Esophageal Diverticulum Associated With a Trichobezoar in a Cat

A 9-year-old, castrated male, domestic longhaired cat was evaluated for persistent regurgitation over the previous month. The cat had presented 9 months earlier and was diagnosed with esophageal obstruction secondary to a trichobezoar. The trichobezoar had been removed endoscopically, and the cat was subsequently fed a canned prescription diet. The owners noted only infrequent regurgitation over the following 9 months. After signs recurred, contrast radiography with fluoroscopy revealed an esophageal diverticulum at the thoracic inlet, with an ovoid filling defect. Decreased esophageal motility was noted distal to the diverticulum. Esophagoscopy confirmed the presence of a trichobezoar within an esophageal diverticulum. Following removal of the trichobezoar and therapy to prevent trichobezoar formation, the cat did well for 2 months until it died suddenly with signs of hyperventilation and open-mouth breathing. **J Am Anim Hosp Assoc 2009;45:000-000.**

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Introduction

An esophageal diverticulum is a congenital or acquired, pouch-like sacculation of the esophageal wall.[1] Congenital diverticula are rare and are caused by developmental abnormalities involving bronchopulmonary-foregut malformations.[2] Acquired esophageal diverticula are classified as either traction or pulsion diverticula.

Traction diverticula are caused by disorders of the thorax secondarily affecting the esophagus. Periesophageal inflammation leads to fibrosis and contraction, which pulls the wall of the esophagus out, forming a pouch. Pulsion diverticula are caused by disorders within the esophagus that increase intraluminal pressure, such as obstruction by strictures or foreign bodies.[1] In humans, pulsion diverticula (in particular, those in an epiphrenic location) are commonly associated with an underlying esophageal motility disorder.[3]

Reports of esophageal diverticula in cats are rare. One cat has been described with a "diverticulum-like" esophageal lesion, which appeared to be a dilated segment of the cranial intrathoracic esophagus.[4] Recently, a cat with a pharyngeal diverticulum and recurrent oropharyngeal foreign bodies was reported.[5] Surgical excision of a diverticulum is usually recommended; however, since clinical signs may not occur with a small diverticulum, a conservative approach may be warranted.[1] This case report highlights an unusual case of an esophageal diverticulum in a cat and possible management for such cases.

Case Report

A 9-year-old, castrated male, domestic longhaired cat was presented for evaluation of a prolonged history of intermittent vomiting and anorexia and a recent onset of regurgitation. Episodes of vomiting and anorexia had been a long-standing problem and were suspected by the local veterinarian to be from inflammatory bowel disease or dietary intolerance. However, no gastrointestinal biopsies had been performed, and signs

resolved with antiemetics. The current episode of vomiting and anorexia was accompanied by regurgitation and did not respond to empirical treatment by the referring veterinarian. Such treatment included famotidine[a] (0.5 mg/kg per os [PO] *q* 24 hours), aminopentamide hydrogen sulfate[b] (0.25 mg/kg PO *q* 12 hours), enrofloxacin[c] (dose not specified), metronidazole[d] (dose not specified), subcutaneous fluid therapy, and a diet change to a hypoallergenic diet.[e] The cat was then referred to the authors' hospital for further evaluation.

On presentation, the cat was hypothermic (temperature 96˚F) and in poor body condition (body condition score was 2/5; body weight was 4.4 kg). The cat was assessed to be approximately 7% dehydrated. Heart and respiratory rates were within normal limits. A grade II/VI parasternal systolic heart murmur was ausculted, but the owners declined additional testing with an echocardiogram. Pain was elicited on abdominal palpation, and the cat vomited a foul-smelling, brownish-colored fluid during the physical examination.

Blood tests at that time revealed azotemia, hyperphosphatemia, hyperproteinemia, hyponatremia, hypochloremia, and a high anion gap metabolic acidosis. The changes were consistent with vomiting and secondary dehydration, and they resolved with fluid therapy.[f]

Thoracic radiographs revealed a fluid-filled, dilated esophagus with an associated esophageal mass or foreign body at the base of the heart and changes consistent with aspiration pneumonia [Figure 1]. A contrast esophagram (without fluoroscopy) and esophageal ultrasound were performed. Findings suggested an intraluminal esophageal foreign body, rather than a mass lesion, as barium was able to pass on all sides of the lesion.

