REGURGITATION – MUCH, MUCH MORE THAN MEGAESOPHAGUS

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Esophageal Weakness

Acquired esophageal weakness is usually (but not always) easy to distinguish from obstruction radiographically, especially when a barium contrast radiograph is performed. However, the severity of the radiographic lesion (i.e., the degree of dilatation) does not always correlate well with the clinical severity. Acquired esophageal weakness is typically difficult to resolve because it is hard to find the underlying cause. Myopathy, neuropathy, myasthenia gravis, dermatomyositis, dysautonomia, esophagitis, Addison's disease, *Spirocerca lupi*, tick paralysis, central nervous system disease, or infiltrative non-obstructive esophageal tumors are possible causes. Generalized myopathies and neuropathies often affect the esophagus because it is composed of striated muscle in the dog. Signs of lower motor neuron disease in these patients are sometimes seen and can include loss of muscle mass, weakness, an inability to bark, or a change in the quality of the bark. Some clients report that their animal has laryngitis, which may seem likely because these pets typically have repeated respiratory infections due to aspiration pneumonia. Treatment of the myopathy or neuropathy should resolve the problem, but symptomatic therapy for the esophageal dilatation is indicated.

Generalized myasthenia gravis usually presents as weakness during exertion which resolves after resting; however, generalized myasthenia can present in a variety of ways, including apparent lameness or permanent weakness. Electromyography and assay for circulating antibodies to acetylcholine receptors are the most definitive tests. Localized myasthenia in the dog is a syndrome in which the esophagus is the only muscle which is obviously weak. Up to 25-30% of dogs with acquired esophageal weakness have this syndrome. Third degree heart block may also be seen in some patients with megaesophagus due to myasthenia. This is diagnosed in dogs with esophageal weakness by detecting serum antibodies to acetylcholine receptors. The antibodies are relatively stable and require little special handling other than refrigeration. If myasthenia is strongly suspected but the titer is negative, it can be valuable to repeat the titer as they sometimes seroconvert later. You cannot perform an edrophonium response test to diagnose localized myasthenia. Myasthenia gravis will sometimes spontaneously resolve. Treatment for myasthenia gravis that does not spontaneously resolve may include anti-acetylcholinesterase drugs and/or cytotoxic agents. Azathioprine and mycophenolate seem to be effective drugs for this purpose. In general, we try to avoid steroids as they seem to be associated with more problems. In really severe cases, we can place a percutaneous gastrostomy tube to support the patient and lessen aspiration while waiting for the drugs to have an effect. However, a gastrostomy tube will not prevent all aspiration as the dog is still swallowing saliva which can be regurgitated and aspirated.

Hypoadrenocorticism may be responsible for causing esophageal weakness, even when the serum electrolytes are normal. This is especially true in standard sized, black poodles, but it can occur in any breed. Treatment for hypoadrenocorticism is steroids, which can make the esophagus start functioning again. However, if your diagnosis is wrong and you give steroids because you suspect the dog may have hypoadrenocorticism, all you are doing is making

aspiration pneumonia and subsequent death that much more likely. Therefore, testing is much preferred to steroid trials.

Idiopathic megaesophagus (i.e., either congenital megaesophagus or acquired megaesophagus for which a cause cannot be found) can only be treated with symptomatic therapy, which usually consists of feeding the animal 3-4 meals of gruel from an elevated platform and making the pet remain in the near vertical position from 5-10 minutes after eating. Near-vertical means just that. It is useless for the dog to just lift its head up while eating; it should be standing on its back legs. The Bailey chair is a very useful device. You can find out more about it on the web (http://www.caninemegaesophagus.org/support.htm). If necessary, use a portable ladder or put the dog in a large trash can to help it remain vertical during this time. This approach is a time-honored treatment, but it does not always work. Some animals with idiopathic esophageal weakness are controlled as well (or better) if they are fed free-choice dry food from an elevated platform. Some can even be fed from the floor. Free-choice feeding encourages the pet to eat small amounts of food throughout the day, thus avoiding intermittent large meals which are more likely to be retained and further dilate the esophagus. If there is any esophageal motility remaining, the dry food may be easier for the esophagus to propel then gruel. It is difficult to predict which feeding regime will work best for a particular patient, and both of these feeding regimes may need to be tried. While most dogs with idiopathic megaesophagus die from aspiration, there are enough of them that respond well that it is very much worth trying. A reasonable percentage of dogs with idiopathic, congenital megaesophagus will spontaneously improve and have normal or near normal function. You cannot predict response to therapy or spontaneous remission; all you can do is support the patient and see what happens.

