Baylisascariasis

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Importance

Baylisascariasis is a parasitic disease of mammals and birds that can be caused by at least 11 species of nematodes in the genus *Baylisascaris.* The adult worms reside in the intestines of definitive hosts and tend to be carried without clinical signs unless they occur in large numbers. However, their larvae can infect a wide variety of hosts and may cause disease as they migrate through tissues, especially when they invade the eye or brain. *Baylisascaris procyonis*, an intestinal nematode of raccoons (*Procyon lotor*), has been responsible for a small number of clinical cases in humans. While these cases seem to be rare, most were serious, and survivors of neural larva migrans often had permanent damage. Other species of *Baylisascaris* may vary in their impact; however, *B. schroederi* is considered to be the most important parasite of giant pandas (*Ailuropoda melanoleuca*), its definitive host, with both adult nematodes and larvae causing illnesses and deaths in this species.

Etiology

Baylisascariasis is caused by intestinal nematodes (family Ascarididae) in the genus *Baylisascaris*. Most research has been done on *Baylisascaris procyonis*, a raccoon parasite that is known to affect humans. This organism is thought to be particularly virulent for paratenic hosts, as its larvae migrate extensively, continue to grow considerably within these hosts, and occasionally invade the CNS or eye. Other species of *Baylisascaris*, including *B. melis, B. columnaris*, *B. potosis*, *B. transfuga*, *B. devosi*, *B. schroederi*, *B. tasmaniensis, B. ailuri, B. laevis* and *B. venezuelensis,* can also cause disease. The various organisms differ in the size of their larvae and extent of larval migration, influencing the likelihood that they will cause clinical signs.

Species Affected

Definitive hosts

Baylisascaris spp. mature to adults in the intestines of one or more definitive hosts. Raccoons are the usual definitive host for *B. procyonis*. This organism can also mature sometimes in dogs. Surveys have not found *B. procyonis* eggs in any wild canids, as of 2020, though one wolf had DNA in its feces. In 2011, a report from the U.S. described this organism in kinkajous (*Potos flavus*); however, those worms might have been *B. potosis,* a related parasite of kinkajous that was discovered a few years later.

B. columnaris uses skunks, especially the striped skunk (*Mephitis mephiti*s), as its definitive hosts, and *B. devosi* occurs in various mustelids (e.g., fishers, martins). European badgers (*Meles meles*) are definitive hosts for *B. melis*. The organism in North American badgers (*Taxidea taxus*) may also be *B. meles,* though it was reported as *B. columnaris* in the past, and *B. devosi* might also be a possibility. *B. transfuga* is found in most species of bears; however, South American spectacled bears (*Tremarctos ornatus*) carry *B. venezuelensis. B. laevis* is a parasite of rodents, especially groundhogs/ woodchucks (*Marmota monax*) but also various ground squirrels, marmots and other species. Other definitive hosts include kinkajous for *B. potosis,* Tasmanian devils (*Sarcophilus harrisii*) and quolls (*Dasyurus* spp.) for *B. tasmaniensis,* giant pandas for *B. schroederi* and red pandas (*Ailurus fulgens*) for *B. ailuri. Baylisascaris* spp. eggs were recently found in the feces of a wild Virginia opossum (*Didelphis virginiana*) but the species was not identified.

Paratenic hosts

More than 130 mammals and marsupials are known to act as paratenic, intermediate or dead-end hosts for *B. procyonis*. While most natural or experimental infections have been described in rodents (mice, guinea pigs, prairie dogs and other species), lagomorphs and nonhuman primates, symptomatic cases have also been seen in dogs (which can act as both definitive and paratenic hosts for this organism), foxes and other canids; various Australian marsupials; opossums, porcupines, American badgers, groundhogs, sea otters and fruit bats. *Baylisascaris* larva migrans is not reported to be an issue in livestock, and experimental infections with *B. procyonis* in sheep, pigs or goats resulted in little migration of the larvae. However, one case of larva migrans, possibly caused by *B. procyonis*, occurred in a newborn lamb infected before

birth. *B. procyonis* is also known to affect at least 40 birds including poultry (chickens), game birds (bobwhite quail, commercial pheasants), various psittacines and other avian species.

