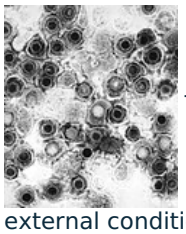
Herpesviruses

It is a broad group of DNA viruses that cause predominantly latent infections in animals and humans. The most important for a person are:

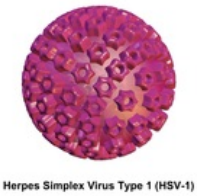
- **herpes simplex virus types 1 and 2** (HSV 1, 2)
- **varicella zoster virus** (VZV)
- **cytomegalovirus** (CMV)
- **Epstein-Barr virus** (EBV, HHV 4)
- **human herpes viruses** (HHV 6, 7, 8)

Virion



The capsid is coated with a lipid membrane. The virus is about 100-180 nm in size and is highly sensitive to external conditions (mainly fatty solvents and oxidizing agents). Between the shell and the capsid is a **protein layer**. The packaging predetermines some properties - viruses are sensitive to acidic pH and drying, as well as to non-polar solvents and detergents. Thus, the infection is transmitted only by **direct contact** with an infected person.

Herpes simplex virus type 1 (HSV 1)



Disease

- clinical manifestation:
  - gingivostomatitis with fever, local lymphadenopathy - in early childhood
  - tonsillitis
  - keratoconjunctivitis
  - meningoencephalitis - can manifest itself throughout life, has a difficult course, high lethality and leaves severe consequences after healing.
  - **herpes labialis** - manifestation in adults
- complications of primary infection - Kaposi's variceliform eruption (Eczema herpeticatum; vesiculo-pustular)

🔎 *For more information see Herpes simplex virus.*

Herpes simplex virus type 2 (HSV 2)

Disease

- most often affects the mucous membranes of the **genital tract**(glans penis, cervix, vagina), especially in puberty. Fever and blisters are characteristic.
- the virus may be excreted by cervical secretion.
- in neonatal infections, the development of severe generalized disease

Pathogenesis

- Gate of entry: the mucous membranes most often of the oral cavity
- Multiplication of the virus
- **Spread across neurons** to the sensory part of the trigeminal ganglion (Ggl. Gasseri), where it persists.
- immunosuppression activates endogenous infection and herpes (herpes)
- the gateway to HSV 2 is the mucosa of the genital tract and the site of persistence = nerve ganglia in the small pelvis

Diagnostics

- virus isolation from saliva, blister content, cerebrospinal fluid
- PCR
- antibody serological test - ELISA

Epidemiology

The source of the virus is a person who excretes it **saliva, cervical secretion** (symptoms of the disease may not be present).

🔎 *For more information see Herpes simplex virus.*

Varicella zoster virus (VZV)

Disease

Clinically, the virus manifests as (chickenpox). The course is usually light with a slightly elevated temperature. Complications can occur if primary infection occurs in adulthood. Reactivation of the endogenous virus manifests itself clinically as shingles.

Pathogenesis

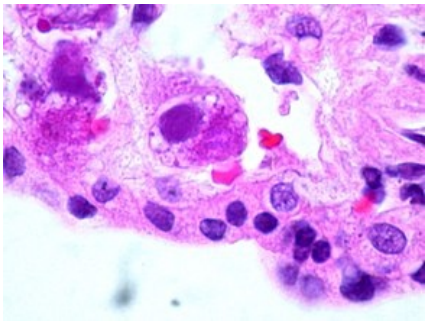
- entrance gate - respiratory tract
- multiplication
- hematogenous spread to the skin and mucous membranes, where it causes blistering
- persistence in the spinal ganglia of the intercostal nerves - when the immunity is weakened, it spreads anterogradely into the sensitive area of the given nerve and shingles is formed.

Diagnostics

- PCR
- ELISA

Cytomegalovirus

It infects 90% of the adult population.

