Noise disease refers to persistent, irreversible morphological changes in the hearing organ caused by exposure to industrial noise

With an acute overpowering effect of noise and sounds, the death of the spiral (Corti's) organ, rupture of the eardrums, and bleeding from the ears are observed.

With chronic exposure to industrial noise, atrophy of the spiral organ with its replacement by fibrous connective tissue is observed. Changes in the sensitive nerve may be absent. Stiffness is observed in the joints of sensitive bones.

Meteosensitivity and diseases caused by atmospheric pressure.

Weather sensitivityis the organism's reaction to the influence of meteorological (weather) factors. Meteosensitivity is quite widespread and occurs under any, but more often unusual for a given person climatic conditions. About a third of the inhabitants of temperate latitudes "feel" the weather. The peculiarity of these reactions is that they occur in a significant number of people simultaneously with the change in meteorological conditions or slightly ahead of them. Meteosensitivity has long caused surprise and even fear of man before an incomprehensible phenomenon of nature. People who sense the weather were called "living barometers", "storm forecasters", "weather prophets".

Already in ancient times, doctors guessed about the influence of weather on the body. In Tibetan medicine, it is indicated that "pain in the joints increases in the rainy season and in the period of high winds." Paracelsus wrote: "He who has studied the winds, lightning and weather knows the origin of diseases."

The manifestation of weather sensitivity depends on the initial state of the organism, age, the presence of any disease and its nature, the microclimate in which a person lives, and the degree of his acclimatization to it. Meteosensitivity is more often noted in people who are rarely in the fresh air, engaged in sedentary, mental work, who do not engage in physical education. It is in them that the zones of the socalled microclimatic comfort are narrowed. As a rule, meteorological fluctuations are not dangerous for a healthy person. With a sudden change in weather conditions, it becomes harder for them to concentrate. Hence, the number of accidents may increase. As a result of diseases (influenza, sore throat, inflammation of the joints, joint diseases, etc.) or overfatigue, the body's resistance and reserves decrease. That is why weather sensitivity is noted in 35-70% of patients with various diseases. So, every second patient with diseases of the cardiovascular system feels the weather. Significant atmospheric changes can cause overstrain and disruption of adaptation mechanisms. Then the oscillatory processes in the body — biological rhythms — are distorted, become chaotic. Physiological (asymptomatic) weather reaction can be compared to a calm lake, on which waves are flowing from a light breeze. A pathological (morbid) weather reaction represents a kind of vegetative "storm" in the body. Disturbances in the regulation of the autonomic nervous system contribute to its development. The number of vegetative disorders has recently been increasing, which is connected with the effect of adverse factors of modern civilization, stress, haste, hypodynamia, overeating and malnutrition, etc. In addition, the functional state of the nervous system is far from the same in different people. This determines the fact that that diametrically opposite weather reactions, favorable and unfavorable, are often noted for the same diseases. More often, weather sensitivity is observed in people with a weak (melancholic) and strong unbalanced (choleric) type of nervous system. People of a strong balanced type (sanguines) are sensitive to the weather only when the body is weakened.

The body is affected by both the weather as a whole and its individual components. Fluctuations in barometric pressure act in two ways: they reduce blood oxygen saturation (the effect of barometric "pits") and mechanically irritate the nerve endings (receptors) of the pleura (the mucous membrane that lines the pleural cavity), the peritoneum (which lines the abdominal cavity), the synovial membrane of the joints, and also vascular receptors. On the European territory of the country, atmospheric pressure is most variable in the Baltic region, in the northwest and north. It is here that weather sensitivity is most often noted in patients with cardiovascular diseases. The wind causes disturbances in the nervous system, irritating skin receptors. In recent years, the study of the influence of weather conditions on organism, the so-called "syndromic meteopathology", which includes the symptoms of meteopathy caused by the combined effect of barometric pressure and atmospheric anomalies, such as thunderstorms, hot and dry winds, fogs, snowfall, etc. So, for example, the syndrome of the midday wind in France; the syndrome of the southwest wind in Switzerland, the syndrome of the northern winds (nords) blowing on the Absheron peninsula (Baku), according to various scientists, affect the health of approximately 75% of the population of these areas. They provoke angina attacks with coronary heart disease

Air humidity plays a role in maintaining the density of oxygen in the atmosphere, affects heat exchange and sweating. Patients with hypertension and atherosclerosis are especially sensitive to high humidity. In most cases, exacerbation of diseases of the cardiovascular system occurs at high relative humidity (80-95%). For many people, rainy days leave an impression even on their appearance, often the face becomes pale. Sudden temperature changes cause outbreaks of acute respiratory infectious diseases. In January 1780, the air temperature in St. Petersburg rose from -44° to +6° during one night, as a result, about 40,000 residents fell ill. A significant increase in cases of acute respiratory diseases was noted in Tashkent in November 1954, when the air temperature dropped from 4-15° to -21°. In addition, a sharp north wind blew, which raised masses of drops of water, sand and microbes that were in them into the air, outbreaks of infectious diseases arose in the city. An excess of positive aerophones, which is observed in hot and humid weather, has an adverse effect on the body, which can cause an exacerbation of heart diseases. In recent years, great importance has been attached to changes in solar activity and the Earth's magnetic field (geomagnetic disturbances and storms). their effect on the body is revealed 1-2 days before the weather changes, while other meteorological factors affect directly before or during the passage of air masses (cyclone or anticyclone). Unusual persistent weather, as a rule, also has an adverse effect on the body. In November 1977, warm, humid weather with heavy fog persisted for a long time in the city of Saratov. It had an oppressive effect on that raised masses of water droplets, sand and microbes contained in them into the air, outbreaks of infectious diseases arose in the city. 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There are three degrees of weather sensitivity. A mild degree is only a subjective malaise. With severe weather sensitivity (moderate degree), pronounced objective shifts are noted: changes in blood pressure, electrocardiogram, etc. With a severe degree of meteosensitivity, sharply expressed violations are observed, it is manifested by five types of meteopathic reactions. With the cardiac type, there are pains in the region of the heart, shortness of breath. The cerebral type is characterized by headaches, dizziness, noise and ringing in the head. The mixed type is a combination of cardiac and nervous disorders. In the asthenoneurotic type, increased excitability, irritability, insomnia, changes in blood pressure are noted. There are people who cannot clearly localize the manifestation of weather sensitivity. This is an unspecified reaction type: generalweakness, pain and aches in joints, muscles, etc.

The nature and magnitude of damage caused by atmospheric pressure depends on the magnitude (amplitude) of atmospheric pressure deviations and, mainly,from the speed of its change.

Decompression sickness most often occurs in divers (during deep-sea diving), in pilots, workers in caissons (caisson disease) as a result of saturation of blood and body tissues with nitrogen or helium and other gases during a person's stay in a high-pressure zone with its subsequent decrease - decompression . The saturation of the tissues of the body with nitrogen or helium in the zone of increased pressure continues until the pressure of these gases in the inhaled air equalizes with theirpressure in tissues. This process usually lasts several hours, and different tissues are saturated with nitrogen or helium at different rates. Blood, for example, is saturated faster than adipose tissue, but the latter dissolves nitrogen 5 times more than blood and other tissues. Saturation of tissues with nitrogen at a pressure of up to 4 at. h (when observing the rules for creating increased pressure) does not have an adverse effect on the body. However, during a rapid transition from a high-pressure zone to a low-pressure zone, excess dissolved nitrogen does not have time to be excreted through the lungs, which results in the transition of blood and tissue gases from a dissolved state to a gaseous state with the formation of bubbles.

The immediate cause of decompression sickness is the blockage of blood vessels by gas bubbles or their suppression of adjacent tissues. Concomitant factors are of significant importance: hard physical work of cooling the body, injuries, etc.

Symptoms of the disease most often appear within the first hour after leaving the high pressure zone, but often much later. The disease is manifested by skin itching, joint and muscle pain. The most

severe clinical symptoms occur when the blood vessels of the brain, lungs and other vital organs are blocked by gas bubbles?

When brain vessels are damaged, dizziness, stupor, vomiting, weakness, fainting, sometimes paresis and paralysis are observed. When pulmonary vessels are damaged, chest pains and a sharp cough occur. Depending on the severity of the disease, death can occur either a few minutes after decompression or withinone day to three weeks.

With the rapid onset of death, there is a strong emphysema of the subcutaneous tissue of the trunk, neck, and face. Crepitation is audible when palpating the skin (resembles the crunch of snow underfoot). Due to the presence of gas in the blood vessels and uneven blood filling of the vessels of the hemomicrocirculatory channel, the skin acquires a marble appearance. The blood collected in the veins remains liquid (due to hypoxia) and acquires a foamy appearance. During the microscopic examination of the internal organs in the vessels, an abundance of air bubbles (gas embolism) is noted. Swelling, perivascular hemorrhages, interstitial emphysema are found in the lungs, fatty dystrophy in the liver. There are multiple small ischemic foci of gray softening in the brain and spinal cord.

With long-term exposure to elevated atmospheric pressure, foci of rarefaction with perifocal sclerosis are found in the tubular bones, deforming osteoarthritis in the joints.

The most effective method of treatment is decompression, i.e. increasing the pressureits subsequent slow decrease."

Radiation damage

Radiation is energy contained in electromagnetic waves and particles. The types, frequencies and biological effect of electromagnetic radiation are summarized in the table. About 80% of radiation comes from natural sources, including cosmic rays, ultraviolet light, and natural radionuclides, especially radon gas. The other 20% arise from various man-made sources: sources of radio and microwave radiation, nuclear, power plants, etc. Though. that the pathological effect of high doses of radiation is probably proven, the effect of low dosessometimes it turns out to be the exact opposite. Electromagnetic radiation is divided into ionizing and non-ionizing.

Non-ionizing radiation includes radiation with a long wavelength and a low frequency of radio waves, microwave, ultraviolet and infrared radiation, visible light. This radiation will lead to vibration and rotation of the atoms of biological molecules. Short-wave radiation can ionize andknock out electrons.

Ionizing and non-ionizing electromagnetic radiation

X-ray, gamma and cosmic radiation are classified as ionizing radiation. There is also radiation of elementary particles: alpha or beta electrons, neutrons, mesons and neutrinos. The energy of these particles is measured in megaelectronvolts (MZV).

The dose of ionizing radiation is measured in the following units: — X-ray: the dose of ionizing radiation, when acting on 1 cm of air ions carrying a charge of one electrostatic unit will be formed; — rad: dose of radiation under the influence of which 1 gram of tissue absorbs 100 Erg; —gray (Gy): radiation dose under the influence of which 1 kg of tissue absorbs 1 J of energy; —ber: radiation dose that produces a biological effect is equal to the action of 1 rad

X-ray or gamma radiation. — sievert (Zv): the dose of radiation that produces a biological effect is equal to effects of 1 Gy of X-ray or gamma radiation; 1 Sv is equal to 100 Ber. **Cellular mechanisms of radiation damage**

The acute effect of the lesion can vary from pronounced necrosis at high doses (>10 Gy), death of proliferating cells at medium doses (from 1 to 2 Gy) to the absence of histopathological effect at doses of less than 0.5 Gy. At such low doses, intracellular structures, especially DNA, are damaged; however, adaptive and reparative response mechanisms to low doses of radiation are activated in most cells. Delayed (late) effects of ionizing radiation can be observed in surviving cells: mutations, chromosomal aberrations, genetic instability. These genetically damaged cells can become the basis for the emergence of malignant tumors; fast-growing tissues are most severely affected. Most tumors

are induced by ionizing radiation with a power of more than 0.5 Gy. Acute cell death, especially endothelial, can lead to delayed organ dysfunction several months or even years after exposure to radiation. In general, this delayed damage occurs as a result of several pathological processes: atrophy of parenchymal organs, ischemia as a result of vascular damage and fibrosis. Acute and delayed effects of ionizing radiation are presented in the table and described below.

Acute effects. Ionizing radiation can cause various types of DNA damage: the formation of crosslinks in DNA proteins, cross-links between DNA chains, oxidation and destruction of bases, destruction of carbohydrate-phosphate chains, breaking one and two DNA chains. These damages can occur both as a result of the direct action of elementary or short-wave radiation particles, and as a result of the action of free radicals and soluble substances formed during lipid peroxidation. Acute injuries and delayed complications under the action of ionizing radiation.

Body	Acute injury	Delayed injury
Bony	Atrophy.	Hypoplasia, leukemia
Skin	Erythema	Atrophy of the epidermis and fibrosis, dermis: cancer
Heart		Interstitial fibrosis
Lungs	Edema, death of epithelial and endothelial cells	Interstitial and intraalveolar fibrosis; cancer
gastrointestinal tract	Edema, mucosal ulcersshell	Ulcers; fibrosis; strictures; cancer
Liver	Veno-occlusive diseases	Cirrhosis; liver tumors
Kidneys	Vasodilatation	Atrophy of the cortical substance, Interstitial fibrosis
Bladder	Erosions of the mucous membrane	Submucosal fibrosis; cancer
Mainbrain	Edema, necrosis	White matter necrosis, gliosis brain tumors
Testicle	Necrosis	Tubular atrophy
Ovary	Atresia of follicles	Fibrosis of the stroma
Thyroid	-	Hypothyroidism; cancer
Mammary gland	-	Fibrosis; cancer
Thymus, lymph nodes	Atrophy	Lymphoma

Acute disturbances in the genetic apparatus of cells occur even under the influence of small doses (less than 0.5 Gy). Such damages include increased expression of c-IO5, c^shi and c-tus proto-

oncogenes, induction of cytokines, such as tumor necrosis factor (TNF), and activation of antioxidant protective enzymes, for example, superoxide dismutase. Free radicals, which are formed directly or indirectly under the action of ionizing radiation, can lead to the development of "oxidative stress", which leads to the activation of the transcription of some substances that enhance the synthesis of various proteins. DNA damage itself causes increased synthesis of proteins involved in DNA repair, cell division arrest, and apoptosis. As is known, the p53 tumor suppressor gene is activated in various types of DNA damage: its protein product changes into an activated form as a result of post-translational transformation. Under its influence, the cell cycle stops, DNA repair is activated, and if the integrity of DNA cannot be restored, the mechanism of apoptosis is triggered.

*Fibrosis.*An important late complication under the influence of ionizing radiation, usually in the doses used for radiotherapy of tumors, is the replacement of normal parenchymal tissue by fibrous tissue, which leads to scarring of the organ and disruption of its function. These fibrotic changes can develop both as a result of acute cell necrosis in organs with incomplete regeneration, and as a result of ischemic damage due to damage to blood vessels. In addition, in the mammary gland and lungs during irradiation, damaging cytokines and growth factors that contribute to sclerosis are released, which persist for several weeks after irradiation.

*Carcinogenesis.*As a result of exposure to ionizing radiation, the risk of various malignant tumors increases, especially skin cancer, leukemia, osteogenic sarcomas, and lung cancer. The disease most often develops 10-20 years after exposure. Thus, Japanese survivors of the atomic bombings of Hiroshima and Nagasaki had an increased incidence of all types of leukemia, except for chronic lymphocytic leukemia. In children, there was an increased incidence of cancer of the mammary and thyroid glands andto a lesser extent - cancer of the urinary tract and urinary organs.

The mechanism responsible for late carcinogenesis is still poorly understood. The long latent period between exposure to radiation and the development of cancer is attributed by some to the occurrence of the so-called induced genetic instability. Quantitative analysis of mutated genes in irradiated cells showed that pathological genes can be transmitted in the population of cells for several generations.

Disorders of growth and development. The embryo and the child's body are very sensitive to ionizing radiation. The greatest sensitivity is observed in the following 4 phases of development:

1. Embryo implantation. When the mother's body is irradiated before implantation, the embryo dies.
2. Critical phases of embryogenesis. When the mother's body is irradiated, even with for diagnostic purposes, from the moment of implantation to the 9th week of pregnancy is observed a large number of different developmental disorders, which in most cases are fatal. During this period, the greatest susceptibility is observed not only to radiation, but also to other teratogenic factors.
3. Fatal period. From the 9th week to the end of pregnancy, exposure to ionizing radiation leads to impaired development of the central nervous system and reproductive organs. Ischemic damage, atrophy and fibrosis are observed in the organs supplied with blood through the affected vessels.

Skin. Hair follicles and epidermis are most sensitive to the effects of ionizing radiation. Desquamation of the epidermis is often observed, its foci are replaced by atrophic epidermis with hyperkeratosis, hyper- or hypopigmentation. Vessels can thin and expand, they are often surrounded by dense bundles of collagen fibers. Impaired wound healing, increased sensitivity to infections and ulceration are observed. These changes are called contact dermatitis. As already mentioned, skin cancer, especially basal cell and squamous cell, can develop 20 or more years after exposure.

Heart. The heart and pericardium are often damaged as a result of radiation therapy in the chest area for lymphomas, lung and breast cancer. Fibrosis of the pericardium leads to the development of constructive pericarditis. Less often, myocardial ischemia and, as a result, cardiosclerosis develop as a result of damage to the coronary arteries.

LungsLungs are easily damaged by ionizing radiation. Acute pulmonary insufficiency often develops, in the later Term — radiation pneumonia. They develop both intra-alveolar and interstitial fibrosis. The risk of lung cancer is much higher in smokers, because there is a synergistic effect of these two factors in carcinogenesis. In cigarette smoke, in addition to carcinogenic substances, two radionuclides are detected: Pb210 and Po210. Sometimes in minesdetect Ka222. These miners often have a mutation (guanine -> thymidine) at codon 249 in p53 gene suppressor tumors.

Kidneys and bladder. Kidneys are moderately susceptible to radiation damage. They gradually develop peritubular necrosis, damage to blood vessels, hyalinization of glomeruli, which will ultimately lead to hypertension and atrophy of the kidneys. Acute necrosis of the epithelium can be observed in the bladder, then submucosal fibrosis, contractures, bleeding, and ulceration develop.

