Vesicular Stomatitis

Overview
• Organism
• Economic Impact
• Epidemiology
• Transmission
• Clinical Signs
• Diagnosis and Treatment
• Prevention and Control
• Actions to Take

In today’s presentation we will cover information regarding the organism that causes vesicular stomatitis and its epidemiology. We will also talk about the economic impact the disease has had in the past. Additionally, we will talk about how it is transmitted, the species it affects (including humans), clinical and necropsy signs seen, and diagnosis and treatment of the disease. Finally, we will address prevention and control measures for the disease as well as actions to take if vesicular stomatitis is suspected.

The Organism

Vesicular Stomatitis Virus

• RNA Vesiculovirus
  – Family Rhabdoviridae
  – Major serotypes
    • VSV-NJ and VSV-I
  – Affects horses, cattle, swine, camelids, humans
    – Sheep and goats resistant
  – Closely resembles exotic vesicular diseases including FMD

Vesicular stomatitis virus (VSV) is a bullet-shaped RNA virus in the genus Vesiculovirus in the family Rhabdoviridae. There are many different serotypes found around the world, but the New Jersey (VSV-NJ) and Indiana (VSV-I) types predominate in the Americas. This vesicular disease can cause lesions in the mouth and on the feet of a wide range of animals, but it primarily affects horses, donkeys, cattle, swine, and South American camelids. Sheep and goats are resistant to VSV and rarely show clinical signs. Humans can also become infected producing influenza-like symptoms. Vesicular stomatitis closely resembles three vesicular diseases exotic to the U.S.: foot-and-mouth disease (FMD), swine vesicular disease, and vesicular exanthema of swine.

Photo: USDA APHIS.
Vesicular Stomatitis

Importance

History
- Early 1800s
  - Horse illness resembling VSV
- 1927: Virus identified
- 1950s: Human infections recorded
- 1982-83: Outbreak in western U.S.
  - Previously, epizootic waves typical
  - Now an annual occurrence in U.S.

Recent Outbreaks
- 1998-99: NM, CO, TX; 130 positives
- 2004-05: TX, NM, CO; 470 positives
- 2005: Nine states; 786 positives
- 2006: WY; 29 positives
- 2009: TX, NM; 7 positives
- 2010: AZ; 4 positives

Economic Impact
- 1928: California dairy herds
  - $97 to 202 lost per head
- 1995: New Mexico beef herd
  - $53 lost per head
- Losses due to:
  - Increased culling, increased mortality
  - Reduced milk production
  - Labor, medicine, veterinary costs

Although the exact etiology was not known, an illness resembling VSV was first noted in horses with “sore tongue” in the early nineteenth century. The Civil War in 1862 disabled 4000 horses with a disease resembling VS. In 1927 the virus was identified in horses. Human infections were thought to occur during the first half of the 20th century but weren’t recorded until the early 1950’s. Epizootic waves tend to occur approximately every 10 years and there was a major outbreak from 1982 to 1983 in the western U.S. Since then, the VSV-New Jersey serotype has been found each year in the U.S.

In May 1998, a U.S. outbreak started in a horse in New Mexico and spread to other horses in Colorado and Texas through January 1999. In all, 130 premises were positive and VSV was isolated from 27 horses. Another outbreak occurred from May 2004 to January 2005. States involved included Texas, New Mexico, and Colorado; in all there were 294 quarantined premises with 470 positive animals (405 equidae, 63 bovine, and 2 llamas). By the end of 2005, 445 premises from nine states (AZ, CO, ID, MT, NE, NM, TX, UT, and WY) had been quarantined. VS cases in 2006 were limited to the state of Wyoming (13 premises quarantined, 29 positive animals). In 2009, VS was detected in both Texas and New Mexico (five premises quarantined, 7 positive animals). In 2010, VS was limited to Arizona (2 premises quarantined, 4 positive animals).

Based on economic data collected after the 1982 VS outbreak in two California dairy herds, dollars lost per cow varied from $202 to $97. Both were intensively managed, dry lot dairies with close animal contact that facilitated disease spread. During the 1995 outbreak in the western U.S., New Mexican beef cattle owners put the cost per head at $53 for each case of VS. Losses were attributed to increased culling, reduced milk production, increased mortality, and labor, medicine, and veterinary costs.
Vesicular Stomatitis

**Epidemiology**

Vesicular stomatitis has historically been a western hemisphere disease; it is indigenous to North and Central America and the northern part of South America. However, according to the OIE, VS was suspected in Bahrain and Laos in 2009. VS also occurred in Pakistan in 2009, although disease was limited to certain regions of the country. In the southwest U.S., outbreaks tend to occur in the warmer regions around riverways and valleys but occasionally occur in more temperate regions. In the southeast U.S., an enzootic cycle exists in which sandflies serve as vectors.

