Rift Valley Fever - A Fatal Viral Disease of Neonatal Animals

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Abstract

Rift Valley fever (RVF), a mosquito-borne disease of animals and humans, is caused by Phlebovirus which belong to the family Bunyaviridae. The disease in animals is characterized by fever, anorexia, diarrhea, hepatitis and abortion. It causes high mortality in neonatal animals and high incidence of abortion in sheep and cattle. The disease is of great economic significance as losses mainly occur due to the death of young lambs and calves. Laboratory help is required to confirm the diagnosis of RVF. The immunization and vector control are the main strategies to reduce the incidence of RVF. It is considered as an occupational disease of livestock handlers, dairy farmers, abattoir workers and veterinarians. Hence, it is imperative to take all necessary precautions to protect from infection.

Key words: Abortion, Mosquito, Neonate, Rift Valley Fever, Viral disease

Introduction

Rift Valley fever (RVF), also known as enzootic hepatitis, is an important vector borne viral disease of animals as well as humans (WHO, 1982; Pal, 2007). The disease was first described in sheep of the area of Rift Valley in Kenya (Africa) in 1931. Epidemics of RVF have been reported from several counties of Africa and the Arabian Peninsula- Saudi Arabia and Yemen (Gerdes, 2004; Woods et al., 2004; WHO, 2007). Mosquitoes of several genera play significant role in disease transmission (OIE, 2004). It is characterized by short incubation period, fever, hepatitis and death in young animals (Radostits et al., 1994). The lambs, calves, kids, kittens and puppies are very susceptible to RFV. The disease is most severe and fatal in neonatal animals. The aim of the present communication is to delineate the etiology, host range and susceptibility, transmission, clinical spectrum, gross lesions and histopathology, epidemiology, diagnosis, immunization and control of RVF in animals particularly in neonates. In addition, the public health significance of RVF is also mentioned.
Etiology

Rift Valley fever is caused by a single serotype of *Bunyavirus* of the genus *Phlebovirus* of the family *Bunyaviridae* (OIE, 2004). The single stranded RNA virus has a lipid envelope and two surface glycoproteins G1 and G2. The genome has three segments L (large), M (medium) and S (small). RVF virus replicates in the mosquitoes and in the vertebrate animals. The liver, spleen and brain are the major sites of viral replication. The virus can be inactivated by disinfectants such as calcium hypochlorite, sodium hypochlorite and acetic acid; and be maintained for 8 years when stored below 0°C.

Host Range and Susceptibility

Natural infection due to RVF virus has been recorded in antelope, buffalo, camel, cattle, goat, monkey, rodent and sheep besides man (Radostits et al., 1994; Pal, 2007). Significant mortality and morbidity due to RVF has been reported in sheep, cattle and man (Radostits et al., 1994; Gerdes, 2004; Kahn and Limes, 2005). Several species of domestic, pet, farm and, laboratory animal are susceptible RVF virus (Gerdes, 2004). The kids, lambs, puppies, kittens, hamsters and mice are highly susceptible to Rift Valley fever virus. Amphibians and reptiles are resistant to RVF virus. Hitherto, disease is not recorded in birds (Gerdes, 2004).

Transmission

Animals and man get infection following the bites of many species of mosquitoes. The virus is transmitted transovarially among Aedes species mosquito, and survives for very long-periods in the mosquito eggs. Cattle and sheep are primary amplifiers of the virus. Humans may also acquire infection through inhalation of aerosol from infected animals, or by direct contact of the mucous membrane or abraded skin, wound with the discharges of diseased animals (WHO, 1982; Pal, 2007). Low concentration of RVF virus in the milk of sick animal may pose health risks to man if the milk is consumed raw or unpasteurized (WHO, 2007). It is interesting to mention that humans have the potential to introduce RVF virus via mosquitoes to animals in uninfected areas (Kahn and Lime, 2005).

