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Insights into Nipah Virus: A Review of Epidemiology, Pathogenesis, and Therapeutic Advances

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Abstract:- Emerging as a WHO priority pathogen, Nipah virus (NiV) – an RNA virus within the Paramyxoviridae family – first ignited outbreaks in 1998 Malaysia. Closely related to Hendra virus, NiV continues to threaten South and Southeast Asia. A zoonotic threat, Nipah virus (NiV) jumps from its natural reservoir, fruit bats, to pigs and then humans. This BSL-4 threat, with no cure or shield, compels us to harmonise the voices of humans, animals, and the environment in a One Health symphony to prevent future outbreaks. A 2018 Chinese study identified populations at high risk for Nipah virus infection are Fruit farmers, traders, palm wine brewers, Cattle herders, especially pig farmers and Tourists. Nipah virus exhibits remarkable zoonotic versatility, with transmission pathways between humans and animals varying based on geography. Factors such as diverse livestock breeding practices, local eating habits, and the interplay with the natural reservoir - fruit bats - contribute to this fascinating mosaic of infection routes. Unravelling these complexities is crucial for designing effective control strategies tailored to specific regions. Following exposure to the Nipah virus (NiV), symptoms typically appear within two weeks, ranging from 4 days to 2 months. Fever, headache, dizziness, and vomiting are common initial signs, potentially progressing to severe encephalitis. A promising development in the fight against Nipah virus emerges as the National Institute of Allergy and Infectious Diseases (NIAID) initiates an early-phase clinical trial for an investigational vaccine.

I. INTRODUCTION

Emerging from South and Southeast Asia, Nipah virus infection (NiV) erupts in sporadic outbreaks, fueled by its zoonotic nature. Bats harbour the virus, transmitting it to humans or through pigs acting as intermediate hosts. NiV presents a double-edged threat, manifesting in two distinct syndromes: a devastating encephalitis or a severe respiratory illness, depending on the specific viral strain. This activity delves into the clinical evaluation of Nipah virus infection, emphasising the vital role of collaborative inter-professional teams in coordinating care for this highly lethal disease. A zoonotic threat, Nipah virus (NiV) jumps from its natural reservoir, fruit bats, to pigs and then humans. This BSL-4 threat, with no cure or shield, compels us to harmonise the voices of humans, animals, and the environment in a One Health symphony to prevent future outbreaks .Emerging as a WHO priority pathogen, Nipah virus (NiV) – an RNA virus within the Paramyxoviridae family – first ignited outbreaks in 1998 Malaysia. Closely related to Hendra virus, NiV continues to threaten South and Southeast Asia. [1]

Newly discovered in 1999, Nipah virus, a zoonotic paramyxovirus related to Hendra, ignited simultaneous outbreaks in pigs and humans across Malaysia and Singapore. With pigs acting as intermediary hosts, the virus caused 276 cases and 106 deaths before ending with the culling of over 1 million pigs. [2,3]

In late September 1998, the first cluster of cases emerged in pig-farming villages near Ipoh, Perak, West Malaysia. This outbreak continued until February 1999. Shortly after, in December, a second cluster appeared near Sikamat, Negeri Sembilan. December also saw the start of the largest cluster, near Bukit Pelandok in the same state.The outbreak initially resembled Japanese encephalitis (JE), a mosquito-borne viral disease with a history of porcine-associated outbreaks in Malaysia. This suspicion was bolstered by the detection of JE-specific immunoglobulin M (IgM) in four out of 28 tested patient samples, alongside the presence of JE nucleic acids in some patients' sera. These findings prompted immediate control measures, including mosquito fogging and intensified JE vaccination campaigns.[4,5,6]

Despite limited human cases (<700) and contained outbreaks, Nipah virus's lethality makes each incident a major public health concern. Its impact extends beyond individuals, affecting families, healthcare systems, and even economies through potential agricultural involvement. To effectively prevent infections and deaths, this study analyses 20 years of scientific advancements in Nipah virus epidemiology and biology, highlighting key knowledge gaps that require urgent attention.[7]

