Baylisascaris procyonis is an ascaridoid parasite of the raccoon that is related to Toxocara canis, the canine roundworm. Other species of Baylisascaris occur in different wildlife species; the more common species include Baylisascaris melis of European badgers, Baylisascaris transfuga of bears, and Baylisascaris columnaris of skunks and American badgers, Baylisascaris devosi of fishers and martins, and Baylisascaris laevis of marmots. Due to the domestic nature of the raccoon and the damage that the larvae Baylisascaris procyonis induces in hosts other than the raccoon, this species is the most important of the genus relative to its potential to cause zoonotic disease.

Baylisascaris procyonis is commonly found in raccoons in the fall of the year. The worms are relatively common throughout the range of this host, although it appears to be relatively uncommon in raccoons living in the coastal area of Louisiana. The worms are common, however, in coastal Texas [1]. When surveys are performed it is important to bear in mind that the prevalence of infection may be higher in the autumn than during other times of the year [2]. The range of Baylisascaris procyonis has been dramatically expanded in recent years by the exportation of raccoons and this parasite to Europe. Recent surveys in Germany have revealed that around 70% of raccoons were infected with this worm [3]. Prior to the introduction of the raccoon to Europe, this host species was limited to North America. Overstreet [4] described a Baylisascaris procyonis from a kinkajou (Potos flavus) in Colombia, but there have been no additional reports from Mexico, Central, or South America of the finding of this parasite in other members of the Procyonidae.

The adult worms are larger than Toxocara canis and live within the small intestine of the raccoon (Fig. 1). Eggs are shed in the feces. The eggs are similar to those of other ascaridoids of terrestrial mammals in that they have a thick shell and are passed in the feces in a state containing a single cell (Fig. 2). After the eggs are deposited on the soil, they take a week or two under optimal environmental conditions of temperature, moisture, and aeration to embryonate to the infective stage. Raccoons become infected through either the ingestion of embryonated eggs or via the ingestion of paratenic hosts that have ingested the egg.
The paratenic hosts often show significant signs of disease due to the larva that grows during its migration. The larvae often make their way to the central nervous system where they cause significant neurologic signs. Natural infections with this parasite have been recorded in gallinaceous birds, ratites, various rodents, lagomorphs, nutria, sea otters, woodchucks, armadillos, porcupines, primates, and other hosts. The larva can be easily recognized in histopathologic section by its distinctive morphology [5] (Fig. 3).

Humans have also been infected with this parasite causing severe disease manifestations and sometimes death. Visceral larval migrans has resulted in several deaths [6; 7; 8]. Not all visceral infections with accompanying meningitis have led to a fatal outcome [9]; although the disease caused serious disease and permanent disability. Other outcomes include the loss of vision when the worms migrate into the retina. This has occurred in the United States and in Germany [10; 11].

It seems that dogs can act as either adult or paratenic hosts. There have been two reports of puppies, one a 12-week-old beagle [12] and one a 10-week-old Walker hound [13] that developed neurologic disease. When the beagle presented, it circled to the left, had hind-limb paresis, and was lethargic, blind, and ataxic. The Walker hound had an acute onset of ataxia that progressed to recumbency within 48 hours. The dogs were euthanized and necropsied, and both were found to have nematodes in their central nervous systems that were diagnosed as being the larvae of Baylisascaris procyonis. In both of these cases, the puppies had been housed in areas where a caged raccoon had been maintained.

Dogs can also serve as the hosts for the adult stage of this parasite. This was first reported in dogs from Iowa by Greve and O'Brien [14]. Since this first report, additional dogs have been reported infected with adult worms in Minnesota [15] and Prince Edward Island [16]. Additional dogs have been found infected in Indiana and Michigan. In all these cases, diagnosis has been made by the discovery of the distinctive eggs in the feces (Fig. 4 and 5). These dogs are not just ingesting fresh raccoon feces containing unembryonated eggs. Necropsies or treatment and examination of the feces of the animals for the presence of worms has revealed the presence of the adult worms in the intestinal tract.

Treatment of the adult worms in dogs is probably relatively easy. Conboy [16] treated a 5-month-old Dalmation puppy with a patent infection using piperazine at 220 mg/kg body weight. German workers [17] treated naturally infected raccoons and found that following oral administration of the drugs that all raccoons could be cleared with pyrantel emboate at 20 mg/kg, ivermectin at 1 mg/kg, moxidectin at 1 mg/kg given only one time or by a 3 day course of treatment with albendazole or fenbendazole at 50 mg/kg body weight or flubendazole at 20 mg/kg body weight. Hill et al. [18] tried to treat naturally infected raccoons with intramuscular ivermectin at 2 mg/kg body weight, and one of the ten treated raccoons continued to shed eggs in its feces.

Treatment of larval disease is much more difficult. In the child that survived the infection [9], therapy consisted of a seek of thiabendazole (50 mg/kg/day) and prednisone (2 mg/kg/day). There was no improve in
the neurologic status at this time so treatment was followed with a single dose of ivermectin (175 µg/kg). The patient left the hospital after a two-week stay on an oral regimen of prednisone that was gradually tapered. Two years after diagnosis, the patient still had severe developmental delay, cortical blindness, and hemiparesis.

The major concern with this parasite is that it is moving into the canine population. Three have been more than two-dozen dogs diagnosed with patent infections. Dogs, unlike raccoon, are indiscriminant fecal shedders. Raccoons are dangerous because they tend to use "latrines," where they routinely defecate. Thus, large numbers of eggs can build up in a small space. Dogs, on the other hand, tend to often defecate wherever, and as is verified by the percentage of people that develop antibodies to *Toxocara canis*, the eggs do make their way into people because of their close association with humans. People tend to never know that they are hosts to *Toxocara canis*. The larvae of *Baylisascaris* grow after they are ingested and are very capable of causing disease. Thus, there is every reason for significant concern relative to the threat posed by dogs perhaps having patent infections.

Dogs should have regular fecal examinations if their owners do not have them on a monthly product that also treats intestinal roundworm infections. It is highly likely that products that prevent heartworm and treat canine roundworms, are capable of clearing dogs of their infections with this parasite. In fact, personal experience with dogs infected with *Baylisascaris procyonis* would indicate that this is the outcome. Thus, this is another good reason to have dogs on monthly preventative treatment year-round. In areas where there is only minimal heartworm transmission or none, veterinarians need to consider this as a reason to maintain vigilance in their fecal diagnostic work ups or to suggest that dogs be placed on preventative therapy as for heartworm. It should be remembered that although raccoons are not domestic animals, they thrive in locations near humans. The populations in cities can be very high and people are often now aware of how many raccoons there actually are in a given neighborhood. Although they are cute, it should not be forgotten that many of them will be infected with this parasite.

References


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