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Understanding Nipah Virus: Epidemiology, Transmission, Clinical Impact, and Emerging Therapeutics

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Abstract

The Nipah virus (NiV), an emerging zoonotic pathogen with high mortality rates and major consequences for public health, is the cause of occasional but severe outbreaks in Southeast Asia. It can transmit from person to person and is mostly carried by fruit bats, who are its natural reservoir. 1998 saw the first identification in Malaysia. Human infections can cause anything from minor respiratory problems to deadly encephalitis, with an average case fatality rate of more than 50%. Because of its high lethality and the absence of specialized antiviral therapies or vaccinations, the virus is categorized as a Biosafety Level 4 (BSL-4) pathogen. Human NiV infections can cause a wide range of clinical signs, from deadly encephalitis and severe respiratory sickness to no symptoms at all. The Nipah virus poses a major threat to public health, especially in endemic areas like Southeast Asia, with a case fatality rate that varies across outbreaks but typically ranges from 40% to 75%. Developing efficient therapies and preventative measures requires an understanding of the pathophysiology of the Nipah virus. The virus causes widespread vascular damage, inflammation, and cellular death when it enters the human body and targets endothelium cells, neurons, and respiratory epithelial cells. Complex clinical symptoms resulting from this multi-organ involvement necessitate a multidisciplinary approach to treatment. Since there are currently no licensed antiviral medications or Nipah virus-

specific vaccinations, the only available treatment options are supportive care. However, more investigation is required to confirm these results in human populations. However, prompt identification and containment efforts are complicated by the intermittent character of epidemics and the quick progression of disease in patients. Preventive measures continue to be the most effective way to limit Nipah virus epidemics in the absence of specialized treatments. These include creating public health policies that incorporate a One Health approach that addresses the relationships between human, animal, and environmental health, educating at-risk populations about avoiding contact with bats and their secretions, and putting infection control measures in place in healthcare settings. In order to lessen the threat of the Nipah virus and stop future outbreaks, this review emphasizes the necessity of coordinated scientific efforts and public health actions.

Keywords: Nipah virus, Zoonotic infection, Respiratory illness, Encephalitis, Transmission, Outbreak.

Highlights

- The Nipah virus is a zoonotic disease that can cause severe and fatal encephalitis.
- It is native to Southeast Asia and the western Pacific; outbreaks have been documented in Bangladesh, Malaysia, Singapore, the Philippines, and India.
- Although neurological symptoms are often common, it can also result in recurring, late-onset encephalitis.
- Risk factors for transmission include contact with fruit bats, the virus's reservoir host, known intermediate hosts including pigs, or known Nipah virus patients.
- The Nipah virus (NiV), a WHO priority pathogen endemic to Southeast Asia, is now diagnosed using in-house laboratory-based serological and nucleic acid amplification techniques.

Since there are currently no licensed medications or vaccines to stop the spread of this potentially pandemic virus, prevention and education are crucial.

1. Introduction

The Nipah virus (NiV) is an emerging zoonotic virus that is known to be extremely deadly and capable of causing pandemics and epidemics. It is a negative-sense, single-stranded RNA virus that can infect humans and other mammals. It is a member of the genus Henipavirus and family Paramyxoviridae.[1] Infected pigs served as the intermediate amplifying hosts during the 1998–1999 pig farmer pandemic in Malaysia and Singapore. Since then, repeated outbreaks with varying transmission patterns and case fatality rates have mostly been documented in South and Southeast Asian nations, particularly Bangladesh and India. [2] Unlike the Malaysian outbreaks, the outbreaks in Bangladesh and India have demonstrated substantial human-to-human transmission, raising concerns about the potential for the virus to spread more broadly. The danger of zoonotic spillover and disease onset has also significantly increased due to environmental changes, including urbanization, deforestation, and more human-animal contact. Pteropus-swine-man, human contagion through eating contaminated food, and inter-human contagion are the many mechanisms of transmission; direct bat-human infection is also postulated. The only actions that may be taken when pig outbreaks arise are isolation, movement restrictions, and the death of affected animals. [3, 4] Close proximity, such as touching, feeding, or visiting an infected person, increases the risk of droplet NiV infection. NiV droplets (aerosol exposure) may be involved in the transmission of NiV during close contact, according to recent experimental research using aerosolized NiV in Syrian hamsters.[5] In addition to conducting serological tests and assays to amplify viral nucleic acids, isolation of the virus can confirm both human and animal NiV infections. Both NiV isolation and propagation need biosafety level-4 (BSL-4) laboratory facilities.[1] Between 1998 and 1999, an outbreak of neurological and respiratory symptoms of febrile encephalitis caused by NiV was recorded in 246 people in Singapore and Malaysia, as well

