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CASE REPORT

Severe chronic idiopathic suppurative lymphoplasmacytic rhinitis in a dog

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Abstract

A 5-year-old, intact male, Border collie dog was evaluated because of a 10-day history of right epistaxis. On physical examination, there was no evidence of nasal discharge. A mild depigmentation of the right nasal planum was observed. The infectious panel was negative for *Leishmania i.*, *Dirofilaria i.*, *A. vasorum* and tickborne infectious diseases. CT scan of the head, rhinoscopy, histopathologic examination of the right nasal mucosa, fungal culture and PCR sequencing were performed at initial diagnostic workup and follow-up. A severe and diffuse chronic idiopathic suppurative lymphoplasmacytic rhinitis was diagnosed, along with isolation of *Trichosporon ashaii*. Because of the unsatisfactory clinical response to marbofloxacin, piroxicam, itraconazole and clotrimazole, oral prednisone was administered. After the initial 2 weeks, prednisone was progressively reduced and discontinued 9 months later. The dog showed complete disappearance of clinical signs, without recurrence after therapy discontinuation.

BACKGROUND

Chronic idiopathic lymphoplasmacytic rhinitis is a common inflammatory disorder of the nasal cavity in dogs.[1–4] Young to middle-aged dolichocefalic large-breed dogs are affected most commonly.[2,2] Chronic idiopathic lymphoplasmacytic rhinitis is characterized by non-pathognomonic clinical signs, including unilateral or bilateral nasal discharge, epistaxis, reverse sneezing, stridor and inspiratory dyspnea.[1,2,4,5] Diagnosis usually is based on histopathologic identification of infiltrating plasma cells and lymphocytes in the nasal mucosa and exclusion of other underlying diseases such as nasal neoplasia, fungal rhinitis, or foreign body rhinitis.[2,6] A histologic nasal inflammation scoring system has been recently proposed in order to more objectively classify rhinitis.[7] The aetiology of chronic idiopathic lymphoplasmacytic rhinitis remains unknown. Several hypotheses have been proposed, such as innate immunity and hypersensitivity, odontogenic causes and bacterial or fungal infections.[1–3,8] Nasal CT scan may show lesions that are completely unremarkable or disclose unilateral or bilateral moderate turbinate destruction with mucus accumulation within air passages and sinuses.[9] Occasionally, the turbinate destruction may be severe, mimicking that seen with fungal rhinitis or neoplastic disease.[9,10] The most common rhinoscopic abnormalities seen are unilateral or bilateral erythema or hyperemia and oedema of the nasal mucosa with the presence of mucus within air passages. Turbinate atrophy or loss is appreciated occasionally.[11,2,6,9,10] Chronic idiopathic lymphoplasmacytic rhinitis is not only a diagnostic but also a therapeutic challenge for clinicians, since treatment is extremely frustrating, with cure rarely achieved. No specific protocols are available for treating the disease, and only a single clinical trial of 6 weeks' duration has been reported.[12] Although chronic idiopathic lymphoplasmacytic rhinitis is not a life-threatening disease, owners are often distraught at the dog's nasal obstruction or the need to clean up nasal discharge or nasal blood frequently in the house. Despite earlier reports in the literature, systemic or topical corticosteroids are seldom effective in controlling clinical signs[12] and actually may worse them.[2] Long-term administration of antibiotics with immunomodulatory effects combined with nonsteroidal anti-inflammatory agents can be helpful in some dogs.[2] Nasal biopsy specimens from dogs with chronic idiopathic lymphoplasmacytic rhinitis have been reported to display higher transcription of fungal genes than those from dogs with nasal neoplasia.[2] Preliminary experience with the administration of itraconazole occasionally led to improvement and even resolution of signs in some dogs with disease.[13,14] A few dogs with chronic idiopathic lymphoplasmacytic rhinitis have been treated empirically with topical antifungal medications (clotrimazole, enilconazole) to address a possible undiagnosed fungal rhinitis; however, improvement in clinical signs was not noted.[13]

