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HERPETIC STOMATITIS IN CHILDREN

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КАФЕДРА СТОМАТОЛОГИИ ДЕТСКОГО ВОЗРАСТА

Н. В. Шаковец, О. С. Романова

ГЕРПЕТИЧЕСКИЙ СТОМАТИТ У ДЕТЕЙ

HERPETIC STOMATITIS IN CHILDREN

Учебно-методическое пособие

Минск БГМУ 2021

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Посвящено проблеме герпетической инфекции у детей. Освещены вопросы химико-биологических свойств герпесвирусов, этиологии и патогенеза развития острой и хронической герпетической инфекции, подробно изложены особенности клинических проявлений различных форм герпетического стоматита. Представлены современные подходы к лечению и профилактике герпетической инфекции у детей.

Предназначено для студентов 4-го курса медицинского факультета иностранных учащихся, обучающихся на английском языке по специальности 1-79 01 07 «Стоматология».

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INTRODUCTION

Viral infections in the oropharynx are common. One widespread viral pathogen that infects the epithelium of this region is herpes simplex virus (HSV) (Tables 1, 2). Herpetic stomatitis is an infectious disease caused by the herpesvirus hominis. Herpes simplex virus 1 and 2 (HSV-1 and HSV-2), also known by their taxonomical names Human alphaherpesvirus 1 and Human alphaherpesvirus 2, are two members of the human Herpesviridae family, a set of new viruses that produce viral infections in the majority of humans. Both HSV-1 (which produces most cold sores) and HSV-2 (which produces most genital herpes) are common and contagious. They can be spread when an infected person begins shedding the virus. Almost 100 % of urban adult populations are carriers of this virus and have neutralizing antibodies to the virus. This acquired immunity suggests that the majority of childhood infections are subclinical (Fig. 1).

Table 1

Herpesvirus

Subgroup	Type
α	herpesvirus hominis type I
	herpesvirus hominis type II
	virus zoster
β	cytomegalovirus
	herpesvirus hominis type VI
	herpesvirus hominis type VII
γ	virus Epstein-Barr
	herpesvirus hominis type VIII

Table 2

Herpesviruses and human pathology

Herpesviruses			Diseases associated with primary infection	Diseases arising from activation of latent infection
Subgroup		Type		
α	– tropism to ectodermal tissues; – short reproductive cycle; – rapid spread; – lytic reproduc-tion cycle; – latency mainly in the nerve ganglia	**herpesvirus hominis type I** (herpes simplex virus 1, HSV-1)	– Congenital and neonatal herpes; – acute herpetic stomatitis; – herpes of the skin of the face and upper extremities; – ophthalmic herpes; – meningoencephalitis	– Herpetic stomatitis; – exudative multiform erythema; – ophthalmic herpes; – meningoencephalitis

Continuation of Table 2

Herpesviruses			**Diseases associated with primary infection**	**Diseases arising from activation of latent infection**
Subgroup		**Type**		
		herpesvirus hominis type II (*herpes simplex virus* 2, HSV-2)	– congenital and neonatal herpes; – genital herpes; – herpes of the skin of the lower extremities; – meningoencephalitis	– herpes of the genitals; – herpes of the skin of the thighs, buttocks, lower extremities; – myelitis, encephalitis
		virus zoster (HHV-3, *herpes zoster, varicella zoster virus*, VZV)	– chicken pox; – pre- and perinatal infection	shingles
β	– tropism for lymphoid and glandular tissues; – long reproductive cycle; – an increase in infected cells; – long-term persistence; – latency	**cytomegalovirus** (HHV-5, *cytomegalovirus*, CMV)	– teratogenic effects; – congenital infection; – immunodeficiency; – pathology of the respiratory system; – ulcerative necrotizing gingivostomatitis; –disseminated infection	– retinitis; – colitis; – stomatitis; – encephalitis
		herpesvirus hominis type VI (HHV-6)	– HIV cofactor; – chronic fatigue syndrome; – oral carcinoma; – cervical carcinoma	– systemic pathology during organ and tissue transplantation; – Langerhans cell histiocytosis
		herpesvirus hominis type VII (HHV-7)	– chronic fatigue syndrome; – exanthema of newborns	– systemic pathology during organ and tissue transplantation
γ	– lymphotropicity (specificity for T- or B-lymphocytes); – tissue proliferation and oncogenesis; latency	**virus Epstein-Barr** (HHV-4, *Epstein–Barr virus*, EBV)	– Infectious mononucleosis; – B-lymphoproli-ferative diseases; – carcinoma; – lymphoepithelioma	– nasopharyngeal carcinoma; – Berkitt's lymphoma; – idiopathic lympho-cytic pneumonia; – leukoplakia
		herpesvirus hominis type VIII (HHV-8)	unknown	– Sakoma Kaposi; – primary spreading lymphoma

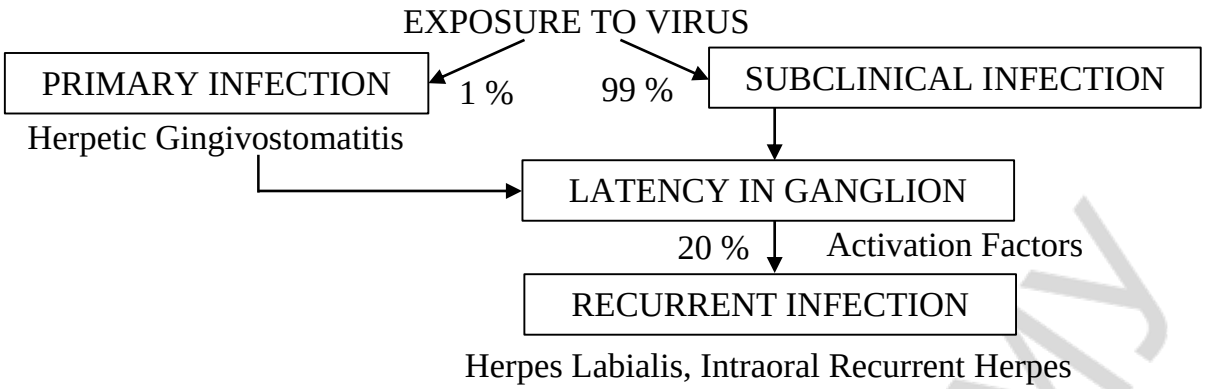

Figure 1. Natural history of herpes simplex virus type 1

CLASSIFICATION OF HERPESVIRUS INFECTION

The herpes simplex 1 genomes can be classified into six clades. Four of these occur in East Africa, one in East Asia and one in Europe and North America. This suggests that the virus may have originated in East Africa. The most recent common ancestor of the Eurasian strains appears to have evolved ~60,000 years ago. The East Asian HSV-1 isolates have an unusual pattern that is currently best explained by the two waves of migration responsible for the peopling of Japan. Herpes simplex 1 and 2 diverged about 6 million years ago.

In Belarus each 7th child has recurrent forms of herpetic infection after acute herpetic stomatitis.

International classification ICD-10:

B00 HERPESVIRAL [HERPES SIMPLEX] INFECTIONS
B00.0 Eczema herpeticum
B00.1 Herpesviral gingivostomatitis and pharyngotonsilitis
B00.3 Herpesviral meningitis
B00.4 Herpesviral encephalitis
B00.5 Herpesviral ocular disease
B00.7 Disseminated herpesviral disease
B00.8 Other form of herpesviral infections
B00.9 Herpesviral infection unspecified
Excludes 1: Congenital herpesviral infections (P35.2)
Excludes 2: Anogenital herpesviral infection (A60)
Gammaherpesviral mononucleosis (B27.0)
Herpangina (B08.5)

CHARACTERISTIC OF HERPESVIRUS

Many of those who are infected never develop symptoms. Symptoms, when they occur, may include watery blisters on the skin or mucous membranes of the mouth, lips, nose, or genitals. Lesions heal with a scab characteristic of

herpetic disease. Sometimes, the viruses cause mild or atypical symptoms during outbreaks. However, they can also cause more troublesome forms of herpes simplex. As neurotropic and neuroinvasive viruses, HSV-1 and -2 persist in the body by hiding from the immune system in the cell bodies of neurons. After the initial or primary infection, some infected people experience sporadic episodes of viral reactivation or outbreaks. In an outbreak, the virus in a nerve cell becomes active and is transported via the neuron's axon to the skin, where virus replication and shedding occur and cause new sores.

