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Review Article

Nipah Virus: An Emerging Zoonotic Threat with Pandemic Potential, Therapeutic Control and Vaccine Development

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Abstract

Throughout history, viral outbreaks of varying intensities and frequencies have caused disaster and terror in the world. The Nipah virus (NiV), which is extremely virulent, has a high case fatality rate, high pandemic potential, and a contagious virus outbreak of zoonotic origin. This encapsulated virus poses a significant risk of frequent outbreaks in Southeast Asia, and its glycoproteins are necessary for it to enter the host cells. Both neurological and respiratory symptoms are associated with pathogenesis. Like monoclonal antibodies and ribavirin antiviral drugs used as potential remedies, but the treatment is modest. Preventive approaches, such as stringent infection control protocols in hospitals and healthcare facilities and population-wide interventions, can help control NiV outbreaks. Prospects for the future suggest that to improve preparedness, there is a need to put a lot of effort into the development of vaccines and preparing antivirals. A comprehensive strategy for addressing NiV should include community education, rigorous surveillance, and epidemiological surveys. A concerted One Health approach that supports human, animal, and environmental surveillance is also essential for NiV management and prevention. In this review, the outbreak of NiV, along with the routes of transmission, prevention and control strategies used, potential causes of the outbreaks, and the precautions that private-public initiatives should take to ensure a lower incidence of disease are discussed.

Keywords: Nipah virus, Hendra virus, Henipavirus, zoonosis, Bat-borne disease, Pathogenesis, Pandemic potential

Introduction

The NiV is a highly contagious virus, belongs to the family *Paramyxoviridae* and genus *Henipavirus*, which has become one of the most significant zoonotic risks to public health over the last 20 years. It is an emerging, pathogenic virus that has been causing outbreaks in South Asia on an annual basis, resulting in lethal respiratory and neurological infections [1]. In 1998, an unknown cause caused several encephalitis outbreaks in Perak, Malaysia. At the start, this outbreak was considered Japanese encephalitis (JE) because the disease showed almost the same signs and symptoms clinically. But after keen research, the disease-causing virus was named NiV in 1999, after the name of the Nipah River, according to the Centers for Disease Control and Prevention (CDC) and the Malaysian Ministry of Health [2]. Fruit bats of the genus *Pteropus* serve as the virus's natural reservoir hosts and are essential to its maintenance and spread. Humans are frequently exposed to contaminated food sources, such as raw date palm sap, through direct contact with diseased animals, or, most concerning, through human-to-human transmission. After being discovered, the NiV was also found in many other countries that were involved in trade with Malaysia. It is highlighted due to its highly zoonotic impact

on people and its immense ability to create a pandemic outbreak [3]. It has been defined among the top 10 emerging contagious viruses. NiV is classified as a Category C priority infection by the World Health Organization (WHO) and is still a potential candidate for the next possible pandemic outbreaks, with a case fatality rate that can vary from 40% to up to 75% depending on the outbreak. Clinically, a NiV infection can present with a wide range of symptoms, from mild fever, illness, or no symptoms to acute respiratory syndrome and lethal encephalitis [4]. Survivors frequently experience neurological sequelae, which increases the impact on public health. Crucially, the absence of efficient antiviral therapy has compelled the use of supportive care exclusively, which has minimal effect on halting the progression of the illness or lowering death. It requires immediate research and development in case of a public health emergency by the WHO, the United Kingdom Vaccine Network, and the Coalition for Epidemic Preparedness Innovations (CEPI), which have identified it as a priority to develop a vaccine against NiV [5]. The pandemic potential, epidemiology, pathophysiology, methods of transmission, zoonotic potential, therapeutic control strategies, and the present state of vaccine research, including its obstacles and advancements, are discussed here.

