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RIFT VALLEY FEVER - A NEGLECTED PATHOGENIC VIRUS OF CAMELIDAE

U. Wernery
Central Veterinary Research Laboratory, Dubai, UAE

ABSTRACT

New World camels and dromedary camels can contract Rift Valley Fever with severe consequences. RVF has not been documented in Bactrian camels. RVF outbreaks are regularly observed after heavy rains in many different African countries and has also entered Saudi Arabia affecting human beings and livestock in 2000. RVF epidemics regularly cause serious economic hardship to animal owners due to loss in production and fatalities. The virus causes abortion storms in dromedary camels at all stages of pregnancy and systemic disease in young stock has been reported. RVF virus has been isolated during the RVF outbreak in Mauritania in 2010. Inactivated and live-attenuated vaccines are available, but have not been scientifically evaluated for use in camelids.

Key words: Epidemiology, prevention, Rift Valley Fever, treatment

Many pathogenic livestock viruses do not disease the *Camelidae* family. They are robust animals and show a high resilience to many diseases. Dromedary camels are resistant to Foot-and-mouth Disease (FMD), Peste des Petits Ruminants (PPR), Blue Tongue (BT), West Nile Fever (WNF) and Wesselbron disease. However, in recent years it had become obvious that New World Camels (NWCs) and Old World Camels (OWCs) can contract Rift valley fever (RVF).

Rift Valley fever (RVF) is an arthropod-borne viral disease of animals including humans, but mostly found in ruminants. Infection in humans is primarily a result of contact with material from infected carcasses (Hoogstraal *et al,* 1979). In addition to human health hazards, RVF epidemics regularly cause serious economic damage to animal owners through loss in production and fatalities, exacerbated by the 100% abortion rate at all stages of pregnancy. Strikingly, all of the RVF epizootics described to date have followed unusually severe rainy seasons, probably indicating a very large insect population as a vector prerequisite (Heubschle, 1983). RVF does not occur in very arid areas. In NWCs, the RVF virus (RVFV) causes not only abortions, but also systemic disease, whereas, in dromedaries, abortion at any stage of gestation is the predominant clinical sign in addition to haemorrhagic fever and pneumonia. RVF is an acute to peracute zoonotic disease of domestic ruminants predominantly in Africa. Floodwater-breeding mosquitoes of the *Aedes* genus, and less importantly biting flies, are considered to be epidemic vectors.

Aeitology

The Rift Valley virus (RVFV) is a member of the *Phlebovirus* genus of the family *Bunyaviridae*. The *Bunyaviruses* are spherical, are 80-120µm in diameter and have a host-cell-derived lipid bilayer envelope through which virus-coded glycoprotein spikes project. No significant antigenic differences have been detected between RVF isolates, but differences in virulence have been demonstrated. RVFV is a single-serotype vector-borne RNA virus. The non-structural protein NSs is a multifunctional protein that enables RVFV to evade the host's antiviral response. This protein is unique among the *Bunyaviruses*, as it forms a filamentous structure in the nucleus, but the virus replicates in the cytoplasm. Altogether, it appears that NSs has multiple functions to counteract the host cell interferon defence mechanism (Pepin *et al,* 2010).

Human implication

Rift Valley fever in humans appears as an influenza-like syndrome in 95% of infections, and the disease can be confused with malaria. This mild form may last for a week. However, during RVF outbreaks in Egypt and Mauritania, severe disease also developed in some patients showing the ocular, the meningo-encephalitic and the haemorrhagic forms. The last-mentioned form is certainly the most

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severe form, as it always leads to the death of the patient. Prevention of the disease depends on the implementation of an early warning system based on the epidemiological monitoring of animals. Human vaccines exist, but are not commercially available and are administered only to people at high risk.

Epidemiology

For more than 90 years, RVF epidemics have occurred at prolonged intervals in eastern and southern Africa. It has been accepted that the virus is endemic in indigenous forests, where it circulates in mosquitoes and vertebrates, spreading to livestock-rearing areas when heavy rains favour the breeding of mosquito vectors. The *Phlebovirus* is transmitted by mosquitoes from 23 species belonging to the *Anopheles, Culex, Aedes and Mansonia genera*. RVF in ruminants is mainly inapparent in non-pregnant adult animals, but in outbreak situations, it is responsible for many abortions and high neonatal mortality, particularly in European breeds and NWC crias. The virus was first isolated in 1931 in livestock on a farm located in the Rift Valley of Kenya. The virus is now endemic in much of sub-Saharan Africa, with epidemics occurring also in West Africa (Saluzzo *et al,* 1987). It has also spread into Egypt, Sudan, Kenya and Mauritania and clearly has the potential to spread elsewhere with climate change and ever increasing unpredictable flooding in different parts of the world. In South Africa, RVF occurs at regular intervals: the first known outbreak was reported in the 1950s, a major outbreak in 1974–1976 and small localised outbreaks in 1981 and 1999. In 2008, RVF reoccurred, and isolated outbreaks were reported in 2009, followed by an extensive epidemic in 2010 affecting most parts of South Africa. In 2000, the disease for the first time affected humans and livestock outside Africa, when it was diagnosed in the southern parts of Saudia Arabia and Yemen (Khan *et al,* 2002; Shoemaker *et al,* 2002). The globalisation of trade and altered weather patterns are a concern for the future spread of RVF, as the causative agent, the RVFV, is capable of utilising a wide range of mosquito vectors. In the last outbreak in Mauritania, at the end of September to the beginning of October 2010, unprecedented rainfall created large ponds of water in the oases of the Saharan region of Adrar, northern Mauritania. Such rain had not been observed for decades, and the locals refer to 1956 (locally known as the 'year of the fever') to describe the recent similar events.

