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Review

Nipah Virus Infection: An Overview of Current Knowledge

Sevil Akbulut Zencirci

Department of Public Health, Faculty of Medicine, Bilecik Şeyh Edebali University, Bilecik, Türkiye

Abstract

Nipah virus (NiV) is a bat-borne zoonotic virus capable of causing severe clinical outcomes and human-to-human transmission. First identified in Malaysia in 1998, it has caused recurrent outbreaks, particularly in Bangladesh and India. NiV can be transmitted through contact with infected animals, consumption of contaminated food, and close contact with infected individuals. Its ability to cause severe encephalitis and respiratory disease in both humans and animals may lead to rapid clinical deterioration. Reported epidemiological and clinical variations across regions reflect the dynamic nature of the infection. The presence of human-to-human transmission and the persistence of sporadic cases indicate the potential for spread beyond traditionally endemic areas. As there is currently no specific antiviral therapy or approved vaccine available, preventive and control measures remain essential. Given its zoonotic origin and complex transmission pathways, integrated approaches addressing human, animal, and environmental health are necessary. This review aims to summarize the existing literature on NiV and contribute to ongoing research and control efforts.

Keywords: Epidemiology, Nipah virus, Prevention

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In recent years, the intensification of interactions among humans, animals, and the environment has increased the risk of the emergence and re-emergence of zoonotic pathogens with high pathogenicity and epidemic potential, thereby posing significant challenges to global public health systems.[1] Indeed, the World Health Organization (WHO) emphasizes that outbreaks caused by zoonotic viruses such as Ebola, Nipah, and Lassa may have serious implications for global health security.[1] Among these pathogens, Nipah virus (NiV) stands out as a particularly concerning threat due to its high case fatality rates, zoonotic origin, and capacity for human-to-human transmission.[2,3]

NiV is a zoonotic RNA virus belonging to the family *Paramyxoviridae* and the genus *Henipavirus*, with fruit bats serving as its natural reservoir.[3] The virus was first identified in 1998 in Malaysia in serum samples obtained from patients presenting with encephalitis, and it was named after the village of Kampung Sungai Nipah, where these cases were reported.[4,5] In subsequent years, NiV infection has been associated with recurrent outbreaks in South and Southeast Asia, particularly in Bangladesh and India, with cases reported almost annually in some regions.[6,7] Laboratory-confirmed cases reported in January 2026 in the state of West Bengal, India, and in February 2026 in Bangladesh have demonstrated that the infection further illustrate the ongoing and regionally sustained nature of NiV activity.[8,9]

High mortality rates, the absence of specific treatment and a licensed vaccine, and the wide geographic distribution of its natural reservoir render NiV a threat that requires preparedness not only in affected countries but also in regions where no outbreaks have yet been reported.[7] Recognizing

Address for correspondence: Sevil Akbulut Zencirci, MD. Department of Public Health, Faculty of Medicine, Bilecik Şeyh Edebali University, Bilecik, Türkiye
E-mail: sevil.zencirci@bilecik.edu.tr
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these risks, the WHO has classified NiV among its priority diseases under the Research and Development Blueprint and has recommended strengthening early diagnosis, surveillance, infection prevention and control, and community-based prevention strategies.[10,11]

In this review, the transmission dynamics, epidemiology, clinical course, and preventive approaches related to NiV are examined in light of the current literature.

Natural Reservoir and Animal Hosts

The primary natural reservoir of NiV consists of fruit bats belonging to the family *Pteropodidae*, with species of the genus *Pteropus* playing a central role in the ecological cycle of the infection.[12] Although these bats are infected with NiV, they generally do not exhibit overt clinical signs of disease; the virus can be shed into the environment through saliva, urine, and feces, and the predominantly asymptomatic course of infection enables sustained viral circulation within the natural reservoir.[3]

Fruit bats of the genus *Pteropus* are distributed across a broad tropical and subtropical region extending from the South Pacific islands and Australia to Southeast Asia, South Asia, and Madagascar, and the emergence of NiV in different regions is closely associated with this geographic distribution. These bats commonly inhabit areas near human settlements and pose a risk of exposure to domestic animals and humans due to their feeding on fruits collected from cultivated date palm trees or on fresh flowing date palm sap.[12]