Etiology

NiV is a negative-sense, ssRNA enveloped, pleomorphic, spherical, and thread-like virus that belongs to the family *Paramyxoviridae* and genus *Henipavirus*. Other members of this family include measles virus, Newcastle disease virus, Mumps, parainfluenza, and Hendra virus (HeV). NiV is also closely related to the Hendra virus. The complete linear genome of NiV is approximately 18.2kb nucleotides [6]. The NiV genome is a non-segmented, negative-sense RNA molecule encoding six major structural proteins, including RNA polymerase or large protein (L), matrix protein (M), fusion glycoprotein (F), attachment glycoprotein (G), phosphoprotein (P), and nucleoprotein (N). It also encodes three non-structural proteins, including C, V, and W, which are produced from the P gene and play roles in viral virulence. So, F and G proteins play a role in the virion-cellular attachment and, afterward, entry into the host cell. The N, P, and L proteins are involved in the viral RNA attachment that results in the virus ribonucleoprotein (vRNP) [7]. Two main, different genotypes of NiV were identified by phylogenetic analysis: NiV-Malaysia (NiV-MY), which is recognized in both Malaysia & Cambodia, and NiV-Bangladesh (NiV-BD), which is recognized in Bangladesh as well as India. Additionally, compared to NiV-BD, NiV-MY genotypes have demonstrated greater virulence, which is directly correlated with the clinical appearance, pathogenicity, mode of transmission, and severity of the disease [8]. The morphology of the NiV is shown in *Fig. 1*.

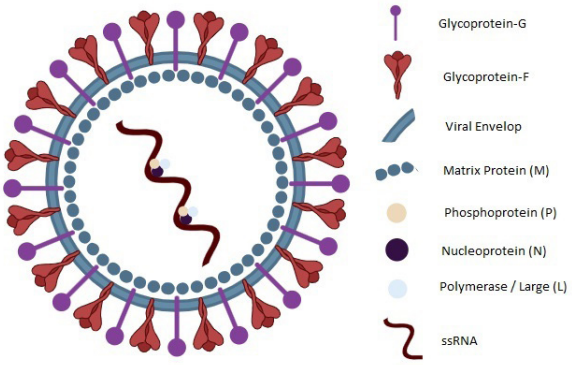

Fig 1. Morphology of NiV. Viral envelope, single-stranded RNA, 6-structural protein: Attachment protein (G), Fusion protein (F), Matrix protein (M), Phosphoprotein (P), Polymerase protein (L), Nucleoprotein (N)

Epidemiology and Pandemic Potential

NiV is a renowned zoonotic viral disease that spreads from animals to people and represents a great threat to both animal and public health. The epidemiology is influenced by the interactions between intermediate animal species, reservoir hosts, human social behaviors, and ecological shifts. NiV also has a high pandemic potential and spreads throughout the South and Southeast Asia [9]. In 1998, the first epidemic outbreak was observed in the pigs in Malaysia. The second outbreak of NiV occurred in a small town in Negri Sembilan in the winter of 1999, and the third and largest outbreak took place near Bukit Pelandok, one of the major pig farming settlements. In this region, the target of NiV infection was pigs, which subsequently spread to humans. They showed the signs of acute respiratory distress and encephalitis [3]. Approximately 265 cases of acute NiV encephalitis were reported, out of which almost 105 people died. So, the infected pigs were exported to other countries by Malaysia, and the NiV disease disseminated there. Beyond Malaysia, NiV cases were reported in neighboring countries, including Singapore, the Philippines, and South Asia (Bangladesh and India), where the cases were reported almost annually [6]. NiV infection reached Singapore in the latter part of February 1999. Since 2001, periodic NiV outbreaks have also been reported in Bangladesh and India, and by 2014, the virus had also infected other countries in South Asia [10]. As Bangladesh is an Islamic country, it didn't import pigs as a source of food. Therefore, the disease was spread to humans through the consumption of contaminated fruits. The virus was believed to have been transmitted by infected horses in the Philippines, where 17 cases with the symptoms were noted. The mortality rate of acute encephalitis was higher at 82 percent. There are also endemic nations such as Thailand, Cambodia, Indonesia, Madagascar, and Ghana. Compared to past infectious

outbreaks, NiV had a higher fatality rate [5]. It is highly virulent and is studied under biosafety level 4 (BSL-4) in a laboratory that is safe for zoonotic emerging infectious diseases. The pandemic potential of NiV is becoming much more significant. It is more likely to spread as a pandemic due to a number of factors, including human sensitivity to the virus and its quick person-to-person transmission. Climate change has also been a concern, with deforestation the risk of occurrence of human-animal contact increases and the chances of spreading of NiV to other tropical regions of the world escalates [11].

