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A Review On Chikungunya

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***Abstract*: This paper presents an overview on chikungunya.**

***Keywords*: Chikungunya.**

1. Introduction

Chikungunya is an infection caused by the Chikungunya virus (CHIKV). Symptoms include fever and joint pain. These typically occur two to twelve days after exposure. Other symptoms may include Headache, muscle pain, joint swelling, and a rash. Symptoms usually improve within a week; however, occasionally the joint pain may last for months or years. The risk of death is around 1 in 1,000. The very young, old, and those with other health problems are at risk of more severe disease. This is usually found in tropics and hence the reason why Chikungunya is predominantly seen in Asian countries. Anyone can get infected with Chikungunya virus. The virus infection can be seen in new born to old people. It is an RNA virus that belongs to the alphavirus genus of the family Togaviridae. The name "chikungunya" derives from a word in the Kimakonde language, meaning "to become contorted", and describes the stooped appearance of sufferers with joint pain (Arthralgia). Rash from chikungunya pronunciation specialty infectious disease symptoms Fever, joint pain complications long term joint pain usual onset 2 to 12 days after exposure duration usually less thana week.

The virus is spread between people by two types of mosquitos: Aedesalbopictus and Chikungunya. Rash from chikungunya pronunciation specialty infectious disease symptoms fever, joint pain complications long term joint pain usual onset 2 to 12 days after exposure duration usually less than a week.

The virus is spread between people by two types of mosquitos: Aedesalbopictus and Causes Chikungunya virus (CHIKV) spread by mosquito's Diagnostic method blood test for viral RNA or antibodies Differential diagnosis Dengue fever, Zika fever Prevention Mosquito control, avoidance of bites Treatment Supportive care Prognosis Risk of death ~ 1 in 1,000 Frequency.

2. History

Historically, chikungunya has been present mostly in the developing world. The disease causes an estimated 3 million infections each year. Epidemics in the Indian Ocean, Pacific Islands, and in the Americas, continue to change the distribution of the disease. In Africa, chikungunya is spread by a sylvatic cycle in which the virus largely cycles between other non-human primates, small mammals, and mosquitos between human outbreaks. During outbreaks, due to the high concentration of virus in the blood of those in the acute phase of infection, the virus can circulate from humans to mosquitoes and back to humans. The transmission of the pathogen between humans and mosquitoes that exist in urban environments was established on multiple occasions from strains occurring on the eastern half of Africa in non-human primate hosts. This emergence and spread beyond Africa may have started as early as the 18th century. Currently, available data does not indicate whether the introduction of chikungunya into Asia occurred in the 19th century or more recently, but this epidemic Asian strain causes outbreaks in India and continues to circulate in Southeast Asia. In Africa, outbreaks were typically tied to heavy rainfall causing increased mosquito population. In urban centers, the virus has spread by circulating between humans and mosquitoes. Global rates of chikungunya infection are variable, depending on outbreaks. When chikungunya was first identified in 1952.Beginning in the 1960s, periodic outbreaks were documented in Asia and Africa. However, since 2005, several decades of relative inactivity, chikungunya has re-emerged and caused large outbreaks in Africa, Asia, and the Americas. In India, chikungunya re-appeared following 32 years of absence of viral activity. Outbreaks have occurred in Europe, the Caribbean, and South America, areas in which chikungunya was not previously transmitted. Local transmission has also occurred in the U.S. and Australia, countries in which the virus was previously unknown. In 2005, an outbreak on the island of Réunion was the largest then documented, with an estimated 266,000 cases on an island with a population of approximately 770,000. In a 2006 outbreak, India reported 1.25 million suspected cases. Chikungunya was recently introduced to the Americas, and from 2013-14 in the Americas, 1,118,763 suspected cases and 24,682 confirmed cases were reported by the PAHO. Analysis of chikungunya virus's genetic code 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 as a vector in addition to the more strictly tropical main vector, Aedesaegypti. 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 co-infections. Chikungunya cases reported. 1952 - Outbreak of Chikungunya detected in Makonde Plateau. 1955 - Marion Robinson and W.H.R. Lumsden identified and described Chikungunya. 1963/64 - Chikungunya detected Indian cities mainly Calcutta, Maharashtra and Vellore.

