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Chandipura Virus: An Emerging Cause of Acute Encephalitis in Children and a Persistent Public Health Challenge in India

Dr. Amit Sachdeva[1*], Mr Nasim Ahmed[2], Mrs. Tamanna Rahman[3], Dr. Anju Sachdeva[4]

[1]Assistant Professor, Department of Community Medicine, Indira Gandhi Medical College, Shimla, Himachal Pradesh
[2]Independent Researcher, Guwahati Assam, India Email: nasim@iarcon.org
[3]MSc in Herbal Science and Technology , Anandaram Dhekial Phookan College under Guwahati University, Assam
[4]Physiotherapist, Shimla, Himachal Pradesh

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Abstract: Background: Chandipura virus (CHPV) is an arthropod-borne vesiculovirus associated with rapidly progressive acute febrile encephalopathy, predominantly among children in India. Although recognized since 1965, CHPV gained major public health importance after high-fatality outbreaks in central and western India. The large outbreak reported in 2024 and renewed transmission during 2026 demonstrate that the virus remains an inadequately understood epidemic threat. **Objective:** This narrative review critically examines the virology, epidemiology, transmission, clinical spectrum, diagnosis, treatment and prevention of CHPV and identifies priorities for surveillance, research and outbreak preparedness. **Key findings:** CHPV is an enveloped, negative-sense RNA virus belonging to the genus *Vesiculovirus* in the family *Rhabdoviridae*. Phlebotomine sandflies are considered the principal vectors, but the natural transmission cycle and vertebrate reservoir remain uncertain. Clinically recognized disease occurs mainly in children and may progress within hours from fever and vomiting to seizures, altered consciousness, cerebral oedema, shock and death. The 2024 Gujarat-centred outbreak showed that CHPV infection can also cause a non-neurological febrile illness and that asymptomatic exposure may be common. Diagnosis depends on timely reverse-transcription polymerase chain reaction, virus isolation or immunoglobulin M testing because routine cerebrospinal-fluid findings may be non-specific. No licensed vaccine or proven antiviral treatment is available. Rapid recognition, intensive supportive care and management of raised intracranial pressure are therefore critical. Vector control is recommended, but intervention effectiveness is poorly quantified because the relevant vectors, breeding habitats and transmission pathways may vary between outbreaks. **Conclusion:** CHPV should be incorporated more systematically into acute encephalitis and acute febrile illness surveillance in endemic and neighbouring regions. Progress requires rapid decentralized diagnosis, prospective clinical studies, integrated human–vector–animal surveillance, standardized case management and development of vaccines and therapeutics.

Keywords: Chandipura virus; acute encephalitis syndrome; sandfly; vesiculovirus; paediatric encephalitis; India

INTRODUCTION

Chandipura virus is a neurotropic arthropod-borne virus associated with explosive outbreaks of acute encephalitis syndrome (AES), principally among children in India. It was first isolated in 1965 from the blood of two adults with a dengue-like febrile illness in Chandipura village, Maharashtra. For several decades it was regarded as an obscure arbovirus of uncertain clinical importance. This perception changed after the 2003 outbreak in Andhra Pradesh and Maharashtra, when hundreds of children developed rapidly progressive encephalitic illness with a high case-fatality rate.[1]

The disease is unusual because deterioration can be exceptionally rapid. A child may initially have fever, headache or vomiting and progress within 24–48 hours to seizures, coma, cardiorespiratory instability and death. This narrow therapeutic window places exceptional demands on families, peripheral health facilities, ambulance systems and paediatric intensive-care services.

CHPV is endemic in parts of India, with outbreaks and sporadic cases documented in Andhra Pradesh, Maharashtra, Gujarat, Madhya Pradesh, Odisha and neighbouring areas. The 2024 outbreak was the largest reported in approximately two decades. Between early June and August 15, 2024, India reported 245 AES cases and 82 deaths; 64 cases were laboratory-confirmed as CHPV infection.[2] The outbreak was concentrated in Gujarat but included cases from Rajasthan, Madhya Pradesh and Maharashtra.

