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Rift Valley Fever - A Review

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Abstract

Rift Valley fever is an acute or per-acute arthropod-born zoonotic disease of domestic ruminants. The disease is predominantly confined to Africa and belongs to the genus Phlebovirus, in the family Bunyaviridae. It is a seasonal disease with a higher occurrence during heavy rainfall seasons, which allows the vector population to breed, and its appearance is correlated with vector density. For endemic areas, diagnosis depends on epidemiology, clinical signs, and microscopic lesions, but confirmation of diagnosis made in the laboratory by using virus isolation or immunological tests is needed. The risk of introducing RVF into disease-free countries via the importation of an infected animal or mosquito is real, and the consequent restriction of access to export markets may induce dramatic economic consequences for national and local economies. With high numbers of vector species present in disease-free regions, the intensification of international trade, and the effects of climate change, Rift Valley fever is now considered a major challenge in global zoonotic disease control. Therefore, epidemiological studies to know the extent of the disease and the provision of pre-exposure vaccines for highly risk groups, as well as pre-export animal inspections, are important control measures to be practiced.

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Introduction

Rift Valley fever (RVF), also known as enzootic hepatitis, is an acute febrile arthropod-borne disease of sheep, goats, cattle, and humans present in most countries of sub-Saharan Africa. This disease is characterized by high mortality rates in young animals and abortion in pregnant ruminants (Smith, 2009).

A virus of the genus Phlebovirus and of the family Bunyaviridae (Smith, 2009) causes RVF. Aedes mosquitoes of sub-genera Aedimorphus and Neomelaniconion are the principal vectors (Knipe and Howley, 2007). The Rift Valley fever virus (RVFV) was initially reported in the Rift Valley of Kenya in 1931 but now exists as an epizootic throughout sub-Saharan Africa, with recent extensions into Egypt, Madagascar, Mauritania, and most recently to the Arabian Peninsula (Radostits *et al.*, 2007). Outbreaks of the disease tend to occur unpredictably in eastern and southern Africa at intervals of five or more years and are associated with abnormally heavy rains and a dramatic rise in the vector population (Quinn *et al.*, 2002). Since Rift Valley fever is one of the most important diseases that seriously affect humans and animals, it is important to understand the overall characteristics of this disease.

Therefore, the objectives of this paper are:

To review the available literature on Rift Valley fever.

Etiology

The Rift Valley fever virus belongs to the family Bunyaviridae, genus Phlebovirus. They are spherical virions with a diameter of 80 to 120 nanometers and a host cell-derived bilipid envelope that projects virus-encoded glycoprotein peaks (G1 and G2) (Kahn, 2005). It has a 3-segment, single-stranded, negative-sense RNA genome with a molecular weight of 4-6×106, and each fragment (large, medium, and small) is contained within a separate nucleocapsid in the virion. Viruses are resistant to alkaline media but are inactivated at pH < 6.8. Viruses can be inactivated by disinfectants such as calcium hypochlorite, sodium hypochlorite, and acetic acid and maintained for 8 years when stored below 0°C (Davies *et al.*, 2003).

Epidemiology

Occurrence

The Rift Valley fever virus was originally reported from the Rift Valley of Kenya in 1931 but has now persisted and appeared in animal epidemics throughout sub-Saharan Africa, with near extensions to Egypt and Madagascar, Mauritania, and more recently to the Arabian Peninsula. It has the potential to spread to other countries (Radostits *et al.*, 2007). Epidemics tend to occur unpredictably in eastern and southern Africa over a period of 5 years or more and are associated with unusually heavy rains and significant increases in vector populations (Quinn *et al.*, 2002). Recent outbreaks of RVF in North and West African countries, which occur independently of rainfall in arid countries, appear to be related to breeding vectors in large rivers and dams (Cagnolati *et al.*, 2006). Major outbreaks of RVF have been recorded in Egypt (1977/78 and 1993), Mauritania (1987), Madagascar (1990/91), Kenya and Somalia (1997). RVF was first recognized outside of mainland Africa in 2000, with outbreaks reported in Saudi Arabia and Yemen. The last outbreak of RVF in East Africa was recorded in late 2006 and early 2007 (OIE, 2009).

