Teschovirus Encephalomyelitis and Porcine Teschovirus Infections

Enterovirus Encephalomyelitis,
Porcine Teschovirus Polioencephalomyelitis
Teschen Disease,
Talfan Disease,
Poliomyelitis Suum,
Benign Enzootic Paresis,
Klobouk's Disease

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Importance

Porcine teschoviruses (PTV) frequently circulate in swine populations without causing clinical signs; however, some of these viruses can cause a central nervous system (CNS) disease called teschovirus encephalomyelitis. Severe outbreaks of teschovirus encephalomyelitis, caused by certain strains of serotype PTV-1, were common in parts of Europe in the 1940s and 1950s, and resulted in major economic losses to the swine industry. This illness was considered to be one of the most dangerous animal diseases of the time. Pigs of all ages were affected, and mortality rates were high. During recent decades, most reported outbreaks of teschovirus encephalomyelitis have been milder, often affecting only young animals in a few herds. Extensive outbreaks have become unusual, although a PTV-1 strain caused a major outbreak among backyard pigs in Haiti in 2009. Why some porcine teschoviruses cause teschovirus encephalomyelitis, while others circulate inapparently, is still under investigation. Some evidence suggests that outbreaks might be influenced by factors such as poor immunity to a PTV virus, co-infection with other pathogens, or the introduction of new or unusually virulent PTV strains. Porcine teschoviruses have also been implicated occasionally in other syndromes, including respiratory and enteric disease.

Etiology

Porcine teschovirus is the common name for the members of the viral species *Teschovirus A*, which belongs to the genus *Teschovirus* and family Picornaviridae. At least 13 serotypes, PTV-1 through PTV-13, have been recognized as of 2018. Serotypes 1 through 10 contain the viruses previously known as porcine enterovirus (PEV) serotypes 1-7 and 11-13. PTV 11-13 were discovered after these viruses were transferred to the new *Teschovirus* genus, and should not be confused with the former PEV serotypes 11-13.

Teschovirus encephalomyelitis has traditionally been defined as the severe illness caused by certain highly virulent strains of PTV-1. It was originally called Teschen disease. Other strains of PTV-1, as well as other serotypes of PTV, can cause similar, but typically milder, neurological syndromes. These diseases have been given a variety of names including Talfan disease, benign enzootic paresis and poliomyelitis suum. Using different names helped distinguish teschovirus encephalomyelitis, which was reportable to the World Organization for Animal Health (OIE), from milder illnesses. However, this disease is no longer reportable to the OIE, and "teschovirus encephalomyelitis" is sometimes used now for all cases with neurological signs, regardless of severity. This terminology is used in this factsheet.

Species Affected

Pigs (*Sus scrofa*) are the only known hosts for porcine teschoviruses. These viruses appear to be maintained in populations of wild boar, as well as in domesticated pigs.

Zoonotic potential

There is no indication that porcine teschoviruses affect humans.

Geographic Distribution

Porcine teschoviruses are widespread in swine populations, but the circulating strains may differ between locations. Outbreaks of severe teschovirus encephalomyelitis caused by PTV-1 strains were first reported in Europe in 1929, and were common there in the 1940s and 1950s; however, these viruses seem to have been eliminated from western Europe by 1980. Some countries in other regions, such as Madagascar, also reported serious outbreaks at the time. Mild to moderately severe outbreaks of encephalitis, caused by various serotypes, continue to be reported sporadically in a number of countries.

Transmission

Porcine teschoviruses can enter the body by ingestion or via the nasal cavity. These viruses are reported to be shed in feces, urine, oral fluids, and possibly

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respiratory secretions. The fecal-oral route is thought to be the predominant method of transmission. Convalescent animals can excrete PTV in feces for up to 7 weeks. The possibility of persistent and/or latent infections has been suggested, based on the detection of viruses in some laboratory-housed pigs that had previously tested PTV-negative by serology and RT-PCR.

