IJCRT.ORG





INTERNATIONAL JOURNAL OF CREATIVE RESEARCH THOUGHTS (IJCRT)

An International Open Access, Peer-reviewed, Refereed Journal

Ebola Virus Disease (EVD): A REVIEW

¹Bhosale Mohini Gundappa, ²Sakhare Raghunath S., ³Dhumal Dipali G., ⁴Gotmukle Omkar N.

¹B Pharm Student, ²Assistant Professor, Dept. of Pharmaceutics, ³B Pharm Student, ⁴B Pharm Student Latur College of Pharmacy, Hasegaon, Maharashtra, India

Abstract: The Ebola Virus Disease (EVD) also known for hemorrhagic fever initially seen in the democratic republic of Congo. Endemic to Africa the outbreaks are recorded in six other countries. The fruit bat is considered to be the natural host for the Ebola virus disease. The Ebola virus transmit through the close contact with blood and body fluids of infected person. The Ebola virus replicate through their own genetic material insert in DNA of host cell and hijack all cellular progresses. The incubation period of Ebola virus is between 6 to 16 days. The symptoms of Ebola virus is diagnosed by the several ways, symptoms of Ebola virus disease includes the high onset of fever, headache in early phase, vomiting, diarrhoea and possible progression to hemorrhagic fever rash and at last phase multi organ failure. The infected person should stay in isolation and quarantine. The anti-viral therapy for EVD, immunotherapies, vaccines are some ways to treat the Ebola virus.

Keywords: Ebola virus, outbreaks, Ebola hemorrhagic fever

I. INTRODUCTION

Ebola virus disease (EVD) is called as Ebola hemorrhagic fever is a sever disease caused by Ebola virus. It occurs in human and non-human primates. The Ebola virus disease was identified in 1976 in almost continuous outbreaks in the Democratic Republic of the Congo and Sudan. Since 2014 World Health Organization (WHO) reported major outbreak of Ebola virus disease in quinoa, Sierra Leone and Liberia of Western American countries. This outbreak of EVD reported from the remote villages of West Africa and this outbreak caused by the ZEBOV virus is the longest and largest dead list and the most complex in history.

There are 4 species of Ebola virus are known to cause disease in human:-

- 1. Zaire Ebola virus (EBoV)
- 2. Sudan Ebola virus (SUDV)
- 3. Bundibugyo Ebola virus (BDDV)
- 4. Tai forest Ebola virus (TAFV)

RESTV (Reston Ebola virus) cause major illness in non-human primates. It was first identified in 1989 in rest on (US) in monkey imported from the Philippines and cause outbreaks in non-human primates in U.S and Italy. In 2018, the Bombali Ebola virus was discovered in bats in sierra Leone and yet it is not known that this species is pathogenic to human.one of the reason that Ebola virus is so dangerous due to its symptoms are various and appears quickly and the hemorrhagic fever is not diagnosed rapidly resembles to other viruses. The infection is characterized by the high fever, the subsequent sign of viral infection indicates multi system involvement such as respiratory, gastro intestinal, neurological manifestation and symptoms are easily mistaken with the malaria, dysentery, influenza, typhoid fever and several bacterial infection. After initial infection the fever often progresses to cause serious symptoms like bleeding from mucous membrane, internal and external hemorrhage from nose, mouth and anus and bleeding from brain can leads to severe depression. The span time between infections to death is 6-16 days.

Source/Reservoir:

The natural reservoir for the Ebola virus disease has not identified until today, but in Africa fruit bats mainly species from the pteropodiadae family are considered possible natural host for Ebola virus. As a result the geographical distribution of Ebola virus may extend with the range of the fruit bats. There are three types of fruit bats such as Hypsignathusmonstrous, Myonscteristorquatas and Epomops carry the virus. Although non-human primates are known to have been source of infection for humans in a no. of previous EVD Outbreaks. They are considered as accidental rather than they are not host reservoir.

Transmission:

Ebola virus is introduced into the human population although close contact with blood, organ or other body fluids of an infected animals. The first human Ebola virus disease outbreak (2014 to 2016) in the West Africa. Ebola virus can be spread through person to person through direct contact with the blood, broken skin of the body fluids of infected person and indirect contact with environmental things that contaminated with such fluids such as needles or soil clothing that have been contaminated with infectious secretions. The transmission does not occurs during the incubation period only occurs once an infected person present with EVD symptoms. Burial ceremonies that involve the direct contact with the body of infected person that play a role in transmission of Ebola virus. The Ebola virus can carry in some area of body even after little illness. These areas include the testes, placenta, CNS and interior of the eye. The Ebola virus can be present in semen for few months after the recovery from EVD. The WHO states that only sick peoples are able to spread the EVD via saliva and virus has not been reported to be transmitted through sweat. There is no evidence of transmission of Ebola virus through contact with skin or through coughing or sneezing.

