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TYZZER'S DISEASE IN MUSKRATS

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Abstract: An outbreak of Tyzzer's disease occurred in a colony of wild-caught muskrats. Deaths occurred suddenly and gross lesions were limited to focal liver lesions, and hemorrhages and necrosis in the cecum and colon. Bacilli typical of *Bacillus piliformis* were demonstrated in liver and intestinal lesions. Subsequently, Tyzzer's disease was diagnosed in two other muskrats from the same river.

In 1969, daths occurred in a group of wild-caught muskrats *(Ondatra zibethica)* housed in individual cages in the laboratory. They had been trapped at Guelph and at Hespeler, Ontario, points about 16 km apart on the Speed River, during the period October 10-17. Some of the muskrats had been experimentally infected with *Schistosomatium douthitti.*

On October 23 the first death occurred. By October 27, five of 22 muskrats had died. On gross pathological examination, a diagnosis of Errington's disease was made.[3] Four more muskrats died in the period November 4-6 and on November 6 chloramphenicol was added to the drinking water at a level of 10 mg/oz. for the remaining muskrats. Three muskrats died after chloramphenicol treatment was instituted, during the interval November 17 to 26. Six healthy muskrats remained at the end of the outbreak, which extended over a period of 35 days.

One muskrat trapped at Guelph on December 19 was housed in another building. This animal was found dead on December 28.

On March 18, 1970 a dead muskrat was found at the confluence of the Speed and Grand Rivers at Preston, a further 16 km downstream from Hespeler. Pathological and bacteriological studies provided evidence of common etiology in all of these cases.

Clinical Signs

Premonitory signs were seldom noticed. Sometimes the animals were noticed to leave food uneaten the day before death. Depression was noticed in some instances, immediately preceding death.

Pathology

Gross lesions consisted of focal yellowish areas, 1-2 mm in diameter, visible on the surface of the livers and scattered throughout the liver parenchyma, and hemorrhages in the walls of the intestines, particularly in the cecum and colon, associated with edema and patchy necrosis of the mucosa. The hearts, spleens, kidneys and adrenals appeared normal. Some of the lungs had patchy red consolidation. Stomachs were usually well-filled with ingesta (carrots, apples, lettuce and pelleted mouse diet — the laboratory diet) but the lower intestine often contained blood. Sometimes there was evidence of discharge of blood from the rectum.

Smears of intestinal contents were usually free of parasitic ova and oocysts.

Histological examination of sections from formalin-fixed and paraffin-embedded tissues revealed long, slender, beaded or banded bacilli in hepatic cells at the periphery of areas of coagulation necrosis (Figure 1) and in epithelial and smooth muscle cells in the hemorrhagic and necrotic lesions in the cecum and colon (Figures 2 and 3). These bacilli stained poorly with hematoxylin, were Gram-negative, and stained faintly with Giemsa. They were best demonstrated with the periodic acid-Schiff (PAS) technique, although even with PAS they

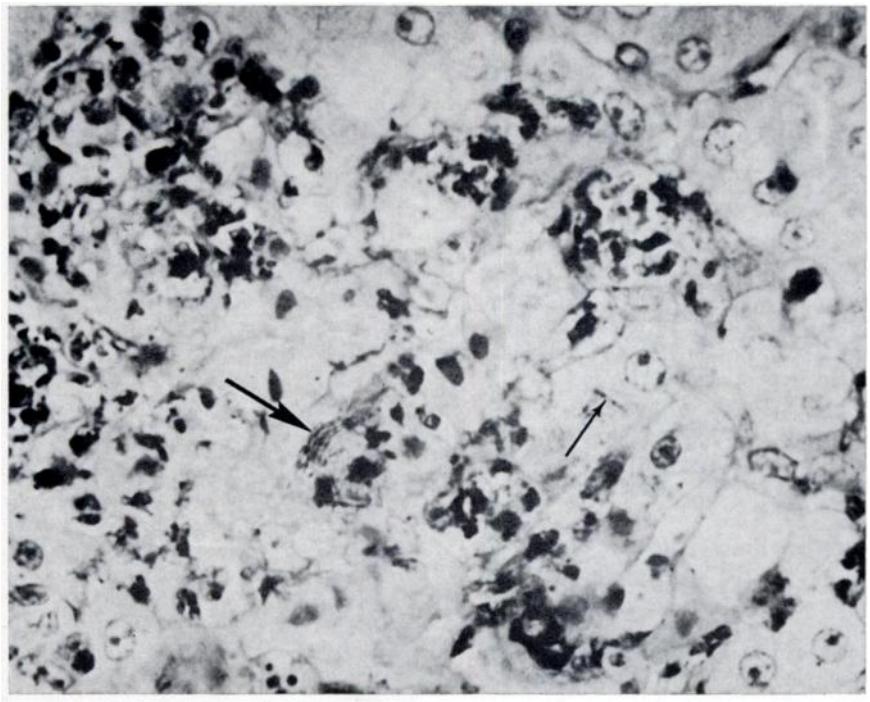

FIGURE 1. Muskrat liver. Slender beaded bacilli (arrows) are in the cytoplasm of hepatic cells at the edge of an area of necrosis. PAS-hematoxylin stain, X 800.