Porcine teschoviruses are readily spread on fomites, and inadequately heat-treated pig swill was thought to be significant in transmission during historical outbreaks in Europe. These viruses are reported to persist in the environment for more than 5 months at 15ºC (59ºF), and can survive in liquid manure for prolonged periods. They are reported to tolerate a wide pH range, remaining viable from pH 2 to pH 9.

Disinfection

Porcine teschoviruses and related picornaviruses are resistant to a number of disinfectants, but they can be inactivated by sodium hypochlorite. In one test of commercial disinfectants, an agent that contained peracetic acid, hydrogen peroxide and acetic acid was also effective. Disinfectants used during historical outbreaks in the 1950s and 1960s included chloramine, chloride of lime and caustic soda. Viruses in manure can be inactivated by aeration, ionizing radiation or anaerobic digestion.

Incubation Period

The incubation period for teschovirus encephalomyelitis has been estimated to be 1 to 4 weeks. In experimentally infected piglets, the highly virulent 'Zabreh' strain of PTV-1 produced clinical signs in 5-7 days. In other syndromes such as respiratory disease, the initial signs occurred as soon as 2 days after experimental inoculation.

Clinical Signs

Outbreaks of teschovirus encephalomyelitis caused by highly virulent strains of PTV-1 can affect pigs of all ages, and are typically characterized by fever, anorexia, depression and incoordination, followed by overt neurological signs. Hypersensitivity is common; affected pigs may grind their teeth, smack their lips or squeal as if they are in pain. Muscle tremors, stiffness or rigidity, nystagmus, seizures, changes in or loss of the voice, opisthotonos and clonic spasms of the legs may also be seen. In the final stage, animals usually develop progressive, ascending paralysis, starting in the hindquarters. Death is usually the result of respiratory muscle paralysis, and often occurs within 3-4 days after the onset of the illness. Animals with milder clinical signs sometimes recover.

Other PTV viruses tend to circulate subclinically; however, some can cause neurological signs, especially in young animals. Many milder outbreaks have been characterized by ataxia and paresis, occasionally progressing to paralysis, but other CNS signs can also be seen. During an outbreak that appeared to be caused by a PTV-13 virus, the clinical signs included sudden death in some animals, and flaccid paralysis, convulsions, nystagmus, opisthotonos, paddling and stereotypical chewing motions in others. Necrosis of the ear, catarrhal to suppurative keratoconjunctivitis and occasional corneal opacity were also noted in some pigs. Systemic signs are not always reported in milder outbreaks. For instance, the neurological signs in PTV-1 infected piglets in Japan were not preceded by either anorexia or fever. The mortality rate in mild to moderately severe outbreaks is variable, but mildly affected pigs can recover. Chronic loss of condition, reduced weight gain and/or persistent neurological deficits have been reported in some surviving animals.

Enteric and respiratory signs have also been seen in some pigs infected with porcine teschoviruses, including some experimentally infected pigs. Mild to moderate diarrhea, often with watery yellow feces, seems to be the most common gastrointestinal sign. Respiratory signs in various outbreaks ranged from rhinitis to dyspnea. In most cases, respiratory and/or enteric involvement was reported in animals with teschovirus encephalomyelitis. However, in one recent outbreak, acute diarrhea, respiratory signs and deaths were associated with PTV-8 infections in newly introduced 7-10-week-old pigs on a breeding farm in China, but there were no CNS signs. Pigs that were experimentally infected with this virus also developed respiratory and enteric signs.

Additional syndromes, such as myocarditis and pericarditis, have been attributed rarely to porcine teschoviruses. There are also a few reports suggesting that these viruses might be involved in reproductive losses. A PTV-1 virus was recovered from an aborted fetus during the 1970s in Europe, and in 2009, a PTV-8 virus was detected in aborted fetuses from a herd with reproductive failure characterized by abortions, stillbirths and weak piglets in China. It is often difficult to determine whether some clinical signs are caused by porcine teschoviruses, as concurrent infections are common in the field.