Most other species of *Baylisascaris* have been confirmed to migrate in at least a few experimentally infected mammals (e.g., laboratory rodents) and/ or birds, but some organisms (e.g., *B. potosis, B. transfuga*) are less likely to cause clinical signs than *B. procyonis*. There are relatively few reports of naturally acquired clinical cases. Some were thought to be caused by *B. columnaris* or *B. melis*, whose larvae are similar to *B. procyonis*. *B. transfuga* was thought to be responsible for neural larva migrans in Japanese macaques at one zoo. It was also found in two clinically affected, wild, newborn moose calves, as well as in apparently healthy wild rodents in Russia. *Baylisascaris* infections have not been reported in poikilotherms.

Zoonotic potential

B. procyonis is the only species that has been reported in humans as of 2021; however, it is possible that other *Baylisascaris* species also affect people.

Geographic Distribution

Raccoons, the definitive hosts for *B. procyonis*, are native to the Americas, from Canada to Panama. Infected raccoons have been found throughout the U.S. but seem to be particularly common in the Mid-Atlantic, Northeast and Midwest and along the Pacific coast. Raccoons were introduced into Europe, the former U.S.S.R. and Asia for the commercial fur trade, and into Japan as pets, and they have become naturalized in some of these areas. The distribution of infected raccoons is not always clear in these regions; however, *B. procyonis* is known to be maintained in wild raccoons in Europe, and some captive (zoo and/or pet) raccoons are infected in Japan and China.

There is less information about other species of *Baylisascaris*, but they probably occur in most areas where their definitive hosts can be found. *B. devosi* and *B. transfuga* infect multiple species and appear to be widespread. As of 2021, both organisms have been reported in North America, Europe and parts of Asia. *B. devosi* is also known to occur in the Middle East (Iran). *B. columnaris* has been reported in North America (and in pet skunks in Europe), while *B. melis* has been documented in Europe, and might also infect badgers in North America. *B. potosis* was identified in captive kinkajous in Japan; however, kinkajous are native from Mexico to Brazil, and the infected animals appear to have originated in Guyana. *B. schroederi* and *B. ailuri* are limited to parts of Asia where pandas can be found, and *B. tasmaniensis* occurs only in Australia. *B. venezuelensis* was found in South America, and *B. laevis* in North America. Parasites may also occur outside these areas in captive wildlife.

Transmission and Life Cycle

Mature *Baylisascaris* nematodes reside in the intestines of their definitive host(s) and release unembryonated eggs in the feces. *B. procyonis* can produce very large numbers of eggs: each worm is estimated to lay up to 179,000 eggs per day, and raccoons carry an average of about 40-50 worms. The worm burden seems to be higher in juvenile raccoons than adults. In most environments, *B. procyonis* eggs take 2-4 weeks or more to develop to the infective stage, which contains second stage larvae, though embryonation can occur more rapidly (11-14 days) under ideal conditions in the laboratory. Although they can be killed eventually by extreme heat and desiccation, *Baylisascaris* eggs are resistant to inactivation, survive harsh winters and may remain viable for years. In some types of soil, they may remain near the surface during this time. They adhere readily to fur and various fomites.

Embryonated *Baylisascaris* eggs can complete their life cycle immediately if they are eaten by a definitive host. In raccoons, *B. procyonis* larvae hatch in the intestines, then develop for a time in the intestinal wall before maturing to adult worms in the lumen. Extraintestinal migration does not seem to occur frequently in raccoons, though larvae have occasionally been found in tissues. However, *B. procyonis* larvae can migrate more extensively in dogs, and some other *Baylisascaris* species undergo extraintestinal migration in their usual definitive hosts.

