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CHILDRENS
EXANTHEMATOUS INFECTIONS

(Training manual for students of English-language

higher education studying in the specialty "Medicine")

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LIST OF CONDITIONAL ABBREVIATIONS

VZV - varicella zoster virus
RZK - is a complement fixation reaction
RNGA - indirect hemagglutination reaction
ELISA - immunofluorescence analysis
CNS - the central nervous system
ECG - electrocardiogram
SARS - is an acute respiratory viral infection
PCR - polymerase chain reaction
ITS - is an infectious-toxic shock
CAA - viral hepatitis A
ESR - erythrocyte sedimentation rate
EA - is an early antigen
VCA - is a capsid antigen
MA - is a membrane antigen
EVNA - is a nuclear antigen
EBV - is an Epstein-Barr virus

CONTENT

1. CHICKENPOX

Chickenpox- is an infectious anthroponotic disease with an airborne transmission mechanism that occurs during primary infection with Varicella zoster virus (VZV) and characterized by the presence of maculopapular-vesicular rash on the skin and mucous membranes on the background of moderate intoxication.

Etiology

The causative agent is the VZV virus, which belongs to the family Herpesviridae, subfamily α-Herpesviridae type 3, genus Varicella viridae. The size of the virus ranges from 150 to 200 nm. The virus is DNA-containing, sensitive to environmental factors. It lasts up to 10-15 minutes outside the human body. It is adversely affected by sunlight, high temperatures, disinfectant solutions. The virus is transmitted with air currents at a distance of up to 20 meters.

The chickenpox virus has tropism to the epithelium of the skin and mucous membranes and to a lesser extent to the cells of the nervous system.

By examining the causative agent of chickenpox under an electron microscope, it was found that it is located in the nuclei of infected cells, consisting of a central body, which is surrounded by a shell. Elementary particles that are outside the cells have a double shell and resemble bricks in shape.

Using special staining methods, elementary bodies can be observed under a conventional optical microscope. When staining the contents of the bubbles with silver, you can identify many very small coccoid formations. These formations are called Aragao bodies.

Epidemiology

The source of chickenpox infection is a person with chickenpox or shingles. The entrance gate for the pathogen is the mucous membrane of the upper respiratory tract. The pathogen from the decaying enanthe elements is intensively released into the environment. The source of chickenpox infection is a person in the last two days of the incubation period and up to five days after the cessation of the rash on the skin. The mechanism of infection - mostly drip, but possible contact, rarely vertical. With the flow of air, the virus is transmitted over relatively long distances (in neighboring rooms, apartments, floors). The contagiousness index is 95-98%. You can get chickenpox at any age, but the maximum number of patients is found at the age of 2 to 7 years. The largest number of diseases occurs in autumn and winter. After suffering from chickenpox, there is a stable lifelong immunity.

Pathogenesis

The entrance gate of the chickenpox virus is the mucous membrane of the upper respiratory tract. When a person is infected, VZV enters the cells of the mucous membranes at the site of penetration, where the virus reproduces and accumulates. At the beginning of the incubation period, the virus replicates in regional lymph nodes and spreads with the bloodstream to the liver, spleen and other organs of the reticuloendothelial system. At the end of the incubation period, secondary viremia

occurs. Possessing a pronounced dermatotropy, VZV penetrates the epithelial cells of the skin, where it multiplies, causing characteristic changes in the affected cells and surrounding tissues.As a result of vasodilation, small spots first appear on the skin, then serous edema of the skin develops and papules form. The formation of chickenpox begins with the defeat of the epidermis-spiny-layer cells. Cells became hyperplastic, intranuclear and intraplasmic eosinophilic inclusions appear in them, that followed by balloon cell dystrophy until necrosis. As a result of cell death, cavities are formed where interstitial fluid accumulates, which leads to the formation of typical single-chamber vesicles. With the reverse development of the bubbles, the liquid is absorbed, crusts are formed, which then disappear. Usually only the superficial layers of the skin are damaged, so no scars are formed, except in severe cases of disease or layering of secondary infection.

During generalization, viruses can enter internal organs. At the same time on mucous membranes of respiratory tracts, a digestive tract and other bodies there are multiple vesicles which are quickly eroded. Hemorrhages, rounded foci of necrosis are found in the tissues of the affected organs, and around.

- multinucleated cells and eosinophilic inclusions without a pronounced inflammatory reaction. Severe liver damage with areas of necrosis and hemorrhage may develop.

The virus is also trophic to nervous tissue. Necrotic and degenerative processes develop in the spinal nerve ganglia, roots of sensory nerves, horns of the spinal cord. Electron microscopy reveals intranuclear inclusions and virus in ganglion cells and adjacent cells, which is characteristic of the virus persistence.

VZV can also affect the substance of the brain, and there are changes characteristic of encephalitis: areas of necrosis, perivascular edema, petechial hemorrhage.

During the presence of the virus in the body, cellular and humoral immunity is stimulated, virus-neutralizing and complement-binding antibodies appear; all this contributes to the release of the virus circulating in the blood and the virus-damaged cells. Viruses that have penetrated the cells of the nerve ganglia, where the infection remains in a "dormant", latent state for a long time, are inaccessible to the action of protective factors. The mechanism of latency in the infection caused by VZV is insufficiently studied.

Autoimmune reactions play an important role in the development of complications (encephalitis, leptomeningitis, myocarditis.)

Cellular immunity is essential in the recovery process from chickenpox.

Clinic

The incubation period for chickenpox lasts 11 to 21 days. Usually the disease begins acutely with fever and rash. Sometimes there are prodromal symptoms - low-grade fever, malaise, loss of appetite. Some patients develop a prodromal rash the day before the typical rash, most often scarlet fever or erythematous, less often crusty. Prodromal rash disappears without a trace within 1-2 days.

The rash first appears on the skin of the torso, limbs, then on the face, scalp with rashes for 3-7 days, in some cases up to 14 days. On the palms, soles, mucous

membranes of the mouth, upper respiratory tract, eyes, external genitalia, the rash is less common, usually in severe forms of the disease, and on the mucous membranes it can appear a day earlier than on the skin.

The process of rash does not occur simultaneously, but in a push. Therefore, on the skin next to the newly formed papules there are fresh transparent blisters and crusts. This is the so-called false polymorphism characteristic of this disease. Each subsequent rash is accompanied by a new rise in body temperature and deterioration of the general condition. Sleep is disturbed, appetite is reduced, irritability appears. The rash on the skin is accompanied by itching. Blisters often appear at the same time on the mucous membrane of the mouth, conjunctiva, less often - the larynx and genitals. Elements of the rash on the mucous membranes quickly macerate with the formation of surface erosions. At the same time moderate pain is possible. Erosions heal in 3-5 days.

The element of wind rash appears as a spot. Then a papule is formed in this place, and then a vesicle. In the typical form the vesicle is up to 0.5 cm in diameter, single-chambered, surrounded by a small rim of redness, filled with serous contents. Then an umbilical indentation appears in the center of the vesicle, the blister begins to dry up and a crust forms in its place, which disappears after a few days, leaving no scars.

Usually lymph nodes are enlarged (submandibular, cervical, axillary and inguinal), their size and number are determined by the number of rashes and their predominant location. During stomatitis, the submandibular and cervical lymph nodes respond more. The appearance of rashes on the genitals leads to an increase in inguinal lymph nodes.

General intoxication syndrome is manifested by aches in the body, general malaise, poor appetite, sleep disturbances, mental lability, headache. Nausea and vomiting may occur. The pulse corresponds to the temperature. Blood pressure tends to hypotension.

Classification

Type	Difficulty	Course
Typical forms Atypical forms: - erased (rudimentary), - pustular, - hemorrhagic, - gangrenous bullous, - generalized (visceral).	Mild Moderate Severe Indications: hyperthermia, significant rash, hemorrhagic syndrome, neurotoxicosis with convulsive syndrome or meningoencephalic reactions, croup syndrome.	Smooth (without complications) With complications: strepto- and staphylodermia, erysipelas, phlegmon, abscess, encephalitis, etc.

Typical forms

With mild severity, the patient condition remains satisfactory. In most patients there is a subfebrile temperature. The number of rash elements is small. Rash on the mucous membranes is observed in no more than 25% of patients, often 1-2 elements. The rash lasts 2-4 days.

At average severity of rash elements is much more, including on mucous membranes. The period of rashes lasts 4-6 days. The temperature rises to 39ºC, moderate intoxication.

There is a high body temperature (39.5º - 40ºC), in severe forms. There is anorexia, sleep disturbances, severe itching of the skin. At the height of the disease, neurotoxicosis with convulsive syndrome and meningoencephalic reactions is possible. Abundant rashes are on the skin and mucous membranes. The rash lasts 7-9 days, sometimes up to 14 days.

Atypical forms

Rudimentary form - occurs in children who were injected with immunoglobulin, plasma or blood during incubation. It is characterized by the appearance of roseola-papular rash with some underdeveloped, barely noticeable bubbles, body temperature is normal. The general condition of the child is not disturbed.

The bullous form is characterized by the presence in the stage of rash, along with the typical vesicles, of large (1-2 cm in diameter) flabby blisters with turbid contents. It is observed seldom, mainly at children till 2 flyings. Some authors consider it a complicated chickenpox due to the addition of bullous streptoderma.

Pustular form is characterized by the presence in the stage of rashes, along with typical vesicles filled with transparent contents - vesicles with purulent contents (pustules).

Hemorrhagic form - develops in exhausted children who suffer from hemoblastosis, hemorrhagic diathesis, sepsis or have long received glucocorticosteroids or cytostatics This form can also occur if chickenpox is layered on another infectious disease such as measles, scarlet fever, shigellosis and others. The content of blisters on day 2-3 of the rash becomes hemorrhagic. Hemorrhages appear in the skin, mucous membranes. There may be bleeding from the nose, gums, stomach, intestines. This form is difficult and often the prognosis is unfavorable.

Gangrenous form develops in exhausted, weakened individuals with poor care, which contributes to the accession of secondary infection. In this form, along with the usual rash elements on the skin, necrotic scabs are formed, the rejection of which exposes deep ulcers. Ulcers increase, gangrene often affects the deep layers of the skin to the fascia and muscles. The course of the disease is severe, mortality is high.

Generalized or visceral forms are rare, seen in infants or older children who have been taking glucocorticosteroids for a long time due to severe illness.

Chickenpox ranks first among viral diseases, the clinical course of which is greatly complicated by the use of corticosteroids before or during the disease. Clinically, this form is characterized by significant toxicosis, hyperthermia, a significant number of rash elements, a long period of rash and specific lesions of internal organs. The

section reveals multiple small foci of necrosis in all internal organs, bone marrow. This form gives the highest mortality rate.

Chickenpox clinic in newborns and young children

The incidence of chickenpox in children 1 year of age is about 25% of the total incidence. In newborns and children of the 1st year of life, the disease often begins with general infectious manifestations. From the first days there is lethargy, restlessness, loss of appetite, often vomiting, body temperature subfebrile or normal. The rash appears on day 2-5, abundant, polymorphic: papules, vesicles and even pustules, but sometimes the elements of the rash look like frozen in one stage of development. At the height of the rash, body temperature can reach significant numbers, increasing toxicosis, possible convulsions, fainting, rash may become hemorrhagic. The course of the disease is often severe. In such cases, neurotoxicosis is exacerbated, meningoencephalitis reactions appear, and visceral lesions are possible. In this age group, chickenpox is often accompanied by a secondary infection with the development of purulent foci of inflammation (pyoderma, phlegmon, abscesses, pneumonia, etc.). It should be noted that in newborns and young children, chickenpox can be mild and rudimentary, which is possible in the presence of residual immunity received from the mother, or if the child received immunoglobulin, plasma or blood transfusions shortly before infection.

Congenital chickenpox

If a woman becomes ill with chickenpox in the last days of pregnancy, congenital chickenpox is possible. It includes all cases of the disease that occurred in a newborn under the age of 11 days. The incubation period is reduced to 6-16 days, and the severity of the disease is determined by the duration of infection. When a woman is ill just before childbirth, chickenpox in a child manifests itself on day 5-10 of life, is severe and can lead to death. If a woman becomes ill 5-10 days before childbirth, the first clinical signs of the disease in the baby will appear immediately after birth. The course of chickenpox in these cases is easier, because the mother has time to produce specific antibodies that are transmitted to the fetus transplacentally. When a pregnant woman develops chickenpox in the first four months of pregnancy, the fetus and then the newborn have the syndrome of "chickenpox" - intrauterine dystrophy, hypoplasia of the extremities, cataracts and even blindness, retardation in psychomotor development.

Complications

Complications can be specific (caused by a virus) and the result of a stratified bacterial infection. Among the specific complications, the most important are encephalitis, meningoencephalitis, laryngotracheobronchitis and pneumonia.

Lesions of the nervous system in chickenpox most often occur during the rash (5-10 days of illness), and sometimes much later 18-21 days after the onset of the disease. 90% of all lesions of the nervous system are encephalitis. Encephalitis most often occurs in boys and at the age of 1 to 5 years. Most often, these encephalitis are characterized by cerebellar lesions and are manifested by cerebellar ataxia. Clinical manifestations begin with a new increase in body temperature, headache, vomiting, lethargy; meningitis are usually absent. There is a shaky gait, dizziness, speech

becomes dysatric, quiet, slow. Positive toe, knee and heel test, Romberg symptom, determine nystagmus, decreased muscle tone and tendon reflexes. Cerebrospinal fluid without pathological changes. The course of encephalitis with cerebellar lesions is favorable. Sometimes encephalitis in chickenpox can occur with the involvement of other brain structures in the inflammatory process. The symptoms of the disease will be as follows: headache, vomiting, convulsions, loss of consciousness, paresis, lesions of the cranial nerves, cerebellum. There may be delusions. Hallucinations, psychomotor agitation, cortical disorders. Mortality can reach 35%, and residual effects (paralysis, oligophrenia, recurrent seizures) are found in 12-15% of patients.

In addition to cerebellar ataxia and encephalitis, chickenpox can cause myelitis, encephalomyelitis, polyneuropathy, optic neuritis, and serous meningitis.

The development of Ray syndrome is possible, especially during aspirin treatment.

Swelling of the laryngeal mucosa can cause the development of false croup and acute respiratory failure. Damage to the mucous membranes with the formation of deep erosions and ulcers can cause bleeding, sometimes quite serious, especially if they are combined with thrombocytopenia. Hemorrhage in the adrenal glands can lead to the development of acute adrenal insufficiency.

Chickenpox can be complicated by the development of pneumonia. At the same time the patient's condition worsens, intoxication increases, the temperature rises to 39-40ºC. Worries about a sore throat and a dry painful cough with an asthmatic component. Radiologically, the picture may resemble miliary tuberculosis, which is due to the rash of a large number of vesicles on the mucous membranes of the bronchi with increased pulmonary pattern due to enlargement of the mediastinal lymph nodes.

Despite the severe manifestations, clinical recovery occurs rapidly.

Secondary infection may result in abscesses, phlegmon, erysipelas, streptoderma, stomatitis, lymphadenitis. Sometimes hematogenous spread of bacterial infection leads to sepsis, pneumonia, arthritis, osteomyelitis, nephritis.

Complications in the vast majority of cases occur in children with immunodeficiency. Chickenpox, which occurs against the background of glucocorticoids, is especially dangerous.

Diagnostic methods

General clinical diagnostic methods. In the general analysis of blood find leukopenia, lymphomonocytosis at normal ESR. Cerebrospinal fluid is examined in the presence of meningeal syndrome. It is usually serous with a moderate increase in protein content and lymphocyte count, although there may be a mixed cell composition represented by lymphocytes and neutrophils. Biochemical examination often reveals a decrease in glucose and sodium.

Specific diagnosis. For laboratory tests, blood and cerebrospinal fluid of patients are used, as well as elements of the rash (vesicle contents), separated from the nasopharynx, skin biopsies.

The research is done in a fluorescent microscope. X-ray diffraction is fast and quite specific. Paired sera are used for serological testing. An increase in antibody titer of 4 or more times over 10-14 days is considered diagnostic. Studies are performed using RZK, RNGA, ELISA, RIA.

Differential diagnosis

Chickenpox usually has to be differentiated from skin and infectious diseases that are accompanied by skin rashes.

In the past, it may have been necessary to make a differential diagnosis with smallpox in the first place. Currently, the epidemiological situation in the world is considered favorable, but the smallpox virus is still present in some laboratories around the world, so it is necessary to remember about this terrible disease. Common symptoms of chickenpox and smallpox are fever, intoxication, the presence of a rash that gradually passes from spot to crust.

Smallpox is characterized by the following symptoms:

- severe intoxication;
- rash appears on the 3rd - 4th day;
- with the onset of rash, the temperature drops to normal or subfebrile figures, rises again with the beginning of the period of suppuration;
- staged rash (first rash appears on the face and limbs, then on the torso;
- the characteristic presence of a rash on the palms and soles;
- on a certain limited are of the rash is monomorphic, is at one stage of development;
- all vesicles fester, the pustule has an umbilical indentation in the center;
- after the crusts fall off, scars are formed.

The generalized form of the disease caused by the **herpes simplex virus** can be very similar to chickenpox in the presence of intoxication, similar rashes, and multiple lesions. Its differences:

- possible prolonged septic fever;
- the most abundant rashes occur on the skin in the mouth, nose, genitals; they are arranged in groups and retain uniformity in the process of transformation of vesicles into pustules and crusting;
- rashes on the skin precede the onset of fever.

In early congenital syphilis in children:

- rashes are mostly bullous or macular in nature;
- rashes are mainly located on the palms, soles, multiple papules appear around the nose and mouth;
- often long-lasting scarring around the mouth;
- often appears "syphilitic rhinitis" with bloody discharge;
- when examining scrapings from the affected areas under a microscope in the dark field you can find treponema.

The formation of abscesses is characteristic of ***pyoderma***, its differences:

- pathogen - more often staphylococcus:
- rashes are localized mainly on exposed areas of the body, often on the face;
- phenomena intoxication is usually absent,
- the course is long.

In true eczema, the vesicular nature of the rash, followed by the formation of crusts can be the cause of diagnostic errors. The following features help to distinguish true eczema:

- the absence of epidemic nature of the disease;
- the disease, even with large lesions, occurs against a background of normal temperature;
- intoxication is practically absent;
- rash in the form of group rashes is localized mainly on exposed areas of the body (face, hands);
- large wetting erosive surfaces are quickly formed at the site of the rash, which are later covered with golden crusts, after the rejection of which scars remain;
- characterized by severe itching of the skin in the affected area.

With ***bullous dermatoses***, fever, intoxication, lesions of the mucous membranes are possible. Their main differences:

- the presence, along with small rashes, large blisters on the torso and extremities;
- rashes on the face are usually absent;
- a positive symptom of Nikolsky (exfoliation of the epidermis with sliding pressing on its surface);
- blisters are sluggish, quickly open, exposing wet ulcers and erosive areas.

Allergic dermatitis has its own distinctive features:

- severe itching of the skin in the affected area;
- rash is more often erythematous-papular;
- no pain in the course of rashes and nerve trunks;
- in the anamnesis there are indications of allergy;
- characteristic symmetry of rashes;
- eosinophilia.

Treatment

1. In mild and moderate forms of chickenpox in immunocompetent patients, therapy is aimed at preventing secondary bacterial complications. To do this, change clothes daily, bed linen, lubricate the vesicles with 1% solution of diamond green or 1-2% solution of potassium permanganate, after a meal rinse your mouth with a solution of antiseptics.

2. Acyclovir is an etiotropic treatment for chickenpox. Indications for the use of acyclovir are:

- patients with oncohematological diseases;
- recipients of organs, bone marrow;
- patients receiving corticosteroids,
- children with congenital immunodeficiencies,
- children with HIV;
- congenital chickenpox;
- chickenpox, which is complicated by lesions of the nervous system, hepatitis, thrombocytopenia, pneumonia;
- severe forms of chickenpox

In addition to acyclovir for chickenpox are effective drugs such as valaciclovir, famciclovir, ganciclovir.

Antiviral therapy is prescribed from the first day of the disease. Acyclovir is administered intravenously at 10 mg / kg body weight 3 times a day. The course lasts 7 days or 48 hours after the appearance of the last elements of the rash. Immunocompetent children older than 2 years and adolescents with severe forms of the disease acyclovir can be administered orally at a dose of 80 mg / kg per day in 5 doses.

3. In severe, generalized forms of chickenpox, especially in newborns and children of the first year of life, it is possible to use a specific varicella-zoster immunoglobulin at a dose of 0.2 ml / kg body weight.

At defeat of mucous membranes of an eye it is possible to apply in the form of drops of DNA gas, interferon, and also ointments (florenal, tebrofen). At stomatitis use anti-inflammatory and antiseptic means (furatsilin, kalanchoe juice) for rinsings.

At layering of a secondary infection use of antibiotics is justified.

Prevention

Passive immunization. For emergency prophylaxis use antiherpetic immunoglobulin in the amount of 2 ml, administered intramuscularly. Prevention of chickenpox with normal immunoglobulins without determining the level of specific antibodies is considered ineffective. It is administered no later than 72 hours.

Immunoglobulin can also be prescribed to pregnant women who became ill with chickenpox no earlier than 5 days before the childbirth, to women who became ill no later than 48 hours later after childbirth, premature infants born before 28 weeks of pregnancy and weighing up to 1000 g.

Passive immunization is indicated for children with leukemia, malignant tumors receiving immunosuppressants. It should be borne in mind the possibility of superinfection of VZV. There is no consensus on the feasibility and effectiveness of such immunoprophylaxis.

A live attenuated vaccine has been obtained in Japan. It is administered to seronegative people (women who want to have a child, medical staff of obstetrics, gynecology and intensive care units). As the experience of vaccinations has shown, vaccination has proved to be harmless and highly effective.

Mass vaccination of children in our country and in most foreign countries is not carried out. Vaccination is considered inappropriate because children generally carry chickenpox easily. But it is indicated for children with immunodeficiency.

Test questions (chickenpox)

1. Define chickenpox.
2. Etiology of chickenpox.
3. Epidemiology of chickenpox.
4. In which case can chickenpox in a newborn be considered congenital?
5. What are the symptoms of congenital chickenpox?
6. Pathogenesis of chickenpox.
7. Classification of chickenpox.

8. Clinic of a typical form of chickenpox.
9. Clinic of atypical forms of chickenpox.
10. Chickenpox clinic in newborns and young children.
11. What two groups can be divided into complications that occur in chickenpox?
12. What are the specific complications of chickenpox.
13. What are the complications of chickenpox in the case of a bacterial infection?
14. What are the methods of diagnosing chickenpox?
15. With what diseases it is necessary to carry out differential diagnosis of chickenpox?
16. What is the treatment of chickenpox?
17. What is the prevention of chickenpox?

2. ENTEROVIRUS INFECTIONS

Enterovirus infections - infectious diseases caused by numerous enteroviruses (Coxsackie and ECHO), characterized by a variety of clinical manifestations associated with intoxication, fever and lesions of the nervous and muscular systems (poliomyelitis , encephalitis, neonatal encephalomyocarditis, epidemic myalgia, pericarditis, herpangina, epidemic exanthema, fever, enterovirus diarrhea, etc.).

Etiology

According to the current classification developed by the International Committee, the enterovirus belongs to the Picornoviridae family in the nomenclature of human viruses.

Coxsackie viruses are the first place in the United States isolated) include 23 serotypes from group A and 6 serotypes from group B. Viruses ECHO (English abbreviation Enteric Cytopathogenic Human Orphan - orphan intestinal cytopathogenic human viruses), represented by 31 serotypes. These viruses, as well as enteroviruses of types 68, 69, 70 and 71 have a size of 20-30 nm, contain RNA.

General properties of enteroviruses:

- small size (25-35 nm);
- contain RNA;
- resistant to ether, 70% alcohol, 5% lysol, to freezing;
- can grow on a variety of primary and transplantable tissue cultures
- inactivated by 0.3% formaldehyde solution,

chlorine-containing solutions (at a chlorine concentration of 0.3-0.5 g / l), when heating, drying and UV irradiated.