3. Pathogenesis

The genus Alphavirus contains approximately 30 members, which probably diverged a few thousand years ago. Some alphaviruses are not pathogenic to humans, whereas others are highly infectious, with the associated clinical diseases ranging from mild to severe. Alphaviruses can be broadly divided into New world and Old-world viruses. These two groups have evolved distinct ways of interacting with their respective hosts and differ in their pathogenicity, tissue and cellular tropism, cytotoxicity and interference with virus-induced immune responses. It should be noted that most alpha viral infections in humans and domesticated animals are considered a 'dead end' — that is, the virus cannot be transmitted to a new host, so the evolutionary pressures driving viral diversification may be linked to their true host species. For CHIKV, a thorough exploration of other zoonotic viral reservoirs has not been carried out. From a clinical perspective, the two groups of alphaviruses are subdivided into those associated with encephalitis (predominantly New world viruses) and those associated with polyarthritis and a rash (predominantly Old world viruses). Although CHIKV is a member of the arthritogenicalpha viruses, during the recent outbreak there were documented cases of meningoencephalitis and hemorrhagic disease indicating that these signs are important sequalae of acute CHIKV infection. Unlike typical encephalogenicalpha viruses, which infect neurons, CHIKV seems to infect the stromal cells of the central nervous system and, in particular, the lining of the choroid plexus. Transmission of CHIKV occurs through a bite by infected Aedesaegypti or Aedes albopictus, although in the recent epidemic some cases were the result of maternal–fetal transmission. Following transmission, CHIKV replicates in the skin and then disseminates to the liver and joints, presumably through the blood. The incubation period is 2–4 days and is followed by a sudden onset of clinical disease with no prodromal phase. Symptoms of CHIKV infection include high fever, rigors, headache, photophobia and a petechial rash or maculopapular rash. In addition, most infected individuals complain of severe joint pain that is often in capacitating (see also the WHO guidelines on the clinical management of chikungunya). The cellular tropism of alphaviruses is regulated by many parameters. For example, RRV envelope glyco proteins allow the infection of mouse DCs but not human DCs67, and the ability of Sindbis virus (SINV) and VEEV69 to infect DCs is determined by a single amino acid substitution in the E2 envelope protein. Further work should examine the sensitivity of Langerhans cells to CHIKV and other alphaviruses. The use of rhabdo viruses and lenti viruses pseudo typed with CHIKV envelope glycoproteins may facilitate the study of early entry or post-entry events. Type I IFNs (IFNα and IFNβ) are also major regulators of tissue tropism and virulence. For example, they prevent the widespread dissemination of Semliki Forrest virus (SFV) in mouse extra neural tissues, and this is associated with reduced sensitivity to type I IFNs and enhanced virus pathogenicity. More generally, type I IFN induction in vivo, as well sensitivity to type I IFN treatment in cell culture, differs markedly between different alphaviruses. The interplay between CHIKV and the innate immune system is discussed below. Infected fibroblasts have also been reported in biopsy material taken from acutely infected patients. There is a debate about the sensitivity of primary blood monocytes to CHIKV infection. Sourisseau et al. reported that the high viral load in blood plasma (ranging from 105 to 108 RNA copies per ml) during acute infection does not correspond to detectable levels of viral RNA in blood cells. They also found that, in vitro, peripheral blood mononuclear cells (including B cells, T cells and monocytes) are not susceptible to CHIKV infection. By contrast, her et al. observed that CHIKV antigens are detected in vitro in monocytes exposed to high viral inocula (multiplicity of infection = 10–50). CHIKV antigen-positive monocytes were also isolated from acutely infected patients but definitive evidence of productive infection was not established. Acutely infected patients, the presence of negative-strand viral RNA must be assessed to determine whether productive infection of monocytes does occur and whether monocytes are true targets of CHIKV. There are notable cell tropism variations among alphaviruses, which probably influences the pathogenesis of disease. For example, human monocyte-derived dendritic cells (DCs) and plasmacytoid DCs (PDCs) are not sensitive to CHIKV Venezuelan equine encephalitis virus (VEEV) can infect DCs and macrophages in lymphoid tissues and cultures, whereas this is not the case for Eastern equine encephalitis virus (EEEV). Interestingly, EEEV infection of myeloid-lineage cells is restricted after virus binding and entry, by inhibiting translation of incoming EEEV genomes. Jumping species — an atypical vector for CHIKV is endemic to Africa, India and Southeast Asia and is transmitted to humans by several species of mosquito, with geographical variations. Although A. aegypti is the classical vector for CHIKV, the 2005 outbreak in La Réunion was associated with an atypical vector, A. albopictus. Other Aedes species are sensitive to experimental CHIKV infection, but their role in field transmission has not been shown. why did CHIKV adopt A. albopictus as its host? The transmission success of arboviral diseases depends on many