**Geographic Distribution**

- Western hemisphere
  - North, Central, and South America
- Emergence in eastern hemisphere?
  - 2009: Bahrain, Laos (suspected)
  - 2009: Pakistan (limited regions)
- Southwest U.S.
  - Outbreaks in warmer regions
  - Southeast U.S.: enzootic cycle

**Morbidity/ Mortality**

- Morbidity
  - Range: 5 to 90%
  - Most animals seroconvert
- Mortality
  - Higher in adults
  - Death rare in cattle and horses

The morbidity rate for vesicular stomatitis is highly variable, and ranges from 5% to more than 90%. Typically, 5-20% of the animals in a herd are symptomatic, but up to 100% seroconvert. Most cases occur in adults; young cattle and horses under a year of age are uncommonly affected. Deaths are very rare in cattle and horses, but higher mortality rates have been seen in some pigs infected with VSV-NJ.

**Transmission**

Transmission of VSV can occur in many ways. Vectors, such as sand flies (*Lutzomyia shannoni*) and black flies (*Simuliidae*) can transmit the virus to other animals through bites (injection); they can also pass the virus transovarially to their offspring. There is ample evidence to indicate this is an arthropod borne disease due to its seasonal patterns; cases typically begin in spring/early summer and disappears after first frost. However, the reservoir and amplifying hosts for VSV are unknown. Direct contact with infected animals’ saliva, exudate, epithelium of open vesicles or contaminated objects is also effective. This is likely the mode of transmission in concentrated animal populations.

Photo: The top photo is of a sandfly and the bottom photo of a blackfly.
Humans may be infected by contact with the lesions or secretions from infected animals, particularly vesicular fluid and saliva. In addition, some people are probably infected through insect bites. Aerosol transmission occurs in laboratories.

The incubation period in animals is 3 to 5 days. Affected animals develop fever and clinically the lesions resemble those of foot and mouth disease. Unlike FMD, horses are affected very severely with oral and coronary band vesicles. This causes drooling, chomping, mouth rubbing, and lameness.

Photo: The above photo depicts an ulcerated lesion on the upper gums of a horse. From USDA APHIS.

Cattle and pigs develop oral vesicles, causing salivation, and vesicles on the mammary gland, coronary band and interdigital region leading to lameness. These vesicles seem to isolate to one area of the body unlike other vesicular diseases. Recovery is within 2 weeks if there is no secondary infection.

Photo: The photo is a vesicle on the tongue of a pig with vesicular stomatitis. From www.vet.uga.edu/esp/OLD_Svd/VS_tongue.
Clinically, all vesicular diseases produce a fever with vesicles that progress to erosions in the mouth, nares, muzzle, teats and feet. Vesicular diseases are clinically indistinguishable from one another, especially in swine as this chart shows, and diagnosis can only be made through virus isolation initially. Any disease with vesicles and fever should be reported to a state or federal veterinarian.

Erosive ulcerative lesions can be seen post mortem, generally in the mouth. Also, the teats and coronary bands of cattle may show lesions. Histologically, edema and degeneration of epithelial cells can be seen.

Photos: The top photo is an ulcerative lesion on the tongue of a horse (antemortem) and the bottom photo shows the clinical signs seen in cattle: teat vesicles and erosions. From USDA APHIS.

Differential diagnosis for oral lesions in cattle include FMD, rinderpest, infectious bovine rhinopneumonitis (IBR), bovine virus diarrhea (BVD), malignant catarrhal fever (MCF), bluetongue and chemical or thermal burns. In pigs, differentials include FMD, swine vesicular disease, vesicular exanthema in swine, foot rot, chemical and thermal burns. For sheep, be suspicious of FMD, bluetongue, contagious ecthyma, lip and leg ulceration, foot rot, and chemical or thermal burns.

Contact authorities if you are suspicious of a vesicular disease. Samples must be properly obtained, securely packaged, and sent to authorized laboratories for diagnosis. Call before sampling as a USDA trained Foreign Animal Disease Diagnostician (FADD) will need to collect and ship the samples.

Clinically, vesicular diseases are indistinguishable from one another, especially in swine. However, vesicular stomatitis should be suspected in horses with salivation or lameness related to vesicles. VSV is not as contagious nor does it spread as rapidly through a group of animals as FMD. Also, VSV lesions are often found in one area of the body.

