Tracheal Collapse: A Common Cause of Cough

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Cough is a common reason for dogs to be presented for veterinary care, and tracheal collapse is a common cause of chronic cough in dogs. The tracheal lumen remains open during both inspiration and expiration thanks to the support of cartilaginous rings. In the dog, rings are incomplete dorsally, forming a "C" shape where the dorsal tracheal membrane connects the edges of the C via the longitudinal tracheal muscle and connective tissue. The principal bronchi which diverge from the trachea are formed by complete cartilaginous rings. Principle bronchi rapidly divide into lobar (or secondary) bronchi. Lobar bronchi divide into segmental (or tertiary) bronchi, which in turn branch dichotomously into small bronchi. Branching continues until respiratory bronchioles are formed. The primary function of the trachea is to serve as a conduit to gas flow from the larynx to the bronchi. However, and additional vital function is the removal of particulate and infectious materials. Tracheal collapse may interfere with both conduit and protective functions of the trachea.

Tracheal collapse in dogs may involve compromise of the airway lumen due to redundant tracheal membrane, weakening of the cartilaginous rings, or both. Pathogenesis is unknown, and likely multifactorial. The lumen of the entire trachea may be affected, or only sections may be compromised. The most commonly involved section is found at the thoracic inlet where the cartilage is thinnest. Although the condition is called tracheal collapse, weakened cartilaginous support can result in bronchial collapse (typically mainstem bronchi) along with tracheal collapse.

Severity of collapse varies from minimal to near complete occlusion of the tracheal lumen. The laws of physics dictate that when the lumen of the airway is compromised by 50%, resistance to airflow does not double but rather increases by 16-fold. This means that in a dog with moderate tracheal collapse of 50%, respiratory muscles must increase generated force by 16 times to maintain ventilation.

Tracheal collapse is a dynamic condition. Pressures within the airway lumen decrease during inspiration (figure?). Because the negative pressure in the pleural space during inspiration is even greater, the walls of the intrathoracic trachea tend to be held open. Simultaneously, the walls of the cervical trachea tend to collapse since surrounding pressure in the neck is relatively positive. In health, collapse is prevented by the rigid cartilage matrix. During expiration, the reverse process occurs. During expiration the walls of the intrathoracic trachea tend to collapse while the walls of the cervical trachea are forced open by changes in relative pressure.

Clinical presentation

Collapsing trachea is primarily a disease of small dogs (eg, Yorkshire terriers, Pomeranians, Chihuahuas), although large dogs and cats are sometimes affected. Clinical signs are usually manifest as the dog reaches middle age and may become more severe over time. Often, dogs with collapsing trachea have a waxing and waning course with periodic exacerbations of clinical signs. The most consistent clinical sign is cough best described as reminiscent of a "goose honk". Cough is typically precipitated by excitement or exercise and tends to occur in paroxysms. Because the cough may be accompanied by a terminal retch and/or gagging, owners may mistake the cough for vomiting. Other clinical signs include increased respiratory rate and/or effort, exercise intolerance, respiratory distress, or collapse with cyanosis.

Abnormalities on physical examination are usually limited to the respiratory system. A dry cough is usually easily elicited on tracheal palpation, but this characteristic is not pathognomonic. An occasional finding which strongly suggests tracheal collapse is an auditory "click" heard with or without a stethoscope when the large airways collapse together, most often at the end of a cough. Because up to 30% of dogs with tracheal collapse may have laryngeal paralysis, inspiratory stridor may be detected. In severely affected dogs, palpation may reveal a flattening in the shape of the normally cylindrical cervical trachea. When present, respiratory distress may occur on either inspiration, expiration, or both phases of respiration. Cardiac murmur is a common concurrent finding in dogs with tracheal collapse, perhaps because AV valvular endocardiosis is common in the same breeds that develop tracheal collapse most often.