Recently, the concurrent presence of upper respiratory and digestive endoscopic and histopathologic abnormalities was observed in some dogs with chronic idiopathic lymphoplasmacytic rhinitis.[15] Interestingly, most of the dogs that received only a gastrointestinal therapeutic approach showed complete remission or sustained improvement of respiratory clinical signs.[15]

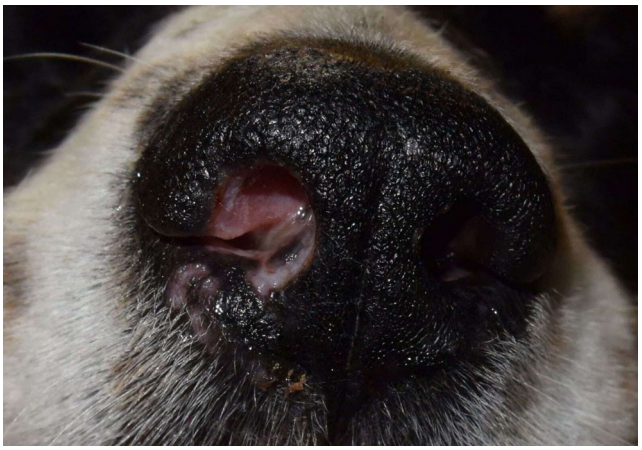


FIGURE 1 Depigmentation of the right nasal planum

This case describes a severe chronic idiopathic lymphoplasmacytic rhinitis in a dog, along with isolation of *Trichosporon ashaii*, its diagnostics and the excellent clinical response to long-term oral glucocorticoids. Moreover, this case highlights the importance of a systematic stepwise approach in the differential diagnosis of canine nasal diseases.

LEARNING POINTS/TAKE-HOME MESSAGES

- Chronic idiopathic lymphoplasmacytic rhinitis is a diagnosis of exclusion that requires an accurate stepwise diagnostic evaluation.
- Clinical history and physical examination findings generally offer an indication of primary nasal disease as opposed to systemic or extranasal diseases.
- If the dog's owners are willing to undertake expensive diagnostic workup, CT and rhinoscopy are indicated early in the disease evaluation.
- A positive fungal culture can be found in the absence of primary fungal rhinitis. Results of fungal cultures should be interpreted in light of the results of cytological and histopathological analyses and ultimately of the response to systemic or topical antifungal treatments.
- Historically, most dogs with chronic idiopatic lymphoplasmacytic rhinitis have exhibited a poor response to oral glucocorticoid treatment. However, after a fungal infection is ruled out and in case of an unsatisfactory clinical response to alternative therapies, oral glucocorticoids should be considered.

CASE PRESENTATION

The patient was a 5-year-old male intact Border collie dog of 20 kg bodyweight, which presented with a 10-day history of right epistaxis. During the last month, the dog showed several episodes of sneezing. The owner did not notice any additional clinical sign. There were no previous medical problems, nor a history of nasal trauma. The dog was current with vaccinations. Diagnostics performed by the referring veterinarian included blood work and enzyme-linked immunosorbent assay snap tests for *Ehrlichia canis*, *Ehrlichia ewingii*, *Borrelia bugdorferi*, *Anaplasma phagocytophilum*, *Anaplasma platys*, *Dirofilaria immitis*, *Leishmania infantum* and *Angiostrongylus vasorum*. Blood work revealed increased globulins and no other abnormalities. Snap tests were all negative. Amoxicillin-clavulanic acid was prescribed for 2 weeks without improvement.

On physical examination, the dog was bright, alert and responsive. The temperature was 38.3°C, the pulse was 96 beats/min, and the respiratory rate was 30 breaths/min. There was no evidence of nasal discharge. A mild depigmentation of the right nasal planum was observed (Figure 1). The remainder of the physical examination was unremarkable.

