Swine Vesicular Disease

Porcine Enterovirus Infection, Enfermedad Vesiclar del Cerdo, Maladie Vesiculose du Porc

Last Updated: December 2017

Importance

Swine vesicular disease (SVD) is a viral disease of pigs that is characterized by the formation of vesicles and erosions on the hooves and around the mouth. The clinical signs can vary in severity, but the illness is short and not life-threatening. Its main significance is the strong resemblance to other vesicular diseases, particularly foot-and-mouth disease. Rapid differentiation of these two diseases is critical, as the introduction of foot-and-mouth disease could result in severe economic losses in disease-free regions. Swine vesicular disease can also cause economic losses from export restrictions. Because many of the currently circulating strains cause a mild illness or infect pigs subclinically, this virus can be difficult to detect unless a country conducts active surveillance with laboratory testing.

Etiology

Swine vesicular disease virus (SVDV) is a member of the genus *Enterovirus* in the family Picornaviridae. It is currently assigned to the viral species *Enterovirus B*. There is only one SVDV serotype, but a number of strains with differing virulence have been identified. Genetic and antigenic analyses have classified SVDV isolates into at least four phylogenetically distinct groups. Two of these groups contain viruses found before 1981; the other groups contain more recent European isolates.

SVDV appears to have evolved from enterovirus B strains that circulate in humans. It is suspected to have originated from human coxsackievirus B5 (CVB5), possibly by recombination with coxsackievirus A9 (CVA9), and may have emerged shortly before the first SVD outbreaks in the 1960s. SVDV may not be the only human enterovirus to have caused outbreaks in pigs. One SVDV-like virus, which was isolated during the 1970s in the Soviet Union, appears to have resulted from the transfer of human coxsackievirus B4 (CVB4) into pigs. However, this virus did not spread widely or become established in swine populations.

Species Affected

Domesticated pigs (*Sus scrofa*) are the only known natural hosts for SVDV. There is no definitive evidence for infections in other members of the Suidae; however, Eurasian wild boar (*S. scrofa*) are likely to be susceptible. Serological surveys suggest that wild boar probably do not serve as reservoir hosts in Europe.

Sheep and one-day-old mice have been infected experimentally: sheep housed with experimentally infected pigs seroconverted, and the virus was shed for a short time, but clinical signs were not seen. Field observations during outbreaks suggest that sheep do not have a significant role in the epidemiology of this disease. Attempts to transmit SVDV to cattle directly or by contact with pigs were unsuccessful. Guinea pigs, rabbits, hamsters and donkeys also did not appear to be susceptible.

Zoonotic potential

Humans have been infected with SVDV while working with this virus in the laboratory. However, there are no reports of seroconversion or disease in farmers or veterinarians after contact with infected pigs.

Geographic Distribution

Swine vesicular disease was formerly endemic in much of Europe, but it has been eradicated except in southern Italy. This disease also occurred in parts of Asia, where the last report of an outbreak was in China in 1999. Many countries report never having found swine vesicular disease. One issue with detecting SVDV is that recent strains often circulate without causing significant clinical signs, and unless routine surveillance is conducted, these viruses may not be found.

Transmission

SVDV can be acquired by direct contact or from a contaminated environment, via mucous membranes or broken skin and by ingestion. Airborne transmission is insignificant, and this virus may not spread between pens unless there is a source of environmental or fomite-mediated transmission, such as a common open drainage

The Center for Food Security & Public Health

Institute for International Cooperation in Animal Biologics

Iowa State University College of Veterinary Medicine

World Organisation for Animal Health Founded as OIE

USDA

system, or the pigs are moved or mixed. Pigs can shed SVDV in nasal and oral fluids, feces, urine and semen. Shedding can begin up to 48 hours before the onset of clinical signs. This virus also occurs in vesicles. Raw or undercooked tissues from swine can transmit SVDV if they are fed to other pigs. Most animals eliminate this virus within two weeks, but in rare cases, they may remain infected for up to three months. Virus has been detected in the nasal secretions and tonsils of these pigs, and for particularly long periods in the feces.

SVDV can survive for long periods in the environment, and fomites are important in transmission. This virus has been found in and on worms in the soil where infected pigs were buried, and in the nasal passages of farmers. It is relatively resistant to heat, and it can survive desiccation, freezing and a wide pH range. Viable virus has been detected after 4-11 months at pH 2.5 to 12, when the temperature is between 12°C (54°F) and –20°C (-4°F). Under some conditions, SVDV can survive up to 2 years in dried, salted or smoked meat; under other conditions, it may be inactivated within a year.

