Novel therapeutic approach to relieve pharyngoesophageal spasm after total laryngectomy

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Total laryngectomy results in a variety of functional changes including the loss of normal laryngeal voice and alterations in swallowing physiology.[1] The current literature recognizes tracheoesophageal (TE) voice restoration as the preferred alaryngeal speech alternative in most patients with total laryngectomy. The method depends on the use of a unidirectional valved prosthesis that maintains the TE puncture and also allows pulmonary air flow into the esophagus for phonation while preventing aspiration during swallowing. Although total laryngectomy disrupts the neurophysiology of the oropharynx, patients who have undergone total laryngectomy rarely report significant swallowing impairments. The most common reason for TE speech failure after total laryngectomy is constrictor muscle hypertonicity or what has been referred to as pharyngoesophageal spasm (PES). PES is known to increase peak intraesophageal pressure measurements during phonation and is correlated with levels of TE speech fluency.[2] PES has also been shown to impede cricopharyngeal functioning, resulting in problems with food transit during swallowing as well as preventing the superior egress of airflow for sound production.[3]

Although several methods have been used with variable success to relieve PES, chemical denervation of the PE segment by using *Clostridium* botulinum toxin A (BTA) that acts on the presynaptic cholinergic nerve fibers to prevent the release of acetylcholine at the neuromuscular junction has become the treatment of choice to facilitate TE speech and swallowing after total laryngectomy. Currently, BTA is injected percutaneously into the pharyngeal constrictor muscles along 1 side of the neopharynx just superior and lateral to the stoma. The optimal technique uses electromyographic (EMG) guidance to the site of injection that was previously marked by the speech pathologist during videofluoroscopic recording. However, percutaneous injection is not always possible, particularly in patients with significantly distorted cervical anatomy, severe postradiation fibrosis, postural difficulties that prevent accurate injection, or anxiety or the inability to tolerate the procedure. Therefore, the purpose of this case series is to assess the efficacy of the BTA injection through the EGD in patients with PES.

METHODS

Eight sessions of BTA injections were performed through the EGD in 4 laryngectomized patients (3 male, 1 female) with TE puncture. All patients had decreased TE speech fluency because of PES[3]; 3 patients also had dysphagia because of evidence of a shortened duration of PES relaxation during swallows on videofluoroscopic examination. Videofluoroscopic examination was performed by the speech pathologist before each injection to identify the location of the spasm.[3] The location of the spastic segment was identified at the time of EGD by transillumination of the marked area on the skin surface of the neck identified as the area of spasticity by the speech pathologist at the time of videoflouroscopic recording. Additionally, during endoscopy, nonstrictured smooth narrowing/spasticity above the TE prosthesis was identified by resistance to air insufflation without resistance to the passage of the endoscope. A total of 100 IU of BTA (Botox [onabotulinumtoxin A]; Allergan, Irvine, Calif) were injected into 4 quadrants of the PES segment through the EGD (Fig. 1). Our intention was to inject the muscular layer followed by gentle "massage" with back and forth movement of an 18-mm controlled radial expansion (CRE) balloon dilator (Boston Scientific Corp, Natick, Mass) to facilitate uniform distribu-

Abbreviations: BTA, botulinum toxin A; CRE, controlled radial expansion; EMG, electromyographic; MBS, modified barium swallow; PES, pharyngoesophageal spasm; SCC, squamous cell carcinoma; TE, tracheosophageal.

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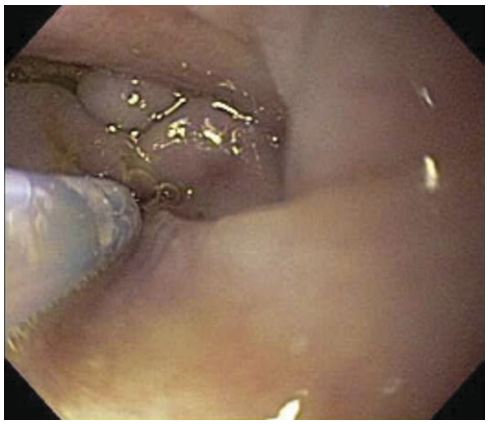

Figure 1. Botulinum toxin A injection through a gastroscope.

tion of the BTA. None of the patients had evidence of anastomotic stricture on modified barium swallow (MBS) or endoscopic view, and during the procedure, there was no resistance to the passage of the endoscope or to the back-and-forth movement of the CRE balloon dilator. All procedures were performed by using monitored anesthesia care.

TE speech fluency was rated before and after injection by the speech pathologist. Fluent TE speech was defined by the ability to produce 10 to 15 syllables per breath and sustain vowel production (/a/) for a minimum of 10 seconds.[3] Intraesophageal air insufflation was performed in all patients.[2] Fluent TE speech and/or a decrease in peak intraesophageal pressure defined improved TE speech production after injection. Diet level was graded according to the Performance Status Scale of Head and Neck Surgery normalcy of diet subscale in all patients before and after injection.[4] An increase in Performance Status Scale of Head and Neck Surgery normalcy of diet subscale score and/or decreased gastrostomy dependence defined improved swallowing function after injection.

Institutional review board approval was not required for this small case series.

RESULTS

Patient 1

A 67-year-old white man with a history of total laryngectomy for squamous cell carcinoma (SCC) of the larynx presented with phonation difficulty and dysphagia requiring gastrostomy tube feedings. The pretreatment MBS study showed PES (Fig. 2). His speech improved with percutaneous lidocaine injection at the PES segment performed in a clinical setting. Subsequent percutaneous BTA injection failed to improve speech or swallowing dysfunction. The first BTA injection through the EGD yielded no response. The decision was made to perform a second procedure because the patient had responded to the lidocaine injection. After the second BTA injection by using

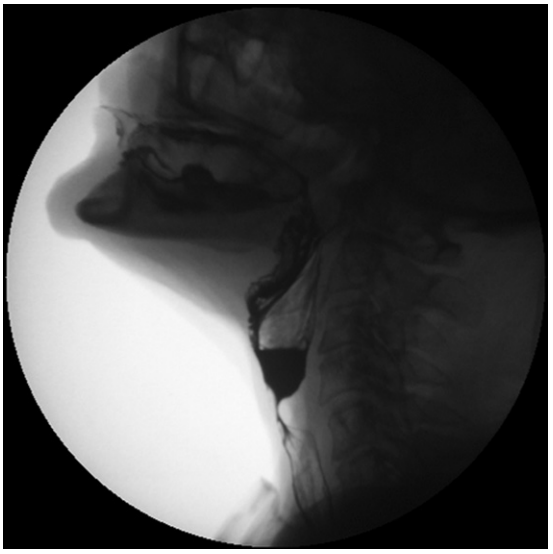

Figure 2. Incomplete bolus passage from a hypertonic cricopharyngeal bar.

Figure 3. Complete relaxation of the cricopharyngeal bar for bolus passage.

the EGD, his speech improved immediately with relaxation of the PES (Fig. 3), and he remained symptom free for 10.5 months. His dysphagia improved gradually, and he was weaned off gastric tube feeding. He responded to