



THE HARP SEAL, *Pagophilus groenlandicus* (ERXLEBEN, 1777) XI. CONTRACAECUM SP. INFESTATION IN A HARP SEAL

Authors: WILSON, T. M., and STOCKDALE, P. H.

Source: Journal of Wildlife Diseases, 6(3) : 152-154

Published By: Wildlife Disease Association

URL: <https://doi.org/10.7589/0090-3558-6.3.152>

BioOne Complete (complete.BioOne.org) is a full-text database of 200 subscribed and open-access titles in the biological, ecological, and environmental sciences published by nonprofit societies, associations, museums, institutions, and presses.

Your use of this PDF, the BioOne Complete website, and all posted and associated content indicates your acceptance of BioOne's Terms of Use, available at www.bioone.org/terms-of-use.

Usage of BioOne Complete content is strictly limited to personal, educational, and non - commercial use. Commercial inquiries or rights and permissions requests should be directed to the individual publisher as copyright holder.

BioOne sees sustainable scholarly publishing as an inherently collaborative enterprise connecting authors, nonprofit publishers, academic institutions, research libraries, and research funders in the common goal of maximizing access to critical research.

THE HARP SEAL, *Pagophilus groenlandicus* (ERXLEBEN, 1777)

XI. CONTRACAECUM SP. INFESTATION IN A HARP SEAL

Case History

In May, 1969, an adult female harp seal, that had been in captivity for two months, was submitted for necropsy. There was no history of specific illness, however, terminal vomition of bile colored fluid, hemorrhage from the anus and convulsions were observed.

Pathological Findings

Gross lesions were confined to the alimentary tract. Masses of thin, pale worms, approximately 4 cm in length, were attached to the alimentary tract mucosa from the esophageal orifice to the rectum. The esophageal wall was markedly thickened and edematous with petechial hemorrhages throughout the mucosa. Individual parasites and clusters of parasites were attached to all portions of the hyperemic and thickened gastric mucosa. The small intestine contained blood and the large intestine contained melena. Traction on parasites embedded at all levels of the alimentary tract indicated that they were firmly attached to the mucosa.

Histological Findings

The esophageal mucosa was necrotic and replaced by an inflammatory exudate. Neutrophils, fibrin and cellular debris were present in the submucosa to the depths of the submucosal glands. Many superficial submucosal glands were dilated. There was necrosis of the glands and loss of acinar epithelial cells (Fig. 1). Edema and hemorrhage were present throughout the esophageal wall with edema most evident between the submucosa and internal muscular layer and

between the external longitudinal muscular layer and the serosa.

Histological examination of various areas of the gastric mucosa revealed similar lesions. The anterior ends of the parasites, associated with necrosis and inflammation of the surrounding gastric mucosa, were observed in both superficial and deep areas of the gastric mucosa (Fig. 2). A mixed leucocyte infiltration, consisting predominantly of neutrophils, and colonies of bacteria were present in the inflamed areas. Adjacent to the embedded parasites the gastric mucosa appeared structureless and uniformly eosinophilic. Penetration of the parasites deep into the gastric pits extended the inflammatory process into the deep submucosa. The small intestine had marked mononuclear leucocyte and eosinophil infiltration throughout the mucosa.

No significant findings were reported from bacterial cultures of the lung, spleen, liver, kidney, mesenteric lymph node and intestine.

Parasitological Findings

The nematode parasites from the esophageal, gastric and small intestinal areas of the alimentary tract were identified as immature specimens of *Contracaecum* sp.

Diagnosis

Verminous esophagitis, gastritis, enteritis and intestinal hemorrhage caused by *Contracaecum* sp. nematodes was considered to be the cause of death.