Disinfection

SVDV is resistant to many commonly used disinfectants such as alcohols, lipid solvents, phenolics, quaternary ammonium compounds and 2% citric or acetic acid. It can be inactivated with sodium hydroxide (NaOH), a strong alkali; however, some other alkali disinfectants, such as sodium carbonate and sodium metasilicate, have limited or poor efficacy. Formaldehyde was effective in one study, but glutaraldehyde was ineffective. Disinfectants reported to inactivate SVDV in the absence of organic matter, given a sufficient contact time and concentration of the agent, include some oxidizing agents (e.g., sodium hypochlorite) and tincture of iodine. Certain combinations of agents (e.g., NaOH combined with iodine) have also demonstrated efficacy. Iodophors alone had only moderate activity against SVDV in one study, but they are reported to be useful for personal disinfection when combined with detergents, in the absence of gross organic matter. A commercial accelerated hydrogen peroxide-based disinfectant could inactivate dried films of SVDV at the manufacturer's recommended concentration, but required double the recommended concentration and contact time for wet films. SVDV is also reported to be susceptible to 56°C (133°F) for one hour or 60°C (140°F) for 10 minutes.

Incubation Period

The incubation period is usually 2 to 7 days, but it might be longer if the dose of virus is small.

Clinical Signs

Swine vesicular disease may be subclinical, mild or severe, depending on the virulence of the strain and the husbandry conditions. Most recent outbreaks have been caused by less virulent strains.

Clinical cases are characterized by the development of vesicles on the legs, and, less frequently, around the mouth. Vesicles can rupture quickly, turning into shallow erosions. Blanching of the epithelium may be noted before the vesicles appear. On the legs, common sites of vesicle formation include the coronary bands and interdigital spaces, but lesions can also occur at other locations, particularly pressure points such as the knees. Affected pigs may temporarily become lame. The hoof wall can separate from the underlying tissues in some cases, but complete hoof detachment is uncommon. SVD lesions tend to be more severe when pigs are housed on concrete, particularly damp concrete, while pigs on straw bedding or in grass may have few or no signs. Vesicles also occur occasionally on the snout, lips, tongue and teats, although they are reported to be uncommon in the oral cavity. Systemic signs may include a transient, mild fever and a decreased appetite for a few days. Weight loss is normally slight and the weight is regained within a short time. Pregnant animals do not usually abort. Neurological signs have been reported but are rare; reported signs include shivering, an unsteady gait and chorea (rhythmic jerking) of the legs. Most pigs recover completely within 2-3 weeks; however, a dark horizontal line may be seen on the hooves where growth was temporarily interrupted.

Post Mortem Lesions

Click to view images

The only post mortem lesions are the vesicles seen in live pigs.

Diagnostic Tests

SVDV, its antigens and/or nucleic acids may be detected in lesions (e.g., vesicular fluid, the epithelial covering, scrapings, deep swabs of erosions), nasal and oral swabs, feces and some other secretions and excretions. However, some assays are not sensitive enough for use with all types of clinical samples. While SVDV is a stable virus, samples should be handled as if they may contain either SVDV or the more fragile foot-and-mouth disease virus.

Viral antigens can be detected in vesicular lesions with ELISAs. Antigen concentrations in the feces are usually too low to be found with this test. Other types of antigen-detection assays are uncommonly used, although immunohistochemistry can be employed and complement fixation was used in the past.

RT-PCR assays can detect nucleic acids in many types of clinical samples including lesion material, oral and nasal swabs, and feces. Fecal samples are particularly useful for recognizing subclinically infected pigs. Oral fluids also appear promising. Some published multiplex RT-PCR assays can simultaneously detect SVDV and the viruses that cause other vesicular diseases, such as foot-and-mouth disease, vesicular stomatitis and vesicular exanthema of swine. Loop-mediated isothermal amplification assays and lateral flow tests for SVDV have also been published.

