Review Article

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Nipah Virus: An emerging zoonotic threat at the human–animal–environment interface

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ABSTRACT

Nipah Virus (NiV) is a highly virulent zoonotic pathogen that poses significant global public health concerns. Since its first recognition in the late 1990s, the virus has triggered numerous outbreaks with significant fatality rates, particularly in South and Southeast Asia. Fruit bats of the genus Pteropus serve as the natural reservoir of the virus, while transmission to humans can occur through direct contact with the reservoir, through an intermediary animal, or through human-to-human transmission in certain situations. In humans, NiV infection generally begins with nonspecific initial symptoms, such as fever and malaise, which can then rapidly progress to serious neurological disorders and acute respiratory failure. The rapid and progressive clinical course, coupled with limited diagnostic facilities in many endemic areas, often hinders early detection and effective outbreak control efforts. Currently, there is no specific antiviral drug or vaccine widely used for the prevention of this disease, so case management still focuses on supportive therapy and strict implementation of infection control measures. In addition to its impact on human health, the NiV outbreak also has widespread social and economic consequences, including disruption to the livestock industry, increased pressure on the healthcare system, and reduced productivity. This demonstrates that the NiV is not merely a clinical problem but also a multidisciplinary challenge involving various sectors. Therefore, this review article presents a comprehensive discussion of the NiV, covering aspects of etiology, epidemiology, clinical manifestations, and its implications for public health. The One Health approach is emphasized as a crucial strategy in NiV prevention and control efforts through the integration of surveillance activities, health system strengthening, and the development of sustainable preventive interventions.

Introduction

Nipah Virus (NiV) is a highly virulent zoonotic pathogen belonging to a group of viruses with the potential to cause serious outbreaks in both humans and animals (Madhukalya *et al*., 2025). Since its first report in the late 1990s, this virus has attracted global attention due to its high case fatality rate, severe clinical manifestations, and ability to transmit between species (Singh *et al*., 2019). These characteristics make NiV a continuing threat to public and animal health, while also potentially disrupting the stability of health systems, particularly in areas with intense interaction between humans, animals, and the environment (Ganguly *et al*., 2025).

NiV belongs to the genus Henipavirus in the family Paramyxoviridae and has a natural reservoir in fruit bats of the genus Pteropus (Hauser *et al*., 2021). Transmission of the virus to humans can occur directly from the natural reservoir or through intermediate hosts, such as domestic animals, influenced by ecological and social factors in a given region (Bruno *et al*., 2022). This diversity of transmission routes demonstrates the complexity of NiV epidemiology and distinguishes it from many other zoonotic pathogens, which generally have more restricted transmission patterns (Branda *et al*., 2025).

From a clinical perspective, NiV infection is often associated with acute encephalitis and severe respiratory distress that can progress rapidly to death (Khadka, 2026). The high mortality rate, coupled with the potential for human-to-human transmission, further raises concerns about NiV's potential as a global health threat (Khan *et al*., 2024). This situation is further complicated by the fact that early symptoms of infection tend to be nonspecific, making early diagnosis often difficult without adequate laboratory support (Garbuglia *et al*., 2023).

To date, there are no specific antiviral drugs or vaccines widely used to prevent NiV infection in humans (Lv *et al*., 2025). Patient management still focuses on supportive therapy and strict implementation of infection control measures (Cubelo *et al*., 2024). This situation makes prevention the primary approach to reducing the impact of the disease and emphasizes the urgency of developing effective and accessible medical interventions (Orosco, 2023).

Beyond its impact on health, the NiV outbreak also has significant social and economic implications (Zobayer *et al*., 2025). Disruptions in the livestock sector, restrictions on economic activity, and increased pressure on the healthcare system demonstrate that NiV is not only a medical issue but also a challenge to development and community resilience (Smith *et al*., 2019). Therefore, NiV control efforts require a cross-sectoral approach that integrates human health, animal health, and environmental management (Hossain *et al*., 2025).

In recent years, NiV has consistently been listed as a priority pathogen by various international health organizations (Mohapatra *et al*., 2024). This reflects global recognition of NiV's potential to trigger outbreaks with impacts that can transcend national borders (Ganguly *et al*., 2025). Therefore, the One Health approach is increasingly emphasized as a strategic framework for understanding and managing the risks posed by NiV, given the close interrelationship between biological, ecological, and social factors in its transmission dynamics (Singhai *et al*., 2021).

Based on this background, this review article aimed to provide a comprehensive overview of the NiV, emphasizing key aspects, including its virology, epidemiology, clinical manifestations, and impact on public health and the economic sector. By integrating the latest scientific findings, this review is expected to contribute to improving understanding and strengthening preparedness for potential NiV threats, both now and in the future.

Etiology

NiV is a zoonotic virus belonging to the family Paramyxoviridae, subfamily Orthoparamyxovirinae, and genus Henipavirus (Singh *et al*., 2019). This genus also includes Hendra virus (HeV), which shares genetic, antigenic, and biological characteristics with NiV (Gazal *et al*., 2022). Viruses in the genus Henipavirus are known as highly virulent pathogens, have a broad host range, and can cause severe to fatal infections in both humans and animals (Ganguly *et al*., 2025).

NiV is an enveloped virus with a single-stranded, negative-polarity, non-segmented RNA genome approximately 18.2 kb in length (Nasir *et al*., 2025). The viral genome encodes six major structural proteins arranged sequentially: nucleoprotein (N), phosphoprotein (P), matrix protein (M), fusion protein (F), attachment glycoprotein (G), and RNA-dependent RNA polymerase (L) (Salleh, 2025). In addition to these structural proteins, the P gene can also produce additional non-structural proteins through RNA editing mechanisms, including the V and W proteins, which are known to play important roles in modulating the host immune response (Yoneda *et al*., 2010).

The NiV virion has a pleomorphic shape with a helical nucleocapsid enveloped by a lipid layer derived from the host cell membrane (Skowron *et al*., 2022). The viral envelope contains glycoproteins G and F, which play a crucial role in the infection process (Salleh, 2025). The G protein binds to the cellular receptors ephrin-B2 and ephrin-B3, which are widely distributed in endothelial cells, neurons, and epithelial tissues, thus explaining NiV's tropism for the central nervous system and vascular system (Kim *et al*., 2026). Meanwhile, the F protein mediates the fusion process between the viral membrane and the host cell membrane, allowing the nucleocapsid to enter the cell's cytoplasm (Jordan *et al*., 2026).

