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Oromandibular Dystonia: Long-Term Management With Botulinum Toxin

Catherine F. Sinclair, MD; Lowell E. Gurey, MD; Andrew Blitzer, MD, DDS

Objectives/Hypothesis: To review the long-term management of patients with oromandibular dystonia (OMD) treated using botulinum toxin.

Study Design: Retrospective chart review at a clinical research center.

Methods: Between 1995 and 2011, 59 patients with a diagnosis of OMD were treated with botulinum toxin. Data were collected on patient demographics, disease characteristics, and long-term treatment outcomes. Differences in management between an earlier published series of the first 20 OMD patients treated with botulinum toxin at this center and subsequent patients were analyzed.

Results: Patients were more commonly female (72% vs. 28%) with an average age at first botulinum treatment of 56.6 years. The median number of treatments was five (range, 1–35 treatments). Average time between treatments was 3.8 months ($\pm$5.2). Overall, 47.5%, had the jaw-closing form of OMD, which was associated with a preferential deviation to one side in 53.6%. These patients received initial injections to the masseter $\pm$ temporalis muscle; the external pterygoid was injected for associated lateral jaw deviation. Internal pterygoid injections were rarely used (3.4%). For the jaw-opening form, injections were initially administered to the external pterygoid, with the addition of anterior digastric for ongoing symptoms. When compared with patients in the older series, more patients since 1988 had treatments to the external pterygoid ($P = .001$) and anterior digastric ($P = .006$) in accordance with an increase in the diagnosis of jaw-opening OMD ($P = .05$).

Conclusions: Long-term management of OMD with botulinum toxin has minimal morbidity and is useful for all clinical forms. Injections can be titrated by dose and location to address the predominant muscle groups involved.

Key Words: Oromandibular dystonia, muscle dystonia, botulinum toxin, movement disorders.

Level of Evidence: 4

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INTRODUCTION

Oromandibular dystonia (OMD) refers to involuntary repetitive or twisting spasms of masticatory, lingual, and pharyngeal muscles. Clinically, it may present with jaw-closing oromandibular dystonia (JCOD), jaw-opening oromandibular dystonia (JOOD), lateral jaw deviation, or a combination of these abnormal movements.[1] It was first reported in 1910 and has an estimated annual incidence of 3.3 to 6.9 cases per 1 million people.[2–4] OMD can be idiopathic (focal or as part of segmental or generalized dystonias), tardive, or secondary to other movement or neurological disorders.[1,5] Focal OMD is rare, and OMD more commonly presents as part of a spectrum of segmental or generalized dystonia.[5–8] There is no known cure for OMD, and although oral medications may benefit approximately one-third of patients, none is universally effective and compliance is often limited by side effects.[9,10] In 1989, Blitzer et al. reported the first series of OMD patients managed with botulinum toxin injection, and since then, many other OMD patients have been managed at the same practice.[11] The purpose of the current article is to compare disease and management characteristics from this initial 1989 case series of 20 patients to a more recent series of 59 patients treated by the same otolaryngologist, and to use the lessons learned from almost 30 years experience in treating this condition to formulate a novel management scheme for use of botulinum toxin in OMD.

MATERIALS AND METHODS

Two groups of patients were compared in this study. For the new data subset, all patients who underwent botulinum toxin injection for OMD between October 1995 and March 2011 in the treatment rooms of the senior author were included. Disease and treatment characteristics for these patients were compared to data from the 1989 case series of the initial 20 patients with OMD injected with botulinum toxin by the senior author (Table I).[11] During their OMD work-up, all patients were reviewed by a neurologist and underwent full neurological testing, including brain imaging. Prior to toxin injection, all patients signed a written informed consent form.

From the New York Center for Voice and Swallowing Disorders, St. Luke's Roosevelt Medical Center, New York, New York, U.S.A.

