



International Journal of Research Publications

Ebola: the endemic disease

Haleema Sadia^a, Qandeel Zahra^a, Abbas Khokhar^b, Ayesha Alam^c, Shahid Raza^c

^aBaluchistan University of Information Technology, Engineering and Management Sciences, Quetta, Pakistan.

^bKind Edward Medical University, Lahore, Pakistan

^cLahore Garrison University, DHA Phase VI, Lahore, Pakistan.

Abstract

Ebola hemorrhage fever is an endemic, zoonotic and viral disease of family filoviridae. It was first observed in remote villages in Central Africa, near tropical rainforests. Later it occurred in the village near river named Ebola. In West Africa the outbreaks of 2014-2016 ruined major urban areas as well as rural ones. Natural host of the virus is fruit bats and the incubation period is 2-21 days. Symptoms starts fever, headache, sore throat and muscle pain, impaired kidney and liver function. But it can proof fatal if untreated. The outbreak control relies on the prevention and control practices. The early supportive care improves the survival. There is no treatment yet identified but some new drug therapies are promised to be evaluated to decrease the risk. A vaccine rVSV-ZEBOV proofed effective against this infection. Moreover, there is no Ebola virus patient in Pakistan but still it is at higher risk of outbreak. A preventive measure such as proper hygiene is needed to reduce the risk. The purpose of this research is to spread awareness and discuss the emergence of destructive and dangerous infection all over the world and how should we protect our country from this burning issue.

© 2018 Published by IJRP.ORG. Selection and/or peer-review under responsibility of International Journal of Research Publications (IJRP.ORG)

Keywords: Zoonotic disease, fruit bats, vaccine, prevention, Pakistan.

1. Introduction:

Ebola is also referred as hemorrhage fever. It is a dreadful illness which can prove fatal if not treated. It firstly appeared in 1976 in South Sudan and Yambuku. Later it arose in a village near a river called Ebola and from this it gains its name ‘Ebola Virus disease’. Ebola virus belongs to the family Filoviridae. The family includes three genera i.e. Marburgvirus, Ebolavirus and Cuevavirus. The Ebola virus contains five species, Tai Forest, Reston, Sudan, Zaire and Bundibugyo. A large number of outbreaks came in Africa due to Sudan, Bundibugyo and Zaire species (http://www.who.int/csr/don/2003_05_07/en/, accessed 7 May 2003).

2. Structure

Ebola virus has a large and unique structure. They contain variable length filamentous capsid which is sometimes branched. Some of them are 14000 nm long. They are single stranded RNA virus and their helical capsid is enclosed inside the membrane. Several viral protein and glycoprotein are sprinkled within the membrane. Its surface protein is extensively glycosylated due to which it inhibits generation of antibodies against protein. The most important protein is the vp30 present in the matrix of the virus which plays a vital role during binding of the virus from membrane. The nucleoprotein is the primary capsid protein, its N-terminal is in contact with viral RNA and the rest of the portion interacts with other viral protein in the envelope. L protein is associated with viral RNA which functions in replication of the virus on infection (Jahrling et al, 1990).

3. Genome of Ebola virus

The virus contains single stranded negative RNA in its nucleic acid. Its genome is 19kb long and codes for seven genes and seven transcriptional units which are formed in mRNA during infection. The encoding structural gene include,

1. Virion envelope glycoprotein, it produce two gene product soluble 60-70 kDa proteins and 150-170 kDa proteins that enters the viral membrane through transcriptional editing (Volchkov et al, 1995; Sanchez et al, 1996)
2. nucleoprotein
3. matrix protein

4. structural protein i.e. VP24 and VP40
5. nonstructural protein i.e. VP30 and VP35
6. Viral polymerase (Garbutt et al, 2004).

Polyadenylated tails and caps are added on it during the creation of mRNA.