In endemic regions, outbreaks largely coincide with the date palm sap collection period and are associated with contamination of sap collection containers by bats through licking the sap and contaminating it with urine or feces.[13] It has been reported that viral shedding increases particularly during the winter months and during bat pregnancy periods; the overlap of this period with the date palm sap harvesting and consumption season in endemic areas is considered to influence the seasonal distribution of outbreaks.[7]

Beyond fruit bats as the natural reservoir, NiV has been reported to cause infection in certain animal species serving as intermediate hosts. In particular, pigs are among the most frequently identified animal species associated with NiV infections; the infection may present with respiratory and central nervous system manifestations, while in some cases clinical signs may be mild or nonspecific and therefore remain unrecognized.[3,7]

Furthermore, NiV infections have also been reported in horses, dogs, cats, and certain livestock species.[3,7] During the 2014 outbreak reported in the Philippines, infections and deaths were documented in horses, and infections were also reported in dogs and cats.[14] In Bangladesh, serological evidence of exposure has been reported in cattle and goats.[15]

Transmission

Transmission in NiV infection is shaped by ecological dynamics characterized by intensified human–animal–environment interactions. The proximity of bat habitats to human settlements, the expansion of agricultural activities, deforestation, and environmental changes that increase wildlife–human contact are among the principal ecological determinants that elevate the risk of viral spillover from its natural reservoir to intermediate hosts and humans.[16,17]

NiV infection is a zoonotic disease with multiple routes of transmission. The virus can be transmitted from animals to humans through direct contact, through the consumption of food contaminated by fruit bats, or, under certain conditions, via human-to-human transmission.[2]

Animal-to-human transmission was reported as the primary route of infection during the initial outbreaks in Malaysia and Singapore, where NiV was first identified. In these outbreaks, the majority of human infections developed following close contact with infected pigs, and transmission was thought to occur particularly through unprotected exposure to respiratory secretions, urine, and contaminated tissues.[7] It was demonstrated that fruit bats of the genus *Pteropus* constitute the natural reservoir and that the virus was transmitted to pigs via fruits contaminated by bats.[16,18]

During the outbreak reported in the Philippines in 2014, human cases were documented following contact with infected horses, exposure to contaminated body fluids during slaughter, and consumption of meat from sick horses, indicating that horses may also serve as potential intermediate hosts.[14]

In outbreaks reported in Bangladesh, fruits and fruit products contaminated with the urine or saliva of fruit bats have been identified as the primary source of animal-to-human transmission. In particular, the consumption of raw date palm sap harvested between December and March in the west-central regions of Bangladesh has been shown to be the most common source of primary infections reported in the country.[19] Field studies have demonstrated that fruit bats access date palm trees at night to consume the flowing sap and contaminate collection containers with their saliva or feces.[20]

Human-to-human transmission has been documented particularly in outbreaks in Bangladesh and India and has occurred mainly through close contact with the bodily fluids of infected individuals. This mode of transmission has been

observed predominantly among family members, caregivers, and healthcare workers, with prolonged and close contact with infected individuals reported to increase the risk of transmission.[21] In addition, nosocomial transmission has been reported in healthcare settings, especially during aerosol-generating procedures such as intubation or in situations involving inadequate use of personal protective equipment.[22]

Epidemiology

The epidemiology of NiV infections exhibits a dynamic pattern shaped by the interaction of numerous environmental, sociocultural, and health system–related factors that vary across temporal and spatial contexts.[6,19]

Malaysia/Singapore

In 1998, cases characterized by fever, headache, and altered consciousness were reported among pig farmers in the Kinta region; following the detection of Japanese Encephalitis (JE) IgM positivity in four cases, the outbreak was attributed to JE, and mosquito control measures along with vaccination campaigns were implemented. However, these interventions proved insufficient to halt the outbreak.[23–25]

