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Rift Valley Cholera: Science and the Study of Disease Transmission

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Abstract

Rift Valley Fever (RVF) is a viral zoonosis spread by mosquitoes. It was first discovered in Kenya in 1930, and since then, it has spread to many African nations and the Arabian Peninsula. Human infection can result in a wide range of clinical outcomes, from self limiting febrile illness to life threatening hemorrhagic diatheses and miscarriage in pregnant women. The RVF virus primarily infects domestic livestock (sheep, goats, cattle) resulting in high rates of neonatal mortality and abortion. RVF has been responsible for numerous outbreaks in Africa and the Arabian Peninsula since its discovery, with significant effects on human and animal health. However, the lack of licensed human vaccines or therapeutics limits options for controlling RVF outbreaks. The World Health Organization places RVF at the top of its priority list for urgent research and development of measures to prevent and control future outbreaks. The current understanding of RVF, including its epidemiology, pathogenesis, clinical manifestations, and vaccine development status, are highlighted in this review.

Keywords: Zoonosis; Hemorrhagic; Miscarriage; Pathogenesis; Epidemology

Introduction

In 1930, Rift Valley Fever Virus (RVFV) was discovered to be the cause of an outbreak of "enzootic hepatitis" near Lake Naivasha in Kenya's Rift Valley. Blood from a diseased lamb was injected into an unaffected lamb, which reproduced the disease, through a Chamberland porcelain filter to determine whether it was caused by a bacterium or a virus. The outbreak occurred during a time of high mosquito activity, leading the researchers to believe that mosquitoes play a role in disease transmission. Healthy sheep were either relocated to a higher altitude where mosquitoes were absent or placed under mosquito netting in an effort to contain the outbreak. Both measures worked, and the lack of apparent direct animal-to-animal transmission supported the hypothesis that mosquitoes are involved in disease transmission. RVFV was later isolated from several naturally infected species of Aa13(en)19 sd-of animal infection studies that were carried out shortly after the outbreak and in the years that followed. These studies documented the susceptibility of a diverse range of animal species. We highlight recent advancements in our understanding of RVF, including its epidemiology, pathogenesis, clinical characteristics, and vaccine development status, in this review [2-5].

Epidemiology

The ability to transmit the virus to its offspring has been demonstrated by a single species of Aedes that was incorrectly identified as Aedes lineatopennis prior to 1985 and later identified as Aedes (Neomelaniconion) mcintoshi . During the dry season, RVFV may be able to remain viable in the eggs of this species before hatching when the rains return. Further research is needed to determine whether this is the only species capable of vertical transmission and to what extent it permits RVFV to circulate during IEPs. Seropositivity in sheep and goats that have not experienced an RVF outbreak suggests that the virus can circulate at a low level in livestock. Wild ungulates can also be infected with RVFV by mosquitoes.

In point of fact, a wide variety of species, including African buffalo, giraffe, black rhino, impala, and African elephants, among others, have been found to have neutralizing antibodies that target RVFV . While some of these species, like buffalo and giraffe, appear to be immune to RVF, others, like elephants, appear to be.

RVF epizootics, in which a large number of livestock become infected, can occur when there are periods of exceptionven.aalo aeat-9(e) en1.5 populations. Rainfall data and changes in vegetation have been used to predict RVF outbreaks because of the correlation between the weather and RVFV infection. However, models that predict RVF outbreaks frequently rely on inaccurate data on the variables. Because of this, these models' predictive value varies [5]. In Kenya, synchronous monitoring of livestock herds during times when conditions appear favorable for RVF outbreaks has also been utilized as an early warning system. Farmers may be more aware of RVF and more likely to get vaccinated as a result of these systems.

RVFV can be transmitted by a wide variety of mosquito species. The virus can infect other arthropods like midges, ticks, and sandflies, which could potentially serve as mechanical vectors. More than 53 species of mosquitoes caught in the field tested positive for RVFV, according to a study. Additionally, more than 65 species have been identified as potential vectors, most of which are *Aedes spp*. and *Culex spp.*. Depending on the species, some of these potential RVFV vectors may be able to successfully transmit the virus [6].

The majority of human infections, in contrast to those that affect

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animals, are caused by contact with infected tissues or uids rather than by a mosquito bite. Various instances of human transmission during the 1930-1950s happened via unintentional lab contamination. In fact, these case reports were the source of much of our early understanding of the disease in humans.

Direct human-to-human transmission has not been recorded. ere is no evidence of nosocomial transmission even during epidemics in hospitals with inadequate personal protective equipment. In one instance, viral RNA was isolated from an immunosuppressed patient's urine and sperm four months a er the onset of symptoms. However, it is unknown whether RVFV can be transmitted sexually. Ex vivo experiments have shown that RVFV can directly infect human placental tissue, and vertical transmission from mother to child has been documented in human cases [7,9].

Numerous studies on seroprevalence in human populations have provided insight into the populations most at risk for infection. Having contact with susceptible animals and participating in the slaughter process is the most signi cant risk factor. Adults are more likely than children to be seropositive, either because they are older and have had more time to come into contact with RVFV or because of the increased occupational risk.

However, it is unlikely that all of the larger outbreaks, like the one in Egypt in 1977, were caused by direct animal contact. *Culex pipiens* was mentioned for the rst time in this instance. ere are documented cases of human infections that were attributed to mosquito bites, despite having no direct connection to livestock [8-11].

It is currently unclear why some individuals develop more severe disease with long-term issues while others remain asymptomatic or

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