

An overview: chikungunya fever



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Introduction

The derivation of chikungunya came from Tanzania, when there were reports of a dengue-like symptom outbreak in 1952-1953, in the Newala and Masasi Districts of the Southern Province. However, dengue was later excluded on the basis that this epidemic involved patients suffering from debilitating joint pains and shorter incubation period which are not clinical features of dengue. Consequently, the illness was called chikungunya; (Robinson 1955) a word from the Makonde dialect describing patient's contorted posture (Lumsden 1955). Chikungunya is an arthropod borne virus (arbovirus) of the genus: Alphavirus from Togaviridae family. It is transmitted to humans mainly by day biting mosquito species *Aedes aegypti* and *Aedes albopictus* (Townson and Nathan 2008). Chikungunya virus (CHIKV) contains a positive-sense single stranded RNA genome, enclosed in an icosahedral nucleocapsid (combination of nucleic acid and capsid) all enclosed in a phospholipid bilayer envelope. Embedded in the envelope are multiple copies of two encoded glycoproteins E1 and E2, a small glycoprotein E3, and a hydrophobic peptide 6K (Strauss and Strauss 1994).

History

There were no previous reported cases of chikungunya or dengue in Tanzania before the chikungunya epidemic in 1952 – 1953 thus, the inhabitants were highly susceptible in acquiring CHIKV infections (Robinson 1955). Prescott et al, 2002 stated that herd immunity; that is, the immunity that is acquired by a large proportion of the population either through vaccination, or from previous infections, thereby rendering the population resistant to the spread of the diseases. Furthermore, other factors could

have contributed to the epidemic including records of unusually high rainfall during February to May 1952. Consequently, this condition was thought to provide suitable breeding environment for the mosquitoes to expand their population, suggesting that the main vector of transmissions were mosquitoes (Lumsden 1955). However, not only high rainfall was implicated in enhancing mosquitoes breeding environment, but also water filled clay jars dug in the inhabitants' huts floor in preparation for the drought season. Consequently, these jars are rarely completely emptied, which further enhanced mosquitoes populations. Another contributing factor to the epidemic could be the living conditions and lifestyles of the indigenous populations. Animals such as fowl, pigeons and occasionally goats were reared in the inhabitant's huts. Consequently, these vertebrates could also serve as CHIKV hosts, and thus, this has provided opportunities for transmissions between animals and humans. During early January 1953, the incidence of the epidemic has reached its peak (Lumsden 1955). However, once a person has acquired the infection, that person will become immune to further infections with the same virus (Robinson 1955). Subsequently, this means that as herd immunity increases, the number of viraemic inhabitants decreases.

Aim

Consequently, it has inspired this literature review on chikungunya in assessing its outbreaks incidence and prevalence, its associations with dengue and the vectors. Furthermore, from this to determine if it poses a risk that Western medicine should be planning for.

Clinical Features

Chikungunya has a viraemic incubation period of 3-12 days (Robinson 1955). Rezza et al. (2007) described chikungunya as a mild and self limited disease in most of the patients. with patients presenting with clinical features such as fever ranging from 39-39. 8oC, fatigue, skin rash (sometimes itches), headache, joint pain (arthralgia), muscle pain (myalgia), diarrhoea, vomiting, photophobia, and conjunctivitis. Furthermore, Kannan et al. (2009) observed most of the above symptoms with inclusion of oedema, oral ulcers, nausea, and haemorrhage. Arthralgia and myalgia involves extremities such as wrists, ankles, hands, feet and phalanges. However, arthralgia, myalgia, oedema, lethargy, and weakness persisted even after fever had subsided (Kannan et al. 2009). However, the La Reunion outbreak observed vertical transmissions from mother to child, with newborns presenting with chikungunya infection without prior involvement of mosquitoes. These infants presented with fever, pain, poor feeding, disseminated intravascular coagulation (DIC), petechiae, distal joint oedema, and thrombocytopenia. Also involved were severe neonatal infections including encephalitis and haemorrhagic fever. Furthermore, there were cases whereby transient brain haemorrhages were complicated by the presence of DIC (Gérardin et al. 2008).

Table 1: represents different clinical features that were associated with chikungunya infection during several outbreaks.