Gastrointestinal tract. As a result of exposure to ionizing radiation, there is a delay in the neuropsychological development of children. The risk of childhood leukemia and tumors also increasesnervous tissue.

Postnatal period. When irradiated in childhood, there is a violation of growth and differentiation of bone tissue. The development of the nervous system, eyes and teeth can also be disturbed.

Congenital mutations. On Ogoizorpiia flies and mice it was proved that mutations thatarise under the action of ionizing radiation, can be inherited. Despite the fact that chromosomal aberrations in blood cells are found in people who survived the atomic bombing and workers of nuclear power plants, such changes are not found in their descendants. Geneticists believe that some recessive mutations can still be passed on to offspring and accumulate in the population. However, a clear relationship between the number of mutations in human germ cells and the received dose was not found.

Delayed manifestations of exposure. After several months or years, late complications may occur (carcinogenesis was discussed above). As a result of such complications, the normal function of vital organs may be disturbed: lungs, heart, kidneys, central nervous system. Infertility can also develop in both men and women. Vision may be impaired due to the development of cataracts, and intestinal obstruction is also sometimes observed as a result of the growth of connective tissue in the intestines. Fibrous structures and chronic ulcers can be observed on the skin, in the gastrointestinal tract, bladder, and vagina. Chronic disorders in small vessels and excessive formation of connective tissue can complicate various surgical interventions. Healing is often disrupted earlier, they develop infectious processes.

Blood vessels. After the initial inflammatory reaction, accompanied by necrosis of the endothelial cells, subendothelial fibrosis, fibrosis of the muscular membrane, destruction of the internal elastic membrane, significant narrowing of the lumen of the vessel develops in the blood vessels in the irradiated area. Capillaries can become thrombosed, obliterated or, conversely, dilated (capillary ectasia). INabsorption of electrolytes and fluid. As a result of vascular damage, ischemia, ulceration, and atrophy of the mucous membrane occur. As a result of fibrosis, structures can develop that lead to intestinal obstruction.

Mammary gland. Even diagnostic chest X-rays can increase the risk of developing breast cancer. Radiotherapy of breast cancer leads to the development of a pronounced fibrotic reaction with a high polymorphism of epithelial cells.

Ovaries and testicles. Spermatogenic cells are very sensitive to radiation; even small doses can lead to disruption of meiosis and infertility. As a result of sclerosis of blood vessels, fibrosis of seminiferous tubules is observed, while Sertoli cells and interstitial cells of Leydig are not damaged. Follicles in the ovary are rapidly destroyed.

Eyes and nervous system. The lens is unstable to the action of ionizing radiation, cataracts often develop in it. The vessels of the retina and ciliary body are often damaged. Foci of necrosis and demyelination of nerve fibers can develop in the brain. As a result of irradiation of the spinal cord, sclerosis of blood vessels occurs in it, which leads to necrosis of cells, demyelination of fibers and, as a result, paraplegia. This process is called transverse myelitis.

Ultraviolet radiation

Sunlight contains radiation with wavelengths from 200 to 4000 nm, including ultraviolet, visible, and infrared. Depending on the wavelength, ultraviolet radiation is divided into three types - UV-A, UV-B and UV-C (see Table 24.4). Ultraviolet radiation makes up from 3 to 5% of the total flow of sunlight that penetrates to the earth's surface. The Earth's ozone layer plays a very important role because it completely absorbs UV-C and partially UV-B. Ordinary glasses, which completely absorb UV-B, but pass UV-A, also play a protective role against ultraviolet radiation. Ultraviolet radiation has two main effects: it accelerates skin aging and increases the risk of skin cancer.

Acute changes under the influence of UV-A and UV-B are reversible and disappear quickly. These include erythema, pigmentation, and damage to Langerhans cells and keratinocytes in the skin. At the same time, the mechanisms and mediators involved in the process differ depending on the type of radiation. Depending on the duration of exposure, erythema, swelling and acute inflammation occur as a result of the release of histamine from smooth cells in the dermis and the synthesis of arachidonic acid metabolites. When exposed to UV-B, interleukin 1 is also released. When exposed to UV-Athere is a rapid temporary darkening of melanin as a result of its oxidation, which is most pronounced in people with dark skin. Tanning under the influence of UV-A and UV-B occurs as a result of an increase in the number of melanocytes, the elongation and spread of their appendages, and the transfer of melanin to keratinocytes. Tanning determines the resistance of the skin to UV-B and partly to UV-A. Both UV-A and UV-B lead to the destruction of Langerhans cells and, as a result, to disruption of immune processes in the skin. UV-B causes apoptosis of keratinocytes, while "sunburn cells" appear in the epidermis, not in make-up keratin.

Repeated exposure to ultraviolet radiation leads to the appearance of signs of aging in the skin (wrinkles, solar elastosis, uneven pigmentation). Unlike ionizing radiation, which activates tissue collagenization, ultraviolet radiation leads to the destruction of elastin and collagen, which results in the formation of wrinkles and a decrease in skin elasticity. These changes are irreversible. The reason for this process is an increase in the activity of the elastin gene and synthesis of metalloproteases that destroy collagen. As a result, enzymatic destruction of type I collagen occurs.

Damage to the skin under the influence of UV-B occurs as a result of the formation of active oxygen-containing substances and damage to natural pigments, for example, melanin. Also, ultraviolet radiation leads to DNA damage, which manifests itself in the form of formation of pyrimidine dimers between adjacent pyrimidine bases in the same DNA strand. Pyrimidine-pyrimidone-(6-4)-phosphoproducts, breaks in one of the DNA strands, and crosslinks in DNA proteins can also be formed. When studying the genetic apparatus of skin cancer cells, the same changes are often found in the p53 gene: the replacement of T by T or DTC by TT. These observations confirm the role of ultraviolet radiation in the development of skin cancer.

Electromagnetic poles

Non-ionizing electromagnetic poles can have frequencies from 1 Hz to 100 Hz (microwave radiation from radars). There is evidence that exposure to loud noise with a frequency of only 5060 Hz increases the risk of leukemia in children. There are reports of an increased incidence of leukemia and brain tumors among electricians working on high-voltage power lines.However, these facts were not proven in various experiments on animals.

1. Theoretical questions Questions for self-control:

1. Define the concept of occupational diseases.
2. Classification of occupational diseases.
3. Clinical and morphological manifestations of diseases associated with insufficient and excessive nutrition.
4. Clinical and morphological manifestations of pneumoconiosis.
5. Clinical and morphological manifestations of atmospheric pressure disorders.

6. Morphological manifestations of diseases under the influence of industrial noise.
7. Morphological manifestations of diseases under the influence of electromagnetic waves and radio frequencies.
8. Vibration disease and its pathomorphology.
9. Morphological manifestations in the human body under the action of electricity.
10. Morphology of injuries from temperature effects, burns, heat stroke, hypothermia.
11. Pathomorphology of acute and chronic radiation sickness.
12. Iatrogenic pathology, medicinal disease, morphological characteristics.

2 Practical tasks

66. Prepare an abstract on the topic: "Clinical and morphological manifestations of pneumoconiosis.."

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
- http://www.webpathology.com/- Web Pathology

Topic #16: "Quarantine infections.»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:

The student should know:

65. definition of "infection", "infectious process", "infectious disease".

66. principles of classification of quarantine diseases

67. morphology of local and general changes in internal organs during quarantine diseases

68. pathomorphosis of quarantine diseases.

The student should be able to:

59. define quarantine diseases

60. explain the morphological features of quarantine diseases

61. conduct a macro- and microscopic examination of organs with signs of quarantine diseases

62. to systematize the main signs specific for each individual type of quarantine diseases

Topic content:

ESPECIALLY DANGEROUS INFECTIONS (THEY)

ONI is a group of acute contagious human diseases that can appear suddenly, spread quickly and cover the population en masse; are characterized by a severe course and high mortality.

The THEY group includes:

1) plague, cholera, smallpox, yellow fever (these are the so-called conventional diseases subject to the International Sanitary Regulations, the former name is quarantine infections);

2) typhus and relapsing typhus, influenza, poliomyelitis, malaria (diseases subject to international supervision);

3) AIDS, anthrax, thrush, melioidosis, tularemia, brucellosis, rickettsiosis, ornithosis, arbovirus infections, botulism, histoplasmosis, blastomycoses (diseases subject to regional or national surveillance).

Cholera

Cholera is an acute infectious disease from the group of diarrheal diseases with predominant damage to the stomach and small intestine, which is characterized by a general serious condition and dehydration of the body. Cholera, like the plague, belongs to the number of

particularly dangerous diseases, to the group of quarantine or conventional diseasesinfections and extremely contagious.

The causative agent of cholera – Vibrio cholerae (cholera vibrio), isolated by R. Koch in 1884 is a gram-negative bacillus in the form of a coma.

Cholera belongs to anthroponoses. A person becomes infected from cholera patients, as well as from bacterial carriers that secrete vibrios with feces, and sick people - when vomiting. Infection occurs when drinking water, less often food products contaminated with secretions containing vibrios (when eating vegetables grown in fields and gardens that are fertilized with non-disinfected sewage, when washing dishes with contaminated water). Flies contribute to the spread of pathogens. Infection is possible while swimming in contaminated reservoirs. Some serotypes of Vibrio cholera, for example, Vibrio El-Tor, are able to live in the body of frogs and oysters. In these cases, human infection can occur indirectly, in the absence of patients.

The incubation period ranges from several hours to 5 days (more often 2-3 days).

Pathogenesis. After overcoming the acid barrier of the stomach (cholera was induced in volunteers only after neutralization of gastric juice), Vibrio cholerae enters the small intestine. Vibrios never penetrate the epithelium of the mucous membrane, and huge accumulations of the pathogen are contained in the lumen of the small intestine and secrete enterotoxin, which is encoded by a virulent phage. According to its chemical characteristics, the enterotoxin of Vibrio cholera is almost identical to the enterotoxin of E. Coli.

Pathomorphology. Structural changes are most pronounced in the small intestine. The lumen of the intestine is sharply expanded, filled with a colorless or pink liquid, sometimes stained with bile. This liquid looks like rice broth. The watery nature of bile in the gallbladder is also described. The wall of the intestine, in particular its mucous membrane, is swollen, with red spots (multiple small point hemorrhages). These changes are designated as serous-hemorrhagic cholera enteritis. Microscopically, sharp swelling, including intracellular, and full blood prevail, small hemorrhages are also noted. With a relatively mild course, there are also significant accumulations of cells, among which lymphocytes and plasma cells predominate. Enteritis may be accompanied by serous or serous-hemorrhagic gastritis. With cholera gastroenteritis, the phenomena of enteritis increase, epithelial cells become vacuolated, desquamation of microvilli is observed. The serous membrane of the intestine is dry, with dotted hemorrhages, matte, painted in a pinkish-yellow color.
Transparent, sticky mucus in the form of threads appears between the loops of the small intestine.

Profuse diarrhea leads to a rapid loss of water and electrolytes (sodium, potassium, bicarbonates), and dehydration leads to hypovolemic shock and metabolic acidosis, blood thickening and hypoxia, increasing oliguria and a drop in body temperature, which characterizes algid (from lat. algor - cold) cholera period Progressive exicosis and electrolyte imbalance play a leading role in the occurrence of cholera coma.

The acute period of cholera may end: —

fatal outcome;

— recovery;
— sometimes with the development of the picture of cholera typhoid.

It is believed that cholera typhoid is not a stage of the disease caused by sensitization, as was previously believed, but a manifestation of the complication of this disease as a result of the addition of secondary, mainly bacterial microflora. At this time, the main changes are already developing in the large intestine. Fibrinous inflammation of the mucous membrane occurs in it, ulcers may form later.

At the autopsy, there is a sharp pronounced cadaveric emaciation, which persists for several days. The body of the deceased acquires an unusual shape, which is designated as "gladiator's pose". As the cadaver embalming disappears, the position of the corpse may change, which creates the impression of its movement. Such a movement of the corpse in space caused panic to the relatives of the dead and the servants of the cholera barracks. They believed that the dead from cholera were buried alive. This was the reason for the so-called "cholera riots" in the past century.

As a result of the rapid onset of cadaveric tanning, the skin resembles goosebumps, it is dry, wrinkled, especially on the fingers ("washerwoman's hands"). The muscles are dry, dark red. The blood in the veins is thick and dark. The spleen is reduced, its capsule is wrinkled, the follicles are atrophic, hemosiderosis of the pulp is noted. Dystrophy of hepatocytes and focal necrosis of the parenchyma develop in the liver. In the kidneys, necrosis of the epithelium of convoluted tubules is noted. There are dystrophic and necrobiotic changes in the myocardium and brain.

Complication. With cholera, the development of necrotic nephrosis is possible, as well as rapidly progressing glomerulonephritis, which are accompanied by uremia. In addition, cholera patients develop pneumonia, abscesses, phlegmon, dysentery, and sepsis.

DeathCholera patients mainly suffer from dehydration, coma, intoxication. Death is also possible from complications of cholera, among which uremia is the most common. With the massive introduction of liquid solutions of sodium bicarbonate into the body, mortality decreases from 50% to 1%.

Plague

Plague (pestis) is an acute infectious disease, which is characterized by a general serious condition of the patient, an inflammatory process in the lymph nodes, lungs and other organs.

Etiology. The causative agent of plague, Yersinia Pestis (former name Pasteuriella pestis) is an encapsulated, gram-negative, non-motile, rather pleomorphic bacillus that can take the form of a rod or a coccus. It is a facultative anaerobe. Various toxins and substances have been found in the plague bacillus: some are associated with bacterial virulence, in particular the polysaccharideprotein complex in the shell or wall of the bacillus (it is known as "fraction 1"); two antigens called V and W; fibrinolysis and coagulation factors and bacterial pigment.

Epidemiology. Plague belongs to the group of anthroponoses. Although plague affects many species of small wild animals, the main reservoir of the disease is the squirrel. A person becomes infected with the plague from a sick person, from the bites of fleas that contain pathogens, as well as from rodents (in nature, marmots, gophers, gerbils, squirrels, rats, and others; in populated areas - mice and rats), at home - from cats and camels . It is necessary to know that ticks and fleas can also be carriers of the disease. Epidemics (enzootics) of plague, which have the character of hemorrhagic septicemia, are not uncommon among rodents.

There are two ways of human infection: more often from diseased rodents with flea bites (bubonic or bubonic plague), less often - by airborne droplets from a sick person with plague pneumonia (primary pneumonic plague). A person becomes infected with the plague when bitten by fleas or through damaged skin during direct contact with sick animals - sources of pathogens (in fishing, when skinning rodents, dissection of a carcass.

Yersinia Pestis, entering the human body, does not cause clinical manifestations of the disease from several hours to 3-6 days (incubation period).

The following clinical and morphological forms of plague are distinguished:

— bubonic plague;

— pulmonary;

— septic

Bubonic form of plague– the most frequent (from 90% to 95%). The disease begins suddenly with a rise in temperature to 39-40°C and proceeds with high fever, chills, tachycardia and cerebral manifestations. Severe headache, dizziness, often nausea and vomiting are noted. Patients suffer from insomnia, they lose consciousness, and hallucinations appear.

The place of penetration of microbes can be unnoticed. But sometimes a vesicle, pustule or small necrotized ulcers appear.

Initially, there is a sharp increase (up to 5 cm in diameter) of regional lymph nodes in relation to the gate of infection. Such lymph nodes are called plague buboes. They can be single or multiple. The lymph nodes are soft, full-blooded, bright red, and bleed when cut. Microscopically, fibrinous and hemorrhagic inflammation is detected at an early stage. At a later stage, necrosis is observed and, as a reaction to necrosis, purulent inflammation develops with melting of the tissue of the lymph node. The surrounding subcutaneous adipose tissue and skin are involved in the inflammatory process. With a favorable outcome, ulcers are scarred. The formation of fistulas and phlegmon is possible. A huge number of bacilli are found in necrotized tissues when smears are stained.

In many cases, hemorrhagic-necrotic lymphadenitis is detected in almost all lymph nodes of the body.

The spleen and liver are dramatically increased in volume as a result of marked hyperplasia of macrophage cells of the reticulo-endothelial system. The spleen is septic, weighs up to 600 g, is flaccid, gives a significant pulp scraping, with foci of necrosis and a leukocyte reaction to necrosis.

With lymphogenic spread, new papules appear, where the same morphological changes as in the regional lymph node are noted. Hematogenous spread leads to the rapid development of plague bacteremia and septicemia, which are manifested by rashes, multiple hemorrhages, hematogenous damage to lymph nodes, spleen, secondary plague pneumonia, dystrophy and necrosis of parenchymal organs. With severe bacteremia, thrombosis, widespread foci of hemorrhages and necrosis are observed in the skin, spleen, liver, mucous membrane of the respiratory system, genitourinary tract and gastrointestinal tract, in the serous membranes of internal organs, in the endocardium.

Widespread foci of hemorrhagic necrosis, which create a dark, almost black pattern on the skin, similar to a geographical map, gave this form the name "black plague".

Approximately 5% of patients with the bubonic form develop the pulmonary form - or secondary, initial (primary) pneumonia.

*Primary pneumonic plague*extremely contagious. The duration of the disease is 2-3 days. There is partial pneumonia with lesions of the pleura - pleuropneumonia. The lungs are red, heavy and swollen. On the section, the lung tissue is variegated: next to the bright red, there are areas of grayyellow color. Pleurisy - fibrinous and hemorrhagic. Microscopically, the lumen of the alveoli contains serous-hemorrhagic exudate; later, stasis, hemorrhages, foci of necrosis and secondary suppuration are added. Leukocyte infiltrates are represented by PML. Pronounced phenomena of severe intoxication. Plague pneumonia, which is accompanied by cough, discharge of sputum mixed with blood, a feeling of lack of air and general malaise, often ends in the death of the patient. The secretions are large, bloody, they contain Yersinia Pestis. Decimating bacteremia develops very quickly.