Epidemiology

The source of infection is a sick person or a virus carrier, erased and inapparent forms are important. The most common factors in the transmission of infection are food and water contaminated with patients feces. Flies can be carriers of the disease. The main mechanism of infection transmission is fecal-oral, possibly airborne and

transplacental. Susceptibility to enterovirus infection is high. Sick children of all ages and adults, but most often - children under 3-10 years. Children under 6 months of age are almost not ill due to transplacental immunity. A severe form (encephalomyocarditis) is typical in newborns, enterovirus diarrhea develops in the first months of life, children aged 1-3 years suffer from paralytic polymyelitis-like forms, preschoolers and schoolchildren suffer from meningitis.

The greatest intensity of the disease is acquired in the summer-autumn period, when outbreaks of diseases are observed (especially in children institutions), but sporadic cases are registered throughout the year.

Later, enterovirus infection ceases to be a threat due to immunity, which develops after asymptomatic forms.

Pathogenesis

The entrance gates of infection for Coxsackie and ECHO viruses are the nasopharynx (airborne route) and the intestinal mucosa (fecal-oral transmission). Transplacental transmission of the infection is also possible, which contributes to the appearance of various defects in newborns. The development of the pathological process is associated with the tropism of viruses to some organs, their biological properties, as well as the state of cellular and humoral immunity. From the lymphatic system, where replication and adaptation take place, the virus enters the bloodstream and promotes the development of viremia. The virus has tropism to muscles (myalgia syndrome), heart (myocarditis), intestinal mucosa (gastroenteritis), nervous system (meningitis, encephalitis), lymphatic system (mesadenitis), etc. Possible damage to several organs and systems (combined forms).

Morphological changes in Coxsackie and ECHO infection have been insufficiently studied (mortality is insignificant). In the presence of encephalomyocarditis of newborns, the phenomena of encephalitis (edema and hyperemia of meninges, polynuclear infiltration of the inflammatory focus, degeneration and death of nerve cells), myocarditis (edema of intermediate tissue, infiltration, focal degeneration and necrosis) are detected.

Clinic

The incubation period in case of infection with Coxsackie virus and ECHO lasts 1-10 days. The onset of the disease is acute: body temperature rises rapidly to 39-40 ° C, there is a headache, sleep is disturbed, weakness, appetite is reduced, there is often repeated vomiting. Hyperemia of the skin of the face and neck is typical, sometimes with the appearance of maculopapular rash. There is also redness of the throat, back of the throat, conjunctiva. The rise in body temperature lasts 3-5 days. Repeated waves of its increase are often noted.

Hematological changes are unstable: ESR is accelerated, relative neutrophilia, lymphopenia.

Against the background of general infectious symptoms there are various typical signs that determine clinical forms of enterovirus infection: aseptic serous meningitis , epidemic myalgia, paralytic (poliomyelitis) form, herpetic sore throat, enterovirus fever, epidemic (enterovirus) exanthema, respiratory catarrhal form, enterovirus

diarrhea (gastroenterochemical myocardial infarction), encephalomycaritis foot-and-mouth disease syndrome), etc.

Aseptic serous meningitis

Aseptic serous meningitis is one of the most common forms of enterovirus infection, caused by Coxsackie virus A 2, 4, 9, 10, Coxsackie B 1-6, ECHO 1-11, 13-22, 24, 25, 27, 29-31 and enterovirus 71. Children aged 5-9 years are more often ill, there are both sporadic cases and outbreaks in children's institutions. The incubation period lasts 2-14 days.

Pathomorphological changes are characterized by serous inflammation of the soft membranes, ventricular ependyma of the brain with the phenomena of hydrocephalus-hypertension syndrome.

The disease begins suddenly: the body temperature rises to 39-40 ° C, intense headache, loss of appetite, nausea, repeated vomiting, abdominal pain, delirium and convulsions. The face is hyperemic, hyperemia of the mucous membrane of the oral cavity with granularity of the soft palate, tonsils, throat. From the first day of the disease there are meningeal signs: stiffness of the occipital muscles, bilateral signs of Kernig, Brudzinski. Abdominal reflexes are reduced. In young children, the umbilicus is tense, throbbing. Occasionally there is an asymmetry of the face, tendon and skin reflexes. All symptoms of CNS damage are unstable, quickly disappear in the event of a decrease in temperature.

Neutrophil-lymphocytic, from the 7th to the 8th day - lymphocytic, decreases from the 10th day. The amount of protein is normal or slightly increased, the Pandy reaction is negative, the amount of sugar and chlorides is normal or moderately reduced. From leukocytes there are no special changes, ESR is moderately accelerated.

The total duration of the febrile period is 1-10 days, normalization of cerebrospinal fluid occurs only at 3-4 weeks of illness. Sometimes there is a repeated increase in body temperature on the 1-7th day (two-wave temperature). The disease ends with recovery, but recurrences are possible (on the 15th-30th day) in the form of short-term fever, headache, vomiting.

After the disease for 2-3 months there is asthenia, symptoms of hypertension (headache, vomiting, increased tendon reflexes).

Occasionally there are forms in which meningeal symptoms are absent and changes in cerebrospinal fluid are significant (asymptomatic fluid-positive meningitis); conversely, in the presence of typical signs of meningitis, cerebrospinal fluid remains normal (meningism with hypertension). At careful inspection of patients it is possible (but not always) to reveal hyperemia of a mucous membrane of tonsils and a back wall of a pharynx with an injection of vessels, the phenomena of granulosa pharyngitis are possible.

Epidemic myalgia (pleurodynia, Bornholm disease)

The disease is caused by Coxsackie virus B 1-5.

Clinical manifestations are acute: body temperature 38-40 ° C, chills, headache, sharp pain in muscles of the chest, upper abdomen, back, limbs. The pain is spastic in

nature, occurs in attacks (up to 10-30 minutes), exacerbated by coughing, movements. Significant pain disrupts breathing, which becomes shallow, more frequent and very painful. Sometimes pain is observed in the hypochondrium and iliac regions, near the navel, which mimics appendicitis.

However, there are no symptoms of peritoneal irritation. Cutaneous hyperesthesia is atypical. During an attack of pain, young children are anxious, cry, and older children become forced. The pain is accompanied by profuse sweating. In the lungs, single dry or wet rales, intermittent hepatolienal syndrome.

The disease has a wavy course, pain attacks appear several times a day, the duration of the disease is 3-5-7-14 days. Possible recurrences in the form of short-term fever, muscle pain, sometimes clinical manifestations of other forms of enterovirus infection (serous meningitis, sore throat). In the blood leukopenia neutrophilia, accelerated ESR.

Herpetic sore throat ("vesicular pharyngitis")

Acute disease with vesicles on the soft palate, tonsils, tongue, back of the throat, associated with Coxsackie viruses A 2, 3, 4, 5, 6, 8, 10, 22, Coxsackie B 3, 4, ECHO 9, 10, 11, 16. It often combined with other forms of enterovirus diseases (serous meningitis, myalgia, etc.) The disease occurs suddenly: body temperature reaches 39-40 ° C, observed headache, vomiting, abdominal pain Typical changes in the throat: redness of the mucous membrane, on the brackets, tonsils, tongue, the back of the throat appear small (up to 1-2 mm) papules, which quickly turn into vesicles. resorbed, larger ones burst, turning into shallow gray-yellow ulcers surrounded by a red rim. The number of vesicles reaches 5-10, sometimes very much. Swalking at this time is painful, submandibular lymph nodes are enlarged, sensitive to touch, reflecting the activity of secondary infection

Blood changes are insignificant, ESR is accelerated. The duration of the disease is 1-7 days, ulcer healing occurs on the 4-7th day. The disease ends with complete recovery.

Enterovirus fever ("minor illness")

The most common form, caused by Coxsackie or ECHO viruses. It is characterized by short-term (within three days) fever, minor headache, sometimes vomiting, nausea During the examination, there is hyperemia of the throat, coated tongue, catarrhal phenomena, enlarged lymph nodes, liver and spleen, a slight crusty rash.The duration of this clinical form is 2-3 weeks, sometimes there is a wavy course of the disease. The clinical diagnosis is confirmed during the outbreak of the disease in the presence of other clinical forms.

Epidemic (enterovirus) exanthema (Boston disease)

Caused by ECHO viruses 4, 9, 2, Coxsackie A 16, 9. It is more common in older children. The incubation period is 4-5 days. The disease begins acutely with a rise in body temperature to 37.5-38 ° C, headache and muscle pain, redness of the throat, minor catarrhal phenomena. After 1-2 days, the temperature drops, the general condition improves. At the same time there is a rash on the face, chest, limbs. It is pink, erythematous, spotty-papular, the skin is unchanged. Spotted enanthema is

observed on mucous membranes. The rash disappears after 2-4 days, there is pigmentation. Some patients have rhinitis, pain when swallowing, conjunctivitis, polyadenitis, hepatolienal syndrome.

Disease lasts up to one week, pigmentation is short-lived (5-6 days), skin peeling does not occur.

Respiratory-catarrhal form ("summer flu")

Caused by Coxsackie viruses A, B, ECHO. Characterized by fever, headache, runny nose, dry cough, sometimes nausea and vomiting. Facial hyperemia, conjunctivitis , hyperemia of the throat, there may be an increase in lymph nodes, liver.The disease is characterized by mild course, without complications, lasts 1-5 days.The general condition is disturbed a little.Clinical diagnosis is difficult (helps the presence of other clinical forms in case of outbreak).

ECHO viruses mainly affect the lower respiratory tract, trachea, bronchi, so the leading symptom is cough. Of course, more, in this case there will be intoxication. Lung tissue is not saved by the virus. The course will usually be easy and short.

Enteroviral diarrhea (intestinal form)

It is more common in children aged several days to 4 years. This is one of the most common enterovirus infections. The disease is due to the fact that enteroviruses multiply in the cells of the intestinal mucosa. It begins acutely with a body temperature of 38-39 ° C, sometimes vomiting, abdominal pain and diarrhea (stools are liquid, watery, sometimes greenish in color, without blood impurities, occasionally with mucus). Occasionally there is bloating; tenesmus and gaping of the anus are absent. From the first days, digestive disorders are combined with signs of catarrhal phenomena: nasal congestion with serous discharge, redness of the mucous membranes, dry cough. The course of the disease is benign, lasting up to 2 weeks.

Encephalomyocarditis and neonatal myocarditis

Caused by Coxsackie B viruses and observed in newborns and children in the first months of life. Infection can be from the mother or patients, as well as in utero. The disease begins acutely with a temperature of 38-40 ° C, there is lethargy, drowsiness, vomiting, diarrhea, rapidly developing cardiovascular failure: cyanosis of the extremities, lips, tachycardia, shortness of breath, systolic murmur, dilation of the heart, enlarged liver. Along with these changes, there are phenomena of encephalitis with tonic or clonic convulsions, impaired consciousness and changes in the cerebrospinal fluid (cytosis up to 0.1 * 109 / l-0.3 * 109 / l, increased protein). The electrocardiogram shows a decrease in the voltage of all teeth, a negative tooth T, an exacerbation of the tooth P, the expansion of the QRS complex, the shift of the interval S-T. Within one or two days, the disease can be fatal. In addition to neonatal myocarditis, myopericarditis occurs in older children, but their course is mild: fever, vomiting, abdominal pain, chest pain, pallor, tachycardia, minor changes in the ECG.

Coxsackie infection in pregnant women can cause embryopathy (heart disease and CNS).

Paralytic (poliomyelitis) form

Paralytic form is caused by Coxsackie viruses B2, 5, A4, 9, 10, ECH02, 4, 6, 9, 11, 16, 30 and enterovirus 71. Most often children aged 4-8 years are sick. The incubation period is 2-5 days. Clinical manifestations on the first day reflect the severity of the course. In mild cases - a slight malaise, body temperature remains normal. Various neurological disorders allow them to be associated with a previous illness. Focal symptoms of the CNS are manifested in the form of sluggish monoparesis of the extremities (impaired gait, weakness in the legs and arms), weakness of the muscles of the buttocks, thighs, calves and sometimes facial muscles. Muscle tone is reduced, tendon reflexes are reduced, especially on the affected side. Cerebrospinal fluid without changes. There are lesions only of the facial nerve of the peripheral type. Violations recover quickly, there are no residual effects. In more severe cases, the disease often begins acutely with a rise in body temperature to 38-39 ° C and the appearance of a general toxic syndrome (headache, brokenness, sleep disturbances). There is muscle pain, but not as intense as in polio. Signs of meningitis and then encephalitis may appear on the first day. The pressure of cerebrospinal fluid rises to 300-600 mm of water. Art., the content of total protein is slightly increased, lymphocytic pleocytosis is detected. Virological examination reveals one of the serotypes of Coxsackie virus, ECHO or enterovirus 71. The peculiarity of the residual phenomena is not only the preservation of tendon reflexes on the affected side, but sometimes their revival.

An important clinical feature is that neurological disorders are very common combined with other signs of Coxsackie and ECHO infection -herpangina, skin rash.

Vesicular stomatitis with exanthema (foot-and-mouth disease syndrome)

Caused by Coxace viruses A5, 10, 11, 16, VZ, 7, C71. Skin rashes can be combined with damage to the oral mucosa. Younger children get sick most often. In this form of the disease may be a prodromal period of 1-2 days, which is characterized by a feeling of brokenness, sore throat, loss of appetite, may be subfebrile body temperature. Then, within a few hours, the body temperature rises to 38-39 ° C, which lasts 1-2 days. Simultaneously with the fever, rashes appear on the skin in the form of blisters up to 5-8 mm in diameter, localized on the extremities, can even be found on the palms and soles. But these blisters do not fester, they are absorbed within a week, leaving no trace.

Almost simultaneously with the rash on the skin or after 12-24 hours on the mucous membrane of the mouth there is an exanthema, which very quickly turns into blisters that burst, leaving aphthae. The rash on the oral mucosa disappears after 48-72 hours.

Epidemic hemorrhagic conjunctivitis

Caused by enterovirus-70. The disease begins acutely. Usually one eye is affected first, after 1-3 days the process captures the other eye. There is photophobia, tearing, foreign body sensation. The general condition of the patient remains satisfactory. On examination, there is swelling of the eyelids, redness and swelling of the conjunctiva, hemorrhage in the conjunctiva, often the upper eyelid, separated scanty,

mucopurulent or serous. The cornea is rarely affected. Normalization occurs in 10-14 days.

Complications

Most clinical forms of enterovirus infection have a favorable course. Complicated cases are determined by the state of background reactivation of the organism, age of the child, type and strain of the pathogen, nature of treatment and other factors.

After the transferred aseptic meningitis sometimes of various duration (till 1 year and more) disturbance of motor function in the form of indistinct coordination of movements, a muscular spasm, involuntary movements is observed.

In acute encephalitis, mainly in young children, coma, severe convulsive syndrome may develop. Damage to the vascular centers can cause varying degrees of hypertension, possibly a decrease in visual acuity.

At defeat of a muscle of heart and a pericardium sudden cardiac arrest, cardiomegaly, persistent disturbance of a warm rhythm is possible. Describe chronic adhesive pericarditis, pulmonary edema.

In newborns, enterovirus infection may be complicated by the development of acute hepatic encephalopathy with massive liver necrosis, renal failure, and massive bleeding.

There is a hypothesis that enteroviruses trigger a cascade of reactions that lead to juvenile diabetes. Due to the involvement of the genitourinary system in the pathological process there are various forms of glomerulonephritis, hemolytic-uremic syndrome, hemorrhagic cystitis, ulceration of the vaginal mucosa.

Diagnostic methods

General clinical methods

General blood test is uninformative, but sometimes, in the first days of the disease there may be a slight leukocytosis.

General urine test is usually unchanged, but in those cases when vesicles appear on the mucous membrane of the bladder or urinary tract, leukocytes and erythrocytes may appear in the urine.

In the presence of diarrheal syndrome, a large number of leukocytes may be found in the coprocytogram.

Is informative in aseptic meningitis. There is an increase in cerebrospinal fluid pressure, the number of leukocytes can be from several hundred to two or three thousand in 1 mm3.

Specific diagnosis

The diagnosis of enterovirus infection can be confirmed by virological examination. The material for the study are the patient's feces (especially in the presence of diarrheal syndrome), nasopharyngeal lavage, cerebrospinal fluid. The period of maximum virus secretion is the first week of the disease; The virus is excreted from the nasopharynx within 2-3 weeks, from the intestine to 6-8 weeks of the disease and longer.

Especially from faeces (there can be a healthy carrier), positive data of serological researches (RZK, RN) are required.But even high antibody titers are not evidence of the enterovirus etiology of the disease, only a fourfold increase in antibody titers detected in a study of paired sera (taken from the patient in the first days of the disease and 2-3 weeks from the convalescent) can be reliable evidence of enterovirus nature. A fourfold increase in titer is not always possible in patients with immunodeficiency, with severe enterovirus disease, in newborns. Enzyme-linked immunosorbent assay is used for quantitative and qualitative determination of virus antigens. Use methods of direct and indirect immunofluorescence, the method of nucleic acid hybridization (allows to determine the genetic code of viruses).

Differential diagnosis

Given the polymorphism of clinical manifestations, multiorgan lesions, differential diagnosis with other diseases and syndromes is difficult.

Aseptic serous meningitis is clinically similar to ***bacterial meningitis.*** Common: acute onset, high fever, meningeal signs. Moreover, neutrophilic cytosis may be detected in the cerebrospinal fluid on the first day, and moderate or even significant leukocytosis may be present in the blood.

The main differences of enteroviral meningitis:

- short-lived fever;
- short course;
- in many cases (uncomplicated) convalescence occurs without the use of active therapy;
- neutrophil cytosis in the cerebrospinal fluid for the second day is replaced by lymphocytic
- often occurs against the background of other manifestations of enterovirus infection.

Aseptic serous meningitis should also be differentiated from ***tuberculous meningitis***, which is characterized by:

- gradual onset;
- prolonged low-grade fever that does not correspond to severity of patient's condition;
- relative bradycardia;
- the process is constantly progressing;
- no changes in the oropharynx;
- it is usually possible to detect the primary source of tuberculosis infection (more often in lungs)
- Mycobacteria of tuberculosis are found on cerebrospinal fluid microscopy.

Enteroviral meningitis, unlike tuberculous meningitis, is not characterized by damage to the cranial nerves.

At carrying out differential diagnosis of *enterovirus encephalitis* with ***encephalitis of other etiology*** it is necessary to consider seasonality, a geographical zone, age of the patient.A very important feature of enterovirus encephalitis is the combination with other forms of enterovirus disease, most often with aseptic meningitis, rapid improvement of the patient's condition and laboratory parameters.

The main differences between ARI *caused by enteroviruses* from ***influenza and ARVI caused by other pathogens:***

- the disease is registered mainly in summer;
- mainly young children are ill;
- the disease is often combined with other manifestations of enterovirus infection (herpangina, diarrhea).

Fever with exanthema, depending on the nature of the rash, requires the exclusion of many infectious diseases in which there are rashes, which may be scarlet fever, measles, rubella, erythema. Differential diagnosis should take into account the location, timing of the rash, the dynamics of the rash, the nature of residual effects.

Enteroviral diarrhea should be differentiated from intestinal diseases of bacterial nature: typhoid fever, paratyphoid A and B, shigellosis, salmonellosis, cholera, etc. It should be remembered that enteroviral diarrhea is characterized by the absence of significant intoxication, the appearance of diarrhea on the background of catarrhal manifestations, as well as epidemiological data (patients with various syndromes of enterovirus diseases - myalgia, serous meningitis, etc.)

Vesicular stomatitis (foot-and-mouth disease syndrome) must be differentiated from ***foot-and-mouth disease***, which is characterized by:

- the presence of a prodromal period;
- fever lasting up to two weeks or more;
- changes in the oral cavity: the mucous membrane is swollen, sharply hyperemic, blisters have a cloudy content and are localized both on the anterior and posterior parts of the nasopharynx, especially on the mucous membrane of the gums, cheeks, tongue;
- blisters turn into ulcers;
- enlarged and painful submandibular lymph nodes.

Epidemiological history (contact with sick animals) facilitates the recognition of foot and mouth disease.

Treatment

The need for hospitalization is determined individually, it depends on the severity of the patient and the clinical form of the disease (taking into account the degree of infectivity of the patient), sanitary conditions at the place of residence.

At signs of meningitis, meningoencephalitis the patient is necessarily hospitalized.

Bed rest, no diet changes required. Specific therapy - not developed. Pathogenetic and symptomatic therapy is performed. Apply globulin 1-6 ml in the first days of the disease and RNAse 0.5 mg per 1 kg of body weight 6 times a day, leukocyte interferon, recombinant interferons (reaferon, viferon), interferonogens (cycloferon), possible appointment of immunoglobulins for intravenous administration (sandoglobulin, pentaglobin).

The complex of pathogenetic and symptomatic therapy is associated with clinical manifestations of the disease: antipyretics, dehydration, detoxification, analgesics. Corticosteroids (for encephalomyocarditis), thermal physiotherapy (for myalgia, parapoliomyelitis) are also prescribed.

In the presence of cerebral edema, diuretics (Lasix, Mannitol) are prescribed. Antibiotics should be prescribed at the risk of developing a mixed viral-bacterial infection. The choice of antibiotic is determined by the location and nature of secondary lesions. But it should be remembered that antibiotics do not act on enteroviruses.

In diarrhea, vomiting, correction of water-electrolyte disorders is of great importance. For this purpose, taking into account the loss of fluid and electrolytes, solutions "Acesil", "Trisil", "Rehydron" and others are introduced.

The prognosis

Is favorable in most cases; severe in paralytic form and encephalitis, unfavorable in neonatal encephalomyocarditis.

Prevention

Specific prevention has not been developed. Early diagnosis and hospitalization of patients (up to 10 days) is important. Children in the foci of infection are prescribed y-globulin, in recent years interferon (5 drops 4 times a day) for up to 10-15 days. Contacts are isolated for 14 days. Mandatory in the center of infection is the use of current disinfection, masks, adhere to the sanitary and hygienic regime, cancel meetings, UFO.

Control questions (enterovirus infections)

1. Define enterovirus infections.
2. Etiology of the disease.
3. Epidemiology of enterovirus infections.
4. Pathogenesis of the disease.
5. Clinic of aseptic serous meningitis.
6. Bornholm Fever Clinic (Epidemic Myalgia).
7. Clinic of hypertensive sore throat ("vesicular pharyngitis").
8. Clinic of enterovirus fever ("minor illness").
9. Clinic of epidemic (enterovirus) exanthema (Boston disease).
10. Clinic of respiratory catarrhal form ("summer flu").
11. Clinic of enterovirus diarrhea (intestinal form).
12. Clinic of encephalomyocarditis and neonatal myocarditis.
13. Clinic of paralytic (polymyelitis) form.
14. Clinic of vesicular stomatitis with exanthema (foot-and-mouth disease syndrome).
15. Clinic of epidemic hemorrhagic conjunctivitis.
16. Complications of enterovirus infection.
17. Diagnosis of enterovirus infection.
18. Differential diagnosis of enterovirus infection.
19. Treatment.
20. Prevention.

3. HERPES SIMPLEX

Herpes simplex is an infectious disease with a predominantly airborne transmission mechanism caused by the herpes simplex virus (HSV-1 and HSV-2) and characterized by a long latent course with periodic recurrences accompanied by vesicular rash on the skin. and mucous membranes, lesions of the CNS and internal organs.

Etiology

HSV was discovered by V. Gruter in 1912. They belong to the family Herpesviridae, subfamily α-Herpesviridae. HSV has a complex structure consisting of a capsid, an outer lipoprotein shell, a protein around which is linear DNA. In addition, the virion contains spermine, spermidine, lipids, lipoproteins, glycoproteins. The size of the viral particles is 120-200 nm. The virus is sensitive to drying and high ambient temperatures, but resistant to low temperatures, long-lasting in the dried state.

