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Review on Ebola Virus

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Abstract: Ebola virus (EBOV) is one of the genera of filoviridae. It has five species *Coted'Ivoire* EBOV (ICEBOV), (ICEBOV), *Reston* EBOV (REBOV), *Bundibugyo* EBOV (BEBOV), *Sudan* EBOV (SEBOV) and *Zaire* EBOV (ZEBOV). All of the known human pathogenic filoviruses are endemic only in sub-Saharan Africa. The Zaire species of Ebola virus is the causative agent of the 2014-2015 epidemic in West Africa. *Reston virus* (RESTV), is not thought to cause disease in humans. Ebola virus is classified as a filovirus (*filo*, Latin for worm). The overall shape of the virions visualization by electron microscopy varies considerably; U-, shepherd's crook-, 9- or eye bolt-shapes, or other or circular/ coiled appearances. Each virion contains one molecule of linear, single-stranded, negative-sense RNA genome. Ebola viruses can survive in liquid or dried material for a number of days. Fruit bats are considered as a reservoir hosts and humans are dead-end hosts. The virus transmits from wild animal to persons and person to person by different ways of contaminations. After entering in to the body it infects different cells most probably macrophages and dendritic cells. The onset of EVD is sudden and early symptoms include flu-like illness, fever, muscle pain (myalgia), fatigue (weakness), headache and sore throat. The virus is diagnosed by non- specific and specific laboratory tests. Treatment is primarily supportive in nature. To control virus, the infected person should be in barrier-isolation from other people and stop eating eating bush meat. Ebola vaccine candidates had been developed in the decade prior to 2014.

Key words: Filoviridae Ebola Outbreak Reservoirs transmission Hemorrhagic Fever

INTRODUCTION

Ebolavirus (EBOV) is one of the genera of *Filoviridae*. Filoviruses are enveloped, non-segmented, single-strand negative sense RNA molecules taxonomically assigned within the order *Mononegavirales* and family *Filoviridae.* Five species of EBOV have been identified: *Coted'Ivoire* EBOV (ICEBOV), *Reston* EBOV (REBOV), *Bundibugyo* EBOV (BEBOV), *Sudan* EBOV (SEBOV) and *Zaire* EBOV (ZEBOV). The Zaire species of Ebola virus is the causative agent of the 2014-2015 which is epidemic in West Africa. The natural reservoir of Ebola virus is believed to be bats, particularly fruit bats and it is primarily transmitted between humans and from animals to humans through body fluids [1].

Ebola virus was first identified as a possible new strain of Marburg virus in 1976. At the same time, a third team introduced the name Ebola virus, derived from the Ebola River- a river that was at first thought to be in close proximity to the area in Democratic Republic of Congo, previously called Zaire, where the 1976. Zaire Ebola virus outbreak occurred in the summer season. The International Committee on Taxonomy of Viruses (ICTV) identifies Ebola virus as Zaire Ebolavirus, which is included into the genus Ebolavirus, family Filoviridae and Order Mononegavirales [2].

In 2000, the virus name was changed to *Zaire* Ebola virus and in 2002 to species *Zaire ebolavirus.* However, most scientific articles continued to refer to Ebola virus or used the terms Ebola virus and *Zaire ebolavirus* in parallel. Consequently, in 2010, a group of researchers recommended that the name Ebola virus be adopted for a sub classification within the species *Zaire ebolavirus*, with the corresponding abbreviation EBOV. Previous abbreviations for the virus were EBOV-Z (for Ebola virus Zaire) and ZEBOV (for *Zaire ebolavirus*). In 2011, the ICTV explicitly rejected a proposal (2010) to recognize this name, as ICTV does not designate names for subtypes, variants, strains, or other subspecies level groupings [3].

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The viral genomic RNA is always associated with multiple copies of a viral nucleoprotein (NP), forming a helical NP-RNA complex. The single stranded RNA genome approximately 19, 000 nucleotide long. Non-retroviral RNA viruses lack the coding for reverse transcriptase and the integration machinery needed for successful transfer to DNA genomes [4].

Ebola virus disease (EVD; also Ebola hemorrhagic fever, or EHF) is a disease of humans and other primates caused by Ebola viruses. Signs and symptoms typically start between two days and three weeks after contracting the virus with fever, sore throat, muscle pain and headaches. Then, vomiting, diarrhea and rash usually follow, along with decreased function of the liver and kidneys. At this time some people begin to bleed both internally and externally [5]. EHF begins with the abrupt onset of fever and malaise, followed over several days by a fall in blood pressure leading to profound shock and the development of severe coagulation defects. In some patients, antigen-specific immune responses develop in time to restrict viral replication and bring about survival, otherwise death occurs one to two weeks after the onset of symptoms [6].

The virus spreads by direct contact with body fluids, such as blood, of an infected human or other animals. This may also occur through contact with an item recently contaminated with bodily fluids. Spread of the disease through the air between primates, including humans, has not been documented in either laboratory or natural conditions. Fruit bats are believed to be the normal carrier in nature, able to spread the virus without being affected by it [1].

Prevention includes limiting the spread of disease from infected animals to humans. This may be done by handling potentially infected bush meat only while wearing protective clothing and by thoroughly cooking it before eating. It also includes wearing proper protective clothing and washing hands when around a person with the disease. The medical services include rapid detection of cases of disease, contact tracing of those who have come in to contact with infected individuals, quick access to laboratory services, proper healthcare for those who are infected and proper disposal of the dead through cremation or burial and economic crisis in different aspect [5].

