

A Review On Ebola Virus Disease

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Abstract- *Ebola Virus Disease (EVD) is a deadly hemorrhagic fever caused by the Ebola virus, a member of the Filoviridae family. Due to increasing global migration, the disease has become a significant international public health concern. In the early stages, patients typically exhibit general flu-like symptoms, which may progressively worsen and lead to shock and failure of multiple organs. At present, no definitive or targeted treatment is available for EVD; therefore, management mainly relies on supportive care and symptomatic relief. This review article offers an in-depth discussion of the Ebola virus, including its clinical features, oral manifestations, diagnostic methods, differential diagnoses, prevention strategies, and treatment approaches.*

Keywords- Ebola virus Disease, Taxonomy, Epidemiology of EVD Pathophysiology, Pathogenesis of Ebola virus, Diagnosis, Treatment

I. INTRODUCTION

Ebola, previously known as Ebola Hemorrhagic Fever (EHF), is an extremely deadly disease that primarily affects humans as well as nonhuman primates. Ebola Virus Disease (EVD) is caused by a virus belonging to the Filoviridae family and the genus *Ebolavirus*. [1] Since its discovery, EVD has created significant diagnostic difficulties and has remained a global public health concern. The disease was first identified in 1976 in Zaire, Africa (now the Democratic Republic of Congo), by Dr. Peter Piot while he was investigating a suspected yellow fever case. [2] The term “Ebola” originated from the nearby Ebola River in Congo, where the outbreak was initially observed. [3]

Fruit bats of the Pteropodidae family—particularly *Hypsignathus monstrosus*, *Epomops franqueti*, and *Myonycteris torquata*—act as natural reservoirs of the Ebola virus in Africa. Nonhuman primates may become infected by consuming partially eaten fruits contaminated by these bats and can subsequently transmit the virus to humans. [4] India faces a potential risk of EVD, as the Pteropodidae fruit bat family is also found within the country’s geographical region. [5]. Certain traditional burial rituals have been strongly linked to disease spread. [6] Sexual transmission has been documented, although airborne spread is considered

unlikely. [7] EVD often presents with unusual and nonspecific symptoms, especially during its early phase, making diagnosis challenging. Initial signs include fever, muscle pain, headache, vomiting, and diarrhea. [8] Bleeding from body openings is a characteristic feature of the disease. [9] Oral manifestations may include gum bleeding, painful swallowing (odynophagia), and other atypical oral lesions. [10]

On August 8, 2014, the World Health Organization (WHO) declared the EVD outbreak a “Public Health Emergency of International Concern.” [5] With a large immigrant population, India is assessing the possibility of a future EVD outbreak. The Ministry of Health and Family Welfare, in cooperation with various agencies, has reviewed the situation and issued travel and health guidelines for air, land, and sea routes to protect health care workers and the general population. [11]

History of Ebola outbreaks

The earliest documented outbreak of the Ebola virus occurred in 1976, involving two simultaneous incidents of different Ebola strains in Yambuku, northern Zaire (currently the Democratic Republic of the Congo, DRC), and in southern Sudan. The strain identified near the Ebola River was later named Zaire ebolavirus. [12] In 1994, an ethnologist became infected after handling a deceased chimpanzee in the Taï National Park in Ivory Coast. [13] This virus was found to be different from the strains responsible for earlier outbreaks in the DRC and Sudan and was therefore classified as Taï Forest ebolavirus. [14] Another outbreak of an unknown illness occurred in 1990 in Reston, Virginia, USA, among cynomolgus (crab-eating) macaque monkeys imported from the Philippines. [15] A new strain, Bundibugyo ebolavirus, was identified in 2007 following an outbreak in the Bundibugyo district of western Uganda, marking the discovery of the fifth known Ebola virus strain. [16]

Between 2013 and 2015, West Africa experienced the most severe Ebola outbreak in history. [17,18] In 2018, a new strain known as Bombali ebolavirus (BOMV) was detected in bats in Sierra Leone; however, its ability to cause illness in animals or humans is still uncertain. A chronological

summary of major Ebola virus disease outbreaks is presented in Table 1.[19]

