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Article Review

Rift Valley Fever threaten the animals and human health

Sahar N. Mohamady[*], Hend, M. Megahed[**], Dalia T. Mohamed[**] and Abd-Allah S. Hamed[*]

Clinical Pathology& Pathology Department[*], Biochemical department[**], Animal Health Research Institute, (AHRI) Zagazig Branch, Agriculture Research Center (ARC), Egypt.

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ABSTRACT

Rift Valley fever (RVF) is a zoonotic disease primarily transmitted by mosquito species such as *Aedes* and *Culex*. Humans can also become infected through direct contact with the blood or organs of infected animals. The disease leads to substantial economic losses, largely due to livestock deaths and abortions associated with RVF infections. The Rift Valley fever virus (RVFV), part of the Phlebovirus genus and the Bunyaviridae family, is the causative agent of the disease. The most severe cases are observed in sheep, goats, newborns, and young animals, as they are particularly vulnerable to infection. The clinical symptoms comprise elevated temperature, nasal fluid secretion, and eye discharge, lymph node inflammation, and vomiting. Diagnosis depends on recognizing particular IgM or IgG antibodies, identifying RVFV nucleic acids, conducting virus isolation, and performing histopathological analysis. Vaccines for animals consist of inactivated and weakened vaccines derived from highly virulent isolates of RVFV. With global climate change, competent vectors are becoming more widely distributed in non-endemic areas, and RVF may spread across national borders. RVF can be controlled by vaccinating livestock, restricting the movement of livestock during epidemics, and destroying vector and vector mosquito habitats.

INTRODUCTION:

Rift Valley fever (RVF) is considered one of the most important pathogens in Africa. It is a zoonotic, vector-borne infectious disease, and classified as a haemorrhagic fever **(Daubney &Hudson 1932, Coetzer &Tustin 2005)**.

RVF is causing financial losses in affected areas, with elevated rates of mortality and miscarriage. RVFV infects bovine, ovines, caprines, and dromedaries, where young creatures suffer significantly more than adults. RVFV primarily impacts animals but can also lead to illness in humans. Mosquitoes (especially the *Aedes* and *Culex*) carry the virus. Humans generally acquire RVF by interacting with infected livestock, but they can also contract it through bites from infected mos-

Corresponding author: Sahar N. Mohamady, Clinical Pathology& Pathology Department, Zagazig Provincial lab, Animal Health Research Institute (AHRI), Agricultural Research Center (ARC), Giza, Egypt
Email address: sahar_nasr_2012@outlook.com.
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quitoes (**Pepin et al 2010, Linthicum et al. 2016 and Kwaśnik et al. 2021).**

In humans, RVF leads to a serious influenza-like illness, which can sometimes involve more severe hemorrhagic problems and death. Moreover, it triggers significant outbreaks at irregular intervals of 5 to 35 years **(FOA 2000).**

Epidemiology:

RVFV was initially extracted in 1931 during an outbreak among sheep in the Rift Valley in Kenya. **(Daubney &Hudson 1932)**. Additionally, the significant outbreaks impacting animals and humans have taken place in various nations in Africa, such as Egypt, Kenya, South Africa, Madagascar, Mauritania, Senegal, and Gambia. **(Jäckel et al. 2013, Sow et al. 2014**, **Nanyingi et al. 2015 , Sang et al. 2017 and Fawzy & Helmy 2019).** The weather conditions, occasional flooding, an increased greenness of vegetation index, and the resulting rise of mosquito vectors infecting vulnerable ruminant hosts result in the development of severe epidemic RVF (**Nanyingi et al. 2015).**

In 2020, the European Food Safety Authority (EFSA) published a study regarding the potential for RVFV to enter Europe as a result of the disease's spread to new areas and the discovery of seropositive animals in Tunisia and Turkey **(Nielsen et al 2020).**

Etiology:

