Rift Valley fever

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Summary
Rift Valley fever (RVF) is an arthropod-borne viral disease of ruminants, camels and humans. It is also a significant zoonosis which may be encountered as an uncomplicated influenza-like illness, but may also present as a haemorrhagic disease with liver involvement; there may also be ocular or neurological lesions. In animals, RVF may be inapparent in non-pregnant adults, but outbreaks are characterised by the onset of abortions and high neonatal mortality. Jaundice hepatitis and death are seen in older animals. Outbreaks of RVF are associated with persistent heavy rainfall with sustained flooding and the appearance of large numbers of mosquitoes, the main vector. Localised heavy rainfall is seldom sufficient to create conditions for an outbreak; the simultaneous emergence of large numbers of first generation transovarially infected mosquitoes is also required. After virus amplification in vertebrates, mosquitoes act as secondary vectors to sustain the epidemic.

Keywords

Introduction and history
A zoonosis may be defined as a disease that is naturally transmitted between humans and animals. The most serious zoonoses are viral, and Rift Valley fever (RVF) falls into this category. This arthropod-borne infection affects a wide range of vertebrates, but clinical disease is limited to domestic ruminants and humans. The course of the disease varies with host species and may be inapparent to peracute.

Rift Valley fever virus was first discovered and characterised in the Rift Valley of Kenya in 1931 (12), but outbreaks probably occurred before this time.

Many sub-Saharan tropical and sub-tropical countries in Africa have reported outbreaks of RVF and the disease is encountered in an enzootic or epizootic form along the east and south coast of Africa and also in Madagascar. The virus has spread as far north as Egypt and more recently an outbreak occurred in the Arabian Peninsula (Fig. 1). Large outbreaks have occurred in South Africa, which were particularly severe between 1950 and 1951 and even more widespread between 1974 and 1975. Because clinical disease is generally not evident in indigenous breeds, RVF has not been identified as a livestock problem in many other countries in Africa, although large losses in indigenous livestock have occurred in Egypt and West Africa. There have been no Rift Valley fever epizootics in smaller countries such as Mali, Gabon, Congo, Chad, Botswana, Angola, Nigeria and Uganda, but evidence of antibodies to the disease has been found in humans and/or livestock. Large outbreaks in cattle, manifested by abortions, have been reported in Zimbabwe and Zambia. Monitoring of sentinel cattle in Zambia indicates an annual emergence of the virus after the seasonal rains, with antibody prevalence varying from 3%-8% to 20% (14).

The ability of RVF to move outside traditionally endemic areas, or even out of the continent of Africa, lies in the fact that there is an unusually large range of vectors capable of transmitting the virus and a level of viraemia in ruminants and humans that is sufficiently high to infect mosquitoes. Such a movement of the virus occurred following the
El Niño floods of 1997-1998 in the Horn of Africa countries, and there was an RVF epizootic in the Arabian Peninsula in 2000. Although it has not been proven that the virus was carried from infected to non-infected areas by viraemic animals, it is a possibility in this case, given the proximity of the Saudi and Somali port cities and the accelerated trade which takes place just before the Hajj festival. Viraemic animals may also have had a role to play in the spread of RVF from Northern Sudan into Egypt in 1977. This type of transmission deserves further consideration in a world where globalisation of trade and short transport times are now the norm.

The importance of Rift Valley fever for animal and public health

The words attributed to Louis Pasteur ‘the microbe is nothing, the terrain is everything’ are particularly applicable to RVF. Flooding in northeast Africa in the El Niño year of 1997-1998 gave rise to the most important epidemic of RVF ever recorded. The greater than normal rainfall caused the combined flooding of two rivers in Somalia, and further flooding in Kenya, creating a huge inland lake and setting up the conditions for an outbreak of RVF that affected 89,000 people in Kenya and Somalia and caused 250 deaths.

Other pathogens, including haemorrhagic fever viruses, circulated in the hot, moist conditions, which were described by the World Health Organization (WHO) as ‘a viral soup’. Bluetongue killed many sheep in Kenya, and the final number of livestock deaths was greatly increased because of the countless animals that drowned (32).

