Esophagitis in Cats and Dogs

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KEYWORDS

• Esophagitis • Gastroesophageal • Reflux • Esophageal • Biopsy • Dog • Cat

KEY POINTS

- Esophagitis is mostly a consequence of increased exposure to gastroduodenal reflux owing to various primary causes.
- An exception is eosinophilic esophagitis, an emerging primary inflammatory disease of the esophagus with a presumed allergic etiology.
- Clinical signs can vary and it can be difficult to differentiate esophagitis from other upper (mostly food-responsive) gastrointestinal diseases.
- When no extraesophageal disease can explain clinical signs, gastroesophageal reflux disease owing to an incompetent lower esophageal sphincter, similar to the well-known condition in people, is suspected.
- Esophagitis owing to gastroesophageal reflux can be confirmed with wireless esophageal pH-monitoring (Bravo capsule), endoscopy, or esophageal endoscopic biopsies and histology.

Video content accompanies this article at http://www.vetsmall.theclinics.com.

INTRODUCTION

Esophagitis denotes a localized or diffuse inflammation of the esophageal mucosa. It is generally thought to result from a caustic or chemical (ie, gastric acid, bile acids) injury. In humans, the most frequent mechanism causing esophagitis is gastroesophageal reflux (GER) leading to GER disease (GERD). GERD is thought to result from lower esophageal sphincter (LES) incompetence.[1] In small animal medicine, GERD secondary to a primary functional LES abnormality is poorly understood, most probably because diagnosing GERD based on a detailed questionnaire on perceived symptoms, as it is done in human medicine, is not applicable. The situation becomes even more complicated when considering that the nonerosive form of GERD (ie, absence of visible lesions on esophagoscopy and the presence of reflux-associated symptoms) comprises the majority of patients in human medicine.[1]

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It should also be noted in this context that, independent of the cause of esophagitis, esophageal inflammation in itself can cause esophageal hypomotility and thus gastroesophageal sphincter weakness by impairing the excitatory cholinergic pathways to the gastroesophageal sphincter. In cats, induction of experimental esophagitis has been shown to attenuate the release of acetylcholine and lowered gastroesophageal pressures. These changes were reversible on healing of the esophagus.[2,3]

CONDITIONS ASSOCIATED WITH ESOPHAGITIS

Esophagitis is for the most part not an independent disease in cats and dogs, but develops secondary to gastro(duodenal) reflux, which in turn can have various causes. The most common scenario for esophagitis in cats and dogs is perianesthetic reflux. Clinical signs usually begin a couple of days after anesthesia, and the diagnosis is usually based on a combination of medical history and ensuing clinical signs. It has been reported that intraabdominal procedures have a higher risk for GER, also the duration of preoperative fasting and choice of preanesthetic drugs influence the incidence of GER during anesthesia.[4–7] In extreme cases, lethal esophageal rupture secondary to perianesthetic acid reflux is possible[8]; however no information exists on associations between magnitude of intraoperative esophageal acid exposure and the risk of subsequent esophageal inflammation. Further causes are medication-induced cases of esophagitis. Cats seem to be particularly susceptible to pill-induced esophagitis when receiving peroral medication, and antibiotics such as clindamycin and doxycycline (doxycycline hyclate is known to cause a strong acidic solution [pH 2–3] compared with doxycycline monohydrate) have been frequently implicated.[9,10] Thus, the routine administration of a small water bolus to facilitate esophageal clearance is recommended in this species.[11] Further causes are lodged foreign bodies, frequent vomiting, malpositioned esophageal feeding tubes, and distal esophageal neoplasia (often leiomyoma),[12,13] all of which permit increased exposure of the esophageal mucosa to gastro(duodenal) reflux. Primary gastric hyperacidity conditions such as Zollinger–Ellison syndrome[14,15] or delayed gastric emptying owing to acute pancreatitis or pyloric outflow obstructions can also lead to esophagitis when refluxing gastric contents injure the esophageal mucosa. Hiatal hernias also predispose to esophagitis in dogs and cats because of the altered functional anatomy of the gastroesophageal pressure barrier (loss of the intrinsic support of the crural diaphragm) and impaired esophageal acid (Video 1).[16–18] Hiatal hernias and associated GER resulting from upper airway obstruction are common problems in brachycephalic dog breeds.[19–21] Less commonly, non–breed-specific upper airway obstruction may also cause reflux esophagitis.[22,23] The supposed pathomechanism is the negative intrathoracic pressure generated by increased inspiratory effort. Yeast esophagitis (*Candida* spp) can rarely develop as a secondary complication in cases of idiopathic megaesophagus owing to the chronic effects of lodging food.