Diagnosis

Often the clinician has a strong suspicion that tracheal collapse exists based on signalment, history, and physical exam. Major differential diagnosis for collapsing trachea include chronic bronchitis, infectious tracheobronchitis, tracheal obstruction due to intraluminal disease or extraluminal compression, elongated soft palate, endocardiosis resulting in extraluminal compression of airways or congestive heart failure, or pneumonia. Imaging studies

Thoracic and cervical radiography may delineate areas of airway narrowing and help rule out differential diagnosis and complicating conditions. Both inspiratory and expiratory lateral films are ideal when tracheal collapse is suspected since collapse is a dynamic process that varies with the phase of respiration. Although radiographic evidence of tracheal collapse is common, overlying soft tissue structures can confuse or obliterate the radiographic diagnosis. In a recent study, radiographs often underestimated the severity of collapse.

Additional imaging techniques can be useful. Flouroscopy may detect airway collapse when static films do not. Ultrousound has been used to demonstrate alteration in the shape of the tracheal lumen in the ventrodorsal projection in dogs with cervical tracheal collapse. Bronchoscopy

Bronchoscopy in perhaps the single most sensitive test for tracheal collapse. Because concurrent upper airway disease is common, laryngeal function and soft palate should be evaluated prior to bronchoscopy. Bronchoscopy allows visual detection of collapse, grading of degree of collapse, and facilitates airway sampling for cytologic and microbiologic testing. Degree of compromise in the most severely affected segments forms the basis for grading of tracheal collapse. Mainstem bronchi are also evaluated for collapse. Bronchoalveolar lavage allows sample collection for both cytologic and microbiologic assessment. Multiple studies have documented high rates of bacterial recovery from the airways of dogs with collapsing trachea. Cytologic identification of septic neutrophilic inflammation is suggestive of respiratory infection that may complicate or exacerbate tracheal collapse.

Concurrent conditions and complications

Dogs affected by collapsing trachea often have concurrent and/or complicating conditions which impact cardiopulmonary health. Tracheal collapse may be triggered by inflammation or infection of the airways, or even by endotracheal intubation. Collapse itself results in inflammation, initiating vicious cycle; cough induces inflammation, which worsens the cough, which worsens inflammation. Laryngeal paresis or paralysis, elongated soft palate, and everted laryngeal saccules are all reported in dogs with tracheal collapse. The same breeds affected by tracheal collapse are often affected by atrioventricular valve endocardiosis as well, and a third or more of dogs with tracheal collapse have concurrent hear murmur. When both conditions are present, atrial enlargement may worsen clinical signs of collapse. Interestingly, tracheal collapse is often associated with hepatopathy and increases in serum ALP as well as bile acids; improved respiratory function is associated with improved hepatic function.

Treatment options

Medical therapy

Medical therapy forms the mainstay of treatment for most dogs with collapsing trachea. Life style modifications (eg, weight control, harness vs. collar) are almost always a part of medical therapy for dogs with tracheal collapse. Antitussive medications reduce the major clinical signs of tracheal collapse (cough). Initially dose is adjusted upwards if required to control cough. Once the cough has been controlled for several weeks, the dosage may be titrated down to the lowest dose and longest dose interval which retains efficacy.

Bacterial infection does not cause tracheal collapse. However, infection may exacerbate clinical signs of collapse. Additionally, the abnormal airway in animals with tracheal collapse may have impaired mucociliary escalator function leading to an increased incidence of infection. Bacteriologic results should ideally be combined with cytologic evidence of septic inflammation to confirm infection. In the author's opinion, a short course (7 to 14 days) of an antimicrobial which reaches adequate concentrations in the airway (e.g., doxycycline) is reasonable during a disease exacerbation.

Additional therapies have been recommended to treat dogs with tracheal collapse (table). Corticosteroids are sometimes used to decrease airway inflammation; judicious use of a short course of prednisone at an antiinflammatory dose (0.5 to 1 mg/kg/day) during periods of exacerbation can be very helpful in reducing severity of clinical signs but chronic use of corticosteroids is contraindicated. Although bronchodilators do not "dilate" large airways, they are often used in an attempt to decrease airway pressure gradients. They may be particularly useful in dogs with small airway disease and intrathoracic tracheal collapse.

