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CHIKUNGUNYA: THE POSITIVE STRANDED RNA VIRUS FROM AEDES SPECIES

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ABSTRACT
Chikungunya fever is a viral disease transmitted to humans by the bite of infected mosquitoes. The virus is a member of the genus Alphavirus, in the family Togaviridae. Chikungunya fever is a viral disease transmitted to humans by the bite of infected mosquitoes. Chikungunya virus is a member of the genus Alphavirus, in the family Togaviridae. The fever is diagnosed based on symptoms, physical findings (e.g., joint swelling), laboratory testing, and the possibility of exposure to infected mosquitoes. There is no specific treatment for chikungunya fever; care is based on symptoms. Chikungunya infection is not usually fatal. Steps to prevent infection with chikungunya virus include use of insect repellent, protective clothing, and staying in areas with screens. Chikungunya virus was first isolated from the blood of a febrile patient in Tanzania in 1953, and has since been cited as the cause of numerous human epidemics in many areas of Africa and Asia and most recently in limited areas of Europe. Chikungunya virus (CHI-V), a mosquito-borne alphavirus, has become an important re-emerging pathogen with its rapid spread to many non-endemic areas. It is an acute viral disease characterized by fever and painful arthralgia. The arthritic symptoms associated with chikungunya can be debilitating and may persist for months or even years in some patients. The virus is transmitted by Aedes mosquitoes. The infection is highly symptomatic, with fever, skin rash and incapacitating arthralgia, which can evolve to chronic arthritis and rheumatism in elderly patients. Chikungunya is endemic throughout Africa, and over the past decade, it has also spread throughout the Indian Ocean, Asia, the South Pacific, southern Europe, the Caribbean and Central America. The rapid emergence of CHIKV has been linked to expansion of the mosquito vector species, Aedes aegypti and Ae. albopictus, throughout most tropical and subtropical regions of the world. Furthermore, mutations in some strains of CHIKV have been associated with increased transmissibility of the virus. In, 2006 an estimated 1.38 million people across southern and central India developed symptomatic disease. The disease is self-limiting febrile illness and treatment is symptomatic. As no effective vaccine and antiviral drugs are available, mosquito controlled by evidence-based intervention is the most appropriate strategy to contain the epidemic for future outbreaks.

KEYWORDS: Alpha virus, Aedes mosquito, Endemic, Nucleocapsid, Genome, Virion, Outbreak, Epidemiology.

INTRODUCTION
Chikungunya is a temporarily debilitating disease caused by **Alpha virus** and spread through mosquitoes. The disease is transmitted predominantly by *Aedes aegypti* and *Aedes albopictus* mosquitoes, the same species involved in the transmission of dengue. Traditionally, CHIKV epidemics have shown cyclical trends, with inter-epidemic periods ranging from 4 to 30 years. Since 2004, CHIKV has expanded its geographical range, causing sustained epidemics of unprecedented magnitude in Asia and Africa. Although areas in Asia and Africa are considered to be endemic for the disease, the virus produced outbreaks in many new territories in the Indian Ocean islands and in Italy.[1]

This recent reemergence of CHIKV has heightened the world's public health awareness and concern about this virus. Between 2006 and 2010, 106 laboratory-confirmed or probable cases of CHIKV were detected among travelers returning to the United States, compared to only 3 cases reported between 1995 and 2005. There also have been nine imported CHIK cases reported in the French territories in the Americas since 2006-three in Martinique, three in Guadeloupe, and three in Guyana. To date, none of the travel-related cases have led to local transmission, but these cases document an ongoing risk for the introduction and possible sustained transmission of CHIKV in the Americas. In 2014 more than a million suspected cases occurred. In 2014 it was occurring in Florida in the continental United States but as of 2016 there were no further locally acquired cases.

Chikungunya virus is a small (about 60–70 nm-diameter), spherical, enveloped, positive-strand RNA virus. (Figure-1).[2]

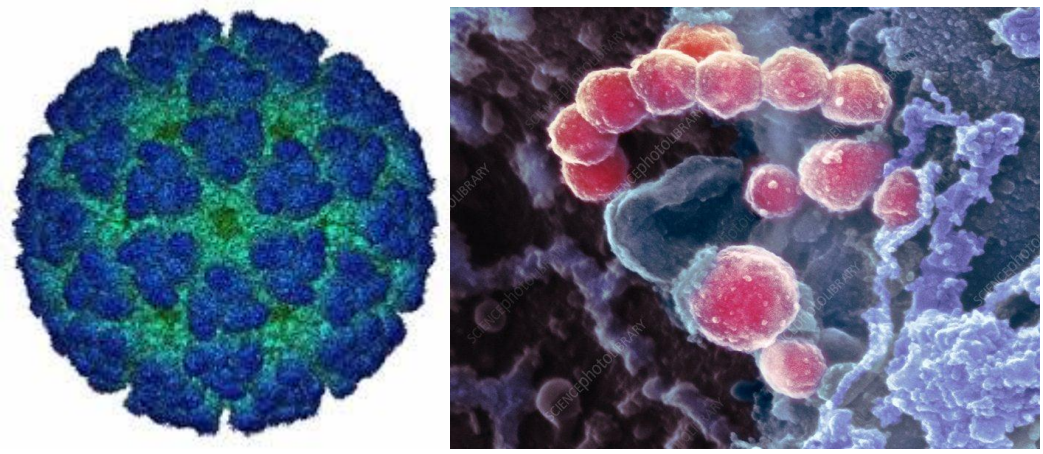

Figure 1: Chikungunya virus particle and electron microscopic picture of chikungunya viruses.

