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"Review on Nipah Virus: A Deadly Disease"

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I. INTRODUCTION

The Nipah virus (NiV) is an RNA virus in the Paramyxoviridae family. It is a member of the Henipavirus genus, which also includes the recently discovered Cedar virus and the Hendra virus (HeV). Henipaviruses are naturally stored in bats.[1] NiV and HeV induce fatal neurologic and/or respiratory illness, whereas Cedar virus has not been discovered to be pathogenic to any animals.[2] It is mostly spread by Pteropus spp., a particular kind of fruit bat. The Pteropus bats, specifically P. vampyrus, P. hypomelanus, P. lylei and P. Giganteu, were linked to outbreaks of the Nipah viral disease in no. of south and southeast Asian nations, including Bangladesh, Cambodia, East Timor, Indonesia, India, Malaysia, Papua New Guinea, Vietnam and Thailand.[3] . NiV is an extremely lethal virus that could endanger the safety of world health.

The National Institute of Allergy and Infectious Diseases (NIAID) and the Centres for Disease Control and Prevention (CDC) have classified this pathogen as a Category C priority pathogen. Its clinical manifestations include fever, encephalitis and in severe cases respiratory and pulmonary disorders affecting numerous body systems.[4] It was discovered that the initial human NiV infections were linked to contact with the swine and it was subsequently established that NiV could be isolated from pigs noses and oropharynxes.[6] Meningitis, encephalitis, and/or 14 day fever were the main symptoms of human infections, which also cause fast neurological degeneration and eventually led to coma within 24-48 hours.[7]

Hendra Virus, which was first appeared in 1994 in Australia, was the first member of this genus. Large scale human outbreak brought on by the Nipah Virus in Malaysia during 1994 and 1998, there were five outbreaks that followed between 2001 and 2005 [9].

Nipah is named after a Malaysian hamlet where the initial outbreaks was initially noted 1998-1999.[10] Between 1998 and 1999, Singapore and Malaysia reported the first case of acute febrile encephalitis in humans after it was first identified in Sungai Nipah (also known as Nipah River Village) where it got its name from.[5] NiV disease first emerged in Southeast Asia in September 1998 and by December 1999, the epidemic had caused 283 human encephalitis cases and 109 deaths in Malaysia and 11 cases with encephalitis or respiratory symptoms and 1 death in Singapore.[8] More than 250 cases of febrile encephalitis among agricultural and abattoir workers were associated with Nipah Virus (NiV) illness outbreak in Malaysia. This pandemic significantly disrupted society and the economy and generated widespread concern. The virus has caused epidemics in other regions of the world, namely in Bangladesh and India, despite the fact that no additional infections from Malaysia have been documented.[11] In May 2018, the Nipah Virus broke out again in Kerala, reigniting interest in this re-emerging illness. Numerous methods of NiV transmission, including human to human and food borne infection have been reported.[4]

Research on this syndrome has been hampered by the relatively small number of patients and difficulties in diagnosis. Because NiV is classified as a biological safety level 4 (BSL 4) pathogen, many countries have restricted access to these types of laboratories. Research on epidemiology, mechanisms of transmission, and practical preventative and control measures is desperately needed. The environment, domestic and peri-domestic animals and humans all need to be taken into account for a One Health approach to effectively control disease.[12]

Virology

The Nipah virus is an enclosed, single-stranded, non-segmented RNA virus with negative sense and helical symmetry. Six genes, namely nucleocapsid (N), phosphoprotein (P), matrix (M), fusion glycoprotein (F), attachment glycoprotein (G) and long polymerase (L) are arranged consecutively in the RNA genome from the 3´ to the 5´. The virus ribonucleoprotein (vRNP) is formed by the N, P and L bonded to the viral RNA. The host cell entry that follows the virion's cellular

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attachment is brought about via the F and G proteins. The fusion (F) protein fuses viral and cell membranes to allow for cellular entrance, whereas the G glycoprotein promotes binding to host cell surface receptors. When the NiV G protein attaches to host ephrin B2/3 receptors, it undergoes conformational changes that cause the F protein to refold. Viral control of host cell machinery has been recently shown to target the nucleolar DNA-damage response (DDR) pathway by inhibiting the nucleolar Treacle protein, which raises the production of Henipavirus (Hendra and Nipah virus). [13]

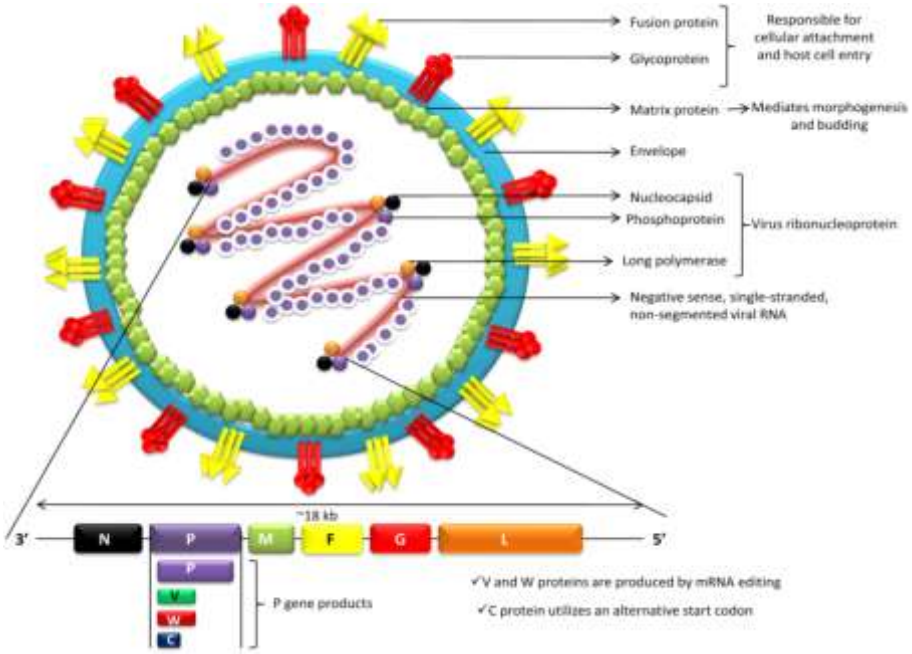

Fig 1: Structure of Nipah Virus

Transmission

Consumption of foods contaminated with the virus and contact with infected animals or human bodily fluids are the two main ways that NiV is spread. Close proximity such as touching, feeding, or staying with a virus-infected person, increases the risk of contracting a droplet NiV infection. Recent experimental research using aerosolized NiV in Syrian hamsters suggested that close contact could be a potential source of NiV transmission via NiV droplets (aerosol exposure). After research in Bangladesh, three routes of the Nipah virus's transmission have been found. The most common way is to consume fresh date palm sap; however, tari, or fermented date palm juice, can also be a potential means of viral transmission.[13]

