Rare but Rising: The Epidemiological Resurgence of Neglected Viral Diseases in the 21st Century

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Abstract

In the last 20 years, the global health environment is increasingly challenged by the re-emergence or resurgence of viral diseases that previously were considered rare or geographically restricted. There have been frequent, widespread, and more lethal appearances of pathogens like Crimean-Congo hemorrhagic fever virus, Nipah virus, Oropouche virus, and Powassan virus frequently across the globe and in areas once thought to be non-endemic. The appearance of these re-emerging diseases is not random, rather the result of overlapping ecological, socio-political, and epidemiological forces, including climate change, deforestation, urbanization, diminished surveillance capacity, and increased interaction between humans and animals. Many of these viruses have no approved vaccines or targeted therapeutic regimens and obviously behold serious threats to global health security. This article first describes the underlying relationships to emerging patterns, discusses the public health implications, and identifies the need for integrative, predictive, and proactive approaches to surveillance, vaccine development, and cross-sectoral collaboration. As the lines between endemic and epidemic taints are now becoming blurred, it is clear that "neglected" viral diseases are not fixed in time - requiring a recalibration of global priorities for preparedness.

Keywords: Neglected viral diseases, emerging infections, zoonotic spillover, vector-borne viruses, Crimean–Congo Hemorrhagic Fever (CCHF), Nipah virus, Oropouche virus, epidemiological resurgence, climate change and disease emergence, One Health, viral

hemorrhagic fevers, global health security, disease surveillance, arboviruses, infectious disease epidemiology.

INTRODUCTION

This era of 21st century has witnessed a paradox of various global health problems, where many infectious as well as deadly diseases like HIV, Malaria, Dengue, Tuberculosis and various other diseases have been the main focal point of the different health authorities. Even after the tremendous efforts of these regulatory authorities a silent resurgence of different rare and neglected viral diseases has been found to increase at huge rate. These diseases were previously ignored but with the span of time numbers of patients who have been diagnosed from this disease are found increasing at a larger rate.

These diseases like Oropouche fever in south America and Chandipura virus were previously confined to specific locations. However, shifts in climate, urbanization, global travel, and ecological disruption have expanded their reach and altered their epidemiological dynamics. Many of these viruses exhibit complex transmission cycles involving arthropod vectors or zoonotic reservoirs, complicating detection, surveillance, and control. This review aims to spotlight these underrecognized viral threats, examining the multifactorial causes of their emergence, their clinical and public health implications, and the urgent need for integrated global strategies to address them before they evolve into the next major outbreak.

These diseases are known as **"neglected tropical diseases,"** which, according to the World Health Organization, are a varied category of illnesses caused by a variety of pathogens (including viruses, bacteria, parasites, fungi, and toxins) and are associated with devastating health, social, and economic consequences. NTDs are primarily found in poor tropical societies, while some have a considerably wider geographical range. NTDs are predicted to impact over 1 billion individuals, with 1.495 billion requiring NTD therapies (including preventative and curative).

The prevalence of NTDs is complicated and frequently linked to environmental factors. Many of them are vector-borne, rely on animal reservoirs, and have complicated life cycles. All of these issues complicate their public health control efforts.

This article reviews about the phenomenon of disease emergence and reemurgence. The World Health Organization (WHO) predicts that more than 1.495 billion individuals globally should be targeted for illness prevention and treatment each year. Noncommunicable diseases (NTDs) cause considerable mortality and morbidity, costing developing countries billions of dollars each year. They also cause disability, stigma, social isolation, and prejudice, as well as financial pressure for sufferers and their families. Despite their relevance, NTDs have received little attention in global health policy, with objective 3.3 of the Sustainable Development Goals being added in 2015.

So, this article mainly focuses on the topic of explaining about the various rare viral diseases that have been seen increasing in the recent years. Here are few examples of various viral diseases like-

1) Oropouche Virus
2) Nipah Virus
3) Chandipura Virus
4) Toscana Virus

Here is a detailed explanation about these viruses, their Epidemiology & geography, Transmission dynamics, clinical features, Mortality, morbidity and different emerging trends for treating and diagnosing these diseases.

