Classical Swine Fever

Hog Cholera,
Swine Fever,
European Swine Fever,
Peste du Porc,
Colera Porcina,
Virusschweinepest

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Importance

Classical swine fever (CSF) is a highly contagious and economically significant viral disease of pigs. The severity of the illness varies with the strain of the virus, the age of the pig, and the immune status of the herd. Acute infections, which are caused by highly virulent isolates and have a high mortality rate in naive herds, are likely to be diagnosed rapidly. However, infections with less virulent isolates can be more difficult to recognize, particularly in older pigs. The range of clinical signs and similarity to other diseases can make classical swine fever challenging to diagnose.

Although classical swine fever was once widespread, many countries have eradicated this disease from domesticated swine. Reintroduction of the virus can be devastating. In 1997-1998, an outbreak in the Netherlands spread to involve more than 400 herds and cost $2.3 billion to eradicate. Approximately 12 million pigs were killed, some in eradication efforts but most for welfare reasons associated with the epidemic. Other European counties have also experienced outbreaks, and the ongoing presence of the virus among wild boar presents a risk of reintroduction to domesticated swine. North America is also at risk for the reintroduction of classical swine fever, which is still endemic in South and Central America.

Etiology

Classical swine fever (hog cholera) results from infection by classical swine fever virus (CSFV), a member of the genus *Pestivirus* and family Flaviviridae. There is only one serotype but several genotypes and subgenotypes. CSFV is closely related to pestiviruses found in ruminants, and some of the latter viruses can cause serological reactions in pigs that may be mistaken for CSF.

Species Affected

CSFV appears to be capable of infecting most or all members of the pig family (Suidae). Clinical cases occur in domesticated pigs and Eurasian wild boar. CSFV has been detected in a white-lipped peccary (*Tayassu pecari*), and experimental infections have been established in common warthogs (*Phacochoerus africanus*), bush pigs (*Potamochoerus larvatus*) and collared peccaries (*Tayassu tajacu*).

Experimental infections without clinical signs have been reported in cattle, sheep, goats and deer, but there is no evidence that these species become infected in nature. Strains of CSFV can also be adapted to passage in rabbits.

Zoonotic potential

There is no evidence that CSFV infects humans.

Geographic Distribution

Classical swine fever is endemic in parts of Asia, parts of South and Central America, and on some Caribbean islands. CSFV has been eradicated from a number of countries, including the U.S., Canada, New Zealand, Australia, Iceland and Japan. It is also absent from the domesticated pig populations of most of western and central Europe, although it is still present among wild boar in some regions. The status of classical swine fever in some areas of Africa may be uncertain, due to limited or no surveillance; as of 2014, it was reported to the World Organization for Animal Health (OIE) as endemic in Madagascar, suspected in Equatorial Guinea in 2013, and either absent or eradicated in other reporting countries.

Transmission

Pigs are mainly thought to become infected by the oral or oronasal routes. CSFV may also enter the body via other mucus membranes (including genital transmission in semen), and skin abrasions. It can be shed in oronasal and ocular secretions, urine, feces and semen; one study reported that pigs infected with strains of low virulence excreted the virus mainly in oronasal secretions. Shedding can begin before the onset of clinical signs. Because CSFV can persist in blood and tissues after death, it is readily spread by feeding uncooked swill that contains tissues from infected pigs. Aerosol transmission has been demonstrated experimentally with some strains. It is most likely to occur between mechanically ventilated buildings in close proximity,

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Institute for International Cooperation in Animal Biologics

Iowa State University College of Veterinary Medicine

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when there are large concentrations of animals. Piglets and wild boar infected before birth or shortly thereafter (i.e., on the day of birth in one experiment) may become persistently infected without developing an antibody response to CSFV. These animals can shed the virus continuously or intermittently for months.

CSFV can be spread on fomites, and may be transmitted on live mechanical vectors such as insects. Estimates of its persistence in the environment differ, and are influenced by the initial concentration of virus and the presence of organic matter. Some studies suggest that CSFV is inactivated on some fomites, in feces (or slurry) and in urine within a few days to 2 weeks at room temperature (e.g., 20ºC). Others describe survival at 4-5ºC for 1-3 months when protected by material such as pig slurry. One study reported that this virus also persisted in pig slurry for at least 70 days at 17ºC. CSFV can remain infectious for nearly three months in refrigerated meat and for more than four years in frozen meat. In this proteinaceous environment, it does not appear to be inactivated by smoking or salt curing. Reported virus survival times in cured and smoked meats vary with the technique, and range from 17 days to more than 180 days.

