



Review Article

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Chandipura virus: The 2024 resurgence and Gujarat outbreak

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ABSTRACT

Originally discovered in 1965 in Maharashtra, India, the Chandipura virus has become a serious hazard to public health, especially in rural areas. Acute encephalitis syndrome, which mostly affects children and has a high death rate, has been associated with Chandipura virus, an arbovirus largely spread by sandflies (*Phlebotomus* spp.). The virus was first thought to be a benign infection, but after many fatal outbreaks, most notably in Gujarat (2005 and 2024) and Andhra Pradesh (2003), it has come to light. The aggressiveness of the virus is highlighted by the quick development from fever to severe neurological symptoms, such as seizures and coma. Public health initiatives concentrate on vector management and supportive care because there is no particular antiviral medication or vaccine available. Recently, Gujarat has witnessed the biggest outbreak of Chandipura virus in 2024 and it may become a big health concern. This paper discusses about Chandipura virus and its recent cases. The virus's propagation, which is made worse by climate change, makes future outbreaks of the virus more likely. The epidemiology, transmission, and clinical consequences of Chandipura virus are highlighted in this review, along with the necessity of more research, monitoring, and public health readiness to lessen its effects.

KEYWORDS: Chandipura virus; Gujarat viral attack; Sandfly; *Phlebotomus*; Acute encephalitis syndrome

1. Introduction

The Chandipura virus (CHPV), not as well-known as other viral infections, is becoming a serious public health issue in several areas, most notably India[1]. Scientists at the Virus Research Centre in Pune, India, Dr. Bhatt and Dr. Rodrigues made the initial discovery of the virus in 1966. The virus was recovered during an investigation into a febrile sickness in the village of Chandipura, which is situated

in northern Maharashtra close to Nagpur. The scientists found a novel virus, which they dubbed Chandipura after the place where it was found, while looking through cases for possible dengue or chikungunya virus etiology[2]. The CHPV was discovered unusually. In the course of looking into a fever epidemic in Chandipura village, Bhatt and Rodrigues discovered a virus in the blood samples of those who were afflicted that had never been found before. With the discovery of the CHPV, the Virus Research Centre's database of arboviruses grew[3]. In 1967, the Virus Research Centre turned over control of the collection to the Indian Council of Medical Research, which called it the National Institute of Virology. The CHPV was a relatively unknown disease for a long time, drawing little interest from virologists. The virus was recovered from individuals with transient fevers, hedgehogs, sandflies (*Phlebotomus* spp.), and *Aedes aegypti* mosquitoes by scientists; however, because of the restricted and mild symptoms it seemed to induce, the virus was not thought to be of great significance. The virus was thought to be spread throughout India based on the identification of virus-specific antibodies in people and a variety of animals from diverse parts of the country[4]. However, such information was kept under concealment, mostly because the virus was not initially linked to any serious illnesses in humans[2]. When Rodrigues and colleagues reported isolating the CHPV from the blood of an 11-year-old kid who passed away from acute encephalopathy syndrome in 1983,

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it was the first time that the virus was clinically linked to a serious illness. Though it would take an additional twenty years for the virus to become widely recognized in the scientific world, this signaled the start of increasing knowledge of the virus's tendency to cause serious neurological sickness[5].

2. Epidemiology

CHPV can lead to outbreaks of acute encephalitis syndrome, especially in children living in rural India. Since its discovery, CHPV has been linked to many epidemics in various parts of India, such as Maharashtra, Gujarat, and Andhra Pradesh[2]. Rapid neurological symptom onset is a common feature of these outbreaks, which raises the case-fatality rate significantly, particularly in young children. Based on the epidemiological pattern, it appears that CHPV outbreaks are localized and irregular, frequently occurring during the monsoon season as the sandfly vector develops in warm and humid conditions. Due to the difficulty in diagnosing the virus and the underreporting of cases, it is still impossible to determine the exact prevalence of CHPV, especially in isolated rural regions with potentially inadequate healthcare facilities[6]. CHPV is not limited to India, despite its relationship with this country. Senegal, Nigeria, and Trinidad are among the other nations where the virus has been found, suggesting a wider global dispersion. However, because of the severity of the epidemics there, the Indian subcontinent has received the majority of study and attention, leaving the epidemiological impact of CHPV outside of India poorly recognized[7].

