Acta Scientific Veterinary Sciences (ISSN: 2582-3183)

Volume 5 Issue 1 January 2023

Conceptual Paper

Herpes Virus: Immune System Mediated Latency or Lysis of Host Cell

Sheikh Uzma Farooq[1], Aditya Sharma[2]*, Sumaiya Bashir Khanday[3], Parth Sharma[4] and Janvi[4]

[1]Department of Veterinary Pharmacology and Toxicology, Khalsa College of Veterinary and Animal Sciences, Amritsar, India

[2]Department of Veterinary Pathology, Khalsa College of Veterinary and Animal Sciences, Amritsar, India

[3]Department of Veterinary Physiology and Biochemistry, Khalsa College of Veterinary and Animals Sciences, Amritsar, India

[4]3rd Professional Year Student, KCVAS, Amritsar, India

***Corresponding Author:** Aditya Sharma, Department of Veterinary Pathology, Khalsa College of Veterinary and Animal Sciences, Amritsar, India.

DOI: 10.31080/ASVS.2022.05.0586

Received: November 07, 2022

Published: December 09, 2022

Abstract

Herpes virus disease is caused by herpes virinae family. It is a dsDNA virus and the family Herpesviridae can be categorized into three major subfamilies: alpha herpesvirinae, beta herpesvirinae and gamma herpesviridae. The herpes virus has unique feature: It replicates inside the host cell and produces pathogenic virions which either manifest the disease or set up latency in the host cell. The severity of pathogenesis is determined by host immune system. In immunocompromised patients, the virus reactivates again and produces a potent disease. It is a harmonious pathogen but can produce grave prognosis depending upon the host cell response. The site of latency, host range and time taken for replication is dependent upon type of herpes virus. Transmission is mainly through direct contact, no vertical transmission has been reported. Majority of herpes virus infections go unnoticed as they are asymptomatic.

Keywords: Herpes Virus; Alpha Herpes Virus; Beta Herpes Virus; Gamma Herpes Virus; Latency; Lysis; Immunodeficiency

Etiology

Herpes virus is a dsDNA virus. It is enveloped virus having a linear genome and is about 125-290 kilo base pair in size. It replicates in the nucleus of host cell. It consists of three types of genes: alpha, beta and gamma, which transcript and translate to produce alpha, beta, gamma proteins respectively. The replication is unique as it produces a high ratio of non-infectious to infectious virions. Another unique feature is establishment of latency in host specific cell. Herpes simplex virus type 1, 2 and Zoster virus all establish latency primarily in the dorsal root ganglia of host while Epstein-Barr virus can set up latency within salivary glands and B lymphocytes of host. The latent virus can be reactivated any time due to many factors like stress, immunodeficiency, exposure to UV light and has capability to produce potent disease [1].

The family Herpesviridae can be categorized into three subfamilies: Alpha herpesvirinae, Beta herpesvirinae and Gamma herpesvirinae. Alpha herpes virus has extremely brief reproductive cycle (hours). It is known to cause infection in humans and have potential to replicate in wide variety of host tissue. They harbor the host cell and end up killing the host cell. The latency is primarily in the sensory ganglia. The subfamily consists of Herpes simplex virus type 1, 2 and Zoster virus. They have a vast host range. Beta herpes virus is known to cause infection in rodents and elephants. They have a highly narrow host range and their replication is slow, infection progresses slowly in host cell and cell death is comparatively slow as compared to alpha herpes virus. A unique characteristic of virus is ability to form syncytial cells. They set up latency in secretory glands, kidney and REC system. Gamma herpes virus is known to cause infection in humans, it has limited host range. They replicate in lymphoblast cell, it has lytic properties. This virus becomes latent in lymphocytes. Epstein Barr virus is the subfamily of gamma herpes virus. Other sub families are Herpes virus type 6 and type 7. Kaposi sarcoma virus is categorized in this family and is genetically similar to Epstein Barr virus [2].

Transmission

Wild mice serve as natural reservoir host for infection. Herpes virus of rodents is used to study human herpes virus infections. Herpes viruses of humans don't replicate in rodents or if they do replicate, they fail to produce any sign or symptom. Murine cytomegalovirus (MCMV) infects mice and serves as useful tool for research and experimental study of humans CMV disease. The herpes virus has capability to remain latent in the previously infected host. They usually adopt two modes of lifecycle. One is latent cycle and other one is lytic cycle. Herpes virus usually after a productive infection set up latency and factors like stress, immunodeficiency, UV light, micro environment, cell autonomous factors and viral elements determine the reactive episodes of the virus causing potent disease.

Herpes Simplex virus: It is transmitted through direct contact. Herpes simplex virus type 1 is transmitted through saliva (Kissing) while as Herpes virus type 2 is known to be transmitted by sexual contact. Herpes virus type 2 is also reported to spread to newborn babies in intensive care units (Nosocomial infection) [3]. Varicella-Zoster virus: It is transmitted via aerosol route. Cytomegalovirus: It is prevalent virus which is spread by intimate contact. It is reported to be excreted in urine and saliva for prolonged period of time. Vertical transmission has not been reported but mother to child transmission is via breast milk secretion, saliva (kissing). Epstein Barr virus and Herpes virus type 6, type 7 is transmitted horizontally. Type B virus or Kaposi's sarcoma herpes virus harbors in rhesus monkeys. The environmental stress leads to virus reactivation and determine the titre of virus load in host body. The virus is excreted in saliva and can spread to humans through improper handling by personnel.