4. Pathogenesis

The glycoprotein plays vital role in displaying the EVD infection. The viral contents of Ebola are released into monocytes and macrophages by the help of GP gene of Ebola virus and then these immune cells release cytokines. Grounds inflammation and fever occurs as a response in endothelial cells which damages the vascular integrity, while the transmembrane glycoprotein is contributing to hemorrhagic fever symptoms when the virus affect the lining of blood vessels and to the cells of reticuloendothelial network and the immune system is altered by small secreted glycoproteins (SGP) which inhibit the neutrophils (Volchkov et al, 2001; Simmons et al, 2002; Prins et al, 2009).

5. Mode of Transmission

The disease is transferred by the fruit bats which belongs to the family Pteropodidae are natural hosts of EVD. In human population it is transmitted through close contacts with blood, secretions, organs or other body fluid of infected animals like, gorillas, monkeys, chimpanzees, forest antelope, fruit bats and porcupines found ill or dead in the rainforest (Feldmann et al, 1994).

Among humans it is transmitted through direct contact with blood secretions, organs or other bodily fluids of infected people, and with all those materials having infectious agents exposure (Rewan and Mirdha, 2014).

6. Symptoms

Incubation period of EVD is 2-21 days (Villinger et al, 1999). Humans do not become infectious until and unless they get symptoms. It starts with the sudden onset of fever, headache, fatigue, sore throat and muscle pain. After that rashes may occur, vomiting, diarrhoea, impaired kidney and symptoms of abnormal liver function and sometimes in severe cases the external and internal bleeding may occur (Stroher et al, 2001).

7. Persistent virus in people recovering from Ebola virus disease

The people who have recuperated from disease. The Ebola virus persists in immune-privileged sites (the sites able to tolerate the entry of antigens without provoking an inflammatory immune

response). These sites includes the central nervous system, inside of eyeball and testicles. In pregnant woman having infection of virus, the virus remains inside the amniotic fluid, fetus and the placenta. The virus persist in breast milk in the breast feeder womens (Sui and Marasco, 2002; Takada et al, 2004; Rolison and Hanosch, 2015).

8. **Diagnosis**

It might be very complicated to differentiate the EVD from other infectious dieses like malaria, meningitis etc. Following methods are used to confirm that the symptoms are caused by Ebola virus infection.

- Serum neutralization test
- Electron microscopy
- ELISA
- Antigen-capture detection test
- RT-PCRA (reverse transcriptase polymerase chain reaction assay)
- Virus issolation by cell culture

The selection of diagnostic test must be considerd very carefully, on which the disease incidence and prevelance, technical specification, social and medical implication of test results depends.

World health organizer also recommended some test which include

- Automated or semi-automated nucleic acid test for routine diagnositic purpose.
- Rapid antigen detection test when nucleic acid test are not readily available.

The specimen required for diagnostic use include:

- Blood collection in ethylenediaminetetraacetic acid from patients who reveals the symptoms.
- Oral fluid in universal transport medium collected from defunct patients if blood collection is not possible .

Biological containment conditions should be adopted in case of laboratory testing on inactivated samples. All national and international transports should be carried out by using triple packing system (Schneider et al, 1998; Zaki et al, 1999).

9. **Treatment and vaccines**

There is no yet proven treatments for EBD but some treatments like blood products,therapies such as drug therapies and immune therapies have been presently upraises. However, an vaccine at experimental stage called rVSV-ZEBOV proves very effective against this infection (Gupta et al, 2001). This vaccine was studied in Guinea in trial involving 11841 people during 2015. Among 5837 people, whoever got the vaccine , no ebola virus was found during or after ten days of vaccination. On the contrary almost 23 cases of those who were not vaccinated were started to have the infection in 10 days. The trail was then led by WHO incollaboration with other international partners. This ring vaccine was choosen as the trial where people were vaccinated right after the case is detected and some were vaccinated after 3 weeks (Ploegh, 1998; Giesbert et al, 2002).

