Other worms

Meningeal worm (deer worm)

Parelaphostrongylus tenuis commonly referred to as P. tenuis, deer worm or meningeal worm, is a parasitic worm of small ruminants and modified ruminants. Its natural (i.e., definitive) host is white-tailed deer. Up to 90% of deer harvested during the hunting season in the Northeastern US are infected. It rarely causes any disease or health issues in this species. Unfortunately, it also readily infects abnormal hosts such as goats, sheep, alpacas, llamas, elk, moose, and fallow deer. Even just one worm can cause serious neurological damage in these other species. The worm has an "indirect" life cycle. Although it reproduces in white-tailed deer, the immature larvae shed in deer feces must further develop in an intermediate host (either land snails or slugs) before being able to infect another ruminant.

The natural host of the meningeal worm is the white-tailed deer.

Image credit: Susan Schoenian

LIFE CYCLE

Immature (stage 1) deer worm larvae are shed in the mucus coating of feces of infected white-tailed deer. They are then either ingested by or burrow into the "foot" of land snails or slugs feeding on the feces. During the next 2 to 3 months, the larvae develop from stage 2 to stage 3 larvae in the snail or slug. Unlike the stage 1 and 2 larvae, the stage 3 larva is infectious to ruminants. Eventually the stage 3 larva is excreted in the snail or slug's slime trail where it can persist for many days.

Sheep and goats become infected by eating fresh forage that is either contaminated with infected snails or slugs or with their infected slime trail. Once ingested by the small ruminant, the mature larvae migrate out of the true stomach into the abdomen and follow nerves in the body wall until they reach the spinal cord. In the definitive host (white-tailed deer), the larvae then follow an orderly and benign pattern of maturation in the grey matter of the spinal cord before migrating in approximately 40 days to the head where they mate and lay eggs in the tissues surrounding the brain. The eggs pass through the bloodstream to the lungs where they hatch into stage 1 larvae, and are then coughed up, swallowed, and passed into the deer's feces.

However, in abnormal hosts such as small ruminants, the larvae become disoriented as they migrate through the meningeal layers protecting the spinal cord into the grey matter. Their travel through the spinal cord and nearby nerves is more active and prolonged. This erratic activity, jointly with the body's inflammatory response, can cause damage to the central nervous system leading to the neurologic signs of the disease discussed next.

IMPACT IN THE ANIMAL

There are two common ways that deer worm damage presents itself in infected goats or sheep. In the first phase, migrating larvae irritate individual nerves where they merge with the spinal cord, causing a constant itch at the area of skin to which the nerve runs. The animal rubs and/or bites this area incessantly, leading to hair loss and possibly raw wounds along an area on one side of the body. In the second more serious scenario, the animal's hindquarter locomotion is affected, and the animal may progress from mild stumbling, especially when turning, knuckling over of the toes of one or both hind legs, or general hind end weakness to total paralysis of the hind legs, leaving it in a dog-sitting posture or down and unable to get up. Sometimes, the front limbs are affected instead or in addition.

Fortunately, even in cases of total limb paralysis, some animals recover fully given proper treatment and/or time. Less commonly, the neurological damage can affect the brain and cause head tilt, circling, rapid eye flickering, and difficulty chewing. Appetite and body temperature typically remain normal in animals affected with deer worm unlike in goats and sheep infected with other common neurological diseases

TREATMENT

Treatment for deer worm centers around two goals: 1) Kill the worm before it does further damage and 2) Repair or mitigate the existing nerve damage. Fenbendazole (Safe-guard®, Panacur®) is often the dewormer of choice because of its ability to cross the blood-brain barrier into the cerebrospinal fluid to effectively kill the worm when given at high dosages for many days -- while still being safe for the host animal. Theoretically, ivermectin (Ivomec®), although also relatively safe at high dosages, is unable to penetrate the blood-brain barrier to kill larvae already in the central nervous system. Instead, it is only effective at killing additional larvae that have not yet entered the spinal cord.

One possible sign of deer worm damage is constant itching, hair loss and even raw spots in a vertical band below the spinal cord.

