Research Article

Chandipura Virus: an Integrated View of Virology, Disease and Public Health

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Abstract

Chandipura virus (CHPV) is a newly emerging arbovirus in India that is mainly spread by sandflies and is connected to episodes of acute encephalitis in kids. Typically, grown-ups do not show clinical signs. CHPV was first identified in Maharashtra in 1965 and has since been associated with AES outbreaks in various states like Maharashtra, Gujarat, Telangana, and Madhya Pradesh. The virus primarily impacts children below 15 years old, especially in rural regions with substandard living conditions. It is most prevalent during the monsoon season when sandflies are most active. The illness progresses rapidly with fever and encephalitis, often resulting in a high mortality rate. Resemblances to other arboviral infections make managing outbreaks challenging due to diagnostic complexities. Present-day public health measures focus on controlling vectors, maintaining cleanliness in the environment, and raising community awareness, but the absence of specific antiviral therapies and vaccines poses a significant obstacle. This evaluation underscores the necessity for enhanced surveillance, diagnostic methods, and vaccine creation to reduce the potential public health consequences of CHPV in India. A holistic One Health strategy, integrating human, animal, and environmental health, is vital for the efficient handling and prevention of CHPV outbreaks.[1]

Chandipura virus (CHPV) is an emerging neurotropic arbovirus belonging to the *Vesiculovirus* genus of the *Rhabdoviridae* family and is an important cause of acute encephalitis syndrome (AES) in India. The infection predominantly affects children, particularly those below 15 years of age, and may progress rapidly from an acute febrile illness to severe neurological disease with a high case-fatality rate. [1,3] CHPV is mainly spread by diseased phlebotomine sandflies, and its frequent presence in various Indian states underscores its ongoing significance for public health.[4] Typically, the symptoms of the disease consist of a sudden onset of high body temperature, head pain, throwing up, convulsions, changes in awareness, and additional signs related to the nervous system. In serious cases, the infection can quickly advance to inflammation of the brain, unconsciousness, and mortality, especially among young individuals.[6] Ever since its initial discovery in 1965, CHPV has been linked to repeated episodes in various Indian regions such as Maharashtra, Andhra Pradesh, Gujarat, Odisha, and Rajasthan.[9,13] the significant increase documented in Gujarat in 2024 and resurgence of CHPV activity in Gujarat and Rajasthan in 2026 highlight the ongoing danger presented by this virus. [14] at present, there is no specific antiviral therapy or licensed vaccine for CHPV, making early diagnosis, supportive management, vector control, surveillance, and public-health preparedness essential for outbreak prevention and control.[4]

In general, CHPV continues to be a significant new viral danger in India, particularly affecting children. Enhancing laboratory testing, tracking of disease spread, genetic surveillance, controlling sandfly vectors, raising community knowledge, and studying efficient antiviral treatments and preventive measures are crucial to decrease illness and death related to CHPV and to enhance readiness for upcoming outbreaks.

Keywords: Chandipura virus; Acute encephalitis syndrome; Arbovirus; Vesiculovirus; Rhabdoviridae; Phlebotomine sandflies; Children; Vector-borne disease; India; Surveillance; Vector control; One Health.

INTRODUCTION

Chandipura virus (CHPV) is a newly identified virus transmitted by arthropods from the Vesiculovirus group of the Rhabdoviridae family. In India, Chandipura virus predominantly impacts individuals below the age of 15. Data from epidemiological studies and outbreaks indicate that adults are seldom affected or show symptoms, even though the

virus was first discovered in 1965 in two adult patients with a fever in Chandipura village, Maharashtra, India.[2] There is currently a lack of significant international data on adult cases comparable to that of India. [3] Originally believed to cause mild febrile illness, subsequent outbreaks revealed that CHPV can lead to severe acute encephalitis syndrome (AES), particularly in children under 15. The illness is spreading quickly with a high fatality rate and continues to be a significant public health issue in regions where it is prevalent.

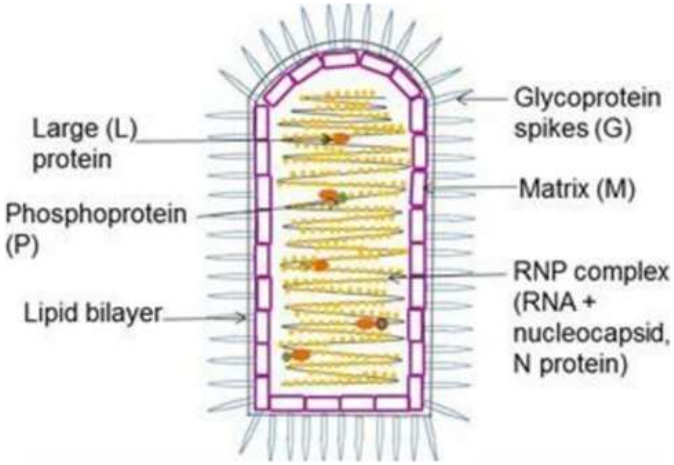

Fig.1 Structure of CHPV Deshwali ZH, Sharma V. A review on Chandipura virus: outbreaks and emerging infectious disease for children in India. Asian J Res Infect Dis. 2025;16(1):1-12. doi:10.9734/AJRID/2025/v16i1413.[4]

Chandipura virus (CHPV) has various crucial structural elements that are necessary for infecting host cells and replicating within them. The virus is enclosed by a lipid bilayer membrane, originating from the host cell membrane during the process of viral budding, and this membrane includes glycoprotein (G) spikes. These spikes are crucial for maintaining structure, aiding in binding to host cell receptors, and acting as significant antigenic markers for immune response and the creation of vaccines. Underneath the membrane is the matrix (M) protein, which is essential for assembling the virus.

