MINISTRY OF HEALTH OF UKRAINE

DANYLO HALYTSKY LVIV NATIONAL MEDICAL UNIVERSITY

PEDIATRIC INFECTIOUS DISEASES DEPARTMENT

GUIDELINES

TO PRACTICAL TRAINING OF PROFILE COURSES OF CHOICE

“OBSTETRICS AND GYNECOLOGY”

FOR 6TH YEAR STUDENTS OF THE MEDICAL FACULTY

SPECIALTY

"GENERAL MEDICINE"

TOPIC:

"HIV infection and AIDS in children. TORCH- infection in children. Prophylaxis of infectious diseases in children: specific and non-specific"

LVIV-2021

Guidelines are made according to the Study program on Pediatric infectious diseases for students of the second (Master Dergree) level of higher education in the field of knowledge 22 " Health Care " specialty 222 "General Medicine"

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for students of the 6th year of General Medicine Faculty
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Guideline to lesson for 6th year students

(practical classes - 6 hours)

«HIV infection and AIDS in children.

Prophylaxis of infectious diseases in children: specific and non-specific»

I

Aim: to know diagnostic criterions of HIV/AIDS in children, how to examine a patient with this disease, and prove the diagnosis, differentiate, give the individual treatment and prevention.

Professional motivation:

As of 2018, of the estimated nearly 38 million people worldwide living with HIV, approximately 1.7 million are children under 15 years of age. Since 2010, new HIV infections among children have declined by 41%, but only half (54%) of all children living with HIV are getting treatment and 100,000 children died of AIDS-related illnesses in 2018. About 500 children are newly infected with HIV every day**.**

More than 90 percent of HIV infections in children result from mother-to-child-transmission, where the virus is passed from a mother living with HIV to her baby during pregnancy, childbirth, or breastfeeding. The risk of this form of transmission increases in direct relation to the severity of the mother's HIV infection.

Basic level

1. To know how to ask complaints, history of the disease and life in children [propedeutic pediatrics].
2. To perform clinical examination of the child [propedeutic pediatrics]To know microbiology, pathophysiology, pathomorphology and clinical features of HIV/AIDS [Microbiology, pathophysiology, and pathomorphology, Pediatric infectious diseases].
3. To diagnose HIV/AIDS after clinical, laboratory and instrumental examination of sick person [infection diseases, propedeutic pediatrics, microbiology, and pathophysiology].
4. To give etiological, pathogenetical and symptomatical treatment of HIV/AIDS [pharmacology].
5. To prevent diseases that may be complicated by HIV/AIDS.

II. Primary aims of the study

A student should know:

- Etiology and epidemiology of a AIDS/HIV infection; TORCH- infection in children
- Pathogenesis and pathological anatomy;
- Classification of a AIDS/HIV infection;
- The clinical characteristic of different kinds of a AIDS/HIV infection; TORCH- infection in children
- Laboratory methods of examination at AIDS/HIV infection; TORCH- infection in children

- Complications, which are observed at a AIDS/HIV infection; TORCH- infection in children
- Differential diagnostics of a AIDS/HIV infection; TORCH- infection in children
- Medical tactics;
- Preventive and antiepidemic measures in the locus.

A student should be able:

• to question a patient in order for obtaining of information on disease history and epidemiologic anamnesis;

• to perform clinical examination of a patient;

• to formulate and to substantiate the diagnosis of AIDS/HIV infection; TORCH- infection

• to prepare a plan of additional patient examination;

• to evaluate results of laboratory examination;

• to make differential diagnosis to distinguish between similar diseases;

• to prescribe adequate pathogen and etiotropic treatment.of AIDS/HIV infection; TORCH- infection

III. Educational aims of the study

- to conduct clinical examination of a AIDS/HIV infection patient and other acute intestinal diseases;
- to formulate and substantiate a clinical diagnosis;
- to prepare a plan of paraclinic patient examination;
- to take samples of material for virologic and other quick analysis methods examination;
- to evaluate results of paraclinic patient examination;
- to organize hospitalization and treatment of a AIDS/HIV infection patient; TORCH- infection
- to plan and organize prophylactic measures against AIDS/HIV infection ; TORCH- infection

IV. Interdisciplinary integration

Table 1

Subjects	To Know	To Know How
1	**2**	**3**
Human Anatomy	The main anatomic characteristics of system target systems.	
Physiology	Functions of the respiratory, nervous and immune systems	To explain a variety of clinical signs and laboratory abnormalities
Pathological Physiology	Pathogenesis of disease	To explain the main symptoms and manifestations appearance, causes of relapses, failure of inadequate therapy
Pathological Anatomy	Pathology	To explain the pathogenesis of complications and causes of death
Microbiology	Etiology (classification, morphologic characteristic of the pathogen, methods of revealing and identification)	To culture the organism
Pharmacology	The main antiviral and antibacterial agents. Regimens of treatment. Treatment of complicated influenza. Supportive care	To write the scheme of treatment of severe HIV/AIDS.
Histology	Histological changes in different clinical forms of influenza	Explanation of appearance of clinical signs

Continuing **Table 1**

1	2	3
Propedeutics of Internal Diseases	History of disease. Examination of a patient.	To gather information about patient's history and chief complaints, to distinguish the ones, most important for diagnosis of HIV/AIDS. To examine the patient, to reveal the main symptoms and signs of disease. To distinguish the set of diagnostic features of HIV/AIDS. To argue the diagnosis.
Surgery	Chest pain, cough, respiratory failure	Differential diagnostics of surgical disorders, diagnostics of complications
Internal Diseases	Chest pain, cough, respiratory insufficiency	To differentiate with other disorders of respiratory system
Neurology	Severe headache, vomiting, meningeal signs, delirium, altered consciousness	Differential diagnostics of encephalitis, meningitis, stroke
Clinical immunology and allergology	Immunologic changes as a part of pathogenesis and host defenses	To explain confirmative serologic tests
Epidemiology	The routes of transmission, main sources of infection	Epidemiological history
Themes integration		
Encephalitis, meningitis, common cold, ARVI, diphtheria, tonsillitis, intestinal infection.	To know peculiarities of manifestations, laboratory diagnostics, treatment	To differentiate HIV/AIDS from other infectious diseases with similar symptoms

V. The contents of the theme

Secondary immunodeficiency syndrom is caused by a virus and characterized by severe immune deficiency resulting in opportunistic infections, malignancies, and neurologic lesion in individuals without prior history of immulogic abnormality.

Etiology. The cause is a retrovirus that has been termed the human T-lymphotrophic virus Type III (HTLV-III), lymphadenopathy-associated virus (LAV), and the AIDS-associated retrovirus (ARV) by different laboratories. More recently, it has also been referred to as the human immunodeficiency virus (HIV), the term that will be used here.

Retroviruses are very small viruses composed of a single strong of RNA, the intermediate nucleic acid in the production of proteins. Normally, the flow genetic information starts with a piece of DNA, which makes a piece of RNA, which in turn codes for, protein. Everything flows in that direction.

Retroviruses contain an enzyme called reverse transcriptase that can convert viral RNA in the cytoplasm into DNA, which may replicate from extrachromosomal sites or move into the cell nucleus where it becomes part of the host cell DNA. These integrated viral genes are duplicated with normal cellular genes, and all progeny of the originally infected cell will contain the viral genes. Expression of the viral genes for some retroviruses may be oncogenic, converting the cell into a cancer, or may have other pathologic effects which may alter normal cell function or produce cell death. Retroviruses have been known to cause malignant and nonmalignant diseases, and the same virus may cause different diseases in different animals; e.g., bovine leukemia virus causes a B cell lymphoma in cows, a T cell lymphoma in sheep, and an immunodeficiency disorder similar to AIDS in rabbits.

There are 3 groups of retroviruses that affect humans, and all have a remarkable affinity for lymphocytes, particularly for T4 lymphocytes. HIV preferentially infects the major subset of T-cells, defined phenotypically as T4 and functionally as "inducer/helper" cells, which are then depleted, resulting in a reduced ratio of T4 helper (Th) to T8 suppressor (Ts) cells. However, the virus also is capable of infecting some nonlymphoid cells, such as macrophages and nervous tissue cells, and presumably remains present for life.

Epidemiology. The major transmission routes of human immunodifficiency virus are sexual contact, parenteral exposure to blood and blood products and perinatal transmission. Early in the AIDS epidemic, epidemiological studies establish that receptive rectal intercourse was the predominant mode of HIV-1 acquisition by homosexual man. Other practices that could traumatize the rectal mucosa appeared to increase further the infection risk for the receptive partner. Insertive rectal sex could also place a men at risk for HIV-1 infection, although the insertive partner would be at lower risk than the receptive partner.

On a world-wide basis sex between man and women apparently is the most common mode of acquiring HIV-1 infection heterosexual transmission accounts for the vast majority of cases. In other country where AIDS cases attributed to heterosexual transmission, although still a small percentage of the total number of reported cases comprise the most rapidly growing category. Therefore an understanding of the rate at which HIV-1 is transmitted between heterosexual couples and of the factors that may impede or enhance heterosexual transmission is important in slowing the worldwide HIV-1 epidemic.

In the country, where HIV-1 infection is more common in men than women, studies of female-to-male transmission of HIV-1 infection are both fewer and smaller than studies of male-to-female transmission. Available date suggest female-to-male transmission may be less efficient than male-to-female transmission.

Overall these American and European studies suggest that heterosuxual transmission from HIV-infected persons to their regular sex partner is relatively-inefficient, especially female-to-male transmission. Furthermore, the risk of heterosexual transmission is not related simply to the number of episodes of sex with HIV-infected person because some people have remained uninfected after hundreds of such contacts whereas others have become infected after a single episode of intercourse.

Infectivity may be higher during early infection before the development of antibodies to HIV-1, also genital ulcer diseases and inflammation of the genital tract lead to increased susceptibility to HIV infection.

Perinatal or vertical transmission. Mother-to-infant transmission of HIV apparently is relatively efficient; without treatment approximately one in the Sour infants born to seropositive mothers is infected. With one rapid spread of infection to women of reproductive age perinatal transmission is now a major consequence of HIV epidemic. The precise rate of perinatal transmission in a given setting has been difficult to define because of problems in the infant and the difficulties in maintaining long-term follow-up. Uninfected children born to seropositive mothers may retain passively aquired maternal antibody for 6 to 18 months.

