Esophageal leiomyoma in a dog causing esophageal distension and treated by transcardial placement of a self-expanding, covered, nitinol esophageal stent

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CASE DESCRIPTION

A 10-year-old spayed female Rottweiler was referred for evaluation because of a 2-month history of regurgitation and weight loss, despite no apparent change in appetite. The dog had received antiemetic and antacid treatment, without improvement.

CLINICAL FINDINGS

Physical examination revealed a low body condition score (2/5), but other findings were unremarkable. Diffuse, global esophageal dilatation was noted on plain thoracic radiographs, and normal motility was confirmed through videofluoroscopic evaluation of swallowing. Transhepatic ultrasonographic and CT examination revealed a circumferential, intraparietal lesion in the distal portion of the esophagus causing distal esophageal or cardial subobstruction and no metastases. Incisional biopsy of the lesion was performed, and findings of histologic examination supported a diagnosis of esophageal leiomyoma.

TREATMENT AND OUTCOME

In view of numerous possible complications associated with esophageal surgery, the decision was made to palliatively treat the dog by transcardial placement of a self-expanding, covered, nitinol esophageal stent under endoscopic guidance. Two weeks after stent placement, radiography revealed complete migration of the stent into the gastric lumen. Gastrotomy was performed, and the stent was replaced and fixed in place. Twenty-four months after initial stent placement, the dog had a healthy body condition and remained free of previous clinical signs.

CLINICAL RELEVANCE

Diffuse benign muscular neoplasia should be considered as a differential diagnosis for acquired esophageal dilatation in adult and elderly dogs. In the dog of this report, transcardial stent placement resulted in resolution of the clinical signs, with no apparent adverse effect on digestive function. The described procedure could be beneficial for nonsurgical treatment of benign esophageal tumors in dogs. (J Am Vet Med Assoc 2018;252:330–335)

ABBREVIATIONS

PEG  Percutaneous endoscopically placed gastrostomy

A 10-year-old spayed female Rottweiler was referred for evaluation because of a 2-month history of progressive regurgitation (at least 10 times/d) and weight loss, despite no apparent change in appetite. No change in voice or signs of weakness had been noticed by the owner. Vaccination status and deworming treatments were up-to-date. The dog was receiving a dry digestive care diet. Treatments previously prescribed by the referring veterinarian to address the regurgitation included metoclopramide\(^a\) (0.35 mg/kg [0.16 mg/lb], PO, q 12 h) and cimetidine\(^b\) (5 mg/kg [2.3 mg/lb], PO, q 12 h), which the dog had been receiving for 2 months with no improvement.

Physical examination revealed a poor body condition score (2/5), but other findings were unremarkable. Results of neurologic examination were also unremarkable, as were results of a CBC and serum biochemical analysis (including serum sodium, potassium, magnesium, and ionized calcium concentrations). Plain thoracic radiography revealed marked diffuse gaseous distension of the esophagus associated with a ventral alveolar pattern in the left cranial lung lobe consistent with aspiration pneumonia (Figure 1). Barium esophagography revealed diffuse, global esophageal dilatation with no evidence of an intraluminal filling defect. At this point, differential diagnoses included a distal esophageal obstruction (stricture, foreign body, or neoplasia), motility disorder (acquired idiopathic megaesophagus, focal myasthenia gravis, or esophageal achalasia), or other causes (toxicant ingestion, esophagitis, or hypoadrenocorticism).

Esophageal motility was evaluated by means of a videofluoroscopic swallowing evaluation. For this evaluation, the dog was given liquid barium sulfate (3 mL/kg [1.4 mg/lb]) and a 50% mix of barium and soft food. Fluoroscopic imaging was subsequently performed, revealing an unremarkable pharyngeal phase of swallowing with unremarkable primary and sec-
ondary esophageal peristaltic waves. However, a reduction in the passage of food through the gastroesophageal junction was noticed. A motility disorder was therefore excluded as a diagnosis because these findings were compatible with distal esophageal or cardial subobstruction. Focal distal myasthenia gravis could not formally be excluded owing to the delay in obtaining test results, and further diagnostic imaging was performed to investigate gastroesophageal function.

