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Ebola Virus Disease: A Comprehensive Review of Epidemiology, Pathogenesis, Diagnosis, Treatment, and Prevention

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ABSTRACT

Ebola Virus Disease (EVD) is a severe, often fatal zoonotic disease caused by viruses of the genus Ebolavirus (family Filoviridae). It is characterized by hemorrhagic fever, gastrointestinal symptoms, multi-organ dysfunction, and shock, with mortality reaching up to 90% in some Zaire ebolavirus infections. Fruit bats of the family Pteropodidae are considered the natural reservoir, with transmission occurring through direct contact with infected animals or human bodily fluids. Human-to-human transmission is the primary route during outbreaks. Viral proteins such as GP, VP24, and VP35 contribute to immune evasion and disease progression. Diagnosis involves RT-PCR and serological assays such as ELISA. Although supportive care remains essential, approved therapeutics, including Inmazeb and Ebanga, have improved treatment outcomes. The rVSV-ZEBOV (Ervebo) vaccine provides effective protection. A One Health approach integrating human, animal, and environmental health is crucial for surveillance, prevention, preparedness, and global outbreak control.

Introduction

Ebola Virus Disease (EVD) is a highly infectious and often fatal disease that affects humans and nonhuman primates. Disease is caused by viruses of the family Filoviridae, genus Ebolavirus. EVD is characterized by severe hemorrhagic fever and a high case-fatality rate. The genus Ebolavirus includes six species: Zaire ebolavirus (EBOV), Sudan ebolavirus (SUDV), Taï Forest ebolavirus (TAFV), Bundibugyo ebolavirus (BDBV), Reston ebolavirus (RESTV), and Bombali ebolavirus (BOMV). Among these, EBOV, SUDV, TAFV, and BDBV are known to cause disease in humans, with EBOV being the most lethal, exhibiting case-fatality rates that can reach up to 90% (Feldmann and Geisbert, 2011).The Ebola virus is a zoonotic pathogen. The primary natural reservoir of the Ebola virus is believed to be fruit bats of the Pteropodidae family, although the exact reservoir species has not been definitively identified. Humans can contract the virus through direct contact with the blood, secretions, organs, or other bodily fluids of infected animals such as fruit bats, chimpanzees, gorillas, monkeys, forest antelope, and porcupines found ill or dead in the rainforest (Leroy et al., 2005).Once the virus

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has entered the human population, it spreads through human-to-human transmission via direct contact with the blood, secretions, organs, or other bodily fluids of infected individuals, as well as surfaces and materials (e.g., bedding, clothing) contaminated with these fluids. Healthcare workers and family members are at particularly high risk during outbreaks due to their close contact with patients (Beeching et al., 2014; Chertow et al., 2014). EVD is marked by a rapid onset of symptoms, beginning with a nonspecific febrile illness that progress to severe gastrointestinal symptoms, such as vomition and diarrhea. In highly viraemic patients, the disease advances to a multiple organ dysfunction syndrome, leading to shock, severe coagulopathy, and death. The immune response to Ebola virus infection is often dysregulated, contributing to the high mortality rate (Baize et al., 2009; McElroy et al., 2020).The significance of EVD extends beyond its high fatality rate. The disease poses a considerable threat to global health security due to its potential for rapid spread and the severe impact it has on affected communities and healthcare systems. The outbreaks occurring from 2013-2016 in West Africa, which resulted in over 28,000 cases and more than 11,000 deaths, highlighted the devastating potential of EVD and the critical need for effective surveillance, response strategies, and medical countermeasures (Briand et al., 2014; Coltart et al., 2017). Recent advances in medical research have led to the development and approval of vaccines and therapeutics targeting the Ebola virus. These advancements represent significant progress in the fight against EVD, offering hope for better prevention and treatment options. A systematic review reported that Inmazeb, administered at 3 ml/kg IV, reduced mortality by 17%, while Ebanga showed a 35.1% mortality rate with a single 50 mg/kg IV dose (Sivanandy et al., 2022). Nevertheless, continued vigilance, international cooperation, and investment in research are essential to mitigate the impact of future outbreaks and enhance global health security (Sullivan et al., 2009; Henao-Restrepo et al., 2017).

