Letter to the Editor

I would like to comment on the article "Botulinum Toxin Type A for Treating Temporomandibular Joint Dysfunction" (Miller TD, Neuhaus IM, Zachary C. *Cosmet Dermatol.* 2008;21:104-106). This article is a nice description of a single-patient treatment with botulinum toxin type A (BTX-A) for temporomandibular joint (TMJ) dysfunction. Although the article may represent the first description in dermatologic literature, treating TMJ with BTX-A has been well documented in oral and maxillofacial surgery literature for many years.

As Miller et al[1] state, TMJ dysfunction is a poorly understood syndrome that is related to many factors, including osteoarthrorois, TMJ disc displacement, occlusal abnormalities, inflammatory mediators, and spasms of the masticatory muscles. The main complaints with TMJ dysfuction are pain, clicking, popping and crepitus, locking of the TMJ, and muscular pain that is exacerbated by movement. This complex disorder is treated by conservative therapy including heat, physical therapy, acrylic splints, occlusal adjustment, anti-inflammatories, and muscle relaxants. More complex treatments include intra-articular steroid injection, lysis and lavage of the TMJ, arthroscopy, and open-joint surgery. Advanced cases may require total TMJ replacement.

The advent of BTX-A has provided an additional powerful tool to deal with the conservative treatment of the muscular component of TMJ. The authors treated their patient with success, but required 7 treatment sessions. In addition, the injections were made while the patient's mouth was open.

Before limiting my oral and maxillofacial surgery practice to cosmetic facial surgery, I had a profound interest in TMJ dysfunction and facial pain. I have treated many TMJ patients with BTX-A during the past 12 years and would like to expound on the technique used by Miller et al.

Bruxism, or grinding the teeth at night, is one of the hallmarks of TMJ dysfunction, and patients who wear acrylic mouth guards at night often grind through the plastic. BTX-A can decrease bruxing by decreasing the bite force. I have worked with local prosthodontists who have performed bite-force measurements before and after BTX-A injection, revealing that the bite force can be significantly decreased. By decreasing the strong function of the masseter muscle, the TMJ can be relieved from the heavy load of the bite force. The BTX-A assists by decreasing the microtrauma from the heavy grinding and by decreasing the spasm within the treated masseter. I speculate that there may be other unknown systemic effects of BTX-A injections that assist muscle function and dysfunction.

My injection sequence is somewhat different from the authors' description. I prefer to inject the masseter while the patient's mouth is clenched. By asking the patient to clench the mouth, it becomes easier to palpate the most active, hypertrophic, and tender areas of the masseter. The masseter is composed of 2 bellies, and because I use a significantly higher dose of BTX-A when treating TMJ dysfunction, the bulk and mass of the masseter allows for safe injection of higher doses than many other commonly treated areas. The flexed hypertrophic masseter muscle has several prominent projections of muscle bulk, and after the patient's mouth is clenched, I mark the greatest prominence of the clenched muscle and inject 10 U of BTX-A into the deep muscle belly. While the patient continues to clench the mouth, I inject another 5 U into the next largest prominence, sometimes injecting into a third. I have injected as much as 50 U per side, although 20 to 30 U is the more common amount during the first appointment. I then have the patient return in 2 weeks to repeat the process. My end point is pain resolution and decrease of bite force, with a third injection rarely performed. In my experience, the BTX-A effects seem to last much longer when treating the masseter than the cosmetic paralysis seen in the upper face. I also frequently inject the temporalis muscle, which is also a large mass muscle. TMJ dysfunction patients can usually pinpoint pain foci in the temporalis muscle, and 5 U of BTX-A in multiple areas can augment the masseter injection. Although the lateral and medial ptergoid muscles are also involved in TMJ and myofacial pain dysfunction, they are more difficult to inject, especially the lateral ptergoid, without using electromyography. Finally, TMJ dysfunction patients have other head and neck concomitant muscle tenderness and spasm. Injecting small amounts of BTX-A (eg, 2–3 U) in the occipital or posterior nuchal regions also seems to be effective. I cannot quantify this, and admittedly my findings are empirical.

I congratulate the authors on shedding light on this simple treatment for this complex disease process and hope that other practitioners are made aware of this option to assist their patients.

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REFERENCE

1. Miller TD, Neuhaus IM, Zachary C. Botulinum toxin type A for treating temporomandibular joint dysfunction. *Cosmet Dermatol.* 2008;21:104-106.