**LESSON 16.  
Microbiology diagnosis of infections, caused by *Herpesviridae, Picornaviridae* families and *Rubivirus* genus**

**LESSON PLAN:**

• Herpesviridae family, general properties (classification, morphology, cultivation).

• Morpho-biological characteristics, epidemiology, diseases, pathogenesis, microbiological diagnosis, treatment and prevention of simple herpesviruses (SHV-I and SHV-II simple herpes viruses).

• Chicken pox - shingles virus (Varicella-zoster virus), morpho-biological characteristics, epidemiology, diseases caused by it, pathogenesis, microbiological diagnosis, treatment and specific prevention.

• Epstein-Barr virus, morpho-biological characteristics, epidemiology, pathogenesis and microbiological diagnosis of infectious mononucleosis

• Cytomegalovirus, morpho-biological characteristics, epidemiology, pathogenesis, microbiological diagnosis and treatment of diseases caused by it

• Other human herpesviruses (HIV-6, IHV-7, IHV-8)

• Picornaviridae family, general properties (classification, morphology, cultivation, antigens, persistence).

• Morpho-biological characteristics of poliomyelitis viruses, epidemiology, pathogenesis of poliomyelitis, microbiological diagnosis. Specific prevention and treatment.

• Morpho-biological features of Coxsackie and ECHO viruses, epidemiology, diseases caused by them, pathogenesis, microbiological diagnosis.

• Rubivirus genus - Rubella virus, general, cultural properties, pathogenicity, epidemiology and pathogenesis of rubella, teratogenic effects of rubella virus. Microbiological diagnosis of measles. Specific prevention.

**HERPESVIRUSES**

**Trigger Words**

ᑏ HSV-1 and HSV-2: neurotropic, Cowdry type A inclusion bodies, syncytia, vesicle, Tzanck smear

ᑏᑏVZV: neurotropic, (V) all stages of lesions, (Z) lesions along single dermatome

ᑏᑏ EBV: lymphotropic: B cell, heterophile-positive mononucleosis, Burkitt lymphoma

ᑏᑏCMV: large cell and owl’s eye inclusion body, opportunistic, mononucleosis, congenital disease

ᑏᑏ and HHV7: lymphotropic, roseola

ᑏᑏHHV-8: Kaposi sarcoma, AIDS-related disease

ᑏᑏ B virus: monkey, fatal encephalopathy

**Biology, Virulence, and Disease**

ᑏᑏ Large, enveloped, icosadeltahedral capsid, DNA genome

ᑏᑏ Encodes polymerase and other proteins (HSV and VZV: thymidine kinase)

ᑏᑏ Cell-mediated immune response essential for control

ᑏᑏ Lytic, latent, recurrent infections; EBV and HHV-8 also associated with cancers

ᑏᑏ HSV: oral/genital, encephalitis, keratoconjunctivitis, neonatal HSV; recurs from neurons

ᑏᑏVZV: pneumonia in adults, varicella, zoster; recurs from neurons

ᑏᑏ EBV: heterophile-positive mononucleosis, B-cell lymphomas; recurs from memory B cell

ᑏᑏ CMV: opportunistic disease, congenital CMV, retinitis; recurs from monocyte and stem cell

ᑏᑏ HHV-6: roseola

ᑏᑏ HHV-8: Kaposi sarcoma

**Epidemiology**

ᑏᑏ Ubiquitous viruses

ᑏᑏ Transmitted by direct contact, bodily fluids

ᑏᑏVZV transmitted by aerosol and direct contact

**Diagnosis**

ᑏᑏCulture, immunologic tests (EBV serology), PCR and genome

analysis

**Treatment, Prevention, and Control**

ᑏᑏVaccines for varicella and zoster

ᑏᑏAntiviral drugs for HSV, VZV, and CMV *CMV,* Cytomegalovirus; *EBV,* Epstein-Barr virus; *HHV,* human herpesvirus; *HSV,* herpes simplex virus; *PCR,* polymerase chain reaction; *VZV,* varicella-zoster virus.

**Unique Features of Herpesviruses**

Have large, enveloped, icosadeltahedral capsids containing double-stranded DNA genomes.

Encode many proteins that manipulate the host cell and immune response.

Encode enzymes (DNA polymerase) that promote viral DNA replication and are good targets for antiviral drugs.

DNA replication and capsid assembly occurs in the nucleus.

