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Biological and Immunological Aspects of Emerging and Re-emerging Avian Influenza and Ebola Diseases

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Abstract

The countries located in west Asia and other parts of Asia are positioned in important and strategic areas due to specific political and geopolitical conditions. These countries have faced various military and political threats, and it is crucial for them to strengthen their ability to foresee, prevent, and prepare for any biological threats. Meanwhile, with the progress made in science and technology, particularly in the field of biology, new biological threats and wars have surfaced. These threats and wars are self-replicating and can significantly expand the contaminated area, requiring advanced equipment and resources in the community in order to combat them effectively. The field of threat detection, cleanup, and contamination has been identified as an area of concern. Additionally, the hidden use of these factors in peacetime poses a potential danger, as does the possibility of deliberate casualties and damages, along with the incidence of intense fear and panic in the target population. Given the recent outbreak of the avian influenza virus in some countries, there is a concern that avian influenza virus outbreaks could be a precursor to a more widespread and severe biological attack, such as the Ebola virus, which presents with widespread biological and clinical symptoms.

Keywords: Avian Influenza, Ebola, Hemorrhagic Fever, Surface Water-Borne Viruses

1. Background

The global perception of biological and chemical weapons as potential threats was greatly influenced during World War I. Some theories suggest that the 1918 Spanish Influenza was the outcome of German biological warfare, with suspicions that the disease was spread intentionally through product contamination, possibly using aspirin produced by the German pharmaceutical company, Bayer.[1] After World War I, in 1925, the Geneva Protocol was established. Its purpose was to prohibit the use of poisonous gases, bacteriological methods, and asphyxiating substances in warfare. Although many countries reserved the right to respond to biological warfare agent use, the treaty did not address issues related to research, development, pathogen production, and storage.[2] In March 1995, a chemical attack using sarin gas by Aum Shinrikyo occurred in the Tokyo subway. Investigation of this cult's activities revealed attempts to carry out other attacks using pathogens like spores, anthrax, cholera, biological toxins, and chemical agents. Numerous historical instances demonstrate the utilization of bioterrorism and biological agents by some countries during wars.[3] It is suggested that some countries, including Russia, the United States, the United Kingdom, France, Japan, and Canada, have been conducting research in this field for many years and are likely to make use of it. The countries situated in Asia are geopolitically and politically significant owing to their strategic and sensitive location.[4] As technology advances, nations are prioritizing access to valuable data, both military and civilian, and addressing political conflicts that have become institutionalized in their objectives. However, this has also led to an increasing need to develop prevention and preparedness capabilities against potential threats from transnational entities and adversaries. Evidence has shown that transgressive forces try to undermine a nation's will and national strength before targeting vital centers. In a meeting that lasted for 4-5 months in Geneva, experts from the World Health Organization (WHO) and the International Bureau of Animal Disease Epidemic jointly defined emerging common diseases in their report. An emerging common disease is one that has recently gained prominence or has recently seen increased prevalence and geographic expansion, affecting hosts and carriers, even though it may have existed before.[5] There have been recent reports suggesting that certain infectious agents that cause

diseases may undergo changes, making it easier for them to spread from person to person.[6] One of these infectious agents is avian influenza, a respiratory viral disease that affects both domestic and wild birds. It is caused by a type of influenza virus in the orthomyxoviridae family.[7] Antigenic changes in the avian influenza virus can lead to global epidemics and pandemics. Influenza viruses are categorized into various subtypes based on genetic and antigenic differences in their surface glycoproteins.[8] So far, two subtypes have been identified based on hemagglutinin and three subtypes based on neuraminidase, all of which have originated from birds. Some of these subtypes have been found to infect humans in different communities.[9] Seroepidemiological studies have shown that bird-specific subtypes such as H_5N_1 and H_9N_2 can spread among humans.[10] The H_5N_1 avian influenza virus was first identified in Hong Kong. It is highly pathogenic in humans and can cause fatalities.[11] In South China, the H_9N_2 subtype was first isolated from throat swabs of patients displaying influenza symptoms,[12] and it also appeared in Hong Kong in subsequent years.[11] Reports suggest that the avian influenza virus is prevalent in industrial poultry and can potentially transmit to humans.[13] The Ebola virus was first discovered in 1976 during an outbreak in southern Sudan and northern Zaire (now the Democratic Republic of the Congo) when it was isolated from patients living near the Ebola River. This virus is a significant cause of hemorrhagic fever and poses a significant threat due to its high mortality rate, lack of specific therapies, and effective vaccines. Ebola virus and Marburg virus are both part of the filoviridae family and are classified as Class A bioterrorism agents, similar to diseases such as plague, smallpox, and anthrax.[14] Recent outbreaks of diseases like the avian influenza virus have highlighted the importance of understanding the ecological context of biomedical attacks. Any such attack would require a thorough understanding of the relevant ecological factors. It is also important to consider the potential impact of more deadly viruses, such as Ebola, which have many biological and clinical similarities to avian influenza. Ultimately, a comprehensive understanding of these factors is necessary to prevent and respond to potential biomedical attacks effectively.[15] In this scenario, two of the prominent reasons we have presented are the influenza and Ebola diseases (Table 1).