Therefore the objective of the paper is:

- To review the nature, epidemiology, zoonotic importance, diagnosis, treatment, prevention, control mechanism and global economic crisis of Ebola virus.

Overview of Ebola Virus

Etiology: The Filoviridae (from the Latin *filo* meaning thread or worm) because its structure seen under the electron microscope resembles that of a thread or worm referring to their filamentous shape [7]. It is enveloped negative-sense single-stranded RNA viruses [8]. The genus *ebolavirus* is divided into five species (*Zaire, Sudan, Ivory Coast, Bundibugyo and Reston).* The previous four species cause disease in human's pathogenic Ebola viruses are endemic only in sub-Saharan. Ebola virus (EBOV, formerly *Zaire ebolavirus*), since it was first recognized in 1976, has caused multiple large outbreaks in Central Africa, with mortality rates ranging from 55 to 88 percent. This causative agent is epidemic in West African. The species *Zaire ebolavirus*, is most dangerous of the known EVD causing viruses and is responsible for the largest number of outbreaks [9].

Sudan virus (SUDV) has been associated with a case-fatality rate of approximately 50 percent in four epidemics: two in Sudan in the 1970s, one in Uganda in 2000 and another in Sudan in 2004[6]. *Taï Forest* virus (TAFV) (*Côte d'Ivoire Ebola virus*) has only been identified as the cause of illness in one person and that individual survived. The exposure occurred when an ethologist performed a necropsy on a chimpanzee found dead in the *Tai Forest*, where marked reductions in the great ape population had been observed [10]. *Bundibugyo* virus (BDBV) emerged in Uganda in 2007, causing an outbreak of Ebola virus disease with a lower case-fatality rate (approximately 30 percent) than is typical for the *Zaire* and *Sudan* viruses. Sequencing has shown that the agent is most closely related to the *Ivory Coast* species [11].

The fifth Ebola species, *Reston* virus (RESTV), differs markedly from the others, because it is apparently maintained in an animal reservoir in the Philippines and has not been found in Africa. The Ebola Reston virus was discovered when it caused an outbreak of lethal infection in macaques imported into the United States in 1989. Nothing further was heard of the Reston virus until 2008, when the investigation of an outbreak of disease in pigs in the Philippines unexpectedly revealed that some of the sick animals were infected both by an arterivirus (porcine reproductive and respiratory disease virus) and by Ebola *Reston* virus. It is not thought to cause disease in humans, but has caused disease in other primates [12].

Viral Morphology: The overall shape of the virions after purification and visualization (e.g., by ultracentrifugation and electron microscopy, respectively) varies considerably; simple cylinders are far less prevalent than

structures showing reversed direction, branches and loops (e.g., U-, shepherd's crook-, 9- or eye bolt-shapes, or other or circular/coiled appearances), the origin of which may be in the laboratory techniques applied. The characteristic threadlike structure is, however, a more general morphologic characteristic of filoviruses (alongside their GP-decorated viral envelope, RNA nucleocapsid, etc.) [13].

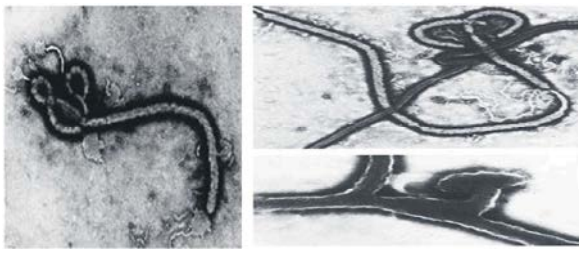

Fig. 1: Morphology of Ebola virus under Electron photomicrograph of a specimen from cell (tissue culture) passage. Source: [14].

EBOV carries a negative-sense RNA genome in virions that are cylindrical/ tubular and contain viral envelope, matrix and nucleocapsid components. The overall cylinders are generally approximately 80 nm in diameter and have a virally encoded glycoprotein (GP) projecting as 7-10 nm long spikes from its lipid bilayer surface [5]. The cylinders are of variable length, typically 800 nm, but sometimes up to 1000 nm long. The outer viral envelope of the vision is derived by budding from domains of host cell membrane into which the GP spikes have been inserted during their biosynthesis. Individual GP molecules appear with spacing of about 10 nm. Viral proteins VP40 and VP24 are located between the envelope and the nucleocapsid in the matrix space [15].

Genomic Structure: The genomes of the five different Ebola viruses (BDBV, EBOV, RESTV, SUDV and TAFV) differ in sequence and the number and location of gene overlaps. Each virion contains one molecule of linear, single-stranded, negative-sense RNA, 18, 959 to 18, 961 nucleotides in length. The 3' terminus is not poly adenylated and the 5' end is not capped [4]. This viral genome codes for seven structural proteins and one non-structural protein. The gene order is 3' – leader – NP – VP35 – VP40 – GP/s GP – VP30 – VP24 – L – trailer – 5'; with the leader and trailer being non-transcribed regions, which carry important signals to control transcription, replication and packaging of the viral genomes into new virions. Sections of the NP, VP35 and the L genes from filoviruses have been identified as endogenous in the genomes of several groups of small mammals [16].