Chikungunya virology: The virion consists of an envelope and a nucleocapsid. The chikungunya virus genome is 11,805 nucleotides long and encodes for two polyproteins – the non-structural polyprotein consisting of four proteins (nsP1, nsP2, nsP3 and nsP4) and the structural polyprotein consisting of five proteins (Capsid, E3, E2, 6K and E1) (Figure-2).

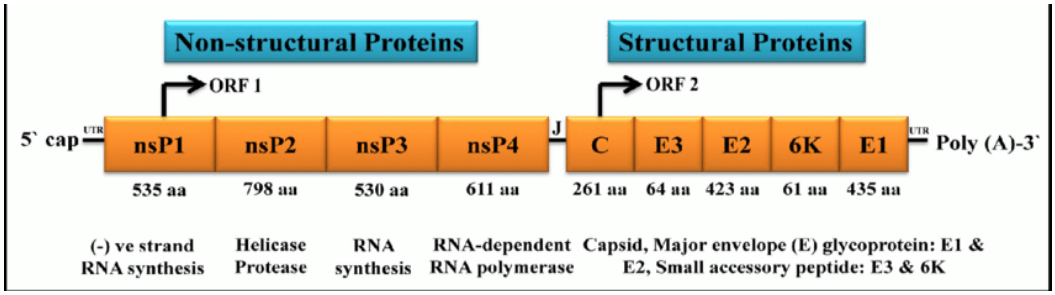

Figure 2: Chikungunya virus genome structure.

The 5' end of the RNA molecule is capped with a 7-methylguanosine while the 3' end is poly-adenylated. A subgenomic positive-strand RNA referred to as 26SRNA is transcribed from a negative-stranded RNA intermediate. This RNA serves as the mRNA for the synthesis of the viral structural proteins. Alphaviruses have conserved domains that play an important role in the regulation of viral RNA synthesis. These domains are found at the 5' and 3' ends as well as at the intergenic region. The E1 and E2 glycoproteins are expected to form heterodimers that associate as trimeric spikes on the viral surface covering the surface evenly. The envelope glycoproteins play a role in attachment to cells. Virions located on the surface of the cell membrane enter the host cells by fusion and endocytosis of the viral envelope. The uncoating of the virions occurs in the cytoplasm. The site of mRNA transcription is in the cell cytoplasm. Replication is not restricted to a particular tissue or organ of the host so the virus replication occurs in various organs. The insect host initiates the virus replication. The genome replication is done in the cytoplasm (Figure-3).

Steps should be taken now to put in place the necessary measures that will decrease the impact that this new arbovirus could have in our Region.[3]

Background: Chikungunya fever is a self-remitting febrile viral illness that has been associated with frequent outbreaks in tropical countries of Africa and Southeast Asia, where the virus has been found to circulate in a sylvatic cycle between forest-dwelling mosquitoes and nonhuman primates. In these areas, sporadic human cases occur, but large human outbreaks were not common. Since 2004, Chikungunya virus (CHIKV) has been causing large epidemics of Chikungunya fever (CHIK), with considerable morbidity and sufferings. The disease reached Mombasa on the Kenyan coast in November–December 2004 and the Comoros, where it resulted in a large outbreak in 2004–2005. Approximately 63% of the population may have been infected with CHIKV in Grande Comore. The disease also reemerged in the rest of Asia, with significant outbreaks in Sri Lanka in 2006, in Malaysia in 2007, and in Thailand and Singapore in 2008–2009.In Malaysia, CHIKV reemerged in April–May 2006, 7 years after the first outbreak in 1998. In Cambodia, outbreaks were

detected in May–December 2011 in several locations within the country and again in 2012. Studies of the circulating 2008–2010 strains from Malaysia, Singapore, Thailand, Cambodia, and China highlighted the widespread establishment of the ECSA, with strains harboring the E1-A226V mutation.In 2007, an outbreak was reported for the first time in northeastern Italy. A total of 217 cases were reported in July–September 2007.

