



ROLE OF BOTOX IN DENTISTRY

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Abstract

Botulinum toxin (BTX) commonly known as Botox, is a neurotoxic protein produced by the bacterium *Clostridium botulinum* and related species. Initially recognized for its therapeutic potential in treating conditions like strabismus and blepharospasm. Botox has been FDA-approved for various medical applications since its introduction. Its utility extends beyond traditional medical treatments to diverse dental procedures, including the management of temporomandibular disorders (TMD), bruxism, and clenching. Additionally, Botox has gained popularity in cosmetic dentistry for enhancing facial aesthetics by addressing issues such as wrinkles, gummy smiles, and black triangles. The toxin works by inhibiting acetylcholine release thereby reducing muscle contraction and glandular secretion. This minimally invasive treatment modality offers promising results with minimal trauma and discomfort for patients. As dental treatment options expand the application of botulinum toxin in dentistry provides a versatile, safe, and effective alternative for both aesthetic and functional dental conditions. Dental professionals with their detailed knowledge of facial anatomy are well-positioned to incorporate Botox into their practice offering innovative solutions for refractory or traditionally invasive conditions.

Key words: Botulinum toxin A, Temporomandibular joint disorder, Bruxism, Gummy smile.

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Introduction

Botox the commercial name for botulinum toxin is a powerful neurotoxin produced by the bacterium *Clostridium botulinum*. It is one of the most poisonous biological substances known significantly more toxic than common toxins like tetanus and curare. Despite its toxicity Botox has become widely used since its introduction in the 1980s especially in plastic surgery and its applications have expanded to fields like dentistry, dermatology, ophthalmology and general medicine.¹

Initially recognized for its therapeutic potential in 1981. Botox received FDA approval in 1989 for treating conditions such as strabismus, blepharospasm, and hemifacial spasm. By 2000 its use extended to treating cervical dystonia and cosmetic concerns like glabellar lines.² Botox works by inhibiting acetylcholine release at neuromuscular junctions causing temporary muscle paralysis that lasts about 3-6 months. This mechanism makes it effective for reducing facial wrinkles, alleviating pain and treating muscle-related disorders.^{3,4}

Botulinum toxin exists in several serotypes (A-G) with types A and B commonly used in medical treatments.⁵ These neurotoxins are produced as inactive polypeptide chains that become active when cleaved into heavy and light chains linked by a disulfide bond.⁶

In dentistry Botox is used for both therapeutic and aesthetic purposes. It is effective in treating temporomandibular joint (TMJ) disorders, bruxism and chronic facial pain. Aesthetically it helps address issues like gummy smiles, high lip lines and post-orthodontic muscle relaxation. Botox offers a minimally invasive solution, broadening the scope of dental practice and enhancing patient care.⁷

Botox's versatility and efficacy have made it a valuable tool in dentistry, providing innovative solutions for a range of dental and facial concerns. Its role continues to expand, driven by ongoing research and increasing acceptance in the dental community.

History

The history of botulinum toxin is fascinating tracing back to ancient times when it likely caused poisoning in humans. Between 1735 and 1793 in Germany several people died from consuming uncooked blood sausages initially thought to be poisoned by *Atropa belladonna* but later identified as botulinum toxin from pork stomach sausages called "Blunzen."⁸ German physician Justinus Christian Kerner studied botulinum naming the toxin "Sausage poison" though he couldn't identify the

biological source. In 1895 Emile Van Ermengem isolated *Clostridium botulinum* from ham that had poisoned people in Belgium naming it "Bacillus Botulinus." In 1949 Dr. Burgen's group discovered that botulinum toxin works by blocking neuromuscular transmission.⁹

During the World Wars botulinum toxin was refined for weaponization. In 1973 Alan B. Scott used botulinum toxin type A (BTX-A) to treat strabismus. By 1979 the FDA approved BTX-A for strabismus treatment, and in 1989 for blepharospasm and hemifacial spasm. Botox's use in oral and maxillofacial surgery began in 1982 with Jan Carruthers who used it to reduce muscle mass and smooth the skin. Its application expanded in 1990 for treating bruxism in patients with brain injury. The first paper on cosmetic uses of Botox was published in 1996. By 2002 Botox was FDA-approved for cosmetic use to improve glabellar lines. In the 1920s Sommer isolated botulinum toxin type A in a purified form and in 1946 Schantz isolated it in crystalline form.¹⁰

