Home on the Range: Where Deer and Antelope Shouldn't Play

Breanna Caitlin Brown

Mississippi State University College of Veterinary Medicine

Class of 2020

Clinicopathological Conference

November 1, 2019

Advisor: David Smith, DVM, PhD, DACVPM (Epidemiology)

Mikell and Mary Cheek Hall Davis Endowed Professor
Department of Pathobiology and Population Medicine

Introduction

Wild animal parks provide opportunities for visitors to observe non-native species in a natural setting. Although wild animal parks are educational and entertaining to the public, the utmost care must be taken to ensure all animals remain healthy. Wild animal parks sometimes combine species that have not evolved together which may lead to unintended consequences. Housing animals in non-native ecosystems with a variety of different species of animals can cause a plethora of issues. For example, commingling of species, especially at greater population densities than typically seen in nature may result in a greater likelihood that some animals may be exposed to non host-adapted pathogens or acquire accidental end-stage infections which may be deadly.

History and Presentation

A recently established 466-acre wild animal park in northern Mississippi houses a variety of different species. The property is split into four, large, high fence pastures with a shared watering source. Species are split between the pastures and include (in no particular order) Grants Zebra, Grevy's Zebra, Chapmans Zebra, Sable Antelope, Kudu Antelope, Addax Antelope, Aoudad Antelope, Gemsbok Antelope, Wildebeest Antelope, Nyala Antelope, Sitatunga Antelope, Blesbok Antelope, Beisa Antelope, Eland Antelope, Springbok Antelope, Impala Antelope, Arabian Oryx Antelope, Fringe Eared Oryx Antelope, Nile Lechwe Antelope, Red Lechwe Antelope, Waterbuck Antelope, Nilgai Antelope, Grants Gazelle, Dama Gazelle, Thompson Gazelle, Axis Deer, Fallow Deer, Pere David Deer, Swamp Buffalo, Watusi Cattle, Dromedary Camels, Reticulated Giraffe, Flamingo, and Ostrich.

The herd included 12 castrated 8-month old Nilgai. On February 5th, 2019, an approximately eight-month Nilgai antelope became stiff in the front limbs and hunched in the back. Over the course of a week, two more Nilgai began to show similar clinical signs. On February 9th, 2019, the population medicine department visited the park. At the time of presentation, one Nilgai had already died and was submitted for necropsy at Mississippi State University College of Veterinary Medicine. Prior to death, the deceased Nilgai (Nilgai #1) had stiff legs and a hunched back; he had been treated by the referring veterinarian with an unknown amount of flunixin meglumine , lactated ringers, and vitamin B12 but did not recover. Two other Nilgai and one 6-month old llama were also showing neurologic signs similar to the first nilgai. Nilgai #2 was treated with flunixin meglumine, intravenous fluids, and a 150-200lb dose of fenbendazole on February 9th, 2019, by the population department. Nilgai #3 was treated with flunixin meglumine and fenbendazole as well. Upon examination by the population medicine rotation students, Nilgai #2 was non-ambulatory with the right thoracic limb showing no neurological deficits. The left thoracic limb had decreased proprioception, absent proprioception in the left pelvic limb, and increased proprioception in the right pelvic limb. He had absent nociception in the left pelvic limb and decreased nociception in the right pelvic limb. A decreased menace response was noted on the left and the remainder of the cranial nerves were intact. These neurological signs classified Nilgai #2 as non-ambulatory paraplegic in the left rear and paraparetic in the right rear. Nilgai #3 was not as severely affected but was still showing neurologic signs of circling, generalized proprioception defects and inappropriate mentation. The owner and referring veterinarian noted Nilgai #3 was showing the “early signs” for this disease outbreak. The llama was found laterally recumbent and was moved to a nursing pen prior to our visit. He

was treated with intravenous fluids and palliative care. At the time of presentation, the llama was ambulatory paraparetic but was eating and drinking normally.

