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Blepharospasm Management with Botulinum Toxin in a Patient with Comorbid Psychiatric Illness: A Case Report and Literature Review

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Abstract

Blepharospasm is a focal dystonia of the orbicularis oculi muscles that frequently co-occurs with psychiatric comorbidities such as anxiety and depression. Botulinum toxin type A (BoNT-A) remains the first-line therapy and is highly effective; however, management becomes more complex when psychiatric symptoms require pharmacotherapy, as some psychotropics can worsen movement disorders. We present a 58-year-old Nigerian male with a 9-month history of disabling blepharospasm and comorbid depression and anxiety. He was treated with serial BoNT-A injections and psychotropic medications, including fluoxetine, mirtazapine, and lorazepam. The patient showed robust motor improvement with BoNT-A and gradual psychiatric stabilization with mirtazapine, which also coincided with better control of blepharospasm. This case highlights the importance of an integrated neurology-psychiatry approach in managing blepharospasm with psychiatric comorbidities. It further suggests that mirtazapine may provide dual benefits in mood stabilization and dystonia management.

Keywords: *Blepharospasm, Botulinum Toxin, Psychiatric Comorbidity, Mirtazapine, Focal Dystonia, Case Report.*

1. INTRODUCTION

Blepharospasm is a chronic focal dystonia primarily affecting the orbicularis oculi muscles, characterized by sustained, involuntary contractions that may progress to forceful eyelid closure, photophobia, and functional blindness [1]. Although it is a movement disorder of neurological origin, its clinical burden extends well beyond motor symptoms. Psychiatric comorbidities, particularly anxiety and depression, are highly prevalent in patients with blepharospasm and may precede the onset of dystonia, suggesting a bidirectional relationship between mood and motor dysfunction [2, 3].

From a pathophysiological standpoint, blepharospasm is believed to result from dysfunction in basal ganglia-thalamo-cortical circuits, with impaired inhibitory control over motor pathways [1]. Neuroimaging and neurophysiological studies support the involvement of abnormal sensorimotor integration and reduced cortical inhibition. Psychiatric comorbidities in dystonia may arise from shared pathophysiological mechanisms, including neurotransmitter dysregulation (dopamine, serotonin, and GABA), or may reflect the psychosocial burden of a highly visible and disabling movement disorder [3].

The mainstay of treatment is **botulinum toxin type A (BoNT-A)**, which acts at the neuromuscular junction to block acetylcholine release and reduce spasms [4]. Most patients experience symptomatic relief lasting 2–4 months, but repeated injections are necessary. While effective for motor symptoms, BoNT-A does not address psychiatric comorbidities, which require concurrent treatment with psychotropic medications or psychotherapy. The choice of psychiatric treatment is particularly challenging, as several classes of psychotropic agents—including typical antipsychotics and certain antidepressants—may worsen movement disorders [5, 6].

This necessitates careful consideration of pharmacologic interactions and multidisciplinary management strategies.

We present the case of a 58-year-old Nigerian male with severe blepharospasm and psychiatric comorbidity who was successfully treated with BoNT-A and mirtazapine. This case highlights the complexity of managing overlapping neurological and psychiatric conditions and underscores the importance of integrated, individualized care.

1.1. Case Presentation

A 58-year-old Nigerian male with no significant prior medical history presented with a 9-month history of progressive, involuntary bilateral eyelid closure, accompanied by intermittent upper body twitching.

These symptoms caused severe visual impairment, preventing him from performing daily activities such as reading and driving. He also reported persistent anxiety, insomnia, and depressed mood.

1.2. Medications at Presentation

- **Psychiatric:** Lorazepam 1 mg daily, Fluoxetine 20 mg daily, Mirtazapine 15 mg daily (later titrated to 30 mg)
- **Other:** Baclofen, Gabapentin, Acetaminophen, Latanoprost, Pantoprazole, Famotidine, Vitamin D3, Melatonin

Neurological examination revealed sustained bilateral orbicularis oculi spasms causing frequent and prolonged eyelid closure. Psychiatric evaluation confirmed depression, anxiety, and sleep disturbance.

