SKIN

IN-DEPTH REVIEW

Clinical Approach to Botulinum Toxin in Cosmetic Dermatology and Neurological Conditions

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ABSTRACT

Introduction: Botulinum toxin is a mainstay of treatment in cosmetic dermatology and neurology. Initially approved for ocular disorders, it is now widely used for cosmetic procedures and neurological conditions, such as chronic migraine and spasticity. This review explores the clinical approach to botulinum toxin in patients with overlapping aesthetic and therapeutic needs, highlighting current evidence, safety considerations, and interdisciplinary coordination.
Methods: A review of literature on botulinum toxin in cosmetic dermatology and neurology was performed. Original and review articles published before July 4th, 2025 were evaluated for relevance.
Discussion: Botulinum toxin inhibits acetylcholine release at the neuromuscular junction by cleaving SNAP-25, leading to reversible muscle paralysis. It reduces dynamic facial wrinkles by relaxing overactive muscles and has additional uses like treating hyperhidrosis and possibly enhancing dermal remodeling. In neurology, botulinum toxin inhibits pain neurotransmitters in chronic migraine and reduces spasticity in conditions like stroke or multiple sclerosis by blocking excessive motor neuron activity. It also relieves dystonia by targeting abnormal muscle contractions. Botulinum toxin is a safe treatment, with common side effects limited to mild local reactions; serious risks are rare but include unwanted diffusion and antibody formation. Careful dosing, anatomical precision, and patient selection minimize complications and improves outcomes.
Conclusion: A coordinated, interdisciplinary approach is essential for patients needing both therapeutic and cosmetic treatment. Synchronizing injection timing, dosing, and communication between providers ensures optimal outcomes and minimizes risk.

INTRODUCTION

Botulinum toxin, a neurotoxic protein produced by *Clostridium botulinum*, has revolutionized both therapeutic and cosmetic medicine through its unique mechanism of neuromuscular blockade.[1] Since its initial approval by the U.S. Food and Drug Administration (FDA) for the treatment of strabismus and blepharospasm in the 1980s, the clinical applications of botulinum toxin have dramatically expanded across various medical disciplines, particularly in cosmetic dermatology and neurology.[2] The toxin's ability to inhibit acetylcholine release at the neuromuscular junction results in reversible chemodenervation, making it an ideal agent

for conditions characterized by muscular overactivity or hypercontraction.[3,4]

In cosmetic dermatology, onabotulinumtoxinA is widely used to reduce dynamic facial wrinkles, including glabellar lines, crow's feet, and forehead rhytides.[5–9] These applications are among the most frequently performed non-surgical aesthetic procedures worldwide, largely due to their favorable safety profile, minimal downtime, and high patient satisfaction rates.[5,9] The toxin's cosmetic benefits arise from its ability to induce temporary muscle relaxation, thus preventing repetitive muscle contractions that contribute to wrinkle formation over time[1]. Despite its widespread popularity, the use of botulinum toxin in cosmetic medicine requires careful attention to dosage, injection technique, and anatomical considerations to optimize outcomes and minimize adverse effects such as asymmetry, ptosis, and unwanted diffusion to adjacent muscle groups.[3,9]

Beyond cosmetic applications, botulinum toxin is a critical therapeutic option for neurological conditions.[4] Chronic migraine poses a significant burden on patients and healthcare systems due to its debilitating nature and impact on quality of life.[2,4,10,11] The FDA approved onabotulinumtoxinA for chronic migraine prophylaxis in 2010, following pivotal clinical trials that demonstrated its efficacy in reducing headache frequency, severity, and associated disability.[10,11] The proposed mechanisms by which botulinum toxin alleviates migraine include inhibition of peripheral and central sensitization pathways, suppression of pro-inflammatory neuropeptide release (ie, calcitonin gene-related peptide, substance P, and glutamate), and modulation of nociceptive neurotransmission.[2,3,12] Botulinum toxin is commonly used in neurological disorders that trigger spasticity, such as stroke, multiple sclerosis, head trauma, spinal cord trauma, dystonia, and motor neuron diseases (spastic paraparesis, amyotrophic lateral sclerosis)[4]. Although botulinum toxin's utility is well established, concerns remain regarding the development of neutralizing antibodies with repeated administration, which may reduce therapeutic efficacy.[13] However, recent meta-analyses suggest that the incidence of clinically relevant antibody formation remains low, particularly with onabotulinumtoxinA, supporting its long-term safety and sustained benefit across indications.[13] Furthermore, ongoing research continues to elucidate new therapeutic indications and refine dosing strategies to maximize benefit.

