Zika Virus - A Mini Review

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Abstract

Zika infection is an arthropod-borne infection originally portrayed in 1947. A few ZIKV flare-ups have been recorded in various nations since. This is for the most part spread by Aedes mosquitoes, and fever, joint agony, red eyes, migraine, and maculopapular rash signs are intently taking after chikungunya and dengue. The results of ZIKV disease remember the chance of microcephaly for pregnant ladies and other intrinsic mind irregularities. This review deals about Zika infection sickness flare-ups including its epidemiological history, transmission pathways, introduction, and determination and counteraction.

Keywords: zika virus, microcephaly, aedes mosquito

Background

During a study on Yellow fever Zika virus (ZIKV) was isolated for the first time by Dick, Kitchen and Haddow in 1947 [1]. They further isolated ZIKV from the Yellow fever causing Aedes africanus Mosquito [2]. First human ZIKV isolates were obtained from Nigeria during 1968. During serological studies humans were reported to have neutralizing antibodies for ZIKV [3].

Introduction

Zika virus (ZIKV) is an emerging vector born Flavivirus that belongs to the "Flaviviridae" family and is characterized by a mild febrile viral disease causative agent transmitted by the bite of an infected Aedes mosquito (female) i.e. Aedes aegypti, and Albopticus Aedes [4]. ZIKV was initially isolated from a feverish Rhesus monkey in Uganda in 1947 [5]. During a study based on serology, the transmission of ZIKV to humans was confirmed in 1952 [6]. In 2007, first epidemic outbreak was reported in Yap state of Micronesia in which 74% people of age group ≥3 years had developed the IgM antibodies and 18% patients had clinical illness. During the outbreak in French Polynesia in 2013 a study done on ZIKV showed its association with Microcephaly [7]. In 2015 first confirmed outbreak of Zika virus in Brazil was reported by the Pan American Health Organization (PAHO) [8]. According to WHO, till early 2016 America along with 23 countries had reported ZIKV transmission. In India, first confirmed case of ZIKV was reported in the State of Rajasthan. ZIKV is closely related to Dengue, Yellow fever and Chikungunya [9] [2].

During pregnancy, ZIKV causes birth defects in new-borns called Microcephaly characterized by condition of having small head and brain damage, along with other congenital defects while in adults it leading to Guillain-Barre syndrome and temporary paralysis further leading to other neurological defects. It is also transmitted by sexual contact with the patient infected with ZIKV, blood transfusion and through the bite of the infected rhesus monkey [10]. ZIKV is an envelope, non-segmented, single stranded positive sense RNA having icosahedral symmetry surrounded by M-membrane, its genome is ~10.8 kilo base, which encodes 3,419 amino acids [11]. The incubation period for ZIKV is 3-14 days. Majority of people don’t develop symptoms and 80% of cases are likely to be asymptomatic. When the symptoms appear, they are
typically low-grade fever (37.4°C-38.0°C), self-limiting and nonspecific. Commonly found symptoms are conjunctivitis, headache, fever, myalgia, arthralgia; rashes are the prominent outcome of maculopapular rashes which spreads through the extremities within the onset of infection (1-4) days [3].

ZIKV is spreading along human-to-human chains of transmission in the Pacific Islands and in South America. In this study it shows the relation between the Asian lineages with NS1 codon adaptation to human housekeeping genes [12]. Currently there is no approved vaccine against ZIKV. Prevent mosquito bite at the time of infected stage. Treatment for ZIKV is sedative and it includes rest and fluid intake. Paracetamol and Acetaminophen are used to reduce the symptoms. Other non-steroidal anti-inflammatory drugs are used to avoid haemorrhagic complications[13]. Aspirin, ibuprofen and naproxen, should be avoided until dengue can be ruled out to reduce the risk of increased bleeding[14]