In addition to flooding, changing land use is another major factor in the emergence of disease, and in the case of RVF this is reflected in the building of dams. Up to 1977, RVF had been seen largely as a veterinary problem in Africa, but in Egypt in that year, six years after the completion of the Aswan dam, more than 200,000 people became sick, 598 deaths occurred and livestock losses resulted in meat shortages (18). The mode of spread of the virus, from an isolated focus in Northern Sudan to the Nile valley, is unclear: it could have been spread by infected stock or people, or transported by mosquitoes (1). The dam had created many hectares of floodlands and increased the breeding ground of mosquitoes, adding impetus to the outbreak. Rift Valley fever was not enzootic in Egypt before 1977 and the virus disappeared in 1981, before re-emerging in 1993.

A second dam-linked incident occurred in 1987 in West Africa. One year after the construction of the Diama dam along the Senegal River, an outbreak of RVF was recorded in northern Senegal and Mauritania. Many thousands of people became sick and about one fifth of those died (22). Abortion losses in livestock were also heavy. This incident, which was the result of the mass emergence of mosquitoes in northern Senegal, was interesting, given the fact that the first RVF virus isolate from mosquitoes in West Africa was discovered as early as 1974 in southern Senegal and that the virus appears to be enzootic in the livestock there (45).

The East African outbreaks, particularly the earlier ones, were considered to be ‘veterinary/animal epidemics’. For example, in the 1930-1931 hepatitis outbreak in the Rift Valley of Kenya there were heavy losses in sheep due to death and/or abortion. In one documented example, 60 out of 80 lambs aged three to seven days died in the space of 24 h and there were also reports of 3,500 lambs and 1,200 ewes being lost in a single month (12). In South Africa between 1950 and 1951, 100,000 sheep died and 500,000 aborted. Similar heavy losses occurred in cattle in Zambia and Zimbabwe.

However, the Egyptian outbreak of 1977 and the West African outbreak of 1987 in Senegal and Mauritania were ‘people epidemics’. The outbreak in the Nile valley and delta caused nearly 600 human deaths. Among the 200,000 people who became sick, retinitis was present in a large proportion of infected individuals, as well as liver necrosis, haemorrhagic fever, and encephalitis (24). Ocular involvement was characterised by retinal macular lesions and was first noticed as an abrupt loss of visual clarity. The
habitats in an endemic cycle between mosquitoes and an unknown vertebrate host. Alternatively there may be virus activity each year, with low-level transmission to livestock associated with Aedine mosquitoes that breed in low-lying depressions which flood when abnormally heavy rainfall raises the water table sufficiently. (In sub-Saharan Africa, RVF is endemic because of transovarial transmission in Aedes Neomelaniconion.)

Once there is evidence of past virus activity, countries are likely to remain permanently infected. Some 20 countries in Africa and Madagascar are infected and 23 species of mosquitoes are involved in the epizootic/enzootic transmission cycles of RVF. Countries in north and west Africa are arid and outbreaks occur independently of rainfall, with mosquitoes that breed in large rivers and dams, not floodwater aedines. In Egypt, there are no mosquitoes capable of transovarial transmission and the major vector is a Culex sp. which breeds in polluted waters (25).

The human toll in Mauritania was also high, with over 200 deaths.

Aetiology and classification

The Bunyaviridae is a large family of viruses and contains five genera, four of which infect vertebrates, while the remaining genus, Tospovirus, contains a group of plant viruses. Three of the vertebrate-infecting genera, Bunyavirus, Phlebovirus and Nairovirus are associated with arthropods (and are further subdivided into complexes of closely related members), while the last genus, Hantavirus, has no known invertebrate association.

Rift Valley fever virus, in physical, chemical and morphological terms, is a typical member of the Bunyaviridae of the genus Phlebovirus. This genus also includes the sandfly fevers. Rift Valley fever virus is an enveloped spherical virus of up to 120 nm in diameter, with short glycoprotein spikes projecting through a bilayered lipid envelope (Fig. 2). The single stranded ribonucleic acid (RNA) genome is divided into three segments each in its own nucleocapsid. These segments are the large (L), medium (M) and small (S) segments expressing many copies of the N (nucleocapsid) and few copies of the L (transcriptase) structural proteins (46).

The S segment has bi-directional coding or ambisense RNA while the other two segments have negative sense RNA genomes (37).