Pathogenesis

In humans, NiV enters the body through ingestion or breathing. Epithelial cells, especially those located in the bronchiole, are the first replication sites and may serve as a means of diagnosing NiV early in the course of infection. The bronchi and, in some cases, the alveoli have been found to contain viral antigens. Acute respiratory distress syndrome (ARDS) like illness results from the release of cytokines from the affected respiratory tract epithelium caused by NiV infection [12]. In the later stages, the airway epithelium also secretes inflammatory mediators, including interleukin and granulocyte-colony stimulating the tissues. In both the early and late stages of infection, respiratory or urinary tract epithelial cells play a critical role in viral replication, particularly when it comes to viral shedding and transmission through urine and airway secretions [13].

The virus enters endothelial cells of the surrounding vessels (capillaries) from the respiratory epithelium. As the illness progresses, the virus enters to the bloodstream. Multi-organ failure can result from attacks on the brain, in addition to the pulmonary, digestive, and excretory systems [14]. NiV enters the central nervous system (CNS) through the choroid plexus, a network of blood vessels in the cerebrum. A number of neurological issues can arise as a result of this dissemination of infection across the blood-brain barrier (BBB). Necrosis can result from the presence of virus particles in the central nervous system. Over time, the infection can also propagate to the CNS through the olfactory bulb. Finally, the virus travels down the olfactory tubercle across the entire ventral brain [15]. The pathogenesis of the NiV is shown in *Fig. 2*.

Viral membrane integration with the host cell membrane is made possible by the viral initial attachment to cell surface receptors (ephrin-B2/B3). Transmembrane glycoproteins, which are encoded by all *Paramyxovirus* members, aid in the attachment and fusion of the virus, which in turn allows the virus to enter cells and disseminate during the early phases of infection [16]. Two kinds of envelope glycoproteins are seen on the surfaces of NiV, referred to as G-glycoprotein for attachment and F-glycoprotein for pH-independent fusion activities. The direct transmission of the pathogenic virus from affected to unaffected adjacent cells is mediated by these glycoproteins, which also cause cytopathogenicity. The cytopathic impact of NiV infection causes endothelial cells to develop membrane fusion-mediated syncytia, which is triggered by these two viral glycoproteins. A hallmark that sets NiV illness apart is the development of symptoms in the endothelial cells of the blood vessels [17].

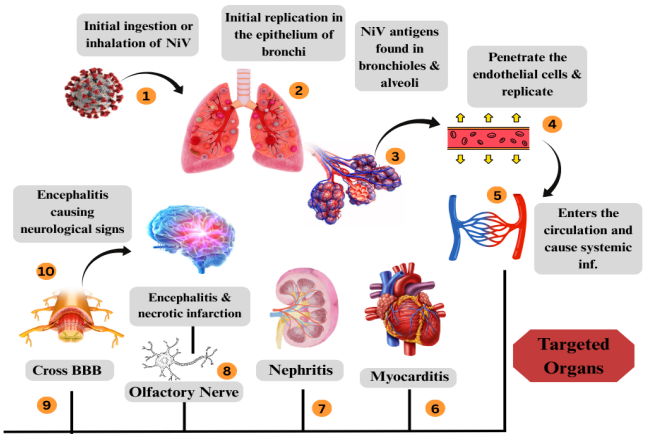

Fig 2. Pathogenesis of NiV. 1. Virus enters the epithelial cells of 2. Bronchioles (Lungs) 3. Viral antigens found in alveoli 4. Penetrate into endothelial cells & replicate 5. Enters circulation 6. Myocardial infarction 7. Renal dysfunction 8. Olfactory nerve leads to encephalitis 9. Crossing the BBB causes neurological signs

Henipaviruses, which belong to the *Paramyxoviridae*, such as NiV, attach to host cell receptors. It's interesting to note that NiV proteins engage alternative host cell receptors because their glycoproteins lack hemagglutination and neuraminidase activity. In contrast, other *Paramyxoviruses*, including *Rubulaviruses, Respiroviruses,* and *Avulaviruses*, bind to receptors that contain sialic acid and exhibit neuraminidase activity. Human neurons, sinus linings of lymphatic nodes, vascular cells, the spleen cells, and placental tissue, arteries surrounding smooth muscle, and airway epithelial cells all express ephrin-B2 receptors; lymphoid cells and the central nervous system express ephrin-B3, a substitute entry receptor [18].