HSV-1 is a double-stranded DNA virus with a genome size of 152 kb encoding for at least 84 different polypeptides. During an in vitro acute infection, the lytic nature of the virus is driven by a sequential cascade of genes (referred to as lytic genes) expressed collectively over the course of the first 8–12 hours following entry into the host cell and includes the immediate early or α genes, early or β genes, and late or γ genes. It is now appreciated that many of these genes encode proteins that serve dual functions: assist in the replication of virus and counter the innate or adaptive immune response to the pathogen. Ultimately, the success rate of the virus within the human host is dependent upon its ability to establish a latent infection and then reactivate at opportune times and shed into bodily secretions that are passed vertically or horizontally to a naive patient.

FEATURES OF α-HERPESVIRUS HOMINIS

– Contain DNA.
– Group of neuroviruses.
– Live in epithelial cells of the host.
– Short reproductive cycle.
– Quick spreading.
– Latent period predominantly in nervous ganglions.
– Lytic cycle of multiplication.

VIRAL STRUCTURE

Animal herpes viruses all share some common properties. The structure of herpes viruses consists of a relatively large, double-stranded, linear DNA genome encased within an icosahedral protein cage called the capsid, which is wrapped in a lipid bilayer called the envelope. The envelope is joined to the capsid by means of a tegument. This complete particle is known as the virion. HSV-1 and HSV-2 each contain at least 74 genes (or open reading frames, ORFs) within their genomes, although speculation over gene crowding allows as many as 84 unique protein coding genes by 94 putative ORFs (Fig. 2). These genes encode a variety of proteins involved in forming the capsid, tegument and envelope of the virus, as well as controlling the replication and infectivity of the virus (Fig. 3).

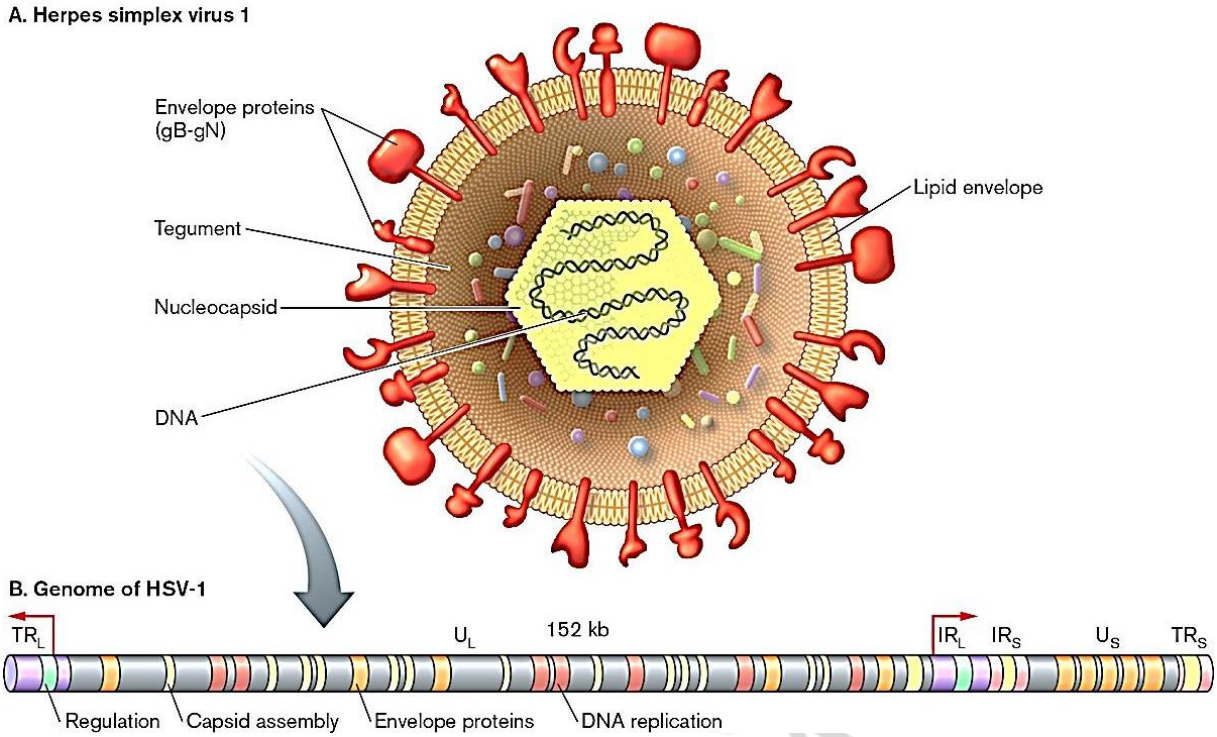

Figure 2. Scheme of the herpes simplex virus 1

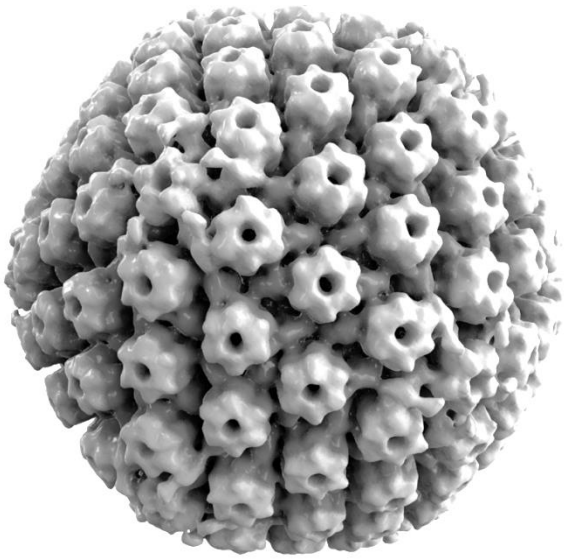

Figure 3. 3D reconstruction of the HSV-1 capsid

The genomes of HSV-1 and HSV-2 are complex and contain two unique regions called the long unique region (UL) and the short unique region (US). Of the 74 known ORFs, UL contains 56 viral genes, whereas US contains only 12. Transcription of HSV genes is catalyzed by RNA polymerase II of the infected host. Immediate early genes, which encode proteins that regulate the expression of early and late viral genes, are the first to express the following infection. Early gene expression follows, to allow the synthesis of enzymes involved in

DNA replication and the production of certain envelope glycoproteins. Expression of late genes occurs last; this group of genes predominantly encode proteins that form the virion particle.

Entry of HSV into a host cell involves several glycoproteins on the surface of the enveloped virus binding to their transmembrane receptors on the cell surface (Fig. 4). The envelope covering the virus particle then fuses with the cell membrane, creating a pore through which the contents of the viral envelope enters the host cell.

