Chikungunya Fever

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Abstract
Chikungunya fever is a viral disease resembling dengue, transmitted to humans by the bite of an infected Aedes aegypti mosquito. It is endemic in East Africa and parts of Asia. Its recent epidemic has badly hit India - especially southern and central India - and like malaria and dengue, this infection has almost become endemic. Symptoms of sudden onset of fever, chills, headache, nausea, vomiting, joint pain with or without swelling, low back pain, and rash are very similar to those of dengue. But, unlike dengue, there is no haemorrhagic or shock syndrome. Chikungunya is a self-limiting illness that requires no specific treatment.

Introduction
Chikungunya is a relatively rare and benign form of viral fever caused by an alphavirus that is spread by mosquito bites from the infected Aedes aegypti mosquito. The name chikungunya, is given by Lumsden’s initial 1955 report, which is derived from the Makonde word ‘kungunyala’, meaning to dry-up or become contorted. Subsequently, Marion Robinson1 who first described the disease following an outbreak in 1952 on the Makonde plateau, between Tanganyika and Mozambique, glossed the Makonde term more specifically as “that which bends up.” This refers to the stooped posture adopted by the patient as a result of the arthritis symptoms that the patient develops.

In India it was first reported in 1963 at Calcutta2. Its recent epidemic which started in December 20053, involving southern and central India has grabbed much attention. Chikungunya is not considered to be fatal. However, in 2005 - 2006, 200 deaths have been associated with chikungunya of Reunion Island and widespread outbreak in southern India.

Clinically, it is characterised by abrupt onset of fever, chills, headache, joint pain, and swelling especially involving small joints. Various types of rashes develop usually after the subsidence of fever and in the convalescent phase.

Chikungunya is closely related to O’nyong-nyong virus4, and clinical features are very similar to those of dengue fever. However, unlike dengue, there is no haemorrhagic or shock syndrome form.

Aetiology
Causative organism – Chikungunya virus
Alternate name – Buggy Creek virus
ICTV acronym – CHIKV

Virus classification
Group: Group IV [(+) SS RNA]
Family: Togaviridae
Genus: Alphavirus
Species: Chikungunya virus

Mode of transmission
Chikungunya is spread by the bite of an infected Aedes mosquito, primarily Aedes aegypti.

Recently, research by the Pasteur Institute in Paris has found a mutation that enables it to be transmitted by Aedes albopictus (Tiger mosquito), which appears to be the cause of the recent epidemic in Asia.

Few cases of mother to foetus infection have been reported from Asia, which occurs between 3 and 4 months of pregnancy.

Reservoir
Humans are the major source of reservoir of chikungunya virus for mosquitoes.

Some non human primates like monkeys are the reservoir in Africa, in which it is transmitted by Aedes fureifer and africans.

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Epidemiology and recent outbreaks

Chikungunya was first described in Africa (Tanzania) in 1952. Then an outbreak was seen in Port Klang in Malaysia in 1999 affecting 27 people. It is endemic in parts of Africa (Transvaal, Uganda, Congo, Nigeria, Ghana, Zimbabwe, Senegal, Burkina Faso, and Cameroon), Southeast Asia (Philippines, Malaysia, Cambodia) and the Indian subcontinent (Pakistan and southern India).

In February 2005, an outbreak was recorded on the French island of Reunion in the Indian Ocean where 258,000 residents had been hit by the virus and 219 official deaths have been associated. In Mauritius, an outbreak has been recorded in 2005.

In 2006, there was a big outbreak in Andhra Pradesh in India where the initial cases were reported at Hyderabad and Anantpur districts in December 2005 and is continuing unabated. There have been reports of large scale outbreaks of chikungunya in Gulbarga, Tumkur, Bidar, Raichur, Bellary, Kolar, and Bijapur districts in Karnataka state since December 2005.

