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Review article

A background review on zika virus infection with current update on Indian scenario

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ABSTRACT

It was in Uganda that the Zika virus was discovered for the first time in 1947. Later in 1952, researchers isolated an infectious agent from rhesus monkeys. 60 years later, in 2007, the first outbreak of Zika virus happened in Brazil. Afterwards cases of infection were reported from Tanzania, Egypt, South Asia, India, Bangladesh, Pakistan, Vietnam, Indonesia, Thailand, Malaysia, Philippines. The most common reason for infection was travelling to infected regions. The main concern in this infection is microcephaly in new born babies and foetuses. In 2016, the first outbreak of Zika virus infection occurred in India, the first three cases were discovered in Gujarat and later one case was found in Tamil Nadu. After two years in two Indian states another outbreak was reported in Rajasthan and Madhya Pradesh with 153 and 130 cases, respectively. Infection spreads due to *Aedes aegypti* mosquito bites or sexual transmission. Zika virus symptoms include mild fever, rash, joint pain and conjunctivitis. It is necessary to conduct some serological and molecular analyses to diagnose infection. There is no specific vaccine or treatment for Zika virus infection till yet. In this review, we explored all the factors related to zika virus infection in detail, including virology, pathogenesis, diagnosis, prevention, and symptoms with special emphasis on cases in India.

Key words: Zika virus, Aedes Aegypti, Yellow Fever, Flaviviridae

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INTRODUCTION

Yellow fever was first discovered in 1947. The Zika virus was found in the forest of Uganda. The meaning of Zika fever is overgrown in Luganda (Ugandan language). In1952, researchers isolated the infectious agent from monkey serum (Rhesus766). Eventually, it was separated from the victims of the tropical regions of Nigeria [1-3]. Recently, victims of Micronesia and Yap, previously found in Brazil, the Cook Islands and New Caledonia, have been reported. Serological evidence of human deer virus was reported in Tanzania, Egypt, South Asia, India, Bangladesh, Pakistan, Vietnam, Indonesia, Thailand, Malaysia, Philippines. In Malaysia, Zika virus was isolated from *Aedes aegypti*. In addition, according to the CDC, recent outbreaks outside Africa, Asia, and many other places [4,5].

Virology

Virus has positive strand RNA with the size of 11 kb in the Flaviviridae family. Zika virus is 4060 nm in diameter and has a lipid envelope. RNA has one long open reading frame, flanked by 50 and 30 terminal noncoding regions that form specific secondary structures required for genome replication and translation [6-8]. Essential component of the virus surface is the E protein, which plays a role in receptor binding and membrane fusion. Domain III of the E protein consists of a panel of neutralizing antibodies, serological tests, and epitopes by the vaccine. Study of molecular evolution of Zika virus based on virus strains collected from four West African countries between 1947 and 2007 identified multiple sites within the Zika virus genome that are under strong negative selective pressure. This finding suggests frequent flushing of harmful substances, polymorphisms in functionally significant genes and possibility of rare recombination flavi virus [9]. Zika virus antigen found only in the nucleus of infected cells. This discovery of a replication site distinct from other flavi viruses makes it worthy of further investigation [10].

Pathogenesis

Zika virus remains in mosquito salivary glands for weeks and is transmitted to humans through bites of *Ades aegypti* mosquitoes [11,12]. However, transmission from mother to child could pose a serious threat and could lead to fibroblasts, keratinocytes, and Langerhans cells all contain (Tyro3 and AXL, Tim-1 and DC-sign) respectively, which can serve as pathways for virus entry [13]. The ethology of microcephaly due to Zika virus infection is still unknown due to limited experimental evidence. Recently, several studies have demonstrated the pathogenic potential of Brazilian strains of Zika virus between fetal developments in animal models. These studies concluded that Zika virus infects the placenta first and then the fetal brain preferentially infects precursors. It destroys cells and reduces viability and growth as neurospheres by down regulating the genes