Disinfection

CSFV can be inactivated with sodium hypochlorite, phenolic compounds, detergents, organic solvents, quaternary ammonium compounds and aldehydes (formaldehyde, glutaraldehyde). It is also sensitive to drying, heat and ultraviolet light. This virus is reported to be destroyed by heating for a minute or less at 90-100ºC or 5 minutes at 70°C. In meat, CSFV is susceptible to a temperature of 65.5ºC or higher, maintained for 30 minutes. It is stable at pH 5-10, but inactivated by pH $\leq$ 3 or pH > 10.

Incubation Period

The incubation period can range from 2 to 15 days, and is often 3-7 days in acute cases. Under field conditions, the disease may not become evident in a herd for 2 to 4 weeks or longer.

Clinical Signs

The clinical signs vary with the strain of CSFV, and the age and susceptibility of the pigs. While highly virulent strains were prevalent in the past, most outbreaks are now caused by moderately virulent strains, and the clinical signs are often less severe and distinctive.

Highly virulent strains of CSFV tend to cause acute, severe illness in naive herds. Common clinical signs in the acute form include a high fever, huddling, weakness, drowsiness, anorexia and conjunctivitis, which can cause severe crusting of the eyelids. Constipation, with the passage of hard fecal pellets, is typically followed by, or intermittent with, watery diarrhea. Pigs may be incoordinated or exhibit an unsteady, weaving or staggering gait, which often progresses to posterior paresis. Some pigs may vomit yellow, bile-containing fluid, or develop respiratory signs. The skin can become hyperemic, and may develop hemorrhages (especially on the abdomen, inner thighs, ears) or a purple cyanotic discoloration, which tends to be seen on the snout, ears and tail. Severe leukopenia is a common laboratory abnormality. Pigs with acute classical swine fever often die within 1-3 weeks, and convulsions may occur in the terminal stages. The subacute form is similar; however, the signs are less severe, the course is prolonged, and the mortality rate lower. Blotching of the ears has been described in both subacute and chronic cases.

Chronic disease tends to be seen with less virulent strains or in partially immune herds, and may affect only a few animals. In the initial stages, it can resemble the other forms, with signs such as anorexia, depression, elevated temperature, leukopenia, and periods of constipation and/or diarrhea. Affected pigs usually improve after several weeks, but after a period where they may appear relatively normal, these signs can recur. Additional signs include wasting or stunted growth, alopecia and skin lesions, and immunosuppression may lead to concurrent infections. The clinical signs can wax and wane for weeks to months, and the outcome is often fatal.

Poor reproductive performance may be the only sign in some breeding herds infected with less virulent strains. Sows in these herds may abort or give birth to stillborn, mummified, malformed, weak or dead piglets. Some piglets may be born with a congenital tremor or congenital malformations of the visceral organs and central nervous system. Others can be persistently infected but asymptomatic at birth. These piglets become ill after several months, with signs of "late onset" disease such as inappetence, depression, stunted growth, dermatitis, diarrhea, conjunctivitis, ataxia or posterior paresis. Although congenitally infected pigs can survive for 2 months or more, all typically die within a year.

Clinical signs in wild boar appear to be similar to the signs in domesticated pigs. Experimentally infected bushpigs also became ill and some cases were severe, with fever, anemia, prolonged clotting times upon blood collection, diarrhea and conjunctivitis. Most experimentally infected warthogs in the same study did not develop clinical signs, despite virological and serological evidence of infection. One warthog had moderate to severe diarrhea. Short-term fever and nonspecific signs were reported in collared peccaries inoculated with CSFV.

Post Mortem Lesions

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The lesions of classical swine fever are highly variable. During outbreaks, the likelihood of observing the characteristic necropsy lesions is better if four or five pigs are examined. In acute disease, the most common lesion is hemorrhage. The skin may be discolored purple and the lymph nodes may be swollen and hemorrhagic. Petechial or ecchymotic hemorrhages can often be seen on serosal and

mucosal surfaces, particularly on the kidney, urinary bladder, epicardium, epiglottis, larynx, trachea, intestines, subcutaneous tissues and spleen. In the intestines, the lesions may include hemorrhagic lesions in the stomach, mild to moderate catarrhal enteritis in the small intestine, and button ulcers in the colon. Straw–colored fluid may be found in the peritoneal and thoracic cavities and the pericardial sac. Severe tonsillitis, sometimes with necrotic foci, is common. Splenic infarcts (raised, dark, wedge-shaped lesions) are seen only occasionally with the currently circulating strains of CSFV, but are highly suspicious of this disease if detected. The lungs may be congested and hemorrhagic, and encephalitis may be noted in the brain. In some acute cases, lesions may be absent or inconspicuous.

