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BOTULINUM TOXIN TYPE A: MULTIFACETED THERAPY FOR NEUROLOGICAL, AUTONOMIC AND OCULAR DISORDERS

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ABSTRACT

Introduction: Botulinum toxin, a neurotoxin produced by Clostridium botulinum, is an effective form of therapy for treating conditions associated with excessive muscle activity or disorders of the autonomic nervous system. Its mechanism of action is based on the inhibition of acetylcholine release at presynaptic nerve terminals, leading to a reversible blockade of neuromuscular transmission and a subsequent reduction in muscle tone. This enables effective control of conditions such as dystonia, spasticity, chronic migraine, overactive bladder syndrome, strabismus, blepharospasm and nystagmus.
Objective: The aim of this study is to present the current state of knowledge on the use of botulinum toxin in the treatment of dystonia, spasticity, chronic migraine, overactive bladder (OAB), strabismus, blepharospasm and nystagmus taking into account clinical efficacy and safety.
Material and methods: A review of the literature available in PubMed, Scopus, and Web of Science databases was conducted, including randomized clinical trials, meta-analyses, and scientific society guidelines.
Results: Botulinum toxin type A (BoNT-A) is a recognized and safe therapeutic intervention for the treatment of neurological and autonomic disorders such as dystonia, spasticity, chronic migraine, and neurogenic overactive bladder. In patients with dystonia and spasticity, BoNT-A reduces muscle tone, limits involuntary contractions, and alleviates pain, supporting rehabilitation and preventing contractures. In chronic migraine, the therapy reduces both the frequency and severity of pain attacks, while in neurogenic overactive bladder, it alleviates urinary urgency and incontinence. In the treatment of strabismus, it enables correction of muscle imbalance and improvement of eye alignment. In blepharospasm, it reduces the frequency and strength of eyelid spasms. In nystagmus, it reduces the amplitude of eye tremors and improves fixation stability.
Numerous studies indicate that BoNT-A is also effective in cases resistant to standard treatment, with the safety and effectiveness of the therapy depending on individual dose adjustment and systematic monitoring of clinical effects.
Conclusions: Botulinum toxin type A (BoNT-A) is an effective and safe therapy for the treatment of neurological and autonomic disorders such as spasticity, dystonia, chronic migraine, neurogenic overactive bladder, strabismus, blepharospasm and nystagmus. The use of BoNT-A leads to symptom relief, pain reduction, and improved quality of life for patients, especially in cases resistant to conventional treatment, while maintaining a favorable safety profile through individualized adjustment and monitoring of clinical effects.

KEYWORDS

Botulinum Toxin (BoNT-A), Dystonia, Spasticity, Chronic Migraine, Neurogenic Overactive Bladder, Strabismus, Blepharospasm, Nystagmus

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Introduction

Botulinum toxin, widely known for its use in aesthetic medicine, has for many years also been extensively employed as a therapeutic agent in the management of neurological, urological, ophthalmological disorders and chronic pain syndromes. Initially used mainly in the treatment of strabismus and dystonia, it has become the subject of numerous clinical studies that have confirmed its efficacy and safety in the treatment of conditions such as chronic migraine, muscle spasticity, overactive bladder, blepharospasm and nystagmus.

Among the clinically available botulinum toxin preparations, onabotulinumtoxinA is the most extensively studied in terms of safety and efficacy. Currently, botulinum toxin type A (BoNT-A) preparations have been approved by regulatory agencies such as the FDA and EMA for the treatment of many clinical indications beyond aesthetic applications.

This publication presents an overview of the current clinical indications for botulinum toxin outside of aesthetic medicine, with particular emphasis on its applications in neurology, urology, ophthalmology and pain management. The mechanisms of action, safety of therapy and the most important clinical studies underlying the recommendations are discussed.

Botulinum toxin

The use of botulinum toxin, which began in the 1970s, has expanded significantly in recent years. It now covers a wide range of clinical indications, both in aesthetic and therapeutic medicine. [1]

The mechanism of action of all known serotypes of botulinum toxin is based on selective disruption of communication between neurons and muscle cells. This toxin prevents the release of acetylcholine, the primary neurotransmitter in neuromuscular synapses, which leads to the interruption of nerve impulses and, as a result, causes temporary immobilisation of the muscle groups involved [2].

