COMPANION OR PET ANIMALS

Chronic pneumonia and focal bronchiectasis in a Siberian husky dog

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Received 11 October 2017
Revised 11 January 2018
Accepted 2 February 2018

SUMMARY

An eight-year-old male Siberian husky dog was evaluated for chronic coughing. Thoracic radiography revealed a diffuse bronchointerstitial pattern and dilatation of the left cranial and left caudal lobar bronchi. Bronchoscopy confirmed marked dilatation of the primary bronchi in the left cranial and caudal lung lobes. Bronchoalveolar lavage revealed septic, neutrophilic inflammation. A diagnosis of severe, focal, cylindrical bronchiectasis secondary to chronic bronchopneumonia was made. Initially, the dog responded well to antimicrobial therapy; however, clinical signs returned after cessation of therapy and generalised bronchiectasis developed after seven months. Chronic bacterial bronchopneumonia should be considered as a cause of focal and generalised bronchiectasis in dogs.

BACKGROUND

Bronchiectasis is a permanent and debilitating result of chronic airway injury that is characterised by dilatation of the bronchi. The disease is not well described in veterinary medicine, and focal disease is the least commonly reported distribution. This is a report of focal bronchiectasis in a dog.

CASE PRESENTATION

An eight-year-old, 26.8 kg, male, neutered, Siberian husky dog presented with a two-year history of continual non-productive cough. Amoxicillin clavulanic acid (17 mg/kg/day for 10 and 7 days on two separate occasions), cephalexin (18 mg/kg/day for 14 days) and oxytetracycline (8.9 mg/kg/day for 25 days) had been administered with limited response. Glucocorticoid (prednisolone 1 mg/kg/day for five days followed by 0.7 mg/kg/day for 40 days) therapy was subsequently administered with minimal response. No medications had been administered in the two months preceding referral other than a course of fenbendazole (50 mg/kg/day for seven days), which had been completed one week prior to referral. No concurrent diseases were reported and the dog was fully vaccinated.

At presentation, physical examination revealed increased adventitious lung sounds throughout both lung fields and loud moist crackles were heard after coughing. Tracheal pinching induced severe, non-productive, paroxysmal coughing. Resting respiratory rate was 40 breaths per minute. Cardiac auscultation was unremarkable with a heart rate of 80 beats per minute. The remaining physical examination was unremarkable.

INVESTIGATIONS

Routine clinicopathological testing was performed. Haematology revealed mild monocytosis 2.16 x 10^9/l (reference interval 0–1.35 x 10^9/l), a neutrophil count of 7.73 x 10^9/l (reference interval 3–11.5 x 10^9/l) and an eosinophil count of 1.24 x 10^9/l (reference interval 0–1.47 x 10^9/l). Examination of a blood smear revealed occasional reactive mononuclear cells, with otherwise normal morphology of red and white blood cells. All plasma biochemical parameters were within reference interval. *Angiostrongylus vasorum* antigen test (Angio Detect Test, IDEXX Laboratories) was negative. Faecal parasitology (modified Baermann technique) was negative.

Thoracic radiography revealed a diffuse, moderate bronchointerstitial pattern throughout all lung lobes, with persistent dilatation of the left cranial and left caudal lobar bronchi and secondary bronchi on several projections (Fig 1A,B) consistent with focal bronchiectasis. Bronchoscopy revealed normal tracheal structure, with mild, diffuse hyperaemia of the mucosa. The mucosa at the level of the carina was moderately thickened and severe dilatation of the left cranial and caudal primary lobar bronchi was seen (Fig 2A,B). Secondary and tertiary bronchi were moderately dilated. The bronchial mucosa within the cranial and caudal left lobes was diffusely hyperaemic and friable. Intraluminal mucopurulent exudate was present throughout the affected areas. The right primary and secondary bronchi were also hyperaemic with mild to moderate thickening of the interbronchial septa with moderate amounts of white mucus. Bronchoscopically guided bronchoalveolar lavage (BAL) from left caudal and right cranial lung lobes was performed. Cytological examination of BAL fluid (Fig 3) revealed a large number of cells in a dense background of necrotic cellular material and large numbers of thin, filamentous chains of dark bacterial cocci and small numbers of larger cocci. Markedly degenerate neutrophils were recognised as the predominant (>90 per cent) cell type. These changes were considered consistent with septic inflammation. *Escherichia coli* and *Streptococcus canis* were isolated. Both bacteria were susceptible to amoxicillin clavulanic acid, determined by broth microdilution.

