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Research Article | www.enlivenarchive.org | Enliven: Microbes and Microbial Techniques

ISSN: 2576-2540

Eastern Equine Encephalitis: A Highly Virulent Emerging and Re-Emerging Viral Metazoonosis

Mahendra Pal[1], and Raj Kumar Singh[2]*

[1]*Narayan Consultancy on Veterinary Public Health and Microbiology, Aangan, Jagnath Ganesh Dairy Road, Anand-388001, Gujarat, India*

[2]*Indian Veterinary Research Institute, Izatnagar- 243122 India*

***Corresponding author**: Mahendra Pal, Founder of Narayan Consultancy on Veterinary Public Health and Microbiology, Aangan, Jagnath Ganesh Dairy Road, Anand-388001, Gujarat, India, E-mail: palmahendra2@gmail.com

Received Date: 25th March 2019
Accepted Date: 26th March 2019
Published Date: 1st April 2019

Citation: Pal M(2019) Eastern Equine Encephalitis: A Highly Virulent Emerging and Re-Emerging Viral Metazoonosis. Enliven: Microb Microbial Tech 6(1): 00e1.

Vector is defined as an arthropod or invertebrate host, which transmit the infection by inoculation into the skin or mucous membrane by biting or by depositing of infective material on the skin or on food or other objects. A number of vectors, such as mite, mosquito, tick, sand fly, tsetse fly, etc. are known to transmit many infections to humans and animals including birds. An infectious disease transmitted biologically by invertebrate vector is known as metazoonosis. There are several mosquitoes borne viral metazoonoses, such as chikungunya fever, dengue fever, eastern equine encephalitis, Ilheus fever, Japanese encephalitis, Murray Valley encephalitis, Rift Valley fever, Rossa river fever, Saint Louis encephalitis, Sindbis fever, Venezuelan equine encephalitis, western equine encephalitis, West Nile fever, and yellow fever, which are reported from many countries of the world [1]. Among the mosquitoes transmitted zoonoses, eastern equine encephalitis (eastern equine encephalomyelitis, eastern encephalitis) is an important life threatening vector borne viral disease of humans as well as equines. Eastern equine encephalitis (EEE) is an emerging and re-emerging arboviral zoonosis of major public health concern as case fatality rate in humans may reach up to 70 % [1,2]. In USA, eastern equine encephalitis is one of the most severe mosquito borne viral diseases with around 33% mortality. Human infections are diagnosed every year in North America. However, most cases of EEE have been reported from Florida, Massachusetts, New York, and North Carolina. It is pertinent to cite that outbreaks of disease in horses and pheasants frequently precede human cases. Furthermore, outbreaks of EEE may occur in humans and horses during the late summer and fall [3]. A massive outbreak of EEE killed 12,000 horses in Louisiana, USA in 1947 [4]. Outbreaks of eastern equine encephalitis in equids were reported from May 2008 to August 2009 in the Brazil, which affected 229 equines with a case fatality rate of 72.92% [3].

Eastern equine encephalitis (EEE) is characterized by high fever, severe headache, lethargy, and seizures. The disease was described for the first time by Feemster [5] during an outbreak in Massachusetts, USA. During the outbreak, Farber and co-investigators [6] reported 8 cases of eastern equine encephalitis in children Since then, the disease is endemic to the eastern United States and the Gulf Coast [7,8]. Natural infection is recorded in humans and also in a wide variety of domestic mammals (alpacas, cattle, deer, dog, donkey, goat, horse, llamas, mule, pig, seal, sheep), and birds (chukar, egret, emu, ostrich, partridge, pheasant, pigeon, turkey) [1,4]. An outbreak of eastern equine encephalitis in humans was reported in Jamaica during 1962 [3]. The epidemics of EEE in humans have occurred in Massachusetts during 2004–2006 and in New Hampshire during 2005 and 2007 [8]. Disease is caused by eastern equine encephalitis virus (EEEV) that belongs to Genus Alphavirus, and Family Togaviridae. It is a single stranded, enveloped icosahedral, nucleocapsid, linear, positive-sense RNA genome. The infectivity of virus is reduced after exposure to irradiation , and inactivated at pH 1-3.The virus is sensitive to heat, formaldehyde, detergents, chloroform, ether, trypsin, b-propiolactone, and can be inactivated by ethanol (50%) for one hour [3,4]. Due to high virulence, EEE virus is classified as a bioterrorism agent by CDC.

Transmission of infection occurs following bites of infected mosquitoes [1,9]. In the United States, EEE virus has been isolated from 27 species of mosquitoes. It is important to mention that Culiseta melanura, which primarily feeds on birds, is the most important vector in the enzootic cycle [4]. Further, bridge vectors like Aedes canadensis, Ae.vexans, Coquillettidia perturbans, and Culex salinarius may transmit EEE virus from reservoir avian species to humans, horses, pigs, and pheasants. Mosquito species, such as Culex erraticus, Cx. peccator, and Uranotaenia sapphirina may also serve as enzootic vectors in some regions of the United States [3,4]. The virus is found in Canada, and USA. In addition, EEE virus also occurs in the Caribbean and regions of Central and South America, particularly along the Gulf coast [3].

