

Vocal Cord Dysfunction: Two Case Reports

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Abstract

Vocal cord dysfunction is an uncommon condition characterised by adduction of vocal cords that can masquerade as or coexists with bronchial asthma. The glottic dysfunction is due to a functional (non-organic) cause. If unrecognised, incorrect diagnosis may result in patients being unnecessarily treated as refractory or severe asthma with high doses of corticosteroid. This may result in unwarranted steroid toxicity. Clues that should raise clinical suspicion to the diagnosis of vocal cord dysfunction include lack of response to bronchodilators, poor reproducibility of spirometric indices due to inconsistent effort and truncation of the inspiratory limb of the flow-volume loop. Definitive diagnosis is made by direct visualisation of the vocal cords during an attack. We report two patients with vocal cord dysfunction and review the literature on this disorder.

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Introduction

Vocal cord dysfunction (VCD) is an uncommon but important condition that can masquerade as or coexists with bronchial asthma that physicians should be aware of. Considerable morbidity, particularly steroid-related complications may be avoided with appropriate diagnosis and treatment. We report two cases of VCD and review the literature.

Case Reports

Case 1

An 18-year-old university student was referred for control of asthma. She was first seen in 1991. She gave a history of recurrent episodic shortness of breath associated with wheezing precipitated by cold weather, dust and exercise over the preceding one year. Her brother had asthma in childhood. She had received therapy consisting of inhaled salbutamol, inhaled beclomethasone dipropionate up to 2000 mcg per day and short courses of prednisolone. She had also worked as a temporary clinic assistant. Between 1991 and 1996 she was admitted to the hospital on seven occasions for asthma. Typically, she would refuse admission despite medical advice and would request discharge at her own risk once she felt better. Two of these admissions were to the intensive care unit and on one occasion she was intubated and mechanically ventilated.

Clinical examination during the attacks often revealed

inspiratory and expiratory rhonchi. She was unable to vocalize during the attacks. Inspiratory and expiratory stridor was noted on two occasions.

Full blood count, chest and lateral neck roentgenogram were normal. Her peak expiratory flow rate (PEFR) varied between 150 L/min and 320 L/min. Nomocapnic hypoxaemia (PaO₂ 53.1, PaCO₂ 38.2) was documented during one of her attacks.

She had a positive histamine bronchoprovocation challenge (PD₂₀ FEV₁ = 390 mcg). Skin prick tests were negative for multiple common allergens. Spirometric values in 1994 were as follows: forced expiratory volume in one second (FEV₁) 2.72 L (92% of predicted), forced vital capacity (FVC) 3.08 L (84% of predicted), and FEV₁/FVC ratio 88.3%. Flow-volume loop during an attack revealed reduction of inspiratory flow, consistent with variable extrathoracic upper airway obstruction (Fig. 1). The diagnosis of VCD was confirmed at an otolaryngologic examination during an attack in April 1994 which revealed paradoxical closure of vocal cords during inspiration. She was able to get off inhaled steroids but subsequently defaulted speech therapy and follow-up.

Case 2

A 62-year-old female housewife and lifetime non-smoker presented in 1995 with chronic cough of three months duration. The paroxysms of cough were associated with shortness of breath. There was no past history of asthma or

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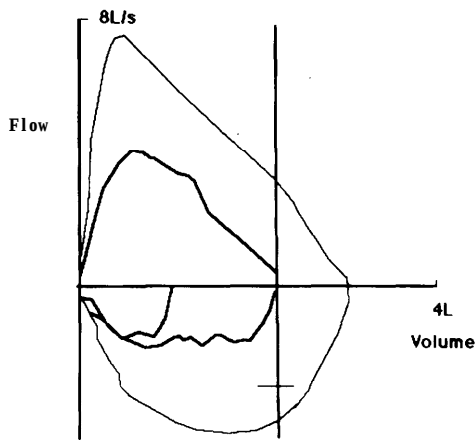


Fig. 1. Flow-volume loop of case one showing truncation of the inspiratory limb.

family history of asthma. She did not have symptoms of rhinitis or gastroesophageal reflux. Clinical examination was unremarkable. Her arterial blood gas was normal with a normal alveolar-arterial (A-a) oxygen gradient. PEFR varied between 240 L/min and 320 L/min. Full blood count and chest roentgenogram were normal. Histamine bronchoprovocation challenge was positive ($PD_{20}FEV_1 = 394$ mcg). Skin prick tests were positive for pollen, feather and dog hair. Barium swallow was normal and no gastroesophageal reflux was demonstrated. Spirometry revealed the following: FEV_1 1.48 L (93% of predicted), FVC 1.86 L (88% of predicted), and FEV_1/FVC ratio 79.7%. Flow-volume loop showed reduction of inspiratory flow, consistent with variable extrathoracic upper airway obstruction (Fig. 2). Bronchoscopy revealed paradoxical adduction of the vocal cords during inspiration thus confirming the diagnosis of VCD. She responded to treatment with inhaled β_2 agonists and inhaled corticosteroids for her asthma and speech therapy for her VCD.