A detailed 2025 outbreak investigation subsequently demonstrated a broader clinical and epidemiological spectrum than previously appreciated. CHPV immunoglobulin M was identified among patients with acute febrile illness without encephalitis, while neutralizing antibodies were detected in substantial proportions of sampled humans and some animals.[3] These findings suggest that severe paediatric encephalitis may represent only the visible portion of a larger spectrum of infection.

The renewed outbreak investigation initiated in Gujarat and Rajasthan in August 2026 underscores the continuing relevance of CHPV. However, the precise vector, reservoir, transmission intensity and final burden of the current outbreak remain under investigation. Public communication should therefore distinguish established knowledge from preliminary field observations.

Virology

CHPV belongs to the order *Mononegavirales*, family *Rhabdoviridae* and genus *Vesiculovirus*. It is structurally related to other vesiculoviruses but is clinically distinct from rabies virus, despite belonging to the same viral family.

The virion is enveloped and typically bullet shaped. Its approximately 11-kilobase, single-stranded, negative-sense RNA genome encodes five principal structural proteins: nucleoprotein, phosphoprotein, matrix protein, glycoprotein and the large RNA-dependent RNA polymerase. The surface glycoprotein mediates attachment and entry and is an important target for neutralizing antibodies. The nucleoprotein protects viral RNA, while the large protein and phosphoprotein form the viral polymerase complex.

CHPV replicates rapidly in susceptible cells and can produce marked cytopathic effects. Experimental studies indicate neurotropism and the capacity to induce neuronal injury, inflammatory signalling and disruption of the blood–brain barrier. Proposed mechanisms of severe neurological disease include direct viral replication in neural tissue, endothelial injury, cytokine-mediated inflammation, cerebral oedema and disturbed cerebral perfusion.

Genomic studies have identified multiple viral lineages circulating in India. Analysis of the 2024 Gujarat isolate showed close similarity to viruses detected in Gujarat during 2012 and 2014 rather than evidence of an entirely novel lineage.[3] This finding argues against assuming that every large outbreak is caused by a newly emerged hypervirulent strain. Outbreak magnitude may instead reflect interactions among vector abundance, population susceptibility, climate, housing, delayed detection and health-system access.

Transmission Ecology

Phlebotomine sandflies are considered the most important vectors. CHPV has been isolated from sandflies in India and parts of Africa, and experimental studies have demonstrated replication and transmission in sandfly species. Sandflies are small, weak-flying insects that rest in dark, humid locations, including cracks in mud walls, animal shelters, crevices and organic debris.

The specific vector responsible for each outbreak is not always established. Experimental or field evidence has suggested possible roles for mosquitoes, ticks or other arthropods, but this evidence is substantially weaker than that for sandflies. During the 2024 Gujarat investigation, collected vector pools did not yield CHPV, illustrating the difficulty of identifying an infected vector after transmission has occurred.[3]

The vertebrate reservoir remains unknown. Antibodies have been found in domestic and wild animals, but seropositivity establishes previous exposure rather than reservoir competence. To qualify as a reservoir, an animal population would need to sustain viral circulation and produce viraemia sufficient to infect vectors. Detection of antibodies in cattle, goats or other animals does not prove that they transmit infection to humans.

Human-to-human transmission has not been established. Consequently, routine isolation of patients as if CHPV were a directly contagious respiratory infection is not supported. Standard infection-prevention

precautions remain appropriate in clinical facilities, while outbreak control focuses primarily on rapid case detection and reduction of vector exposure.

Outbreaks frequently occur during or around monsoon periods, although the relationship between temperature, rainfall and transmission is complex. Rainfall may increase humidity and breeding habitats, whereas very heavy rain can disrupt some vector populations. Mud housing, wall cracks, animal proximity, poor waste management and limited access to vector protection may contribute to exposure. These associations require stronger analytical study before they are translated into precise causal claims.

