

# The Comprehensive Review on The Chikungunya Fever

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**Abstract:** Chikungunya fever, a debilitating arthritic disease spread by mosquitoes, is caused by the chikungunya virus (*CHIKV*), which is the etiological agent of the disease. Over the course of the past seven years, the virus has spread from Africa to the Indian Ocean islands and Asia, causing a significant amount of morbidity and some mortality among humans, including newborn babies. Starting from the main reports of its presence in Africa during the 1950s, in excess of 1500 logical distributions on the various parts of the sickness and its causative specialist have been delivered. According to an examination of these publications, despite the absence of autochthonous cases in developed nations and several studies in the 1960s and 1970s, the scientific community maintained a low level of interest. In any case, in 2005 chikungunya fever out of the blue reappeared through wrecking pandemics in and around the Indian Sea. Mutations in the viral genome that facilitated the virus's replication in *Aedes albopictus* mosquitoes were linked to these outbreaks. An acute febrile illness known as chikungunya fever is accompanied by severe, frequently crippling polyarthralgias. Chikungunya virus (*CHIKV*), an arthropod-borne virus that is primarily transmitted to humans by the bite of infected mosquitoes, is the cause of the disease. Millions of cases of the disease have been reported in countries in and around the Indian Ocean since the virus's reemergence in 2004 and its spread to new locations, such as Europe. Due to the high attack rates associated with recurrent epidemics, the high levels of viremia in infected humans, and the worldwide distribution of the *CHIKV*-transmitting vectors, the risk of *CHIKV* importation into new regions is always present.

**Keywords:** CHIKV, hikungunya virus, Chikungunya fever, Arbovirus, Alphavirus, Antiviral therapy

## INTRODUCTION: -

The bite of infected *Aedes* mosquitoes transmits the viral disease known as chikungunya fever. The illness typically manifests as an acute fever, skin rash, and arthralgia that is incapacitating. In Makonde, an African dialect, the word "chikungunya" means "to walk bent over," and it refers to the incapacitating arthralgia seen in affected patients. [1] Chikungunya virus (*CHIKV*) is the etiological agent and a member of the Alphavirus genus in the family *Togaviridae*. [2] There are cases of chikungunya in Asia, Africa, and the Indian subcontinent. For several years, human infections in Africa have been relatively low. In December 2013, two laboratory-confirmed autochthonous cases of were reported in the French portion of the Caribbean island of St. Martin. Local transmission has been confirmed in over 43 American nations and territories since then. [3] This was the main reported episode of *CHIKV* with autochthonous transmission in the Americas; Consequently, it is a topic of significant concern on our continent. Chikungunya is a virus that infected mosquitoes transmit to humans. The chikungunya virus (*CHIKV*) is the cause.

This was the main reported episode of *CHIKV* with autochthonous transmission in the Americas; Consequently, it is a topic of significant concern on our continent. Chikungunya is a virus that infected mosquitoes transmit to humans. The chikungunya virus is to blame. A *CHIKV* infection results in severe joint pain and fever. Muscle pain, joint swelling, headache, nausea, fatigue, and a rash are additional symptoms. Chikungunya-related joint pain is frequently crippling and can last for a variety of lengths of time. There is neither a specific drug nor vaccine for the virus currently. The goal of the treatment is to get rid of the symptoms of the disease. Africa, Asia, and India account for most cases. However, sporadic outbreaks are observed elsewhere, and a significant outbreak occurred in several countries of the Americas Region in 2015. In areas where they are prevalent, the disease can be misdiagnosed because it shares some clinical symptoms with Zika and dengue. Chikungunya-related severe cases and deaths are extremely uncommon and almost always associated with other health issues. There is no accurate estimate of the annual number of people affected by chikungunya worldwide because accurate diagnosis is difficult. A significant factor in the risk of contracting chikungunya is the proximity of human habitation to breeding grounds for mosquitoes.

Chikungunya is a virus spread by mosquitoes. It was first discovered during a 1952 outbreak in southern Tanzania. It is an RNA virus that is a member of the family *Togaviridae* and the alphavirus genus. A word in the Kimakonde language that means "to become contorted" is the source of the term "chikungunya," which refers to the stooped appearance of those who suffer from joint pain (arthralgia). The Chikungunya virus (*CHIKV*) is what causes the disease chikungunya. Fever and joint pain are symptoms. Between two and twelve days after exposure, these typically occur. A rash, muscle pain, joint swelling, and a headache are additional signs. Within a week, symptoms usually improve; However, the joint pain can occasionally last for months or years. The chance of dying is about one in 1,000. More severe diseases are a possibility for the elderly, very young, and those with other health issues. us in *CHIKV* The virus is spread between people by two types of mosquitos: *Aedes albopictus* and *Aedes aegypti*. They mainly bite during the day. The virus may circulate within several animals including birds and rodents. Diagnosis is by either testing the blood for the virus's RNA or antibodies to the virus. The symptoms can be mistaken for those of dengue fever and Zika fever. It is believed most people become immune after a single infection. The best method for anticipation is mosquito control and the aversion of chomps in regions where the illness is normal. Using insect repellent and mosquito nets, reducing the mosquito population's access to water, and other measures, could help. As of 2016, there is neither a specific treatment nor a vaccine. Suggestions incorporate rest, liquids, and drugs to assist with fever and joint agony. Since the 2000s, outbreaks of the disease have been reported in Europe and the Americas. In 2014, more than a million suspected cases occurred in Florida in the continental United States; however, as of 2016, there had been no additional locally acquired cases. In 1952, the disease was first discovered in Tanzania. The Kimakonde language uses the phrase, which means "to become contorted."

