Decrypting the Outbreak of Nipah Virus in Kerala

Raghuram.Janagam1, Nagaraja.Jarugula2, Dr.M.Prasadrao3, Akrambaig.Moghal4
1. Student department of pharmacy practice, M.A.M College of Pharmacy, Kesanupalli, Narasaraopet, Guntur, A.P, India.
2. Assistant professor, M.A.M College of Pharmacy, Kesanupalli, Narasaraopet, Guntur, A.P, India.
3. Principal, M.A.M College of Pharmacy, Kesanupalli, Narasaraopet, Guntur, A.P, India.
4. Student department of pharmacy practice, M.A.M College of Pharmacy, Kesanupalli, Narasaraopet, Guntur, A.P, India

Corresponding Author: RaghuRam

ABSTRACT: Nipah virus, microscopically called as Paramyxovirus had been retrospectively investigated as the probable etiologic agent for an outbreak of causing Acute Encephalitis in Kerala , as it recently re-emerged , but there is no evidence of suggesting that it has been transmitted by fruit bats as suspected initially . It is a highly contagious disease which exacerbates on time as it was mis-diagnosed with typical encephalitis which is caused by Japanese encephalitis virus, Herpes simplex virus, leptospire bacteria primarily targets CNS. In Kerala, at Kozhikode’s Baby Memorial hospital a patient was suspected with encephalitis and is samples were examined by the concerned doctors at Manipal center of Virus & research under trains of United States for disease control and prevention to test for nipah virus as the only pathogen capable of causing symptoms and leading to sickness in families simultaneously. Presently the majority of physicians are recommending RIBAVIRIN as the drug of choice for symptomatic relief. The Surveillance & enhancement of diagnostic measures to determine Nipah Virus infection are recommended. Later on, the rules & regulations passed on by Government of Kerala were made strict and till May 30, no individual was suspected.

Keywords: Nipah virus, Encephalitis, misdiagnoised, Ribavirin, catastrophic.

I. INTRODUCTION
Nipah virus causes encephalitis in humans and has a high fatality rate. Species of fruit bats in the Pteropus genus are the natural reservoir of Niv. Niv has been isolated and or Niv RNA has been identified or observed in bats, recently in India at Kerala. The Nipha virus had killed 16 lives, including a nurse. In late 1998 and early 1999, an outbreak of acute encephalitis with high mortality rates among pig handlers in Malaysia took place. This virus can kill the hundreds of them. To this a novel paramyxovirushas named Nipah virus. This virus was subsequently named Nipha virus after kumpungsugaiNipha (its a Nipha river village) where the 1st viral isolates were obtained. After that during Jan – Feb. of 2001 an outbreak of febrile illness with altered sensorium was obtained in siliguri, west Bengal (India). The outbreak occurred among hospitalized patients; family contacts of the patients and with the medical staff of hospitals. Japanese encephalitis which is endemic in this area was initially suspected a different disease. Now recently in India an outbreak of NIPHA re-emerged at Kerala there is no evidence to suggest that the Nipah virus in Kerala was transmitted by fruit bats as suspected initially. Nearly about 19 peoples are suffered with this disease out of them 17 are dead.

II. WHAT IS NIPHA?
It’s an emerging zoonotic (the virus that transmitted from animals to humans) pathogenic disease with disturbing abilities. And causes death to humans, But they can show the asymptomatic condition in flying fox i.e. fruit bats (pteropodidiae) and often infects animals such as pigs.

III. HOW IS IT INFECTED?
Humans are directly or indirectly in contact with infected carriers like pigs and bats, this way the disease can cause. The consumption of raw date palm sap contaminated with bat feces is the way to get the disease. Infected human to human transmission or virus transmitted to their family members or transmission to the hospital staff like doctors, nurses, ward boys, etc.
IV. EPIDEMIOLOGY

In 1999 at Malaysia an outbreak of Nipha virus this disease can file 300 human cases with over 100 death were reported. To stop the outbreak more than millions of pigs were euthanized, causing tremendous loss for Malaysia.

