Molly's Laryngeal Malady

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Class of 2022

Clinicopathologic Conference
October 15th, 2021

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Introduction:

Laryngeal paralysis occurs when the arytenoid cartilages either partially or completely fail to abduct during inspiration, leading to an upper respiratory obstruction. This obstruction results in progressive inspiratory stridor, exercise intolerance, and hoarse voice changes. Laryngeal paralysis is common in large-breed, male dogs.[3] It has been reported but is uncommon in cats.[7] The disease can be either congenital or acquired, but the most common form is acquired idiopathic laryngeal paralysis which is now thought to be part of a generalized, progressive polyneuropathy. This form is most commonly seen in older dogs, most commonly Labrador retrievers.[6]

History and Presentation:

Molly, a 12-year-old female spayed Labrador retriever, presented to the MSU-CVM Small Animal Surgery Service on 6/22/2020 for ongoing respiratory issues. In February 2020, Molly presented to her primary veterinarian for waking during the night seeming out of breath, exercise intolerance, and having a "weak bark". Thoracic radiographs revealed mild right heart enlargement and mild pulmonary edema. Molly was prescribed Enalapril and Lasix. At a recheck exam in April, gabapentin and prednisone were also prescribed. On June 15th, a sedated laryngeal exam revealed suspected laryngeal paralysis. During this visit, Molly's primary veterinarian repeated thoracic radiographs and discontinued all medications except enalapril. Molly historically had issues with her hips, suspected to be osteoarthritis, and received a joint supplement in the past.

On presentation, Molly was bright, alert, and responsive. She weighed 33.4 kg with a body condition score of 7/9. Rectal temperature was mildly elevated at 103.8° F. She had a pulse of

148 beats per minute and a respiratory rate of 36 breaths per minute. Her mucous membranes were pink and moist with a normal capillary refill time of less than 2 seconds. Cardiopulmonary auscultation revealed no murmurs or arrhythmias. Stridor and mildly increased respiratory effort were noted. Mild dental tartar was present. Mild waxy debris was noted within both external ear canals. The abdomen was tense but non-painful. Peripheral lymph nodes were small, soft, and symmetrical. Numerous masses/nodules were noted on the face, chest, abdomen, and dorsum. The remainder of her physical exam was within normal limits.

Pathophysiology:

The etiologies of this condition can be divided into two broad categories: congenital or acquired. Congenital laryngeal paralysis is seen in young dogs, where clinical signs are typically recognized by 4-6 months old.[7] Several breeds including Siberian huskies, Bouviers des Flandres, bull terriers, and white-coated German Shepherd Dogs have been discovered to have congenital, hereditary forms of laryngeal paralysis.[3] In Bouviers des Flandres, it has been identified as an autosomal dominant trait that causes secondary Wallerian degeneration of the recurrent laryngeal nerves.[4] Dalmatians, Rottweilers, Leonbergers, and Afghan hounds are just a few of the breeds that are reported to be associated with a congenital laryngeal paralysis polyneuropathy.[4] Axonal degeneration and loss of myelinated nerve fibers in the recurrent laryngeal nerve and paralaryngeal nerves in Dalmatians and Rottweilers has been shown.[4] Acquired laryngeal paralysis is most commonly associated with what is now being termed GOLPP (geriatric onset laryngeal paralysis polyneuropathy). This syndrome displays itself in middle aged to older dogs as a progressive generalized polyneuropathy, where within approximately 1 year of a dog's diagnosis of laryngeal paralysis they develop systemic neurologic signs as well. This historically has been termed acquired idiopathic laryngeal

paralysis (AILP), however it has recently been shown with histopathology and electrodiagnostic tests that this is actually a type of progressive generalized polyneuropathy.[3] One study showed that 50% of dogs who presented with idiopathic laryngeal paralysis had diffuse neurologic co-morbidities in the pelvic limb and/or esophageal deficits. Another study showed that 31% of dogs with laryngeal paralysis had conscious proprioceptive deficits at the time of diagnosis, and within one year 100% of them did.[1] GOLPP is most commonly seen in Labrador retrievers, but may also be seen in Rottweilers, Afghan hounds, Irish setters, golden retrievers, St. Bernards, and standard poodles.[4,6] Acquired laryngeal paralysis may less commonly be due to damage of the recurrent laryngeal nerve. This damage could be caused by trauma, iatrogenically via surgery, or cervical/intrathoracic neoplasia. Diseases such as hypothyroidism, myasthenia gravis, and systemic lupus erythematous may also be the cause of laryngeal paralysis. [3,4]