History of Ebola Virus Disease

EVD was first identified in 1976, marking the onset of a series of outbreaks that would capture global attention due to the high fatality rate of the disease and devastating impact on affected communities. The initial outbreaks occurred almost simultaneously in two different regions: near the Ebola River in Zaire (now the Democratic Republic of Congo) and in Nzara, Sudan. These outbreaks resulted in 318 cases and 280 deaths in Zaire and 284 cases with 151 deaths in Sudan. The outbreaks were characterized by high fatality rates, severe hemorrhagic symptoms, and rapid transmission, laying the foundation for the subsequent scientific and medical scrutiny of the Ebola virus (Feldmann and Geisbert, 2011). In 1989, a new strain of the Ebola virus, named Reston ebolavirus (RESTV), was discovered in monkeys imported to Reston, Virginia, from the Philippines. This strain was notable because, unlike other strains of the Ebola virus, it did not cause fatalities in humans. The discovery of RESTV highlighted the potential for cross-species transmission and raised concerns about the risk of importing and handling primates from regions where Ebola viruses might be present. In 1995 outbreak occurred in Kikwit, Democratic Republic of Congo, which was one of the major epidemics of EVD, resulting in 315 cases and 254 deaths. This outbreak garnered significant international attention and underscored the need for robust public health responses and international collaboration in managing and containing such outbreaks. The Kikwit outbreak also provided critical data and insights into the clinical manifestations, transmission dynamics, and potential control measures for EVD (Leroy et al., 2005). In 2000-2001, outbreak of EVD in Gulu, Uganda, led to 425 cases and 224 deaths. This outbreak was notable for the effective mobilization of resources by the Centers for Disease Control and Prevention (CDC) and the World Health Organization (WHO), which played a crucial role in containing the virus. The Uganda outbreak demonstrated the importance of rapid response and international cooperation in managing EVD outbreaks (Chertow et al., 2014; World Health Organization, 2020). The West Africa epidemic of EVD from 2014-2016 was the largest and most severe outbreak of EVD to date. It began in Guinea in December 2013 and quickly spread to Liberia and Sierra Leone. By the end of the epidemic, over 28,000 cases and 11,000 deaths were reported. The epidemic was declared an international crisis by the WHO in August, 2014. The scale of this outbreak highlighted the challenges of managing EVD in densely populated urban areas and underscored the need for improved healthcare infrastructure, surveillance, and community engagement. Experimental vaccines and treatments were deployed during this epidemic, marking a significant step forward in the fight against Ebola virus (Briand et al., 2014; Coltart et al., 2017). A major outbreak of EVD occurred in the eastern Democratic Republic of Congo between 2018 and 2020, resulting in over 3,400 cases and 2,280 deaths. This outbreak posed significant challenges due to ongoing conflict and insecurity in the region, which hindered response efforts. Despite these challenges, the deployment of new vaccines and treatments helped to control the outbreak and reduce mortality rates (Henao-Restrepo et al., 2017). In 2019, the European Medicines Agency approved the Ebola vaccine rVSV-ZEBOV (Ervebo). The WHO later prequalified this vaccine, facilitating its use in outbreak response. The approval of Ervebo marked a significant advancement in preventive measures against EVD, offering a valuable

tool for protecting at-risk populations and controlling future outbreaks (Sullivan et al., 2009). In 2021, a new outbreak occurred in Guinea, linked to the strain from the 2014-2016 West Africa epidemic. This outbreak served as a reminder of the persistent threat posed by EVD and the need for continued vigilance, surveillance, and readiness to respond to new cases. The recurrence of the virus in previously affected regions underscored the importance of maintaining robust public health systems and community engagement to prevent and control outbreaks.

