HIATAL HERNIA AND DIAPHRAGMATIC EVENTRATION IN A LEOPARD (PANTHERA PARDUS)


Abstract: A 1-yr-old male leopard (Panthera pardus) presented for intermittent anorexia, emaciation, and generalized muscle wasting. Plain radiographs, ultrasonography, and esophageal endoscopy led to a diagnosis of diaphragmatic eventration with probable concurrent hiatal hernia. An exploratory laparotomy confirmed both diagnoses, and surgical repair and stabilization were performed. After surgery, the leopard was maintained on small liquid meals for 4 days, with a gradual return to normal diet over 2 wk. By 4 wk after surgery, the leopard was eating well and gaining weight, and it showed no recurrence of clinical signs for 2 yr subsequently, becoming mildly obese.

Key words: Esophagitis, eventration, gastropexy, hiatal hernia, leopard, Panthera pardus.

CASE REPORT

A 1-yr-old male leopard (Panthera pardus) was examined for intermittent anorexia, emaciation, and apparent weight loss of at least 3 mo duration. Physical examination under chemical restraint using estimated weight revealed emaciation with generalized muscle wasting. Hematologic abnormalities (based on normal reference values from the International Species Information System, Apple Valley, Minnesota 55124, USA) included a nonregenerative normocytic normochromic anemia (hematocrit = 22.9%, reference = 37.0 ± 5.4%; 5.21 ± 10 red blood cells [RBC]/µl, reference 8.28 × 10^6 ± 1.46 × 10^6 RBC/µl; MCV = 43 fl, reference = 45.3 ± 5.4 fl; MCH = 14.4 pg, reference = 15.2 ± 1.8 pg) and a leukocytosis with left shift, neutrophilia, and eosinophilia (total white blood cells [WBC] = 29,100/µl, reference = 13,740 ± 4022 WBC/µl; 16,300 segmented neutrophils/µl, reference = 10,340 ± 3872 neutrophils/µl; 7,860 bands/µl, reference = 1,216 ± 2,455 bands/µl; 3,780 lymphocytes/µl, reference = 1,746 ± 974 lymphocytes/µl; 1,160 eosinophils/µl, reference = 528 ± 516 eosinophils/µl). Serum chemistry analysis, urinalysis, and feline infectious disease serology (feline leukemia virus, feline immunodeficiency virus, feline infectious peritonitis, and Toxoplasma) were unremarkable.

Radiographs revealed a large soft tissue opacity contiguous with the dorsal diaphragm. Contents of the mass appeared to include a partially gas-filled stomach, small intestine, and several triangular soft tissue opacities thought to represent liver lobes and spleen. A megaesophagus was present cranial to this mass (Fig. 1).

An esophagram using barium sulfate (Sol-pace, E-Z-EM Inc., Westbury, New York 11590-5021, USA, diluted 50/50 by volume with water, 3.5 ml/kg) showed megaesophagus with irregular margins caudally and ventral deviation of the cardiac. Ultrasonography confirmed the presence of stomach, small intestine, and congested liver tissue within the mass observed on radiographs. A presumptive diagnosis of diaphragmatic eventration was made. From these diagnostic tests, it was difficult to ascertain whether a hiatal hernia was present as well. Diaphragmatic hernia was included in the differential diagnoses, but it appeared less probable on the basis of the radiographic appearance of the diaphragm.

Endoscopic examination (Fujinon gastroscope, EPX301A, 9.6 mm, Wayne, New Jersey 07470, USA) of the esophagus revealed mild inflammation orad, with fluid pooling. More caudally, severe erosions were observed with multifocal areas of discoloration and hemorrhage. Cranial to the gastroesophageal junction, an eccentric intramural mass almost completely obstructed the esophageal lumen. Samples of the esophageal mucosa were taken for cytology, and biopsies of the intraluminal mass were submitted for culture and histopathology. The stomach appeared grossly normal.

