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Case Report

Efficacy of Botulinum Toxin-A Injections in Masticatory Muscles for the Management of Bruxism: A Clinical Perspective

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Abstract

Bruxism, characterized by involuntary, repetitive jaw-muscle activity including clenching and grinding of teeth, can lead to significant dental and muscular complications. Conventional treatments—such as occlusal splints and behavioral therapy-may not fully address muscular hyperactivity. Botulinum toxin type A (BoNT-A), a neurotoxin that inhibits acetylcholine release at neuromuscular junctions, has emerged as a promising therapeutic option. This article reviews clinical studies regarding the efficacy of BoNT-A injections into masticatory muscles for bruxism management, with a focus on clinical outcomes, safety, and practical considerations.

Introduction

Bruxism is a parafunctional activity involving repetitive contraction of masticatory muscles, which may occur during wakefulness (awake bruxism) or sleep (sleep bruxism). It affects approximately 8% - 31% of the general population, with sleep bruxism being more prevalent. The etiology is multifactorial, encompassing psychological stress, genetic predisposition, and disturbances in central nervous system neurotransmission. Chronic bruxism may result in tooth wear, Temporomandibular Joint Disorders (TMD), masticatory muscle hypertrophy, headaches, and orofacial pain.

Traditional interventions, such as occlusal appliances, cognitive behavioral therapy, and pharmacological agents (*e.g.*, muscle relaxants, anxiolytics)-have variable efficacy and patient compliance. In recent years, BoNT-A has gained attention as a neuromodulatory therapy capable of reducing muscle overactivity without compromising muscle function entirely.

Mechanism of action of BoNT-A

Botulinum toxin type A acts by cleaving SNAP-25, a synaptosomal-associated protein required for acetylcholine vesicle fusion and release at the neuromuscular junction. This inhibition results in a reversible chemodenervation and muscle relaxation that persists for approximately 3 to 6 months. When injected into hyperactive masticatory muscles (commonly the masseter, temporalis, and sometimes medial pterygoid), BoNT-A diminishes muscle contractility and alleviates symptoms associated with bruxism [1].

Clinical efficacy

Multiple randomized controlled trials (RCTs) and observational studies have demonstrated the clinical benefits of BoNT-A in managing bruxism:

- **Symptom reduction:** Studies show significant reductions in the frequency and intensity of bruxism episodes following BoNT-A injection. Polysomnographic studies confirm reduced electromyographic activity during sleep in treated individuals.
- **Pain relief:** Patients often report decreased masticatory and facial muscle pain [2], especially when bruxism is associated with myofascial pain syndromes or TMD [3].

- **Muscle hypertrophy:** Aesthetic and functional improvements, both clinician-assessed and patient-reported, have been documented in patients with masseteric hypertrophy secondary to chronic bruxism.
- **Tooth protection:** By attenuating muscle strength, BoNT-A may reduce mechanical wear on dentition and protect dental restorations.

One study by Guarda-Nardini, et al. [2] showed that patients receiving BoNT-A reported significant improvements in pain and bruxism indices compared to placebo over a 12-week period. Another RCT conducted by Shim, et al. [4] demonstrated similar efficacy with a sustained reduction in masseter activity and subjective bruxism scores [5].

Dosage and injection protocol

Dosing regimens vary, but commonly range from, typically, 20–30 units of BoNT-A are injected into the masseter and 10–20 units into the temporalis muscle per side [4]. Injections are typically performed using anatomical landmarks and Electromyographic (EMG) guidance is sometimes used to enhance injection accuracy. Clinical effects are observed within 3–7 days post-injection, peaking at around 2–4 weeks.

Safety and adverse effects

BoNT-A is generally well tolerated. Reported adverse events include mild, transient side effects such as local pain, hematoma, chewing fatigue, or temporary asymmetry. Systemic toxicity is rare when administered correctly. Long-term safety appears favorable based on currently available evidence, although muscle atrophy with repeated dosing should be monitored.

Limitations and considerations

Despite its efficacy, BoNT-A does not address the central neurological components of bruxism and may need to be combined with therapies such as behavioral therapy or occlusal splints. The need for repeated injections and associated cost may be barriers for some patients. More longitudinal studies are warranted to assess optimal dosing intervals, long-term outcomes, and potential resistance.

Conclusion

Botulinum toxin-A injection into masticatory muscles represents an effective and relatively safe treatment for refractory cases of bruxism, particularly when associated with muscular pain or hypertrophy. While not a first-line therapy for all patients, it offers significant clinical benefits for those unresponsive to conventional approaches. Further longitudinal and large-scale clinical studies are essential in the multidisciplinary management of bruxism.

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