On the following day, the cat was anesthetized for upper gastrointestinal endoscopy. A large foreign body consisting of hair and tuna was removed from the midthoracic esophagus by elongated rat tooth forceps. Severe esophagitis localized to the area of the trichobezoar was present. The remainder of the esophagus was grossly normal. In light of the previous history of vomiting and anorexia, biopsy specimens of the stomach were obtained for histopathology, which were later reported as within normal limits. The diagnosis was an esophageal trichobezoar causing obstruction, with secondary esophagitis and aspiration pneumonia.

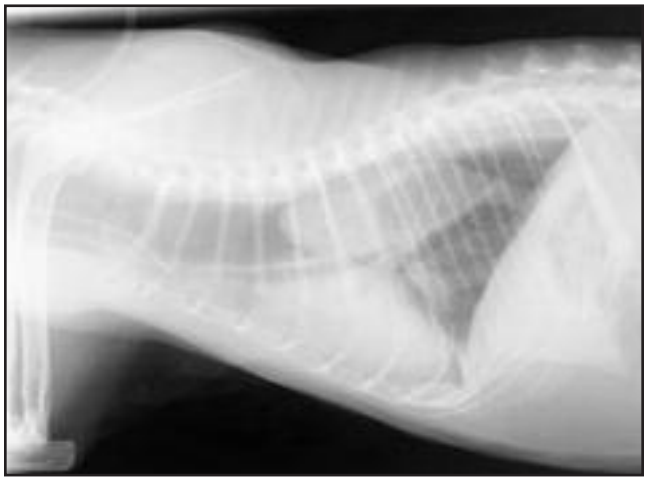

Figure 1—Thoracic radiograph revealing a dilated, fluid-filled esophagus and aspiration pneumonia.

The cat was treated with total parenteral nutrition (rather than the enteral route because of the history of vomiting), intravenous fluids,[f] omeprazole[g] (1.5 mg/kg suspension [2 mg/mL of omeprazole per mL of 8.4% sodium bicarbonate prepared as described[6,7]] PO *q* 24 hours), sucralfate[h] (0.5 gm PO *q* 8 hours), metoclopramide[i] (0.3 mg/kg subcutaneously *q* 8 hours), and amoxicillin-sulbactam[j] (30 mg/kg intravenously *q* 8 hours). The cat improved clinically and approximately 7 days later was released from the hospital. Owners were instructed to give the cat sucralfate (0.5 gm PO *q* 8 hours) and famotidine (0.5 mg/kg *q* 24 hours) for 10 days and a strict diet of a canned, high-calorie prescription diet.[k] This diet was chosen for its high caloric density and palatability, as well as its consistency. An underlying esophageal motility disorder was considered as a potential predisposing cause for the esophageal trichobezoar, but further evaluation of esophageal motility with a contrast study and fluoroscopy was not conducted at that time.

On subsequent rechecks, the owners reported that the cat continued to have intermittent regurgitation, which seemed worse when the cat was shedding or eating any food other than the prescription diet.[k] Potential causes for regurgitation included an underlying esophageal motility disorder, healing of severe esophagitis with an esophageal stricture, or recurrent esophageal trichobezoars. Repeated survey thoracic radiographs (at 3 months and at 8 months after discharge) showed no signs of megaesophagus or esophageal foreign body. Mild peribronchial infiltrates were noted. These infiltrates were consistent with feline asthma, bronchitis, neoplasia, or pulmonary inflammation; however, no clinical signs of respiratory disease had been reported by the owners. A follow-up contrast esophagram with fluoroscopy was recommended on several occasions but was declined by the owners. The cat was continued on famotidine (0.5 mg/kg *q* 24 hours), trichobezoar treatment,[l] and the prescription diet.[k] The cat gained approximately 4 kg of weight and was reportedly doing well.

Approximately 9 months after the cat's first presentation, the owners returned the animal to the authors' hospital for evaluation of persistent regurgitation over the past month. The regurgitation had progressed to the point where it was now occurring at almost every mealtime, despite the cat eating the prescription diet.[k] The cat had a markedly decreased appetite and was lethargic and hiding during the 2 days prior to presentation. On physical examination, excessive salivation and weight loss of approximately 0.6 kg were noted. Temperature, pulse, and respiratory rate were within normal limits. The previously noted heart murmur was unchanged. In light of the cat's previous history, differential diagnoses included esophageal foreign body, esophageal stricture, or worsening of an esophageal motility disorder.

