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The Tactical Strategy, Readiness, Epidemiology and Reaction to Potential Treatments for the Possibly Lethal Ebola Virus: A Review

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Authors' contributions

This work was carried out in collaboration between all authors. Authors RV and NG designed the study, performed the statistical analysis, wrote the protocol, and wrote the first draft of the manuscript. Authors KF, RP, Varnika, PS and RB managed the analyses of the study. Authors KB, Arushi Semwal, Ayushi Semwal, SJ and VM managed the literature searches. All authors read and approved the final manuscript.

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Review Article

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ABSTRACT

Over the preceding 38 years, a few outbreaks have been brought on by the Ebola virus, which produces the Ebola illness. No specific treatment has been approved for EVD. Problem management and supportive care are the cornerstones of treatment. Effective outbreak control requires a multidisciplinary team effort that includes case care, infection prevention and control protocols, contact tracing and surveillance, a high-quality laboratory service, dignified and safe funerals, and social and community mobilization. The 2014 Ebola outbreak started in Africa and swiftly spread to other continents before turning into a pandemic. The illness gained international interest because to its relatively peculiar design, lethality and contagiousness, difficulty in containing its spread, and absence of a reliable treatment. Two medications have received FDA approval to treat EVD. Ebanga is a single monoclonal antibody, whereas Inmazeb is a mixture of three monoclonal antibodies. Individuals utilizing any of the two FDA-approved treatments had a significantly greater overall survival rate. In this article, the known history of the Ebola virus, its mode of infection, epidemiology, lifecycle, and possible treatments are briefly reviewed.

Keywords: EVD; ebola scourge; infectivity and lethality; epidemiology; symptoms.

1. INTRODUCTION

The new pandemic of Ebola infection illness (previously known as Ebola haemorrhagic viral sickness) started in Guinea in December 2013. After and report on Walk 23rd, 2014, WHO (World Health Organization) became involved when advised of the extent of the issue (WHO, EVD in spain,2014). Ebola infection sickness (EVD) was first found in 1976 in the rule of Democratic, (DRC) Republic of Congo, which was previously known as Zaire town close to the Ebola River. As indicated by the important viral and epidemiologic information, the Ebola infection has long existed before any recorded outbreaks. In any case, because of urbanization development, attack of forested regions, and close connection with untamed life creatures, the spread of the Ebola infection has made the deadliest illnesses creatures and people. Until now, the regular supply host of the Ebola infection has not been identified at this point, however, researchers accepted that the African natural product bats are logically engaged with the spread of the Ebola infection and may try and be the repository. Researchers keep looking for convincing proof of the bat's part in the transmission of Ebola and the latest Ebola infection recognized was the Bombali infection which was distinguished in examples from bats gathered in Sierra Leone. Ebola disease refers to a range of fatal diseases in humans caused by four ebolaviruses in the genus Ebolavirus. Ebola disease epidemics in humans occur on a regular basis, particularly on the African continent [35,43]. Each of the four ebolaviruses that cause human sickness, together with their associated viral species and disease name, are listed below:

1. Ebola infection (species Zaire ebolavirus) causes Ebola infection sickness.
2. Sudan infection (species Sudan ebolavirus) causes Sudan infection sickness.
3. Taï Woods infection (species Taï Woodland ebolavirus, previously Côte d'Ivoire ebolavirus) causes Taï Timberland infection sickness.
4. Bundibugyo infection (species Bundibugyo ebolavirus) causes Bundibugyo infection sickness (cases of Ebola Diagnosed in the United States, 2014) [36].

Ebola infection (EBOV) has a place with the family Filoviridae, the sort of Ebolavirus, and often causes lethal contamination in people. EBOV sickness (EVD) may show numerous, sequential, and vague illness side effects including high fever, cerebral pain, heaving, anorexia, looseness of the bowels, and throbbing muscles. Unexplained draining in the eyes, nose, gums, and stomach happens in the high-level stages. The main flare-up of EVD was accounted for in 1976 in the Popularity based Republic of the Congo. Since then, there have been reports of small EVD outbreaks in some countries in Central Africa, including Sudan and Ugandal, with an estimated 2350 cases of EVD occurring between the 1970s and 2013 (*International Commission, 1978*). The illness can consequently be viewed as endemic to certain areas of Focal Africa. In Walk 2014, an episode