Classification and etiology

Ebola virus is an enveloped, filamentous virion belonging to the family *Filoviridae* (order *Mononegavirales*), with characteristic thread-like morphology ranging from 800-1000 nm in length and 80 nm in diameter (Bharat et al., 2012). The viral envelope, derived from host cell membranes during budding, contains trimeric glycoprotein (GP) spikes that mediate host cell attachment and entry (Lee et al., 2008). These GP spikes are the primary targets for neutralizing antibodies and vaccine development (Pallesen et al., 2016). The virus possesses a non-segmented, single-stranded negative-sense RNA genome of approximately 19 kb that encoding seven structural proteins: Nucleoprotein (NP), Polymerase cofactor (VP35), Matrix protein (VP40), Glycoprotein (GP), Transcription activator (VP30), Minor matrix protein (VP24), and RNA-dependent RNA polymerase (L protein). The genome adopts a helical nucleocapsid structure formed by NP-VP35-RNA complexes, surrounded by VP40 and VP24 matrix proteins that facilitate virion assembly and budding (Wan et al., 2017). Notably, VP24 serves as a interferon antagonist, while VP35 functions as both a polymerase cofactor and innate immune evasion protein (Basler, 2017).

Ebolavirus species

There are six recognized species within the Ebolavirus genus, each differing in their hosts, characteristics, and geographic distribution. *Zaire ebolavirus* (EBOV) primarily infects humans and nonhuman primates. *Sudan ebolavirus* (SUDV) is associated mainly with humans and fruit bats. *Taï Forest ebolavirus* (TAFV) primarily affects humans, with bats considered a likely reservoir. Similarly, *Bundibugyo ebolavirus* (BDBV) mainly infects humans and is also suspected to be associated with bats. *Reston ebolavirus* (RESTV) has been detected primarily in nonhuman primates and pigs and is considered nonpathogenic to humans. Bombali ebolavirus (BOMV), the most recently identified species, is believed to be associated with bats, although its epidemiological significance and host range remain under investigation.

Etiology

EVD is a zoonotic disease caused by viruses belonging to the genus Ebolavirus. Fruit bats of the family Pteropodidae are considered potential natural reservoir hosts, as certain species may harbour the virus without showing apparent clinical signs (Leroy et al., 2005). Non-human primates, including chimpanzees and gorillas, are highly susceptible to infection and may serve as sources of human transmission through contact with infected blood, tissues, carcasses, or contaminated bushmeat (Feldmann and Geisbert, 2011). Among domestic animals, pigs are susceptible to Reston virus infection and have demonstrated the potential for virus transmission among themselves; however, their role in transmitting Ebola viruses to humans remains unclear (Chertow et al., 2014).

Ebola virus in humans

EVD historically referred to as Ebola haemorrhagic fever (EHF), is a severe and potentially fatal infectious disease in humans caused by viruses belonging to the genus Ebolavirus. The species associated with human disease include Zaire ebolavirus (EBOV), Sudan ebolavirus (SUDV), Bundibugyo ebolavirus (BDBV), and Taï Forest ebolavirus (TAFV). Reston ebolavirus (RESTV) has been identified in humans but has not been associated with symptomatic disease. EVD is characterized by an incubation period of 2–21 days, after which symptoms typically begin with fever, fatigue, headache, and muscle pain, followed by gastrointestinal manifestations and, in severe cases, organ dysfunction and haemorrhagic complications.

Transmission of EVD

EVD outbreaks typically originate from zoonotic transmission, with index cases often linked to direct contact with infected wildlife (e.g., fruit bats, nonhuman primates) or their bodily fluids (WHO, 2021). The virus then spreads through human-to-human transmission via direct contact with infectious fluids or contaminated fomites (Jacob et al., 2020).

Human-to-human transmission pathways

Human-to-human transmission of Ebola virus occurs primarily through direct contact with the blood and other infectious bodily fluids of symptomatic individuals or deceased patients. The major transmission pathways include:

Direct contact with infectious bodily fluids: The virus spreads through blood, feces, vomit, saliva, urine, sweat, breast milk, and semen (particularly persistent in semen

for months post-recovery) (Sissoko et al., 2017). High viral loads in late-stage patients make healthcare workers especially vulnerable during patient care (Briand et al., 2014).

