Inside the Outbreak: Science and Prevention of Nipah Virus

[1]Riddhi Vadnere, [2]Gitanjali Bhatjire, [3]Dr. Atul Bendale, [4]Anil Jadhav
[1]Student, Mahavir Institute of Pharmacy, Mhasrul-Varvandi Road, Nashik, Maharashtra 422202.
[2]Assistant Professor, Mahavir Institute of Pharmacy, Mhasrul-Varvandi Road, Nashik, Maharashtra 422202.
[3]Head of Department, Mahavir Institute of Pharmacy, Mhasrul-Varvandi Road, Nashik, Maharashtra 422202.
[4]Principal, Mahavir Institute of Pharmacy, Mhasrul-Varvandi Road, Nashik, Maharashtra 422202

***Abstract*—Nipah virus, an newly discovered zoonotic paramyxovirus of the henipavirus genus, continues to creates a threat to global health due to its high invading rate, recurring spillover events, and limited therapeutic options. This comprehensive review synthesizes current knowledge on niv epidemiology, transmission dynamics, clinical presentation, diagnosis, treatment, and preventive strategies across regions affected since its emergence in malaysia in 1998. Outbreak analyses from Philippines, Singapore, Bangladesh, Malaysia and India highlight the virus's complex ecology, shaped by human animal interfaces, pteropus bat reservoirs, and region-specific exposure patterns such as date palm sap consumption and livestock contact. The review underscores the absence of licensed antivirals or vaccines, although experimental candidates such as ribavirin, favipiravir, monoclonal antibodies, viral-vector vaccines, and subunit g-glycoprotein platforms show promising preclinical efficacy. A strong emphasis is placed on the one health approach, integrating human, animal, and environmental surveillance to strengthen outbreak preparedness, as demonstrated effectively in kerala's coordinated response. Future perspectives highlight the need for enhanced genomic surveillance of bats, real-time sequencing during outbreaks, ecological monitoring, and the development of rapid diagnostics and scalable vaccines. Collectively, this review emphasizes that sustained research, robust surveillance, cross-sector collaboration, and community level interventions remain crucial for mitigating the ongoing and future risk of niv epidemics.**

***Index Terms*—Nipah virus, Zoonotic spillover, Henipavirus epidemiology, One Health approach, Outbreak preparedness.**

I. INTRODUCTION

Both Nipah virus (NiV) and Hendra virus have long been associated with severe, often fatal respiratory and neurological disease in animals and humans [1]. It is a single-stranded RNA virus belongs to the family; Paramyxoviridae, is classified under the Henipavirus genus alongside this virus and the more recently identified Cedar virus. Fruit bats of the Pteropus genus are recognized as the primary natural reservoirs for these viruses, although Cedar virus has not yet been linked to clinical disease [2]. Due to its high fatality rate and capacity to cause repeated outbreaks, NiV is listed by the World Health Organization as a priority pathogen requiring accelerated research and development [3]. Since its first detection during the 1998 Malaysian outbreak, the virus has continued to cause recurrent episodes across South and Southeast Asia, infecting multiple mammalian hosts and posing a persistent public health threat through both zoonotic and person-to-person transmission [4].

The global distribution of Pteropus bats suggests that regions within their ecological range remain vulnerable to future spillover events, a concern highlighted by the recent appearance of NiV cases in a previously unaffected district of Kerala, India [5]. Epidemiological investigations in Malaysia also revealed that Malay villages reported no cases despite being located near affected Chinese-owned pig farms; adherence to Islamic practices that restrict contact with pigs likely lowered their exposure risk [6]. Following the COVID-19 pandemic, pathogens with high epidemic potential particularly NiV have drawn increased scientific attention. Identified initially in 1998 [7], NiV remains one of the WHO's top priority agents due to its high virulence and continuous emergence [8], while global health organizations such as CEPI [9] and the UK Vaccine Network [10] have prioritized it for vaccine development.