Epidemiology and transmission

The distribution of large RVF outbreaks is neither seasonal nor annual, but linked to the presence of water. Water is important to most blood-feeding insects, as they have aquatic or semi-aquatic immature stages and this automatically limits the choice of breeding sites. In the south and east of Africa, outbreaks of RVF are associated with heavy rainfall, while in the drier north and west they are linked to irrigated lands. Epizootics of RVF are cyclical in nature and characterised by long inter-epizootic periods. These cycles may be comparatively short, i.e. five to fifteen years in wetter areas, or much longer, i.e. from fifteen to thirty years or more where it is drier. In the inter-epizootic periods the virus may be present in forest-edge habitats in an endemic cycle between mosquitoes and an unknown vertebrate host. Alternatively there may be virus activity each year, with low-level transmission to livestock associated with Aedine mosquitoes that breed in low-lying depressions which flood when abnormally heavy rainfall raises the water table sufficiently. (In sub-Saharan Africa, RVF is endemic because of transovarial transmission in Aedes Neomelaniconion.)

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In the rest of Africa, inter-epizootic survival depends on transovarial transmission of the virus or venereal transmission between mosquitoes with low-level circulation in livestock. In Egypt, new outbreaks are the result of infected adult mosquitoes coming out of hibernation, re-introduction of the virus via the transport of infected animals, or wind-borne transmission from infected neighbouring countries. The latter is thought to have occurred in 1977 in Egypt, when unusual southerly winds were documented for a week at the end of
July/beginning of August, probably bringing large numbers of mosquitoes up from the north of Sudan. The waters of the Aswan dam had already significantly increased mosquito-breeding sites in Egypt.

Rainfall, and the presence or absence thereof, is an important factor in the establishment of breeding sites, and it is above average and sustained rainfall which creates conditions for an outbreak of RVF. Persistent rainfall raises the water table and floods grassland and the shallow depressions which characteristically dot the plateau regions of some African states. Eggs of the ground pool breeding Aedes spp. are attached to vegetation at the edge of these depressions, and when they flood large numbers of floodwater breeding mosquitoes emerge (13). Outbreaks may be less frequent and less widespread in highly populated areas where there is intensive land use, as this decreases the presence of water and thus reduces the number of mosquito breeding sites.

Epidemics do not occur as a result of lateral spread from a single source, but because of the intensification of vector activity due to the simultaneous emergence of a great number of vectors. This would explain why localised heavy rainfall does not produce an epidemic. When outbreaks do occur there are often large distances between foci of infection, and one theory being tested to account for this is that mosquito eggs may be dispersed by wading birds (National Institute for Communicable Diseases, Johannesburg, South Africa, personal communication). Secondary, opportunistic mosquitoes then serve to propagate the epizootic.

There are many arthropods involved in the transmission of RVF – whether by biological or mechanical means. In southern Africa alone, the virus has been isolated from wild mosquitoes of the Aedes, Culex, Anopheles, Mansonia and Eretmapodites species. The floodwater-breeding aedines are of particular importance in the sub-genera Aedimorphus and Neomelaniconion. In Africa alone, 23 species of mosquito from five genera are involved and, while mosquitoes are the most important arthropod vectors, virus may be mechanically transmitted by Culicoides, Simulium, and even tick species. There is a complex cycle of enzootic maintenance and epizootic transmission of the virus by different mosquito species. Transovarial transmission within the Aedes species allows a single generation of infected mosquitoes to emerge after flooding. These infect some livestock, and if the pools created by the floodwater do not disappear then other mosquito species breed rapidly and act as secondary vectors.

There are two different types of secondary cycles by which the virus has been transmitted by zoophilic mosquitoes. In South Africa between 1974 and 1976, 14.5% of people working in this industry were seropositive, with seven reported deaths. Urban consumers of animal products are not affected by the sylvatic cycle as the virus does not survive below pH 6, and the pH of meat generally falls to 6 or less during processing (41). The second type of secondary cycle is the urban peridomestic cycle, in which humans become infected with RVF through the bites of anthropophilic mosquitoes. The large number of human cases in Egypt in 1977 and 1978 occurred largely as a result of this type of transmission; the antibody prevalence in people reached 29.6%, and there were 598 recorded deaths. Human transmission takes place when the virus comes into contact with abraded skin or mucous membranes (aerosol with intranasal infection is also a possibility and is thought to have occurred in Saudi Arabia during the ritual slaughter of animals). There is also circumstantial evidence that infection can occur through contact with raw milk.

