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A REVIEW ON DISEASE AND TREATMENT OF EBOLA VIRUS

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Abstract

Ebola virus Diseases (EVD) has mainly affected economically impoverished countries as restricted resources negatively influence a country's infrastructure and administration. Explored into the factors that showed to the universal outbreak, surroundings forth plans to counter EVD cases in developing countries, and conceving specific measures to maximum the lay out of the diseases are the necessary treads that must be instantly taken. In this review we condensed the pathogenesis of EVD and the elements that showed to its spread. We also focus the mediation worked by determined countries that have strongly restricted the epidemic, and add some preventive estimate after studying the present data. According to the available data barriers to prevent and control the disease in affected include irresolute and disorganized health system, substandard sanitary condition, poor personal hygiene practices, chief authorities in spreading countries must conceive plans, the present resources in intellect to deal with the breakout before it happens. As a first tread, communities should be instructed on EVD's indication, history, mode of transmission, and methods of protection, which include the importance of personal hygiene implementation, through seminars newspapers, and other social media. A popular opinion leader (POL) sharing this information would more help to remove the error about the nature of the diseases and incidentally enhance the quality of life of pompous patients and their families.

Keywords: Ebola haemorrhagic fever, Epidemiology, Ebola virus, physiology, transmission, prevention and control.

INTRODUCTION

EBOV infects primarily humans, monkeys, and bats; but other species such as mice and fawns may also contact infection. There are five identified species of EBOV, four species (Zaire Ebola virus, Sudan Ebola virus, Tai Forest Ebola virus, and Bundibugyo Ebola virus) are known to infect humans and cause disease, whereas Reston Ebola virus is non-human primate pathogen [1-3]. Ebola virus are the constructive source of Ebola hemorrhagic fever (EHF). The first infection was recognized in 1976 in the Northern Democratic Republic of Congo, in Zaire as well. Since then, E. virus disease (EVD) became endemic in Africa. Between the particular epidemic areas, 318 cases were recorded in DRC, and 284 cases were recorded in Sudan and two dissimilar species of EBOV were identified: EBOV-Zaire and EBOV Sudan. In 1977, one fatal case due to EBOV was reported in Zaire, and EBOV subsequently remerged with 34 cases, 22 of which were fatal in Sudan in 1979. No further cases were recorded until 1994. In 1995, EVD due to EBOV remerged in the DRC [3-7].

Structure of E. virus

E. virus is a filamentous shape virus with dimensions of 800 nm long and 80 nm in diameter and has an encapsulated single-stranded negative RNA [9]. There are seven indicates the proteins by Ebola: Nucleoprotein (NP), glycoprotein (GP), RNA-dependent RNA polymerase (L), and four constitutional viral proteins: (VP24), (VP30), (VP35), and (VP40) (Fig. 1) [10-13]. The role of these proteins is summarized as follows:

• NP: Essential for RNA encapsulation

• GP: Essential for the attachment of the virus to the host cell membrane and entering the nucleo capsid of the virus into the host cytoplasm.

• VP24: Essential for virus assembling and in transcription by being a part of the nucleo capsid structure [14]

• VP30: Suppression of viral RNA silencing

• VP35: Secure to NP to reduce the nucleocapsid to facilitate the transcriptional declaration

• VP40: Required for virus localization out of the host cell membrane and gives filamentous shape to virus together with GP and helps maintain the structural integrity of the virion [15-17].

LIFE CYCLE

The natural reservoir host of E. virus is fruit bats and accidental hosts are humans and non-human primates. E. virus can be directly transferred by blood or body fluids such as urine, saliva, sweat, faces, breast milk, and semen. E. virus also can be transferred by sexual contact [19-23]. After entering the body through small wounds on the skin or mucous membranes, the virus targets monocyte/macrophages and dendritic cells. The infection then spreads through the lymphatic vessels to regional lymph nodes and from there causes secondary viremia infecting the spleen, liver, and adrenal glands (Fig. 2) [13]. Steps of the virus life cycle: Viruses attach to the host receptors by GP which is endocytosed into vesicles in the host cell. Then, the viral membrane fuses with the vesicle membrane, and the nucleocapsid is released into the cytoplasm. The transcription of RNA process begins with the binding of the polymerase complex to a single binding site located within the leader region of the genome. The complex then slides along the RNA template and sequentially transcribes the individual genes in their 3'–5' order. Encapsidated, negative-sense genomic ssRNA is used as a template for the synthesis (3′–5′) of polyadenylated, monocistronic mRNAs and, using the host cell's ribosomes, tRNA molecules, etc., the mRNA is translated into individual viral proteins, with an increase of viral protein levels, a switch occurs from translation to replication. Assembly starts by the nucleocapsids which accumulate in the perinuclear region; then it is