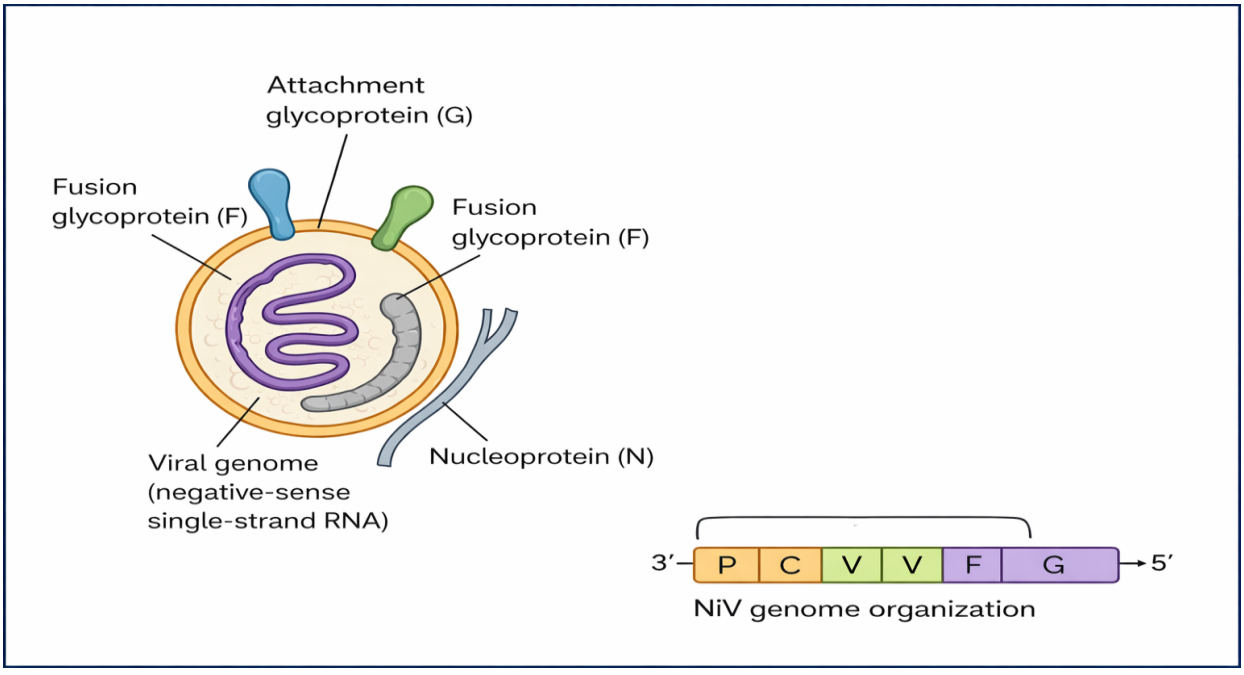

Figure 1: Structure of Nipah Virus and its Genome organization.

NiV is an enveloped virus containing a negative-sense, single-stranded RNA genome and belongs to the genus Henipavirus within the Paramyxoviridae family [11,12]. Its genome is non-segmented, measuring roughly 18 kb in length [12,13,14,15]. This genome translates six essential structural proteins: the nucleoprotein (N), phosphoprotein (P), matrix protein (M), fusion glycoprotein (F), add-on glycoprotein (G), and the large RNA polymerase (L). Additionally, the P gene gives rise to three accessory proteins W, V and C through mRNA editing and alternative translation initiation. Comparative genomic studies have revealed two primary Nipah virus lineages: the M genotype, which encompasses Malaysian isolates (NiV-M), and the B genotype, which includes strains identified in Bangladesh (NiV-B) and India (NiV-I) [13,16,17]. Although these variants exhibit high genomic similarity (with NiV-M and NiV-B sharing approximately 91.8% homology, and NiV-I showing 85.14-96.15% similarity to both), infections caused by B-genotype strains are reported to be more severe than those associated with the M genotype [16,18,19,20].

II. METHODS

A comprehensive literature search was carried out using the electronic databases pubmed, google scholar, and the cochrane library. Search terms included the following mesh keywords and related phrases: “nipah virus infection,” “nipah virus epidemiology,” “clinical features of nipah virus,” “nipah virus diagnosis,” “nipah virus surveillance,” “nipah virus vaccine,” “nipah virus malaysia,” “nipah virus bangladesh,” “nipah virus india,” and “nipah virus philippines.”

III. NIPAH VIRUS OUTBREAK

Nipah virus (niv) was first identified in malaysia as a disease circulating in pig farms that subsequently infected humans, triggering a major encephalitis outbreak with approximately 40% fatality, totaling 276 confirmed cases and 106 deaths.