There appears to be little contact transmission between animals in spite of the presence of virus in nasal discharge and saliva. Laboratory infection has been reported and people at risk are now vaccinated and required to take protective measures.

Rift Valley fever transmission and the effects of global warming

At this point, it is also worth considering global warming and its effect on extending the range of RVF vectors. Global warming is set to modify temperature, rainfall, wind and sea-levels and will therefore alter vector, vertebrate and virus interactions. One American study on the effects of global warming on mosquito-borne arboviruses reported that two American mosquito species had moved northwards to slightly cooler areas to escape escalating temperatures (33). Various other studies have been undertaken, including a WHO Task Group investigation, the results of which were released in a 1990 report on the ‘potential health effects of climate change’ (43). The sensitivity of diseases and their vectors in relation to global temperature changes was rated on a scale of zero to three. Among the dozen human illnesses evaluated none scored zero or ‘no effect’. Malaria scored three, dengue two, and all other insect-borne viral diseases scored one.

The biology of infection

Diagnosing RVF can be a challenge because while some outbreaks are obvious there are also cryptic cycles which are not seen. Severe clinical disease in young animals indicates that a breed is highly susceptible to the disease.
Table I lists the host range of vertebrates according to their degree of susceptibility to the virus, ranging from highly susceptible to resistant (31).

Epidemics are characterised by sudden abortion storms, almost 100% mortality in newborns of less than one week old and an influenza-like illness in humans (2). Complications may include hepatitis with generalised haemorrhages and cerebral or ocular infections in some individuals (15). Camels, which do not exhibit disease will, however, abort, as do buffalo. Newborn kids and lambs are most susceptible and mortality may vary from 70% to 100%. The disease follows a peracute course, with collapse and death occurring 12 h after the onset of pyrexia (40°C to 42°C). After the incubation period, which can last from between 12 h and 36 h, animals become listless and exhibit abdominal pain. Adult sheep and goats or older lambs and kids are less susceptible and disease may vary from acute to inapparent, depending on the breed. There is an incubation period of 24 h to 72 h followed by an elevated temperature, lymphadenitis, and vomiting or diarrhoea. The animal is listless, becomes recumbent and may have haemorrhagic diarrhoea. Icterus can develop and abortion may occur. Mortality varies from 10% to 70%, although in adults with a more sub-acute form of the disease it may only reach 20%. Calves less than ten days old will also suffer a peracute form of RVF and die within 24 h. Icterus is more commonly seen in older calves and adult cattle and death in adults is often less than 10% (21). The animal develops anorexia, diarrhoea and dysgalactia and exhibits salivation and nasal discharge (17). Abortion may occur as a result of infection of the foetus or as a result of a febrile reaction. The foetus is often autolysed.

In indigenous breeds of African cattle, infection with RVF is often retrospectively diagnosed serologically and closer examination may then reveal abortions and a drop in milk production.

Wild ruminants seroconvert after inapparent infection but, as mentioned above, buffalo abort.

### Human infection

Disease in man is often an indicator of a larger problem, particularly in dry areas where livestock are not intensively managed and abortions are not evident. Human exposure to the virus is often occupational, either through handling infected livestock or their products or by breathing in aerosols released at slaughter. Mosquito bites, as seen in Egypt, and the consumption of raw milk have been documented as routes of exposure. In a high density population area like the Nile delta, man may even be considered to be an amplifying host, because humans may develop viraemia, thus enabling biting mosquitoes to transmit the virus to additional hosts.

Human illness may take one of four forms, as follows:

a) an uncomplicated, febrile, influenza-like illness

b) a haemorrhagic fever with liver involvement, thrombocytopenia, icterus and bleeding tendencies

c) encephalitis following a febrile episode with confusion and coma. Death is infrequent but there may be some residual damage

d) ocular involvement with reported blurred vision and loss of visual acuity due to retinal haemorrhage and macular oedema. There may also be residual damage (31).

Exposure to the virus is followed by a short incubation period of three to seven days, chills, headache and a biphasic fever. The patient experiences muscle and joint pains, nausea and tenderness in the liver area. In most cases, recovery is uneventful and complications are the exception rather than the norm.

