Medicine
TEAM 437

MED437
KING SAUD UNIVERSITY

Herpes viral infections

Objectives :

1. To know the clinically important HHVs.
2. To know the common characteristics of HHVs.
3. To know the common modes of transmission of different HHVs
4. To know the clinical features of these infections, diagnostic methods and treatment.

Done by :

Team leader: Salem Al-Ammari

Team members: - Fahad alfaiz. - Batoul AlRuhaimi

- Mohammed AlAsqah

Revised by :

Aseel Badukhon

Resources :

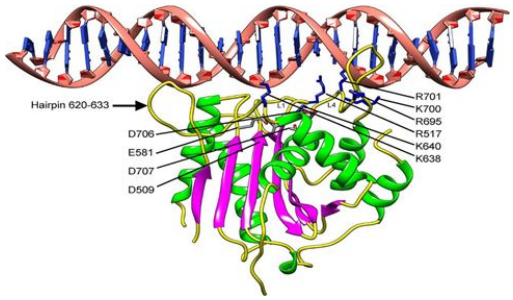
Doctor's slides + Team 436
Lecturer: Dr. Mazin Barry
Same as 436 slides: No
Davidson's Self-assessment in Medicine

Herpes Virus

Virus	Infection
Herpes Simplex Virus type 1 (HSV 1)	1. Herpes labialis ('cold sores') 2. Keratoconjunctivitis 3. Finger infections ('whitlows') 4. Encephalitis 5. Primary stomatitis (inflammation of the mouth and lips, not related to the stomach) 6. Genital infections (especially oral sex but more common in type 2)
Herpes Simplex Virus type 2 (HSV 2)	1. Genital infections 2. Neonatal infection (acquired during vaginal delivery)
Varicella Zoster Virus (VZV)	1. Chickenpox (العنقز) 2. Shingles (herpes zoster) (الحزام الناري)
Cytomegalovirus (CMV)	1. Congenital infection (3rd trimester of pregnancy) 2. Disease in immunocompromised patients 3. Pneumonitis 4. Retinitis 5. Colitis 6. Systemic infection
Epstein Barr Virus (EBV)	1. Infectious mononucleosis (triad of sore throat, fever, neck lymphadenopathy) 2. Burkitt's lymphoma 3. Nasopharyngeal carcinoma 4. Oral hairy Cell leukoplakia (AIDS patient) (and post transplant lymphoproliferative disorders)
Human Herpesvirus 6 (HHV-6) and 7 (HHV-7) Roseolovirus	1. Exanthem subitum (Roseola): three day fever in children 2. Disease in immunocompromised patients and children 3. Bell's Palsy (all other herpes can cause it but HHV-6,7 are the most common)
Human Herpesvirus 8 (HHV-8)	Associated with Kaposi's sarcoma (purple papules or plaques around the mouth and swelling in the scrotum.)
Herpes simiae (Herpes B or Monkey B Virus)	Fatal human cases of myelitis and hemorrhagic encephalitis have been reported following bites, scratches, or eye inoculation of saliva from monkeys.

Herpes Virus

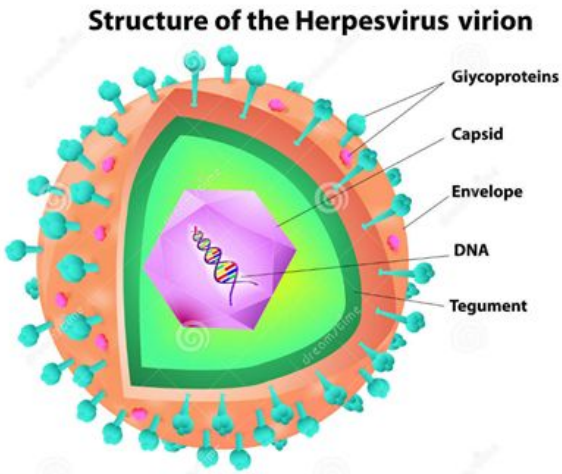
Characteristics



- All DNA viruses
- All encapsulated
- All have latency after the initial infection
- Mostly require **close contact** for transmission