Table 1: The chronology of previous EVD outbreaks

S.No.	Year	Country	EVD	Cases	Death
1	2018–2019	The DemocraticRepublic of the Congo	Zaire	Ongoing	
2	2018	The DemocraticRepublic of the Congo	Zaire	54	33
3	2017	The DemocraticRepublic of the Congo	Zaire	8	4
4	2015	Italy	Zaire	1	0
5	2014	Spain	Zaire	1	0
6	2014	UK	Zaire	1	0
7	2014	USA	Zaire	4	1
8	2014	Senegal	Zaire	1	0
9	2014	Mali	Zaire	8	6
10	2014	Nigeria	Zaire	20	8
11	2014-2016	Sierra Leone	Zaire	14124*	3956*
12	2014-2016	Liberia	Zaire	10675*	4809*
13	2014-2016	Guinea	Zaire	3811*	2453*
14	2014	The DemocraticRepublic of the Congo			
15	2012	Democratic Republic of Congo	Bundibug	57	29
16	2012	Uganda	Sudan	7	4
17	2012	Uganda	Sudan	24	17
18	2011	Uganda	Sudan	1	1
19	2008	Democratic Republic of Congo	Zaire	32	14
20	2007	Uganda	Bundibug	149	37
21	2007	Democratic Republic of Congo	Zaire	264	187
22	2005	Congo	Zaire	12	10
23	2004	Sudan	Sudan	17	7
24	2003	Congo	Zaire	35	29
25	2003	Congo	Zaire	143	128
26	2001-2002	Congo	Zaire	59	44
27	2001-2002	Gabon	Zaire	65	53
28	2000	Uganda	Sudan	425	224
29	1996	South Africa	Zaire	1	1
30	1996	Gabon	Zaire	60	45
31	1996	Gabon	Zaire	31	21
32	1995	Democratic Republic of Congo	zaire	315	254
33	1994	Côte d'Ivoire	tai Forest	1	0
34	1994	Gabon	zaire	52	31

35	1979	Sudan	sudan	34	22
36	1977	Democratic Republic of Congo	zaire	1	1
37	1976	Sudan	Sudan	284	151
38	1976	Democratic Republic of Congo	Zaire	318	280

transported to the budding sites at the plasma membrane. Budding come about at the plasma membrane where VP40 and GP play a vital roles in the budding procedure. Ultimately, the particles is released [25-28]

Symptoms of the diseases:

The incubation period usually extends 5–7 days, although it can be as minimum as 2 days and as maximum as 21 days. Approximately 95% of the patients show signs within 21 days after the infection which is the recommended period for the follow-up of the disease. Typical symptoms include fever, profound weakness, diarrhea, abdominal pain, cramping, nausea, and vomiting for 3–5 days and may persist for up to a week. Laboratory complications including elevated aminotransferase levels marked lymphocytopenia, and thrombocytopenia may occur. Clinical EHF is presented by unexpected arrival of fever, fatigue, chills, general malaise, headaches, myalgia, anorexia, and gastrointestinal distress within 3–13 days following submission to the virus [23, 30-32].

DIAGNOSIS:

Rapid and reliable diagnosis of EVD is essential for appropriate and effective patient management. Diagnosis of suspected cases is confirmed by EBOV-specific laboratory tests that detect the EBOV genome (e.g., reverse transcriptase polymerase chain reaction [RT-PCR]) [33]. During the period of Ebola infection, viral RNA can be detected by RT-PCR in saliva, tears, sweat, breast milk, urine, vaginal fluid, and seminal fluid regardless of the acute disease [9,12]. Infection can also be diagnosed by measurement of the EBOV antigen or specific antibodies [34], IgM antibodies can be detected starting from 2 days after the first symptoms appear and disappear after 30–168 days. IgG response is generally considered to start between 6 -18 days post-onset of illness and remains detectable for years [3]. In the past 10 months, the West Africa EVD outbreak has stimulated the development of new diagnostic tests, including rapid antigen detection tests and nucleic acid detection tests such as loop-mediated isothermal amplification assays [34].

INFECTION CONTROL AND TREATMENT:

Immediate isolation of infected cases is very important before proceeding in any action [32]. The risk of E. virus infection can be decreased by averting contact with blood or body fluids from infected people, addition to avoid visiting the patients in the hospital, and by careful hand washing and hygiene [12]. It is necessary to abstain from breastfeeding for the possibility of transmission of the virus through the milk, in addition to safe sex practices, especially after the appearance of infection after the recovery of infected people [19]. So far, there is no authorized safe and effective treatment for EVD. Current treatment is merely supportive, including the control of pain and secondary infections, as well as fluid therapy [35]. Symptoms and complications of EVD should be treated immediately after they occur. Hypovolemia due to massive fluid loss through vomiting and diarrhea is the most common symptom of EVD. Thus, it is necessary to maintain fluid volume by modulation. The electrolyte ratios regulate the daily fluid input and output. It was also observed that antiemetic and antidiarrheal drugs may limit the massive loss of fluids and should be approved [36] when disseminated intravascular coagulation develops. They must control the coagulation factors, the remedy of thrombocytopenia and anemia. In addition, respiratory failure is more often secondary to EVD complications, and therefore, oxygen therapy in severe cases should be used [17,37-39]

WHO has issued a document for the classification, testing, and use of drugs in patients who are infected with the E. virus [11]. With the global effect of the West Africa outbreak EVD, research and development for new Ebola vaccine candidates have been stimulated, though no authorized vaccine is currently available. Previously, the development of vaccine candidates has led to the initiation of Phase I, II, and III human clinical trials [29,41].

CONCLUSION:

The current EVD outbreak urges the health care and public health systems to respond to infectious disease emergencies and develop the healthcare infrastructure in developing countries and to increase awareness in countries at risk for EVD imported cases. Human Ebola outbreaks usually occur unexpectedly with a subsequent rapid spread from person to person. E. viruses are highly contagious infectious. Understanding the clinical aspects, immediate diagnosis and suitable treatment are major steps toward the prevention of death and transmission of the virus to other people.

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