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Chandipura virus disease: an ongoing fatal acute encephalitis syndrome

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Abstract

The current ongoing outbreak of Chandipura virus in the state of Gujarat has shown its epidemic potential, leading to the death of 22 children, and deployment of the National Joint Outbreak Response Team to Gujarat and Rajasthan by the Indian Home Ministry. The virus is known to be acutely fatal following episodes of encephalitis syndrome, primarily in pediatric patients (aged 2.5-15 years) running a shorter incubation period of 24-48 h. The infection is vector borne; primarily mediated by bite of infected sandfly i.e., *Phlebotomus spp.* Despite, high case fatality rate ranging from 45-75%, and epidemic potential, the disease remains largely under managed due to the absence of effective treatment and vaccines. Therefore vector control strategies are the central paradigm to contain and prevent the episodes of outbreaks.

Introduction

The Chandipura virus (CHPV), a (-) ssRNA virus, belonging to genus Vesiculovirus, family *Rhabdoviridae* was first isolated in 1965 from two adult patients suffering from a febrile illness in Chandipura village (Nagpur region) in India during chikungunya and dengue viruses outbreak. The virus was therefore named after its first place of isolation. Despite a much longer history of discovery, it gained recognition for epidemic potential only after the acute encaphiltis outbreak occurred in Andhra Pradesh, 2003 where around 51% of clinical samples were found to be positive with a resulting case fatality rate (CFR) of 52.3%. Since then, CHPV has become one of the major etiology of encephalitis syndrome in pediatric population (2.5 years to 15 years) with a high CFR. Similar epidemics have been reported from Gujarat in 2004 (CFR 75%), Odisha in 2015, Nagpur (43.6%), Gujarat and Rajasthan (2024) and Gujarat (2026). The

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disease primarily affects children (<15 years) and runs an acute course with symptoms rapidly progressing to coma and death 48 h post infection (PI).

Transmission

CHPV is vector (sand fly) borne virus, with sandfly belonging to genus *Phelobotomus* being incriminated as the major vector. The transmission of CHPV occurs after a bite of infected sandflies. The virus has also been isolated from other sandflies like *Sergentomyia* spp. Previous experimental studies on *Phlebotomus papatasi* have revealed transmission of virus via vertical, venereal and horizontal routes, thus attributing to the potential of *P. papatasi* in maintaining the virus naturally during non-epidemic periods, followed by an upsurge in clinical cases upon favourable conditions for proliferation of sandfly. Apart from sandfly, A*edes aegypti* (the major vector of Yellow fever), has also been experimentally implicated in CPHV transmission. Fortunately, no human to human transmission has been reported till date.

Pathogenesis

While the exact mechanism of entry of CPHV into the neuron and subsequent death is relatively unknown, the virus gains entry into the central nervous system via retrograde (ascending) movement from peripheral nerves or olfactory nerves. Further, hematogenous spread via destruction of blood brain barrier (BBB) and/or endothelial cells is mediated by cytokines and chemokines produced as a result of invasion of BBB. Subsequently initiation of neuronal death is mediated by mounting of immune response via production of stress factors, proinflammatory cytokines, nitrogen species, and the release of reactive oxygen species (ROS).

Clinical symptoms

The symptoms arrive shortly after the infection, *i.e.*, following an incubation period of 24-48 h, characterised by high fever, altered sensorium (mental state), and seizures. The manifestations and illness are, however, of major concern in pediatric patients falling into the age group 2.5 years to 15 years of age, as evident in reported outbreaks. The symptoms progress within 24-48 h into acute encephalitis syndrome (brain swelling), characterised by seizures, ultimately culminating in coma and death if left untreated. Apart from the CNS symptoms, other non-specific clinical manifestations as recorded from previous outbreaks include vomiting, diarrhoea, cough, headache, etc.

Outbreaks in India

Although CPHV was discovered in 1965, the disease remained neglected until the 2003 Andhra Pradesh outbreak, where around 11 districts of the state encountered the outbreak with a CFR of 56%. Since, then multiple outbreaks have occurred throughout Indian states, including Andhra Pradesh (2003–2005, 2007-2008), Gujarat (2005, 2009–2012), Vidarbha

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region of Maharashtra (2007, 2009–2012), Bihar (2018), Gujarat and Rajasthan (2024; largest outbreak in two decades; CFR 46%, elaborated across 43 districts), and currently ongoing outbreak in Gujarat and Rajasthan (2026).

Diagnosis

The sudden onset of acute encephalitis syndrome (AES), high CFR (45-75%), and CNS manifestations, aid in diagnosis, however serology alone cannot be used to accurately diagnose CPHV. Therefore, diagnosis is done based on its epidemiology and clinical manifestations followed by confirmation using molecular tools *viz.*, Reverse transcriptase PCR (RT PCR), and Immunoflourescence assay (IFA). Among the molecular tools, RT PCR targeting N gene, demonstrates 100% specificity and sensitivity as low as 10 pfu/ml. Immunofluorescence Assay (IFA) is employed to detect both antibodies and antigens specifically providing distinct identification features for the virus under investigation. Further, animal cell culture based virus isolation from humans and sandflies can be attempted using cell lines like, Vero E6 cells, BS-C-1, Rhabdomyosarcoma, and chicken embryo etc. Furthermore, laboratory animal models like Swiss albino mice are experimentally explored for studies on viral pathogenesis, where experimental studies reported mice mortality upon intracranial, intraperitoneal, subcutaneous, intradermal, nasal and oral routes of administration of CPHV. However, histopathological findings are marked by perivascular cuffing (brain), mild congestion and collapsed areas in lungs, focal degenerative changes in liver, and increased intracellular spaces in heart.

Management of CPHV

Currently, there exists no specific treatment for CPHV, however clinical cases are managed based on their symptoms inclusive of fluid therapy, anticonvulsants, and anti-pyretics. Further, anti-viral drugs like ribavirin, favipiravir (T705), vesiculopoins (novel antiviral compound) have been found to suppress CPHV replication *in vitro*. The public health impact of CPHV infections are further aggravated due to absence of licensed vaccines and treatment. However, few potential vaccine candidates are being explored in labs currently.

Prevention and Control Strategy

The absence of effective treatment and vaccines against CPHV direct the prevention and control strategy towards management and control of the involved vector i.e., *Phlebotomus.* The female sandflies survive under warm, humid conditions especially in monsoons, reside and breed in cracks, crevices in walls, decaying animal excreta, and stagnant water in and around animal shelters. Therefore anti-adult sandfly measures like pre-monsoon application of insecticides, indoor residual spraying (IRS), and fogging prove effective in fly control and consequently dampens the chances of outbreaks. Additionally, mapping the sandfly prone areas

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along with geographic information system (GIS) and remote sensing can help in predicting hotspots for future associated outbreaks.

Conclusion

Chandipura virus is one of the etiology of acute encephalitis syndrome (AES), with a short incubation period of 24-48 h and results in case fatality rate ranging from 45-75%. The infections significantly infect pediatric patients of 2.5-15 years of age, culminating into a fatal encephalitis associated death. The infections are primarily mediated by bite of infected sandfly falling under the genus; *Phlebotomus* The clinical cases are managed based on the symptoms manifested by the patients mainly including fluid management, anti-pyretics and anti-convulsants, however antiviral compounds like ribavirin has demonstrated anti-viral acivity in vitro. The absence of effective treatment and vaccine, and relatively high CFR in young patients underscore the need for development of effective treatment and vaccine candidates.

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