1) **Oropouche Virus:** The Oropouche virus (OROV) is a member of the genus Orthobunya- virus, family Peribunyaviridae, and order Bunyavirales. It belongs to the Orthobunyavirusoropoucheense species. The single-stranded RNA virus OROV has a spherical lipid-enveloped virion and is negative-sense. The three single-stranded RNA segments (L, M, and S) that make up the viral genome are contained within a helical nucleocapsid. These segments encode the nucleocapsid protein (N), viral envelope glycoproteins Gn and Gc, and RNA-dependent RNA polymerase (RdRp), all of which are necessary for viral replication and assembly.

OROV is further categorized under the Simbu serogroup of the genus Orthobunyavirus, which contains 129 known viruses (along with nine other related but unclassified species). Two main evolutionary subclades comprise this serogroup:

Subclade A: Manzanilla and Oropouche
Subclade B: Simbu, Akabane, Sathuperi, Shamonda, and Shuni viruses. [1]

History and Spread of the Disease:

OROV is a febrile arboviral infection that is most typically found in Brazil and the Amazon. The first case was recorded in Trinidad and Tobago in 1955, and a second strain was discovered in Trinidad in 1960. The virus was initially discovered in Brazil in 1960 from sloth blood and Ochlerotatus serratus mosquitos.

In 1960, a significant outbreak of Oropouche fever was recorded in Belém City, affecting an estimated 11,000 persons. Since then, the virus has spread to cities in other South American states and other nations. In 2009, the final epidemic of OROV was recorded in Altamira and Santa Barbara in Pará State, as well as Mazagão in Amapá.[2]

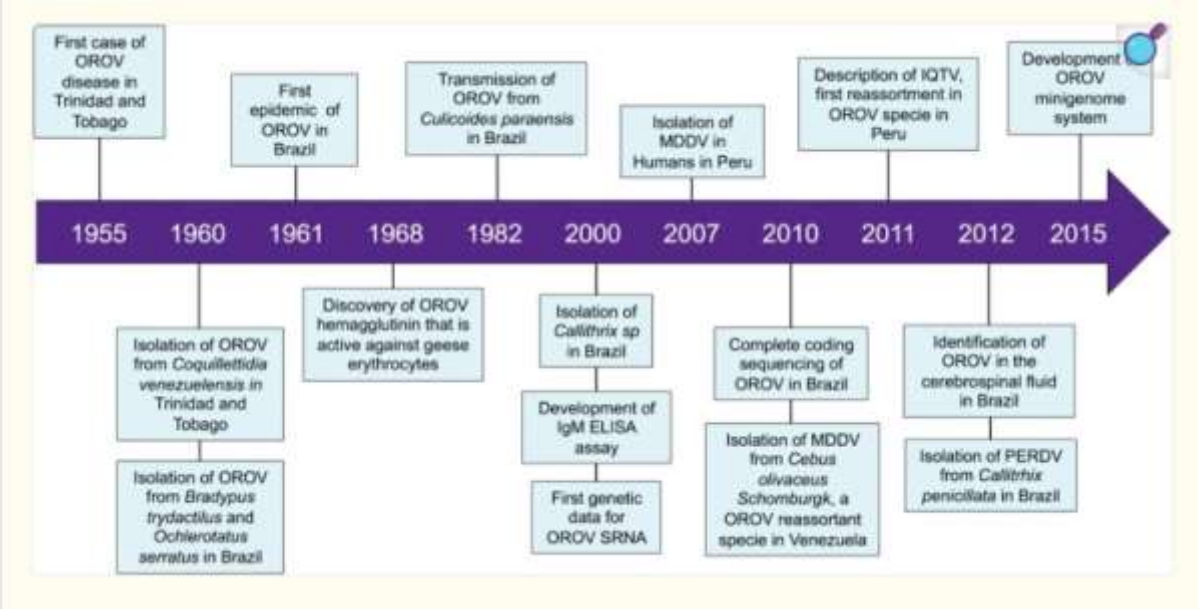

Figure 1: Time line of Advances in Oropouche Virus [3].

Pathogenesis: The highly detectable virus known as OROV enters the bloodstream and brain pathways, where it causes an initial inflammatory response in the central nervous system, which in turn leads to a systemic infection. Most research focuses on figuring out how OROV affects the central nervous system. When given intra-cerebrally, it has been demonstrated to infect experimental animals, especially mice and hamsters. Hepatocyte death and Kupffer cell hyperplasia are signs of fatal unembellished hepatitis, according to an early study on intra-cerebral inoculation of hamsters.

