

Feline asthma



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Introduction

Asthma may be defined as an obstructive, reversible disease that affects the lower respiratory tract. It is characterized by bronchial hyper-reaction that causes a reduction in the bronchial diameter and

KEY POINTS

- ➔ Feline asthma is an inflammatory disease that affects the lower respiratory tract and is characterized by bronchial hyper-reaction to different stimuli
- ➔ The reduction in air flow is typically the result of a combination of inflammation, mucous accumulation, and the contraction of airway smooth muscle
- ➔ Typical signs include coughing, wheezing, distress, orthopnea, tachypnea, and dyspnea with an excessive expiratory effort. Clinical signs may be permanent or intermittent, mild, moderate or severe
- ➔ The treatment for asthma as a disease must be focused on the control of inflammation in order to prevent bronchoconstriction
- ➔ Inhalation therapy is considered a key solution for human asthma management and nowadays it is widely recommended to treat feline asthma
- ➔ Alternative therapies that may be beneficial to manage feline asthma are available, but more investigation is required in order to prove their efficacy

an excessive mucous secretion which results in a variety of signs, including coughing, wheezing and respiratory distress. It is a rare condition in the animal kingdom that has only been described with these characteristics in the feline and human species (1).

First recognized in 1906 (2), asthma is one of the most common lung diseases in cats with considerable morbidity and eventual mortality. This respiratory condition has been given various names: lower airway feline disease, allergic feline asthma, allergic acute bronchitis, immune-mediated lower respiratory tract disease and feline bronchial disease. In any case, it must be clear that feline asthma is caused by an exaggerated immune reaction towards an inhaled allergen that generates specific chemical and structural changes in the tracheo-bronchial tree. Clinically, feline asthma is evidenced by the relatively variable presence of coughing, wheezing, exercise intolerance and respiratory distress that can settle spontaneously or in response to medical treatment (1,3,4).

This article aims at reviewing current knowledge on the pathophysiology of asthma in cats, recognizing the most frequent clinical signs and available diagnostic methods, and addressing a practical approach for the treatment and management of these cases.

Pathophysiology and pathogenesis

As with human asthma, the pathophysiology of feline asthma is not completely understood. However, recent research with experimental antigen-induced inflammatory bronchial disease models has helped to better characterize the mechanisms of this condition (5). Asthma is a predisposition to chronic airway inflammation with reversible bronchoconstriction episodes. There are three basic pathogenic events associated with feline asthma: immune response alteration, adrenergic-cholinergic system imbalance, and increased mucous production.

Immune response alteration: T lymphocytes, mast cells and eosinophil interaction

An allergic Type I hypersensitivity reaction is probably the reason for feline asthma, as is the case with human asthma. In cats with asthma, exposure to airborne allergens that would be harmless to normal cats stimulates the production of allergenic-specific IgE antibodies. The process starts when the dendritic cells of the respiratory tract in the asthmatic patient take the antigenic particle and then migrate to the lymphatic nodes to present the novel substance to the T₁ helper lymphocytes (TH₁ cells). Interaction between TH₁ cells and T₂ helper lymphocytes (TH₂ cells) induces cellular differentiation of B lymphocytes to produce specific IgE against the antigen. These IgE antibodies will then blend with mast cells and basophils in the respiratory mucous layer making them more sensitive towards a future exposure to the same antigen (1,3,4).

If re-exposure to the allergen occurs, the IgE on the surface of the sensitized mast cells attaches to the allergen, triggering a reaction that acutely releases preformed mediators, mainly histamine and serotonin. Histamine is a vasoactive amine that, when joined to other mediators, is believed to contribute to mucous secretion, increase capillary permeability and promote granulocyte chemotaxis. It has recently been demonstrated that serotonin is a primary mediator in feline mast cells which contributes to smooth muscle contraction. This mediator is not present in human, horse and dog respiratory airways. During an acute asthmatic episode in cats, the release of serotonin from mast cells provokes a sudden contraction of smooth muscles in the bronchi (1,3). For a long time, it had been assumed that the histamine released from feline mast cells was the chemical that caused the acute bronchoconstriction. This presumption has recently changed because of the finding that nebulized histamine in the cat's airways has unpredictable effects that can vary from one individual to another. In fact, histamine can have no effect at all, or can unleash bronchoconstriction, or can even dilate a cat's airways (1).