3. Transmission and propagation

The spread of the CHPV is primarily by the biting of infected sandflies, especially those belonging to the species *Phlebotomus*. These tiny, blood-feeding insects are the main CHPV vectors, and knowing how they spread the virus is essential to understanding its epidemiology[8]. A major factor in the transmission of CHPV is the life cycle of sandflies and their interactions with human populations. Usually popular in rural and semi-rural settings, sandflies are frequently seen near human habitations. Their natural habitats include leaf litter, soil rich in organic matter, and animal burrows, where they procreate. Sandfly activity rises in monsoon season, during which the majority of CHPV outbreaks occur. As they need blood meals to produce their eggs, female sandflies bite people and animals, which spreads the virus. After a sandfly becomes infected, the virus multiplies and spreads to more victims during future blood feeds. Multiple hosts are involved in the relatively intricate CHPV transmission cycle[9]. Although CHPV outbreaks mostly affect humans, it is thought that the virus spreads through

sylvatic cycles involving other vertebrates, maybe including small mammals or birds, which serve as reservoir hosts. These animals allow the virus to remain in the environment because they contain it without necessarily exhibiting symptoms of sickness. Although the exact makeup of these reservoir hosts is yet unknown, they play a critical function in keeping the virus inside the ecosystem in which it lives, where it can be transmitted to humans *via* sandfly bites. Since there is no evidence of CHPV spreading from person to person, arthropod vectors are still the main source of transmission. *Aedes aegypti* mosquitoes are among the insects in which CHPV has occasionally been found, which raises questions about the possibility of wider transmission paths[10]. Despite this, the bulk of human cases are attributed to sandflies as the primary vector.

4. Outbreaks

CHPV was not linked to serious human illness for a considerable amount of time following its discovery. The virus was mostly identified as the cause of minor, self-limiting feverish infections; it had little effect on public health overall. However, in the early 2000s, a number of outbreaks brought attention to the virus's ability to cause fatal neurological illnesses[11]. The first significant outbreak that made CHPV well known was in 2003 when researchers from the National Institute of Virology discovered that a significant number of young infants in Andhra Pradesh were suffering from severe neurological illnesses. With 329 cases and 183 deaths, the epidemic was marked by the quick onset of encephalitis symptoms such as fever, altered mental state, seizures, and vomiting. Children comprised the majority of those impacted, and the number of fatalities was shockingly high. The majority of survivors recovered quickly and experienced no long-term effects, whereas deaths frequently happened within 24 hours after the beginning of symptoms[12]. Investigations into a few of the hospitalized children during this outbreak produced strong evidence that the CHPV was the cause. White blood cells, a common indicator of inflammation in encephalitis, were not significantly elevated in the cerebrospinal fluid, according to a retrospective analysis of another group of 104 children hospitalized in Hyderabad during the same period. However, the clinical features of the group of children were consistent with encephalopathy rather than classic encephalitis. Acute fever, altered sensorium, seizures, diarrhea, and vomiting were the main symptoms of the sickness. These manifestations resulted in the clinical diagnosis of "brainstem encephalitis", with CHPV being determined to be the most likely cause[13].

After this epidemic, a second cluster of "acute encephalitis" cases primarily affecting tribal children was reported in Gujarat's eastern regions in 2005. Despite its lesser scope, this epidemic led to 26 cases and 18 deaths. Further scientific research was prompted by the

recurrent pattern of severe neurological disease and high mortality among children in these outbreaks, which generated serious concerns about the potential influence of CHPV on public health[12,14].

5. Scientific and medical response

CHPV, which was once thought to be a minor pathogen, drew the attention of scientists again after widespread outbreaks in the early 2000s. In order to learn more about the behavior, virulence, and potential for large-scale outbreaks of the virus, a number of additional studies were carried out. Scientists started examining the CHPV's growth kinetics in a variety of cell lines and chicken eggs[15]. These results shed light on the virus's capacity to infect a variety of host species, including both vertebrates and invertebrates.

