PAPER

Aspiration pneumonia in the Irish wolfhound: a possible breed predisposition

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Background: Anecdotal reports suggest a recent high prevalence of aspiration pneumonia in Irish wolfhounds, prompting further investigation into the incidence of the disease in this breed.

Objectives: To investigate the possibility that Irish wolfhounds have an increased incidence of aspiration pneumonia, and to consider possible predisposing causes in this breed.

Methods: Retrospective review of medical records from the Small Animal Specialist Hospital, Sydney, from January 2008 to December 2012 inclusive to determine the total hospital incidence and individual breed incidences of aspiration pneumonia.

Results: The total hospital incidence of aspiration pneumonia was 0·5%. The Irish wolfhound had the highest breed incidence, with 9 of 25 dogs (36%) diagnosed with aspiration pneumonia. Four of the Irish wolfhounds had a predisposing cause identified; one having a choking episode, one having gastric bloat, while two were diagnosed with laryngeal paralysis after it was specifically investigated because of clinical suspicion. Five had no underlying cause of their aspiration pneumonia determined.

Clinical Significance: On the basis of the hospital population studied, the Irish wolfhound has a high incidence of aspiration pneumonia. Further investigation into the possible predisposing cause(s) in this breed is warranted.

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INTRODUCTION

The Irish wolfhound is an uncommon breed in Australia, comprising just 0·15% of dogs registered with the Australian National Kennel Council (Australian National Kennel Council 2013), and 0·09% of the cases seen at a busy small animal referral hospital in Sydney.

Despite the rarity of the breed, a subjectively high number of Irish wolfhounds have recently been treated for aspiration pneumonia (AP) at the Small Animal Specialist Hospital, North Ryde, prompting further investigation into the incidence of the disease in this breed.

The Irish wolfhound is considered predisposed to multiple disease processes, affecting the cardiovascular, cutaneous, endocrine, gastrointestinal/hepatic, immune, coagulation, skeletal, ophthamological and respiratory systems (Gough & Thomas 2004). The best known predisposed respiratory disease is rhinitis/bronchopneumonia syndrome, where a chronic rhinitis is the primary disease process while bronchopneumonia develops secondarily, with the disease typically occurring in young dogs and often being present from birth (Clercx *et al.* 2003, Wilkinson 1969). Immunodeficiency was proposed as the cause of the rhinitis/bronchopneumonia syndrome in three related dogs in one study (Leisewitz *et al.* 1997). There has been no other reported predisposition to the development of pneumonia in the Irish wolfhound. A review of the veterinary literature found no manuscripts discussing pneumonia in the Irish wolfhound, and only one manuscript discussing bacterial pneumonia in dogs that had a single Irish wolfhound as part of a case series (Wingfield *et al.* 1997).

Pneumonia can develop secondary to opportunistic normal airway flora, entry of microorganisms into the lower respiratory tract via inhalation/aspiration, local extension from intrathoracic structures or haematogenous spread (Lee-Fowler & Reinero 2012).

To establish a definitive diagnosis of bacterial pneumonia, airway sampling via broncho-alveolar lavage (BAL), trans-tracheal wash or endotracheal wash is required (Schulze & Rahilly 2012). However, it is common in a clinical setting for veterinarians to rely exclusively on thoracic radiographs, clinical signs and other clinicopathological results (such as haematology) to presumptively diagnose pneumonia, particularly when diagnosing AP (Kogan *et al.* 2008a, Tart *et al.* 2010). A retrospective case series by Kogan *et al.* (2008a) and Tart *et al.* (2010) found that in a clinical setting, only 5 and 38% of cases respectively that were diagnosed with AP had sampling of the airways to document microbial infection. In human medicine the term AP specifically refers to the development of a radiographically evident infiltrate in patients who are at increased risk for oropharyngeal aspiration (Marik 2001).

The radiographic changes that support a diagnosis of pneumonia in dogs are the presence of alveolar and/or interstitial pulmonary patterns. AP typically has these changes in dependent lung lobes, with the right middle, right cranial and caudal segment of the left cranial lung lobe most commonly affected (Kogan *et al.* 2008b, Lamb 2007, Tart *et al.* 2010). The primary differentials for a radiographic alveolar or interstitial pulmonary pattern include pneumonia, pulmonary oedema, haemorrhage, neoplasia and atelectasis (Cohn 2010), with the primary disease process being determined based on history, clinical signs, additional testing and a response to treatment.

