



Exotic Animal Diseases Bulletin

Rift Valley Fever

The warning

NASA's Goddard Space Flight Center sounded the alarm back in September 2006, warning that rising sea temperatures accompanied by heavy rains in the Pacific and Indian Oceans could spark an outbreak of Rift Valley fever (RVF) in the Horn of Africa. The climatic conditions were similar to those in 1997–98, when a major outbreak of RVF occurred in Tanzania, Somalia and Kenya.

This warning was carried in the United Nations' Food and Agricultural Organisation's (FAO) November 2006 edition of EMPRES WATCH.¹ FAO encouraged those countries at risk to activate their early warning, prevention and contingency plans for RVF.

The disease

RVF is an acute arthropod borne viral disease of ruminants, camels and humans which was first identified in Kenya's Great Rift Valley in 1930.

In Africa indigenous ruminants including sheep, goats and *Bos indicus* cattle are relatively resistant to RVF infection and show few clinical signs. Introduced livestock species, their crosses and humans act as the indicator species, as they develop clinical signs following infection.

In cattle, sheep and goats the disease is most severe in young animals, in which high mortalities can occur. In peracute cases animals are found dead, or collapse when driven.

In acute cases, the incubation period is less than 24 hours followed by fever, weakness, unsteady gait, mucopurulent nasal discharge and vomiting. Death occurs in less than 72 hours. Haemorrhagic diarrhoea and petechial or ecchymotic haemorrhages in mucous membranes may be observed.

Subacute disease is more common in adult animals. Fever is followed by weakness and anorexia. Jaundice and abdominal pain may be observed, and abortion of pregnant animals may result. Infection may be inapparent in adult animals.

On examination the predominant signs are petechial or ecchymotic haemorrhages in most internal organs. The liver is swollen, congested and friable. There may be ascites, hydropericardium, hydrothorax and pulmonary oedema present. The fluid is frequently blood stained and the carcass may be jaundiced.²

RVF is a significant zoonosis. In humans it causes a severe influenza-like disease (fever, headache, bleeding, malaise, muscle pain, back pain, vomiting, extreme weight loss) with many patients recovering about a week after the onset of symptoms. Severe disease may develop in 8% of patients resulting in the death of 0.5 to 1.0% of patients. The severe form of the disease can present as a generalised haemorrhagic syndrome, encephalitis or retinitis.³ Human deaths may result from post-recovery meningoencephalitis.

People can become infected by handling material from infected animals, such as butchering infected stock or through veterinary and obstetrics procedures, or more commonly from the bites of infected arthropod vectors.

Vectors

At least 30 species of mosquitos have been found to be naturally infected with RVF virus. *Aedes* sp are believed to be the major vectors, however *Culex* sp and *Mansonia* sp also are important vectors. The virus has also been recovered from ticks and *Culicoides* sp. Transovarial transmission has been demonstrated in mosquitoes and is

believed to maintain the virus in the environment during inter-epizootic periods.

Recent epizootics in Kenya have shown the importance of heavy rainfall and crop irrigation practices in providing ideal conditions for vector multiplication.

World distribution

Until the late 1970s RVF was confined to eastern Africa. In 1977–80 an outbreak occurred in Egypt, resulting in 200,000 human cases and 598 deaths. In 1987 an outbreak occurred in southern Mauritania and northern Senegal. Further outbreaks occurred in Egypt in 1993 and 1997. An outbreak in 1996–97 across the Horn of Africa resulted in an estimated 89,000 human cases and 478 deaths. In 2000 RVF spread to the Arabian Peninsula in Saudi Arabia (800 human cases) and Yemen (1000 human cases).^{3,4}

The 2006–07 outbreak

The traditional nomadic tribespeople in Somalia and Kenya herd cattle, fat tailed sheep and goats and are scattered over large areas, making communications and vaccination programs difficult to implement. The tribespeople are exposed to insects and may kill and eat a sick animal. Initial indications of the outbreak were sketchy due to extensive flooding in Kenya (many areas of north eastern Kenya were unreachable by road) and civil unrest in Somalia. Aborting and dying livestock and human deaths were reported in north-eastern Kenya.

On 9 January 2007 the Kenyan authorities advised the World Organisation for Animal Health (OIE) that the outbreak had commenced on 4 December 2006 (Figure 1). 1500 cases

were reported in sheep with 105 deaths, 1500 cases in goats with 95 deaths, 500 cases in cattle with 30 deaths and 500 cases in camelids with 5 deaths.

As of 12 March 2007 the World Health Organisation (WHO) reported 684 suspected human cases, including 155 deaths, of which 234 cases had been laboratory confirmed. 114 suspected human cases, including 51 deaths, were reported in Somalia.⁵

Most of the early reported cases in Kenya were in herdspeople. One of the deaths was of a veterinarian who died after undertaking a post mortem examination on a goat.

In Tanzania cases of RVF in animals were first reported on 18 January 2007 followed by human cases in early February.

Prof Glyn Davies⁵ has indicated that in endemic areas RVF virus is transovarially transmitted in *Aedes* mosquitoes breeding in flood waters. Amplification occurs with each successive cycle of reproduction. The flood waters allow the generation of huge mosquito populations that feed on indigenous ruminants, which are unlikely to manifest clinical signs. The epidemic's duration is dependent on the persistence of flood waters, which can last for up to 4 months.

Vaccination

A modified live virus vaccine is available in Africa. It is relatively cheap and easy to produce, but may cause abortions and foetal abnormalities if used in pregnant susceptible sheep. An inactivated vaccine is available for use in pregnant animals. No commercial human vaccine is available.

The use of vaccines is contra-indicated at any time when virus transmission is occurring. This is because the virus spreads via contaminated needles from animal to animal, extending the outbreak⁶. Hence FAO developed the predictive model to provide an early warning system to allow vaccination to be carried out in the pre-epizootic phase. Unfortunately this season widespread vaccination was not carried out.

Other control measures implemented in affected countries include the strict enforcement of a ban on the livestock slaughter, restricted livestock movement,



Figure 1. Map of current outbreak (OIE 2007)

and an integrated vector control program, including the use of larvicides. Public health measures included the use of indoor residual insecticide sprays, repellents and treated mosquito nets.

Relevance to Australia

In principle, RVF could be introduced into Australia through the importation of infected vectors or hosts, including humans.

Australia's quarantine procedures for inbound overseas vessels minimise the risk of introduction of infected vectors. The probability that RVF would be introduced into Australia in this way would appear to be low.

It may be possible for the disease to be introduced by an infected person before clinical disease became apparent. Some potential vector species for RVF virus exist in Australia but no competency studies for the Australian strains have been undertaken. The risk of introduction of the disease by an infected person appears to be low: RVF has never been diagnosed in Australia.

Whilst it is unlikely that RVF will spread to Australia, the use of climate models to predict the likely occurrence of

disease does have application for vector borne diseases, such as bluetongue.

For further information see the AUSVETPLAN Rift Valley Fever Disease Strategy at the www.animalhealthaustralia.com.au.

References

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