DIFFERENTIAL DIAGNOSIS

Differential diagnosis for chronic cough

1. infectious (bacterial, parasitic pneumonitis, protozoal, viral, fungal);
2. inflammatory/allergic (eosinophilic bronchopneumopathy, chronic bronchitis, allergic bronchitis);

To cite: Nerhagen S, Shiel RE. *Vet Rec Case Rep* Published Online First: [*please include* Day Month Year]. doi:10.1136/vetreccr-2017-000543

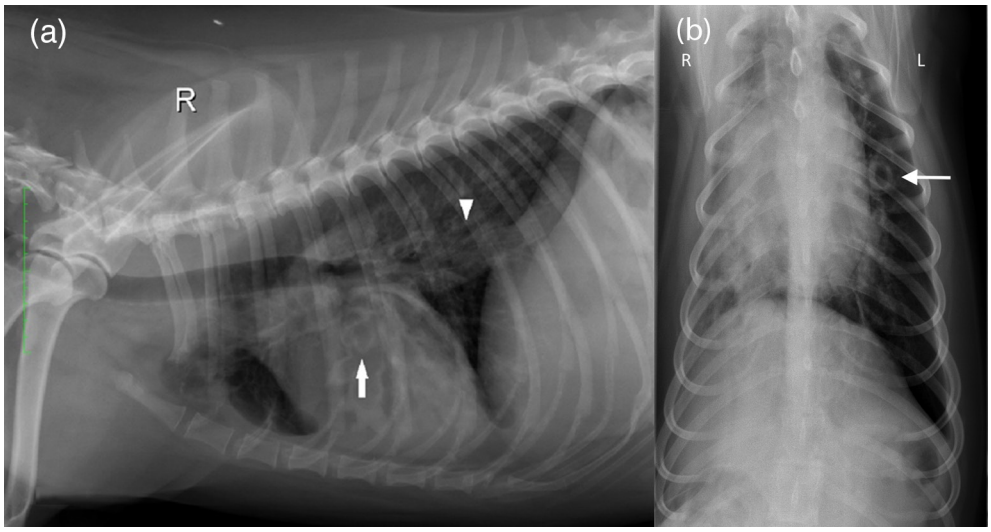

FIG 1 Thoracic radiographs. (a) Right lateral view of a dog with chronic coughing demonstrating cylindrical bronchiectasis of the left cranial (arrow) bronchus. A mild diffuse interstitial pattern is seen throughout all lung lobes (arrowhead). (b) Cylindrical bronchiectasis also demonstrated in dorsoventral (DV) view. Note that the increased opacity in the right hemithorax is secondary to recumbency artefact.

3. obstructive (tracheal collapse, bronchomalacia, laryngeal paralysis, laryngeal collapse, obstructive neoplasia, foreign body);
4. cardiovascular (eg, pulmonary oedema [cardiogenic or non-cardiogenic], cardiomegaly);
5. neoplastic (eg, primary, metastatic);
6. other (eg, dysphagia, gastrointestinal reflux, tracheo-oesophageal or broncho-oesophageal fistula).

Differential diagnosis for bronchiectasis

1. infectious (eg, chronic bronchopneumonia);
2. inflammatory (eg, eosinophilic bronchopneumopathy, chronic bronchitis, chronic pulmonary fibrosis);
3. obstructive (eg, foreign body, broncholithiasis, obstructing neoplasia);
4. congenital diseases (eg, primary ciliary dyskinesia, primary immune deficiency).

Based on history and physical examination findings in this case, certain diagnoses were considered less likely: parasitic pneumonitis was unlikely due to negative testing and recent fenbendazole therapy, pulmonary fungal disease is very uncommon in Ireland and uncomplicated viral infections are typically not associated with such a chronic disease process. Bacterial infection was identified in this case; however, an underlying, predisposing disease could not be excluded.