Resistance of the Virus in the Environment: Ebola viruses can survive in liquid or dried material for a number of days. However, Ebola virus can be inactivated by UV radiation, gamma irradiation, heating for 60 minutes at 60°C or boiling for five minutes. The virus is susceptible to sodium hypochlorite and disinfectants. Freezing or refrigeration will not inactivate the virus [18]. Outside the host Filoviruses capable to survive on contaminated surfaces have been reported particularly at low temperature (4°C). When dried in tissue culture media on to glass and stored at (4°C), *Zaire Ebolavirus* survive for over 50 days [19].

Epidemiology: Geographic distribution: Outbreaks of Ebola virus disease have been confined to Sub-Saharan Africa. An epidemic caused by the *Zaire* species caused several hundred cases in 1995 in Kikwit, Democratic Republic of the Congo and the *Sudan* virus infected more than 400 people in Gulu, Uganda in 2000. The 2014-2015 Ebola epidemic, caused by the *Zaire* species of virus, is not only the first to occur in West Africa (Guinea, Sierra Leone, Liberia, Mali and Nigeria), but is far larger than all previous outbreaks combined [5].

Ebola virus has also spread to non-human primates, apparently as a result of their contact with an unidentified reservoir host (possibly bats) [20]. This has contributed to a marked reduction in chimpanzee and gorilla populations in Central Africa and has also triggered some human epidemics due to handling of and/or consumption of sick or dead animals by local villagers as a source of food [21].

World Health Organization (WHO) reported a major Ebola outbreak in Guinea, a Western African nation in March 2014. The disease then rapidly spread to the neighboring countries of Liberia and Sierra Leone. It is the largest Ebola outbreak ever documented and the first recorded in the region. Never before in recorded history has a biosafety level four pathogen infected so many people so quickly, over such a broad geographical area, for so long. In 2014 Ebola virus disease spread outside Africa example; United States and Spain [22].

Reservoir Hosts: Reservoir hosts are carrier hosts but that do not show clinical sign. Perhaps the greatest mysteries regarding the filoviruses are the identity of their natural reservoir(s) and the mode of transmission to wild apes and humans [23]. Only Ebola virus sequences, not infectious virus, have been detected in samples collected from bats in Central Africa. The natural reservoir for Ebola has yet to be confirmed; however, bats are considered to be the most likely candidate species.

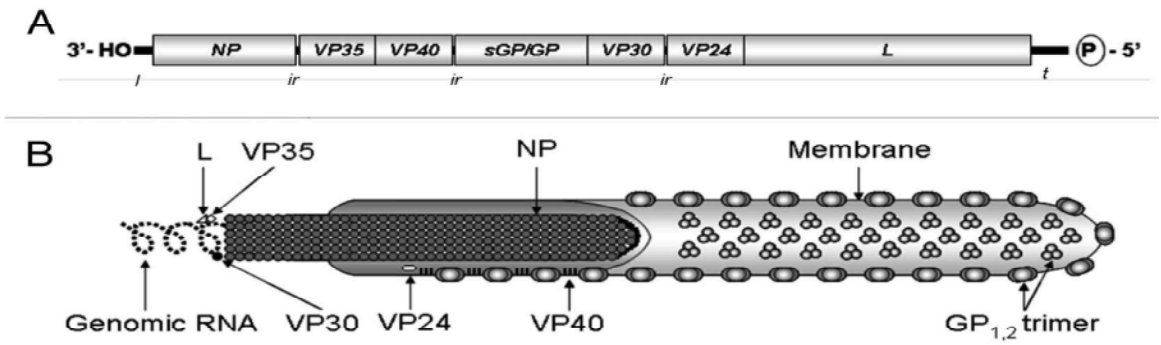

Fig. 2: Schematic representation of (A) organization of filovirus genomic RNA (ZEBOV depicted). (B) Filovirus particle. Source: [17]
Hint: l, Leader sequence; NP, nucleoprotein gene; VP, viral protein gene; GP, glycoprotein gene; L, RNA-dependent RNA polymerase gene; Ire, intergenic region

Fig. 3: Ebola virus reservoir fruit bats. Source: [25]

Three types of fruit bats (*Hypsignathusmonstrosus*, *Epomopsfranqueti*and *Myonycteristorquata*) were found to possibly carry the virus without getting sick. Plants, arthropods and birds have also been considered possible viral reservoirs [24].

Bats were known to roost in the cotton factory in which the first cases of the 1976 and 1979 outbreaks were observed and they have also been implicated in Marburg virus infections in 1975 and 1980. Of 24 plant and 19 vertebrate species experimentally inoculated with EBOV, only bats became infected. The bats displayed no clinical signs of disease, which is considered evidence that these bats are a reservoir species of EBOV. In a 2002–2003 survey of 1, 030 animals including 679 bats from Gabon and the Republic of the Congo, 13 fruit bats were found to contain EBOV RNA [26]. Antibodies against *Zaire* and *Reston* viruses have been found in fruit bats in Bangladesh, suggesting that these bats are also potential hosts of the virus and that the filoviruses are present in Asia [27].

