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IMPROVING THE EFFECTIVENESS OF TREATMENT OF HERPETIC STOMATITIS IN PATIENTS OF DIFFERENT AGE GROUPS

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✓ ***Resume***

Here presented historic data on herpetic infection, characterized different representatives of herpetic virus. Pathogenesis of this virus is described, classification of it is given and phases of virus development in the human body, ways of decease transmission. Illustrated prevalence of herpetic stomatitis in different age groups. Presented clinic classification of herpetic stomatitis, clinics of rapidly relaps-ing herpetic stomatitis depending on level of decease. Given differential diagnostics of oral herpetic infection in comparison with a range of similar deceases. Described basic treatment principles of herpetic stomatitis, offered a scheme of the most rationaltreatment of above decease with a use of media for local and overall therapy of her- petic stomatitis. Recommendations are given for prophylactic actions, which deter theoccurrence and relapse of this decease.

One of the urgent problems of modern clinical medicine is viral diseases. A special place among them is occupied by herpes simplex (PG).

The term "herpes" (from "herpo"to crawl) has been known in medicine for almost 25 centuries. Febrile herpes ("cold") it was described by the Roman physician Herodotus in 100 BC. Mention of it is found in the treatises of Hippocrates, Avicenna. In 1912, the German scientist W.Gruter isolated the herpes simplex virus (HSV). It caused herpetic keratitis in rabbits when the contents of the patient's vesicle were applied to the scarified cornea. In 1919, another German scientist A. Lowenstein published the results of similar experiments [1, 2, 3].

Keywords: HSV, herpetic stomatitis.

ПОВЫШЕНИЕ ЭФФЕКТИВНОСТИ ЛЕЧЕНИЯ ГЕРПЕТИЧЕСКОГО СТОМАТИТА У БОЛЬНЫХ РАЗНЫХ ВОЗРАСТНЫХ ГРУППАХ

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✓ ***Резюме***

В статье приставлены исторические данные о герпетической инфекции, охарактеризованы различные представители герпетического вируса. Описан патогенез этого вируса, дана его классификация и фазы развития вируса в организме человека, пути передачи заболевания. Проиллюстрирована распространенность герпетического стоматита в разных возрастных группах. Представлена клиническая классификация герпетического стоматита, клиники быстро рецидивирующего герпетического стоматита в зависимости от степени заболевания. Дана дифференциальная диагностика герпетической инфекции полости рта в сравнении с рядом аналогичных заболеваний. Описаны основные принципы лечения герпетического стоматита, предложена схема наиболее рационального лечения вышеуказанного заболевания с использованием сред для местной и общей терапии герпетического стоматита. Даны рекомендации по профилактическим мероприятиям, которые предотвращают возникновение и рецидив этого заболевания.

Ключевые слова: ВПГ, герпетический стоматит.

ТУРЛИ ЁШДАГИ БЕМОРЛАРДА ГЕРПЕТИК СТОМАТИТНИ ДАВОЛАШ САМАРАДОРЛИГИНИ ОШИРИШ

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✓ ***Резюме***

Илмий мақолада герпетик инфекция ҳақида тарихий маълумотлар тақдим этилди, герпетик вирус турли вакиллари характерланади. Ушбу вируснинг патогенези тасвирланган, унинг классификацияси берилган ва инсон организмида вирус ривожланиш босқичлари, децаза юқиш йўллари. Турли ёш гуруҳларида герпетик стоматитнинг тарқалиши тасвирланган. Герпетик стоматитнинг клиник таснифи, decease даражасига қараб тез қайталанадиган герпетик стоматит клиникалари тақдим этилди. Шунга ўхшаш десеасес бир қатор билан солиштирганда оғзаки герпетик инфекция берилган дифференциал диагностика. Герпетик стоматитнинг асосий даволаш тамойиллари тасвирланган, юқорида кўрсатилган десеазни маҳаллий ва умумий даволаш учун оммавий ахборот воситаларидан фойдаланган ҳолда энг оқилона даволаш схемасини таклиф қилди. Профилактик ҳаракатлар учун тавсиялар берилади, бу эса бу десеазнинг пайдо бўлиши ва қайталанишини олдини олади. Замонавий клиник тиббиётнинг долзарб муаммоларидан бири вирусли касалликлардир. Улар орасида herpes simplex (ПГ) алоҳида ўрин тутади.

