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A REVIEW ON CURRENT SCIENARIO OF JAPANESE ENCEPHALITIS

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ABSTRACT

Japanese encephalitis is one of the neurological Infection. It is a Serious mosquito – Borne viral infection with broad range of manifestation. It mainly affects Animals, Humans and transmiited by Mosquito in Human causing inflammation of the membrane around the brain. Japanese encephalitis virus causes Japanese encephalitis by Flavivirus and is closely related to St. Louis Encephalitis and west nile Encephalitis. Approximately 20-30% of JE cases are Fatal and 30-50% of surviviour have significant neurological sequelae. In India Japanese encephalitis has claimed many lives in Bihar, West Bengal, Assam, Arunachal Pradesh etc currently there is no cure for Japanese encephalitis virus and treatment is mainly symptomatic and supportive therapy. A number of Anti Viral Agents have been investigated how ever none of these should convincingly improve their out come of Japanese encephalitis virus

KEYWORDS: Mosquito –Borne, Flavivirus St. Louis Encephalitis, Neurologic Sequelae.

INTRODUCTION

Japanese encephalitis is caused by the Japanese encephalitis virus (JEV), a flavivirus, and is closely related to St. Louis encephalitis and West Nile encephalitis. It occurs primarily in rural areas of Asia. Japanese encephalitis is spread through these regions by bites of culicine mosquitoes, most often Culex tritaeniorhynchus.[1] It is a single stranded, positive-sense polarity RNA genome of approximately 11 kb in length. The virion of JEV contains three structural proteins – nucleocapsid or core protein (C), non- glycosylated membrane protein (M) and glycosylated envelope protein (E), as well as seven non-structural (NS) proteins – NS1, NS2A, NS2B, NS3, NS4A, NS4B and NS.[2] Japanese encephalitis (JE) is among the most important viral encephalitis in Asia, especially in rural and suburban areas where rice

culture and pig farming coexist.[3-5] JE is an inflammatory disease affecting the central nervous system including the cerebrum, the cerebellum, and the spinal cord. JE is the most common vaccine-preventable encephalitis in Asia and occurs in annual epidemics or endemically in many Asian countries.[6-9] Humans are a dead end host and as such there is no human-to-human transmission. This mosquito species preferentially breeds in rice paddies and is a dusk and evening feeder. Approximately 99% of JE infections are asymptomatic; however, JE in symptomatic patients can be a devastating disease with a mortality of approximately 30%. Following an incubation period of 4–14 days, symptomatic patients can present with high fever, chills, headache, myalgia and confusion. In children gastrointestinal complaints like pain, vomiting can dominate initially and 75% will experience seizures.[10]

