Network for wildlife health surveillance in Europe

Diagnosis Card

Aujeszky's Disease

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Etiology

Suid Herpesvirus 1 (SuHV-1). Double-stranded DNA virus of the genus *Varicellovirus,* subfamily: *Alphaherpesvirinae;* Family: Herpesviridae; Order: *Herpesvirales*. (synonym: pseudorabies virus: PrV; Aujeszky's disease virus: ADV).

Affected species (wildlife, domestic animals, humans)

Swine are the only natural host and reservoir for PrV, although it can infect other mammals including carnivores, ruminants, and rodents causing fatal disease. Reports of horses contracting PrV are very rare. Humans are resistant against natural PRV infection.

Epidemiological characteristics and disease course

PrV can be transmitted through secretions, excretions (saliva, nasal discharge), sexual encounters, aerosols and from eating contaminated feed/carcasses. Within wild/feral swine PrV appears to be preferentially transmitted by oro-nasal and venereal route. Incubation period in swine normally range between 1-8 days up to 3 weeks. Usually, after oro-nasal and venereal infection of the natural host and primary replication in epithelial cells of the upper respiratory and genital tract, respectively, the virus gains access to the olfactory, trigeminal and glossopharyngeal nerves. A hallmark of PrV is their capacity to persist for the lifetime in their host in a latent state. Trigeminal ganglia, sacral ganglia and tonsils are the most common sites of PrV latency. Whereas highly virulent PrV strains are predominantly neuroinvasive, strains of moderate or low virulence exhibit weak neuroinvasiveness, but distinct pneumotropism. Despite successful elimination of PrV from domestic pigs in several parts of the world including large regions in Western and Central Europe, PrV is widespread in populations of wild/feral swine across the world.

Clinical signs

Domestic swine: Presence and severity of clinical signs as well as morbidity and mortality vary depending on age, immunological status, route of infection, and virulence of the PrV strain. In fully susceptible swine PrV infection results in high morbidity and mortality, especially in juveniles which develop predominantly meningoencephalitis and viremia-associated signs.

Neonatal pigs (< 7 days): sudden death with few, if any, clinical signs.

Weaning and post-weaning pigs, (2 to 3 week old): severe signs of central nervous system affliction (shivering, incoordination, convulsion, tremor, ataxia, and paralysis) with mortality rates up to 100%.

3 to 20 weeks old: may still show neurological signs, but usually develop age-dependent resistance-reduced mortality of 50% up to 5% in 4 week and 5 months old pigs. Co-infections with other swine viruses often result in severe and fatal proliferative and necrotizing pneumonia. Generally, high fever is followed by anorexia, listlessness, excessive salivation, vomiting, coughing, sneezing, dyspnoea, and aspiration pneumonia, trembling and eventually marked incoordination (hind legs).

Adult swine: high morbidity due to predominantly respiratory signs. Clinical signs can be present for 6 to 10 days. Most animals recover within a few days but present with less weight. In finishing and fattening pigs, clinical signs can amplify and animals often die from secondary bacterial pneumonia. Signs in gilts and sows depend on phase of gestation and include embryonic death, resorption of foetuses, mummified foetuses, abortion, or stillbirth, in addition to respiratory signs and fever.

Wild/feral swine: clinical signs are rare indicating a high adaptation of prevailing PrV variants to the host population. Cases of spontaneous disease clinically and pathologically identical to AD in domestic pigs are rare.

Other mammals: peracute fatal course of disease with incubation periods of only 2 to 3 days is characteristic with predominantly progressive neurological signs. Often extreme pruritus which can result in severe automutilation, is the only clinical sign.

Gross lesions

No typical gross lesions, at least not in terms of being characteristic for the disease. Multifocal tissue necrosis, exudative kerato-conjunctivitis, serous to fibrinonecrotic rhinitis, necrotizing laryngotracheitis, bronchointerstitial pneumonia, necrotizing tonsillitis, and leptomeningeal hyperemia (CNS) may be present. Multiple small foci of acute hemorrhagic necrosis may be seen in organs and placenta. In aborted sows, necrotizing placentitis and endometritis are observed; aborted fetuses may be macerated or, occasionally, mummified (SMEDI). In fetuses or neonatal pigs, necrotic foci in liver and spleen, lungs and tonsils are common. In carnivores, parts of the body and particularly the upper extremities are often characterized by widespread skin eruption due to automutilation.

