VOLGOGRAD STATE
MEDICAL UNIVERSITY
Department of Pathological Anatomy

Air-respiratory infections: SARS, influenza, parainfluenza, adenovirus infection, diphtheria, measles, scarlet fever, whooping cough, meningococcal infection.

Guidelines for students
III year of medical faculty
for practical exercises in pathological anatomy

Volgograd, 2020

1. Purpose: to study the epidemiology, causes, pathogenesis, characteristic signs and significance of childhood infections.

2. Requirements for the level of the student in the development of the discipline - pathological anatomy.

1. Give a definition of influenza, explain its etiology and pathogenesis; characterize local and general morphological changes in influenza; Assess the significance of complications and the cause of death in influenza.
2. Define diphtheria, explain its etiology and pathogenesis; characterize local and general morphological changes in diphtheria; evaluate the significance of complications and the cause of death in diphtheria.
3. Give a definition of scarlet fever, explain its etiology and pathogenesis; characterize the morphology of scarlet fever depending on the period and severity of its course; evaluate the significance of complications and the cause of death in scarlet fever.
4. Give a definition of meningococcal infection, explain its etiology and pathogenesis; characterize the morphology of various forms of meningococcal infection; evaluate the significance of complications and the cause of death in meningococcal infections.
5. Define measles, explain its etiology and pathogenesis; characterize local and general morphological changes in measles; evaluate the significance of the complications and causes of death in measles.

INFORMATION BLOCK

Infection is a biological phenomenon, the essence of which is the introduction into the human body and the multiplication of harmful prokaryotic and eukaryotic organisms in it, followed by the development of various forms of interaction - from asymptomatic carriage of invading agents to severe illness. In practical medicine, the term “infection” refers to an infectious disease, or an infectious process, that is, a complex of pathological changes and reactions to the introduction and reproduction of disease pathogens. Closely related to this is the concept of “infection” (infection) —the ingestion of the causative agent of an infectious disease into cells or tissues of the body. Infection can lead either to the occurrence of an infectious disease or to the carriage of pathogens in the human body (including endocytobiosis) without any signs of the disease. Despite the improvement of living conditions, mortality from infectious diseases remains quite high.

In order to attribute the disease to infections, it is necessary to observe the postulates of the outstanding German bacteriologist Robert Koch (R. Koch, 1843-1910):

• the causative agent of infection should be detected in all cases with this disease, but should not occur in other diseases or in healthy people;

• the pathogen can be isolated or isolated from the patient’s body in a pure culture;

• experimental introduction of a pure microbe culture into an organism that is sensitive to this pathogen causes this disease in an experimental animal.

When characterizing the pathogen, its virulence is taken into account - a measure of the pathogenic ™ pathogen ™ in relation to a given organism, which includes infectivity (ability to penetrate into the body), invasiveness (ability to spread in the organism) and toxicity.

Parasites are any organisms belonging either to prokaryotes or eukaryotes, i.e. viruses, microbes, protozoa, fungi, helminths, etc. that use another organism as a habitat and (or) a food source (in particular, the human body), causing harm in most cases. In such a broad interpretation, the concepts of "parasite" and "pathogen" are almost equal. Parasites (pathogens) are obligate (able to exist, or parasitize, only being in another organism) and optional, that is, capable of existing both inside the body (including inside its cells) and outside it. In practical medicine, the term "parasite" often refers only to eukaryotic organisms, considered separately from viruses and bacteria. They are involved in parasitology, which studies parasitic diseases caused by unicellular and multicellular eukaryotes. There are

endoparasites living inside the cavities, tissues and cells of the macroorganism, and ectoparasites living on the surface of the body.

Infection mechanisms

Respiratory tract. Daily, city dwellers inhale an average of about 10,000 micro-beans, including viruses, bacteria and fungi. Alveoli reach only particles of 5 microns or less. They are attacked and, as a rule, absorbed by alveolar macrophages or neutrophils, attracted by cytokines. This purification system is normally quite effective, but its role can be reduced by smoking, secretion of a viscous substrate with hereditary cystic fibrosis, aspiration of gastric contents and traumatic intubation. It also "weakens" with viral infections. Some viruses of the respiratory group (for example, influenza pathogens) possess hemagglutinins, which attach carbohydrates to the surface of the epithelium and thereby prevent mucociliary clearance, that is, cleansing with mucus and cilia. Bacteria are found (from the hemophilic, pertussis, and pertussis groups) that produce toxins that paralyze the activity of epithelial cilia. Tubercle bacillus settles in the alveoli due to its resistance to the killer action of inactive macrophages.

Viruses can spread from cell to cell by fusion of the latter or by axon transport (poliovirus). They are also able to penetrate the bloodstream and move with the help of either vagrant macrophages (HIV-1) or red blood cells (the causative agent of tick-borne Colorado fever).

Thus, in some cases, the most important signs of the infection process are formed and appear in areas remote from the gates of infection. So, measles and chickenpox viruses enter the body through the respiratory tract, but the first signs of the disease appear in the form of a skin rash.

Flu. Influenza (from French grippe - catch) is an acute highly contagious epidemic disease that usually occurs in the cold season and is caused by an RNA virus tropic to the epithelium of the respiratory tract (pneumotropic virus) belonging to the Orthomyxoviridae family. Another name for the disease is influenza (from Italian. Influenza - influence).

Etiology. There are 3 serological types of influenza virus: A (represents the greatest epidemic danger), B (causes local outbreaks and epidemics), C (usually leads to sporadic cases).

Pathogenesis. The virus causes 3 stages of the disease. Stage 1 - the introduction and primary reproduction of the virus, occurring with the help of its RNA polymerase. The duration of this stage, corresponding to the incubation period of the disease, ranges from several hours to 2-4 days. Stage 2 - viremia, accompanied by prodromal phenomena. Stage 3 - secondary reproduction of the virus in tropic cells, leading to generalization of the infection and the height of the disease, manifested by acute subfibril or fibril fever, headache, catarrhal rhinitis, cough and (less commonly) conjunctivitis, joint and muscle pain. The whole variety of developing changes in the body is due to the following properties (action) of the virus: Cytopathic (cytolytic) action, leading to degenerative lesions of the epithelial cells of the respiratory tract with subsequent necrosis, desquamation, which is often accompanied by a violation of the drainage function of the respiratory epithelium; Immunosuppressive effect with the development of transient immunodeficiency, manifested in a significant reduction in the patient's phagocytic activity of neutrophils, macrophages, suppression of chemotaxis, the appearance of circulating toxic immune complexes; A vasopathic (vasoparalytic) effect that causes hyperemia, stasis, plasma impregnation, edema, plasmorrhagia and hemorrhage; The neuropathic effect due to the effect, first of all, on the neurovegetative, neuroendocrine and neurohumoral centers of the medulla oblongata and hypothalamus, where a high concentration of toxins is created due to large vascularization.

Pathological anatomy. The morphological picture is due to a combination of local and general changes. The first changes affect the respiratory tract and are associated primarily with the cytopathic and vasopathic effects of the virus. In the cytoplasm of affected epithelial cells, the presence of small round rounded basophilic (clusters of viruses) and fuchsinophilic (organelle destruction under the influence of the virus) bodies is noted. The most sensitive morphological methods for detecting the virus are immunohistochemical methods, in particular immunofluorescence. General changes are caused by viremia and intoxication, leading to dystrophic, discirculatory disorders and inflammation in the inter-

nal organs, skin, serous membranes. Depending on the severity of existing local and general changes, mild, moderate and severe clinical and morphological forms of the disease are distinguished.