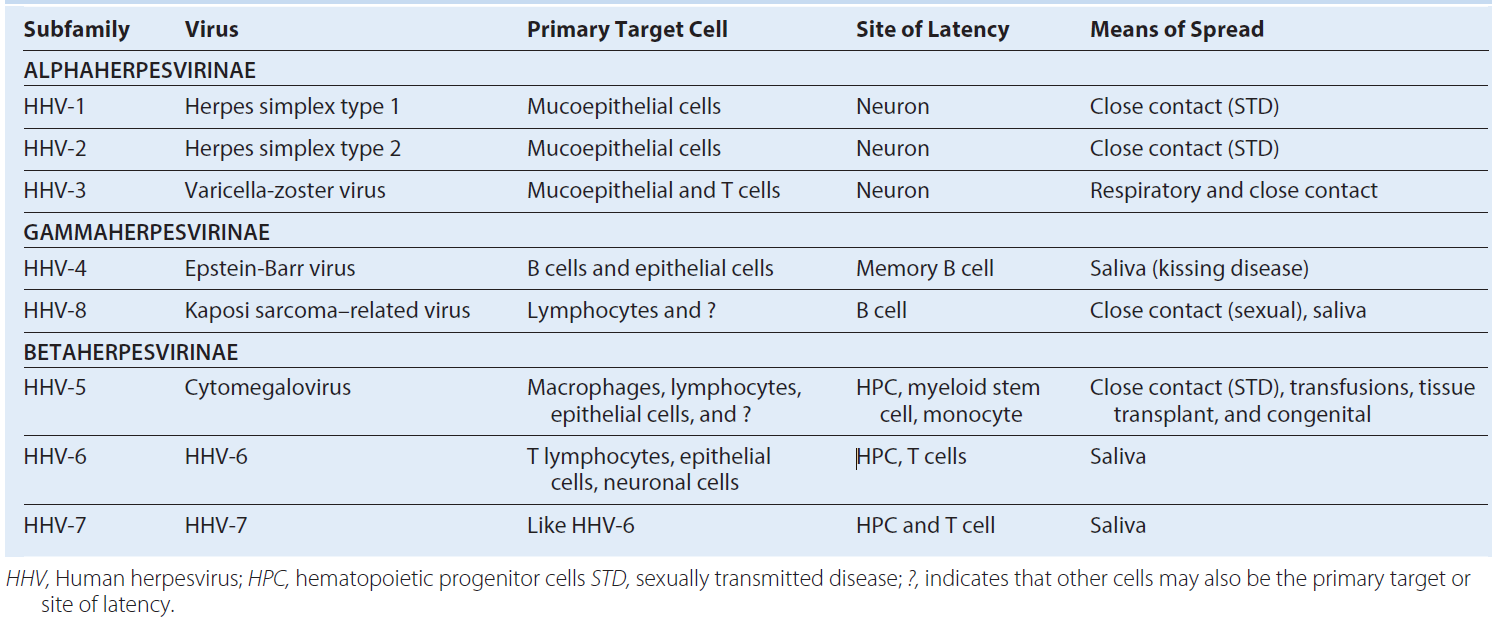
Virus is released by exocytosis, by cell lysis, and through cell-to cell bridges.

Can cause lytic, persistent, latent, and (for Epstein-Barr virus) immortalizing infections.

Ubiquitous.

Cell-mediated immunity is required for control.

**Properties Distinguishing the Herpesviruses**



**Disease Mechanisms for Herpes Simplex Viruses**

Disease is initiated by direct contact and depends on infected tissue (e.g., oral, genital, brain).

Virus causes direct cytopathologic effects.

Virus avoids antibody by cell-to-cell spread and syncytia.

Virus establishes latency in neurons (hides from immune response).

Virus is reactivated from latency by stress, ultraviolet B light, or immune suppression.

Cell-mediated immunity is required for resolution, with a limited role for antibody.

Cell-mediated immunopathologic effects contribute to symptoms.

**Triggers of Herpes Simplex Virus Recurrences**

Ultraviolet B radiation (skiing, tanning)

Fever (hence the name “fever blister”)

Emotional stress (e.g., final examinations, big date)

Physical stress (irritation)

Menstruation

Foods: spicy, acidic, allergies

Immunosuppression:

Transient (stress related)

Chemotherapy, radiotherapy

Human immunodeficiency virus

**Epidemiology of Herpes Simplex Virus**

**Disease/Viral Factors**

Virus causes lifelong infection.

Recurrent disease is a source of contagion.

Virus may cause asymptomatic shedding.

**Transmission**

Virus is transmitted in saliva, in vaginal secretions, and by contact with lesion fluid (mixing and matching of mucous membranes).

Virus is transmitted orally and sexually and by placement into eyes and breaks in skin.

HSV-1 is generally transmitted orally; HSV-2 is generally transmitted sexually, but not exclusively.

**Who Is at Risk?**

Children and sexually active people are at risk for primary disease of HSV-1 and HSV-2, respectively.

Physicians, nurses, dentists, and others in contact with oral and genital secretions are at risk for infections of fingers (herpetic whitlow).

Immunocompromised people and neonates are at risk for disseminated life-threatening disease.

**Geography/Season**

Virus is found worldwide.

There is no seasonal incidence.

**Modes of Control**

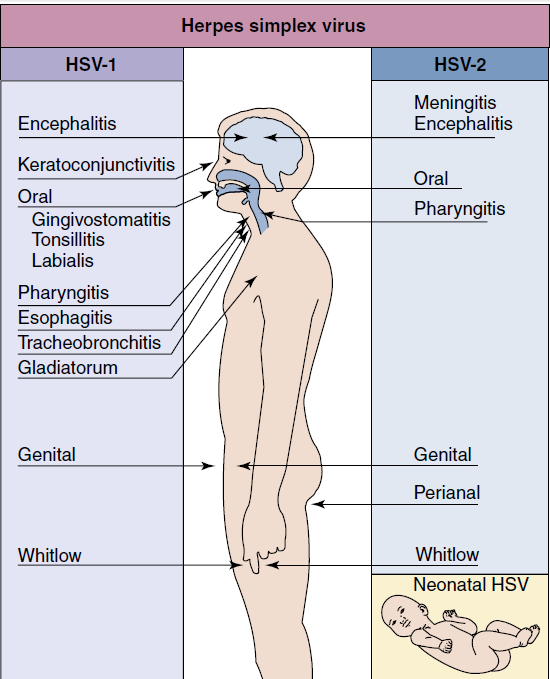
Antiviral drugs are available for treatment and prophylaxis.

No vaccine is available.

