Fin Was Still Alive, but Barely Breathing

Courtney C. Caugh

Mississippi State University

College of Veterinary Medicine

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Advisor: Seth Kettleman, DVM

Introduction

Brachycephalic airway syndrome (BAS) or brachycephalic obstructive airway syndrome (BOAS) refers to a combination of anatomical findings that have been propagated through breeding dogs (and cats) with these deformities as they have increased in popularity.[4] Any brachycephalic dog (meaning "short-headed") can be affected, but over half of the cases of BOAS reported in one study were English Bulldogs.[5] Animals may possess one or all five of the BOAS components, which include elongated soft palate, stenotic nares, everted laryngeal saccules, laryngeal collapse, and hypoplastic trachea.[4,5,6,11] Multiple studies have reported elongated soft palate as the most consistent finding.[5] Clinical signs range in severity and owners may present their pets for routine surgical correction or in need of airway stabilization due to severe respiratory distress.

History and Presentation

Fin is an approximately 10-year-old male Boston Terrier who presented to Mississippi State University College of Veterinary Medicine Ophthalmology Service on May 27, 2020 for squinting and redness of his left eye for several days to a week. He had a history of difficulty breathing after playing outside two days prior to presentation, but he recovered from the brief episode and had been behaving normally at home since that time. He had no prior history of ophthalmic disease.

On initial presentation, Fin was bright, alert, and anxious. He weighed 11 kilograms and had a body condition score of 5/9. His vitals included a heart rate of 100 beats per minute and a rectal temperature of 102.1 degrees Fahrenheit. His mucous membranes were pink and moist with a capillary refill time of less than two seconds. He was panting and had a mildly increased respiratory effort. His nares were stenotic, which is a consistent anatomic finding with his breed.

On cardiothoracic auscultation, a grade III/VI left systolic heart murmur and marked upper airway noise were appreciated. No crackles or wheezes were heard. His abdomen was tense upon palpation, but no overt masses or abnormalities were appreciated. A brief neurologic exam did not reveal any deficits.

During ophthalmologic examination, trace aqueous flare, mild conjunctival hyperemia, and corneal edema were noted in the left eye. He had normal ocular pressures and there was no fluorescein stain uptake in either eye. The remainder of his ophthalmic exam did not reveal any abnormalities, but it was very brief as Fin became increasingly agitated. His previously described increased respiratory effort progressed to respiratory distress. Cyanosis of his tongue and overall weakness were noted, and he was transferred to MSU-CVM's Emergency Service for airway stabilization.

After being rushed to the ICU, Fin received 0.4 mg/kg of butorphanol and 0.1mg/kg of acepromazine intravenously. Flow-by oxygen was administered as well. The decision to intubate Fin and start a propofol CRI was made after he continued to show signs of respiratory distress following an intravenous injection of 0.14 mg/kg dexamethasone SP. The Small Animal Surgery Service was notified of Fin's distress and arrived shortly after his initial stabilization to place a tracheostomy tube. Once stabilized, Finn was placed in an oxygen cage and was closely monitored overnight.

Diagnostic Approach

As mentioned previously, diagnostics performed at the time of Fin's initial presentation were limited due to his critical state. An upper airway examination was performed and revealed swelling and edema of various tissues, including Fin's tongue, soft palate, and pharynx. Additionally, his soft palate was elongated and everted laryngeal saccules were observed.

The following morning, blood was drawn and submitted after Fin was transferred to the Surgery Service for further diagnostics and treatment. A complete blood count revealed a stress leukogram characterized by a mild neutrophilia of 12,613.2 /uL (3100.00-11800.00/uL) and mild lymphopenia of 822.6/uL (1100.0-4800.0/uL). Chemistry panel revealed a slightly elevated ALT and ALP of 92 U/L (10-90 U/L) and 207 U/L (11-140 U/L) respectively. CK was moderately increased at 1067 U/L (50-300 U/L), as well.