as in pigs raised for food.[6]A total of 639 human cases of NiV infection have been reported from Bangladesh (261 cases), India (85 cases), Singapore (11 cases), the Philippines (17 cases), and Malaysia (265 cases), with a mortality rate of approximately 59%.[7] Notably, outbreaks among the livestock population cannot be prevented in areas where date palm sap contamination plays a significant role in the spread of NiV infection. However, if livestock immunization is made inexpensive, it might work in some areas.[7] Numerous vaccine candidates, including vectored and subunit vaccines that provide protective immunity, have been found through extensive research encompassing preclinical trials in a variety of animals and nonhuman primates. One vectored vaccination that uses the vesicular stomatitis virus has demonstrated protection against hamsters, African green monkeys, and inferrets[10]. Despite these advancements, the academic community continues to struggle with funding for human clinical studies of potential vaccinations. Despite the high death rate, pharmaceutical corporations are reluctant to fund research on the creation of vaccines for rare diseases like Nipah. Human vaccination is a crucial component of preventing NiV infection. Vaccination of livestock (particularly pigs and likely horses) in endemic areas is another aspect of prevention.[8] This review goes into great detail about the biology of NiV, its transmission and epidemiology, pathology, and advancements in diagnosis, vaccine design, and appropriate prevention and control studies that should be used to combat this emerging pathogen.[11]

2. Molecular Biology of the Nipah virus (NiV)

NiV is an enveloped pleomorphic virus that is a member of the Paramyxoviridae family's genus Henipavirus.

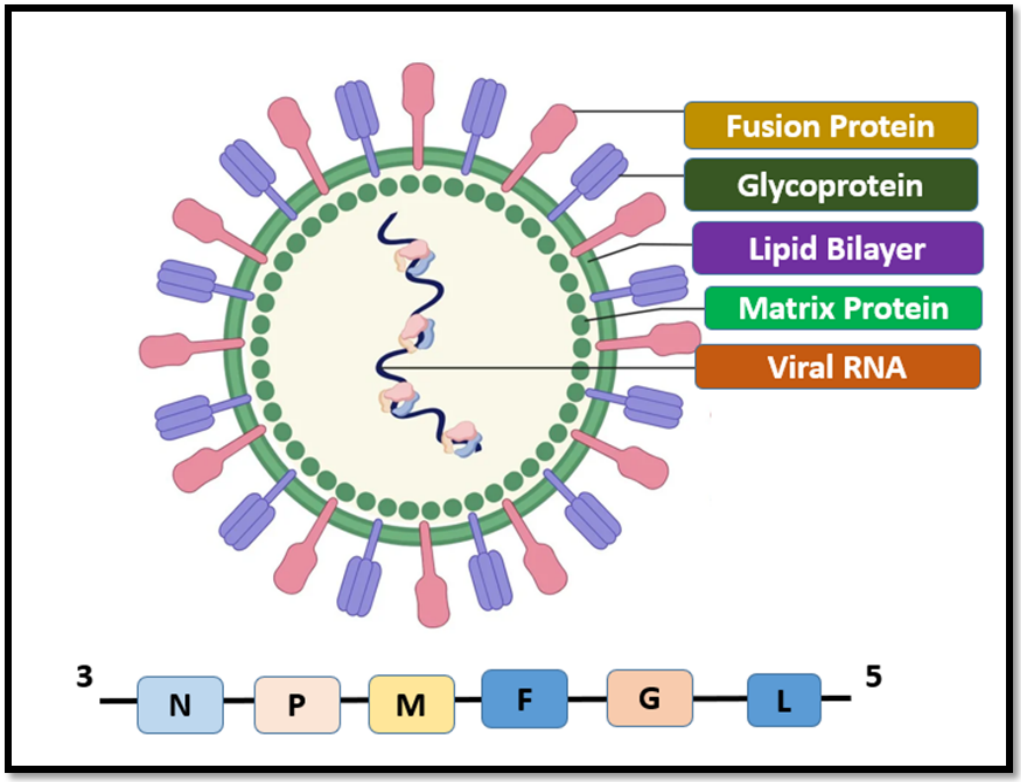

Figure: 1 illustration of the anatomy of the Nipah virus (NiV). The ribonucleoprotein complex, which is made up of the RNA genome linked to the N, P, and L proteins, is encased in a lipid bilayer envelope that is studded with two envelope glycoproteins, G and F. Six structural proteins- N, P, M, F, G, and L- are encoded by the RNA. The inner portion of the envelope is associated with the matrix protein M. The lipid bilayer, RNA, and structural proteins are shown in various colors.[14]

Fig 1. Nucleocapsid (N), phosphoprotein (P), matrix protein (M), fusion protein (F), glycoprotein (G), and RNA polymerase (L) are the six structural proteins that are encoded by the virus's non-segmented negative sense single-stranded RNA, which is roughly 18.2 kb long. The ribonucleoprotein complex, which is essential for controlling transcription and viral RNA production, is made up of the N, P, and L as well as the viral RNA. The F and G proteins that span the envelope12 regulate attachment and entrance into the host cell. While the F protein causes viral-cell membrane fusion to enable the entry of viruses, the G protein promotes virus attachment and binds to the host cells Ephrin-B2 and -B3 receptors of virion [13,14].