3.1. Malaysia-Singapore Outbreak (1998–1999):

Retrospective investigations traced the epicenter to a pig farm near the village of ipoh, where the earliest human infection was documented in january 1997. Through the movement and sale of livestock, the virus spread across peninsular malaysia and later reached singapore during 1998–1999 [21,22]. Subsequent transmission from infected pigs to farm workers and others in contact with livestock triggered the human outbreak [23].

3.2. Bangladesh

Since Nipah Virus Was First Identified In Bangladesh In 2001, The Country Has Experienced Repeated Outbreaks And Remains One Of The Regions With The Highest Number Of Documented Human Infections[24]. This Typically Occurs When People Consume Raw Date Palm Sap That Has Been Contaminated By Bat Secretions, Allowing Transmission Without The

Involvement Of Livestock. These Spillover Events Often Follow A Predictable Seasonal Pattern, Commonly Referred To As The "Nipah Season."[25,26,27]. Patients In Bangladesh Frequently Present With Respiratory Symptoms In Addition To Neurological Involvement, A Clinical Profile That Aligns With The Regular Occurrence Of Person-To-Person Transmission Through Respiratory Droplets. The Case Fatality Rate In These Outbreaks Is Notably High, With An Average Mortality Approaching Three-Quarters Of Those Infected.[21,25] . The epidemiology in bangladesh differs significantly from other affected regions. Since its first detection in 2001, seasonal outbreaks have repeatedly occurred during the winter months, predominantly in central and northwestern districts, commonly referred to as the "nipah belt," where most spillover events are reported [28]. Fruit bats of the genus pteropus are recognized as the natural reservoir for the virus [29]. While some patients have reported contact with pigs, livestock have not played a major role in outbreaks in bangladesh, likely due to differences in animal husbandry practices compared with malaysia. Most domestic animals are kept in small numbers by individual households, limiting opportunities for virus amplification among livestock [30]. Other animals, including cattle and goats, have shown evidence of niv exposure in serological studies, though their contribution to human transmission is minimal [31,32]

3.3 India

India has experienced several notable nipah virus (niv) outbreaks. Unlike in bangladesh, the consumption of date tribute sap is not common in this area. As of june 1, 2018, 18 cases were confirmed, with 17 fatalities [33]. The affected individuals were primarily in the economically productive age group, and there was no significant difference in incidence between males and females [34]. During the 2001 siliguri outbreak, the source patient remained unidentified. Subsequent transfers of these patients to other hospitals led to further spread, infecting 25 healthcare staff members and eight visitors [35]. The 2007 outbreak began with a single person infected through consumption of alcohol derived from date palm sap, and subsequent cases, including a healthcare worker, resulted from close contact with this individual [36]. In the 2018 kerala outbreak, at least one healthcare professional contracted niv within a medical facility [37].

3.4. Existence of virus in kerala

In the past five years, kerala has faced multiple nipah virus outbreaks, with the latest reported in 2023. The outbreaks, which occurred in 2018, 2019, 2021, and 2023, have largely impacted the kozhikode district and nearby regions, while the 2019 incident was recorded in the ernakulam district**.**[38]. In the 2018 kerala outbreak, every case apart from the index patient was linked to human-to-human transmission. Possible exposure routes included activities such as cleaning an abandoned well inhabited by bats or entering the nearby forested areas[39]. In 2021, the national institute of virology (niv), pune, reported that antibodies found in bat samples were associated with the rousettus genus, expanding earlier findings that had identified nipah-related strains in pteropus bats.[40]. During september, 2023, the kerala state government reported six laboratory-confirmed nipah infections, including two deaths, both among male patients aged 40 and 49 years.