In 2000 the FDA approved Botox for treating cervical dystonia and in 2004 for severe underarm sweating (primary axillary hyperhidrosis).^{11,12} By 2010 Botox was approved for chronic migraine treatment. Throughout the mid-1990s Botox was used for treating crow's feet, temporomandibular disorders (TMDs) and masseter muscle injection.¹³ Over the years Botox has been approved for numerous medical conditions including chronic migraine, urinary incontinence and upper limb spasticity with ongoing research exploring new medical and cosmetic applications.¹⁴

Mechanism of Action

Botulinum toxins across all serotypes disrupt neural transmission by impeding the release of acetylcholine the primary neurotransmitter at the neuromuscular junction. When administered intramuscularly the toxin induces muscle paralysis by blocking acetylcholine release from presynaptic motor neurons. These toxins target four distinct areas in the body:

1. The neuromuscular junction
2. Autonomic ganglia
3. Postganglionic parasympathetic nerve endings and
4. Postganglionic sympathetic nerve endings.¹⁵

Temporomandibular disorder

Temporomandibular disorder (TMD) encompasses various conditions affecting the masticatory system, including issues with the temporomandibular joint and masticatory muscle dysfunction. Symptoms include facial pain, joint sounds, headaches, peri-auricular pain, neck pain,

and limited jaw movement often affecting both sides of the face. TMD is more common in women aged 20-40 and frequently involves a myogenic component with causes like bruxism, stress, oromandibular dystonia, and psychomotor behaviours. Traditional treatments such as intraoral appliances, occlusal adjustments, dental restoration and surgery are invasive, irreversible and expensive.¹⁶

Botulinum toxin A (BT-A) offers a viable conservative alternative by reducing muscle spasticity. Administered intramuscularly into the masseter and temporalis muscles under electromyographic or ultrasonic guidance. BT-A reduces bruxism, pain and inflammation improving mouth opening and parafunctional habits.¹⁷ It is also effective for treating recurrent mandibular dislocation by relaxing the lateral pterygoid muscles.¹⁸

Bruxism

Bruxism is characterized by repetitive clenching or grinding of teeth and jaw thrusting occurring while awake or asleep. This disorder leads to tooth wear, temporomandibular joint (TMJ) pain, muscle pain, and joint locking.¹⁹ Its exact cause is unknown, though psychological factors, emotional stress and malocclusion are suspected.²⁰

One of the earliest reports on use of botulinum toxin type A for bruxism was by Van Zandlicke and Marchau, demonstrated successful treatment in a brain-injured patient with severe bruxism using 100 U of BT-A injected into the temporalis and masseter muscles.²¹ Ivanhoe et al. found that injecting 200 IU of BT-A into the masseter muscle improved bruxism symptoms over 19 weeks.²² BT-A is recommended for sleep bruxism with a single injection lasting about a month.²³ Injections are guided by palpation during clenching with doses of 25-100 U per side injected into three sites in the thickest parts of the masseter muscles.²⁴

Gummy smile

A gummy smile characterized by an excessive display of gingival tissue greater than 3mm when smiling occurs in 7% of young males and 14% of young females. Several anatomical factors influence its appearance including lip length, crown length, vertical maxillary excess and oral muscular behaviour. The levator labii superioris alaeque nasi muscle is commonly involved in gummy smiles.²⁵ Various surgical techniques such as those by Rubinstein and Kostianovsky, Miskinyar and Rees and LaTrenta have been reported for correcting hyperfunctional upper lip elevator muscles but these are not commonly used. More prevalent surgical corrections include LeFort I maxillary

osteotomies for skeletal vertical maxillary excess and gingivectomies for delayed passive dental eruption with excessive gingival display.