Noninvasive transhepatic ultrasonography revealed focal, circumferential thickening in the muscular layer of the gastroesophageal junction wall oral to the cardia (length of lesion, 1.4 cm; width of lesion, 3.5 cm; width of muscular layer, 14 mm; Figure 2). This finding was consistent with an infiltrative parietal lesion, and the differential diagnoses included neoplasia, granuloma, or circumferential hypertrophy secondary to achalasia. A thoracic CT scan with contrast enhancement confirmed the presence of a circumferential, intraparietal mass of the distal portion of the esophagus at the level of the esophageal hiatus and revealed no metastases in the lungs or regional lymph nodes.

Gastroscopy was then performed with an 8.8-mm pediatric videogastroscope (Figure 3). Progression of the endoscope into the stomach was difficult owing to presumed high tonus of the lower esophageal sphincter. No luminal mass or mass effect was visible. Furthermore, no medication known to affect lower esophageal sphincter motility had been administered (eg, midazolam, propofol, or isoflurane). Retroflexion was performed to inspect the cardia region, which appeared grossly normal. No ulceration or inflammation was noted. A PEG tube was placed for nutritional support. An antacid (esomeprazole; 0.7 mg/kg [0.32 mg/lb], PO, q 24 h) and a cytoprotective agent (sucralfate; 2 doses, PO, q 24 h) were prescribed.

Owing to the absence of a mucosal lesion, biopsy via endoscopy was deemed of little usefulness. Considering the size of the mass, and to provide a 1-cm margin around the suspected neoplasm, resection of 6 cm of the esophagus was deemed necessary. However, the complication rate for esophageal surgery in dogs is report-
edly high (33% mortality rate following resection of a third of the thoracic portion of the esophagus). Consequently, a dual minimally invasive treatment approach was chosen, consisting of surgical diagnostic biopsies performed via lateral thoracotomy and placement of a self-expanding, covered, nitinol esophageal stent to palliate the obstruction.

The dog was premedicated for the procedures with methadone (0.2 mg/kg [0.1 mg/lb], IV) and midazolam (0.2 mg/kg, IV), anesthesia was induced with propofol\(^b\) (5 mg/kg, IV), and the dog was endotracheally intubated. Anesthesia was maintained with isoflurane in oxygen, and mechanical ventilation was provided. The dog was positioned in right lateral recumbency. A 5-cm-long intercostal thoracotomy was performed between the left 9th and 10th ribs in the dorsal quarter of the thorax. Palpation of the esophagus revealed a lesion at the level of the esophageal hiatus that was deemed unresectable because of extensive and diffuse muscular thickening. The muscular layer was incised without penetrating the esophageal lumen, and the lesion was bluntly dissected. A 1 X 1-cm biopsy specimen was collected, and the esophagus was sutured with 4-0 monofilament synthetic absorbable material. A pleural catheter was then placed, and the thoracic wall was closed routinely.

A self-expanding, covered, nitinol esophageal stent\(^1\) (diameter, 12 mm; length, 80 mm) was inserted into the esophagus under esophagoscopic guidance. The stent was first positioned aborally until it partially extended into the stomach, and then it was deployed under external control (Figure 1). The stent was fixed in the gastric mucosal layer by gastrostomy with 3 sutures of 4-0 monofilament synthetic absorbable material.\(^1\) Postoperative thoracic radiography was performed to confirm appropriate stent positioning (Figure 1).