### Pathogenesis and lesions

If an examination of blood is undertaken, the following three findings indicate the presence of RVF:

- profound leucopaenia

- elevated blood enzymes associated with severe liver damage

- thrombocytopenia.

The main sites of viral replication are the liver and spleen and often the brain (3, 30). The brain is a good specimen to submit in the case of an autolyzed foetus. In extremely susceptible animals, peracute liver damage is seen (16). Susceptibility decreases with age and adult sheep and goats are often only moderately susceptible, as are cattle and human beings. This varies according to breed and individual and a hepatic form of the disease may occur with vasculitis and haemorrhagic manifestations. The RVF virus replicates in many cells, and lesions in target organs are lytic in nature. Virus antigen has been demonstrated in...
the walls of small vessels, such as the adrenocortical cells and the glomeruli of the kidney, as well as in most areas of the spleen and all cells of the liver (42).

In an aborted foetus, virus may be found in visceral organs and the brain. Virus may be recovered from a placenta which does not show any visible lesions. Hepatic necrosis is the most common lesion observed in foetuses at autopsy and severe lesions are seen in newborn lambs. The liver is enlarged and congested and bronze to yellow in colour. It is soft and friable with a patchy congestion, haemorrhages and scattered gray-white necrotic foci (Figs 3 and 4). Because of the peracute nature of the disease the necrotic foci become indistinct in the severely congested discoloured liver (9, 11). Icterus is not seen in lambs, although in adult sheep with less severe lesions, this may be a feature.

Calves and cattle display more localised liver lesions, necrotic foci are visible and lobulation is more distinct (10). There is a generalised lymphadenopathy and often a severe haemorrhagic gastro-enteritis. The gut contents may be chocolate brown in colour, particularly in newborn calves. There is a blood-stained accumulation of fluid in the body cavities and widespread ecchymotic and petechial haemorrhages on subcutaneous and serosal surfaces. The wall of the gallbladder is oedematous with haemorrhages (Fig. 5).

A severe lytic liver necrosis with dense aggregates of nuclear and cellular debris, fibrin and inflammatory cells, is found in neonates and aborted foetuses of all species (Fig. 6). In adult animals necrosis is mostly multifocal and less diffuse.

Diagnosis

Single cases of RVF may be confused with many viral diseases of sheep where there is sudden death, generalised lymphadenopathy and haemorrhages throughout the carcass. However, RVF should be considered when there is a sudden onset of abortions at all stages of pregnancy, sudden death in young animals following an acute febrile
disease and obvious liver involvement in all cases. Such an outbreak may also be accompanied by severe flooding, the presence of large aggregates of mosquitoes and an influenza-like illness in human beings exposed to infected material. The presence of haemorrhagic fever in some individuals can create a sense of panic, sometimes leading to a public health crisis.

Liver lesions are pathognomonic for RVF, however, laboratory confirmation is advisable. In the case of live animals, specimens should include heparinised blood and serum, while the tissues of choice from dead animals are the liver, spleen, kidney, lymph nodes and heart blood. The brain of an autolysed foetus should also be included. Samples should be securely packed and labelled to indicate the dangerous nature of the contents. Specimens should be sent on ice at 4°C or, if there is likely to be a delay in transit, tissues may be sent in formalin for histopathology and also placed in glycerol or saline. Paired serum samples should be considered due to the confusing clinical presentation of RVF in adult animals.

Viral antigen may be rapidly detected using a reverse transcriptase polymerase chain reaction technique (34, 36). Virus replicates rapidly in infant or unweaned mice, on hamster or monkey cell lines such as Vero, BHK or CER, and on mosquito cells. Primary ruminant cells of calves and lambs may be useful as well.

Antibody tests include immunodiffusion, complement fixation, haemagglutination inhibition (HAI), indirect immunofluorescence (IFA) and virus neutralisation (8, 40). Immunoglobulin G (IgG) antibodies are produced early in an infection, but of the available enzyme-linked immunosorbent assay (ELISA) tests, immunoglobulin M (IgM) ELISAs are favoured for a rapid diagnosis (29).

Although African phleboviruses other than RVF are unlikely to cause disease or cause confusion in the diagnostic interpretation, it should be noted that there may be cross-reactivity associated with HAI tests. Screening tests may be done with HAI, IFA and ELISAs, but neutralisation tests are considered to be specific.

