

Rapid communication

Vocal cord dysfunction with nocturnal awakening

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Vocal cord dysfunction (VCD) is an involuntary disorder caused by paradoxical movement of the true and false vocal cords. It has been well documented to masquerade as disorders of both the upper and lower respiratory tract, most frequently asthma.¹ Historically, one of the key differentiating factors between asthma and VCD has been the absence of nocturnal awakening with VCD.² We report on four patients with VCD who experienced nocturnal awakening as part of their symptom complex.

We believe that the presence of nocturnal awakening does not reliably differentiate between these two groups.

CASE 1

N.B. was a 40-year-old woman with a history of perennial rhinitis since childhood with seasonal exacerbation, particularly in the summer and fall, who was treated with antihistamines and cromolyn nasal solution. She was diagnosed with "mild" asthma at age 25 and treated with albuterol and cromolyn sodium metered-dose inhalers (MDIs) on an as-needed basis. Two years before evaluation, she was exposed to paint and glue in her office, and since then, had noted a marked exacerbation in her asthma symptoms. Asthma symptoms consisted of cough, shortness of breath, chest and throat tightness, difficulty breathing both on inspiration and exhalation, voice changes, wheezing, and significant decrease in her effort tolerance. She experienced frequent nocturnal awakening caused by these symptoms. There was no significant improvement in her condition with administration of β_2 -agonists. The patient had required evaluation for acute asthma exacerbations, approximately eight times per year in the preceding 2 years, by either an emergency department physician or her gen-

Abbreviations used

MDI: Metered-dose inhaler

VCD: Vocal cord dysfunction

eral practitioner. These exacerbations were usually treated with an oral steroid burst, increased use of β_2 -agonists, and inhaled steroids and oral antibiotics. Triggers included an exacerbation in her rhinitis symptoms, exposure to car exhaust, and exercise. The patient did not have symptoms suggestive of gastroesophageal reflux disease.

Results of physical examination were significant for nasal mucosal edema and erythema bilaterally with tenderness to percussion of the frontal and maxillary sinuses bilaterally. The rest of the results of the physical examination were normal; however, there was a mildly obstructed flow-volume loop, with the FEV₁ and forced vital capacity being approximately 75% of predicted value. The inspiratory portion of the flow-volume loop was normal.

Skin prick tests could not be performed because of the use of astemizole. Nasal saline irrigation and administration of beclomethasone dipropionate nasal spray twice a day were instituted, resulting in some decrease in her rhinitis symptoms. There was no reduction in her pulmonary symptoms with the use of inhaled β_2 -agonists. The result of a methacholine challenge was negative at 25 mg/ml (Fig. 1). This was followed by an exercise test, the result of which was also negative. Approximately 15 minutes after the exercise test, she experienced throat and chest tightness, voice changes, and mild stridor. Her FEV₁ values fluctuated by 20% from baseline and then returned to normal. Rhinolaryngoscopy revealed marked adduction of the true vocal cords on inspiration (Fig. 2). She was treated with speech therapy, psychosocial consultation, and nasal saline irrigation followed by beclomethasone dipropionate nasal spray twice a day and has experienced significant reduction in her symptoms at 9 months' follow-up.

CASE 2

J.T. was a 60-year-old woman who had no significant medical history until 5 years before evaluation, when she experienced an upper respiratory tract infection followed by perennial rhinitis and increasing shortness of breath with progressive dyspnea on exertion. At the time

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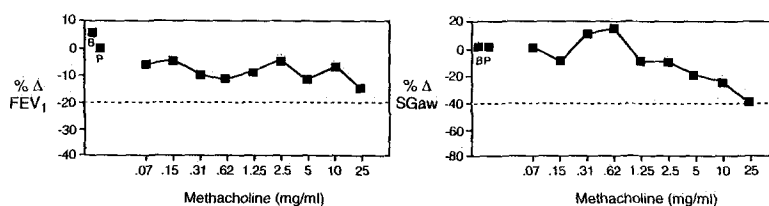


FIG. 1. Negative methacholine challenge response at 25 mg/ml.

