SMALL ANIMALS

Demographic, clinical, and radiographic features of bronchiectasis in dogs: 316 cases (1988–2000)

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Objective—To determine demographic, clinical, and radiographic features of bronchiectasis in dogs.

Design—Retrospective study.

Animals—289 dogs identified through the Veterinary Medical Database (VMDB) and 27 dogs examined at the North Carolina State University Veterinary Teaching Hospital.

Procedure—Demographic characteristics of dogs identified through the VMDB were compared with characteristics of the entire population of dogs entered in the VMDB. Medical records of dogs examined at the teaching hospital were reviewed; the diagnosis was confirmed through review of thoracic radiographs.

Results—Analysis of data from the VMDB indicated that American Cocker Spaniels, West Highland White Terriers, Miniature Poodles, Siberian Huskies, English Springer Spaniels, and dogs > 10 years old had an increased risk of bronchiectasis. Among dogs examined at the teaching hospital, coughing was the most common clinical sign. There was evidence for excessive airway mucus but not hemorrhage. A variety of bacterial organisms were isolated from tracheal wash and bronchoalveolar lavage samples. On thoracic radiographs, cylindrical bronchiectasis, generalized disease, and right cranial lung lobe involvement were most common. Seven of 14 dogs for which follow-up radiographs were available did not have any progression of radiographic lesions. Median duration of clinical signs prior to diagnosis of bronchiectasis was 9 months (range, 1 day to 10 years). Median survival time was 16 months (range, 2 days to 72 months).

Conclusions and Clinical Relevance—Results suggest that despite substantial clinical abnormalities, dogs with bronchiectasis may survive for years. Certain purebred dogs and older dogs may have an increased risk of developing bronchiectasis. (*J Am Vet Med Assoc* 2003;223:1628–1635)

Bronchiectasis refers to persistent dilatation of the bronchi resulting from chronic airway inflammation with destruction of the structural integrity of the bronchial walls.[1] Dilatation of the bronchi occurs secondary to traction by surrounding lung tissue on weakened airways. Because bronchiectasis is defined morphologically, it is most commonly diagnosed antemortem through thoracic radiography or computed tomography, although survey thoracic radiography is generally considered to be useful only in the diagnosis of advanced bronchiectasis, as it is insensitive for detection of early abnormalities.[2-5]

Although bronchiectasis is well known to occur in dogs, few scientific publications have primarily addressed this abnormality. The condition has been included in some studies[6,7] of lower respiratory tract disease and has been described in numerous reports[8-13] of ciliary dyskinesia in dogs. A retrospective study[2] emphasizing the radiographic features of bronchiectasis was published over 25 years ago, and since then a single case of acquired bronchiectasis has been described.[14] Therefore, we undertook a review of a national veterinary database and the medical records of patients at a veterinary teaching hospital to determine demographic, clinical, and radiographic features of bronchiectasis in dogs. In addition, we hoped to identify potential risk factors (eg, age, sex, and breed) for bronchiectasis.

Criteria for Selection of Cases

The terms "bronchiectasis" and "bronchiectasis due to infection" were used to search the **Veterinary Medical Database**[a] **(VMDB)** to identify dogs in which a diagnosis of bronchiectasis had been made between January 1, 1988, and December 31, 2000. Information obtained on dogs with bronchiectasis included breed, approximate age at the time of diagnosis (reported in ranges), and sex. For comparison, population statistics for all dogs entered into the VMDB during the same period were also obtained. Follow-up visits by the same patient were excluded from consideration.

In addition to dogs identified through the VMDB, a computer search of the medical records of the North Carolina State University Veterinary Teaching Hospital was performed to identify dogs in which a diagnosis of bronchiectasis had been made between 1988 and 2000. Because the veterinary teaching hospital uses a different coding system than the VMDB, dogs examined at the teaching hospital were not included in the records of dogs entered in the VMDB.

Dogs examined at the teaching hospital were included in the study only if a complete set of thoracic radiographs (ie, ventrodorsal or dorsoventral view and right or left lateral view) was available for review. To verify the diagnosis, thoracic radiographs from dogs

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considered for inclusion in the study were randomly mixed with thoracic radiographs from control dogs examined at the veterinary teaching hospital for reasons other than respiratory tract disease. Each set of radiographs was independently examined by a board-certified radiologist (CRB) and a board-certified internist (ECH) for features consistent with bronchiectasis. Case dogs were included in the study only if both evaluators agreed that radiographic changes were consistent with a diagnosis of bronchiectasis.