The purpose of this study was to determine the incidence of AP in the Irish wolfhound in a selected population, as well as to assess the possible condition(s) that may predispose to AP in this breed.

MATERIALS AND METHODS

Case selection

Medical records of the Small Animal Specialist Hospital, Sydney, were searched to identify all Irish wolfhounds that had presented to the hospital over a five-year period, from January 2008 to December 2012 inclusive. Medical records of these dogs were reviewed to determine the clinical diagnosis for each case. For those that had received a presumptive clinical diagnosis of AP, the medical records and digital radiographs were retrieved for detailed analysis.

To allow a comparison to the study population, a medical record search was also performed to identify all patients diagnosed with presumptive AP during this period. This allowed establishment of both the total incidence and individual breed incidences of presumptive AP in the hospital population.

Inclusion and exclusion criteria

Inclusion criteria were Irish wolfhounds that had received a clinical diagnosis of presumptive AP with complete medical records and radiographs for review.

Cases were excluded if the medical records were incomplete, radiographs were not available for review, or if there was a finding or suspicion of underlying pulmonary pathology not consistent with AP after review of the radiographs and medical records.

Presumptive diagnosis

AP was established as a presumptive diagnosis based on a radiographic finding of alveolar or interstitial pulmonary patterns in dependent lung lobes, acute onset of clinical signs associated with the respiratory tract, and no other disease process or abnormality was detected to explain the presence of radiographic pulmonary changes and clinical signs.

Review of medical records

Records of the Irish wolfhounds that had presumptive AP as a clinical diagnosis were evaluated, assessing the signalment, history, presenting clinical signs and any possible condition(s) that may have predisposed to the development of AP. Digital radiographs of these patients were reviewed by a board-certified radiologist to confirm the presence of alveolar +/– interstitial patterns in dependent/ventral lung lobes, with the absence of other pulmonary pathology, thereby supporting the clinical diagnosis of AP.

RESULTS

The total number of animals that had received a clinical diagnosis of AP over the study period was 138, giving an overall incidence for the hospital population of 0·5%. Twenty five Irish wolfhounds had presented for assessment during the time period examined. Nine of the 25 (36%) had AP listed as a clinical diagnosis. Table 1 lists the incidence of presumptive AP for individual dog breeds in the hospital population. For those breeds not listed, the incidence was found to be less than 2%.

For all nine Irish wolfhounds, the medical records were considered complete and radiographs were available for review. All nine dogs had clinical signs and radiographic pulmonary changes consistent with pneumonia. The radiographic changes involved the ventral/dependent lung lobes in all dogs (Fig 1). All nine dogs had been clinically well before the acute onset of respiratory

Table 1. Different dog breeds and the incidence of aspiration pneumonia

Breed	Incidence of aspiration pneumonia (%)
Irish wolfhound	36
Maremma sheepdog	12·50
Basset hound	8·80
Bulldog	6·40
Old English sheepdog	5·00
Dogue de Bordeaux	4·70
Griffon bruxellois	4·50
Pekingese	4·40
Welsh springer spaniel	4·20
Rhodesian ridgeback	3·80
Boston terrier	2·90
Poodle (miniature)	2·90
Scottish terrier	2·90
Great Dane	2·70
Shetland sheepdog	2·60
Keeshond	2·50