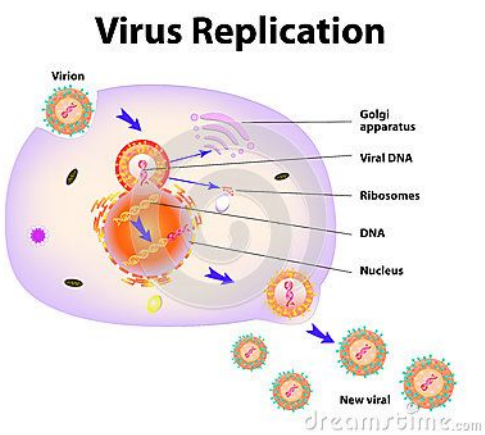
Structure

- Herpesviruses have a unique four layered structure:
  - A core containing the large double stranded DNA genome
  - Genome is enclosed by an icosadeltahedral capsid which is composed of capsomers
  - The capsid is surrounded by an amorphous protein coat called the tegument (between the envelope and the capsid).
  - It is encased in a glycoprotein bearing lipid bilayer envelope. (envelope derived from the host' nuclear membrane).



Viral Replication

- Upon entry into the host cell nucleus, three distinct phases of gene transcription and protein synthesis are initiated producing the immediate-early, early, and late proteins
- Viral nucleocapsid assembly occurs within the host cell nucleus.
- The virus acquires its final envelope by budding into cytoplasmic vesicles



HSV 1 and HSV 2

HSV1	HSV2
Oral (Non-genital herpes infection)	Genital Herpes infection
Encephalitis (because it's close to the brain "trigeminal ganglion")	Neonatal infection (vertical transmission at time of delivery) is associated with congenital malformations, intrauterine growth retardation (IUGR), chorioamnionitis (AKA intra-amniotic infection) , and even neonatal death.

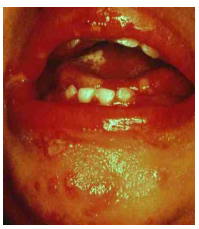
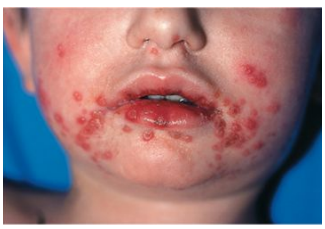
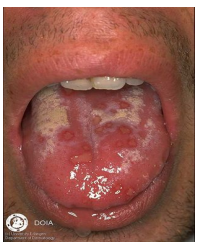
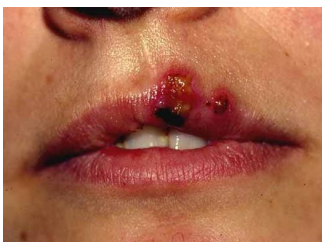
- Both can cause similar illness (genital or oral lesions): e.g. oral sex may transmit HSV1 to genitals
- They both cause primary and recurrent infections (primary infection is more severe and causes systemic manifestations : fever, malaise…)

Transmission and Pathophysiology

- Transmission is by close direct contact with body secretions
- Exposure to HSV at mucosal surfaces or abraded skin sites permits entry of virus and initiation of its replication in cells of the epidermis and dermis
  - After initial infection the virus infects the sensory and autonomic nerves and becomes dormant in the ganglion (latent infection) (**trigeminal nerve for HSV-1** 'usually reactivates in the face' and **sacral route for HSV-2** 'usually reactivates in the genital area')
- Incubation period 2-12 days, lesions last 3- 4 weeks

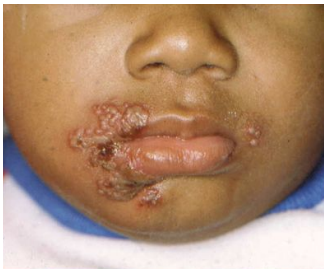
HSV-1 Gingivostomatitis

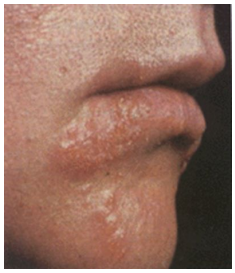
- HSV 1 is very common in children especially daycare and schools
- Gingivostomatitis and pharyngitis are the most frequent clinical manifestations of first-episode HSV-1 infection
- Primary infection is usually asymptomatic and unnoticed, but symptomatic primary infection is associated with:
  - Systemic manifestations (e.g., fevers, sore throat, tender cervical lymphadenopathy, malaise)
  - As well as oral lesions (groups of vesicles on patches of erythematous skin) painful blisters around mouth and in oral mucosa

