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HERPES SIMPLEX VIRUS (HSV)

PRESENTED BY

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I N T R O D U C T I O N -

- Herpes (Greek : Creep or Crawl)
- Herpes Simpler Virus Belong to the Ubiquitous Herpesviridae Family.
- Human Herpes Simplex Virus Causes Contagious Infection With a Large Reservoir in the General Population.
- HSV Has a Potential For Significant Complications In The Immunocompromised Host.

M O R P H O L O G Y

- **1. Necleocapsicl** :- Virions Are Spherical .

150-200 NMIN Diameter.

-Symmetry - Icosahedral Symmetry.

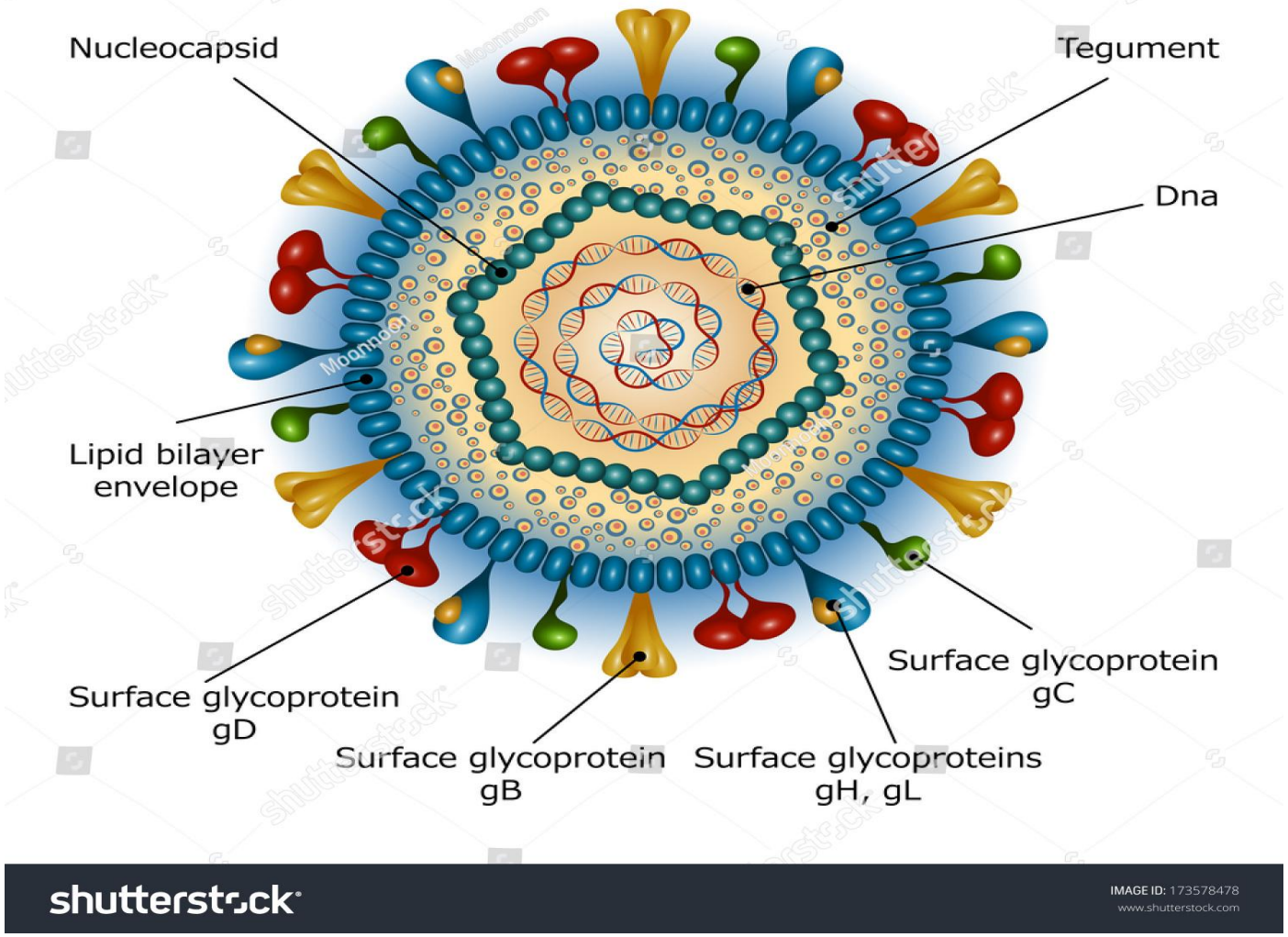
-Linear Double- Standard DNA

-Nucleotides Surrounded By Capsids & Capsid Composed Of Capsomeres’

-162 Capsomers

Herpes Simplex Virus

Baltimore Group I (dsDNA)



2) ENVELOPE

-Lipoprotein.

- Nucleocapsid Surrounded by Envelope.

(i) Lipid Part - Nuclear Membrane

. (ii) Protein Part – Bind to Host Cell Receptor Help in Viral Entry.
. -Glycoprotein Spikes help in Entry).

3) TEGUMENT –

-Between Capsid & Envelope.

. -Asymmetric Structure Present.

-Replication Takes Place In Host Cell Nucleus Like Other Double Standard
. DNA..

- - Only Difference In Linear Double Standard DNA Becomes Circular Inside Host Cell & Replicate Be Rolling Circle Machine.

- **CLASSIFICATION OF HUMAN HERPERVIRUS :-**
- 1 Subfamily Of HSV :--
- i) Alpha
  ii) Beta
  iii) Gamma

Official Name	Species(8)	Common Name
1) Alpha	Genus HSV-1 Simlex HSV -2 Vericella HHV -3	Herpes Simples Virus-1 Herpes Simplex Virus-2 Varicella Zoster Virus