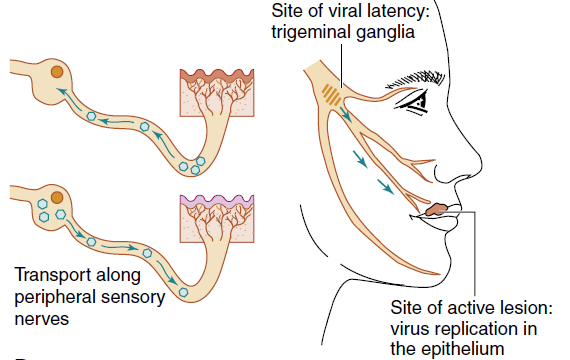
Health care workers should wear gloves to prevent herpetic whitlow.

People with active genital lesions should refrain from intercourse until lesions are completely reepithelialized.

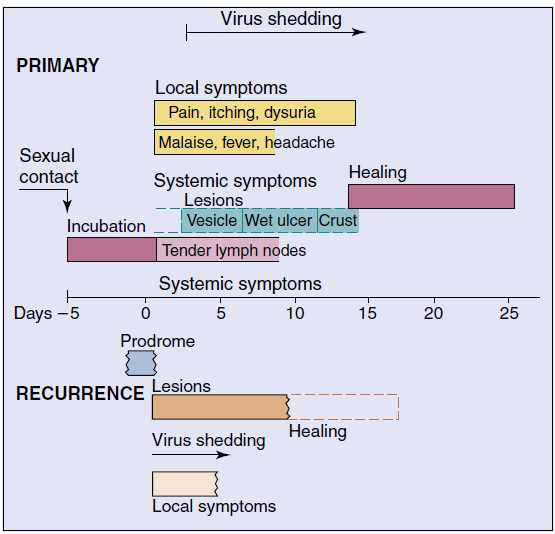
**Disease syndromes of herpes simplex virus *(HSV).* HSV-1 and HSV-2 can infect the same tissues and cause similar diseases but have a predilection for the sites and diseases indicated.**



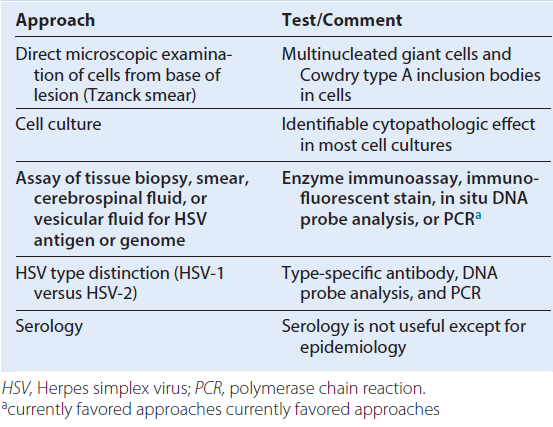
**(A) Primary herpes gingivostomatitis. (B) Herpes simplex virus establishes latent infection and can recur from the trigeminal ganglia.** (A, From Hart, C.A., Broadhead, R.L., 1992. A Color Atlas of Pediatric Infectious Diseases. Wolfe, London, UK. B, Modified from Straus, S.E., 1993. Herpes simplex virus and its relatives. In: Schaechter, M., Eisenstein, B.I., Medoff, G. (Eds.), Mechanisms of Microbial Disease, second ed. Williams & Wilkins, Baltimore, MD.)



**Clinical course of genital herpes infection. The time course and symptoms of primary and recurrent genital infection with herpes simplex virus type 2 are compared. *Top,* Primary infection; *bottom,* recurrent disease.** (Data from Corey, L., Adams, H.G., Brown, Z.A., et al., 1983. Genital herpes simplex virus infection: clinical manifestations, course and complications. Ann. Intern. Med. 98, 958–972.)



**Laboratory Diagnosis of Herpes Simplex Virus Infections**



**U.S. Food and Drug Administration–Approved Antiviral Treatments for Herpesvirus Infections**

**Herpes Simplex 1 and 2**

Acyclovir

Penciclovir

Valacyclovir

Famciclovir

Trifluridine

**Varicella-Zoster Virus**

Acyclovir

Famciclovir

Valacyclovir

Varicella-zoster immune globulin

Zoster immune plasma

Live or adjuvanted subunit vaccine

**Epstein-Barr Virus**

None

**Cytomegalovirus**

Ganciclovira

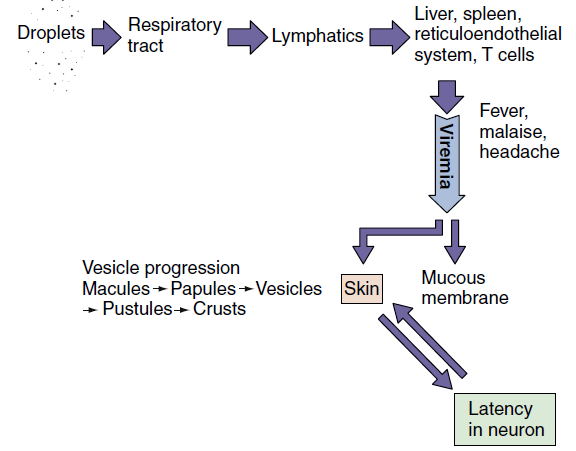
Valganciclovira

Foscarneta

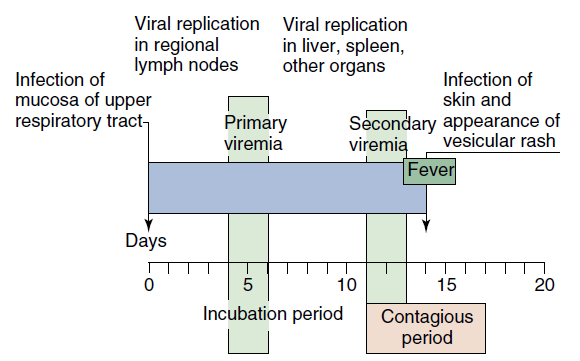
Cidofovira

aAlso inhibits herpes simplex and varicella-zoster viruses.