Baylisascaris eggs can also hatch if they are ingested by mammals or birds other than the definitive host. These hosts are generally called paratenic (transport) hosts, as they are not essential for the completion of the parasite's life cycle. However, rodents and some other animals can also be considered intermediate hosts for *B. procyonis*, as the larvae mature from second to third stage larvae. In a paratenic/ intermediate host, *B. procyonis* eggs penetrate the intestinal wall and are carried via the bloodstream through the liver to the lungs. From here, they can re-enter the circulation and may be distributed to a variety of organs/ tissues, where they can migrate for a time. Eventually they encyst, most often in connective tissue and muscles. Other species of *Baylisascaris* undergo a similar migration, though its extent may vary.

Paratenic/intermediate hosts can transmit *Baylisascaris* to definitive hosts only if their tissues are eaten. Larvae have been reported to survive in the tissues of a dead host for several days. When a raccoon ingests encysted *B. procyonis* larvae from a paratenic host, these larvae immediately mature into adult worms in the intestine. As a result, the prepatent period is shorter in animals that eat paratenic hosts (e.g., 32-38 days) than eggs (50-76 days). Paratenic hosts play no significant role in the life cycles of parasites that have herbivores as their definitive hosts (*B. laevis*, *B. schroederi* and *B. ailuri*).

Transplacental infection has been seen in paratenic hosts, including two newborn sibling moose calves infected with *B. transfuga*, and a newborn lamb thought to be

infected with *B. procyonis* or a morphologically similar organism (e.g., *B. melis* or *B. columnaris*). There was no evidence of intrauterine transmission of *B. procyonis* to newborn raccoons in one study.

Disinfection

B. procyonis eggs are highly resistant to disinfectants and can become embryonated even in a weak formalin solution. Desiccation and heat, such as strong sunlight on an exposed surface or heat in a dry attic, will eventually destroy the eggs, but it is not known how long this will take. One group found that, under experimental conditions, *B. procyonis* eggs were no longer viable if they were heated to 62°C (144 °F) or desiccated for 7 months. However, the eggs remained viable after freezing at -15°C (5°F) for 6 months, and also survived desiccation for 6 months.

High heat (e.g., a propane torch, boiling water or incineration) is usually used to decontaminate fomites. Boiling lye water could also be employed but is caustic. Eggs can be washed off surfaces with a 1% sodium hypochlorite solution, which stops them from sticking; however, the eggs are not killed by this treatment, and one study reported that treatment with undiluted household bleach for 90 minutes did not affect their viability.

Infections in Animals

Incubation Period

The incubation period is thought to be at least a week, and probably 2-4 weeks or longer, in most paratenic hosts. Experimentally infected mice developed clinical signs in 7 to 20 days. The onset of clinical signs, if any, is less predictable in definitive hosts.

Clinical Signs

B. procyonis in definitive hosts

Raccoons infected with *B. procyonis* are usually asymptomatic, but massive infections in young animals may cause intestinal obstruction or other complications such as perforation. Intestinal infections in dogs, which have fewer mature worms, have generally been found during routine fecal examination, and are unlikely to cause significant clinical signs. However, dogs can be affected by larva migrans, as they can also act as paratenic hosts.

B. procyonis in paratenic hosts

Nonspecific signs such as lethargy, depression, and a rough hair coat or disheveled feathers may be seen in some animals affected by migrating larvae. Most published clinical cases were caused by larvae that entered the brain or eye. Neurological signs may develop acutely and progress quickly, or they can be insidious and slowly progressive. The signs may wax and wane in some cases, and they sometimes stabilize if the larvae become encapsulated. Affected animals have had diverse signs such as circling, rolling, torticollis, ataxia, tremors, progressive weakness, paralysis, hypertonia, extensor rigidity, seizures and dysphagia. Birds may have difficulty perching or become unable to fly. Visual defects can include blindness, with granulomatous chorioretinitis, optic nerve atrophy and/or retinal depigmentation. Larvae may also damage other organs, especially the liver or lungs, and transient respiratory signs occurred shortly after inoculation in some experimentally infected animals. Overt signs of organ dysfunction seem to be uncommon, though there are rare reports of conditions such as myocarditis.

