Assessment of Infection Control Practices Among Nurses, Laboratory Staff, and Pharmacists in Nipah Virus Care-An Updated Review

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Abstract

Background: Nipah virus (NiV) is a highly pathogenic zoonotic RNA virus within the *Henipavirus* genus, responsible for recurrent outbreaks in South and Southeast Asia. It is associated with high case fatality rates, severe encephalitis, and respiratory failure, and is classified as a Biosafety Level 4 pathogen due to the absence of licensed vaccines or definitive antiviral therapy. Healthcare workers, particularly nurses, laboratory personnel, and pharmacists, are at increased risk of occupational exposure during outbreak response, making infection control practices critical for prevention and containment.

Aim: This updated review aims to evaluate infection control practices among nurses, laboratory staff, and pharmacists in the context of Nipah virus care, with emphasis on transmission prevention, biosafety adherence, and multidisciplinary preparedness.

Methods: A structured narrative review approach was applied, synthesizing current peer-reviewed literature, WHO guidance, and outbreak reports related to Nipah virus infection control. Evidence was analyzed focusing on clinical transmission risks, laboratory biosafety procedures, pharmaceutical handling protocols, and nursing-level infection prevention strategies in healthcare settings.

Results: Findings indicate that Nipah virus transmission occurs through direct contact with infected animals, contaminated food products, and human-to-human spread via body fluids. High-risk exposure is amplified in hospital settings without strict infection control adherence. Effective prevention relies on isolation protocols, personal protective equipment, environmental decontamination, and early detection through RT-PCR diagnostics. Laboratory staff face significant biosafety risks requiring BSL-4 containment measures. Nurses play a central role in barrier nursing and patient isolation, while pharmacists contribute to antiviral stewardship and investigational drug management. Gaps remain in rapid diagnostic availability and standardized antiviral therapy.

Conclusion: Effective infection control in Nipah virus care depends on coordinated multidisciplinary practices. Strengthening training, biosafety compliance, and outbreak preparedness among nurses, laboratory staff, and pharmacists is essential to reduce transmission and improve outbreak response.

Keywords: Nipah virus, infection control, nurses, laboratory staff, pharmacists, biosafety, outbreak management

Introduction

Nipah virus (NiV) represents a highly pathogenic RNA virus classified within the family *Paramyxoviridae* and the genus *Henipavirus*. This genus also encompasses other significant zoonotic viruses, including Hendra virus, Langya virus, Mojiang virus, and Cedar virus. Since its initial identification in 1998 during an outbreak in Malaysia, NiV has continued to emerge sporadically, producing multiple outbreaks across regions of South and Southeast Asia. The recurrent nature of these outbreaks, combined with the virus's capacity for rapid transmission and severe clinical outcomes, has led the World Health Organization (WHO) to designate Nipah virus as a priority pathogen requiring urgent research and preparedness efforts due to its high epidemic potential and public health significance.[1] Nipah virus is fundamentally a zoonotic pathogen, with its primary natural reservoir identified as fruit bats of the genus *Pteropus*. Transmission to humans is not typically direct from bats alone; rather, spillover events often involve

intermediate hosts, most notably domestic pigs, which can amplify viral transmission before human infection occurs. Once introduced into human populations, the virus can spread through direct contact with infected individuals or contaminated bodily fluids, contributing to nosocomial and community transmission patterns observed in previous outbreaks. The epidemiological behavior of NiV underscores its complex transmission ecology, which is influenced by interactions between wildlife reservoirs, domestic animals, and human populations.[2]

From a biosafety perspective, Nipah virus is categorized as a Biosafety Level 4 (BSL-4) pathogen due to its high case fatality rate, severe clinical manifestations, and the absence of approved specific antiviral therapy or licensed vaccine. The lack of definitive treatment options significantly heightens the risk associated with laboratory handling and clinical management, necessitating the highest level of containment measures to prevent accidental exposure and secondary transmission. Clinical infections are often associated with severe encephalitis and respiratory complications, further emphasizing the need for stringent infection control protocols in healthcare environments. Given its zoonotic origin and multifactorial transmission pathways, effective prevention and control of Nipah virus necessitate an integrated One Health approach. This framework emphasizes the interdependence of human health, animal health, and environmental conditions in the emergence and propagation of infectious diseases. Coordinated surveillance across veterinary and human health sectors, alongside ecological monitoring of bat populations, is essential to identify and mitigate spillover risks at an early stage. Strengthening interdisciplinary collaboration and implementing comprehensive public health strategies are therefore critical to reducing the likelihood of future outbreaks and limiting the global impact of this high-consequence pathogen.