Table 1. Reasons for Examining these Two Viruses

1	The similarities of the clinical symptoms of two diseases
2	The similarities of Ebola's clinical symptoms with other viral diseases of hemorrhagic fever
3	Rapid transmission of two influenza and Ebola viruses between animals and humans
4	Environmental and geographical conditions in most countries where there is drought
5	Stability of covered viruses such as influenza and Ebola at a relative humidity below 5%
6	Both are zoonotic viruses (common human-animal disease)
7	Both viruses infect surface water.[16,17]

2. Objectives

The purpose of the study was to prevent a wide-scale biological attack, such as Ebola, by providing a detailed report. Additionally, since outbreaks of acute bird flu are found in many countries, knowledge about the virus's ecology and how it spreads in the region could serve as a basis for future biological attacks by the adversary, such as Ebola.

3. Methods

For this study, we included all articles published between 2000 and 2019 in both Farsi and English that contained keywords related to the Ebola virus, avian influenza, viral hemorrhagic fevers, rapid transmission of Ebola virus and avian influenza between humans and other organisms, common human-animal diseases, and surface water-borne viruses. Initially, we screened the titles of all relevant articles to remove unrelated and duplicate articles. Then, we reviewed the abstracts of the remaining articles and thoroughly examined and studied the original articles.

4. Results

4.1. Ebola Virus, Group A

The Ebola virus (EBOV) belongs to the Ebolavirus genus within the Filoviridae family, closely related to the Marburg virus. [18] EBOV is characterized by a single-stranded negative-sense ~19-kb linear RNA, and it poses a significant threat to human health, causing Ebola hemorrhagic fever, one of the deadliest and most serious diseases known to mankind (Figure 1).[19] There are five known strains of the Ebola virus, each named after the location where they were first discovered.[20] The Zaire Ebola (ZEBOV) strain was first identified near the Ebola River Valley (Yambuku) in Zaire (now the Democratic Republic of the Congo)[21] in 1977, resulting in an 88% mortality rate among infected patients. The Ebola Sudan (SEBOV) strain was found in 1976 and has a mortality rate of 53%.[22] The Ivory Coast Ebola (CIEBOV) strain was identified in 1994 but didn't result in deaths.[23] The Ebola Bundibugyo (BEBOV) strain emerged during an outbreak in Bundibugyo, Uganda, between November 2007 and February 2008, causing a 25% mortality rate.[24] The Ebola Reston (REBOV) strain was discovered in Reston, Virginia, in imported primates (Cynomolgus monkeys) from the Philippines in 1989-1990. REBOV infects humans without causing disease or death, in contrast

to the other four strains, which lead to hemorrhagic fever.[25] The exact origin of the Ebola virus is unknown, but it is believed to be a zoonotic virus native to Africa.[26] Evidence suggests that interactions with primates (such as monkeys, chimpanzees, and gorillas), forest antelopes (in the case of Ivory Coast), pigs, horses, and fruit bats may contribute to disease transmission.[27] There is no evidence of gender or race-based susceptibility to Ebola Virus Disease (EVD), but it appears to disproportionately affect adults compared to children and individuals under 17 years of age.[28] During the outbreak of the disease in the Democratic Republic of the Congo (DRC) in 1995, 27 (8.6%) of the 315 cases were children, although approximately 50% of the DRC population was less than 17 years old at the time. Exact evidence for age selection is not available at present, however, it is well known that hemorrhagic fever selectively affects adults more than children.[29] The Ebola virus has an approximate diameter of 80 nm and is enveloped by a host cell-derived phospholipid membrane. Its genome is about 18.9 kb and consists of a single-stranded, non-segmented, and negative-sense RNA molecule. The genome accounts for about 1.1% of the total virion volume, with a mass of approximately 0.4×10^6 Da.[30]

