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Medical treatment of tracheal collapse

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Tracheal collapse is commonly encountered as a cause of cough and airway obstruction in the dog, particularly in toy breed dogs. The etiology of tracheal collapse is unknown, but some dogs have been shown to have a reduction in chondrocytes in tracheal cartilage that allows flattening and elongation of the tracheal rings. Collapse occurs in a dorsoventral direction with prolapse of an elongated dorsal tracheal membrane into the lumen of the airway. This dynamic collapse leads to mechanical irritation of the mucosa, which enhances tracheal edema and inflammation. The cervical trachea collapses during inspiration while the intrathoracic portion of the trachea collapses on expiration. Many dogs with tracheal collapse have collapse of both the cervical and intrathoracic trachea. The principal bronchi may also be affected (bronchomalacia) in large or small breed dogs, and small airway disease (bronchitis) is variably present.

Recognition of tracheal collapse is often a clinical diagnosis based on history and signalment. However, documentation of the degree of respiratory embarrassment, identification of predisposing conditions that could exacerbate coughing, and early intervention with appropriate therapy are essential for optimal patient management.

Tracheal collapse is most commonly recognized in small or toy breed dogs, such as the Yorkshire terrier, Pomeranian, Poodle, Maltese, and Chihuahua. No sex predilection has been recognized. Affected dogs range from 1-15 years of age, although many are middle-aged when first presented. At our institution, over 2/3 of dogs diagnosed with tracheal collapse are overweight or obese. In contrast, lower airway collapse (bronchomalacia) can be seen in any size of dog, although these dogs are also often overweight.

Most dogs with tracheal or airway collapse have a chronic history of waxing and waning respiratory difficulty or cough that has grown progressively worse over time or has become refractory to treatment. Exacerbation of cough after eating and drinking or with excitement is common. The cough in dogs with tracheal collapse can be described as paroxysmal, dry, or as a “honking” cough. Owners may mistake the cough for vomiting or will report gagging or retching in association with the cough as the animal attempts to clear the airways of secretions. Worsened tachypnea, exercise intolerance, and respiratory distress tend to occur in stressful situations, such as during physical exertion, with heat stress, or in humid conditions. This may be the result of airway collapse alone, chronic lower airway disease, and/or concurrent laryngeal obstruction (edema, subglottic stenosis, or laryngeal paralysis). Cyanosis or syncope occurs in severely affected animals due to complete airway obstruction or pulmonary hypertension.

Cervical auscultation can reveal musical or wheezing sounds caused by turbulent airflow through the narrowed lumen. Dramatic stridor over the upper airway can be suggestive of concurrent laryngeal paresis or paralysis, which has been reported in 30% of dogs with tracheal collapse, however it can also result from tracheal collapse alone. A flattened cervical trachea may be palpable in severe tracheal collapse. Caution is warranted when performing tracheal examination in dogs with reports of severe coughing episodes since palpation may induce a life-threatening crisis of coughing or cough syncope.

Lung sounds may be difficult to assess in dogs with tracheal collapse due to tachypnea, obesity, or referred upper airway sounds. Crackles associated with mucus plugging and airway closure can sometimes be ausculted when concurrent chronic bronchitis or lower airway collapse is present. A loud end-expiratory snapping sound is suggestive of large airway closure or bronchial collapse. Careful cardiac auscultation should be performed because many middle-aged, small breed dogs have mitral valve insufficiency in addition to pulmonary disease and/or tracheal collapse. Hepatomegaly is a common finding in dogs with tracheal collapse. This could be associated with fatty infiltration or a non-specific hepatopathy.

Although the diagnosis of tracheal collapse can be strongly presumed based on the signalment, history, and physical examination findings, a complete diagnostic work-up should be performed to define concurrent disorders and provide appropriate therapy. Routine hematologic testing (CBC, chemistry panel, urinalysis) may detect predisposing conditions or concurrent disease in dogs with tracheal collapse. Increased liver enzymes are not uncommon in dogs with airway collapse, and elevations in serum bile acids have also been reported, although the cause for this is unclear.

Radiographs are essential to detect concurrent pulmonary or cardiac disorders. Inspiratory and expiratory lateral views are helpful in identifying luminal changes in tracheal diameter, and a dorsoventral view should also be performed to complete the examination. On full inspiration, radiographs may show collapse of the cervical trachea with dilation of the
intrathoracic trachea. Peak expiratory radiographs can sometimes show collapse of the mainstem bronchi and/or the intrathoracic trachea, with ballooning of the cervical trachea. Unfortunately, static radiographs falsely identify collapsed airways yet underestimate the degree of collapse when present, do not always identify the appropriate site of collapse, and are less helpful in documenting intrathoracic airway collapse than cervical disease. Fluoroscopy, where available, is beneficial in providing information on the degree of dynamic airway obstruction, and it also allows correlation of airway collapse with cardiac and respiratory cycles.

Concurrent bronchial disease may appear as a generalized increase in interstitial or peribronchial markings throughout the lung fields. Pulmonary infiltrates may be difficult to distinguish in obese dogs due to superimposition of fat over the thorax and within the mediastinum. Cautious interpretation of the cardiac silhouette is warranted in obese dogs with tracheal collapse. Fat around the pericardial space and reduced lung volume can lead to the impression of cardiomegaly. Right-sided heart enlargement may be present in dogs with severe tracheal collapse, pulmonary disease, or other factors that predispose to the development of pulmonary hypertension. In addition to identifying cardiopulmonary abnormalities, dorsoventral radiographs are helpful in documenting the degree of obesity. The centimetres of fat present between the thoracic cage and the skin should be noted for client communication on obesity control and for follow-up comparisons.