Post Mortem Lesions

There are no characteristic gross lesions in teschovirus encephalomyelitis, although meningitis is reported to be common over the cerebellum. Nonsuppurative polioencephalomyelitis with lymphocytic perivascular cuffing is found on histopathological examination. It occurs mainly in the gray matter of the cerebellum, diencephalon, medulla oblongata and ventral horns of the spinal cord. In very young animals, the dorsal horns of the spinal cord may be affected. Microscopic lesions are also common in the dorsal root ganglia and trigeminal ganglia.

Interstitial pneumonia and associated gross lesions have been reported in some pigs infected with porcine teschoviruses. The large intestine of some experimentally infected pigs (PTV-1) with diarrhea was reported to be flaccid, edematous and thin-walled. During a recent

outbreak of teschovirus encephalomyelitis thought to be caused by a PTV-13 virus, common gross lesions included generalized reddening of the carcass, serosanguinous exudates in body cavities (hydrothorax, ascites and hydropericardium), enlargement of the mesenteric lymph nodes, and lung lesions associated with interstitial pneumonia.

Diagnostic Tests

Teschovirus encephalomyelitis

Porcine teschoviruses, their nucleic acids and antigens can be detected in the brain and/or spinal cord of pigs with teschovirus encephalomyelitis. As these viruses are very common in asymptomatic pigs, their recovery from tissues other than the CNS is not considered to be diagnostic for this condition.

Samples for virus isolation should be taken from pigs that died very recently or were killed for necropsy. Recovery of PTV is most likely to be successful early in the course of the disease. These viruses can be isolated in porcine cells or cell lines, particularly cells from the kidney (e.g., PK-15 cells). The identity of the virus and its serotype can be confirmed with serological tests including virus neutralization and indirect immunofluorescence, as well as by genetic techniques. Serotyping can be complicated by cross-reactivity when using polyclonal antisera.

Reverse-transcription polymerase chain reaction (RT-PCR) assays can detect PTV nucleic acids in clinical samples; however, this technique is not available in all laboratories. Loop-mediated isothermal amplification assays have also been published. Teschovirus antigens may sometimes be detected in the CNS with immunohistochemistry, but these antigens are reported to be very difficult to find.

Teschovirus encephalomyelitis can also be diagnosed by serology. Microtiter virus neutralization or ELISAs are often used, but other tests such as complement fixation have been employed. A four-fold rise in the titer should be seen. Because antibodies to porcine teschoviruses are common among swine, a single titer is not diagnostic.

Histopathology of the brain and spinal cord helps support the diagnosis. Microscopic lesions are found mainly in the cerebrum, cerebellum, diencephalon, medulla oblongata, and cervical and lumbar spinal cord.

Other diseases and subclinically infected herds

Porcine teschoviruses have occasionally been implicated in conditions other than teschovirus encephalomyelitis based on a rise in antibody titers, the presence of the virus in affected tissues and the exclusion of other causative agents. A definitive diagnosis is difficult, as teschoviruses are common in asymptomatic herds, and co-infections are common. In some cases, the involvement of PTV was substantiated by inoculating the virus into experimentally infected swine; however, this would not be practical for routine diagnosis.

PTV is commonly found in the intestines, tonsils and ileal lymph nodes of subclinically infected animals. These viruses also occur sometimes in visceral organs (e.g., spleen, kidney), with or without clinical signs. More than one PTV serotype may be found in an animal.

Treatment

There is no treatment for teschovirus encephalomyelitis other than supportive care.

Control

Disease reporting

Veterinarians who encounter or suspect teschovirus encephalomyelitis should follow their national and/or local guidelines for disease reporting. In the U.S., state or federal authorities should be consulted for the current reporting requirements.

