FACT SHEETS ON ZIKA VIRUS DISEASE EPIDEMIOLOGY

Deepali Danave1, Vanita Kulkarni2

1Associate Professor, Department of Microbiology, RCSM Government Medical College, Kolhapur.
2Professor and HOD, Department of Microbiology, RCSM Government Medical College, Kolhapur.

ABSTRACT

OBJECTIVES
Zika virus (ZIKV) disease was a neglected entity with only 14 documented cases worldwide up to 2006. The first major outbreak in Yap Islands, Micronesia changed this. It has spread to African and American countries since then causing major outbreaks. It has also been temporally associated with neurological syndromes and congenital anomalies. This study is an attempt at documenting the facts related to ZIKV.

MATERIALS AND METHODS
This study is a retrospective analysis. Possible documented sources of ZIKV articles and internet have been scanned. Conclusions have then been drawn from it.

RESULTS
ZIKV disease has spread gradually to encompass African and American countries resulting into a pandemic. Also its temporal and spatial link with neurological syndromes and congenital anomalies has been documented.

CONCLUSIONS
From being a neglected subject Zika virus has evolved into a slow pandemic. Apart from its dengue like presentation, its temporal association with neurological syndromes and congenital anomalies have raised concern worldwide.

KEYWORDS
Zika virus, Neurological Syndromes.


INTRODUCTION
Zika virus (ZIKV) disease is caused by Zika virus which belongs to the family of arboviruses which include other viruses as Yellow fever virus, West Nile virus, Dengue virus, Chikungunya virus and Tick-borne encephalitis virus. It is transmitted to people through the bite of an infected mosquito from the Aedes genus, mainly Aedes aegypti, which usually bite during the morning and late afternoon hours. Transmission from an infected pregnant mother to her baby during pregnancy or around the time of birth has also been implicated.[1]

The classic clinical presentation resembles dengue fever but also chikungunya: a fever accompanied by polyarthralgia, myalgia, maculopapular rash and headache. These symptoms are usually mild and last for 2-7 days. Only one out of four infected people develop symptoms of the disease. Zika virus is diagnosed through PCR (Polymerase chain reaction) and virus isolation from blood samples.[2] Diagnosis by serology is not recommended as the virus can cross-react with other arboviruses.[2] Zika virus has been named after the Zika region of Uganda where it was first isolated from a Macaca monkey.[3]

The first human case was detected in Nigeria in 1954.[4] Zika was an insignificant and neglected pathogen. This situation changed in the 21st century with large outbreaks in French Polynesia and Brazil in 2013 and 2015 respectively.[1] Outbreaks of the virus have been recorded in Africa, the Americas, Asia and the Pacific.[1] The clinical presentation of ZIKV disease is acute but self-limiting. However microcephaly in the new born and other neurological syndromes (Guillain Barre syndrome i.e. GBS) have been found temporally associated with Zika virus infection.[1]

We began this study to analyse the current situation of ZIKV disease in the world and its implications in various settings.

MATERIALS AND METHODS
This work is a retrospective analysis of the various studies available in relation to ZIKV. The information available through different sources as internet and journal articles has been compiled together and inferences have been drawn through the available information. All the data and figures mentioned in this study are as per those quoted in references.

RESULTS
Table 1 shows the geographical spread of ZIKV across various continents. Table 2 shows the distribution of documented cases on record. Table 3 shows the number of cases with neurological manifestations and congenital anomalies having history of prior exposure to ZIKV.
DISCUSSION

Since only 14 documented human cases of ZIKV disease existed up to 2006 Zika virus has been a neglected entity. The situation changed drastically in the twenty first century with large scale outbreaks in Pacific islands beginning on Yap in Micronesia in 2007. The geographical spread of the virus from 1950s till date has been elaborated in Table 1. Phylogenetic analysis of ZIKV reveals two main lineages based on sequencing of non-structural protein gene (NS5)-African lineage, Asian lineage. Twenty one full length Zika virus genomes are currently available in Gen Bank and nine of those have collection date information in their Gen Bank record. The evolution of the ZIKV disease into a slow pandemic from 2007 onwards with a staggering increase in the number of suspected or recorded cases has been shown in Table 2.

The clinical presentation of the viral infection is acute and self-limiting. But the increase of congenital anomalies, Guillain-Barre syndrome (GBS) and other neurological and auto-immune syndromes in areas where Zika virus is circulating have pointed out its far fetched implications. During the Zika virus outbreak in French Polynesia, 74 patients had presented with neurological syndromes or autoimmune syndromes after the manifestation of symptoms consistent with Zika virus infection. Of these 42 (55%) were classified as GBS. In July 2015 Brazil reported detection of 76 patients with neurological syndromes of which 42 were confirmed as GBS. Among the confirmed GBS, 26 (62%) had a history of symptoms consistent with Zika virus infection. In January 2016, El Salvador reported an unusual increase of GBS since early December 2015 which was 46 cases when on an average they had 14 cases of GBS recorded per month.

In October 2015, Brazil recorded an unusual increase in microcephaly cases in Northeast Brazil, with 3530 cases recorded in 20 states. In January 2016, ophthalmological findings were reported in three children with microcephaly and cerebral calcifications detected by CT scan and presumable intrauterine ZIKV infection. On 13th January 2016, the Brazil Ministry of Health reported the detection of Zika virus genome, through the RT-PCR technique in four cases of congenital malformation in the state of Rio Grande do Norte. All these neurological syndromes and congenital anomalies have been illustrated in Table 3.

All the above incidences indicate that currently there is only ecological evidence of an association between increased microcephaly, neurological and auto-immune syndromes and prior infection with Zika virus. These findings are consistent with a temporal and spatial link between Zika virus circulation and increase of GBS. Apart from intrauterine transmission strong possibility exists of sexual transmission.

There is no vaccine or drug available to prevent/treat Zika virus disease at present. Based on available information World Health Organization (WHO) has declared Zika virus disease to be a Public Health Emergency of International concern (PHEIC) on 1st February 2016. Pan American Health Organization/WHO reinforces the recommendations relating to the Zika virus surveillance, including monitoring neurological syndromes and congenital anomalies.

As of now the disease has not been reported in India. However, the mosquito that transmits Zika virus namely Aedes aegypti is widely prevalent in India. Ministry of Health and Family Welfare, Government of India has also issued advisory, enhanced surveillance alerts regarding Zika virus.

CONCLUSIONS

From being a neglected subject Zika virus has evolved into a slow pandemic. Apart from its dengue like presentation, its temporal association with neurological syndromes and congenital anomalies have raised concern worldwide.

REFERENCES


