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Mini Review Article

Rift Valley Fever in Livestock Wildlife and Humans: A Mini Review

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Abstract
Rift Valley fever is an arboviral disease that mainly affects both animals and humans, associated with symptoms like strong chills, malaise, weakness, nausea, a severe headache, or a feeling of fullness around the hepatic region. It is mainly caused by a family of *Bunyaviridae* and the genus *Phlebovirus* of Rift Valley Fever (RVF). The virus spreads through mosquitoes and domestic animals in humans. The incubation period for RVF usually lasts four to six days. The majority of cases of RVF were non-fatal and self-limiting, whereas thrombosis, severe dengue, neurological problems, eyesight loss, or abortions in pregnant females have also been reported to be associated with the fever. Since 2000, multiple outbreaks have hit a wide range of Sub-Saharan African countries and the Arabian Peninsula. This review article mainly demonstrates how the virus affects humans, its causes, and conditions associated with RVF and currently available treatments.
Keywords: Rift Valley Fever Virus (RVFV), Etiology, Pathogenesis, Hemorrhagic fever

Introduction

Rift Valley Fever (RVF) is a viral illness that mostly affects farmed animals in Sub-Saharan Africa, including cattle, buffalo, sheep, goats, and camels. People can get the disease from the blood, body fluids or tissue of an infected animal, or from a mosquito bite (Rift Valley fever n.d.). The Rift Valley Fever Virus (RVFV) poses a significant threat as a growing zoonotic disease, especially to disadvantaged African communities that are susceptible to financial and environmental challenges. (Bracci, 2022). In reality, several past epidemics of RVF infection in Africa were first discovered because of infections among veterinarians and their assistants after they performed extensive tests on sick animals. Such persons are at risk for infection via direct exposure to infected patients and animals, as do farmers and slaughterhouse employees (Bird et al., 2009). A zoonotic arbovirus called RVFV may infect both humans and animals and cause serious illness. Ever since its detection in 1931, the virus has often produced epidemics in the Middle East and Africa (Nanyingi et al. 2015a; Clark et al., 2018; Oymans et al., 2020).

Epidemiology

Office International des Epizooties (OIE) and Animal Disease Notification System (ADNS) collected epidemiological statistics about

RVF outbreaks for animal outbreaks in Africa and Mayotte & France (MS), respectively, and WHO for notifications of human outbreaks (Nielsen et al., 2020). Around 40,000 animals, including sheep, goats, camels, and cattle, were said to have perished in Saudi Arabia during the epidemic of 2000, while 8,000 to 10,000 of them gave birth. In 2007, the epidemics in Sudan resulted in Saudi Arabia imposing restrictions on cattle imports from Sudan. This had a major impact on the livestock markets of both nations (Himeidan et al., 2014; Bett et al., 2019).

Etiology

A Single-strand, spherical, enveloped RNA Arbovirus of the genus *Phlebovirus* is the cause of RVF. The *Bunyaviridae* family is the habitat of such a virus (Kapoor, 2008). The genome is divided into three segments: large, medium, or small. Mostly mosquitoes and vertebrate animals can reproduce the RVFV. Viral replication mostly occurs in the liver, spleen, and brain (Pal et al., 2012). The prevalent risk factors included being bitten by mosquitoes and either handling or leaving newborn infants. Other risk factors include ingesting or handling ill animal products, working with livestock as herders, handling fetal tissue, sleeping with animals, touching blood, and attending to animals during childbirth (Nanyingi et al., 2015b).

How RVF affects Humans

The outbreak of RVF is through different routes in the human body. The primary mode of disease transmission is through mosquito bites, specifically from Aedes and Culex species. In addition, coming into contact with sick animals can also be a source of transmission (McMillen and Hartman, 2018). Furthermore, the transmission of the RVFV through hematophagous flies is also possible (Ikegami and Makino, 2011).

Outbreak since 2000 of RVF

According to the Saudi Ministry of Health, there were 516 instances of rift valley fever in 2000, with 87 deaths. In the year 2000, Yemen's Ministry of Public Health recorded 1087 people who were suspected of having a health issue, with 121 of them having died. The Egyptian Ministry of Health diagnosed 148 instances of RVF in 2003, including 27 deaths. In 2006, from the period 30 November 2006 to 12 March 2007, a sum of 684 sufferers as well as 234 deaths of humans was noticed in Kenya. From December 2008- May 2009, the Ministry of Health reported that Madagascar diagnosed 236 infected cases with additional seven deaths. Furthermore, from 16 September 2012 to 13 November 2012, a total of 36 cases including 18 deaths were reported in six regions of Mauritania. On 11 October 2016, a group of 105 human subjects was diagnosed with RVF, including 28 deaths in the Tahoua region of Niger (Ikegami and Makino, 2011).