Cytomegalovirus	
Herpesviridae	
	
placental inflammation caused by CMV	
Type NK	dsDNA
Source	human
Transmission	vertically (transplacentally, childbirth, breast milk), body fluids
Occurrence	cosmopolitan
Infection	mononucleosis syndrome, hepatitis, retinitis, encephalopathy
Diagnostics	serology , virus isolation, Ag detection, PCR
Therapy	symptomatic, antivirals, hyperimmune Ig
Vaccination	in development
MeSH ID	D003587
Medscape	215702

Primary infection

Primary infection is usually mild or asymptomatic. It manifests itself as:

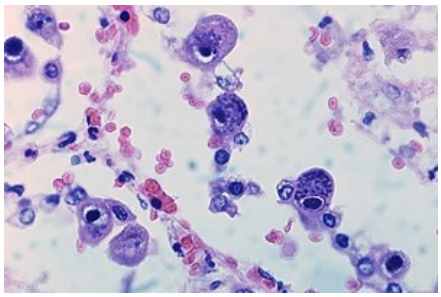
- infectious mononucleosis syndrome,
- febrile illness with lymphadenitis,
- hepatitis (especially in infants),
- **severe congenital infections** (microcephaly, blindness, hepatomegaly, purpura),
- congenital or postnatal infection - usually asymptomatic,
- rarely pneumonia, colitis, meningitis.

Reactivation in **immunosuppressed** manifests as:

- febrile illness with lymphadenitis,

- pneumonia,
- septic disease,
- colitis, oesophagitis,
- retinitis (especially in HIV positive),
- encephalitis.

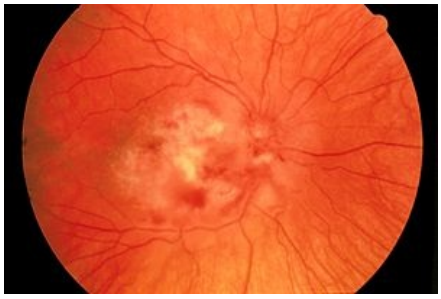
Following a kidney transplant, a virus infection can cause transplant rejection.


Cytomegalovirus infection

Pathogenesis

The gateway is the respiratory tract or upper gastrointestinal tract. Then there is multiplication and subsequent hematogenous spread. It is associated with atherosclerosis and restenosis after heart surgery. The virus persists in salivary gland cells, renal tubules and leukocytes. The infected individual occasionally **excretes viruses through saliva, urine, cervical secretion, and breast milk.**

Diagnostics


CMV Retinitis

Cultivation

Cultivation is easy. CMV grows with a typical **cytopathic effect** in 5-25 days on human diploid cells. Identification can be accelerated by evidence of **nuclear inclusions** with a mononuclear antibody (24-48 hours). We can use urine, saliva, vaginal secretion, breast milk (difficult), leukocytes (difficult) for cultivation.

Quick diagnostics

Antigen detection and microscopy are insensitive. Nucleic acid detection (PCR) is used. It is fast and reliable. Most sample types can be used. It is also possible to perform quantitative determinations. PCR is more sensitive than culture.

Evidence of antibodies

IgG, IgM or IgA are determined. IgG is anamnestic, for the diagnostics of reactivation they have a little importance, it is possible to determine the avidity of IgG. Both IgM and IgA are important in acute infections and reactivations. Non-specific reactions are described. Serology is only an orientation method. Antibodies do not indicate immunity.

Therapy

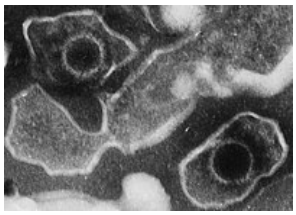
- Hyperimmune gamma globulin
- Antiviral treatment:
  - ganciclovir;
  - foscarnet;
  - special new antivirals (cidofovir).
- Prophylaxis in high-risk patients;
  - aciclovir (not suitable for the treatment of advanced infection).

Prevention

Vaccine attempts have so far failed. There are epidemiological measures in organ recipients (CMV negative recipients should not receive organs from a CMV positive donor) - due to the high population density, this is difficult to follow.