**Mechanism of spread of varicella-zoster virus (VZV) within the body. VZV initially infects the respiratory tract and is spread to the reticuloendothelial system and T cells and then by cell-associated viremia to the skin.**



**Time course of varicella (chickenpox). The course in young children, as presented in this figure, is generally shorter and less severe than that in adults.**



**Disease Mechanisms of Varicella-Zoster Virus**

Initial replication is in the respiratory tract.

Infects epithelial cells, fibroblasts, T cells, and neurons.

Can form syncytia and spread directly from cell to cell.

Spread by viremia in T cells to skin and causes lesions in successive crops.

Life-threatening pneumonia occurs in adults with primary infection caused by vigorous inflammatory response.

Can evade antibody clearance, and cell-mediated immune response is essential to control infection.

Disseminated life-threatening disease can occur in immunocompromised people.

Establishes latent infection of neurons, usually dorsal root and cranial nerve ganglia.

Herpes zoster is a recurrent disease; it results from virus replication along the entire dermatome.

Herpes zoster results from depression of cell-mediated immunity.

**Epidemiology of Varicella-Zoster Virus**

**Disease/Viral Factors**

Causes lifelong infection.

Recurrent disease is a source of contagion.ᑏ

**Transmission**

Virus is transmitted mainly by respiratory droplets but also by direct contact.

**Who Is at Risk?**

Children (aged 5 to 9 years) experience mild classic disease.

Teenagers and adults are at risk for more severe disease with potential pneumonia.

Immunocompromised people and newborns are at risk for life-threatening pneumonia, encephalitis, and progressive disseminated varicella.

Elderly and immunocompromised people are at risk for recurrent disease (herpes zoster [shingles]) caused by a waning immune response.

**Geography/Season**

Virus is found worldwide.

There is no seasonal incidence.

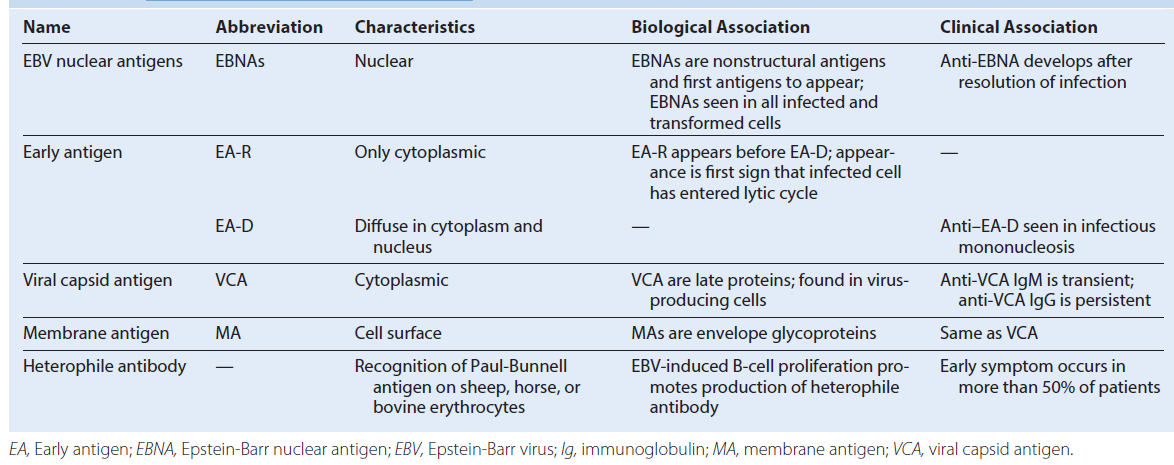
**Modes of Control**

Antiviral drugs are available.

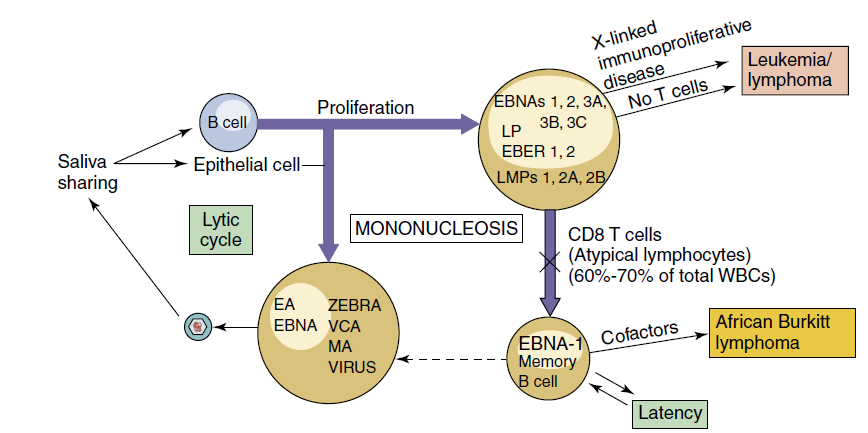
Varicella-zoster immunoglobulin is available for immunocompromised people and staff exposed to virus, as well as newborns of mothers showing symptoms within 5 days of birth.

Live vaccine (Oka strain) is available for children (varicella) and adults (zoster). Adjuvanted subunit vaccine also is available for zoster.