Cytology of the esophageal mucosa showed evidence of inflammation with sepsis. All bacteria cultured were normal enteric organisms. No evidence of neoplasia was noted on cytology or histopathology. These findings were most consistent with a diagnosis of reflux esophagitis, which strengthened the suspicion that a hiatal hernia was also present, causing gastroesophageal reflux.

Eight days after initial presentation, an exploratory laparotomy was performed. On the basis of
an estimated body weight of 23 kg, the leopard was restrained with ketamine (Ketaset®, Fort Dodge Laboratories Inc., Fort Dodge, Iowa 50501, USA; 5 mg/kg i.m.) and xylazine (BenVenue Laboratories, Bedford, Ohio 44146, USA; 1 mg/kg i.m.), intubated, and maintained on isoflurane (IsoFlo®, Abbott Laboratories, North Chicago, Illinois 60064, USA) in oxygen. A standard ventral midline approach to the abdomen revealed an abnormally enlarged esophageal hiatus, allowing herniation of abdominal organs. Viscera that had moved through the hiatus into the thoracic cavity included the left medial and quadrate liver lobes with gallbladder, approximately 20 cm of small intestine, and the entire spleen and stomach. In addition, a large portion of the diaphragm just lateral to the hiatus appeared to be thin-walled and flaccid, and it protruded cranially. Additional small intestine was displaced cranially into this cupola. All viscera appeared viable and were placed in their normal anatomic positions. The left and right ventrolateral aspects of the esophagus were sutured to the diaphragm along the edge of the hiatus with two simple interrupted 0 polypropylene sutures (Prolene®, Ethicon, Johnson and Johnson Co., Somerville, New Jersey 08876, USA) on each side (esophagopexy). The redundant diaphragmatic tissue was then plicated with seven simple interrupted sutures of 0 polypropylene. The excess diaphragmatic tissue was incorporated until the left hemidiaphragm protruded no farther cranial than the normally positioned right hemidiaphragm. Finally, a left-sided incisional gastropexy, using the fundus of the stomach, was performed with 0 polypropylene suture.2

After surgery, the leopard was maintained on Canine/Feline A/D (Hills Pet Nutrition Inc., Topeka, Kansas 66601, USA) for the first 4 days, followed by a gradual return to a normal amount and consistency of food over the next 2 wk. Trimethoprim-sulfamethoxazole (BioCraft Laboratories Inc., Elmwood Park, New Jersey 07407, USA; 30 mg/kg b.i.d. p.o.) and sucralfate (Marion Merrell Dow Inc., Kansas City, Missouri 64114, USA; 1 g, b.i.d. p.o.) were administered to treat the reflux esophagitis. By 4 wk after the operation, the leopard was no longer anorectic or lethargic and appeared to be gaining weight. There was no recurrence of clinical signs. At a follow-up examination, the body weight was 41 kg. Radiographs taken 5 yr postoperatively were normal (Fig. 2).

DISCUSSION

Eventration is a thinning of the diaphragmatic tissue, resulting in a more cranial position of the
intact diaphragm. This condition is more frequently reported in humans than in animals. It is usually congenital, but it may be acquired secondary to trauma. In humans with diaphragmatic eventration, the normal anatomy of the gastroesophageal angle is disrupted by displacement of the stomach into the expanded diaphragmatic sac.

A hiatal hernia occurs when enlargement of the esophageal hiatus of the diaphragm allows abdominal contents to protrude into the thorax. There are two main types of hiatal hernias in animals. The sliding type, in which the gastroesophageal junction and frequently the stomach are displaced into the thorax and the one present in this leopard, is the most commonly reported. Reflux esophagitis is a frequent sequel. Large sliding hernias are uncommon and, as in this case, may include liver, spleen, and intestines. Paraesophageal hernias, in which the stomach is displaced through a diaphragmatic defect adjacent to the hiatus, are rarely reported in dogs and cats. The gastroesophageal junction remains in the abdomen and reflux esophagitis is therefore less prevalent. Although most cases reported in dogs and cats are congenital, acquired hiatal hernias have occurred secondary to trauma or after diaphragmatic hernia repair. Eventration may exist with or without concurrent hiatal hernia. Congenital diaphragmatic eventration with coexisting esophageal hiatal hernia has been reported in a Shetland sheepdog. In cases without hiatal hernia, reflux esophagitis is not an expected sequel because the lower esophageal sphincter is not displaced into the thorax.