Image credit: Mary C. Smith

Steroids such as dexamethasone and/or non-steroidal anti-inflammatory drugs such as flunixin meglumine (Banamine®) are used to reduce inflammation and further damage to the nervous tissue. Please note that dexamethasone should not be used in late-pregnant animals as it may induce labor. Withdrawal periods (the number of days after treatment that an animal's milk or meat cannot be used) vary widely for different drugs and need to be considered in consultation with the prescribing veterinarian when deciding on a treatment. Some animals spontaneously recover without any treatment, while not all treated animals inevitably recover. To date, there have been no controlled studies with goats and sheep comparing the effectiveness of various treatments for deer worm to no treatment at all. However, a four-year study at Cornell University compared two treatment protocols in naturally-infected goats and sheep on ten farms near Ithaca, New York.

Enrolled animals were randomly assigned within herd to a "treatment plus ivermectin" (T+I) protocol or to a "treatment only"(TO) protocol, with protocols alternating for succeeding animals from the same farm. The T+I animals received oral fenbendazole (25 mg/kg live weight) and subcutaneous ivermectin (0.5 mg/kg) for 5 days and intramuscular dexamethasone (0.2 mg/kg) for 3 days followed by 0.1 mg/kg for 2 days);

the TO animals received the same 5-day course of fenbendazole and dexamethasone but received a placebo rather than ivermectin subcutaneously.

Examinations were conducted before and after the 5 days of treatment to assign animals a neurological score from 1 (unable to stand) to 5 (no detectable neurologic deficits or only an expert would notice) scale, with a score of 4 indicating likely to function in the herd for breeding though noticeably impaired. After the 5-day treatment period, animals were classified as "recovered" if they had a score of 4 or higher. All 11 goats on the T+I protocol recovered, while 6 of the 9 TO treated goats recovered without further treatment, and 3 goats required additional treatment to recover. Six of 9 T+I sheep recovered but 3 had to be euthanized. Five of 9 TO sheep recovered without further treatment, two required additional treatment and 2 had to be euthanized.

The better outcomes for goats are probably explained by closer observation of goats (coincidentally all goats were returned to barns at night while all sheep were left in pastures 24/7), which resulted in earlier identification of deer worm damage in the goats. Studies with larger numbers of animals are needed to definitively state whether including ivermectin improves the effectiveness of deer worm treatment. Inclusion of ivermectin for 5 days increases the drug withdrawal period for the standard protocol to 96 days or more according to FARAD, the Food Animal Residue Avoidance Databank. If there is no concern about the withdrawal period, the Cornell study cannot rule out the possibility that including ivermectin in the fenbendazole/dexamethasone protocol may benefit the treatment of highly valuable animals.

PREVENTION

Regular, frequent deworming to prophylactically treat for deer worm before it enters the spinal cord is costly, time-demanding, and inadvertently selects for stomach and intestinal worms resistant to the drug,

In more advanced cases, the animal may experience paralysis of the hind limbs.

Image credit: Mary C. Smith

rendering that dewormer ineffective at controlling these damaging gastrointestinal parasites. Therefore, it is generally not advised for goats and sheep. There is no selection for resistant deer worm larvae because they never complete their life cycle in sheep and goats.

The most effective way to prevent deer worm infection is to limit goat and sheep exposure to infected slugs, snails, and deer. Places where deer bed down, and newly cleared brush or woodlands areas are particularly high risk. Low, moist areas are conducive environments for infection, especially in the late summer and fall when L3 larvae are more likely to have matured in the snails and slugs readily found that time of year in leaf litter and fresh forage. Daily patrolling by guardian dogs during the winter and grazing season and other deterrents such as human activity and/or deer-proof fencing to discourage deer from frequenting pastures will also help reduce the incidence of disease. There is some question as to whether having guinea hens or ducks in the grazing paddocks may help control the incidence of slugs and snails and cut down on infection.

IDENTIFICATION

Fecal sampling to identify worm larvae is not useful in diagnosing deer worm. Even in white-tailed deer, this long-lived worm is generally present in low numbers. The eggs do not pass to the feces and fecal larvae counts are low. The worm does not appear to successfully mature, mate, and produce eggs and larvae in abnormal species such as goats and sheep. Instead, the diagnosis of deer worm in these species relies on the grazing history of the animal and the physical signs of the disease. The fluid that surrounds the brain and spinal cord(cerebrospinal fluid) will also often have characteristic changes to it when viewed microscopically. An absolute diagnosis can be made with difficulty post-mortem by screening the nervous tissue to find the live worms or the microscopic damage they have done.

Videos showing typical signs of deer worm in goats and sheep and more information on Cornell studies on immune responses to deer worm exposure, treatment protocols and life cycles can be found at https://blogs.cornell.edu/smallruminantparasites/chemical-treatment-protocols/. *Image credit: Susan Schoenian*

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