An In-Depth Look:

FELINE BRONCHIAL ASTHMA

Feline Bronchial Asthma: Pathophysiology and Diagnosis*

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ABSTRACT:

Feline bronchial asthma is a common respiratory condition caused by altered immunosensitivity of the respiratory tract to inhaled allergens. This hyperresponsiveness causes various structural and chemical changes in the tracheobronchial tree, including adrenergic–cholinergic imbalance and abnormal mucus production. All of these modifications may manifest as commonly recognized clinical signs, most notably coughing, wheezing, and expiratory dyspnea. No pathognomonic clinical sign(s) and/or laboratory assay(s) have been identified, and definitive diagnosis of feline bronchial asthma is based on history, physical examination findings, and exclusion of other disease entities from the differential diagnosis.

Feline bronchial asthma, which was first recognized by Hill[1] in 1906, is a reversible obstructive airway disease manifesting as extensive narrowing of the airways with excessive airway mucus, resulting in respiratory distress. This disease entity has been referred to by many different names, including feline lower airway disease, feline allergic asthma, acute allergic bronchitis, and immune-mediated airway disease.[2] Veterinarians should understand that feline bronchial asthma results from enhanced immunoreactivity to an inhaled allergen, evoking specific chemical and structural changes within the tracheobronchial tree. Emergency treatment of this condition is truly challenging because affected patients are often in critical condition. Handling for imaging and restraint for other diagnostic and therapeutic procedures may result in death in some severe cases.[3] A patient presenting with feline bronchial asthma typically has a history of paroxysmal coughing frequently associated with episodes of respiratory distress. However, cats demonstrating these clinical signs may often appear normal

*A companion article on treatment appears on page 426.

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between events. Physical examination findings are variable and depend on the severity of clinical signs. Common diagnostic procedures include a complete blood cell count, serum biochemical profile, thoracic radiography, electrocardiography, fecal examination, heartworm antibody and antigen assays, endotracheal wash, and/or bronchoscopy with bronchoalveolar lavage.[4]

NORMAL ANATOMY AND PHYSIOLOGY

Knowledge of the normal anatomy and function of the respiratory tract is necessary to understand the pathogenesis of feline bronchial asthma. The tracheobronchial tree extensively arborizes, terminating to form the pulmonary alveoli involved in gas exchange.[5] Each successive division of the respiratory tract brings changes to its structural components—alterations necessary for proper functioning of the terminal airways. Cellular components of the respiratory tract change with the transition from trachea to alveoli. Specifically, the relative amount of cartilage in the airways decreases, and the amounts of elastic fibers and smooth muscle increase as airway passages become narrower. Mucus-secreting goblet cells are plentiful in the main bronchi but sparsely present in the lower airways. However, bronchial glands are present in the narrower passages, secreting aqueous fluid into the respiratory tract. The epithelial cells are ciliated to the level of the respiratory bronchioles, whereas the alveolar walls are primarily composed of type 1 and 2 pneumocytes to facilitate gas exchange.

The respiratory tract has myriad protective mechanisms to combat a variety of irritants. Mechanical filtration occurs in the nose and nasopharynx, and the mucociliary apparatus removes foreign substances from the lower portions of the respiratory tree. If particles reach the respiratory bronchioles, alveolar ducts, and/or alveoli, alveolar macrophages phagocytize the substances, and the antigens are eventually expelled via mucociliary clearance. Secretory IgA produced in the bronchial submucosa also traps irritants in respiratory tract secretions.[6] If antigens are not effectively removed from the tracheobronchial tree, an inflammatory or allergic reaction ensues.

The respiratory tract has parasympathetic and sympathetic innervation supplied by the vagus nerve and thoracic sympathetic trunk, respectively.[7] Cholinergic stimulation triggers glandular secretion, contraction of bronchial musculature, increased mucus production, and vasodilation. The adrenergic system of the respiratory tract has β_2 receptors, and activation causes bronchial relaxation and decreased mucus production.