*Septic form of the disease*characterized by numerous hemorrhages in the skin, mucous membranes and various organs. This form also runs hard. Rashes can be represented by pustules, papules, erythema, but with obligatory hemorrhagic and necrotic components, often with the formation of ulcers. In this form, the intestine may be affected, which is accompanied by diarrhea, sometimes with an admixture of blood and mucus in the feces. Multiple hemorrhages are found in the internal organs, mucous membranes, and skin.

To establish a diagnosis of plague on the dissection table, a bacterioscopic and bacteriological examination with the sowing of a culture of the plague bacillus is mandatory, as well as verification of the diagnosis with the help of a biological experiment: the introduction of material taken at autopsy into animals (in the case of plague, the death of animals occurs on the 3-6th the day of the experiment from hemorrhagic septicemia). When dissecting corpses, special preventive measures are observed.

Complication. In the case of plague, they are usually fatal. With bubonic, skin-bubonic and primary septic forms of plague, patients die from septicemia or cachexia - plague marasmus, with primary pneumonic plague - from intoxication or pulmonary complications. Before the era of antibiotics, the fatality rate from the bubonic form ranged from 50 to 90%, and the pulmonary and septic forms were 100% fatal. Today, thanks to antibiotic therapy, the mortality rate does not exceed 5-10%.

1. Theoretical questions Questions for self-control:

1. Definition of the concept of "infection", "infectious process", "infectious disease".
2. Describe the causative agents of quarantine diseases
3. Determine the ways of penetration of pathogens of quarantine diseases into the human body
4. The main clinical and morphological signs of quarantine infections
5. Classification of clinical and morphological forms of plague
6. Describe the changes in the human body during cholera

2 Practical tasks

67. Prepare an essay on the topic: "Definition of the concept of "infection", "infectious process", "infectious disease""
68. Compile the graphological structure "Classification of clinical and morphological forms of plague".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory http://www.webpathology.com/- Web Pathology

Topic #17: "Rickettsiosis. Prion infections..»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:

The student should know:

69. Definition of the concept of "infection", "infectious process", "infectious disease".

70. Know the typical patterns of morphological manifestations of inflammation caused by rickettsial pathogens

71. The main clinical and morphological signs of rickettsioses and prion infections

72. Classification of clinical and morphological forms of rickettsioses

73. Morphological characteristics of rickettsioses

74. Morphological characteristics of prion infections.

The student should be able to:

63. Interpret morphology, clinical and morphological forms of infectious processes
64. Explain the pathogenesis and morphogenesis of infectious diseases
65. Consider and compare the clinical and morphological manifestations of pathologies based on an infectious lesion of a macroorganism
66. To work out the peculiarities of morphological changes in different infectious diseases in different organs on drugs.
67. To be able to explain the complications and consequences that can occur with various infectious diseases

Topic content:

Rickettsioses

Rickettsioses are a group of infections of humans, many warm-blooded animals and some arthropods that are intracellular parasites.

The reservoir of rickettsial infection in nature is wild and domestic animals, they are a source of infection of arthropod bloodsuckers - fleas, ticks.

People develop diseases with characteristic rashes on the skin and peculiar impressions of vessels in the form of vasculitis and thrombovasculitis of varying severity. The infection passes through slightly damaged skin, into which the causative agents of lice and fleas are rubbed with feces.

Classification of rickettsioses

1) typhus group, which includes epidemic typhus, which is transmitted by lice, and endemic or rat typhus, transmitted by fleas.
2) a group of tick-borne spotted fevers (rocky mountain spotted fever, Marseilles, North Australian tick-borne typhus and North Asian tick-borne typhus).
3) a group of endemic fevers, the causative agents of which are transmitted by the larvae of redbodied ticks (Ttsutsugamushi fever).
4) group of pneumotropic rickettsioses or Ku-fever group.
5) group of paroxysmal rickettsioses (Volyn or trench fever).
6) group of rickettsioses of animals.

These rickettsioses are characterized by the following signs:

1) severe vascular
pathology; 2) febrile
condition; 3) rash.

Epidemic typhus is an infectious disease that affects the vessels of the microcirculatory channel and the central nervous system and is characterized by a specific rash and fever. Mortality - 520%. Sporadic cases suddenly occur nowadays. In 1976, the resident of Odesa City Hospital O.O.

Mochutkovsky proved that the transmission of pathogens is carried out by clothes lice.

Incubation period - 10-12 days. The disease begins with a rise in temperature. On the 5-8th day, a rash appears on the skin of the abdomen and chest: roseolae and petechiae. Rickettsia develop in the endothelium of vessels, it swells, proliferates and peels off. Morphological picture. An elementary form of vascular impression is:

1) warty endovasculitis (destruction of the endothelium, necrosis, formation of a wall coagulation thrombus in the form of a wart);
2) proliferation of endothelial and intimal cells;
3) necrosis of the entire thickness of the vessel wall, when all three membranes are necrotized. The vessel collapses, obstructs, thromboses. This is destructive thrombovasculitis.

Changes are more common in the brain. Nodes or granulomas formed from neuroglia (Popov's nodes) form around the affected blood vessels from brain tissue. In the autonomic nervous system - changes in the upper cervical nodes. The skin is hyperemic, roseolae-petechial rashes. Smears from typhoid exanthema reveal rickettsiae. There are foci of necrosis on the retina. In the adrenal glands - dystrophic changes, lipid depletion, edema, hyperemia, tendency to collapse. Myocardium - acute focal interstitial myocarditis or mixed parenchymal-interstitial myocarditis. Spleen - increases 3-4 times, weighs 300-500 g. The pulp is hyperplastic. Pneumonia in the lungs. Granulomas of lymphocytes, macrophages, and plasma cells are formed around the centers of vascular impression.

The cause of death is intoxication with severe circulatory and central nervous system disorders, heart paralysis against the background of collapse. Complications of typhoid fever - hypostatic or aspiration pneumonia, gangrene and bedsores, purulent inflammatory processes in the salivary glands - parotitis, phlegmon, abscesses, purulent otitis, septicopyemia, toxic neuritis of the auditory nerve.

Endemic typhus. The disease is benign, rash occurs in 75% of cases, petechiae occur rarely. The nervous system suffers little. Mortality is almost absent, pathological anatomy is poorly studied.

Prion infections

Prions are an unprecedented class of infectious agents that consist only of reduced host protein molecules. Prions do not contain nucleic acids and, thus, differ from all known microorganisms, such as bacteria, fungi. viruses and virus-like particles. After multiple passages in culture, it was proven that pathogenic prion proteins capable of transmission are mutants of the cellular isoform of the normal prion protein. To date, 18 different mutations of the human RgR gene have been identified, which are associated with various prion diseases.

Prion protein (PgR) is a sialoglycoprotein with a molecular weight of 33,000-35,000 daltons, or 33-35 kD, which is encoded by a single gene located in human chromosome 20. It consists of approximately 254 amino acids in humans, including a 22-membered N-terminal signal peptide. The RgR-c prion is found in all mammals. Its half-life is several hours, but it is well preserved during development. Prions are very resistant to various physical and chemical influences. Peonies are resistant to boiling for 30-60 minutes, drying for up to 2 years, freezing - 3 times more than known viruses, chemical treatment with alcohols, formaldehyde, acids, UV irradiation, gamma radiation, hydrolysis by enzymes. The most effective effect is found in doses that denature almost all proteins. In other words, of all living things, the prion is the last to die. RgR-c is part of the outer cell membranes, is connected to the outer surface of cells by a glycolipid anchor and participates in endocytosis and cell catabolism. Despite the fact that the highest level of PgR concentrations is found in neurons, it can be synthesized by many other cells of the body. The role of normal prion protein (PgR) in healthy individuals is still not completely known. Prion protein is necessary for normal synaptic function. It is assumed that prions are involved in intercellular recognition and cellular activation. Some believe that their function is to suppress age-related processes, and therefore prion diseases are similar in their clinical and morphological characteristics to gerontological diseases. Prion protein (PgR) exists in two forms:

- in the form of a normal, non-infectious form, which occurs in the brain both in normal and infected patients. This form is designated as the cellular prion protein, or PgRc;
- isoform, or RgR-Sc (commonly called "scrarie" - sheep disease), which is a pathological, infectious form and accumulates in the brain only in sick people and animals suffering from spongioform transmissible encephalopathy.

Classification

Today, there are two groups of human diseases that are caused by prions:

- spongiform transmissible encephalopathies
- spongiform myositis with prion-associated inclusions.

The most studied to date are spongiform transmissible encephalopathies.

Pathogenesis of prion encephalopathies. Based on the established fact that prion diseases are unique from the genetic and infectious point of view, Pruziner proposed in 1991 a modern concept of the pathogenesis of spongiform transmissible encephalopathies. Its essence is that a person can be infected with prions in two ways:

1. Mendelian inheritance (autosomal dominant type of inheritance). However, this is a sequential inheritance - due to the previous gene autoreplication of the infectious agent.
2. Transmission of an infectious agent by alimentary or iatrogenic means.

Prion diseases are both infectious and hereditary diseases. They can also be sporadic in the sense that there are cases when no known risk factor is detected, although the infection was most definitely acquired by one of the two previously indicated methods. Based on current knowledge, the transmission of prion encephalopathies is determined by three factors: the dose of infection, the route of infection, and the species barrier. The dose of the infectious agent received by the host depends on the amount of infected tissue and its virulence (infectious titer). But it is always necessary to remember that with repeated exposure there is necessarily a risk of a cumulative effect.

The route of prion infection plays an important role in developmental diseases and has its own hierarchy.
According to the degree of significance, the ways of infection can be divided in the following sequence:
- intracerebral;
-andintravenous;
- intraperitoneal; -oral.

Pathomorphology of prion encephalopathies.
The neuropathology of human prion diseases is characterized by classic microscopic signs:
• spongy changes;
• loss of neurons
•astrocytosis;
• formation of amyloid plaques.
Macroscopically, in all cases of prion encephalopathies, an insignificant decrease in the mass of the brain was noted, in some observations there was a moderate atrophy of the gyri, mainly in people with a prolonged course of the disease.
Microscopically, prion spongioform encephalopathy is characterized by the presence of many oval vacuoles (spongiosis) from 1 to 50 microns in diameter in the neuropil of the gray matter of the terminal brain. Vacuoles can be found in any layer of the cerebral cortex. These can be individual vacuoles or groups divided into sections. On paraffin sections, vacuoles appear optically empty, however, in some of them, when stained with hematoxylin and eosin, fine granularity is often revealed. Vacuoles can merge into microcysts, as a result of which the cytoarchitectonics of the cortex is significantly created. Vacuolation can also be detected in the cytoplasm of large cortical neurons.
In addition to the cortex, spongy changes in the neuropil and vacuolation of the cytoplasm of neurons are determined by the course of all fields of Ammon's horns, by the course of the dentate fascia; in the area of subcortical nuclei, thalamus and cerebellar cortex. Involvement of the cerebellum in the pathological process is the most characteristic manifestation of this disease, although the degree of spongiosis in it is very variable. Fusion of vacuoles is not characteristic of cerebellar lesions. Spongiosis is more often represented by microvacuoles with a diameter of 150 microns, which are located in the molecular layer. Spongiform changes are constantly accompanied by a decrease in the number of neurons in various sections of the cortex. Neurons of the III-VI layer are mainly affected. In some preserved neurons, cytoplasmic vacuolation is noted, some neurons are wrinkled, hyperchromic. The degree of loss of neurons correlates with the severity of spongiform changes and corresponds to the duration of the disease.
Various dystrophic changes were detected in proliferating astrocytes, starting with vacuolization of the cytoplasm and ending with the appearance of ossified forms followed by clasmatodendrosis. Myelin fibers of the cortex remain preserved.
With a long course of the disease, the most pronounced vacuolization and loss of neurons, accompanied by spongy status, with widespread gross vacuolization in all departments, complete collapse of the cerebral cortex in the form of an irregularly distorted framework of glial tissue with small inclusions of preserved neurons. In general, the longer the course of the disease, the clearer the microscopic changes will be. In the basal ganglia and thalamus, severe neuronal death can be combined with gliosis and atrophy. Sharp dystrophic changes are observed in the cerebellum up to the death of granule cells and Purkinje cells. Preserved Purkinje cells are hyperchromic, swollen, with phenomena of tigrolysis and nuclear lysis. Myelin fibers, which are adjacent to the cortex and nuclear groups of the cerebellum, are often varicosely swollen, with fragmentation phenomena. However, it must be emphasized that

One of the morphological signs of prion encephalopathies is the presence of prion-protein (PgR) plaques, which have the appearance of rounded eosinophilic structures. The study of the

structure and topography of RgR plaques is of great theoretical and practical importance. The number, localization, and even microscopic signs of plaques vary with different forms and types of prion encephalopathies. Such plaques are characteristic of Kuru disease.Many authors call them Kuru plaques. They occur less frequently in sporadic and familial forms of CreutzfeldtJakob disease, but very often (more than 70%) in its new form. In isolated observations, they are described in familial fatal insomnia. Very often, PgR-amyloid plaques are localized in the cells of the granular layer of the cerebellar cortex, but they can also be located in the molecular layer and in the white matter. They are usually surrounded by a pale pink halo. The intensity of plaque coloring is different. Perhaps that is why it is not always possible to detect them. For this, standard immunohistochemical methods with RgR antibodies are used. Green birefringence is observed in the detected protein polymers after congo-rot staining under polarizing microscopy. Visually, the spinal cord is practically preserved. Only sometimes a significant decrease in the number of motoneurons is noted. No demyelination of the white matter of the spinal cord was detected in any of the observations. Despite the relatively high concentration of prions observed in peripheral nerves, there are no pronounced structural changes in them.

The clinic of all forms of prion encephalopathy can be represented by various neurological symptoms caused by vacuolization and death of neurons (the main mechanism of action of prions at the cellular level) in almost any part of the gray matter of the brain, including the cerebellum. Typical are:

1. Disorders of the sensitive sphere: varying degrees of amnesia, loss and distortion of sensitivity, loss of sensory functions.
2. Disturbances in the motor sphere, ataxia, immobility, atrophy of muscles, including respiratory muscles, paralysis.
3. Mental disorders: loss of professional skills, depression, drowsiness, aggressiveness, reduced intelligence up to complete dementia.

With the development of clinical manifestations, there are no signs of inflammation, no biological abnormalities in the blood or in the encephalo-arachnoid fluid, no non-invasive, direct, or indirect tests that would allow a confident diagnosis. The clinical diagnosis is confirmed only by histological examination of the central nervous system: spongiosis with vacuolization of neurons, proliferation of astrocytes and glia without signs of inflammation and demyelination.

The group of human prion subacute transmissible spongiform encephalopathies includes:

- Creutzfeldt-Jakob disease;
- Gerstmann-Straussler-Sheinker disease;
- "fatal familial insomnia" syndrome;
- Kuru disease;
- chronic progressive childhood encephalopathy, or Alpers disease.

Creutzfeldt-Jakob disease (CJD) is a subacute encephalopathy characterized by slow progressive death of neurons. The disease occurs mainly in adults and is characterized by the rapid development of dementia, which is accompanied by pyramidal and extrapyramidal symptoms. Infection occurs when eating meat from cows suffering from a similar disease. Cases of humanto-human transmission have been described during implantation
intracranial electrodes, corneal transplants and, most often, with the introduction of growth hormones extracted from the human pituitary gland. Diffuse atrophy of the cerebral cortex with spongiform changes, especially in the neocortex, and widespread neuronal dystrophy is most often observed in cerebral lesions. On the section, foci of softening of the brain substance are visible, sometimes in the form of hollow, filled with dull grayish-pink mushy contents. Microscopically, there is a decrease in the number of neurons and reactive proliferation of astrocytes. Numerous small vacuoles are found in the processes of neurons and astrocytes, as a result of which the term "spongiform encephalitis" appeared. In this disease, there are no signs of an inflammatory response in the brain tissue.

Creutzfeldt-Jakob disease is presented in three classic forms:

- sporadic form (85-90% of all cases);
- family form (10-15%);
- iatrogenic form (% not yet definitively established).

In addition, at the suggestion of British researchers, today one more form, the so-called "new atypical form" of Creutzfeldt-Jakob disease, has been identified.

The sporadic form of Creutzfeldt-Jakob disease is one of the rare diseases. It is common on all continents. CKD occurs in different age groups - from 17 to 82 years old, depending on the form of the disease. The duration of the disease varies from a few weeks to eight years. However, the average life expectancy from the onset of the disease is six months.

Iatrogenic forms are the result of neurosurgical infection due to insufficiently disinfected surgical instruments or electrodes during transplantation of the cornea, dura mater or during treatment with derivatives of the human pituitary gland (growth hormones and gonadotropins). The incubation period of iatrogenic and other forms of CKD depends on many factors: the method and gate of the infection entering the body, its phenotype, the dose of the infection, and the recipient's genotype. In those cases where the penetration of the agent occurred directly into the central nervous system, the incubation period was from 10 to 30 months, and the first sign in the clinic was dementia. At the time when the infection entered the body from the periphery, for example, with the introduction of growth hormones or gonadotropins, the incubation period increased to 5 years or more, sometimes reaching 35 years.

1. Theoretical questions

Questions for self-control:

1. Variants, forms and stages of the course of rickettsiosis;
2. Typical patterns of morphological manifestations of inflammation caused by rickettsial pathogens;
3. Sites of primary penetration (entrance gate) of infection in rickettsiosis and prion infections;
4. The essence of the infectious process in rickettsiosis;
6. Micro- and macroscopic characteristics of changes in rickettsioses and prion infections.