Transmission and its Zoonotic Spillover

Like the related *Hendra virus, Lyssaviruses, Filoviruses,* and *Coronaviruses,* the naturally occurring reservoir host of the NiV is the *Pteropus* fruit bats. Fruit bats (sometimes called megabats) belong to the *Pteropodidae* family. The fruit bats, also known as flying foxes, are primarily responsible for the spread of NiV. Although NiV is a contagious and zoonotic virus, it may potentially spread from person to person through close contact and through either direct or indirect contact with infected animals or their body fluids, like a bat's urine or saliva [19]. The pigs and horses frequently serve as intermediate hosts for NiV. As the outbreak occurred in

Malaysia and its transmission was linked to infected pigs, but in the case of Bangladesh, the pandemic outbreak was due to the consumption of the contaminated date palm sap, and in the Philippines, the infected horses play a role in this zoonotic impact of the NiV disease [20]. The following are the main transmission routes:

People can get the disease by coming into close contact with sick pigs and bats or their waste products, including their urine, feces, or saliva. For a specific period of time, NiV can endure on surfaces in the environment. Transmission can occur if people touch their mouth, nose, or eyes after coming into contact with contaminated surfaces, tools, or items without practicing good hand hygiene, such as people climbing on the date palm tree [21]. In several outbreaks, raw date palm sap tainted with infected fruit bat pee or saliva was consumed by the susceptible host, leading to the spread of the NiV. For a specific period, the virus may survive in date palm sap, making it a possible source of outbreak. The NiV may spread from person to person via direct contact with an infected individual's body fluids, including blood, urine, saliva, and respiratory secretions. The major problem during epidemics is this kind of transmission, especially in medical facilities where exposed patients or their tainted medical equipment may be present [22]. Mistakes in infection control procedures have led to NiV epidemics in medical facilities. When providing treatment for patients infected with the NiV, healthcare professionals and caregivers run the risk of contracting the disease if they fail to take the proper measures, such as wearing personal protective equipment (PPE) [23]. The transmission and zoonotic potential of NiV is shown *Fig. 3*.

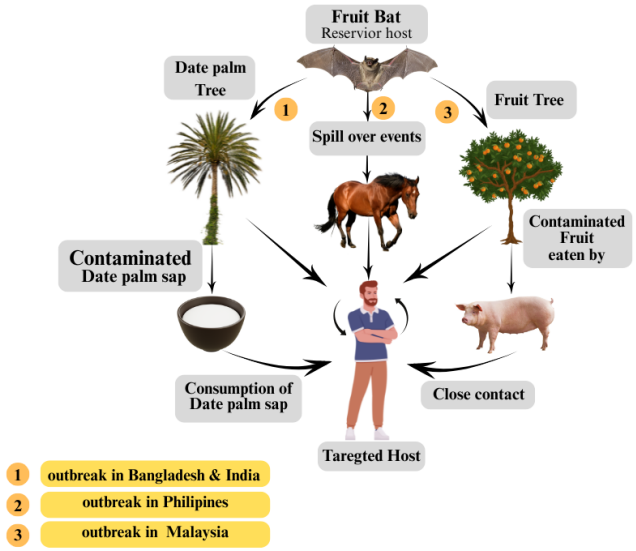

Fig 3. Transmission of NiV and its Zoonotic Spillover. 1. Outbreak in Bangladesh & India by direct contact and consumption of contaminated date palm sap, 2. Outbreak in the Philippines due to the consumption of raw meat of affected horses, 3. Outbreak in Malaysia by contact with infected pigs and fruit bats

Clinical Manifestations

There are several clinical manifestations caused by NiV infection in both humans and animals, depending on the basis of the varying intensities of exposure to the infection.

Humans

Although the NiV is most recognized for its effects on the neurological system, and may also harm the respiratory system. The incubation period for the NiV is 4-45 days. The primary symptoms of NiV are fever, headache, respiratory discomfort, muscle aches, vomiting, poor coordination, disorientation, and uncontrollable walking [24]. There could be bleeding from the gut, renal injury, and the development of septicemia. In extreme cases, the seizures are followed by encephalitis within 24 to 48 hours, which ultimately leads to coma. Approximately 14% to 29% of individuals in Malaysia were reported to have respiratory problems at the time of the initial outbreak [25].