In mild influenza, which is the most common variant of the disease, acute catarrhal (serous, mucous, desquamative) rhinolaryngitis develops, less commonly rhinolaryngotracheobronchitis. The mucous membranes of the nose, larynx, trachea, bronchi (occasionally and sinuses) are swollen, covered with abundant exudate, full-blooded, sometimes with puncture hemorrhages. Microscopic examination noted hydropic degeneration, necrosis and desquamation of affected epithelial cells, slight lymphocytic infiltration, increased secretory activity of goblet cells and glands. An immunohistochemical (immunofluorescence) study, which determines the influenza virus and its serological type, shows cytoplasmic inclusions of the pathogen in smears of the mucous membrane of the upper respiratory tract. The disease after 5-6 days ends in complete recovery. However, in some cases, the process can progress and give a number of complications. That is why in the treatment of patients with even mild influenza, bed rest and detoxification therapy are so important.

Moderate influenza is characterized by the addition to the above changes of a more significant lesion of the mucous membrane of the trachea, bronchi and alveoli, where serous hemorrhagic or fibrinous hemorrhagic inflammation develops with lympho-macrophage infiltration, extensive areas of necrosis and desquamation of the epithelium. The latter circumstance, along with a decrease in the production of surfactant by pneumocytes due to the cytopathic effect of the virus and the formation of thick mucous plugs, leads to obstruction of the bronchi with the formation of atelectasis characteristic of the disease. At the same time, the lung is enlarged in size, with dense, sinking in the water areas, bluish-red or gray-red. In addition, in the lungs, interstitial and focal (acinous, lobular, confluent) serous or serous-hemorrhagic pneumonia, acute perifocal emphysema develops. The interalveolar septa are significantly thickened due to interstitial inflammation. Recovery in most cases occurs after 3-4 weeks, however, bronchopulmonary complications are possible. In influenza caused by A2 virus (Hong Kong, etc.) and proceeding compared to other types of pathogen, it is usually more severe, more pronounced hypertrophy of alveolocytes is observed with the formation of large mononuclear cells that are larger than alveolar macrophages and containing basophilic inclusions of viruses and fuchsinophilic (oxyphilic) bodies.

Severe influenza occurs in two ways: toxic and with pulmonary complications.

Toxic flu is manifested not only by serous-hemorrhagic inflammation of the upper respiratory tract and lungs with an increase in hemorrhagic and necrotic components, but also by severe general changes. Perhaps the development of hemorrhagic pulmonary edema, hemorrhagic syndrome (multiple hemorrhages in the brain, mucous membranes and serous membranes, skin, internal organs), serous hemorrhagic meningitis, cerebral edema, and acute lymphoid organ hyperplasia. Parenchymal organs and nervous tissue are full-blooded, with the phenomena of fatty and protein dystrophy. In sick children, individual cases of adrenal hemorrhage (Waterhouse-Fridericksen syndrome), development of false croup (edema of the laryngeal mucosa with spasm of its lumen and asphyxia) have been described.

Influenza with pulmonary complications occurs when a secondary bacterial infection (strepto-, staphylococcus, pneumococcal, pseudomonas, etc.) is associated with the development of severe bronchopneumonia, usually observed a week after the onset of the disease. It should be noted that the presence of bacteria is evidenced by the development of purulent inflammation in the affected areas, which is not characteristic of “pure” viral infections. The patient has fibrinous hemorrhagic (rarely necrotic) laryngitis and tracheitis, while in the bronchi there is serous-purulent, fibrinous-purulent, purulent hemorrhagic bronchitis, as a rule, capturing the entire thickness of the bronchus molten in separate sections of the bronchial wall (segmental destructive panbronitis). This explains the frequent development of subsequent bronchiectasis, atelectasis and obstructive emphysema. In the lungs, severe focal-confluent serous-hemorrhagic pneumonia forms, rapidly giving way to purulent-hemorrhagic pneumonia, sometimes capturing entire segments with purulent fusion of the pulmonary parenchyma. The affected lung is enlarged in size, uneven color, airiness and density due to the alternation of reddish-gray or reddish-green dense, bulging foci of pneumonia, sunken bluish or gray-red airless atelectasis, swollen light ash areas of acute emphysema, dirty and gray abscesses red hemorrhage. Such a lung was called the "large motley lung." Spleen with influenza is enlarged, as a rule, slightly, gives a small scrap-

ing of hyperplastic pulp. Minimal regional lymphadenitis is noted. In early childhood, the flu is usually more severe than in adults, with more pronounced intoxication, hemorrhagic syndrome, damage to the nervous system and frequent complications. In addition, it is noteworthy that the increase in the incidence among children is somewhat behind the peak of the epidemic in adults.

Complications and causes of death. All flu complications can be pulmonary and extrapulmonary. Influenza pneumonia often leads to the development of bronchiectasis (bronchiectasis), pneumosclerosis, areas of carnification, the formation of chronic obstructive emphysema. In severe cases, fibrinous, less often hemorrhagic or purulent pleurisy, up to pleural empyema, sometimes purulent mediastinitis is noted. Often, pericarditis develops, which contributes to diligence of the lung and pleura to the heart shirt. In some cases, serous meningitis, arachnoiditis, encephalomyelitis, purulent encephalitis, neuritis, glomerulonephritis are possible. Sometimes toxic myocarditis, acute warty or ulcerative endocarditis develops, catarrhal or catarrhal-purulent otitis media, sinusitis, frontal sinusitis, ethmoiditis occur. In elderly patients, activation of chronic diseases may be observed. Relatively rarely, patients die from intoxication, hemorrhagic pulmonary edema, hemorrhages in the vital centers of the brain, which is possible with severe toxic flu already on the 4th – 5th day of the disease (fulminant form of the flu, "acute influenza toxicosis"). Most often, death occurs at a later date from cardiopulmonary or pulmonary heart disease due to pneumonia and its complications. Asphyxia is possible due to true (due to croupous inflammation) or false croup, which is noted, as a rule, in children.

Diphtheria

Diphtheria (from the Greek. Diphthera - skin, film) is an acute infectious disease characterized mainly by fibrinous inflammation in the focus of primary fixation of the pathogen and general intoxication associated with absorption of exotoxin. Most often, children from 4 to 6 years old get sick. Currently, adults and children over 7 years old are ill.

The causative agent of diphtheria is Corynebacterium diphtheriae. Infection occurs mainly by airborne droplets. Diphtheria bacillus is well preserved in the environment. It survives 17 days in water and milk, on dishes, books, toys, linen can be stored for several weeks, dies in a few hours under the influence of sunlight; all disinfectants (lysol, phenol, mercuric chloride, chloramine, hydrogen peroxide, formalin) in normal concentrations kill it. The main source of infection is sick with diphtheria, which is dangerous for those around the entire period of the disease and even some time after recovery. When coughing, sneezing, talking with droplets of saliva, sputum, mucus, the patient releases pathogens into the environment. A healthy person becomes infected by inhaling contaminated air. The source of infection can be a bacteriocarrier - a healthy child or an adult without visible signs of the disease, but secreting diphtheria bacilli. Their carriers are often children. Diphtheria bacillus affects the mucous membranes of the nasopharynx, pharynx, upper respiratory tract (larynx, trachea). More rarely, bacteria enter the mucous membrane of the external genitalia in girls, the umbilical wound in newborns, and damaged skin. Diphtheria bacillus survives on the mucous membrane, but the toxin secreted by it spreads blood and lymph throughout the body. The toxin at the site of introduction and reproduction of the bacillus causes inflammation of the mucous membrane with the formation on it of a dense, filmy coating of gray-white color, tightly soldered to tissues. Depending on the place of penetration and propagation of diphtheria bacilli, various forms of the disease are observed. The incubation period of the disease lasts from 2 to 10 days.