Fomite transmission: Contaminated surfaces (e.g., bedding, medical equipment) remain infectious, with the virus surviving for hours to days depending on environmental conditions. Improperly sterilized needles have caused iatrogenic transmission in outbreak settings (Osterholm et al., 2015).

Mucocutaneous exposure: The virus enters through broken skin or mucous membranes (ocular, nasal, oral), emphasizing the importance of proper PPE use. Notably, unlike respiratory viruses, Ebola is not transmitted via airborne routes under natural conditions (WHO, 2021).

Zoonotic origin

EVD) is a zoonotic disease that can be transmitted from infected animals to humans through direct contact with infectious materials. Fruit bats belonging to the family Pteropodidae are considered potential natural reservoir hosts and are believed to harbour the virus without exhibiting apparent clinical signs (Leroy et al., 2005). Human infections have been associated with contact with infected wildlife, particularly through the handling or consumption of fruit bats and exposure to the blood, bodily fluids, or tissues of infected non-human primates, including monkeys and apes, often during hunting, carcass handling, and consumption of contaminated bushmeat (Feldmann and Geisbert, 2011).

Molecular Pathogenesis of Ebola virus

EVD is a severe viral infection associated with systemic inflammation, immune dysregulation, vascular dysfunction, and high mortality in severe cases. Its molecular pathogenesis involves complex interactions between viral proteins and host cellular factors that facilitate viral entry, replication, immune evasion, and tissue injury. The infection primarily affects the liver, adrenal cortex, lymphatic tissues, and immune cells, while disturbances in coagulation and vascular integrity contribute to systemic complications.

Glycoprotein (GP)

The surface glycoprotein (GP) of Ebola virus plays a crucial role in viral entry and disease pathogenesis. Viral entry: GP mediates viral attachment and entry into host cells through endocytosis, followed by membrane fusion. Host factors, including TIM-1, which may facilitate viral attachment, and Niemann–Pick C1 (NPC1), which is essential for intracellular entry, contribute to this process (Miller et al., 2012). Cytotoxic effects: Ebola virus GP can disrupt endothelial cell integrity, increasing vascular permeability and contributing to fluid leakage, haemodynamic instability, and shock in severe EVD cases (Yang et al., 1998).

VP24 and VP35 Proteins

Ebola virus proteins VP24 and VP35 are important determinants of viral replication and immune evasion. VP24: This protein interferes with interferon (IFN) signalling by inhibiting the nuclear import of phosphorylated signal transducer and activator of transcription 1 (STAT1), thereby limiting the host's antiviral response (Reid et al., 2006). VP24 also interacts with host cellular proteins involved in immune signalling and intracellular trafficking, further contributing to viral immune evasion (Zhang et al., 2012). VP35: VP35 suppresses the production of type I interferons by interfering with pathways responsible for activating interferon regulatory factors 3 and 7 (IRF-3/7), which are essential for IFN gene expression (Hartman et al., 2004). Additionally, VP35 can suppress RNA interference (RNAi)-mediated antiviral responses, thereby supporting viral replication and persistence within the host (Haasnoot et al., 2007).

Viral RNA Polymerase (L)

The large (L) protein of Ebola virus functions as an RNA-dependent RNA polymerase and is essential for viral genome replication and transcription. During replication, the L protein catalyses the synthesis of complementary RNA using the viral RNA genome as a template, with viral RNA synthesis occurring in association with intracellular replication complexes and inclusion bodies (Mühlberger et al., 1999). The L protein also transcribes viral messenger RNAs (mRNAs) from the negative-sense RNA genome. These transcripts encode structural proteins, including nucleoprotein (NP) and VP40, as well as regulatory proteins such as VP24 and VP35, which are required for viral replication, assembly, and release (Weik et al., 2002).

Targeted Tissues and Organ Damage

Ebola virus infection affects multiple tissues and organs, resulting in cellular injury, immune dysfunction, and systemic complications.