PATHOPHYSIOLOGY

The clinical signs associated with feline bronchial asthma result from an altered immune response, adrenergic–cholinergic imbalance, and dysfunction of the mucociliary apparatus. Patients are initially exposed to an inhaled particle, resulting in a type 1 hypersensitivity reaction.[8] Specifically, dendritic cells in the respiratory tract engulf the antigenic particle, subsequently migrating to lymph nodes to present the foreign substance to lymphocytes (helper T1 [T_H1] cells). Interactions among T_H1 cells and helper T2 (T_H2) cells evoke plasma cell differentiation to produce IgE. IgE then attaches to high-affinity receptors on mast cells and basophils, sensitizing them for future exposure to the same antigen.

There are currently no pathognomonic clinical signs or authoritative diagnostic assays for definitively diagnosing feline bronchial asthma. However, common clinical signs may include wheezing, coughing, and expiratory dyspnea.

If an animal is reexposed to the identical irritant, IgE cross-linking occurs on the surface of sensitized mast cells—a reaction that releases preformed mediators, most notably histamine. In addition, the arachidonic acid pathway is activated, and arachidonic acid is converted by cyclooxygenases to prostaglandins and thromboxane A_2 and by lipoxygenases to leukotrienes. Histamine is a vasoactive amine believed to trigger bronchoconstriction and, with other mediators, is also thought to enhance mucus secretion, increase capillary permeability, and promote granulocyte chemotaxis. Mast cells also contain eosinophil chemotactic factor; interleukin (IL)-1, 2, 3, 4, and 5; granulocyte–

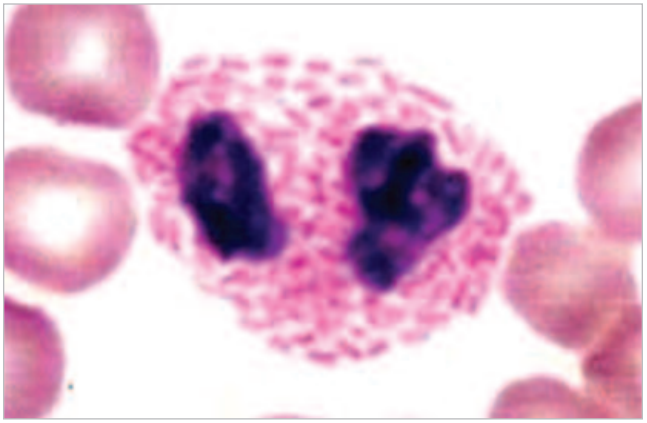


Figure 1. Presence of eosinophils in bronchoalveolar lavage samples of dyspneic cats may be supportive of bronchial asthma. There is no scientifically proven correlation between peripheral eosinophilia and the incidence of feline bronchial asthma.

macrophage colony-stimulating factor; interferon-γ; and tumor necrosis factor-α.

IL-5 promotes eosinopoiesis in bone marrow of both domestic animals and humans, resulting in increased release of mature eosinophils into circulation[9,10] (Figure 1). However, previous studies have failed to document a consistent relationship between peripheral eosinophilia and a definitive diagnosis of feline bronchial asthma.[11,12] Furthermore, IL-3 promotes differentiation of the multiple various precursors of eosinophils.[9] These granulocytes enter the area of inflammation under the influence of various chemokines and cytokines, most notably eosinophil chemotactic factor, leukotrienes, and a breakdown product of histamine called *imidazolacetic acid*.[9] In addition, a human immunology study documented that eosinophil survival is prolonged by IL-5 and granulocyte–macrophage colony-stimulating factor released from degranulated mast cells.[13] Activated eosinophils also release inflammatory mediators, particularly eosinophil major basic protein, perpetuating airway injury.[9] In addition, human research identified inflammatory changes in asymptomatic patients, indicating persistent chronic changes in the absence of clinical signs.[13]