Diphtheria pharynx begins with malaise and fever up to 38 ° -39 ° C. There is a sore throat, swelling of the submandibular lymph nodes. Redness of the mucous membrane is found in the pharynx, on the tonsils and less often on the soft palate - white or grayish-white membranous deposits. The more extensive the raids, the stronger the intoxication of the body and the more severe the course of the disease. The so-called toxic form of diphtheria may develop; it begins acutely, the temperature rises to 39 ° -40 ° C, there may be severe pain when swallowing, repeated vomiting. General weakness and lethargy appears, the pulse is frequent, the face is pale. There is edema of the subcutaneous tissue in the submandibular lymph nodes, which extends to almost the entire neck, sometimes to the chest. One of the earliest signs of toxic diphtheria is swelling of the throat, when the tissues of the tonsils and soft

palate are closed, almost leaving no clearance. A filmy coating covers the palate, nasopharynx, breathing becomes wheezing, the mouth is half-open, later abundant discharge from the nose appears.

Diphtheria of the nose is characterized by a persistent runny nose. The general condition of the child may not be disturbed, the temperature is normal, in connection with which parents in most cases late seek medical help.

Morphology and morphogenesis. After infection of a person, bacteria most often settle on the mucous membrane of the upper respiratory tract and digestive tract, primarily on the tonsils. Diphtheria bacillus multiplies in the area of the entrance gate on the mucous membranes and releases exotoxin, the absorption of which is entirely dependent on the structural features of the mucous membrane and the depth of local changes. With a mild course of the disease, catarrhal inflammation is observed. More often, exotoxin causes epithelial necrosis, paretic expansion of blood vessels with a violation of their permeability, tissue edema and the release of fibrinogen from the vascular bed. Fibrinogen coagulates under the influence of tissue thromboplasty. A fibrinous film forms on the surface of the damaged mucous membrane. At the same time, local paralytic expansion of blood vessels occurs, accompanied by a slowdown in blood flow and a sharp increase in vascular porosity. A large number of coarse proteins are mixed with the exudate and the inflammation becomes fibrinous. In addition to fibrin, exudate contains few leukocytes, macrophages, and red blood cells. With the hypertoxic form of diphtheria, the process sometimes takes on a hemorrhagic character. The development of severe toxic and hyper-toxic forms of diphtheria is due to increased sensitivity due to sensitization to diphtheria toxin.

Macroscopically, with diphtheria of the pharynx and tonsils against a background of moderate hyperemia, a whitish or yellowish film about 1 mm thick is determined, mostly tightly connected to the underlying tissues - multilayer flat epithelium and underlying fibrous connective tissue (diphtheria inflammation). With this form of diphtheria, toxic changes are most pronounced. During recovery, either the film melts under the action of proteolytic enzymes, or its rejection as a result of demarcation inflammation with the formation of ulceration. In addition to diphtheria of the pharynx, the most frequent localization of the local inflammatory process, it may develop in other parts of the body.

Distinguish diphtheria: nose; larynx; eye; ears genital mucous membranes; skin (in the area of wounds).

In some cases, several organs can be affected simultaneously - combined diphtheria. Lymphatic and hematogenous spread of the pathogen with diphtheria is relatively rare. A somewhat larger role may be played by intracanalicular dissemination. In this way, corynebacteria can spread from the throat to the nasopharynx, through the respiratory tract to the lungs. In all areas, the occurrence of fibrinous inflammation is possible. Significantly more severe and common changes occur due to exposure to toxins when they are absorbed from the site of infection. Among them, it should be noted first of all changes in regional lymph nodes. They significantly increase in size due to sharp plethora and hemorrhage. Plots of necrosis usually occur in the centers of the follicles. Often, edema of the mucous and submucous membranes of the pharynx and pharynx, as well as fiber of the muscles of the neck, develops. Along with this, cellular infiltrates and focal necrosis of the neck muscles are detected. In cases with the highest toxicosis, numerous hemorrhages occur. In addition to such changes, with diphtheria, vascular thrombosis is often noted. Exotoxin Corynebacterium diphtheriae has the ability to suppress the biosynthesis of respiratory cycle enzymes, therefore it paralyzes tissue respiration, changes cholinergic processes, disrupts the synthesis of catecholamines and leads to their accumulation in tissues. Exotoxin acts primarily: on the heart; peripheral nervous system; adrenal glands. The release of exotoxin from the body is accompanied by damage to the predominantly tubular epithelium of the kidneys. Necrotic nephrosis is detected in the kidneys, and in severe cases of toxic diphtheria, massive necrosis of the cortical layer. Of great importance is the defeat of the nervous system, especially peripheral. The most characteristic is the selective damage to nerve fibers with periaxonal decay of myelin. The reaction from the cells of the membranes and changes in the axial cylinders are insignificant. First of all, peripheral nerves are located that are closer to the pharynx: glossopharyngeal, vagus, sympathetic and phrenic nerves, III cervical sympathetic ganglion and nodose ganglion of the vagus nerve. Alterative neuritis develops with the breakdown of myelin, axial cylinders suffer less. Circulatory disorders, dystrophic changes in nerve cells, up to cytolysis, are observed in the nerve ganglia. Changes, gradually

increasing, appear after 1.5-2 months in the form of so-called late paralysis of the soft palate, diaphragm, heart with lesions of the glossopharyngeal, diaphragmatic and vagus nerves, respectively. Possible regeneration of elements of the peripheral nervous system. A significant role in diphtheria is played by damage to the cardiovascular system. In the early stages of the disease, paresis of blood vessels is noted. In arterioles, as well as in the walls of arteries, fibrinoid necrosis can be detected. In the myocardium, at first, alterative changes are determined (lysis, vacuolization or block decomposition of muscle fibers), sometimes combined with lipidosis - alternative toxic myocarditis. Cavities of the heart are widened across, the muscle is dull, flabby, mottled on the cut, there may be parietal thrombi. With a combination of alterative changes with serous edema and moderate lymphocytic infiltration by the end of the 1st week, we can talk about serous interstitial myocarditis. Later, (starting from the 2nd week) after the appearance of histiocytic infiltration, the term interstitial productive myocarditis is used. Dystrophic changes and hemorrhages in the structures of the conducting system of this organ are essential for the development of heart lesions. Macroscopically, the muscle of the heart is flabby, grayish or yellowish in color. In its expanded cavities, as well as in large vessels, especially in the veins, blood clots often form. Significant changes occur in the adrenal glands and paraganglia. There are sharp circulatory disorders, often with hemorrhages, necrosis of part of the cortical cells. Hemorrhages, dystrophy and necrosis of cells are noted in the adrenal medulla, in the cortical layer - the disappearance of lipids, small foci of necrosis. Perhaps the development of toxic changes in other organs. In the spleen, hyperplasia of the B-zone with pronounced karyorexis in the centers of reproduction of the follicles, plethora of the pulp are noted. Death with pharyngeal diphtheria (untimely administration of antitoxic serum) or toxic forms occurs from early heart failure with myocarditis (at the beginning of the 2nd week of illness) or late heart failure or diaphragm associated with alterative neuritis. Diphtheria of the respiratory tract is characterized by lobar inflammation of the larynx, trachea, bronchi with easily separated fibrinous films. The films easily depart, since the mucous membrane of the upper respiratory tract and bronchi is lined with prismatic and cylindrical epithelium, loosely connected to the underlying connective tissue. Abundant mucus also contributes to the separation of the film. Therefore, the toxin is not absorbed and there are no general toxic effects with this form of diphtheria. Croupous inflammation of the larynx with diphtheria was called true croup, the spread of the process to small branches of the bronchial tree - descending croup, which may be accompanied by the development of focal pneumonia. Complications of diphtheria of the respiratory tract are associated with the use of intubation or tracheotomy, in which the formation of pressure sores is possible. Pressure ulcers with secondary infection lead to purulent perichondritis of the cartilage of the larynx, phlegmon, purulent mediastinitis. The timely use of antibiotics prevents these complications. The death of patients with laryngeal diphtheria can be caused by asphyxia (spasm of the larynx with true croup or airway obstruction with fibrinous films) or associated pneumonia and purulent complications.

