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The management of suspected a

DO you think you have seen a dog with hay fever or asthma? It is well-accepted that allergic responses can trigger respiratory tract signs in humans and allergic triggers are suspected to underlie feline allergic bronchitis (feline asthma). In dogs, however, the poten-

tial for allergy as a trigger for respiratory signs is less generally accepted and gains little mention in standard veterinary textbooks, which understandably concentrate on the more readily-characterised disorders such as chronic bronchitis, neoplasia and infection.

Since allergic disease is accepted as a cause of dermatological signs and gastro-intestinal signs, for example, there is no reason to suppose that allergy could not trigger airways signs in dogs, as it does in humans and other veterinary species. Published reports support this conclusion (Corcoran *et al.*, 1991, Clerx *et al.*, 2000).

Clinical signs associated with inflammation and irritation of the anatomic regions of the airway are outlined in Table I. With all signs, of course, a wide differential diagnosis must be considered. Additional features that might indicate a possible allergic trigger include a seasonality or variability in clinical signs. As with hay fever in humans, pollen may be an aeroallergen for dogs and clinical signs may be worse only at certain times of the year. Similarly, signs may be triggered by exposure to allergen in certain areas of the normal environment.

Allergic disease is typically characterised by the presence of eosinophils in inflammatory infiltrates of tissue, representing an inappropriate acute response to benign antigens as a result of immune sensitisation. In nature these responses are typically directed at parasites such as intestinal helminths, and an important tenet of clinical management of all cases where allergy is suspected is to ensure that there is no associated parasitism either through diagnostic testing, therapy or both.

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looks at canine allergic airway disease and examines four typical case studies

dander (Clercx et al., 2000).

For allergic airway disease, there are no specific diagnostic tests available to identify either allergy or the triggering allergen, other than exposure: withdrawal:exposure regimes. Unfortunately, unlike the skin, the airways are not amenable to localised exposure testing, and this could, potentially, trigger serious and life-threatening responses.

It is unusual, therefore, to be able to definitively identify possible triggers. The suspicion of allergy, however, is increased upon identification of an eosinophilic component in diagnostic samples such as nasal flushes, pharyngeal swabs and bronchoalveolar lavage, although the presence of eosinophils should not be considered an absolute prerequisite.

Therapeutic options for suspected airway diseases include allergen avoidance, irritant avoidance, anti-inflammatory therapy and palliative therapy such as bronchodilation. Allergen avoidance may be possible if a specific trigger can be recognised, and a process of trial and error in exposing the patient to various regions of its normal environment may help. Similarly, a small proportion of cases appear to respond to exclusion diets, and there is a justification for a diet trial in most cases (Corcoran et al., 1991).

In all cases of inflammatory airway disease, additional irritants should be avoided if at all possible, including tobacco smoke, house dust and other noxious and irritating substances a dog might be exposed to through typical indiscriminate sniffing. Anti-inflammatory drugs, particularly corticosteroids, are an important tool in managing airway inflammation and can be administered orally or by inhalament and both oral and inhaled medications may be used.

Case studies

In this article, I will use some case examples to illustrate where allergy appeared to play a role in the generation of a variety of clinical signs related to the airway and a range of options for therapy.

Jethro a seven-year-old male English bull terrier. Jethro presented with a complicated medical history including chronic allergic skin disease, hypoadrenocorticism and lungworm, which had been appropriately treated. The presenting complaint was of expiratory dyspnoea and coughing. On examination Jethro was bright and alert and in good to slightly obese body condition. There were marked crackles over both lung fields and Jethro demonstrated a wheezy cough during the consultation.

Haematology showed a mild neutrophilia, lymphocytosis and monocytosis, consistent with chronic inflammation.

Thoracic radiographic examination showed a generalised broncho-interstitial pattern (Fig**ure I**). An echocardiographic examination was unremarkable. No lungworm larvae were found in faeces. A bronchoscopic examination showed excess airway mucus and dynamic small airway collapse. A BAL grew no organisms and on cytology there was mild neutrophilic inflammation. Possible underlying causes for the bronchitis were considered to be allergy, infection or irritation.

Notwithstanding the lack of a specific identified trigger, anti-inflammatory and bronchodilator therapy was indicated and, to avoid complicating the management of Jethro's other medical conditions, he was considered a good potential candidate for inhalation therapy. Jethro was weaned onto inhaled budesonide (Pulmicort, Astra-Zeneca) 100μ g twice-daily and salbutamol (Ventolin, Allen and Hanburys) 100 μ g three to four times daily, according to owner instructions shown later. (Note that Pulmicort is presently unavailable and fluticasone (Flixotide, Allen and Hanburys) appears an acceptable alternative). Jethro was re-examined five weeks later, when the owners reported that there had been no difficulty in getting Jethro to accept inhaled treatment. They reported minimal

coughing and markedly improved exercise tolerance.

Examination revealed some expiratory effort and occasional crackles were audible over the ventral lung fields. A repeat thoracic radiograph showed a marked reduction in the lung pattern previously noted (**Figure 2**). The owners were advised that they could withhold salbutamol unless needed for bronchospasm relief and to maintain once-daily budesonide inhalation for continued suppression of inflammation in the airway.

• Corrie, a five-year-old female neutered border collie. Corrie presented with a history of exercise intolerance, occasional wheezing and low-grade coughing. The owner was a keen hill walker and had noticed a reduction in Corrie's normal stamina on long walks, although Corrie was well able to tolerate normal exercise. She was otherwise considered to be well with no other clinical signs.

Clinical examination was unremarkable. Haematology and biochemistry screens were unremarkable. Faecal parasitology was negative. Thoracic radiographs showed a mild generalised bronchial pattern and, on bronchoscopic examination, there was hyperaemia of the bronchial mucosa. A BAL revealed a mild, mixed neutrophilic-eosinophilic inflammation with no organisms. An allergic bronchitis was suspected and, to explore possible triggers, a turkey and rice exclusion diet (James Wellbeloved Turkey and Rice Kibble) was started. Within two weeks the owner reported a marked improvement in clinical signs and, after a further two weeks, re-introduction of a normal diet was associated with a noted deterioration

An exclusion diet was maintained and Corrie was considered well and normal for four years. She represented after four years because of a recurrence of coughing and exercise intolerance, and repeat investigations suggested a recurrence of eosinophilic-neutrophilic bronchitis. This did not respond to dietary management at this point, so therapy with inhaled budesonide and salbutamol was initiated and a good response was identified by the owner, with reduced coughing and panting and improved exercise tolerance. Ellie, a two- and a half-yearold female golden retriever. Clinical signs in this dog were difficult to define. The owner complained that there was exercise intolerance, abnormal breathing and a dark tongue colour on exercise. Ellie was also reported to have a tendency to gag and cough after drinking.

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tion. Similarly, bronchodilators (methylxanthines, beta-2 agonists) may be administered to relieve bronchospasm when there is small airway involve-

TABLE 1. Signs associated with possibleallergic airway disease

- Rhinitis Sneeze, nasal
- discharge (serous or mucoid).
- May be accompanying conjunctivitis.

Pharyngitis

- Snorting, reverse sneezing, excessive swallowing, gulping and gagging.
- Tracheobronchitis
 Coughing (often harsh and dry).
 Expiratory dyspnoea (if small airways involved).
- Exercise intolerance.Heat intolerance.
- Increased panting.
- Pneumonitis
 - Coughing (often deep and soft), inspiratory and expiratory dyspnoea, malaise, fever.

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