STOP HONKING AT ME! – MANAGING TRACHEAL COLLAPSE

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INTRODUCTION

Managing tracheal collapse (TC) can be challenging, frustrating, and sometimes frightening, for the pet, pet owner, and veterinarian. An overview of the disease will be provided with emphasis on tying therapeutic options (environmental, medical, and interventional) to symptomology.

PATHOPHYSIOLOGY

The trachea is comprised of 35 to 45 C-shaped hyaline cartilage rings.[1] The rings are connected dorsally by the tracheal membrane. The respiratory cycle results in variation in the pressure within the tracheal lumen. During inspiration, increased negative pressure in the chest results in outward radial forces on the intrathoracic trachea. Simultaneously, the cervical trachea experiences inward radial forces. During the expiration phase of respiration, the opposite occurs. This results in a tendency for the cervical tracheal to collapse during inspiration and the intrathoracic trachea to collapse during expiration. The rigidity of the normal tracheal rings prevents significant variation in tracheal size during the respiratory cycle.

There are two recognized types of tracheal TC: traditional (TTC) and malformation (MTC).[2] Traditional collapse (TTC) is a result of progressive tracheal chondromalacia and weakening of the tracheal rings. This results in a dynamic collapse of the trachea. The second type, tracheal malformation (MTC), is characterized by an abnormal shape of the cartilage rings. Rather than a "C" shape, the affected rings take on more of a "W" shape with a ventrally located invagination of the cartilage. Patients with the tracheal malformation type experience static collapse of the trachea.

Describing Tracheal Collapse

TC tends to be a progressive disease both in severity and the location of the collapse. The severity of the collapse is graded on a scale of I-IV.[1] Remember collapse can be either dynamic or static and is the qualitative nature of the collapse is noted in addition to the grade. Lastly, the location of the collapse should be documented. The most common location for tracheal collapse is at the thoracic inlet.[3] Some patients will have more significant cervical collapse, others may experience more significant intrathoracic collapse, and some patients will experience collapse of the entire length of the tracheal. The grade, location, and character of the collapse should be documented at the time of diagnosis. Example: Grade III dynamic collapse at the thoracic inlet.

Grade I	Tracheal lumen reduced by less than 25%
Grade II	Tracheal lumen reduced by 50%
Grade III	Tracheal lumen reduced by 75%
Grade IV	Tracheal lumen reduced by 90%

HISTORY, CLINICAL SIGNS, AND PHYSICAL EXAM FINDINGS

TC occurs most commonly in middle-aged small-breed dogs with Yorkshire Terriers accounting for one-third to two-thirds of cases.[1,4] Onset of signs are reported to occur in around 24% of cases within the first 6 months of life.[5] Cough, typically described as goose-honking, is a common clinical sign in patients with TC.[1,2] Other clinical signs include raspy breathing, exercise intolerance, cyanosis, collapse/syncope, and respiratory distress. Clinical signs are often exacerbated with exercise or excitement.[1] Some patients experience a long history of

waxing/waning clinical signs whereas others have minimal historical problems and present with severe respiratory distress as their initial clinical sign.

Obesity is common in patients with TC with approximately 50% of patients affected.[1] This is important to note because normalization of body condition is an important component of medical treatment. In general, other than their respiratory disease, many patients with TC are in good health. Patients can present with raspy inspiration or expiration, depending on the location of the collapse. Direct auscultation of the trachea may reveal a high-pitched or musical sound as air flow is altered as it passes through the narrowed lumen. Palpation of the cervical trachea may reveal soft or malleable tracheal rings or, with tracheal malformation, invagination of the ventral aspect of the tracheal ring. Additionally, some patients experience increased tracheal sensitivity and have an easily inducible cough. Lung auscultation is generally clear unless concurrent pulmonary disease, like pneumonia, is also present. Some patients with intrathoracic collapse will also have concurrent collapse of the carina or mainstem bronchi. In some patients, the bronchi can be heard snapping closed on expiration. Given the higher incidence of mitral valve disease in small breed dogs, a mitral murmur may also be noted concurrently; particularly in older patients.

DIAGNOSIS

For many patients, TC should be suspected based on historical clinical signs and physical examination findings. Confirmation of TC can be performed with radiographs, fluoroscopy, or bronchoscopy. These various modalities have benefits and disadvantages.