2 Practical tasks

69. Prepare an essay on the topic: "The essence of the infectious process in rickettsiosis."

70. Make a graphological structure "Variants, forms and stages of the course of rickettsioses".

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
- http://www.webpathology.com/- Web Pathology

Topic #18: "Children's infections.»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:
The student should know:

75. Etiology, pathogenesis, pathomorphology of children's acute respiratory infections.

76. Prognosis, complications of these diseases.

77. Local pathological manifestations of diphtheria.

78. Pathomorphology of various forms of meningococcal infection.

79. Pathomorphology of mild and severe forms of whooping cough.

The student should be able to:

68. To interpret the pathomorphology of scarlet fever depending on the period and severity of its course.

69. Pathogenesis of exanthems in scarlet fever, measles, rubella, chicken pox, pseudotuberculosis, meningococcemia.

70. Assess the importance of complications and explain the causes of death in whooping cough.

71. To evaluate the importance of complications and causes of death in diphtheria.

72. Define measles.

Topic content:

Measles is an acute highly contagious infectious disease of children, which is characterized by catarrhal inflammation of the mucous membranes of the upper respiratory tract, conjunctiva and a spotted-papular rash on the skin. Transmission is carried out by airborne droplets. Children under 3 years old and adults rarely get measles. However, cases of measles in adults are also registered today. The duration of the disease is 2-3 weeks. The causative agent of measles is a 150-nm RNA virus from the Paramyxoviridae family, which reproduces primarily in the respiratory system. Focal serous - macrophage inflammation occurs in the affected areas, and giant multinucleated cells from the epithelium are formed right there.

The measles virus is able to reduce the barrier function of the epithelium, phagocytic activity, and also cause a drop in the titer of protein-infectious antibodies. This state of anemia sharply increases the susceptibility of patients to secondary infection or exacerbation of an existing chronic process, for example, tuberculosis. Transferred measles leaves persistent immunity.

Macroscopically, the process at this time has the character of catarrhal pharyngitis, laryngotracheobronchitis. The mucous membrane is swollen, full of blood, secretion of mucus is sharply increased, which is accompanied by a runny nose, cough, lacrimation. In severe cases, necrosis may occur, the mucous membrane becomes dull, grayish-yellow in color. Swelling and necrosis of the mucous membrane of the larynx can cause a reflex muscle spasm with the development of asphyxia (false croup). In addition, as a rule, small red pneumonia foci are found in

the lungs. Measles is characterized by metaplasia of the epithelium of the mucous membranes into a multi-layered flat one, which sharply reduces the barrier function of the epithelium. With an uncomplicated cortex, miliary and submiliary foci of proliferation of lymphoid, histiocytic and plasma cells are formed in the interalveolar septa of the lungs. The development of interstitial pneumonia is possible, in which strange giant cells are formed in the walls of the alveoli - giant cell cow pneumonia. Along with this, catarrhal conjunctivitis occurs. Hematogenous dissemination of the virus in the body soon occurs. In many organs, including the mucous membrane of the nose and pharynx, the tissues of the tonsils and appendix, there is hyperplasia of reticular cells and giant cell metamorphosis of affected cells, primarily epithelial. At the same time, a measles-specific lesion of the mucous membranes (enanthema - Filatov's - Koplik's spots) develops. In these areas, full blood, focal edema with vacuolization of epithelial cells and small lymphohistocytic infiltrates are determined. Macroscopically, enanthema initially looks like small red foci. Soon their central areas - a place, where the peeling of the epithelium begins - they become whitish. Later, an exanthema appears - a large papular rash on the skin: first behind the ears, then on the face, neck, trunk, finally on the limbs, more on the extensor surfaces. At the same time, skin appendages are affected.

Complication. Among the complications, the central place is occupied by damage to the bronchi and lungs, associated with the addition of a secondary viral and bacterial infection. Not only the inner lining of the bronchi (endo-bronchitis), but also the middle (mesobronchitis) and the outer (peribronchitis) are damaged. Panbronchig often has a necrotic or purulent-necrotic nature. Affected bronchi on lung autopsy have the appearance of grayish-yellow foci, very similar to tuberculous nodules. Such panbronchitis is the source of the development of bronchiectasis, lung abscesses, purulent pleurisy. The transition of the process to the peribronchially located lung parenchyma leads to the development of peribronchial pneumonia, and later - to chronic lung damage with the outcome of pneumosclerosis. The most severe, although too rare, manifestation of the generalization of the disease is damage to the central nervous system. Measles virus penetrates nerve cells, in which, under light microscopy, it can appear in the form of inclusions. First of all, the neurons of the large hemispheres are affected. They swell, vacuolize or shrivel. Focal disintegration of myelin is determined in the white matter, mostly in those areas where the axoplasm of nerve fibers is most damaged. These changes are accompanied by the proliferation of astrocytes, microglial cells and lympho-monocytic perivascular infiltrates. Infectious-allergic mechanisms play a significant role in the pathogenesis of bovine encephalitis. These changes are accompanied by the proliferation of astrocytes, microglial cells and lympho-monocytic perivascular infiltrates. Infectious-allergic mechanisms play a significant role in the pathogenesis of bovine encephalitis. These changes are accompanied by the proliferation of astrocytes, microglial cells and lympho-monocytic perivascular infiltrates. Infectious-allergic mechanisms play a significant role in the pathogenesis of bovine encephalitis.

Diphtheria is an acute infectious disease characterized mostly by fibrinous inflammation in the focus of primary fixation of the pathogen and general intoxication associated with the absorption of exotoxin. Most often, children from 4 to 6 years old are sick. Adults and children older than 7 years are now sick. The causative agent of diphtheria is Corynebacterium diphtheriae. Infection occurs mainly by airborne droplets. Diphtheria bacillus is well preserved in the environment. The main source of infection is a person suffering from diphtheria, who is dangerous for those around him during the entire period of illness, even for some time after recovery. When coughing, sneezing, and talking, along with droplets of saliva, sputum, and mucus, the patient releases pathogens into the environment. A healthy person becomes infected by inhaling contaminated air. The source of infection can be a carrier - a healthy child or an adult without visible signs of the disease, but who secretes diphtheria plaques. Carriers are more often children. Diphtheria bacillus infects the mucous membranes of the nasopharynx, pharynx, and upper respiratory tract (larynx, trachea). The diphtheria bacillus takes root on the mucous membrane, but the toxin released by it is carried by blood and lymph throughout the body. The toxin at the site of penetration and reproduction of the bacillus causes

inflammation of the mucous membrane with the formation of a dense gray-white film coating on it, which is closely soldered to the tissues. The diphtheria bacillus takes root on the mucous membrane, but the toxin released by it is carried by blood and lymph throughout the body. The toxin at the site of penetration and reproduction of the bacillus causes inflammation of the mucous membrane with the formation of a dense gray-white film coating on it, which is closely soldered to the tissues. The diphtheria bacillus takes root on the mucous membrane, but the toxin released by it is carried by blood and lymph throughout the body. The toxin at the site of penetration and reproduction of the bacillus causes inflammation of the mucous membrane with the formation of a dense gray-white film coating on it, which is closely soldered to the tissues.

Depending on the place of penetration and reproduction of diphtheria bacilli, different forms of the disease are observed. Diphtheria is characterized by the fact that it:

1. belongs to the number of characteristic so-called local infections, the causative agents of which, as a rule, are found only in the area of the primary localization of the process;
2. the lifelong symptom complex of diphtheria and all the underlying anatomical changes are the result of exposure to the body of diphtheria toxin, so there is not a single symptom in the entire picture of the disease that could not be obtained experimentally with the help of one poison without the participation of living pathogens.

The patho-anatomical characteristic consists in the fact that the most constant reaction of local tissues to the penetration of the causative agent into them is fibrous inflammation in the form of diphtheritic or croupous. Diphtheria is a crust, a film, the cause of fibrinous inflammation in the place of localization of the diphtheria microbe and mainly, in its ability to extremely deeply disrupt the activity of the cardiovascular system in general and especially in the part that is located in the region of toxin formation, that is, the entrance gate. The result of the effect of the toxin on the vascular system is an unusually strong paralytic expansion of local blood vessels, a slowing of the blood flow and a sharp increase in the porosity of the vascular walls, all of which contribute to the appearance of a protein-rich inflammatory exudate. In addition,

The sequence of development of the process on the mucous membrane is presented as follows. Damage to the mucous membrane under the influence of diphtheria bacillus toxin begins with coagulation necrosis of the epithelium, which creates the ground for further development of the pathogen. The poison penetrating through the epithelium causes an inflammatory process in the underlying layers of the mucous membrane, where the expansion and overflow of blood vessels occurs, as well as the rapid formation of liquid exudate. This exudate penetrates the epithelial cover and, reaching the necrotic zone, thanks to the action of diphtheria toxin, easily coagulates under the influence of thrombokinase, which is released during cell necrosis, forming a visible film on the surface of the mucous membrane. As long as only the surface layer of the mucosa is exposed to necrotizing influence, fibrin shedding is limited to the epithelial cover and is easily separated. With deeper tissue necrosis and damaged connective tissue bases, the fibrinous exudate captures the submucosa, and the film is closely related to the underlying tissue, and its removal is difficult and accompanied by bleeding. The most frequent place of primary localization of the process is the tonsils, as well as the mucous membrane of the pharynx and upper respiratory tract. In the throat, as a rule, we observe diphtheritic inflammation, and in the respiratory tract - croupous inflammation, which depends on the epithelium covering the mucous membrane and the ease of removing the film. While the films of the multi-rowed ciliated epithelium of the respiratory tract are relatively weakly connected to each other and the underlying border membrane, due to which they are easily peeled off in whole layers, the multilayered flat epithelium of the tonsils, on the contrary, is a very compact cell mass, all elements of which are extremely strongly connected with each other, so with the fibrous connective tissue directly adjacent to it. Therefore, the fibrous film that forms on the surface of the tonsils, the arch of the soft palate is strongly connected to the underlying tissue and its separation is slow, or with an ulcerated defect on the surface of the mucous membrane. As for the cylindrical epithelium of the mucous membrane of the respiratory tract, it is easily separated together with the fibrinous mass,

which is facilitated by the accumulation of mucus under the film, which is released in large quantities from the secretory glands. Thanks to this, patients sometimes cough up whole fibrinous casts of the respiratory tract. The natural removal of the films takes place either by removing them from the underlying tissue with the help of demorcation inflammation, or the films undergo purulent melting and then disappear gradually, this process takes place between the 2nd and 7th day from the beginning of their formation. In their place, very superficial excoriations or ulcers remain, which granulate and epithelize very quickly.

In relation to diphtheria, the proposition that the degree of intensity and spread of the local process corresponds to the severity of intoxication is verified, that is, there is a parallelism of clinical and anatomical data.

Damage to the digestive tract: in the digestive tract, the tonsils are most often in the first place, and the process can be limited to them.

At the same time, the tonsils significantly increase in volume and due to sharp hyperemia, they get a dark red color on the section (in places it changes to black-red). Against this background, pale grayish-yellow foci of necrosis are clearly distinguished, often penetrating the tonsil tissue to a considerable depth and located along the walls of the crypts. In other cases, the process can spread to adjacent areas of the pharynx and pharynx, the root of the tongue, gums, nasopharyngeal region, epiglottis, sometimes to the Eustachian tube and the middle ear. At the same time, fibrinous deposits cover these formations either with a solid cover or in the form of islands of larger or smaller size. The color of the deposits is mostly whitish, with hemorrhages, the color becomes muddy gray. The mucosa outside the deposits is red. In exceptional cases, the process spreads further, involving the tongue, gums, hard palate,

Damage to the respiratory tract: first of all, it is necessary to indicate damage to the nasal cavity, as it often serves as the primary location of the process in children. The process has the character of catarrhal inflammation with bloody secretions, indicating deeper damage to the vascular system.

Complications of rhinitis - transition from the usual form of diphtheria, spread to neighboring organs, complicated pneumonia. Diphtheria is characterized by the corrosive effect of secretions on the skin, as a result of which patients have superficial ulcers on the edges of the nostrils and on the upper lip, often covered with crusts. From the lower parts of the respiratory tube, the larynx and the upper part of the trachea are most often damaged, and the process can be isolated, but is more often combined with damage to the pharynx and pharynx. Often croup of the larynx shows a tendency to spread down the respiratory tract in the form of a so-called descending croup, capturing the trachea, bronchi, sometimes to small branches. Lobular bronchopneumonia is observed in lung tissue with descending croup.

Eye diphtheria. Eye damage with diphtheria is also secondary, either passing from the nasal cavity or transferred by the child's fingers.

The process begins with the conjunctiva of the eyes. By its nature, the inflammation is either granular, causing the formation of delicate films, or more often diphtheritic, in which the deposits take on a muddy yellowish-gray color and a greasy appearance. In severe cases, the entire mucosa throughout has a uniformly muddy-sebaceous appearance, with areas of hemorrhage. Then purulent exudate, scarring appears.

Damage to the nervous system, especially peripheral, is of great importance. The most characteristic is selective damage to nerve fibers. The periaxonal nature of dystrophic changes, which consist in the breakdown of myelin, is especially typical. The reaction from the cells of Schwann's membrane is insignificant. Weakly expressed changes in axial cylinders. Intraganglionic nerve fibers of intervertebral nodes are partially damaged. In the vagus nerve, not all areas are damaged in the same way - the process captures mainly the thick soft fibers of its branches, which depart at the level of the ganglion nodosum. Often the roots of the spinal cord, anterior, are involved in the pathological process, the damage in this case has a pronounced segmental character, accompanied by paralysis.

Damage to the cardiovascular system. In the early stages of the disease, paresis of blood vessels is noted, fibrinoid necrosis is often found in arterioles, areas of necrosis can also occur in the walls of arteries. Dystrophic and necrotic changes in the form of lysis, vacuolization, or deep disintegration of muscle fibers are first detected in the myocardium, later - on the 2nd or 3rd week of the disease - acute interstitial myocarditis, in which there is focal infiltration of the intermuscular connective tissue layers by histiocytes and lymphocytes with impurities of plasma cells of neutrophils, eosinophilic leukocytes. Changes occur more often in the left ventricle. The muscles of the heart are flaccid, yellowish in color, blood clots form in the veins. Myocarditis can be the cause of acute heart failure with a fatal outcome - early heart paralysis, after 2-2, 5 months - late paralysis of the heart. It is caused by toxic damage to the vagus nerve.

Changes in the organs of the chromophore system (adrenal glands, paraganglia), - sharp disturbances in blood circulation, often with hemorrhages, necrosis of part of the cells of the cortex. In the medulla of the adrenal glands - a decrease in the content of adrenaline.

Frequent changes in the kidneys. When the local process is localized only in the respiratory tract, absorption of toxins almost does not take place, therefore changes related to intoxication are not observed. With this form of diphtheria, the most important complication is asphyxiation. It is due to the mechanical closure of the lumen of the respiratory tract, reflex spasm of the laryngeal muscles and swelling of the mucous membrane.

Scarlet fever is an acute infectious disease with initial damage most often to the pharynx. The name comes from the word "scarlatina" - bright red. The causative agent of the disease is hemolytic streptococcus.

Scarlet fever mainly affects children, more often at the age of 3 to 10 years. Infection is transmitted from a person, mainly by aerosol, rarely through objects. The incubation period ranges from several hours to several weeks. Diseases occur in the form of sporadic cases.

Streptococci settle on the mucous membrane of the upper respiratory tract or digestive tract, especially often on the tonsils. They begin to multiply in the depth of one or more crypts of the tonsils. Necrotization of the crypts and lymphatic tissue of the tonsils takes place.

Around the focus of necrosis - full blood, edema, leukocyte reaction with the formation of a zone of demarcation inflammation. Fibrin falls out on the surface of the tonsils.

Macroscopically, the tonsils are enlarged, swollen, bright red with white or yellow dots in the mouths of the crypts, lamellar deposits of fibrin. Soft palate and nasopharynx full of blood (burning throat). The tongue is covered with a grayish-yellow coating, crimson. The development of a primary affect in the lungs and skin is possible. Such forms are called extrabuccal. Not every streptococcal sore throat, even a necrotic one, is scarlet fever. First of all, lymphogenic spread of streptococci occurs, first of all, in the regional submandibular lymph nodes. In the future, the inflammatory process develops with the advantage of an alternative component. Permanent damage, along with tonsillitis, of the regional lymph nodes allows us to talk about the primary scarlatinous complex. The inflammatory process can spread beyond the nodes to adipose tissue and m'

Sometimes it is accompanied by septic thrombophlebitis or erosion of blood vessels. Later, hematogenous desquamation may occur. It passes after the development of thrombophlebitis in the veins of the tonsils, later septicopyemia occurs, which is accompanied by metastatic abscesses in various organs, including purulent inflammation of large joints - elbow, hip, and knee. Intracanalicular spread of streptococci is often observed. There is damage to the mucous membrane, underlying tissues, ethmoid bone (ethmoiditis). The transition of the process from the vein to the eyes, meninges and brain tissue is possible with the development of purulent meningitis or brain abscess, the spread of infection through the Eustachian tube, into the middle ear, developing otitis, thrombophlebitis of the memoid sinus, purulent meningitis or brain abscess. Streptococcal toxins also spread throughout the body. This is most pronounced in the first 3 days of the disease. Rashes are formed. A sharp focal hyperemia and edema is determined, then perivascular lymphohistiocytic infiltrates, necrosis of the surface layers of the epithelium. Macroscopic - rashes of bright red color,

microcellular, appear on the chest, back, neck, on the nasopharyngeal triangle - at the end of the first, at the beginning of the second week.

In addition to rashes, toxic manifestations include dystrophic changes in parenchymal organs and acute swelling of the brain with severe damage to nerve cells. Interstitial inflammation occurs in parenchymal organs. Depending on the predominance of septic or toxic damage, toxic, septic, and toxic-septic forms of scarlet fever are distinguished.

All considered changes refer to the first period of scarlet fever (1-2.5 weeks). On the third Sunday, some patients have a second period of scarlet fever. All changes are less pronounced, there are no rashes. Sowing of streptococci from tonsils due to superinfection is noted. This process causes severe allergic damage, among which is glomerulonephritis.

Meningococcal infection. It is one of the forms of meningococcal infection with predominant damage to the soft meninges of the brain and spinal cord. In addition to meningitis, nasopharyngitis and meningococcemia of a meningococcal nature are distinguished.

Meningococcal meningitis is more common in children under the age of five.