Control dogs were identified by searching the medical records of dogs examined by the North Carolina State University veterinary cardiology and oncology services during the study period. Dogs were considered for inclusion in the control group only if a complete set of thoracic radiographs (ventrodorsal or dorsoventral view and right or left lateral view) was available for review and if bronchiectasis was not mentioned in the radiology report. Two control dogs were matched to each potential case dog on the basis of age group (< 1, 1 to 7, or > 7 years old). One of the control dogs was of the same breed as the potential case dog; the other control dog was of mixed breeding. When multiple complete sets of thoracic radiographs from various hospital visits were available for a potential case dog, radiographs from the earliest date on which bronchiectasis was diagnosed and the most recently obtained radiographs were used. Sets of thoracic radiographs from potential case dogs and the control dogs were numbered and presented to the evaluators in random order, as determined with a random number table.

Procedures

Review of medical records—Medical records of dogs examined at the veterinary teaching hospital in which a diagnosis of bronchiectasis was confirmed were reviewed, and the following information was obtained: breed, age, sex, initial clinical signs, duration of clinical signs of respiratory tract disease at the time bronchiectasis was first diagnosed, results of cytologic examination and culture of tracheal wash and bronchoalveolar lavage fluid, bronchoscopic findings, concurrent diseases, and survival time. If there was insufficient follow-up information in the medical record to determine survival time, the referring veterinarian was contacted by telephone for additional information. Records of results of fluoroscopy for evaluation of collapse of the trachea and mainstem bronchi were also reviewed.

In dogs in which > 1 airway fluid sample (ie, tracheal wash or bronchoalveolar lavage fluid) had been collected, results of cytologic examination and bacterial culture of the sample collected most closely to the time of the initial diagnosis of bronchiectasis were used. To investigate possible associations between bronchiectasis and infection with *Bordetella* spp, *Mycoplasma* spp, or *Pseudomonas* spp, culture information was obtained for all airway fluid samples to determine whether these organisms were isolated. The time between last antimicrobial administration and collection of airway fluid samples was also recorded.

Review of thoracic radiographs—All thoracic radiographs were systematically reviewed for signs of bronchiectasis, pulmonary parenchymal disease, tracheal abnormalities, pleural disease, and cardiac disease by a board-certified radiologist (CRB). Bronchiectatic lesions were further characterized as cylindrical (tubular) or saccular (cystic). Cylindrical bronchiectasis referred to dilatation of the bronchi without tapering toward the periphery. Saccular bronchiectasis referred to airway dilatation that included focal saccular dilatations or cyst-like structures. The extent of involvement was described as single lobe, multiple lobe (2 or 3 lobes), or generalized (> 3 lobes). When there was single or multiple lobe involvement, affected lung lobes were identified.

Severity of the bronchiectasis was subjectively classified as mild, moderate, or severe on the basis of location and extent of involvement. Mild bronchiectasis was characterized by single lobe involvement with dilatation of the lobar bronchus as it extended away from the pulmonary hilum toward the periphery; lesions tended to be in a central location. Moderate bronchiectasis was characterized by multiple lobe involvement with predominantly central bronchial changes or single lobe involvement with peripheral extension of bronchiectasis. Severe bronchiectasis was characterized by generalized lung lobe involvement with dilatation of principal bronchi extending into the periphery of multiple lung lobes.

Pulmonary abnormalities other than bronchiectasis were classified as bronchial, interstitial, or alveolar, and severity of these pulmonary changes was subjectively graded as mild, moderate, or severe. Mild or nonspecific bronchial changes such as airway mineralization were not considered, as they could not be distinguished from age-related changes. Mild interstitial pulmonary changes were characterized as an increased interstitial pattern thought to be excessive for age, moderate interstitial changes were characterized as generalized increased lung opacity with border effacement of major and minor pulmonary vessels, and severe interstitial changes were characterized as generalized pulmonary soft tissue opacity with border effacement and minimal air bronchogram formation. Focal pulmonary abnormalities with air bronchograms were considered evidence of mild alveolar pulmonary changes. Moderate alveolar changes involved an entire lung lobe with a discrete lobar sign or multifocal, peripheral air bronchograms. Severe alveolar changes included multilobar changes with discrete lobar signs and border effacement with the cardiac silhouette or diaphragm, depending on location.

Tracheal abnormalities were characterized as narrowing or excessive dilatation; location of the abnormality was recorded. Pleural disease was described as fibrosis (fissure lines only) or effusion. If effusion was identified, the severity was recorded as mild, moderate, or severe. Cardiac disease was described by specific cardiac chamber (if this could be determined) or great vessel enlargement, as described.[15]

All radiographic results were recorded and tabulated independently after the review process. For dogs with bronchiectasis that had multiple sets of radiographs obtained during various visits to the teaching hospital, changes in radiographic findings between the time of diagnosis and the most recent radiographic study were evaluated.

Statistical analyses—Odds ratios (ORs) and their 95% **confidence intervals (CIs)** were calculated for potential risk factors[16]; standard software[b] was used. Fisher exact tests or χ^2 tests with the Yates correction were used to determine whether breed, age, or sex was significantly associated with bronchiectasis. For dogs with bronchiectasis, an unpaired *t* test[c] was used to compare time intervals between sequential thoracic radiographs for dogs that had radiographic evidence of disease progression with time intervals for dogs that did not have radiographic evidence of disease progression. For all analyses, values of $P < 0.05$ were considered significant.

