

AUJESKY'S DISEASE (PSEUDORABIES) IN THE HORSE

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Summary

Pseudorabies, caused by suid (porcine) herpesvirus-1, is an acute, contagious disease affecting primarily pigs, but transmission to other species including horses can occur. Pseudorabies has been reported around the world, although it has been eradicated from many countries, including the UK. The pig is the natural host of suid herpesvirus-1, and other species, including horses, are generally infected as a result of close contact with pigs (aerosol spread). Species other than pigs are considered to be dead-end hosts, and they invariably die as a result of acute neurological disease. Natural pseudorabies infections in horses have been rarely reported. Severe neurological signs develop over a period of 1–3 days, followed by death.

Introduction

Pseudorabies (also known as Aujeszky's disease, Herpesvirus suis disease, infectious bulbar paralysis, mad itch), caused by suid (porcine) herpesvirus-1, is an acute, contagious disease affecting primarily pigs, but transmission to other species including cattle, sheep, goats, dogs, cats and horses can occur. Since its recognition as a disease entity by Aujeszky in 1902, the disease has become of major economic importance. The name pseudorabies was given to the disease because of its similarity to bovine clinical rabies. Pseudorabies has been reported around the world, including Britain, Ireland, continental Europe, North Africa, Asia, South America, New Zealand and the USA, however since the 1980s the

disease has been eradicated from many countries. Infection results in neurological signs in piglets, respiratory disease and poor growth performance in fattening pigs, and reproductive failure in sows (Van Oirschot 2004). Older pigs usually survive the acute infection, but carry the virus in a latent form for their entire lives.

Aetiology

Suid herpesvirus-1 has the broadest range of the animal herpesviruses (Crandell 1985). Natural infections of pseudorabies have been observed in pigs, cattle, sheep, goats, horses, dogs, cats, rodents, mink and wild animals. Human infections have been reported, but currently pseudorabies is not considered to be a public health threat. Replication of the virus with cytopathological changes occurs in cell cultures derived from a wide variety of animal species. The virus survives 2–7 weeks in the environment, and up to 5 weeks in meat.

Epidemiology

The pig is the natural host of suid herpesvirus-1. Pigs generally become infected by the nasal route, and the virus is most commonly spread by aerosols. Infection is by consumption of contaminated food or milk, but spread via semen (especially by artificial insemination) and unwashed embryos is also possible. The brown rat may be important in disease transmission from farm to farm (Timoney *et al.* 1988). Other species, including horses are generally infected as a result of close contact with pigs (aerosol spread). Species other than pigs are considered to be dead-end hosts, and they invariably die as a result of acute neurological disease (Van Oirschot 2004).

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Pathogenesis and clinical signs

The respiratory tract is the natural route of infection in pigs. The primary site of viral replication is the nasopharyngeal region and respiratory tract. The virus enters the olfactory nerves and travels along the glossopharyngeal nerve to the medulla, or to the pons and medulla by way of the trigeminal nerve.

In a natural infection in pigs, the incubation period is 1 week and the disease lasts 2–8 days. Recovered pigs may become latent carriers that actively shed the virus during periods of stress. Infected pigs aged <1 month of age usually die of neurological disease, whereas growing and finishing pigs usually exhibit respiratory disease, and sows abort (Inch 1998). In suckling pigs, the morbidity is high, and mortality can reach 80–100%, but mortality falls to <5% in pigs aged >4 months. In cattle, intense pruritus of some portion of the skin is the principle manifestation ('mad itch'), and generally appears on one of the flanks or the hind legs (Timoney *et al.* 1988). If the part is accessible, the animal licks at it incessantly until the skin becomes abraded and reddened. Intense pruritus is also commonly seen in dogs and cats, in addition to bulbar and pharyngeal paralysis.

Horses do not appear to be particularly susceptible to natural infection with suid herpesvirus-1, and they have been reported to often remain unaffected on farms where other livestock, dogs and cats are dying of the disease (Crandwell 1985). Natural pseudorabies infections in horses have been rarely reported. Van den Ingh *et al.* (1990) described one case in a horse that was pastured next to a maize field that had been manured with pig slurry; a strong wind blew manure into the pasture. Another natural infection in a horse was reported by Kimman *et al.* (1991). This horse originated from a farm where breeding sows, fattening pigs, cattle, ponies and horses were housed in the same barn. In species other than the pig, including the horse, the incubation period following infection is up to one week. Severe neurological signs develop over a period of 1–3 days, followed by death. Clinical signs include anorexia, depression, fever, muscle tremors, hyperexcitability, chewing, hypersalivation, severe pruritus (resulting in self-mutilation), head pressing, nystagmus, iridocyclitis, blindness, ataxia, recumbency and paralysis.

Experimental infection of 2 ponies by instillation of virus into the conjunctiva and nostrils resulted

in the development of fever after 7 days (Kimman *et al.* 1991). They subsequently started to show behavioural changes, and developed severe neurological signs 9 days after infection. One pony became excited and one became depressed. One pony died on the ninth day after inoculation and the other was subjected to euthanasia on the tenth day. Both ponies had a significant increase in serum antibody titre against the virus.

Diagnosis

Analysis of cerebrospinal fluid reveals nonsuppurative meningitis. *Post mortem* examination shows nonsuppurative meningoencephalitis with perivascular cuffing (**Fig 1**), focal and diffuse gliosis, neuronal necrosis and focal malacia. Intranuclear inclusion bodies may be seen in intact and necrotic neurons and swollen astrocytes. A definitive diagnosis is made by virus isolation or the demonstration of suid herpesvirus-1 antigen in neurons by immunohistochemistry or the fluorescent antibody technique. A variety of serological assays can be used to demonstrate specific suid herpesvirus-1 antibody, including serum neutralisation, agar-gel immunodiffusion test, microimmunodiffusion test, enzyme-linked immunosorbent assay, indirect solid-phase microradioimmunoassay, modified direct complement-fixation test, countercurrent immunoelectrophoresis and the indirect haemagglutination test.

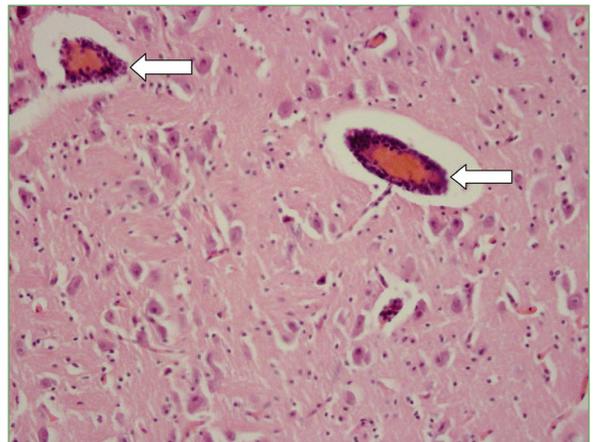


FIGURE 1: Photomicrograph of pig brain infected by Aujeszky's disease. Perivascular cuffing (arrows). Haematoxylin and eosin.

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