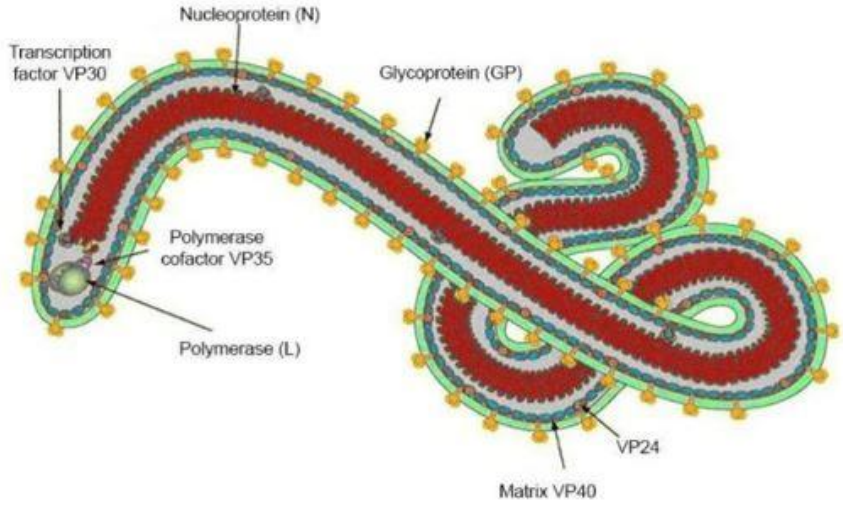

Figure 1. The Structure of the Ebola Virus

4.2. Ebola Virus Immunology, Infection, and Replication

The EBOV infections primarily target mononuclear phagocytes, including macrophages, monocytes, dendritic cells, Kupffer cells, and other immune cells involved in antigen presentation.[31] Endothelial cells also serve as a secondary target for EBOV infection. Currently, the exact mechanism of viral replication and transcription remains unknown due to the virus's high infection rate and hazardous nature.[32] However, it is believed that EBOV employs a mechanism similar to other negative-sense RNA viruses for transcription, translation, and replication. For instance, it is currently hypothesized that once inside the host cell, EBOV undergoes transcription to produce a polyadenylated genomic messenger RNA (mRNA). This mRNA comprises specific genetic sequences, including '3 leader,' Nucleoprotein (NP), viral proteins (VP35), VP40, glycoprotein (GP), VP30, VP24, and polymerase protein (L). Following transcription and translation, seven structural polypeptides are produced. Among these, VP35, VP30, NP, and protein L are associated with the virus's genomic RNA within a ribonucleoprotein complex involved in transcription and viral replication. VP24 and VP40 matrix proteins are associated with this complex and play a role in nucleocapsid formation.[33,34] The VP30 is presumed to act as a transcriptional activation factor and is essential for the EBOV replication cycle. It has been reported that 80% of GP gene transcription products encode a non-structural soluble glycoprotein precursor known as Pre-SGP.[35] Subsequently, post-translational cleavage of Pre-SGP occurs through enzymatic activity, resulting in the secretion of glycoproteins (SGPs), delta-peptides (Δ-peptides), triglyceride protein 2, and glycoprotein 1, GP1,2. Additionally, a recently discovered soluble secretory glycoprotein (ssGP) is produced. After production, sGP, Δ-peptide, and ssGP are systematically secreted in infected individuals and can be detected in blood samples from EBOV-infected individuals.[36,37] As of now, the exact function of sGP remains unknown, although various hypotheses have been proposed. For instance, Kinderelski et al. (2000) demonstrated that sGP interacts with neutrophils by binding to the neutrophilic membrane CD/6b receptor, which is a neutrophil-specific type III FC-γ receptor involved in interacting with the FC domain in HCG and modulating its function.[38]

