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Review Article

A Comprehensive Review on Chandipura Virus

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ABSTRACT

Chandipura virus (CHPV), a member of the Rhabdoviridae family and Vesiculovirus genus, is an emerging neurotropic virus identified primarily in India. It causes acute encephalitis-like illness, mainly affecting children under 15 years, with a rapid clinical course and high case fatality rate. The virus is transmitted primarily by sandflies, with potential mosquito involvement. Since its discovery in 1965, CHPV outbreaks have posed significant public health concerns, especially in endemic regions. There is currently no specific antiviral therapy or vaccine available, making early diagnosis, vector control, and supportive treatment critical in managing outbreaks. Increased surveillance, molecular diagnostics, and "One Health" initiatives are key strategies to mitigate the impact of this deadly virus.

INTRODUCTION

Chandipura virus (CHPV) is an emerging viral pathogen belonging to the Vesiculovirus genus of the Rhabdoviridae family. It was first isolated in 1965 from a patient in Chandipura village, Maharashtra, India[1]. The virus is genetically related to the rabies virus and shares the characteristic bullet-shaped morphology typical of rhabdoviruses. CHPV is a negative-sense, single-stranded RNA virus that primarily exhibits neurotropism—meaning it preferentially infects and damages neural tissue. Since its discovery, the virus has been implicated in several outbreaks of acute encephalitis-like illness, particularly affecting children under the age of 15 years.

Transmission of Chandipura virus is predominantly vector-borne, with sandflies (Phlebotomus species) identified as the primary vectors[3]. However, some entomological studies also suggest the potential role of mosquitoes such as Aedes aegypti and Culex species in viral transmission. Seasonal outbreaks tend to occur during the monsoon months, coinciding with heightened vector activity. Endemic regions in India, especially in Andhra Pradesh, Maharashtra, Gujarat, and Madhya Pradesh, have reported

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sporadic outbreaks with alarmingly high case fatality rates ranging from 55% to 75%, making the virus a significant pediatric health concern in affected areas.

Clinically, CHPV infection is characterized by a sudden onset of high-grade fever, vomiting, altered mental status, seizures, and rapid progression to coma and death in severe cases. The disease course is fulminant, with many children succumbing within 24–48 hours of symptom onset. Due to the absence of a specific antiviral treatment or licensed vaccine, the current management is limited to supportive care and intensive monitoring. The World Health Organization (WHO) and Indian Council of Medical Research (ICMR) recognize CHPV as a virus of public health importance, emphasizing the need for enhanced surveillance, vector control, and research into effective diagnostics, therapeutics, and vaccine.

ETIOLOGY

Chandipura virus (CHPV) is a negative-sense, single-stranded RNA virus belonging to the genus Vesiculovirus within the family Rhabdoviridae. Like other rhabdoviruses, it exhibits a characteristic bullet-shaped morphology. The viral genome is approximately 11 kilobases in length and encodes five structural proteins: Nucleoprotein (N), Phosphoprotein (P), Matrix protein (M), Glycoprotein (G), and Large protein (L) or RNA-dependent RNA polymerase. These proteins play critical roles in viral replication, transcription, and assembly. Among these, the G protein is responsible for attachment and entry into host cells, while the L protein facilitates RNA synthesis. CHPV exhibits marked neurotropism and has the capacity to invade the central nervous system (CNS), leading to severe neurological manifestations in infected individuals, particularly children.

The principal vector implicated in CHPV transmission is the Phlebotomus species of sandflies. These vectors acquire the virus during blood feeding on viremic hosts and transmit it to new hosts during subsequent feeding. Although sandflies are the primary vector, some entomological studies have indicated that mosquitoes such as Aedes aegypti and Culex quinquefasciatus may act as secondary or mechanical vectors, especially in areas where sandflies and mosquitoes coexist. The transmission cycle is not completely understood but is believed to be maintained in nature through a zoonotic reservoir involving small mammals or other vertebrates. Seasonal prevalence, particularly during and after the monsoon, corresponds with increased vector activity, which aligns with the timing of reported outbreaks in endemic regions of India.

EPIDEMOLOGY

Chandipura virus (CHPV) is primarily endemic to India, with major outbreaks reported in the states of Andhra Pradesh, Maharashtra, Gujarat, and Madhya Pradesh. The virus predominantly affects children aged 2–14 years, with case fatality rates ranging from 55% to 75% during outbreaks. The first isolation occurred in 1965, but large-scale epidemics were documented in 2003 (Andhra Pradesh), 2007 (Maharashtra), and more recently in 2019 in the Nagpur region. CHPV outbreaks typically occur during the monsoon and post-monsoon seasons, correlating with increased vector activity—particularly Phlebotomus sandflies. Serological studies have also detected CHPV antibodies in asymptomatic individuals and animals, suggesting subclinical infections and possible zoonotic reservoirs. Although the majority of reported cases are from India, seroprevalence and viral RNA detection in Nepal

and Sri Lanka indicate the potential for broader regional distribution.