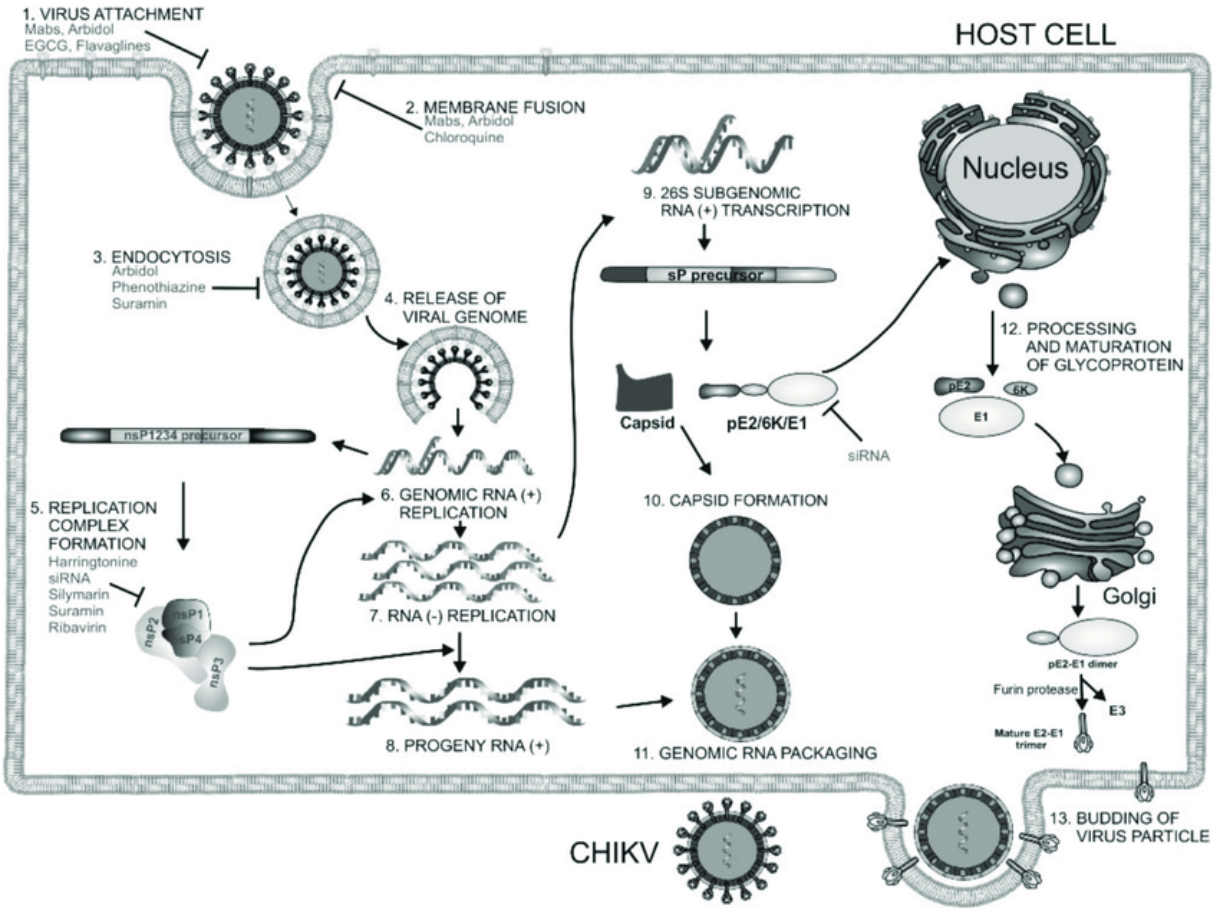

Figure 3: Chikungunya virus replication cycle.

A limited outbreak was reported in September 2010 in southern France, where an enhanced surveillance of febrile illness, focusing on CHIKV and DENV, has been in place since 2007 in areas where an albopictus is present. The virus in both the 2010 and 2014 outbreaks belonged to the ECSA genotype but did not possess the E1-A226V mutation. On the Caribbean island of St Martin Island, a cluster of clinical cases with onset of febrile illness starting in mid-October 2013 resulted in negative laboratory results for dengue in November 2013. This document is meant to serve as a guideline that individual countries can use as the basis for their CHIKV surveillance, prevention, and control programs.[4]

Causes of Transmission: It is a mosquito borne viral disease caused by Chikungunya virus. The virus is transmitted to human by *Aedes aegypti* mosquito. The disease is transmitted by the following causes.

•The mosquito breed on stagnant water.

•The mosquitoes bites during day time with peaks in early morning and late afternoon.

•Mosquitoes acquire the virus from humans and after a period of approx 8-10 days they transfer it to other human while feeding.

•The virus circulates in blood of infected person for several days at approximately the time when the person gets its fever.

•The number of cases varies depending on the immunity of the exposed population. It has been estimated that about 40% of the 700,000 residents of Reunion Island were infected during the 2005-2007 epidemic, and as part of the same outbreak, a subsequent 1.3 million cases have occurred in India.

Signs and Symptoms: While the incubation period of chikungunya disease is between two to six days, the symptoms usually start appearing four to seven days post-infection. Though the result of this disease is not death, the symptoms may persist for months or even more. These may be severe and disabling. Newborns who are infected around the time of birth, adults aged ≥65 years and people with high blood pressure, diabetes or heart disease are more susceptible or at higher risk for more severe disease.

