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"NORTH OSSETIAN STATE MEDICAL ACADEMY»
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Department of Infectious diseases

METHODOLOGICAL GUIDE

CHILDREN'S INFECTIOUS DISEASES IN ADULTS

for students studying in the specialty
31.05.01 General medicine (specialty)

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Otaraeva B.I., Plieva Zh.G.,
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Reviewers:

Plakhtiy L.Ya. - Doctor of Medical Sciences, Professor, Head of the Department of Microbiology, State Budgetary Educational Institution of Higher Professional Education SOGMA of the Ministry of Health of Russia

Kusova A. R. - doctor of medical science, Professor, head of Department of General hygiene FGBOU VO SOGMA Ministry of health of Russia

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CLINICAL SIGNIFICANCE OF THE TOPIC

Diseases such as measles, rubella, scarlet fever, chickenpox, and mumps are mainly reported in children. In everyday life, these diseases have acquired the name "children's infections". However, these diseases are also found in adults. Moreover, in recent years, they are increasingly observed in persons over the age of 15, and measles is registered mainly in adults, who sometimes become sources of infection, bringing measles to children's institutions. Children's infections in adults are often more severe than in children, have certain features, sometimes other manifestations, complications. Delayed diagnosis leads to late isolation, delayed detection of complications, the appointment of adequate therapy, increases the number of contacts.

Purpose of the lesson.

The purpose of studying this topic is the early diagnosis of children's infectious diseases in adults and learn the tactics of management and treatment of these patients.

As a result of studying the topic, the student should know:

1. Etiology and epidemiology of diseases.
2. Pathogenesis and pathological anatomy.
3. Clinic.
4. Methods of diagnostics.
5. Treatment.
6. Measures in the focus of infection and prevention issues.

Medical practical skills mastered by students on the topic:

The student should be able to:

1. Correctly to collect the anamnesis of disease and epidemiological history.
2. To conduct an objective study of the patient, to identify the most significant features of each childhood infection.
3. Identify and assess exanthema and enanthema in measles, rubella, scarlet fever, and chickenpox (type of rash, number of elements, localization, timing of appearance, and residual events).
4. Examine the oropharynx and salivary glands to judge the nature of their lesions.
5. Examine patients to identify the most frequent lesions of various organs and systems in these infectious diseases.
6. make a differential diagnosis of childhood infections in adults with diseases that occur with similar clinical symptoms.
7. Identify clinical and epidemiological indications for hospitalization.
8. Conduct sanitary and educational work in the environment of the patient.

VARICELLA

Chickenpox is an acute infectious disease that is accompanied by a papulovesicular rash on the skin and mucous membranes.

ETIOLOGY

The causative agent of chickenpox is a virus from the family of Herpesviridae, known as "strongyloplasma Aragao" in honor of the Brazilian scientist Aragao, who discovered the virus in 1911. The pathogen is a virus of large size (150-200 nm), it can be seen in a conventional light microscope. The varicella-zoster virus is contained in large quantities in the varicose vesicles in the first 3-4 days of the disease, then its number quickly decreases and after the 7th day it cannot be detected.

The virus has a Central core, a protein shell, an outer membrane with ciliated protrusions, contains DNA, and when found in epithelial cells forms intracellular inclusions. It lives and reproduces only in the human body. The main properties of the virus that are important in epidemiology are its volatility and extremely low resistance. In the external environment, the virus quickly dies: in the droplets of mucus, saliva, the virus persists for no more than 10-15 minutes; UV radiation, heating, and sunlight quickly inactivate it.

In the material from vesicles in the presence of anti-denaturing drugs (milk, sucrose) and at-70 ° C, the virus persists for 5 years; in the culture fluid at the same temperature, it dies after 2-3 months.

EPIDEMIOLOGY

Chickenpox is a very contagious disease, with almost 100% susceptibility to it. The source of infection is patients with chicken pox, sometimes shingles. Smallpox patients become infectious no earlier than 20-24 hours before the appearance of exanthema and continue to be dangerous until the 5th day after the appearance of the last element of the rash. The path of transmission is airborne, and the virus is released in huge quantities when coughing, talking and sneezing. A vertical mechanism of transmission of the virus to the fetus from a mother who was ill with smallpox during pregnancy is possible. Some authors believe that if a newborn gets sick before the 10th day of life, that is less than the minimum time of incubation of smallpox, the disease should be regarded as congenital, resulting from in utero transmission. V / smallpox in newborns is characterized by a severe course, sometimes with the appearance of extremely severe visceral forms of a disseminated character.

It is especially dangerous for a newborn if the mother becomes ill with smallpox less than 5 days before delivery. If a woman is ill during the last month of pregnancy, the probability of newborn disease is 25%. The severity and prognosis depend on the timing of the rash, in particular, severe course and fatal outcomes are more likely to occur if the rash in the mother appeared in the last 4 days before childbirth or on the 5-10 th day of life of newborns. The virus from mother to fetus gets through the placenta during viremia in the mother. Born children acquire the so-called chickenpox syndrome, which is expressed in immobility of the limbs with disfiguring scars on the skin, mental disorders and abnormalities of the visual organ. Up to 24% of these newborns die in the first days of life.

The peak incidence occurs in the autumn-winter period. As a result of transferred smallpox virus, post-infectious immunity is formed, which is preserved for life.

PATHOGENESIS

When the virus enters the body, it is initially fixed on the cells of the mucous membranes, it is introduced into them and during the incubation period it multiplies, and then it penetrates into the blood and spreads throughout the body. In infected people, the virus is found in the nasopharynx in the last days of incubation and in the first days of the disease. About viremia is indirectly evidenced by the appearance of a generalized rash, common almost all over the surface of the body. The virus of smallpox, having ectodermally, affects the mucous membranes and skin.

Antiviral protection is provided by cellular immunity, which controls viruses located intracellularly. If this form of immunity is damaged, viral infection is disseminated. V. N. Vertsner et al. (1974), F. I. Nagimova (1991) believe that in the formation of the pathological process in smallpox, the leading role is played by the T-lymphocyte system, the suppression of which is accompanied by a more severe course of the disease and the occurrence in some cases of visceral lesions of internal organs. This statement is supported by the fact that smallpox is much more severe in people who received hormone and radiation therapy, before or during the disease, which often leads to generalization of the process and dissemination of chickenpox rashes in internal organs, in particular in the lungs and liver, as well as in patients older than 70 years. The cessation of anticancer therapy prior to the beginning of smallpox reduced the risk of dissemination of lesions.

In the surface layer of the skin at the site of fixation of the b/smallpox virus, there is a limited expansion of blood capillaries in the form of a spot, serous edema, papules appear, and later, when the epidermis is detached, a vesicle is formed, which in some cases turns into a pustule. As a result of the multiplication of the virus, the accumulation of toxic products, allergic restructuring of the body, fever and other manifestations of an infectious disease develop.

If you experience a pustular rash intoxication, the temperature reaction become more pronounced.

After a b / smallpox infection, there is a persistent lifelong immunity, which, however, may weaken in old age or due to the occurrence of immunosuppression. Immunity of adults is the result of active immunity acquired in childhood, but sometimes smallpox occurs in them as a result of a sharp decrease in the intensity of immunity and re-infection.

PATHOLOGICAL ANATOMY

The pathoanatomic picture in smallpox is insufficiently studied, in addition, with fatal outcomes, it often develops against the background of other diseases. There is information about morphological changes in visceral forms of smallpox. Visceral complications of V/smallpox are rare, are diagnosed mainly on the section and are characterized by a generalized spread in the internal organs, mainly in the

liver, lungs, spleen, kidneys, pancreas of yellowish-white foci of necrosis, which have histological similarity to chicken pox on the skin. Rounded eosinophilic inclusions are detected in the nuclei of necrotised cells.

CLINIC

The incubation period is 10-21 days. Prodromal phenomena in children are expressed little, in adults often marked malaise, lethargy, a sense of exhaustion, headache, decreased appetite and can occur with significant General intoxication (nausea ,sometimes vomiting and abdominal pain), fever. In the vast majority of adults and in all children, the prodromal period lasts 1-2 days. The prodromal period in adults makes it difficult to recognize smallpox and timely isolation of the patient, which has a certain epidemiological significance. B/smallpox fever accompanies the beginning of a mass appearance of a rash and disappears with the end of the rash. The fever usually lasts for 2-5 days, but with abundant and prolonged rashes, it lasts up to 7-8 days. The maximum temperature rise is observed during the period of the greatest rash, especially in cases of pustules. There is no characteristic temperature curve for b/smallpox. Since the rash in b/smallpox occurs periodically, the T-curve can also have a wavy character, accompanying the appearance of new elements of the rash. The severity of fever can be different, and in some patients it may not be present. The febrile reaction in adults is more common and has a longer duration than in children. When prodromal phenomena are absent or poorly expressed, the first sign of the disease is a rash that appears unexpectedly, in the midst of complete health. The rash in V/smallpox occurs not at once, but in 3-5 doses, with intervals of 24-48h., and between the first and last-6-8 days.

The initial elements of the rash can appear on any part of the body: on the stomach, chest, shoulders, thighs, then spreading throughout the body without a specific pattern. In contrast to natural smallpox, with V / smallpox on the face, the rash is less abundant and appears later than on the limbs and trunk. Exanthema on the palms and soles is very rare, but the appearance of a rash on the scalp is quite typical for smallpox. With a mass rash, a thick rash appears, primarily on the hairy surface of the head, torso and to a lesser extent in the inguinal and axillary folds. On day 3-4 of the disease, a rash polymorphism can be observed on the same area of the skin: spots, vesicles, pustules, crusts.

The number of elements of the rash varies and ranges from a single to several hundred. Further rashes are mainly observed on the extremities, but the new elements become smaller and smaller, the number of them decreases, and the rash gradually stops. Rash in smallpox/V / successively passes through a number of stages of development. At first, round or oval red or pink spots appear in the size of a pinhead to a lentil grain (2-4 mm), which after a few hours turn into papules, and then some of them – into vesicles filled with transparent content. Vesicles have a single-chamber structure, very tender and surrounded by a Corolla of redness. Soon, the transparent contents of the bubbles become cloudy, and after 2 days, the vesicles begin to dry out and surface crusts of yellowish, dark red or brownish color are formed, which on the 6-8 th day fall off, leaving no scars in most cases. The time for crusts to fall off can also be longer. The rash is often accompanied by itch-

ing. Vesicles can fester and turn into pustules, in which the ulceration is deeper, leaving behind small depressions of the skin-smallpox, noticeable after many years. Since the rash with smallpox occurs unevenly, the rash finally disappears after 15-20 days from the beginning of the disease.

Rashes in V / smallpox are often accompanied by an increase in the lymph nodes, and in adults, the lesion of the lymph nodes is much more common than in children.

In adult patients, the phenomena of General intoxication are more pronounced. V / smallpox with lesions of internal organs and complications occurs mainly in adults. Most adults complain of General weakness and yellowing, scratching and sore throat. Less common are nasal breathing difficulties, coughing, insomnia and irritability.

Severe manifestations of smallpox include bullous, hemorrhagic and gangrenous forms. When bullous form on the skin, instead of the characteristic bubbles, bubbles with sluggish healing ulcers form. This form is more often observed in patients with various concomitant diseases.

In the hemorrhagic form of smallpox, hemorrhages in the form of rashes occur, it is more common in patients with thrombopenic purpura and other manifestations of hemorrhagic diathesis.

Gangrenous form occurs in very weakened patients and occurs usually during the first week of the disease, sometimes even on the first day of the rash and is characterized by the fact that the vesicles increase in size and quickly fill with bloody fluid, and when they dry out, a black crust is formed, surrounded by a red inflammatory rim. When the crust is rejected, necrotic areas of the skin are found. The gangrenous form of b / smallpox is characterized by a severe course and in some cases can lead to a fatal outcome.

The b / smallpox virus can cause specific lesions of internal organs: lungs, liver, spleen, kidneys, and pancreas. Damage to internal organs is associated with an altered reactivity of the body, a decrease in its resistance, and the suppression of T-lymphocytes.

Along with the typical forms, there are also erased forms of smallpox, characterized by the presence of only a single rash, not accompanied by a temperature reaction. Erased forms of b/smallpox, especially outside the foci, are recognized with great difficulty and can cause outbreaks of diseases.

Complications of b / smallpox are rare, but they can occur, be severe, and even lead to death. More often, these complications are caused by secondary bacterial flora and less often by the virus itself. From the complications caused by secondary infection, there are pneumonia, pleurisy, false croup, abscesses, pyoderma, pyelitis, erysipelas, otitis media, meningitis, encephalitis, polyneuritis, nephritis, etc.

Encephalitis and nephritis more often complicate the course of smallpox in adults than in children, which is associated with a greater allergization of the adult body.

The absolute majority of children and adults carry smallpox without any serious consequences, the mortality rate does not exceed 0.01-0.05%. In patients suf-

fering from other severe diseases or who have suffered from them, smallpox can cause serious damage to internal organs.

B / smallpox poses a significant risk to pregnant women, especially in the first months of pregnancy. There may be a pathological effect on the development of the fetus, sometimes miscarriages, and later there may be severe visceral forms of smallpox in newborns, which can lead to a fatal outcome.

DIAGNOSTICS

In typical cases, b/smallpox recognition does not cause much difficulty, but in adult patients in some cases, chickenpox is diagnosed late, because doctors often do not think about the possibility of adult children with a drop infection, and the presence of a pronounced prodromal period and the later appearance of specific rashes also make it difficult to diagnose. Untimely recognition of smallpox contributes to the lack of data on the epidemiological history and anamnesis of the disease, the timing of the appearance and nature of the rash.

