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Организатор: кафедра Патологической анатомии ТМА.

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MUNDARIJA – ОГЛАВЛЕНИЕ - CONTENTS

IMPLEMENTATION AND RESULTS OF THE SURVEY HELD ON THE PATHOLOGICAL ANATOMY OF THE EBOLA VIRUS

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INTRODUCTION.

The Ebola virus, the prototype of the Ebolavirus genus in the Filoviridae family of negative-sense single-stranded RNA viruses, was discovered in 1976 during the first documented outbreak of Ebola virus disease in Yambuku, northern Congo. The Ebola virus has since caused sporadic outbreaks of varying sizes in Equatorial African nations. In March 2014, an Ebola virus variant named Ebola Makona was detected in Guinea. This variant caused a 3-year epidemic affecting tens of thousands in multiple West African countries, collapsing healthcare systems. The Ebola Makona epidemic spread through both rural and urban areas, underscoring previously poorly understood features like sexual transmission and virus persistence after recovery.

Scientific and clinical knowledge of human Ebola disease before West Africa was very limited. Few human cases occurring in remote areas limited research, as did filovirus research confinement to high-security laboratories. Studies on Ebola pathophysiology have been hampered by a lack of susceptible small animal models with competent immunity. For example, mice are resistant to the non-adapted Ebola virus.

Previously, Ebola virus disease was described as acute hemorrhagic fever, earning its name Ebola hemorrhagic fever. Reported case fatality rates reached 90%. The disease was characterized by lymphopenia, disseminated intravascular coagulation, immunosuppression, and systemic inflammatory response resembling septic shock. While many observations stem from the West African outbreak, some hypotheses have been revised. Surprisingly, few human cases presented with bleeding, and bleeding did not correlate with severity. This changed the disease name from Ebola hemorrhagic fever to Ebola virus disease. Moreover, robust immune activation rather than immunosuppression correlates with Ebola, and virus persistence in fluids after recovery changed views of Ebola, prompting new research and policies. We aim to integrate novel findings within the current human Ebola model and discuss future directions.

While Ebola virus disease differences exist between viruses, this review focuses on Ebola disease caused by all known ebolaviruses pathogenic for humans. Most available data come from Ebola virus infections rather than the Sudan virus, Bundibugyo virus, and Taï Forest virus. Related Marburg viruses will be mentioned where data exists but, unfortunately, information on Marburg disease is still lacking.

RELEVANCE

- ✓ To assess whether the Ebola virus is prevalent among the public
- ✓ To examine the factors of sources people to know about the Ebola virus
- ✓ To determine the solution to the existing problem

PURPOSE

- ➢ To create awareness about the Ebola virus among young people
- ➢ To study the pathological anatomy of the Ebola virus

MATERIALS AND METHODS

This study used online E-library sources to obtain some of the statistical resources regarding the Ebola virus. The **search for literary sources** was carried out using the bibliographic databases Web of Science, Scopus, DBLP, and PubMed. When selecting sources, they paid attention to experimental articles, literary reviews, and the number of their citations over the past year.

RESULTS

The available data spanning four decades indicates that human infection with the Ebola virus primarily occurs through close contact with infected bodily fluids. This likely happens during both spillover events (e. . contact with infected blood while butchering bushmeat) and human-to-human transmission. There is no evidence direct contact with bats causes the Ebola virus to spill over into humans, but infection with the Marburg and Ravn viruses through direct or indirect contact with Egyptian fruit bats has been documented. Human visits to caves or mines where these bats roost have been directly linked to Marburg virus disease, strongly indicating mucosal or skin contact with bat droppings is enough to initiate Marburg virus infection in people. Except for the initial Ebola outbreaks in Zaire tied to substantial needle transmission, most data since the early 1990s suggest exposing skin and mucous membranes to the Ebola virus while conducting activities like washing bodies during traditional funerals or caring for sick relatives at home is enough for human-to-human transmission. Early data from lab exposure to the Sudan virus even suggest skin abrasions may not be needed for the virus to enter through the skin. This raises questions about how Ebola virus infection occurs in skin and mucous membranes, and which cells are involved in initial virus amplification.

Antigen-presenting cells are a potential initial target for Ebola virus infection. Research in animal disease models has indicated that dendritic cells and macrophages are early and preferred targets that support virus replication. Both cell types can be productively infected by the Ebola virus in vitro, and the virus prevents dendritic cell activation mainly through VP24 and VP35.

Much research over the last decade has focused on defining the development and specific functions of dendritic cell subsets in mice and humans. The emerging picture is that several subsets exist with overlapping and distinct functions, roughly classified as classical, plasmacytoid, and inflammatory dendritic cells in humans. Whether Ebola can equally infect different subsets remains unknown, but evidence suggests it cannot. For example, several C-type lectins on dendritic cell surfaces have been involved in Ebola virion attachment to target cells like DC-SIGN. However, dendritic cells of the epidermis and mucosal epithelium do not express these molecules. Initial Ebola virus infection may depend on attachment to target cells via TIM-1 and TIM-4, highly expressed in mucosal epithelia. Elucidating the initial steps by which Ebola establishes infection in a host is needed to understand how the virus spreads from the entry site and perhaps design countermeasures to prevent spread.

CONCLUSION

As an objective of this survey, this paper presented to elaborate on the Ebola virus to the public.

Ebolaviruses cause outbreaks of severe fevers that spread from person to person. Despite how dangerous the Ebola virus is, experts know very little about what happens inside the human body during Ebola virus disease (EVD) or the immune system responses that help people survive and develop immunity. This lack of knowledge likely comes from a lack of clinical and lab data from past outbreaks. The huge EVD outbreak in West Africa from 2013 to 2016 allowed researchers to study clinical, epidemiological, and immunological factors in many patients using modern lab tools. This review will summarize what researchers know about how the human body physically responds and the immune system reacts to infections with filoviruses like Ebola.