NiV replication occurs entirely within the host cell's cytoplasm (Madhukalya *et al*., 2025). After the virus enters the cell, the ribonucleoprotein complex is released and serves as a template for the L protein-mediated transcription of viral mRNA (Hu *et al*., 2025). Next, viral protein synthesis is followed by genome replication and assembly of viral particles at the plasma membrane (Yang *et al*., 2024). In this stage, the M protein plays a crucial role as a link between the nucleocapsid and the viral envelope. Mature virions are then released from the cell through a budding mechanism (Yasukochi *et al*., 2025).

Phylogenetically, NiV is divided into two main clades, the Malaysian strain and the Bangladeshi strain, which exhibit differences at the nucleotide level and in their biological characteristics (Mire *et al*., 2016). This genetic variation is associated with differences in transmission patterns and clinical manifestations, although it does not fundamentally alter its taxonomic position (de Campos *et al*., 2024). Various virological characteristics of NiV, such as genome stability, replication strategy, and interaction with the host immune system, contribute to its ability to cause severe infections and make it a priority pathogen in the context of global health security (Garbuglia *et al*., 2023).

Epidemiology

NiV has a geographic distribution primarily focused on South and Southeast Asia, which aligns with the natural distribution of its primary reservoir, the fruit bats of the genus Pteropus (Nidom *et al*., 2025). The presence of this reservoir, combined with environmental factors and human activities, has led to a pattern of NiV outbreaks that tends to be sporadic but can recur (Singh *et al*., 2019).

NiV outbreaks were first reported in Malaysia and Singapore in the late 1990s, with pigs acting as intermediate hosts (Tan *et al*., 2024). Following this outbreak, the epicenter of NiV shifted to South Asia, particularly Bangladesh and India, which continue to report outbreaks almost annually (Veggalam *et al*., 2025). In these regions, transmission is more common without the involvement of intermediate hosts, reflecting differences in epidemiological dynamics between regions (Kiran *et al*., 2025).

NiV infections in Bangladesh and India are generally localized with limited geographic coverage, but exhibit high fatality rates (Satter *et al*., 2025). Cases are often concentrated in rural areas characterized by intense interaction between humans, domestic animals, and wildlife (Branda *et al*., 2025). Furthermore, socioeconomic factors and specific cultural practices also play a role in influencing the pattern of case distribution in these areas (Yeasmin *et al*., 2025).

Although most NiV cases are reported from Asia, the geographic distribution of Pteropus bats, its natural reservoir, spans a much wider area, including Southeast Asia, South Asia, and Australia (Gurley and Luby, 2011). The presence of reservoirs in areas that have not yet reported outbreaks suggests that the potential distribution of NiV may be broader than indicated by reported human cases (Epstein *et al*., 2020). This raises concerns about the possibility of outbreaks in new regions if ecological factors and human behavior favor transmission (Anish *et al*., 2024).

Environmental changes, including deforestation, urbanization, and agricultural expansion, also influence the geographic distribution of NiV by increasing the likelihood of contact between humans and the viral reservoir (Deka and Morshed, 2018). These factors can shift the boundaries of risk areas and open up opportunities for the virus to spread to previously unaffected areas (Ganguly *et al*., 2025).

Pathogenesis

NiV infection generally begins when the virus enters the body through the mucosa of the respiratory or gastrointestinal tract following exposure to a contaminated source (Cline *et al*., 2022). The initial stage of pathogenesis is characterized by interactions between viral glycoproteins and receptors on host cells (Mehnaz *et al*., 2024). NiV's G glycoprotein has a specific affinity for ephrin-B2 and ephrin-B3 receptors, which are highly expressed on epithelial cells, vascular endothelium, and neurons (Palomares *et al*., 2013). This receptor distribution pattern explains NiV's tissue tropism and the multi-organ system involvement often found in severe infections (Thiel *et al*., 2008).

After successfully entering the host cell, NiV replicates in the cytoplasm, followed by viral protein synthesis and the formation of new viral particles (Liew *et al*., 2022). Further viral spread occurs locally through cell-cell fusion, resulting in the formation of syncytia (Ringel *et al*., 2019). This process allows the virus to spread without direct exposure to the humoral immune response. This mechanism contributes to efficient viral replication while accelerating progressive tissue damage (Singh *et al*., 2019).

One of the key characteristics of NiV pathogenesis is the involvement of the vascular system (Erbar and Maisner, 2010). Infection of endothelial cells can trigger systemic vasculitis, characterized by impaired endothelial function, increased vascular permeability, and the formation of microthromboses (Wong *et al*., 2002). This vascular damage plays a major role in the development of edema, hemorrhage, and tissue ischemia, particularly in organs such as the brain and lungs (Cline *et al*., 2022). Consequently, neurological and respiratory disorders often appear as the dominant clinical manifestations (Faus-Cotino *et al*., 2024).

NiV has the ability to cross the blood-brain barrier, either through hematogenous spread or through the migration of infected cells (Tyagi *et al*., 2025). After reaching the central nervous system, the virus can infect neurons and glial cells, triggering necrotizing encephalitis, characterized by widespread inflammation, focal necrosis, and the formation of viral inclusions (Wong *et al*., 2002). This damage to the nervous tissue is closely associated with the appearance of severe neurological symptoms and the high fatality rate of the infection (Vindel-Zuñiga *et al.*, 2026).

In addition to its direct cytopathic effects, NiV also has mechanisms to evade the host immune response (Madhukalya *et al.*, 2025). The nonstructural proteins V and W are known to inhibit the type I interferon pathway by interfering with the activation of key transcription factors, thus diminishing the early-phase antiviral response (Shaw *et al.*, 2004). This disruption of immune regulation allows the virus to replicate continuously and contributes to worsening disease progression (Ganguly *et al.*, 2025).

Immune response

The host immune response to NiV infection is complex and often unable to effectively inhibit viral replication in the early stages (Johnston *et al.*, 2025). After entering cells, NiV is recognized by pattern recognition receptors (PRRs), such as Retinoic acid-inducible gene I (RIG-I)–like receptors and Toll-like receptors, which generally trigger the activation of the type I interferon pathway (Huang *et al.*, 2022). However, this initial antiviral response is often inhibited, allowing the virus to replicate and spread further before an optimal adaptive immune response develops (Liew *et al.*, 2022).

