Concurrent Dysphagia with Paradoxical Vocal Fold Movement

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Abstract

Inappropriate adduction of vocal cords during inspiration is defined as paradoxical vocal fold movement (PVFM). Patients are usually present with signs and symptoms of respiratory distress mimicking asthma. However, by fiber optic laryngoscopy the differential diagnosis can be easily made if PVFM is kept in mind. Exact diagnosis prevents unnecessary medications and surgical interventions for PVFM. Even though shortness of breath and dyspnea are seemed to be the major concerns affecting patients’ well-being, concurrent dysphagia may accompany as a bothering problem in some of the cases. Dysphagia management program, respiratory exercises and psychological counseling carry an important role in management of non-organic PVFM cases.

ABBREVIATIONS

PVFM: Paradoxical Vocal Fold Movement; EMG: Electromyography; GER: Gastroesophageal Reflux

INTRODUCTION

Inappropriate adduction of vocal cords during inspiration is defined as paradoxical vocal fold movement (PVFM) [1,2]. There are several synonyms for this entity such as paradoxical vocal cord motion, paradoxical vocal cord adduction, laryngeal asthma, hysterical stridor, and vocal cord dysfunction [3-8]. Patients are usually seen in emergency services with complaints of shortness of breath, dyspnea and dysphonia. The degree of respiratory distress changes from patient to patient and severe forms of PVFM are often misdiagnosed as asthma. Despite the absence of arterial hypoxemia, patients are often treated with standard asthma medications and sometimes urgent intubations or even tracheotomies [1,2,9]. Multiple etiological factors have been considered so far. A classifying scheme for PVFM according to its organic or non-organic etiology was proposed and seemed to ease the management of the disease [3]. PVFM mostly presents with airway obstruction symptoms. Dysphagia in PVFM is very rare and only reported in organic cases [5,10].

Here is a case of non-organic PVFM with severe dysphagia and the therapy procedure performed for this entity is presented.

CASE PRESENTATION

The patient was a 36-year-old female working as a nurse in a psychiatry clinic, presented with an acute onset of short of breath and dysphonia a month before the admission. Dysphagia had begun a couple of days after the initial symptoms. She complained about losing approximately 5 kilograms after the beginning of the symptoms. The patient’s medical history included epilepsy for 22 years and Bickerstaff encephalitis one year ago. The patient was on sodium valproate therapy for epilepsy and was neurologically disease free for 6 months. The patient’s local hospital diagnosed her as bilateral vocal cord paralysis and proposed for a tracheotomy. She had withstood the surgical operation; thus, she was put on corticosteroid and bronchodilator therapy, which had no effect on the symptoms. The patient’s local hospital diagnosed her as bilateral vocal cord paralysis and proposed for hera tracheotomy. She had withstood the operation; thus, she was put on corticosteroid and bronchodilator therapy, which had no effect on the symptoms. The patient was referred to our clinic for further evaluation of the respiratory distress and dysphagia complaints [11,12].

The patient was very fraught at first sight. Dyspnea with exertion and a loud inspiratory stridor was conspicuous. This clinical scene was thought to be supporting for the tracheotomy decision. However, at fiber optic pharyngolarygoscopic evaluation (FPE), observing the adduction of the vocal cords during inspiration and a chinking at the posterior third of the cord, PVFM diagnosis was put on. The fiber optic views of the
vocal folds at rest position, during inspiration and phonation are seen in Figures 1, 2, 3. She was given an Oral Peripheral Examination, the Bedside Swallow Evaluation (BSE), and Modified Barium Swallow Study (MBSS) according to our clinics’ management plan for dysphagia. Structured oral peripheral examination included a thorough assessment of the oral cavity, including tongue strength and mobility, palatal elevation, and the diadochokinetic measures. All those structures were measured to be normal and oral/speech apraxia was absent.

In the BSE, she was given 5 cc x 5 liquids (cherry juice x 2, peach nectar x 2, and water x 1). As solids, she was given 1 piece of graham cracker, 1 small banana, and ½ cup fruit flowered yogurt. Her initial EAT 10 (Eating Assessment Tool-10) [13] score was a potential dysphagia problem. Her DHI (Dysphagia Handicap Index) [14] score was also indicative of severe dysphagia with an equal number of distribution of the scores of DHI Subcategories (Functional Problems, Physical Problems, Emotional Problems) (total score of 90/100, score of 30 for each category).

Following BSE, she underwent the Modified Barium Swallow Study (MBSS). Due to the fear of swallowing solids; the patient’s MBSS was completed with thin and thick liquid consistency (1 cc, 3 cc, and 5 cc of each at anterior-posterior and lateral positions) opaque contrast matter. In Figure 4, MBSS showing, 3 cc bolus passing do not trigger the opening of the Upper Esophageal Sphincter, the triggering of the swallow reflux is also delayed during oro-pharyngeal pass. Anterior-posterior bolus introduction with 1cc and 3 cc barium indicated a problem with the opening of the upper esophageal sphincter (Figure 4); however, 5-cc bolus had no problem in advancing into the esophagus (Figure 5). There was no penetration nor aspiration observed during MBSS.

