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Zika Virus Induced Neuropathy

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Abbreviations: ZIKV: Zika virus; MCPH: Microcephaly;

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The association between Zika Virus (ZIKV) epidemic and neurological disorder has raised an urgent global alarm. The current epidemic of ZIKV has triggered quick responses in the scientific world. The first case of ZIKV was reported in 2015 from Brazil and now has spread over 30 countries. Nearly four hundred cases of traveler associated ZIKV infection have also been reported in the United States. ZIKV is primarily transmitted by mosquito belongs to the genus *Aedes* that are widely distributed throughout the world. Additionally, the virus can also be transmitted from male to female by sexual contact. The epidemiological investigations during the current outbreak found a causal link between infection in pregnant women and development of microcephaly (MCPH) in their unborn babies. This finding is a cause for grave concern since MCPH is a serious neural developmental disorder that can lead to significant post-natal developmental abnormalities and disabilities. Recently, published data indicates that ZIKV infection severely affects the growth of fetal neural progenitor cells and cerebral neurons resulting into malformation of cerebral cortex leading to MCPH. Recently, it has been reported that ZIKV infection deregulates the signaling pathway of neuronal cell and inhibit the neurogenesis resulting into dementia.

Zika virus (ZIKV) outbreak reported for the first time in 2015 from Brazil and has now been spread to over 30 countries. Currently, four hundred cases of ZIKV infection have been reported in United States of America. ZIKV is primarily transmitted by mosquitos of the genus Aedes that are widely distributed throughout the world. Additionally, the virus can also be transmitted from male to female by sexual contact [1]. The phylogeny of ZIKV suggests two important lineages known as African and Asian, evaluating from one ancestry, mostly apparent in Uganda [3]. Probably the vectors are *Aedes polynesiensis* and *Aedes aegypti,* a species of Aedes found in French Polynesia, reported in Yap [4-6]. *Aedes species* found in various parts of USA [2,4]. The viral RNAs has 3419 amino acids and host and viral proteases help in the translation and cleavage of polyprotein into structural and non-structural proteins [7]. Initially, the ZIKV attached with host endosomal membrane by endocytosis and then ssRNA is released into host cytoplasm followed by translation and cleavage of protein and finally resulted in formation of viral proteins. In the next step, the dsRNA produced after replication in endoplasmic reticulum followed by production of additional ssRNAs to form new virus progeny. The newly emerged virus particles move into Golgi complex and finally released into the intracellular spaces to infect the neighboring cells [8]. There is a very close link between flaviviruses such as Zika, Chikungunya and Dengue, which have common symptoms like a headache, rash, myalgia, arthralgia fever, and maculopapular. The published information provided a convincing evidence about the development of neurological symptoms like Dementia in South America

and Polynesia [9,10]. Based on published data the ZIKV infection to pregnant women will lead to the development of MCPH in the developing fetuses [1]. This is a cause for grave concern since MCPH is a serious neural developmental disorder that can lead to serious lifelong developmental abnormalities and disabilities in the affected babies. This disease is characterized by a smaller head circumference with reduced brain size due to a poorly-developed cerebral cortex. The current paradigm is that a key factor in ZIKV-induced MCPH is apoptosis of infected fetal cortical neural stem/progenitor cells those results in depletion of neurons from the cortex and consequent malformation of the cerebral cortex leading to MCPH.

Summary and Future Prospects

In this column, we have provided an updated information demonstrating the link between ZIKV epidemic and MCPH to the risk of dementia. Based on the published information provided, it is well known that placental infection of the progenitor neuronal cells in the growing embryo, mainly in the first trimester, is the main primary region for the ZIKV infection linked neuronal malformation. ZIKV efficiently infects human fetus brain cells and tissue both *in vitro* as well as *in Vivo*. Animal and mouse models have also repeated critical attitude of human viral pathology related to fetus demise, fetus neuroids epidemiology and cortical growth *in vivo*. This is apparent that the macular degeneration identified with few cases of the microcephalic newly born babies along with a result of ZIKV infection in the growing fetal retinal construction, as it was also observed in virus affected mice, newborn babies. ZIKV deregulate Akt host cell signaling pathway which is very essential for brain development in adult human as well as the fetus. There is few evidence has been reported for the capability of ZIKV to target central nervous system of adult human and induced dementia like symptom, but considerable number of cases are required to analyze further confirmation and association of ZIKV in dementia development. Based on current published reports, there is an urgent need to conduct more research to gather updated information so that an effective disease management strategy can be designed and developed to protect the spread of ZIKV infection.

References

1. Li C., *et al. "*Zika Virus Disrupts Neural Progenitor Development and Leads to Microcephaly in Mice". *Cell Stem Cell* 19.1 (2016): 120-126.
2. Fahim Ahmad. "ZIKV leads to Microcephaly". *Ec Microbiology* ECO.01 2016: 12-13.
3. Lanciotti RS., *et al*. "Genetic and serologic properties of Zika virus associated with an epidemic, Yap State, Micronesia, 2007". *Emerging Infectious Diseases* 14.8 (2008): 1232–1239.
4. Hennessey M. "Zika virus spreads to new areas-region of the Americas, May 2015–January 2016". *Morbidity and Mortality Weekly Report* 65.3 (2016): 1031–1034.
5. Cao-Lormeau VM., *et al*. "Zika virus, French Polynesia, South pacific, 2013". *Emerging Infectious Diseases* 20.6 (2014): 1085-1086.
6. Duffy MR., *et al*. "Zika virus outbreak on Yap Island, Federated States of Micronesia". *The New England Journal of Medicine* 360 (2009): 2536–2543.
7. Haddow AD., *et al.* "Genetic characterization of Zika virus strains: geographic expansion of the Asian lineage". *PLOS Neglected Tropical Diseases* 6 (2012): 1477.
8. MicrobeWiki. "Reproductive Cycle of a Zika virus in a Host Cell". Flaviviridae Swiss Institute of Bioinformatics. 2016.
9. Mlakar J., *et al.* "Zika Virus Associated with Microcephaly". *The New England Journal of Medicine* 374 (2016): 951–958.
10. Ioos S., *et al.* "Current Zika virus epidemiology and recent epidemics". *Medicine ET Maladies Infectieuses* 44.7 (2014): 302-307.