Occasionally, dogs with tracheal collapse present in severe respiratory distress or such distress develops with the excitement and stress of handling. Complicating factors such as congestive heart failure or laryngeal paralysis should be addressed appropriately. A combination of sedatives, corticosteroids, and an oxygen rich environment may allow stabilization of the animal. Butorphanol or acepromazine are reasonable sedative choices for most dogs.

Interventional therapy

For a small minority of dogs with tracheal collapse, medical therapy is inadequate. Interventional therapy should be considered for dogs with dyspnea, cyanosis, syncope, or frequent, severe paroxysms of cough unrelieved by medical therapy. Interventional therapy includes placement of extralumenal or intraluminal tracheal stents. Owners must understand that stent placement is designed to allow a patent airway but does not eliminate cough, the most common clinical sign associated with collapsing trachea.

Extraluminal stents are designed to hold the airway open from the outside. Surgical placement of the stents is invasive and technically demanding. These stents are best suited for dogs with cervical or thoracic inlet tracheal collapse. Complications of extraluminal stent placement can be fatal, including laryngeal paralysis and tracheal necrosis. Loosening or failure of the implant are additional complications. Because the cartilage of the entire trachea is often abnormal, stenting of a severely affected cervical trachea may simply allow collapse to develop in the thoracic trachea, much like a chain breaking at its weakest link.

A variety of non-surgically placed intraluminal tracheal stents have been used in dogs with tracheal collapse. Most of these stents are placed with fluoroscopic or bronchoscopic guidance. Stents cannot be placed in bronchi, nor can they be placed so as to touch the larynx. Reported complications of the procedure include incorrect placement, potentially fatal laryngeal spasm, tracheal perforation, transient tracheal hemorrhage, pneumomediastinum, and stent fracture. Additional complications include infection and granulation. Because the stent interferes with mucociliary clearance, airway and lung infections may occur; ideally, the epithelium grows over the

mesh stents re-establishing the escalator. Granulation tissue may grow through the stent resulting in a diminished luminal diameter or even completely occluding an airway. Additionally, the unstented segments of trachea and the bronchi are still susceptible to collapse after stent placement.

Summary

Tracheal collapse is a common cause of chronic cough in small breed dogs, and is an occasional cause of severe respiratory distress. Diagnosis of tracheal collapse is based on radiographic or bronchoscopic visualization of collapse. Because collapse is often dynamic, timing of radiographs is crucial. The diagnosis may be missed entirely if radiographs are not obtained in the appropriate phase of respiration. Each dog diagnosed with collapsing trachea should be carefully evaluated for concurrent or complicating medical conditions. Addressing such conditions appropriately often makes management of the tracheal collapse far more successful.

It is important for owners to understand clearly that tracheal collapse is not a curable condition. Instead, efforts are made to mitigate clinical signs and improve quality of life. In the majority of cases, life-style changes and medical management are adequate to control clinical signs related to tracheal collapse. However, for a proportion of affected dogs interventional therapy may be warranted. If collapse is limited to the cervical trachea, extraluminal prosthetic stents may be an option. For any segment of tracheal, placement of an intraluminal stent may bring relief from the most severe clinical signs.

Table 1: Some medications used in the therapy of collapsing trachea in the dog

medication	dosage	medication	dosage
Antitussive		**Anti-inflammatory**	
Hydrocodone	0.22 mg/kg q8 hrs	Prednisone	0.5-1.0 mg/kg/day; taper
Butorphanol	0.05-1 mg/kg PO q6-8 hrs	Dexamethasone sodium-phosphate	1 mg/kg once as emergent therapy
Lomotil	0.2-0.5 mg q12 hrs	Solu-delta cortef	50 mg/kg once as emergent therapy
Antibiotic		**Bronchodilator**	
doxycycline	5-10 mg/kg PO q24 hrs	Theophylline*	10 mg/kg* PO q12 hr
		Sedative	
		Acepromazine	0.55-2.2 mg/kg PO or 0.055-0.11 mg/kg SQ, IV, IM
		Butorphanol	0.1-0.5 mg/kg SQ, IM q2-6 hr
Other			
Stanozolol	0.15 mg/kg PO q12 hr		

Depends on preparation and manufacturer

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