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EBOLA VIRUS UPDATE: A REVIEW ON PATHOGENESIS, CLINICAL MANAGEMENT, AND GLOBAL CONTAINMENT (1976–2026).

Medical Microbiology

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INTRODUCTION

The Ebola virus is one of the most significant threats to global health security, characterised by its high mortality rate ranging between 20-80% and on an average of 50%. Further, it has the potential for rapid cross-border spread. Since its discovery in 1976, our understanding of the virus has evolved from managing mysterious "haemorrhagic fevers" to implementing precise molecular diagnostics, targeted monoclonal antibody therapies, and highly effective vaccines. This review article provides an updated clinical and scientific overview of the Ebola virus, drawing on the latest 2026 guidelines to outline the current state of taxonomic classification, viral morphology, pathogenesis, and the rigorous infection control measures required to contain outbreaks.[1]

Taxonomic Position and Classification

Ebola viruses belong to the order *Mononegavirales,* the family *Filoviridae*, and the genus *OrthoEbolavirus*. This family is characterised by filamentous, enveloped viruses that contain single-stranded, negative-sense RNA. Within the genus there are 4 species that are proven to cause human disease. Zaire ebolavirus is the most virulent species, responsible for the classic Ebola virus disease (EVD) and the most devastating historical outbreaks. Sudan Ebolavirus is responsible for Sudan virus disease (SVD). Bundibugyo Ebolavirus causing Bundibugyo virus disease (BVD) and Tai Forest ebolavirus associated with Tai Forest virus disease. The Reston and Bombali species had so far not caused any clinical disease in humans, though *Reston* is pathogenic in non-human primates.[1]

Viral Architecture

The virion measures approximately 80–100 nm in diameter and can reach lengths up to 14,000 -19000 nm. It appears as a filamentous, thread-like particle that is highly pleomorphic, often assumes alphabet "U" or numeric "6" shape or can be curved. The architecture of the virus consists of seven structural proteins and two non-structural [NS] or regulatory proteins. From outside to inside we have the viral envelope a lipid bilayer derived from the host cell's plasma membrane during the budding process. The primary antigen is the surface glycoproteins (GP) spikes that protrude from the envelope and is critical for host cell attachment to folate receptor α and various lectins and mediates membrane fusion. Besides it a secretory glycoprotein (sGP) is also synthesized by the virus which is antigenic decoy to absorb neutralizing antibodies. Further, it is the primary antibody target and candidate vaccine. The matrix layer is **c**omposed of VP40 (Matrix Protein) and VP24 (Viral Protein 24) proteins that provides structural integrity and is essential for assembly and budding. The central helical nucleocapsid core contains the negative-sense RNA genome wrapped in the nucleoprotein (NP) which protects the genome and play an essential role in viral replication. The polymerase complex consists of two different proteins VP35 (Viral Protein 35) a replication cofactor and the L protein is a large polymerase enzyme (RNA-dependent RNA polymerase) that carries out the superfast viral replication. VP 30 is the transcription activator.[2,3]

Virulence Factors: Subverting the Host Défense

The extreme virulence of *Zaire ebolavirus*, with case fatality rates (CFR) historically reaching **90%**, is because of a multi-pronged assault on the host immune system. The virus uses specific structural and regulatory proteins to subvert defences actively. VP35, acts as a potent immune antagonist by blocking the induction of Type I Interferons (IFNs). Moreover, it masks the presence of viral double-stranded RNA, preventing the host cell from sensing the infection and entering an antiviral state. VP24, binds to nuclear transporters (karyopherins), effectively blocking STAT signalling molecules from entering the nucleus. This shuts down the transcription of interferon-stimulated genes, crippling the body's ability to mount an effective défense. Glycoprotein (GP) facilitates entry and fusion of membrane. The virus also produces a secreted form (sGP) that is released into the blood and acts as a "decoy," binding to neutrophils and neutralising antibodies, which further impairs the immune response. VP40, beyond its structural role, it triggers prolonged inflammatory pathways that contribute to widespread tissue damage.[2,3]

