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Zika Virus: Pathogenesis, Diagnosis, Treatment and Prevention

Shankar V. Pandhare[1], Bhimashankar S. Hucche[2], Shyamlila B. Bavage[3], Nandkishor B. Bavage[4]

[1]B. Pharm Final Year Student, Latur College of Pharmacy Hasegaon, Tq. Ausa, Dist. Latur-413512 Maharashtra, India
[2]Department of Pharmaceutics, Latur College of Pharmacy Hasegaon, Tq. Ausa, Dist. Latur-413512 Maharashtra, India
[3]Department of Pharmacognosy, Latur College of Pharmacy Hasegaon, Tq. Ausa, Dist. Latur-413512 Maharashtra, India
[4]Department of Pharmaceutical Chemistry, Latur College of Pharmacy Hasegaon, Tq. Ausa, Dist. Latur413512 Maharashtra, India

ABSTRACT

In 1947, a study on yellow fever revealed the first isolation of a new virus, in the blood of the sentinel rhesus macaque that was implanted in the Zika Forest in Uganda. Zika virus remains hidden for about 70 years; and within a year, the Zika virus was introduced to Brazil from the Pacific islands and spread rapidly throughout the Americas. Zika virus (ZIKV) is an arthropod-borne virus that was first isolated in 1947. Since then, numerous ZIKV outbreaks have been reported in various countries. It is highly transmitted by Aedes mosquitoes, and the symptoms of fever, joint pains, red eyes, headaches, and maculopapular outbreaks are similar to those of chikungunya and dengue. The virus can also be transmitted through body fluids, sex and direct transmission to mothers in the womb. Asymptomatic presentation is common. When symptoms occur, people show mild fever, maculopapular rash, arthralgia, or conjunctivitis 2 to 7 d after infection. The diagnosis should be made in people with appropriate symptomatology and a history of exposure. Zika virus diagnostic tests are available and vary depending on the duration of symptoms. The treatment is supportive, and is recommended for all pregnant women. Previous infections are thought to provide protection against future poses. Prevention and education are key to reducing the spread of disease.

Keywords : Zika Viruse, Zika Infection, Flaviviruse Infection, etc.

Introduction

On March 26, 2015, an Zika Virus (ZKV) outbreak was transmitted by Aedes mosquitoes to a blood sample of 24 patients in a small town in Bahia, Brazil. The spread of the virus quickly became a global epidemic of anxiety. The epidemic in Brazil alone is estimated at 440 million to 1.3 million. The 2013-2014 ZKV outbreak in French Polynesia has been the largest outbreak to date. Experts believe the virus was introduced in Brazil during the 2014 World Cup soccer tournament, where the four Pacific states (New Caledonia, French Polynesia, Easter Island, and the Cook Islands) where ZKV broadcast in 2014 had teams that participated in the tournament. [1] Zika virus has recently caused serious concern around the world. Recent emergencehave become a major challenge due to the shift from its previous treatment features to now visible neurological problems. The Zika virus (ZIKV) was first isolated from Dick, Kishi, and Hadadow in 1947 in albino mice using the blood of rhesus monkey found in Uganda's Zika forest. It is transmitted by the Aedes mosquito. In 1952, human infection was indicated by the presence of an antibody, and in 1964, the virus was isolated from people in Uganda. Thereafter, until the beginning of 2007, the spread of Zika remained restricted to Africa and Asia. [2]

The challenge of diagnosing ZVD in resource-intensive conditions, especially in Africa, is enormous. ZIKV exhibits mild to moderate severity similar to the most common febrile illness in tropical areas. The diagnosis of ZVD by reverse transcription polymerase chain reaction (RT PCR) should be interpreted with caution based on the sample used and the time of the initial symptoms and the date of collection of the sample. Bingham et al. it was found that only 56% of Zika RT PCR were separated from serum samples within the first five days of symptoms compared to 95% of urine samples collected on the same day. Therefore, incorrect PCR does not determine Zika infection. [2]

