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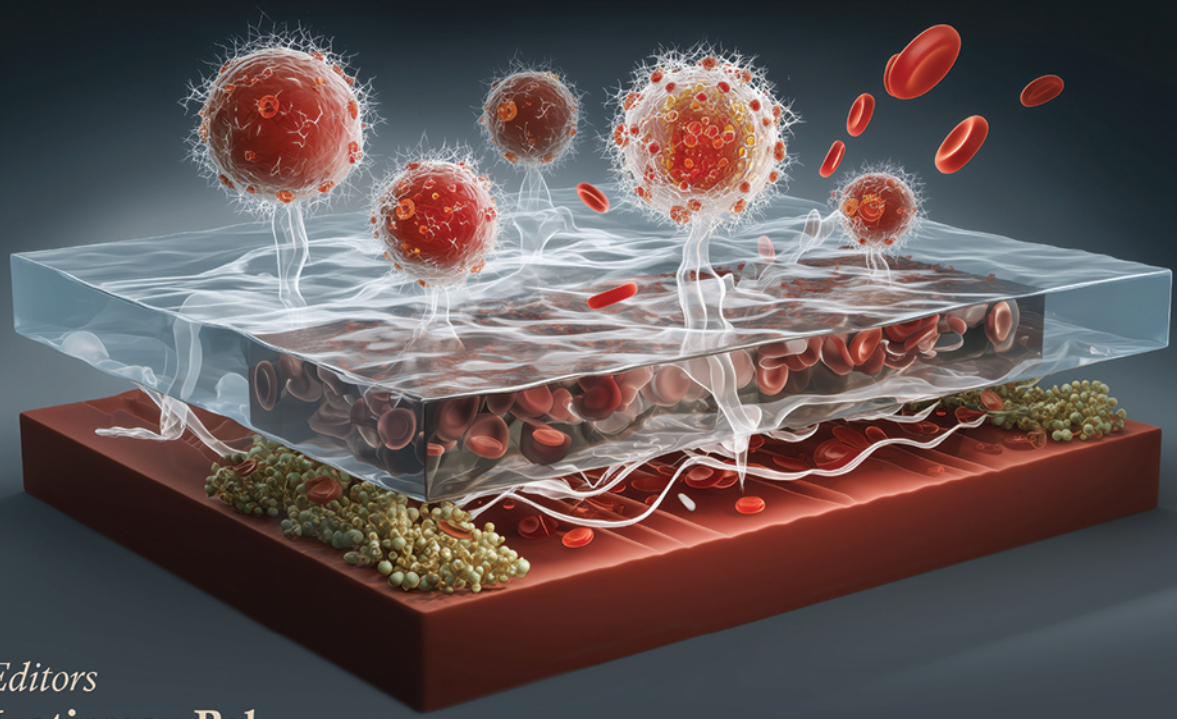
Monograph on Tropical Fever



Editors
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Foreword
Krishantha Jayasekera

2nd Edition

JAYPEE

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CHAPTER 9

Zika and Chikungunya

Anivita Agarwal, Nandini Chatterjee, Animesh Ray, Tanuka Mandal

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INTRODUCTION

Zika virus (ZIKV) is an arbovirus belonging to the genus *Flavivirus* and *Flaviviridae* family. This positive-sense single-stranded ribonucleic acid (RNA) virus is transmitted by the bite of infected mosquitoes. The disease usually presents as an acute febrile illness with rash, arthralgia, and conjunctivitis. ZIKV infection has been associated with significant neurological complications as well as congenital microcephaly.

Chikungunya is also an arthropod-borne single-stranded RNA virus, presenting as an Alphavirus febrile illness with rash. The virus belongs to the family *Togaviridae*, genus alphavirus, and causes characteristic debilitating joint pains. Though usually a self-limiting illness, a substantial proportion of the population develops chronic disease in the form of characteristic, incapacitating polyarthralgia and inflammatory arthritis lasting for months to years. Millions of cases across the globe and the association with severe disease with multiorgan failure have been lately been a major public health concern lately.

Overlapping clinical features of acute onset fever with rash and joint pains with other arboviral diseases pose a diagnostic challenge for both these viral fevers in tropical countries **(Table 1)**. Currently, there are no therapeutic drugs available, and vaccines are under trial.

TABLE 1: Clinical features, laboratory parameters, and diagnostic tests to differentiate dengue fever, chikungunya fever, with Zika fever.