However, some researchers have contested this notion, suggesting that sGP might function as a decoy when released during an EBOV infection. For instance, sGP shares a neutralizing epitope with GP1 and GP2 transcripts,

and it may bind to circulating neutralizing antibodies, diverting the immune response and thereby evading immune surveillance.[39] Studies have also indicated that sGP could act as a mediator in the activation of target cells and contribute to increased endothelial permeability, which can lead to hemorrhage and shock.[40] It's important to note that the U.S. Biological Weapons Program has imposed restrictions on the production of biological weapons, which include known vaccines and antibiotic treatments. However, the former Soviet Union specifically targeted biological agents for which there were no known cures and produced significant quantities of the Ebola virus in aerosol form until 1992.[41] As reported by Albic, the Ebola virus was categorized as N2 by the Soviet Biological Weapons Program and was stored in various facilities, including the Ultra-pure biopreparations institute in Lenin Grad, Omotninsk, Cairo, Lubolensk (outside Moscow), the Lyubuchany Immunology Institute in Chekhov (near Moscow), and the Vector Testing and Research Center in Novosibirsk (a small town called Colt Su) in Siberia.[42] In 1993, an extremist group attempted unsuccessfully to obtain the Ebola virus in Zaire as part of a biological weapon program aiming for a global devastation. Due to the significant potential for Ebola transmission, its associated disease and mortality, the CDC has classified Ebola as a Group A biological agent, signifying its high level of biological security concern.[43]

4.3. Symptoms of Ebola Virus Disease

EBOV is transmitted through exposure to contaminants from infected individuals, including blood, body fluids, excretions, vomit, breast milk, urine, semen, and even organs. For instance, research has indicated that EBOV can persist in semen for approximately 70 to 90 days after infection.[44] Close contact with a patient, whether deceased or alive, has frequently been linked to infection, especially during traditional burial ceremonies associated with EVD. Family members who wash and prepare the deceased for burial are particularly at risk.[45] Furthermore, evidence suggests that indirect transmission can occur through coughing or sneezing. Direct exposure to bedding, clothing, or other inanimate objects previously in contact with an infected person, especially after death, can also lead to disease transmission.[46] Numerous infection cases have been reported among individuals who fail to follow proper disinfection procedures after contact with an EVD patient. This highlights the importance of adhering to infection control measures, particularly for healthcare workers at outbreak sites.[47] EBOV has a wide range of cellular targets, including mononuclear phagocytic cells, endothelial cells of the respiratory and digestive tracts,[48] hepatocytes, fibroblast cells, and parenchymal cells in the liver, spleen, and lymphoid tissues, leading to extensive necrosis.[49]

Besides the rapid proliferation of EBOV in mononuclear phagocytic cells and endothelial cells, the virus triggers the release of various pro-inflammatory cytokines, such as RANTES, monocyte chemotactic protein-1, macrophage inflammatory protein α-1, tumor necrosis factor-α, interleukin-δ, interleukin-8, and growth-oncogenic α.[50] Notably, the Ebola virus inhibits the secretion of interferon-α, a crucial immune regulator and antiviral cytokine, in infected cells.[51] Following the entry of EBOV into the system, there is an incubation period of approximately 4 to 10 days (as stated in the WHO leaflet number 103, the range can extend up to 21 days) before the first symptoms of EVD become apparent.[51] The EVD exacerbations include symptoms such as flu-like symptoms (approximately 40 °C or 104 °F), chills, restlessness, muscle and joint pain, fatigue, severe insomnia, and general muscle weakness. Patients may experience an imbalance and a sore throat, especially when swallowing. Following these initial symptoms, they may suffer from lethargy, nausea, vomiting, diarrhea, excessive fatigue, asthma, anorexia, and pain.[52] A maculopapular rash typically emerges first in the lateral regions of the trunk, groin, and armpits, rapidly spreading throughout the body except for the face.[53] Figure 2 shows the symptoms of the disease.

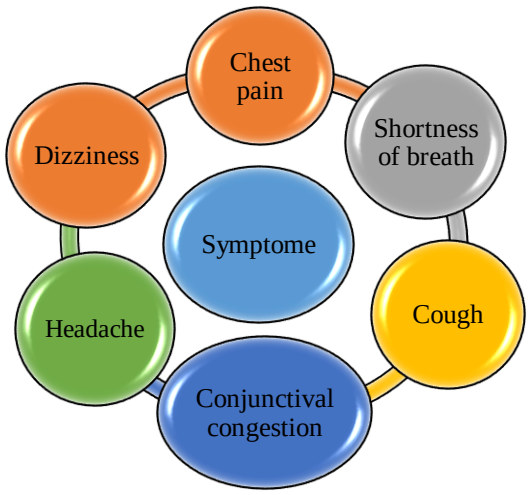

Figure 2. The Symptoms of the Disease.