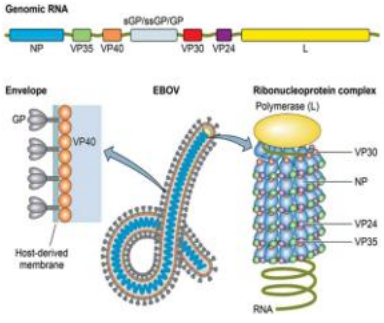

Fig.1 Showing Ebola Virus Morphology and Virulence Factors

Epidemiology and Transmission Dynamics

Ebola is a zoonotic disease with a natural reservoir as fruit bats (family *Pteropodidae*). "Spillover" events occur when humans encounter the blood, secretions, or organs of infected animals, including bats and non-human primates (apes, monkeys, or forest antelopes). Human-to-human transmission occurs through direct contact with body fluids (blood, vomit, faeces, urine, saliva, sweat, etc.) of an infected person who is symptomatic or through indirect contact with contaminated surfaces (bedding, clothing, medical equipment). Handling of the deceased bodies which remain highly infectious also results in disease transmission. However, Ebola is NOT airborne. A person is only contagious once they begin exhibiting symptoms. The incubation period ranges from 2 to 21 days, though most cases present within 8 to 10 days.[1]

History of Outbreaks (1976–2026)

Since the first recorded outbreaks in 1976 in the Democratic Republic of the Congo (formerly Zaire) and Sudan, the timeline of Ebola has been marked by periodic, devastating clusters. While primarily restricted to West and Central Africa, occasional laboratory-acquired outbreaks have occurred in the UK, Europe and Russia. The 2014–2016 West African outbreak remains the largest in history, though more recent outbreaks through 2026 have benefited from the rapid deployment of vaccines and therapeutics.

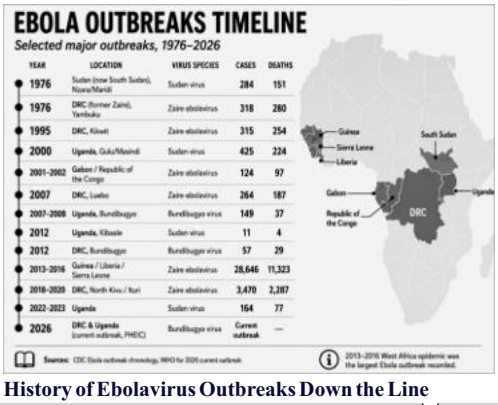

Fig.2 History of Ebolavirus Outbreaks Down the Line

Pathogenesis of Ebola Virus Disease – [From Infection to Multiorgan Failure]

Initial infection sets in following exposure to body fluids of a confirmed/suspected case of EBOD or infected animals and fruit bats. The virus primarily infects innate immune cells like macrophages, monocytes, and dendritic cells by attaching to host receptors like the TIM -1 & DC- SIGN. These cells then transport the virus to the lymph nodes, liver, spleen, and adrenal glands. This is followed by immune suppression mediated by VP24 and VP35, which blocks interferon signalling, allowing for unchecked replication and the death of lymphocytes (apoptosis). Hyper inflammation and cytokine storm results in when infected cells release great amounts of pro-inflammatory cytokines TNFα, IL6 and IL8 along with chemokines which manifest as fever, fatigue, and systemic shock. During illness, the virus directly infects the microvascular endothelium compromising the vascular integrity and leading to fluid leakage and hypovolemic shock. This triggers expression of tissue factor and consequent coagulation defects and disseminated intravascular coagulation with hallmark symptoms of internal and external bleeding [haemorrhage]. Massive tissue necrosis and widespread viral replication leads to focal necrosis [cell death] characterized by multiorgan failure [MOF] prominent in the liver and kidneys with deranged liver and renal function and ultimately death. Following acute disease, the virus persists for few weeks in the immune privileged sites [eye, brain, testis, placenta] in the survivors.[2,3]

Clinical Case Definition as per WHO: A possible case is one with compatible symptoms and epidemiological risk (travel/contact) within 21 days. While a probable case is a possible case with high-risk exposure (e.g., contact with body fluids) or an epidemiologically linked death where testing was unavailable. On the hand a confirmed case is any case with positive laboratory detection (RT-PCR or antibody).[4,5,6]