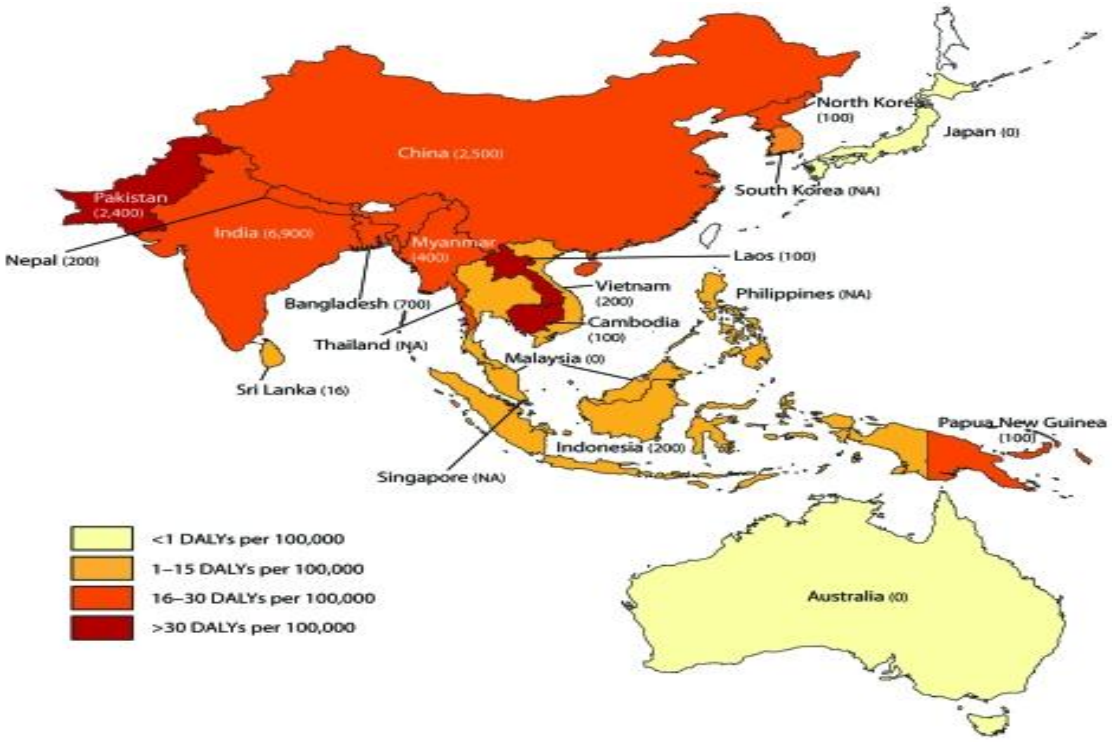
This article reviewed JVE literature from 2000-2016 outlining the indian scenario, deplication, diagnosis and prevention of this deadly disease.[11]

SOURCES OF INFECTION

- Water birds of the family Ardeidae; herons and egrets (also known as bitterns).
- Mosquito Vector.
- Once infected, swine amplify JE virus and high titres in blood, provide more infectious agent to vectors.
- JE virus could be transmitted in boar semen.[12]

EPIDEMIOLOGY

Japanese encephalitis is one of the most important forms of epidemic and sporadic encephalitis in the tropical regions of Asia, including Japan, China, Taiwan, Korea, Philippines, all of Southeastern Asia, and India; however, related neurotropic viruses are spread across the globe. Countries with proven epidemics of JE include India, Pakistan, Nepal, Sri Lanka, Burma, Laos, Vietnam, Malaysia, Singapore, Philippines, Indonesia, China, maritime Siberia, Korea, and Japan. In the past 50 years, the geographic areas affected by JEV have expanded. JE is the main cause of viral encephalitis in many Asian countries; it is endemic with seasonal distribution in parts of China, the Russian Federation's south-east, and South and South-East Asia. there has been no local transmission of JEV detected in Africa, Europe, or the Americas. There area reported 30, 000e50, 000 clinical cases of JEV annually, with an estimated mortality ranging from 10 to 15,000 deaths It is more common in rural areas where regular rice cultivation and flood irrigation occurs.[13]