Source of infections

This virus is thought to be perpetuated by a cycle involving vector mosquitoes, wildlife, and domestic animals and by ovary transmission in certain species of flood-resistant Aedes mosquitoes. Disease outbreaks occur in endemic areas of zoos when wet flood conditions favor the expansion of vector populations in the presence of susceptible livestock. Ruminants are highly sensitive and serve as primary amplification hosts. A pronounced but brief viremia occurs in infected animals and facilitates the spread of disease by insect bites. During the viral period, the blood, tissues of affected animals, milk, feces, aborted fetuses, and livestock are sources of infection (Quinn *et al.*, 2002). An unidentified wildlife reservoir host may also exist (Radostits *et al.*, 2007).

Method of transmission

The Rift Valley fever virus is an arbovirus transmitted transovarially by mosquito vectors. More than 30 mosquito species were found to be infected by RVFV (McIntosh *et al.*, 1981), belonging to seven genera, of which Aedes and Culex are considered the most important from the point of view of vector competence (other genera are Anopheles, Coquillettidia, Eretmapodite, Mansonia and Ochlerotatus). The capacity of the RVF virus to transmit without the involvement of arthropod vectors raises concern over the possibility of the virus's importation into non-enzootic areas through contaminated materials, animal products, viremic humans, or no livestock animal species (Edward *et al.*, 2011). Contagion is not considered important in livestock, as opposed to the case in humans (Blood *et al.*, 1993). In addition, wild ruminants may play a role in the epidemiology of RVF in areas where their population density is high (Evans *et al.*, 2008). Humans are also easily infected by aerosols from infected animals when humidity is high or by contact with infected animal tissue, aborted fetuses, mosquito bites, and laboratory procedures. There is a risk of mosquitoes transmitting disease to animals in uninfected areas. No human-to-human transmission has been documented (Kahn, 2005), but placental RVFV transmission can occur in vertebrates, including humans, resulting in abortion and a high neonatal mortality rate. (Arishi *et al.*, 2006).

Risk factors

Environmental risk factors

Environmental factors such as climate (temperature, humidity, annual precipitation, and rainfall intensity), hydrology (near a lake or dam, irrigation, water accumulation, proximity to rivers), and topography (altitude, land cover) can affect vector capacity (Maclachlan and Dubovi, 2011). The incidence of the disease varies with the size of the vector population. It is highest during the heavy rainy season, which allows vector populations to increase and expand from fixed

waters to spawn in surface waters in normal areas (Radostits *et al.*, 2007). New zoonotic diseases can occur at any time in Africa, even in arid regions, when rural development projects of strong ecological significance are multiplied (Lefèvre and Shimshony, 2010).

Animal Risk Factors

Cattle, sheep, camels, water buffalo, monkeys, humans, mice, rats, ferrets, and hamsters are very susceptible, and goats to a moderate degree, but not pigs, rabbits, guinea pigs, or poultry (Blood *et al.*, 1993). Viremia in animals varies by species and age. It is short in neonatal sheep because it appears 16 hours after infection and persists until death, usually occurring within 36–48 hours. In contrast, in sheep, goats, and adult cattle, viremia can be detected one to two days after infection, with a peak of three to five days and lasting up to seven days (Lefe'vre *et al.*, 2010).

The miscarriage rate in infected pregnant sheep is almost 100%. Sheep seem to be more sensitive than cattle or camels. The native cattle breeds showed marked resistance to RVF compared with the imported breeds. Trafficking in animals suspected of being a source of infection in areas was previously free (Davies and Martin, 2003).