Transmission in humans and animals

1. Fruit bats are a natural source of Nipah viruses. Date palm sap is the food source for fruit bats carrying the NiV virus. Viral infections can occur in high-sugar solutions, such as fruit pulp.
2. A virus spread to people by date palm sap ingestion.
3. Upon visiting these fruit trees, fruit bats of Pteropus spp., reservoirs of NiV, had the chance to naturally spill the virus-containing drop into the farm, contaminating the farm's soil and product.
4. Pigs and other animals eat contaminated fruits. Pigs serve as both an amplifying host and an intermediary. The close proximity of fruit trees, fruits such as date palm, fruit bats, pigs, and humans collectively serve as the foundation for the emergence and dissemination of new and deadly zoonotic virus infections like Nipah.
5. NiV-contaminated pork meat is exported to other regions.
6. Eating contaminated pork can expose humans to infection.
7. NiV can spread to other people through close contact with an infected individual.

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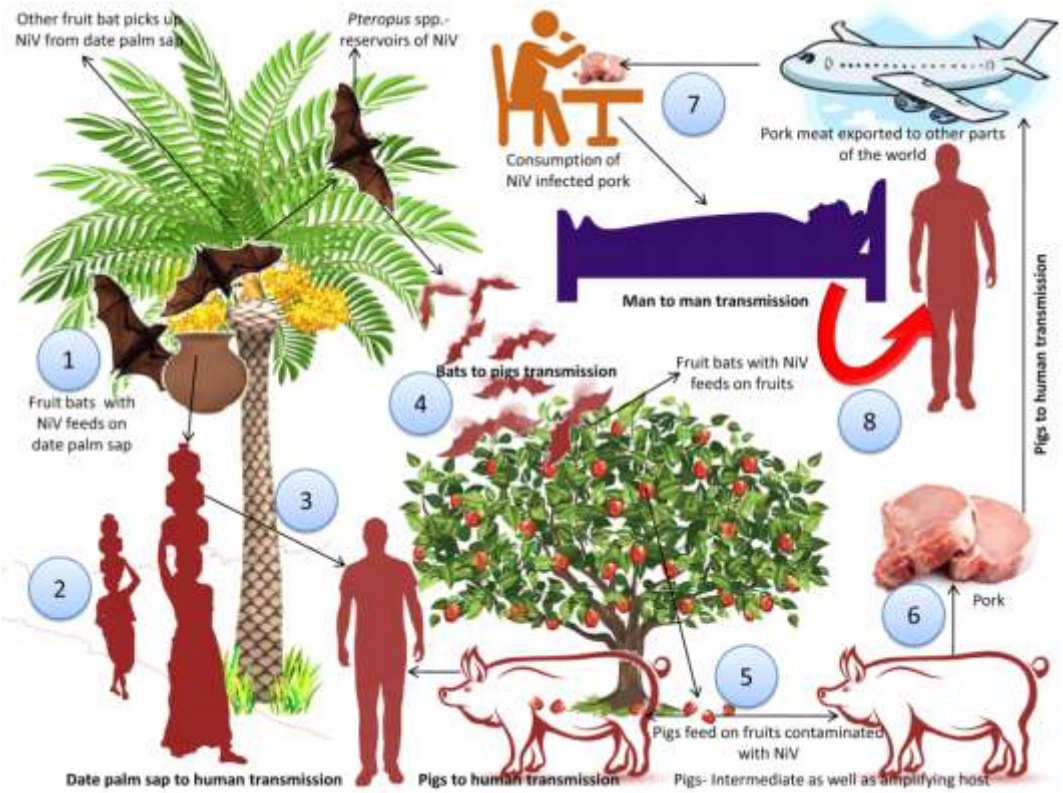

Fig 2: Transmission of Nipah Virus

Epidemiology

Since virus research requires a biosafety level 4 (BSL-4) laboratory facilities, the epidemiology of NiV remains incompletely understood. The flying fox, or Pteropus fruit bat (genus Pteropus, order Chiroptera), is thought to be one of the natural animal reservoirs for NiV. These flying foxes are found in Asia, China, Australia, parts of Africa, and the Pacific Islands. There are roughly sixty different species of them. It remains to be established experimentally whether the NiV infection causes these flying foxes to acquire subclinical illness or not. In Malaysia, Thailand, Bangladesh, and Cambodia, sero-surveillance investigations were carried out to detect NiV in flying fox samples. 9% to 25% of the bats were positive for the virus. Based on urine samples acquired from flying foxes in Malaysia and Cambodia, the NiV was isolated. Pigs served as a mediating host for humans in the Malaysian outbreak, whereas flying foxes served as NiV's native host. In the endemic locations, pigs became infected through indirect contact with flying foxes carrying the NiV virus. NiV outbreaks in humans have been reported from Bangladesh, India, Malaysia, and Singapore. Until June 2018, NiV was the cause of 643 laboratory-confirmed cases and at least 380 (59%) human deaths in five countries: Malaysia, Singapore, Bangladesh, India, and the Philippines.[14]

