Tracheal Collapse in Dogs

Jeffrey “Jay” Adams
Mississippi State University College of Veterinary Medicine
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CPC Advisor:
Todd Archer, DVM, MS, Diplomate ACVIM
Associate Professor, Small Animal Internal Medicine
Department of Clinical Sciences

I. Introduction

Tracheal collapse refers to the narrowing of the tracheal lumen resulting from weakening of the cartilaginous rings, a redundancy of the dorsal tracheal membrane, or both.[1] Tracheal collapse most commonly occurs in middle-aged toy and miniature dogs, Toy Poodles, Yorkshire Terriers, Pomeranians, Maltese, and Chihuahuas.[1,2] Tracheal collapse in larger dogs is usually associated with trauma, deformity, or intraluminal or extraluminal masses and should not be equated with tracheal collapse in toy breed dogs.[2] The primary clinical feature in most dogs is a nonproductive couch, described as a "goose honk", and the cough often worsens during excitement, exercise, or when the collar exerts pressure on the neck.[1]

The normal trachea is composed of an open lumen maintained during all phases of quiet respiration by cartilaginous rings, which are connected by fibroelastic annular ligaments to maintain flexibility, thereby allowing movement of the neck without compromising the airway.[1,4,5] The dorsal cartilaginous rings are incomplete distally, the dorsal tracheal membrane consists of the longitudinal tracheal muscle and connective tissue.[1,4,5]

The ultimate cause of tracheal collapse is unknown. Congenital and acquired components have been proposed as causes for tracheal cartilage weakness. A credible theory of the pathogenesis of tracheal collapse is that certain dogs are predisposed to collapse because of inherent abnormalities in their cartilage but are initially asymptomatic.[1] Proposed causes include genetic factors, nutritional factors, allergens, neurologic deficiency, small airway disease, and degeneration of cartilage matrices.[2] Affected tracheal cartilages become hypocellular, and their matrices degenerate; normal hyaline cartilage is replaced by fibrocartilage and collagen fibers, and the quantities of glycoprotein and glycosaminoglycans are diminished.[2] The cartilages lose their rigidity and their ability to maintain normal tracheal conformation during the respiratory

cycle.[2] This becomes an exacerbating problem from increased respiratory efforts, airway inflammation, and/or cough.[1] In some other cases, it is an acquired tracheal lesion that follows trauma, compression caused by extraluminal masses, peritracheal inflammation, and flawed tracheotomy or transtracheal aspirate techniques.[3]

Changes in intrathoracic and airway pressures during increased respiratory efforts or cough likely assist in the progression of disease, and the chronic presence of inflammatory mediators (e.g., collagenases and proteases) within the trachea likely further weaken its structure.[1] Any narrowing of the trachea results in greatly increased resistance to air flow and local turbulence because the resistance to airflow is proportional to the reciprocal of the radius of the lumen to the fourth power.[1] This increased resistance may further contribute to a cycle of increased respiratory efforts, cough, and inflammation.[1] A continuing cycle of inflammation is plausible as a result of mucosal damage. Mucus hypersecretion and airway obstruction impair normal mucociliary clearance, and inflammatory mediators amplify the response to irritants and organisms.[1] As a result of progressive disease, changes in intrathoracic and airway pressures tend to affect specific segments of the trachea. Collapse of the cervical trachea as well as the thoracic inlet (the extrathoracic trachea) tends to occur during the inspiratory phase of respiration as pressure within the lumen drops and the walls are susceptible to atmospheric pressure.[1,2] The intrathoracic trachea, mainstem, and lobar bronchi tend to collapse during the expiratory phase of respiration.[1,2]

Clinical signs often progress with age and include abnormal respiratory noise, dyspnea, exercise intolerance, cyanosis, and syncope.[2] Signs may occur acutely but then slowly progress over months to years.[1] Tracheal collapse is classically described as occurring in middle-aged or older toy breeds (average, 6 to 8 years).[2] However, it frequently begins to show signs in dogs

between 6 months and 5 years of age.[2] Respiratory noises include wheezing, hacking, coughing, and stridorous breathing.[2] The cough maybe productive, but classically in most dogs will exhibit a nonproductive cough, as described as a "goose honk."[1,2] Stertorous sounds may be heard during periods of increased respiratory efforts, and such signs are usually the result of extrathoracic tracheal collapse.[1] Clinical signs could be elicited or exacerbated by tracheal infection, tracheal compression, exercise, excitement, eating, drinking, or hot humid weather.[2] Noxious stimuli (i.e., smoke and other respiratory irritants) may also precipitate clinical signs.[2]