Photo: The photo depicts a cow drooling due to oral vesicles. From USDA APHIS.
Vesicular Stomatitis

Laboratory Diagnosis
- Virus isolation
- Viral antigen detection
  - Vesicular fluid or epithelium
  - ELISA, complement fixation, virus neutralization
- Antibody tests
  - Paired serum samples
  - ELISA, complement fixation, virus neutralization

VSV can be isolated in tissue culture, embryonated chicken eggs or mice. Viral antigen can be detected in vesicular fluid or the base of the vesicle epithelium using ELISA, complement fixation, or virus neutralization tests. Antibody tests can also be done on paired serum samples (acute and convalescent).

Treatment
- No specific treatment available
- Supportive care
  - Fresh, clean water
  - Electrolytes if necessary
  - Soft feeds
- Antibiotics for secondary infection
- Good prognosis
- Production animals may suffer losses

As for most viruses, there is no treatment available. Supportive care such as fresh, clean water with/without electrolytes and soft feed, such as silages, and fresh grasses, will decrease the anorectic period. If secondary infection is present, antibiotics should be used. Prognosis is good for VSV infection but production losses can be permanent if the udder of cattle is affected.

Clinical Signs in Humans
- Incubation period: 1 to 6 days
- Influenza-like symptoms
  - Headache, fever, retrobulbar pain when moving eyes, malaise, nausea, limb and back pain, and rarely, oral vesicles. It is a self-limiting disease and treatment consists of supportive care. Recovery can be prolonged but death is rare.

Disease in Humans

Following an incubation of 1 to 6 days, humans may display influenza-like symptoms. These include headache, fever, retrobulbar pain when moving eyes, malaise, nausea, limb and back pain, and rarely, oral vesicles. It is a self-limiting disease and treatment consists of supportive care. Recovery can be prolonged but death is rare.

Clinical Signs in Humans
- Incubation period: 1 to 6 days
- Influenza-like symptoms
  - Headache, fever, retrobulbar pain, malaise, nausea, limb and back pain, oral vesicles (rare)
- Self-limiting disease, supportive care
- Recovery can be prolonged
- Death is rare

Clinical diagnosis is difficult as many patients only exhibit flu-like symptoms and never seek treatment. Differentials include Coxsackie A group viruses (which includes hand, foot, and mouth disease), herpes simplex, and very rarely foot and mouth disease. Diagnosis is through serology as virus isolation has been unsuccessful.

Diagnosis in Humans
- Clinical diagnosis difficult
  - Flu-like illness
  - Many do not seek treatment
- Differentials include:
  - Coxsackie A group viruses (Hand, foot and mouth disease)
  - Herpes simplex
- Diagnosis via serology
Humans that are handling tissues infected with VSV should take biosafety level 3 precautions. If in the field, wearing proper protective equipment to protect the mucous membranes and broken skin from direct contact will help decrease the chance of infection. VS is a zoonotic disease of low incidence and those affected display an influenza-like virus that rarely causes vesicle formation. Recovery occurs within 4 to 7 days if not secondarily infected.

If you suspect a case of vesicular stomatitis, state or federal authorities should be notified immediately. Animals suspected with vesicular stomatitis should be isolated, and the farm should be quarantined until definitive diagnosis is determined.

Many different disinfectants will inactivate VSV when contact is 10 minutes or longer and the item is free of organic matter. Phenolic and halogen-based disinfectants work the best. Others include 2% sodium carbonate (soda ash), 2% iodophores, chlorine dioxide, 1% sodium hypochlorite (chlorine bleach), 1% cresylic acids, quaternary ammonium (Rocco 1:200), and poly ethoxyethanol- iodine complex (4% Wescodyne).

Commercial vaccines are used in some endemic regions of Central and South America, but are not available in the U.S. There are inactivated and attenuated vaccines that may be made available during an outbreak but efficacy is unknown. Contact your state veterinarian for availability information.
Prevention

• Do not buy from positive herds for 3 months post-infection
• Avoid grazing at peak insect feeding hours
• Segregation and isolation necessary for controlling spread
• Sanitation
• Insect control programs

Advise producers not to purchase animals from herds that have had VSV in the last three months, as they could introduce it into the naïve herd. Avoid grazing in insect-laden areas or at peak insect feeding hours if in an area of concern for VSV. If VSV is diagnosed in the herd or area, segregation and isolation of animals is necessary to control the spread of the disease. Animals on pasture should be moved to stables to prevent further spread. Good sanitation practices (cleaning out feed twice daily, disinfect feed bunks and water troughs) and quarantine are essential for control until the viral infection fades from the area. Insect control programs need to be implemented to prevent vector-spread.

Additional Resources

• World Organization for Animal Health (OIE) – www.oie.int
• Center for Food Security and Public Health – www.cfsph.iastate.edu

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