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Editorial

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Viral Encephalitis: Bangladesh Perspective

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The spectrum of infections of CNS is broad and encompassing bacterial/aseptic meningitis and encephalitis[1]. Viral Infections of the central nervous system (CNS) are potentially life threatening for the patients if these infections are not diagnosed and treated early. The initial clinical presentations of many CNS infections are nonspecific, making a definitive etiologic diagnosis challenging. Viral CNS infections are usually self-limiting, but in children less than 1 month old and in immune-compromised patients, the infection can be more serious. Some infected patients may develop serious squeal such as longterm cognitive impairment, short term memory loss, psychiatric morbidity and chronic fatigue syndrome[2].

Many viral pathogens both new and emerging are creating heavy burden on health care system in Bangladesh. Common conventional viruses that may cause acute, sub-acute and chronic encephalitis are Poliomyelitis, PML, Herpes simplex, Varicellazoster, Rubella, Measles, Cytomegalovirus, Rabies, SSPE, Echovirus, HIV, Coxsackie A & B, HTLV, Arbo-Virus, Japanese Encephalitis, West Nile and Dengue etc. The JEV, Rabies, Enteroviruses, HSV, Measles are presented as isolated acute viral encephalitis. Both brain and spinal cord (Encephalomyelitis) may be infected by Measles, VZV, HIV. Acute transverse myelitis may be caused by Poliovirus, Enteroviruses, and Dengue. Sub acute/ chronic viral infections of CNS includes Subacute Sclerosing Pan-encephalitis (SSPE), Measles.

The target cells with specific viral tropism are Neuron, Astrocytes, Oligodendrocytes, Microglia and Endothelialcells. Neurons are target of HSV, Rabies, SSPE, JE, Poliomyelitis, and Cytomegalovirus. The Astrocytes are attacked by SSPE, HSV(Rarely), PML(Rarely), Cytomegalovirus. The JEV has the affinity for oligodendroglial cells. In many viral encephalitis intracellular viral inclusions are diagnostic. Specific type of Intranuclearinclusions are found in Herpes Simplex, Varicella zoster, SSPE and PML induced encephalitis. Diagnostic intracytoplasmic inclusions are found in Japanese encephalitis and Rabies. HIV (AIDS), HTLV1, Poliomyelitis and Cytomegalovirus have no cellular inclusion.

Many of the encephalitis are characterized by long incubation period. These have, no inflammation, minimalimmune response, no RNA or DNA, no identifiable viral particle and no inclusions but these are transmissible. These unconventional virusesleads to Protein Misfoldingencephalopathy and Transmissible Spongiform Encephalopathies. The causative agents are abnormal Prion Protein (PrPsc). PrPscare fibrillar structures present in presynaptic terminals but their function is not clear. Probably these proteins play a role in synaptic transmission. PrPscproteins can be identified by immunohistochemistry. In the brain these neuropils are accumulated around vacuoles. Plaques of PrPsc arealso found in grey matterand occasionally in the subcortical white matter. The Prion disease like Kuru- Human, CreutzfeldtJakob Disease-Human etc. are rareencephalopathies. A few new Variant of CJD (vCJD)-Human, PrP-cerebral amyloid angiopathy-Human are observed as abnormal Prion protein encephalopathy.

There have been significant developments in the diagnostic methods for rapid, accurate, and early identification of the causative viruses; no major breakthroughs in the treatment of viral meningitis have been reported. Efforts aimed at early identification of the causative viruses are critical for optimal clinical management of patients with viral encephalitis. Immune responses to neurotropic viruses can promote viral clearance or latency, but sometimes give rise to pathology and disease. HIV persists in CNS myeloid cells (Macrophages and microglia), giving rise to chronic innate and adaptive immune responses. This pro-inflammatory milieu can eventually cause neuronal damage and dementia. By contrast, herpes simplex virus latency in sensory ganglion neurons is maintained without injury, in part by innate cytokines and virus-specific T cells[3]. Intrauterine CMV, Rubella, HSV-2 encephalitis may cause Hydrocephalus or Microcephaly. Though etiology is not known but Reye's syndrome and Guillain-Barre Syndrome are presumed virus related disorder.

Viral infections of the Central Nervous System often result in a spectrum of movement disorders ranging from slowness and rigidity to hyperkinetic movements such as chorea, ballism, dystonia, and myoclonus[4]. The basal ganglia are especially susceptible to some viruses, because of their intrinsic neurotropism, a predilection of opportunistic infections for the deep gray matter of the brain, and possibly the mounting of an autoimmune response against basal ganglia antigens. Hyperkinetic movement disorders are associated with Prion diseases.

Several viral infections involving CNS have been reported from Bangladesh. These viral infections of the CNS in adults and children are associated with high morbidity and mortality in Bangladesh. Among these the Nipah virus, Rabies, Herpes simplex, Japanese encephalitis, Dengue and Enteroviruses are the most common viruses identified in Bangladesh. Rubella virus, Varicella zoster, Mumps and Cytomegalovirus are also frequently reported in the laboratory[5]. There have been significant developments in the last few decades in the understanding of the pathogenesis of the disease process by the application of new technologies[6]. Advancements in brain imaging and newer diagnostic modalities are providing scope for early diagnosis and management. Many of the new diagnostic modalities including PCR, Immunostain are available in Bangladesh. Further establishment of high-tech diagnostic facilities including viral genome sequencing, postmortem death review with brain cell and tissue diagnostic facilities are essential for diagnosis and research to reduce the public health burden.

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