Rift Valley fever (RVF)

Author: Prof JAW Coetzer
Licensed under a Creative Commons Attribution license.

Introduction
Rift Valley fever (RVF) is a primarily mosquito-borne, multi-species zoonotic viral disease of which the causative organism, Rift Valley fever virus, was first isolated in the early 1930s in the Rift Valley in Kenya. It is the cause of severe disease in small stock resulting in high mortality and abortion storms particularly in sheep and goats.

Rift Valley fever is a good example of a disease where the principles of the One Health concept are applicable. Proper studies or control strategies of Rift Valley fever required a more integrated or holistic approach to human, animal and ecosystem health and collaborative efforts of multidisciplinary teams working nationally and internationally. The expected environmental or ecosystem changes in large parts of Africa as a results of climate change may well have a significant bearing on the ecology or epidemiology of the disease and frequency of outbreaks in animals and humans.

Salient features of RVF
Rift Valley fever is caused by a Phlebovirus of the family Bunyaviridae. No significant antigenic differences have been detected between Rift Valley fever virus isolates and laboratory passaged strains from many countries.

The disease is mainly of economic importance in sheep, goats and cattle with new-born animals being most susceptible. Deaths in wild animals are rare, but mortality has been reported in several wildlife species, including African buffaloes, waterbuck, giraffe, sable and springbuck.

Hepatic disease occurs in all species but it is most severe in extremely susceptible hosts, such as new-born lambs and kids. In these hosts hepatic lesions rapidly progress to a massive necrotic hepatitis just before death. In less susceptible animals, such as adult sheep and goats, the hepatic lesions tend to be more focal in nature.

The haemostatic derangement which manifests as a viral haemorrhagic fever with a bleeding tendency and disseminated intravascular coagulopathy is most severe in the fatal hepatic syndrome in animals and humans.

In new-born lambs and kids the incubation period is usually 24-36 hours. In animals less than one week old mortality is 90 % or more. In adult sheep mortality rates varying from 5 to 30% and abortion rates of 40 to 100% have been reported in outbreaks. In adult cattle it is usually an inapparent infection, but some develop peracute or acute disease. The mortality rate is 0-5% and about 10% in adult cattle and calves respectively. An abortion rate of up to 40% may occur in pregnant cows.

The hepatic lesions of RVF are essentially similar in all domestic animals and humans, varying mainly with the age of the affected individual. The most severe lesions
occur in aborted sheep foetuses and new-born lambs in which the liver is usually moderately to greatly enlarged, soft, friable and yellowish-brown to dark reddish-brown in colour. The hepatic lesions in new-born lambs are almost invariably accompanied by numerous petechiae and ecchymoses in the mucosa of the abomasum, and its contents are dark chocolate-brown as a result of the presence of partially-digested blood. Rift Valley fever is often a very haemorrhagic disease in cattle.

The vast majority of human infections result from direct or indirect contact with the blood or organs of infected animals. The virus can be transmitted to humans through the handling of animal tissue during slaughtering or butchering, assisting with animal births, conducting veterinary procedures, or from the disposal of carcasses or foetuses. The majority of RVF infections in humans is inapparent or is associated with moderate to severe, non-fatal, influenza-like illness, but a minority, probably less than 1%, develops ocular lesions, encephalitis or severe fatal hepatic disease with haemorrhagic manifestations.

Protective clothing such as gloves and masks should be used when doing necropsies on suspected cases of RVF or handling infected tissues. No vaccine is currently freely available to protect humans against the disease.

Where does RVF occur?

With the exception of the 2000-2001 outbreak on the Arabian Peninsula all other outbreaks or serological evidence of Rift Valley fever have been limited to the African continent, Madagascar and the Comoro Islands.

For most of the 20th century, outbreaks were confined to countries in Africa. The virus was isolated for the first time outside continental Africa in 1979 in Madagascar where the virus is now endemic. In 2000-2001 Rift Valley fever virus was introduced across the Red Sea and caused a major outbreak of disease in livestock and humans in Saudi Arabia and Yemen on the Arabian Peninsula. During 2008, it was detected on the French island of Mayotte in the Archipelago of Comores, located between Mozambique and Madagascar.

The epidemiological characteristics of the disease justify the perception that the unusually wide range of mosquitoes that can transmit the virus, and the sufficiently high levels of virus that circulate in livestock to infect mosquitoes, would render many parts of the world receptive to the virus.

What triggers an outbreak of RVF?

Epidemics, associated with above average rainfall, have tended to occur in eastern, central and southern African countries usually at irregular intervals of 5 to 15 years or longer. Sporadic cases or small outbreaks occurred between epidemics.