Clinical Spectrum

The onset of disease is sudden and dramatic with initial signs depending upon the breed and genotype and age of the target animals. The sudden onset of abortions among sheep, cattle, goat or camels and high mortality in neonates (young lambs, calves and kids) in an area is probably the most significant signs of the disease. The incubation period in lambs is 12-96 hours. The most severe reactions occur in newborn animals particularly the kids and lambs which succumb to death within hours of infection. In young lambs, kids, calves and puppies, the onset of disease is rapid with fever of 40-42°C, anorexia, listlessness,
rapid respiration, abdominal pain, diarrhea, weakness and death. Fatality in less than one week old lambs exceed over 90 per cent in 36 hours. Calves less than 10 days old suffer a peracute form of RVF and die within 24 hours. However, in lambs and calves of more than one week age, mortality may be less than 20 per cent (Radostits et al., 1994). The abortion may occur as a result of infection of foetus or due to febrile reaction. The foetus is often autolysed. The icterus is more commonly seen in older calves. The animals at one to four months of age may suffer an acute febrile illness with prostration and 10-40 per cent fatalities (Glyn Davies and Martin, 2003).

Gross Lesions and Histopathology

Hepatic necrosis is the primary lesion observed in RVF. In aborted fetuses and neonatal animals, particularly the lambs and calves, the liver is soft, enlarged, friable and yellowish brown to dark in color. In addition, the oedema and hemorrhages in the wall of gall bladder, hemorrhagic enteritis, enlarged oedematous peripheral and visceral lymph nodes, widespread cutaneous hemorrhages, accumulation of blood stained fluids in the body cavities and extensive subcutaneous and serosal hemorrhages are also observed. The rapid decaying of the carcass may be a consequence of the severe liver damage. Histopathologically, the liver lesions in the diseased lambs are pathognomonic. In lambs, there is a focal to diffuse coagulative necrosis of hepatocytes in the affected liver. Intramuscular inclusion bodies are also noticed (Glyn Davies and Martin, 2003).

Epidemiology

Rift Valley fever is a peracute or acute viral zoonotic disease of domestic animals. The incidence of RVF peaks in late summer. The virus is spread epidemically by many species of mosquitoes. Since its first outbreak among the sheep in 1931, the disease has been reported in several other species of animals and man. Countries with endemic disease and substantial outbreaks of RVF include Egypt, Gambia, Kenya, Madagascar, Mauritania, Mozambique, Namibia, Saudi Arabia, Senegal, South Africa, Sudan, Yemen, Zambia and Zimbabwe (OIE, 2004). The cyclic epidemics have occurred at 5 to 20 years intervals in drier areas. In the periods between epidemics, the virus is believed to be dormant in eggs of the mosquitoes. Many countries like Angola, Botswana, Burkina Faso, Cameroon, Chad, Congo, Ethiopia, Giabon, Guinea, Malawi, Mali, Niger, Nigeria, Somalia, Tanzania and Uganda are known to have some cases, periodic isolation of virus, or serologic evidence of RVF. In India, presence of antibodies to RVF or a closely related virus in goats and sheep was first demonstrated in Rajasthan in 1995. Subsequently, an outbreak of disease among sheep was recorded in Tamil Nadu. The virus was not isolated from sheep during the outbreak. However, detection of anti-RVF IgG antibodies in the sera of sheep was suggestive of RVF-like illness (Joshi, 2004).
The disease has a great economic and public health importance. A massive epidemic of RVF which occurred in Kenya, Somalia and Tanzania in 1997-1998 caused deaths of many thousands of camels, goats and sheep and over 90,000 human cases with more than 500 deaths (Woods et al., 2002). It is an occupational viral zoonosis of cattle owners, shepherds, farmers, animal handlers, abattoir workers and veterinarians. The economic impact of RVF can be catastrophic for meat and dairy producers due to serious illness and high mortality among the affected livestock herds. The epidemic of disease occurs over a large area of a country following heavy rains and flooding. The disease carries a high mortality in neonatal calves, lambs and kids.