Radiography is widely available and generally considered low risk to the patient. As patients can have dynamic TC, radiography may result in a false negative result for TC or, more commonly, underestimate the degree and location of the collapse.[3] To improve the diagnostic utility of radiographs in TC, both inspiratory and expiratory images should be collected. It is also important to include the entire length of the cervical trachea in the images. Obtaining respiratory phase specific images can be difficult in patients with increased respiratory rates, who are coughing, or are in respiratory distress. If tracheal collapse is noted on plain film radiographs, I generally recommend fluoroscopy or bronchoscopy be performed to evaluate the degree and location of the collapse.

Fluoroscopic evaluate of patients with TC is beneficial as this allows the observer to evaluate the entire length of the trachea, carina, and mainstem bronchi during both phases of respiration. Observing the trachea real-time during respiration allows for more accurate assessment of the degree and location of the collapse. Some patients do not experience TC during normal respiration. In these cases, a cough can usually be induced by gentle manipulation and the trachea can then be evaluated for collapse during coughing.

Endoscopic evaluation will allow for examination of the length of the trachea, carina, and mainstem bronchi for collapse. The secondary and tertiary bronchi can also be examined for collapse, which cannot be evaluated with fluoroscopic examination. Another advantage of endoscopic examination is that the airways can be sampled for culture and cytology to evaluate for concurrent infectious components. Anesthesia is required to allow for endoscopic examination. Patients with significant TC may experience a difficult recovery from anesthesia and clinical signs may be temporarily increased following anesthesia and intubation.

EMERGENCY MEDICAL TREATMENT

Some patients will present in acute respiratory distress and/or cyanosis. The mainstays of acute management are minimal handling, oxygen therapy, sedation, antitussive therapy, and possibly administration of short-acting steroids. Depending on the stability of the patient, acepromazine (0.01 – 0.05 mg/kg) and butorphanol (0.05 –

0.1 mg/kg) can either be administered IM or IV. If the patient is stable enough for IV catheter placement at the time of presentation this can be performed, and medication administered IV. For patients with severe distress, medication should be administered IM. If available, a patient can then be placed in an oxygen cage and monitored closely. Repeat doses of sedative and/or antitussive can be given to reduce clinical signs. Diagnostics should be delayed until the patient's level of distress has improved and respiratory function is improved. If there is concern for laryngeal or tracheal edema, dexamethasone SP can be administered either IV or IM at a dose of 0.1 – 0.2 mg/kg. Patients that fail to respond may need to be intubated to receive adequate ventilation and oxygenation.[6]

Bronchodilators are generally unhelpful in patients with TC. TC is a disease affecting large airways which are comprised of larger amounts of cartilage and less smooth muscle. Bronchodilators are more effective in small bronchioles which have less cartilaginous support and greater amounts of smooth muscle.

CHRONIC MEDICAL THERAPY

For patients that present with less severe clinical signs or following management of respiratory distress, chronic medical therapy can be initiated. Medical therapy has been reported to be effective in 65% to 78% of patients with TC for greater than 12 months.[6] The goal of medical therapy is to minimize clinical signs and improve the quality of life as there is no definitive treatment for TC. Chronic therapy is usually comprised of environmental & lifestyle modifications, antitussive agents, anti-inflammatories, and judicious use of anxiolytics or sedatives and antibiotic therapy.

Environmental & Lifestyle Considerations

As noted earlier, obesity is a common finding in patients with TC. An increase in adiposity decreases lung expansion and increases the work of breathing. This phenomenon is categorized as Pickwickian Syndrome, or obesity hypoventilation syndrome. Patients with tracheal collapse are generally less tolerant of exercise and therefore weight loss must be achieved through reduced caloric intake. This author has seen significant reduction in clinical signs with appropriate weight loss.

Patients with TC have increased tracheal sensitivity. Physical simulation of the trachea or inhaled irritants are more likely to trigger cough. The use of collars or neck leads should be avoided in TC patients. A harness that avoids any compression at the neck is recommended. The home environment should be carefully discussed with clients and all possible inhaled irritants removed. This includes smoke (cigarette, pipe, etc.) as well as home fragrances like incense, diffusers, essential oils, carpet fresheners, etc. In severely affected patients, I also recommend that owners temporarily discontinue perfumes/colognes and heavily fragranced lotions. Some of these items can be reintroduced one at a time to see if they trigger a decline in patient clinical signs.