Other Baylisascaris spp.

Most definitive hosts probably do not suffer significant ill effects from moderate numbers of worms in the intestines; however, there are occasional reports of enteric impactions or other complications, such as rare reports of intestinal perforation or peritonitis in captive skunks infected with *B. columnaris.* The migrating larvae of some *Baylisascaris* species may cause clinical signs in their definitive hosts as well as paratenic hosts. *B. schroederi* is reported to affect giant pandas in both the larva migrans and intestinal stage, with diarrhea, constipation and/or nonspecific signs of illness, and occasional deaths. Heavy infestations of *B. transfuga* in bears are also thought to result in illness, including reduced body condition or even death. In one report, a juvenile bear that drowned due to other causes had granulomatous peritonitis. *B. venezuelensis* was thought to be the cause of death in one spectacled bear, which was in poor condition and had signs of larval migration (congestion and hemorrhagic foci) in its lungs. *B. laevis* has also caused liver or lung lesions in some experimentally infected animals. A groundhog infected with this organism had signs of pneumonia, and two guinea pigs died after developing dyspnea, bloody stools and ataxia and becoming emaciated.

Paratenic hosts can have signs of larva migrans similar to those seen with *B. procyonis*. *B. melis* and *B. columnaris* may cause extensive damage in some experimentally infected rodents and rabbits. Skunks were associated with one outbreak of neural larva migrans in nonhuman primates at a zoo, suggesting that *B. columnaris* may have been responsible. Some other species of *Baylisascaris,* such as *B. potosis* or *B. transfuga*, seem to be less pathogenic and invade the brain less often; however, *B. transfuga* was found in the CNS of two orphaned, newborn moose calf siblings born in the wild .One calf was weak and unable to swallow or suckle properly; the other was euthanized after breaking its leg. This organism was also the most likely agent in Japanese macaques with CNS signs at a zoo.

Post Mortem Lesions   ⬛ **Click to view images**

Nematodes may be found in the intestines of the definitive host. Mature *B. procyonis* are large tan roundworms. The female is 20 to 22 cm long, approximately twice the size of the male.

Migrating larvae can cause hemorrhagic or necrotic lesions and tracks, as well as granulomas, in many tissues. Larvae that invade the CNS can cause focal areas of palpable softening and discoloration, as well as small multifocal hemorrhages. However, fatal cases with microscopic lesions

but no gross abnormalities are possible, especially in birds. Larvae may be found both within lesions and in areas of the brain that appear to be normal. In some cases they may no longer be present by the time the brain is examined. Inflammation from migrating larvae may also result in pneumonia, myocarditis, hepatitis, myositis, peritonitis and other lesions outside the CNS. Extraneural granulomas occur less often in birds than mammals; the larval burden in clinically affected birds can be very low, and the lesions may be limited to the brain.

Diagnostic Tests

Intestinal infections in definitive hosts can be diagnosed by identifying the eggs in feces, or worms in the feces or vomitus. Fecal samples should be collected for at least 3 days before concluding that a raccoon is not infected with *B. procyonis*. *Baylisascaris* and *Toxocara* eggs are very similar and readily confused; however, *Baylisascaris* eggs are darker and somewhat smaller, with a finely granular surface on close examination, rather than having the coarsely pitted surface of *Toxocara*. PCR tests to identify *B. schroederi* in the feces have been developed for use in giant pandas. Commercial coproantigen tests (ELISAs) that detect *Toxocara* infections in dogs can cross-react with *Baylisascaris*.