The incubation period in humans is 7 to 10 days. The infection in humans has a range of clinical presentations, from asymptomatic to fatal encephalitis. The disease is manifested by sudden onset of fever, severe headache, vomiting, anorexia, chills, sore throat, arthralgia, myalgia, abdominal pain, diarrhea, lethargy, neck stiffness, irritability, restlessness, cyanosis, confusion, stupor, disorientation, tremors, seizures paralysis, coma, and death [1,4,10]. In children, sometimes periorbital edema, facial edema or generalized edema is observed. Encephalitis occurs most frequently in children less than 15 years and people over 55 years of age. Death usually occurs 2 to 10 days after onset of symptoms. The case fatality rate may vary from 30% to 70% [11]. The morbidity is very high (90%).The people who do not develop neurological signs usually recover completely after an illness of 1 to 2 weeks [4]. It is mentioned that neurologic sequelae, such as seizures, paralysis, and mental retardation can occur in up to 80% of the survivors, particularly in children and aged persons . Many patients with severe sequelae die within a few years [3].

Clinical findings should be supported with laboratory tests to confirm the disease. Computed tomography and magnetic resonance imaging may help to detect lesions in brain. Immunological techniques, such as hemagglutination inhibition, complement fixation, and serum neutralization are useful to demonstrate antibodies in the sera of patients [3,4]. The isolation of virus from the clinical specimens can be attempted in Vero cells (African green monkey kidney), baby hamster kidney cells, and duck embryo fibroblasts. Detection of eastern equine encephalitis virus can be done by reverse transcription polymerase chain and nucleic acid amplification assay [3,12]. It is necessary to create containment level 3 facilities in the laboratory to work with EEEV. Regular disinfection of working table in the laboratory with 1% sodium hypochlorite is highly imperative. It is important to mention that serologic test remains the mainstay for diagnosing the infection [3].

Presently, no antiviral therapy is available for eastern equine encephalitis. Therefore, supportive treatment is focused mainly on managing complications, such as seizures and increased intra cranial pressure. The paracetamol can be taken to bring down fever and reduce joint pain.

Currently, there is no commercial vaccine to immunize humans for eastern equine encephalitis virus. Therefore, isolation of patient in screen room, provision of protective clothing, use of mosquito net, application of repellent cream (N. N-diethylbenzamide) on uncovered skin, installation of screen and nets in dwellings, treatment of clothes and gear (pants, socks, boots, and tents) with permethrin, removal of standing water sources around the home, covering of water containers, spraying of insecticides, precautions during necropsies on horses, restriction on the movement of diseased horse, vaccination of equines, and health education of the public regarding the mode of spread, severity of disease and preventive measures are imperative to mitigate the incidence of this metazoonosis [1,3,4]. It is stressed that permethrin should not be applied directly on the skin. Parents are advised not to use insect repellent on babies younger than 2 months old [3].

It is emphasized that person with neurological symptoms in endemic regions should be investigated for EEE infection. There is a need to undertake surveillance in birds for predicting outbreaks of disease.

Further work on the development of potent, safe, and low cost antiviral chemotherapeutic agent and vaccine for the humans may be rewarding.

Acknowledgments

The author wishes to thank Prof. Dr. R. K. Narayan for going through the manuscript and Anubha for computer help. This paper is dedicated to Wing Commander Abhinandan Varthaman of Indian Air Force for his great valor, courage, and brave action by shooting down enemy F-16 aircraft by his MiG-21 aircraft on February 27, 2019. India feels very proud on such dedicated soldier.

References

1. Pal M (2007) Zoonoses. First Edition, Satyam Publishers, Jaipur, India.
2. Gibney KB, Robinson S, Mutebi JP, Hoenig DE, et al. (2011) Eastern equine encephalitis: an emerging arboviral disease threat, Maine. Vector Borne Zoonotic Dis 11: 637-639.
3. Pal M (2013) Public health importance of emerging and re-emerging viral zoonoses. Ph.D. Lecture Notes. Addis Ababa University, College of Veterinary Medicine, Debre Zeit, Ethiopia 1-36.
4. CFSPH (2017) Eastern, Western and Venezuelan equine encephalitis. Centre for Food Security and Public Health, Iowa State University, Ames, USA. 1-12.
5. Feemster RF (1938) Outbreak of encephalitis in man due to the eastern virus of equine encephalomyelitis. Am J Public Health Nations Health 28: 1403-1410.
6. Farber S, Hill A, Connerly ML, Dingle JH (1940) Encephalitis in infants and children caused by the virus of the eastern variety of equine encephalitis. JAMA 114:1725- 1731.
7. Calisher CH (1994) Medically important arboviruses of the United States and Canada. Clin Microbiol Rev 7:89-116.
8. CDC (2006) Eastern equine encephalitis—New Hampshire and Massachusetts, August–September 2005. Morbidity and Mortality Weekly Report 55: 697-700.
9. Hachiya M, Osborne M, Stinson C,Werner BG (2007) Human eastern equine encephalitis in Massachusetts: predictive indicators from mosquitoes collected at 10 long-term trap sites, 1979-2004. Am J Trop Med Hyg 76: 285-292.
10. Deresiewicz RL, Thaler SJ, Hsu L, Zamani AA (1997). Clinical and neuroradiographic manifestations of eastern equine encephalitis. N Eng J Med 336:1867-1874.
11. Przelomski MM, O'Rourke E, Grady GF, Berardi VP, Markley HG (1988) Eastern equine encephalitis in Massachusetts: a report of 16 cases, 1970–1984. Neurology 38: 736-739.
12. Lambert AJ, Martin DA, Lanciotti RS (2003) Detection of North American eastern and western equine encephalitis viruses by nucleic acid amplification assays. J Clic Microbiol 41:379-385.