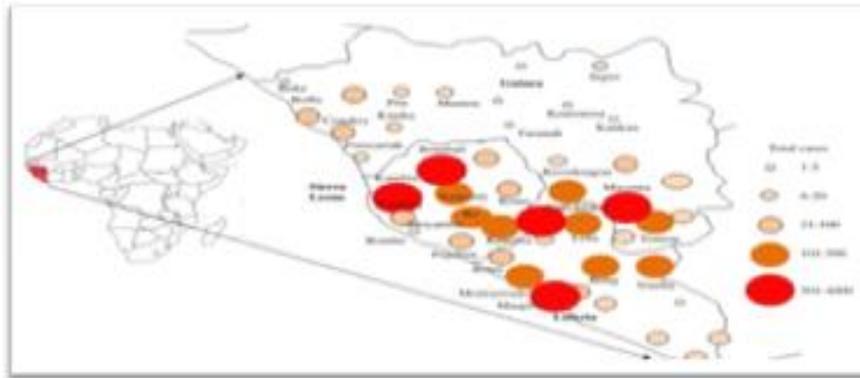


Fig 1 Detailed geometric data of the EVD outbreak.

The data shown are based on a report by the WHO:

Year	Country/village	Ebola virus subtype	Number of human cases	Number of deaths	Mortality	Source and spread infection
2000-2001	Uganda	Sudan virus	425	223	53%	Providing medical care to Ebola case-patient without using adequate personal protection measures
2001-2002	Gabon	Ebola virus	65	53	82%	Outbreak occurred over border of Gabon and Republic of Congo
2001-2002	Republic of the Congo	Ebola virus	57	43	75%	Outbreak occurred over border of Gabon and Republic of Congo
2002-2003	Republic of the Congo	Ebola virus	143	128	89%	Outbreaks in the district of Mboma and Kelle in Cuvette Quest Department
2003	Republic of the Congo	Ebola virus	35	29	83%	Outbreaks in the villages of Mboma district, Cuvette Quest Department
2004	Sudan, Yambia	Sudan virus	17	7	41%	Outbreak concurrent with an outbreak of measles, and several cases were later reclassified as measles
2004	Russia	Ebola virus	1	1	100%	Laboratory infection
2007	Democratic Republic of the Congo	Ebola virus	264	187	71%	The outbreak was declared on November 20. Last death on October 10
2007-	Uganda	Bundibugyo	149	37	25%	First reported

2008		virus				occurrence of a new strain
2008	Philippines	Reston virus	6	0		Six pig farm workers developed antibodies; did not become ill
2008-2009	Democratic Republic of the Congo	Ebola virus	32	15	47%	Not well identified
2011	Uganda	Sudan virus	1	1	100%	The Uganda Ministry of Health informed the public that a patient with suspected Ebola died on May 6 th 2011
2012	Uganda, Kibaale	Sudan virus	11	4	36%	Laboratory tests of blood samples were conducted by UVRI and CDC
2012	Democratic Republic of the Congo	Bundibugyo virus	36	13	36%	This outbreak has no link to the contemporaneous Ebola outbreak in kibaale, Uganda
2012-2013	Uganda	Sudan virus	6	3	50%	CDC assisted the ministry of Health in the epidemiology and diagnosis of the outbreak
2014	Democratic Republic of the Congo	Zaire virus	66	49	74%	The outbreak was unrelated to the outbreak of West Africa

Table no. 1. Outbreak of EVD

Source of Infection

Close physical contact with patients during the acute phase of the illness, as well as exposure to blood or bodily fluids from deceased individuals, represents the primary route of Ebola virus transmission.[20]. In fact, unsafe burial rituals were responsible for nearly 68% of infections during the 2014 Ebola outbreak in Guinea.[21]EBOV RNA can persist in rectal, conjunctival, and vaginal secretions for up to one month, and the virus may remain detectable in semen for as long as three months, indicating that recovering patients may still harbor the virus.[22]Another study reported a male convalescent patient whose semen tested positive for Ebola antigens almost three months after the onset of symptoms.[23] Although asymptomatic carriers were long believed to be non-infectious and not significant contributors to Ebola outbreaks—especially based on field observations in West Africa[24]—this belief was challenged when an asymptomatic infection was documented during the 1996 North Gabon

epidemic.[25] This underscores that exposure to infected blood and bodily fluids is the major mode of transmission.[26]Consumption of raw or undercooked meat

from infected animals, such as bats and chimpanzees, plays a significant role in oral transmission of Ebola, particularly in African regions.[27] The detection of Ebola virus in pigs in the Philippines in 2008 further expanded concerns regarding potential animal reservoirs.[28]