An abrupt onset of fever, frequently accompanied by joint pain, is the most common symptom. Muscle pain, headache, nausea, fatigue, and a rash are additional symptoms. Although severe joint pain typically subsides within a few days, it may last for months or even years. Serious confusions are phenomenal, yet abnormal extreme cases can cause long haul side effects and even demise, particularly in more seasoned individuals. Polyarthralgia, a maculopapular rash, and an abrupt febrile illness are all symptoms of chikungunya fever. Shock or severe bleeding in chikungunya fever are uncommon; Compared to dengue disease, the onset is quicker and the fever lasts less time. The most common symptom is an abrupt onset of fever, frequently accompanied by joint pain. Additional symptoms include muscle pain, a headache, nausea, fatigue, and a rash. Even though severe joint pain usually goes away after a few days, it can last for months or even years. Serious confusions are amazing, but unusual extreme cases can cause long-term side effects and even death, especially in older people. Chikungunya fever is characterized by polyarthralgia, a maculopapular rash, and an abrupt febrile illness. In chikungunya fever, shock or severe bleeding are uncommon; The onset and duration of the fever are shorter in this illness than in dengue fever. There are cases of chikungunya in Asia, Africa, and the Indian subcontinent. For a few years, human infections in Africa have been relatively low. In the French portion of the Caribbean island of St. Martin, two laboratory-confirmed autochthonous cases were reported in December 2013 by France. From that point forward, neighborhood transmission has been affirmed in more than 43 nations and domains in the American region. [3] This was the principal reported flare-up of CHIKV with autochthonous transmission in the Americas; Consequently, it is a topic of significant concern on our continent. Chikungunya fever, a rash, acute febrile illness, and arthralgia are all symptoms of this virus. Chikungunya fever can progress into potentially long-term, crippling arthritis that can last for months or years. The evaluation and treatment of chikungunya fever are demonstrated in this activity, which also discusses the interprofessional team's role in enhancing patient care.

The most reliable method for diagnosing Chikungunya fever is viral culture. Real-time loop-mediated isothermal amplification and reverse transcription polymerase chain reaction have also been found to be useful. More frequently, serodiagnostic methods are utilized for the detection of Chikungunya virus immunoglobulin M and G antibodies. The chikungunya virus can be cured on its own; However, life-threatening severe manifestations like meningoencephalitis, fulminant hepatitis, and bleeding manifestations can occur. Supportive and symptomatic treatment is used. Since there is currently no commercially available vaccine for this condition in India, prevention by educating the community and public health officials and vector control measures appear to be the most effective means of controlling Chikungunya fever. Due to its explosive onset, rapid spread, high morbidity, and numerous clinical manifestations, the recent resurgence of Chikungunya fever has attracted global attention. Chikungunya fever has become a significant illness among returning travelers since 2006, even in areas where it is not endemic. Indeed, travelers have emerged as the disease's sentinels, carriers, and transmitters. The social and financial effect of Chikungunya fever has likewise been huge particularly in India.

### CHIKV Genome, Structure And Replication

Icosahedral symmetry characterizes the enveloped plus-strand RNA virus known as chikungunya virus. The virion is 70 nm in measurement and it is made from rehashing units of the E1 and E2 transmembrane glycoproteins (240 heterodimers of E2/E1 organized as trimeric spikes on its surface), the capsid (C), a host-determined lipid bilayer, and a solitary particle of genome RNA. The genome is roughly 12 kb long and encodes the non-structural proteins (nsPs) at the 5' end and the underlying proteins at the 3' end. The structural proteins and the nsPs are translated from sub-genomic RNA. Endocytosis is how RNA Alphaviruses enter their target cells. The viral replication complex is made up of these proteins and produces an intermediate with full-length negative strand RNA. Both subgenomic (26S) and genomic (49S) RNAs are synthesized from this as a basis for their respective structures. The autoprotolytic processing of the C---pE2---6K---E1 polyprotein precursor is triggered by the subgenomic RNA. The capsid is let go, and the pE2 and E1 glycoproteins are made by further processing. Together, PreE2 and E1 form the Golgi and export to the plasma membrane, where pE2 is broken down into E2 and E3. Restricting of the viral nucleocapsid to the viral RNA and the enlistment of the membrane-associated envelope glycoproteins advance viral gathering. The icosahedral core of the assembled alphavirus particle buds at the cell membrane [5,7].