Subsequently, purpura and hemorrhagic manifestations may develop, including petechiae, ecchymosis, uncontrolled bleeding from venous injection sites, nasal bleeding, hematuria, and melena. Thrombocytopenia, lymphopenia, and leukopenia are also common.[54] Secondary symptoms may include edema, hypotension, hypovolemia, tachycardia, and abnormal renal and hepatic function, affecting thymocytes and hepatocytes.[55] In a few days following infection, patients may exhibit neutrophilia and increased concentrations of aspartate aminotransferase and alanine aminotransferase. In the later stages of infection, most patients experience external hemorrhage from body orifices, such as the nose and mouth.[56] This hemorrhage results from the formation of numerous small blood clots in the body, clinically referred to as Disseminated

Intravascular Coagulation (DIC). Tissues located where these clots are found may die due to a lack of nutrients and oxygen. [57] In an attempt to counteract the clots, the body releases substantial amounts of plasmin and tissue factors into the bloodstream, leading to uncontrollable bleeding in various parts of the body.[58] During the final stages of EVD, progressive symptoms like myocardin and pulmonary edema are common. Ultimately, patients may go into shock, experience severe metabolic syndrome, and exhibit a peculiar facies expression without any apparent movement.[59] Most patients, often in the second week of the illness, succumb to the disease due to tachypnea (increased respiration), hypotension, anuria, and, frequently, coma.[60] It is believed that the Ebola virus can cross the placental barrier and infect the fetus. Evidence has shown that in 15 cases of pregnant women with EVD, nearly all experienced miscarriages, with only one giving birth at the time of infection. Unfortunately, this newborn also succumbed to a severe fever just three days after birth.[61]

4.4. Bird's Influenza

The influenza virus is an RNA virus belonging to the Orthomyxoviridae family. It is categorized into three subtypes: A, B, and C, based on the central protein of the virus. Type B influenza primarily affects humans and typically leads to limited epidemics every two to four years.[62] Type C influenza also affects humans and pigs but usually results in mild clinical symptoms with minimal clinical signs. In contrast, Influenza A is a highly prevalent virus.[63] It has the capability to infect various mammal and avian species and is more virulent compared to Influenza B and C.[64] Influenza A virus possesses a glycoprotein envelope housing two key antigens: hemagglutinin (H) and neuraminidase (N).[65] Both humans and animals generally produce antibodies against these antigens. Influenza A can be classified into 16 hemagglutinin (H_1-$H1_6$) and nine neuraminidase (N_1-N_9) subtypes, each identified by a unique combination, host type, year, and geographic region of the first virus isolation, such as H_5N_1 avian influenza in Hong Kong in 1997.[66,67] Among these strains, all 16 hemagglutinin types can cause the disease in birds. The influenza virus genome is fragmented, consisting of eight segments, making genetic changes during replication highly likely. Recombination, especially involving influenza A, is more common than the other influenza types and can affect either N, H, or both antigens, though H antigenic changes are more frequent than N changes.[68] Minor changes due to point mutations in the RNA coding sequence can result in alterations of one or more amino acids in the 69-amino acid hemagglutinin structure, leading to changes in the virus's antigenicity. Every few years, influenza A acquires new antigens, with some shifts being more pronounced due to genetic mutations that can be transmitted between different animal and human species.[69,70] Antigenic shifts occur when the genomes of two different human and avian influenza viruses merge and create a new virus with increased pathogenicity. Such viruses can be more virulent, spread rapidly, and result in a pandemic, as individuals lack prior exposure to the new strain.[71] In terms of pathogenicity, influenza viruses can be categorized as non-pathogenic, low-pathogenic, or high-pathogenic. Avian influenza viruses with hemagglutinin subtypes H_1 to H_4 are considered highly pathogenic (Figure 3).[72]

