Common lower airway diseases in the dog and cat

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This is the second in a series of two articles describing the more common lower respiratory diseases in dogs and cats. Although they have been separated into two articles (see also Bronchial disease in the dog and cat UK Vet Vol 10 No 2), one needs to be aware that there is often overlap between the anatomical localisation of the different pathologies. Pulmonary neoplasia will not be discussed in this article.

BRONCHOPNEUMONIA
Aetiology

Infectious bronchopneumonia is in most cases a secondary bacterial pulmonary colonisation (*Staphylococcus* spp, *Streptococcus* spp, *Proteus* spp, *Klebsiella* spp, *Pseudomonas* spp, *Pasteurella* spp) after a primary viral infection (Distemper, canine adenovirus II, canine parainfluenza, in the dog, Fig. 1;

feline calicivirus and feline herpesvirus in the cat, Fig. 2). Some agents are known to act as primary pathogens (*Bordetella bronchiseptica* in the dog and cat, *Beta-haemolytic streptococci*, *Pasteurella* spp in the cat). Only recently a very virulent form of a mutated equine influenza virus has been described in the USA. *Mycoplasma* spp. are currently also emerging. Fungal infections of the lower airways are very rare in the UK.

Bronchopneumonia can be a complication of aspiration of food or gastric acid (e.g. with dysphagia, megaoesophagus, anaesthetic reflux: often a cranioventral distribution of the lesions, Fig. 3), inhalation of a foreign body (often localised to the right middle lung lobe, Fig. 4) or toxic substances (smoke inhalation), pulmonary haemorrhage

Fig. 1: *A Cavalier King Charles Spaniel with infectious bronchopneumonia and intranasal oxygen administration.*

Fig. 2: *A cat with bronchopneumonia showing a lingual ulcer secondary to calicivirus infection.*

Fig. 3: *The typical cranioventral distribution of the radiographic pulmonary lesions associated with aspiration pneumonia.*

Fig. 4: *Grass seeds are common foreign bodies in the airways.*

(coagulopathies, trauma) and many chronic airway diseases (brachycephalic upper airway syndrome, chronic bronchitis, feline asthma).

In cases of recurrent bronchopneumonia in the absence of any of the above causes an underlying immuno-incompetence (e.g. ciliary dyskinesia, neutrophilic chemotactic dysfunction, rhinitis/ bronchopneumonia syndrome in Irish Wolfhounds, FIV/FeLV in the cat) should be considered.

Clinical signs

A productive cough and pyrexia (often associated with anorexia and lethargy) are the most common clinical signs. In severe cases and depending on the extension of the lesions an expiratory dyspnoea and cyanosis can also be observed.

Diagnosis

A full history and a thorough physical examination (fever, crepitations on auscultation) are highly valuable. Thoracic radiography is essential for confirming the diagnosis but one has to be aware that there is not always a correlation between the severity of the clinical signs and the radiographic changes (Fig. 5). Bronchoscopy will show the

Fig. 5: *A detailed view of an air-bronchogram.*

presence of purulent material in the lower airways but is mainly used to obtain a selective cytology and culture sample (bronchoalveolar lavage, BAL). A tracheal wash is an alternative when there is no access to BAL. A transtracheal wash and FNA of the lungs can be useful in those animals that would not tolerate a general anaesthesia.

A neutrophilia with left shift is only observed in about 25 % of the cases. PCR tests are now widely available for many viral pathogens.

Treatment

Critical cases require hospitalisation for oxygen and fluid therapy (Fig. 1). An initial intravenous broad spectrum antibiotic treatment covering Gram negatives, Gram positives and anaerobes, or based on

culture and sensitivity results should be used for a minimum of three days (preferably). This is followed by a course of oral antibiotics for a minimum of six weeks and until two weeks after complete resolution of the radiographic signs. Nebulisation with saline humidifies the airways and helps mucociliary clearance. Adding gentamicin in the nebulisation liquid might reduce the population of *Bordetella bronchiseptica* in the airway of infected dogs but its use is only recommended as an adjuvant treatment. Respiratory physiotherapy (coupage, at least four times daily) is an important part of the treatment of bronchopneumonia. It helps eliciting a cough which improves the clearance of mucopurulent material. Corticosteroids and anti-tussives are strongly contraindicated.