Results

Demographic characteristics of dogs in the VMDB—Between January 1, 1988, and December 31, 2000, records for 582,786 canine accessions were submitted to the VMDB. Bronchiectasis was diagnosed in 289 (0.05%) of these dogs. Fifty-five of the 289 dogs were of mixed breeding; the remainder represented 60 breeds. Five breeds were significantly overrepresented, compared with all dogs entered in the VMDB during the study period (**Table 1**).

Of the 287 dogs for which an approximate age was available, 148 (51.5%) were > 10 years old, whereas only 15.6% of the general population of dogs entered in the VMDB were > 10 years old (OR, 5.78; 95% CI, 4.59 to 7.29; $P < 0.001$). Only 10 dogs (3.5%) were < 1 year old, with the youngest dogs with bronchiectasis being between 2 and 6 months old (n = 6).

The proportion of American Cocker Spaniels with bronchiectasis that were between 2 and 4 years old (10/46; 22%) was significantly ($P < 0.001$) higher than the proportion of all other dogs with bronchiectasis that were in this age group (17/241; 7.1%). Only 16 of the 46 (35%) American Cocker Spaniels were > 10 years old, which was significantly ($P = 0.02$) less than the proportion of all other dogs with bronchiectasis that were this old. All 8 of the affected West Highland White Terriers were ≥ 10 years old.

Table 1—Breed distribution of dogs with bronchiectasis entered in the Veterinary Medical Database between 1988 and 2000 versus all dogs entered in the database during this period

Breed	No. with bronchiectasis (%)	No. in database (%)	OR	95% CI
Mixed	55 (19.0)	129,421 (22.2)	0.82	0.61–1.10
American Cocker Spaniel	46 (15.9)	25,455 (4.4)	4.15	3.03–5.69
Miniature Poodle	15 (5.2)	10,898 (1.9)	2.88	1.71–4.84
Golden Retriever	13 (4.5)	31,016 (5.3)	0.84	0.48–1.46
Labrador Retriever	13 (4.5)	46,787 (8.0)	0.54	0.31–0.94
Siberian Husky	9 (3.1)	6,488 (1.1)	2.86	1.47–5.55
English Springer Spaniel	9 (3.1)	7,737 (1.3)	2.39	1.23–4.64
West Highland White Terrier	8 (2.8)	3,710 (0.6)	4.45	2.20–8.99
Toy Poodle	7 (2.4)	7,823 (1.3)	1.83	0.86–3.86
All dogs	289	582,786	NA	NA

OR = Odds ratio. CI = Confidence interval. NA = Not applicable.

Information on sex was available for 284 of the dogs with bronchiectasis. Sex was significantly ($P < 0.001$) associated with bronchiectasis, with sexually intact female dogs significantly underrepresented (26/284; 9.2%), compared with other dogs (OR, 0.36; 95% CI, 0.23 to 0.55). Castrated male dogs were overrepresented (87/284 [30.6%]; OR, 1.89; 95% CI, 1.47 to 2.43), and neutered dogs were overrepresented (182/284; 64.1%), compared with sexually intact dogs (OR, 1.86; 95% CI, 1.45 to 2.40).

Demographic characteristics of dogs examined at the veterinary teaching hospital—Between 1998 and 2000, 40,340 dogs were examined at the North Carolina State University Veterinary Teaching Hospital. Bronchiectasis was diagnosed in 31 (0.08%) of these dogs; however, only 27 (0.07%) met the criteria for inclusion in the study following review of thoracic radiographs.

Of the 27 dogs included in the study, 8 (30%) were American Cocker Spaniels, 6 (22%) were Miniature Poodles, 3 (11%) were Toy Poodles, and 2 (7%) were Labrador Retrievers. In addition, there was 1 mixed-breed dog and 1 dog of each of the following breeds: Afghan Hound, Basset Hound, German Shorthaired Pointer, Irish Setter, Lhasa Apso, Shih Tzu, and West Highland White Terrier. Compared with the breed distribution of all dogs examined at the teaching hospital during the study period, American Cocker Spaniels (OR, 7.49; 95% CI, 2.83 to 17.95; $P < 0.001$) and Miniature Poodles (OR, 14.73; 95% CI, 4.85 to 37.86; $P < 0.001$) were significantly overrepresented.

Dogs with bronchiectasis ranged from 4 months to 18 years old (mean, 11.0 years; median 12.0 years). Of the 27 dogs, 19 (70%) were > 10 years old and 1 (4%) was < 1 year old. Information on age of all dogs examined at the teaching hospital was not available for statistical comparisons.

Five (19%) of the 27 dogs were sexually intact females, 5 (19%) were spayed females, 7 (26%) were sexually intact males, and 10 (37%) were castrated males. Sex distribution of dogs with bronchiectasis was not significantly different from sex distribution of all dogs examined at the teaching hospital during the study period.

