

Editorial**Acute Encephalitis Syndrome: The Lethal Hazard of Chandipura Virus****Kamran Ahmad****Department of Pharmacy, Nibha Institute of Pharmaceutical Sciences, Rajgir, India.***ARTICLE INFO**

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Editorial

Acute Encephalitis Syndrome (AES) is a critical and frequently fatal syndrome marked by the swift development of cerebral inflammation, resulting in symptoms including fever, disorientation, seizures, and, in severe instances, irreversible neurological impairment or mortality. The Chandipura virus (CHPV), a lesser known yet perilous infection, has surfaced as a notable cause of AES in specific regions of India, despite many viruses being recognised as responsible for the condition. This virus, chiefly spread by mosquitoes, poses a multifaceted and developing challenge to public health, with its severe repercussions frequently intensified by delayed diagnosis and treatment.

The Emerging Threat of Chandipura Virus

The Chandipura virus, member of the Rhabdoviridae family, is a mosquito-borne pathogen that has caused multiple outbreaks of Acute Encephalitis Syndrome (AES), especially in the Indian states of Maharashtra, Madhya Pradesh, and Andhra Pradesh. Initially identified in 1965 in India, the virus has attracted interest due to its correlation with lethal encephalitis outbreaks in both rural and urban demographics.

The Chandipura virus is transmitted via the bites of infected mosquitoes, primarily by *Phlebotomus* species. The virus primarily impacts children, particularly those under 10 years old, who are at the greatest risk of suffering severe encephalitis. The incubation period for CHPV is brief, with symptoms generally appearing within 4 to 8 days following an infected mosquito bite.

Pathophysiology of Acute Encephalitis Syndrome

The Pathophysiology of Acute Encephalitis Syndrome As a result of the Chandipura Virus

Upon entering the body, the virus disseminates via the bloodstream to the brain, inducing inflammation in the encephalitic regions. The characteristic of AES induced by the Chandipura virus is fast advancement, with individuals frequently experiencing decline within hours of symptom beginning. The clinical manifestations may commence with an abrupt high temperature, headache, and vomiting, rapidly progressing to disorientation, agitation, seizures, and loss of consciousness. In extreme instances, the infection may result in irreversible cognitive and physical impairments, along with mortality.

The virus predominantly affects the central nervous system, resulting in considerable neurological impairment. In contrast to other types of encephalitis, which involve immune-mediated brain inflammation due to infection, Chandipura virus infection directly impairs neuronal function, resulting in the syndrome's accompanying symptoms.

Challenges in Diagnosis and Treatment

A significant obstacle in addressing AES caused by the Chandipura virus is the difficulty of early detection. The clinical manifestations of AES coincide with those of other viral and bacterial encephalitic diseases, complicating the differentiation of aetiologies. Moreover, the diagnostic infrastructure in rural and distant regions, where Chandipura virus outbreaks frequently arise, is typically inadequate, resulting in delays in acquiring essential laboratory confirmation. Detection of the Chandipura virus by laboratory tests

necessitates specialised equipment, including PCR (Polymerase Chain Reaction) testing, which may not be readily accessible in impacted regions. Consequently, numerous instances remain misdiagnosed until the illness has advanced to a critical stage, hampering treatment initiatives. Management of AES induced by Chandipura virus mostly consists on supportive care. No specific antiviral medication exists for CHPV; the main strategy focusses on symptom management—regulating fever, alleviating cerebral oedema, and averting consequences such as seizures. In critical instances, patients may necessitate mechanical ventilation in the event of respiratory failure. The absence of a conclusive treatment or vaccine for the virus renders early discovery and supportive care crucial for enhancing survival and recovery prospects.

Societal Implications and Necessity for Intervention

The incidence of Chandipura virus-induced AES has considerable socio-economic and emotional impacts on families and communities. The virus predominantly impacts children, resulting in significant repercussions for families, including enduring emotional trauma and the possibility of lasting disability in survivors. Communities in endemic regions experience persistent worry and anxiety due to the unpredictable nature of outbreaks, which jeopardises the welfare of their youth. Moreover, the financial strain is substantial. Families may incur substantial medical expenses resulting from hospitalisation, specialised treatment, and rehabilitation for survivors. The demand for a proficient workforce to attend to individuals with chronic neurological impairments further exacerbates the load on already overwhelmed healthcare facilities. Resolving this public health concern necessitates coordinated efforts from governments, healthcare practitioners, and researchers. Public health initiatives targeting vector control—such as eradicating mosquito breeding sites, advocating for insect repellent usage, and distributing mosquito nets—are crucial for preventing the transmission of the Chandipura virus. Moreover, enhancing awareness of the signs and symptoms of AES

in rural populations can facilitate early detection and augment survival rates.

Advancing: Investigation and Mitigation

Although initiatives to address AES induced by the Chandipura virus are in progress, further research is critically required to produce vaccines and antiviral therapies. The scientific community must prioritise the advancement of diagnostic instruments and therapeutic strategies specific to this virus, while also investigating innovative approaches to address mosquito-borne diseases broadly. Furthermore, healthcare infrastructure in high-risk areas must be fortified. Investing in medical facilities, enhancing quick diagnostic capabilities, and training healthcare personnel to identify symptoms of Chandipura virus-induced AES could substantially improve outbreak management. Cooperation among federal health agencies, the World Health Organisation, and local public health organisations is essential for fostering resilience to the infection.

Conclusion

Acute Encephalitis Syndrome induced by the Chandipura virus poses a covert yet significant risk to public health, especially in certain regions of India. The swift emergence, severe neurological consequences, and absence of targeted therapies provide considerable obstacles for healthcare systems, particularly in rural areas where the virus is most widespread. Nonetheless, by concentrated initiatives in prevention, early identification, and research on vaccines and antiviral treatments, it is feasible to alleviate the effects of this lethal virus. The imperative of resolving this issue is paramount, since each outbreak advances our efforts to mitigate the destruction wrought by this elusive and formidable infection.

References

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