REGURGITATION: MUCH MORE THAN JUST MEGAESOPHAGUS

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Esophageal foreign objects usually consist of bones but may be rawhide treats, food, dental chew toys, toys, balls, rocks, wood, etc. They usually lodge at the thoracic inlet, base of the heart, or lower esophageal sphincter. A history of a patient that begins to regurgitate (as opposed to vomit) acutely is very suggestive of acquired esophageal obstruction due to a foreign object. These patients may continue to drink water, but they typically refuse solid food because the food bolus cannot pass by a partial esophageal obstruction and causes pain whenever it tries to. A casual, careless history that fails to raise the suspicion of regurgitation will typically lead the clinician to suspect an acute gastritis. However, the realization that the patient is regurgitating (as opposed to vomiting) should be a "red flag". Too often, a pet which has ingested a foreign object is treated conservatively while we wait and see if the supposed gastritis spontaneously resolves. This is problematic because foreign bodies can erode and perforate the esophagus much quicker than they would stomach or intestines.

Plain radiographs should be performed first. Bones are a common cause of obstruction, and plain films that are made with proper technique and then carefully evaluated are diagnostic in most cases. Remember that poultry bones are not as radiodense as the patient's bones, which means that excellent radiographic technique is required to see them. Foreign bodies in the esophagus can perfectly mimic pulmonarv or mediastinal masses; you often cannot tell the difference with plain radiographs. If poor contrast in the region of the esophagus, pleural effusion or pneumothorax are seen, one must seriously consider esophageal perforation and mediastinitis. If plain films are not diagnostic, then contrast films can be performed. Barium provides better contrast, but iodide is safer if there is an unsuspected perforation. Esophageal perforation may occur at variable times after ingestion of a foreign object. Even a blunt object, if tightly lodged in the esophagus, can cause ischemia and perforation in 2-3 days. The prognosis for animals with esophageal perforation and severe mediastinitis is guarded to poor, depending upon their condition at the time of diagnosis.

Endoscopy is almost always the preferred method of removing foreign objects, but fluoroscopic and surgical techniques can be effective if the operator is well trained. Rigid endoscopes allow much more control of the foreign object and are preferred to flexible scopes for removal of these foreign objects. It is especially useful to be able to pull the object into a rigid endoscope and then withdraw it and the scope as a unit, thus protecting the esophagus. The main disadvantage of rigid endoscopes is that they are often not long enough in larger dogs. Finesse is required; brut force can easily lacerate/perforate the esophagus. If a large object or a bone cannot be easily dislodged, do not force it lest you perforate a previously intact esophagus; instead, you can use rigid equipment to “chew” it up and hopefully dislodge it. If that fails,
passing a large Foley catheter behind the foreign object and inflating the balloon often helps; it
distends the esophagus (thus freeing the foreign object) and then is used to pull the object out. If
you cannot pull a foreign object out of the esophagus, you can try to push it into the stomach.
However, do not push bones or other foreign objects into the stomach unless you are sure that it
is smooth on the aborad side and will not further damage the mucosa. Finally, be careful if you
insufflate the esophagus lest you rupture a weakened area in the mucosa and/or cause a fatal
tension pneumothorax.

Fish hooks terrify many clinicians, but they can often be successfully removed
endoscopically. Fish hooks have usually penetrated the mucosa (and sometimes the muscular
tunics); you will often have to use rigid equipment to carefully force the tip of the hook back out
of the mucosa. A small hole is left, but there are very seldom any complications. After
removing the foreign object, retake plain chest radiographs to be sure that a pneumothorax
(which would indicate a perforation) is not present. Antibiotics are indicated if there is
substantial esophageal mucosal ulceration (and especially if you remove a fish hook which had
been used with various baits that can harbor anaerobic bacteria). Depending upon the amount of
damage, corticosteroids may be used to try to prevent cicatrix formation; however, it is not clear
that they are effective. Rarely there may be severe hemorrhage.

Primary esophageal carcinomas occur rarely although gastric carcinomas may spread into
the lower esophagus. Most primary esophageal tumors are asymptomatic until they become very
large. We have diagnosed a few primary esophageal tumors fortuitously when routine chest
radiographs revealed a density in the diaphragmatic lung fields. Esophageal sarcomas are
usually due to Spirocerca lupi, which is discussed below. Most esophageal tumors are secondary
to mediastinal or thyroid tissues, and they cause esophageal obstruction by extramural pressure.
Thyroid carcinomas may also invade the esophagus. The prognosis is usually poor.

