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Review Article

Deciphering the Spectrum of Herpes Virus- An Overview

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ABSTRACT

Infections caused by herpesviruses are severe particularly in immuno-susceptible individuals. Initial infection is followed by latency phase in ganglionic neurons. It is the period in which virus particles are not produced and no neuronal damage takes place. Once the virus is activated it results in viral replication which produces shingles in tissues that innervate neurons and causes consistent radicular pain called postherpatic neuralgia. Other complications include cranial nerve palsies, stroke, meningitis, retinitis, ulcers and pancreatitis. Herpes viruses are abundantly associated with many other several pathological conditions associated with both humans and animals. Thus far, numerous approaches used to avert these infections have been ineffective because these viruses can circumvent different immune mechanisms. For this reason, better comprehension is needed to understand their complicated lifecycle. Currently, mass spectrometry has emerged as a crucial method that has permitted primitive findings in virology. In this review we discuss the prospective prosecution of high-through proteomic techniques to study proteomic streaming along with the post translational modifications that help in improving the molecular aspect of viral infection. It is hope in future that synergistic efforts will facilitate in the emergence of new procedure to fight against these viruses.

Keywords: herpesvirus, postherpatic neuralgia, latency, ganglionic neurons

Introduction

Family Herpesviridae is diverged into three subfamilies namely, the alpha, beta and gamma-herpesvirinae.[1] These subfamilies are colloquially known as alpha-, beta-, and gamma-herpesviruses. Alpha herpesvirinae is the subfamily of Herpesviridae and it is responsible for causing virus action in humans. It includes two types of viruses namely herpes simplex viruses' types 1 and 2 (HSV-1 and HSV-2) and varicella-zoster virus (VZV). They are known for causing skin vesicles but clinical manifestations of areas other than the cutaneous area is of more engrossed. [2] Both viruses are normally present during childhood and eventually develop a lifelong indiscernible infection in sensory neurons. The reactivation and transport of virus to mucosae causes the epithelial cells infection and propagate in the individuals. HSV-1 causes the genital and oral herpes and is responsible for viral encephalitis and ocular disease. Varicella-zoster cause chickenpox and herpes zoster or shingles upon reactivation from dormant state and go along with chronic pain termed as post-herpetic neuralgia. Although vaccines have been made to avert herpes zoster in adults and varicella in children. HZ vaccines are cost effective, provides partial protection along with painful side effects. Currently no vaccine is available to prevent HSV. Understanding the biological mechanisms displayed by these two viruses could help in developing the targets to establish the antiviral therapies. Gene expression of herpesvirus is regulated by both viral and host proteins. The size of HSV-1 and VZV genome is 152 and 125 kb. HSV and VZV comprise of 90 and 71 genes which codes for more than 84 and 71 proteins which are expressed timely and classified into three namely, immediate early, early and late transcripts. The timely regulated protein expression of HSV-1 and VZV establish the interactivity that is linking viral and host proteins in the course of replication.[2] Proteomics is a powerful strategy in order to investigate virus structure and comprehend process that how virus particles reproduce and disseminate by means of host cellular machinery. Commonly, cellular feedback due to the virus infection is examine by investigating the relative abundance of cellular proteins, post translational modifications and interaction between the viral and host proteins. Currently, mass spectrometry

has emerged as avital tool which has permitted the preliminary inventions in virology. [1] After the first infection caused by varicella, the VZV enters a dormant stage in sensory ganglia and cranial nerves. Cellular immunity against VZV turn down as age progresses and immunosuppression occurs. Eventually, this results in reactivation and dissemination of virus to skin through sensory neurons, thus initiating a distinct preliminary irritation accompany with a rash. VZV infection is a possibility in one out of every five individuals worldwide. According to statistics, one million instances of herpes zoster are reported to arise per annum in the United States, and roughly one-third of infected individuals will erupt with HZ throughout their lifetime. Onset of herpes zoster can happen at any stage of life and is known to be less complicated in young adults and children. High rate of morbidity and mortality is seen in elders and in people have low immunity. Complications of HZ include the vasculitis, post herpetic neuralgia and myocardial infarction. On account of the severity of clinical complications, there is an immediate need for the diagnosis of the biomarkers which surely will help in elucidating the mechanism governing the clinical picture.[3]

