

Self Assessment

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A four-year-old female neutered domestic shorthair cat presented with a three-month history of intermittent bouts of acute onset dyspnoea and tachypnoea, with episodes usually occurring approximately once a week. Coughing was also a prominent feature, and was harsh, non-productive and intermittent, with paroxysmal bouts occurring every few days. Appetite was good, drinking was unchanged, and no weight loss had been noted.

On clinical examination the cat was bright, alert and in good bodily condition. Mucous membrane colour, CRT, heart rate, pulse quality and temperature were normal. The cat was moderately tachypnoeic (40 breaths/min), and thoracic auscultation revealed a diffuse increase in respiratory noise, with wheezes audible bilaterally. There was no evidence of a lymphadenopathy or oculonasal discharge. Tracheal palpation did not elicit a cough reflex. Abdominal palpation was unremarkable.

The cat was admitted to the hospital for further investigations. Routine haematology and biochemistry were generally unremarkable, with the exception of a moderate eosinophilia ($3.12 \times 10^9/l$ (reference range $0-1.5 \times 10^9/l$)). The cat was sedated with ketamine and midazolam for lateral and dorsoventral thoracic radiographs (Figs. 1 and 2).



Fig. 1: Lateral thoracic radiograph.



Fig. 2: Dorsoventral thoracic radiograph.

QUESTIONS

1. Describe the changes on the thoracic radiographs.
2. Construct a list of the five most likely differential diagnoses.
3. What further investigations would you perform?

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ANSWERS

1. The cardiac silhouette is within normal limits on both the lateral and dorsoventral views. There is a moderate to severe generalised bronchial lung pattern evident. The lung fields extend cranially beyond the first rib and back to the 13th rib suggesting overinflation.

2. Differential diagnosis

- Feline asthma
- Chronic bronchitis
- *Aelurostrongylus abstrusus* infection
- Pulmonary neoplasia
- *Bordetella bronchiseptica* infection

3. Further investigations

Due to the diffuse nature of the bronchial changes on radiographs, the next diagnostic test would be bronchoscopy and bronchoalveolar lavage.

The cat was anaesthetised and bronchoscopy performed with a 4 mm flexible video-bronchoscope, which revealed mild airway hyperaemia and small amounts of mucus. An aspiration catheter passed down the biopsy channel of the bronchoscope was directed down a bronchus. Bronchoalveolar lavage was performed by instilling five millilitres of warmed sterile saline down the catheter followed by immediate re-aspiration. This was repeated with the catheter passed down a different bronchus.

Bronchoalveolar lavage produced a fluid with a high cell count. The majority of cells were inflammatory cells and eosinophils predominated (64%) (Fig. 3). There was no cytological evidence of infection and culture was negative.

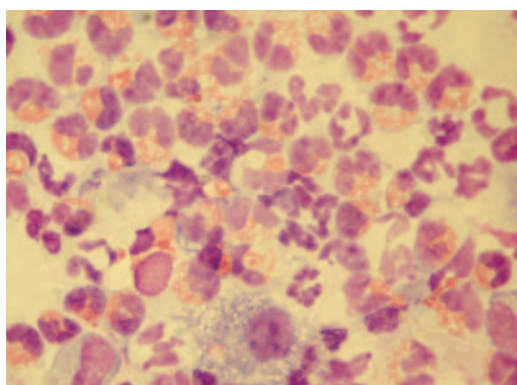


Fig. 3: Bronchoalveolar lavage cytology demonstrating many eosinophils.

Faecal analysis was also performed and did not reveal evidence of larval stage of *Aelurostrongylus abstrusus*. A diagnosis of feline asthma was made.

TREATMENT AND OUTCOME

Oral treatment with prednisolone (Prednicare Tablets; Animalcare) 1mg/kg q 12h was commenced. Inhaled steroid therapy was also initiated with fluticasone propionate (Flixotide Evohaler 220mcg; Allen & Hanburys), two puffs q 12h via a paediatric spacer chamber (Babyhaler; Allen & Hanburys), and a face-mask (Fig. 4).



