CEREBROSPINAL NEMATODIASIS CAUSED BY Parelaphostrongylus tenuis IN ANGORA GOATS IN TEXAS

Authors: FRED S. GUTHERY, and SAMUEL L. BEASOM
Source: Journal of Wildlife Diseases, 15(1): 37-42
Published By: Wildlife Disease Association
URL: https://doi.org/10.7589/0090-3558-15.1.37
CEREBROSPINAL NEMATODIASIS CAUSED BY
Parelaphostrongylus tenuis IN ANGORA GOATS IN TEXAS

FRED S. GUTHERY and SAMUEL L. BEASOM, Department of Wildlife and Fisheries Sciences, Texas A&M University, College Station, Texas A&M University, College Station, Texas 77843, USA.

LARRY JONES, Texas Veterinary Medical Diagnostic Laboratory, Drawer 3040, College Station, Texas 77840, USA.

Abstract: A natural infection of meningeal worm (Parelaphostrongylus tenuis) accounted for the death of 11 of 17 (65%) Angora goats in a study in South Texas during 1975. Clinical signs, gross pathology and histopathology in Angoras were similar to other abnormal hosts.

INTRODUCTION

Parelaphostrongylus tenuis is a common parasite of white-tailed deer (Odocoileus virginianus) in much of the eastern United States and Canada. Generally the nematode is silent in white-tailed deer but has produced neurologic disease in naturally-infected elk (Cervus canadensis), moose (Alces alces), caribou (Rangifer tarandus) and domestic sheep. The parasite is known from Louisiana, Arkansas and Oklahoma, three states which border Texas. Distribution in Texas is limited to eastern and southern regions (R. M. Robinson, pers. comm.).

In January, 1975, a study was initiated in the South Texas Plains to determine sources of mortality to flocks of Angora goats grazed on rangeland. Coyote (Canis latrans) predation and meningeal worm infection were the major causes of death. The purpose of this paper is to describe the prevalence, clinical signs, gross pathology and histopathology of P. tenuis in a naturally-infected population of Angora goats.

MATERIALS AND METHODS

In January, 1975, and February, 1976, 211 and 204 adult Angora nannies, respectively, were placed on two experimental pastures in northern Zavala County, Texas. The goats were individually marked with numbered "punch-through" ear tags and drenched with levamisole HCl for internal parasites prior to release. Each flock was checked 5-7 times per week during the two study periods until the goats were removed in late June, 1975, and late July, 1976. Data on morbidity were maintained for each goat. When carcasses were found, the major organs were examined for gross lesions and the general condition of the individual was noted.

Seven nannies showing clinical signs of neurologic disease were taken to the Texas Veterinary Medical Diagnostic Laboratory, College Station, Texas, for detailed study. Five of these were electrocuted and examined at necropsy. The brain and spinal cord (CNS) were removed and selected tissues from all organs were placed in 10% phosphate...
buffered formalin. Gross examination of the CNS was done after 4-5 days fixation. Serum and unclotted blood (using EDTA) were taken upon arrival and prior to euthanasia.

Tissues were routinely processed, embedded in paraffin, sectioned at 5-6 μm (some CNS sections were 10-12 μm) and stained with hematoxylin and eosin.

RESULTS

Prevalence

Clinical signs of neurologic disease, manifested as posterior paresis, first appeared in a nanny on 1 April 1975, 60 days after the flocks were introduced to the experimental pastures. Subsequently, 13.6% of 206 adult goats developed slight to severe clinical signs of meningeal worm infection. Of 28 presumably infected nannies, 11 died in the field, 10 survived to 27 June when they were sold and 7 were removed for diagnostic examination.

In 1975 prevalence of infection and mortality varied dramatically between two flocks, which were pastured 7 km apart. In one pasture, 22% of 103 goats were infected and 9.7% died. In the second the figures were 4.8 and 1.0%, respectively.

In 1976, 1 of 204 goats showed clinical signs of meningeal worm infection and none died. The different prevalence between years possibly resulted from different densities of the gastropods that serve as intermediate hosts for P. tenuis. Considerable rainfall in 1975 favored snails, transmission of the disease, and dispersion of larvae, whereas 1976 was a dry year.

Neurologic disease was the major source of mortality among adult Angora goats in 1975, accounting for 11 of 17 (65%) deaths. Coyotes killed three goats and three were lost to undetermined causes. Kids did not show signs of neurologic involvement with P. tenuis either year.

Clinical Signs

The clinical signs shown by goats infected with P. tenuis were similar to those described for other incompatible hosts: lack of fear, lethargy, paraplegia, posterior incoordination, general weakness, posterior paralysis (Fig. 1), drooping ears and head held at unusual angles. When the head was moved to the normal position, it immediately was rotated back to an abnormal angle. The signs differed somewhat among nannies; probably this was because clinical effects of meningeal worm vary with the activity, longevity and position of the nematode in the CNS. Prehension was normal except when a goat became severely paralyzed.