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Month/Year	Country	Location	No. of cases	No. of Deaths	% Fatality rate
Sept 1998-April 1999	Malaysia	Perak, Selangor, Negeri Sembilan states	265	105	39.6
Mar-1999	Singapore	Singapore	11	1	9
Jan-Feb 2001	India	Siliguri	66	45	68.2
Apr-May 2001	Bangladesh	Meherpur	13	9	69.2
Jan 2003	Bangladesh	Naogaon	12	8	66.7
Jan-Apr 2004	Bangladesh	Rajbari, Faridpur	67	50	74.6
Jan-Mar 2005	Bangladesh	Tangail	12	11	91.7
Jan-Apr 2007	Bangladesh	Kushtia, Naogaon, Natore, Pabna, Thakurgaon	18	9	50
Apr 2007	India	Nadia	5	5	100
Feb-Apr 2008	Bangladesh	Manikganj, Rajbari	11	9	81.8
Jan 2009	Bangladesh	Gaibandha, Nilphamari, Rangpur, Rajbari	4	1	25
Feb-Mar 2010	Bangladesh	Faridpur, Gopalganj, Kurigram, Rajbari	17	15	88.2
Jan-Feb 2011	Bangladesh	Comilla, Dinajpur, Faridpur, Lalmohirhat, Nilphamari	44	40	90.9
Jan 2012	Bangladesh	Joypurhat	12	10	83.3
Jan-Apr 2013	Bangladesh	Gaibandha, Manikganj, Naogaon, Pabna, Natore	24	21	87.5
Jan-Feb 2014	Bangladesh	13 Districts	18	9	50
Mar-May 2014	Philippines	Philippines	17	9	52.9
Jan-Feb 2015	Bangladesh	Faridpur, Magura, Naogaon, Nilphamari, Ponchoghor, Rajbari	9	6	66.7
May 2018	India	Kozhikode and Malappuram	18	17	94.4
Total			643	380	59

Clinical manifestation in humans

Patients with NiV infection can present with a variety of clinical symptoms, from atypical pneumonia with minor neurological illness and a low death rate to full-blown, fast advancing encephalitis that is lethal. The duration of incubation ranges from 4 to 21 days. It is unclear whether the infection's infectivity period coincides with the incubation period.[15]

Rapid acute encephalitis with an increased death rate is caused by NiV. During the Malaysian outbreak, the incubation period varied from 4 days to 2 months, and most cases reported within 2 weeks or fewer. Fever, headache, nausea, vertigo and lowered consciousness were the main clinical signs. Segmental myoclonus, hypertension, tachycardia, areflexia and hypotonia were among the distinctive clinical symptoms. The symptoms of encephalitis, particularly when the brainstem was impacted, were te most likely the direct cause of death. Patients who remained aware throughout the infection were less likely to experience signs like tachycardia and aberrant doll's eye reflex, which are linked to a poor progenesis[16]

Acute Signs/Symptoms

Except for one, every patient reported having an elevated temperature and experiencing overall weakness and exhaustion. 10 individuals (45%) reported having stomach issues, including nausea and vomiting. Seizures (5; 23%) and impaired mental status (17;77%) were the predominant neurological symptoms of acute illness. Electroencephalography was not carried out during an acute illness; instead, seizures were always identified based on clinical presentation and patient's reaction to antiepileptic medication. No one with acute sickness reported having tremor, myoclonus or any other movement problems.

Persistent neurological features

Persistence behavioural or personality changes, persistence subjective difficulties with memory or concentration, brain MRI abnormalities, cranial nerve palsies, myoclonus, persistence atacsia or gait issues, confluent hyperintensities on T2- and FLAIR weighted images, cortical atropy and persistence hyper intensities on T2- and FLAIR weighted images.[17]

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Volume 11, Issue 1 Jan - Feb 2026, pp: 689-702 www.ijprajournal.com ISSN: 2456-4494

Clinical manifestation in animals

The illness is also referred to as one-mile cough, barking pig syndrome in Malaysia and Porcine Respiratory and Encephalitis Syndrome (PRES) in pigs. Pigs under six months of age have been documented to have an acute febrile sickness, characterized by the development of a respiratory illness ranging from rapid laborious breathing to harsh non productive cough. Young piglets are the only exception to generally low death rate. Morbidity rates in confined animals can get close to 100%. The involvement of the neurological system can cause tremors, muscle twitches, weakness in the rear legs and varied degrees of flaccid or spastic paresis. In addition to seizures, nystagmus can also occur in boars and sows.

When dogs have NiV infection, they may experience renal syncytia development, glomeruli and tubule necrosis and lung inflammation. In cats, multiple organ vasculopathy may coexist with the development of endothelium syncytia. When experimental NiV infection occurs in variety of animals, including African Green Monkey, Guinea pigs, hamsters and chick embryos, it causes vasculopathy and lesions to develop in the CNS parenchyma. However clinical appearance lacking in both rats and mice.[18]

Pathology

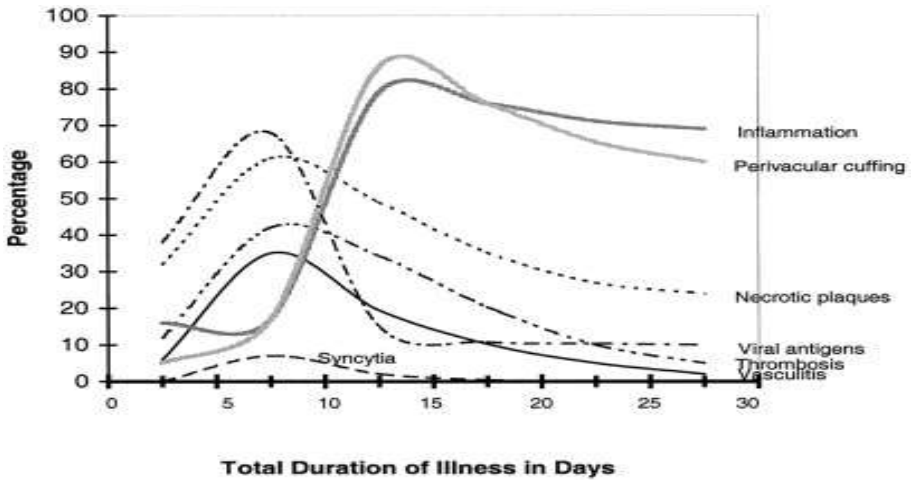
The microscopic characteristics lacked specificity. Lesions in the CNS were usually difficult to diagnose, but occasionally tiny, distinct, haemorrhagic, necrotic lesions were discovered in few patients. Out of 10 brains examined, only 2 had a clear herniation. Several organs, blood arteries and parenchyma showed histopathological alterations which are reported appropriately.