ZIKV is most commonly transmitted by female mosquitoes Aedes aegypti and Aedes albopictus. The latter is widespread in Southeast Asia and, thanks to its adaptability, has been found in the Americas, the Pacific Islands, Australia, and Africa. A. albopictus is a potential vector in more than 20 arboviruses, including key members of the Flaviviridae and Togaviridae families such as Dengue (DENV) and Chikungunya (CHIKV), respectively. Both species may be present in the same regions. Zika fever is considered a minor illness with symptoms such as headache, fever, pain and joint pain. However, since its revival, ZIKV has been linked to an increasing number of cases of microcephaly, Guillain-Barré syndrome, meningoencephalitis, and myelitis in the most affected areas such as the Brazilian North Eastern State. [3] Zika virus (ZIKV) shares genus flavivirus with West Nile virus (WNV), yellow fever virus (YFV), dengue virus (DENV), and Japanese encephalitis virus, all of which contain a single polyprotein (þ) ssRNA genome that is able to replicate quickly as cell enters. Codes of a single open reading framework for the three genes and seven non-genetics are combined with viral and host genes, while non-translated regions ultimately contribute to duplication, tolerance, and protection of viruses by secondary structure and sub-genomic flavivirus RNA production. [4]

The Zika virus is a single arthropod carrying Ribonucleic acid (RNA) carried by arthropod that is transmitted primarily by the bite of the female Aedes mosquito. Although several species of Aedes subgenus have been linked to the Zika virus outbreak, Aedes aegypti and Aedes albopictus are thought to be the main carriers. Fetal effects associated with Zika infection in mothers can damage and include microcephaly, ocular manifestations, such as loss of foveal reflex, macular neuroretinal atrophy, lens subluxation, and iris coloboma, loss of early pregnancy, and death for a child. There have also been reports of Guillain-Barre syndrome associated with Zika virus. [5]

Pathogenesis

ZIKV is an enveloped, icosahedral, non segmented, sense positive, single stranded RNA virus. It is 40 nm wide with an outer envelope (E) and a thick inner core. Its genome consists of a single module of a single RNA encrypted 10794 kb in length with 2 uninterrupted shortcuts (5 'and 3' NCR) and one long open reading frame incorporating polyprotein: 5 'C prM E NS1 N S2A NS2B NS3 NS4A NS4B NS5 3 composed of capsid (C), which precedes the membrane, envelope and seven non-protein proteins (NS). Glycoprotein coverage is ZIKV's main antigenic compound. Zika belongs to the family of flaviviridae and phylogenetic analysis has shown that it has three lines: West Africa (Nigerian group), East Africa and Asia. In 2016, the gene sequence that is part of ZIKV E was performed on a specific ZIKV RT PCR in North Central Nigeria (GenBank entry number MF926508 / Nigeria / 2016), this is the fifth ZIKV to report data sequences from Africa to date. [10] Phylogenetic analysis revealed that it was a descendant of West African descent. ZIKV can be transmitted in two main ways: Vector to person and person-to-person. Human transmission of ZIKV is entirely possible through the following procedures: By transplacental route, leakage of the virus by trophophlastic plug, transmission of the virus to the amniotic sac during fertilization, or during childbirth by an infected mother to her newborn baby. In addition, human transfusions have been reported in a sexual way and there have been reports of transfusions in Brazil. Sexual transmission occurs when a person is strongly infected with Zika, this is especially important when the exposed partner is pregnant due to possible neurological effects on the fetus. ZIKV is a neurotropic; crosses the blood-brain barrier to the fetus and attaches itself to neuronal cells in the brain. The RNA virus binds its genome to neuronal cells, causing apoptosis and ultimately leading to disruption of neuronal growth, neuronal proliferation and migration. [2]

ZIKV infection neuropathology is clearly demonstrated in the Asian genus. However, microcephaly was not detected following the Zika infection of Africa. Interestingly, in vitro studies have confirmed a significant increase in African stress through high viral replication and cell lysis in both neuronal and non-neuronal cells. More than 30 laboratory-based studies have suggested that African Zika species are capable of producing the same, or worse, damage to cells of the central nervous system (CNS), as well as reproductive and immune systems such as Asian problems circulating in the United States. [2]