Scarlet fever

Scarlet fever (from Italian. Scarlatum - crimson, purple) is one of the forms of streptococcal infection in the form of an acute infectious disease with local inflammatory changes, mainly in the throat, accompanied by a typical common rash. More often children from 2 to 7 years old, sometimes adults. The causative agent is Streptococcus pyogenes (b-hemolytic group A streptococcus of various serological variants). The highest incidence of scarlet fever occurs in the autumn-winter period. Infection occurs from a sick child who is dangerous to others throughout the illness and even some time after recovery. The source of infection can also be patients in whom scarlet fever occurs in a very mild, worn-out form, sometimes (for example, in adults) in the form of tonsillitis (tonsillitis). The causative agent of scarlet fever, located in droplets of sputum, saliva, mucus of the patient, coughs, sneezes, talks into the air and then penetrates through the respiratory tract into the body of a healthy child (airborne transmission of infection). The causative agent of scarlet fever can remain for some time on objects used by the patient, and they can also be a source of infection. Most often, streptococci enter the body through the pharynx, less often through damaged skin. The incubation period is from 2 to 7 days. Clinic. The disease begins suddenly: the temperature rises quickly, general malaise appears, sore throat

when swallowing, there may be nausea, as well as vomiting, sometimes multiple. In the first 10-12 hours of illness, the skin is clean, dry and hot. In the throat, bright redness, tonsils enlarged. The rash appears at the end of the first or beginning of the second day of the disease, first on the neck, upper back and chest, then quickly spreads throughout the body. It is especially abundant on the flexion surfaces of the arms and lower abdomen. The rash is red or bright pink in the form of small, about the size of a poppy seed, densely spaced spots. Itchy skin is often noted. The chin and skin above the upper lip and nose remain pale on the face, forming the so-called white scarlet fever. The tongue is dry and covered with a whitish coating; on the 3rd day it is cleaned and becomes raspberry red (raspberry tongue). These manifestations of the disease persist for several days, and then gradually disappear. By the end of the first or at the beginning of the second week, lamellar peeling appears on the site of the rash, first on the neck, earlobes, and then on the tips of the fingers and toes, on the palms and feet. On the body, peeling is pityriate. Peeling ends in 2-3 weeks.

Pathogenesis. For the occurrence of streptococcal infection, it is very important to pre-damage the epithelial cover (mucous membranes or skin) most often with viruses. In the development of scarlet fever, two periods are distinguished. The first period is due to direct toxic or septic effects on body tissues. The second period is manifested by allergic reactions from the skin, joints, kidneys, blood vessels, heart. The primary focus in scarlet fever is usually localized in the pharynx (pharyngeal form of scarlet fever) with a maximum lesion of the tonsils and much less often in other organs and tissues, especially in the skin (extrafaringeal form of scarlet fever). The former name is buccal and extra buccal scarlet fever. Streptococci after infection of a person most often settle on the mucous membrane of the nasopharynx, mainly on the tonsils, where they begin to multiply in the depths of one or more crypts.

Macroscopically tonsils enlarged, swollen, bright red (catarrhal sore throat). Microscopic examination in the mucous membrane and tissue of the tonsils shows sharp plethora, foci of necrosis, along the periphery of which streptococcus chains are found in the area of edema and fibrinous effusion, and a slight leukocyte infiltration is found at the border with healthy tissue. Under the influence of their toxins, necrosis of the crypt epithelium occurs, and then the lymphatic tissue of the organ. Around the focus of necrosis, plethora, edema, and then a leukocyte reaction with the formation of a zone of demarcation inflammation are noted. Fibrin often falls on the surface of the tonsil. Soon on the surface and in the depths of the tonsil tissue, grayish, dull foci of necrosis appear - a necrotic tonsillitis typical of scarlet fever. Depending on the severity of the course, necrosis can spread to the soft palate, pharynx, auditory (Eustachian) tube, middle ear, from the lymph nodes to the fiber of the neck. With the rejection of necrotic masses, ulcers form. In the case of the spread of the infectious process to surrounding tissues, a pharyngeal abscess occurs. Due to the paralytic state of small blood vessels, the soft palate and nasopharynx are sharply full-blooded (“flaming pharynx”). Streptococci and their toxins naturally spread throughout the patient's body. Especially often the lymphogenous spread of bacteria occurs, especially in the regional lymph nodes. In the future, an inflammatory process develops with a predominance of the alternative component. The inflammatory process can spread beyond the nodes to the fatty tissue and muscles of the neck (hard phlegmon). Later, hematogenous dissemination also occurs. Often there is an intracanalicular distribution of streptococci. When they enter the nasopharynx and nose, damage occurs not only to the mucous membrane, but also to the underlying tissues, including the ethmoid bone. Occasionally, infection spreads through the auditory tube into the middle ear. Less commonly, dissemination of streptococci through the digestive tract. Along with this, streptococcal toxins are distributed throughout the patient’s body, which is especially pronounced in the first 3 days. The most important manifestation of toxemia for diagnosis is a rash (the disease is only called scarlet fever in this case). Histological examination of the skin reveals focal congestion, edema, as well as hemorrhages, later small perivascular, mainly lymphohistiocytic, infiltrates are formed. Macroscopically, a rash of bright red color, small-pointed, appears first on the skin of the neck, then spreads to the chest, back, and finally captures, in typical cases, the entire body except the nasolabial triangle. The cervical lymph nodes are enlarged, juicy, full-blooded, there may be foci of necrosis and the phenomena of severe myeloid infiltration (lymphadenitis). Dystrophic changes and interstitial lymphohistiocytic infiltrates are noted in the liver, myocardium and kidneys. In the spleen, lymphoid tissue of the intestine, B-zone hyperplasia with plasmaization and myeloid metaplasia are observed. These changes vary

depending on the severity and form of scarlet fever. In the brain and autonomic ganglia, there are dystrophic changes in neurons and circulatory disorders.

The rash usually appears on the 1-2 day of the disease in the upper half of the body and then spreads to the limbs. Soles and palms remain free of rashes. The rash is small-dot, consists of tiny papules, so the skin feels like sandpaper to the touch. In addition, there is a pallor of the nasolabial triangle, a strawberry tongue (a lined tongue with protruding red papillae, later the plaque disappears), a bright rash in the skin folds in the form of lines (Pastia symptom). After 6-9 days, the rash is resolved, and a little later there is peeling of the hands and feet.