Animals

NiV shows a vast diversity in the viral disease, mostly in pigs or some species of bats. A series of clinical manifestations that cannot be fully understood without the complications of the viral dynamics in the intermediate hosts, as well as in the natural reservoirs.

Various clinical manifestations may occur in pigs, which are intermediate hosts of the NiV infection in humans. These symptoms indicate the characteristic manifestation of the virus, which is to attack the respiratory system, causing difficulty in breathing and frequent coughing [26]. More insights on the virus-to-host interaction can be obtained through neurological manifestations, including apparent trembling, muscle weakness, and poor coordination. It is important to note that miscarriage and stillbirth in pregnant sows are linked to infections caused by NiV in pigs. This is the fact of this virus that it even leads to unexpected and sudden death of the infected pigs without apparent clinical manifestation beforehand [27]. Fruit bats are literally the ones referred to as natural reservoir hosts of the NiV. Strikingly, these bats are unable to show clinical signs. They incubate the virus in their body fluids and waste products, and in doing so, they become the main source of virus transmission. This special attribute highlights the effects of bat-to-animal or bat-to-human through contaminated excretions and secretions [28].

Diagnostic Approaches and Laboratory Confirmations

For the purposes of reversing the high mortality rate associated with NiV infection, the identification of the disease early and accurately is a must. To meet this end, a comprehensive diagnostic procedure involves taking a range of specimens from infected animals and humans.

Samples collected in the case of human patients include cerebrospinal fluid (CSF), urine, blood, throat, and nasal swabs used in diagnosis. In the same breath, diseased animals provide excellent diagnostic specimens that could be used in the isolation and detection of NiV, including their kidney, lungs, and spleen [29]. The NiV diagnostic tests should be performed at highly controlled and dedicated institutions, especially in Biosafety Level 4 (BSL4) laboratories. The RT-PCR tests of the blood, urine, nasal passage, throat, and cerebrospinal fluid can be carried out to detect early development of the NiV infections. Other diagnostic options also include DNA amplification, DNA sequencing, immunofluorescence test, histopathology, viral isolation, neutralization, and high-throughput to carry out complete genome sequencing of NiV infecting individuals and animals [30]. Following WHO and conventional diagnostics of NiV, PCR is the most sensitive procedure to detect an active NiV disease. Nevertheless, NiV-specific IgM ELISA is an alternative serological test in conditions where PCR is inaccessible. ELISA forms a reliable technique in the detection of NiV, though with low sensitivity and specificity compared to molecular detection [31].

Prevention & Control Measures

As the NiV may have various implications, prevention of its infection is essential. As NiV is transmitted to humans through animals, control measures such as avoiding contact with infected animals and secretions of the fruit bats should be taken. Public health measures are necessary in order to slow the spread, including isolating the patients who have been confirmed. Additionally, a quarantine policy might be implemented for those who have had close contact with the reported cases, and symptom observation could be conducted to lessen the chance of transmission [32]. To prevent the transmission of a disease, it is critical to undertake control measures, especially in the case of healthcare workers (HCWs). This has meant that, due to the lessons learnt through previous outbreaks such as the Ebola virus and severe acute respiratory syndrome (SARS), strong recommendations are set regarding the protection of HCWs. An efficient infection prevention and control plan incorporates standard precautions, strict hand hygiene, and personal protective equipment (PPE) use as its guiding principle. These precautions are necessary when handling all patient care operations, even those that make aerosols [33]. This can include separating sick patients in the same rooms or those with single-patient rooms to minimize contact with vulnerable persons. In regions prone to viral epidemics, healthcare facilities ought to be prepared to handle NiV cases. This preparedness also involves broad screening and admissions, effective triage systems. Standard precautions must be observed in every respect of medical care, such as how a patient is handled, collection of the samples, cleaning, and disposal of wastes [34].

Therapeutic Interventions

Currently, there are no specific effective antiviral medications or vaccines specifically designed to treat NiV infection. The WHO only recommends therapies for severe respiratory and neurological problems; therefore, prevention and intensive and supportive care will be the cornerstones of management [35]. Many antiviral medications were studied for the treatment of NiV, which are listed in *Table 1*.