**Markers of Epstein-Barr Virus Infection**



Progression of Epstein-Barr virus (EBV) infection. Infection may result in lytic, latent, or immortalizing infection, which can be distinguished on the basis of production of virus and expression of different viral proteins and antigens. T cells limit the outgrowth of the EBV-infected cells and maintain the latent infection. *CD,* Cluster of differentiation; *EA,* early antigen; *EBER,* Epstein-Barr–encoded RNA; *EBNA,* Epstein-Barr nuclear antigen; *LMPs,* latent membrane proteins; *LP,* latent protein; *MA,* membrane antigen; *VCA,* viral capsid antigen; *WBCs,* white blood cells; *ZEBRA,* peptide encoded by the *Z* gene region.



**Disease Mechanisms of Epstein-Barr Virus**

Virus in saliva initiates infection of oral epithelia and tonsillar B cells.

There is productive infection of epithelial cells and B cells.

Virus promotes growth of B cells (immortalizes).

T cells are stimulated by infected B cells; they kill and limit B-cell outgrowth. Tcells are required for controlling infection.

Antibody role is limited.

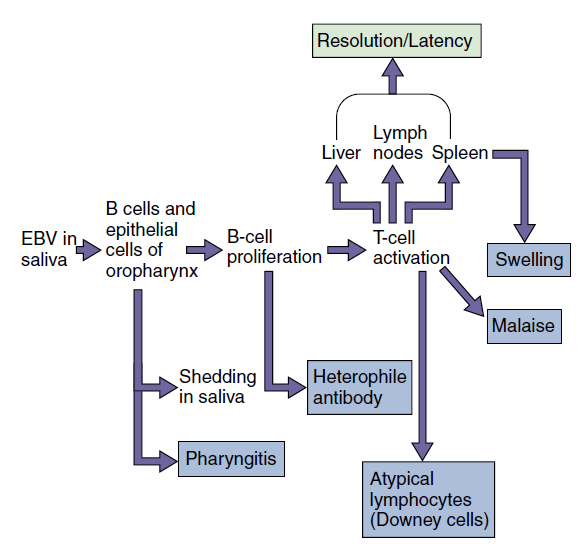
EBV establishes latency in memory B cells and is reactivated when the B cell is activated.

T-cell response (lymphocytosis) contributes to symptoms of **infectious mononucleosis.**

There is causative association with lymphoma in immunosuppressed people and African children living in malarial regions (African Burkitt lymphoma) and with nasopharyngeal carcinoma in China.

EBV-associated B-cell lymphomas may result from immunosuppression.

Pathogenesis of Epstein-Barr virus *(EBV).* EBV is acquired by close contact between persons through saliva and infects B cells. Resolution of the EBV infection and many of the symptoms of infectious mononucleosis result from activation of T cells in response to the infection.



**Epidemiology of Epstein-Barr Virus**

**Disease/Viral Factors**

Virus causes lifelong infection.

Recurrent disease is primary source of contagion.

Virus may cause asymptomatic shedding.

**Transmission**

Transmission occurs via saliva, close oral contact (“kissing disease”), or sharing of items such as toothbrushes and cups.

**Who Is at Risk?**

Children experience asymptomatic disease or mild symptoms.

Teenagers and adults are at risk for infectious mononucleosis.

Immunocompromised people are at highest risk for life-threatening neoplastic disease.

**Geography/Season**

Infectious mononucleosis has worldwide distribution.

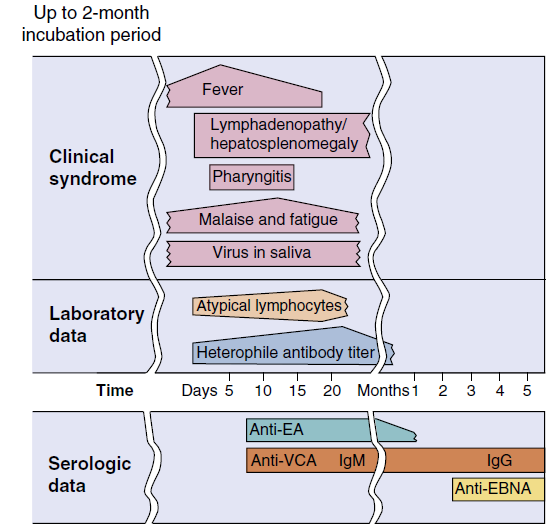
There is causative association with African Burkitt lymphoma in the malarial belt of Africa.

There is no seasonal incidence.

**Modes of Control**

There are no modes of control.

Clinical course of infectious mononucleosis and laboratory findings of those with the infection. Epstein-Barr virus infection may be asymptomatic or may produce the symptoms of mononucleosis.The incubation period can last as long as 2 months. *EA,* Early antigen; *EBNA,* Epstein-Barr nuclear antigen; *Ig,* immunoglobulin; *VCA,* viral capsid antigen.



