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Objective—To determine clinical signs and rhinoscopic, computed tomographic, and histologic abnormalities in dogs with idiopathic lymphoplasmacytic rhinitis.

Design—Retrospective case series.

Animals—37 dogs.

Procedure—Clinical information was obtained from medical records. Nasal computed tomographic images and histologic slides of biopsy specimens were reviewed.

Results—Dogs ranged from 1.5 to 14 years old (mean, 8 years); most (28) were large-breed dogs. Nasal discharge was unilateral in 11 of 26 (42%) dogs and bilateral in 15 of 26 (58%) dogs. In dogs with unilateral disease, duration of clinical signs ranged from 1.5 to 36 months (mean, 8.25 months; median, 2 months), and in dogs with bilateral disease, duration of signs ranged from 1.25 to 30 months (mean, 6.5 months; median, 4 months). Computed tomography (n = 33) most often revealed fluid accumulation (27/33 [82%]), turbinate destruction (23/33 [70%]), and frontal sinus opacification (14/33 [42%]). Rhinoscopy (n = 37) commonly demonstrated increased mucus and epithelial inflammation; turbinate destruction was detected in 8 of 37 (22%) dogs. Bilateral biopsy specimens from all 37 dogs were examined. Four dogs had only unilateral inflammatory changes. The remaining 33 dogs had bilateral lesions; in 20, lesions were more severe on 1 side than the other.

Conclusions and Clinical Relevance—Findings suggest that idiopathic lymphoplasmacytic rhinitis is a key contributor to chronic nasal disease in dogs and may be more common than previously believed. In addition, findings suggest that idiopathic lymphoplasmacytic rhinitis is most often a bilateral disease, even among dogs with unilateral nasal discharge. (J Am Vet Med Assoc 2004; 224:1952–1957)

Chronic nasal discharge is a common clinical complaint in dogs. Treatment is most effective when the etiopathogenesis is known; however, the underlying cause of rhinitis is not always apparent. The most common causes of chronic nasal discharge in dogs include neoplasia, aspergillosis, nasal foreign body, rhinitis secondary to dental disease, and idiopathic lymphoplasmacytic rhinitis (LPR; sometimes referred to as immune-mediated or chronic inflammatory rhinitis). Several studies have identified neoplasia and aspergillosis as the 2 most common causes of chronic nasal discharge in dogs. Both of these disorders typically result in unilateral nasal discharge or unilateral nasal discharge that progresses to bilateral nasal discharge over time. Similarly, rhinitis related to dental disease or a nasal foreign body typically is associated with unilateral nasal discharge. In contrast, rhinitis related to infection, chronic inflammation, allergy, or immune stimulation would be expected to result in bilateral nasal discharge.

To our knowledge, only 2 previous reports describe clinical signs, results of diagnostic testing, and outcome in dogs with idiopathic LPR, and those reports involved only 5 dogs each. Idiopathic LPR has been referred to as immune-mediated and allergic rhinitis, but specific characteristics of the disease have not been fully described. Specifically, the duration and progression of clinical signs have not been addressed, and imaging abnormalities have not been reported. More extensive study of idiopathic LPR is needed to improve the recognition and diagnosis of this disorder.

The purpose of the study reported here, therefore, was to determine clinical signs, effectiveness of past treatment, rhinoscopic findings, computed tomographic abnormalities, and histologic abnormalities in dogs with idiopathic LPR. We hypothesized that dogs with unilateral nasal discharge would have a shorter duration of clinical signs and less severe tomographic and histologic abnormalities than dogs with bilateral nasal discharge, indicating that dogs with unilateral nasal discharge were examined earlier in the course of disease.

Criteria for Selection of Cases

Medical records of dogs examined at the University of California, Davis, Veterinary Medical Teaching Hospital between 1997 and 2002 were searched to identify all dogs examined because of chronic nasal discharge. Dogs in which a histologic diagnosis of LPR had been made and no underlying cause of the lesions (eg, aspergillosis, neoplasia, foreign body, or oronasal fistula) could be identified were eligible for inclusion in the study. Dogs were included in the study only if biopsy specimens had been obtained from both nasal passages.