Beyond tracheal irritants, other environmental modifications may be necessary. Heat, which leads to panting, can worsen clinical signs. Owners should be instructed to avoid exposing pets to higher ambient temperatures. For some patients, excitement will also acutely worsen clinical signs. Owners should monitor for these environmental triggers and adjust as necessary. It may be impossible to remove all environmental triggers, but any reduction is of benefit for many patients.

Medications

Antitussives: Antitussive therapy is one of the more critical treatments in managing patients with TC. Opioids are generally effective at controlling cough. They work peripherally by reducing the perceived irritation of the cough

stimulus as well as centrally in the cough center.[7] There are several opioid options including hydrocodone, butorphanol, codeine, and diphenoxylate/atropine (Lomotil®). Additionally, there are a few non-schedule antitussive therapies, dextromethorphan and maropitant. Of those listed, I have had good success with hydrocodone, diphenoxylate/atropine, and maropitant. Maropitant (Cerenia®) has been demonstrated to significant reduce cough in patients with TC.[8] The neuropeptide Substance P is found in sensory peripheral nerves and the central nervous system. Substance P and its receptor, neurokinin 1, play a role in the sensitization to cough and airway inflammation. As a NK-1 receptor antagonist, maropitant works to block the effect of Substance P and subsequent minimize cough.

- Hydrocodone: 0.2 – 0.5 mg/kg PO every 6 to 12 hours.[9] Doses of up to 1 mg/kg every 6 hours have been reported. I typically start at 0.25 mg/kg every 6 hours. If effective, I gradually increase the duration, but generally try to administer at least twice daily, even in well controlled patients. Schedule II drug.
- Diphenoxylate/atropine (Lomotil®): 0.2 – 0.4 mg/kg PO every 8 to 12 hours.[9] If effective at this frequency, I will try to decrease the frequency to every 12 hours. Schedule V drug.
- Maropitant (Cerenia®): 2 mg/kg PO every 48 hours.[8]
- Butorphanol – Starting dose of 0.55 – 1.1 mg/kg every 6 to 12 hours.[9] Schedule IV drug.
- Dextromethorphan – This medication has poor oral absorption and a short half-life in dogs and would not be recommended for use in patients with TC as effective antitussive agent.

Sedatives: Sedatives are helpful with patients whose clinical signs are exacerbated by excitement. Opioids can have a sedative effect, particularly at higher doses, but may be insufficient alone. Sedatives can be administered prior to know periods of excitement or owners can be instructed to administer them if the patient is progressing to a respiratory crisis. Oral acepromazine is a long-acting phenothiazine sedative. It does not have appreciable effects on the respiratory system but does depress the cardiovascular system. I tend to utilize acepromazine for patients who have infrequent anxiety or excitement events. Trazodone, a serotonin 2A antagonist/reuptake inhibitor, is a good anxiolytic that is well-tolerated in dogs. For me, this is the preferred anxiolytic for patients with TC. Some patients require routine administration of an anxiolytic and trazodone has less risk than regular administration of acepromazine. Trazodone has a wide safety margin and supplemental doses can be given as needed for more severe events.

- Acepromazine: 0.55 – 2.2 mg/kg PO every 6 to 8 hours.[9]
- Trazodone: 2.5 – 5 mg/kg PO every 12 to 24 hours.[9] Much higher doses have been reported as well as dosing intervals of every 8 hours.

Anti-inflammatories: Inflammation is a key trigger in chronic cough or nuisance cough. In addition to the contribution to clinical signs, inflammation of the respiratory track can lead to structural changes in the respiratory tract.[8] Antitussives increase the cough threshold and subsequently decrease cough, but fail to address the trigger, which is inflammation. Glucocorticoids are primarily utilized in patients with TC and can be administered systemically and/or via inhalation. Maropitant theoretically may also have some anti-inflammatory effects, but did not result in a decrease number of inflammatory cells in one study.[8] Additional studies are needed to fully evaluate the anti-inflammatory potential of maropitant in patients with TC. As with any glucocorticoid use, the dose is tapered to find the lowest effect dose for the patient. I typically initiate therapy

with systemic glucocorticoid and slowly transition to inhaled steroid, patient permitting. NSAIDs are generally not recommended given the need for glucocorticoids in these patients.