Liver: Ebola virus replication in hepatocytes contributes to cellular injury, apoptosis, and hepatic dysfunction. Impaired liver function can reduce the synthesis of coagulation factors and other essential proteins,

thereby contributing to coagulopathy and impaired haemostasis (Schnittler and Feldmann, 2003; Wauquier et al., 2010).

Coagulopathy: Ebola virus disease may cause disseminated intravascular coagulation (DIC), characterized by dysregulated coagulation activation, consumption of clotting factors and platelets, and impaired haemostatic balance. These disturbances can contribute to bleeding manifestations and severe systemic complications (Bray, 2005).

Adrenal cortex: Infection and injury involving adrenocortical cells may impair the production of cortisol and aldosterone, disrupting fluid balance, electrolyte regulation, and cardiovascular stability. These effects may aggravate hypotension, shock, and multiple-organ dysfunction syndrome (MODS) in severe cases (Martines et al., 2015).

Lymphatic tissues and immune cells: Ebola virus infection can cause lymphoid tissue damage and depletion of immune cell populations, compromising immune function and contributing to impaired host defence. These changes may increase susceptibility to secondary infections and worsen disease progression (Gupta et al., 2014).

Mechanism of Immune Dysregulation and Systemic Effects

The combined activities of Ebola virus proteins, particularly GP, VP24, VP35, and L, contribute to immune dysregulation, viral replication, and systemic disease progression. These mechanisms enable the virus to evade host defence responses and induce widespread tissue injury.

Immune evasion: Ebola virus proteins interfere with innate immune signalling pathways, particularly those involved in interferon production and response, thereby weakening antiviral defences and facilitating viral replication and dissemination within the host (Hartman et al., 2004; Reid et al., 2006).

Systemic manifestations: Severe Ebola virus disease is associated with excessive inflammatory responses, coagulation abnormalities, increased vascular permeability, and multiple-organ dysfunction. These complications arise from a combination of direct viral effects, immune cell dysfunction, endothelial injury, and dysregulated cytokine production, which may lead to a cytokine storm and contribute to disease severity (Baize et al., 2002).

Signs and Symptoms

The clinical manifestations of EVD typically develop within an incubation period of 2-21 days following exposure. Early symptoms are generally nonspecific and include fever, headache, muscle pain, profound fatigue, weakness, sore throat, and chills, which may progress rapidly to more severe clinical manifestations (Feldmann and Geisbert, 2011). As the disease advances, patients may develop gastrointestinal disturbances, particularly vomiting and diarrhoea, accompanied in some cases by a skin rash. Severe infection may be associated with coagulation abnormalities, including disseminated intravascular coagulation (DIC), petechiae, ecchymoses, and mucosal bleeding. Progressive organ involvement can lead to hepatic and renal dysfunction, jaundice, and other manifestations of multiple-organ impairment. In advanced cases, severe fluid loss, vascular dysfunction, and systemic complications may result in hypotensive shock, which is a major contributor to mortality in EVD (Feldmann et al., 1993; Bray, 2005).

Pathology of EVD

The pathological features of Ebola virus disease (EVD) described in this article are primarily based on observations from human cases and validated non-human primate studies, which provide important insights into disease pathogenesis. Gross lesions commonly include hepatomegaly, hepatic congestion, mottling, haemorrhagic foci, and hepatocellular necrosis (Schnittler and Feldmann, 2003). The spleen may exhibit enlargement, congestion, and surface haemorrhages (Gupta et al., 2004). Haemorrhagic manifestations, including petechiae and ecchymoses on the skin and mucous membranes, reflect vascular injury and coagulation abnormalities, while gastrointestinal, respiratory, and other mucosal haemorrhages may occur in severe cases (Feldmann et al., 1993; Baize et al., 2002). Microscopically, the liver exhibits extensive hepatocellular necrosis, sometimes accompanied by Councilman bodies, which are eosinophilic apoptotic bodies, along with sinusoidal congestion and erythrocyte accumulation (Wauquier et al., 2010; Schnittler and Feldmann, 2003). The spleen and lymph nodes show lymphoid cell necrosis and depletion, accompanied by sinusoidal congestion and haemorrhage (Gupta et al., 2004; Feldmann et al., 1993). Renal lesions may include necrosis of tubular epithelial cells, whereas pulmonary pathology can involve interstitial pneumonia, inflammatory cell infiltration, and alveolar haemorrhage (Martines et al., 2015).