Pathophysiology

The non-structural protein encoded on the S segment (NSs) of the RVFV is the sole component that has been discovered to directly affect the hosts, even though that certain elements of the RVFV's RNAs play a key part in the virus' pathogenesis. In contrast to the host's interferon (IFN) antiviral response, NSs are hostile and belligerent. The immune system requires interferons to combat viral infections within a host. The first is competitive inhibition of the transcription factor's synthesis, which is believed as the origin of this inhibitory mechanism. The RNA polymerase I and II-required component on this transcription factor interacted with and bound by NSs. This interaction results in competitive inhibition with another transcription factor component (McMillen and Hartman, 2018). The majority of RVF patients experience a febrile, self-limiting sickness. However, some people get thrombosis, severe dengue, neurological problems, or eyesight loss. There were a lot of diametrically lab cases within the 1930s and 1940s because there were insufficient bio-safety protocols in place. However, the majority of those and subsequent outbreaks' victims had non-fatal, self-limiting illnesses. Incubation for RVF usually lasts 4 to 6 days. Strong chills, malaise, weakness, nausea, a severe

headache, and/or a feeling of fullness around the liver area are the first symptoms to appear, as shown in Figure 1. Following these symptoms are a high body temperature (38.8 to 39.5 °C), lowered blood pressure, back, shoulder, neck, or leg discomfort, rigidity, shivering, flushed face, red eyes with sores, constipation, sleeplessness, and/or photophobia (Petrova et al., 2020).