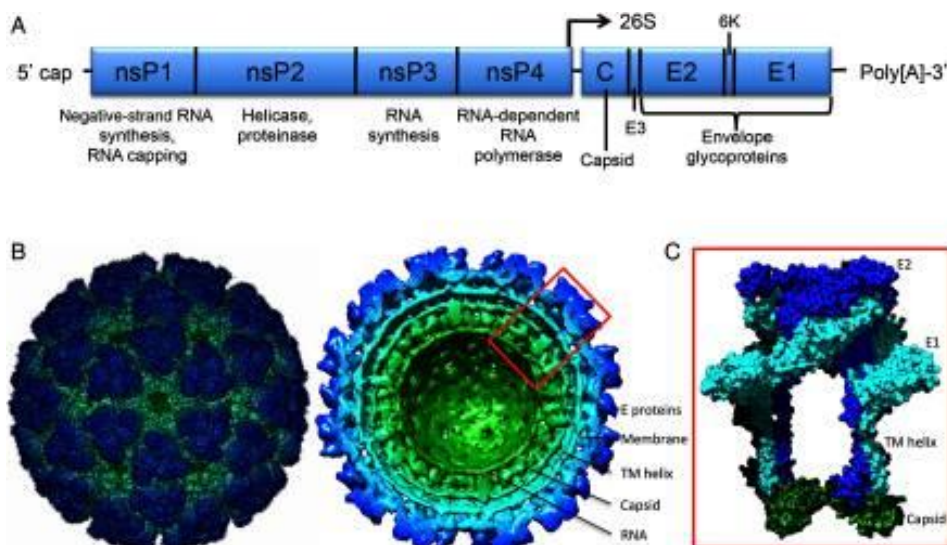


Fig. 1

**Vectors, Transmission And Reservoirs :-**

There are clearly two distinct transmission cycles: urban and enzootic. Arboreal mosquitoes, particularly *Aedes* spp., serve as vectors in Africa, where an enzootic cycle occurs in forested habitats. Based on their high rates of seroprevalence, documented infection and viremia in nature, and viremia levels in response to experimental infection, nonhuman primates appear to be the primary reservoir and amplification hosts in the enzootic cycle. [8] The enzootic transmission cycle can spread to people who live nearby, and enzootic mosquito vectors may be involved in interhuman transmission during small outbreaks. When CHIKV enters urban areas where the more anthropophilic vectors, *Aedes aegypti* and *Aedes albopictus*, can initiate human-to-human transmission, epidemics also occur in Africa. CHIKV is equipped for starting a supported, metropolitan transmission cycle that depends just on *A. aegypti* or potentially *A. albopictus* and human enhancement hosts. [9] This endemic/pandemic cycle brings about elevated degrees of human openness to mosquito transmission, especially because these vectors live in closeness to individuals. *A. albopictus* is both zoophilic and anthropophilic, aggressive, silent, active all day, and has a longer lifespan than other mosquitoes (up to 8 weeks). In particular, the behavior and ecology of *A. aegypti* are ideal for epidemic transmission because adult females prefer to feed on humans, frequently take several partial blood meals during a single gonotrophic cycle, oviposit in artificial containers as their preferred larval sites, and rest inside houses. Although the infectivity of various CHIKV strains varies widely for both *A. aegypti* and *A. albopictus*, humans develop high-titer viremias that typically persist during the first four days after the onset of symptoms, with the peak estimated at approximately 10<sup>9</sup> viral RNA copies/ml<sup>14</sup> and infectious titers sometimes exceeding 10<sup>7</sup> PFU/ml.<sup>15</sup> These titers typically exceed the oral infectious dose 50% levels for both epidemic vector. In the absence of human cases, monkeys, rodents, and birds serve as the virus reservoir outside of these times, sustaining environmental virus circulation.

**EXTRINSIC INCUBATION**

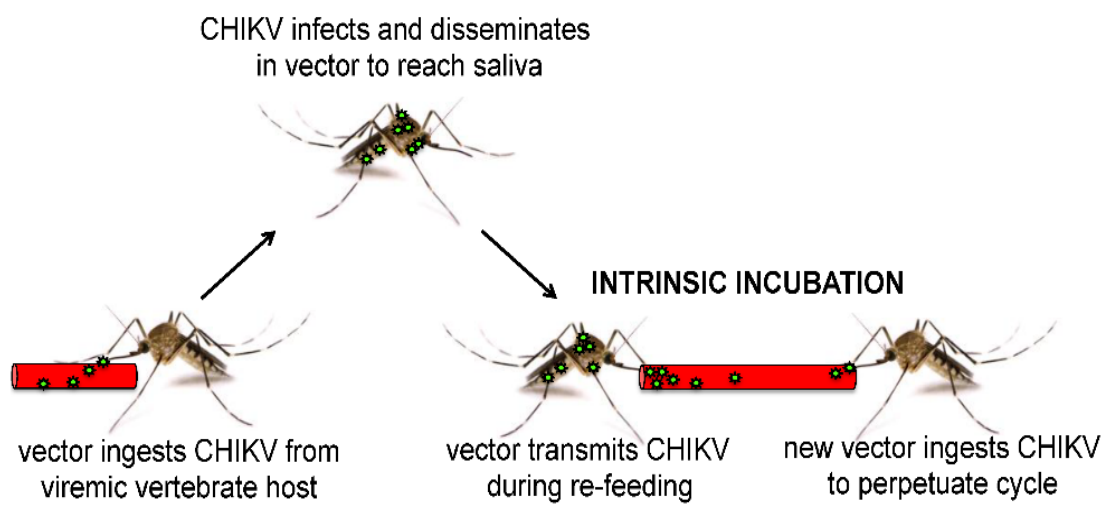


Fig. 2

**Geographic Distribution :-**

Most cases of CHIKV transmission can be found in Southeast Asia and Africa. Since its discovery in 1952, CHIKV has caused numerous epidemics in these locations. [19,20] The most recent major outbreak began in Kenya in 2004 and spread to La Réunion in 2005 through neighboring islands. [21] From there, the virus spread to several Indian Ocean islands and India. [1,22] From India, it spread to Sri Lanka, Thailand, Malaysia, and Italy in 2007. [23,24] In 2009, CHIKV transmission resumed in La Réunion, which led to CHIKV reimportation to Europe in May. CHIKV was reported in:

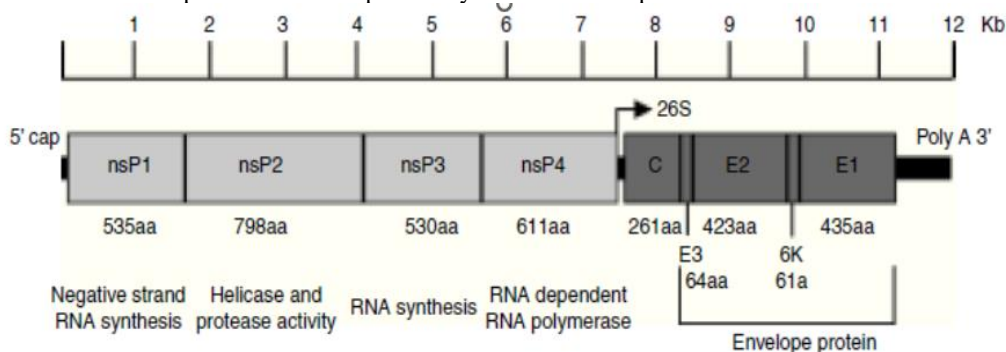


Fig. 3

Asia's western, southern, and southeast; Oceania; West and Central Africa; and Islands in the Western Indian Ocean. During 2013, CHIKV was communicated in Southeastern, Southern and Eastern Asia and Oceania. [20] The ebb and flow episode began in, the Caribbean Island, Holy person Martin on December 6, 2013. [26] During December 2013 and January 2014 it spread to the neighbor islands. [27] In February, it kept spreading and arrived at French Guiana. [28] In May, Guiana and practically all the Caribbean Islands detailed autochthonous CHIKV infections. [29] In June the principal instances of El Salvador were reported. [30] By July, autochthonous transmission was accounted for in Florida, USA, Costa Rica, Panama and Venezuela. [31] By September, cases were accounted for in Guatemala, Colombia and Brazil. [32] In October, Nicaragua and Paraguay revealed cases interestingly

and Guatemalan cases rose.[33] Toward the finish of November, Mexico announced its most memorable autochthonous transmission in the southern territory of Chiapas. Likewise by this month, Belize and Honduras announced cases.3 As per the Container American Wellbeing Association (PAHO), since the ongoing episode began, there have been 1,280,953 thought autochthonous transmission cases and more than 26,300 have been affirmed in America.[35] The new reports got from Mexico uncovers 405 affirmed autochthonous transmissions.[35] By and by, this numbers does exclude patients that didn't search for clinical guide.

**Phylogeny**

There are four known CHIKV lineages, each with its own unique antigenic and genotypic features. According to the results of the initial phylogenetic study, CHIKV originated in Africa, where two main lineages were present: East/Central/South Africa and West Africa. Posteriorly the ECSA ancestry spread to Asia and started the Asian lineage.[36,37] Until 2004, these were the just recognized CHIKV heredities that were circling. The ECSA lineage was found to be the source of the 2005 Indian Ocean pandemic.[38] When the pandemic started in Kenya in 2004, the first CHIKV isolates from La Réunion had an alanine at residue 226

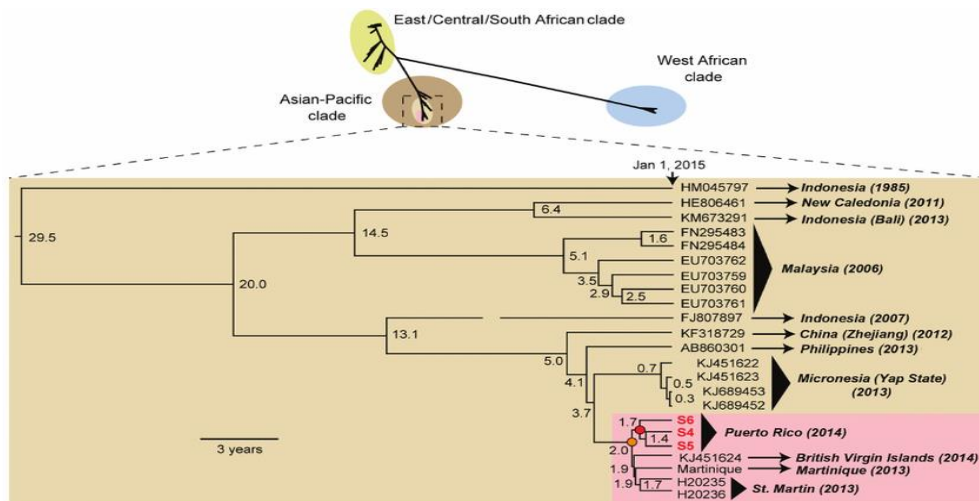


Fig. 4

of the E1 envelope protein. Later isolates had an A226V substitution. This and extra replacements led to the fourth ancestry, Indian Sea lineage.[38]

**Pathogenesis :-**

Although the cells and organs involved in viral replication have been discovered thanks to recent outbreaks, the pathogenesis of CHIKV infection in humans remains poorly understood. CHIKV directly enters the subcutaneous capillaries and infects susceptible skin cells following intradermal inoculation by infected mosquitoes: limited replication occurs in macrophages, fibroblasts, and endothelial cells.40 Locally produced viruses are transported to secondary lymphoid organs, where they infect migratory cells, release viruses into the lymph circulation, and then enter the blood.[20] Once in the blood, the virus can enter the liver, muscle, joints, and brain.[19] The infection is accompanied by a significant infiltration of mononuclear cells in these tissues. CHIKV infection elicits strong systemic innate responses, primarily involving the production of antiviral IFN- as well as a few pro-inflammatory cytokines, chemokines, and growth factors.[24]