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Nipah virus exhibits environmental resilience, persisting for up to 3 days in certain fruit juices and mango, and at least 7 days in artificial date palm sap under specific conditions (13% sucrose, 0.21% BSA, pH 7.0, 22°C). Its half-life in fruit bat urine reaches 18 hours, highlighting its relative stability. While viable at 70°C for 1 hour (with reduced concentration), complete inactivation necessitates heating at 100°C for over 15 minutes (de Wit et al., 2014). However, NiV's environmental stability in its natural habitat remains variable, subject to diverse conditions. Fortunately, soaps, detergents, and common disinfectants like sodium hypochlorite readily inactivate the virus (Hassan et al., 2018).[8,9]

II. AETIOLOGY

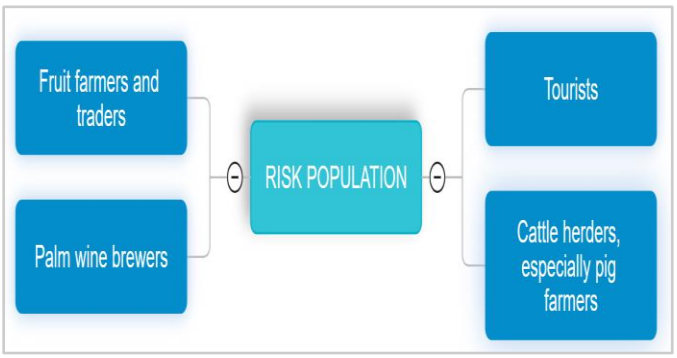

Fig.1 Risk Population Prone for Nipah Virus.

Fruit farmers and traders: Fruit bats, the natural reservoir, readily transmit the virus to those handling fruits. Palm wine brewers: Collecting date palm sap in earthen pots exposes brewers to bat urine or saliva, potential contamination sources. Cattle herders, especially pig farmers: Close contact with livestock, particularly pigs, increases the risk of transmission. Tourists: Visiting rural areas brings potential contact with the aforementioned high-risk groups or, in rare cases, directly infected. Fruit bats, specifically the Pteropus genus, act as natural reservoirs for Nipah virus. Widely distributed across the southern hemisphere, these bats – found in West Africa, Madagascar, and Southeast Asia – have shown NiV seropositivity, raising concerns about potential zoonotic transmission.[10]

III. EPIDEMIOLOGY

Nipah virus exhibits remarkable zoonotic versatility, with transmission pathways between humans and animals varying based on geography. Factors such as diverse livestock breeding practices, local eating habits, and the interplay with the natural reservoir - fruit bats - contribute to this fascinating mosaic of infection routes. Unravelling these complexities is crucial for designing effective control strategies tailored to specific region.[11]

Country	Year/regions	Primary route of transmission	Cases	Death	Fatality rate [%]
Malaysia	1998–1999	Contact with pigs	265	105	39.6
Singapore	1999	Contact with pigs	11	1	9
India	2001 Siliguri	Human-to-human–close direct contact Contact with bats from the *Pteropus* spp.	66	45	68.2
	2007 Nadia		5	5	100
	2018 Kerala		18	17	94.4
	2021 Kerala		1	1	100.0
Bangladesh	2001 Meherpur	Consumption of contaminated fruits and palm sap Person-to-person–close direct contact	13	9	69.2
	2003–2007 Naogaon, Rajbari, Tangail, Kushtia, Natore, Pabna, Thakurgaon		99	78	78.8
	2008–2015 Manikganj, Rajbari, Gaibandha, Nilphamari, Rangpur, Faridpur, Gopalganj, Kurigram, Comilla, Dinajpur, Lalmonirhat, Joypurhat, Naogaon, Natore, Pabna, Magura, Ponchoghor		139	106	76.3
Philippines	2014	Contact with horses Consumption of horse meat	17	9	52.9

Fig.2 Adopted from Skowron K, Bauza-Kaszewska J et. al, 2022

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➢ ***Malaysia:***

The emergence of Nipah virus (NiV) in Malaysia during the 1998 epidemic marked a significant shift in the landscape of zoonotic viral infections. Initially misattributed to Japanese encephalitis (JE) due to its presence in adults and its association with pig farms, NiV's distinct transmission patterns and clinical presentation necessitated further investigation. After months of meticulous research, the virus was successfully isolated from the cerebrospinal fluid of a patient, formally identifying NiV as the causative agent. Notably, the epidemic primarily impacted male pig farm workers, with minimal cases reported in young children. Fever, headache, and decreased consciousness were the most prevalent symptoms among affected individuals. Reported case and fatality figures for the Malaysian outbreak vary slightly depending on the source, ranging from 238 to 265 cases and 105 to 109 deaths, respectively. This high mortality rate, subsequently confirmed in subsequent outbreaks, underscored the severity and urgency of understanding NiV's pathogenesis and control strategies.