Discussion

VCD was first recognised in 1974.¹ It is due to inappropriate adduction of otherwise normal vocal cords.² The glottic dysfunction is due to a functional (non-organic) cause and may be associated with underlying emotional or psychiatric factors.³ Hence, the terms "Munchausen's stridor"¹ and "factitious asthma"⁴ have been used to describe the condition.

VCD is an important differential diagnosis of wheeze that clinicians need to recognise." A majority of patients presenting with refractory or steroid-dependent asthma at the Ohio State University Medical Center were found to have VCD and if they were compliant with the treatment of VCD, they were able to wean off corticosteroid therapy.⁶ Morbidity from steroid toxicity can be avoided if patients with VCD masquerading as refractory or steroid dependent asthma are identified.

Patients with VCD are typically female, aged 20 to 40 years, and often have previous psychiatric illness and employment in the paramedical field.⁷ Notably, both our patients were females and the first patient had worked in healthcare-related occupation. Interestingly, the second patient had a family member in the healthcare profession. The onset of dyspnoea and wheezing in the first patient had coincided with her entry into tertiary education. Educational stress in a meritocratic society that places heavy emphasis on academic performance could have precipitated her VCD.

As in both our patients, asthma and VCD can coexist.⁸ In pure VCD, arterial blood gases and the alveolar-arterial oxygen tension difference are usually normal. Hypoxaemia has been reported in patients with VCD⁹⁻¹² and its presence does not rule out this condition. Pronounced variability in spirometric tests due to variable effort may in fact be a clue to the diagnosis.² As in both our patients, flow-volume loops performed during the attack characteristically show evidence of variable extrathoracic airway obstruction, manifesting as a flat inspiratory loop with an increased ratio of forced expiratory flow at 50% vital capacity to forced inspiratory flow at 50% vital capacity.² This feature of the flow-volume loops provides suggestive clues to the diagnosis of VCD.

Visualisation of the vocal cords during an attack is essential for the definitive diagnosis of VCD. The classic findings are inspiratory adduction of the anterior two-thirds of the vocal cords with a diamond-shaped chink in the posterior section of the cords.^{5,13} Laryngoscopy is often normal in an asymptomatic patient.⁸ Provocative challenges with methacholine or exercise will often reproduce the VCD.³ A multidisciplinary approach with involvement of the physician, psychiatrist and speech therapist is most effective in the treatment of patients with vocal cord dysfunction.^{5,7} Speech therapy is utilised in order for the patient to learn relaxed throat breathing.⁷ Heliox therapy

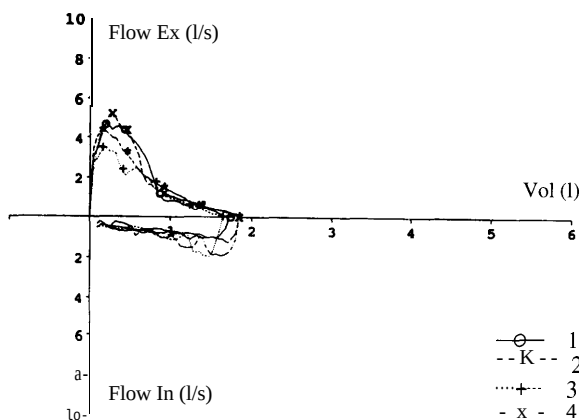


Fig. 2. Flow-volume loop of case two.

(helium-oxygen mixture) is effective in the acute treatment of VCD. The low density of this mixture allows less turbulent flow past the adducted vocal cords, thereby alleviating the dyspnoea and often interrupting the attack.^{2,14}

The long-term prognosis of VCD is unknown as present knowledge is based on limited case reports with short follow-up. In many cases, a good prognosis follows a positive reaction to the initial explanation of the diagnosis.²

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