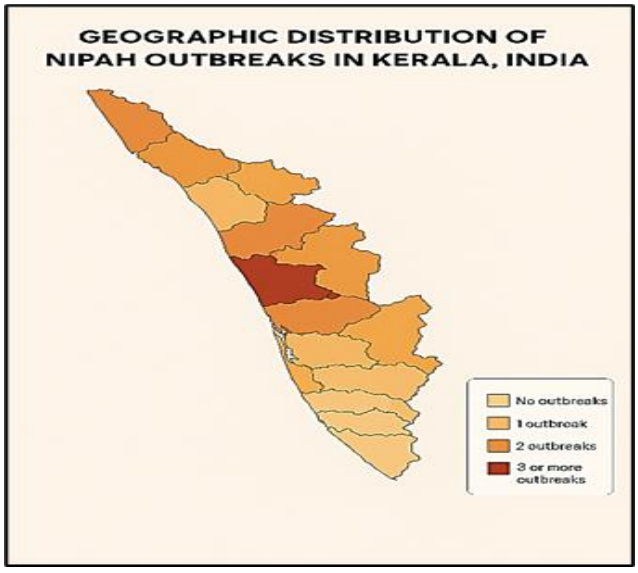

Figure 2:Geographical distribution in Kerala, India

3.5. Philippines

In 2014, the philippines experienced an outbreak of nipah virus (niv) infection involving seventeen confirmed patients, resulting in a case fatality rate of 82%. Many of the affected individuals ten in total reported either handling horses or consuming horse meat prior to falling ill. During the same period, ten horses also died, nine of which displayed neurological signs, although none of the animal samples were tested for niv. This finding raises the possibility that niv strains may evolve within bat populations or mutate during repeated spillover events, increasing the chances of enhanced transmission capabilities [41].

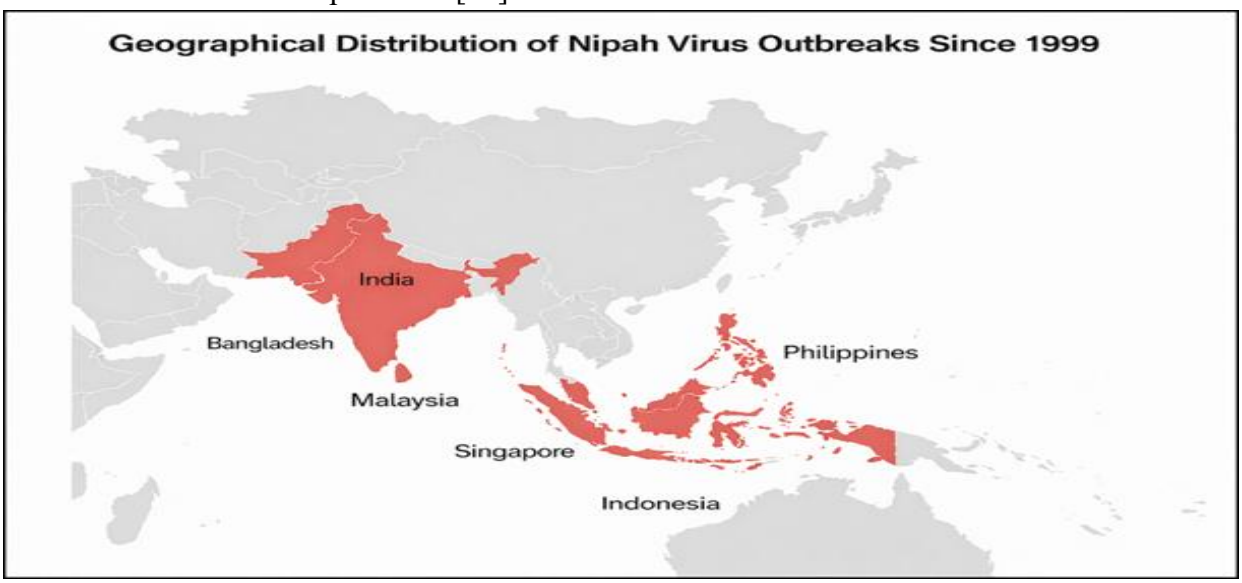

Figure 3: Since 1999, In Malaysia, singapore, bangladesh, india and the philippines reported niv's sporadic cases.

IV. CLINICAL PRESENTATION

In humans, the incubation period for nipah virus (niv) infection varies widely, ranging from approximately four days to as long as two months, although over 90% of cases manifest symptoms within the first two weeks of exposure [42]. Neurological features were broad and varied, involving conditions such as aseptic meningitis, widespread encephalitic changes, and localized brainstem pathology. A distinctive aspect of niv infection is the occurrence of relapsing or delayed-onset encephalitis, which may appear months or even years after the initial infection. In a cohort described by tan, among 160 individuals who survived their first episode of encephalitis, 12 patients (7.5%) experienced true relapses after initial recovery, while 3 patients (3.4%) developed late-onset encephalitis despite having no neurological involvement during their acute illness [43]. Remarkably, the most prolonged interval before the appearance of late-onset encephalitis documented in the literature was 11 years [44].