Between 1976 and 1998, in 30, 000 mammals, birds, reptiles, amphibians and arthropods sampled from regions of EBOV outbreaks, no Ebola virus was detected apart from some genetic traces found in six rodents (belonging to the species *Mus setulosus* and *praomys*) and one shrew (*Sylvisorexollula*) collected from the Central African Republic [28]. Traces of EBOV were detected in the carcasses of Gorillas and Chimpanzees during outbreaks in 2001 and 2003, which later became the source of human infections. However, the high rates of death in these species resulting from EBOV infection make it unlikely that these species represent a natural reservoir for the virus [27].

Transmission: Epidemics of Ebola virus disease are generally thought to begin when an individual becomes infected through contact with the bush meat (from chimpanzee, bats, gorilla, etc.) or body fluids of an infected animal and humans. Once the patient becomes ill or dies, the virus then spreads to others who come into direct contact with the infected individual's blood, skin, or other body fluids. Studies in laboratory primates have found that animals can be infected with Ebola virus through droplet inoculation of virus into the mouth or eyes [29].

Contact with Infected Animals: As Ebola virus is zoonosis human infection with Ebola virus can occur through contact with wild animals (e.g., hunting, butchering and preparing meat from infected animals).

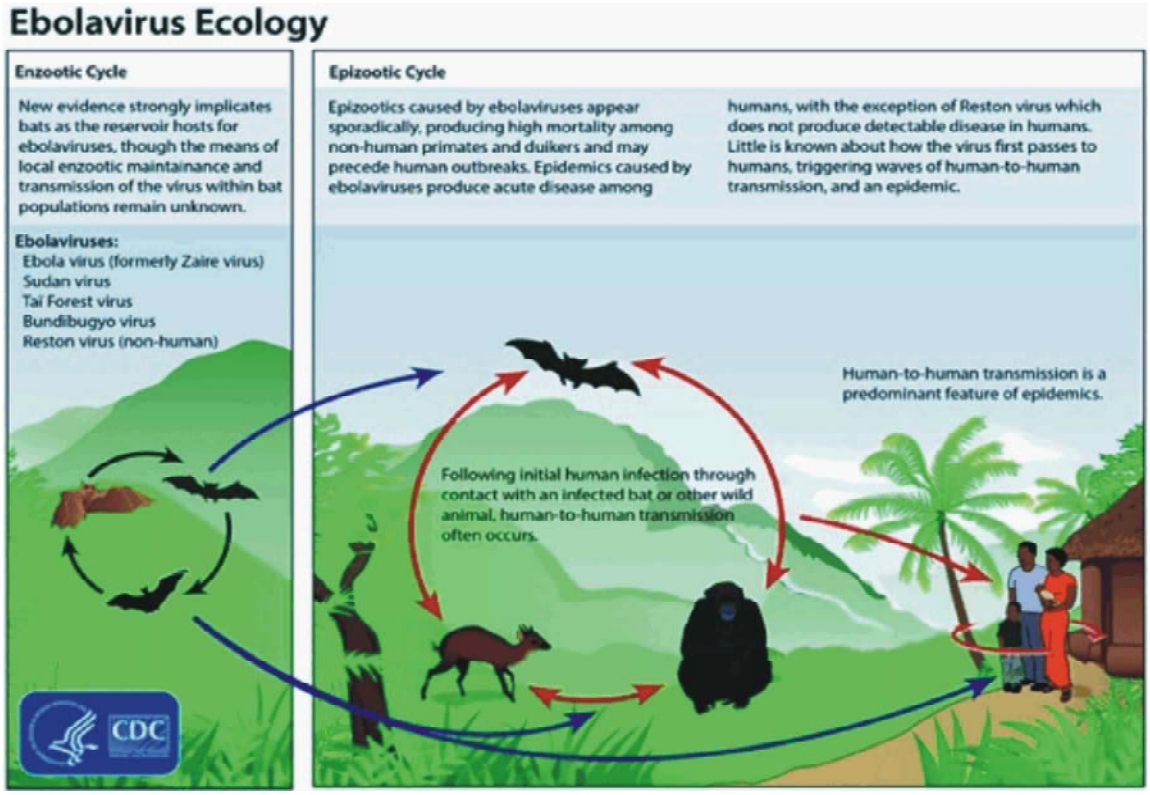

Fig. 4: Enzootic and epizootic transition cycle of Ebola virus [33]

Bush meat being prepared for cooking in Ghana. In Africa, wild animals including fruit bats are hunted for food and are referred to as bush meat [30]. In Equatorial Africa, human consumption of bush meat has been linked to animal-to-human transmission of diseases, including Ebola virus [8]. Although it is not entirely clear how Ebola virus initially spreads from animals to humans, the spread is believed to involve direct contact with an infected wild animal or fruit bat. Besides bats, other wild animals sometimes infected with EBOV include several monkey species, chimpanzees, gorillas, baboons and duikers [31].

Evidence indicates that both domestic dogs and pigs can also be infected with EBOV. Dogs do not appear to develop symptoms when they carry the virus and pigs appear to be able to transmit the virus to at least some primates. Although some dogs in an area in which a human outbreak occurred had antibodies to EBOV, it is unclear whether they played a role in spreading the disease to people [32]. Person-to -person transmission also occurs through direct contact with blood, body fluids, or skin of patients with Ebola virus disease, including those who have died from the infection [33].