Probably the only primary inflammatory esophageal disease without an underlying condition predisposing to GER is eosinophilic esophagitis (EE). This entity is a recently recognized inflammatory disorder of the esophagus in dogs and cats suspected to be a hypersensitivity disorder that results in marked accumulation of eosinophils within the esophageal tissue.[24,25] Eosinophil presence and activity then results in tissue damage, edema, inflammation, and fibrosis. Only case reports have been reported up to now and it is currently unclear whether EE is underdiagnosed in cats and dogs.

Reflux esophagitis owing to a LES incompetence similar to GERD in humans is also frequently suspected in dogs and cats based on a variety of clinical signs assumed to reflect esophageal pain. Specific diagnostic criteria for GERD are lacking and a

tentative diagnosis is made based on clinical and endoscopic findings, exclusion of other esophageal and extraesophageal diseases, and an adequate response to treatment.[18,26–29] The crux is that diagnosing GERD in people is largely based on symptom perception, and ideally a close correlation between reflux and symptom during ambulatory esophageal pH-metry.[1] However, although symptoms are by definition inherently subjective experiences and communicated to the health care professional by the human patient, cats and dogs naturally exhibit clinical signs that can or cannot reflect sensations associated with esophagitis. This is why GERD is mostly a diagnosis of exclusion in our patients and ideally based on a clear response to treatment. Recently, the 3-year incidence of suspected canine GERD presented to a specialty practice was estimated at 0.9%, diagnoses have been based on a combination of historical and clinical signs, as well as radiographic, endoscopic, or histopathologic findings without actual demonstration of increased esophageal reflux events.[29] It is still controversial if GERD is truly an independent disease in cats and dogs, because inflammatory small bowel disease can present with similar clinical signs.[28,30] In the authors' experience, many patients with clinical signs originally attributed to reflux esophagitis have chronic inflammatory enteropathies and can in fact be successfully managed in the long term with dietary changes, high-dose multistrain probiotics or budesonide.

Cats with chronic gingivostomatitis have recently been shown to have a high incidence of concurrent esophagitis based on endoscopy and histology.[31] It remains difficult to determine to what extent esophageal inflammation causes clinical signs in these patients as clinical features are limited and cannot distinguish with certainty stomatitis from esophagitis. Dogs with idiopathic laryngeal paralysis can also have increased acidic reflux events compared with clinically normal dogs with refluxes even reaching the most proximal part of the esophagus.[32] However, these findings were not accompanied by endoscopic evidence of esophagitis and esophageal biopsies were not collected in that study.

CLINICAL SIGNS

Esophagitis may lead to immediate signs caused by inflammation or delayed signs caused by stricture formation. Animals with mild esophagitis may show no clinical signs, whereas animals with more severe esophagitis can show decreased appetite, anorexia, odynophagia or dysphagia, ptyalism, increased empty swallowing motions, extension of head and neck while swallowing, retching, vomiting, regurgitation, sudden unexplained discomfort, belching, drooling, excessive grass eating, or surface licking. Brachycephalic breeds (especially French Bulldogs) usually show a mixture of regurgitation and retching of frothy material mostly during increased activity or excitement.

Clinical signs reported in the literature include retching, gagging, repeated swallowing motions, smacking, discomfort at night, sudden nervousness, restlessness or discomfort, and refusal to eat despite apparent interest in food.[18,24–26,28,29]

Pruritus and other manifestations of allergic skin disease may be seen in cases of EE. Concurrent borborygmi may indicate that it is a small bowel disease after all, even in the absence of diarrhea. Although it would seem to be plausible that the severity of clinical signs depends on the extent and depth of esophageal lesions, they do not always correlate and may vary greatly. Hoarseness, stridor, and cough could reflect injury to the epiglottis, larynx, and upper airway.[30] The onset of anesthesia-associated reflux esophagitis varies from 1 to 3 days to 2 weeks after a causative anesthetic event.[33,34] On physical examination, patients may have evidence

of halitosis and laryngeal signs with redness, hyperemia, and edema of the vocal folds and arytenoids. However, the majority of patients have normal physical examinations. Clinical signs of strictures depend on the degree of obstruction; the narrower the stricture the more pronounced is the regurgitation; however, regurgitation also could be caused by painful swallowing. Strictures closer to the pharynx may cause more immediate regurgitation. Mild to moderate strictures may cause no clinical signs until the patient swallows a large food bolus or firm food (**Fig. 1**A, B). Sometimes there is gradual progression in severity as the stricture continues to contract, causing greater luminal narrowing.