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Send correspondence to Andrew Blitzer, MD, New York Center for Voice and Swallowing Disorders, St. Luke's Roosevelt Medical Center, 425 West 59th Street, 10th Floor, New York, NY 10019.
E-mail: ab1136@aol.com

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TABLE I.
Oromandibular Dystonia—Summary of Patient Demographic Data.

	New Data	Old Data	*P*
Total patients	59	20	.14
Female, no. (%)	40 (67.8)	17 (85.0)	
Male, no. (%)	19 (32.2)	3 (15.0)	
Age ≥60 years, no. (%)	8 (13.6)	12 (60.0)	.01
Prior oral medications, no. (%)	45 (76.3)	20 (100)	.02
Predominant symptoms, no. (%)			
Pain	12 (34.3)*		
Speech/eating impairment	19 (54.3)*		
Dental effects	7 (20.0)*		
Age, mean (SD), yr	56.6 (14.0)	59.7 (12.9)	.01
Female	51.3 (11.4)	60.8 (13.1)	
Male	50.8 (12.4)	53.3 (11.9)	
Symptom duration, yr	3.5	6.25	

*n = 35 due to missing patient demographic data.
SD = standard deviation.

Outcome Measures

Data from patients in the current series were analyzed for patient and disease characteristics, and botulinum toxin management specifics including dose, injection location, response to initial injection, and timing and characteristics of subsequent injections. These data and those from the older series of 20 patients were used to create a management algorithm for use of botulinum toxin in OMD.

Injection Technique

Lyophilized botulinum toxin A (BOTOX; Allergan Pharmaceuticals, Irvine, CA) was obtained and stored frozen at −20°C until reconstitution with sterile saline (without preservative) at the time of injection. The toxin was reconstituted to a concentration of 2.5 U per 0.1 mL or 5 U per 0.1 mL depending on the amount required for injection, where 1 U represents the median lethal dose for mice. Injections were performed using electromyographic (EMG) guidance. Visual inspection and palpation of muscular areas with maximal hypertrophy were used to guide initial placement of a 27-gauge, monopolar, Teflon-coated, hollow EMG recording needle. The recording needle was connected to the EMG machine, and needle placement was confirmed by EMG evidence of contractions during voluntary muscle activation. Feedback from the EMG machine was then used to target the injection to the areas of maximal muscular activity. Injections were individualized for each patient based on clinical type of OMD and symptom severity. For JCOD, injection into the masseter and/or temporalis muscle was performed percutaneously, with temporalis injection being directed to the anterior and mid muscle belly. Injection of internal pterygoid was performed percutaneously from below, with the needle inserted under the angle of the mandible and directed superiorly using EMG control. For JOOD, injection into the external pterygoid (EP) was performed via an intraoral approach. The pterygomandibular raphe was palpated intraorally, and the EMG needle was inserted into the EP between the raphe medially and mandibular ramus laterally, posterosuperior to the last maxillary molar tooth. Needle placement was verified by electrical activity on side-to-side jaw motion. The BOTOX injection was spread throughout the length of the muscle, with the needle directed toward the mandibular condyle. Anterior digastric was injected percutaneously using the EMG trace to ensure correct needle placement. Injection should be placed superficially, closer to the mentum than hyoid, to minimize risk of toxin diffusion into the base of tongue and deep submental musculature. The platysma was injected percutaneously by using very superficial needle placement while the patient grimaced to facilitate identification of platysmal bands. If lateral deviation OMD was present concurrently or as an isolated symptom, injection was into the EP using the technique described above with or without anterior temporalis injection.