A separate outbreak of chikungunya fever was reported in Maharashtara and Orissa in March 2006. In May 2006, in Bangalore, the state capital of Karnataka, there appeared to be an outbreak of chikungunya with arthralgia/arthritis and rashes. On 23/06/2006, fresh cases of this disease were reported from Chennai, Tamil Nadu. On 24th August 2006, The Hindu newspaper reported that the Indian states of Tamil Nadu, Karnataka, Andhra Pradesh, Maharashtra, Madhya Pradesh, Gujarat, and Kerala had reported 1.1 million cases.

Recently, in August-September 2006, many cases of fever, arthralgia/arthritis and rashes have been reported from Ajmer, Bhilwara district of central Rajasthan, where 3 cases were found to be positive for chikungunya in Hurda village, district Bhilwara.

Re-emergence of chikungunya virus

Chikungunya virus is no stranger to the Indian subcontinent. Since its first isolation in Calcutta in 1963, there have been several reports of chikungunya virus infection in different parts of India. The last outbreak of chikungunya virus infection occurred in India in 1971.

A study conducted at the National Institute of Virology (NIV), Pune, India, has confirmed CHIKV as the causative agent for large outbreaks of fever with arthralgia and arthritis in 3 Indian states. Thus, chikungunya fever has emerged in an outbreak form after 32 years.

A recent report of large scale outbreaks of chikungunya virus in southern India has confirmed the re-emergence of this virus.

The precise reasons for the re-emergence in the Indian subcontinent as well as in southern India, of this viral infection, are due to a variety of social, environmental, behavioural and biological changes.

Genetic analysis of chikungunya viruses have revealed that two distinct lineages were delineated. One containing all isolates from Africa, and the second comprising all African as well as Asian strains. Phylogenetic trees corroborated historical evidence that the virus originated in Africa and subsequently was introduced into Asia. The Indian viruses isolated from 1963 through 1973 belonged to the Asian genotype, whereas the current isolates from the 3 Indian states and the Yawat isolate belonged to the central/east African genotype. A simplistic view is that the lack of herd immunity within the country is probably responsible for the epidemic. A serosurvey conducted in Calcutta a decade ago revealed that only 4.3% of the sera tested were positive – these are of 51 to 55 years age group. No child or young adult was found to be positive.

Clinical features

Full-blown disease is most common among adults in whom the clinical picture may be dramatic.

The abrupt onset of clinical manifestations follows an incubation period of 2 to 3 days. Silent CHIKV infections do occur, but the number and incidence is not yet known. Fever is sudden onset, high grade (> 40° C, 104°F) with chills and rigors; fever is biphasic or saddle back (fever subsides in 2 to 3 days and then comes back after 1 day); the second phase of fever is usually associated with bradycardia. Fever is associated with constitutional symptoms such as headache,
photophobia, conjunctivitis, anorexia, nausea, and abdominal pain.

- Arthralgia/arthritis in chikungunya has been quite crippling in recent outbreaks in southern India.

- Migratory polyarthritis mainly affects the small joints of hands, wrists, ankles, and feet. Rash may appear at the outset or several days after the illness i.e., in the convalescence phase.

- Dermatological manifestations observed in a recent outbreak of chikungunya fever in southern India include the following:
  1. Maculopapular rash.
  2. Blotchy, nasal erythema.
  3. Freckle-like pigmentation over centro-facial area.
  4. Flagellate pigmentation on face and extremities.
  5. Lichenoid eruption and hyperpigmentation in photo distributed areas.
  6. Multiple aphthous-like ulcers over the scrotum, crural areas, and axilla.
  7. Lymphoedema in acral distribution (bilateral/unilateral).
  8. Multiple ecchymotic spots (children).
  9. Vesiculobullous lesions (infants).
 10. Subungual haemorrhage.
 11. Phototrichia.

Fever typically last for two days and then abruptly comes down. However, joint pain, headache, insomnia, and various degrees of prostration last for a variable period, usually for about 5 to 7 days. Chikungunya is a self-limiting disease, and recovery is the rule, but little mortality has been reported in the recent outbreaks in southern India. In July 2006, a team analysed the virus RNA and determined the genetic changes that have occurred in various strains of the virus and identified the genetic sequence which led to the increased virulence of the recent strains.