10. **Prevention and control**

People should avoid the contact of infected fruit bats and monkeys by covering the food properly Use proper gloves while handling the animals. Animal meat should be cleaned and thouroughly cooked before consumption. Human to human transmission should be stoped by avoiding the direct and close contact with the fluids of people having ebola virus symptoms. Doctors , nurses and medical staff should wear protective coverings and dresses while taking care of ill patients. People should take care of proper hygiene i.e. washing their hands after visitng hospitals and as well as while taking care of patients (Briand et al, 2014).

11. **Prognosis**

Agreeing fromWHO, the average ebola virus disease (EVD) case fatalness rate is about 50%. In the past, during other outbreaks, the rate has vacillated from 25% to as high as 90% (Scanchez et al, 1998; Ito et al, 1999).

12. **EBD outbreak in Pakistan**

WHO representative in pakistan Dr. Michel Theiren said that ‘’ ebola virus is a big threat, since ebola virus is spreading very fastly across world , Pakistan is at higher risk, though there is no ebola virus disease (EBD) effected patient in pakistan but the government should take steps toward this deadly disease. If a single patient affected form ebola virus disease (EBD) enters the country without screening, can cause the outbreak’’ (Xu et al, 1998; Khan and Ahmad, 2015).

13. **Conclusion**

Ebola virus is a very dangerous infection which causes viral haemorrhagic fever and it could be a reason of high mortality rate among the different countries. The virus is transmitted through wild life animals and also through human. There is no treatment discovered yet but a trailed vaccine have evaluated along with some therapies.

14. Recommendation

Prevention is better than cure, people should avoid every thing that can result in this fatal infection. As in Pakistan yet there is no ebola virus disease (EBD) outbreak , but we should not ignore it , as it can spread if any of the person having this disease enters the country or if we donot take care of the food we eat as it is a zoonotic disease. Government should spread awarness throughout the country and everyone should be serious about one's own health. We should keep our environment clean and hygeinic so that there could not be any single risk of this infectious disease in our country.

References

- Briand S, Bertherat E, Cox P, Formenty P, Kieny MP, Myhre JK ... and Dye C (2014). The international Ebola emergency. *New England Journal of Medicine*, 371(13), 1180-1183.
- Feldmann H, Nichol ST, Klenk HD, Peters CJ, and Sanchez A (1994). Characterization of filoviruses based on differences in structure and antigenicity of the virion glycoprotein. *Virology*, 199(2), 469-473.
- Garbutt M, Liebscher R, Wahl-Jensen V, Jones S, Möller P, Wagner R, ... and Ströher U (2004). Properties of replication-competent vesicular stomatitis virus vectors expressing glycoproteins of filoviruses and arenaviruses. *Journal of virology*, 78(10), 5458-5465.
- Geisbert TW, Pushko P, Anderson K, Smith, J, Davis KJ and Jahrling PB (2002). Evaluation in nonhuman primates of vaccines against Ebola virus. *Emerging infectious diseases*, 8(5), 503.
- Gupta M, Mahanty S, Bray M, Ahmed R and Rollin PE (2001). Passive transfer of antibodies protects immunocompetent and immunodeficient mice against lethal Ebola virus infection without complete inhibition of viral replication. *Journal of Virology*, 75(10), 4649-4654.
- Ito H, Watanabe S, Sanchez A, Whitt MA, and Kawaoka Y (1999). Mutational analysis of the putative fusion domain of Ebola virus glycoprotein. *Journal of virology*, 73(10), 8907-8912.
- Jahrling PB, Geisbert TW, Johnson ED, Peters CJ, Dalgard DW and Hall WC (1990). Preliminary report: isolation of Ebola virus from monkeys imported to USA. *The lancet*, 335(8688), 502-505.

Khan H and Ahmad I (2015). Threat of Ebola virus disease for Pakistan. Gomal Journal of Medical Sciences, 12(3).