Although there is no cure for ZIKV, in most cases, only 20% of patients who present clinical symptoms will be resolved within 2 to 7 days, and rarely require hospitalization (Peteren et al. 2016; Saaib et al. 2016). The clinical manifestations of ZIKV infection have been shown to be similar to other mosquito-borne diseases such as Chikungunya (CHIKV) and Dengue fever virus (DENV), but with much lower mortality than DENV (Fox 2015). In addition, ZIKV-related symptoms are usually mild and short-lived, and manifest with a mild fever of about 98.9 ° F (37.2 ° C) and a combination of one of the following symptoms: conjunctivitis, macular or popular rash, arthralgia, myalgia, headache, retro-orbital pain, edema, vomiting and weakness. [6]

ZKV is a mosquito-borne flavivirus associated with dengue virus, yellow fever virus, and West Nile virus. ZKV is a single-membered RNA virus (10,794-nt genome), closely related to the Spin virus and is transmitted by several Aedes mosquitoes, including Aedes. africanus, Aedes. hensilli, Aedes. luteocephalus, Aedes. Aegypti, etc. ZKV was first detected in rhesus monkeys in 1947 during a syphilis yellow fever in Uganda's Zika Forest, and was reported in 1952. In two well-known lines of ZKV (Africa and Asia), Phylogenetic studies show that the closest form of ZKV to that, which originated in Brazil, was isolated from samples taken from French Polynesia and spread among the Pacific Islands, and that of Asian descent. [1]

Zika virus usually follows the sylvatic transmission cycle. It is classified as an arbovirus that is transmitted from one vertebrate to another by mosquito bites. Virions exist such as immature (non-infectious), mature (infectious), and fusogenic (host membrane binding). Humans are not responsible for what happens in the lifetime of virions. Monkeys and chimpanzees can also act as pathogens, and some studies have found that sheep, elephants, and goats have antibodies against Zika, suggesting that potential habitats can also be established. The cycle begins when the Aedes mosquito sucks blood containing the Zika virus after biting an infected person. The virus begins to replicate in the epithelial cells of the midgut and then travels to the inflamed parts of the mosquito. After a 10-day incubation period, saliva becomes infected with the virus, making the mosquito a vector of infection. When it enters a person's skin, the virus infects dermal fibroblasts that act as receptors for the Zika virus. [7]

Diagnosis

There are two types of ZIKV diagnoses. The first type involves detection of the virus and / or parts of the virus. RT-PCR, immunoassay, and antibodies were developed to detect ZIKV RNA, viral proteins (especially NS1), and live viruses, respectively. These methods are used for mosquito monitoring and the diagnosis of the patient's species. Among them, RT-PCR is the most popular test due to its sensitivity and specificity, while viral segregation remains a gold standard but requires additional cell culture laboratory infrastructure. Because up to 80% of infected people have no symptoms, blood donors, blood bank samples, and transplant organs may sometimes need to be tested for ZIKV infection or contamination. [8]

The prevalence of ZIKV infection symptoms prompts laboratory testing. Serologic test results should be interpreted with caution because there may be recurrence of other flaviviruses, including people vaccinated against yellow fever or Japanese encephalitis. Infections with flaviviruses such as Yellow fever and Dengue fever are common in Africa and the yellow fever vaccine makes recurrence a major problem in explaining the test results in these settings. Current algorithms suggest a combination of IgM tests followed by plaque-reduction neutralization tests (PRNTs) in cases of positive or equitable positive diagnostic results. If the results of the ZIKV IgM test are positive, equivocal or inconsistent, testing for neutralization antibodies using PRNT should be done to determine whether ZIKV IgM shows a recent ZIKV infection or a false positive result. PRNT titre> 10 should be translated as proof of recent infection with ZIKV when PRNT goes to another

flaviviruses tested by <10. The gold standard for ZIKV infection diagnosis is based on the detection of RNA virus from a variety of clinics. Direct diagnosis is only possible within the first 3-5 days after the onset of symptoms. Salivas and urine types to detect the virus genome by RT PCR can be a good diagnostic example. Bingham et al. it was found that only 56% of Zika antigens were isolated from serum samples within five days of onset of symptoms compared to 95% urine samples collected on the same day. It is important to use PCR trials identifying both the Asian and African ZIKV lines identifying the saved genetic regions of the envelope or the NS5 region. This is to prevent false positives. It is also important to note that the actual results of the reverse transcriptase polymerase chain reaction (rRT PCR) do not exclude Zika infection due to the late viraemia and inaccuracy in reporting the onset of Zika symptoms. [2]