Blood vessels

It is noticed where immune-staining and histological lesions are distributed. When Nipah virus is present, there is a significant involvement of blood vessels in brain, lungs, heart and kidney. However CNS blood vessels were most seriously damaged. Vasculities were usually seen in tiny arteries, arterioles, capillaries and venules. Large arteries (such as aorta and pulmonary trunk) and medium sized vessels (such as renal artery and vein, anterior and middle cerebral arteries) did not exhibit vasculities. The current encephalitis case did not have any vasculities.

Pathologic findings	Brain no. %	Lung no. %	Heart no. %	Kidney no. %	Spleen no. %
Necrosis	28/30 (93)	17/29 (59)	1/29 (3)	10/29 (34)	10/24 (42)
Vasculities	24/30 (80)	18/29 (62)	9/29 (31)	7/29 (24)	0/24 (0)
Viral antigens	27/32 (84)	7/29 (24)	4/24 (17)	6/25 (24)	1/21 (5)

Table 1 : Pathological findings in blood vessels



International Journal of Pharmaceutical Research and Applications
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CNS

Vasculities, thrombosis, parenchymal necrosis, and the presence of viral inclusions were the primary pathogenic findings in the central nervous system. Whole CNS vascular involvement of gray and white matter was seen. Eight individuals had their spinal cords investigated, and three of them had pathological lesions that were identical to those seen in other parts of the brain. Spinal cords showed pathological lesions resembling those elsewhere in the central nervous system. Plaques in the gray and white matter showed varying degrees of necrosis. The round or oval-shaped necrotic plaques had sizes ranging from around 0.2 mm to more than 5 mm. Near these plaques, different degrees of parenchymal edema and inflammation, thrombosis, and vasculitis were commonly observed. Reactive microglia, lymphocytes, macrophages, and neutrophils made up the inflammatory cellular infiltration. The most frequent locations for microcystic degeneration were those near necrotic plaques. On rare occasions, microcystic alteration without any nearby plaques was also seen. Sometimes injured axons in white matter developed axonal spheroids resembling those observed in diffuse axonal injury. There were no significant regional infarctions of the kind linked to the blockage of medium- or large-sized arteries. Focal neuronophagia, microglial nodule development, and perivascular cuffing were observed elsewhere in the parenchyma. Parenchymal inflammation was found in 67% of patients overall.

Both the cytoplasm and the nuclei of neurons contained viral inclusions, however the latter were often more difficult to locate. Near necrotic plaques or vasculitic vessels, the majority of inclusions were discovered. Typically, cytoplasmic inclusions were eosinophilic, distinct, tiny, and occasionally numerous. With the exception of a narrow chromatin ring around the outside, nuclear inclusions were less frequently observed and occupied the majority of the nucleus. Despite the fact that inclusions were discovered in 63% of cases, after a thorough search, they were frequently only discovered in a small number of neurons.

The recurrent encephalitis patient had a slightly different CNS pathology than other cases. Viral inclusions were much more widespread and noticeable; they either filled the entire cytoplasm of the cell or just distributed more peripherally. The neuropil was likewise full of inclusions. The parenchymal lesions were more confluent, occasionally hemorrhagic, and accompanied by gliosis, a large number of macrophages, and substantial neuronal loss. Perivascular cuffing was not a significant characteristic, and there was no evidence of vasculitis or typical necrotic plaques.

Non CNS

62% of cases had lung vasculitis, and 59% had fibrinoid necrosis. Fibrinoid necrosis was frequently linked to small vessel vasculitis and frequently affected many neighbouring alveoli. Occasionally, alveolar gaps next to necrotic areas were found to have multinucleated giant cells with intranuclear inclusions. Aspiration pneumonia, pulmonary oedema, and alveolar haemorrhage were frequently seen. With the exception of one instance in which a big bronchus displayed substantial transmural inflammation and ulceration, histopathological alterations of the bronchiolar epithelium were rare.

The periarteriolar sheaths of the spleen displayed acute necrotizing inflammation together with a decrease of white pulp. Furthermore, the parenchyma of one case showed huge conspicuous multinucleated giant cells with intranuclear inclusions. There was no evidence of major blood vessel vasculitis. Large reactive mononuclear cells, hemophagocytosis, and sporadic necrosis were observed in lymph nodes. Multinucleated giant cells were infrequently seen in the lymph node cells lining the subcapsular sinusoids. In 34 percent of cases, localized glomerular fibrinoid necrosis was observed in the kidney. Inflammation in several cases completely destroyed the glomeruli. On rare occasions, interstitial inflammation, thrombosis, and vasculitis were observed (Figure 8A). Seldom was synchytial development observed involving the tubular epithelium and glomerulus' periphery. The transitional epithelium of the pelvis and renal calyx did not exhibit syncytia. In 31% of instances, vasculitis was seen in the heart. A patient who had been unconscious for more than two weeks had a significant myocardial infarction linked to vasculitis. Another patient who lived for almost a month showed signs of vasculitis-related localized cardiac fibrosis. Vasculitis has also been observed in the pancreas, adrenal gland, and mesentery, among other tiny arteries in other organs. The examination of the liver, skeletal muscle, and other tissues revealed no notable abnormalities.[19]