Risk of transmission through different body fluids: Transmission is most likely to occur through direct contact of broken skin or unprotected mucous membranes with virus containing body fluids from a person who has developed signs and symptoms of illness. According to the World Health Organization, the most infectious body fluids are blood, feces and vomit [34].

Ebola virus can also be spread through direct contact with the skin of a patient, but the risk of developing infection from this type of exposure is lower than from exposure to body fluids. The risk of Ebola transmission also depends upon the quantity of virus in the fluid. During the early phase of illness, the amount of virus in the blood may be quite low, but levels then increase rapidly and may exceed 108 RNA copies/mL of serum in severely ill patients [35]. As an example, an epidemiologic study found that family members were at greatest risk of infection if they had physical contact with sick relatives (or their body fluids) during the later stages of illness, or helped to prepare a corpse for burial [36].

Risk of Transmission Through Contact with Contaminated Surfaces: Ebola virus may be transmitted though contact with contaminated surfaces and objects. The Centers for Disease Control and Prevention (CDC) indicates that virus on surfaces may remain infectious from hours to days. There is no high-quality data to confirm transmission through exposure to contaminated

Table 1: Levels of risk of transmission of Ebola virus according to type of contact with an infected patient

Risk level	Type of contact
Very low or no recognized risk	Casual contact with a feverish, ambulant, self-caring patient. Examples: sharing a sitting area or public transportation; receptionist tasks.
Low risk	Close face-to-face contact with a feverish and ambulant patient. Example: physical examination, measuring temperature and blood pressures.
Moderate risk	Close face-to-face contact without appropriate personal protective equipment (including eye protection) with a patient who is coughing or vomiting, has nosebleeds or who has diarrhea.
High risk	Percutaneous, needle stick or mucosal exposure to virus-contaminated blood, bodily fluids, tissues or laboratory specimens in severely ill or known positive patients

Source: [38]

surfaces [37], but it is clear that the potential risk can be greatly reduced or eliminated by proper environmental cleaning [34].

Risk of Airborne Transmission: The apparent lack of airborne transmission among humans is believed to be due to low levels of the virus in the lungs and other parts of the respiratory system of primates, insufficient to cause new infections. A number of studies examining airborne transmission broadly concluded that transmission from pigs to primates could happen without direct contact because, unlike humans and primates, pigs with EVD get very high Ebola virus concentrations in their lungs and not their blood stream [39].

Nosocomial Transmission: Transmission to healthcare workers may occur when appropriate personal protective equipment is not available or is not properly used, especially when caring for a severely ill patient who is not recognized as having Ebola virus disease [40].

Weaponization: Ebola virus is classified as a biosafety level four agent, as well as a category a bioterrorism agent by the Centers for Disease Control and Prevention. It has the potential to be weaponized for use in biological warfare [41] and was investigated by biopreparat for such use, but might be difficult to prepare as a weapon of mass destruction because the virus becomes ineffective quickly in open air [42].

Pathogenesis: Because of the difficulty of performing clinical studies under outbreak conditions, almost all data on the pathogenesis of Ebola virus disease have been obtained from laboratory experiments employing mice, guinea pigs and non-human primates. However, case reports and large-scale observational studies of patients in the 2014-2015 West African outbreak are providing urgently needed data on the pathogenesis of the disease in humans [43].

Cell Entry and Tissue Damage: Local Damage at the Primary Site of entry after entering the body through mucous membranes, breaks in the skin, viruses replicate at the primary site of entry, resulting in spread to nearby epithelial cell surfaces, or parenterally, Ebola virus infects many different cell types. Macrophages and dendritic cells are probably the first to be infected; filoviruses replicate readily within extracellular fluid [44].

Replication: Being acellular, viruses such as Ebola do not replicate through any type of cell division; rather, they use a combination of host and virally encoded enzymes, alongside host cell structures, to produce multiple copies of themselves. These then self-assemble into viral macromolecular structures in the host cell. The virus begins its attack by attaching to host receptors through the glycoprotein (GP) surface peplomer and is endocytosed into macropinosomes in the host cell. As viral protein levels rise, a switch occurs from translation to replication. Using the negative-sense genomic RNA as a template, a complementary (+) ssRNA is synthesized; this is then used as a template for the synthesis of new genomic (-) ssRNA, which is rapidly encapsulated [45].

Gastrointestinal Dysfunction: Patients with Ebola virus disease commonly suffer from vomiting and diarrhea, which can result in acute volume depletion, hypotension and shock [46]. Systemic inflammatory response Ebola virus also induces a systemic inflammatory syndrome by inducing the release of cytokines, chemokines and other proinflammatory mediators from macrophages and other cells [9]. Infected macrophages produce tumor necrosis factor (TNF)-alpha, interleukin (IL)-1beta, IL-6, macrophage chemotactic protein (MCP)-1 and nitric oxide (NO). These and other substances have also been identified in blood samples from Ebola-infected macaques and from acutely ill patients [6].