The timing mechanisms, and risk factors for perinatal transmission might occur and guiding the development of effective interventions. These factors include maternal stage of disease maternal antibody response to infection, viral titer, variations in viral genotype and phenotype, and obstetric factors such as preterm birth, mode of delivery, and maternal or placental coinfection. Perinatal HIV transmission can occur both in utero or at birth. Several lines of evidence support the occurrence of human placenta tissue express CD4 receptors and are suseptible to HIV infection. Virus has been isolated from amniotic fluid and has been identified in fetal abortus tissue by culture, PCR and in situ hybridization. However, other investigators have not found HIV in fetal tissue and it is difficult to exclude contamination of these tissues by maternal blood. Clinically, the fact that subsets of infected infants have detectable virus at birth immunologic abnormalities in the neonatal period and rapid progression to AIDS in the first Sour months of life suggest in the utero transmission. The proportion of infants actually infected in utero and the time during gestation when this is most likely to occur, however, are not known.

Intraportum transmission, analogous to the vertical transmission of hepatitis B, likely occurs through direct contact with maternal blood secretions as the infant is delivered through the birth canal. HIV has been isolated from cervical secretions. Also the virus might be able to pass directly through maternal-fetal

transfusions, particularly during placental separation at birth. The fact that many infected children are born without detectable virus or immunologic abnormalities supports the likelihood that delivery represents a high risk for HIV transmission. Interestingly there has not been strong evidence that that delivery by caesarean section is protective. However, a recent report based on an international twin registry suggests that being the first of two twins delivered and vaginal delivery are risk factors for infection in twin births. This hypothesis and its relevance for singleton births warrant further study. Although it has not been shown that intrapartum fetal scalp monitoring facilitates transmission, avoiding invasive of the fetus whenever possible, seem prudent.

Postpartum perinatal transmission of HIV through breast-feeding has been reported. Free virus has been found in the cellfree fraction of breast milk and might directly penetrate the infant's gastrointestinal mucosa. However, data from several cohort studies suggest that the additional risk of postpartum transmission is low in pregnant mother already infected with HIV. These finding may result from low viral titers in breast milk of previously infected women, concomitant IgA antibody or some other factor.

Extensive laboratory research and epidemiological studies indicate that HIV is not transmitted by shaking hands, hugging, kissing, contacting bodily secretion such as sweat, mucus (as in sneezing or coughing) or salive. Nor is HIV transmitted by food, swimming at a pools, drinking at a water fountain and also bloodsucking mosquitoes or other arthropods.

Pathogenesis. Following infection across a genital surface, involving infection of CD-4 bearing cells in the mucosa or submucosa, the virus presumably migrates to a regional lymph node, where viral replications occurs. A number of rounds of viral replication than occur within the bounds of the regional lymph node as no detectable virus or immune respons occurs for up to 42 days post infection. When the quantity of infected cells exceeds a threshold,viremia occurs,and the symptoms of an acute non-specific viral illness with tende adenopathy, sore troath, diffuse macular rush, arthralgia and fever. Following the acute viremia, when up to 10^7 viral particles/mL plasma can be found, a primary immune response develops with antibodies to viral proteins and a cytotoxic T-cells response, which limit viral replication and clear viral particles from the plasma. The reduction the viral load in plasma is not matched by a clearance of provirus in peripheral blood mononuclear cells, and cellular viremia continues in the face persistent and sustained cellular and humoral immune response for the duration of the infection. Even while plasma viral load is suppressed by the immune response, CD-4 T-lymphocyte number foil in linear manner over time. The most plausible explanation for the pathogenesis of AIDS over time is the sustained less CD-4 cells by ongoing HIV viral replication in nature peripheral blood T-cells and by a slight failure of production of match peripheral destruction of HIV CD-4 cells. However, recent controversy over the pathogenic mechanisms and homestasis of T-cells has revealed that simple viral cytopathic effect on CD-4 cells may be overly simplistic model.

During the course of HIV infection, CD-4 cells continue to decline in peripheral blood and plasma viral load slowly rises. Over a definite period CD-4 cells number declines from 800 to 200 mL; at this level, the probability of the cellular immune system containing latent or environmental infections such as *P. carinii* falls and clinical opportunist infection becomes* increasingly possible. As the viral 1 retrovirus load rises. HIV isolates with altered co-receptor usage appear which can use the CXCR-4 chemocine receptor rather than CCR-5; these isolates are more cytopathic in vitro, and may lead to wider tissue distribution of HIV in later disease, AIDS is therefore, the clinical condition of an immune system which is sufficiently compromised by HIV infection that there is an inability to protect against the growth of low grade pathogenes or viral indeced tumors.

The fact that this virus, after infection of the host, besides destroyed strong immune system also can spread to many body tissues. The ultimate outcode of the infection depends on the host's immune reaction to the virus either through suppression of HIV replicationor through killing of the infected cell. In some individuals an active immune system has prevented development of the disease for years. The factors important in maintaining this immune response are not yet know and merit close attention. The immune deficiency produced by HIV infection makes patients suseptible to infection by a variety of organisms, including viryses, bacteria, fungi and parasites that are of low pathogenecity in the normal individual and of variable prevalence in different part of the world. In some individuals the immune system appears to make enhancing antibodies to HIV and this phenomenon occurs particularly with progression of disease. It is related to change to antibodies made and in some cases to modifications in the virus so that is more sensitive to enhancing antibodies. Moreover, the immune system can hyperreact, with production of antibodies that

might also hasten the development of disease. Clearly changes in the virus and the immune response of the host play important roles in the ultimate steps leading to AIDS.

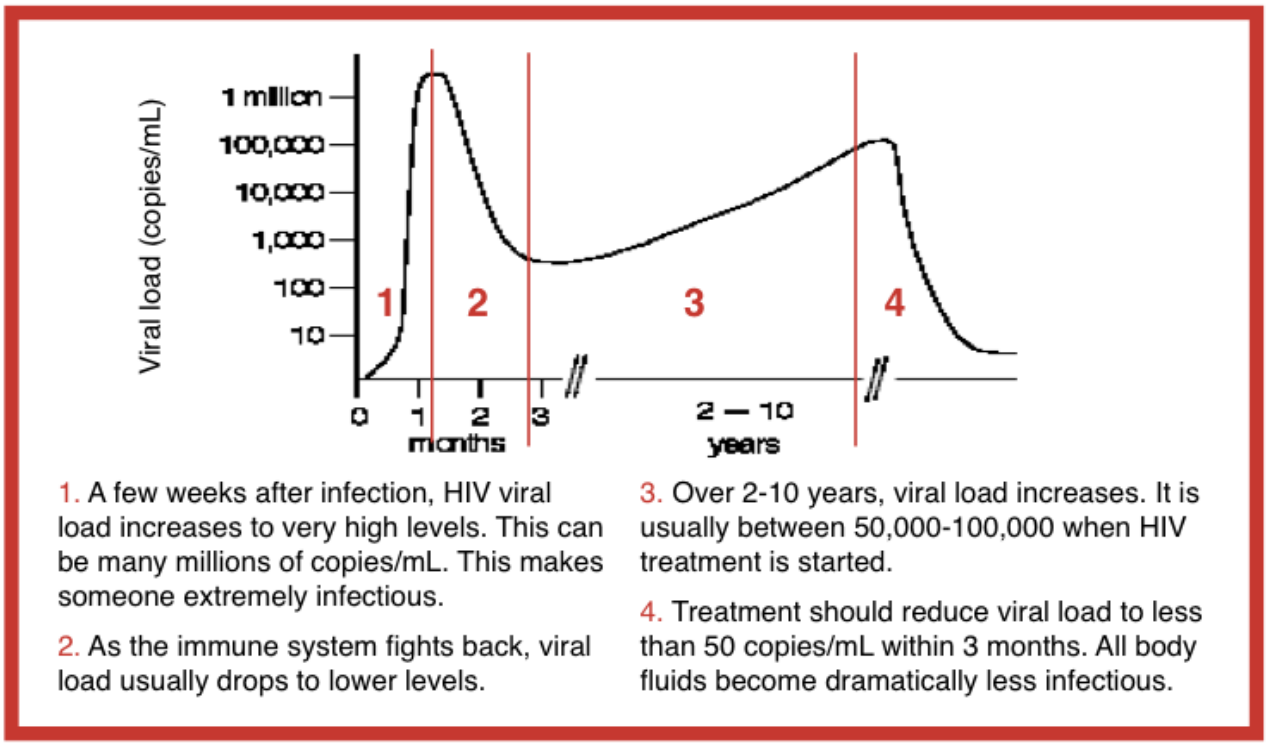
Anatomic pathology. Forty to sixty percent of AIDS patients develop neurologic dysfunction and up to 90 % have neuropathologic changes at autopsy. HIV itself can cause brain disease manifested as meningoencephalitis, mild cognitive dysfunction, or frank dementia. It is felt that the pathogenesis of this neurologic damage is related to the presence of infected tissue macrophages that may release viral proteins or cytokines that result in brain dysfunction, inflammation, and tissue destruction. In this regard, studies of brain tissue from AIDS patients have shown that the predominant cell type infected with HIV is the monocyte/macrophages (M/M).

Infected M/M may release factors resulting in reactive glial cell growth, and, because glial cells have been shown to be infectable with HIV in vitro, infected brain M/M may provide a source of infectious HIV to these glial cells. The HIV envelope protein can inhibit neuronal growth in vitro; this may be due to competition between neuroleukin and gpl20 for binding to the neuroleukin receptor, because there is partial sequence homology between these two proteins. It is possible, but not yet demonstrated, that infected M/M in the brain may release large quantities of gpl20 resulting in the inhibition of neuronal growth.

A wide variety of hematologic abnormalities occur in HIV-infected individuals including pancytopenia and myelodysplasia. Although the etiology of these multiple abnormalities has not been completely delineated, it has been shown that the CD34+ bone marrow myeloid progenitor cell can be infected with HIV in vitro with the resultant production of large amounts of predominantly intracellular virus and minimal cytopathic effects. More recently, CD34+ cells isolated from the bone marrow of some infected individuals have been shown to be infected with HIV. Whether these precursor cells produce large amounts of virus in the bone marrow in vivo and the potential contribution of these cells to the hematologic abnormalities observed are currently unknown. Infected macrophages within the bone marrow have been reported to produce factors, presumably cytokines, which appear to suppress hema-topoiesis through their effects on the CD34 + precursor cell. Whether bone marrow macrophages are an important reservoir of HIV has not been definitively determined.

Finally, cells of the monocytic lineage that populate other organs are susceptible to infection with HIV and may contribute to pathogenesis of disease at these sites. Specifically, lung alveolar macrophages, Kopffer cells of the liver, and peritoneal macrophages are infectable in vitro with HIV, and alveolar macrophages from HIV-infected individuals are clearly infected in vivo. It is currently unknown whether these cells contribute to tissue-specific disease, such as the diffuse pulmonary fibrosis that occurs frequently in pediatric AIDS patients.

