Clinical Signs of Pseudorabies in the Dog and Cat: A Review of 40 Cases

Barbara A. Hawkins, BS, DVM*
Greg R. Olson, MS, DVM, PhD**

INTRODUCTION

Pseudorabies (Aujeszky's disease, "mad itch", infectious bulbar paralysis) has been present in the United States since 1813.[1] In 1902, Aladar Aujeszky in Hungary determined that the disease was of non-bacterial origin.[2] Aujeszky investigated two fatal cases of pseudorabies (PR), one affecting a bull and another a dog, and differentiated this syndrome from rabies.[3,4,5] Although PR is most prevalent in swine in the United States, it is a devastating disease in cattle, sheep, goats, dogs and cats. Infection in highly susceptible domestic animals results in rapid death, but these dead-end hosts contribute little to the spread of PR. Adult swine are more resistant to pseudorabies virus (PRV) infection and can develop latent infections, serving as natural reservoirs for the virus. This condition allows for the survival and spread of the virus not only to swine but to other domestic animals as well.

Herpesvirus suis, the etiologic agent of PR, may survive in an infected environment for two to seven weeks.[6] The virus may survive on hay for up to forty-six days during the winter.[2] It is relatively resistant to changes in pH and may remain viable for many years in tissues at low temperatures. The virus is rapidly inactivated by one-half percent sodium hydroxide and formalin but may be resistant to three percent phenol and cresolic disinfectants.[2,6]

PRV infection in swine is manifested by a variety of clinical syndromes. The viral strain, route of exposure, and the animal's immune status all contribute to the clinical picture, but the primary factor is the age at which the animal becomes infected.

Suckling piglets are more susceptible than older pigs with mortality rates often approaching one hundred percent. Sudden death may be the only apparent finding in this age group with PR, while others may be febrile, anorectic, and incoordinated with muscle tremors, vomiting, and diarrhea prior to death.[6,7]

Growing and finishing pigs are more resistant to PRV infections and therefore suffer fewer death losses. Clinical signs in this age group include fever, anorexia, depression, vomition, and respiratory signs with sneezing, coughing, nasal discharge, and labored breathing.[6,7]

In adult swine, the disease is relatively mild. Some mature pigs show no clinical signs or experience only mild appetite depression. Respiratory distress is more characteristic in this age group although some may develop a CNS syndrome, depending on the viral strain causing the infection.[6,7] Sows infected while pregnant can experience early embryonic deaths, expulsion of mummified feti, abortions, or birth of live, weak pigs depending upon when in gestation the sow was exposed to the PRV.[6,7]

It is important for the veterinarian to be aware of PR in dogs and cats for the following reasons: (1) Iowa and other midwestern states, being large swine-producing areas, are experiencing an increase in the incidence of PR not only in swine herds but also in other farm animals, including dogs and cats; (2) the clinical disease in dogs and cats is very short (death usually ensues in these species often before PR appears clinically in the swine herd), therefore, a diagnosis of PR in these species may aid the pork producer in control-

*Dr. Hawkins is a 1985 graduate of the College of Veterinary Medicine at Iowa State University.
**Dr. Olson, a former Iowa State University Instructor in the Department of Veterinary Pathology, is presently a Veterinary Pathologist at Pathology Associates, Inc. in Cincinnati, Ohio.

ling the disease in the swine herd; (3) the clinical signs that are noted in dogs and cats with PR can be misleading to the practitioner resulting in a misdiagnosis of rabies, distemper, or insecticide/herbicide toxicosis if a diagnostic laboratory is not consulted.

CLINICAL DATA

Forty PR infected dogs and cats admitted to the Iowa State University Veterinary Teaching Hospital from 1975 through 1982 are summarized in this paper. The majority of these cases were diagnosed positive for PR by the Iowa State University Veterinary Diagnostic Laboratory using either the Fluorescent Antibody technique or virus isolation from submitted brain tissues.

HISTORY: According to the obtainable histories of these cases, the majority of the dogs and cats that were infected with PRV lived on or near farms housing swine. In almost all instances, PR was not diagnosed in the swine herd until after the dog(s) and/or cat(s) had died from the disease. However, one cat with PR was presented with a history of living on a farm where four feedlot heifers had died after showing signs of profuse salivation, pruritus, and self-mutilation. Another case involved a dog dying of PR that had contact with two of the neighbors' calves that had died of a "neurological disorder." Consumption of contaminated swine and cattle tissues or uncooked offal by dogs and cats is usually the route of exposure.[8,9,10] No consistencies were noted among the signalments of these forty animals reviewed.

CLINICAL SIGNS: Twelve of the forty animals, two cats and ten dogs, died suddenly without clinical signs. Of the remaining twenty-eight dogs and cats, some of the clinical signs observed included: increased salivation, depression and lethargy, rapid, labored breathing, fever, vomiting, convulsions, reluctance to move, recumbency, and intense pruritus. Table 1 summarizes these clinical observations and the frequencies in which these signs occurred.

