



Neurotropic Viral Infections in Bangladesh: Burden and Challenges

Mohammad Enayet Hussain

Assistant Professor, Department of Neurology, National Institute of Neurosciences & Hospital, Dhaka, Bangladesh; Email: enayetdmc@yahoo.com

Neurotropic virus infections continue to cause major disease and economic burdens on society¹. It poses a major challenge to human health care systems due to the associated morbidity and mortality worldwide. This creates a unique problem in providing treatment to the patients involved. This is largely due to unique features of the central nervous system (CNS), with a plethora of interconnected and interdependent cell types, complex structures and functions, reduced immune surveillance and limited regeneration capacity. Infection by neurotropic viruses as well as the local induced immune responses can irreversibly disrupt the complex structural and functional architecture of the CNS, frequently leaving the patient with a poor or fatal prognosis. Besides immediate and direct effects, there are several neurological disorders often associated with autoimmune mechanisms that are assumed to be delayed virus-induced disorders: multiple sclerosis, Guillain-Barré syndrome, narcolepsy and encephalitis lethargica². Neurotropic pathogens can access the brain by various routes including retrograde axonal transport along motor and olfactory neurons, haematogenous spread across the blood-brain barrier (BBB), blood-cerebrospinal fluid barrier, meningeal-cerebrospinal fluid barrier, via direct infection of endothelial cells or via spread of infected leukocytes across the BBB into the brain parenchyma³.

Influenza virus infections are the most common viral disease in Bangladesh. Overcrowding is the main reason of this disease burden. The most common extra-respiratory complication of influenza is the development of CNS disease⁴. Influenza virus infections cause some acute CNS diseases. Acute CNS diseases include febrile seizures, acute onset brain dysfunction, meningitis, encephalitis and

encephalopathies. In addition, influenza viruses have been linked to the development of Guillain Barré syndrome, Kleine Levin syndrome and transfer myelitis⁵. Maternal influenza has been associated with schizophrenia and bipolar disorder (BD) in the offspring. Acute demyelinating encephalomyelitis (ADME) typically occurs in measles patients. Subacute sclerosing panencephalitis (SSPE) occurs on average 4–10 years following acute MV infection. Mumps virus was the leading cause of aseptic meningitis in the pre-vaccine era. Pathologically, mumps encephalitis resembles ADME and is characterised by extensive perivascular demyelination in the white matter of the cerebral and cerebellar hemispheres, basal ganglia, midbrain, pons, medulla and spinal cord with areas of demyelination associated with the perivascular infiltration of immune cells, microglia and gliosis. Outbreak of Nipah virus (NiV) has been reported in 2006 in Rajbari district of Bangladesh³. Prodromal signs of henipavirus infection are general and include fever, lethargy and headache and occur following an incubation period of 5–14 days. This rapidly progresses to neurological signs of infection including limb weakness and segmental myoclonus. Rabies is common disease in Bangladesh.

It is a neurological disease characterized by fatal encephalitis. In the neurological phase, furious rabies patients may develop paresthesia at the site of exposure, anxiety or agitation, dysautonomia (autonomic dysfunction) including inspiratory spasms, hypersalivation hydrophobia, sometimes in combination with aerophobia and fluctuating mental state. The paralytic form of rabies is rather characterized by progressive weakness and paralysis, with absence of many of the symptoms seen in the neurologic phase of furious rabies.

Paralytic rabies is often misdiagnosed as Guillain–Barré syndrome (GBS), autoimmune disease or stroke.

Chikungunya viral infection is not common in Bangladesh. However, there was an outbreak in 2008 at Rajshahi District caused by *Aedes albopictus* mosquito commonly presented with high fever, headache, maculopapular rash and painful arthralgia⁴. Furthermore central nervous system involvement can also be found by Chikungunya viral infection like brain swelling, disseminated intravascular coagulation, cerebral or cerebellar hemorrhage, scattered parenchymal petechiae, cerebellar hematoma and hematemeses⁷. In children and adults, neurological manifestations include altered levels of consciousness, cranial nerve deficits, seizures, decreased deep tendon reflexes, psychosis, hemi/paraparesis, paraplegia and involuntary movements. A subset of children of <1 year presented with hypotonia, tense fontanelle and status epilepticus⁸. Herpes simplex virus (HSV) infection is the most common cause of infectious encephalitis in humans⁹. However, in Bangladesh Uddin et al² have reported HSV-2 in a hospital based study and have found that female is more commonly affected than male in young age group though male are affected later. HSV-1 is responsible for up to 90.0% of HSV encephalitis (HSE) cases, whereas HSV-2 infection being less common and more often presenting clinically as meningitis. Clinically, HSE patients present with headache, fever and alteration of mental status such as confusion, psychosis and also alteration of consciousness from somnolence to stupor and coma. Sequelae are still found and include focal or generalized seizures, personality changes, impaired memory and/or cognition, motor deficits or aphasia. Epstein–Barr virus is the causative agent of infectious mononucleosis (IM) in naïve children and young adults, with clinical symptoms evident 4–6 weeks following infection. Less than 5 % of primary EBV infections cause CNS disease that present as meningitis, encephalitis, cerebellitis,

cranial or peripheral neuropathies and polyradiculomyelitis. To prevent these neurotropic viral diseases development of more effective intervention and antiviral treatment regimens is an urgent need.

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