of EVD was accounted for without precedent for Africa (West Africa), in Guinea, and it spread quickly to adjoining nations including Sierra Leone and Liberia and, making a serious epidemic. This has caused significant wellbeing concerns both in and past the locale, with the WHO (World Health Organization) and various nations starting wellbeing checking and regulation measures [28,29]. We portray here the past and current plagues, the study of disease transmission, clinical elements, conclusion, and treatment of EVD as depicted to date in the literature [6, 47]. Recombinant vesicular stomatitis virus-based vaccine expressing a ZEBOV glycoprotein (rVSV-ZEBOV) was given to a patient who developed Ebola during the 2018–2020 outbreak in the Democratic Republic of the Congo's North Kivu province. He recovered in 14 days thanks to treatment that contained an Ebola virus (EBOV)-specific monoclonal antibody (mAb114). But six months later, he returned with EBOV viremia and a severe EVD-like sickness, and this time, he passed away. We started epidemiologic and genetic studies, which revealed that the patient had experienced an acute EVD relapse. This set off a chain of transmission that resulted in 91 cases over the course of four months, spanning six health zones.The Ebola virus (EBOV) is the cause of the severe and often fatal sickness known as Ebola virus disease (EVD). EVD outbreaks usually begin with a single incidence of likely zoonotic transmission, which is then followed by human-to-human transmission by contact with contaminated food or bodily fluids. Multiple organ dysfunction syndrome, gastrointestinal symptoms, fever, and EVD have a high case-fatality rate. A combination of laboratory testing, usually real-time reverse transcription PCR to identify viral RNA or quick diagnostic tests based on immunoassays to identify EBOV antigens, are needed to make the diagnosis. European and US regulatory agencies have approved an EBOV-targeted vaccination as a result of recent advancements in medical countermeasure research. Two monoclonal antibody products that target the EBOV membrane glycoprotein were shown to improve survival in a randomized clinical study of experimental therapies for EVD. New insights into EVD and viral persistence in survivors of EVD have been gained from the extraordinary 2013–2016 Western African EVD outbreak, which was the largest in history, as well as the ongoing EVD outbreak in the Democratic Republic of the Congo. These insights have led to the development of new approaches to infection prevention, clinical management optimization, acute illness outcomes, and patient attention to clinical care needs [59].

2. MODE OF INFECTION

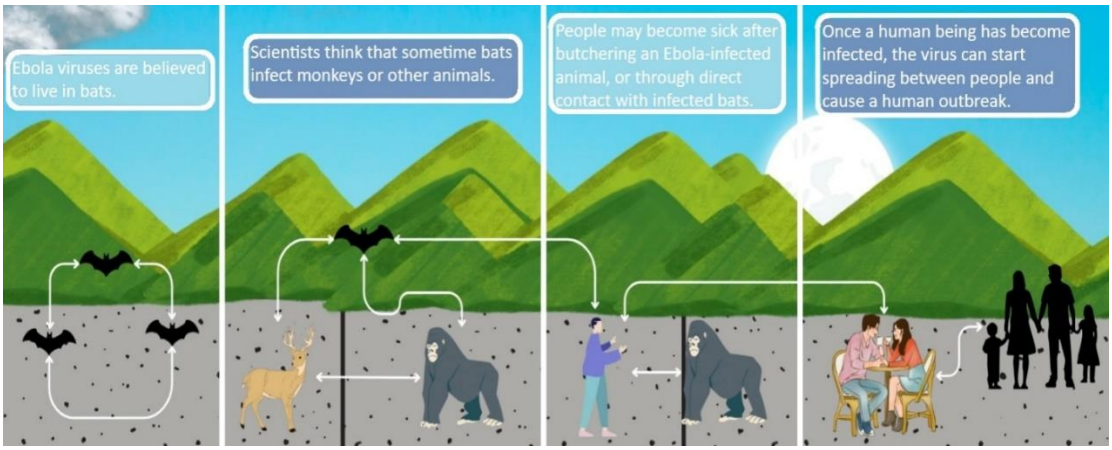
Scientists think people are initially infected with an ebolavirus through contact with an infected animal, such as a fruit bat or nonhuman primate. This is called a spillover event. After that, the virus spreads from person to person, potentially affecting many people (*Kerstiëns and Matthys, 1999)*. These viruses can spread from person to person by contact with polluted fluids, which makes them more prevalent in impoverished settings. The natural reservoir for the Ebolavirus is thought to be the African fruit bat, or Rousettus aegyptiacus. These bats can spread the virus to antelopes, monkeys, and apes that live in forested environments. Living in forested environments, handling dead bodies infected with the Ebolavirus, and consuming such animals are all regarded as risk factors linked to cultural and religious practices that impede the containment of epidemics in these locations. Uses spread through contact (such as through mucous membranes in the eyes, broken skin, nose, or mouth) with:

a. Objects (like bedding, garments, needles, and clinical equipment) defiled with body liquids from a debilitated individual has or with kicked the bucket from Ebola infection [17].
b. Infected fruit bats or nonhuman primates (such as apes and monkeys).
c. Blood or body fluids (saliva, sweat, urine, vomit, feces, breast milk, semen, and amniotic fluid) of a person who is sick has died from Ebola diseasse [17,56].
d. Semen from a man who recovered from Ebola disease (through oral, vaginal, or anal sex). Ebolaviruses can remain in certain body fluids (including semen) of a patient who has recovered from Ebola disease, even if they no longer have symptoms of severe illness. There is no evidence that ebolaviruses can spread through sex or other contact with vaginal fluids from a woman who has had Ebola disease [17,56]
e. Previous outbreaks give opportunities to study the transmission of Ebola virus from person to person. Within hospitals spread has been recorded numerous times, and amplification of outbreak has happened in both SUDV and EBOV healthcare settings. Early investigations of epidemic revealed the