PATHOPHYSIOLOGY

Vector Bite (Sandfly/Mosquito)
↓
Viral Entry into Skin & Local Replication
↓
Primary Viremia (Virus enters bloodstream)
↓
Systemic Dissemination
↓
Crossing the Blood–Brain Barrier (BBB)
↓
CNS Invasion – Neurotropism (Infects neurons)
↓
Activation of Immune Response
- Cytokine storm
- Glial cell activation
↓
Neuroinflammation
- Cerebral edema
- Neuronal necrosis
- Perivascular cuffing
↓
Clinical Manifestation
- Fever, seizures, altered consciousness
- Rapid progression to coma/death

Chandipura virus (CHPV) enters the human host primarily through the bite of infected Phlebotomus sandflies. Upon transmission, the virus first replicates locally in the dermis and regional immune cells. It then spreads systemically via the bloodstream, resulting in primary viremia. The virus has a high affinity for neural tissues and is capable of breaching the blood–brain barrier—either by infecting endothelial cells or through Trojan horse mechanisms involving infected leukocytes. Once inside the central nervous system (CNS), CHPV directly infects neurons, triggering robust neuroinflammation. This inflammatory cascade is characterized by the release of pro-inflammatory cytokines, chemokines, and activation of microglia, which amplifies the damage to neuronal tissues. Neuropathological changes such as cerebral edema, perivascular lymphocytic infiltration, microglial nodules, and neuronal necrosis are commonly observed. Clinically, this leads to rapid deterioration with symptoms like high-grade fever, seizures, altered sensorium, and death within 24 to 48 hours in severe cases, especially in young children. The aggressive neurotropic nature of CHPV and the host's hyperinflammatory response are central to its high case fatality rate.

CLINICAL FEATURES

1. Incubation Period

- The incubation period ranges from 1 to 6 days, though most symptomatic cases begin within 2–4 days after the infective sandfly bite.

2. Initial Symptoms

- The disease often begins abruptly with high-grade fever, headache, and myalgia.
- Unlike some viral encephalitis, there are usually no prominent prodromal signs, making the onset appear sudden and severe.

3. Neurological Manifestations

- Altered sensorium (confusion, lethargy, stupor) develops early in the course of illness.
- Seizures, particularly generalized tonic-clonic seizures, are a hallmark feature.
- Vomiting and irritability are frequently observed, especially in infants and young children.
- As the infection progresses, patients may become unconscious, and coma can ensue within hours.
- Neck rigidity and other signs of meningeal irritation may be present in some cases.

4. Rapid Deterioration

- In many outbreaks, children have been reported to die within 24–48 hours of symptom onset, particularly when seizures and coma appear early.
- The case fatality rate has ranged from 55% to 75% in various reported outbreaks, highlighting the urgency of prompt recognition and supportive care.

5. Neurological Sequelae in Survivors

- Children who survive the acute phase may develop long-term neurological deficits such as:

a) Cognitive impairment
b) Behavioral issues
c) Speech and motor deficits
d) Seizure disorders (e.g., epilepsy)

- The severity of sequelae depends on the degree of central nervous system involvement and the timeliness of medical intervention.

6.Atypical Features

- In some rare cases, symptoms like photophobia, papilledema, and focal neurological deficits (e.g., hemiparesis) have been reported.

DIAGNOSIS

Early and accurate diagnosis of Chandipura virus infection is critical due to the rapid clinical progression and high mortality, particularly among children. The gold standard diagnostic method is reverse transcription polymerase chain reaction (RT-PCR), which detects CHPV RNA in serum or cerebrospinal fluid (CSF) during the acute phase of the illness. RT-PCR is highly sensitive and specific and is most effective when performed within the first 3–5 days of symptom onset. Another confirmatory method includes virus isolation using cell cultures such as Vero or C6/36 cell lines, although this technique is time-consuming and limited to specialized virology laboratories.

Serological tests, such as IgM-capture ELISA, can detect antibodies against CHPV and are useful in the later stages of illness or for retrospective confirmation. However, due to the rapid progression of disease, many patients may succumb before mounting a detectable antibody response. Neuroimaging (such as CT or MRI) may reveal features like cerebral edema, but findings are typically nonspecific. In endemic regions, diagnosis is often based on a combination of clinical symptoms (acute febrile encephalopathy in children), epidemiological linkage (seasonal outbreaks), and exclusion of other common causes of acute encephalitis syndrome (e.g., Japanese encephalitis, herpes simplex virus, dengue, or bacterial meningitis). A strong index of suspicion, especially during monsoon seasons in outbreak-prone areas, is essential for timely diagnosis and supportive care.