There are **two forms of scarlet fever**:

-toxic;

- septic.

In severe toxic form, death occurs in the first 2-3 days from the onset of the disease, in the throat there is a particularly sharp hyperemia, which extends even to the esophagus. Hyperplasia in lymphoid tissue is less pronounced, dystrophic changes and severe circulatory disorders predominate in organs. With a severe septic form in the affect area, the process assumes a widespread purulent-necrotic character with the formation of a pharyngeal abscess, otitis anthritis and purulent temporal bone osteomyelitis, purulent-necrotic lymphadenitis, neck phlegmon, soft - with purulent fusion of tissues, hard - with a predominance of necrosis. Phlegmon can lead to arrosion of large vessels of the neck and fatal bleeding. Purulent inflammation from the temporal bone can pass to the venous sinuses of the dura mater with the formation of brain abscess and purulent meningitis. In lymphoid organs, myeloid metaplasia predominates with the displacement of lymphoid tissue. With reduced body resistance, streptococci sometimes penetrate the bloodstream, which leads to sepsis. Such forms of the disease are more common in young children (1-3 years). At 3-4 weeks, sometimes later, from the onset of the disease in some patients, a second period of scarlet fever occurs. The second period of the disease can never be foreseen, since it does not necessarily occur, regardless of the severity of the first. They are characterized by the same changes as at the beginning of the disease, but they are less pronounced and are not accompanied by a toxic symptom complex. This repeated inflammatory process causes peculiar severe allergic lesions in a person sensitized to streptococci, the most characteristic of which is acute ("post-streptococcal") or chronic glomerulonephritis. There are no streptococci in the kidneys at this stage of the process, however, immune complexes containing streptococcal antigen are detected here. Vasculitis, serous arthritis, recurrent-warty endocarditis, less often fibrinoid changes in the walls of large vessels with an outcome in sclerosis, can be observed. Due to the use of antibiotics, as well as changes in the properties of the pathogen itself, at present, allergic and purulent-necrotic processes in scarlet fever almost do not develop. Death can occur from toxemia or septic complications.

Measles

Measles is an acute viral disease characterized by fever, general intoxication, enanthema, maculopapular rash, damage to the conjunctiva and upper respiratory tract.

Etiology. The causative agent of measles (Polinosa morbillarum) belongs to paramyxoviruses (family Paramyxoviridae, genus Morbillivirus). The genus of measles viruses also includes the subacute sclerosing panencephalitis virus, the dog plague virus and the cattle plague virus. Morphologically, the measles virus is similar to other paramyxoviruses, the diameter of its virion is 120-250 nm. The shell contains 3 layers - a protein membrane, a lipid layer and external glycoprotein protrusions. Contains RNA, has hemagglutinating and hemolytic activity. Hemolyses and agglutinates red blood cells of monkeys, but unlike other paramyxoviruses it does not agglutinate red blood cells of chickens, guinea pigs, and mice. Pathogenic for monkeys. It is cultivated on the cells of human kidneys and monkeys. Attenuated measles virus strains are obtained that are used as a live measles vaccine. Measles virus is rapidly inactivated by heating, ultraviolet radiation, under the influence of disinfectants.

Epidemiology. The source of infection is only a sick person who releases measles virus into the environment from the last 2 days of the incubation period to the 4th day after the rash. Transmission occurs by airborne droplets. Persons who do not have measles and are not vaccinated against it remain

highly susceptible to measles throughout their lives and can become ill at any age. Prior to the introduction of measles vaccination, 95% of children had measles before the age of 16 years. After the widespread use of measles vaccines, the incidence of measles has significantly decreased, but the incidence of measles has persisted and in recent years there has been an upward trend. For complete measles protection, immunization of 94-97% of children under 15 months of age is necessary. This is difficult to achieve even in developed countries. Outbreaks of measles are also observed among those vaccinated (67-70% of all outbreaks). A large number of cases are noted among older age groups (school children, adolescents, military personnel, students, etc.). This is due to a significant decrease in immunity 10-15 years after immunization. The incidence is high in African countries; measles is especially severe here.

Pathogenesis. The gates of infection are the mucous membrane of the upper respiratory tract. The virus multiplies in the epithelium of the respiratory tract, as well as in other epithelial cells. Electron microscopy of material taken from Filatov-Koplik spots and skin rashes, accumulations of measles virus are detected. From the last days of incubation, within 1-2 days after the rash appears, the virus can be isolated from the blood. The causative agent is hematogenously distributed throughout the body, fixed in the organs of the reticuloendothelial system, where it multiplies and accumulates. At the end of the incubation period, a second, more intense wave of viremia is observed. The causative agent has pronounced epithelial activity and affects the skin, conjunctiva, mucous membranes of the respiratory tract and oral cavity (Belsky-Filatov-Koplik spots). The virus can also be found in the mucous membrane of the trachea, bronchi, sometimes in the urine. In some cases, the virus can be introduced into the brain, causing the development of specific measles encephalitis. In hyperplastic lymphoid tissues, in particular in the lymph nodes, tonsils, spleen, thymus, giant reticuloendotheliocytes (Uortin-Finkeldean cells) can be found. In many leukocytes, destroyed chromosomes are detected. The airway epithelium can necrotic, which contributes to the stratification of a secondary bacterial infection. From the 3rd day of the rash, viremia is sharply reduced, and from the 4th day the virus is usually not detected. From this time, neutralizing antibodies begin to be detected in the blood.

With measles, a specific allergic rearrangement of the body develops, which persists for a long time. In vaccinated, the titers of antibodies to measles virus sharply decrease over time, while allergization persists for a long time. This causes an atypical course of measles in vaccinated patients who become ill 5-7 years after vaccination. There is evidence of a relationship between measles virus and the so-called slow infections that occur with degenerative processes in the central nervous system (chronic encephalitis). In particular, patients with subacute sclerosing panencephalitis found high titers of measles antibodies. However, at present, several strains of the virus have been isolated from the brains of those who died from subacute sclerosing panencephalitis, which differed in properties slightly from measles virus and were closer to the dog plague virus in antigenic structure. Measles leads to a state of anergy, which is manifested in the disappearance of allergic reactions (to tuberculin, toxoplasmin, etc.) in infected individuals, as well as in exacerbation of chronic diseases (dysentery, tuberculosis, etc.). Immunosuppression persists for several months. As established in African countries, within a few months after an outbreak of measles, the incidence and mortality rate among children who have had measles is 10 times higher compared to children who have not had measles. On the other hand, the premorbid state of the immune system affects the clinical symptoms and course of measles. The measles problem in HIV-infected people is becoming increasingly relevant.

Immunity after suffering a natural measles infection is persistent. Recurrent measles diseases are rare. Immunity after vaccination is shorter (10 years after vaccination, only 36% of vaccinees retain protective titers of antibodies).