INVESTIGATIONS

Complete blood count, serum chemistry panel, urinalysis, serum protein electrophoresis, coagulation profile, immunofluorescent antibody assays for detection of *Leishmania infantum* antibodies, systolic blood pressure measurement and thoracic radiographs were performed. Only mild hyperglobulinaemia and hyperfibrinogenaemia were observed. At that point, the dog was considered a good candidate for general anesthesia. Contrast-enhanced CT scan of the head followed by rhinoscopy was then performed in order to identify

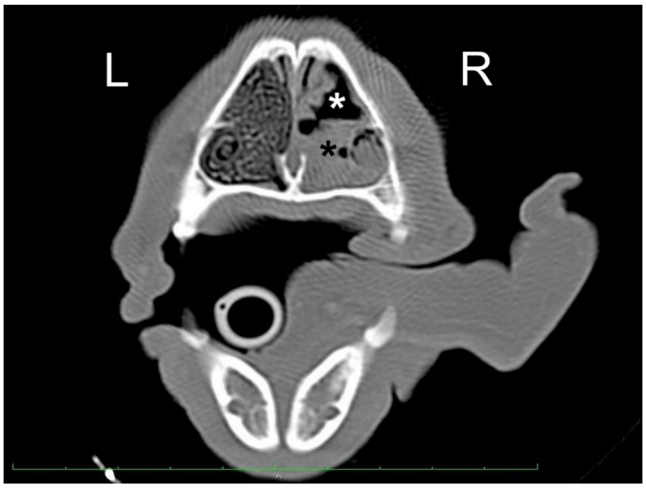


FIGURE 2 CT image from a 5-year-old M Border collie dog with a 10-day history of right epistaxis. The right nasal cavity (medium and ventral meatuses) is filled with hyperdense material (black*) with moderate destruction of the nasal turbinates (white*)

the underlying nasal, paranasal and nasopharynx disorders. On CT scan, moderate cavitary destruction of the turbinates of the right ventral meatus, modest amount of hyperdense material without enhancement in the right maxillary recess and right mandibular lymph node enlargement were observed (Figure 2). On rhinoscopy of the right nasal cavity, a considerable amount of blood from the choana, a slightly hyperemic and oedematous mucosa and an increase in the airspace of the ventral meatus along with a slightly irregular mucosal surface were observed (Figure 3). On rhinoscopy of the left nasal cavity, mild oedema and mucosal hyperemia were observed. Nasal tissue specimens from both nasal cavities were submitted for cytological examination, and from the right nasal

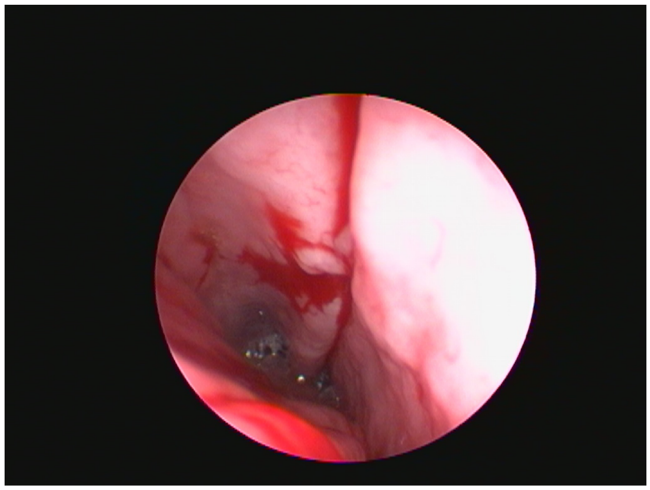


FIGURE 3 Endoscopic view of the right nasal cavity. A small amount of blood, a slightly hyperemic and edematous mucosa with a slightly irregular surface, and a small increase in the airspace can be seen