3. Pathogenesis of Nipah Virus Infection

The infectious disease known as NiV, or Nipah virus, is first spread from animal to human and from human to human. Bats and pigs can transmit this type of illness. Humans can contract the disease if they eat fruits that have already been tainted by a sick pig or bat[15]. People, especially kids, eat a variety of fruits in the summer, including litchis and mangoes. Fruits on trees may be bitten by bats at night and then sold in the market the following morning, making it challenging to verify whether or not the fruits in the trees were consumed by bats [16]. These animals' saliva and urine can also transmit the virus to humans. Therefore, fruits or sap tainted with an infected animal's urine or saliva can also be a powerful way to spread the virus.[17] Additionally, bats can infect humans when climbing trees by leaving their saliva or secretions on them. This infection can be tested by determining the antigen, isolating the virus, and employing serology. Detection can be done via histopathology. Numerous tissues, blood, and respiratory secretions have been used to detect the Nipah virus in pigs. It is also possible to test for the virus in feline blood, urine, and respiratory secretions. However, dogs' brain, spleen, liver, lung, kidney, adrenal gland, and other organs are infected with the virus.[18] The route that causes the disease is described in **Fig. 2** Nipah virus disease-causing pathway.

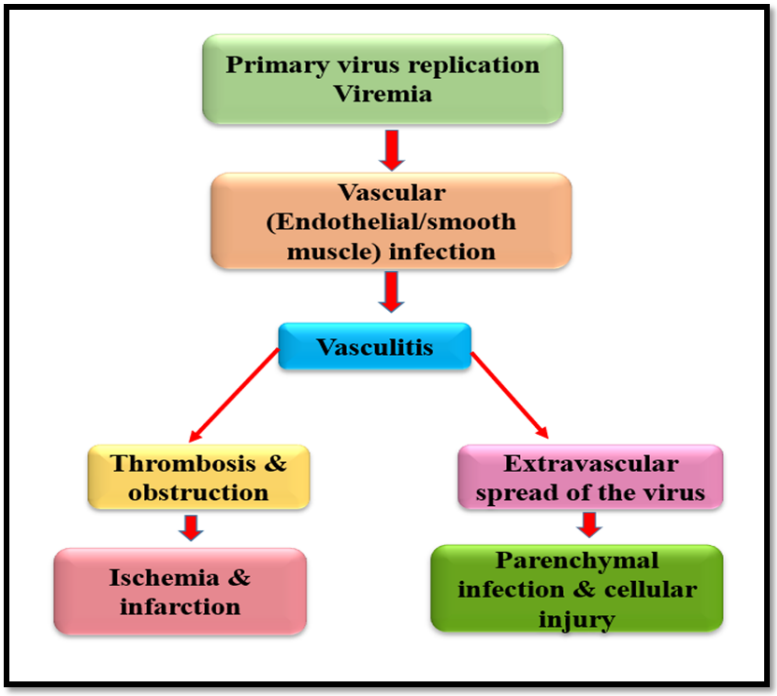

Figure 2: Demonstrates how primary viral replication results in viremia, which makes it possible for smooth muscle and vascular endothelial cells to become infected. The ensuing vasculitis results in extravascular viral transmission, thrombosis, ischemia, infarction, parenchymal infection, and cellular damage.[19]

One of the main ways that the Nipah virus spreads from person to person is through human respiratory secretions.[19] Sneezing, coughing, urine, saliva, and other bodily fluids can transmit the infection. The fusion (F) and attachment (G) proteins regulate cellular connections. The newly generated precursor (F) protein (F0) cleaves two components, namely F1 and F2, by the host protease. The F1 subunit contains the virus's merging peptide, which aids in the binding of the virus to the host cell.[20] The M protein of the virus plays a role in both budding and morphogenesis. NiV infectivity can be neutralized by antibodies against G proteins.[21] The enveloped Henipavirus, which contains NiV, naturally invades target cells after binding because of the coordinated efforts of glycoprotein fusion and attachment. NiV glycoprotein (G) in host cells undergoes conformational changes as a result of interactions with class B ephrin (viral receptor), which activates and fuses the membrane of F glycoprotein.[22] The host ephrin B2/3 receptor is bound by the Nipah G protein, which accelerates the G protein's conformational shift that results in F protein refolding.[23]

4. Clinical Presentation in Humans and Animals

The duration of incubation varies from four days to two weeks. A tiny percentage of patients may appear with encephalitis, pulmonary involvement, or an asymptomatic illness.[24] NiV was detected in the Malaysian outbreak patients, and it was discovered that traces of NiV were found in urine samples in addition to saliva and throat swab samples. Therefore, exercise caution when using the restroom with someone who is afflicted.[25]**Fig 3.** The illness with fever and headache for 3–14 days, followed by the opposite symptoms, indicates exposure and an incubation period of 5–14 days.[26] Sleepiness, fever, headache, and bodily discomfort are the initial symptoms, which are followed by confusion and disorientation. In as little as 24 to 48 hours, these symptoms may worsen and result in a coma.