Symptoms and course. The incubation period lasts 9-11 days. With the prophylactic administration of immunoglobulin, it can be extended up to 15-21 days, less often - longer. Separate manifestations of the disease are noted from the second half of the incubation period (weight loss of the child, swelling of the lower eyelid and conjunctival hyperemia, low-grade fever in the evenings, cough, slight runny nose). The initial or prodromal period is characterized by an increase in body temperature to 38-39 ° C, weakness, general malaise, and a decrease in appetite. A runny nose intensifies, a rough "barking" cough appears, conjunctival hyperemia is sharply expressed. There is a measles enanthema in the form of small red spots located on the mucous membrane of the soft and hard palate, pathogenomic for mea-

sles spots Belsky-Filatov-Koplik. These spots are often localized on the mucous membrane of the cheeks. They are small whitish, slightly rising above the level of the mucous membrane spots, surrounded by a narrow reddish border, and firmly sit on the mucous membrane. In appearance they resemble semolina or bran. With the advent of exanthema, they disappear. At the end of the initial period (3-4th day), body temperature decreases, then with the appearance of measles rash again rises to higher numbers. General intoxication and damage to the respiratory tract are amplified.

Measles exanthema is characterized by the stages of rash: on the 1st day, elements of the rash appear on the face, neck; on the 2nd day - on the body, arms and hips; on the 3rd day, the rash seizes the legs and feet, and on the face begins to turn pale. The most densely elements of the rash are located on the face, neck and upper body. The rash consists of small papules (about 2 mm), is surrounded by an irregularly shaped spot, the diameter of the spot is usually more than 10 mm. Elements of the rash tend to merge, forming complex figures with scalloped edges. However, even with the thickest rash, patches of perfectly normal skin can be detected. In some cases, against the background of measles exanthema, hemorrhages (petechiae) can be noticed. After 3-4 days, the elements of the rash turn pale, brownish spots remain in their place - pigmentation, especially pronounced and prolonged in the presence of hemorrhagic transformations of the rash. At the site of the rash, pityriasis peeling is subsequently observed (on the face and trunk).

Pronounced conjunctivitis is characteristic, sometimes with purulent discharge, gluing eyelashes in the morning. Peripheral lymph nodes (posterior cervical, occipital, axillary) are enlarged, sometimes sensitive to palpation. Scattered dry rales, sometimes medium-bubbly wet rales, are heard over the lungs. In the case of pneumonia, shortness of breath appears, with percussion, certain areas of shortening of percussion sound are noted, sonorous, fine-bubbly wet rales are heard. Some patients have abdominal pain, loose stools. The appearance of diarrhea may be due to other pathogenic agents (campylobacter, giardia, rotaviruses, etc.), superimposed on measles infection.

Koplik spots - small whitish papules with a red rim. They can be confused with heterotopically located sebaceous glands - Fordyce's disease. However, the latter do not have a hyperemic rim and are not accompanied by disturbances in the general condition.

Koplik spots are bluish-white with a diameter of 1-2 mm, with a bright red border. When examining a patient in low light, they are easy to miss. Koplik spots, sometimes numerous, are usually located on the mucous membrane of the cheeks opposite the second molars. They are found only with measles. With the appearance of the rash, Koplik's spots fade and soon disappear.

Even in the absence of symptoms of central nervous system damage in measles, changes on the EEG are very often detected. Clinically pronounced encephalitis with fever, headache, confusion, coma, convulsions develops in 1 out of 1,000 cases. Usually, symptoms appear in the first days after the rash, in rare cases, a few weeks later. Mortality in acute measles encephalitis is about 10%. After it, neurological defects often remain, in particular mental retardation, epilepsy. Less commonly, measles is complicated by transverse myelitis. In most cases, damage to the central nervous system is caused by an immune response to myelin proteins (post-infection encephalomyelitis), and not by direct exposure to the virus. In patients with weakened immune systems, encephalitis can progress and lead to death after 1-6 months. Sometimes measles encephalitis develops in the absence of a clinically pronounced measles history (the virus is isolated by autopsy).

Complications. A measles virus infection of the mucous membrane of the respiratory tract can lead to the development of bronchitis, false croup, bronchiolitis, and also cause the most common measles complication - pneumonia. By genesis, it is viral-bacterial. A major role is played by the layered secondary bacterial microflora. But with some forms of pneumonia, the virus plays a major role. Such complications include interstitial giant cell pneumonia, which most often develops in people with immunodeficiencies (in cancer patients it is detected in 50-60%, in HIV-infected patients in 60-82%), it is severe, accompanied by shortness of breath, in the lungs infiltrative changes, multinucleated giant cells can be found in sputum.

Conjunctivitis is a mandatory manifestation of measles, but in some patients, in addition to conjunctiva, the cornea can also be affected. Keratoconjunctivitis is a complication that can sometimes lead to blindness. Rare complications include myocarditis, hepatitis, glomerulonephritis. With second-

ary bacterial pneumonia, a lung abscess may develop. A serious complication is damage to the central nervous system (encephalitis, meningoencephalitis), which is observed in 1 in 1000 patients with measles (in people with a weakened immune system, encephalitis was observed in 20% of cases). Signs of encephalitis often appear a week after the appearance of exanthema, although they may develop later (after 2-3 weeks). The body temperature rises again, signs of general intoxication, drowsiness, lethargy, sometimes loss of consciousness, amimia, lack of abdominal reflexes, nystagmus, damage to the facial nerve, paralysis of the extremities appear. Serious consequences can result in measles lesion of the optic and auditory nerve. When involved in the process of the spinal cord, there may be pelvic disorders.

The most difficult to diagnose atypical measles in vaccinated. It is necessary to differentiate from rubella, enteroviral exanthema, Rosenberg infectious erythema, allergic (drug, serum) rash, from infectious mononucleosis. In these cases, it is advisable to use laboratory methods. In the initial period and in the first two days after the rash appears in stained smears of sputum, nasal mucus or urine, multinuclear giant cells can be detected. Measles virus can be isolated from the same materials in cell culture. Measles antigen can be detected in the epithelium of the respiratory tract by immunofluorescence. Serological methods are also used (RSK, RTGA, RIF, etc.). Diagnostic is considered to be a titer increase of 4 times or more.

Meningococcal infection

Meningococcal infection is an acute infectious disease caused by meningococci (Neisseria meningitidis) and characterized by periodic epidemic outbreaks. These outbreaks occur at intervals of 25-30 years, more often children under 5 years old are affected, but people of any age can be sick. It is customary to distinguish between localized (meningococcal carriage and acute nasopharyngitis) and generalized forms (meningococcemia and purulent meningitis or meningoencephalitis). Meningococcus (Neisseria meningitidis) has the form of coffee beans located both extra- and intracellularly, and is determined in smears from the nasopharynx or cerebrospinal cerebrospinal fluid, is very sensitive to external influences (temperature, pH, humidity), therefore, outside the body and in the corpse quickly dies. The source of meningococcus is a sick person or a carrier of bacteria. Infection occurs predominantly by aerosol. Standing out with droplets of mucus from the upper respiratory tract, the pathogen enters the body of a healthy person when inhaling air containing meningococci. The incidence is higher in February - April (seasonal rise).

Pathological anatomy and pathogenesis. The ingress of meningococcus into the mucous membrane of the nasopharynx only in 10-30% of cases causes the development of meningococcal nasopharyngitis. The main clinical symptoms of nasopharyngitis are: pain and sore throat, dry cough, nasal congestion, runny nose with scanty mucopurulent discharge, sometimes bloody, headache, fever. Dizziness, nosebleeds are possible. Macroscopically, meningococcal nasopharyngitis is characterized by catarrhal inflammation of the mucous membranes with especially pronounced hyperemia, swelling of the posterior pharyngeal wall and hyperplasia of the lymphatic follicles. This form is of great epidemiological importance, as it is often not clinically diagnosed.

