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Hiatal Hernia in a Harbor Porpoise (*Phocoena phocoena*)

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ABSTRACT: We observed the displacement of the first compartment of the stomach through the diaphragmatic hiatus into the thoracic cavity in an immature harbor porpoise (*Phocoena phocoena*). This hiatal hernia, coupled with a severe pneumonia, contributed to the emaciation and death of the animal.

Key words: Harbor porpoise, cetacean, hernia, hiatus, *Phocoena phocoena*.

Hiatal hernias are infrequently described in the veterinary literature. Two basic types of hiatal hernia, with variations of each type, are recognized (Skinner, 1985). The type I hernia, or sliding hiatal hernia, is due to an enlargement of the hiatal muscular channel which allows a portion of the gastric cardia to herniate slightly upward. In these cases it is gastroesophageal reflux rather than the herniated portion of the stomach that causes the symptoms (Skinner, 1985; Barker and Van Dreumel, 1985). In the type II, or rolling hernia, a portion of the stomach herniates through a localized weakness or defect in the hiatus. With type II hernias large portions of the stomach, along with other organs such as the colon and small intestine, may enter the hernia sac within the thorax (Skinner and Belsey, 1967). More severe clinical signs develop when herniated organs or the fluids they produce diminish intrathoracic space (Wilson and Muir, 1983). Diaphragmatic hernias in veterinary medicine are most frequently the result of blunt trauma with only a small proportion being congenital in origin (Wilson and Muir, 1983). We present a case of type I hiatal herniation of the first compartment of the stomach which contributed to the emaciation and death of a harbor porpoise (*Phocoena phocoena*).

In February 1991, a dead male harbor porpoise was found on a rocky beach in Kildonan Bay (49°12'N, 125°00'W) on the west coast of Vancouver Island, British Columbia, Canada. The discovery of the carcass was not preceeded by any severe weather conditions. Three lacerations, ranging from 4 to 6 cm in length, were present on the left lateral aspect of the thoracic wall. Due to the lack of a tissue response, these lesions were considered to be post-mortem injuries. The animal measured 1,347 mm in length and weighed 25 kg. The maximum girth, measured cranial to the dorsal fin, was 762 mm. The blubber layer of the porpoise was markedly reduced, measuring only 0.7 mm in the sternal region. The porpoise's dentition and length were consistent with that of an immature animal. Based on published reports of the biometrics of harbor porpoises (Bamfield, 1974; Read, 1990), this animal was judged to be considerably emaciated.

Upon necropsy, the entire first compartment of the stomach was found to be displaced through the diaphragmatic hiatus into the porpoise's thoracic cavity. No acute traumatic lesions were noted on the diaphragm although small fibrous tags were found adjacent to the hiatus. The herniated forestomach was tightly packed and distended by a dry mass consisting primarily of small fish bones and otoliths. The remainder of the gastrointestinal tract distal to the hernia was devoid of food and feces. Several partial thickness ulcers were present at the junction of the second and third stomach compartments. Numerous nematodes, presumably *Anasakis*, were found attached to the mucosa of the second gastric compartment but were not associated with the ulcerated areas. There was a lack of internal fat stores plus serous atrophy of perirenal and epicardial fat depots in this animal. Tissue sample were preserved in 10% buffered formalin, sectioned, and stained with hematoxylin and eosin (Humanson, 1967).

A severe, multifocal necrotizing bron-

chopneumonia was the other significant finding made during the necropsy. Gross examination of the lungs revealed multiple dark, congested regions throughout the pulmonary parenchyma. Numerous necrotic areas were also seen throughout the lung lobes. A stable froth was present in the trachea and the primary bronchi. Histologic examination of the necrotic tissue showed extensive fibrinous exudation admixed with increased numbers of macrophages, neutrophils and necrotic debris. Numerous bacterial colonies were noted within the center of these necrotic patches. Further bacterial examination was not performed. Adult nematodes, presumably *Halocercus*, were present in several areas of the pulmonary parenchyma. Because of the lack of histologic evidence of a significant verminous pneumonia, it was felt that these parasites likely were not a major factor in the death of the porpoise.

Histological study of the intestine demonstrated prominent lymphoid follicles with moderate infiltration of the medullary sinuses by neutrophils. The cortical portion of these follicles had focal aggregations of multinucleated giant cells surrounded by neutrophils. Granulomatous foci were noted in the tunica submucosa of the main stomach. The mesenteric lymph nodes appeared grossly enlarged. The splenic lymphoid follicles were prominent and demonstrated occasional active germinal centers.

Although the majority of diaphragmatic hernias in veterinary medicine are traumatic in origin, developmental and heredity etiologies have been identified in several species (Wilson and Muir, 1983; Fox and Crary, 1973). There was no direct pathological evidence in this case with which to diagnose a cause for the hiatal hernia. When a medical history is lacking, an acute traumatic hernia can often be deduced by the presence of other traumatic lesions, such as an abdominal wall hematoma (Wilson and Muir, 1983). The fibrous tags noted in this porpoise are similar to those described by Brinkley (1990) for a cat struck by a car one year prior to admission for the repair of an extensive hiatal hernia, suggesting a chronic course for this porpoise's hernia. Type II hernias in humans progressively enlarge and can result in herniation of a large portion of the stomach over time (Skinner, 1985). Congenital hiatal hernias in small children typically result in a patient with postural regurgitation, vomiting, anemia, and a failure to thrive and develop (Skinner and Belsey, 1967). Given this animals age and poor condition, a congenital etiology for the hernia cannot be ruled out.

Although gastric ulceration in cetaceans has been associated with chronic stress and prolonged anorexia, the presence of these lesions does not help to determine the course of the porpoise's illness. Gastric ulcers are not uncommon in cases of gastric herniation. "Riding ulcers" can be seen where the gastric mucosa overrides the underlying ridge of the hiatus (Skinner, 1985). Disruption to the blood flow supplying the gastric mucosa is another possible cause for this animal's ulcers as the lesions were found in the unherniated portion of the stomach as opposed to the region over the hernia ridge. Increased feed histamine levels, gastric nematodes, and bacterial infections, all potential factors in this case, also have been associated with gastric ulceration in cetaceans (Sweeney and Ridgeway, 1975).

In the face of the severe, necrotizing bronchopneumonia and granulomatous lymphadenitis, one may question the role of the partial gastric herniation, as a contributing cause of the death of this harbor porpoise. The forestomach of a harbor porpoise functions to liquify ingested food and present it to the main stomach (Smith, 1972). Based on the impaction of the forestomach and lack of food material distal to the hernia, it can be concluded that the hernia prevented adequate delivery of food to the gastrointestinal tract. Based on the near complete depletion of fat stores, this animal probably was severely nutritionally compromised. We postulate that sufficient

liquified feed passed from the herniated forestomach to sustain a young, healthy porpoise, but was insufficient to meet the increased demands created by the debilitation associated with the more acute respiratory disease and the demands of foraging as the animal matured. An alternative explanation for the combination of lesions present in this case could be one of aspiration of regurgigated or vomited material. However, the anatomical separation of the digestive and respiratory tracts of cetaceans makes this possibility unlikely. One may therefore conclude that, although not the immediate cause of the porpoise's death, the hiatal hernia made a significant contribution to the debilitation of the animal, thus speeding its demise.

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