Fig. 1. Clinical manifestations.



The symptoms vary depending on the age of the child. The following are the most common symptoms of HIV infection. However, each infant, child, or adolescent may experience symptoms differently. Symptoms may include:

- **Infants.** HIV status may be difficult to determine in the first year of like, so repeated tests may be done. Symptoms may include:
- **Failure to thrive.** Delayed physical and developmental growth as evidenced by poor weight gain and bone growth.
- **Swollen abdomen.** This is due to swelling of the liver and spleen.
- **Swollen lymph nodes**
- **Intermittent diarrhea.** Diarrhea that may come and go.
- **Pneumonia**
- **Oral thrush.** A fungal infection in the mouth that is characterized by white patches on the cheeks and tongue. These lesions may be painful to the infant.

- **Children.** Symptoms seen in children older than 1 year of age can be divided into three different categories, from mild to severe. They may include the above symptoms, but may also include the following:

Mild	Moderate	Severe
Swollen lymph nodes	Pneumonitis--swelling and inflammation of lung tissue	Two serious bacterial infections in a two-year period (meningitis, blood infection, or pneumonia)
Swelling of the parotid gland (salivary glands located in front of the ear)	Oral thrush that lasts for more than two months	A yeast infection that occurs in the digestive track or lungs
Constant or recurring sinus infections	Constant or recurring diarrhea	Encephalopathy--an inflammation of the brain
Constant or recurring ear infections	A fever that persists for more than one month	Tumors or malignant lesions
Dermatitis--an itchy, rash on the skin	Hepatitis--an inflammation of the liver that is often caused by an infection	Pneumocystis jiroveci pneumonia (the type of pneumonia most commonly seen with HIV)
Abdominal swelling from increased liver and spleen size	Complicated chickenpox	
	Kidney disease	

- **Adolescents.** Symptoms of HIV in teens may be the same as in children, and may also be more similar to the symptoms commonly seen in adults with HIV. Some teens and adults may develop a flu-like illness within a month or two after exposure to the HIV virus, although, many people do not develop any symptoms at all when they first become infected. In addition, the symptoms that do appear, which usually disappear within a week to a month, are often mistaken for those of another viral infection. Symptoms may include:

- Fever
- Headache
- Malaise (not feeling well)
- Enlarged lymph nodes

Persistent or severe symptoms may not surface for 10 years or more, after HIV infection first enters the body in teens and adults. This "asymptomatic" period of the infection is highly variable from person to person. But, during the asymptomatic period, HIV is actively infecting and killing cells of the immune system. Its most obvious effect is a decline in the blood levels of CD4+ cells (also called T4 cells)--the immune system's key infection fighters. The virus initially disables or destroys these cells without causing symptoms.

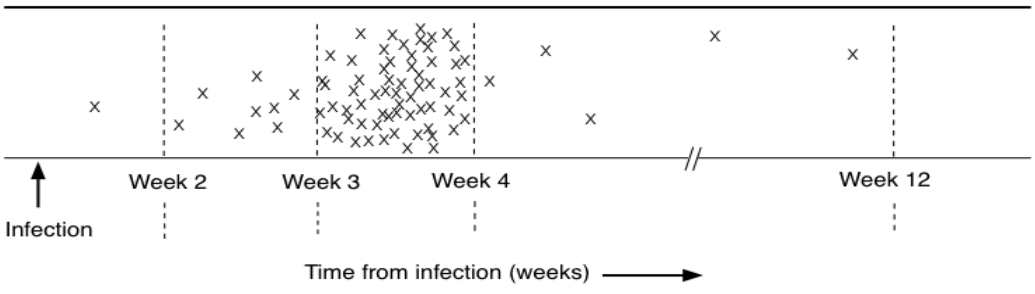
An HIV-infected child is usually diagnosed with AIDS when the immune system becomes severely damaged or other types of infections occur. As the immune system deteriorates, complications begin to develop. The following are some common complications, or symptoms, of the onset of AIDS. However, each child may experience symptoms differently. Symptoms may include:

- Lymph nodes that remain enlarged for more than three months
- Lack of energy
- Weight loss
- Frequent fevers and sweats
- Persistent or frequent yeast infections (oral or vaginal)
- Persistent skin rashes or flaky skin
- Pelvic inflammatory disease that does not respond to treatment
- Short-term memory loss
- Severe or unusual infections (opportunistic infections)

Some people develop frequent and severe herpes infections that cause mouth, genital, or anal sores, or a reactivation of chickenpox known as shingles.

The symptoms of an HIV infection may resemble other medical conditions. Always consult your child's doctor for a diagnosis.

Diagnosis. Fig. 2. Time to develop antibodies: 95% people do this by week 4 and more than 99.9% by week 12



Each 'x' represents the time when a different person develops HIV antibodies. Testing is only useful when the majority of infections would be detected. Testing after 2 or 3 weeks is not very useful.

Fig. 3. Timeline for HIV infection, immune responses and window period for tests

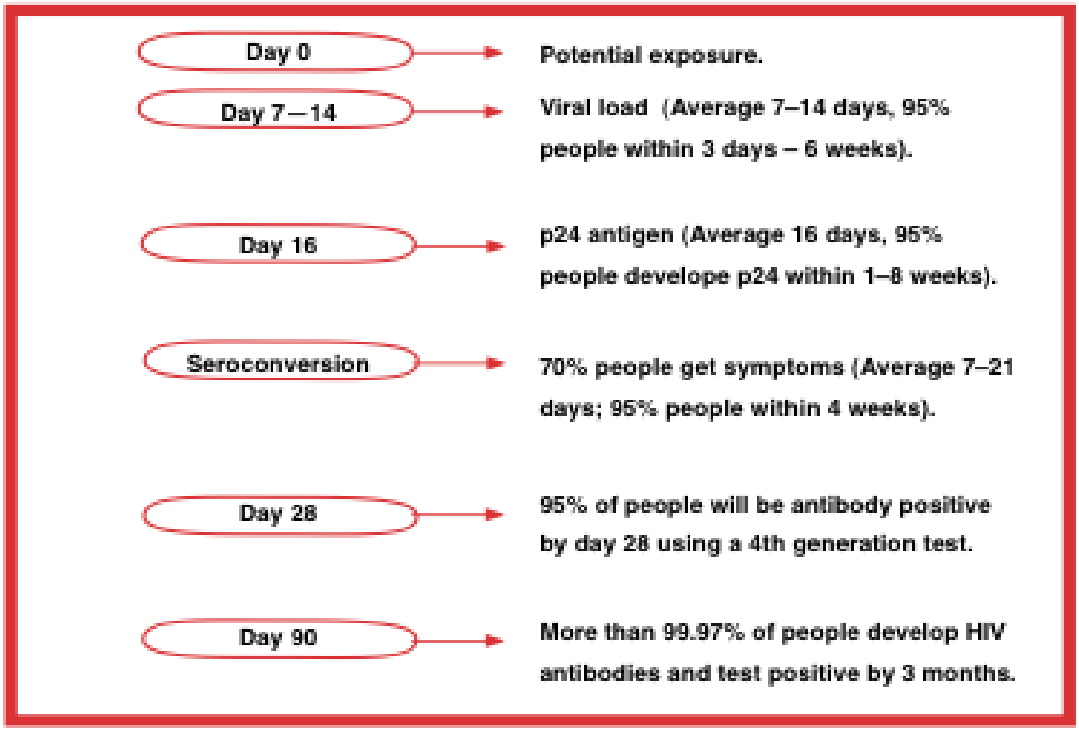


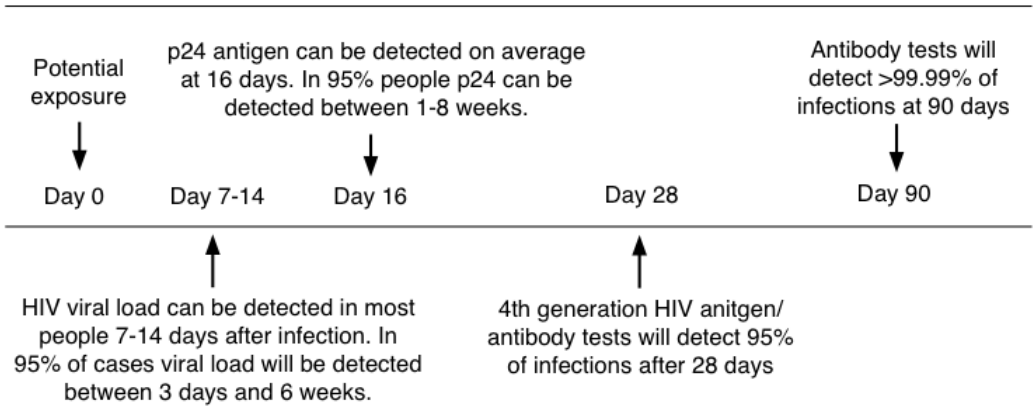
Figure 2 shows the range of times that people can take to respond to HIV infection.

- The earliest marker is HIV viral load. This is in the first weeks after infection (usually from 1 to 6 weeks after exposure). A high viral load is related to seroconversion symptoms.
- The first HIV protein (antigen) that can be measured is p24 (from 1 to 8 weeks after exposure).
- Viral load and p24 tests are not accurate for diagnosing early HIV if the results are negative.
- An HIV antibody response can be detected as early as two weeks in a few people and in more than 99.9% of people by 12 weeks. An antibody test at 4 weeks will detect 95% of infections.
- Antibody testing at 4 weeks can give you a good indication of your HIV status, but you need a test at 12 weeks after the exposure to be considered HIV negative.

Fig. 4. Different types of HIV test

Type of test	What the test look for?		
	RNA/ DNA *	Anti-gen	Anti-body
PCR/viral load	●		
p24 only test (Ag)		●	
4th generation antigen/antibody (Ag/Ab) tests (p24+ ELISA, ELI, MEIA/ ELFA/ECLIA): includes Architect, Duo, Combo/Combi etc		●	●
1st/2nd/3rd generation antigen only tests (ELISA, ELI, MEIA/ELFA/ ECLIA): includes TriDot etc			●
Rapid tests: finger prick and oral swab test are antibody only: includes OraQuick.			●
Western blot tests look for antibodies to specific HIV proteins. They confirm a positive HIV antibody test result.			●
* Viral genetic material			

Fig. 5. Average time after exposure to detect HIV antigens and antibodies



The pool of human lymphocytes possesses specific glycoproteins of their surface that play an important role in the cells activity and function CD-4 positive lymphocytes are the primary target of HIV infection, and the CD-4 receptor is the primary binding site of HIV. Throughout the course of chronic HIV infection the number of CD-4 lymphocytes is depleted and the loss of these cells is associated with development of the characteristic opportunistic infection and malignancies of AIDS. Thus the measurement of CD-4 positive lymphocytes is one of the most impotent determinates for clinically staging the disease status of HIV infected patients. In uninfected controls normal values for the CD-4/CD-8 ratio are 2.0 to 1.0. Normal values for CD-4 counts are generally 500 to 1,000 cells/mL[3] in adults.