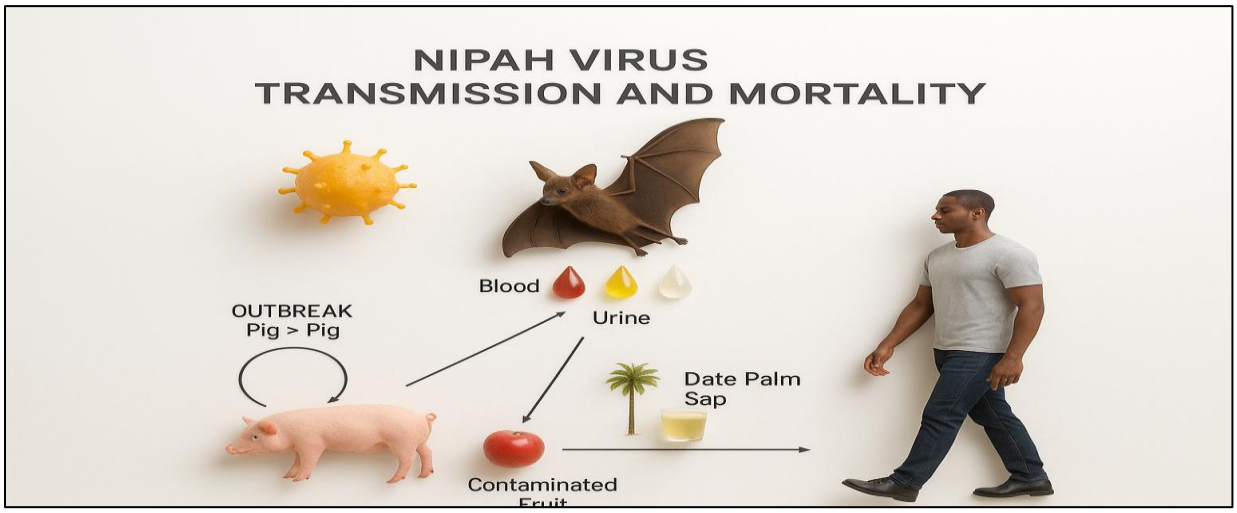

Figure 4: Transmission dynamics of nipah virus.

4.1 Respiratory involvement

Although nipah virus infection is primarily recognized for its neurological manifestations, involvement of the respiratory system has been reported across multiple outbreaks. In malaysia, respiratory features were observed in approximately 14-29% of infected individuals, but it remained uncertain whether these symptoms were part of the early clinical picture or developed later due to complications such as aspiration or ventilator-associated pneumonia.

4.2 Neuroradiology findings

During the malaysian outbreak, brain mri examinations often demonstrated widespread abnormalities involving the cerebral cortex, temporal lobes, and pons. Individuals who later experienced recurrent symptoms or delayed-onset encephalitis typically showed numerous irregular and merging cortical lesions on imaging.[45]. In contrast, the radiological pattern seen in singapore patients differed significantly. Their scans commonly displayed many tiny, bilateral lesions usually under one centimeter situated in the subcortical and deep white matter(fig no.5)