Activated mast cells also have the ability to release other mediators: eosinophilic chemotactic factor, interleukins 1, 2, 3, 4 and 5, granulocyte

and macrophage colony stimulating factors, interferon γ , tumor necrosis factor α , prostaglandins, thromboxane A₂ and leukotrienes. Interleukin-5 promotes eosinopoiesis in the bone marrow, increasing the release of mature eosinophils into the bloodstream. Moreover, interleukin-3 also promotes the differentiation of multiple eosinophil precursors. These granulocytes enter the inflammation site in the airways via the influence of several chemokines and cytokines, mainly eosinophilic chemotactic factor, leucotrienes and a product of histamine degradation called imidazole-acetic acid. The survival of eosinophils is increased because of the action of interleukin-5 and granulocyte and macrophage colony stimulating factors released from the mast cells. The activated eosinophils also release inflammatory mediators, particularly the main basic protein from the eosinophil, which renders damage to the airways permanent (1,4,6).

Adrenergic-cholinergic system imbalance

Inappropriate contraction of bronchial smooth muscle is directly associated with inflammation, modifying the sympathetic-parasympathetic balance of the bronchial tree. The adrenergic system acts on the airways via β_2 -adrenergic receptors whose stimulation increases the production of cAMP causing bronchodilation and a reduction in mucous production. Cholinergic stimulation is opposed to β_2 -adrenergic action by means of the generation of cGMP causing respiratory smooth muscle to contract (bronchoconstriction), increasing mucous production and provoking vasodilation. TH₂ cells and eosinophil activity contribute to the imbalance of adrenergic-cholinergic systems of the respiratory tract. This imbalance is responsible for the typical severe hyper-reaction in feline asthma that makes an asthmatic cat prone to acute narrowing of airways with only low levels of antigen stimulation (4).

Increased mucous production

An increase in mucous production is a key factor in the development of asthma, which definitely contributes to the morbidity and mortality of the disease. Goblet cells and submucosal gland cells are responsible for producing mucin in the airways. Mucin is a glucoprotein that represents the main ingredient of mucous in airways and the fundamental agent for its viscoelastic and

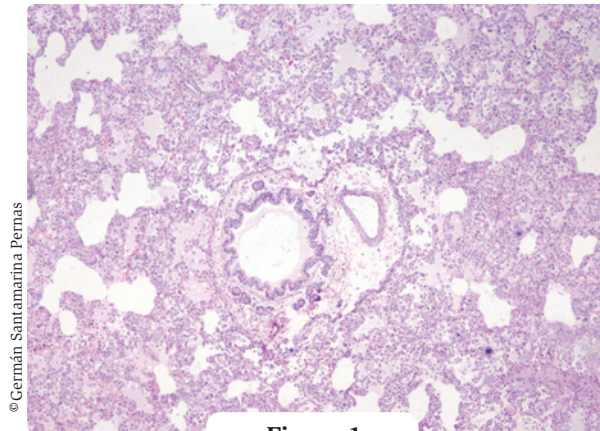


Figure 1.

Histological image of a normal bronchiole in a cat. It highlights the absence of inflammatory cell infiltrates and a normal number of submucous glands.

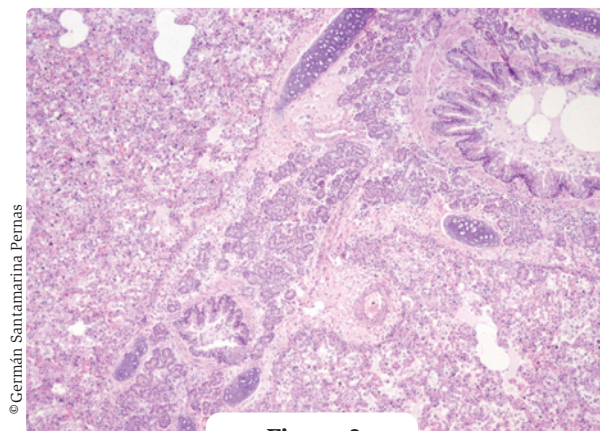


Figure 2.