Differential diagnosis. The differential diagnosis of the acute retroviral syndrome includes a number of other illnesses: infectious mononucleosis; other viral infections such as influenza, measles, rubella, and

herpes simplex; and secondary syphilis. Evaluation of patients presenting with an illness consistent with acute retroviral infection should include a careful history to elicit risks for HIV infection, laboratory tests to rule out mononucleosis and syphilis, HIV antibody and antigen tests, and complete blood counts and differential. Sequential HIV antibody tests may need to be performed over several months to confirm the diagnosis.

The differential diagnosis of persistent generalized lymphadenopathy (PGL) includes HIV infection and a wide variety of other processes that are associated with generalized lymphadenopathy: sarcoid, secondary syphilis, and Hodgkin's disease, for example. In patients with HIV infection, lymphadenopathy may also be caused by mycobacterial infections, KS, and lymphoma. In patients with clinical findings suggesting opportunistic disease, needle aspiration of lymph nodes may help establish a specific diagnosis. Examination of aspirates with cytologic, acid-fast, and Gram stains is valuable in identifying infection or malignancy. If a specific diagnosis is not determined after staining and culture of node aspirates, then lymph node biopsy is indicated. Aspiration of lymph nodes in patients with PGL usually reveals benign cells. Biopsy specimens show follicular hyperplasia, with the normal architecture distorted by greatly expanded germinal centers composed of B lymphocytes. It is now known that active viral replication is occurring in these follicular cells and dendritic cells, although the patient may appear well clinically.Most patients with PGL require no invasive evaluation and can be managed expectantly for the occurrence of other AIDS-related manifestations.

A limited differential diagnosis of isolated thrombocytopenia in an HIV-infected person includes drug-induced thrombocytopenia, particularly in heroin addicts and alcoholics, consumptive thrombocytopenia, or splenic sequestration. Some patients with thrombocytopenia may also present with leukopenia or anemia. The presence of constitutional symptoms and pancytopenia suggests an opportunistic infection, particularly disseminated mycobacterial or fungal infection, or a lymphoma.

Treatment. Basic therapy consists of indication of antiviral agents. There are used preparations, that inhibit the return transcriptasa of the virus: azydotymidin (AzT), didanosin (ddi), zalcytobyn (ddc), stavodin (d4T), lamivudin, abacavir (ABC), nevirapapin (NVP).

Till now monotherapy AZT (retrovir, zidovudin) was used. The preparations are prescribed 0.2 gm 3 times per day constantly or courses, duration is not less than 3 months. Treatment will be carried out under the control of the general blood analysis with 2 times per one month during the first 2 months and subsequently once per month. In a stage of preAIDS (secondary diseases) AZT is necessary to indicate till disappearance of a clinical symptomatology. If the clinical picture is not better zidovudin is indicated only for that patient in which blood concentration are less than 500 cells in 1 mkL. With such treatment it is possible to prolong patients life, the number of resistant viruses to a preparation however is marked. So, monotherapy AZT is recommended only for prophylaxis of infection of fetus from mother.

Among new means with other mechanism of action a specific inhibitor of proteases krixivan is used, which is effective concerning resistant to AZT populations of a virus 0.8 gm every 8 hour. Preparations of a choice may be rotonavir, nelfinavir, sacvinar-SGC, amprenavir.

Recently it is proved, that efficiency of treatment essentially can be increased using a combination of two or three antiviral preparations. Therefore monotherapy was changed for polytherapy. The most frequently combination of two inhibitors of virus return transcriptasa (stavudin + didanosin, stavudin + lamivudin, zidovudin+didanosin, zidovudin + lamivudin, zidovudin+abacavir) and one inhibitor of a protease is used. At patients with high risk of disease progress (viraemia over 1 million copies/mL), and also in urgent cases the two inhibitors of proteases and 1-2 inhibitors of virus return transcriptasa are used.

Efficiency of specific treatment is controlled by monitoring with following criteria: 1) level HIV RNA in plasma; 2) quantity of T-lymphocytes CD4; 3) a clinical condition of the patient; 4) morphology and biochemistry of a blood (for detection of undersirable effects of an organism). Level HIV RNA in plasma is researched after 4-8 and 12-16 weeks from the beginning of treatment and subsequently each 3-4 months. The major condition of successful antiretrovirus therapy is its usage during all life of the patient, however it is interfered by a high toxicity of preparations and the complications connected with them. Complete treatment of patients with AIDS remains an unsolved problem. Last combination is considered the most effective, but also it does not cure patients with AIDS.

It is not less important preventive treatment of secondary diseases at AIDS. Against pneumocystic pneumonias the basic agent is bactrim. For initial prophylaxis of this disease bactrim is indicated 1 tablet during 3 days each week. At occurrence of pneumonia daily reception of preparation is prescribed. In case of an intolerance of bactrim it is possible to indicate dapsone or primachin in a combination with clindamicin. At presence of herpetic infections indicate acyclovir.

Against criptococus and other funguses amphotericinum is used, against bacteria - the appropriate antibiotic. At Kaposi's sarcoma freezing of eruption elements by liquid nitrogen, irradiation, chemotherapy is indicated. The immunotherapy of AIDS is at developing stage.

Prophylaxis. The most effective method for preventing mother-to-child transmission (PMTCT) of HIV is by initiating HIV-positive pregnant women on antiretroviral therapy (ART) as early as possible. ART decreases viral levels in the mother’s bloodstream, thus reducing the risk that she will transmit the infection to her infant. ART should also be administered to a child before and after birth; treatment will help a baby’s body resist infection.

Globally, an estimated 82% of pregnant or breastfeeding women living with HIV were receiving antiretroviral medicines to prevent transmission of HIV to their children in 2018.

Planning of the lesson

Table 5

n/n	The main stages of a lesson, their contents	The methods of control	Methodical equipment	Time in % from total time of a lesson
1	**2**	**3**	**4**	**5**
				10-20 %
1	Organization stage			
2	Purposes of a lesson		Relevance of the Theme. Tutorial goals of a lesson	1-3min
3	Basic knowledge and skills control	Control questions	The list of control questions	10-15 min
	1.Etiology, epidemiology, classification of disease	Test-control (first grade)	Tests of the first level	
	2. Manifestations in connection with pathogenesis	Methods of the second grade: Individual questioning in oral and writing form. Standard task's solution. Second grade test-control	Questions Clinical cases (tests of the second grade) Theory tasks for writing answers. Second grade tests	
	3. Treatment	Methods of the third grade: 1. Solution of complicated tasks. 2.Third grade test-control	Third grade questions and tasks Third grade tests	
	4. Prevention			

Continuing **Table 5**

1	2	3	4	5
				70-80 %
1	Professional skills formation	Patients with studied disease and similar diseases, patient's histories, medical cases.		
	To master the skills of: a) Diagnosis b)Laboratory confirmation c) Treatment	Laboratory data of the patients, antibacterial drugs and drugs for supportive care		
	Independent work with patients	Patients, patient's histories, medical cases.		
	Differential diagnosis	Drawing schemes of pathogenesis and clinical course of disease; making up a differential diagnosis table and list of prescriptions for intensive care.		
				10 %
	Teacher's control, recommendations, the task for the next lesson		10-15 min	

Tasks and assignments for self-assessment

Questions for self-control:

1. Infectious agent of AIDS, its biological properties.
2. Epidemiology of AIDS, contingents of the promoted risk of AIDS infection.
3. Mechanisms and ways of contamination.
4. Pathogenesis of AIDS.
5. Basic periods of AIDS development, their clinical symptoms.
6. Manifestation of AIDS in children.
7. Epidemiological and clinical criteria of diagnosis.
8. Laboratory diagnosis.
9. Principles of medical treatment.
10. Prophylaxis of AIDS.

Tests for self-control:

1. Criteria for classification of HIV infection by WHO, approved by the Ministry of Health of Ukraine (2006) for use in our country:
   A. clinical manifestations
   B. viral load in the blood
   C. the number of CD4 lymphocytes in the blood
   D. G. duration of the disease more than 5 years
   E. All of the above are correct except for item G.
2. HIV positive is dangerous as a source of infection:
   A. from the first day after infection
   B. a few weeks after infection and throughout life
   C. only during the clinical manifestation of the disease
   D. throughout life after a period of acute HIV infection.
   E. only in the chronic phase of the disease
3. The clinical signs of HIV infection are:
   A. fungal lesions of the tonsils
   B. causative diarrhea within 2 weeks
   C. micropoliadenopathy for 3 weeks
   D. persistent fever for 1 month or longer
   E. all answers are correct
4. The leading factors for HIV infection are:
   A. Blood transfusion and its preparations

B. Kisses
C. Sex, including homosexuality in men
D. fecal-oral in the presence of relevant HIV-associated infectious diseases
E. Transmissive, especially in Africa

5. Etiotropic treatment of HIV infection is carried out by:
A. antiretroviral monotherapy:
B. antiretroviral therapy with several drugs
C. combined antibiotic therapy with a wide range of drugs
A. D, sulmetoprim in combination with immunomodulators
D. a combination of 2 antiretroviral agents with laser blood irradiation.

6. Complaints of the patient with pneumocystis pneumonia:
A. chills with fever, weakness. Dry cough, shortness of breath
B. cough with a large amount of serous purulent sputum
C. complaints are absent in the majority of patients
D. cough in the absence of radiological changes
E. cough with bloody sputum

7. The acute stage of HIV infection is most often manifested by:
A. generalized form of candidiasis
B. mononucleoside syndrome
C. Cachexia
D. total immunodeficiency
E. diarrhea

8. AIDS-related infections include:
A. recurrent erysipelas
B. typhus
A. C.Malaria
C. disseminated histoplasmosis
D. pneumoconiosis

9. Source of the causative agent of HIV infection:
A. Rodents
B. Wild animals
C. Pets
D. Man
A. E.Human monkeys

10. HIV infection is:

A. Anthroponosis, the sensitivity of the human body to this disease depends on race
B. zoanthroponosis, 100% sensitivity of the human body to this disease
C. anthroponosis, sensitivity depends on the presence of a mutation of the gene CCR5
D. anthroponosis, sensitivity depends on the antigens of the histocompatibility complex HLA
E. zooanthroponosis, the most sensitive are black people

Test answers

1	2	3	4	5	6	7	8	9	10
E	B	D	C	B	A	B	D	D	B

TASK 1

Patient T, 10 years old was admitted to the infectious hospital with complaints of fever to 37.3 C for 2 months, a general weakness. Over the last 6 months weight loss by 7kg.

