# Effect on the mandibular bone of botulinum toxin in dentistry when applied to chewing muscles

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# **Keywords**

Neurotoxin Type A, Bone Loss, Muscle Atrophy, Temporomandibular Joint, Mandibular Condyle, Alveolar Process, Alveolar Bone Loss

# Abstract

The injection of neurotoxin A (BTX A) within the masticatory muscles, to SOURCE its temporary paralysis, may be an EXTENSIVELY used intervention for clinical disorders like or mandibular dystonia, sleep bruxism, and masseteric hypertrophy. Considering that contraction is required for mechano-transduction to take care of bone homeostasis, it's relevant to deal with the bone adverse effects related to muscle condition after this intervention. The objective of this text is to condense the present and relevant literature about lower jaw loss after BTX A intervention within the masticatory muscles.

# Introduction

Temporomandibular disorders (TMDs) are the foremost common sort of facial pain condition, affecting approximately 10% to 12% of the population, with about 25% of people having a minimum of one TMD episode during their lifetime.

TMDs, which fall into the category of musculoskeletal disorders, may be a collective term wont to describe variety of related conditions affecting the mandibular joint (TMJ) and masticatory muscles, all of which have three common symptoms: orofacial pain, joint noises, and restricted jaw opening during jaw movements like speaking or chewing. Many terms are wont to describe TMDs, including TMJ syndrome and TMJ disease; however, the term TMD is that the preferred term employed by the American Academy of Orofacial Pain (AAOP) and other professionals. Various classifications and clinical diagnostic criteria exist to explain TMD. Myogenic TMD, the more common form, only involves functional alterations of the masticatory muscles round the TMJ without TMJ problems. It always occurs in persons with high stress and is caused by nocturnal bruxism (teeth grinding) or day- or nighttime teeth clenching.

# Treatment

Since the first focus of pain is round the ear, the individual may first visit an ear, nose, and throat physician or otolaryngologist. Having eliminated the likelihood of headache, ear, or sinus problems, subsequent step is to think about the likelihood of TMJ pain and dysfunction. The dentist should be subsequent health care provider

that the individual seeks. Management of TMJ dysfunction may involve the utilization of medicines , one among the most recent treatment options for TMJ-induced muscle spasms is injection of neurotoxin.

When neurotoxin A (Botox ®) is injected in minute doses into the affected muscles round the TMJ, acetylcholine released from nerve terminals is inhibited, thus blocking neuromuscular transmission and rendering the muscle unable to contract. Unfortunately, this amelioration usually lasts for less than three to four months, so most patients require repeated injections over a few years. The long-term effects of this therapy can cause bone resorption. Here, we compile evidence to clinical studies, demonstrating that BTXA induced masticatory muscle atrophy promotes lower jaw loss. While bone loss has been detected at the condylar process or alveolar bone, cellular and molecular mechanisms involved during this process must still be elucidated. Further basic research could provide evidence for designing strategies to regulate the undesired effects on bone during the therapeutic use of BTXA. However, within the meantime, we consider it essential that patients treated with BTXA within the masticatory muscles be warned a few putative collateral lower jaw damage.

# **Patients and Methods**

Botulinum toxin A injection protocol was followed for the orofacial pain management within the sort of a freeze-dried powder (Onabotulinumtoxin A; BOTOX<sup>®</sup>, Allergan Inc., Irvine, CA, USA) was reconstituted at concentration of 20 units/mL (2 units/0.1 mL, 100 units in 5 mL of sterile saline), and used immediately after preparation. The BTX preparation was injected into each temporalis and superficial masseter muscle at a dose of 20 units and 25 units per muscle, respectively.

# **Experimental protocol**

Injections were performed within the 4 main masticatory muscles, that is, the 2 M. masseter and therefore the 2 M. temporalis muscles, no matter the symptoms being unilateral or bilateral, as is that the current practice for patients with TMDs. All injections were administered percutaneously and intramuscularly. Each injection was performed by employing a 1-mL syringe and a 26-gauge needle, at a dilution of 100 U/mL of injectable saline. Each patient received a complete dose of 100 U—30 U for every M. masseter and 20 U for every M. temporalis. Ten points of injection were performed for every patient—3 per M. masseter and a couple of per M. temporalis (Figure 1).



**Fig. 1.** Injection sites of botulinum toxin in masticatory muscles: 3 sites for M. masseter and 2 for M. temporalis.

The injection sites corresponded to areas of greatest muscle mass on palpation. The depth of the injection decided as follows: When the needle came into contact with bone, it had been removed for a distance of about 5 mm to inject BTX directly into the muscle.

## **Experimental protocol**

3-D condylar analysis showed significant bone changes in most of the patients within the study. Six patients presented bone changes within the anterior area of the condyle on the combined image: bone formation (in 3 patients) and bone loss (in 3 patients). Two patients had significant bone loss within the anterior portion of the proper condyle (Figure 5B). Seven patients had bone changes within the upper condylar por-tion: new bone formation in 5 and bone loss in 2. Seven patients presented bone changes within the posterior area: new bone formation in 3 and bone loss in 4. One patient presented new bone formation of the processus coronoideus on each side. No significant differences might be seen at the mandibular angle.



**Fig. 2.** (A) Color map of the 3-dimensional (3-D) model of a mandible showing surface differences at 1 year, expressed in millimeters on the color bar. Note the significant bone thick-ening at the left digastric fossa. (B) Color map of 3-D model of a right condyle showing a significant thinning at the ante-rior part at 1 year.

## Conclusions

In the present study, we found that 1 year after a BTX injection into masticatory muscles, bone changes were evidenced by texture analysis and 3-D reconstruction of the mandible. Half the study patients presented bone changes, with new bone formation or bone loss, depending on the bony territories and the redistribution of muscle strains. These bone changes can have clinical expression, and some recently published case reports tend to confirm this hypothesis. Mandibular bone-related adverse effects involve cellular and metabolic changes, microstructure degradation, and morphological alterations. While bone loss has been detected at the mandibular condyle or alveolar bone, cellular and molecular mechanisms involved in this process must still be elucidated. Further basic research could provide evidence for designing strategies to control the undesired effects on bone during the therapeutic use of BTX A. However, in the meantime, we consider it essential that patients treated with BTX A in the masticatory muscles be warned about a putative collateral mandibular bone damage

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