Pathogenesis and Pathology

After penetration by mosquito bites, skin wounds, or aerosols through the oropharynx, there is an incubation period of 30–72 hours, during which the virus invades the liver parenchyma and lymphatic reticular organs (Dubovi, 2011). In pregnant women, the virus has a particular predisposition to the placenta and fetus, causing rapid fetal death. Disseminated intravascular coagulopathy is the cause of many lesions (Lefèvre and Shimshony, 2010). Multiple foci of gray-white necrosis, 0.5–1.0 mm in diameter, are always present in the parenchyma, but they may not be clearly seen due to organ discoloration. There may be edema and bleeding in the gallbladder wall and hepatic lymph nodes. Numerous petechiae and bruises are present in the lining of the stomach and small and large intestines and contain dark chocolate brown substances due to the presence of partially digested blood. Splenomegaly, mild or moderate, with intracapsular hemorrhage in domestic ruminants, peripheral and visceral lymph nodes are enlarged, edematous, and possibly petechiae. Other changes include generalized subcutaneous, serum, and visceral hemorrhage, mild to moderate effusion, often bloody, in body cavities, congestion, and pulmonary edema (Cagnolati *et al.*, 2006).

Clinical Signs

Rift Valley fever affects many animals, but the severity of clinical signs varies with age. The main manifestation is an epidemic of abortion and neonatal mortality (Lefèvre *et al.*, 2010). The incubation period is 12 to 36 hours in sheep, with biphasic fevers that can reach 106°F (41°C). The sheep usually die within two days. Affected animals are listless, reluctant to move or feed, and may show signs of colic (Kahn, 2005). In field conditions, 90–100% of pregnant ewes miscarry, and mortality in ewes is 90% and in adult ewes is 20–60%. Clinical disease and similar findings in goats. In cattle, the disease is less severe, with 10–30% mortality in calves and cows, but 90–100% of pregnant cows abort (Murphy *et al.*, 1999) and also have fever (40–41). °C), dry and/or dull hair, watery eyes, runny nose, excessive salivation, loss of appetite, weakness, bloody or bad-smelling diarrhea, and decreased milk supply. In camels, after a brief viral infection, miscarriage is the only visible sign of infection. Horses, dogs, and cats are susceptible to RVFV infection but do not show clinical signs. Thus, as part of an unknown group of infections, wild animals also belong to this group and develop antibodies only (Lefèvre and Shimshony, 2010). The majority of human RVF infections are either equivocal or associated with moderate to severe, non-fatal flu-like illnesses (fever (37.8–40 °C), headache, myalgia, asthenia, nausea and epigastric discomfort, photophobia), but a few, possibly less than 1%, develop fatal eye damage, encephalitis, or fatal liver disease with hemorrhagic manifestations (Kahn, 2005).

Clinical Pathology

Antibodies appear in the serum about a week after infection, and persistence depends on the type of antibody. There is severe leukopenia associated with RVF infection, which is most evident in the early stages of infection. Severe liver damage leads to increased serum levels of enzymes involved in this condition, e.g., glutamic dehydrogenase, and leads to thrombocytopenia (Davies and Martin, 2003).

Because of its wide geographical distribution and the explosive ability to colonize new areas where livestock production is widespread, laboratory confirmation of the presence of RVFV is considered diagnostic (Maclachlan and Dubovi, 2011). In areas where RVF is endemic or

nearby, any significant increase in lamb and lamb mortality, combined with abortions, should be considered suspicious. Generally, such a case, which is associated with a human flu-like illness, would rule out RVF. During an animal outbreak, hepatic changes typical of acute RVF may indicate a preliminary diagnosis, but laboratory confirmation is required. In the subacute form and when miscarriage is the only sign, biological studies are the only diagnostic tool (Lefèvre and Shimshony, 2010).

Laboratory Diagnosis

The Rift Valley fever virus can be isolated from serum and blood collected in anticoagulants during the febrile phase of the disease in the liver, spleen, and brain of dead animals or aborted fetuses. All precautions should be taken when sampling (Radostits *et al.,* 2007). Primary isolation is usually performed on different types of cultured cells, such as African green monkey kidney cells, baby hamster kidney cells, chicken embryo mesh, or primary cells derived from bovine or sheep. Alternatively, hamsters, adult or lactating mice, embryonic chicken eggs, or 2-day-old lambs can be used for primary virus isolation (OIE, 2008). Rapid diagnosis can be obtained using the supernatant of homogenized samples as antigen in virus neutralization assays; immunofluorescence staining of impression smears from infected liver, spleen, brain, or cell cultures; or by demonstration of virus in serum, obtained during the febrile phase of the disease, by enzyme immunoassay or immunodiffusion. The presence of characteristic histopathological lesions in the liver is helpful in the diagnosis (OIE, 2008). All routine serological tests can be used to detect antibodies against RVFV and are useful in epidemiological studies. The IgM ELISA test can demonstrate a recent infection using a single serum sample (Kahn, 2005).