On physical examination a cough can usually be elicited by palpation of the trachea.[1] Palpation may elicit paroxysmal coughing.[2] Flaccid tracheal cartilages with prominent lateral borders occasionally are evident on palpation of the cervical trachea.[2] A soft end-expiratory snapping together or clicking of the tracheal wall may be heard during auscultation if intrathoracic collapse is present.[1,2] In advanced cases or after exercise, increased inspiratory effort may be observed in dogs with extrathoracic collapse and increased expiratory effort observed in those with intrathoracic collapse, often accompanied by audible sounds.[1]

Collapsing trachea is most often suspected from the signalment and clinical signs. Imaging or direct visualization of the trachea through tracheobronchoscopy is needed to obtain a final diagnosis. Radiographs of the neck to evaluate the size of the lumen of the extrathoracic trachea are taken during inspiration, when narrowing caused by tracheal collapse is more evident because of negative airway pressure.[1,2] Conversely, the size of the lumen of the intrathoracic trachea is evaluated on thoracic radiographs taken during expiration, when increased intrathoracic pressures make collapse more apparent.[1,2] However, radiographs are only a snapshot in time, and many cases of tracheal collapse look normal on radiographic evaluation of the trachea. Fluoroscopy facilitates evaluation of dynamic movement of the trachea and

mainstem bronchi through all phases of respiration. This imaging modality is better than survey radiographs to evaluate for possible tracheal collapse, as evaluation of the changes in luminal diameter is easier to observe this way.[1] The sensitivity of fluoroscopy in detecting airway collapse is enhanced if the patient can be induced to cough during the evaluation by applying pressure on the trachea.[1]

Tracheobronchoscopy is indicated with suspected tracheal collapse to confirm and grade the severity of collapse, to evaluate the entire tracheobronchial tree, and to collect airway samples for cytology and culture.[2] The collection of airway specimens (such as by bronchoalveolar lavage) is useful for identifying exacerbating or concurrent conditions.[1] Tracheal conformation should be evaluated to determine the location and severity of the collapse.[1] There is a grading scheme for tracheal collapse that evaluates tracheal luminal obstruction and tracheal cartilage integrity. Grade 1 tracheal collapse is a reduction in lumen diameter up to 25%, with the trachealis muscle being slightly pendulous and the cartilages maintaining a somewhat normal shape.[2] Grade 2 tracheal collapse is a reduction in lumen diameter between 25 and 50%, with the trachealis muscle stretched and pendulous and the cartilage beginning to flatten.[2] Grade 3 tracheal collapse is a reduction in lumen diameter between 50 and 75%, with the trachealis more stretched and pendulous and cartilages nearly flatten.[2] Grade 4 tracheal collapse means that the lumen is essentially obliterated, collapsed between 75 and 100%, and the tracheal cartilages are completely flattened and may invert to contact the trachealis muscle.[2] Evaluation of the health of the mainstem bronchi and lower airways is indicated during tracheobroncoscopy. Collapse of mainstem bronchi and bronchomalacia were identified by bronchoscopy in 50% of dogs with tracheal collapse.[10]

Medical therapy is recommended for all animals with tracheal collapse that are not suffering from significant dyspnea, because it can result in improvement in clinical signs in the majority of dogs.[1,2] Environmental modifications are a key part of successful therapy, and considerations include using a harness instead of a collar and ensuring removal of any possible environmental allergens (such as cigarette smoke or other noxious substances in the air). Other key measures include weight loss as indicated as well as management of any concurrent underlying conditions.[1] Medical therapy is often multimodal, with considerations for cough control, bronchodilation, glucocorticoid therapy, sedation, as well as other possible medications. Antitussive medications include: Butorphanol Tartrate (Torbutrol) 0.5-1.0 mg/kg PO q6-12hr or 0.055 mg/kg SC q6-12hr, Hydrocodone Bitartrate (Hycoden) 0.5 mg/kg PO q8hr, and Diphenoxylate with Atropine (Lomotil) (2.5 mg diphenoxylate hydrochloride + 0.025 mg atropine per 5 ml or per tablet) 0.2 to 0.5 mg/kg diphenoxylate PO q8-12hr.[2] Anecdotally, maropitant has been suggested to be beneficial for the treatment of bronchitis.[11] Although placebo-controlled studies are necessary, the results of the study offer a possible explanation for the perceived improvement in clinical signs, namely, a possible antitussive effect of maropitant.[11] The dose and frequency of administration of antitussives are adjusted as needed.[1] Bronchodilators are used to decrease resistance in the respiratory airway and increase airflow to the lungs. Possible medications include: Theophylline 10 mg/kg PO q12hr, Aminophylline 10 mg/kg PO, IM, or IV q8hr, Albuterol 20-50 mcg/kg Puffs, q6-8h, and terbutaline 3-5 mcg/kg SQ q4-6h.[2,12] Pediatric metered dose inhalers used with spacers and facemasks may be used in cooperative dogs to administer aerosolized bronchodilators.[2] Anti-inflammatory doses if glucocorticoids can be given for a short period during exacerbation of signs (prednisone, 0.5 to 1 mg/kg q12h for 3 to 5 days, then tapered and discontinued over 3 to 4 weeks).[1] Sedation with