factors, including the geographical and temporal distribution of the insect vectors, their growth rate and the viral incubation period inside them. A. albopictus is a competent vector for dengue virus and numerous arboviruses, and its distribution has expanded recently, even replacing A. aegypti in some places. It is native to Southeast Asia and has colonized both tropical and temperate regions. It was identified in Europe (first in Albania) and in North America in the early 1980s, probably having been introduced through shipments of used car tyres from Asia. Currently, A. albopictus is present in at least European countries and in around 25% of the United States. There are several features of A. albopictus that make it a good viral vector: it survives in both rural and urban environments; it was probably first zoophilic and then progressively became anthropophilic it is long lived (4–8 weeks); it has a flight radius of 400–600 meters; and it can successfully infect humans and animals because it is aggressive, quiet and diurnal. Furthermore, the mosquito's eggs are highly resistant and can remain viable throughout the dry season, giving rise to larvae and adults.

4. Epidemiology

It is a mosquito-borne alpha virus that was first isolated after a 1952 outbreak in modern-day Tanzania. The virus has circulated in forested regions of sub-Saharan African in cycles involving nonhuman primate hosts and arboreal mosquito vectors. Phylogenetic studies indicate that the urban transmission cycle —the transmission of a pathogen between humans and mosquitoes that exist in urban environments—was established on multiple occasions from strains occurring on the eastern half of Africa in non-human primate hosts. This emergence and spread beyond Africa may have started as early as the 18th century. Currently, available data does not indicate whether the introduction of chikungunya into Asia occurred in the 19th century or more recently, but this epidemic Asian strain causes outbreaks in India and continues to circulate in Southeast Asia. A number of chikungunya outbreaks have occurred since 2005. An analysis of the chikungunya virus's genetic code suggests that the increased severity of the 2005– present outbreak may be due to a change in the genetic sequence, altering the virus' viral coat protein, which potentially allows it 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, Aedesaegypti. In July 2006, a team analyzed the virus' RNA and determined the genetic changes that have occurred in various strains of the virus and identified those genetic sequences which led to the increased virulence of recent strains.

A. Causes

Virus Realm: Riboviria Chikungunya virus (CHIKV), is a member of the genus Alphavirus, and family Togaviridae. It was first isolated in 1953 in Tanzania and is an RNA virus with a positive-sense single-stranded genome of about 11.6kb. It is a member of the Semliki Forest virus complex and is closely related to Ross River virus, O'nyong'nyong virus, and Semliki Forest virus.