TREATMENT

Currently, there is no specific antiviral treatment available for Chandipura virus infection. Management primarily revolves around supportive care to alleviate symptoms and prevent complications. As the disease progresses rapidly—often leading to coma and death within 24 to 48 hours—early hospital admission and intensive monitoring are critical. Patients presenting with high-grade fever and altered sensorium, particularly children during outbreak seasons in endemic areas, should be treated as medical emergencies.

Supportive treatment includes antipyretics to manage fever and anticonvulsants such as phenytoin or levetiracetam for seizure control. In

cases of raised intracranial pressure or cerebral edema, osmotic diuretics like mannitol may be administered to reduce brain swelling. Adequate hydration, electrolyte balance, and maintenance of oxygenation through supplemental oxygen or mechanical ventilation are essential aspects of care in severe cases. Continuous neurological assessment and prompt intervention can help prevent further deterioration.

Experimental treatments have been attempted during past outbreaks, including the use of ribavirin, an antiviral agent known to inhibit RNA viruses. However, clinical outcomes have been inconclusive, and there is no established evidence of benefit in CHPV. Some reports also mentioned the use of corticosteroids to mitigate inflammatory responses and cerebral inflammation, but again, data supporting their efficacy remain limited. Due to the lack of a proven antiviral or immunomodulatory therapy, the focus remains on preventing secondary complications through intensive supportive management.

Moreover, intensive care unit (ICU) support is vital for critically ill patients. Facilities should be equipped to handle pediatric encephalitis cases during seasonal outbreaks. Long-term rehabilitation may be necessary for survivors who suffer from neurological sequelae such as motor deficits or cognitive impairments. Efforts are ongoing to develop antiviral therapies targeting Chandipura virus replication and immunopathology, but none are currently available for clinical use.

VACCINATION

1. Recombinant Vesiculovirus-Based Vaccine

- Type: Live attenuated recombinant vaccine
- How Given: Intramuscular (IM) injection in animal models
- Mechanism: Engineered vesiculoviruses express Chandipura glycoproteins to stimulate an immune response.
- Status: Preclinical (tested in mice); showed good immunogenicity and survival benefit.

2. DNA Vaccine

- Type: Plasmid DNA encoding CHPV glycoprotein
- How Given: Intramuscular injection with or without electroporation
- Mechanism: Host cells produce viral proteins that trigger immune response.
- Status: Preclinical trials in mice; showed antibody production, but protection level under evaluation.

3. Inactivated Whole-Virus Vaccine

- Type: Formalin-inactivated CHPV
- How Given: Subcutaneous or intramuscular injection (animal models)
- Mechanism: Inactivated virus particles stimulate immune system without causing infection.
- Status: Laboratory and animal studies; safe but needs human trials.

4. Virus-Like Particle (VLP) Vaccine

- Type: Recombinant proteins forming virus-like particles
- How Given: Not yet tested in animals; assumed intramuscular route
- Mechanism: Mimics virus structure without containing genetic material, inducing immunity.
- Status: Early research phase; shown promise in lab experiments.

PREVENTION

Prevention of Chandipura virus primarily revolves around vector control and minimizing human exposure to sandflies, the main vectors responsible for virus transmission. In endemic areas, efforts should focus on reducing sandfly breeding sites by improving sanitation, clearing vegetation near homes, and eliminating damp soil and organic debris where sandflies lay eggs. Regular use of indoor residual insecticide spraying, especially during peak sandfly seasons (monsoon), has proven effective in reducing vector populations. Insecticide-treated bed nets and mosquito repellents (containing DEET or permethrin) can also serve as a personal line of defense, particularly for children who are at higher risk.

Public awareness and health education are crucial components of prevention. Families in rural and tribal regions—where most outbreaks occur—must be informed about the symptoms of CHPV, such as high-grade fever, seizures, and altered mental status, and the need for urgent medical attention. Community participation in vector control programs and reporting of suspected encephalitis cases during outbreak seasons can greatly aid in early detection and containment. Schools, healthcare workers, and local governance systems should be involved in seasonal awareness campaigns to reinforce hygiene and vector-prevention practices.

Surveillance and early outbreak response systems play a pivotal role in preventing widespread transmission. Strengthening laboratory capacities for CHPV diagnostics (e.g., RT-PCR and ELISA) at district levels enables faster identification of cases and implementation of control measures. Sentinel surveillance in high-risk zones, along with a "One Health" approach involving coordination between veterinary, entomological, and human health sectors, can help identify zoonotic and vector-based threats early. Until a safe and effective vaccine is available, prevention through vector control, public engagement, and timely healthcare intervention remains the most effective strategy to combat Chandipura virus outbreaks.

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