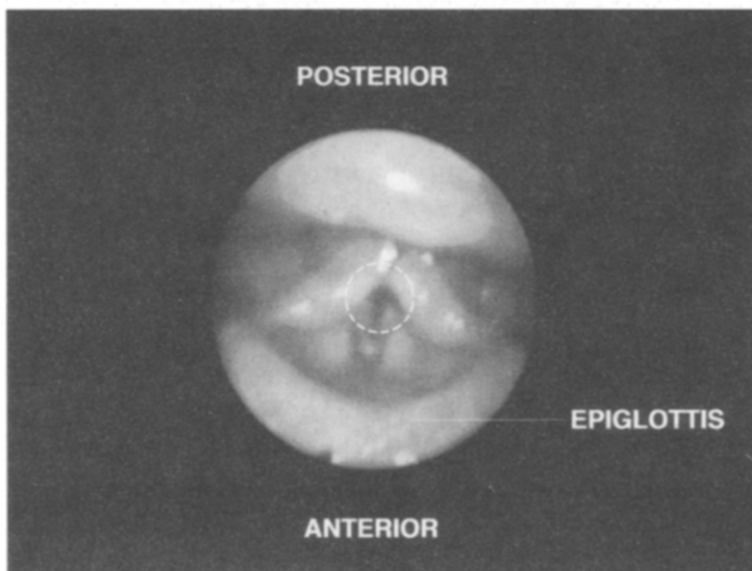


FIG. 2. Rhinolaryngoscopy demonstrating marked adduction with classic diamond-shaped posterior "chinking" of true vocal cords (dashed circle).

of evaluation, her effort tolerance was limited to 100 feet. Her symptom complex consisted of shortness of breath, chest tightness, persistent cough, and throat clearing with nocturnal awakening caused by both shortness of breath and cough. The patient's symptoms were not controlled by inhaled steroids and β_2 -agonists. Of note, the onset of her symptoms coincided with significant stress within her marriage. The patient did not have symptoms suggestive of gastroesophageal reflux disease. Results of physical examination were negative except for mildly obstructed spirometry with a truncated inspiratory portion of her flow-volume loop (Fig. 3). The result of a methacholine challenge was negative at 25 mg/ml. However, the patient was experiencing symptoms, and rhinolaryngoscopy revealed marked adduction of the vocal cords both on inspiration and on expiration (i.e., biphasic VCD). She has undergone speech therapy and psychosocial consultation with an excellent response at 1-year follow-up.

CASE 3

A.S. was a 24-year-old man who experienced his first episode of shortness of breath after a soccer game at age 20. Symptoms resolved spontaneously but recurred 1 week later under similar circumstances. He was evalu-

ated by his primary care physician, and a diagnosis of exercise-induced bronchospasm was made on clinical grounds. He was treated with albuterol MDI, 2 puffs, before exercise. Over the past 4 years, his symptoms had waxed and waned and tended to be exacerbated by increasing stress. His symptoms were markedly exacerbated over the month before evaluation at this institution. Psychosocial stresses included the impending purchase of a house, recent marriage, and job loss. Symptoms consisted of shortness of breath on inspiration and on expiration, chest tightness, wheezing, and nocturnal awakening caused by these symptoms. Symptoms would occur suddenly with no obvious triggers. The patient noted a poor response to albuterol MDI and required 2 puffs every 2 hours.

Results of physical examination were normal except for a truncated inspiratory flow-volume loop. The patient underwent a methacholine challenge, the result of which was positive at 0.371 mg/ml. However, the FEV₁/forced vital capacity ratio never changed. Rhinolaryngoscopy revealed adduction of the vocal cords both on inspiration and on expiration. Of note, his spirometry returned to normal immediately after this test without medication. He has undergone speech therapy and psychosocial consultation with an excellent response. He

underwent a repeat methacholine challenge 9 months later, and the result was positive at 1.15 mg/ml. Results of repeat rhinolaryngoscopy were negative. In this patient one cannot rule out asthma on the basis of these data. However, he has not required β^2 -agonists for 9 months subsequent to the interventions described above, and a repeat methacholine challenge demonstrated no significant change in his baseline airway reactivity.

CASE 4

J.J. was a 38-year-old woman who had a childhood history of perennial asthma. Her parents moved from New York to Arizona when she was 10 years old, and she experienced a marked reduction in asthma symptoms. She still had mild exercise-induced bronchospasm, which would respond to as-needed administration of Bronkaid. At age 19 she moved to Denver, and her asthma symptoms remained quiescent, requiring very infrequent use of Bronkaid. At age 33 she was having marital problems and began experiencing flashbacks related to early childhood sexual abuse issues. This was immediately followed by the onset of shortness of breath, neck tightness, difficulty breathing both on inspiration and expiration, and voice changes. She was admitted to a psychiatric hospital overnight, and her symptoms resolved spontaneously. Her symptoms had progressively worsened, occurring more frequently with frequent nocturnal awakening caused by shortness of breath. Three months before evaluation at National Jewish Center for Immunology and Respiratory Medicine, she was seen by her physician and was prescribed albuterol MDI (2 puffs four times daily), theophylline, and prednisone but experienced no improvement in her condition. She was referred to this institution for further treatment.