Калит сўзлар: ҲСВ, герпетик стоматит.

Relevance

Viral diseases are one of the urgent problems of modern clinical medicine. A special place among them is occupied by herpes simplex (PG).The term "herpes" (from "herpo" - to crawl) has been known in medicine for almost 25 centuries. Febrile herpes ("cold") it was described by the Roman physician Herodotus in 100 BC. Mentions of him are found in the treatises of Hippocrates, Avicenna. In 1912, the German scientist V. Gruter isolated the herpes simplex virus (HSV). This caused herpetic keratitis in rabbits when the contents of the patient's vesicle were applied to the scarified cornea. In 1919, another German scientist A. Levenstein published the results of similar experiments [1, 2, 3].

Epidemiology

In recent years, worldwide, there has been a trend in the spread of HSV. Primary infection, as a rule, is asymptomatic, and 20-30% of herpes patients develop a relapse of the disease during the first 2-3 years, so it is not possible to establish the true number of cases. The increase in morbidity is largely associated with the spread of asymptomatic and undiagnosed forms of the disease, but this objective process, unfortunately, is not accompanied by radical changes in the attitude of both doctors and the population to this disease [4].

According to WHO, the disease caused by HSV, as the cause of death, ranks second (15.8%) after influenza (35.8%) [1,2].

According to modern estimates, the infection rate of the HSV population is very high and even approaching 100% [1,2,6]. Epidemiological studies conducted over the past 12 years have shown that by the age of 15, about 83% of people are infected with HSV, and at the age of 30 years and older more than 90% of the population [1,4,5].

The prevalence of herpetic infection is ensured by a large number of virus carriers: about 20 million people in the world are asymptomatic carriers of the virus, half of them are infected with the HSV-2 virus. A persistent trend towards an increase in the number of infected has been revealed: the number of carriers of HSV-I and HSV-2 alone has increased by 30% over the past 10 years. HSV-1 is transmitted by the orolabial route and is detected in 70-100 million people in the USA alone [3].

At the initial stages of the study of HSV, there was an opinion about the electoral defeat of HSV-1 exclusively in the facial area, and HSV-2 in the genital area. Modern data refute this point of view. The role of HSV-1 in the development of genital herpes worldwide is increasing, reaching, according to some estimates, 50%. At the same time, it was noted that the herpes virus associated with HSV-1 recurs

much less frequently, and more than 95% of its exacerbations are associated with HSV-2 infection. Cases of labial herpes caused by HSV-2 have become more frequent [2,6]. The wide prevalence of HSV is caused by a number of adverse factors, including environmental (environmental pollution), occupational hazards, bad habits (smoking, alcohol consumption), alimentary (poor nutrition, vitamin deficiency) [7].

Herpes affects the skin, central nervous system, mucous membranes, and sometimes internal organs. Over the past few years, a significant increase in herpetic infection has been observed in dermatovenerology, neurology, obstetrics and gynecology, ophthalmology, otorhinolaryngology, as well as in dentistry [1,5].

Acute herpetic stomatitis (OGS) occupies one of the leading places in pediatric infectious pathology, primarily because this stomatitis accounts for more than 80% of all diseases of the oral mucosa in children [8].

Children of different age groups suffer from OGS, however, most often at the age of 6 months to 3 years, which is explained by the disappearance of antibodies received from the mother interplacentally, and the peculiarities of the structure of the mucous membrane of the oral cavity (SOPR) at this age. Currently, it has been noted that newborn children also suffer from OGS (from 2 to 43 days after birth), following ante- and postnatal infection, while intrauterine infection was noted in 1/3 of cases [9].