This climatic event lead to unusually profuse growth of vegetation, attracting shepherds and pastoralists from remote areas, including the south and south-east regions of the country. It also favoured the multiplication in high densities of several species of mosquito, mainly from the genera *Culex* and *Anopheles (Culex quinquefaciatus, Anopheles pharoensis, Anopheles protoriensis, Culex poicilipes, Anopheles gambiae, Aedes vexans, Culex antenatus, Anopheles rufipes, Mansonia uniformis, Anopheles ziemani)*, including competent vectors for major arboviruses.

A few weeks after this period of rain, severe outbreaks of malaria and RVF were reported in several oases ('Graret') of the Adrar region. Interestingly, the first probably reportable case in livestock was a dromedary camel that became sick during the last week of October 2010 in the Aoujeft area (Ould Al Mamy *et al,* 2011). Dromedaries in the area exhibited an RVF immunoglobulin M (IgM) seroprevalence of 33% and suffered from two clinical forms: a peracute form with sudden death within 24 hours and an acute form with fever, ataxia, blood-tinged nasal discharge, blindness, haemorrhages of the gum and central nervous system signs. Abortions also occurred. The virus was isolated from four dromedaries. It seems that dromedaries play a major role in the epidemiology of RVF outbreaks.

Epidemiological studies of RVF have always been performed during episootics or immediately afterwards. This was the case for epidemics in Sudan, Kenya and Egypt. Several studies also included the local dromedary populations. Scott *et al* (1963) reported outbreaks of RVF in cattle, following severe rainfall in Kenya, parallel to a drastic increase in abortions in dromedaries. Antibodies to RVF were found in 45% of the dromedaries examined during this outbreak. The authors stated that the RVFV was responsible for the increased rate of abortions; however, no virological studies were performed to substantiate this supposition. Meegan *et al* (1979) also observed an increased abortion rate in dromedaries during an RVF epizootic in Egypt. In this case, the epidemic was supposedly carried by Sudanese dromedaries to Egypt (Abd El-Rahim *et al,* 1999; Hoogstraal *et al,* 1979), as severe epidemics were raging in northern Sudan at the time (Eisa *et al,* 1977). During this period, Hoogstraal *et al* (1979) registered 31 RVF reactors in dromedaries. Other than the increased abortion rate during outbreaks of RVF, no other clinical signs have been observed so far in camels (Davies *et al,* 1985). Aly (1979) found antibodies with the Haemagglutination inhibition (HI) test in 15.6% of dromedaries in Egypt, and Walker (1975) described abortions and deaths in

young one-humped camels during RVF outbreaks. Peters and Meegan (1981), however, observed only a subclinical form of RVF. Olaleye *et al* (1996) examined 180 dromedaries with the haemagglutination inhibition test and the serum neutralisation test in Nigeria and detected 3.3% of cases to be positive. The authors stressed the involvement of camels in the transmission cycle of RVFV. Out of 1,119 serum samples from dairy camels in Dubai, four (0.35%) were positive in the RVF inhibition enzyme-linked immunosorbent assay (ELISA), which has, according to Paweska *et al* (2005), a 100% specificity and sensitivity for camelids. These four samples were from adult dairy dromedaries. None of the calves had seroconverted. The low incidence of RVF is not unexpected because the Arabian Peninsula experiences very little rainfall, unfavourable for the breeding of high numbers of mousquitoes. However, it should be kept in mind that camels which are imported from countries that regularly experience RVF outbreaks should be tested (Wernery *et al,* 2008).

Imam *et al* (1978) and Eisa (1984) were able to isolate the virus from a healthy, naturally infected dromedary. Experimental infections with the RVFV have failed to induce clinical signs in non-pregnant dromedaries (Davies *et al,* 1985). In spite of high RVF antibody titres, the same authors were not able to determine an increased rate of abortion in infected dromedaries.