Care should be taken to evaluate the respiratory system as, analogous to humans, it is surmised that esophagitis can also cause inflammatory airway disease. Pulmonary manifestations of esophagitis may include aspiration pneumonia, chronic bronchitis, and potentially interstitial pulmonary fibrosis. Concerning this aspect, it is of particular interest to note that idiopathic pulmonary fibrosis is seen nearly exclusively in older West Highland White Terriers, a breed that is at the same time notoriously famous for lodged esophageal foreign bodies.[35–37] Chronic intermittent microaspiration of gastric acid secondary to an esophageal dysmotility may be a contributing causative event in this breed. A recent study found that, although gastric juice microaspirations occurred in various canine respiratory diseases, bile acids measured in bronchoalveolar lavage fluid as a surrogate marker for microaspirations of gastroduodenal content were detected only in healthy West Highland White Terriers.[38]

DIAGNOSIS

Diagnostic Imaging

Although historical and clinical findings may be suggestive of esophagitis, the results of routine laboratory testing are usually normal. Radiography is of limited use for the detection of esophagitis; compatible findings may be mild esophageal dilations or fluid

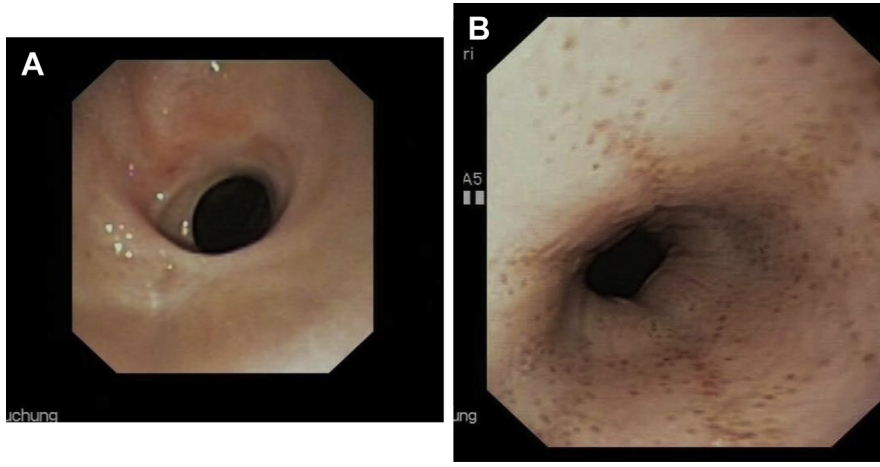


Fig. 1. (*A*) Partial midthoracic esophageal stricture in an 8-year-old King Charles Cavalier Spaniel with esophagitis secondary to a lodged foreign body (chewing bone) that had been sitting in the esophagus for 2 days. The dog started to regurgitate again 5 days after removal of the foreign body when fed a regular amount. Small portion were well-tolerated. (*B*) Distal esophagus of the same dog with multiple brownish dots representing a proliferative response of submucosal glands to increased acidic reflux.

accumulation in the distal esophagus. However, foreign bodies, hiatal hernias, esophageal dilation, ring anomalies, or masses could be detected and a pathologic lung pattern may reflect aspiration injury to the lungs. Mediastinal or pleural air or liquid accumulation may indicate esophageal perforation. If a perforation is considered likely, an iodinated contrast medium should be used instead of barium. Although contrast esophagrams are inexpensive, readily available, and noninvasive tests, they are only useful in demonstrating stenotic narrowing or intramural masses of the esophagus. The sensitivity of barium esophagram to detect esophagitis is low. Mucosal irregularities and a prolonged retention of the contrast medium can be seen with moderate to severe inflammation, whereas milder forms of esophagitis will be missed.[4] Fluoroscopic swallow studies have the benefit to assess esophageal motility during the whole swallow with less of a chance to miss the moment, when the contrast medium passes a narrow point, as could be the case with static images. It is of importance to do wet swallows with liquid contrast medium and dry swallows with a barium–food mixed bolus, because liquids can sometimes pass a partial stricture, whereas a food bolus may be retained. Although swallow studies can assess causes (reflux episodes, hiatal hernia [see Video 1], mural masses) as well as consequences (dysmotility, strictures, reflux episodes) of esophagitis, it is not a useful modality to diagnose esophagitis.