Necropsy Findings

Nilgai #1 was submitted for necropsy at Mississippi State University College of Veterinary Medicine. Necropsy revealed subacute to chronic extensive myelitis of the spinal cord with myelomalacia and intralesional nematodes (morphology consistent with *Parelaphostrongylus tenuis).* The spinal cord was malacic from T5 caudally. Suppurative and necrotizing cystitis was noted with focal rupture leading to a uroabdomen. The abdominal cavity was filled 3 liters of red-tinged, thin, opaque fluid that smelled of ammonia. The bladder was markedly distended with widely disseminated ecchymotic hemorrhages and a small perforation which leaked urine. Subcutaneous and intramuscular hemorrhage was widely disseminated and severe; most notably along the ventral abdomen, sternum, and inguinal region. Subacute, focal pulmonary thrombosis and severe diffuse serous atrophy of fat was also observed. Due to the neurologic deficits, this nilgai lost motor and urinary function leading to a urinary tract infection, subsequent bladder rupture and ultimately a septic uroabdomen. The cause of death was reported to be septicemia.

Histopathology findings

In two sections of the spinal cord between T5 and T6, there are 5 nematode cross sections that are surrounded by neutrophil necrosis and hemorrhage. The nematodes are characterized by 2-4um thick eosinophilic cuticle, polymyarian-coelomyarian musculature, accessory hypodermal chords, a pseudocoelom, an intestine lined with few multinucleate cells, and testes. These nematode characteristics are consistent with *P tenuis*. Scattered throughout other sections of the spinal cord white matter, there are widespread digestion chambers containing glitter cells or

eosinophilic cell debris. Multifocally, the myelin sheaths are variably dilated and contain swollen eosinophilic axons, known as Wallarian degeneration.

Pathophysiology

Parelaphostrongylus tenuis, also known as the meningeal worm, is a nematode which resides in approximately 80% of white tail deer (*Odocoileus virginianus)* in endemic areas of the United States (Gandolf/Beest). *Parelaphostrongylus tenuis* infection has also been reported in a variety of other species including: moose, caribou, reindeer, elk, wapiti, mule deer, black-tailed deer, fallow deer, eland, domestic sheep, domestic goats, domestic cattle, domestic horses, guinea pigs, sable antelope, scimitar-horned orynx, bighorn sheep, blackbuck antelope, llama, camels, bongo, and pronghorn antelope (Simmons, Gandolf/Beest, Pinn, Nagy). Domestic cattle are seemingly resistant to infection whereas the llama appears to be the most sensitive to the development of clinical disease (Nagy).

White tail deer serve as the natural host of the meningeal worm and are usually only subclinically affected. The lifecycle is complex and begins with the adult parasite residing in the subarachnoid space of the white tail deer. Eggs leave the central nervous system via the venous sinus and embryonate into L1 larvae. First stage larvae enter the pulmonary parenchyma and migrate through the trachea where they are coughed up, swallowed, move through the gastrointestinal tract and exit the deer through the feces. The larvae then enter gastropods (slugs and land snails), where they develop into the infective L3 stage. The L3 larvae are hardy in the environment and have the ability to overwinter in the environment prior to ingestion by the white tail deer or during spring grazing. The larvae are released from the gastropod during digestion in the abomasum and begin to migrate through body tissues to the central nervous system. The larvae then enter the dorsal horn of the spinal cord and develop into an adult, usually between

20-30 days after infection. From the dorsal horn, they migrate to the subarachnoid space using the dorsal nerve roots. The prepatent period in the white tail deer is approximately 90 days, with a complete lifecycle occurring every 4 months (Nagy).

Infection of *P. tenuis* through the ingestion of gastropods leads to migration of the larvae into the spinal cord, but in species other than white tail deer, the migration of the nematode is non-directional and the larvae die prior to reaching the brain in most cases (Gandolf, Pinn, Nagy) resulting in more severe neurological disease than is typically observed in white tail deer. Within the spinal cord are subsections of white matter and grey matter. Migration tracts are more common in the white matter of the spinal cord, however migration through the grey matter is also possible (Pinn). The spinal cord is responsible for receiving and distributing information to the peripheral nervous system, local integration of the sensory and motor functions, and relaying sensory and motor information to and from the brain (Thomson). When these areas are damaged due to larvae migration, neurological signs develop. The location of the inflammation/ migration tract dictates the clinical signs. Signs can range from single limb lameness or rear limb weakness to head tilt, ataxia, circling, blindness, progressive loss of motor function and death (Gandolf). Overall presentation of the animal can be summarized as unilateral or bilateral asymmetric signs of spinal cord disease which progresses cranially (Pinn, Nagy).