Classification and Phylogeny

ZIKV full genome ZIKV MR766 prototype strain was completely sequenced in 2007. It is a Flavivirus and encodes E glycoprotein which helps the virus to enter the host and counterbalance the antibodies of other Flavivirus. Positive sense RNA encodes a single polyprotein which splits into 3 structural proteins (C, PrM / M, E) C encodes Capsid, PrM for pre-membrane/membrane and non-structural proteins NS1, NS2A, NS2B, NS3, NS4A, NS4B and NS5 [15]. The protein act as the vital constituent for the virion with which the virion is fixed to the membrane of the host cell (CDC). Virus makes conformational changes after entering the acidic environment of the endosomes thereby encouraging the binding with endoplasmic membrane which causes the release of the viral genome into the cytoplasm. [16]. After the release of viral RNA, it changes into viral proteins leading to the initiation of replication. Immature particles of viral material gathered within the endoplasmic reticulum (ER) are coiled and maturation of the virion particles take place by the Cellular Secretory Pathway. Furin protease cleave the PrM releasing a peptide leading to the formation of mature M protein [12]

During the study three different lineages of ZIKV have been reported i.e. two African lineages MR766 clump and Nigerian clump and one Asian lineage [8]. NS5 and MR766 gene are used for targeting the primers and sequencing respectively [17]

Transmission:

ZIKV is similar to other Flaviviruses in various modes of transmission. ZIKV is transmitted by sylvatic transmission in which 2 sub-genera of Aedes are involve. Likelihood of A.aegypti in causing outbreaks is found less as compared to A. albopictus which is high [18]. ZIKV is frequently transmitted through the bite of an infected mosquito. Transmission of virus from infected mother to foetus through placenta is also known, wherein it leads to congenital defects like Microcephaly leading to neurological effects in foetus. [13].

Furthermore, ZIKV can be transmitted through blood transfusions (Hayes.et, al.2009), through laboratory exposure [18] and by sexual contact with acute ZIKV infected patients [10], [19].
Clinical Manifestation:
ZIKV is reported to be asymptomatic in 80% of the cases. Symptoms resolve within 2 to 3 weeks and are usually mild with low grade fever, retro-orbital pain, headache, non-purulent conjunctivitis, maculopapular rash, vomiting, leg pain, anorexia, lymphadenopathy, stomach ache, dizziness and diarrhoea [9], [18]. Males usually develop symptoms like hematospermia and prostatitis characterized by mild dysuria and perineal pain [20].

Laboratory testing and Diagnosis:
Laboratory findings for ZIKV are limited. Complete blood count and other biochemistry test results are reported to be normal in most of the patients, although some patients report symptoms like neutropenia, leukopenia, thrombocytopenia. [8], [15] and liver markers shows elevation.

Serological diagnosis of ZIKV is confirmed by the presence of antibody IgM in serum. IgM antibodies are detectable generally 3 to 4 days of onset of infection for which Antibody Capture Enzyme-Linked Immunosorbent Assay (Zika MAC-ELISA) is used. However, recent studies reveal the limited cross reactivity of ZIKV IgM sera with other Flavivirus. ZIKV ELISA NS1 is found to be a more specific serological test [14].

Nucleic Acid Test (NAT):
It is based on molecular techniques used to detect viral genomic material and provide the evidence of the infection [7]. NAT holds various well equipped techniques for detection among which real time polymerase chain reaction (RT-PCR) is the golden standard for ZIKV detection with high specificity and sensitivity being effective in serum, saliva and semen in 1-2 weeks post infection (CDC)[2], [4], [12], [14].

Preventive Measure:
Several preventive and control measures are reported by government to check ZIKV spread it includes various factors like:
Preventing bites from mosquitoes by remaining indoors, wearing long-sleeved shirts and trousers [17].
Insect repellents licensed using Environmental Protection Agency [15]. Using nets for mosquitoes. Avoid non-essential travel to countries facing ZIKV infection wrath. Preventing mosquito bites by staying indoors, wearing long-sleeved shirts and long pants [1], [18].
Using Environmental Protection agency registered insect repellents. Use mosquito nets. Avoiding non-essential travel to countries currently facing the wrath of ZIKV infection. [21]. Women of reproductive age living in affected countries were advised not to become pregnant as a result of an rise in cases of microcephaly. [20]. Chemical treatment includes the use of pyrethroids, organochloride and organophosphorus that function on the vector nervous system. [15], [19], [21]

Conclusion
Although the Zika virus is not deadly, the complications associated with it can threaten quality of life, particularly in pregnant women and children. With proper interventions and health management it can be easily avoided. With annual global outbreaks, all health agencies will prepare well before the peak season.
References