Differentiating parameter	*Zika*	*Chikungunya*	*Dengue*
Clinical features:			
• Fever	++	+++	+++
• Rash	+++	++	+
• Arthralgias	++	+++	+
• Conjunctivitis	++	+	–
• Hemorrhage	–	–	++
• Shock	–	–	+
Laboratory parameters:			
• Leukopenia	–	+	+
• Thrombocytopenia	±	±	+
Diagnostic tests:	RT-PCR if <2 weeks, IgM if >2 weeks	PCR if <5 days, IgM if >5 days	NS1 antigen if <3–5 days, dengue IgM if >5 days

(IgM: immunoglobulin M; RT-PCR: reverse transcription polymerase chain reaction)

ZIKA VIRUS

Epidemiology

Zika virus was first isolated from nonprimate hosts in 1947, then from the *Aedes africanus* mosquito in 1947, and later described in a human case from Nigeria in 1954. The virus was further isolated periodically from various serosurveys across Asia and Africa. However, major outbreaks in the Western Pacific Islands of Yep (2007), French Polynesia in the South Pacific (2013–2014), and Brazil (2015–2016) brought focus due to the large numbers of affected population. Based on genomic sequencing of the NS-5 encoding gene and phylogenetic studies, ZIKV has been classified into three lineages: East African, West African, and Asian, having varied clinical presentations. Indian outbreaks in 2017–2018, ascribed to the Asian lineage and the recent outbreak in Kerala in 2021, were not reported to have any Zika-associated microcephaly. **Figure 1** illustrates the geographical distribution of outbreaks of ZIKV in India. With growing globalization and travel, over 86 countries and territories have reported cases of ZIKV as per the World Health Organization (WHO).

Pathogenesis

The primary mode of transmission of ZIKV is by *Aedes* (*Aedes aegypti, Aedes albopictus*, etc.) mosquito bite, which is also shared by the Dengue and Chikungunya viruses. Recently described, alternative modes of transmission include spread via the transplacental route, sexual intercourse, blood transfusion, organ transplant, and breast milk. ZIKV RNA has also been found to be shed in urine, semen, saliva, blood, female genital tracts, breast milk, amniotic fluid, and cerebrospinal fluid (CSF), etc., for prolonged periods of time.

The virus is transmitted via the bite of an infected mosquito harboring the virus in its salivary glands after a blood meal from another infected vertebrate (extrinsic incubation period of 8–12 days). Humans are accidental hosts, whereas animals are reservoirs. The exact pathogenesis remains largely unknown, but the virus is believed to replicate in dendrites at the site of a mosquito bite. After an (intrinsic) incubation

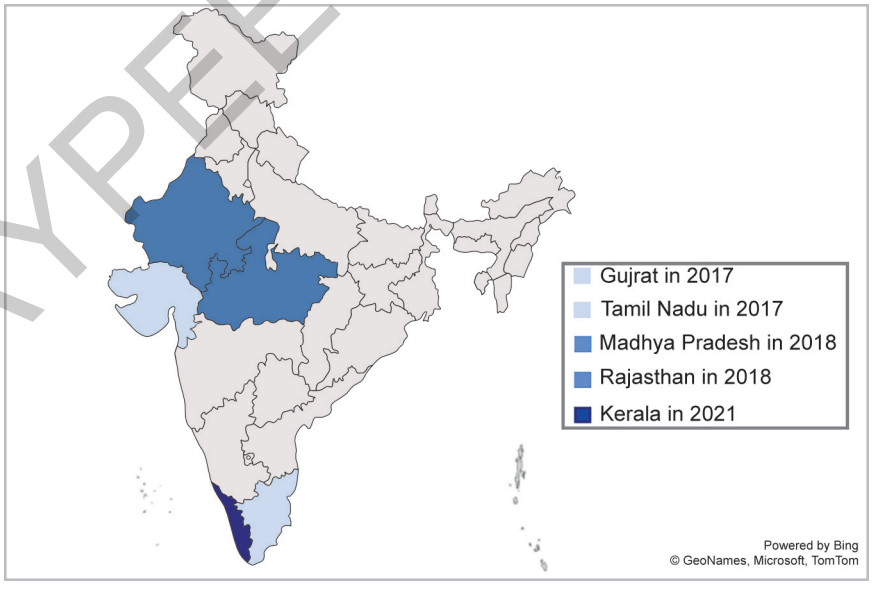

Fig. 1: Geographical distribution of outbreaks of Zika virus in India.