Histological image of an asthmatic cat's bronchiole with significant hyperplasia of peribronchial glands.

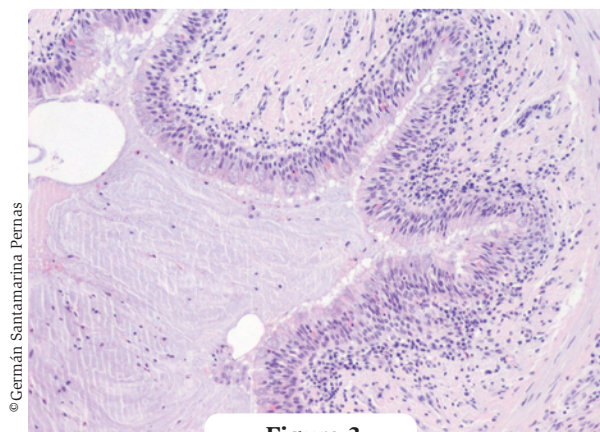


Figure 3.

Histological image of an asthmatic cat's bronchial epithelium with peribronchial inflammatory cell infiltrate, hyperplasia of Goblet cells and abundant mucous material in the bronchial lumen.

adhesive properties. Several publications have documented severe hyperplasia and/or hypertrophy of mucous secreting cells in severe asthma cases, which represents airway remodeling (**Figures 1, 2, 3**). This situation leads to an increase in stored mucins and in mucins secreted in sputum. The functional aftermath of these changes include the increase in mucous production and narrowing of airways contributing to asthma aggravation. Currently available data suggest that the action of TH₂ cytokines (especially interleukin-13) plays a very important role in increasing mucous production by stimulating hyperplasia of Goblet cells in asthma cases (1, 4).

⊗ Pathophysiological consequences

Due to the cellular infiltrate associated with chronic inflammation, airway edema and excessive mucous secretion produce narrowing of the pulmonary tracts which impairs proper ventilation. In addition asthmatic cats can suffer from an acute narrowing of the airways due to constriction of bronchial smooth muscles leading to severe respiratory distress. Clinical signs such as coughing, wheezing and lethargy are the result of airflow restriction (1,4). Coughing can also be caused by stimulation of mechano-receptors located in the inflamed and contracted smooth muscles of the airways. Complete blockage of a main bronchus may cause atelectasis in the corresponding lung lobe due to the inability of air to enter or leave the lungs (1,3,7).

Another typical characteristic of asthma in cats is expiratory dysfunction. The airway caliber is bigger during inspiration than during expiration. In this way a bronchial tube that is partially obstructed during inspiration can become totally blocked during expiration, leaving air trapped in the alveoli. This is why it becomes necessary to increase expiratory efforts to overcome the obstruction caused by bronchospasm and mucous excess. Under these circumstances, dramatic increase of intraluminal pressure may occur, and may even cause a permanent dilation of airways (bronchiectasis) and loss of elastic support structures (emphysema) (3,4,7).

⊗ Clinical presentation

The incidence of feline asthma is probably underestimated but it is thought to affect 1% of the

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population. It is the most frequent cause of coughing in cats. Although it affects cats of any age, it is more common in young and middle-aged cats. The Siamese breed may be over-represented with a prevalence that can be up to 5%. This suggests that there is a genetic predisposition similar to the human situation (1,8).

Clinical signs may be quite different according to the degree of affliction. The most frequent signs are coughing, wheezing, and respiratory harshness, which can vary from day to day. In mild cases, clinical signs are limited to short and occasional coughing bouts. These signs tend to be chronic or to progress slowly. Some cats with asthma may remain asymptomatic between occasional episodes of acute airway obstruction. In severe cases, cats can develop a daily persistent cough with frequent acute dyspneic crises. Aggravation or induction of clinical signs may occur associated with exposure to potential allergens or irritant agents such as a new bed, cigarette or chimney smoke, cleaning products or perfumed air fresheners, dust or seasonal pollen. Clinical signs tend to worsen with stress or exercise. Weight loss can be evident in cats that have chronic respiratory disease. However, those cats which restrict their activity due to respiratory disease may become overweight (1,3,6,8).