It is diagnosed in / smallpox, in typical cases, by a specific polymorphic rash, characterized by a sequence of transformation of the elements of the rash and the simultaneous presence of various forms of development among them, when along with fresh vesicles or pustules, there are also drying crusts. Sometimes in adult patients with moderate to severe forms, a few vesicles and pustules may appear on the palms and soles with hyperemia around and soreness on palpation. In patients, the entire period of rash is accompanied by fever and leukopenia.

Laboratory confirmation of the diagnosis is obtained by staining the contents of vesicles or pustules with silvering and detecting the virus (Taurus Aragao) using a light microscope or immunofluorescence method. The b / smallpox virus is most easily detected in the transparent contents of newly appeared vesicles.

Antibodies to the b / smallpox virus can be detected by serological reactions, in particular, RSC, indirect immunofluorescence method, virusneutralization in sensitive cell culture, immune hemagglutination reaction. However, all these methods are time-consuming and complex, which makes it difficult to apply them in General medical practice. Diagnosis is made mainly on the basis of clinical and epidemiological data.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis of chickenpox is carried out with impetigo, vesicular rickettsiosis, pemphigus, with Kaposi's syndrome, and in previous years it was also carried out with smallpox.

Differentiation with natural smallpox was carried out primarily in the presence of pustular rashes in V / smallpox, which are more common in adult patients. When differentiating these diseases, it was taken into account that natural smallpox begins acutely, with chills, vomiting, headache, pain in the Sacro-lumbar region,a rapid increase in temperature to 39-40.5 ° C, insomnia, whereas with V/smallpox, most patients have moderate manifestations of General intoxication in the form of headache, dizziness, General weakness, sometimes coughing, runny nose, nausea. Fever in most patients with chickenpox does not exceed 38-39 ° C and lasts, on av-

erage, from 3 to 7 days, does not have a regular character, with repeated sweats, periodic temperature rises are observed, followed by remissions.

In patients with natural smallpox, the fever wave in the prodroma period lasts for about 3 days, changing into remission for 3-4 days and again increasing to 39.0-40.0 ° C with significant fluctuations when pustular rash appears.

If V / smallpox rashes on the skin appear in jerks without a certain sequence, then with natural smallpox there is a strict sequence: the rash first appears on the face and scalp, on the hands, on the second day - on the trunk, on the third day-on the lower limbs. Significant differences are also noted in the nature of the rash, which is polymorphic in smallpox. The elements of the rash are simultaneously in different stages of development, while the vesicles are single-chamber, located on a non-infiltrated base. In patients with natural smallpox, all the elements of the rash are quite dense and located on an infiltrated base, the vesicles are multicameral and when punctured, unlike vesicles in smallpox, do not fall off. On a limited area of skin, all elements of the rash are in the same phase of development, i.e. the rash is monomorphic. If in chicken pox, the indentation in the center of the vesicle - the "navel" - is rare, then in natural smallpox, most vesicles and pustules have an umbilical indentation.

The mucous membranes of the larynx and genitals are rarely affected by smallpox. the rash is not abundant. after opening the vesicles, surface erosions are formed. At the same time, most patients with natural smallpox are affected by the mucous membranes of the soft palate, nose, larynx, gums, and conjunctiva.

Blood in smallpox during the rash period is characterized by leukopenia, neutropenia, relative lymphocytosis, and in natural smallpox-moderate leukocytosis, neutrophilosis with a shift to the left.

Impetigo differs from b / smallpox by flabby vesicles located mainly on the hands and face. The bubbles easily burst and form purulent crusts.

TREATMENT

Treatment is usually carried out at home. There are no means of etiotropic therapy. With severe intoxication with abundant pustular rashes, it is recommended to prescribe antibacterial agents. Recently, there have been reports of a positive effect of antiviral drugs (acyclovir, vidarabine) in people with impaired immune status, as well as leukinferon (a new-generation IFN drug), which when prescribed in the early stages of the disease shortens the febrile period, faster stop falling asleep, less often develop complications.

Therapeutic measures are mainly aimed at caring for the skin and mucous membranes. For faster drying of the bubbles and prevention of secondary infection, the elements of the skin rash are smeared with 1% aqueous solutions of methylene blue or diamond green, a concentrated solution of potassium permanganate. Apply a 0.1% aqueous solution of etacridine lactate or Castellani liquid. Aphthous formations are treated with a 3% solution of hydrogen peroxide or a 0.1% solution of etacridine lactate. Severe itching can be relieved by smearing the skin with glycerin, wiping with water with vinegar or alcohol.

Prescribe antihistamines. In severe cases, especially in the weak and elderly, it is recommended to prescribe a specific immunoglobulin.

PREVENTION

Hospitalization of smallpox patients is carried out according to epidemiological and clinical indications. More often, patients are isolated at home. Isolation of patients ceases 5 days after the appearance of the last fresh element of the rash. A b/smallpox patient becomes contagious to others not earlier than 20 hours before the appearance of exanthema. If the first sick child in the group has stopped visiting a child care facility more than 24 hours before the onset of a mass rash, then the quarantine in the child care facility can not be established. In the DDU, children who communicate with the patient are separated for 21 days. If the day of communication with the source of infection is established accurately, disconnection is carried out from the 11th to the 21st day of the incubation period. If there are repeated cases in the DDU, the separation is not carried out. Because of the low resistance of the pathogen, final disinfection in foci of smallpox is not carried out. Weakened children who have not had chickenpox are intramuscularly injected with 1.5-3 ml of immunoglobulin obtained from the blood serum of reconvalescents. Dispensary monitoring of patients who have been ill is not regulated.

RUBELLA (RUBEOLA)

Rubella is an anthroponotic viral infection with an aspiration mechanism for transmitting the pathogen. It is characterized by moderate fever, mandatory development of generalized lymphadenopathy and the appearance of a small-spotted rash.

ETIOLOGY

The causative agent is an RNA-genomic virus of the genus Rubivirus of the family Togaviridae. All known strains belong to the same serotype. In the external environment, the virus is quickly inactivated under the influence of UV rays, disinfectants and heating. At 56 ° C, the virus dies after 30 minutes, at 100 ° C-after 2 minutes, when exposed to UV rays - after 30 seconds. At room temperature, the virus persists for several hours, and it tolerates freezing well. It shows teratogenic activity.

EPIDEMIOLOGY

The source of infection is a person with a clinically expressed or asymptomatic infection. Of great epidemiological importance are children with congenital rubella, in whose body the virus can persist for up to 1.5 years or more, as well as patients with a latent form of infection, which among adults are 6 times more than patients (in 30-50% of patients, rubella occurs in an asymptomatic form). A sick person begins to secrete the virus from 7-8 days after infection and in the first 4-5 days of the appearance of a rash. However, there is evidence that patients remain infectious for 7 days and even 3-4 weeks after infection.

The transmission mechanism is aspiration, the transmission path is air-drop. The virus is released from the patient's body with the secret of the upper respirato-

ry tract mucosa. It can be detected in the urine and feces of patients, but food and contact-household transmission routes are not of significant epidemiological significance. Due to the instability of the virus in the external environment, infection through third parties and objects does not occur. Possible vertical mechanism of transmission of the pathogen to the fetus from the mother who became ill in the first 3 months of pregnancy. As a result of infection, fetal death, miscarriages, and the birth of children with malformations are possible. In children with congenital rubella, there may be a long-term persistence of the virus, its isolation with nasopharyngeal mucus and urine.

According to existing regulations, a rubella patient is isolated from the children's team for a period of at least 5 days from the moment of the appearance of the rash. Children who have been in contact with the sick are not isolated.

In rubella, unlike other drip infections, the immune layer in the population is less active and does not cover all sensitive individuals, so 15-50% of women of childbearing age still have a potential risk of rubella infection during pregnancy. One of the reasons for the slower formation of the immune layer in rubella is that rubella is less contagious than measles or chickenpox, so the circulation of rubella virus among the population is more limited.

If the stratum of non-immune women of childbearing age becomes higher than 20%, then there is a threat of an epidemic outbreak of rubella. If people who are immune to rubella do not come into contact with rubella patients for a long time, then their level of immunity may decrease so much that they may reinfect themselves if they encounter the virus again.

The main sign of reinfection is seroconversion, but clinical manifestations of rubella, including exanthema, are rare, which, apparently, is due to the absence of pronounced virosemia, resulting in the release of the virus from the nasopharynx is short and non-intensive, so those who have undergone reinfection do not play a significant epidemiological role.

Specific antibodies in reinfection consist exclusively of Ig G, whereas in primary rubella disease they occur in fractions of Ig G and Ig M and the immune response begins with the appearance of specific IG m antibodies, which in the first 5-6 days of the disease predominate over Ig G. In the future, specific Ig Gantibodies dominate, reaching a peak 3-4 weeks after the onset of rubella and remaining in a high titer for several months and even years. Therefore, if specific Ig G-antibodies appear and increase after 8-10 days or later after contact with a rubella patient Ig G-антитела и отсутствуют and IG M-antibodies are absent, then this indicates in favor of reinfection. At the same time, the presence of Ig M-antibodies in the blood serum of the subject after 18-25 days and even later after contact with the patient Ig Mindicates primary rubella disease.

PATHOGENESIS

The pathogenesis of rubella is insufficiently studied. The entrance gate for acquired rubella is the upper respiratory tract, through which the virus enters the regional lymph nodes, where the virus multiplies, accumulates and then enters the blood. Virology in rubella occurs even before the appearance of clinical symptoms

of the disease, that is, in the incubation period. Due to Virology, the infectious origin of hematogenous spread throughout the body. Having lymphotropic and dermotropic properties. the virus causes changes in the lymph nodes, which increase in size already in the incubation period, as well as skin damage.

In congenital rubella, the virus enters the fetus with the mother's blood, infecting the epithelium of chorionic villus and endothelium of placental blood vessels, from where it is carried into the fetal bloodstream, infecting the cells of the embryo. Rubella virus has a tropism to the embryonic tissue, resulting in a violation of fetal development. Among the mechanisms of formation of congenital anomalies, direct cytopathogenic action of the virus, cell growth disorders and fetal tissue ischemia associated with placental vascular disorders are suggested. The virus disrupts mitotic activity, slows down the growth of cell populations, causes chromosomal changes, which leads to a lag in the physical and mental development of the child, the formation of various deformities.

Those organs and systems of fetal organs that are in the formation period are affected. Critical periods of intrauterine fetal development are 3-11 weeks for the brain, 4-7 weeks for the eyes and heart, 7-12 weeks for the hearing organ, and 7-12 weeks for the palate-10-12-I am a week, etc. Thus, the nature and severity of deformities in congenital rubella depend on the timing of fetal development. The virus has a destructive effect on the lens of the eye and the cochlea of the labyrinth of the inner ear. Cataracts, glaucoma, and heart defects develop with the mother's rubella disease in the first 2 months of pregnancy, and fetal psychomotor disorders develop with the mother's disease at 3-4 months. Already formed fruit is relatively resistant to the effects of the virus. Despite the presence of antibodies to the rubella virus in the blood, the pathogen persists for a long time in the child's body, which can be a source of infection for others for a long time. Rubella disease of women at the 3-4 th week of pregnancy causes congenital deformities in 60% of cases, at the 9-12 th week - in 15% and at the 13-16 th week of pregnancy in 7% of cases.

Immunity after congenital rubella is less stable. This is probably due to the fact that the formation of immunity in congenital rubella occurs in the conditions of an immature fetal immune system.

CLINIC

The incubation period is the same in children and adults and is 15-24 days; sometimes it can be reduced to 10 days.

In children, the prodromal period is usually absent, and rubella begins directly with a rash, but sometimes a headache and slight malaise are noted the day before the rash. catarrhal phenomena are poorly expressed and are not observed in all patients.

In adults, there is a more distinct prodromal period, during which catarrhal phenomena can be expressed: a runny nose, soreness and a feeling of scratching in the throat, dry cough, photophobia, lacrimation. The prodromal period usually lasts from a few hours to a day.

Both children and adults at least 24 hours before the appearance of skin rashes, there is an increase in the lymph nodes. The most typical is an increase in

the occipital, parotid, and lateral lymph nodes, which is an important diagnostic sign, but lymphadenopathy is not found in all patients. In adults, sometimes the increase in lymph nodes occurs in stages, with the first increase in anterior cervical, the next day lateral and another day - occipital. Lymphadenopathy can persist for quite a long time, on average 2-3 weeks.

One of the most important and permanent diagnostic signs of rubella is a rash that first appears on the face and behind the ears, on the scalp and within 10-12 hours spreads to the entire body, while creating the impression of a momentary rash, which reaches its maximum severity after 24 hours from the beginning of the appearance of exanthema. The evolution of the rash often occurs so rapidly that by the time it appears on the torso on the face, it has already faded. The rash is mainly localized on the extensor surfaces of the extremities, buttocks, back; less intense rash on the face, neck. There is no exanthema on the palms and soles. Rash elements are usually round in shape, do not rise above the skin level, the size of a pinhead to a lentil (2-5 mm), they are located on the unchanged skin and usually do not merge. A pale pink rash. In adult patients, the rash is more abundant and has a tendency to merge, which sometimes results in erythematous fields, which greatly complicates the differential diagnosis of rubella with measles and scarlet fever. In 20-30% of patients, the rash may be absent. with a weakly expressed rash, sometimes it helps to detect it by provoking a rash, for which a venous stasis is created on the arm by easily pulling it with the help of a blood pressure cuff, tourniquet, or simply with your hands, while the pulse should be felt. After 1-3 minutes. the rash, if present, will be more noticeable.