6. Life cycle and pathophysiology of CHPV

Knowledge of the viral life cycle is important for fully understanding how the virus spreads and eventually causes disease in humans, especially encephalitis. It is thought that tiny animals and perhaps birds are the CHPV's natural reservoirs, where the life cycle of the virus starts[16]. Although they do not exhibit any symptoms, these animals carry the virus, which keeps them alive in the ecosystem. Then infected sandflies (*Phlebotomus* spp.), which are considered to be the principal vectors of the virus, bite people and other animals. Through feeding on the blood of a reservoir host, infected sandflies pick up the virus. The virus starts growing in the midgut of the sandfly before moving on to the salivary glands. The virus enters the host's circulation through the sandfly's saliva when it feeds on another host[17]. Insects such as *Aedes aegypti* mosquitoes have also been shown to transmit CHPV, despite sandflies being the main vectors. This raises worries about wider transmission, especially in urban areas where these mosquitoes are common.

After entering a human host, the virus begins to replicate, first in the blood artery lining endothelial cells and then maybe in other cell types like macrophages[18]. The virus must replicate in order to cause viremia, a situation in which the bloodstream allows the infection to spread throughout the body. The virus may spread widely during the viremia phase *via* the blood-brain barrier, which is a vital stage in the development of severe neurological disorders[19]. The host's immune system builds a defense to stop the illness as it progresses. Interferons and other cytokines are part of the innate immune response, which is crucial in the fight against viral reproduction. But occasionally, especially in young people, the immune system can become dysregulated, which can result in a "cytokine storm". This severe inflammatory reaction can exacerbate the condition by seriously damaging tissue, particularly brain tissue. The potential

of CHPV infection to penetrate the central nervous system is a particularly alarming feature[20]. One of the key events in the virus's pathogenesis that causes encephalitis is its breach of the blood-brain barrier. The brainstem, a crucial region of the central nervous system that regulates vital physiological processes including breathing and heart rate, is the main target of CHPV infection. Although the precise process by which CHPV causes neurological damage is not entirely understood, it is thought to entail both secondary immune-mediated harm and direct viral cytopathic effects, through which the virus directly affects brain cells. Quick and severe clinical manifestations, such as fever, convulsions, altered consciousness, and a quick progression to coma and death, are frequently the outcome of brainstem involvement[21]. When a person has a CHPV infection, their symptoms usually start unintentionally and get worse very rapidly. Typically, patients first experience a sharp increase in body temperature, which is followed by neurological symptoms including nausea, vomiting, and seizures. In many cases, the disease advances quickly and results in death 24 to 48 hours after the beginning of symptoms[2]. It is fairly rare for viral encephalitis survivors to recover so fast and often without permanent neurological abnormalities; this implies that the damage may be reversible if the immune response can be properly regulated.

CHPV vectors like sandflies get infected when they ingest the blood from an infected human or animal, continuing the virus's cycle of infection. While vector bites are the primary means of transmission, there is evidence that the virus can also transmit through physiological fluids including blood and saliva during the acute stage of sickness[22]. Nonetheless, there remains a lack of verified human-to-human transmission, and vectors continue to be the predominant mode of transmission. The pathophysiology of CHPV is shown in a schematic diagram (Figure 1) with parts that correspond to the many stages of the virus's journey[23,24].

7. Symptoms of CHPV infection

The onset of CHPV infection is typically marked by a sudden high fever. This fever is often accompanied by severe headaches, which can be debilitating for the patient. Vomiting is another common symptom, adding to the overall discomfort and contributing to the risk of dehydration, which can exacerbate the patient's condition. As the infection progresses, more severe neurological symptoms begin to manifest. Convulsions or seizures are a prominent feature of CHPV infection, indicating that the virus has begun to affect the central nervous system[25]. These seizures can be frequent and intense, posing a significant risk to the patient's health. Another critical symptom is altered mental status. Patients may experience confusion, irritability, and varying degrees of consciousness, ranging from drowsiness to full-blown delirium. In many cases, these