Between 1998 and 1999, a large-scale outbreak occurred in Malaysia, predominantly affecting adults and exhibiting epidemiological characteristics inconsistent with those typical of JE.[23,25] Detailed virological investigations identified the causative agent as a novel paramyxovirus, which was subsequently named NiV.[23,24] Following its identification, extensive control measures were implemented, including the culling of all pigs considered infected, burial of carcasses with lime, strict disinfection procedures in farms and slaughterhouses, and mandatory use of personal protective equipment for personnel in contact with animals; these interventions successfully brought the outbreak under control.[26,27] The virus was isolated from the cerebrospinal fluid of patients in April 1999 and was named NiV after the locality where it was first identified.[4,28,29] Between September 1998 and December 1999, a total of 283 cases were reported in Malaysia, of which 109 resulted in death.[16]

Following the emergence of NiV in Malaysia, the infection was introduced into Singapore through the importation of live pigs. In March 1999, 11 abattoir workers were hospitalized with encephalitis and pneumonia, and one case resulted in death. All affected individuals had contact with infected pigs imported from farms involved in the Malaysian outbreak, and virological analyses demonstrated that the causative agents in the two countries were genetically identical.[30] Case–control studies identified exposure to the urine and feces of infected pigs as the primary risk factor.[31] Serological screening detected evidence of infection in 22 (1.5%) of 1,469 at-risk individuals, some of whom remained asymptomatic.[32] The outbreak was brought under control following the ban on pig imports and the closure of the implicated abattoirs, and no further cases were reported thereafter.[30]

Bangladesh

In April–May 2001, 13 cases of encephalitis were reported in the Meherpur region of Bangladesh, nine of which resulted in death. Following the exclusion of other common etiologies such as JE, dengue, and malaria, serological analyses identified reactive antibodies against NiV in two serum samples, leading to the recognition of these cases as the first documented NiV infections in Bangladesh.[33]

In subsequent years, NiV infections were reported regularly in Bangladesh and were characterized by high mortality rates. In 2003, 12 cases (eight deaths) were reported in Naogaon, and in 2004, 36 confirmed cases with a 75% mortality rate were documented in Faridpur; evidence of human-to-human transmission was identified in these outbreaks.[34,35] The 2007 outbreak in Thakurgaon further supported transmission through close contact and exposure to respiratory secretions.[19,36]

In the following years, cases were predominantly reported in clusters during the winter months (December–April) and were associated with the consumption of raw date palm sap. This pattern indicates that date palm sap contaminated by fruit bats (*Pteropus* spp.) constitutes the principal route of transmission, with limited human-to-human transmission also occurring.[34,37–39]

Most recently, on 3 February 2026, a confirmed NiV case from the Rajshahi region in northwestern Bangladesh was reported to WHO. The case involved a woman aged 40–50 years residing in Naogaon district, who died following symptom onset on 21 January; the diagnosis was confirmed by PCR and ELISA. The patient had a history of consuming raw date palm sap prior to symptom onset; 35 contacts were monitored, and no additional cases were identified.[8]

India

Over the past approximately two decades, NiV has caused recurrent outbreaks in India. The first outbreak occurred in January–February 2001 in Siliguri, West Bengal, where 66 cases were identified, and approximately two-thirds of the cases resulted in death. The outbreak was largely characterized by nosocomial transmission, with a substantial proportion of cases occurring among healthcare workers and hospital contacts. Molecular analyses demonstrated that the Siliguri strain was genetically more closely related to strains circulating in Bangladesh than to Malaysian isolates.

The findings indicate that human-to-human transmission, particularly through contact with patients' respiratory secretions and other bodily fluids, played a significant role in the outbreak.[22,40]

In 2007, an outbreak occurred in the Nadia district of West Bengal, during which five cases were reported, all of which resulted in death. The index case had a history of consuming palm wine suspected to have been contaminated by bats; subsequent cases demonstrated evidence of human-to-human transmission.[41,42] During the outbreak that emerged in Kerala state in 2018, a total of 23 cases were identified, 18 of which were laboratory-confirmed, and 21 deaths were reported. The outbreak spread largely within healthcare settings; transmission was shown to occur through direct or indirect contact with the index case and via respiratory secretions.[43,44]

