Case Report

Optic Neuritis Visual Loss after Intragastric Botulinum Toxin A Injection: A Case Report

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Abstract

Intragastric botulinum toxin A is increasingly used as an endoscopic adjunct for weight reduction, and although generally safe, rare neurological and ophthalmic adverse effects have been reported. We describe the case of a 37-year-old woman who developed dimming of vision in the left eye twelve days after intragastric botulinum toxin A injection. Visual evoked potential revealed delayed P100 latency in the affected eye, while the contralateral eye was normal, and magnetic resonance imaging demonstrated inflammation of the left optic nerve. Demyelinating disease was excluded through appropriate investigations. The close temporal relationship between botulinum toxin exposure and symptom onset, along with the absence of alternative etiologies, supported a diagnosis of toxin-related optic neuritis. This case highlights that intragastric botulinum toxin A may rarely precipitate optic nerve involvement and emphasizes the importance of patient counseling and prompt ophthalmologic assessment when visual symptoms occur.

Keywords: Botulinum Toxins; Intragastric; Optic Neuritis; Visual Evoked Potentials; Obesity; Adverse Effects.

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Introduction

Obesity is a major global health challenge associated with significant comorbidities and reduced quality of life [1]. Management strategies range from lifestyle modification and pharmacotherapy to bariatric surgery, with varying degrees of efficacy and risk [2]. In recent years, intragastric injection of botulinum toxin A has been introduced as a minimally invasive endoscopic approach to delay gastric emptying and promote satiety, thereby assisting weight reduction [3]. While botulinum toxin A is widely regarded as safe, its systemic diffusion and rare neurological or ophthalmic complications have been documented, including ptosis, diplopia, and optic nerve involvement in non-ocular applications [4]. The visual evoked potential is a sensitive tool for detecting abnormalities of the visual pathway, particularly in optic neuritis [5]. Reporting unusual presentations is essential to expand current understanding of the potential risks associated with intragastric botulinum toxin A and to alert clinicians to the possibility of rare but vision-threatening complications. This case describes optic neuritis with delayed P100 latency following intragastric botulinum toxin A injection for weight loss. Written informed consent was obtained from the patient for publication of this case report and any accompanying images. The study protocol and reporting were reviewed and approved by the institutional ethics committee, and all procedures were carried out in accordance with the principles of the Declaration of Helsinki.

Case Report

A 37-year-old woman was referred to Basir Eye Clinic for assessment because of reduced vision in her left eye, while her right eye remained normal. She reported that the visual problem had started twelve days after she underwent an intragastric injection of botulinum toxin A for weight reduction.

On electrophysiological testing, different stimulation methods were applied according to visual status. Because of the markedly reduced visual acuity in the left eye, flash stimulation was used to record the visual evoked potential, whereas pattern-reversal checkerboard stimulation was used for the right eye, which maintained full acuity. The left eye showed a flat response with a delayed P100 peak latency of 98 milliseconds, while the right eye produced responses within the laboratory's normal range.

Magnetic resonance imaging revealed inflammation of the left optic nerve but did not show any additional central nervous system lesions. A systemic pathological work-up was unremarkable, and laboratory testing excluded demyelinating disease.

The combination of symptom onset closely following intragastric botulinum toxin A exposure, unilateral delay in visual evoked potential latency, and radiological evidence of optic nerve inflammation, together with the exclusion of alternative causes, pointed toward a diagnosis of optic neuritis most likely related to the toxin injection.

Discussion

This case illustrates a rare but important complication of intragastric botulinum toxin A injection. Although botulinum toxin A is widely used in neurology, ophthalmology, and gastroenterology for both therapeutic and cosmetic indications, its systemic spread can occasionally lead to unexpected adverse effects [6].

In our case the patient's visual symptoms developed twelve days after intragastric botulinum toxin A administration, and this

close timing between the intervention and onset of optic neuritis supports a possible causal association.

The electrophysiological findings support optic nerve involvement. Visual evoked potential testing demonstrated delayed P100 latency in the affected eye, consistent with impaired conduction along the visual pathway. This aligns with prior evidence showing that VEP is sensitive for detecting optic nerve dysfunction, particularly in optic neuritis [7]. The normal VEP in the contralateral eye confirms the focal nature of the injury.

Neuroimaging provided further corroboration, with MRI demonstrating left optic nerve inflammation and no additional central nervous system lesions. Importantly, demyelinating disease was excluded through laboratory testing, which reduces the likelihood of multiple sclerosis or neuromyelitis optica spectrum disorder as underlying causes. The absence of systemic abnormalities further supports a toxin-induced mechanism.

Comparable cases have been documented in the literature. Khalili et al., [8] reported optic neuropathy in a patient who received botulinum toxin A in the medial rectus muscle for post-traumatic diplopia, where optic neuritis developed within two days and responded to corticosteroid therapy. Another report by Chun et al., [9] has described optic nerve involvement after botulinum toxin A injection into the masseter muscle, with visual field loss and abnormal retinal circulation findings. These examples, combined with the present case, highlight that ocular and optic nerve complications can occur even when botulinum toxin is injected away from the orbit.

The mechanism by which intragastric botulinum toxin A could induce optic neuritis remains uncertain. Possible explanations include systemic diffusion of the toxin, immune-mediated reaction, or idiosyncratic neurotoxicity affecting susceptible individuals [8].

Although rare, the event emphasizes the importance of careful patient selection, informed consent, and vigilance for visual symptoms following treatment. Overall, this case expands the spectrum of potential adverse effects associated with intragastric botulinum toxin A, underlines the diagnostic value of VEP and MRI in confirming optic nerve involvement, and stresses the role of early ophthalmological evaluation to prevent permanent visual loss.

Conclusion

This case shows that optic neuritis with delayed visual evoked potential and MRI evidence of optic nerve inflammation can occur after intragastric botulinum toxin A injection. The findings point to a likely toxin-related optic nerve involvement and stress the need for rapid ophthalmologic assessment when visual symptoms develop.

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Footnotes and Financial Disclosures

Conflict of interest:

The authors have no conflict of interest with the subject matter of the present manuscript.