INTERNATIONAL MULTI DISCIPLINARY JOURNAL FOR RESEARCH & DEVELOPMENT
eISSN 2394-6334 Impact factor: 8.854 Volume 13, issue 04 (2026)

NIPAH VIRUS INFECTION: A REVIEW

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Abstract: This review presents a comprehensive analysis of the Nipah virus (NiV), encompassing its structure, replication, epidemiology, and recent advances in therapeutics and vaccines. The significance of this work lies in the timely synthesis of emerging research, which offers valuable insights for scientists, policymakers, and healthcare professionals. It serves as a critical resource to guide future research directions, enhance outbreak preparedness, and accelerate the development of effective medical countermeasures against NiV.

Keywords: Nipah virus (NiV), zoonotic disease, paramyxovirus, viral transmission, preventive measures, vaccine

Introduction

The emergence of novel infectious diseases poses a serious threat to global public health [1, 2]. The Nipah virus (NiV) is a highly lethal and contagious pathogen that causes severe epidemics with high morbidity and mortality rates. The initial cases of Nipah virus infection were recorded in 1998 during an outbreak of neurological and respiratory illnesses on pig farms in Malaysia. This epidemic affected 265 individuals and resulted in 108 deaths [3]. Subsequently, Nipah virus was documented in Bangladesh during the winters of 2001, 2003, and 2004 [4, 5]. It is believed that its natural reservoir consists of bats of the genus *Pteropus* [6]. Infections with Nipah virus have been documented among bats in Malaysia, Bangladesh, and Cambodia [7].

The clinical manifestations of Nipah virus infection are highly variable, ranging from subclinical forms to severe respiratory disease and fatal encephalitis [8]. Nonspecific symptoms such as fever, headache, and myalgia often progress to severe complications, including seizures, coma, and encephalitis, which carry a high risk of death. Although Nipah virus has the potential to cause large-scale epidemics, no licensed vaccine or targeted antiviral drugs are currently available [8].

According to World Health Organization data, in January 2026, two cases of Nipah virus infection were reported in the People's Republic of Bangladesh (one of which was fatal), and two cases (among healthcare workers) were reported in India.

This study examines the epidemiology and transmission dynamics of the Nipah virus, its clinical presentation, and the challenges associated with diagnosis and treatment. It also discusses strategies to mitigate the risk of future epidemics, including vaccine development, improved epidemiological surveillance systems, and effective public health measures. Lessons learned from previous Nipah virus outbreaks provide valuable insights for the global community in strengthening preparedness and response strategies against threats posed by emerging infectious diseases.

Nipah virus is a dangerous zoonotic infection characterized by a severe course and high mortality rate. Although the risk of its introduction into our country is low, physicians must be informed about the disease. Due to international tourism and migration, there is a possibility of importation. Its symptoms resemble those of other viral fevers and encephalitides, making knowledge essential for differential diagnosis. Early suspicion facilitates epidemiological surveillance, isolation, and prevention of spread.

Virological and Molecular Characteristics. The Nipah virus (NiV) is a pleomorphic virus with a diameter of 120–150 nm, which is significantly larger than most paramyxoviruses [9]. The viral surface is characterized by a single layer of surface projections with an average length of 17 nm. The nucleocapsid diameter is approximately 5 nm, with a periodicity of 17 nm. A notable feature is the presence of reticular cytoplasmic inclusions near the endoplasmic reticulum, which distinguishes NiV from other viruses in the family *Paramyxoviridae* and contributes to its unique morphology and nomenclature [10]. NiV possesses a helical nucleocapsid and a single-stranded, non-segmented, negative-sense RNA genome with a total length of approximately 18.2 kb. The 3'-5' RNA genome encodes three non-structural and six structural proteins: nucleocapsid (N), phosphoprotein (P), matrix protein (M), fusion glycoprotein (F), attachment glycoprotein (G), and large RNA polymerase protein (L) [11]. The P gene of the Nipah virus encodes not only the phosphoprotein (P) but also three non-structural proteins—C, V, and W—through alternative open reading frames and RNA editing mechanisms. These accessory proteins contribute to the diversity of protein functions and the complex biology of Nipah viruses. All of these factors are important for the emergence and development of viral diseases [12].

Geographic Distribution and Endemic Areas. Sun et al. [13] documented 749 cases of Nipah virus infection in Bangladesh, India, Malaysia, Singapore, and the Philippines. Among patients with available demographic data, 89% (358 out of 402) were adults aged 15–59 years; males constituted 74% (391 out of 530) and females 26% (139 out of 530). The most common occupational group was livestock farmers, accounting for 68% (238 out of 351 cases). Among 489 patients with identified exposure, 69% (336) had contact with animals, 26% (127) with infected humans, and 5% (26) with activities related to date palm trees. Epidemiological patterns, such as gender, exposure, and timing of illness, varied across the five countries. The case-fatality rate for Nipah virus infection in humans is 55% [13]. Additionally, Nipah virus has been detected in 425 bats across seven countries: Thailand, Cambodia, Malaysia, Indonesia, East Timor, Bangladesh, and India.