Some individuals have tried using cisapride in selected patients with idiopathic esophageal weakness that do not respond well to nutritional modification. Theoretically, cisapride would not be expected to work in these animals because cisapride primarily works on smooth muscle and canine esophagus is striated muscle. Furthermore, cisapride is expected to tighten up the lower esophageal sphincter, thus making it harder for food to pass out of the esophagus and into the stomach. Perhaps cisapride helps patients when gastroesophageal reflux is part of the problem.

More recently, achalasia-like disease has been diagnosed in some dogs with megaesophagus. Diagnosis requires fluoroscopy. If diagnosed, it can be treated with balloon dilation or injection with Botox. Sildenafil might (?) be helpful.

Esophagitis

Esophagitis is much more common than many clinicians are aware. The difficult partly arises from the fact that esophagitis can present with clinical signs that lead one to believe the dog is vomiting instead of regurgitating. Furthermore, mild esophagitis may only cause minor signs (mild regurgitation of mucus and phlegm) while severe esophagitis can cause so much pain that patients refuse to swallow water or even saliva. Because there can be so wide a range of clinical signs, it is easy to forget that esophagitis is a differential for a patient. It is critical to identify that esophagitis is present as delayed diagnosis can have serious clinical repercussions.

Substantial inflammation of the esophageal mucosa causes muscular weakness by interrupting the reflex arcs within the esophagus and/or between the esophagus and the brain. However, this weakness is not always reflected by finding megaesophagus. Most patients have very minor esophageal distention and yet can have major signs. Likewise, barium exophoras can have relatively minor changes and not reflect the severity of the esophagitis. Esophagoscopy typically shows an edematous, reddened, bleeding esophageal mucosa, + structure formation, making it the diagnostic method of choice to find esophagitis. However, in rare cases, there may be more subtle changes with thickening and discoloration (especially at the lower esophageal sphincter of cats).

Adding to this problem is the fact that there is such a wide range of causes of esophagitis. Severe esophagitis may be caused by anesthetic procedures in which animals are placed in dorsal recumbency and then have gastric acid pool in their esophagus for relatively long periods of time. However, gastroesophageal reflux from any cause can be responsible. Hiatal hernias occasionally are responsible for such reflux. Rare animals ingest caustic substances (e.g., lye), and some cats will like caustic disinfectants off their fur. However, a surprisingly large number of animals are administered caustic substances by veterinarians. In particular, tetracyclines, NSAIDs, ciprofloxacin and clindamycin are recognized as having substantial potential to cause esophagitis. Pills and capsules are notorious for lodging in the esophagus of cats, and it is therefore not surprising that doxycycline is a recognized cause of esophageal stricture in cats. Esophagitis may also be secondary to any cause of protracted vomiting. In particular, parvovirus enteritis may cause such intense vomiting that esophagitis results. If a vomiting animal has the character of its vomitus change, which seems to suggest regurgitation, consider the possibility that esophagitis has occurred secondary to the persistent vomiting. Gastrinomas (a tumor which secretes gastrin and results in massive gastric acid secretion) also causes esophagitis because of the vast and unending amounts of acid the esophagus is exposed to as the dog continually vomits. Gastroesophageal reflux may be potentiated by or even caused by esophagitis (which may be caused by reflux in the first place). Thus, there may be a positive feedback loop which can be hard to break (i.e., esophagitis causes more reflux which causes more esophagitis which causes more reflux which causes ...). Rarely there can be spontaneous inflammation, as seen with eosinophilic esophagitis in dogs. Brachycephalic dogs seem to have an increased incidence of gastroesophageal reflux, esophagitis and perhaps hiatal hernia. Finally, esophageal foreign bodies typically cause varying degrees of esophagitis. The esophagus is far more susceptible to pressure necrosis from a foreign body than are the stomach or intestines.