Herpes Labialis (cold sores)

- Recurrent herpes labialis is the most frequent clinical manifestation of reactivation HSV infection
- Disease usually less severe than first episode
- Symptoms typically include a burning pain followed by small blisters or sores

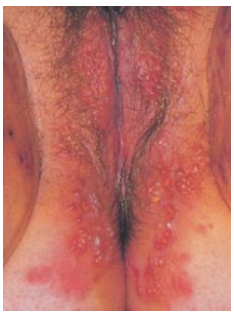


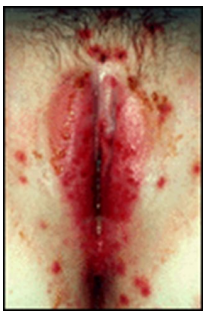


- Usually at the area where mucosa of lips meets skin. Recurrent disease is less severe, it could be multiple recurrences and usually patients can predict when sores are coming (prodrome), they feel tingling around their lips, as soon as tingling sensation starts patients are infectious even before sores appear.

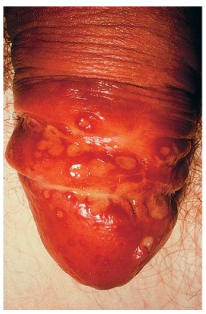
Genital HSV2

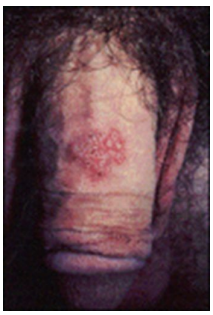
- Lasting up to 3 weeks:
- First attack: Very painful genital vesicles or pustules other findings are tender inguinal lymphadenopathy and vaginal and/or urethral discharge, myalgias,itching, and dysuria + constitutional symptoms (e.g. fever, headache, malaise, pain)
- Recurrent attack: no systemic symptoms.
  - Recurrent is less severe and without systemic manifestations, only a rash so a lot of women get the vesicles of the recurrent attack and they don't even realise it's there. That's why it's important when a pregnant woman goes in labor that the OB should examine her for active herpetic lesions.





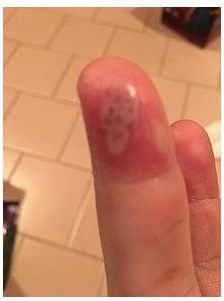
Herpetic ulceration of the vulva





Penile herpes simplex (HSV-2) infection

- Inoculation lesion on the finger gives rise to a paronychia termed a 'whitlow' → in contacts of patients with herpetic lesions. It was formerly seen in health-care workers and dentists, but is prevented by protective gloves.
- HSV-1 is the cause in 60% of cases of herpetic whitlow, and HSV-2 is the cause in the remaining 40%



Diagnosis

- Can be made clinically when characteristic lesions are recognized.
- Serology: diagnostic test: Direct fluorescent assay and ELISA
  - IgM (+ after 1 to 2 weeks of infection "acute")
  - IgG (+ 3-4 weeks after infection and stays forever "latent")
- Viral culture: not used
- Cytology
- PCR of CSF(only if presented with meningoencephalitis)

Treatment

- Start treatment with prodrome symptoms (tingling/burning) before lesions appear
  - Acyclovir (first line) 400 mg po 5 x/day (q4h while awake) x 5 days
  - Famciclovir 500 mg po bid x 7 days
  - Valacyclovir 2 gm po q12h x 1 day
- Topical treatment (mild cases)
  - Penciclovir 1% cream q2h during day x 4 days
  - Acyclovir 5% cream 6x/day (q3h) x 7 days

- For mild disease or recurrent: topical Acyclovir or Penciclovir
- Systemic antivirals should be started as the pt develops the tingling sensation bc it shortens the duration of disease if you start early
- Master the boards: Foscarnet is used in acyclovir resistant herpes)

