CHRONIC BRONCHITIS AND ASTHMA IN DOGS AND CATS

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INTRODUCTION

Chronic bronchitis (CB) is an inflammatory airway disease that, in association with tracheobronchial collapse, is probably the most common chronic canine airway disorder. Although the cause(s) of most cases of CB in dogs is unclear, the result is chronic airway inflammation, chronic cough and excessive mucus production. Because dogs do not expectorate, excessive mucus may be difficult to recognize. Therefore, the diagnosis of CB is usually based on chronic cough alone. Importantly, because the diagnosis of CB is based on clinical criteria (cough), the diagnosis should not be made until you rule out other causes of chronic cough such as heart failure, heartworm infestation, pneumonia, lung tumor etc. The purposes of this article are to 1) summarize the main clinical features of canine CB, 2) highlight the most important tests used to confirm the diagnosis of canine CB, and 3) emphasize practical treatment principles and specific treatment strategies.

CLINICAL FINDINGS IN DOGS WITH CHRONIC BRONCHITIS

No one really knows the typical age, breed(s) and sex of dogs with CB. Most of the available information regarding dogs with CB has been based upon relatively few, isolated case reports and one retrospective post mortem evaluation of 24 dogs with histories compatible with CB. In 1990 we reviewed the clinical presentation, pathophysiology, and efficacy of treatment for 18 dogs with confirmed CB. This information, case reports, anecdotal reports from practitioners across the country, and my experience form the basis for the following:

Signalment

Dogs diagnosed with CB are generally ≥ 8 years of age. There does not seem to be a clear sex or breed predilection although lots of small and toy breeds such as Poodles and Pomeranians have been clinically diagnosed with CB. I think that many of these dogs cough because of extrathoracic tracheal collapse which is not associated with CB.

History

By definition, dogs with CB have a chronic cough. The quality of this cough is generally deeper and “throatier” than the high pitched “honking” cough caused by extrathoracic tracheal collapse, and yet harsher than the “soft moist” cough caused by pneumonia. If you ask the question carefully you will find that the cough terminates in gagging or retching. This means the dog is bringing up and swallowing mucus.

Most dogs with CB are otherwise absolutely normal while some will be severely exercise limited by their disease. Although easily fatigued, these animals are otherwise bright, alert and in all other respects systemically well. The clinical signs of depression, lethargy, anorexia etc. are not consistent with the diagnosis of CB. If they are present you should think about another diagnosis, or an additional medical problem.

PHYSICAL EXAMINATION

Inspection is usually unremarkable. You might be able to detect a prolonged expiratory phase accompanied by an increased expiratory effort. Palpation of the chest wall is unremarkable. Although deep palpation of the trachea will often cause the dog to cough, this finding is common to most dogs with cough from any cause and is not by itself a marker for CB. Chest percussion is not really helpful to me. Thoracic auscultation may be normal or reveal diffuse crackles in all lung fields. If you hold off one nostril (of the dog) for 10-15 seconds the dog will inhale deeply when the nostril is released. Auscultation at this time may reveal previously occult crackles especially at the lung bases. Conversely, crackles may disappear for a few breaths immediately following a cough, and this would not be an appropriate time to determine whether abnormal lung sounds are present.

DIAGNOSTIC TESTS

Because the diagnosis of CB is based on a history of chronic cough, it is only necessary to perform those diagnostic tests which are required to rule of the presence of other disorders which cause cough. Still, some test results are very helpful to make you comfortable with the diagnosis of CB.

THORACIC RADIOGRAPHS
Thoracic radiographs of dogs with CB may appear normal. More commonly however, thoracic radiographs reveal the presence of “doughnuts” and/or “tram lines” which are prominent and thickened bronchial walls seen on end or in parallel, respectively.

**Bronchopulmonary Cytology**

Neutrophils are usually the predominant cell recovered from specimens taken by tracheal wash; these cells do not independently indicate current or past infection. Intracellular bacteria and/or a toxic appearance of neutrophils would of course suggest the presence of bacterial infection. Mucus is generally abundant even when a relatively small volume of fluid is recovered. Lesser numbers of lymphocytes, eosinophils, and epithelial cells are recovered in most samples.

Eosinophils may be recovered from tracheobronchial secretions of dogs with flea allergy dermatitis and an otherwise normal respiratory tract. But in general, dogs with large numbers of eosinophils in airway secretions are frequently symptomatic on a seasonal basis only (suggesting an environmental source of the offending antigen and cause for the subsequent cough), and 2) these cases also seem to respond most dramatically to anti inflammatory therapy (see Glucocorticoid Therapy below). The “snow dogs” ie; husky’s, Samoyeds etc are overrepresented as breeds that experience eosinophilic bronchitis. In these cases, the dogs may be younger, the symptoms seasonal, and the response to glucocorticoids is dramatic.

**Tracheobronchial Culture**

The airways and lungs of dogs, cats, horses and humans are frequently inhabited by a broad range of inhaled bacterial flora. In most cases, bacteria recovered from the airways of bronchitic dogs reflect innocuous colonization rather than infection.