This review aims to provide a comprehensive overview of the clinical approach to botulinum toxin in patients with overlapping cosmetic dermatology and neurological conditions. The paper will discuss current evidence and practice recommendations, and emerging research that continues to shape the evolving landscape of botulinum toxin therapy.

DISCUSSION

Botulinum Toxin Pathophysiology

Mechanism of Action

Botulinum toxin is a potent neurotoxin derived from *Clostridium botulinum*, with seven serotypes from A-G identified, of which type A (BoNT-A) is most commonly used clinically.[2,14] Serotype A exists in several formulations: onabotulinumtoxinA, abobotulinumtoxinA, and incobotulinumtoxinA, prabotulinumtoxinA, and daxxibotulinumtoxinA.[15]

The toxin exerts its effects by inhibiting acetylcholine release at the neuromuscular

junction.[2–4,12] After injection, botulinum toxin binds with high affinity to the presynaptic cholinergic nerve terminals via a two-step process: binding and internalization.[1] Once internalized through endocytosis, the light chain of the toxin cleaves SNAP-25, a synaptosomal-associated protein essential for vesicle fusion with the presynaptic membrane.[1,2] This blockade prevents the release of acetylcholine, leading to chemodenervation and reversible muscle paralysis.[1,2] Over time, nerve terminals sprout new synaptic contacts, restoring neurotransmission typically within 3 to 6 months.[4]

Pathophysiology in Cosmetic Dermatology

In cosmetic dermatology, botulinum toxin's primary target is dynamic facial wrinkles, which result from repetitive contraction of facial muscles over time.[6,14] The reduction of acetylcholine release leads to temporary paralysis of targeted muscles, such as the corrugator supercilii, frontalis, and orbicularis oculi14. This relaxation of hyperactive muscles smooths overlying skin, diminishes the appearance of dynamic rhytids, and prevents the formation of deeper static wrinkles with repeated treatments.[6,14] Additionally, BoNT-A modulates activity in non-muscle tissues, such as eccrine glands, making it useful for conditions such as axillary and palmoplantar hyperhidrosis14. By inhibiting sympathetic cholinergic stimulation of sweat glands, BoNT-A effectively reduces localized sweating.[2,14]

The aesthetic application of botulinum toxin also appears to influence fibroblast activity and collagen remodeling. Recent studies suggest that BoNT-A may indirectly contribute to dermal remodeling by reducing microtrauma from muscle contraction and potentially affecting fibroblast-mediated extracellular matrix production, leading to improved skin texture over time.[14]

Pathophysiology in Neurological Conditions

Within neurology, botulinum toxin's mechanism extends beyond the neuromuscular junction, affecting both motor and sensory pathways. In chronic migraine, the exact mechanism is multifactorial. BoNT-A is believed to inhibit the release of key pain-mediating neurotransmitters such as calcitonin gene-related peptide (CGRP), substance P, and glutamate from peripheral nociceptive fibers.[1,12] This action reduces peripheral sensitization, which in turn dampens central sensitization, one of the major contributors to chronic migraine pathology.[1,12]

In conditions such as spasticity from stroke, multiple sclerosis, traumatic brain injury, or spinal cord injury, BoNT-A reduces involuntary muscle hyperactivity by interrupting excessive alpha motor neuron activity at the neuromuscular junction[4]. The toxin provides functional improvements in mobility, posture, and pain control by relaxing spastic muscles without compromising overall strength.[4] In dystonia, BoNT-A reduces abnormal muscle contractions by decreasing aberrant cholinergic transmission at the dystonic muscle groups, providing symptomatic relief.[2,4]

While botulinum toxin's core mechanism revolves around inhibition of acetylcholine release, its therapeutic applications in both dermatology and neurology demonstrate its far-reaching physiological versatility.