Epidemiology and Major Outbreaks

After its original identification in 1965, CHPV was only occasionally isolated from humans. The 2003 outbreak established its association with severe childhood encephalitis. Rao and colleagues investigated 329 children with acute encephalitis in Andhra Pradesh; 183 died, and virological evidence implicated CHPV in the outbreak.[1]

An outbreak in eastern Gujarat during 2004 further demonstrated severe paediatric disease and rapid progression.[4] Subsequent cases and outbreaks occurred in Maharashtra, Gujarat and other states, although reported burden varied according to laboratory access and surveillance intensity.

The concentration of severe disease among children remains incompletely explained. Adults may have partial immunity following earlier exposure, while developmental differences in immune response, blood–brain-barrier function or neuroinvasion may also contribute. Serological findings suggesting widespread exposure among adults support the possibility that many infections are asymptomatic or mild.

The 2024 outbreak provided several important insights. WHO reported a crude case-fatality proportion of approximately one-third among all notified AES cases, although not all AES cases were laboratory-confirmed as CHPV.[2] The dedicated virological investigation of confirmed cases reported a higher case-fatality proportion, emphasizing that estimates depend on the denominator and case definition used.[3]

This distinction is essential. The case-fatality rate among laboratory-confirmed encephalitis cases cannot be applied to all CHPV infections because mild and asymptomatic infections are likely undercounted. Conversely, calculating fatality among all AES cases may dilute CHPV-specific severity by including other aetiologies.

The ongoing 2026 investigation has expanded surveillance to include acute febrile illness as well as AES, community serosurveys, animal sampling, vector studies and whole-genome sequencing. This wider approach may clarify the proportion of infections that remain mild and whether children with encephalitis differ immunologically or virologically from those with uncomplicated fever.

Clinical Spectrum and Pathogenesis

The incubation period is not precisely established but is thought to be short. Initial manifestations commonly include abrupt high fever, headache, vomiting, malaise and altered behaviour. Diarrhoea or other non-specific symptoms may occur.

Neurological deterioration may follow rapidly, with irritability, drowsiness, seizures, confusion, loss of consciousness and coma. Severe cases may develop cerebral oedema, shock, respiratory failure and multiorgan dysfunction. Some historical reports described normal or minimally inflammatory cerebrospinal fluid despite profound neurological illness, contributing to debate over whether the syndrome represents conventional encephalitis, encephalopathy, vasculopathy or a combination of these processes.

Neuroimaging has shown cerebral oedema, cortical abnormalities and, in some cases, vascular or infarct-like changes. These observations have led to hypotheses involving endothelial injury, vasospasm and impaired cerebral perfusion. However, imaging findings are not uniform, and severe CHPV disease should not be reduced to one pathological mechanism.

The recognition of CHPV-positive acute febrile illness without neurological involvement is a recent and important development.[3] It raises the possibility of a clinical continuum from asymptomatic infection to uncomplicated fever and fulminant encephalitis. Prospective surveillance is needed to determine what proportion of febrile infections progress and which host or viral factors predict deterioration.

Survivors may recover completely, particularly when intensive care is available promptly. Nevertheless, neurological sequelae—including epilepsy, weakness, cognitive impairment or behavioural changes—can occur. Long-term outcome data remain limited because most outbreak studies focus on acute mortality rather than standardized neurodevelopmental follow-up.

Diagnosis and Differential Diagnosis

The clinical presentation is not specific enough for diagnosis. During the monsoon, childhood AES in India has a broad differential diagnosis, including Japanese encephalitis, dengue, scrub typhus, herpes simplex encephalitis, enteroviruses, bacterial meningitis, cerebral malaria, leptospirosis, toxic encephalopathy and metabolic disorders.

CHPV should be suspected in a child from or recently present in an affected area who develops acute fever followed by rapid neurological deterioration. Suspicion should increase during a recognized cluster, but laboratory confirmation remains necessary.