Immunity Against NiV Infection

Immunity against the NiV has different characteristics in natural infections compared to experimental infections. The innate and adaptive immune response that develops in spontaneous infections has a significant impact on the onset of the NiV infection and its clinical prognosis. Interferon signaling, pro-inflammatory cytokine activation, and antibody synthesis are all involved in the immune response [45]. Viral glycoprotein antibodies are usually neutralized, resulting in long-term protective immunity in survivors. The kinetics of antibody production, cytokine expression, and T-cell activation can all be studied in experimental infection models, such as those in hamsters, ferrets, and non-human primates. Survival is linked to the production of neutralizing antibodies and virus-specific T-cell responses early in the infection. These immunological dynamics are crucial to understanding how to formulate effective vaccines and interventions against NiV [46].

The first defense against NiV infection is innate immunity, which occurs immediately upon viral entry before the adaptive immune system is stimulated. Neutrophils surround viral particles, which produce reactive oxygen species, antimicrobial peptides, and neutrophil extracellular traps (NETs), but excessive NET production causes tissue damage. NiV initially infects endothelial cells, epithelial cells, and selected immune cells upon natural infection, where it also induces recognition via pattern recognition receptors (PRRs), which include Toll-like receptors (TLRs) and RIG-I-like receptors (RLRs) [47]. These receptors sense viral components, especially viral RNA, and cause the activation of signaling pathways, including NF-κB and IRF3/7, which promote type I interferons (IFN-α and IFN-β) production and pro-inflammatory cytokines. NiV infection stimulates production that is necessary in order to restrain viral replication. Important innate cytotoxic cells, including natural killer (NK) cells, recognize and kill NiV-infected cells by identifying stress-induced ligands and down-regulated MHC class I. Neutrophils and macrophages facilitate early viral clearance by phagocytosis and secretion of inflammatory mediators [48]. Nevertheless, in spite of these defenses, NiV contains a few structural and

Table 1. *Therapeutics evaluated against NiV*

Drug	Target	Mechanism of Action	Dose/Route	Key Findings	Advantages	Limitations	Study Type	Reference
Ribavirin	Humans/Syrian hamsters	Nucleoside analog; inhibits viral RNA synthesis	Oral/IV; early-phase administration	Reduced mortality in early human cases; limited effect if delayed	Moderate efficacy, Broad antiviral spectrum;	Weak evidence; toxicity at high doses	Retrospective/ *In vivo*	[36]
Remdesivir	African green monkeys	RNA-dependent RNA polymerase inhibitor	5-10 mg/kg, IV; pre- and post-exposure	Complete protection when given early; reduced viral load	High efficacy, Broad antiviral spectrum; already FDA-approved for other viruses	IV only; high cost	*In vivo*	[37]
Favipiravir	Syrian golden hamsters	Inhibits viral RNA polymerase, causing lethal mutagenesis	300 g/kg/day; within 24 h post-infection	Enhanced survival and reduced virus titer	Moderate to high efficacy, Oral bioavailability; Stockpiled for influenza	Teratogenicity; limited NiV human data	*In vivo*	[38]
Chloroquine	Vero E6 cells	Blocks endosomal acidification, interfering with viral entry	10-25 µM Within 24 h	Inhibited viral entry *in vitro;* lacked *in vivo* efficacy	Ineffective in vivo, Low cost; widely available	limited animal or clinical benefit	*In vitro*	[39]
Monoclonal antibody (m102.4)	Ferrets/African green monkeys	Neutralizes G glycoprotein, blocking viral attachment	15-50 mg/kg Single IV dose; 24-48 h post-exposure	Protected against disease progression and death	High efficacy, Strong preclinical/early human safety data	IV only; needs cold chain	*In vivo*	[40]
Monoclonal antibody cocktail (1F5 + m102.4)	African green monkeys	Broad neutralization of G glycoprotein variants	20 mg/kg IV combination; early post-exposure	Improved survivals and monotherapy; reduced viral load	Very high efficacy in animals may prevent escape mutants	High cost; logistical complexity	*In vivo*	[41]
Galidesivir (BCX4430)	Animal models (hamsters, NHPs)	Adenosine analog; inhibits viral RNA synthesis	50-100 mg/kg; early administration	Reduced mortality in filoviruses; NiV data suggest potential benefit	Broad-spectrum RNA virus activity	No human NiV trials	*In vivo*	[42]
Remdesivir + mAb combination	African green monkeys	Polymerase inhibition + viral neutralization	50 mg/kg IV combo; early post-exposure	Synergistic protection; improved survival	Very high efficacy, Combines two mechanisms; lowers resistance risk	Resource-heavy; IV	*In vivo*	[43]
Acyclovir	Vero cells	DNA polymerase inhibitor (herpesviruses)	10-50 µM	No significant antiviral effect against NiV	Ineffective for RNA viruses, Safe; widely available	Not active against RNA viruses	*In vitro*	[44]