➢ ***Singapore:***

In March 1999, Singapore faced a concerning public health challenge: Nipah virus infections emerged among slaughterhouse workers handling pigs imported from Malaysia. The virus attacked both the respiratory system and the brain, causing illness in 11 men between the ages of 24 and 66. Sadly, one life was lost during this outbreak. Notably, all affected individuals were males, mostly of Chinese ethnicity with one Indian individual.Faced with this new threat, Singapore implemented a ban on the import of pigs from Malaysia.

➢ ***Bangladesh:***

Between 2001 and 2013, Bangladesh experienced a series of Nipah virus outbreaks, predominantly during winter months, affecting numerous regions, some recurrently. Initial outbreaks exhibited a concerningly high mortality rate, rising from 69% in 2001 to 83% by 2013. Over this 12-year period, 209 cases were recorded, with 161 fatalities. Research efforts elucidated unique epidemiological features of Bangladesh's Nipah strain. The virus was identified as an "etiological factor" in the 2004 Faridpur outbreak, hinting at potential environmental and cultural influences. identified distinct Bangladeshi-specific Nipah genomic sequences, establishing a separate genetic clade within the virus. Subsequently, a new classification system was proposed, separating the Malaysian and Cambodian strains (M genotype) from those isolated in Bangladesh and India (B genotype). A critical finding distinguishing the Bangladeshi strain was its unusual transmission routes. Studies highlighted the significance of date palm juice consumption and direct contact with infected individuals' secretions, including respiratory fluids. This novel understanding proved crucial for developing targeted prevention strategies specific to this unique Nipah variant.

➢ ***India:***

India's Nipah virus landscape, while less pronounced than in neighbouring Bangladesh, has witnessed its own series of outbreaks since 2001. The inaugural incident, occurring in West Bengal directly across the border from the Bangladeshi hotspot, marked a grim precedent with 66 cases and 45 fatalities, the highest recorded at the time. A subsequent outbreak in the same region in 2007 claimed five lives. However, it was the 2018 outbreak in Kerala that truly cast a spotlight on India's Nipah threat. Characterised by acute respiratory syndrome and encephalitis, this event recorded 18 cases and a devastating 17 deaths, exceeding 90% mortality and surpassing all previous Indian outbreaks. Phylogenetic analysis revealed the Kerala strain's close kinship with the Bangladeshi genotype B, while also exhibiting unique intra-clade variations. Further investigation utilising both human and bat samples confirmed the fruit bat reservoir and established a near-identical match between the human and bat viral strains. A solitary confirmed case in 2019, culminating in the patient's full recovery, offered a brief glimmer of hope. However, the tragic death of a 12-year-old boy in Kerala's Kozhikode district in 2021 served as a stark reminder of the virus's persistent threat, with subsequent contact tracing among family, friends, and healthcare workers yielding negative results, adding an element of uncertainty to the trajectory of India's Nipah narrative.

➢ ***Philippines:***

In 2014, the southern Philippines was struck by a deadly epidemic, with 17 cases and a staggering mortality rate exceeding 80%. The infections, primarily linked to horse meat exposure or consumption, were caused by a strain closely related to the Malaysian variant. Fruit bats were likely the strongest source of transmitting the infection to horses.[12]