To further evaluate Fin's respiratory and cardiovascular systems, cervical and thoracic radiographs were performed. Thoracic radiographs revealed a large amount of subcutaneous emphysema due to tracheostomy tube placement, an elongated soft palate, and severe edema of the soft palate and pharyngeal and cervical soft tissues. Nasopharyngeal attenuation was also noted, indicating the need for a head CT to further evaluate the structure and the cause of the soft tissue swelling. An enlarged cardiac silhouette was noted on thoracic radiographs, but it was unknown if this finding was due to valvular degeneration or a normal breed variant, which has been documented in one study comparing vertebral heart scores of brachycephalic breeds to Beagle dogs.[2] Due to Fin's history of a heart murmur, an echocardiogram was recommended and performed later that morning. Both mitral valve and tricuspid valve degeneration were noted with possible pulmonary hypertension, which has been associated with BOAS.[7]

That afternoon (5/28), Fin was sedated with 5mcg/kg of dexmedetomidine and 0.2mg/kg of butorphanol prior to re-evaluation of his airway and computed tomography of the head. Airway examination revealed substantial soft tissue swelling, consistent with radiographic findings. Head CT with contrast confirmed severe upper airway edema without indication of a mass and several incidental findings which included an enlarged pituitary gland, chronic bilateral otitis externa and media, and periodontal disease.

Pathophysiology

BOAS consists of a combination of primary anatomic findings including elongated soft palate, stenotic nares, and hypoplastic trachea.[5,6] While brachycephalics have short and compressed skulls, they develop oral tissues and nasal turbinates comparable in size to non-brachycephalic dogs.[11] A shorter muzzle leads to more compression, which correlates to increased risk of obstruction.[11] In contrast, Parker *et al.* concluded that dogs with a muzzle length at least half that of the skull did not have any risk of obstruction.[11] Additional factors such as obesity and stress perpetuate clinical signs and probability of BOAS in brachycephalics.[5,11]

Due to the primary components listed above, affected dogs have decreased pathways for airflow and experience increased resistance.[4,5,11] Decreased airflow drives higher respiratory rates and encourages secondary changes, including everted laryngeal saccules, everted tonsils, and laryngeal collapse (the most severe of these being laryngeal collapse).[4,5,11] The secondary components create a vicious cycle by inducing more edema, which can lead to a higher risk of obstruction of the larynx by the soft palate and subsequent respiratory distress.[5] In Fin's case, stenotic nares, elongated soft palate, and everted laryngeal saccules were present.

Clinical signs may vary but typically include some degree of stertor, snoring, panting, and exercise intolerance.[4,5,6,11]Severely affected animals can present in respiratory distress, with cyanosis, abdominal effort, collapse, and hyperthermia due to altered normal cooling.[5,11] A "wide-based stance with abducted forelimbs" has been described as a common posture on presentation, as dogs attempt to move more air throughout the respiratory tract.[5,11]

In addition to respiratory disease, dogs with BOAS may have gastrointestinal clinical signs such as vomiting, regurgitation, subsequent aspiration pneumonia, and diarrhea. While the exact pathophysiology is still being studied, it is proposed that excessive negative pressure caused by

increased airflow resistance may cause reflux and aerophagia. French Bulldogs have been reported to have the highest prevalence of GI signs when compared to other brachycephalic breeds across multiple studies. When surgical correction is performed, there is an improvement in gastrointestinal signs regardless of breed.[8]

Baseline bloodwork, including CBC, chemistry, and blood gas analysis (or SpO2) should be performed on animals presenting with BOAS. When compared to non-brachycephalic dogs, dogs with BOAS had a consistently lower SpO2 and were at risk for hypoxemia.[1] Due to the obstructive nature, respiratory acidosis is not uncommon.[1] Interestingly, dogs with BOAS were found to be hypercoagulable on thromboelastography (TEG) when compared to non-brachycephalics in a study that compared the disease to sleep apnea in humans.[3] While not all veterinary hospitals have TEG available, it may be a useful tool in guiding treatment for BOAS animals.

