Parasitic Encephalitis Due to *Baylisascaris* sp. in Wild and Captive Woodchucks (*Marmota monax*)

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Abstract. Twelve of 102 woodchucks submitted for necropsy to the New York State College of Veterinary Medicine had clinical histories of central nervous system disorder. All had multifocal eosinophilic encephalitis with formation of granulomas. Nematode larvae, identified as *Baylisascaris* sp. were found in six of the 12 and were assumed to be the etiologic agent in all affected woodchucks. Both wild and captive (wild-trapped) woodchucks were affected, and all occurrences were between mid-June and early September.

The woodchuck, *Marmota monax*, is a common rodent in North America, having an extensive normal range that extends northeasterly into Nova Scotia, along the Atlantic coast southward into Georgia, westward into Missouri, and north and west into Canada and Alaska [5]. Although it lives in proximity to man, the woodchuck is generally a wary creature, and no direct contact between man and free-ranging woodchucks usually is encountered. The woodchuck, however, has gained increased importance for use as a laboratory animal for biomedical research in such areas as acute and chronic viral hepatitis, hepatic neoplasia, adipose tissue metabolism, and endocrine regulatory mechanisms [17]. The majority of these laboratory woodchucks are originally wild-caught. Deviations from normal behavior are obvious and may be expressed as increased tameness or viscousness. These behavioral changes often are associated with increased human contact and bite or scratch wounds are suffered occasionally by those people involved. Many woodchucks subsequently are examined for rabies exclusively, and this yields no definitive diagnosis [3, 10, 14] when the rabies diagnosis is negative.

This report describes encephalitic nematodiasis due to the migration of aberrant parasitic larvae in both wild and captive woodchucks which have been submitted to the New York State College of Veterinary Medicine for necropsy examination (table I).
Table I. Signalment and history of affected woodchucks

<table>
<thead>
<tr>
<th>Affected woodchuck number</th>
<th>Age</th>
<th>Sex</th>
<th>Wild or captive</th>
<th>Parasite found</th>
<th>Primary clinical complaint</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>J</td>
<td>F</td>
<td>Wild</td>
<td>−</td>
<td>Chasing people on college campus</td>
</tr>
<tr>
<td>2</td>
<td>J</td>
<td>F</td>
<td>Wild</td>
<td>+</td>
<td>Found in yard unable to use hindlegs</td>
</tr>
<tr>
<td>3</td>
<td>J</td>
<td>M</td>
<td>Captive</td>
<td>−</td>
<td>Head tilt, seizures, depression</td>
</tr>
<tr>
<td>4</td>
<td>J</td>
<td>M</td>
<td>Captive</td>
<td>+</td>
<td>Head tilt, rolling to side, unable to stand</td>
</tr>
<tr>
<td>5</td>
<td>A</td>
<td>F</td>
<td>Wild</td>
<td>+</td>
<td>Found on back porch</td>
</tr>
<tr>
<td>6</td>
<td>A</td>
<td>M</td>
<td>Wild</td>
<td>−</td>
<td>Found in yard with family pets</td>
</tr>
<tr>
<td>7</td>
<td>A</td>
<td>F</td>
<td>Captive</td>
<td>−</td>
<td>Depressed, anorexic</td>
</tr>
<tr>
<td>8</td>
<td>J</td>
<td>F</td>
<td>Captive</td>
<td>−</td>
<td>Head tilt, uncoordinated</td>
</tr>
<tr>
<td>9</td>
<td>A</td>
<td>F</td>
<td>Captive</td>
<td>+</td>
<td>Circling, uncoordinated</td>
</tr>
<tr>
<td>10</td>
<td>J</td>
<td>F</td>
<td>Captive</td>
<td>−</td>
<td>Head tilt, uncoordinated</td>
</tr>
<tr>
<td>11</td>
<td>J</td>
<td>F</td>
<td>Wild</td>
<td>+</td>
<td>Acting strangely, head tilt</td>
</tr>
<tr>
<td>12</td>
<td>J</td>
<td>M</td>
<td>Wild</td>
<td>+</td>
<td>Head tilt, circling, nystagmus</td>
</tr>
</tbody>
</table>

J = juvenile; A = adult.

Case Histories

Since August, 1978, 12 of 102 woodchucks have been submitted for necropsy with histories of unusual behavior or nervous signs, and all occurred between mid-June and early September. There was no apparent age or sex predilection. Typical histories included “Found chasing people on college campus,” “Came to back porch and was not afraid of people,” or “Bites at metal strips in cage.” Clinical signs included head tilt, circling, ataxia, incoordination, depression, seizures, voracious appetite, or anorexia. Six captive woodchucks had clinical signs noted within two days to two months of captivity. The remaining six woodchucks were non-captive. The captive woodchucks were killed by injection and the non-captive woodchucks were killed by blunted cerebral trauma or gun shot.