Objective status: Normal skin, palpable small peripheral lymph nodes. Pulse 84 beats / min, rhythmic, blood pressure 110/70 mm Hg Heart tones are muted, vesicular breathing is above the lungs. The abdomen is a soft, painless, hepatolyenial syndrome. Dysuric disorders were not detected. There is no meningeal syndrome. In the general analysis of blood: leukocytes - $3{,}2x10^9$/L.

What is the most likely diagnosis? Which laboratory tests for diagnosis are informative?

TASK 2

Patient K, 14 years old, was admitted to the infectious hospital with complaints of fever up to 38 C, general weakness, pain in the rectum, rare faeces 4-5 times a day with a mixture of mucus and blood, some hoarseness of voice. Over the past 6 months, weight loss of 11 kg, and diarrhea, pain in the rectum and fever lasted more than 2 months.

In the objective status, attention was paid to micropoliadenopathy, white gentle plaques in the oropharynx, hepatolyenial syndrome. In EGFD: candidiasis esophagitis.

Rectromanoscopy: on the background of minor hyperemia of the mucous membrane of the lower third of the rectum ulcers up to 0.5 cm in diameter. In the general blood test - leukopenia, lymphopenia.

What is the most likely diagnosis? Which laboratory tests for diagnosis are informative?

TASK 3

Having a 28-year-old woman suffering from HIV. There were no women's consultations at the dispensary. There was no specific therapy during pregnancy. Pregnancy and childbirth passed without complications. She gave birth to a baby weighing 3.2 kg without signs of pathology.

What is the doctor's tactics for the patient and her child? What is a child screening plan? What is the child's treatment plan?

Aids and material tools: Charts "HIV", "AIDS", "HIV/AIDS

Student's practical activities:

I. To perform the diagnosis:

1. Ask complaints, anamnesis and life history.
2. Examine the patients, find clinical features of disease
3. Make diagnose due to clinical and laboratory dates.

II Provide the treatment of HIV/AIDS and prevention of diseases that may be complicated by croup syndrome.

Students must know :

1. Etiology, epidemiology and pathogenesis of HIV/AIDS.
2. Clinical diagnostic features of HIV/AIDS.
3. Laboratory data in patient with HIV/AIDS.
4. Differential diagnosis of HIV/AIDS in children.
5. Main treatment of HIV/AIDS.
6. Prevention of HIV/AIDS.

Student should be able to

1. Separate anamnesis data, which told us about risk factors of HIV/AIDS.
2. Find diagnostic clinical criterions of HIV/AIDS during examination of patients.
3. To perform differential diagnosis among diseases which have the same clinical features.
4. To learn main tendentions of the HIV/AIDS treatment.
5. To perform prevention of HIV/AIDS.

«Prophylaxis of infectious diseases in children: specific and non-specific.»

Aim: to know how to prescribe measures in the foci of different infections (nonspecific prevention), specific prevention of children infectious diseases according to the immunization schedule.

Professional motivation: Protection from infectious disease is referred to immunity. That's why immune prophylaxis is the most potential method of infectious diseases prevention. World experience shows that potential risk of postimmunization reactions and complications is very low. And risk of infectious diseases' complications and mortality really overweight them. Ambulatory pediatrician plays the main role in immune prophylaxis program. Organization of immune prophylaxis is the main prophylactic work of the ambulatory pediatrician.

Basic level

1. Epidemiological peculiarities of "controlled" infections: mumps, measles, rubella, diphtheria, pertussis, poliomyelitis, tetanus, viral hepatitis [microbiology, children infectious diseases].
2. Immunization schedule [children infectious diseases].

Planning of the lesson **Table 1**

n/n	The main stages of a lesson, their contents	The methods of control	Methodical equipment	Time in % from total time of a lesson 25
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				10-20 %
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	4. Prevention			

Continuing

Table 1

1	2	3	4	5
				70-80 %
1	Professional skills formation	Patients with studied disease and similar diseases, patient's histories, medical cases.		
	To master the skills of: a) Diagnosis b)Laboratory confirmation c) Treatment	Laboratory data of the patients, antibacterial drugs and drugs for supportive care		
	Independent work with patients	Patients, patient's histories, medical cases.		
	Differential diagnosis	Drawing schemes of pathogenesis and clinical course of disease; making up a differential diagnosis table and list of prescriptions for intensive care.		25

Subject's integration

Table 2

Subjects	To Know	To Know How
1	**2**	**3**
Human Anatomy	The main anatomic characteristics of immune system	
Physiology	Respiratory, nervous and immune system function	To explain a variety of clinical signs and laboratory abnormalities
Pathological Physiology	Pathogenesis of disease	To explain the main symptoms and signs appearance, causes of relapses, failure of inadequate therapy
Pathological Anatomy	Pathology	To explain the pathogenesis of complications and causes of death
Microbiology	Etiology (classification, morphologic characteristic of the pathogen, methods of revealing and identification)	To culture the organism
Pharmacology	The main antiviral and antibacterial agents. Regimens of treatment. Treatment of complicated influenza. Supportive care	To write the scheme of treatment of severe influenza. To understand vaccines
Histology	Histological changes in different clinical forms of postvaccinal complications	Explanation of clinical signs appearance

Continuing **Table 3**

1	2	3
Propedeutics of Internal Diseases	History of disease. Patient's examination.	To gather information about patient's history and chief complaints, to distinguish those, most important for diagnosis of postvaccinal complications. To examine the patient, to reveal the main symptoms and signs of disease. To distinguish the set of diagnostic features of postvaccinal complications. To argue the diagnosis.
Surgery	Chest pain, cough, respiratory failure	Differential diagnosis with surgical disorders, diagnosis of complications
Internal Diseases	Chest pain, cough, respiratory insufficiency	To differentiate with other disorders
Neurology	Severe headache, vomiting, meningeal signs, delirium, altered consciousness	Differential diagnosis with encephalitis, meningitis, stroke
Clinical immunology and allergology	Immunologic changes as a part of pathogenesis and host defenses	To explain confirmative serologic tests
Epidemiology	The routs of transmission, main sources of infection	Epidemiological history

Students' independent study program.

1. Objectives for students' independent studies.

You should prepare for the practical class using the existing textbook and lectures. Special attention should be paid to the following:

The immunoprophylaxis task is management by immunological answer to prevent the disease beside separate persons and groups of the population.

The ways of the immunoprophylaxis:

Active - stimulation of own antibodies production,

Passive - introduction of ready antibodies.

Vaccinal preparations characteristic

1. Vaccines, which include complete killed microorganisms (pertussis, typhoid, cholera) or inactivated viruses (influenza, poliomyelitis Salk vaccine)
2. Anatoxins, which contains inactivated toxin of the bacteria (diphtheria, tetanus)
3. The vaccines from alive attenuated viruses (measles, mumps and others.)
4. Vaccines, which contains crossing alive microorganisms (BCG)
5. Chemical vaccines from fraction of killed microorganisms (pneumococcal, meningococcal)
6. Gene-engineering recombinant, chemical synthesized (hepatitis B, influenza)
7. Associated (in composition of which enters several vaccines)

Composition of vaccines:

1. Active or immunizing antigens
2. Fluid base
3. Preservatives, stabilizers, antibiotics
4. Auxiliary facilities

Ways of the vaccination

1. Intramuscular (DTP, DT, DT-M, antirhabic, meningococcal B)
2. Subcutaneous (measles, mumps, rubella, meningococcal A+C)
3. Intracutaneous (BCG)
4. On skin (plague, tularemia, brucellosis)
5. Peroral (poliomyelitis)
6. Intranasal (Influenza, inactivated)

Recommended immunization schedule for infants and children **Table 3**

Age	Recommended immunizations
4 months	Diphtheria, tetanus, and pertussis (DTP) Polio (OPV or IPV) Hepatitis B (HBV) Haemophilus influenzae type B (HiB) (1)
6 months	Diphtheria, tetanus, and pertussis (DTP) Hepatitis B (HBV) Haemophilus influenzae type B (HiB)
12-15 months	Haemophilus influenzae type B (HiB) Tuberculosis test (2)
12-18 months	Diphtheria, tetanus, and pertussis (DTP) Polio (OPV or IPV) Varicella zoster (chicken pox) vaccine (VZV)
15 months	Measles, mumps, and rubella (MMR) vaccine Hepatitis B (HBV)
4-6 years	Diphtheria, tetanus, and pertussis (DTP) Polio (OPV or IPV) Measles, mumps, and rubella (MMR) vaccine (3)
12-14 years	Varicella zoster (chicken pox) vaccine (VZV) (4)
14-16 years	Tetanus-diphtheria booster (5)

Ukrainian Immunization Schedule **Table 4**

Immunization	Age										
	1 day	3-5 days	2 months	4 months	6 months	12 months	18 months	6 years	14 years	16 years	Adults
Hepatitis B	+		+		+						
Tuberculosis		+									
Measles, mumps, and rubella						+		+			
Diphtheria, tetanus			+	+	+		+	+		+	Every 10 years
Pertussis			+	+	+		+				
Polio			+	+	+		+	+	+		
Hib-infection			+	+		+					

Vaccinal process - is a change of homeostasis, which appear in organism in response to introduction of vaccinal preparation and include the complex of reactions to which belongs: formation of antibodies, adaptation and postvaccinal reactions, postvaccinal complications.

Vaccinal reactions appear in response to entering the vaccines, are characterized by appearance of clinical manifestations typical to this type of vaccine, which have a round-robin duration, are short, do not cause serious changes of vital activity in the organism.

Postvaccinal complications - all pathological phenomena, which appear after vaccination and are not inherent to the usual vaccinal process, but obvious, their relationship with performed vaccination:

1. Postvaccinal unusual reactions and complications, caused strictly by vaccine ("true").
2. Joining of intercurrent infections in postvaccinal period.
3. Exacerbation of chronic diseases and primary manifestations of latent diseases.

