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Chandipura virus infection in domestic and laboratory Animals: Vector-host dynamics, pathogenesis, and one health implications

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Abstract

Chandipura virus (CHPV), an emerging neurotropic rhabdovirus, presents a growing public health threat due to its high case-fatality rates in paediatric populations. While human outbreaks are well-documented, the role of livestock, companion animals, and wildlife in the sylvatic and domestic transmission cycles remains poorly defined. This review compiles current knowledge on CHPV infection in animals, emphasizing host susceptibility, seroprevalence trends, and neuropathogenesis in laboratory models. Sero-surveillance reveals widespread subclinical exposure among domestic livestock (cattle, buffaloes, sheep, goats, and pigs), hinting at their potential role as amplifying or maintenance hosts. Conversely, neonatal and juvenile murine models uniquely replicate the severe, age-dependent encephalitic syndrome seen in humans, serving as crucial systems for studying blood-brain barrier breach, microglial activation, and toll-like receptor 4 (TLR4)-mediated neuroinflammation. Integrating veterinary surveillance, entomological tracking, and human clinical diagnostics through a One Health approach is critical for mapping viral persistence, identifying natural reservoirs, and preventing future cross-species spillover events.

Keywords: Chandipura virus, encephalitis, one health, vector-borne disease, pathogenesis, animal models

1. Introduction

Chandipura virus (CHPV) is an arthropod-borne virus belonging to the genus *Vesiculovirus* within the family *Rhabdoviridae* (Mayo, 2002; Walker *et al.*, 2018) ^[13, 29]. First isolated in 1965 from human patients during a febrile outbreak in Chandipur, Maharashtra, India (Bhatt & Rodrigues, 1967) ^[3], the virus has transitioned from an obscure tropical pathogen into a major driver of explosive acute encephalitis syndrome (AES) outbreaks (Rao *et al.*, 2004) ^[18]. Although clinical research has long prioritized human pathology, the cross-species transmission risk and hidden veterinary footprint of CHPV demand closer scrutiny.

In nature, the virus cycles between haematophagous arthropods and vertebrate hosts. While humans are considered incidental, dead-end hosts who do not pass the virus to others, a variety of domestic livestock and wild vertebrates show strong evidence of exposure (Joshi & Rodrigues, 1987) ^[9]. This suggests complex sylvatic and synanthropic cycles that maintain the virus during inter-epidemic periods. Understanding how CHPV operates in animals is vital for mapping its geographic spread and identifying potential natural reservoirs. Furthermore, experimental animal models have become indispensable tools for cracking open the molecular secrets of CHPV neuroinvasion and testing novel therapies (Lad & Gupta, 1992) ^[11]. This review provides a comprehensive look at CHPV in both domestic and experimental animals, framing it as a critical priority for global One Health surveillance.

2. Virology and Genomic Architecture

CHPV is an enveloped, bullet-shaped virion measuring roughly 180 nm in length and 75 nm in width. Its core holds a linear, single-stranded, negative-sense RNA genome of approximately 11 kilobases (kb) (Arankalle *et al.*, 2005) ^[1]. The genome contains a 49-nucleotide leader

region at the 3' end, followed by five highly conserved structural genes (Marriott & Easton, 1999) ^[12]:

- **Nucleoprotein (N):** Encapsidates the viral RNA genome, shielding it from host nuclease degradation and serving as the primary structural template for replication.
- **Phosphoprotein (P):** Acts as an essential cofactor for the viral polymerase, playing a dual role in regulating viral transcription and blunting the host's innate antiviral interferon responses.
- **Matrix Protein (M):** Drives virion assembly and budding at the host cell membrane. It is highly cytotoxic and triggers apoptosis while shutting down host cellular transcription to favor viral replication (Basak *et al.*, 2007) ^[2].
- **Glycoprotein (G):** Forms spikes on the viral envelope that bind to host surface receptors (such as low-density lipoprotein receptors), dictating cell tropism and facilitating fusion (Ghosh *et al.*, 2016) ^[6]. It is also the primary target for neutralizing antibodies.
- **Large Protein (L):** Functions as the multi-domain viral RNA-dependent RNA polymerase (RdRp), managing viral transcription, replication, capping, and polyadenylation.

The high mutation rate of the RdRp enzyme allows CHPV to adapt rapidly to different insect vectors and mammalian hosts, raising the risk of altered virulence and cross-species spillover.

3. Vectors and Transmission Dynamics

The survival and spread of CHPV rest on a complex network of blood-feeding vectors and susceptible animal hosts.

3.1 Primary and Secondary Vectors

Phlebotomine sandflies, particularly *Phlebotomus papatasi* and *Phlebotomus argentipes*, serve as the primary vectors for CHPV (Geevarghese & Mishra, 2011) ^[4]. These insects thrive in warm, humid climates, making the monsoon and post-monsoon seasons (May to September) prime windows for viral transmission. The virus multiplies within the sandfly's salivary glands and is injected into mammalian hosts during blood meals. Crucially, *P. papatasi* can transmit the virus both venereally and transovarially (from mother to offspring), allowing CHPV to persist across generations even when susceptible vertebrate hosts are scarce.