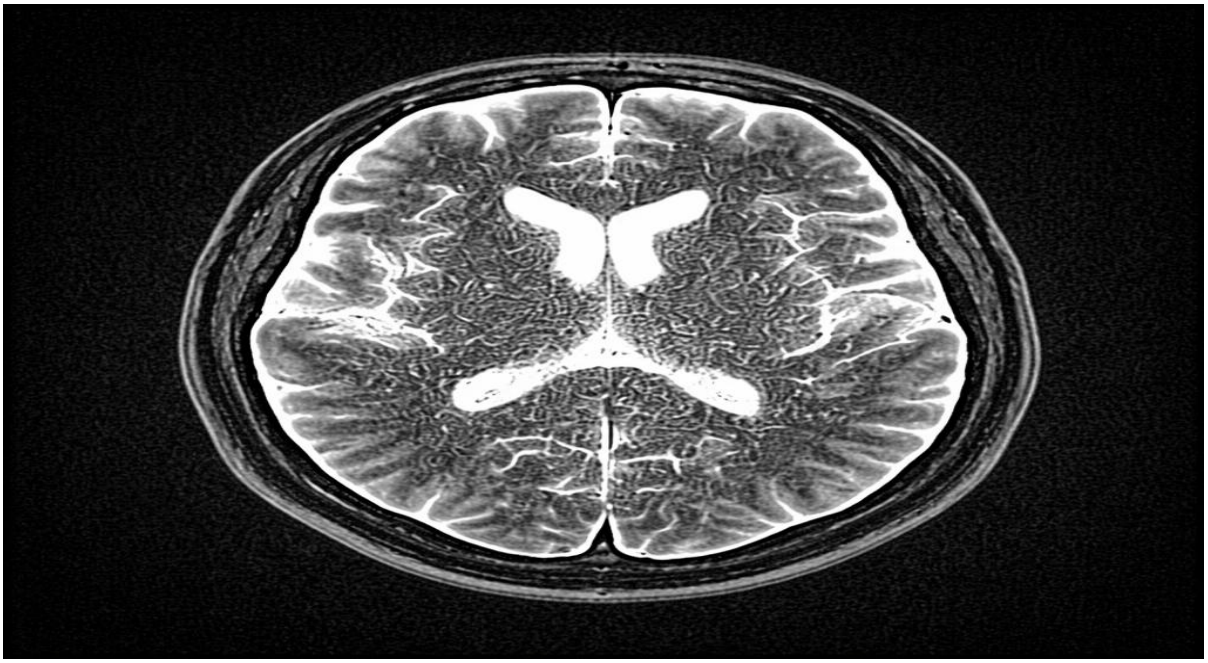

Figure 5: Typical mri pattern of multiple small white matter lesions. (a) multiple punctate white matter lesions. (b) the largest lesion is more prominent on corresponding diffusion-weighted image

V. DISCOVERY OF THE VIRUS

Researchers identified a previously unknown virus while examining at the university of Malaya in 1999, cerebrospinal fluid from a patient suffering from severe encephalitis. When the samples from several fatal cases were introduced into vero cell cultures, the cells exhibited prominent syncytia formation, indicating viral infection. Subsequent electron microscopy revealed structural characteristics consistent with members of the paramyxoviridae family. The virus was later named nipah virus, reflecting its first isolation from a patient in kampung sungai nipah, a community located in negeri sembilan [46].

VI. PATHOGENSIS

For evaluating genetic relationships, representative fragments of approximately 1599 bp from the n gene (27 isolates) and about 1809 bp from the g gene (15 isolates) were analyzed. Sequence data for niv isolates originating from malaysia, cambodia, bangladesh, india, and thailand submitted to the ncbi genbank database between 2001 and 2018 were included in the study.[47] phylogenetic reconstruction was carried out with the maximum likelihood (ml) approach using mega version 6.06, applying 1000 bootstrap replications for statistical support [48]. The optimal evolutionary models for dendrogram construction were selected through the “find best dna/protein model” feature in mega and cross-validated using the findmodel online platform (posada & crandall,

1998). Based on these assessments, the khy + g substitution model was chosen for the n gene, while the t92 model was identified as most suitable for the g gene.[49]

VII. ONE HEALTH APPROACH AGAINST NIPAH

By improving coordination among different sectors, the mission helps close operational gaps and enhances the nation's ability to respond quickly and effectively to public health threats. Through improved data-sharing and communication among major stakeholders, the program underscores the importance of the interconnected nature of human, animal, and environmental health an essential perspective for tackling zoonotic infections such as nipah virus (niv)[50]. This cross-sector collaboration supported early detection of the infection source, more effective containment strategies, and swift implementation of control measures. Kerala's integrated one health model demonstrated how coordinated action across multiple disciplines can significantly improve outbreak management and mitigation efforts[51].