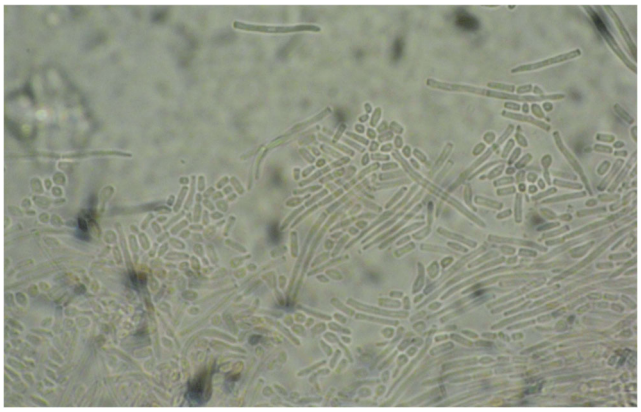


FIGURE 5 Microscopic morphology of the yeast-like colonies. Hyphae and arthroconidia (400 x magnification)

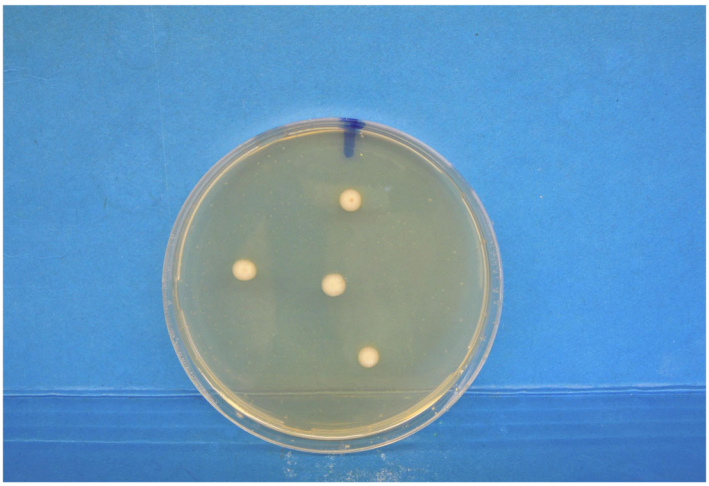
cavity for histopathological examination and fungal culture. Copious bleeding occurred after biopsy. Foreign material, although not visualized, could not be excluded. Therefore, multiple nasal lavages with warm and sterile solution were performed after tissue specimen collection. Cytology and histopathology of the right nasal cavity were consistent with a mixed neutrophilic-lymphoplasmacytic inflammatory process, and severe and diffuse chronic suppurative lymphoplasmacytic rhinitis, respectively. The total inflammatory score was 9.[7] Cytology of the left nasal cavity was consistent with normotypic-hyperplastic epithelial cells. On fungal culture, yeast-like colonies grew from the points of inoculation of the biopsy fragments (Figure 4). The microscopic morphology was consistent with that of the genus *Geotrichum* or *Trichosporon* (Figure 5). Using molecular techniques (amplification and sequencing of the internal transcribed spacer rRNA gene), the isolate was identified as *Trichosporon ashaii*.

DIFFERENTIAL DIAGNOSIS

The past history, signalment and physical examination ruled out a local idiopathic nasal planum depigmentation and local autoimmune dermatoses. Systemic autoimmune dermatoses were ruled out as the physical examination did not indicate the presence of other cutaneous or mucocutaneous lesions. Among nasal disorders, viral rhinitis was excluded because the dog was current with vaccinations and did not show other consistent systemic, respiratory, ocular, gastrointestinal, dermatologic or neurologic clinical signs.

Following the aforementioned diagnostic workup tooth root abscess, benign polyp and foreign body were excluded. Nasal mites were excluded either because clinical signs were not consistent or because of the lack of identification of parasites at cytological and histopathological examinations. Nasal washing examination for eggs was not performed because parasites were considered less likely due to the extent of destructive rhinitis identified at CT scan and rhinoscopy. At that point, fungal, bacterial, or idiopathic rhinitis and neoplasia were still on the differential diagnostic list. A neoplasia was considered unlikely since neither a mass nor neoplastic cells at cytological and histopathological analyses were identified. However, it was not definitely ruled out because severe nasal inflammation could have caused false negative results and the copious bleeding after endoscopic specimen collection made impossible to obtain multiple, big size samples. Since primary bacterial rhinitis is uncommon, while secondary bacterial rhinitis occurs more frequently due to disruption of normal mucosal integrity, the neutrophilic inflammatory infiltrate identified in this case supported this latter hypothesis. The loss of normal nasal turbinates could indicate destructive rhinitis due to fungal infection or severe idiopathic