**Diagnosis**

The field veterinarian can suspect RVF if he encounters high abortion rates possibly approaching 100 per cent among the cows and ewes; very high mortality approximately 100 per cent in calves and lambs of less than 7 days of age, extensive liver lesions in aborted fetuses and neonatal animals, an influenza like disease in man particularly in individuals associated with livestock and occurrence of disease during a period of high insect activity (Glyn Davies and Martin, 2003; OIE, 2004).

The clinical specimens such as the liver, spleen, brain, lymph nodes, kidney, heart, and blood should be collected aseptically in sterile containers for virus isolation. In case of an autolysed foetus, the brain is a good specimen to be submitted on ice to the laboratory for diagnosis. Several types of cell cultures such as baby hamster kidney (BHK), African green monkey kidney (Vero), chicken embryo reticulum (CER), or primary kidney and testis cell cultures of lambs and laboratory animals like mice and hamsters can be employed for the isolation of virus (OIE, 2004; Kahn and Line, 2005; Pal, 2007).

Two sera samples at an interval of 30 days should be obtained to demonstrate antibodies against RVF by ELISA, AGID, HI, CF and virus neutralization methods (OIE, 2004; Pal, 2007). The virus antigen can be detected by reverse transcriptase polymerase chain reaction (RT-PCR) (Sall et al., 2002). It is a very specific and sensitive molecular tool for the diagnosis of RVF in the early phase of disease. Histopathological examination of the liver of the affected animal reveals extensive hepatic necrosis which is the characteristic lesion in RVF (Radostits et al., 1994).

Differential diagnoses in animals include brucellosis, enterotoxaemia, blue tongue, vibriosis, heart water disease, Nairobi sheep disease, ephemeral fever, ovine enzootic abortion and trichomoniasis (Radostits et al., 1994; Gerdes, 20004; Kahn and Lines, 2005).
Immunization

The livestock can be protected by immunization. Mutagenized vaccine does not cause any adverse effect in neonatal lambs and calves (Morill et al., 1997). One inoculation induces rapid immunity. A serum neutralization titer of 20 or greater is protective. Lambs and calves that receive colostrum from a convalescent dam or dam vaccinated with an attenuated virus are passively protected for about 3 months. It is advisable to vaccinate the lambs routinely at six months of age for protection. The pregnant cows and ewes should preferably be vaccinated with a formalin-inactivated vaccine. Revaccination after three months is advisable to induce an immunity that will last for one year and to confer cholesterol immunity to offspring (Kahn and Line, 2005). Live attenuated vaccine is not recommended for pregnant sheep as it may cause abortion, fetal death and some anomalies (Radostits et al., 1994).

Control

RVF can be controlled by vaccination of all susceptible animals to prevent infection of amplifying hosts and thus infection of vectors. Use of insecticides to kill mosquitoes and ban on export of livestock during RVF epizootic periods is also recommended. As RVF is a zoonotic disease, all precautions should be taken to protect the health of the persons engaged in livestock industry (Pal, 2007; WHO, 2007).

Public Health Significance

Rift Valley fever is a significant viral zoonosis which causes high morbidity and mortality in persons particularly dealing with diseased animals. The slaughter of infected animals, autopsy procedures and laboratory manipulation of tissues and isolation of virus are high professional risks of acquiring RVF. The epidemic of RVF recorded in several countries involved many humans. The risk of human-to-human infection through direct contact appears to be very low (WHO, 2007).

Conclusions

Rift valley fever, a metazoonosis of viral etiology, is endemic in many countries of the world. The disease is reported in a wide variety of animals and man. It carries a high mortality in neonatal animals chiefly in lambs. Transmission of RVF is primarily by the bites of the mosquitoes of several species. Man acquires the infection from the infected animals. In neonatal animals, pathognomic lesions are noticed in the liver. Diagnosis is confirmed by isolation of virus, demonstration of antibodies in the sera and histopathology of the liver. Immunization of animals, destruction of mosquitoes and restriction on the movement of animals during epizootic can help in the control of RVF. Considering the public health implications of RVF, the persons dealing with the sick animals must take strict measures to prevent the transmission of
disease. It is emphasized to conduct disease surveillance in vulnerable areas; and also to assess economic and public health consequences of RVF.

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References