acepromazine (0.025-0.1 mg/kg [maximum 1 mg] IV, IM, or SC q8hr) and/or diazepam (0.2 mg/kg IV q12hr) and supplemental oxygen may be required in severely dyspneic patients.[2] Mucolytics and saline nebulization may benefit those with excess mucus production and infection.[2] Antibiotics are not indicated for the routine management of a collapsing trachea.[1] Dogs in which tracheal wash or bronchial alveolar wash fluid analysis has revealed evidence of infection should be treated with appropriate antibiotics based on the results of sensitivity testing.[1] Response to medical therapy in dogs with mild to moderate disease can be rewarding initially, but since this is a progressive disease, the effects may wear off over time. [2]

Placement of a tracheal stent can be considered for dogs that are no longer responsive to medical management, usually because of respiratory difficulty.[1] Placement of such stents for tracheal collapse is considered a salvage procedure.[6] It is a consideration for all dogs with moderate to severe clinical signs, often with 50% or greater reduction of the tracheal lumen, and in which there is a failure of response to aggressive medical management.[2] The goal of stent placement is to support tracheal cartilages and trachealis muscle, allowing increased amounts of air to reach the lungs.[2]

Placement of intraluminal stents for tracheal collapse has greatly reduced the morbidity and improved the success of these procedures as compared to surgical placement of extraluminal rings.[1] Advantages of intraluminal stents include short anesthetic time, immediate improvement in clinical signs related to tracheal collapse, and the ability to noninvasively place the stent within the cervical or thoracic regions.[6] The most commonly used stents are self-expanding and made of nickel-titanium alloys.[1] The flexibility of the stents is comparable to that of tracheal cartilage and allows the stent to conform closely to the shape of the entire length of the trachea, rather than exert focal amounts of pressure at specific locations along the tracheal epithelium.[8]

Intraluminal stents can address collapse at any level of the trachea; however, they are expensive, require fluoroscopic placement and can be associated with rare serious complications.[2] Possible complications include granuloma formation and stent fracture.[1]

Measurements for stent selection are performed with the patient under general anesthesia with thoracic radiographs obtained while airway pressure is held at 20 cm H_2O positive pressure.[9] Each radiograph is corrected for magnification by comparison to an esophageal probe with radiopaque markers of known increments.[6] Tracheal diameter is measured in predetermined locations along the entire length of the trachea, from the dorsal tracheal membrane to the ventral margin of the tracheal cartilages.[6] Stent diameter selection is determined by adding an additional 10–20% to the maximal diameter measured at 20 cm H2O positive pressure ventilation.[6] Stent length is chosen to specifically span the collapse seen in the trachea. This often includes stenting the maximal length of the trachea in small dogs, as measured ideally from just below the cricoid cartilage to above the tracheal bifurcation.[6] Stent selection is based upon manufacturer's shortening chart.[6]

For stent placement, patients are placed under general anesthesia, positioned in lateral recumbency, and positioned for fluoroscopy use during deployment. .[6] The stent delivery system is inserted into the trachea through the endotracheal tube and positioned appropriately distal to the collapse using fluoroscopy. [6] The stent is then slowly deployed using fluoroscopy, ensuring that the stent is maintained in the correct position along the length of the trachea. As long as the position of the stent appears correct during deployment, deployment continues until the stent is completely deployed and in correct position within the lumen of the trachea. If the stent needs adjustment during deployment, it can be resheathed, repositioned, and re-deployed. Once fully deployed, the stent delivery system is carefully removed. Final radiographs are then obtained

prior to recovering the patient from anesthesia. Patients should be continuously monitored during recovery; acute respiratory distress may occur postoperatively secondary to inflammation, edema, and/or laryngeal paresis or paralysis.[2] Medications used in medical management such as antitussives, bronchodilators, analgesics, and sedatives may be given as necessary to control coughing and excitement.[2]

Immediate improvement in clinical signs may be seen after stent placement; however, significant improvement in clinical signs may also take several weeks because of tracheitis, peritracheal swelling, and irritation.[2] Significant clinical improvement (e.g., decreased respiratory noise, less respiratory effort, increased exercise tolerance, fewer tracheobronchial infections) should be noted within 2 to 3 weeks of stent placement.[2] The quality of life is improved for most patients, but stent placement does not cure the condition.[2]

In most dogs, clinical signs can be controlled with targeted medical management for the individual patient, with diagnostic evaluations performed during episodes of persistent exacerbations of signs.[1] The prognosis depends on the location and severity of the tracheal collapse as well as any concurrent respiratory problems, such as laryngeal paralysis, main stem bronchi collapse, or bronchial disease[2] Dogs with laryngeal or bronchial disease do not improve clinically as much as those with tracheal collapse alone.[2] Approximately 80% to 90% of dogs initially improve after stent implantation, but late complications can worsen the prognosis.[6]