ENDOSCOPY

Endoscopic examination is the most sensitive method to diagnose esophagitis, although reliable endoscopic criteria for diagnosis of esophagitis as well as grading of severity of esophagitis have not been established in small animals. Of note, no descriptive endoscopic work larger than case reports has been published on canine or feline esophagitis, and it is likely that subtle lesions go undetected. Only 1 in 22 dogs presenting with clinical signs attributed to esophagitis also had endoscopic evidence of esophagitis in a recent study.[28] Early signs of esophagitis are erythema and edema usually above the LES (**Fig. 2**), but these findings depend on the quality of endoscopic equipment. Other signs include increased vascularity, because enlarged capillaries develop in response to acid near the mucosal surface (**Fig. 3**A, B). Prolonged acidic injury leads to proliferation of submucosal esophageal glands in dogs,[39] and their excretory ducts can readily be seen as round dots (**Fig. 4**). Another common sign is increased granularity; the mucosal surface seems to be rough and puckered (**Fig. 5**). Findings compatible with severe esophagitis are areas of exudative pseudomembranes and ulcerative mucosa. The esophageal mucosa of published cases of EE of cats and dogs seems to be clearly abnormal with marked proliferative mucosal lesions similar to a cobblestone appearance, friable and hyperemic mucosa with exudative and ulcerated mucosa, and the presence of benign structure formation.[24,25] Typical gross endoscopic findings in humans with EE comprise concentric rings, furrows, exudate, and strictures. However, a normal endoscopic examination has been reported in up to 40% of cases and EE can be missed without biopsies.[40] This author has been actively looking for EE in esophageal biopsies from patients with compatible clinical signs and unremarkable esophageal linings, but has not found a case as of yet. Although typical for reflux esophagitis, circular inflammation just above the LES should not be confused with the squamocolumnar junction (the demarcation line between the squamous esophageal lining and the columnar gastric lining, the so-called z-line), that may seem to be sharply delineated in cats and dogs with erythematous and reddened gastric mucosa. This is especially the case with esophageal overinsufflation. Strictures are usually obvious at endoscopy; exceptions could



Fig. 2. Distal esophagitis in a 1-year-old male French Bulldog presenting for daily regurgitation. Streaky erosions can be seen just above the LES (*arrows*). Final diagnosis was reflux owing to brachycephalic obstructive airway syndrome.

be large breeds with esophageal diameters much bigger than the scope where strictures could be missed. In these cases, generous air insufflation will usually help to demonstrate the focally reduced esophageal diameter. The endoscopic examination should always include a full gastric inspection with special attention to the cardia and pylorus to exclude underlying abnormality, such as obstructive leiomyoma or radiolucent foreign bodies, to confirm that the esophagitis is the primary problem. Concurrent duodenoscopy and biopsies seems reasonable given the unspecific clinical signs that could also reflect the presence of small bowel disease.

Narrow band imaging uses only blue and green light that is absorbed by vessels but reflected by mucosa, and can better capture the microstructures of the superficial mucosa and reveal subtle changes of patients with esophagitis in human medicine.[41] The value of narrow band imaging has still to be determined in canine and feline esophagitis (**Fig. 6**).

ESOPHAGEAL HISTOLOGY

It could be argued that esophagoscopy without biopsy is insufficient to rule out esophagitis, because cases with grossly normal appearing mucosa on endoscopy and necropsy, but histopathologic evidence of esophagitis, have been reported in cats and dogs.[12,26,31,42,43] This finding is in accordance with findings in humans, where endoscopic findings in patients with GERD vary from no visible mucosal damage (termed nonerosive esophageal reflux disease) to esophagitis, peptic strictures, or Barrett's esophagus.[1] Unfortunately, it can be difficult to obtain adequate esophageal biopsies, except in the more severe cases of esophagitis, because the esophagus is lined with a robust stratified squamous epithelium, and even repeated biopsies with serrated forceps from the same location may yield only epithelial bits. Münster and colleagues[44,45] advocate taking biopsies from the gastroesophageal junction using standard forceps