Severe RVF epidemics have recently occurred in East Africa (Anonymous, 1998). Many domestic animals and humans were affected in vast areas of Kenya, southern Sudan and northern Tanzania in December 1997 and January 1998. It was claimed that the 1998 Kenyan outbreak most probably eliminated the entire camel calf population, as perhaps as many as 150,000 animals died (Mungere, 2000). During the Mauritanian outbreak in 1998, immunoglobulin G antibodies were detected in dromedaries, but no virus was isolated (Nabeth *et al,* 2001). It is of great relevance to carry out proper investigations to elucidate the role of RVF in dromedaries because of the zoonotic potential of this disease. Intensive investigations were conducted by Munyua *et al* (2005) during the 2006–2007 RVF outbreaks in Kenya in which thousands of cattle, sheep, goats and dromedaries were affected. Again, abortion was the main clinical sign in dromedaries, with a prevalence of up to 38%. However, no attempts were made to isolate the virus from aborted foetuses or placentas. Reports emerged in 2011 of many llamas and alpacas dying from RVF in 2010 and 2011 in South Africa. South Africa's NWCs are used to guard sheep from predators, such as jackals and leopards, and on some farms they are also reared for their wool, which has a cashmere quality. The disease broke out in the Western Cape Province in January 2010. Not all alpacas die when infected with the RVFV; some get flu-like clinical signs and recover quickly. In these outbreaks, the morbidity rate was 12.5% and the mortality rate 0.13%. Interestingly, although the farm on which the fatalities occurred is an alpaca enterprise comprising alpacas of all ages, only the yearling group was affected. The disease was also observed in young llamas. Rift Valley Fever outbreaks in South Africa occurred in 2009, 2010 and last in 2011. Although South Africa has an alpaca population of around 3000 only 22 animals succumbed to RVF in 2010 and 2011.

In 2024 and 2025, disease outbreaks were reported again in some parts of Ethiopia and Kenya after heavy rain in these areas. Several clinical signs observed in dromedary camels during these outbreaks were similar as described by Oud El Mamy *et al* (2011) with fever, staggering, ventral oedema, blood stinged nasal discharge, severe conjunctivitis, abortions, oedema of neck and head. Currently serological and viral testing is performed and will be reported later.

Clinical signs

During the most recent RVF outbreaks in East Africa, the WOAH (OIE) received many reports of high mortality in camels throughout the affected areas. Some descriptions of morbidity and mortality were highly suggestive of camelpox or parapox (*Ecthyma contagiosum*), with ballooning of the head and upper neck, swollen eyes and huge mucoid membrane sloughs in the mouth covering some ulcers.

However, the general disease pattern was that of fever and abortion, which were the predominant features, but early neonatal death and jaundice were also observed. As RVFV was not isolated from camels during these outbreaks, it is not clear whether the disease was caused by RVF. However, death and abortions in many dromedaries were observed in the Saudi Arabian RVF outbreak of 2000 and the 2006 outbreak in Kenya (WOAH, 2007), but no thorough investigations were carried out. The clinical signs of RVF in NWCs were described during the outbreak in South Africa. The disease had an abrupt onset and short course, lasting only between 4 and 6 h. The four affected alpaca crias were anorexic,

severely depressed and pyrexic and showed signs of abdominal discomfort. They also developed respiratory distress, and, despite supportive therapy, they died. During necropsy, widespread petechiae and ecchymoses were seen in the buccal mucosa, subcutis, serosal surfaces, epi- and endocardium, liver, lungs and lymph nodes. Histologically, the liver showed coagulative to lytic necroses. In two animals, intranuclear inclusions were detected, and real-time polymerase chain reaction (PCR) and immunohistology staining confirmed RVF (Gers and Grewar, 2010).

In general, RVF affects a wide range of animal species, but the severity of clinical signs varies according to age, and, in sheep, peracute, acute, subacute and inapparent RVF forms have been described. In young bovine calves under ten days of age, the acute form, with hyperthermia, profuse fetid diarrhoea and depression combined with respiratory distress, is frequent. Death is very rapid (within 48 h), and the mortality rate may exceed 50%. Goats are generally considered to be more resistant to RVF than sheep, but kids may be affected and show the same clinical signs as lambs.

In adult dromedary camels, after a brief period of viremia, abortion seems to be the only visible sign of infection which can also be caused by brucellosis, camelpox and *Trypanosma evansi* infections. In younger dromedary camels, Oud El Mamy *et al* (2011) were the first who described the clinical features which were fever, ventral oedema, swollen head, staggering, severe conjunctivitis, blood tinged nasal discharge with haemorrhages of eyes and tongue. These lesions were also seen in the 2024, 2025 outbreaks in Kenya and Ethiopia (Cran, Pers. Communication 2025). Gross pathological lesions were icteric enlarged liver, petechiae on organ surfaces and free blood in abomasum and intestines which resemble alterations seen in entertoxaemias or haemorrhagic diathesis (HD, Wernery *et al,* 2014). RVF has not been documented in Bactrian camels, as RVF outbreaks have not been reported where Bactrians are reared.