NiV has an effective strategy for suppressing the innate immune response (Seto *et al.*, 2010). Nonstructural proteins derived from the P gene, particularly the V and W proteins, act as interferon antagonists by interfering with the phosphorylation and nuclear translocation of Signal Transducer and Activator of Transcription 1 (STAT1) and Signal Transducer and Activator of Transcription 2 (STAT2) (Yoneda *et al.*, 2010). Inhibition of this pathway reduces the expression of interferon-induced genes, thereby reducing the host cell's ability to suppress viral replication (Rodriguez *et al.*, 2002). This leads to an increased viral load that is associated with disease severity (Hantabal *et al.*, 2026).

The adaptive immune response to NiV infection involves the activation of T lymphocytes and the formation of neutralizing antibodies (Pelissier *et al.*, 2019). Cluster of Differentiation 8 positive ($CD8^+$) T cells function to eliminate infected cells, while Cluster of Differentiation 4 positive ($CD4^+$) T cells play a role in assisting B cell activation and regulating the immune response (Topchyan *et al.*, 2023). Antibodies directed against glycoproteins G and F can neutralize the virus by inhibiting the process of viral attachment and fusion with host cells (Dang *et al.*, 2019). However, in many fatal cases, this adaptive immune response often appears too late or is not strong enough to prevent extensive tissue damage (Singh *et al.*, 2019).

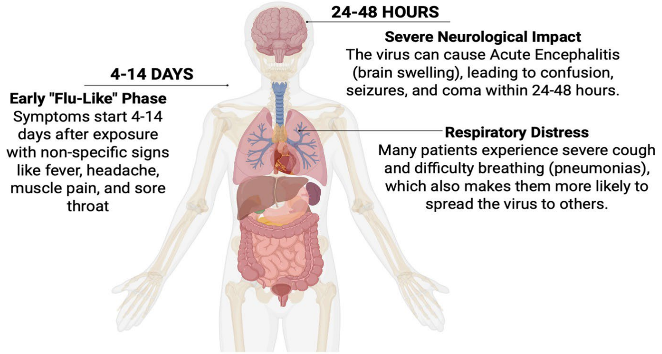
Immune system dysregulation is a key feature of NiV infection (Kaundal *et al.*, 2024). Excessive and uncontrolled immune activation can trigger the release of high levels of pro-inflammatory cytokines, which contribute to endothelial cell damage and increased vascular permeability (Fosse *et al.*, 2021). This exacerbates systemic vasculitis and tissue edema, particularly in the brain and lungs, which are major factors determining mortality (Brown *et al.*, 2023).

Furthermore, NiV infection can trigger an unstable long-term immune response. In some patients who have recovered from the acute phase, relapses or delayed-onset encephalitis have been reported, possibly related to viral persistence or an incompletely resolved immune response (Tan *et al.*, 2002). This phenomenon suggests limitations in the development of long-term protective immunity to NiV.

Pathology

NiV infection is characterized by pathological changes involving multiple organ systems, reflecting the virus's propensity to infect endothelial cells and nervous tissue (Levine *et al.*, 2025). The primary lesions are associated with extensive vascular damage, which plays a key role in the development of severe clinical manifestations in both humans and animals (Lv *et al.*, 2025). The relatively uniform lesion patterns across species indicate a common pathogenesis, although disease severity can vary (Madhukalya *et al.*, 2025).

Macroscopically, the most frequently involved organs are the brain and lungs (Kulkarni *et al.*, 2013). The brain may exhibit diffuse edema with focal hemorrhages, while the lungs typically exhibit congestion and severe edema (Mehnaz *et al.*, 2024). Spleen and lymph node enlargement have also been reported in some cases, reflecting a systemic inflammatory response (Bruno *et al.*, 2022). Figure 1 illustrates the main clinical manifestations of NiV infection, including neurological symptoms and respiratory distress associated with vascular and tissue damage in affected organs.



The virus can cause Acute Encephalitis (brain swelling), leading to confusion, seizures, and coma within 24-48 hours.

Symptoms start 4-14 days after exposure with non-specific signs like fever, headache, muscle pain, and sore throat

Many patients experience severe cough and difficulty breathing (pneumonias), which also makes them more likely to spread the virus to others.

Figure 1. Clinical signs of Nipah Virus

Histopathological examination shows that necrotizing vasculitis is a characteristic lesion of NiV infection (Torres-Velez *et al.*, 2008). This disorder is characterized by endothelial cell damage, mononuclear inflammatory cell infiltration, and thrombus formation in small to medium-sized blood vessels (Diederich *et al.*, 2023). This disruption of vessel wall integrity triggers the extravasation of blood cells and plasma proteins, which subsequently contribute to tissue edema and hemorrhage (Wong *et al.*, 2002).

In the central nervous system, pathological changes are characterized by multifocal encephalitis accompanied by neuronal necrosis, gliosis, and inflammatory cell infiltration around blood vessels (Cline *et al.*, 2022). Viral inclusions can be detected in endothelial cells and neurons, particularly in areas with severe vascular damage (Al-Obaidi *et al.*, 2024). The interaction between ischemia triggered by vasculitis and direct infection of nerve cells contributes to the development of progressive neurological disorders in NiV infection (Faus-Cotino *et al.*, 2024).

The lungs exhibit pathological changes such as interstitial pneumonia, alveolar edema, and hemorrhage (DeBuysscher *et al.*, 2013). Damage to the pulmonary capillary endothelium disrupts gas exchange and contributes to acute respiratory failure (Fosse *et al.*, 2021). Lesions with similar characteristics can also be found in other organs such as the heart, kidneys, and liver, although their frequency and severity are relatively lower in NiV infection (Levine *et al.*, 2025).

Immunopathologically, lesion formation is a consequence of the interaction between viral replication and the host's inflammatory response (Prescott *et al.*, 2012). The activation of various immune cells and the release of pro-inflammatory mediators can exacerbate tissue damage, particularly to blood vessel structures (Escudero-Pérez *et al.*, 2023). However, the adaptive immune response in the early phase of infection is often unable to effectively eliminate NiV (Hassan *et al.*, 2026).

Clinical manifestations

NiV infection exhibits a wide range of clinical signs in both humans and animals. This variation reflects the multisystem and neurotropic nature of the pathogen and is influenced by differences in host species and transmission routes (Madhukalya *et al.*, 2025). Table 1 summarizes the spectrum of clinical manifestations of NiV infection in humans and various animal species.