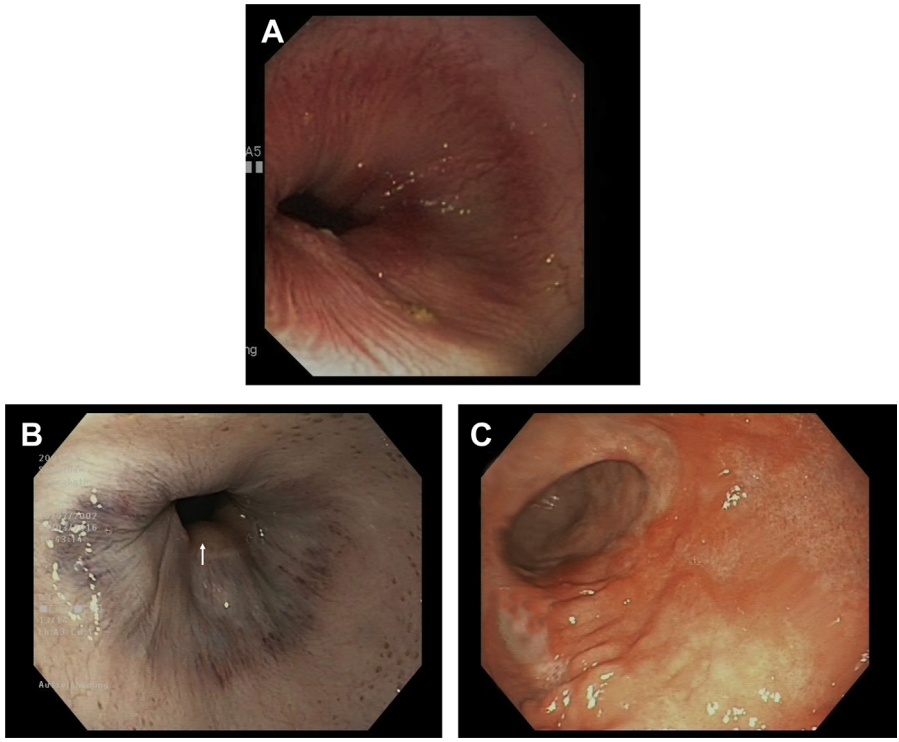


Fig. 3. (*A*) Distal esophagitis just above the LES in a 6-year-old female spayed Bernese Mountaindog presented for inappetence. No cause could be identified. (*B*) Esophagoscopy of a 9-year-old Labrador Retriever presented for increased lip smacking and empty swallowing. There is circumferential increased vascularity around the LES beginning at the Z-line (*white arrow*). Gastroscopy (*C*) of the dog under 3B revealed increased granularity, erythema, and protruding bulges. Histopathology revealed severe lymphocytic gastritis with fibrosis, a differential diagnosis was small cell lymphoma.

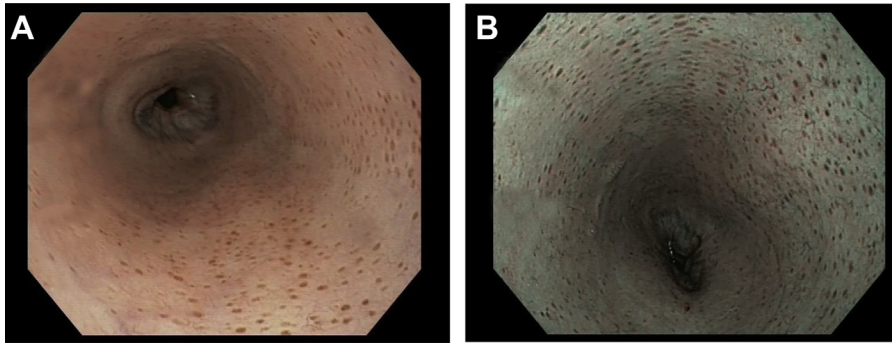


Fig. 4. (*A*) Same dog as in Fig. 3B and C. Prolonged acidic injury leads to proliferation of submucosal esophageal glands and their excretory ducts can be seen as round brown spots. (*B*) Narrow band imaging.