Diagnosis

Diagnostic techniques are well described in the WOAH (OIE) chapter "Rift Valley Fever" (OIE, 2018). The definitive diagnosis of RVF depends on virological and serological investigations, as other arthropod-borne virus diseases tend to occur under the same climatic conditions. This is especially true for Wesselsbron disease, which can also cause mortality in lambs, kids and calves and abortion in ewes. However, RVF is associated with higher mortality and abortion rates. Lesions in the livers of young animals also differ in RVF and Wesselsbron disease. Wesselbron disease have not been described in *Camelidae*. Hepatic changes are usually less extensive in RVF than in Wesselsbron disease. Specimens for laboratory confirmation should include heparinised blood, liver, spleen, kidney, lymph nodes and brain from aborted foetuses for virus isolation on Vero and BHK 21 cells or suckling and weaned mice. Antibodies to RVF can be demonstrated by the complement fixation test, agar gel immunodiffusion test, Haemagglutination inhibition test and ELISA. Results by Paweska *et al* (2005) showed that the inhibition ELISA is a highly accurate diagnostic tool for RVF disease surveillance and control programmes, and for monitoring the immune response after vaccination. It has the advantage of being independent of the species tested. This was confirmed by Martin-Folgar *et al* (2010), who tested dromedary sera with the competative ELISA after the 2006/2007 RVF outbreak in Kenya. Viral antigen can also be detected by immunofluorescence in impression smears of infected tissues. It is noted that the available IgM ELISA kits are not validated in camels.

Treatment and prevention

Measures, such as the chemical control of vectors, movement of livestock to higher altitudes or the confinement of animals to mosquito-proof stables, are usually impractical or too late. Immunisation remains the only effective way to protect livestock. The main purpose of a RVF vaccine is to prevent the spread of the virus into species of economic interest like cattle, sheep, goats and camelids and limit the impact on animal and public health.

Although it has still not been determined decisively whether dromedaries actually develop RVF, Guillaud and Lancelot (1989) have concerned themselves with the production of a vaccine. The authors determined that the attenuated vaccine strain MVP-22 has yielded satisfactory results in the dromedary. Following a single subcutaneous vaccination, 18 of 22 dromedaries developed neutralising antibodies. A challenge infection with the RVFV was not performed.

Currently, there is no specific RVF vaccine designed for camelids. It is not known whether the sheep vaccine, which has been used in South Africa to protect alpacas from RVF, is effective or not, and this cannot be determined until proper vaccine trials

have been conducted. In Dubai, two dromedaries were vaccinated with a live attenuated (Namibia: NSR 0580) and an inactivated (Namibia: NSR 0966) vaccine, both of which were from Onderstepoort and designed for cattle, sheep and goats. With a cattle dose, even after five injections, no antibodies were detected using the inhibition ELISA.

Further serological investigations with an inactivated RVF vaccine should be initiated, as the number of dromedary camels used in this immunisation was far too small. However, it is known that formalin inactivated RVF vaccines gave a short-lived immunity in cattle and require multiple administrations. Vaccination with classical live attenuated RVF vaccines has been associated with foetal malformations, still birth and foetal demise and should therefore be only used in non-preganant animals. However, in cattle they give a life-long immunity and may be tried in camels too. Two live attenuated vaccines are commercially available: Smithburn strain, which was isolated in Uganda, and clone 13 vaccine. Both vaccines are effective with one administration. Clone 13 vaccine is a natural RVFV isolate that contains a large (70%) deletion in its NSs gene. This vaccine appears to be a very good potential vaccine strain without any side-effects (Wichgers Schreur *et al,* 2023; Wichgers Schreur *et al,* 2025). However, a large experiment with pregnant ewes demonstrated that the clone 13 RVFV vaccine spread to the foetus, resulting in malformation and still births (Makoschey *et al,* 2016). Next generation live attenuated RVF vaccine are in the making and recombinant vaccines using either Capripox virus or vaccinia virus have been developed but not really used in proper field trials.

Conclusion

It is now agreed that NWCs and OWCs cannot only contract a RVFV infections but also develop a disease. In adult dromedary camels the main clinical sign of RVF is abortion at any stage and young stock may develop a systemic disease. RVF outbreaks are regularly observed after heavy rains in many different African countries and climate change has the potential that RVF virus may enter countries outside Africa. Inactivated and live attenuated RVF vaccines are available and have not been scientifically evaluated for the use in *Camelidae*. Both have their advantages and disadvantages in ruminating animal species.

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