CLINICAL FEATURES

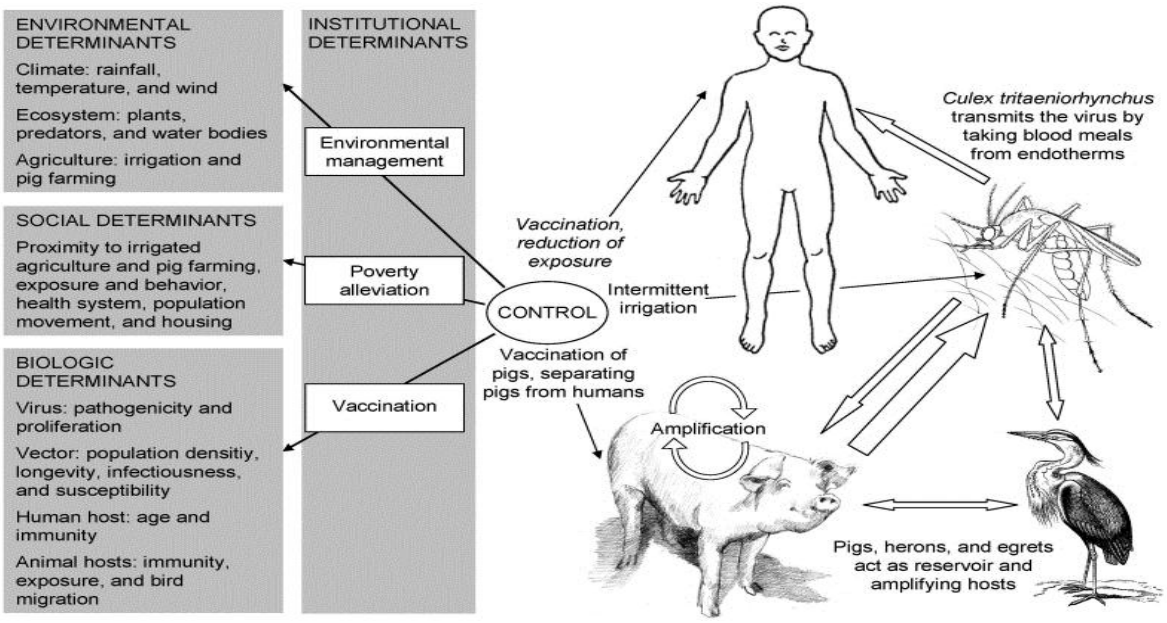
JE is primarily a disease that afflicts children but can occur among people of any age. People infected with JEV mainly are asymptomatic; infection is symptomatic in less than 1% of cases, and typically involves severe encephalitis. The incubation period of JE is 5-15 days and its earliest symptoms are lethargy, fever, headache, abdominal pain, nausea, and vomiting. Prodromal symptoms can evolve over several days to 1 week, when characteristic mental changes, focal neurological deficits, movement disorders and generalized weakness may develop over the next few days, thereafter advancing to progressive confusion, delirium, and coma.[14]

TRANSMISSION

Japanese encephalitis virus is usually transmitted by mosquitoes in the genus Culex. The specific mosquito vectors vary with the region; however, Culex tritaeniorhynchus is important in spreading this virus to humans and domesticated animals across a wide geographic range. *C.* tritaeniorhynchus breeds in rice paddies and connecting canals, and is active at twilight JEV is usually transmitted in mosquito bites, although lizards and bats can also become infected by eating infected mosquitoes. Humans and most domesticated animals are incidental hosts, with low viremia, and are not considered to be important in virus transmission. For example, while horse-to-horse transmission via mosquitoes has been demonstrated in the laboratory In addition to mosquito bites, infections in people have been

reported after exposure to JEV in the laboratory or during tissue sample collection. This virus can be transmitted through mucous membranes or broken skin, inhaled in aerosols, or acquired by needlestick injuries.

Japanese encephalitis virus does not survive well outside a living host. How the virus persists during the winter in temperate climates is uncertain, although various mechanisms have been suggested.[15]



ETIOLOGY

Japanese encephalitis is caused by a flavivirus, which can affect both humans and animals. The virus is passed from animals to humans through the bite of an infected mosquito.

Pigs and wading birds are the main carriers of the Japanese encephalitis virus. A mosquito becomes infected after sucking the blood from an infected animal or bird. If you get bitten by an infected mosquito, it can pass on the virus.

The mosquitoes that carry Japanese encephalitis usually breed in rural areas, particularly where there are flooded rice fields or marshes, although infected mosquitoes have also been found in urban areas. They usually feed between sunset and sunrise. Japanese encephalitis can't be passed from person to person.[16]