Of all known biological substances, botulinum toxin is characterised by the highest toxicity, even in extremely small doses. [3]

Also known as the ‘miracle poison’, [2] it now plays an important role in the treatment of many different diseases. Although mainly associated with aesthetic medicine, it has also found wide application in the treatment of ophthalmic neurological, muscular and pain disorders. [4]

Seven different serotypes of botulinum toxin have been described in the medical literature, each with unique antigenic characteristics. Among these, serotypes A and B are most commonly used in clinical practice. Serotype A, designated BTX-A or Bo-NT-A, is considered the most effective in terms of potency, making it the preferred choice in many therapies.[1]

Botulinum toxin therapy in neurological muscle tone disorders

Botulinum toxin type A (BTX-A) has long been recognised as one of the key therapeutic tools in the treatment of neurological disorders characterised by increased muscle tone. Its applications include the treatment of dystonia, both focal and generalised, and spasticity associated with damage to the central nervous system. The effectiveness of this method has been confirmed in a number of clinical trials and observations conducted in many centres, making BTX-A an important element of modern therapeutic strategies in neurology [5,6].

Botulinum toxin in the treatment of dystonia

BTX-A is considered the standard treatment for focal dystonia, including cervical dystonia, blepharospasm and hemifacial spasm. When botulinum toxin is administered to muscles affected by dystonia, it causes localised, well-controlled and predictable peripheral paresis, which lasts for approximately three months. [7] Studies have shown a significant improvement in symptom severity and quality of life in patients after the use of BTX-A. Its effectiveness in reducing clinical symptoms and improving quality of life has been confirmed in numerous clinical studies, both older and current.

A study conducted by Simpson et al. evaluated the effectiveness of botulinum toxin type A (BTX- A) therapy in 205 patients with cervical dystonia in whom previous treatments had proven ineffective. A total of 1,074 BTX-A injections were administered during 505 treatment sessions. Analysis of the results showed that as many as 71% of participants experienced significant clinical improvement in dystonia symptoms. In a group of 89 patients reporting pain, 76% experienced almost complete pain relief. The duration of maximum improvement was 11.2 weeks on average, indicating a lasting therapeutic effect. Adverse effects were reported in 28% of the subjects, with the most commonly reported complications being mild dysphagia and weakness of the neck muscles, which were transient and did not require discontinuation of treatment. [8]

The effectiveness of BTX-A in cervical dystonia is also confirmed by the results of a Cochrane review from 2020. The analysis included randomised studies involving adults and showed that BTX-A significantly reduces the severity of symptoms compared to placebo. However, the authors noted the limited amount of data on comparisons between individual preparations and long-term effects [9].

In another study involving 219 patients, the long-term efficacy of incobotulinumtoxinA (Xeomin) was evaluated. Patients received 120 or 240 units of BTX-A at flexible intervals (minimum 6 weeks), with up to 5 treatment cycles over a period of 68 weeks. A sustained improvement in the TWSTRS scale was achieved, with a low incidence of adverse events, mainly transient dysphagia and muscle weakness [10].

Botulinum toxin in the treatment of spasticity

Spasticity is a significant clinical problem in the course of many neurological disorders, such as stroke, multiple sclerosis and cerebral palsy.It manifests itself as a pathological increase in muscle tone, which can occur in the form of continuous tonic contractions, irregular phasic contractions, or a combination of both. Lack of adequate control over this condition leads to adaptive structural changes, such as permanent shortening of muscles and fascia, resulting in the development of contractures that limit range of motion. [11]

Conventional methods of treating spasticity primarily include physiotherapy interventions, oral pharmacotherapy and, in selected cases, surgical procedures. In recent years, significant progress in therapy has been made with the use of local injections of botulinum toxin type A (BoNT-A), which are highly effective in reducing muscle tone.

