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Popular Article

ZIKA VIRUS: An Emerging Zoonotic Disease

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Abstract

Zika Virus Disease (ZVD) is a mosquito-borne (Aedes) viral disease caused by Zika virus (ZIKV). The clinical presentation of Zika fever is nonspecific and can be misdiagnosed as other infectious diseases, especially those due to arboviruses such as dengue and chikungunya. ZIKV infection was associated with only mild illness prior to the large French Polynesian outbreak in 2013 and 2014, when severe neurological complications were reported, and the emergence in Brazil of a dramatic increase in severe congenital malformations (microcephaly) suspected to be associated with ZIKV. Laboratory diagnosis of Zika fever relies on virus isolation or detection of ZIKV specific RNA. Serological diagnosis is complicated by cross reactivity among members of the Flavivirus genus. There is a high potential for ZIKV emergence in urban centers in the tropics that are infested with competent mosquito vectors such as *Aedes aegypti* and *Aedes albopictus*.

Introduction

Zika virus (ZIKV) is an arthropod-borne virus (arbovirus) in the genus Flavivirus and the family Flaviviridae. ZIKV is usually transmitted by the bite of infected mosquitoes. It presents as mild fever, rash (mostly maculopapular), headaches, arthralgia, myalgia, asthenia, and non-purulent conjunctivitis, occurring about two to seven days after the mosquito bite. One out of five people may develop symptoms, but in those who are affected the disease is usually mild and may last between two and seven days.

Its clinical manifestation is often similar to dengue, also caused by the same vector. The adaptation of ZIKV to an urban cycle involving humans and domestic mosquito vectors in tropical areas where dengue is endemic suggests that the incidence of ZIKV infections may be underestimated.

Epidemiology

ZIKV was first isolated from a nonhuman primate in 1947 and from mosquitoes in 1948 in Africa, and ZIKV infections in humans were sporadic for half a century before emerging in the Pacific and the Americas. The virus was first identified in a rhesus monkey in the tropical Zika Forest in Uganda in April 1947 by the scientists of the Yellow Fever Research Institute. The first human Zika Fever infection was identified in Nigeria in 1954. Until 1981, evidence of human infection with Zika virus was reported from other African countries, such as the Central African Republic, Egypt, Gabon, Sierra Leone, Tanzania, and Uganda, as well as in parts of Asia including India, Indonesia, Malaysia, the Philippines, Thailand, and Vietnam.

Reservoir of infection: Unknown.

Immunity: Once a person has been infected, he or she is likely to be protected lifelong.

Vector: Zika virus is transmitted primarily by *Aedes aegypti* mosquito. *Aedes albopictus* mosquito also might transmit the virus. *Aedes hensilli* was the predominant vector species identified in the Yap outbreak.

About the vector: Aedes mosquito is common vector to transmit the viruses that cause dengue, chikungunya, West Nile fever and ZVD. Aedes mosquito can be recognized by white markings on its legs and a marking in the form of a lyre on the upper surface of the thorax. An infective female Aedes mosquito acquires the virus while feeding on the blood of an infected person and then viruses are passed on to healthy humans through its bites. The density of Aedes mosquito is more during monsoon and post-monsoon season. Female Aedes mosquito is a day biter and is most active during daylight, for approximately two hours after sunrise and several hours before sunset. It bites many times to complete full blood meal, therefore it is known as indiscriminate feeder. The male mosquito does not bite humans or animals. Aedes prefers to breed in man-made containers, viz., water storage containers, coolers, discarded buckets and plastic containers, bottles, tyres and coconuts shells, etc. in which water stagnates for more than a week. It prefers to lay its eggs singly on damp surfaces just above the water line, in clean water which contains no other living species. In unfavourable conditions, the eggs can be viable for over a year in dry state, which allows the mosquito to re-emerge after winter or dry spell. The life cycle lasts for 8-10 days in favourable conditions. *Aedes aegypti* rests indoor, in dark corners of the houses, on dark cloths, umbrellas, under furniture- beds, shelves, coolers, behind hanging, shoes, besides household articles, curtains etc. but rarely on walls. Outside it rests in cool and shaded places. The flight range for Aedes is generally 100 metres but it can fly up to 400 metres.

Mode of Transmission

Zika virus is primarily transmitted by the bite of an infected mosquito from the *Aedes* genus, mainly *Aedes aegypti*, in tropical and subtropical regions. *Aedes* mosquitoes usually bite during the day, peaking during early morning and late afternoon/evening. This is the same mosquito that transmits dengue, chikungunya and yellow fever. Zika virus is also transmitted from mother to fetus during pregnancy, through sexual contact, transfusion of blood and blood products, and organ transplantation.

Incubation period:

The incubation period (the time from exposure to symptoms) of Zika virus disease is estimated to be 3–14 days. The majority of people infected with Zika virus do not develop symptoms. Symptoms are generally mild including fever, rash, conjunctivitis, muscle and joint pain, malaise, and headache, and usually last for 2–7 days.

