Encephalitis in two porcupines due to
Baylisascaris larval migration

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Encephalitis is associated with the migration of *Baylisascaris* sp. larvae in a number of animal species, including mice, squirrels, woodchucks, rabbits, foxes, various birds, and humans. The species of *Baylisascaris* most often incriminated has been *B. procyonis* from raccoons. In a recent report of *Baylisascaris* sp. infection in American porcupines (*Erethizon dorsatum*) with concurrent *Toxoplasma* infection, the relative contribution of the 2 agents to the clinical problem could not be determined. In the current report, we describe the clinical signs, microscopic lesions, and epidemiologic investigation of *Baylisascaris* sp. infection causing progressive central nervous system (CNS) disease in 2 captive American porcupines. One porcupine also had bronchointerstitial pneumonia due to a virus that had not been previously reported in this species.

An immature male porcupine developed ataxia, somnolence, rear limb paresis, and head tilt. Antibiotic therapy was initiated, but the animal died several weeks later, following a protracted course of progressive neurologic dysfunction. No gross lesions were observed at necropsy. Formalin-fixed tissues were submitted to the Animal Disease Diagnostic Laboratory, Purdue University, for microscopic evaluation. All sections of CNS tissue examined, including the cerebrum, cerebellum and brainstem, had multifocal malacia and encephalitis. The inflammatory infiltrate consisted predominantly of gemistocytic astrocytes, with scattered neutrophils, eosinophils, and rare multinucleated giant cells. The infiltrate was predominantly perivascular, including meningeal and submeningeal vessels of the cerebellum.

The lung had a diffuse bronchointerstitial pneumonia characterized by large numbers of neutrophils filling bronchi, bronchioles, and alveolar spaces. Additionally, proteinaceous fluid filled many alveolar spaces, and dissolution of the alveolar walls was commonly observed (Fig. 1). Many alveolar epithelial cells contained large basophilic intranuclear clearances (Fig. 2). Electron microscopic examination of lung tissue revealed numerous viral particles within the cytoplasm and nucleus of degenerate alveolar epithelial cells (Fig. 3). These viral particles wereicosahedral and were approximately 30 nm in diameter. Based upon the morphology and size, these virus particles were presumed to be either a parvovirus or a picornavirus. Fresh lung tissues were not available for virus isolation. There has been 1 report of suspected parvovirus in porcupines; however, this was associated with necrotizing enteritis, and virions were not identified within inclusions.

The liver was the only other organ examined that had morphologic lesions. Centrilobular hepatocytes had vacuolar change consistent with fatty degeneration, most likely secondary to the long-standing inappetance of the animal.

Six weeks later, a second immature porcupine that had been housed in the same cage as the first animal developed ataxia that progressed to recumbency. This porcupine had progressive neurologic disease despite antibacterial and corticosteroid therapy. The porcupine was euthanized and necropsied, and formalin-fixed tissues were submitted to the Animal Disease Diagnostic Laboratory. Microscopic lesions within the brain were limited to the cerebral peduncles and caudal brain stem bordering the fourth ventricle. Affected CNS tissue was characterized by multifocal areas of malacia, gliosis, and infiltration by numerous neutrophils forming microabscesses admixed with lesser numbers of eosinophils and gemistocytic astrocytes (Fig. 4). Lymphocytic perivascular cuffing, including meningeal and submeningeal vessels, was also a prominent feature of the inflammatory reaction. Rare cross sections of nematode larvae were present within inflamed and malacic areas, as well as within otherwise normal neuropil. These larvae were 60-66 pm in greatest diameter, with prominent single lateral alae and a large centrally located intestine flanked by smaller triangle-shaped lateral excretory columns (Fig. 5). Both the size and morphology of the larvae were consistent with *Baylisascaris* sp. This porcupine also had a chronic diffuse interstitial pneumonia, characterized by thickening of the alveolar septa by histiocytic infiltration and prominent type II pneumocyte hyperplasia.

Based on the microscopic identification of *Baylisascaris* larvae in the second porcupine brain, additional sections of the brain from the first animal were examined. A single nematode larva, with similar morphology, was identified within the brain stem.

An on-site investigation of the central Indiana farm was conducted to determine the source and species of the *Baylisascaris* larvae involved. The premises had, at one time, housed 5 porcupines that were originally obtained in Michigan. One adult male was caged separately indoors and had been in residence for 3 years. During the spring of 1989, 4 additional porcupines were added; 1 adult female and 1 juvenile were purchased, and 2 juveniles were wild-caught from separate localities. The 2 purchased porcupines were kept in an 8- x 8- x 10-ft wire outdoor enclosure. The 2 wild-caught juveniles were kept outdoors in a 3- x 3- x 4-ft raised wire cage.