The dog recovered well from anesthesia. Postoperative medical treatment included gastroprotectants (sucralfate and esomeprazole), an antiemetic (maropitant sulfate\(^a\)); 1 mg/kg [0.45 mg/lb], SC, q 24 h), antimicrobials (amoxicillin–clavulanic acid\(^c\); 20 mg/kg [9.1 mg/lb], PO, q 12 h), and metronidazole\(^a\) (15 mg/kg [6.8 mg/lb], PO, q 12 h). For analgesia, the dog received morphine at a dosage of 0.3 mg/kg (0.14 mg/lb, IV, q 4 h for 24 hours) and then at 0.2 mg/kg (IV, q 4 h for 2 days), then buprenorphine at 20 µg/kg (9 µg/lb, IV, q 6 h for 2 days), and finally tramadol at 3 mg/kg (PO, q 12 h for several days at home). Feeding was performed through the PEG tube. Histologic examination of the biopsy specimen revealed findings consistent with esophageal leiomyoma.

One week after stent placement, regurgitation frequency had decreased to once a day. The PEG tube was still in place. Radiography revealed a 1.5-cm displacement of the stent toward the gastric lumen (Figure 1).

Two weeks after stent placement, frequent vomiting was reported. Radiography revealed complete migration of the stent into the gastric lumen. A gastrotomy was performed with the dog anesthetized (same protocol as before) to securely attach the esophageal portion of the stent. The stent was repositioned in the esophagus and fixed in the stomach with 4 absorbable sutures in a cruciate pattern.

Three weeks after stent placement, the owner reported that the dog’s clinical signs had progressively improved. The dog was able to eat the total number of calories required to achieve a healthy body weight. Appropriate positioning of the stent was confirmed by thoracic radiography. The PEG tube was removed 2 months after stent placement.

No additional regurgitation was reported at the 9-month follow-up examination, by which time the dog had regained a healthy body condition. Plain radiography revealed the absence of esophageal dilatation but migration of the stent into the gastric lumen (Figure 1). The control CT scan\(^d\) revealed a persistence of the focal circumferential eccentric thickening of the wall of the cardia without evidence of progression (Figure 4). A control esophagoscopic examination performed with the dog anesthetized allowed easy progression of the endoscope through the cardia to the stomach. No inflammatory lesion was observed. The stent was easily and definitively removed by gastroscopy.

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\(^{a}\) Small Animals, Exotic, & Avian

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Figure 3—Endoscopic image of the aboral portion of the esophagus in the dog of Figure 1 before (A) and after (B) stent placement.

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Figure 4—Computed tomographic images in the transverse plane (A), sagittal reconstruction (B), and dorsal reconstruction (C) at the level of the cardia in the dog of Figure 1 obtained 9 months after stent placement. Images were obtained in the standard soft tissue window. In panel A, notice the persistence of a circumferential eccentric thickening of the wall of the cardia (between calipers) and the absence of progression, compared with findings in previous CT examinations (Figure 2). The pericardial mass is visible (arrowheads).
Two years after stent placement, the dog remained free of the previously observed clinical signs.

**Discussion**

To the authors’ knowledge, transcardial placement of an esophageal stent to relieve neoplastic obstruction has not been reported before in the veterinary literature. Diffuse benign muscular neoplasia should be considered as a differential diagnosis for acquired esophageal dilatation in adult and older dogs, as suggested in a previous case report. A few reported cases of esophageal achalasia in dogs, involving esophageal dilatation secondary to abnormal tonus of the lower esophageal sphincter, may have some similarities with the case reported here. Nevertheless, although no swallow-pressure topographic assessment was performed for the dog of the present report, the evidence obtained via diagnostic imaging and histologic evaluation of a full-thickness biopsy specimen indicated leiomyoma, with secondary acquired megaesophagus.