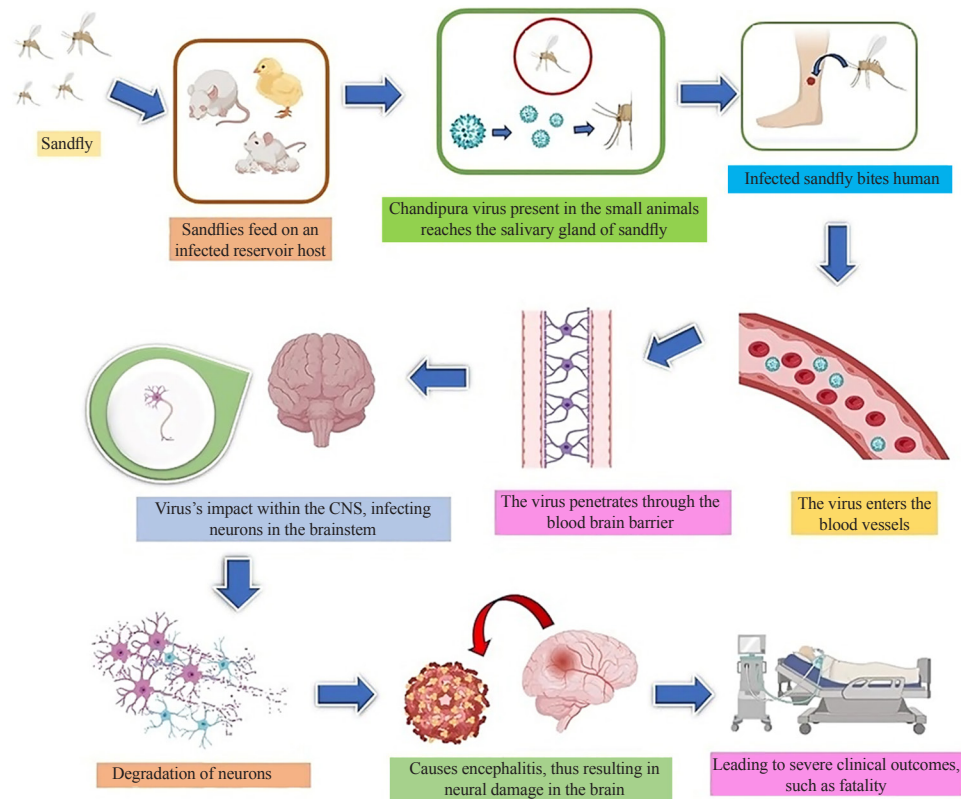


Figure 1. Pathophysiology of Chandipura virus.

symptoms rapidly progress to more severe neurological impairment, and if left untreated, they can lead to coma. The progression from initial symptoms to severe neurological complications can occur within a matter of days, making the disease particularly dangerous[26].

8. Treatment of CHPV infection

Currently, there is no specific antiviral treatment or vaccine available for CHPV infection. The management of the disease primarily involves supportive care aimed at alleviating symptoms and preventing complications. For patients with severe symptoms, hospitalization is often required, particularly for children, who are the most vulnerable to the severe effects of the virus. In a hospital setting, healthcare providers can closely monitor the patient's condition and provide the necessary care to manage symptoms[27]. One of the critical aspects of supportive care is maintaining proper hydration, especially in patients experiencing frequent vomiting. Dehydration can worsen the patient's condition and lead to further complications, so intravenous fluids may be administered to ensure adequate hydration. Medications, such as antipyretics, are used to manage fever, while anticonvulsants are given to control seizures, which are a common and dangerous symptom of CHPV infection. In cases where the patient exhibits severe neurological symptoms, intensive care may be necessary[28]. This can include respiratory

support and other interventions to manage complications that arise from the infection's impact on the central nervous system. Due to the lack of a specific antiviral treatment, preventive measures, such as controlling sandfly populations and limiting exposure to these vectors, are crucial in reducing the risk of infection. Public health efforts focus on educating communities about the dangers of sandfly bites and implementing strategies to control the vector population in areas prone to outbreaks[29].