Clinical signs—The most common clinical sign was coughing, which was identified in 23 of the 27 dogs (85%). Coughing was the primary initial complaint in 17 dogs, and cough and respiratory distress were the primary initial complaints in 1 dog. In addition, coughing was reported in 5 dogs that were examined with primary problems unrelated to the respiratory tract. Two dogs were examined because of respiratory distress alone. The remaining 2 dogs did not have any history of respiratory tract disease. One of these dogs was examined because of a rectal mass, and thoracic radiographs were obtained to identify metastases. The bronchiectasis was considered moderate and involved 2 lung lobes. The other dog was brought to the teaching hospital for surgical removal of cystic calculi. Crackles were ausculted on physical examination, and thoracic radiographs were obtained prior to anesthesia. Bronchiectasis in this dog was classified as generalized and severe. The duration of respiratory tract signs (cough or respiratory distress) from the time of onset to the time of diagnosis of bronchiectasis ranged from < 1 day to 10 years (mean, 18 months; median, 9 months).

Radiographic abnormalities—Of the 27 dogs, 19 (70%) had cylindrical bronchiectasis **(Fig 1)**, and 8 (30%) had saccular bronchiectasis **(Fig 2)**. A single lobe was involved in 3 (11%) dogs, 2 or 3 lobes were involved in 8 (30%) dogs, and > 3 lobes were involved in 16 (59%) dogs. The right cranial lobe was affected in 25 (93%) dogs, including 1 dog with single lobe involvement and all dogs with multiple lobe or generalized involvement.

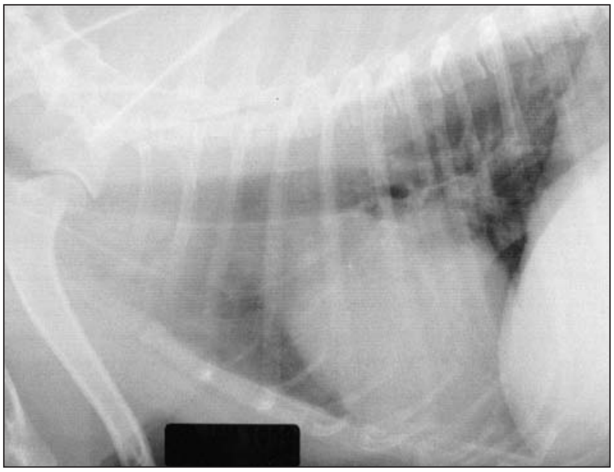

Figure 1—Lateral radiographic view of the thorax of a 13-year-old dog with cylindrical bronchiectasis. Notice that the right cranial lung lobe bronchus is abnormally dilated and does not taper in the periphery. There is also dilatation of the right middle bronchus and a caudal lung lobe bronchus centrally and an ill-defined interstitial lung opacity surrounding the abnormal bronchi.

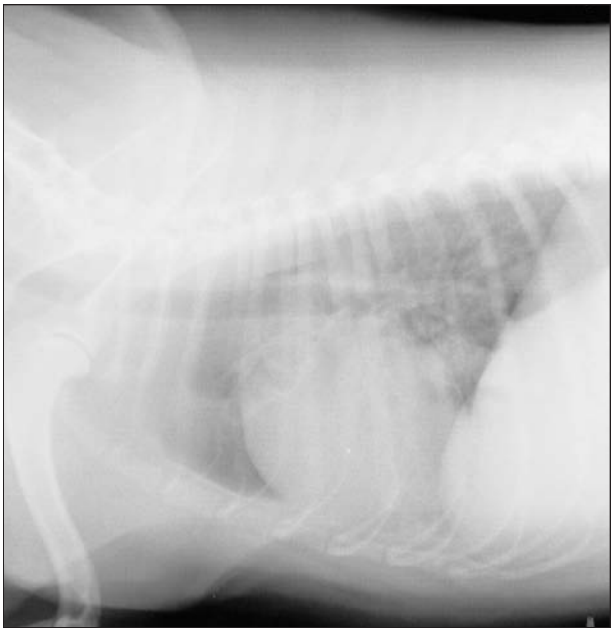

Figure 2—Lateral radiographic view of the thorax of a 2-year-old dog with saccular bronchiectasis. The left cranial lung lobe bronchus has a saccular dilatation and does not taper in the periphery. There is marked thickening of the bronchial wall consistent with chronic inflammatory changes.

Bronchiectasis was considered to be mild in 6 (22%) dogs, moderate in 16 (59%) dogs, and severe in 5 (19%) dogs.

