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Botulism Presented with Clinical Head Drop, Approach to Botulinum Toxin A: A Case Report

Klinik Olarak Kafa Düşmesi ile Prezente Olan Botulism Olgusu, Botulinium Toxin A'ya Yaklaşım, Olgu Raporu

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Anahtar Kelimeler: Botulinium toksini, BoNT, advers olay, elektrodiagnoz, baş düşmesi

Dear editor,

Botulinum toxin (BoNT) is a proteinaceous exotoxin. Of all biotoxins, BoNT has the greatest neurotoxic potential. In fact, BoNT is a strong, fatal, and irreversible neurotoxin, and if administered incorrectly, it can cause botulism. However, BoNT-A is stable, since it is the most powerful neurotoxin that is frequently used in medicine and is simple to prepare, having the longest function duration at low temperatures (1).

The effects of BoNT on cholinergic motor nerve terminals, flaccid paralysis of the muscles, and transient denervation have been observed. This exotoxin has the potential to kill an organism's neurological system, which might result in symptoms such as lightheadedness, breathing difficulties, and muscular weakness. It is used therapeutically to treat abnormalities of muscular movement, including facial spasms. The use of BoNT has been adopted to treat conditions like hyperhidrosis, strabismus, facial tics, refractory headaches, and persistent migraines (2).

The cosmetic injection of BoNT has had a powerful impact throughout the world since the interest in looking young has become widespread (3).

Typically, a clinical history and laboratory confirmation are used to diagnose botulism. Botulism is occasionally misdiagnosed as a stroke, Guillain-Barré syndrome, or myasthenia gravis.

High dosages of BoNT can result in botulism when used to treat conditions such as hereditary spastic paraparesis, extremities dystonia, hemiparesis, paralysis, hyperhidrosis, and neurogenic bladder. In the vast majority of reported instances, diagnosis is made using clinical criteria. However, electrodiagnostic confirmation is not included in many publications (1).

Our patient was a 50-year-old woman. After receiving 100 units of Dysport (abobotulinumtoxinA) for her masseters on March 30, 2022, the patient complained of weakness and difficulties in swallowing and was later sent to the critical care unit on April 5, 2022. Here, BoNT was administered to the patient's masseters for cosmetic reasons. It was the first time BoNT was used on her masseter muscles.

The patient was fatigued from March 30, 2022 until she was taken to the hospital's critical care unit on the fifth day of sickness, due to widespread weakness and the inability to support her neck backwards (Figures 1, 2). After receiving BoNT, the patient experienced diarrhea for the first 2 days, followed by constipation that has persisted ever since. From the first day, the patient suffered from severe dry mouth. About 4–5 days after receiving Botox, she experienced trouble swallowing and had trouble consuming small meals. In time, speech problems began to emerge. The patient had never suffered from urinary incontinence. Localized pain occurred in the jaw and neck. Around the chin and face, there was extensive redness. The patient made no mention of injection site bleeding, flu-like symptoms, or pneumonia. Botox antitoxin was administered while she was in the intensive care unit.

The patient was admitted to the neurology clinic on April 22, 2022, following a 2-day stay in the critical care unit. The patient was conscious and willing to cooperate; she was orientated but had dysphonia.

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Light reflexes and eye movements were both normal. The motor assessment revealed the presence of finger abduction (4/5), forearm flexion and extension (5/5), forearm flexion and extension (2/5), neck flexion (2/5), neck extension (1/5), upper extremity bilateral arm abduction (4/5), arm adduction (5/5), and wrist extension and flexion (5/5). Leg extension in the lower extremities was 4/5; leg flexion, leg abduction, foot dorsiflexion, and foot plantar flexion were 5/5. Deep tendon reflexes were hypoactive in four limbs. Plantar reflexes were flexor on both sides.