This is followed by the activation of the adaptive immune system through the activation and proliferation of CD8+ T cells in the early stages of the disease.[32,28] Pain is associated with the infiltration of mononuclear cells and viral replication in muscles and joints. Later in the acute phase, there is a typical switch to a CD4+ T-cell response and the production of the anti-inflammatory proteins IL-1RA and IL-2RA. CHIKV infection causes a significant inflammatory response, which may be orchestrated by the production of IL-16, IL-17, monocyte chemoattractant protein 1 (MCP-1), IP-10, and MIP-1. The production of proinflammatory MIF, MIP-1, SDF1, IL-6, and IL-8 marks the end of the acute phase. CCL5, MCP-1, IP-10, MIP-1, and IL-8 are produced by activated macrophages that are susceptible to CHIKV infection.[30] These chemokines play a major role in leukocyte recruitment to infection sites and coordinate the deployment of effective antiviral defenses.

CCL5 (RANTES) levels were also high in all patients during the first week after symptom onset. Infection with CHIKV also triggers a robust cellular immune response. The presence of cellular responses was suggested by elevated plasma levels of IL-4, IL-7, and IL12p40, cytokines that support adaptive immunity.[14] Natural killer cells play a crucial role in the removal of infected cells. Furthermore, in the advancement of CHIKV arthralgia has likewise been suggested.[5]

The B cell-advancing cytokines IL-4 and now and again IL-10, were additionally up directed in the initial not many days after side effect beginning presumably starting the development of CHIKV-explicit IgG. Furthermore, CD4+ T lymphocytes, which are likewise associated with the advancement of humoral reactions, were emphatically actuated around the finish of the intense phase.[12] IgG antibodies are distinguished in the principal week after contamination, demonstrating fast seroconversion and elevated degrees of neutralizer reactions among CHIKV-contaminated individuals. Explicit IgM goes on for 3- - 4 months from the beginning of the sickness, and that IgG endures more than 6 months.[17] Nonetheless, their job in persistent arthralgia isn't very surely known.