9. Rise of CHPV in India in 2024

CHPV, which was discovered in a Maharashtra village in 1965, has resurfaced as a serious hazard to public health, especially in that region of India. The virus came to light in 2003 after an outbreak in Andhra Pradesh, where 329 children were afflicted and 183 of them died from the illness, even though it was discovered decades before. This startling death rate indicated that CHPV, which mostly affects youngsters and causes serious neurological problems, had the potential to be extremely lethal. With 26 cases documented and an alarming 78% death rate, the 2005 Gujarat epidemic brought the virus's lethal effects even further to light. CHPV epidemics are now known to be characterized by this pattern of significant mortality, especially in children. Gujarat continues to be a CHPV hotspot based on recent data.

The CHPV primarily affects children under the age of 15,

presenting with symptoms such as sudden high fever, altered sensorium, and seizures, which can quickly progress to coma and death if not treated promptly. A three-year-old boy from Dungarpur in Rajasthan tested positive for the virus, as confirmed by the National Institute of Virology in Pune[30]. By July 31st 2024, India had recorded 53 confirmed cases of CHPV, with 51 cases in Gujarat and 2 cases in Rajasthan, according to Union Health Minister J. P. Nadda, who provided this information in a written response to the Rajya Sabha on August 6, 2024. Out of these 53 cases, 19 people have died, all of whom were from Gujarat. Minister Nadda also detailed the steps taken to control the outbreak, including the deployment of a National Joint Outbreak Response Team to assist the Gujarat government in implementing public health measures and conducting detailed epidemiological investigations[31]. The rapid spread of the CHPV across India has raised significant concerns among health experts. Notably, Rajasthan detected its first case after Gujarat reported 51 cases. In response, the Rajasthan Medical and Health Department issued an advisory outlining necessary precautions[32].

Public health officials and medical professionals are seriously concerned about the virus's quick spread and the severity of its symptoms. The relationship between rising CHPV incidence and climate change has drawn the attention of experts. Vector-borne infections, such as those carried by sandflies, are thought to proliferate due to rising temperatures and shifting environmental factors. These vectors' reproductive season gets delayed by the warmer weather, which also broadens their geographic distribution and may expose additional populations to the virus. This pattern raises concerns since it may indicate that CHPV will spread more widely and cause more frequent and severe outbreaks[33,34]. The high death rate which can range from 56% to 75% depending on the outbreak emphasizes the necessity of efficient public health initiatives. National Joint Outbreak Response Teams have been sent by the Indian government to help state health officials contain the outbreak. To stop the virus from spreading, teams of scientists from the National Institute of Virology Pune, the Indian Council of Medical Research, and the National Centre for Disease Control are carrying out epidemiological studies and putting vector control measures into place[30].

Research on entomology is essential to comprehending the vectors that transmit CHPV. Public health professionals can more effectively target control efforts by identifying the particular species of sandflies or other insects that are responsible for transmitting the virus. To lower the number of vectors and stop the virus's transmission, these measures include indoor residual spraying in the afflicted areas. Accredited Social Health Activists, Auxiliary Nurse Midwives, and health fast response teams are actively engaged in monitoring and early case detection, guaranteeing timely referral to healthcare facilities equipped with specialist services and ventilator support.

The absence of a vaccine or targeted antiviral medication continues to be a major obstacle in the fight against CHPV, notwithstanding recent efforts. The virus is a dangerous infection since it can quickly and severely harm the nervous system, especially in young patients. With the ongoing expansion of CHPV, there is an increasing risk not just to India but also to other countries, particularly given the effects of climate change. From its discovery in 1965 to the present, the history of CHPV shows how the virus has changed from being a relatively unknown infection to a major public health issue. In order to tackle this lethal virus, there is an urgent need for further research, monitoring, and public health preparation, as demonstrated by the outbreaks in India, especially in Gujarat. It is essential to take preventive steps to safeguard vulnerable communities and stop future outbreaks from reaching the catastrophic levels witnessed in recent years, as climate change is expected to worsen the spread of vector-borne illnesses[35]. The recent cases of this virus are demonstrated in Figure 2.