Etiology

Nipah virus (NiV) is taxonomically classified within the genus *Henipavirus*, which is part of the subfamily *Orthoparamyxovirinae* under the broader family *Paramyxoviridae*. It is structurally characterized as an enveloped virus containing a single-stranded, negative-sense RNA genome. This genomic configuration is associated with a relatively high mutation capacity and efficient replication within host cells, contributing to its pathogenic potential and adaptability across multiple species. The viral genome is organized into six functional genes that encode essential structural and non-structural proteins. These include the nucleocapsid (N), phosphoprotein (P), matrix (M), and large polymerase (L) genes, which collectively form the ribonucleoprotein complex responsible for viral replication, transcription, and assembly. The matrix protein plays a central role in viral particle organization and budding from infected host cells, facilitating efficient viral dissemination. The polymerase complex is critical for RNA synthesis and genome replication, ensuring sustained viral propagation within susceptible hosts. In addition to these internal structural components, Nipah virus encodes two key surface glycoproteins, namely the fusion (F) protein and the attachment (G) glycoprotein. These proteins are essential determinants of viral infectivity and host cell entry. The G glycoprotein mediates initial viral attachment by binding to specific host cell receptors, while the F protein facilitates membrane fusion between the viral envelope and host cell membrane, enabling the entry of viral genetic material into the host cytoplasm. Importantly, these glycoproteins are also primary targets for neutralizing antibodies, making them significant in immune recognition and vaccine development strategies.[3]

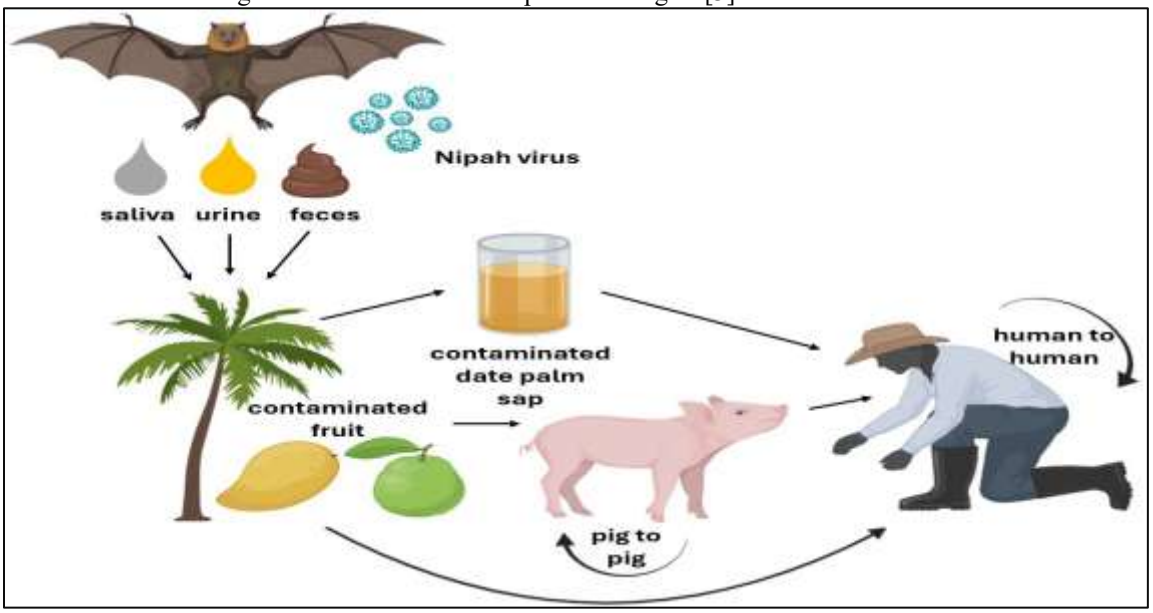

Fig. 1: Etiology of Nipah Virus.

A critical aspect of NiV pathogenesis lies in the interaction between the G glycoprotein and host cell receptors, particularly ephrin-B2 and ephrin-B3. These receptors are widely expressed in endothelial and neuronal tissues, which explains the virus's strong tropism for the vascular and central nervous systems. The broad distribution of these receptors contributes to the multisystemic nature of Nipah virus infection, including severe neurological and

respiratory manifestations observed in infected individuals. This receptor distribution also underlies the virus's ability to cross species barriers, enabling infection across a wide range of mammalian hosts. The primary natural reservoir of Nipah virus is the *Pteropus* species of fruit bats, which maintain the virus in ecological cycles without exhibiting significant disease. Spillover events from these reservoir hosts to intermediate animals such as pigs have been documented, which subsequently facilitated transmission to humans during initial outbreaks. Beyond pigs, NiV has demonstrated the ability to infect other mammalian species, including rodents, weasels, and various non-human primates, indicating a broad host range and significant zoonotic potential.[2] The molecular structure, receptor specificity, and multi-host infectivity of Nipah virus collectively define its etiology and explain its emergence as a high-consequence pathogen with significant public health implications.