Fig. 5. Distal esophagus of an 18-month-old male neutered Maine Coon cat 14 months after surgical correction (Y-U cardioplasty) and subsequent ballooning of a congenital esophageal stenosis. The cat could eat again and thrived to a normal sized clinically healthy Maine Coon cat. A side effect of the surgery was incomplete closure of the LES, and twice daily omeprazole was prescribed. The esophageal mucosa seems to be edematous, with patchy erythema and increased granularity, the typical ringed structure of the feline esophagus is only partially visible.

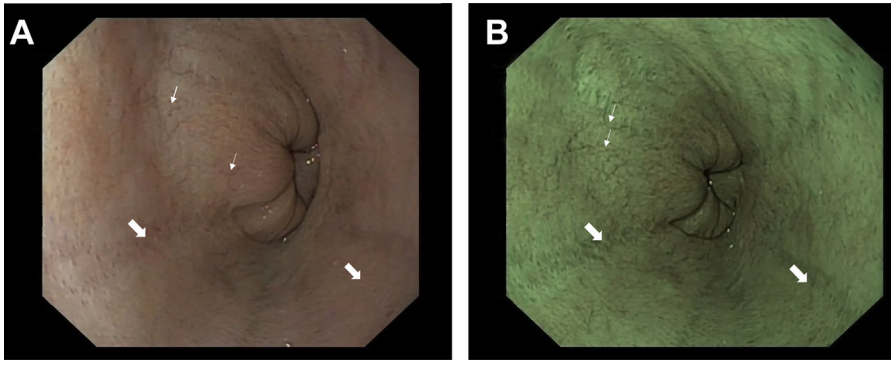


Fig. 6. LES of an 8-year-old male Jack Russel Terrier with intermittent vomiting, lip smacking, and refusal to eat. White light endoscopy (*A*) shows mild mucosal vein dilation (*thin arrows*) and discrete longitudinal stripes (*thick arrows*) reflecting activated excretory ducts of submucosal glands. (*B*) Narrow band imaging (*blue green* endoscopy) accentuates these findings. Wireless esophageal pH-monitoring revealed a fractional time pH less than 4 of 1% (0%–3.1%). Histopathology revealed moderate gastric and duodenal lymphoplasmacytic inflammation. No improvement was seen with gastric acid suppression, and clinical signs finally disappeared with a novel protein diet and budesonide.

for single use with open elliptical branches, and a lancet. Structural microscopic abnormalities such as fibrosis, inflammation, elongation of the stromal papillae, and increased thickness of the basal cell layers can be found in biopsies comprising only the squamous epithelium and the lamina propria mucosae.[27,29] Before the advent of esophageal pH-metry, esophageal histology was used in the research on GERD in people, and hyperplastic thickening of the basal cell layer and abnormal elongation of stromal papillae of the lamina propria were found to reflect excessive regeneration and thus were considered pathognomonic for GERD in humans.[46,47] Histologic evidence of hyper-regeneratory changes in esophageal biopsies was recently also reported in 20 dogs presenting with clinical signs suggestive of reflux esophagitis (**Fig. 7**).[29] So far, the sensitivity of these histologic lesions for the diagnosis reflux esophagitis has not been evaluated against a quantifiable gold standard, such as intraesophageal pH-metry, but the predominant clinical signs regurgitation and ptyalism improved in 8 of 11 dogs with hyper-regeneratory esophagopathy after treatment with proton pump inhibitors.[29] The specificity of esophageal histologic lesions has not been assessed so far, and this endeavor seems to be challenging because clinically silent reflux esophagitis cannot be ruled out with certainty. But still, hyper-regeneratory esophageal lesions were significantly less common in age-matched control dogs (n = 19) undergoing endoscopy for clinical signs unrelated to the esophagus (eg, chronic diarrhea).[29] Clearly, further work is necessary to clarify the prevalence and thus significance of esophageal histologic lesions of cats and dogs. Endoscopic findings of severe chronic acid exposure such as Barrett's esophagus (replacement of the normal squamous epithelium of the distal esophagus with metaplastic columnar epithelium) are exceedingly rare in cats and dogs, further illustrating that GERD might not be as common as in humans.[18,48]