Swine vesicular disease can also be diagnosed by virus isolation; however, this is now infrequently done. The World Organization for Animal Health (OIE) recommends that isolation of SVDV be attempted if RT-PCR or an antigen-

detection ELISA indicates that it may be present, but this is not supported in the herd by clinical signs, serology or an epidemiological link to an outbreak. SVDV can be recovered in porcine cell cultures including IB-RS-2 cells, and identified by antigen-detection ELISA or RT-PCR. False negative results are possible if other enteroviruses are present in the sample. While these viruses can be distinguished when confirming the virus's identity, they may outgrow SVDV or affect its growth.

Swine vesicular disease is often diagnosed by serology, especially during surveillance or export certification. The most commonly used serological tests are virus neutralization (the microneutralization test) and ELISAs. Transient false positive reactions are seen occasionally, although the cause is not known: approximately 0.2-0.4% of unexposed pigs are positive or equivocal in ELISAs, and about half of these samples are also positive when they are retested by virus neutralization. These 'singleton reactors' can be identified by retesting them and their cohorts. The absence of seropositive cohorts and a constant, declining or negative second titer suggests that the animal is not infected. Serum from a singleton reactor also contains only antigen-specific IgM, while sera from infected pigs usually have specific IgG, or both IgG and IgM. In an immunoblot, the serum from singleton reactors displays a wide variety of patterns, while sera from positive animals react almost exclusively with the VP1 protein. Only one singleton reactor is usually identified in a herd, but rare incidents with multiple reactors have been published.

Treatment

There is no specific antiviral therapy for swine vesicular disease. In countries where treatment is allowed, supportive measures may be helpful.

Control

Disease reporting

A quick response is vital for containing outbreaks in disease-free regions. Veterinarians who encounter or suspect infection with SVDV should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal veterinary authorities should be informed immediately.

Prevention

In SVDV-free areas, preventive measures include screening imported pigs, restricting the importation of pork products that may contain virus, restricting swill feeding, and regulating the disposal of garbage from international airplanes and ships. Some countries conduct routine surveillance and pre- and post-export testing, particularly in Europe. The detection of SVDV is complicated by the existence of strains that produce very mild disease or subclinical infections. Some pigs infected with these viruses have only low antibody titers, which can be missed by routine surveillance using ELISAs. In endemic areas, biosecurity measures that exclude potentially infected animals and contaminated fomites can be very helpful for protecting uninfected farms. No commercial vaccines are available.

Outbreaks are controlled by quarantining infected farms and regions, tracing pigs that may have been exposed, culling infected and in-contact pigs, and cleaning and disinfecting the affected premises. While swine vesicular disease is considered only moderately contagious, rapid transmission is possible in swine-dense areas, and depopulation of groups of farms may be necessary in this situation. The persistence of SVDV in the environment complicates eradication, and if disinfection is inadequate, it can become re-established from this source. Some outbreaks in Italy were linked to inadequately disinfected vehicles used to move swine. Disposal methods for carcasses must also be adequate.

Morbidity and Mortality

SVDV seems to have emerged sometime around 1960 from a human enterovirus, and caused a number of outbreaks in Europe and Asia. Since that time, the circulating strains have evolved to become less virulent, and they may cause few or no clinical signs. These strains can circulate inapparently in pigs.

Mortality is negligible in swine vesicular disease, while the morbidity rate varies between herds, depending on factors such as the virulence of the strain, the age of the animals, and the husbandry conditions. Clinical signs tend to be more severe in young pigs, and in pigs housed on concrete floors, particularly when they are damp. All pens on a farm may not be affected, but in individual pens, the morbidity rate can reach 100%.

Public Health

Seroconversion to SVDV and clinical cases have only been reported in laboratory workers. Most symptomatic cases were mild and characterized by flu-like illnesses. However, one case of meningitis was associated with SVDV.

Internet Resources

The Merck Veterinary Manual

United States Animal Health Association. Foreign Animal Diseases

Public Health Agency of Canada. Pathogen Safety Data Sheets

World Organization for Animal Health (WOAH)

WOAH Manual of Diagnostic Tests and Vaccines for Terrestrial Animals

WOAH Terrestrial Animal Health Code

Acknowledgements

This factsheet was written by Anna Rovid Spickler, DVM, PhD, Veterinary Specialist from the Center for Food Security and Public Health. The U.S. Department of

Agriculture Animal and Plant Health Inspection Service (USDA APHIS) provided funding for this factsheet through a series of cooperative agreements related to the development of resources for initial accreditation training.