Epidemiology

The Nipah virus is primarily associated with frugivorous bats, mainly of the genus Pteropus within the family Pteropodidae. These animals typically do not become ill but can shed the virus through saliva, urine, or feces [14]. Direct transmission involves contact with infected bats or their contaminated excretions. Indirect transmission occurs through intermediate hosts such as pigs.

Transmission of Nipah virus occurs as follows: (1) Frugivorous bats serve as the primary natural reservoir of Nipah virus; (2) Saliva, urine, blood, or feces can contaminate pigs, fruits, or date palm sap; (3) Humans can become infected through direct contact with pigs or consumption of contaminated fruits; (4) Bat excreta may enter the human body; (5) Human-to-human transmission occurs via biological fluids.

The initial spread of Nipah virus in Malaysia was linked to large-scale pig farming, facilitating transmission from bats to pigs and subsequently to humans [15]. Human-to-human transmission is a significant concern, as the virus can spread through direct contact with infected biological fluids. This mode has been observed in hospital settings and among close relatives of infected patients, indicating the risk of nosocomial spread and person-to-person transmission. In Bangladesh, food- and waterborne transmission, particularly through contaminated date palm sap, has been identified as a major risk factor. Epidemiological studies have established a strong association between consumption of contaminated food products, such as fresh date palm sap, and Nipah virus infection. The sap is likely contaminated during harvesting when it comes into

contact with infected bats. Therefore, preventing transmission of the Nipah virus requires addressing potential sources of infection, especially through contaminated food products [14].

Pathogenesis

Detection of the Nipah virus in the upper respiratory tract epithelial cells during the early stage of disease indicates that the virus may enter via respiratory secretions or aerosols. The susceptibility of respiratory epithelium suggests that this site serves as the primary portal of entry [16]. Histopathological analysis of fatal human cases suggests that endothelial cells may be the main target cells, although sufficient evidence to fully confirm this role is still lacking. The Nipah virus uses the G protein for cell attachment, binding to ephrin B2/B3 receptors, which activates the F protein to mediate membrane fusion and viral entry into host cells [17].

In the later stages of disease, viral replication occurs in the respiratory epithelium and subsequently spreads to the endothelial cells of pulmonary blood vessels [18]. In response to infection, levels of inflammatory cytokines such as IL-6, IL-4, TNF-α, IFN-γ, IFN-λ, and IFN-β increase in the small airways. Immune cells are subsequently recruited to the airways and lungs, ultimately leading to the development of clinical conditions characterized by respiratory failure [19]. The severe destructive and lethal effects of the Nipah virus are attributed to its P proteins, which suppress interferon-dependent antiviral signaling pathways and dampen the innate immune response [20].

NiV infection induces vasculitis in small blood vessels and capillaries, while larger and medium-sized vessels often remain unaffected [18]. The virus enters the bloodstream through damaged capillaries, causing viremia [16]. By binding to heparan sulfate, NiV attaches to circulating leukocytes without infecting them, thereby utilizing leukocytes as “Trojan horses” for dissemination within the host [21].

The Nipah virus spreads throughout the body via the bloodstream and infects various organs. There are no specific macroscopic pathological signs unique to Nipah virus infection; however, it commonly causes vasculitis in the brain, lungs, heart, kidneys, and spleen, accompanied by endothelial cell necrosis and inflammatory cell infiltration [18]. Vasculitis was observed in 62% of cases, and fibrinoid necrosis in the lungs in 59% of cases. In the spleen, loss of white pulp and acute necrotizing inflammation of the periarteriolar sheath were noted. No pathological changes were observed in the liver, skeletal muscles, or other tissues [18].

Inflammatory mediators disrupt the integrity of the blood-brain barrier, leading to the manifestation of central nervous system (CNS) symptoms in patients [22]. The Nipah virus infects T cells expressing CD16; CD16 is a potent receptor for the leukocyte adhesion molecule ALCAM (CD166), which is highly expressed on microvascular endothelial cells forming the blood-air and blood-brain barriers. This explains the virus’s predilection for infecting small blood vessels in the lungs and brain [23].