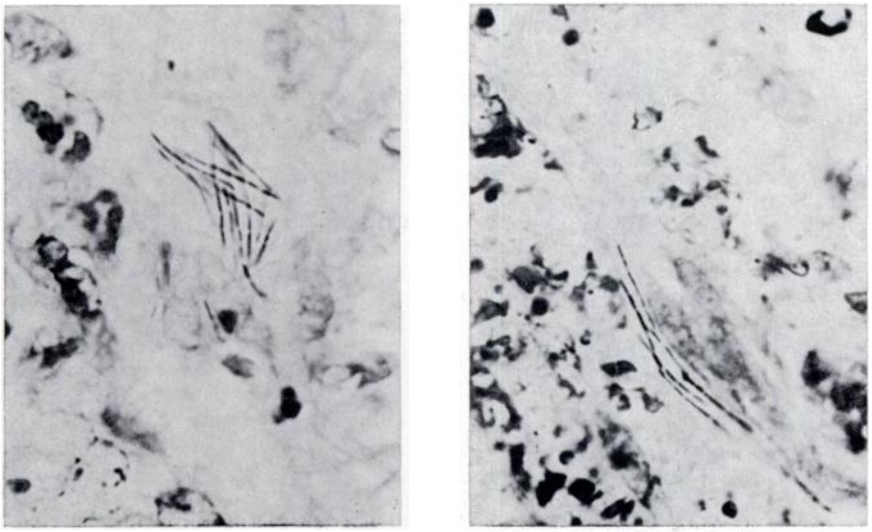

FIGURE 2 and 3. Muskrat cecum. Slender beaded or banded bacilli typical of **Bacillus piliformis** longitudinally or in criss-cross fashion in the cytoplasm of smooth muscle cells at the edg of an area of coagulation necrosis. PAS-hematoxylin stain, X 1500.

stained erratically and never uniformly PAS-positive. Some of the bacilli contained clear oval spore-like structures which appeared as empty vacuoles with PAS.

Intestinal lesions consisted of patchy acute necrosis of large areas of the mucosa, with necrosis extending in some cases into the muscularis mucosa and muscularis externa. These lesions were often associated with thrombosis of adjacent arteries and veins. Liver lesions were disseminated, their distribution near portal veins suggesting relationship to a noxious agent arriving from the gut via the portal circulation.

Lung lesions were variable, limited to congestion and edema. Lesions were not found in other organs sectioned; kidneys, adrenals, pancreas, spleen and heart.

Bacteriology

Blood agar and MacConkey's agar plates inoculated with wire loop scrapings from liver and intestinal lesions were incubated aerobically and anaerobically. Bacteria were not isolated from most of the livers. Livers from some muskrats which had been dead for several hours, and the intestinal cultures, yielded a variety of organisms; *Escherichia coli, Proteus* sp., and *Parcolon* organisms being the most common. None of these bacteria was grown consistently and none was considered to be responsible for deaths of the muskrats.

Suspensions of liver tissues from several muskrats were inoculated intraperitoneally, without antibiotics, into pairs of adult guinea pigs, without effect. Cysteine agar slants were inoculated with the same suspensions of liver tissues in unsuccessful attempts to isolate *Pasteurella tularensis.*

DISCUSSION

The presence in all liver and intestinal lesions of bacilli typical of *Bacillus piliformis,* coupled with failure to demonstrate the Gram positive *Clostridium* said to be the etiologic agent of Errington's disease, made us discard the latter diagnosis in favour of a diagnosis of Tyzzer's disease.[6] Absence of spleen lesions and the prevalence of intestinal lesions made infection with *Listeria monocytogenes, Pasteurella tularensis* and *Pasteurella pseudotuberculosis* seem to be less likely causes of death. Failure to culture these organisms and to infect guinea pigs, removed these diseases from further consideration. Virological studies were begun but not continued when organisms typical of *B. piliformis* were demonstrated consistently. Most attempts to culture *Bacillus piliformis* in artificial media have been unsuccessful.[2]

Tyzzer's disease has been described in mice,[6] rats,[5] gerbils and hamsters,[7] and rabbits.[1] To our knowledge this is the first report of its occurrence in muskrats. Since the first deaths occurred approximately 10 days after the muskrats were brought into captivity, and laboratory mice were being cared for by the same caretakers, it seemed possible that infection was picked up from a reservoir in latently infected mice. However, the occurrence of the disease in a muskrat trapped in the same river area and kept in isolation in another building, supported the idea that muskrats were infected in the wild. Some of the muskrats may have been latently infected or in the incubatory stages of the disease when they were trapped. The occurrence of Tyzzer's disease in free-living muskrats was confirmed when the last case was found 5 months later, in a muskrat picked up dead about 16 km downstream on the same river.

The possible efficacy of chloramphenicol in controlling this disease could not be confirmed.

Although several of the muskrats which died had been infected experimentally with *Schistosomatium douthitti,* this infection seemed to have no influence on the occurrence of Tyzzer's disease. In the absence of Tyzzer's disease, the schistosomes had no apparent effect on the health of the muskrats.

It is possibly significant that a diagnosis of Errington's disease was at first made. This disease, studied by Lord et al,[4] was believed to be caused by a spore-forming *Clostridium* organism. Although several previous cases of such a disease

in muskrats have been diagnosed as Errington's disease by one of us (Karstad), on the basis of hemorrhagic enteritis and focal liver lesions, it has never been possible to confirm the diagnosis by demonstration of a specific etiologic agent. In future cases of similar disease, a diagnosis of Tyzzer's disease will be considered, and attempts will be made to demonstrate *B. piliformis* in the lesions. Errington's disease and Tyzzer's disease may be one and the same.

Acknowledgement

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