Clinical signs in humans

The clinical manifestations of NiV infection in humans vary widely, ranging from asymptomatic infection to severe, potentially fatal disease involving multiple organ systems (Ganguly *et al*., 2025). The incubation period generally lasts 4–14 days, although some reports have shown it to be longer than 30 days (Garbuglia *et al*., 2023). This variability in clinical presentation is influenced by the characteristics of the virus, the route of exposure, and the host's immune response (Satter *et al*., 2025).

Early symptoms of NiV infection are generally nonspecific and resemble those of an acute viral infection, including fever, headache, myalgia, fatigue, and sore throat (Alam, 2022). At this stage, establishing a clinical diagnosis is often difficult because the symptoms are similar to those of other viral infections endemic in the affected area (Kiran *et al*., 2025). As the disease progresses, most patients develop central nervous system involvement, a key characteristic of NiV infection (Hassan *et al*., 2026).

Neurological manifestations of NiV infection can include decreased level of consciousness, disorientation, seizures, tremors, and focal neurological deficits suggestive of acute encephalitis (Gavali *et al*., 2021). Neuroimaging often reveals multifocal white matter and gray matter lesions, consistent with vasculitis and neural tissue necrosis (Tyler, 2009). This condition can progress rapidly and can progress to coma within a short time (Satter *et al*., 2026).

In addition to neurological disorders, respiratory symptoms are also frequently reported, particularly in outbreaks associated with the Bangladeshi strain of NiV (Levine *et al*., 2025). These manifestations can range from coughing, shortness of breath, to acute pneumonia, which increases the potential for human-to-human transmission via droplets or respiratory secretions (Aditi and Shariff, 2019). Severe pulmonary involvement is generally associated with a poorer prognosis (Kulkarni *et al*., 2013).

Even in patients who survive the acute phase, long-term complications can still arise (Aquib *et al*., 2026a). Some individuals experience persistent neurological impairment, such as decreased cognitive and motor function (Sejvar *et al*., 2007). Furthermore, cases of recurrent or delayed-onset encephalitis have been reported months to years after initial NiV infection, indicating possible viral persistence or reactivation of the inflammatory response in the central nervous system (Ludlow *et al*., 2016).

Clinical Signs in animals

NiV infection in animals exhibits a variety of clinical manifestations influenced by several factors, such as host species, age, infectious dose, and route of exposure (Sarkar *et al*., 2025). Infected animals generally exhibit respiratory and nervous system involvement, although disease severity can vary significantly across species (de Wit and Munster, 2015). Understanding clinical signs in animals is crucial for early outbreak detection and efforts to prevent zoonotic transmission.

In pigs, which act as amplifying hosts, NiV infection is generally characterized by respiratory symptoms such as coughing, tachypnea, and frothy nasal discharge (Orosco, 2023). Neurological manifestations can also occur, particularly in piglets, including tremors, ataxia, limb weakness, and behavioral changes (Lv *et al*., 2025). Morbidity in pig populations is typically high, while mortality tends to be lower than in humans (McLean and Graham, 2019). However, the high viral shedding rate from infected pigs makes this species an important source of infection (Paliwal *et al*., 2024).

Fruit bats of the genus Pteropus, which serve as the natural reservoir for NiV, generally do not exhibit any obvious clinical signs (Field *et al*., 2001). Infection in bats is usually subclinical, although the virus can be detected in saliva, urine, and feces (Garbuglia *et al*., 2023). The lack of clinical symptoms in this natural reservoir contributes to the persistence of the virus in the environment and increases the potential for undetected transmission to other animals and humans (Yeasmin *et al*., 2025).

In other domestic animal species, such as horses, goats, dogs, and cats, reports of NiV infection are relatively rare and generally come from sporadic cases or experimental studies (Skowron *et al*., 2022). Clinical manifestations in these species can include fever, respiratory distress, and neurological signs of varying severity (Hassan *et al*., 2026). Meanwhile, in non-human primates, often used as research models, symptoms typically reflect a more severe disease, characterized by encephalitis and respiratory distress that resemble those seen in humans (Lee *et al*., 2020).

Diagnosis

Diagnosis of NiV infection requires an accurate and integrated labo-

Table 1. Clinical signs of NiV infection in humans and animals.

Host	Main organ system	Major clinical signs	Special characteristics
Humans	Systemic (early)	Fever, headache, myalgia, fatigue, and sore throat	Early nonspecific symptoms resembling acute viral infection; difficult to differentiate from other endemic viral diseases
	Central nervous system	Decreased level of consciousness, disorientation, seizures, tremors, and focal neurological deficits	Associated with acute encephalitis; multifocal lesions on neuroimaging; can progress rapidly to coma
	Respiratory system	Cough, shortness of breath, and acute pneumonia	More prominent in the Bangladesh strain; increases risk of human-to-human transmission; severe pulmonary involvement linked to poor prognosis
	Long-term complications	Cognitive and motor impairments, recurrent or delayed-onset encephalitis	May appear months to years after acute infection; may indicate viral persistence or reactivation of CNS inflammation
Pigs	Respiratory system	Cough, tachypnea, and frothy nasal discharge	High morbidity; serve as amplifying hosts; important source of virus shedding
	Nervous system	Tremors, ataxia, limb weakness, and behavioral changes	More evident in piglets; relatively low mortality; neurological signs milder than in humans
Fruit bats (*Pteropus spp.*)	Systemic and excretory	Generally asymptomatic	Subclinical infection; virus excreted in saliva, urine, and feces; natural reservoir of NiV
Other domestic animals (horses, goats, dogs, cats)	Systemic, respiratory, and nervous	Fever, respiratory distress, and neurological signs	Sporadic or experimental cases; severity varies; early recognition important for zoonotic risk
Non-human primates	Nervous and respiratory systems	Severe encephalitis and respiratory distress	Clinical manifestations resemble human infection; often used as research models for severe disease

ratory approach due to the nonspecific nature of clinical symptoms and the high pathogenicity of the virus (van den Hurk *et al*., 2025). Case determination is generally based on a combination of molecular, serological, and pathological methods, with the choice of technique tailored to the phase of infection, the type of specimen being examined, and the capabilities of laboratory facilities (Garbuglia *et al*., 2023). Table 2 summarizes the various diagnostic approaches for NiV infection, including molecular, serological, virological, and pathological methods.