Results of a complete blood count and biochemical profile were unremarkable. Thoracic radiographs revealed widening of the cranial mediastinum with the cranial lung

lobes pushed caudally. A large area of heterogenous, foamy soft-tissue density was noted in the ventral portion of the cranial mediastinum, and the thoracic esophagus over the heart contained air. The previously noted peribronchial changes in the lungs were more obvious. A barium esophagram (performed with liquid barium only) with fluoroscopic evaluation revealed a large diverticulum at the thoracic inlet with an ovoid filling defect that nearly completely filled the diverticulum [see Video]. Esophageal motility distal to the diverticulum was decreased, with incomplete clearing of the barium bolus on both primary and secondary peristaltic waves. However, the cat was occasionally able to form a normal secondary bolus. No evidence of gastroesophageal reflux or a hiatal hernia was seen.

Video—Barium esophagram showing a large, ovoid filling defect at the level of the thoracic inlet.

Endoscopy was performed under general anesthesia, and an esophageal foreign body was noted at the level of the thoracic inlet [Figure 2]. The foreign body, which consisted of hair mixed with food, was removed; this allowed better visualization of the diverticulum [Figure 3]. No evidence of esophagitis within the diverticulum was noted. The remainder of the esophagus was endoscopically normal. After the cat recovered from general anesthesia, a neurological examination, thyroid level, and myasthenia gravis titer were performed to further evaluate for causes of an underlying esophageal motility disorder. All results were within normal limits. Treatment options for the diverticulum were considered in consultation with the surgical service. A trial of conservative medical therapy was recommended prior to considering more aggressive options such as surgical resection of the diverticulum. The cat was discharged from the hospital with recommendations for the owners to keep the animal's coat closely shaved (to prevent trichobezoars) and to continue feeding the same canned prescription diet.[k] Additional esophageal promotility therapy would be considered in the future if the cat continued to have signs of regurgitation.

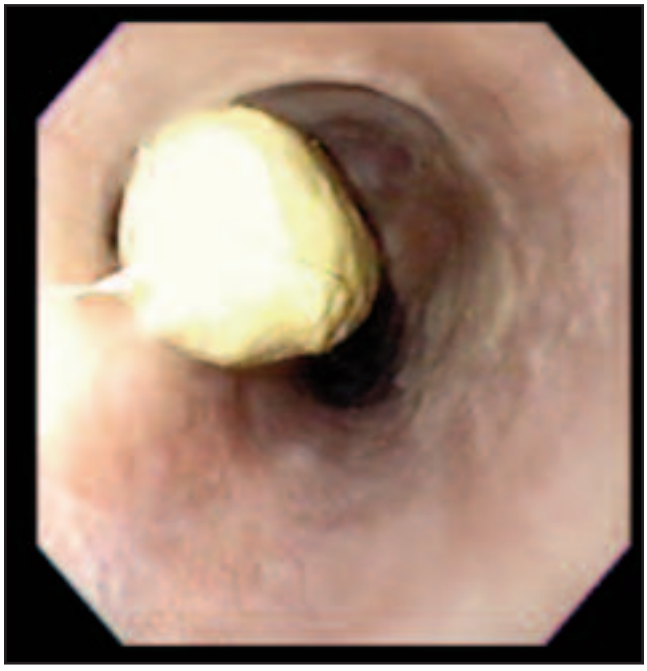

Figure 2—Esophagoscopy showing a large trichobezoar at the level of the thoracic inlet.

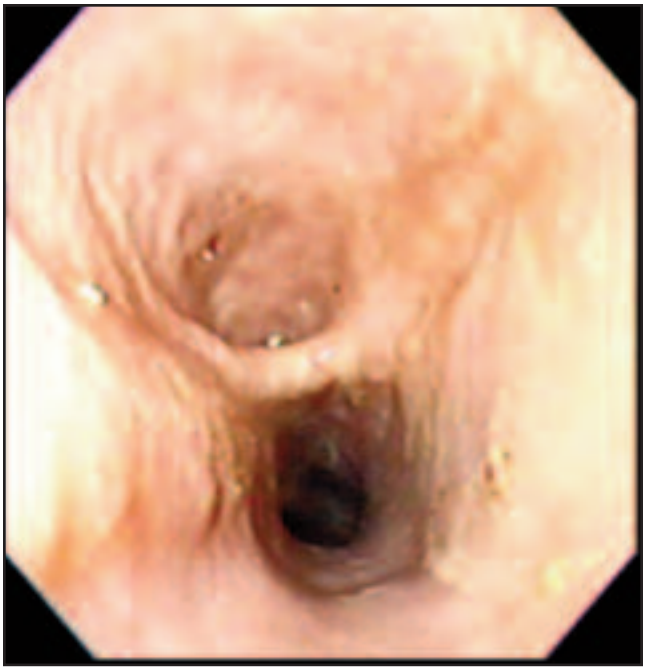

Figure 3—Esophagoscopy revealing a diverticulum (i.e., a blind, pouch-like sacculation) in the esophagus, at the level where the trichobezoar had been removed. No evidence of esophagitis was noted.