Procedures

Medical records were examined, and information was recorded on history, signalment, response to previous treatment, results of clinicopathologic testing, com-
puted tomographic findings, and rhinoscopic abnormalities. Information on the response to treatment before and after a diagnosis of idiopathic LPR had been made was collected when available. Diagnostic testing to identify the underlying cause of the nasal disease was performed at the discretion of the admitting clinician, but a minimum database was collected for all dogs.

Dogs were anesthetized for computed tomography of the nasal cavity, and computed tomography was performed with a standard or helical high-speed scanner. For purposes of the present study, tomographic images were reviewed by a board-certified radiologist (EJH) for lateralization of abnormalities and focal or diffuse involvement of the nasal cavity, frontal sinus, and cribiform plate. Specific tomographic findings related to fluid density, gas pocketing, turbinate destruction, soft tissue opacification, frontal sinus involvement, and plaque-like lesions were characterized as absent, mild, moderate, or severe.

Rhinoscopy was performed with a 5° rigid endoscope. For the present study, all endoscopic findings were reviewed. Abnormalities recorded during examination included mucosal hyperemia or edema, mucus accumulation, and turbinate destruction. Biopsy specimens were obtained from both sides of the nasal cavity with 2- or 4-mm cup biopsy instruments. One to 12 (mean, 6) biopsy specimens were obtained from each side of the nasal cavity and placed in a single cassette for analysis. Biopsy specimens were immersion-fixed in neutral-buffered 10% formalin, routinely processed, embedded in paraffin, and sectioned at a thickness of 4 μm. Sections of all specimens were stained with H&E; in addition, sections from selected specimens were stained with trichrome, periodic acid–Schiff, Grocott methanol silver, or Brown and Brennam stain for detection of microorganisms. For the present study, histologic slides of nasal biopsy specimens were reviewed by a board-certified pathologist (HEVDC); each side of the nasal cavity was individually scored. Overall severity of inflammation was classified as mild, moderate, or severe on the basis of the relative extent of inflammation for all dogs, compared with the normal nasal histologic appearance. Turbinates lesions were characterized as mild, moderate, or severe depending on the degree of bone remodeling and destruction. Additional features described included whether there was evidence of epithelial hyperplasia, erosions, adherent mucus, or vasculitis with or without hyaline degeneration of the vascular wall.

Statistical analyses—One-way ANOVA was used to compare duration of clinical signs and histologic severity of inflammation between dogs with unilateral versus bilateral nasal discharge. Values of \( P < 0.05 \) were considered significant. Computed tomographic abnormalities, rhinoscopic findings, and histologic lesions were reported as unilateral or bilateral, and the level of agreement between clinical signs (ie, unilateral vs bilateral nasal discharge) and computed tomographic, rhinoscopic, and histologic findings was assessed by calculating the \( \kappa \) statistic. The \( \kappa \) statistic assesses the level of agreement between clinical signs and diagnostic test results beyond that expected on the basis of chance alone. A \( \kappa \) of 0 indicates no agreement beyond that expected on the basis of chance alone, while a \( \kappa \) of 1 indicates perfect agreement.

Results

Records of 55 dogs in which a histologic diagnosis of LPR had been made during the study period were identified. However, 15 of these dogs were excluded from the study because an underlying cause was identified (aspergillosis, 8 dogs; neoplasia, 4; intraluminal foreign material, 2; and oronasal fistula repair, 1). In the remaining 40 dogs, no specific cause for inflammatory nasal infiltrates was identified, and a diagnosis of idiopathic LPR had been made. However, 3 of these dogs were excluded from the study because bilateral nasal biopsy specimens had not been obtained. The remaining 37 dogs were included in the study.