The RVF virus, which is linked to the genus Phlebovirus and the family Bunyaviridae, is the cause of RVF. These spherical virions have a diameter of 80–120 nanometers and are enveloped in a bilipid layer formed from the host cell through which spikes of virus-coded glycoprotein protrude **(Kahen 2005)**. This single-stranded RNA virus has two surface glycoproteins, G1 and G2, and a lipid envelope. The three portions of the genome are called L (Large), M (Medium), and S (Small). The RVF virus reproduces in both vertebrates and insects. The liver, spleen, and brain are the main locations where viruses replicate. The virus is resistant to alkaline conditions and is inactivated at pH values below 6.8. Disinfectants including calcium hypochlorite, sodium hypochlorite, and acetic acid can inactivate the virus, and it can be preserved for eight years at temperatures below zero degrees Celsius **(Davis et al. 2003).**

Vector:

The *Aedes* and *Culex* genera are regarded as the primary vectors. (**Linthicum et al. 2016 and Lumley et al. 2017)**. Primary vectors like floodwater Aedes (such as *Aedes mcintoshi, Aedes ochraceus, Aedes sudanensis, and Aedes dentatus*) also keep the virus viable in their eggs during dry spells. It is suggested that the virus may persist during inter-epidemic and overwintering times through vertical transmission (from adult to egg). **(Linthicum et al. 1985 and Mohamed et al. 2013)**. Disease outbreaks might happen. Infected eggs hatch as the rainy season starts, and infected adult female mosquitoes spread the infection to animals in the area. The RVFV secondary vector spreads the virus horizontally from animals with high virality to people. The illness may spread geographically as a result of secondary vectors such as mosquitoes, mostly from the Culex (*Culex pipiens, Culex poicilipes, and Culex univittatus*), *Anopheles, and Mansonia* species moving into areas with sick animals **(Sang et al. 2017).**

Transmission

The main way that RVF is spread is through mosquito bites from a variety of species. Infected animals and bug bites are the two ways that humans get the virus. Contact with blood, body fluids, or the tissues and organs of infected animals or fetuses is the main method by which humans get infected. Inhaling aerosols of infected bodily fluids can result in infection. Thus, exposure as an occupational hazard cannot be avoided in any kind of work; among the activities least safe from RVFV infection risk are veterinary procedures and the skinning or killing of diseased animals. Another known risk factor for RVFV infection is the consumption of raw or unpas-

teurized milk. **World Health Organization (2018).**

Pathogenesis

Depending on the animals' susceptibility or resistance, three different infection patterns are typically seen in both animals that are naturally infected and those that are experimentally infected. Uncontrolled viraemia and a severe acute infection are possible outcomes. The second pattern is one in which the viraemia rapidly declines and the illness is moderate to asymptomatic. The third pattern is characterized by delayed infection-related consequences **(Coetzer and Ishak 1982 Pepin et al. 2010).**

Hepatocellular alterations brought on by infection may develop to necrosis, which is indicated by elevated liver enzyme levels, leukopaenia, or thrombocytopenia **(Findlay 1932).** The incubation period varies from 1 to 6 days. The incubation duration is 12-72 hours for lambs that are newborn, 24-72 hours for adult sheep, goats, and cattle, and 3-6 days for humans **(USDA 2011).**

Clinical symptoms of RVF:

Include increased frequency of abortions (sometimes known as "abortion storms"), a high death rate in young animals, vomiting, stomach colic, hemorrhagic diarrhea, prolonged prostration, jaundice, dysgalactia, high fever, nasal and ocular secretions, and lymphadenitis in adult animals. (**Ikegami and Makino 2011**). There is a noticeable difference between juvenile and adult animals' vulnerability to and advancement of RVF. The illness takes 24 to 36 hours to incubate in sheep. Multi-focal liver necrosis and sporadic moderate splenomegaly were discovered during post-mortem examination. **(FAO 2003).**

Concerning experimental infection showed mortality in adult sheep is between 20 and 30%, while in newborn lambs it is 95–100% **(Easterday 1965)**; When ewes are pregnant, the probability of abortion is nearly 100% (**Ikegami and Makino 2011)**.

Compared to sheep and goats, cattle are less prone to illness. Adult RVFV infections often have no symptoms, but they can potentially become acute and have a 5% fatality rate **(Ikegami and Makino 2011)**.