Twenty (74%) dogs had abnormal pulmonary parenchymal patterns at the time of initial diagnosis of bronchiectasis. No bronchial abnormalities other than bronchiectasis were identified. Interstitial changes were identified in 13 dogs, and alveolar changes were identified in 7. Interstitial changes were classified as mild in 7 dogs and moderate in 6. Alveolar changes were classified as moderate in 3 dogs and severe in 4. Tracheal narrowing was identified in 4 (15%) dogs. The narrowing was at the thoracic inlet in 2 dogs, in the middle of the intrathoracic portion of the trachea in 1 dog, and at the carina in 1 dog. Mild tracheal dilatation was identified in 2 (7%) dogs; dilatation was located at the thoracic inlet in 1 dog and in the middle of the intrathoracic portion of the trachea in the other. Pleural fibrosis was identified in 20 (74%) dogs. Abnormalities of the cardiac silhouette were not seen.

Complete sets of radiographs taken ≥ 1 month after the initial diagnosis of bronchiectasis were available for 14 dogs. Time between initial radiographs and the most recent radiographs ranged from 7 weeks to 5 years (mean, 18 months; median, 17 months). The type of bronchiectasis changed to saccular in 4 of 9 dogs that initially had cylindrical bronchiectasis and remained as saccular in the other 5 dogs. The extent of bronchiectasis increased to generalized in 1 dog that initially had single lobe involvement and in 2 of 5 dogs that initially had multiple lobe involvement. The extent of bronchiectasis decreased to multiple lobe involvement (3 lobes) in 1 of 8 dogs that initially had generalized disease. The severity of bronchiectasis increased in 3 dogs, decreased in 2 dogs, and stayed the same in 9 dogs.

Seven of the 14 dogs had no evidence of disease progression in regard to any variable evaluated (ie, change from cylindrical to saccular, increase in the number of affected lobes, or increase in the severity of disease). The interval between radiographs for dogs without any evidence of disease progression (mean, 10 months; median, 11 months; range, 2 to 19 months) was shorter than, although not significantly ($P = 0.07$) different from, the interval between radiographs for dogs with evidence of disease progression (mean, 25 months; median, 23 months; range, 2 to 61 months).

Fluoroscopic abnormalities—Ten of the 27 dogs were evaluated fluoroscopically, and 9 of the 10 had evidence of collapse of the trachea or mainstem bronchi. Three of the dogs evaluated were Miniature Poodles (2 with airway collapse), 2 were American Cocker Spaniels, 2 were Toy Poodles, 1 was a Lhasa Apso, 1 was a German Shepherd Dog, and 1 was a Shih Tzu. Mainstem bronchi collapse alone was seen in 4 dogs, intrathoracic tracheal collapse alone was seen in 2 dogs, collapse of the mainstem bronchi and intrathoracic portion of the trachea was seen in 2 dogs, and collapse of the mainstem bronchi and extrathoracic portion of the trachea was seen in 1 dog.

Results of cytologic examination and culture of airway fluid samples—Tracheal wash or bronchoalveolar lavage fluid samples were obtained from 18 of the

27 dogs, with multiple samples collected from 7 of the 18. In all dogs, the sample collected most closely in time to the initial diagnosis of bronchiectasis was a tracheal wash sample. Some information about antimicrobial administration prior to collection of these tracheal wash samples was available for 14 of the 18 dogs. Eight dogs were reported to have been receiving antimicrobials within 1 week prior to collection of the tracheal wash sample, and 5 dogs had been receiving antimicrobials prior to collection of the sample, but information on when administration was discontinued was not available.

Various clinical pathologists performed cytologic examinations of the airway fluid samples without any uniform format for cytologic interpretations. Tracheal wash samples from 17 of the 18 dogs were considered to be of sufficient quality for cytologic examination. Neutrophilic inflammation was identified in all 17 samples. Neutrophils had abnormal morphology (eg, degenerative changes, toxic changes, and karyolysis) in 9 samples, and bacteria were seen in 7 of these 9 samples. Excessive numbers of eosinophils were identified in 10 samples, and in 2 of these samples, the number of eosinophils was similar to the number of neutrophils. Macrophages were seen in 10 samples, and in 2 of these samples, the number of macrophages was similar to the number of neutrophils. All 3 cells types were seen in 9 samples. Nine samples contained mucus.

Aerobic bacterial culture yielded bacterial growth for 17 of the 18 tracheal wash samples. Eleven samples had light or moderate growth of at least 1 organism. In the remaining 6 samples, bacterial growth was obtained only after incubation in enrichment medium. A single organism was isolated from 7 samples, and ≥ 2 organisms were isolated from 10. Light or moderate growth of the following organisms was obtained: α-hemolytic *Streptococcus* spp (n = 1), group D *Streptococcus* spp (3), group G *Streptococcus* spp (1), *Enterococcus faecium* (2), *E fecalis* (1), *Staphylococcus hemolyticus* (1), *S intermedius* (1), *S simulans* (1), *Escherichia coli* (2), *Pseudomonas aeruginosa* (2), *Pasteurella multocida* (1), *Pasteurella-Actinobacillus* spp (1), *Klebsiella pneumonia* (1), *Lactobacillus* spp (1), and *Micrococcus* spp (1). Mycoplasma cultures performed on 6 samples did not yield any growth.