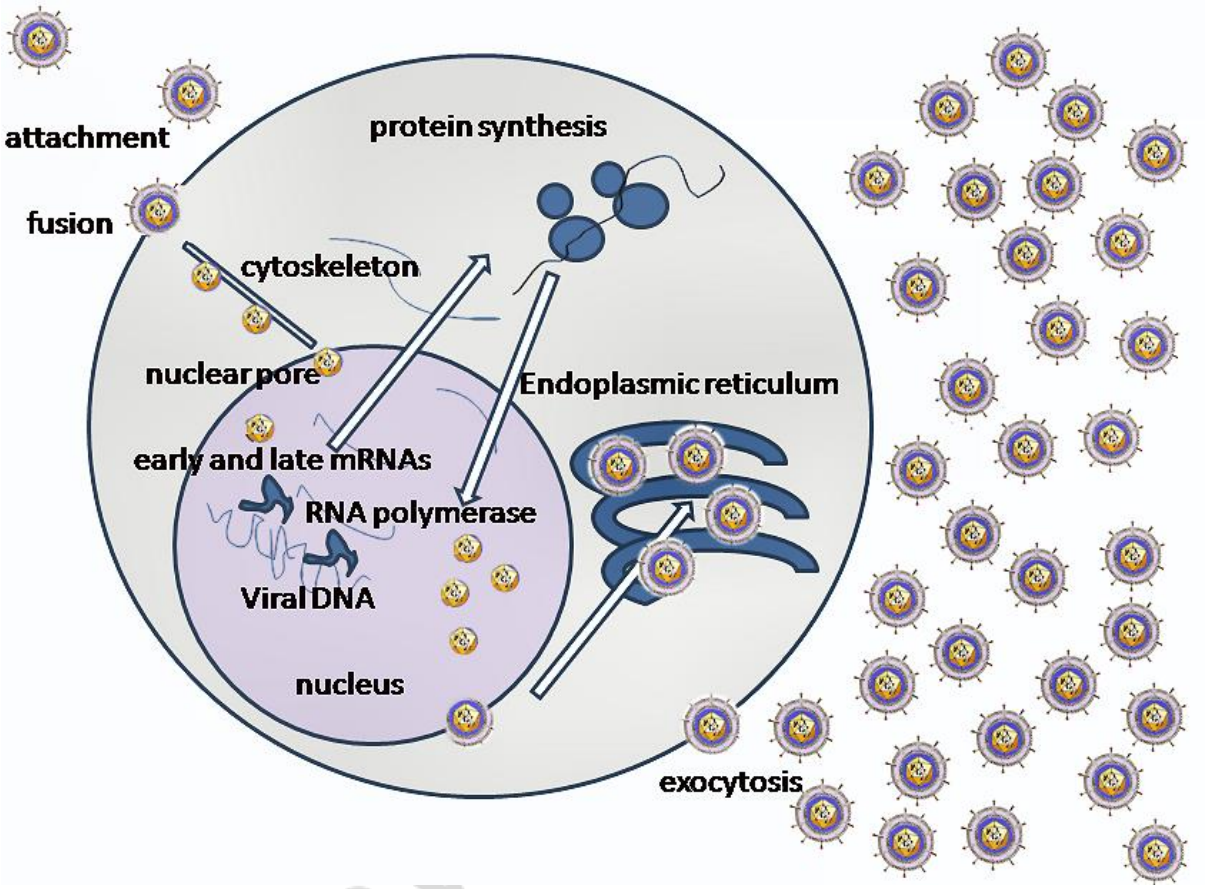

Figure 4. Virus replication in the cell

The sequential stages of HSV entry are analogous to those of other viruses. At first, complementary receptors on the virus and the cell surface bring the viral and cell membranes into proximity. Interactions of these molecules then form a stable entry pore through which the viral envelope contents are introduced to the host cell. The virus can also be endocytosed after binding to the receptors, and the fusion could occur at the endosome. In electron micrographs, the outer leaflets of the viral and cellular lipid bilayers have been seen merged; this hemi fusion may be on the usual path to entry or it may usually be an arrested state more likely to be captured than a transient entry mechanism.

GENETIC INOCULATION

After the viral capsid enters the cellular cytoplasm, it is transported to the cell nucleus. Once attached to the nucleus at a nuclear entry pore, the capsid ejects its DNA contents via the capsid portal. The capsid portal is formed by 12 copies of portal protein, UL6, arranged as a ring; the proteins contain a leucine zipper sequence of amino acids, which allow them to adhere to each other. Each icosahedral capsid contains a single portal, located in one vertex. The DNA exits the capsid in a single linear segment.

REPLICATION

Following infection of a cell, a cascade of herpes virus proteins, called immediate-early, early, and late, is produced. Research using flow cytometry on another member of the herpes virus family, Kaposi's sarcoma-associated herpesvirus, indicates the possibility of an additional lytic stage, delayed-late. These stages of lytic infection, particularly late lytic, are distinct from the latency stage. In the case of HSV-1, no protein products are detected during latency, whereas they are detected during the lytic cycle.

The early transcribed proteins are used in the regulation of genetic replication of the virus. On entering the cell, an α-TIF protein joins the viral particle and aids in immediate-early transcription. The virion host shutoff protein (VHS or UL41) is very important to viral replication. This enzyme shuts off protein synthesis in the host, degrades host mRNA, helps in viral replication, and regulates gene expression of viral proteins. The viral genome immediately travels to the nucleus, but the VHS protein remains in the cytoplasm.

The late proteins form the capsid and the receptors on the surface of the virus. Packaging of the viral particles including the genome, core and the capsid — occurs in the nucleus of the cell. Here, concatemers of the viral genome are separated by cleavage and are placed into formed capsids. HSV-1 undergoes a process of primary and secondary envelopment. The primary envelope is acquired by budding into the inner nuclear membrane of the cell. This then fuses with the outer nuclear membrane, releasing a naked capsid into the cytoplasm. The virus acquires its final envelope by budding into cytoplasmic vesicles.

PATHOGENESIS OF HERPESVIRUS INFECTION

Necrosis sites appear in organs and tissues as the result of:
– cells damage by the virus;
– immune destruction of the cells;
– formation of the blood clots in capillary.
Cytopathic changes (Fig. 5, 6, 7):
– ballooning degeneration or cytoplasmic vacuolization;
– nuclear chromatolysis;

– cellular lyses;

– nuclear and/or cytoplasmic inclusion bodies (viral aggregates or foci where viral replication has been completed).

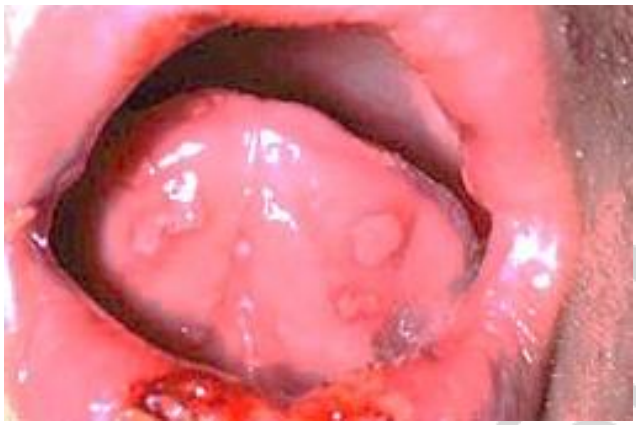

Figure 5. Vesicules with exudat and inflamatory cells

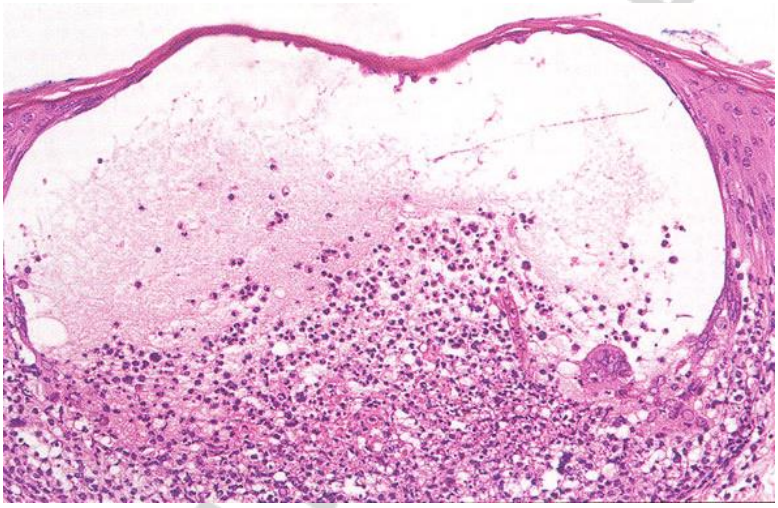

Figure 6. The infected cells fuse and form multinucleated cells containing herpesvirus nuclear inclusions

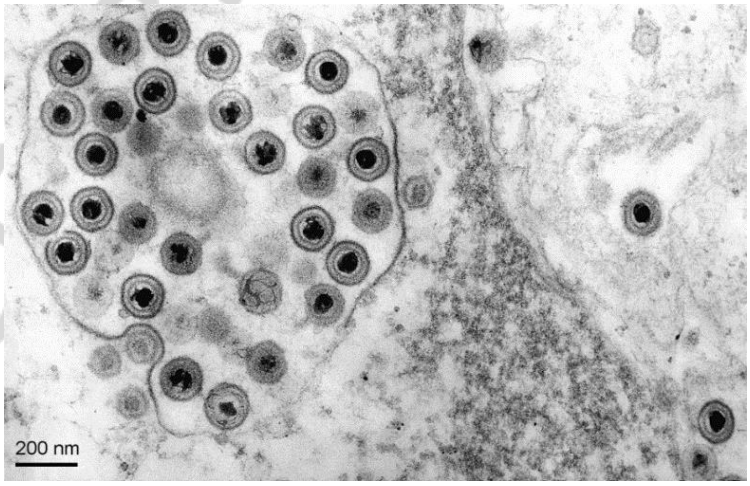

Figure 7. New virus generation in the cell

Factors reducing antiviral immunity:

– hormonal imbalance (puberty, menstruation);

– adverse environmental factors;

– the quality of nutrition (deficiency of protein, vitamins, trace elements);

– immunosuppressive effects of disease associated with the pathology of the immune system (concurrent infectious, endocrine and autoimmune diseases, blood pathology);

– immunosuppressive therapy in autoimmune diseases, cancer and transplant of organs and tissues;

– physical or emotional stress;

– exposure to ultraviolet light;

– nerve or tissue damage.

Influence of the herpesvirus on the host:

– Intoxication;

– Necrosis;

– Immunosuppression;

– Disorder of blood coagulation;

– Activation of Bacteria and Candida;

– Sensibilisation (Fig. 8);

– GIT disorders.