Laboratory infections are common and appropriate measures should be taken to protect staff members, including mandatory immunisation.

Surveillance and control

Rift Valley fever is intimately connected to sustained, heavy rainfall with flooding. Cycles are irregular and inter-epizootic periods may be long. Continuous control efforts may be impractical, but it is possible to monitor enzootic virus activity in livestock.

Routine screening of abortions, where practical, should be encouraged. Sentinel herd monitoring during the rainy season will yield useful data. Herds should be strategically placed near water sources which are known to flood and should consist of young seronegative animals. These animals should be regularly sampled for IgG/IgM antibodies. Data collected may alert authorities to the presence of virus emergence/circulation in sufficient time to contemplate control measures.

An even earlier warning system, five months in advance, using satellite weather data, was used to predict the RVF outbreak in Mauritania that erupted in 1987 after the completion of the Diama Dam on the Senegal River. This type of remote sensing satellite warning system collects the following data:

a) southern oscillation index (SOI) – measures the difference in atmospheric pressure between Tahiti (east Pacific) and Darwin (west Pacific). Heavy rainfall is associated with a negative SOI

b) sea surface temperature (SST) – of the Indian and Pacific oceans. Conditions are neutral if the SST remains between 0.5°C above or below the norm for a given area of ocean. In the El Niño year of 1997-1998 the Pacific SST was 5°C above the norm and severe flooding occurred in the Horn of Africa

c) normalised differentiated vegetation index (NDVI) – measures the greenness and browness of the vegetation. The green leaf biomass or photosynthetic capacity of a region is an indicator of the amount of moisture in the soil (23).

The correlation between increased vector activity and heavy rainfall predictions from weather satellite data has been investigated and used successfully in a number of important human and animal disease studies (6). The analysis of data sets may be as simple as assessing information about El Niño to predict a wet season or as complex as comparing land-based weather station minimum and maximum temperatures and monthly rainfall with satellite derived NDVI, middle InfraRed reflectance (MIR), land surface temperature (LST) and air temperature (TAIR).

This data is a useful tool for predicting weather conditions that favour an RVF outbreak, but even if a season is particularly wet, an outbreak will not occur unless there is also active virus circulation and a large susceptible vertebrate population.

Rift Valley fever remains difficult to predict but devastating in its effect on both humans and animals.

Control measures include the following:

a) movement control with respect to trade and export

b) vector control with larvicides in vector breeding sites rather than aerial spraying of adults
c) vaccination of livestock – this is important for two reasons, (i) livestock amplify the virus and mosquitoes preferentially feed on livestock (ii) the young/newborn are most at risk of dying, but colostral immunity confers protection against virulent RVF virus challenge.

The vaccine currently in use is a live, attenuated product incorporating the neurotropic attenuated mouse brain Smithburn strain, which was derived from the Entebbe strain in 1944, developed in 1949, and has been used in South Africa since 1952. Attenuation was achieved by passage in suckling mice and embryonated eggs (39). Serial intra-cerebral passage enhances neurotropism and reduces hepatotropism. The vaccine is not used in pregnant animals except in the face of an outbreak, because a percentage of animals may abort (44). This vaccine is still neurotropic and affects the central nervous system of the foetus and induces foetal teratology and hydramnios (hydrops amnii) in the dam. Use of this attenuated live vaccine in non-endemic areas may be considered during an outbreak, because the importance of preventing the negative effects of the virus in humans and livestock may well outweigh the risk that the virus may re-assort or regain virulence (35). Vaccines under development include two live strains not yet available for field use. One is derived from a mutagenised human field isolate from Egypt, MP 12; it is an efficient immunogen, but is still abortogenic. A mutagen attenuated RVF vaccine of MP 12 has however been found immunogenic and non-abortogenic in pregnant ewes and immunogenic and non-pathogenic in neonatal lambs in the face of virulent challenge exposure. This vaccine produces low-level transient viraemia in cows. The other vaccine, clone 13, is also derived from a field strain of a naturally mild human case. It is a stable vaccine candidate with not much likelihood of reversion, as this clone has a large deletion of 549 nucleotides in the non-structural protein gene (7, 20, 26, 27, 28).