Leiomyomas of the lower esophageal sphincter are occasionally seen. Endoscopy is the
best tool to find them. They are best seen when one looks at the lower esophageal sphincter
from the stomach using a retroflexed view. It is worth looking for these tumors as they are
potentially curable with timely surgery.

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 bouginage is usually effective;
it is also more likely to be successful than surgery and resection of the affected area. In general,
surgical resection should be a last ditch resort and only used if esophageal ballooning or
bouginage has failed despite repeated dilatations. However, you must use proper esophageal
balloons because Foley catheters and endotracheal tubes with inflatable cuffs will often not allow
you to dilate a dense or mature stricture. More difficult cases (i.e., those with extensive strictures
or with concurrent severe esophagitis) may benefit from a couple of techniques. Endoscopic
administration of intralesional steroids may help minimize reformation of the stricture.
Typically we put 1-2 ml of Vetalog at the site of the stricture either before or after ballooning.
Another technique is to make 3-4 equidistant cuts into the stricture using an electrocautery
device (i.e., either a snare or a knife) prior to ballooning. This helps the stricture to “break” open at multiple spots with the idea that there will be 3 or 4 smaller, less deep lacerations at the stricture site instead of one major, deep laceration which is more likely to restrict. However, you should not attempt to use cautery through an endoscope unless you have some training less you cause too much trauma to the tissues or destroy your endoscopic equipment.

Another technique is to “paint” the site where the stricture was broken down with Mitomycin C (NOT mithromycin C, there is a difference). A 5 mg bottle is reconstituted and soaked up into a gauze sponge. Then this sponge is endoscopically placed on the site where the stricture was broken up for 5 min. Then it is flushed off with 60 ml of water.

Finally, for particularly difficult cases, stents may be placed in the esophagus. These must be sutured in place. The stents are made by Infiniti corporation (http://www.infinitimedical.com/p_stents.html) and the suture device is made by Pare Surgical Inc (http://www.infinitimedical.com/p_stents.html). 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. If you are treating an esophageal stricture, remember that you may need to do 1-15 dilatations. If esophagitis is diagnosed, you need to treat it aggressively in order to help prevent the stricture from recurring quickly.

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, corticosteroids 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.

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.

Some owners elect to have a permanent gastrostomy tube placed in the patient. This will not eliminate all regurgitation or aspiration, because the patient is still swallowing saliva which will remain in the esophagus until it is regurgitated. However, gastrostomy tubes will help eliminate most of the regurgitation and can markedly prolong such a patient's quality,
comfortable life.

Aspiration pneumonia is a major problem and cause of death in dogs and cats with esophageal weakness causing regurgitation. If the respiratory disease cannot be stopped by alleviating the regurgitation by dietary therapy, then you have to try to control it with antibacterial drugs. A transtracheal wash with cytology and culture will help identify optimal antibiotics. Until culture results are known, use of broad-spectrum, bactericidal drugs (i.e., amikacin plus either cephalothin or amoxicillin; enrofloxacin plus amoxicillin or clindamycin) are used. In severe cases of aspiration pneumonia, one may have to bypass the esophagus with a gastrostomy tube to try to prevent or at least minimize further aspiration. These tubes can be placed with the aid of a flexible endoscope and be used for months unless there are major problems.

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 when severe esophagitis is present because 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 esophagrams 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. Gastrinoma (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. In addition, gastric acid secretion should be minimized and preferably abolished. H-2 receptor antagonists (e.g., cimetidine, ranitidine, famotidine) suppress gastric acid secretion, but they do not eliminate it. This is because they are competitive inhibitors. That means that there is constantly some degree of competition between the H-2 receptor antagonists and the stimuli for acid secretion. 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. You can try to achieve greater efficacy with the H-2 receptor antagonists by doubling or tripling their dose, but the proton pump inhibitors are usually more effective.

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. If you use it, it should be administered as a slurry.

A combination of omeprazole and cisapride seems to be the most effective medical treatment regime. Antibiotics are used to treat secondary infections, but nobody really knows if they do anything in this regard. 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. 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.

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 bouginage 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.

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.