Recent studies have employed the subcutaneous technique to introduce OROV into experimental animal models, simulating the arbovirus's natural mode of infection. The OROV vaccine caused a systemic infection in hamsters, which resulted in neurological motor dysfunction and paralysis. Hematogenous transmission to the liver and brain was shown by the virus's accumulation in these organs. A Trojan horse mechanism most likely breaks through the blood-brain barrier (BBB), enabling the virus to multiply in its target organs and tissues. Because of its three RNA genome segments, OROV exhibits a great deal of genetic variety, allowing for both re-assortment processes and novel evolutionary features. The first recorded deaths linked to the virus occurred in 2024, when a genetically distinct re-assortant strain was identified as the cause of the OROV outbreak in Bahia, Brazil. [3]

Clinical Manifestations: The Oropouche virus (OROV) is the cause of Oropouche fever, an acute febrile illness that manifests as a high temperature, headache, myalgia, arthralgia, malaise, and skin rash. Relapses are frequent, and viremia typically manifests during the first few days of infection. When an OROV infection is severe, it can lead to neurological problems including meningitis or encephalitis. According to experimental research, OROV has been detected in the brain and spinal cord of adult golden hamsters and newborn BALB/c mice, indicating that the virus may first infiltrate the central nervous system before spreading to the brain. [4]

2) **Nipah Virus:** Nipah virus (NiV) is an RNA virus from the Paramyxoviridae family, specifically the genus Henipavirus, which also includes Hendra virus (HeV) and the recently identified Cedar virus. It is extremely pathogenic to a wide spectrum of mammals and has the potential to cause pandemics due to zoonotic and person-to-person transmission. Pteropus bats, the reservoir of infection, have a global distribution, and recent outbreaks in Kerala, India, underscore the need for urgent research on epidemiology, transmission routes, and possible preventative and control techniques. NiV is designated as a biological safety level 4 (BSL4) pathogen, and access to laboratories is restricted in several countries. To achieve successful control, a one Health strategy must take into account humans, domestic and peri-domestic animals, and the environment. [4]

History and Spread of the Disease: Between September 1998 and April 1999, a large outbreak of illness in pigs and humans in Peninsular Malaysia claimed the lives of 105 people and forced the slaughter of almost 1.1 million pigs. The disease in pigs was highly contagious, with severe fever, respiratory involvement, and occasionally nervous symptoms in all age groups. The most common clinical syndrome in humans was encephalitic, with symptoms including fever, headache, myalgia, sleepiness, and disorientation, which could develop to coma within 48 hours.

Most human patients had close contact with live pigs, and the majority were pig farmers. The principal illness cause in pigs and humans was eventually discovered to be a previously unknown virus of the Paramyxoviridae family. The predominant method of transmission on pig farms was thought to be respiratory. Retrospective investigations indicate that NiV has caused disease in pigs in Peninsular Malaysia since late 1996, but it was not recognized as a new syndrome due to comparable clinical symptoms, morbidity, and mortality rates. [5]

The study of NiV in humans and pigs was impacted by the success of targeted wildlife surveillance, the scarcity of resources, and the parallels between NiV and HeV. Because of their close association, Malaysian bat species were given priority. For serology and virus isolation, wild-caught bats were the focus because Malaysia lacked wildlife rescue networks. Twenty-one bats of five species have neutralizing antibodies to NiV. NiV was successfully isolated from a seropositive flying fox colony in a recent attempt.

The virus's effective transmission and maintenance in pig herds suggested a wide range of possible secondary hosts. Pig farms were frequently surrounded by peridomestic birds and small mammals. 72 of the 465 dog samples that were analyzed exhibited antibodies, while 92 of the dogs that were sampled from disease-endemic areas close to Bukit Pelandok had an antibody frequency of 46%. Following the removal of pigs, peridomestic rodents, insectivores, and birds all showed negative serologic data, suggesting that they were not secondary reservoirs for NiV. NiV did not spread laterally among dog populations, as seen by the reduced antibody frequency and the confinement of infection to within 5 km of the endemic area, while dogs were easily infected after close contact with sick pigs. [5]