Structure of Herpes Virus

All herpes viruses including alpha, beta and gamma are comprised of double stranded DNA which is surrounded by an icosahedral capsid made up of proteins. Around the capsid there is an amorphous covering of proteins termed as tegument which is the characteristic feature of herpesviruses. A lipid envelops made up of host derived lipids and viral glycoproteins enclosed tegument. Few host proteins like annexins and tubulins along with mRNAs are integrated into virions, although they are not crucial for the life cycle of a virus but had distinct function if present.[4] (Fig. 1A). In Herpesviridae family, tegument protein forms a link between nucleocapsid and lipid envelop. The tegument is further segmented into several outer and inner teguments depending on its connection with envelop proteins or the capsid. Tegument outer layer is amorphous, depicts asymmetrical ordering of glycoproteins. On contrary, inner tegument contains icosahedral symmetry due to its immediate interaction with icosahedral capsid of virus. Tegument is the important constituent of herpes virus structure and reflects diverse roles that are crucial for virus life cycle. Tegument is formed by protein-protein interaction among capsid proteins, glycoproteins and tegument. There are about 17 to 38 tegument proteins known to present in Herpesviridae.[5, 6]

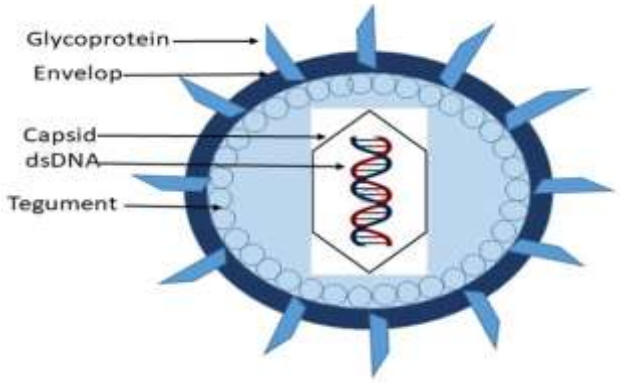

Figure 1. Structure of Herpes Simplex Virus

HSV-1

The characteristic feature of HSV-1 is that this virus is double stranded, enveloped and neurotropic. The genome size of HSV-1 is 152 kb which encodes 74 or more different genes.[7] Due to the widespread nature of this virus it can be transmitted by means of direct contact among people. According to WHO an estimated of 3.7 billion people that age under 50 are infected worldwide with HSV-1. Once enter into the human body by means of skin or mucosa, HSV-1 takes its route to sensory neurons to the axons followed to the trigeminal ganglion and establishes a latent infection. [8]

Upon stimulation, reactivation of the latent virus escort the symptomatic or asymptomatic infections, causing blisters and cold sores.[9] HSV-1 can also enter in CNS, often causing fatal neurological disease like herpes simplex encephalitis. However, strong correlation has been found between HSV-1 infection and Alzheimer's disease (AD).[10] Till date no antiviral therapy is available that could eradicate HSV-1 infection because virus go through dormant infection and thus elude drug interaction. Consequently, there is a need of more underlying research in order to reveal interactions of HSV-1 so that novel

therapeutic procedures can be developed against viral infections. [11,12.]

Varicella zoster virus (VZV)

Varicella zoster virus (VZV) is also known as herpesvirus 3, with double stranded DNA genome. VZV naturally infects humans only, and it attacks particularly epithelial cells, ganglia and T lymphocytes. Origin of varicella or chickenpox is the result of primary infection afterwards virus remains inactive in ganglionic neurons. With a decline in cell mediated immunity, person becomes immunocompromised, VZV becomes active again and causes zoster or shingles. Other deleterious neurological and ocular disease include meningoencephalitis, vasculopathy and retinopathy. Other disorders include pancreatitis, hepatitis and ulcers. [13, 14]

Pathogenicity of Herpes Simplex Virus infection

Lytic Infection: Subsequently, after the transmission of virus to vulnerable host, VZV escalate to oral pharynx and attack on T cells which goes into the blood stream and propagate virus to other body organs. Innate immunity becomes active to control the infection first. [15, 16] VZV can modify various T cell so that skin transport can be assisted. [17] DNA of VZV is identified in T cells within 10 days before the appearance of a rash and remains for a week after that. At first, innate immunity impedes the viral replication in skin, which imparts time for the development of acquired immunity. [15] In due course, virus control the cutaneous innate immune response, resulting in viral replication in skin, forming the typical rash called varicella. High concentration of non-cellular VZV emerge in skin that later on spread VZV to others. In this lytic infection cycle, VZV expresses variety of genes. As explained for other herpesviruses, gene expression takes place in sequence of events, starting from immediate early genes, early genes then late genes. Latency stage is followed with no gene expression. Reactivation, results in the expression of all genes, therefore starting the lytic cycle again. [14]