Infection by aerosol from a sick person and a carrier. The incubation period is 2-3 days. Duration of clinical manifestations - several weeks. Occurs in outbreaks or sporadic cases.

A moderately pronounced inflammatory process occurs in the nasopharynx, the morphology of which is little studied. In some cases, the infection is limited to a catarrhal process in the nasopharynx - meningococcal nasopharyngitis.

Spreading by hematogenous route, IDNS is most often affected. Reproduction of cocci in the subarachnoid space. The surrounding tissues are sharply full of blood, an effusion of serous exudate. After a few hours, the process becomes serous-purulent, and after 1-2 days - purulent or fibrinouspurulent.

Localization of inflammation on the surface of the large hemispheres, it also happens at the base (behind the intersection of the optic nerves), often the process involves the spinal cord, it can spread deep with the development of encephalitis or into the cerebral ventricles with the appearance of purulent ependymocytes.

Macroscopically, in the midst of the disease, meningitis has a characteristic appearance. The brain is covered with a purulent cap, the meninges are swollen, cloudy, blood vessels are sharply dilated. During recovery, the meningococci die first, and the exudate dissolves. Complications may occur with prolonged flow. Circulation of the cerebrospinal fluid may be disrupted if the outflow tracts are blocked by exudate or obliterated, with its organization internal hydrocephalus develops.

The transition of the inflammatory process to the arteries of the brain with their thrombosis and disruption of the blood supply to areas of the brain in which there are foci of softening is possible.

Meningococcemia may develop with the most severe course of the disease. In this case, foci of inflammation appear in various organs and tissues. Damage to soft meninges sometimes does not have time to develop and patients die within 24-48 hours. Typical changes in blood vessels, hemorrhages in various organs, in the skin.

Hemorrhages in the adrenal glands, which are accompanied by acute adrenal insufficiency, are especially dangerous. Vascular thrombosis and necrotic changes in the skin are observed.

Whooping cough is an acute infectious disease of children with a cyclic course and characteristic attacks of convulsive cough.

Bordetella pertussis, the causative agent of whooping cough, is a short rod-shaped bacterium. Discovered by Belgian scientists Bordet and Frenchman Jeangou in 1906. The infection is transmitted by airborne droplets. Infection is possible only when communicating with a patient, because Bordetella pertussis dies quickly outside the body. The danger of infection through surrounding objects is practically excluded.

Most often, children from 1 to 5 years old are sick, sometimes even children up to 1 year old. In adults, the disease is rare. Whooping cough leaves stable immunity, repeated diseases are very rare.

The incubation period lasts from 2 to 15 days (5-9 days on average).

Clinic. At first, a small cough appears, which increases day by day. The temperature rises, the child becomes nervous, sleep and appetite worsen, this period is called catarrhal and lasts up to 2 weeks. All manifestations of the disease continue to increase; gradually the child's well-being worsens, the cough becomes more prolonged and severe, and at the end of the second - beginning of the third week it acquires a paroxysmal character: the disease passes into the third period - spasmodic, which lasts 1-5 weeks. Attacks of convulsive cough are the main and permanent symptom of the disease. The cough begins with two or three deep coughing movements, followed by a series of short movements, they come one after the other and end with a deep whistling breath due to a convulsive narrowing of the larynx. After that, coughing fits begin again. The severity of the disease depends on the duration and frequency of attacks. In young children, coughing fits are long (up to 2-3 minutes), consist of short exhaling impulses without whistling breaths. During an attack, the patient's face turns red, and after that it has a bluish tint. Tears appear in the eyes, sometimes hemorrhages form in the white membrane of the eyes, the tongue sticks out of the mouth, neck veins swell, involuntary separation of feces and urine is possible. The attack ends with the release of viscous sputum and often vomiting. Coughing attacks are repeated from 5 to 30 or more times a day. The face becomes puffy, the eyelids swell, and hemorrhages may appear on the skin of the face. In the intervals between coughing attacks, the digas feel quite satisfactory. The cough gradually weakens, the attacks become less frequent - the recovery period begins, which lasts an average of 1-3 weeks.

The total duration of the disease is from 5 to 12 weeks. A child is considered contagious within 30 days from the onset of the disease. Mass vaccinations have led to the emergence of so-called erased forms of whooping cough, when the spasmodic period can be very light or completely absent.

Pathogenesis. Bordetella pertussis reproduces mainly on the mucous membrane of the respiratory tract. Their epithelium undergoes dystrophic changes and peels off, signs of catarrhal inflammation are revealed. Bronchial openings contain serous exudate with a small admixture of leukocytes and macrophages. Bordetella are found in the exudate, lying freely or phagocytosed. Sometimes there is a bronchogenic spread of the inflammatory process to the respiratory departments. In these cases, small foci of pneumonia occur. The areas with progressive changes in the alveoli contain serous-macrophagal-leukocyte or serous-leukocyte exudate. Along with this, severe functional changes are revealed: acute emphysema is noted in the anterior parts of the lungs; in the back sections, areas of emphysema alternate with areas of atelectasis.

The decay products of the causative agent (endotoxins) cause irritation of the nerve receptors of the larynx, impulses are generated that go to the brain and lead to the formation of a persistent focus of irritation in it. Due to the reduction of the threshold of excitation of nerve centers and receptors, a small non-specific irritation can be enough to cause an attack of spastic cough. A "respiratory tract neurosis" develops, which is clinically manifested by jerky exhalations coming one after the other, which are replaced by a convulsive deep breath, repeated many times and ending with the release of viscous sputum or vomiting. Spasm of the larynx, bronchial muscles, peripheral vessels, vomiting and other symptoms indicate irritation of not only the respiratory but also other vegetative centers. Attacks of spastic cough cause stagnation in the system of the superior vena cava, which increases blood circulation disorders of central origin, and lead to hypoxia. Whooping cough is especially severe in infants, they do not have spastic cough attacks, their equivalent is apnea attacks with loss of consciousness and asphyxiation.

Today, thanks to seroprophylaxis and mass vaccination, the severity of the course and morbidity have decreased significantly, the mortality rate does not exceed tenths of a percent.

Pathomorphology. At the autopsy, the face is swollen, acrocyanosis, hemorrhages on the conjunctiva, facial skin, oral mucosa, pleural sheets and pericardium are noted.

Macroscopically, there is a moderate fullness of the respiratory tract with slight semi-liquid overlays on the mucous membrane. The lungs are swollen, bullous emphysema is often detected at the front edge of the lungs. In the back sections, the lungs are gray-red, often with dotted hemorrhages.

On the section, you can see separate small gray or gray-red foci of compaction that protrude, and more numerous sunken dark-red areas (atelectases).

Microscopically, catarrhal inflammation is found in the mucous membrane of the larynx, trachea, and bronchi: vacuolization of the epithelium, increased mucus secretion, hemoptysis, edema, moderate lymphohistiocytic infiltration; in the lungs - small bronchi in a state of spasm, in the parenchyma of the lungs - edema, hemoptysis, atelectasis. Small foci of pertussis pneumonia may develop in infants. At the same time, a serous-leukocyte and even fibrinous exudate with a large number of pertussis rods is found in the alveoli.

In other organs, including the brain, there are blood circulation disorders, in places with plasma and hemorrhages.

Complications: pneumonia (especially in children from 1 to 3 years old), nosebleeds, respiratory arrest. In thoracic and weakened dggei, whooping cough can be very difficult: the catarrhal period is short, sometimes a spasmodic period immediately begins, and often coughing fits lead to respiratory arrest.

1. Theoretical questions

Questions for self-control:

1. Etiological and pathogenetic factors of diphtheria.
2. Classification and typical pathomorphological picture of different localizations of diphtheria (tonsils, larynx, nose, skin, external genitalia).
3. Basic properties of the causative agent of measles. Epidemiological features.
4. The main links of the pathogenesis of measles.
5. Etiology, epidemiology of scarlet fever;
6. Pathogenesis of scarlet fever, pathomorphological manifestations of the disease.
7. Etiology, epidemiology of rubella.
8. The main links of the pathogenesis of rubella;
9. Diagnostic criteria of acquired, congenital rubella. Etiology, epidemiology, pathogenesis of chicken pox;
10. Pathomorphological changes in chicken pox

2 Practical tasks

71. Prepare an essay on the topic: "Etiological and pathogenetic factors of diphtheria."

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory

- http://www.webpathology.com/- Web Pathology

Topic #19: "Diseases caused by protozoa, helminths. Mycoses..»

Purpose: as a result of independent study of this topic, students should know the topic for studying the topic at clinical departments. In the practical work of a doctor, it is necessary for the clinical and anatomical analysis of sectional observations. .

Basic concepts:
The student should know:

80. Morphogenesis, morphological characteristics, complications, consequences of trichinellosis, echinococcosis, cysticercosis, opisthorcosis, schistosomiasis

81. Classification and features of the structure of mycoses.

82. Etiology, pathogenesis, pathomorphology of the most common mycoses.

83. Prognosis, complications of these diseases.

84. Clinical and morphological forms of mycoses, pathomorphosis and complications accompanying certain forms of mycoses in both children and adults.

The student should be able to:

73. To interpret the modern classification of mycoses.

74. To characterize the etiology of mycoses.

75. To characterize the features of the pathomorphosis of mycoses.

76. Classify diseases caused by helminths.

77. To characterize the etiology, pathogenesis and morphological essence of diseases caused by helminths.

78. To characterize the etiology, pathogenesis and morphological essence of diseases caused by protozoa

79. To characterize the etiology, pathogenesis and morphological essence of diseases caused by helminths.

80. Carry out clinical and laboratory diagnostics with microscopy of certain types of mycoses, helminths, protozoa.

Topic content:

Mycoses

1. The essence of mycoses and mycotoxicoses.

Mycoses are diseases in which the active parasitism of pathogenic fungi occurs in the body of animals and pathological changes occur mainly in the places of their parasitism. They have a chronic course.

Mycotoxicoses are diseases that arise as a result of eating feed contaminated with toxic mushrooms or their waste products (toxins). The nature of pathological changes depends on the resistance of the organism, the virulence of the fungus, and the environmental conditions. These diseases occur when eating fodder affected by fungi.

The diagnosis of fungal diseases is made on the basis of epizootic data, clinical-anatomical and histological studies.

Pathological-anatomical changes are characterized by damage to the digestive tract, the liver, which receives blood that washes the intestines with toxins that have been absorbed, and the kidneys, an excretory organ. Microscopic studies of the affected organs make it possible to identify drusen and fungal vultures. 1. Classification of mycoses:

1) Visceral:
-primary
-secondary (opportunistic)
-epidermicosis: epidermophytes

2) Dermatomycosis:
-superficial dermatomycoses: the main changes are emphasized in the epidermis, but the dermis is also affected
- deep dermatomycoses: together with the dermis, the epidermis is affected.

Visceral mycoses O.K. Khmelnytskyi in 1962 divided into:
I. Primary acute infectious visceral mycoses (obligate pathogenic):
- cryptococcosis, blastomycosis, coccidomycosis, histoplasmosis, rhinosporidosis II. Opportunistic mycoses (facultatively pathogenic):
- candidiasis and mold mycoses (aspergillosis, mucorosis, penicillosis)

Actinomycosischronic disease, the causative agent is the anaerobic radioactive fungus Actinomyces Israeli, which thrives in tonsil crypts and carious teeth. Endogenous infection usually occurs with injuries and microtraumas of the organs of the oral cavity. Drusen causes positive neutrophil taxis, i.e. a focus of purulent inflammation is formed, then macrophages, plasma cells, undifferentiated connective tissue cells proliferate perifocally, xanthoma cells appear, autogenous vessels form, granulomas merge. Areas of purulent inflammation, surrounded by granulation and mature connective tissue, have the appearance of bee honeycombs on the section, and grains of actinomycetes drusen are visible in the pus. The actinomycotic infiltrate spreads through the cellular and connective tissue layers of organs, forming a fistula course.

There are two clinical and morphological forms:

I. Destructive - in which tissue destruction with the formation of large abscesses prevails;

P. Destructive - proliferative with growth of connective tissue.

According to localization, the following are distinguished:
- cervicofacial actinomycosis (most often); - abdominal
- actinomycosis of the lungs and chest cavity;
- bone-articular, muscular, skin;
- actinomycosis of the nervous system.

Candidiasis (candidiasis)

A yeast-like fungus of the genus Candida saprophytes on the surface of the skin and mucous membranes, parasitises when the resistance of the macroorganism is weakened. Primary candidiasis (without provoking factors) can develop in young children. Secondary candidiasis - autoinfection (after the provoking effect of any factors: antibiotic, corticosteroid therapy, viral infection, etc.). probably, the elimination of endogenous microflora during antibiotic therapy promotes adhesion of fungi to the epithelium without cornification of the oral cavity, esophagus, and vagina. The fungus is tropic to the glycogen-rich multilayered squamous epithelium of mucous membranes. When fungi

penetrate the underlying tissue, oval or rounded yeast-like cells can transform into pseudomicellar (filamentous) forms, which carry out invasive growth. Defensive reactions, which develop in response to the introduction of candida in the tissue, are controlled by the immune system and are provided by neutrophils. Neutrophils are capable of active phagocytosis of fungi, characteristic stringing of neutrophils on a thread of pseudomycelium and their mass death. Local candidiasis: on the skin or mucous membrane, brown overlays of pseudomycelium threads, desquamated epithelium and neutrophils, there may be foci of mucosal necrosis with demarcation purulent inflammation. When pseudomycelium germinates in the vessel lumen, metastasis occurs in the vascular organs.

Two forms of tissue reactions in visceral candidiasis:

1. exudative-necrotic, - around the fungi there is a cellular infiltrate of disintegrating neutrophils strung on a thread of pseudomycelium, and necrotic ones predominate when ICS weakens. And not inflammatory changes.
2. tuberculoid granulomatous reaction - with a protracted course, a productive reaction prevails. The center of the granuloma usually contains only fragments of the fungus and cellular detritus, surrounded by macrophages, epithelioid and giant polynuclear cells, and on the periphery by lymphocytes. As the granuloma matures, fibroblasts appear in it.

Candida can parasitize in the cytoplasm of macrophages and giant cells (endocytobiosis), so it is impossible to speak with certainty about their complete phagocytosis by macrophages. Incomplete phagocytosis can contribute to the dissemination of the pathogen and the development of hematogenous forms of candidiasis.

Gastrointestinal candidiasis. The favorite localization of candidiasis, along with the skin, is the upper part of the digestive tract; lesions of the mucous membranes of the stomach and intestines are somewhat less common. Penetration of fungal threads deep into the wall leads to the formation of ulcerative defects, sometimes leading to perforation.

- esophageal candidiasis - characterized by stenosing films
- intestinal candidiasis is characterized by pseudomembranous overlays and the formation of ulcers.
- candidiasis of the stomach can be accompanied by perforation of the stomach. If the fungi are localized at the bottom of the stomach ulcer, they can become the source of a generalized process.

*Candidiasis of the urinary tract*the kidney arises in an ascending way. Possible candidal urethritis, cystitis, pyelonephritis. Small abscesses, foci of necrosis or granulomas form in the cortical layer of the kidneys.

Candida laryngitis, tracheitis, bronchitis, as well as pneumonia, which develop both as a result of aerogenic and hematogenous entry of the pathogen into the lung tissue, are characteristic. Small foci of fibrinous inflammation with necrosis in the center are found in the lungs, cavities are formed after their suppuration. With a long course of perifocal, there is a productive tissue reaction with subsequent fibrosis.

P. Generalized candidiasis, with hematogenous spread, there are either isolated metastatic foci (in the brain, kidneys, heart, bone marrow, striated muscles), or widespread metastases in many organs. Forms of generalized candidiasis:

- candidal septicemia;
- candidal septic endocarditis;
- chronosepsis;
- chronic granulomatous candidiasis of children.

Cryptococcosis(European blastomycosis, Brusse-Buschke disease, torulosis) - the causative agent is a blastomycete - a yeast-like fungus that has a gelatinous capsule that delays the migration of leukocytes to it, so the early forms of the lesion are characterized by an almost complete absence of an inflammatory reaction.

Later, polymorphic granulomas are formed, consisting of epithelioid and giant polynuclear cells, containing a small amount of the pathogen, including and its degenerative forms.

The primary object of the lesion is the skin and lungs, from where hematogenous metastasis to the central nervous system (into the substance of the brain and meninges) is characteristic, further metastasis leads to vascular lesions.

North American blastomycosis (Gilchrist's blastomycosis) is a chronic mycosis of the skin of granulomatous and ulcerative nature, which is complicated by metastases in internal organs, and disseminated forms may occur. Primary damage to the lungs (and not the skin) is possible.

Histoplasmosis(Darming's disease, reticuloendothelial cytoplasmosis) deep mycosis, can be in a generalized or localized form. A characteristic lesion of the mononuclear macrophage system: the pathogen is localized in macrophages, giant multinucleated cells, and cells of the macrophagehistiocytic system of the spleen, liver, lymph nodes, and bone marrow.

Three clinical and morphological forms:

1. Benign pulmonary histoplasmosis, both primary, caused by inhalation of fungal spores, and secondary, which develops as a result of lympho-hematogenous spread of the fungus from other organs;
2. Primary histoplasmosis of mucous membranes and skin;
3. Disseminated.

Three types of tissue changes are possible:

- intracellular (in histiocytes) localization of the pathogen without a tissue reaction;
- hyperplasia of reticuloendothelial cells containing pathogens, which may be accompanied by perifocal necrosis;
- granulomatous inflammation (epithelioid-cellular granulomas with single giant cells and a significant admixture of leukocytes).

Coccidiosis- extremely deep mycosis, which is rare, occurs in two stages:

- primary - the lungs, skin and gastrointestinal tract are affected
- secondary - has a chronic, malignant, progressive course with the one that generalizes the process, accompanied by the defeat of lymph nodes, internal organs, central nervous system and bones.