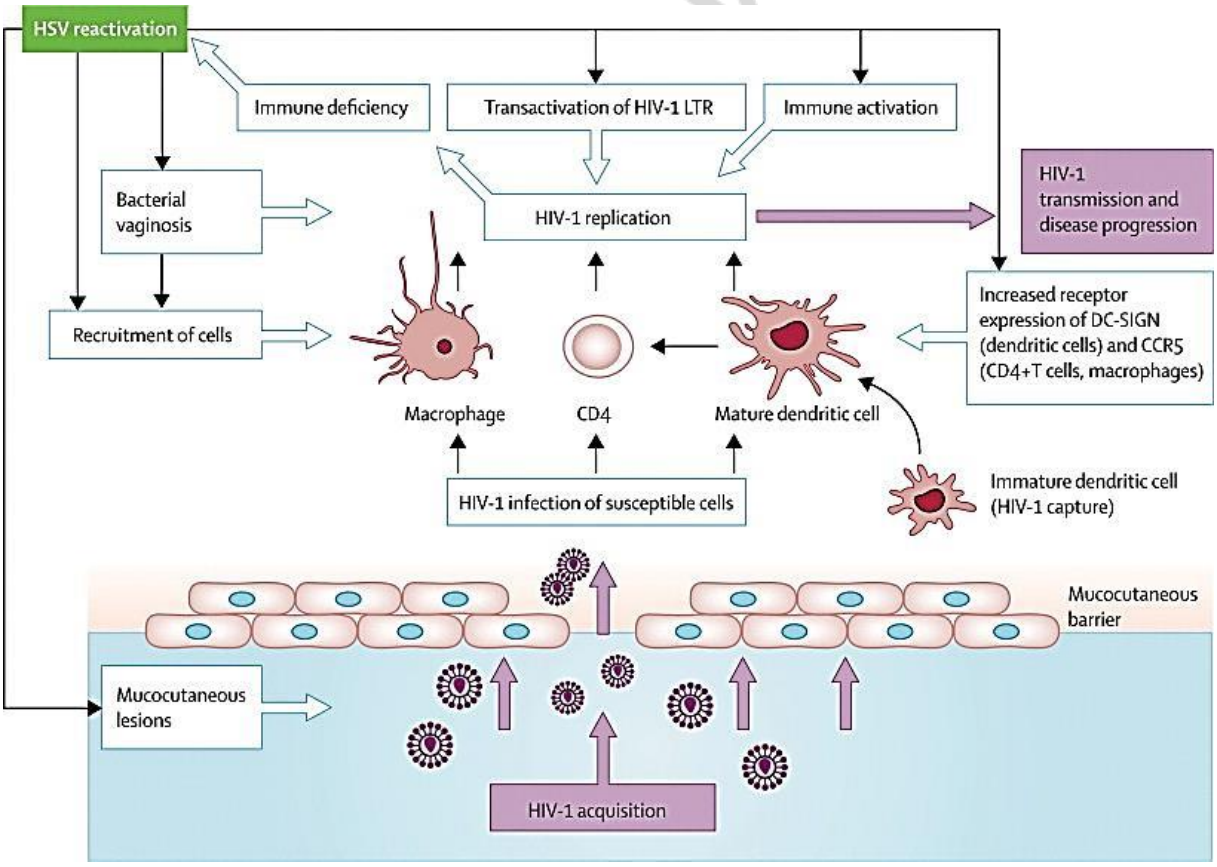

Figure 8. Immune response to acute herpes infection

Sources of infection — persons with recurrent infection (Fig. 9).

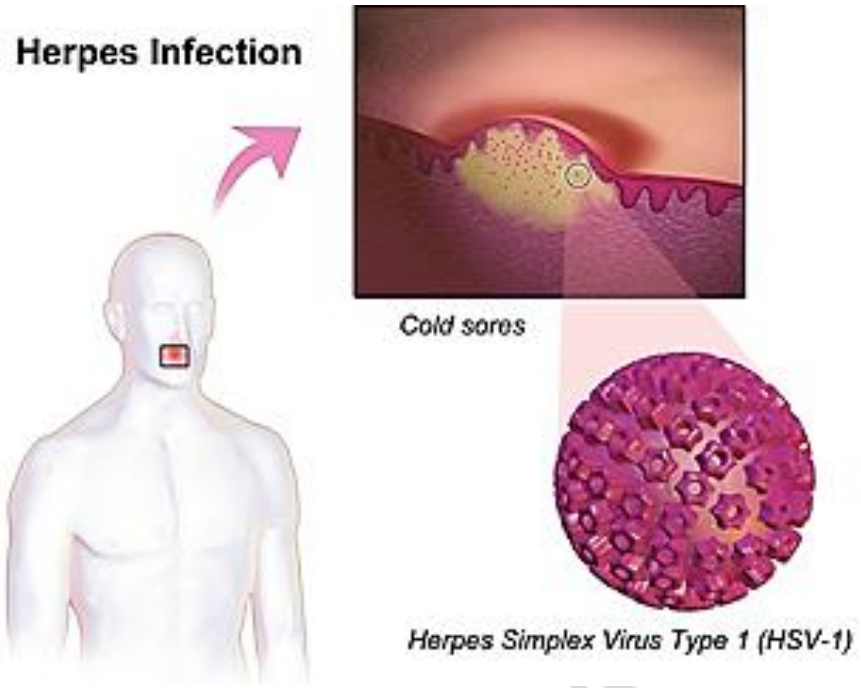

Figure 9. Herpes infection

Viral transmission routes are:

– contact (eating from the same utensils, sharing lip balm, kissing, etc.);
– droplets;
– sexual contacts;
– haemotransfusion;
– transplacental.

Herpes simplex viruses can affect areas of skin exposed to contact with an infected person (although shaking hands with an infected person does not transmit this disease). An example of this is herpetic whitlow, which is a herpes infection on the fingers (Fig. 10). This was a common affliction of dental surgeons prior to the routine use of gloves when conducting treatment on patients.

Viruses may also be transmitted vertically during childbirth. However, the risk of infection transmission is minimal if the mother has no symptoms or exposed blisters during delivery. The risk is considerable when the mother is infected with the virus for the first time during late pregnancy.

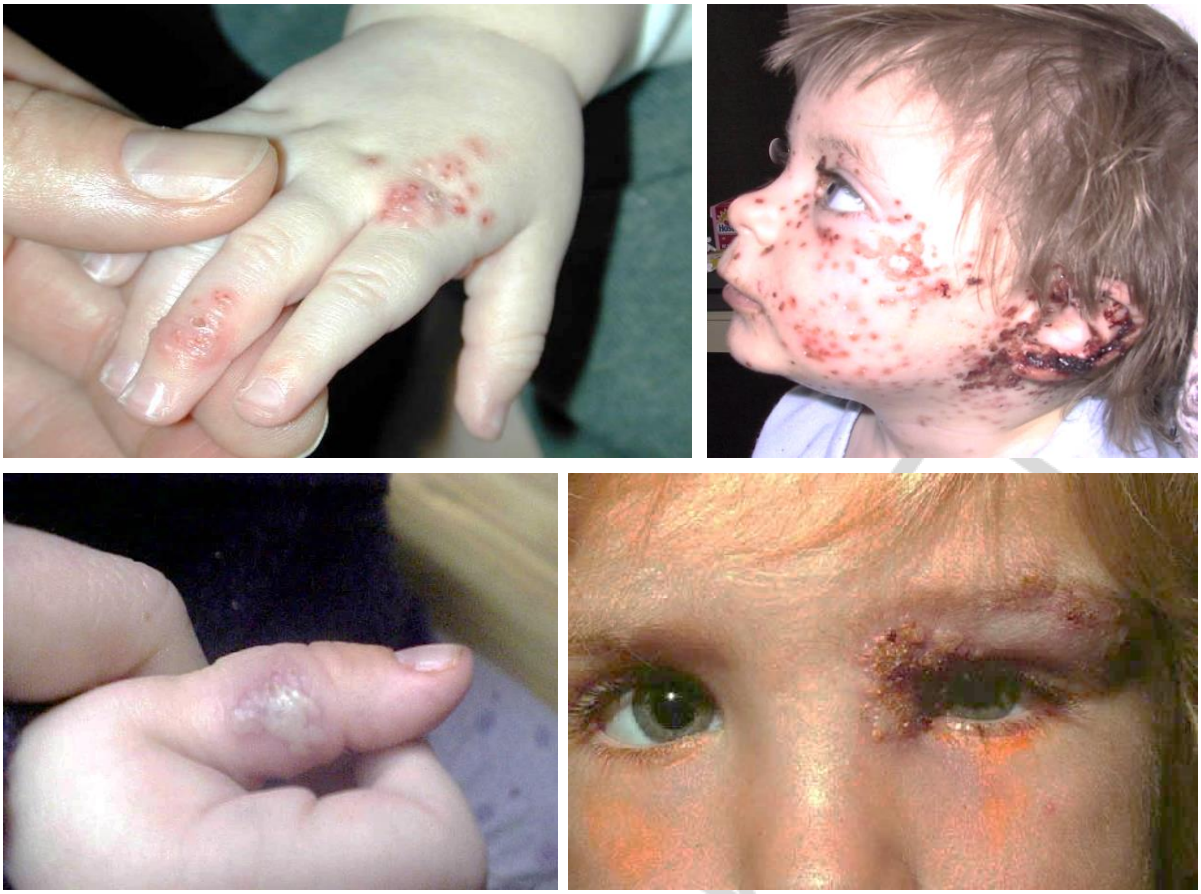

Figure 10. Saliva transmission of herpes virus to fingers, eyes, and ears

FEATURES OF HERPESVIRUS INFECTION IN CHILDREN

– The primary infection is most frequently seen in children between 6 months and 3 years of age, although older age groups can be affected.