TREATMENT AND OUTCOME

Amoxicillin clavulanic acid (20 mg/kg twice daily for seven weeks) treatment was initiated with good response. The frequency of coughing was greatly reduced after three weeks of treatment. The owner declined further investigation such as repeat radiography or CT to consider surgical removal of the affected lung lobes (left cranial and caudal). The dog was referred back to the care of the primary veterinarian.

The cough persisted at lower frequency over most of the next seven months. Intermittent courses of antimicrobial drugs (amoxicillin clavulanate and doxycycline) were prescribed

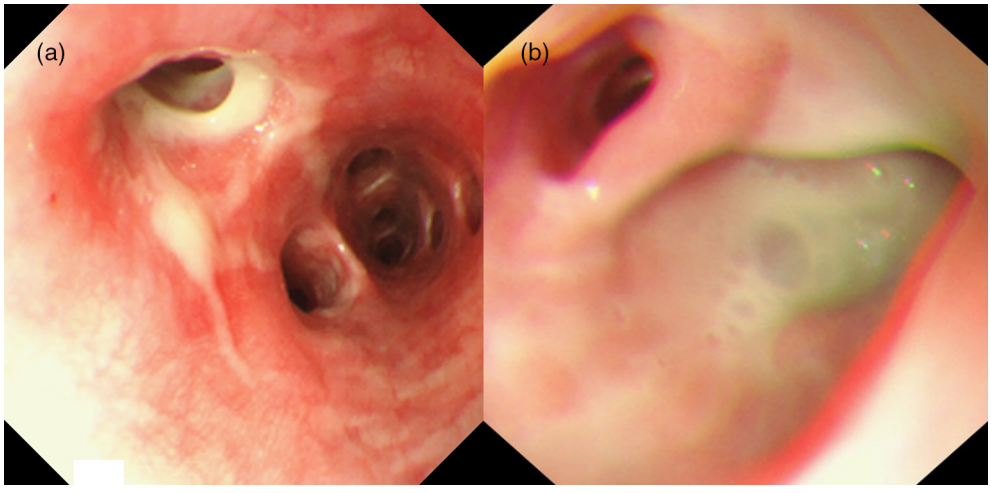

FIG 2 Bronchoscopy. (a) Endoscopic image at the level of the left cranial lobar bronchus. Diffuse mucosal hyperaemia and a moderate amount of mucopurulent material are evident. (b) Endoscopic image at the level of the left caudal lobar bronchus. Partial to full obstruction of the lumen with yellow-green mucopurulent material is evident.

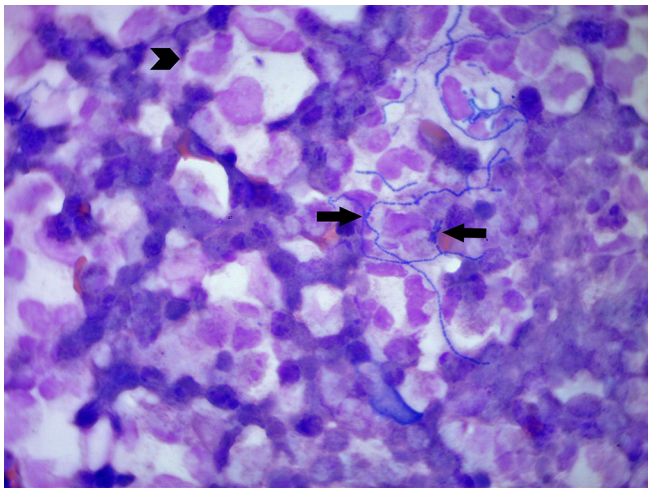

FIG 3 Bronchoalveolar lavage (BAL) cytology obtained from the left caudal lung lobe. Markedly degenerate neutrophils (arrowhead) and a large number of extracellular and intracellular cocci (arrows) are evident.

during periods of relapse. Prednisolone was also prescribed at anti-inflammatory doses (initial dose 0.7 mg/kg/day, decreasing to 0.3 mg/kg/day). This resulted in a marked decrease in the severity and frequency of cough, which was limited to periods of excitement. Radiographs repeated after seven months revealed diffuse bronchiectasis of left and right lung lobes, with marked dilatation of the left lobar bronchi and secondary bronchi.