involved in cell and organ development and gene up regulation involved in the immune response. This inhibits cell proliferation and differentiation, resulting in neuronal Apoptosis, cortical thinning and macroscopic features Similar to microcephaly [14]. Zika virus is a positive-sense, single-stranded RNA virus. The Flaviviridae family includes several other clinically significant mosquitos borne viruses. The closest relative of Zika virus is the Spondweni virus. Zika virus genome contains 10,794 nucleotides encoding 3,419 amino acids. Like other flavi viruses, Zika virus consists of a capsid, a membrane precursor, an envelope, and seven non-structural proteins. According to phylogenetic analysis, Zika virus is classified into various African and Asian subspecies. Both originated in East Africa in the late 19th or early 19th century 1900's. In Malaysia, Asian bloodlines emerged during the virus migration from Africa to Southeast Asia. Zika virus spreads from that island in the Pacific, separated from Yap and French Polynesia, as well as New Caledonia, Cook Islands, Easter Island, and America. One study showed that human skin fibroblasts, keratinocytes, and immature dendritic cells allow Zika virus to enter the body. After entering the cell, flavi viruses typically replicate within the endoplasmic reticulum vesicles. However, Zika virus antigens have only been found in the nucleus of infected cells. This finding suggests a replication site distinct from that of other flavi viruses. [15-17]. The virus has an intrinsic latency period of 4–5 days within the human host, infects another vector during blood feeding, where it spends an 8–12-day exogenous latency period before spreading and manifesting in the vector's saliva[18].

Symptoms

Up to 4 out of 5 people infected with the Zika virus have no signs or symptoms. Symptoms usually develop 2 to 14 days after being bitten by an infected mosquito. Symptoms usually last about a week and most people recover completely. Signs and symptoms of the Zika virus most commonly include [Mild fever, Rash Joint pain, particularly in the hands or feet, Redeyes (conjunctivitis), Microcephaly symptoms are (Small head, abnormalities in brain function can cause death in the foetus) [19-21].

Diagnosis

Clinical Presentation

In the absence of other arbo virus epidemics, diagnosis can be made on clinical grounds alone. However, as already mentioned, Zika virus outbreaks are usually associated with other arbo virus epidemics and require diagnostic investigation to clarify the clinical picture [22].

Serological analysis

Detection of IgM antibodies in a patient's serum by ELISA is a valid method, but not available in most laboratories. Additionally**,** cross reactivity with other antibodies to other arbo viruses reduces the specificity of this technique [23-25]. In a recent study, serum samples from 21acuteundifferentiatedfever patients in Thailand were tested for immuno reactivity to Zika virus infection, dengue**,** Japanese encephalitis**,** and chikungunya envelope antigens. This study showed evidence of immuno reactivity to the ZIKV envelope, suggesting that the Zika virus outbreak may have spread to Thailand. However, due to the cross reactivity of serological analyses**,** urges need of more specific diagnostic methods [26].

Molecular Diagnostics (RT-PCR)

Molecular diagnostics can be performed using reverse transcriptase polymerase chain reaction (RT-PCR) [27]. Diagnostic studies suggest that serum maybe positive for viral particles as soon as illness and fever appear, but begins to decline when a rash appears. Nucleic acids remain detectable for approximately 20 to 60 days after the onset of symptoms [28,29]**.** During an epidemicin French Polynesia, Kutuna et al. reported positive viral RNA in urine, buta serum sample from the same patient was negative [30]. Courinat et al. reported that virus was detectable at in the urine of infected individuals 3 days after the onset of illness [25]. These data are consistent with previous studies suggesting long-term detection of viral RNA from other flavi viruses such as dengue virus and West Nile virus in urine samples. These reports highlight the role of urinary virus detection as a diagnostic tool for Zika virus infection during the epidemic [31,32].

Treatment

The infection can be prevented simply by avoiding mosquito bites, reducing sexual transmission and avoiding unnecessary travel to areas of ongoing Zika virus infections [33]. Currently development of an effective Zika virus vaccine has become a major priority globally, as there is no confirmed vaccine and selective antiviral drug against this infection [34]. As a part of primary treatment, patients are advised to drink plenty of fluids to prevent dehydration [35]. Many existing drugs such as ivermectine, daptomycin, sofosbuvir and chloroquine have been tried in vitro with some success. A natural product, nanchangmycin, derived from bacteria, can inhibit early entry of viruses into the host [36,37]. Zika virus infection leads to neurological complications which can be tackled by using NMDAR (N-Methyl-D-aspirate receptor) blockers viz. Memantine, dizocilpine [38]. As per recent report zika virus patents are treated using traditional Chinese medicine (250 mg xivyanping IV) as antiviral therapy [39].