Molecular detection is the primary approach in diagnosing NiV infection in the acute phase (Daniels *et al*., 2001). Methods such as reverse transcription polymerase chain reaction (RT-PCR) and real-time RT-PCR are used to identify the viral genome in various clinical specimens, including blood, cerebrospinal fluid, respiratory swabs, and organ tissue (Madhukalya *et al*., 2025). These techniques have high sensitivity and specificity, allowing confirmation of infection before the onset of a humoral immune response (Guillaume *et al*., 2004). In addition to their use in the diagnosis of individual cases, molecular approaches are also important in analyzing the genetic characteristics of the virus and in epidemiological tracking during outbreaks (Ambat *et al*., 2019).

Serological testing is generally used to identify late-stage infections or for surveillance purposes (Mazzola and Kelly-Cirino, 2019). Detection of specific antibodies to NiV, such as Immunoglobulin M (IgM) and Immunoglobulin G (IgG), is typically performed using an enzyme-linked immunosorbent assay (ELISA) (Shete *et al*., 2022). Additionally, a virus neutralization test can provide more specific confirmation by assessing the presence of functional antibodies capable of inhibiting viral infection (Liew *et al*., 2022). However, the application of this method is relatively limited due to the requirement for high-level biosafety facilities and more complex laboratory procedures (Jin *et al*., 2025).

Virus isolation from clinical specimens provides definitive evidence of the presence of NiV, but this practice is rarely performed outside of reference laboratories (Yadav *et al*., 2021). The procedure requires biosafety level 4 (BSL-4) facilities due to the high risk of transmission (Madhukalya *et al*., 2025). Therefore, virus isolation is more commonly used for research and outbreak confirmation purposes than for routine diagnosis (Daniels *et al*., 2001).

Histopathology and immunohistochemistry serve as supportive methods, particularly in fatal cases or for post-mortem investigations (Aquib *et al*., 2026b). Histology can identify characteristic lesions such as necrotizing vasculitis and multifocal encephalitis, while immunohistochemistry allows direct detection of NiV antigens in endothelial cells and neurons (Cline *et al*., 2022). This approach provides a clear link between the presence of the virus and tissue damage (Hassan *et al*., 2026).

Diagnosing NiV infection faces several challenges, including limited access to high-level biosafety facilities, symptoms similar to other infectious diseases, and limited diagnostic data in animals (Hafeez *et al*., 2025). Therefore, a diagnostic strategy combining molecular, serological, and pathological methods is the most effective approach to ensuring a correct diagnosis while supporting a rapid and efficient public health response (Mazzola and Kelly-Cirino, 2019).

Differential diagnosis

Differential diagnosis of NiV infection presents a significant clinical challenge because early symptoms are nonspecific and mimic those of various viral and non-viral infections endemic in affected areas (Ganguly *et al*., 2025). A differential diagnosis approach is crucial, particularly in the early stages of the disease and during outbreaks, to prevent delays in treatment and reduce the risk of further transmission (Rahman *et al*., 2024).

In its early stages, NiV infection is often difficult to distinguish from other acute febrile illnesses, such as influenza, dengue, chikungunya, malaria, and typhoid fever (Mazzola and Kelly-Cirino, 2019). Similar symptoms, including fever, headache, myalgia, and fatigue, make the initial clinical diagnosis less specific (Levine *et al*., 2025). Therefore, epidemiological history—such as contact with animals, consumption of risky foods, or interaction with confirmed cases—plays a crucial role in narrowing down the diagnostic possibilities (Al-Askari *et al*., 2023).

The progressive involvement of the central nervous system makes NiV infection a consideration in the differential diagnosis of acute encephalitis (Faus-Cotino *et al*., 2024). Diseases such as Japanese encephalitis, herpes simplex encephalitis, rabies, and encephalitis caused by West Nile virus or enteroviruses also require consideration (Banerjee *et al*., 2019). While neurological symptoms can be similar, NiV is typically characterized by rapid disease progression and, in some cases, respiratory distress, which can help differentiate it from other causative agents (Khadka, 2026).

In patients presenting with predominantly respiratory symptoms, particularly during outbreaks with human-to-human transmission, NiV infection should be differentiated from other severe viral pneumonias, such as avian influenza, SARS-CoV, MERS-CoV, and COVID-19 (Singh *et al*., 2019). The presence of acute pneumonia concurrent with encephalitis, particularly in individuals with a history of epidemiologically relevant exposure, should raise suspicion of NiV infection (Chowdhury *et al*., 2025).

In animals, the differential diagnosis includes a variety of infectious diseases affecting the respiratory and nervous systems (Bruno *et*

Table 2. Diagnostic methods for NiV infection based on principles, specimen types, and clinical roles.

Diagnostic method	Principle	Specimen type	Infection phase	Advantages	Limitations
RT-PCR / real-time RT-PCR	Detection of viral RNA genome through molecular amplification	Blood, cerebrospinal fluid, respiratory swabs, and organ tissue	Acute phase	High sensitivity and specificity; allows early confirmation of infection; supports epidemiological tracking	Requires molecular laboratory facilities and strict biosafety handling
ELISA (IgM/IgG)	Detection of specific antibodies against NiV	Serum	Late phase / recovery	Suitable for surveillance and seroepidemiological studies; relatively easy to perform	Not optimal for early acute phase; possible cross-reactivity
Virus neutralization test	Measurement of functional antibodies that inhibit viral infection	Serum	Late phase	High specificity; confirms protective immune response	Complex procedure; requires high-level biosafety facilities
Virus isolation	Culture of live virus from clinical specimens	Blood, tissues, and body fluids	Acute phase	Definitive evidence of virus presence	High risk; only performed in BSL-4 laboratories; not for routine diagnosis
Histopathology	Identification of characteristic tissue changes	Organ tissue (post-mortem)	Fatal cases	Shows specific patterns of tissue damage	Does not detect virus directly
Immunohistochemistry	Detection of viral antigens in target cells	Organ tissue	Fatal cases / outbreak investigation	Links presence of virus to tissue lesions; supports pathogenesis studies	Requires specific antibodies and technical expertise

al., 2022). In pigs, NiV infection can mimic classical swine fever, African swine fever, pseudorabies, or complex respiratory viral infections (Yin *et al*., 2025). In horses and other domestic animals, viral or bacterial neurological diseases must also be considered, making laboratory confirmation crucial (van den Hurk *et al*., 2025).

Transmission

NiV is transmitted through multiple pathways, demonstrating the virus's ability to move across species and adapt to different ecological conditions (Rani *et al*., 2026). NiV transmission involves a natural reservoir, an intermediate host, and direct human-to-human transmission, with environmental factors and human behavior also influencing infection (Bhate *et al*., 2025). Figure 2 illustrates the NiV transmission cycle, emphasizing the role of fruit bats as a natural reservoir, the involvement of intermediate hosts, and the spillover pathways that allow the virus to reach humans.

