pulmonary and critical care pearls

Recurrent Asthma Despite Corticosteroid Therapy in a 35-Year-Old Woman

Clay B. Marsh, M.D.; Michael D. Trudeau, Ph.D.; and Jeffrey E. Weiland, M.D., F.C.C.P.

A 33-year-old woman with a history of asthma presented with shortness of breath and voice loss. Her asthma began 4 years previously, after an inhalational exposure to irritating fumes. She subsequently experienced frequent wheezing associated with voice loss when exposed to dust or chemical fumes despite preventive therapy with bronchodilators and corticosteroids. The patient denied a family history of lung disease, respiratory problems as a child or young adult, or psychiatric illnesses; however, she had felt "down" since developing her work-related pulmonary condition. Her lung function tests between asthma episodes were always normal. Current medications included theophylline, inhaled albuterol, and prednisone (20 mg/d). She did not smoke and denied use of illicit drugs.

Physical Examination

Vital signs: temperature, 99°F; pulse, 88 beats/min; respirations, 18/min; BP, 115/65 mm Hg without paradox. General: anxious-appearing woman


Laboratory Findings

Hematocrit, 38 percent; WBC, 7,200/µl; electrolytes, normal; O₂ saturation (pulse oximeter, room air), 94 percent. Chest radiograph: normal. Pulmonary function tests were performed (Fig 1).

What diagnostic test would you perform next?
Diagnosis: Fiberoptic laryngoscopy demonstrated inspiratory adduction of the vocal cords, confirming the presence of acute vocal cord dysfunction

Patients with shortness of breath and wheezing present a differential diagnosis that includes several conditions that can simulate bronchial asthma. It is important to accurately diagnose nonasthmatic causes of wheezing in order to avoid complications of long-term corticosteroid therapy inappropriately prescribed for a misdiagnosis of intractable asthma.

Acute vocal cord dysfunction is one of the conditions that can mimic recurrent asthma exacerbations. Variously termed episodic laryngeal dyskinesia, Münchhausen's stridor, factitious asthma, vocal cord dysfunction, and paradoxical vocal cord motion, this disorder produces intermittent upper airway obstruction when the vocal cords adduct at inappropriate times during the inspiratory and expiratory respiratory cycles. Because of this inappropriate adduction, patients have only a small glottic "chink" through which airflow occurs. The resulting increase in airway resistance causes progressive dyspnea, wheezing, loss of voice, and tachypnea.

In addition to its role in generating speech, the glottis normally participates in respiration by varying its caliber to adjust airflow, thereby regulating the duration of inspiration and expiration and the size of end-expiratory lung volumes. Phasic adduction of the true vocal cords is an important element of this regulatory mechanism. Dysfunction of the timing of vocal cord adduction results in glottic narrowing or intermittent glottic closure independent of fluctuations of lower airway caliber. Although the obstruction is located in the upper airway within the glottis, the limitation of airflow in this condition produces wheezing similar in pitch to that in asthma; however, the wheezing is dissimilar in timing, being equally evident during inspiration and expiration.

Although the etiology of vocal cord dysfunction is unknown, both a speech pathology basis and a psychiatric basis have been invoked. Some affected patients receive a secondary gain from their respiratory disability; a high incidence of affective disorders, such as depression and employment in paramedical fields, has also been noted in this patient population.

Acute vocal cord dysfunction occurs most commonly in women who are 20 to 40 years old. The diagnosis is suggested when the severity of dyspnea appears out of proportion to the observed physical or laboratory findings and no response to aggressive asthma therapy occurs. The presence of wheezing during inspiration and expiration that is heard best over the larynx with poor transmission to the chest wall further supports the diagnosis. Arterial blood gases usually display a normal alveolar-arterial oxygen gradient. Pulmonary function tests often detect poor reproducibility on spirometry and pronounced variability of inspiratory flow loops, often with a characteristic oscillating baseline (Fig 1). A flow-volume loop may show variable extrathoracic airway obstruction with or without additional expiratory obstruction during an acute attack. Chest radiographs are normal, in contrast to the hyperinflated lung volumes often demonstrated by patients with an acute exacerbation of asthma.

The diagnosis of vocal cord dysfunction is confirmed by laryngoscopy during an acute episode, which demonstrates almost complete adduction of the vocal cords throughout the respiratory cycle. Narrowing of the glottis with a small posterior chink is frequently observed; the chink serves as the patient's airway during symptomatic episodes. Patient sedation, inhalation of a mixture of 80 percent helium and 20 percent oxygen, positive-pressure face-mask ventilation, or reassurance may terminate an episode. When the patient is examined laryngoscopically between episodes of vocal cord dysfunction, glottic function appears normal. With the use of video stroboscopy (examination with a fiberoptic laryngoscope with strobe-light capability to track high-frequency vocal cord motion), abnormalities in inspiratory glottic tone may be seen after different phonation exercises. This finding may help suggest the diagnosis in the absence of a symptomatic exacerbation.

Treatment involves a multidisciplinary approach by the primary physician, a consulting psychiatrist, and a speech therapist. On occasion, patients experience clinical improvement after gaining insight into the functional nature of their problem. Currently utilized techniques include relaxation exercises combined with speech therapy techniques designed to improve awareness of vocal cord position. Prognosis is generally good if the condition is recognized before patients develop long-term complications of corticosteroid therapy for a misdiagnosis of asthma.

The present patient demonstrated typical clinical

![Figure 2. Laryngoscopic view during inspiration shows that vocal cords are adducted with a small posterior glottic chink (solid arrow). Open arrow identifies the epiglottis.](image-url)
features of vocal cord dysfunction and underwent laryngoscopy during her episode of wheezing. Her vocal cords were adducted during inspiration with a small posterior glottic chink (Fig 2). After the initiation of therapy and psychiatric counseling and speech therapy, the patient improved markedly. No further “asthmatic” episodes occurred during follow-up despite discontinuation of all asthma drugs.

**Clinical Pearls**

1. Symptoms out of proportion to objective findings, wheezing during inspiration, poor response to aggressive asthma therapy, and a normal alveolar-arterial oxygen gradient should suggest vocal cord dysfunction in patients presenting with acute asthmalike dyspnea.

2. The laryngoscopic examination during acute episodes of vocal cord dysfunction confirms the diagnosis by demonstrating inspiratory adduction of the vocal cords, which form a small posterior glottic chink.

3. Patients with vocal cord dysfunction are typically women aged 20 to 40 years who frequently have underlying affective disorders, experience secondary gain from their respiratory condition, and work in paramedical fields.

4. Therapy begins with discontinuation of asthma medications followed by a multidisciplinary approach with a speech pathologist, the primary physician, and a psychiatrist employing patient insight and relaxation exercises.

**Suggested Reading**