Table 5

Postvaccinal reactions	Postvaccinal complications
DTP-vaccination	
1. Temperature 37.5-39 ºC, anxiety, poor sleeping, rarely - vomiting. 2. Local reaction (more often on revaccination) - in the manner of hyperemia, infiltration. 3. Febrile seizures (on background of the quick ascent of the temperature). 4. Reinforcement of the allergic manifestations (in children with exudative-catarrhal diathesis)	1. Kvinke's edema. 2. Anaphylactic shock, collapse. 3. "Croup" (on background of ARVI). 4. Prolonged cry more than 4-5 hours. 5. Afebrile seizures, absances. 6. Encephalitis (stratification of the disease)
OPV-vaccination	
Does not call any reactions	1. Reinforcement of the allergic manifestations (in children with exudative-catharrhal diathesis) 2. Kvinke's edema, urticaria. 3. Vaccinassocated poliomyelitis in immunised and in contact persons (on background of immunedeficiency) - 1:1,500000.
Vaccination against measles	
1. Specific vaccinal reaction from 4 to 14 days: - temperature 37,5-38 ºC, - catarrhal manifestations, conjunctivitis, - pale-rose rash in a small amount, - duration - 2-3 days, - is not contagious	1. in children with exudative-catarrhal diathesis: Kvinke's edema, urtica 2. Lymphadenopathy. 3. Hemorrhagic vasculitis. 4. Hyperthermia 39-40 ºC with febrile seizures
Vaccination against mumps	
1. In some cases from 4 to 12 days - fever, catarrhal manifestations. 2. Rare - a short increase of parotid glands.	1. Hyperthermia. 2. Febrile seizures. 3. Abdominal syndrome. 4. Allergic rash. 5. Very rare - serous meningitis.
BCG-Vaccination	
1. Local reaction: papule, vesicle, rib; lymphadenitis	1. Subcutaneous cool abscess (BCGitis). 2. Purulent lymphadenitis. 3. Kelloid scars. 4. Lymph nodes calcification. 5. Generalized BCG-infection on background of immune deficiency (4: 1000000). 6. Osteites with dominating damage of long bones.

LIST OF THE MEDICAL CONTRAINDICATIONS TO THE PREVENTIVE VACCINATIONS

Table 6

Vaccine	Contraindications
All vaccines and anatoxins	• Severe complications on previous dose in the manner of anaphylactic shock. • Allergy on any component of the vaccine. • Progressing diseases of the nervous system, hydrocephalus and hydrocephalic syndrome in stage of decompensation, epilepsy, epileptic syndrome with seizures 2 times per month and more. • Anemia with level of hemoglobin below 80 g/l (preventive vaccinations are conducted after hemoglobin level increasing).
All alive vaccines	• Congenital combined immune deficiencies, primary hypogammaglobulinemia (giving of the vaccines is not contraindicated at selective immune deficiency of Ig A and Ig M), hemoblastoses and malignant tumors, pregnancy, AIDS
BCG	• Weight of the child less than 2,000 g: prematurity of the 2nd degree (the weight is 1,500 - 1,999 g) vaccination do not performed before 1 month of life, prematurity of the 3rd degree (the weight 1,000 - 1,499 g) - vaccination do not performed before 2 months of life, complicated reactions on previous vaccination (lymphadenitis, cool abscess, ulcer of the skin more 10 mm in diameter, kelloid scar, osteomyelitis, generalized BCG-infection, tub. infection)
OPV	• Children, whom alive vaccines are contraindicated, as well as members of their families are recommended to be vaccinated by inactivated poliomyelitis vaccine (IPV)
DTP	• Seizures in anamnesis (instead of DTP enter DT or vaccine with acellular component)
living vaccine against measles, living mumps vaccine, vaccine against rubella or trivaccine (measles, mumps, rubella)	• Allergic reactions on Aminoglucosides • Anaphylactic reactions on eggs protein

Passive immunization is indicated

1. To children with insufficient antibodies syntheses as a result of congenital or acquired cellular defects of B-lymphocytes.

2. At absence of vaccines against infection, when single way of protection is introduction of ready antibodies.

3. If required immediate preventive maintenance of the disease for epidemiological causes (the contact with sick on measles, preventive maintenance of rabies, tetanus).

4. For neutralization of the antigen-toxin by specific antitoxic antibodies.

5. With medical purpose at the beginning of the diseases (at diphtheria, botulism, tetanus).

Tests and assignments for self-assessment

Choose the correct answer / statement:

1. To the child, 1 year old, was diagnosed transitory hypogammaglobulinemia. Define how he should be vaccinated against poliomyelitis.

A. It is absolute contraindication to vaccination

B. vaccination according to immunization calendar

C. vaccination according to immunization calendar by alive OPV (the oral poliomyelitis vaccine)

D. vaccination according to immunization calendar by IPV inactivated poliomyelitis vaccine)

E. vaccination after normalization of immunoglobulins' level

2. The child, 1 year old, during 2.5 months was receiving immunosuppressive therapy. His mother has addressed to immunologist with the question about vaccination of the child according to calendar. Vaccination against what disease is contraindicated to this child?

A. Tuberculosis

B. Measles

C. Mumps

D. Rubella

E. Poliomyelitis

3. The child, 7 years old, came to district pediatrician for vaccination against tuberculosis. What examination must be done before this vaccination?

A. General blood test

B. General urinalysis

C. Biochemical blood test

D. ECG

E. Mantu Test with 2 tuberculin units

4. The Child has recovered from diphtheria of the pharynx. How to immunize this child against diphtheria later?

A. Vaccination should be done through 6 months after the disease

B. Vaccination should not be done

C. Vaccination should be done by antidiphtherial serum

D. Vaccination should be done to children, which did not receive the specific treatment

E. Vaccination should be done after stimulation of the immune system

5. The girl, 4.5 months, was in contact with child who had whooping cough. It is known that the girl was immunized according to calendar. What is her further immunization against whooping cough?

A. To give her human immune globulin immediately

B. Vaccination should not be done

C. Vaccination should be done according to calendar

D. Vaccination should be done by acellular pertussis vaccine

E. Vaccination should be done on background of the chemotherapy

Answers for the self-control:

Tests: 1-D, 2-A, 3-E, 4-A, 5-B.

Task 1

Suddenly, the temperature in the refrigeration chamber, where the unlawful serum, tetanus toxoid, AKDP, polio vaccines and hepatitis B were stored, dropped to -20ºC.

Which of these drugs can eventually be used for vaccinations? 2. What is the procedure for writing off unsuitable IIBPs? 3. How should IAPB be disposed of?

Task 2

You need to take part in organizing an office for preventive vaccinations at a children's district clinic.

1. What is the amount of space required for the proper organization and optimal operation of such an office? 2. What equipment should be in the office? 3. Who is responsible for organizing the vaccinations?

Task 3

A child of 3 years of age at the time of administration of the DTP vaccine had suffocation, severe acrocyanosis, cold, sticky sweat, nausea, dizziness. Pulse filiform, blood pressure - 60/30 mm Hg 1. What is your diagnosis? 2. The emergency care algorithm.

Aids and material tools: Charts "Immunization schedule".

Student's practical activities:

I. Writing of individual Immunization schedule to healthy children.
II. Writing of individual Immunization schedule to children in case of contraindications, late immunization and other problems.
III. Prescribe epidemiological measures in the focus of infection, specific prevention of the disease.
IV. Diagnosing, treatment and prevention of postimmunization reactions and complications.

Students must know:

1. Recommended immunization schedule for infants and children.
2. Ukrainian immunization schedule.
3. DTP Vaccine: characteristics, immunization schedule, risks, contraindications.
4. MMR vaccine: characteristics, recommendations, precautions and contraindications, adverse reactions.
5. Polio vaccine: characteristics, immunization schedule, risks, contraindications.
6. Varicella zoster (chicken pox) vaccine: characteristics, immunization schedule, risks, contraindications. Varicella-zoster immune globulin.
7. Hepatitis B virus vaccine: characteristics, immunization schedule, risks, contraindications.
8. Hepatitis A virus vaccine: characteristics, indications, contraindications, side effects.
9. Influenza vaccine: characteristics, immunization schedule, risks, contraindications.
10. Normal postimmunization reactions and complications: clinical features, treatment prevention.

11. epidemiological measures in the focus of infection,
12. Specific passive prevention of diseases by immune globulin.

Student should be able to

1. Write individual Immunization schedule to healthy children.
2. Write individual Immunization schedule to children in case of contraindications, untime immunization and other problems.
3. Prescribe epidemiological measures in the focus of infection, specific prevention of the disease by immune globulin.
4. Diagnose, treat and prevent of postimmunization reactions and complications.

TORCH-infections

Actuality of theme:

Infections with a vertical transmission mechanism is one of the urgent problems of modern neonatology, pediatrics, infectology. The frequency of this pathology has increased, which is due to the increase in infection of women of childbearing age with pathogens that can cause intrauterine infections of the fetus (Pozdnyakov SV, 2001). Intrauterine infections account for 10 to 61% of neonatal mortality. Of particular interest are infections of the TORCH group. The acronym TORCH combines several diseases that are transmitted vertically: T - toxoplasma, O - others, R - rubella, C - cytomegalovirus, H - herpes simplex virus. Congenital infections can infect the fetus, cause malformations that are incompatible with life, or cause disability.

Learning objectives:

Learning objectives: to learn to make a nosological clinical diagnosis, to have an idea of the importance of infections TORCH-group in the structure of childhood infectious diseases, congenital diseases, malformations and developmental anomalies, to conduct differential diagnosis of diseases belonging to the group TORSN-infections clinically similar diseases, to assess the dynamics of the main clinical manifestations and laboratory parameters, to identify complications, to appoint examinations necessary for diagnosis.

In class, **the student must be able to:**

• follow the basic rules of work at the patient's bedside;
• collect a history of the disease with an assessment of epidemiological data;
• conduct a clinical examination of the patient and identify the main symptoms of the disease;
• make a differential diagnosis;
• make a plan for laboratory examination of the patient;
• make an individual treatment plan taking into account the etiology, pathogenesis, age of the child, severity, period of illness, the presence of complications.

III. Materials for classroom independent work.

III.1. Basic knowledge, skills and abilities needed to study the topic.