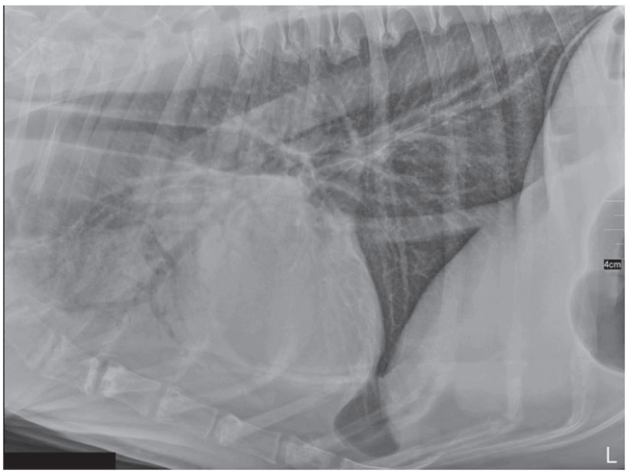

FIG 1. Left lateral thoracic radiograph of dog 3, demonstrating an alveolar pattern in the right cranial lung lobe. The small volume of air in the oesophagus, also the stomach, was thought secondary to aerophagia

signs. On the basis of the history, physical examination findings, additional clinicopathological testing and response to treatment, other differentials for the pulmonary changes, including pulmonary oedema, pulmonary haemorrhage and pulmonary neoplasia, were excluded. Therefore no cases were excluded from the study.

The details of the nine cases (signalment, history, physical examination findings, clinical pathology results and radiographic findings) are summarised in Table 2.

Two dogs were reported to have had an episode before presentation known to predispose to aspiration, with one choking on a pig's ear and one having gastric bloat. Two were diagnosed with laryngeal paralysis, a disease known to predispose to AP (Barton 2004), after undergoing laryngeal examination to investigate a clinical suspicion of laryngeal dysfunction owing to a reported recent change in their bark. For the remaining five dogs the cause of the AP remained unknown, but laryngeal function was not assessed in these dogs.

No dogs underwent investigation of oesophageal function, but none had a history of dysphagia or regurgitation, and none had evidence of megaoesophagus on plain thoracic radiography. None of the nine dogs were reported to have had an anaesthetic or been sedated in the two weeks before presentation. No dogs underwent cardiac evaluation, because of a low clinical suspicion of cardiac disease as the cause of the radiographic pulmonary changes. No dogs had viral titres performed to investigate the possibility of viral disease as the precipitating cause of the pneumonia. No dogs were reported to have recently been in boarding kennels or exposed to large groups of dogs.

All dogs received treatment, one as an outpatient while eight were admitted for in-hospital treatment. Only one of the nine dogs had airway sampling with a BAL, which was performed five days after admission to hospital because of clinical deterioration despite treatment, which diagnosed a multidrug-resistant *Klebsiella pneumoniae* infection. This was the dog that had choked on a pig's ear and it did not survive to discharge. Eight dogs responded to treatment with resolution of clinical signs and resolution of pulmonary radiographic changes. For the five dogs where the cause of AP remained unknown, three had multiple recurrent episodes while one had a single repeat episode of AP, each of which required treatment. Two dogs were ultimately euthanised (one 12 months and one 2-years post initial diagnosis) because of recurrent episodes. A necropsy was not performed on either dog.

DISCUSSION

This study found that 36% of the Irish wolfhounds who presented to a single hospital during the study period were diagnosed with presumptive AP. The overall hospital incidence of AP during the same time period was 0·5%. There are no published reports of the incidence of AP in dog breeds, so in addition to the overall hospital incidence, the individual breed incidences were also determined for comparison. The Irish wolfhound had the highest breed incidence at 36%; with the second and third highest being 12·5 and 8·8%, in the Maremma sheepdog and Basset hound, respectively. For both the Maremma sheepdog and the Old English sheepdog, less than 20 individuals of each breed were treated in the hospital for any problem during the five-year period. Therefore, the incidence of AP in these breeds may have been falsely elevated given the low case numbers seen. The Irish wolfhound constituted 6·5% of the total cases of AP, while the breed made up only 0·09% of all hospital cases during the study period. These findings are supportive of the clinical suspicion that in this hospital population the Irish wolfhound has a higher incidence of AP compared with other dog breeds.

An obvious limitation of this study is its retrospective nature, relying on the accurate recording of case details, and specifically the obtaining and recording of a full clinical history for possible predisposing causes of AP. Being retrospective also means that complete investigation was not performed in all cases, with the final diagnosis of bacterial AP not being definitive, but rather presumptive in eight of the nine dogs, with only one dog having airway sampling performed to document pulmonary bacterial infection. However the history, clinical signs, radiographic changes and response to treatment were consistent with AP in all cases, based on the clinical diagnostic criteria of Tart *et al.* (2010) and Kogan *et al.* (2008b).