The pathogenesis of feline bronchial asthma may be explained by an imbalance between the β_2-adrenergic and cholinergic systems. The β_2-adrenergic receptor is adenyl cyclase on bronchial smooth muscle cell membranes. Adenyl cyclase catalyzes cAMP production, which causes bronchial smooth muscle relaxation. Therefore, β_2-adrenergic stimulation increases cAMP levels, causing bronchodilation. Cholinergic action opposes β_2-adrenergic action through the generation of cGMP, which causes smooth muscle contraction.[14,15] The activities of both T_H2 cells and eosinophils help evoke the imbalance between the adrenergic and cholinergic systems of the respiratory tract, resulting in profound airway hyperresponsiveness characteristic of feline bronchial asthma.

Patients with feline bronchial asthma have abnormal mucus production and flow. Several reports have documented profound hyperplasia and/or hypertrophy of mucus-secreting cells.[2,10,16] Furthermore, a recent canine study documented that mucus is more abundant and transport up the tracheobronchial tree is greatly reduced because of epithelial denudation.[17] Thus mucus plugs accumulate, leading to considerable narrowing of the airways.

Diagnostic investigation of patients suspected of having feline bronchial asthma routinely includes a complete blood cell count, serum biochemical profile, thoracic radiography, fecal analysis, electrocardiography, and heartworm antibody and antigen assays.

Dramatic alterations of pulmonary function secondary to airway obstruction are characteristic features of feline bronchial asthma. Simply stated, ventilation is inadequate because of increased airflow resistance caused by bronchospasm, mucus plugs, and turbulent airflow. Expiratory dysfunction is a principal clinical sign of an asthmatic cat. In animals, the airway lumen is larger during inspiration than during expiration; thus partial obstruction during inspiration may become complete obstruction during expiration. Therefore, a cat with

Major Diagnostic Differentials for Feline Tachypnea/Dyspnea

- Cardiomyopathy
- Feline bronchial asthma
- *Aleurostrongylus* spp infection
- *Paragonimus* spp infection
- Toxoplasmosis
- Pyothorax
- Chylothorax
- Foreign body obstruction
- Hemothorax
- Acute or chronic bronchitis
- Viral pneumonia
- Upper respiratory infection
- FIP
- Systemic fungal infection
- Diaphragmatic hernia
- Mediastinal lymphoma
- Pulmonary neoplasia

asthma has air trapped in the alveoli, necessitating increased expiratory effort to overcome the obstruction caused by bronchospasm and excessive mucus.

DIAGNOSIS

A classic scenario frequently encountered by veterinarians is presentation of a dyspneic cat exhibiting opened-mouth breathing. The clinician must rapidly and efficiently assess the patient and institute lifesaving therapy based on often limited historical, physical examination, and diagnostic data.

The most common clinical signs associated with feline bronchial asthma include dyspnea, tachypnea, orthopnea, paroxysmal coughing, and increased expiratory effort (see box on this page). Furthermore, acute asthmatic patients are frequently severely stressed, may breathe with an opened mouth, and may bilaterally abduct their elbows when in sternal recumbency. Unfortunately, a combination of many of the clinical signs may also be seen with several other potential processes; therefore, clinicians must try to determine whether a patient's clinical signs are referable to primary cardiac or primary pulmonary disease.

