Vocal cord dysfunction: A case report

LCDR Erik C. Cline, CRNA, MSN, NC, USN
LCDR Roger Davis, CRNA, MSN, NC, USN
Okinawa, Japan

CDR Joseph F. Burkard, CRNA, DNSc, NC, USN
San Diego, California

Vocal cord dysfunction (VCD) is a respiratory condition characterized by paradoxical closure of the vocal cords. This condition results in a myriad of symptoms that would be expected from an upper airway obstruction including anxiety, hyperventilation, wheezing, stridor, shortness of breath, dyspnea, and suprasternal and neck muscle retraction. The present case report describes a 30-year-old woman with known VCD who underwent local anesthesia with intravenous sedation for perianal skin tag removal. Postoperatively, the patient experienced respiratory distress, prompting interventions and investigation. A review of the literature revealed limited information on VCD, and no anesthesia literature was found regarding this entity.

Key words: Airflow, airway limitation, asthma, vocal cord.

Vocal cord dysfunction (VCD) is a respiratory condition characterized by paradoxical closure of the vocal cords. In normal breathing, the vocal cords are alternatively activated such that they are abducted during inspiration to decrease airway resistance and slightly adducted during expiration to limit expiratory flow rates and prevent alveolar collapse. Although occasionally involving the expiratory phase, VCD is manifested most commonly by abnormal narrowing of the vocal cords during inspiration, resulting in an airflow limitation at the level of the larynx. This condition results in a myriad of symptoms that would be expected from an upper airway obstruction including anxiety, hyperventilation, wheezing, stridor, shortness of breath, dyspnea, and suprasternal and neck muscle retraction. Interestingly, despite the apparent respiratory distress, affected patients rarely are hypoxic or appear cyanotic. Fortunately, such potentially life-threatening breathing problems lead to a difficult differential diagnosis.

Vocal cord dysfunction is a condition that closely resembles asthma, making the 2 entities difficult to differentiate symptomatically. Its misdiagnosis often leads to inappropriate treatment, and, often, patients have been treated unsuccessfully with high-dose corticosteroids and bronchodilators that have led to iatrogenic side effects. Occasionally, acute manifestations lead to intubation or tracheotomy for upper airway obstruction. These interventions have, in turn, led to increased length of hospitalization and psychological and behavioral dysfunction. Diagnosis rests with high clinical suspicion and direct observation. The “gold standard” for the diagnosis of VCD is laryngoscopy with direct visualization of the paradoxical motion of the vocal cords when the patient is symptomatic.

The following case report describes a 30-year-old woman with known VCD who underwent local anesthesia with intravenous (IV) sedation for perianal skin tag removal. Postoperatively, the patient experienced respiratory distress, prompting interventions and investigation. A review of the literature revealed limited information on VCD, and we found no anesthesia literature regarding this entity.

Case report

A 30-year-old, 63-kg woman was admitted to the operating room for perianal skin tag excision. The patient was classified as ASA physical status II for a medical history that included asthma and VCD. Medications included fluticasone, 100 mg, salmeterol, 50 µg, inhalation powder (Advair) daily, and albuterol metered-dose inhaler as needed. The diagnosis of asthma was made 2 years earlier, following a 5-day intensive care unit admission for pneumonia during which the patient was intubated.

The patient provided informed consent for an anesthesia plan that included a subarachnoid block to anesthetize the sacral roots. The preprocedure vital signs were as follows: blood pressure, 120/66 mm Hg; heart rate, 60 beats per minute; respiratory rate, 14 breaths per minute; temperature, 98.7°F; and SpO2, 98% while breathing room air. Physical examination findings were unremarkable; the airway examination revealed a Mallampati 2, a thyromental distance of finger breadths, and full neck range of motion.

The patient was transported to the main operating room, standard monitors were applied, and she was placed in the sitting position for the subarachnoid block. The patient was premedicated with 2 mg of midazolam IV. Multiple attempts at placing the block were unsuccessful, and after consultation with the
surgeon, the anesthetic plan was changed to local anesthesia with IV sedation. The patient then was placed in the prone position, and an additional 2 mg of midazolam was given before placement of 9 mL of 1% plain lidocaine to the surgical site by the surgeon. The patient also received 25 µg of fentanyl IV and 20 mg of propofol IV. The total surgical time was 4 minutes. The patient then was assisted to the supine position and transferred to the hold area as a “fast track” patient for direct admission to our facility’s phase 2 recovery area.

While in the hold area, the patient had increased work of breathing with audible stridor, tracheal tugging, and accessory muscle use. The decision was made to transport the patient to the postanesthesia care unit (PACU) for further evaluation and stabilization of her condition. On arrival in the PACU the patient’s vital signs were as follows: blood pressure, 104/50 mm Hg; heart rate, 91 beats per minute; respiratory rate, 16 breaths per minute; and Spo2, 100%. Audible stridor was still present with lower respiratory wheezing.

