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Case Report

Iatrogenic Ptosis Mimicking Cranial Nerve III Palsy Following Cosmetic Botox Injection: A Case Report.

Moath Alshanqity [1*], Lujain Filimban[2], Sadaqah Wazzan[3]

[1]Assistant Consultant, Emergency Medicine, King Abdulaziz Medical City, Ministry of National Guard Health Affairs, Jeddah, Saudi Arabia
[2]Emergency Medicine Resident, King Abdulaziz Medical City, Ministry of National Guard Health Affairs, Jeddah, Saudi Arabia
[3]Emergency Medicine Resident, King Abdulaziz Medical City, Ministry of National Guard Health Affairs, Jeddah, Saudi Arabia
[*]Corresponding author: Moath Alshanqity – Email: Moath.alshanqity@gmail.com

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Abstract

We present a 40-year-old female who developed iatrogenic ptosis mimicking cranial nerve III palsy after undergoing a cosmetic treatment to treat periorbital hyperpigmentation. Within 48 hours of receiving what was believed to be tranexamic acid (TXA) injections, the patient developed isolated left ptosis and generalized headache. Stroke, cerebral venous thrombosis (CVT), subarachnoid hemorrhage (SAH), and cranial nerve injury were among the differential diagnoses upon presentation. No insult to the central nervous system was found during the initial workup, which included neuroimaging. Although TXA was initially suspected due to its prothrombotic potential and the patient's history of pulmonary embolism, it has no known neurotoxic effects, and the pattern of findings did not support a thrombotic or central etiology. Botulinum toxicity (BoNT) was not initially suspected. It was discovered that a BoNT injection was mistakenly given into the patient's upper eyelid without her consent, according to a follow-up with the patient and her dermatological clinic. This case highlights the significance of patient safety procedures, follow-up correspondence, and a comprehensive history in aesthetic practice.

Keywords: Cranial nerve palsy; botulinum toxin; oculomotor nerve; cosmetic injection; iatrogenic botulism; medical error; patient safety

1. Introduction

A neurotoxic protein labeled botulinum toxin (BoNT) is frequently utilized in aesthetic therapy to alleviate dynamic facial wrinkles and relax muscles. Even though its cosmetic application is thought to be safe, adverse effects such as ptosis, diplopia, and facial asymmetry have been documented. Involvement of the cranial nerves, especially isolated oculomotor palsy, is exceedingly uncommon and typically linked to incorrect dosage, method, or toxin diffusion [1-3].

This report describes a patient who, after receiving cosmetic periorbital injections initially believed to be

TXA, developed iatrogenic ptosis mimicking cranial nerve III palsy. Follow-up revealed that BoNT had been accidentally injected into the upper eyelid, leading to localized neuromuscular blockade of the levator palpebrae superioris, rather than a true axonal injury to the oculomotor nerve.

2. Case Presentation

A 40-year-old Middle Eastern woman arrived at King Abdulaziz Medical City (KAMC), Jeddah's emergency department, with a three-day history of progressive drooping of her left eyelid and a diffuse headache. The day following a cosmetic procedure that involved injecting TXA into the periorbital area to treat hyperpigmentation, the symptoms started. Given her past medical history of provoked pulmonary embolism, TXA's known prothrombic potential initially raised concern for a vascular complication. However, the patient denied experiencing visual loss, dysphagia, diplopia, or systemic weakness. Her earlier medical history included several elective cosmetic treatments and obstructive jaundice treated with ERCP. She denied having any recent infections and was not on regular medications. On neurological examination, the patient was alert and oriented. Cranial nerve testing revealed complete left eyelid ptosis and mild outward deviation of the globe, but extraocular movements were full and symmetric without nystagmus, and pupils were reactive and equal. Motor strength, sensation, reflexes, coordination, and gait were normal. At the time of presentation, the working diagnosis included stroke, cerebral venous thrombosis (CVT), or a possible peripheral cranial neuropathy. BoNT exposure was not initially suspected due to lack of documentation or consent for its use.

- **Investigations**

Complete blood counts, electrolytes, liver and kidney function panels, and inflammatory markers were among the first tests performed in the emergency room; all of them were within normal ranges. A urinalysis was obtained as part of routine workup and revealed positive findings for nitrites and leukocyte esterase. Although these results were incidental and not related to the presenting neurologic symptoms, the patient was empirically started on ceftriaxone. To rule out dural venous sinus thrombosis, a non-contrast CT venogram of the brain was carried out; the results were unremarkable.

- **Follow-up and Outcome**

The patient was admitted for additional assessment after being referred to neurology. An MRI of the brain and orbits showed no signs of structural abnormalities, mass effect, or infarction. A thrombophilia screen revealed a slight decrease in protein S activity. Tests for acetylcholine receptor antibodies and autoimmune screening were both negative. These results ruled out an infectious origin, demyelinating illness, or central lesion. After receiving symptomatic treatment, the patient was discharged.
She was told to follow up with the dermatology clinic to investigate a potential medication or administration error during her neurology outpatient appointment. In a subsequent follow up call initiated by the Emergency Department the patient then reported that the dermatology clinic had verified, following an internal investigation and review of CCTV footage, that a syringe containing BoNT had been accidentally provided and injected into the left upper eyelid in place of the intended TXA. The patient claimed that she had not given permission for BoNT to be administered. The dermatologist and the assistant nurse miscommunicated, according to the clinic. Despite the procedural error, the patient later provided verbal informed consent for publication of her case, including all clinical details and follow-up outcomes.

