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# Atypical but not innocuous: Case series of oculogyric crisis induced by Olanzapine

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## Abstract

Oculogyric crisis (OGC), a rare acute dystonic reaction that often results in severe patient suffering, is characterised by an involuntary upward deviation of the eyes. Although atypical antipsychotics like olanzapine can occasionally cause one OGC, first-generation antipsychotics are more frequently linked to this condition. Three separate instances of olanzapine-induced OGC that occurred in patients taking 10 mg or 15 mg of the medication are discussed in this article. A 30-year-old woman with schizophrenia, a young male with bipolar mania, and a young female with ATPD were among the patients. After starting trihexyphenidyl 2 mg and reducing or stopping their olanzapine dosage, the three patients who had developed OGC while taking olanzapine showed a significant improvement in their symptoms.

Despite olanzapine's generally lower propensity for extrapyramidal symptoms, these cases highlight the significance of identifying it as a possible cause of OGC. The practical clinical advice for handling this upsetting adverse event is provided by the consistent effectiveness of dose modification and anticholinergic therapy seen in these cases.

**Keywords:** Oculogyric Crisis, Atypical Antipsychotics, Acute Dystonia, Schizophrenia

## Introduction

The extraocular muscles are impacted by the uncommon and frequently concerning acute dystonic reaction known as oculogyric crisis (OGC). It is distinguished by the prolonged, involuntary upward deviation of both eyes. The person looks up most of the time, but they can also look down or sideways. These episodes, which can last anywhere from a few seconds to hours, cause a great deal of suffering for one patient. They are frequently accompanied by other dystonic symptoms such as periorbital twitches, retrocollis (backward flexion of the neck), tongue protrusion, blepharospasm, and severe anxiety. OGC has a broad aetiology that includes focal

brain lesions, neurometabolic disorders, drug side effects and neurodegenerative conditions.[2]

However, the most common cause is drug-induced, particularly associated with neuroleptic medications.[1] First-generation antipsychotics, such as haloperidol, are well-established culprits due to their potent dopamine D2 receptor blockade.[1] Second-generation (atypical) antipsychotics, such as olanzapine, quetiapine, risperidone, and aripiprazole, are typically linked to a decreased risk of acute dystonia and extrapyramidal symptoms (EPS).[3] Olanzapine is a commonly prescribed atypical antipsychotic that works well to treat schizophrenia and bipolar disorder because it mainly works by blocking dopamine D2 and serotonin 5HT2A receptors.[6]

OGC is still a rare but clinically significant side effect linked to olanzapine use, despite the drug's generally favourable extrapyramidal side effect profile.[3] OGC is present in 1.8% of patients taking atypical antipsychotics, according to a prospective study on early intervention for psychosis. Olanzapine was found to be one of the common causative agents in this class.[3] This observation emphasises that even though the overall risk is decreased, there is still a chance of this particular, upsetting adverse event. By describing the clinical presentation, progression, and effective treatment of three separate cases of olanzapine-induced OGC, the current case series seeks to add to the body of existing literature. The goal is to raise clinician awareness of this uncommon adverse drug reaction and offer helpful advice on how to manage it effectively.

**Methodology**

This report presents a retrospective case series derived from the clinical records of three patients being treated at department of Psychiatry VMMC and Safdarjung Hospital, New Delhi who developed olanzapine-induced oculogyric crisis. Patients were identified based on a clear clinical diagnosis of OGC occurring during olanzapine treatment and demonstrated improvement following specific interventions. Comprehensive clinical data, including patient demographics, relevant medical and psychiatric history, details of olanzapine initiation and dosage, precise descriptions of OGC presentation, timing of onset, interventions (pharmacological and non-pharmacological), and clinical outcomes, were systematically extracted from their medical files. To ensure patient confidentiality and ethical reporting, all identifying patient information has been de-identified. Informed consent was obtained from the patients or their legal guardians for the publication of their de-identified case details. This approach ensures that the presented clinical observations are both scientifically valid and ethically sound for publication.