Disciplines	Know	Be able
Microbiology	Features of structure, taxonomic position, pathogens of THORCH infections.	
Anatomy, pathoanatomy, histology	Intrauterine development, organogenesis, age features, main pathomorphological changes in infectious lesions.	
Physiology, pathophysiology, immunology	Pathogenesis of inflammation.	
Propaedeutics of pediatric's diseases	Defense systems and mechanisms of immune response.	Conduct clinical laboratory and instrumental examination of the patient, evaluate the results of the examination.
Pharmacology	The main stages and methods of clinical examination of the patient, the symptoms of various organs and systems	Prescribe basic antiviral and antitoxoplasmic drugs.
Pediatrics, neonatology, pediatric surgery.	Mechanism of action of antiviral and antiparasitic drugs.	Carry out differential diagnosis.
Obstetrics and gynecology	Clinic and diagnosis of congenital diseases.	Conduct clinical laboratory and instrumental examination of the patient, evaluate the results of the examination.

Contents of the topic

TOXOPLASMOSIS

Toxoplasmosis is a widespread zoonotic parasitic disease characterized by polymorphism of clinical manifestations and diversity of course.

Etiology.

The causative agent of the infection, Toxoplasma gondii, is a sporozoa from a number of coccidia. The pathogen exists in three forms: 1) tachyzoites (trophozoites) - can infect any mammalian cells, replicate by endocytosis, forming a parasitic vacuole (pseudocyst), destroy the cells of the host, cause acute infection, very sensitive to environmental factors; 2) bradyzoites - exist in the form of true cysts, can persist in body tissues until the end of life, providing a chronic latent infection, can be a source of recurrent infection; they cause infection when eating raw or undercooked meat; 3) sporozoites (oocysts) - develop only in the cells of the intestine of members of the feline family. Once in the soil, after 2-5 days, the oocysts turn into mature sporozoites and become invasive. In warm moist soil, sporozoites remain invasive for 18 months.

Epidemiology.

The infection is ubiquitous. The level of infection of the population in different countries is from 5-10 to 80-90%. Toxoplasma is found in more than 200 species of mammals and 100 species of birds, but the main source of infection is cats. The main factor of transmission is raw or semi-raw meat with Toxoplasma cysts, less often - vegetables, water, eggs, steamed milk contaminated with oocysts. There are several ways of infection transmission: alimentary, transcutaneous, transplacental.

Pathogenesis.

Toxoplasmas have a cytopathogenic effect on cells and in the places of their penetration inflammatory granulomas, necrosis are formed, where calcium salts fall out and calcifications are formed. Tissue cysts are formed, which can persist in the body for decades and can lead to recurrence with the generalization of the process in the event of a child's immunodeficiency. In congenital toxoplasmosis or in immunosuppressed persons, the acute infectious process causes severe necrotic lesions of the brain, lungs, heart. After the disease, an immunocompetent person develops non-sterile immunity.

Pathomorphology.

Histological changes in the lymph nodes are characterized by reactive follicular hyperplasia, accumulation of epithelioid histiocytes, local overflow and distension of the sinuses by monocytes. In the central nervous system there may be local or diffuse meningoencephalitis with focal necrosis, perivascular mononuclear inflammation. Necrotized areas can later calcify, causing strictures of the sylvian aqueduct, Moro's hole, forming a hydrocephalus syndrome, episyndrome. When the eye is affected, chorioretinitis is formed, which can become recurrent, leading to blindness. In the myocardium, skeletal muscles may be tissue cysts, aggregates of microorganisms, mononuclear infiltrates. Toxoplasmosis hepatitis with hepatomegaly is common. Glomerulonephritis with deposits of toxoplasmic complexes (antigen-antibody) may develop in the kidneys.

Clinical signs

The infection can be transmitted to the fetus transplacentally from a first (acutely) infected immunocompetent mother. Toxoplasmosis can be transmitted from a mother with a compromised immune system both in acute infection and in the reactivation of chronic latent infection. If the mother's infection occurs in the first trimester of pregnancy, the risk of infecting the fetus is on average 25-27%. Such a pregnancy can end in miscarriage, stillbirth or the birth of a child with severe damage to the CNS, eyes (cataracts, coloboma), less often damage to other organs, leading to profound disability or death in the first year of life. The frequency of fetal infection in the mother's disease in the second trimester of pregnancy reaches 52-54%. An infected child is born with the classic manifestations of toxoplasmosis: hydrocephalus with convulsive syndrome, calcifications in the brain tissue and inflammation of the eyes. If a pregnant woman has acute toxoplasmosis in the third trimester, the probability of infection of the fetus reaches 65%. About 10% of infected children are born with clinical manifestations of acute toxoplasmosis (jaundice, hepatolienal syndrome, congenital carditis, serous meningitis, pneumonitis, prolonged fever, papular-hemorrhagic rash). Almost 90% of children infected in the 3rd trimester have no symptoms of congenital toxoplasmosis at birth, but without specific treatment in most cases in the future there is the

development of eye lesions, impaired motor and mental activity, convulsive syndrome, hearing loss.

Diagnosis.

To diagnose toxoplasmosis, parasitological diagnostic methods are used (cell culture method, bioassay method in mice) - isolation of T. gondii from biological fluids or biopsy material of body tissues; serological methods - determination of specific IgG, IgM or IgA antibodies in the blood or cerebrospinal fluid; methods for determining DNA (PCR - polymerase chain reaction).

Treatment of toxoplasmosis.

Treatment is indicated for infected newborns, regardless of whether they have clinical manifestations of the disease or not.

To date, the most effective method of treatment of toxoplasmosis infection is the appointment of a combination of pyrimethamine and sulfadiazine. These drugs act synergistically and provide a blockade of folic acid metabolism in replicating tachyzoites. Additional administration of folinic acid (leucovorin) can prevent the toxic effects of pyrimethamine on the red bone marrow. Pyrimethamine is administered to children for the first 2 days at a saturation dose of 2 mg / kg / day (maximum 50 mg / day), and then at a maintenance dose of 1 mg / kg / day (maximum 25 mg / day). Sulfadiazine saturation dose 75 mg / kg / day, maintenance dose 50 mg / kg every 12 hours. Leucovorin is prescribed at a dose of 5-20 mg 5 times a week for the duration of treatment and another 1 week after taking pyrimethamine. Other etiotropic agents used to treat toxoplasmosis, including in pregnant women and infants, are spiramycin, clindamycin, and azithromycin.

Prevention.

The basis of prevention of toxoplasmosis are measures aimed at limiting the spread of the pathogen in the environment (control of the wild cat population, exclusion from the diet of domestic cats of raw meat and offal) and prevention of individual infection. Serological screening of pregnant women.

RUBELLA

Rubella is an acute viral infection that can take the form of an acquired or congenital process with different transmission mechanisms and different consequences.

Etiology.

The causative agent of rubella belongs to the family Togoviridae, and is the only member of the genus Rubivirus. The virus contains RNA. Viral particles have a spherical shape with a diameter of 65-70 nm. The rubella virus is unstable in the environment, dies at room temperature within a few hours, but is well preserved when frozen.

Epidemiology.

Rubella is an anthroponotic infection. The source of infection is a patient with acquired, congenital rubella or viral infection. In children with congenital rubella virus excretion with sputum, feces, urine can continue for 1.5-2 years. Transmission routes: airborne, vertical. Susceptibility to rubella is high. The contagiousness index is 70% -90%. Innate immunity is absent in children whose mothers are susceptible to rubella. The epidemic process is characterized by winter-spring rises in incidence. Immunity after rubella - stable, long.

Pathogenesis.

In congenital rubella, the virus enters the fetus through the mother's bloodstream. The frequency of infection of a pregnant woman in contact with a rubella patient depends on her immunity to this infection. In the population, the incidence of rubella in pregnant women reaches 15%. In half of infected pregnant women the disease is subclinical. The virus infects the epithelium of the chorionic villi and the endothelium of the blood vessels of the placenta, from where it enters the bloodstream of the fetus and its cells in the form of emboli. The development of the pathological process is of the type of chronic infection. The level of embryonic infection reaches 80% -90% in pregnant women in the first trimester, 25% -35% in the second trimester and 8% -10% in the third trimester. With rubella infection after 20 weeks of gestation, the risk of malformations is significantly reduced, but infection during this period can lead to the development of chronic disease with damage to the nervous system and sensory organs. Possible mechanisms of congenital anomalies may be a direct destructive effect of the virus on embryonic cells, fetal ischemia as a result of placental vascular damage. The most affected are those organs that are in the process of formation. Such a critical period for the brain is the 3-11th week of pregnancy, for the heart - 4-7th, for the ear - 7-12th, for the eye - 4-7th, for the palate - 10-12th. Heart defects, cataracts, glaucoma develop in the mother's disease in the first 8 weeks of pregnancy, hearing damage - in the 12th week of pregnancy. After the birth of a child, the persistence of the virus remains in the body. This creates conditions for the development of immunopathological processes, the result of which is the development of a child with congenital rubella, deafness, retinopathy, thyroiditis, diabetes, encephalitis.

Clinical signs.

Norbert Greg first described the classic triad of congenital rubella: cataracts, heart disease, deafness. In addition to the classical triad of congenital rubella, there is an extended syndrome of congenital rubella, in which in addition to these syndromes there are microcephaly, glaucoma, cleft palate, interstitial pneumonia, hepatitis, myocarditis, meningoencephalitis, lesions of the vestibular apparatus, anemia, vadicoma .

Among the heart defects most often (78%) there is non-overgrowth of the botal duct, there are also defects of the aortic valve, aortic stenosis, aortic coarctation, ventricular septal defect and pulmonary artery stenosis. Defects of the "blue type" in rubella are almost non-existent. Cataract is the result of the direct cytopathogenic action of the rubella virus, which can persist in the lens of the eye for several years. Cataracts can be unilateral or bilateral and are often combined with microphthalmia. This abnormality may be absent at birth and develop later in the neonatal period. Glaucoma is much less common than cataracts. Retinopathy is often found among visual defects, which is characterized by areas of dark pigmentation and depigmentation located on the retina. Changes in the eyes can occur several years after birth. The most common defect in congenital rubella is deafness. It can be light or strong, one-sided or two-sided. Deafness is often accompanied by vestibular dysfunction, the severity of which correlates with the degree of deafness.

Nervous system disorders in congenital rubella are manifested by loss of consciousness, drowsiness, irritability, convulsions, decreased muscle tone, paralysis. In the future there are

varying degrees of movement disorders, hyperkinesis, convulsions. Neurological symptoms are joined by mental retardation.

Thrombocytopenic purpura occurs immediately after birth and is most pronounced in the first week after birth. Hemorrhagic rash on the skin may persist for 2-3 months.