Pathogenesis:

The pathophysiology of Ebola virus is not yet understood. The incubation period is vary and it is depending on the type of explorer. The WHO finding that the mean incubation period is 11.4 days which is not different in different country. The symptoms of viral infection occurs in 8 to 10 days. The incubation period is related to the infection route. The Ebola virus is migrates from the infection site to lymph nodes, liver, spleen and adrenal gland and then resulting in decrease in the lymphocyte count, hepatic cellular necrosis is occur and it is associated with the deregulation of clotting factors and coagulopathy. Adreno cortico necrosis is also found and it is associated with hypotension and impaired steroid synthesis. Inadequate support, care it resulting in organ failure and death within 10 days of symptoms occurs in humans.

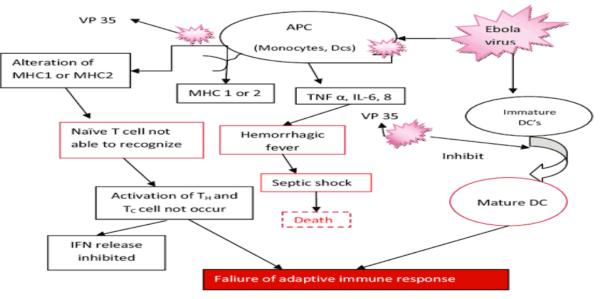


Fig.1: Pathophysiology of Ebola Virus Disease

Entry:

There are two types of cell entry proteins first is host encoded Niemann-pick C1 (NPC1) which is cholesterol transport protein essential for entry of virus into the host cell and for its replication and second is TIM1 which binds to the receptor binding domain of EBoV glycoprotein to increase the receptivity of Vero cells. After entering the body through the mucus membrane Ebola virus infects many cells. The macrophages and dendrites and cells are first infected. Filo virus replicate fastly in these cells causing necrosis and releasing new viral particles into extracellular fluid. Quick fundamental spread is added by infected prompted 10 concealment of type1 infection responses. Dissemination to provincial lymph nodes brings about additional rounds of replication followed by spread through the circulation system to dendrites cells and fixed and portable macrophages in the liver, spleen, thymus and other lymphoid tissues.

Sign and symptoms

The incubation period of Ebola virus disease (EVD) is about 2 to 21 days. EVF is characterized by the acute viral illness and sudden onset of fever (greater than 38°c to 101° F). The muscle pain, illness, weakness, sore throat, headache, vomiting, rashes, diarrhea, impaired kidney dysfunction, internal and external bleeding, low WBC count and liver enzymes elevation observed in patient. In man after 61 days cure from EVD Ebola virus was found in semen. The peoples who cures from EVD develops antibodies and antibodies present in that person for at least 10yrs.

The clinical features can be divided into four phases:

Phase-I: In phase I, the sign & symptoms are non specific such as high fever, nausea, headache, etc.

Phase-II (day1 to 6): In phase II, the fever is not responds to antimalarial drugs, intense fatigue followed by diarrhoea, vomiting, and abdominal pain.

Phase-III (day 7 to 8): In this phase patient feel better & seek food and health is also improve in some patients who are recover during this phase.

Phase-IV (day 9): If health status worsted not recover the skin manifestation purpura (skin rash), cardiovascular distress & respiratory disorder are observed. Diagnosis

Early research Centre affirmation of through clinical hemorrhagic fever cases is fundamental for execute suitable control measures. Conclusive finding of associated cases with EHF is generally made by PCR discovery and infection on Vero cells. As a class 4 microorganism Ebola infection culture require a greatest control office. Extra lab symptomatic tests in corporate ELISA for the discovery of Ebola IgG and IgM expluct antibodies and infection antigens, more particular sub-atomic testing is likewise accessible however isn't promptly accessible In the clinical setting. The negative test result within the first 48 cycles after infection does not rule out EBoV infection. Because of the rapid onset of auto illness, serological testing has no role in diagnosis. The diagnosis of acute Ebola virus disease is occur by viral genome detection via RTPCR after infection virus is detected in 48 hours in both lethal and non-lethal cases. After the last Ebola virus outbreak in kaluamba, DRC (2008-2009), the EBoV analytical advance of ELISAs for recognition of antigens and IgM immunizer, and RT-PCR have been removed to INRB in Kinshasa.