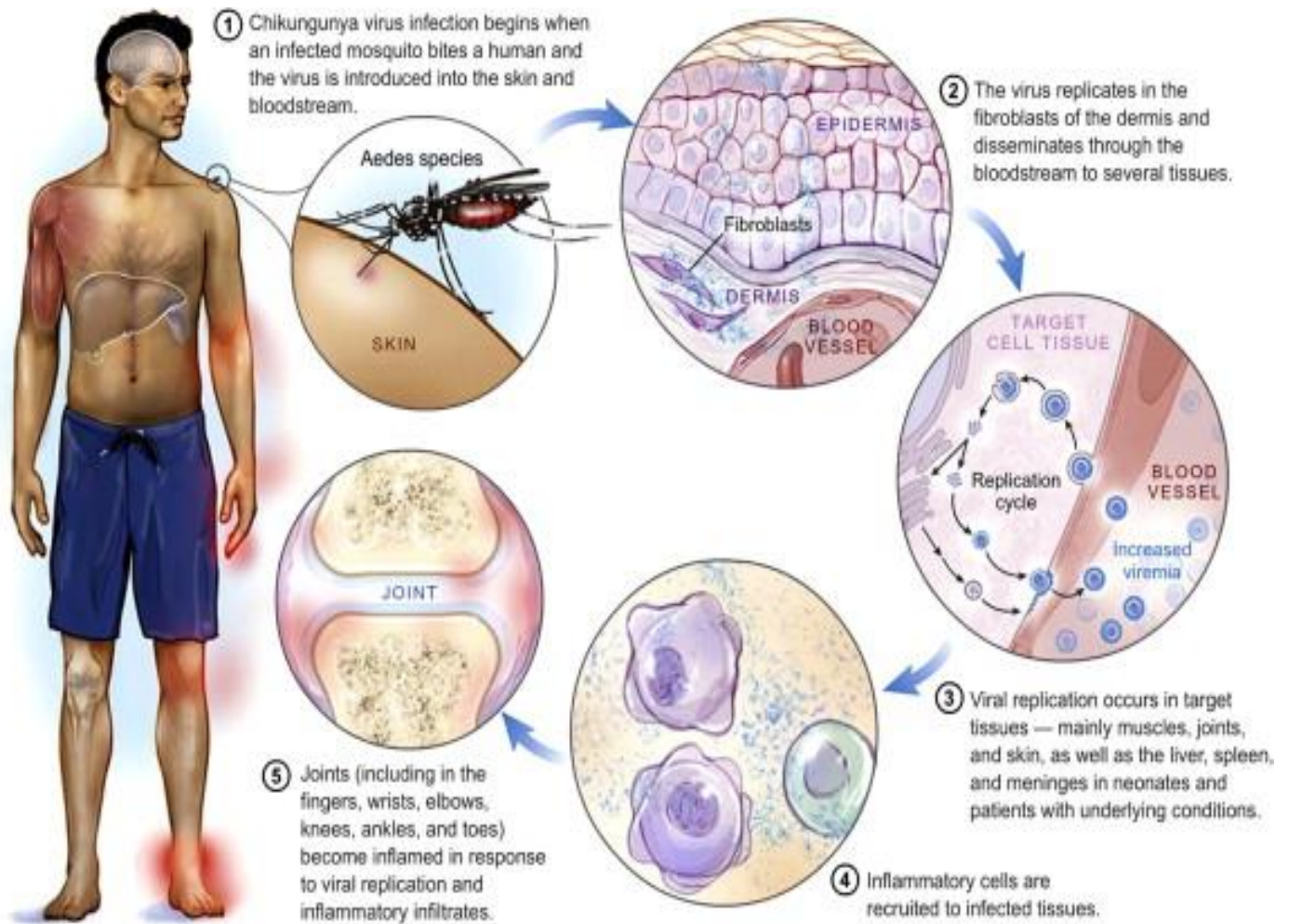


Fig. 5

### Clinical Manifestations :-

Polyarthralgia, a maculopapular rash, and an abrupt febrile illness are all symptoms of chikungunya fever. Studies conducted on infected patients during the La Réunion outbreak revealed that arthralgia was bilateral and symmetrical in 78.4% of the patients. The incubation period lasts two to four days (ranging from one to twelve days), and asymptomatic infections occur in five to fifteen percent of cases. 49—52 Rash was present in 54% of patients, primarily on the trunk and arms, and it mostly affected the ankles, knees, hands, wrists, feet, and elbows. [25] percent of patients reported experiencing periarticular edema, with an increased incidence affecting the ankles. 72% and 63% of the patients, respectively, had myalgia and headache. Iridocyclitis and retinitis are the most common ocular manifestations associated with CHIKV infection and have a benign course with complete resolution and preservation of vision.[15] The acute signs and symptoms typically resolve in less than two weeks, but arthralgia may last for weeks, months, or even years; [26,27] this is a clinical symptom that may distinguish CHIKV infection from dengue virus infection. Radiological findings are normal, and biological markers of inflammation like eryth In addition, in a univariate analysis, CHIKV-infected patients had fewer gastrointestinal symptoms than dengue-infected patients did, as well as a higher prevalence of myalgia and arthralgia.[9,14]

Chikungunya is generally not thought to be life-threatening. However, severe forms are also possible. Patients with severe chikungunya fever who need to be hospitalized are typically older and have comorbid conditions like diabetes, which are independent risk factors for severe disease.[29,26] Severe chikungunya can cause encephalopathy and encephalitis, myocarditis, hepatitis, and multiorgan failure. Neonates are also at risk for severe infection that is associated with neurologic signs, and these rare forms can result in death and typically affect patients who have other medical conditions.60,61 When neonates from viremic mothers are exposed to the virus during birth, the infection rate can reach 50%, resulting in severe illness and encephalopathy with long-term neurological effects and poor outcomes.

The prognosis for CHIKV infection is generally favorable. However, patients older than 45 are more likely to develop recurrent and persistent chronic rheumatic musculoskeletal pain.[13] A study found that subjects with severe initial rheumatic involvement (six or more painful sites with at least four other symptoms) were more likely to develop chronic rheumatic musculoskeletal pain on follow-up.63 Additionally, a positive association between high titers of CHIKV-specific IgG in the plateau phase and long-lasting arth 2 would direct the physician throughout the process of case definition and treatment. Palacios-Martinez's algorithm was adapted into this one. [38]

### Diagnosis :-

The diagnosis of chikungunya infection is based on clinical, epidemiological, and laboratory criteria. A possible CHIKV case is one with an acute onset of fever and severe arthritis that cannot be explained by other medical conditions.[20] There are three primary types of laboratory tests used to diagnose CHIKV infection: serology, virus isolation, and reverse transcriptase-

polymerase chain reaction Virus isolation can be carried out using acute serum specimens or mosquitoes collected in the field (less than 8 days). A susceptible cell line or suckling mouse can be inoculated with serum from whole blood collected during the first week of illness at a reference laboratory. Several RT-PCR assays for the detection of CHIKV RNA have been published.

This can be accomplished by transporting the sample cold (between 2 and 8 °C or dry ice) as soon as possible (within 48 hours).[22] Due to their reduced risk of contamination and increased sensitivity, real-time closed system assays should be used. PAHO recommends using the CHIKV RT-PCR protocols from the Centers for Disease Control and Prevention and the Institute Pasteur[14,70] in light of the virus's sensitivity. Both PCR testing and virus isolation are performed using serum taken from whole blood. In the enzyme-linked immunosorbent assay (ELISA), serum taken from whole blood is used for serological diagnosis. The serum (or blood) specimen should not be frozen during transport at temperatures between 2 and 8 °C. CHIKV-specific IgM antibodies or a four-fold increase in IgG titer in acute and convalescent specimens can be used to make a serologic diagnosis.[28] IgM can be determined using a variety of commercially available methods. However, it is important to keep in mind that techniques that make use of the entire virus as the antigen offer superior sensitivity to those that make use of recombinant proteins.

It is recommended that in-house methods for IgM/IgG ELISA be implemented using the purified viral antigen and adhering to the CDC protocols because the initial commercially available kits produced poor results.[30]