Herpes Encephalitis

- Clinical Setting:
  - **HSV-1 is most common** cause of sporadic encephalitis
  - Risk factor: use of natalizumab for treatment of multiple sclerosis or Crohn's disease
  - Survival and recovery from neurological sequelae are related to mental status at time of initiation of therapy. Elderly, dementia, and previous CVA patients are unlikely to recover
  - **Early** diagnosis and treatment imperative immediate treatment is essential for good prognosis
- Etiologies:
  - HSV-1
  - HSV-2 causes occasional cases
- Diagnosis: it has a tendency to infect the temporal lobe
  - PCR analysis of CSF for HSV-1 DNA is 100% specific and 75–98% sensitive. Sensitivity depends on the timing of the lumbar puncture
  - 25% CSF samples drawn **before day 3 are negative** by PCR if clinical suspicion is high, you should repeat CSF after 72 hours
  - Negative PCR is associated with decreased protein and <10 WBC/mm3 in CSF. You should always examine their oral mucosa and if they have active herpes sore that's a very strong clue that they have Herpes Encephalitis.
- Treatment: Acyclovir IV 12.5 mg/kg IV (infuse over 1 hr) q8h x 21 days (prolonged therapy), make sure it's IV and prolonged course!

Varicella Zoster Virus (VZV)

Characteristics

1. Primary infection: Chickenpox (العنقز)
2. Recurrent infection: Herpes zoster (shingles) (الحزام الناري)

Clinical Syndrome

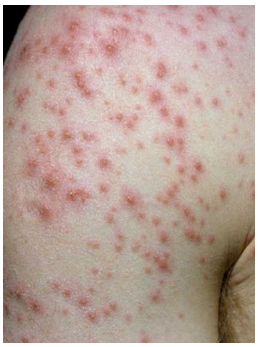
- Chickenpox
- Shingles (single dermatomal or multiple dermatomes) it's the relapse form of chickenpox
- Disseminated VZV disease/organ involvement could involve the brain causing encephalitis
- Emerging data suggests VZV may cause vasculopathy of cerebral, temporal, and other arteries
- Suggested as possible cause of Giant Cell arteritis

Pathophysiology

- The virus is spread by the **respiratory route** (airborne and contact) and replicates in the nasopharynx or upper respiratory tract.
- Followed by localized replication at an undefined site, which leads to seeding of the reticuloendothelial system and, ultimately, viremia.
- The virus establishes latency within the dorsal root ganglia especially lumbar and sacral spines.(During reactivation which is common in shingles it affect the dermatome of that spine). New researches linked VZV as the cause of idiopathic vasculitis such as Giant cell arteritis.
- Chickenpox incubation period: 10 to 21 days

Clinical Features of Chickenpox

Even 3 weeks after exposure patient may present with chickenpox



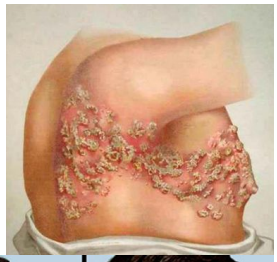
- Overall, chickenpox is a disease of **childhood**, because 90% of cases occur in children younger than 13 years of age.
- Highly contagious
- Symptoms include: fever, headache, malaise, Iching ,blisters-like vesicular rash:
  Appears first on chest, back, and face, and then spread over the entire body. the rash lasts for 2 weeks , there are different ages of these rashes ,some of them would be fresh and others are older that would scab over and dry out. Patients are considered infectious until all the lesions scab over and dry out

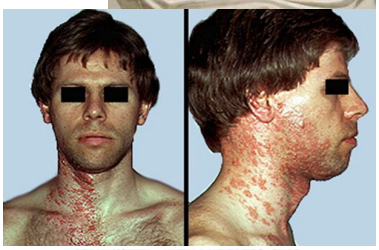
Complications

Rare and usually in adults > 20

- Pneumonia
- Encephalitis
- Bacterial skin and soft tissue infections

Clinical Features of Herpes Zoster (Shingles)





Reactivation of VZV leads to VZ

- It could involve a single dermatome or multiple dermatomes it can also cause disseminated VZV with multiple organ involvement in immunocompromised . unilateral distribution most often appears as a single stripe of blisters that wraps around either the left or the right side of your trunk, hence the latency in the lumbar spine.