Although Angora goats have a strong flocking instinct, those infected by P. tenuis frequently became separated from the flock. They usually stood in a "humped up" position and ran with a spastic gait. Some nannies weakened by

FIGURE 1. Angora nanny with posterior paralysis.
the infection became entangled in thorny brush and died.

Partially paralyzed nannies scraped saucer-shaped areas with their hind feet when struggling to arise. An accumulation of fecal pellets was present at these sites. These circumstances suggested P. tenuis infection when a nanny disappeared with no record of clinical signs, but whose decomposed carcass was found later.

The time between onset of clinical signs and death varied considerably among individuals. In two cases, nannies lacking clinical signs one day were paraplegic the next day. One nanny showing severe neurologic impairment of her hind quarters survived in the field for 57 days. Two that were moderately affected apparently underwent complete remission of clinical manifestations after being removed from the field. Such remission has occurred in naturally infected sheep.\(^{2}\)

Gross Pathology

Field necropsy of four nannies by Guthery did not reveal any gross pathologic conditions. All nannies apparently were in excellent condition with substantial kidney, heart and omental fat deposits. Ample food was present in the rumens and the feces appeared normal.

The five goats examined at necropsy also were in good nutritional condition. Three had grossly evident areas of cavitation, malacia and/or hemorrhage in various parts of the spinal cord. One had lesions in the medulla, pons and ventral portions of the midbrain. One had several enlarged upper cervical and mediastinal lymph nodes. These nodes contained small pockets of yellowish-green, caseous material from which Corynebacterium pseudotuberculosis was cultured.\(^{3}\) Other gross lesions were not observed.

Histopathology

CNS lesions in all five goats were similar in type and pattern, with random areas throughout the brain (especially the stem) and spinal cord consisting of malacia with large clusters of gutter cells (Fig. 2). Often, immediately adjacent to the malacic areas the blood vessels were lightly cuffed with lymphocytes, occasional eosinophils and, more rarely, plasma cells. Degenerating and/or swollen axons, particularly in ventral and ventrolateral columns, were evident within sections of affected spinal cord. Nematodes (Fig. 3) rarely were observed and seldom were they surrounded by an inflammatory cellular infiltrate. Lesions in other organs and tissues were minimal or not remarkable.

The morphologic changes in the CNS of Angoras were similar to other cases involving metazoan parasites and parasitic migration through the CNS.\(^{1,4,16}\)

No ova resembling those of P. tenuis were found in lungs or meninges, nor were cross sections of gravid parasites observed.

Hemograms (leucocytes, erythrocytes, hematocrit, hemoglobin, differential) were essentially normal in all goats. In two goats whole-blood levels of acetylcholinesterase were within the normal range.

DISCUSSION

To our knowledge a natural infection of Angora goats in Texas by P. tenuis has not been confirmed previously. Natural infections have occurred in other breeds in New York.\(^{2}\) Spanish goat kids orally infected with third-stage larvae suffered high mortality from necrotizing colitis and peritonitis associated with migration of the nematodes.\(^4\) Kids injected intraperitoneally with infective larvae

---

\(^{2}\) Identification by Dr. Howard Whitford, Texas Veterinary Medical Diagnostic Laboratory, Drawer 3040, College Station, Texas 77843, USA.
FIGURE 2. Medulla oblongata showing areas of malacia (1) and degenerating axons (2). ×65.

FIGURE 3. Cross sections of Parelaphostrongylus tenuis. ×65.
showed clinical effects ranging in severity from mild posterior ataxia to general paralysis.¹⁰

Some mortality of goats in portions of South Texas historically attributed to other sources probably has been caused by meningeal worm. Two enzootic poisonous plants, guajillo (Acacia berlandieri) and coyotillo (Karwinskia humboldtiana), produce clinical signs resembling neurologic disease. Death attributed to coyote predation also could have been an indirect result of neurologic disease. Abnormal behavior in goats, such as separating from the flock, predisposes them to coyote predation.¹⁵

Little is known about P. tenuis in Texas, although it occurs in restricted areas in eastern and southern portions of the state. A comprehensive study of meningeal worm’s distribution, ecology and potential for range expansion into important Angora goat production areas seems warranted.

Acknowledgements
We thank Dr. D. B. Pence for critical review of the manuscript. J. Williams and D. Nobles assisted in field data collection. Mr. and Mrs. J. Lee granted permission to conduct the study on their ranch.

LITERATURE CITED

Received for publication 8 June 1978