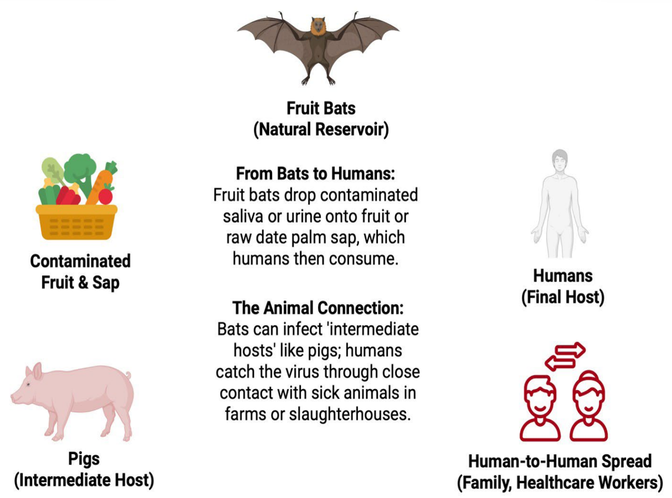


Figure 2. The transmission loop

The natural reservoir of NiV is fruit bats of the genus Pteropus, which can carry and shed the virus without showing any symptoms of disease (Sayed *et al*., 2019). The virus is excreted in urine, saliva, and feces, which can then contaminate the surrounding environment or food sources (Singh *et al*., 2019). Exposure to these biological materials allows transmission to humans and domestic animals, either through direct contact or indirectly through contaminated surfaces or food (Branda *et al*., 2025).

Foodborne transmission of NiV is a major route of transmission, particularly in South Asia (Joshi *et al*., 2023). Raw food products contaminated by bat excretions, such as fresh date palm sap, have been shown to be a source of infection for humans (Khan *et al*., 2025). NiV's ability to survive under certain environmental conditions allows the virus to remain infectious upon consumption, making this route a crucial public health concern (Mohapatra *et al*., 2024).

NiV transmission from animals to humans is most likely to occur when an intermediary animal, particularly pigs, is present (Sirait *et al*., 2024). The virus multiplies in large numbers in infected animals, being shed through respiratory secretions and body tissues (Sarkar *et al*., 2025). Intense contact during animal husbandry, transportation, or slaughtering increases the likelihood of human exposure to the virus, facilitating interspecies transmission (Luby, 2013).

In addition to the zoonotic route, human-to-human transmission also plays a role in the spread of NiV, particularly in outbreaks involving certain strains (Hossain *et al*., 2025). Transmission occurs through close contact with infected individuals, including exposure to respiratory droplets and bodily fluids (Ganguly *et al*., 2025). Respiratory involvement in some patients increases the effectiveness of transmission, particularly in household settings and healthcare facilities (Yeasmin *et al*., 2025).

Healthcare facilities can become amplification points for transmission if infection prevention and control measures are not properly implemented (Pritchard and Hornsey, 2025). Exposure of medical staff to patient secretions or aerosols during medical procedures can further spread the virus, underscoring the importance of implementing strict biosafety protocols (Orosco, 2023).

Risk factors

The occurrence of NiV infection is influenced by the interaction of ecological factors, human behavior, social conditions, and contact with contaminated animals or environments (Goswami *et al*., 2026). This combination of factors triggers spillover events from natural reservoirs to humans and allows for further transmission in the community (Bhate *et al*., 2025). Table 3 presents a summary of risk factors for NiV infection, including ecological, behavioral, social, and individual aspects.

Contact with natural reservoirs is a major risk factor for infection (Luby *et al*., 2009). Fruit bats of the genus Pteropus can shed the virus through urine, saliva, and feces, increasing the risk of exposure to people living or working near their habitat (Epstein *et al*., 2020). This risk increases with ecosystem changes, such as deforestation and land conversion, which lead to closer interactions between humans, domestic animals, and wildlife (Debnath *et al*., 2021).

Consuming certain types of food also increases the risk of infection

Table 3. Risk factors for NiV infection based on source of exposure and mechanism of transmission.

Risk factor category	Main source or condition	Mechanism of increased risk	Example context
Ecological factors	Presence of fruit bats (*Pteropus spp.*)	Exposure to viral excretions (urine, saliva, and feces) in the environment	Living or working near bat habitats
Environmental changes	Deforestation and land-use conversion	Increases interactions between humans, wildlife, and domestic animals	Clearing land for agriculture or settlements
Food consumption practices	Consumption of raw or contaminated food	Oral transmission through unprocessed food products	Drinking fresh date palm sap without protective measures
Contact with infected animals	Exposure to domestic animals (especially pigs)	Contact with respiratory secretions, blood, or tissues	Farm workers and slaughterhouse personnel
Farming system	High animal density and poor biosecurity	Facilitates virus amplification and spread	Intensive farms without disease control measures
Behavioral factors	Close contact without protection	Direct human-to-human transmission	Caring for patients without personal protective equipment
Social factors	Limited healthcare infrastructure	Suboptimal infection prevention and control	Crowded households and communities
Individual factors	Advanced age and certain health conditions	Increases susceptibility and disease severity	Repeated exposure in high-risk individuals

(Khadka, 2026). Raw food products contaminated by bat excretions, such as fresh date palm sap, have been linked to NiV infections in several endemic areas (Khan *et al*., 2025). Lack of protection of food sources and low public awareness further increase the likelihood of transmission through this route (Hossain *et al*., 2025).

Direct interaction with infected animals is a significant risk factor, especially for people working in livestock (Chew *et al*., 2000). Contact with infected pigs or other animals—through respiratory secretions, blood, or body tissue—increases the chance of infection, particularly in intensive farming systems with high animal densities and inadequate biosecurity practices (Ganguly *et al*., 2025).

Behavioral and social factors also play a role in the spread of NiV (Zobayer *et al*., 2025). Caring for patients without adequate personal protective equipment, close contact within households, and traditional care practices can facilitate transmission between individuals (Cubelo *et al*., 2024). Furthermore, limited healthcare facilities and poor adherence to infection prevention measures increase the risk of secondary transmission (Hossain *et al*., 2025).