The 37 dogs included in the study ranged from 1.5 to 14 years old (mean, 8 years; median, 8.5 years) at the time of examination. There were 21 spayed females, 11 castrated males, and 5 sexually intact males. Body weight ranged from 1.3 to 60 kg (2.9 to 132 lb; mean, 27.3 kg [60.1 lb]; median, 28.4 kg [62.5 lb]). Twenty-eight of the 37 (76%) dogs were classified as large-breed dogs. Twenty-two (59%) dogs were purebred, but German Shepherd Dog (n = 6) was the only breed represented by > 1 dog. Thus, 6 of the 22 (27%) purebred dogs were German Shepherd Dogs, whereas only 4% of all dogs examined at the veterinary teaching hospital annually were German Shepherd Dogs. Five of the 15 dogs that were not purebred were classified as Labrador Retriever mixes and 3 were classified as German Shepherd Dog mixes. Thirty-five dogs had mesaticephalic skulls, 1 had a dolichocephalic skull, and 1 had a brachycephalic skull.

Nasal discharge was the most common clinical complaint and was reported in 35 of the 37 (95%) dogs. The discharge was described as mucoid or mucopurulent in 25 of the 35 (71%) dogs, hemorrhagic or blood-tinged in 6 (17%), and serous in 4 (11%). Whether the nasal discharge was unilateral or bilateral was not recorded for 9 dogs. Eleven of the remaining 26 (42%) dogs were reported to have unilateral nasal discharge, and 15 (58%) were reported to have bilateral nasal discharge. Duration of clinical signs prior to examination at the veterinary teaching hospital ranged from 6 weeks to 36 months (mean, 8.25 months; median, 2 months) for the 11 dogs with unilateral nasal discharge and from 5 weeks to 30 months (mean, 6.5 months; median, 4 months) for the 15 dogs with bilateral nasal discharge. Duration of clinical signs was not significantly different between groups. Other common clinical complaints included sneezing (19/37 [51%]), coughing (15/37 [41%]), epistaxis (15/37 [41%]), reverse sneezing (6/37 [16%]), stertorous breathing (5/37 [14%]), ocular discharge (4/37 [11%]), and pawing or rubbing at the muzzle (2/37 [5%]).

Information on previous treatment was available for 32 dogs, although efficacy of this treatment was not recorded for all 32. Thirty of the 32 (94%) dogs had been treated with antimicrobials, including 14 (44%) that were treated with antimicrobials alone. Enrofloxacin was the most commonly used antimicrobial (16/30 [53%]), followed by amoxicillin-clavulanic acid (8/30 [27%]). Twelve dogs were treated with multiple antimicrobials. Effectiveness of treatment was reported for 21 of the 30 dogs treated with antimicrobials alone or in combination with other drugs. Ten of the 21 (48%) reportedly did not have any improve-
ment, 2 (10%) had slight improvement, 4 (19%) had some improvement with a return of clinical signs after antimicrobial administration was discontinued, and 5 (24%) had initial improvement followed by a return of clinical signs while antimicrobials were still being administered.

Antihistamines had been administered to 10 of the 32 (31%) dogs, with diphenhydramine administered most commonly (9/10 dogs). Effectiveness of treatment had been reported for 7 dogs treated with antihistamines alone or in combination with other drugs, and 3 reportedly did not have any improvement, 1 had slight improvement, and 1 had some initial improvement followed by a return of clinical signs while antihistamines were still being administered.

Glucocorticoids had been administered in 10 of the 32 (31%) dogs, and clinical efficacy was reported for 5. Three had had no reduction in clinical signs, and 2 had had slight improvement. Itraconazole had been administered in combination with antimicrobials in 1 dog, and clinical signs improved initially but then returned.

On initial physical examination at the veterinary teaching hospital, nasal discharge was apparent in 19 dogs, and dry discharge or crusting around the nares was evident in 9. Decreased nasal airflow was mentioned in the records of 3 dogs. Lung sounds were increased in 2 dogs and decreased in 3. Three dogs had referred upper airway noises on thoracic auscultation. Lymphadenopathy was identified in 9 dogs; 6 dogs had mandibular lymph node enlargement, 1 had superficial cervical lymph node enlargement, 1 had superficial cervical and mandibular lymph node enlargement, and 1 had mandibular, superficial cervical, and popliteal lymph node enlargement. Lymph node aspirates were obtained from 4 of the 9 dogs with peripheral lymphadenopathy. Results of cytologic examination of the aspirates were unremarkable for 1 dog and compatible with moderate reactive lymphoid hyperplasia for the other 3. Fever (rectal temperature \( \geq 39.4^\circ\text{C} \) \( [103^\circ\text{F}] \)) was documented on admission in 6 dogs (range, 39.4° to 40°C [103° to 104°F]). Four dogs coughed during the examination, and 3 dogs sneezed. Stertor, audible nasal congestion, and increased respiratory effort were each noted in 1 dog.