The central part of the virus is made up of a ribonucleoprotein (RNP) complex created by the negative-sense, single-stranded RNA genetic material (~11 kb) surrounded by the nucleocapsid (N) protein. There are five structural proteins produced by the genetic material: nucleocapsid (N), phosphoprotein (P), matrix (M), glycoprotein (G), and large polymerase (L). CHPV possesses an unsegmented, negative-sense RNA genome of around 11 kb that carries instructions for five primary proteins: nucleoprotein (N), phosphoprotein (P), matrix protein (M), glycoprotein (G), and large polymerase (L). These proteins are crucial for various stages of the virus's life cycle such as replication, transcription, assembly, entry, and dissemination. Together, these structural parts facilitate the process of viral entry, multiplication, formation, and transmission in host cells, which highlights their significance as key areas for the development of antiviral medications and vaccines.

CHPV is transmitted by sandflies, particularly Phlebotomus spp., although mosquitoes and ticks could also serve as carriers. Various Indian states, including Andhra Pradesh, Gujarat, Maharashtra, and Odisha, have experienced numerous virus outbreaks since the early 2000s, indicating a broader spread. While there is a possibility of the virus spreading in an epidemic manner, there is currently no designated antiviral medication or approved vaccine. Therefore, timely detection, effective vector management, continuous monitoring, and raising public health awareness remain essential to avoid and manage CHPV infections. [4]

CHPV GENOME STRUCTRE

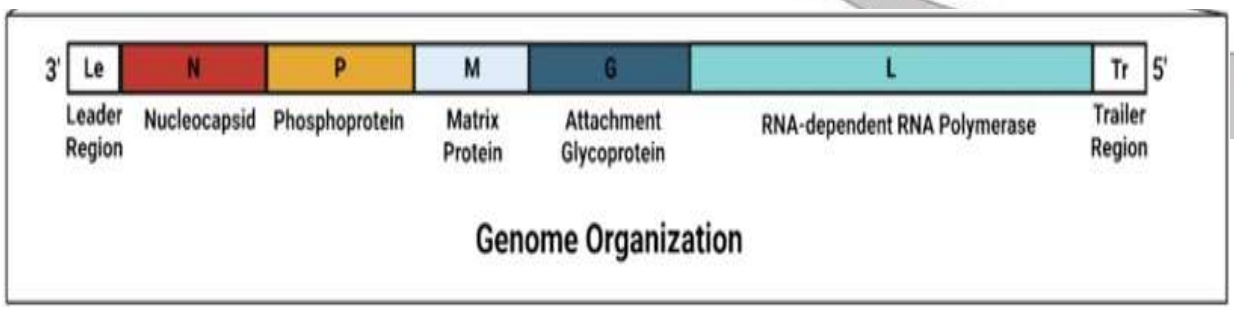

Figure: 2 CHPV Genomic Features https://www.antibodysystem.com/archive/634.html[5]

1. Nucleoprotein (N): Packages viral RNA to create the nucleocapsid, which is the fundamental structural element of the viral ribonucleoprotein complex.
2. Phosphoprotein (P): Serves as a specific escort for N, preventing its random clustering during duplication to guarantee correct nucleocapsid formation. It also controls viral transcription and duplication.
3. Matrix Protein (M): Positioned below the envelope, it upholds the structural stability of the virion.
4.Glycoprotein (G): The single spike on the surface of the virus, facilitating recognition of receptors and merging the viral envelope with the membrane of the host cell. Computational simulations also indicate that G proteins of Indian and West African variations differ at the interface responsible for binding to receptors, which might clarify why the Indian strain is more harmful to humans.
5.Polymerase (L): Responsible for catalysing the replication and transcription of viral RNA. [5]

CLINICAL FEATURES AND SYMPTOMS

Chandipura virus (CHPV) infection typically has an incubation period of 2 to 14 days following a bite from a sandfly or mosquito carrying the virus. Symptoms often start with a sudden high fever, intense headache, shock in severe instances, body pain, and overall fatigue.
As the infection advances, neurological signs become more noticeable. Individuals might experience seizures, disorientation, sleepiness, restlessness, and, in extreme situations, unconsciousness or a coma. Indications of meningeal inflammation, like stiffness in the neck and sensitivity to light, could also be detected.
Certain individuals may encounter stomach-related issues like queasiness and throwing up at the initial phase of the sickness. While it is not as frequent, a skin eruption and muscle fatigue might also manifest.
In serious cases of infection, CHPV can lead to sudden inflammation of the brain, causing quick neurological decline. Individuals who survive a severe illness might experience lasting neurological issues such as motor problems, paralysis, or cognitive impairment. Prompt identification and proper care are crucial due to the swift advancement and high likelihood of death associated with this condition.[6]