Reverse-transcription polymerase chain reaction is most useful during the early viraemic phase. Serum, cerebrospinal fluid and other specimens should be collected as early as possible, ideally before antibody responses dominate and before repeated freeze–thaw cycles degrade RNA. Immunoglobulin M enzyme-linked immunosorbent assay can support diagnosis later in illness. Virus isolation and genomic sequencing are generally confined to reference laboratories.

A negative result does not completely exclude infection when specimens are collected late, transported poorly or obtained after viraemia has declined. Conversely, detection of antibody alone may require interpretation in relation to timing and background population exposure.

Laboratory systems should test for multiple AES pathogens rather than stopping after one agent is suspected. Coinfection, incidental seropositivity and outbreak-related diagnostic anchoring can otherwise lead to misclassification.

Table 1: Chandipura Virus: Evidence, Clinical Implications and Public Health Priorities

Domain	Current evidence	Clinical or public health implication	Major uncertainty	Priority action
Virology	Enveloped negative-sense RNA vesiculovirus with five major structural proteins	RT-PCR and sequencing can provide early confirmation and molecular surveillance	Viral determinants of neurovirulence are incompletely defined	Expand genomic and functional studies using standardized clinical metadata
Vector	Sandflies have the strongest field and experimental support	Vector control should include habitats relevant to sandflies	Responsible vector may differ between outbreaks; mosquitoes and other arthropods remain under investigation	Conduct longitudinal entomological surveillance before, during and after outbreaks
Reservoir	Antibodies have been detected in several animal species	One Health investigation is justified	Seropositivity does not prove reservoir competence	Study viraemia, vector infectivity and ecological maintenance in candidate hosts
Human transmission	Direct person-to-person spread has not been established	Community isolation and contact quarantine are not primary control measures	Rare or context-specific transmission cannot be excluded without surveillance	Maintain standard clinical precautions and monitor unusual clusters
Clinical spectrum	Ranges from asymptomatic exposure and febrile illness to fulminant encephalitis	Acute febrile illness surveillance may improve early detection	Frequency and predictors of neurological progression are unknown	Establish prospective cohorts across the full disease spectrum
Severe disease	Fever may progress rapidly	Immediate referral and	Optimal neurocritical-care protocol is not	Develop standardized case-

	to seizures, coma, cerebral oedema and shock	paediatric intensive supportive care are critical	established	management and referral algorithms
Diagnosis	Early RT-PCR, IgM testing and reference-laboratory confirmation are available	Specimens should be collected promptly and tested within an AES panel	Short viraemia and transport delays reduce sensitivity	Strengthen regional laboratories and sample-transport systems
Treatment	No proven antiviral or licensed vaccine	Management is supportive, including airway, seizure, shock and intracranial-pressure care	Candidate antiviral evidence is largely preclinical	Conduct therapeutic screening, animal studies and adaptive clinical trials
Vector control	Indoor residual spraying, insecticides, housing improvement and personal protection are recommended	Integrated measures may reduce exposure	CHPV-specific effectiveness data are sparse	Embed intervention evaluation within outbreak response
Surveillance	Existing AES and IDSP platforms can detect clusters	CHPV can be integrated into established reporting systems	Surveillance focused only on encephalitis misses mild infections	Link AES, acute febrile illness, vector and serological surveillance
Long-term outcomes	Neurological sequelae occur in some survivors	Follow-up should extend beyond discharge	Prospective neurodevelopmental data are scarce	Create survivor registries and standardized outcome assessments
Risk communication	Early neurological warning signs require urgent referral	Community awareness may reduce treatment delay	Sensational reporting may cause fear and misinformation	Use clear, non-stigmatizing, locally adapted communication

Treatment and Case Management

No antiviral agent has demonstrated clinical efficacy against CHPV, and no licensed vaccine is available. Management is therefore supportive and time critical.