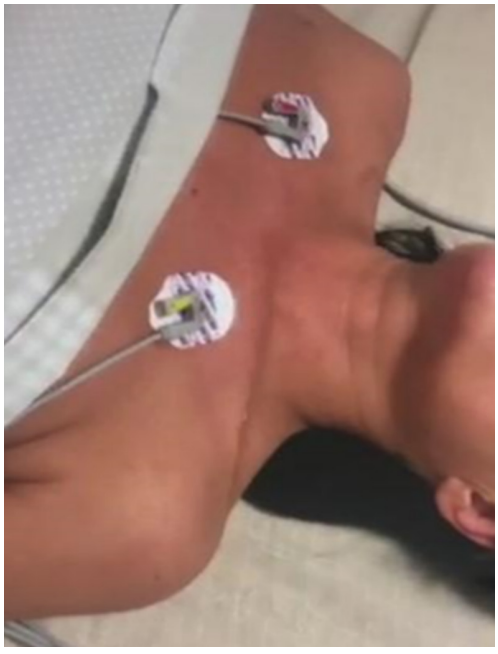

Figure 1. Intensive care unit while the patient is lying flat

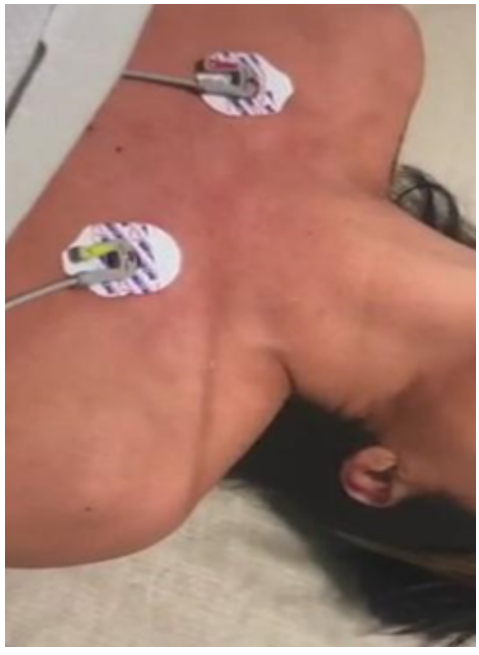

Figure 2. Intensive care unit; head drops back when the body is slightly raised

Tests were planned to differentiate the patient's diagnosis. On the second day after being admitted to the neurology unit, a nerve conduction study and repetitive stimulation study were performed on the patient.

A Synergy on Nicolet Viking Quest EMG instrument was used to conduct the electrodiagnostic assessment. Both the motor nerve conduction study and the sensory nerve conduction study were determined to be normal 9 days following the injection conduction measures. According to needle EMG, the left sternocleidomastoid muscle revealed uncommon denervation findings.

Repetitive stimulations at 3, 15, and 20 Hz were administered on the peroneus nerve (recording from the extensor digitorum brevis muscle), the N. ulnaris nerve (recording from the abductor digiti minimi muscle), and the N. accessorius nerve (recording from the trapezius muscle). Repetitive 3 Hz stimulation response in the N. peroneus and N. ulnaris was normal, followed by an incremental response to repetitive 15 Hz stimulation.

A 3 Hz repeated stimulation of the left trapezius muscle innervated by the left N. accessorius nerve was normal, with a decremental response at 15 Hz. As a result, the study found that neuromuscular junctions were involved (Figures 3, 4, 5). However, when the left N. accessorius nerve was stimulated, there was a decremental response, which we attributed to structural denervation of the left cervical muscles. The patient's left sternocleidomastoid muscle was noticeably atrophic.

Mild spinal stenosis on C4-C5-C6 was detected by cervical magnetic resonance imaging. Radicular root involvement was not visible. Likewise, there was no evident spinal cord disease. The test results were as follows: muscle tyrosine kinase antibody:

Anatomy / Train	Rate Hz	Ampl. mV	Ampl 4-1 %	Fac Ampl %	Area mVms	Area 4-1 %	Fac Area %	Time
R Peroneal - EDB								
Baseline	2	3.9	-3	100	14.8	-6.6	100	0:00:00
Post Exercise @0:00	3	4.0	-4.9	102	15.7	-10.1	106	0:00:22
@0:30	5	4.1	-3.6	104	15.6	-12.8	106	0:01:00
@1:00	15	3.9	29.2	100	15.3	-16.4	103	0:01:30
@2:00	50	50.1	-89.2	1280	30.4	-74.5	206	0:02:30
R Ulnar - ADM								
Baseline	2	8.3	-4.9	100	24.1	-1.6	100	0:00:00
Post Exercise @0:00	3	7.6	-2	90.9	24.3	-3.7	101	0:00:39
@0:30	15	8.3	9.2	99.6	23.0	-8.9	95.4	0:01:34
@1:00	30	28.7	9.9	344	55.7	-24.3	232	0:02:09
R Accessory (spinal) - Trapezius								
Baseline	2	3.6	10.1	100	25.3	3.7	100	0:00:00
Post Exercise @0:00	3	3.8	-9.8	105	21.8	1.1	86.1	0:00:19
@0:30	15	3.0	-21.8	83.2	17.3	-16.5	68.3	0:00:44
@1:00	20	3.0	-5.7	81.3	17.0	-25.3	67.2	0:01:30