**Diagnosis of Epstein-Barr Virus**

1. Symptoms

a. Mild headache, fatigue, fever

b. Triad: lymphadenopathy, splenomegaly, exudative pharyngitis

c. Other: hepatitis, ampicillin-induced rash

2. Complete blood cell count

a. Hyperplasia

b. Atypical lymphocytes (Downey cells, T cells)

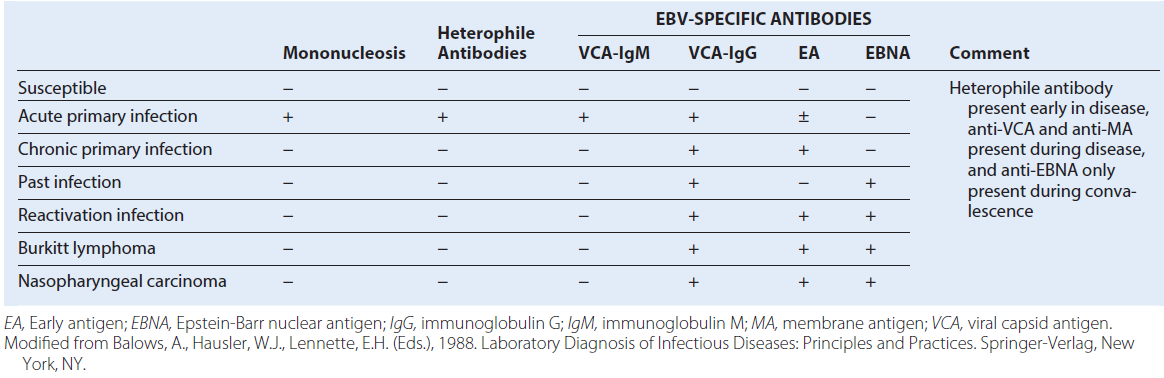
3. Heterophile antibody (transient)

4. EBV–antigen-specific antibody

5. Genome detection by PCR

*EBV, Epstein-Barr virus; PCR, polymerase chain reaction.*

**Serologic Profile for Epstein-Barr Virus Infection**



**Disease Mechanisms of Cytomegalovirus**

Acquired from blood, tissue, and most body secretions.

Causes productive infection of macrophages, epithelial cells, and other cells.

Establishes latency in hematopoietic stem cells and monocytes

Cell-mediated immunity is required for resolution and maintenance of latency and contributes to symptoms.

The role of antibody is limited.

Suppression of cell-mediated immunity allows recurrence and severe disease.

CMV generally causes subclinical infection.

**Sources of Cytomegalovirus Infection**

Neonate - Transplacental transmission, intrauterine infections, cervical secretions

Baby or child - Body secretions: breast milk, saliva, tears, urine

Adult - Sexual transmission (semen), blood transfusion, organ graft

**Epidemiology of Disease/Viral Factors**

Virus causes lifelong infection.

Recurrent disease is source of contagion.

Virus causes asymptomatic shedding.

**Transmission**

Transmission occurs via blood, organ transplants, and all secretions (urine, saliva, semen, cervical secretions, breast milk, and tears).

Virus is transmitted orally and sexually, in blood transfusions, in tissue transplants, in utero, at birth, and by nursing.

**Who Is at Risk?**

Babies

Babies of mothers who experience seroconversion during term are at high risk for congenital defects

Sexually active people

Blood and organ recipients

Burn victims

Immunocompromised people: symptomatic and recurrent disease

**Geography/Season**

Virus is found worldwide.

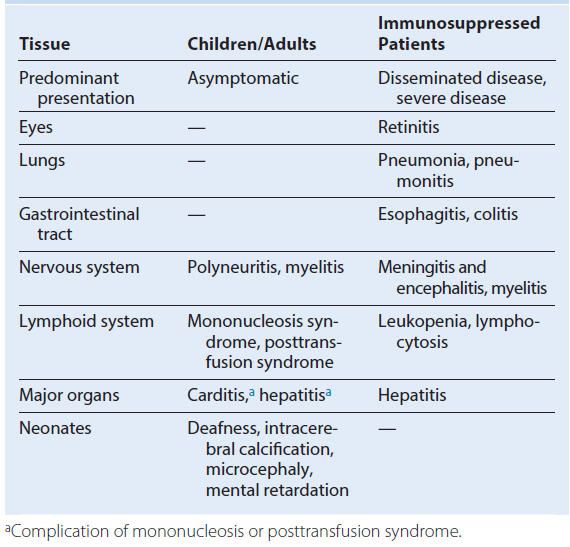
There is no seasonal incidence.

**Modes of Control**

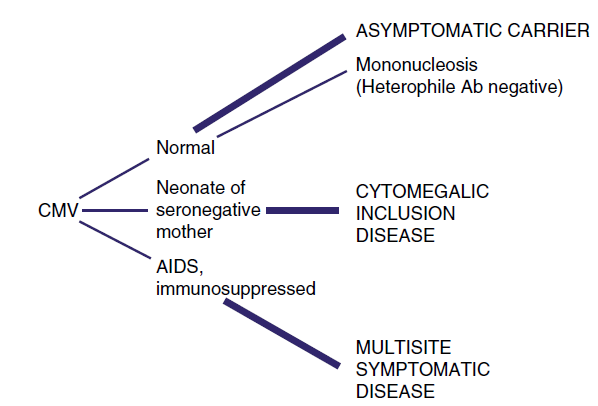
Antiviral drugs are available for serious disease.

Screening potential blood and organ donors for cytomegalovirus reduces transmission of virus.

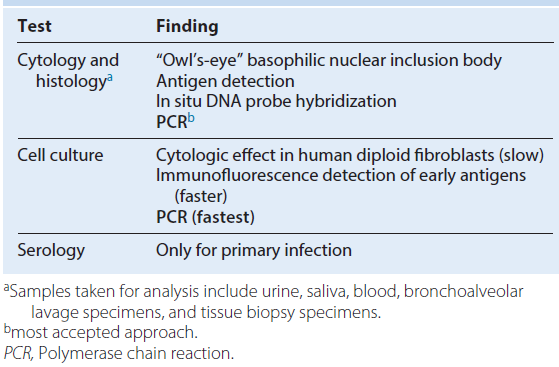
**Cytomegalovirus Syndromes**



Outcomes of cytomegalovirus *(CMV)* infections. The outcome of CMV infection depends very heavily on the immune status of the patient. *Ab,* Antibody; *AIDS,* acquired immunodeficiency syndrome.