Treatment Options

Treatment for BOAS consists of surgical correction that is dependent on the individual's components and severity at presentation. Multiple anatomical components are typically present in patients, requiring multiple procedures at the time of surgery.[5] As stated previously, elongated soft palate is the most frequent finding.[5] Staphylectomy, or soft palate resection, is commonly performed with either scissors or laser.[6] It is imperative that the proper amount of soft palate be removed to prevent further respiratory complications, such as aspiration pneumonia.[6] It should be excised so that 1-2mm of soft palate extends over the epiglottis.[6] The nares may be resected with a #11 scalpel blade in a variety of ways, including vertical, horizontal, or dorsolateral wedge resections, with all techniques resulting in a noticeably wider nostril.[6] Laryngeal sacculectomy can also be performed but may carry a higher risk of post-operative swelling than

the other procedures.[6] Complication rates following BOAS surgery have been reported around 12% regardless of which procedure(s) was performed,[4,5] but can depend on the dog's clinical status at the time of surgery. There are many studies demonstrating the benefits of surgery outweigh the risks, as up to 90% of dogs experience clinical improvement and thus a better quality of life.[4,5] If significant swelling and edema occurs post-operatively, the airway will need to be secured.

Fin presented in severe respiratory distress, necessitating stabilization via tracheostomy prior to correcting his anatomical abnormalities. Tracheostomy tubes serve as an airway when obstruction is severe and normal respiration is diminished.[6,9] To facilitate tube placement, patients are ideally anesthetized and pre-oxygenated, but intubation is not always possible with extreme airway swelling. In dorsal recumbency, the ventral neck is shaved and sterilely prepped with surgical scrub solutions. Caudal to the larynx, an approximately 5cm skin incision is made over the midline of the trachea. Blunt dissection is performed until the sternohyoid muscles are visualized and can be separated. Gelpi Retractors are used in order to further visualize the trachea under the musculature. Two stay sutures are placed cranial and caudal to the tracheal ring that will be incised (between the 3rd and 5th rings). A horizontal incision is made in the annular ligament with a #15 scalpel blade between the two stay sutures for tracheostomy tube placement. The tracheostomy tube size and type (single vs double-lumen) is chosen based on the patient's tracheal diameter (up to 50%) and surgeon preference, respectively. After insertion of the tube, it is secured behind the patient's neck to avoid dislodgment. Any excess skin cranial and caudal to the site is sutured.[6]

Tracheostomy, like corrective BOAS surgery, is not free of complications. The most common complications in one study included pneumomediastinum, followed by tracheostomy

tube obstruction, dislocation, aspiration pneumonia, and edema of the tracheostomy site. In the same study, Bulldogs appeared to have higher complication rates and more severe complications that led to higher euthanasia and death rates than other breeds. Overall, surgeons should expect high complication rates from tracheostomy and must stress good nursing care and in-hospital management to increase success rates.[9]

Case Progression and Outcome

Brachycephalic obstructive airway syndrome can affect dogs of any age, but studies have described the mean age at presentation between two-three years old.[5] Fin's presentation was interesting as he was geriatric and had no prior history of respiratory distress, despite being active throughout his life. Twenty-four hours after stabilization via tracheostomy, initial diagnostics were performed. Fin was able to oxygenate appropriately during the work-up and his first tracheostomy tube change occurred later that afternoon. He remained on dexamethasone SP at 0.14mg/kg intravenously once a day to reduce airway swelling with the tentative plan to go to surgery for correction of his brachycephalic airway obstructive syndrome.

Fin continued to do well with tracheostomy tube cleanings every four hours and daily tube replacements until May 30. That day, mucous production at the site increased and occluded his tube, which is a known complication of tracheostomies.[9] This occurs due to a well-documented change in the mucosa following surgical placement.[10] Fin's occlusion became apparent when he displayed labored breathing, tachypnea, and an episode of collapse. After recovering from the acute distress episode, Fin's tube changes were increased to every 12 hours with an emphasis on nursing care in between changes, including nebulization, coupage, and proper suctioning of the tube.

On June 1, six days after initial presentation, Fin had another episode of labored breathing and pale mucous membranes. He was sedated with propofol and intubated to re-evaluate his airway, which revealed static edema and inflammation. His tracheostomy tube changes were increased to every 6 hours prior to surgery. On day 7, Fin underwent BOAS surgical correction which included a staphylectomy, laryngeal sacculectomy, and rhinoplasty. Additionally, a sample of the epiglottis was collected and submitted for biopsy, which revealed inflammation. He recovered uneventfully from surgery. Post-operatively, Fin experienced mild, serous nasal discharge, which was to be expected. On day 9, the nasal discharge became mucopurulent and he began having increased upper airway sounds, which prompted the need for thoracic radiographs. At that time, there was no radiographic evidence of aspiration pneumonia, but Fin was started on a course of Unasyn at 30 mg/kg every eight hours for broad-spectrum bacterial coverage.