Because HSV consists of more than 20% lipids, it is easily inactivated by ether, alcohols and other organic solvents. . According to antigenic properties, HSV is divided into HSV-1 and HSV-2.

Epidemiology

The population of HSV-1 is 90-97% and HSV-2 is about 40%. Infection of a child occurs before the age of 5 years. The source of infection is patients with herpes simplex and virus carriers. HSV-1 is transmitted by airborne and contact routes and affects the skin, mucous membranes, central nervous system. Seasonality and epidemic outbreaks are not typical. HSV-2 is sexually transmitted and causes genital and neonatal herpes. The greatest susceptibility in children aged 5 months to 3 years. Children in the first months of life do not get herpes due to the presence of transplacental immunity.

HSV is a weak inducer of interferon, inactivation of viral DNA in cells does not occur, so the virus in the presence of virus-neutralizing antibodies persists in the body for lifelong, causing recurrences of the disease.

Pathogenesis

Entrance gates of infection - mucous membranes of the lips, mouth, conjunctiva, genitals. The virus multiplies in cells of ecto- and endodermal origin, causes the destruction of epithelial cells and is characterized by the appearance of a typical vesicular rash. HSV-1 is stored in trigeminal nerve cells. HSV-2 - in the nodes of the sacral nerve. In the future, the spread of the virus (neurogenic, hematogenous and lymphogenic) is associated with the state of the immune system, the patient's age, infection, HSV type, and so on. Generalized herpes develops as a result of virus dissemination in newborns, in children with immune system defects, and in the case of immunosuppressive therapy. Activation of viruses is associated with insufficient activity of macrophages, T-helper lymphocytes, cytotoxic lymphocytes, as well as a

decrease in the production of immune mediators. Herpes infection is an indicator of AIDS (due to immunosuppression).

Herpes encephalitis caused by HSV-1 occurs both in primary infection (30%) and in the reactivation of latent infection (70%). Ways of penetration of a virus into a brain - hematogenous or neural (retroaxonal). The spread of the virus in the nervous system is associated with its penetration into the cerebrospinal fluid. The primary replication of the virus occurs in the mesenchymal cells of the meninges, ventricular epindymus with subsequent damage to neurons and glia. HSV-1 affects all brain cells. With HSV-2 encephalitis, the baby is more likely to be infected through the mother's birth canal or transplacental route.After the virus enters the skin and mucous membranes, it begins to replicate and then spreads from cell to cell, and then into the blood and lymph. When transplacental transmission of the infection, the virus immediately enters the blood and from it through the blood-brain barrier into the brain. HSV-2 belongs to cytolytic viruses. Necrotic and inflammatory processes develop in infected cells. In the brain, necrosis is localized in gray and white matter, often diffuse in nature and spread to the deep layers of the brain, cerebellum.

Classification of herpes infection (according to AP, Kazantsev, 1980) Herpes infection of the skin (localized and widespread

1. Herpes infection of the mouth (stomatitis) and upper respiratory tract.
2. Genital herpes
3. Herpetic keratitis and keratoconjunctivitis (superficial, deep)
4. Herpetic encephalitis and meningoencephalitis
5. Visceral forms of herpes infection
6. Generalized neonatal herpes

Clinic

The incubation period lasts from 2 to 14 days, then there is a general intoxication syndrome with fever and other symptoms of intoxication. According to the mechanism of infection, there are acquired and congenital infection. Acquired infection can be primary and secondary (recurrent), localized and generalized. There are also latent forms of herpes infection.

Primary forms of herpes infection include neonatal herpes infection, encephalitis, gingivostomatitis, primary herpes of the skin, eye, herpetic panaritium, keratitis.

Secondary, recurrent forms include herpes of the skin and mucous membranes, ophthalmoherpes, genital herpes.

The primary infection occurs as a result of the first contact with the virus. This usually occurs at an early age (up to 5 years). In adults (16 to 25 years) who do not have antiviral immunity, primary herpes infection may be caused by HSV-2. In 80-90% of primary infected children the course of the disease is latent and only in 10-20% of cases its clinical manifestations are observed.

The most common form of primary herpes is ARI, aphthous stomatitis, can be manifested by various lesions of the skin, conjunctiva, and cornea.

The localized form of herpes is characterized by the appearance of a vesicular rash near the mouth, nose, ear, face, and other areas of the skin. Before the rash there is a

moderate itching of the skin, burning and pain, and after 1-2 days there are small vesicles with a transparent content on a hyperemic basis, which are located in groups. On day 3-4, the blisters dry up, crusts form in their place, which disappear after 5-7 days. Sometimes the blisters merge to form a large blister with a serous content, after the opening of which an erosion is formed, which gradually heals without changes in the skin. Possible recurrent rashes, often in the same place, the healing time in such cases increases to 2-4 weeks. In children with dermatoses (eczema, neurodermatitis, etc.) with the presence of erosive skin lesions, HSV can cause the development of herpetic eczema. There are other names of the disease in the literature: "vaccine-shaped pustulosis", herpetiform eczema", "varioliform pustulosis". This form is more common in young children. The onset of the disease is acute: rapid increase body temperature up to 40 ° C, the appearance of large herpetic blisters on eczematous skin (usually the face). The vesicles are single-chambered, burst quickly and crusts are formed. There is an increase and soreness of the lymph nodes. In addition to the skin, the nervous system and visceral organs are also involved in the pathological process, in connection with which a fatal outcome is possible.

Skin lesions in herpes infection in some cases may be zosteroform, hemorrhagic, hemorrhagic-necrotic or ulcerative-necrotic.

Zosteroform herpes is characterized by the localization of the rash along the nerve trunks, more often in the face, buttocks and lower extremities. This form of herpes simplex differs from Herpes Zoster in the absence of severe pain along the nerves.

In the hemorrhagic form, the blisters have a bloody content. The hemorrhagic-necrotic form is characterized by the appearance of necrosis at the site of the rash.

The ulcerative-necrotic form, as a rule, develops against the background of severe immunodeficiency of any genesis. If ulcerative-necrotic manifestations persist for more than 3 months, they are classified as AIDS-marker diseases.

Erythematous, papular, edematous are atypical forms of herpes simplex. At these forms vesicles are not formed, and there are hyperemia, small papules, there is a fabric hypostasis. Frequent recurrences of this form in the same place can cause lephantiasis (elephantiasis-like herpes).

There is an abortive form of herpes infection, which also belongs to atypical forms. The entrance gate is often the skin of the fingers and palms. Typical vesicles are absent. The disease is accompanied by itchy skin, swelling and redness. In this case, the diagnosis of "panaritium" is often made.

The defeat of mucous membranes by herpes gives aphthous recurrent gingivostomatitis (most often in children 6 months - 3 years), which is clinically manifested by fever up to 39-40 ° C, toxicosis, redness and swelling of the mucous membranes of the lips, cheeks, tongue, gums. Appearance of typical herpetic blisters in these places, which burst and small ulcers appear. Children do not eat well, are very restless, sleep is disturbed. Layering of a bacterial infection is possible.

In case of HSV lesions of the mucous membranes of the upper respiratory tract, an acute respiratory disease (ARI) occurs, which is clinically indistinguishable from such other etiology. This form of herpes infection accounts for 5-7% of all ARIs.

Eye lesions (ophthalmoherpes) can be primary or recurrent. More than 90% of cases are recurrent ophthalmoherpes. With herpetic conjunctivitis, the mucous

membrane of the eyelids, eyeballs are hyperemic. Characterized by moderate photophobia and lacrimation. Superficial lesions of the anterior segment of the eye are characterized by the development of conjunctivitis, combined lesions of the conjunctiva and eyelids - blepharoconjunctivitis, canaliculitis, keratitis. The development of recurrent corneal erosion, diffuse episcleritis is possible. Deep lesions of the anterior segment of the eye are characterized by the spread of inflammation and may be manifested by keratitis, keratoiridocyclitis. The latter form can result in corneal opacity and decreased visual acuity. Occasionally, optic neuritis develops. Newborns may develop cataracts, chorioretinitis or uveitis.

Genital herpes is more commonly diagnosed in adolescents and adults with a sexually transmitted infection. In children of younger age, genital lesions occur secondarily after the appearance of other manifestations of herpes infection. The infection occurs through infected hands, towels, linen. Clinically, genital herpes is manifested by redness and swelling of the genitals, vesicular rash on the labia majora and labia minora in girls and the skin of the penis, scrotum in boys. The blisters peel quickly, forming erosive or erosive-ulcerative surfaces. The disease is accompanied by itching, pain in the affected areas, body temperature usually rises.

In 50-75% of patients, genital herpes has a recurrent course, the nature of clinical manifestations is determined by the state of the immune system. Lymphatic vessels can be involved in pathological process. In this case, the development of lymphostasis is likely, which can lead to elephantiasis of the genitals.

The causative agent of HSV-2 is associated with cervical cancer.

HSV-2 has tropism to nervous tissue. More often it occurs as encephalitis or meningoencephalitis. Encephalitis can be caused by both HSV-1 and HSV-2. Brain damage is often isolated or may be one of the syndromes of generalized herpes infection that observed mainly in newborns and young children. It is believed that in newborns the etiological factor is mainly HSV-2, and in older children and adults NSV-1.

At the most typical course of herpetic encephalitis there are 5 periods:

1. General infectious (1-21 days) - fever, catarrh of the upper respiratory tract, possible blistering rash on the skin and mucous membranes.

2. Encephalic (1-10 days) - headache, vomiting, mentalexcitement, delirium, hallucinations, aphasia, apraxia, agnosia, pyramidal disorders.

3. Encephalitic (comatose) (1-50 days) - disorders of consciousness, convulsions, signs of coma.

4. Early convalescence (1-12 months) - retrograde and fixation amnesia, apraxia, agnosia, regression of physical development.

5. Residual effects - mental disorders, hyperkinesis, paresis of the extremities (months - years).

Acute encephalitis occurs as a result of primary infection, re-infection or on the background of activation of chronic herpes infection. The onset of encephalitis often coincides with the recurrence of herpes of the skin and mucous membranes or after contact of a child with adults with exacerbation of herpes of the skin, mucous membranes.

The presence of 70-75% of children with herpetic encephalitis characteristic cortical disorders makes it possible to suspect the disease before the development of coma and to begin etiotropic therapy in a timely manner.

In some patients, the encephalic period may be absent, the disease begins immediately with the appearance of general cerebral and focal symptoms.

The so-called pseudotumorous variant of herpetic encephalitis is possible, in which against the background of normal or subfebrile body temperature for several weeks the symptoms of CNS damage, signs of intracranial hypertension increase, there are changes in the fundus.

Cases of herpetic encephalitis with wavy and recurrent course are described. In children, the course of encephalitis is possible in the form of recurrent organic psychoses. Forms of herpetic encephalitis such as stroke, stem, encephalomyelitis and some other variants are also possible.

Mortality in herpetic encephalitis without antiviral treatment is 60-70%, in the case of treatment - 15-20%. Residual effects in the form of a gross organic defect (apalic syndrome, dementia, epileptic seizures, paresis, hypertensive, hydrocephalus, etc.) develop in 90% and 10-15% of cases, respectively.

Herpetic meningitis develops as serous, aseptic and is often combined with primary genital herpes. Herpetic meningitis has a prolonged course, with the risk of complications such as enuresis, polyradiculoneuropathy, myelitis, recurrent meningitis, timely diagnosis and early specific etiotropic treatment. Visceral forms of herpes infection are manifested by acute parenchymal hepatitis, pneumonia, nephritis. They occur more often in newborns, but can also occur in older children.

Herpetic hepatitis is more likely to be a manifestation of a primary herpes infection in newborns and children in the first months of life. It is accompanied by high fever, severe symptoms of intoxication, vomiting, enlarged liver, spleen, jaundice, hemorrhagic syndrome. It often has a prolonged course with severe cholestasis and the development of acute hepatic encephalopathy, liver failure, possibly fatal.

Herpetic pneumonia and focal nephritis are not clinically different from lung and kidney damage of other etiologies.

The course of herpes simplex in 15-20% of patients is acute, and most relapses occur. Clinical manifestations of recurrence are the same as in acute forms.

In older children with compromised immune system, there is a chronic generalized form of herpes infection, characterized by subacute course, slow increase in headache, low-grade fever, general weakness, memory loss.

Herpes in HIV-infected people develops more often as a result of activation of latent herpes infection. The disease becomes generalized. Signs of generalization are the spread of the virus on the mucous membranes with the subsequent occurrence of herpes pneumonia, the development of chorioretinitis, meningoencephalitis. Herpetic rash does not disappear, and ulcers form in its place. Herpes infection in HIV-infected people does not tend to heal spontaneously.

Congenital herpes infection

The frequency of herpes infection in newborns in different countries is 1 case per 2000-3500 births. The infection is caused mainly by HSV-2. After infection, HSV

persists in the body for life in a latent form, and the host's immune system creates barriers to the activation of herpes infection. During pregnancy, there is a transformation of immune responses, which contributes to the activation of HSV. Both direct and indirect effects of HSV on pregnancy have been proven. There are the following ways of HSV penetration into the embryo and fetus:

1) ascending;
2) hematogenous (transplacental);
3) transovarian

Clinical symptoms of congenital herpes infection depend on the time of infection, virulence of the virus and the state of the protective forces of the pregnant woman and the fetus.

Some researchers point to the possibility of developing malformations in the case of infection of the fetus in early pregnancy (microcephaly, microophthalmia, chorioretinitis, etc.). But most authors note that HSV has no teratogenic effects.

Activation of HSV in the expectant 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%. Premature births can begin or the baby becomes ill in the first hours after birth.

The incidence of miscarriage is 55% for genital herpes in the early stages and 25% in the late stages. 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 passage of the infected mother through the birth canal, the disease develops during the incubation period - from 2 to 12 days, often 4-7 days after birth.

Forms of herpes infection in newborns:

1) localized;
2) herpetic lesion of the CNS (encephalitis);
3) generalized form.

Laboratory diagnosis

Today, the most common ELISA, which can detect specific Ig M and Ig G antibodies in the patient blood. Detection of Ig M class antibodies indicates acute disease, reinfection or exacerbation of latent infection. However, this immunoglobulin appears in the serum only on day 10-14 of the disease, which reduces its diagnostic value. The presence of Ig G class antibodies in the blood indicates a chronic infection, and a fourfold increase in Ig G titer in the dynamics - the activation of herpes infection.

The HSV genome in the blood or cerebrospinal fluid is detected by PCR. In terms of sensitivity and specificity, PCR is not inferior to the method of virological examination, but significantly ahead of it in speed. Virus identification has not been used recently due to the complexity and duration of this method.

Treatment of herpes infection

Treatment of patients with herpes simplex should be carried out in stages:

I stage - treatment in the acute period of the disease or during relapse:

1. Antiviral drugs.
2. Natural antioxidants.

3. With a pronounced exudative component of inflammation - prostaglandin inhibitors (indomethacin, etc.).

4. Interferon drugs or its inducers.

Stage II - therapy in remission (early convalescence):

1. Immunomodulators.

2. Adaptogens of plant origin.

Stage III - in stable clinical and immunological remission (prevention of intercurrent diseases).

The "gold standard" in the treatment of herpes infection is acyclovir, which is prescribed periodically at a dose of 200 mg 5 times a day, 7-10 days with a subsequent transition to an anti-relapse dose - 100 - 200 mg per day for several months with exacerbation of herpes of the skin and mucous membranes. In children under 2 years, the dose of acyclovir is reduced by 2 times. You can also use ointments, gels that contain acyclovir, interferon, etc.

With repeated use of acyclovir in further treatment, it may become ineffective, then it is replaced by valaciclovir, famciclovir, inosine drugs.

With herpetic encephalitis acyclovir is prescribed at a dose of 10-15 mg / kg every 8 hours intravenously for 10-14 days, then -orally 200 mg 5 times a day for children older than 2 years for 2-3 weeks. Early discontinuation of acyclovir therapy may lead to recurrence of herpes encephalitis. According to the literature, early use of acyclovir in herpetic encephalitis reduces mortality by 20% versus 60-70% without its use and reduces the number of residual effects from 50% to 10-15%.

Effective intravenous administration of acyclovir at a dose of 10-15 mg / kg every 8 hours for 10-14 days in generalized forms of herpes infection in newborns, which reduces the number of adverse effects by 50%.

In ophthalmoherpes, idoxuridine, trifluridine should be used. Valaciclovir, famiclovir, and idoxuridine are recommended in the treatment of genital herpes, but they have been shown to be effective only if administered no later than day 3 of the disease.

In forms of herpes infection caused by HSV that is resistant to acyclovir in some cases effective treatment with foscarnet.

The use of glucocorticosteroid hormones is indicated in comatose forms of herpes encephalitis.

In the complex therapy of patients with herpes infection used immunological drugs that help normalize the cellular and humoral parts of the immune system, activate the interferon defense system.

Prevention

In the acute form of herpes infection, common and generalized forms of children should be isolated in separate wards. Newborns who have been in contact with patients with herpes infection should be examined for infection. If they suspect a herpes infection, antiviral therapy should be initiated.

Children with dermatitis, eczema, immunodeficiency, AIDS, and those receiving immunosuppressive therapy should be isolated from patients with herpes.

In children who attend preschool groups and have manifestations of skin herpes, it is necessary to cover the affected areas with clothing, bandages, etc. or isolate them from the team during the exacerbation of the process.

Manipulations on the head should be avoided in children born to mothers with suspected genital herpes.

Pregnant women who have been diagnosed with genital herpes are recommended a caesarean section, and children born to such mothers should be screened for herpes.

Ultraviolet radiation can provoke recurrences of herpes, so patients with recurrent herpes should avoid direct sunlight.

Creation of antiherpetic immunity at a chronic infection is promoted by introduction of the killed vaccine from a virus (on 0,1-0,2 ml in 2-3 days 5 times, not less than twice a year).

Anti-epidemic measures are not carried out in the center.

Control questions (herpes simplex)

1. Define the disease common herpes.
2. What is the etiology of herpes simplex?
3. What is the epidemiology of herpes simplex?
4. Pathogenesis of herpes simplex.
5. What are the different forms of herpes infection according to the mechanism of infection?
6. What can be a herpes infection?
7. What clinical forms belong to the primary forms of herpes infection?
8. When and who develops a primary herpes infection?
9. What are the clinical manifestations of primary forms of herpes infection?
10. What are the clinical forms of herpes infection according to the prevalence of the virus?
11. To which tissues is HSV-1 trophic?
12. To which tissues is HSV-2 trophic?
13. Clinical symptoms of congenital herpes infection.
14. Laboratory diagnosis of herpes simplex.
15. Treatment of herpes infection.
16. Prevention of herpes infection.

4. INFECTIOUS ERYTHEMA

Infectious erythema is a group of weakly wasted acute infectious diseases caused by the virus and manifested mainly intoxication and erythematous rash.

In children there are the following forms of erythema: infectious Tshamer, Rosenberg, sudden exantemia, nodular.

Infectious erythema Tshamer

Infectious erythema Tshamer - for the first time this disease called "local rubella" described A. tshamer in 1886. The unresolved issue of the pathogen was obviously the reason for the lack of study of this disease as infectious people and dermatologists. However, precisely dermatologists are mostly solved by its

differential Diagnostics. Patients with infectious erythema are often falling to dermatologists, because in the clinical course of the disease, skin symptoms are dominant, it can simulate pathology of the skin.

Etiology

Practically unknown. Probably an infectious erythema of Tsmmer is a viral disease. The question of its existence as an independent nosological unit was in doubt. The assumption was assumed that this is a clinical syndrome that occurs in various viral infections that it can be due to enterers, however, the scientists did not come to a single opinion. Some authors believe that the pathogen of an infectious erythema is **Parvovirus in 19 (HPV).**

Epidemiology

This disease refers to diseases with insignificant contagiousness and is observed in the form of sporadic cases or minor outbreaks in families and children institutions, but in the opinion of some scholars, can take the nature of epidemics. According to some authors, large outbreaks of infectious erythema are described in Germany and the United States (up to 600-1000 cases).

Although the source of infection is not established, but it is believed that a person is ill with an infectious erythema. Transmission of infection occurs an air-droplet pathway, although the contact path is not excluded. Patiently children aged 2 to 15 years old, adults are rare, so most authors consider an infectious erythema Tsmer as a children's disease, which is more common in the spring or early summer. The disease leaves a steady lifelong immunity.

Pathogenesis

Not learned.

Classification

Not developed. However, it is possible to classify the severity of the disease: light, medium gravity and severe form; Some authors, depending on the presence and degree of manifestations of the characteristics of symptoms, isolate the inparant (subclinical) form, which is especially found during epidemic outbreaks, but such a classification is rare.

Clinic

The incubation period lasts from 2 to 20 days, an average of 9-14 days. Usually has an insignificant course. Begins acute, without prior prodromal phenomena; Characteristic appearance of chills, body temperature in this normal or subfebrile, but sometimes it can reach 39 ° C. The symptoms of intoxication are absent or expressed slightly, but in older children can be more pronounced. The duration of the subfebrile period usually does not exceed 1 -2 days, but in some cases (especially in older children), the temperature can stay 3-7 days.

The main symptom of the disease, which is diagnosed, is a rash. The rash appears on the first day of the disease, less often to the second. First, the rash appears on the

face - mainly on the cheeks in the form of protruding elements of the Friday-Papulosis character the size of 3-5 mm, disappearing when tensioning the skin. After several hours, the spots are increased, merge, forming a general bright blush. On the back of the nose - a clear red membrane that in general on the face has the appearance of "wings of a butterfly". The section of the nasal triangle, as a rule, remains clean of rashes. On the hair cover, the heads of rash does not happen. The rash can be on the lobby and on the chin. In The rash period may appear for the feeling of heartburn and itching skin. After 1-3 days, the rash appears on the hands (preferably on extensions), simultaneously or a bit later appears on the buttocks, lower extremities and torso. The rash elements are located symmetrically, its practically never happens on palms and feet -Sini shades, bright red kaymum, forms a variety of figures in the form of rings, garlands, coins, loops, mesh-circular character rashes. On shoulders, buttocks, hips rash occupies white. lash part of the surface. On the trunk, a rash in the form of separate ridiculas or macular elements of pink color, uncomfortable, but may have a form of drain erythema. Duration of storage of rash 3-13 days, rarely up to 20 days. The disappearance of rash, as a rule, occurs gradually, through the stage of unstopped marble of skin, much less often there is light pigmentation and in single cases -tender peeling. No significant changes from internal organs are observed.

According to D.V. Polishko, an infectious erythema can run in the form of a light, medium-hard and severe form.

The severe form of the disease is characterized by the following symptoms: the temperature of 39 ° C and above, sometimes there is a burning place, a strong headache, insomnia, abundant rash.

Average shape: an increase in temperature to 38-39 ° C, moderate excitation or adynamia, moderate headache, insomnia, abundant rash.

Light form: The temperature is normal or subfebrile, the general condition is disturbed slightly, non-gentle, and sometimes and abundant rash.

Sometimes there may be catarrhal phenomena in the form of a catarrh upper respiratory tract, conjunctivitis. In general analysis of blood -normocytosis or leukopenia, eosinophilia, a rodder shift, an increase in ESR.

Diagnostics

Since specific diagnosis is not yet developed, the diagnosis of the infectious erythema of the Tsmmer is determined on the basis of a clinical picture and hemogram. Take into account the age of a false child and epidemiological data.

Differential diagnostics

Sometimes it is necessary to carry out with a ***rubella,*** from which it is characterized by a greater brightness of rash, the mergers of rash, forming a variety of figures that are not characteristic of rubella, duration of rash, lack of rash in the area of the nasopalla triangle, absence of an increase in occipital and rear cervical lymph nodes;

From the ***Bark***, the differences are also sufficiently expressed in the form of a lack of prodromal period, catarrhal changes, patches of filato-coil, such a strict rash stage, as well as the character of rash.