Baylisascaris larva migrans is difficult to diagnose in live animals. A presumptive diagnosis can be made based on a history of exposure to raccoons or other definitive hosts, combined with clinical signs and laboratory results suggestive of a parasitic infection. Peripheral eosinophilia can be seen in mammals with neural larva migrans from *Baylisascaris*; however, eosinophilia may only be found in cerebrospinal fluid (CSF) in birds. Serology is uncommonly employed for diagnosis in animals, but *B. schroederi* ELISAs have been published for giant pandas, and an ELISA used in humans appeared to be useful for detecting antibodies to *Baylisascaris* in some nonhuman primates (apes, Old World monkeys). Serology must be interpreted in conjunction with other findings; the latter ELISA found a number of seropositive animals in zoos, none of which were symptomatic. A rising titer is expected to be helpful in clinical cases. Imaging studies (computed tomography, magnetic resonance imaging) are not considered practical in most cases, though lesions in deep white matter appear to be suggestive of *Baylisascaris* neural larva migrans in nonhuman primates.

A definitive diagnosis of larva migrans depends on the identification of the parasite within tissues in a biopsy sample or, more often, at necropsy. *Baylisascaris* larvae can be distinguished from some other nematodes such as *Toxocara* by their large size (*B. procyonis* larvae are up to 80 μm in diameter and up to 1900 μm in length) and their morphology. However, larvae in tissues can be difficult to identify, and misidentification is common. Species of *Baylisascaris* have traditionally been distinguished by morphology, but some organisms appear very similar. PCR tests, used mostly in research, can be helpful in definitive

identification, though some organisms such as *B. columnaris* and *B. procyonis* are closely related even in genetic tests. Epidemiological evidence, such as a history of exposure to raccoons but not skunks or badgers, is suggestive.

Treatment

Definitive hosts can be treated with various anthelmintic drugs including piperazine, pyrantel, ivermectin, moxidectin, albendazole, fenbendazole and flubendazole. Some monthly heartworm/intestinal worm preventive tablets may also be effective, although one treatment was not always sufficient to clear all of the worms. Neural larva migrans is treated with anthelmintic drugs that penetrate well into the CNS (e.g., albendazole, mebendazole), but the prognosis is guarded as the damage may be irreversible. Corticosteroids are employed concurrently to control inflammation, which contributes to the pathology and can be exacerbated by the death of the larvae. Supportive treatment is administered as appropriate.

Control

Disease reporting

Baylisascariasis is not generally reportable; however, veterinarians should consult their national and/or local guidelines for confirmation. State regulations should be checked in the U.S.

Prevention

Infections with *Baylisascaris* are difficult to prevent completely, as the hosts are common, the eggs can remain viable for long periods in the environment and decontamination is difficult. Prevention relies mainly on avoiding contact with feces and areas of fecal contamination. However, some monthly heartworm/ nematode preventives appear to be useful against intestinal infection with *B. procyonis* in dogs. In high-risk areas, dogs that are not on these agents should receive regular fecal examinations. Anthelmintic baiting has been explored in wild raccoons, and appears to be promising, though its practicality, long-term sustainability and potential for encouraging drug resistance must be considered.

Housing in zoos and wildlife exhibits should be designed to minimize paratenic hosts' exposure to fecal contamination from raccoons, skunks and other definitive hosts. Paratenic hosts are sometimes treated prophylactically with anthelmintics, especially after a high risk exposure. Captive definitive hosts should be tested regularly and dewormed as necessary, and carnivorous species should not be fed wild paratenic hosts that might carry larvae. Newly acquired definitive hosts should be quarantined until they are parasite-free. Once contamination of a definitive host's environment has occurred, it can be difficult to remove the eggs completely. Keeping these animals in dedicated cages that can be cleaned, if necessary, with the harsh methods required to destroy *Baylisascaris* eggs can be helpful.

Morbidity and Mortality

B. procyonis is a common parasite in wild raccoons, particularly young animals. In the U.S., its prevalence in different regions is reported to range from < 5% to 60% or higher. A seasonal peak in egg shedding seems to occur in the fall in northern temperate climates, followed by a decline over winter and early spring. Patent intestinal infections with this organism seem to be uncommon in dogs. One survey in the U.S. found *B. procyonis* eggs in 0.005% of canine fecal samples, with regional averages varying from 0.0004% to 0.007%. It was, however, detected in 0.4% of dogs on Prince Edward Island, Canada. A recent study found that *B. procyonis* was less readily established in dogs than raccoons, infections were less likely to become patent, and dogs with patent infections stopped shedding eggs within 1-2 months. *B. procyonis* migrates minimally during its development in raccoons and is rarely symptomatic in that species even when the number of parasites is large. However, severe or fatal illnesses can occur in dogs with larva migrans.