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IV. PATHOGENESIS

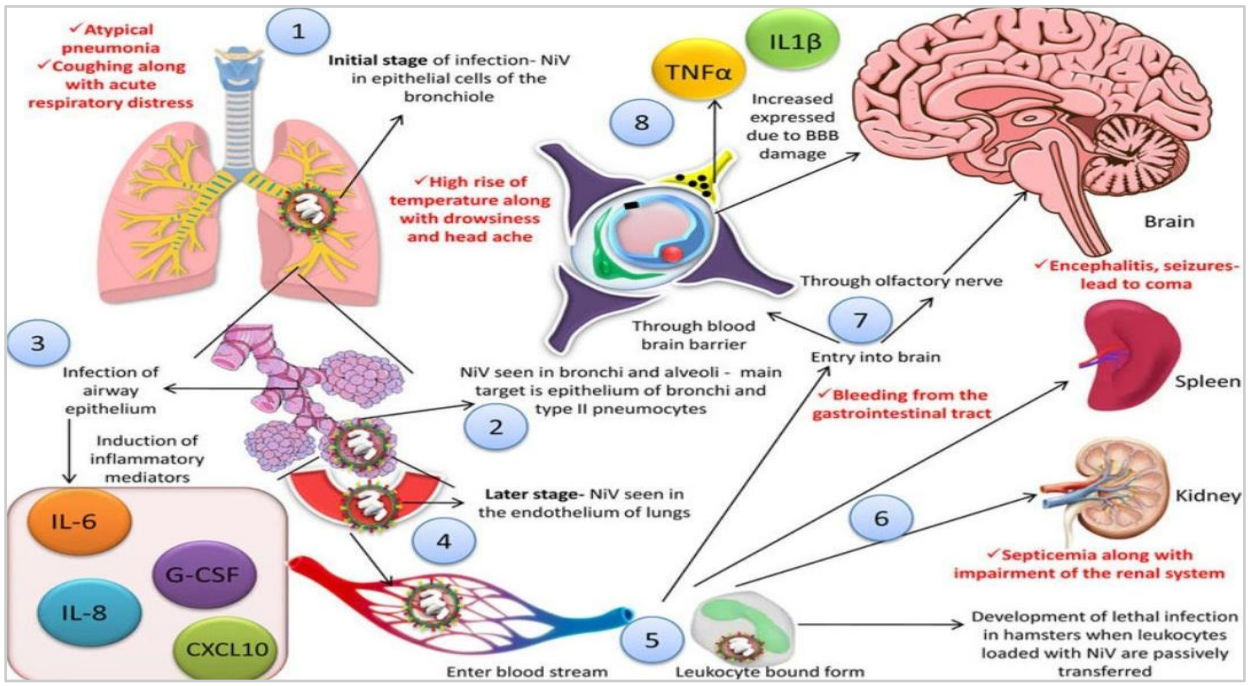

Fig.3 Adopted from Singh RK, Dhama K et. al, 2019

Nipah virus (NiV) exhibits a multi-stage pathogenesis marked by progressive dissemination and organ damage.

- Initial respiratory involvement: NiV initially targets the bronchiolar epithelium.
- Pulmonary progression: Viral antigen detection in bronchi and alveoli indicates further respiratory compromise.
- Inflammatory response: Airway epithelial infection triggers the release of proinflammatory mediators.
- Vascular endothelial cell invasion: In later stages, NiV invades the pulmonary endothelium.
- Hematogenous dissemination: The virus escapes into the bloodstream, either freely or within host leukocytes, facilitating multi-organ involvement.
- Multi-organ targeting: NiV reaches the brain, spleen, and kidneys .
- Central nervous system (CNS) access: NiV utilises two pathways for CNS entry:
  - Hematogenous route
  - Anterograde transmission via the olfactory nerve.
- Blood-brain barrier disruption and neuroinflammation:
- infection disrupts the blood-brain barrier, with tumour necrosis factor (TNF)-a are seen due to infection of the CNS by the virus which then leads to development of neurological signs.Symptoms seen in humans are red font.[13]

V. NIPAH VIRUS ORIGIN / HISTORY

The Nipah virus, a highly pathogenic zoonotic disease, first breached the species barrier leaping from its natural reservoir in fruit bats to infect both animals and humans. The Malaysian village of Sungai Nipah became the epicentre of this initial outbreak in 1998, with pig farms acting as the primary transmission bridge. Nearly 300 human cases were documented, resulting in over 100 tragic fatalities within a year. Initially understood as a porcine-driven neurological and respiratory illness, the Nipah virus's zoonotic potential soon became evident, posing a significant threat to both animal and human populations. While searching for the natural host of Nipah virus, researchers stumbled upon an unexpected discovery: a completely new paramyxovirus in the urine of flying fox bats.[27]