B. Signs and symptoms

The incubation period ranges from one to twelve days, and is most typically three to seven. The disease may be asymptomatic, but generally is not, as 72% to 97% of those infected will develop symptoms. Characteristic symptoms include sudden onset with high fever, joint pain, and rash. Other symptoms may occur, including headache, fatigue, digestive complaints, and conjunctivitis. Sign and Symptoms. Information gained during recent epidemics suggests that chikungunya fever may result in a chronic phase as well as the phase of acute illness. Within the acute phase, two stages have been identified: a viral stage during the first five to seven days, during which viremia occurs, followed by a convalescent stage lasting approximately ten days, during which symptoms improve and the virus cannot be detected in the blood. Typically, the disease begins with a sudden high fever that lasts from a few days to a week, and sometimes up to ten days. The fever is usually above 39 °C (102 °F) and sometimes reaching 40 °C (104 °F) and may be biphasic—lasting several days, breaking, and then returning. Fever occurs with the onset of veremia, and the level of virus in the blood correlates with the intensity of symptoms in the acute phase. When IgM, an antibody that is a response to the initial exposure to an antigen, appears in the blood, viremia begins to diminish. However, headache, insomnia and an extreme degree of exhaustion remain, usually about five to seven days.

Following the fever, strong joint pain or stiffness occurs; it usually lasts weeks or months, but may last for years. The joint pain can be debilitating, often resulting in near immobility of the affected joints. Joint pain is reported in 87–98% of cases, and nearly always occurs in more than one joint, though joint swelling is uncommon. Typically, the affected joints are located in both arms and legs, and are affected symmetrically. Joints are more likely to be affected if they have previously been damaged by disorders such as arthritis. Pain most commonly occurs in peripheral joints, such as the wrists, ankles, and joints of the hands and feet as well as some of the larger joints, typically the shoulders, elbows and knees. Pain may also occur in the muscles or ligaments.

Rash occurs in 40–50% of cases, generally as a macula popular rash occurring two to five days after onset of symptoms. Digestive symptoms, including abdominal pain, nausea, vomiting or diarrhea, may also occur. In more than half of cases, normal activity is limited by significant fatigue and pain. Infrequently, inflammation of the eyes may occur in the form of iridocyclitis, or uveitis, and retinal lesions may occur. Temporary damage to the liver may occur. Rarely, neurological disorders have been reported in association with Chikungunya virus, including Guillain–Barré syndrome, palsies, meningoencephalitis, flaccid paralysis and neuropathy. In contrast to dengue fever, Chikungunya fever very rarely causes

hemorrhagic complications. Symptoms of bleeding should lead to consideration of alternative diagnoses or co-infection with dengue fever or coexisting congestive hepatopathy.

C. Chronic disease

Observations during recent epidemics have suggested chikungunya may cause long-term symptoms following acute infection. This condition has been termed chronic Chikungunya virus-induced arthralgia. Long-term symptoms are not an entirely new observation; long-term arthritis was observed following an outbreak in 1979. Common predictors of prolonged symptoms are advanced age and prior rheumatological disease. During the La Reunion outbreak in 2006, more than 50% of subjects over the age of 45 reported long-term muscular skeletal pain with up to 60% of people reporting prolonged painful joints three years following initial infection. A study of imported cases in France reported that 59% of people still suffered from arthralgia two years after acute infection. Following a local epidemic of chikungunya in Italy, 66% of people reported muscle pains, joint pains, or asthenia at one year after acute infection. Currently, the cause of these chronic symptoms is not fully known. Markers of autoimmune or rheumatoid disease have not been found in people reporting chronic symptoms. However, some evidence from humans and animal models suggests chikungunya may be able to establish chronic infections within the host. Viral antigen was detected in a muscle biopsy of a person suffering a recurrent episode of disease three months after initial onset. Additionally, viral antigen and viral RNA were found in macrophages in the synovial joint of a person experiencing a relapse of musculoskeletal disease 18 months after initial infection. Several animal models have also suggested Chikungunya virus may establish persistent infections. In a mouse model, viral RNA was detected specifically in joint associated tissue for at least 16 weeks after inoculation, and was associated with chronic synovitis. Similarly, another study reported detection of a viral reporter gene in joint tissue of mice for weeks after inoculation. In a nonhuman primate model, Chikungunya virus was found to persist in the spleen for at least six weeks.