Management and Treatment:

The board of patients experiencing EVD involves steady consideration for example, keeping up the fluid alongside electrolyte balance, circulatory strain & oxygen immersion. This likewise incorporates treating complexities emerging from optional diseases. Non etheless, numerous, clinical, immunotherapy and nucleic corrosive treatment approaches have been reported and are under additional examination.

1. Anti-viral therapy:

Due to the prerequisite of biosafety level 4 facilities in a lab. Explores have utilized switch hereditary qualities to distinguish new focuses inside viral genomes of the EBOV for medication and immunization improvement. Turn around hereditary qualities permits the development of recombinant viruses, for example, EBOV. Containing key quality successions however are non-replicating and subsequently non-

infective. This strategy has been utilized by Martinez and partners to understand quality capability in EBOV examination to concentration infection passage, replication and gathering. Ribavirin and lamivudine have been attempted as a way to treat EVD.

Ribavirin disrupts the covering of the viral mRNA, while lamivudine is a nucleoside simple that meddles in quality replication. Nonetheless ribavirin brought about diminished mortality in human instances of Lassa fever and monkeys with Fracture Valley fever infection yet has not been viable in that frame of mind of loviral and Baviviral infections. Moreover, no obvious endurance benefit was seen with lamivudine treatment. Another enemy of viral special is known as T-705 (favipiravir) has gone through creature preliminaries to consider its efficacy in contrast to EBoV. In spite of the fact that it was at first evolved by Fujišim, Japan for treating influenza infection contamination by restraining a viral chemical, the creature studies have now confirmed that favipiravir is compelling in treating creature stained with the air borne E718 kind of EBOV.

2. Immunotherapy:

Uninvolved resistant treatment or recovering immune plasma for treatment of EVD was uniquely utilized in a 1995 flare-up in Kikwit, Zaire. Mu-daddy and his partners used this treatment to treat eight patients with EVD, out of which seven endure. This procedure utilizes plasma from recuperated EVD patients to kill antibodies. WHO gave ongoing rules for the expected utilization of blood items from EVD survivors. This rule resolved different issues going from identification of appropriate blood or plasma benefactors among EVD survivors, give resent and selection, contributor blood assortment, as well as stock piling of entire blood and plasma alongside transportation.

3. vaccines:

Vaccines Due to the pathogenicity of the infection, no customary vaccines consisting of inactivated/killed infection, or produced using a lessened viral strain, are being created for Ebola as a result of the gamble of incomplete inactivation of the infection or of inversion to a completely dynamic structure. Instead, these antibodies depend on generally new methodologies that have been made conceivable with the progression of sub-atomic science and recombinant hereditary advances presented during the last 10-20 years.

Clinical preliminaries for a few competitor immunizations are in various phases and a safe and effective antibody is expected. Currently, two antibody up-and-comers are entering efficacy preliminaries in humans. The first is cAd3-ZEBOV created by GlaxoSmithKline and tested by the US Public Organization of Sensitivity and Irresistible Diseases (NIAID). The second is the rVSV tried by the New Connection Genetics Corporation after being authorized from the General Wellbeing Organization of Canada. The two antibodies showed promising paces of efficacy in non-human primates. The many clinical trials are firstly initiate in Africa & Australia, while the United State, Europe and Asia started the manufacturing of new vaccines of EVD. Anti-Ebola virus vaccine have been placed in several clinical trials and finally the Food and Drug Administration of US approved the Ebola vaccines rVSV-ZEBoV and this vaccine is a single dose infection & it is a live, attenuated vaccine genetically engineered to contain a protein from the Zaire Ebola virus.

Conclusion:

The regular episodes of Ebola virus have caused numerous mortalities and morbidities. Since the infection might prompt a pandemic, its counteraction has been happened to most extremely significance as it is profoundly equipped for causing significant physical and monetary economic burden so there is need to conduct clinical trials on EBoV to establish possible treatment to prevent further outbreaks.