Most effected animals do not return to normal function due to a permanent damage of the spinal cord known as myelomalacia. Myelomalacia is ischemic or hemorrhagic necrosis of the spinal cord which is most commonly noted in deep pain negative dogs following intervertebral disk herniation (Platt). In the case of meningeal worm, the localized ischemic event is the migration/death of the larvae which leads to an inflammatory response and cell death. The necrotic portion of the lesion can spread cranially or caudally. Animals with known infection of

P. tenuis who were successfully treated and remained in the herd had chronic changes to the spinal cord years later.

Diagnostic Approach/Considerations

Diagnosing *P. tenuis* is difficult. Antemortem diagnosis is impossible with *P. tenuis,* therefore a tentative diagnosis must be made based off of clinical signs, history of exposure to white tail deer, the presence of eosinophils in the CSF, and response to treatment (Pinn, Pierre-Charles Lefevre). Increased CSF eosinophil percentage and eosinophilic pleocytosis strongly supports a presumptive clinical diagnosis of *P. tenuis* in camelids (Pinn, Nagy). One report shows the CSF of clinically effected animals have increased concentration of proteins, RBCs, WBCs, and eosinophils (Smith). The use of CSF tap is strongly recommended in animals showing the correct neurological signs. Differential diagnoses include: aberrant migration of other parasites, protozoal myelitis, listeriosis, lead intoxication, caprine arthritis encephalitis, scrapie, rabies, trauma, copper deficiency, vitamin E/selenium deficiency, spinal cord/brain abscess, salt toxicity, osteomylelitis of the spinal cord, and polioencephalomalacia (Schoenian, Nagy).

Definitive diagnosis can only be made post mortem by demonstrating parasites in the spinal cord (Nagy). This can be challenging, especially if the animal has been "successfully treated" and the nematodes are no longer present in the spinal cord. Histopathology of the spinal cord will reveal microcavitation and spongiosis with gliosis and mononuclear cell infiltrates with scattered eosinophils (Nagy).

Treatment and Management

The current treatment of choice includes high dose fenbendazole (20-50mg/kg orally once daily for 5 days) and/or high dose ivermectin (0.3-0.4 mg/kg subcutaneously daily for 3-5 days)

(Lankaster). Successful treatment requires early diagnosis and aggressive treatment. The problem lies in the inability for most anthelminthic to penetrate the blood-brain barrier and the presence of the P-glycoprotein pump that actively removes drugs from the central nervous system (Nagy).

Each affected Nilgai was treated with 50mg/kg of fenbendazole orally, and 0.3mg/kg of ivermectin subcutaneously. Recommended future treatment included fenbendazole at a 50mg/kg dose administered orally every 24 hours for 3-5 days. Ivermectin at a 0.3 mg/kg dose was given subcutaneously every 30 days. Flunixin meglumine at a 0.5 to 1 mg/kg dose was administered every 12 hours until signs resolved. Supportive care was crucial for these sick animals. This included intravenous fluids, B vitamins, appropriate antibiotics to treat secondary infections, easy access to food and water, and physical therapy such as rotating the non-ambulatory Nilgai to ensure he did not develop pressure sores or nerve damage on the down limbs. Nonsteroidal anti-inflammatories and steroids have been used in these cases due to concerns of the inflammatory response to the parasite migrating through the spinal cord, rather than the structural damage caused by the migration, may account for the majority of clinical signs (Nagy).