Further history was significant for the onset of rhinitis symptoms approximately 5 years earlier. Her symptoms were perennial, exacerbated in the spring months, and consisted mainly of nasal congestion. She had no symptoms suggestive of gastroesophageal reflux disease. Her family history was significant for paternal asthma, and an older sister had asthma and rhinitis.

Review of prior office data was significant for a flow-volume loop with a truncated inspiratory portion. Results of physical examination were normal except for minimal nasal mucosal edema bilaterally. Home peak flows showed no significant variability, even when she was experiencing symptoms. Screening computed tomography of the sinuses demonstrated mild chronic ethmoid sinusitis. The result of a methacholine challenge was positive with a PC_{20} FEV_1 of 0.429 mg/ml. Rhinolaryngoscopy was significant for marked adduction of the true vocal cords on inspiration.

The patient was initially treated with inhaled triamcinolone, 2 puffs four times daily, as-needed albuterol (for symptoms with a 20% decrease in home peak flows), nasal saline irrigation, and beclomethasone dipropionate nasal spray twice a day. In addition, she underwent extensive psychosocial counseling and speech therapy. At follow-up 2 weeks later, she had marked reduction in

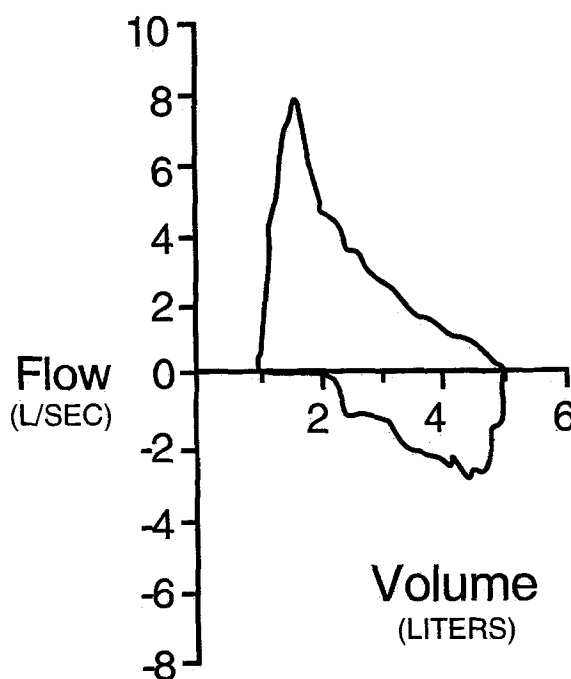


FIG. 3. Abnormal flow-volume loop demonstrating a truncated inspiratory portion.

her symptoms, she was able to abort the episodes of shortness of breath with various breathing maneuvers, and she had not required albuterol. Her nasal symptoms were also decreasing. A slow taper of inhaled triamcinolone was outlined. At follow-up 6 weeks later, she had stopped all asthma medications of her own accord without recurrence of symptoms. At follow-up 10 months later, she continued to abstain from taking all her asthma medications, was performing daily nasal saline irrigations, and was using beclomethasone dipropionate nasal spray on an as-needed basis. She was able to control all episodes of breathing difficulties with breathing maneuvers. Her nocturnal awakening had resolved completely.

Although one cannot rule out asthma in this patient, she has responded well to specific management of her VCD with no specific asthma interventions.

DISCUSSION

VCD has been well documented to masquerade as disorders of both the upper and lower respiratory tract, most frequently asthma. VCD that is first seen in this manner may be inadvertently treated with high-dose oral steroids, β_2 -agonists, and inhaled steroids. It usually does not respond significantly to these medications,³ and there is a substantial risk of iatrogenic complications. Unfortunately, these two diseases frequently coexist, and differentiation of VCD from asthma on the basis of history and physical findings is unreliable.

Kellman and Leopold² noted the absence of stridor in one of their hospitalized patients during sleep. Of note, she had been successfully treated earlier that day with

oral diazepam. No mention is made of the method used to observe this patient or whether she was monitored continuously during that one observation. There were no follow-up observations.

Other authors have noted the disappearance of VCD with induction of general anesthesia⁴ or with the use of benzodiazepam or morphine.⁵ To our knowledge, there is one prior report of nocturnal awakening caused by isolated VCD in the literature.⁶

In this case series, two of our patients had isolated VCD (negative methacholine challenge response), and two of our patients had VCD with reactive airways disease. However, the two with concomitant reactive airways disease responded to specific treatment of their VCD and stopped all asthma medication with no recurrence of their symptoms during 9 months' follow-up. We

conclude that the presence of nocturnal awakening does not reliably exclude the diagnosis of VCD.

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