The tissue forms of Coccidioides immitus are spherical in shape and contain endospores. Morphological changes in the affected tissues are related to the life cycle of the fungus. In response to the rupture of the spherule and the release of endospores, an acute purulent inflammation develops, as they mature, a tubercle-like granuloma with few giant cells forms perifocally in the spherule. After maturation and rupture of the spherule, in response to the release of endospores, macrophage leukocyte infiltration is observed again. In the chronic course of the disease, the growth of granulation tissue with the phenomena of fibrosis is observed, and the causative agent occurs more often in the form of deformed and devastated spherules.

Rhinosporoidosischaracterized by papillomatous growths on the mucous membranes. The tissue reaction to the pathogen is expressed by productive inflammation with the development of granulation tissue: polypous inflammation develops in the mucous membranes of the nasal cavity and nasopharynx, less often it is localized in the mucous membrane of the vulva, urethra, and in the skin.

Aspergillosis. Aerobic fungi of the genus Aspergillus constantly vegetate in the soil. Can cause damage to the skin and respiratory tract of a person. In the tissues of the host, they are found in the form of uniformly septated hyphae (threads), grow radially, fan-like, as they divide dichotomously, but can form mycelium balls. Fungal threads are found among necrotized tissues, and a leukocyte reaction develops around the necrosis zone, and leukocytes are characterized by karyopyknosis and karyorrhexis. With a prolonged course, granulomas are formed - a productive inflammatory reaction develops around the zone of necrosis or abscess with the accumulation of histiocytes and fibroblasts, giant and a small number of epithelioid cells are present. In the center of the granuloma, only fragments of the cells of the fungus are found. Visceral forms of aspergillosis are divided into:

- bronchopulmonary (bronchitis, pneumonia);
- cerebral;
- gastrointestinal;

- urogenital;
- generalized.

The most characteristic pulmonary aspergillosis is distinguished by four types:

1. Non-purulent pulmonary aspergillosis with the formation of gray-brown dense infiltrative foci with a whitish center, where a cluster of fungi is determined among the infiltrate;
2. Purulent pulmonary aspergillosis with focal necrosis and suppuration;
3. Aspergillema (aspergillosis-lycetoma) of the lung - a spherical growth of the fungus, which is formed as a result of its growth on the inner surface of the cavity (bronchoectatic, or abscess cavity), forms wrinkled membranes that fit into this cavity;
4. Tuberculous pulmonary aspergillosis with the formation of granulomas similar to epithelioid tubercles.

Aspergillosis is often associated with chronic lung diseases (bronchitis, bronchiectasis, abscess, lung cancer, fibrous-cavernous tuberculosis), and the wall of the bronchus or cavity is lined with a thin layer of mold. CNS damage is observed when the process spreads from the air sinuses and orbit of the eye as a result of hematogenous metastasis.

Generalized aspergillosis develops during hematogenous spread more often in bronchopulmonary forms and is characterized by single or multiple metastatic foci in internal organs.

Mucormycosis (mucorosis). Deep chronic mycosis, which, in addition to superficial damage, damages the respiratory organs. With hematogenous generalization, the process is accompanied by tissue and organ infarctions.

Inflammation is manifested in weak leukocyte and lymphocyte infiltration with a significant content of eosinophils, necrotic processes prevail. Visceral forms of mucorosis quickly end in death, so granulation tissue rarely develops. If this happens, it is characterized by a large number of histiocytes and giant cells. In connection with the growth of fungi in vessels, thromboangitis and mycotic thrombosis develop, which leads to the development of multiple heart attacks. Moreover, ascending thrombosis is characteristic. The growing ascending thrombosis in the pulmonary arteries reaches the main trunk of the pulmonary artery, the ascending thrombosis of the vessels of the eye socket and additional nasal cavities contributes to the transition of the process to the brain tissue, as a result of which necrosis and hemorrhage are formed.

Echinococcosis -human and animal disease caused by the larval stage (finnoy) of echinococcus from the class of tapeworms and the family of tapeworms. Etiology. Eshinococcus granularis, which causes the hydatid form of echinococcosis, and Eshinococcus milticularis, which causes the alveolar form of echinococcosis, or alveococcosis, are of the greatest importance in human and animal pathology. Hydatidous echinococcosis is more common than alveococcosis. Epidemiology and pathogenesis. In the development of hydatidous echinococcosis in humans, the obligate host of sexually mature tapeworms - the dog, in which the parasite lives in the intestines - plays an important role. The alveococcus larva, which differs from the hydatidous echinococcus larva, is found in rodents and humans. Humans are likely to become infected when handling rodent skins. Pathological anatomy. With hydatid echinococcosis, bubbles (or single bubbles) of one size or another (from a walnut to the head of an adult) appear in the organs. They have a whitish layered chitinous shell and are filled with a transparent colorless liquid. There is no protein in the liquid, but it contains succinic acid. From the internal germinative layering of the bladder, daughter bladders with scolexs arise. These daughter cysts fill the chamber of the mother cyst (unicameral echinococcus). The tissue of the organ in which the single-chamber echinococcus develops undergoes atrophy. At the border with the echinococcus, connective tissue grows, forming a capsule around the bubble. Vessels with thickened walls and foci of cellular infiltration with an admixture of eosinophils are found in the capsule. In the areas of the capsule directly adjacent to the chitinous membrane, with giant cells of foreign bodies appear, phagocytizing elements of this shell. More often, the echinococcal cyst is found in the liver, kidneys, lungs, less often - in other organs. Cytoplasmic outgrowths are formed in alveococcal vesicles, and the growth of the vesicles occurs by budding outward, and not inside the mother vesicle, as is the

case with unicameral echinococcus. As a result, in alveococcosis, more and more bubbles are formed, penetrating the tissue, which leads to its destruction. Therefore, alveococcus is also called multichambered echinococcus. Therefore, the growth of alveococcus has an infiltrating character and is similar to the growth of a malignant neoplasm. Toxic substances released from the bubbles cause necrosis and a productive reaction in the surrounding tissues. There are many eosinophils and giant cells of foreign bodies in the granulation tissue, phagocytizing membranes of dead bubbles. Alveococcus is primarily found more often in the liver, less often in other organs. In the liver, it occupies a whole portion. It is very dense (the density of a board), on a section it has a porous appearance with layers of dense connective tissue. A decay cavity sometimes forms in the center of the node. Alveococcus is prone to hematogenous and lymphogenic metastasis. Hematogenous metastases of alveococcus at its primary localization in the liver appear in the lungs, then in the organs of the large blood circulation (kidneys, brain, heart, etc.). in this regard, alveococcus clinically behaves as a malignant tumor. Complications in echinococcosis are more often associated with the growth of a bubble in the liver or metastases of alveococcus. The development of amyloidosis is possible. Alveococcus is primarily found more often in the liver, less often in other organs. In the liver, it occupies a whole portion. It is very dense (the density of a board), on a section it has a porous appearance with layers of dense connective tissue. A decay cavity sometimes forms in the center of the node. Alveococcus is prone to hematogenous and lymphogenic metastasis. Hematogenous metastases of alveococcus at its primary localization in the liver appear in the lungs, then in the organs of the large blood circulation (kidneys, brain, heart, etc.). in this regard, alveococcus clinically behaves as a malignant tumor. Complications in echinococcosis are more often associated with the growth of a bubble in the liver or metastases of alveococcus. The development of amyloidosis is possible. Alveococcus is primarily found more often in the liver, less often in other organs. In the liver, it occupies a whole portion. It is very dense (the density of a board), on a section it has a porous appearance with layers of dense connective tissue. A decay cavity sometimes forms in the center of the node. Alveococcus is prone to hematogenous and lymphogenic metastasis. Hematogenous metastases of alveococcus at its primary localization in the liver appear in the lungs, then in the organs of the large blood circulation (kidneys, brain, heart, etc.). in this regard, alveococcus clinically behaves as a malignant tumor. Complications in echinococcosis are more often associated with the growth of a bubble in the liver or metastases of alveococcus. The development of amyloidosis is possible. in section it has a porous appearance with layers of dense connective tissue. A decay cavity sometimes forms in the center of the node. Alveococcus is prone to hematogenous and lymphogenic metastasis. Hematogenous metastases of alveococcus at its primary localization in the liver appear in the lungs, then in the organs of the large blood circulation (kidneys, brain, heart, etc.). in this regard, alveococcus clinically behaves as a malignant tumor. Complications in echinococcosis are more often associated with the growth of a bubble in the liver or metastases of alveococcus. The development of amyloidosis is possible. in section it has a porous appearance with layers of dense connective tissue. A decay cavity sometimes forms in the center of the node. Alveococcus is prone to hematogenous and lymphogenic metastasis. Hematogenous metastases of alveococcus at its primary localization in the liver appear in the lungs, then in the organs of the large blood circulation (kidneys, brain, heart, etc.). in this regard, alveococcus clinically behaves as a malignant tumor. Complications in echinococcosis are more often associated with the growth of a bubble in the liver or metastases of alveococcus. The development of amyloidosis is possible. then in the organs of the large circle of blood circulation (kidneys, brain, heart, etc.). in this regard, alveococcus clinically behaves as a malignant tumor. Complications in echinococcosis are more often associated with the growth of a bubble in the liver or metastases of alveococcus. The development of amyloidosis is possible. then in the organs of the large circle of blood circulation (kidneys, brain, heart, etc.). in this regard, alveococcus clinically behaves as a malignant tumor. Complications in echinococcosis are more often associated with the growth of a bubble in the liver or metastases of alveococcus. The development of amyloidosis is possible.

Cysticercosis- chronic helminthiasis, which is caused by the larval stage (finnoy) of the armed (pork) hookworm (solitary).

*Etiology, epidemiology, pathogenesis.*The disease develops in humans, as well as in some animals (pigs, dogs, cats), which are intermediate hosts of the parasite and its phynozoic stage. Animals become infected by eating human feces containing helminth eggs. A person becomes infected by consuming pig meat, in which the finna (pisticercus) is a parasite. The development of finna into an adult parasite occurs in the human intestine. Humans can develop cysticercosis when parasitised by the pig solitaire in the intestines. This happens when the solitaire eggs fall into the stomach, where their shell dissolves, the embryos penetrate through the stomach wall into the lumen of the vessels, are transferred to various tissues and organs, where they turn into cysticerci. Pathological anatomy. A cysticercus has the appearance of a bubble the size of a pea. A head with a neck extends inward from its wall. An inflammatory reaction develops around the cysticercus. The infiltrate consists of lymphocytes, plasma cells, fibroblasts and eosinophils. Young connective tissue gradually appears around the infiltrate, which matures and forms a capsule around the cysticercus. In brain tissue, microglial cells participate in the formation of the capsule around the cysticercus. Over time, the cysticercus dies and calcifies.

Opisthorchosis- a disease of humans and mammals caused by parasites of the trematode species. The first description of the morphology of opisthorchosis belongs to the Russian pathologist K. N. Vinogradov (1891).

*Etiology.*For humans, the invasion of the cat fluke is of the greatest importance. Epidemiology and pathogenesis. Humans and carnivores infected with opisthorchiasis are a source of infection for Bithynia molluscs, which ingest the eggs of the parasite that have entered the water with the feces of sick people and animals. In the body of molluscs, the larval stages of the helminth multiply, which ends with the release of cercariae into the water. They penetrate through the skin of fish into their subcutaneous tissue and muscles, transforming here into metacercariae. Infection with opisthorchosis in humans and mammals occurs when raw fish with helminth larvae (metacircarchia) are consumed.

*Pathological anatomy.*The main changes develop in the bile ducts and liver parenchyma. Inflammation develops in the intrahepatic bile ducts, where a large number of parasites are located. The duct walls are infiltrated with lymphoid elements, plasma cells, and eosinophils. The epithelium forms reactive growths with the formation of iron structures in the subepithelial layer. As a result, sclerosis of duct walls and periductal sclerosis develops. Necrosis particles appear in the liver parenchyma, which are replaced by growing connective tissue. Sclerotic changes in the liver have a focal nature and are associated with the predominant localization of parasites in the biliary tract. Inflammation also occurs in the wall of the gallbladder. In the pancreas, the expansion of the ducts in which clusters of helminths, hyperplasia of the mucous membrane are found, inflammatory infiltrates in the wall of the ducts and in the stroma of the gland. Complications are associated with the addition of a secondary infection of the biliary tract, which leads to the development of purulent cholangitis and cholangiolitis. With a long course of opisthorchosis, cirrhosis of the liver is possible. Cholangiocellular liver cancer sometimes develops as a result of prolonged and perverse proliferation of the bile duct epithelium.

Schistosomiasischronic helminthiasis with predominant damage to the genitourinary system and intestines.

*Etiology.*The causative agent of this helminthosis in humans is schistosomes from the group of trematodes: Schistosoma haematobium, Schistosoma mansoni and . Schistosoma japonicum. Schistosoma haematobium causes genitourinary schistosomiasis, which was first discovered by Bilharts and is therefore called bilhartsiosis.

*Epidemiology and pathogenesis.*The eggs of the parasite go through their development cycle in the body of freshwater molluscs to the stage of cercariae, which are introduced through the skin into the human body. Cercariae mature very quickly and turn into schistosomula, penetrating into the peripheral veins, where sexually mature individuals are formed. From here, fertilized females go to

their favorite place of residence: pelvic veins, mesenteric and hemorrhoidal veins, as well as the wall of the large intestine. Here, females lay eggs, which causes tissue damage. Part of the eggs is excreted with urine and feces into the external environment, being a source of spread of helminthiasis. Outbreaks of urinary schistosomiasis are mainly found in Africa. Schistosoma mansoni is found in South and Central America, Africa, Schistosoma haematobium - in Japan and Southeast Asian countries.

*Pathological anatomy.*With schistosomiasis, changes are observed first of all in the places where eggs are laid, that is, in the bladder, the wall of the large intestine. The most common is urinary schistosomiasis, which affects the bladder. In the early period of the disease, inflammation, hemorrhages, desquamation of the epithelial layer develop in the surface layers of the mucous membrane of the urinary bladder. Then the changes spread to the deeper layers of the wall. Leukocyte infiltrates appear in the submucosal layer around schistosomiasis eggs, they cover the entire thickness of the mucous membrane, in which ulcers form. Over time, the exudative tissue reaction changed to a productive one, a granulation tissue with a large number of epithelioid cells formed around the eggs, and a schistosome granuloma formed. The process acquires a chronic course, the result of which is sclerosis and deformation of the bladder wall. Dead eggs are calcified. The spread of the parasite in the veins of the small pelvis leads to the appearance of lesions in the prostate gland, epididymis. With slow healing of bladder ulcers and a long course of the disease, the development of cancer is possible. In case of schistosomiasis of the large intestine, similar changes develop in the walls, ending with sclerosis. There are cases of schistosome appendicitis. Hematogenous spread of the process is possible. Parasites enter the liver, lungs, and brain, and inflammatory infiltrates of lymphocytes, neutrophils, and epithelioid cells appear at the site of their introduction. Granulation tissue is quickly formed, sclerosis develops. With slow healing of bladder ulcers and a long course of the disease, the development of cancer is possible. In case of schistosomiasis of the large intestine, similar changes develop in the walls, ending with sclerosis. There are cases of schistosome appendicitis. Hematogenous spread of the process is possible. Parasites enter the liver, lungs, and brain, and inflammatory infiltrates of lymphocytes, neutrophils, and epithelioid cells appear at the site of their introduction. Granulation tissue is quickly formed, sclerosis develops. With slow healing of bladder ulcers and a long course of the disease, the development of cancer is possible. In case of schistosomiasis of the large intestine, similar changes develop in the walls, ending with sclerosis. There are cases of schistosome appendicitis. Hematogenous spread of the process is possible. Parasites enter the liver, lungs, and brain, and inflammatory infiltrates of lymphocytes, neutrophils, and epithelioid cells appear at the site of their introduction. Granulation tissue is quickly formed, sclerosis develops. brain and inflammatory infiltrates of lymphocytes, neutrophils, and epithelioid cells appear at the place of their introduction. Granulation tissue is quickly formed, sclerosis develops. brain and inflammatory infiltrates of lymphocytes, neutrophils, and epithelioid cells appear at the place of their introduction. Granulation tissue is quickly formed, sclerosis develops.

Trichinosis -chronic helminthiasis with a predominant lesion of the striated muscles, where the young forms of the parasite are localized.

*Etiology and pathogenesis.*The causative agent of the disease is Trichinella spiralis. Human infection occurs when consuming pig meat infected with trichinella. Pathological anatomy. Trichinosis is characterized by typical changes in the striated muscles. However, they are affected unevenly. Thus, the respiratory muscles, diaphragm, masticatory muscles, pharyngeal muscles, oculomotor muscles, etc. are most intensely affected. At a macroscopic examination, it can be seen that the muscles are dotted with very small nodules, sometimes yellowish, then whitish, soft or dense up to calcified. Trichinella in various stages of development are detected microscopically in the muscles. It has been established that young trichinella bore the sarcolemma, penetrate the muscle fiber, grow, twist into a spiral, and the muscle fiber itself swells, the transverse striae disappears. After the death of ulcer fibers, an inflammatory infiltrate consisting of histiocytes, lymphocytes, and single eosinophilic leukocytes appears around them. A picture of trichinellosis myositis develops. Gradually, a

connective tissue capsule forms around the spirally bent parasite, the parasite dies and calcifies. From all of the above, it follows that trichinella, which has entered the muscle fibers of a person, inevitably dies and its epidemiological role in the spread of the disease ends. Trichinella are carried by the blood stream not only into the muscles, but can get stuck in the capillaries of internal organs, causing inflammatory reactive changes around them. Gradually, a connective tissue capsule forms around the spirally bent parasite, the parasite dies and calcifies. From all of the above, it follows that trichinella, which has entered the muscle fibers of a person, inevitably dies and its epidemiological role in the spread of the disease ends. Trichinella are carried by the blood stream not only into the muscles, but can get stuck in the capillaries of internal organs, causing inflammatory reactive changes around them. Gradually, a connective tissue capsule forms around the spirally bent parasite, the parasite dies and calcifies. From all of the above, it follows that trichinella, which has entered the muscle fibers of a person, inevitably dies and its epidemiological role in the spread of the disease ends. Trichinella are carried by the blood stream not only into the muscles, but can get stuck in the capillaries of internal organs, causing inflammatory reactive changes around them.