Differential Diagnosis

Rift Valley fever, Wesselsbron disease, and other arthropod viral diseases tend to occur under the same climatic conditions. Rift Valley fever must be distinguished from Wesselsbron disease because both diseases can cause death in lambs and calves and miscarriage in ewes.

However, VFR is associated with much higher mortality and abortion rates than Wessels-Brøns disease. Fatal agents associated with liver injury, hemorrhage, and/or superficial jaundice that may resemble RVFs in domestic ruminants include botulism as well as bacterial sepsis such as coccidiosis. Sepsis, salmonella, and anthrax infections, as well as diseases that cause miscarriage, including brucellosis, leptospirosis, and chlamydia, can be confused with RVF. Nairobi sheep disease can also be confused with RVF (Radostits *et al.,* 2007).

Prevention and Control

There is no specific treatment for RVF. Vector control and quarantine can help protect livestock and people in endemic areas, but vaccination is the most practical and economical control measure. Quarantine can help protect livestock and people in endemic areas, but vaccination is the most economical and practical control measure (Radostits *et al.,* 2007).

Vaccination

The prevention of RVF is based on the vaccination of livestock to avoid the occurrence of epidemic diseases in animals and thus epidemics (Lefèvre and Shimshony, 2010). There is a dead virus vaccine (inactivated virus vaccine) that requires a booster shot for good immunity, and a live attenuated virus vaccine provides good protection for at least 28 months, but it is not recommended for pregnant women as it causes abnormal death and teratogenicity (Radostits *et al.,* 2007). Another sheep-attenuated RVF virus vaccine, produced from rat brains and embryonic eggs, is found to be effective and inexpensive, but it still causes abortions in pregnant ewes (Murphy *et al.,* 1999).). Inactivated vaccines prepared from highly immunogenic strains of RVFV are suitable for use in pregnant animals and can be used in RVF-free countries bordering endemic areas. (Quinn *et al.,* 2002). In pregnant ewes and cattle, vaccination should be repeated after 3 months to induce immunity that lasts for 1 year and confers immunity in the colon to the offspring (Kahn, 2005).

Vector Control

Control of vectors and host movement is necessary to interrupt the epidemiological cycle of the RVF virus and thus minimize the potential impact of an outbreak by reducing the transmission rate. Effective vector control methods include hormone inhibitors such as methoprene, extensive use of aerial insecticides or vehicles, and strategic treatment of mosquito habitat and soil with larvicide and pesticides, respectively (Davies and Martin, 2003).

Prevention of introduction

To prevent the entry of RVF into disease-free countries, importation of all susceptible species should be prohibited, and all necessary measures should be taken to prevent the entry of infectious insects and materials that are biologically infected (Blood *et al.*, 1993).

Recommendations

RVF was first recognized in Africa and has a great potential to spread to other continents and cause high lamb and calf mortality and abortion in adult animals and flu-like disease in humans. RVF is an economically important disease. In addition to its impact on animal health, the impact it results from import and export restrictions is significant, particularly in those countries in which livestock contributes a great share of their economies. As Rift Valley fever needs insects (mosquitoes) for its life cycle and transmission, its epidemics have cyclical occurrences. Immunization and vector control are the main strategies to reduce the incidence of RVF.

Therefore, based on the above conclusion, the following recommendations are forwarded:

- Repeated and regular epidemiological studies are required to know the extent of the disease.
- Provision of a pre-exposure vaccine for highly risky groups and immunization of animals in enzootic areas should be practiced.
- Pre-export animal inspections should be practiced to prove the absence of RVF virus activity in the areas from which the animals originated.
- In RVF-free countries, the importation of all susceptible species should be prohibited.

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