- Facial infection with VZV can cause Ramsay hunt syndrome characterized by: painful rash on the outer ear , Lower motor neuron paralysis of the facial nerve, Loss of taste sensation on the anterior two third of the tongue.
- In case of facial infection patient should be immediately referred to ophthalmology and ENT because it can cause blindness and deafness

Investigations

- Clinical picture Very clear
- Serology Most commonly used , IgM(4-6 weeks after infection) positive in chickenpox / IgG positive in shingles) because it happens in reactivated reactivation!
- Viral culture Not used
- PCR biopsy of the lesions + CSF

Treatment of Chickenpox

- Acyclovir 800 mg po 5x/day x 5-7 days, start within 24 hrs of rash
- Valacyclovir 1000 mg po TID x 5 days
- Famciclovir 500 mg po TID
- immunocompromised: Acyclovir 10–12 mg/kg **IV** (infused over 1 hour) q8h x 7 days

(Acyclovir oral 1st line, IV in immunocompromised at a lesser dose and duration than encephalitis)

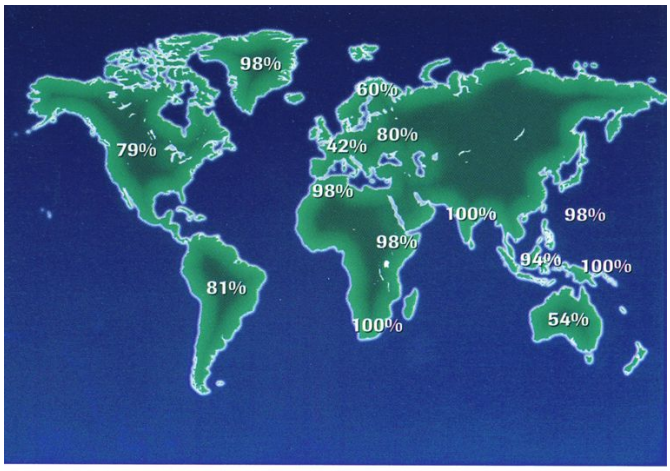
Prevention

- **VZV vaccination** at a minimum age of 1 yr: 2 dose series (12-15 months and 4-6 years)
- <5% of cases of varicella but >50% of varicella-related deaths occur in adults >20 yrs of age:
- **VZV immunoglobulin (VZIG)** post-exposure prophylaxis (125 units/10 kg) in susceptible persons at greater risk for complications (immunocompromised such as HIV, malignancies, pregnancy, and steroid therapy) as soon as possible after exposure (<96 hrs).If varicella develops, initiate treatment quickly (<24 hrs of rash) with Acyclovir
- If admitted need airborne infection **isolation and contact precautions** (HCW 'health care worker' with VZV Ab IgG positive or history of chickenpox do not need PPE 'Personal protective equipment'). Rare complications of VZV (pneumonia, superinfection cellulitis, Encephalitis)
- Susceptible adults should be vaccinated, check Antibody in adults with negative or uncertain history of varicella (10-30% will be Ab-neg) and vaccinate those who are Ab-negative.

Cytomegalovirus (CMV)

Characteristics

- Worldwide distribution; The largest virus that infects human beings.
- Latency after primary infection.
- Infection ranges from asymptomatic to severe multisystemic disease.



CMV Seroepidemiology

Clinical Features

- Primary infection:
  - Asymptomatic
    OR
  - Infectious mononucleosis syndrome: clinically identical to that caused by EBV. It is estimated that about 8 percent of infectious mononucleosis (IM) cases are caused by CMV. Persistent fever, Sore throat, and lymphadenopathy are characteristic IM symptoms.
- Secondary infections in Immunocompromised patients esp.solid organ transplant (SOT) and hematopoietic stem cell transplant (HCT) recipients

Cytomegalovirus in Transplant Patients (The doctor stressed that its important)

- SOT recipient's disease onset was early post-transplant (first 100 days) but with the use of effective prophylaxis disease is now often "late-onset" and occurs following the discontinuation of prophylaxis

Risk Factors important

- SOT: Seropositive donor (D+) and seronegative recipient (R-). Lymphocyte depleting antibody therapy (thymoglobulin, ATG, OKT-3, alemtuzumab)
- HCT: Seronegative donor (D-) and seropositive recipient (R+) .T-cell depleted or cord blood transplants, Graft versus host diseases (GVHD)

Clinical Disease

- "CMV syndrome" (fever, leukopenia, and thrombocytopenia w/o other end-organ disease)
- Gastrointestinal disease (colitis, esophagitis, enteritis)
- Hepatitis very high ALT + AST
- Pneumonitis multilobar patch disease
- CNS disease (meningoencephalitis, myelitis)
- Retinitis (common in AIDS pts should be routinely seen by an ophthalmologist to look specifically for CMV Retinitis bc it can progress without the pt knowing and cause blindness)
- Multisystem (cystitis, nephritis, etc.)