Clinical Features

An estimated 80% of persons who are infected with Zika virus are asymptomatic. In the majority of cases, ZVD is a self-limiting disease. The symptoms are similar to other arbovirus infections such as dengue, and include fever, skin rashes, conjunctivitis, muscle and joint pain, malaise, and headache. These symptoms are usually mild and last for 2-7 days. Other reported symptoms are dizziness, oedema of the extremities, retro-orbital pain, anorexia, photophobia, gastro-intestinal disorders, sore throat, cough, aphthous ulcers, back pain, sweating and lymphadenopathies. None of these symptoms are specific and ZVD can be misdiagnosed with other bacterial and viral infections, especially with other arboviruses in endemic areas.

Guidelines for sample collection:

During the first week after onset of symptoms, ZVD can often be diagnosed by performing reverse transcriptase-polymerase chain reaction (RT-PCR) on serum by both conventional and real time methods. Virus-specific IgM and neutralizing antibodies typically develop toward the end of the first week of illness; cross-reaction with related flaviviruses (e.g., dengue and yellow fever viruses) is common and may be difficult to discern. Plaque-reduction neutralization testing can be performed to measure virus-specific neutralizing antibodies and discriminate between cross-reacting antibodies in primary flavivirus infections. Viral isolation is not regarded as a diagnostic tool and is recommended only for supplemental research studies in public health surveillance. Pan American Health Organization (PAHO) has issued interim guidance for laboratory detection and diagnosis of ZVD surveillance in the Americas. An algorithm has been developed for detection of introduction of the virus into a specific area. This algorithm is addressed to reference laboratories with established capacity (molecular/antigenic and serological) to detect dengue, chikungunya), and Zika viruses. A BSL2 containment level is required to handle suspected samples. These recommendations are subject to modifications that take into account advances in knowledge of the disease and the etiologic agent.

Laboratory Diagnosis

Sample storage

- Blood (5ml in plain vial) of suspect case.
- Keep refrigerated (2-8 degree C) if it is to be processed (or sent to a reference laboratory) within 48 hours.
- Keep frozen (-10 to -20 degree C) if it is to be processed after the first 48 hours or within 7 days.
- Keep frozen (-70 degree C) if it is to be processed after a week. The sample can be preserved for extended periods.

Transportation of the sample to the reference laboratory

Always use triple layer packaging and ship within 48 hours of collection under cold chain (dry ice or at least with cooling gels). The original samples should be packed, labelled and marked. Always include the completely filled out clinical and epidemiological record.

Molecular diagnosis:

Blood (5ml in plain vial) of suspect case (see previous section for case definition) should be collected during first five days of illness and sent to the laboratory for RT PCR with prior intimation. ZIKV RNA also has been detected in urine over an extended period in the acute phase, which means that could be an alternative sample to be considered. However, and since more studies are needed, it is recommended that the serum sample be taken during the first 5 days after the onset of symptoms for RT PCR.

Serological diagnosis:

As on date, this has not been standardized in India. However, the diagnosis can be undertaken by detecting ZIKV-specific IgM antibodies by ELISA or immunofluorescence assays in serum specimens from day 5 after the onset of symptoms. Since a single serum in the acute phase is presumptive, it is recommended that a second sample be taken 1–2 weeks after the first sample to demonstrate seroconversion (negative to positive) or a fourfold increase on the antibody titter (with a quantitative test). The interpretation of the serological tests is especially important for the diagnosis of ZIKV. In primary infections (first infection with a flavivirus) it has been demonstrated that antibodies cross-reaction is minimal with other genetically related viruses. However, it has been demonstrated that sera of individuals with a previous history of infection from other flaviviruses (especially dengue, yellow fever and West Nile) can cross-react in these tests. As on date no commercial kits have been approved or validated for the serological determination of ZIKV.

Treatment

ZVD is usually relatively mild and requires no specific treatment. People sick with Zika virus should get plenty of rest, drink plenty of fluids, Receive symptomatic treatment with acetaminophen (paracetamol) for pain and fever and antihistaminic for pruritic rash. If symptoms worsen, they should seek medical care and advice.

Prevention And Control

The key to control ZVD is adoption of a comprehensive approach by way of regular vector surveillance and integrated management of the Aedes mosquitoes through biological and chemical control that are safe, cost effective; and environmental management, legislations as well as action at household and community levels.

Integrated vector management:

Vector Surveillance Several indices have been described and are currently used to monitor Aedes population.

Larval surveys: Water holding containers are examined for the presence of mosquito larvae and pupae in the house or premises. Indices commonly used to monitor Aedes are: House index (HI), Container Index (CI), Breteau Index (BI) and Pupae Index (PI).

Adult Surveys are done by Landing/biting collection, resting collection, Oviposition traps.

Environmental Management

- Environmental modification: Long lasting physical transformation of vector habitats. Improved water supply, mosquito proofing of overhead tanks, cisterns or underground reservoirs.

- Environmental manipulation: Temporary changes to vector habitats as a result of planned activity to produce conditions unfavourable to vector breeding.

Complications of Zika virus disease

Zika virus infection during pregnancy is a cause of microcephaly and other congenital abnormalities in the developing foetus and new-born. Zika infection in pregnancy also results in pregnancy complications such as fatal loss, stillbirth, and preterm birth.

Zika virus infection is also a trigger of Guillain-Barré syndrome, neuropathy and myelitis, particularly in adults and older children. Research is ongoing to investigate the effects of Zika virus infection on pregnancy outcomes, strategies for prevention and control, and effects of infection on other neurological disorders in children and adults.