Laryngeal Disease Case Report

Sherry L. Blackmon

Advisor: Dr. Andrew Mackin

Introduction

A history of voice change and stridor (high pitch wheeze or whistle) in dogs is highly suggestive of laryngeal disease. The most common laryngeal disease in dogs is laryngeal paralysis, which refers to the failure of the arytenoid cartilage to abduct during inspiration, effectively creating a partial upper airway obstruction [1, 2, 4]. Other differential diagnoses for laryngeal disease in dogs includes laryngeal neoplasia, obstructive laryngitis or inflammatory disorders, acute laryngitis, laryngeal collapse, trauma, foreign body, extraluminal mass, and webbing or post-surgical complications (fibrotic adhesions) [1,3]. Diagnosis of laryngeal disease is usually made based upon history, clinical signs, and direct laryngeal examination. Treatment depends upon the underlying pathology but, in cases of laryngeal paralysis, treatment is often categorized as emergency treatment in cases of respiratory distress, conservative medical management or surgical treatment for severely affected dogs [1, 2, 4].

History and Presentation

A 4-year-old 26.1-kg (57.5-lb) intact male Mountain Cur/Mixed Breed dog trained for feral swine hunting was referred to the Mississippi State University College of Veterinary Medicine (MSU-CVM), Department of Internal Medicine, for acute voice loss that occurred approximately 6 months previously. His owner first noticed the voice loss while hunting when the dog attempted to bay at feral swine but was unable to do so. The owner reported hearing the dog bay every weekend previously that month. Approximately 1-2 weeks following the acute voice loss, his owner noticed a harsh breathing sound, especially apparent during periods of excitement and increased physical activity. The dog's voice never returned, and the harsh breathing sounds persisted unchanged. Laryngeal exam performed by the referring veterinarian was suggestive of laryngeal paralysis, and the dog was referred to MSU-CVM for additional

diagnostics and treatment recommendations. Incidentally, the referring veterinarian also diagnosed the dog as heartworm positive.

Upon presentation at MSU-CVM, the dog was bright, alert, and responsive, and his vitals were clinically unremarkable. When observed at rest and kennel confined, his respiratory sounds were quiet and normal; however, when excited a loud stridor was easily appreciated. There were healed abrasions over many parts of his body, including his mouth, thorax, and abdomen and bilaterally along the lateral tarsal region, consistent with his history of hunting and trapping feral swine. Auscultation of his lungs revealed referred upper airway sounds but was otherwise normal. Auscultation of his heart was unremarkable. His neurological exam was within normal limits with no evidence of cranial nerve or thoracic or pelvic limb abnormalities. The remainder of his physical exam was clinically unremarkable.

Pathophysiology

Based upon the patient history and presentation, including the clinical signs of voice change and subsequent loud stridor and exercise intolerance, the differential diagnoses were limited to disease processes of the larynx. Laryngeal paralysis is the most common laryngeal disease in dogs and was the suspected diagnosis, although the dog did not meet the typical signalment for acquired laryngeal paralysis (> 9-years, Labrador and giant breeds predisposed) [1]. Laryngeal paralysis occurs when the arytenoid cartilage fails to abduct during inspiration, effectively creating a partial upper airway obstruction [1, 2, 4]. The etiology of the disease may be congenital or acquired (idiopathic, secondary to trauma, neoplasia, generalized polyneuropathy, hypothyroidism and myasthenia gravis) and may occur unilaterally or bilaterally [1, 2, 4].

In the case of this patient, laryngeal exam revealed attempted movement of the arytenoid cartilage, effectively ruling out laryngeal paralysis; however, arytenoid movement was impeded by a markedly abnormal anatomical conformation of the larynx. The airway opening (rima glottidis) was smaller than normal. The epiglottis had a straight-edge appearance at the cranial margin that was not the leaf-like shape typical of a normal epiglottis and the cranial margin was connected to the frenulum of the tongue. Both corniculate processes of the arytenoid cartilage were abnormally flattened and partially fused dorsally. The arytenoids were stiff and did not abduct normally bilaterally.

Biopsies were obtained of the arytenoids and epiglottis. The arytenoid tissue was histologically unremarkable and without evidence of inflammation or fibrosis. The epiglottis tissue was suggestive of chondroid metaplasia. The epiglottis was composed of squamous epithelium overlying connective tissue and within the connective tissue there was cartilage and dense collagen. The cartilage contained plump chondrocytes irregularly spaced and occasionally binucleated with one very large chondrocyte with a large nucleus that was unusual. The chondroid tissue in the epiglottis was unusual in that the chondrocytes were somewhat disorganized and within a poorly staining matrix. Within the sample it was unknown as to how the chondroid tissue interfaced with the cartilage of the epiglottis.