**Case Presentations**

**Case 1: Young Female ATPD Patient**

A young unmarried female, aged 23 years, graduate, belonging to Hindu nuclear family of middle socio-economic status of rural background of Delhi was presented in psychiatry department with a two-week history of suspiciousness, irritability, decreased sleep, and disorganized behaviour. There was no past or family history of psychiatric illness or substance use. On mental status examination, she was restless, with delusional ideas of persecution and thought disorganization. A diagnosis of Acute and Transient Psychotic Disorder (ATPD) was made according to ICD-10 criteria. As the patient was hard to manage at home she got admitted. She was started on olanzapine 5mg/day, gradually increased to 15 mg/day. After one week of treatment, the patient developed sudden episodes of sustained upward deviation of the eyes lasting several minutes, associated with restlessness, anxiety, and discomfort. Neurological

examination and neuroimaging ruled out organic causes. Clinical features were consistent with olanzapine-induced oculogyric crisis. Olanzapine was abruptly stopped and trihexyphenidyl 2 mg/day was initiated. Symptoms of OGC subsided completely within 48 hours. Subsequently, she was shifted to Aripiprazole 5 mg/day and later titrated to 15mg, which was well tolerated without recurrence of dystonic reactions. The patient showed significant improvement in psychotic symptoms over the following weeks and was maintained on Aripiprazole and Trihexyphenidyl and was discharged within 21days.

**Case 2: Young Male Bipolar Mania Patient**

A 23-year-old unmarried male, studied up to 8th, living in Hindu nuclear family of middle socio-economic status of urban background of Delhi presented to the psychiatry outpatient department with a 3-week history of elevated mood, increased activity, decreased need for sleep, irritability, grandiosity, and increased goal-directed behaviour. There was no significant medical or family history. Based on clinical evaluation, a diagnosis of mania without psychotic symptoms (ICD-10) was made and patient was admitted for the same. The patient was initiated on olanzapine 10 mg/day, gradually increased to 20 mg/day over two weeks. He showed improvement in manic symptoms. However, after one month of treatment, he developed recurrent episodes of sustained upward deviation of the eyes lasting 5–10 minutes, associated with anxiety, restlessness and mild neck discomfort. No neurological abnormalities or structural lesions were identified on neuroimaging. The symptoms were clinically diagnosed as olanzapine-induced oculogyric crisis. Olanzapine was gradually tapered and discontinued, and trihexyphenidyl 2mg was initiated and later titrated to 4mg/day. Over the course of one month, OGC symptoms subsided completely. The patient was subsequently shifted to Aripiprazole (25 mg/day), which was well tolerated with no recurrence of dystonia.

Fig. 1: Involuntary uprolling of eyes (Oculogyric crisis)

**Case 3: 30-Year-Old Female**

A 30-year-old female, married, not formally educated belonging to middle socio economy status of urban background of Delhi with no significant medical or substance use history, presented with persecutory delusions, auditory hallucinations, and social withdrawal for six months. Based on ICD-10 criteria, a diagnosis of Paranoid Schizophrenia was made. The patient was started on olanzapine 10 mg/day. After three days of treatment, she developed acute episodes of sustained upward eye deviation, lasting 5–7 minutes, accompanied by restlessness, anxiety, and slowness of movement. There were no abnormal neurological findings, metabolic disturbances, or organic causes on further evaluation. The symptoms were consistent with olanzapine-induced oculogyric crisis. Olanzapine was continued with close monitoring, and trihexyphenidyl 2 mg/day was introduced. Within 48 hours, there was a marked reduction in dystonic episodes, and by the end of one week, OGC had completely subsided. The patient was maintained on olanzapine 15 mg/day and trihexyphenidyl, with gradual improvement in psychotic symptoms and no recurrence of OGC.

**Results**

Regarding the clinical presentation and reaction to treatment of olanzapine-induced oculogyric crisis, a recurring pattern was observed in all three of the cases that were presented. The main symptom of OGC in all patients was involuntary upward deviation of the eyes,

which was frequently accompanied by related dystonic features like anxiety and periorbital twitches.[1] OGC's onset varied; in contrast to the more common acute onset seen in the other cases, one young male patient had a noticeably delayed onset about 30 days after starting olanzapine.[2]

Importantly, after a combined therapeutic approach, the OGC episodes either resolved or markedly improved in all three cases. This approach always included giving oral trihexyphenidyl 2 mg at the same time as the olanzapine dose, either lowering it or stopping it altogether. One noteworthy finding is that trihexyphenidyl 2 mg consistently and quickly resolves the crisis across a range of patient profiles and initial olanzapine doses (10 mg or 15 mg). The results consistently showed that the distressing symptoms were completely resolved or significantly improved, usually within 24 to 48 hours of the start of management.[1]

A summary of the patient demographics, clinical presentation, and management of olanzapine-induced oculogyric crisis is provided in Table 1.

