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# Phenomenon of "Sismonasty" in the Use of Botulinum Toxin for Treatment of Hyperkinetic Facial Wrinkles: A Case Report

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## 1. Introduction

The adverse effects of medications are of paramount scientific importance for their clinical approval, often outweighing their efficacy and safety. Drugs that exhibit high safety profiles with wide therapeutic windows and effective outcomes can still prompt re-evaluation if they produce recurring or severe adverse effects when used on a larger population.

Botulinum toxin type A, produced by the bacterium *Clostridium botulinum*, has a significant history of research. Initially identified in 1817 by German physician Justinus Andreas Christian Kerner while investigating food poisoning cases, the term "botulism" derives from the Latin word "botulus," meaning "sausage," due to its association with sausage-related poisoning[1].

Research on botulinum toxin gained momentum during World War II, driven by its high toxicity and potential for use as a biological weapon. Despite the nefarious intentions of this period, advancements in understanding the toxin's mechanisms and dose response relationships occurred[2]. The toxin's first medical use was approved in 1979 for treating strabismus, followed by FDA approval in 2002 for treating glabellar hyperkinetic wrinkles, marking its entry into aesthetic applications[3].

The literature indicates that botulinum toxin type A has a broad therapeutic range. A dose exceeding 3,000 units is required to achieve lethality in a 75 kg individual via intramuscular injection. Lethality concentrations vary by absorption route: 1 µg/kg for oral ingestion, 10-13 ng/kg for inhalation, and 1-2 ng/kg for intravenous or intramuscular routes[4].

This case report aims to describe an adverse effect of involuntary contraction of the frontal muscles induced by mechanical stimulus (nastic) following botulinum toxin type A treatment for hyperkinetic facial wrinkles. It also introduces the concept of similarity with the botanical phenomenon of sismonasty to elucidate the possible mechanism responsible for this phenomenon. Although the phenomenon was self-limiting and did not harm the patient, it serves as a cautionary note for practitioners using botulinum toxin type A. Despite extensive use and numerous studies, gaps remain in understanding its mechanisms and potential adverse effects. The use of botulinum toxin type A should not be trivialized, as it has the potential for serious consequences.

## 2. Case Report

In accordance with CONEP Resolution No. 166/2018, this report has been approved by the institution's Research Ethics Committee and adheres to CNS Resolution 196/96 guidelines.

The patient was a 32-year-old healthy white male with no comorbidities, no prior botulinum toxin treatments, and no history of neuromuscular disorders. Clinical examination revealed static wrinkles in the forehead and glabellar region (Glogau 3) and dynamic wrinkles in the periocular region (Glogau 2). The frontal and glabellar muscles were well-developed. A treatment plan was devised using botulinum toxin type A without proteins (Xeomin®), with a total dose of 100 IU, individualized for each region (Figure 1).

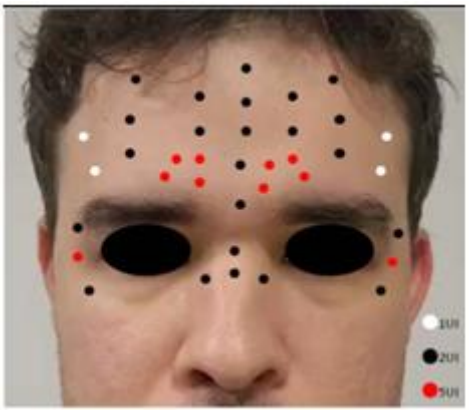

**Figure 1.** *Demonstration of the therapeutic plan carried out on the patient based on the muscle mass of each region.*

The treatment was administered on May 9, 2023, without complications. After 48 days, on June 26, 2023, the patient observed involuntary contraction of the frontal muscles with eyebrow tail elevation following vigorous massage of the right and left frontal regions (Figure 2). This contraction had a latency of 3 to 5 seconds and lasted 10 to 15 seconds (Figure 3), causing slight asymmetry and formation of hyperkinetic wrinkles (Figure 4). When attempting voluntary contraction, the affected regions appeared paralyzed by the toxin (Figure 5).