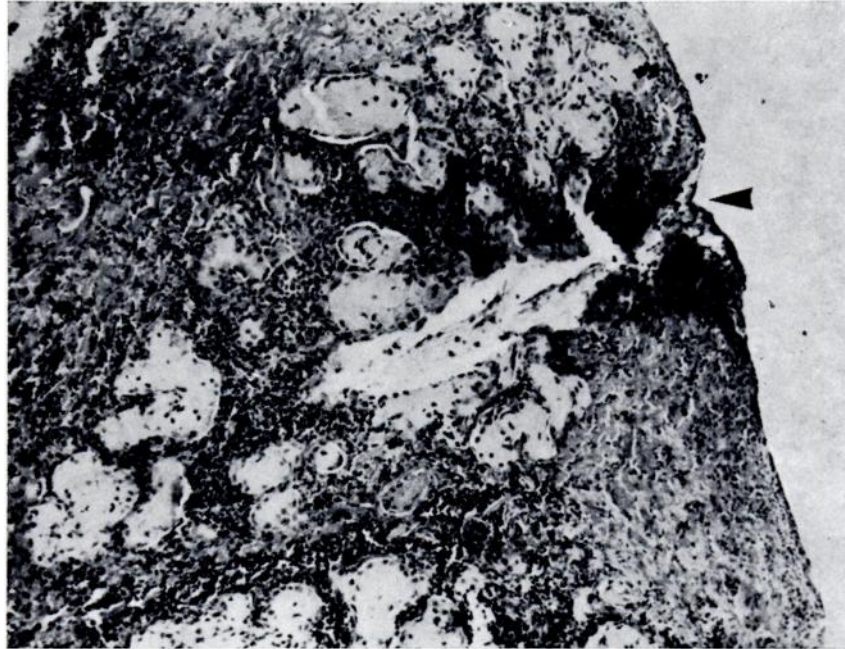


FIGURE 1. Esophageal wall illustrating loss of epithelium (arrow) and inflammation of the submucosa. H & E Stain: X 100

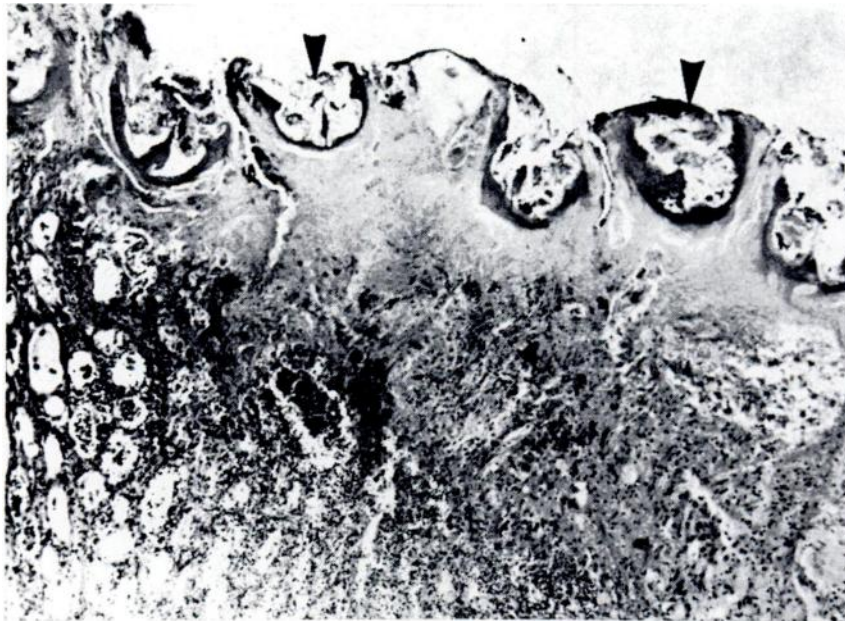


FIGURE 2. Necrosis and inflammation of gastric mucosa associated with parasitic (arrow) infiltration. H & E Stain: X 40

Discussion

Contracaecum sp. nematodes are common alimentary tract parasites in pinnipeds but little is known of their pathogenicity (Delymure, 1955. *Helminthofauna of Marine Mammals*. Academy of Sciences, USSR. p. 225-239). Heavy gastric infestations of wild harp seals by this parasite are common and considered by some authorities to be non-pathogenic (Personal communication, Ronald, K., University of Guelph, Guelph, Ontario, Canada).

Other reports, however, have consistently shown an association between *Contracaecum* sp. infestation in pinnipeds and gastritis and gastric ulceration. Schroeder and Wegeforth (1935, J.A.V.M.A. 87: 333-342) in a study of gastric lesions in a wide range of pinnipeds, reported the presence of *Contracaecum* sp. in most cases of gastritis and gastric ulcers. Young and Lowe (1969, J. Comp. Path. 79: 301-313) stated that *Contracaecum* sp. does cause severe gastric lesions in wild grey seals (*Halichoerus grypus*).

Their gross and histological descriptions of the gastric lesions are similar to those contained in this report. Recently Johnston and Ridgway (1969, J.A.V.M.A. 155: 1064-1072) reported marked gastric degeneration and ulceration in a California sea lion (*Zalophus californianus*) infested by *Contracaecum* sp. Keyes (1965, J.A.V.M.A. 147: 1090-1095) reported gastric ulceration in northern fur seals (*Callorhinus ursinus*) caused by *Contracaecum osculatum* to be common and suggested that mortality associated with this parasite might be significant.

Review of our necropsy files indicates that in post mortem examinations of 10 California sea lions and 11 harp seals, no *Contracaecum* sp. parasites were found in either esophagus or intestine. One sea lion and one harp seal each had fewer than twenty-four *Contracaecum* sp. worms in its stomach.

It is our opinion that more significance should be given to this parasite as a cause of disease in pinnipeds.

Acknowledgements

We acknowledge the assistance of personnel of the Marine Mammal Laboratory, Zoology Department, University of Guelph, and the excellent assistance of Mr. E. W. Eaton.

T. M. WILSON

P. H. STOCKDALE

Department of Pathology
Ontario Veterinary College
University of Guelph
Guelph, Ontario, Canada

February 13, 1970