The paradigm used for subsequent injections was that if patients had <50% improvement on a 1% to 100% of normal function rating scale (validated and previously reported for dystonia)[12] 2 weeks after initial injection, they were given an additional dose of the same amount of toxin into previously injected muscles with or without injection to additional related muscle groups. If these patients subsequently still had <50% response, no further toxin was given. If patients had between 50% and 80% response, they received additional injections into the same muscles with dose increases of 5 to 10 U and/or additional injection into associated muscles. Those patients who had >80% response to initial dose were given the same dose for subsequent injections, with the timing of injection based on return of clinical symptoms (Table III). With regard to the number of injections per muscle, patients were treated with five points in each masseter and temporalis, three points in each external and internal pterygoid and platysma, and one to two points per anterior digastric. From the point of injection, the toxin diffuses 1 to 1.5 cm, and thus successive injections into a single muscle should be placed 1 cm away from any prior injection site in three dimensions.

RESULTS

Patient Characteristics

There were 59 patients included in the recent study, with a 2:1 female-to-male predominance and average age at diagnosis of 56.6 years (i.e. Table 1). Demographic data on clinical symptoms at presentation and classification of OMD into focal, segmental, or generalized form were unavailable for 24 patients due to inadequate chart documentation. Data for all other patients, diseases, and treatment demographics were complete. Most patients (91.5%) had idiopathic (primary) OMD, with five patients having a secondary or tardive form. At diagnosis, patients had been symptomatic for an average of 3.5 years with the predominant symptom being functional impairment of speech, chewing, and eating. When compared with data from the pre-1988 series, patients treated since 1995 were significantly younger and had less frequently been on long-term oral medications. All of the patients except one, who had not previously trialed oral medications, had focal OMD of varying types. Mean follow-up was 4.3 years (±3.4 years). Twenty patients (33.9%) were followed for >5 years.

Disease Characteristics

Disease characteristics are noted in Table II. Two-thirds of patients had OMD as part of a segmental or generalized dystonia syndrome. Almost half of the patients (28 patients, 47.5%) had JCOD, and of these, 53.6% (15 patients) had some degree of lateral jaw deviation. Similarly, six of the 21 patients (28.6%) with JOOD had a lateral component. Ten patients (16.9%) had

TABLE II.
Disease and Botulinum Toxin Treatment Characteristics.

	New Data	Old Data	*P*
Other muscles, other disease, no. (%)*			
No, focal OMD alone	12 (34.3)†	4 (20.0)	.25
Yes	23 (65.7)†	16 (80.0)	
OMD type, no. (%)			
Closing	13 (22.0)	18 (90.0)	.03
Closing with lateral deviation	15 (25.4)		
Opening	15 (25.4)	2 (10.0)	
Opening with lateral deviation	6 (10.2)		
Lateral deviation	10 (16.9)		
Muscles treated, no. (%)‡			
External pterygoid	49 (83.1)	7 (35.0)	.001
Masseter	31 (52.5)	18 (90.0)	.002
Temporalis	25 (42.4)	12 (60.0)	.18
Digastric (anterior belly)	17 (28.8)	0	.006
Platysma	7 (11.9)	2 (10.0)	.82
Medial pterygoid	2 (3.4)	3 (15.0)	.44
Genioglossus/hyoglossus	0	2 (10.0)	.01
Injection frequency, mean (SD), mo			
Overall	3.8 (5.2)		>.05
Jaw-closing OMD (± lateral deviation)	3.9 (6.6)		
Jaw-opening OMD (± lateral deviation)	4.0 (2.5)		
Lateral deviation OMD	3.5 (4.5)		
Botulinum toxin initial dose, mean (SD), U			
External pterygoid	9.6 (4.3)		
Masseter	19.7 (7.2)		
Temporalis	15.8 (7.1)		
Digastric (anterior belly)	4.6 (1.0)		
Platysma	7.5 (2.2)		
Medial pterygoid	20 (0)		
No. of treatments per patient, median (range)			
Overall	5 (1–35)		.30
Jaw-closing OMD (± lateral deviation)	8.0 (1–35)		
Jaw-opening OMD (± lateral deviation)	4.5 (2–26)		
OMD with lateral jaw deviation alone	2.5(1–12)		

*Includes patients with segmental or generalized dystonia or orobuccolingual dyskinesia.
†n = 35 due to missing patient demographic data.
‡The number of patients with that muscle injected with botulinum toxin at some stage during their disease course.
OMD = oromandibular dystonia; SD = standard deviation.

documented concomitant lingual involvement, which was significantly more common among JOOD patients (eight vs. two patients, $P = .01$). Compared with the older data set, significantly more patients in the current series had JOOD ($P = .03$). There was no correlation between dystonia form and gender, predominant clinical symptoms, age of symptom onset, or focal versus segmental/generalized presentation ($P > .05$).