Meningococcemia. Depending on the state of the body's immune reactivity, meningococcus can cause sepsis, called meningococcemia, which sometimes has a fulminant course. The basis of vascular lesions in meningococcemia is bacterial shock, resulting from the intensive decay of phagocytosed bacteria with the release of their endotoxin. Structural changes in meningococcemia consist of moderately expressed inflammatory changes and sharply prevailing in the clinical and morphological manifestations of circulatory disorders. Paresis of small vessels is observed with the development of stasis, thrombosis, hemorrhage and, then, necrosis in the organs. Meningococcemia begins acutely, is manifested by fever, the occurrence of a characteristic rash on the 1-2 day of the disease, more often in the form of irregular shape of asterisks of various sizes, less often - small-point or extensive hemorrhages. In the latter cases, the disease is very difficult, with impaired cardiovascular activity, bleeding and hemorrhage in the internal organs. With timely treatment, the prognosis in most cases is favorable. Meningococcemia is characterized by generalized damage to the microvasculature, skin rash, joint changes

(purulent arthritis), serous membranes (purulent pericarditis), choroid (purulent iridocyclitis and uveitis), adrenal glands and kidneys. If the patient dies in the first 24-48 hours, meningitis may be absent.

Microscopic examination is typical of the detection in the tissues of various organs of small accumulations of meningococci, surrounded by very moderate loose neutrophilic lymphocytic infiltrates. Changes of this kind are more often observed in the soft meninges, somewhat less often in the heart, liver, kidneys, skin, and occasionally in the thymus and lungs. Macroscopically inflammatory changes, with the exception of a slight thickening and turbidity of the meninges, are not determined. Among the circulatory disorders, a mandatory component is the manifestation of thrombohemorrhagic syndrome. In typical cases, a rash is of great diagnostic value. With a minimum degree of its intensity, plethora of small venules with swelling of the vascular endothelium is detected. With a greater severity of the process, extravasates and moderate lymphocytic leukocyte infiltration are detected, mainly the walls of the blood vessels. Thrombi are determined in the lumen of small vessels. In addition, diffuse blood impregnation of all layers of the skin and areas of necrosis are noted. Macroscopically, small elements are initially revealed in the skin that can be rose-colored and rose-papular. Subsequently, the elements of the rash increase in size, it becomes hemorrhagic, often acquiring a star shape. In the center of large drainage elements, yellowish areas of necrosis may be noted. The most typical localization of the rash on the buttocks, back of the thighs and legs. Another manifestation of thrombohemorrhagic syndrome is adrenal gland damage, usually with total or subtotal hemorrhages, followed by necrosis of both the cortical and brain layers. This is accompanied by acute adrenal insufficiency (Waterhouse-Friedericksen syndrome). Among other localizations of hemorrhages during meningococcal infection, the mucous membranes of the digestive tract, trachea, and bladder should be noted. Necrotic nephrosis is detected in the kidneys. In smears from the affected organs, meningococci can be detected if the autopsy is performed no more than 10-18 hours after death. Meningococcemia often occurs against the background of violations of the immunogenesis system and generalized viral infections, especially influenza. The death of patients with fulminant course occurs from bacterial shock, the severity of which is aggravated by hemorrhages in the adrenal glands, acute renal failure is less common (in adults). With a longer course, the death is due to septicopyemia or purulent meningitis.

Meningitis also usually begins suddenly and within 1-2 days a pronounced clinical picture develops. Relatively rarely, mainly in young children, meningococcus spreads by the hematogenous route, overcomes the blood-brain barrier and is fixed in the pia mater, where it causes purulent meningitis. The predominant disease of children in the first 5 years of life is associated with the structural immaturity of this barrier. With purulent meningococcal meningitis (meningoencephalitis), gradually increasing infiltration of the soft meninges, mainly represented by neutrophilic leukocytes with an admixture of lymphocytes, monocytes and a small number of eosinophils, in some cases with fibrin prolapse, is determined. The most dense infiltrates are located around the blood vessels. Accumulations of neutrophilic leukocytes and small thrombi are usually determined in the lumen of blood vessels. Similar, although much less pronounced, changes are noted in the wall of the cerebral ventricles. In the substance of the brain, inflammatory changes are usually defined as purulent vasculitis. In addition, pronounced ischemic and dystrophic changes in nerve and glial cells are detected. Macroscopically, the inflammatory process that develops in the meninges is initially serous or serous-purulent in nature. The soft meninges become sharply full-blooded, impregnated with a slightly unclear serous exudate. The exudate gradually thickens, acquires a greenish-yellow color and purulent character. From 2-3 days, the exudate becomes purulent and accumulates mainly in the form of yellowish-gray masses in the furrows around the veins. Purulent infiltration is localized on both the basal and convexital surfaces, located here in the form of a yellowish-greenish “bonnet” or “cap”. In some cases, the whole brain is covered with a continuous layer of yellowish purulent masses. By the 5th-6th day, the exudate is even more condensed from the addition of a fibrinous effusion. From the medulla oblongata, the exudate can directly go to the membranes of the spinal cord, as well as the membranes of the cranial and spinal nerves. Changes in the spinal cord are inconsistent and usually mild. In some cases, pus-like masses are determined in the lumen of the ventricles, and the ependyma lining them looks edematous, cloudy. With a long course of the disease, cell lysis and exudate resorption gradually occur. The number of granulations, and subsequently the connective tissue, in contrast to pneumococcal lesions, is small. In

the later stages of the disease, mainly around the ventricles, glial proliferation is noted. Ventricular ependyma and vascular plexuses can also be involved in the process with the development of purulent ependimitis and piocephaly, which are more often observed in children of the first 2-3 years of life. Microscopically, the vessels of the soft meninges are sharply full-blooded, the subarachnoid space is dilated, saturated with leukocyte exudate, penetrated by fibrin threads. The process from the choroid can pass to the brain tissue with the development of meningoencephalitis. Starting from the 3rd week of the disease, the exudate undergoes resorption. With a large amount of fibrin, it is organized with obliteration of sections of the subarachnoid space of the middle and lateral openings of the IV ventricle and difficulty in circulating cerebrospinal fluid. The consequence of this is progressive hydrocephalus with increasing atrophy of the brain substance. Death can occur in the acute period from swelling of the brain with insertion of the tonsils of the cerebellum into the large occipital foramen and infringement of the medulla oblongata in it or, in subsequent periods, from meningoencephalitis, purulent ependymitis, and later from general cerebral cachexia due to hydrocephalus and cerebral atrophy.

Sometimes the same patient develops all these forms of the disease. Carriage of meningococci is often observed in the absence of symptoms of the disease - meningococcal carriage, due to which the pathogen circulation in the teams is mainly maintained. The most dangerous from an epidemiological point of view is a patient with nasopharyngitis, accompanied by a runny nose, cough, sneezing, which contributes to the intensive spread of pathogens.

3. Lesson plan

To study the following macropreparations, to describe them according to the scheme for the description of macropreparations.

Macropreparations.

1. **Measles pneumonia.** To study the macropreparation “Measles pneumonia”. Characterize the sectional view, indicate the macroscopic features of the lung with measles.
2. **Diphtheria angina, croupous laryngitis and tracheitis**. Describe the macropreparation "Diphtheria angina, croupous laryngitis and tracheitis." Pay attention to the condition of the mucous membranes of the pharynx, larynx, trachea.
3. **Purulent meningitis**. To study the macropreparation purulent meningitis. Pay attention to the localization of the pathological process (indicate which lobes are affected). Describe the state of the meninges and ventricles of the brain.