Diagnosis of esophageal tumor usually involves standard radiography and endoscopy. As was performed for the dog of the present report, contrast radiography and fluoroscopy may be used to identify an intraluminal mass or esophageal dilatation oral to the lesion. Transabdominal ultrasonography of the gastroesophageal junction has been used in healthy and clinically affected humans. Healthy and abnormal gastroesophageal junctions in dogs, including in a dog with a leiomyoma, were characterized in a previous study. Five ultrasonographic layers, with a mean ± SD total thickness of 10.8 ± 1.6 mm, were identified in the esophageal wall of healthy dogs weighing from 20 to 29.9 kg (44 to 65.8 lb) in that study, compared with a muscular layer of 14 mm in the dog of the present report; however, calculation of a ratio between thicknesses at 2 different esophageal regions would have provided a more useful comparison in this respect. The precise measure of the lesion in the dog of the present report was confirmed via transabdominal ultrasonography.

Esophagoscopy allows exploration of a possible distal esophageal subobstructive neoplastic lesion and visual inspection of secondary lesions (e.g., inflammation, degree of obstruction, hemorrhage, or ulcerative lesions). For the dog of the present report, esophagoscopy allowed confirmation of the absence of mucosal involvement and distal and focal narrowing of the esophageal lumen. Lesion biopsy with forceps could not have been an effective diagnostic approach because such action may have only harvested the superficial esophageal layers. Moreover, biopsy is contraindicated for leiomyomas in humans because of the risk of secondary infection, hemorrhage, and perforation. A definitive diagnosis was possible for the dog of the present report only through histologic evaluation of a biopsy specimen obtained via thoracotomy.

The consensus in human medicine for obtaining a definitive histologic diagnosis in patients with symptomatic esophageal leiomyoma is to consider surgical resection. Reports of such surgery in veterinary medicine are rare, and the procedures that have been described are associated with numerous complications (e.g., poor exposure, narrow surgical approach, presence of tension at anastomosis, dehiscence, fistula formation, or stricture). The lesion in the dog of the present report was diffuse, and the dog was in poor body condition, so a minimally invasive procedure was selected. A literature search revealed reports of a total of 7 dogs with distal esophageal leiomyoma (at the level of the lower esophageal sphincter in the distal portion of the esophagus or at the level of the diaphragm) that were treated surgically (5 for which treatment was a success, 1 that was euthanized, and 1 that was lost to follow-up).

For several years, stents have been used in human medicine to manage dysphagia resulting from benign esophageal strictures or to palliatively treat obstructive esophageal cancer. In contrast, only a few veterinary reports describe the use of esophageal stents to palliate a malignant esophageal tumor in a dog and refractory benign esophageal stricture in dogs and cats. In a study involving 84 dogs with experimentally induced esophageal achalasia, performance and efficacy of temporary transcardial placement of a covered stent were evaluated. The stents in that study were well tolerated and led to improved cardial function. For the dog of the present report, after selection of an appropriate stent length and a transcardial position, a covered stent was chosen because the histologic characteristics of the esophageal lesion were unknown at the time of stent placement. Moreover, the stent could have been removed had the dog developed signs of discomfort or pain attributable to the stent being placed in an atypical position.

Nevertheless, covered stents are also associated with a higher migration rate than noncovered stents, even though the flared ends are designed to facilitate anchorage. In a previous study, stent migration occurred in 15 of 60 dogs with experimentally induced achalasia in which a covered stent was placed. Stent migration has been identified in dogs 3 and 8 months after placement, even when the stent was sutured in place. In the dog of the present report, stent migration was observed 2 and 9 months after the procedure, despite the intragastric suture. Reasons for this migration could include superficial placement of the sutures in the gastric mucosa, esophagogastric motility, or acid dissolution of the absorbable component of the suture material.

Some innovations, such as silica gel membranes or antierosion coating, may enhance stent resistance to gastric acidity. Despite these improvements, in human studies, stent migration occurred in 12% to 67% of patients treated for benign stricture with a partially and totally covered metallic stent or plastic stent. However, our clinical observations suggested that the temporary presence of the stent in the dog of the present report helped to dilate the distal portion...
of the esophagus and led to considerable improvement of clinical signs. These observations were corroborated by the results of a previous study involving dogs with achalasia, in which the optimal timing of stent retrieval to ensure maximum improvement of esophageal function was between 2 weeks and 1 month after placement.