Figure 3. Repetitive stimulation of N. peronealis, N. ulnaris, and N. accessorius

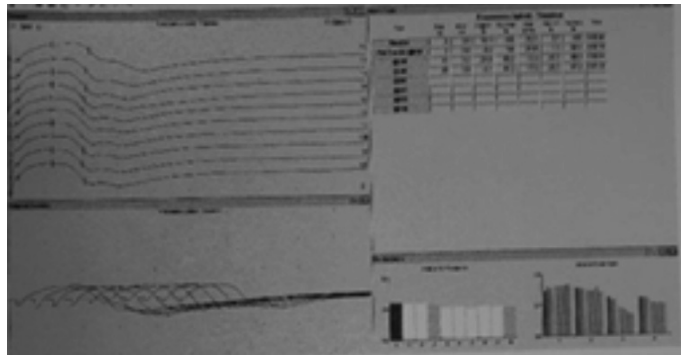

Figure 4. Left N. accessorius 3 Hz repetitive stimulation

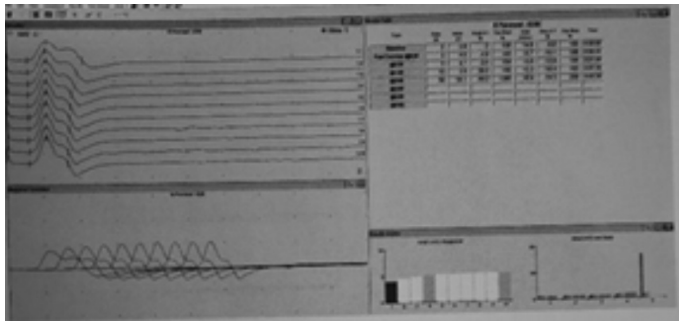

Figure 5. Right N. peronealis 3 Hz repetitive stimulation

0.2 (negative), and anti-acetylcholine receptor antibody: 0.01 (negative).

Numerous neurological illnesses can benefit from the use of botulinum neurotoxin as a treatment. Distant effects from local therapy are possible. BoNT was shown to diffuse to neighboring muscles. Shorter intervals between doses, the use of greater amounts of fluid for injection, higher single injection dosages, and axonal transmission can all lead to botulism. AbobotulinumtoxinA exhibits more dissemination than onabotulinumtoxinA with regard to the type of toxin utilized. When injected into face muscles, where the gap between the targeted and untargeted muscles may be incredibly narrow, this propagation feature may be more significant than other considerations (4).

In systemic non-iatrogenic botulism, the electrodiagnostic assessment often reveals normal conduction rates. A reduced compound muscle action potential is a possible finding; however, this generally occurs exclusively in the proximal muscles and may go unnoticed in regular assessments (5). Iatrogenic botulism is thought to be caused by the direct injection of toxins into the vascular system, even though it is common procedure to inspect the syringe before injection to ensure it is not in a vein. Iatrogenic botulism has been observed at places far from the local injection via anterograde and retrograde axonal transmission. Overall, the risk of adverse effects from BoNT injections rises with the amount of the toxin and varies depending on the target disease.

When BoNT is injected into the masseter muscles, as it was in our case, a rare adverse reaction of botulism called "drop-head" results.

Under no circumstances can we forget that BoNT is an extremely potent neurotoxin, and that only highly qualified medical professionals with extensive expertise should use BoNT to prevent botulism.

Ethics

Informed Consent: Obtained.
Peer-review: Externally peer-reviewed.

Authorship Contributions

Surgical and Medical Practices: K.İ.B., Concept: K.İ.B., Design: K.İ.B., Data Collection or Processing: K.İ.B., Analysis or Interpretation: H.O., Literature Search: K.İ.B., S.K., Writing: K.İ.B.

Conflict of Interest: No conflict of interest was declared by the authors.

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