By inhibiting the release of acetylcholine at the neuromuscular junction and causing temporary muscle paralysis, botulinum toxin type A (BTX-A) aims to reduce spasticity and, consequently, improve joint function. [12]

A multicentre, randomised, placebo-controlled phase III trial was conducted to evaluate the efficacy and safety of incobotulinum toxin A in the treatment of lower limb spasticity in Japanese patients. The phase III trial included 208 Japanese patients with lower limb spasticity who received incobotulinumtoxin A (400 units) or placebo. After a single injection, a significant improvement in spasticity was observed in the treatment group (mean change in Ashworth scale −7.74 vs. −4.76 in placebo; $p = 0.0041$). The effects persisted for 12 weeks, and further improvement was seen during open-label extension therapy. The safety profile was favourable, with rare and mild adverse events. The results confirm the efficacy and good tolerability of the therapy [13].

Another multicentre, prospective observational study evaluated the long-term efficacy and safety profile of botulinum toxin type A (BTX-A) in patients with spasticity resulting from various neurological conditions: traumatic brain injury (TBI), spinal cord injury (SCI) and multiple sclerosis (MS). The study included 120 patients who received standard doses of BTX-A for 12 months. The efficacy of the therapy was measured using the Modified Ashworth Scale (MAS) and the Modified Rankin Scale, while safety was assessed based on the frequency and type of adverse events. The most pronounced reduction in spasticity and improvement in motor function was observed in patients with MS, where the mean reduction in MAS was −1.8 ($p < 0.01$). Moderate improvement was observed in the TBI and SCI groups, with mean MAS reductions of −1.2 and −1.3, respectively ($p < 0.05$). Adverse events reported were sporadic and mild in nature, mainly involving local reactions at the injection site. The results indicate that long-term use of BTX-A is an effective and well-tolerated treatment for spasticity in patients with various neurological conditions. [14]

Another randomised, controlled clinical trial evaluated the effect of a stretching exercise programme on the efficacy and durability of botulinum toxin type A (BTX-A) in patients with spasticity following a stroke. The study involved 60 participants who, after receiving BTX-A, were divided into two groups: one undergoing a regular stretching exercise programme and a control group without additional physical therapy. After 24 weeks, a significantly greater reduction in muscle tone was observed in the exercise group, as assessed using the modified Ashworth scale (mean change −2.1 vs. −1.3 in the control group, $p < 0.01$). In addition, patients following the stretching programme showed improved motor function and greater satisfaction with the therapy. No significant differences in the incidence of adverse events were observed between the groups. The results suggest that incorporating stretching exercises into standard botulinum toxin treatment may significantly increase the effectiveness of spasticity therapy after stroke. [15]

Botulinum toxin in the treatment of migraine

Migraine is one of the most common neurological disorders, with a complex, multifaceted nature. [16] It is characterised by periodic, intense headaches, [16] which result from the interaction of many biological, environmental and genetic factors.

In typical cases, migraine is episodic and lasts from several hours to up to three days. These attacks are often accompanied by symptoms such as nausea, vomiting, and hypersensitivity to light and sound stimuli. Some patients experience a so-called aura – temporary visual or sensory disturbances or other neurological symptoms that precede the actual headache. [17]

Migraine is a complex neurological disorder in which the trigeminovascular system plays a key role in its pathophysiology [16]. Crucial to its pathophysiology is the activation of the trigeminovascular system – comprising the sensory fibres of the trigeminal nerve (ganglion trigeminale) innervating the dura mater and large cerebral vessels – which, in response to triggering stimuli, release vasodilatory neuropeptides, including calcitonin gene-related peptide (CGRP) and substance P. In some cases, there is also a phenomenon of spreading cortical depression (CSD), which is a wave of depolarisation of neurons in the cerebral cortex that can initiate a pain attack. These processes result in the sensitisation of pain neurons in the central nervous system, which leads to the onset of characteristic throbbing headaches and accompanying symptoms such as photophobia and hypersensitivity to sound. [18]