The patient received a 5-mg dose of albuterol via a handheld nebulizer. The patient’s vital signs remained stable with an unchanged Spo2 value. The patient then was medicated with a total of 6 mg of midazolam during a 10-minute period with a concurrent nebulized dose of racemic epinephrine. Between nebulizer treatments, the patient received assisted positive-pressure ventilation via a Jackson Reese circuit. After medication with 4 mg of midazolam IV in the PACU, the patient’s objective level of anxiety was decreased, but lower airway wheezing persisted, so the decision was made to administer ketamine, 30 mg IV. Ketamine was chosen primarily for its bronchodilator properties.

The patient’s condition remained hemodynamically stable throughout the preceding interventions. At this point, a consultation was requested of the pulmonary medicine specialist who reassessed the patient and initiated guided imagery (a form of relaxation therapy) and pursed-lip breathing, which resulted in resolution of symptoms. The patient was transported to the phase 2 recovery area and discharged to home. The total PACU stay was 2 hours.

Discussion

The overall prevalence of VCD in the population is unknown. It affects all ages and sexes; however, the literature reveals that VCD is diagnosed predominantly in white women between the ages of 20 and 40 years with an increased body mass index.6-8 Vocal cord dysfunction can coexist with asthma. Typically, VCD is misdiagnosed as exercise-induced asthma and refractory to treatment. The National Jewish Center for Immunology and Respiratory Medicine found that of patients referred for treatment of refractory asthma, 30% had VCD and asthma and 10% had VCD alone.6

A subsequent retrospective study at the National Jewish Center found asthma to coexist with VCD in 56% of 95 patients diagnosed with VCD.5

Because VCD is found in asthmatics and mimics the symptoms found in patients with only asthma, it often is overlooked and can remain undiagnosed for 5 to 10 years.9 In the previously mentioned study at the National Jewish Center, in the year before their diagnostic admissions, the group of 95 patients diagnosed with VCD had an average of 9.7 emergency department visits and 5.9 admissions; 28% of these patients were intubated.1 Patients with VCD tend to seek care frequently for recurring symptoms before the correct diagnosis is made.

The cause of VCD is unknown, but there are several triggers, including exercise, environmental irritants, gastroesophageal reflux disease (GERD), rhinitis, and conversion disorder. There seems to be a large psychological component to VCD.1,4-8 In an early retrospective study, 73% of patients with VCD had a major psychiatric disorder, 38% had a history of abuse, and 37% had a personality disorder.1 Many investigators believe that VCD is a conversion reaction rooted in a variety of psychiatric conditions, including abuse, depression, emotional and, possibly, physical stress.3,5 Conversion disorders, although not intentional, are characterized by repressed emotional conflicts that are converted to physical symptoms with no underlying organic cause. Although the influence of gender is speculative, VCD predominantly affects females, and this influence of gender is consistent with the observed increase of somatoform disorders, conversion disorders, and anxiety among female patients. Psychogenic causes are supported by the fact that symptoms subside with reassurance, support, distraction, and placebo and the fact that patients cannot produce the abnormal laryngeal movements voluntarily.5,9

However, several studies found little evidence to support conversion disorder in patients with VCD because depression and somatization were frequent findings in patients with VCD and patients with asthma. Due to the retrospective nature of many of these studies, the number of patients in whom psychological problems developed after the onset of respiratory symptoms is unknown. The chronic, unpredictable nature and failure to respond to treatment can lead to frustration and increased emotional distress that can contribute to depression and anxiety.5,10

More recent studies have suggested alternative trig-
gers to VCD. It has been documented that irritant exposure may lead to laryngeal dysfunction or VCD. A temporal relationship between the onset of VCD and occupational or environmental exposure to irritants such as ammonia, solvent vapors, cleaning solutions, and smoke was found. This temporal association suggests a direct inflammatory effect of the irritant on the vocal cords. The same study found the incidence of GERD to be much higher in the VCD population than in the general community owing to its role in the irritating effects of refluxing gastric contents on the vocal cords. Canine models have shown GERD to induce laryngeal spasm and sensitize mucosal chemoreceptors. The pathophysiology of irritant-induced VCD also remains unknown, although it has led to several interesting hypotheses, most of which seem to center on a theory of laryngeal hyperresponsiveness. One such hypothesis states that an initial inflammatory insult, possibly caused by antigens in the laryngeal mucosa, results in a persistent autonomic imbalance in which subsequent stimuli, such as cold air or psychological stressors, initiate local parasympathetic reflexes, causing vocal cord narrowing. Another hypothesis states that direct stimulation of nociceptive or olfactory nerve endings within the respiratory tract may initiate higher center processing or local reflex arcs that lead to paradoxical vocal cord closure. Short- or long-term irritant inhalation causes an alteration in the number and/or regulation of sensory nerve endings, leading to a lowering of their threshold for stimulating the glottic closure reflex.