Botulinum Toxin Indications

Cosmetic Dermatology

The most common cosmetic indication for botulinum toxin is the temporary reduction of dynamic wrinkles, lines formed by repetitive muscle contractions.[2,5,7,9,14] Anatomical locations for treatment include glabellar lines (frown lines), horizontal forehead lines, and lateral canthal lines (crow's feet)[5]. Injections into specific muscles, ie, corrugator supercilii, frontalis, orbicularis oculi, achieve localized chemodenervation and can softening expression lines and producing a more youthful appearance.[5]

Beyond wrinkle reduction, BoNT-A is increasingly used for facial contouring[16]. Masseter reduction for facial slimming is common in patients with bruxism.[16] Injections into the depressor anguli oris and mentalis muscles can elevate oral commissures and smooth the chin, respectively.[17] Advanced aesthetic uses include brow lifts, correction of "gummy smile," treatment of platysmal neck bands, and reduction of perioral rhytides and nasal "bunny lines".[18–21]

Non-facial cosmetic procedures are increasingly gaining popularity as well. These include treatment of horizontal neck lines (the "Nefertiti lift"), axillary and palmoplantar hyperhidrosis, and aesthetic reduction of lower leg bulk through gastrocnemius injection.[22–24] Cosmetic BoNT-A results are typically visible within 3–5 days, with peak effect at 1-2 weeks and duration of benefit lasting 3–6 months depending on formulation.[15] Repeated injections of BoNT-A may cause muscular atrophy in the injected regions, thereby extending the duration of effects.[15]

Neurological Conditions

In neurology, botulinum toxin serves as a therapeutic mainstay for a variety of movement disorders and neurological syndromes. It is FDA-approved for chronic migraine, cervical dystonia, blepharospasm, spasticity (including post-stroke and cerebral palsy-related), and adult upper limb spasticity[4,10,12,25–27].

For chronic migraine, BoNT-A is injected across multiple head and neck muscle groups according to the PREEMPT protocol[10,11,28]. Clinical trials demonstrate significant reduction in headache frequency and severity[10,11,28]. In stroke, spinal cord injury, multiple sclerosis, and traumatic brain injury, focal spasticity may impair function or cause pain. BoNT-A reduces tone in affected muscles, improving limb posture, hygiene, and in some cases, function[25,26]. Individualized dosing and EMG or ultrasound guidance enhance targeting.

Dystonias, including cervical dystonia, oromandibular dystonia, and hemifacial spasm, are among the most robust indications, with BoNT-A providing reliable symptom relief[25,27]. Notably, type B botulinum toxin is FDA-approved for cervical dystonia[29].

In amyotrophic lateral sclerosis or hereditary spastic paraparesis, focal injections can palliate excessive tone or drooling, improving quality of life even in progressive disease[30].

In the neurological setting, botulinum toxin is administered every 3–4 months, with careful monitoring of functional goals and potential adverse effects such as weakness or dysphagia. As a highly targeted therapy, it offers symptomatic relief without systemic side effects, making it especially valuable in medically complex populations.

Botulinum Toxin Safety

Botulinum toxin is a very safe medication with relatively few adverse effects. Since it is administered as a local injection, systemic side effects are limited.[2,15] The most common adverse effects are injection site reactions such as bruising, swelling, or discomfort, along with transient ptosis, headache, and

mild weakness in adjacent muscles.[2] These effects are usually self-limiting and resolve within a few weeks.[2]

