Case Report *Open Access*

International Journal of Clinical Drug Practice & Pharmacotherapy

DOI: *doi.org/10.63721/26IJCDPP108*

A Rare Presentation of Olanzapine-Induced Oculogyric Crisis Associated with Glossoptosis

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Citation: *Sebin Sabu, Neena Bhatti, Girish Joseph, Abha Singh, Dinesh K Badyal (2026) A Rare Presentation of Olanzapine-Induced Oculogyric Crisis Associated with Glossoptosis . Int J. of Cli Drug Pract & Pharmaco 1:(1), 1-5. WMJ-IJCDPP-108*

Abstract

Adverse drug reactions (ADRs) are a significant cause of morbidity in patients receiving psychotropic medications. Oculogyric crisis (OGC) is a rare extrapyramidal adverse effect characterized by sustained upward deviation of the eyes due to disruption of dopaminergic neurotransmission in the basal ganglia. Although more commonly associated with first-generation antipsychotics, OGC has also been reported with second-generation agents such as olanzapine.

We report the case of a 17-year-old female who developed oculogyric crisis associated with glossoptosis following prolonged treatment with olanzapine.

The adverse drug reaction was identified during pharmacovigilance monitoring at Christian Medical College, Ludhiana. Causality assessment using the Naranjo Adverse Drug Reaction Probability Scale indicated a probable association. The patient showed complete clinical recovery following withdrawal of the offending drug. This case highlights a rare and unusual presentation of olanzapine-induced extrapyramidal symptoms and emphasizes the importance of early recognition and pharmacovigilance reporting.

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Submitted: 24.04.2026 **Accepted**: 29.04.2026 **Published**: 15.05.2026

Keywords: Olanzapine, Oculogyric Crisis, Glossoptosis, Adverse Drug Reaction, Pharmacovigilance

Introduction

Oculogyric crisis (OGC) is a rare dystonic reaction characterized by sustained, involuntary upward deviation of the eyes due to abnormal contraction of the extraocular muscles. It is most commonly associated with medications that block dopamine receptors, re-

sulting in disruption of the dopaminergic-cholinergic balance within the basal ganglia [1].

Although OGC has traditionally been linked to first-generation antipsychotics, increasing evidence suggests that second-generation antipsychotics, including olanzapine and risperidone, can also precipitate this condition. Olanzapine is widely prescribed for the management of schizophrenia and bipolar disorder due to its relatively favorable safety profile compared with older antipsychotics. However, rare neurological adverse reactions, including dystonic reactions such as OGC, have been reported in patients receiving olanzapine therapy [1-3].

In addition to ocular manifestations, involvement of other muscle groups may occur when disruption of dopaminergic pathways extends beyond the extraocular muscles. Abnormal contraction of orolingual musculature may lead to unusual clinical features such as glossoptosis, characterized by posterior displacement of the tongue. This presentation is exceedingly rare and has been scarcely reported in association with antipsychotic-induced movement disorders.

Data from the World Health Organization pharmacovigilance database (VigiAccess) indicate that while a large number of adverse drug reactions have been reported with olanzapine, oculogyric crisis remains uncommon, and glossoptosis is extremely rare [4].

In this context, we report a rare case of olanzapine-induced oculogyric crisis associated with glossoptosis in an adolescent patient. This case underscores the need for heightened clinical vigilance, even with commonly used second-generation antipsychotics, and emphasizes the role of pharmacovigilance in identifying atypical presentations of drug-induced movement disorders.

Case Presentation

This case was collected as a part of the Pharmacovigilance elective under the Department of Pharmacology, Christian Medical College, Ludhiana, which is an adverse drug reactions (ADR) Monitoring Centre. The case was assigned a Worldwide Unique Number (WUN): IN-IPC-301229586.

A 17-year-old female presented to the Emergency Department at Christian Medical College, Ludhiana, with complaints of sudden episodes of abnormal eye movements associated with difficulty in speech. She had been receiving olanzapine 10 mg once daily and escitalopram 10 mg once daily for schizophrenia and depression for approximately six months prior to the onset of symptoms. The patient developed intermittent episodes characterized by sustained upward deviation of both eyes lasting several minutes. During these episodes, she remained conscious but was unable to voluntarily control the eye movements. The episodes were associated with anxiety and significant discomfort.

In addition to ocular symptoms, the patient developed glossoptosis, manifested as posterior displacement of the tongue leading to difficulty in speech. There was no history of trauma, seizure disorder, or prior neurological illness. On examination during an episode, conjugate upward deviation of the eyes was observed with preserved pupillary reflexes. No focal neurological deficits were identified. Neuroimaging revealed no structural abnormalities.

Given the temporal association with olanzapine therapy, a drug-induced adverse reaction was suspected. Olanzapine was discontinued, and supportive management was initiated. Following withdrawal, the patient showed gradual and complete resolution of symptoms, with no recurrence during follow-up.

Causality, Severity, and Preventability Assessment

Causality assessment of the suspected adverse drug reaction was performed using the Naranjo Adverse Drug Reaction Probability Scale, a validated tool that estimates the likelihood of a causal relationship between a drug and an observed clinical event. The patient achieved a total score of 6, which categorizes the reaction as a probable adverse drug reaction. This assessment was based on a clear temporal relationship between olanzapine exposure and onset of symptoms, improvement following drug withdrawal (positive dechallenge), absence of alternative etiologies, and the presence of objective clinical findings [5].

The severity of the adverse drug reaction was evaluated using the Modified Hartwig and Siegel Severity Assessment Scale, which classified the reaction as Level 4 (moderate severity. This classification reflects that the reaction required discontinuation of the suspected drug and active medical management, along with the need

for hospital-based care, but did not result in permanent harm or disability [6].

