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TYZZER'S DISEASE IN MUSKRATS: OCCURRENCE IN FREE-LIVING ANIMALS

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Abstract: Tyzzer's disease was diagnosed in four muskrats (*Ondatra zibethica*) found dead over a 2 month period in a single feed-house in a Saskatchewan marsh. No dead animals were found elsewhere in the marsh, although several hundred apparently healthy animals were trapped during this period. Similarities in the pathology and epizootiology of Tyzzer's and Errington's diseases of muskrats support an hypothesis that these diseases are a single entity.

INTRODUCTION

Tyzzer's disease (infection with *Bacillus piliformis*) has been described in recently trapped captive muskrats (*Ondatra zibethica*) in Ontario¹¹ and British Columbia.³ Karstad *et al.*¹¹ found a single dead muskrat in the area of origin of the Ontario animals. Hall and van Kruiningen¹⁰ referred to the diagnosis of this disease in a muskrat in Connecticut, but no details were provided. In addition to muskrats, a variety of domestic and laboratory animals are affected and serologic titres to the organism have been reported in cottontail rabbits (*Sylvilagus floridanus*) in Maryland.⁸ Tyzzer's disease recently has been reported in captive coyote (*Canis latrans*) pups treated with corticosteroids.¹⁵

This report describes the occurrence of Tyzzer's disease in free-living muskrats from a marsh in Saskatchewan, and discusses the relationship between Tyzzer's and Errington's diseases of muskrats.

HISTORY

On 17 December 1976 the skinned carcasses of three muskrats were submitted by a conservation officer to the

Department of Veterinary Pathology, University of Saskatchewan for examination. Two of the animals had been found dead by a trapper in a muskrat feed-house □ in Pike Lake, a small ox-bow lake located about 20 km from Saskatoon. The third animal was one of several apparently normal muskrats which had been trapped within the feed-house. The trapper had noted fresh blood passing from the rectum of the dead muskrats prior to skinning.

On 26 January 1977, the trapper found another dead muskrat at the same site, and the skinned carcass together with those of 12 apparently normal muskrats trapped in the general area of the marsh were submitted for necropsy.

On 17 February we visited the marsh with the trapper and recovered another dead muskrat from the same site.

During the winter the trapper opened many lodges and feed-houses on the lake and trapped several hundred muskrats. Dead animals were found only at the one site.

METHODS

Necropsies were performed and tissues were collected for histopathology and microbiology. Inocula from liver, lung,

□ A small structure composed of vegetation and mud with a small central chamber located over a plunge-hole in the ice.

spleen, and intestine were cultured aerobically at 37 C on blood and MacConkey agar, and on cysteine heart agar under 10% carbon dioxide for possible isolation of *Francisella tularensis*. A variety of tissues from the muskrats found dead in December was submitted to Dr. John Iversen, Department of Veterinary Microbiology, University of Saskatchewan for attempted isolation of *Chlamydia*.

Tissues for histopathology were fixed in 10% neutral buffered formalin, processed routinely, sectioned at 6 μ m and stained with hematoxylin and eosin (H&E). Selected sections were stained with Gram and Giemsa stains¹⁴ and by silver impregnation (Warthin-Faulkner technique¹).

PATHOLOGY

The four muskrats (two males, two females) found dead in the feed-house were in only fair body condition and had less body fat than did trapped animals from the same area. The most striking lesion was the somewhat dilated cecum, the serosal surface of which was extremely dark red-black. The lumen of the cecum was filled with a dark mixture of blood and ingesta, the wall was thickened by intra-mural hemorrhage and edema, and the mucosa was irregularly necrotic. The liver contained variable numbers of 1-3 mm white foci on both capsular and cut surfaces. The stomach of all animals contained food, and the small intestine was normal in three muskrats. In the fourth, the ileum was slightly dilated and the ingesta was more fluid than normal. In two muskrats the wall of the proximal spiral colon was involved similarly to that of the cecum and there was fresh blood in the terminal colon of three animals. Spleen and lymph nodes were normal, and the lungs were mildly congested.