BoNT/A inhibits neurotransmitter exocytosis by cleaving SNARE proteins, which blocks the release of neuropeptides such as CGRP, substance P and glutamate. This action reduces the activation of pain pathways and the transmission of pain signals within the trigeminal vascular system. [19] The toxin affects the surface expression of certain ion channels and receptors, which may contribute to a reduction in the sensitivity of neurons to pain stimuli and a decrease in central sensitisation [19]. After peripheral administration, BoNT/A is captured by nerve endings and then transported retrogradely to ganglia and centrally to trigeminal nuclei. There, the toxin can cleave SNARE proteins, blocking neurotransmitter release and reducing the activation of pain neurons [20]. Botox is used in patients with chronic migraine, defined as headaches occurring on ≥15 days per month, of which ≥8 days meet the criteria for migraine. [21] Treatment consists of cyclical (every 12 weeks) administration of the preparation to specific points on the head and neck, in accordance with an established protocol (e.g. PREEMPT).

BoNT-A therapy is effective in reducing the number of days with headaches, the severity of symptoms and improving quality of life. It is well tolerated and is an important therapeutic option, especially in patients who do not respond to classic preventive treatment.

The most compelling data on the efficacy of botulinum toxin type A (BoNT-A) in the treatment of chronic migraine come from the PREEMPT programme, comprising two large, randomised, placebo-controlled trials (PREEMPT-1 and PREEMPT-2) involving a total of 1,384 patients.

The drug was administered every 12 weeks at a dose of 155–195 units, injected into 31–39 points on the head and neck. After 24 weeks of therapy, the number of days with headache decreased by an average of 8.4 days per month in the BoNT-A group, compared to 6.6 days in the placebo group ($p < 0.001$). An improvement was also observed in the severity of symptoms and their impact on daily functioning (HIT-6).

After one year of treatment, nearly 70% of patients achieved at least a 50% reduction in the number of pain days. The therapy was well tolerated – the most common adverse events were mild and transient and included neck pain, muscle weakness and eyelid drooping. [22]

Botulinum toxin in the treatment of overactive bladder syndrome (OAB)

Overactive bladder syndrome (OAB) is a common condition characterised by uncontrolled bladder contractions leading to sudden urge to urinate, frequent urination and nocturnal urination (nocturia), despite the absence of any diagnosed urinary tract disease. Large studies indicate that this condition affects more than 10% of the population. [23]

The first line of treatment is usually lifestyle modifications, bladder training, and anticholinergic drugs or beta-3 agonists. However, many patients do not achieve satisfactory improvement or experience significant side effects, creating a need for alternative therapeutic solutions. [24].

In recent years, there has been growing interest in the use of intradetrusor injections of botulinum toxin type A (BoNT-A) in the treatment of overactive bladder (OAB), both idiopathic and neurogenic (neurogenic detrusor overactivity – NDO). The mechanism of action of BoNT-A in the bladder involves inhibition of acetylcholine release at the nerve endings innervating the detrusor muscle, modulation of TRPV1 and P2X3 receptors in the bladder mucosa, and reduction of afferent stimulation of the bladder. The effect of these actions

is a reduction in the frequency of uncontrolled detrusor muscle contractions and a significant improvement in clinical symptoms such as sudden urge and urinary incontinence. [25][26][27][28]

In patients in whom standard treatment (including anticholinergic drugs, beta-3 agonists, bladder training) does not produce satisfactory results or is poorly tolerated, botulinum toxin injections into the bladder detrusor muscle (intradetrusor) are considered. [29] Some experimental studies have also evaluated submucosal administration, showing similar clinical efficacy, but this method is not currently the standard of care.