The worst side effect of a NiV infection is encephalitis. Early in the infection, respiratory illness may also be present. [27] Nipah case patients who have respiratory issues are more prone to spread the infection than those who do not. In the setting of an epidemic outbreak, symptomatic individuals are suspected of having the sickness. Fever, nausea, headaches, coughing, sore throats, and muscle aches are among the first signs in humans. [28] Septicemia can also result in gastrointestinal bleeding and renal system damage. Additionally, some patients may have breathing problems, which can vary from acute respiratory disturbance to atypical pneumonia. [28] Seizures, sleepiness, disorientation, mental confusion, and certain neurological indicators of encephalitis are among the severe symptoms that follow. Within a few days, the illness may cause a coma and death. [29] According to statistical research, this viral disease has a death rate of between 40 and 75 percent. Some long-term negative effects, such as convulsions, personality abnormalities, and other neurological diseases, have been observed in Nipah virus survivors.[30] Porcine respiratory and encephalitis syndrome is another term for this illness in pigs. In Malaysia's peninsula, it is also known as "one-mile hack" or "barking pig syndrome." It has been found that pigs younger than six years old are more likely to acquire an acute feverish sickness. [31]

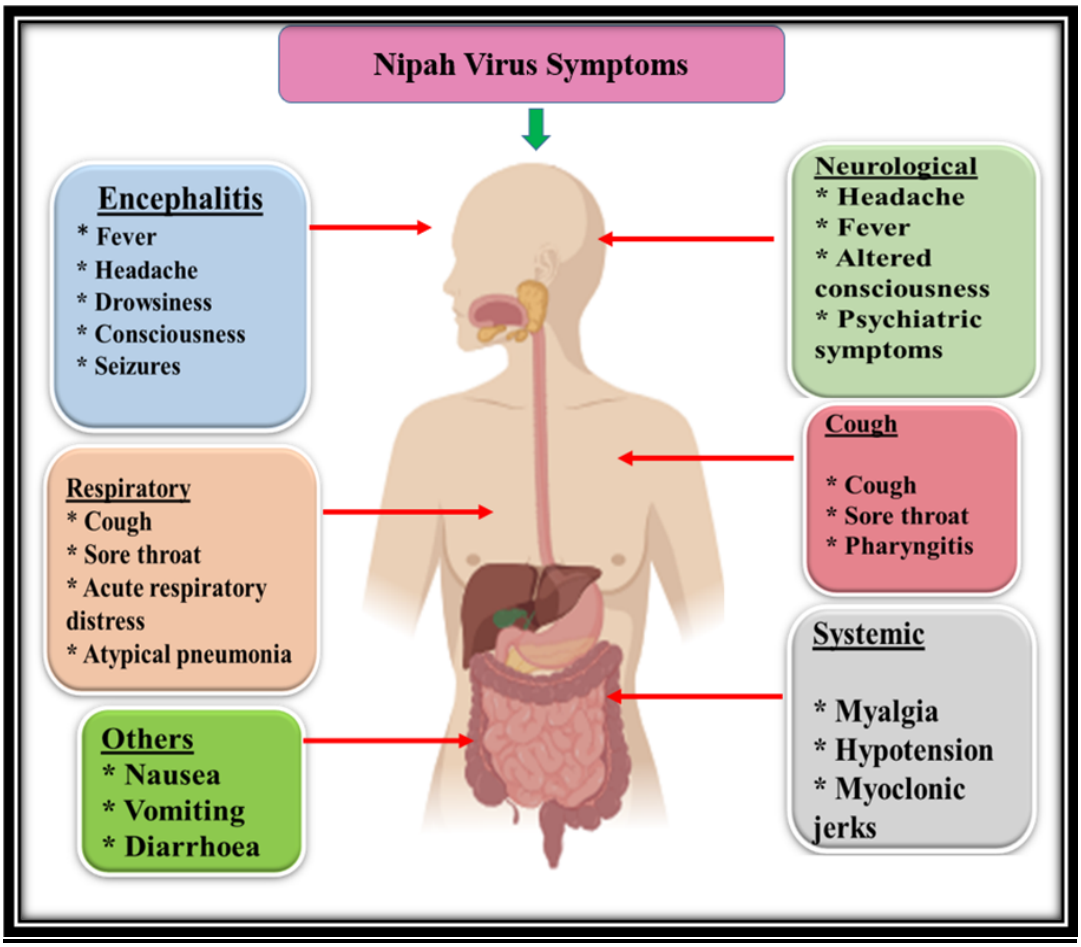

Figure 3: The picture highlights the Nipah virus's multisystem involvement and severe disease progression in infected persons by summarizing the main clinical signs of the infection, including neurological, encephalitic, respiratory, systemic, and gastrointestinal symptoms.[31]