In contrast, the histologic findings of EE seem to be straightforward; eosinophils are not found in the healthy esophagus, making the presence of these cells indicative of disease. Guidelines in humans recommend that a diagnosis of EE is made when a patient presents with symptoms of esophageal dysfunction and esophageal biopsies demonstrate 15 or more eosinophils in a high power field in the absence of competing



Fig. 7. pH capsule attached to the distal esophageal mucosa for continuous esophageal pH monitoring in a dog with suspicion of reflux esophagitis.

causes such as GERD.[49] Reported eosinophil counts in mucosal biopsies of cats and dogs with EE ranged from 20 to 40 eosinophils per high power field.[24,25]

ESOPHAGEAL pH-METRY

All these procedures may aid in the diagnosis of esophagitis, but still fail to detect and quantify reflux when GERD is suspected. In humans, catheter-free esophageal pH monitoring has become the gold standard in diagnosing GERD. This technique not only provides information on esophageal acid exposure, but is also able to assess symptoms associated with acid reflux episodes. A widely used system in humans is the Bravo system. It includes a small capsule (26.0 mm × 5.5 mm × 6.5 mm) containing an antimony pH electrode with internal reference, miniaturized electronics with radiofrequency transmitter and battery, and a capsule delivery system, as well as an external receiver to monitor intraesophageal pH. This wireless pH monitoring system was found to be a useful tool for extended (generally 4 days; some capsules adhere up to 7 days to the mucosa and esophageal pH measurement can be extended accordingly) ambulatory catheterless esophageal pH monitoring in dogs.[28] Endoscopic capsule placement was quick and easy, and adverse clinical signs have not been observed.[28] Current disadvantages of the system are the high cost of the pH capsule, receiver, and software, as well as the need for brief anesthesia for accurate placement. Our clinical experience indicates that this technique seems to be safe and patients from 6 to 55 kg body weight tolerate the measurements well. The capsule is positioned in the distal esophagus 3 to 5 cm above the LES and attached (ie, pierced) to the mucosa (see **Fig. 7**). Mucosal attachment is achieved by the use of vacuum suction and a lock and pin mechanism. Once released from the delivery system, pH data are recorded by a receiver attached to the dog's collar and owners are instructed to maintain a logbook to record all events presumed to be related to GER. The receiver has so-called symptom buttons that can be individually programmed. At discharge, owners are familiarized with the receiver and instructed to press the appropriate buttons if the dog shows the corresponding clinical signs. An association between clinical signs and reflux is considered positive if the clinical signs occurs within 1 to 2 minutes of the reflux event. In people, the so-called symptom index is defined as the percentage of symptom events that are temporally related to a reflux episode. A symptom index of 50% usually is considered positive.[50]

Our results contradicted the hypothesis that minute amounts of acid can severely damage the esophagus in dogs. In healthy dogs, the number of refluxes (defined as an esophageal pH of <4) and the duration of long refluxes (>5 minutes) varied considerably over the course of 96 hours. The median total number of refluxes was 10 (range, 1–65), the median number of refluxes lasting longer than 5 minutes was 1 (range, 0–4), the median duration of longest reflux was 8 minutes (range, 0–27). The overall fraction time of a pH of less than 4 was low and usually ranged between 0% and 3%.[28] These numbers are actually lower compared with what has been established as normal in humans (4.0%–6.7%).[51,52] Results in dogs with clinical signs commonly attributed to reflux esophagitis were insofar surprising as clinically relevant reflux episodes could not be demonstrated in 17 of 22 suspected dogs and a temporal relationship between clinical signs observed by owners and reflux episodes could also not be established. Still, dogs with clinical signs had overall higher esophageal pH parameters when compared with healthy dogs.[28] A clear limitation of the study was that esophageal pH recordings were available from only 7 healthy control dogs and therefore no robust reference range exists for comparison of esophageal pH data at this point. Another conclusion from that study was that the differential diagnosis diet-responsive

enteropathy should be ruled out as best as possible before pursuing treatment for esophagitis, because both diseases can have similar clinical signs.[28] Since that study, we have used this system frequently in dogs with clinical signs suggestive of esophagitis, but could detect abnormal esophageal pH profiles in only a handful of dogs. In fact, complete resolution of clinical signs is rarely achieved with gastric acid suppression alone,[30] and most dogs ultimately respond to dietary changes, probiotics, and less commonly budesonide.