Fig. 4: Metered dose inhaler connected to paediatric spacer device and face mask.

The dose of prednisolone was gradually reduced after one week and stopped after a further two weeks. The owner was also advised to avoid the use of possible precipitating or exaggerating factors such as aerosols and plug-in air fresheners, and to exclude the cat from bedrooms.

The dose of fluticasone has been reduced to 1 puff q 24h, although if the owner suspects the cat may have another asthmatic episode, the dose and frequency of inhaled medication is temporarily increased. Clinical signs have resolved and no side-effects from inhaled medication have been observed.

DISCUSSION

The term 'chronic bronchial disease' is used by some authors to include both chronic bronchitis and feline asthma, as the distinction between these two entities is difficult. Chronic bronchitis is a condition that causes a chronic cough for which other causes of cough, such as pneumonia or bronchopulmonary neoplasia, have been ruled out. Asthma is a disorder characterised by spontaneous bronchoconstriction that may resolve spontaneously or in response to therapy. The clinical signs of intermittent bouts of acute onset dyspnoea and tachypnoea, as seen in the present case, would be more suggestive of feline asthma than chronic bronchitis.

Asthma is a syndrome characterised by airway hyper-reactivity resulting in acute, reversible, bronchoconstriction (Padrid 2000). The hypersensitivity response can be aimed at a variety of inhaled allergens, and results in airway inflammation. Airway inflammation induces reversible

airflow obstruction through smooth muscle constriction, bronchial wall oedema, and hypertrophy of mucous glands, resulting in signs ranging from intermittent coughing to respiratory distress (Johnson 2000).

There is no one diagnostic test that allows for a definitive diagnosis of feline asthma, and diagnosis was one of exclusion. The history of coughing with intermittent episodes of acute onset tachypnoea and dyspnoea, in an otherwise healthy cat was very suggestive of feline asthma. Serum biochemical and haematological changes are generally non-specific, and only 30% of cases have circulating eosinophilia (Padrid 1991). Thoracic radiography was very useful in this case as it revealed bronchial pathology. The most important radiographic finding is bronchial wall thickening, however interstitial, bronchial and alveolar patterns may be seen (Johnson 2000). Cytology of tracheobronchial secretions was consistent with a diagnosis of asthma (Padrid 2001a), and revealed evidence of airway inflammation, with the presence of large numbers of eosinophils. However healthy cats have been found to exhibit up to 25% eosinophils (Padrid 1991).

Aleurostrongylus abstrusus infection should be ruled out, preferably by examination of several faecal samples, as a cause of coughing and peripheral blood and airway eosinophilia, although most infected cats are probably asymptomatic.

Human asthmatic airways show evidence of chronic ongoing inflammation whether or not the patient is symptomatic, and treatment is aimed towards underlying chronic inflammation, with the mainstay of treatment involving the use of anti-inflammatories and bronchodilators. The most effective treatment for feline asthma is high-dose, long-term oral corticosteroids (Padrid 2000). However, long-term therapy can lead to undesirable side-effects. Inhaled corticosteroids have the advantage of delivering a high drug concentration very quickly directly into the airway, so minimizing systemic side-effects (Padrid 2001b). Oral prednisolone was used in the case initially, since it can take up to 14 days for optimal drug levels of inhaled corticosteroids to be achieved. This also allowed time for the owners to become proficient in using the inhalation device. Inhaled fluticasone was chosen, as it is highly effective in cats with virtually no systemic side-effects, although inhaled beclomethasone is also a suitable choice. A spacer chamber allows the cat to inhale medication, and a paediatric type was chosen because of its very low resistance. Recently, a purpose made, low resistance, feline spacer device has become available (Aerokat Feline Aerosol Chamber).

Feline asthma is a chronic disease, and it is likely that the present case will require long-term continuous medication. Other medication such as leukotriene receptor antagonists or cyclosporin may be of benefit in the future (Johnson 2001). Unfortunately in a number of cases irreversible fibrotic changes and chronic hypoxia can ensue.

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