non-structural proteins, including V, P, W, and M, which inhibit the IFN signaling by attacking the interaction between the proteins and the signaling transducer. IFN-I has been demonstrated to play a role in host defense against NiV infection in vivo in hamsters, ferrets, and mice, and interventions that drive IFN-I signaling, such as poly(I)-poly(C12U), can improve survival. Excessive release of pro-inflammatory cytokines, such as TNF-α and IL-1β, is also involved in the neurological pathology and disrupts the blood-brain barrier [49].

Adaptive immunity is very significant in the control and clearance of NiV infection, which is observed following the primary innate reaction. B lymphocytes generate virus-specific antibodies and memory B cells to produce humoral immunity against NiV to provide both long and short-term protection. After the first exposure, IgM antibodies are produced, and then IgG, IgA, and other immunoglobulin subclasses are also produced. And when re-exposed, there is a fast, robust response by means of memory B cells [50]. There is a limited amount of human data, although a 2018 NiV outbreak in India involving human subjects reported that survivors produced NiV-specific IgM and IgG antibodies, with an increase in the number of B cells, which is an indication that adaptive immunity has worked. In animal experiments, it has been further shown that neutralization antibodies are produced in 1-2 weeks post-infection in swine, although viral RNA may remain in the presence of antibodies [51]. Late B-cell response was observed to be linked with the fast disease progression in African green monkeys, and the early production of IgM and IgG correlated with the survival of monkeys. These findings collectively indicate that humoral responses are of great importance in viral clearance and protection, but the kinetics and magnitude of B-cell and antibody responses vary across species and define disease outcome [52].

T lymphocytes are the cellular immune mediators that play a crucial role in the control and removal of NiV infection. The coordinating activity of T cells is that CD8+ cytotoxic T cells identify viral peptides displayed on MHC class I molecules, killing infected cells directly by the process of apoptosis and cytolysis, whilst CD4+ helper T cells promote B-cell activation and cytotoxic effects by secreting cytokines. The expansion and functioning of these effector cells are coordinated by cytokines like IFN-γ, TNF-α, and IL-2. The adaptive response in the memory B and T cells induces long-lasting immunity, allowing rapid and intense reactions to a new exposure [53]. Animal models, including African green monkeys, swine, mice, and others, were involved in the process of virus clearance accompanied by enhanced production of cytokines and chemokines, which supported the role of both CD4+ and CD8+ effector and memory T cells in the process. Mice vaccine experiments demonstrated that NiV-specific T cell responses could be induced against F and G protein epitopes, suggesting potential vaccine targets. In addition, T cells that had cross-reacted during earlier exposure to other *Paramyxoviridae* viruses, such as measles and human parainfluenza, were able to recognize and kill NiV-infected cells, which indicated that cellular immunity can be more broad-based in terms of protection against antigenically related viruses [5].

Vaccine Development

NiV is a life-threatening disease because it is widely distributed and has no effective cure. Preventing the NiV infections to reduce the spread, through the development of vaccines, would help relieve it among vulnerable populations. But making vaccines against the reservoir of the virus, the bats, is very impractical since working with a live pathogen may cause biohazards and other possible issues of administration. The immunogenic agents of the traditional vaccine are usually weakened pathogen or their proteins, but other types of antigens are more favoured by the investigators due to the fear of biohazards [54]. An interesting approach is through subunit vaccines, which use fragments of protein or a glycoprotein of a pathogen and produce a protective immune reaction to it, e.g., in the cat family, soluble G glycoprotein, when administered subcutaneously, alone is capable of inducing serum-neutralizing antibodies. In cats that were kept vaccinated, the levels of antibody were significantly higher (titer ~20,000) up to 2 months [55].