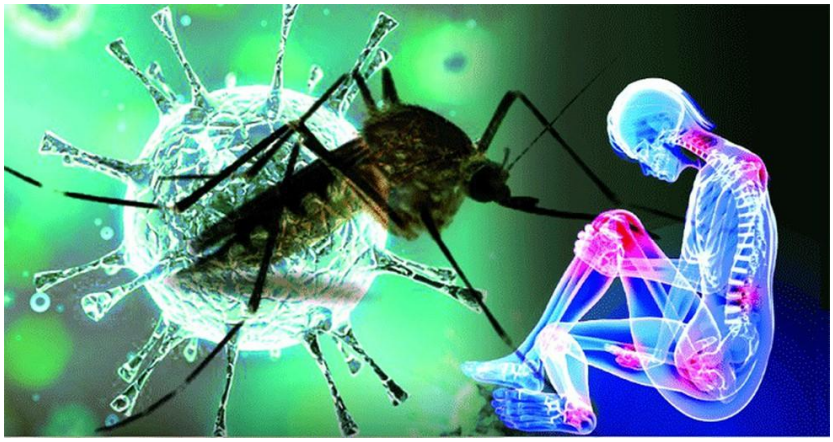

Figure 4: Chikungunya Virus.

It is said that if a person is infected once, he or she will likely to be protected from further infections in future. The common sings & symptoms of Chikungunya virus is given below-

- Chikungunya fever is an acute febrile illness with an incubation period of 3-7 days.
- It affects all age groups and both sexes equally, with an attack rate (percentage of individuals who develop illness after infection) of 40%-85%.
- Patients present with abrupt onset of high-grade fever often reaching 102°-105°F, with shaking chills that last 2-3 days.
- The fever may return for 1-2 days after an afebrile period of 4-10 days, hence called a "saddle-back fever."
- Others symptoms may include headache, muscle pain, joint swelling or rash.
- Most of the patients recover fully, but some cases joint pains may persist for weeks or months.
- As per WHO children less than 1 year and elderly are at greater risk of severe disease.

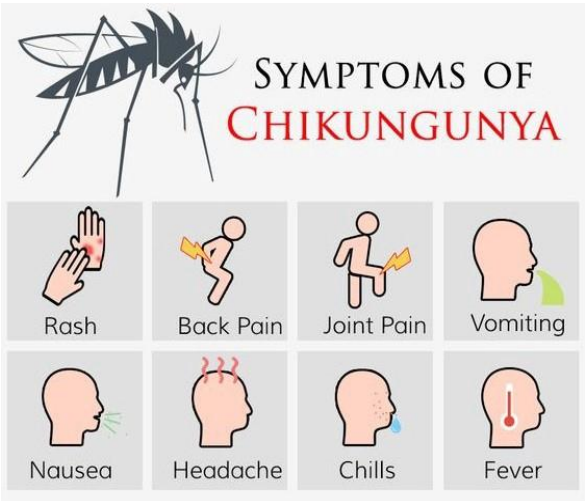

Figure 5: Symptoms of chikungunya.

MECHANISM: The immunopathological mechanisms responsible for CHIKV disease are poorly understood, primarily owing to the lack of suitable animal models of disease. However, much can be learnt about alpha viral disease mechanisms by drawing on current RRV studies and disease models. Not only could these studies provide an insight into the mechanisms of CHIKV disease, they may also provide essential clues for the development of a much-needed mouse model of CHIKV disease that mimics the adult human arthritic disease. A recent study has established a mouse model for CHIKV disease using neonatal and type-1 interferon (IFN)-deficient mice. This model mimics the more severe cases of CHIKV, resulting in flaccid paralysis, lethargy, viral dissemination to the CNS and sometimes mortality. Mice with partial deficiency in type-1 IFN provided a model for benign CHIKV infection, where no lethality or mortality was observed but infectious virus was obtained from various tissues. Wild-type mice, however, failed to exhibit any signs of infection. Unfortunately, these mouse models do not mimic many aspects of the human disease as the models do not show prominent limb weakness and inflammatory response in bone and joint tissues. Clearly, more research is needed to develop an adult mouse model appropriate for this form of the disease.[5]