Ascariasis- chronic helminthiasis with localization of the parasite in the small intestine.

*Etiology and pathogenesis.*The causative agent of the disease - roundworms (Azsapz 1шпЬпсо1с1е5) belong to the class of nematodes, of different sexes. The female lays a huge number of eggs in the intestines of the patient, which are released into the external environment with feces. A person becomes infected by ingesting roundworm eggs with water or contaminated food. In the small intestine, the egg shell dissolves and the released larva, penetrating into the blood and lymphatic vessels, enters the lungs and is released into the lumen of the respiratory tract (migratory stage of ascariasis, migroascaridosis). Microascaridosis continues for 7-15 days after infection. Then the larvae are swallowed with saliva, again end up in the intestines, where mature roundworms develop (intestinal stage of ascariasis). The pathological anatomy of ascariasis is mainly related to the presence of ascaris in the small intestine. Their number can reach several hundreds. Adult roundworms can penetrate into the excretory ducts of the pancreas and liver, into the lumen of the appendix and cause inflammatory processes in them. If too many roundworms accumulate in the lumen of the small intestine, intestinal obstruction or, in some cases, breakthrough of the wall of the small intestine by roundworms can occur. During the migration of the larvae, hemorrhages are observed in the lungs. Sometimes, during the migration of ascaris larvae, quickly appearing and disappearing foci of inflammation ("volatile infiltrates" in the terminology of clinicians) appear in the lungs, which are accompanied by a small discharge of sputum containing a huge number of eosinophils. There is eosinophilia in the blood, a rash on the skin. Morphologically, in these cases, acinous-lobular foci of inflammation, rich in eosinophils, are found in the lungs. Ascaris larvae are usually not detected in areas of inflammation.

Malaria- an acute or chronic recurrent infectious disease that has different clinical forms depending on the maturation period of the causative agent.

*Etiology and pathogenesis.*The disease is caused by Plasmodium, discovered in erythrocytes by Laveran (1880). The vector of the pathogen is the mosquito (Anopheles). Having entered the blood during a mosquito bite, plasmodia go through a complex development cycle, parasitize in human erythrocytes, reproducing asexually, which is called schizogony. Parasite schizonts accumulate particles of a dark brown pigment - hemomelanin - in the cytoplasm. During hemolysis, parasites and hemelanin are released from the erythrocyte. Moreover, the pigment is phagocytosed by the cells of the macrophage system, and the schizonts are again introduced into the erythrocytes. In this connection, hemolytic anemia, hemelanosis, and hemosiderosis of elements of the reticuloendothelial system develop, ending with sclerosis. During periods of hemolytic crisis, acute vascular disorders (stasis, diapedesis hemorrhages) appear. in connection due to persistent antigenemia in malaria, toxic immune complexes appear in the blood. Their action is associated with damage to the microcirculatory channel (increased permeability, hemorrhages), as well as the development of

glomerulonephritis. There are several types of malaria plasmodium, which differ in terms of their maturation. In this regard, three-day, four-day and tropical forms of malaria are distinguished.

*Pathological anatomy.*In the three-day, most frequent form of malaria, anemia develops in connection with the destruction of erythrocytes, the severity of which is aggravated by the property of plasmodia of three-day malaria to settle in young erythrocytes - reticulocytes (M.V. VoynoYasenetsky). Products released during the breakdown of erythrocytes, especially hemelanin, are captured by the cells of the macrophage system, which leads to an increase in the spleen and liver, bone marrow hyperplasia. Organs loaded with pigment acquire a dark gray, and sometimes black, color. The spleen increases especially quickly, first as a result of full blood, and then - hyperplasia of cells that phagocytose pigment. In this connection, its pulp becomes dark, almost black. In the acute stage of malaria, the spleen is loose, full-blooded, in the chronic stage, it becomes dense as a result of the developing sclerosis; its weight reaches 3-5 kg (malarial splenomegaly). The liver is enlarged, full-blooded, with a gray-black surface on the section. Marked hyperplasia of stellate reticuloendotheliocytes with hemomelanin deposition in their cytoplasm. In chronic malaria, the thickening of the liver stroma and the growth of connective tissue in it are emphasized. The bone marrow of flat and tubular bones has a dark gray color, the hyperplasia of its cells and the deposition of pigment in them are emphasized. There are areas of bone marrow aplasia. Hemomelanosis of the organs of the reticuloendothelial system is combined with their hemosiderosis, suprahepatic (hemolytic) jaundice also develops. The pathological anatomy of fourday and three-day malaria is similar. In tropical malaria, the changes are little different from those described in the three-day form, although they have some peculiarities. They are explained by that erythrocytes containing maturing schizonts of tropical malaria accumulate in the terminal areas of the bloodstream, which leads to the development of parasitic stasis. In places of accumulation of maturing schizonts during the period of their division into merozoites, neutrophils and macrophages phagocytize both infected erythrocytes and immature schizonts, as well as decay products and pigment, plasmodia that appear after division (M. V. Voyno-Yasenetsky). Parasitic stasis is associated with life-threatening changes in the brain, which are observed in malarial coma. In such cases, the cortex and other areas of the gray matter of the brain have a dark brown (smoky) color. In the white matter, there are numerous point hemorrhages that surround vessels filled with agglutinating erythrocytes, with parasites in the cytoplasm or hyaline thrombi. Around such vessels, in addition to hemorrhagic ones, 1.5-2 days after the onset of coma, the reactive growth of glial cells occurs, which leads to the formation of peculiar nodules - the so-called Durk's granuloma.

A complication of acute malaria can be glomerulonephritis, chronic - exhaustion, amyloidosis.

Death is usually observed in tropical malaria complicated by coma.

Amoebiasis- a chronic parasitic disease based on ulcerative colitis.

*Etiology and pathogenesis.*Amebiasis is caused by protozoans from the class of rhizopods - Entamoeba hystolytica. The causative agent was discovered by F.A. Lesh (1875) in the stools of patients with amebiasis. The disease occurs mainly. image in countries with a hot climate. Infection occurs through the alimentary route with encysted amoebae, protected from the action of digestive juices by a special membrane that melts in the cecum, where the most pronounced morphological changes are usually observed. The histological properties of the amoeba explain its deep penetration into the intestinal wall and the formation of non-healing ulcers. What conditions contribute to the transition of the carrier to the disease remains unclear.

*Pathological anatomy.*Getting into the wall of the large intestine, the amoeba and its waste products cause swelling and histolysis, mucosal necrosis, and ulcer formation. Areas of necrosis of the mucous membrane slightly protrude above its surface, they are colored dirty gray or green. The section shows that the zone of necrosis penetrates deep into the submucosal and muscle layers. When an ulcer is formed, its edges become undercut and hang over the bottom. As the necrosis progresses, the size of the ulcer increases. Amoebae are found at the border between dead and preserved tissues. It is characteristic that the cellular reaction in the intestinal wall is weakly expressed. However, as the

secondary infection joins, a leukocyte reaction occurs, pus appears. Sometimes a phlegmonous and gangrenous form of colitis develops. Deep ulcers heal with a scar. However, it is not uncommon for Regional lymph nodes are somewhat enlarged, but amoebas are not detected in them, amoebas are usually found in the blood vessels of the intestinal wall.

Complications of amoebiasis are divided into intestinal and extraintestinal. Of the intestinal ones, the most dangerous are breakthrough ulcers, bleeding, and the formation of body infiltrates around the affected intestine, which often stimulate the tumor. The most dangerous of extraintestinal complications is the development of a liver abscess.

Balantidiasis.characterized by the development of chronic ulcerative colitis. An isolated lesion of the appendix is rarely emphasized.

*Etiology.*The causative agent of balantidiasis is Valantidium coli infusoria.

Pathological anatomy. Changes in balantidiasis are similar to those in amebiasis, but in balantidiasis, which is much less common than amebiasis, the intestinal damage is not so pronounced. First, there is damage to the surface layers of the mucous membrane with the formation of erosions. Later, as the balantidium penetrates into the submucosal layer, ulcers of different sizes develop and form; their edges are undermined, gray-dirty remnants of necrotic masses are visible at the bottom. Balantidium are usually found in the vicinity of foci of necrosis, as well as in the crypts and thickness of the mucous membrane far from ulcers. They can penetrate into the muscle layer, into the lumen of lymphatic and blood vessels. Local cellular reactions in balantidiasis are weakly expressed, eosinophils predominate among the cells of the infiltrate. The most important complications of balantidiasis are ulcer rupture with the development of peritonitis. Joining the ulcerative process of a secondary infection can lead to septicopyemia.

Toxoplasmosis- a disease that is caused by simple unicellular microorganisms and occurs both in humans and animals. By origin, toxoplasmosis can be congenital or acquired. Congenital toxoplasmosis affects newborns and young children, acquired - older children and adults. It has been established that toxoplasmosis is widespread among the population of many countries, and a large number of people suffering from abortive and latent forms are emphasized.

*Etiology*toxoplasmosis is established. The causative agent of the disease is toxoplasma (from the Greek toxon - arc), which has an arc-shaped shape. For the first time, toxoplasma was discovered in 1908 by Nicole and Manso in Gonda rodents.

*Pathogenesis.*The ways of toxoplasma penetration into the body have not been definitively established. It is suggested that parasites are introduced through damaged skin or mucous membranes (for example, gastrointestinal tract, respiratory tract), through bites of blood-sucking ectoparasites. A person usually gets infected from dogs or cats if hygiene rules are not followed in dealing with them. Transmission of the disease from person to person is possible only through the placenta of the fetus from the infected mother, which is observed in congenital toxoplasmosis. Getting into the bloodstream, toxoplasmas spread throughout the body, penetrate the cytoplasm of cells of various organs and multiply by simple division. Cells containing toxoplasmas increase in size and are called pseudocysts. When the pseudocysts are destroyed, the parasites enter the bloodstream again and infect new cells.

Pathological anatomy. Congenital toxoplasmosis is accompanied by changes in the brain and eye membranes. Foci of necrosis appear in the brain and eye membranes, which quickly calcify. In the brain, these foci of necrosis have a yellowish color, the size of a millet grain to a pea and are located in the cortex of the large hemispheres and in the subependymal zone of the lateral ventricles of the brain. Reactive, mostly productive, inflammation occurs around foci in the brain tissue, as well as in adjacent areas of soft meninges and in the ependyma. On this soil, adhesive processes develop in the meninges, ventricles of the brain, the circulation of liquid is disturbed and hydrocephalus occurs. It gradually increases, and by the time the fetus is born, it can reach a significant degree and lead to atrophy of the brain substance. Upon microscopic examination, toxoplasmosis pseudocysts and free-lying toxoplasmas are found in fresh foci of necrosis. Gradually, pseudocysts and masses of necrosis

become calcified. In the eye, foci of necrosis are formed in the retina and vascular tract, accompanied by productive inflammation. Formation of connective tissue and deformation of eye tissues. Infection of the fetus in the early stages of pregnancy leads to delayed brain development and improper formation of other organs. Thus, toxoplasmosis is one of the etiological factors in the formation of ugliness. Formation of connective tissue and deformation of eye tissues. Infection of the fetus in the early stages of pregnancy leads to delayed brain development and improper formation of other organs. Thus, toxoplasmosis is one of the etiological factors in the formation of ugliness. Formation of connective tissue and deformation of eye tissues. Infection of the fetus in the early stages of pregnancy leads to delayed brain development and improper formation of other organs. Thus, toxoplasmosis is one of the etiological factors in the formation of ugliness.

Acquired toxoplasmosis is characterized by weak necrotic phenomena and the predominance of productive inflammatory changes in the organs. With the septic form of acquired toxoplasmosis, meningoencephalitis, hyperplasia of the spleen and lymph nodes, and interstitial inflammatory processes in internal organs develop. Acquired toxoplasmosis often proceeds in a hidden form and is recognized only by immunological reactions. In some cases, women learn about toxoplasmosis only after childbirth, when the newborn is diagnosed with this disease.

1. Theoretical questions

Questions for self-control:

1. Malaria, balantidiasis, amoebiasis: morphogenesis, morphological characteristics, complications, consequences.
2. Trichinellosis, echinococcosis, cysticercosis, opisthorchosis, schistosomiasis: morphogenesis, morphological characteristics, complications, consequences.
3. Mycoses: pathological anatomy, complications, consequences in dermatomycoses.
4. Pathological anatomy, complications, consequences of visceral mycoses (actinomycosis, candidiasis, aspergillosis).

2 Practical tasks

72. Prepare an essay on the topic: "Malaria, balantidiasis, amoebiasis: morphogenesis, morphological characteristics, complications, consequences."

3. Test tasks for self-control:

4. Individual tasks

1. Make an outline on this topic

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory
- http://www.webpathology.com/- Web Pathology

Topic #20: "Preparation for the final control for the year.»

Purpose: as a result of independent study of this topic, students should prepare to pass the final examination for the year. .

Basic concepts:
The student should know:

85. The structural basis of the development of diseases and their clinical manifestations, the structural basis of recovery, complications and consequences.

86. Pathomorphological research methods: autopsy, biopsy, study of biopsy material.

87. Pathology of the cell and general pathological processes, the totality of which determines the manifestations of one or another disease;

88. Pathogenesis and pathological changes in diseases at different stages of their development (morphogenesis), structural bases of complications and consequences of the disease;

89. Consequences arising from changes in human living conditions and treatment and diagnostic and therapeutic manipulations.

The student should be able to:

81. Solve typical and complex specialized tasks and practical problems in the learning process, which involves research and/or innovation and is characterized by the complexity and uncertainty of conditions and requirements.

1. Theoretical questions

Questions for self-control:

1. Pathological anatomy as a science, a field of practical medicine and a subject of study. Problems of pathological anatomy.
2. Levels of research on the structural basis of diseases. Material (objects) and methods of pathomorphological research.
3. The main stages of the development of pathological anatomy. Contribution of domestic scientists to the development of world pathomorphology.
4. Concept of ultrastructural cell pathology. Damage to the cytoplasmic membrane, mitochondria, endoplasmic reticulum, Golgi apparatus, lysosomes. Reversible and irreversible nuclear damage.

Damage to mitosis, causes, types.

5. Definition of the term "dystrophy", causes of dystrophy. Pathogenesis and mechanisms of dystrophy.
6. Classification of dystrophy. Morphogenesis of parenchymal (intracellular) protein dystrophies. Morphogenesis and morphology of parenchymal fatty dystrophies (lipidoses). Morphogenesis and morphology of parenchymal carbohydrate dystrophies.
7. Stromal-vascular (extracellular) protein dystrophies, varieties, morphology, mechanisms, causes, outcomes.
8. Stromal-vascular lipidoses and carbohydrate dystrophies, varieties, morphology.
9. Definition of mixed dystrophy, classification. Classification of hemoglobinogenic pigments.

Types of violations of their exchange.

10. Violation of the metabolism of lipidogenic pigments.

11. Violation of nucleoprotein metabolism.
12. Disorders of calcium metabolism, types of calcinosis, its causes and morphology.
13. Definition of necrosis, its causes, types, depending on the mechanism of action of the pathogenic factor. Morphological signs of necrosis. Early morphological and histochemical changes. Morphological signs of necrosis in the nuclei. Morphological signs of necrosis in the cytoplasm and intercellular substance.
14. Clinical and morphological forms of necrosis. Coagulation necrosis, causes of development, types, microscopic and macroscopic changes in areas of necrosis. Enzymatic and non-enzymatic fat necrosis, localization, causes.
15. Gangrene, definition, classification. Dry gangrene, localization, macroscopic changes in necrotic tissue. Wet gangrene, localization, causes, morphological changes. Bedsores, features of development, localization.
16. Collective (wet) necrosis, location, macro-microscopic changes. Exits of necrosis.
17. Apoptosis, definition, morphological manifestations of apoptosis. The influence of external factors on the regulation of apoptosis. Categories of autonomous apoptosis.
18. Signs of general death, mechanisms and terms of their development.
19. General idea of edema, composition of tissue fluid, classification, localization of fluid accumulation. Local edema, its regulation, mechanism of development, types. General edema, its varieties and mechanisms of occurrence.
20. Dehydration of the body, mechanisms of development, degrees of dehydration.
21. Varieties of general arterial congestion. Local arterial congestion, types, causes, morphology. Pathomorphology, consequences of stasis.
22. General venous congestion, types, causes of development, changes in the lungs and liver in chronic venous congestion.
23. Blood thickening, causes, morphological changes in organs. Thinning of the blood, causes, meaning.
24. Bleeding, definition, causes of development, classification. Hemorrhage, types, morphology.
25. Shock, definition, classification. Stages of development of shock, morphological changes. Morphological changes of kidneys, lungs, liver, myocardium, stomach and intestines during shock. 26. Heart attack, definitions, causes. Types of heart attacks. Mechanisms of development and morphological changes in the infarct zone. Myocardial infarction, localization, morphology, outcome. The result of a heart attack.
27. Disorders of lymph circulation, causes, classification. Acute and chronic local lymphedema. Morphology of acute and chronic general lymphedema.
28. Thrombosis, definition, causes and mechanisms of thrombosis. Morphology and types of blood clots. Favorable and unfavorable outcomes of thrombosis.
29. Definition of DVZ syndrome, causes of occurrence. Stages of DVZ syndrome, morphological signs.
30. Definition of embolus, types of embolus. Ways of movement of emboli. Morphology of thromboembolism of the pulmonary artery and vessels of the great circle of blood circulation. 31. Definition of inflammation, etiology. Morphological signs of inflammation. Morphological changes during alteration, exudation and proliferation.
32. Classification of inflammation by morphology, course depending on the reactivity of the organism. Forms of exudative inflammation. Serous inflammation, etiology, localization, morphology, outcome.
33. Fibrinous inflammation: etiology, types, localization, morphology, outcomes.
34. Purulent inflammation: etiology, forms, localization, morphology, outcomes.
35. Non-independent forms of inflammation. Catarrhal inflammation; etiology, localization, types, morphology, exits. Hemorrhagic and purulent inflammation: etiology, morphology, outcomes. 36. General characteristics of productive inflammation, classification, outputs. Intermediate (interstitial) inflammation, morphology, outcome. Granulomatous inflammation, definition of

granuloma, etiology, stages of granuloma. Productive inflammation with the formation of polyps and acute condylomas; localization, etiology, consequences.