During the summer of 1989, 1 raccoon was purchased locally and kept on the premises in a large raised wire cage for 5 days. The raccoon was intractable but had no signs of illness. The raccoon was sold, and the 2 wild-caught juvenile porcupines were placed into the raccoon’s cage, from which they were periodically removed and given access to the ground surrounding the cage and to an open area in the yard. Two months later, the first wild-caught juvenile porcupine developed CNS signs. Six weeks after that, the second porcupine exhibited CNS signs.
Multiple superficial soil samples were collected from under and around the raccoon cage, from within the large enclosure, and from the open area of the yard. These samples were examined for parasite eggs using a centrifugal flotation method. Embryonated *Baylisascaris* eggs were found in 4 of 10 soil samples and were most numerous directly under and around the raccoon cage; no eggs were found in the enclosure or in the open area.

The raccoon was therefore the source of *Baylisascaris procyonis* eggs that led to infection of the 2 wild-caught porcupines. Whether the porcupines were infected with eggs from the cage or the ground is unknown. However, the ground was the more likely source of the infection because it was where most of the raccoon feces accumulated, and the young porcupines ate grass and twigs from the contaminated area. This relatively minor contamination resulted in an ongoing clinical problem. However, large numbers of *B. procyonis* eggs could have been present because infected raccoons shed an average of 20,000 eggs/g of feces and a maximum of 250,000 eggs/g of feces. Embryonated *B. procyonis* eggs can...
remains viable in the soil for years if adequate moisture is present. 7,8 The delay of several months in the onset of CNS disease is consistent with the 3-4 week period necessary for Baylisascaris eggs to become infective, and the time necessary for infection, larval migration from the intestine to the CNS, and the initiation of CNS damage. Depending on the number of eggs ingested, animals may develop CNS disease 2-4 weeks or more after infection. 8 The 6-8 month period of captivity before clinical signs developed in the 2 porcupines makes their acquisition of the infection from the wild unlikely.

The exact cause of the pulmonary disease in the 2 porcupines is unknown. The virus particles described have not been previously reported in association with pneumonia in porcupines. The viral infection in the first porcupine may have predisposed the animal to secondary bacterial bronchopneumonia. Whether or not both porcupines were affected by the same viral and bacterial agents was not deter-

Figure 4. Photomicrograph of the brain of a porcupine. Microabscess contains necrotic debris, neutrophils, eosinophils, and 3 cross sections of nematode larvae (arrows). HE stain.

Figure 5. Higher magnification of Fig. 4. Note prominent lateral alae (arrows) and triangular excretory columns (E) on both sides of the intestines, characteristic of Baylisascaris sp. HE stain.
The incidence of congenital diaphragmatic hernias in swine was not considered to be the result of Baylisascaris larval migration in either animal.

The owner was advised of the Zoonotic potential of this parasite and proper disinfection methods for the premises and affected cages; disinfection is best done using some form of incineration to destroy the eggs. Furthermore, the owner was advised not to keep any more raccoons or skunks on the premises, because either could reintroduce the problem. To date, the remaining 3 porcupines on the premises and the 6 rabbits housed in an elevated outdoor wire pen have remained healthy and serve as sentinels for environmental contamination by Baylisascaris eggs.

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References


Epizootics of diaphragmatic hernias in swine

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Diaphragmatic hernias in swine occur sporadically, often in a single animal, and are generally assumed to be congenital or the result of traumatic accidents. In 1 survey, the incidence of congenital diaphragmatic hernias in swine was 0.02%. Evidence for hereditary predisposition to diaphragmatic hernias in swine is lacking.

Swine practitioners and veterinary diagnosticians are occasionally confronted with epizootics of fatal diaphragmatic hernias in growing swine. The cause of these epizootics is neither known nor easily studied. An excellent clinical description of an epizootic in Ohio remains the only thorough report of this condition although an anecdotal report of an outbreak in Kansas is also of interest?

Four Iowa swine herds that experienced an acute onset of fatal diaphragmatic hernias in a large number of growing pigs were investigated in 1990. In all 4 epizootics, mortality due to diaphragmatic hernias occurred in weaned swine between 3 and 16 weeks of age. No fatal diaphragmatic hernias were observed in the offspring of previous identical matings of sires and dams in any of the 4 herds.

In herd A, 4 sows farrowed 3 weeks later than did the other 18 in the group. The 36 pigs from these later litters were segregated until they were commingled with older pigs when weaned at 4 weeks of age. Twenty-six of 36 pigs subsequently died between 5 and 8 weeks of age. Necropsies performed on 7 of the animals confirmed the presence of diaphragmatic hernia with no other disease processes evident grossly. The other 19 animals were not necropsied, but deaths were attributed to diaphragmatic hernias by the attending veterinarian or the producer, based on clinical appearance or sudden deaths.

In herd B, 240 8-week-old pigs were delivered to 2 different sites for growing and finishing. The pigs that were delivered to site 1 were isolated from other swine, and no deaths due to diaphragmatic hernias were reported. At the second site, 47 of 122 pigs died between 10 and 16 weeks of age. All were found either dead or moribund, exhibiting respiratory distress. All of 15 pigs that were necropsied on the premises by the attending veterinarian had diaphragmatic hernias, with no other gross lesions deemed significant. The feed and its source, the environment, and the management were similar at both sites. There was direct contact between affected and older unaffected pigs at site 2, but no contact between these groups at site 1.

Herds C and D were both on small farms. On both farms,