Retinitis

Investigations

Diagnosis almost always depends on laboratory confirmation and cannot be made on clinical grounds alone.

- Serologic tests (antigen detection): Serological tests can identify latent (IgG) or primary (IgM) simple detection of anti-HCMV antibody is not generally useful because the incidence of HCMV infection in the population is so high, and periodic inapparent recurrent infections occur frequently.
- PCR (most common test, used on serum, CSF, tissue) from blood , qualitative and quantitative PCR to know the viral load and for follow up after treatment
- Viral cultures: from blood, urine, tissue, lacks specificity. (slowly growing)
- pp65 antigen: less commonly used, not recommended in neutropenic patients
- Histopathology (gold standard to confirm end-organ disease) bronchoscopy and biopsy in pneumonitis , colonoscopy and biopsy in colitis ,The virus can also be identified in tissues by the presence of characteristic intranuclear 'owl's eye' inclusions (see the fig). Detection of CMV in urine is not helpful in diagnosing infection, except in neonates, since CMV is intermittently shed in the urine

Treatment (acyclovir is NOT used with CMV)

- Ganciclovir 5 mg/kg IV q12h (1st line treatment) OR
- Valganciclovir 900 mg po q12h
- Foscarnet (90 mg/kg IV q12h) or cidofovir (5 mg/kg IV once weekly) (If CMV is resistant to ganciclovir)
- Treatment duration should be individualized: Continue treatment until:
  - CMV PCR or antigenemia has become undetectable
  - Clinical evidence of disease has resolved . At least 2-3 weeks of treatment

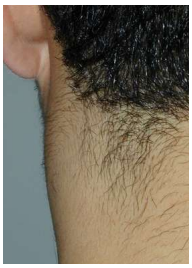
Epstein-Barr Virus (EBV)

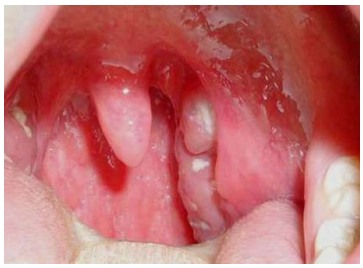
Characteristics

- Ubiquitous human herpes virus.
- By adulthood 90 to 95% of most populations are positive.
- Spread occurs by intimate contact between susceptible individuals and asymptomatic shedders of EBV. Even cheek to cheek kisses can transmit it
- Mostly causes asymptomatic infections.
- Carcinogenic: Strong association with African Burkitt's lymphoma & Nasopharyngeal carcinoma.

Clinical Features

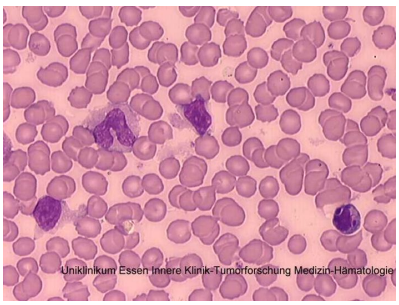
- Infectious mononucleosis, symptoms include:
  - Fever
  - Sore throat
  - Lymphadenopathy tender

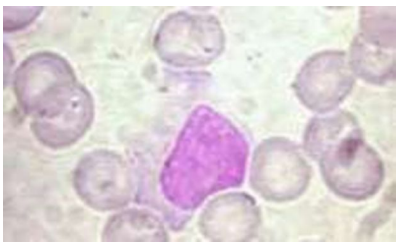




Investigations

- Serologic:
  - Transient appearance of heterophile antibodies (weak antibodies) 70-92% sensitivity and 96-100% specificity 📍 diagnostic test which is detected by the Paul–Bunnell or 'Monospot' test, (heterophile AB is weak antibodies produced by non specific B cells)
  - Later on, Permanent emergence of antibodies to EB
- Hematologic Findings: in peripheral blood smear
  - >50% mononuclear cells
  - Lymphocytosis (>10% atypical lymphocytes as demonstrated in the pics.)
  - Neutropenia
  - Thrombocytopenia
  - EBV specific antibodies