Examine the following micropreparations, sketch them, indicate and mark the pathological changes with arrows, using the atlas of micropreparations.

Micropreparations.

1. To study the micropreparation "**Measles pneumonia**" to note the presence of giant multinucleated cells. Characterize changes in the alveolar septum, peribronchial tissue.
2. Examine the “**Measles encephalitis”** micropreparation. Pay attention to perivascular infiltrates from glia cells and mesenchymal elements.
3. To study the micropreparation "**Parenchymal myocarditis in diphtheria**." Pay attention to the foci of cardiomyocyte necrosis, vascular congestion and interstitial infiltration by lymphoid cells, individual neutrophilic leukocytes.
4. To study the micropreparation "**Necrotic tonsillitis with scarlet fever**." Pay attention to the foci of necrosis that capture the underlying tissues, cell infiltration: polymorphic nuclear leukocytes. Plethora of vessels.
5. Examine the “**Purulent meningitis**” micropreparation. pay attention to a sharp thickening of the pia mater and diffuse neutrophilic leukocyte infiltration, swelling of the brain tissue.

Solve the following situational tasks using the tutorial.

Situational tasks

Situational task 1.

A girl 20 years old in childhood suffered from complicated measles. Currently, severe pulmonary heart failure, shortness of breath, fingers in the form of drumsticks, nails in the form of watch glasses, cough with collected mucopurulent sputum (especially in the morning). An X-ray examination determines a sharp expansion of the bronchi. Is this condition related to measles? What pathological process does the respiratory system take? What is the cause of pulmonary heart disease?

Situational task 2.

A 15-year-old patient with signs of general cerebral cachexia was delivered to the clinic. From the anamnesis it is known that 2 years ago he suffered meningococcal meningitis. the examination revealed hydrocephalus. What pathological changes in the brain led to the development of hydrocephalus?

Situational task 3.

A child of 8 years old had severe pain when swallowing, swelling of the neck, body temperature increased to 39C. During the examination, it was found: Difficult to separate grayish films on the tonsils, enlargement of the cervical lymph nodes, systolic murmur in the heart, atrial fibrillation. Pronounced general intoxication. Your diagnosis. What is the development of symptoms associated with? what is the possible outcome of the disease?

Situational task 4.

A child of 6 years had a sore throat, body temperature increased. On the 2nd day from the onset of the disease, a small point rash was found covering the surface of the body, with the exception of the nasolabial triangle. Upon examination of the throat - bright red pharynx and tonsils, raspberry tongue, on the surface of the tonsils - small grayish foci of necrosis. What is your diagnosis?

Answer the following questions of the current test control.

1. **Diphtheria is infected by all of the following routes, except:**

a) transmission;
b) contact household;
c) alimentary;
g) airborne droplets.

2. **When diphtheria is most often affected:**

a) nose;
b) oropharynx;
in the eyes;
d) external genitalia;
e) skin.

3. **For a localized form of diphtheria of the oropharynx, the occurrence of:**

a) mild sore throat;
b) hyperemia of the mucous membranes of the pharynx;
c) membranous raids on the tonsils, passing on the arches and tongue

4. **The material for the isolation of the causative agent of diphtheria is:**

a) mucus from the nose and pharynx;
b) blood;
c) urine;
d) feces.

5. **For the specific treatment of patients with diphtheria is used:**

a) antitoxic antidiphtheria serum;
b) anti-diphtheria gamma globulin;
c) diphtheria toxoid

6. **Source of measles infection:**

a) mammals;
b) a person;
c) rodents;

d) arthropods.

7. **Threatened age with measles:**
a) after 14 years;
b) adults;
c) children 1-5 years old;
d) children under 1 year old.

8. **The main variant of exanthema for measles:**
a) stain;
b) papule;
c) stain + papule;
d) petechia;
e) vesicle.

9. **Pathognomonic symptom with measles:**
a) trismus;
b) hydrophobia;
c) spastic syndrome;
d) Belsky-Filatov-Koplik spots.

10. "**Vaccination Measles" is:**
a) measles after глоб-globulin prophylaxis;
b) after preliminary vaccination;
c) measles after blood transfusion;
d) after damage to the skin (inoculation).

11. **The causative agent of scarlet fever:**
a) E. coli;
b) yersinia;
c) β-hemolytic streptococcus;
g) spirochete.

12. **What is not typical for a typical exanthema with scarlet fever:**
a) hyperemic background;
b) necrosis of the elements of the rash;
c) concentration in the folds of the skin;
d) a pale nasolabial triangle;
d) subsequent lamellar peeling.

13. **What are the complications of scarlet fever:**
a) myocarditis;
b) synovitis;
c) otitis media;
g) mastoiditis;
e) septicemia, septicopyemia.

14. **Which of the following does not apply to measures to prevent scarlet fever:**
a) separation of contact;
b) quarantine of the team;
c) vaccination.

15. **The most threatened contingent for scarlet fever infection:**
a) from 2 to 7 years;
b) from 2 to 10 years;
c) 10-14 years;
d) adults.

16. **The causative agent of scarlet fever:**
a) pneumococcus;
b) β-hemolytic streptococcus;
c) green streptococcus;
g) enterococcus.

17. **What disease is this rash characteristic of: "small-pointed, against a hyperemic background, with predominant localization in the folds":**
a) measles;
b) rubella;
c) scarlet fever.

4. *List of recommended literature:*

Basic literature:

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

Additional literature:

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3. “General and Systemic Pathology” Joseph Hunter, 2002.

4. “General and Systematic Pathology ” Ed. J.C.E. Underwood – Edinburgh: Churchill Livingstone, 1996 (2^{th}).

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6. “Histopathology. A Color Atlas and Textbook” Damjanov I., McCue P.A. – Baltimore, Philadelphia, London, Paris etc.: Williams and Wilkins, A Waverly Co., 1996.

7. “Muir’s Textbook of Pathology” Eds. R.N.M. MacSween, K. Whaley London: ELBS, 1994 (14^{th}).

8. “Pathology” Eds. Rubin, J.L. Farber – Philadelphia: Lippincott Raven Publ, 1998 (3^{th}).

9. “Pathology Illustrated” Govan A.D.T., Macfarlane P.S., Callander R. – Edinburgh: Churchill Livingstone, 1995 (4^{th}).

10. “Robbins Pathologic Basic of Disease” Eds. R.S.Cotran, V.Kumar, T.Collins – Philadelphia, London, Toronto, Montreal, Sydney, Tokyo: W.B.Saunders Co., 1998 (6^{th}).

11. “Wheater’s Basic Histopathology. A Color Atlas and Text” Burkitt H.G., Stevens A.J.S.L., Young B. – Edinburgh: Churchill Livingstone, 1996 (3^{th}).

12. “Color Atlas of Anatomical Pathology” Cooke R.A., Steward B. – Edinburgh: Churchill Livingstone, 1995 (10^{th}).

13. “General Pathology ” Walter J.B., Talbot I.C. – Edinburgh: Churchill Livingstone, 1996 (7^{th}).

14. “Concise Pathology” Parakrama Chandrasoma, Glive R. Taylor.

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16. “Short lectures on pathology” Zagoroulko A., 2002

17. “Robbins pathologic basis of diseases” Cotran R., Kumar V., Collins T.

18. “General pathology” Dr. Fatma Hafez, 1979.

19. “Anderson’s Pathology ” Damjanov I., Linder J. – St. Louis: Mosby Inc., 1995 (10^{th}).

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