PATHOGENESIS

After attachment of the Japanese encephalitis virus to a host cell membrane, local membrane disruption may lead to entry of the virus into the cell itself. The virus initially propagates at the site of the bite and in regional lymph nodes. Two cellular characteristics are critical to the pathogenesis: (1) the M protein, which contains hydrophobic domains that help to anchor the virus onto the host cell, and (2) the E protein, which is the principal immunogenic feature and which is expressed on the membrane of infected cells. The E protein mediates membrane fusion of the viral envelope and the cellular membrane, promoting viral entry into the host cell. The Japanese encephalitis virus replication cycle includes initial host cell receptor interaction of the virus followed by receptor-mediated endocytosis, fusion of the viral and host cell membranes, subsequent cytoplasmic release of viral genome, and several other transcription and pretranslation steps. Maturation of virus particles occurs in the Golgi complex, followed by ultimate release of the virus Subsequently, viremia develops, leading to inflammatory changes in the heart, lungs, liver, and reticuloendothelial system. Most infections are cleared before the virus can invade the CNS, leading to subclinical disease. Subclinical or mild forms of Japanese encephalitis resolve in a few days if the CNS is not involved. In such cases, the infection may not produce symptoms and therefore remains undetected. However, given the neurotropic character of Japanese encephalitis virus, neurologic invasion can develop, possibly by growth of the virus across vascular endothelial cells, leading to involvement of large areas of the brain, including the thalamus, basal ganglia, brain stem, cerebellum (especially the destruction of the cerebellar Purkinje cells), hippocampus, and cerebral cortex. Overall, Japanese encephalitis virus is believed to result in increased CNS pathology because of its direct neurotoxic effects in brain cells and its ability to prevent the development of new cells from neural stem/progenitor cells (NPCs). Japanese encephalitis virus likely represents the first mosquito-transmitted viral pathogen to affect neural stem cells. These cells can serve important roles in injury recovery; consequently, Japanese encephalitis–induced disruption of neural stem cell growth may be particularly important to further morbidity and mortality.[17]

DIAGNOSTIC TESTING

Japanese encephalitis patients should be consider with the evidence of neurological infection like (meningitis, encephalitis) who have recently travelled to endemic area or resident in endemic region JE generally accomplished by testing of serum or cerebrospinal fluid (CSF)

to detect virus-specific IgM antibodies. JE virus IgM antibodies are usually detectable 3 to 8 days after onset of illness and persist for 30 to 90 days. Past IgM antibodies.

Occasionally may reflect a past infection or vaccination Serum collected within 10 days of illness onset may not have detectable IgM, and the test should be repeated on a convalescent sample. For patients with JE virus IgM antibodies, confirmatory neutralizing antibody testing should be performed. In fatal cases, nucleic acid amplification, histopathology with immunohistochemistry, and virus culture of autopsy tissues will be useful.[18]

CLINICAL MANIFESTATION

Most of JE infections are mild(fever and head ache) or with out apparent symptoms. 1 in 250 infections results in severe clinical illness. Severe disease is characterized by rapid onset of high fever, headache, neck stiffness, disorientation, coma, seizures, spastic paralysis and ultimately death. The case-fatality rate can be as high as 30% among those with disease symptoms.[19] Patients with JEV infection have a history of mosquito exposure in an endemic area, with the subsequent occurrence of the following signs and symptoms: The prodromal period is characterized by fever, headache, nausea, diarrhea, vomiting and myalgia, which may last for several days.[20]

MANAGEMENT

Treatment of JE is essentially supportive. Most of the case should be managed by intensive care unit. Supportive measures include maintenance of airways, breathing and circulation, hydration, electrolyte status, and control of pyrexia and convulsions. It is prudent to use appropriate parenteral antibiotics to cover for bacterial infection. Raised intracranial tension should be controlled with mannitol infusion., intravenous furosemide or intermittent positive pressure ventilation to keep arterial carbon dioxide tension.proper nursing care is important to prevent aspiration pneumonia and bedsore. Role of steroid usage is still debateable.

Interferon therapy has not met with great success. A recent clinical trial of oral ribavarin administration was also not encouraging.[21]

PREVENTION

Orientation planning and consulation works shop should be conducted. JE campaign planning meetings should be held for the districts undertaking the JE campaign. Information about

cause prevention and mosquito bites. Educting publics about mosquito breeding places filling pools, weekly drainage of accumulated water, lowering of water levels in rice fields etc.[22]

CONCLUSION

Japanese encephalitis is still one of the major health problem in india. No antiviral Drug against JEV is not available for treatment only prevention can control the disease. Environmental and ecological factors are responsible for spread of JE. Control can be made after developing high quality immunization program.