Signs of stent-associated discomfort have been reported for dogs. Despite opioid treatment, the dog of the present report had persistent regurgitation, apparent nausea, and vomiting during the first 2 weeks after stent placement, which presumably resulted in stent migration. Although the possibility of pain could not be ruled out, this apparent intolerance might have been due to the transcardial position of the stent, even though this has not been reported. Similar signs have been reported for humans, who can develop reflux esophagitis and rare chest pain following stent placement.

The dog of the present report remained free of previously observed clinical signs for almost 2 years after the stent was repositioned. No signs of dysphagia were observed. We surmised that the radial force exerted by the stent against the esophageal wall helped to reduce the obstruction. A limitation in case management was the lack of performance of a control swallow evaluation to assess esophageal motility at the 9-month follow-up examination. The prognosis for the dog was difficult to predict. Of the 7 reported cases of esophageal leiomyoma in dogs that were left untreated or treated surgically, dogs were free of clinical signs from 2 to 37 months after the surgery or stent placement. However, follow-up information was incomplete for some patients.

The present report described the use of a transhepatic ultrasound approach and stent placement for the management of benign neoplasia of the distal portion of the esophagus in a dog. Such tumors should be considered as a differential diagnosis for acquired esophageal dilatation in adult dogs. Transcardial esophageal stent placement contributed to a complete improvement of clinical signs. Moreover, suppression of the high-pressure barrier between the esophagus and stomach for several months had no apparent adverse effect on digestive function.

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**Footnotes**

a. Emeprid, CEVA Santé Animale, Libourne, France.
d. BrightSpeed, General Electric, Velizy-Villacoublay, France.
e. Video esendoscope GIF 160, Olympus, Andover, Mass.
g. Ulcer, Sanofi Aventis, Paris, France.h. Propovet, Axience, Pantin, France.i. Biosyn, Medtronic, Minneapolis, Minn.j. Ultralinx esophageal stent, Boston Scientific, Natrick, Mass.

**References**

23. Cowling MG, Adam A. Gastrointestinal stenting: indications and techniques. In: Brandon JC, Teplick SK, eds. *Nonsurgi-
From this month’s AJVR

Effect of trotting speed on kinematic variables measured by use of extremity-mounted inertial measurement units in nonlame horses performing controlled treadmill exercise

Antonio M. Cruz et al

**OBJECTIVE**
To assess effects of speed on kinematic variables measured by use of extremity-mounted inertial measurement units (IMUs) in nonlame horses performing controlled exercise on a treadmill.

**ANIMALS**
10 nonlame horses.

**PROCEDURES**
6 IMUs were attached at predetermined locations on 10 nonlame Franches Montagnes horses. Data were collected in triplicate during trotting at 3.33 and 3.88 m/s on a high-speed treadmill. Thirty-three selected kinematic variables were analyzed. Repeated-measures ANOVA was used to assess the effect of speed.

**RESULTS**
Significant differences between the 2 speeds were detected for most temporal (11/14) and spatial (12/19) variables. The observed spatial and temporal changes would translate into a gait for the higher speed characterized by increased stride length, protraction and retraction, and flexion and extension; mediolateral movement of the tibia; and symmetry, but with similar temporal variables and a reduction in stride duration. However, even though the tibia coronal range of motion was significantly different between speeds, there was large variability for this variable, which raised concerns about whether these changes were clinically relevant. For some variables, the lower trotting speed apparently was associated with more variability than was the higher trotting speed.

**CONCLUSIONS AND CLINICAL RELEVANCE**
At a higher trotting speed, horses moved in the same manner (eg, the temporal events investigated occurred at the same relative time within the stride). However, from a spatial perspective, horses moved with greater action of the segments evaluated. The detected changes in kinematic variables indicated that trotting speed should be controlled or kept constant during gait evaluation. (Am J Vet Res 2018;79:211–218)