PARASITIC INFESTATION

Aetiology

Oslerus osleri, *Filaroides hirthi*, *Crenosoma vulpis*, *Angiostrongylus vasorum*, *Pneumocystis carinii* and migratory ascarides are the more common causes of pulmonary parasitic infestation in the dog. *Aelurostrongylus abstrusus* and migratory ascarides are possible causes in the cat. Mainly younger animals are affected. Pulmonary involvement with *Toxoplasma gondii*, a protozoal disease, is common in cats with acute disease.

Clinical signs

Most animals do not show any clinical signs. Those that do show clinical signs often have a violent, gagging-like, productive cough. *Angiostrongylum vasorum* infestation has been associated with systemic signs like DIC, right-sided heart failure, thrombo-embolism, posterior paresis and other neurological signs. *Toxoplasma gondii* often causes pulmonary lesions (in cats) but the clinical signs can vary tremendously depending on the other organs that are involved in the disease process (hepatic, neurological, muscular and ocular signs).

Diagnosis

The physical examination and thoracic radiography are often non-specific. Bronchoscopy helps in

Fig. 6: *Bronchoscopic image of the airways infested with adult lungworm.*

identifying larvae, adult worms (Fig. 6) or nodules at the tracheobronchial bifurcation (mainly *O. osleri*). Bronchoalveolar lavage will occasionally reveal the associated eosinophilic inflammation, and will occasionally show the presence of nematode eggs (Fig. 7). Detection of larvae in a faecal sample examined via the Baermann technique is confirmative but a negative sample does not exclude the presence of lungworm. Therefore, sample collection over multiple days (at least three days) is strongly indicated. *P. carinii* can be diagnosed by FNA of the lungs (Fig. 8). In case of suspicion of an *A. vasorum* infestation investigation of coagulation parameters (platelet count, buccal mucosal bleeding time, PT, APTT, D-dimers) and the presence of pulmonary hypertension (prominent pulmonary arteries on radiographs, echocardiography) might be indicated.
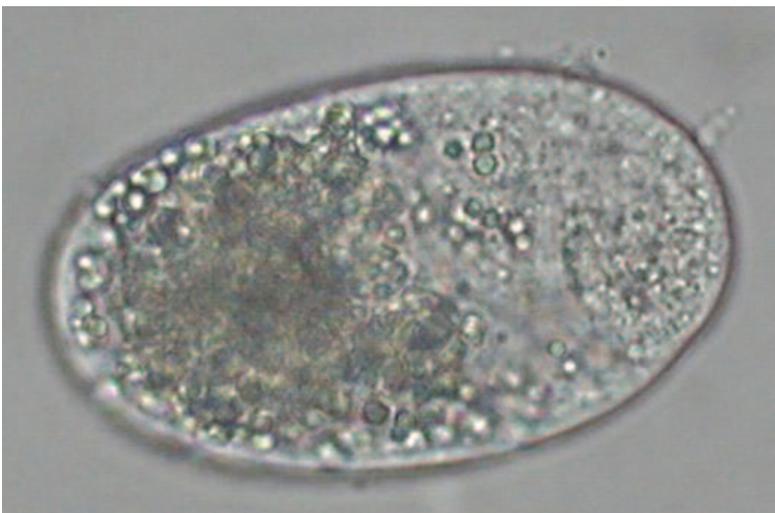

Fig. 7: *A nematode egg as seen in a broncho-alveolar lavage sample.*

Toxoplasma gondii gives a variety of pulmonary radiographic signs (from fluffy alveolar patterns to more consolidated and granulomatous disease, occasionally associated with pleural effusion). For toxoplasmosis, IgM/IgG antibody titres are helpful in differentiating acute disease from old infection. PCR on a broncho-alveolar lavage sample might help in confirming the disease, as does the presence of tachyzoites.
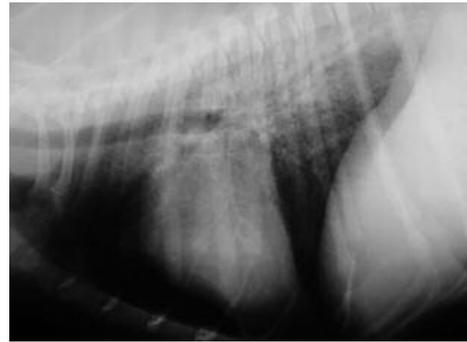

Fig. 8: *Radiographic image of a dog with* Pneumocystis carinii *infestation.*

Treatment (Table 1)
(N.B. These drugs are not all licensed in the UK and owner consent should be obtained.)