At the same time with skin rashes or shortly before them, in some patients, enanthema is detected in the form of single small spots on the hard palate and oral mucosa (Forchheimer's spots), sometimes there are spot hemorrhages on the Palatine tongue and soft palate.

Internal organ damage in rubella usually does not occur, but sometimes a feverish reaction causes a slight tachycardia, muffling of heart tones. In some cases, at the height of the disease, the liver and spleen may increase. Intestinal dysfunction and dyspeptic disorders are atypical for rubella.

Along with typical, severe forms of measles often erased form of the disease, which are also accompanied by release of virus into the environment, viremia and the appearance of specific antibodies, and sometimes changes in the blood. Erased forms of rubella are as dangerous for pregnant women as rubella with severe symptoms.

Rubella in pregnant women does not have any differences in the clinical picture, difficulties arise in cases where the disease occurs atypically or in an erased form. The possibility of erased forms of rubella in pregnant women should be suspected in all cases, if after contact with a patient with rubella after 15-21 days, even a minor catarrh of the upper respiratory tract appears.

COMPLICATIONS

With rubella, complications are rare. Among them, pneumonia, otitis media, arthritis, angina are the most frequent, and thrombocytopenic purpura occurs less

frequently. The development of complications is usually associated with the addition of secondary bacterial infections. Extremely rarely (mainly in adults), severe encephalitis, meningoencephalitis and encephalomyelitis are observed. Encephalitis begins acutely with the appearance of high body temperature, vomiting, impaired consciousness, convulsions. Develop paresis and paralysis, damage to the cranial nerves, focal symptoms appear. When punctured, the cerebrospinal fluid flows out under pressure, and when analyzed, it reveals a slight increase in the level of protein and glucose, pleocytosis.

DIAGNOSTICS

In the hemogram for rubella, leucopenia, lymphocytosis, and an increase in ESR are often detected. Plasma cells are sometimes found in adults. In General, changes in the hemogram are subject to significant fluctuations depending on the age of patients and the severity of the disease.

Serological diagnosis of rubella is carried out with the use of rtga, RSC, ELISA and RIA. All serological reactions allow you to make a diagnosis only retrospectively. Therefore, they are more widely used for the diagnosis of rubella in pregnant women. For diagnostic purposes, serological reactions are performed with paired serums: one is taken in the acute period of the disease (1-3 days of the disease or immediately after contact with a patient with rubella), the second – 2-3 weeks after the rash, the interval between blood collection should be at least 10 days. A four-fold increase in the antibody titer is considered diagnostic. The presence of antibodies in the first blood serum, which should be taken immediately after contact, but no later than 12 days, indicates that the woman has suffered rubella earlier, so there is no danger of fetal damage and the pregnancy can be preserved. The absence of antibodies in the first serum indicates a woman's susceptibility to rubella infection, and the increase in titer or appearance of antibodies in the second serum – an active rubella infection and the danger of fetal damage. If an active rubella infection is detected in the first 3 months of pregnancy, then this gives grounds for its termination.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis of rubella is most often carried out with measles, scarlet fever, enterovirus infections, and an allergic rash.

Rubella from measles is characterized by a longer incubation period, less pronounced catarrhal phenomena and conjunctivitis, minor intoxication,the absence of Filatov-Koplik spots, pigmentation and peeling, phagocytosis of the rash. With measles during the first day, a rash appears on the face and upper chest, on the 2nd-on the trunk and upper limbs, on the 3rd – on the lower limbs. The measles rash disappears in the same order as it appears, leaving behind pigmentation that lasts 3-10 days, as well as peeling. Catarrhal phenomena, high temperature, conjunctivitis, often "barking" cough appear with measles 2-3 days before the appear-

ance of the rash. The rash with rubella is less bright and is located mainly on the extensor surfaces of the body, back and buttocks.

From scarlet fever, rubella differs in the nature of rashes, less pronounced intoxication. With rubella, there is no sore throat, no bright hyperemia of the oropharyngeal mucosa and "crimson" tongue. Rash with scarlet fever is more abundant, small-point, located on a hyperemic basis, elements of a rash of a smaller size; it is localized mainly on the neck, on flexor surfaces and in the skin folds of the extremities, in the axillary and inguinal regions, in the lower abdomen, on the chest, often accompanied by pinpoint hemorrhages in the articular folds. Thickening of the rash in places of natural folds is a symptom of Pastia.

For scarlet fever is characterized by white dermographism and pale nasolabial triangle. In the blood of scarlet fever, neutrophilic leukocytosis with a shift to the left, eosinophilia are noted; and in rubella, leukopenia with relative lymphocytosis, as well as plasma cells, are detected.

Rubella differs from an allergic rash by its cyclical course, the presence of lymphadenitis and the nature of the rash. Often, an allergic rash is no different from rubella, while only a study of the condition of the peripheral lymph nodes helps to establish a correct diagnosis.

Enteroviral infections accompanied by rashes are diagnosed on the basis of the absence of enlargement of the occipital lymph nodes, the presence of diarrhea.

TREATMENT

Treatment for rubella is symptomatic. Patients with uncomplicated rubella can be treated at home, hospitalization is carried out for epidemiological indications. Prescribe a complex of vitamins (ascorbic acid, Riboflavin, thiamine), antihistamines. With considerable conjunctivitis in eyes buried 15-20% solution of sulfacetamide. In cases of severe lymphadenitis on the area of the lymph nodes, the effect of dry heat, UHF is shown. The occurrence of rubella meningoencephalitis is an indication for urgent hospitalization. Such patients are prescribed large doses of prednisone (80-120 mg per day), as well as dehydration, detoxification and anticonvulsant therapy.

In severe cases of rubella, the interferon-type drug leikinferon is used, which practically has no side effects and can be used for the treatment of pregnant women.

The prognosis for rubella is favorable, but in cases of encephalitis, the mortality rate can reach 20-40%. With congenital rubella, the prognosis depends on the severity of the process and the duration of pregnancy, often serious: heart defects, damage to the organs of vision, hearing, the nervous system, and malformations of the skeleton and skull are noted. Most developmental abnormalities remain for life.

PREVENTION

Early isolation of patients is indicated for prevention purposes. Disinfection is not carried out, they are limited to wet cleaning and ventilation of the room. The patient is subject to isolation for a period of at least 5 days from the moment of rash.

When isolating patients, special attention is paid to the exclusion of their contact with pregnant women. Pregnant women who have not previously had rubella should avoid contact with patients for at least 3 weeks. As an emergency prevention, pregnant women who have communicated with the patient are injected with anti-reddening immunoglobulin. In order to prevent secondary cases of the disease in the focus within 72 hours from the moment of detection of the first patient, the following categories of persons (aged 12 months to 35 years) from among those who communicated with the patient are subject to vaccination (revaccination) :

- who have not previously had rubella and have not been vaccinated against it;
- who have not previously had rubella and have been vaccinated against it once (if no more than 6 months have passed since the vaccination);
- persons with an unknown infectious and vaccination history of rubella.

Persons who have been in contact with the sick are not subject to isolation, however, it should be borne in mind that they may have erased forms of rubella. All methods of quarantine order were ineffective, so the main importance in the prevention of rubella is given to vaccination. Virtually all vaccines used in the world are harmless and highly immunological effective.

Live viral rubella vaccine is contraindicated for pregnant women. After vaccination, a woman should be protected from pregnancy for at least 3 months. to avoid infection of the fetus. Other contraindications include immunodeficiency States, severe diseases with high body temperature, hypersensitivity to certain components of the vaccine, the use of antimetabolites, steroids. After the introduction of the vaccine, sometimes there is an increase in body temperature, the occipital lymph nodes may increase, rash, arthritis and arthralgia may appear.

In accordance with the order of the Ministry of health of the Russian Federation No. 229 dated 27.06.01, vaccination against rubella is included in the national calendar of mandatory vaccinations. Children aged 15-18 months and girls aged 12-14 years are subject to vaccination. In Russia and was allowed to use the following foreign vaccine containing a live uses an attenuated virus strain Wistar RA 27/3: mumps-measles-krasnushnaya vaccine MMR-2 (Merck sharp & Dohme, USA), mumps-vaccine krasnushnaya MR-VAX-2 (Merck sharp & Dohme, USA), krasnushnaya vaccine MERUVAX-2 (Merck sharp & Dohme, USA), mumps-vaccine krasnushnaya RUVAX and krasnushnaya vaccine RUDIVAX (Aventis-Pasteur, France).

MEASLES (MORBILLI)

Measles is an acute viral anthroponotic disease with an aspiration mechanism for transmitting the pathogen. It occurs with a characteristic fever, catarrhal inflammation of the mucous membranes of the eyes, nasopharynx and upper respiratory tract, specific rashes on the mucous membrane of the mouth and a spotty-papular rash on the skin.

ETIOLOGY

Measles virus-belongs to the family Paramyxoviridae, the genus Morbillivirus, has an outer villous shell, a helical nucleocapsid and hemagglutinin, does not contain neuraminidase. The measles virus contains RNA. There is only one known antigenic type of the virus, similar in structure to the causative agent of mumps and parainfluenza. The measles virus is isolated from nasopharyngeal mucus, blood of the patient and urine in the catarrhal period and at the very beginning of the rash. The virus is not resistant to physical and chemical factors and does not persist in the external environment for more than 30 minutes, but air flows together with droplets of mucus can be transported over considerable distances. At 56 ° C, the measles virus dies within 2-3 minutes, but is resistant to low temperatures (- 70 ° C), at 60 ° C – dies instantly. Sensitive to sunlight and UV rays, it is easily inactivated by ether, formalin. The virus can be cultured in cultures of the renal epithelium of a human embryo or rhesus macaque.

EPIDEMIOLOGY

The source of infection is measles patients who secrete the virus into the external environment in the last 2 days of the incubation period and up to the 4th day after the rash. The total duration of the infectious period is 8-10 days. The most contagious patient is in the catarrhal period. From the 5th day of the rash, the patient is considered already non-infectious and his isolation can be stopped.

High contagiousness of the patient in the catarrhal period is associated with an abundant discharge of the virus from the nasopharynx when coughing, sneezing, talking. With the flow of air, it can be carried over considerable distances. Infection is possible even with fleeting contact with the patient. Due to the low resistance in the external environment, the infection is not transmitted by contact and household means. Measles is widespread all over the world, the susceptibility is very high, and the infection rate is almost 100%.

The characteristic alternation of periodic rises and decreases in measles incidence still persists. Before the start of planned vaccination, periodic rises in morbidity were registered everywhere with intervals of 2-3 years. The epidemic process in measles depends on the level of collective immunity, determined by the proportion of people who have overcome measles among the population. Mass immunization has made changes in the epidemiological manifestations of infection: it has increased the intervals between rises in morbidity to 8-10 years, shifted seasonality to the spring and summer months, and also contributed to the "maturation" of the infection. People of all ages can get measles, and children aged 1 to 5 years are more likely to get sick among the unvaccinated. Up to a year, children rarely get sick due to the small number of contacts and the presence of passive immunity received from the mother. Immunity is maintained for life, repeated cases of the disease are rare.

Currently, there are prerequisites for the complete elimination of measles.:

- absence of carrier and reservoir of infection among animals;
- presence of only one serotype of the pathogen;
- the presence of a stable immune system after a disease;
- obtaining a highly effective vaccine.

PATHOGENESIS AND PATHOLOGICAL ANATOMY

Measles is a common, generalized infectious disease that affects mainly the respiratory system, the Central nervous system, and the digestive tract. The virus enters the body through the mucosa of the upper respiratory tract and, possibly, the conjunctiva. The virus settles on the mucosa, penetrates into the submucosa, then into the regional lymph nodes, where it is fixed and primary reproduction occurs.

The virus is detected in the blood already from the 3rd day of the incubation period, the first wave of virosemia occurs, reaching the highest intensity in the catarrhal period and on the 1st day of the rash. In the initial period, the amount of the virus is still small and can be neutralized by the introduction of gamma globulin, which is the basis for passive immunization, conducted in contact with a measles patient. In the middle of the incubation period in the lymph nodes, the spleen have a high concentration of virus is used for recording and reproduction in the cells of macrophage system, lymph nodes, tonsils, liver, spleen, myeloid tissue in the bone marrow. From the 3rd day of the rash, the amount of virus released decreases sharply, and by the 5th day it ceases to be detected in the blood. At this time, virusneutralizing antibodies appear in the blood.

With measles, there is a decrease in both local and General immunity, which, along with extensive damage to the mucous membranes of the respiratory tract and digestive tract, creates favorable conditions for the penetration of secondary infections, which occupy a dominant position in the occurrence of complications in this disease. In patients with measles, vitamin metabolism (especially the metabolism of vitamins C and A) suffers, which in turn creates additional conditions for the occurrence of complications. In the catarrhal period, inflammation of the oropharynx, larynx, trachea, bronchi is noted, and focal pneumonia often develops. On the mucous membrane of the lips and cheeks appear spots Filatov-Koplik-Belsky.

Morphological changes in measles occur primarily in the places of primary reproduction of the virus, i.e. in the upper respiratory tract, where there is damage to the scleral epithelium, dystrophic changes occur in the cells of the respiratory mucosa, which increase in size, various inclusions begin to be detected in their cytoplasm, and the number of nuclei increases. In the lumen of the respiratory tract and alveoli, in addition to the deflated alveolocytes, serous fluid containing individual red blood cells, white blood cells can be detected. In the mucous membrane of the upper respiratory tract, trachea of the bronchi, lymphohistiocytic infiltrates occur, the endothelium of blood vessels of the respiratory organs swells, in some cases undergoes fibrinoid transformation. In cases of severe measles, necrosis of the surface layers of the mucous membrane can occur, most often in the larynx, in the vocal cords, spreading down to the upper part of the trachea and up to the entrance to the larynx. This leads to hoarseness of the voice, which is also affected by inflammatory infiltration and tissue edema. In the case of transition of necrosis to the connective tissue base of the respiratory mucosa, ulcers occur. The pathological process can completely invade the mucous membrane of the respiratory tract, af-

fecting the bronchi, bronchioles, alveoli, causing bronchitis, peribronchitis, pneumonia.