In a large phase III study conducted by Dmochowski et al. (2010) in 557 patients, the use of 100 U of onabotulinumtoxinA resulted in a significant reduction in urge episodes and urinary incontinence compared to placebo, as well as improved results in quality of life questionnaires (ICIQ-OABqol). [30]

In a prospective study of women with idiopathic OAB, 100 J of intradetrusor botulinum toxin was administered and the clinical and urodynamic effects were evaluated: after the first injection, patients reported significant improvement for up to 9 months; when the injection was repeated, the effects were comparable. Mild complications included an increase in residual urine volume, dysuria and urinary tract infections. [31]

Another study involving women with OAB assessed the effect of the procedure on nocturnal symptoms (nocturia and nocturnal incontinence episodes). After administering 100 units of botulinum toxin type A to the bladder in 76 women with overactive bladder, the number of nocturia

episodes decreased by an average of 0.98 and nocturnal incontinence by 0.37 ($p < 0.001$). The average volume of urine passed at night increased by 92.6 ml, and the subjective assessment of treatment efficacy on the VAS scale improved by an average of 58 points. Three patients (3.9%) required catheterisation due to urinary retention. [32]

Botulinum toxin type A is an effective treatment for neurogenic overactive bladder, but its use requires careful monitoring of the patient in terms of treatment efficacy and potential adverse effects, such as urinary retention or urinary tract infections. [30][31][32] The decision to use this therapy should be made on an individual basis, taking into account the characteristics of the disease, the patient's needs and current scientific research confirming the safety and efficacy of BoNT-A.

Botulinum toxin therapy in ophthalmology

Botulinum toxin plays a significant role in treating various ophthalmological disorders, such as strabismus, nystagmus, and blepharospasm.

Botulinum toxin has a therapeutic effect in the treatment of strabismus and nystagmus through selective, reversible blockade of acetylcholine release at the neuromuscular junction, resulting in temporary weakening of extraocular muscle tension and enabling correction of eye alignment disorders.

The long-lasting effect of eye alignment correction results from two specific mechanisms:

(1) disruption of the balance between antagonistic and synergistic extraocular muscles and

(2) reestablishment of central regulation of eye alignment by the binocular vision system.

In the case of strabismus, temporary paralysis of the extraocular muscle leads to a change in the position of the eyeball. In nystagmus, short-term immobilisation of all extraocular muscles results in a reduction in the intensity of abnormal eye movements. In blepharospasm, on the other hand, temporary inhibition of the activity of the extraocular extensor muscles suppresses involuntary contractions. [33]

Strabismus

Strabismus, or misalignment of the eyes leading to binocular vision disorders, can be treated both surgically and with botulinum toxin type A. Clinical studies indicate that injections of this toxin can effectively reduce the angle of deviation with a low risk of complications, making this method an attractive alternative, especially in cases with a smaller angle of deviation or a high risk of surgical complications.

In a study involving 74 patients with intermittent exotropia, BTA was administered to both lateral rectus muscles. The mean angle of deviation decreased significantly (at distance and near). Success (defined as deviation ≤ 10 PD) was achieved in approximately 56.7% of patients. [34]

A retrospective study conducted at an ophthalmology centre in Riyadh (Saudi Arabia) included patients with exotropia treated with botulinum toxin type A (Botox) injections between 2014 and March 2020. The main objective was to determine the extent to which a deviation angle of less than 10 prism dioptres could be achieved. A total of 97 cases (57 males and 40 females) aged between 2 months and 40 years were examined. In this group, 28 patients (approximately 29%) achieved an angle of deviation ≤ 10 prism after treatment, while 49 patients (approximately 50%) achieved ≤ 20 prism. Women responded better than men: 32.5% of women achieved ≤ 10 prisms and 60% achieved ≤ 20 prisms. In addition, it was shown that a higher dose of toxin correlated with a greater change in the

deviation angle. Conclusions: Botulinum toxin therapy may be a safe alternative to surgery in selected types of exotropia — the best results were achieved in 'ordinary 'forms of exotropia, while the presence of significant visual impairment or amblyopia was associated with poorer results. [35]

Another study evaluated the efficacy of botulinum toxin type A injections in children with neurological disorders and strabismus. Fifty children with esotropia (34) or exotropia (16), mainly with cerebral palsy or hydrocephalus, participated in the study. The mean angle of deviation decreased from 42.5 PD before treatment to 12.8 PD after injection, and 60% of children achieved alignment within 10 PD. The success of the therapy was more frequent in esotropia, shorter duration of strabismus and smaller angle of deviation. The authors indicate that botulinum toxin is an effective and safe alternative to surgery, especially in cases of high surgical risk. [36]