Children with fever and neurological warning signs require immediate assessment of airway, breathing and circulation. Hypoxaemia, shock and hypoglycaemia should be corrected promptly. Seizures require rapid treatment according to paediatric protocols. Fluid therapy must balance restoration of circulation against the risk of worsening cerebral oedema.

Raised intracranial pressure should be suspected in children with deteriorating consciousness, abnormal pupils, repeated vomiting, hypertension with bradycardia or other neurological signs. Management may include head elevation, airway control, ventilation, osmotherapy and neurocritical monitoring according to available resources and clinical indications. Routine use of interventions without physiological assessment should be avoided.

Empirical treatment for bacterial meningitis, herpes encephalitis, malaria or other treatable conditions may be appropriate until diagnostic evidence permits de-escalation. There is no established role for corticosteroids specifically for CHPV encephalitis, and their use should be based on a defined alternative indication or research protocol.

Outcome probably depends strongly on time to recognition and access to intensive care. Rural families may first seek local or informal treatment because early symptoms resemble common febrile illness. Public-health messaging should therefore focus on urgent referral for fever accompanied by persistent vomiting, abnormal drowsiness, confusion, seizures or loss of consciousness.

Prevention and Outbreak Control

In the absence of a vaccine, prevention centres on vector control, housing improvement, personal protection and rapid surveillance. Measures commonly recommended include insecticide spraying in affected villages, filling cracks in mud walls, improving sanitation around houses and animal shelters, using bed nets or screens and reducing resting habitats for sandflies.

These recommendations are biologically plausible, but CHPV-specific intervention trials are lacking. Conventional mosquito-control approaches may be insufficient because sandflies are smaller, have different resting behaviour and may breed in organic-rich microhabitats rather than obvious standing water.

Vector control should therefore be guided by local entomological investigation. Indoor residual spraying may be appropriate where vectors rest on treated surfaces, while environmental management should address wall cracks, animal shelters, waste and damp organic matter. Insecticide susceptibility should be monitored rather than assumed.

Active case finding should cover both AES and acute febrile illness in affected districts. Rapid reporting of fever clusters among children can trigger sample collection before neurological deterioration. Peripheral clinicians and community health workers require simple case definitions and clear referral pathways.

A One Health approach is justified because the natural maintenance cycle remains unresolved. Human case surveillance should be linked with vector collection, animal serology, ecological assessment and viral sequencing. However, animal culling or restrictions should not be introduced without evidence that a species functions as a reservoir or source of infection.

Public Health Significance

CHPV illustrates the vulnerability created when a rare but rapidly fatal disease occurs in rural children and requires advanced critical care. Although total annual case numbers are smaller than those of dengue or Japanese encephalitis, the speed of progression and high mortality among recognized encephalitis cases give it disproportionate public-health importance.

The disease also exposes weaknesses in AES surveillance. AES is a syndromic category rather than a single diagnosis. If laboratory capacity is limited, CHPV may be missed or attributed to "unknown viral encephalitis." Conversely, during a publicized outbreak, every childhood encephalitis death may be inaccurately labelled Chandipura.

CHPV is also an equity issue. Many outbreaks have affected rural, tribal or socioeconomically disadvantaged communities living in housing that facilitates vector exposure and located far from paediatric intensive care. Prevention cannot rely solely on advising individual families to avoid bites. Housing, sanitation, transport, laboratory access and emergency referral are structural components of control.

Recent Advances

The 2024–2025 investigations broadened understanding of CHPV in several ways. Identification of IgM-positive non-neurological febrile cases suggests that surveillance restricted to encephalitis substantially underestimates infection.[3] High neutralizing-antibody prevalence among sampled populations supports the possibility of widespread exposure and partial population immunity.

Whole-genome sequencing has improved the ability to compare viruses across outbreaks and identify whether large epidemics reflect new lineages or re-emergence of established strains. Genomic surveillance must nevertheless be integrated with clinical severity, vector and exposure data; genetic differences alone do not prove altered virulence.