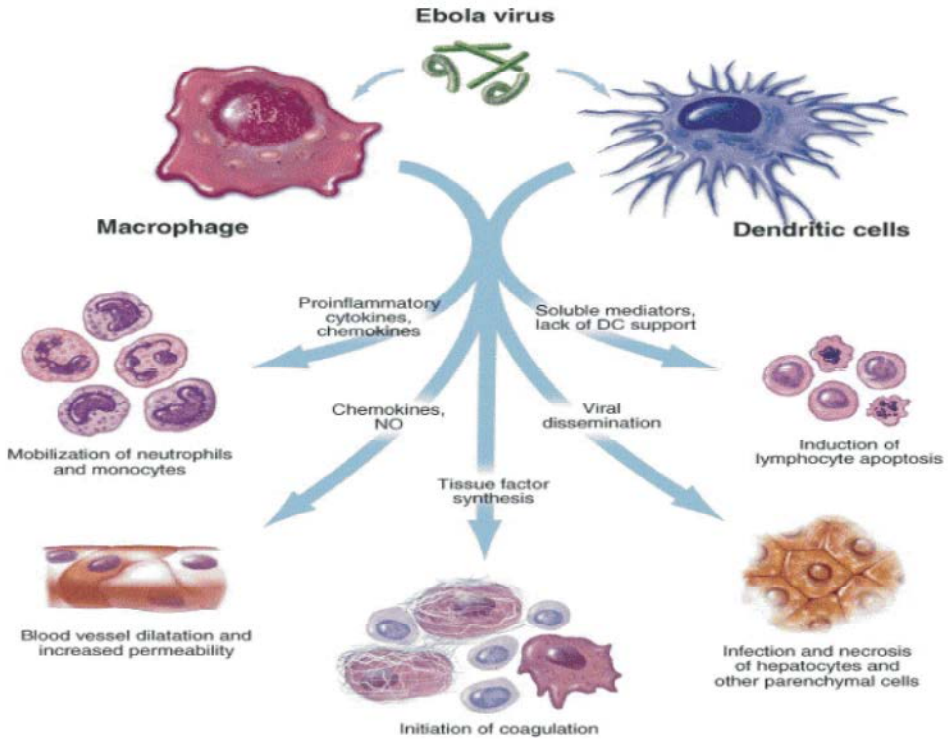

Fig. 5: ZEBOV-infected macrophages and DC play a central role in inducing the clinical features of EHF. Source: [8]

Coagulation Defects: The coagulation defects seen in Ebola virus disease appear to be induced indirectly, through the host inflammatory response. Virus infected macrophages synthesize cell-surface tissue factor (TF), triggering the extrinsic coagulation pathway; proinflammatory cytokines also induce macrophages to produce TF [47].

Impairment of Adaptive Immunity: Failure of adaptive immunity through impaired dendritic cell function and lymphocyte apoptosis helps to explain how filoviruses are able to cause a severe, frequently fatal illness [9]. Ebola virus acts both directly and indirectly to disable antigen-specific immune responses. Dendritic cells, which have primary responsibility for the initiation of adaptive immune responses, are a major site of filoviral replication. In *vitro* studies have shown that infected cells fail to undergo maturation and are unable to present antigens to naive lymphocytes, potentially explaining why patients dying from Ebola virus disease may not develop antibodies to the virus [6].

Signs and Symptoms: The length of time between exposure to the virus and the development of symptoms (incubation period) is between two to twenty-one days; However, recent estimates based on mathematical models predict that around 5% of cases may take greater than 21 days to develop [1].

The onset of EVD is sudden and early symptoms include flu-like illness, fever, muscle pain (myalgia), fatigue (weakness), headache and sore throat [9]. The next stage of the disease is characterized by symptoms and clinical manifestations from several organ systems. Symptoms can be gastrointestinal (vomiting, diarrhea, anorexia and abdominal pain), neurological (headaches, confusion), vascular (conjunctival/pharyngeal injections), cutaneous (maculopapular rash) and respiratory (cough, chest pain, shortness of breath) and can include complete exhaustion (prostration). During the first week, patients often deteriorate suddenly, while diarrhoea and vomiting are getting worse. All of these symptoms correspond to the prodromal phase of EVD [48].

After one week hemorrhagic manifestations can appear in more than half of the patients (bloody diarrhea, nosebleeds and hematemesis, petechiae, ecchymosis and puncture bleedings). Some patients develop profuse internal and external hemorrhages and disseminated intravascular coagulation (CDC, 2014). Bleeding into the whites of the eyes may also occur. Heavy bleeding is uncommon; if it occurs, it is usually located within the

Table 2: Laboratory diagnosis of Ebola and Marburg hemorrhagic fever

Test	Source	Target	Remarks
Polymerase chain reaction (PCR)	Blood, serum, tissues	Viral nucleic acid	Rapid and sensitive but requires special equipment
Antigen ELISA	Blood, serum, tissues	Viral antigen	Rapid and sensitive, but requires special equipment
Immunohistochemistry	Tissues (e.g., skin, liver)	Viral antigen	Inactivated material, but require time
Fluorescence assay(FA)	(e.g. liver)	Viral antigen	Rapid and easy, but interpretation is subjective
Electron microscopy	Blood, tissue	Viral particle	Unique morphology (immune staining possible), but intensive and require expensive equipment
Immunofluorescence assay	Serum	Virus specific antibodies	Simple to perform, but prone to non-specific positive and negative interpretation
Virus isolation	Blood, tissue	Viral particle	[0] Virus available for studies, but needs bio contaminant require time

Source: [51]

gastrointestinal tract [8]. Recovery may begin between seven to twenty-four days after first symptoms Death, if it occurs, follows typically six to sixteen days from first symptoms and is often due to low blood pressure from fluid loss. Additionally, they develop antibodies against Ebola that last at least 10 years, but it is unclear if they are immune to repeated infections. If someone recovers from Ebola, they can no longer transmit the disease [49].