Computed tomography was performed in all 37 dogs; however, tomographic studies for only 33 dogs were available for review. In 4 dogs, results of computed tomography were considered unremarkable, whereas in 29 dogs, tomographic abnormalities were seen (Fig 1).

Eight dogs had unilateral tomographic lesions, and 21 dogs had bilateral lesions. In 12 of the 21 dogs with bilateral lesions, the lesions were more severe on 1 side than on the other. Fluid accumulation was evident in 27 of the 33 (82%) dogs and graded as mild in 7, moderate in 13, and severe in 5. Soft tissue opacification was evident in 25 of the 33 (78%) dogs and graded as mild in 10, moderate in 12, and severe in 3 dogs. Plaque-like lesions were identified in 24 of the 33 (73%) dogs and graded as mild in 13, moderate in 9, and severe in 2. Turbinate destruction was detected in 23 of 33 (70%) dogs and graded as mild in 15, moderate in 6, and severe in 2. Gas pocketing was found in 23 of the 33 (70%) dogs and graded as mild in 9, moderate in 11, and severe in 3. Frontal sinus involvement was observed in 14 of the 33 (42%) dogs and graded as mild in 9, moderate in 2, and severe in 3. No dogs had tomographic evidence of cribriform plate involvement. Localization of lesions could be determined in 27 dogs. Lesions were diffuse in 16 (59%) dogs, rostral in 8

![Figure 1](image-url)
Nasal samples from 15 dogs were submitted for microbial culture. Samples from 5 dogs did not yield any growth. Samples from 9 dogs yielded a growth of bacteria. A single bacterial organism was isolated from 5 of these 9 samples, and 2 or more bacterial organisms were isolated from the remaining 4. The most common bacterial species isolated included Staphylococcus (3), and Corynebacterium (2). Fungal growth was obtained from nasal samples from 2 dogs, 1 of which also had bacterial growth. For 1 of these dogs, 2 colonies of Candida parapsilosis and 1 colony of Trichosporon asahii were obtained. For the other, a single colony of Cladosporium spp was isolated, along with a growth of bacteria.

Histologic sections from 70 nasal biopsy specimens obtained from the 37 dogs had evidence of chronic lymphoplasmacytic infiltration of the mucosa. Superimposition of neutrophilic infiltration was evident in 31 dogs, with neutrophils sequestered in mucus adherent to the epithelial layer or in subepithelial abscesses or pustules. Six biopsy specimens from 5 dogs contained hemosiderin, while 5 biopsy specimens from 4 dogs contained cosinophils. Evidence of hyalinization of the vascular wall sometimes in association with vasculitis was present in 17 biopsy specimens from 10 dogs. Special stains failed to reveal microorganisms in any of the sections examined.

Of the 74 biopsy specimens examined, all but 4 had evidence of mucosal inflammation. The overall severity of inflammation was graded as mild in 40 (54%), moderate in 22 (29%), and severe in 8 (11%). Epithelial changes were mild in most specimens, with hyperplasia identified in only 8 of the 74 (11%) biopsy specimens and epithelial erosions identified in 3 (4%). Turbinate structures could be assessed in 68 of the 74 biopsy specimens. Mild turbinate remodeling was identified in 17 of these 68 (25%) specimens, mild turbinate destruction was identified in 12 (18%), moderate turbinate destruction was identified in 1 (1.5%), and severe turbinate destruction was identified in 5 (7%).

