

Feline asthma

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Introduction

Feline asthma is a true allergy caused by a IGE-mediated Type 1 hypersensitivity reaction. Initially inhaled allergens like pollen or dust mites elicit the early immune response. The released mediators also induce chemotactic recruitment and activation of eosinophils and other inflammatory cells leading to a slowly progressive chronic inflammation and airway remodeling. Asthma is an active chronic inflammatory disease.

There is a breed predisposition for Korat, Siamese and Birman cats. Young to middle-aged cats are most commonly affected. Clinical signs consist of cough and increased breathing effort, which vary in severity. Clinical signs often are chronic or slowly progressive. In some cats, signs go unnoticed by the owner for long periods of time. Owners may notice their cat coughing several times per day, but often they misinterpret coughing for passing a hairball. Cats with severe exacerbations "status asthmaticus" can present with open mouth breathing, dyspnea, and cyanosis. Physical examination may be normal or reveal tachypnea, expiratory distress, a barrel-shaped thorax, increased lung sounds and wheezing.

An expiratory breathing pattern is characteristic for diseases of the lower airways. The airway lumen is larger during inspiration than during expiration. Therefore, partial airway obstruction due to bronchoconstriction, inflammation or mucus plugs during inspiration may become complete obstruction during expiration, especially in peripheral airways with small diameters.

In cats presented with acute respiratory distress diagnostics should be postponed until the patient has improved. (butorphanol, oxygen, bronchodilator). Differentials for acute respiratory distress in cats and typical findings are: Asthma (the history of chronic cough, increased expiratory effort, wheezing, intermittent respiratory distress); heart disease (murmur, galopp, arrhythmia, tachy/bradycardia, mixed or inspiratory dyspnea); pleural effusion (muffled heart sounds, inaudible lung sounds, horizontal percussion dullness, mixed or inspiratory tachypnea, shallow breathing) and upper airway disease (stridor, inspiratory dyspnea).

Cough is a major finding in feline asthma and the history or presence of cough in a cat points toward the diagnosis of a lower airway disease. Cough is always caused by an airway irritation, since cough receptors are located in the larynx, the trachea and the bronchi but not in bronchioles or alveoles. Differentials for chronic cough are: asthma; chronic bronchitis (chronic, inflammatory, non-infectious, non-allergic disease with signs similar to asthma but no history of asthma attacks, no eosinophilia, little or no response to bronchodilators and an older age of affected cats); Lung worms (*Aelurostrongylus abstrusus*, *Eucoleus aerophilus* (*Capillaria aerophila*); worms with a lung passage (*Toxocara cati*)); mycoplasma; HARD (heartworm-associated respiratory disease) and bronchial neoplasia.

Diagnostics

Blood work reveals a peripheral eosinophilia in 17-46% of affected cats but may be seen in other disorders. The classic **thoracic radiographic** pattern in cats with feline asthma includes a bronchial pattern (doughnuts or tramlines), an overinflated lung (flattened diaphragm, tenting) and signs of right middle lobe atelectasis. Rarely a pneumothorax can be seen. **Fecal examination** is mandatory as part of the diagnostic work-up, to rule out/detect lung worms as origin for the eosinophilic airway infiltration. A Baermann method (*Aelurostrongylus abstrusus*) and a fecal flotation (*Toxocara cati*) should be performed. A heartworm antibody/antigen test should be performed in endemic regions. **Bronchoscopy** typically reveals mucosal erythema and edema as well as reduction and collapse in airway luminal diameter. In addition, excessive mucus and/or mucus plugs are typical findings. **Cytologic examination of airway samples**, obtained by bronchoscopy and BAL (bronchoalveolar lavage) or endotracheal wash (blind BAL) typically provides evidence of eosinophilic airway inflammation. A sterile neutrophilic airway inflammation might be seen in acute asthma exacerbations or in chronic bronchitis. The BAL fluid (BALF) should also be sent for culture and sensitivity and mycoplasma culture or PCR, although the pathogenicity of mycoplasma in the feline respiratory tract is still a matter of debate. Bacteria in the respiratory tract may be contaminants, commensals or true infection which may be primary or a consequence of airway remodelling. The decision to treat with antibiotics should be based on the clinical picture.

Barometric whole body plethysmography (BWBP) is a method for lung function testing in unrestrained cats. BWBP can be used for estimating spontaneous airflow limitation,

as well as airway hyperresponsiveness. Skin allergy testing or serum IGE testing cannot yet be recommended for clinical practice. Other diagnostics like biomarkers in BALF or exhaled breath condensate collection may be beneficial in the future.

Treatment

In cats presented with acute respiratory distress, stress should be minimized (additional administration of butorphanol 0.2mg/kg) and oxygen should be supplemented. Parenteral therapy with a bronchodilator (e.g. the beta-2 agonist terbutaline 0.01 mg/kg IV, IM or SC) should be administered promptly. An inhaled short acting bronchodilator may be used (salbutamol/albuterol 100-200 µg /1-2 actuations/Spacer) but it may pose additional stress to the patient not used to the inhalation device and the drug may not reach the bronchi in an animal with severe airflow limitation. If bronchodilators alone are not efficient a rapidly acting glucocorticoid (e.g. prednisolon 2-5 mg/kg or dexamethasone 0.25-0.5 mg/kg IV or IM) should be added. (This may influence BALF results!)

The treatment of chronic disease comprises the **avoidance** of specific and unspecific triggers (tobacco smoke, dust, open fires, perfumes, household cleaners), if possible. **Glucocorticoids** are the mainstay in the treatment of asthma, due to their anti-inflammatory, anti-allergic and anti-fibrotic effects (e.g. prednisolone 1-2 mg/kg PO q 12 h for 1-2 weeks, followed by gradual taper).

Bronchodilators can be used additionally, if glucocorticoids alone are insufficient. Bronchodilators should not be used as a monotherapy, since their anti-inflammatory potential is weak and they may mask asthma signs. (e.g. terbutalin 0,625 mg PO 2x day; theophylline slow release 25mg/PO 1 x day).

Cyclosporine has been officially used in a cat with asthma. Alternative treatments like allergen specific immunotherapy, omega-3 fatty acids, lidocaine inhalation, stem cell therapy and tyrosine-kinase inhibitors have been investigated in asthma models. Some of them may be beneficial in the future, but they cannot yet be recommended for clinical cases.

The use of inhaled medications using a metered dose inhaler, a spacing chamber and a face mask is becoming commonplace. Corticosteroid-induced side-effects can be eliminated and inhaled glucocorticoids can also safely be used in cats with heart disease or diabetes mellitus.

Inhaled glucocorticoids:

Budesonid (e.g. Budecort[®] MDI, Budair[®]) 200µg inhaled 2 puffs q12h, in stable patients reduce to 1 puff q 12h

Fluticason (e.g. Flixotide) 125µg 1 puff q12h

Combination: Fluticason 125µg/Salmeterol 25µg (e.g. AirFluSal MD[®] Serevent[®]) 1 puff q12h

Prognosis

Most cats are well controlled with oral or nebulized therapy, if the diagnosis has been settled early. Feline asthma It is a chronic, slowly progressive inflammatory disease which requires lifelong treatment. Ongoing, unnoticed airway inflammation can lead to irreversible remodeling, resulting in a decline in lung function. In chronic cases, when remodeling has become severe treatment can become challenging and asthma can lead to euthanasia.