Rapid, laborious breathing is a symptom of respiratory illness.[31] The morbidity rate can reach 100% in animals who are kept in extreme confinement or have limited mobility. Due to the neurological system's dysfunction, symptoms include tremors, flaccid or spastic paresis, hind limb weakness, and muscle twitch.[32] There have been reports of a disorder known as nystagmus in adult female sows and boars, or common wild male pigs, where the eyes move erratically, impairing vision, balance, and coordination. Additionally, pig seizures are caused by this.[33] Lung inflammation occurs in dogs with NiV infection. Other disorders include the development of syncytia in the kidneys along with necrosis of the glomeruli and tubules. Endothelial syncytia and vascular abnormalities in many organs are observed in infected cats. [34] When NiV is experimentally infected in a number of additional species, including as guinea pigs, hamsters, and African green monkeys, certain primary clinical symptoms are seen. The symptoms include weight loss, significant vascular abnormalities in various organs, and lesions in the parenchyma of the central nervous system. [34]

4.1Encephalitis

A feverish sickness with neurological abnormalities, such as convulsions, weakness, disorientation, and neuropsychiatric symptoms, is a common presentation of encephalitis. Additionally, patients may exhibit ambiguous symptomscase studies of NiV epidemics show that the majority of patients had non-specific symptoms, including as fever, headache, myalgia, and gastrointestinal and respiratory problems. [35]

4.2 Nipah virus encephalitis symptoms

Table 1: Shows the wide range of clinical characteristics, highlighting the serious neurological issues and long-lasting functional impairments linked to Nipah virus infection.

Incubation period	Ranges from a few days to months; majority within 2 weeks
Acute non-specific signs and symptoms	• Fever • Headache • Vomiting • Respiratory symptoms – atypical pneumonia, cough, acute respiratory distress syndrome[32,33,34]
Acute neurological signs and symptoms	• Reduced level of consciousness or altered Glasgow coma scale • Areflexia or hyporeflexia • Focal weakness • Brain stem dysfunction – abnormal pupils or dolls-eyes reflex, tachycardia, hypertension • Seizures • Behavioural change
Sub-acute signs and symptoms	• Late-onset encephalitis symptoms – reduced level of consciousness or altered Glasgow coma scale; brain stem dysfunction; seizures; behavioural change
Long-term sequalae	• Relapse encephalitis • Persistent neurological deficits • Cognitive deficits including memory disturbances • Depression • Behavioural change • Functional difficulties

5. Epidemiology

5.1 Malaysia and Singapore

The Nipah virus (NiV) outbreak was first identified in Malaysia in 1998-1999. The virus was later named after the Sungai Nipah village where it was first isolated from the spinal fluid of infected patients. [36] Several patients had immunoglobulin M (IgM) antibodies against Japanese encephalitis virus and were initially misdiagnosed as having Japanese encephalitis. However, the fact that the disease spread among humans despite control measures to stop it led to the identification of a new virus of animal origin called NiV. [37] Singapore also reported an outbreak among 11 slaughterhouse workers, including one death. Epidemiological studies have shown that the main source of human infection is close contact with infected pigs or pig feces. [38] Pigs have been shown to be effective breeding hosts because they are exposed to fruit bat viruses through ingestion of contaminated fruit. Large-scale and intensive pig farming practices have increased the rate of spread of infection among pigs, increasing the likelihood of infectious events leading to human infection. [39] Control measures that led to a rapid decline in infections included the culling of more than 1 million pigs, as well as large-scale decontamination, trade restrictions and increased local biosecurity. [40] Although dogs were also infected and dog mortality on affected farms was identified as an additional risk factor, there was no evidence of sustained human-to-human transmission during the outbreaks in Malaysia and

Singapore. However, serological studies performed on dogs and humans in these countries have found no evidence of Nipah virus infection in either species. However, subsequent ecological studies identified the Pteropus bat as the natural host of Nipah virus. [41]

5.2 Bangladesh

Since 2001, Bangladesh has faced recurring seasonal outbreaks of the Nipah virus, predominantly occurring during the colder months and primarily concentrated in the central and northwestern Nipah belt. In contrast to Malaysia, where outbreaks were associated with extensive pig farming, pigs in Bangladesh are generally kept as domestic animals. [42] Consequently, the primary animal reservoir responsible for Nipah outbreaks in Bangladesh is the Pteropus bat. Transmission of the Nipah virus from bats to humans occurs via raw date sap that is tainted with bat excretions and subsequently consumed by humans. [43, 44] In Bangladesh, Nipah virus outbreaks have been linked to the season for collecting date palm sap. Studies investigating seropositivity to the Nipah virus in pigs, cattle, and goats have detected the presence of the virus, indicating that these animals may contribute to transmission; however, they do not seem to be the main catalysts for outbreaks. [45,46] A notable feature of Nipah outbreaks in Bangladesh is the considerable number of secondary cases arising from person-to-person transmission. Research has shown that transmission can occur from an infected individual to another through close contact with the fluids of the infected person, including saliva. [47] Furthermore, the virus has been identified in the saliva of infected individuals, which aids in its dissemination among people. The potential for other transmission pathways, such as the consumption of fruits contaminated by bats or exposure to environments beneath bat roosts, remains unverified. [48] Therefore, the primary modes of Nipah virus transmission in Bangladesh are from bats to humans via date palm sap and subsequent vigorous human-to-human transmission. [48]