The outbreak that occurred in Ernakulam in 2019 was rapidly brought under control through isolation of the index case and monitoring of approximately 300 contacts.[45,46] In 2021, a 12-year-old child in Kozhikode died due to NiV infection that developed after the consumption of wild fruits.[47] In September 2023, another outbreak in the same region resulted in six confirmed cases and two deaths; the cases were identified as close contacts of the index patient.[48]

Between May and July 2025, a total of four confirmed NiV cases were reported in the Malappuram district of Kerala and, for the first time, in Palakkad district; two of these cases resulted in death. The cases were reported to be epidemiologically unrelated, suggesting possible independent zoonotic spillover events. Notably, the presence of *Pteropus* fruit bats, the natural reservoir of NiV, has been documented in the affected regions.[49]

Most recently, in January 2026, confirmed NiV infections were reported in two healthcare workers employed at the same hospital in the state of West Bengal, India. One of the cases was managed in intensive care, while clinical recovery was observed in the other. Approximately 190 contacts were monitored, no additional cases were identified, and surveillance and infection prevention and control measures remain ongoing.[9]

Philippines

The NiV outbreak that occurred in 2014 on Mindanao Island in the Philippines was reported in association with horse slaughter and the consumption of horse meat. A total of 17 suspected cases and two deaths were recorded, and anti-NiV IgM positivity was detected in three patients.[14,50] Analyses indicated that the responsible strain was genetically more closely related to Malaysian isolates, and it was determined that 10 cases were associated with horse slaughter or consumption, while five cases—including two healthcare workers—were linked to contact with infected patients.[14]

Pathogenesis

The principal route of entry for NiV infection is oro-nasal exposure; however, early stages of viral replication have not been fully elucidated, as most data derived from human tissues originate from advanced-stage cases. Nevertheless, the detection of high levels of viral antigen in lymphoid tissues as well as in alveolar and vascular structures of the respiratory system suggests that initial viral replication may occur in these regions.[51]

Early viremia enables the virus to enter the systemic circulation and disseminate to various tissues, particularly the vascular endothelium. Vascular injury associated with endothelial involvement has been reported to contribute to systemic spread and organ dysfunction in NiV infection.[51] Attachment of NiV to host cells occurs through the interaction of the viral G glycoprotein with ephrin-B2 and ephrin-B3 receptors. The high expression of these receptors in brain and lung tissues, as well as in vascular structures, is considered one of the principal mechanisms underlying the neurological and respiratory manifestations of the infection.[52] The high degree of conservation of the ephrin-B2 receptor across different mammalian species further supports the broad host range of the virus.[53,54]

Central nervous system involvement may develop through both hematogenous dissemination and direct invasion via cranial nerves. Experimental animal models have demonstrated that the olfactory nerve pathway, in particular, represents an important early route of invasion.[55] The severe clinical course of NiV infection is partly attributed to the virus's capacity to suppress the innate immune response. In particular, products of the P gene have been shown to inhibit the interferon response and weaken antiviral defense mechanisms.[56,57]

The genetic diversity of NiV partially explains the observed differences in clinical and epidemiological characteristics. It has been suggested that NiV strains circulating in pigs in Malaysia are genetically distinct from those identified in Bangladesh and India, and that these strains may have diverged genetically within natural reservoir populations across different geographic regions.[41,58–60]

Clinical Features

NiV infection primarily manifests with acute encephalitis and respiratory system involvement.[42] Case fatality rates may reach 40–75%, and the incubation period generally ranges from 3 to 14 days, although it may extend up to 45 days in rare cases.[2] In the early phase of infection, nonspecific symptoms such as fever, headache, myalgia, and vomiting are commonly observed.[61] Approximately 6–11% of infec-

tions may remain asymptomatic;[31,62] however, in symptomatic cases, the clinical course may progress rapidly. As the disease advances, severe pneumonia, acute respiratory distress syndrome (ARDS), and fatal encephalitis may develop.[61]

Regional differences in clinical course and mortality have been observed. In outbreaks reported in Bangladesh and India, mortality rates have been higher (exceeding 70%) compared with those in Malaysia, and respiratory involvement has been reported to be more prominent.[22,63] In contrast, during the Malaysian outbreaks, lower mortality rates were observed, and respiratory manifestations were reported to be more limited.[64]