In Bangladesh from 2001 – 2015 nearly 298 human cases are filed out of 209 are killed 70% of mortality rate has been observed in Bangladesh.

In India, From Bangladesh this virus can cross the border twice this outbreak occurred in 2001 and 2007 in the districts of Siliguri and Nadia, in west Bengal, India killing 70 deaths were reported.

In south India at Kerala Nipha had smashed the lives of 17 humans out of 19 in 2018 and the mortality rate was 89% has been observed.

V. STRUCTURE OF THE NIPAH VIRUS

VI. ETIOLOGY

Its majorly transmitted by FRUIT BATS AND PIGS these are act as the intermediates which can transmitted this disease. The period of incubation is 14 days.

VII. PATHOPHYSIOLOGY

By preliminary autopsy findings the major target of this virus is the CNS. Endothelial syncytia, comprised of multi nucleated giant endothelial cells are frequently found in NiV infections and are mediated by the fusion and the attachment envelope glycoproteins. Ephrin B2 is a functional receptor for NiV. Ephrin B2 is expressed on endothelial cells and neurons, which is consistent with the known cellular tropism for NiV. Firstly the virus replicate at the specific site and causes vascular infection which leads to vasculitis and causes thrombosis and obstruction which leads to ischemia and infraction. Some times it spreads extra vascularly and causes parenchymal infections and cellular injury.

SYMPTOMS

Encephalitis (brain inflammation); nerve disorders; respiratory problems; fever; headache; myalgia; sore throat; vomiting; dizziness.
VIII. DIAGNOSIS
Serological test; CBC; RTPCR (Reverse Transcriptions Polymerase Chain Reaction) and Virus isolation; swab examination, antibody detection by ELISA test.

IX. TREATMENT
There is no particular treatment for nipah virus. Right now to decrease symptoms RIBAVIRIN is generally suggested by the physician. A monoclonal antibody which is targeted on the viral G glycoprotein has been beneficial in a ferret model of the disease.

9.1 Ribavirin: These are the commonly used drugs in anti viral infections like HCV; RSV infection: viral hemorrhage and right now it’s a drug for NiV infection it can’t cure the disease but it reduces the symptoms.

9.2 Mechanism of action: Ribavirin is a guanosine (ribonucleic) analog used to stop viral RNA synthesis and viral mRNA capping, thus, it is a nucleoside inhibitor. Ribavirin is a prodrug, which resembles purine RNA nucleotides after when it gets metabolized. Then, it interferes with RNA metabolism required for viral replication. Combined chloroquine and ribavirin treatment does not prevent death in a hamster model of NiV and HeVs infection. As ribavirin and chloroquine proved to be active in inhibiting henipavirus release from infected cells, we asked whether both drugs would show an increased antiviral effect when given in combination, indicative of a favorable drug–drug interaction. To investigate this, we took 8–12-week-old hamsters with a lethal dose of 104 TCID50 of NiV or HeV, and treated them with chloroquine and ribavirin post-challenge. Cell culture experiments had shown the effectiveness of both drugs in inhibiting virus spread when added up to 12 h. Based on these results and a likely best-case post-human-exposure scenario, we chose to initiate treatment at 6 h and compared the survival rate of animals receiving the following drug.

X. TREATMENT
(i) five animals received ribavirin individually at a dose of 30 mg kg−1 every 12 h.
(ii) five animals received chloroquine individually at a dose of 50 mg kg−1 every other day, and
(iii) five animals received a combination of ribavirin and chloroquine using the described concentrations and dosing schedules. Virus-infected control animals received vehicle solution, and received drugs only.