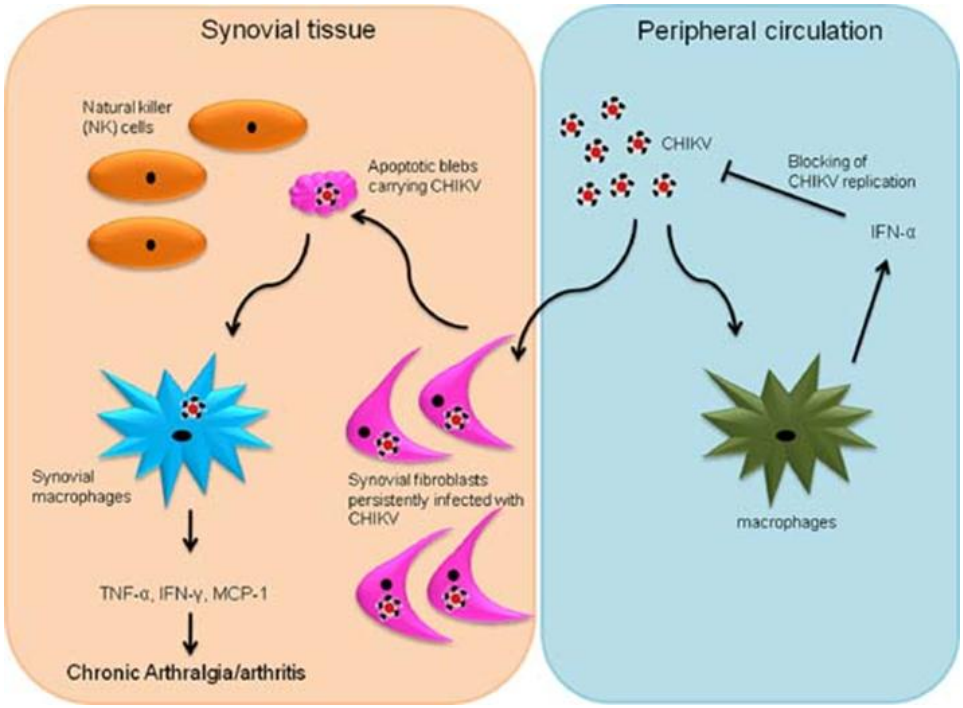

Figure 6: Chikungunya transmission.

The initial studies involving a mouse animal model for CHIKV infection have similarities to early RRV studies. For example, in 1973, studies into RRV infection in outbred mice showed a similar spread of virus through a wide range of tissues, including the CNS, although no evidence of RRV replication or RRV-mediated pathology was found in the joint tissues. The targeting of connective tissue of joints and skeletal muscles have been identified as sites of viral replication for both CHIKV and RRV within the respective mouse models. Similar to what was seen for CHIKV in these and previous studies, we have found that RRV is unable to replicate in mouse blood leukocytes and primary human monocytes, except when infection is performed in the presence of sub-neutralizing concentrations of anti-RRV antibody through a process known as antibody-dependent enhancement of infection .The early RRV mouse models were originally thought to be unsuitable models for human RRV-induced arthritic disease due to the lack of RRV-mediated pathology in the joint tissues. After further development, however, an RRV disease model in outbred mice was established, with characteristics that clearly mimicked that of the human disease. Mice developed arthritic symptoms in the hind limbs at 7–9 days post-infection, with a dramatic infiltration of monocytes and macrophages into the hind-limb muscle, causing associated disruption to the muscle structure. Full recovery of mice was seen after approximately 25 days post-infection. This model was successfully reproduced using inbred mice, resulting in similar clinical features and pathology. Since the newly developed CHIKV disease mouse models also do not mimic the less severe form of CHIKV disease, showing either severe symptoms or not producing detectable arthritic symptoms, the lessons learnt from the establishment of an arthritic RRV mouse model could provide valuable clues for the development of a more appropriate mouse model.[6]

Diagnosis: Chikungunya infection is diagnosed on the basis of clinical, epidemiological and laboratory criteria. An acute onset of fever and severe arthralgia or arthritis that is not explained by other medical disorders is considered a possible CHIKV case. Diagnosis can be delayed due to the possible confusion of symptoms with those of dengue fever or Zika. Fever and polyarthralgia give 84% sensitivity, 71% positive predictive value (PPV), and 83% negative predictive value (NPV). Enzyme-linked immunosorbent assays (ELISA) can be used to confirm the presence of anti-CHIKV antibodies, with IgM antibody levels highest three to five weeks post-infection and persisting for up to two months. PCR can also be used to genotype the virus.

Treatment

- There are no proper medicines or vaccines for the treatment of chikungunya.
- Take proper rest for quick recovery.
- Always take enough fluid to prevent yourself from dehydration.
- Take Acetaminophen, Ibuprofen or Paracetamol to reduce fever & pain.
- Don’t take NSAIDS (non steroidal anti inflammatory drug like Aspirin).
- If you are taking medicines for other health issues, talk to your doctor for proper treatment

