Exotic Diseases: Vesicular Stomatitis and Rinderpest

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Vesicular Stomatitis

1. Definition and aetiology

Vesicular stomatitis (VS) is a rhabdoviral disease of the genus *Vesiculovirus* that causes sporadic (cyclic) outbreaks of disease in cattle, horses, donkeys, mules, and pigs. Cattle under 1 year of age rarely show clinical signs. VS is seen in the United States, Mexico, and Central and South America. Human beings have sometimes been infected with an influenza-like disease. Two distinct antigenic strains have been designated, the New Jersey (NJ) and Indiana (I) serotypes. Vesicles occur in the mouth, on the teats, and in interdigital areas. Lesions may occur only on the teats or only in the mouth. The vesicles rapidly turn to painful ulcerations that cause disphagia and reluctance to eat, frothing at the mouth, drooling, agalactia, weight loss, mastitis, and lameness. Death is rare. Epizootic waves of VS have tended to occur at about 10-year intervals, usually in the summer or fall, but since the major epizootic in 1982 and 1983 in the western United States, the NJ serotype has been identified in the United States each year. An outbreak occurred in 1995 in western United States, in New Mexico and Colorado. More cases occurred in 1997. Although the NJ serotype has at least 14 distinct genotypes, only a few of these have been found in recent outbreaks in the United States. The NJ serotype should be considered a collection of serologically related but genetically variant viruses. The I serotype has three subtypes; I-1 occurs in the United States. State and federal regulatory veterinarians should be contacted immediately when VS is suspected so that quarantine and disease identification measures can be used quickly to contain an outbreak.

2. Clinical signs and differential diagnosis

After a mean incubation period of 9 days (the range is 3 to 14 days), there is onset of fever and oral lesions that cause excess salivation and reluctance to eat. From 5% to 60% of cattle on a farm may show clinical signs. Vesicles are only occasionally visible, because the epithelium rapidly necroses and many lesions quickly turn to ulcers. Lesions on the gums and tongue may coalesce to form large eroded areas (Fig. 30-51). Milk yield falls quickly. Teat lesions are common in dairy cattle, and small ulcers in the interdigital area and on the coronary band are occasionally seen. Recovery varies from 2 to 21 days, depending on the severity of the lesions and management factors such as the type of feed and milking sanitation. Actual healing of the lesions may take 34 to 59 days.

The major differential diagnostic consideration is foot-and-mouth disease, which causes almost identical clinical signs. Other diseases of cattle that result in oral lesions (bovine virus diarrhoea, bovine papular stomatitis, bluetongue) do not usually appear as epidemics but rather in one or a few animals (although bovine papular stomatitis may have a high morbidity). Other causes of oral lesions such as bristle grass irritation and toxins should be ruled out.

3. Clinical Pathology and laboratory diagnosis

The VS virus is difficult to isolate from blood, urine, faeces, and oral swabs but has been isolated from tongue epithelium. A complement fixation test and a fluorescent antibody test are available for virus identification. Serum neutralizing (SN) titre rises rapidly after exposure and then gradually falls over the first year. SN titres that follow natural exposure may persist for years. Vaccination with inactivated virus

results in a rapid rise in titre followed by a gradual decline for a year. The presence of an SN antibody titre does not prevent reinfection or development of clinical signs. ELISA's are also available. Haematologic and clinical chemistry findings generally reflect an acute to chronic inflammatory disease and are nonspecific in helping to make a diagnosis.

4. Pathophysiology

After a short incubation period of 24 hours, fever and viral invasion of oral epithelial cells occur. Oral abrasions or trauma may increase susceptibility. Contact of virus with teats or feet can result in lesions in these areas, especially if the teats are chapped or cracked or the feet are traumatized. The lesions progress rapidly from blanched macules to vesicles and soon rupture, leaving sloughed epithelium and ulcerated areas. Healing occurs quite rapidly if feed is soft and nontraumatic.

5. Epidemiology

Older, higher-producing dairy cows that have been in milk longer are more susceptible to clinical disease caused by VS than herd mates. Because the virus cannot penetrate intact mucosa, cattle fed coarse feeds or hard pellets that traumatize the oral mucosa are at higher risk. Cows with chapped or cracked teats and those on farms with poor milking hygiene are more likely to get teat lesions. Cow to cow contact is a major mode of transmission in outbreaks, and increased interpen movement of cattle, as well as shared feed and water troughs, unless cleaned frequently, increases the risk of development of VS. The virus is transmitted by milking machines and human hands during outbreaks. Insect vector also contribute to the mechanical spread.