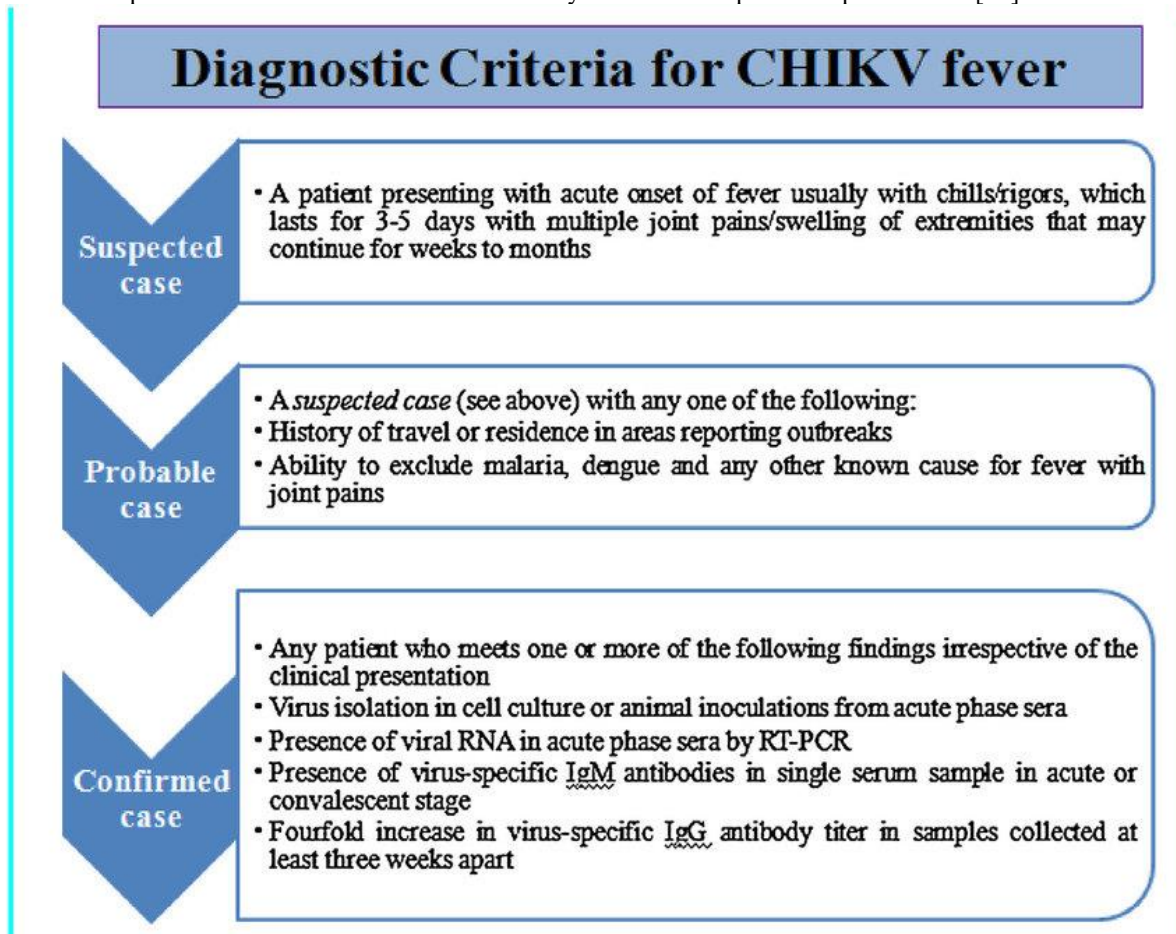


Fig. 6

#### Treatment :-

CHIKV infection cannot be treated with a specific antiviral medication. After excluding more serious conditions like malaria, dengue, and bacterial infections, symptomatic treatment is recommended.[20,21] In an acute infection, supportive treatment consists of rest and the use of acetaminophen to reduce fever (4 g/day). Even though recovery from CHIK is the anticipated outcome, convalescence can be prolonged, and persistent joint pain may require pain management, including long-term anti-inflammatory therapy, [16,11]. Ibuprofen, naproxen, or another non-steroidal anti-inflammatory agent (NSAID) can be used to relieve the arthritic component of the disease.[19,14] In patients with severe joint pains that are not relieved by NSAID, tramadol or narcotics Oral or topical nonsteroidal anti-inflammatory drugs (NSAIDs) and a brief course of oral corticotherapy or corticoid injections into the affected joint are the specific treatments for chronic diffuse post-CHIKV polyarthralgia. Tramadol, antiepileptic medications, and tricyclic antidepressants can be used to treat neuropathic pain.[23]

Although an earlier study suggested that hydroxychloroquine phosphate offered some benefit in arthralgia,74 subsequent studies failed to confirm its efficacy.[23,25,36] Alternative therapies like methotrexate (MTX) can be evaluated in patients with refractory joint symptoms. The specific treatments for chronic diffuse post-CHIKV polyarthralgia are nonsteroidal anti-inflammatory drugs (NSAIDs) taken orally or topically, as well as a brief course of oral corticotherapy or corticoid injections into the affected joint. Neuropathic pain can be treated with tramadol, antiepileptic medications, and tricyclic antidepressants.[17] Although an earlier study suggested that hydroxychloroquine phosphate offered some benefit in arthralgia, subsequent studies failed to confirm its efficacy.[26,35] Patients with refractory joint symptoms can be evaluated with alternative therapies like methotrexate (MTX).

**Prevention :-**

vaccine development, individual protection against mosquito bites and vector control are the only effective preventative measures. The most effective method for preventing CHIKV infection is mosquito control, which employs the same model as dengue control and has been relatively successful in many countries and settings<sup>80</sup>. In order to protect against the day-biting vectors, clothing that minimizes skin exposure is recommended. Breeding sites must be destroyed, frequently emptied, and cleaned or treated with insecticides.<sup>[29]</sup> In strict accordance with the instructions on the product label, repellents can be applied to clothing or skin that is exposed. DEET, also known as N,N-diethyl-3-methylbenzamide, IR3535, also known as 3-[N-acetyl-N-butyl]-aminopropionic acid ethyl ester, or icaridin, also known as 1-piperidinecarboxylic acid, 2-(2-hydroxyethyl)- 1-methylpropylester, should be included in repellents. Other insecticide vaporizers or mosquito coils may also reduce indoor biting.<sup>[33]</sup>



