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## CASE REPORT

# Paradoxical Vocal Cord Adduction Mimicking as Acute Asthma in a Pediatric Patient

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**SUMMARY** **We report an adolescent girl with paradoxical vocal cord adduction who presented with acute onset of hyperventilation, wheezing and stridor that did not respond to bronchodilator and anti-inflammation therapy. The paradoxical vocal cord motion was confirmed by flexible fiberoptic bronchoscopic examination. We found the stridor was induced by hyperventilation, and was caused by paradoxical vocal cord movement. The abnormal cord motion may be psychogenic and could be misdiagnosed as asthma. It is important to investigate the underlying background and social history and to avoid unnecessary use of beta-agonists, steroids, and even endotracheal intubation or tracheostomy.**

Paradoxical vocal cord adduction (PVCA) has been defined as adduction of the vocal cords on inspiration and abduction on expiration.[1-3]. Although the exact etiology of the paradoxical movement is still unknown, multiple etiologies including brainstem compression, motor neuron injury, movement disorder, gastroesophageal reflux, malingering disorder, and somatization/conversion disorder have been reported.[4] It is usually considered to be associated with psychosocial stress and psychiatric disease.[5] PVCA is a rare disorder that may present with symptoms similar to asthma. Vocal cord dysfunction may exist alone or accompany the diagnosis of asthma in as many as 33% or more of these patients.[6] PVCA may present with wheezing mimicking status asthmaticus and stridor[7] which mimics life-threatening airway compromise such as of infectious, traumatic, neoplastic or structural etiologies. It is important to differentiate this benign disorder from wheezing and stridor in the emergency department especially if those patients presented with psychogenic hyperventilation.

### CASE REPORT

A 14-year-old girl with a history of asthma, diagnosed early in childhood but asymptomatic for many years after desensitization, presented with a cold, runny nose, congestion and progressively worsening coughing and wheezing. The patient was seen in the outpatient clinic a few times and was diagnosed acute asthmatic condition. She was given oral erythromycin and bronchodilators. However, the patient continued to have poor appetite, was coughing constantly and lost two to three pounds in one month. She was admitted to a community hospital in the State for further work-up. Because of persistent cough and a history of tuberculosis exposure, a tuberculin test was done and revealed an induration of 18 x 20 mm in diameter but a chest X-ray was negative. Isoniazid 300 mg was recommended for the latent tuberculous infection. From then onwards she developed frequent laryngeal spasmodic coughing spells despite steroid inhalation treatment.

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She was then referred to an oto-rhinolaryngologist. A flexible fiber-optic laryngoscopy demonstrated no evidence of irritation or tumor masses.

She was transferred to the emergency department of Chang Gung Children's Hospital because of inspiratory stridor, expiratory wheezing, shortness of breath and hyperventilation. Arterial blood gas analysis showed pH: 7.637, $pCO_2$: 12.7 mmHg, pO2: 190.2 mmHg and $HCO_3$: 13.3 mm/l.

Physical examination revealed an afebrile adolescent girl with a respiratory rate of 32/minute. Oropharyngeal examination was normal. Breathing sounds were clear on auscultation, except for persistent stridorous sounds. The extremities were warm, with good peripheral pulses. Neurologic examination revealed no abnormalities.

According to the patient's history of asthma exacerbated by upper respiratory tract infection, she was treated with ipratropium bromide (Atrovent) and terbutaline (Bricanyl) nebulization and intravenous hydrocortisone injection (5mg/kg/dose) for 3 days, without improvement. Although serum CAP allergen test (Pharmacia Upjohn Pharmaceutical, Uppsala, Sweden) was performed and showed sensitivity to mite, grass pollen and dog dander allergens, no obvious history of exposure to these allergens could be found preceding this acute attack. Pulmonary function tests revealed a normal expiratory flow rate with FEV1 of 2.91 l (113% predicted), an FVC of 2.99 l (124% predicted), and an FEV1/FVC ratio of 97.3%. The flow-volume relationship showed a flattening of the inspiratory limb, which was suggestive of a variable extrathoracic