In the myeloid tissue of the bone marrow, multicellular cells characteristic of measles are detected, focal fullness and edema of the meninges, edema and focal proliferation of glia, focal lysis of myelin fibers, disorders of blood and lymph circulation are noted in the Central nervous system. In cortical encephalitis, mesenchymal and gliotic proliferation occurs, localized mainly in the white matter of the brain. The reaction from the cervical lymph nodes is usually moderate.

CLINIC

The incubation period for measles is 6 to 17 days, but in most cases it lasts 8 to 11 days. In individuals who have undergone seroprophylaxis, the incubation period is 21-28 days. It is prolonged when combined with measles and other infectious diseases, in persons who received hemotransfusion, plasma administration.

There are three periods of the course of measles: catarrhal, rash period and pigmentation period.

The catarrhal period in children and adults begins acutely, while adults are more pronounced intoxication and temperature reaction. Adult patients complain of lethargy, fatigue, General malaise, sleep disorders, headache. The temperature rises to 38-39 ° C . On the 2nd-3rd day of the disease, the temperature decreases, sometimes to subfebrile digits, but with the beginning of the rash period, there is a new rise in temperature, which after 1-2 days reaches the maximum figures, and by the 4th – 6th day of the rash decreases and returns to normal. In the first days of the disease, vomiting, abdominal pain, and runny stools often occur. There is an abundant discharge from the nose, which is initially mucosal, and then becomes Mucopurulent, there is an obsessive cough, which is one of the constant symptoms, there is a hoarse voice. Sometimes at the beginning of the disease, due to laryngeal edema, croup syndrome appears. Objective examination reveals hyperemia of the oropharyngeal mucosa and granularity of the posterior pharyngeal wall. Such symptoms that are often found in children, such as puffiness of the face, catarrhal angina, a rough "barking" cough, stenosed breathing, are usually not observed in adults. On the 2-3 day of the catarrhal period, the cough increases, there is hyperemia of the conjunctiva, puffiness of the eyelids and photophobia. Already in the catarrhal period, 1-2 days before the appearance of rashes, on the mucous membrane of the cheeks, both in children and adults, there is a typical and pathognomonic symptom of measles - the Filatov-Koplik-Belsky spot. They are small, grayish-whitish papules surrounded by a narrow border of hyperemia, do not merge with each other, are not removed with a cotton-gauze swab. Filatov-Koplik-Belsky spots are usually located on the mucous membrane of the cheeks against small molars, much less often - on the mucous membrane of the lips and gums. At the same time, in adults, they are found up to 3-4 days of skin rashes, while in children, they disappear in the first 2 days of the rash. For 1-2 days before the appearance of exanthema on the mucous membrane of the soft and hard palate appears cortical enanthema: small pinkish-red spots of irregular shape, the size of a pinhead to lentils; after 1-2 days, they merge and become indistinguishable. During this period,

both adults and children sometimes experience dysfunction of the gastrointestinal tract due to viral damage to its mucous membrane. Symptoms of intoxication are increasing up to a maximum of manifestations in the period of the rash. Sometimes in the catarrhal period, an ephemeral prodromal small-point or papular rash appears. The catarrhal period lasts 3-4 days, sometimes longer, especially in adults – up to 5-8 days.

The period of rash in adults retains its classic features; it begins on the 4-7 day of the disease and lasts 3-4 days. The first elements of the rash appear behind the ears, on the scalp, on the bridge of the nose and then during the first day spread to the face, neck, and upper chest. During the next day, the rash spreads to the torso and upper arms and on the 3rd day to the lower limbs and distal parts of the hands. The descending sequence of rashes is characteristic of measles and is an important differential diagnostic sign. Simultaneously with the appearance of a rash, there is an increase in body temperature, which reaches a maximum in 1-2 days. In adults, the rash is more abundant, coarse-spotted, papular and often has a draining character. In some patients with a severe course of the disease (more often in adults), the rash may become hemorrhagic on day 2-3.

During the pigmentation period with a smooth course of measles, the condition of patients becomes satisfactory, body temperature normalizes, appetite and sleep are restored. The transition of the rash to pigmentation occurs in stages - from top to bottom. Pigmentation is also a characteristic and important diagnostic feature. During this period, there is a decrease in all types of immunity (measles anergy), so reconvalescents should be protected from secondary infection. Often after pigmentation, which lasts no more than 5-7 days, patients may experience bran-like peeling, mainly on the face, which is also an important diagnostic sign in the retrospective diagnosis of measles.

In modern conditions, when determining the form of measles, it is advisable to use the classification of A. A. Koltypin and M. G. Danilevich. According to this classification, there are 2 forms of measles: typical, which has the main classical signs of the disease, and atypical, which has any significant deviations in the clinical picture. According to severity, there are 3 forms: light, medium and heavy. Atypical measles can occur in two ways: an aborted and motivirovano. Abortive measles begins as a typical measles and ends after 1-2 days from the onset of the disease, while the rash occurs only on the face and torso, and the body temperature, as a rule, appears only on the 1st day of the rash.

Mitigirovannaya measles proceeds much easier and is noted in persons who have received a vaccination or previously had measles. It is characterized by a shortened prodromal period, moderate catarrhal phenomena occurring against the background of normal or subfebrile body temperature. In addition, the stages of rashes are absent, the rash is sparse, quickly disappears, leaving barely noticeable short-term (from a few hours to 2-3 days) pigmentation. Enanthema, Filatov-Belsky-Koplik spots are expressed slightly or absent. There are also forms that occur almost without pathological manifestations. Their diagnosis is possible only with the help of a serological study of paired blood serums. The severity of measles is determined by the severity of symptoms of intoxication, local manifesta-

tions, and the presence of complications. The most severe cases of measles occur in people living in territories that are "free" from this infection for a long time and in people over 70 years of age.

With uncomplicated measles, the blood is marked by leukopenia, rod-shaped shift, lymphocytosis, plasma cells may appear, ESR increases.

COMPLICATIONS

The appearance of complications in measles contributes to a decrease in both humoral and cellular immunity, which leads to a decrease in the body's resistance to secondary infections. Measles is characterized by complications such as pneumonia (while many authors do not consider pneumonia a complication if it occurred in the acute period of the disease), otitis media, laryngitis, cortical croup (more common in children), stomatitis, and cortical encephalitis. Pneumonia can appear in any period of the disease, but early emerging pneumonia and having the character of bronchopneumonia are more severe, with pronounced intoxication, changes in the nervous and cardiovascular systems. Late pneumonia occurs, as a rule, on 4-5 days from the beginning of the rash. In these cases, the body temperature rises again, the General condition worsens, shortness of breath and a large number of wet wheezes appear.

Such complications as meningoencephalitis, serous meningitis, polyneuritis, develop later, usually on the 10-15 day of the disease, during the pigmentation period.

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

DIAGNOSTICS

Diagnosis of measles is based on typical clinical signs and epidemiological data. In favor of measles is evidenced by the acute onset of the disease, elevated temperature, the presence of catarrhal phenomena, conjunctivitis, Filatov-Belsky-Koplik spots that appear on the 1-3 th day of the catarrhal period on the oral mucosa, spot-papular rash that appears on the 3-4 th day of the disease, and different stages of rashes in the next 3 days.

To confirm the diagnosis, especially of erased, atypical forms of infection, serological diagnostic methods are used - rnga, rtga, PH. Since the increase in the titer of antibodies in paired serums is taken into account, the specific diagnosis is retrospective. The first blood sample is taken no later than the 3rd day of the rash period, the second after 10-14 days. The diagnosis is considered verified only when the antibody titer increases by 4 times or more. The diagnosis can be confirmed by detecting the virus in nasopharyngeal flushes both in tissue culture and by immunofluorescence. The antigen is detected before the 3rd-4th day of the rash.

DIFFERENTIAL DIAGNOSIS

When differentiating measles with other respiratory diseases, it should first of all be borne in mind that with respiratory viral infections such as influenza, parainfluenza, adenovirus, respiratory syncytial infection, the mucous membranes of the cheeks remain clean, pale pink, shiny, whereas with measles, early spots appear Belsky-Filatov-Koplik, which persist for 2-3 days, occasionally 4-5 days, and after their disappearance, the mucous membrane of the cheeks acquires a velvety appearance.

The use of antibiotics in the prodromal period of measles leads to less pronounced symptoms and a febrile reaction, but the mandatory symptom in these cases remains a cough.

In influenza and other acute respiratory diseases, there is no spot-like papular rash, Belsky-Filatov-Koplik spots. Although with adenovirus infection, conjunctivitis and catarrhal phenomena are also noted, but there is no Belsky-Filatov-Koplik symptom, a characteristic measles rash.

Scarlet fever is not characterized by catarrhal phenomena. Pharyngeal hyperemia in scarlet fever is sharply delineated.

With rubella, the catarrhal period is less pronounced, the rash appears on the 1st day of the disease and in a few hours spreads to the trunk and limbs, there is no stage in the rash. Rash elements in rubella are smaller and paler, do not tend to merge, are polymorphic, localized on the back and extensor surfaces, disappear after 1-2 days without subsequent pigmentation. The mucous membranes of rubella are shiny, smooth, of normal color, characterized by an increase in the lymph nodes, especially the occipital and posterior ones. With scarlet fever, the most bright and large rash occurs on the 1st day of appearance, on the 2nd-it pales significantly, especially on the face, and decreases in size to a fine-spotted one. On the 3rd day, the rash on the face becomes even paler, barely noticeable, but it persists well on the buttocks and outer surfaces of the thighs. On the 4th day, the rash disappears without a trace, without transition to pigmentation. With measles, the rash is the most bright, large, draining becomes on the 2nd and 3rd day of the rash. From the blood side, patients with rubella have leukopenia with lymphocytosis and the presence of plasma cells.

Measles also needs to be differentiated from infectious erythema, drug-induced and allergic rashes, serum sickness, and enterovirus infections.

Allergic rashes, unlike measles, appear without a previous catarrhal period and have no stages. In addition, the elements of the rash sometimes acquire annular, urticarial whimsical outlines, accompanied by itching.

Morbilliform rash is sometimes accompanied by serum sickness, with no recurrence of rash and catarrhal phenomena. Often the rash appears in the places where the serum is administered, it is symmetrical, there are blisters, skin itching.

TREATMENT

Only patients with severe measles are hospitalized, so most patients are treated at home. Patients from closed children's institutions, dormitories, boarding schools, as well as for epidemiological reasons are hospitalized without fail. The patient should be provided with bed rest during the entire febrile period.

There is no etiotropic treatment for measles. Therapy of measles patients is currently carried out by pathogenetic and symptomatic means and is largely aimed at preventing complications of a microbial nature and at their timely treatment. Specific anti-measles gamma globulin, which is an effective preventive agent provided it is administered to contact persons in the first half of the incubation period, does not significantly affect the course of measles, if it is administered at the end of the incubation period or during the disease. Early administration of gamma globulin helps to neutralize viral reproduction due to the presence of virusneutralizing antibodies in the drug. In addition, the introduction of gamma globulin in measles patients has a certain desensitizing and stimulating effect, which is used in medical practice.

Treatment of uncomplicated measles is to combat General intoxication and to influence local catarrhal changes from the respiratory tract and eyes.

Recently, interferon-type drugs have been increasingly used in the treatment of patients with viral diseases. Administration of the 4th generation drug-leukinferon in the early stages of the disease reduces the febrile period and reduces the number of complications in measles. At a high temperature, antipyretics are prescribed, and when signs of increased intracranial pressure appear, dehydration therapy is performed. To reduce dry cough, dionine, codeine, and expectorants are prescribed. Three or four times a day, the patient's eyes are washed with water or a 2% solution of soda and after removing pus and purulent crusts, 15-20% R-R of albucide is buried, as well as R-R of vitamin A, which protects the sclera from drying out and prevents the development of keratitis. In case of corneal ulcers, the eyes are washed with 2% boric acid solution, 15-20% albucide solution is instilled, and 1% prednisolone ointment is applied to reduce inflammation.

Dry lips are smeared with vaseline, the nose is cleaned with a wet swab and buried in the nasal passages 3-4 times a day with vaseline oil.

In some cases, with a severe and complicated course of measles, it is possible to prescribe antibiotics. In case of complications, treatment is carried out depending on their nature.

False croup occurs due to laryngospasm, and if stenosis of the 2nd degree develops, then prednisone is prescribed for 5-7 days with an initial dose of 15-20 mg, IV 2.5% R-R aminazine is administered with 1% R-R Dimedrol and 0.5% solution of novocaine 0.5 ml. Resort to a hot bath, apply a warming compress on the neck, give a warm drink (hot milk with soda or Borjomi), in rare cases with stenosis of the third degree, it is necessary to perform a tracheostomy.

After the endured measles, the condition of asthenia can persist for a long time, which should be taken into account when developing a sparing regime of study, work and rest of the patient. The prognosis for timely and correctly started treatment for measles is favorable.