Clinical Features of Ebola Virus Disease [EBOD]

The incubation period varies from 2-21 days. Maximum infectivity is observed following onset of symptoms. The symptoms are initially like any other febrile illness e.g. influenza, malaria, typhoid fever, shigellosis, cholera, leptospirosis, rickettsial fever, sepsis and viral haemorrhagic fevers [dengue fever, Crimean Congo Haemorrhagic fever, Marburg virus disease and other similar VHFs]. The clinical presentation of EBOD is often divided into "dry" and "wet" phases. Early ("Dry") phase is characterized with sudden onset of fever, muscle pain (myalgia), headache, and fatigue. This is followed by the Late ("Wet") phase with symptoms of vomiting, diarrhoea, and unexplained bleeding or bruising. Discharge policy states that patients whose tests results are negative in two consecutive RT-PCR assays, taken at least 48 hours apart can be discharged from the hospital.[1,5,6]

The case fatality rate (CFR) is high as discussed above and those who recover from acute disease will face complications. Acute complications include severe metabolic and electrolyte derangements (acidosis), acute kidney injury, hepatitis, and neurological symptoms such as seizures or meningoencephalitis. While the Post-Ebola Syndrome manifest as extreme fatigue and muscle/joint pain. Patients with ocular complications presents with light sensitivity, eye pain, vision loss and even blindness. Peripheral neuropathy and peeling of skin are some other noted complications.[1,5,6]

Laboratory Diagnosis

The clinical and diagnostic pathway follows specific algorithms provided by WHO/CDC/EOCD guidelines. In first place screening and triage of patients is done which includes thorough history taking and clinical evaluation. This includes relevant travel or exposure history within the last 21 days alongside Ebola-compatible symptoms (e.g., fever, severe headache, muscle pain, vomiting). Facilities utilize prediction scores to categorize individuals into low, intermediate, or high-risk. Specimens like whole blood, serum, plasma, faeces, urine and semen are useful in diagnosis of EBOD. Specimens are to be collected from suspected and probable cases by healthcare workers strictly following BSL-2 containment measures.

Primary NAAT/RT-PCR is the gold standard for laboratory confirmation, detecting viral RNA. Window-Period Retesting is indicated because viremia may be undetectable in the early phase of infection, WHO guidelines dictate that if a living patient tests negative within 72 hours of symptom onset, a second test must be performed on blood drawn after the 72-hour mark. Diagnosis must be conducted under maximum biological containment typically BSL-4.

Besides RT-PCR several point of care tests (PoC's) based on immunochromatography principle are available for screening and triage of patients to minimize the spread of disease. They are under different stages of approval by FDA & WHO. The "Assured Criteria" is recommended by WHO for developing PoC devices. The diagnostic target for antigen detection is the nucleoprotein, VP40 or glycoprotein antigen in whole blood, plasma, serum, oral swabs and urine with varied sensitivity and specificity ranging between 50- 97% and 90-98%. Most of them are based on lateral flow assay principle. Rapid immunochromatography diagnostic test [RIDT] focusing on antibody detection are also available.

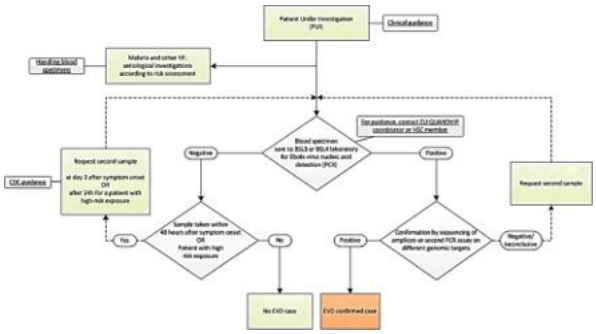

Fig.6 Algorithm for Laboratory Diagnosis of Ebola Virus Disease Adapted from ECDC