Regardless of the underlying cause of laryngeal paralysis, it affects the dogs all in the same way. The recurrent laryngeal nerves innervate the laryngeal abductor and adductor muscles, including an abductor called the cricoarytenoideus dorsalis muscle. When there is atrophy of this muscle due to one of the underlying causes listed above, the arytenoid cartilages and the vocal cords cannot be abducted, and instead remain in a paramedian approach during inspiration.[3] This narrowing in the glottic lumen decreases airflow into the respiratory tract and increases resistance, resulting in turbulence which is heard as stridor.[3,4] To compensate for this increased airflow through the larynx the intraglottic pressure drops, causing the arytenoid cartilages and vocal cords to be sucked in medially which worsens the obstruction. This insufficient abduction can predispose the dog to aspiration pneumonia.[3] Dogs may present in acute respiratory distress if they become too overexerted from excitement or heat and go into a “vicious cycle of increased respiratory rate, trauma to the arytenoid mucosa, inflammation, swelling and hyperthemia.”[1]

Differential Diagnoses:

Other diseases that cause upper respiratory obstructions should be considered when compiling a differential diagnoses list. These may include brachycephalic syndrome, laryngeal collapse, tracheal collapse, and masses or trauma involving the upper airway.[3] Masses may be a result of a foreign body causing swelling and infection, or of neoplasia- specifically squamous cell carcinoma or lymphoma. Other less commonly reported diseases to consider may include granulomatous laryngeal disease and fungal mycetoma.[2]

Diagnostic Approach/Considerations:

Signalment and history are essential parts of reaching a diagnosis, as they allow you to determine the underlying cause of the laryngeal paralysis (congenital vs acquired). This may be further aided by a full neurologic exam. In acquired laryngeal paralysis, the patient may show evidence of muscle wasting, weakness, or exhibit other neurologic deficits.[3] While laryngeal paralysis cannot be detected on radiographs, cervical and thoracic radiographs should still be performed in order to rule out other differentials for upper respiratory obstructions as well as to identify any comorbidities or secondary illnesses such as megaesophagus, post-obstructive pulmonary edema, or aspiration pneumonia.[3,4] Bloodwork to evaluate total T4 and TSH levels should be completed to rule out a hypothyroid neuropathy, as 30-40% of dogs with acquired laryngeal paralysis have been found to be concurrently hypothyroid.[3]

A sedated, direct laryngeal exam is the mainstay for definitive diagnosis. During respiration the movement of the larynx must be visualized, as dogs that are affected with laryngeal paralysis do not have abduction of their arytenoid cartilages during inspiration and remain in a paramedian position. Visualizing edema of the corniculate processes or laxity of the vocal cords can also aid

in diagnosis.[5] During this exam, it is helpful to have an assistant who can call out the phases of respiration while you are visualizing the larynx to avoid confusing paradoxical movement with abduction of the arytenoids. Paradoxical movement occurs when air being released on expiration causes the arytenoid cartilages to move and can appear similar to abduction.[1] If necessary, a respiratory stimulant called doxapram may be administered to encourage deeper breaths and facilitate visualization of arytenoid cartilage abduction.[5]

Treatment and Management:

Treatment of laryngeal paralysis is dependent on the severity of clinical signs (primarily respiratory distress) that the patient is experiencing. If a dog has mild respiratory signs, then it may be possible to manage them with several life-style changes. This may include maintaining a lean body weight or losing weight, decreasing their activity level, using a harness as opposed to a collar, avoiding situations that cause them to become excitable or stressed, and limiting time in high ambient temperatures.[3,4] If the dog goes into acute respiratory distress, emergent medical treatment is needed that typically involves: supplemental oxygen, sedation, corticosteroids and active cooling. In some cases, temporary endotracheal intubation or tracheostomy must be implemented until the episode of acute distress is over.[1] If a dog's quality of life becomes significantly impacted by their laryngeal paralysis and medical management is no longer sufficient, then surgery is often the next step. While there are several different types of surgery available, currently the unilateral arytenoid lateralization surgery (AKA tieback) is considered the gold standard.[1]