All dogs were over one year of age at the time of their presentation and diagnosis of presumptive AP. The only dog that had nasal discharge on examination was eight years of age, with none of the dogs having a prior history of nasal discharge, suggesting that rhinitis/bronchopneumonia syndrome and/or an immunodeficiency syndrome was not the cause of the pneumonia in these dogs. The eight dogs who did not have airway sampling all survived to discharge, with repeat thoracic radiography demonstrating resolution of the alveolar/interstitial changes. The fact that these eight dogs had treatment directed at pulmonary bacterial infection, while none were treated for pulmonary oedema or haemostatic abnormalities, with all making a clinical recovery and having resolution of their pulmonary radiographic changes supports the clinical suspicion that the radiographic changes

Table 2. Clinical details of nine Irish wolfhounds with aspiration penumonia

Dog	Signalment	History/presenting complaint	Pertinent physical examination findings at presentation	Abnormalities on clinical pathology	Radiography	Cause of aspiration pneumonia	Outcome
1	Nine-year FN	Acute onset panting and retching two hours before presentation.	Temp: 38·6°C RR: 72/minute HR: 120/minute Thoracic auscultation unremarkable.	Haematology: no abnormalities. Biochemistry: no abnormalities.	Alveolar pattern in the left cranial lung lobe.	Undetermined	In-hospital treatment. Discharged home. Recurrent episodes of AP. Euthanasia 2·5 years post discharge due to recurrent episodes.
2	Seven-year M	Acute onset abnormal breathing/panting and restlessness 24 hours before presentation.	Temp: 38·4°C RR: Panting HR: 120/minute ↑ respiratory sounds, but no crackles/wheezing.	Haematology: WBC: 22 × 10^9/L (5·1 to 16·8), Neut: 19·7 × 10^9/L (2·9 to 11·6). Biochemistry: no abnormalities	Alveolar pattern in the right middle lung lobe	Undetermined	In-hospital treatment. Discharged home. Multiple recurrent episodes of AP. Chronic antibiotic therapy. Currently stable 2·5 years post discharge.
3	Eight-year FN	Panting, coughing, lethargy for three days, then nasal discharge 24 hours before presentation.	Temp: 40·3°C RR: Panting HR: 144/min Left nasal discharge. Abnormal respiratory sounds right mid-thorax.	Haematology: not performed. Biochemistry: not performed.	Alveolar pattern in the right cranial lung lobe and the caudal segment of the left cranial lung lobe.	Undetermined	Outpatient treatment. Signs resolved. Recurrent episode 8 months post discharge. Euthanised 12 months post discharge due to AP recurrence.
4	Eight-year F	Acute onset panting, mild non-productive retching and coughing three hours before presentation.	Temp: 38·7°C RR: 80/minute HR: 100/minute Normal lung sounds.	Haematology: Lymp: 0·7 × 10^9/L (1·1 to 5·1) Biochemistry: Amyl: 343 U/L (500 to 1500), Lip: 188 U/L(200 to 1800)	Alveolar pattern in the right middle and cranial lung lobes, interstitial pattern in the caudal segment of the left cranial lung lobe.	Undetermined	In-hospital treatment. Discharged home. Currently well 18 months post discharge, with no further AP episodes.
5	Two-year M	Choked/gagged on pig's ear three days prior. Panting and lethargy last 48 hours.	Temp: 39·8°C RR: panting HR: 100 Harsh lungs on auscultation.	Haematology: no abnormalities. Biochemistry: no abnormalities. Airway sampling: *Klebsiella pneumonia*	Alveolar pattern in the right middle, right caudal and caudal segment of left cranial lung lobes. Diffuse interstitial pattern.	Aspiration secondary to a choking episode	In-hospital treatment. Death in hospital.
6	Seven-year M	Intermittent retching and gagging for 12 months. Worse in last one week. Laboured respiration, coughing and restlessness last 24 hours. Recent change in bark.	Temp: 39·6°C RR: panting140/minute HR: ↑ lung sounds ventral left thorax, reduced in ventral right thorax.	Haematology: not performed Biochemistry: not performed	Alveolar pattern in the right middle lung lobe, interstitial pattern in the right cranial lobe.	Aspiration secondary to laryngeal paralysis	In-hospital treatment. Discharged home. Multiple recurrent episodes of AP. Euthanasia 12 months post discharge due to the recurrent episodes.
7	One-year MN	Acute onset retching and gagging two hours before presentation. Progressive abdominal distension over the same time period.	Temp: 37·2°C RR: 40/minute HR: 160/min Cyanotic mucous membranes. Abdominal distension.	Haematology: not performed. Biochemistry: not performed.	Alveolar/interstitial pattern in the ventral two thirds of the entire right lung.	Aspiration secondary to gastric bloat	In-hospital treatment. Discharged home. Lost to follow-up.
8	Four-year FN	Acute onset rapid breathing one hour before presentation. Recently altered bark.	Temp: 38·9°C RR: 72/minute HR: 80/min Normal respiratory sounds.	Haematology: WBC: 2·9 × 10^9/L (5·1 to 16·8), Neut: 2·3 × 10^9/L (2·9 to 11·6), Lymp: 0·48 × 10^9× 10^9/L (1·1 to 5·1) Biochemistry: GGT: 12 U/L (0 to 7)	Alveolar pattern in the caudal segment of the left cranial lobe.	Aspiration secondary to laryngeal paralysis	In-hospital treatment. Discharged home. Recurrent episodes of AP. Euthanasia three months post discharge.
9	Two-year MN	Dullness and inappetence last four days. Panting and restlessness last six hours. No coughing, no vomiting, but some throat clearing.	Temp: 39·4°C RR: panting HR: 112/minute Thoracic auscultation unremarkable.	Haematology: Mono: 1·34 × 10^9/L (0·16 to 1·12) Biochemistry: Urea: 2·3 mmol/L (2·5 to 9·6), Lip: 150 U/L (200 to 1800)	Alveolar pattern in the ventral half of the right cranial and middle lung lobes.	Undetermined	In-hospital treatment. Discharged home. Recurrent episode five months post discharge. Thirteen months post discharge remains well, with no further AP episodes.