The following format can be used to cite this factsheet**.** Spickler, Anna Rovid. 2017. *Swine Vesicular Disease*. Retrieved from http://www.cfsph.iastate.edu/DiseaseInfo/factsheets.php.

References

Althouse GC, Rossow K. The potential risk of infectious disease dissemination via artificial insemination in swine. Reprod Domest Anim. 2011;46 Suppl 2:64-7.

Bellini S, Alborali L, Zanardi G, Bonazza V, Brocchi E. Swine vesicular disease in northern Italy: diffusion through densely populated pig areas. Rev Sci Tech. 2010;29(3):639-48.

Bellini S, Santucci U, Zanardi G, Brocchi E, Marabelli R. Swine vesicular disease surveillance and eradication activities in Italy.Rev Sci Tech. 2007;26(3):585-93.

Blackwell JH, Graves JH, McKercher PD. Chemical inactivation of swine vesicular disease virus. Br Vet J. 1975;131(3):317-23.

Blomström AL, Hakhverdyan M, Reid SM, Dukes JP, King DP, Belák S, Berg M. A one-step reverse transcriptase loop-mediated isothermal amplification assay for simple and rapid detection of swine vesicular disease virus. J Virol Methods. 2008;147(1):188-93.

Bruhn CA, Nielsen SC, Samaniego JA, Wadsworth J, Knowles NJ, Gilbert MT. Viral meningitis epidemics and a single, recent, recombinant and anthroponotic origin of swine vesicular disease virus. Evol Med Public Health. 2015;2015(1):289-303.

Burrows R, Mann JA, Goodridge D, Chapman WG. Swine vesicular disease: attempts to transmit infection to cattle and sheep. J Hyg (Lond). 1974;73(1):101-7.

Elbers AR, Dekkers LJ, van der Giessen JW. Sero-surveillance of wild boar in The Netherlands, 1996-1999. Rev Sci Tech. 2000;19(3):848-54.

Escribano-Romero E, Jiménez-Clavero MA, Ley V. Swine vesicular disease virus. Pathology of the disease and molecular characteristics of the virion. Anim Health Res Rev. 2000;1:119-26.

Fernández J, Agüero M, Romero L, Sánchez C, Belák S, Arias M, Sánchez-Vizcaíno JM. Rapid and differential diagnosis of foot-and-mouth disease, swine vesicular disease, and vesicular stomatitis by a new multiplex RT-PCR assay. J Virol Methods. 2008;147(2):301-11.

Ferris NP, Nordengrahn A, Hutchings GH, Paton DJ, Kristersson T, Merza M. Development and laboratory evaluation of a lateral flow device for the detection of swine vesicular disease virus in clinical samples. J Virol Methods. 2010;163(2):477-80.

Garner G, Saville P, Fediaevsky A. Manual for the recognition of exotic diseases of livestock: A reference guide for animal health staff [online]. Food and Agriculture Organization of the United Nations [FAO]; 2003. Swine vesicular disease. Available at: http://www.spc.int/rahs/.* Accessed 28 Dec 2007.

Hälli O, Ala-Kurikka E, Nokireki T, Skrzypczak T, Raunio-Saarnisto M, Peltoniemi OA, Heinonen M. Prevalence of and risk factors associated with viral and bacterial pathogens in farmed European wild boar. Vet J. 2012;194(1):98-101.

Hole K, Ahmadpour F, Krishnan J, Stansfield C, Copps J, Nfon C. Efficacy of accelerated hydrogen peroxide® disinfectant on foot-and-mouth disease virus, swine vesicular disease virus and senecavirus A. Appl Microbiol. 2017;122(3):634-9.

International Committee on Taxonomy of Viruses [ICTV]. Virus Taxonomy: 2016 Release EC 48, Budapest, Hungary, August 2016; Email ratification 2017 (MSL #31). *Enterovirus.* ICTV; 2017. Available at: https://talk.ictvonline.org/taxonomy/. Accessed 11 Dec 2017.

Lin F, Kitching RP. Swine vesicular disease: an overview. Vet J. 2000;160:192-201.

Lomakina NF, Shustova EY, Strizhakova OM, Drexler JF, Lukashev AN. Epizootic of vesicular disease in pigs caused by coxsackievirus B4 in the Soviet Union in 1975. J Gen Virol. 2016;97(1):49-52.