On recheck evaluation 2 weeks later, the owners reported that the cat was doing very well and did not regurgitate as long as he ate only the prescription diet.[k] On two instances, the cat ate the housemate's dry food and subsequently regurgitated. Repeat thoracic radiographs revealed a widened cranial mediastinum (possibly due to obesity or the esophageal diverticulum) and a large area of soft-tissue opacity, consistent with the previously noted diverticulum in the cranial thoracic esophagus; however, no evidence of a foreign body was seen. The cat was released from the hospital with recommendations for a follow-up barium esophagram to be performed in approximately 6 months. According to the owners, the cat did well for 2 months following discharge but then died acutely at home following an episode of hyperventilation and open-mouth breathing. A necropsy of the cat was declined by the owners.

Discussion

This case illustrates an esophageal diverticulum, an unusual condition in cats. In this case, the authors knew that the

diverticulum was acquired, because it was not present on the original contrast study or endoscopy. No history of trauma or evidence of intrathoracic disease was seen that would cause a traction diverticulum, so it was most likely an acquired pulsion diverticulum. The cause of the diverticulum could have been increased intraluminal pressure secondary to obstruction by a trichobezoar, or the trichobezoar could have developed secondary to accumulation of hair in the esophageal diverticulum. The thoracic inlet is a common area for foreign bodies to lodge in the esophagus, as it represents a physiological narrowing of the esophageal lumen. This cat had fluoroscopic evidence of esophageal dysmotility, which could also have contributed to the development of the trichobezoar.

A review of the literature revealed only one report of an esophageal diverticulum-like pouch in an 8-month-old Himalayan cat with a 1-week history of coughing, nasal discharge, and a 5-day history of postprandial vomiting.[4] A barium esophagram revealed marked dilatation and redundancy of the cranial intrathoracic esophagus. No evidence of obstruction was noted during surgical exploration, and the 5-cm dilated segment was surgically resected. The cat died 2 days later. The authors discussed whether the esophageal lesion might be better termed an esophageal pouch than a diverticulum. That cat was also diagnosed with asthma, although the relationship (if any) between the dilated esophagus and the respiratory disease was unknown.

Esophageal obstruction by a trichobezoar is believed to occur when a trichobezoar that is vomited from the stomach becomes lodged in the esophagus.[8-13] In some cases, an underlying esophageal motility disorder has been suspected to predispose to esophageal trichobezoars.[8,10] In one case report, a 3-year-old cat developed recurrent esophageal trichobezoars with no accompanying megaesophagus. A functional esophageal motility disorder was suspected, although the cat recovered with only symptomatic therapy (i.e., shaving and trichobezoar treatment).[8] One cat with a possible congenital megaesophagus developed an esophageal trichobezoar.[10] That cat died following esophagotomy to remove the trichobezoar. Four other cats have been reported to develop esophageal obstruction secondary to trichobezoars without apparent underlying esophageal motility disorders.[9,11-13] All of these cats responded to symptomatic therapy involving shaving the hair coat and administration of oral, petroleum-based treatments for trichobezoar. Compared to the number of cats that routinely pass trichobezoars (either through the feces or the vomitus), the number of reported cases of esophageal trichobezoars causing problems is very low.

The cat in this report showed evidence of a mild, diffuse esophageal motility disorder on the barium study and fluoroscopy, which may have been a predisposing factor contributing to recurrent esophageal trichobezoar obstruction. An underlying motility disorder would also explain the persistent regurgitation when the cat ate anything other than canned soft food during the 6- to 8-month period between confirmed instances of esophageal trichobezoars. An esophageal motility disorder may also have contributed to the long-standing unexplained signs of intermittent vomiting and anorexia, since common presenting gastrointestinal signs in a large series of cats with esophageal motility dysfunction included vomiting, regurgitation, dysphagia, and anorexia.[14] In that series, 43% of the cases of esophageal motility dysfunction were considered idiopathic, and 57% were associated with conditions known to cause esophageal motility dysfunction (e.g., myasthenia gravis, dysautonomia, esophagitis associated with hiatal hernia or gastroesophageal reflux) or conditions secondary to esophageal obstruction (e.g., mediastinal masses, vascular ring anomaly, esophageal stricture).[14]

The cause of the esophageal motility disorder in this cat was not determined, and no evidence for risk factors cited in the above series of cats could be identified. A thyroid level was performed to evaluate for possible hypothyroidism. Thyroid function is often evaluated in dogs with acquired megaesophagus; however, a relationship between hypothyroidism and esophageal motility disorders in dogs has been questioned.[15] Whether hypothyroidism is associated with esophageal motility dysfunction in cats is unknown.