NECROPSY FINDINGS: Seventeen of the forty dogs and cats which died from PR were necropsied. The other twenty-three animals were either too autolytic for lesion identification or were unavailable for necropsy. Consistent postmortem lesions were noted in the gastrointenstinal tract, heart, and lungs.

Twelve of the seventeen animals necropsied had lesions involving the gastrointestinal tract. Multifocal to diffuse petechial hemorrhages were noted in the esophagus, stomach, and small intestine. Frank and digested blood was present in at least one section of the gastrointestinal tract in a majority of the animals. Ulcers were noted on the soft palate of one dog and on the duodenum of another dog examined. In two other animals, the liver was congested and slightly enlarged. One dog's liver had several 1–2 mm pale yellow, depressed foci scattered over its surface.

Along with the gastrointestinal lesions, the heart was frequently affected grossly by PRV. Several petechial and ecchymotic hemorrhages were noticed on the endocardium and epicardium of four animals infected. The heart in another dog was enlarged and flabby.

Four of the animals examined displayed diffusely congested lungs. All but one of these animals had gastrointestinal lesions in addition to the abnormalities noted in the lungs. Edema was present in the congested lungs from one of the PR infected dogs.

TABLE 1

Clinical Sign/ Physical Abnormality	Frequence of Sign Observed	Percentage
Increased salivation	17/28	60.7
Depression and lethargy	12/28	42.8
Dyspnea	8/28	26.8
Vomiting	7/28	25.0
Convulsions	7/28	25.0
Recumbency	5/28	17.8
Unwillingness to walk but not recumbent	5/28	17.8
Intense pruritus	5/28	17.8
Other CNS signs (headpressing, circling)	5/28	17.8
Incoordination and ataxia	4/28	14.3
Hyperesthesia	4/28	14.3
Dehydration	4/28	14.3
Irregular pulse	3/28	10.7
Muscle spasms and trembling	3/28	10.7
Unresponsive pupils	3/28	10.7
Nystagmus	3/28	10.7
Anorexia	3/28	10.7
Diarrhea	2/28	7.1
Flaccid anus	2/28	7.1
Congested mucous membranes	2/28	7.1
Personality changes	2/28	7.1

*Death resulted in all cases within 8–120 hours after onset of clinical signs.

Other sporadic gross pathological findings included: enlarged protruding tonsils, congested spleen, clotted and unclotted blood in both pleural and peritoneal cavities, and congested meninges.

DIFFERENTIAL DIAGNOSES: Of the forty cases reviewed, rabies, distemper, lead poisoning, organophosphate, strychnine, and inorganic arsenic toxicoses were included as differential diagnoses by the submitting veterinarians. Table 2 compares the prominent clinical signs of these conditions with those of PR.

CONCLUSION

To aid the veterinarian in a diagnosis of PR in dogs and cats, it is important to obtain a complete history including the animal's environment, i.e. whether or not it lives on or near a swine farm, possible encounters with wild animals such as skunks or racoons, roaming habits, or exposure to herbicides, insecticides, or other chemicals used on the premises.

Due to the nonspecific clinical signs associated with PR infected dogs and cats, this disease is often overlooked. Many practitioners associate PR with the "mad itch" syndrome. However, because intense pruritus was reported in only 17.8 percent of the clinically affected dogs and cats admitted to the Iowa State University Veterinary Teaching Hospital, the absence of this clinical sign cannot be used to rule out PR.

It is not surprising that PR in dogs and cats can be misinterpreted as other infectious diseases or toxicoses due to the ambiguity of clinical signs associated with PRV infection. However, the conscientious veterinarian should be aware of the clinical appearance of PR in dogs and cats. A tentative diagnosis of PR in a dog or cat can aid the practitioner in advising the swine producer of an impending PR outbreak in his swine herd.

TABLE 2

Signs Observed In Dogs and Cats With PR	Clinical Signs Observed With: Distemper[11]	Rabies[11]	Lead[12]	Organo-Phosphate[12]	Strychnine[12]	Inorganic Arsenic[12]
Increased salivation		+		+		+
Depression and lethargy	+		+	+		+
Dyspnea	+			+	+	
Vomiting	+		+	+		+
Convulsions	+	+	+	+	+	
Recumbency		+				
Unwillingness to walk but not recumbent		+				
Intense pruritus						
Other CNS signs	+		+			
Incoordination and ataxia	+	+	+	+	+	+
Hyperesthesia			+		+	
Dehydration		+				+
Irregular pulse						+
Muscle spasms and trembling			+	+	+	+
Unresponsive pupils		+		+	+	
Nystagmus						
Anorexia	+	+	+			+
Diarrhea	+		+	+		+
Flaccid anus						
Congested mucous membranes						+
Personality changes		+				
Sudden death					+	

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