

SHORT REPORT

Late presentation of Nipah virus encephalitis and kinetics of the humoral immune response

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Abstract

Nipah virus is a newly discovered paramyxovirus transmitted directly from pigs to humans. During a large encephalitis outbreak in Malaysia and Singapore in 1998-9, most patients presented acutely. A 12 year old child is described who developed encephalitis 4 months after exposure to the virus. She was diagnosed by a new indirect IgG enzyme linked immunosorbent assay (ELISA), which is also described. The late presentation and IgG subclass responses had similarities to subacute sclerosing panencephalitis. Nipah virus should be considered in patients with encephalitis even months after their possible exposure.

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Between September 1998 and June 1999, an outbreak of severe viral encephalitis occurred in peninsular Malaysia and Singapore, caused by the newly discovered Nipah virus.^{1,2} This paramyxovirus, closely related to Hendra virus,³ caused illness in pigs, and humans in close contact with pigs or their secretions, such as farmers and abattoir workers. During the outbreak, the diagnosis was made by virus isolation from CSF, or by serological tests against Hendra virus, because serological tests for Nipah virus had not yet been developed.⁴ Most patients infected with Nipah virus presented with acute encephalitis,^{2,5,6} but three patients had a neurological relapse up to 39 days after an initial mild illness.⁴ We report here a patient who presented with mild Nipah encephalitis 4 months after exposure to the virus; in addition we describe the development of an indirect IgG enzyme linked immunosorbent assay (ELISA) for diagnosing Nipah virus infection, and the IgG subclass responses.

Case report

A 12 year old girl presented to Sibu hospital, Sarawak in April 1999 with a 2 day history of fever and right frontal headache, and a 5 minute episode of sudden jerking movements of the left leg, which was followed by weakness and numbness of the same limb.

She was born in Sarawak, on the island of Borneo, but had moved with her two sisters

and parents to a pig farm in Negeri Sembilan, peninsular Malaysia, in 1997. Her work here included feeding the pigs, artificial insemination of sows, delivering piglets, and injecting sick pigs. In December 1998, when the Nipah virus outbreak reached Negeri Sembilan, her father became ill with encephalitis. In the same week our patient had a 2 day febrile illness with a runny nose, cough, headache, and blurred vision, which resolved spontaneously. In January 1999, after her father's death, she returned to Sarawak with her family. In February she had another brief episode of fever and chills which resolved with paracetamol. Her mother and sisters had remained well throughout. As a child the patient had been immunised with BCG, hepatitis B, oral polio vaccine, measles, diphtheria, pertussis, and tetanus. During the encephalitis outbreak she, along with many others, was vaccinated against Japanese encephalitis virus (JEV).

On examination at Sibu hospital, the patient was afebrile, and looked well. General medical examination was normal. She was fully conscious, but during the examination developed a further 4 minute episode of rhythmic jerking of the left leg. This limb was weak (power grade 4/5 in all groups), reflexes were normal, but tone was increased in both the left leg and left arm. She had no sensory loss, although she continued to report paraesthesia of the left leg. Over the next few hours her residual weakness and paraesthesia resolved.

Initial investigations including full blood count, urea, electrolytes, and liver function tests, were normal, except for a slightly low sodium concentration at 132 mmol/l. Examination of CSF showed a pleocytosis with 170 cells/mm³ (100% lymphocytes), a protein concentration of 82 mg/dl, and a glucose concentration of 48 mg/dl. No organisms were seen and bacterial culture was negative. Brain CT was normal.

Over the next 2 days, the patient had an intermittent mild pyrexia, and two brief episodes of generalised tonic-clonic seizures, which resolved without treatment. After loading with intravenous phenytoin (10 mg/kg) she had no further convulsions. Because of the CSF pleocytosis she was treated with penicillin and chloramphenicol, but she made an uneventful recovery and was discharged well, 10 days after admission. At 1 week, 1 month, 6

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