Neurological involvement is observed in a substantial proportion of cases, with headache, confusion, and altered consciousness among the initial manifestations. In later stages, seizures, coma, and focal neurological deficits may develop;[51,65] severe disease, particularly in cases presenting with prominent neurological symptoms, may result in cerebral edema (encephalitis) and death.[2]

Although a substantial proportion of patients who experience acute encephalitis recover completely, long-term neurological sequelae—such as cognitive impairment, cranial nerve palsies, ataxia, and behavioral changes—have been reported in some cases.[66] In addition, clinical manifestations associated with systemic vasculitis and multiorgan involvement have been described, affecting cardiac, renal, and other organ systems.[65,67]

Diagnosis

Because the clinical manifestations of NiV infection may resemble those of other febrile illnesses, viral encephalitides, and severe pneumonia, clinical differentiation can be challenging. Therefore, early diagnosis is critical both for appropriate patient management and for the control of potential outbreaks.[68] NiV infection should be suspected in individuals presenting with acute encephalitis syndrome or severe acute respiratory infection who have a history of residence in endemic areas or possible exposure.[7]

Definitive diagnosis is established through laboratory methods. In the presence of clinical and epidemiological suspicion, the combined evaluation of molecular and serological tests is recommended. RT-PCR is the primary diagnostic method for detecting viral RNA in respiratory specimens, blood, or cerebrospinal fluid and is particularly prioritized during the acute phase. For serological diagnosis, the detection of IgM and IgG antibodies by ELISA is employed; these methods are used for both diagnostic and surveillance purposes.[69,70]

Specimens should be collected as early as possible, using appropriate personal protective equipment and in accordance with biosafety protocols. NiV is an agent requiring high-level biosafety measures, and handling non-inactivated specimens necessitates advanced laboratory infrastructure.[2,7]

Treatment

Currently, there is no licensed specific antiviral therapy or vaccine for NiV infection; therefore, clinical management largely relies on early diagnosis and effective supportive care. The classification of NiV among priority pathogens under WHO's Research and Development Blueprint has contributed to accelerating therapeutic and vaccine development efforts.[7,10]

The current therapeutic approach focuses on the timely recognition and appropriate management of respiratory failure, neurological complications, and hemodynamic instability, particularly in severe cases. Maintenance of fluid–electrolyte balance and provision of adequate nutritional support play a decisive role in determining prognosis. In addition, close monitoring of complications such as cerebral edema and pneumonia is essential, with advanced life-support interventions—including oxygen therapy, mechanical ventilation, and renal replacement therapy—implemented when indicated.[2]

Evidence regarding specific antiviral agents remains limited. Ribavirin has been administered empirically in some outbreaks, and observational studies have reported a possible reduction in mortality; however, robust and controlled clinical data confirming its efficacy are lacking. Similarly, remdesivir and various monoclonal antibodies have been evaluated primarily in experimental models or early-phase studies and are not part of routine clinical use.[3]

Prevention

Although there is currently no licensed vaccine for humans against NiV, several candidate vaccines—including virus-like particle–based, adenoviral vector–based, recombinant vesicular stomatitis virus (rVSV)–based, and mRNA-based platforms—have demonstrated promising results in animal studies.[71–78] Nevertheless, no approved vaccine or specific therapy against NiV is currently available; therefore, the primary strategy at present is to strengthen measures aimed at interrupting transmission chains.[7] Communication and community engagement strategies that support the adoption of preventive practices may enhance the feasibility and sustainability of these measures in the field.[79,80]

Prevention of Bat-to-Human Transmission

Since *Pteropus* bats constitute the natural reservoir of NiV, preventing transmission from bats to animals and humans is of critical importance. In particular, measures to prevent bat access to date palm sap and avoidance of consuming

raw sap that may be contaminated are among the primary preventive strategies.[17,81,82]

Date palm sap collectors hang containers to collect the flowing sap from trees; however, these containers may often become contaminated with bat excreta. In some regions, collectors filter the sap using nets or cloth before sale, but there is no evidence that this practice eliminates viral contamination.[83] Covering the sap collection area of date palm trees with a physical barrier known as a skirt has been shown to prevent bats from accessing the sap.[84,85] Additionally, washing or peeling fruits before consumption and maintaining proper hand hygiene during preparation are important preventive measures.[86]