Results of culture of all tracheal wash and bronchoalveolar lavage samples were reviewed for any reported growth of *Bordetella* spp, *Mycoplasma* spp, or *Pseudomonas* spp. *Bordetella* spp were not isolated from any sample, even following incubation in enrichment broth. In 1 dog, *Mycoplasma* spp were not isolated during culture of an initial tracheal wash sample, but were isolated from bronchoalveolar lavage fluid collected 6 months later. *Pseudomonas* spp were isolated from 11 samples from 3 dogs. Light growth was obtained from 6 samples; for the remaining 5 samples, growth was obtained only after incubation in enrichment broth. *Pseudomonas aeruginosa* was isolated from 8 of the 11 samples. *Pseudomonas cepacia* (*Burkholderia cepacia*) and *P diminuta* (*Brevundimonas diminuta*) were each isolated once from 2 samples from the same dog.

Bronchoscopic findings—Written reports of bronchoscopic findings were available in the medical records of 6 dogs. All 6 dogs were considered to have generalized airway inflammation on the basis of excessive secretions, hyperemia, edema, or mucosal fragility. Bronchiectatic airways were identified in 5 dogs. In 4 dogs, there were large volumes of exudate or inspissated material throughout multiple lobes with occlusion of 1 or more lobar bronchi. The exudates pooled in dependent airways. In another dog, excessive mucus was seen. Collapse of mainstem or lobar bronchi was seen in 4 dogs; 2 of these dogs had also had fluoroscopic evidence of intrathoracic airway collapse, while the other 2 dogs did not have fluoroscopy performed.

Concurrent diseases—Only 1 dog underwent specific testing for ciliary dyskinesia. Electron microscopic examination of tissue collected at the time of death from a 4-month-old Labrador Retriever failed to confirm a diagnosis of congenital ciliary dyskinesia. No dogs underwent specific testing of immune function.

Twenty-four of the 27 dogs had problems involving 1 or more organ systems besides the respiratory system. Otitis externa was identified in 7 of the 27 (26%) dogs, 4 of which were American Cocker Spaniels. Dermatitis was identified in 3 (11%) dogs, 2 of which were Toy Poodles. Hyperadrenocorticism was diagnosed in 3 dogs and suspected in 1. Two of these dogs were Toy Poodles, and 2 were Miniature Poodles.

Survival data—The date of death was available for 17 of the 27 dogs with bronchiectasis. Survival time ranged from 2 days to 72 months (mean, 21 months; median, 16 months). Three of the 17 dogs died within 10 days after bronchiectasis was diagnosed. Twelve dogs survived > 1 year, with 3 of those dogs surviving > 3 years. Ten of the 17 dogs died or were euthanatized for reasons other than respiratory tract disease, although chronic disease could have entered into the decision.

Case numbers were insufficient for statistical analysis of prognostic indicators, but there were no obvious associations. Of the 3 dogs that died within 10 days after bronchiectasis was diagnosed, 2 had mild, cylindrical bronchiectasis with multiple lobe involvement in 1 and generalized disease in the other, and 1 had generalized, moderate, saccular bronchiectasis. Of the 3 dogs that survived > 3 years, 1 had severe, cylindrical bronchiectasis with generalized involvement that changed to moderate, saccular bronchiectasis involving 3 lobes; 1 had moderate, cylindrical bronchiectasis involving 2 lobes that progressed to moderate, saccular bronchiectasis with generalized involvement; and 1 had moderate, saccular bronchiectasis involving 1 lobe that progressed to severe, saccular bronchiectasis with generalized involvement. The latter dog had surgery to remove the initial affected lobe 2 months after initial diagnosis (70 months prior to death). Subsequently, bronchiectasis appeared in the remaining lobes. In the 2 dogs that survived the longest, *P aeruginosa* was isolated from initial airway fluid samples, and *Pseudomonas* spp were subsequently isolated from follow-up samples. The success of various treatments in prolonging survival time could not be assessed because accurate records of treatment were not available.

Discussion

Bronchiectasis is a result of chronic airway inflammation leading to destruction of the airway walls and involves complex interactions among microorganisms, inflammatory cells, and inflammatory cell products.[1,17-20] The normal structure and function of the airways are compromised, impairing mucociliary clearance. The resulting retention of secretions and decreased ability to eliminate infections perpetuates the inflammatory response. Because alterations in airway defenses have occurred by the time of diagnosis, it is difficult to determine whether infection identified at the time of diagnosis was an initiating cause or a consequence of the disease, and the inciting cause of inflammation may not always be infection.[21] Furthermore, bronchiectasis is not a universal consequence of chronic airway inflammation, and preexisting abnormalities in pulmonary clearance mechanisms or immunologic and inflammatory host responses are important.[1,17,19,20]

Dogs were included in the present study only if they had clearly identifiable radiographic evidence of bronchiectasis. However, studies[3,4] in people have found the sensitivity of thoracic radiography in the diagnosis of bronchiectasis to be as low as 37% to 47% in some instances. Therefore, it is likely that all dogs in this study had advanced disease and that bronchiectasis is more common in dogs than our data suggest.