Infection prevention is practically not carried out, since too many people are involved in social activity and most of them are lifelong virus carriers. Thus, infection and morbidity are constantly growing, outstripping the natural growth of the planet. Compared with the 80s, by the end of the millennium, the number of registered herpes patients increased in the USA by 13-40%, in European countries - by 7-16%, the highest percentage is observed in Africa and is 30-40% [4].

Etiology and pathogenesis

The causative agent of HSV is a DNA-containing virus, once in the body, HSV persists throughout life, periodically causing relapses of the disease, which, as with primary infection, occur with different degrees of severity and location. Primary herpes in 80% of patients is asymptomatic. Usually, a few days after infection, specific antibodies appear in the blood serum. More than 85% of 3-year-old children have virus neutralizing antibodies. In addition to antibodies, cellular immunity plays an important role as protective factors against reinfection, which suppresses the reproduction of the virus - the production of interferon the cage itself. A significant role in protecting the body is played by viruria – the removal of the virus from the body in various ways (with urine, sweat, sputum) [1, 3,9,10,11,12 13].

HSV is capable of affecting the epithelium, passing into a latent form and reactivating after a while, leading to relapses.

HSV with a size of 120-150 nm consists of a nucleotide located in the center, a capsid surrounding the core of the virion, and an outer shell in which these structures are enclosed [14, 15].

Currently , 8 types of pathogenic human herpesviruses are known

(human herpes virus- HHV 1-8). Depending on the nature

of virus reproduction, the structure of the genome, the type of cells in which the process takes place, as well as immunological and other features, herpesviruses are divided into 3 sub-families - alpha, beta and gamma.

Alpha-herpesviruses include HSV-HSV-1, HSV-2 and Varicella zoster virus – VZV (HHV-3) with rapid replication and cytopathogenetic effect on infected cell cultures. Their reproduction takes place in various types of cells, viruses persist mainly in the ganglia in a latent state [17].

Beta-herpesviruses include cytomegalovirus-CMV(HVV-5), HHV-6, HVV-7. They are species-specific, affect various types of cells, increase them to huge sizes (cytomegaly) and can cause an immunosuppressive state [18]

The Epstein–Barr virus (HHV–4) and (HHV-8) belong to the gamma virus subfamily. These viruses are characterized by tropicity to lymphoid cells (T- and B – lymphocytes), where they persist for a long time and after transformation can cause lymphomas and sarcomas [19].

A more detailed classification is presented in Table No. 1.

Table 1
Classification of human herpes virus

Human herpesviruses	Designations	The main diseases associated with this type of herpesviruses
Herpes simplex virus type 1	HSV-1	Labial herpes. Herpes of the skin and
HSV-1	HSV-2	mucous membranes. Ophthalmogerpes. Genital herpes. Herpetic encephalitis.
Herpes simplex virus type II HSV-2	VVZ	Genital herpes. Neonatal herpes
Varicella	(VOG)	Chickenpox. Herpes zoster.
Epstein-Barr virus (EBV-HHV-	VEB	Infectious mononucleosis.
4)	CMV	kitt's lymphoma.
Herpes virus type VI and	VHF-6 VHF-7	Congenital lesions of the central nervous system. Retinopathy. Pneumonitis. Hepatitis.

HSV penetrates into the body through damaged skin and mucous membranes and goes through 4 phases of development.

In the 1st phase, the virus is introduced into the epithelial cells, where its multiplication occurs. Cells die, lymphocytes and macrophages migrate to the focus of inflammation, inflammatory mediators are released, capillaries and connective tissue are damaged.

In phase 2, the virus penetrates into the nerve endings and ganglia. Virus replication occurs in the nuclei of neurons. HSV has a cytopathic effect: it destroys the cells in which it multiplies [1,15].