With differential diagnosis with ***scarlet*** fever, it is necessary to take into account the lack of angina, the symptom of raspberry tongue, lifting rash in the folds of the skin, lamellar peeling, taking into account the localization of the rash (on extensible extremities), its morphology (large spots, excellent blossom patterns) and other symptoms in patients with infectious erythema Tshamer;

The presence of erythema on the face in the type "butterfly" may require the differentiation of this disease with ***red lupus***, for the latter characteristic duration of the process, expressed infiltration of foci, their clear limits, the presence of follicular hyperkeratosis and scar atrophy on the site of past rashes.

Allergic diseases and medical disease were excluded on the basis of anamnestic data: the absence of signs of allergies in the past, contact with any allergen or administration of drugs, there are no characteristic rashes for infectious erythema (on the face in the form of a "butterfly", on the body in the form of figures), mainly symmetrical placement of rash on extensible surfaces. The infectious erythema of the Tshamer needs to be differentiated with a windy vacuum, polymorphic exudative erythema, infectious mononucleosis, etc.

Treatment

With mild form of disease - symptomatic treatment. In severe form, disinclusion and general mixing agents are used, as well as glucocorticoids.

Prevention

Specific prevention is not designed. Patients with an infectious erythema Tsmmer need isolation. Preventive measures in the cell are not carried out.Quarantine for people contacting patients with erythema Tshamer is not installed.

Control questions (infectious erythema)

1.Determination of infectious erythema.
2.Etiology of infectious erythema Tshamer.
3.Epidemiology of infectious erythema Tshamer.
4.Clinic of Infectious erythema Tshamer.
5.Differential diagnostics of the infectious erythema of the Tshamer.
6.Treatment of contagious erythema Tsmmer.
7.Prevention of infectious erythema Tsmmer.

Infectious erythema Rosenberg

In 1925, N.K.Rozenberg described a peculiar variant of infectious erythema in adults. He considered her hard the disease accompanied by a boom lean. Patients with an infectious erythema Rosenberg are found quite often, but practical doctors are still not well aware of this disease and often do not recognize it, therefore, a comprehensive study of the disease is definitely important.

Etiology

The pathogen is not installed, a virus is considered.

Epidemiology

The disease occurs in the form of sporadic cases throughout the year, mainly in the autumn-winter period. Contagiousness is insignificant. The ways of transmission of infection are not studied. Ill in the main person of the young and middle-aged.

Pathogenesis

Not learned.

Classification

Depending on the severity of the disease, 3 forms can be distinguished: light, medium gravity and severe.

Clinic

The incubation period is not installed. The beginning of the disease is sharp. Increasing body temperature is accompanied by chills. The fever is already on the first or on the second day of the disease reaches 38-39 ° C and in the future maintained at the same level. Of course, it has a weakening character, with severe leakage - permanent. Patients complain about on a strong headache, which is localized predominantly in the front-temporal area, sleep disorders and increasing general weakness, often distinguish between joint pain, limbs, lumbar.

One of the most characteristic clinical signs of erythema Rosenberg is a plenty spotted or spotted-papular rash that appears on 4-6 days of illness. It is localized on the extensor surface of the limbs, mainly in the area of large joints (shoulder, elbow, radiotherapy, knee and shin) and on buttocks, much less on the torso, very little or even it is not on the face. The development of rash elements usually occurs within the first two days. Initially, the rash elements are placed isolated from each other, have clear edges, rounded shape and size not more than 3-5 mm. Then, together with an increase in the amount of rash, individual elements are increased in diameter to 1.5 cm or more. The edges become less clear, and the form is less correct. Subsequently, the rash is often merged, forming a pattern of irregular shape or large erythematous fields, especially on buttocks, in the area of knee and ships. The rash first has a pink color, after 2-3 days it becomes purple red, then gradually brilliant and pale. The rash disappears 5-6 days from the moment of appearance. At the same time, the tussle occurs on the trunk, and on the palms and soles in a number of cases there is a large-lamellar peeling, as in scarlet fever.Patients are excited, especially at the beginning of the disease. The face is hyperemic, somewhat swollen. The eye conjunctiva is hyperemic, vessels sclera injected. Often increased and sensitive in palpation of cervical lymphatic nodes. Changes in the cardiovascular system are not expressed. The liver and spleen are usually increased. In the area of the soft palate is often determined by a spotted enanteme.

The disease lasts an average of 8-12 days. Reducing body temperature is reduced lysis. After its normalization, patients quickly restore their strength. In the analysis of blood - normocytosis or leukopenia, eosinophilia, a rodder shift, increasing ESR.

Diagnostics

Similar diagnosis of an infectious erythema of the Tshamer.

Differential diagnostics

Conducted with the same diseases as erythema Tsmera (Cyrus, rubella, scarlet fever, allergic diseases and medical disease, windpox, infectious mononucleosis, etc.), as well as directly with the infectious erythema of the Tshamer.

Treatment and prophylaxis

The same as in the infectious erythema of the Tshamer.

5. SUDDEN ERYTHEMA (EXANTEME)

Disease first described in 1900 titled Roseola infantum, later it was described and called a three-day critical fever with an exanteme, sixth illness.

Etiology

Most authors consider it an independent disease with a pathogen of viral nature. Until recently, the virus causing this disease was unknown, subsequently thought that a sudden exanthema is due to adenoviruses, enteroviruses, but now it is proved that in children this disease causes herpes simplex virus IV type.

Pathogenesis

Not learned.

Epidemiology

The disease occurs rarely, mainly in young children.

Clinic

Incubation period 3-7 days. The beginning of the disease is acute - the body temperature increases to 39-40 ° C and above. In spite of a strong fever, the well-being of children can remain satisfactory, but often there are moderate manifestations of intoxication: headache, posiness, restriction, loss of appetite. Sometimes vomiting, diarrhea, abdominal pain and lower extremities, seizures. The harder leakage is noted in older children. Fever lasts 3 days, in Individual cases up to 5 days, after which the temperature critically decreases to the norm and simultaneously (for 3-4 days) appears rash on the skin of the back (less rash begins to reduce the temperature, or after 1-2 days), then on the stomach, breast cage, neck, head and limbs. Rash is abundant, fine-spotted, pale pink on unchanged skin, edges are uneven, often with a pale isola, the propensity of them to the merger is weak. The rash is localized mainly on the back, less on the face and the bending surface of the upper extremities. The

process of rash lasts for several hours, after 2-3 days, the exanteme disappears without peeling and pigmentation. Pulse frequent, heart tones are weakened. Sometimes there are moderately increased regional lymph nodes. Quite characteristic changes in blood: from the first days of the disease there is leukocytosis, then leukopenia due to an increase in neutrophilic granulocytes, a rodder shift, relative lymphocytosis (up to 90%). In the urine, protein, leukocytes are determined. The failure of the disease is benign. In the fever sometimes it develops serous meningitis.

Diagnostics, Differential Diagnosis, Treatment and Prevention

Similar infectious erythema Tshamer and Rosenberg.

Knotted erythema

Etiology

Etiology is unknown, most likely viral etiology. The nodal erythema may be one of the manifestations of tuberculosis,rheumatism, ishersiniosis, benign lymphoretics, as well as other bacterial, viral, fungal diseases, and often a predictor of severe diseases of joints, colon, blood.

Epidemiology

The source of the infection is unknown. Assume an air-droplet mechanism of infection. There are sporadic, rarely - group diseases mainly in the spring and autumn. More likely to get sick.

Pathogenesis

In the pathogenesis of nodal erythema, an allergy is played by the pathogens of tuberculosis, streptococci and staphylococci. Changes are mainly localized in subcutaneous fatty tissue, they are productive-infiltrated, places - destructive nature, up to fibrinous necrosis of the vascular wall.

Clinic

The incubation period lasts 1-4 days. Beginning acute or subacute with an increase in body temperature for 1-2 days to 38-39 ° C, sometimes with chills. In children patients with tuberculosis, the clinic begins with weight loss, malaise, subfebrile body temperature, angina, lymphadenopathy. Children complain of headache, joint pain, occasionally possible catarrhal phenomena, intestinal dysfunction, posiness, poor sleep.

The rash appears on 3-5 days of illness in the form of dense,painful in palpation of nodes, symmetrically located on the anterior-internal surfaces of the legs, forearm, around the radiation joints, on the outer surface of the hips and butt painful in palpation of nodes, symmetrically located on the anterior-internal surfaces of the legs, forearm, around the radiation joints, on the outer surface of the hips and buttocks. The number of elements varies from single to several dozen, dimensions - from several millimeters to 2-5 cm. Nodes are dense, protrude above the skin, sharply painful to the touch, first bright red, and then purple-blue, greenish-yellow. Nearly located nodes can be drained, forming an erythematous area similar to Beshchi.

Increased temperature is constant, sometimes remitting. With the advent of node rash, the symptoms of intoxication are enhanced. Often there is an increase in

submandibular and cervical lymph nodes, an increase in the spleen. After 2-3 weeks, the nodes are absorbed without suppuration, the body temperature is reduced by a literally. The course of the disease is benign.

The nodal erythema can be observed in the form of such forms: a typical, migrating, dense, indefinite paniculitis. In streptococcal infection, relapses are possible. In the blood there are moderate leukocytosis, an increase in ESR.

Diagnostics

Based on a characteristic clinical picture and hemogram. Take into account the age of the patient and epidemiological data. Specific methods of laboratory diagnostics are not developed.

Treatment

Assign antibiotics, more often a penicillin series, antihistamines, in severe cases glucocorticide preparations.

Prevention

Prevention and measures in the infection cell are not developed.

Checkbooks (erythema)

1. Give an infectious erythema of Rosenberg.
2. Clinic of an infectious erythema of Rosenberg.
3. Treatment of an infectious erythema of Rosenberg.
4. Allow a sudden erythema (exanthem).
5. Etiology of sudden erythema.
6. Clinic of sudden erythema.
7. Treatment of sudden erythema.
8. Give the definition of nodal erythema.
9. What is the etiology of nodal erythema.
10. Epidemiology of nodal erythema.
11. Pathogenesis of nodal erythema.
12. Clinic nodular erythema.
13. Treatment of nodal erythema.

6. INFECTIOUS MONONUCLEOSIS

Infectious mononucleosis (synonyms: Filatovs disease, glandular fever, monocytic angina, Pfeiffera disease, etc.) - an acute anthroponized infectious disease caused by an Epstein-barrier virus from the HerpesViridae 4-type family with a drip transmission mechanism characterized by intoxication, fever, generalized lymphadenopathy, and the appearance of atypical mononuclears in the general blood test.

Etiology

The pathogen is an Epstein-Barr virus - represents a B-lymphotopic human virus belonging to a group of herpes viruses (family – Gerpes Viridae, gamma herpes

virinae submarity). This is a human herpes virus, type 4. The virus contains a two-disperse DNA. The virion consists of a capsid diameter of 120-150 nm surrounded by a lipid shell. Barr virus has a tropism to B-lymphocytes containing surface receptors to this virus. In them, there is a synthesis of full particles of virion, or only its separate components. Recent research revealed the presence of a virus and in the epithelial cells of the mouth and nasopharynx. Epstein Barr virus is detected not only in infectious mononucleosis, but also with other diseases - Burkitts lymphoma, nasopharyngeal carcinoma and with some lymphomas in persons with weakened immunity. The virus can be persisted for a long time in host cells in the form of latent infection. It has a number of antigenic components that are common with other Herpes group viruses. In general, there are 4 main EBV antigens:

• Early antigen (Early antigen - EA) appearing in the nucleus and cytoplasm, preceding the synthesis of viral particles. Contains D- I R-components.

• capsid antigen (Viral Capside Antigen - VSA) contained in the virus nucleocapside. In infected cells,containing the EBV gene, but in the cytoplasm of which there is no VSA, the replication of the virus does not occur.

• Membrane antigen (Membrane Antigen - MA).

• Nuclear antigen (EBSTAIN-BARR Nuclear Antigen - EBNA), consisting of a complex of polypeptides.

Isolate A and in strains of EBV. Between the strains of the virus allocated from patients with different clinical forms of mononucleosis, there are no significant differences.

The time of appearance and biological significance of these antigens are uneven. Knowledge of the terms of various antigens and the detection of antibodies to them give an opportunity with sufficient reliability to diagnose acute, latent and chronic EBV infection. In acute infection, antibodies to early antigens appear first, then to a nuclear antigen. At latent infection, antibodies to capsid and membrane antigens are detected in the absence of antibodies to early antigens.

Epidemiology

The source of infection is a patient with an infectious mononucleosis (especially with erased, abortive and latent forms), as well as viruses. The mechanism of air-droplet transmission (due to droplets) and contact (through toys, dishes, at a kiss), recently note the transmission of infection with hemotransfuzia. The cases of vertical transmission of EBV and sexually are described. Input gates are mucous membrane of rotoral and upper respiratory tract.The virus is released with saliva in the external environment within 12-18 months. After the primary infection, proved by studies of materials taken from the oropharynx. If you take the washing of the oropositive healthy individuals, then a virus also reveals 15-25%.

The disease is more often recorded in the form of sporadic cases, sometimes there are outbreaks of disease in schools and kindergartens. Infectious mononucleosis is recorded year-round, but mainly in spring and autumn periods. Among children, the first peak of morbidity accounts for 2-10 years, the second - among girls for 14-16 years, among boys - for 16-18 years. At the age of 2 years, children are rarely ill, and in the event of a disease, it has a subclinic course. The contagiousness index is not

installed. Small contagiousness is associated with a high percentage of immune persons (more than 50%), the presence of erased and atypical forms of mononucleosis, which are usually not detected, as well as a low concentration of the virus in saliva. Activation of infection promote factors for reducing general and local immunity.

Pathogenesis

Input gates of infectious mononucleosis are mucous membrane and lymphoid fabric of naso- and oropharynx, where the primary reproduction and accumulation of the virus occurs. The mucous membrane edema is formed, almonds and regional lymph nodes are increased. EBB gets in B-Lymphocytes, where replicated, accumulates and spread throughout the body with lymphogenic and hematogenous. The virus penetrates into lymph nodes, liver, spleen, causes proliferation of lymphoid and reticologistocytic elements, which is clinically manifested by lymphadenitis, hepatosplenomegaly. Activation of conditionally pathogenic microflora leads to the development of acute tonsillitis. Under the influence of virus B-lymphocytes proliferated and vary in atypical mononuclears (Virocytes).

Recently, great attention is paid to an infectious mononucleosis as an immune system disease. The virus does not destroy infected cells (B-lymphocytes), and stimulates their reproduction; can persist in lymphocytes long.

In response to fixation of EBB on the surface of B-lymphocytes, activation of T-lymphocytes-suppressors, natural T-killers, as well as the mechanisms of an antibody of dependent K-cell cytolysis are included. These mechanisms, on the one hand, inhibit proliferation and differentiation of B-lymphocytes, and on the other hand, lead to the lysis of infected B-lymphocytes, which contribute to the release of the virus into free circulation followed by its elimination with humoral specific antibodies. The described mechanisms contribute to the benign course of infectious mononucleosis.

After the primary infection of EBV stored in a human body in a small amount permanently. Cytotoxic T-lymphocytes and natural killers restrict primary infection and hold the pool of immortal EBV-infected in-lymphocytes under control. When any element of the immune response is broken, the small pool of EBV - infected cells can be expanded. This leads to the development of lymphoproliferative syndrome, lymphoma with a reduced immune response, and vice versa, if the answer is too active, fatal infectious mononucleosis may be observed or vertible.

Classification

The only generally accepted classification is not designed. In clinical practice it is convenient to use the following classification of infectious mononucleosis:

1. By manifestation of clinical symptoms:
- manifest form,
- subclinical form.
2. By the nature of the course:
- typical,
- Atypical:
- erased

- visceral.

3. By degree of severity:

- a light run,
- the course of medium gravity,
- severe course.

If identifying, in diagnosis indicate primary is a disease or relapse.

Clinic

The incubation period lasts from several days to 1-2 months, more often - within 20-50 days. Typically, the disease begins with prodromal phenomena: weakness, myalgia appears, headache, chills, loss of appetite, nausea. Such a state can last from a few days to 2 weeks, in the future there are sore throat, a temperature reaching 38-39 ° C. Often, in children, the disease develops acute, without prodrom. In most patients, a classic clinical triad is characteristic of infectious mononucleosis: fever, lymphadenopathy, sore throat.

The fever is a fairly frequent symptom, although there are possible cases of the disease in which the temperature does not rise. Chills and sweating are not characteristic. The character of the temperature curve is a variety of - permanent, remitting, although among children it is more likely to have an incorrect form. The duration of fever - from a few days to 1 month. And even longer, more often - 1-3 weeks. The correlation between the character of the temperature curve and other clinical manifestations is usually absent.

Lymphadenopathy is one of the most common signs of the disease, manifests itself early and disappears later than other clinical manifestations. The first increases the cervical lymphatic nodes placed in the form of garland along m. STERNOCLEIDOMESTOIDEUS. In the midst of the disease, you can find an increase in other groups of lymph nodes - peripheral (smeared, inguinal), internal (mesenteric, peribronchial). Increasing the internal lymph nodes may determine the appearance of additional symptoms - abdominal pain, cough, difficulty breathing. Increased lymph nodes can be a size from 1 to 4 cm The sore throat is due to local inflammatory changes. The mucous membrane of the posterior wall is hyperemic, swollen, hypertropholic follicles (granular pharyngitis). The tonsils are enlarged, often covered with a gentle white bloom. Possible activation of secondary infection, in this case, a dirty gray riot that is easily removed on tonsils appears on tonsils. In connection with the defeat of the palatine and nasoglovnyky tonsils, there is a nasal laying, the difficulty of nasal breathing, the emergency of voice. The frequent feature of the disease is hepato and splenomegaly developing usually 3-5 days of illness. Increase in the liver is determined palpation in half of patients, with ultrasound - in 85-90% of cases. In this case, there is always an increase in cytolytic enzymes, and in part of patients children exhibit a yellow skin color, sometimes only an ice-like glass. In the recovery period, the liver gradually decreases in size, although possible option and long hepatomegaly (within a few weeks). Enzymatic indicators are normalized before. It often increases the spleen, but it is not always possible to propagate. Increased spleen is dense, elastic, painless in palpation, significant increase in it causes a sense of discomfort in the left hypochondrium. The maximum pronounced

hepato-lainal syndrome becomes 5-10 years of illness. It should be noted that in some cases, a significant increase in the spleen is possible that during its palpation can lead to a bread of organ.

In 3-19% of patients with skin and mucous membranes there is a rash (urban, spotted, hemorrhagic, scarlet fever, crude), an embodiment is possible on mild palatine. In the literature, cases of occurrence of a rash with infectious mononucleosis in patients receiving ampicillin and derivatives thereof: amoxicillin and amoxicillin + clavulonic acid, etc.

Often, there is a flaw and swelling of the eyelids, which is associated with lymphostasis, which arises as a result of the defeat of nasogla and lymph nodes.

Possible tachycardia, a slight muffling of heart tones, sometimes systolic noise disappearing as a child recovery. Changes to ECG are not detected. Temporary violations of the cardiovascular system are treated as "infectious heart".

Changes in the lungs arise only as complications associated with screws of acute respiratory infections and activating microbial flora.From the nervous system with infectious mononucleosis, cases of serous meningitis, encephalitis, polyneuritis, myelitis, neuritis of cranial nerves, cerebellum ataxia, psychosis and human lymphoma are described. These manifestations can be observed separately, or during acute infectious mononucleosis.

The disease lasts at least 2-4 weeks. The first 2 weeks correspond to the midst of the disease, at this time an increase in temperature, manifestations of general intoxication. After 2-3 weeks, the period of reconvalescence begins: the temperature of the body decreases, the manifestations of intoxication decreases, the lymph nodes, liver, spleen are reduced, the hemogram is normalized. But the process can be extended to 2-3 months and even longer.

In children of the first year of life, an infectious mononucleosis has clinical features. Thus, it often turns out to be the Face Facility, swelling of the eyelids, neck, conjunctivitis, early film angina, dyspeptic phenomena. The course of illness is favorable.

Atypical forms of infectious mononucleosis are characterized by the lack of any leading symptom of the disease or unusual expressiveness of any symptoms (local lymphadenopathy, sharply expressed jaundice, etc.).

At erased, the course of clinical manifestations are fuzzy, which determines the high frequency of diagnostic errors.

Prolonged course is defined in case of conservation hematological changes and lymphadenopathy to 6 months.

Chronic forms are small. Prolonged persistence of EVV is associated with the presence of immunodeficiency in the patient, including HIV-infected children. In addition, the development of neoplastic processes and autoimmune diseases is related to EVV. Therefore, patients who have suffered infectious mononucleosis in the duration of the process would months and longer, residual phenomena in the form of astheno-vegetative syndrome, dyspeptic phenomena, subfebrile. For the final definition of chronic form, the puncture of the bone marrow, lymph nodes, liver is required. Taking into account the ability of EVV to persistence, one should remember

the possibility of developing mixt pathology against the background of Persistency EVV.

At the same time, it is believed that recurrences and chronic course of infectious mononucleosis in children are not.

At the same time there is an opinion that recurrences and chronic course infectious mononucleosis does not occur in children.

Complication

They develop seldom, but in case of their occurrence the forecast is considerable deteriorating.

They are mainly based on autoimmune processes neurological complications are more common in children (meningitis, encephalitis, meningoencephalitis). Meningitis develops in the acute period of the disease. Patients complain of the main pain, nausea, vomiting, which does not bring relief, may convulsions, loss of consciousness, meningeal signs. At examination of cerebrospinal fluid is detected lymphocytic pleocytosis, sometimes with the presence of mononuclear cells. The duration of such meningitis is from several days to several weeks. More often the process ends with complete recovery, fatalities are rare.

Encephalitis developing on the background of mononucleosis poses a significant danger. Localization of the process diverse, which causes a large polymorphism of clinical manifestations; chorea-like movements, paralysis, lesions respiratory center, coma. The phenomena of encephalitis can to be combined with defeat of a spinal cord, peripheral and cranial nerves. Sometimes such patients develop mental disorders (psychomotor agitation, hallucinations, etc.). The danger of encephalitis is its ability to rapid progression. If the process is eliminated quickly, then residual effects after the disease does not happen.

At primary infection development of a syndrome is possible Hyena-Barre (ascending acute polyradiculoneuritis with protein-cell dissociation in the cerebrospinal fluid), Bell's palsy (facial muscles due to damage to the facial nerve), transverse myelitis.

Hematological complications are manifested by leukopenia, pronounced agranulocyte reaction, thrombocytopenia. Significant thrombocytopenia may be accompanied bleeding, thrombocytopenic purpura. Hemorrhagic syndrome is sometimes manifested by hemorrhage into the retina.

The development of autoimmune anemia is possible.

A serious complication is the rupture of the spleen, which is almost always leads to death. The reason for the break may be sharp movement of the patient, rough palpation of the organ.

In young children, infectious mononucleosis can complicated by sharp swelling of the tonsils and mucous membranes pharynx, accompanied by the development of obstruction respiratory ways. The cause of obstruction is an increase paratracheal lymph nodes.

Enlargement of the liver - one of the most characteristic manifestations infectious mononucleosis, but in some patients it is accompanied by jaundice with a clear increase activity of cytolytic enzymes, which can be classified as hepatitis.

Some patients may develop myocarditis, pericarditis, or confirmed by ECG examination.

During convalescence the development of interstitial nephritis of autoimmune origin is possible. Less often lesions of endocrine glands with development are found mumps, orchitis, pancreatitis, thyroiditis. The course of mononucleosis may be complicated by exogenous attachment or activation endogenous infection.

Diagnosis

Infectious mononucleosis is diagnosed on the basis of the following clinical manifestations such as fever, nasal breathing difficulties, edema, moderate hyperemia of the oropharynx, polyadenitis, hepatosplenomegaly and characteristic hematological picture.