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period ranging from 3 to 12 days, it spreads to the regional lymph nodes and distant organs such as the nervous system along the hematogenous route. In pregnant females, it may infect the fetus through transplacental transmission. ZIKV may persist in whole blood, immune-sanctuary sites, semen, and urine for weeks to months.[1]

Clinical Features and Complications

Incubation period from the bite of a mosquito to symptom onset may vary between 3 and 12 days. Over 75–80% infected are likely to develop asymptomatic disease. Historically, symptoms were believed to be a mild, self-limiting disease, with a low case fatality rate. However, in the recent past, many cases of severe disease with multiorgan failure requiring intensive care and mortality have also been seen. Predominant feature is fever (72%), which is typically low grade, usually resolving within 1–2 weeks. Maculopapular pruritic rash spreading proximally to extremities, including face, palms, and soles, which spontaneously resolves in 1–4 days, is a characteristic finding. Associated symptoms such as arthralgias and myalgias (65%) and nonpurulent conjunctivitis (63%) are also commonly noted.[2] Other reported symptoms include headache, retroorbital pain, and rarely of pericarditis, myocarditis, facial puffiness, and uveitis.

The Brazil ZIKV outbreak (2015) was notable for the dreaded complication of the disease in pregnant women, resulting in infants born with microcephaly was reported in 20/10,000 live births compared to a background rate of 0.5/10,000 live births. Pregnant women are susceptible to ZIKV infection in all trimesters, with maternal-fetal transmission. The severe form commonly arises due to infection in the first or second trimester. Congenital Zika syndrome could be present as fetal losses, microcephaly, facial dysmorphisms, sensorineural hearing loss, and ocular abnormalities. Studies have demonstrated the incidence of microcephaly as <7%.[3]

Severe neurological manifestations, first noticed in the French Polynesia outbreak (2013–2014), have also been described, such as Guillain–Barré syndrome, meningitis, and meningoencephalitis.[4] Other neurological manifestations include transverse myelitis, encephalitis, neuropsychiatric symptoms, and cognitive symptoms.

Acute febrile illnesses such as dengue, chikungunya, malaria, leptospirosis, scrub typhus, and enteric fever must be considered as a differential diagnosis in India due to significant overlap of clinical features.[5] Zika fever should be suspected in patients with clinical manifestations as well as epidemiological exposure (travel to an area where ZIKV infection has been reported, or unprotected sexual contact with a person who meets these criteria).

Diagnosis

The Centers for Disease Control and Prevention (CDC), the National Health Service (NHS), and WHO have classified it as a level 2 pathogen requiring a biosafety level 2 laboratory for sample handling.

Reverse transcription polymerase chain reaction (RT-PCR) in serum, whole blood, or urine samples within weeks of symptom onset can be used to diagnose ZIKV. A positive RT-PCR confirms the diagnosis; a negative test, however, does not rule it out and should be followed by serology testing. Urine RT-PCR is found to have higher sensitivity.

Immunoglobulin M (IgM) capture enzyme-linked immunoassay (ELISA) serology/CSF analysis performed after 1–2 weeks of illness has good specificity. However, structural similarities between Dengue virus and ZIKV can lead to cross-reactivity of antibodies produced against flaviviruses; hence must be interpreted with caution. Testing for neutralizing antibodies via PRNT (plaque reduction neutralization test) can be performed to determine false positivity.

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Treatment and Prevention

Being essentially a self-limiting disease, management is primarily supportive. Symptomatic management with rest, intake of fluids, antipyretics, and analgesics are to be provided. ZIKV could lead to immune-mediated thrombocytopenia (ITP); hence, nonsteroidal anti-inflammatory drugs (NSAIDs) should be avoided to prevent the risk of bleeding. Currently, there is no approved drug or vaccine against ZIKV, although several are being investigated. Vector control and prevention from mosquito bites remains the sole preventive and most effective strategy against flaviviruses.

CHIKUNGUNYA VIRUS

Epidemiology

Chikungunya was first isolated in 1952 from an outbreak in Tanzania, in a patient having severe rheumatological manifestations. The disease was named in their Kimakonde language as chikungunya, meaning "to bend over," referring to the patients' posture assumed with disabling joint pains. Across the globe, various outbreaks have occurred in the tropics and subtropics, causing millions of human infections and serious public health concerns.

Reported in India first in 1963, the illness reemerged in 2005 (The Indian Ocean lineage) and subsequently resulted in periodic outbreaks all over the country, making the disease endemic. A notable mutation, E1-A226V, among many others, has been identified that has led to its enhanced fitness, survival adaptability (to *Aedes albopictus*), and severity, causing numerous Indian outbreaks.[6]

As dengue, Zika, and chikungunya viruses are all transmitted via the same vector, it is often difficult to differentiate these epidemiologically. Dengue and Chikungunya coinfections have also been frequently reported. **Table 1** delineates the differentiating features between dengue, Zika, and chikungunya.