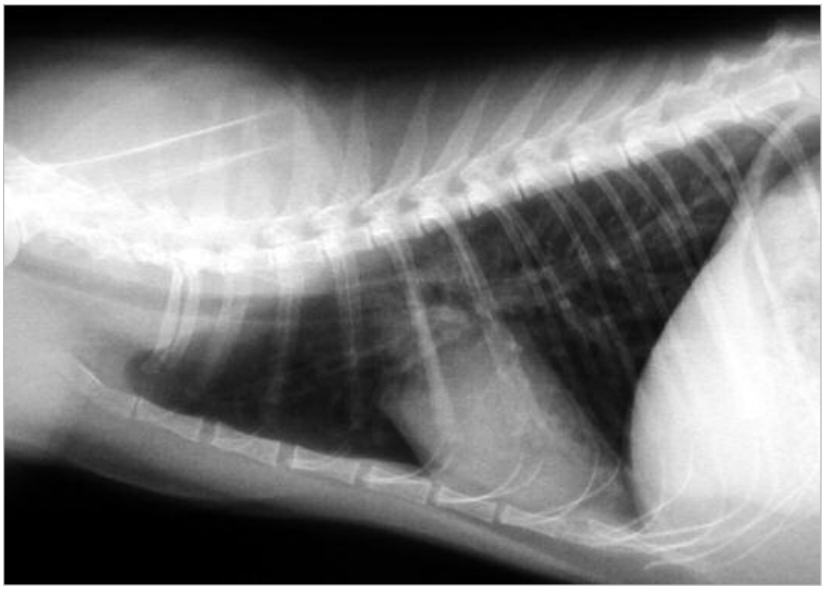
Some significant and potentially useful physical examination findings may help preliminarily distinguish between primary cardiac disease and primary respiratory dysfunction. Patients with cardiogenic shock are frequently hypothermic secondary to reduced cardiac output and subsequently reduced tissue perfusion.[18] Conversely, feline asthmatic patients typically have normal body temperatures. Thoracic auscultation of an asthmatic patient frequently yields expiratory wheezes.[2,3,8,11,12] Coughing in asthmatic cats is a frequent complaint of owners, whereas cats with cardiomyopathy rarely cough.[2,3,8,11,12] In our experience, the presence of a heart murmur should not be used as the sole factor to differentiate between primary cardiac and primary pulmonary disease.

To complicate matters further, there is no single diagnostic test for obtaining a definitive diagnosis of feline bronchial asthma. Indeed, ancillary diagnostic tests primarily serve to eliminate other potential rule outs from the differential diagnosis. Nevertheless, astute clinicians should conduct a thorough diagnostic evaluation after initial stabilization of acute asthmatic patients.

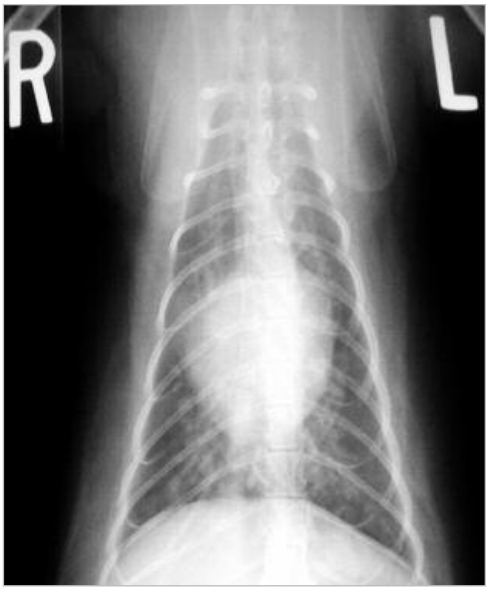
When presented with a patient in acute respiratory distress, clinicians should follow a standard and consistent protocol to assess and hopefully stabilize the patient's respiratory and cardiovascular systems (i.e., airway, breathing, circulation). Thoracic auscultation may reveal bilaterally increased bronchovesicular sounds as well as increased expiratory effort. Cats in acute respiratory distress are critically ill and may be at risk of respiratory arrest, even with minimal handling. Therefore, we recommend and provide flow-by or mask oxygen supplementation while conducting a triage examination. In case intubation and mechanical ventilation are necessary, appropriate supplies, including a laryngoscope, endotracheal tube(s), and an ambu-bag, should be within reach. One attempt to place a peripheral intravenous catheter should be made to facilitate administration of emergency intravenous medications. Appropriate bronchodilators and corticosteroids may initially be administered via inhalation and/or the intravenous route if intravenous catheterization was successful. The patient should then be placed in a temperature-controlled oxygen-enriched environment (i.e., oxygen cage) for observation. However, if inhaled medications and/or a patent intravenous catheter are not available, venipuncture should not

Endotracheal wash and/or bronchoscopy with bronchoalveolar lavage are frequently invaluable as appropriate advanced diagnostic tests to help eliminate other diagnostic differentials.