– A degree of immunity is transferred to the newborn from circulating maternal antibodies so an infection in the first 6 months of life is rare.

– The majority of childhood infections are subclinical.

HSV infection causes several distinct medical disorders. Common infection of the skin or mucosa may affect the face and mouth (orofacial herpes), genitalia (genital herpes), or hands (herpetic whitlow). More serious disorders occur when the virus infects and damages the eye (herpes keratitis), or invades the central nervous system, damaging the brain (herpes encephalitis). People with immature or suppressed immune systems, such as newborns, transplant recipients, or people with AIDS, are prone to severe complications from HSV infections. HSV infection has also been associated with cognitive deficits of bipolar disorder, and Alzheimer's disease, although this is often dependent on the genetics of the infected person.

EVOLUTION OF THE SKIN AND MUCOUS LESION ELEMENTS

The evolution of lesion elements in herpetic stomatitis on the skin and oral mucosa is different (Fig. 11, 12).

Figure 11. Evolution of the skin lesion elements

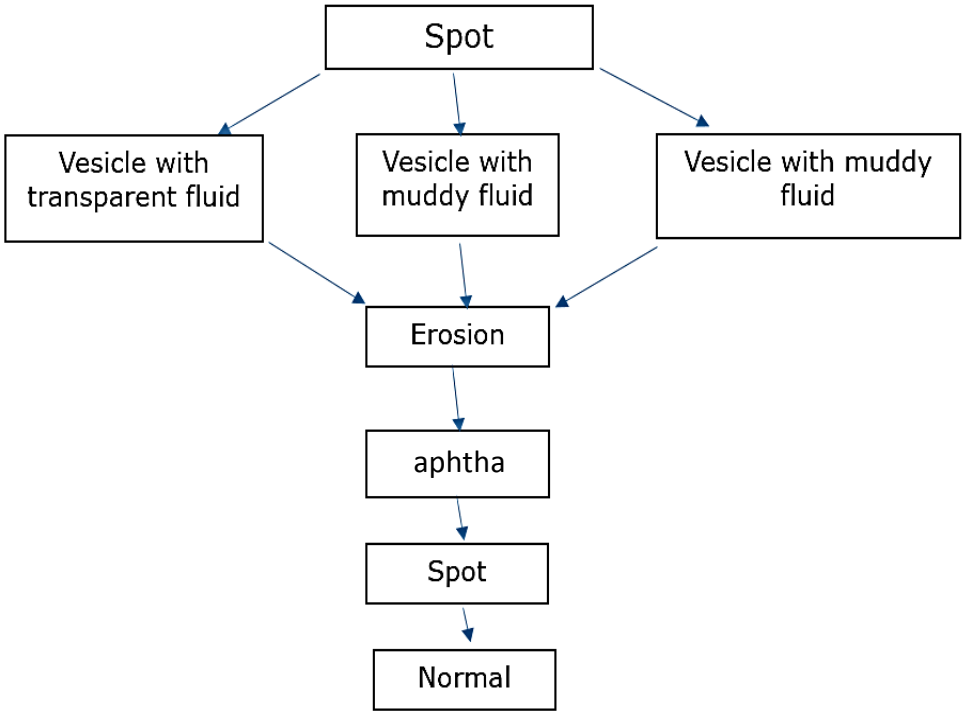

Figure 12. Evolution of the mucous lesion elements

ACUTE HERPETIC STOMATITIS

Clinically, the viral infection begins with prodromal signs or symptoms, such as mild itching that progresses rapidly to blisters or vesicles. After these burst, erosions and ulcers appear and, finally, scabs appear at the end of the herpes cycle. During the first days, pain can be sufficient to cause a lack of appetite, provoking intense systemic involvement. The virus is disseminated when children are irritated by the itching and scratch, bursting the blisters and spreading the virus, very often into the eyes and nose, and other parts of the body.

STAGES OF THE HERPESVIRUS INFECTION

1. **Incubation period** (2–17 days) — virus penetration into the host; primary virus replication in cells around the zone of penetration; cells damage, primary virusaemia.

2. **Febrile stage** — virus spreading inside the host, secondary virusaemia, passing of the virus according to the tissue tropism.

3. **Ulcerative stage** — virus replication in sensitive cells; their damage; immune reaction.

4. **Period of extinction** — mobilizing of all immune factors.

5. **Period of clinical recovery** — virus persistence, latent virus.

Severity of the acute herpetic stomatitis

– mild;
– moderate-severe;
– severe.

MILD FORM OF ACUTE HERPETIC STOMATITIS

– common status is satisfactory;
– temperature is 37–37.5 °C;
– the amount of the oral lesions is 3–5 (Fig. 13);
– oral lesions appear once;
– gingivitis in front teeth;
– submandibular lymphadenitis;
– the length of the period is 1–2 days;
– the protective mechanisms of saliva are expressed.

It is necessary to note such an important symptom of acute herpetic stomatitis as submandibular lymphadenitis: it is diagnosed in ulcerative stage, lasts throughout the disease and persists for 7–10 days after the complete epithelization of the lesions.

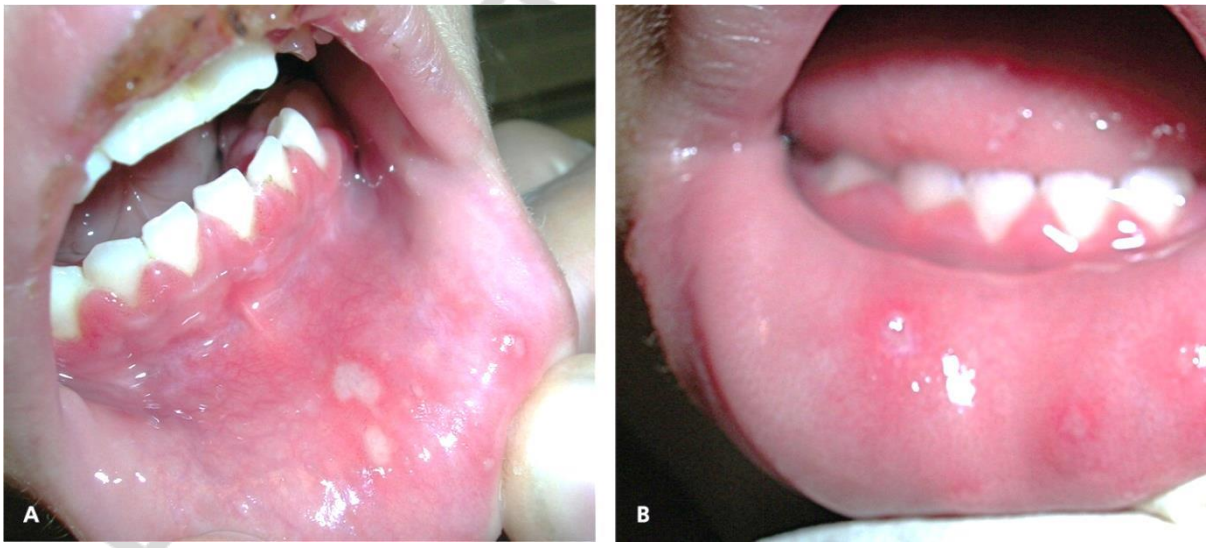

Figure 13. Mild form of the herpetic stomatitis

MODERATE-SEVERE FORM OF ACUTE HERPETIC STOMATITIS

– Headaches, malaise, oral pain, mild dysphagia, and cervical lymphadenopathy are the common symptoms that accompany the fever (38.5–39.0 °C) and precede the onset of a severe, oedematous marginal gingivitis;

– The amount of the oral lesions is 10–25 (Fig. 14, 16);
– Lesions appear several times;
– Submandibular lymphadenitis;
– Gingivitis (Fig. 15);
– Skin lesions (Fig. 14);
– Oedematous marginal gingivitis;
– Decreased immunity;
– Drooling;
– Candida lesions;
– Leucopenia, lymphocytosis;
– GIT disorders;
– The length of the period is 7–10 days.

