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Case report

Co-existence of vocal cord dysfunction with pulmonary conditions other than asthma: A case series

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ABSTRACT

Background: Vocal cord dysfunction (VCD) is defined as inappropriate movement of the vocal cords resulting in functional airway obstruction and symptoms including cough, wheezing, and dyspnea. VCD is often misdiagnosed with asthma but can also co-exist with asthma. The association of VCD with other serious pulmonary conditions has not been described to date.

Case reports: We describe the first case series of two adult patients evaluated at a university asthma clinic who in addition to having VCD also had significant pulmonary pathology other than asthma. Patient 1 had VCD and pulmonary veno-occulsive disease which necessitated a lung transplant. Patient 2 had VCD and a patent ductus arteriosis who necessitated surgical closure.

Conclusion: It is important to recognize that VCD can exist with pulmonary conditions other than asthma. Lack of improvement in respiratory symptoms after appropriate treatment for VCD should alert the clinician to evaluate for additional conditions.

1. Introduction

In 1902, Sir William Osler defined vocal cord dysfunction (VCD) as spasms of laryngeal muscles occurring during inspiration and in times of great distress [1]. Currently, VCD is described as exaggerated adduction of vocal cords during inspiration and/or expiration causing respiratory and laryngeal symptoms [2]. Laryngoscopy, the gold standard for diagnosis, involves direct visualization of abnormal vocal cord motion during an acute attack or provocation with known triggers. Classic laryngoscopic findings include inspiratory vocal cord adduction of the anterior two thirds with a posterior diamond-shaped chink [3]. Additionally, abnormal spirometric findings may include flattening of the inspiratory flow-volume loop and increased FEF-50/FIF-50 (ratio of forced flow at 50% of expiration to forced flow at 50% of inspiration) [3–5]. The common respiratory symptoms observed in VCD include cough, dyspnea, wheezing and chest tightness. Because of the overlapping symptoms, the relationship between VCD and respiratory diseases is of particular interest for physicians specializing in pulmonary conditions.

The association between VCD and asthma has been well documented [6–8]. VCD can exist in isolation, can coexist with asthma, or can mimic asthma. Our group has shown that VCD and asthma co-existed in 32.6% of patients seen in a university asthma practice [6]. Comorbid VCD and asthma resulted in increased frequency of long-acting β-agonist use and worse quality of life [6,9]. In our cohort of VCD patients, 42.4% had been previously misdiagnosed as having asthma for an average of 9.0 years, contributing to more ED visits and systemic steroid use [6,10]. To date VCD has not been described in association with pulmonary diseases other than asthma. We describe a case series of two patients with co-existing VCD and non-asthma pulmonary conditions.

1.1. Case 1

A 21-year-old woman, diagnosed with exercise induced asthma at age 14 by exercise spirometry, presented with worsening exertional dyspnea over the past six months. She reported shortness of breath after walking 160 m (2 blocks) that would last 1–3 hours. Despite previous asthma treatment with inhaled corticosteroids, long-acting muscarinic agents, short acting beta agonists and nedocromil, she experienced minimal symptom improvement. Due to lack of improvement in her symptoms, she underwent a methacholine challenge test which was interpreted as positive. Interestingly, there was a parallel decrease in both the FEV1 and FVC without significant change in the FEV1/FVC

