Cricothyroid Muscle Botulinum Toxin Injection to Improve Airway for Bilateral Recurrent Laryngeal Nerve Paralysis, A Case Series

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Summary: Bilateral vocal fold paralysis most commonly results from iatrogenic trauma to the recurrent laryngeal nerve during surgical procedures in the anterior neck. Patients may require tracheostomy because of acute or gradual onset of dyspnea and airway compromise. The intralaryngeal injection of Botox has been considered as a possible therapy for these airway symptoms of bilateral vocal fold paralysis. Chronic unopposed activity of intact cricothyroid muscles could potentially result in gradual medialization of the vocal folds in patients with bilateral recurrent laryngeal nerve paralysis. This case series describes three patients who successfully underwent injections of botulinum toxin into the bilateral cricothyroid muscles to offer sustained relief of dyspnea resulting from bilateral vocal fold paralysis.
Key Words: Bilateral vocal fold paralysis–Botox–Cricothyroid muscle injection–Bilateral recurrent laryngeal nerve paralysis.

INTRODUCTION

Bilateral vocal fold paralysis most commonly results from iatrogenic trauma to the recurrent laryngeal nerve during surgical procedures in the anterior neck.[1] This may result in airway compromise and respiratory distress requiring tracheostomy in some patients. Other patients experience early breathy dysphonia and aspiration, as the vocal folds may be positioned in a more lateralized position. However, over time, the airway may worsen as unopposed action of the cricothyroid muscle continues in patients with bilateral recurrent laryngeal nerve paralysis. Patients begin to experience improved voice but notice gradually worsening dyspnea. The intralaryngeal injection of Botox (Allergan, Irvine, California) has been considered as a possible therapy for these airway symptoms of bilateral vocal fold paralysis. However, it is sparsely considered in the clinical literature; moreover, when it has been documented, the injection of Botox is most commonly into the thyroarytenoid muscle, and patients require serial injections until finally receiving more definitive therapy. In this series, we consider three patient cases in which injection of Botox into the bilateral cricothyroid muscles was successfully employed to decrease airway symptoms of bilateral vocal fold paralysis.

CASE REPORTS

Case 1

A 51-year-old woman presented with the chief complaints of wheezing, worsening shortness of breath, "feeling strangled," and loss of ability to sing. The patient had a total thyroidectomy for Graves disease and toxic diffuse goiter 9 months earlier, and her recovery was complicated by hematoma and subsequent neck exploration on postoperative day four. The patient complained of decreased exercise capacity as a result of her exertional dyspnea. Her decreased ability to phonate interfered with her day-to-day work requirements as a home childcare provider. She greatly feared airway compromise as she had previously had a tracheostomy; her goal was to avoid this outcome if at all possible. On examination, her voice was noted to be "raspy," and her breathing was minimally noisy on deep inspiration and without stridor at rest. Videostroboscopy revealed bilateral immobile true vocal folds, a 3-mm glottic chink, and the presence of an equal mucosal wave bilaterally with complete closure. Her voice handicap index (VHI) at the initial examination was 97. Options for therapy were discussed with the patient and among those considered included Botox injection of the bilateral cricothyroid muscles to decrease the gradual closure of her paralyzed folds due to unopposed action of the cricothyroid muscles. She returned to clinic 1 month later with ongoing dyspnea. Botox injections were recommended to potentially improve her airway. The patient's first treatment was 2.25 units of Botox injected into both cricothyroid muscles.

Over the next 5 months, the patient returned to clinic to undergo two additional injections of Botox (three units per side at 3 months and again at 5 months after initial injections) to the bilateral cricothyroid muscles. Between each injection, she experienced minor, progressive recurrence of dyspnea. However, at no time did her symptoms return to the severity that she had experienced before treatment. After her last injection, 13 months after presumed injury to the bilateral recurrent laryngeal nerves, she has not required further intervention. The patient experienced sustained relief of airway symptoms after her series of three Botox injections to the bilateral cricothyroid muscles, although vocal fold motion did not return as noted by flexible laryngoscopy with stroboscopy. She is back to normal voice use. Her clinical follow-up has been on an as needed basis since that time.

Case 2

A 23-year-old woman presented to our clinic with chief complaints of dysphonia, shortness of breath, and a choking

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sensation. She had a total thyroidectomy for Graves disease 4 months before presentation. An electromyography (EMG) 2 months earlier by another provider had reportedly demonstrated severe fibrillation potentials of the laryngeal musculature suggesting bilateral vocal fold paralysis. On examination, the patient's voice was breathy; her VHI was 118 (P38, F40, E40) with a maximum phonation time (MPT) of only 3 seconds. Videostroboscopy revealed bilateral immobile vocal folds in the paramedian position with absence of mucosal wave, incomplete closure, and no evidence of obstruction.