Secondary vectors, including sandflies of the *Sergentomyia* genus (Gokhale & Mishra, 2009) ^[7], *Aedes aegypti* mosquitoes (Sudeep & Gokhale, 2014) ^[27], and certain ixodid ticks, have also yielded wild viral isolates or shown vector competency in lab settings. This diverse vector profile highlights the virus's high ecological flexibility.

3.2 Domestic and Sylvatic Cycles

The domestic transmission cycle is closely tied to rural farming environments. Mud-plastered houses, thatched roofs, and livestock sheds provide ideal breeding grounds for sandflies, which find shelter and organic nutrients in accumulated cow dung and leaf litter. Here, vectors feed interchangeably on humans and domestic animals.

In contrast, the sylvatic cycle operates in wilder landscapes, involving wild rodents, bats, and non-human primates. These wild populations are thought to harbor the virus quietly, feeding small-scale spillover events into domestic zones when environmental conditions line up

4. Susceptibility and Seroprevalence in Domestic Livestock

Unlike humans, adult domestic livestock naturally exposed to CHPV do not typically develop overt neurological disease. Instead, they undergo subclinical infections characterized by brief, low-level viremia followed by robust seroconversion.

Sero-surveillance programs in endemic regions have uncovered high rates of neutralizing and neutralizing-like antibodies against CHPV in several domestic species:

- **Cattle and Buffaloes:** Frequently show high seroprevalence rates in agricultural zones, reflecting heavy exposure to sandfly populations in livestock shelters (Joshi & Rodrigues, 1987) ^[9].
- **Pigs:** Exhibit significant seropositivity, making them important sentinels for tracking viral circulation near human dwellings (Shrivastava & Arankalle, 2011) ^[24].
- **Goats and Sheep:** Regularly show anti-CHPV antibodies, confirming that small ruminants participate actively in the peri-domestic viral cycle (Somani & Mishra, 2015) ^[26].

The high rate of subclinical seropositivity in livestock suggests these animals may act as temporary amplifying or maintenance hosts. While their short-lived viremia may not always be enough to spark massive human outbreaks, their presence keeps vector populations fed and infected, stabilizing the virus within rural ecosystems.

5. Experimental Pathology and Neuroinvasion in Laboratory Models

Because natural infections in adult livestock are quiet and subclinical, researchers rely heavily on laboratory animal models to study the aggressive neurotropic nature of CHPV.

5.1 Murine Models and Age-Dependent Resistance

Mice are the gold-standard model for CHPV research, showcasing a stark, age-dependent susceptibility that mirrors human patterns (Lad & Gupta, 1992) ^[11]:

- **Neonatal and Juvenile Mice (<14 Days Old):** Highly susceptible to peripheral inoculation (intraperitoneal or subcutaneous). They quickly develop severe clinical signs, including hyperexcitability, ataxia, tremors, generalized convulsions, quadriplegia, and death within 48 to 96 hours (Ghosh & Basu, 2009) ^[5].
- **Adult Mice (>21 Days Old):** Resistant to peripheral infection due to a fully matured blood-brain barrier (BBB) and an advanced innate immune system. However, they remain vulnerable to direct intracranial injection, which triggers fatal encephalitis.

5.2 Mechanisms of Neuro-invasion

CHPV uses two primary routes to breach the central nervous system (CNS):

1. **Hematogenous Route:** Following local replication in the dermis and regional lymph nodes, the virus enters the bloodstream, causing primary viremia. High viral loads can compromise the BBB by directly infecting brain capillary endothelial cells or altering tight-junction proteins (Paquet & Rout, 2012) ^[16]. Alternatively, the virus can slip across using a "Trojan Horse" strategy inside infected monocytes or macrophages (Samuel & Diamond, 2006) ^[21].
2. **Retrograde Axonal Transport:** The virus can bypass the BBB entirely by invading peripheral or olfactory

nerve endings at the bite site, traveling backward along axons directly into the spinal cord and brain (Ghosh & Basu, 2009) ^[5].

5.3 Histopathology and Cellular Stress

Once inside the CNS, CHPV selectively targets and destroys neurons, leaving astrocytes and oligodendrocytes mostly intact (Ghosh & Basu, 2009) ^[5]. Classical histopathological findings in infected animal brains include severe cerebral and cerebellar edema, perivascular lymphocytic cuffing, and microglial nodules. Widespread neuronal degeneration, central chromatolysis, and ischemic necrosis are prominently observed within the cerebral cortex, thalamus, and Purkinje cell layer of the cerebellum.

At the molecular level, viral replication inside neurons triggers intense cellular stress, causing a massive release of reactive oxygen species (ROS) and nitric oxide (NO) (Verma & Mishra, 2016) ^[28]. This oxidative stress disrupts mitochondrial function, activating death-domain pathways and driving rapid neuronal apoptosis.