THERAPY

Treatment should ideally be directed at the underlying causes of the esophagitis and it is important to eliminate predisposing factors (eg, oral medication). In case of a chronic obstructive upper respiratory problem, corrective surgery minimizing or eliminating the obstruction may also resolve inflammatory esophageal lesions.[19] The same would apply to hiatal hernias. Because anesthesia is the most common cause of esophagitis, it would be desirable to prevent acidic reflux in patients undergoing anesthesia, especially in those that already have a history of anesthesia-related esophagitis. Pretreatment with high doses of metoclopramide yielded conflicting results and the administration of ranitidine before surgery also did not reduce the incidence of GER.[53,54] Although preanesthetic administration of cisapride and esomeprazole together decreased the number of reflux episodes in anesthetized dogs in a recent study, the administration of esomeprazole alone was felt to be less effective because it only decreased the acidity of refluxes but not the overall quantity of reflux episodes.[55] In cats, esophageal pH during anesthesia could be significantly increased when 2 oral doses of omeprazole were given 18 to 24 and 4 hours before anesthesia, and no reflux episodes (defined as a pH of <4) were recorded during anesthesia compared with placebo-treated cats.[56]

The same principle of increasing the LES pressure together with gastric acid suppression to prevent further injury and allow esophageal healing has been recommended in the treatment of suspected reflux esophagitis. In cats, experimentally induced esophagitis decreases esophageal peristalsis, decreases the LES pressure, and diminishes esophageal clearance.[3] These changes were reversible with healing of the esophagus.[3] Contradictory to common belief, orally administered metoclopramide administration did not result in significant changes in the LES pressure in dogs. Only cisapride administration significantly increased LES pressure in a placebo-controlled study.[57] A mainstay of treatment for suspected esophagitis is a combination of gastric acid suppression and sucralfate. In dogs and cats, proton pump inhibitors (ie, omeprazole) provide superior gastric acid suppression compared with H2 receptor antagonists (ranitidine, famotidine) and should therefore be considered more effective for the treatment of acid-related disorders.[58]

Twice daily dosing (1–1.5 mg/kg) is advised to maximize gastric acid suppression in canine and feline esophagitis. An exception may be esomeprazole in dogs, because once-daily oral dosing in a recent study also lead to an adequate increase of intragastric pH.[59] Sucralfate, an aluminum salt of a sulfated disaccharide, is a mucosal protectant that binds to inflamed tissue to create a protective barrier. It is supposed to block diffusion of gastric acid and pepsin across the esophageal mucosa and inhibit the erosive action of pepsin and possibly bile.[58] Although the rationale for its effectiveness is based on its protective adherence to denuded mucosal surface in an acidic environment, and the canine esophageal milieu is weakly alkaline,[28] clinically it seems to soothe the patient's discomfort when dosed multiple times daily.[58] In people, intensive high-dose sucralfate therapy (2 g of sucralfate given orally every 2 hours for the first

3 days, followed by 2 g 8 times daily for 21 days) has been shown to be beneficial in enhancing mucosal healing and preventing stricture formation in advanced-grade corrosive esophagitis.[60] EE has been successfully treated with dietary antigen elimination (ie, hydrolyzed diet) in a cat and with corticosteroids in a dog.[24,25] When present, strictures may need esophageal bougienage or balloon dilation. Budesonide orodispersible tablets have recently been shown to be a promising new option for the induction of clinical and histologic remission in a placebo-controlled trial in humans with EE.[61] Lifestyle changes are part of the initial management of reflux esophagitis in people and include losing weight if overweight, avoiding bed time snacks, and elevating the head at night. Although this therapy intuitively makes sense, no data are available on this strategy in small animals.

SUMMARY

Esophagitis in cats and dogs is largely a consequence of prolonged esophageal exposure to acid, which in turn can have various underlying causes. Analogous to GERD in humans, an incompetent LES is also often suspected to cause clinical signs, but the validity of these clinical signs remains currently unclear. The diagnosis can be confirmed endoscopically, with esophageal biopsies, with esophageal wireless pH monitoring or through a clear response to therapeutic trials with sufficiently dosed proton pump inhibitors. In unclear cases that do not respond satisfyingly to treatment, chronic enteropathy should be suspected and strict dietary trials should be tried out.

DISCLOSURE

The author has nothing to disclose.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found online at https://doi.org/10.1016/j.cvsm.2020.08.003.

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