**Bronchoscopy**

The airways of dogs with CB are universally erythematous and usually have a roughened or granular appearance. The mucosa is often thickened, irregular and edematous. Excessive and viscid mucus may be seen to span the lumen of an airway or gather together as a mucus plug, which can occlude smaller airways. Collapse of the dorsal tracheal membrane into the lumen of the airway is common in dogs with CB. In my experience, dogs with intrathoracic airway collapse respond only marginally to therapy and in general have a less fortunate prognosis than dogs whose intrathoracic airways are unaffected by passive expiration.

**Biopsy and Histopathology**

Chronic bronchitis is a clinical diagnosis and does not require tissue biopsy for confirmation.

**Therapeutic Options**

The primary treatment of CB is based entirely on controlling airway inflammation. The goal of therapy is to decrease the inflammation upon which all clinical signs result. The guiding principle of any therapy must always be “if in doubt, do no harm”.

**Corticosteroids**

Glucocorticoids (GC) have been used to treat humans with bronchial disease for over 50 years. They are clearly the single most effective means of ameliorating the symptoms of CB in people. Even though GC have no primary antitussive activity, by decreasing inflammation they may decrease stimulation of airway sensory nerves which are responsible for initiating cough in canine CB. Additionally, GC markedly decrease the volume of mucus produced by bronchitic airways. In my experience GC are the most effective drugs available to treat dogs with CB, and should be considered the mainstay of chronic therapy.

I treats new cases of CB with prednisone 1mg/kg PO, q 12h for 7 days, followed by 0.5mg/kg PO, q 12h for 7 additional days. At this point the clinical signs will have greatly improved for the vast majority of dogs with CB. The owner should continue to give the drug on an alternating day basis, while the dose is gradually decreased over the ensuing two months to the least amount of drug needed to adequately, if not completely control clinical signs. A maintenance dose of prednisone of 0.1-0.25 mg/kg PO q 12h, every other or every third day is ideal. Additionally, after and additional 2-4 months an attempt can be made to gradually stop the drug entirely. Commonly, signs may not worsen for months afterward, at which time prednisone may be reinstituted using the schedule described above.

If the side effects of steroid therapy are significant, I use the inhaled steroid Flovent. Although only 10-20% of inhaled steroids reach the small airways, they are rapidly absorbed into the lung and therefore bioavailability is assumed to be high. Additionally, direct absorption into the lung greatly diminishes the side effects of systemically administered corticosteroids. Please see the article on feline asthma for details of administration.
**Bronchodilators**

The rational use of bronchodilators to treat dogs with CB is based on 2 assumptions; 1) some degree of bronchoconstriction exists, and 2) this bronchoconstriction causes clinical signs. I rarely use bronchodilators for dogs. In my experience the exclusive use of some combination of oral and inhaled steroids predictably controls 75-90% of the initial clinical signs. This is a realistic treatment outcome.

**Antibiotics**

There is no objective evidence that bacterial infection plays a significant role in the majority of cases of canine CB. Similarly, there is no objective evidence that antibiotic therapy has any effect on the duration or intensity of signs displayed by the dog with CB. I don’t routinely perform tracheobronchial wash or prescribe antibiotics for dogs with newly diagnosed CB. This is because, in my experience, dogs with newly diagnosed CB have a more favorable response to corticosteroids than to antibiotics, and because these dogs do not have a better therapeutic response when antibiotics are given concurrently with corticosteroids. Additionally, I have never recognized the development of bacterial pneumonia in dogs with CB who were given corticosteroids and not antibiotics. Having said that, the most commonly recognized bacteria within the respiratory tree in dogs are staph, strep, e coli, bordetella, klebsiella and pseudomonas. My first choice for these organisms is enrofloxacin, 5 mg/kg PO SID for 10 days. Chloramphenicol is an excellent choice based on lipid solubility and common sensitivity patterns. I use a dose of 50 mg/kg PO TID for 10 days. IF YOU USE THIS DRUG YOU MUST FOLLOW FDA GUIDELINES REGARDING HANDLING.

**Cough Suppressants**

Chronic inflammatory disorders of the lower airway often result in the production of excessive viscid mucoid secretions. Coughing serves to clear these secretions, and thus may be viewed as a protective physiologic reflex. However, there are many cases in which the cough is dry and non productive. In these situations the cough is not protective, and serves to further irritate the airway, leading to a vicious cycle of cough-irritation-cough. In addition, some dogs with chronic cough are unable to sleep, and may awake their owners at night. Occasionally, some dogs with chronic cough may become syncopal. In each of these clinical settings cough suppression may be indicated. I use hydrocodone bitartrate, 0.22 mg/kg PO q 6-12h as needed. This is a starting dose and often needs to be increased by 50-100% increments to achieve the desired clinical effect. Although in theory any morphine-like drug can induce respiratory depression, in practice the most common side effects of over administration of hydrocodone in dogs is drowsiness and constipation. Given at night, the side effect of drowsiness may be a welcome advantage to both the dog and the owner.

**PROGNOSIS AND CONCLUSIONS**

Canine CB is a common, progressive and chronic airway disorder. Clinical signs can be controlled but the underlying disease cannot be cured. There is great value in establishing excellent client communications so that client expectations are realistic.