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Ebola Virus disease: New and emerging infectious diseases

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Abstract

Human-to-human transmission fuels sporadic epidemics of severe, systemic febrile disease caused by Ebola virus strains. Even though Ebola viruses, particularly Ebola virus (EBOV), are well-known agents of viral haemorrhagic fever and there is international concern about Ebola virus disease (EVD) outbreaks, very little is known about the human immune correlates of survival and immune memory and the pathophysiology of EVD in humans. The absence of clinical and laboratory data from previous outbreaks is probably to blame for this lack of fundamental understanding of EVD's physiological characteristics. For the first time, cutting-edge laboratory equipment has been used to evaluate clinical, epidemiological, and immunological parameters in a significant number of patients as a result of the unprecedented scale of the EVD epidemic that swept through West Africa from 2013 to 2016. Human pathophysiologic and immunologic responses to filovirus infection will be summarized in this literature review.

"An emerging disease is one that has appeared in a population for the first time, or that may have existed previously but is rapidly increasing in incidence or geographic range," states the World Health Organization (WHO). Emerging infections are defined as "infectious diseases whose incidence in humans has increased in the past two decades or threatens to increase in the near future" by the Centers for Disease Control and Prevention (CDC). These diseases, which do not discriminate based on nationality, include: new infections brought on by changes or evolution in organisms that are already present; known infections reemerging as a result of antimicrobial resistance in known agents or breakdowns in public health measures, as well as previously unrecognized infections appearing in areas undergoing ecological transformation or spreading to new geographic areas or populations.

Keywords: Ebola virus genus; Ebola Viruses; Immune memory; Human transmission; Haemorrhagic fever

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Introduction

The prototype of the Ebola virus genus, Ebola virus (EBOV), belongs to the Filoviridae family of negative-sense, single-stranded RNA viruses. The Ebola virus (EBOV) was first discovered in 1976 during the first known outbreak of the Ebola virus disease (EVD) in the town of Yambuku in northern Zaire, now the Democratic Republic of the Congo [1]. Since then, EBOV has caused sporadic outbreaks of varying degrees of human disease in Equatorial African nations. Guinea was the first location where an EBOV variant known as EBOV Makona was found in March 2014. This variant was the cause of an epidemic that lasted three years and affected tens of thousands of people in several West African nations, destroying three of their healthcare systems. EBOV Makona spread throughout both urban and rural areas, highlighting previously unknown aspects of EVD, such as sexual transmission and virus persistence following recovery [2]. Prior to its appearance in West Africa, human EVD was poorly understood

from a scientific and clinical standpoint. Research was constrained to biosafety level 4 containment laboratories due to the lack of human cases and their prevalence in rural areas of Equatorial Africa. Additionally, the absence of immune-competent small animal models that are susceptible to EVD has hampered fundamental research into the disease's pathophysiology. No adapted EBOV, for instance, is completely resistant to laboratory mice, a prevalent disease model [3].

Prior to 2014, EVD was known as Ebola haemorrhagic fever (EHF), as it was referred to as an acute haemorrhagic fever. Cases had been found to have fatality rates of up to 90%. Lymphopenia, disseminated intravascular coagulation (DIC), immunosuppression, and a systemic inflammatory response resembling septic shock were the hallmarks of the condition. While the findings from the West African EVD outbreak have strengthened many of these observations, some of the previous hypotheses have been revised. The lack of correlation between bleeding and disease severity and the overall low number of human cases presenting with bleeding may have been one of the most surprising findings [4]. The disease's name was changed from Ebola haemorrhagic fever to Ebola virus disease as a result of these findings. In addition, the fact that EVD is associated with robust immune activation rather than immunosuppression, as well as the virus's capacity to persist in multiple bodily fluids long after recovery, has altered our current understanding of the disease and prompted new research directions and public health policies. In this paper, we will discuss future research directions and attempt to incorporate these novel findings into the current human EVD model [5].

Portals of Ebola virus

Over the past forty years, epidemiological data have shown that close contact with infected body fluids is the most common way for humans to become infected with EBOV. This most likely occurs during both human-to-human transmission and spillover events. Although infection with MARV and RAVV has been documented through direct or indirect contact with Egyptian rousettes, there is no evidence that direct contact with bats causes EBOV spillover into humans [6]. Mucosal or skin contact with bat droppings is sufficient to initiate MARV infection in humans, as human visits to caves or mines where these bats roost have been directly linked to the development of MVD [7].