The outbreaks of Rift Valley fever in countries in North and West Africa occurred independently of rainfall in dry regions, apparently in association with vectors which breed in large rivers and dams.

It is currently postulated that Rift Valley fever virus in sub-Saharan Africa is maintained in inter-epidemic periods principally by transovarial transmission in aedine mosquitoes particularly in areas where there are dambos or broad vleis, with a low level of transmission to livestock or wildlife. Aedine mosquitoes overwinter as eggs. The eggs can survive for long periods in dried mud possibly for several seasons if pans, dambos or vleis remain dry.

Maintenance of virus during the inter-epidemic period is possible by means of vertical (transovarial) transmission by Aedes species. Serological surveys in cattle and wildlife indicate that varying amounts of virus activity occur each year in certain areas in eastern and
southern Africa without epidemics occurring. In southern Africa the onset of epidemics tends to be recognized late in summer. It is thought that epidemics are precipitated by abnormally heavy rains which lead to an explosive increase in epidemic mosquito vectors and spread of the disease from these endemic foci.

The flooding of dambos or vleis and the humid weather conditions prevailing in epidemics favour the breeding not only of the aedine maintenance vectors such as Aedes mcintoshi, Aedes unidentatus and Aedes juppi and the non-aedine mosquitoes such as Culex and Anopheles species which serve as epidemic vectors. Contagion is not considered to be important in livestock.

**Prevention and control**

One should suspect Rift Valley fever when heavy rains are followed by the occurrence of abortions in sheep, goats and cattle together with fatal disease, particularly in young animals, which is characterized by necrotic hepatitis and haemorrhages in the abomasum and serosal surfaces. Frequently there is also an influenza-like illness in humans closely associated with livestock industries.

Specimens to be submitted for laboratory confirmation of the diagnosis include heparinized or clotted blood, plasma or serum of live affected animals, or tissue samples, including liver, spleen, kidney, lymph nodes and heart blood of dead animals. Specimens should be securely packaged and submitted on ice to a suitable laboratory for isolation of virus or demonstration of antibody.

Virus can be isolated readily in a variety of cell cultures, or in suckling and weaned mice or hamsters inoculated intracerebrally or intraperitoneally. Viral antigen detection is rapidly done in tissue sections by immunoperoxidase staining and in serum by ELISA. Viral nucleic acid can readily be detected in serum and other tissues of infected livestock and humans, as well as mosquitoes by means of a variety of highly sensitive polymerase chain reaction assays.

In animals that survive the disease, paired serum samples, one taken during the acute illness and the other 2-3 weeks later, should be submitted for retrospective diagnosis by means of antibody detection.

Tissue specimens from the liver, spleen, and lymph nodes should also be collected in 10% buffered-formalin for histopathological examination.

In southern Africa, outbreaks tend to terminate abruptly soon after the first frosts of winter which suppresses vector activity. In contrast, virus activity may persist in those parts of Africa which experience warmer winters. Vector control is of limited or no use in the control of Rift Valley fever and immunization remains the only effective way to protect livestock.

Epidemics of RVF tend to occur at irregular intervals of many years and it is usually difficult to persuade farmers to vaccinate livestock during long inter-epidemic periods. Hence it is advisable in African countries with large sheep and goat populations to immunize the offspring of vaccinated ewes and nannies on a regular basis at 6 months of age, when colostral immunity has waned, with a single dose of the modified live Smithburn vaccine. The Smithburn virus is unfortunately not entirely avirulent. A range of anomalies of the central nervous system may occur.

It is advised that only inactivated vaccines should be used when it is considered necessary to immunize animals in countries where the presence of Rift Valley fever has not been proven. In contrast to the live Smithburn vaccine, formalin-inactivated vaccines are safe for use even in pregnant animals.
The shortcomings of the Smithburn vaccine have led to continuing research, and several vaccines have been on trial during the past 3 decades but few have yet passed the rigorous evaluation for successful registration. A new avirulent candidate virus (clone 13), was found to be safe and efficacious.

The economic losses associated with epidemics of Rift Valley fever are considerable, and include inter alia mortality and abortion particularly in sheep and goats, international trade bans, and the costs involved in the production, distribution and administration of vaccines. When mortalities occur among valuable species of wildlife, the cost can be very high and the concomitant ban on hunting and translocation of wild animals can have a devastating effect on the hunting industry and the sale of wild animals.

Rift Valley fever is a disease that must be reported to the World Organization for Animal Health by the veterinary authorities of member countries.

Find out more
The CPD course on RVF describes its history, aetiology and epidemiology, how to recognise it and confirm the diagnosis, its potential for transboundary spread and the challenges of controlling the disease.