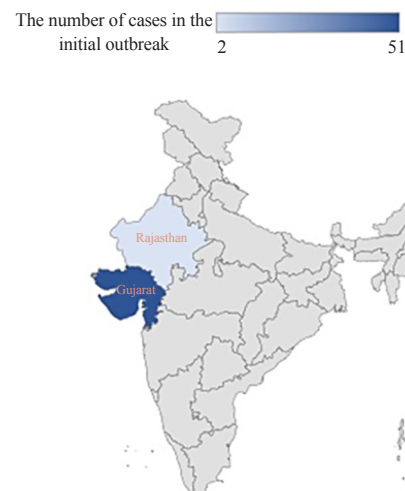


Figure 2. Recent cases of Chandipura virus in India in 2024[31].

10. Prevention

A comprehensive strategy centered on vector control, personal protection, environmental management, and early diagnosis is needed to stop the spread of CHPV. Sandflies are not only vectors for Chandipura virus, but also for *Leishmania* parasites, causing diseases like visceral leishmaniasis (Kala-azar) and cutaneous leishmaniasis in India. These diseases affect various regions, with *Phlebotomus argentipes* identified as a primary vector for visceral leishmaniasis[36]. It is critical to manage the main vector, sandflies by spraying insecticides, removing vegetation and standing water from breeding grounds, and using insect repellents. To keep sandflies out of houses, personal precautions should be taken including putting on bed nets sprayed with pesticide, putting in fine mesh screens on windows and doors, and wearing long sleeves[3]. Preventive

measures mostly rely on raising community knowledge. Crucial actions include informing the public about the dangers, promoting appropriate sandfly management, and advocating for personal safety. Sandfly populations are further decreased by environmental management practices including keeping the surrounding area clean. For quick diagnosis and treatment, early detection through surveillance systems and healthcare worker training is crucial[10].

11. Future prospective

CHPV represents a looming threat to Gujarat and India as a whole, particularly as climate change and environmental shifts create more favorable conditions for its primary vector. With rising global temperatures and altered weather patterns, the habitats of sandflies could expand, increasing the geographic range of CHPV and the frequency of outbreaks across India. In Gujarat, the virus has already demonstrated its capacity for rapid and deadly outbreaks. The state's large rural population, combined with its hot and arid climate, provides an ideal environment for sandflies to thrive, increasing the risk of the virus becoming endemic in certain regions. If not controlled, CHPV could terrorize Gujarat by causing repeated, severe outbreaks, particularly in areas with limited access to healthcare and vector control measures. On a broader scale, the spread of CHPV across India could have devastating consequences. The virus could potentially extend its reach to new states, especially those with similar climatic conditions to Gujarat. Without effective interventions, including vector control, public health education, and the development of antiviral treatments or a vaccine, CHPV could cause significant public health crises across the country. High mortality rates, as seen in past outbreaks, could lead to widespread fear and panic, disrupting daily life and overwhelming healthcare systems.

The economic impact could also be severe, with repeated outbreaks affecting tourism, agriculture, and other key sectors in Gujarat and beyond. The potential for CHPV to cause widespread fear and high mortality rates underscores the urgency of addressing this threat. Without decisive action, CHPV could terrorize Gujarat and other parts of India, leading to severe public health, social, and economic consequences.

12. Conclusion

CHPV poses a public health threat in India. The correlation between the rising prevalence of CHPV and climate change emphasizes how urgent it is to confront this new threat. Moreover, the lack of a vaccine or particular antiviral therapy makes effective public health measures more crucial. Proactive public health interventions,

improved surveillance, and intensified research are essential to averting future epidemics and safeguarding susceptible groups.

Conflict of interest statement

The authors declare that there is no conflict of interest.

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Data availability statement

The data supporting the findings of this study are available from the corresponding authors upon request.

Authors' contributions

RD conceptualized and designed the work, carried out data collection, and drafted the article. PAC performed data analysis and interpretation, critically reviewed the article, and was responsible for final approval of the version to be published.

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