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EPIZOOTIOLOGY OF AN OUTBREAK OF CEREBROSPINAL NEMATODIASIS IN COTTONTAIL RABBITS AND WOODCHUCKS

H. A. JACOBSON,[1] P. F. SCANLON,[1] V. F. NETTLES[2] and W. R. DAVIDSON[2]

Abstract: An epizootic of cerebrospinal nematodiasis in cottontail rabbits (*Sylvilagus floridanus*) and woodchucks (*Marmota monax*) caused by *Baylisascaris procyonis* larvae followed the establishment of an ascarid-infected raccoon (*Procyon lotor*) population in a woodlot. Five of seven raccoons examined from the woodlot harbored ascarids, with one heavily infected animal shedding approximately 27,500 eggs per gram of feces. A laboratory-reared cottontail rabbit developed neurologic disease due to larval migration 80 days after infection with *B. procyonis* eggs from the raccoons.

INTRODUCTION

Recently, we described an epizootic of cerebrospinal nematodiasis in cottontail rabbits (*Sylvilagus floridanus*) caused by ascarid larvae, presumably of raccoon (*Procyon lotor*) origin.[5] The epizootic began during the winter of 1974-75 when 16 of approximately 60 cottontail rabbits captured in a woodlot (Center Woods) had signs typical of neurologic disease. The present report represents epizootiologic studies following the outbreak, including demonstration of the helminth in the definitive host, laboratory transmission of the agent, and additional occurrences of the disease.

HISTORY

Raccoons, which are considered the definitive host for *Baylisascaris procyonis*,[3,7,8] were not known to inhabit the woodlot prior to 1970 (Henry S. Mosby, Department of Fisheries and Wildlife Sciences, Virginia Polytechnic Institute and State University, Blacksburg, personal communication). During the summer of 1970, raccoons were first noted when they caused damage to a vegetable garden 1 km from Center Woods. Thereafter, they frequently were observed in the woodlot and surrounding areas. In winter 1974, three raccoons were removed from the woodlot in response to depredations on deer feeders in the research enclosure. When depredation occurred again in June, 1975, a second trapping effort was initiated. Fifteen raccoons were removed during July and August, 1975. During this same period, three additional raccoons were killed by automobiles on the bordering highway.

Following the initial outbreak, additional cases of neurologic disease were observed in animals within the woodlot and from nearby areas. Two of 12 cottontail rabbits live-trapped in April, 1975, 2 km from the woodlot on the Virginia Polytechnic Institute and State University campus developed signs typical of the neurologic disease.[5] Three woodchucks (*Marmota monax*) were observed with neurologic disease in the woodlot, two in July and one in October, 1975. They had symptoms similar to those previously reported,[6,9] and were easily caught by hand. An additional sick woodchuck was observed on the university golf course 2 km from the woodlot in June, 1975.

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MATERIALS AND METHODS

Six raccoons trapped in August, 1975, from Center Woods were examined for ascarids. Necropsy was conducted on three of these and feces of the remaining three were examined for ascarid eggs by fecal floatation. One raccoon killed on the highway adjacent to the woodlot also was obtained for necropsy. Prior to the killing of one raccoon, ascarid egg production was recorded for a 10 day period by the Stoll dilution technique.[1]

Approximately 100 ascarid eggs obtained by fecal sedimentation from the aforementioned raccoons were placed on an apple slice and fed to a pen-raised juvenile cottontail rabbit. Three days following onset of clinical signs, the rabbit was killed and a necropsy performed. The brain, spinal cord, and tissues having gross lesions were examined histologically.

The three sick woodchucks from the woodlot were killed and examined for gross lesions. The brains and spinal cords were examined histologically for the presence of ascarid larvae and associated lesions.

RESULTS

At necropsy, three of the four raccoons were found to harbor adult *B. procyonis*. Ascarid burdens ranged from 17 to 93 worms per raccoon. The raccoon with 93 *B. procyonis* (49 females, 44 males) was shedding 25,750 (±3,912 S.E.) eggs per gram of feces. Two of the three raccoons examined by fecal floatation also were shedding large numbers of ascarid eggs.