There are no reports in canine patients regarding chondrometaplasia in the larynx. In human medicine, there is a case report regarding a history of laryngeal trauma and acute clinical signs that helped differentiate chondrometaplasia from cartilaginous tumors (i.e. chondromas and low grade chondrosarcomas) [5]. The human case report stated that chondrometaplasia caused by mesenchymal degeneration secondary to trauma was characterized by well-organized submucosal nodules of cartilage tissue with minimal atypia [5]. Some atypia was also seen in the

histology of our canine patient; however, given the sparse information in the literature it is difficult to know if this is within the acceptable standards for chondrometaplasia.

Congenital laryngeal disease is relatively rare across species, and anatomical and histological differences present a challenge in cross-species comparisons. Within these limitations, dogs are frequently the preferred animal model for studying human laryngeal diseases [6]. In human literature, the most commonly reported congenital laryngeal disease and cause of stridor in infants is laryngomalacea which results from flaccid (collapsing) supraglottic tissue [7, 8, 9]. Symptoms are usually self-limiting and resolve within 24 months of age, whereas severe cases are managed surgically with transoral supraglottoplasty [8, 9]. Other congenital human laryngeal diseases include vocal fold paralysis, which may be unilateral or bilateral and is similar to congenital laryngeal paralysis in dogs, laryngeal atresia and subglottic stenosis, laryngeal-esophageal cleft, laryngeal web and subglottic hemangiomas [10].

Less is known regarding laryngeal disease in cats, although they share many of the same differential diagnoses as dogs [1, 11]. In one retrospective study of 39 suspected laryngeal disease cases referred to the University of Bristol during 1996-2006, cats were diagnosed with laryngeal paralysis (n=14), laryngeal neoplasia (n=10), laryngeal inflammation (n=6) or miscellaneous laryngeal disease (n= 5) (four patients were excluded for lack of definitive diagnosis) [11]. Of the five cats classified in the miscellaneous laryngeal disease category, two cats were littermates (3-year-old FS Burmese cats) and suspected to have a congenital anomaly exacerbated by a recent inflammatory process. The littermates were presented with a ten day history of inspiratory sounds, dyspnea, coughing and gagging/retching following a cattery stay. The cats were negative for FeLV, FIV, FHV, and FCV. Echolaryngography was suggestive of severe thickening of the vocal folds and direct laryngeal exam under anesthesia of the most symptomatic cat confirmed

severe swelling and a small lumen. Clinical symptoms improved for both cats following treatment with prednisolone and doxycycline. Repeat echolaryngography performed one month later showed resolution of the swelling; however, the lumen of both cats remained abnormally small. In retrospect, the owners felt that the cats showed mild symptoms of stridor and dysphonia even as kittens. Based upon the history and clinical presentation, researchers concluded the cats likely suffered a congenital anomaly exacerbated by a recent inflammatory process. One of the cats continued to experience dyspnea and was euthanized at 1080 days, while the other had mild symptoms of increased inspiratory effort and was lost to follow-up at 720 days.

Diagnostic Approach and Considerations

A definitive diagnosis of laryngeal disease may utilize laryngeal radiography, ultrasonography, fluoroscopy, computed tomography/MRI, laryngoscopy/endoscopy and laryngeal biopsy [1, 4, 12]. Laryngoscopy/endoscopy and laryngeal biopsy were elected in this case. The pre-anesthetic minimum database consisted of thoracic radiographs, a serum chemistry, CBC and urinalysis. These results were clinically unremarkable and indicated low anesthetic risk. The plane of anesthesia is an important consideration when performing the laryngeal exam, and a light plane of anesthesia is necessary to prevent a false-positive diagnosis of laryngeal paralysis [4]. In the case of this patient, laryngeal exam ruled out laryngeal paralysis; however, arytenoid movement was impeded by the abnormal anatomical conformation of the larynx. Biopsies were obtained of the arytenoids and epiglottis and two absorbable (4-0 Vicryl) sutures were placed in the left corniculate process. Dilute epinephrine was used to control bleeding and an anti-inflammatory dose (0.1mg/kg) of dexmethasone SP was administered to reduce post-biopsy laryngeal inflammation. Once laryngeal bleeding resolved, the patient was extubated and recovered without complication.