From additional research methods the most important is general blood test.At the beginning of the disease in most patients there is a decrease the content of segmental and increase rod-shaped neutrophils. Leukocytosis gradually increases ($9\text{-}10^9$- $15\text{-}10^9$/ l) with a moderately increased number of mononuclear cells (atypical mononuclear cells, lymphocytes, monocytes) up to 15-50%. These changes persist for 3-6 months. Intercurrent diseases convalescents are often accompanied mononuclear reaction. ESR is accelerated moderately (up to 20-30 mm / hour). The most characteristic sign of the presence in the blood atypical mononuclear cells. In most children atypical mononuclear cells can be detected within 2-3 weeks from onset disease. In some cases, mononuclear cells disappear already at the end of the first week of illness, although sometimes they can appear in the blood for more than one month. Number of atypical mononuclear cells in the blood of patients ranges from 5-50%. There is connection between the number of atypical mononuclear cells and severity of the disease.

In the presence of meningeal syndrome in the cerebrospinal fluid detect low lymphomocytic pleocytosis.

When conducting biochemical research, even for absence of jaundice there is an increase (2-3 times) activity of ALT, AST, alkaline phosphatase. With the appearance of jaundice, bilirubin level increases, activity of cytolysis enzymes significantly increases.

The essence of specific research methods is conducting serological tests for antibodies to virus antigens (EA, MA, VCA, NA), the antigens or titers themselves heterophile antibodies.

Antibodies to EA are detected as in patients with infectious mononucleosis, and Burkitt's lymphoma and nasopharyngeal carcinoma. They appear a few weeks later onset of the disease and disappear a few months later recovery. These antibodies are markers of severe course infectious mononucleosis. Antibodies to VCA appear in the first days of the disease, but the increase in titers occurs slow. At the primary infection, are detected antibodies class lgM, which in 1-2 months disappear. IgG antibodies appear at the same time, but remain for life. Antibodies to NA appear after 3-4 weeks and persist a long time. Their lifelong detection indicates persistence of the virus.

Detection of heterophilic antibodies in children often gives false-negative reaction due to imperfect development immune system, therefore, especially for young children, this method is ineffective. Most often put the Paul-Bunnel-Davidson reaction, which reveals high titers antibodies (up to 1: 1024). It should be borne in mind that the reaction gives positive result also with rubella, scarlet fever, influenza, malaria.

With the help of the Goff-Bauer reaction it is possible to quickly and reliably detect membrane (MA) and capsid (VCA) antigens in latent infection and early (EA) and nuclear (NA) antigens in acute infection.

Use the detection of EBV nucleic acid by PCR in blood, saliva, lymphatic tissue. Often in the plan of examinations include other additional methods, in particular ultrasound, chest radiography, ECG, etc.

Differential diagnosis

Bacterial angina. Similar: lesions of the tonsils for anginal type. Different: bacterial angina characterized by severe sore throat with hypertrophy tonsils without pharyngitis, enlargement and pain only submandibular lymph nodes, no hepatolienal syndrome, no mononuclear reaction.

Diphtheria. Similar: lesions of the tonsils for anginal type. Different: diphtheria is characterized by dirty-gray plaques, which removed with difficulty, exposing the bleeding surface; enlarged only submandibular and cervical lymph nodes, swelling of the neck tissue, no hepatolienal syndrome,neutrophilic leukocytosis.neutrophilic leukocytosis.

Rubella. Similar: lymphadenopathy, small-spotted rash. Different: with rubella, enlarged only the neck and occipital lymph nodes, no symptoms of pharyngitis and sore throat, the rash appears from the first day of illness, the liver and spleen do not enlarged, atypical mononuclear cells are absent.

Scarlet fever. Similar: fever, sore throat, rash. Different: scarlet fever is characterized by very bright redness of the mucous membrane of the oral cavity, “raspberry tongue”, lesion tonsils have all the features of streptococcal angina, facial skin is brightly hyperemic with a pale nasolabial fold triangle, small-spotted rash appears at first days, no generalized lymphadenopathy and hepatolienal syndrome.

ARVI. Similar: fever, enlargement of the cervical lymph nodes, enlargement of the liver and spleen. Different: for ARVI are typical conjunctivitis, rhinitis, cough, no pronounced mononuclear reaction.

Viral hepatitis A. Similar: jaundice, enlargement liver and spleen, fever. Different: with VHA fever lasts no more than 2-5 days; sore throat absent, absent local and generalized lymphadenopathy, not typical mononuclear reaction.

Mumps. Similar: enlargement of the submandibular and parotid lymph nodes. Different: with mumps is a lesion of the salivary glands, their soreness, a positive symptom of Murson, no symptoms of tonsillitis, no hepatosplenomegaly, there is no mononuclear reaction.

Lymphocytic leukemia. Similar: fever, hepatolienal syndrome. Different: with lymphocytic leukemia is pronounced hemorrhagic syndrome, tonsillitis is not typical,

the appearance normoblasts, significantly accelerated ESR, erythropenia and thrombocytopenia.

Lymphogranulomatosis. Similar: fever, enlargement cervical lymph nodes. Different: with lymphogranulomatosis in blood test-neutrophilic leukocytosis, in punctate with lymph nodes – Berezovsky-Sternberg cells.

Treatment

Hospitalization of patients is not mandatory. Basic indications for hospitalization: severe course, presence or threat occurrence of complications, the course of the disease in the background severe immunosuppression, children under 1 year.

Regime– exercise restrictions

Diet - exclusion of spicy, fried extractives dishes. No specific treatment has been developed.

Symptomatic therapy includes antipyretics drugs, antihistamines.

In severe forms prescribe glucocorticosteroids (12 m / kg / day for prednisolone) – 3-5 days.

When joining a secondary bacterial infection - antibacterial drugs: macrolides, cephalosporins.

Ampicillin and its analogues are contraindicated.

Terms of discharge for infectious mononucleosis:

-satisfactory condition

-normalization of body temperature

-appearance of manifestations of acute tonsillitis

- normalization of the size of the liver and spleen (or clear tendency to decrease their size)

-in the general analysis of blood: normalization of quantity atypical mononuclear cells (less than 10%), normalization of ESR.

-Normalization of ALT activity, if it increased during illness.

Prevention

Patients with EBV infection do not need isolation. Adults who suffered from infectious mononucleosis, cannot be donors. Children after infectious mononucleosis for some time can not go in for sports, they are contraindicated large physical activity, as is possible spontaneous rupture spleen. Carrying out preventive measures in the hearth the disease is not foreseen. Vaccines (polypeptide, genomic) against EBV are under development.

Control questions (infectious mononucleosis)

1. Determination of infectious mononucleosis.
2. Etiology of the disease.
3. Epidemiology of the disease
4. Pathogenesis of infectious mononucleosis.
5. Classification.
6. Clinic of infectious mononucleosis

7. Complications of infectious mononucleosis.
8. Diagnostics of the disease.
9. What changes in the general blood test is the most characteristic a sign of infectious mononucleosis?
10. Which of the serological reactions are used as method of rapid diagnosis of infectious mononucleosis?
11. In the detection of which antibodies to Epstein-Barr virus can confirm infectious mononucleosis?
12. Clinic of infectious mononucleosis.
13. Treatment of infectious mononucleosis.
14. What antibiotic can not be prescribed during treatment infectious mononucleosis?
15. Differential diagnosis of infectious mononucleosis.
16. Prevention of infectious mononucleosis.

7. MEASLES

Measles is an acute infectious disease that is caused virus, transmitted by airborne droplets, characterized by two-wave fever, catarrhal inflammation of the mucous membranes of the respiratory tract, eyes, presence of Filatov-Koplik spots, and with a new increase body temperature – a staged occurrence on the body characteristic maculopapular scarring, leaving pigmentation.

Etiology

The causative agent of measles is the virus Polinosa morbillarum family of Paramyxoviridae of the genus Morbillivirus, contains RNA, but in contrast other paramyxoviruses, it does not contain neurominidase.

Birion with a diameter of 120-180 nm, oval shape. Virus has hemagglutinating, hemolytic and symplast-forming activity. Emit 6 major antigens of the virus, three of which are bound to its shell: membrane shell protein (M), glycoprotein (H), glycoprotein F, which has the highest immunogenicity, as well as a large protein (L), phosphoprotein (P), nucleocapsid protein (NP).

The measles virus is volatile, outside the human body the virus dies within 30 minutes, so the final disinfection of measles is not carried out.

Enidemiology

The source of measles infection is only a sick person who becomes contagious from the last two days of the incubation period and more five days after the appearance of the rash, in the presence of complications this period is extended to 10 days. The mechanism of transmission of infection – respiratory droplets. The virus can be transmitted to large distances (to adjoining rooms, apartments, floors). Index contagiousness is 95-98%. Due to the transplacental immunity children under 3 months do not get sick, and at the age of 3 to 6 months, gett sick very rarely. The incidence is registered during year, but its maximum increase is observed in the

autumn-winter and spring periods. After the infection formed stable immunity, cases of recurrent diseases range from 0.5-3% of all cases.

Pathogenesis

The initial gate is the mucous membranes of the upper respiratory tract and eye conjunctiva. The virus settles on the mucous membranes, penetrates into the submucosal space and regional lymph nodes, where it is fixed and primary replication. From the third day of the incubation period, the virus enters the bloodstream- primary viremia. The amount of virus is still small in blood and can be quickly neutralized by immunoglobulin. This fact was the basis of passive immunoprophylaxis meales. From blood the measles virus enters to a spleen, liver, bone marrow, lymph nodes, infects mononuclear cells and lymphocytes. From 7 day of the incubation period develops recurrent viremia, which leads to fixation of the virus in epithelial cells of the skin, respiratory tract, eye conjunctiva, intestines with the development of dystrophic changes in them.

Increased concentration of the virus in the blood leads to it fixation and further reproduction in cells reticuloendothelial system of all organs (lymph nodes, tonsils, spleen, liver, etc.), formation reticuloendotheliocytes (giant multinucleated cells Warten-Finkeld).

Measles rash is an infectious-allergic dermatitis pronounced exudative component that occurs due to the interaction between sensitized lymphocytes and viral antigens in the capillary endothelium and skin cells. The same inflammatory process occurs in the mucosa the oral cavity, where the necrotized epithelium is cloudy and formed small white foci of necrosis (spots Koplik- Filatov).

In addition to the destruction of cells of mucous membranes, affected T-lymphocytes, which leads to lymphopenia. Develops a state of anergy, it's a decrease in total and local immunity, which lasts up to a month. During viremia when measles virus enters the cells of the CNS, which can lead to such a severe complication as measles encephalitis and encephalomyelitis. Its occurrence is associated with a violation regulation of the immune response in measles, in which immunocompetent cells lose the ability to distinguish "own" / "foreign", and formed immune reactions against their own tissue antigens by type of hypersensitivity. However, a number researchers believe that encephalomyelitis at measles is not the only one autoimmune disease, but also the result of the virus on walls of vessels of a brain and a spinal cord with secondary damage to neurons and nerve fibers.

Classification

There is no generally accepted clinical classification of measles, however, majority clinicians are distinguished depending on the course:

-easy;

-moderate;

-severe course of the disease: a) without hemorrhagic syndrome;
b) with hemorrhagic syndrome.

The statistical classification of measles given in ICD-10, includes the following options:

1. Measles complicated by encephalitis;
2.Measlescomplicated by meningitis;
3. Measles complicated by pneumonia;
4. Measles complicated by otitis media;
5. Measles with intestinal complications;
6. Measles with other complications (keratitis, measles keratoconjunctivitis);
7. Measles without complications.

Given the severity of the main clinical manifestations of the disease allocate course:

- typical;
-atypical, with options: 1. abortive,
2. mitigated,
3. erased;
4. asymptomatic.

Clinic

The incubation period of measles is 9-17 days and lasts up to 21 days with the introduction of immunoglobulin, or other blood products. In the clinical picture of measles points out three periods: catarrhal (prodromal), rash and pigmentation. The catarrhal period lasts 3-5 days. It begins with fever up to 38-39°C, cough, runny nose. By 2-3 days the cough worsens, appears conjunctival hyperemia, scleritis, photophobia, which can accompanied by blepharospasm. Mucous membranes oral cavity, soft palate become bright-red, swollen, fluffy. On the soft palate appears enanthema, on the mucous membranes -pathognomonic for a meameasl a spots of Filatov-Koplik. More often they are found on The mucous membranes of the cheeks in the area of small molars as well on the mucous membranes of the lips, conjunctiva, genitals, etc. During the same period, a grayish plaque may appear on the mucosa gums. Stools often become liquid. Measles enanthema appears 1-2 days before exanthema. Sometimes in catarrhal period of measles on the skin appears speckled scarlet fever or urticarial rash (rach-rash), which disappears in 1-2 days or after the appearance of a measles rash.

Through 4-5 days against strengthening symptoms of intoxication, catarrh of mucous membranes, temperature rise to 39-40°C, appears spotty-papular rash and begins rash period. On the first day of the rash, the first elements of the rash appear behind the ears and then on the face and neck. On the 2nd day the rash occurs on the torso. On the 3rd day the rash appears on distal parts of the upper and lower extremities. In modern conditions measles rash can appear in 2 stages: in the first day- the face, torso; on the other-on the extremities. Such staged rash is a very important diagnostic feature. Measles rush evenly covers the skin, prone to fusion and located on unaltered skin. The rash can be profuse, sometimes acquire a hemorrhagic character or, conversely, very much scanty, in the form of individual elements. The patient has a typical appearance: swollen face, red eyes, abundant nasal discharge. Body temperature on the 1st day of the rash is significant higher than in the catarrhal period. Sometimes 1-2 days before the rash the temperature

decreases and a new increase on the 1st day rash gives a temperature curve of two-humped nature. The temperature remains elevated throughout the rash.

The measles rash very quickly begins to darken, later becomes brown and the period of pigmentation begins. Pigmentation begins in the order of origin, first on the face, then the torso, and then on the limbs. Withheld pigmentation 1-1.5 weeks, sometimes longer, and then there is a small shelling. During the period of pigmentation, the temperature normalizes. The general condition is normalized. During the convalescence of measles asthenia and anergy has been observed for a long time.

Atypical course. Happens in people who have a partial immunity against measles as a result of conducted in the long term vaccination or received after contact with patients with prophylactic specific immunoglobulin.

Abortive course. Characterized by typical initial manifestations of the disease, the appearance of rashes on the mucosa oral cavity and facial skin, and then the rash does not spreads, the temperature normalizes, is fast process regression.

Mitigated measles. It is characterized by elongation incubation period up to 4 weeks. Followed by all symptoms erased: t rarely exceeds 38°C, catarrhal symptoms are insignificant, often not found, Belsky – Filatov-Koplik spots.

Erased current. The body temperature rises slightly or remains normal, catarrhal syndrome limited to a slight sore throat, nasal congestion, rashes pale and sparse.

Asymptomatic course. The appearance of specific antibodies in people who have never had measles or been vaccinated against it him, an increase in antibody titer.

Measles in children of the first year of life

Measles in children can be a congenital infection. At fetal infection of the disease is subacute sclerosing panencephalitis, the symptoms of which appear at the first days after the birth of a child. In pregnant women with measles may be premature births and miscarriages.

If the mother did not have measles immunity, then her childn can get measles from the first days of life. The course of the measles in them light and can even be erased. Catarrhal phenomena are pronounced Moderate, body temperature normal or subfebrile. Rash sparse, there may be no phasing. But they have more often there are secondary complications, especially of the digestive tract, that primarily manifested by diarrhea. After the transfer the disease in the neonatal period is specific anti-measles immunity does not occur in children in case of recurrence infections can cause measles again.

Complications of measles can be primary, that caused by the virus itself (false croup, pneumonia, encephalitis, nosebleeds, diarrhea, keratitis, nephritis), and secondary, that caused by stratification bacterial infection (otitis, otogenic meningitis, sinusitis, frontitis, pneumonia, mediastinitis, pleurisy, coma, skin gangrene, acute appendicitis, abscesses).

The most dangerous complication of measles is the occurrence subacute sclerosing panencephalitis.

Subacute sclerosing panencephalitis is a classic slow viral infection, which is more common in adolescents and young people. It can occur 1-6 years later transferred

measles. Characteristic of this disease are progressive neurological disorders in the form of epileptic syndrome, ataxia, hyperkinesis, paresis, blindness, decreased intelligence. The duration of the disease is 1-3 years, the consequences are unfavorable.

Diagnostics

In the general analysis of blood leukopenia, lymphocytosis, eosinophilia, thrombocytopenia.

Birological method is complex, rarely used.

Cytological examination (cytoscopy) of smears-prints with oropharynx-identification of multicore typical of measles giant cells.

Serological methods (RTGA and RPGA) – titer increase antibodies in the dynamics of 4 or more times.

Enzyme-linked immunosorbent assay (IFA) – detection of antibodies to measles virus class lg M (acute period), increasing the titer of the class Ig Gb 4 times or more (transferred disease).

Immunofluorescence method. At the end of the prodromal period and during the rash conduct research smears- fingerprints from the nasal mucosa, treated with a specific luminescent serum to detect antigens measles virus

Differential diagnosis

In the first days of the disease, when the leading are catarrhal and general toxic syndromes, differential diagnosis should be performed with influenza, parainfluenza, adenovirus disease, pneumonia.

Distinctive features of **influenza** from measles:

- no manifestations during the incubation period;
- leading symptom-tracheitis, no hoarseness;
-there is no predominant swelling of the lower eyelids;
- presence of a rash is not typical.

Parainfluenza distinguishes:

-slught intoxication;
-lack of conjunctivitis;
-no rash.

From moments of measles rash must be differentiated from rubella, enterovirus exanthema, scarlet fever, infectious mononucleosis, meningococcemia, medicamentous exanthemas.

Differences between rubella and measles:

-relatively minor intoxication;
-early appearance of exanthema (1-2 days);
- no staged rash,
-insignificant catarrhal syndrome;
- the nature of the rash: small, not prone to merging;
- generalized lymphadenopathy;
- arthritis.

Scarlet fever is distinguished by:

- features of catarrhal syndrome (tonsillitis);

- early appearance of rash (1st day);
-nature of the rash (small, draining, located on hyperemic skin);
- features of exfoliation after rash (bran-shaped, and on the palms of the soles- large-plate);
- "raspberry tongue".
-nature of complications (myocarditis, nephritis);
- leukocytosis, increased ESR.

Assist in making a differential diagnosis can specific methods.

Treatment

The question of hospitalization in the infectious department decided individually.

Patients of the 1st are subject to hospitalization years of age, with severe forms of measles, with complications and for endemic indicators (children's institutions of the closed type). It is necessary to provide isolation to patients.

Bed rest is assigned for the entire fasting period. Food should be easily digestible, not contain irritants. Patients should receive sufficient the amount of liquid in the form of juices, compotes.

Complicated sticky, moderate measles and atypical forms measles does not require medical treatment.

Patients are prescribed:

-hygienic measures of the oral cavity and eyes;
-frequent ventilation of premises;
-when colds -vasoconstrictors;
-when coughing – mucolytic drugs;
-vitamin A orally.

Prevention

General prevention. Timely detection and isolation patients, contact monitoring. When it occurs cases of measles in closed children's groups quarantined for 17 days.

Emergency prevention. Contact children are administered anti-measles human immunoglobulin at a dose of 3 ml. In younger children are mainly subject to such prevention (from 2 months to 3 years), as well as pregnant women, persons with acute and chronic infection accompanied by immunodeficiency.

Specific planned prevention (vaccination). It is performed at the age of 12 months: it is injected once subcutaneously live cow vaccine (0.5 ml). Re-introduction of the vaccine conducted on seronegative children aged 6 years. If the child was not vaccinated against measles under the age of 2-3 years, in case of her contact with a measles patient, an emergency is performed vaccination with live attenuated vaccine. To vaccinate proved effective, it should be done no later 3-4 days after contact. After vaccination is created enough intense immunity for a period of 10-15 years.

Control questions (measles)

1. Define measles.
2. Etiology of measles.
3. Epidemiology of measles
4. Pathogenesis of measles.
5. In what term after contact with the patient it is expedient introduction of immunoglobulin to prevent disease?
6. Classification of measles.
7. Clinic.
8. What periods are allocated in the measles clinic?
9. What does the term “staged rash” mean?
10. Clinic of atypical forms of measles?
11. Clinic of measles in children of the first year of life.
12. Complications of measles are caused by a virus (primary).
13. Secondary (bacterial) complications of measles.
14. What is the most dangerous complication of measles?
15. Diagnostics of measles?
16. Differential diagnosis of measles?
17. Treatment.
18. Prevention.

8. RUBELLA

Rubella is an acute anthroponotic infectious disease preferably with an air-drop transmission mechanism that caused by rubella virus and characterized small-spotted rash, minor catarrhal phenomenas and enlargement of lymph nodes (mainly posterior cervical and occipital).

Etiology

The causative agent of rubella virus containing RNA refers to genus Rubivirus, family Togaviridae.

Virus has a spherical shape, a diameter of about 60 nm, RNA- relevant. The virus is surrounded by a supercapsid on the surface of which there is glycoprotein spikes E1 and E2 up to 10 nm long, that provide the reception of the virus on the target cell and penetration its inside this cell. Glycoproteins possess hemagglutinating properties. The rubella virus in the cell-target of the infected organism penetrates by endocytosis. In the cytoplasm of the cell is then the release of genomic RNA. In the future, there is an active replication of viral nucleotides. At the final stage, the nucleocapsid, passing through the cell membrane, covered by a section of this membrane, budding from the cell, gets a closed outer shell (supercapsid).

Virus is relatively unstable in the environment. At room temperature dies in a few hours, when boiling-in a few minutes. Poorly tolerates drying, action direct sunlight, UV, various disinfectants. Virus is thermolabile. But when frozen it remains good (several years at a temperature of -70 ° C).

Epidemiology

The source of infection is a sick person (even in the absence obvious clinical manifestations), as well as viruses. Excretion virus in external environment with nasopharyngeal mucus, sputum person begins in the incubation period for 1-2 weeks before the appearance of rashes. Excretion of the virus stops after 2-3 weeks after the onset of the rash, continuing thus and when the person considers himself healthy. The most intense excretion of virus from the body of a sick person occurs in the first 5 days after the appearance of the virus.

The easier the disease, the shorter it is usually the period of virus isolation. It is especially long when congenital rubella – up to 1.5-2 years or more, and the virus also found in urine and feces.

Rubella is highly contagious diseases. The index of continuity is approaching in different ways sources up to 90-98%. This causes its rapid spread in children's non-immune groups.

Most cases the disease occurs at the age of 2 to 9 years, and before period of adulthood is about 70-80% of people have in their blood specific anti-rubella antibodies. The main route of infection-air- droplets contact, but not excluded also transmission mechanism. In addition, there is another way of infection-transplacental. Transmission of the virus in this way is possible in all periods of pregnancy, but infection is especially dangerous in the 1st trimester of pregnancy.

Rubella is characterized by a certain seasonality – winter and spring, because at this time the virus is able to last longer stored in the environment, and closer communication of people indoors.

The transferred disease leaves lifelong immunity. In addition, children under 6 months are also not sick since most of them have immunity inherited from the mother.

Pathogenesis

Virus enters the body through the mucous membrane of the upper respiratory tract and multiplies in the lymph nodes and then enters in the blood. The virus infects the vascular endothelium, creating their increased permeability, edema and hemodynamic disorders in tissues. Clinically, this is manifested by catarrhal syndrome, intoxication. In the vascular endothelium of the surface layers of the skin virus forms a focal inflammatory reaction that causes the appearance rash. 2-3 days later in the blood appear virus-neutralizing antibodies, which leads to the release of the body from the pathogen and the formation of long-term immunity.

In addition, viruses can enter leukocytes (lymphocytes), where they can be found a week before emergence clinical symptoms.