FIGURE 4 Yeast-like colonies growth from the points of inoculation of the biopsy fragments



lymphoplasmacytic rhinitis. The lack of off-white to grey fungal plaques across the surface of the nasal mucosa and the lack of fungal elements at cytological and histopathological examinations made nasal aspergillosis unlikely. The isolation of *T. asahii* raised a diagnostic dilemma. Though there are few reports of infections in immunocompetent people,[16,17] this fungus is considered an opportunistic pathogen. Therefore, the positive culture without a cytological or histopathological confirmation could indicate only tissue colonization. On the other hand, the growth in purity from different inoculum points suggested considering a possible pathological role.

TREATMENT

The dog was treated with a combination of marbofloxacin (2 mg/kg orally every 24 h) and piroxicam (0.3 mg/kg orally every 24 h). Since fungal rhinitis was still on the differential diagnostic list, the use of glucocorticoids was thought not safe. In order to evaluate the clinical response of the dog, itraconazole (5 mg/kg orally every 12 h) was added to the treatment plan due to its low toxicity, limited costs and occasional improvement reported in dogs with chronic idiopathic lymphoplasmacytic rhinitis.

OUTCOME AND FOLLOW-UP

During the following 3 weeks, the owner referred mild inappetence, disappearance of epistaxis, followed by appearance of ipsilateral mucopurulent nasal discharge and worsening of sneezing. At the follow-up examination, on day 32, the dog resented manipulation of the muzzle, and the depigmentation of the right nasal planum was slightly worsened. Airflow was reduced on right side. The dog appeared in good general condition. Because of the lack of satisfactory clinical response to the systemic antifungal treatment and the worsening of signs, a second contrast-enhanced CT scan of the head followed by rhinoscopy was scheduled, along with cytology of the right mandibular lymph node and additional fungal cultures from ocular and nasal swabs and right mandibular lymph node aspirate. Mucosal samples were collected for cytology from both nasal cavities, and for histology, immunohistochemistry and fungal culture from the right nasal cavity. The nasal cavities were then treated with clotrimazole (1%) infusion administered topically through catheters placed endoscopically (Figure 6). Except for a worsening of the turbinate destruction of the right nasal cavity, CT and endoscopic features were similar to those observed during the previous evaluation. No fungal growth nor fungal elements were identified both at cytological and histopathological analysis and cultures. At that point, a diagnosis of severe diffuse chronic idiopathic suppurative lymphoplasmacytic rhinitis was posed. Primary fungal rhinitis and neoplasia were excluded based on negative fungal culture, immunohistochemistry and histopathological results. Piroxicam and itraconazole were interrupted. Prednisone (1 mg/kg orally every 12 h for 2 weeks) was added to the therapeutic plan. Marbofloxacin was discontinued 2 weeks later, while oral prednisone, after the initial 2 weeks, was progressively reduced and discontinued 9 months later. In the meantime, the dog showed a progressive improvement of the respiratory signs with complete disappearance of epistaxis, sneezing and mucopurulent discharge and marked improvement of nasal planum depigmentation (Figure 7). No recurrence of signs after therapy discontinuation was reported.