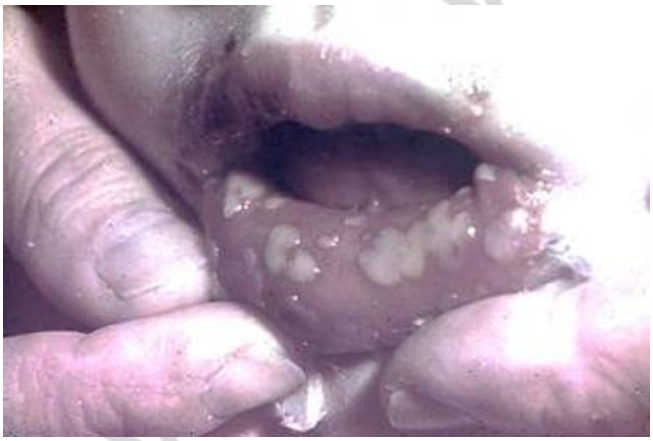

Figure 14. Herpetic lesions of the lips

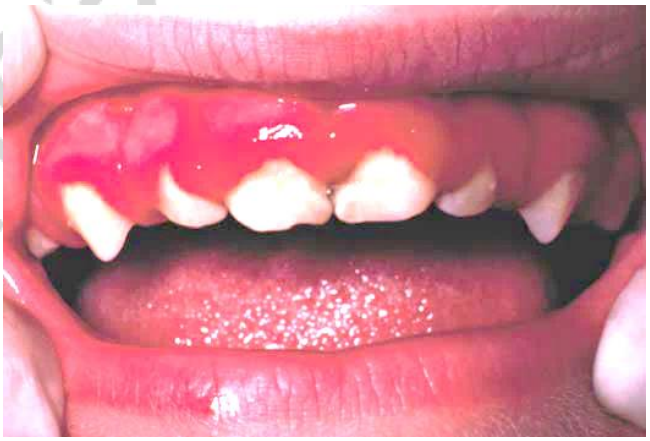

Figure 15. Herpetic gingivitis

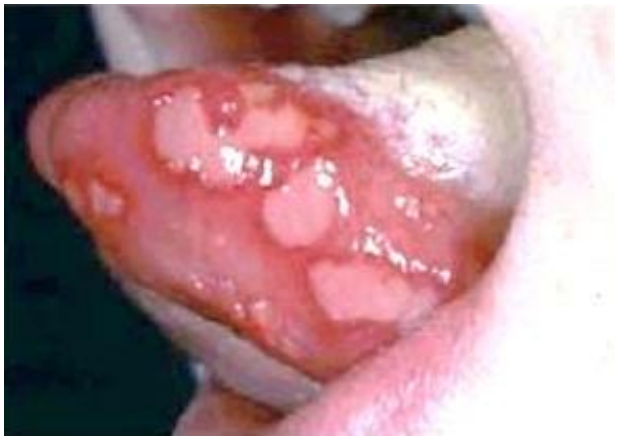

Figure 16. Herpetic lesions of the tongue

SEVERE FORM OF ACUTE HERPETIC STOMATITIS

– temperature is 39–40 °C;

– fluid-filled vesicles (25 and more) appear on the gingiva and other areas such as the tongue, lips, buccal, and palatal mucosa (Fig. 18);

– The vesicles, which have a grey, membranous covering, rupture spontaneously after a few hours to leave extremely painful yellowish ulcers with red, inflamed margins;

– lesions appear several times;

– submandibular lymphadenitis;

– necrotizing ulcerative gingivitis;

– skin lesions (Fig. 17);

– cervical lymphadenopathy;

– leukopenia, lymphocytosis;

– GIT disorders;

– decreasing of all factors of the immunity;

– the length of the period is more than 10 days.

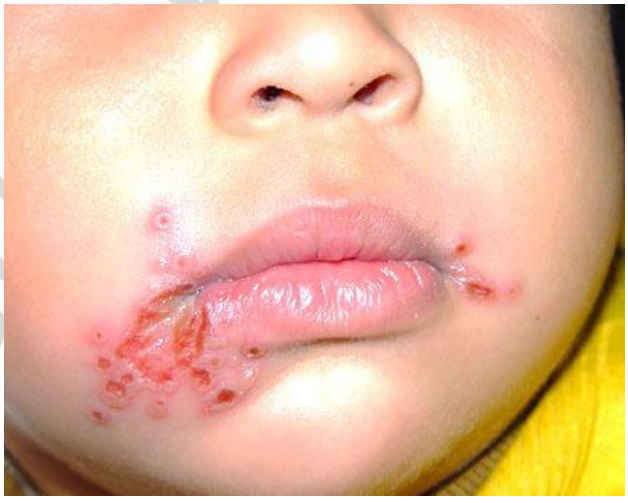

Figure 17. Skin lesions

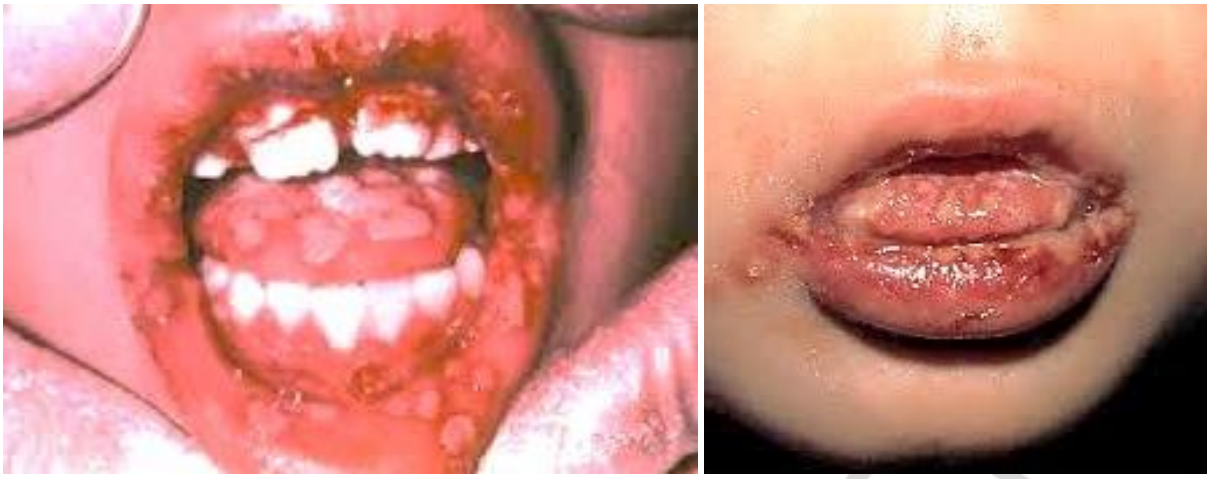

Figure 18. Severe form of the herpetic stomatitis

DIAGNOSTICS OF HERPESVIRUS INFECTION

The clinical features, history, and age group of the affected children are so characteristic that diagnosis is rarely problematic. If in doubt, however, smears from recently ruptured vesicles reveal degenerating epithelial cells with intranuclear inclusions. The virus protein also tends to displace the nuclear chromatin to produce enlarged and irregular nuclei.

Diagnostic methods:

– PCR diagnostics (Polymerase chain reaction);
– Virological investigation;
– Method of gene sensing;
– Immunofluorescence reactions (RIF, IF);
– Enzyme immunoassay (ELISA, TFIIFA or ELISA);
– Radioimmunoassay (RIA);
– Immunoblotting (IB).

For successful laboratory rapid diagnosis of herpetic infection, a simultaneous study of the material taken from several lesions in different ways should be planned.

! For the correct diagnose the doctor should note:

1. The type of the infection: acute or chronic.

2. The form of the disease: mild, moderate-severe or severe.

3. The stage of the disease: incubation, febrile, ulcerative, period of extinction or period of clinical recovery.

For example: Acute herpetic stomatitis, mild form, ulcerative stage.

DIFFERENTIAL DIAGNOSTICS OF HERPESVIRUS INFECTION

Gingivostomatitis symptoms in infants may wrongly be dismissed as teething. Coincidentally, primary tooth eruption begins at about the time that infants are losing maternal antibody protection against the herpes virus. Also, reports on teething difficulties have recorded symptoms which are remarkably consistent with primary oral herpetic infection such as fever, irritability, sleeplessness, and difficulty with eating.

Younger infants with higher residual levels of antibodies would experience milder infections and these would be more likely to go unrecognized or be dismissed as teething difficulty.

Acute herpesvirus gingivostomatitis must also be differentiated from herpangina, another disease that also commonly causes ulcers in the oral cavity in children, but is caused by the Coxsackie A virus rather than the herpes virus.

