Botulinum Toxin A in the Treatment of Myofascial Pain and Dysfunction: The Case Against Its Use

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Since the Food and Drug Administration approval of botulinum toxin (BT) for the temporary treatment of blepharospasm and strabismus in 1989 and for the treatment of cervical dystonia in 1984, it has been used clinically to treat a variety of other conditions possibly involving muscle spasm or hyperactivity. These have included low back pain, whiplash-associated neck pain, chronic migraine of cervical origin, chronic tension-type headache, facial tics, orofacial dyskinesia, masseteric hypertrophy, bruxism, and myofascial pain. Despite its wide application, the literature supporting its efficacy in many of these conditions is weak, consisting mainly of uncontrolled, open-label studies rather than double-blinded, randomized clinical trials. The purpose of this report is to present the arguments against the use of BT in the management of myofascial pain and dysfunction (MPD) involving the muscles of mastication.

Arguments Against the Use of BT in MPD

Ideally, in selecting a treatment for any condition, the decision should be based on the therapy addressing the etiology of the problem rather than the symptoms involved. In the case of BT for treating MPD, this has not been the situation. Currently, the etiology of masticatory muscle pain and dysfunction remains unknown and the therapeutic use of BT is based on several different unproved theories. Probably the most frequently cited theory is that the pain and dysfunction are caused by muscle hyperactivity. The problem with this concept is that electromyographic studies of the muscles of mastication in patients with MPD do not always show an increase in resting muscle activity. Therefore, on this basis, the use of BT to produce muscle paresis or paralysis as a treatment is questionable.

Most studies in the literature supporting the use of BT for the management of myogenous pain and dysfunction have been open-label trials in a heterogeneous group of patients. Those studies that have used a prospective, randomized, double-blinded approach provide the opposite answer. For example, Wheeler et al tested BT against placebo for chronic neck pain and found no difference. Likewise, Nixdorf et al found no difference in pain intensity and muscle tenderness in a group of female patients with chronic moderate to severe jaw muscle pain. As would be expected, those who received BT had less maximum mouth opening than those who received placebo because of the muscle weakness. A more recent randomized, double-blinded, placebo-controlled study on the therapeutic effects of BT in treating neck and upper back pain of myofascial origin also showed no difference in the visual analog pain scale values and the neck disability index between the 2 groups. In the most current randomized, controlled, double-blind, crossover study, which compared BT with isotonic saline injections for persistent temporomandibular myofascial pain disorders, the results showed no clinically relevant effect for BT. The investigators concluded that BT type A injections are not an effective adjunct to conventional treatment in such patients.

A second proposed theory for the etiology of MPD suggests that there is inflammation in the muscle causing the pain and dysfunction and that decreasing muscle activity decreases this inflammatory process. This concept is based on the clinical observation that BT injections can relieve tension-type headaches, which are supposed to have an inflammatory basis. The use of BT for MPD is based on an unproved concept, because there is no evidence that it is caused
by myositis. Moreover, although animal studies have shown that BT not only has a paralytic effect on muscle by inhibiting the release of acetylcholine from the motor nerve endings at the neuromuscular junction, but also has an independent analgesic effect by blocking the action of such neurotransmitters as substance P, glutamate, and calcitonin gene-related peptide on peripheral nociceptors, it does not have an anti-inflammatory effect.

Another proposed explanation for the development of MPD is a chronic tooth clenching-and-grinding habit. On this basis, paralyzing the muscles of mastication should be effective in resolving the problem, and there are studies to show its effectiveness in such situations. However, bruxism can be managed just as effectively and with less risk using a bite appliance, which involves a single rather than multiple treatments. Moreover, although there are no significant risks involved using a proper bite appliance, this is not true for BT. Not only can the injection of BT into the muscles of mastication be accompanied by the same minor complications as any other intramuscular injection (pain, bruising, and swelling), but also there may be more serious complications. About 1% of patients who receive BT may develop severe, debilitating headaches that can persist for several weeks. A small group of patients also may develop antibodies, especially when receiving BT at frequent intervals. Another risk is muscle atrophy and possible deformity from the long period of decreased muscle activity, particularly when using multiple injections as would be necessary to control bruxism. This has been reported as an hourglass deformity occurring in the temporalis muscle from multiple injections used to treat chronic tension headache and in the intrinsic muscles of the hand from intrapalmar injections for the treatment of hyperhidrosis. Neurogenic muscle atrophy also has been shown in biopsy specimens from the gastrocnemius muscle in healthy volunteers up to 1 year after a single BT injection. An even more serious complication of BT is the development of paresis or paralysis in areas adjacent to the primary injection site, with the potential for causing difficulty in swallowing and respiratory problems.

The final theory on the cause of MPD relates to the presence of trigger points in the muscle. These are described as hyperirritable spots that are associated with palpable nodules in taut bands of muscle fibers. Although there are reports in the literature regarding their identification in the larger skeletal muscles, there is no consistency in the diagnostic methodology used, and there is no single theory on how they arise. Moreover, those areas of tenderness that occur in the masticatory muscles do not have the characteristics that would classify them as trigger points. However, even if they are trigger points, the recommended spray and stretch techniques, manipulative therapy, and local anesthetic injections used in treating these patients seem preferable to the use of muscle paralysis. Supporting this idea is a systematic review of the literature performed in 2007, where 4 of the 5 randomized controlled studies identified in which BT was compared with placebo for injection of trigger points causing myofascial pain concluded that BT was not an effective treatment.

Another consideration in evaluating the results of trigger point injections is whether any positive results are actually due to the BT or merely to a needling effect. In an attempt to answer this question, Cummins and White performed a systematic review of the literature in 2001 and concluded that dry needling of myofascial trigger points is just as effective as injection of an active drug. In a more recent prospective, randomized, controlled study, Ay et al also found that dry needling of trigger points in patients with cervical myofascial pain was just as effective as a local anesthetic injection in improving pain and range of motion. Thus, there appears to be no reason to use BT for injection of trigger points.

In summary, there are several reasons why BT should not be used for the treatment of MPD involving the masticatory muscles. First, the mechanism of action of BT does not address the etiology of the problem, but rather attempts to manage the symptoms. Moreover, there are insufficient prospective, randomized, double-blind, placebo-controlled studies to show that it is effective even for this purpose. A second reason for not using BT injections is the fact that it generally represents a temporary rather than a permanent form of therapy, because muscle function returns in a period of weeks to months, usually requiring retreatment. Third, BT injections incur greater risks to the patient than most other forms of medical management that are equally effective in controlling MPD. The current recommendation of the American Association for Dental Research for treating patients with MPD states, “... unless there are specific and justifiable indications to the contrary, treatment of TMD patients should be based on the use of conservative, reversible and evidence-based therapeutic mo-
BT does not fit this description. The fact that there are still patients that some clinicians find difficult to manage is generally due to misdiagnosis and mistreatment rather than to a lack of effective therapies. As with any new form of treatment, clinicians are always seeking additional applications for its use. To paraphrase an old adage, “BT is a treatment looking for new diseases.” However, MPD should not be one of them!!

References