TRANSMISSION

Vector-borne: primarily through the bite of diseased Phlebotomus sandflies.
Reservoirs: Mice, cows, swine, and canines. Brief viremia can be sustained, aiding the virus's persistence in the wild.
Human exposure: Located in rural regions close to animal shelters and inadequately ventilated areas with high organic content that are conducive to the reproduction of sandflies. [7]

PATHOPHYSIOLOGY OF CHANDIPURA VIRUS

The Chandipura virus (CHPV) is primarily transmitted to humans by the bite of infected Phlebotomus sandflies. Once the virus enters the body, it replicates at the bite site and then spreads through the blood causing initial viraemia. The virus disseminates throughout the body and eventually breaches the blood-brain barrier (BBB) to infiltrate the central nervous system. CHPV has a strong affinity for the nervous system, targeting neurons and triggering brain inflammation. The body's immune response to the infection involves the release of inflammatory cytokines and activation of microglial cells. This robust inflammatory reaction results in brain swelling, neuronal harm, and inflammation around blood vessels. The outcome is high body temperature, vomiting, seizures, changes in

consciousness, and sudden encephalitis. This progression can quickly lead to unconsciousness and death, particularly in young kids.[8]

HISTORICAL EVOLUTION AND RECENT OUTBREAKS OF CHANDIPURA VIRUS IN INDIA/GLOBAL

The Chandipura virus (CHPV) was initially discovered in 1965 from individuals in Chandipura village close to Nagpur, Maharashtra. Later on, between 1967 and 1969, it was found in phlebotomine sandflies, indicating their involvement in transmitting the virus in 1980.[9] CHPV was identified in individuals with sudden inflammation of the brain in Madhya Pradesh, confirming its link to brain disorders.[10] Following this, significant surges were documented in various regions of India. In the year 2003, Andhra Pradesh had a notable encephalitis outbreak, with a major outbreak occurring in Gujarat in 2004. Additional outbreaks or groups of cases were recorded in Maharashtra and Telangana between 2005 and 2006. In 2009, Odisha, in 2012, Nagpur, and in 2018, Bihar experienced the repeated and varied geographical spread of CHPV in India. [11] In 2024, there was a significant increase, mainly in Gujarat, with 245 cases of acute encephalitis syndrome (AES) and 82 deaths reported across the country by August 15, 2024. Out of these, 64 cases were confirmed to be caused by CHPV through laboratory tests, with 61 originating from Gujarat and three from Rajasthan. This outbreak was recognized as the most extensive CHPV-related incident in India in about twenty years. [12,13] In 2026, there have been new reports of CHPV activity in Gujarat and Rajasthan. By the beginning of August 2026, Gujarat had documented 184 suspected cases, 35 confirmed CHPV cases, and 22 fatalities. [14] In general, the recurrent occurrences spanning from 1965 to 2026 highlight the ongoing significance of CHPV in India's public health, especially in children. They underscore the necessity for enhanced AES monitoring, quick detection, genetic tracking, and efficient control of sandfly vectors. Chandipura virus (CHPV) has not caused any major global outbreaks.[15]

CONCLUSION

This review examines the present knowledge of the epidemiology, virology, transmission, pathogenesis, clinical symptoms, diagnosis, prevention, and recent progress in Chandipura virus studies. It underscores the importance of enhanced monitoring and the creation of successful treatments and preventive measures. [16]

Chandipura virus (CHPV) is now a significant factor in causing acute encephalitis syndrome (AES) in India, especially affecting children. Despite its discovery in Maharashtra in 1965, its frequent appearance in various Indian regions over the years emphasizes its ongoing significance in epidemiology and public health. The virus mostly impacts children, swiftly advancing from fever to severe neurological symptoms, and is linked to high mortality rates in severe cases, underscoring the importance of early detection and prompt supportive care.[1,2,6] The structural and genetic features of CHPV, such as its single-stranded negative-sense RNA genome and the N, P, M, G, and L proteins, offer vital focal points for comprehending viral replication, disease development, antiviral investigations, and vaccine creation.[5,17]The repeated occurrences documented from 1965 to 2026, such as the significant increase in cases in Gujarat in 2024 and the resurgence in Gujarat and Rajasthan in 2026, highlight the importance of ongoing monitoring for AES, quick and trustworthy lab testing, genetic surveillance, and efficient control of sandfly vectors.[16,14] Currently, the lack of a particular antiviral remedy and approved vaccine poses a significant obstacle. Therefore, a combination of vector control, sanitation practices, community education, and enhanced public-health readiness are crucial elements for preventing and managing CHPV outbreaks.[4] In general, continuous monitoring and a unified One Health strategy involving human, animal, and environmental health are essential to enhance early identification, decrease spread, and lessen the consequences of future CHPV outbreaks in India.

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