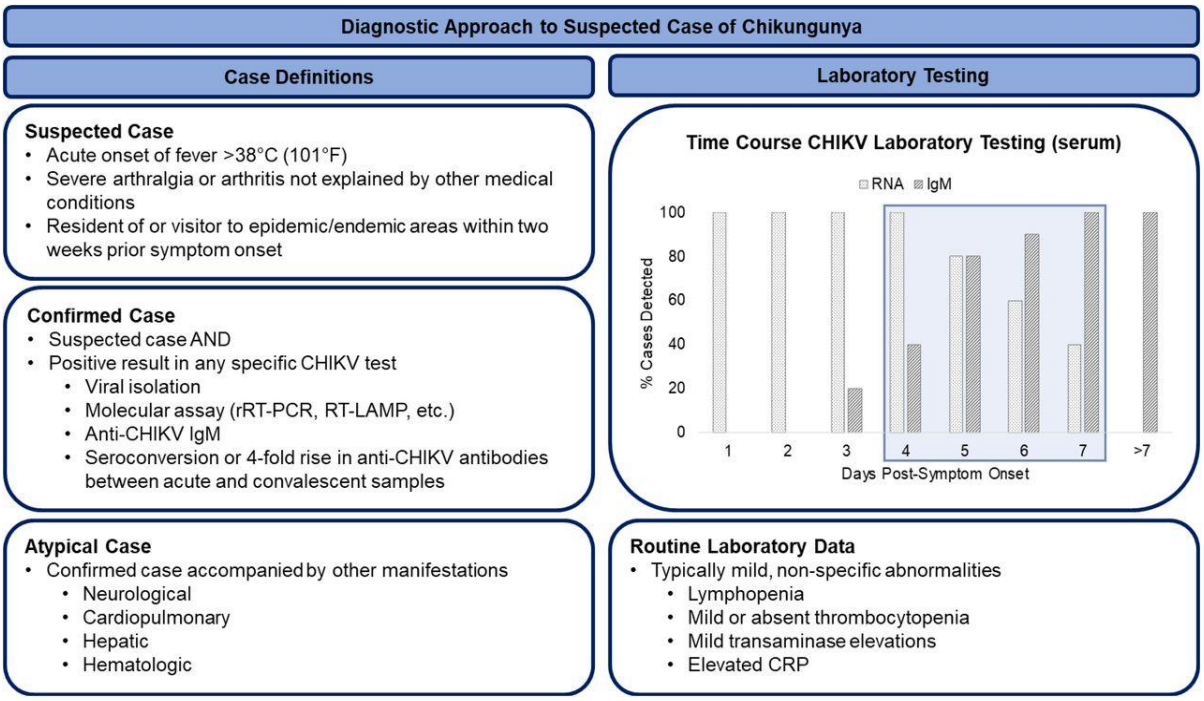

Figure 7: Diagnostic flowchart.

Prevention

As we all know, Chikungunya is a mosquito borne disease. So, the best way to prevent chikungunya is to avoid mosquito & mosquito bites.

- Try to cover most of your body with full sleeve dresses.
- For longer protection use active DEET, oil of lemon, oil of eucalyptus, IR535 as insect repellent.
- Using of mosquito netting is the best idea to prevent chikungunya.
- Try to stay indoor as much as possible.
- You can also try the clothes which are generally treated with permethrin or other mosquito repellent.
- Keep your surrounding clean as much as possible.
- Always consider a health professional, if you are traveling to an area endemic with chikungunya.
  And if you have chikungunya, must be prevent yourself form mosquito bite because in the first week of infection, the virus can be found in your blood and can infect a normal people through the mosquito.

Geographic Distribution: Historically, chikungunya has been present mostly in the developing world. The disease causes an estimated 3 million infections each year. CHIKV is typically found being transmitted in Africa and Southeast Asia. Since its discovery in 1952, CHIKV has caused several epidemics in these places.[7]

The last huge outbreak started in Kenya in 2004 and spread through neighbor islands to La Réunion in 2005. After that, the virus spread to several islands in the Indian Ocean and India. From India it spread to Sri Lanka, Thailand, Malaysia and finally to Italy in 2007. In 2009 CHIKV transmission restarted in La Réunion and lead to CHIKV re-importation to Europe in May 2010.

During 2011, CHIKV was transmitted in Oceania, Central Africa, Southern and Southeastern Asia, Europe and Western Indian Ocean Islands. In 2012, CHIKV was reported in: Southeastern, Southern and Western Asia; Oceania; Central and Western Africa; and Western Indian Ocean Islands. During 2013, CHIKV was transmitted in Southeastern, Southern and Eastern Asia and Oceania.

The current outbreak started in, the Caribbean Island, Saint Martin on December 6, 2013. During December 2013 and January 2014 it spread to the neighbor islands. In February, it continued spreading and reached French Guiana. In May, Guiana and almost all the Caribbean Islands reported autochthonous CHIKV infections. In June, the first cases of El Salvador were reported. By July, autochthonous transmission was reported in Florida, USA, Costa Rica, Panama and Venezuela. By September, cases were reported in Guatemala, Colombia and Brazil. In October, Nicaragua and Paraguay reported cases for the first time and Guatemalan cases rose. By the end of November, Mexico reported its first autochthonous transmission in the southern state of Chiapas. Also by this month, Belize and Honduras reported cases.

According to the Pan American Health Organization (PAHO), since the current outbreak started, there have been 1,280,953 suspected autochthonous transmission cases and more than 26,300 have been confirmed in America. The recent reports obtained from Mexico reveals 405 confirmed autochthonous transmissions.

Nevertheless, this numbers does not include patients that did not look for medical aid.[8]

Table-1: Recent selected large-scale CHIKV epidemics in the 2000s.

Location	Year/Duration	Affected
Lamu Island, Kenya	2004	13,500
La Réunion	2005–2006	255,000
India	2005	1,380,000
Mauritius	2006	13,500
Gabon	2007	20,000
Thailand	2008-2009	49,000
Republic of Congo	2011	8000
Martinique-Guadeloupe	2014	308,000
French Polynesia	2014-2015	66,000

An analysis of the genetic code of Chikungunya virus suggests that the increased severity of the 2005–present outbreak may be due to a change in the genetic sequence which altered the E1 segment of the virus' viral coat protein, a variant called E1-A226V. This mutation potentially allows the virus to multiply more easily in mosquito cells. The change allows the virus to use the Asian tiger mosquito (an invasive species) as a vector in addition to the more strictly tropical main vector, Aedes aegypti. Enhanced transmission of Chikungunya virus by A. albopictus could mean an increased risk for outbreaks in other areas where the Asian tiger mosquito is present. A albopictus is an invasive species which has spread through Europe, the Americas, the Caribbean, Africa and the Middle East.