DISCUSSION

Bronchiectasis is a permanent and abnormal dilatation of bronchi, which usually develops because of chronic non-specific airway inflammation with subsequent destruction of bronchial wall integrity.[1 2] Dilatation occurs secondary to weakening or destruction of the bronchial wall from loss of elastin and destruction of muscle and cartilage.[1] The damage impairs mucociliary clearance, which results in reduced clearance of airway secretions, providing an environment for infection. Infection exacerbates inflammation which leads to a self-perpetuating cycle of damage.[2–4] However, not all dogs with chronic inflammatory or infectious lung disease develop bronchiectasis, indicating additional abnormalities in immune response or pulmonary clearance.[4]

Bronchiectasis in people is generally divided into cystic fibrosis (CF) and non-CF (N-CF) related causes.[2 5] Most people with CF develop bronchiectasis, with bronchiectatic changes found in more than 50 per cent of end-stage CF lungs on autopsy.[6] However, the prevalence varies highly between geographic areas and age groups.[7] Prevalence in children with CF is unknown, but in Australia an estimated prevalence of 50–70 per cent by three to five years has been reported.[8] The pathophysiology of bronchiectasis in patients with CF has not yet been fully elucidated, but both reduced mucous production and increased sodium and decreased chloride absorption, causing thickening of mucus, have been proposed as a cause of reduced mucociliary clearance leading to bronchiectasis.[9]

Although bronchiectasis is a common complication of CF in human medicine, most bronchiectasis cases are not associated with CF.[7] In the N-CF group, a number of diseases are included: congenital defects of large airways (eg, bronchial cartilage deficiency), ciliary dysfunction, lung injury (eg, tuberculosis and aspiration pneumonia), hypersensitivity (eg, allergic bronchopulmonary aspergillosis), obstructions, autoimmune diseases (eg, rheumatoid arthritis) and immune deficiency (eg, HIV and immunoglobulin deficiency).[5]

In dogs, the underlying causes of bronchiectasis are not well characterised. The diseases most commonly associated with this condition are pneumonia, eosinophilic bronchopneumopathy and inflammatory airway disease such as chronic bronchitis.[4 10 11] Other less common causes are bronchial foreign bodies and primary ciliary dyskinesia.[1]

In people, bronchiectasis can be classified morphologically as cylindrical, saccular, cystic or varicose.[1 4] Both cylindrical and saccular forms have been reported in dogs.[4] The cylindrical pattern is more common and characterised by loss of the normal distal tapering of the thick-walled bronchi resulting in a uniform, tubular dilatation.[1] By contrast, saccular bronchiectasis is characterised by balloon-like dilatation of the distal bronchi and is thought to represent a more advanced manifestation of the cylindrical form.[4]

Bronchiectasis can also be classified by distribution as diffuse, multifocal or focal.[1 10] Lesion distribution often reflects the nature of the underlying disease; as most causative diseases are diffusely distributed, diffuse or multifocal bronchiectasis is most common.[11] In dogs, focal bronchiectasis is relatively rare, accounting for 5–11 per cent of all cases.[1 10] Although this may reflect a local underlying disease, the process leading to diffuse bronchiectasis may initially be associated with asymmetric or focal bronchiectasis; one dog with focal bronchiectasis without any obvious underlying disease was reported to develop diffuse disease after lobectomy of the first-affected lung lobe.[1]

Radiographic changes may not be apparent in cases of early or mild bronchiectasis. Sensitivity of thoracic radiography has not been reported in larger studies in dogs, but in a recent retrospective study, thoracic radiology was suggestive of bronchiectasis in only 60 per cent of subsequently confirmed cases.[10] In people, the sensitivity of thoracic radiography is reported to be as low as 37–47 per cent, and the preferred diagnostic modality is high-resolution CT (hr-CT) because it detects small changes in the airways more readily.[1 2 5] In dogs, both bronchoscopy and hr-CT have been shown to be highly useful; in the study by Johnson *et al.* in 2016,[10] bronchiectasis was diagnosed by bronchoscopy in 92 per cent, and hr-CT in 100 per cent, of cases. It is important to remember that both modalities have advantages and limitations. Bronchoscopy can be limited by the inability of the endoscope to access more peripheral airways and the image can be obscured by mucus plugging; however, bronchoscopic sampling is often essential to identify the underlying disease process. CT can be limited by user variability, spatial resolution and slice thickness, but provides more comprehensive images of bronchopulmonary structures.[4]