Pathogenesis

Being an arbovirus, CHIKV (Chikungunya virus) is primarily transmitted by the bite of its vectors, *Aedes aegypti* and *Aedes albopictus* mosquitoes. Mosquito replicates the CHIKV in its midgut and reaches the salivary glands during the extrinsic incubation period (of 10 days). After the bite of the infected mosquito, it replicates within dermal fibroblasts at the site of inoculation and then spreads via the bloodstream to various organs. Animal models suggest that the virus directly infects the synovium and replicates in the joints, causing the production of proinflammatory cytokines and chemokines.

Chikungunya virus is also transmitted to the eggs from adult mosquitoes through vertical transmission. Mother-to-child transmission, although very rare, has also been reported in pregnant women who developed chikungunya fever in their last trimester of gestation, potentially leading to poor pregnancy outcomes.[6] Occasionally, it may also be transmitted via blood products.

The transmission is via two types of cycles: (1) Sylvatic cycle, where there is enzootic transmission between nonprimates (which acts as both host and reservoir) and mosquitoes, and (2) urban cycle, where *Aedes* mosquitoes spread the disease to human hosts.[7]

Clinical Features and Complications

After an incubation period of 2–6 days from the bite of a mosquito, clinical presentations may range from asymptomatic to severe disease.

Chikungunya virus causes a fever with arthritis syndrome, with symptoms usually occurring in 72–97% of the infected patients.

Acute Phase

It is characterized by high fever and severe joint pain that can compromise daily life activities. The rashes are erythematous macular or maculopapular rashes appearing in the first 2–3 days of the illness and subsiding within

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7–10 days. It can be patchy or diffuse on the face, trunk, and limbs. It may be pruritic at times. Fever, myalgia, and Rashes usually resolve or decrease in intensity in 1 or 2 weeks, although the acute phase may persist up to 21 days.

Postacute Phase

Postacute phase—3 weeks to 90 days. Presentation includes polyarthralgia, polyarthritis, exacerbation of comorbidities, chronic fatigue, and worsening of preexisting degenerative or traumatic arthritis.

Chronic Phase

- *Chronic phase*: arthralgia for >3 months. In addition to joint pain, exacerbation of comorbidities, tenosynovitis, tendinitis, and neuritis have been reported.
- Chronic chikungunya arthritis may mimic rheumatoid arthritis clinically to the extent that it may satisfy the 2010 American College of Rheumatology (ACR) diagnostic criteria for rheumatoid arthritis and lead to confusion.
- Chronic chikungunya arthritis commonly affects middle-aged women. The metacarpophalangeal, proximal interphalangeal, and wrist joints, as well as the knees and ankles, are involved in a symmetric, polyarticular pattern. Also, tenosynovitis, bursitis, and tendonitis may occur. Other features are fatigue, insomnia, myalgias, morning stiffness, memory or concentration problems, and asthenia or depression.

Patient Subsets

- *Elderly patients*: evidence of arthralgia persisting for >15 months and high mortality.
- Patients with comorbidities such as hypertension and diabetes have a higher risk of severe disease, hospitalization, and mortality.
- *Pregnant female*: Chikungunya fever does not confer an increased risk for obstetric hemorrhage, preterm birth, or low birth weight.
- Children born with low virus levels may manifest fever, pain, and suckling difficulties within a week.
- *Congenital chikungunya (mother-to-child chikungunya virus infection)*: 50% chances if the mother is infected with a high viral load during the early stages of labor. Complications such as chikungunya virus-associated central nervous system disease (encephalopathy) and multiple organ dysfunction (MOD) may occur in about half of these newborns.

Differential Diagnosis

- The differential diagnosis varies according to geographical distribution, travel history, and exposures.
- Dengue, Zika, and chikungunya viruses are transmitted by the same vectors, occur in similar environs, and have comparable clinical features.
- Occasional coinfections in the same patient have been found.
- Chikungunya virus infection is more likely to cause high fever, severe arthralgia, arthritis, rash, and lymphopenia, while dengue virus infection is more likely to cause neutropenia, thrombocytopenia, hemorrhage, or shock.

The clinical presentation of ZIKV is mild, though there may be congenital infections.

Other conditions to be considered in differential diagnosis are:

Infections

leptospirosis, malaria, rickettsia, group A streptococcus, rubella, measles, parvovirus, enteroviruses, adenovirus, other alphavirus infections (Mayaro, Ross River, Barmah Forest, o'nyong-nyong, and Sindbis viruses)

Post-infectious Arthritis

Rheumatologic conditions like RA, SLE, Sjogren Syndrome

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Complications, including neurological manifestations such as encephalitis, meningitis, Guillain–Barré syndrome; cardiovascular features such as myocarditis and tachyarrhythmias, have also been reported in the elderly and patients with underlying medical conditions. Deaths have been reported during chikungunya outbreaks in India and other countries.