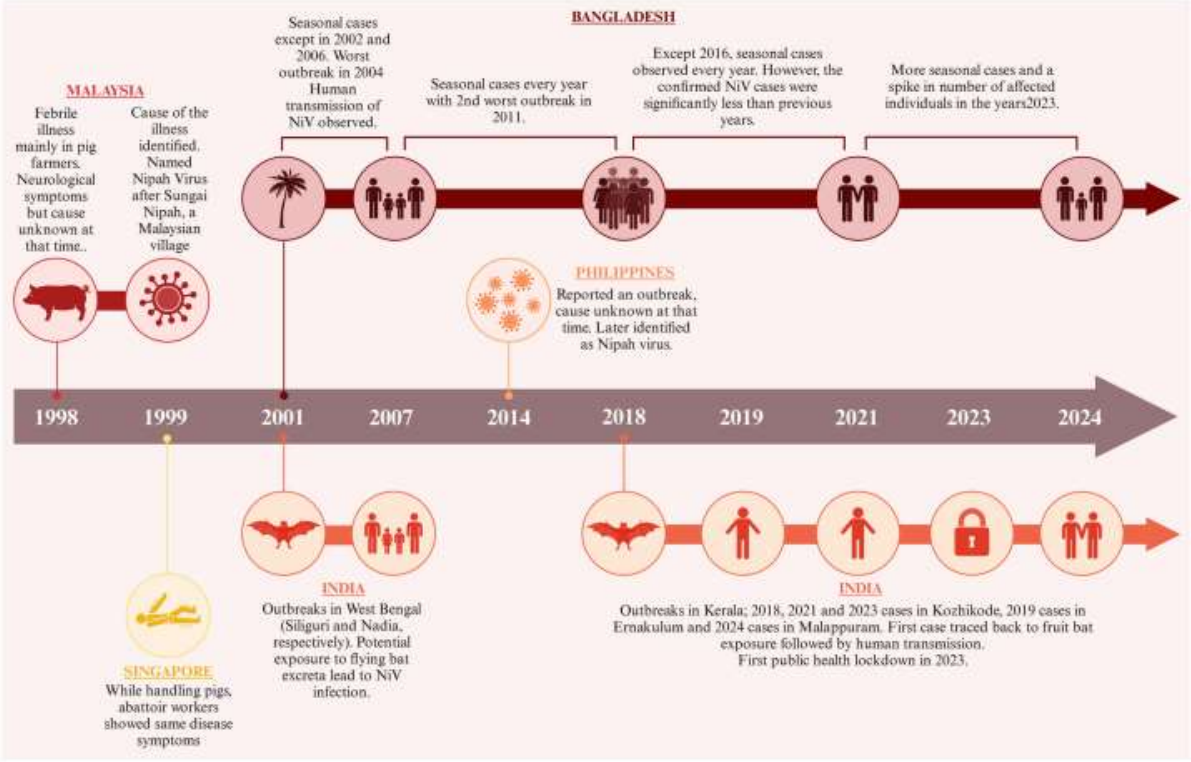

Figure 2: Timeline of Nipah virus (NiV) outbreaks from 1998 to 2024.

Pathogenesis: Nipah virus (NiV) is a fast spreading and frequently lethal virus that attacks the central nervous system and respiratory tract. It replicates in respiratory mucosa epithelial cells or immune cells such as macrophages, binds to ephrin-B2 and ephrin-B3 receptors, and infects a wide spectrum of cells before spreading throughout the body. The virus enters the bloodstream and causes viremia, which spreads to many organs. Its affinity for endothelial cells results in necrotizing vasculitis, vascular leakage, and tissue destruction.

The brain is especially vulnerable because NiV passes the blood-brain barrier. In the central nervous system, NiV causes diffuse encephalitis, neuronal necrosis, perivascular inflammation, and, in certain cases, viral inclusion bodies. In the lungs, NiV infects alveolar epithelial cells, causing severe pneumonia, alveolar bleeding, and acute respiratory distress syndrome (ARDS). The virus's non-structural proteins disrupt type I interferon signaling, allowing it to elude detection and proliferate unchecked. The condition usually appears after 5-14 days, with nonspecific symptoms such as fever, headache, and muscle soreness. The case fatality rate is disturbingly high, often reaching from 40% to 70%, and survivors may experience long-term neurological consequences. [6]