D. Mechanism

Chikungunya virus is passed to humans when a bite from an infected mosquito breaks the skin and introduces the virus into the body. The pathogenesis of chikungunya infection in humans is still Mechanism. poorly understood, despite recent outbreaks. It appears that in vitro, Chikungunya virus is able to replicate in human epithelial and endothelial cells, primary fibroblasts, and monocyte-derived macrophages. Viral replication is highly cytopathic, but susceptible to type-I and -II interferon. In vivo, in studies using living cells, chikungunya virus appears to replicate in fibroblasts, skeletal muscle progenitor cells, and myofibers.

The type-1 interferon response seems to play an important role in the host's response to chikungunya infection. Upon infection with chikungunya, the host's fibroblasts produce type-1 alpha and beta interferon (IFN-α and IFN-β). In mouse studies, deficiencies in INF-1 in mice exposed to the virus cause increased morbidity and mortality. The chikungunya-specific upstream components of the type-1 interferon pathway involved in the host's response to chikungunya infection are still unknown. Nonetheless, mouse studies suggest that IPS-1 is an important factor, and that IRF3 and IRF7 are important in age dependent manner. Mouse studies also suggest that chikungunya evades host defenses and counters the type-I interferon response by producing NS2, a nonstructural protein that degrades RBP1 and turns off the host cell's ability to transcribe DNA. NS2 interferes with the JAK-STAT signaling pathway and prevents STAT from becoming phosphorylated.

In the acute phase of chikungunya, the virus is typically present in the areas where symptoms present, specifically skeletal muscles, and joints. In the chronic phase, it is suggested that viral persistence (the inability of the body to entirely rid itself of the virus), lack of clearance of the antigen, or both, contribute to joint pain. The inflammation response during both the acute and chronic phase of the disease results in part from interactions between the virus and monocytes and macrophages. Chikungunya virus disease in humans is associated with elevated serum levels of specific cytokines and chemokines. High levels of specific cytokines have been linked to more severe acute disease: interleukin-6 (IL-6), IL-1β, RANTES, monocyte chemoattractant protein 1 (MCP-1) monokine induced by gamma interferon (MIG), and interferon gamma-induced protein 10 (IP-10). Cytokines may also contribute to chronic Chikungunya virus disease, as persistent joint pain has been associated with elevated levels of IL-6 and granulocyte macrophage colony-stimulating factor (GM-CSF).[38] In those with chronic symptoms, a mild elevation of C-reactive protein (CRP) has been observed, suggesting ongoing chronic inflammation. However, there is little evidence linking chronic Chikungunya virus disease and the development of autoimmunity.

E. Diagnosis

Chikungunya is diagnosed on the basis of clinical, epidemiological, and laboratory criteria. Clinically, acute onset of high fever and severe joint pain would lead to suspicion of chikungunya. Epidemiological criteria consist of whether the individual has traveled to or spent time in an area in Diagnosis which chikungunya is present within the last twelve days (i.e.) the potential incubation period). Laboratory criteria include a decreased lymphocyte count consistent with viremia. However, a definitive laboratory diagnosis can be accomplished through viral isolation, RTPCR, or serological diagnosis. The differential diagnosis may include infection with other mosquito-borne viruses, such as dengue or malaria, and infection with influenza. Chronic recurrent polyarthralgia occurs in at least 20% of chikungunya patients one year after infection, whereas such symptoms are uncommon in dengue. Virus isolation provides the most definitive diagnosis, but takes