- Standard Test: Real-Time Reverse Transcription-Polymerase Chain Reaction (RT-PCR) is the gold standard for detecting viral RNA in blood. It is most accurate between days 3 and 10 of symptoms.
- Specimen Requirements: A minimum of 4 mL of whole blood in plastic EDTA tubes. Glass and heparinized tubes are rejected as they interfere with PCR.
- Screening Protocol: If a symptomatic patient tests negative within the first 48 hours, they must be re-tested several days later to confirm the result.
- Discharge: Patients are only cleared for discharge after two consecutive negative RT-PCR tests, taken at least 48 hours apart.[8]

Treatment and Targeted Therapeutics

The cornerstone of management is optimized supportive care combined with specific monoclonal antibodies. Supportive care includes aggressive IV fluid and electrolyte resuscitation to combat the massive loss of fluids from vomiting and diarrhoea. This also includes blood pressure management and symptom control (pain/fever). Targeted therapy is by administering monoclonal antibodies early in the course of infection. The FDA and WHO approved highly effective targeted treatments for *Zaire ebolavirus* are Inmazeb (atoltivimab, maftivimab, and odesivimab-ebgn) and Ebanga (ansuvimab). Many newer drugs in the form of combination of monoclonal antibodies are in pipeline for effective management of the disease. [8]

Vaccination is the primary tool for breaking chains of transmission in at-risk populations during outbreaks or epidemics. The two WHO approved vaccines are Ervebo (Merck & Co.) administered in single dose to prevent disease caused by *Zaire ebolavirus*. Another vaccine called Zabdeno and Mvabea (Janssen) is a two-dose vaccine regimen. There are several candidate vaccines for other strains of Ebola virus like Sudan virus (SVD) and Bundibugyo virus disease (BVD) in various stages of clinical development. Ring vaccination strategy is recommended by CDC/WHO during outbreaks and epidemics for containing the spread. [9]

Infection Prevention and Control (IPC) Measures for EBOD

Besides routine standard precautions, specific transmission-based precaution are to be followed by health care professionals, people engaged in handling of deceased cadavers, family members or close contacts of EBOD cases. CDC provides comprehensive guidelines for IPC measures to be observed for different categories of EBOD disease i.e. confirmed unstable cases, confirmed stable cases and suspected cases of EBOD. An interim guidance is also available on CDC website for environmental control, spill management and waste disposal for EBOD. Health care professionals providing care to patients with confirmed, suspected EBOD disease must be trained by skilled personnel and their competency to be assessed prior to placement. Subsequently their IPC practices to be monitored by trained supervisors on regular basis. Moreover, the CDC guidance states that

anyone who is found noncompliant to IPC measures in health care setting is to be immediately removed from providing patient care.[10]

In general IPC measures for EBOD include isolation of patient in a single room with negative pressure and accompanying anteroom facility for change of PPE, use of patient dedicated equipment as far as possible. Use of single use disposable set of personal protective equipment like double pair of gloves with inner pair of gloves with cuff, coverall or impermeable gowns, additional splash proof plastic aprons, N95 respirator or PAPR, face shield, goggles and shoe covers.[10]

Waste generated during patient care to be double-bagged and disposed as per the recommendation for containment category of pathogens of class 4. The housekeeping staff too are to be trained for handling of patient waste and managing accidental spills of patient's body fluids. Further their competency to be assessed and monitored periodically by the skilled supervisor. Body fluid spills are to be managed using 0.5% sodium hypochlorite solution with exposure time of minimum 5 minutes or any other recommended chemicals in the CDC document. Environmental cleaning to be frequent and to be done with recommended chemicals in the guidance document available on the website.[10]

CONCLUSION

The 2026 outlook for Ebola control is one of cautious optimism. While the virus remains inherently dangerous due to its molecular ability to subvert the immune system, the integration of rapid RT-PCR diagnostics, effective monoclonal antibody treatments, and proactive ring-vaccination strategies has transformed the clinical landscape. However, the success of these interventions depends entirely on the rigorous application of infection prevention and control measures. Healthcare systems must remain vigilant, ensuring that staff are trained in the meticulous donning and doffing of PPE and that public health responses including contact tracing and safe burial of diseased individuals immediately upon the identification of a suspected case.

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