5.3 India

India has experienced multiple outbreaks of the Nipah virus that exhibit epidemiological characteristics akin to those observed in Bangladesh. The first recorded outbreak occurred in Siliguri, West Bengal, in 2001, resulting in 66 probable cases and 45 deaths. [49] A subsequent outbreak took place in 2007 in the Nadia district of West Bengal, which involved five confirmed cases and a case-fatality rate of 100%. [50] Both outbreaks demonstrated considerable person-to-person transmission, particularly within healthcare settings. In the Siliguri outbreak, the source patient, whose identity remains unknown, transmitted the virus to not only hospital patients but also healthcare personnel and visitors, highlighting the significant impact of transmission within hospitals. [51] Likewise, the 2007 outbreak originated from the index case consuming fermented date palm sap, which led to further spread among family members and healthcare workers. In 2018, another outbreak emerged in the Kozhikode and Malappuram districts of Kerala, resulting in 18 confirmed cases and 17 deaths, illustrating the Nipah virus's capacity to manifest in regions not previously recognized as endemic. [52] Additional outbreaks in Kerala in 2019, 2021, 2023, and 2024 reinforced the trend of zoonotic spillover and underscored the urgent need for rapid laboratory diagnostics, comprehensive contact tracing, stringent infection control measures, and well-coordinated public health responses. [53,54]

Table 2: Epidemiological and clinical features and outcomes in Nipah virus infections. [54]

Clinical/Epidemiological Feature	Malaysia–Singapore	Bangladesh–India
Affected population	Predominantly adult pig farmers and individuals with occupational exposure to pigs.	Affects adults, children, family members, and healthcare workers exposed to infected patients.
Primary route of spread	Transmission mainly occurred from fruit bats to pigs, followed by pig-to-human infection.	Infection primarily results from direct exposure to fruit bats through consumption of bat-contaminated date palm sap or fruits, with occasional involvement of domestic animals.
Human-to-human transmission	Person-to-person transmission was uncommon and occurred only sporadically.	Human-to-human transmission is frequent and contributes substantially to outbreak propagation.
Respiratory manifestations	Respiratory symptoms were relatively infrequent; pneumonia was reported in a small proportion of patients, sometimes without encephalitis.	Respiratory illness is common, with cough and breathing difficulty frequently observed, and some patients developing acute respiratory distress syndrome (ARDS).
Neurological manifestations	Encephalitis commonly presented with segmental myoclonus in approximately one-third to one-half of patients.	Encephalitis is common; however, segmental myoclonus has rarely been documented.
Magnetic resonance imaging (MRI) findings	MRI typically demonstrates multiple small, disseminated hyperintense lesions throughout the brain.	MRI findings are generally limited and often show larger confluent hyperintense lesions.
Relapsed or delayed-onset encephalitis	Recurrent or late-onset encephalitis occurs in approximately 5–10% of survivors.	Delayed neurological complications have been reported in a subset of recovered patients during follow-up.
Long-term neurological sequelae	Persistent neurological deficits occur in about one-fifth of survivors.	Approximately one-third of survivors experience lasting neurological impairment.
Case fatality rate	Mortality ranges from **32–41%**, reflecting comparatively lower fatality.	Mortality is substantially higher, reaching approximately **70%**, making outbreaks considerably more severe.

6. Transmission of Nipah Virus

The Nipah virus can be transmitted through direct contact with infected animals such as bats, pigs, and horses, or their bodily fluids including blood, urine, and saliva. **Fig 4.**Additionally, consumption of contaminated food products like palm sap or fruit, as well as close contact with an infected individual or their bodily fluids (including nasal or respiratory droplets, urine, or blood), can facilitate transmission. [55] NiV enters the human body through the oro-nasal route and can infect other hosts as well. High levels of antigen found in lymphoid and respiratory tissues indicate that these areas are likely the initial sites of replication. [55] NiV targets the epithelial cells of the respiratory tract, triggering the release of inflammatory cytokines, which can result in a disease resembling acute respiratory distress syndrome. Beyond the lungs, other organs such as the kidneys, spleen, and brain may also be affected, potentially leading to multiple organ failure. [56]