Diagnosis and Specific Laboratory Testing: Nonspecific laboratory testing: Laboratory work with the viruses is permitted only in biosafety level four (BSL4) laboratories [33]. Possible laboratory indicators of EVD include a low platelet count. An initially decreased white blood cell count followed by an increased white blood cell count. Elevated levels of the liver enzymes Alanine aminotransferase (ALT) and Aspartate aminotransferase (AST) and abnormalities in blood clotting often consistent with Disseminated intravascular coagulation (DIC) such as a prolonged prothrombin time, partial thromboplastin time and bleeding time [37].

The diagnosis of EVD is confirmed by isolating the virus, detecting its RNA or proteins, or detecting antibodies against the virus in a person's blood. Isolating the virus by cell culture, detecting the viral RNA by Polymerase chain reaction (PCR) and detecting proteins by Enzyme-linked immunosorbent assay (ELISA) are best methods used in the early stages of the disease. Detecting antibodies against the virus is most reliable in the later stages of the disease and in those who recover [1]. Immunoglobulin M (IgM) antibodies are detectable two days after symptom onset and Immunoglobulin N (IgG) antibodies can be detected six to eighteen days after symptom onset [50].

During an outbreak, isolation of the virus via cell culture methods is often not feasible. In field or mobile hospitals, the most common and sensitive diagnostic methods are real-time PCR and ELISA. In 2014, with new mobile testing facilities deployed in parts of Liberia. Laboratory test results were obtained three to five hours after sample submission [22].

Differential Diagnosis: Early symptoms of EVD may be similar to those of other diseases common in Africa, including malaria and dengue fever. The symptoms are also similar to those of Marburg virus disease and other viral hemorrhagic fevers. The complete differential diagnosis is extensive and requires consideration of many other infectious diseases such as typhoid fever, shigellosis, rickettsial diseases, cholera, sepsis, EHEC enteritis, leptospirosis, scrub typhus, plague, Q fever, candidiasis, histoplasmosis, trypanosomiasis, visceral leishmaniasis, measles and viral hepatitis among others [52].

Non-infectious diseases that may result in symptoms similar to those of EVD include acute promyelocytic leukemia, hemolytic uremic syndrome, snake envenomation, clotting factor deficiencies/platelet disorders, thrombotic thrombocytopenic purpura, hereditary hemorrhagic telangiectasia, Kawasaki disease and warfarin poisoning [53].

Prognosis for EVD has a high risk of death in those infected which varies between 25 percent and 90 percent of those infected The highest risk of death was 90 percent in the 2002–2003 in Republic of the Congo outbreak [22]. Death, if it occurs, follows typically six to sixteen days after symptoms appear and is often due to low blood pressure from fluid loss. Early supportive care to prevent dehydration may reduce the risk of death [54].

Treatment: Treatment is primarily supportive in nature. Early supportive care with rehydration and symptomatic treatment improves survival. Rehydration may be via the oral or by intravenous route. These measures may include management of pain, nausea, fever and anxiety [55]. The World Health Organization recommends avoiding the use of aspirin or ibuprofen for pain due to the bleeding risk associated with use of these medications [22].

Blood products such as packed red blood cells, platelets or fresh frozen plasma may also be used. Other regulators of coagulation have also been tried including heparin in an effort to prevent disseminated intravascular coagulation and clotting factors to decrease bleeding. Antimalarial medications and antibiotics are often used before the diagnosis is confirmed, though there is no evidence to suggest such treatment helps. A number of experimental treatments are being studied [55].

Prevention and Control: Bush meat, an important source of protein in the diet of some Africans, should be handled and prepared with appropriate protective clothing and thoroughly cooked before consumption. Some research suggests that an outbreak of Ebola disease in the wild animals used for consumption may result in a corresponding human outbreak. Since 2003, such animal outbreaks have been monitored to predict and prevent Ebola outbreaks in humans [20].

The infected person should be in barrier-isolation from other people. All equipment, medical waste, patient waste and surfaces that may have come into contact with body fluids need to be disinfected [56]. During the 2014 outbreak, kits were put together to help families treat Ebola disease in their homes, which include protective clothing as well as chlorine powder and other cleaning supplies. Education of those who provide care in these techniques [57].

Ebola viruses can be eliminated with heat (heating for 30 to 60 minutes at 60.2°C or boiling for five minutes). To disinfect surfaces, some lipid solvents such as some alcohol-based products, detergents, sodium hypochlorite (bleach) or calcium hypochlorite (bleaching powder) and other suitable disinfectants may be used at appropriate concentrations. Education of the general public about the risk factors for Ebola infection and of the protective measures individuals may take to prevent infection is recommended by the World Health Organization. These measures include avoiding direct contact with infected people and regular hand washing using soap and water [5].

Vaccine: In addition to these therapeutic treatments, the development of effective prophylaxis to prevent EBOV infection would be beneficial for communities, healthcare workers in filovirus-endemic regions, as well as against accidental laboratory exposure. Many Ebola vaccine candidates had been developed in the decade prior to 2014 outbreak [58].