PREVENTION

A measles patient is subject to early isolation for a period of at least 5 days from the onset of the rash, but if the disease is complicated by pneumonia, this period is extended to 10 days. Since the measles virus is not resistant to the external

environment, disinfection in the foci is not carried out. The room where the patient was should be ventilated for 30-45 minutes. Measures among those who have come into contact with a measles patient include accurate accounting of all contacts, quarantinization, surveillance, and passive immunization. Children who have been in contact with a measles patient are subject to separation and are not allowed to enter children's institutions (nurseries, kindergartens and the first 2 classes of school) from the 8th day of incubation to the 17th day unvaccinated and until the 21st day-subjected to passive immunization. For the first 7 days from the beginning of contact, children are not subject to separation. Students who have had contact with a measles patient are not exempt from attending educational institutions. In the hearth daily preventive examination and temperature measurement is carried out for early detection and isolation of patients.

Children who are actively immunized against measles are not subject to quarantine.

Children who have not received active immunization against measles for medical reasons and have not previously been ill with this infection are given gamma globulin, and children (older than 3 years) who do not have contraindications, but have not been vaccinated in a timely manner, are given emergency vaccination.

Passive immunization (single administration of immunoglobulin in the first 5 days after contact with the patient) is indicated for children under 3 years of age, pregnant women, tuberculosis patients and persons with weakened immune systems.

Reactions to the administration of gamma globulin, as a rule, are absent. The highest preventive effect is achieved when gamma globulin is administered no later than 6 days from the moment of contact. The duration of action of gamma globulin is 3-4 weeks (approximately 28 days). In the case of repeated contact with a measles patient after this period, an additional half of the dose of gamma globulin is administered.

Older children who have not had measles and have not been vaccinated against measles, gamma globulin is administered only for medical reasons in contact with measles patients.

To control the state of immunity of the population, selective serological studies are conducted. The who regional Committee for Europe, at its 48th session (1998), decided that the goals of the Health 21 programme were to eliminate measles from the Region by 2007 or earlier. By 2010. elimination of the disease must be registered and certified in each country.

MUMPS (Parotitis Epidemika)

Mumps is an acute viral anthroponotic disease with an aerosol transmission mechanism, accompanied by intoxication and damage to glandular organs, mainly the salivary glands, as well as the nervous system.

Currently, in terms of prevalence in the group of airborne infections, it takes the 3rd place after chicken pox and rubella. Thanks to routine immunization, the incidence has declined annually, but some vaccinated after 5-7 years, the concentration

of protective antibodies is significantly reduced, thereby increasing the susceptibility to disease of adolescents and adults. Diagnosis of children's drip infections, including mumps, is usually delayed, because doctors often do not think about the possibility of this disease. All this leads to the spread of mumps among the unvaccinated. Meanwhile, the clinical picture of mumps in adults has its own characteristics - it often causes severe and often irreversible consequences: orchitis can lead to infertility, bilateral neuritis of the auditory nerve - to deafness, etc.

ETIOLOGY

The mumps virus belongs to the genus paramyxovirus (Paramyxovirus parotitidis), the family (Paramyxoviridae), which also includes a number of measles and a number of pneumoviruses. In natural conditions, the mumps virus is pathogenic only to humans.

The mumps virus has a size of 100 to 200 microns, and electron microscopy shows a dome - shaped, flat, elongated and irregular shape.

The virus contains RNA that are sensitive to physical and chemical factors, a fully inactivated for 20 min at t º 55-60^0 C, is quickly destroyed by irradiation with ultraviolet rays, dies in 12-hour treatment with 0.1%-s ' solution of formalin, at 3-5 minute exposure to a 1% solution of Lysol, 50% alcohol or ether, but long (up to 9 10мес.) stored at low temperatures (-25^{0}C - 70^{0}C), remains viable for 8 days at 37^0 C. It is not sensitive to antibiotics and other chemotherapeutic agents. The optimal pH of the medium for the causative agent of mumps is 6.5-7.0.

The virus is cultivated in chicken embryos in cell culture of human amnion and kidney of the Guinea pig.

The mumps virus has hemagglutinin and hemolyzing activity against human erythrocytes, the number of animals and birds. There is only one known virus serotype, which has two antigens: viral-v (virus) and soluble -s (solube), which is not associated with viral particles. The existence of only one type of mumps virus creates favorable conditions for laboratory diagnostics and the creation of an effective mumps vaccine.

EPIDEMIOLOGY

Mumps is an anthroponosis and is spread in various geographical and climatic zones. The only source of infection is a sick person who secretes the mumps virus 1-2 days before the onset of clinical symptoms (before the swelling of the parotid glands) and before the 6-9 th day of the disease. The virus is secreted from the body with saliva and urine and is transmitted from person to person by airborne droplets. Possible infection through objects infected with the virus, as well as transplacental transmission. The contamination index is 30-50%.

The intensity of virus spread in the environment is small, which is due to the absence of catarrhal phenomena of the upper respiratory tract. The mumps virus is released into the environment along with a large-drop aerosol of saliva, which quickly settles and does not spread far, without infecting a large number of susceptible individuals. In the spread of infection, the duration of contact of healthy people with sick people is of great importance. However, the sensitivity is still quite high. Children under one year of age rarely get sick due to a small number of contacts and ma-

ternal immunity. Epidemic outbreaks can be registered at any time of the year, but are somewhat more frequent in late winter and spring. Among the adult population, epidemic outbreaks are more often registered in closed and semi-closed collectives (barracks, dormitories, ship crews). Crowding, poor living conditions also contribute to the spread of mumps. The disease occurs in both sexes, but men get sick more often. Individuals over 50 years of age rarely get sick. Immunity after mumps is stable.

PATHOGENESIS

The gate of infection is the mucosa of the upper respiratory tract. After entering the human body, the virus multiplies in the epithelial cells of the respiratory tract and is carried by the blood flow to all organs, of which the most sensitive to it are the salivary, genital and pancreatic glands, as well as the Central nervous system. There, it accumulates in large quantities, causes an inflammatory reaction with successive inclusion in the pathological process of a number of organs with the appearance of appropriate changes in them (mumps, meningitis, orchitis). Early virosemia and the possibility of damage to various organs and systems that are remote from each other indicate the hematogenous spread of infection.

The virus enters the salivary glands primarily because here the pathogen finds the most favorable conditions for its reproduction and development. Late salivoglandulitis is probably the result of late Virology. By the 7th-9th day of the disease, virucidal antibodies begin to accumulate in the body, which leads to the reverse development of the disease and recovery. Saliva inhibitors play a role in the mechanism of sanogenesis, which suppress the activity of the virus and prevent the virus from entering cells.

Damage to other glandular organs and the nervous system develops not from the first days of the disease, but 5-10 days after the onset of inflammation of the parotid salivary glands. This "lag" is explained by less favorable conditions for reproduction of the virus in other organs.

PATHOLOGICAL ANATOMY

Pathoanatomic changes in mumps in the parotid glands are manifested by hyperemia and significant edema of the interstitial tissue of the glands, its capsule and located around the tissues. In the area of the ducts of the gland, round-cell infiltrates consisting of epithelioid and lymphoid cells are determined. There is a catarrhal inflammation of the glandular passages, often with the expansion of the excretory ducts filled with a protein mass with a small admixture of cellular elements.

If the submandibular and sublingual salivary glands are involved in the pathological process, edema of the pharynx, larynx and tongue is possible.

In mumps orchitis, the pathological process is manifested by diffuse infiltration of interstitial tissue. Foci of hemorrhage and necrosis of the glandular epithelium with blockage of the tubules with cellular detritus, fibrin and leukocytes may occur. There are degenerative changes in the epithelium of the seminal tubules. In severe cases, as a result of orchitis, testicular atrophy can occur. Ovaries in women are rarely affected, and complications such as acute vulvovaginitis, bartholinitis and specific mastitis can also occur.

In mumps meningoencephalitis, there is hyperemia, edema of the brain and its membranes, serous-fibrinous exudate is found in the grooves of the brain and around the brain vessels. Histologically, there is a perivascular infiltration of the meninges, areas of demyelination in the white matter of the brain, and scattered foci of hemorrhage. Any specific changes in mumps lesions of the Central nervous system have not been established.

In cases of pancreatitis, there is fullness of the gland, interstitial edema, moderate degeneration of islet cells and the appearance of fat necrosis. With severe damage to the pancreas and the development of atrophy, diabetes can occur. Acute glomerulonephritis sometimes occurs in the kidneys. In the first days of the disease, spleen hyperplasia is possible. Lesions of the peripheral nerves, cornea and iris of the eyes, heart, muscles, and hearing organ in mumps are very rare.

CLINIC

The incubation period in children and adults is on average 11-26 days (fluctuations from 3 to 35 days). The prodromal period in adults is more common than in children.

In some patients, General malaise, weakness, arthralgia, headaches, unpleasant sensations in the parotid salivary gland, muscle pain, sleep disorders, dry mouth, and yellowing may occur for 1-2 or even 3 days.

The disease begins acutely, with chills and an increase in body temperature to $39\text{-}40^0$ S, with tinnitus, pain when opening the mouth and chewing, as well as pain and swelling of the parotid salivary gland, usually at first on one side, and after 1-2 days on the other. One of the early signs of mumps is soreness behind the earlobe, in the place of future edema (Filatov's symptom). In front of the ear, on one side, there is a swelling that quickly spreads and fills the retromandibular space. Puffiness spreads in three directions: up - on the area of the mastoid process, down and posteriorly - on the neck and anteriorly on the cheek. With an increase in the parotid salivary gland, the earlobe rises up, the face takes on a characteristic mumps-like appearance, especially noticeable when examining the patient from behind.

During this period, as a result of compression of the auditory meatus and Eustachian tube, patients may experience noise and pain in the ears, possibly hearing loss; when the swelling disappears, hearing is quickly restored. Maximum edema is observed by the 3rd-5th day of the disease, then it gradually decreases and disappears on the 6th-9th day. In adults, the enlargement of the gland lasts longer, often up to 10-16 days of illness.

The skin over the inflamed gland is stretched, while its color does not change. Sometimes there is a slight soreness on palpation, more in the center, on the periphery the soreness is expressed very little or may be absent. When pressing on the tumor, the fossa does not remain. Due to inflammation and edema of the parotid gland, patients experience difficulty opening their mouths, soreness when chewing and swallowing. During this period, salivation is reduced, the visible mucous membranes are dry, and patients complain of thirst. In the area of the parotid duct on the mucous membrane of the cheeks, redness and swelling are noted (a positive symptom of Mursu).

The affected submandibular and sublingual salivary glands become enlarged, their consistency becomes testy; often there is a swelling of fiber that spreads down to the neck. Puffiness is better seen when the patient turns his head in the opposite direction. Submaxillitis is 2 times more common in adults than in children.

Sublinguitis is manifested by pain in the chin area, under the tongue, which increases when the tongue is stuck out, the presence of a small, slightly painful, test-like swelling, which can spread from the chin area to the upper part of the neck. Insulated sublingual rare, usually combined with mumps.

The lymph nodes in mumps are usually not involved in the inflammatory process.

The internal organs of isolated mumps are not changed. In some patients, tachycardia, muffling of heart tones, noise at the top, hypotension are noted. Changes in the Central nervous system are manifested by headache, malaise, insomnia. Hepatolienal syndrome is unusual. Fever, as a rule, lasts no more than 5 days, but when many glands and meninges are involved in the pathological process, the temperature response is delayed for a longer time - up to 15-20 days.

The severity of mumps is determined by the severity and duration of the temperature reaction, damage to the nervous system and individual glandular organs. There are light, medium-heavy, heavy and erased course of the disease. Adults are more likely to have a severe course of the disease.

In the interpretation of complications in mumps, there are two points of view. Some authors (M. A. Selimov, V. M. Sukharev) believe that lesions of any glandular organ or nervous system should be regarded as a typical manifestation of mumps, and complications should include lesions caused by the addition of secondary flora. According to other authors (A. p. Kazantsev), in a non-complicated form of the disease, only the salivary glands are involved in the process, and pathological changes in other glands or the Central nervous system should be considered as a complication.

With mumps, orchitis, pancreatitis, serous meningitis, meningoencephalitis and other lesions often occur.

Mumps orchitis occurs mainly only in adults, and is extremely rare in boys under 14 years of age. The frequency of mumps orchitis increases with the age of the patient. Orchitis usually occurs on the 5-6 day of the disease, against the background of disappearing swelling of the parotid gland, while the condition of patients deteriorates sharply: the body temperature rises to 39-40^{0}C, there may be headache, vomiting, chills, pain in the testicle and inguinal region. The testicle increases in size, becomes painful and dense. The skin above it is hyperemic, usually affects one testicle, bilateral orchitis is much less common. Pronounced clinical manifestations of orchitis last no more than 3-5 days, then go on the wane and by 10-15 days of the disease usually disappear. Sometimes orchitis can lead to testicular atrophy. Isolated orchids are rare. The development of orhiepididimita is observed mainly in the 5 to 9-th day of illness. With mumps orchiepididimitis, there is a violation of spermatogenesis, which in the future can lead to a decrease in sexual and reproductive functions. Women may have oophoritis, bartholinitis, mastitis. Mastitis can develop not only in women, but also in girls and even in men.

Частым проявлением эпидемического паротита является панкреатит, который нередко протекает латентно и может быть выявлен только с помощью лабораторных методов диагностики. Панкреатит чаще развивается у детей и подростков и клинически выражается в повышении температуры тела, появлении опоясывающих болей в верхней части живота, тошноте и рвоте, а также снижении аппетита. При очень выраженном болевом синдроме иногда возникает картина острого живота. Течение панкреатитов обычно доброкачественное, они заканчиваются через 5-10 дней выздоровлением.