significance transmission of parenteral via nonsterile needles, albeit this has since been overlooked [21]. Furthermore, researchers have discovered that healthcare employees are particularly vulnerable. During previous SUDV and EBOV epidemics, the use of surgical masks and barrier protection appeared to be sufficient to prevent most nosocomial transmission. However, in one SUDV outbreak, out of 22 the 14 (64%) healthcare workers became sick after the important barrier precautions were applied or implemented, resulting in the reinforcement of important infection control practices. Three studies (two on SUDV and one on EBOV) looked at the risk of illness among the household contacts based on a history of direct the physical contact with a important primary case [49,18]. Since none of the 23 family contacts who had been in the same room as the primary patient contracted the virus in the first instance, the investigators came to the conclusion that the virus is not readily transmitted by air. In the second trial, researchers found that the greatest risk was from direct contact with the bodily fluids of sick patients; however, sharing a hut or a mat posed an independent risk, suggesting that other routes of transmission were involved [3,46]. According to the third study's authors, although they did not find any evidence of "small-particle aerosol transmission," they estimated that the maximum risk of this form of transmission was only 4% based on their study's confidence bounds [58,13,53,54].

f. Out of 316 cases linked to an EBOV outbreak, investigators found 55 individuals to have no known source of exposure during the preliminary evaluation of a fourth report addressing the disease. Of the 23 people who were exposed to a probable EVD case, 19 had visited the primary case prior to becoming ill; 14 had merely touched the case (with no apparent body fluid exposure); and 5 had neither physical contact nor body fluid exposure [6]. They also obtained additional information from surrogates for 40 deceased patients and from four survivors [6] According to Takada and Kawaoka [53], the authors conjectured that the route of transmission for the five individuals who did not record personal touch could have been explained by "large droplets, aerosolized particles, or fomites". A few other perspectives are relevant to understanding the spread of the Ebola virus from person to person. First off, blood infection levels rise significantly throughout the course of the illness; this likely affects the amount of viral shedding, which in turn affects a patient's long-term desirability [33,22]. Second, given the presence of infection in various bodily liquids, patients in the latter stages of their illness who are generally irresistible and who are experiencing severe the runs, retching, dying, or hacking may be obligated to shed irresistible infection with spray particles of different sizes. In the third place, examiners have increased the likelihood of "superspreading occasions." In one study, it was evident that a single SUDV disease infected ten family members; further details on this case are not available. According to an EBOV epidemic investigation, exposures most likely occurred during primary-case burials. One instance was clearly the source of 38 secondary cases, while another case was the source of 21 secondary cases. Both cases had gastrointestinal haemorrhaging. The burial of a patient who passed away in May 2014 has been linked to more than 300 cases in Sierra Leone. Fourth, it appears that 10 or fewer viral particles are necessary to cause infection in humans, suggesting that the infectious dosage for Ebola viruses is extremely low [50,19]. Health care workers may find this to be especially important while donning contaminated PPE, since improper donning could expose them to minuscule viral particles [15,16,55,56].

g. The propagation of the present West African outbreak is poorly understood. One startling finding is the large proportion of infected medical staff. The WHO claims that this incident was caused by improper use of personal protective equipment (PPE), a lack of medical personnel, poor training in specialised treatment, and an incorrect diagnosis of EVD. The precise mode of infection for a nurse in Madrid, Spain who treated a returning missionary, and for two American nurses who contracted the infection while tending to a patient in Dallas, Texas, is still unknown [34,39]. The Dallas patient underwent aerosol-generating procedures (including intubation) before he passed away, but it's unclear if such treatments helped spread to the medical personnel [24,26].



Picture 1. Model showing Mode of infection

Table 1. Symptoms and signs of Ebola Virus Disease in the 1976 Sudan Epidemic (N = 183)

Symptoms and sign	Frequency (%)
1.Bleeding	71
a. Melena	59
b. Bleeding (survivors)	48
c. Bleeding (nonsurvivors)	91
2.Fever	100
3.Headache	100
3.Chest pain	83
4.Diarrhea	81
5.Emesis	59
6.Sore throat	63
7.Maculopapular rash	52
8.Cough	49

- This data shows a high prevalence of fever, headache, and various gastrointestinal symptoms (melena, diarrhea, vomiting) in the cases.
- Chest pain and sore throat are also common, indicating potential respiratory and systemic involvement.
- Bleeding, especially in the form of melena, appears to be associated with the cases.
- The distinction between survivors and non-survivors in terms of bleeding indicates a potential severity factor.
- The presence of a maculopapular rash is notable and could indicate a viral origin.
- Cough, while present in almost half the cases, is less prevalent compared to other symptoms.