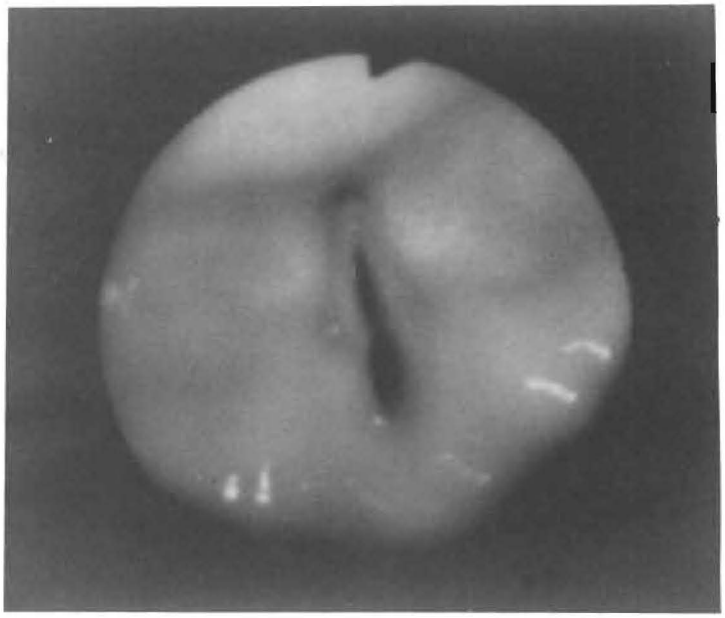

**Fig. 1** Flexible bronchoscopy from this patient with videotape, with the photograph taken during inspiration and expiration. During deep inspiration, the vocal cords show closure anterior and the posterior glottis is partially open.

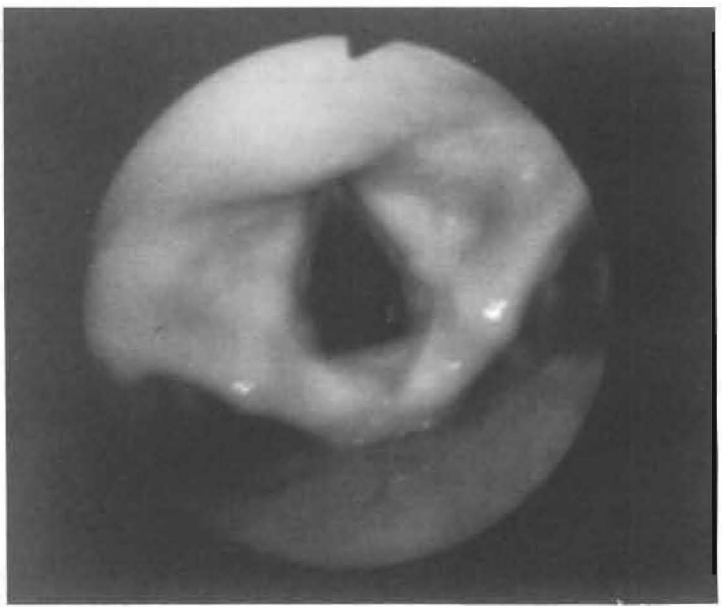

**Fig. 2** In contrast, the vocal cords show maximal abduction during deep expiration. The vocal cords are in paradoxical movement.

obstruction. Because of persistence of recurrent inspiratory stridor after anti-asthmatic therapy, a flexible bronchoscopy examination was performed, which showed paradoxical vocal cord movement with abduction of the vocal cords on expiration and adduction of the vocal cords on inspiration (Figs. 1 and 2). But no paradoxical vocal cord adduction nor inspiratory stridor was observed after midazolam treatment.

