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Editorial

Chikungunya: A Neglected Tropical Disease

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Editorial

Chikungunya, a mosquito-transmitted viral disease endemic to tropical regions is recurring in epidemic waves over the last 15 years, with outbreaks becoming increasingly severe, infecting over one million people annually and causing debilitating joint pain. The virus caused outbreaks throughout the world with an ability to spread to non-endemic areas. The Chikungunya Virus (CHIKV) was first described during an outbreak in 1952 on the Makonde plateau, in the Newala district, along the border, between Mozambique and Tanganyika, the mainland part of modern-day Tanzania in East Africa. It was subsequently introduced into Asia and caused several periodical outbreaks in various African and South-East Asian countries (India and Thailand) since the 1960s. Though chikungunya virus was first isolated in 1952, its biology, transmission, and mechanism of pathogenesis has been poorly understood, and deserves more attention [1].

The name chikungunya is derived from the native *makonde* root verb or *kimakonde* word *kungunyala*, from Tanzania meaning “that which bends up”, “to become contorted”, or “to walk bent over”. Subsequently, several authors overlooked the references to the *Makonde* language and assumed the term have been derived from *Swahili* language, the *lingua franca* of the region [2].

Epidemics of chikungunya virus infection occurred in Africa, Thailand in 1958, and India, in Kolkata in 1963, and in South India 1964 (Pondicherry, Chennai, Vellore). The disease was endemic till 1973, along the East Coast of India and Maharashtra. Since then, it was clinically quiescent and no outbreaks were reported between 1973 and 2005, except for a few sporadic cases, which occurred in various places in the world including India (Maharashtra) [3].

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In 2005-2006, chikungunya reemerged in Indian Ocean Islands of Reunion affecting almost one-third of country's population where *Aedes albopictus* was the vector. This re-emergence in 2005 was due to a mutation (Alanine in the 226 position of E1 glycoprotein gene is replaced by valine) in the virus. This mutation led to a shift of vector preference, and mutated virus (E1-A226V strain) was found to be highly infective to Asian tiger mosquito *Aedes albopictus* (New vector) than *Aedes aegypti*. Following this re-emergence, chikungunya virus caused several outbreaks in India, other South-East Asian and African countries and spread to some areas of America and Europe [4].

At present, chikungunya has been identified in over 60 countries in Asia, Africa, Europe and the Americas. An outbreak of chikungunya in India in 2006 affected more than 1,500,000 cases transmitted by vector *Aedes aegypti* mosquito. At present, in India, chikungunya is endemic in several states including Karnataka, Tamil Nadu, Andhra Pradesh, Telangana, and West Bengal [3]. From 2014 to 2016, about 19,435 cases were reported in Colombia. A major outbreak in 2015 affected several countries of the Region of the Americas with 6,93,489 suspected cases and 37,480 confirmed cases of chikungunya being reported to the Pan American Health Organization (PAHO) [4].

Chikungunya virus belongs to the genus *Alphavirus*, in the family *Togaviridae*. Chikungunya virus has three genotypes Asian, West African, and East/Central/South African (ECSA), all named after their geographical distributions [5]. Virus particles are spherical with a diameter of 50-70 nm. Capsid is composed of 42 capsomers and genome is positive-sense single-stranded RNA of about 11.8 kb. It is an enveloped virus, the capsid being surrounded by a lipid envelope that contains two glycoproteins (E1 and E2) possessing haemagglutinating activity. The virus consists of four Nonstructural proteins (NS) and three structural proteins. The structural proteins are the capsid and two envelope glycoproteins (E1 and E2), which form 240 heterodimeric spikes on the virion surface. During replication, E2 binds to cellular cell adhesion molecule (Mxra8) and enter the host cell through receptor-mediated endocytosis. E1 glycoprotein contains a fusion peptide which causes membrane fusion that allows the release of nucleocapsids into the host cytoplasm, promoting infection [2].

Chikungunya virus is closely related to several other alpha viruses, including Ross River virus, Barmah Forest virus, O'nyong-nyong virus, Sindbis group of viruses, and Mayaro virus, all of which are known to cause arthritis. Because it is transmitted by arthropods (mosquitoes), it can also be referred to as an *arbovirus* (*arthropod-borne virus*). In the United States, it is classified as a category C priority pathogen, and work requires biosafety level III precautions [1].

Neonates and old people above 65 years and persons with underlying hypertension, diabetes or heart disease constitutes high risk group. The infection may be asymptomatic, but 72 to 97% of those infected will develop symptoms. After an incubation period of three to seven days, patient develops an abrupt onset of fever frequently accompanied by severe crippling joint pains (due to arthritis). 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. Arthritis is polyarticular, migratory and edematous (joint swelling), affecting the joints of wrists,

phalanges, ankles, knees, and lower back. The joint pain is often very debilitating, and can vary in duration, most patients recover fully within a week, but in some cases joint pain may persist for several months, or even years. Hence the virus can cause acute, subacute or chronic disease. Other common signs and symptoms include muscle pain, headache, nausea, fatigue, tenosynovitis, rash and slight photophobia. A maculopapular rash was common in 1963 Kolkata outbreak where the disease first appeared but hemorrhagic lesions were rare afterwards. Occasional cases of eye, neurological and heart complications have been reported, as well as gastrointestinal symptoms, including abdominal pain, nausea, vomiting or diarrhea [6-8].

The disease shares some clinical signs with dengue and zika, and can be misdiagnosed in areas where they are common. In general, chikungunya is less severe, less acute and hemorrhages are rare compared to dengue. Though chikungunya is not very fatal, the symptoms and chronic nature of the disease indicate that it should not be ignored. Biological markers such as IL-1 β and IL-6 are increased and RANTES (Regulated on Activation, Normal T-cell Expressed and Secreted) levels are decreased in chikungunya virus infection [6,7].

The virus is transmitted from human to human by the bites of infected female *Aedes aegypti* and *Aedes albopictus* mosquitoes, which can also transmit dengue virus. Chikungunya virus is maintained in nature through urban and sylvan transmission cycle. The urban cycle refers to transmission from human to mosquito to human, while sylvatic transmission is animal to mosquito to human. In urban cycle chikungunya virus is primarily maintained in more densely populated areas, in which humans act as the major reservoir hosts and mosquitoes of the genus *Aedes aegypti* and *Aedes albopictus* act as vectors. Sylvatic cycle occurs in African forests, where the virus is maintained between the wild primates e.g. monkeys, which serve as reservoir hosts and forest species of *Aedes* mosquitoes such as *A. furcifer*, *A. taylori* and *A. luteocephalus*, which serve as vectors. The virus may circulate within a number of animals including birds and rodents. Rarely, vertical transmission from mother to fetus and by blood transfusion also reported [7].

There is no specific antiviral drug treatment for chikungunya, but chikungunya is cured by immune system in almost all cases. Treatment is directed primarily at relieving the symptoms, including the joint pain using antipyretics and non-steroidal anti-inflammatory drugs, and maintaining proper fluid levels. In those who have more than two weeks of arthritis, ribavirin may be useful [6,7].

There is no chikungunya vaccine. The best means of prevention is overall mosquito control and the avoidance of mosquito bites. Recently, a few vaccine trials are ongoing for chikungunya. MV-CHIK, and VRC-CHKVLP059-00-VP vaccines have successfully completed phase I clinical trials. The MV-CHIK vaccine is a recombinant measles virus (Shwartz strain) that expresses chikungunya virus surface proteins. VRC-CHKVLP059-00-VP is a Virus-Like Particle (VLP) vaccine [8].

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