Latency: VZV is dormant in neurons of dorsal root ganglia, cranial nerve and enteric ganglia. Because sites of zoster particularly comprehend to those that include varicella. VZV enters in epidermal nerve endings and propagate to retrograde axonal transport and make its way to ganglia cell bodies. [18] DNA of dormant VZV is circular and do not replicate, although histones associated posttranslational modifications shows that there is close association between euchromatin and immediate early genes, thus facilitate the gene expression, but no association is found with late VZV genes. [19] Immediate early proteins 63 (IE63) interrelate with anti-silencing function protein 1 (ASF1) and up regulate the binding of histone. The function of ASF1 is to deposit and clear histones from DNA while transcription is going. IE63 when interact with ASF1 it facilitates the transcription of viral genes, a probability that needs verification.[20]

Reactivation of VZV causes ganglia to become hemorrhagic and necrotic. VZV proteins are abundantly found in neuronal as well as non-neuronal cells, whereas amplification of MHC proteins (class I and II) along with invasion of CD4+ and CD8+T cells is the hallmark of ganglionitis. [21,22] Late viral proteins like envelope glycoprotein E, exhibit that lytic cycle of VZV has started. All proteins of latency stage are cytoplasmic but in the course of infection they become nuclear. [23]

Prevalence of Herpes Virus

Prevalence and intensity of HZ markedly increase after 50 years of age. [24-26] HZ is painful with erythematous and maculopapular rash that last at 2 to 4 weeks. However, antiviral therapy considered as an efficacious treatment for HZ to lessen duration and illness.[27] Postherpetic neuralgia is the utmost complication of accompanying herpes zoster with constant pain persistent for 3 months after shingles outbreak and 20% of HZ patients experienced this pain specifically older people. [28] Although, the reason for the reactivation of VZV is not known but immunosuppression and older age are well documented but the epidemiology cannot be explained on these grounds only. [29] Other risk factors include ethnicity, female gender[30], family history of HZ[31], inflammatory bowel disease [1], chronic obstructive pulmonary disorder and diabetes[32]. However, these findings are in disagreement. Further, literature of HZ mostly comes from electronic health records which may

miscalculate the prevalence of HZ among population [33, 34]. Evidence collected form population surveys is very limited. Additionally, other risk factors include lifestyle and in appropriate health electronic records. [35]

Clinical manifestations

The degree of pain accompanying HZ is characterized by burning, aching and stabbing. People with herpes zoster also encounter sensitivity upon touch, ache elicit by insignificant stimuli and insufferable tingling. Postherpetic neuralgia (PHN) is condition in which pain persist for at least 3 months after the inception of rash. Pain with PHN disturbs daily life. Individual with PHN experienced social withdrawal, depression and decrease life quality. Other clinical manifestation includes cardiovascular events, palsy and neurological sequelae. Serious cases may often require hospitalization.[36] Zoster paresis causes weakness of arm and diaphragm; leg weakness or urinary retention occur following lumbar or sacral zoster. Acute infection of VZV may cause meningitis. Reactivation of VZV in cerebral arties results in vasculopathy that leads to the ischemic stroke. VZV is also the leading cause of stromal keratitis manifesting corneal anesthesia and retinal necrosis.[37]

Transmission

Transmission of VZV essentially occurs when airborne droplets nuclei are inhaled, dispersed from vascular skin lesion or respiratory tract. Virus dispersion has been reported with both primary varicella and HZ and cases with secondary varicella have also been documented in patient having localized HZ. [38] VZV may persevere in blood and saliva after HZ and vulnerability to these fluids may account for several possible routes of transmission. Varicella infection is common when person comes in close contact with patient whereas pool do not have a significant standing in transmission.[39] Transmission prevention in both community as well as in healthcare setting relies on universal vaccination, along with proper isolation of infected patients. Recent guidelines suggest standard provision for hospitalized patients suffering from localized HZ. Patients with HZ in community are usually allowed to join group settings like work place or school in case if lesions have been covered.[40]