Other species of *Baylisascaris* are also likely be common in their definitive hosts. In Europe, *B. transfuga* has been found in 14% to ≥ 50-60% of bears in some regions, while *B. columnaris* was detected in approximately 60% of striped skunks in Utah and 25% of captive striped skunks in Europe, and *B. schroederi* occurs in up to half of all pandas in some parts of China. Some definitive hosts, such as pandas infected with *B. schroederi*, can have significant adverse effects from parasitism, while others, such as skunks infected with *B. columnaris*, seem to be affected only rarely.

The effect of larva migrans on paratenic hosts is influenced by the host, the species of parasite and the location of the larvae. Some organisms (e.g., *B. transfuga*, *B. potosis*) have smaller larvae and/or migrate less extensively, decreasing the risk of damage to important organs such as the brain or eye. However, even *B. procyonis* does not affect these sites in most animals: only 5-7% of this organism's larvae are estimated to enter the CNS in experimentally infected mice. Clinical cases with CNS signs are often serious, and can result in residual neurological sequelae even if the animal survives. One outbreak in an aviary of 35 cockatiels (*Nymphicus hollandicus*) eventually affected 34 of the birds, and only one symptomatic bird survived. *B. procyonis* also seems to cause significant morbidity and mortality in some wild species, such as certain North American populations of the Allegheny woodrat (*Neotoma magister*) and the white-footed mouse (*Peromyscus leucopus*). However, many exposures are probably subclinical. One study found antibodies to *Baylisascaris* in 22-57% of healthy nonhuman primates (great apes, lesser apes, Old World monkeys) at US zoos.

Infections in Humans

Incubation Period

The incubation period in humans is not well established, but neural larval migrans may be seen as soon as 2-4 weeks after ingesting eggs.

Clinical Signs

Relatively few cases of baylisascariasis have been described in humans, and the full spectrum of disease may be incompletely understood. Serological studies suggest that some exposures are subclinical, and the symptoms in clinical cases are likely to depend on the location and number of migrating larvae.

Most reported cases have involved the CNS and/or eye. Neural larva migrans has been seem mainly in young children. It affected the brain in most reports, but spinal cord involvement has been described. Clinical signs vary, but motor deficits and seizures are common, and patients may also have altered levels of consciousness, behavioral changes, loss of vision and/or speech and other signs. Fever seems to be absent in most cases. The initial signs may be mild, with subtle behavioral changes (e.g., lethargy, somnolence, irritability), weakness, speech defects and/or mild changes in vision, but they can rapidly become severe. However, there is one report of a child who experienced gradually progressive ataxia, quadriparesis and developmental issues, and was thought to have been infected several years before baylisascariasis was eventually diagnosed.

Neural larva migrans can be fatal, and many surviving patients have been left with serious neurological defects. However, there were a few cases with better outcomes, including apparent full recovery in one child who was diagnosed with relatively mild symptoms. Very young patients may continue to improve over time, to a greater or lesser extent, as the developing brain grows and forms new connections.

Ocular larva migrans sometimes occurs concurrently with CNS signs, but it can also be an isolated syndrome. The latter condition tends to affect older children and adults. Ocular larva migrans is typically characterized by inflammatory and degenerative changes mainly in the retina and optic disk, and usually affects only one eye. Common lesions include retinochoroiditis, vitreitis, the formation of intraocular granulomas and, in more severe cases, retinal detachment and/or panophthalmitis. Clinical signs may include transient obscuration of vision, photophobia, other signs of diffuse unilateral subacute neuroretinitis (DUSN) and loss of vision. Some visual defects can be permanent.