**Laboratory Tests for Diagnosing Cytomegalovirus Infection**



**Clinical Summaries**

**Herpes Simplex Virus**

**Primary oral herpes:** A 5-year-old boy has an ulcerative rash with vesicles around the mouth. Vesicles and ulcers are also present within the mouth. Results of a Tzanck smear show multinucleated giant cells (syncytia) and Cowdry type A inclusion bodies. The lesions resolve after 18 days.

**Recurrent oral HSV:** A 22-year-old medical student studying for examinations feels a twinge at the crimson border of his lip and 24 hours later has a single vesicular lesion at the site.

**Recurrent genital HSV:** A sexually active 32-year-old woman has a recurrence of ulcerative vaginal lesions with pain, itching, dysuria, and systemic symptoms 48 hours after being exposed to ultraviolet B light while skiing. The lesions resolve within 8 days. Results of a Papanicolaou smear show multinucleated giant cells (syncytia) and Cowdry type A inclusion bodies.

**Encephalitis HSV:** A patient has focal neurologic symptoms and seizures. Magnetic resonance imaging results show destruction of a temporal lobe. Erythrocytes are present in the cerebrospinal fluid, and polymerase chain reaction is positive for viral DNA.

**Varicella-Zoster Virus**

**Varicella (chickenpox):** A 5-year-old boy develops a fever and a maculopapular rash on his abdomen 14 days after meeting with his cousin, who also developed the rash. Successive crops of lesions appear for 3 to 5 days, and the rash spreads peripherally.

**Zoster (shingles):** A 65-year-old woman has a belt of vesicles along the thoracic dermatome and experiences severe pain localized to the region.

**Epstein-Barr Virus**

**Infectious mononucleosis:** A 23-year-old college student develops malaise, fatigue, fever, swollen glands, and pharyngitis. After empirical treatment with ampicillin for a sore throat, a rash appears. Heterophile antibody and atypical lymphocytes are detected from blood.

**Cytomegalovirus**

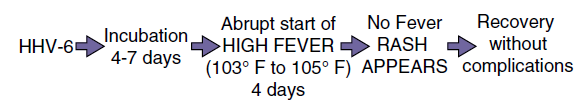
**Congenital CMV disease:** A neonate exhibits microcephaly, hepatosplenomegaly, and rash. Intracerebral calcification is noted on a radiograph. The mother had symptoms similar to mononucleosis during the third trimester of her pregnancy.

**Human Herpesvirus 6**

**Roseola (exanthem subitum):** A 4-year-old child experiences a rapid onset of high fever that lasts for 3 days and then suddenly returns to normal. Two days later, a maculopapular rash appears on the trunk and spreads to other parts of the body.

*CMV,* Cytomegalovirus; *HSV,* herpes simplex virus.

**Time course of symptoms of exanthem subitum (roseola) caused by human herpesvirus 6 *(HHV-6).* Compare these symptoms and this time course with those of fifth disease, which is caused by parvovirus B19**

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**PICORNAVIRUSES**

**Trigger Words**

Polio: flaccid paralysis, major and minor disease, fecal-oral

Coxsackievirus A: vesicular diseases, meningitis; coxsackievirus B (body): pleurodynia, myocarditis

Other echovirus and enteroviruses: like coxsackievirus and hepatitis A virus

Rhinoviruses: common cold, acid labile, does not replicate above 33° C

**Biology, Virulence, and Disease**

ᑏ Small size, icosahedral capsid, positive RNA genome with terminal protein

ᑏᑏGenome is sufficient for infection

ᑏᑏ Encodes RNA-dependent RNA polymerase, replicates in cytoplasm

**Picornaviridae**

**Enterovirus**

Poliovirus types 1, 2, and 3

Coxsackievirus A 24 types

Coxsackievirus B 6 types

Echovirusa 34 types

Parechovirus 16 types

Enterovirus 4

**Hepatovirus**

Hepatitis A virus

**Rhinovirus: >100 types+**

**Cardiovirus**

**Aphthovirus**

a*E*nteric, *c*ytopathic, *h*uman, *o*rphan + virus.

**Unique Properties of Human Picornaviruses**

Virion is a **naked, small** (25 to 30 nm), **icosahedral** capsid enclosing a single-stranded positive RNA genome.

Enteroviruses are resistant to pH 3 to pH 9, detergents, mild sewage treatment, and heat.

Rhinoviruses are labile at acidic pH; optimum growth temperature is 33° C.

**Genome is an mRNA.**

Naked genome is sufficient for infection.

Virus replicates in cytoplasm.

Viral RNA is translated into **polyprotein,** which is then cleaved into enzymatic and structural proteins.

Most viruses are **cytolytic.**

*mRNA, Messenger ribonucleic acid.*

Pathogenesis of enterovirus infection. The target tissue infected by the enterovirus determines the predominant disease caused by the virus*. Coxsackie,* Coxsackievirus; *echo,* echovirus; *HAV,* hepatitis A virus; *polio,* poliovirus.

**Disease Mechanisms of Picornaviruses**

Enteroviruses enter via the oropharynx, intestinal mucosa, or upper respiratory tract and infect the underlying lymphatic tissue; rhinoviruses are restricted to the upper respiratory tract.