Figure 2. Left lateral and ventrodorsal thoracic radiographs of a 4-year-old, neutered, domestic shorthaired cat revealing a severely diffuse bronchial pattern. Note the extensive peribronchial infiltration appearing as "doughnuts" and "tramlines."



Left lateral view.



Ventrodorsal view.

be continued. Instead, medications should be given via appropriate alternate routes (i.e., intramuscularly and/or subcutaneously), and then the patient should be placed in an oxygen cage. Clinicians should then obtain a complete medical history from the owner. Information provided by clients may be quite varied, ranging from a history of acute onset of respiratory distress to chronic coughing with no evidence of further dyspnea. Clinicians should be sure to ask clients about the duration of clinical signs, characteristics of the surrounding environment (i.e., indoor versus outdoor cat), presence of other animals in the household, and travel history. In addition, owners should be questioned about the cat's exposure to second-hand tobacco smoke because human studies and veterinary anecdotal evidence suggest an increased prevalence of bronchial asthma in chronically exposed humans and cats.[19,20]

Once the patient is stable, a complete physical examination should be performed. If possible, orthogonal (i.e., two views at right angles to each other) thoracic radiography is also indicated. Asthmatic cats typically have a diffuse bronchial or bronchointerstitial pattern (Figure 2). In addition, the diaphragm may appear flattened, indicating alveolar air-trapping characteristic of feline bronchial asthma. Pulse oximetry may be measured to assess hemoglobin saturation (SaO_2). SaO_2 should be maintained at a minimum of 92% because lower values correlate to unacceptably low partial arterial oxygen pressures (PaO_2). According to the oxyhemoglobin concentration curve, SaO_2 values below 92% or PaO_2 values below 75 to 80 mm Hg indicate moderate to severe hypoxemia requiring aggressive intervention.[21]

Blood should be collected for a complete blood cell count, a serum biochemical profile, and heartworm antibody and antigen assays to help eliminate potential diseases from the differential diagnosis. Serologic testing for *Toxoplasma gondii* should also be considered. Feces should be collected for Baerman analysis to help rule out *Aleurostrongylus* and/or *Paragonimus* spp infection. Electrocardiography is indicated to determine cardiac conductance abnormalities and possible atrial and/or ventricular chamber enlargements. Patients with significant and persistent electrocardiographic abnormalities should be further evaluated with echocardiography.

Additional diagnostic tests may include bronchoscopy with bronchoalveolar lavage and/or endotracheal wash. These procedures are not commonly performed but may provide invaluable data for clinicians attempting to eliminate other disease processes from the differential diagnosis.[22,23] Bronchoscopy typically reveals mucosal erythema and edema as well as circumferential reduction in airway luminal diameter. In addition, excessive mucus and/or mucus plugs may be identified. No specific cytologic findings from bronchoalveolar lavage and/or endotracheal wash fluid are pathognomonic for feline

bronchial asthma.[22–24] A previous study identified a mean eosinophil count of up to 18% in the bronchoalveolar lavage fluid of normal cats.[24] Furthermore, culture and sensitivity analysis as well as a Baerman analysis may be conducted on bronchoalveolar lavage fluid to further screen for the presence of infectious organisms. Thus a presumptive diagnosis is based on a complete history, physical examination findings, and supportive diagnostic test results that have helped eliminate other disease processes from the differential diagnosis.