- Prednisone: 0.5 – 1 mg/kg PO per day. Dose can be given once daily or divided and given twice daily.
- Fluticasone: 110 – 220 mcg/puff every 12 to 24 hours. Should be administered with an appropriate delivery device like the AeroDawg® chamber.

Antibiotic Therapy: For most dogs with TC, cough is not caused by underlying bacterial infection. However, some patient may have secondary bacterial infection. Ideally the decision to utilize antibiotic therapy is based on cytologic examination and culture/sensitivity testing on airway samples. However, I will occasionally utilize a short course of broad-spectrum antibiotic therapy in patients that experience an acute decline in clinical signs who were previously well controlled where another trigger cannot be identified. If this fails to resolve clinical signs, additional diagnostics and interventions should be performed. Routine use of antibiotic therapy is not recommended.

INTERVENTIONAL & SURGICAL THERAPY

TC tends to be a progressive disorder. For many, the degree of collapse and location of the collapse worsen even if clinical signs are well controlled.[6] Patients with disease that fails medical therapy, or for patients who are not stable to receive medical therapy, additional options exist including tracheal stent placement and extraluminal tracheal prosthetics.

Tracheal Stent Placement

There are currently no specific guidelines for when stent placement should be performed. It is generally accepted that patients who present in severe respiratory distress or those who fail to respond to medical therapy are good candidates. Cough is not an indicator for tracheal stent placement. If cough is the predominate clinical sign, more aggressive medical therapy should be implemented.

Currently, the tracheal stent is woven from a single strand of nitinol. Nitinol is an allow of nickel and titanium which is malleable/elastic in nature and returns to its original shape ("shape memory"). Other materials, like stainless steel, are more prone to material fatigue and fracture when placed in high motion areas. Stainless steel can only be stretched to 0.3% of its original length and return to its normal shape. Conversely, nitinol can be manipulated by over 10% and retain its original configuration.[10] The superelastic feature of this compound makes it ideal for placement within the trachea.

Tracheal stent placement methods have been refined over the years which has led to improved outcomes and fewer complications. Patients receiving stent placement are reported to have improvement in goose-honking or raspy breathing (89%) and dyspnea (84%) and have a median survival time of 1,005 days (2.75 years) in one study.[11] Possible significant complications include stent fracture and/or ingrowth of granulation tissue into the tracheal lumen. These complications can be reduced through appropriate stent sizing/placement, aggressive post-stent medical therapy, and routine re-evaluation.

A recent study evaluated TC patients who received medical therapy alone and those that received stent placement. In this study, the median survival time for patients who received medical therapy was 3.7 years

compared to 5.2 years in the group that received stent placement. For patients with severe disease, the median survival time in the medial therapy group was 12 days compared to 1,338 days for dogs receiving tracheal stent placement.[2]

Intralumenal stents offer several advantages in that they can be placed relatively quickly using a non-invasive procedure. Techniques are described using fluoroscopy, plain film radiography, and by bronchoscopy. The small tracheal diameter of many affected patients generally prevents stent placement using bronchoscopy. Fluoroscopic placement is the preferred method so that entire deployment process can be monitored and adjusted as needed to ensure correct placement.

Extraluminal Tracheal Prosthetics

There are several types of prosthetic devices available, but the general approach is the same. Prosthetics are placed around the trachea and sutured into place. These external structures provide support and prevent collapse and reduce clinical signs. Patients with cervical and/or thoracic inlet collapse are preferred candidates for this procedure. Success rates are reported to be 75% to 85%.[6] There is risk of damage to the recurrent laryngeal nerve resulting in laryngeal paralysis (10-20% of patients)[12] as well as disruption to the tracheal blood supply and tracheal necrosis (uncommon). In one earlier study, ~20% of dogs required tracheostomy and tracheal ring migration has been reported in one case report[13,14]. Long survival times (>2,500 days) have been reported for patients with cervical tracheal collapse receiving extraluminal tracheal prosthetics.[15] As such, extraluminal tracheal prosthetic placement may be an excellent option for some patients.

CONCLUSIOIN

Tracheal collapse can be a difficult disease to manage, and the therapeutic approach need to be customized for each patient. Fortunately, many patients can be managed long term with environmental, lifestyle, and medical treatments. Improvements in materials and practices have resulted in subsequent improved outcomes for patients that require tracheal stent placement.

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