Outbreaks are often associated with the movement of animals from another area, but disease epidemics not associated with new animals do occur. The infection tends to be seasonal (occurring in the summer and autumn in temperate areas and at the end of the rainy season in the tropics) and behaves like an arthropod-borne virus. The reservoir and vectors appear to be the sand fly and the black fly. Antibodies have been found in a number of wild species of animals, (deer, raccoon, bobcat, monkey), which may provide a reservoir. Active cases of VS occur in Mexico between US epidemics, giving rise to the possibility that cattle from Mexico arriving in the United States may also act as sources of VS virus. Sheep and goats in contact often seroconvert, although clinical signs in these species are rare. The virus survives several weeks in cool soils and is very resistant to pH changes. Cattle generally show a high morbidity rate and a low mortality rate (1% to 5%). Although many in-contact cattle do not show obvious clinical signs of disease, many more have oral lesions if closely examined, and most animals in the herd seroconvert.

The economic losses associated with an outbreak of VS can be severe, especially in dairy cattle. In the 1982 epizootic in California, losses on two dairies (principally decreased milk production and culling for mastitis) totaled US$225,000 during 2 months.

6. Necropsy findings

Deaths are rare and usually are attributed to secondary bacterial diseases, including environmental mastitis and pneumonia. Cattle become gaunt and weak as a result of dysphagia and resultant reduced food intake. Erosive and ulcerative lesions are usually confined to the mouth. The teats frequently are involved in lactating cows, and lesions on the coronary band and interdigital area occasionally may be seen.

Histologically, intracellular and extracellular edema, ballooning and degeneration of epithelial cells, and vesicle formation accompanied by neutrophilic infiltration are present. There are no inclusion bodies. The characteristic bullet-shaped structure of the VS virus sometimes can be seen with electron microscope examination of fresh lesions or vesicular fluid.

7. Treatment and prognosis

Mortality can be almost completely prevented if ill cattle are offered shade, fresh water, clean bedding, and soft feed. Offering soft feed hastens recovery and reduces the anorectic period. Debilitated cattle should be given broad-spectrum antibiotics in an effort to control secondary bacterial pneumonia. Cattle with teat lesions are at high risk of developing mastitis and should be carefully milked last and monitored closely for mastitis. The prognosis for survival is very good, but agalactia and mastitis may result in culling of a large number of animals.

8. Prevention and control

During an outbreak of VS, quarantine of the premises and isolation of sick animals are required. Regulatory officials will help organize and maintain a quarantine. Feed should be soft and fine, because coarse or hard-pelleted feeds increase the spread of the virus and prolong the recovery time. Leftover feed should be removed from feed bunks twice daily and the bunks disinfected. Water troughs should be cleaned and disinfected daily. Disinfection can be accomplished by using 1% formalin, organic iodines, hexachlorophene, or phenyl-phenolic preparations. Two hours of exposure to lye (2% NaOH) will *not* deactivate the virus.

Vaccination using killed or live virus vaccines is rarely practiced preventively because the disease occurs as rare epidemics in small areas and because vaccination interferes with serologic testing and monitoring. Vaccination with a killed virus vaccine may be used by regulatory veterinarians in at-risk animals during an epidemic. Owners and managers should consult state and federal veterinarians before considering VS vaccination.

Rinderpest (Cattle Plague)

1. Definition

Rinderpest is an acute, highly contagious and often fatal disease of ruminants. Cattle and water buffalo are most frequently affected, but the disease also occurs in sheep, goats, pigs, camels and other cloven-hoofed animals, in which it is usually less severe.

2. Aetiology

Rinderpest virus is in the family paramyxoviridae, genus *Morbillivirus*. There is only one known strain.

It is closely related to peste des petits ruminants (PPR), which is essentially small ruminant rinderpest. Other morbilliviruses are canine distemper and the 1995 Australian morbillivirus that occurred in horses and humans.

3. Distribution

The virus is currently most active in Africa because it is maintained in wild ruminants, and frequently jumps to cattle. It is also found in the Middle East and Asia.