In the 3rd phase (2-4 weeks after infection), with a normal immune response, the resolution of the primary disease occurs, the elimination of HSV from tissues and organs. However, in the paravertebral ganglia, the pathogen persists throughout a person's life.

In the 4th phase, HSV reproduction is reactivated, it moves along nerve fibers to the epithelial cells of the SOPR (gate of infection) with the recurrence of a specific infectious and inflammatory process and possible dissemination of infection [20].

Herpetic infection is reactivated under the influence of external and internal factors that reduce the protective functions of the body (hypothermia, overheating, acute respiratory disease, hormonal changes) [1].

In some patients, in the absence of clinical manifestations of herpes, there is an active release of HSV with biological secretions – asymptomatic herpes, which plays an important role in the infection of contact persons.

Thus, HSV has the properties of both an acute infection and a pathogen with a chronic persistent course [3].

Several ways of infection with herpes are known:

- airborne;
- contact (direct or indirect contact);
- transplacental;
- transfusion [1].

Classification of herpetic stomatitis.

Herpes simplex manifests itself in two forms:

1) acute herpetic stomatitis or acute aphthous stomatitis

2) chronic recurrent herpes or chronic recurrent herpetic stomatitis [3, 11].

By severity:

1) easy;

2) medium-heavy;

3) heavy [3].

Classification by WHO: K12. Stomatitis and related lesions

HSV penetrates into the body through damaged skin and mucous membranes and goes through 4 phases of development.

In the 1st phase, the virus is introduced into the epithelial cells, where its multiplication occurs. Cells die, lymphocytes and macrophages migrate to the focus of inflammation, inflammatory mediators are released, capillaries and connective tissue are damaged.

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Thus, HSV has the properties of both an acute infection and a pathogen with a chronic persistent course. [3].

Several ways of infection with herpes are known:

- airborne;
- contact (direct or indirect contact);
- transplacental;
- transfusion [21].

Treatment

Treatment of patients with herpetic stomatitis presents certain difficulties and depends on the mechanism of infection, the form and severity of the infectious process, as well as the localization of the lesion [24].

Treatment of herpes simplex is complex (general and local).

The general treatment boils down to the following:

1. a high-calorie diet is prescribed, copious drinking.

2. antiviral drugs –bonafton 0.1 g3 times a day for 5-10 days; alpizarin 0.1 g 4 times a day; chelipin 0.1g 4 times a day for 5-10 days; acyclovir adults 200 mg 5 times a day [2,3,9,12,25].

3. desensitizing therapy - diphenhydramine, suprastin, pipolfen, diprazine, diazoline, fencorol, tavigil.

4. restorative therapy -vitamin C up to 2.0 g per day, multivitamins.

5. analgesic, antipyretic, anti-inflammatory drugs.

6. immunocoregulatory therapy (roncoletin) in combination with antiviral agents [26].

Local treatment includes the following actions:

1. anesthesia of the oral mucosa - 2% oil solution of anesthetic, 1% pyromycoin ointment, 5% lidocaine gel, 1% trimecaine solution, solcoseryl adhesive paste [9, 27].

2. antiseptic treatment of the oral mucosa -1.5% solution of hydrogen peroxide, 00.6% chlorhexidine, potassium permanganate 1:5000, etc ;

3. proteolytic enzymes are used to reject necrotic films - trypsin, chymotrypsin, chymopsin, lysozyme [3,9].

4. application of antiviral ointments 3-4 times a day 3% acyclovir, 3% zovirax, 3% herpevir.

5. epithelizing therapy - application of keratoplastic preparations, 2-3 times a day: vitamin A in oil, vitamin E in oil, carotoline, rosehip oil, sea buckthorn oil, solcoseryl adhesive paste [3,9].

Prevention of viral diseases.

1. isolation of a patient with a viral disease from the collective, even with mild severity;

2. elimination of chronic foci of infection;

3. herpetic vaccination [3].

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