JAPANESE ENCEPHALITIS

Dr Tom Solomon, Lecturer in Neurology and Medical Microbiology
University of Liverpool

Epidemics of encephalitis were described in Japan from the 1870s onwards, and Japanese encephalitis virus was first isolated from a fatal case in the 1930s. It is a small enveloped RNA virus, a members of the genus Flavivirus (family Flaviviridae), named after the prototype Yellow fever virus (in Latin yellow = flavus). The flaviviruses are relatively new viruses, derived from a common ancestor 10-20,000 years ago, that are rapidly evolving to fill new ecological niches. Japanese encephalitis virus is transmitted in an enzootic cycle between small birds by Culex mosquitoes, pigs are important amplifying hosts. Humans become infected by Culex mosquitoes coincidentally, but are not part of the natural cycle.

Japanese encephalitis virus has always been recognised as a killer. Over the last 50 years it has spread relentlessly across Southeast Asia, India, southern China, and the Pacific reaching Australia in 1998. Culex mosquitoes are unavoidable in rural Asia, and almost everyone is exposed to the virus. Only about 1 in 300 infections results in disease, and there is a wide range of presentations from a simple febrile illness to a severe meningoencephalitis, as well as a newly recognised polio-like acute flaccid paralysis. There are estimated to be 50,000 cases of Japanese encephalitis annually, with 15,000 deaths. The actual numbers may become clearer with the application of new simple rapid diagnostic tests. In addition to the high mortality, approximately half the survivors have severe neuropsychiatric sequelae, with their associated socioeconomic burden.

Recent findings in Asia raise important issues about the spread, control and pathogenesis of Japanese encephalitis. It is thought to be spread by birds, but mosquitoes blown between Pacific islands may contribute too. An expensive formalin inactivated and newer live attenuated vaccine against Japanese encephalitis are available, but not for the majority of the 2.8 billion people living in affected regions. For them, the factors determining who, of all those infected with Japanese encephalitis virus, develops neurological disease may be critically important. The relative contributions of the human immune response, and viral strain differences are currently being investigated.

References


© Copyright The Encephalitis Support Group 2002 All rights reserved.

The views expressed in any quoted resources represent those of the authors and are not the views or official policy of the Encephalitis Society and its Professional Panel.