Fig . 7

**Vaccines :-**

Several technologies have been used to develop CHIK vaccines, including inactivated viral vaccines, live-attenuated viruses, alphavirus chimeras, recombinant viral vaccines, consensus-based DNA vaccines, recombinant subunit vaccines, and most recently, a virus-like particle (VLP) vaccine. However, there is currently no commercial vaccine for CHIKV.<sup>81,82</sup> Phase I trials for two vaccine candidates have concluded: a chikungunya vaccine based on live recombinant measles virus and the VRC-CHKVLP059-00-VP, VLP vaccine. The VLP vaccine, VRC-CHKVLP059-00-VP, was also immunogenic, safe, and well tolerated<sup>84</sup>. The live recombinant measles-virus-based chikungunya vaccine had good immunogenicity even in the presence of measles immunity, was safe, and had a generally acceptable tolerability profile.

**Economic Burden :-**

In 2006, the chikungunya pandemic in India resulted in a significant decline in the community's productivity as well as an enormous burden for epidemiologists. Public weight of chikungunya was assessed to be 25,588 DALYs lost during 2006 plague. Persevering arthralgia was found to force significant weight, representing 69% of the complete DALYs. The efficiency misfortune as far as pay predestined was assessed to be at least 6 million USD.<sup>85</sup> Different examinations made in India announced that the weight for Andhra Pradesh was 6600 DALYs (cost: US\$12,400,000). The burden was not too great, but the costs were high, and most out of pocket. A review made with military cops at La Réunion in June 2006 revealed that most suggestive patients (93.7%) whined of an ongoing phase of the sickness, which is described by torments in joints or bones, or both, albeit the request was made a half year after the pestilence top. With these antecedents, if the outbreak spreads throughout Mexico, the infected working adults will be incapacitated, which will increase the economic burden. Additionally, the majority of working adults suffer from depression, hand disability, and loss of mobility, all of which can last for weeks or months.<sup>[27,30]</sup>

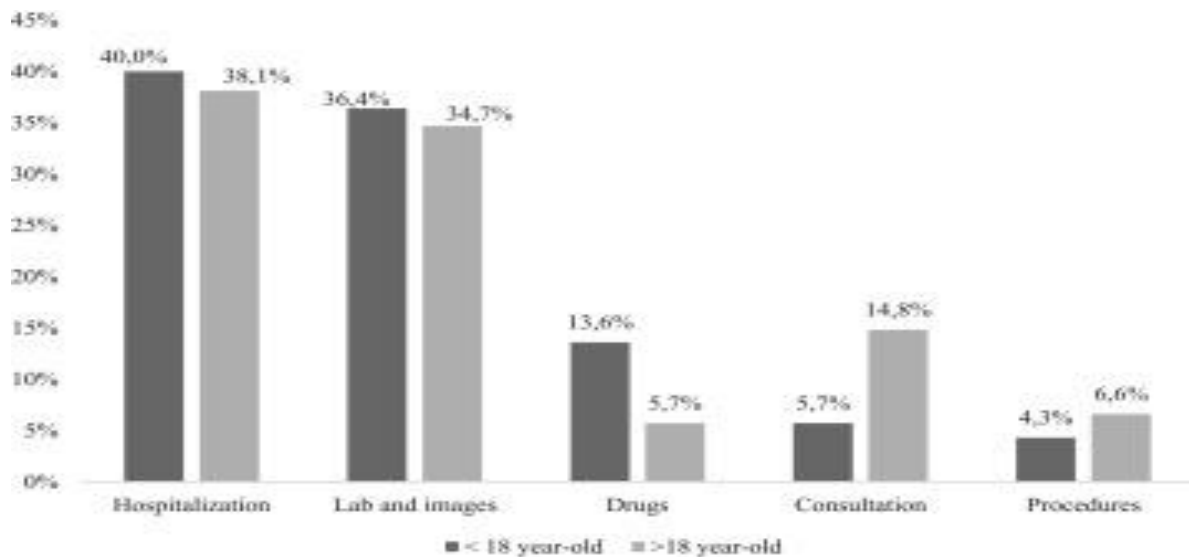


Fig. 8

### Conclusion :-

The introduction of CHIKV to the United States will present a significant financial and public health threat. Due to the prevalence of vectors, autochthonous transmission is very likely to occur elsewhere in Mexico and the United States. Countries are not shielded from diseases spread by vectors because of economic development; Travel, the aging of the population, and the production of solid waste that provides *Aedes* mosquitoes with a haven may amplify an epidemic. "Prevention is better than treatment" is a saying. This is profoundly huge in the event of chikungunya assault. The episode of chikungunya is currently a question of concern. There is a vaccine or a specific medicine that has not yet been discovered that can be used as a treatment. Although the pattern of treatment is like that of dengue, the severe pain is different. Chikungunya fever can be avoided by avoiding mosquito bites because this virus is carried by mosquitoes. Chikungunya virus is a member of the alphavirus family and is prevalent in Southeast Asia and Africa. The bites of *Aedes* mosquitoes can spread it to humans. Fever, headache, arthralgia, myalgia, and conjunctivitis are among the symptoms, which are comparable to those of dengue fever. These symptoms go away after two or three days, and a maculopapular rash appears. The fever might come back. Arthralgia can last for weeks or even months at a time. In most cases, antibody tests are used to make a diagnosis, but in the early stages of the disease, these tests may come back negative. when PCR can be used to make the diagnosis. Treatment is suggestive with analgesics and antipyretics.

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