Social history revealed that she moved to Los Angeles, California 5 years ago and currently lives with her mother and a younger sibling. She is the elder sister of two children to a kind, hardworking father and a doting mother. Asthma had been well controlled for 4 years. Because of the persistent shortness of breath and stridor, she had to suspend schooling for about one month and displayed a dysphonic anxious mood two weeks before admission. Although she had done well in school and there was no obvious evidence of stress or emotional disturbance, the psychological impact resulting from her fear of a protracted and ineffaceable disease should be considered. After relaxation training and behavior therapy, she went home and was followed at our Out Patient Department with gradual improvement.

## DISCUSSION

Paradoxical vocal cord motion results from a true vocal cord adduction during inspiration. Stridor is caused by a high velocity air flow through a small passage, such as hypopharynx, larynx, trachea and mainstream bronchi.[8] Previous reports described that the paradoxical vocal cord adduction may present with wheezing, stridor mimicking bronchial asthma[1,9] sometimes accompanied with hyperventilation.[10] Like other cases reported previously, our patient had a history of asthma before PVCA was diagnosed. We believe that stridor was induced by exercise or hyperventilation[10] and was caused by PVCA. If PVCA is found to be psychogenic, early recognition may prevent unnecessary respiratory treatment and lead to definite psychiatric assessment and management.

The first report of paradoxical vocal cord movement was made in a child on initial presentation of wheezing to an emergency department by Poirrier *et al.*[11] in 1996. The previous reported pediatric patients all carried the diagnosis of asthma for several months to ten years before the diagnosis of PVCA was made. Vocal cord dysfunction can masquerade as asthma and is indeed often coexisting with asthma in 33-56% of patients, according to Brugman and his colleague[6,12] in 1993. Due to a high probability of coexistence of asthma history, unsuccessful invasive treatments such as intubation and even tracheostomy have been done. Therefore, correct diagnosis in emergency departments is most important.

The true etiology of PVCA is unknown. A classification scheme for paradoxical vocal cord motion was proposed recently by Maschka *et al.*[4,13] in 1997. Isolated laryngeal dysfunction with abnormal vocal cord movements has been shown to occur both in various organic as well as non-organic conditions.[4,13] It is generally considered to be a symptom of psychosocial stress and/or psychiatric disease.[5,14] In addition, association with a history of sexual abuse has been reported by Freedman *et al.*[15] in 1991. The possible causes of PVCA are classified in Table 1.[4] Organic causes of PVCA occur less frequently than

**Table 1** Classification scheme for paradoxical vocal cord adduction

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| **Non-organic causes** |
| Factitious or malingering disorder |
| Somatization/conversion disorder |
| Major depression |
| Munchausen's syndrome |
| Obsessive compulsive disorder |
| Adjustment disorder |
| Sexual abuse |
| Post-traumatic stress disorder |
| **Organic causes** |
| Brainstem compression |
| Cortical or upper motor neuron injury |
| Nuclear or lower motor neuron injury |
| Movement disorders |
| Post-operative complication |
| Gastroesophageal reflux |
| Cystic fibrosis |

non-organic causes. The non-organic laryngeal dysfunction with a functional component is being increasingly recognized, the symptoms of which can be easily confused with those of asthma, as in this patient.

It is important to explore and correct the underlying life-threatening organic causes of PVCA such as brainstem compression, motor neuron injury, etc. Besides that, many methods about treatment of patients with paradoxical vocal cord adduction have included oxygen, bronchodilators, and epinephrine as well as intubation, and even tracheostomy.[2,14,16] Pharmacological treatments reported as successful include breathing of a mixture of helium 80%/oxygen 20%, benzodiazepines[2] and antidepressant drugs.[5] Educating the patient with speech therapy has proven very useful to abort the acute attack and prevent recurrence.[5,14,16] Treatment of PVCA is aimed at normalizing vocal cord movement through resolving the underlying cause, such as a central nervous system lesion or a psychogenic problem. Distinguishing the PVCA from asthma is very important since the treatment is quite different. Evaluation of patients with PVCA requires identification of the underlying causative factors to direct appropriate diagnostic evaluations and treatment which can decrease cost as well as morbidity of the patient.

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