During the dyspneic crises, cats tend to adopt a typical position; lying on the floor in sternal recumbence, their head and neck extended, the mouth opened and with wheezing respiration. If bronchoconstriction lasts long enough, the cat can become cyanotic and the thorax can adopt a barrel shape due to air trapped inside. Dyspnea intensity may be assessed by the degree of the lateral retraction of nostrils and the corners of the mouth (8).

🔍 Diagnostic tests

Physical examination

Many asthmatic cats may have a normal physical exam at rest. However, in cases where the cat is severely affected due to the blockage of the lower respiratory tract, a significant expiratory distress will be typically noticed with adventitious sounds such as crackling. Wheezing can be detected when the disease is advanced or during

Table 1.

Main differential diagnoses in cats with dyspnea-tachypnea

<p>Acute pulmonary edema</p> <ul style="list-style-type: none"> • Heart failure (cardiomyopathy)
<p>Pleural effusion</p> <ul style="list-style-type: none"> • Chylothorax • Pyothorax • FIP • Heart failure (cardiomyopathy)
<p>Pulmonary parasites</p> <ul style="list-style-type: none"> • <i>Aelurostrongylus</i> spp. • <i>Paragonimus</i> spp. • <i>Capillaria</i> spp.
<p>Infectious bronchitis</p> <ul style="list-style-type: none"> • <i>Bordetella</i> • <i>Mycoplasma</i>
<p>Foreign body</p>
<p>Mediastinal lymphoma</p>
<p>Thoracic trauma</p> <ul style="list-style-type: none"> • Diaphragmatic hernia • Hemothorax

acute aggravation episodes. Blocked airways may have air trapped distally which reduces thoracic compressibility; as noted above this may cause the thorax to become barrel-shaped (1,3,6,8).

Blood tests

Approximately only 20% of cats with feline asthma have peripheral eosinophilia. This is not a very specific finding because there are other conditions that can demonstrate this (e.g. lung parasites, gastrointestinal parasites, heart worms, ectoparasites...) (3,6). Cats with asthma may show a stress leucogram, but this is also not specific. Blood chemistry seldom provides relevant information regarding lung disease, yet there are certain tests that can help eliminate some potential conditions from the differential diagnosis. It is worth considering a serum test for *Toxoplasma gondii*, and in areas where *Dirofilaria immitis* is endemic, it is recommended to perform serum tests for detection of antigen and heart worm antibody in cats with respiratory signs (3,4).

Fecal exam

Aelurostrongylus abstrusus, *Paragonimus kellicotti* or *Capillaria aerophila* infections can cause coughing and respiratory harshness in cats. In

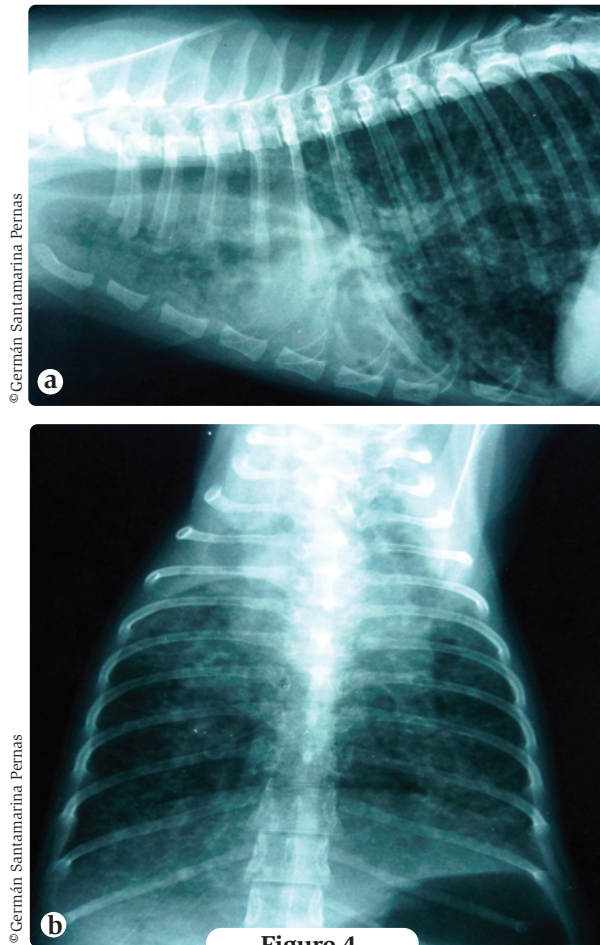


Figure 4.