Bronchoscopy can document tracheal collapse when radiographs or fluoroscopy are inconclusive. Bronchoscopy also provides the following benefits: laryngeal structure (edema, saccular eversion) and intrinsic motion or function can be evaluated; the location and degree of tracheal ring flattening can be graded (see below); protrusion of the dorsal tracheal membrane into the tracheal lumen can be accurately, though subjectively, assessed; the extent of tracheal collapse can be defined for possible surgical intervention; tracheal inflammation or irritation can be visualized; intrathoracic airway collapse can be confirmed or denied; both dynamic and structural changes in the lower airways can be visualized; the contribution of small airway disease to pulmonary dysfunction can be defined.

Bronchoalveolar lavage (BAL) should be performed in all dogs in which bronchoscopy is performed to document infection and to detect inflammation by cytologic examination. Appropriate antibiotic and/or anti-inflammatory therapy can be prescribed based on BAL results. The risk of bronchoscopy in dogs with tracheal collapse can be significant, especially in obese animals with severe tracheal sensitivity. A slow recovery from anesthesia is advisable to minimize stress, and an oxygen-enriched environment should be available. One cc of 1% lidocaine sprayed into the distal trachea at the end of bronchoscopy will decrease the cough reflex in small dogs.

Inpatient management should focus on minimization of stress, and oxygen supplementation can be beneficial. Mild cough suppression and sedation with butorphanol (0.05-0.1 mg/kg SC q4-6 hours) and/or acepromazine (0.01 -0.1 mg/kg SC) may be useful. This combination of drugs seems to provide synergistic sedation, so caution should be employed when using the drugs together. If heavier sedation is required, morphine might be considered (0.25 - 0.5 mg/kg SC or IM), although this drug may induce vomiting and commonly causes panting, both of which can worsen respiratory distress. To decrease laryngeal inflammation or alleviate tracheal irritation, a single dose of dexamethasone-SP (0.1 - 0.2 mg/kg IV) can be administered.

Outpatient management should be designed to correct risk factors identified in the diagnostic work-up. If chronic bronchitis is diagnosed, corticosteroids should be employed as detailed (see proceedings). For treatment of local tracheal inflammation, only a short course of steroids would be recommended, and inhaled steroids are likely preferred to limit side effects or systemically administered steroids. Airway infection is treated with appropriate antibiotics when identified. Pending culture results, a trial on doxycycline (3-5 mg/kg PO BID) is often employed to treat potential Mycoplasma infection and for anti-inflammatory effects. Bronchodilators may be useful in animals with airway collapse or small airway disease by improving respiration and decreasing the likelihood of intrathoracic airway collapse. Suggested drugs include sustained release theophylline (10 mg/kg PO BID), terbutaline (1.25-5 mg/dog PO BID-TID) and albuterol syrup (50 μg/kg PO TID). Finally, narcotic cough suppressants are often required to control cough and should be administered often enough to control cough without inducing severe sedation. Suggested drugs include hydrocodone (0.22 mg/kg PO BID-QID) and butorphanol (0.55 - 1.1 mg/kg PO PRN). Start with frequent administration and increase the dosing interval as the dog responds in order to decrease the likelihood of inducing tolerance.

Ancillary measures include avoidance of collars and decreased exposure to heat and humidity. Encouraging weight loss for obese animals is essential since this alone can result in significant reductions in cough and improvement in overall health. Specific goals should be designed that allow the owner and animal to achieve steady, sustainable weight loss. Use of technical staff to follow adherence to the program can increase compliance. The specific diet to use depends on the individual and an exercise program can be designed depending on the tolerance of the animal. In some instances, exercise may trigger worsening of cough. Upper airway surgery, if needed, can also improve the dog’s presentation. Dogs that fail to respond to aggressive medical therapy or are presented in extreme respiratory distress may require interventional therapy to sustain life. Cervical tracheal collapse can be treated surgically with application of prosthetic rings to the outside of the trachea, and good success rates have been reported when experienced individuals perform the procedure. The primary complication is laryngeal paralysis associated with damage or bruising of the recurrent laryngeal nerve. Placement of expandable nitinol stents (produced by Infiniti Medical, www.infinitimedicale.com) within the airway has resulted in relief of clinical signs in some dogs with intractable disease due to intrathoracic tracheal collapse. The stents are placed using bronchoscopy or with fluoroscopic guidance and are in permanent position following deployment. Use of stents has not been fully evaluated in dogs with diffuse concurrent lower collapse, however they may be required as a life-saving intervention. Complications of stent placement include fracture and migration of stents.
Grades of tracheal collapse

Grade I: The cartilage ring structure of the trachea remains circular and is almost normal. Slight protrusion of the dorsal tracheal membrane into the lumen reduces the diameter by < 25%.

Grade II: Flattening of the tracheal cartilage leads to lengthening of the dorsal tracheal membrane and further reduces the luminal diameter to approximately 50%.

Grade III: The tracheal cartilage rings are severely flattened and the trachealis muscle contacts the inner surface of the tracheal cartilage. The lumen is reduced by 75%.

Grade IV: The trachealis muscle is collapsed onto the inner surface of the cartilage leading to complete obstruction of the lumen. A double lumen may be seen in some cases.