Diagnosis

Based on clinical manifestations and epidemiological clues, the diagnosis of chikungunya must be suspected. Laboratory diagnosis can be based on viral isolation, RT-PCR, or serological testing [immunoglobulin G (IgG) and IgM antibodies] using ELISA or rapid immunochromatographic assays. RT-PCR, not widely available, has very high sensitivity and specificity for the detection of CHIKV and can be performed on blood or CSF samples within the first week of illness. ELISA/immunochromatographic assays for IgM antibodies are used for detection after five days from illness. These persist for weeks to three months and must be correlated with IgG antibodies, clinical features, and epidemiological setting.

In endemic areas, many acute febrile illnesses may be misdiagnosed on the basis of serological tests due to cross-reactivity of IgM and coinfection with other arboviruses. Hence, physicians must keep a high index of suspicion for alternative etiologies.

Treatment and Prevention

There is no specific antiviral therapy or effective vaccine available for chikungunya fever. Management is essentially supportive, including rest, fluids, antipyretics, and anti-inflammatory or analgesics. NSAIDs must be carefully administered after ruling out dengue fever in the acute phase due to the risk of bleeding manifestations. Polyarthritis usually resembles inflammatory arthritis on imaging and typically responds to conventional anti-inflammatory drugs. However, for persistent symptoms or nonresponsiveness after even two weeks of NSAID therapy, with elevation of inflammatory markers, corticosteroid administration may be considered.

For patients with persistent joint pain, the use of nonsteroidal anti-inflammatory drugs, and physical therapy are common.

Recommendations for treatment with methotrexate (MTX), sulfasalazine (SSZ), leflunomide, hydroxychloroquine (HCQ), NSAIDs, or immunobiologicals have been put forward.

Several publications advocate the use of HCQ as first-line therapy for CHIKF arthritis, although there is limited evidence of efficacy. The dose being (6 mg/kg/day), with evaluation every 6 weeks, followed by the addition of SSZ (1,000 mg/day). If still not controlled after 36 weeks, to initiate MTX (at an initial oral dose of 10 mg/week).

Prevention

Prevention of chikungunya virus disease:

- Community-level mosquito control programs.
- Personal protective measures to decrease exposure to infected mosquitoes.
- Personal protective measures include the use of insect repellents, mosquito nets, and wearing long-sleeved shirts and long pants.
- Travelers should choose a hotel or lodging with air conditioning or screens on windows and doors.

CHIKUNGUNYA VACCINE

The Advisory Committee on Immunization Practices (ACIP) recommendations for use of chikungunya vaccine among travelers:

- Persons aged ≥18 years traveling to a place where there is a chikungunya outbreak.
- Persons traveling to a place without an outbreak but with evidence of chikungunya virus transmission among humans within the last 5 years.

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- Persons aged >65 years, particularly those with comorbidities, who are likely to be exposed to mosquitoes.
- Persons staying for a cumulative period of 6 months or more.

Vector control in the form of insecticide sprays, biological control, and personal protection from bites of mosquitoes are the most useful steps that are essential for the prevention of arboviral diseases such as chikungunya.

CONCLUSION

Zika and Chikungunya infections are caused by arboviruses and have the same vectors. Their clinical presentations are also somewhat similar. But clinical suspicion based on geographical distribution, travel history and milder presentation in Zika are helpful for management.

TAKE HOME MESSAGE

- ZIKV leads to a relatively uncommon arboviral disease in India, transmitted via the bite of an Aedes mosquito.
- The most dreaded complication due to ZIKV infection is microcephaly, which occurs due to the disease in pregnant females typically in their first or second trimesters.
- Chikungunya disease, caused by the same vector, leads to an acute febrile illness with severe joint pain.
- In some patients, Chikungunya can lead to relapsing or intermittent disabling joint pain, which requires treatment in line with inflammatory arthritis.
- The management of both Zika and Chikungunya is essentially supportive, while vector control and personal protection form the backbone of the preventive strategy.

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Monograph on
Tropical Fever

Salient Features

- Comprehensive analysis of infections prevalent in tropical regions
- Syndromic diagnostic strategies for tropical febrile illnesses
- Recent updates on managing drug-resistant infections
- Focus on neglected tropical diseases and the resurgence of certain infections
- New reader friendly format has been added
- With take away messages at the end
- Latest development in management have been incorporated

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