The majority of data since the beginning of the 1990s suggest that exposing skin and mucosae to EBOV while performing activities like body washing during traditional funerals or caring for sick relatives in the household is sufficient for human-to-human transmission of EBOV, with the exception of the first EVD outbreaks in Zaire, which were linked to substantial percutaneous needle transmission (Ebola haemorrhagic fever in Zaire 1976–1978) [8]. These outbreaks were the only cases in which the transmission Abrasions of the skin may not be necessary for Ebola virus entry through the skin, according to early laboratory data. These results raise questions about how and which cells are involved in the primary amplification of the Ebola virus in skin and mucosae.

Epidemiology and transmission

A review of CRE's biology, epidemiology, and management has been done. Newer antibiotic treatments for CRE have been discussed in recent articles. The mean duration of colonic carriage in hospitalized CRE colonized patients was greater than one year, according to follow-up.100 However, HCP are rarely, if ever, colonized. None of the health care providers evaluated in a study of fecal carriage among HCP in a hospital endemic for CRE were colonized [9].

The human gut is the primary reservoir for CRE infections. The majority of transmission mechanisms involve direct and indirect contact between people. Endoscopes, particularly duodenoscopes, have been contaminated, causing numerous outbreaks in hospitals. These outbreaks have occurred despite the fact that endoscopes have been thoroughly cleaned and disinfected in accordance with current guidelines. A review of approaches to providing endoscopes free of pathogens has been done. It has been demonstrated that the hospital's water sources, particularly sinks, serve as reservoirs for CRE [10]. A review of CRE elimination strategies and interventions' success rates has been conducted.

Conclusion

The sensitivity of C auris to germicides has been the subject of several reviews. Numerous researchers have investigated the vulnerability of C auris to germicides. Evaluated the germicidal potential of chemical sterilants and/or high-level disinfectants and found that, with the exception of 0.55 present ortho-phthalaldehyde, which only inactivated E. coli by 2.3 log10, all of the agents achieved a C auris reduction of less than 4.1 log10 importantly; these in vitro experiments were carried out in difficult circumstances. When used appropriately, it is likely that all high-level disinfectants currently approved by the FDA are effective against C. auris.

Pathogens can be transmitted vertically via breastfeeding, transplacental, or other routes. By sampling the placenta, researchers can examine disease processes in living patients and learn more about how vertical transmission works. SUDV or BDBV antigen was found in fetal trophoblastic cells in this study, indicating that these viruses can infect and possibly cross the placental epithelial barrier, causing the fetus to be infected transplacental. EBOV infection of the fetus transplacental has previously been documented in stillbirths through PCR analysis of fetal swabs, blood, and amniotic fluid. Even after the virus has been removed from the mother's blood, the virus may persist in these cases due to the placenta's immunoprotective function. Some herpes viruses, the human immunodeficiency virus, the Zika virus, treponema, and toxoplasma viruses are among the human pathogens that are able to successfully cross the placental barrier and infect the fetus. The trophoblastic, which is the primary cellular barrier against fetal infection, is made up of two main types: the extra villous trophoblastic, which invades the maternal decidua and directly contacts maternal cells, such as lymphocytes and decidua stromal cells, and the villous trophoblastic, which is directly exposed to maternal blood. Immunohistochemical analysis revealed Ebola virus antigen in both the extra villous

trophoblastic and the syncytiotrophoblast in this study.

Mammalian species differ significantly in the cellular structure of placentas, as does the degree to which fetal trophoblasts are exposed to maternal blood. The likelihood of pathogen vertical transmission and/or placental tropism in natural versus incidental hosts may be affected by these structural differences. To fully comprehend the pathogenesis of EVD in pregnant women and their offspring and ultimately to develop methods for infection prevention or treatment, future sampling of placental tissue is required. In addition, future research into the interaction and clinical outcomes associated with this confection ought to be a top priority due to the fact that EBOV-malaria parasite confections were prevalent during the 2013-2016 West African outbreaks and the extremely high rates of malaria in many areas where filo virus outbreaks occur.

Acknowledgement

None

Conflict of Interest

None

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