Although camels are less vulnerable than cattle, an acute RVFV infection can nevertheless cause a serious illness and even death. Abortions, foot lesions, hemorrhages, and eye discharge are some of the symptoms (**Fawzy and Helmy 2019).**

Human infections usually result in a self-limiting feverish sickness. While some infected persons may have flu-like symptoms, around 50% do not exhibit any clinical symptoms. A tiny proportion may experience severe clinical manifestations, such as encephalitis, eye problems, or hemorrhagic fever with liver illness (**Easterday 1965 and Ikegami and Makino 2011).**

Clinical pathology:

Severe leukopenia, elevated blood levels of enzymes such as glutamic dehydrogenase (GLDH), linked to liver damage and thrombocytopenia, are frequent observations **(FAO 2003)**.

Diagnosis:

Clinical symptoms:

Fever (40–42 degrees Celsius), anorexia, depression, weakness, mucopurulent nasal discharge, vomiting, jaundice, and hemorrhagic diarrhea are all symptoms of the affected animals **(Swanepoel 2004).** By the identification of certain IgM or IgG antibodies. During the acute (febrile) stage of the illness, entire blood or serum samples, as well as other post-mortem organs like the brain, liver, or spleen, are obtained from fresh corpses or aborted fetuses to isolate the virus or identify viral RNA. Milk can also be found to have the virus.

Conventional and real-time RT-PCR assays are presently the quickest and most sensitive tests for detecting and quantifying RVFV during outbreaks (**Garcia et al 2001)**.

The identification of the nucleoprotein of RVFV was established through a lateral flow

immuno-chromatographic strip test or A pen-side test. This form of assay aids in improving the early diagnosis and management of RVF during ongoing outbreaks. **(Cêtre-Sossah et al 2019).** The liver's histology confirms the diagnosis. It changed color from yellow-brown to red-brown, got somewhat bloated, and was pliable. Punctate areas of bleeding and tissue death may also be seen in the liver, creating a speckled look on its surface. In lambs, spots of petechiae and ecchymosis on the abomasal mucosa are frequently found, with digested blood detected in the lumen and within the small intestine. Adult animals may have a significant amount of new blood in the intestinal lumen, as well as hemorrhages and oedema in the abomasal plica, and several organs were impacted as:

Haemorrhagic foci in the spleen might occasionally manifest as subcapsular marginal infarctions. There are petechial and oedema in the lymph nodes. There are endocardial and sub-epicardial hemorrhages in the heart. The lungs exhibit emphysema, necrosis in the per-bronchial lymphoid tissue, interlobular septum, and alveolar and interstitial oedema. Additional observations include pulmonary oedema, per-renal oedema, serous or sanguineous and serous liquids in the bodily spaces, and subcutaneous serosal bleeding and nephrosis **(Ikegami and Makino .2011)**

Control of disease and prevention

Limiting an animal's mobility under epizootic conditions can aid in the management of RVF. In addition, all vulnerable animals should be vaccinated to avoid vector infection by amplifyinghosts.

Immunizations: RVF does not have a particular therapy. Nonetheless, two vaccines—a live attenuated vaccine and a formalin inactivated vaccine—are accessible and often utilized in endemic nations to prevent RVF. Sheep and goats who get the live attenuated Smith burn vaccination develop lifetime protection. It is not advised to use the Smith burn vaccine extensively in non-endemic nations or during outbreaks because to the possibility of reversion. Since the inactivated vaccine does not provide long-term immunity, booster shots and yearly revaccinations are necessary for ongoing infection prevention. It is advised to use the inactivated vaccination on pregnant animals and in RVF-free nations where outbreaks are occurring **WHO** (**2007)**. Additionally, it is advised to use pesticides to eradicate mosquitoes and to prohibit the export of cattle during RVF outbreaks (**Pal 2007)**. Control of mosquito egg laying sites, larvae and adult must be done.

CONCULION

RVF threatens the health of humans and animals, so we need to limit the spread of infection, especially in Africa by improving vaccination campaigns, and creating vaccine banks for many diseases, which represents a significant step forward in combating the worldwide dissemination of animal diseases. Additionally, a greater comprehension of the transmission routes within Africa may facilitate the prevention of RVFV's spread from Africa to other continents. Additionally, the world combine should have a plan to address one of the most significant arbovirus risks to human and animal health in a way that is genuinely One Health One World.

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