Hospital-acquired transmission has also contributed to Ebola spread, especially in areas with poor infection control practices. Reuse of contaminated needles was linked to the outbreaks in Sudan and Zaire in 1976.[29,30] Similarly, inadequate sterilization and poor hygiene were key factors behind the 1967 Yambuku outbreak.[31]

Taxonomy

Among these, EBOV is responsible for the most severe form of Ebola hemorrhagic fever, producing the highest mortality rates (57%–90%). SUDV follows with fatality rates between 41% and 65%, while BDBV shows around 40% mortality.[34]

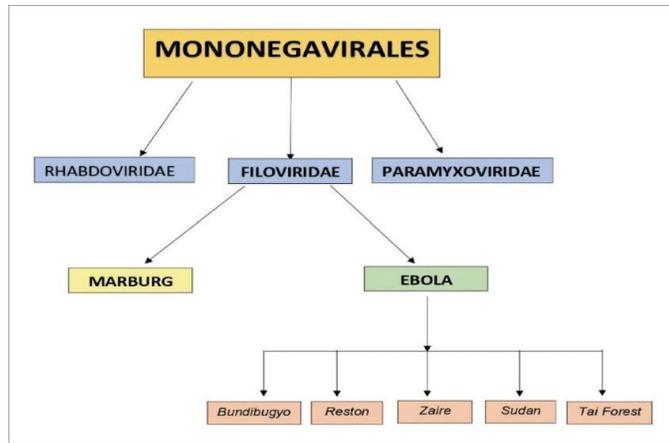


Fig 2 show the taxonomy of ebolaviruse

Epidemiology of EVD

1. Host and reservoir range

The exact range of hosts and reservoirs for the Ebola virus is still not fully understood. Evidence suggests that its primary hosts include primates such as humans, chimpanzees, gorillas, and monkeys.[35,36] Fruit bats belonging to the Pteropodidae family are believed to serve as the natural reservoirs of the virus. Other animals—such as duikers, non-human primates, cats, foxes, hogs, antelopes, porcupines, and rodents—are considered potential intermediate or incidental hosts. Unlike bats, however, these animals generally develop severe or fatal disease when infected. They may harbor the virus without symptoms and shed it through bodily fluids, enabling transmission to humans who hunt, handle, or consume bushmeat from these animals.[35,37,38] Research has also shown that the virus can persist in various body fluids and tissues—including semen, vaginal secretions, sweat, aqueous humor, urine, and breast milk—of individuals who have recovered from Ebola virus disease.[39,40] This persistence may contribute to relapse or reactivation of infection in previously recovered patients.[36]

2. Transmission among humans

Ebola virus is transmitted to humans primarily through direct contact with the blood, organs, or other bodily fluids of infected animals. The index case of the 2014–2016 West Africa outbreak was linked to exposure to bats.[41] Beyond bats, infections have also occurred in individuals who

handled infected chimpanzees, gorillas, and forest antelopes—whether alive or dead—in regions such as Gabon, the Republic of the Congo, and Côte d’Ivoire.[42]

The virus can enter the human body through the nose, mouth, eyes, ears, cuts, open wounds, or mucous membranes.[43] Transmission through sexual contact with an Ebola survivor or convalescent patient has also been reported.[44] In individuals with Ebola virus disease (EVD), the virus is present in all bodily fluids, including blood, vomit, urine, stool, sweat, tears, breast milk, semen, mucus, saliva, and other secretions.[45]

3. Pathogenesis

The Ebola virus specifically targets cells of the mononuclear phagocyte system. After entering the body through the skin or mucous membranes, the virus infects monocytes, macrophages, and dendritic cells—key immune cells responsible for its widespread dissemination.[46] The viral surface spike glycoprotein enables attachment and entry into these macrophages and dendritic cells, a process regarded as a crucial step in the pathogenesis of the disease.[47] As a consequence, the adaptive immune response becomes severely impaired. This includes reduced chemokine co-stimulation, inadequate upregulation of major histocompatibility complex (MHC) molecules, and failure of lymphocyte activation and differentiation. Additionally, the virus suppresses interferon production, further weakening host immunity.[48,49]

