Guest Editorial

RE-EMERGENCE OF CHIKUNGUNYA VIRUS IN INDIA

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Chikungunya virus is no stranger to the Indian subcontinent. Since its first isolation in Calcutta,¹ in 1963, there have been several reports of chikungunya virus infection in different parts of India.²⁻⁴ The last outbreak of chikungunya virus infection occurred in India in 1971. Subsequently, there has been no active or passive surveillance carried out in the country and therefore, it 'seemed' that the virus had 'disappeared' from the subcontinent.⁵ However, recent reports of large scale outbreaks of fever caused by chikungunya virus infection in several parts of Southern India have confirmed the re-emrgence of this virus.⁶⁻⁸ It has been estimated that over 1,80,000 cases have occurred in India since December 2005. Andhra Pradesh (AP) was the first state to report this disease in December 2005, and one of the worst affected (over 80,000 suspected cases). Several districts of Karnataka state such as Gulbarga, Tumkur, Bidar, Raichur, Bellary, Chitradurga, Davanagere, Kolar and Bijapur districts have also recorded large number of chikungunya virus related fever cases. Over, 2000 cases of chikungunya fever have also been reported from Malegaon town in Nasik district, Maharashtra state, India between February-March 2006. During the same period, 4904 cases of fever associated with myalgia and headache have been reported from Orissa state as well.8 According to the National Institute of Virology, Pune, out of 362 samples collected from different places in AP such as Kadapa, Secunderabad, Chittoor, Anantapur, Nalgonda and Prakasam, Kurnool and Guntur districts, 139 were found positive for chikungunya, antibodies, six for dengue fever and 15 related to both the fevers. Apart from India, several small countries in the southern Indian Ocean such as the French Reunion Islands, Mauritius, Seychelles and other countries have also been reporting large scale outbreaks of chikungunya virus infection this year.

Chikungunya is a relatively rare form of viral fever caused by an alphavirus that is spread by bite of an infected *Aedes aegypti* mosquito. The name is derived from the Makonde word meaning "that which bends up" in reference to the stooped posture developed as a result of the arthritic symptoms of the disease. The disease was first described in 1952, following an outbreak on the Makonde Plateau, along the border between Tanganyika and Mozambique. Symptoms of this infection include abrupt onset of fever, chills, headache, and severe joint pain with or without swelling (usually the smaller joints), low back pain, and rash. The symptoms are most often clinically indistinguishable form those observed in dengue fever. Indeed, simultaneous isolation of both dengue and chikungunya from the sera of the same patients has been reported earlier indicating the presence of dual infections.⁹ Therefore it is very important to clinically distinguish dengue from chikungunya virus infection. Unlike dengue, hemorrhagic manifestations are relatively rare and as a rule shock is not observed in chikungunya virus infection. Most often chikungunya is a self limiting febrile illness. However, neurological complications such as meningoencephalitis have been reported in a small proportion of patients during the first Indian outbreak as well as the recent French Reunion islands outbreaks.¹⁰⁻¹¹ Mother to child transmission of chikungunya virus was a new observation recorded during the recent French Reunion islands outbreak.11

The precise reasons for the re-emergence of chikungunya in the Indian subcontinent as well as the other small countries in the southern Indian Ocean are an enigma. Although, it is well recognized that re-emergence of viral infections are due to a variety of social, environmental, behavioural and biological changes, which of these contributed to the re-emergence of chikungunya virus would be interesting to unravel. Genetic analysis of chiungunya viruses have revealed that two distinct lineages were delineated,¹² one containing all isolates from western Africa and the second comprising all southern and East African strains, as well as isolates from Asia. Phylogenetic trees corroborated historical evidence that the virus originated in Africa and subsequently was introduced into Asia.13 Such studies need to be conducted on virus isolates obtained during the current outbreak in order to understand if any mutation has occurred in the virus that has facilitated the large scale spread of this virus in the region. Alternatively, one could take the simplistic view that the lack of herd immunity within the country probably lead to its rapid spread across several states. A serosurvey conducted at Calcutta a decade ago did reveal that only 4.37% of the sera tested were positive for chikungunya antibodies with the highest seropositivity rates observed in the age group of 51-55 years and no chikungunya antibodies detected in the young and young adults. These findings probably suggest that there is indeed lack of herd immunity to chikungunya virus. Yet another challenge faced during this large outbreak in the country has been the lack of rapid diagnostic facilities. Although, the National Institute of Virology at Pune, has been of great help in determining the etiology of the outbreak relying on one institute in the country to render diagnostic help for case

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management would be foolhardy. It would be therefore desirable to ensure that several virology laboratories in the country are enrolled and networked to deliver rapid diagnosis in large outbreaks such as this as well other emerging viral infections like Chandipura and Avian influenza.

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