5.4 The Hyperinflammatory Cascade

The severe tissue damage seen in CHPV encephalitis is driven as much by the host's immune response as by the virus itself. Animal studies show that CHPV activates Toll-Like Receptor 4 (TLR4) on microglia and astrocytes (Rajasekharan & Chandy, 2014) ^[17]. This triggers a massive, uncoordinated surge of pro-inflammatory cytokines and chemokines, including Tumor Necrosis Factor-alpha (TNF- α), Interleukin-1 beta (IL-1 β), Interleukin-6 (IL-6), and Monocyte Chemoattractant Protein-1 (MCP-1) (Saxena & Kumar, 2013) ^[22].

While intended to fight the infection, this intense cytokine storm damages the BBB further, draws in destructive inflammatory cells, and accelerates widespread bystander neuronal death.

6. Diagnosis of CHPV Infection in Animals

Accurate veterinary diagnosis is essential for effective surveillance and outbreak prediction.

6.1 Molecular Assays

Reverse Transcription Polymerase Chain Reaction (RT-PCR) and real-time quantitative RT-PCR (qRT-PCR) are the gold standards for detecting CHPV during early infection. These assays target highly conserved regions of the viral N or P genes, offering exceptional sensitivity and specificity (Gunjikar & Arankalle, 2010) ^[8]. They can identify viral RNA in blood, cerebrospinal fluid (CSF), and tissue homogenates (brain, spleen) during the brief viremic phase.

6.2 Serological Testing

Because viremia passes quickly, serological tests are invaluable for broader surveillance:

- **Enzyme-Linked Immunosorbent Assay (ELISA):** Capture ELISAs detect anti-CHPV IgM antibodies, indicating recent exposure (Shrivastava & Joshi, 2013) ^[23]. Indirect ELISAs track IgG antibodies to assess historical exposure and map long-term herd immunity.
- **Serum Neutralization Test (SNT):** The definitive standard for confirming specific exposure, SNT measures the ability of animal sera to neutralize live virus in cell culture (Rowson & Mahy, 1985) ^[19], eliminating cross-reactive false positives from related vesiculoviruses.

6.3 Virus Isolation and Immunohistochemistry

- Plating samples on susceptible cell lines (such as Vero, BHK-21, or MDCK) allows researchers to isolate the live virus and observe its rapid, destructive cytopathic effects (CPE). In post-mortem studies, immunohistochemistry (IHC) using specific monoclonal antibodies can pinpoint viral antigens within brain lesions, confirming a definitive diagnosis (Ghosh & Basu, 2009) ^[5].

7. Prevention, Control, and Therapeutics

There are currently no approved vaccines or targeted antiviral drugs for CHPV in either human or veterinary medicine. Management relies on integrated vector control and biosecurity.

7.1 Environmental and Chemical Vector Management

Controlling sandfly populations is the most effective way to break the transmission cycle (Mourya & Yadav, 2015) ^[15]:

- Regular application of indoor residual sprays (IRS) containing pyrethroids or organophosphates in human homes and animal shelters.
- Treating livestock with topical insecticide pour-ons or insecticide-impregnated ear tags to lower bite rates.
- Modifying environments around farms by clearing organic debris, managing manure properly, and sealing wall cracks to destroy sandfly breeding sites.

7.2 Preclinical Insights from Animal Models

Experimental animal models are driving the search for future treatments. Preclinical trials in mice have highlighted several promising avenues:

- **Antiviral Drugs:** Broad-spectrum antivirals like favipiravir have shown success in curbing viral replication and boosting survival rates in lethal mouse challenges (Kumar & Singh, 2018) ^[10].
- **Neuroprotective Agents:** Minocycline, an antibiotic with potent anti-inflammatory properties, has been shown to reduce microglial activation, lower cytokine surges, and ease brain edema in infected mice (Mishra & Basu, 2012) ^[14].
- **Vaccine Candidates:** Experimental designs—including inactivated whole-virus vaccines (Shukla & Arankalle, 2012) ^[25], recombinant vesicular stomatitis virus (rVSV) platforms displaying the CHPV-G protein (Roy & Chattopadhyay, 2016) ^[20], and DNA-based formulations—have triggered strong neutralizing antibody responses and fully protected animal models from lethal exposure.

8. Conclusion and Future Directions under a One Health Paradigm

Chandipura virus can no longer be viewed strictly as a human medical emergency. Its intricate reliance on sandfly vectors, combined with widespread, quiet exposure across domestic livestock and wild animals, positions it as a classic One Health challenge (World Health Organization, 2023) ^[30]. Focusing only on human clinical cases leaves public health frameworks reactive, missing the broader ecological shifts that drive outbreaks.

To get ahead of the virus, future efforts must focus on

1. **Systematic Veterinary Surveillance:** Establishing routine serological screening of livestock in high-risk zones to map viral activity during inter-epidemic periods.

2. **Ecological and Climate Modeling:** Studying how shifting weather patterns, deforestation, and changing agricultural practices alter sandfly habitats and densities.
3. **Refining Translational Models:** Developing next-generation animal models to better understand how the virus persists in asymptomatic hosts and crosses the species barrier.

By uniting human medical experts, veterinary scientists, and entomologists under a shared One Health framework, we can build robust early-warning systems, develop protective vaccines, and ultimately defuse the threat of CHPV before it triggers the next major epidemic.

Conflict of Interest

Not available

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