The key to a typical diagnosis of Zika virus infection is to detect viral nucleic acid by RT-PCR and to detect IgM antibodies via the IgM-capture enzyme-linked immunosorbent assay (MAC-ELISA). The detection of viral nucleic acid in serum provides a clear diagnosis; however, in most cases viremia passes, and the RT-PCR diagnosis was most successful within 1 week after the onset of clinical illness. In contrast, viral RNA is found in serum about ten weeks after infection in a pregnant woman whose fetal name is evidence of congenital infection. In addition, viremia is usually low, which makes it difficult to distinguish viruses from clinical samples. Although the exact time and duration of the IgM antibody response to the Zika virus found by MAC-ELISA has not been determined, extensive information on other related flaviviruses indicates that IgM will appear as viremia wanes during the first week after the onset of the disease and will continue for a few months. . Therefore, RT-PCR testing of serum samples obtained during the first week of clinical illness and MAC-ELISA testing of samples that could be tested by RT-PCR or found to be defective by RT-PCR may have a much higher diagnosis. [9]

Treatment and Prevention

No special treatment is available for ZKV. Supportive care includes relaxation, antipyretics, analgesics, and watching coagulopathy or multiple organ failure are important care goals. Antihistamines can be considered for marked symptoms. Intravenous fluids, oxygen (as needed), and monitoring of vital signals are measures of care. Given the similarity of the signs and distribution of areas, suspected cases of ZKV should be tested and controlled for possible dengue or chikungunya virus infection. Non-steroidal anti-inflammatory drugs (NSAIDs) and aspirin should be delayed until dengue can be removed to reduce the risk of bleeding. [1]

Like other mosquito-borne flaviviruses, the treatment of Zika virus infection focuses on these symptoms. There is no Zika virus vaccine available; therefore, prevention and control measures focus on preventing mosquito bites, reducing sexually transmitted infections, and controlling the mosquito vector. Potential preventive measures aimed at reducing the risk of infection in pregnant women include avoiding unnecessary travel to ongoing Zika infection sites, avoiding unprotected sexual contact with partners at risk of Zika infection and using mosquito repellent, permethrin treatment, bed nets, nets.

window screens and cool air. A very efficient A controller. Egypt relies on an integrated process that includes the elimination of A-mosquito breeding sites. aegypti, the use of worms, and the use of pesticides to kill adult mosquitoes. However, each of these methods has its limitations. Communities are often mobilized to reduce A-breeding areas. Aegypti, however, this strategy often fails, in part because of incompatible participation between families and the existence of secret breeding grounds in modern urban areas. [9]

Zika presents without symptoms or with minor symptoms, symptoms can be controlled with bed rest, intravenous fluids and acetaminophen. The biggest challenge for Zika is the problem of infection, efforts should be made to antiviral agents, vaccines and other means of prevention. About 30 approved antiviral agents have been tested and found to have anti-Zika viral properties. However, the anti-bacterial effects were largely determined using Asian pressures. It is therefore important to get this anti-bacterial activity in Africa. [2]

Common methods of prevention include preventing mosquito bites and preventing sexually transmitted infections. Methods to prevent mosquito bites include: wearing long sleeves and long sleeves, permethrin-infested clothing, insecticide-treated indoor spraying, door and window inspections for mosquitoes and other environmental control measures aimed at reducing or eliminating mosquito breeding. Preventing sexual transmission is very important when a sexual partner is pregnant. Sexual transmission can be prevented by contraception or by using a condom. Because of transfusion-related blood transfusions, it is necessary to provide affordable blood transfusion services. This could include pre-delivery testing to prevent potential Zika infection and to call for active Zika serological testing with high sensitivity. This is especially important for pregnant women who need a blood transfusion. There are several vaccines used in various stages of development, some of which are lively, nonexistent and some are genetically engineered vaccines. Among these vaccines, there is one that targets both Dengue and ZIKV. It will be very helpful to develop a flavivirus vaccine that can be used in limited settings for infections with high and high flavivirus infections. [2]

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