Host Immune Response in EVD

The immunological features of Ebola virus disease (EVD) are primarily characterized by interactions between innate and adaptive immune responses, as demonstrated in human cases and validated non-human primate studies. The innate immune response begins when macrophages and dendritic cells recognize viral components through pattern recognition receptors, triggering the release of pro-inflammatory cytokines, including tumour necrosis factor-alpha (TNF-α) and interleukin-6 (IL-6). Excessive cytokine production may result in a cytokine storm, contributing to systemic inflammation and disease progression (Sullivan et al., 2003; Gupta et al., 2014). Ebola virus proteins VP24 and VP35 interfere with interferon signalling and antiviral immune mechanisms, while impaired dendritic cell function may delay the development of effective adaptive immunity and antibody responses during acute infection (Basler, 2017; Messaoudi et al., 2015). In contrast, survivors develop durable humoral and cellular immune responses, including neutralizing antibodies against viral glycoproteins, memory B cells, and functional T-cell responses (Bornholdt et al., 2016; Rijal et al., 2019). The protective role of adaptive immunity is further supported by the efficacy of the recombinant vesicular stomatitis virus-based Ebola vaccine (rVSV-ZEBOV) (Henao-Restrepo et al., 2017; Diallo et al., 2016). Long-term immune protection in survivors is associated with the persistence of Ebola virus-specific antibodies and memory T cells, which contribute to sustained immunological memory and may provide protection against subsequent infection, although the extent and duration of protection can vary (McElroy et al., 2020).

Diagnosis of EVD

The diagnosis of EVD relies on molecular, serological, virological, and immunohistochemical techniques to confirm infection, assess disease progression, and support outbreak surveillance. Reverse transcription polymerase chain reaction (RT-PCR) is a primary diagnostic method for detecting Ebola virus RNA in clinical specimens, including blood, plasma, serum, and oral swabs, and is widely used for early case confirmation and outbreak management (Broadhurst et al., 2016; Mbala-Kingebeni et al., 2019). Serological assays, particularly enzyme-linked immunosorbent assays (ELISA), detect virus-specific immunoglobulin M (IgM) and immunoglobulin G (IgG) antibodies, supporting the assessment of recent or previous infection, immune responses, and vaccine-induced seroconversion (Feldmann et al., 1993; WHO, 2022; Rivera et al., 2015). Virus isolation in cell culture enables detailed virological investigations, including viral characterization and studies of replication and antiviral susceptibility, but requires specialized high-containment facilities, generally biosafety level 4 (BSL-4), because of the high pathogenicity of Ebola virus (Feldmann et al., 2003; Halfmann et al., 2019; Mehedi et al., 2011). Quantitative RT-PCR provides estimates of viral RNA levels, with higher viral loads, often reflected by lower cycle-threshold (Ct) values, having been associated with poorer clinical outcomes; however, Ct values must be interpreted in the context of the assay and clinical findings (de la Vega et al., 2015; Sissoko et al., 2017). The detection of IgM followed by IgG seroconversion can help characterize the evolving antibody response during infection and recovery, while serological testing also supports retrospective surveillance and population-level investigations (Liu et al., 2018; Ravichandran et al., 2022). Immunohistochemistry (IHC) is useful for detecting viral antigens in tissues and investigating viral distribution and tissue tropism, including in macrophages within the spleen, liver, and lymph nodes, as well as in organs such as the brainstem and gastrointestinal tract in non-human primate models (Mani et al., 2025; Banadyga et al., 2021). Molecular testing can also support species-specific identification of Zaire ebolavirus (EBOV), Sudan ebolavirus (SUDV), and Reston ebolavirus (RESTV), while serological assays contribute to epidemiological investigations and studies of host exposure. RT-PCR is particularly important for diagnosing acute EBOV and SUDV infections, whereas testing of animal samples, including those from pigs and non-human primates, can assist RESTV surveillance. The selection and interpretation of diagnostic methods depend on the stage of infection, specimen type, and epidemiological context. Collectively, these approaches are essential for laboratory confirmation, clinical assessment, epidemiological surveillance, and effective outbreak control.