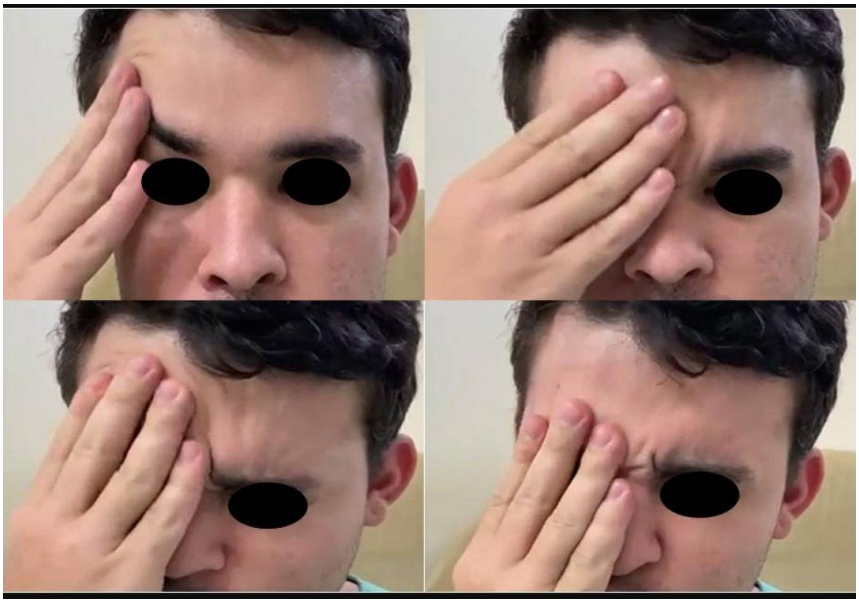

**Figure 2.** *Demonstration of Mechanical stimulus through vigorous massage in the right frontal region, with the same triggering mode of contraction on the left side*

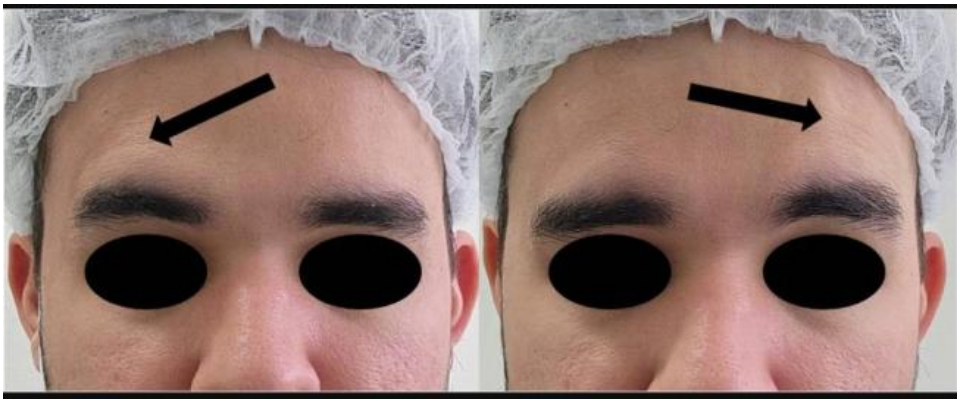

**Figure 3.** *Demonstration of the involuntary muscle contraction that occurs after mechanical stimulus, demonstrating the formation of hyperkinetic wrinkles in the tail portion of the eyebrow*

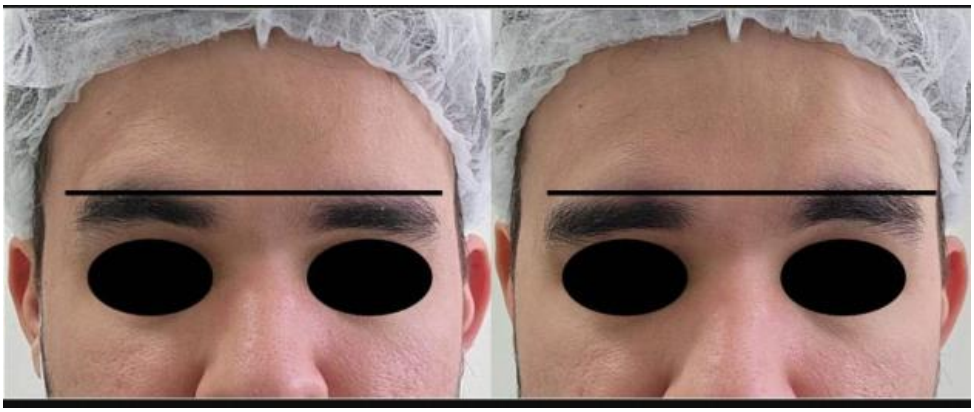

**Figure 4.** *Demonstration of the transient asymmetry caused by the involuntary contraction of the frontal muscles*

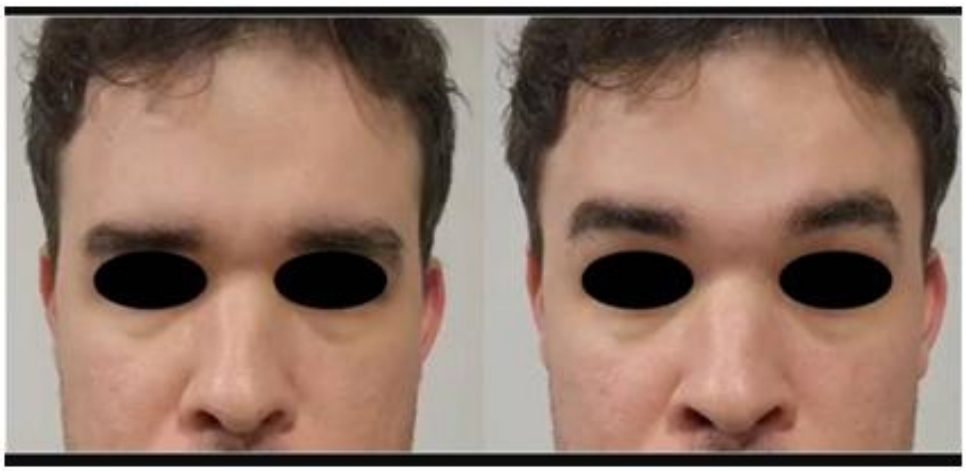

**Figure 5.** *Demonstration of the action of botulinum toxin with the patient's voluntary contraction, noting that there is no contraction of the muscles that contract involuntarily nor the formation of hyperkinetic wrinkles in the body and eyebrow tail regions as occurs after mechanical stimulus.*