Lateral (a) and ventrodorsal (b) thoracic radiographs of a cat with asthma: significant bronchial pattern, hyperinflation (caudal displacement of the diaphragm) and atelectasis of the right cranial lobe are evident.

endemic areas, these lung parasites should be excluded by proper fecal testing, including flotation with or without centrifugation techniques (to detect *Paragonimus* and *Capillaria* eggs) and Baermann sedimentation (to detect larvae of *Aelurostrongylus*) (1,3,4). For more information, see article 7 on Respiratory parasitic diseases p.44.

Thoracic X-Ray

The classic radiographic pattern of a cat with asthma tends to show swelling of the bronchial walls, generally described as “donuts” and “railway lines” (Figure 4). In addition there may be evidence of increased radiolucency of the lungs and flattening and caudal displacement of the diaphragm, recognized when observing the diaphragmatic pillars around the lumbar vertebrae area L₁-L₂. Around 15% of asthmatic cats display an increased density of the right middle lung lobe and a central

displacement towards the right. This sign is related to the presence of atelectasis in the lung lobe as a result of accumulation of mucus in the bronchi. Even though atelectasis may affect several lobes, it is particularly common in the right middle lobe, as its bronchus is oriented ventrally from the bottom of the main right bronchial tube; this means that mucus tends to build up due to gravity (3,8).

It is important to highlight that some cats with asthma may present a normal radiograph; therefore a diagnosis of asthma cannot be disregarded based only on the absence of radiographic signs.

Therapeutic trial

When a cat has respiratory symptoms that are thought to indicate asthma, a therapeutic trial can be carried out by the administration of bronchodilators. Coughing and wheezing in cats with asthma normally disappear 10 minutes after administering bronchodilators (terbutaline 0.01 mg/kg, IV, IM or SC; albuterol 100 µg inhaled (Figure 5)). Most asthmatic cats respond to treatment in 5-7 days with a high-dose corticosteroid therapy and, if no evident improvement is shown, the diagnosis for feline asthma should be reviewed (1,3).

Bronchoscopy

Bronchoscopy is not a procedure frequently undertaken when investigating a possible case of feline asthma. In cats with coughing and respiratory compromise, bronchoscopy involves certain risks and will rarely be necessary to determine a definite diagnosis of asthma in a patient. Nevertheless, on a few occasions it might be useful to eliminate other pathologies from the differential diagnosis. The bronchoscopy in feline asthma typically reveals erythema and mucous edema, reduction of airway luminal caliber and (sometimes) evidence of excessive mucous and mucous plugs (1,3,9,10).

Endotracheal/bronchoalveolar wash procedures

Cytological findings obtained during endotracheal and/or bronchoalveolar wash procedures are not pathognomonic of feline asthma. Generally in cats suffering from asthma, evidence of airway inflammation is noted with an increase in the number of eosinophils recovered from

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Figure 5.

Inhaled therapy with a measured dose inhaler (MDI) attached to a spacing chamber and connected to a facial mask. The spacing helps the drug move from the inhaler into the cat's lungs. The chamber has a releasing valve that opens when the patient inhales. The protocol is described in **Table 2**.

bronchial secretions. However, several studies have made it clear that an eosinophilic preponderance (20-25% of total cell count) recovered from tracheobronchial wash procedures may be found in many normal cats. Thus eosinophilia in bronchial samples must not be taken into account when seeking a definitive diagnosis of feline asthma (1,3,9,10).