The predominant clinical sign in patients with hiatal hernia is regurgitation, which appears to be due to the gastroesophageal reflux. In normal animals, several factors are involved in preventing gastroesophageal reflux. These factors include normal anatomic position of the lower esophageal sphincter (LES), adequate tension of the gastroesophageal ligament, and presence of a high-pressure zone in the caudal esophagus. These conditions are compromised when the LES is displaced into the thorax. Resulting gastroesophageal reflux causes an esophagitis, which is associated with decreased esophageal motility. Many animals develop a megaesophagus and subsequent aspiration pneumonia. Other clinical signs that may occur include...
ptyalism, dyspnea, weight loss, and anorexia. A concurrent diaphragmatic eventration may also cause dyspnea because of decreased functional residual capacity and abnormal diaphragmatic movement. This leopard was unusual in that emaciation and intermittent anorexia were the only presenting signs. Despite the presence of a megaesophagus and severe esophagitis, no regurgitation was observed.

Survey radiographs facilitate diagnosis of hiatal hernia and eventration. Radiographic diagnosis of diaphragmatic eventration is based on the presence of a bulging or arched diaphragm with unbroken continuity. A sliding hiatal hernia implies cranial displacement of the gastroesophageal junction through the hiatus into the caudal mediastinum. It may be diagnosed radiographically by identification of the abnormally located gastroesophageal junction cranial to the diaphragm. In this case, at the time radiographs were made, the gastroesophageal junction was contiguous with the diaphragm as it bulged forward. The bulging diaphragm created ventral bending of the caudal mediastinum, distorting the gastroesophageal junction. Diagnosis of this disorder may be aided by positive contrast fluoroscopic examination revealing reduced esophageal motility or gastroesophageal reflux due to LES incompetence. Endoscopy is helpful in assessing the severity of the reflux esophagitis. In this leopard, eventration was diagnosed radiographically, but final diagnosis of the hernia was not conclusive until surgery, when it was apparent that the stomach and other organs had since moved into the thoracic cavity and a large hiatal hernia was visualized.

In many cases, hiatal hernia may be managed medically. Dietary modification, with or without the use of antisecretory drugs and prokinetic agents, may be enough to keep less severe cases asymptomatic. In cases where endoscopic examination shows severe esophagitis, or when medical therapy has yielded unsatisfactory results, surgical repair is indicated. The goal of surgical repair is to anatomically reduce and stabilize the hernia. Stabilization is accomplished using plication of the diaphragm to narrow the hiatus and eliminate any concurrent eventration, esophagopexy involving fixation of the caudal esophagus to the diaphragm, and gastropexy to prevent cranial displacement of the stomach.

In humans, surgical repair of hiatal hernias involves fundoplication for structural reinforcement of the LES. This procedure has previously been used in dogs but has been associated with complications such as vomiting, reherniation, and gastric tympany. Since dogs and cats do not appear to have primary sphincter incompetence, as is commonly observed in humans with hiatal hernias, structural reinforcement of the sphincter should not be necessary. The recommended surgical management in dogs and cats is simple reduction and stabilization. The preferred treatment for diaphragmatic eventration is diaphragmatic plication, particularly in symptomatic cases. In this case, the plication of the diaphragm performed during the hiatal hernia repair simultaneously eliminated the eventration.

This case of a hiatal hernia and diaphragmatic eventration in a leopard was managed by simple reduction and stabilization, and by plication, respectively. It is the first report of either disorder in this species. Since long-term medical therapy would have been impractical in a large exotic cat, it was critical that surgical intervention result in a successful outcome. The unusual presentation in this case underscores the importance of considering hiatal hernia as a differential diagnosis in young cachectic animals, even when regurgitation is not observed.

**LITERATURE CITED**


Received for publication 6 November 1998