XI. SPREADING OF NIPAH IN KERALA
Mohammed Salih, aged 28-years he is an architect from Kerala’s Perambra town, came to Kozhikode’s Baby Memorial Hospital with his family. Salih was having chief complaints of vomiting, had a high fever, and he was in a mentally agitated state. A critical care physician A.S. AnoopKumar, knew that these were the symptoms meant to be encephalitis, which is an inflammation of brain tissue that kills hundreds of people in India every year. Kumar tried to stabilize the condition of Salih, but by around 9 a.m., when the hospital’s neurologists came to examine him. Though Salih was receiving top-end care, his condition was worsening timely. He had some very distinct symptoms, recalls ChellentonJayakrishnan, one of the neurologists who treated him. His heart rate is increasing over 180 beats/min and BP had also increased. His limbs were limp, seen no reflexes. These symptoms were improbably of any encephalitis cases that the team had ever seen. Jaya Krishnan and his colleagues ruled out the common causes of encephalitis, one by one. In usual the mosquito-borne infection typically doesn’t affect more than one person in a household and his younger brother Sabith expired about 12 days ago after showing similar symptoms. His father and aunt, too. Salih couldn’t have Japanese encephalitis. If so the family had been exposed through a common pet, they would have fallen sick at the same time, says Jaya Krishnan. Salih had fallen sick days after Sabith did. So, is this a case of poisoning?

The team ruled this out, too. Rabies, is another possible cause of encephalitis, was ruled out too. Toxins can trigger the encephalitis-like symptoms but were usually not accompanied by fever. Virologist Arun Kumar head of manipal centre of viruses and research and doctors at baby memorial hospital comes to conclusion that the recent attack with encephalitis in kerala is a nipah virus. The nipah virus around Kozhikode, kerala by the checking of the sample of the first patient Sahib of first day. Arunkumar was interested to make a test to findNipah for two reasons. Among them the First is that the States covered by his surveillance project were Tripura and Assam, both across the border from Bangladesh and potential geographies for Nipah and the second is that the virus is thought to be a probable bioterrorism agent. The Manipal laboratory is the only second facility in India capable of doing so, other than the Pune’s National Institute of Virology (NIV). It was a serendipitous move. On the time when Arunkumar received Sahib’s samples on May 18, he also ruled out common causes of encephalitis such as the Japanese encephalitis virus, the Herpes Simplex virus and Leptospira bacteria. Only one pathogen is capable of causing Salih’s symptoms and leading to sickness among several family members at the same time. It wasNipah, said by Arunkumar. In August 2017, the MCVR team was trained by the United States’ Centers for Disease Control and Prevention to test for the Nipah virus. That made the Manipal laboratory only the second facility in India capable of doing so, apart from Pune’s National Institute of Virology (NIV). It was an accidental discovery.
11.1 Victim: 26yr old sabith died on may 5 along with him about 17 people also lost their lives. Sabith first came as outpatient at Parambra hospital for high fever and body pains on may 2. On may 3rd he was admitted into hospital and it is suspected that four people including a night duty sister picked the virus from him. As his condition was worsened he is shifted to the medical college hospital on may 4 for CT-scan where he was died on may 5. His younger brother admitted to the hospital with same symptoms and the test revealed that he is nipah positive. At last he was also died. Then the strict rules were passed by the government of kerala and after that no nipah infected person was suspected till may 30.

11.2 Health dept’s Honour: Meanwhile, the Department of Health is honoring the team at an event to be held at Nalanda Auditorium on Sunday. Doctors who were noted for their exemplary service in 2017-18 will also be honored on the occasion by Minister for Health and Social Justice K.K. Shylaja. Excise and Labour Minister T.P. Ramakrishna will preside over the function, while Transport Minister A.K. Saseendran will be the guest of honour.

XII CONCLUSION

The nipah virus vastly spreading through the Kerala, causing encephalitis has been discovered by the MCVR by isolating the samples of that suspected individual carried by serological evidence. But, the source of virus that initiated the out-break in Kerala remained as an open question.

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