Treatment

- Treatment of infectious mononucleosis is largely supportive because more than 95% of the patients recover uneventfully without specific therapy .
- Corticosteroids (in patients who are unable to swallow and severe cases)

Cases from the Doctor

Case 1

36 Y/O Indian professor presents to ID clinic with recurrent oral vesicular lesions that lasts for 10 to 14 days, 4 to 5 lesion around his lip, slightly painful, interferes with his lecturing, No fever, no oral cavity lesions, gets these episodes almost on a monthly basis. Previously diagnosed with HSV-1, took acyclovir on occasion with good results, Brought on occasionally with stress but he's is still annoyed by the recurrency of the disease

- You recommend? Chronic suppressive therapy with acyclovir
- The treatment of recurrent HSV-1 infection (herpes labialis) is ACYCLOVIR, but if it was not responding well, we give it with steroids .

Case 2

32 Y/O 40 weeks pregnant lady who is in labor, O.B. found her to have genital vesicular lesions suspected to be HSV-2, She had similar genital lesions twice in the past 4 years, O.B. calls for medical opinion.

- You recommend? C-Section if possible with IV acyclovir till delivery
- Refer baby to neonatal ID once born. to start him on acyclovir

Summary:

HSV1 Oral **(Trigeminal)**	HSV2 Genital Herpes **(Sacral)**
1st episode: Gingivostomatitis and pharyngitis Recurrent: herpes labialis	1st episode: +Systemic symptoms Recurrent: - Systemic symptoms
Complication: Encephalitis ("trigeminal ganglion") Risk factor: use of natalizumab	Complication: Neonatal infection (vertical)

- Transmission: close direct contact with body secretions > then go to sensory and autonomic nerves > dormant in the ganglion (latent infection)
- Incubation period: 2-12 days, lesions last 3- 4 weeks

- Diagnosis: Could be clinically, Serology (IgM post 1st 2nd weak, IgG post 3rd 4th weak and stays forever), PCR (in Encephalitis ~if (-) repeat after 72 hours)

- Treatment: with prodrome symptoms before lesions appear: Acyclovir (5d)/ Famciclovir (7d)/ Valacyclovir.

  Topically: Penciclovir (4d), Acyclovir (7d).

  Encephalitis: Acyclovir IV (21d)

  ~~~ if resistant Foscarnet.
  ~~~

Summary:

Varicella Zoster Virus (VZV)

spread by the **respiratory route** (airborne and contact) > URT > RES > Viremia+Chickenpox ...> latency in the dorsal root ganglia > if ↓immune > Shingles

Primary infection: **Chickenpox**	**Recurrent** infection: Herpes **zoster (shingles)**
- Incubation period: 10 to 21 days - Complication: Pneumonia, Encephalitis, Skin and soft tissue	Complication: • **Ramsay hunt syndrome:** painful rash on the outer ear, facial nerve paralysis, Loss of taste on the anterior 2/3 of the tongue. • should be immediately referred to ophthalmology and ENT because it can cause blindness and deafness
Symptomes:	
Fever, headaches, fatigue	
Itching, blisters-like vesicular rash each lasts for 2 weeks - 1st chest, back, face > All body	In specific Dermatomes: Severe pain, Rash, Paresthesia, Itching.
Serology: IgM(4-6 weeks post infection)	Serology: IgG
	Oral Acyclovir start within 24 hrs of rash (IV in immunocompromised)

- Diagnosis: generally, clinically, **Serology** (Most commonly used), PCR.
- Prevention**: VZV vaccination** at a minimum age of 1 yr: 2 dose series (12-15 months and 4-6 years)
- **VZV immunoglobulin (VZIG)** post-exposure prophylaxis in susceptible persons at greater risk for complications (immunocompromised such as HIV, malignancies, pregnancy, and steroid therapy) as soon as possible after exposure (<4 days).
- If varicella develops, initiate treatment quickly (<24 hrs of rash) with Acyclovir
- If admitted need airborne infection **isolation and contact precautions** (HCW with VZV Ab IgG positive or history of chickenpox do not need PPE).
- Susceptible adults should be vaccinated, check Antibody in adults with negative or uncertain history of varicella (10-30% will be Ab-neg) and vaccinate those who are Ab-negative.