Botulinum Toxin Dosing and Injection Characteristics

Dosing characteristics for each muscle group are shown in Table II. Larger muscles, such as the masseter and temporalis, received higher initial toxin doses than smaller muscles, such as the anterior digastric and EP. Median initial doses were 25 U to the masseter (range, 5–30 U), 15 U to the temporalis (range, 5–25 U), 7.5 U to the EP (range, 2.5–25 U), 5 U to the anterior digastric (range, 1.5–5 U), and 7.5 U to the platysma (range, 5–10 U). Both patients having medial pterygoid injections received 20 U per muscle. All except one platysma injection were performed on patients with JOOD. Significantly more patients in the current series received EP and anterior digastric injections when compared to data from the 1989 case series. Most patients received injections of the same dose of toxin bilaterally to identical muscle groups. Eleven patients (18.6%) undergoing EP injections had different initial doses given per side

TABLE III.
Treatment Algorithm Based on Oromandibular Dystonia Type.

Muscles for Initial Injection*	Subsequent Injections
Closing	
Masseter 25 U (50 U if hypertrophy):	If no response: 1) consider internal pterygoid (external injection) 10U, 2) double initial dose to previously injected muscles; if partial response: 1) consider internal pterygoid (external injection) 10 U, 2) increase masseter ± temporalis dose by 5–10 U
→ Consider anterior/mid temporalis 15–25 U;	
→ If concurrent lateral deviation, add 7.5 U external pterygoids (intraoral) and consider anterior portion temporalis 15–25 U	
Opening	
External pterygoid 7.5 U:	Consider anterior digastric 5 U if not previously injected; if no response, double initial dose to previously injected muscles; if partial response, increase external pterygoid dose by 5–10 U
→ Consider anterior digastric 5 U;	
→ If concurrent lateral deviation, consider anterior portion temporalis 15–25 U	
→ If concurrent platysmal contraction, consider platysma 7.5 U	
Lateral deviation	
External pterygoid 7.5 U:	Consider anterior temporalis if not previously injected; if no response, double external pterygoid dose; if partial response, increase initial external pterygoid dose by 2.5–10 U depending on level of desired weakness
→ Consider anterior temporalis 15–25 U	
→ Depending on predominant direction of jaw movement, consider higher dose to contralateral external pterygoid and ipsilateral anterior temporalis	

*The number of injection points per muscle is five for each masseter and temporalis, and three for each external and internal pterygoid/digastric/platysma.

based on degree of jaw deviation. The median number of treatments per patient was five (range, 1–35 treatments), with the most treatments per patient occurring for JCOD patients (Table II). Twenty-nine patients (49.2%) had five or more botulinum toxin injections, and 20 patients (33.9%) had two or fewer treatments.

Overall, 26 patients (44.1%) returned for reinjection within 1 month of their initial toxin dose (11 JCOD, 11 JOOD, four lateral deviation). These patients were managed according to the algorithm shown in Table III. Thirty-nine patients (66.1%; 17 JCOD, 17 JCOD, five lateral deviation) had >2 injections overall, and 23 of these patients (59.0% or 39.0% of total) did not require dose adjustment within the first month of treatment. Mean time between initial and subsequent injection for these 23 patients was 4.2 ± 2.0 months, with 11 patients receiving subsequent dose increases (± additional muscles groups added), three having dose decreases, and nine having equivalent doses to initial injection. Ten patients had only a single treatment (16.9%; six JCOD, one JOOD, three lateral deviation), and 10 patients (five JCOD, three JOOD, two lateral deviation) had only two injections in total, the second being performed within 1 month of initial injection. Genioglossus and hyoglossus were not injected in this series. Two patients in the older data set did receive genioglossus and hyoglossus injections, resulting in significant dysphagia. Apart from incomplete response to initial injection necessitating dose increase and/or treatment of additional muscle groups, there were no complications reported by patients in the current series. There was no correlation between clinical form of dystonia and total number of injections per patient or time between injections ($P > .05$).