Damage to fetal tissues is exacerbated by hypoxia due to damage to the vessels of the placenta and hemodynamic disorders in it.

Classification

Statistical classification (according to ICD-10, 1995):

1. In 06 Rubella;

2. P 35.0 Congenital rubella;

- B 06.0 Rubella with neurological complications:

a) encephalitis (G 05.1);
b) meningitis (G 02.0);
c) meningoencephalitis (G 05.1);

- B 06.8 Rubella with other complications:

a) arthritis (M 01.4);
b) pneumonia (J 17.1);

- At 06.9 Rubella without complications.

Clinical classification of Y. V. Lobzin (2000):

A. Acquired:
a) manifest form - typical (mild, medium, severe);
- atypical (without rash);
b) inapparent (subclinical):
- with complications;
- without complications;
B. Congenital:
- impressions of the nervous system;
- congenital heart disease;
- hearing impressions;
- eye impressions;
- mixed impressions;
- residual phenomena of congenital rubella.

Clinic

The incubation period for rubella is 11-24 days (14-21). In the clinical picture of rubella there are two periods: catarrhal (prodromal) and rash.

The prodromal period is very short - from a few hours to 1-2 days. At this time, patients may experience slight chills, drowsiness, sometimes a sore throat, cough, minor rhinitis. At careful inspection it is possible to notice in most cases easy hyperemia of a conjunctiva, and sometimes on a mucous membrane of a soft palate small red-pink spots (Forsheimer's spots).

Then begins a period of rash - a characteristic sign of rubella, and sometimes its first symptom. They appear on the face and spread for several hours without any sequence to the whole body and limbs. Their localization on extensor surfaces of extremities, a back, buttocks is characteristic. Rash small-spotted, 2-4 mm in diameter, rarely - spotted-papular, pale pink, round or oval, with clear contours, smooth surface on unaltered skin, has no tendency to merge. Disappears after 1-3 (4) days, leaving no pigmentation or peeling. The pathognomonic symptom for rubella is an increase in all peripheral lymph nodes, especially occipital, auricular, posterior. No other disease is accompanied by such a significant increase, compaction and often soreness of these groups of nodes. Lymph node enlargement is the first longest symptom of rubella, as it lasts for 2-3 weeks after the rash disappears, and in some cases longer. There is no correspondence between the intensity of the rash and

lymphadenitis. Lymphadenitis is a constant sign of rubella, although there may be no rash.

In the midst of the disease, there may be signs of catarrhal inflammation of the upper respiratory tract in the form of a slight runny nose and conjunctivitis. Unlike measles, there is no photophobia, in most cases of rubella there is enanthema - individual pink spots on the soft palate.

The course of rubella in children 2-14 years is the most typical and mild.

Atypical forms of rubella are very diverse. Sometimes it begins immediately with a rash, without any prodromal signs. There may be asymptomatic forms (inapparent), which are diagnosed only after laboratory tests.

Congenital rubella. In the case of a pregnant woman with rubella in a manifest or asymptomatic form, the risk of developing fetal abnormalities is close to 100% when infected in the first weeks of pregnancy, 40% - in the 2nd month, 10% - in the 3rd month, 4% - in II-III trimesters. Congenital rubella syndrome manifests itself in the form of a triad of predominant anomalies - cataracts, heart disease, deafness (Gregg, 1941). Later, these anomalies included microphthalmia, occlusal anomalies, craniocerebral deformities (microcephaly, hydrocephalus), encephalopathy, cleft palate, hepatitis, myocarditis, glaucoma, malformations of the genitourinary system, dermatitis, thrombocytopenia, hypogammaglobulinemia.

In 40% of cases, there is fetal death and miscarriage. Some developmental defects caused by the virus may be detected at a later period. Damage to the fetal brain leads to the development of chronic meningoencephalitis, clinically it manifests itself in infants in the form of drowsiness, lethargy, or, conversely, increased excitability, seizures. Further microcephaly is defined.

Early neonatal signs of congenital rubella include numerous hemorrhagic rashes on the background of thrombocytopenia lasting for 1-2 weeks, hemolytic anemia with reticulocytosis, hepatosplenomegaly, hepatitis with hyperbilirubinemia, interstitial pneumonia. Most changes disappear within 6 months. In addition, these children have low body weight and small growth at birth. They may lag behind in physical and mental development.

Diagnosis

With acquired rubella:

- General blood test (leukopenia, neutropenia, lymphocytosis, plasma cells, normal ESR;
- Virological - isolation of the virus from washes from the nasopharynx, blood, feces, urine.
- Serological method (RN, RPGA, RZH, RIF) - increase in antibody titers in the dynamics of 4 or more times;
- Enzyme-linked immunosorbent assay (ELISA): determination of specific Ig M class antibodies in the acute period of the disease and Ig G after infection in the blood, if necessary in the cerebrospinal fluid;
- PCR of blood, urine, saliva, if necessary, cerebrospinal fluid - the release of virus RNA.
- *With congenital rubella:*

- ELISA: detection of specific antibodies of class Ig M;
    - Serological method (RPGA): stable positive result;
    - Detection of virus RNA (blood, urine, saliva, feces, cerebrospinal fluid) by PCR.

Differential diagnosis

The leading clinical manifestations of rubella are exanthema and lymphadenopathy. First of all, differentiate rubella from measles.

There are **measles**:

- very high contagiousness;
- acute, sudden onset, severe intoxication and catarrh;
- scleritis, conjunctivitis, photophobia, lacrimation;
- no generalized lymphadenopathy;
- later (3-4 days) the appearance of a rash with a clear slow (about 3 days) stages of rash;
- the presence of spots Koplik-Belsky;
- pigmentation and bran-like peeling after the rash disappears.

Unlike **scarlet** fever, which is characterized by the following:

- the disease occurs with severe intoxication and fever;
- characteristic sore throat on the background of sore throat;
- enlarged and painful only submandibular lymph nodes;
- small-spotted rash, located on a hyperemic background;
- there is an abundant rash on the face with a pale nasolabial triangle;
- "crimson" tongue;
- small-scale peeling in places of rash on the skin (6-9 days);
- tachycardia, possible myocarditis;
- neutrophilic leukocytosis, increased ESR.

Rash, generalized lymphadenopathy may be in infectious mononucleosis. But unlike rubella in **mononucleosis**:

- more pronounced general intoxication syndrome;
- characteristic sore throat, tonsillitis;
- rash occurs on the background of drug therapy (more often);
- lymph nodes are significantly enlarged, sometimes in the form of packages, there is no predominant impression of cervical and occipital lymph nodes;
- there is an increase in the liver and spleen;
- leukocytosis with lympho- and monocytosis, the presence of> 10% of atypical mononuclear cells.

For **enterovirus exanthema**, in contrast to rubella is characterized by:

- mostly summer-autumn seasonality;
- acute onset, intoxication with fever up to 39 ° C and above;
- later appearance of the rash (2nd - 3rd day);
- absence of catarrhal phenomena, conjunctivitis;

- no lymphadenopathy;
- diarrhea, herpangina, myalgia, meningeal signs;
- no significant changes in the hemogram.

At **drug toxic exanthemas**:

- a clear link between taking the drug and the appearance of a rash;
- rash more often in the form of zones of hyperemia, although variations are possible;
- often itchy skin;
- there are "spills" on the background of the drug;
- generalized lymphadenopathy is often absent;

- the duration of the course is determined by the duration of the drug.

With regional lymphadenitis, the rash is **felinosis (cat scratch disease)**. But at the same time:

- there is a primary affect;
- after 10-20 days, mainly inguinal, popliteal lymph nodes increase (on the side where the scratches were applied);
- enlargement of the nodes is quite significant (up to 3-5 cm), can fester;
- rash (scarlet fever, rubella) appears in 1-6 weeks after lymph node enlargement;
- characteristic leukocytosis, increased ESR.

In addition, there are many other diseases that have a course with rashes and lymphadenitis (AIDS, toxoplasmosis, listeriosis, brucellosis, leukemia, etc.).

Complication

Rare. The most serious complication of rubella is encephalitis. Encephalitis occurs during or after a rash. It begins acutely with fever, sometimes against the background of normal temperature with signs of disturbance of consciousness, generalized clonic-tonic convulsions, focal symptoms. The most threatening are central disorders of the heart and breathing. Often the meninges are involved in the process - meningoencephalitis develops. The prognosis of lesions of the central nervous system in the case of rubella is serious - in 20-35% of cases fatalities, and in 30% of children who relapsed, note the residual effects of the nervous system (paresis, paralysis, convulsive syndrome and others).

Lesions of the internal organs are rarely noted. Girls and women may develop arthritis, which is clinically manifested by pain, redness, swelling of the joints. The metacarpophalangeal joints of the fingers are more often affected, followed by the knees and elbows. Symptoms of joint damage usually appear a week after the rash appears and disappear within the next week. The course of arthritis and synovitis is short and favorable. Some authors report the possibility of chronic rubella arthropathy. Testalgia is possible in school-age boys.

Thrombocytopenia rarely develops. Decreased platelet count in combination with the appearance of peripheral small capillaries may cause hemorrhagic syndrome (nasal and gingival bleeding, hematuria), but they are not threatening, although the development of thrombocytopenic purpura is possible.

Otitis, pneumonia, nephritis are also possible.

Treatment

Treatment of patients with uncomplicated course of acquired rubella is carried out at home:

- bed rest during the acute period;
- general hygienic measures;
- frequent ventilation of premises;
- symptomatic therapy: antipyretics with fever (paracetamol, ibuprofen), etc.

Treatment of patients with congenital rubella - depending on the nature of the main clinical syndromes in a specialized hospital in an isolated ward.

Prevention

General prevention is to identify patients, isolate them and treat them. Patients, if possible, are isolated at home for 5 days after the rash, sometimes it is advisable to extend the period. Disinfection is not performed. The contacts are quarantined for 21 days. Pregnant women who have not had rubella before should be protected from contact with patients for at least 3 weeks. At disease of women in the first trimester of pregnancy abortion is shown (in the absence at it in blood of antibodies to a rubella virus)

Specific prevention is vaccination. Currently, various types of vaccines have been developed, including a live trivalent vaccine (against measles, rubella and mumps), with which the WHO plans to deal with this widespread and dangerous "childhood" infection in the coming years through mass vaccination. Due to viremia, vaccination of pregnant women is contraindicated, moreover, after vaccination, a woman should avoid pregnancy for at least 3 months.

In Ukraine, according to the vaccination calendar, specific prevention of rubella is carried out with a live vaccine at the age of 12 months, followed by revaccination at 6 and 15 years (girls). Vaccination can be carried out as a monovaccine and trivaccine (measles, rubella, mumps).

Test questions (rubella)

1. Define rubella.
2. What causes rubella?
3. What is the epidemiology of rubella?
4. Pathogenesis of rubella.
5. Classification of rubella.
6. Diagnosis of rubella.
7. What ELISA results indicate congenital rubella?
8. What changes in the general analysis of blood are characteristic of rubella?
9. How long does it take to secrete the virus in congenital rubella?
10. Clinic of typical acquired rubella?
11. In what period of pregnancy rubella is most dangerous for the unborn child?
12. What developmental abnormalities are characteristic of congenital rubella?
13. Treatment of rubella.
14. Differential diagnosis of rubella.
15. Prevention of rubella.

9. MENINGOCOCCAL INFECTION

Meningococcal infection- is an acute anthroponotic infectious disease with airborne transmission mechanism caused by meningococcus, with a variety of localization and severity of the clinical picture - from localized forms - nasopharyngitis, to generalized forms such as meningococcemia, meningitis and encephalitis. organs and systems.

Etiology

The causative agent of meningococcal infection - meningococcus, belongs to the family Neisseriaceae, Neisseria. The genus Neisseria includes two species of pathogenic microorganisms: N. meningitidis and N. gonorrhoeae, N. meningitidis has 13 serogroups - A, B, C, D, X, Y, Z, 29E, W-135, H, I, K, L , which differ in the composition of a specific capsular polysaccharide. The greatest role in human pathology is played by meningococci of groups A, B, C, Y. Meningococci have a round shape. Occur in the form of diplococci, gram-negative. Pathogenic and virulent properties of meningococci are due to the presence of endotoxin, allergenic substance, SIgA protease, capsules and cilia. Endotoxin has a multifactorial damaging effect - disorders in the blood coagulation and complement system, drop in vascular tone, pyrogenic effect, etc., it also has pronounced sensitizing properties due to the presence of an allergenic substance. Endotoxin often leads to severe vascular damage with the formation of hemorrhages. Meningococci cross the blood-brain barrier. Meningococci have low resistance to various environmental factors. The optimum temperature of their existence is about + 37ºC, outside the human body they die in 30 minutes. Disinfectants, low temperatures, direct sunlight, ultraviolet rays, high temperatures have a detrimental effect on the pathogen. The influence of environmental factors on meningococci is manifested in their tendency to variability and the formation of α-forms. The latter on media that do not contain antibiotics are easily reversed to the original bacterial form. Meningococci are able to develop resistance to chemotherapy. Sulfanilamides are ineffective in the treatment of meningococcal infection, preference in treatment should be given to antibiotics.

Epidemiology

The source of infection is a patient with a clinically manifest or asymptomatic (carrier of meningococcus) form of meningococcal infection. The route of transmission of meningococcal infection is airborne. Prolonged contact at close range (up to 0.5 m) and crowding indoors contribute to the infection. The seasonality of the disease is also characteristic - January-April. The susceptibility to meningococcal infection varies in different age groups. Newborns do not get sick until 6-10 months due to transplacental immunity. Young children get sick most often. Boys get sick more often and more severely. In cases where meningococcus is introduced into an isolated group, whose members have not previously encountered this pathogen, age differences in the frequency of the disease are almost absent. The contagiousness index is 10-15%. There is a familial predisposition to meningococcal infection. The ratio of patients with generalized form and carriers is 1: 180-200 (and even 1: 2000). Frequency of outbreaks in 10-25 years. After the transferred disease there is a steady type-specific immunity. Recurrent diseases are very rare.

Pathogenesis

The entrance gate for meningococcus is the mucous membrane of the upper respiratory tract, often the nasopharynx.

In most cases, the presence of the pathogen on the surface of the mucous membrane is not accompanied by clinical manifestations, this condition is regarded as meningococcal. This form of meningococcal infection accounts for 85-90% of all cases. However, it is an infectious process, it is accompanied by the activation of local protective reactions (secretory immunoglobulins, interferon, lysozyme, etc.), the end result of which is the purification of the mucous membrane from microorganisms. However, already in the process of carrier increases, albeit slowly, the level of specific antibodies in the serum, due to the penetration of pathogens and their antigens into the bloodstream in small quantities that can not cause disease.

In order for the disease to occur, the pathogen must penetrate the submucosal layer by overcoming local barriers of protection. This is accompanied by the development of a local inflammatory reaction. The picture of nasopharyngitis develops. At this stage, the development of the pathological process may stop.

The development of the generalized process is based on a variety of factors associated with both meningococcus and the characteristics of the human body.

Meningococci can enter the bloodstream directly from meningococcal-containing macrophages or lymphogenically. Mass entry of the pathogen into the blood and its mass death due to the bactericidal action of blood, accompanied by the release of endotoxin, leads to the development of disorders of homeostasis.

Direct and indirect action of endotoxin on blood vessels is manifested in dystrophic and necrotic damage to their walls, increased permeability. The consequence of this generalized process is a violation of the blood supply and functions of all vital organs. Hemorrhages, in this process they are visible on the skin, occur in almost all organs. Rashes are bacterial thrombi with perivascular vascular lesions and extravasations.

With meningococcal disease there are coagulation disorders of varying degrees, up to the development of DIC syndrome, which further exacerbates the dysfunction of all organs and systems. In the first phase of DIC syndrome (hypercoagulation stage) there is an activation of coagulation factors, resulting in the formation of blood clots in small vessels. In the future, due to the active consumption of coagulation factors, there is a deficiency of them - a hypocoagulation stage develops with a predominance of anticoagulant factors. Bleeding that occurs can even be the cause of death. Thus, in DIC syndrome, thrombosis is combined with bleeding.

Leading in the pathogenesis of the fulminant (lightning) form is infectious-toxic shock (ITS), which is caused by massive bacteremia and toxemia. Endotoxin shock leads to impaired hemodynamics, microcirculation, DIC syndrome, to profound metabolic disorders. In turn, they activate secondary pathogenetic factors, cause shock progression and disorders of vital organs and systems.

Of particular interest is the lesion of the adrenal glands in the most acute meningococcal sepsis. They are so common that many researchers have considered hemodynamic disorders to develop as a consequence of acute adrenal insufficiency resulting from massive necrosis and hemorrhage into the adrenal glands. However,

not all ITS deaths showed significant adrenal damage and cortisol deficiency, and doses of hormones prescribed to shock patients should be several times higher than those required for replacement therapy. However, hemorrhage into the adrenal glands is the development of severe acute adrenal insufficiency (Watergaug-Friedrichsen syndrome), certainly exacerbates the severity of the condition and vascular collapse. Acute adrenal insufficiency is accompanied by a decrease in blood pressure, blood clotting, electrolyte imbalance, nitrogen retention and other severe manifestations.

The rate of development and extent of lesions occurring in the first hours of the disease, hemorrhagic syndrome with hemorrhage into the mucous membranes, adrenal infarction bring the pathogenesis of lightning meningococcemia with the phenomenon of Schwartzman-Sanarelli, which is believed to be the basis of senses.

Against the background of generalization of meningococcal infection, pathogens can enter various organs with the formation of foci of inflammation in them and the appearance of characteristic clinical symptoms. Such organ lesions can occur both on the background of typical meningococcemia and atypical (without skin rashes), which is reflected in some classifications as independent clinical forms.

The reason for the development of such lesions as arthritis, nephritis, pericarditis, episcleritis, vasculitis can be not only meningococci themselves, but also immune complexes.

CNS damage occurs not only due to intoxication (toxic damage to blood vessels with increased permeability, which leads to swelling of the meninges and the development of meningism). Meningococcus may penetrate the blood-brain barrier with the development of purulent inflammation. But hematogenous is not the only way for meningococcus. It can enter the CNS from the primary site of inflammation (nasopharynx) through the lattice bone and vaginal nerves. Meningococcus, getting on the soft meninges, causes their swelling, and then serous-purulent inflammatory process. From the meninges of the brain, the inflammatory process can spread to the meninges of the spinal cord, cranial nerves. Particularly dangerous involvement in the process of stem formations in the bottom of the IV ventricle of the brain, which leads to respiratory and cardiac disorders. Meningitis, meningoencephalitis can be combined with meningococcemia.

Immunity after the disease persists for several years, its stress is determined by the state of the patient's immune system, the clinical form of the disease, the serogroup of meningococcus.

Classification

I. Localized forms:
- meningococcosis;
- nasopharyngitis.

II. Generalized forms:
- meningitis;
- meningoencephalitis;
- typical meningococcemia (with hemorrhagic skin rashes);
- atypical meningococcemia (without hemorrhagic skin rashes).

Both of these forms can run:

a) without metastases in the internal organs;

b) with metastases in the internal organs (myocarditis, pericarditis, pneumonia, arthritis, etc.).

- combined forms.

The course of meningococcal infection by severity is:

- easy;
- medium severity;
- heavy;
- very severe (hypertoxic, fulminant form).

The duration of acute (up to 3 months), prolonged (more than 3 months) and chronic (more than 6 months) course of meningococcal infection.

Clinic

The incubation period is 2-10 days.

Meningococcosis has no clinical manifestations. The diagnosis is established on the basis of isolation of the pathogen from the nasopharynx and (or) retrospectively by increasing the titer of specific antibodies.

Meningococcal nasopharyngitis

Clinical manifestations depend on the severity of the course. Body temperature can be normal, subfebrile and febrile. The fever lasts 1-3 days. The child complains of headache, sore throat and sore throat, nasal congestion, mucopurulent discharge from the nose. The mucous membrane of the posterior wall of the pharynx is hyperemic with follicular hypertrophy and purulent "track", the mucous membrane of the nose is also hyperemic. Herpetic rashes may appear on the lips. Lethargy, lethargy, pallor are noted. Peripheral blood may have moderate neutrophilic leukocytosis. In 50% of patients the blood picture does not change. It is almost impossible to diagnose meningococcal nasopharyngitis on the basis of the clinical picture.

Epidemiological anamnesis and bacteriological examination of nasopharyngeal mucus help. Nasopharyngitis is often the beginning of a generalized form. In the center of the infection it is necessary to examine all contacts, conduct a laboratory examination for the presence of the pathogen, isolate children with manifestations of nasopharyngitis and prescribe treatment.

Meningococcal meningitis (meningoencephalitis)

In young children, the first signs are convulsions. Meningitis begins suddenly, in the midst of complete well-being, parents can name the time when the child became ill.

One of the first symptoms is a persistent stabbing headache, often it occurs simultaneously with a rise in temperature to 39-400C. Vomiting (often repeated, which does not bring relief), hyperesthesia. Typical posture of the patient: lying on his side with his head tilted back and knees brought to the abdomen. Focal symptoms may appear - impressions of III, V, VI, VII, VIII pairs of cranial nerves. Meningeal signs (symptoms of Kernig, Brudzinski, occipital muscle rigidity) can already be detected in the first hours of the disease. Typical meningeal signs are not typical for infants. They have tension (protrusion) of the umbilicus, a symptom of suspension

(Lesage). In some cases, infants develop cerebral hypotension, which is accompanied by toxic-exicosis, intestinal dysfunction. Cerebral hypotension may be the result of excessive dehydration, administration of large doses of the potassium salt of benzylpenicillin.

Ependymatitis can sometimes develop. The reason is inadequate treatment. Against the background of normal temperature and normalization of the cerebrospinal fluid, the child's condition worsens: disturbance of consciousness, drowsiness, sometimes agitation, vomiting, "brain" crying, explosion of the umbilicus, divergence of the skull sutures. Then there are cramps, paresis and paralysis of the muscles of the neck, sphincters. Characteristic posture - legs crossed at the shins and extended, toes clenched into fists. Without adequate therapy, cachexia and severe mental disorders occur rapidly. The cerebrospinal fluid is characterized by high protein content and xanthochromia, the number of cells is normal.

Changes in the cerebrospinal fluid are of great importance for the diagnosis of meningitis. On the first day of illness, the cerebrospinal fluid may be clear. Very quickly it becomes cloudy, purulent due to neutrophils. The number of cells reaches several thousand in 1 μl. The amount of protein is increased, and sugar and chlorides are reduced. Sanitized cerebrospinal fluid is considered when 1 μl contains up to 100 lymphocytes.

Meningococcal meningococcemia.

Meningococcemia is one of the most severe forms of meningococcal infection. More often the disease occurs acutely, suddenly: the temperature rises to 39-400C, there is a general weakness, headache, muscle pain in the back and extremities, thirst, paleness, agitation, shortness of breath, tachycardia. After 4-6 hours (no later than the second day) there is a rash of hemorrhagic nature, which is localized mainly on the buttocks, thighs, legs, torso. Later, superficial and deeper necrosis appears in the center of the rash. Subsequently, necrotic masses are rejected with the formation of scars. Possible necrosis and dry gangrene of the ears, nose, phalanges of the fingers and even the hands and feet. The elements of the rash have an irregular star shape, ranging in size from small petechiae to large ecchymoses, dense to the touch, slightly protruding above the skin surface.

In the initial period of the disease with hemorrhagic rash may be combined with roseola-papular elements, but they quickly (after 1 day) disappear without a trace.

Hemorrhages on the mucous membrane of the oral cavity, sclera, conjunctiva often appear simultaneously with skin rashes, and more often - before them. With iridocyclochorioiditis, the iris becomes rusty. More severe eye lesions, up to panophthalmitis, are rare.

At patients with a meningococcemia defeat of joints, mainly small is observed. The skin on them is hyperemic, swollen, limited movements, painful. Their course is benign, the function is restored completely.