37. Primary organs of immunogenesis, their role in the development of immune reactions. Secondary organs of immunogenesis, their role in the development of immune reactions. Types of lymphocytes, their localization in organs of immunogenesis, functional features. Types of immune reactions.

38. Definition of immunopathological processes, classification. Violations of immunogenesis associated with pathology of the thymus and pathology of peripheral lymphoid tissue. 39. Mechanisms of development of hypersensitivity reactions of immediate and delayed type. Classification of hypersensitivity reactions. Morphological characteristics of delayed-type hypersensitivity (HST) and immediate-type hypersensitivity (HNT) reactions. Morphological characteristics of the reaction of transplant rejection.

40. Definition and classification of autoimmune diseases. Characteristics of organ-specific autoimmune diseases. Diseases with autoimmune disorders, mechanisms of appearance of autoantigens.

41. Amyloidosis, chemical composition and physical properties of amyloid. Classification of amyloidosis. Characteristics of primary, hereditary (genetic), secondary, localized and senile amyloidosis. Types of amyloidosis depending on the specificity of the fibril protein. Methods of micro- and macroscopic detection of amyloid. Appearance of organs in amyloidosis, result. 42. Classification of immunodeficiency states. Classification of primary immunodeficiency syndromes. Combined immunodeficiency syndromes, types, state of organs of immunogenesis, clinical manifestations. Syndromes of insufficient cellular immunity, state of organs of immunogenesis, clinical manifestations. Humoral immunity deficiency syndromes, state of organs of immunogenesis, clinical manifestations.

43. Reasons for the development of secondary immunodeficiency states.

44. Definition of regeneration, classification. Regulation and phases of the regenerative process. Characteristics of physiological regeneration. Types of reparative regeneration. Characteristics of complete regeneration. Characteristics of incomplete regeneration. Pathological regeneration, conditions of occurrence, types.

45. Blood regeneration.

46. Regeneration of blood and lymphatic vessels.

47. Regeneration of connective and adipose tissue. Pathological regeneration of connective tissue. Regeneration of smooth and skeletal muscles.

48. Regeneration of bone tissue, regeneration conditions, characteristics of an uncomplicated bone fracture. Morphological characteristics of secondary bone fusion.

49. Regeneration of cartilage tissue. Regeneration of the epithelium. Regeneration of the brain and spinal cord. Regeneration of peripheral nerves.

50. Types of wound healing. Wound healing by primary and secondary tension.

51. The concept of compensation and adjustment. Stages of the compensatory process. Manifestations of adaptive processes. Hypertrophy and hyperplasia, definition, classification.

52. Working (compensatory) hypertrophy, causes of development. Characteristics of cardiac hypertrophy, causes, macro- and microscopic changes. Vicarious hypertrophy, conditions of development. Neurohumoral hypertrophy and hyperplasia. True and false hypertrophy, morphological changes in organs.

53. Atrophy, definition, classification. Types of general pathological atrophy, morphological changes in organs, appearance of patients. Types of local pathological atrophy, causes, morphology.

54. Definition of organization, encapsulation, cirrhosis and sclerosis, morphology.

55. Metaplasia and dysplasia, definition, morphological characteristics. Degrees of dysplasia.

56. Tumors, definitions, modern theories of carcinogenesis. Mechanisms of blastomatous action of pathogenic agents.

57. Tumor morphogenesis, morphogenetic variants of tumor formation. Structure of the tumor. Types of tumor growth.
58. Tumor atypism, definitions, types. Morphological characteristics of tissue and cellular atypism. Precancerous (precancerous) conditions and changes, morphology. Metastasis: types, regularities, mechanisms. Relapse, definition.
59. Modern classification of tumors. Morphological features of benign tumors. Morphological features of malignant tumors.
60. General characteristics and nomenclature of tumors from tissues originating from mesenchyme. Benign and malignant tumors from connective tissue, muscle tissue, blood and lymphatic vessels. Benign bone-forming and cartilage-forming tumors. Benign and malignant tumors from adipose tissue.
61. Classification and morphological features of tumors of the central nervous system. Benign neuroectodermal tumors. Low-differentiated and embryonic neuroectodermal tumors.
62. Benign and malignant tumors of the meninges.
63. Mature and immature tumors of peripheral nerves. Benign and malignant tumors of sympathetic ganglia.
64. Nomenclature of tumors that develop from melanin-producing tissue. Nevi - definition, classification, morphology. Melanoma, stages of development. Different types of melanoma morphology.
65. Nomenclature of epithelial tumors. Morphological features of epithelial tumors without specific localization. Benign and malignant tumors from the covering epithelium.
66. Benign and malignant tumors of the liver. Benign tumors of the stomach and intestines from Kulchytsky's enterochromaffin cells.
67. Organ-specific tumors of the thyroid gland, kidneys, skin: benign and malignant 68. Benign and malignant uterine tumors, types, morphology
69. Tumors of salivary glands and oral cavity.
70. Features of tumor growth in children compared to adults. Classification of childhood tumors. Dysontogenetic tumors in children. Theories of teratoma development, histological variants of teratoma. Morphological structure of mature and immature teratomas. Morphological features of hamartoma and hamartoblastoma.
71. Tumors in children arising from embryonic cambial tissues in the central nervous system, sympathetic ganglia and adrenal glands.
72. Hamartomas and hamartoblastomas of vascular origin. Hamartomas and hamartoblastomas of muscle tissue. Hamartoblastomas of internal organs: nephroblastoma (Willms tumor), hepatoblastoma. Definition of teratoma, typical localization. Sacrococcygeal teratoma and teratoblastoma. Peculiarities of the morphological structure. Ovarian and testicular teratomas.
73. Benign and malignant tumors in children, which develop according to the type of tumors in adults.
74. Organ-specific hormonally active tumors of the adrenal glands: classification, morphological features.
75. Benign and malignant breast tumors.
76. Definition, classification and morphological characteristics of anemias.
77. Definition, classification, morphological characteristics of thrombocytopenia and thrombocytopenia. Classification, morphological characteristics of coagulopathies.
78. Definition, classification, general morphological characteristics of leukemias.
79. Types, stages of the course, morphological characteristics of acute leukemia.
80. Types, stages of the course, morphological characteristics of chronic leukemia.
81. Pathohistological types, morphological characteristics of Hodgkin's disease, causes of death. General characteristics, classification, morphological manifestations and prognosis of nonHodgkin's lymphomas.
82. Definition of atherosclerosis, risk factors, modern theories.

83. Morphogenesis of macroscopic changes in atherosclerosis. Morphogenesis of microscopic changes in atherosclerosis. Clinical and morphological forms of atherosclerosis, organ lesions in atherosclerosis.
84. Definition, risk factors, connection of ischemic heart disease with atherosclerosis and hypertension. Morphology of acute, recurrent and repeated myocardial infarction. Consequences, complications, causes of death in myocardial infarction.
85. Morphological characteristics, complications, causes of death in chronic ischemic heart disease.
86. Hypertensive disease: definition, risk factors. Morphological changes in blood vessels, heart, changes in organs in hypertensive disease.
87. General characteristics of systemic diseases of connective tissue: disturbance of immune homeostasis and systemic progressive disorganization of connective tissue in rheumatic diseases.
88. Classification, morphogenesis, morphological characteristics of rheumatism. Endocarditis, myocarditis, pericarditis and pancarditis: classification, morphological characteristics, complications.
89. Morphology of Bekhterev's disease. Morphogenesis, pathomorphology, complications and causes of death in systemic lupus erythematosus. Pathological anatomy, visceral manifestations, complications, causes of death in systemic scleroderma. Pathological anatomy of dermatomyositis. Complications, causes of death.
90. Pathomorphology of systemic vasculitis: nonspecific aortoarteritis, nodular periarteritis, Wegener's granulomatosis, obliterating thromboangiitis.
91. Pathological anatomy of acquired heart defects. Pathological anatomy of acquired (secondary) cardiomyopathies.
92. General characteristics, classification, background diseases and risk factors of cerebrovascular disease. Infarct (ischemic stroke) of the brain: morphological characteristics. Morphological characteristics, consequences of hemorrhagic stroke.
93. Morphological characteristics, complications of spontaneous intracranial hemorrhage. Morphological characteristics, complications of spontaneous subarachnoid hemorrhage. 94. Morphological characteristics, complications of Alzheimer's disease. Morphological characteristics, complications of multiple sclerosis. Morphological characteristics, complications of amyotrophic lateral sclerosis.
95. Morphological characteristics, complications of postresuscitation encephalopathy.
96. Morphological characteristics, complications of diseases of the peripheral nervous system.
97. Morphological characteristics of acute bronchitis. Modern classification of pneumonia. Morphological characteristics and complications of acute focal pneumonia.
98. Morphological characteristics and complications of lobar pneumonia.
99. Morphological characteristics and complications of acute interstitial pneumonia. Morphological characteristics of acute destructive processes of the lungs.
100. Definition and classification of chronic non-specific respiratory diseases. Morphological characteristics and complications of chronic bronchitis.
101. Morphological characteristics of chronic obstructive emphysema. Morphological characteristics and complications of bronchiectasis.
102. Morphological characteristics and complications of bronchial asthma.
103. Morphological characteristics of idiopathic pulmonary fibrosis.
104. Morphological characteristics of lung cancer.
105. Diseases of the esophagus: morphological characteristics. Morphological characteristics of chronic gastritis. Pathomorphology of ulcer disease. Complications of ulcer disease.

106. Stomach cancer. Macroscopic and histological forms. Peculiarities of metastasis.
107. Pathomorphology of non-specific ulcerative colitis. Pathomorphology of Crohn's disease. Intestinal tumors.

108. Clinical and morphological forms of appendicitis. Complication of appendicitis. 109. Morphological characteristics, prognosis of fatty hepatosis. Definition, morphological characteristics, prognosis of toxic liver dystrophy.
110. Morphogenesis, forms, morphological characteristics of acute hepatitis. Morphological characteristics of chronic hepatitis, degree of activity and chronicity.
111. Morphological characteristics of the most important types of cirrhosis. Liver cancer, morphological characteristics.
112. Pathomorphology of gallstone disease. Pathomorphology of acute and chronic cholecystitis.

113. Morphological characteristics, complications of acute and chronic pancreatitis. Tumors of the pancreas, morphological characteristics.
114. Morphological characteristics, complications and causes of death in Itsenko-Cushing's disease.

115. Morphological characteristics, complications of acromegaly. Morphological characteristics of diabetes insipidus.
116. Morphological characteristics of diabetes. Complications of diabetes mellitus: morphological characteristics of diabetic macro- and microangiopathy.
117. Multinodular goiter. Morphological characteristics, complications, consequences. Graves' disease (diffuse toxic goiter, Based's disease): morphological features of the thyroid gland, visceral manifestations.
118. Hypothyroidism. Cretinism. Myxedema. Morphological characteristics. Definition, pathomorphology of Hashimoto's thyroiditis.
119. Primary chronic insufficiency of the cortical substance of the adrenal glands (Addison's disease): morphological manifestations. Waterhouse-Friederiksen syndrome: morphological manifestations.
120. Morphological manifestations of inflammatory diseases of the endometrium and myometrium. Morphological manifestations of precancerous processes and tumors of the endometrium and myometrium. Morphological characteristics, complications, consequences of inflammatory diseases of the mammary glands. Morphological characteristics of fibrocystic changes of mammary glands.

121. Morphological characteristics, complications, consequences of benign nodular hyperplasia of the prostate gland. Morphological characteristics of inflammatory diseases of the testicles.

122. Modern clinical and morphological classification of kidney diseases. Postinfectious glomerulonephritis: morphological characteristics, consequences. Rapidly progressive: morphological characteristics, consequences.
123. Chronic glomerulonephritis: morphological characteristics, consequences. Classification, morphological manifestations of idiopathic nephrotic syndrome. Morphological manifestations of membranous nephropathy.
124. Morphological characteristics, prognosis of necrotic nephrosis.
125. Morphological characteristics, prognosis of tubulointerstitial nephritis. Morphological characteristics, prognosis of acute and chronic pyelonephritis.
126. Morphogenesis and morphological characteristics of nephrolithiasis, consequences Chronic renal failure. Nephrosclerosis. Pathological anatomy.
127. Morphological changes of bones in hyperparathyroid dystrophy. Morphological characteristics, complications of Paget's disease.
128. Morphological characteristics, complications of fibrous dysplasia.
129. Morphological characteristics, complications of osteomyelitis.
130. Morphological characteristics, causes of death in Duchenne muscular dystrophy.

Morphological characteristics, causes of death in myotonia.

131. Classification, morphological diagnosis, complications and consequences of ectopic pregnancy.
132. Classification, morphological characteristics of ORN-gestoses.
133. Classification, morphological characteristics and prognosis of trophoblastic disease. 134. Morphological manifestations, impact on the fetus and the woman's body, consequences of infectious processes in the placenta. Morphological manifestations of blood circulation disorders in the placenta.
135. Morphological characteristics, prediction of delay in intrauterine development of the fetus.
136. Morphological characteristics of intrauterine infections of the fetus.
137. Birth injury: classification and morphology.
138. Morphological characteristics of hemolytic disease of infants. Morphological characteristics of hemorrhagic disease of infants.
139. Morphological characteristics, complications of pneumopathies.
140. Morphological characteristics, consequences of asphyxia.
141. Morphological characteristics, consequences of non-infectious fetopathy: diabetic and alcoholic fetopathy.
142. Classification and morphology of congenital malformations.
143. Morphological characteristics of impaired and insufficient nutrition.
144. Pathological anatomy, consequences, causes of death in the case of injuries related to the influence of physical factors of the external environment: industrial noise, electromagnetic waves of radio frequencies, ionizing radiation, electric current, temperature effects. Variants of local and general reactions in infections. Morphological characteristics, complications, consequences, causes of death in scarlet fever.
145. Morphological characteristics, complications, consequences, causes of death in bacterial dysentery.
146. Morphological characteristics, complications, consequences, causes of death in typhoid fever, salmonellosis.
147. Morphological characteristics, complications, consequences, causes of death in respiratory viral infections.
148. Morphological characteristics, complications, consequences, causes of death in typhoid fever.
149. Morphological characteristics, complications of prion lesions of the central nervous system.
150. Morphological characteristics, complications, causes of death in AIDS.
151. Morphological characteristics, complications, consequences, causes of death in childhood viral infections: measles, infectious mononucleosis, epidemic parotitis, poliomyelitis
152. Morphological characteristics, complications, consequences, causes of death in diphtheria. 153. Morphological characteristics, complications, consequences, causes of death in whooping cough.
154. Tissue reactions in tuberculosis. Pathological anatomy of primary tuberculosis complex. Morphology of progression of primary tuberculosis. Pathological anatomy of the chronic course of primary tuberculosis.
155. Morphological characteristics, complications, consequences, causes of death in hematogenous tuberculosis with predominant lung damage.
156. Morphological characteristics, complications, consequences, causes of death in secondary tuberculosis. Modern pathomorphosis of tuberculosis.
157. Clinical and anatomical forms of sepsis: septicemia, septicopyemia, septic (infectious) endocarditis.
158. Plague: clinical and morphological forms, complications, causes of death.
159. Tularemia: clinical and morphological forms, causes of death.
160. Anthrax: clinical and morphological forms, causes of death.

161. Cholera: clinical and morphological forms, complications, causes of death.
162. Pathomorphology of congenital syphilis. Pathomorphology of acquired syphilis.
163. Morphological characteristics, complications, consequences, causes of death in diseases caused by protozoa: malaria, balantidiasis, amebiasis.
164. Morphological characteristics, complications, consequences, causes of death in diseases caused by helminths: trichinellosis, echinococcosis, cysticercosis, opisthorchosis, schistosomiasis.

2 Practical tasks

3. Test tasks for self-control:

4. Individual tasks

5. List of recommended literature:

Main:

- Atlas of micropreparations in pathomorphology / I.I. Starchenko, B.M. Filenko, N.V. Royko, etc.; VDZU "UMSA". - Poltava, 2018. - 190 p
- The basics of pathology according to Robbins: in 2 volumes. Volume 1 / Vinay Kumar, Abul K. Abbas, John C. Astaire; translation of the 10th Eng. edition. Publisher: AllUkrainian specialized publishing house "Medytsyna". – X II. - 2019. - 420 p.
- Pathomorphology. General pathomorphology: a study guide / edited by Ya. Ya. Bodnara, V.D. Voloshina, A.M. Romanyuk, V.V. Gargin. - New Book, 2020. - 248 p.

Additional:

Pathomorphology: National handyman / V.D. Markovskyi, V.O. Tumanskyi, I.V. Sorokina [and others]; edited by V.D. Markovsky, V.O. Tumanskyi. - K.: VSV "Medicine", 2015. - P. 20-129.

Electronic information resources

- http://moz.gov.ua- Ministry of Health of Ukraine
- www.ama-assn.org– American Medical Association /American Medical Association
- www.who.int- World Health Organization
- www.dec.gov.ua/mtd/home/- State Expert Center of the Ministry of Health of Ukraine
- http://bma.org.uk– British Medical Association
- www.gmc-uk.org- General Medical Council (GMC)
- www.bundesaerztekammer.de– German Medical Association
- http://library.medicine.utah.edu/WebPath/webpath.html- Pathological laboratory http://www.webpathology.com/- Web Pathology