Clinical Manifestations: Nipah virus infection presents with a wide spectrum of clinical manifestations, ranging from mild flu-like symptoms to severe, life-threatening encephalitis and respiratory failure. After an incubation period of 5 to 14 days, patients typically develop fever, headache, myalgia, sore throat, and fatigue. As the disease progresses, many develop acute neurological symptoms such as disorientation, seizures, and coma due to encephalitis,

often within 48 hours. Respiratory symptoms like cough, shortness of breath, and acute respiratory distress syndrome (ARDS) are more prominent in outbreaks caused by the Bangladesh strain. In severe cases, multiorgan involvement, hypotension, and death can occur, with case fatality rates ranging from 40% to 75%. Some survivors experience long-term neurological complications or relapsing encephalitis. [7]

3) **Chandipura Virus:** The Chandipura virus (CHPV) was first discovered in 1966 by Dr. Bhatt and Dr. Rodrigues at the Virus Research Centre in Pune, India. The virus was discovered during an investigation into a febrile sickness in Chandipura village, near Nagpur. The virus was discovered unusually, in the blood samples of those afflicted. The Virus Research Centre's database of arboviruses grew, and in 1967, control of the collection was handed over to the Indian Council of Medical Research, the National Institute of Virology. The virus was initially thought to be spread throughout India based on the identification of virus-specific antibodies in people and animals. However, it was not initially linked to serious illnesses in humans. In 1983, the virus was clinically linked to an 11-year-old boy who died from acute encephalopathy syndrome. Although it took twenty years for the virus to become widely recognized, it signalled the start of increasing knowledge of its tendency to cause serious neurological sickness.[8]

CHPV, a disease endemic to India, is a common cause of acute encephalitis in pediatric populations. It was first isolated in 1965 in Maharashtra state, but outbreaks likely occurred a decade prior in 1957. The disease primarily affects children under 15 and has a rapid progression with symptoms of high fever and vomiting. No human-to-human transmission of CHPV has been identified, but it is transmitted by the bite of vectors such as sandflies, mosquitoes, and ticks.

The virus is present in African countries, Nigeria, Senegal, Bhutan, Nepal, and Sri Lanka, raising concerns about its potential spread to other regions. Outbreaks have occurred sporadically across different Indian states and regions since 2003, but they have largely been small and well contained. The most recent outbreak of acute encephalitis (AES) in 20 years was the largest in 20 years, with 245 cases reported between June and August 2024, resulting in 82 deaths, 64 of which were confirmed to be due to CHPV. Experts warn of a potential resurgence due to the ongoing monsoon season, urbanization, population movement, and ecological changes, which influence vector populations and increase human exposure to the virus.

History and Spread of the Disease: CHPV, a virus found in India, Bhutan, Nepal, Sri Lanka, and Africa, has been detected in various parts of the country. It was first identified in 1965, but it was first identified in India in 1957-58. CHPV was first isolated from sera collected from clinically confirmed encephalitis cases in 1983 and 1993. In 2003, it re-emerged in Andhra Pradesh, Gujarat, Maharashtra, and Gujarat, with a high fatality ratio. In 2005, it was found in Gujarat's Vadodara district with a 70% case fatality rate. CHPV has

been associated with several encephalitis epidemics in India, Nigeria, and Sri Lanka. It has also been isolated from hedgehogs and macaques in Nigeria and Sri Lanka. [9]

Table 1: History and Spread of the Chandipura Virus

Year(s)	Location/Region	Event/Findings
1965	Nagpur, Maharashtra, India	First detection and isolation of CHPV from two febrile cases
1997 & 2002	Warangal district, Andhra Pradesh	Investigations suggested wide circulation of CHPV
2003	11 districts, Andhra Pradesh	Encephalitis outbreak with Case Fatality Rate (CFR) of 56%
2003	Andhra Pradesh	CHPV isolates obtained
2004	Vadodara district, Gujarat	CHPV outbreak with 70% CFR in pediatric population
2004	Gujarat State	CHPV outbreak with 78.3% CFR among children; virus isolated
2004–2005	Not specified	Anti-CHPV neutralizing antibodies detected in 65.3% sera; CHPV isolated
2005–2006	Andhra Pradesh	Hospital-based surveillance revealed 54.4% CFR
2007–2008	Nagpur, Maharashtra	CHPV investigation showed CFR 43.6%; viral RNA detected in sandflies
2008	Maharashtra	CHPV RNA detected in two sandfly pools
2009	Andhra Pradesh & Hyderabad region	CHPV infection confirmed in 8/10 encephalitis cases by RT-PCR; only 1/132 contact sera positive for anti-CHPV IgM
2010–2011	Panchmahal district, Gujarat	Anti-CHPV IgM antibodies detected in 10.56% (n=587) fever cases
2012	Maharashtra and Gujarat	Anti-CHPV IgM ELISA and RT-PCR confirmed 4.7% CHPV positivity in encephalitis cases (n=130)
Till date	India, Bhutan, Sri Lanka, Nepal; Nigeria, Senegal	CHPV presence recorded in Indian subcontinent and Africa (notably in hedgehogs)