Preventability assessment was conducted using the Schumock and Thornton Preventability Scale, which indicated that the reaction was probably preventable. Although oculogyric crisis is a recognized adverse effect of antipsychotic therapy, the absence of documented routine monitoring for extrapyramidal symptoms during prolonged treatment may have contributed to delayed recognition. Early identification and closer clinical monitoring might have reduced the severity or prevented the progression of symptoms [7].

A summary of the assessment findings is presented in Table 1.

Table 1: Summary of ADR Assessment

Assessment Tool	Key Criteria Applied	Result	Interpretation
Naranjo Adverse Drug Reaction Probability Scale[5]	Temporal relationship with drug intake, improvement after withdrawal (dechallenge), absence of alternative causes, objective clinical evidence	Score: 6	Probable adverse drug reaction
Modified Hartwig and Siegel Severity Assessment Scale[6]	Drug withdrawal required, hospitalization and active medical management required, no permanent harm	Level 4	Moderate severity adverse drug reaction
Schumock and Thornton Preventability Scale[7]	Known adverse effect of drug, lack of documented monitoring for extrapyramidal symptoms	Probably preventable	Probably preventable adverse drug reaction

Discussion

Adverse drug reactions (ADRs) are defined by the World Health Organization as responses to a medication that are noxious and unintended and occur at doses normally used in humans. Oculogyric crisis (OGC) is a rare neurological condition characterized by sustained upward deviation of the eyes due to abnormal contraction of extraocular muscles. It is primarily associated with disturbances in dopaminergic neurotransmission within the basal ganglia and is commonly linked to medications that block dopamine receptors [1,8].

Although atypical antipsychotics are generally associated with a lower incidence of extrapyramidal adverse effects, cases of OGC have been reported with these agents. A study described delayed-onset OGC in a patient receiving olanzapine therapy, highlighting that second-generation antipsychotics are not completely free from neurological adverse reactions. Similarly, another study reported a case of OGC associated with risperidone treatment, further supporting the role of dopamine receptor blockade in its pathogenesis [1,2].

Another case documented the occurrence of OGC in a patient treated with olanzapine for psychiatric illness, reinforcing that such adverse reactions may occur even with medications considered relatively safer in terms of extrapyramidal side effects. In addition, emerging evidence suggests that atypical antipsychotics can produce a broader spectrum of extrapyramidal manifestations. A recent report of atypical antipsychotic-induced rabbit syndrome demonstrated that agents such as olanzapine and quetiapine may cause rare and under recognized movement disorders, particularly with prolonged use. This finding supports the concept that dopaminergic blockade, even with second-generation antipsychotics, can disrupt basal ganglia pathways sufficiently to produce atypical motor manifestations [3,9].

In the present case, the patient developed OGC after approximately six months of olanzapine therapy. The delayed onset suggests that such adverse reactions may occur even after prolonged exposure, possibly due to cumulative dopaminergic blockade or adaptive neurochemical changes. The clear temporal association and complete resolution of symptoms following drug withdrawal strongly support a causal relationship. An unusual feature observed in this case was glossoptosis, which has rarely been reported in association with antipsychotic-induced movement disorders. This may be

explained by the involvement of adjacent motor pathways due to dopaminergic imbalance, affecting orolingual musculature and resulting in abnormal tongue positioning and speech difficulty. Early recognition of drug-induced OGC is essential, as the condition may be mistaken for seizure activity or worsening psychiatric symptoms. Prompt discontinuation of the offending medication usually leads to complete resolution of symptoms, as observed in this case.

Limitations

This case report has certain limitations. As a single-patient observation, the findings cannot be generalized to a broader population. Although a clear temporal relationship and resolution of symptoms following drug withdrawal support a causal association, rechallenge was not performed due to ethical considerations, limiting definitive confirmation. Additionally, the patient was receiving concomitant psychotropic medication (escitalopram), which may act as a potential confounding factor, although its contribution to the observed reaction is less likely. Objective rating scales for extrapyramidal symptoms were not utilized, which could have provided a more quantitative assessment of symptom severity. Furthermore, serum drug levels were not measured, limiting pharmacokinetic correlation with the adverse event.

Future Directions

Further studies are required to better understand the incidence, risk factors, and mechanisms underlying atypical antipsychotic-induced dystonic reactions, particularly rare manifestations such as glossoptosis. Prospective studies evaluating dose–response relationships and individual susceptibility factors may help guide safer prescribing practices. There is also a need to incorporate routine monitoring for extrapyramidal symptoms in patients receiving long-term antipsychotic therapy, especially in vulnerable populations such as adolescents. The use of standardized assessment tools may facilitate early detection and improve clinical outcomes. Strengthening pharmacovigilance systems through increased reporting of rare adverse drug reactions is essential to enhance real-world safety data. Such efforts will contribute to improved understanding of the full spectrum of antipsychotic-related movement disorders and support the development of safer therapeutic strategies.

Conclusion

This case highlights a rare presentation of olanzapine-induced oculogyric crisis accompanied by glossoptosis. Although olanzapine is generally considered to have a lower risk of extrapyramidal adverse effects compared with first-generation antipsychotics, it may still precipitate uncommon neurological reactions. The present case emphasizes the importance of maintaining a high index of suspicion for drug-induced movement disorders, even with second-generation antipsychotics. Early recognition, prompt discontinuation of the offending drug, and appropriate management are essential to ensure complete recovery and prevent unnecessary investigations or misdiagnosis. Furthermore, this case underscores the value of pharmacovigilance in identifying rare and atypical adverse drug reactions, thereby contributing to improved drug safety and clinical awareness.

Acknowledgements

Artificial intelligence assistance was used for language refinement and manuscript preparation. The authors retain full responsibility for the content and conclusions.

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