Histological lesions

Microscopic lesions reflect neuroinvasive and epitheliotropic properties of PrV. Evidence of non-specific histological lesions can be observed when brain tissues from diseased animals are examined microscopically: nonsuppurative meningoencephalomyelitis, ganglioneuritis of trigeminal and paravertebral ganglia, panencephalitis (piglets), encephalomyelitis with perivascular cuffing. In swine, other histological lesions may include epithelial lesions in parenchymatous organs; necrosis of bronchial, bronchiolar, and alveolar epithelium; multifocal to diffuse lymphohistiocytic endometritis and vaginitis; necrotic placentitis; degeneration of seminiferous tubules; necrotic foci in the tunica albuginea of testicles; spermatozoa abnormalities; necrosis in parenchymatous organs (aborted or stillborn piglets together). Additionally, presence of intranuclear eosinophilic inclusion bodies, which are more common in lesions outside the nervous system is considered characteristic for AD.

Differential diagnosis

Swine: Rabies (lyssavirus), porcine polioencephalomyelitis (teschovirus infection), classical (CSF – pestivirus) and African swine fever (ASF – Asfarvirus), swine influenza, encephalomyocarditis (EMC), infections with highly virulent strains of porcine reproductive and respiratory syndrome virus (PRRSV) and porcine circovirus type 2 (PCV 2), Japanese encephalitis, hemagglutinating encephalomyelitis, bacterial meningoencephalitis including *Streptococcus suis* infection, salt poisoning, hypoglycemia, organic arsenic or mercury poisoning, congenital tremor, other diseases causing abortion.

Other mammals: Rabies, scrapie (sheep), bovine spongiform encephalopathy (BSE) and diseases or conditions causing CNS symptoms e.g. persistent itching need to be excluded.

Criteria for diagnosis

Detection of viral antigens directly in infected organ tissue, or isolation of virus, detection of viral DNA (PCR, etc.), detection of gE (field strains specific) antibodies (latent infection)

Recommended diagnostic method(s) and preferred samples (incl. recommended amount and appropriate storage)

Post mortem diagnosis should be performed on fresh organ tissues, preferably from brain, nervous ganglia (trigeminal/sacral), tonsils, lungs, fetuses and/or placenta. Viral antigen can be detected using immunoperoxidase and/or immunofluorescence staining with polyclonal or monoclonal antibodies on cryosections of tissues. Diagnosis is confirmed by virus isolation in cell cultures. PCR (conventional, real-time) is the method of choice for detection of viral DNA. Indirect or competitive ELISAs, seroneutralisation (SNT), latex agglutination tests (LAT) and immunoblotting detect PrV specific antibodies. Rabies diagnostics should be performed in parallel for suspect specimens.

Shipment and sample storage: Specimens for diagnosis should be shipped refrigerated or frozen, (temperature: +4 °C or –20 °C) according to the national and international regulations for shipment of infectious substances to avoid exposure. For long-distance shipment of isolates or tissues, proper packing and freezing on dry ice or in liquid nitrogen is recommended. Upon arrival in the laboratory, specimens preferably should be stored refrigerated or frozen (-20°C) for a short period before testing.

APHAEA protocol (for harmonization at large scale)

ELISA (i.e. the most reliable, specific, sensitive, cheap and quick method to estimate the status of PrV infections in wild/feral swine populations, and applicable to poor quality and haemolysed sera).

Laboratories that can be contacted for diagnostic support

Institute of Molecular Biology, Friedrich-Loeffler Institute, Südufer 10, 17493 Greifswald - Insel Riems, Germany (http://www.fli.bund.de)

ANSES – OIE Reference Laboratory for Aujeszky's Disease Laboratoire de Ploufragan- Plouzané, Unité de Virologie et Immunologie Porcines BP 53 « Les Croix » 22440 PLOUFRAGAN (uvip@anses.fr)

Recommended literature

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