As the condition was benign, self-limiting, and induced only by local mechanical stimulation, it was decided to monitor the phenomenon. By July 24, 2023, 76 days post-procedure and 28 days after the manifestation, the phenomenon had resolved, with the intended paralysis of the frontal muscles persisting.

## 3. Discussion

Seven types of botulinum toxins are currently known, with type A being the most toxic. Botulinum toxins are among the most potent substances, with a lethal dose of approximately 1 ng/kg, far exceeding the toxicity of cyanide[5].

The mechanisms of botulinum toxins are not fully understood but are primarily characterized by the inhibition of acetylcholine release at the neuromuscular junction. The action of the toxin can be categorized into four main effects: 1. Muscle relaxation, 2. Antinociceptive action, 3. Action on the autonomic nervous system, and 4. Direct and indirect effects on the central nervous system[6].

This report focuses on muscle relaxation. The observed phenomenon resembles sismonasty in botany, where plants exhibit contraction or closure of their leaves in response to mechanical stimuli. In plants, sismonasty results from mechanical stimulation causing the release of calcium and potassium into the extracellular medium, leading to osmotic loss of intracellular fluid and cell volume reduction, thereby causing leaf closure[7].

Botulinum toxin type A acts by blocking acetylcholine release at the neuromuscular junction, which involves several stages: diffusion to the cholinergic nerve terminal, binding to high-affinity receptors, endocytosis, and subsequent inhibition of acetylcholine exocytosis[6]. The toxin cleaves SNARE proteins, specifically SNAP-25, inhibiting the fusion of acetylcholine vesicles with the nerve terminal membrane, thus preventing muscle contraction[6].

In the context of the reported phenomenon, the mechanical stimulus may stretch muscle spindles, triggering afferent signals via fibers Ia

and II. This stretch activates alpha-motoneurons and gamma-motoneurons, which stimulate intrafusal fibers. The botulinum toxin's partial reduction of afferent signals may still allow for some activation of gamma-motoneurons, leading to the observed involuntary contraction. The phenomenon likely resolves as the toxin's effects on afferent signals and intrafusal fibers diminish over time, with a partial return of muscle activity after approximately three months[6] (Figure 6).

## 4. CONCLUSION

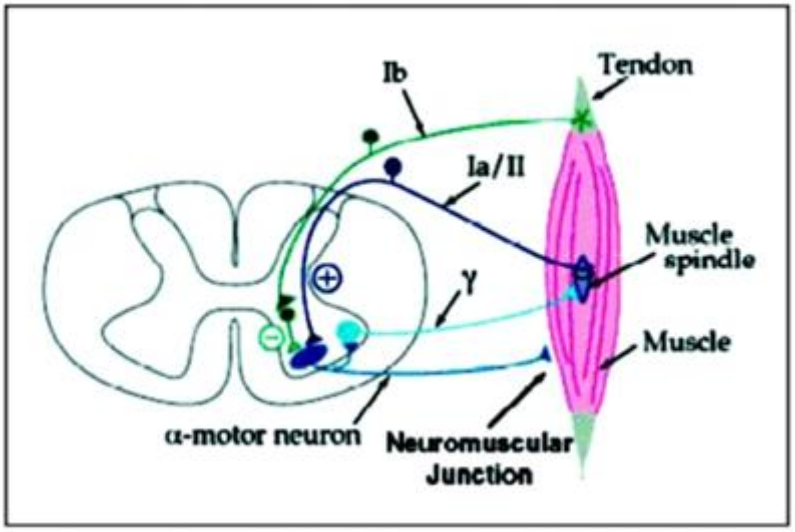


**Figure 6**. *Demonstration of the spinal cord stretch reflex. Stretching of the muscle spindle, triggering conduction of signals through fibers la and Il to the alpha-motoneuron. Stimulation of intrafusal fibers through the collateral pathway that stimulates the gamma-motoneuron. This path would be the explanation of the observed seismonasty phenomenon. (Source: Dressler D, Saberi FA, Barbosa ER. Botulinum toxin: mechanisms of action. Arg Neuropsiquiatr, 2005:63(1):180 185).*

Despite its extensive use, gaps remain in understanding the mechanisms of botulinum toxin type A. This report highlights a novel adverse effect that was benign and resolved without intervention. Further research is necessary to fully comprehend the range of effects and mechanisms associated with this treatment.

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