Botulinum Toxin Therapy for Abductor Spasmodic Dysphonia

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Summary: Botulinum toxin has been widely accepted as an effective therapy for controlling the symptoms of adductor spasmodic dysphonia (ADSD). Reported experience with botulinum treatment for abductor spasmodic dysphonia (ABSD) has been less impressive. Factors that may impair outcomes for ABSD include differences in the pathophysiology of ADSD and ABSD and limitation of maximal dose from airway restriction with posterior cricoarytenoid muscle (PCA) weakness. We report our experience with botulinum injection of the PCA with an asymmetric dose escalation protocol, based on clinical observations that in ABSD, abductor spasms are often stronger on one side, usually the left. The nondominant side was injected with 1.25 units. Dominant side dose began at 5 units, with step-wise increments of 5 units per week until one of three endpoints was reached: Elimination of breathy voice breaks, complete abductor paralysis of the dominant side, or airway compromise. Fourteen of 17 patients achieved good or fair voice, with dominant-side doses ranging from 10 to 25 units. Exercise intolerance limited PCA dose in two patients. One patient had persisting breathiness that improved with medialization thyroplasty. Asymmetric botulinum toxin injection into PCA muscles can suppress abductor spasm in patients with ABSD, but breathiness may persist, because of inadequate glottal closure.

Key Words: Spasmodic dysphonia—Botulinum—Abductor.

INTRODUCTION

Spasmodic dysphonia (SD) is a disabling speech disorder in which episodic bursts of activity in laryngeal muscles intermittently disrupt vocal function. Its most common form is adductor SD (ADSD), wherein involuntary tight glottal closure causes frequent voice breaks. In abductor SD (ABSD), the voice is impaired by breathy voice breaks and/or a sustained whispering quality to the voice.[1] In a series of more than 900 SD patients, Blitzer et al[2] found that only 13% had ABSD. Botulinum toxin has been widely accepted as an effective therapy for controlling the symptoms of ADSD. In contrast, reported experience with

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botulinum treatment for ABSD has been variable and generally less impressive than for ADSD.[2,3] Whereas botulinum toxin can substantially improve symptoms in nearly all ADSD patients, fewer ABSD patients obtain benefit, and the degree of improvement is less profound. In the Blitzer et al[2] series, ADSD patients had an average benefit of 90% of normal, whereas ABSD patients had an average benefit of 66% of normal. The disappointing results of botulinum toxin therapy for ABSD had led to studies of alternative therapies, such as medialization thyroplasty.[4,5]

Factors that may contribute to less favorable outcomes include difficulty in localizing the PCA for injection and airway restriction with significant PCA weakness. Additionally, electromyographic studies indicate that the pathophysiology of ADSD and ABSD are significantly different. Although ADSD is characterized by spasms in adductor muscles, ABSD is more complex. Not only are there spasms in the abductor muscle of the larynx, the posterior cricoarytenoid muscles (PCA), there is also diminished phonatory activity in the adductor muscles.[6]

Variability in the outcome of botulinum toxin for ABSD is also related to the lack of standard measurement for documenting response to treatment. Edgar et al[7] performed multivariate analysis on several acoustic measures in patients with ABSD, to identify useful parameters. They found that voice onset time for voiceless consonants was significantly longer for ABSD patients than for normal subjects ($P = 0.15$) and identified the best acoustic parameter to be perceptual counts of breathy voice breaks. However, most reports of botox therapy for ADSD have designated subjective patient response as the primary outcome measure.[2,8–10] Bielamowicz et al[11] found little correlation of subjective benefit with blinded measures of breathy breaks or perceptual ratings by speech pathologists. They speculated that these results could indicate inadequacy of external measures to account for improvement in the effort of speaking. Alternatively, patients were only reporting a placebo effect. They also suggested that ABSD could be a ''negative dystonia,'' with delayed or inadequate activation of adductor muscle activity at the onset of phonation. Inadequate active adduction could explain persistent breathiness after elimination of spasmodic abduction. However, another possibility is that the doses in that study were not optimal for diminishing PCA spasms. Variability in efficacy is undoubtedly related to differences in the doses prescribed as well as the muscles targeted. Various dosing schemes have been recommended, and most recommend beginning with a unilateral injection of 3 to 5 units, and then adding contralateral injections as needed.[2,11] In this protocol, most patients have required bilateral injection, and maximum dose has been limited by airway obstruction, because of suppression of inspiratory abduction of the larynx.

The authors began using botulinum toxin to treat patients with ABSD in 1990. In our initial group of patients, none obtained significant benefit from unilateral PCA injection. Addition of a small amount of botulinum toxin in the contralateral PCA was found to be dramatically effective in some patients. We noted that the abductor spasms were nearly always asymmetric, and that the amplitude was generally greater on the left side. Furthermore, we reasoned that because unilateral laryngeal paralysis does not commonly produce respiratory compromise in adults, patients should tolerate significantly higher unilateral doses than the doses that have been reported in previous studies with bilaterally symmetric injections. Subsequently, we adopted an asymmetric dose escalation protocol with 1.25 units in the nondominant side, and stepwise increases in the dose to the dominant side, to as high as 25 units.

This article recounts our clinical experience in managing ABSD patients. First, we present a retrospective review of pretreatment transnasal flexible laryngoscopy video examinations, which assesses asymmetry of abductor spasm. Second, we report the outcomes of 20 patients treated by an asymmetric dose escalation protocol for botulinum injection of the PCA muscles.

METHODS

Video analysis

We reviewed our transnasal flexible laryngoscopic archives of patients with ABSD or mixed SD and identified 23 pretreatment video segments that provided a clear view of the larynx during connected speech, including plosive consonants followed by