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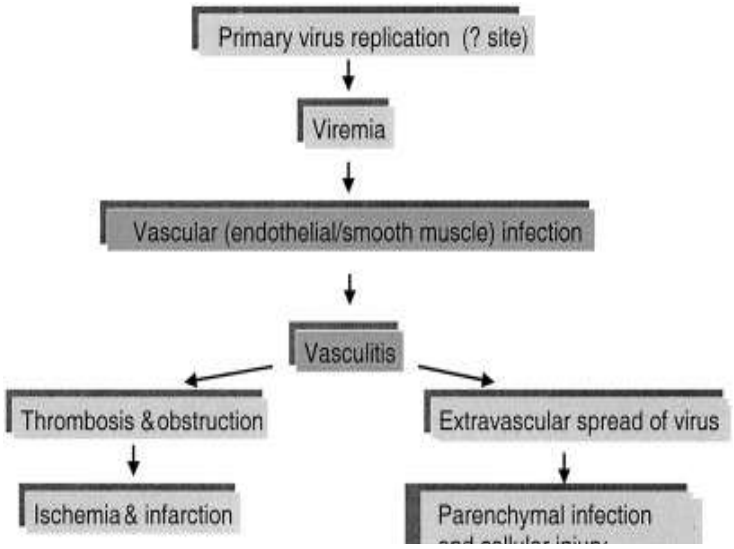

Pathogenesis of Nipah Virus Infection

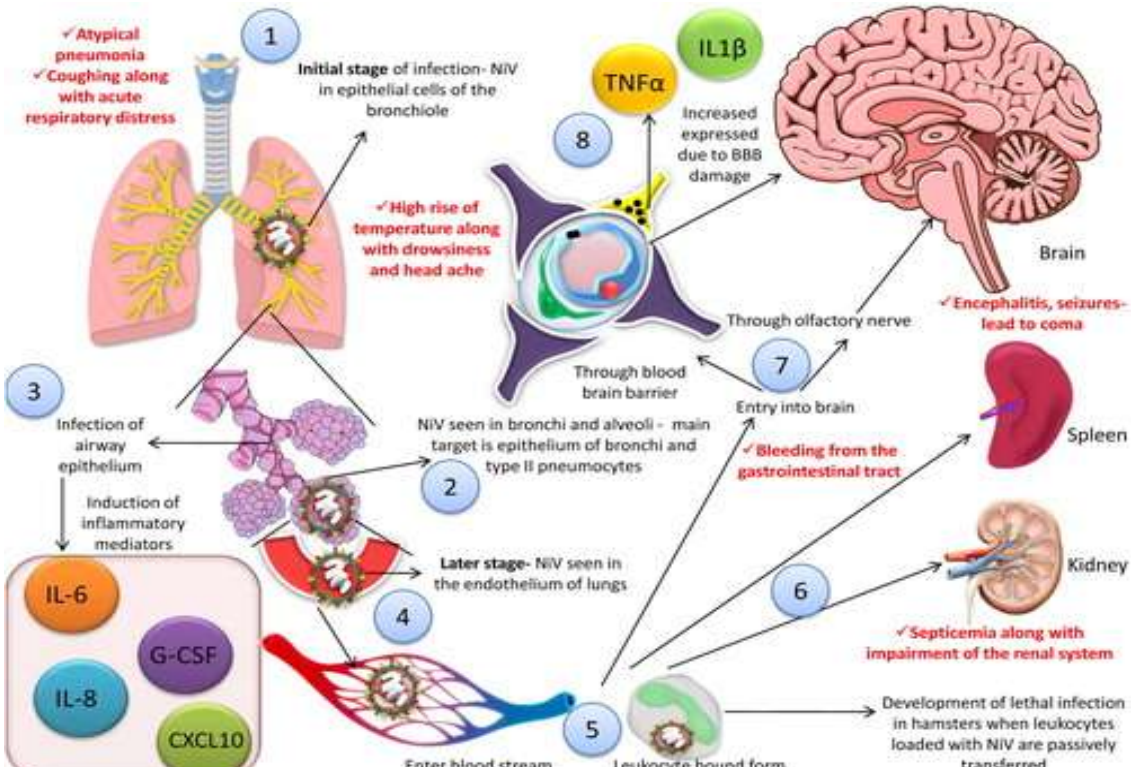

Fig 3: Pathogenesis of Nipah Virus

Diagnosis

Viral nucleic acid amplification assays, serological testing, and virus isolation can all be used to confirm NiV infections in humans and animals. NiV isolation and propagation require laboratory facilities that meet the requirements of Biosafety Level 4 (BSL-4). BSL-3, however, might work well enough to isolate the virus from suspected clinical materials in the first place. After the virus has been confirmed in infected cells (fixed with acetone) using an immunofluorescent method, the culture fluid needs to be transferred right away to a BSL-4 facility. In this regard, it is important to remember that the institutes responsible for managing NiV in Bangladesh are the Institute of Epidemiology Disease Control and Research

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(IECDR) and the International Centre for Diarrhoeal Disease Research, Bangladesh (ICDDRB). A BSL-4 laboratory has been set up at the National Institute of Virology (NIV) in Pune, India. The National Institute of Animal Health in Japan has created a monoclonal antibody-based immunohistochemistry diagnostic method.

ELISAs based on viral antigen capture provide a low-cost, high-throughput approach to screening questionable samples. The detection and separation of NiV from HeV have been reported using a monoclonal antibody-based antigen capture ELISA. For evaluating human and swine sera, indirect IgG ELISAs have been established, and an IgM capture ELISA employing a recombinant N protein of NIV has also been reported. It has been claimed that a sandwich ELISA using a rabbit polyclonal anti-NiV G protein is a rapid test for the diagnosis of the illness. NiV antigen-containing infected cell lysate can be utilized as a coating agent for ELISA testing. These antigens are supplied by the Centres for Disease Control and Prevention in Atlanta and the Australian Animal Health Laboratory (AAHL) in Geelong.