Epidemiology

The epidemiology of Nipah virus (NiV) reflects a complex zoonotic transmission cycle driven by interactions between wildlife reservoirs, domestic animals, environmental contamination, and human populations. Transmission occurs through multiple routes, primarily involving direct exposure to infected animals such as fruit bats, pigs, and horses, or contact with their biological fluids, including blood, urine, and saliva. Human infection can also result from ingestion of contaminated food sources, particularly raw palm sap or partially consumed fruits contaminated by bat secretions. In addition, human-to-human transmission has been clearly documented, occurring through close contact with infected individuals or exposure to infectious body fluids such as respiratory droplets, nasal secretions, urine, and blood. A notable example of sustained human-to-human transmission was observed during the 2023 outbreak in Kerala, India, highlighting the ongoing public health risk posed by nosocomial and community spread.[4] Seasonal variation plays an important role in the epidemiological pattern of NiV, particularly in South Asia. The incidence of infection tends to increase between December and May, a period that coincides with the harvesting of raw date palm sap, which is frequently consumed fresh and unprocessed. This seasonal behavior is strongly associated with spillover events from bats to humans, as fruit bats contaminate sap collection sites during feeding activities. Although most cases occur during this seasonal window, sporadic infections have also been reported as late as July, indicating that environmental and behavioral factors may extend risk periods beyond the primary transmission season.[5] In contrast, in Southeast Asia, NiV outbreaks have generally been nonseasonal and closely linked to intensive pig farming systems, particularly in Malaysia and Singapore, where pigs served as amplifying hosts facilitating widespread transmission among humans and animals.[6][7]

Environmental persistence of the virus significantly contributes to its transmission dynamics. Nipah virus has demonstrated the ability to remain viable for several days on fruit pulp and palm sap, thereby extending the window of exposure for human infection. Additionally, studies have shown that the virus can persist in bat urine with a half-life of approximately 18 hours, further increasing the likelihood of environmental contamination and indirect transmission.[8][9][10] Following exposure, the incubation period typically ranges from 4 to 14 days, although variability in clinical onset has been reported depending on the route and intensity of exposure as well as host factors.[6] From a virological standpoint, two major genetic strains of Nipah virus have been identified: the Bangladesh strain and the Malaysian strain. These strains differ in their epidemiological behavior and clinical outcomes. Notably, strains circulating in South Asia, particularly Bangladesh and India, exhibit a greater capacity for human-to-human transmission compared to the Malaysian strain, contributing to more frequent and clustered outbreaks in these regions.[11][12] The first recognized outbreak of Nipah virus infection occurred in 1998 near Ipoh in Perak, Malaysia, initially involving pig farmers and leading to significant economic and public health consequences. In 1999, the virus was successfully isolated from the cerebrospinal fluid of a patient in Sungai Nipah village, from which the virus derives its name.[1][2] A subsequent outbreak in Singapore in 1999 resulted in 11 confirmed cases and one fatality, linked to imported pigs from Malaysia.[13]

In Bangladesh, the epidemiological pattern shifted significantly following the first documented outbreak of encephalitis in Meherpur in 2001. Unlike Malaysia, pig involvement was absent due to cultural and agricultural practices that limit pig farming. Instead, repeated seasonal outbreaks have been observed, each believed to originate from independent spillover events from fruit bats. This pattern underscores the direct role of environmental contamination and foodborne transmission in this setting.[2] A similar outbreak occurred in Siliguri, West Bengal, India, in 2001, geographically adjacent to Bangladesh. Retrospective investigations strongly suggest Nipah virus as the causative agent, with transmission likely linked to contaminated date palm sap consumption.[14][15] Further expansion of the epidemiological footprint of NiV was documented in 2014 in the Philippines, where transmission was associated with exposure to infected horses, demonstrating similarities to Hendra virus epidemiology observed in Australia.[2][16] More recently, between 2018 and 2025, the southern Indian state of Kerala has experienced multiple recurrent outbreaks, totaling eight distinct episodes. These outbreaks have consistently implicated fruit bats as the primary reservoir, reinforcing the ongoing risk of zoonotic spillover in regions where human activity overlaps with bat habitats.[17][18][19]

Pathophysiology

The pathophysiology of Nipah virus (NiV) infection is driven by a complex zoonotic cycle involving reservoir hosts, intermediate amplifying hosts, and susceptible human populations. The primary natural reservoir of the virus in Bangladesh and India is the Indian flying fox (*Pteropus giganteus*), which maintains viral circulation without

manifesting overt disease, thereby serving as a long-term ecological source of infection.[20][21] In addition to this species, several other *Pteropus* bats across different geographical regions, including Cambodia, Thailand, Indonesia, and Madagascar, have been shown to possess anti-Nipah virus antibodies, indicating prior exposure and supporting the widespread distribution of henipaviruses within bat populations.[22][23][24][25] Although less common, non-*Pteropus* bat species have also been identified as potential reservoir hosts in certain ecological settings, suggesting that viral maintenance may not be strictly limited to a single bat genus and may involve broader chiropteran biodiversity under specific environmental conditions.[23][26][27]