Pathogenecity

Rodent infected with HSV-1 may lead to development of virus induced CNS demyelination or encephalitis which can lead to death of rodent. Primary infection of mucosa lead to local multiplication of virus, sensory nerve endings infection, and spread of virus through retrograde axonal transport to ganglia of peripheral nervous system and as a result proliferative infection of neurons occurs.

Herpes simplex virus: The virus replicates in epithelial cells initially. The proliferation is characterized by cell lysis which leads to inflammation and vesicle formations. It spreads through lymph nodes and causes viremia. It then, ascends to dorsal ganglia and set up latency period. During any immunocompromised condition, latent virus reactivates again and descends back to epithelial cells. They start proliferation and manifest a potent infections disease. The severity of disease depends on the immunodeficiency of host. Varicella- Zoster virus replicates in nasopharyngeal cells. It proliferates slowly and causes cell lysis. It is known to produces vesicular rash. The virus also is reported to set up latency in dorsal ganglia. Cytomegalovirus harbors in salivary glands and also has affinity for kidney cells. They proliferate slowly and produce multi nucleated intranuclear and intracytoplasmic inclusion bodies. The virus is excreted via saliva and urine. Epstein Barr virus replicate in oropharyngeal cells and also lymphocytes. The presence of multinucleated cells in parotid glands and lymphatic tissue has been reported. Kaposi sarcoma virus has high affinity for neuronal cells, They proliferate slowly and cause encephalitis [4].

Gross lesions

The herpes rash appears like clusters of small, fluid filled blisters. The sore appear red, white or yellow in colour filled with a clear liquid. They can be anywhere on skin but appear in genital area in case of genital herpes and around mouth, conjuctiva and cornea in case of oral herpes virus infection.

Microscopic lesions

Intraepidermal blister and acantholysis with solitary keratinocytes within the blister cavity. Keratinocytes will show nuclear changes like margination of nuclear chromatin. Cowdry type inclusions are seen. Majorly neutrophils and lymphocytes are seen with some eosinophil cells also.

Diagnosis

Majority of herpes virus infections go unnoticed as they are asymptomatic. Therefore, it is difficult to diagnose it. A clinical diagnosis of genital herpes infection should be checked by laboratory testing. It can be done by use of direct tests for viral isolation, the detection of antigen or the most recent method which is by the use of molecular diagnostic techniques. Testing for serotypes should be done because of the different anticipating and counseling inference. The confirmatory diagnosis of genital herpes depends on showing the presence of HSV in the genital region, either by virus isolation or detection of antigen. Serological testing is sometimes

beneficial in symptomatic patients when direct methods have shown negative results or in asymptomatic patients to determine past or present infection.

Specimens taken from vesicular lesions within the first three days after their appearance are favoured, but other lesion material from older lesions or swabs of genital secretions should be taken if suspicion of HSV infection is high. Once crusting and healing have started, the recovery rate of HSV falls sharply. The use of alcohol or iodophors to cleanse the lesions may lead to inactivation of the virus and thereby, should be avoided. Calcium alginate swabs are toxic to HSV and therefore should be avoided.

For collection of sample, a sterile needle with plastic shaft should be carefully inserted at the site of lesion so that epithelial cells could be collected for further investigation. At a time, more than one lesion should be sampled. In the same way for ulcerative lesions, a swab should be firmly twisted in the base of one or more lesions. The swab(s) should be immediately inserted into viral transport medium such as M5 transport medium. The swab's shaft should be broken before the cap is changed so that the shaft will not interfere with closure and leakage will not occur. The specimen should be held at 4°C and transported to the laboratory for further processing within 48 h. During transportation, the specimen should be protected from heat by including a cold pack or ice cubes in a sealable plastic bag. Virus specimen collection swabs with matching transport tubes are commercially available and used.

Conclusion

Herpes virus is a cytolytic virus. The alpha, beta or gamma herpes virus has rapid, slow or progressive reproductive cycle and depending upon type of herpes virus, cell death is brief or prolonged. The virus has two modes of lifecycle. One is cell lysis and another is latency. The site of establishing latency also depends upon type of herpes virus infection. The latent virus can be reactivated any time due to immunodeficiency. The latent virus descends back to predilection site, replicates and produces potent pathogenesis.

Bibliography

1. Barton ES., *et al*. "Herpesvirus latency confers symbiotic protection from bacterial infection". *Nature* 447 (2007): 326-329.

2. Bennett JE and Dolin R (eds). "Principles and Practice of Infectious Diseases". 4th Edition, Churchill Livingstone, New York, (1995).

3. Straus S. "Introduction to herpesviridae". In Mandell GL, Bennett JE, Dolin R (eds): Principles and Practice of Infectious Diseases, 4th Ed., Churchill Livingstone, New York, (1995): 1330.

4. Whitley RJ and Schlitt M. "Encephalitis caused by herpesviruses, including B virus". In Scheld WM, Whitley RJ, Durack DT (eds). Infections of the Central Nervous System. Raven Press, New York. (1991): 41.