In addition to other factors, an individual's demographic characteristics and health status also influence susceptibility to infection and disease severity (Hassan *et al*., 2026). Repeated exposure to the virus, advanced age, and certain health conditions can increase the likelihood of severe disease (Clayton *et al*., 2016). However, data on the role of individual factors is limited and requires further research.

Public health importance

NiV poses a serious threat to public health due to its high mortality rate, severe symptom spectrum, and potential for transmission, which involves complex interactions between humans, animals, and the environment (Veggalam *et al*., 2025). In humans, infection often progresses to acute encephalitis and serious respiratory distress, with high mortality rates reported in various outbreaks (Asokan *et al*., 2026). This significant mortality makes NiV a pathogen with a significant health impact despite the relatively small number of cases (Aquib *et al*., 2026a).

From an epidemiological perspective, NiV presents significant challenges in early detection and outbreak control (Bhowmik *et al*., 2025). The nonspecific nature of early symptoms makes clinical diagnosis difficult, risking delays in the public health response (Ahmed *et al*., 2024). Furthermore, evidence of person-to-person transmission, particularly through close contact and exposure to bodily fluids, increases the potential for spread within healthcare facilities and communities, particularly in areas with limited medical resources (Sun *et al*., 2024).

The lack of a specific antiviral agent or a widely available vaccine underscores the urgency of NiV as a global public health concern (Mehnaz *et al*., 2024). Current case management is supportive, making prevention efforts the primary strategy for mitigating the disease's impact (Rahman *et al*., 2024). This situation leaves the health system vulnerable, especially during recurrent outbreaks that could overwhelm the capacity of medical services and healthcare workers (Cabuhat *et al*., 2026).

NiV has far-reaching impacts beyond the health sector, including social and economic ones (Zobayer *et al*., 2025). Restrictions on movement, disruptions to agricultural and livestock activities, and public fear of transmission can lead to social instability and economic losses, particularly in endemic areas (Wegner *et al*., 2022). Thus, the effects of NiV not only impact individual health but also impact the resilience of communities as a whole (Radhakrishnan *et al*., 2021).

Globally, NiV is categorized as a priority pathogen due to its potential to trigger outbreaks with international impact (Ganguly *et al*., 2025). Its nature as a zoonotic virus with the potential to adapt to humans emphasizes the importance of a One Health approach to risk mitigation (Branda *et al*., 2025). Integrating surveillance, public education, health system strengthening, and interdisciplinary collaboration are key strategies to protect public health from current and future NiV threats (Zobayer *et al*., 2025).

Economic impact

The NiV outbreak has had a significant economic impact, both directly and indirectly, especially in areas heavily dependent on agriculture and livestock (Kiran *et al*., 2025). The direct economic impact stems primarily from healthcare costs, such as treating patients with severe symptoms, increasing the capacity of medical facilities, and implementing outbreak control measures (Aditi and Shariff, 2019). These costs increase with the length of treatment and the high demand for medical resources due to the lack of specific therapies (Yeasmin *et al*., 2025).

The livestock sector, particularly the pig industry, was among the hardest hit during the NiV outbreak (Kulkarni *et al*., 2013). Policies such as the mass culling of infected or high-risk animals, restrictions on livestock movement, and temporary farm closures resulted in significant economic losses for farmers and their associated supply chains (Espinosa *et al*., 2020). These impacts included not only the loss of livestock populations but also long-term productivity declines and reduced market confidence in animal products (Bose and Kumar, 2025).

Indirect impacts are also felt in the trade and labor sectors. Restrictions on economic activity, reduced public mobility, and concerns about disease transmission can hinder the distribution of goods and services (Smith *et al*., 2019). At the local level, outbreaks have the potential to lead to decreased household incomes, increased temporary unemployment, and reduced purchasing power, particularly in vulnerable rural communities (Sirait *et al*., 2024).

Furthermore, the NiV outbreak has prompted increased government investment in disease surveillance, research, and health preparedness (Soni *et al*., 2024). While these measures are crucial for long-term prevention, allocating significant resources in a short time can divert funds from other development sectors (van den Hurk *et al*., 2025). Thus, NiV has the potential to impact national economic planning and government budget priorities (Klingelhöfer *et al*., 2025).

Treatment

To date, there is no specific antiviral therapy widely approved for the treatment of NiV infection (Lv *et al*., 2025). Therefore, clinical management is primarily supportive, aimed at maintaining vital organ function, preventing complications, and improving the patient's chances of recovery (Hassan *et al*., 2025). This approach reflects the limited intervention options that can directly suppress viral replication (Chan *et al*., 2025).

Supportive care includes careful monitoring of neurological and respiratory conditions, as these systems are commonly affected in NiV infection (Skowron *et al*., 2022). Patients with encephalitis require seizure control, intracranial pressure management, and correction of metabolic disorders (Banerjee *et al*., 2019). In cases of respiratory distress, oxygen support or mechanical ventilation are often essential components of intensive care (Levine *et al*., 2025).

Several antiviral agents have been tested in limited use and experimental studies. Ribavirin was used in early outbreaks, but available clinical evidence has not shown consistent effectiveness (Chan *et al*., 2025). Monoclonal antibody-based therapies targeting the viral surface glycoprotein have shown protective effects in preclinical studies, but their application in humans is still under development and further evaluation (Leyva-Grado *et al*., 2024).

In addition to pharmacological treatment, infection prevention measures in healthcare facilities are crucial for case management (Pritchard and Hornsey, 2025). Patient isolation, proper use of personal protective equipment, and implementation of infection control protocols play a significant role in preventing nosocomial transmission, particularly given the potential for person-to-person transmission in certain outbreaks (Orosco, 2023).

Vaccination

Developing a vaccine for NiV is a top priority in prevention efforts, given the high mortality rate and the lack of effective curative therapies (Asokan *et al*., 2026). Vaccination is considered the most sustainable strategy to reduce the risk of outbreaks, protect at-risk populations, and break the chain of transmission, particularly in endemic areas and among groups with high exposure rates (Rahman *et al*., 2024).

Various vaccine platforms have been tested in preclinical stages, including subunit vaccines, viral vectors, and nucleic acid-based vaccines (Lv *et al*., 2025). Most candidates target the NiV surface glycoprotein, which plays a key role in viral attachment and fusion with host cells (Zhou *et al*., 2025). This target was chosen because of its ability to trigger protective humoral and cellular immune responses in animal models (Salleh, 2025).

Preclinical studies have shown that several vaccine candidates can provide protection against viral challenge, either by preventing the onset of clinical disease or by reducing viral replication (DeBuysscher *et al*., 2014). These findings underscore the potential of vaccination as an effective prevention strategy, although the evidence is still limited to animal models and not yet fully applicable to humans (Satapathy *et al*., 2025).