one to two weeks for completion and must be carried out in biosafety level III laboratories. The technique involves exposing specific cell lines to samples from whole blood and identifying Chikungunya virus-specific responses. RT-PCR using nested primer pairs is used to amplify several chikungunya-specific genes from whole blood, generating thousands to millions of copies of the genes in order to identify them. RT-PCR can also be used to quantify the viral load in the blood. Using RT-PCR, diagnostic results can be available in one to two days. Serological diagnosis requires a larger amount of blood than the other methods, and uses an ELISA assay to measure chikungunya-specific IgM levels in the blood serum. One advantage offered by serological diagnosis is that serum IgM is detectable from 5 days to months after the onset of symptoms, but drawbacks are that results may require two to three days, and false positives can occur with infection due to other related viruses, such as o'nyong virus and semliki forest virus. Chikungunya is diagnosed on the basis of clinical, epidemiological, and laboratory criteria. Clinically, acute onset of high fever and severe joint pain would lead to suspicion of chikungunya. Epidemiological criteria consist of whether the individual has traveled to or spent time in an area in Diagnosis which chikungunya is present within the last twelve days (i.e.) the potential incubation period). Laboratory criteria include a decreased lymphocyte count consistent with viremia. However, a definitive laboratory diagnosis can be accomplished through viral isolation, RTPCR, or serological diagnosis. The differential diagnosis may include infection with other mosquito-borne viruses, such as dengue or malaria, and infection with influenza. Chronic recurrent polyarthralgia occurs in at least 20% of chikungunya patients one year after infection, whereas such symptoms are uncommon in dengue.

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F. In other case

Chikungunya is diagnosed on the basis of clinical, epidemiological, and laboratory criteria. Clinically, Chikungunya begins as an acute febrile illness (high fever) and severe joint pain would lead to suspicion of chikungunya. Other common symptoms include headache, muscle pain, joint swelling, and rash. Epidemiological criteria consist of whether the individual has travelled to or spent time in an area in which chikungunya is present within the last twelve days (i.e. the potential incubation period). Laboratory criteria include a decreased lymphocyte count consistent with viremia. However, a definitive laboratory diagnosis can be accomplished through viral isolation, RT-PCR, or serological diagnosis. The differential diagnosis may include infection with other mosquito-borne viruses, such as dengue or malaria, and infection with influenza. Chronic recurrent polyarthralgia occurs in at least 20% of chikungunya patients one year after infection, whereas such symptoms are uncommon in dengue. Virus isolation provides the most definitive diagnosis, but takes one to two weeks for completion and must be carried out in bio-safety level III laboratories. The technique involves exposing specific cell lines to samples from whole blood and identifying chikungunya virus-specific responses. RTPCR using nested primer pairs is used to amplify several chikungunya-specific genes from whole blood, generating thousands to millions of copies of the genes in order to identify them. RT-PCR can also be used to quantify the viral load in the blood. Using RT-PCR, diagnostic results can be available in one to two days. Serological diagnosis requires a larger amount of blood than the other methods, and uses an ELISA assay to measure chikungunya-specific IgM levels in the blood serum. One advantage offered by serological diagnosis is that serum IgM is detectable from 5 days to months after the onset of symptoms, but drawbacks are that results may require two to three days, and false positives can occur with infection due to other related viruses, such as o'nyong'nyong virus and Semliki Forest virus. Presently, there is no specific way to test for chronic signs and symptoms associated with Chikungunya fever although nonspecific laboratory findings such as C reactive protein and elevated cytokines can correlate with disease activity.

G. Treatment

Currently, no specific treatment for chikungunya is available. Supportive care is recommended, and symptomatic treatment of fever and joint swelling includes the use of non-steroidalanti-inflammatory drugs such as naproxen, non-aspirin analgesics such as paracetamol (acetaminophen) and fluids. Aspirin is not recommended due to the increased risk of bleeding. Despite anti-inflammatory effects, corticosteroids are not recommended during the acute phase of disease, as they Treatment may cause immune suppression and worsen infection.

