Herpes simplex viruses infection

Herpes simplex viruses (HSV-1, HSV-2; Herpesvirus hominis) produce a variety of infections involving mucocutaneous surfaces, the central nervous system, and on occasion – visceral organs.

Etiology

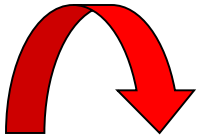
- Herpes simplex virus - ***DNA***
- ***Alphaherpesviridae*** family
- Is very sensitive to ultraviolet light and various disinfectants.
- HSV forms intranuclear inclusions in infected cells
- There are two types of HSV:

✓ ***Herpes simplex virus 1*** (HVS-1) affects facial skin, mucous membranes of the oral cavity and eyes.

✓ ***Herpes simplex virus 2*** (HVS-2) - also affects mucous membranes and skin of genital organs.

Herpes simplex viruses

- Herpes simplex virus type 1 (HSV-1) is usually associated with infections of the lips, mouth and face.
- It is the most common herpes simplex virus and many people develop it in childhood.



- HSV-1 often causes sores (lesions) inside the mouth, such as cold sores (fever blisters), or infection of the eye (especially the conjunctiva and cornea). It can also lead to infection of the lining of the brain (meningoencephalitis).
- It is transmitted by contact with infected saliva.
- By adulthood, 30 - 90% of people will have antibodies to HSV-1.

- **Herpes simplex virus 2 (HSV-2)** is usually, but not always, sexually transmitted. Symptoms include genital ulcers or sores.
- However, some people with HSV-2 have no symptoms. Up to 30% of adults have antibodies against HSV-2.
- Cross-infection of type 1 and 2 viruses may occur from oral-genital contact. That is, you can get genital herpes on your mouth, and oral herpes on your genital area.

Epidemiology

- The source of infection is a **person with various clinical forms**.
- Infection is transmitted be infected by their mothers with passing through delivery passages.
- Transplacental transmission of infection may occur.
- Susceptibility to the infection is high.
- Presence of antiviral antibodies does not prevent relapses. Frequency of relapses depends on the condition of specific cell immunity.

Pathogenesis

- Portal of entry is skin and mucous membranes. Primary infection occurs in the first 3-5 years of age.

- After primary infection HSV penetrates in the sensorium ganglia of posterior nerve roots of the spinal cord and it nerves, and persists there for life in forms which are inaccessible to the influence of the immune system.

- When immunity decreases the infection passes over to the productive form and HSV spreads along the nerves into skin and mucous membranes - typical clinical manifestations .

Pathohistological lesions

- Balon dystrophy with cell death and accumulation of serous exudate in epidermis

- Foci of coagulation necrosis with poor cell infiltration in brain and other organs in infection generalized forms

- Petechiae, perivascular infiltrates, large cells with intranuclear inclusions are typical.

Clinical manifestations

In primary infection an incubation period is from **2 to 14 days** (an average 4-5 days).

There are some clinical forms of herpesviral infection depending on the process localization.

Oral – Facial Infections: gingivostomatitis and faringitis are most common of first – episode HSV-1 infection, recurrent herpes labialis is the most common clinical manifestation of reactivation HSV-1 infection.

Herpes labialis (cold sore)

- Is characterized by appearance of small vesicles on the mucous membranes of nose and lips.
- The vesicles appear in groups and are surrounded by an area of hyperemia.
- Subjective sensations are itch, pain and burning pain. They contain clear fluid, which gradually then becomes turbid.
- The vesicles dry up and crusts.
- Crusts fall off in 5-7 days and the affected skin is again covered with epithelium.

Acute herpetic gingivostomatitis

- Usually infects children from **6 months to 3 years old.**
- The disease frequently occurs in primary infection.
- Acute onset with severe toxemic fever (up to 40-41°C). Child stops eating, complains of sore mouth, severe hypersalivation. At the same time hyperemia and edema of mucous membranes of cheeks, gums, tongue, and lips appear.
- Soon after typical herpetic eruptions occur in their place; they burst rapidly, resulting in aphthae.
- Regional lymph nodes are enlarged and painful.
- Duration of the disease is from **7 to 10 days.**

Herpetic eye infections

- appear in the form of **conjunctivitis** and **keratoconjunctivitis.** Typical eruptions occur on the conjunctiva, cornea, sclera. Marginal ulcer of the cornea, parenchymatous keratitis, cataract, choreoretinitis may result too. If the disease has a relapsing course, blindness may occur.
- HSV infection of the corneal epithelium produces pain, tearing, photophobia, and corneal ulcers that often have a branching pattern.

Eczema herpeticum

- Occurs usually in babies which suffer eczema.
- The onset with a fever up to 40°C and higher.
- Severe toxemia.
- Vesicular rash appears on eczematous skin. The vesicles fuse, burst, dry up rapidly and result in crust. Recurrent waves of eruptions occur frequently.
- The disease has a severe course and lethal outcome may result.

CNS HSV infections

- Appear in the form of **serous meningitis** and **meningoencephalitis.**
- Clinical peculiarities of the disease are: severe spastic syndrome (with involvement of temporal and parietal brain lobes), and necrotic changes in CNS.
- The disease has a very severe course, **morbidity goes up to 50-80%.**

Generalized herpesviral infections

- In neonates and children with severe immunodeficiency severe generalized forms of the disease with lethal outcome may occur.
- Severe signs of toxemia, lesion of brain, liver, kidneys, lungs, adrenal glands.
- If specific therapy is absent, outcome is usually unfavorable.

Diagnosis

- Antigens of HSV may be discovered using the immunofluorescence assay of skin and mucous lesions, blood cells, cerebrospinal and amniotic fluid, pathoanatomical material
- Viral culture of the lesion (vesicles, blood, CSF, tissues)
- Blood test for antibodies of HSV (serology)
- PCR (DNA-HSV-1, DNA-HSV-2)
- ELISA (antibodies cl.IgM and IgG)

Treatment

- **Acyclovir** for 7-10 days; **Famciclovir** , **Valacyclovir** .
- Antiviral and antiseptics may be used locally on affected areas of skin and mucous membranes.
- Symptomatic and pathogenetic therapy.
- Treatment of herpetic encephalitis should be conducted according to the general principles.
- In relapses of herpetic infection the recurrent courses of vaccination using herpetic vaccine, or prolonged (up to 3-5 years) anti-relapsing therapy by Acyclovir .