Pulmonary function testing has been used extensively in humans and horses to identify various pulmonary diseases and assess therapeutic response.[25,26] Current techniques include spirometry, tidal breathing flow–volume loop analysis, barometric whole body plethysmography, dynamic and static compliance, and lung and upper airway resistance.[27,28] A description of each test is beyond the scope of this article; clinicians interested in learning more about pulmonary function testing are encouraged to consult appropriate physiology texts and refereed journals. The results of each assay provide physicians with meaningful data with which to augment patient assessment. However, pulmonary function testing has not gained widespread use in veterinary medicine, and its use is currently limited to veterinary educational institutions. Nevertheless, pulmonary function testing may afford veterinarians invaluable data with which to classify various respiratory diseases, including feline bronchial asthma, and thus promote enhanced patient care.

Research is being conducted in both human and veterinary medicine to document the use of biochemical markers for detecting myocardial injury and/or failure to help differentiate between cardiac and noncardiac causes of dyspnea. Specifically, cardiac troponin I and T and brain natriuretic peptide have shown promise in human, canine, and feline studies.[29–32] To our knowledge, research is ongoing at several academic institutions, including North Carolina State University and Tufts University, to develop an emergency room test that will allow accurate, affordable, and rapid differentiation between primary cardiac and primary pulmonary disease. This type of assay would be an invaluable diagnostic asset to veterinary practitioners.

CONCLUSION

Feline bronchial asthma is a respiratory condition characterized by hyperresponsiveness of the lower airway to an inhaled allergen. An imbalance in the adrenergic and cholinergic systems, abnormal mucus production, and dramatically altered pulmonary function may result from a type 1 hypersensitivity reaction. Altered pulmonary function may manifest in several ways, but clinical signs most often include coughing, wheezing, and expiratory dyspnea.

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1. Which type of hypersensitivity reaction causes altered immunosensitivity of the tracheobronchial tree in cats with bronchial asthma?

a. I
b. 2
c. 3
d. 4

2. Which statement regarding airway protective mechanisms is true?

a. Mechanical filtration occurs at the level of the carina.
b. Foreign substances are removed from the lower airways by mucociliary elevation.
c. IgE is produced by the bronchial mucosa and traps irritants in respiratory tract secretions.
d. An inflammatory response is suppressed if an allergen is not effectively removed.

3. Histamine

a. is an active hydroxyl molecule.
b. is released from mast cells following IgA cross-linking of IgA receptors.
c. triggers severe bronchodilation.
d. is thought to enhance mucus secretion.

4. Which statement regarding adrenergic and cholinergic imbalance is true?

a. The β_2-adrenergic receptor is acetylcholinesterase on the cell membranes of bronchial smooth muscle.
b. Adenyl cyclase catalyzes production of cAMP.
c. cAMP causes bronchial smooth muscle contraction.
d. Cholinergic action augments β_2-adrenergic action.

5. Which of the following antagonizes β_2-adrenergic action?

a. adenyl cyclase
b. cGMP
c. ATP
d. protein C/protein S complex

6. Which of the following is not a common clinical sign of feline bronchial asthma?

a. paroxysmal coughing
b. tachypnea
c. inspiratory dyspnea
d. coughing

7. Which of the following is not a possible radiographic finding in cats with bronchial asthma?

a. a bronchiolar pattern
b. a flat appearance of the diaphragm
c. a bronchointerstitial pattern
d. cranial mediastinal lymphadenomegaly

8. Clinicians should strive to maintain Sao_2 above

a. 92%.
b. 60%.
c. 88%.
d. 60 mm Hg.

9. Which diagnostic test for feline bronchial asthma is not recommended?

a. electrocardiogram
b. fecal analysis
c. trypsin-like immunoreactivity assay
d. bronchoscopy

10. Which immunoglobulin is predominantly secreted by bronchial submucosa as a protective mechanism of the tracheobronchial tree?

a. IgG
b. IgA
c. IgE
d. IgM