INTRODUCTION
In 1994, there was an outbreak of respiratory and neurologic infections affecting horses and humans who had been in close contact with the sick horses. To date, there have been seven human cases, with four mortalities. A new zoonotic virus from the paramyxovirus family was discovered and was named Hendra virus (HeV). In 1998–1999, there was an outbreak of fatal encephalitis among the pig-farming community in Malaysia, involving more than 350 patients, with a mortality rate of 40%. Isolation of a new zoonotic paramyxovirus closely related to HeV, in cerebrospinal fluid (CSF) samples of patients in Sungai Nipah village, Malaysia, led to the discovery of Nipah virus (NiV) as the etiologic agent. Although there have been no more outbreaks since May 1999 in Malaysia, there have been two outbreaks in West Bengal, India: the first in Suliguli in 2001, and the second in Nadia district in 2007. There have been outbreaks almost annually in Bangladesh between 2001 and 2012. These outbreaks were associated with more prominent respiratory symptoms and a higher mortality rate of 70%. The natural reservoir host for the henipaviruses was identified as fruit bats of the Pteropus species based on the presence of neutralizing antibodies to the virus, and isolation of virus from their urine and saliva. The wide geographic distribution of Pteropus bats as the disease vector, which expands from Asia and Australasia to West Africa, makes Henipavirus an important emerging cause of fatal encephalitis.

THE HENIPAVIRUS
HeV and NiV can be isolated from patients’ CSF, saliva, urine, pharyngeal and nasal secretions. They grow easily in all types of mammalian cells, but not in insect cell lines (Chua et al., 1999, 2000). They are single-stranded RNA paramyxoviruses, in the genus Henipavirus. Hence, they share many common characteristic features with other members of the Paramyxoviridae family. In tissue culture, they show a typical cytopathic effect pattern of cell fusion and formation of giant syncytial cells. Histologically, they exhibit a pleomorphic lipid bilayer envelope, with the formation of intracytoplasmic inclusions of viral nucleocapsids that gives rise to the typical “herringbone” appearance in infected cells under negative staining (Chow et al., 2000; Goldsmith et al., 2003).

Like their other Paramyxoviridae family, the genomic makeup of both HeV and NiV consists of six genes (N-P-M-F-G-L) flanked by a 3’ leader and 5’ trailer region. Both viruses have identical leader and trailer sequence lengths and hexamer-phasing positions for all of their genes (Chan et al., 2001). However, they demonstrate longer genomic length of 18 246 nucleotides for the Malaysian strain of NiV, 18 252 nucleotides for the Bangladesh strain, and 18 234 nucleotides for HeV, as compared to the average of 15 500 nucleotides in other paramyxoviruses (Chan et al., 2001; Harcourt et al., 2001). The Bangladesh NiV strain also exhibits the most genetic variability, with a 92% homology to the Malaysian strain (Harcourt et al., 2005), hence the probability of the more virulent nature of the Bangladesh NiV, associated with a higher mortality rate among the population affected.

EPIDEMIOLOGY
The first outbreak of HeV infection occurred in September 1994, in Hendra, Brisbane, Australia, involving 18 horses and two humans. This resulted in 14 deaths among the horses and one human fatality (Selvey et al., 1995). Retrospectively, in August 1994, there was a similar case reported in Mackay, North Queensland, where a person caring and assisting in the autopsy of two sick horses developed aseptic meningitis, initially with full}

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