Treatment of Ebola Virus Disease

The management of Ebola virus disease (EVD) involves comprehensive supportive care and specific therapeutic interventions aimed at reducing mortality and preventing complications. Supportive care remains fundamental and includes oral or intravenous fluid and electrolyte replacement to manage dehydration caused by vomiting, diarrhoea, and fever; oxygen supplementation and appropriate blood pressure management to stabilize patients experiencing respiratory distress or shock; and prompt treatment of secondary infections, sepsis, and organ-specific complications, particularly hepatic and renal dysfunction. Early and intensive supportive interventions are essential for improving clinical outcomes, especially in patients with severe disease (Jaspard et al., 2022; Sivanandy et al., 2022). Among antiviral therapies, remdesivir, a nucleotide analogue that inhibits viral RNA-dependent RNA polymerase, has been investigated for

its activity against Ebola virus, while favipiravir has also been evaluated for its potential to inhibit viral replication; however, remdesivir is not an approved standard treatment for EVD, and clinical evidence has not established favipiravir as an effective therapy (Mulangu et al., 2019; Letafati et al., 2023). Monoclonal antibody therapies have demonstrated greater clinical benefit, particularly for Zaire ebolavirus infection. Earlier candidates, such as ZMapp, and the monoclonal antibody mAb114 contributed to the development of targeted treatment strategies (Mulangu et al., 2019). Currently, the approved treatments Inmazeb, a combination of atoltivimab, maftivimab, and odesivimab, and Ebanga (ansuvimab) are important therapeutic options for EVD caused by Zaire ebolavirus. Inmazeb combines three monoclonal antibodies targeting the viral glycoprotein, whereas Ebanga is a single monoclonal antibody directed against the same viral target; both are administered intravenously and have demonstrated survival benefits in clinical studies (Sivanandy et al., 2022). Convalescent plasma therapy involves administering plasma collected from recovered EVD patients to provide passive immunity through virus-specific antibodies. Although this approach has been investigated during Ebola outbreaks, its clinical effectiveness remains uncertain, and outcomes may vary according to antibody concentration, donor characteristics, and the timing of administration (Mulangu et al., 2019; Franchini et al., 2021). Overall, early diagnosis, high-quality supportive care, and prompt administration of effective monoclonal antibody therapy, where indicated and available, remain central to improving outcomes in patients with EVD.

Control and Prevention Measures

Effective control and prevention strategies are essential for limiting the transmission of Ebola virus disease (EVD) and containing outbreaks. Key measures include the prompt isolation of confirmed cases, appropriate monitoring and quarantine of individuals with relevant exposure histories in accordance with public health guidelines, and the implementation of rigorous infection prevention and control practices. Consistent use of appropriate personal protective equipment (PPE), proper hand hygiene, safe handling and disposal of infectious materials, and thorough disinfection of contaminated surfaces are particularly important in healthcare settings to minimize the risk of transmission (Woolsey et al., 2021). Vaccination is another important component of outbreak prevention, particularly against Zaire ebolavirus. The recombinant vesicular stomatitis virus-based vaccine rVSV-ZEBOV (Ervebo) has demonstrated high effectiveness in ring vaccination campaigns and is used as a preventive measure during outbreaks. Its protective effectiveness depends on the timing of administration, with protection developing over time following vaccination (Meakin et al., 2024).