It is interesting to speculate that an underlying esophageal motility disorder could also have contributed to esophageal diverticulum formation in this cat. Although primary esophageal motility disorders are an important predisposing cause of acquired esophageal diverticula in humans, the motility disorders are usually those associated with functional esophageal obstruction causing increased intraluminal pressure, such as achalasia (i.e., failure of the gastroesophageal sphincter to relax) or diffuse esophageal spasm.[2,3,16] However, nonspecific primary hypomotility disorders have also been documented.[3,16]

An esophageal diverticulum as described in this cat does not appear to be a common sequela of idiopathic esophageal hypomotility disorders in cats, based on the absence of a description of this complication in a large series.[14] A combination of factors (i.e., esophageal hypomotility and lodging of hair and foreign material at the thoracic inlet) likely led to the development of the diverticulum, although this conclusion is not definite.

Diverticulectomy and reconstruction of the esophageal wall are the preferred treatment method for large diverticula in humans.[1,17] Small diverticula can be managed conservatively with measures that reduce the likelihood of diverticulum impaction, such as feeding a liquid or semiliquid diet.[1] The authors chose to manage this cat medically, instituting trichobezoar prevention measures and continuing the semiliquid diet, prior to recommending a more aggressive surgical approach. With the mild clinical signs the cat was experiencing, the risks of surgery were considered to outweigh the potential benefits. The role that the mild esophageal motility disorder played in the cat's intermittent signs of regurgitation was also unclear, which could have led to a higher failure rate of surgery.

Other therapy measures that could have been attempted include providing the food in an elevated position, holding

the cat upright after feeding, and administering prokinetic drugs to improve esophageal motility. In the Moses study, 78% of the cats with various esophageal disorders responded to treatment with H2 receptor antagonists and prokinetics (i.e., metoclopramide or cisapride).[14] Cisapride would appear to be a reasonable choice for an esophageal promotility drug in cats because of its effect on esophageal smooth muscle (present in the distal esophagus of cats), although no studies have demonstrated proof of clinical efficacy.[18]

Humans with small esophageal diverticuli may be asymptomatic for the diverticulum, with clinical signs resulting from the underlying motility disorder.[2] The cat's initial response to conservative medical management in this study was encouraging, and consequently the treatment was continued. Without a necropsy, the cause of sudden death in this cat could not be determined. Potential causes of death that could be related to the diverticulum include regurgitation and secondary aspiration or impaction of the diverticulum with mucosal necrosis and perforation. Unrelated causes of the acute signs of hyperventilation, open-mouth breathing, and sudden death include asthma or uncharacterized cardiac disease.

Conclusion

Esophageal diverticula are rare in cats, but they should be considered a differential diagnosis in cats with chronic regurgitation, esophageal hypomotility, and recurrent esophageal trichobezoars. In this case, the clinical signs were relatively mild, and it was difficult to separate whether signs were from the esophageal motility disorder or from the acquired diverticulum. This cat responded, at least for a few months, to medical management consisting of diet change and trichobezoar prevention. Therefore, conservative treatment of small esophageal diverticula should be considered.

Footnotes

[a] Pepcid AC; Merck, Whitehouse Station, NJ 08889
[b] Centrine; Fort Dodge Animal Health, Fort Dodge, IA 50501
[c] Baytril; Bayer, Shawnee Mission, KS 66201
[d] Flagyl; Pharmacia and Searle, Chicago, IL 60680
[e] Green Pea and Duck; Innovative Veterinary Diets; Royal Canin USA, Inc., St. Charles, MO 63301
[f] Plasmalyte 148 KCl supplementation; Baxter International, Inc., Deerfield, IL 60015
[g] Prilosec; Procter & Gamble, Cincinnati, OH 45202
[h] Carafate; Axcan Scandipharm, Inc., Birmingham, AL 35242
[i] Reglan; A.H. Robins Co., Inc., Philadelphia, PA 19101
[j] Unasyn; Roerig (Pfizer, Inc.), New York, NY 10017
[k] A/D; Hill's Pet Nutrition, Inc., Topeka, KS 66601
[l] Catlax; Pharmaderm Animal Health, Melville, NY 11747

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