Conventional Vaccines: Conventional inactivated vaccines have been generated through inactivation of EBOV by heat, formalin, or γ-irradiation. Several conventional vaccine candidates were not effective at stimulating protective immune responses [59]. Inactivated Ebola virus vaccines were shown to not promote an adequate immune response to the real pathogen. Several promising vaccine candidates that integrate viral subunits have been shown to protect non-human primates (usually macaques) against lethal infection. Despite altering the route of administration (subcutaneous (S.C.) vs. Intramuscular (I.M.) injection) or adjusting the timing of booster injections [60].

Sub-Unit Vaccines (Non-Viral): EBOV genes inserted into a DNA plasmid can be injected directly into a patient's muscle, where expression of the antigen can elicit an immune response to the corresponding virus particle. The use of DNA vaccines can be advantageous as they can lead to the generation of antibody and cytotoxic T lymphocytes [61].

Virus-like Particles (VLPs) as a Candidate Vaccine: VLP vaccines, unlike viral vaccines inactivated by heat, chemical or ??-irradiation, can present filoviral antigens in a presumably native form. EBOV-like particles have been examined as potential vaccine candidates and can be generated by the expression of VP40 alone or along with GP. Mice vaccinated with VLPs expressing ZEBOV VP40 and ZEBOV GP followed by either two booster injections, or one booster with QS-21 adjuvant, resulted in complete protection from a challenge with a lethal dose of mouse-adapted ZEBOV [62].

Vesiculovirus (VSV)-Based Candidate Vaccines: Candidate vaccines based on replication competent recombinant VSV can grow to high titers and induce a strong humoral and cellular response in humans. Mice vaccinated and boosted with recombinant VSV expressing ZEBOV GP (VSV-GP) survived a challenge of ZEBOV with complete protection [63].

Vector-Based Vaccines: Viruses can be used as vaccine vectors when genes encoding antigens of EBOV are inserted and expressed from the viral carrier. Viral vectors can be replication competent or defective. While replication competent vectors generally elicit strong and long-lasting immune responses following immunization, these platforms may not be recommended for use in

immunocompromised individuals. Defective viral vectors, while potentially safer, may require multiple doses to achieve optimal immunity [64].

Impact and Implications of the Ebola Crisis: The current Ebola outbreak is concentrated in Sierra Leone, Liberia and Guinea: countries with limited state capacity which are recovering from political instability and conflict [65]. The indirect consequences of the Ebola epidemic and its disruption of public and private services threaten the lives and livelihoods of more than 22 million people in Ebola-affected areas As well as its immediate impact on people's health, the crisis is likely to have longer-term socio-economic and political consequences which present risks to the countries' development and stability [66].

Political Impact and Implications: The political impact of the crisis has not yet been assessed deeply. However, initial analysis suggests that as a result of the governments' poor management of the Ebola crisis, the epidemic threatens to become a political crisis in any or all of the affected countries because of the deep frustration it has generated and the gradual deterioration of the security situation [67].

Economic Impact and Implications: The Ebola crisis has had a negative impact on the economies of the countries it has affected and its impact can be felt in many different sectors. The economic impacts include loss of gross domestic output, threat to food security, fall in employment and livelihoods and decline in foreign investment [68].

Social Impact and Implications: The Government of Sierra Leone and its partners predict that progress in human development is likely to be reversed due to the impact of the Ebola crisis on health, education and standard of living. Many development organizations have suspended their operations, leaving their target groups without any input [69].

Security Impact and Implications: The population of the various countries are deeply frustrated with their governments' poor response to the epidemic. Security forces have played a central role in the crisis response, especially in relation to enforcing quarantines [67]. Under current caseloads, the impact of the West African Ebola crisis has deepened, particularly in Liberia and Sierra Leone 2014 financing gaps for the three core countries range from US $80 million to US $120 million, summing to over US $290 million. Slow containment scenarios will lead to even greater financing gaps in 2015 [70].

CONCLUSION

Ebola virus is a type of filovirus which is endemic in some African countries. EBOV carries a negative-sense RNA genome in virions that are cylindrical/tubular and contain viral envelope, matrix and nucleocapsid components. The virus is transmitted from bats non-human primates to humans by direct contact, hunting and eating of bush meat this is known as spillover transmission. High risk of transmission occurs percutaneous, needle stick, mucosal exposure to virus contaminated blood bodily fluids and tissue or laboratory specimens in severely ill or unknown patients. After enters in to the body first it affects macrophages and dendritic cells. The patient shows different sign and symptoms such as gastrointestinal, neurological, vascular cutaneous and respiratory and can include complete exhaustion (prostration). During the first week, patients often deteriorate suddenly, while diarrhea and vomiting are getting worse. After diagnosis supportive and different blood products are administer to the patient. In order to prevent disease outbreak peoples should have to take care in; any form of close contact with wild animals, consumption of any type of bush meat, contact with symptomatic patients and/or their bodily fluids and hygienic condition of humans.

Based on the above conclusion, the following are recommended to:

- Supportive therapy, different blood products and heparin should be administered to the patient
- Avoiding any form of unsafe contact with symptomatic patients and/or their bodily fluids and wild animals (including monkeys, forest antelopes, rodents and bats), both alive and dead.
- Avoid consumption of any type of bush meat which is not properly cooked is recommended.

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