3. EBOLA VIRUS RESPIRATORY TRANSMISSION: A HYPOTHESIS

Given that individuals exposed to infectious aerosols are also more likely to be in close proximity to and in direct contact with an infected case, it has proven challenging to prove or rule out that at least some degree of Ebola virus transmission currently occurs via infectious aerosols generated from the gastrointestinal tract, the respiratory tract, or medical procedures. Thus far, scientists have not discovered any indications of Ebola virus respiratory transmission (by large droplets or small-particle aerosols) in humans [25,45]. This could be due to the fact that, in light of the small number of studies that have thoroughly examined transmission patterns, such transmission either does not occur or has not been seen. It's important to take into account whether primary pulmonary infections and Ebola virus respiratory transmission could occur in the future, even in the absence of epidemiological evidence to support this theory. A significant body of research suggests that this kind of transmission could occur even in the absence of significant genetic changes or rapid development in Ebola viruses (however viral evolution over time may enhance

the likelihood). First off, viral particles have been discovered in the pulmonary alveoli of human corpses, suggesting that infectious aerosols may be released from the respiratory system. Ebola viruses may also be identified from saliva [25,45]. Second, macrophages and epithelial cells are among the respiratory cell types that can be infected by Ebola viruses. Third, although coughing is known to release aerosols and can be a sign of EVD, the frequency of cough reports in case series ranges, ranging from "rare" to 49%. Fourth, research on animals demonstrates that aerosols can transmit EBOV and that pneumonitis-related respiratory illnesses can develop from this mode of inoculation. Fifth, the RESTV experience suggests that animals and even humans can contract that species' respiratory disease [15]. The Ebola virus is in the respirable range, which is between 800 and 1,000 nm. Lastly, people can make and emit aerosols with a range of particle sizes, including small particles that can penetrate the lower respiratory tract and infect susceptible cells. Aerosolized Ebola virus may cause primary pulmonary infections in uninfected individuals (animal studies have demonstrated this), which will lead to active viral shedding from the respiratory tract and possibly initiate a human respiratory transmission cycle akin to pneumonic plague outbreaks. Scholars examining a nosocomial outbreak of Lassa fever virus, an additional African hemorrhagic fever virus that is usually transmitted by contact with rodents, especially through rodent urine, postulated that respiratory transmission might have occurred. Similarly, researchers studying a nosocomial cluster of the vector-borne disease Crimean-Congo hemorrhagic fever, which affects Eastern Europe and Africa, found that aerosol-producing medical procedures were probably the cause of aerosol transmission. Aerosols are the most likely means of dispersal for hemorrhagic fever viruses, which have long alarmed specialists as possible bioterrorism agents. Filoviruses, such as the Marburg and Ebola viruses, are especially dangerous. This apprehension arises from lack of knowledge regarding the possibility of these viruses spreading through aerosol [44,52]. To highlight the threat, the Soviet Union turned a variant of the Marburg virus into a weapon. Prominent public health organisations have stated that it is unlikely that Ebola viruses would spread by air in the future due to specific genotypic changes in the virus. This refers to transmission by small-particle aerosols moving over time and distance [10, 20, 12]. We both agree that this is an unlikely (but not impossible) scenario; however, due to changes in the virus's phenotypic, there is still a chance that droplets of different sizes from cases in close proximity to uninfected individuals could spread aerosolly and potentially cause respiratory infections if primary pulmonary infections occur. The West African Ebola epidemic surprised even the most astute infectious disease experts in the international public health community, so we shouldn't assume that Ebola viruses won't surprise us in the future [14].

4. THE FILOVIRUS LIFE CYCLE

Electron microscopy concentrates on show that the Ebola infection has a filamentous appearance regularly 800 nm long and 80 nm in width. Each popular molecule or virion comprises of a nucleocapsid comprising of the negative ssRNA genome encompassed by the VP35, the polymerase cofactor, and nucleoprotein NP, the infection explicit record activator VP30, and the viral RNA polymerase L proteins [5]. This nucleocapsid is embodied by an external viral envelope starting from the host cell film with trademark 10 nm long popular glycoprotein (GP) spikes. The lattice between the external viral envelope and the nucleocapsid is involved by the VP40 and VP24 viral proteins [5].

The viral RNA polymerase attaches at the leader end of each gene to commence successive transcription. During this step, the L protein caps and polyadenylates newly transcribed mRNAs. Notably, the major mRNA generated from the GP gene produces sGP, a tiny, non-structural, soluble protein secreted into blood by infected host cells. RNA editing produces a fully functioning glycoprotein, which is produced on the cell surface as GP spikes. These GP spikes aid in virion anchoring and membrane fusion, and are a critical element in Ebola virus pathogenicity (*UN News Service, 2007; WHO, 2012*).

The matrix protein VP40 is required for the virion's structural stability. It is also linked to endocytosis and virus budding, and it can escape from cells even in the absence of other viral proteins. The second matrix protein, VP24, inhibits host cell interferon production. Furthermore, VP24, together with VP35 and NP proteins, is required for the effective assembly of a functioning nucleocapsid (Gatherer, et al.2014). The remaining proteins, NP, VP35, VP30, and L, are structural components of the nucleocapsid. Additionally, these proteins catalyze genome transcription and replication [37].