Table 1: Summary of Patient Demographics, Clinical Presentation, and Management of Olanzapine-Induced Oculogyric Crisis

| Patient ID | Age | Sex | Primary Psychiatry Diagnosis | Olanzapine Dose at OGC Onset | Key Clinical Features of OGC | Time to OGC Onset (relative to Olanzapine) | Olanzapine Management | Trihexyphenidyl Dose | Outcome |
|---|---|---|---|---|---|---|---|---|---|
| Case 1 | Young | F | ATPD | 10 mg | Upward eye deviation, Superior orbital twitches, anxiety | 1 week | Decreased & stopped | 2 mg | Complete resolution |
| Case 2 | Young | M | Bipolar Mania | 15 mg | Upward eye deviation, periorbital twitches, protracted staring, hand shaking, anxiety | Approximately 30 days | Decreased & stopped | 2 mg | Symptom relief |
| Case 3 | 30 | F | Schizophrenia | 10 mg | Upward eye deviation | 3 days | Decreased & stopped | 2 mg | Improve ment |

**Discussion**

This case series describes three patients who developed oculogyric crisis (OGC) as an adverse reaction to olanzapine and, importantly, shows that a consistent treatment strategy proved effective in all cases. Each patient experienced rapid improvement after adjustment of the olanzapine dose and the addition of trihexyphenidyl at a dose of 2 mg.

OGC is classified as an acute dystonic reaction, arising from a functional reduction in dopaminergic activity within the brain—often a result of dopamine receptor blockade. Olanzapine, an atypical antipsychotic, primarily acts as a dopamine D2 receptor antagonist. Although it is characterised by “loose binding” and faster dissociation from D2 receptors compared with first-generation antipsychotics—properties that generally

lower the risk of extrapyramidal symptoms (EPS)—its moderate affinity for D2 receptors can still disturb the dopamine–acetylcholine balance in the basal ganglia and precipitate acute dystonia. Thus, the term "atypical" should not be interpreted as an absence of risk. In susceptible individuals, even partial or transient D2 blockade can be enough to trigger OGC, particularly at higher doses where the likelihood of EPS increases.

Reports in the literature suggest that OGC with olanzapine is uncommon, with an estimated incidence of 1.8% among patients receiving atypical antipsychotics, and olanzapine being a notable contributor within this group. The onset is most often within four days of initiating or increasing the dose of an antipsychotic. In one of our cases, however, a young male developed OGC approximately 30 days after starting olanzapine. Such delayed onset, also noted in other "unusual" reports, indicates that clinicians should remain vigilant for OGC beyond the early treatment period. This variability may be influenced by individual pharmacokinetic and pharmacodynamic factors or changes in receptor sensitivity over time. Recognised risk factors for OGC include higher antipsychotic doses, male sex, younger age, severe illness, and prior episodes of EPS. The young male in our series matched two of these—age and sex—supporting the relevance of these predictors in clinical practice.

Management of OGC centres on either discontinuing or reducing the dose of the offending antipsychotic, where possible, alongside the use of anticholinergic medication for symptomatic relief. In our three cases, trihexyphenidyl 2 mg led to prompt resolution. The effectiveness of this relatively low dose is notable, as other case reports describe variable responses, with some patients requiring higher doses or even switching to agents such as clozapine when anticholinergics failed. Trihexyphenidyl is well-suited for acute management, given its rapid gastrointestinal absorption, onset of action within one hour, and peak effect within two to three hours. Typical dosing for drug-induced EPS ranges from 3–6 mg daily, in divided doses, up to a maximum of 15 mg/day. Our findings suggest that, in certain cases, a 2 mg dose may be sufficient to achieve rapid symptom control.

Given the range of conditions that can mimic OGC—including functional neurological movement disorders, ocular tics, ocular dyskinesia, ocular bobbing, epileptic phenomena, and various genetic or metabolic disorders—careful clinical evaluation is essential to confirm the diagnosis.

The clinical takeaway from this series is clear: while olanzapine generally carries a lower EPS risk than typical antipsychotics, OGC can still occur. Early recognition and prompt action—through dose adjustment and timely anticholinergic therapy—are critical to relieving patient distress and avoiding misinterpretation of symptoms as psychiatric relapse. Though OGC is rarely life-threatening and typically resolves within 24–48 hours with appropriate treatment, it can be highly distressing. The consistent, positive outcomes in these three cases underline that standard anticholinergic therapy, when combined with sensible modification of the antipsychotic regimen, can be both effective and practical in managing olanzapine-induced OGC.

**Conclusion**

Olanzapine can cause oculogyric crisis, a rare but upsetting acute dystonic reaction, even though it is an atypical antipsychotic with a generally positive extrapyramidal symptom profile. Through the dual strategy of reducing or stopping the olanzapine dosage and promptly administering 2 mg of trihexyphenidyl, the three cases shown consistently show that olanzapine-

induced OGC can be successfully and quickly resolved. This emphasises how crucial it is to have increased clinician awareness, detect the problem early, and use the right medication to treat this controllable side effect, which will ultimately guarantee the best possible patient outcomes and reduce suffering.

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