Summary:

Cytomegalovirus (CMV)

- The largest virus that infects human beings.
- CMV persists for the lifetime of its host; reactivation may therefore occur.
- Mostly Infectious mono caused By EBV is mono test (+), while if caused by CMV it's (-)

Primary infection	Secondary infections
Asymptomatic or Infectious mononucleosis syndrome	Risk Factors important: ~SOT: Seropositive donor (D+) and seronegative recipient (R-). Lymphocyte depleting antibody therapy (thymoglobulin, ATG, OKT-3, alemtuzumab). Used to be early but now due to prophylaxis is now late. ~HCT: Seronegative donor (D-) and seropositive recipient (R+) . T-cell depleted or cord blood transplants, Graft versus host diseases (GVHD)

Clinical Disease:

- Retinitis (AIDS)
- Colitis
- Esophagiti
- Pneumonitis
- Encephalitis

Investigations: (NOT CLINICALLY)

- Serologic tests (not useful)
- PCR (most common test, used on serum, CSF, tissue)
- Viral cultures: from blood, urine, tissue, lacks specificity.
- pp65 antigen: less commonly used, not recommended in neutropenic patients
- Histopathology (gold standard to confirm end-organ disease)

Treatment:

- Ganciclovir (1st line treatment) – Foscarnet/cidofovir (If CMV is resistant to ganciclovir)

- Continue treatment until:
  - CMV PCR or antigenemia has become undetectable
  - Clinical evidence of disease has resolved . At least 2-3 weeks of treatment

Summary:

Epstein-Barr Virus (EBV)

Carcinogenic, Strong association with African **Burkitt's** lymphoma & Nasopharyngeal carcinoma.

Mostly asymptomatic	• Infectious mononucleosis, symptoms include:
	  ○ Fever
	  ○ Sore throat
	  ○ Lymphadenopathy tender

- Serologic:
  - Transient appearance of heterophile antibodies (weak antibodies) 70-92% sensitivity and 96-100% specificity 📍 diagnostic test which is detected by the Paul–Bunnell or 'Monospot' test, (heterophile AB is weak antibodies produced by non specific B cells)
  - Later on, Permanent emergence of antibodies to EB
- Hematologic Findings: in peripheral blood smear
  - >50% mononuclear cells
  - Lymphocytosis (>10% atypical lymphocytes as demonstrated in the pics.)
  - Neutropenia
  - Thrombocytopenia
  - EBV specific antibodies

- Treatment of infectious mononucleosis is largely supportive
- ~Corticosteroids (in patients who are unable to swallow and severe cases)

Questions

1- EPV is usually associated with which of the following malignancies ?
a) esophageal carcinoma
b) multiple myeloma
c) Burkitt's lymphoma
d) small cell carcinoma of the lung

2- 34 years old sexually active female presented to clinic with fever ,multiple painful genital vesicles and dysuria what is your next step?
a) start ganciclovir IV
b) serology
c) start Acyclovir oral
d) PCR

3- A 22 year old bed bound man after a RTA was brought to ED with SOB and was diagnosed as acute asthma exacerbation and treated with salbutamol and IV steroids. 24 hours later he developed rash around mouth and ear. What drug should be given at the time?
a. Acyclovir
b. Imatinib
c. Vancomycin

4- Which one of the following presents with a rash in all the developmental stages?
a. Chicken pox
b. Measles
c. Rubella
d. Rift Valley hemorrhagic fever

Answer:
1- C
2- C
3- A
4- A

11.5. A 26 year old pregnant woman, in the seventh month of pregnancy, presents concerned that she was visited 5 days ago by her niece who the next day developed an itchy vesicular rash. The niece stayed in her house for 3 days. The niece saw her family physician on her return home and has been diagnosed with chickenpox. The woman is concerned because she does not remember ever having chickenpox as a child, a fact confirmed by her mother. You arrange to check a varicella zoster serology, which is negative. Which of the following should you offer to prescribe?

A. Aciclovir orally for 7 days
B. Intravenous immunoglobulin
C. Vaccination against varicella zoster virus
D. Valaciclovir orally
E. Varicella zoster immunoglobulin

11.5. Answer: E.
The patient is non-immune to varicella zoster virus (VZV) and has had a significant exposure during pregnancy within the last 7 days so should receive passive immunisation with varicella zoster immunoglobulin. Immunoglobulin would not have as high levels of antibodies. Vaccination would take too long to generate immunity. Treatment with aciclovir or valaciclovir is not indicated