



Bilateral Vocal Cord Abductor Paralysis in Advanced Parkinson's Disease

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Abstract

Parkinson's disease (PD) is one of the most common neurologic disorders, affecting approximately 1% of individuals over 60 years of age. The annual incidence of Parkinson's disease ranges from 4.5 to 21 cases per 100,000 population [1]. PD is characterized by a combination of motor and non-motor manifestations, including rigidity, tremor, bradykinesia, speech impairment, and gait disturbances. The histopathologic hallmark of the disease is the progressive degeneration of pigmented neurons in the substantia nigra.

Keywords: Parkinson's disease

Introduction

Vocal cord paralysis may lead to life-threatening upper airway obstruction and has been reported as a cause of mortality in patients with neurodegenerative disorders such as Parkinson's disease and multiple system atrophy (MSA) [2]. Although vocal cord paralysis is relatively uncommon in Parkinson's disease, it is more frequently observed in patients with multiple system atrophy. To the best of our knowledge, only a limited number of cases of vocal cord paralysis associated with PD have been reported in the English-language literature. Chia Chan Tsai et al. [3] reported a case of bilateral vocal cord paralysis in a patient with PD, while Plasse et al. [4] reported two cases and Isozaki et al. [5] described three cases.

Case report:

A 57-year-old woman with stage 5 Parkinson's disease (PD) presented with a 1-week history of stridor. She had been diagnosed with Parkinson's disease more than 10 years earlier, initially presenting with tremor, rigidity, and bradykinesia. Her functional status progressively deteriorated, and over the preceding 4 years, her disease had reached stage 5 according to the Hoehn and Yahr scale. She was subsequently bedridden and required Ryle's tube feeding. She had also experienced multiple previous admissions for bronchopneumonia. The day of the admission, the patient developed inspiratory stridor, which became more pronounced during episodes of worsening tremor and bradykinesia. There was no history of thyroid disease, diabetes mellitus, smoking, or alcohol consumption. The patient subsequently developed acute respiratory distress, characterized by severe dyspnea and progressive oxygen desaturation, with associated vocal cord paralysis on the nasofibroscopy and impaired airway protection. Given the severity of respiratory compromise, the patient was urgently admitted for emergency tracheostomy to secure the airway. No anterior neck mass was detected. Indirect laryngoscopy (Fig. 1, 2) demonstrated both vocal cords in a paramedian position, with pooling of secretions. Laryngeal sensation was also reduced. Laboratory investigations were unremarkable. A chest X-ray showed no significant abnormality, with a normal mediastinal contour.

The patient's antiparkinsonism medication was optimized by the neuromedical team, and she was closely monitored in the ward for more than one week. Her oxygen saturation was continuously monitored and remained above 95% throughout the hospitalization. Following careful adjustment of her antiparkinsonism medication, a reduction in tremor, rigidity, and dyskinesia was observed.

A repeat indirect laryngoscopy (Fig. 1, 2) demonstrated findings similar to those of the initial examination, with the vocal cords remaining in a paramedian position. the patient remained clinically stable without significant oxygen desaturation after tracheostomy

Discussion

Upper airway obstruction secondary to bilateral vocal cord paralysis in Parkinson's disease (PD) is an uncommon complication and is more frequently observed in patients with Multiple System Atrophy (MSA). In the series reported by Huppler et al., only two of 633 patients with unilateral or bilateral vocal cord paralysis had Parkinsonism [6]. Similarly, Holinger et al. reported a low incidence, with only four patients with Parkinson's disease among 389 patients presenting with partial or complete bilateral vocal cord paralysis [7].

The laryngeal and esophageal musculature is innervated through the nucleus ambiguus and is functionally influenced by the basal ganglia and their descending fibers within the vagus nerve. The pathophysiological mechanism underlying vocal cord paralysis in Parkinson's disease remains poorly understood. One proposed mechanism involves degeneration of the nucleus ambiguus, a finding that has been demonstrated in cadaveric studies of patients with multiple system atrophy [5]. Multiple system atrophy is a neurodegenerative disorder characterized by varying combinations of Parkinsonism, cerebellar dysfunction, and autonomic disturbances [8]. Liberman et al. suggested that the mechanism responsible for vocal cord paralysis may be similar in patients with Parkinson's disease and multiple system atrophy [2].

Isozaki et al. reported several differences in the clinical and pathological characteristics of vocal cord palsy between PD and MSA. First, clinical laryngoscopic examination showed that vocal cord abductor paralysis (VCAP) in PD was not exacerbated during sleep, unlike in MSA. Second, histological examination of the intrinsic laryngeal muscles revealed no abnormalities of the posterior cricoarytenoid muscle in PD, whereas neurogenic atrophy of this muscle was observed in MSA. Third, severe dysphagia requiring tube feeding was more commonly observed among patients with PD than among those with MSA [5].

Regarding the management of bilateral vocal cord paralysis in PD, treatment options include tracheostomy to relieve upper airway obstruction or optimization of antiparkinsonism medical therapy. Several cases have been managed with tracheostomy, as reported by Vas et al. [9], Plasse & Lieberman [4], Read & Young [10], Corbin & Williams [11], Lew et al. [12], Nakane et al. [13], Onoue et al. [14], and Qayyum et al. [15]. In contrast, only a limited number of reported cases have been successfully managed with medical treatment alone, including the cases described by Chia et

al. [3] and Corbin & Williams [11].

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