Management Algorithm

A treatment scheme based on our experience with this disorder over the last 29 years is proposed in Table III. Although in general, bilateral muscle groups can be injected with the same dose, but the suggested doses per muscle per side may need to be tailored individually, especially when unilateral pathology predominates.

DISCUSSION

OMD is a rare neurological condition that may present focally, or more commonly, as part of a segmental craniocervical or generalized dystonia syndrome.[6] A diagnosis of OMD carries with it significant quality of life issues due to its effects on chewing, swallowing, and talking with resultant social embarrassment and cosmetic disfigurement.[13,14] Oral trauma often results from wear or early loss of teeth due to persistent grinding and tongue biting, and dental appliances are useful in some patients.[1] As with other forms of dystonia, OMD may be alleviated by afferent proprioceptive sensory inputs (sensory tricks) including relaxing, talking, singing, humming, lip biting, tongue posturing, swallowing, and chewing gum.[3,7] Spasms are absent during sleep and generally aggravated by stress. Diagnosis may be challenging, and early misdiagnosis has been common in the past, particularly among the dental community.[3,11,15,16] Over the past 20 years, there have been many reports of the use of botulinum toxin in OMD, particularly for those patients with symptoms refractory to systemic medications[3,11,14–17] The initial injection of botulinum toxin for OMD was performed in 1983, with the first case series being published in 1989.[11] At the beginning of this study, there were no standard guidelines for the treatment of OMD, and thus the reported approach to treatment was empirical, beginning with small doses and titrating them according to patient symptoms. Experience with patients from that study and over subsequent years has enabled refinement of technique and dosing characteristics and has made it possible to

formulate a treatment scheme for the different forms of OMD as outlined in Table III. Specific changes in management have included avoidance of injection to submental or floor of mouth muscles (apart from superficially into the anterior digastric), performance of all EP injections intraorally (as described in the Materials and Methods section), and refinement of dosing and sequence of muscle injection for each clinical OMD form.

The type of OMD is the best guide as to which muscles should be treated initially, and severity of symptoms serves as the best guide of botulinum toxin dose required. EMG guidance facilitates accurate needle placement and is recommended, despite not having been used in a large prior prospective trial.[1] The anatomy of the muscles affected by OMD has been described in a previous publication.[14] The EP muscle is responsible for depressing the mandible and is thus the predominant muscle involved in JOOD. It can be approached internally or externally; however, we find the internal approach with EMG guidance more accurate and reliable, as it allows toxin distribution along the length of the muscle for maximal effect with less chance of diffusion to other palatal muscles. Submental muscles (especially anterior digastric) also contribute to mouth opening. Care must be taken when injecting in this region due to a risk of significant postinjection dysphagia. This risk is minimized by ensuring that injections are superficial and located closer to the mentum than hyoid, thus minimizing toxin diffusion into tongue base muscle attachments. Treatment of JOOD is often reported to result in higher complication rates than JCOD[1,18]; however, we did not observe this in the current study likely due to avoidance of deep high-dose injections into the submentalis complex and use of EMG guidance to ensure accurate needle placement. EP also plays an important role in grinding motions and thus contributes to forms of OMD with a component of lateral jaw deviation, as do the anterior fibers of temporalis. The masseter is a strong elevator of the mandible and is active in JCOD, with occasional contribution by temporalis (anterior/midfibers) and internal pterygoid. Although lingual and pharyngeal musculature may be affected by the dystonic process, and injection of these muscles has been reported,[11,14] success rates are low, and there is a high risk for undesirable side effects, particularly severe dysphagia as noted in the old data set where two patients received hyoglossus and genioglossus injections (Table II).