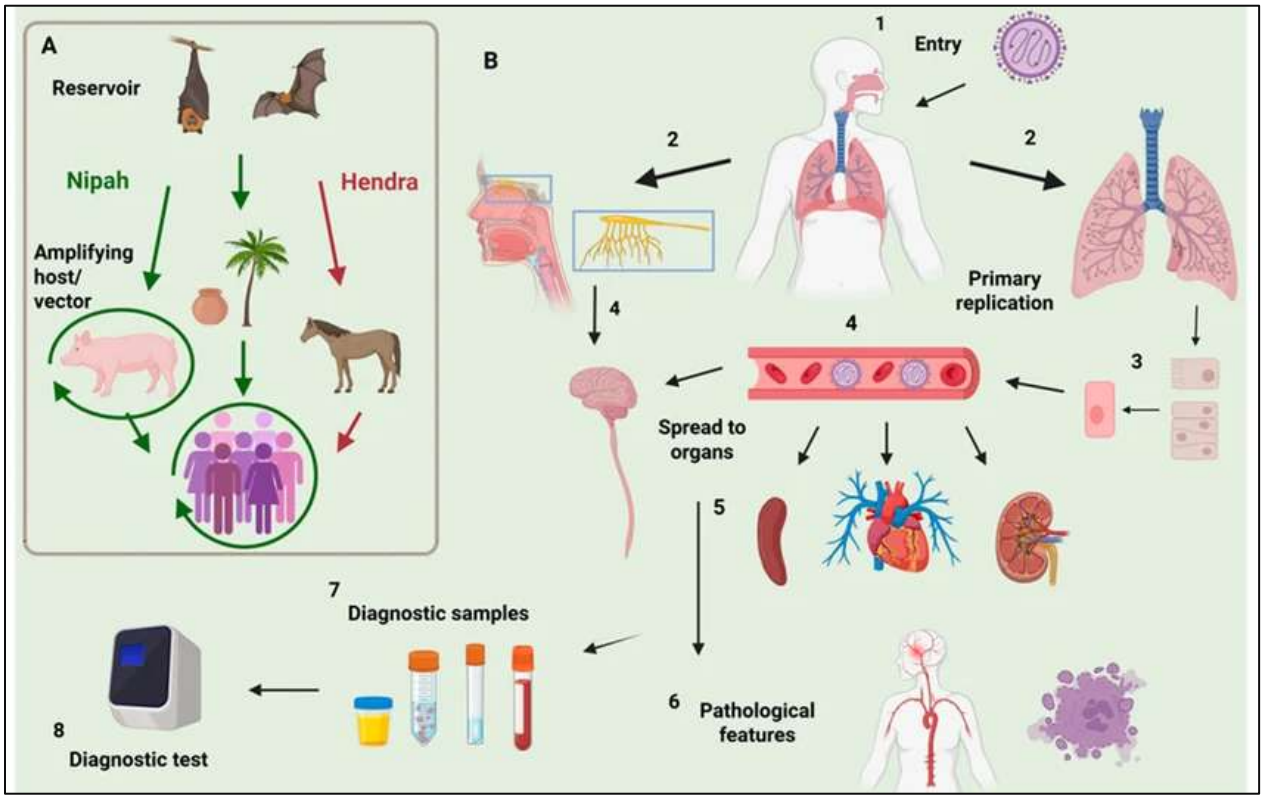

Fig. 2: Nipah Virus Pathogenesis.

Intermediate hosts play a critical role in amplifying viral transmission and facilitating spillover to humans. During the initial outbreaks in Malaysia and Singapore, domestic pigs served as the principal intermediate hosts, enabling efficient viral replication and transmission within agricultural settings and significantly increasing human exposure risk.[28] In contrast, during the outbreak in the Philippines, horses were identified as intermediate hosts, demonstrating that NiV can adapt to different mammalian species depending on ecological and farming contexts. These variations highlight the flexibility of the virus in crossing species barriers and establishing transmission chains in diverse agricultural systems. Human infection typically occurs through the oronasal route, following exposure to contaminated secretions or materials. After entry into the host, the virus demonstrates a predilection for epithelial and lymphoid tissues, where high antigen concentrations suggest these sites as primary loci of viral replication. Early replication in these tissues facilitates systemic dissemination through lymphatic and vascular pathways, enabling widespread organ involvement. A key determinant of viral entry is the attachment glycoprotein (G protein), which mediates binding to host cell receptors ephrin-B2 and ephrin-B3. These receptors are widely expressed in endothelial cells, smooth muscle cells, and neurons, providing a mechanistic explanation for the broad tissue tropism and severe systemic manifestations associated with NiV infection.[29]

Following receptor binding, viral entry is facilitated by membrane fusion processes that allow the viral RNA to access the host cytoplasm, initiating replication. Infection of the respiratory tract epithelium is a central event in disease progression and is associated with a robust inflammatory response characterized by the release of pro-inflammatory cytokines. This immune activation contributes to pulmonary pathology resembling acute respiratory distress syndrome (ARDS), which is often observed in severe clinical cases.[30] The inflammatory cascade, combined with direct cytopathic effects, leads to progressive tissue injury and respiratory compromise. Beyond the respiratory system, Nipah virus demonstrates a strong propensity for multisystem involvement. Viral dissemination can affect the central nervous system, kidneys, and spleen, reflecting its widespread endothelial tropism. In the brain, infection of neurons and endothelial cells contributes to encephalitis, a hallmark of severe NiV disease. Renal and splenic involvement further exacerbates systemic dysfunction, often culminating in multiorgan failure in advanced stages of infection.[10] The combination of direct viral cytotoxicity, endothelial damage, and dysregulated host immune responses forms the core pathological framework underlying the high morbidity and mortality associated with Nipah virus infection.