REFERANCE

1. http://emedicine.medscape.com/article/233802-overview
2. T.J. Chambers, C.S. Hahn, R. Galler, C.M. Rice Flavivirus genome organization, expression, and replication Annu Rev Microbiol, 1990; 44: 649-688.
3. Halstead SB, Jacobson J. Japanese encephalitis vaccines. In: Plotkin SA, Orenstein WA, Offit PA, editors. Vaccines. 5th ed. Philadelphia: Elsevier, 2008; 311-52.
4. Burke DS, Leake CJ. Japanese encephalitis. In: Monath TP, editor. The arboviruses: epidemiology and ecology, Vol. 3. Boca Raton: CRC Press, 1988; 63-92.
5. Fischer M, Lindsey N, Staples JE, Hills S; Centers for Disease Control and Prevention (CDC). Japanese encephalitis vaccines: recommendations of the Advisory Committee on Immunization Practices (ACIP). MMWR Recomm Rep, 2010; 59(RR-1): 1–27. PMID: 20224546.
6. Campbell GL, Hills SL, Fischer M, Jacobson JA, Hoke CH, Hombach JM, Marfin AA, Solomon T, Tsai TF, Tsu VD, Ginsburg AS. Estimated global incidence of Japanese encephalitis: a systematic review. Bull World Health Organ, 2011; 89: 766–774.
7. Hanna JN, Ritchie SA, Phillips DA, Shield J, Bailey MC, Mackenzie JS, Poidinger M, McCall BJ, Mills PJ (1996) An outbreak of Japanese encephalitis in the Torres Strait, Australia, 1995; Med J Aust 165: 256.
8. Hanna JN, Ritchie SA, Phillips DA, Lee JM, Hills SL, van den Hurk AF Pyke AT, Johansen CA, Mackenzie JS (1999) Japanese encephalitis in north Queensland, Australia, 1998. Med J Aust, 170: 533–536.
9. Travelers' health: yellow book. Available at: http://wwwnc.cdc.gov/travel/yellowbook/2014/chapter-3- infectious-diseases- related-to-travel/japanese-encephalitis.

10. Japanese Encephalitis Vaccines: WHO position paper – February 2015. Wkly Epidemiol Rec, 2015 Feb 27; 90(9): 69–87.
11. Review article Japanese encephalitis: a review of the Indian perspective in The Brazilian Journal of INFECTIOUS DISEASES ELESVIER Publication braz j infect dis, 2012; 16(6): 564–573.
12. A Recent review on Japanese encephalitis in word journal of pharmaceutical science, 3(3): 545-556. Review Article ISSN 2278–4357.
13. Review Article Current recommendations for the Japanese encephalitis vaccine Journal of the Chinese Medical Association, 2015; 78: 271e275.
14. *Japanese B Encephalitis, Arbovirus B, Mosquito–borne Encephalitis Virus* Last Updated: January 2016.
15. Rethinking Japanese Encephalitis Virus Transmission: A Framework for Implicating Host and Vector Species.
16. A review of Japanese encephalitis in Uttar Pradesh, India September 14, 2017, IP: 117.242.162.214].
17. Epidemiology, Disease Transmission and Pathogenesis Caused by JE Virus: Its Prevention and Control American Journal of Infectious Diseases and Microbiology, 2015; 3(1): 38-64.
18. Center for disease control and prevention (CDC) CDC 24/7: saving lives, protecting peoples.
19. http://www.who.int/mediacentre/factsheets/fs386/en/
20. Japanese Encephalitis—A Pathological and Clinical Perspective Debapriya Ghosh, Anirban Basu* National Brain Research Centre, Manesar, Haryana, India PLOS Neglected tropical Disease.
21. Prevention, diagnosis and management of Japanese encephalitis in children kumar R in Dovepress.
22. Role of Education and Counseling for the Prevention of Japanese Encephalitis in the Eastern U.P, India Priyanka Srivastava Abhishek Sing Anuj Kr Srivastava, Abhay P Singh and Dev Prakash epidemiology open access.