

Canine Chronic Rhinitis

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Canine chronic inflammatory rhinitis represents a frustrating, multifactorial, and often lifelong condition in which the clinician's goal is not cure but establish meaningful symptom control. This presentation will initially review the normal nasal anatomy and provide a broad differential list for chronic nasal discharge that emphasizes inflammatory rhinitis, neoplasia, fungal disease, dental pathology, and foreign bodies which dominate the clinical landscape. Because clinical signs such as sneezing, stertor, and nasal discharge—progressing from serous to mucopurulent to epistaxis—are nonspecific, a systematic diagnostic approach is essential. Initial testing includes CBC, chemistry, urinalysis, coagulation panel, blood pressure, and a sedated oral exam, while nasal cytology, culture, and skull radiographs are often discouraged due to being a low diagnostic yield process. It should be noted that nasal cultures are “not reliable” and are often site-dependent. Computed Tomography (CT) is considered the single most valuable diagnostic tool, providing detailed assessment of nasal passages, sinuses, dentition, and middle ears, and guiding rhinoscopic evaluations. Rhinoscopy typically reveals hyperemic, friable mucosa with variable discharge, but histopathology remains the only definitive diagnostic method, as emphasized by the line: *“the only way to truly diagnose this disease.”*

Chronic idiopathic lymphoplasmacytic rhinitis (LPR) is considered a diagnosis of exclusion—noninfectious, nonallergic, and nonneoplastic—accounting for 20–40% of chronic nasal disease. Dogs show highly variable signs, with 95% exhibiting nasal discharge and roughly equal frequency of unilateral and bilateral involvement. The etiology is likely immune-mediated involving aberrant innate and adaptive responses, increased Toll-like receptor expression, and TH₂-skewed cytokine patterns. Treatment focuses on a multimodal, long-term management approach. Antihistamines and oral corticosteroids provide little durable benefit and are not recommended as primary therapy. Saline therapies—including sprays, nebulization, and “voluntary head dunking”—help reduce mucus burden and improve delivery of intranasal medications. Intranasal corticosteroids (e.g., fluticasone, mometasone, budesonide) are a cornerstone of therapy with anecdotal success and low cost. Although the intranasal administration poses challenges to uncooperative patients, the incidence of systemic side effects are uncommon. Inhaled MDI corticosteroids may help but have variable deposition intranasally. Leukotriene inhibitors (montelukast, zafirlukast) show limited efficacy alone but may benefit in 20–25% of dogs when combined with select antihistamines. Antibiotics such as doxycycline or azithromycin may reduce secondary bacterial populations and inhibit metalloprotease activity, but are not curative. NSAIDs—particularly piroxicam, meloxicam, and firocoxib—emerge as one of the most evidence-supported treatments; a retrospective study showed NSAID use was “significantly associated with clinical improvement,” making them a strong first-line option. Experimental or emerging therapies involving lokivetmab, JAK

inhibitors, and low-dose external beam radiation will be discussed. Ultimately, chronic inflammatory rhinitis requires patience, realistic expectations, and individualized multimodal therapy aimed at achieving a >50% reduction in symptom severity—what you describe as determining “what is tolerable” for each patient.