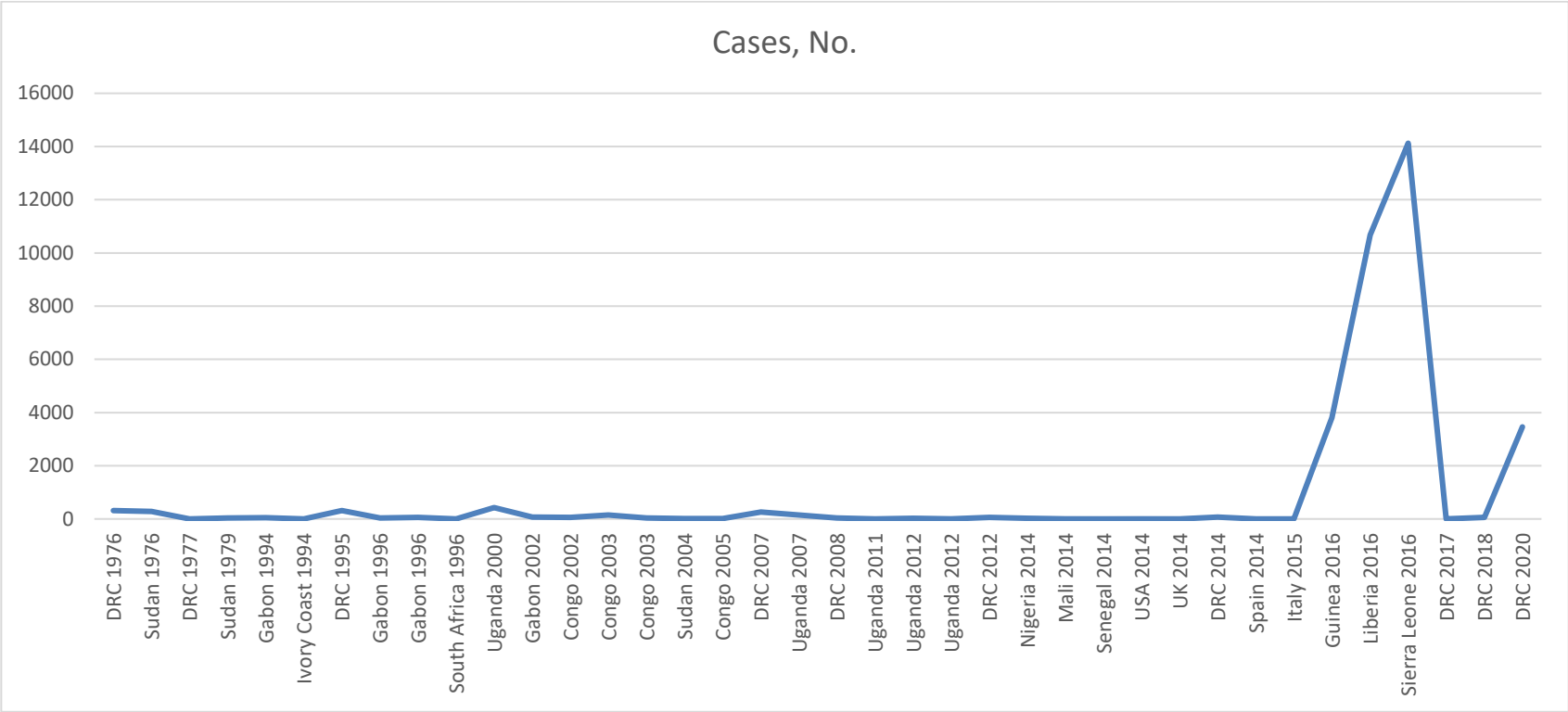


Fig. 1. Number of cases of ebolaviruses infection till 2020

4.1 Invasion of the Host Immune System

The virus's first targets are the host immune system's monocytes and macrophages. Dendritic cells, liver cells, and endothelial cells are other targets. The Ebola virus uses a variety of ways to disrupt or even totally bypass the host immune system. The structural proteins of the virus are involved in the majority of these host immune system attack activities [1]. The antibody-dependent enhancement (ADE) mechanism is one such mechanism in which host antibodies (Abs) promote or increase the virus's adhesion to host cells, enhancing infection in these cells. The Abs bind to antibody receptors at their Fc sites, whereas the virus binds to the antigen-binding site at the Abs6's free end. In vitro investigations on Ebola revealed that the virus stimulates the complement system's classical route. The Ebolavirus first connects to its receptor on the host cell surface. Following that, Abs bind to the virus's glycoprotein (GP) spikes, and the complement system's C1q component binds to the Ab-GP complex. The C1q component improves the Ab-GP complex's ability to attach to C1q ligands on the host cell surface, boosting the virus's contact with its receptor on the host cell surface. In this manner, the virus's GP spikes employ the host immune system (Abs and complement components) to strengthen their attachment to target cells [50].

Aside from ADE, the virus's protein VP35 inhibits the immune system's interferon (IFN) pathways, which are comprised of numerous cytokines that exert anti-viral reactions. VP35 inhibits IFN response by competing with proteins such as the retinoic acid-inducible gene 1 (RIG1) protein for IFN pathway activation. VP24, like VP35, inhibits IFN pathway activation. VP24 inhibits transcription factors such as STAT1 that regulate the transcription of immune system genes [41]. As previously stated, the GP gene's major mRNA transcript encodes the soluble sGP, which is thought to have an anti-inflammatory role during infection, aiding the virus's escape from host immune system reaction. Furthermore, because sGP and GP share many epitopes, it could potentially sequester or absorb host Abs to prevent their downstream function. As a result, the viral proteins impair several immune system components in order to adhere to the host cell for subsequent invasion [7,42].