In herpangina, ulcers are usually isolated to the soft palate and anterior pillar of the mouth. In herpetic gingivostomatitis, lesions can be found in these locations, but they are almost always accompanied by ulcerations on the gums, lips, tongue or buccal mucosa and/or by hyperemia, hypertrophy or hemorrhage of the gums.

MANAGEMENT OF ACUTE HERPETIC STOMATITIS

Treatment of herpetic stomatitis, as well as any other infectious disease, should be ethiologic, pathogenic and symptomatic. It is divided into general and local (Table 3). The choice of specific drugs and their dosage is determined by the severity of stomatitis, its stage and the possibility of the infection generalization.

Table 3

Treatment of acute herpetic stomatitis

Type of treatment	Object	Drug
Ethiologic	Virus herpes simplex	Antiviral
Pathogenic	Immunodeficiency	Immunotherapy
	Dehydration	Drinking, hydrotation
	Intoxication	
	Fever	Antipyretic
	Inflammation of oral mucous	Antiphlogistic
Symptomatic	Pain	Diet, analgetics, anaesthetics
	Bleeding	Tanning, haemostatics

Since we are talking about a systemic infection with manifestations in the oral cavity, the recommendations in treatment of herpetic stomatitis are the following:

– Herpetic gingivostomatitis does not respond well to active treatment. Bed rest and a soft diet are recommended during the febrile stage and the child should be kept well hydrated.

– Pyrexia is reduced with a paracetamol suspension and secondary infection of ulcers may be prevented with chlorhexidine.

– A mouthrinse (0.2 %, two to three times a day) may be used in older children who are able to expectorate, but in younger children (under 6 years of age) a chlorhexidine spray can be used (twice daily) or the solution can be applied with a sponge swab.

– Gels for anaesthesia (with lidocaine hydrochloride).

– In severe cases of herpes simplex, systemic acyclovir can be prescribed in the form of suspension (200 mg) for internal use, five times daily for 5 days (Fig. 19).

– In children under 2 years the dose is halved.

– Acyclovir is active against the herpesvirus but is unable to eradicate it completely. The drug is most effective when given at the onset of the infection.

– Period of extinction — ointment and solutions for epithelization (gel Solcoseryl, Caratolyn, Vinilyn, oil vit. A, vit. E.

Children over 2 years old — the full dose:

– Acyclovir 400 mg twice a day;

– Famciclovir 250 mg once a day;

– Valacyclovir 500 mg once a day.

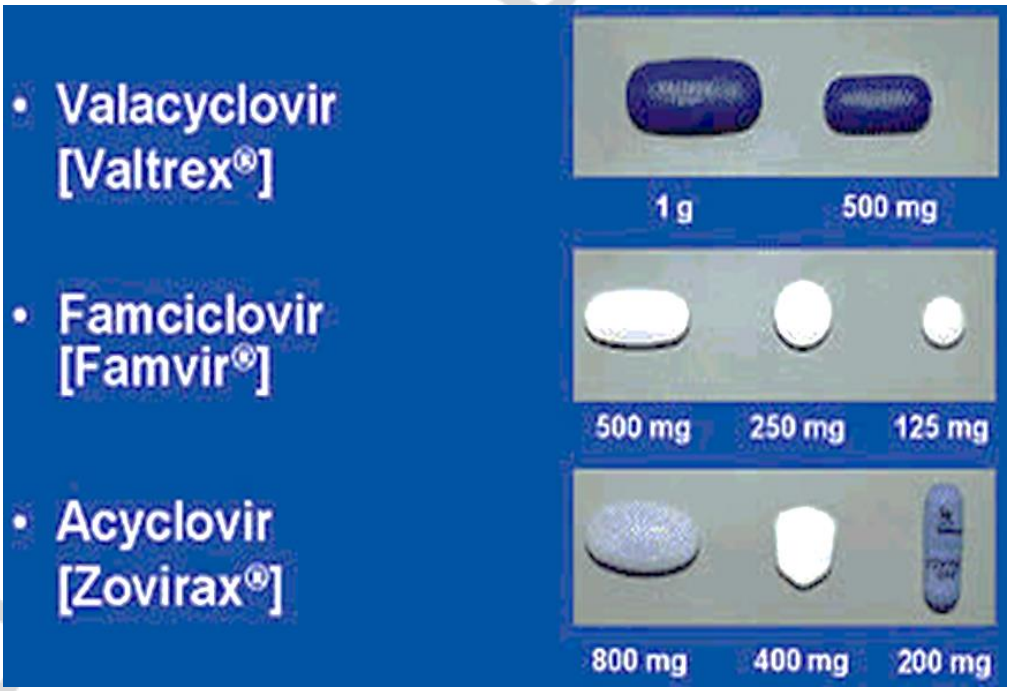

Figure 19. Examples of antiviral drugs

❗ ***No method eradicates herpes virus from the body, but antiviral medications can reduce the frequency, duration, and severity of outbreaks.***

❗ ***Antibiotics cannot affect viruses so it is useless to administer them to the patient.***

As herpetic lesions are very painful, children do not want to eat or drink and the greatest fear of medical professionals is dehydration. Parents must concentrate on making sure that the child drinks plenty of liquids. If any signs or symptoms of dehydration occur such as unusual sleepiness, lethargy or persistent fever, contact the pediatrician. Instruct your child not to put his or her hands in mouth or rub eyes when sores are present to keep the virus from spreading to other body parts. Wash your child's hands with soap and hot water often.

Clean your child's mouth by gently brushing his or her teeth. Don't give the child salty, acidic or spicy food as these will just aggravate the pain. Make sure that your child also gets enough rest and plenty of sleep.

Several studies have demonstrated good results from the use of low-level laser therapy (LLLT), primarily due to acceleration of the healing process and pain relief, which make it a promising resource in treatment of this pathology.

Laser light is a form of radiation that does not ionize, it is highly concentrated and, in contact with different types of tissue and depending on the type of laser, results in photochemical, photoelectric, photothermal, or photomechanical effects. Laser radiation is not invasive, it is well tolerated by tissues, does not have mutagenic effects, and can be used repeatedly without risks.

Low-level laser therapy has many physiological effects, such as anti-inflammatory and analgesic properties and stimulation of wound healing. Low-level lasers or healing lasers, use an energy level below 500 W and do not cause temperature elevation in the target tissue. These lasers also produce a photobiological or photochemical effect on the target tissue.

CHRONIC RECURRENT HERPETIC STOMATITIS

After the primary infection the herpesvirus remains dormant in epithelial cells and in the nerve ganglia of the host. Reactivation of the latent virus or reinfection in subjects with acquired immunity occurs in adults.

Recurrent, like acute infection, can be either unapparent or clinical. The latency mechanisms and virus reactivation are not well studied. There are two groups of factors that determine the development of recurrent infections: an increase in HSV activity and a decrease in the effectiveness of immunity (Fig. 20).

⇧ Of virus activity:
– Acute infection;
– Inflammation;
– Changing of neuron condition (trauma, local anaesthesia, nevritis);
– Chemical activators (nicotine)

⇩ Of immunity:
– Herpesvirus infection
– Hormonal changes
– Stress
– Malnutrition (proteins, vitamins, microelements)
– Immune pathology
– Insolation

Figure 20. Evaluations factors of chronic recurrent herpesvirus

FORMS OF CHRONIC RECURRENT HERPETIC STOMATITIS

Recurrent disease presents as an attenuated intraoral form of the primary infection or as herpes labialis, i.e. the common "cold sore" on the mucocutaneous border of the lips.

!*70 % of children have chronic recurrent herpesvirus stomatitis after acute herpesvirus stomatitis.*

Recurrent HSV infection generally appears as single vesicles or a small cluster of vesicles that rupture quickly, forming ulcers. The ulcers usually occur on the keratinized epithelium of the hard palate, gingiva and skin (Fig. 21).

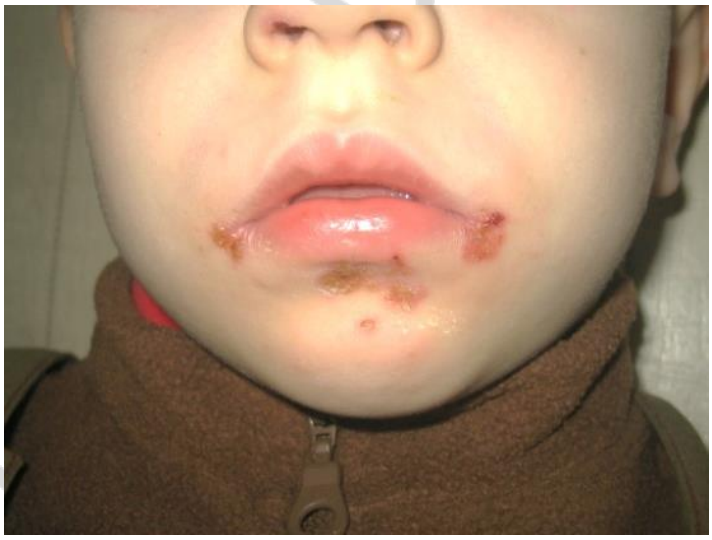

Figure 21. Patient with chronic recurrent herpesvirus

Forms of the chronic recurrent herpesvirus:
– Mild form — 1–2 times in 3 years;
– Moderate-severe form — 1–2 times a year;
– Severe form — 4 and more times a year or permanent form.