При эпидемическом паротите часто отмечается поражение ЦНС ввиду тропности вируса к нервным клеткам. Серозные менингиты и менингоэнцефалиты при эпидемическом паротите характеризуются теми же проявлениями, что и менингиты другой этиологии. Серозные менингиты обычно сочетаются с поражением других органов и возникают на 3-6-й день болезни, при этом отмечается повышение температуры тела, появляются резкая головная боль, рвота, бессонница ригидность мышц затылка, положительные симптомы Брудзинского, Кернига, брадикардия. В первые дни паротитного менингита вследствие отека и напухания вещества головного мозга у больных могут наблюдаться энцефалитические реакции в виде нарушения сознания, судорог, бреда, психомоторного возбуждения. Санация цереброспинальной жидкости у взрослых протекает медленнее, чем у детей (дольше месяца), и значительно отстает от клинического выздоровления. Течение менингита обычно доброкачественное, но иногда длительно (несколько месяцев) могут сохраняться симптомы астении.

При эпидемическом паротите иногда возникают менингоэнцефалиты, при которых появляется очаговая симптоматика. У взрослых могут отмечаться кратковременные парезы отводящих нервов, гемипарезы, появление патологических рефлексов. При эпидемическом паротите часто поражаются слуховые и вестибулярные нервы, что в отдельных случаях может привести к понижению слуха и глухоте.

Изменения крови при эпидемическом паротите незначительны и характеризуются лейкопенией (у взрослых иногда лейкоцитоз), относительным лимфоцитозом, моноцитозом, СОЭ слегка увеличена или нормальная.

ДИАГНОСТИКА

Диагностика эпидемического паротита основана главным образом на характерной клинической картине и эпидемиологическом анализе. Для выделения возбудителя исследуемым материалом служит слюна, спинномозговая жидкость (6 дней от начала заболевания), моча (до 14 дня от начала болезни). В последние годы все шире стали использовать серологические методы диагностики, однако диагноз в этих случаях удается подтвердить лишь ретроспективно. Наиболее часто используют РСК и РТГА. Высокий титр S и низкий V-антител, в острый период болезни может служить признаком эпидемического паротита. Подтвердить диагноз можно при повторном исследовании сыворотки реконвалесцентов спустя 2-3 недели после начала заболевания, при этом в 4 раза и более должен увеличиться титр V-антител, а уровень S-антител должен оставать-

ся стабильным. При РСК диагностическими титрами при однократном исследовании считают разведения 1:80; РТГА 1:10 – 11:20.

Иногда для диагностики используют внутрикожную реакцию с паротитным антигеном в виде инактивированного вируса, однако эта реакция становится положительной только в период реконвалесценции, при этом инфильтрация кожи и покраснение достигает 1-3 см в диаметре. Так же для диагностики паротита определяют уровень амилазы и диастазы в крови и моче, содержание которых повышается у большинства больных и достигает максимума через неделю от начала болезни.

ДИФФЕРЕНЦИАЛЬНАЯ ДИАГНОСТИКА

Заболевание следует отличать от гнойных воспалительных заболеваний околоушных желез, их увеличения при обтурации стенонова протока слюнным камнем, паратонзиллярном абсцессе, инфекционном мононуклеозе, а также при дифтерии, сопровождающейся отеком подкожной клетчатки (токсической форме).

Для эпидемического паротита характерно острое начало с озноба, повышения температуры тела и других признаков интоксикации, появление болезненной (больше в центре) тестообразной припухлости в проекции околоушных, реже подчелюстных или подъязычных слюнных желез с чувством напряжения и боли. Поражение чаще носит двусторонний характер. Наиболее важные и ранние признаки паротита- появление выраженной болезненности при надавливании позади мочка уха (симптом Филатова) гиперемия и отек слизистой оболочки вокруг отверстия стенонова протока (симптом Мурсу).

ЛЕЧЕНИЕ

При неосложненных формах лечение амбулаторное. Больных обычно госпитализируют по эпидемиологическим показаниям или в случаях возникновения осложнений. Средства этиотропной терапии отсутствуют. Постельный режим рекомендуют в лихорадочный период независимо от тяжести заболевания. В первые дни болезни больным дают преимущественно жидкую или полужидкую пищу. Особое значение приобретает уход за полостью рта: частое питье, полоскание кипяченой водой или 2% раствором соды, тщательная чистка зубов. На область околоушных желез применяют сухое тепло (сухие согревающие компрессы, облучение лампой соллюкс), назначают местные физиотерапевтические процедуры в виде ультрафиолетового облучения, УВЧ- терапии, диатермии. При выраженном токсикозе проводят дезинтоксикационную терапию с назначением небольших доз глюкокортикоидов. В некоторых клиниках получены положительные результаты после применения ИФН (лейкинферона) в ранние сроки заболевания.

При развитии орхита кроме постельного режима рекомендуют применять суспензории, местно в первые 3-4 дня- холод, а в последующие дни – тепло. Также проводят раннее лечение средними дозами глюкокортикоидов.

ПРОФИЛАКТИКА

Для специфической профилактики применяют живую вакцину (ЖПВ) из аттенуированного штамма вируса паротита Л-3, выращенного на культуре клеток эмбрионов японских перепелов. Профилактические прививки проводят в плановом порядке детям в возрасте 12 мес, не болевшим паротитом, с последующей ревакцинацией в 6 лет ассоциированной вакциной против кори, паротита и краснухи. Эффективность вакцинации против паротита достаточно высока, она способствует резкому уменьшению заболеваемости паротитом и снижению количества осложнений (менингитов, орхитов, панкреатитов). Оправданы вакцинация и ревакцинация подростков и взрослых по результатам серологических обследований.

МЕРОПРИЯТИЯ В ЭПИДЕМИЧЕСКОМ ОЧАГЕ

Противоэпидемическую работу в очаге начинают с изоляции больных. Госпитализации подлежат больные с тяжелыми формами и из организованных закрытых коллективов, общежитий. Чаще больного изолируют дома до исчезновения клинических признаков, но не менее чем на 9 дней. Помещение, где содержится больной, часто проветривают, проводят влажную уборку, больному выделяют отдельную посуду, белье, игрушки и др. Дети до 10 лет, не болевшие эпидемическим паротитом, подлежат разобщению на 21 день с момента контакта с больным. В связи с длительной инкубацией и контагиозностью только в последние дни этого периода лица, общавшиеся с больным, могут посещать детские коллективы в первые 10 дней инкубационного периода, но с 11-го по 21-й день подлежат разобщению. При отсутствии противопоказаний к вакцинации ранее не привитым детям следует ввести ЖПВ. Экстренную вакцинопрофилактику также можно проводить по результатам серологического скрининга. Для этого используют моно- или ассоциированные живые вакцины с паротитным компонентом, зарегистрированные на территории России.

Переболевших можно допускать в коллектив после клинического выздоровления даже при появлении в детском учреждении повторных заболеваний. Диспансерное наблюдение за переболевшими не регламентировано. Однако существуют рекомендации о необходимости наблюдения в течение 2 лет за перенесшими паротитный серозный менингит или менингоэнцефалит в связи с возможностью отклонений в неврологическом или психическом статусе.

СКАРЛАТИНА (Scarlatina)

Скарлатина – острое антропонозное заболевание, проявляющееся интоксикацией, поражением ротоглотки и мелкоточечной экзантемой.

Название заболевания происходит от итальянского слова scarlatina – багровый, пурпурный.

ЭТИОЛОГИЯ

Возбудитель скарлатины - β-гемолитический стрептококк группы А из семейства Streptococcaceа. Имеет округлую форму, в мазках обнаруживается в виде различной длины цепочек. Грамположителен. Относится к аэробам, но хорошо размножается и в анаэробных условиях. При посеве на кровяной агар вызывает гемолиз. Серологическая классификация проводится по антигенным

свойствам С-полисахарида. Группа А стрептококков, к которым относится возбудитель скарлатины, включает более 80 серотипов. Несмотря на исключительный интерес к проблеме стрептококковых инфекций и огромному числу солидных работ в этой области, до сих пор не удается получить ясный ответ на вопрос о специфических свойствах типов стрептококков А, способных вызвать скарлатину. Известно, что возбудитель продуцирует эритрогенный (скарлатинозный) токсин.

β-Гемолитический стрептококк группы А устойчив во внешней среде. Выдерживает кипячение в течение 15 минут, устойчив к воздействию многих дезинфицирующих средств (сулема, хлорамин, карболовая кислота).

ЭПИДЕМИОЛОГИЯ

Скарлатина - строгий антропоноз, источником инфицирования является больной человек в острый период болезни и в период реконвалесценции, если имеется реконвалесцентное бактериовыведение. Возможно заражение и от носителей гемолитического стрептококка А, не имевших и не имеющих симптомов скарлатины; частота такого носительства возрастает осенью при формировании детских коллективов.

Механизм передачи инфекции - аэрогенный, преобладающий путь инфицирования - воздушно - капельный во время различных экспираторных актов (чиханье, кашель, крик и т. д.) в виде капельной фазы аэрозоля как фактора передачи возбудителя. Имеет значение пылевой аэрозоль, загрязняющий одежду, постельное белье, игрушки, мебель. Возбудитель сохраняется на них в течение нескольких суток, что повышает риск инфицирования в тесных маленьких по объему помещениях и при скученности людей.

Возможно инфицирование контактным механизмом (актуален при экстрабуккальной скарлатине).

Восприимчивы к скарлатине лица, не имеющие специфического антитоксического иммунитета - дети и взрослые. Дети первых 6-12 мес жизни имеют обычно пассивный иммунитет, приобретенный от матери и болеют очень редко (1-2 % от общего числа больных). Считается также, что чувствительность к возбудителю уменьшается после 20 лет и падает после 40 лет - эти возрастные контингенты редко вовлекаются в эпидемиологический процесс. Индекс восприимчивости равен 0,4.

Скарлатине свойственна осенне-зимняя сезонность.

Иммунитет после скарлатины стойкий, ненапряженный, антитоксический.

ПАТОГЕНЕЗ И ПАТОЛОГОАНАТОМИЧЕСКАЯ КАРТИНА

Входными воротами являются зев и носоглотка. Здесь возбудитель фиксируется и продуцирует токсины. Основным из них являются эритрогенный экзотоксин (токсин Диков, или токсин общего действия, или токсин сыпи), обусловливающий интоксикацию и ответственный за большинство симптомов скарлатины. Он обладает антигенными свойствами и приводит к формированию антитоксического иммунитета. Выделяются также эндотоксины, иногда

называемые токсинами «частного приложения», определяющие инвазивность и агрессивность β-гемолитического стрептококка А. К ним относят стрептолизин, лейкоцидин, энтеротоксин и различные ферменты (стрептокиназа, гиалуронидаза и т.д.). Иммунитет к ним типоспецифичен и нестоек. Все сказанное объясняет тот факт, что повторные случаи скарлатины в общем редки и что человек, переболевший скарлатиной, может легко заразиться другими стрептококковыми инфекциями (рожей, ангинами, пиодермиями и т.д.).

Патогенез скарлатины сложен, в дидактических целях в нем искусственно выделяют токсический, септический (бактериальный), аллергический компоненты. Доминирует токсический компонент. Токсины обусловливают токсемию, которая является причиной генерализованного расширения мелких сосудов во всех органах, в том числе в коже и слизистых оболочках. Отсюда яркая гиперемия кожных покровов и резкое полнокровие языка и зева, которые так типичны для скарлатины. Точечная сыпь - тоже проявление токсемии, результат расширения сосудов кожи, идущих перпендикулярно или тангенциально к поверхности покровов. Одновременно наблюдаются небольшая периваскулярная инфильтрация и умеренный отек дермы. Эпидермис соответственно очажкам гиперемии пропитывается экссудатом, в нем развивается паракератоз, при котором между ороговевшими клетками сохраняется прочная связь [Ивановская Т. Е., 1989]. Этим объясняется отторжение крупных пластин рогового слоя кожи, особенно там, где он самый толстый (ладони, подошвы), что в клинической картине проявляется пластинчатым шелушением в исходе скарлатинозной сыпи. В головном мозге и вегетативных ганглиях возникают расстройства кровообращения и в особо тяжелых случаях - дистрофические изменения нейронов.

В конце 1-й - начале 2-й недели нарастает роль аллергического компонента патогенеза в результате сенсибилизации продуктами жизнедеятельности и особенно распада микроорганизмов. В клинической картине проявления аллергии возможны (но не обязательны) на 2-й, чаще 3-й неделе заболевания. Соответствующая перестройка иммунной системы, нарушение проницаемости защитных барьеров могут привести к развитию гломерулонефрита, артериитов, поражению сердца и другим осложнениям иммунопатологического характера.

С другой стороны, эти изменения иногда способствуют распространению возбудителя из лимфатических образований зева по контакту и гематогенно, в результате чего образуются септические очаги с соответствующей патолого-анатомической картиной. В лимфатическом аппарате зева видны глубокие очаги некрозов, в лимфатических узлах - очаги некроза и лейкоцитарной инфильтрации вплоть до гнойного воспаления. В селезенке развиваются типичные септические изменения. В остальных органах - септическая инфильтрация клетками миелоидного ряда с большим количеством эозинофилов, что типично именно для скарлатины. Развитие гнойного лимфаденита с зонами глубокого некроза может привести к флегмоне шеи с последующей аррозией крупных сосудов и тяжелым кровотечением. Распространение гнойно-некротических процессов в этой области может привести к отитам, ситам височной кости, перейти на твердую мозговую оболочку, венозные синусы с тяжелейшими последствиями.

КЛИНИКА

Инкубационный период продолжается 1-11 дней, в среднем составляет 5-6 дней.

Заболевание начинается остро. Кардинальными признаками скарлатины является лихорадка, поражение зева, первичный лимфаденит и сыпь.