4. Clinical manifestations

Ebola virus disease (EVD) in humans is characterized by a wide spectrum of clinical symptoms. The illness typically begins suddenly, following an incubation period of 2 to 21 days after exposure.[51]

Clinical presentation varies from person to person, and not all individuals exhibit the full range of symptoms. However, patients who become symptomatic are highly infectious during the acute stage of the illness, emphasizing the importance of rapid identification and isolation to prevent further spread of the virus.[53]

Pathophysiology

The Ebola virus is an intracellular parasite with a filamentous, enveloped structure that contains non-segmented, single-stranded RNA. Five genetically distinct Ebola virus genomes—ZEBOV, BDBV, REBOV, TAFV, and SEBOV—vary in their gene sequences and the arrangement of overlapping genes. Among these, VP35, VP30, the

nucleoprotein, and RNA-dependent RNA polymerase are essential components involved in viral replication and pathogenesis.

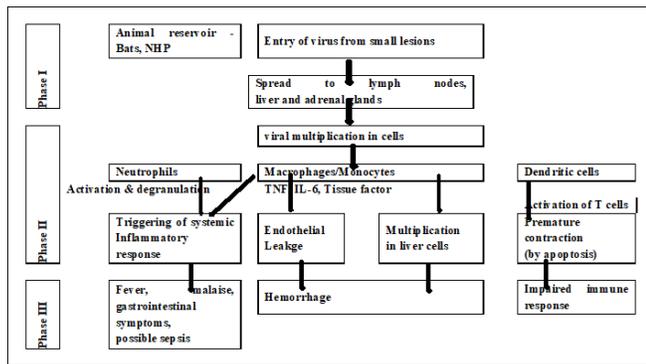


Fig.: 3 Pathogens of EVD

Pathogenesis of Ebola virus

Ebola virus can enter the human body through mucosal surfaces, breaks in the skin, close physical contact with infected individuals or their bodies, or via direct parenteral exposure. Once inside, EBOV preferentially targets several immune cell types—such as dendritic cells, monocytes, and macrophages—as well as endothelial and epithelial cells, hepatocytes, and fibroblasts. Within these cells, the virus undergoes active replication by manipulating host genes and inducing apoptosis, leading to markedly high levels of viremia.

Transmission routes

Although the complete life cycle of EBOV species is not fully understood, fruit bats are widely believed to serve as the natural reservoir. Evidence shows that Ebola virus can spread from bats to certain primate species, meaning that infected bats or primates can transmit the virus to humans when they are handled or consumed.

Clinical features of EVD

EVD is known to produce one of the most severe forms of viral hemorrhagic fever humans. Most patients initially develop sudden flu-like symptoms, including high fever, chills, fatigue, and muscle pain. These early signs may progress to widespread gastrointestinal issues such as vomiting and diarrhea, as well as respiratory complaints like chest pain and coughing.

Diagnosis

Diagnosing Ebola virus disease (EVD) is challenging because it requires a detailed patient history and thorough clinical evaluation. In regions where the virus is common, infections often occur through hunting or consuming bushmeat or bats, and through direct contact with sick individuals or corpses. In outbreak areas, laboratory methods such as ELISA, PCR, immunofluorescence, and histochemical techniques are used to confirm Ebola infection. Since the virus is abundant in skin tissues, post-mortem skin biopsies are commonly performed to verify Ebola infection. Earlier, serological diagnosis relied on the indirect fluorescent antibody test, but due to issues with sensitivity and clarity, it was replaced by the more reliable direct IgG ELISA test for assessing infection prevalence.

Treatment

There is no specific cure for Ebola hemorrhagic fever (EHF). Treatment primarily focuses on maintaining proper hydration, managing organ failure, and providing nutritional support along with antibiotics when necessary. To reduce trauma and prevent further spread of infection, invasive procedures such as injections, catheterization, and parenteral interventions were minimized. Certain medications—including aspirin, NSAIDs, anticoagulants, and steroids—are strictly avoided as they may worsen the condition.

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