Once aseptic samples are obtained from an endotracheal or bronchoalveolar wash, cultures and antimicrobial sensitivity tests may be performed to check for aerobic bacteria and *Mycoplasma* spp. However, it is worth noting that some organisms normally considered as pathogenic, such as *Klebsiella* or *Pseudomonas* spp., can be found in samples obtained from healthy cats. In fact, the bacteria isolated from an asthmatic cat's airways will most likely reflect colonization rather than infection. The finding of *Mycoplasma* may be an exception, as these microorganisms have been isolated only from the airways of cats with respiratory disease, not from healthy cats. The role of *Mycoplasma* in feline respiratory disease is not yet known, but it has been noted that this organism can contribute to the degradation of neutral endopeptidase, the enzyme responsible for the destruction of substance P, a protein capable of causing bronchoconstriction and edema in feline airways. *Mycoplasma* – along with certain viruses which can remain dormant in the cat's airways for long periods – is thought to be the responsible agent for the increase in substance P levels

Table 2.

Administering inhaled medications to a patient (13,15)

1. Shake the inhaler several times and insert the measured dose inhaled (MDI) into the spacing chamber connected to the mask.
2. Hold the unit on the cat's face to avoid leaks.
3. Fully press the inhaler to ensure the drug enters the spacing chamber.
4. Allow the cat to breathe 10-15 times.
5. Where two inhalations are necessary, leave at least 1-2 minutes before repeating the procedure.
6. If the animal gets frightened when pressing the inhaler, press the dispenser before placing the mask on the cat's face. Note this means the cat will receive a lower dose and may then require a second administration to guarantee it receives enough drug for the treatment.
7. If the cat is treated with fluticasone and albuterol combined, treatment should always begin with the bronchodilator.

contributing to spontaneous bronchoconstriction in asthmatic cats. It is important to bear in mind that isolation of *Mycoplasma* is difficult and requires specialized growth mediums, so handling and submission of laboratory samples must be done correctly (1,9).

Lung function test

Lung function tests, typically used in human medicine to check for pulmonary disease, are difficult to implement in veterinary practice due to the limited cooperation by animals. However, some methods have been developed to assess pulmonary function in cats, yet are rarely implemented in veterinary practice and are currently restricted to teaching and research institutions. Measurement of respiratory tidal volume-flow curves have confirmed that cats with bronchial disease have an increased ratio in the expiration time: inspiration time, a reduction in the area below the expiration curve, lower rates of expiratory flow, lower expiratory tidal volumes and increased median pulmonary resilience. These

Table 3.

Treatment protocols suggested for cats with asthmatic crises

Symptoms	Recommendations
Mild and intermittent	<ul style="list-style-type: none"> Inhaled albuterol (100 µg), as necessary
Mild to moderate	<ul style="list-style-type: none"> Oxygen therapy Inhaled albuterol (100 µg), as necessary, or terbutaline (0.325-0.625 mg/cat PO bid or tid) Long-acting theophylline (25 mg/kg PO at night) Prednisolone (1 mg/kg PO bid for 5 days, then 1 mg/kg PO sid for 5 days and 1 mg/kg PO every 48 hours for 5 days) Inhaled fluticasone (110-220 µg bid)
Severe signs Initial management	<ul style="list-style-type: none"> Oxygen therapy Inhaled albuterol (100 µg) every 30-60 minutes until respiratory effort is resolved or minimized; or terbutaline (0.01 mg/kg SC or IM every 4 hours) Dexamethasone (1 mg/kg IV or IM) Consider administering magnesium coadjuvant
Severe signs After stabilization	<ul style="list-style-type: none"> Prednisolone (1 mg/kg PO bid for 5 days, then 1 mg/kg PO sid for 5 days and 1 mg/kg PO every 48 hours for 5 days) Inhaled fluticasone (110-220 µg bid and reduce to the lowest possible dose according to the patient's clinical response) Inhaled albuterol (100 µg), as necessary

changes in resilience during the expiratory phase are compatible with an obstructive disease of the lower respiratory tract. Full-body plethysmography is another technique being researched for evaluation of lung mechanisms and has proved useful to assess a normal cat's airway reactivity. The application of this technique in cats with asthma would allow confirmation of airway hypersensitivity and permit the clinician to measure the response to bronchodilators in asthmatic cats (3).

⊗ Treatment

Not every asthmatic cat develops the disease to the same degree, therefore disease management may vary depending on the severity of the condition (11,12). On the other hand, respiratory crises are feasible in any asthmatic cat, and the control of a patient with an acute dyspneic crisis will differ from the treatment of a cat that is not undergoing a crisis (*Table 3*).

Table 4.