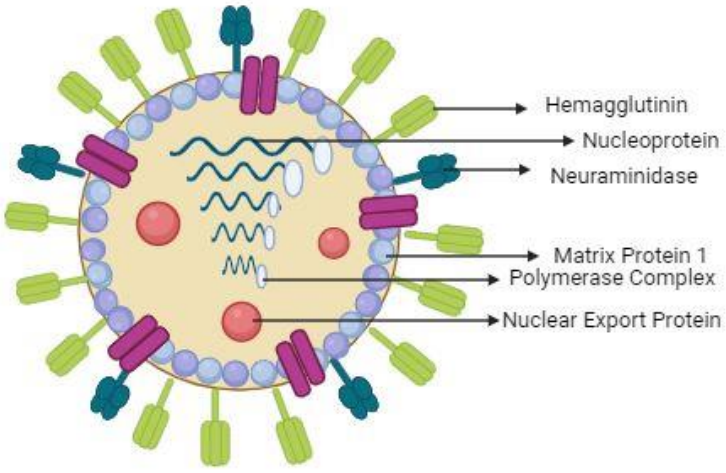

Figure 3. The Structure of Avian Influenza Virus.

4.5. Pathogenesis of Avian Influenza

Both H_7 and H_5 avian influenza strains have the potential to be highly pathogenic, and virulence in the avian influenza virus appears to be strongly associated with these specific hemagglutinin subtypes.[73] This shift in virulence significantly impacts the host's response to the virus, with more virulent strains often inducing a stronger inflammatory response.[74] Post-mortem pathological examinations reveal severe histopathological changes in the pulmonary cord and widespread alveolar destruction.

These changes include alveolar spaces being filled with fibrinoid exudates and red blood cells, as well as the formation of hyaline membranes, vascular congestion, and lymphocytic infiltration in the interstitial and proliferative spaces.[75, 76] Additionally, hemophagocytosis is observed in lung biopsies from patients. During autopsies, a notable reduction in the number of lymphocytes with the presence of atypical lymphocytes is observed in these patients, along with lobular center necrosis in the liver.[77]

4.6. Clinical Signs and Differential Diagnosis of Ebola Virus and Avian Influenza

Distinguishing EBOV infection from other viral diseases, such as avian influenza, can be challenging. In the early stages of the EBOV infection, it shares symptoms with diseases like Lassa fever, malaria, and typhoid fever, making initial diagnosis difficult.[78] Physicians can differentiate EVD by looking for more severe symptoms such as sore throat, pharyngitis, and edema in the later stages. Hemorrhagic symptoms of EVD, except for mucosal bleeding, can be identified through widespread clotting issues and uncontrolled bleeding throughout the body.[79] It is also challenging to differentiate hemorrhagic complications from other Viral Hemorrhagic Fevers (VHFs) from EVD. However, EVD is distinct in its presentation, including features like helplessness and weight loss. Maculopapular rashes are typically only seen in dengue virus infection and not in other VHFs.[80] Accurate diagnosis of VHF requires laboratory tests and confirmation.[81] Clinical symptoms are commonly described in patients admitted to the hospital. Therefore, patients with atypical clinical symptoms or those not requiring hospitalization, such as encephalopathy and gastroenteritis, have not been well-documented.[82] It is generally believed that the disease primarily affects children, adolescents, and young adults with no underlying health conditions. Common clinical signs include fever, excessive tearing, runny nose, shortness of breath, conjunctivitis, muscle pain, cough, headache, sore throat, nausea, vomiting, and, in severe cases, worsened symptoms.[83] Given the high prevalence of the disease, healthcare personnel must strictly adhere to health protocols to prevent its spread. This includes wearing protective clothing, gloves, and boots that are properly disposed of after use.[84] Table 2 shows a comparison of clinical signs between Ebola and avian influenza viruses.

Table 2. Comparison of Clinical Symptoms between Ebola and Avian Influenza Viruses