At the High Security Animal Disease Laboratory (HSADL), Bhopal, an ELISA based on recombinant N protein has been developed to evaluate pig serum samples. Under BSL-2 circumstances, a serum neutralization test for NiV can be carried out using pseudo typed particles. An artificial vesicular stomatitis virus that expresses secreted alkaline phosphatase (SEAP) is used in this assay. SEAP activity can be measured to determine the neutralization titre. Pig and ruminant serum, such as those of goats and cattle, have been utilized to detect antibodies against NiVsG, a glycoprotein, using the microsphere technique (based on luminex). Recently, ELISA has also been established to detect virus-specific antibodies in porcine serum samples utilizing recombinant full-length N protein and truncated G protein. Serum samples were first screened for Henipavirus infection using NiV N ELISA, whereas NiV G ELISA identified only NiV infections. These ELISAs are useful diagnostic tools for swine population seromonitoring, as well as maybe livestock and wildlife monitoring. NiV infection can be detected molecularly using techniques including reverse transcription polymerase chain reaction (RT-PCR), real-time RT-PCR (qRT-PCR), and duplex nested RT-PCR (nRT-PCR), with amplicon nucleotide sequencing providing confirmation. A novel set of primers aimed at the N gene has been published. In nRT-PCR assays for NiV RNA detection, internal controls could also be incorporated. Furthermore, in Thailand, two distinct viral strains from Pteropuslylei have been found thanks to this type of nRT-PCR. NiV infection diagnosis has also been found to benefit from the development of qRT-PCR techniques for Henipavirus detection. Reports of N gene-specific primer-based SYBR-Green I dye-based qRT-PCR have also been published. In order to quantitatively identify NiV replicative viral RNA, a unique one-step qRT-PCR technique targeting the intergenic region separating the F and G genes has recently been reported.This test avoids the need for viral mRNA amplification and may be more accurate than the traditional qRT-PCR. It is imperative to conduct an optimal exploration of advancements in the realm of diagnosing new zoonotic infections through an integrated One Health strategy.[20]

Direct detection of the agent

Polymerase chain reaction (PCR) is the most effective test for direct detection because of its high sensitivity, specificity, and quick reporting of results. Urine, swabs, tissue samples, and CSF are examples of specimens that may be utilized. Animal diagnosis by direct detection may be challenging due to the low sensitivity of virus detection.

PCR

The US Centres for Disease Control and Prevention (CDC) have developed conventional PCR that targets the N (nucleocapsid protein) gene. Real-Time PCR (RT-PCR) can detect NiV RNA in respiratory secretions, urine, or cerebrospinal fluid. These tests are frequently used for diagnosis because of their excellent sensitivity and specificity. A 2004-developed TaqMan probe-based test with a ~1 pfu sensitivity can identify the N gene. It can be used for diagnosis during an outbreak and is specific for NiV RNA. Additionally, an assay based on SYBR Green that targets a distinct region of the N gene has been created. It detects HeV and has a lesser sensitivity (~100 pfu).

Immunohistochemistry

Tissue that has been formalin-fixed can be utilized for immunohistochemistry. Vascular endothelium is the site of viral replication, hence a variety of tissues, including the brain, lung, spleen, kidney, and lymph nodes, may be employed. In pregnant animals, the uterus, placenta, and products of fertilization are also examined. Previously, immunohistochemistry was performed using

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human serum during convalescence. This has since been replaced by anti-NiV rabbit serum.

Virus isolation

A BSL-4 laboratory is required for the isolation of viruses from respiratory secretions, urine, cerebrospinal fluid, or other tissue specimens. The Vero cell line is the preferred cell line for both NiV and HeV. Additionally, pteroid bat cell lines have been created. It takes three days to notice cytotoxic effects. After the cells divide into syncytia, the syncytia rise off the surface, causing punctate holes to appear in the monolayer. Compared to HeV, NiV generates larger syncytia, and the two can be distinguished from one another based on variations in the distribution of nuclei inside the syncytia. By using PCR or immunohistochemistry, the virus from cell culture can be definitively identified.

Electron microscopy and sequencing, which are used to characterize viruses, are further tests that might be employed. They are not appropriate for primary diagnosis and are not always available.

Antibody detection

Serum or CSF containing IgM antibodies is used for diagnosis. IgG antibody detection is a useful technique for identification in reservoir animals during epidemiological investigations, as well as for surveillance in humans. During outbreaks, it has also been utilized for human diagnostics.

ELISA

Because of its great sensitivity, quickness, simplicity, and safety, it is the most widely used test for serological diagnosis. In Malaysia, the diagnosis was confirmed using ELISAs that were designed by the CDC for the detection of IgG and IgM. Since then, Bangladesh has utilized it for surveillance during NiV outbreaks. The highly conserved N antigen has been used to produce further recombinant protein-based diagnostics. 50% of patients had detectable IgM antibodies on day 1 of sickness, and after day 18, 100% of patients have positive IgG antibodies. The presence of IgG lasts for several months.

Serum Neutralisation Test

Although it needs to be done in a BSL-4 facility, this test is regarded as the gold standard. Test sera are allowed to infect Vero cells after being cultured with the virus. Test results can be examined three days after testing, as positive sera prevent the onset of cytopathic consequences. A modified neutralization test has been created that can be read 24 hours a day. Here, the viral-serum mixture is extracted following an adsorption period, and the virus is detected by immunostaining. One possible application for pseudo typed viruses is the surrogate neutralization test. An enveloped virus that has one or more foreign envelope proteins is known as a pseudo typed virus. While positive sera can neutralize the NiV envelope proteins, these viruses can be handled safely in a BSL-2 facility.