B. procyonis might also be able to cause visceral larva migrans in humans. Common signs of visceral larva migrans caused by other genera of parasites (e.g., *Ascaris*, *Toxocara*) include eosinophilia, nonspecific clinical signs such as low-grade fever, nausea and lethargy and, in some cases, enlargement of the liver and/or symptoms of pneumonitis.

Eosinophilia has been reported in most patients with baylisascariasis and sometimes in asymptomatic family members; and mild enlargement of the liver accompanied CNS signs in two patients. One group noted that mild respiratory signs shortly before the onset of neurological signs in some patients might have been related to parasite migration through the lungs, but this is speculative. In one fatal case of *B. procyonis* larva migrans, an eosinophilic mass resembling a tumor was found in the heart. A macular rash, seen mainly on the face and trunk, has also been described.

Diagnostic Tests

Antemortem diagnosis of baylisascariasis can be difficult. In ocular larva migrans, an ophthalmoscopic examination may occasionally reveal large, motile larvae in the retina, as well as chorioretinitis and other signs of DUSN. Unless a brain biopsy is done and a larva is found in that sample, antemortem diagnosis of neural larva migrans is usually based on serology, with a history of probable exposure and supportive evidence from other tests, including blood and CSF findings suggestive of a parasitic infection.

Serological tests for *Baylisascaris,* including ELISAs, indirect immunofluorescence and immunoblotting (Western blotting), are not widely available in commercial laboratories but may be provided by university research laboratories or public health agencies (e.g., the CDC in the U.S. or the National Reference Centre for Parasitology in Canada; see Internet Resources). Because the currently available tests detect IgG and healthy people can be seropositive from past exposure, a rising titer in serum is helpful. Antibodies to *Baylisascaris* can also be found in CSF. Imaging techniques (diffuse deep white matter changes on MRI) and encephalography provide supportive evidence and help rule out other causes, such as *Toxocara* neural migrans.

A definitive diagnosis can be made retrospectively if larvae are identified in the CNS at autopsy.

Treatment

Early diagnosis and treatment provides the best chance of a good outcome. The optimal treatment of baylisascariasis is still uncertain, but albendazole is currently thought to be the drug of choice in patients with CNS signs, and should be started as early as possible. Corticosteroids are given concurrently to suppress inflammation caused by the death of the larvae, as well as to dampen the existing inflammatory response. Laser photocoagulation is the treatment of choice for ocular larva migrans if the larva is not in a critical area, but other therapies may also be used.

Prevention

The risk of infection with *B. procyonis* can be decreased by avoiding contact with raccoon feces and contaminated sites. Raccoons sometimes defecate spontaneously at single use sites, but they often establish "latrines" visited by multiple animals. Latrines are typically found at the base of trees, in the forks of trees, or on raised horizontal surfaces such as fallen logs, stumps, large rocks, woodpiles, decks and rooftops. Raccoons will also defecate in attics, garages and haylofts if they have access. Their feces is typically dark and tubular, with a particularly pungent odor. It often contains undigested seeds, fragments of corn or other foods, bones and/or shells.

Raccoons should be blocked from entering attics and basements, and discouraged from visiting residential areas by measures such as rendering food attractants (including garbage and birdseed) inaccessible. Sandboxes should be covered when not in use, to prevent raccoons from defecating in them. Public health authorities often advise that raccoons not be kept as pets, especially in homes with young children. All captive raccoons should be examined regularly for *B. procyonis* eggs, and dewormed if necessary. Regular fecal examination and anthelmintic treatment also reduce the risk that dogs may shed *B. procyonis*, though infections generally seem to be uncommon in this species.