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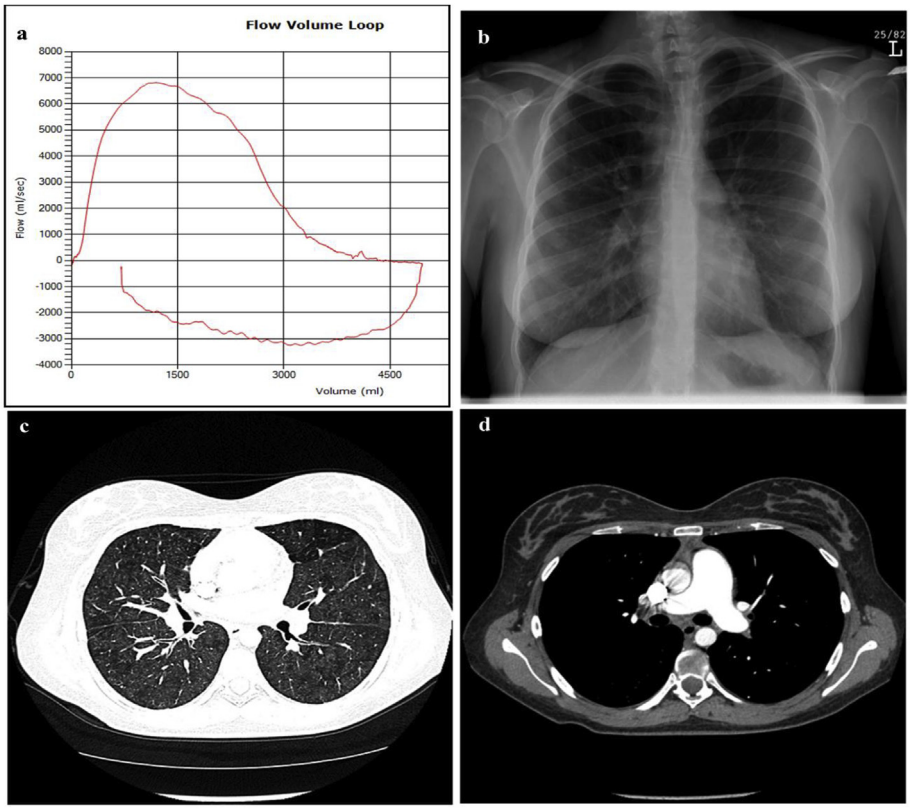

Fig. 1. a. Flattening of inspiratory flow volume loop; b. CXR with interstitial changes in lung bases; c,d. CT Chest with ground-glass opacities (c) and air-trapping and enlarged pulmonary artery (d).

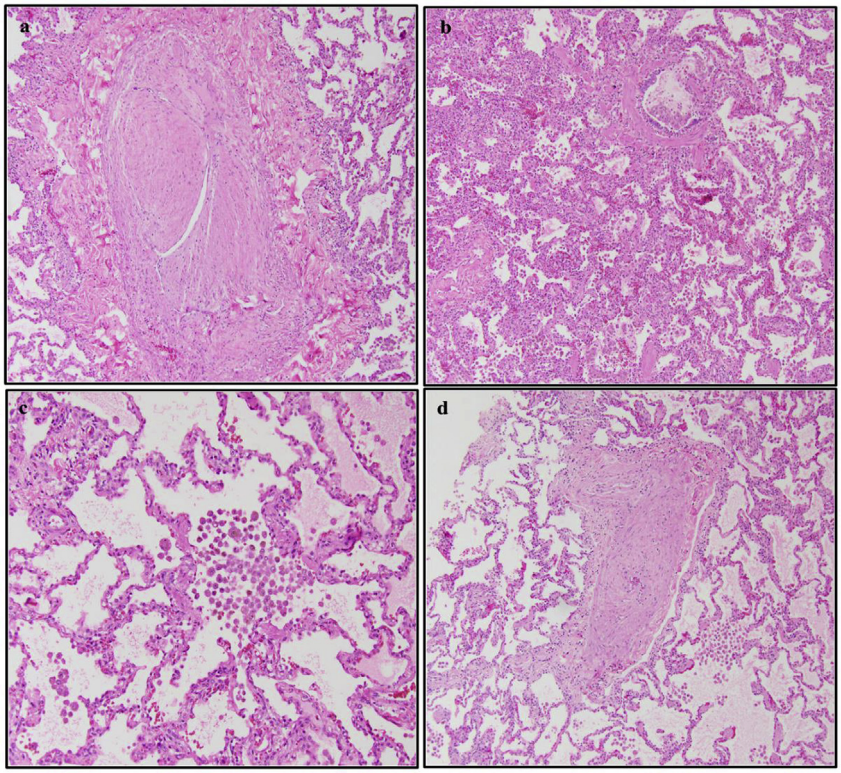

Fig. 2. a–d. Explanted lungs showing intimal fibrosis of veins and venules, diffuse alveolar septal thickening, alveolar hemosiderosis and intimal fibrosis of pulmonary arteries and hypertensive arterial changes.

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