To quickly identify and contain an outbreak in non-endemic areas where NiV outbreaks have not happened, a high index of suspicion is necessary. Guidelines on what constitutes a Suspected, Probable, and Confirmed case of NiV infection were released by the NCDC in India, and they have been successfully applied to contain recognized outbreaks in that country.[21]

Table 2: Laboratory and radiological diagnosis of Nipah Virus

Diagnostic Tests	Clinical Presentation
Routine Haematological Test	• Thrombocytopenia • Leucopenia • Raised liver enzymes • Hyponatremia
Cerebrospinal fluid analysis	• Lymphocytic pleocytosis • Raised proteins • Normal glucose levels
Imaging	• 2-7 mm multifocal discrete lesions in the subcortical and deep white matter.
NIV specific tests	• ELISA for detection of antibodies • Positive PCR reaction • Virus isolation

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Prevention

The primary goal of NiV management should be prevention because there is now no effective treatment option and the vaccine against NiV is still in the preclinical stage. It is essential to appropriately inform the populations that are at risk on how the virus spreads. Fruits that have come into contact with bats shouldn't be fed to farm animals. In order to lower the chance of bat-to-human transmission, raw palm sap should be avoided as it is also a potential cause of infection. Nonetheless, certain cultures find this to be a difficult step. Handling any dead or sick patient or animal requires the use of safety gear, gloves, and appropriate procedures. The National Centre for Disease Control of India has strongly suggested stringent and adequate hand hygiene by using soap and water after coming into contact with any ill person or animal due to the present epidemic in Kerala, India. Additionally, they cautioned against consuming toddy or raw palm sap. Individuals were cautioned not to handle dead bodies properly, get into abandoned wells, or eat partially chewed fruits. The Coalition for Epidemic Preparedness Innovations (CEPI) was established in January 2017 by pharmaceutical firms and foreign governments with the goal of funding and advancing research towards developing a vaccine that is safe, inexpensive, and effective against the disease. But before a vaccine is authorized for widespread use, years will pass.[22]

The main goals of prevention efforts have been to stop date palm sap from becoming contaminated, raise public knowledge of the risks associated with date palm sap consumption, and stop the spread of the disease from person to person. It has been discovered that using skirts to cover the date palm plants' sap-producing regions efficiently keeps bats away. According to a 2015 study evaluating the behaviour of those who consumed raw date palm sap, relatively few of them were aware of the NiV virus, and those who were just as inclined to ingest it as those who were not. In 2017, a randomised controlled study was carried out to evaluate a behaviour change communication intervention. The results indicated that while spreading a message prohibiting sap intake altogether did not reduce exposure to potentially contaminated sap, doing so reduced exposure to safe sap. When there is an ongoing outbreak, the WHO advises against consuming bat-bitten fruits, raw date palm sap, toddy, or juice, as well as pig and bat exposure. Gloves and other protective gear should be used when handling sick animals or their tissues, as well as during the slaughter and culling processes, to lower the danger of animal-to-human transmission.

The application of infection control measures, such as patient isolation, the use of personal protective equipment, and proper hand hygiene, is part of the prevention of person-to-person transmission. Identified contacts using contact tracing are subjected to testing and monitoring until a negative result is obtained. It has been discovered that hospital surfaces around patients are contaminated with NiV. When providing treatment for suspected or confirmed instances of NiV infection, healthcare facilities are required to implement and monitor adherence to established infection prevention and control procedures. When a suspected NiV patient is present, healthcare personnel should report the incident to the authorities and be tested for the virus. It is advised that contacts of infected individuals refrain from having extended, intimate personal contact with them. Funeral customs that necessitate coming into close touch with the dead are discouraged.[23]

Treatment

The main line of defence against catching any kind of virus is vaccination. Patients with NiV infection can only be treated with supportive and preventative care as there are currently no licensed vaccinations or drugs available to treat the infection. Essential clinical practices include maintaining hydration and electrolyte balance, airway patency, mechanical breathing, and venous thrombosis prophylaxis. Furthermore, patients infected with NiV are given broad-spectrum antibiotics. In cell cultures, chloroquine shown effectiveness in inhibiting the Nipah virus; however, animal models were unable to confirm this finding. Favipiravir administration produced positive outcomes in hamsters. A large investigation that involved preclinical testing in a variety of animals led to the identification of several vaccine kinds. A vector vaccine against the vesicular stomatitis virus has shown protective effects in hamsters, ferrets, and African green monkeys. Advanced vaccinations have been produced, such as DNA vaccines, live and recombinant virus vectors, and particles that resemble viruses. Research has been conducted on a number of viral vectors in an effort to generate experimental vaccines, including the rhabdovirus, the canarypox virus, and the vesicular stomatitis virus glycoprotein (VSV-G). It has been found that the use of a recombinant measles virus (rMV) vaccination in people shows promise. It was

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reported that recombinant VSV-vectored immunization showed good effectiveness in a hamster model.

In fruit juices or mango fruit, the NiV can survive for up to three days; in artificial date palm sap kept at 22°C, it can survive for at least seven days. In fruit-bat urine, the virus has a half-life of eighteen hours. To prevent and control human NiV infection, many measures are necessary in addition to vaccinations and medicines. There are further tips to prevent contracting the virus from the US Centres for Disease Control and Prevention (CDC). These include often washing your hands with soap, avoiding ill bats and other animals like pigs, avoiding areas where bats are known to reside, and avoiding the blood or bodily fluids of someone who has been diagnosed with NiV. Additionally, it's best to stay away from raw date juice. Furthermore, it is better to use physical barriers that prevent bats from accessing the sap stream of date palm trees. Farms should be planned to reduce overcrowding to stop the rapid spread of disease among animals, and they shouldn't be situated adjacent to fruit trees that draw bats.[6] It's crucial to stay away from contaminated food and direct touch with virus hosts and their secretions. When doing chores that need you to be close to animals, it is imperative that you wear the appropriate protective clothing, especially when disposing of and slaughtering animals. Direct human-to-human contact is a critical preventive measure that can effectively reduce the amount of NiV transmission.[24]

Antiviral

Riboflavin and Chloroquine

Ribavirin was the first antiviral medication to be used against NiV, and it was given during the 1998 outbreak in Malaysia. A research with 140 individuals infected with NiV showed that ribavirin treatment lowers the death rate from acute NiV encephalitis. Ribavirin has also been shown to be effective against HeV in vitro experiments, as evidenced by a reduction in viral output of more than 50 times. Furthermore, treatment with ribavirin delayed mortality from viral illness by two days, however it was not able to prevent death, according to a study utilizing a hamster model for NiV infection.

Moreover, research conducted in vitro have demonstrated that the antimalarial medication chloroquine has strong antiviral efficacy against NiV and HeV infections. In light of these findings, four patients were treated during the most recent HeV outbreak in Queensland with a combination of oral Chloroquine and intravenous ribavirin. Subsequent in vivo tests, however, revealed that the ribavirin-chloroquine combination did not offer any defence against the transmission of the virus. Moreover, ribavirin did not significantly affect hamsters infected with HeV, even if it postponed mortality in those with NiV infection, and hamsters receiving either ribavirin alone or in combination with chloroquine did not benefit from it.