Botulinum toxin (BT) has shown promising results for gummy smile correction. Small carefully titrated doses of BT can limit muscular over-contraction of the upper lip reducing gum exposure when smiling. Researchers at Yonsei University College of Dentistry in Seoul, Korea have proposed the "Yonsei point" for BT injections. This point is located at the center of a triangle formed by the levator labii superioris, levator labii superioris alaeque nasi and zygomaticus minor muscles. A dose of 3 U is recommended at each injection site.¹⁶

Masseteric hypertrophy

Chronic jaw clenchers often develop enlarged masseter muscles noticeably altering their facial appearance and making the jaw appear swollen and misshapen. Historically the primary treatment was surgical resection which could result in significant contracture. However, several clinical studies have demonstrated that small Botox injections (approximately 30 U per side) into the masseter muscles can effectively reduce hyperactivity. This treatment has led to a substantial decrease in muscle size with a maximum reduction of 35.4%. Once the underlying cause of hyperactivity is addressed the reduction in masseter hypertrophy tends to be long-lasting even after stopping Botox treatments.²⁶

Mandibular spasm

Mandibular spasm involves the muscles of mastication and other related muscles of the jaw leading to restricted mouth opening also known as "trismus." This condition can cause several issues such as difficulties in maintaining proper oral hygiene, performing dental procedures and carrying out functional movements like eating and talking. Injecting botulinum toxin into the targeted muscles induces paralysis effectively reducing muscle hyperactivity and spasms.²⁷

Trigeminal neuralgia

The International Headache Society defines trigeminal neuralgia (TN) as a condition characterized by recurrent, unilateral, brief electric shock-like pains that abruptly start and stop, confined to one or more divisions of the trigeminal nerve and triggered by harmless stimuli.²⁸ Botulinum toxin A (BT-A) has proven effective in treating trigeminal neuralgia without major adverse effects. It is becoming a preferred minimally invasive treatment over other invasive therapy. A study by Boru et al. found that injecting BT-A into the roots of the maxilla and mandible significantly

decreased pain severity and attack frequency in TN patients.²⁹ According to Elcio, BOTOX injections can substantially relieve the excruciating pain associated with trigeminal nerve inflammation. As an adjunctive treatment Botox reduces pain severity by acting on nerve endings.³⁰

Diagnostic Application

Botulinum toxin A (BT-A) can serve both prophylactic and diagnostic purposes in patients with chronic intermittent toothache. It helps determine whether the pain is muscular or pulpal. For instance, muscle pain from the anterior temporalis is often referred to the teeth. Treating this muscle pain with BT-A can prevent unnecessary and irreversible dental procedures.³¹

Application in Denture Wearers:

Jaw muscles can change in size, cross-sectional area and properties to adapt to functional demands. Patients struggling to adapt to new dentures due to irregular and uncoordinated muscle activity can benefit from botulinum toxin A (BT-A) treatment. For those who have been edentulous (toothless) for a long time, BT-A therapy provides muscle relaxation facilitating easier adjustment to new dentures.³²

Complications

The complications of botulinum toxin-A (BTA) injections can be categorized as systemic, local and reduced therapeutic effects due to antibody formation. Complications are more commonly observed when BTA is used for therapeutic purposes rather than cosmetic purposes due to the higher doses involved in therapeutic treatments.³³

A loss of treatment efficacy may occur due to the development of neutralizing antibodies against BTA. Approximately 7% of patients receiving BTA experience resistance, prompting the exploration of botulinum toxin B as an alternative therapy. The risk of producing neutralizing antibodies increases with frequent BTA injections over a short period, high doses and dose escalation of BTA injections. During the course of therapy most complications are local and usually mild.³⁴ These can include:

- Erythema
- Pain
- Ecchymosis at the injection site
- Mouth droop
- Dry eyes
- Asymmetry of facial expressions during dynamic movements
- Ptosis and lid edema
- Facial muscle weakness
- Transient dysphagia
- Infection

- Weakness
- Difficulty in chewing
- Aspiration risk during breathing
- Recurrent jaw dislocation
- Salivary duct calculi
- Xerostomia
- Nasal speech
- Nasal regurgitation
- Headache
- Blurred vision
- Dizziness
- Gastrointestinal upset
- Voice changes
- Local injuries to carotid arteries or branches of facial nerves¹³

Conclusion

The transformation of botulinum toxin from a deadly substance to a therapeutic drug marks a significant achievement in the field of medicine. Beyond its well-known cosmetic applications, botulinum toxin is also used for conditions such as TMJ, facial pain, and gummy smiles. Proper knowledge of administration and procedures is essential to avoid complications. The success of botulinum toxin treatments hinges on patient satisfaction.

However, there are unresolved issues and concerns with this agent, including the lack of standardization in the biological activity of different preparations, a limited understanding of toxin antigenicity and variations in injection methods. These factors can lead to occasional complications typically involving muscle weakness and the high cost remains a significant drawback that needs to be addressed in the future.

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