Prevention

Vaccination

Till the date there is no vaccine have been developed yet. According to a report published by the Institute of Medicine in March 2016, eighteen educational institutions and pharmaceutical corporations are currently developing various types of vaccines for ZIKV such as purified inactivated vaccines, stay-attenuated vaccines, DNA vaccines, and viral vectored vaccines [40]. Some of the vaccines that have been tested in animal studies have shown promising results,

and most of them are still in preclinical phases of testing [41]. In the first half of this year, we anticipate that four vaccines initial phase of clinical trial will begin on clinicaltrials.gov (NCT02840487, NCT02887482, NCT02809443, and NCTOI 967238) and are expected to be completed by the end of the year. Although the timeline to provide an effective vaccine for Zika virus is being condensed, the pathway to licensure of an effective vaccine may take four to five years at a minimum [42].

Vector prevention

Other precautions can be learned from the yellow fever virus. Vector control measures such as using insect repellents and home screens, wearing long-sleeved shirts, sleeping in air-conditioned rooms, and eliminating household trash that breed mosquitoes have been suggested as the most effective preventative measures. The World Health Organization (WHO) recommends DEET (N,N-diethyl-3-methylbenzamide), IR3535 (3-[N-acetyl-N-butyl]-amino propionic acid ethyl ester), or Icaridine (I-piperidinecarvone). acid), 2-(2-hydroxyethyl)-1-methylpropyl ester) [43].

Travel and pregnancy measures

CDC recently recommended avoiding sexual contact with men or women who live in or recently returned from, areas with active Zika virus infection. Women living in the area shall avoid pregnancy. However limited access to contraceptives in infected area may lead to unwanted pregnancies [45, 46].

Cases of zika virus infection in India

India's first reports of Zika virus infection reported in 1950s. In 1954, the Indian National Virology Institute analysed and found 16.8% of its samples positive for Zika virus infection. A first outbreak of Zika virus infection in India was reported in the state Gujarat and Tamil Nadu in 2016-2017. Since then, health organizations have monitored suspected cases with strict surveillance. The incidences of infection were observed in Rajasthan and Madhya Pradesh in October 2018 [47-49]. There were 153 (Male-50, Female-103, Pregnant-63), and 130 (Male-34, Female-96, Pregnant-42) cases in Rajasthan and Madhya Pradesh respectively [53]. In 2017- 2018, a subtype of Asian lineages irresponsible for the outbreak in India. Routes of spread of Zika virus infection are vector and non-vector borne. An *Ades aegypti* mosquito bite leads to vector borne infection, while non-vector borne infections can be transmitted through sexual contact, breast feeding, mother-to-foetus via the placenta, and any kind of blood transfusion. *Ades aegypti* mosquitoes are also carriers of dengue and chikungunya [50,51]. The state of Gujarat and Tamil Nadu tested 18000 mosquitoes in 2017 that later were found to carry Zika virus infection. During the Rajasthan outbreak, Zika virus infection was confirmed by RT-PCR, which on sequencing had similar serological strains as human infection [52]. However, till date there is no report of microcephaly in India. All the reported cases had a common symptom, which is fever with or without rashes [55]. Control of infection could be still challenging in future outbreak.

CONCLUSION

The Zika virus can cause often a mild to moderate fever viral illness that is spread by mosquitoes in most people, but it could have serious repercussions for unborn children and foetuses. As new incidences to become known, management guidelines will be significantly altered. Therefore, it is crucial that those who offer obstetric medical care stay current with the quickly changing informational environment that has so far been associated with this outbreak. Currently, there is a growing problem of Zika virus infection in India that is affecting the healthcare. To prevent further large-scale outbreaks in India, there needs to be continued effort toward vector control and surveillance in the aforementioned states of India.

Conflict of interest:
Authors declare that there is no conflict of interest.

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