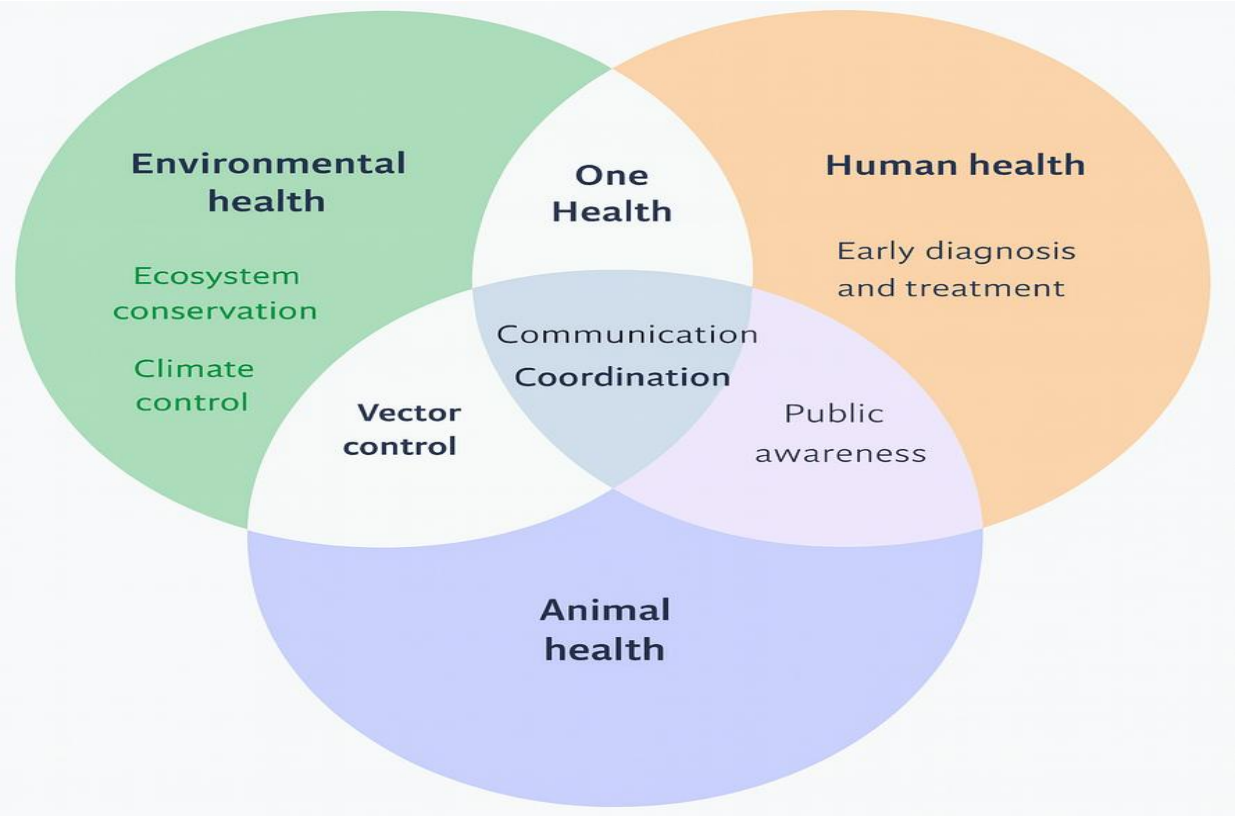

Figure 6:one health approach against nipah virus

VIII. TREATMENT

The clinical behaviour of nipah virus (niv) infection differs markedly from many other viral illnesses, largely because it can spread directly between humans and still lacks any approved antiviral therapy or licensed vaccine. [52,53] during earlier outbreaks, drugs such as ribavirin and

acyclovir were used in an attempt to reduce disease severity. Reports from the malaysian outbreak indicated that mortality decreased to approximately 36% when ribavirin therapy either oral or intravenous was administered, while acyclovir was utilised in singapore as part of clinical management. Nevertheless, the true therapeutic value of these agents has not been clearly established.[54,55]. Promising results have emerged from experimental studies evaluating favipiravir (t-705), which demonstrated notable antiviral activity in niv-infected hamster models[56].

IX. DIAGNOSIS

Clinical samples for nipah virus (niv) testing are obtained from individuals showing symptoms or during post-mortem investigations. As per the national centre for disease control (ncdc), india, recommended specimens include throat swabs placed in viral transport medium, along with urine, blood and cerebrospinal fluid. All samples must be handled with strict biosafety precautions and transported using triple-layer packaging at 2–8 °c; if retention beyond 48 hours is required, storage at −20 °c is advised. Although processing infectious material traditionally requires a bsl-4 laboratory, irradiation-based viral inactivation has been suggested as a potential method to allow safe handling of samples in bsl-2 settings [57,68].