In the absence of serum antibody, enterovirus spreads by viremia to cells of a receptor-bearing target tissue.

Different picornaviruses bind to different receptors, many of which are members of the immunoglobulin superfamily (i.e.,intercellular adhesion molecule-1).

The infected target tissue determines the subsequent disease.

Viral, rather than immune, pathologic effects are usually responsible for causing disease.

The secretory antibody response is transitory but can prevent the initiation of infection.

Serum antibody blocks viremic spread to target tissue, preventing disease.

Enterovirus is shed in feces for long periods.

Infection is often asymptomatic or causes mild, flulike, or upper respiratory tract disease.

**Epidemiology of Enterovirus Infections**

**Disease/Viral Factors**

Nature of disease correlates with specific enterovirus

Severity of disease correlates with age of person

Infection often asymptomatic, with viral shedding

Virion resistant to environmental conditions (detergents, acid, drying, mild sewage treatment, and heat)

**Transmission**

Fecal-oral route: poor hygiene, dirty diapers (especially in day-care settings)

Ingestion via contaminated food and water

Contact with infected hands and fomites

Inhalation of infectious aerosols

**Who Is at Risk?**

Young children: at risk for polio (asymptomatic or mild disease)

Older children and adults: at risk for polio (asymptomatic to paralytic disease)

Newborns and neonates: at highest risk for serious coxsackievirus, echovirus, and enterovirus disease

**Geography/Season**

Viruses have worldwide distribution; wild-type polio virtually eradicated in most countries because of vaccination programs

Disease more common in summer

**Modes of Control**

For polio, live oral polio vaccine (trivalent OPV) or inactivated trivalent polio vaccine (IPV) is administered

For other enteroviruses, no vaccine; good hygiene limits spread

**Transmission of enteroviruses. The capsid structure is resistant to mild sewage treatment, salt water, detergents, and temperature changes, allowing these viruses to be transmitted by fecal-oral routes, by fomites, and on hands**.

**Summary of Clinical Syndromes Associated with Major Enterovirus Groups a**

**Advantages and Disadvantages of Polio Vaccines**

*IPV,* Inactive polio vaccine; *OPV,* live oral polio vaccine.

**Clinical Summaries**

**Poliovirus**

**Polio:** A 12-year-old girl from Nigeria has headache, fever, nausea, and stiff neck. Symptoms improve and then recur several days later, with weakness and paralysis of her legs. She has no history of polio immunization.

**Coxsackievirus A**

**Herpangina:** Vesicular lesions on the tongue and roof of the mouth of a 7-year-old patient accompany fever, sore throat, and pain on swallowing.

**Coxsackievirus B (B for body)**

**Pleurodynia:** A 13-year-old boy has fever and severe chest pain with headache, fatigue, and aching muscles lasting for 4 days.

**Coxsackievirus or Echovirus**

**Aseptic meningitis:** A 7-month-old infant with fever and rash appears listless, with a stiff neck. A sample of his cerebrospinal fluid contains lymphocytes but has normal glucose and no bacteria. Full recovery occurs within 1 week.

**TOGAVIRUSES**

**Trigger Words**

Rubella: German measles, congenital disease, rash, vaccine

**Biology, Virulence, and Disease**

ᑏ Small size, envelope surrounds icosahedral nucleocapsid, (+) RNA genome

ᑏᑏ Encodes RNA-dependent RNA polymerase, replicates in cytoplasm

ᑏᑏ Early and late mRNA and proteins produced

ᑏᑏVirus spreads in blood to target tissues, including neurons and brain

ᑏᑏ Antibody can block viremia and disease

ᑏᑏ Prodrome of flulike symptoms caused by interferon and cytokine response

ᑏᑏ Arboviruses: equine encephalitis viruses (WEE, EEE, VEE)

ᑏᑏ Rubella: benign childhood rash, swollen glands. Adult complications include

arthritis, encephalitis. Congenital infection: teratogenic, cataracts, deafness, microcephaly, etc.

**Epidemiology**

ᑏᑏ Rubella: aerosol spread, only infects humans, unvaccinated individuals at

risk, fetus at high risk

**Diagnosis**

ᑏᑏ RT-PCR, ELISA

**Treatment, Prevention, and Control**

ᑏᑏ Live attenuated rubella vaccine at 1 year of age in MMR; booster at 4 to 6 years

Spread of rubella virus within the host. Rubella enters and infects the nasopharynx and lung and then spreads to the lymph nodes and monocyte-macrophage system. The resulting viremia spreads the virus to other tissues and the skin. Circulating antibody can block the transfer of virus at the indicated points *(X)*. In an immunologically deficient pregnant woman, the virus can infect the placenta and spread to the fetus.

**Epidemiology of Rubella Virus**

**Disease/Viral Factors**

Rubella infects only humans.

Virus can cause asymptomatic disease.

There is one serotype.

**Transmission**

Respiratory route

**Who Is at Risk?**

Children: mild exanthematous disease.

Adults: more severe disease with arthritis or arthralgia.

Fetus <20 weeks: congenital defects.

**Modes of Control**

Live attenuated vaccine administered as part of the measles mumps-rubella vaccine.

**Prominent Clinical Findings in Congenital Rubella Syndrome**

Cataracts and other ocular defects

Heart defects

Deafness

Intrauterine growth retardation

Failure to thrive

Mortality within the first year

Microcephaly

Mental retardation

**Rubella:** A 6-year-old girl from Romania developed a faint rash on her face, accompanied by mild fever and lymphadenopathy.

Over the next 3 days, the rash progressed to other parts of the body. She had no history of rubella immunization.