Epstein-Barr virus (EBV)

🔎 *For more information see EBV.*



Widespread virus, from 10 years of age is infected almost. A small proportion is clinically manifest. It has been linked to **Burkitt's lymphoma**children in Africa and **nasopharyngeal carcinoma** in Asia.

Disease

- clinical manifestations as coating angina with lymphadenitis, fever and hepatic impairment.
- relatively long convalescence

Pathogenesis

- entrance gate - respiratory tract
- B - lymphocyte multiplication and persistence
- persistent presence of antibodies and immunity to reinfection.

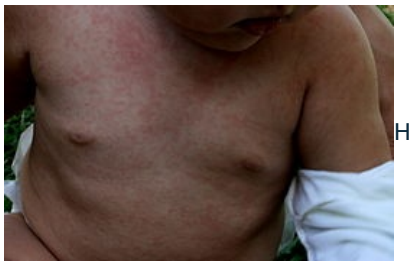
Diagnostics

- insulation is complicated and practically impossible.
- serological diagnostics
  - evidence of non-specific antibodies (Paul-Bunnell reaction, ...)
  - evidence of specific antibodies - indirect immunofluorescence, ELISA

Epidemiology

The virus spreads by droplets and is widespread worldwide.

HHV-6 and HHV-7



HHV-6 and HHV-7 belong to the family Herpesviridae. HHV-6 is divided into two subspecies - HHV-6A and HHV-6B. They are enveloped viruses that contain double-stranded DNA. The size of the virion is 120-150 nm. They replicate in the nucleus, they mature in the cytoplasm. They are lymphotropic viruses similar to CMV, they persist in the body for life.

Diagnostics

- Based on clinical signs;
- serology - IgG and IgM antibodies by immunofluorescence or ELISA methods;
- cultivation - demanding, on special lymphocyte soils;
- PCR - from different tissues, the most sensitive method.

Manifestations of infection

Primary infection usually takes place in childhood, often without symptoms. **HHV-6** causes one of the most well-known manifestations of these viruses - a mild **febrile condition in infants and young children**either with rash - exanthema subitum (sixth childhood disease) or without skin speeches. Severity increases with the incidence of **febrile convulsions**. Similar manifestations are also caused by **HHV-7**. The infection spreads through the air. After an incubation period of 5-12 days, the high temperature **without catarrhal manifestations** lasting about 3 days, which is typical for the disease, begins. When the temperature drops, a small rash is sown, which must be distinguished from toxoallergic rash (after antibiotics, which are often given).

Rare clinical manifestations are **encephalitis, hepatitis**, or **infectious mononucleosis syndrome**. [1]