In 4 of the 37 dogs, histologic evidence of inflammation was strictly unilateral with mild inflammation in 1 side of the nasal cavity and no histologic lesions in the contralateral side. In the other 33 dogs, bilateral histologic lesions were seen. In 13 dogs, inflammatory lesions were judged to be of equivalent severity in the left and right sides of the nasal cavity, with 9 dogs having mild inflammation and 4 having moderate inflammation. In the other 20 dogs, histologic lesions were more severe in 1 side of the nasal cavity than in the other. Six dogs had severe inflammation in 1 side of the nasal cavity and mild inflammation in the other, 12 had moderate inflammation in 1 side of the nasal cavity and mild inflammation in the other, and 2 dogs had severe inflammation in 1 side of the nasal cavity and moderate inflammation in the other.

For 33 dogs, histologic slides containing turbinate structures from both sides of the nasal cavity were available for review. Ten dogs did not have any evidence of turbinate remodeling or destruction. Fifteen dogs had unilateral turbinate lesions, with mild bone remodeling or destruction in 13 dogs and severe bone remodeling or destruction in 2. Seven dogs had evidence of mild (n = 6) or severe (1) bilateral bone remodeling or destruction, with severity of changes in the left and right sides of the nasal cavity being similar. The remaining dog had moderate bone remodeling in 1 nasal cavity and severe bone remodeling in the other.

Whether a unilateral or bilateral nasal discharge was seen clinically did not correlate with whether tomographic (κ = 0.41), rhinoscopic (κ = 0.41), or histologic (κ = 0) lesions were unilateral or bilateral.

Concurrent diseases that were identified included pneumonia (3 dogs) and keratoconjunctivitis sicca, discoid lupus erythematosus, masticatory myositis, hypercalcemia, shifting limb lameness, CNS disease, T3-L3 myelopathy, lumbar myelopathy, urinary tract infection, and bronchitis (1 dog each).

In 21 of 37 dogs, medications were prescribed to treat the idiopathic LPR. Sixteen (76%) received antimicrobials, 12 (57%) received corticosteroids (3 by means of a nasal spray), 6 (29%) received antifungal medication (intranasal clotrimazole infusion), and 6 (29%) received antihistamines. Information on effectiveness of treatment was available for only 11 dogs. Of these, 5 reportedly had a sustained improvement in clinical signs. Three of these 5 dogs were treated with a combination of amoxicillin-clavulanic acid and glucocorticoids for 2 weeks. One of these dogs had unilateral disease rhinoscopically and histologically and sneezed out a foreign body (foxtail) 2 months after discharge. It was not known whether this was the cause of the dog's clinical signs; however, the owner reported that the dog had returned to normal after this time. The remaining 2 dogs that responded to treatment consisted of a dog treated with diphenhydramine in which clinical signs resolved within 1 month but returned if diphenhydramine administration was discontinued and a second dog treated with diphenhydramine and an intranasal corticosteroid spray for 1 week. In 4 of 6 dogs treated with intranasal administration of clotrimazole, no response to treatment was noted; treatment effectiveness was not recorded in the other 2 dogs treated with clotrimazole.

**Discussion**

Results of the present study suggest that idiopathic LPR is most often a bilateral disease, even among dogs with unilateral nasal discharge. A previous study of LPR in 5 dogs reported that dogs were between 3 and 10 years old, whereas dogs in the present study...
were between 1.5 and 14 years old, reinforcing the idea that idiopathic LPR is a disease of middle age to older dogs. German Shepherd Dogs appeared to be overrepresented in the present study, but no other breed predilections were identified. The duration of clinical signs prior to examination at the veterinary teaching hospital ranged from 1 month to 3 years, emphasizing the chronicity of this disorder and possibly helping to explain why tomographic abnormalities that were seen were so severe.