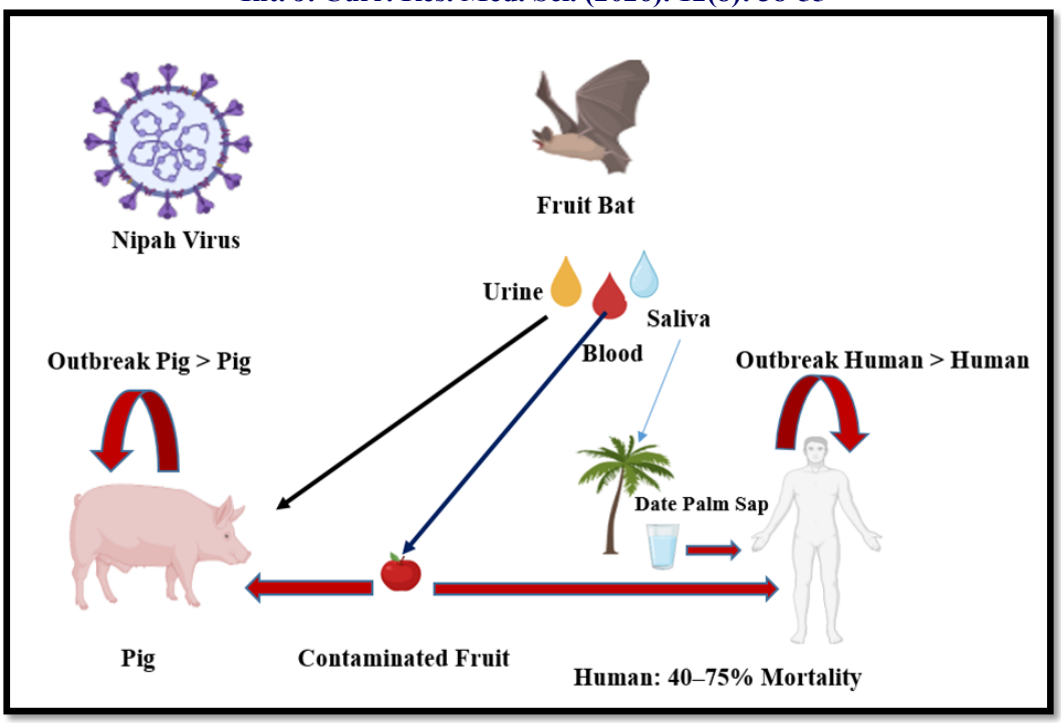

Figure 4: The illustration illustrates the transmission of the Nipah virus from fruit bats to pigs and humans via contaminated fruits, date palm sap, and bodily fluids, followed by human-to-human transmission that results in a mortality rate of 40–75%.

6.1 Transmission in humans

Direct interaction with unsanitary pigs, various infected animals, or through contaminated fruits (such as partially consumed fruits discarded by fruit bats), as well as direct contact with ill individuals, are identified as the primary causes of outbreaks. [57]Individuals at heightened risk for Nipah include:

- Those who work in close proximity to infected pigs
- Family members and caregivers of individuals diagnosed with Nipah
- Healthcare professionals attending to patients with Nipah
- Individuals who come into contact with food or beverages contaminated by infected animals
- People who ascend trees that are frequently inhabited by infected bats

Table:3 Major Transmission Routes of Nipah Virus.

Transmission Route	Primary Source	Mode of Transmission
Bat-to-Human	Fruit bats (*Pteropus* spp.), the natural reservoir of NiV	Infection occurs through the consumption of food or fresh date palm sap contaminated with bat saliva, urine, or feces, as well as through contact with contaminated fruits or environmental surfaces.
Bat-to-Animal-to-Human	Domestic pigs (particularly during the Malaysia outbreak)	Humans acquire infection by handling infected pigs or through exposure to their respiratory secretions, urine, or other contaminated excretions.
Human-to-Human	Individuals infected with Nipah virus	Person-to-person transmission occurs through close contact with infected patients, particularly via respiratory droplets, body fluids, or contaminated fomites during caregiving or healthcare exposure.

7. Diagnosis of Nipah Virus infection

The isolation of the virus, along with serological testing and assays for the amplification of viral nucleic acids, is essential for confirming the presence of infections in both humans and animals. These serological tests are primarily antibody tests that detect antibodies in the bloodstream, yielding a positive result when an infection is present. To manage NiV containment and its spread, biosafety level-4 (BSL-4) facilities are necessary. [58] A monoclonal neutralizer-based antigen ELISA has been developed for the detection of NiV. IgG ELISAs have been employed for testing sera from pigs and humans, while an IgM-mediated ELISA utilizing a recombinant N protein of NiV has proven to be highly effective in identifying viral contamination. [59] Additionally, a sandwich ELISA using a polyclonal neutralizer against the NiV G protein has been established and has demonstrated rapid diagnostic capabilities for the disease. By utilizing pseudo-typed particles, a serum neutralization test for NiV can be conducted under BSL-2 conditions. Microsphere assays have been applied to detect antibodies against the glycoprotein in the sera of pigs and ruminants, such as goats and steers. An ELISA employing recombinant full-length N and G proteins has also been developed. [60]

Table: 4 Laboratory Diagnosis of NiV Infection.

Diagnostic Method	Clinical Significance
Routine haematological tests	• Thrombocytopenia • Leucopenia • Raised liver enzymes • Hyponatremia
Cerebrospinal fluid analysis	• Lymphocytic pleocytosis • Raised proteins • Normal glucose levels
Imaging	• 2-7mm multifocal discrete lesions in the subcortical and deep white matter
NiV specific tests	• ELISA for detection of antibodies • Polymerase Chain Reaction • Virus isolation

8. Treatment of Nipah Virus infection

NiV is classified as a priority pathogen by the WHO due to its significant pandemic potential, necessitating increased attention. At present, there are no effective medications or vaccines available for NiV, which means that treatment for patients is limited to supportive care and preventive strategies.[61] Key clinical approaches to manage NiV infection post-detection include ensuring respiratory function, reducing the risk of venous thrombosis, and maintaining fluid and electrolyte balance. Additionally, broad-spectrum antibiotics are prescribed to individuals infected with NiV.[62]

Table: 5 Antiviral agents investigated for the treatment of NiV infection.

