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Omphalitis and Peritonitis in a Young West Indian Manatee (*Trichechus manatus*)

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ABSTRACT: Mortality data for the West Indian manatee (*Trichechus manatus*) indicates that from 1979 to 1984 16% of the recorded deaths involved young juveniles. Necropsy of a young manatee from the west coast of Florida revealed an active infection of the umbilical area (omphalitis) extending down the umbilical artery and veins. A generalized peritonitis was present. Bacterial cultures revealed *Streptococcus faecium*, *Plesiomonas shigelloides*, *Pseudomonas putrefaciens* and *Escherichia coli*.

Key words: *Trichechus manatus*, West Indian manatee, omphalitis, umbilical infections, peritonitis, case report.

The West Indian manatee (*Trichechus manatus*), an endangered species, ranges from the Carolinas in the United States to Brazil in South America. Less than 2,000 individuals inhabit the waters surrounding Florida, where the greatest numbers are concentrated in North America.

Part of the efforts to preserve the manatee in Florida involve identifying morbidity and mortality factors. Causes of reported mortality include intussusception (Forrester et al., 1975), toxoplasma encephalitis, gram negative bacterial encephalitis, red tide intoxication, hemorrhagic enteritis, severe pustular dermatitis and pneumonia (Beurgelt and Bonde, 1983; Beurgelt, 1984; Beurgelt et al., 1984). Approximately 16% of the manatee deaths recorded involve individuals less than 150 cm in length (Delaney et al., 1985). This group includes newborn animals and juveniles which are still dependent on maternal care. We examined one such animal, a young 139-cm 40-kg female manatee found at a river junction along the west coast of Florida in September 1985. It was

placed on ice and transported to Sea World of Florida for examination.

There were no signs of trauma present externally. A 3.0-cm piece of decaying umbilical tissue was still present which when removed had a caseous attachment. Skin measurements ranged from 0.6 cm ventrally to 1.5 cm laterally. The outer blubber layers (subcutaneous layer) measured 0.7 cm ventrally and 0.4 cm laterally. Inner blubber layers are separated from the outer layer by a layer of musculature. The ventral inner blubber layer was 0.5 cm thick and the lateral layer was 0.2 cm in thickness.

The abdominal cavity contained approximately 2 liters of serosanguinous fluid. Numerous fibrin tags were present on the serosal surface of the organs. The umbilical area, containing the residual umbilical vessels, and the urachus were grossly enlarged. The umbilical vein measured 1.8 cm in cross section and was filled with clotted blood, while the umbilical arteries were 1.0 cm in cross section.

The bladder was fusiform in shape with the urachus still present but not patent. There was very little urine and the bladder walls appeared grossly thickened. Approximately 6.0 cm caudal to the umbilicus the urachus was adhered in a 3.0-cm circular area to the underlying loops of bowel. Grossly, the bowel wall did not appear to be perforated. Aerobic cultures of the abdominal fluid grew *Streptococcus faecium*, *Plesiomonas shigelloides*, *Pseudomonas putrefaciens* and *Escherichia coli*. Anaerobic cultures also yielded *Streptococcus faecium*.

The stomach and small intestine were

empty and partially gas filled. Portions of the serosa of the jejunum and ileum were adhered to the urachus ventrally. The cecum and colon were distended with gas. There were early signs of liver decomposition such as the presence of small gas pockets in the tissue parenchyma. Gross lesions were not noted in the heart, adrenals, lungs or brain. There were no fractures present in the axial or appendicular skeleton.

Histopathologic evaluation of these tissues demonstrated acute, often severe, multiorgan inflammatory changes. The umbilicus and multiple peritoneal foci were characterized by extensive infiltrates of primarily eosinophils. The umbilicus also had considerable diffuse necrosis and hemorrhage. The liver was characterized also by marked diffuse infiltrates of eosinophils with focally extensive hepatocellular necrosis and congestion.

There was moderate multifocal acute inflammation of the renal glomeruli and moderate diffuse chronic to active inflammation of the small intestine. Numerous lymph nodes had moderate diffuse lymphoid depletion and congestion. The remaining organs were microscopically unremarkable. Special histochemical stains including Brown and Brenn, methenamine silver and giemsa did not elucidate the etiology of the above organ changes.

The cause of this manatee's death was probably a result of compromise to the function of the liver and other organs, and terminal septicemia. A possible portal of entry for a pathogenic organism in a neonate included the umbilicus. Umbilical infections (omphalitis, omphalophlebitis, "naval ill") have been implicated as a cause of bacteremia, septicemia, and joint disease in other species of newborn animals (Pederson et al., 1983; Elhasuani, 1984; Cushing, 1985).