You should seek to prevent further gastroesophageal reflux by keeping the stomach as empty as possible by using prokinetics such as metoclopramide or, preferably, cisapride. Studies in people show that cisapride is clearly more effective than metoclopramide. The only real advantage of metoclopramide is that it can be given by injection; a useful fact in animals that are regurgitating profusely. Most importantly, gastric acid secretion should be abolished. H-2 receptor antagonists are poor drugs for achieving this goal. Omeprazole, lanosprazole, pantoprazole and esomeprazole are non-competitive inhibitors of gastric acid secretion. Therefore, these drugs can be noticeably more effective and for much longer than the H-2 blockers. Omeprazole is given at 1 mg/kg PO bid. Giving it twice daily is particularly important.

Sucralfate is of uncertain value in patients with esophagitis. Unless there is some gastric acid reflux into the esophagus (which you are desperately trying to stop in the first place), it is doubtful that the sucralfate is of much use in healing the esophagitis. However, it can help alleviate pain. Sucralfate should be administered as a slurry. In patients with severe pain that refuse to even swallow their saliva can receive viscous lidocaine (OTC).

A combination of omeprazole and cisapride seems to be the most effective medical treatment regime. Glucocorticoids have been thought to help retard fibrous connective tissue proliferation and cicatrix, but their effectiveness is uncertain (and they might predispose to infection). Placing a PEG tube seems to have some real advantages in patients with very severe disease such that they will not eat or cannot refrain from regurgitating. First, we will then know that the cisapride and omeprazole tablets will reach the stomach. Second, we will also know that the animal will receive its caloric and protein needs, and hopefully with less irritation to the esophagus than would have occurred otherwise.

Esophageal Stricture

If there is severe esophagitis, cicatrix may form and obstruction develop subsequently. Diagnosis of stricture is best accomplished by esophagoscopy IF the operator is familiar with such obstructions. It is surprisingly easy to pass a slender endoscope through a stricture and never recognize the stricture. It is also surprisingly easy to miss a partial obstruction due to a stricture with a barium esophagram. If you suspect a stricture and must use a barium esophagram to make the diagnosis, use barium mixed with solid food. Balloon-dilatation or bougienage is recommended if a stricture has occurred. Many animals need to have 2-6 dilatation procedures (all the while being treated for esophagitis), although some only need one procedure and some need more than 15. Do not try to resect the stricture unless you have had prior dilatation procedures fail.

Cicatrix (i.e., scarring) may occur after an episode of severe esophagitis from any cause (including foreign objects). It is particularly easy to miss this problem on a barium swallow if only liquid barium is used. If radiographs using liquid barium are nonrevealing, repeat the study using barium mixed with food, which is more likely to stop at a partial obstruction. Endoscopy is very good at finding these lesions; however, you must keep in mind the size of the patient as you evaluate the esophageal lumen. A partial stricture will be very obvious in a 10 lb dog or cat but may not be apparent in an 85 lb animal. Balloon-dilatation or bougienage is usually effective; it is also more likely to be successful than surgery and resection of the affected area.

For particularly difficult cases, stents may be placed in the esophagus. These must be sutured in place. The major point to remember is that if an animal starts to have problems days to weeks after anesthesia, consider strongly the possibility that an esophageal stricture has developed secondary to esophagitis. BE-tubes are the latest technique used for difficult strictures. In this technique, the owners balloon the stricture twice daily at home with an indwelling tube that doubles as a balloon and a feeding tube.

Hiatal Hernias

Hiatal hernias may be more common than suspected. Shar Pei's seem to have a relatively high incidence of hiatal hernias. They can be difficult to diagnose unless you know how to look for them. Sometimes seen on plain radiographs and simple barium contrast radiographs, the more occult cases sometimes need more aggressive diagnostics. Sometimes one must manually put pressure on the abdomen during film exposure to try to push the stomach through the hernia and into the chest so that it can be diagnosed radiographically. Endoscopic diagnosis is not always straightforward. You may need to put the endoscope into the stomach and retroflex it in order to see the abnormality. Even when found, the big question is whether the hiatal hernia is causing a problem or is an "innocent bystander". In particular, if you have an older dog or cat (i.e., > 1-2 years old) that just started having clinical signs, you should strongly consider that the hiatal hernia is a fortuitous finding that is not responsible for the clinical signs.