A vaccine for use in pregnant animals and in non-endemic areas is produced from a pathogenic field isolate. This virus is cell culture adapted and formalin inactivated. There are disadvantages, which include the use of pathogenic strains, the necessity of two vaccinations and the costs involved (4, 5, 19). A human vaccine is produced in the United States for use in people at risk.

**Conclusion**

Rift Valley fever is a zoonosis and a haemorrhagic fever which can generate a sense of panic in the public and there has been speculation as to where it might appear if the virus were to ‘escape’ from Africa.

The virus could exit the country via either wind-borne spread of infected mosquitoes, as was probably the case in 1977 when the virus spread from Sudan to Egypt, or vehicular transport of viraemic persons or animals, as happened in 2000 when the virus spread from the Horn of Africa to Saudi Arabia. In both these situations, the virus was deposited in an already flooded area with an extremely large local mosquito population and naive hosts. This is the key to the generation of an outbreak – the obligatory presence of flood conditions and large numbers of mosquitoes.

The particular risk factors associated with the movement of infected animals are the high, intense viraemia and the wide range of vectors capable of transmitting the virus, including mechanical vectors such as ticks and various biting flies such as *Glossina*, *Simulium*, *Stomoxys* and *Culicoides* spp. The viraemic period is about seven days, but the virus may persist in the spleen of sheep for much longer. Although the incubation period of RVF is short, travel to a country with a dam, large irrigation project or large delta system is a concern. Potential risk areas include locations such as where the Tigris and Euphrates rivers empty into the Persian Gulf and the area where the Indus River delta flows into the Arabian Sea. Should the virus spread to an arid or semi-arid one, a cryptic cycle might be the result, where an odd abortion or human case could pass unnoticed.

Given the right conditions, this transboundary disease has the potential to spread over large distances.

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La fièvre de la Vallée du Rift

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Résumé
La fièvre de la Vallée du Rift est une maladie virale transmise par les arthropodes qui touche les ruminants, les camélidés et les humains. C'est aussi une zoonose importante qui peut se manifester comme une maladie pseudo-grippale sans complication, mais peut aussi prendre la forme d'une maladie hémorragique avec atteinte hépatique ; elle peut également provoquer des lésions oculaires ou neurologiques. Chez les animaux, la fièvre de la Vallée du Rift peut ne pas se manifester chez les femelles adultes non gestantes, mais les foyers se caractérisent par l'apparition d'avortements et d'une forte mortalité néonatale. Chez les animaux plus âgés, on constate des icteries, des hépatites et des décès. Les foyers de fièvre de la Vallée du Rift sont associés à de fortes pluies persistantes accompagnées d'inondations durables et à l'apparition de grandes quantités de moustiques, qui sont le principal vecteur. Il est rare que des fortes pluies localisées suffisent à créer les conditions d'un foyer ; il faut aussi l'émergence simultanée de nombreux moustiques de première génération infectés par voie transovarienne. Après l'amplification du virus chez les vertébrés, les moustiques agissent comme vecteur secondaire pour prolonger l'épidémie.

Mots-clés

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Resumen
La fiebre del Valle del Rift es una enfermedad vírica transmitida por artrópodos que afecta a rumiantes, camélidos y seres humanos. También es una importante zoonosis, que a veces se presenta como afección de tipo gripal sin complicaciones, pero otras veces da lugar a una enfermedad hemorrágica con afectación hepática. Puede ocasionar asimismo lesiones oculares o neurológicas. En los animales, la fiebre del Valle del Rift puede ser asintomática en adultos no gestantes, pero los brotes se caracterizan por la aparición de abortos y una elevada tasa de mortalidad neonatal. En ejemplares de más edad se observan ictericia, hepatitis y muerte del animal. Los brotes de esta enfermedad vienen asociados a lluvias fuertes y persistentes, con inundaciones continuas y la aparición de gran número de mosquitos, que son su principal vector. La presencia de fuertes lluvias localizadas no crea por sí sola las condiciones necesarias para un brote, pues se requiere también la aparición simultánea y en gran número de una primera generación de mosquitos infectados por vía transovárica. Tras la ampliación del virus en vertebrados, el insecto actúa como vector secundario y perpetúa así la epidemia.

Palabras clave
References