Materials and Methods

Complete necropsies were done on all woodchucks. One longitudinally sectioned half of each brain of eight of the woodchucks (all of the non-captive and two of the captive woodchucks) were submitted for immunofluorescent rabies examination. All were negative. The remaining portion of brain and sections of other major organs including liver, kidney, spleen, pancreas, lung, and heart were fixed in 10% neutral buffered formalin, embedded in paraffin, sectioned at 4 to 6 μm, and stained with hematoxylin and eosin (HE) for light-microscopic examination.

Results

No specific gross lesions other than those incurred at death were observed at necropsy in all woodchucks.

Histologic lesions of multifocal to locally extensive and severe, subacute or chronic eosinophilic encephalitis were observed consistently in the brain. Lesions were distributed throughout the brain, but were observed most frequently in the cerebral
Fig. 1: Characteristics of mammalian ascarid larvae [1]: distinct lateral alae, paired excretory gland cells enclosed in lateral cords; intestine with few uninucleated intestinal cells per diameter within a typical focus of inflammation and necrosis surrounded by eosinophils and glial cells.

Fig. 2: Larvae; rounded anterior end and tail which end in a round knob bent dorsally. Esophagus uniform in diameter, ending in bulb. Distinct lateral alae, near anterior end of esophagus to near the level of anus. Measurements (average) in μm: total length, 1200; greatest width, 63; length of esophagus, 191; distance of nerve ring from anterior end, 91; and distance from anus to tail tip, 39. Bar = 100 μm.

cortex and midbrain. Affected areas consisted of foci of malacia, with or without hemorrhage, large aggregates of eosinophils with fewer neutrophils and macrophages, glial cell proliferation, and occasional multinucleated giant cells. Within scattered foci, sections of characteristic mammalian ascarid larvae [1] were observed in six of
the 12 woodchucks (fig. 1). Similar lesions without sections of parasites were observed in the other six woodchucks. There were large perivascular aggregates of eosinophils with fewer neutrophils. The meninges appeared normal unless they were in close proximity to focal parenchymal lesions, in which instance there was focal eosinophilic meningitis.

Lesions were observed in the heart and the lung and were similar to those in the brain. They consisted of focal granulomas composed of eosinophils, mononuclear cells, multinucleated giant cells, and proliferating fibroblasts. Sections of nematode larvae often were present within these granulomas.

The remaining formalin-fixed tissues were compared with sections on the glass slides, and appropriate tissues were selected for dissection to obtain intact parasites for genus identification (fig. 2). Measurements of morphologic features such as total length, greatest width, length of esophagus, distance of nerve ring from anterior end, and distance from anus to tail tip correlated with those of *Baylisascaris* sp. [1, 12, 13].

**Discussion**

Encephalitis due to ascarid larval migrations in aberrant hosts has been reported in both experimental animals [2, 7, 11, 14] and naturally occurring outbreaks [3, 4, 6–10, 16]. Although various species have been involved as aberrant hosts in the naturally occurring disease, *Baylisascaris* sp. larvae have been identified as the etiologic agent, where a specific parasite could be identified. The skunk is the usual definitive host for *B. columnaris* and the raccoon is the usual host for *B. procyonis*. Contact with contaminated raccoon or skunk feces could be proven in outbreaks involving flocks of domesticated avian species [6, 9, 16]. One must assume that woodchucks and other wild animals are exposed readily to the parasite-contaminated excrement of raccoons and skunks. Skunks, cottontail rabbits, foxes, weasels, and chipmunks have been observed to occupy woodchuck burrows, either to seek protection from the elements or predators or to raise their young [5].

Physical measurements of larval features in affected woodchucks included in this report and literature reports on experimentally induced nematodiasis in woodchucks implicate *B. columnaris* or *B. procyonis* as the cause of this disease. *B. procyonis* has been considered the more pathogenic of the two for rodents [3, 15].

All twelve woodchucks in this report were affected between mid-June and early September. It has been suggested [3] that the seasonal incidence of this disease may be attributed to a combination of the seasonal activity of woodchucks, to the greater probability of woodchucks being observed by man in the summer months, and to a seasonal peak in ascarid egg production in the raccoon or skunk.

Attaining a definitive diagnosis in woodchucks with nervous signs has increased importance as the woodchuck emerges as an important laboratory animal and is required for controlled studies. These laboratory woodchucks often are trapped in the wild and the probability of human exposure has increased markedly. Frequently no basis for this unusual behavior is diagnosed as immunological tests for rabies...
rarely are positive and no tissue remains for further diagnostic tests. Histologic examination of brain tissue or examination of Baermannized brain tissue [3] can provide a definitive diagnosis in most instances and relieve concern about rabies.

References


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