TREATMENT OF CHRONIC RECURRENT HERPETIC STOMATITIS

Treatment of chronic recurrent herpesvirus should be ethiologic, pathogenic and symptomatic. It is divided into general and local.

During ***the recurrent period***, treatment can be started already in the prodromal period in the family: when signs of infection appear (burning, itching, edema in the place of the future element of the lesion), local, and better systemic use of antiviral drugs should be started, the so-called "strategy for episodic treatment of chronic recurrent herpesvirus".

On exacerbation of the recurrent chronic herpesvirus, the treatment tactics are the same as those described above for acute herpetic stomatitis, corresponding to mild, moderate-severe or severe degrees of the severity.

The peculiarities of antiviral treatment of chronic recurrent herpesvirus stomatitis in immunosuppression are:

– the appointment of acyclovir systemic treatment (often in high doses) in order to prevent the generalization of herpes infection;

– readiness to replace acyclovir with Cidofovir or even Foscarnet in cases (5–10 %) of HSV resistance to acyclovir and its analogs.

In ***the interrecurrent period***, general treatment is aimed at ensuring control of the activity of persistent herpes infection; therefore, children with chronic recurrent herpesvirus infection are dispensary registered with a dentist.

The main attention in a ***mild form*** of the herpesvirus infection is to eliminate factors which can provoke the recurrence of the disease:

– identification and elimination of oral cavity infection (sanitation of the oral cavity);

– identification and elimination of bad habits that traumatize oral mucosa and the vermilion border (parents' motivation);

– the use of photo protective lipsticks in spring and summer.

In case of a ***moderate-severe form*** of chronic recurrent herpesvirus stomatitis the elimination of local provoking factors is combined with general effects on the child's immunity:

– family motivation to harden the child;

– carrying out immunostimulation in the spring-autumn period (in agreement with the pediatrician).

In a ***severe form***, immunocorrection is added to the measures carried out for mild and moderate-severe forms, which must be carried out with the participation of an immunologist.

An alternative method of treatment for severe and permanent forms of chronic recurrent herpesvirus stomatitis is, the so-called "strategy of preventive anti herpetic treatment" (syn. suppressive therapy), which requires the constant daily intake of acyclovir 200 mg — 2 to 5 times a day (or valacyclovir 200 mg 1–2 times a day) for 1–3 years. This strategy cannot prevent the onset of relapse,

but provides an ability to stop the reproduction of an activated virus at the beginning. The method is highly effective (it prevents about 80 % of clinical relapses), but it is psychologically and economically difficult.

During predicted risk periods of an increase of chronic herpesvirus stomatitis recurrence the method of prophylactic antiherpetic treatment can be used as a compromise: during hypothermia and stress, during immunosuppressive therapy, etc.

RECURRENT HERPES SIMPLEX LABIALIS

Recurrent herpes simplex labialis, also known as oral herpes, is an infection of the mouth area caused by the herpes simplex virus (Fig. 22). It's a common and contagious infection that spreads easily. According to the American Sexual Health Association, over half of adults in the United States carry this virus.

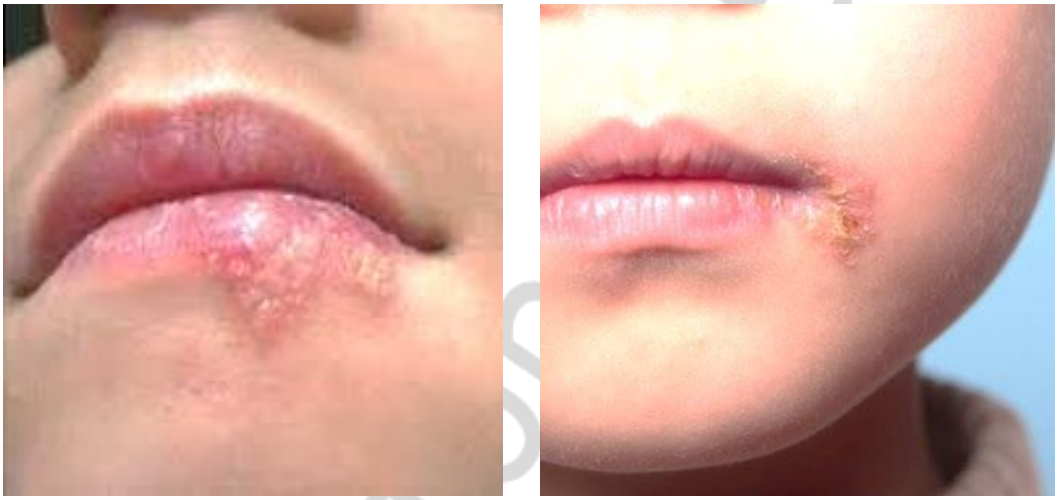

Figure 22. Patient with recurrent herpes simplex labialis

The infection causes blisters and sores on the lips, mouth, tongue or gums. After an initial infection, the virus stays dormant inside the nerve cells of the face. Later on in life, the virus can reactivate and result in more sores. The herpes is recurrent. These are commonly known as cold sores or fever blisters.

Recurrent herpes simplex labialis is usually not serious, but relapses are common. Many people choose to treat the recurrent episodes with over-the-counter creams. The symptoms will usually go away without treatment in a few weeks. A doctor may prescribe medications if relapses occur often.

Herpes simplex labialis is the result of a virus called herpes simplex virus type 1 (HSV-1). The initial infection usually occurs before age 20. It typically affects the lips and areas around the mouth.

Sources of the virus: close personal contact with someone who has the virus; from touching objects where the virus may be present (these include towels, utensils, razors for shaving, and other shared items).

After the first infection, the virus lays dormant inside the nerve cells of the face for the rest of a person's life. This means that symptoms aren't always present. However, certain events can make the virus reawaken and lead to a recurrent herpes infection. Events that trigger a recurrent infection of oral herpes might include:

– fever;
– menstruation;
– a high-stress event;
– fatigue;
– hormonal changes;
– upper respiratory infection;
– extreme temperature;
– a weakened immune system;
– recent dental work or surgery.

Treatment of the recurrent herpes simplex labialis should be ethiologic, pathogenic and symptomatic. It is divided into general and local. For the local treatment antiherpetic lipsticks, creams and gels can be used (Fig. 23, 24).

Figure 23. Antiherpetic lipstick

Figure 24. Antiherpetic cream and gel

Recognizing the Signs of Recurrent Herpes Simplex Labialis

The primary infection may not cause symptoms at all. If it does, blisters may appear near or on the mouth within one to three weeks after your first contact with the virus. The blisters might last up to three weeks. In general, a recurrent episode is milder than the initial infection.

Symptoms of a recurrent episode may include:

– blisters or sores on the mouth, lips, tongue, nose, or gums;

– burning pain around the blisters;

– tingling or itching near the lips;

– outbreaks of several small blisters that grow together and may be red and inflamed.

Tingling on or near the lips is usually a warning sign that the cold sores of recurrent oral herpes are about to appear in one or two days.

PREVENTION OF HERPESVIRUS INFECTION

About 90 % of the population carries HSV. There's little the parents can do to prevent their child from picking up the virus sometime during childhood. The child should avoid all close contact with people who have cold sores. So if the caregiver gets a cold sore, he can't kiss the child until the sore is gone. The child should also avoid other children with herpetic stomatitis.

If the child has herpetic stomatitis, parents should avoid spreading the virus to other children:

– Make the child wash her/his hands often.

– Keep toys clean and don't share them with other children.

– Don't allow children to share dishes, cups, or eating utensils.

– Don't let the child kiss other children.

HSV-1 is most contagious during an outbreak of symptomatic oral herpes, but can also be transmitted when no symptoms are felt or visible. People with active symptoms of oral herpes should avoid oral contact with others and sharing objects that have contact with saliva.

Dentists are responsible for the proper evaluation and risk assessment of their patients as well as infection control in the health care setting. Thus, recognition of viral infections, knowledge of standard precautions, and implementation of prevention strategies are important components of the infection control process.

CONTENT

Учебное издание

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Романова Ольга Сергеевна

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