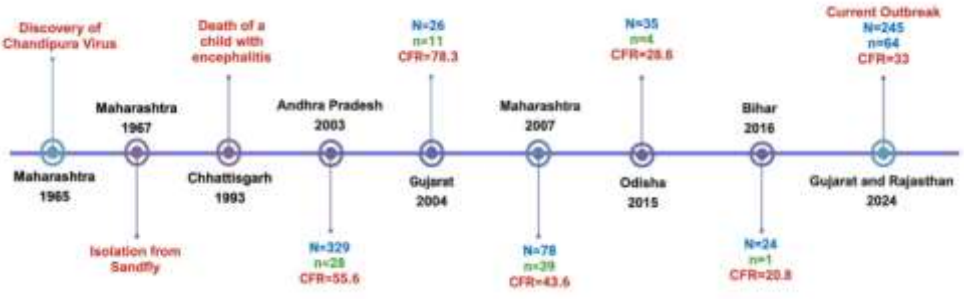

Figure 3: Timeline for the Resurgence of Chandipura Virus[9]

Pathogenesis: Chandipura virus (CHPV), a sandfly-borne virus, is a highly neurotropical virus that enters the body through a bite. It replicates in local immune cells, leading to viremia. The virus then crosses the blood-brain barrier and enters the central nervous system, targeting critical regions like the brainstem, thalamus, and basal ganglia. This leads to neuronal necrosis, apoptosis, inflammation, and microglial activation. The host's immune response, including overproduction of cytokines, exacerbates the damage. The virus causes acute encephalitis, seizures, coma, and death, particularly in children under 15. [10]

Clinical Manifestations: Chandipura virus infection starts with fever, vomiting, and diarrhea, often mistaken for other febrile illnesses. As the disease progresses, neurological symptoms become prominent, indicating early central nervous system involvement. In pediatric cases, the infection can lead to serious complications like encephalitis and coma within 24 to 48 hours. Early diagnosis and intervention are crucial in managing CHPV infections. [11]

4) Crimean–Congo Hemorrhagic Fever (CCHF)

CCHFV belongs to the family of tickborne viruses known as the Bunyaviridae, specifically the Nairovirus genus. of the five genera that make up the Bunyaviridae family. Hemorrhagic fever-causing viruses belong to three genera: Phlebovirus, Nairovirus, and Hantavirus. There are seven serogroups of nairoviruses. The prototype of the CCHF serogroup, which also contains the Hazara virus, is CCHFV; it has not been shown that the Hazara virus can infect humans.

A tick-borne CCHF virus (CCHFV) is the cause of the vector-borne zoonotic disease known as Crimean Congo hemorrhagic fever (CCHF). The virus, which infects a variety of animals and several bird species, is common throughout Africa, the Middle East, Eastern Europe, and parts of Asia. To remain fit in a variety of settings, hosts, and vectors, the virus have developed into several lineages. In humans, its case fatality rate varies from 10% to 50%. A number of ixodid ticks are thought to be effective and natural vectors of CCHF, which has a complicated and poorly understood transmission cycle.