History and Physical

The clinical presentation of Nipah virus (NiV) infection typically begins with a nonspecific febrile prodrome that makes early recognition challenging. Initial symptoms commonly include fever, headache, dizziness, and episodes of vomiting, which may be mistakenly attributed to other viral or systemic infections during the early phase of illness. In some patients, respiratory system involvement develops concurrently or shortly after the onset of systemic symptoms, reflecting early viral replication in respiratory epithelium and associated inflammatory responses. Clinical manifestations vary significantly depending on the viral strain and geographic region. Infections caused by strains circulating in India and Bangladesh are more frequently associated with prominent respiratory involvement, whereas patients infected with Malaysian strains generally present with minimal or absent respiratory symptoms.[14] Despite the severity often associated with Nipah virus infection, epidemiological studies have demonstrated that a considerable proportion of infections may remain asymptomatic. Serological surveys indicate that between 8% and 45% of infected individuals may not develop overt clinical disease, suggesting a broader spectrum of infection severity than previously recognized.[6][31] This asymptomatic or subclinical carriage plays an important role in understanding transmission dynamics and may contribute to unrecognized viral circulation in endemic regions. Following the initial febrile phase, the disease frequently progresses rapidly to involve the central nervous system, resulting in acute encephalitis. Patients may develop a rapid decline in neurological function characterized by lethargy, progressive disorientation, and eventual coma in severe cases. Neurological examination often reveals a wide range of abnormalities, including decreased level of consciousness, brainstem dysfunction, myoclonus, areflexia, hypotonia, and cerebellar signs, reflecting widespread involvement of both cortical and subcortical structures. Clinical deterioration is often marked by autonomic instability, including hypertension and tachycardia, alongside progressive impairment of consciousness, indicating worsening intracranial involvement and systemic stress response.[7]

In severe or fulminant cases, Nipah virus infection can progress to multiorgan dysfunction syndrome. Complications may include gastrointestinal bleeding and acute renal failure, both of which contribute significantly to mortality. The systemic nature of the disease reflects the virus's ability to infect endothelial cells across multiple organ systems, leading to vascular injury and widespread inflammation. In outbreaks observed in Bangladesh, respiratory manifestations have been particularly prominent, with up to 69% of patients exhibiting respiratory tract symptoms. A subset of these cases progressed to acute respiratory distress syndrome (ARDS), highlighting the severity of pulmonary involvement in this regional variant.[6][32][33][34] This contrasts with earlier Malaysian outbreaks, where respiratory symptoms were less frequently reported, emphasizing the influence of viral strain and epidemiological context on clinical expression. Long-term neurological sequelae have also been documented among survivors of Nipah virus encephalitis. These complications may persist for months or years following acute infection and include chronic fatigue, ocular motor palsies, cervical dystonia, focal motor deficits, and facial nerve paralysis. In some cases, the disease course is atypical, with delayed onset of symptoms or relapse after apparent recovery from the initial infection. Such relapsing or late-onset neurological disease underscores the potential for persistent or reactivated viral effects within the central nervous system, necessitating long-term follow-up of affected individuals.[35]

Evaluation

The laboratory and diagnostic evaluation of Nipah virus (NiV) infection reveals a pattern of systemic hematological, biochemical, and neurological abnormalities that reflect the multisystemic nature of the disease. Common hematological findings include leukopenia and thrombocytopenia, which indicate bone marrow suppression or peripheral consumption secondary to systemic viral infection and immune activation. Biochemical abnormalities frequently demonstrate hyponatremia, alongside elevated hepatic transaminases, specifically alanine aminotransferase (ALT) and aspartate aminotransferase (AST), suggesting hepatic involvement and generalized inflammatory injury affecting multiple organ systems.[13] These non-specific laboratory abnormalities are often the first indicators of systemic viral infection but lack diagnostic specificity, necessitating further confirmatory testing. Definitive detection of Nipah virus relies on virological assays performed on multiple clinical specimens, including urine, blood, cerebrospinal fluid (CSF), and respiratory secretions. Viral identification can be achieved through culture techniques and molecular methods such as polymerase chain reaction (PCR). Among these, real-time reverse transcription–polymerase chain reaction (RT-PCR) is the most widely utilized and clinically valuable diagnostic modality due to its high sensitivity, specificity, and rapid turnaround time.[36][37][38][39][40] RT-PCR allows early detection of viral RNA during the acute phase of infection, even before serological responses become detectable, making it essential for timely diagnosis and outbreak control.