Common drugs used in the treatment of feline asthma

Drug	Class	Dose	Frequency	Administration	Notes
Prednisolone	Corticosteroid	2.5-5 mg/cat	BID - Q2-3d	PO	Chronic inflammation of airways
Dexamethasone	Corticosteroid	1 mg/kg	One dose - in a crisis	IV	Acute crisis management
Fluticasone MDI*	Inhaled corticosteroid	110-220 µg	BID - EOD	Inhaled	Chronic inflammation of airways, it avoids the systemic effects of steroid administration (14)
Cyclosporine	Immunosuppressant	10 mg/kg	BID (monitor through levels)	PO	Alternative to the use of steroids (18)
Cyproheptadine	Antihistamine and antiserotonine	2 mg/cat	SID - BID	PO	Interferes in smooth muscle contraction (17)
Terbutaline**	β-adrenergic agonist	0.01 mg/kg in a crisis	Repeat q 15-30 min	IV, IM, SQ	Releases bronchoconstriction
		0.625 mg/cat	BID	PO	Long-term bronchoconstriction management
Albuterol MDI*	β-adrenergic agonist	100 µg in a crisis	Repeat q 15-30 min	Inhaled	Releases bronchoconstriction (16)
		100 µg	BID	Inhaled	Long-term bronchoconstriction management
Long-acting Theophylline***	Metilxantine	25 mg/kg (theophylline anhydrous)	SID (at night)	PO	Long-term bronchoconstriction management

*Albuterol and fluticasone concentrations may vary from one country to another.

**In some countries, there is no terbutaline available for parenteral treatment.

***Different commercial presentations of long-acting theophylline display different bioavailability.

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Therapeutic strategies for the treatment of asthma are generally focused towards the suppression of inflammation and the reversal of bronchoconstriction (**Table 4**). The trend among new therapies is towards attempts to eliminate the exaggerated hypersensitivity reaction before it can trigger inflammation and bronchoconstriction of airways.

⊕ Treatment for an acute crisis

Cats that are presented in acute crisis with severe respiratory distress must be handled with care, minimizing stress and delaying, where necessary, diagnostic testing. In these situations, it is important to administer oxygen by placing the cat inside an oxygenation cage with a FiO₂ of at least 40%. This is normally an efficient and low-stress method to administer oxygen to a cat suffering from asthma (**Figure 6**). Initially, the cat will be administered bronchodilators to neutralize the acute bronchoconstriction. First choice bronchodilators for these emergency situations are the β₂-adrenergic antagonists (terbutaline and albuterol). These drugs quickly and selectively stimulate β receptors, and produce almost immediate relaxation of smooth muscle in the airways. If there is no favorable response to the initial dose within 15 - 30 minutes the bronchodilator should be repeated along with a short-term corticosteroid such as dexamethasone. Once the patient has been stabilized it is necessary

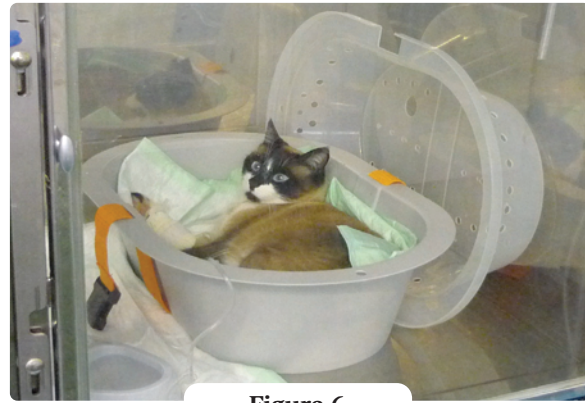


Figure 6.

Cat undergoing oxygen supplement therapy inside a cage to treat an asthmatic crisis.

to continue with the diagnostic evaluation to achieve satisfactory long-term treatment.

⊕ Conclusion

Feline asthma is a condition that any small animal clinician must be able to recognize and treat successfully, although the mechanisms of this disease are not fully understood. There is no single definitive diagnostic test available and it may be necessary to employ a variety of tests, and to eliminate other possible diseases that cause respiratory signs, to reach a tentative diagnosis. Treatment should be geared towards the individual animal and must be based on an accurate appraisal of the animal's needs. ⊕

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