FN Female neutered, M Male, F Female, MN Male neutered, Temp Temperature, RR Respiratory rate, HR Heart rate, WBC White blood cell, Neut Neutrophil, Lymp Lymphocyte, Amyl Amylase, Lip Lipase, GGT Gamma-Glutamyl Transferase, Mono Monocyte

were associated with AP. Although it is possible that some of the dogs may have had a sterile or viral pneumonitis, as no dogs had airway sampling performed in the first 1 to 2 days after clinical signs developed, it is not possible to differentiate between these possibilities and AP in these cases.

All dogs included in this study were considered healthy by their owners before the acute onset of their clinical signs. No dogs were found to have evidence of thoracic trauma or disease, nor a condition that is considered to predispose to bacterial pneumonia developing via opportunistic normal flora nor haematogenous spread (such as poor body condition, metabolic disease, endocrine disease, immunosuppression, sepsis/bacteraemia or another primary pulmonary pathology) (Cohn 2010, Lee-Fowler & Reinero 2012). This suggests that the pneumonia was most likely secondary to aspiration in all nine dogs.

A study by Kogan *et al.* (2008a) found that the most common cause of AP in dogs was megaoesophagus. While no dogs in this study specifically had their oesophageal function assessed, none had either current or previous clinical signs or radiographic changes to indicate the presence of megaoesophagus, suggesting a different pathophysiological process for the presumptive AP in these dogs.