On June 24th, 2020 Molly was anesthetized for a unilateral arytenoid lateralization (left laryngeal tieback). The patient was placed in right lateral recumbency and the left side of the neck was clipped, aseptically prepared with chlorhexidine 4% scrub, alcohol, and Duraprep then draped

for surgery. A skin incision was made using a #10 blade ventral to the jugular vein, beginning at the caudal angle of the mandible and extending slightly caudal to the larynx. The subcutaneous tissues and platysma muscles were incised and retracted with Metzenbaum scissors to expose the laryngeal area. The thyroid cartilage was palpated and the thyropharyngeus muscle was incised along the dorsolateral margin of the thyroid cartilage lamina. Two stay sutures were placed through the thyroid cartilage to retract it laterally. The cricoarytenoideus dorsalis muscle was dissected and transected at the muscular process of the arytenoid cartilage. Two sutures of 3-0 Prolene on a Taper were pre-placed through the caudal aspect of the cricoid cartilage and again through the muscular process of the arytenoid cartilage. The patient was extubated and the larynx was visualized intra-orally to verify sufficient abduction. The sutures were then tied down and the patient was intubated again. The surgical site was lavaged. The platysma muscle was apposed with 3-0 PDS in a simple continuous pattern. The deeper adipose tissues were closed with 3-0 PDS suture in a simple continuous pattern, and the skin was closed in an intradermal pattern with 3-0 Monocryl. The incision site was covered with a Telfa pad and Hypafix. Recovery was uneventful.

It was explained to Molly's family that her disease will continue to require lifelong management, primarily due to the fact that her surgery increased her risk of continually developing life-threatening aspiration pneumonia. Several lifestyle adjustments needed to be made, including: no swimming, offering her water under direct supervision to ensure she does not drink excessive quantities at once, offering her small, frequent, hand-fed meals as "meatballs" or softened kibble. Other necessary adjustments included walking Molly on a harness (instead of a neck collar) to avoid excess pressure on her airways.

Case Outcome:

Molly's surgery and recovery from anesthesia were seemingly without complications; however, the following morning she became febrile and developed a productive cough. Thoracic radiographs were repeated which revealed aspiration pneumonia. She was treated with nebulization and coupage, metoclopramide, as well as intravenous Unasyn and enrofloxacin up until her discharge date on June 30th, 2020. At this point, Molly was switched to oral medications, and her owners were instructed to continue her nebulization and coupage at home. At the time of discharge, Molly was eating, drinking, urinating, and defecating normally. She was comfortably breathing with minimal to no effort on room air. On July 8th, 2020 a follow-up call was made to Molly's owners for an update. They reported the day prior, she had potentially aspirated while eating and had been excessively coughing and regurgitating since. Her owners brought her to their RDVM who noted she was febrile and depressed, and immediately restarted her antibiotics as well as several other medications according to Molly's owners. On July 17th, 2020 was the final communication with Molly's owners, who reported she had greatly improved and was continuing to do well.

Conclusion:

Geriatric-onset laryngeal paralysis polyneuropathy (GOLPP) is the most common form of laryngeal paralysis, and is most commonly seen in older, large-breed male dogs. Approximately 70% of GOLPP cases occur in Labrador Retrievers.[6] Key clinical signs include exercise intolerance, voice changes, and inspiratory stridor.[4] When laryngeal paralysis can no longer be managed medically and with life-style changes alone, unilateral arytenoid lateralization surgery is the treatment of choice. Aspiration pneumonia is the most common complication of this surgery and can occur at any point post-operatively. One study showed that in the three years

following surgery, 31.8% of patients developed aspiration pneumonia.[8] Despite this, the patient's prognosis with surgery is good. Up to 90% of dogs who undergo this surgery will improve, and 70% will have survived after 5 years.[1]

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