IDIOPATHIC PULMONARY FIBROSIS
Prevalence
Idiopathic pulmonary fibrosis (IPF) is a progressive disease most often seen in Terrier breeds (Fig. 9) (mainly West Highland White Terriers) and occasionally in other small breed dogs. It is mainly observed in middle –aged and older dogs. An IPF-like pathology has only recently been described in cats.

Aetiology
The aetiology remains unknown but an allergic component is suspected in some cases, since many WHWT have concurrent atopy. Some drugs (amiodarone, bleomycine) and toxins (paraquat) can also cause similar lesions, but the latter is often rapidly fatal.

Clinical signs
Most animals do not show any clinical signs. Crackles at the end of inspiration are a clinical

TABLE 1: Treatments for parasitic infestation

Antiparasitic treatment should be tailored to the type of infection:	
O. osleri, Filaroides spp:	fenbendazole 50 mg/kg/q 24h for 10 days, repeat 3 weeks later
C. vulpis:	fenbendazole 50 mg/kg/q 24h for 10 days, repeat 3 weeks later; a single oral dose of 0.5 mg/kg milbemycin oxime
A. vasorum:	fenbendazole 50 mg/kg q 24h for 3 weeks, levamisole 10 mg/kg q 24h for 5 days, ivermectine 200μg/kg SQ once (take care with Collies),
P. carinii:	levamisole 5 mg/kg three times a week
A. abstrusus:	fenbendazole 50 mg/kg/q 24h for 10 days; ivermectine 400μg/kg SQ once. In case of secondary infection, a short course of antibiotics might be indicated. Once the infection has been eliminated and if the cough still persists an anti-inflammatory corticotherapy might be indicated. (prednisolone 0.5 mg/kg q 24h for one week followed by alternate day therapy).
Toxoplasmosis:	25 mg q 12-24h

hallmark of the disease. In the more advanced cases expiratory dyspnoea with marked respiratory effort can often be observed. A cough is uncommon and often indicates secondary bacterial infection. Exercise intolerance can be present in the severely affected animals.

Diagnosis

A definite diagnosis of IPF is only confirmed by histopathology of a lung biopsy (Fig. 9). However, in the clinical setting the diagnosis is based on the exclusion of all other causes. End-inspiratory crackles in the predisposed breeds are, in the absence

Fig. 9: *A Terrier with IPF and pulmonary hypertension and oxygen administration via a half closed Elizabethan collar.*

of radiographic evidence of alveolar infiltrates, suggestive for the presence of IPF. Thoracic radiography often shows a marked broncho-interstitial pattern in the caudodorsal lungfields associated with reduced lungfield size and cor pulmonale (Fig. 10). Doppler echocardiography
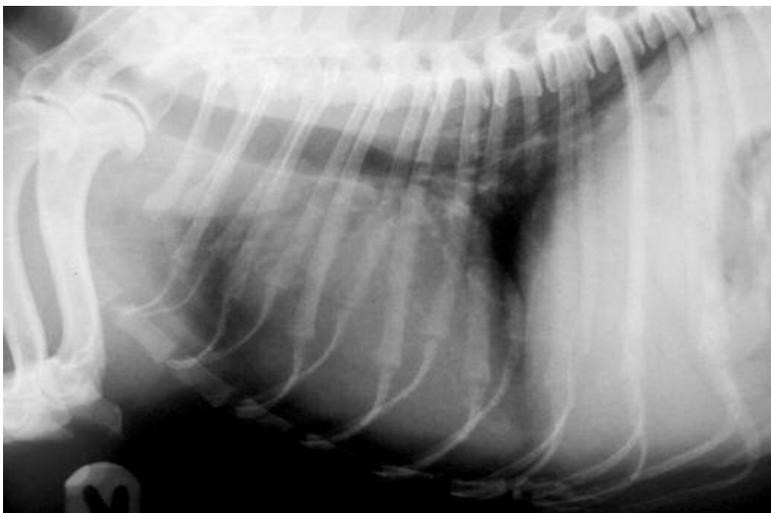
Fig. 10: *Radiographic image of a dog with interstitial pulmonary fibrosis.*