Treatment of choice depends on the underlying condition. Secondary bacterial infections are common in dogs with a variety of airway diseases including bronchiectasis, as previously discussed. Therefore, correct antimicrobial treatment based on culture and susceptibility testing is important.[10–12] Most common bacterial isolates cultured from canine airways include enteric bacteria (*E. coli*, *Klebsiella)*, *Pasteurella* species, *Mycoplasma* species, *Bordetella bronchiseptica*, *Staphylococcus* species and *Streptococcus* species.[11 12] The antimicrobial course length may vary, but it is generally advised to treat 10–14 days after complete clinical and/or radiographic resolution, meaning administration for typically a total of three to six weeks.[11] Recently published guidelines for antibiotic treatment of respiratory diseases state that a re-examination should be performed

10–14 days after commencement of therapy to decide if the duration should be extended.[12] The decision to discontinue or extend therapy should be based on clinical, radiological and haematological findings.[12] Overzealous or prolonged courses of therapy may predispose for development of antibiotic resistance.[5] A recent study assessed the value of C-reactive protein (CRP) to guide antimicrobial therapy in dogs with bacterial pneumonia.[13] Using CRP, treatment duration was significantly decreased without increasing number of relapses, suggesting that empirical treatment duration recommendations may be excessive.[13] The use of CRP may be a good option to guide therapy; however, additional studies are warranted to assess baseline CRP concentrations in dogs with this chronic inflammatory disease.

Adjunctive therapies such as bronchodilators, nebulisation and coupage may be of benefit in cases of bronchiectasis. In people, it is advised to perform a therapy trial with bronchodilators in all patients diagnosed with bronchiectasis and β2 agonists (eg, salbutamol) are recommended for the trial.[5] Routine use of methylxanthines (eg, theophylline) is not recommended due to lack of evidence.[5] In dogs, there are no published recommendations; however, bronchodilators could reduce inflammatory bronchospasm,[4] but the effect may be limited by reduced bronchial airflow (eg, from secretions) and from destruction of the bronchial wall.[4 5] Nebulisation is the delivery of aerosol droplets (typically saline) by the use of a nebuliser to the lower airways. Nebulisation is believed to help clear airway secretions[4 11] and is regularly used in cases of canine bacterial pneumonia[11] and could be beneficial for patients with bronchiectasis. Coupage is a gentle and rhythmic pounding with cupped hands to the lateral thoracic walls to help animals expectorate and is often used in combination with nebulisation[11]; although coupage is commonly recommended in affected animals, objective evidence of benefit is lacking.

Bacterial pneumonia is one of the most common diagnoses in dogs with acute or chronic respiratory disease and the most common cause of canine bronchiectasis.[4 10 11] Primary bacterial pneumonia is uncommon and is usually secondary to an underlying trigger such as initial viral pneumonia, aspiration pneumonia, foreign body inhalation or chronic inflammatory disease.[11] Although bronchiectasis is irreversible, prompt identification and effective treatment is necessary to attempt to slow the cycle of destruction and preserve the integrity of unaffected bronchi. As seen in this case, as well as a case previously reported,[1] focal bronchiectasis can progress to a generalised form.

Surgical removal of abnormal lung lobes is an alternative for dogs with focal disease or a foreign body,[1 4] and should be considered as the presence of bronchiectasis will predispose the dog to recurring infections.

In this case, the diagnosis of a bacterial bronchopneumonia was not unexpected. Unfortunately, the owner refused further diagnostics and more aggressive treatment (eg, surgery) due to improvement on initial antibacterial therapy. However, the dog developed generalised bronchiectasis by seven months after presentation. Dogs with bronchiectasis can be considered to have a fair to good prognosis, but it likely depends to some extent on the ability of owners to seek out and carry through with appropriate veterinary care.

Contributors SN wrote the initial draft of this document and oversaw parts of the treatment. RES oversaw treatment and made final edits to the case report.

Funding This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests None declared.

Provenance and peer review Not commissioned; externally peer reviewed.

Data sharing statement Unpublished information about the case report, e.g. full blood results can be provided should it be needed through the corresponding author.

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*Vet Rec Case Rep*2018 6:
doi: 10.1136/vetreccr-2017-000543

Notes