Clinical Features associated with Chikungunya				
Most common	Joint pain (96. 6%)	Fever	Fever	Fever
	Fever (96. 3%)	Joint pain	Joint pain	Arthralgia

	Headache (71. 2%)	Arthritis	Fatigue	Myalgia
	Muscle pain (62. 6%)	Myalgia	Skin rash	Headache
	Cutaneous eruptions (32. 5%)	Arthralgia	Headache	Anorexia
		Headache	Muscle pain	Nausea
		Rash	Diarrhoea	Itch/ Rash
		Sore throat	Itch/ Rash	Oedema
			Vomiting	Oral ulcer
			Photophobia	Eye pain
			Conjunctivitis	Vomiting
				Eye congestion
Least common				Haemorrhage
Reference	Renault et al. 2007	Yosulf et al. 2008		Kannan et al. 2009

Distribution of Chikungunya

The World Health Organisation (WHO) alerted the Global Alert and Response Network in March 2005, of an impending outbreak of chikungunya fever in the Comoro Islands. It was the first Southwestern Indian Ocean islands to be implicated in the epidemic, subsequently; other countries were later implicated in this epidemic. The emergence of the epidemic in Mayotte was imported from Grande-Comore by mid-April and by the end of April Mauritius was also implicated. Overall, the Southwestern Indian Ocean region was believed to be afflicted by the first emergence of CHIKV (Renault et al. 2007).

In April 2005, a chikungunya infection which started in Grande-Comorre was imported into La Reunion (French Overseas Territory), became its first severe reported case, in March 2004 to April 2005. It resulted in the establishment of a wide operational epidemiologic surveillance system by the island's local authority with the following objectives “ monitor epidemic trends, characterise cases and detect new transmission clusters to provide orientations for prevention and vector control”. Unfortunately, the collection of data was not conducted scientifically, as the figures of reported cases were not scientifically correlated (Renault et al. 2007). Moreover, there were also flaws in the correlation of the epidemic curve as the capacity of the surveillance system was not sufficient to evaluate the number of cases. Consequently, this resulted in an underestimated number of reported cases. (Renault et al. 2007). Approximately 3% cases did not correspond with the criteria defining suspected cases as an abrupt onset of fever $> 38.5^{\circ}\text{C}$ accompanied by debilitating joint pain. However, Renault et al. (2007) argued that these were mainly due to some atypical cases that were

confirmed by laboratory testing and also due to errors in reporting cases. Moreover, there could have been misdiagnosis of chikungunya infection as not all of the atypical cases were laboratory confirmed; and also the previous year there were outbreak of dengue. By April 2006, 203 deaths which were either directly or indirectly attributed to chikungunya infection with the mortality rate of 0.3/1000 people with a median age of 79. Consequently, the number of deaths was minimal. Moreover, the low immune status of the individual as indicated by the age could have been a contributing factor to the death, as some of these individuals were presented with other underlying conditions. However, direct association between death and infection was not confirmed due to the absence of autopsies, consequently; this made it difficult to assess the extent chikungunya virus had on death.

Mayotte, an island of the Comoros archipelago was involved in the first CHIKV outbreak in April 2005 to July 2006 affecting approximately 6346 residents. The outbreak involved two waves, with peak occurring in the second wave. In the Maldives, an outbreak occurred during December 2006 to April 2007 with 11879 confirmed and suspected cases. Out of the 197 inhabited Maldives islands 121 islands reported CHIKV. The epidemic was thought to be associated with unusually high rainfall from October 2006 to March 2007 and post tsunami construction activities which provided breeding sites for mosquitoes. (Yoosuf et al. 2008).

On the east coast of Madagascar, in Toamasina, Chikungunya virus and Dengue type 1 virus outbreak were reported during January to March 2006. The study involved interrogating 4,242 residents in 27 neighbourhoods, of which 2,863 were suspected cases and of these 44 were hospitalised cases.

55 patients serum were sampled after passing the criteria which included having less than five days of fever as well as three of these symptoms: headache, myalgia, arthralgia, retroorbital pain or rash. The results included 24 dengue patients, 4 chikungunya and 10 co-infections. As the study was only based on clinical investigations, and only a few samples were analysed, there were insufficient investigation to measure the Chikungunyas' and Dengues' contributions in the 2 peaks of the epidemic curve. Furthermore, 2 of the 24 Dengue patients had IgM Chikungunya virus. This could be caused by false positive or false negative results, resulting in misdiagnosis or they were in fact co-infections (Ratsitorahina et al. 2008).

The cases represented on the map are either confirmed cases or suspected cases.

Reference: 1 Krastinova et al. 2006, 2 Rezza et al. 2007, 3 Pastorino et al. 2004, 4 Si al. 2008, 5 Lumsden 1955, 6 Tamburro and Depertat 2009, 7 CDC 2009, 8 WHO 2009, Yoosuf et al. 2009.