Table 1. *Timeline of Clinical Course and Interventions*

Date	Clinical Status	Interventions / Notes
Apr 29, 2024	Depressed mood, insomnia	Mirtazapine increased to 30 mg
Jul 10, 2024	Anxiety and insomnia persisted; blepharospasm unchanged	Continued psychiatric therapy
Aug 26, 2024	BoNT-A injections administered	Mood remained low
Oct 26, 2024	Modest improvement in eyelid spasms	Psychiatric symptoms ongoing
Dec 17, 2024	Reduced eyelid closures	Psychiatric instability continued
Mar 4, 2025	Mood swings persisted	Psychotherapy discussed
May 13, 2025	Marked motor improvement	Depressive symptoms improved
Jun 25, 2025	Sustained motor gains	Psychiatric state stable
Aug 20, 2025	Dystonia extended to tracheal/neck region; labored breathing, bulging eyes	BoNT-A therapy continued; mood stable

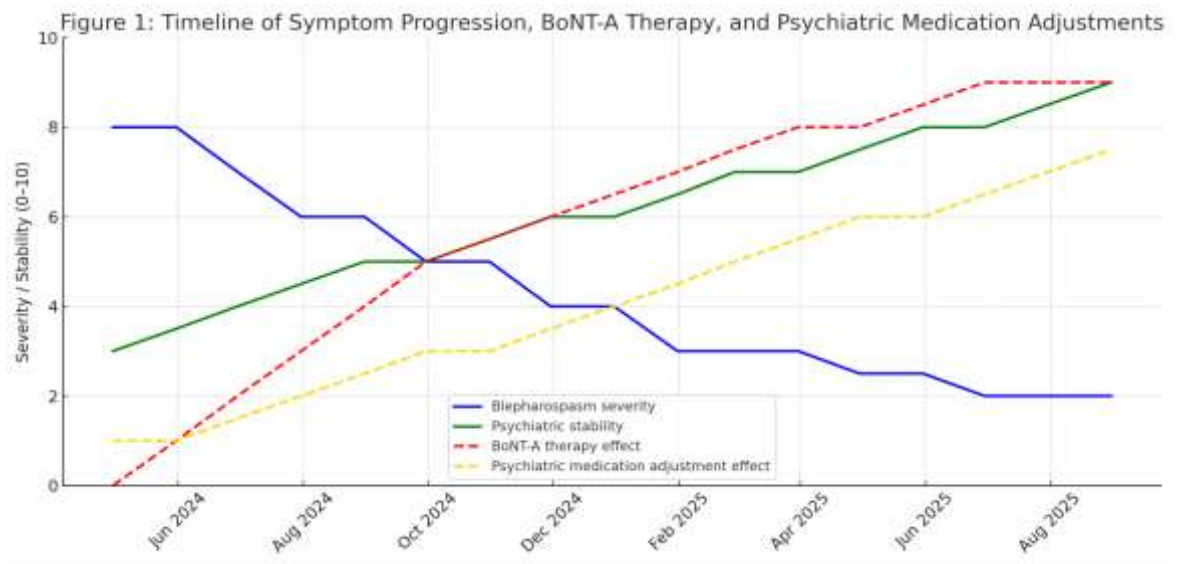

Figure 1. *Shows the progression of blepharospasm severity (blue) and psychiatric stability (green) over time, with markers for BoNT-A injections (red dotted line) and psychiatric medication adjustments (yellow dotted line). Improvement in motor symptoms correlates with BoNT-A therapy, while psychiatric symptoms fluctuate, highlighting the need for integrated care.*

The patient received his first series of BoNT-A injections on August 26, 2024. Follow-up over the subsequent months showed a clear, positive trajectory in his motor symptoms. By May 2025, his blepharospasm was well-controlled, with markedly improved functional vision. His psychiatric symptoms were slower to stabilize but showed gradual improvement with

medication adjustments and supportive therapy. Notably, the administration of mirtazapine was associated with improvement of his blepharospasm. In a later follow-up, the dystonia had progressed to involve cervical and laryngeal muscles, a known evolution of the disorder, which required continued BoNT-A therapy to new muscle groups.

2. DISCUSSION

This case illustrates several critical considerations in the management of blepharospasm with psychiatric comorbidities.

2.1. BoNT-A Remains the Cornerstone of Therapy

Our patient experienced a sustained and robust response to BoNT-A injections, consistent with the literature demonstrating BoNT-A's efficacy in reducing orbicularis oculi contractions and improving quality of life [4]. The improvement in motor function enabled the patient to regain functional vision, independence in daily activities, and overall social functioning—highlighting how effective motor control can have downstream psychosocial benefits.