Figure 1 Vocal folds at rest position.

Figure 2 Vocal folds during inspiration.

Figure 3 Vocal folds during phonation.

The patient underwent speech and swallowing exercises with psychological counseling for non-organic PVFM. During the first 5 days, no improvement was obtained in the clinical scene. On the sixth day, all symptoms recovered suddenly. She has been followed up disease free for 10 months up to date.

DISCUSSION

Vocal cords make an adduction movement on inspiration in order to enlarge the glottic space. Adduction of the cords in PVFM patients causes an obstruction in the upper airway resulting in signs and symptoms that mimic asthma. On this, misdiagnosed patients may be given inessential medications, emergency intubations, even tracheotomies (especially in emergency departments) [1,3-5,8,9]. To prevent these undesirable situations, one must keep PVFM in mind in differential diagnosis of patients presenting acute respiratory distress refractory to asthma therapy. In history, proposal or performance of tracheotomy usually persists and medications including high doses of steroids does not provide any benefits as seen in the presented case. Oxygen saturation is usually in normal ranges even though there are noisy stridor, wheezing, or signs of short of breath [1,3,9]. We also observed this in the presented case. The golden standard diagnostic tool is flexible pharyngolaryngoscopy and observing the inappropriate adduction of the vocal cords on inspiration. Asthma may also co-exist in PVFM patients [13]. The pulmonary function tests also contribute to the diagnosis in an especially symptomatic period. Though the patient had failed to perform this test, asthma is excluded in differential diagnosis by physical examination and considering the lack of benefit of anti-
asthmatic medications. PVFM seems to be more prevalent in the female gender and health care workers [8,9]. The presented case was in agreement with these demographic criteria.

Several factors may underlie in PVFM. Maschka et al. [3] developed a classification scheme for PVFM according to its underlying etiology. In organic group brainstem compression, cortical or upper neuron injury, nuclear or lower motor neuron injury, movement disorder, gastroesophageal reflux (GER) exists. Factitious or malingering disorder and somatization/conversion disorder constitute the non-organic group. In the presented case, organic causes of PVFM were distinguished by neurological evaluation. Since the presented case had no significant sign or symptom implying GER, she was classified as non-organic PVFM.

While reviewing the literature it is interestingly seen that dysphagia is only reported in a low number of PVFM cases. Maschka et al. [5] notified that dysphagia could be associated with organic causes resulting in vagal dysfunction due to brainstem compression as it is seen in Arnold-Chiari malformation. In a retrospective study, Portier et al. [10] reviewed 286 children with Arnold-Chiari malformation and detected PVFM in only 2 cases. Dysphagia were reported in both cases and recovered by decompression surgeries. Perkner et al. [8] classified PVFM according to being exposed to occupational or environmental inhalants. Dysphagia was observed in 5 of the 9 cases (56%) in irritant group and 8 of the 30 cases (27%) in non-irritant group. Because of the classification scheme of the study, it is impossible to realize the incidence of dysphagia in organic or non-organic causes. In the presented case dysphagia was onset at the same time as the respiratory distress and dysphonia. The patient also emphasized a weight loss during the disease.

Alternative treatment modalities were put into practice for PVFM. Botulinum toxin injection to laryngeal muscles under EMG guidance improved the average scores of the rating scale of PVFM patients [13]. Anti-reflux therapy is an effective therapy for GER related PVFM patients [2]. Surgical procedures for brainstem compression in organic PVCM cases may resolve the complaints [10]. Speech therapy and respiratory training are reported as effective treatment modalities for especially non-organic PVFM cases [2,7,9,14].

The therapy of the presented patient was established on mainly gaining the patient's trust and cooperation. In order to provide psychiatric supportive therapy, she was informed about the etiological factors of the disease and the therapy technique. This was also to rule out for any component of phagophobia.
At this point, her being a psychiatry nurse is considered to be a beneficial aspect. Afterwards she began synchronous respiratory retraining and swallowing exercises [19]. A speech-language pathologist carried out the respiratory retraining exercises matching with the procedures published in the previous articles [2,7,9]. The patient was taught to perform coordinated thoracic-abdominal breathing in upright and supine position and laryngeal relaxation techniques. The voice quality was tried to improve by coughing and hard glottic attacks.

In the literature, no articles were found about the management of the dysphagia associated with PVFM. Knowing the origin is non-organic, and patient’s positive response to relaxation and hard glottal attacks made us believe that she would benefit from a dysphagia management program, which consisted of bolus adjustment, head positioning during swallow, exercises, and effortful swallow technique. Introducing bolus with the following schedule: stage I, full liquids (thin, nectar and honey consistency) and stage II, alternating liquids with pureed consistency solids.

The patient did not show any improvement in the first 5 days of the therapy. On the sixth day, all the symptoms were resolved suddenly during speech therapy session and she has been followed up disease free for 10 months up to date.

REFERENCES