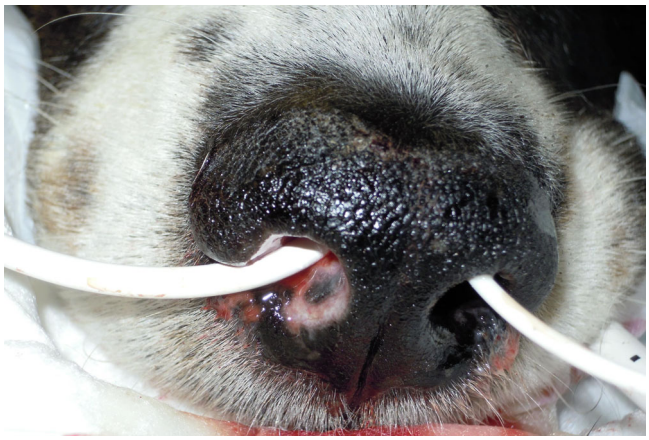


FIGURE 6 Nasal catheters placed endoscopically for clotrimazole infusion

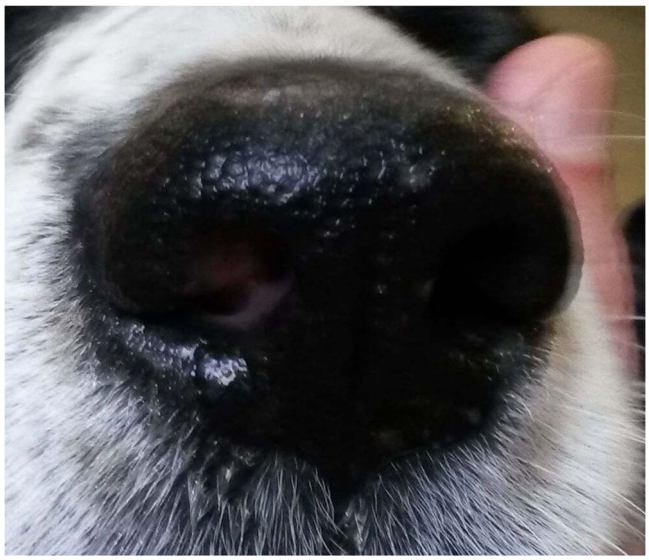


FIGURE 7 Nasal planum at follow-up

DISCUSSION

This case describes the diagnosis and therapeutic management of a 5-year-old, Border collie dog with severe diffuse chronic idiopathic suppurative lymphoplasmacytic rhinitis, along with isolation of *Trichosporon ashaii*, highlighting the usefulness of a systematic stepwise approach and the unusual excellent clinical response to long-term oral glucocorticoids.

Chronic inflammatory rhinitis is commonly found in dogs with chronic nasal disease, and it is characterized by lymphoplasmacytic infiltrates in the nasal mucosa in the absence of an obvious aetiologic process, as in this case.[1,2] Sneezing associated with mucous or mucopurulent chronic discharge is the most commonly reported clinical presentation[5]; while pure or mixed haemorrhagic discharge is more common with nasal neoplasia, foreign bodies and nasal mycosis,[5] unlike what it was observed in this dog. The majority of

dogs with unilateral clinical signs have bilateral nasal mucosal pathology, although severity of inflammation between the two sides of the nasal cavity often varies.[1,2] In this case, both nasal cavities were endoscopically abnormal; however, mucosal collection for histopathology was performed only from the right side because of copious bleeding that occurred after biopsy. Turbinate destruction, mimicking that seen with fungal rhinitis, is only occasionally observed in lymphoplasmacytic rhinitis.[6] Since it was observed here, particular attention was posed to exclude primary fungal rhinitis, as multiple colonies of *T. asahii*, as a single organism, were cultured after the first mucosal endoscopic collection. In a previous study, fungal DNA was detected in biopsies from all dogs with chronic nasal disease, but not in biopsies from healthy control dogs.[18] As expected, dogs with nasal aspergillosis had the highest levels of fungal DNA, but biopsies from dogs with lymphoplasmacytic rhinitis demonstrated significantly higher levels than biopsies of dogs with neoplasia.[18] As stated by the authors, the increased fungal DNA in dogs with chronic nasal disease could reflect poor mucosal defense mechanisms and impaired mucociliary clearance, allowing invasion of potential pathogens. Moreover, significant difference in fungal load between dogs with lymphoplasmacytic rhinitis and dogs with nasal neoplasia might suggest a link between presence or retention of fungal species in the nose and the pathogenesis of lymphoplasmacytic rhinitis. Several studies of chronic rhinosinusitis in humans have demonstrated that fungal DNA can be detected in the nasal mucosa of healthy patients as well as those with the disease.[19] These comparable findings might suggest that chronic nasal inflammation or hypersensitivity response to airborne fungi might contribute to disease in both species.[20] Recently, there have been few reports of human infections by *T. asahii* in the absence of immunosuppressive conditions,[16,17] while in veterinary medicine scattered information is available.[21] In the present case, since a fungal rhinitis was not initially ruled out, and antifungal medications may occasionally be effective in the treatment of chronic idiopathic lymphoplasmacytic rhinitis or undiagnosed fungal rhinitis,[13,14] itraconazole and clotrimazole were used before glucocorticoids. The absence of fungal elements at the second histopathologic examination and the negativity of the second fungal culture can be interpreted in two ways: 1) *T. ashaii* was a simple colonizer, 2) the fungus was responsible for a secondary infection resolved by itraconazole therapy.