- *Clinical Signs And Symptoms*

Following exposure to the Nipah virus (NiV), symptoms typically appear within two weeks, ranging from 4 days to 2 months. Fever, headache, dizziness, and vomiting are common initial signs, potentially progressing to severe encephalitis.[14] Neurological involvement can be diverse, affecting the brainstem, cerebellum, and meninges.[4] A unique feature is the possibility of relapse or late-onset encephalitis even years later.[15] Survivors may experience psychiatric issues, like depression and often personality changes are seen with attention deficits.[16] Especially in the advanced stages of the disease Neurological manifestations are well-documented in severe cases of human outbreaks in Malaysia with development of ARDS (50–60%).[17] While asymptomatic infections occur, their prevalence remains unclear. In various studies the incidence of subclinical infections ranges from 1% to 45%[18]. Vomiting, dysphagia and myalgia are also described.[19] Recognizing uncommon manifestations and maintaining a high degree of suspicion in high-risk areas is crucial for early diagnosis and intervention.[20]

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➢ *Nipah Virus Diagnostic*

For diagnosing Nipah virus (NiV), several methods are employed. The gold standard is virus isolation in Vero cells, which typically shows characteristic cytopathic effects like syncytia formation and cell death, accompanied by vasculitis, within 3 days. Suitable sample types include cerebrospinal fluid, respiratory swabs (preserved in viral transport medium), blood, and urine. However, all testing must be conducted in Biosafety Level 4 (BSL-4) laboratories due to the highly contagious nature of the virus.

Figuring out Nipah virus can be tricky, but we have tools like blood tests and DNA checks to help. ELISA is a quick and cheap blood test that looks for virus "soldiers" your body makes, but it's not always accurate. Recently, the conventional polymerase chain reaction (PCR) technique, once considered standard, has been superseded by increasingly sensitive and specific methods. These include conventional reverse transcription PCR (RT-PCR), nested RT-PCR, real-time RT-PCR employing intercalating dyes (qPCR), real-time RT-PCR utilizing hydrolysis probes (TaqMan), multiplex bead-based real-time RT-PCR, and the RT-LAMP assay. RT-PCR assays for NiV have focused on a highly preserved section within the N, M, or P gene of the viral genome.[12]

➢ *Differential Diagnosis*

When a patient presents with fever, encephalitis (brain inflammation), or ARDS (acute respiratory distress syndrome), especially in areas with Nipah outbreaks or travel history, it's crucial to consider both Nipah and other possible causes. While these symptoms can appear in many illnesses, accurately pinpointing the culprit is key for optimal care.

Patients showing signs of Nipah infection should be evaluated for the following potential differentials:

- Japanese encephalitis (JE)
- Measles
- Rabies
- Dengue encephalitis
- Cerebral malaria
- Scrub typhus
- Leptospirosis
- Herpes encephalitis
- Bacterial meningitis.[17]

➢ *Treatment*

Given the absence of an effective drug against NiV, patient management primarily relies on supportive and prophylactic measures. Upon confirmation of NiV infection, clinical practices involve maintaining airway patency, preventing venous thrombosis, and managing fluid and electrolyte balance. Mechanical ventilation is utilised for severe respiratory symptoms, while broad-spectrum antibiotics are administered to infected individuals.

Several compounds have been investigated in the quest for a NiV-inhibiting drug. The efficacy of ribavirin, used during the Malaysian epidemic, remains uncertain, as does that of acyclovir in Singapore. Chloroquine, an antimalarial drug, demonstrated effectiveness in inhibiting NiV in cell cultures but lacked confirmation in animal models. Encouraging outcomes emerged following the application of Favipiravir (T-705) and monoclonal antibodies m102.4 in animal studies, with the latter advancing to phase I human trials. Eighty-six treatment-related adverse events were reported, with comparable rates between placebo and treated groups, and no fatalities occurred.