The main contraindications to botulinum toxin injection include neuromuscular disorders such as amyotrophic lateral sclerosis, Lambert-Eaton syndrome, myasthenia gravis, and multiple sclerosiss.[2] However, botulinum toxin is routinely used in local injections for a variety of these conditions. Additionally, patients with known allergies to the toxin's constituents, active infections at injection sites, or psychological conditions such as body dysmorphic disorder should be approached with caution.[15] Pregnancy and breastfeeding are considered relative contraindications since botulinum toxin can potentially potentiate neuromuscular blockade in the developing fetus or neonate. Although data are limited, botulinum toxin is generally not recommended during pregnancy due to insufficient safety data in this population.[8] However, emerging research suggests good efficacy with limited side fetal effects for women treated with botulinum toxin for chronic migraines in pregnancy.[31,32]

Rare but serious adverse effects may occur if the toxin spreads beyond the target area. For example, in facial contouring, unwanted effects like asymmetric expression or dysphagia can occur, particularly when anatomical planes are poorly understood or injections are placed too deeply or laterally.[13] Therefore, precise knowledge of facial and regional anatomy is essential for minimizing complications, as adverse events are more likely when an injector lacks specialized training.[33]

Additionally, while higher botulinum toxin doses may extend the duration of therapeutic benefit, they also carry the risk of diffusion into neighboring muscles and resultant side effects. Therefore, a balance must be struck between optimizing efficacy and minimizing complications, especially in delicate anatomical regions like the neck and periorbital area.[8]

Another important consideration in safety is the development of neutralizing antibodies against botulinum toxin. Although relatively rare, repeated high-dose exposures may trigger an immune response that reduces the effectiveness of future treatments.[13] The incidence of clinically relevant antibody formation is less than 2% in most studies, particularly when standard dosing intervals and volumes are followed.[15]

Several botulinum toxin type A formulations are currently approved for clinical use, including onabotulinumtoxinA (Botox®), abobotulinumtoxinA (Dysport®), and incobotulinumtoxinA (Xeomin®), prabotulinumtoxinA (Jeuveau®), and daxxibotulinumtoxinA (Daxxify®). While each product shares the same 150 kDa neurotoxin core, they are manufactured using distinct processes and contain different excipients.[13] These differences result in varying potency units and dosing conversions, yet they all maintain similar safety profiles when used correctly.

Clinical Approach to Botulinum Toxin

Given what is known about the pathophysiologic mechanism of botulinum toxin, its uses in cosmetic dermatology as well as neurology, and its safety profile, a clinical approach to using botulinum toxin is crucial.

Patients with neurological conditions often seek botulinum toxin treatment not only for therapeutic relief but also for cosmetic enhancement. For example, individuals receiving botulinum toxin for chronic

migraine, cervical dystonia, or spasticity may concurrently express interest in aesthetic treatments for forehead lines, glabellar lines, or crow's feet. As both applications rely on similar injection techniques and target facial musculature, clinicians must be prepared to address overlapping needs.

Botulinum toxin typically exerts its clinical effects within 3 to 5 days, reaching peak efficacy by two weeks, and lasts approximately 3 to 4 months in most patients, extending to 6 months with select formulations.[2,15] However, duration can vary based on individual metabolism, muscle mass, and treatment area. For neurological conditions such as spasticity or dystonia, slightly higher doses are used, which may modestly extend the duration of effect. Re-treatment is commonly scheduled at 12-week intervals.[2]

Given this timeframe, a coordinated approach between dermatologists and neurologists becomes essential. To optimize efficacy and patient satisfaction, treatments should be timed to coincide, either during the same visit or within a short window, to avoid desynchronization of therapeutic and cosmetic outcomes. This collaborative model ensures that dosing strategies account for both functional and aesthetic goals, minimizes cumulative toxin load, and reduces the risk of antibody formation due to overly frequent injections.

Clear communication between specialties, including shared treatment plans and anatomical mapping, can streamline care and prevent redundant or conflicting interventions. In patients with dual indications, personalized scheduling not only enhances outcomes but also reinforces a patient-centered, interdisciplinary approach to botulinum toxin therapy.

CONCLUSION

Use of botulinum toxin in cosmetic dermatology and neurology is very common. Understanding botulinum toxin's pathophysiology and its safety profile lends a critical application to clinical approach. Coordinating dermatology and neurology treatment with botulinum toxin is central to effective and patient-centered management.

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