Laryngeal paralysis is known as a disease of large-breed dogs which results in upper airway obstruction and dyspnoea (Millard & Tobias 2009). The pathophysiology of laryngeal paralysis is the development of a loss of the ability to abduct the arytenoid cartilage(s) and vocal fold(s) leading to narrowing of the glottis. The loss of function also results in reduced protection of the airway during swallowing, making it a potential predisposing cause of AP (Barton 2004). According to Bernoulli's principle, the airway narrowing results in an increase in velocity of airflow, turbulence and negative pressure, which causes laryngeal oedema and swelling and further glottis narrowing and resistance to airflow. This perpetuating cycle ultimately results in clinical dyspnoea when insufficient air is able to enter the lungs to maintain normal respiratory function (Amis *et al.* 1986). It is interesting to note that for the two dogs in this study that were found to have laryngeal paralysis, neither had shown any clinical signs of dyspnoea before the presenting episode of AP. It is hypothesized that this was due to the relatively large size of the Irish wolfhound larynx, where the loss of laryngeal function resulted in glottis narrowing, but not enough to result in the subsequent pathophysiological changes that lead to dyspnoea. If this hypothesis is correct it would make the clinical signs of laryngeal paralysis in the Irish wolfhound more similar to left recurrent laryngeal haemiplegia in horses, where patients do not present for signs of dyspnoea, but rather for inspiratory stridor and/or poor performance (Stick *et al.* 1999). Being domesticated pets that are routinely confined to backyards, Irish wolfhounds may not undergo exertion sufficient to result in inspiratory stridor or notable poor performance. It is therefore possible that laryngeal paralysis may be a subclinical disease in this breed, which is only investigated if an altered bark is noted, or may remain undiagnosed and manifest as the development of AP. This may be similar to what has been found in humans, where subclinical aspiration is known to be a contributor to the development of pneumonia in humans with a variety of disorders (Ramsey *et al.* 2005). The signalment of the Irish wolfhounds in this study, with the median age of seven years, is similar to the signalment associated with laryngeal paralysis in dogs (particularly given the shorter average life span of the Irish wolfhound compared with other dog breeds), which has a mean age at presentation of approximately 10 years (Millard & Tobias 2009).

Given the supposition above, it is possible that had the remaining five dogs with no identified cause of the presumptive AP undergone examination of their laryngeal function, some of them may have also been found to have laryngeal paralysis/paresis. This hypothesis may explain why three of these five dogs went on to have multiple recurrent episodes of AP after the initial episode treated at the hospital.

While all dogs in this study had clinical signs of respiratory tract disease, the most common owner report was that of persistent panting and restlessness. Of the clinical signs associated with pneumonia, coughing is often the first mentioned in reference books discussing the disease (Brady 2004, Cohn 2010). However, recent studies by Kogan *et al.* (2008b) and Tart *et al.* (2010) reported the prevalence of coughing at 57 and 46% respectively, suggesting it is lower than previously thought. In the present study the prevalence was even lower, with only three of the nine cases (33%) having coughing as a presenting clinical sign. While these findings may appear unexpected, physiologically it is intuitive, as the airflow and shear forces generated in smaller airways during a cough are typically inadequate to clear luminal debris (Widdicome 1995). This is supported experimentally, where it has been shown to be difficult to induce a cough from the smaller airways and alveoli (Widdicombe 1995). This suggests that in the absence of coughing as a clinical sign, the possibility of lower airway and alveolar disease should not be excluded.

Tart *et al.* (2010) reported the prevalence of pyrexia at presentation as 43%, similar to that of 44% on admission in this study.

The results of this study warrant consideration that the Irish wolfhound may be predisposed to the development of AP. Therefore, for any Irish wolfhound who presents for assessment of becoming acutely unwell, particularly if persistent panting is a reported clinical sign, even in the absence of a cough and with normothermia, AP should be considered as a differential diagnosis, which may warrant including thoracic radiography as part of the diagnostic evaluation. If AP is found to be present and if no predisposing cause or underlying disease process is identified, then assessment of laryngeal function should be considered to try to establish the cause of the disease and guide future treatment and/or management for the patient. A prospective, multi-institutional study assessing laryngeal functional in any Irish wolfhound diagnosed with AP would more accurately determine the incidence of laryngeal paralysis and its possible association with AP in this breed.

Additional testing that could also be considered would include the assessment of oesophageal function, and if found to be abnormal, further investigation into possible causes (including myasthenia gravis, primary oesophageal disease and lead/organophosphate toxicity) should be considered.

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Conflict of interest

None of the authors of this article has a financial or personal relationship with other people or organisations that could inappropriately influence or bias the content of the paper.

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