3. Discussion

BoNT is a potent neurotoxin that inhibits acetylcholine release at neuromuscular junction, resulting in temporary muscle paralysis. Although its effects are typically localized, unintended diffusion or misplacement can result in unintentional paralysis. Rarely, BoNT injections have been linked to oculomotor nerve palsy, frequently because of incorrect technique or dosage [1-3].
In this case, the clinical picture was more consistent with a cranial nerve III palsy mimic rather than true axonal nerve injury. The patient presented with isolated upper eyelid ptosis, preserved extraocular movements, and normal pupillary responses suggesting localized paresis of the levator palpebrae superioris from BoNT diffusion, not a central or peripheral nerve lesion. The rapid onset of symptoms (within 24 hours), focal nature of findings, and absence of radiologic abnormalities support a diagnosis of localized BoNT induced neuromuscular blockade. TXA, although used off-label

for periorbital hyperpigmentation, has not been reported to cause cranial neuropathies [4]. While the patient had an incidental finding of positive urinalysis and a history of thrombophilia, these findings were not clinically contributory. Empirical ceftriaxone was administered during the initial workup, but the patient showed no systemic signs of infection, and the treatment had no effect on the neurologic outcome. Importantly, the discovery of the injection error during follow-up- confirmed through CCTV review was pivotal in establishing the diagnosis and raises significant concerns regarding clinic medication safety, procedural communication, and informed consent. The sequence of events is consistent with previous reports of early-onset periocular complications, even though the patient's ptosis developed within 24 hours of the injection, which is slightly earlier than the typical onset window of 24 to 72 hours for BoNT effects, with peak activity typically occurring at 7 to 14 days [5]. As early as the first day following injection, ptosis has been demonstrated to result from diffusion to the levator palpebrae superioris muscle, especially when the toxin is misdirected into the upper eyelid or brow region [6]. In the present case, a localized cause rather than systemic botulism or vascular injury is further supported by the fact that the ptosis did not worsen over the next few days. Management of BoNT induced eyelid ptosis is primarily conservative, as the condition results from temporary neuromuscular blockade and typically resolves as the toxin is degraded over time. Symptom onset usually occurs within 24 to 72 hours, with improvement beginning in 2 to 8 weeks and full recovery expected within 3 to 6 months in most cases, depending on the dose, injection site, and extent of diffusion [5].In this case, the patient was prescribed lubricating eye drops to protect the exposed cornea from drying and advised to use eyelid taping to assist with functional vision and maintain ocular surface hydration. No pharmacologic reversal agents were initiated. While topical alpha-adrenergic agonists such as apraclonidine 0.5% may be used to stimulate Müller's muscle and temporarily elevate the eyelid by 1–3 mm, their effectiveness varies, and they are most useful in patients with mild to moderate ptosis [6]. BoNT antagonists such as antitoxins are not indicated for localized cosmetic overdosing and are reserved for systemic toxicity or large-dose accidental exposure [7]. The patient was informed that full recovery could take up to six months, and she is currently being monitored in follow-up. This case highlights the importance of early identification, supportive care, and comprehensive patient counseling on the prognosis and time course of iatrogenic complications from cosmetic injections. This case also highlights the critical importance of **procedural safety systems** in aesthetic practice. Preventive measures such as clear medication labeling and double-checking syringe contents before administration are essential to reduce the risk of procedural errors. Such safeguards are especially important in outpatient cosmetic clinics where medications like BoNT and TXA may be used concurrently and stored in similar formats. Incorporating standardized verification steps into routine workflow can reduce the risk of miscommunication and medication substitution, ultimately protecting patient safety.

4. Conclusions

The present case emphasizes how crucial it is to maintain vigilance on cosmetic treatments, make sure medications are properly verified, and have clear patient consent.

While BoNT is generally considered safe when properly administered, unintended delivery or diffusion can result in localized neuromuscular complications that mimic cranial nerve palsy. In this case, **iatrogenic ptosis mimicking** oculomotor nerve dysfunction occurred due to inadvertent upper eyelid injection. Prompt follow-up and patient communication were essential in uncovering the true etiology.

Such events highlight the need for vigilance in aesthetic practice and reinforce the value of detailed post-procedural assessment in identifying delayed or subtle iatrogenic errors.

Author Contributions:

All authors contributed to case analysis, manuscript drafting, and final review. All authors approved the submitted version.

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Conflicts of Interest:

The authors declare no competing interests.

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Informed Consent Statement: Informed consent was obtained from the patient for publication of this case report, including the clinical course, diagnostic workup, and follow-up findings. The patient understood that identifying details would be omitted and that no images would be published. The lack of consent for the BoNT procedure itself is described in the case narrative and is unrelated to the publication consent process.

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