A number of factors may have been involved in the development of omphalitis, including an inadequately developed immune system, the presence of an infectious

agent of increased pathogenicity or numbers in the surrounding environment, and an available route of pathogen entry. It cannot be ascertained if the animal in this report received adequate colostral protection, if the environment that the animal inhabited was abnormal such as a stagnant or polluted canal, or if there was some trauma to the umbilical cord at birth. The significance of the bacteria isolated from the abdominal fluid is questionable. The exact time of death was unknown so there is a possibility that some of the bacterial species may be opportunistic invaders or were species originating partially from the intestinal tract. While the origin of the bacteria found is difficult to pinpoint, it cannot be ruled out that some or all of the bacteria found may be involved in the early stages of the pathogenesis of the illness.

Representative pathologic specimens from this study are deposited in the Registry of Comparative Pathology (Armed Forces Institute of Pathology, Washington, D.C. 20306, USA; Accession Number 2091218-4). This paper is Sea World contribution number SWF-8510 F.

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BOOK REVIEW . . .

The Microsporidia of Vertebrates, E. U. Canning and J. Lom. Academic Press, Ltd., 24/28 Oval Road, NW1 7DX, England. 1986. 289 pp. \$64.00 U.S.

This reference text is designed to aid recognition of microsporidial parasites by nonspecialists and aid species diagnosis by specialists. Overall, the book is successful in achieving its goals. Although Canning and Lom worked together, Canning was overall editor and wrote Chapters 1, 3 and 4; Lom wrote Chapter 2 with Dykova; and Canning and Lom wrote Chapter 5. The text has more or less uniform style and content.

The volume is divided into five chapters. Chapter 1 (Introduction to the Microsporidia) describes the economic importance of microsporidia, and their general characteristics and development (i.e., diagnostic features, and meronts and merogony, sporonts and sporogony, sporoblasts, spores and transmission). This chapter provides definitions of all genera of microsporidia parasitic in vertebrates.

Chapter 2 discusses microsporidia of fish, beginning with a list of fish hosts. It would be useful to have references with this list. The next section describes pathogenicity of microsporidia on fish hosts at the cellular and tissue level both histologically and ultrastructurally. Species descriptions usually have the following format: Species Name, Synonyms (where applicable), Hosts (including type of environment), Geographical Distribution, Site of Infection (within host), Signs of Infection and Pathology, Structure and Life Cycle, and Remarks. Several species descriptions are augmented by some or all of the following: Organization of the Xenoma, Relation to Host Age, Development in the Central Region, Commercial Importance, Transmission, Seasonal Fluctuation of Infection, Host Specificity, Taxonomy, and Treatment.

Unfortunately, the format of Chapters 3 and 4 are not consistent with Chapter 2. Neither Chapter 2 nor 3 has a host list.

Chapter 3 describes microsporidia parasitic in amphibians and reptiles. Each species description (usually) had the following format:

Species Name, Synonyms, Hosts and Localities, Morphology, and Lesions. The above headings were sometimes supplemented by Structure and Life Cycle, Transmission, Notes, and Comment.

Chapter 4 describes microsporidial parasites of birds and mammals. A brief section on birds precedes the mammal section. The species format included Species Name, Host and Locality, Lesions and Signs, Morphology (and Development), and Notes. The description of *Encephalitozoon cuniculi* is extended by Transmission, Diagnosis, Pathogenesis, Mechanisms of Resistance in *E. cuniculi* Infections, Interaction of *E. cuniculi* with Concurrent Infections, Relation between *E. cuniculi* and Tumors, and Control and Therapy of *E. cuniculi*. The section on the medical importance of microsporidia would be more appropriate in Chapter 1, integrated with the section on economic importance.

Chapter 5 is a brief guide to methods used for handling and studying microsporidia. References to specialized techniques are provided. This "techniques" chapter should be moved up as Chapter 2.

Although the book is published by Academic Press and is expensive, the paper is of lesser quality than usual; numerous pages of the reviewer's copy were damaged. Also, the quality of the photomicrographs is uneven. The grouping of the micrographs causes the reader to constantly "flip ahead and back" to match pictures with the text. Numerous line drawings of good quality, including schematics of life cycles, are used throughout the book. There is an extensive Table of Contents and an extensive Index. Relatively few errors (i.e., typographical, terminology) were noted. The References include references published in 1986.

Overall, the volume is a well written and adequately illustrated reference text of microsporidial parasites of vertebrates. Both nonspecialists and specialists will find the book useful.

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