II. Case Presentation

Lil Man is an 11-year-old intact male Yorkshire Terrier that presented to Mississippi State University College of Veterinary Medicine Internal Medicine Department on May 1st, 2017 for tracheal collapse. Lil Man has had a history of intermittent episodes of “goose honk” coughing

for the past couple of years. Early on the morning of April 29th, 2017 the coughing was significantly worse and he was seen by his referring veterinarian. Collapsing trachea was diagnosed on chest radiographs and Acepromazine and Prednisolone/diphenhydramine were prescribed. Lil Man improved the following day but then worsened again the morning of May 1st. He presented to MSU-CVM on the morning May 1st.

Upon presentation, Lil Man appeared bright, alert, and responsive. He was immediately brought back to ICU to ensure proper oxygenation. He had pink mucous membranes and CRT of less than 2 seconds. His vital parameters (Temperature: 101.5F, Pulse: 140 bpm, and Respiration: 32 bpm) were all within normal limits. SPO2 measured at 93-94% and flow by oxygen was provided. He weighed 3.1 kg with a body condition score of 5/9 (ideal). Physical exam findings include very audible stridor "honking cough" with expiratory noise heard over the trachea, severe dental tartar, and a distended non-painful abdomen. All other physical exam findings were unremarkable. He was placed in the oxygen cage. Lab work was performed (CBC, Chemistry, and urinalysis) and no clinically significant findings were seen. A snap 4DX test was negative. Thoracic and abdominal radiographs were performed on May 1st, 2017. Thoracic radiographs revealed a focal severe narrowing/collapse of the trachea at the thoracic inlet as well as suspected small airway collapse. Abdominal radiographs revealed an enlarged liver and spleen with rounded margins as well as some mineralization of the subcutaneous tissues and stomach. Antitussive medications butorphanol (0.1 mg/kg IV for rescue), hydrocodone (0.5 mg/kg PO q12hr), albuterol (1 puff PO q12hr), and cerenia (2 mg/kg PO q8hr) were administered in hospital.

On May 2nd, 2017 bronchoscopy was performed under anesthesia. A broncho-alveolar lavage was then performed and samples from the lower airways were taken for bacterial culture

and cytology. Thickened tissue was observed on the right epiglottitis and a sample was taken to be evaluated. The trachea had grade 1-2/4 collapse (25-50% collapse) within the thoracic cavity and a grade 4/4 (100% collapse) at the thoracic inlet. The left side of his lower airways in the lungs were partially to completely collapsed as well. Cytology of the samples taken during bronchoscopy revealed no infectious agents or abnormal inflammation. Recovery from anesthesia was uneventful, although butorphanol was needed during recovery within the ICU.

On May 3rd, 2017 an "oxygen challenge" test was performed. The amount of oxygen was reduced in the oxygen cage to see if he could breathe sufficiently in a room air environment. He failed the test, as he needed the higher concentration of oxygen for proper oxygenation. His clinical signs had not improved to this point and he required maintenance within the oxygen cage as well as light sedatives. Due to the lack of response to medical management and the requirement of the oxygen cage to live, the decision was made to place a tracheal stent. On May 4th, 2017 thoracic radiographs were acquired to measure for the appropriate sized stent. Anesthesia and recovery were uneventful.

On May 5th, 2017, a tracheal stent was placed in radiology with the guidance of fluoroscopy. During the procedure, Lil Man went into cardiac arrest, but was recovered while under anesthesia. As a complication of this, regurgitation and aspiration occurred. Once stabilized, the tracheal stent was successfully deployed with the assistance of fluoroscopy, and Lil man recovered from anesthesia uneventfully. After stent placement, he was kept in the oxygen cage, the "goose honk" coughing (stridor) was no longer observed, and he was breathing well. He was placed on intravenous antibiotics for treatment of the aspiration that occurred during the procedure. The following morning of May 6th, 2017 he was placed into a normal cage, and he continued to show no signs of coughing. At this time, his lungs sounded normal on auscultation,

and his temperature was within normal limits. Medical management at this time consisted of antibiotics, nebulization and coupage, as well as antitussive drugs.

Lil Man was sent home with the following medical management: Hydrocodone, Cerenia, Theophylline, Clavamox, and Baytril. To help prevent future coughing episodes, we recommend weight loss, reduce/eliminate possible environmental trigger, limit the amount of stress and excitement, and to only walk Lil Man with a harness leash. Follow up care was managed by the referring veterinarian. A recheck appointment in July revealed that Lil Man's clinical signs had significantly improved, radiographs displayed resolution of the previously described aspiration pneumonia and no complications associated with the placement of the tracheal stent.

III. References

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