In any case, since the dog showed a poor clinical response to itraconazole, primary fungal rhinitis could be excluded. Newer azoles, such as voriconazole are purportedly superior to itraconazole for treatment of *T. ashaii* and could be considered for therapeutic use in selected cases, depending on their availability and costs.[22]

Topical nasal steroid drops may be applied if the dog will tolerate administration. Topical glucocorticoid sprays such as fluticasone propionate have been used with variable success in human chronic rhinosinusitis patients and have shown anecdotal promise in some dogs with chronic nasal discharge.[2,23] Oral glucocorticoids have rarely proven effective in treating most dogs with chronic idiopathic lymphoplasmacytic rhinitis,[12] and long-term use of oral steroids should be avoided because of systemic side effects. Furthermore, the administration of immunosuppressive doses of glucocorticoids without ruling out a fungal infection could lead to worsening of symptoms or death. Indeed, glucocorticoids exert many quantitative and qualitative immunosuppressive effects that induce cellular immunodeficiency.[24] Although these effects impact all arms of the immune response, the cell-mediated Th1 response, critical for defense against fungal organisms, is particularly inhibited.[24,25] Moreover, the increased host susceptibility to opportunistic fungal infections also depends on glucocorticoid-induced effects on fungi such as increased growth and colonization rates, increased bloodstream translocation from the gastrointestinal tract and increase adherence capacity to mucosal cells.[24] Besides these risks, recognition of such infections may be delayed, since the anti-inflammatory properties of glucocorticoids blunt the symptoms of fungal infection. Last but not least, opportunistic fungal infections associated with glucocorticoid therapy depend on the administration route (inhaled glucocorticoids have a lower potential for causing fungal infections than do systemic glucocorticoids), dose (high doses correlate with high risk of fungal infection), potency (dexamethasone and methylprednisolone are more potent than prednisone) and duration of treatment (restriction of glucocorticoid administration to less than 21 days might reduce infectious complications).[24] Over the past years, the frequency of opportunistic fungal infections has increased substantially not only in humans but also in dogs with the use of multi-agent immunosuppressive therapy to treat immune-mediated diseases.[26,27] Although the prevalence of documented serious fungal infections appears to be relatively low in dogs treated with glucocorticoids and cyclosporin, the poor outcome in the majority of the dogs of a case series[27] highlights the need for caution in the use of immunosuppressive therapy until a fungal infection has been ruled out and frequent clinical monitoring once treatment is started.

In this case, the clinical response to immunosuppressive and long-term anti-inflammatory doses of prednisone was excellent, while the response to antifungals was poor, contrary to what is occasionally reported in the literature. Since a primary fungal infection was finally ruled out here, it is possible that the positive response of dogs with chronic idiopathic lymphoplasmacytic rhinitis to antifungals, as observed in previous studies, was due to the presence of an undiagnosed mycotic infection. Based on these results, an immune-mediated mechanism triggering the inflammatory response cannot be excluded here. However, further studies evaluating the immunoregulatory patterns in the nasal mucosa of dogs with lymphoplasmacytic rhinitis are required to determine the role of immune dysregulation in this disease.

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