2) Beta	Cytomegalo CMV-5 Roselo -6 -7	Cytomegalo Virus Human Herpes Virus-6 Human herpes Virus -7
3) Gamma	Lymphocrypto -4 Rhadinio -8 (HHV -8)	Epstein Barr Virus Kaposi's Sarcoma Associated Herpes virus

EPIDEMIOLOGY –

- HSV -Associated diseases are among the most wide-spread Infections Affecting Nearly 60-95% Of Human Adults.
- Highest Incidence Of HSV-1 Infection Occur Among Children 6 Months to 3 years Of Age.
- Primary Infection By HSV -2 IS More Common In Young Adults.
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- **PATHOGENESIS:-**
- 1 Primary Infection
- 2 Latent Infection
- 3 Recurrent Infection
- **SOURCES :-**
- Saliva
- Skin Lesion
- Respiratory Secretion From Infected Case Or Asymptomatic

- **TRANSMISSION :-**
- HSV-1 – Oropharyngeal –

  Direct Contact,

  Inhalation(Lesion on Face)
- HSV -2 Sexual Contact -

  From Mother to Fetus (Lesion in Genital Area)
- **SITE OF INFECTION :-**

HSV-1 Infect and Replicate At Local Site Or Epithelial Cell Of Skin Or Mucous Membrane And Produce Lesion.

- **More Common :-**

  HSV-1 Lesion –

  Area Above Waist (Around Mouth)

  HSV-2 Lesion –

  Area Below Waist (Genital)

  After Local Multiplication, Cell to Cell Spread Occur, in Lymph Node To.

Spread via NERVE, VIRUS secondary infect nervous tissue

Virus Invade (आक्रमण)

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Local Nerve Ending

↓ transported by

Retrograde axonal flow

↓ to (~~Self direction~~)

dorsal root ganglia

↓

Become Latent

Primary infection are mild & asymptomatic.