Primary transcription starts as soon as ribonucleoprotein (RNP) complexes relax in the cytoplasm. Host ribosomes transform the produced viral mRNAs into viral proteins. Viral proteins that are newly synthesised aid in both secondary transcription and inclusion body-mediated genome replication. RNP complexes, which are replicated genomes, condense and go to budding sites [4]. Numerous pharmacological families, including nucleoside analogues, nucleotide synthesis inhibitors, and polymerase inhibitors, have the ability to hinder the production of viral RNA. In the course of genome replication, NP encapsidates nascent viral genomic RNAs (vRNAs) and antigenomic RNAs (cRNAs). This process is susceptible to disruption by inhibitors of RNP complex formation. Lastly, small interfering RNAs (siRNAs) can target mRNAs as well as vRNAs or cRNAs, and phosphorodiamidate morpholino oligonucleotides (PMOs) can prevent the translation of viral mRNAs. It is noteworthy that while the majority of strategies targeting the transcription and translation of viral RNA are still in the experimental and developmental stages, the siRNA TKM-Ebola (highlighted in bold) and its nucleoside analogues favipiravir and remdesivir are either in clinical trials or have been used in humans in experiments [8].

4.2 Invasion of the Virus into the Host Cell

The precise mechanism by which the Ebola virus penetrates host cells is yet unknown. Endocytosis is a general method used by most enveloped viruses, including the Ebola virus, to infect host cells. According to research, the virus enters the cell via a lipid-dependent, non-clathrin, and dynamin-independent endocytic pathway. The Ebola virus's most likely mechanism is macropinocytosis. This mechanism involves actin polymerization producing outward extensions of the plasma membrane that can fold back on themselves [30]. The distal loop ends of these membrane ruffles or extensions can unite to form a macriopinosome [39]. Actin and its related polymerising proteins so play an important role in viral entrance. The precise method by which the virus promotes macropinocytosis is unknown. It is thought that interactions between GP and host cell surface receptors might cause macropinocytosis, which can lead to viral entry [48].

4.3 Virus Reproduction

With the engagement of the polymerase complex once inside the host cell, the virus begins transcription at the leader end of the genome5.

VP30 is a transcription activation factor that promotes viral genome transcription, whereas VP24 inhibits this process [2]. The precise mechanism of VP24-dependent transcription termination is unknown, but it appears to be critical for transitioning the virus from its transcriptional or replication active form to one oriented for virion assembly and escape from the host cell [51]

4.4 Virus Budding and Expulsion from the Host Cell

The cell loses its link to other cells as well as its attachment to its substrate after duplication. Meanwhile, newly produced genomes are bundled into new buds or virions and egress from the host cell surface via the matrix protein VP40. VP40 interacts with the ubiquitin ligase Nedd4, a component of the human ubiquitination enzyme pathway that connects multiple copies of ubiquitin molecules to VP40. The COPII transport mechanism transports VP40 to the host cell plasma membrane. Once inside the plasma membrane, the virus passes via lipid rafts, where virions are assembled and budded before exiting the host cell [57,58]. Although the virus's basic components are recognized, the precise methods by which it causes disease in people are not fully understood [26]. This presents a significant obstacle for treatment, and as of now, prevention is the best course of action to avert an Ebola outbreak. In the following essay, we will discuss disease prevention, treatment, and prognosis, as well as recognized strategies to limit further spread and recurrence [38].

5. SYMPTOMS

Ebola symptoms can be severe and include fever, lethargy, muscle pain, headache, and sore throat. Then there's vomiting, diarrhoea, a rash, and internal and external bleeding.

The interval between being infected and experiencing symptoms ranges between 2 and 21 days. Once a person has Ebola, they can only spread the disease. People can spread Ebola for as long as the virus is present in their bodies, even after they have died [35,36].

Some people may have symptoms for two years or longer after recovering from Ebola. Among these signs are:

A. tiredness headache muscle and joint pain
B. eye pain and vision issues
C. gaining weight
D. stomach ache and loss of appetite
E. Hair loss and skin issues
F. Sleeping difficulties Memory loss
G. hearing impairment
H. Anxiety and depression.

People should seek medical attention if they exhibit any of the following symptoms: symptoms and have been in an Ebola-infected area or had contact with someone who may have had Ebola (*WHO,Outbreak, 2014; CDC, 2014*).

6. PREVENTIVE MEASURES

The main goals of Ebola virus disease (EVD) prevention strategies are to lower the risk of infection and shield people from it. Avoiding contact with the blood or bodily fluids of ill people, such as their urine, feces, saliva, sweat, vomit, breast milk, amniotic fluid, semen, and vaginal secretions, is one of the most crucial preventive precautions. Second After an individual has recovered from Ebola, do not touch his semen until laboratory testing has verified that the virus has left his semen. Avoiding touch with items that may have come into contact with bodily fluids or blood from an infected person is another way to prevent infection (such as bedding, clothes, needles, and medical equipment). Avoid taking part in funeral or burial customs that require managing the body of an individual who has been diagnosed or proven to have had Ebola. Steer clear of bats, woodland antelopes, nonhuman primates (such chimpanzees and monkeys), and any raw meat that has been prepared from these or unidentified animals [23,31,32].