Laboratory research has identified viral proteins and host pathways that may serve as therapeutic or vaccine targets. Candidate approaches include neutralizing antibodies, glycoprotein-based vaccines, small-molecule inhibitors and host-directed strategies. Most remain at in-vitro or animal-study stages.

The current national investigation has also expanded the surveillance framework to include acute febrile illness, serosurveys, animal testing and multiple arthropod groups. This integrated design may help resolve long-standing uncertainties, provided results are published transparently regardless of whether a reservoir or alternative vector is identified.

Challenges and Limitations

The evidence base remains dominated by outbreak reports, small case series and laboratory studies. Case definitions, diagnostic timing and denominator populations vary, making it difficult to compare fatality rates or estimate infection prevalence.

The natural transmission cycle remains the greatest ecological uncertainty. Sandflies are strongly implicated, but negative vector testing during an outbreak is common because infected insects may be rare, transient or collected after transmission peaks. Conversely, experimental competence in a mosquito or tick does not prove an epidemiologically important field role.

Another controversy concerns disease mechanism. The terms encephalitis, encephalopathy and epidemic brain attack have all been used. Direct neuroinvasion is supported by virological evidence, but vascular injury, systemic inflammation and cerebral oedema may contribute. Clinically, the distinction should not delay emergency neurocritical care.

Climate associations are plausible but underdeveloped. Retrospective correlation between outbreaks and temperature or rainfall cannot establish causation. Longitudinal vector, meteorological and human surveillance is needed.

Finally, media reports often combine suspected AES deaths with confirmed CHPV deaths. Scientific reporting should preserve separate denominators for suspected AES, laboratory-confirmed infection, encephalitic disease and all detected infections.

Future Directions

India should establish year-round sentinel surveillance for CHPV in historically affected and ecologically suitable districts. Surveillance should include children with acute febrile illness as well as AES during high-risk seasons.

Regional laboratories need validated RT-PCR and serological capacity, external quality assurance and rapid specimen transport. Sequencing should be performed on representative samples rather than only unusual or fatal cases.

Prospective clinical studies should define the incubation period, progression from fever to encephalitis, predictors of severe disease, optimal intracranial-pressure management and long-term neurological outcomes. Standardized data collection would permit pooling across outbreaks.

Entomological research should identify vector species, seasonal abundance, infection rates, resting sites and insecticide susceptibility. Animal studies should assess reservoir competence rather than relying only on antibody detection.

Vaccine development is scientifically justified because severe disease affects children and progresses too rapidly for delayed therapy. Candidate vaccines require robust animal models, safety studies and strategies for use in geographically focal outbreaks.

Finally, outbreak preparedness should include paediatric referral mapping, ambulance readiness, critical-care protocols and community communication before the monsoon. Waiting until encephalitis deaths are reported forfeits the limited window for prevention and early treatment.

CONCLUSION

Chandipura virus is a rare but formidable cause of rapidly progressive paediatric encephalitis in India. Its public-health importance arises from its high mortality among recognized severe cases, short interval between fever and neurological collapse, uncertain ecological cycle and concentration in underserved communities.

Evidence strongly implicates sandflies, but the specific vector and vertebrate reservoir responsible for maintaining transmission remain unresolved. Recent findings indicate that infection can also cause uncomplicated febrile illness and that asymptomatic exposure may be common.

There is no proven antiviral treatment or licensed vaccine. Survival therefore depends on early recognition, rapid transport and meticulous supportive neurocritical care. Prevention currently

relies on integrated vector management and housing and environmental measures, although their CHPV-specific effectiveness requires evaluation.

The large 2024 outbreak and renewed 2026 investigations demonstrate that CHPV cannot remain an episodically remembered disease. India needs continuous surveillance, decentralized diagnosis, multidisciplinary ecological research and planned paediatric emergency response.

Future success should be measured not only by controlling visible outbreaks but by detecting transmission earlier, reducing delays in treatment, preventing deaths and resolving the biological uncertainties that have persisted since the virus was first identified more than six decades ago.

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