Common signs of chronic nasal disease in dogs include serous, sanguineous, mucoid, or purulent nasal discharge, sneezing, stertor, epistaxis, and ocular discharge, all of which were variably present in dogs in the present study. The mucopurulent nature of the nasal discharge in many dogs with LPR most likely reflects the chronicity of inflammation because bacterial culture of nasal samples yielded minimal or no growth in this study and the response to antimicrobial treatment historically had been minimal. Several dogs in the present study had had episodes of epistaxis, which is often associated with neoplasia, aspergillosis, nasal foreign bodies, and coagulopathies. Epistaxis may also be caused by vigorous or paroxysmal sneezing. For 5 of the 15 dogs in this study with a history of epistaxis, epistaxis was the primary or only clinical complaint, and 2 of these dogs had no evidence of sneezing. To the authors' knowledge, this is the first report to identify epistaxis as a primary complaint in dogs with idiopathic LPR, although hemorrhagic discharge has been reported previously for dogs with LPR. Fifteen of 37 (41%) dogs also had a history of coughing, which has not previously been associated with LPR. Coughing is normally associated with lower respiratory tract disease, and 3 dogs in this study had a history of pneumonia while 1 dog had a history of bronchitis. Coughing in the remaining dogs may have been a result of unrecognized lower respiratory tract disease or laryngeal irritation associated with retrograde nasal drainage.

Historically, antimicrobials, antihistamines, and glucocorticoids had not been effective in eliminating clinical signs in dogs in the present study. Although antimicrobial treatment had helped to reduce nasal discharge in some dogs, often only the character of the discharge was changed (ie, from mucopurulent to serous), and similar to findings in a previous report, the response to antimicrobials was not sustained. Antimicrobials likely reduce secondary bacterial colonization without diminishing the discharge caused by idiopathic LPR. Although 4 of 5 dogs in a previous report improved when treated with prednisone, most dogs in the present study had historically not responded to glucocorticoid treatment. The large number of dogs for which follow-up information was unavailable makes it difficult to draw any definite conclusions on the efficacy of particular treatments. However, if idiopathic LPR is immune-mediated, as some postulate, a better response to glucocorticoids would be expected. A prospective study is required to monitor treatment efficacy more accurately.

Physical examination findings in dogs in the present study were nonspecific and typical for chronic nasal disease of any cause. Nasal discharge was the most common finding on physical examination, with evidence of current discharge or dry nasal discharge in most dogs. Importantly, unilateral clinical signs were seen in some dogs with idiopathic LPR. Nine dogs also had regional lymphadenopathy, and aspirates from 3 had evidence of moderate lymph node reactivity, which may have been a result of chronic immune stimulation.

A previous study of dogs with chronic nasal disease reported radiographic findings; however, radiography has low sensitivity in differentiating inflammatory rhinitis from neoplasia or mycotic rhinitis. Computed tomography provides better definition of the extent and severity of abnormalities of the nasal cavity, although at least in cats, computed tomography cannot be used to differentiate chronic rhinitis from neoplasia. There is some discordance in the literature as to the extent of turbinate destruction and frontal sinus involvement with chronic inflammatory rhinitis. Whereas some authors have report that turbinate destruction was associated with more destructive processes such as neoplasia or aspergillosis, others have demonstrated the potential for inflammatory rhinitis to cause turbinate destruction. Turbine destruction was seen tomographically in 23 of 33 (70%) dogs in the present study and was severe in 2, demonstrating that idiopathic LPR can result in recognizable turbinate damage. Some authors have reported that inflammatory rhinitis does not involve the frontal sinuses; however, 14 of 33 (42%) dogs in this study had frontal sinus involvement, indicating that idiopathic LPR often extends beyond the nasal passages. Importantly, computed tomography revealed bilateral disease in many dogs with unilateral clinical signs, supporting the use of computed tomography for any dog with chronic nasal disease.

Current literature suggests that the distribution of radiographic lesions in dogs with LPR or chronic inflammatory disease is focused in the rostral portion of the nasal cavity; however, 16 of 27 (59%) dogs in this study had diffuse (rostral, middle, and caudal) tomographic lesions and only 8 had lesions localized to the rostral portion alone. This demonstrates that idiopathic LPR may have a diffuse distribution similar to that reported for neoplasia. Fluid accumulation and soft tissue opacification were the most common tomographic abnormalities in this study.