The symptoms are most often clinically indistinguishable from those observed in dengue fever. Indeed, simultaneous isolation of both dengue and chikungunya from the sera of some patients has been reported earlier, indicating the presence of dual infections. Therefore, it is very important to clinically distinguish dengue from chikungunya virus infection. Unlike dengue, haemorrhagic manifestations are relatively rare, and as a rule, shock is not observed in chikungunya virus infection. Other important clinical conditions for differential diagnosis are West Nile fever, O’nyong-nyong fever.

Diagnosis

The tests available are:

- Detection of antigen and antibody in serum by ELISA test.
- IgM capture ELISA is necessary to distinguish the disease from dengue fever.

These tests are available at National Institute of Virology at Pune, Maharashtra.

Treatment

- There is no specific treatment for chikungunya. The illness is usually self-limiting and will resolve with time. Supportive care with rest is indicated during the acute joint symptoms. Ibuprofen, paracetamol relieve symptoms of fever and aching.
- Movement and mild exercise tend to improve stiffness and morning arthralgia, but heavy exercise may exacerbate rheumatic symptoms.
- In unresolved arthritis refractory to NSAIDs, chloroquine phosphate (250 mg/day for several weeks) has given good results.
- Vaccine for commercial purpose is not available and is undertrial.

Complications

Chikungunya is a self-limiting illness. The major causes for morbidity are severe dehydration, electrolyte imbalance, and hypoglycaemia.

Recovery is the rule, but 10–15% patients had chronic joint pain and stiffness.
Major complications, though rare, are:

1. Bleeding disorders (epistaxis, UGI bleed) as it causes thrombocytopenia, superadded by in judicious use of NSAIDs.

2. Neurological complications:\(^\text{15}\):
   a. Meningoencephalitis.
   b. Paresis of limbs.
   c. Slurring of speech.

3. Cardiovascular decompensation:\(^\text{16}\).

4. Pneumonia and respiratory failure:\(^\text{16}\).

5. Deaths – Few deaths have been reported in the recent epidemic in Reunion Island and in southern India:\(^\text{4}\).

Prophylaxis and prevention of chikungunya

As there is no vaccine available yet, the only way to prevent it is to eliminate mosquito breeding sites and to prevent mosquito bites.

Aedes mosquito vector for this disease breeds in artificial accumulations of water in and around human dwellings, such as water found in disused wares, broken bowls, flower pots, earthen pots. Therefore, these sites should be eliminated by responsible human behaviour and social education.

Aerosol spray of ultra low volume quantities of malathion or sumithion (230 ml/litre) has been found to be effective.

Mosquito net and repellents which contain 20 – 50% DEET (N, N-diethyl-meta-toluidide) should be used to prevent mosquito bites. As mosquito bites during daytime, mosquito repellants should be used during daytime also, and not only during the night.

Use of full sleeve clothing is effective in preventing mosquito bites.

Chikungunya fever with pregnancy

There have been cases of mother-to-foetus infection which have occurred between 3 and 4 months into pregnancy:\(^\text{17}\). Before and after that period in pregnancy, cases have not been recorded. IgG that is produced around day 15, passes through the placenta and confers immunity to the foetus. However, there is a 48 per cent risk of infection at birth if the virus is present in the mother’s blood:\(^\text{18}\). Such an infection in the foetus is rarely serious, and more than 90 per cent of the infected newborns recover quickly without sequelae.

Information to travellers

In 2006, CHIK fever cases also have been reported in travellers returning from known outbreak areas to Europe, Canada, the Caribbean (Martinique), and South America (French Guyana). During 2005 – 2006, 12 cases of CHIK fever were diagnosed serologically and virologically at CDC in travellers who arrived in the United States from areas known to be epidemic or endemic for CHIK fever:\(^\text{19}\). Currently, there is no restriction on travel to islands in the Indian Ocean. Travellers should be careful to take necessary precautions to prevent mosquito bites:\(^\text{20}\).

References