- **LANTENT INFECTION :-**
- HSV-1 – Undergoes Latent In Trigeminal Ganglia.
- HSV-2 – Undergoes Latent to Lumbar & Sacral Ganglia.
- Latent Infection Also Known As non Replication Stage
- HSV Does Not Replicate in Latent Stage Except Small RNA.
- Micro RNA Coded by Latency Associated Viral Gene.
- Micro RNA Helps in Maintain the Latent Infection & Prevent Cell Death.

- **RECURRENT INFECTION :-**

- Reactivation Of Latent Virus Can Occur/Induced By Various Stimuli Like Fever, Axonal Injury & Emotional Stress, UV Light.

-It Migrate Down The Neuron Via Axonal Spread-Virus Goes Back to Peripheral Site Replicate In Skin Or Mucoso Lead To Secondary Lesion.

- Recurrent Infection are Less Serve Because Presence Of pre-Existing Host- Immunity.

Clinical features :- Lesion by HSV-1

1) mucosal : - Common site is buccal mucosa.

Acute gingivostomatitis in children Characterized by fever irritability & vesicular lesion in mouth, which ulcerate become secondary infection.-Lesion heal – 2-3 weeks.

2) CUTANEOUS :-

- Lesions are fever blister and herpes fibrils.
- Caused by reactivation of virus by fever.
- Skin lesion are thin walled umbilicate vesicle.
- Vesicle contain serous fluid filled virus particle & cell debris which breaks down to form ulcers.

- **Seen on face** – On cheeks
- On chin
- Around mouth
- On forehead
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- **Herpetic Whitlow** –
- Occupational disease in dentist, doctor, nurse.
- Occur as pustules lesion of finger.

- **3) Ophthalmic** –

1) Acute Keratoconjunctivitis

- corneal ulcer

- lesion of conjunctival epithelium

2) Follicular conjunctivitis- with vercicle formation on eyelids.

- scarring & blindness – on recurrences

- **4) CNS :-**
- i) Aseptic meningitis & Encephalitis in babies
- ii) Acute necrotizing encephalomyelitis
- High mortality rate In children & adults

- **5) HERPES LABIALIS :-** (fever blisters & cold sore)
- **Lesion by HSV-2 Genital herpes –**
- Characterized by vesiculo ulcerative lesions on penis & urethas in male.
- Cerivx, uvla, vagina & perineum in female.
- Lesion are painful & associated with fever malaise, inguinal lymphadenopathy.
- Infection may be asymptomatic in female.

- **NEONATAL HERPES:-**
- Infection acquired in utero during birth or after birth.
- Infection may be localized-skin mouth or eyes.
- Generalized – involve multiple organs.
- In CNS causes encephalitis

- **LABORATORY DIAGNOSIS :-**
- **1) Specimen :-**
- vesicle fluid, skin swab, corneal scaping, throat swab CSH, brain biopsy.

- **2)Microscopic Examination:-**
- -Smear prepared from scraping obtained from base of vesicle & stained with toluidine blue (1%) if smear is positive –
- The multinucleated giant cell with faceted nuclei.

- **GIEMSA STAIN:-**
- Used to demonstrate intranuclear inclusion bodies in smear.
- **Electron Microscopy:-**
- Used to demonstrate virus particle in smear.
- **Flurucent Antibodies test:-** for Biopsy material.
- Encephalitis diagnosis

- **VIRUS ISOLATION:-**
- Tissue culture is method of choice.
- The specimen ixs inoculated in tissue culture.
- The appearance of typical CPE in cell culture in 2 or 3 day indicate presence of HSV.
- Identification of agent is established by using neutralization test or immune fluorescence test with specific anti sera.
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- **SERERLOGICAL TEST:-**
- Useful in diagnosis of primary infection only.
- Antibody develop in 4-7 days after infection and reach peak in 2-4 weeks.
- Antibody measured by – Neutralization test
- CFT – Compliment Fixation test
- ELIHSA –
- IFT – Immunofluorescence test


Thank
you