Prevention

Severe outbreaks caused by PTV-1 have been controlled by quarantines and movement controls, euthanasia of the herd, ring vaccination and tracing of contacts. Porcine teschoviruses can survive in the environment for months, and the premises must be cleaned and disinfected well. A 28-day waiting period was employed during Czechoslovakian eradication campaigns, before final disinfection and restocking. Although vaccines for teschovirus encephalomyelitis were used in eradication programs in Europe, they are no longer made.

Milder outbreaks that affect young animals may be mitigated by introducing new breeding stock at least one month before breeding. This allows these animals to develop immunity to the strains that circulate in the herd and pass maternal antibodies to their young.

Morbidity and Mortality

Porcine teschoviruses are endemic in many herds, and often circulate without causing clinical signs. One hypothesis is that teschovirus encephalomyelitis resembles diseases such as polio, where intestinal infections are common, a smaller proportion of the viruses invade the visceral organs, and only a small number of cases result in CNS signs. If this view is accurate, teschovirus encephalomyelitis might develop when an animal has insufficient immunity to control teschovirus replication in the CNS, such as when either the viral strain is highly virulent or the animal's immunity to the virus is weak. Co-infections might also increase the severity of some outbreaks. Limited evidence, to date, suggests that immunity to one serotype might not protect animals from other serotypes.

Highly virulent strains of PTV-1, which were first documented in 1929, caused extensive outbreaks in Europe in the 1940s and 1950s. Pigs of all ages were affected, and

up to 90% died within a few days of becoming ill. While there is little definitive information about how these viruses spread, biosecurity measures at the time were poor or absent, and viruses were often thought to be introduced by feeding contaminated pig swill. Czechoslovakia was severely affected in these outbreaks, with teschovirus encephalomyelitis reported on more than 65,000 farms or backyard pig units in 1952, and an incidence of nearly 2800 cases per 100,000 swine. An eradication program was begun in Czechoslovakia that year, with the last cases found there in 1973.

Outbreaks of teschovirus encephalomyelitis reported in the last 20-30 years have usually been less severe and less widespread. In many incidents, the clinical cases seem to be localized, affecting one to a few herds, and only young animals have been affected. These animals are thought to be infected primarily during weaning, when their exposure to other animals increases and maternal antibodies have declined. Cases may also be seen in younger piglets, particularly when a serotype to which they have no immunity is introduced into the herd. Recent reports of laboratory-confirmed teschovirus encephalomyelitis include a PTV-1 outbreak in Japan (2002), which affected 7 of 41 piglets in a herd (all affected piglets were euthanized), and retrospectively diagnosed PTV-1 outbreaks in Canada between 2002 and 2008, which had owner-reported morbidity rates of 0.1% to 2%, and case-fatality rates between < 0.1% and 100%. The outbreak in Japan occurred in 6-week-old pigs, while the Canadian outbreaks mainly affected 4-7 week old pigs. Most of the surviving animals in the Canadian outbreaks were euthanized due to persistent neurological deficits and/or poor weight gain. More recently, an outbreak thought to be caused by a PTV-13 virus affected 6-7-week-old pigs in 2 herds in Spain, with a 20% morbidity rate, 60% case fatality rate, and batch mortality of 10-12%. In contrast, severe and extensive outbreaks caused by a PTV-1 strain were reported among backyard pigs in Haiti and the Dominican Republic in 2009. More than 1500 pigs of all ages were affected in Haiti, with overall morbidity and mortality rates reported to be approximately 60%% and 40%, respectively. However, serological surveys conducted during these outbreaks suggested that many pigs without clinical signs had antibodies to PTV-1, suggesting that subclinical infections might also have been common. It is possible that co-circulating viruses, including classical swine fever virus, contributed to the severity of the outbreak in Haiti.

Internet Resources

The Merck Veterinary Manual
http://www.merckvetmanual.com/

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

OIE Manual of Diagnostic Tests and Vaccines for Terrestrial Animals [Note: Although teschovirus encephalomyelitis no longer appears on the list of diseases reportable to the OIE, information about this disease is still available in the manual].
http://www.oie.int/international-standard-setting/terrestrial-manual/access-online/

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