After the detection of zika virus in Brazil in April 2015, the first ever in the Western Hemisphere, it is now thought some chikungunya and dengue cases could in fact be zika virus cases or coinfections.

Epidemiology: Chikungunya is a mosquito borne alpha virus noticed after a outbreak in 1952 in Tanzania. This virus has found in the area of sub-saharan Africa which involves nonhuman primate hosts &arboreal mosquito vectors. Later spread beyond Africa may have started in 18th century. Chikungunya appears in Asia during 19th century & continues to spread in Southeast Asia. An analysis about genetic coding of chikungunya virus says that the increase severity of 2005 & recent outbreaks is due to the change in genetic sequence which leads to multiple the cell of virus more easily while in the body of mosquito. The changed virus used Asian Tiger mosquito as vector. The RNA & various strains of virus by genetic sequence was recognized by a team in July 2006.

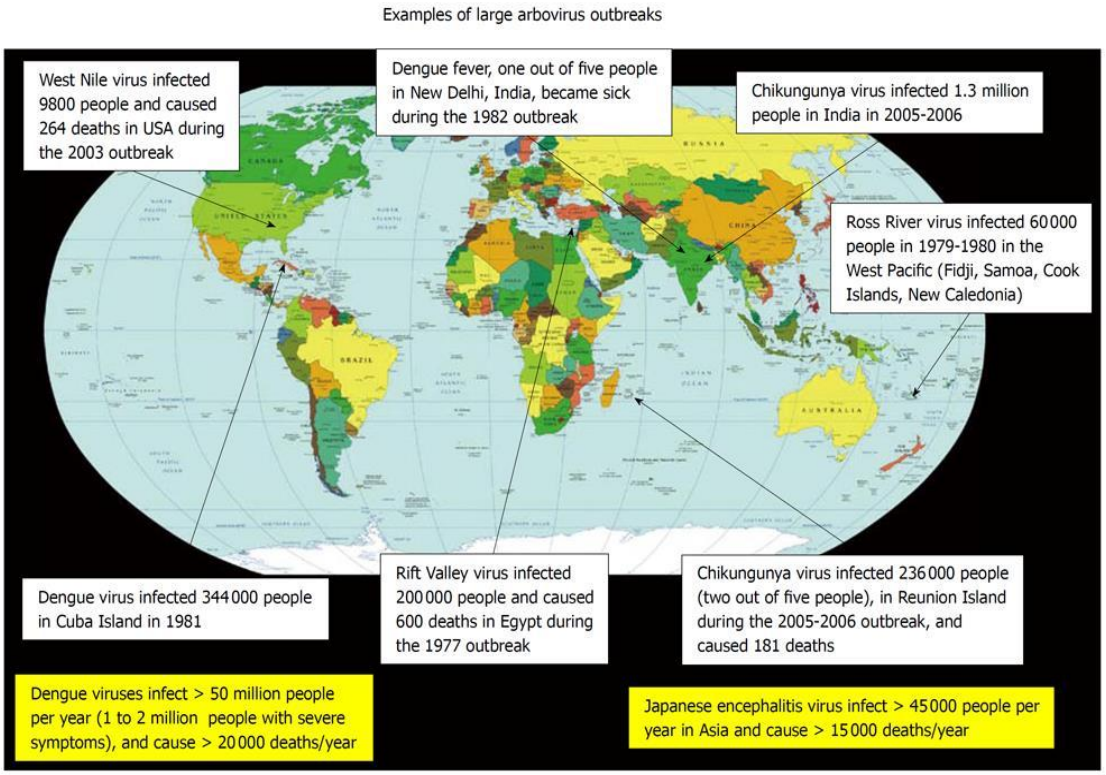

Figure 8: Global threat of chikungunya.

The largest outbreak of chikungunya was recorded in late March 2005 to 2006 on the island of western rim of Indian ocean. About 25000 cases was recorded daily. The amount was decrease gradually& remain stable for some months. But again, in the beginning of October 2005, the amount was increased dramatically as the weather was favorable for mosquito growth. Although number of confirmed cases was very low as these are not recorded or depended upon extrapolation, suggested by physician (as many as 110,000 of the total 800000

population). Other countries such as Mauritius, Seychelles, Madagascar, Comoros, Mayotte of Southwest Indian Ocean also reported some cases of chikungunya virus. In India, states affected by the outbreak of chikungunya were Andhra Pradesh, Andaman & Nicobar Islands, Tamil Nadu, Karnataka, Maharashtra, Gujarat, Madhya Pradesh, Kerala and Delhi which was also in 2006.The major cause of mortality is due to severe dehydration, electrolyte imbalance and loss of glycemic control. Recovery is the rule except for about 3 to 5% incidence of prolonged arthritis. As this virus can cause thrombocytopenia, injudicious use of these drugs can cause erosions in the gastric epithelium leading to exsanguinating upper GI bleed. Also the use of steroids for the control of joint pains and inflammation is dangerous and completely unwarranted. On average there are around 5,300 cases being treated every day. Prior to 2013, chikungunya virus outbreaks had been identified in Africa, Asia, Europe, the Indian and Pacific Oceans. In late 2013, the first local transmission of chikungunya virus was identified in Caribbean countries and territories.[9]

Recent Outbreaks: Chikungunya virus disease cases have also been reported in Asia and Africa during this period. Since the previous update on 17 January 2020, Brazil and Thailand have reported the majority of new cases. Additionally, since the last update, Ethiopia has declared the end of the chikungunya outbreak.