Treatment and Management

The patient was diagnosed with laryngeal anatomic anomaly, etiology unknown; however, his treatment recommendations were based on the clinical signs associated with laryngeal paralysis. Conservative management of laryngeal paralysis includes activity restriction, reducing excitement levels, avoiding exposure to hot, humid weather and use of a harness rather than a neck collar during walks. Dogs that are unresponsive to conservative management measures or have more severe signs of respiratory distress are better managed with surgery. Surgery for laryngeal paralysis may be performed electively or emergently, depending on the severity of the respiratory signs and ability to stabilize the patient without surgery. The goal of surgery is to provide the patient with a larger airway by increasing the size of the rima glottides [4]. Surgery is classified as either intra-laryngeal or extra-laryngeal, including cricoarytenoid cartilage lateralization ("tie-back") [4]. Tie-back is currently considered the procedure of choice, and the objective of this procedure is to prevent passive adduction (failure of abduction) of the arytenoid cartilage during inspiration by fixing it to a neutral or slightly lateral position [4]. Surgical complications include aspiration pneumonia, persistent coughing, stridor, gagging, panting, seroma formation, vomiting, exercise intolerance and webbing (fibrotic lesions) [4].

Case Outcome

The patient was discharged from MSU-CVM following laryngeal examination and biopsies. His owner elected for conservative management which included retirement from hunting and keeping the dog as a pet only. The client was pleased that a surgical consultation by the MSU-CVM Surgery Service was performed concurrently with the laryngeal examination and biopsy. Client education ensured the owner recognized that surgery is most frequently a salvage procedure for dogs with severe clinical signs and surgery would not likely result in a return to

athletic performance. Questions that still remain include whether the underlying cause of the laryngeal abnormalities was congenital or hereditary, or the result of trauma or other acquired pathological process. Hunting feral swine often involves physical contact and the dogs wear Kevlar protective gear; however, the cervical region is frequently exposed. If trauma is responsible for the clinical symptoms and anatomical abnormalities detected during the laryngeal examination, it would have been a significant trauma with external signs likely to have occurred. However, the owner doesn't recall any specific trauma to the cervical region. On the other hand, if the underlying cause of the laryngeal abnormality is a congenital pathological process, one would expect the presentation of clinical symptoms earlier in life. However, as the case of the 3-year-old Burmese littermates suggests, perhaps congenital anomalies can go unnoticed until they are exacerbated by other causes. The client was interested in ethical breeding of a skilled hunting dog and hoped that biopsy would definitively distinguish between congenital and acquired disease, but this was not the case. The client also shared partial ownership of some of this patient's littermates and questioned whether a laryngeal examination was warranted in these asymptomatic dogs. The client was encouraged to follow-up with MSU-CVM or his referring veterinarian if there was any change in the patient's clinical symptoms or if littermates became symptomatic.

References

1. MacPhail C. Laryngeal Disease in Dogs and Cats. Vet Clin North Am Small Anim Pract. 2014; 44:19-31.
2. Hawkins, EC. Chapter 18: Disorders of the Larynx and Pharynx. In: Nelson RW, Couto CG, eds. Small Animal Internal Medicine. 5th ed. St. Louis: Elsevier Mosby, 2014; 253-257.

3. Hawkins, EC. Chapter 16: Clinical Manifestations of Laryngeal and Pharyngeal Disease. In: Nelson RW, Couto CG, eds. Small Animal Internal Medicine. 5th ed. St. Louis: Elsevier Mosby, 2014; 247-248.
4. Kitshoff A, Goethem B, Stegen L, Vandekerckhove P, Rooster H. Laryngeal paralysis in dogs: an update on recent knowledge. J S Afr Vet Assoc. 2013; 84: E1-9.
5. Orlandi A, Fratoni S, Herrmann I, Spagnoli L. Symptomatic laryngeal nodular chondrometaplasia: a clinicopathological study. J Clin Path. 2003; 56: 976–977.
6. Garrett CG, Coleman JR, Reinisch L. Comparative histology and vibration of the vocal folds: implications for experimental studies in microlaryngeal surgery. Laryngoscope. 2000; 110:814-24.
7. Richter GT, Thompson DM. The surgical management of laryngomalacia. Otolaryngol Clin North Am. 2008; 41:837-64.
8. Dobbie AM, White DR. Laryngomalacia. Pediatr Clin North Am. 2013; 60:893-902.
9. Thorne MC, Garetz SL. Laryngomalacia: Review and Summary of Current Clinical Practice in 2015. Paediatr Respir Rev. 2016;17:3-8.
10. Ahmad SM, Soliman AM. Congenital anomalies of the larynx. Otolaryngol Clin North Am. 2007; 40:177-91.
11. Taylor SS, Harvey AM, Barr FJ, Moore AH, Day MJ. Laryngeal disease in cats: a retrospective study of 35 cases. J Feline Med Surg. 2009; 11:954-62.
12. Hawkins, EC. Chapter 17: Diagnostic Tests for the Larynx and Pharynx. In: Nelson RW, Couto CG, eds. Small Animal Internal Medicine. 5th ed. St. Louis: Elsevier Mosby, 2014; 249-252