might indicate the presence of secondary pulmonary hypertension. Bronchoscopy and broncho-alveolar lavage are mostly unremarkable (except in the presence of a secondary infection). Under general anaesthesia reduced lung compliance can be noticed but one has to be aware that these animals are extremely sensitive for lung over-inflation and re-expansion injury. High resolution spiral computed tomography might be a non-invasive tool for diagnosing, staging and therapeutically monitoring canine pulmonary fibrosis.

Treatment

Treatment remains purely palliative. Because of the slow but unfortunately fatal progression of this disease the aim of treatment is to try to slow down its progression. At present, there are no evidence-based therapies for idiopathic pulmonary fibrosis. However, corticotherapy remains the treatment of choice in many cases: prednisolone 1-2 mg/kg q 24h for one week followed by a reduction to alternate day therapy for the rest of the animal's life (average 0.5 mg/kg prednisolone q 48h). Immunosuppressive therapy like azathioprines have been tried but with very little beneficial effect. Colchicine, an anti-fibrotic drug, might be useful in the very early stages of the disease (but unfortunately we only see the advanced cases), but is not without adverse effects. Bronchodilators like theophylline have, besides their bronchodilatory properties, the additional potential of fortifying the diaphragm and other respiratory muscles. Intermittent antibiotics should be considered in case of secondary bacterial infection. In cats, response to therapy (corticosteroids, antibiotics, bronchodilators) is poor, and most cats die within days to months.

PULMONARY INFILTRATE WITH EOSINOPHILS (PIE)
Definition

PIE is a canine disease characterised by eosinophilic infiltration of the lungs and bronchial mucosae.

Aetiology

The exact aetiology remains, despite a strong suspicion for an immunological hypersensitivity Type I component, unknown. However parasitic infestation might give similar infiltrates.

Clinical signs

A productive cough associated with retching and gagging are the most common clinical signs. The presence of dyspnoea depends on the extent of the pulmonary infiltrates.

Diagnosis

The lesions seen on thoracic radiographs can vary tremendously in severity: from a mild broncho-

Fig. 11: *Bronchoscopic image of the 'green' mucopurulent discharge often seen with pulmonary infiltrates with eosinophils.*

interstitial pattern to alveolar infiltrates and a granulomatous nodular pattern. Bronchoscopy and broncho-alveolar lavage are essential for confirming the diagnosis (Fig. 11). A peripheral eosinophilia and/or basophilia might be present. Faecal analysis via the Baermann technique should always be performed to exclude underlying lungworm infestation.

Treatment
Corticotherapy is effective in most cases: prednisolone 1 mg/kg q 24h for 1 week, followed by a gradual reduction of the dosage over 6 weeks and using alternate day therapy. Alternatively the author has had success with the use in of cyclosporine (5 mg/kg) in those animals that did not tolerate the corticosteroid treatment. Lungworm treatment should be administered in endemic areas.

BULLOUS EMPHYSEMA
Aetiology
A destruction of the septae between the alveolae causes the development of bullae within the pulmonary parechyma. Two different groups have been defined:
1. The congenital form which is quite rare.
2. A form secondary to chronic respiratory disorders

Clinical signs
Expiratory dyspnoea and end–expiratory crackles are not uncommon in the acquired form. Recurrent unilateral or bilateral pneumothorax, causing sudden onset dyspnoea, are other presenting signs.

Diagnosis
The radiographic signs are often pathognomonic but the absence of radiographic evidence of pulmonary bullae does not exclude the presence of disease. Spiral CT-scan, as well as thoracoscopy can be a useful additional tool to identify and localise the bullae. Exploratory sternotomy (or thoracotomy if the disease is unilateral) should be considered for diagnostic as well as therapeutic purposes in the presence of recurrent pneumothorax. An underlying disease, like bronchiectasia and pulmonary neoplasia should also be excluded.