For the first 9 days in hospital, Fin was hyporexic to anorexic, but did not display signs of GI upset. On day 10, Fin vomited after morning treatments and was subsequently started on 1 mg/kg of maropitant and 1 mg/kg of pantoprazole intravenously. His appetite improved the following evening and he ate a few “meatballs” of food. He seemed well overall until day 13 (June 8). At that time, increased respiratory effort and rates were noticed. He was placed back in the oxygen cage and repeat thoracic radiographs were taken later that morning. Radiographic findings consisted of a severe, mixed interstitial to alveolar pulmonary pattern in the caudodorsal lung lobes. Differentials included aspiration pneumonia, bacterial pneumonia, or noncardiogenic pulmonary edema. Due to Fin’s recent history of tracheostomy tube occlusions and BAS surgery, aspiration pneumonia was presumed. He had periods of respiratory concern throughout the next several days that were controlled with supplemental oxygen. Terbutaline and enrofloxacin were

added to his treatment plan at 0.01 mg/kg subcutaneously every 8 hours and 10 mg/kg intravenously every 24 hours, respectively.

Thoracic radiographs were repeated again on day 16 (June 11) to monitor progression of Fin's disease and showed worsening in the right caudal and accessory lung lobe and improvement in the other lung lobes. Although he had severe radiographic evidence of disease, he appeared comfortable when challenged without the tube for small periods of time on day 17. Unfortunately, on day 18, Fin relapsed once again and had an episode of respiratory distress and lethargy. Recheck thoracic radiographs were performed again and showed unchanged pneumonia. Furosemide was added to his treatment plan at 2 mg/kg intravenously every eight hours and intravenous fluids were discontinued. Twenty-four hours after administering furosemide, another set of radiographs were taken, which remained unchanged. At that time, terbutaline was removed from his treatment regimen. Two days later (day 21), Fin appeared very stable and had radiographic resolution of aspiration pneumonia. His tracheostomy tube was removed and the incision was left open to heal by second intention. Fin was discharged on June 19, 2020 on enrofloxacin, Clavamox, and furosemide with instructions to return for a recheck appointment in five days. His owners were advised to feed only canned food in small amounts and confine Fin for two weeks to allow proper healing of the tracheostomy site and prevent regurgitation and subsequent aspiration.

Fin spent a total of 24 days in the hospital. His first tracheostomy challenge (removal of the tracheostomy tube and observation for adequate oxygenation) occurred on day 9, but his tube was not successfully removed until day 21. He failed many tracheostomy challenges and had several episodes of respiratory distress during his stay due to tube obstruction and secondary aspiration pneumonia, which is also common following permanent tracheostomies.[10] It is not

surprising Fin experienced complications during his hospital stay, as complications have been reported in approximately 90% of patients with temporary tracheostomy tubes, ranging in severity from minor to life-threatening.[9]

Fin had three recheck appointments to ensure his tracheostomy site was healing appropriately and monitor radiographic signs of pneumonia, with the final occurring on June 27, 2020. At that time, Fin was apparently healthy and had no signs of aspiration pneumonia. His tracheostomy site healed without complications and he was doing well at home.

Social Media Implications

Recently, brachycephalic obstructive airway syndrome has become a hot topic as the United Kingdom Kennel Club updated their breed standards for French Bulldogs in December 2021. The revision was a result of the Kennel Club's concern for the health and future of the breed due to increased breeding of these dogs in the United Kingdom. Specifically, the reformed breed standards called for enlargement of external nares and longer snouts. These guidelines aim to reduce future incidence of complications of BOAS that were previously discussed in this paper, including respiratory distress and heat stroke.[12] It is important to note that these changes were publicly supported by many veterinarians and veterinary surgeons. These doctors serve as exemplary models and should inspire other veterinary professionals to advocate for improved animal welfare standards in regard to responsible breeding.

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