Laboratory experiments examined the effectiveness of GRFT (Griffithsin) and its synthetic trimeric tandemer (3mG) in inhibiting the viruses NiV and those from four other families. Initial testing in live animals demonstrated that oxidation-resistant GRFT (Q-GRFT) provided substantial defence against deadly NiV infections in golden Syrian hamsters. GRFT, a lectin that binds to high mannose oligosaccharides, displayed wide-ranging antiviral activity in live organisms.[12]

➢ *Therapeutics and Vaccines*

While the 1998-1999 outbreak of Nipah in Malaysia left scars, it also yielded valuable insights. Notably, the antiviral drug ribavirin showed a 36% reduction in mortality among those with Nipah encephalitis compared to non-treated groups. Although not a definitive cure, it offered a flicker of hope in the face of this deadly virus.But the battle against Nipah is far from over. There are currently no commercially available vaccines or approved therapeutics for Nipah, leaving supportive care as the primary weapon. Recent research, however, has unveiled a promising target: the Ephrin-B2 receptor. Both Nipah and its close cousin, Hendra virus, utilise this receptor to gain entry into cells. This shared vulnerability opens an exciting avenue for developing effective treatments. Fusion inhibitors that block Ephrin-B2 expression could potentially form the backbone of future vaccines and drugs. While it's still early days, the identification of Ephrin-B2 as a common entry point for Nipah and Hendra viruses brings us a step closer to effective interventions. This discovery marks a significant milestone in the fight against these deadly diseases, offering a future where treatment and prevention become reality.[25]

➢ *Management and Control*

Nipah demands swift isolation and strict infection control. Treatment revolves around supportive care, ensuring stable breathing, circulation, and electrolyte balance. For severe pneumonia, mechanical ventilation, preferably invasive, is crucial. While specific antivirals remain in the pipeline, this comprehensive approach optimises recovery in Nipah infection.

Prognosis Nipah virus packs a punch, with a death toll ranging from 40% to 100%. Age and severe brain-stem involvement, marked by reduced consciousness, vomiting, unusual eye movements and pupil dilation, high blood pressure, and rapid heartbeat during illness, were associated with worse outcomes in the Malaysian outbreak. This highlights the importance of early intervention and aggressive supportive care for those battling Nipah.[23]

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➢ *Disease Prevention*

The risk of illness and death among healthcare workers caring for Nipah patients is deeply concerning. While uncertainties remain about its transmission, we can still draw practical lessons from past outbreaks like Ebola and SARS to safeguard our healthcare workforce. The bedrock of prevention lies in standard infection control practices, meticulous hand hygiene, and appropriate personal protective equipment (PPE).

- *Building Defence At Every Level:*

By implementing these measures, we can build a robust line of defence against Nipah in healthcare settings, protecting our vital workforce and ultimately saving lives. [17]

Fig.4 Disease Prevention

➢ *Complications*

Studies paint a grim picture of Nipah virus encephalitis (NiV encephalitis), highlighting the crucial need for early diagnosis to improve survival and recovery rates. This disease carries a staggeringly high case fatality rate, with reported mortality ranging from 40% to a shocking 91% in various studies. Even for those fortunate enough to survive NiV encephalitis, the battle is far from over. A significant portion of survivors experience persistent neurological and cognitive problems, including debilitating depression, memory deficits, and chronic fatigue. Functional impairment, hindering daily activities, is also a frequent consequence of this devastating disease.[10,28]

