

Original Research Article

A Study of Prevalence of Chikungunya among Children: A Cross-Sectional Study

Girish Shakuntal¹, Prasanna Parashare²

¹Asst professor, Dept of paediatrics, SMBT Institute of medical Sciences & Research Centre, Dhamangaon Tal, Igatpuri, Dist. Nasik

²Senior Resident, Dept of paediatrics, SMBT Institute of medical Sciences & Research Centre, Dhamangaon Tal, Igatpuri, Dist. Nasik

***Corresponding author**

Girish Shakuntal

Email: faridi17@rediffmail.com

Abstract: Chikungunya is a mosquito-borne, viral, acute febrile illness that can be difficult to distinguish clinically. Since the incidence of this disease is increasing, it is necessary to study the incidence and prevalence of the disease to find out the measures to stop the spread of the disease. The study was done as cross-sectional study including of 450 children of the nearby rural as well as urban area. The study was done over a period of 6 months and the children who had chikungunya were considered for the study. The clinical signs and symptoms typical for the diagnosis of chikungunya were only considered for the study. Out of 450 children, 39 (8.6%) had chikungunya in their childhood. Through the recent epidemics, chikungunya has demonstrated its ability to spread and infect large proportions of the population. Therefore measures should be initiated to control the spread of the disease.

Keywords: Chikungunya, Children, Infection

INTRODUCTION

The chikungunya virus (CHIKV) is an enveloped, positive stranded RNA alphavirus belonging to the Togaviridae family and transmitted by Aedes mosquito bites. It causes a dengue-like illness, characterized by fever, rash, painful myalgia, and arthralgia, and sometimes arthritis. It was first isolated by R.W. Ross in 1952 in the Newala district of Tanzania. Its current geographic distribution covers sub-Saharan Africa, Southeast Asia, India, and the Western Pacific where numerous outbreaks have been reported. In these areas, upsurges of re-emergence occur at intervals of 7 to 20 years [1-4].

Chikungunya and dengue are mosquito-borne, viral, acute febrile illnesses that can be difficult to distinguish clinically [5].

Chikungunya virus is an arthropod-borne virus that is transmitted by Aedes mosquitoes. The virus transmission cycle requires infection of female mosquitoes via a viraemic bloodmeal taken from a susceptible vertebrate host and, following a suitable extrinsic incubation period, transmission to another vertebrate host during subsequent feeding [6].

Since the end of 2004, CHIKV has emerged in the Southwestern Indian Ocean islands. Between January and March 2005, over 5,000 cases were reported in the Comoros. Later in 2005, the virus spread to other islands, including Mayotte, Seychelles, La Reunion, and Mauritius. In La Reunion Island, a French overseas department (total population: 787,836), the first declared case was observed in Saint-Pierre (southern area of the island) in the beginning of March 2005 among people returning from the Comoros. The transmission was moderate until the rainy season, which started in December in 2005 and was associated with an epidemic of unprecedented magnitude (300,000 cumulative cases on December 30, 2006), with a peak incidence reached on the fifth week of 2006 (over 45,000 cases). No other known arboviral disease was associated with this chikungunya outbreak [10].

Aedes albopictus was identified as the only vector of a principally urban transmission. During this outbreak, severe or complicated forms of chikungunya were reported in adult patients, including encephalopathy and hemorrhagic fever, which frequently occurred in the context of chronic diseases or

underlying conditions such as diabetes mellitus, chronic obstructive pulmonary disease, ischemic heart disease, chronic renal failure, or alcoholic hepatopathy [4, 9].

Thus conducted a cross-sectional study in order to characterize the epidemiological, clinical, biological, and radiological features and outcomes of all the cases of child chikungunya infections recorded at our institution [4].

MATERIALS AND METHODS

The study was done as cross-sectional study including of 450 children of the nearby rural as well as urban area. The study was done over a period of 6 months and the children who had chikungunya were considered for the study. The clinical signs and symptoms typical for the diagnosis of chikungunya were only considered for the study. Cases in doubt were excluded from the study. Approval of the ethical committee was taken before start of the study and informed consent was also obtained from the parents of each children. All the findings were recorded and the percentage analysis was done.

RESULTS

Out of 450 children, 39 (8.6%) had chikungunya in their childhood. The diagnosis of the condition was made from the relevant history of the patient.