Лихорадка - самый первый симптом скарлатины. Температура тела поднимается внезапно, обычно до высоких цифр - 38-39^0 С и даже 40^0 С, очень часто сопровождается однократной или многократной рвотой. На фоне высокой температуры тела больные остаются подвижными, возбужденными, болтливыми, они бегают, кричат, становятся требовательными и плохо управляемыми. В самых тяжелых случаях ночью развивается бред, а сами больные становятся вялыми, угнетенными.

Пульс частый, степень тахикардии не соответствует высоте температуры тела, превышая обычные соотношения.

Поражение зева при скарлатине представляет собой яркую разлитую гиперемию, охватывающую боковые миндалины (а часто и все кольцо Пирогова-Вальдейера, в которое, кроме боковых, входят носоглоточная миндалина, парные аденоидные образования наружного отверстия евстахиевых труб и язычная миндалина, расположенная у корня языка), дужки, язычок, мягкое небо и заднюю стенку глотки и резко обрывающуюся у того места, где слизистая оболочка мягкого неба переходит в слизистую оболочку, покрывающую твердое небо. Линия обрыва образует заметные неровности края гиперемии. Старые авторы характеризовали подобную картину как «пылающий зев с языками пламени». Иногда на этом фоне видна энантема: очень мелкие, точечные красные пятна, чаще всего в центре мягкого неба, чуть выше язычка.

Иногда, в особенно тяжелых случаях, ко 2-му дню (реже на 3-й день) заболевания на пылающих миндалинах появляются налеты - слизистые, фибринозные и даже некротические. В современных условиях такие налеты встречаются крайне редко.

Резкая гиперемия и отек зева сопровождаются болями в горле, на которые больной жалуется с первых часов заболевания на фоне появившейся лихорадки. В доантибиотическую эру типичные изменения в зеве держались около 6 дней, а при появлении налетов до 8 - 14-го дня. В настоящее время на фоне адекватной антибиотикотерапии и правильного патогенетического лечения сроки поражения зева могут сокращаться.

Упомянутые выше лимфатические образования объединяются в кольцо Пирогова-Вальдейера лимфатическими путями, которые далее соединяют их с регионарными лимфатическими узлами. Первичный лимфаденит тоже является ранним симптомом скарлатины, чаще он двусторонний, реже односторонний. Увеличенные лимфатические узлы плотны на ощупь, слабо болезненны. Чаще увеличиваются передневерхние шейные лимфатические узлы. В современных условиях лимфаденит редко бывает значительным и встречается не у всех больных.

Сыпь появляется, как правило, в первый день болезни, реже на 2-е сутки заболевания. Одной из ее особенностей является то, что она всегда располагается на фоне гиперемированной кожи и лучше всего там, где кожа особенно нежна: на сгибательных поверхностях конечностей, передней и боковой поверхности шеи, боковых поверхностях груди, на животе, внутренней и задней поверхностях бедер со сгущением в местах естественных сгибов - подмышечных, локтевых, паховых, подколенных областях.

Наиболее типичный элемент скарлатинозной сыпи - очень мелкое пятнышко, размером буквально с точку, отсюда ее описание как точечной сыпи (иногда не очень правильно с семантической точки зрения как мелкоточечной). В местах механической травмы, а также в сгибах можно видеть линии (симптом) Пастиа - сгруппированные петехиальные элементы, которые «живут» дольше точечной сосудистой сыпи и позволяют поставить правильный диагноз при запоздалом обращении больного врачу. Редко можно встретить не типичную сыпь в виде очень мелких папул розового цвета - мелкопапулезную сыпь, и совсем редко - так называемую милиарную сыпь, которая имеет вид мельчайших (до 1 мм в диаметре) пузырьков, наполненных серозным содержимым и располагающихся главным образом на коже живота и внутренних поверхностей бедер.

Очень характерно расположение сыпи на лице - она как бы щадит носогубной треугольник, который получил название скарлатинозного (симптом Филатова, который первым указал на эту особенность скарлатины). Видимая бледность кожи в этой области обусловлена раздражением токсином нижней части ганглия тройничного нерва (гассерова ганглия) и соответственно сосудосуживающих волокон III ветви тройничного нерва. Бледность носогубного треугольника особенно подчеркивается горящими щеками и яркими припухшими губами, что придает неповторимое своеобразие внешнему виду больных скарлатиной. Н. Ф. Филатов считал что диагноз скарлатины можно установить во многих случаях, не раздевая больного, по внешнему виду его лица. В типичных случаях это, безусловно, соответствует действительности даже при современном, более легком течении этого заболевания.

При надавливании на покрытую сыпью кожу сыпь исчезает, таким образом можно получить «симптом ладони» (если прижать кожу больного ладонью, ее белый отпечаток некоторое время хорошо виден, но через несколько секунд исчезает, на этом месте вновь обнаруживается сыпь на гиперемированном фоне кожи).

На 4-5-й день болезни (при легких формах и раньше) сыпь начинает бледнеть и исчезает, сменяясь шелушением. Эпидермис отслаивается при скарлатине пластами, особенно на пальцах рук и ног. Пластинчатое шелушение очень характерно для этого заболевания и позволяет в большинстве случаев уверенно ставить ретроспективный диагноз на 2-3-й неделе течения скарлатины.

Весьма характерны при этом заболевании изменения языка. В первые сутки инфекции он покрывается, особенно у корня языка, обильным белым налетом (что обычно наблюдается при всех инфекциях с выраженной интокси-

кацией), но с 3-4-го дня он начинает очищаться с кончика и краев языка, обнажая малинового цвета поверхность с гипертрофированными сосочками. Отсюда название этого симптома - «скарлатинозный малиновый цвет» (за сходство с ягодой малины, а не только за его цвет). К концу 1-й - началу 2-й недели заболевания цвет языка нормализуется, но большие, выступающие сосочки хорошо видны до 3-й недели.

Важное дифференциально-диагностическое значение имеют симптомы симпатикотонии - сухие теплые (горячие) кожные покровы, тахикардия, блестящие глаза, активное поведение больного, выраженный и стойкий белый дермографизм. Нередко это является неоценимым подспорьем в разграничении скарлатины от скарлатиноподобной лихорадки (одной из клинических форм псевдотуберкулеза). При последней дети вялые, с грустными глазами, «мокрые», дермографизм у них обычно красный.

При неосложненном течении заболевания бронхолегочная система не изменяется. Тоны сердца громкие, наблюдается тахикардия, умеренное повышение АД. Печень и селезенка не увеличиваются. При пальпации кишечника обычно никаких изменений обнаружить нельзя, хотя имеется склонность к запорам (что характерно для всякой симпатикотонии). В гемограмме, как правило, обнаруживается умеренный лейкоцитоз с небольшим сдвигом лейкоцитарной формулы влево. СОЭ обычно повышена. Со 2-й недели заболевания возможна эозинофилия. Самые легкие формы скарлатины протекают без гематологических изменений. В осадке мочи могут появляться белок, эритроциты, гиалиновые цилиндры, что свидетельствует об интоксикационном синдроме.

Скарлатина протекает в легкой, средней тяжести и тяжелой форме.

При ***легкой форме*** скарлатины (в наши дни она составляет более 65% случаев заболевания) температура тела поднимается не выше 38,5 С, иногда остается субфебрильной и даже нормальной. Рвота обычно однократная. Жалобы на умеренные боли в горле, недомогание, головную боль. Поражение зева - типичное, без налетов и некрозов, держится 4-5 дней. Точечная сыпь также типична, угасает к 3-4-му дню заболевания и завершается крупнопластинчатым шелушением. Регионарные лимфадениты встречаются редко, если они есть, то увеличение шейных лимфатических узлов незначительное, болезненность их умеренная. В гемограмме нормоцитоз или небольшой лейкоцитоз. В осадке мочи изменений может не быть.

Средней тяжести форма скарлатины встречается в трети всех случаев заболевания. Она характеризуется более выраженной интоксикацией, повышением температуры тела с ознобами и жаром до 39º С и выше, что сопровождается повторной рвотой. Поражение зева ярко выражено; на фоне пылающего зева у некоторых больных можно видеть выпот в лакуны или нагноившиеся фолликулы миндалин. Сыпь типична, сохраняется в течение 5-6 дней, иногда можно видеть единичные или сгруппированные петехии. В гемограмме - лейкоцитоз со сдвигом лейкоцитарной формулы влево, эозинофилия непостоянна. В осадке мочи иногда появляются следы белка, эритроциты, гиалиновые цилиндры как свидетели интоксикации.

Тяжелая форма скарлатины протекает либо с преобладанием симптомов выраженной интоксикации (токсическая форма), либо с септическими проявлениями (септическая форма), либо с сочетанием крайних степеней интоксикации и септических очагов (токсикосептическая форма). При токсической форме температура тела бурно повышается до 40-41º С и выше, синдром интоксикации представлен ярко и во всем объеме, с многократной рвотой и преобладанием угнетения ЦНС (у некоторых больных возможно ее возбуждение с бредом, менингизмом и судорогами). Все характерные для скарлатины симптомы также выражены во всей своей полноте и яркости, часто встречаются геморрагические элементы сыпи наряду с типичной точечной экзантемой. Тахикардия доходит до 150-180 в минуту, тоны сердца приглушаются, у некоторых больных развивается коллапс. В моче - протеинурия, гематурия, цилиндрурия.

Септическая форма - исключительная редкость в настоящее время, она развивается у детей младшего возраста, ослабленных той или иной сопутствующей патологией, страдающих первичными или вторичными иммунодефицитами. В зеве на фоне типичных скарлатинозных изменений в силу нарастающих микроциркуляторных расстройств возникают некрозы, быстро присоединяется условно- патогенная микрофлора и развивается гнойно-некротическая ангина. Стрептококки преодолевают кольцо Пирогова-Вальдейера, развивается лимфаденит, per contituitatem возбудители попадают в близлежащие органы: возникают гнойные отиты, гаймориты, этмоидиты, мастоидиты. При прорыве стрептококков в кровь может развиться сепсис в форме септицемии или септикопиемии. Как и при других этиологических формах сепсиса, увеличиваются печень и селезенка.

Отдельно рассматривается ***экстрабуккальная скарлатина***, при которой входными воротами являются раневая, ожоговая, послеродовая, послеоперационная поверхности. В этом случае поражения зева и шейных лимфаденитов не бывает. Сыпь нередко распространяется по всему телу именно от ворот инфекции. В остальном клиническая картина скарлатины остается типичной

Осложнения. В полном соответствии с патогенезом скарлатины ее осложнения можно разделить на три группы. Первая группа токсических осложнений включает развитие острой сердечно-сосудистой недостаточности (коллапс) и токсико-инфекционного шока. Во вторую группу входят ранние и поздние (вторичные) бактериальные осложнения: отиты, гнойные лимфадениты, заглоточные абсцессы, синуситы, мастоидиты, абсцесс головного мозга, синустромбоз, менингит медиастинит, флегмона желудка, сепсис и т. д. Третья группа - так называемые аллергические (иммунопатологические) осложнения: постстрептококковый гломерулонефрит (с возможным исходом в нефросклероз), миокардит, васкулиты, бородавчатый эндокардит, Фибриноиды интимы крупных сосудов с исходом в склероз.

Особенности скарлатины у взрослых. Взрослые болеют редко; необходима настороженность в отношении экстрабуккальной скарлатины в хирургических, акушерских, ожоговых стационарах. Течение заболевания у взрослых обычно легкое, осложнения крайне редки.

ПРОГНОЗ

В абсолютном большинстве случаев благоприятный, при наличии осложнений - серьезный. Летальность в настоящее время близка к нулю, но в последние годы описано несколько случаев смерти детей от септической и токсико-септической форм скарлатины, протекавших на фоне респираторных вирусных инфекций.

ДИАГНОСТИКА

Базируется на клинических данных с учетом эпидемиологического анамнеза. Подтверждают диагноз выделением β-гемолитического стрептококка группы А. Серологическая диагностика скарлатины не разработана. Для ранней диагностики возможных осложнений делают повторные анализы мочи, контроль гемограммы.

ДИФФЕРЕНЦИАЛЬНАЯ ДИАГНОСТИКА

Скарлатину следует отличать от кори, краснухи, псевдотуберкулеза, лекарственных дерматитов. В редких случаях развития фибринозных налетов и особенно при их выходе за пределы миндалин заболевание необходимо дифференцировать от дифтерии.

Скарлатину отличают яркая разлитая гиперемия ротоглотки («пылающий зев»), резко ограниченная в месте перехода слизистой оболочки на твердое небо, ярко-красный язык с малиновым оттенком и гипертрофированными сосочками («малиновый язык»), мелкоточечные элементы сыпи на общем гиперемированном фоне, сгущение сыпи в виде темно-красных полос на кожных складках в местах естественных сгибов, отчетливо выраженный белый дермографизм, бледный носогубный треугольник (симптом Филатова). При надавливании на кожу ладонью сыпь в этом месте временно исчезает («симптом ладони»), положительны эндотелиальные симптомы. После исчезновения экзантемы отмечают мелкочешуйчатое шелушение кожи (на ладонях и подошвах крупнопластинчатое).

ЛЕЧЕНИЕ

В настоящее время лечение скарлатины осуществляют на дому, за исключением тяжелых и осложненных случаев. Необходимо соблюдать постельный режим в течение 7-10 дней. Этиотропным препаратом выбора остается пенициллин в суточной дозе 6 млн.ЕД (для взрослых) курсом 10 сут. Альтернативные препараты- макролиды (эритромицин в дозе 250 мг 4 раза в сутки или 500 мг 2 раза в сутки) и цефалоспорины 1 поколения (цефазолин по 2-4 г/сут). Курс лечения также составляет 10 дней. При наличии противопоказаний к указанным препаратам можно применять полусинтетические пенициллины, линкозамиды. Назначают полоскания горла раствором фурацилина (1:5000), настоями ромашки, календулы, эвкалипта. Показаны витамины и антигистаминные средства в обычных терапевтических дозах.

