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Source: Journal of Wildlife Diseases, 13(2) : 114-116

Published By: Wildlife Disease Association

URL: https://doi.org/10.7589/0090-3558-13.2.114

TYZZER'S DISEASE IN WILD-TRAPPED MUSKRATS IN BRITISH COLUMBIA

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Abstract: A diagnosis of Tyzzer's disease (*Bacillus piliformis* infection) was made in a group of recently-trapped muskrats (*Ondatra zibethica*). The major lesions consisted of enteritis and focal necrosis of the liver, with organisms resembling *B. piliformis* in hepatocytes on the periphery of these foci. The outbreak was associated with recent capture, housing, and the use of sulfaquinoxaline-medicated feed.

INTRODUCTION

Tyzzer's disease, an enterohepatic infection caused by *Bacillus piliformis*, a pleomorphic Gram-negative, spore-forming rod, has been associated mainly with infections in laboratory mammals. In recent years it has been reported in domestic cats,[7,11] in dogs,[8,10] in horses[3,4,5,9,12] and in muskrats.[6] This paper describes further cases of Tyzzer's disease in wild-trapped muskrats and emphasizes the possible association of the outbreak with certain laboratory management practices.

HISTORY AND LABORATORY EXAMINATIONS

A group of 12 muskrats was live-trapped in the Delta area of the Fraser Valley, British Columbia. At the time of capture, animals were free of injuries and appeared to be in excellent condition. The muskrats were delivered to the University of British Columbia and were housed in the Biological Sciences animal facility in a previously cleaned, heavy sheet metal tank measuring 4.6 m x 1 m x 1.5 m. A water level of approximately .75 m was maintained in the tank, with a nesting-rest area provided by an access port at one end. Water was obtained from Vancouver city water mains and flowed constantly through the tank with no recycling. The muskrats were not in contact with other animals in the facility and were observed six to eight times daily. Feed consisted of unmedicated commercial rabbit pellets, alternating every other day with pellets containing 0.03% sulfaquinoxaline for the prevention of coccidiosis, a disease which had occurred in a previous group of muskrats.

The first loss occurred on post-capture day 5, and by day 21, eight of the 12 animals had died. Four surviving muskrats appeared normal and healthy at all times. In affected animals, a brief clinical course of illness was characterized by depression and a sudden display of bleeding from the anus and preceded death by 12 to 24 hours. Only three of eight animals, a male dead on day 5 and two females dead on days 14 and 21 respectively, were submitted to the laboratory for necropsy. Fecal staining of the perineum was a prominent gross finding. Internally, tissues were paler than normal and except for the liver and cecum, abdominal organs appeared to be normal. Numerous 1-2 mm white to yellow foci were present in the liver. The cecum had slight edema with hyperemia of the serosal surface. The lumen of both the cecum and colon contained very fluid, brown fecal material with hemorrhage and edema of the mucosa.

Bacteriologic examination of tissues from the two females yielded *Citrobacter*

freundi from the lung and liver and *Streptococcus pyogenes* from the liver of one animal; *Bordetella bronchiseptica, Providencia stuartii* and *Pseudomonas aeruginosa* were isolated from the liver of the other animal. Intestinal contents of both animals were negative for *Salmonella* species.

Fecal smears and flotations were negative for coccidia and for the ova of gastrointestinal helminths; no adult or larval stage nematodes or cestodes were observed during gross examination.

No chicken-embryo lethal agents were recovered from 10-day-old embryos inoculated via the chorioallantoic membrane, allantoic sac and yolk sac, with 0.2 ml of a 15-20% homogenate of lung, liver and kidney in phosphate buffered saline containing crystalline penicillin G, streptomycin sulfate and neomycin sulfate.

Routine screening tests for arsenic, antimony, bismuth, silver and sulfur, and a qualitative test for lead, were uniformly negative.

Sections of liver and kidney were fixed in 10% neutral buffered formalin, processed routinely, cut at 6 μm and stained by the hematoxylin and eosin, Giemsa and Warthin-Starry[13] methods.

Histologic lesions were observed only in liver and consisted of numerous foci of necrosis containing much cellular debris, karyorrhectic nuclei, erythrocytes and fibrin with little or no concurrent inflammatory reaction. Organisms resembling *B. piliformis* were best appreciated when stained by the Warthin-Starry method, although they could be discerned by careful examination of Giemsa-stained sections. Long, thin, beaded filamentous organisms could be seen in hepatocytes on the periphery of necrotic foci, as individuals or as clumped groups, often criss-crossing in matchstick fashion. Organisms were observed only in scant numbers.

DISCUSSION

Gross lesions in the liver and in the intestinal tract resembled those of Tyzzer's disease, as reported previously for muskrats;[6] histologically, hepatic lesions were similar to those reported for muskrats[6] and for other species infected with *B. piliformis*.[1-5,7-12] The demonstration of organisms resembling *B. piliformis* in hepatocytes on the periphery of necrotic foci provided further confirmatory evidence of the diagnosis; the organism was not, however, recovered by egg inoculation or other cultural methods. The use of antibiotics in the inoculum may have prevented the growth of *B. piliformis* in embryonated eggs, as observed by Ganaway *et al.*[1] who found a graded inhibitory effect on the organism by the use of streptomycin, penicillin, erythromycin and chlortetracycline. Although post mortem examinations were conducted on three animals only, it was assumed that the remaining five animals died as the result of *B. piliformis* infection, based on the appearance of similar clinical signs in those animals.

The history of the loss of muskrats from Tyzzer's disease associated with the use of sulfaquinoxaline-medicated feed bore notable similarity to the loss, from the same disease, of laboratory rabbits fed sulfonamides, as reported by Ganaway *et al.*[1,2] These investigators observed retrospectively that an outbreak of Tyzzer's disease had occurred in laboratory rabbits given sulfonamides as a treatment for coccidiosis.[2] In a subsequent trial, all sulfaquinoxaline-treated rabbits died of Tyzzer's disease within 30 days, whereas non-treated animals, housed in the same room, remained healthy. In addition, cortisone-treated rabbits, given sulfaquinoxaline and raised on contaminated bedding, died of the disease in a more predictable pattern and with a shortened incubation period.[2]

In view of these observations, it is tempting to suggest that the combination of the effects of capture and housing, plus the use of sulfaquinoxaline-medicated feed on alternate days, may have precipitated the disease outbreak reported herein. *B. piliformis* has been found to be resistant to sulfamethazine as well as to chloramphenicol,[1] an antibiotic used unsuccessfully to treat in-contact muskrats during a previously reported

outbreak of Tyzzer's disease.[6] The early occurrence of Tyzzer's disease in recently-trapped muskrats housed in a clean tank which was provided with constantly flowing, non-recycled water, was consistent with the suggestion of Karstad *et al.*[6] that the animals may have been latently infected or were in the incubatory stage of the disease in their native habitat.

Acknowledgments

The authors wish to express their gratitude to Mr. Peter Drummond, Department of Zoology, University of British Columbia, for assistance in providing history and management details, to Dr. P. C. Simon for assistance with bacterial isolations and to Ms. Barbara Donaldson, Ms. P. Mills and Ms. V. Wong for excellent technical assistance.

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Received for publication 27 July 1976