DISCUSSION

There is a confirmed history of outbreaks during 1963–64 in Kolkata¹⁵ (earlier known as Calcutta) and 1965 in Chennai (earlier known as Madras) when more than 3, 00,000 people were affected. Last epidemic in India was reported from Barsi, Maharashtra in 1973 when a morbidity of 37.5% was reported for the whole town [8, 10].

Chikungunya virus is transmitted by culicine mosquitoes and can alternatively affect vertebrates and arthropods. The arthropods remain infected throughout all its life. Its transmission to humans is mainly through *Aedes* species mosquitoes. *Aedes aegypti*, *Aedes albopictus* and *Aedes polynesiensis* are commonly involved in the transmission although *Culex* has also been reported for the transmission in some cases [11, 12].

Chikungunya was first detected in 1952 in Makonde, United Republic of Tanzania (formerly Tanganyika) and derives its name from kungunya, the Swahili word for the contorted posture of patients because of their arthritic symptoms. It was first described by Robinson and Lumsden in 1953. Epidemics were subsequently noted in the Philippines (1954, 1956 and 1968), Thailand, Cambodia, Viet Nam, India, Myanmar and Sri Lanka [8]. In India, major

epidemics of chikungunya were reported in 1963 in Kolkata, in 1965 in Pondicherry (formerly Pondicherry), Tamil Nadu, Andhra Pradesh, Madhya Pradesh and Maharashtra and again in 1973 in Maharashtra [13].

The infection is of acute onset and variable clinical features are common findings. The symptoms develop after an incubation period of 4 to 7 day (Incubation period lies between 1 and 12 days). In most of the cases the disease is self-limiting and the symptoms disappear within 5 to 7 days even without treatment. Rarely the symptoms may persist for a longer period and occasionally complications may develop [8].

A clinical triad of ‘fever, rashes and arthralgia’ is suggestive of chikungunya fever. The clinical features vary from high fever (more than 40°C, rapid in rise and sometimes associated with rigor), severe headache, chills and rigors, nausea and vomiting. The fever may disappear to return in one or two days giving it the name of ‘Saddle back fever’ [8].

Recent Indian study reported transmission of chikungunya virus by *Anopheles stephensi* too. The Indian Ocean outbreak is caused by transmission by *Aedes* only. The common reservoirs for chikungunya virus are monkeys and other vertebrates. The role of cattles and rodents has also been reported in the transmission of the virus. The CHIKV usually shows a periodicity with occurrence of disease in the community with latency intervals of 3-4 years, probably due to its cycle in monkeys. Following transmission, CHIKV replicates in the skin, and disseminates to the liver, muscle, joints, lymphoid tissue (lymph nodes and spleen) and brain, presumably through the Blood [14].

Typically, joint damage fluctuates over time, but always affects the same parts of the body, mostly the extremities (hands, ankles, knuckles). The mortality rate is low (0.4%), but is higher in babies less than 1 year old (2.8%) and increases in the elderly with concurrent diseases [14].

Pathogenesis

Detailed studies are not available on the pathogenesis of the chikungunya fever. It is expected that once after inoculation, primary viral multiplication occurs in lymphoid and myeloid cells. The arthropod vectors acquire the virus by sucking blood during this period. The virus then spreads to the targeted organs and immune system starts functioning at this stage leading to the activation of both humoral and cellular immunity. This response of the body leads to the clinical features of the disease. The convincing evidence and studies are not available [8].

Laboratory diagnosis relies upon the detection of the virus on early samples and/or specific anti-CHIKV IgM and IgG on blood samples. Commercial kits are available, sometimes with excellent sensitivity and specificity. For example the commercial Chikungunya virus real-time reverse transcription-PCR (RT-PCR) kit, by Panning *et al.*, was 100% sensitive and specific in comparison to a published real-time RT-PCR. This commercial CHIKV kit may assist laboratories in affected regions and serve the needs of outpatient travel medicine clinics worldwide. The capability of quantifying virus RNA concentrations may facilitate the monitoring of disease progression and the assessment of risks of transmission in the nosocomial situation. In addition, this kit may help in regions where CHIKV vectors *Aedes aegypti* and *Aedes albopictus* are subject to virus surveillance [14].

CONCLUSION

Through the recent epidemics, chikungunya has demonstrated its ability to spread and infect large proportions of the population. There is a very good chance this disease will continue to spread unless measures are taken to improve the recognition of the disease, to control the vectors responsible for the transmission, and to rapidly communicate epidemiological information to vector control experts and other public health officials. Hopefully, timely sharing of accurate information will help control the spread and magnitude of future outbreaks.

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