Public Health Measures

Comprehensive public health interventions are essential for preventing the spread of EVD and effectively managing outbreaks. Public awareness campaigns play a vital role in educating communities about transmission pathways, clinical symptoms, and preventive practices, thereby promoting early detection, timely reporting, and appropriate health-seeking behaviour. Safe burial practices, including the use of appropriate personal protective equipment and the careful handling of deceased individuals, are equally important in minimizing exposure to infectious bodily fluids and reducing transmission risks. In addition to its health consequences, EVD can have substantial socioeconomic impacts by placing considerable pressure on healthcare systems and disrupting agricultural activities, trade, tourism, and other economic sectors because of public fear and restrictions associated with disease outbreaks (WHO, 2021).

Potential as a Biological Weapon

The high pathogenicity of Ebola virus and its potential to cause severe disease and substantial mortality have raised concerns regarding its possible misuse as a biological weapon. Its association with severe outbreaks, high case-fatality rates in certain settings, and the challenges associated with clinical management make it a pathogen of public health and biodefence concern (Leitenberg, 2005). Addressing this potential threat requires robust disease surveillance, effective laboratory biosafety and biosecurity measures, strengthened public health preparedness, and international collaboration to facilitate early detection, rapid response, and coordinated outbreak management. These measures are essential for reducing the risks associated with the deliberate misuse of highly pathogenic infectious agents and protecting global health security.

Vaccine Development for Ebola Virus

Animal models, particularly guinea pigs, mice, and non-human primates, play an important role in preclinical Ebola virus vaccine research because they enable the evaluation of vaccine safety, immunogenicity, and protective efficacy. Several vaccine strategies have been investigated, including live-attenuated approaches, recombinant protein vaccines, and DNA vaccines designed to

stimulate virus-specific immune responses. DNA vaccines encoding Ebola virus glycoprotein (GP) and secreted glycoprotein (sGP) have demonstrated protective potential against lethal viral challenge in preclinical animal studies (Mire et al., 2015). These vaccines can stimulate both humoral and cellular immune responses, which are essential components of antiviral immunity (Warfield et al., 2007). Furthermore, prime–boost vaccination strategies, involving sequential administration of different vaccine platforms, such as viral vectors, adenovirus-based vaccines, and DNA vaccines, have been investigated to enhance immune responses and protective efficacy in non-human primate models. The Ebola virus glycoprotein (GP) remains a major target for vaccine development because it mediates viral entry into host cells and contributes to disease pathogenesis (Mire et al., 2015). Accordingly, GP-based vaccines aim to induce specific antibodies capable of neutralizing the virus and preventing infection, thereby providing protective immunity against Ebola virus disease (Warfield et al., 2007).

Conclusion

Ebola virus disease (EVD) remains a significant global health concern, necessitating an integrated One Health approach that addresses the interconnections between human, animal, and environmental health. Strengthening coordinated surveillance systems, monitoring potential wildlife reservoirs, and improving early detection and rapid response mechanisms are essential for reducing zoonotic transmission risks and preventing outbreaks from escalating into public health emergencies. Effective policy frameworks and regulatory measures should promote collaboration among medical, veterinary, and environmental sectors to facilitate coordinated disease prevention and control. Investment in research and development is equally important for advancing diagnostic techniques, vaccines, and therapeutic interventions. Integrating EVD prevention strategies with the Sustainable Development Goals (SDGs), particularly those related to health, food security, and biodiversity conservation, can further support sustainable development in affected regions. In addition, strengthening laboratory infrastructure, enhancing the capacity of healthcare and veterinary professionals, and promoting public awareness of transmission pathways and preventive practices are vital for effective outbreak preparedness and management. Sustained international cooperation, equitable access to medical interventions, and the sharing of scientific knowledge and resources are also essential for protecting vulnerable populations. Ultimately, a coordinated, multidisciplinary, and proactive approach is necessary to reduce the impact of EVD, strengthen global health security, and improve preparedness for future emerging infectious disease threats.

Conflicts of interest and financial disclosures

The authors state that there are no conflicts of interest to disclose.

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