Raccoon feces and contaminated material at sites where people would be expected to contact them (e.g., decks and patios) should be removed and burned, buried or sent to a landfill as soon as possible; the eggs usually take 2-4 weeks or more to reach the infectious stage. Care should be taken to avoid getting the eggs and feces on hands and clothes. Gloves and protective clothing should be worn, and the hands should be washed immediately afterward with soap and water. A face mask may be helpful in dry conditions, to prevent the inhalation of dust and debris that may contain various fecal pathogens, and to prevent accidental contamination of the mouth. Ordinary disinfectants are ineffective against these eggs. but decks, patios and other surfaces may be treated with boiling water, steam cleaned, or flamed with a propane flame-gun, as appropriate. Incineration of contaminated objects is also effective. More extensive measures in a few situations included removing the top few inches of soil or treating the area with a xylene-ethanol mixture. In many cases, some *Baylisascaris* eggs may remain despite rigorous cleanup measures.

Exposure to *B. procyonis* may be difficult to prevent completely, as raccoons are common even in suburban locations and the infective eggs can survive for long periods in contaminated soil. However, behaviors that limit exposure to eggs, especially in large quantities, are expected to be helpful. Children should be stopped from eating dirt and taught not to put objects into their mouths. Good hygiene, especially hand washing after outdoor play or contact with animals, including dogs, should be encouraged. Supervision is advisable for younger children in areas where raccoon feces may be present, as the variety of objects in the feces may attract them and they may become infected when they place their fingers in their mouth. Developmentally disabled individuals should also be supervised. Prophylactic albendazole has been used in some people after exposure to raccoon eggs.

Given the possibility that organisms other than *B. procyonis* might also infect humans, common sense

measures should be taken to reduce exposure to animal feces in general.

Morbidity and Mortality

Baylisascariasis seems to be an uncommon disease in people, with fewer than 50 clinical cases documented worldwide as of 2021. However, it is possible that this disease is underdiagnosed, as some cases might be attributed to other causes. Subclinical exposures also seem likely: parents or siblings of affected children are sometimes seropositive but asymptomatic, and surveys in healthy people found antibodies to *Baylisascaris* in 8% of children in the Chicago area, 8% of adults in Santa Barbara, California, 7% of wildlife rehabilitators in the US and Canada, and 16% of raccoon trappers. In one case, a small number of larvae were found in the brain of an elderly woman who died of unrelated causes and had no recent clinical signs attributed to this organism (mild signs might have been masked by pre-existing Alzheimer's disease). It is possible that severe neurological disease occurs only after exposure to large numbers of eggs, or in hosts who are unusually susceptible.

Neural larva migrans tends to be seen mainly in infants and young children under 2 years of age. One likely reason is that young children are more likely to eat dirt or put contaminated fingers, soil or other fomites into their mouths. The small size of their brain may also contribute to the severity of the damage. Some cases of neural larva migrans occurred in older, developmentally challenged individuals or adults with neurological defects, particularly those who had a history of pica or eating dirt. Isolated ocular larva migrans tends to be seen in older children and adults with no other signs of baylisascariasis. Some of these individuals had no history of exposure to raccoons or the exposure was incidental, and it is possible that this condition can be caused by small numbers of eggs. The risk is expected to be elevated in people who hunt, trap, perform taxidermy and handle wildlife.

The outcome in cases with CNS signs appears to be poor if no treatment is given, or if treatment is delayed. About a dozen cases were reported between 1981 and 2002, with a case fatality rate of approximately 50%, and the survivors had severe, permanent neurological sequelae. Treatment was apparently ineffective in many of these cases, possibly because irreversible damage was already present. The prognosis has improved to some extent in recent years, though it remains guarded. As of 2018, 18 of 23 children with neural larva migrans have survived, and a few had mild to moderate residual neurological deficits or even recovered fully. Earlier diagnosis and treatment probably contributed to the improved outcome in these cases. It is also possible that some milder cases with less extensive lesions are now being recognized and the earliest studies were based on a subset of severe cases.

Internet Resources

U.S. Centers for Disease Control and Prevention (CDC). *Baylisascaris* Resources for Health Professionals

International Veterinary Information Service (IVIS)

The Merck Manual

The Merck Veterinary Manual

National Reference Centre for Parasitology (NRCP) Canada

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