Antiviral Agent	Mechanism of Action	Preclinical and Clinical Evidence	Potential Therapeutic Application
Remdesivir	A broad-spectrum antiviral prodrug that inhibits viral RNA-dependent RNA polymerase, thereby preventing viral RNA synthesis.	Has demonstrated potent antiviral activity by significantly reducing NiV replication in both cell culture (in vitro) and animal (in vivo) studies.	Considered a promising therapeutic option for NiV infection and may serve as an adjunctive treatment to enhance viral clearance and reduce the risk of disease recurrence.
Favipiravir	Selectively inhibits viral RNA-dependent RNA polymerase, disrupting viral replication and transcription.	Exhibits strong antiviral efficacy in vitro, even at low concentrations, and provides complete protection against lethal NiV challenge in Golden hamster models.	Regarded as one of the most promising antiviral candidates for human NiV infection and may also be useful as a post-exposure prophylactic agent.
Ribavirin	A broad-spectrum antiviral drug that interferes with viral RNA synthesis and replication.	Used during the Malaysia–Singapore outbreak either alone or in combination with chloroquine; however, available clinical and experimental findings have produced inconsistent results.	Its clinical effectiveness remains inconclusive, and additional well-designed studies are needed to establish its therapeutic value against NiV infection.
Balapiravir	An investigational RNA polymerase inhibitor developed to suppress viral replication.	Demonstrated antiviral activity against Nipah virus in laboratory-based (in vitro) studies, although in vivo evidence is currently limited.	Represents a potential therapeutic candidate that warrants further evaluation in animal models and clinical studies before routine clinical use.

9. Prevention of Nipah Virus Infection

Outbreaks of NiV necessitate expensive emergency responses globally. NiV presents a heightened risk in areas characterized by various risk factors and low development indicators. [63] With the impacts of climate change and human intrusion into the habitats of flying foxes, it is probable that outbreaks will emerge in new regions. In addition to vaccination, other strategies are vital for the prevention and control of human NiV infections and may prove to be more cost-effective. Preventing infection in livestock could be an effective approach in areas where they act as intermediate hosts. [64] This involves ensuring that fruit and bat roosting trees are situated away from livestock farms and grazing areas that are vulnerable to virus contamination. In certain nations, such as Malaysia, this strategy has already demonstrated significant effectiveness. [65] Control measures are considerably more challenging in regions where contaminated date palm sap is the main source of NiV. In these areas, changes in human behavior, such as refraining from consuming contaminated date palm sap, would be essential. [65] In Bangladesh, the harvesting of date palm sap typically occurs overnight. The nocturnal behaviors of bats, including drinking from, defecating, or urinating in the jars used for collecting date palm sap, have been documented using infrared cameras. Implementing measures

to restrict bat access to the sap stream of the date palm tree, as well as shaving the surface, can reduce the risk of human exposure to NiV in these environments. [66] Raising public awareness about the dangers of consuming raw sap and preventing the contamination of sap collection pots were critical strategies for disease prevention in Bangladesh from 2012 to 2014. Thorough washing of vegetables and fruits is crucial to eliminate any traces of bat excreta. [67]

10. Emerging Opportunities

In recent decades, significant research has concentrated on the pathogenesis of the Nipah virus and its transmission mechanisms. This understanding has the potential to be significantly enhanced in the future. It is crucial to emphasize that this knowledge should be applied judiciously to facilitate the development of a Nipah vaccine for human clinical trials and to mitigate risk factors that contribute to infection. The prevention of this zoonotic disease is of paramount importance. In response to the epidemics in Bangladesh and India, scientists have established a global outbreak network and a response network, highlighting the necessity for improved communication between veterinary and medical services regarding the disease. By engaging multiple industries and employing multi-sector strategies, effective prevention measures can be formulated and executed. Furthermore, it is essential for individuals to maintain awareness of food hygiene alongside public health practices.

11. Conclusion

Despite numerous warnings and recurring outbreaks over the last twenty years, the NiV continues to represent a significant threat to global public health, resulting in considerable morbidity and mortality among both humans and animals. Its elevated case-fatality rate, zoonotic characteristics, and capacity for human-to-human transmission highlight its potential to instigate larger outbreaks with severe health, social, and economic repercussions. The lack of licensed vaccines and specific antiviral treatments further emphasizes the difficulties in managing this emerging pathogen. Enhancing surveillance systems, bolstering outbreak preparedness, raising public awareness, and implementing effective infection-control measures are vital for minimizing disease transmission. Simultaneously, there is an urgent need for accelerated research initiatives and well-structured clinical trials to assess promising therapeutic agents and vaccine candidates. Advancing these strategies will be essential for lessening the impact of future Nipah virus outbreaks and enhancing global health security.

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