The lesions of chronic disease are less severe and may be complicated by secondary infections. Necrotic foci or "button" ulcers may be found in the intestinal mucosa, epiglottis and larynx. Button ulcers in the intestine may be followed by diffuse, diphteroid-necrotizing enteritis. In growing pigs that have survived for more than a month, bone lesions can also occur at the costochondral junction of the ribs and the growth plates of the long bones.

In congenitally infected piglets, common lesions include cerebellar hypoplasia, thymic atrophy, ascites, and deformities of the head and legs. Edema and petechial hemorrhages may be seen in the skin and internal organs.

Diagnostic Tests

CSFV can be detected in blood or tonsil swabs collected from live animals, or in tissue samples (tonsils, pharyngeal and mesenteric lymph nodes, spleen, kidneys, distal ileum) taken at necropsy. Samples from live animals should be collected when they are febrile. Reverse transcriptase polymerase chain reaction (RT–PCR) tests, which detect viral nucleic acids, are often used for diagnosis. Loop-mediated isothermal amplification (RT-LAMP) assays have also been published. Identification to the genotype or subgenotype level, using nucleic acid-based tests, can be useful in epidemiology. CSFV antigens can be detected with direct immunofluorescence (FAT or FATST) or ELISAs. ELISAs are only considered suitable as herd tests. In addition, CSFV can be isolated in several cell lines including pig kidney (e.g. PK–15) cells, with virus identification by direct immunofluorescence, immunoperoxidase staining or RT-PCR.

Ruminant pestiviruses (e.g., bovine virus diarrhea virus and border disease virus) can occasionally infect pigs. Serum neutralization tests, or immunoperoxidase procedures that use monoclonal antibodies, can differentiate CSFV from these viruses. They can also be distinguished with genetic methods such as RT-PCR.

Serology can be useful for diagnosis and surveillance. Antibodies develop relatively late (after 2 to 3 weeks) in most animals, but persist lifelong. However, piglets (including wild boar) infected before or immediately after birth may be immunotolerant and negative on serology. The most commonly used tests are virus neutralization (e.g., the fluorescent antibody virus neutralization [FAVN] and neutralizing peroxidase–linked assay [NPLA] tests) and various ELISAs. Assays that use monoclonal antibodies can distinguish antibodies to CSFV from responses to ruminant pestiviruses. The definitive test is the comparative neutralization test.

Companion ELISAs are also available for use with marker vaccines. At present, these tests are considered suitable as herd tests, but are not reliable in individual animals.

Treatment

There is no treatment for classical swine fever, other than supportive care.

Control

Disease reporting

A quick response is vital for containing outbreaks in CSFV-free regions. Veterinarians who encounter or suspect classical swine fever 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 countries where classical swine fever is endemic, this disease may be excluded from a herd by buying animals from CSFV-free herds, quarantining the new stock for 4 months and testing the animals before allowing them to contact the rest of the herd. Vaccines can be used to protect animals from clinical signs, and may also be employed to reduce the prevalence of infections during an eradication program. Both modified live and subunit (marker) vaccines are manufactured.

Outbreaks in CSFV-free regions are generally eradicated by slaughter of confirmed cases and contact animals, cleaning and disinfection of infected premises, safe carcass disposal, movement controls/ quarantines and surveillance. Pre-emptive slaughter of animals on nearby farms and/or emergency vaccination may also be employed. Because the currently circulating viruses are often less readily detected by clinical signs, some countries conduct routine virological surveillance for CSFV, such as periodic sampling of tonsils from dead pigs.

Controlling endemic infections in wild populations is difficult. Oral vaccination is used in wild boar in Europe. Contact between domesticated herds and wild pigs should be avoided.

Morbidity and Mortality

The severity of classical swine fever is influenced by the viral strain, the age and immune status of the pigs, and other factors such as the animals' general health and viral dose. Highly virulent strains of CSFV, which were

prevalent at one time, cause outbreaks with morbidity and mortality rates that can approach 100%. However, most outbreaks are now caused by moderately virulent strains, and less virulent strains also circulate. Some strains of low virulence have caused only 20% mortality in experimentally infected pigs. Case fatality rates also differ with the form of the disease, and are very high in the acute form, but lower in subacute cases. Mortality tends to be lower in adult pigs, compared to young animals, especially with less virulent strains.

Internet Resources

The Merck Veterinary Manual
http://www.merckvetmanual.com/mvm/index.html

United States Animal Health Association.
Foreign Animal Diseases
http://www.aphis.usda.gov/emergency_response/downloads/nahems/fad.pdf

World Organization for Animal Health (OIE)
http://www.oie.int

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals
http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/

OIE Terrestrial Animal Health Code
http://www.oie.int/international-standard-setting/terrestrial-code/access-online/

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