Prevention of animal-to-human transmission

In the outbreaks in Malaysia and Singapore, the demonstration that human infections were associated with close contact with infected pigs, exposure to their secretions and excreta, and contact with contaminated materials such as raw pork underscored the critical importance of controlling animal contact in preventing infection.[30,62] Accordingly, direct contact with infected animals should be avoided, and gloves and appropriate protective clothing should be used during contact with sick animals, animal tissues, and slaughtering procedures. Furthermore, when planning new pig farms in endemic regions, feed sources and housing structures should be designed to prevent access by fruit bats.[7]

Prevention of Human-to-Human Transmission

In controlling NiV infection, avoiding unprotected and close contact with infected individuals is essential. Attention should be paid to hand hygiene after patient care or visits; contact with blood and other bodily fluids should be minimized, and exposure should also be reduced during funeral practices.[2,7]

During case investigation, potential contacts should be identified; based on risk assessment, contacts should be isolated as appropriate, monitored throughout the incubation period, and those who develop symptoms should be promptly isolated, tested, and provided with appropriate supportive care.[68]

In healthcare settings, suspected or confirmed cases should, if possible, be isolated in single rooms, and contact and droplet precautions should be implemented. Appropriate masks, eye protection, gowns, and gloves should be used during patient care; respirators should be preferred during aerosol-generating procedures.[2,7] Considering reported nosocomial transmission and infections among healthcare workers, strict adherence to infection prevention and control measures is imperative.[87]

One Health Approach: Control of Infection in Animals

As illustrated by NiV, a substantial proportion of emerging infectious diseases today are of zoonotic origin, posing significant threats to global health security and economic sustainability. The Marburg virus outbreak reported in Rwanda, the widespread transmission of mpox across multiple regions, and the COVID-19 pandemic have underscored the importance of understanding zoonotic spillover dynamics and maintaining institutional collaboration among human, animal, and environmental health sectors during inter-epidemic periods.[12,88–91]

NiV outbreaks demonstrate that animal–human–environment interactions constitute a central axis in the emergence and spread of the disease. Practices such as deforestation, urbanization, prolonged droughts, forest fires, and pig farming integrated with agricultural activities have facilitated transmission chains by increasing contact between humans and potential reservoirs or intermediate hosts.[16,17,92] In this context, rapid quarantine of animal facilities in suspected outbreaks, controlled and safe culling of infected animals, and restriction of animal movement are among the primary intervention measures.[7] Experiences from previous outbreaks associated with pig farms have shown that regular cleaning and disinfection practices, strict control of farm entry and exit, and strengthened biosecurity measures are effective in controlling transmission.[2] Furthermore, considering the epidemiological roles of pigs and fruit bats, the implementation of integrated animal health and wildlife surveillance systems is essential for strengthening early warning mechanisms and response capacity in line with the One Health approach.[7]

Conclusion

NiV remains among the zoonotic pathogens of continued global health security concern due to its high case fatality rate, broad host range, and potential for human-to-human transmission. Outbreak patterns reported from different countries indicate that NiV infection does not follow a fixed or uniform epidemiological model; rather, it demonstrates variable dynamics depending on ecological conditions, the presence of intermediate hosts, and sociocultural practices. Moreover, considering increasing global mobility and documented cases of human-to-human transmission, the possibility of geographic spread of the infection cannot be entirely excluded.

In this context, it is important to maintain early warning systems, integrated human–animal surveillance, and infection prevention and control capacity not only in endemic regions but also in countries where no cases have yet been reported. The absence of a specific antiviral therapy

and a licensed vaccine currently makes preventive measures aimed at interrupting transmission the cornerstone of control strategies. Early diagnosis, contact tracing, and supportive care remain key intervention tools in outbreak management.

The experience with NiV highlights the importance of addressing the relationship between ecosystems, animal health, and human health in a holistic manner in the management of zoonotic threats. Maintaining sustainable preparedness capacity during inter-epidemic periods and supporting research and development efforts are crucial for mitigating the impact of potential future outbreaks.

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