4. Clinical signs and differential diagnosis

The incubation period in cattle is 3 to 15 days, which is followed by sudden onset of fever, depression, and anorexia. The nose is dry, and mucous membranes are congested. Within days oral erosions appear where necrotic foci have sloughed. Purulent lacrimation occurs. Diarrhoea is severe and may be bloody. Dehydration and emaciation often lead to death. The disease in sheep and goats usually is mild or subclinical, and mortality in small ruminants.

In endemic areas RP is suspected when the signs described above occur in groups of animals. In nonendemic areas diseases that appear similar clinically are bovine virus diarrhoea, malignant catarrhal fever, asenic poisoning, severe coccidiosis, and severe fulminating infectious bovine rhinotracheitis.

Other causes of severe gastroenteritis and diarrhoea (e.g., salmonellosis) must also be considered. Oral lesions of RP are similar to these seen with vesicular stomatitis. In small ruminants, bluetongue, PPR, and Nairobi sheep disease must be ruled out.

5. Laboratory Diagnosis

Laboratory confirmation of RP can be accomplished by (1) virus isolation; (2) detection of viral antigen by fluorescent antibody testing, virus neutralization, complement fixation, or agar gel immunodiffusion; (3) detection of rising antibody titre by ELISA or virus neutralization; and (4) histopathologic evaluation. Virus isolation is most successful in the first days of infection (often before the onset of diarrhoea). Blood should be taken in heparin. The lymph nodes and spleen are reliable sources of virus, and in cattle, tears and ocular discharges are also reliable sources. Lymph node biopsy after Day 3 of infection is the most reliable means of diagnosis in a living goat. Tissues should be shipped on ice to the laboratory for virus isolation or detection of antigen. Detection of a rising antibody titre can help diagnose the disease retrospectively. Leukopoenia may be noted in the acute early states of RP.

6. Pathophysiology

The virus usually enters through the respiratory mucosa. Lymphoid tissue is the primary target of the rinderpest virus. Lymphocytes are destroyed in the germinal centers of the lymph nodes, Peyer's patches, tonsils, splenic corpuscles, and caecal lymphoid tissue. Immunosuppression occurs as lymphoid tissue is destroyed. The virus also attacks the alimentary tract mucosa; Peyer's patches are the most severely affected. As alimentary mucosa is lost, diarrhoea and emaciation become severe.

7. Epidemiology

RP is highly contagious; it is spread mainly through airborne droplets, direct contact, faeces, and contaminated fomites such as human beings. All secretions and faeces of infected animals are contagious throughout the course of the disease. Wild ruminants frequently are a source of infection for livestock. The morbidity rate often approaches 100% with a 25% to 90% mortality rate, making treatment unrewarding and the prognosis poor. Innate or specific resistance may protect individuals or herds from clinical signs after infection. Recovered animals do not appear to act as carriers. Valuable individuals may be helped by supportive therapy and hydration.

8. Necropsy findings

Lesions are found mainly in the alimentary tract and lymphoid tissues. Subendocardial hemorrhages may also be seen in animals that die of acute illness. Oral erosions, oedema, and congestion of the abomasum are typically seen, and ulcers and hemorrhagic to necrotic Peyer's patches occasionally are seen. The caecal and colonic mucosae are haemorrhagic, ulcerated, or necrotic. The lymph nodes have necrotic germinal centers.

9. Prevention and control

Because there is only one known strain of RPV and protection after infection usually is lifelong, vaccination of cattle in endemic areas with a live cell culture attenuated virus is an effective means of controlling RP. Although there is some debate as to whether this vaccine causes immunosuppression, it has been used safely and effectively in millions of cattle in Africa and Asia. The vaccine does not produce adverse reactions, has a long shelf life, and produces solid immunity. Its major disadvantages are that the lyophilized virus has to be kept cold, and once a vial has been reconstituted, it must be used quickly before the virus dies. A vaccinia-vectored gene vaccine currently under field testing is effective and has the advantages of long-term stability in harsh environments and ease of application (scratch on).

Control of epidemics in areas where the disease is not endemic involves quarantine and slaughter of infected and contact animals, as well as other contact ruminants and swine. Disinfection of premises while under government quarantine is essential in controlling the disease. The virus is susceptible to most disinfectants and survives in the environment only for 2 to 3 days.