Serological testing is also employed in the diagnostic workup of NiV infection, particularly in resource-limited or early-stage clinical settings. However, serology alone lacks specificity due to potential cross-reactivity with related henipaviruses such as Hendra virus. For this reason, confirmatory testing using serum neutralization assays conducted in biosafety level 4 (BSL-4) laboratories or PCR-based methods is required to establish definitive diagnosis. The diagnostic sensitivity of IgM antibodies varies with disease progression, being relatively low at symptom onset, approximately 44% to 50%, increasing to 60% to 71% by day four, and reaching up to 100% by day twelve of illness.[41] IgG antibodies demonstrate even slower early detection, with positivity rates as low as 7% during the first week of symptoms, followed by progressive seroconversion over time. Consequently, IgG testing is primarily used for epidemiological surveillance and seroprevalence studies rather than acute diagnosis, providing valuable data on population-level exposure and transmission dynamics.[42] Currently, there are no validated lateral flow rapid

diagnostic assays available for Nipah virus, which limits point-of-care testing capabilities during outbreaks. Cerebrospinal fluid analysis in affected patients commonly demonstrates elevated white blood cell counts and increased protein levels, consistent with viral encephalitis.[6] In acute cases, virus-specific antibodies are detected in serum samples in more than 70% of patients, whereas CSF samples show positivity in fewer than one-third of cases. The detection of viral RNA or culture positivity in CSF has been strongly associated with increased mortality, indicating more severe central nervous system involvement and poorer clinical outcomes.[43]

Neurophysiological assessment using electroencephalography (EEG) in patients with encephalitis often reveals characteristic findings, including bilateral temporal periodic complexes with diffuse, symmetric sharp and slow wave activity occurring at intervals of one to two seconds. The degree of EEG slowing correlates with the severity of encephalitis, reflecting progressive cerebral dysfunction. However, while EEG is sensitive in identifying encephalopathic changes suggestive of Nipah virus infection, it lacks diagnostic specificity and cannot independently confirm the disease.[Kong et al. Neurol J Southeast Asia. 1999] Neuroimaging, particularly magnetic resonance imaging (MRI), provides further diagnostic support. Typical MRI findings include multiple small lesions ranging from 2 to 7 millimeters in diameter, best visualized on T2-weighted sequences. These lesions are commonly distributed in the subcortical and deep white matter of the cerebral hemispheres, periventricular regions, and corpus callosum. Notably, these lesions are usually not associated with significant cerebral edema or mass effect, distinguishing NiV encephalitis from other forms of acute inflammatory or vascular brain injury. A minority of patients may demonstrate leptomeningeal or parenchymal enhancement on contrast-enhanced MRI, indicating more extensive inflammatory involvement of the central nervous system.[6][45][46]

Treatment / Management

Management of Nipah virus (NiV) infection requires strict infection prevention measures combined with intensive supportive clinical care, as no fully established curative therapy currently exists. Patients with suspected or confirmed infection must be immediately isolated to reduce the risk of nosocomial and community transmission. Infection control protocols are central to management and include the use of personal protective equipment, barrier nursing techniques, and controlled handling of all biological specimens. Given the high transmissibility of the virus through body fluids and respiratory secretions, rigorous adherence to isolation procedures is essential in all healthcare settings. The cornerstone of treatment remains supportive care, focusing on the maintenance of respiratory function, hemodynamic stability, and neurological monitoring. Many patients require intensive care unit admission due to the rapid progression to encephalitis and respiratory failure. Mechanical ventilation may be necessary in cases of severe respiratory compromise or acute respiratory distress syndrome, while careful fluid and electrolyte management is required to address systemic complications. Antiviral therapy has been explored with several agents, although definitive evidence of efficacy remains limited. Ribavirin, an antiviral drug with activity against other paramyxoviruses such as respiratory syncytial virus, has been the most extensively studied agent. Clinical reports present conflicting outcomes, with some observational human studies suggesting a reduction in mortality, while experimental animal models have failed to demonstrate significant benefit.[47][48] Despite these inconsistencies, ribavirin is recommended by the National Centre for Disease Control in India and has been administered during outbreaks, including compassionate use during the Kerala outbreaks, reflecting its continued clinical consideration in the absence of superior alternatives.[6][47][49] Other antiviral agents have also been investigated for potential therapeutic roles. Acyclovir, remdesivir, and chloroquine have been evaluated in preclinical or limited clinical contexts; however, robust evidence supporting their efficacy against NiV infection is currently insufficient.[13][48][50] These agents remain under investigation, and their use is not yet standardized in clinical guidelines. Research into preventive and therapeutic strategies is ongoing, with particular interest in vaccine development. Novel vaccine platforms, including mRNA-based technologies, are being explored for their ability to induce protective antibody responses against Nipah virus antigens, particularly the glycoproteins involved in host cell entry.[51] These efforts aim to establish long-term preventive solutions for populations at risk of repeated zoonotic spillover. Additional experimental therapies have shown promising results in preclinical studies. Favipiravir, an antiviral agent approved in Japan for influenza treatment, has demonstrated efficacy in animal models such as hamsters; however, human clinical data are still lacking, limiting its current application in routine care.[52] A human monoclonal antibody, m102.4, which targets the glycoprotein of Hendra virus, has shown cross-reactive potential against Nipah virus and has been used under compassionate protocols for both treatment and post-exposure prophylaxis during outbreaks, including those in Kerala. This antibody is currently undergoing phase 1 clinical trials in Australia, reflecting ongoing efforts to translate experimental therapies into clinically validated interventions.[49] Discharge and isolation protocols are strictly regulated due to the risk of persistent viral shedding. Patients are typically discharged only after obtaining a negative real-time reverse transcription polymerase chain reaction (RT-PCR) result from throat swab testing. Furthermore, individuals confirmed with NiV infection are generally required to remain in isolation for a minimum period of 21 days following diagnosis, ensuring adequate containment and minimizing the risk of secondary transmission.[1]