Treatment
The underlying disease should be treated but pulmonary lobectomy/pneumectomy is often indicated when the disease is not generalised.

REFERENCES AND FURTHER READING
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TABLE 2: Recommended initial diagnostic tests for the more common bronchial and lower airway diseases

Disease	More common clinical signs	Recommended diagnostic tests
Canine infectious bronchopneumonia	Cough, pyrexia, lethargy	Thoracic X-rays BAL (culture/cytology)
Feline infectious bronchopneumonia	Dyspnoea, anorexia, pyrexia	Thoracic X-rays BAL (culture/cytology) FIV/FelV
Chronic bronchitis	Dry chronic cough	Thoracic X-rays BAL (culture/cytology)
Bronchiectasia	Dry chronic cough	Thoracic X-rays BAL (culture/cytology)
Lungworm	Gagging cough, otherwise fit animal	Thoracic X-rays Bronchoscopy and BAL Baermann faecal analysis
Feline asthma	Chronic dry cough Acute onset life-threatening dyspnoea	Thoracic X-rays Bronchoscopy and BAL
Feline toxoplasmosis	Clinical signs depend on organ involvement but mostly generalised disease	Thoracic X-rays IgM/IgG titres
Idiopathic pulmonary fibrosis	Expiratory dyspnoea, abdominal breathing, exercise intolerance, rarely cough	Thoracic X-rays Spiral CT-scan Pulmonary biopsy
Pulmonary infiltrate with eosinophils	Productive cough	Thoracic X-rays Bronchoscopy and BAL (Bronchial biopsy if possible) CBC, Baermann faecal analysis
Bullous emphysema	Recurrent pneumothorax, Occasionally cough	Thoracic X-rays Spiral CT-scan Thoracoscopy/exploratory sternotomy

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CPD

These multiple choice questions are based on the above text. Readers are invited to answer the questions as part of the RCVS CPD remote learning program. Answers appear on page 99. In the editorial panel's view, the percentage scored, should reflect the appropriate proportion of the total time spent reading the article, which can then be recorded on the RCVS CPD recording form.

1. **Which of the following statements regarding bronchopneumonia is incorrect:**
   a. Infectious bronchopneumonia is in most cases a secondary bacterial pulmonary colonisation after a primary viral infection.
   b. A productive cough and pyrexia are the most common clinical signs.
   c. There is not always a correlation between the severity of the clinical signs and the radiographic changes.
   d. In case of recurrent bronchopneumonia an underlying immunodeficiency should be considered.
   e. Corticosteroids and anti-tussives are strongly indicated.

2. **Which of the following statements regarding lungworm infection is incorrect:**
   a. It is often associated with an eosinophilic pulmonary inflammation.
   b. Nodules at the tracheal bifurcation might indicate *Oslerus osleri* infection.
   c. *Angiostrongylum vasorum* infestation has been associated with systemic signs like DIC.
   d. It occurs mainly in old animals.
   e. Faecal analysis for larvae via the Baermann technique is the diagnostic test of choice but false negatives are possible.

3. **Which of the following statements regarding idiopathic pulmonary fibrosis is incorrect:**
   a. IPF does not occur in cats.
   b. It mainly affects Terrier breeds.
   c. Crackles at the end of inspiration are a clinical hallmark of the disease.
   d. A definite diagnosis of IPF is only confirmed by histopathology of a lung biopsy.
   e. Corticotherapy remains the treatment of choice in many cases.

4. **Which of the following statements regarding pulmonary infiltrates with eosinophils is incorrect:**
   a. *Crenosoma vulpis* infestation can mimic the clinical picture of PIE
   b. Thoracic radiography is pathognomonic and normal radiographs exclude the presence PIE
   c. Bronchoscopy and broncho-alveolar lavage are essential for confirming the diagnosis.
   d. Faecal analysis via the Baermann technique should always be performed.
   e. A peripheral eosinophilia is only present in a limited number of cases.