The salient point of irritant-induced VCD seems to be that chronic irritation of the larynx leads to increased laryngeal sensitivity, predisposing to VCD attacks by various external stimuli. In support of the hypothesis of a local reflex arc, a recent series of studies was done with the use first of humans and then of cats. The study results indicated that glottic narrowing has 2 mechanisms. In the active mechanism, the adductor is activated predominantly over the abductor, resulting in glottic narrowing. In the passive mechanism, air flowing through the narrowed glottic opening increases in velocity, resulting in a decrease in static pressure (the Bernoulli principle), which further narrows the glottic opening. This narrowed glottic aperture increases internal resistance for breathing, generating subglottic negative pressure. Interestingly, the abnormal inspiratory activity of the adductor is almost entirely abolished by relief of this subglottic negative pressure via a tracheostoma. These results indicate that the unusual adductor activity is triggered by a laryngeal reflex arc evoked by potent negative pressure in the subglottic space or by several phenomena attributable to it such as vocal cord vibration, stretch, or distortion. Vocal cord dysfunction seems to form a spectrum of disease with causes that are psychological and physiological.

The diagnosis of VCD is difficult due to its unpredictable character and manifestations. It often is suspected when the clinical picture is inconsistent with that of asthma. Generally, patients with VCD lack sputum production and nocturnal symptoms and have localization of wheezing to the throat and upper part of the chest. In addition, they have no significant abnormalities in blood gases, electrocardiograms, echocardiography, chest radiographs, pulmonary artery pressures, and alveolar-arterial oxygen differences. Because of its unpredictable behavior, it is often necessary to attempt to induce VCD with bronchial challenges such as exercise, histamine, or metacholine.

Current criteria for diagnosis include a posterior “chinking” of the vocal cords during inspiration, early expiration, or both. Posterior chinking is a small diamond-shaped opening at the posterior portion of the vocal cords. Flow-volume loops also have been used to support the diagnosis of VCD. When patients with VCD are symptomatic, they have an abnormal flattening of the inspiratory limb of the flow-volume loop that is consistent with an extrathoracic obstruction. Patients with VCD have this inspiratory truncation of the flow-volume loop 70% of the time during bronchoprovocation testing. To make the diagnosis of VCD, there must be knowledge of its existence and direct visualization of the vocal cords while patients experience symptoms.

Management of VCD differs for the acute and chronic phases. Acute episodes of VCD must be managed in a calm, reassuring manner. Measures found helpful for controlling acute attacks include placing the patient in the “sniff” position; having the patient pant, which widens the glottic aperture; diaphragmatic breathing; and asking the patient to make a soft “s” sound, which diverts their attention from inspiration to expiration. Other measures to treat acute episodes include oxygen; heliox (helium/oxygen, 80:20 or 70:30), which is less dense than air, resulting in decreased turbulence across the narrowed glottis; intermittent positive pressure, and continuous positive airway pressure. Finally, the use of sedatives, anxiolytics, small doses of propofol, and topical lidocaine applied to the larynx have been effective in resolving an acute VCD attack. In severe cases, treatments have included sectioning of the laryngeal nerve with subsequent ipsilateral paralysis of the vocal cord...
and the intralaryngeal injection of botulinum toxin type A, which provides symptomatic relief for several weeks and can be repeated. 6,12,14

The management of chronic VCD requires a multi-disciplinary approach. This approach attempts to educate the patient about the recognition and prevention of a VCD attack. Speech therapy and psychologic counseling are the cornerstones of this management. Psychologic counseling attempts to prevent future attacks by reducing stress or treating an underlying conflict that may lead to a future attack, whereas speech therapy trains the patient to recognize the symptoms of VCD and apply various breathing techniques that help resolve the symptoms. Other treatments successfully used include relaxation training, biofeedback, and self-hypnosis. 4,6,7

Conclusion
Vocal cord dysfunction presents unique challenges to anesthetists, particularly in an emergency situation. If the patient has a known history of VCD, it must be considered in the differential diagnosis, and its unique treatment modalities must be implemented to avoid increased morbidity and possible mortality.

REFERENCES

AUTHORS
LCDR Erik C. Cline, CRNA, MSN, NC, USN, is a staff CRNA in the anesthesia department, US Naval Hospital, Okinawa, Japan.
LCDR Roger Davis, CRNA, MSN, NC, USN, is a staff CRNA in the anesthesia department, US Naval Hospital, Okinawa, Japan.
CDR Joseph Burkard, CRNA, DNSc, NC, USN, is a clinical research coordinator, Navy Nurse Corps Anesthesia Program, San Diego, Calif.

DISCLAIMER
The views expressed in this article are those of the authors and do not reflect the official policy or position of the Department of Defense or the United States Government.