Suggested de-worming protocol

Fenbendazole is an approved product as a medicated pellet for hoofed zoo and wildlife animals with a recommended dose of 7.5mg/kg to be fed over a 3-5-day period (Bliss). It is almost impossible to determine when and how much each animal will eat per treatment so spreading the dose over multiple feeding ensures the most animals will consume an adequate dose. A simple formula can be used to calculate how much to feed each herd. One pound of 0.5% fenbendazole pellets will deworm 750 pounds of wildlife so the producer should determine how much the herd weighs by estimating the average weight of the animals and multiplying that by the number of

animals in the herd. Then divide this number (lbs. of wildlife) by 750 to determine how many pounds of product to feed. A successful deworming protocol requires a seasonal program. Pellets should be fed once in the winter to remove parasite burden from the previous summer grazing season. Pellets should be fed twice in the spring one month apart to reduce contamination of parasite eggs in the environment. The first treatment will kill infective larvae and the larvae consumed from the pasture will be killed by the second treatment. Treatment should begin again in the late summer/early fall prior to breeding season to keep the immune system strong and help with a successful breeding season (Bliss). Current recommendation for the treatment of llamas includes monthly administration of an avermectin anthelmintic with treatment starting 1 month after the last hard frost and stopping following the first hard frost (Nagy). This recommendation is controversial considering such aggressive treatment is likely to lead to resistance in other parasites.

Other Management Strategies

Establishment of gravel roads or other vegetation breaks within the enclosure acts as a barrier to slugs and snails. Gravel can be placed around the perimeter fence or water source to limit migration of gastropods (Gandolf, Simmons, Nagy). Mollusicides must be used with caution due to potential for environmental toxicity. Non-native species who are less susceptible to contracting *P. tenuis* can be grazed on the pasture prior to grazing more susceptible species. The population medicine department determined the most effective way to eliminate *P. tenuis* infection is this situation was to eliminate native white tail deer populations within the fenced in parameter. Fences should be high tensile wire and tall enough to keep deer from entering the parameter. This will break the lifecycle and eliminate the potential for infection (Gandolf, Simmons). Another important management strategy included designing specific feeding

locations for the different species within pastures. This will help to eliminate competition for resources between the different species and should help to maintain adequate body condition score for all animals within the park.

Case Outcome

The owner was contacted in March and confirmed treatment was successful for the effected Nilgai and llama. No further contact has been made with the owner, therefore it is unknown if the white tail deer have been eliminated, if any of our recommended treatment strategies have been put into place, or the current status of the Nilgai.

References

Bliss DH, The Control of Gastro-Intestinal Nematode Parasites of Hoofed Wildlife in North America, MidAmerican Ag Research Technical Bulletin

Gandolf Rae, Beest, Julie Ter, Meningeal worm (Parelaphostrongylus tenuis), American Association of Zoo Veterinarians Infectious Disease Committee Manual 2013

Lefèvre, Pierre-Charles et al. Clinical Signs. Infectious and Parasitic Disease of Livestock Vol. 2 Paris: Lavoisier, 2010, 1684-1685

Nagy, DW, Parelaphostrongylus tenuis and other parasitic diseases of the ruminant nervous system, Vet Clin Food Anim 20 (2004) 393-412

Pinn T, Bender H, et al. Cerebrospinal fluid eosinophilia is a sensitive and specific test for the diagnosis of Parelaphostrongylus tenuis in camelids in the northeastern United States. Journal of Veterinary Diagnostic Investigation 2013: 25(1) 54-60

Platt SR, McConnell, FJ, et al, Magnetic Resonance Imaging Characteristics of Ascending Hemorrhagic Myelomalacia in a Dog, Veterinary Radiology and Ultrasound, 47(1), 2006, pp 78-82

Schoenian, Susan, Meningeal worm- deer worm-brain worm, Maryland Small Ruminant page, 2019

Simmons, HA, Steffer DJ, Parelaphostrongylus tenuis in Captive Pronghorn Antelope (Antilocapra Americana) in Nebraska, Journal of Wildlife Diseases. 38(4); 822-825

Smith MC, Buckner SM, et al. Field trial to evaluate the efficacy of ivermectin in the treatment of small ruminants with presumptive Parelaphostrongylus tenuis infections, Cornell University website

Smith, Bradford. Parelaphostrongylus tenuis in Large Animal Internal Medicine 4th Ed. Mosby, Elsevier, 2009, 1081-1082

Thomson, Christine and Hahn, Caroline in Veterinary Neuroanatomy A Clinical Approach, Hierarchical organisation in the nervous system, Saunders; ELSEVIER, 2012, pg 35