The central nervous system is the most severely affected organ, with primary pathological features consisting of vasculitis, thrombosis, parenchymal necrosis, and viral inclusions. Vasculitis predominantly affects small arteries of the brain, while medium and large arteries are less commonly involved. Brain herniation has been observed in some cases [18]. Finally, the Nipah virus can rapidly enter the central nervous system via the olfactory pathway [24].

Clinical Presentation

Nipah virus causes an acute and rapidly progressive illness that primarily affects the respiratory and central nervous systems. Symptoms typically appear 3–14 days after exposure. Up to 11% of cases may be asymptomatic. In typical cases, respiratory failure and encephalitis may occur. Patients infected with the Nipah virus strain often develop atypical pneumonia and severe respiratory problems, including acute respiratory distress syndrome [25].

Initial symptoms may include fever, headache, myalgia (muscle pain), nausea, vomiting, sore throat, cough, and/or difficulty breathing. Acute encephalitis may manifest with dizziness, drowsiness, altered consciousness, and other neurological signs. In severe cases, encephalitis and seizures can lead to unconsciousness within 24–48 hours. Progression to encephalitis is associated with poor outcomes, with death occurring on average six days after symptom onset [26].

Most individuals who survive encephalitis recover fully; however, approximately 20% of survivors may experience persistent neurological sequelae (seizures, severe fatigue, and behavioral changes). In a small proportion of patients, relapsed or late-onset encephalitis (occurring weeks, months, or even years after recovery) may develop and can be fatal [27, 28].

The case-fatality rate typically ranges from 40% to 75%, but may be higher depending on the viral strain, disease severity, and availability of appropriate and high-quality medical facilities [29, 30, 31].

Laboratory Diagnosis

Reverse transcription polymerase chain reaction (RT-PCR) is the gold standard for detecting NiV, enabling rapid and specific identification of viral RNA in clinical samples. Real-time RT-PCR assays targeting the N and G genes have been developed for use in both outbreak and surveillance settings [32].

Serological tests, including enzyme-linked immunosorbent assay (ELISA) and neutralization tests, are used to detect NiV-specific IgM and IgG antibodies. Seroconversion usually occurs after the first week of illness, making serology particularly useful for retrospective diagnosis and seroprevalence studies [33, 34].

Virus isolation, although definitive, requires Biosafety Level 4 (BSL-4) facilities and is therefore limited to specialized laboratories [32].

Treatment Principles

According to the National Center for Disease Control, treatment is primarily supportive, focusing on rest, adequate hydration, and symptom management. Various pharmacological agents have been tested in animal models to inhibit Nipah virus replication. Ribavirin, the monoclonal antibody m102.4, and favipiravir have been investigated as potential treatments. During the 1998–1999 Malaysian outbreak, ribavirin was used and reduced the mortality rate by 36% without significant adverse effects [35]. The monoclonal antibody m102.4, developed against the ephrin-B2 and ephrin-B3 receptors, effectively neutralizes the virus in vitro and in vivo, demonstrating potential for passive immunotherapy [6]. It has been used experimentally to protect animals from lethal Nipah virus challenge [36].

Research into therapeutics against Nipah virus (NiV) continues. Ribavirin reduced mortality by 36% during the 1998–1999 Malaysian epidemic, remdesivir provided 100% survival in infected African green monkeys, and favipiravir fully protected rats.

Prevention

Avoiding contact with pigs and livestock, refraining from consuming raw date palm sap, and drinking sufficient clean water can reduce the risk of Nipah virus infection. Date palm trees that may serve as bat roosting sites should be planted away from livestock farms and pastures [37]. Healthcare workers are advised to use personal protective equipment when treating Nipah virus patients [38]. Active surveillance, contact tracing, and accurate diagnosis are essential for interrupting transmission. Development of a vaccine against Nipah virus faces significant challenges, including the sporadic nature of the disease, incomplete understanding of protective immunity, limited private-sector involvement, and the scarcity of BSL-4 laboratories for preclinical testing. Priority areas include diagnostics, immunotherapy, vaccines, and antiviral

drugs [39]. Understanding the role of bats in disseminating pathogens is crucial for preventing the spread of deadly viruses between species, particularly between animals and humans.

Conclusion

The Nipah virus is an extremely contagious and highly dangerous pathogen that demands urgent attention. Preventive measures, such as avoiding contaminated food and implementing infection control practices, are decisive in halting its spread. An interdisciplinary "One Health" approach involving collaboration among physicians, veterinarians, and ecologists is essential to mitigate the risks posed by Nipah virus. The development of diagnostic tools, immunotherapies, vaccines, and antiviral agents plays a key role in combating the virus. Given its high mortality rate and potential for human-to-human transmission, comprehensive research and collaborative efforts are urgently needed to effectively interrupt the chain of infection.

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