On the 80th day post-infection, the juvenile cottontail rabbit suddenly began showing neurologic signs of disease. Clinical signs included: head tilt, torticollis, and extreme hypersensitivity to external stimuli. Ascites and hepatomegaly were evident at necrospy. Liver sinuses were engorged and capsular fibrosis was prominent. Histological examination revealed severe hepatic fibrosis with hemorrhage and regenerative areas distributed

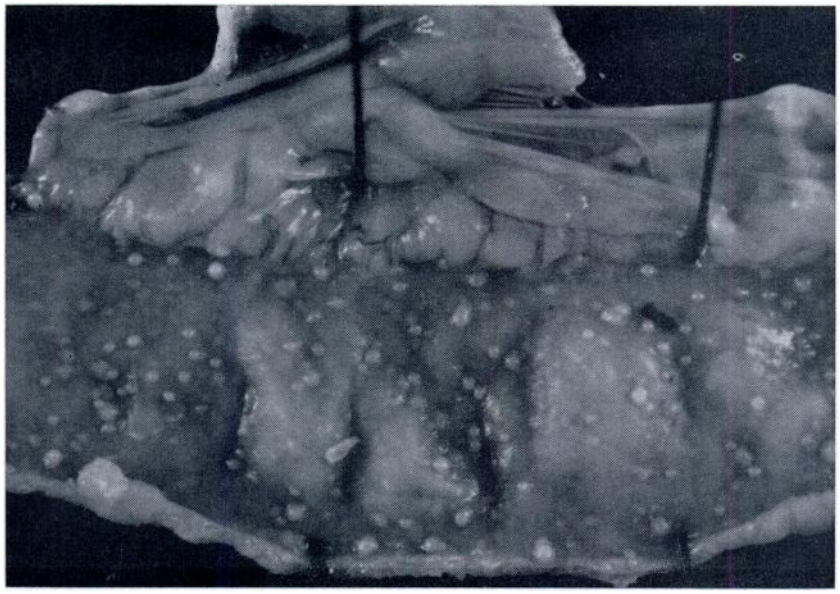


FIGURE 1. Numerous granulomatous nodules containing larvae of **Baylisascaris procyonis** in the small intestine of a woodchuck.

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throughout the organ. Histologic examination of the brain revealed one ascarid larvae 62 μm in diameter in the thalamus. Ascarid-induced lesions, as previously described,[2,5] were quite severe in the white matter throughout the brainstem. Lesions were not present in the spinal cord.

Gross lesions were evident in only one woodchuck. The affected animal had several thousand granulomas which contained ascarid larvae distributed throughout the length of the small intestine (Fig. 1). Parasitic granulomas also were present in the liver. The brains of all three woodchucks contained ascarid larvae 48 to 62 μm in diameter accompanied by typical lesions.[6,9]

DISCUSSION

The raccoon ascarid was previously reported as *Ascaris columnaris*[5] It has since been noted that the correct designation for this parasite is *Baylisascarsis procyonis*.[3,7,8] The demonstration of larvae, lesions, and a neurologic syndrome in the experimentally infected rabbit, which were consistent with previous observations, confirmed that *B. procyonis* was indeed the etiologic agent. That woodchucks, in addition to rabbits, were affected was not surprising since there have been previous reports of this disease in these animals.[6,9]

Circumstances surrounding the epizootic indicate that it was initiated by colonization of the woodlot by *B. procyonis* infected raccoons, since neurologic signs were not observed in several hundred rabbits trapped on the area over the preceeding years.[5] The population density of raccoons was extremely high as 18 raccoons (15 trapped, 3 highway mortalities) were recovered from the 36 ha area. A density of 1 raccoon per 4 ha is considered high.[4] The high percentage of infected raccoons in the population, coupled with the capacity of *B. procyonis* to produce large numbers of eggs, suggest that the woodlot was heavily contaminated. The massive natural infection observed in one woodchuck tends to support this contention.

As noted previously,[5] the ramifications of neurologic disease due to *B. procyonis* in small mammals are not completely understood; however, with raccoon population densities and infection densities at the level observed, clearly losses can be significant. Considering the potential consequences of this disease in small mammal populations, it appears warranted to recommend that raccoons be examined for ascarid infection prior to relocation. Furthermore, the public health aspect of *B. procyonis* larval migration should be explored.

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