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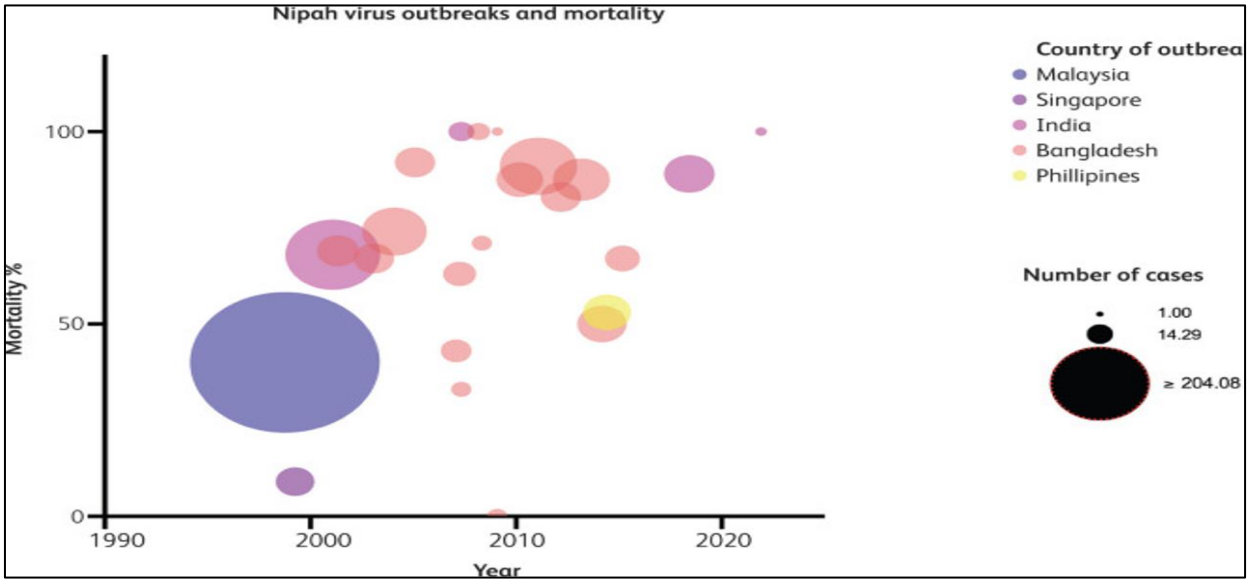

Fig.5 Adopted from Alam AM, 2022

➢ *Clinical Trials*

A promising development in the fight against Nipah virus emerges as the National Institute of Allergy and Infectious Diseases (NIAID) initiates an early-phase clinical trial for an investigational vaccine. Developed by Moderna, Inc., in collaboration with NIAID's Vaccine Research Center, the mRNA-1215 vaccine leverages the same mRNA platform utilized in several successful COVID-19 vaccines. This Phase 1 trial, conducted at the NIH Clinical Center in Bethesda, Maryland, aims to evaluate the vaccine's safety, tolerability, and immunogenicity in 40 healthy adults aged 18-60 years. Employing a dose-escalation design, the study will administer the vaccine in two doses via shoulder muscle injections, spaced either four or twelve weeks apart. Four cohorts of ten participants each will receive different dosages: 25 micrograms, 50 micrograms, and 100 micrograms, with the fourth group's dosage determined by an interim analysis. Throughout the year-long follow-up period, participants will undergo regular clinical assessments and blood draws to monitor their responses to the vaccine. This initial study represents a crucial step towards developing a potential Nipah vaccine. The mRNA-based platform offers advantages in rapid development and scalability, providing hope for an expedited timeline compared to traditional vaccine development methods. Should safety and efficacy be established, subsequent larger-scale trials will be necessary to confirm broader applicability and public health impact. While the fight against Nipah remains ongoing, this trial marks a significant milestone in the quest for effective prevention strategies. The dedication of NIAID, Moderna, and the study participants paves the way for a future where protecting communities from Nipah infection becomes a reality.[26]

VI. CONCLUSION

In conclusion, this review provides a comprehensive overview of the Nipah virus, highlighting its complex transmission dynamics, diverse clinical manifestations, diagnostic challenges, and limited treatment options. Despite advances in understanding this deadly pathogen, significant gaps remain in our knowledge, particularly regarding its reservoirs and potential for future outbreaks. Moving forward, concerted efforts are needed to enhance surveillance, develop effective therapeutics, and strengthen public health infrastructure to mitigate the threat posed by Nipah virus. Collaborative research and international cooperation are essential for advancing our understanding of this virus and improving our ability to prevent and control its spread. By investing in research, surveillance, and preparedness, we can better protect global health security and safeguard against the devastating impact of Nipah virus outbreaks.

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CONFLICT OF INTEREST

Authors do not have any conflict of interest with any individual.

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