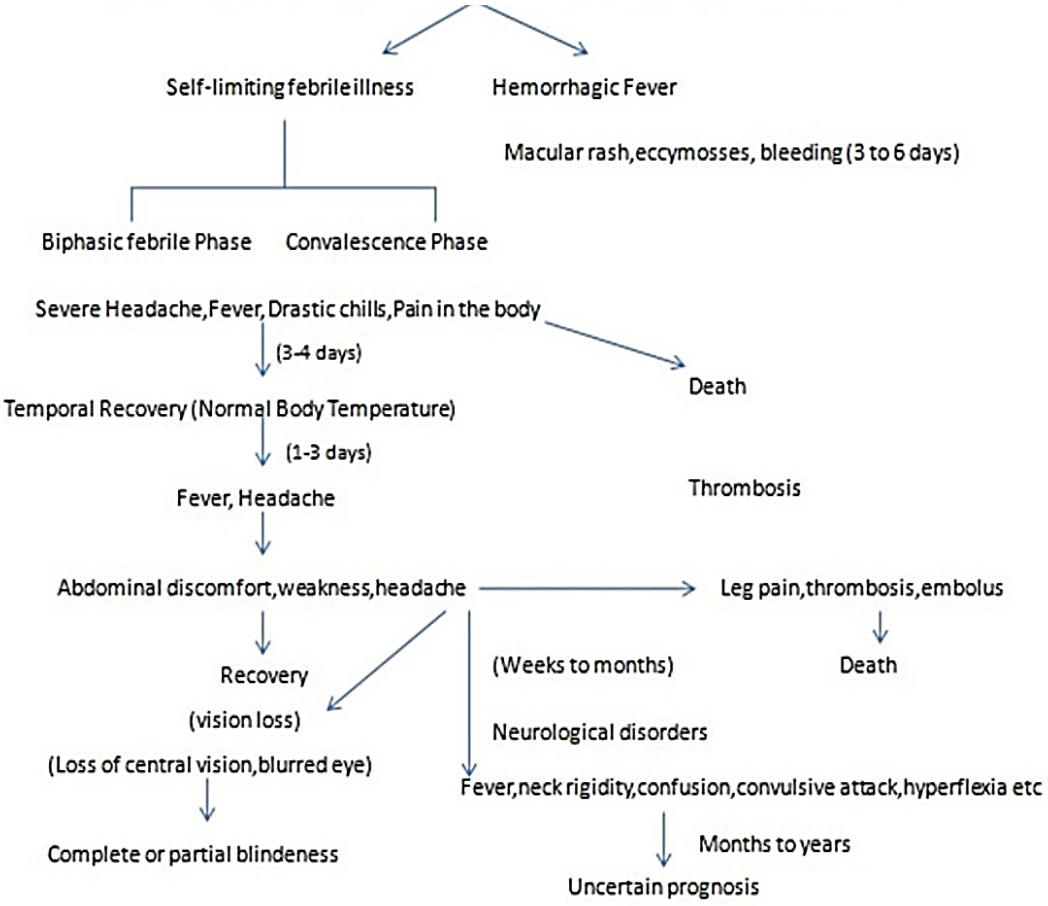

Fig. 1. The pathological form of Rift Valley Fever in humans.

Neurological Disturbances

It has been observed that many patients with RVF experience self-limiting febrile illness, which consists of biphasic febrile and convalescence phase, which mainly shows different medical conditions like neurological disorders, vision loss, and haemorrhagic fever and thrombosis (Petrova et al., 2020). The incubation period of this fever is of four to six days, and symptoms start immediately with severe chills, body aches, dizziness, headache, vomiting, or sensation of fullness in the liver region (Sall et al., 2002; Bird et al., 2009; Mansfield et al., 2015). Some of them experience high-grade fever, long-lasting fever for more than ten days (Sissoko et al., 2009) (Rift Valley fever n.d.). However, during the convalescence phase, some of the individuals develop loss of balance and muscle fatigue symptoms in their body (Benedict et al., 2015). In a study of Encephalitis patients with RVF, it was observed that neck rigidity persisted for five days starting from the twenty-fifth day. Furthermore, some of the individuals exhibited hyperreflexia and fever until fifty days. In addition, the individuals did not experience any significant effects from medications such as Rifampicin, Amantadine, and Dexamethasone during a two-week period (Caroline et al., 2014). Alrajhi et al. (2004) conducted another study on female patients

with encephalitis and retinitis. The subject had an ataxic walk, bilateral retinal haemorrhage, and fever, as well as a low-aware level. She was let out from the hospital on the thirtieth day of her sickness to her home; at the time, she was blind, had urinary incontinence, and was quadriplegic. Furthermore, for the next year, she had no improvement in any of her neurological issues (Peters et al., 1986).

Loss of Vision

Some of the individuals reported retinopathy or maculopathy with RVF (Ayoub et al., 1978; Scharton et al., 2014). These patients experience central vision loss and blurred vision infection either immediately or after a month or year. Macular edema with discharges, including a white mass encompassing the macular area, accompanied by or without retinal haemorrhage injury, vitreous haze, or vasculitis, may be present in both or one eye (Siam and Meegan 1980; Kende et al., 1987; Al-Hazmi et al. 2005; Ikegami and Makino 2011).

Haemorrhagic Fever

Some investigations identified RVF patients with lethal complications, which may result in mortality in some cases (Findlay, 1932). Most patients have a fever, rigor, nausea, vomiting, headaches, injected conjunctive, sleepiness, or body aches. Macular eruption over the entire trunk, ecchymosis on the arms, limbs, or even the eyelids, bleeding from the gums, as well as gastrointestinal oral membranes, low arterial pressure, hematemesis, melena, diarrhea, throat pain, pneumonia, jaundice, and/or hepatosplenomegaly were among the symptoms reported by some of the individuals. In some circumstances, the enzymes alanine aminotransferase (ALT), the enzyme aspartate aminotransferase (AST), the enzyme lactate dehydrogenase (LDH), as well as platelet count and haemoglobin levels rise, while platelet count and haemoglobin levels fall (Kitchen, 1934; Francis and Magill, 1935; Salib and Sobhy 1978; Maar et al., 1979; Alrajhi et al., 2004).

Thrombosis

Some of the deadly RVF cases had thrombosis. According to an investigation done by Schwentker et al. the patient's fever returned to its normal level on day four after the commencement of symptoms; nonetheless, two popular spots of several centimeters in diameter had been found on the patient's thigh and leg around the fifth day of treatment and stayed until the eighth day. (Schwentker and Rivers 1934).