7. POSSIBLE TREATMENTS

Treatment includes intravenous or oral fluids and medications administered by the hospital. It is not safe to care for Ebola patients at home since the patient may infect others. They will not receive the same degree of care at home that they would receive from experts. The Zaire strain of Ebola, which is predominantly found in Guinea and the Democratic Republic of the Congo, has an effective vaccine. Antibodies are used to treat it. These antibody medications are administered intravenously and improve the chances of survival [35,36].

Other kinds of Ebola are being researched for vaccines and therapies. Supportive therapy for all kinds of Ebola saves lives and include the following:

A. Fluids administered orally or intravenously,
B. blood transfusions,
C. medicines for other infections the person may have, such as malaria,
D. medicines for pain, nausea, vomiting, and diarrhoea.

WHO guidelines explain the optimal supportive treatment Ebola patients should receive, from administering necessary tests to managing pain, nutrition, and co-infections (such as malaria), among other techniques that place the patient on the best route to recovery. During the 2018-2020 Ebola outbreak in the Democratic Republic of the Congo, the first-ever multi-drug randomized control trial was carried out to assess the efficacy and safety of medications used in Ebola therapy. The World Health Organization provides ongoing advice on preferred treatments and practices. Inmazeb (atoltivimab, maftivimab, and odesivimab-ebgn) is a monoclonal antibody combination used to treat Zaire ebolavirus infection in adults and children, including neonates born to women who tested positive for the virus [35,36].

Not only are there no known treatments for Ebola virus dis- ease, but very little is known about the mechanisms by which patients develop shock and DIC. The epidemics that have occurred during the past 4 decades have been in low-income countries with limited health care resources. Most patients do not have simple laboratory tests, such as a complete blood cell count, and more costly tests, such as a coagulation panel or cardiac output measurement, are rare [35, 36]. In addition, tests must be performed in a biosafety level-4 laboratory. What we know has been learned from past epidemics and studies in nonhuman primates. Treatment is support- ive. Dehydration is very common, so rehydration should be attempted with an oral balanced electrolyte solution. If the patient cannot maintain fluid balance because of gastroin- testinal illness, IV crystalloid fluids should be administered. Hypoxia is reported to occur with Ebola virus disease, but during the current epidemic, it is not as common as one might expect communication, Robert Fowler, MDCM, (World Health Organization, June, 2014) unless the patient develops multisystem organ dysfunction [11].

Ebola virus illness therapy has been complicated because the virus encodes two glycoproteins: the first is a membrane glycoprotein present in the viral membrane that promotes viral attachment and entry into host cells, and the second is a secreted, non-structural glycoprotein. The latter elicits host non-neutralizing antibodies that cross react with glycoprotein, potentially preventing effective viral neutralization. Many people have survived the Ebola virus. Their convalescent serum has been anecdotally successful in treating patients who were acutely ill with Ebola virus disease. Convalescent serum therapy is more likely to be employed in high-income nations where Ebola virus patients have been treated. Another promising therapy is with monoclonal antibodies [35,36]. These have been shown to reverse infection in non-human primates and to cure infected animals after symptoms and circulating Ebola virus are present. A combination of monoclonal antibodies (ZMapp), derived from 2 previous experiments, rescued 100% of rhesus macaques when given 5 days post challenge, even in the presence of advanced dis- ease. As of November 2014, ZMapp has been administered to 7 patients with the disease on a case-by-case basis under a compassionate use protocol, and additional studies of it and several other treatments and vaccines are under way.

Other therapies being researched to cure the Ebola virus illness include the virus's inhibition of membrane fusion (T-20 Enfuvirtide), transcription/replication inhibitors, nucleoside analogs, antisense oligonucleotides, small-interfering RNAs, maturation inhibitors such as furin inhibitors and budding inhibitors, and cytokine storm modulation by an array of cytokine inhibitors [11].

8. DIAGNOSIS

Clinically, distinguishing Ebola virus disease from other infectious diseases such as malaria, typhoid fever, and meningitis can be difficult. Many pregnancy symptoms and Ebola illness symptoms are very similar. Pregnant women should preferably be tested quickly if Ebola is suspected due to the risks to the pregnancy and to themselves [35]. The following diagnostic procedures are used to confirm that symptoms are caused by Ebola virus infection:

A. ELISA (enzyme-linked immunosorbent test) for antibody capture,
B. Tests for antigen capture,
C. Neutralization test for serum,
D. Reverse transcriptase polymerase chain reaction (RT-PCR) assay,
E. Electron microscopy

F. Virus isolation by cell culture
G. Epidemiological information
H. Post-mortem examination
I. Rapid Diagnostic tests(RDTs)

9. CONTROL AND PREVENTION

People can protect themselves from Ebola by doing the following:

- Surveillance & early detection
- Isolation & treatment
- Contact tracing
- Community Engagement
- Safe Burial Practices
- Infection prevention & control
- Vaccination
- Travel restrictions & cross border collaboration
- Research & development

The Ervebo vaccine has been shown to be effective in protecting people from the species Zaire ebolavirus and is recommended by the Strategic Advisory Group of Experts on Immunization as part of a broader set of Ebola outbreak response tools. Case management, surveillance, and contact tracing, a strong laboratory service, safe burials, and social mobilization are all important components of effective outbreak control. The involvement of the community is critical to the successful containment of outbreaks. Raising public awareness of Ebola risk factors and protective actions (including vaccination) that individuals can take is an effective method to limit human transmission. Several variables should be emphasized in risk reduction messaging:

- lowering the possibility of wildlife-to-human transfer
- lowering the danger of human-to-human transmission outbreak containment methods, such as safe and dignified interment of the dead
- lowering the possibility of sexual transmission
- lowering the risk of transmission from pregnancy-related fluids and tissue.

When providing care for patients infected with the Ebola virus, healthcare professionals should take extreme precautions to prevent infection by avoiding contact with the patient's bodily fluids and blood, as well as contaminated surfaces and items like bedding and clothing. Regardless of the patient's apparent ailment, healthcare professionals should always take the standard measures when caring for them. These include things like proper hand hygiene, respiratory hygiene, wearing personal protection equipment (PPE) to avoid splashes or other contact with infectious materials, safe injection techniques, and safe burial procedures. Workers in laboratories are also at risk [35]. Human and animal samples collected for the examination of Ebola infection should be handled by competent personnel and processed in well-equipped laboratories [35,36].

10. DISCUSSION

EVD is an extremely contagious disease that has spread widely, especially in Africa. Although the Ebola virus's precise origin is still unknown, it is believed to be animal-borne because infected animals can directly infect other species. 55 persons lost their lives in the most recent outbreak, which occurred in November 2020 and resulted in 130 EVD cases from 13 health zones—119 of which were confirmed cases and 11 of which were suspected cases. For EVD, there are currently very few drugs and treatment choices [27]. The most often used drugs, in addition to supportive care, are ZMapp, Inmazeb, and Ebanga. The review found that the posterior probability with Inmazeb and Ebanga performed better than the current standard of care by itself. Treatment with inmazeb, however, lowers mortality by 17%. The most common side effects reported with Ebanga are fever, tachycardia, diarrhea, vomiting, hypotension, tachypnea, and chills when compared to ZMapp and Inmazeb. Overall, Ebanga is a therapy option for patients with cardiovascular problems, however Inmazeb is the medication of choice for treating EVD when compared to ZMapp or other options. Over the years, the DRC has had multiple Ebola outbreaks. In North Kivu Province, there was a notable outbreak between 2018 and 2020. The virus moved to heavily inhabited regions, making containment measures difficult. Following coordinated efforts by regional health authorities, the World Health Organization (WHO), and other partners, the outbreak was deemed to be under control in June 2020. According to a WHO announcement, the current Ebola epidemic in Uganda was formally declared on September 20, 2022, following the discovery of a case of the Sudan virus disease in the central Ugandan region of Mubende [40].

The current Ebola virus disease pandemic has lasted longer, impacted more people, killed more people, and caused more social chaos than all previous Ebola virus disease outbreaks

combined. To put the current pandemic in context, viral hemorrhagic fevers impact over 100 million people and kill 60,000 people each year.The Ebola virus disease has created so much upheaval because so little is known about it, owing to its high fatality rate and clinical manifestations. However, the current pandemic has occurred due to a lack of information (avoidance of bats and infected nonhuman primates), insufficient public health practices (protocols for isolation and implementation of quarantines and unsafe burial practices), ease of travel, insufficient infection control (the nurse in Spain who contracted Ebola virus disease was reported in the media to have "touched" her face with her gloved hand), and insufficient infection control (the nurse in Spain who contracted Ebola virus disease was reported. Based on prior experience, it seems likely that nothing will have changed in a year [9]. However, based on what we have learned, we as anaesthesiologists should take the necessary steps now to better prepare and educate ourselves in order to protect our families from the consequences of such events and provide effective treatment for those to whom we will provide care during this and future.

11. CONCLUSION

Numerous epidemics have been caused by the highly contagious Ebola virus disease (EVD), the majrity of which have struck Africa. Despite much investigation, the Ebola virus's precise origin remains unknown. The majority of scientists, however, emphasize zoonotic transmission, in which the virus is assumed to begin in animals before moving on to humans. This conclusion about the potential for a spillover effect from wildlife reservoirs to human populations is based on the data, since afflicted animals may serve as direct conduits for the spread of disease to other species. Our ability to prevent and manage epidemics in the future will be strengthened by our continued scientific efforts to identify the reservoir host and understand the mechanisms of interspecies transmission. The scientific community is attempting to not only understand the ecological context of the Ebola virus's natural habitat but also to develop more focused and efficient methods of disease prevention and control on a worldwide basis.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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