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CASE REPORT

Severe vocal cord dysfunction resistant to all current therapeutic interventions

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Summary
Vocal cord dysfunction (VCD) is characterised by paradoxical vocal cord adduction during inspiration or throughout the respiratory cycle, it results in wheeze, stridor, cough and dyspnoea. Although asthma and VCD can coexist, patients with VCD are frequently misdiagnosed with refractory asthma. It can severely restrict an individual's level of activity and effective therapeutic control can be difficult to achieve. We report the case of a patient who was treated with all available therapeutic interventions, including intralaryngeal botulinum toxin injection, but failure resulted in a permanent trachesotomy.

Introduction

Vocal cord dysfunction (VCD) is characterised by paradoxical vocal cord adduction during inspiration or throughout the respiratory cycle, it results in wheeze, stridor, cough and dyspnoea.[1] Although asthma and VCD can co-exist, patients with VCD are frequently misdiagnosed with refractory asthma.[2] In the emergency department, exacerbations may be incorrectly triaged as acute asthma or acute upper respiratory tract obstruction, resulting in unnecessary tracheal intubation or tracheostomy.[3]

VCD is most prevalent among young women aged 20–40 yr and is thought to be primarily psychological in causation.[1] Ayres and Gabbott[4] have suggested that laryngeal hyperresponsiveness to stimuli such as cold air and stress may alter autonomic balance and represent the pathophysiology of VCD. It can severely restrict an individual's level of activity and effective therapeutic control can be difficult to achieve. We report the case of a patient who was treated with all available therapeutic interventions, including intralaryngeal botulinum toxin injection, but failure resulted in a permanent trachesotomy.

Case

A 38-yr-old gentleman was referred with poorly controlled adult onset asthma, associated with a raised eosinophil count. At this time his main symptoms were dry cough, dyspnoea and gastro-oesophageal reflux. He had a 20 pack

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year smoking history with normal alpha I anti-trypsin levels. Occupational history included employment in the rubber and printing industry. Despite increasing anti-asthma therapy, hospital admissions were becoming more frequent and the patient was forced to retire from work.

Pulmonary function testing was poorly reproducible but more consistent with restrictive rather than obstructive pulmonary disease but CT scanning of the thorax demonstrated normal lung fields and normal bronchial airways. At first bronchoscopy, the vocal cords functioned normally and saline failed to induce a stridor. Shortly after bronchoscopy, the patient developed an inspiratory stridor and laryngoscopic examination revealed paradoxically adducted vocal cords. An emergency tracheostomy was performed by the on call ENT surgeons who made a diagnosis of bilateral vocal cord paralysis. CT scanning showed normal laryngeal and tracheal airway.

While the tracheostomy was in situ, the patient did not have any respiratory related hospital admissions and a repeat laryngoscopic examination confirmed normal vocal cord function. The tracheostomy was decannnulated seven months after insertion, since the site became chronically infected with methlicillin resistant staphylococcus aureus and no conservative therapies had been attempted.

Following decannulation, the patient had two acute hospital admissions with stridor and other exacerbations lasting up to 1 week which were managed at home. Thus, a co-ordinated multi-disciplinary treatment programme was needed.

Firstly, intensive outpatient and inpatient speech therapy was commenced over a 9-month period, using release and relaxation techniques allied with stress counselling. This approach had some success but did not prevent an acute respiratory admission with stridor, when laryngoscopic examination confirmed the diagnosis of VCD.

A face mask with inspiratory resistance, which decreases vocal cord adduction by reducing inspiratory air flow, was produced for the patient.[5] This approach resulted in a 2 month period of good symptom control without any hospital admissions. However, the success was not maintained and eventually the stridor became continuous while the patient remained awake.

Botulinum toxin injection of the true and false vocal cords was performed, using techniques described in spasmodic dysphonia with doses of 10 units to both thryoarytenoids and 2.5 units to both false cords.[6] Vlahakis et al.[7] have reported mild symptomatic relief following such an approach in VCD. In our case, the symptoms fully resolved within 48 h but unfortunately recurred within 1 week of the procedure. Laryngoscopic examinations during the relapse showed laryngeal closure caused by false and true cord adduction (Fig. 1). In view of recurrent relapses, the patient was discharged with a permanent tracheostomy.

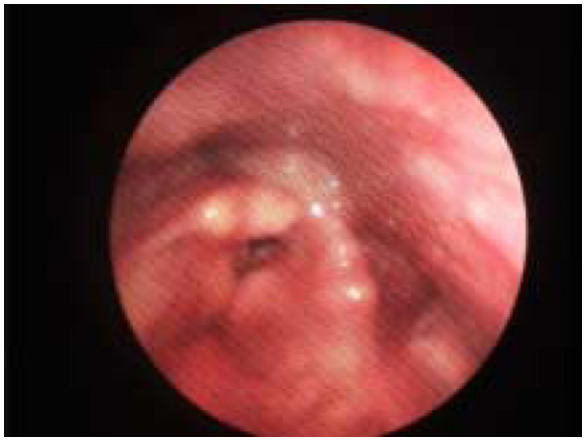

Figure 1 Adducted true and false cords, 1 week after botulinum toxin injection.

Discussion

This is the first documented British case of VCD, in which all recommended and available therapies have been systematically attempted. Unfortunately, despite speech therapy, stress counselling, an inspiratory resistance mask and botulinum toxin injections, tracheostomy reinsertion could not be prevented.

The underlying aetiology in this case includes psychological stress, gastro-intestinal reflux and asthma; these were all addressed in the therapeutic programme outlined. Treatment of VCD challenges respiratory physicians and requires a co-ordinated multi-disciplinary treatment programme to optimise the chance of success.

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