References:

- 1. Gull and A. Fifteen countries are at risk of Ebola outbreak, saysWHO.BMJ2014; 349:g6305.
- 2. KhanAS, TshiokoFK, HeymannDL, LeGuennoB, NabethP, KerstiënsB, etal. The reemergence of Ebolahemorrha gicfever, DemocraticRepublicoftheCongo, 1995. Commission de Luttecontreles Epidémies à Kikwit. JInfect Dis1999;179(Suppl1):S76-S86.
- 3. CDCSpecialPathogensBranch(2010).EbolaHemorrhagicFeverCaseCountandLocationList.Ebola HemorrhagicFeverInformationPacket.
- 4. Johnson, K.M. & Breman, J.G. (1978) Ebolahaemorrhagic feverin Zaire, 1976. Bulletinof the World Health Or ganization56:271-293.
- 5. HoenenT, GrosethA, FeldmannH(July2012). "CurrentEbolavaccines". ExpertOpinBiolTher12:859-72
- 6. Kuhn JH, Becker S, Ebihara H, Geisbert TW, Johnson KM, Kawaoka Y, Lipkin WI, Negredo AI, Netesov SV, Nichol ST, PalaciosG, Peters CJ, Tenorio A, Volchkov VE, Jahrling PB (December2010). "Proposal for a revised taxonomy of the family Filoviridae: Classification, names oftaxaandviruses, and virus abbreviations". Archives of Virology 155:2083–103.
- 7. Klenk, H., & Feldmann, H. (2004). Ebolaand Marburg Viruses: Molecular and Cellular Biology. Wym ondham:HorizonBioscience.
- FunkDJ,KumarA(November2014)."Ebolavirusdisease:anupdateforanesthesiologistsan 8. dintensivists".CanJAnaesth.7.DrazenJM,Kanapathipillai.

9. KalengaOI, MoetiM, SparrowAetal. The Ongoing Ebola Epidemic in the Democratic Republic of Co ngo,2018-2019.NEnglJMed.2019;381,373-83.

Democratic 10. World Health Organization. Ebola virus disease _ Republic of the Congo.Diseaseoutbreaknews25July2018.Retrievedfromhttps://www.who.int/csr/don/25-july-2018-eboladrc/en/LastaccessedMarch16,2020.

- 11. MurrayMJ.EbolaVirusDisease: A Review of Its Pastand Present, Anesth Analg, 2015;121(3), 798-809.
- 12. HasanS, AhmadSA, MasoodR, et al. Ebolavirus: Aglobalpublichealthmenace: Anarrativereview .JFamilyMedPrimCare.2019;8(7),2189-2201.
- 13. CentersforDiseaseControlandPrevention.40YearsofEbolaVirusDiseasearoundtheWorld.2019.Retri evedfromhttps://www.cdc.gov/vhf/ebola/his-tory/chronology.html.LastaccessedDecember25,2019.
- 14. MalvyD,McElroyAK,deClerckH.Lancet.2019;393(10174),936-48.
- 15. "Ebolavirus Pathogen Safety Data Sheets". Public Health Agency of Canada 16.. Laupland KB, Valiquette L. (May 2014). "Ebola virus disease". Can J Infect Dis Med Microbiol 25: 128-9.
- 17. Leroy EM, Kumulungui B. Pourrut X. Rouquet P. Hassanin A, Yaba P. Délicat A. Paweska IT. Gonzalez JP. Swanepoel R (December 2005). "Fruit bats as reservoirs of Ebola virus". Nature 438: 575-576.

18. Olival KJ, Islam A. Yu M, Anthony SJ, Epstein JH, Khan SA. Khan SU, Crameri G, Wang LF, Lipkin WI, Luby SP, Daszak P (February 2013). "Ebola virus antibodies in fruit hats, Bangladesh". Emerging Infect Dis 19: 270-3.

19. Saced, M.F.. Kolokoltsov, A.A. Albrecht, T. & Davey, R.A. (2010). Cellular Entry of Ebola Virus Involves Uptake by a Macropinocytosis-Like Mechanism and Subsequent Trafficking through Early and Late Endosomes. PLOS Pathogens, 6(9).

20. Misasi J, Sullivan NJ (October 2014). "Camouflage and Misdirection: The Full-On Assault of Ebola Virus Disease". Cell 159: 477-86.

21. Chippaux JP (October 2014). "Outbreaks of Ebola virus disease in Africa: the beginnings of a tragic saga". J Venom Anim Toxins Incl Trop Dis 20: 44

22. Goeijenbier M. van Kampen JJ, Reusken CB, Koopmans MP, van Gorp EC (November 2014), "Ebola virus disease: a review on epidemiology, symptoms, treatment and pathogenesis". Neth J Med 72: 442-8.

23. Takada, A., Feldmann, H., Ksiazek, T. & Kawanka, Y. (2003). Antibody-Dependent Enhancement of Ebola Virus Infection. Journal of Virology 77: 7539-7544.

24. Misasi J. Sullivan NJ (October 2014). "Camouflage and Misdirection: The Full-On Assault of Ebola Virus Disease". Cell 159: 477-86.

25. Kühl A. Pohlmann S (September 2012). "How Ebola virus counters the interferon system". Zoonoses Public Health 59: 116-31,