Challenges in developing a vaccine against NiV include limited clinical trial data due to sporadic cases, genetic variation within the virus, and logistical difficulties in vaccine production and distribution (Kim *et al*., 2025). Furthermore, determining the target population for vaccination is crucial, given that NiV typically occurs in small outbreaks and is not endemic in most regions of the world (Bruno *et al*., 2022).

From a public health perspective and the One Health principle, vaccinating intermediate animal hosts or high-risk populations could be an additional strategy to reduce the likelihood of virus transmission to humans (Singhai *et al*., 2021). While its implementation is still limited, this approach represents a more proactive and integrated prevention effort (Halpin *et al*., 2026).

Control

Prevention and control of NiV infection require a comprehensive approach, taking into account the zoonotic nature of the virus, the role of natural reservoirs, and transmission patterns in humans and animals (Singh *et al*., 2019). Because therapeutic and vaccine options are still limited, non-pharmacological measures are the primary focus to reduce the risk of outbreaks and their impact (Faus-Cotino *et al*., 2024). Figure 3 shows the main strategies for NiV prevention and control, emphasizing actions to reduce the risk of spillover, strengthening surveillance systems, and breaking the chain of transmission.

Protect Food Sources
Avoid consuming raw date palm sap and always wash or peel fruit that may have been bitten by bats.

Farm Biosecurity
Keep livestock away from bat habitats and report any sudden outbreaks of coughing or "barking" sounds in pigs.

Healthcare Safety
Use Personal Protective Equipment (PPE like masks and gloves when caring for sick individuals to prevent hospital outbreaks.

Figure 3. The prevention shield

Primary prevention aims to reduce the risk of spillover from natural reservoirs to humans and domestic animals (Safdar *et al*., 2024). Key measures include environmental management, limiting contact between fruit bats and livestock, and improving biosecurity practices on farms (Das *et al*., 2020). Furthermore, public education about risky behaviors, particularly regarding the consumption of potentially contaminated raw food products, also plays a crucial role in prevention efforts (Ahmed, 2024).

Effective epidemiological surveillance is essential for NiV control (Aditi and Shariff, 2019). Early detection of cases in both humans and animals allows for rapid action before the virus spreads further. Integrating surveillance systems across sectors, including human health, animal health, and the environment, enhances the ability to identify threats comprehensively and sustainably (Singhai *et al*., 2021).

In outbreak situations, control focuses on interrupting transmission routes (Pritchard and Hornsey, 2025). Measures such as case isolation, contact tracing, and the implementation of infection prevention and control protocols in healthcare facilities are crucial to prevent human-to-human transmission and nosocomial infections (Yeasmin *et al*., 2025). Protecting healthcare workers through the use of appropriate personal protective equipment is a priority to ensure the safe operation of medical services (Verbeek *et al*., 2020).

Animal control also plays a crucial role, particularly when domestic animals serve as intermediate hosts (Singh *et al*., 2019). Measures such as animal movement restrictions, quarantine, and selective culling may be necessary in certain circumstances to halt the spread of the virus (Tyagi *et al*., 2025). These policies need to be accompanied by social and economic support to ensure their acceptance and effective implementation by affected communities (Hossain *et al*., 2025).

Challenges and future directions

NiV management still faces various challenges, from a scientific, operational, and policy perspective (Hafeez *et al*., 2025). The main obstacle is the limited understanding of the virus's spillover mechanisms from natural reservoirs to humans (Mohapatra *et al*., 2024). The complex interactions between environmental factors, land-use change, and human behavior make predictions difficult, making the development of accurate risk models for early prevention challenging (Deka and Morshed, 2018).

Diagnostic challenges remain significant. Limited access to safe and rapid molecular diagnostic facilities in endemic areas leads to delayed case identification (Ganguly *et al*., 2025). Furthermore, the similarity of early symptoms to other infectious diseases often leads to underdiagnosis, which risks further spreading the disease before interventions can be implemented (Bruno *et al*., 2022).

In terms of therapy and prevention, the lack of vaccines and antiviral drugs that have been extensively tested in humans is a major obstacle (Thakur and Bailey, 2019). Although preclinical developments are underway, conducting large-scale clinical trials faces ethical and logistical challenges due to the low number of cases and the sporadic nature of outbreaks (Johnson *et al*., 2024). This situation slows down the process of validating the effectiveness and safety of medical interventions (Tan *et al*., 2024).

The capacity of health systems in at-risk areas remains an ongoing challenge (Singh *et al*., 2019). Limited resources, inadequate protection of healthcare workers, and limited medical facility preparedness can exacerbate the impact of an outbreak (Ukoaka *et al*., 2024). Furthermore, coordination between sectors and countries is often suboptimal, despite the potential for NiV to spread beyond administrative and geographic boundaries (Ganguly *et al*., 2025).

Going forward, research and policy should emphasize a more proactive and integrated preventive approach (Cabuhat *et al*., 2026). Key priorities include strengthening One Health-based surveillance, developing rapid diagnostic platforms for field use, and innovating vaccines and therapies with broad coverage (Garbuglia *et al*., 2023). Furthermore, integrating ecological, virological, and social data within a predictive framework is expected to improve preparedness for potential future spillovers (Branda *et al*., 2025).

Conclusion

NiV is a highly virulent zoonotic pathogen that significantly impacts human and animal health, as well as global health systems. Its complex transmission pattern—involving natural reservoirs, intermediate hosts, and the potential for human-to-human transmission—makes controlling the virus a multidimensional challenge. The high mortality rate and the limited availability of therapies and vaccines emphasize the importance of more effective and sustainable prevention approaches.

A thorough understanding of etiology, epidemiology, clinical manifestations, and public health and economic impacts is crucial for designing appropriate risk mitigation strategies. Evidence shows that NiV control cannot rely solely on clinical interventions but requires an integration of surveillance, biosecurity practices, public education, and health system preparedness.

The One Health approach is the most appropriate framework for addressing the NiV threat, given the close relationship between human, animal, and environmental health. Strengthening cross-sector collaboration, continued investment in vaccine and therapeutic research, and improving early detection capacity are crucial steps to reduce the risk of future outbreaks. With a coordinated, evidence-based strategy, the impact of NiV can be significantly reduced.

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Conflict of interest

The authors declare that there is no conflict of interest.

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