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Meeting Report

5th Annual Meeting of Japanese Society for Neurovirology

The 5th Annual Meeting of Japanese Society for Neurovirology was held in Osaka, Japan on November 17, 2001. The focus of the meeting was "Recent problems of neurological disorders caused by viruses and related agents in Japan." The meeting was well attended by clinical and basic scientists, as well as media personnel and administrators.

Four plenary sessions were offered. T. Morishima, from Nagoya University, organized the first session on "Post-influenza virus encephalopathy" and gave a presentation entitled "Pathobiology of influenza-encephalopathy and mechanism of the crisis." T. Iwasaki from the National Institute of Infectious Diseases presented "Pathological analysis of influenza encephalopathy." Both speakers reported that influenza-encephalopathy is seen more often in the infant population. It was suggested that a medication used in the treatment of influenza-encephalopathy in infants may be associated with higher rates of sudden death, although the mechanism of this crisis remains unidentified.

The following session, "New activities of neuroviruses," was organized by E. Konishi from Kobe University. K. Morita from the Institute of Tropical Medicine of Nagasaki University presented "Sudden outbreak of West Nile virus in USA." The results from genetic analysis of the virus raised some interesting questions and requires further investigation to determine if this strain originated in migrating birds, imported birds, or infected humans.

The second speaker, K. Ikuta from the Institute of Microbiological Diseases in Osaka University presented "Persistent infection and diseases of the central nervous system related with Borna virus infection." Although the Borna virus has been linked to mental illness, Ikuta reported that any correlation between Borna virus infection and mental illness requires further analysis to confirm a causative relationship. "Epidemic encephalitis due to Enterovirus 71 and Nipah virus" was presented by Thong K. Wong, Department of Pathology at the University of Maraya. Recently, Asia has experienced two major epidemics of viral encephalitis. In Malaysia, Taiwan, Japan, Singapore, and Australia a reemergence of enterovirus 71 caused severe encephalomyelitits in children. Necropsy studies showed severe perivascular cuffing, parenchymal inflammation, necrosis, and neuronophagia in the brainstem, diencephalons, and dentate nucleus of the cerebellum, along with mild inflammation in the meninges and focal areas of the cerebral cortex. Although no viral inclusions were detected, immunohistochemical and *in situ* hybridization studies conclusively demonstrated that the virus was neurotropic. Inflammatory cells participating in neuronophagia also showed evidence of the virus. Thus, viral cytolysis appears to be an important mechanism for neural damage. However, direct evidence of viral presence is usually focal, with inflammation often extending far beyond the areas of neuronal infection. This could suggest that other factors might also be involved in tissue damage. Further studies using human tissue and animal models could shed more light on the pathogenetic mechanisms of enterovirus 71 encephalomyelitis. The second epidemic of viral encephalitis occurred in Malaysia and Singapore and was caused by a novel paramyxovirus called Nipah. This epidemic was mainly concentrated in handlers of pigs. Pathological evidence suggested that endothelium of small blood vessels in the central nervous system were particularly susceptible to infection. This led to disseminated endothelial syncytial formation and damage, which triggered a series of events that included vasculitis, thrombosis, ischaemia, and microinfarction. However, there was also evidence of parenchymal infection by virus in the central nervous system and other organs. Thus, neurological dysfunction in acute Nipah encephalitis may be the result of both microinfraction and direct neuronal infection. Some patients who apparently recovered from the acute encephalitis may suffer a relapse encephalitis. Other infected patients, who soon after infection suffered only mild symptoms but not acute encephalitis, may develop a late-onset encephalitis in terms of clinicopathological features, including evidence to indicate that both were due to viral recurrence. It is now becoming clear that Nipah virus may have originated from fruit bats, which was transmitted to pigs who acted as amplifying host, eventually infecting humans. There is probably human-to-human transmission, but the rate is thought to be very low.

The third session of the meeting, "Measles virus infection and the neuropathogenesis," was organized by H. Ogura from Osaka City University. F. Kofune from the Institute of Medical Science of Tokyo

University presented on "Neuro-tropism of measles virus on a monkey model," followed by a presentation, "Establishment and its application of genetic engineering system of wild strain measles virus," given by K. Takeuchi from the National Institute of Infectious Disease. V. ter Meulen, from Wuerzburg University, was the third presenter to speak in this session. His topic was "Pathogenetic aspects of measles virus and human immunodeficiency virus-infection of the central nervous system." In spite of the development of effective vaccines for the measles virus, cases of the measles virus have recently increased in Japan, due to a decrease in the numbers of people receiving the measles vaccine. This phenomenon is not limited to Japan.

The fourth session was organized by M. Shinagawa of Obihiro University of Agriculture and Veterinary Medicine and was called "Progress in research on prion diseases." The first speaker was K. Doura of Kyusyu University. The title of his presentation was "Diagnosis and treatment of human prion disease, the present state and prospect." The second speaker, T. Yokoyama from the National Institute of Animal Health, delivered a presentation on "Prion diseases in animals." Prion diseases are caused by β-sheet rich prion. Creutzfeldt-Jakob disease (CJD) is one of the human prion diseases. In Japan, about 100 cases of iatrogenic transmission of CJD have been reported after dura transplantations. Additionally, bovine spongiform encephalopathy (BSE), the bovine prion disease, was recently reported in Japan. Early diagnosis of CJD and BSE is important. Methods for early diagnosis are in the developmental stage. Treatment for prion disease involves agents used in malaria medicines and derivatives of chlorophyll. Another approach in treatment includes the destruction of β-sheet rich prion and inhibition of accumulation of the prion, using anti-prion antibodies and detection of X-factor on prion folding. These treatments are still in the early stages of investigation.