Lung O, Fisher M, Beeston A, Hughes KB, Clavijo A, Goolia M, Pasick J, Mauro W, Deregt D. Multiplex RT-PCR detection and microarray typing of vesicular disease viruses. J Virol Methods. 2011;175(2):236-45.

Montagnaro S, Sasso S, De Martino L, Longo M, Iovane V, Ghiurmino G, Pisanelli G, Nava D, Baldi L, Pagnini U. Prevalence of antibodies to selected viral and bacterial pathogens in wild boar (*Sus scrofa*) in Campania Region, Italy. J Wildl Dis. 2010;46(1):316-9.

Nardelli L, Lodetti E, Gualandi GL, Burrows R, Goodridge D, Brown F, Cartwright B. A foot-and-mouth disease syndrome in pigs caused by an enterovirus. Nature. 1968; 219:1275.

Pannwitz G, Haas B, Hoffmann B, Fischer S. [Serological examinations for swine vesicular disease (SVD) in a closed pig breeding herd using ELISA]. Berl Munch Tierarztl Wochenschr. 2009;122(5-6):161-8.

Public Health Agency of Canada [PHAC]. Pathogen Safety Data Sheet – Coxsackievirus. Pathogen Regulation Directorate, PHAC; 2011 Nov. Available at: https://www.canada.ca/en/public-health/services/laboratory-biosafety-biosecurity/pathogen-safety-data-sheets-risk-assessment/coxsackievirus-pathogen-safety-data-sheet.html. Accessed 13 Nov 2017.

Rodriguez-Sanchez B, Sanchez-Vizcaino JM, Uttenthal A, Rasmussen TB, Hakhverdyan M, et al. Improved diagnosis for nine viral diseases considered as notifiable by the World Organization for Animal Health. Transbound Emerg Dis. 2008;55(5-6):215-25.

Roic B, Jemersic L, Terzic S, Keros T, Balatinec J, Florijancic T. Prevalence of antibodies to selected viral pathogens in wild boars (*Sus scrofa*) in Croatia in 2005-06 and 2009-10. J Wildl Dis. 2012;48(1):131-7.

Sedlak K, Bartova E, Machova J. Antibodies to selected viral disease agents in wild boars from the Czech Republic. J Wildl Dis. 2008;44(3):777-80.

Senthilkumaran C, Bittner H, Ambagala A, Lung O, Babiuk S, Yang M, Zimmerman J, Giménez-Lirola LG, Nfon C. Use of oral fluids for detection of virus and antibodies in pigs infected with swine vesicular disease virus. Transbound Emerg Dis. 2017;64(6):1762-70.

Shirai J, Kanno T, Tsuchiya Y, Mitsubayashi S, Seki R. Effects of chlorine, iodine, and quaternary ammonium compound disinfectants on several exotic disease viruses. J Vet Med Sci. 2000;62(1):85-92.

Torres A. Swine vesicular disease. In: Foreign animal diseases, 7th ed. Boca Raton, Fl: United States Animal Health Association, 2008. p. 397-400.

Vengust G1, Valencak Z, Bidovec A. A serological survey of selected pathogens in wild boar in Slovenia. J Vet Med B Infect Dis Vet Public Health. 2006;53(1):24-7.

World Organization for Animal Health [OIE] . Manual of diagnostic tests and vaccines for terrestrial animals [online]. Paris: OIE; 2017. Swine vesicular disease. Available at: http://www.oie.int/fileadmin/Home/eng/Health_standards/tahm/2.08.08_SVD.pdf. Accessed 9 Dec 2017.

World Organization for Animal Health (OIE). Technical disease cards [online]. Swine vesicular disease. Available at: http://www.oie.int/animal-health-in-the-world/technical-disease-cards/. Accessed 11 Dec 2017.

World Organization for Animal Health [OIE]. World Animal Health Information Database (WAHIS) Interface. Swine vesicular disease. Available at: http://www.oie.int/wahis_2/public/wahid.php/Wahidhome/Home. Accessed 11 Dec 2017.

Zoni R(1), Zanelli R, Riboldi E, Bigliardi L, Sansebastiano G. Investigation on virucidal activity of chlorine dioxide. experimental data on feline calicivirus, HAV and coxsackie B5. J Prev Med Hyg. 2007;48(3):91-5.

*Link defunct