Rhinoscopic findings reported in previous studies of dogs with chronic inflammatory rhinitis include hyperemia, inflammation, excessive mucus, edema, and infiltration. Excessive friability of tissue was also reported and may indicate chronic damage to the nasal mucosa. Turbinate destruction was evident rhinoscopically but with lower frequency than was evident histologically or on computed tomographs. This likely was attributable, in part, to the multitude of endoscopists with various levels of experience who performed endoscopy on these dogs. In 2 studies of rhinoscopy as a diagnostic tool for chronic nasal diseases, intranasal mass lesions were associated with evidence of rhinosinusitis or inflammation. Therefore, while rhinoscopy is an important tool in the diagnosis...
of LPR through exclusion of other causes, concurrent assessment by means of computed tomography and histologic examination of biopsy specimens is required to make a diagnosis.

Forty of 74 (54%) biopsy specimens from dogs in the present study had mild inflammation, and epithelial changes such as hyperplasia, erosion, and lymphocytic exocytosis were seen in some specimens. In comparison, an early study\textsuperscript{1} reported epithelial hyperplasia, squamous metaplasia, ulceration, and various degrees of submucosal fibrosis in biopsy specimens from 3 dogs with LPR. This may reflect a different disease process in the previous 5 dogs or a different stage of disease. The presence of some degree of turbinate destruction or remodeling in 35 of 68 biopsy samples demonstrates that idiopathic LPR can cause destructive changes that can be identified microscopically if not macroscopically. Neutrophilic infiltration of the mucosa and adherent mucus identified in specimens from 31 dogs may have represented chronic inflammation and secondary overgrowth of superficial bacteria. Biopsy specimens from 4 dogs contained eosinophils, which might suggest an allergic component of disease in these dogs. The clinical significance of the vascular lesions that were seen is unclear, although they might suggest that an immunemediated process was involved.

The distribution of lesions (ie, unilateral vs bilateral) in dogs with inflammatory rhinitis has been examined in several studies\textsuperscript{3,4,14} and shown to be variable. In 1 study,\textsuperscript{2} of 5 dogs had unilateral signs that progressed to bilateral involvement. In the present study, 11 of 26 (42%) dogs had a history of unilateral nasal discharge, and 15 (58%) had bilateral nasal discharge. One dog had had unilateral nasal discharge for 36 months prior to examination, demonstrating that idiopathic LPR may at times affect a single nasal cavity without progressing to bilateral disease. On the other hand, only 4 dogs had histologic evidence of unilateral disease. This is of particular importance because nasal biopsy specimens represent such a small portion of the nasal cavity. Thus, it is likely that widespread inflammation may be present despite limited clinical signs. The 4 dogs with strictly unilateral histologic changes all had mild inflammation, while the dogs with bilateral histologic lesions generally had more severe inflammation.

Findings in the present study verify that idiopathic LPR is a key contributor to chronic nasal disease in dogs and may be more common than previously believed. While historically considered a benign disorder compared with more destructive diseases such as neoplasia or aspergillosis, this study clearly indicates that idiopathic LPR can cause severe turbinate destruction and frontal sinus involvement. Clinical signs are often typical of other chronic nasal disorders (eg, nasal discharge and sneezing) but can include epistaxis without other clinical signs and cough. Idiopathic LPR has the potential to affect the nasal passages diffusely, even though the history might suggest unilateral disease. The response to treatment with antimicrobials, antihistamines, and glucocorticoids was poor in this study.

Potential causes for idiopathic LPR considered in this study included an unidentified foreign body or undiagnosed neoplastic or mycotic disease. Failure to identify a primary cause is possible, given the selectivity of biopsy sampling but unlikely given the adequate quantity and quality of the biopsy specimens. Other hypotheses for the causes of LPR include immune dysregulation, allergy, or high microbial load. Poor glucocorticoid response in dogs in this study suggests that immunemediated inflammation cannot be the sole mechanism behind idiopathic LPR, as was suggested previously.\textsuperscript{1} Although rhinitis is a common manifestation of allergy in humans, there is little evidence of allergic rhinitis in dogs. Upper respiratory tract signs have been described as signs of allergy in only 2 reports,\textsuperscript{3,15} both of which focused on the predominance of allergic dermatitis and conjunctivitis. Further research is required to identify the cause of this chronic, nonresponsive nasal disease and establish a rationale for specific treatment protocols.

References