Figure 9: Global outbreak.

Detailed overview

Europe: No autochthonous cases of Chikungunya virus were detected in continental EU/EEA countries and the UK in 2019 and 2020.

Americas and the Caribbean

Brazil: In 2020 and as of 25 January, Brazil reported 2 340 suspected cases.

Colombia: In 2020 and as of 9 February, Colombia reported 82 cases, two of which are laboratory confirmed. This represents a lower number of cases in comparison with the same period in 2017, 2018 and 2019 according to national health authorities.

El Salvador: In 2020 and as of 8 February, El Salvador reported 19 suspected cases. For the same period in 2019, El Salvador reported 35 suspected cases.

Asia

India: According to media reports quoting health authorities, three chikungunya cases have been reported in New Delhi in February 2020.

Malaysia: In 2020 and as of 25 January, 170 cases have been reported across the country, with most of the cases being reported in Perak region according to Malaysia Ministry of Health.

The Philippines: According to media reports quoting local health authorities, at least 40 chikungunya cases were reported in Tampakan city, in South Cotabato, in the past weeks.

Thailand: In 2020 and as of 10 February, Thailand reported 559 cases with no deaths associated affecting 37 provinces. Provinces reporting the highest incidences are located in the southern part of the country.

Africa

Ethiopia: According to WHO, the chikungunya outbreak in Ethiopia is considered to be over. Since the beginning of the outbreak in July 2019 and as of 8 December 2019, when the last cases were reported, 54 908 cases with no associated deaths were reported in Dire Dawa city, Araf and Somali regions.

Kenya: According to WHO, a chikungunya outbreak has been reported in Garissa County, Kenya. From 31 December 2019 to 9 February 2020, 163 cases have been reported.

Australia and the Pacific

No outbreaks have been reported since the previous update.[10]

Symptoms

- Most people infected with chikungunya virus will develop some symptoms.
- Symptoms usually begin 3–7 days after being bitten by an infected mosquito.
- The most common symptoms are fever and joint pain.
- Other symptoms may include headache, muscle pain, joint swelling, or rash.
- Chikungunya disease does not often result in death, but the symptoms can be severe and disabling.
- Most patients feel better within a week. In some people, the joint pain may persist for months.
- People at risk for more severe disease include newborns infected around the time of birth, older adults (≥65 years), and people with medical conditions such as high blood pressure, diabetes, or heart disease.
- Once a person has been infected, he or she is likely to be protected from future infections.

Diagnosis

- The symptoms of chikungunya are similar to those of dengue and Zika, diseases spread by the same mosquitoes that transmit chikungunya.
- See your healthcare provider if you develop the symptoms described above and have visited an area where chikungunya is found.
- If you have recently travelled, tell your healthcare provider when and where you travelled.
- Your healthcare provider may order blood tests to look for chikungunya or other similar viruses like dengue and Zika.

Treatment

- There is no vaccine to prevent or medicine to treat chikungunya virus.
- Treat the symptoms:
  - Get plenty of rest.
  - Drink fluids to prevent dehydration.
  - Take medicine such as acetaminophen (Tylenol®) or paracetamol to reduce fever and pain.
  - Do not take aspirin and other non-steroidal anti-inflammatory drugs (NSAIDS until dengue can be ruled out to reduce the risk of bleeding).
  - If you are taking medicine for another medical condition, talk to your healthcare provider before taking additional medication.
- If you have chikungunya, prevent mosquito bites for the first week of your illness.
  - During the first week of infection, chikungunya virus can be found in the blood and passed from an infected person to a mosquito through mosquito bites.
  - An infected mosquito can then spread the virus to other people.[11]

CONCLUSION

In recent years there have been explosive outbreak of Chikungunya fever in several parts of sea. Although the disease is self-limiting, morbidity can be very high in major outbreaks resulting in heavy social and economic roll. Integrated vector management through the elimination of breeding sites, use of anti-adult and anti-larval measures and personal protection will contribute to prevent and outbreak. WHO encourages countries to develop and maintain the capacity to detect and confirm cases, manage patients and implement social communication strategies to reduce the presence of the mosquito vectors. Aerosol sprays may be applied throughout the home, but areas where adult mosquitoes rest (dark, cooler areas) must be targeted, including bedrooms, closets, clothing hampers, etc. Care should be taken to emphasize proper use of these products when advocating their application to the public, in order to reduce unnecessary exposure to pesticides.

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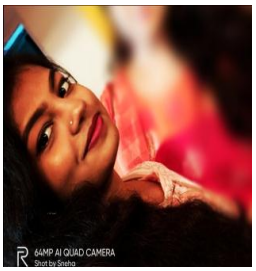

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