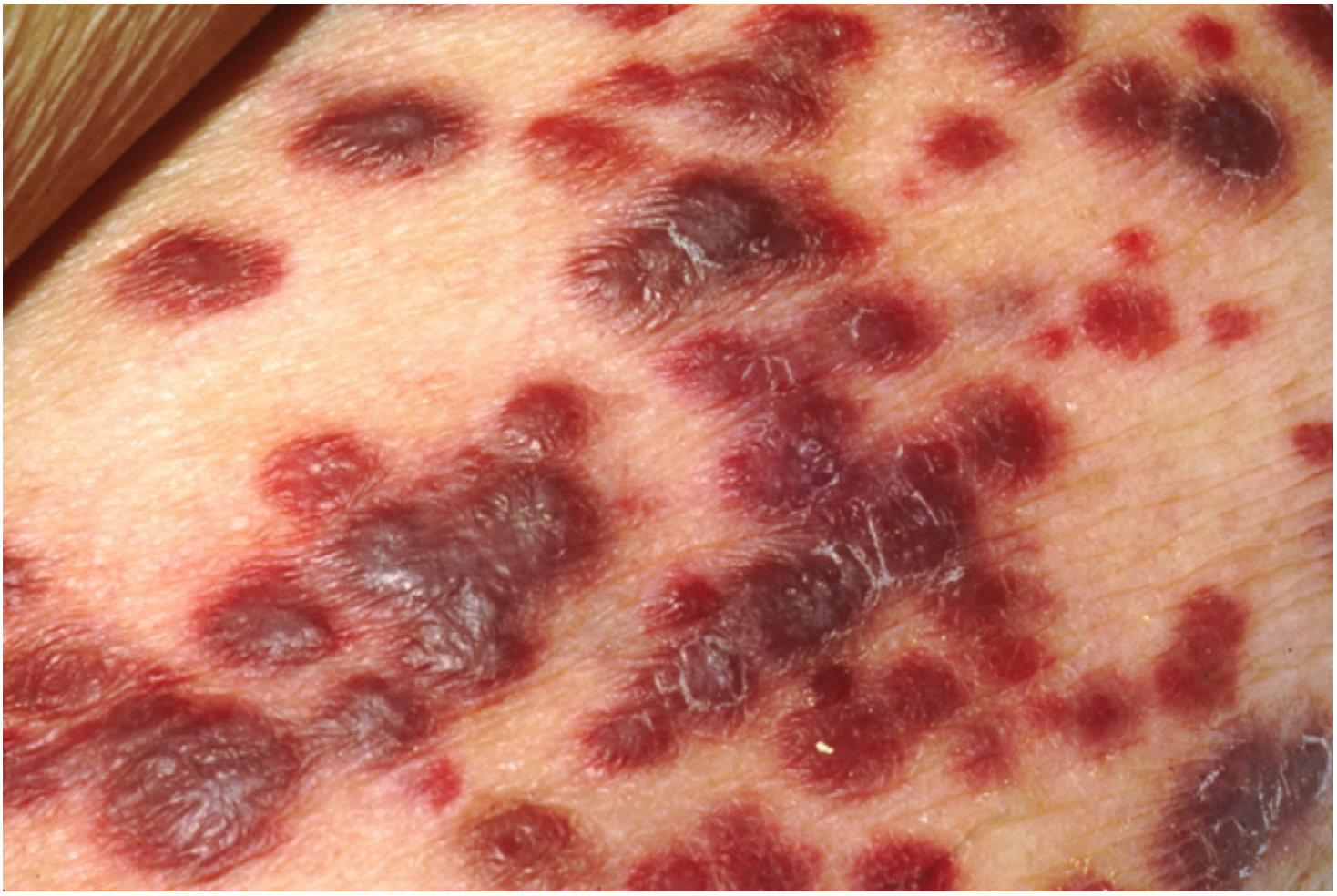
In immunosuppressed (lymphoproliferative diseases, after transplantation ...) there is a possibility of reactivation of the infection.

HHV-6A is more neurotropic, often detectable in patients with inflammatory CNS (multiple sclerosis).

Therapy

Therapy is symptomatic.

HHV-8

Human herpes virus 8	
Herpesviridae	
	
Kaposi's sarcoma	
Type NK	dsDNA
Source	human
Transmission	sexual intercourse, blood
Occurrence	cosmopolitan
Infection	Kaposi's sarcoma (HIV +), other malignancies (angiosarcoma), Castleman's disease
Diagnostics	serology, detection of antibodies against nuclear antigen, HHV-8 DNA
Therapy	does not yet know effective causal treatment
MeSH ID	D019288

- **HHV-8** (*Human Herpesvirus 8*), also referred to as **KSHV** (*Kaposi sarcoma-associated herpesvirus*), is a dsDNA virus from family *Herpesviridae*.
- Described in patients with immune defect (HIV).
- In immunodeficiency causes Kaposi's sarcoma (mesenchymal malignant vascular tumor).
- Other diseases caused by the virus include *primary exudative lymphoma* (when malignant B-lymphoma develops) or Castleman's disease.
- It also occurs in immunocompetent persons, in whom it causes only latent infection.
- Current status diagnostics using PCR only.
- Serological diagnostics: IgG antibodies - anamnestic [2].

Links

Related Articles

- Herpetic encephalitis
- HHV-6 and HHV-7 infections

Used literature

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