2.2. Psychiatric Comorbidities in Blepharospasm are Common and Clinically Significant

Multiple studies have documented elevated rates of depression and anxiety in patients with blepharospasm [2, 3]. These psychiatric conditions may arise from the neurobiology of dystonia itself through alterations in basal ganglia and limbic circuits or as a consequence of the social and functional disability imposed by the disorder. The coexistence of psychiatric symptoms can exacerbate motor disability and negatively influence treatment adherence. In this case, our patient's persistent anxiety and insomnia initially limited the perceived benefit of BoNT-A until psychiatric symptoms were addressed in parallel.

2.3. Psychotropic Management Must be Tailored to Minimize Iatrogenic Worsening of Dystonia

Certain psychotropics, particularly dopamine antagonists and some SSRIs, can exacerbate dystonia or precipitate tardive syndromes [5, 6]. The use of mirtazapine in our patient provided dual benefits: improvement of depressive and anxiety symptoms and stabilization of motor symptoms. Mirtazapine's pharmacological profile as a noradrenergic and specific serotonergic antidepressant (NaSSA) makes it less likely to induce extrapyramidal symptoms compared to SSRIs or antipsychotics. Its sedative and anxiolytic effects may also reduce motor overactivity, indirectly supporting dystonia management [7]. In an open label study, mirtazapine was shown to improve abnormal movement in patients with hemifacial spasm, similar to blepharospasm in terms of involuntary facial muscle contractions in 80% of the patients [8]. The patients' demonstrated significant improvement in involuntary movement within 8 to 60 days of treatment [8]. Emerging case reports suggest mirtazapine may play a stabilizing role in movement disorders, though systematic evidence is still lacking.

2.4. Disease Progression and Longitudinal Care

Despite initial control, our patient's dystonia spread to cervical and laryngeal regions, a known progression of focal dystonia syndromes. This emphasizes the importance of ongoing monitoring, longitudinal treatment planning, and flexibility in expanding BoNT-A therapy to additional muscle groups as the disease evolves.

2.5. The Need for Integrated Multidisciplinary Management

The overlap between neurological and psychiatric manifestations underscores the need for collaborative care models. Neurologists provide targeted motor interventions with BoNT-A, while psychiatrists manage mood and anxiety disorders that can exacerbate disability. Psychotherapy may also provide valuable coping strategies, improving resilience in the face of chronic disease. In our case, psychiatric stabilization was crucial in maximizing the benefits of motor treatment, and motor stabilization reduced the psychosocial burden contributing to depression demonstrating a reciprocal therapeutic synergy.

Overall, this case contributes to the limited but growing body of literature on the intersection of dystonia and psychiatric illness, highlighting opportunities for improved outcomes through careful pharmacologic selection and integrated care pathways.

3. CONCLUSION

This case underscores several key lessons in the management of blepharospasm with psychiatric comorbidities. First, BoNT-A remains the gold standard for motor symptom control and provides profound improvements in functional vision and quality of life. Second, psychiatric symptoms

must be addressed in parallel, as untreated anxiety and depression can amplify disability, limit treatment effectiveness, and diminish quality of life. Third, psychotropic selection requires careful consideration, as some agents may exacerbate dystonia. In our patient, mirtazapine provided dual benefit in improving psychiatric symptoms while coinciding with stabilization of motor function, suggesting it may be a particularly suitable antidepressant in this population.

Beyond pharmacologic management, this case illustrates the necessity of a truly integrated neurology-psychiatry model of care. Blepharospasm is not simply a motor disorder, it is a neuropsychiatric condition with multidimensional consequences. Optimal treatment requires coordinated interventions addressing both movement and mood.

Finally, our case demonstrates the natural progression of dystonia over time, with extension to cervical and laryngeal regions despite effective local treatment. This progression reinforces the need for long-term follow-up, flexible treatment strategies, and continued research into therapies that target both motor and psychiatric dimensions of dystonia. Greater awareness among clinicians of the psychiatric overlap in blepharospasm, and of safe pharmacologic choices such as mirtazapine, may significantly improve patient outcomes.

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Patient Consent: "Written informed consent was obtained from the patient for publication of this case report."

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