Blepharmospasm

Blepharospasm, also known as eyelid spasm, is a neurological disorder characterised by involuntary, recurrent contractions of the extraocular muscles responsible for closing the eyelids. Symptoms include frequent blinking, narrowing of the eyelids that impairs vision, and in extreme cases, complete closure of the eyes, which significantly affects the patient's quality of life. The aetiology of the condition is not fully understood, but it is known to be multifactorial, involving both genetic and environmental factors. The treatment of blepharospasm includes pharmacological and rehabilitation methods, as well as botulinum toxin type A injections, which have been shown in numerous clinical studies to be effective in reducing the severity of spasms and improving patients' quality of life. [37] [38]

In the case of blepharospasm, numerous studies have confirmed that treatment with botulinum toxin brings about a significant improvement in symptoms – spasms are milder, more invasive interventions are less frequently required, and the therapy is well tolerated.

In a retrospective study conducted at the Ophthalmology Clinic at the University of Naples assessed the long-term efficacy and safety of botulinum toxin type A (Botox) in the treatment of blepharospasm. Between 1980 and 2001, 178 patients with blepharospasm were treated at the Ophthalmology Clinic of the University of Naples. Each patient had the severity of their spasms assessed on a four-point scale and the duration of improvement (in months) monitored. Among the 168 patients observed, 93% reported improvement after toxin administration. The average duration of the effect was 3.6 months. In patients who received more than 14 treatments, as many as 76% achieved stable relief of their symptoms. Complete remission of symptoms was achieved in 1.7% of participants (3 people). No systemic or toxic reactions were reported — side effects were localised. The authors conclude that botulinum toxin type A therapy is an effective, safe, simple and repeatable method of reducing eyelid spasms in most patients with blepharospasm. [39]

Nystagmus

Nystagmus is an involuntary, usually rhythmic movement of the eyeballs — lateral, vertical or rotational — caused by disturbances in the eye movement amplification system and/or the vestibular system. [40]

Although the treatment of nystagmus with botulinum toxin is not currently part of standard guidelines, there are increasing reports in the literature indicating its potential effectiveness. Of particular importance may be cases in which nystagmus coexists with changes in the axis of the eyeballs or asymmetry of eye movements, suggesting the possibility of using this therapy as a supplement to conventional treatment methods in selected clinical situations.

Six patients aged 17 to 35, equally divided between women and men, who had exotropia with concomitant nystagmus, were enrolled in the study. Each patient was administered botulinum toxin type A (Dysport, Ipsen) into the lateral rectus muscles of both eyes using a 27G retrobulbar needle. Before the procedure and during subsequent check-ups, comprehensive ophthalmological and orthoptic examinations were performed, which allowed for a thorough assessment of the changes after therapy. After toxin administration, all patients showed marked improvement: nystagmus in the central position of vision was significantly alleviated, and the previous compensatory head posture improved to such an extent that most participants felt significant relief in their daily functioning. The procedure was performed without serious complications, and any side effects were mild and quickly resolved. These results suggest that local administration of botulinum toxin may be an effective and well-tolerated method of alleviating symptoms in patients with exotropia and nystagmus. [41]

Summary

Botulinum toxin type A (BoNT-A) is an effective and safe therapeutic option for the treatment of neurological, motor and autonomic disorders such as dystonia, spasticity, chronic migraine, overactive bladder syndrome, strabismus, blepharospasm and nystagmus. The therapy contributes to the reduction of muscle tension, limitation of involuntary contractions, pain relief and improvement of patients' quality of life, even in cases resistant to conventional treatment. In ocular disorders, BoNT-A specifically improves eye alignment, reduces involuntary eyelid spasms, and stabilizes abnormal eye movements, thereby enhancing visual function and comfort.The efficacy and safety of BoNT-A depend on individual dose adjustment and systematic monitoring of clinical effects.

Author's contribution

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