Sometimes meningococcemia is pleurisy, arthritis, thrombophlebitis, endo-, myo-, and pericarditis. Renal pathology can manifest itself in the form of glomerulonephritis, pyelitis and acute renal failure.

Meningococcemia may be accompanied by the development of ITS, the clinical manifestations of which in various infectious diseases are fundamentally the same. ITS, which develops in meningococcal disease, is characterized mostly by a rapid increase in symptoms: a rapid change in the stages of compensated, subcompensated and decompensated degrees of shock.

In the case of a rapid increase in clinical symptoms of meningococcemia (acute or sudden onset, a typical rash with a significant increase in body temperature) and progression of ITS develops a lightning form of meningococcal infection - subacute meningococcal sepsis, which can be fatal in the first 1-2 days. This malignant (fulminant) form of meningococcal infection mostly develops in children from 6 months. up to 2 years, the anamnesis of which is not burdened by previous diseases. Often these are children who are ahead of their peers in physical development and are overweight.

Meningococcemia may be accompanied by meningitis (meningoencephalitis). In such cases, a combination of symptoms of meningococcemia with symptoms of meningitis is observed.

Atypical meningococcemia.

Generalization of meningococcal infection can occur without skin rashes, but with damage to internal organs. At the same time the signs of defeat of separate bodies and systems (meningococcal myocarditis, endocarditis, endocarditis, pneumonia) with the corresponding symptomatology come to the fore.

Complication:

- ITS;
- acute renal failure;
- ICE syndrome;
- acute adrenal insufficiency (Waterhaus-Friedrichsen syndrome);
- pulmonary edema.

Diagnosis

For laboratory confirmation of the diagnosis, the isolation of the pathogen from the nasopharynx, blood, cerebrospinal fluid is crucial. The rapid method of diagnosing meningococcal infection is bacterioscopy of a thick drop of blood. Results can be obtained immediately. Serological confirmation can be obtained from RNGA with meningococcal erythrocyte diagnosticum serogroups A, B, C. Blood is examined in the dynamics with an interval of 5-7 days. In the diagnosis of meningococcal meningitis is crucial to study the cerebrospinal fluid. The general analysis of blood will be characterized by changes, as for any bacterial infection. In the analysis of urine there are traces of protein, single erythrocytes, cylinders, ie changes that indicate toxic irritation of the kidneys.

Given the instability of meningococcus in the environment, before sending to the laboratory cerebrospinal fluid or other test material should be stored in a thermostat at a temperature of + 370C, but not more than 2-3 hours.

Differential diagnosis

Meningococcal nasopharyngitis has to be differentiated from various lesions of the nasopharynx of viral, bacterial, allergic nature.

In ***SARS,*** the nature of nasal discharge is watery, serous, and in meningococcal infection - purulent, mucopurulent from the first day of the disease. In SARS there are no purulent "tracks" characteristic of meningococcal nasopharyngitis. The blood test will also show changes characteristic of viral infections - leukopenia, lymphocytosis, normal ESR.

Sinusitis differs from meningococcal nasopharyngitis mainly by lesions of one sinus (hence unilateral purulent discharge from the nose), long-term stable course, and a characteristic radiological picture.

Diphtheria of the nasopharynx from meningococcal nasopharyngitis is distinguished by the presence of mucous-bloody secretions from the nose, the formation of rashes and crusts on the mucous membrane at the entrance to the nose; whitish plaques on the nasal mucosa, pharynx, which are difficult to remove and after their removal the bleeding surface is exposed.

Serological confirmation can be obtained from RNGA with diagnostics of meningococcal erythrocyte serogroups A, B, C. Blood is examined in the dynamics within 5-7 days. In the diagnosis of meningococcal meningitis is crucial to study the cerebrospinal fluid. The general analysis of blood will be characterized by changes, as for any bacterial infection. In the analysis of urine, there are traces of protein, single erythrocytes, cylinders, changes that indicate toxic irritation of the kidneys.

Talking about the instability of meningococcus in the environment, before sending cerebrospinal fluid to the laboratory test material should be stored in a thermostat at a temperature of + 37^0C, but not more than 2-3 hours.

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Meningococcemia should be differentiated primarily with diseases whose main symptoms are intoxication and rash.

Scarlet fever is distinguished from meningococcal disease:

- "burning throat", "raspberry tongue";
- abundant small-spot rash on the hyperemic background of the skin, which covers the face, torso, limbs; especially abundant rash in places of natural folds;
- the presence of a pale nasolabial triangle;
- tonsillitis.

Chickenpox from meningococcal disease differs primarily like the rash and its clear dynamics: spot → papule → vesicle → crust.

In infectious mononucleosis, the rash is polymorphic, even hemorrhagic. However, infectious mononucleosis often has angina, characterized by generalized lymphadenopathy, hepatosplenomegaly, and blood tests reveal atypical mononuclear cells on the background of lymphomonocytosis.

Typhoid fever can also be a hemorrhagic rash in combination with severe intoxication. But with typhus, there will be a late appearance of a rash (3-5 days), other localization. A short-term decrease in temperature before the appearance of the rash. Hepatosplenomegaly. The characteristic appearance of the patient is "red eyes on a red face".

Leptospirosis, as well as meningococcemia, is also characterized by severe intoxication and abundant hemorrhagic rash. But in contrast with meningococcemia leptospirosis, it is also characterized by severe myalgia with a predominant localization of pain in the calf muscles; kidney damage is manifested by hematuria with subsequent development of ARF; redness and puffiness of the face, hepatorenal syndrome; arthralgia, and in the hemogram progressive anaemia, sharply increased ESR.

Thrombocytopenic purpura is distinguished from meningococcemia mainly by gradual onset, the absence of intoxication; long chronic course; frequent bleeding, especially from the mucous membranes of the oral cavity; localization of the rash mainly on the skin of the torso and flexor surfaces of the upper extremities; frequent enlargement of the spleen; severe hypochromic anaemia, thrombocytopenia.

Hemorrhagic vasculitis can be accompanied by fever, rash (sometimes hemorrhagic), and arthritis. Hemorrhagic vasculitis lasts longer, the rash is mostly papular, urticarial; placed symmetrically, mainly on the extensor surface of the limbs, around the joints; possible enlargement of the spleen; sometimes there are haemorrhages in a cover and substance of a brain with the corresponding changes in cerebrospinal fluid.

Meningococcal meningitis

Meningococcal meningitis can be distinguished from other meningitis only by changes in the cerebrospinal fluid. It should be known that before the puncture, the patient should be examined by an ophthalmologist: in the case of a stagnant nipple of the optic nerve, it should be temporarily refrained from by prescribing dehydration therapy first. If it is still necessary to carry out the vital signs, the cerebrospinal fluid should be released in infrequent drops, under the cover of a mandrel to avoid wedging.

Treatment

Treatment of nasopharyngitis:

1. Antibacterial therapy: rifampicin 10 mg/day for 3-5 days, or macrolides (erythromycin, spiramycin, azithromycin), or chloramphenicol.

2. Local means: UFO, gargling with disinfectants.

Treatment of meningococcal purulent meningitis

Prehospital stage of treatment:

1. Providing venous access
2. Antibacterial therapy - chloramphenicol sodium succinate 25 mg / kg (single dose) IV
3. Glucocorticoids 1-3 mg / kg of prednisolone
4. Infusion therapy with saline and colloidal solutions
5. Antipyretics
6. Furosemide - 1-2 mg / kg
7. For convulsions – diazepam

Inpatient treatment:

1. Antibacterial therapy: benzylpenicillin 300000-500000 IU / kg / day of administration every 4 hours. Reserve antibiotics: ceftriaxone 100 mg / kg / day, cefotaxime - 200 mg / kg / day. In the presence of ITS - chloramphenicol sodium succinate 100 mg / kg / day.

Treatment of meningococcemia

Prehospital stage of treatment:

1. Providing venous access
2. Antibacterial therapy - chloramphenicol sodium succinate 25 mg/kg (single dose) IV
3. Glucocorticoids - prednisolone, hydrocortisone or dexazone 2-3 mg/kg on prednisolone - without ITS, 5 mg/kg - at ITS of the I degree, 10 mg/kg at ITS of the II degree, 15-20 mg/kg at ITS of the III degree degree
4. Infusion therapy with saline or rheopolyglucin to stabilize BCC.
5. Inotropics (dopamine) - to support hemodynamics.

Inpatient treatment:

1. Depending on the severity of hospitalization in the intensive care unit and resuscitation, or the department of neuro infection of an infectious hospital.
2. Antibacterial therapy: in the presence of ITS drug of choice - chloramphenicol succinate at a dose of 100 mg / kg / day, when removing a patient with ITS prescribe penicillin 200 mg / kg / day, or third-generation cephalosporins - cefotaxime 100-200 mg / kg / day , ceftriaxone 100 mg / kg / day. In severe form and the need to protect against nosocomial infection additionally used aminoglycosides of the 3rd generation - amikacin up to 20 mg/kg/day, netilmicin 1.5-2 mg/kg every 8 hours.
3. Detoxification therapy in moderate forms is performed with glucose-salt solutions, taking into account the daily fluid requirements and pathological costs.
4. Post-syndrome therapy is carried out under the existing syndromes, their treatment is carried out according to the relevant treatment protocols.

Prevention

General prevention is reduced to timely detection, isolation and treatment of patients; carrying out activities in the centre, which includes current disinfection. All contact persons are subject to bacteriological examination of mucus from the nasopharynx to meningococcus (children - 2 times within 3 days, adults - 1 time).

Specific prevention is vaccination. There are monovaccines (A and C), bivalent (A + C), quarter vaccines (A + C + Y + W135).

The vaccine is administered once intravenously, the dose depends on age. A - the vaccine protects for 2 years, C - for 2-4 years.

For convalescents of generalized forms of infection, dispensary supervision is established: after meningococcemia - 6 months, after meningitis and meningoencephalitis - for a year and, if necessary, a neurologist, psychiatrist, ophthalmologist. In the first half of the year, convalescents are examined once every 3 months, in the second - once every six months.

Test questions:

1. etiology of meningococcal infection;
2. epidemiology;
3. pathogenesis;
4. classification;
5. clinic of localized forms;
6. clinic of generalized forms;
7. diagnosis;
8. differential diagnosis;
9. treatment of nasopharyngitis;
10. treatment of meningitis of meningococcal etiology;
11. treatment of meningococcemia;
12. prevention of meningococcal infection.

10. SHINGLES

Shingle is an acute infectious disease that develops mainly due to the activation of the chickenpox virus in the body under conditions of impaired immunological homeostasis, sometimes - due to exogenous infection. It is characterized by an inflammatory process of a limited area of skin (corresponding mucous membranes) with the appearance of grouped spotty-papular-vesicular elements of the rash, which develops against the background of previous local neuralgic pain in the affected area.

Aetiology

Taking about epidemiological observations and virological studies, the identity of shingles and chickenpox viruses is established.

Epidemiology

The source of infection can be both a patient with chickenpox and a patient with shingles. After the initial infection caused by Varicella Zoster, it may become latent, in which the virus persists in the nerve cells of the intervertebral ganglia. T-lymphocytes stimulate virus reactivation. Due to the weakening of T-cell immunity (severe somatic diseases, malignant neoplasms, immunosuppressive therapy, HIV

infection), the development of shingles is possible. And yet shingles is mostly an adult infection.

The route of transmission is airborne, the contagiousness is lower than chickenpox. The disease occurs more often in the cold season and is characterized by sporadic.

Pathogenesis

Herpes zoster virus is neurodermatotropic. Necrotic and degenerative processes develop in the spinal nerve ganglia, roots of sensory nerves, horns of the spinal cord. Electron microscopy reveals intranuclear inclusions and viruses in ganglion cells and adjacent cells, which is characteristic of the persistence of the virus. VZV can affect the brain substance, with changes characteristic of encephalitis: areas of necrosis, perivascular oedema, petechial haemorrhage.

Various factors that reduce the body can contribute to the activation of VZV. This may be due to new infectious diseases, or overheating, hypothermia, ultraviolet radiation, radiation, stress, hormonal disorders, severe diseases (leukaemia, malignancies, taking certain medications (glucocorticosteroids, cytostatics, and immunosuppressants).

Activation of infection is accompanied by degenerative changes
nerve ganglion cells, Schwann cells, nerve fibres,
infiltration of tissues by lymphocytes and polynuclear cells, stasis in
blood vessels.

On nerve trunks, VZV gets into the skin or mucous membranes where there are herpetic rashes along the course of these trunks. Viruses from destroyed cells in the period of exacerbation enter the bloodstream, stimulating both specific immunity and the development of allergic processes. In shingles, in contrast to chickenpox, come to the fore not so much epitheliotropic as neurotropic properties of the virus.

Factors of humoral and cellular immunity take part in the process of clinical recovery. In this case, a more important role is played by cellular immunity. Antibodies enhance the killer function of T lymphocytes to destroy infected cells. Shingle is a marker of immunosuppression.

Classification

Form.
- localized;
- disseminated.

Phase:
- active;
- inactive.

By severity:
- light forms;
- moderate;
- severe forms (bullous, hemorrhagic, gangrenous).

By localization:
- spinal form (cervical, thoracic, lumbosacral);
- craniocerebral.

Clinic

It is almost impossible to determine the incubation period from the moment of virus activation in the nerve ganglia to the beginning of clinical manifestations of the disease (according to various sources from one week to several years).

The onset of the disease is acute: there is fever, general weakness, headache, nausea, and sometimes vomiting. Even before the rash, there is pain along the affected nerve, and in the corresponding area of skin innervation - a burning sensation, itching, pain, usually intense, is exacerbated by changes in body position. Then in places of future rashes, there is infiltration, hyperemia, and on the 3-5th day (sometimes later) there is also a rash.

Rashes with shingles are characterized by the following features:

- they are located on the skin in groups along the nerve trunks;
- from the moment of the rash the pain becomes less;
- Filling lasts 1-2 weeks in the form of separate groups, which merge and can affect large areas of skin;
- the process is unilateral, wherever it is localized (on the skin of the left or right half of the head and face, on one side of the neck, along one limb, etc.).

As a rule, regional lymph nodes increase and become sensitive. With shingles may damage the mucous membranes of the mouth, nose, conjunctiva, bladder, vagina, rectum.

In the area of nerve damage, there are changes in skin temperature, disorders of pain and tactile sensitivity, muscle paresis. In some cases, possible damage to internal organs: lungs, heart, digestive tract.

Variants of the clinical course of shingles

Localization of the lesion	Localization of the lesion	Clinical symptoms, in addition to skin lesions, in the area of the dermatome
Trigeminal nerve	30-90	Conjunctivitis, keratitis, iridocyclitis, visual neuritis and oculomotor nerve, stomatitis
Cervical-thoracic segments CV-TVII	40-50	Flabby paresis of the hands, acute myelopathy
Thoracic segments TVII-XII	Rarely	Paresis of the abdominal muscles
Lumbosacral segments	10- 15	Flabby paresis of the legs, urinary disordersintestinal obstruction
The shell of the head brain	75-80	Serous meningitis
Brain	0,1-1	Encephalitis
Spinal cord, peripheral nerves	0,1-1	Myelitis, Hyena-Barre syndrome

In the abortive form, only papules on erythematous spots are formed. In the absence of gross immune disturbances, symptoms of a disease regress in 2-3 weeks. At hemorrhagic, gangrenous forms, at the bacterial infection of vesicles, the rash remains within 4-8 weeks and longer.

Complication

The most common complication of shingles is postherpetic neuralgia. In 60% of patients, it persists for 1 month after the disappearance of the rash, in 25% - 3 - 6 months, in 15% - more than 6 months. Pain can occur in 30 days - 6 months after peeling.

Relatively rare complications of shingles are encephalitis and myelitis. These complications occur most often in children under one year, with the generalization of the process and on the background of immunosuppression. The disease usually manifests itself in 1-2 weeks after the appearance of a rash on the skin. The patient has impaired consciousness, convulsions, focal symptoms, urination and defecation disorders, sensitivity disorders. Prolonged residual effects in the form of loss of memory, taste, the smell can remain. Some patients encephalitis is accompanied by psychosis, paresis, lesions of the cranial nerves with subsequent persistent disorders of hearing and vision. Mortality from encephalitis is 5-25% or more.

In addition to these complications of the nervous system in shingles are possible: retrobulbar neuritis, Hyena-Barre syndrome, cranial nerve damage, neurogenic bladder, intestinal obstruction, myositis, granulomatous vasculitis of cerebral vessels with subsequent development. В імуноскомпроментованих пацієнтів можлива дисемінація інфекції з ураженням внутрішніх органів (серця, легенів, печінки, головного мозку). The highest risk of the progressive development of shingles was observed in patients with lymphogranulomatosis and lymphoma. Generalized forms occur in 40% of cases.

Diagnostic methods

Diagnosis of shingles is based on clinical and epidemiological data, it should be remembered that this disease develops under certain conditions (immunodeficiency, comorbidities).

Confirm the diagnosis by detecting a specific viral antigen in the contents of the bubbles by immunofluorescence.

Serological diagnosis is reduced to the finding of an increase in the titer of the virus-neutralizing and complement-binding antibodies in the dynamics of the disease.

Differential diagnosis

Differential diagnosis of ***shingles*** should be performed with the following clinical forms: herpes simplex, eczema, pyoderma, erysipelas.

The main differences between herpes simplex and shingles are: - frequent recurrences;

- the absence of severe pain in the affected areas;
- rashes are usually random, not in the process
nerve, not characterized by unilateral localization of rashes;
- predominant localization of rashes - lip area, wings of the nose.

At eczema and shingles similar localization, the vesicular character of rashes, intoxication can be observed. Distinctive signs of eczema:

- severe itching in the affected skin in the absence of pain;
- there is no connection of localization of rashes along the course of nerve trunks;
- not characteristic asymmetry of lesions;
- no intoxication.

In some cases, with abundant rashes, there are common features of ***pyoderma*** and shingles: the pustular nature of the elements of the rash, the formation of crusts, the connection with immunodeficiency. However, there are significant differences, in particular in pyoderma:

- no vesicular elements;
- there is no connection between rashes (their localization) and innervation;
- not characteristic asymmetry of rashes;
- there is no pronounced pain in the affected area;

Rash, in contrast to shingles, is characterized by a predominance of clinical symptoms of sudden or acute onset of the disease with a rise in body temperature to 39°C and above, often with colds. Without previous neuralgic pain, a local inflammatory process develops with clear outlines, significant redness and infiltration of the skin. Patients with erysipelas may have hollow elements on the surface of the skin in the affected area (bullous form), but they are much larger than that characteristic of shingles blisters. The localization of the inflammatory process in patients with erysipelas is not limited to the area of innervation of a single nerve. Rash as streptococcal infection, in contrast to shingles, is accompanied by neutrophilic leukocytosis, increased ESR.

Treatment

Acyclovir is an effective drug for the treatment of patients with shingles. The dose for children under 1 year is 30 mg/kg 3 times or 1500 mg / m2 3 times intravenously. Etiotropic treatment should be started in the first 72 hours after the onset of the disease and continued for 7 days from the onset of the disease or two days after the appearance of the last elements of the rash. Early administration of acyclovir in shingles reduces the duration of the disease, promotes earlier disappearance of pain, prevents complications and dissemination of the process.

Immunocompetent patients with uncomplicated mild herpes zoster acyclovir can be administered orally at a dose of 80 mg 5 times a day. In addition to acyclovir drugs, you can use the shaft acyclovir and ganciclovir.

In severe generalized and complicated forms of shingles can be slowly (over 12 hours) intravenously administered ribavirin at a dose of 15 mg/kg.

In parallel with antiviral drugs of general action in patients with shingles use antiviral ointments: Oxolinic 0.25%, Tebrofen 0.25-0.5%, Riodoxol 0.258-0.5%, Zovirax (acyclovir) in in the form of ointment or jelly. For aseptic purposes, use an alcoholic 1-2% solution of diamond green or 1-3% aqueous solution of methylene blue. In case of accession of a bacterial infection ointments with antibiotics are applied locally.

If necessary, conduct non-specific detoxification therapy. Analgesics, nonsteroidal anti-inflammatory drugs, tranquillizers are prescribed to relieve pain. Physiotherapeutic methods of influencing the pain syndrome (taking into account the nature of local inflammatory changes) are also used: UV irradiation, UHF, ultrasound, electrophoresis with novocaine, novocaine blockade.

As an isotropic therapy for shingles also uses varicella-zoster immunoglobulin at a dose of 0.2 ml/kg, once. In severe forms, the dose of immunoglobulin can be doubled.

It should be borne in mind that shingles are often combined with haematological and other diseases for which glucocorticosteroid therapy is indicated. In such cases, according to the indications, continue pre-treatment.

To stimulate the regenerative processes in the affected ganglia and nerve fibres, according to the indications, drugs are prescribed that promote the transmission of nerve impulses at the synapses (Proserpine, galantamine); glutamic acid, B vitamins, nonsteroidal anabolic drugs (potassium orotate, etc.), tocopherol acetate. It is advisable to use agents that improve microcirculation: xanthinol nicotinate, trental, until.

Correction of herpes zoster disorders associated with shingles should be performed with caution. A comprehensive immunological examination of the patient with the appointment of adequate immunomodulatory therapy is required.

Prevention (see chickenpox)

Control questions (shingles)

1. Define the clinical form of shingles.
2. What is the causative agent of shingles?
3. Who is the source of shingles infection?
4. What is the mode of transmission characteristic of shingles?
5. Pathogenesis of shingles.
6. What is the classification of this clinical form?
7. What is the clinic of shingles?
8. What are the possible options for the clinical course of shingles?
9. What are the most common complications of shingles?
10. What complications of shingles are relatively rare?
11. What are the methods of diagnosing shingles?
12. With what diseases do you need to differentiate shingles?
13. Treatment of shingles?

11. SCARLET FEVER

Scarlatina (Scarlatina) is an acute anthroponotic infectious disease caused by β-hemolytic streptococcus group A, which has a predominantly airborne transmission mechanism, is characterized by fever, intoxication syndrome, acute tonsillitis with regional lymphadenitis, smallpox, and smallpox. and allergic.

Aetiology

Streptococci - gram-positive microorganisms that have a spherical shape belong to the family Lactobacilli. Depending on the ability to hemolyze erythrocytes,

streptococci are divided into β (complete hemolysis), α (partial hemolysis), and γ (absence of hemolysis). In 1981, Nelson created the most complete classification of streptococci. Based on the precipitation reaction, 21 groups of streptococci were identified, which differ in the carbohydrate component of their shell. Each group is denoted by the Latin letter A, B, C, D, and so on. Of all these groups of streptococci, a special position is occupied by group A, which includes S. pyogenes, β-hemolytic streptococcus. There are 80 serovars of β-hemolytic streptococcus.

Streptococci produce toxins, enzymes, hemolysins. 20 extracellular agents secreted by hemolytic group A streptococci have been identified. Of these, erythrogenic toxins (A, B and C) are of the greatest importance for the clinic. The streptococcal toxin has two fractions - thermolabile and thermostable. The thermolabile fraction of erythrogenic toxin is the most important pathogenic product of hemolytic streptococcus in scarlet fever. In addition to toxic properties, streptococci have enzymes: streptolysins (O and S), diphosphopyridine nucleotidase, streptokinase (A and B), deoxyribonuclease (A, B, C and D), hyaluronidase, proteinase, lipase, esterase and more.

The cell wall of group A streptococci includes M-, T- and R-proteins that have antigenic properties, T-protein is present in all strains of streptococci, on this basis is the typing of streptococci into groups, M-protein provides fixation of the pathogen at the site of adhesion and determines its virulence.

Epidemiology

The source of infection is a sick person and a bacterium. Group A streptococci are constantly found in the oropharynx of healthy people, but only 15-20% of them can cause the disease. Incidence rates depend on the age of the child, climatic conditions, season, concentration and frequency of human contact. A significant role in the spread of infection belongs to patients with mild and atypical forms of streptococcal diseases.