The development of the NiV vaccine has been done in various approaches, and the surface glycoproteins have formed a major consideration, G-glycoprotein and F-glycoprotein. The former involves a recombinant subunit vaccine such as Hendra virus subunit glycoprotein (HeVsG) that has been shown to offer protection against NiV and HeV challenge on ferrets, African green monkeys (AGM), as well as rabbits. Applied in Australia to horses (Equivac HeV, Zoetis) as a veterinary vaccine against HeV. The HeVsG is also being evaluated as a possible human vaccination against NiV. Thus, the immunization of ferrets by the HeVsG demonstrated exceptional outcomes with respect to disease prevention and a reduction in NiV replication and protected the ferrets for at least 14 months. Nonetheless, HeVsG vaccination induces cross-neutralising antibodies against NiV in pigs, though they were not at protective levels [56]. The concentrations of cross-neutralizing antibodies in these animals rose about 80-fold after 5-7 days after making the challenge; however, they did not have significant cellular immunological memory and protection. It is interesting to mention that pigs that were exposed to NiV orally and nasally produced a protective antibody response and cell-mediated immune (CMI) memory response. The following are

viral recombinant vectors that have been demonstrated to protect hamsters, pigs, ferrets, and/or AGMs against NiV challenge: recombinant *Rhabdoviruses* (VSV and rabies) expressing NiV F or NiV G, vaccinia viruses encoding both NiV F and NiV G, canarypox encoding NiV F or NiV G, and recombinant measles virus vector expressing NiV G. In spite of the fact that antibodies against the G or F glycoproteins may lead to neutralization of viruses, G appears to be the most prevalent and co-dominant target of neutralizing antibodies [57].

Integrating One Health in NiV Surveillance

It is important that the One Health strategy be included in NiV surveillance in order to manage this new zoonotic threat in a coherent way. This plan unites communities, domains, and sectors across various levels of society to work together in the prevention, forecasting, detection, and response of global health emergencies such as pandemics. Given that the One Health strategy takes into consideration the aspects of the environment, veterinary health, and public health, it can be deployed to improve the monitoring of this virus and minimize the chances of severe NiV outbreaks. Health-based surveillance involves coordinated efforts by veterinarians, animal biologists, environmental scientists, and medical professionals to detect and follow the virus throughout its several reservoirs and transmission pathways [58]. High-risk areas, where agricultural activities, ecological disturbance, and human-wildlife contact raise the possibility of spillover occurrences, should be given priority by surveillance systems. Predictive skills can be further improved by environmental monitoring, which includes land-use changes and climatic variability.

Future Perspective and Conclusions

Most countries do not have specialized surveillance tools, diagnostic instruments, or emergency measures that plan to fight this virus, implying that the world is unprepared for a Nipah pandemic. Even though the WHO and other agencies have classified NiV as a priority research and development pathogen, it is imperative to note that there is an urgent need for more funds to be allocated to monitoring, educating the masses, developing vaccines, thus a possible health emergency in the future can be prevented. In the last decades, particular attention was given to the pathophysiology of the NiV and its transmission method. This knowledge was improved in the next decades. Here, it is important to keep in mind that the prudent application of the same knowledge is needed so that the NiV vaccine can be developed, subject to human clinical trials, and risk factors can be reduced to prevent the occurrence of infection. This zoonotic illness must be prevented at all costs [59]. Given the necessity for better communication between veterinary and medical services on the illness, scientists have established a global outbreak network and a response network, particularly in the wake of the outbreaks in Bangladesh and India. Some strong preventative measures may be created and put into action by incorporating many industries and multi-sector approaches. NiV has a high zoonotic effect and a high mortality rate. Frequent NiV outbreaks have resulted in several human and animal deaths and morbidities over the past 20 years, despite multiple warnings. Furthermore, because of its high mortality rate and propensity to cause a major physical and financial burden, prevention of this illness is crucial owing to its pandemic potential. In order to stop any more NiV outbreaks, health officials must immediately begin clinical trials to determine potential treatment plans.

Declarations

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