X. MANAGEMENT AND CONTROL

Strict isolation of confirmed or suspected nipah virus (niv) cases is essential to prevent further transmission, along with adherence to comprehensive infection prevention and control procedures.

10.1 Antiviral Chemotherapy

Ribavirin, an antiviral commonly used against several paramyxoviruses such as respiratory syncytial virus, was administered during the nipah virus (niv) outbreak in malaysia[71]. Reported literatures showed a reduction in fatality rates with its use, findings from goh et al. [72] during the same outbreak did not demonstrate any clear survival benefit. Subsequent evaluations in various animal models have also indicated that ribavirin does not offer meaningful protection against niv infection [73]. Despite these limitations, the national centre for disease control (ncdc) continues to advise the use of oral or intravenous ribavirin for confirmed niv cases, although it does not recommend the drug for chemoprophylaxis [74]. Acyclovir was administered to some patients during the singapore outbreak, but its therapeutic value remains uncertain due to a lack of conclusive evidence [75]. Chloroquine showed antiviral activity in cell culture experiments; however, studies in hamsters revealed that it failed to prevent mortality, either alone or when combined with ribavirin [76]. Experimental work has also demonstrated that natural ephrin-b2 ligands along with soluble ephrin-b2 can inhibit niv infection under laboratory conditions [77]. Another antiviral, favipiravir, licensed in japan for influenza treatment, has been explored as a potential candidate, although further studies are needed to confirm its effectiveness against niv.

10.2 Surveillance

Routine monitoring for nipah virus (niv) activity is conducted in regions of bangladesh where the disease is known to occur. The national surveillance system uses both event-based and sentinel approaches to detect potential outbreaks. In addition, sentinel surveillance focuses on investigating clusters of encephalitis cases. A cluster is defined as two or more patients presenting within a 21-day period and residing within approximately a half-hour walking distance of each other [78].

10.3 Vaccines

Multiple vaccine platforms targeting nipah virus (niv) have been explored, and several have progressed through testing in different animal models. One of the most extensively investigated strategies involves a subunit vaccine based on the viral g glycoprotein (sg) of both niv and hendra virus (hev). Notably, the hev-derived sg antigen has been shown to induce cross-protective immunity against infections caused by either virus [78,79,80,]. This research ultimately contributed to the development of equivac hev, a licensed hev vaccine for horses currently registered and used in australia. In addition to subunit vaccines, several recombinant viral-vector vaccines have been engineered[81].

XI. PREVENTION

Human exposure through consumption of contaminated food products also requires attention. Raw date palm sap or similar products can become contaminated by bats, making avoidance essential. Since discouraging the intake of traditionally consumed fresh sap may conflict with cultural practices, more practical and culturally acceptable interventions such as installing bamboo skirts or other physical barriers to prevent bats from accessing sap during collection have shown promise [69]. Several vaccine platforms have demonstrated strong protective efficacy in preclinical research using both small-animal models and non-human primates. Among these, vaccine candidates based on vesicular stomatitis virus vectors appear particularly advanced, having provided full protection in hamsters, ferrets, and african green monkeys [70]. Ultimately, effective vaccination strategies will likely need to include not only human populations at risk but also susceptible livestock species such as pigs and potentially horses in regions where niv circulates endemically.

XII. FUTURE PERSPECTIVES

- Understanding ecological and environmental drivers

Future studies must focus on how climate change, deforestation, and habitat fragmentation influence the ecology of nipah virus. Altered bat migration, feeding behavior, and roosting patterns may increase human bat interactions, thereby elevating spillover risk.

- Host dynamics and transmission pathways
- Expanded genomic surveillance

XIII. CONCLUSION

Nipah virus continues to pose a serious public health challenge due to its high mortality and unpredictable spillover events. Changing ecological conditions and close human animal interactions play a critical role in its transmission. Although advances in surveillance and diagnostics have improved outbreak detection, effective vaccines and specific treatments remain limited. Strengthening integrated research, preparedness, and one health based interventions is essential to reduce the risk of future outbreaks.

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