Three months later, she returned to clinic and notably had interval voice improvement and preservation of her airway. A repeat EMG demonstrated chronic denervation of the left thyroarytenoid muscle with a significantly reduced recruitment pattern (∼80%) and ongoing denervation of the right with a less dramatically reduced recruitment pattern (∼50%). Because of interval improvement in clinical examination, observation and close follow-up were recommended. However, her symptoms progressed, and she complained of worsening dyspnea that limited her activity and precluded her work as an emergency medical services officer. Her vocal folds had assumed a near midline position, and the patient had mild stridor at rest (Figure 1). At this point, 14 months after her initial thyroid surgery, 2.5 units of Botox were injected into the bilateral cricoarytenoid muscles.

As a result of the injections, the patient experienced stabilization and improvement in her dyspnea; however, the patient experienced transient cough and increased breathiness in voice quality. She required five additional Botox injections with 2.25–2.5 units per side, between 2 and 4 months apart, for transient improvement in dyspnea. Her relief of airway symptoms was consistently accompanied by worsening of voice quality and occasionally by a transient increase in cough and aspiration of thin liquids. Her most recent evaluation was 6 months after her last Botox injections. She sustained a much longer period of breathiness before her voice returned, but she had no return of shortness of breath, and her vocal folds remained immobile but in a more lateralized position (Figure 2). This finding may suggest that further injections may no longer be needed.

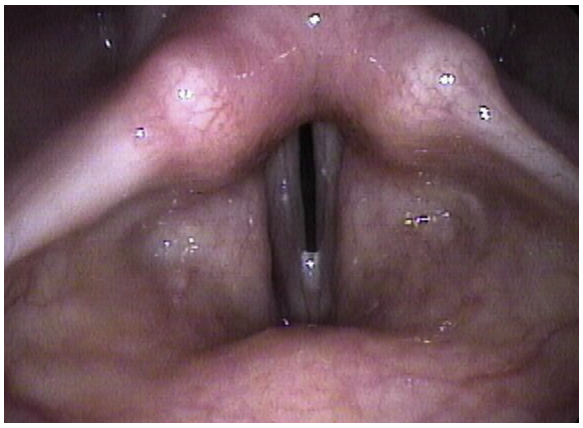

FIGURE 1. Bilateral vocal fold paralysis with the vocal folds in the paramedian position during maximum abduction.

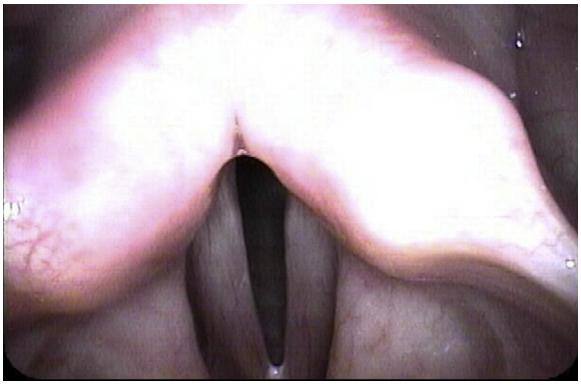

FIGURE 2. The same patient after Botox injections during maximum abduction with the vocal folds in a more lateral position.

Case 3

A 42-year-old woman presented with mild difficulty breathing immediately after total thyroidectomy for a large multinodular goiter. An evaluation at that time noted bilateral vocal fold immobility. She was treated initially with systemic steroids, which improved her breathing. She continued to experience moderate dysphonia. She was discharged with no other intervention. Follow-up 3 weeks later revealed bilateral vocal fold immobility with both vocal folds in a paramedian position. MPT was 7 seconds and her VHI was 62 (physical, 28; functional, 20; and emotional, 14). No treatment was initiated at that time. Her voice gradually improved nearly to the patient's baseline. Two months later, her laryngoscopy showed persistent bilateral vocal fold immobility with minimally lateralized vocal folds (MPT: 16, VHI: 22 [P: 10, F: 7, E: 5]). Over the next 2 months, the patient noted progressive dyspnea, initially only with exertion but gradually becoming constant, even at rest. This was accompanied with some mild inspiratory stridor. Her voice remained strong although she had trouble sustaining long sentences without feeling short of breath. Examination revealed bilateral immobility of the vocal folds, which were in a near midline position, and a 2-mm maximal glottic chink (MPT: 22, VHI: 29 [P: 14, F: 10, E: 5]). An EMG was performed, which showed reduced waveforms >75% with large motor units and markedly reduced recruitment of the thyroarytenoid muscles. The cricothyroid muscles were normal.

At that time, bilateral cricothyroid muscle Botox injections (3.0 units per side) were performed. Two to three days later, the patient began noticing improvement in her breathing and increased exercise tolerance, accompanied by slightly more breathy voice quality. Six weeks later, she was doing well both from a voice and breathing standpoint, but she still had some exercise intolerance with protracted aerobic exercise (MPT: 14, VHI: 32 [P: 18, F: 10, E: 4]). Ten weeks later, the patient returned with improved voice but complained of progressive shortness of breath, although not as significant as her pre-Botox state (VHI: 18 [P: 10, F: 5, E: 3]). Second, bilateral cricothyroid muscle Botox injections were performed (2.5 units per side). She did well for 6 months but again developed gradually worsening exertional dyspnea. She underwent a third set of bilateral Botox injections (2.5 units per side). At her last