Transmission of CHIKV

CHIKV requires an invertebrate and vertebrate host in order to complete its replication cycle (Pardigon 2009). Invertebrate hosts also known as definitive hosts are mainly the Aedes mosquito species. The Aedes mosquito becomes infected subsequent to ingesting viraemic blood meal from infected vertebrate hosts. The blood meal containing CHIKV travel to the gut, there CHIKV undergo replication within the gut wall. Subsequently, CHIKV contain mechanisms permitting its penetration into the mosquitoes' tissues. From there, it passes through either the bloodstream or by other methods (not well known) to different sites of the body such as the salivary glands where it

undergoes further replication. Extrinsic incubation period (EIP) is defined as the time taken for the vector to transmit CHIKV after ingesting a viraemic blood meal. Subsequently, there are factors that could influence the length of the EIP including temperature which increases when the EIP decreases. This is thought to be an inverse proportional relationship. Moreover, the quantity of viraemic blood ingested is another factor (Cook and Zumla 2009). Furthermore, Ross (1956) research demonstrated that a mosquito could harbour viral particles for up to 30 days. Gould and Higgs (2009) suggested that vertical transmission of CHIKV from infected mosquitoes result in infected mosquitoes' eggs. These eggs, due to their desiccated nature, are able to survive for long period of time in the environment where it hatches during the rainy season. Sylvatic transmission cycle involves vertebrate hosts such as primates, birds, and rodents which serve as natural hosts (Pardigon 2009). However, humans were considered accidental hosts, resulting in urban transmission cycles producing epidemics. After taking a viraemic bloodmeal from an infected human the domestic mosquitoes can also become infected thereby contributing to the outbreaks (Gould and Higgs 2009).

Distributions of *Aedes albopictus* and *Aedes aegypti*

The main vectors of chikungunya are *Aedes aegypti* and *Aedes albopictus*. The vector involved in the 2005/2006 Mayotte outbreak was *Aedes albopictus* which resided locally on the island (Sissoko et al. 2008). *Aedes albopictus* was involved in the 2006 Dengue fever and Chikungunya outbreak in Madagascar. The mosquito was found in drums, buckets, coconut shells, discarded cans, pots wet containers and tyres (Ratsitorahina et al.

2008). In the 2007 Italian outbreak, the only vector present was *Aedes albopictus* (Rezza et al. 2007). The main vector associated with recent outbreaks was *Aedes albopictus*.

Effect of climate change

Most of the cases occurred after reports of high rainfall.

Association with Dengue Fever and Malaria

In the 1999-2000 Democratic Republic of Congo outbreak, patients had acquired simultaneous CHIKV and *Plasmodium falciparum* infection.

However, there were not sufficient evidence to prove co-infection between the two but it was assumed by the authors that it was probable (Pastorino et al. 2004). Compared to CHIKV, dengue virus is of the genus *Flavivirus* from *Flaviridae* family and contains 4 serotypes (Dayal-Drager 2004 cited by Seyler et al. 2009). The 2006 Madagascar outbreak showed that the mosquito can harbour both Chikungunya and dengue virus (Ratsitorahina et al. 2008). CHIKV is often masked by dengue fever due to similar clinical features. Consequently, a study by Vazeille et al. (2008) demonstrated that *Aedes aegypti* has a higher susceptibility in Dengue 2 virus and a lower susceptibility to CHIKV. Moreover, studies by Vazeille et al. (2008) and Moutailler et al. (2009) demonstrated that *Aedes albopictus* are more efficient at harbouring CHIKV than dengue 2 virus.

Importation into Europe

France

Between March 2005 to August 2006, 80 visitors from Chikungunya infected countries in the Southwest Indian Ocean region (La Reunion, Mauritius,

Mayotte, Comoros and India) confirmed chikungunya infection at Pitié-Salpêtrière Hospital in Paris, France (Hochedez et al. 2007).

Italy

In 2007, a massive CHIKV outbreak occurred in the Emilia Romagna region in northeastern Italy. There were 205 identified cases with febrile illness reported in Castiglione di Cervia and Castiglione di Ravenna villages (171) separated by the river in Ravenna province, and Cervia (13) and other villages (21), between July 4 to September 27. The epidemic was believed to be imported from Kerala, India (CHIKV epidemic infested area) by a man, who after two days of his arrival in Castiglione di Cervia developed febrile illness. The majority of the cases were the elderly (median age 60) representing increased incidence with age. *Aedes albopictus* which causes Chikungunya was also found in the area of the epidemic which further propagated the virus. Positive CHIKV sequences were detected in 90 captured *Aedes albopictus* mosquitoes from Castiglione di Cervia and 125 from Castiglione di Ravenna. Most cases occurred in Castiglione di Cervia and Castiglione di Ravenna Villages with peak incidence during third week of August. The 13 Cervia cases were local transmission introduced from Castiglione through migration (Rezza et al. 2007).

Other European Countries

Due to travelling, several European countries have encountered CHIKV from viraemic travellers from epidemic countries (Panning et al. 2008). The study was conducted at the Bernhard-Nocht Institute for Tropical Medicine in Hamburg, Germany involved 680 patients.

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