Ploegh HL (1998). Viral strategies of immune evasion. Science, 280(5361), 248-253.

Prins KC, Cárdenas WB, and Basler CF (2009). Ebola virus protein VP35 impairs the function of interferon regulatory factor-activating kinases IKK ϵ and TBK-1. Journal of virology, 83(7), 3069-3077.

Rewar S and Mirdha D (2014). Transmission of Ebola virus disease: an overview. Annals of global health, 80(6), 444-451.

Rolison JJ and Hanoch Y (2015). Knowledge and risk perceptions of the Ebola virus in the United States. Preventive medicine reports, 2, 262-264.

Sanchez A, Trappier SG, Mahy BW, Peters CJ, and Nichol ST (1996). The virion glycoproteins of Ebola viruses are encoded in two reading frames and are expressed through transcriptional editing. Proceedings of the National Academy of Sciences, 93(8), 3602-3607.

Sanchez A, Yang ZY, Xu L, Nabel GJ, Crews T, and Peters CJ (1998). Biochemical analysis of the secreted and virion glycoproteins of Ebola virus. Journal of virology, 72(8), 6442-6447.

Schneider J, Gilbert SC, Blanchard TJ, Hanke T, Robson KJ, Hannan CM ... and Hill AV (1998). Enhanced immunogenicity for CD8⁺ T cell induction and complete protective efficacy of malaria DNA vaccination by boosting with modified vaccinia virus Ankara. Nature medicine, 4(4), 397.

Simmons G, Wool-Lewis RJ, Baribaud F, Netter RC and Bates P (2002). Ebola virus glycoproteins induce global surface protein down-modulation and loss of cell adherence. Journal of virology, 76(5), 2518-2528.

Ströher U, West E, Bugany H, Klenk HD, Schnittler HJ and Feldmann H (2001). Infection and activation of monocytes by Marburg and Ebola viruses. Journal of virology, 75(22), 11025-11033.

Sui J and Marasco WA (2002). Evidence against Ebola virus sGP binding to human neutrophils by a specific receptor. Virology, 303(1), 9-14.

Takada A, Fujioka K, Tsuiji M, Morikawa A, Higashi N, Ebihara H ... and Kawaoka Y (2004). Human macrophage C-type lectin specific for galactose and N-acetylgalactosamine promotes filovirus entry. Journal of virology, 78(6), 2943-2947.

Villinger F, Rollin PE, Brar SS, Chikkala NF, Winter J, Sundstrom JB ... and Peters CJ (1999). Markedly elevated levels of interferon (IFN)- γ , IFN- α , interleukin (IL)-2, IL-10, and tumor necrosis factor- α associated

with fatal Ebola virus infection. The Journal of infectious diseases, 179(Supplement_1), S188-S191.

Volchkov VE, Becker S, Volchkova VA, Temovoj VA, Kotov AN, Netesov SV and Klenk HD (1995). GP mRNA of Ebola Virus Is Edited by the Ebola Virus Polymerase and by T7 and Vaccinia Virus Polymerases. Virology, 214(2), 421-430.

Volchkov VE, Volchkova VA, Mühlberger E, Kolesnikova LV, Weik M, Dolnik O and Klenk HD (2001). Recovery of infectious Ebola virus from complementary DNA: RNA editing of the GP gene and viral cytotoxicity. Science, 291(5510), 1965-1969.

Xu L, Sanchez A, Yang ZY, Zaki SR, Nabel EG, Nichol ST, and Nabel GJ (1998). Immunization for Ebola virus infection. Nature medicine, 4(1), 37.

Zaki SR, Shieh WJ, Greer PW, Goldsmith CS, Ferebee T, Katshitshi J ... and Khan AS (1999). A novel immunohistochemical assay for the detection of Ebola virus in skin: implications for diagnosis, spread, and surveillance of Ebola hemorrhagic fever. The Journal of infectious diseases, 179(Supplement_1), S36-S47.