REVIEW ARTICLE

Nipah Virus in Bangladesh

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Introduction

Nipah virus is a newly recognized zoonotic virus. The virus was discovered in 1999. It caused disease in animals and in humans, through contact with infectious animals. The virus is a member of the virus family *Paramyxoviridae*. Although member of this group of viruses have only caused a few focal outbreaks, the biologic property of these viruses to infect a wide range of hosts and to produce a disease causing significant mortality in humans has made this emerging viral infection a public health concern.[1] The World Health Organization has confirmed two recent human outbreaks of the deadly Nipah virus have occurred in Bangladesh. This virus had previously only been confirmed in Malaysia, where a 1999 outbreak killed 40% of those infected. WHO said that in February 2004, an outbreak of this virus spread across six Bangladesh districts and caused 17 human deaths in 23 cases – a mortality rate of 74%. A second outbreak in the Faridpur district earlier this month killed 18 of 30 cases – a 60% mortality rate.[2] Fruit bats of *Pteropid* species were identified as the natural reservoir hosts.[3,4] A retrospective investigation of two outbreaks of encephalitis in Meherpur and Naogaon, Bangladesh, which occurred in 2001 and 2003, collected serum samples from persons who were ill, their household contacts, randomly selected residents, hospital workers, and various animals were classified as laboratory confirmed or probable. Among them 13 cases were identified (4 confirmed, 9 probable) in Meherpur; 7 were in persons in two households. Patients were more likely than nonpatients to have close contact with other patients or have contact with a sick cow. In Naogaon, there were identified 12 cases (4 confirmed, 8 probable); 7 were in persons clustered in two households . Two pteropus bats had antibodies for nipah virus. Samples from hospital workers were negative for nipah virus antibodies. These outbreaks, the first since 1999, suggest that transmission may occur through close contact with other patients or from exposure to a common source. [5]

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Pathogenesis & Pathology

The pathogenesis of Nipah virus infection is associated with its ability to infect blood vessels and extravascular parenchyma in many organs, particularly central nervous system.[6] Endothelial cell damage, necrosis, and syncytial giant cell formation are seen in affected vessels. Characteristic viral inclusions are seen by light and electron microscopy. Immunohistochemistry (IHC) analysis show widespread presence of nipah virus antigens in endothelial and smooth muscle cells of blood vessels. Abundant viral antigens are also seen in various parenchymal cells, particularly neurons. Infection of endothelial cells and neurons as well as vasculitis and thrombosis seem to be critical to the pathogenesis of this new human disease.[7]

Risk Factors:

1. Close contact with infected animal eg. Pig, other domestic animal.[8]
2. Handling raw pork
3. Slaughterhouse worker[8]
4. Human induced global change including forest clearance and climate change[9]
5. Use and mis use of medical and biological technology such as contaminated equipments
6. Rural to urban migration resulting in high density periurban slums
7. Increasing long distance mobility and trade
8. Changes in personal behaviour
9. Political ignorance, denial and obduracy
10. Social disruption of war and conflict[9]

Transmission

The mode of transmission from animal to animal, and from animal to human is uncertain but appears to require close contact with contaminated tissue or body fluids from infected animals. Nipah antibodies have been detected in pigs, other domestic and wild animals.

It is unlikely that this virus is easily transmitted to man. Despite frequent contact between fruit bats and human there is no serological evidence of human infection. Pigs were the apparent source of infection among most human cases in the Malaysian outbreak of Nipah, but other sources, such as infected dogs and cats, cannot be excluded. Human to human transmission of this virus has not been reported.[1]

Clinical Feature:

The incubation period is between 4 and 18 days. In many cases the infection is mild or inapparent (subclinical). In symptomatic cases, the onset is usually with influenza like symptoms, with high fever, tachycardia, systolic hypertension and muscle pains (myalgia), sore throat. The disease may progress to inflammation of the brain (encephalitis) with drowsiness, disorientation, convulsions and coma. Autonomic disturbances and myoclonic jerks are common. 50% of clinically apparent cases die. The prognosis for the survivors are good. [1,10]

Diagnosis

1. MRI (Features found on MRI in relapsed and late onset encephalitis differed from the features in acute encephalitis in that confluent cortical involvement is the prominent finding in the former, as opposed to discrete focal lesions in the subcortical and deep white matter in the later) is a sensitive and specific diagnostic tool for evaluating Nipah encephalitis.[11]
2. MR spectroscopy depicted reduction in N-acetylaspartate-to-creatine ratio and elevation of choline-to-creatine ratios.[12]
3. Rapid immune plaque assay (Detect neutralizing antibodies)[13]
4. Indirect immunofluorescence assay using anti-Hendra specific hyperimmune mouse ascitic fluid and FITC-conjugated goat anti-mouse IgG. (Nipah virus was isolated from throat swab, urine, nasal swab)[14]
5. CSF examination [15]
6. Complement fixation and / RT-PCR assays (negative for other viral diseases like Japanese encephalitis, herpes simplex, measles and mumps viruses.
7. ELISA[15]

Treatment

No drug therapies have yet been proven to be effective in treating Nipah infection. Treatment relies on providing intensive supportive care. There is some evidence that treatment with anti viral drug, ribavirin, can reduce both the duration of feverish illness and severity of disease. However, the efficacy of this treatment in curing disease or improving survival is still uncertain.[1]

Protection of Health Care Professionals:

The risk of transmission of this virus from sick animals to humans is thought to be low and transmission from person to person has not yet been documented, even in large outbreak. Therefore, the risk of transmission of this virus to health care workers is thought to be low. However, transmission without percutaneous exposure (through a break in the skin barrier) is theoretically possible, as respiratory secretions contain the virus. This is why it has been categorized as a biohazardous agent that should be managed in a high-level biosecurity laboratory. It is recommended that close contact with body fluids and infected tissues be avoided if Nipah infection is suspected.[1]

Prevention:

1. Using personal protective equipments
2. Conducting surveillance for Nipah infection on animal farms specially pig
3. Avoiding handling and processing infected farm animal like pigs.
4. Early detection and notification[16]

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