ПРОФИЛАКТИКА

Основу профилактики респираторных стрептококковых инфекций в организованных коллективах составляют планомерные и систематические лечебно-диагностические мероприятия. Ранняя и активная диагностика, изоляция и полноценное этиотропное лечение заболевших играют в этих условиях решающую роль. Реальность такого подхода объясняется тем, что возбудители сохраняют чувствительность к действию пенициллина и его производных. Применение препаратов группы пенициллина может предупредить групповые заболевания скарлатиной и ревматизмом, а также снизить заболеваемость ангиной и стрептококковыми острыми респираторными заболеваниями.

Для купирования вспышек респираторных стрептококковых заболеваний в организованных коллективах необходимо лечение препаратами пенициллинового ряда больных не только явными, но и скрытыми формами стрептококковой инфекции. С этой целью всем контактировавшим лицам вводят однократно внутримышечно бициллин-5 (дошкольникам-750 000 ЕД, школьникам и взрослым- 1 500 000 ЕД) или бициллин-1 (дошкольникам- 600 000 ЕД, школьникам и взрослым- 1 200 000 ЕД.

Санитарно- гигиенические мероприятия в организованных детских и взрослых коллективах, а также в больничных условиях (уменьшение численности коллектива, его скученности, общие санитарные мероприятия, дезинфекционный режим) уменьшают вероятность реализации воздушно-капельной и

ВОПРОСЫ ДЛЯ САМОКОНТРОЛЯ

1. Понятие «Детские инфекционные болезни у взрослых» (ДИВ).
2. Этиология и эпидемиология кори.
3. Основные симптомы кори у взрослых.
4. Дифференциальная диагностика кори.
5. Меры профилактики при кори у взрослых.
6. Лечение кори.
7. Этиология и эпидемиология краснухи.
8. Симптоматика краснухи у взрослых.
9. Патогенез краснухи.
10. Дифференциальный диагноз краснухи у взрослых.
11. Краснуха у беременных и меры ее профилактики.
12. Этиология и эпидемиология ветряной оспы.
13. Признаки ветряной оспы у взрослых.
14. Дифференциальный диагноз ветряной оспы.
15. Этиология и эпидемиология эпидемического паротита.
16. Клиника паротитной инфекции у взрослых.
17. Дифференциальный диагноз при паротитной инфекции.
18. Лечение и профилактика эпидемического паротита.
19. Этиология и эпидемиология скарлатины.
20. Клинические признаки скарлатины у взрослых.
21. Дифференциальный диагноз скарлатины.
22. Лечение и меры профилактики при скарлатине.
23. Клинические и эпидемиологические показания для госпитализации взрослых пациентов, заболевших ДИВ.

ЗАДАЧА № 1

Больная В. 28 лет жалуется на слабость, повышение температуры тела, головную боль, насморк, кашель. Больной себя считает со вчерашнего вечера, когда почувствовала познабливание, недомогание, насморк. Температуру тела вчера не измеряла. Сегодня утром температура 37,6º С, появился кашель, головная боль, чувство жжения кожи лица. При осмотре: выраженная эритема на лице, На коже спины, ягодиц, разгибательных поверхностях конечностей обильная мелкопятнистая сыпь бледно-розового цвета. Отдельные элементы на спине сливаются. На коже груди и живота сыпи значительно меньше. Конъюктивы умеренно гиперемированы. На слизистой мягкого неба энантема. Лимфатические узлы увеличены: заты-

лочные до 1 см, околоушные до 0,5 см, паховые до 0,5 см, безболезненные, подвижные. Дыхание через нос затруднено. В легких дыхание везикулярное. Тоны сердца ясные, пульс 82 в мин, АД 110/70 мм. рт. ст. Язык чистый. Живот мягкий, безболезненный, печень и селезенка не увеличены. Менингеальных явлений нет. Физиологические отправления в норме.

1) Поставить диагноз и составить алгоритм постановки диагноза.

ЗАДАЧА № 2

Больной П. 25 лет жалуется на слабость, головную боль, ломоту в теле, кашель, слезотечение, сыпь на коже. Болен с 6.03, заболевание началось с кашля, насморка, охриплости голоса, боли в горле. Беспокоила слабость, повышение температуры тела до 38,0 С. Обратился к врачу 8.03. Поставлен диагноз «грипп». Принимал бисептол, витамины. Эти явления продолжались в течение недели, были также неприятные ощущения в области глаз, слезотечение, отечность лица и век, мучительный кашель. Повторно обратился к врачу, к приходу которого температура вновь повысилась до 39,0 С. При осмотре констатированы обильная сливная пятнисто-папулезная сыпь красного цвета на лице и шее. Лицо одутловато. Конъюктивы ярко гиперемированы, склеры инъецированы. Слизистая ротоглотки гиперемирована. На деснах белесоватые наложения, легко снимающиеся шпателем. В легких жесткое дыхание, рассеянные сухие хрипы. Тоны сердца приглушены, ритмичные. Пульс 88 уд/мин, АД 105/70 мм рт.ст. Живот мягкий, безболезненный, Печень и селезенка не увеличены. Физиологические отправления в норме. Менингеальных явлений нет. Госпитализирован. В дальнейшем в течение 3 дней сыпь распространилась на грудь, плечи и бедра, предплечья и голени. Впоследствии на месте сыпи возникла пигментация, на коже лица – отрубевидное шелушение.

1) Необходимо поставить диагноз и составить алгоритм постановки диагноза.

ЗАДАЧА № 3

Больной Ш. 20 лет, рядовой, обратился в медсанчасть 3.04 с жалобами на припухлость в области правой околоушной железы, боль при открывании рта, сухость во рту, слабость, головную боль. Заболел 1. 04, когда появилась слабость, познабливание, боль в области правого сосцевидного отростка. Сегодня заметил припухлость в области угла нижней челюсти справа, боль при открывании рта, головная боль усилилась. Объективно: состояние средней тяжести, температура 37,7 С. Кожные покровы чистые. Периферические лимфатические узлы не пальпируются. Незначительный тризм жевательной мускулатуры. В правой околоушной области отмечается припухлость, слегка болезненная при пальпации, 6/6 см, округлой формы, тестоватой консистенции, не связана с подлежащими тканями, кожа над ней натянута и лоснится. Слизистая глотки чистая, не гиперемирована. В легких жесткое дыхание, хрипов нет. Тоны сердца ритмичные. Пульс 86 уд/мин, ритмичный, удовлетворительных свойств. АД 110/70 мм рт.ст. Язык суховат, густо обложен белым налетом. Живот мягкий, безбо-

лезненный при пальпации. Печень и селезенка не пальпируются. Менингеальных явлений нет. Госпитализирован.

В дальнейшем температура тела стала снижаться и нормализовалась 5.04. Припухлость в области слюнной околоушной железы стала постепенно исчезать. Обращало на себя внимание повышение диастазы мочи до 512 ед. 7.04 появился озноб, температура тела 39,1º С, головная боль, ноющие боли в правом яичке, паховой области. При осмотре: правое яичко увеличено в размере по сравнению с левым в 2 раза, плотное, болезненное, кожа мошонки гиперемирована.

1) Поставить диагноз и составить алгоритм постановки диагноза.

ЗАДАЧА № 4

Больная Г. 28 лет, врач-стоматолог, обратилась к участковому врачу с жалобами на повышенную температуру тела, головную боль, боль в горле, сыпь на теле, сопровождающуюся зудом. Считает себя больной с утра 11.08, когда почувствовала боль в горле при глотании, сильную головную боль, озноб. Температура тела повысилась до 39,9º С. Через несколько часов отметила тошноту, была однократная рвота. Участковый врач диагностировал фолликулярную ангину и назначил олететрин. К вечеру больная заметила на теле сыпь. Утром 12.07 температура тела 38,0º С, сыпь сохранялась, был небольшой зуд кожи. При повторном осмотре было выявлено состояние средней тяжести. На гиперемированном фоне кожи туловища и конечностей обильная мелкоточечная сыпь красного цвета, сгущающаяся в области подмышечных впадин и подколенных ямок, нижней части живота и паховых областях, на сгибательных поверхностях рук. Носогубный треугольник бледный. Выявляется белый дермографизм. Слизистая миндалин, дужек, язычка, мягкого неба ярко-красного цвета с четкой границей. Миндалины увеличены до 1 см, в лакунах имеются гнойные наложения, снимающиеся шпателем при надавливании. Тонзиллярные лимфатические узлы увеличены и болезненны. В легких – без патологии. Тоны сердца ритмичные, приглушены. Пульс 92 уд/ мин. ритмичный. Язык покрыт белым налетом, суховат. Живот мягкий, безболезненный во всех отделах. Печень и селезенка не пальпируются. Стул и диурез в норме. Менингеальных явлений нет.

1) Поставить диагноз и составить алгоритм постановки диагноза.

ЗАДАЧА № 5.

Больной К.25 лет, студент, заболел остро 11.06, когда появилась слабость, чувство разбитости, головная боль, температура тела повысилась до 37.4 С. 12.06 и 13.06 присоединились тошнота, боль в горле, редкий кашель, температура тела поднялась до 38,5 С. 13.06 обратился к врачу. Был диагностирован «грипп», назначено симптоматическое лечение. 13.06 к вечеру заметил на волосистой части головы единичные гнойнички, 14.06 самочувствие несколько улучшилось, новых элементов сыпи не замечал, температура тела 37,6º С, 15.06 отмечал озноб, сильную головную боль, тошноту, была однократная рвота, на туловище и конечностях появилась обильная сыпь. обратился к участковому

врачу повторно и был госпитализирован. Объективно при осмотре: состояние средней тяжести, температура тела 39,1º С , вял. На коже волосистой части головы, лица, шеи, туловища и конечностей имеется обильная полиморфная сыпь (пятна, папулы, везикулы, пустулы и корочки с преобладанием везикул и пустул), сопровождающаяся зудом. Элементы сыпи расположены на неизмененном фоне кожи, мягкие на ощупь, кожа под ними не инфильтрирована. Слизистая глотки гиперемирована. На слизистой твердого и мягкого неба, внутренней поверхности щек имеются везикулы и эрозии. Пальпируются увеличенные, умеренно болезненные шейные лимфатические узлы. В легких без патологии. Тоны сердца ритмичные, приглушены. Пульс 104 уд/мин., ритмичный, удовлетворительных свойств, АД 110/70 мм рт.ст. Язык суховат, обложен белым налетом. Живот мягкий, безболезненный при пальпации во всех отделах. Печень и селезенка не пальпируются. Очаговых и менингеальных явлений нет.

1) Поставить диагноз и составить алгоритм постановки диагноза.

ОТВЕТЫ

Задача № 1

Алгоритм диагностики краснухи у взрослых.

Симптомы интоксикации

Есть

Исследование продолжается

Умеренно выраженные катаральные явления

Есть

Исследование продолжается

Появление мелкопятнистой сыпи с 1-3 дня болезни с распространением ее по телу в течение нескольких часов.

Есть

Исследование продолжается

Генерализованная лимфаденопатия с преимущественным увеличением затылочных и шейных лимфатических узлов.

Есть

Исследование закончено.

Диагноз: Краснуха.

Задача № 2

Алгоритм диагностики кори у взрослых

Симптомы интоксикации

Есть

Исследование продолжается

Выраженные катаральные явления

Есть

Исследование продолжается

Пятна Филатова-Коплика

Есть

Исследование продолжается
Пятнисто-папулезная сыпь с 1-8 дня болезни с поэтапным распространением
Есть
Исследование закончено
Диагноз: Корь, период высыпания.

Задача № 3
Алгоритм диагностики паротитной инфекции у взрослых.

Симптомы интоксикации
Есть
Исследование продолжается
Боль жевании и открывании рта в области слюнных желез.
Есть
Исследование продолжается
Увеличение одной или нескольких слюнных желез (околоушных, подчелюстных)
Есть
Исследование продолжается
Одновременное поражение слюнных желез и поджелудочной железы, яичек молочных желез, развитие серозного менингита
Есть
Исследование закончено

Диагноз: Эпидемический паротит.

Задача № 4
Алгоритм диагностики скарлатины у взрослых.
Симптомы интоксикации
Есть
Исследование продолжается
С 1-2-го дня сыпь мелкоточечная, на гиперемированном фоне с локализацией на сгибательных поверхностях конечностей, шее, в подмышечных впадинах, внизу живота, сгущение в местах кожных складок.
Есть
Исследование продолжается
Стойкий белый дермографизм
Есть
Исследование продолжается
Появление со 2-4-го дня болезни «малинового» «сосочкового» языка
Есть, исследование продолжается.
Появление с 5-6-го дня болезни пластинчатого шелушения кожи кистей, стоп, туловища, а на лице и шее – отрубевидного шелушения.
Есть

Исследование закончено
Диагноз: Скарлатина.

Задача № 5.

Алгоритм диагностики ветряной оспы у взрослых.
Симптомы интоксикации
Есть
Исследование продолжается
Полиморфизм сыпи на 1-3-й день болезни (на лице и волосистой части головы с распространением по туловищу и конечностям)
Есть
Исследование продолжается
Полиморфизм сыпи (наличие на отдельном участке кожи пятен, папул, везикул, пустул, корочек)
Есть
Исследование закончено

Диагноз: Ветряная оспа.