Symptom	Ebola	Avian influenza	Symptom	Ebola	Avian influenza
High fever and chills	+++*	+++	Excessive fatigue	+++	+++
Frailty	+++	+++	Nervous headache	+++	+++
Muscular pain	+++	+++	The lack of balance	+++	++
Joint's pain	+++	+++	Sore throat when swallowing	+++	+++
Helplessness	+++	+++	Nope	+++	+++
Nausea	+++	+++	Abdominal pain	+++	+++
Nausea and Vomiting	+++	+++	Maculopapular rash	+++	+
Diarrhea	+++	+++	Chest pain	+++	++
Fatigue	+++	+++	Shortness of breath	+++	++
Anorexia	+++	+++	Cough	+++	+++
Congenital Congestion	+++	+++	Hemorrhagic petechiae	+++	-
Headache	+++	+++	Hemorrhagic ecchymosis	+++	-
Confused	+++	++	Bleeding	+++	-
Sudden attack	+++	+	nose bleeding	+++	++
Purpura	+++	+	Hematuria	+++	-
Melena	+++	-	edema	+++	-
External bleeding	+++	-	Low blood pressure	++	++
Thrombocytopenia	+++	-	Hypovolemia	++	-
Lymphopenia	+++	-	Tachycardia	++	-
Leukopenia	+++	-	Renal impairment	++	-
Liver disorder	++	-	Tachypnea	++	-
Shock	++	-	Hypotension Mode	++	-
Seizure	++	+	Coma	++	-
Metabolic disorders	++	+	Anuric mode	++	+

5. Discussion

In this study, we examined the Ebola virus and influenza viruses. The Ebola virus is known to cause Ebola disease, and there are both similarities and differences between it and influenza viruses. We also discussed the importance of accurately diagnosing the Ebola virus in the early stages of the disease to delay the initiation of treatment. This is crucial because until the disease is confirmed, it may be transmitted to others. We also emphasized the importance of preventing the transmission of viruses through direct contact with patients and protecting healthcare personnel in healthcare facilities, particularly for influenza viruses. Generally, the topics we discussed in this section demonstrate that diagnosing, preventing,

and treating viruses are vital for improving public health and preventing the spread of diseases. To enhance further knowledge and progress in the diagnosis and treatment of these types of diseases, carrying out more research in virology and virus transmission prevention is essential. Additionally, public education and awareness about methods of virus prevention and care play a significant role in the community.

6. Conclusion

This study recommends that suspected patients of both Ebola and other viruses should be placed in quarantine. Nurses caring for these patients should take necessary precautions to limit contact and rigorously control the handling of the patient's bodily secretions. All nursing procedures and invasive procedures, such as catheter placement, body secretion sampling, venipuncture, and suctioning, should be carried out with the utmost care and scrutiny. Healthcare workers and hospital staff in contact with patients must wear appropriate gloves, masks, and protective gear. Close monitoring should also extend to family members and others who have been in contact with such patients. This monitoring regimen should include daily body temperature measurements, hospitalization, and quarantine for anyone suspected of having the disease, particularly after fever onset. Preventing Ebola hemorrhagic fever is particularly challenging in Africa due to the unknown identity and whereabouts of the Ebola reservoir animals. However, a few fundamental measures have been suggested to mitigate the risk. An essential prevention principle is to avoid direct contact with infected individuals. Healthcare center staff should exercise extreme caution, wearing full protective gear, including masks, gloves, and goggles, to prevent contact with blood and bodily fluids. In the event of a patient's death, close contact with the deceased body should be avoided. As previously mentioned, the early symptoms of Ebola are similar to common diseases, making early detection challenging. When individuals exhibit early Ebola symptoms and doctors have suspicions, quarantine measures should be implemented. During this time, public health professionals can confirm the presence of the disease through laboratory tests. Given the high prevalence of the Ebola virus and its clinical symptom similarity to influenza, it is crucial to differentiate between these two diseases. A person presenting flu-like symptoms could potentially carry the Ebola virus; thus, early diagnosis and isolation are vital to prevent further transmission in healthcare settings.

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Author Contributions

NNSh and SHB, designed and performed the study. NNSh, SHB contributed to writing the manuscript.

Research Highlights

What Is Already Known?

Early symptoms of these viruses can mimic other diseases, making accurate and rapid diagnosis critical. Infection control practices in healthcare settings and public health awareness are crucial to manage outbreaks.

What Does This Study Add?

This study underscores the importance of early detection, accurate differentiation, and laboratory testing to confirm diagnoses. The research highlights the significance of effective infection control measures to limit the spread of these viruses, emphasizing the need for healthcare personnel to take strict precautions when handling patients and their bodily secretions. Overall, this study provides valuable insights into the diagnosis, prevention, and management of Ebola and influenza viruses, emphasizing the need for a proactive approach in tackling these viral threats.

Conflict of Interest Disclosures

All authors declared that they have no conflict of interest.

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