The virus infects hosts at every stage of its growth and can spread transovarially, transstadially, and most likely during co-feeding. Humans can contract the disease by coming into touch with infected animal tissues during the viraemic phase, by being bitten by an infected tick, or by coming into contact with human blood and infectious tissues during the acute phase. There is a high risk of CCHF in many African nations; in 20 of these, outbreaks have been documented. Tick collection or crushing, elderly age, and livestock rearing are risk factors for human illnesses. Studies on CCHF frequently ignore environmental risk factors and regional distribution in favor of concentrating on infection patterns in humans and livestock. [12]

History and Spread

Crimean-Congo Hemorrhagic Fever (CCHF) was first identified in 1944 and has since spread across Eastern Europe, the Middle East, Sub-Saharan Africa, and South Asia, primarily

transmitted by Hyalomma ticks, outbreaks have occurred in countries like Turkey, Iran, Pakistan, India, Spain, and Portugal. Climate change, lack of a vaccine, and high case fatality have led the World Health Organization to categorize CCHF as a priority emerging infectious disease.[13]

Pathogenesis

The main ways that the Crimean-Congo Hemorrhagic Fever Virus (CCHFV) spreads are by direct contact with the blood or tissues of infected humans or animals, or by the bite of an infected Hyalomma tick. Once within the host, the virus first targets endothelium cells, dendritic cells, and macrophages. This causes the virus to replicate early in the local lymph nodes before spreading through the bloodstream. It infiltrates vital organs such the spleen, liver, and vascular endothelium, where it causes immunological activation and direct cytotoxic effects.

The ability of CCHFV to inhibit interferon responses, especially Type I interferons, is a crucial pathogenic characteristic that enables it to avoid innate immune recognition and multiply quickly. Pro-inflammatory cytokines (such as IL-6, IL-10, and TNF-α) are released by infected endothelium and immune cells, starting a cytokine storm those results in capillary leakage, systemic inflammation, and widespread endothelial dysfunction. These side effects encourage thrombocytopenia and disseminated intravascular coagulation (DIC), which can lead to vascular collapse, multi-organ bleeding, and in extreme situations, shock and death. [14]

Clinical Manifestations

After an incubation period of one to three days for tick-borne cases and up to nine to thirteen days for blood-contact exposures, the clinical phase of CCHF usually starts. The four traditional stages of the sickness are incubation, pre-hemorrhagic, hemorrhagic, and convalescent. Abruptly high temperature, intense headache, myalgia, nausea, photophobia, and mood swings are the hallmarks of the pre-hemorrhagic phase. During the first three to five days, patients may have abdominal pain, conjunctival injections, and flushed faces. Signs of bleeding emerge as the illness enters the hemorrhagic phase, which typically occurs between the fourth and fifth day.

These include gingival bleeding, ecchymoses, petechiae, hematuria, hematemesis, epistaxis, and even cerebral hemorrhage. Particularly in severe cases, this stage is linked to shock, hepatic failure, and fast thrombocytopenia. There have also been reports of neurological side effects such unconsciousness, agitation, and confusion. In fatal situations, multi-organ failure usually results in death within the second week. Survivors recover gradually over a few weeks as they approach the convalescent period. [15]

CONCLUSION

The 21st century has witnessed a troubling epidemiological shift: viruses once considered rare, geographically isolated, or biologically constrained are now reemerging with greater intensity, geographic range, and public health impact. Diseases such as Crimean–Congo Hemorrhagic Fever, Oropouche virus infection, Nipah virus encephalitis, and Powassan virus disease, once neglected due to their low global incidence, are increasingly making headlines due to their deadly nature, lack of treatment options, and capacity for nosocomial or zoonotic spread. This resurgence reflects a complex interplay of climate change, deforestation, globalization, urban expansion, and breakdowns in public health infrastructure—factors that have created fertile ground for viral spillover and amplification. The reappearance of these pathogens underscores the fragility of existing disease surveillance and the urgent need to reframe how we categorize “rare” diseases in a rapidly changing world.

FUTURE ASPECTS

Looking forward, addressing the resurgence of neglected viral diseases demands a multi-pronged and anticipatory strategy. First, enhanced global surveillance networks and real-time genomic tracking will be essential for early detection and containment. Second, investment in pan-viral vaccine platforms, especially those adaptable to hemorrhagic fevers and vector-borne threats, must be prioritized. Third one is the integrated One Health approaches that unify human, animal, and environmental healths are critical for predicting and mitigating zoonotic spillovers. Additionally, climate-responsive vector control programs and risk mapping using AI and satellite data could help anticipate future hotspots. Finally, the global health community must reallocate resources toward diseases traditionally labeled as "neglected," recognizing that in an interconnected world, geographic distance offers no true buffer against viral emergence. Only by shifting from reactive to predictive public health can we hope to prevent the next viral crisis from becoming another tragic inevitability.

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