The patient becomes dangerous from the beginning of the disease, the duration of the period of epidemiological danger varies from several days to several weeks (and even months) depending on the quality of antibacterial treatment, nasopharyngeal condition, the possibility of re-infection with new strains of β-hemolytic streptococcus group A. Early use of penicillin promotes the rapid release of the macroorganism from streptococcus: with a uniform course in 7-10 days from the onset of the disease, the child poses virtually no epidemiological danger.

The main route of transmission is airborne. The intensity of the spread of streptococcus increases significantly during coughing, sneezing. This is also facilitated by the presence of dust in the air, close and prolonged contact with the patient. Possible contact and household route of infection through toys, things, consumer goods, as well as through food, mainly dairy.

The lowest incidence rate is registered in children of the first year of life (especially up to 6 months), in the blood of which circulate antibodies that entered the mother through the placenta.

Scarlet fever contagion index - 0.4 (40%).

Children aged 2 - 9 years are most often ill. Seasonality is clearly manifested - the rise in morbidity in the autumn-winter period of the year.

Scarlet fever is unevenly distributed. The highest incidence rates are recorded in countries with cold and temperate climates; in countries with hot climates is rare.

After the disease, the child body develops two types of immunity: antitoxic and antibacterial. Antibacterial immunity has no type specificity, it is homogeneous for all group A streptococci. A feature of antitoxic immunity is its stability and intensity throughout human life. Given that the erythrogenic exotoxin of streptococcus is the main pathogenetic chain of scarlet fever, as well as the features of antitoxic immunity, it can be concluded that scarlet fever occurs once in a lifetime. Antibacterial immunity is unstable, so local streptococcal diseases may recur.

Pathogenesis

The entrance gates are the mucous membranes of the tonsils, sometimes - damaged skin (wound or burn surface), the mucous membranes of the genital tract (in women in labour). In the macroorganism, streptococcus spreads by lymphogenic and hematogenous pathways, through channels and contact with adjacent tissues. Clinical manifestations of the disease are caused by septic, toxic and allergic action of the pathogen (three syndromes of the pathogenesis of streptococcal infection).

Septic (or infectious) syndrome of pathogenesis is characterized by inflammatory or necrotic changes at the site of streptococcal invasion. Inflammation is initially catarrhal but has a tendency to a rapid transition to purulent, purulent-necrotic.

The toxic syndrome is caused mainly by exotoxin, which enters the bloodstream, causes fever and symptoms of intoxication: malaise, small rash, change of throat and tongue, the reaction of regional lymph nodes (in the first 2-3 days of illness), changes in the heart, vascular system. The most pronounced manifestations of the toxic syndrome are observed in toxic forms of scarlet fever. The decreased tone of the sympathetic nervous system, inhibition of the release of corticosteroid hormones on the background of severe CNS damage can lead to a sharp decrease in blood pressure and death from infectious-toxic shock.

Allergic syndrome develops from the first days of scarlet fever, but reaches its peak at 2-3 weeks of illness and persists for a long time. Allergy is mainly specific and is caused by protein substances of streptococcus. It is usually not accompanied by visible clinical manifestations but leads to increased permeability of blood vessel walls, decreased phagocytic activity of leukocytes and other changes. In this regard, there is a risk of complications of infectious-allergic nature (glomerulonephritis, myocarditis, synovitis, rheumatism), which usually develop at 2-3 weeks of illness as a result of infection with other serotypes of streptococcus.

In the pathogenesis of scarlet fever, there is a change in the phases of autonomic nervous activity: at the beginning of the disease, there is an increase in the tone of the sympathetic nervous system ("sympathetic phase"), which is subsequently replaced by the dominance of the tone of the parasympathetic nervous system ("vagus phase").

Pathomorphology

Exudate, exfoliated epithelium and streptococcal accumulations are found in the crypts of the palatine tonsils, and deep necrobiosis and necrosis zones are found in the tissue.

Toxic scarlet fever is characterized by a sharp catarrhal inflammation of the mucous membranes of the tonsils, pharynx and even the oesophagus with superficial necrosis of the epithelium. There is the fatty degeneration of the liver, there may be punctate foci of necrosis. In the spleen, there is slight hyperplasia of the pulp with partial necrosis. There are dystrophic changes in the myocardium, acute swelling and sharp circulatory disorders in the brain.

In the septic form of scarlet fever, deeper necrosis is observed on the palatine tonsils, sometimes on the posterior wall of the pharynx. Large foci of necrosis may be in the regional lymph nodes, purulent tissue melting and the spread of the process is not adjacent subcutaneous tissue with the development of adenophlegmon. Purulent and necrotic foci are also found in various other tissues and organs (joints, kidneys, etc.).

Classification of scarlet fever

By type:

1. Typical.
2. Atypical:
   a) erased;
   b) hypertoxic;
   c) hemorrhagic;
   d) extrapharyngeal (burn, wound, postpartum).

In the septic form of scarlet fever, deeper necrosis is observed on the palatine tonsils, sometimes on the posterior wall of the pharynx.

Large foci of necrosis may be in the regional lymph nodes, often there is purulent tissue melting and the spread of the process is not adjacent subcutaneous tissue with the development of adenophlegmon.

Purulent and necrotic foci are also found in various other tissues and organs (joints, kidneys, etc.).

Classification of scarlet fever

By type:

1. Typical.
2. Atypical:
   a) erased;
   b) hypertoxic;
   c) hemorrhagic;
   d) extrapharyngeal (burn, wound, postpartum).

By severity:

1. Light form.
2. Moderate form.
3. Severe form:

- toxic;
- septic;
- toxic-septic.

Severity criteria:
- severity of intoxication syndrome;
- the severity of local changes.

During:
1. Smooth.
2. Complicated:
   a) allergic in nature;
   b) purulent complications.
3. Abortive.

Clinical picture

Typical forms of scarlet fever are characterized by the presence of a primary focus in the throat and the classic signs of the disease. There is a clear cyclical development of scarlet fever with a change of 4 periods: incubation, initial, rash and convalescence. The incubation period varies from 1 to 12 days, often 2-4 days.

Scarlet fever usually begins acutely. Characteristic intoxication, fever, acute tonsillitis with regional lymphadenitis. Intoxication syndrome is manifested by a violation of the general condition, headache, often nausea and vomiting, tachycardia. Body temperature rises to 38°C and above. Acute tonsillitis syndrome is characterized by sore throat (especially when swallowing), bright pink limited hyperemia of the mucous membrane of the soft palate and tonsils, sometimes small dot enanthema on the soft palate, reaction of anterior cervical (tonsillar) lymph nodes (enlargement, moderate compaction and sensitivity on palpation). Tonsillitis is more often catarrhal in nature, but can be lacunar or follicular. Necrotic sore throat is now rare and is a complication.

The period of the rash. Against the background of the maximum severity of the syndromes of the initial period (intoxication, tonsillitis) there is a small rash.

Exanthema syndrome develops in the early stages, usually in the first 2 days of the disease. Morphologically, it is a small roseola 1-2 mm in size, located close to each other. The color of the rash on the first day is bright, sometimes bright red, up to 3-4 days pale to pale pink.

Rash is often quite intense, less often - scanty, localized mainly on the flexion surfaces of the extremities, anterior and lateral surfaces of the neck, lateral surfaces of the chest, abdomen, lumbar region, inner and posterior surfaces of the thighs and legs, in places of natural flexions - axillary, elbow, inguinal, popliteal.

In these areas, the rash is more intense, brighter, located on a hyperemic background of the skin and persists longer. As a result of mechanical trauma to the skin vessels, small petechiae often appear, which are located in isolation or form hemorrhagic stripes (Pastia lines), which remain for some time after the rash disappears and serve as one of the additional signs in the diagnosis of scarlet fever at a later date.

The patient's skin is dry, rough (due to hypertrophy of hair follicles and the action of the sympathetic division of the autonomic nervous system).

Changes of the tongue are typical for scarlet fever. On the first day of the disease it is covered with a white plaque, from the 2nd to the 4-5th day it is gradually cleared, starting from the tip, and becomes bright crimson, with protruding fungal papillae on the cleaned surface ("raspberry tongue").

In the acute period of scarlet fever there is a characteristic appearance of the patient's face: against the background of a bright blush of the cheeks and cherry or crimson color of the lips, there is a pale nasolabial triangle (symptom of Filatov).

Changes in other organs and systems in the acute period of scarlet fever are usually insignificant. Changes in the cardiovascular system may be observed.

Typical for scarlet fever is the course of the sympathetic phase and vagus phase, associated with toxin damage to the autonomic nervous system.

In the first 3-4 days the sympathetic phase is revealed: tachycardia, increase in arterial pressure, dryness of skin, a negative symptom of Ashner, white dermographism slowly appears and quickly disappears, and from 5-6 days there comes a vagus phase: bradycardia, decrease in arterial pressure, sweating, Ashner's symptom is sharply positive, white dermographism quickly appears and slowly disappears.

The development of symptoms of scarlet fever is very fast, they are most pronounced on day 1-2 of the disease. The further course of the disease is characterized by a natural successive attenuation of scarlet fever symptoms. The manifestations of intoxication are the first to weaken, the body temperature in most patients normalizes by 3-5 days of illness. The rash persists for 2 to 6 days (average 4 days). Changes in regional lymph nodes disappear by 4-5 days, the tongue - by the end of the 2nd week of the disease.

The period of convalescence begins in the 2nd week of the disease and lasts 10-14 days. It is characterized by the presence in some patients of peeling skin and "papillary" tongue. Lamellar peeling is typical of scarlet fever, especially on the fingers and toes. Slight bran-like peeling on the skin of the neck, torso, earlobes is possible. During convalescence, hypersensitivity to streptococcal superinfection persists and the associated risk of infectious-allergic and septic complications.

Atypical forms - extratonsillar (burn, wound, postpartum, postoperative).

Extratonsillar scarlet fever differs from the typical form in the absence of complaints of sore throat, inflammatory changes in the oropharynx and reactions of the tonsillar lymph nodes.

The rash has a characteristic scarlet fever morphology and location, but also thickens at the entrance gate (wounds, burns).

Intoxication is moderate or significant, other clinical manifestations do not differ from those in typical scarlet fever.

There are mild, moderate and severe forms of scarlet fever.

Mild form in modern conditions is the most common and is characterized by a mild intoxication syndrome and the presence of catarrhal tonsillitis.The condition of

children remains satisfactory, body temperature does not exceed 37.5-38.50C. There are no complaints, sometimes there is a short-term headache, sore throat when swallowing, possible single vomiting. Small-spot rash is not bright and not intense, fades by 3-4 days of illness.

Moderate form is accompanied by significant intoxication and pronounced changes in the location of the entrance gate. Children complain of weakness, headache, loss of appetite, pain when swallowing. Body temperature rises to 38.6-39.50C, vomiting is usually repeated. In the oropharynx there are phenomena of tonsillitis with bright limited redness, often with purulent effusion in the lacunae, or purulent follicles. Dotted enanthema is sometimes observed on the mucous membranes of the soft palate. The rash is bright, intense, on a hyperemic skin background, persists for 5-6 days. All patients show changes in the cardiovascular system: tachycardia, muffled heart sounds, increased blood pressure.

Severe scarlet fever can occur with severe symptoms of intoxication (toxic form) or septic lesions (septic form). When combining the pronounced initial symptoms of toxicosis and septic manifestations, the form of scarlet fever is regarded as toxic-septic.

Toxic form of scarlet fever is characterized by pronounced symptoms of intoxication. Repeated vomiting, headache, agitation, delirium, loss of consciousness, convulsions are noted. Body temperature rises to 400C and above. Characteristic appearance of the patient's face: a bright blush of the cheeks with a pronounced pale nasolabial triangle, bright dry lips, injection of scleral vessels. The throat is bright, "burning"; hyperemia reaching the border of the soft palate, punctate enanthema of hemorrhagic nature. The rash on the body is bright, on a hyperemic background of the skin, often with hemorrhages. Symptoms of damage to the cardiovascular system are detected at the beginning of the disease- there is a pronounced tachycardia, muffled heart sounds, increased blood pressure.

As the toxicosis increases, sometimes even in the first day, infectious-toxic shock (ITS) may develop: cyanosis, cold extremities, frequent filiform pulse, muffled heart sounds, a sharp drop in blood pressure, oliguria. In the absence of adequate therapy, death occurs on the first day of the disease.

The septic form of scarlet fever is accompanied by the development of severe inflammatory purulent and purulent-necrotic processes arising from the primary source of inflammation. The patient condition is progressively deteriorating. The body temperature increases, sore throat becomes necrotic, and foci of necrosis appear not only on the tonsils, but also in parentheses, at the base of the tongue.

Purulent lymphadenitis of tonsillar lymph nodes with connection to the pathological process of the surrounding tissue (adenophlegmon), purulent otitis, ethmoiditis, mastoiditis develop. In the absence of adequate etiotropic therapy, the disease progresses rapidly, developing a severe septic condition and death as a result.

The course of scarlet fever (by nature) is regarded as smooth, if the patient after normalization of body temperature and disappearance of symptoms of intoxication there are no complications or concomitant diseases that affect the main process.

The non-smooth course of scarlet fever is characterized by the development of complications (specific, nonspecific), exacerbation of chronic diseases and layering of secondary bacterial flora.

Complication

Specific complications of scarlet fever are divided into toxic, infectious (septic) and allergic; by terms of occurrence - early (developing in the 1st week of the disease) and late (occurring in the 2nd week and later).

Toxic complication is infectious-toxic shock, which occurs in the toxic form of scarlet fever.

Septic complications: sore throat - in the early stages only necrotic, in the late -any nature; lymphadenitis - in the early stages of purulent, in the late - of any nature. Common complications are otitis, adenoiditis, paratonsillar abscess, sinusitis, mastoiditis, laryngitis, bronchitis, pneumonia; especially severe - septicemia, meningitis.

Allergic complications of scarlet fever - infectious-allergic myocarditis, glomerulonephritis, rheumatism, synovitis.

Early complications can be toxic and infectious (septic). The reasons for the development of early complications are the lack of antibacterial therapy or improperly performed etiotropic treatment (inappropriate antibiotic, low dose and irregular administration of the drug, a short course and late start of therapy).

Late complications of scarlet fever, mainly infectious-allergic, are caused by specific sensitization by streptococcus, but can also be septic.

An important role in the development of late complications belongs to secondary infection.

Features of scarlet fever in young children. Scarlet fever in children of the first year of life (especially up to 3 months of age) is rare, as they have antitoxic immunity received from the mother. The peculiarity of scarlet fever in patients of this age group is the low severity of the syndrome of intoxication and other initial manifestations of the disease (catarrhal tonsillitis, minor skin rashes). "Raspberry tongue", large-plate peeling is seldom observed. But more often than in older children, there is a smooth course of the disease associated with the layering of SARS and the development of septic complications (purulent otitis, lymphadenitis, septicemia).

Diagnosis

1. general blood test (leukocytosis, neutrophilia, shift of the formula to the left, eosinophilia, increased ESR).

2. bacteriological examination of mucus from the oropharynx (isolation of β-hemolytic streptococcus group A)

3. serological (increase in titers of antistreptolysin O in the dynamics)

Differential diagnosis

Scarlet fever should be differentiated from diseases accompanied by rashes: rubella, pseudotuberculosis, measles, chickenpox, infectious tshamer erythema, pityriasis, toxicoderma.

Distinctive features of rubella from scarlet fever:

- the presence of a rash in the nasolabial triangle;
- significant enlargement and soreness of the occipital and posterior cervical lymph nodes;
- moderate effects of intoxication

- no tonsillitis;
- there is no significant, limited softness of the soft palate;
- in the general analysis of blood the presence of plasma cells and leukopenia with lymphocytosis.

Distinctive features of pseudotuberculosis from scarlet fever:
- the presence of dyspeptic syndrome
- later appearance of a rash on the skin;
- the presence of catarrhal phenomena in the form of nasal congestion, conjunctival hyperemia, scleritis;
- the presence of a symptom of "gloves" and a symptom of "socks"
- enlargement of the liver and spleen;
- location of the rash on a non-hyperemic skin background

Measles distinguishes:
- the presence of bright manifestations of the prodromal period in the form of cough, lacrimation, conjunctivitis, photophobia, rhinitis;
- the presence of Filatov-Koplik spots
- no acute tonsillitis
- staged rash
- the presence of a period of pigmentation
- no bran-like and large-plate peeling

Prodromal scarlet fever rash in chickenpox (rach) has the following differences from scarlet fever:
- no acute tonsillitis
- the absence of a significant symptom of intoxication
- lack of contact with patients with chickenpox

Infectious tshamer erythema differs from scarlet fever:
- proceeds without the expressed phenomena of intoxication with normal or slightly raised body temperature
- no acute tonsillitis
- the presence of a rash on the chin
- the rash can take the form of garlands, and its color from yellow-blue in the center to bright red on the periphery
- there is no peeling of the rash

At differential diagnosis of scarlet fever from pityriasis it is necessary to consider the following signs which characterize the last:
- Rash occurs more often in children under one year
- localized rash mainly on the neck and chest
- no symptoms of intoxication
- no acute tonsillitis
- when cooled, this rash disappears quickly

Toxicoderma has the following features that distinguish it from scarlet fever:
- The appearance of a rash is associated with medication
- There is a rash more often for 3-4 days after taking medication
- positive allergy history of the child
- peeling occurs immediately after the disappearance of the rash, and not for 5-7 days as in scarlet fever

Treatment

Treatment of patients with scarlet fever is complex, etiopathogenetic;
is carried out both in a hospital, and in house conditions.

Hospitalization is carried out according to clinical (severe and moderate forms), age (children under 1 year) and epidemiological (patients from closed groups, living in dormitories, communal apartments, etc.) indicators. Patients are hospitalized in boxes, which are filled simultaneously. Transfer to other boxes and resettlement of other patients is not allowed.

1. Mode - bed rest during the entire acute period of the disease.
2. The diet should be appropriate for the child age and contain all the necessary food ingredients.
3. Etiotropic therapy: antibiotics - in mild form penicillins or macrolides, in moderate - penicillins, in severe - cephalosporins I-II generation, clindamycin, vancomycin. The course of antibacterial therapy - in mild form 10 days, heart and severe 10-14 days, the route of administration - in mild form - orally, in moderate - intramuscularly, in severe - intravenously.
4. Detoxification therapy: in mild form - a significant amount of drinking, in moderate and severe forms - infusion of glucose-saline solutions
5. Antihistamines
6. Drugs that strengthen the vessel wall (ascorbutin, galascorbin)
7. Antipyretic drugs (paracetamol, ibuprofen)
8. Means of local sanitation: gargling with disinfectant solutions, quartz tube, etc.

Prevention

There is currently no specific prevention of group A streptococcal infection.

The main preventive measures are early detection and isolation of the source of infection. Isolation of a patient with scarlet fever is carried out in a hospital or at home. Children are discharged from the hospital not earlier than 10 days after the onset of the disease with a negative result of bacteriological examination for group A streptococcus. Scarlet fever convalescents are not allowed in preschools and the first 2 classes of school for another 12 days. The same isolation period (22 days) is recommended for patients with sore throat from scarlet fever. Impact on transmission routes: daily and final (on the day of registration of recovery) disinfection by parents and staff.

Contact preschoolers and schoolchildren of grades 1-2 are quarantined for 7 days from the moment of isolation of a patient with scarlet fever with the whole complex of anti-epidemiological measures.

Of great importance in the prevention of scarlet fever are measures aimed at organizing the proper placement of children in bedrooms, playrooms, classrooms (dispersal of groups, keeping the distance between beds, desks in accordance with sanitary norms, etc.).

An important role is played by the regulation of air regime in bedrooms, places of mass classes (ventilation, wet cleaning, ultraviolet radiation).

Test questions (scarlet fever)

1. Define the clinical form.
2. What is the causative agent of scapula?
3. Who is the source of scarlet fever?
4. What is the mode of transmission characteristic of scarlet fever?
5. Pathogenesis of scarlet fever.
6. Classification of scarlet fever.
7. What is the clinic of a typical form of scarlet fever?
8. What complications are characteristic of scarlet fever?
9. What are the methods of scarlet fever?
10. With what diseases you need to differentiate scarlet fever?
11. What is the treatment of scarlet fever?

Additions

Calendar of preventive vaccinations in Ukraine

(Order of the Ministry of Health of Ukraine №947 of 04.06.2018)

Age	**Vaccination against**				
1 day		Hepatitis B			
3-5 days	Tuberculosis				
2 month		Hepatitis B	Diphtheria, pertussis, tetanus	Poliomyelitis,	Hemophilic infection
4 months			Diphtheria, pertussis, tetanus	Poliomyelitis,	Hemophilic infection
6 months		Hepatitis B	Diphtheria, pertussis, tetanus	Poliomyelitis	
12 months				Measles, rubella, mumps	Hemophilic infection
18 months			Diphtheria, pertussis, tetanus	Poliomyelitis	
6 years			Diphtheria, tetanus	Poliomyelitis	Measles, rubella, mumps
14 years				Poliomyelitis	
16 years			Diphtheria, tetanus		
Adults			Diphtheria, tetanus		

Recommended vaccination

Vaccination Anti	Recommended
Hepatitis A	-medical workers; -Children and staff of children, preschool institutions. -Personnel of public catering institutions and food industry enterprises involved in preparation (manufacturing) of transportation and sale of food. -Military, employees of the Ministry of Internal Affairs, firefighters, specialized personnel of special designated (operational services), Personnel for maintenance of water treatment facilities. Water supply networks, servicing sewage systems and sewage treatment facilities. -personnel and patients with closed institutions (psychiatric institutions, institutions to maintain mentally retarded persons, etc.); personnel and persons in punishment facilities; -Students of medical educational institutions. -Persons participating in peacekeeping events have been established by humanitarian assistance, etc.; -Persons who use narcotic substances intravenously, HIV-infected people with venereal diseases. -Persons which often change sexual partners, prostitutes, homosexuals; -Persons traveling in regions with high endemic hepatitis A -Persons who communicated with patients with hepatitis A in infection cells: -Patients in hemophilia, on chronic liver diseases of different etiology, including patients with hepatitis B and C and chronic carriers of hepatitis V virus patients with nonspecific hepatitis; -Patients with a compromised immune system.

Hepatitis B.	-Military, employees of the Ministry of Internal Affairs, firefighters, specialized personnel of special designated (operational services), -personnel and patients with closed institutions (psychiatric institutions, institutions to maintain mentally retarded persons, etc.); personnel and persons in punishment facilities; - the personnel of the service sector, which, according to the specifics of their professional activities, may have contact with human biological fluids (hairdressers, staff of beauty salons, masseurs, etc.), as well as those studying in these specialties; -athletes -Persons who use narcotic substances intravenously, HIV-infected people with venereal diseases. -Persons which often change sexual partners, prostitutes, homosexuals; -children who are not subject to compulsory vaccination and individuals aged 20-40 years, primarily women; -patients with chronic and oncological diseases, with chronic hepatic insufficiency; -Persons traveling to regions with high endemic hepatitis B.
Flu	- to all persons for individual protection in accordance with the instructions on the use of vaccines.
Hemophilic type b infection	- all children who have reached 2 months of age. Vaccination is best done simultaneously with vaccination of diphtheria, tetanus, coughing and poliomyelitis.
Pneumococcal infection	- Children elders in 2 years and adults with asplasty. Lymphogranulomatosis, hemoglobinopathies, chronic renal insufficiency, cardiovascular diseases, lung diseases. diseases associated with metabolism, other states with an increased risk of pneumococcal infection (alcoholism, liver cirrhosis, etc.); - adults with violations of immune status and elderly persons, especially those who live in boarding schools; - HIV-infected persons.
An infectious disease for which a vaccine registered in Ukraine	- to all persons who wish to be muted outside the scheme of the calendar of prevention vaccination (Section 1), can be done in medical and prophylactic institutions or other medical facilities with official permission for vaccination.

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