Differential Diagnosis

The differential diagnosis of Nipah virus (NiV) infection is broad and includes a wide range of infectious and non-infectious conditions that present with acute febrile illness, encephalitis, or severe respiratory compromise such as acute respiratory distress syndrome. Because the initial clinical presentation of NiV is non-specific, involving fever,

headache, vomiting, and rapidly evolving neurological or respiratory symptoms, accurate diagnosis depends heavily on epidemiological context. Factors such as recent travel history, exposure to animals (particularly bats, pigs, or horses), consumption of potentially contaminated food such as raw date palm sap, and known outbreaks in the region are critical in narrowing the diagnostic possibilities. Among the most important infectious differentials are other viral encephalitides. Japanese encephalitis virus remains a leading cause of viral encephalitis in endemic regions and presents with similar neurological deterioration. West Nile virus encephalitis also shares overlapping clinical features, including fever and neuroinvasive disease affecting the central nervous system. Measles virus encephalitis, particularly in the form of subacute sclerosing panencephalitis, can present with progressive neurological decline, though it typically follows a more chronic course. Other members of the Henipavirus genus must also be considered due to their structural and pathogenic similarities to Nipah virus. Additional viral causes include dengue virus encephalitis and human herpesvirus infections, both of which can produce neurological manifestations ranging from mild confusion to severe encephalopathy. Rabies virus infection is a critical consideration in the differential diagnosis, particularly in patients with a history of animal exposure, as it can closely mimic acute viral encephalitis with rapidly progressive neurological decline and near-universal fatality if untreated [53].

Parasitic and bacterial infections also form an essential part of the differential spectrum. Cerebral malaria, caused by Plasmodium falciparum, can present with fever, altered consciousness, and neurological impairment, particularly in endemic regions. Scrub typhus and leptospirosis may also produce febrile illness with central nervous system involvement and multi-organ dysfunction, complicating clinical differentiation in early stages. Bacterial meningitis must also be considered, especially in cases presenting with acute fever, neck stiffness, and altered mental status. In addition to infectious etiologies, autoimmune encephalitis represents an important non-infectious differential diagnosis. This condition can present with subacute neuropsychiatric symptoms, seizures, and cognitive decline, often mimicking viral encephalitis in early stages. However, autoimmune causes typically lack the systemic infectious features and epidemiological associations seen in Nipah virus infection. Overall, the differential diagnosis of NiV infection requires careful integration of clinical presentation, laboratory findings, imaging studies, and epidemiological risk factors. The overlap of symptoms with multiple infectious and inflammatory conditions makes early recognition challenging, emphasizing the importance of high clinical suspicion in endemic and outbreak settings.[53]

Prognosis

The prognosis of Nipah virus (NiV) infection remains extremely poor, reflecting its high virulence, neurotropism, and capacity for rapid systemic deterioration. Reported case fatality rates vary widely between 40% and 100%, depending on the outbreak setting, viral strain, and quality of supportive care. Mortality is particularly high in cases presenting with severe neurological involvement, especially when the brainstem is affected. Brainstem dysfunction is clinically significant and may be indicated by abnormal oculocephalic reflexes, autonomic instability, and tachycardia, all of which reflect advanced central nervous system compromise and poor neurological prognosis.[6][53] Several clinical and laboratory parameters are associated with worse outcomes. Viral isolation from cerebrospinal fluid (CSF) has been strongly correlated with increased mortality, indicating more extensive central nervous system invasion and uncontrolled viral replication.[43] Neurophysiological findings also contribute to prognostic assessment. Electroencephalography often reveals bitemporal periodic complexes in patients with severe encephalitis, particularly in those who progress to coma or death. These findings reflect widespread cortical dysfunction and are commonly observed in advanced disease stages, although they are not independently predictive of survival.[Kong et al. Neurol J Southeast Asia. 1999]

