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Type III Hiatal Hernia in a Harbor Seal (*Phoca vitulina concolor*)

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Abstract

A 1-y-old male harbor seal (*Phoca vitulina concolor*), identified by a flipper tag as an animal previously rehabilitated, was found stranded and was euthanized. *Postmortem* examination revealed a type III hiatal hernia, with the lower esophageal sphincter and approximately 80% of the stomach within the hernia sac. The stomach had an organoaxial volvulus. No ingesta were found in the animal's digestive tract, and the hernia likely contributed to this animal's stranding. During the animal's previous stay in a rehabilitation facility, it had undergone inhalation anesthesia, including intermittent positive pressure ventilation. This appears to be the first description of hiatal hernia in a phocid seal.

Key Words: hiatal hernia, pinniped, seal, harbor seal, *Phoca vitulina concolor*

Introduction

In March 2005, a 1-y-old harbor seal (*Phoca vitulina concolor*) that had formerly been a patient at the Marine Animal Rehabilitation Center (MARC) at the University of New England was found stranded on the coast of New Jersey by the Marine Mammal Stranding Center (MMSC). The animal had been released in Maine 4 mo previously and was identified by a numbered flipper tag. Observers from the MMSC notified MARC and felt it best to euthanize the seal due to its perceived poor condition. No further evaluation was done by MMSC prior to euthanasia. Since the seal had undergone surgical drainage of the right tympanic cavity to treat a chronic otitis interna, evaluation of the carcass was requested to determine the success of the surgical drainage.

Materials and Methods

The seal carcass was shipped from New Jersey to Maine via common carrier and took several days en route. Despite being shipped on ice, there was some degree of *postmortem* autolysis that occurred during shipment. *Postmortem* examination was performed using the standardized protocol for pinnipeds (Dierauf, 1994).

Results

The animal had lost approximately 17 kg, representing almost 36% of its pre-release body weight of 48.2 kg. The only visible external lesion was a slight mucopurulent discharge from the right ear canal. Subcutaneously, blubber ranged from 1 to 1.25 cm over the entire carcass.

The abdomen appeared lacking of viscera, and only a small portion of the stomach, including the pyloric area and distal portion of the body, was visible in the abdomen caudal to the liver. Other organs within the abdomen appeared normal for this animal's age and species.

Upon opening the thorax, the heart, lungs, and associated structures were grossly normal; however, a large mass was visible to the left of the midline, seemingly attached caudally to the diaphragm. The heart and lungs were reflected craniad, demonstrating that the mass was a continuation of the esophagus and was covered by the pleura. Examination of the heart and lungs was unremarkable; a mild infestation of lungworms was found in the distal major bronchi.

The gastrointestinal tract was removed from the proximal esophagus to the duodenum with the diaphragm and liver intact to explore this hiatal hernia anomaly (see Figure 1).

Dissection of the hernia sac revealed approximately 80% of the stomach within the hernia sac. There were no obvious adhesions of the stomach to the sac; however, the hernia sac contents were so tightly compressed as to make manipulation of them through the sac difficult. There was a 180º axial volvulus as well as two redundant folds of the cardia, essentially blocking passage of whole fish, the preferred food of harbor seals. Further dissection of the stomach revealed numerous anasakoid parasites and an absence of ingesta. There were numerous small erosions of the mucosa of

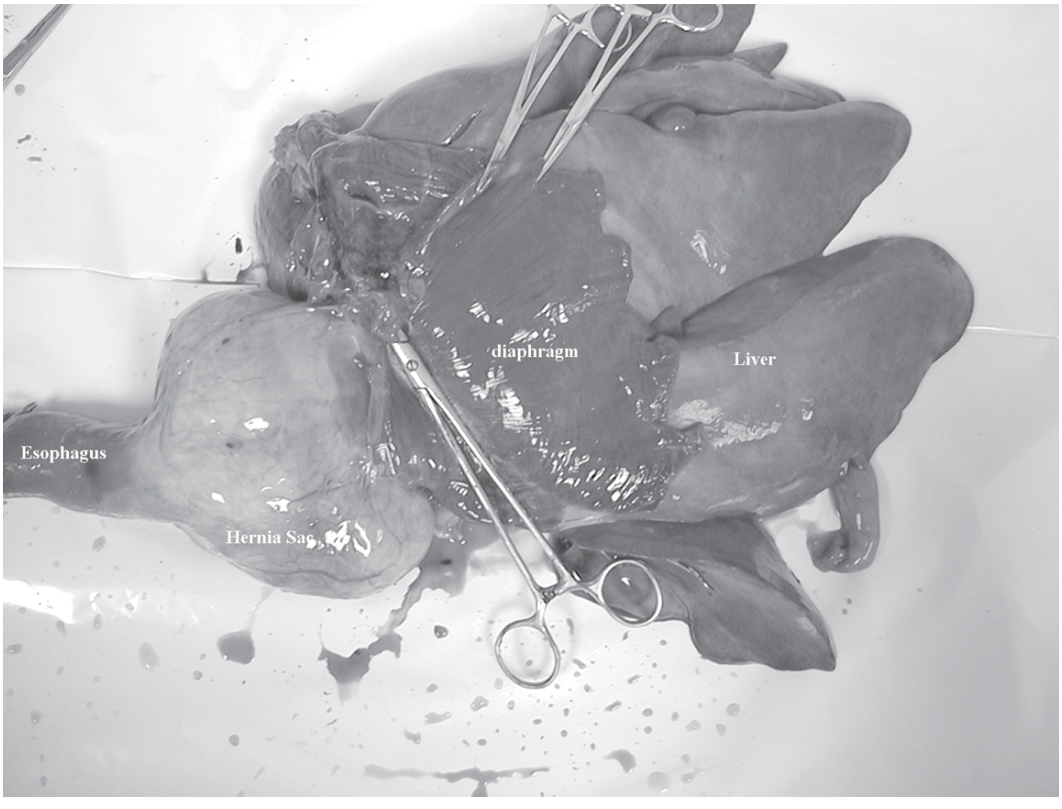

Figure 1. Postmortem view of the esophagus, diaphragm, and liver after removal; the hernia sac is visible cranial to the liver and contains the gastroesophageal sphincter and approximately 80% of the stomach.

the esophagus proximal to the lower esophageal sphincter.