Host pathogen interaction

Elucidating the protein-protein interactions (PPIs) between host and the pathogen can decipher essential biological process and also reveal mechanisms that propagating infectious diseases and thus facilitating the therapeutic procedures.[41] Individual viral proteins such as glycoproteins necessitate the entrance of HSV-1 within the host cell, neurovirulence factor, viral E3 ubiquitin ligase, single stranded DNA-binding protein, major viral transcription factor and so on. In depth availability of data will help in understanding the meshwork of PPI build between human and HSV-1.[42] Variety of methods are available for the prediction of host-pathogen interaction. Among them intra-species PPI traditional methods include interolog mapping, domain motif interaction (DMI)-based method and domain-domain inter action (DDI)-based method. [43]

Diagnosis and prevention

One of the most common tool used for the laboratory diagnosis of VZV is PCR in which material is taken from skin, swabs, scabs cerebrospinal fluid and saliva. VZV antigens are detected by means of direct immunofluorescence, however this procedure is less accurate than PCR.[44] In countries where vaccines are not available for varicella, procedure of passive immunization is used which impart temporary preservation for few weeks. Antiviral remedy has been employed against both varicella and zoster in immune sensitive individuals but did not proved as significant.[45] But today, vaccines for varicella and zoster have drastically upgraded the life quality in United States. Children do not fail to attend their school on account of suffering from varicella and very few employed parents have to stay at home to look after their unwell children. Zoster vaccines have become important with increasing lifespan now. Additionally, basic research has evolved the enormous progress in managing VZV infections, thereby has improvised the standard of life. [45]

Mass spectrometry-based proteomics

Notwithstanding, since ages 2D electrophoresis is only the technique that has been used to examine the proteins in HSV-1 infected cells. Although this technique offers few pitfalls like multiple spots represent the multiple proteins and poor resolution of protein spots. On the other hand, emerging advances in high throughput techniques such as MS-based proteomics have facilitate in protein analysis that are interacting with HSV-1 proteomes. Shotgun analysis on another way can help in elucidating the phosphorylation sites in HSV-1 thus benefiting in revealing the biological phenomena involved in the activation or deactivation of different host signaling pathways in the course of infection. Further, combinatorial efficacy of immunoaffinity chromatography and quantitative mass spectrometry will be valuable tool in characterizing the glycoproteome of HSV-1 infected cells. Eventually, execution of ion mobility and native MS in future will make provision regarding viral-proteins, conformation, protein complex structures, binding affinities topology of protein complex of virus thus expanding knowledge about structural biology of HSV-1.[46]

Post-translational modifications

Numerous structural proteins are phosphorylated but the amplest post-translational modification seen in the structural proteins of herpesvirus is glycosylation. Glycosylation results in glycoprotein aggregation and form large complexes that cannot run through the pores of gel and therefore cannot be examine on SDS-PAGE. Further, glycosylation reduces the effect of trypsin digestion due to steric hindrance and thus impede protein elucidation. Therefore, identification of glycosylated protein is challenging through SDS and mass spectrometry.[47]

Proteogenomics

Proteogenomics is the use of fragmentation

spectra that is required to ameliorate the gene annotation. Consequently, it leads to the improvement of data quality, particularly confirms the open reading frame (ORF) expression. Homologies with distinct sequences sometime are scant for varying groups such as alloherpesviruses. In this situation, few ORFs are considered as recognized coding sequences because it is difficult to find homology with any known protein while genome sequencing. Therefore, it is necessary to identify those proteins that are coded by uncertain sequences in order to understand the virus lifecycle. [48]

Therefore, the objective of this systematic review and meta-analysis is to consolidate available data on the prevalence of HBV infection among blood donors in Pakistan between 1991 and 2025, analyze temporal trends, evaluate screening practices, and provide recommendations to strengthen national blood safety strategies.

Conclusion

Human neurotropic DNA viruses that can enter a state of latency in the nucleus include varicella-zoster virus (VZV) and herpes simplex viruses 1 and 2 (HSV-1 and HSV-2). These are the most pervasive viruses linked with various pathologies in humans and animals. Till date, several strategies have been employed to impede these viral infections Proteomic perusal of structural proteins will help in comprehending the lifecycle of herpesvirus. PTMs analysis is also valuable for targeting cell and infectivity of the particles. Clinical and translational research are increasingly incorporating mass spectrometry (MS)-based proteomics. Proteomic biomarker discovery procedure today routinely process tens to hundreds or even thousands of patient samples in a high-throughput fashion. Liquid chromatography–mass spectrometry (LC/MS)–based proteomics has been effectively used to characterize the proteomes of a variety of human bio fluid specimens.

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