Typical manifestations of congenital rubella include hepatitis, hepatosplenomegaly, hemolytic anemia with characteristic reticulocytosis and deformed erythrocytes, serous meningitis, interstitial pneumonia, tubular bone lesions. There are also more rare lesions: malformations of the genitourinary system (cryptorchidism, hypospadias, hydrocele, bicornuate uterus, dicotyledonous kidneys), malformations of the gastrointestinal tract (pylorostenosis, atresia of the bile ducts), various skin manifestations: dermatitis, pigmented pigments. Children with congenital rubella often have low body weight, short body length, significantly lagging behind in physical development. About 16% of these children die within the first 4 years of life. The cause of death is heart disease, sepsis, damage to internal organs.

Diagnosis.

The diagnosis of congenital rubella is made in the case of isolation of the virus from various substrates of the patient (blood, urine, nasopharyngeal mucus, cerebrospinal fluid, bone marrow), detection of IgM antibodies or a stable positive result in RPHA and the presence of clinical signs of congenital rubella. In the presence of two of the main symptoms (cataract, glaucoma, heart disease, deafness, retinopathy of pigmentation) or one main and one additional symptom (purpura, splenomegaly, jaundice, meningoencephalitis, bone lesions, microcephaly), the disease is not contradictory to the diagnosis of congenital lack of laboratory confirmation.

Treatment.

Specific therapy for rubella has not been developed.

Prevention.

Specific prevention of rubella is carried out with live rubella vaccine at the age of 12-15 months with subsequent revaccination at 6 years and 15 years of girls. Vaccination can be given with a monovaccine or a combination vaccine, which includes vaccines against rubella, measles and mumps. Immunity is formed in 95% of vaccinated.

CYTOMEGALOVIRUS INFECTION

Cytomegalovirus infection (cytomegalovirus) is an infectious disease caused by herpesvirus type 5 and characterized by various clinical forms and course - from asymptomatic to severe generalized form with damage to many organs, from acute to chronic.

Etiology.

The causative agent of cytomegolovirus infection (CMVI) is cytomegalovirus hominis (CMV), which belongs to the family of herpesviruses, a subfamily of □-herpesviruses. The virus contains DNA with a diameter of 120-200 nm. The causative agent of cytomegalovirus is thermolabile: inactivated at a temperature of 56oC, stored for a long time at room temperature, quickly loses its infectious properties during freezing (-20oC). Sensitive to disinfectant solutions. The virus has 3 serotypes.

Epidemiology.

CMVI is one of the most common human diseases. Factors of CMV transmission can be almost all biological substrates and secretions that contain the virus: blood, saliva, urine, cerebrospinal fluid, vaginal secretions, semen, amniotic fluid, breast milk. There are various ways of CMV transmission: airborne, sexual, vertical and parenteral. The main source of infection in children is the mother carriers of CMV. CMVI is the most common transplacental infection. The frequency of intrauterine CMV infection ranges from 1% to 3%. Transplacental infection of the fetus can be both in primary infection of the mother and in the reactivation of chronic infection. Intrauterine infection of the fetus with cytomegalovirus in women with primary infection reaches 30% -50%. In secondary infection (reactivation of latent-persistent infection or infection with a new strain of CMV in seropositive women), the risk of fetal infection and the development of severe CMVI does not exceed 2%.

Pathogenesis.

The entrance gates for CMV in the ante-, intra- and postnatal periods can be the outer coverings of the fetus, the epithelium of the respiratory tract, digestive tract and genitals. At antenatal infection of a fruit infection occurs mainly transplacentally. In intranatal infection, CMV enters the body by aspiration of infected amniotic fluid or secretions of the mother's birth canal. As a result of viremia, CMV is disseminated into the tissues of various organs. CMV replication occurs in these organs, which is accompanied by a cytopathic effect with the formation of cytomegalovirus cells. Epithelial, muscle, and nerve connective tissue are more commonly affected. The inflammatory process ends with the formation of interstitial and cystic fibrosis, multiple calcifications. Under the influence of immune protection factors CMV goes into a state of persistence. In the future, with a decrease in the activity of specific immunity, reactivation of the pathogen is possible**.**

Clinic.

Depending on the gestational age, when CMV infection occurred, there is a different nature of the lesions (blastopathy, embryopathy and fetopathy). At infection on the 1-14th day of gestation blastopathies develop: death of a germ, miscarriage or formation of the system pathology similar to genetic diseases. When infected on the 15th-75th day of gestation, embryopathies develop: malformations at the organ or cellular level (true defects) or miscarriages. Among the defects of development: from the CNS are more common microcephaly, microgyria, hydrocephalus, violations of the architecture of the brain; from the cardiovascular system - a defect of the interventricular and atrial septum, endocardial fibroelastosis, malformations of the aortic valves, pulmonary trunk. Malformations of the gastrointestinal tract, kidneys, lungs, lower extremities and other organs may occur. At infection on the 76-180th day of gestation early fetopathies develop: inflammatory reactions with an alternative and exudative component and an exit in fibro-sclerotic deformation of bodies (false defects). Abortion is possible. At infection from the 181st day and before childbirth various fetopathies develop: manifest inflammatory reactions with defeat of various bodies and systems (hepatitis, encephalitis, thrombocytopenia, pneumonia, etc.). The most typical symptom complexes of the clinical picture of congenital CMVI are: thrombocytopenic purpura, jaundice, hepatosplenomegaly, microcephaly, malnutrition, prematurity, hepatitis, encephalitis, chorioretinitis, interstitial pneumonia, nephritis. In

intranatal infection, the nature of the disease is mainly determined by the characteristics of the premorbid condition of the newborn (maturity, maturity, comorbidities, etc.). In premature, debilitated children with a burdensome perinatal history, the clinical manifestation of CMVI is possible as early as 3-5 weeks of life.

In 90-97% of congenital CMVI occurs in subclinical form, but later in 30% -90% of them in the next 2-7 years develop various hearing, vision, dental defects, mental retardation, behavioral disorders, poor school performance, episyndrome. **Diagnosis.**

If congenital CMVI is suspected, the pathogen is identified in various biological environments of the body (blood, cerebrospinal fluid, urine, saliva, genital swabs, sputum) by PCR; examine umbilical cord blood and newborn blood for the level of specific Ig M and Ig G.

Treatment.

The question of the etiotropic therapy of CMVI is debatable due to the toxicity of antiviral drugs. For etiotropic therapy in congenital CMVI, ganciclovir is used at a dose of 12 mg / kg body weight for 6 weeks. In CMVI, which is caused by ganciclovir-resistant virus isolates, it is possible to use foscarnet at a dose of 60 mg / kg body weight per day. The use of specific immunoglobulins is recommended.

Prevention.

Non-specific measures include serological screening of women of childbearing age, compliance with hygiene rules. A promising area is the use of CMV vaccine.

HERPETIC INFECTION

Herpes infection is a group of diseases caused by the herpes simplex virus and characterized by predominant lesions of the skin, mucous membranes and nervous system.

Etiology.

HSV viruses belong to the Herpesvirus family, a subfamily of □-herpesviruses. According to modern notions, the herpes simplex virus has a complex structure consisting of a capsid, an outer lipoprotein shell, a protein around which is linear DNA. The size of the viral particles is 120-200 nm. The virus is sensitive to high temperatures, the action of alcohols and other solvents, resistant to low temperatures, to the action of ultrasound, can be stored for years in a dried state. According to antigenic properties, HSV is divided into HSV-1 and HSV-2.

Epidemiology.

Infection of the population of the Earth HSV-1 is 90% -97%, HSV-2 - about 40%. The source of HSV infection is people with various forms of the disease and virus carriers. HSV-1 is transmitted by airborne and contact. HSV-2 is sexually or vertically transmitted. The latter route can be performed during childbirth (contact with the mother's birth canal), transplacental or ascending through the cervical canal into the uterine cavity. HSV-2 usually causes genital and neonatal herpes. **Pathogenesis.**

The primary reproduction of the virus occurs at the entrance gate, causing the destruction of epithelial cells. The virus then enters the sensory ganglia, where it undergoes major replication and subsequent latency. When the macroorganism cannot limit the replication of the virus, the spread of infection by neurogenic, hematogenous and lymphogenic routes is

observed. In generalized forms of infection in the internal organs develop foci of coagulation necrosis.

Clinical signs.

When the fetus is infected in the first months of pregnancy, probably due to the general toxic effects of the virus on the fetus, miscarriages are observed. At the same time in a placenta there are necrosis with characteristic eosinophilic inclusions. Activation of HSV in the mother after 32 weeks of pregnancy leads to infection of the fetus in 10% of cases, and on the eve of childbirth - in 40-60%. There may be a premature birth or the baby will get sick in the first hours after birth. The incidence of miscarriage in genital herpes in the mother is 55%, which occurs in early pregnancy and 25% - in late pregnancy. Infection of the fetus during childbirth in women with genital herpes occurs in 50% of cases. If the infection of the fetus occurred during the birth of an infected mother, the disease in the child develops after an incubation period of 2 to 12 days, often 4-7 days after birth.

The localized form is characterized by lesions of the skin and mucous membranes of the mouth and eyes. A sick child has single blisters or groups of them on the background of erythema and edema in various parts of the body. They most often appear on the 5th-14th day of the child's life, with transplacental infection the child is already born with elements of the rash. Over time, erosion is formed at the site of the blisters, followed by pigmentation of the skin in its place. Eye damage occurs in the form of keratoconjunctivitis, uveitis, chorioretinitis, retinal dysplasia. Encephalitis occurs more often at 2-3 weeks after birth. Initially, there is fever, behavioral disorders, tremor, vomiting, loss of appetite, then depression of consciousness, convulsions. The disease in 50% of children is fatal within 6-7 days, and in others it has severe residual effects.

The generalized form is observed in 20% -50% of cases of herpes infection in newborns. The disease develops 5-10 days after birth. The disease in its clinical picture resembles neonatal sepsis. Mortality in generalized forms of GI in newborns can reach 80% -90%.

Diagnosis.

If congenital herpes is suspected, the pathogen is identified in various biological environments of the body (blood, cerebrospinal fluid, vesicle contents, nasopharyngeal washes, conjunctiva of the eyes, genitals, biopsy) by PCR, immunofluorescence reaction; examine umbilical cord blood and newborn blood for the level of specific Ig M and Ig G.

Treatment.

For etiotropic therapy of herpes infection in newborns, acyclovir 10-15 mg / kg body weight is used every 8 hours parenterally. Immunobiological drugs (specific immunoglobulin) are also used in the treatment of generalized forms.

Prevention.

Non-specific measures include prevention of contact with patients with active forms of herpesvirus infection. Pregnant women with confirmed genital herpes, cesarean section is recommended. In some countries, acyclovir chemoprophylaxis is recommended for pregnant women with genital herpes before delivery. A HSV vaccine is being developed.

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