Remdesivir

Remdesivir (GS-5734) is a prodrug that has a broad spectrum of antiviral actions against the replication of filovirus, coronavirus, and paramyxovirus. It functions as a nucleotide analogue. It can also decrease NiV replication in primary human lung microvascular endothelial cells by more than four orders of magnitude, according to in vitro investigations. Additionally, remdesivir was tested in a study to see how well it worked against NiV infection in African green monkeys (AGM). Two of the four AGM treated with remdesivir developed mild respiratory symptoms, and the other two developed severe respiratory symptoms, indicating that remdesivir is a promising antiviral medication against NiV.

Favipiravir

Toyama Chemical Company (Tokyo, Japan) produced Favipiravir (T-705; 6-flouro-3-hydroxy-2 pyrazinecarboxamine; [Avigan]), a viral RNA-dependent RNA polymerase (RdRp) inhibitor. At micromolar concentrations in vitro, favipiravir has been shown to have strong antiviral efficacy against henipaviruses by preventing Nipah and Hendra from replicating and transcribed. In a model using Syrian hamsters, animals given a fatal dosage of NiV were completely protected for 14 days by administering favipiravir subcutaneously once daily or orally twice a day.[25]

Other Antiviral

R1429, also known as 4′-azidocytidine, is a cytidine analogue and its prodrug balapiravir. It is the final nucleoside analogue investigated against NiV to date. Although prior clinical trials for the hepatitis C virus and dengue virus showed limited bioavailability and unfavourable toxic effects, Balapiravir has exhibited in vitro effectiveness against both NiV and HeV. An interferon inducer called poly(I)-poly(C12U) has also demonstrated some effectiveness against NiV in vitro and in a hamster model. It has been demonstrated that soluble ephrinB2, a functional receptor for NiV G glycoprotein, inhibits viral fusion in vitro. The G glycoprotein's ephrin-B2 and B3 receptor binding

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sites are the target of the human monoclonal antibody m102.4, which has been shown to provide post-exposure prophylactic protection against NiV challenge in ferrets and African green monkeys. Similarly, ferret NiV infections have been successfully treated by human monoclonal antibody h5B3.1, which is specific to glycoprotein F.[26]

Table 3 : Drugs with potential antiviral activity against NiV

Drug	Description	Experimental Model
Chloroquine	4-aminoquinoline	In vitro
Ribavirin	Guanosine analogue	In vitro
Acyclovir	Guanosine analogue	Historical review
Favipiravir	Purine analogue	In vitro Syrian hamster
Remdesivir (GS-5734)	Adenosine analogue	In vitro African green monkey
Balapiravir (R1479)	Cytidine Analogue	In vitro
Poly(1)-poly(C12U)	Interferon Inducer	In vitro Hamster
Ephrin B2	G glycoprotein fusion inhibitor	In vitro
Human mAb m102.4	G glycoprotein fusion inhibitor	Ferrets African green monkey
Human mAb h5B3.1	F glycoprotein fusion inhibitor	Ferrets

Table 4: Surveillance case definitions for Nipah virus infection as defined by the National centre for Disease Control, India.

Case type	Description
Suspect case	Person from a community affected by a Nipah outbreak who have: • Fever with new onset altered mental status or seizure and/or • Fever with headache and/or • Fever with cough or shortness of breath
Probable case	Suspect case patient/s who resided in the same village where confirmed case patient/s were living during outbreak period and who died before diagnostic specimens could be collected. OR Suspect case patient/s who came in direct contact with confirmed case patients in hospital setting during the outbreak period and who died before complete diagnostic specimens could be collected.
Confirmed case	Suspected case who has laboratory confirmation of Nipah virus infection either by: • Nipah virus RNA identified by PCR from respiratory secretions, urine or cerebrospinal fluid. • Isolation of Nipah virus from respiratory secretions, urine or cerebrospinal fluid.

Future direction and research gaps

NiV has been included on the WHO's Blueprint priority disease list since 2016. At least 13 vaccine candidates are in the preclinical phases of development as a result of the CEPI efforts, but none of them have received a license, and there is currently no treatment for this infection. As previously mentioned, diagnosis plays a critical role in the success of surveillance efforts and the prompt identification of positive cases, which are still necessary to stop or lessen the effects of human outbreaks. The limited number of BSL4 or BSL3 laboratories that can securely handle suspected samples continues to hinder the diagnosis of NiV, necessitating longer transit times for samples to reach these facilities. In these facilities, traditional diagnosis primarily involved costly and difficult-to-use RT-PCR assays, which needed to be run on specialized equipment by staff with specialized training. It should be inexpensive, simple to use, and consistent with biosafety standards. It should also not require highly skilled

workers, allowing for quicker diagnosis times and accessibility in rural regions of the nation. As long as the assays are appropriately interpreted, serology is still a valuable method for monitoring the spread of virus strains in bats and other species. However, there is currently a lack of standardization for these tests, thus before advocating the use of any particular tests in this situation; a precise comparison should be made. Ultimately, new research indicates that a number of bat species, including Macroglossusminimus, Rhinolophus luctus, Taphozous longimanus, Taphozousmelanopagon, and Rosettusaegypticus, may serve as NiV reservoirs. This means that the list of NiV reservoirs is never conclusive. Therefore, in the future, trustworthy serological testing will be required to assess the virus circulation in a region that may be bigger than initially reported.[28]

II. CONCLUSION

In the near future, all gaps in diagnostic techniques should be closed for improved NiV surveillance and preparedness. Multiple assays based on distinct target genes are already part of the NiV molecular diagnostics panel. Comparative studies must now be conducted in order to evaluate their sensitivity and specificity and provide recommendations for future application. There are fewer serological assays available, but they cannot currently be recommended for use in diagnostic procedures due to the lack of standardization.

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