Histologically, the hepatic foci were areas of coagulation necrosis with little or no inflammatory response. Small

numbers of thin, beaded filamentous organisms lying in parallel or criss-cross arrangement were within the cytoplasm of intact hepatocytes adjacent to the necrotic foci. These organisms were only faintly stained with H&E, did not stain with Gram stain, but stained basophilic with Giemsa and were impregnated with silver.

The mucosa of the colon was focally necrotic and ulcerated, with severe hemorrhage and edema of all layers of the wall. The walls of some submucosal arteries were undergoing necrosis and infiltration by inflammatory cells. With silver impregnation, a variety of bacterial types was visible within the lumen and in necrotic areas of the cecal wall. Large numbers of organisms morphologically similar to those in hepatocytes were found both in epithelial cells and free in the crypts, and lesser numbers were present within cells in the submucosa. The lungs were congested, with areas of alveolar hemorrhage.

MICROBIOLOGY

Small numbers of *Escherichia coli*, *Streptococcus* sp. and *Staphylococcus* sp. were isolated from most of the tissues submitted for culture from these animals. *F. tularensis* or *Chlamydia* were not isolated.

DISCUSSION

The gross and histologic lesions in the present cases were similar to those described in previous reports of Tyzzer's disease in this species.^{3,11} The striking resemblance of the lesions of Tyzzer's disease to those of Errington's disease of muskrats led Karstad *et al.*¹¹ to suggest that the diseases might be identical. Errington⁵ separated what he called the "haemorrhagic disease" into two categories: a pneumonic form "characterized by lung haemorrhages, with minor gross lesions of other sorts" which "seemed to occur much the same during one year-

grouping as another” and an enterohepatic form with “various combinations of liver necrosis and intestinal haemorrhages” which occurred in a highly variable manner from year to year.

The lesions of Tyzzer’s disease resemble those of the entero-hepatic form as described by Errington^{5,6} and by Lord *et al.*¹² and the pathology of these two conditions appears to be distinct from that of other common infectious diseases of muskrats. Focal hepatic necrosis has been described in tularemia,¹⁶ yersiniosis,²⁰ salmonellosis,² epizootic chlamydiosis¹⁸ and coccidiosis¹⁷ in muskrats. In the first three of these conditions the spleen and lymph nodes often contain necrotic foci, which have not been described in either Tyzzer’s or Errington’s disease. Hemorrhagic typhlo-colitis was not a feature of epizootic chlamydiosis of muskrats.¹⁸ Shillinger¹⁷ described “greyish-white granular areas in the liver and inflammation in the small intestine” in muskrats affected with coccidiosis; however, no histopathologic description was offered, the coccidia were not identified, and there was little evidence that the lesions, particularly those in the liver, were due to coccidia.

Lord *et al.*¹² described and illustrated intracellular spore-forming bacteria within hepatocytes adjacent to foci of necrosis in muskrats with Errington’s disease. These bacteria appear to be very similar to those now considered to be diagnostic⁷ for Tyzzer’s disease. Lord *et al.*¹³ suggested that an unidentified *Clostridium* spp. might be the causative

agent of this form of Errington’s disease, and had some success in reproducing the disease experimentally in captive muskrats with that agent. However, in view of the occurrence of spontaneous Tyzzer’s disease in captive muskrats, this experimental transmission using wild-caught muskrats should be re-evaluated. Muskrats placed in cages occupied previously by animals with Errington’s disease, developed that disease and died.¹³ Transmission by exposure to contaminated bedding has been shown to occur in Tyzzer’s disease in both mice^{9,16} and rabbits.¹

In the present case, dead muskrats were found at only one site in the marsh, but mortality occurred there over approximately a 2 month period. This focus of prolonged mortality was strikingly similar to the “deadly foci” of haemorrhagic disease described by Errington.^{5,6} In these foci, muskrats died consistently while muskrats lived securely in adjacent areas. Epizootics usually began at such foci. The occurrence of resistant spore forms and transmission by contact with contaminated bedding which are known to occur in Tyzzer’s disease might explain such a pattern. These similarities in epizootiologic pattern between the entero-hepatic form of Errington’s disease and Tyzzer’s disease in muskrats, together with the similarity of the lesions, strongly support the hypothesis¹¹ that these diseases are a single entity. Lesions resembling the pneumonic form of Errington’s disease have not been reported in muskrats with Tyzzer’s disease and this may be a distinct entity.

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