Possible Vertical Infection

In Egypt, a retrospective study was conducted on pregnant females, results of their study depicted there was no increase in the total number of abortions during the RVF outbreak. Furthermore, the serological conversion rate of aborted females before and after the outbreak was 31.1% and 27.5%, respectively (Schrire, 1951). Pregnant women demonstrated similar research during the outbreak of RVF in Saudi Arabia. Four days prior to delivery, they experienced fever, headache, dizziness, and muscle aches throughout their body. They also produced IgG specific to RVF (Freed, 1951). Her newborn babies existed with an anti-RVFV IgM antibody, as well as ALT, AST elevation, jaundice, the extension of the activated partial thromboplastin time and the prothrombin, and died on the sixth day after birth (Deutman and Klomp, 1981).

Diagnosis

Diagnosis In accordance with WHO guidelines, 23 a real-time polymerase chain reaction (RT-PCR) is desired for such detection of RVFV RNA from blood or plasma and identification of anti-RVFV IgM and IgG antibodies, the detection of the RVFV serology, and/ or the isolation of RVFV. The capacity to identify antigenic (isolated virus, viral RNA) or immunological markers (IgM and IgG) or antigenic timing of sample in relation to illness development determines the best test to use. To confirm RVFV cases, a combination of molecular and serological testing is often required. Other infectious diseases caused by abortifacient agents, including brucellosis, leptospirosis, chlamydiosis, campylobacteriosis, *Coxiella burnetii* infection, and salmonellosis, are differential diagnoses (Schrire, 1951). Traditional virological techniques, such as viral isolation, histopathology, antigen identification, antibody detection, and nucleic acid-

based tests, can identify RVFV (Freed, 1951). Overall, RT-PCR is a quick, sensitive, accurate, and dependable test for early RVFV infection detection, although there are some constraints in terms of its sensitivities, cost, and degree of operator competence. To quickly identify RFV suspicions, it is necessary to regularly use it in combination with IgM detection (Al-Hazmi et al., 2005).

Management

Non-Pharmacological

This type of treatment involves information for medical professionals, a drive to remove mosquito breeding grounds, and advice on avoiding contact with ill recommendations for how to follow all safety steps while performing tasks involving animals, including butchering and slaughter (Siam and Meegan, 1980). To decrease the risk of transmitting diseases from animals to humans due to consuming raw milk, young animal blood or tissue, it is possible to take preventative measures. In epizootic locations, every animal product (blood, meat, and milk) should be thoroughly processed before ingestion (Al-Hazmi et al., 2005).

Pharmacological

Treatment with curcumin reduced RVFV infection to nearly undetectable levels. Sorafenib was one contender that had the best non-toxic ability to suppress viral levels. Researchers looked at sorafenib effects in both the *in vitro* and *vivo* models further to confirm its effectiveness towards RVFV infection (Swanepoel et al., 1979). Broad-spectrum antivirals represent the gold standard for developing effective responses against a wide range of viral diseases. Nebulized ribavirin, additionally being used off-label to manage viral disease pathogens such as Lassa, may be a viable treatment for respiratory syncytial virus infections. Several viruses, especially RVFV, have now been found to be robust with ribavirin in both vitro and in vivo (Abdel-Wahab et al. 1978; Yassin, 1978;). The administration of treatments, which included 0.4% CMC placebo and 70 mcg of ribavirin (a positive control), began one hour after the infection (HPI) and continued twice daily for ten days. A potential pyrazine derivative known as favipiravir (T-705; 6-flouro-3-hydroxy-2-pyrazinecarboxamine) has shown strong antiviral efficacy against a number of RNA viruses (Scharton et al., 2014)]. In order to prevent viral infections in rhesus monkeys, such as simian hemorrhagic fever, rabies, and yellow fever, prophylactic therapies using polyriboinosinic-polyribocytidylic acid stabilized with poly-L-lysine and carboxymethyl cellulose or poly (IRCLC), are successful (Arishi et al., 2006) .

Conclusion

This review article describes RVF etiology, epidemiological characteristics, outbreaks since 2000, and many other important parameters. RVF is a zoonosis that affects both humans and animals. The RVF was initially reported in sub-Saharan Africa during the previous decades. From the above reports, it has been found that several studies have been undertaken on humans and animals. Ongoing research and experiments are being conducted to cure and prevent the harmful effects of RVF on humans and the population. Various evidence studies have been conducted, and still, research on RVF is under-processed to completely cure the disease.

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Not applicable.

Ethical approval

Not applicable.

Conflict of interest statements

The authors declare that there is no conflict of interests.

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