The strain of Nipah virus plays a significant role in determining clinical outcomes. The Malaysian strain has been associated with comparatively lower mortality rates, whereas strains circulating in Bangladesh and India demonstrate higher virulence and greater capacity for human-to-human transmission, contributing to more severe outbreaks and increased fatality rates.[54] This variation highlights the importance of viral genetic diversity in shaping disease severity and epidemiological impact. In addition to acute mortality, Nipah virus infection is also associated with relapsed and late-onset encephalitis, which can occur months to years after apparent recovery from the initial illness. These delayed neurological manifestations may present with recurrent fever, headaches, seizures, and progressive neurological deficits. Neuroimaging during relapse may demonstrate evolving cerebral perfusion abnormalities, transitioning from focal hyperperfusion to hypoperfusion on CT positron-emission tomography. Magnetic resonance imaging may reveal more diffuse and confluent cortical involvement compared to the focal hyperintense lesions typically observed in acute infection, with relatively less involvement of the brainstem.[45] Although relapsed and late-onset encephalitis is generally associated with a lower mortality rate of approximately 18%, it remains clinically significant due to persistent neurological morbidity.[55] The underlying pathogenesis of relapse is not fully understood, but evidence suggests ongoing host–pathogen interactions rather than active viral replication alone. Autopsy findings have demonstrated increased viral inclusions and larger parenchymal lesions in relapsed cases compared to acute disease, alongside persistence of viral antigens despite the absence of detectable live virus. These observations support a model in which immune-mediated mechanisms and residual viral components contribute to delayed neurological deterioration rather than direct cytopathic viral activity alone.

Complications

Nipah virus (NiV) infection is associated with a wide spectrum of long-term neurological and functional complications, reflecting its strong neurotropic characteristics and the extent of central nervous system involvement during acute disease. Survivors often experience persistent neurological deficits that significantly affect daily functioning and quality of life. These complications include behavioral and cognitive changes, cervical dystonia, cerebellar dysfunction, oculomotor palsies, generalized weakness, and facial paralysis.[56] Such manifestations may result from structural and functional damage to cortical and subcortical brain regions, cranial nerve involvement, and residual inflammatory injury following acute encephalitis. The duration and severity of these complications can extend far beyond the acute phase of infection. Neurological sequelae may persist for months or even years, leading to long-term disability and reduced functional independence. Cognitive and behavioral changes may interfere with social reintegration, occupational performance, and psychological well-being, while motor deficits such as weakness or dystonia may limit mobility and physical capacity. Oculomotor abnormalities and cerebellar dysfunction further contribute to impaired coordination and visual-motor integration, increasing the overall burden of disease. Long-term follow-up studies have highlighted the chronic impact of NiV infection on survivors. One study reported that even 10 years after acute infection, a subset of patients continued to experience persistent fatigue, excessive daytime somnolence, and focal neurological deficits.[Siva SR et al. Neurology Asia. 2009][35] These residual symptoms were associated with measurable functional impairment, as assessed by the Modified Rankin Scale, indicating ongoing limitations in daily activities and sustained neurological disability. The persistence of symptoms over such an extended period underscores the potential for NiV infection to produce chronic neurological morbidity rather than being solely an acute self-limiting illness in survivors [18][57].

Given the absence of definitive curative therapy and the severity of long-term outcomes, prevention remains a central pillar of Nipah virus control. Deterrence strategies and patient education are critical components of reducing transmission risk, particularly in endemic regions where zoonotic spillover events are more likely. Public health education should emphasize the avoidance of raw date palm sap consumption, which is a well-documented route of viral transmission, as well as minimizing exposure to bats and pigs, especially in outbreak-prone areas. The World Health Organization also advises avoiding contact with fruits that have been partially eaten by bats or contaminated with bat secretions, as these may serve as indirect sources of infection.[WHO. Nipah Virus. 2018] Occupational exposure risks must also be addressed through targeted preventive measures. Individuals involved in animal handling and slaughtering should use appropriate protective clothing and follow strict hygiene protocols to reduce the risk of zoonotic transmission. In the event of suspected outbreaks, rapid public health response is essential. This includes immediate quarantine of affected facilities, notification of veterinary and agricultural authorities, and implementation of environmental control measures such as culling infected animals and safe disposal of carcasses through burial or incineration. Because Nipah virus is classified as a reportable disease in many endemic regions as well as in countries such as the United States, prompt notification of suspected or confirmed cases is mandatory. Early reporting enables timely activation of infection control measures, including isolation of patients, restriction of movement, and coordinated outbreak response. Human-to-human transmission can be effectively reduced through strict adherence to personal protective equipment use, contact precautions, and structured contact tracing. Quarantine of exposed individuals remains a key strategy in limiting secondary transmission and controlling outbreak spread.[1][18][57]

Conclusion

Nipah virus infection remains a critical global health threat due to its high mortality rate, zoonotic transmission, and potential for rapid outbreaks. The absence of specific antiviral therapy and licensed vaccines increases reliance on strict infection control measures and multidisciplinary healthcare coordination. Nurses, laboratory personnel, and pharmacists each play essential roles in preventing transmission and managing infected patients. Nursing staff ensure effective isolation, barrier precautions, and continuous patient monitoring. Laboratory professionals are responsible for accurate and safe diagnostic procedures under high biosafety conditions, while pharmacists support therapeutic decision-making and management of investigational treatments. The review highlights that gaps in training, biosafety infrastructure, and rapid diagnostic availability continue to challenge outbreak response. Strengthening infection prevention protocols, improving interprofessional collaboration, and enhancing preparedness training are essential to reduce occupational exposure and secondary transmission. Sustainable investment in surveillance systems and healthcare workforce readiness is necessary to mitigate future outbreaks and improve global health security against Nipah virus infection.

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