Histologically, there was erosion of the stratified squamous epithelium of the esophageal mucosa with vacuolation of epithelial cells and intra-epithelial infiltrates of neutrophils. An infiltrate of moderate numbers of neutrophils and lesser numbers of macrophages was present along the deep margin of the ulcer. There were loose interstitial infiltrates of macrophages in the subjacent submucosa. The stomach had no histological lesions.

Dissection of the ear canal revealed a healed tympanic bulla with a fibrotic lining and no obvious otitis interna. Histopathology of the auditory nerve was not performed.

While it is impossible to say without question, it seems likely that the retention of the stomach with volvulus within the hiatus contributed to the animal's stranding.

Discussion

Diaphragmatic hernias occur in several forms. Congenital hernias occur as a result of failure of the diaphragm to completely close during development or due to diaphragmatic tissue weakness. Hiatal hernias represent a specific subgroup of diaphragmatic hernias. Herniation through the esophageal hiatus has been recognized as a clinical entity in numerous species, including humans (Burkitt, 1981), dogs (*Canis familiaris*) (Halpin & Soper, 2001; Hunt et al., 2002), cats (*Felis silvestris catus*) (Mitsuoka et al., 2002), cheetahs (*Acinonyx jubatus*) (Teunissen et al., 1978), a leopard (*Panthera pardus*) (Kearns et al., 2000), and a harbor porpoise (*Phocoena phocoena*) (Stephen, 1993). The majority of cases in nonhuman animals represent Type I and Type II hernias. Type I hernias often are referred to as sliding hiatal hernias as a portion of the proximal stomach and distal esophagus "slides" in and out through the muscular hiatal canal. Patients with Type l hernias often present with no signs or with signs related to gastroesophageal reflux and subsequent reflux esophagitis (DeMeester et al., 1981). Types II and III hernias are paraesophageal hernias. In Type II hernias, a portion of the proximal stomach protrudes into the thorax alongside the esophagus through an enlarged muscular hiatus. In pure Type

II hernias, the lower esophageal sphincter (LES) remains within the abdomen. Type III hernias are characterized by cranial displacement of the LES into the thorax along with variable portions of the stomach. Type IV hernias are a further progression of Type III, including other abdominal viscera in the thorax (Maziak et al., 1998; Sivacolundhu et al., 2002).

Progression from Type II to IV increases the clinical symptoms in human cases from gastroesophageal reflux through early satiety, bloating, and post-prandial discomfort to dyspepsia, vomiting, and esophageal mucosal erosion (Patti et al., 1996). Incarceration and/or organoaxial volvulus (Halpin & Soper, 2001) also have been described. Chronic iron deficiency has been described in humans with hiatal hernia and is thought to be due to blood loss through the esophageal mucosa during periods of decreased venous, a result of vascular compromise within the hernia sac (Maziak et al., 1998).

The pathophysiology of paraesophageal hernia is unclear. Theories discuss an inherent weakness in the membrane attaching the distal esophagus to the diaphragm, with exacerbation due to increased intra-abdominal pressure (Burkitt, 1981). For the harbor seal, pressure changes could occur during diving, increasing the intra-abdominal pressure over the intrathoracic pressure. Intermittent positive pressure ventilation (IPPV) during surgical intervention might contribute to weakening the esophageal hiatus, especially if excessive intrathoracic pressures were reached.

Recently, dysfunction of the longitudinal muscle of the distal esophagus caused either by loss of the inhibitory innervation of the post-diaphragmatic esophagus or by the abnormally increased tone of the longitudinal esophageal muscle layers above the diaphragm has been discussed as a factor.

Standard treatment protocols in both human and veterinary medicine involve surgical reduction (if necessary) and plication of the periesophageal diaphragmatic tissue (Nissen plication) carried out via a laparotomy approach (Wu et al., 1999; Dahlberg et al., 2001; Halpin & Soper, 2001).

The animal described here had undergone approximately 2 h of anesthesia during its initial hospitalization. At that time, anesthesia was induced by intramuscular ketamine (100 mg/ml; KetesThesia, Vetus Animal Health, Burns Veterinary Supply, Inc., Rockville Center, NY, USA) and midazolam (Midazolam HCl, Abbott Labs, North Chicago, IL, USA). Surgical anesthesia was maintained by isoflurane (Aerane, Baxter Healthcare Corp., Deerfield, IL, USA) administered via a cuffed endotracheal tube while it underwent a CAT scan and bulla osteotomy. During the entire time, the seal received IPPV. No problems were reported with IPPV, and inspiratory pressures were within accepted norms. There is no way of knowing if this procedure contributed to the hernia with volvulus.

This appears to be the first report of hiatal hernia in a phocid seal. Previous reports of diaphragmatic hernias fail to describe this type of hernia as well as the portal through which the abdominal contents passed (van der Kamp, 1987, 1994). Due to the rehabilitation status of this animal, it remains unclear whether this was a wholly naturally occurring disease process or a condition that may have been exacerbated by surgical intubation and IPPV. Either way, this case has implications for those who care for similar species in captive and rehabilitation situations.

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