Thoracic radiographs from the dogs in this study were reviewed by a single radiologist to obtain consistent interpretations. Cylindrical bronchiectasis and generalized bronchial involvement were the most common radiographic abnormalities. These findings are in agreement with findings in a previous study[2] of 14 dogs with bronchiectasis. Little clinical difference has been found between types of bronchiectasis in people,[1] although saccular bronchiectasis is generally thought to represent more advanced disease.[5] The progression of lesions over time in some dogs in the present study from cylindrical to saccular, but never from saccular to cylindrical, would support this suggestion.

The right cranial lung lobe was involved in 25 of the 27 (93%) dogs in the present study. Ten dogs with bronchiectasis described by Brownlie[6] in a report of 109 dogs with lower respiratory tract disease had involvement of the cranial and middle lobes. Bronchiectasis has been described as a cyclical process involving decreased airway clearance, microbial colonization, and inflammation.[17] The gravity-dependent location of these commonly involved lung lobes may have further contributed to decreased clearance of secretions and perpetuation of the cyclical process.

Most dogs with bronchiectasis in this study had a history of cough. Productive cough is a common clinical sign in people with bronchiectasis, but it is difficult to accurately classify coughing as productive or nonproductive in dogs. The excessive mucus in some tracheal wash samples and evident during bronchoscopy was consistent with the hypersecretion of mucus and pooling of secretions, which is typical of the disease in people. Hemoptysis or blood-tinged sputum is seen in approximately 50% of people with bronchiectasis,[22] but hemoptysis, cytologic evidence of hemorrhage in airway fluid samples, or bronchoscopically apparent hemorrhage were not identified in any of the dogs in this study. Bronchiectasis was not identified as a cause of hemoptysis in a recent review of 36 dogs with this sign.[23]

Chronic cough can also be a sign of chronic bronchitis, and the relationship between bronchiectasis and chronic bronchitis is difficult to define. In human medicine, chronic bronchitis and bronchiectasis are both classified as obstructive lung diseases, and although they are considered to be distinct clinical entities,[5,17,22,24-26] there is some overlap between these conditions in people. In dogs, chronic bronchitis is defined as coughing for 2 months in the absence of other disease,[27,28] but the cause of chronic bronchitis in dogs has not been established. Chronic bronchitis has been proposed to be a cause of bronchiectasis in dogs and cats[2,28-31]; however, a cause-and-effect relationship has not been proven. Dogs with chronic coughing secondary to early bronchiectasis may not have radiographically evident abnormalities and may be considered, rightly or wrongly, to have chronic bronchitis.

Focal bronchiectasis can result from endobronchial obstruction.[20] A single lung lobe was involved in only 3 of 27 (11%) dogs in the present study. None of these dogs had any evidence of airway obstruction, and no obstruction was found in the 1 dog that underwent lobectomy. This dog subsequently developed generalized bronchiectasis. It is possible that some dogs with airway obstruction and focal bronchiectasis were not included in the present study because medical record codes included the primary diagnosis, such as lung tumor or bronchial foreign body, but did not include bronchiectasis. No focal or obstructive disease was found in a previous study[2] of dogs with bronchiectasis.

None of the dogs in this study had obvious congenital disease, but testing for such conditions was performed in only 1 dog. Ciliary dyskinesia has been associated with bronchiectasis in some dogs,[8-13] but was not identified in the 1 dog in this study in which samples were submitted for electron microscopy. Nineteen of 27 (70%) dogs in this study were > 10 years old at the time bronchiectasis was diagnosed, making congenital disease unlikely.

The association found between bronchiectasis and certain breeds in the present study could be a result of inherited disorders. American Cocker Spaniels, West Highland White Terriers, Miniature Poodles, Siberian Huskies, and English Springer Spaniels were overrepresented, compared with dogs of other breeds. American Cocker Spaniels are at increased risk for a variety of inflammatory and immune-mediated diseases, including atopy,[32] immune-mediated hemolytic anemia,[33] and hypothyroidism.[34] West Highland White Terriers appear to be predisposed to pulmonary fibrosis,[35] but radiographs of 29 West Highland White Terriers with chronic pulmonary disease did not show any evidence of bronchiectasis. Bronchiectasis was reported in 3 dogs that had undergone radiation therapy 3 to 7 months previously and was thought to possibly be a result of fibrosis.[36] Siberian Huskies have been associated with eosinophilic respiratory disease.[37] For dogs in the present study that were identified through a search of the VMDB records, the diagnosis of bronchiectasis was not confirmed by review of radiographs. However, the statistical associations with specific breeds were quite strong, and there was no obvious reason to suspect a bias toward overdiagnosis among dogs of a specific breed.