The pathophysiological mechanism of dystonia is unclear, but likely involves defects in the basal ganglia causing loss of physiological inhibitory control over the thalamus and brainstem with subsequent dysregulation of centrally mediated movements.[19] As seen in this series, it is more common in females with a mean age of onset between 31 and 58 years.[1,20,21] Compared with the 1989 study, the average age of patients in the current report was significantly younger, and prediagnosis symptom duration was significantly shorter. This likely reflects an increase in early diagnosis of all forms of OMD, possibly due to an increased awareness of the condition among primary care physicians, otolaryngologists, and dental specialists to whom these patients initially present. Also, significantly fewer patients in the current series were on oral medications at the time of presentation to the clinic, and all except one of those patients not on medication had focal OMD of varying forms. This finding may again reflect more widespread knowledge about management options for focal forms of OMD within both medical and general public communities, and a desire to avoid systemically active oral medications in favor of a local management option with minimal side effects. It may also represent a slight change in treatment paradigm with preferential use of toxin over systemic medications in isolated focal OMD. Regardless, all OMD patients in the current series were referred to a neurologist for evaluation and consideration of oral medications.

Concerns regarding decreased potency of toxin injections over time or of development of antibodies to toxin with prolonged use are not supported by the current data, where the median number of treatments was five and almost half of patients treated had five or more injections over time with mean follow-up of 4.3 years. In the 1989 series, patients completed self-rating scales for functional activity to assess treatment response.[11,12] Forty percent had 0% to 20% improvement, and 60% had >50% improvement. Unfortunately there were no quantitative data available for the current series, and the presence or absence of improvement was determined from documentation in patient charts combined with our paradigm for subsequent toxin injections as described in the methods section and outlined in Table III. Using this paradigm, patients who had a <50% functional improvement after initial injection returned within 1 month for reinjection, and if they still had <50% improvement, no further toxin doses were given. Thus, at least some of the 20 patients (33.9%) in the current series who had two or fewer injections were likely to be poor responders, and this percentage of nonresponders correlates well with data of the older series and suggests that over 60% of patients treated can expect to have >50% functional improvement. Not all of the patients in the current series who had two or fewer injections were necessarily poor responders; however, patients often travel from distant locations for their first few injections before following up for subsequent injections with more local physicians. Thus, 33.9% is likely an overestimation of toxin ineffectiveness. JOOD is considered more treatment resistant than JCOD, predominantly due to a higher incidence of concomitant lingual involvement.[1,7] Although JOOD patients did have more frequent lingual involvement in the current series, we did not find significant differences between JOOD and JCOD in number of injections per patient, frequency of injections, or extent of subsequent dose increases.

This is a retrospective study and thus has limitations. Although all patients did complete functional rating scales after each toxin injection, these data were unavailable, therefore the response of an individual patient to their botulinum toxin injection could only be inferred from the standard treatment paradigm used (as described in the Materials and Methods section) and from documentation in patient charts. Without a

definitive quantitative scale it is difficult to compare treatment response between the different subtypes of OMD and between the current series and pre-1989 series, which did include a functional rating scale. It would also have been interesting to compare the older and newer series with regard to initial dose of toxin applied to certain muscles, and average time between these injections; unfortunately, these data were unavailable for the older patient series.

CONCLUSION

Long-term management of OMD with botulinum toxin has minimal morbidity and is useful for all clinical forms, including JOOD, JCOD, and lateral jaw deviation. Injections can be titrated by dose and injection site to address the predominant muscle groups involved. Injection into submental, lingual, and pharyngeal musculature should be avoided due to the high risk of severe dysphagia.

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