Passive immunotherapy has potential benefit in treatment of chikungunya. Studies in animals using passive immunotherapy have been effective, and clinical studies using passive immunotherapy in those particularly vulnerable to severe infection are currently in progress. Passive immunotherapy involves administration of anti-CHIKV hyperimmune human intravenous antibodies (immunoglobulins) to those exposed to

a high risk of chikungunya infection. No antiviral treatment for Chikungunya virus is currently available, though testing has shown several medications to be effective in vitro. CHRONIC ARTHRITIS. In those who have more than two weeks of arthritis, ribavirin may be useful. The effect of chloroquine is not clear. It does not appear to help acute disease, but tentative evidence indicates it might help those with chronic arthritis. Steroids do not appear to be an effective treatment. NSAIDs and simple analgesics can be used to provide partial symptom relief in most cases. Methotrexate, a drug used in the treatment of rheumatoid arthritis, has been shown to have benefit in treating inflammatory polyarthritis resulting from chikungunya, though the drug mechanism for improving viral arthritis is unclear.

H. Vaccine

As of 2017, no approved vaccines are available. A phase-II vaccine trial used a live, attenuated virus; to develop viral resistance in 98% of those tested after 28 days and 85% still showed resistance after one year. However, 8% of people reported transient joint pain, and attenuation was found to be due to only two mutations in the E2 glycoprotein. Alternative vaccine strategies have been developed, and show efficacy in mouse models. In August 2014 researchers at the National Institute of Allergy and Infectious Diseases in the USA were testing an experimental vaccine which uses virus-like particles (VLPs) instead of attenuated virus. All the 25 people participated in this phase 1 trial developed strong immune responses. As of 2015, a phase 2 trial was planned; using 400 adults aged 18 to 60 and to take place at 6 locations in the Caribbean. Even with a vaccine, mosquito population control and bite prevention will be necessary to control chikungunya disease.

I. Prevention

Because no approved vaccine exists, the most effective means of prevention are protection against contact with the disease-carrying mosquitoes and controlling mosquito populations by limiting their habitat. Mosquito control focuses on eliminating the standing water where mosquitos lay eggs and develop as larva; if elimination of the standing water is not possible, insecticides or biological control agents can be added. Methods of protection against contact with mosquitoes include using insect repellents with substances such as DEET, icaridin, PMD (p-menthane-3,8-diol, a substance derived from the lemon eucalyptus tree), or ethyl butylacetylaminopropionate (IR3535). However, increasing insecticide resistance presents a challenge to chemical control methods.

Wearing bite-proof long sleeves and trousers also offers protection, and garments can be treated with pyrethroids, a class of insecticides that often has repellent properties. Vaporized pyrethroids (for example in mosquito coils) are also insect repellents. As infected mosquitoes often feed and rest inside homes, securing screens on windows and doors will help to keep mosquitoes out of the house. In the case avoid getting bitten by mosquitoes. Keep the surroundings clear of stagnant water or pools where mosquitoes can breed. Use mosquito nets and close windows in the evening to prevent mosquitoes from entering. Because no approved vaccine exists, the most effective means of prevention are protection against contact with the disease carrying mosquitoes and controlling mosquito populations by limiting their habitat. Mosquito control focuses on eliminating the standing water where mosquitoes lay eggs and develop as larva; if elimination of the standing water is not possible, insecticides or biological control agents can be added. Methods of protection against contact with mosquitoes include using insect repellents with substances such as DEET, icaridin, PMD (p-menthane-3,8-diol, a substance derived from the lemon eucalyptus tree), or IR3535. However, increasing insecticide resistance presents a challenge to chemical control methods of the day-active A. aegypti and A.albopictus, however, this will have only a limited effect, since many contacts between the mosquitoes and humans occur outdoors.

5. Conclusion

Chikungunya is a viral fever caused by the bite of infected mosquito. The symptoms of the disease are high fever and joint pains. It takes time to get relief from pain. There is no allopathic medicine to cure this disease. There are certain ayurvedic and homeopathic medicines and different natural home remedies to cure the disease. The preventive measure is to avoid getting bitten by mosquitoes. Keep the surroundings clear of stagnant water or pools where mosquitoes can breed and use mosquito nets and close windows in the evening.

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