In people, cartilage deficiency, which can cause regions of the bronchi to dilate and narrow with inspiration and expiration, has been associated with bronchiectasis.[1] Such dynamic motion of the airways is seen in dogs with tracheal collapse and in some dogs with chronic bronchitis. In the present study, 9 of 10 dogs that were evaluated fluoroscopically had evidence of intrathoracic airway collapse, and 2 additional dogs had bronchoscopic evidence of airway collapse. Although cartilage defects have been identified in some dogs with tracheal collapse,[38,39] the frequent occurrence of intrathoracic airway collapse in dogs with chronic bronchitis makes it likely that airway collapse was a consequence of weakened airway walls resulting from bronchiectasis, rather than a cause. Even so, it may be possible that airway collapse contributes to the cycle of continued inflammation, obstruction, and destruction of the airways.

Infections with certain bacterial, viral, and fungal agents are thought to predispose individuals to bronchiectasis. Bacteria frequently isolated from people with bronchiectasis include *Staphylococcus aureus*, *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Moraxella catarrhalis*, and *Pseudomonas* spp.[1,5,17,25,40-42] However, many different types of organisms have been isolated from patients with bronchiectasis, including *Mycoplasma pneumoniae*,[22,43] and in dogs, infection with *Mycoplasma* spp has been associated with bronchopneumonia and bronchiectasis.[44]

Unfortunately, conclusions that can be drawn regarding results of bacterial culture of airway fluid samples in the present study are limited because of the high proportion of dogs that had been treated with antimicrobials prior to collection of airway fluid samples. Nevertheless, bacteria were isolated from tracheal wash samples from 17 of 18 dogs. A variety of gram-negative and gram-positive organisms were isolated, and growth of > 1 organism was common. Bacterial growth was likely to be clinically important in most of these dogs because despite recent antimicrobial administration in some dogs, there was cytologic evidence of neutrophilic inflammation and degenerative neutrophils and bacteria could be isolated without incubation in enrichment broth in many of these cases.

Results of cultures of all airway fluid samples from dogs in the present study were scrutinized for evidence of infection with *Mycoplasma* spp, *Bordetella* spp, or *Pseudomonas* spp. *Mycoplasma* spp were isolated from only 1 dog, and *Bordetella* spp were not isolated from any dogs, suggesting that these organisms do not have an important role in the pathogenesis of bronchiectasis in dogs. However, the potential contribution of earlier infection cannot be discounted given the long delay between prior pulmonary events and the onset of clinical signs of bronchiectasis in people.[22]

Pseudomonas organisms were isolated from 11 airway samples from 3 dogs in the present study, with 8 of the isolates identified as *P aeruginosa*. The number of dogs with *Pseudomonas* infection in the current study was insufficient to allow for any analysis for an association with prognosis. However, 2 of the dogs from which *P aeruginosa* was isolated had the longest and third longest survival times (72 and 47 months), and the third was still alive > 1 year after bronchiectasis was diagnosed. The dog that survived for 72 months also had *B cepacia* recovered from a tracheal wash sample > 2 years prior to death.

Allergic airway disease has been associated with bronchiectasis. In people, for instance, a strong association is seen with allergic bronchopulmonary aspergillosis, and it appears that some patients with asthma develop bronchiectasis.[1,18,19,45,46] Although dogs have not been reported to develop allergic bronchopulmonary aspergillosis, there have been reports[37,47] of bronchiectasis in dogs with eosinophilic lung disease. In the present study, 10 of 17 dogs had eosinophils in airway fluid samples, and in 2, the number of eosinophils was similar to the number of neutrophils.

Dogs with bronchiectasis can be considered to have a fair or good prognosis. Signs of respiratory tract disease were present for 10 years prior to the diagnosis of bronchiectasis in 1 dog in this study, and median duration of clinical signs prior to diagnosis was 9 months. Seven of the 14 dogs for which follow-up radiographs were available did not have any radiographic evidence of worsening of bronchiectasis over time. Median survival time for dogs in this study was 16 months, with 3 dogs surviving > 3 years. This is particularly important given that the median age at the time of diagnosis was 12 years. People with bronchiectasis are generally expected to do well if they comply with treatment regimens and practice routine preventative strategies.[25] Similarly, the prognosis for dogs with bronchiectasis likely depends to some extent on the ability of owners to seek out and carry through with appropriate veterinary care, particularly during exacerbations of the disease.

[a]Veterinary Medical Database, School of Veterinary Medicine, Purdue University, West Lafayette, Ind.
[b]StatsDirect, version 1.9.8, StatsDirect Ltd, Herts, UK.
[c]Sigma Stat for Windows, version 2.03, SPSS Inc, Chicago, Ill.

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