Archives of Psychiatry and Behavioral Sciences

Volume 1, Issue 1, 2018, PP: 64-66

Olanzapine-Induced Tardive Oculogyric Crisis Associated With Paroxysmal Psychiatric Symptoms: A Diagnostic Dilemma

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Abstract

We describe a rare presentation of olanzapine-induced tardive oculogyric crisis and discuss its phenomenology as well as management. Oculogyric crisis may present with stereotypic psychiatric symptoms and mistaken by clinicians for exacerbation of psychosis.

***Conclusion:** This case highlights the need for clinicians to be vigilant for such unusual adverse effects during antipsychotic treatment and differentiate them from relapse of primary illness.*

Keywords: *Olanzapine; Tardive; Oculogyric crisis; Paroxysmal psychiatric symptoms; Adverse effects.*

Introduction

Oculogyric crisis (OGC) is a rare but severely distressing iatrogenic movement disorders caused by dopamine receptor blockers (e.g. antipsychotics, metoclopramide etc.) which can hamper normal daily functioning. OGC is characterized by spasmodic deviation of the eyes (most commonly upward and occasionally lateral, downward or oblique) frequently associated with backward and lateral flexion of the neck, mouth opening, tongue protrusion and ocular pain. Occasionally, such episodes are preceded by prodromal symptoms such as symptoms of autonomic arousal, restlessness and agitation.[1-2] Stereotypic paroxysmal psychiatric symptoms such as visual hallucinations and illusions, auditory hallucinations, delusions, catatonic phenomena, obsessive thoughts or panic attacks have been described along with OGC.[1-2] These episodes generally occur in later part of the day, last minutes (ranging from seconds to hours) with spontaneous resolution of psychiatric symptoms.[1]The delayed or 'tardive' form of OGC (occurring after weeks or months of exposure) is relatively uncommon as compared to the acute presentation. The main stay of treatment and prevention of recurrence of antipsychotic induced tardive OGC is the use of anticholinergics, reduction or withdrawal of the offending agent and use of antipsychotics with lower propensity for EPS. We are reporting rare case of a patient presenting with episodes of olanzapine-induced tardive oculogyric crisis associated with paroxysmal psychiatric symptoms.

Case Report

Mr. S, a 30 years old unmarried male presented to us with psychiatric illness of about eleven years' duration with an insidious onset and continuous course with exacerbations. The illness was characterized by fearfulness, aggression, delusion of persecution, auditory hallucinations (2nd person commanding and threatening type), recurrent suicidal attempts of high lethality and significant deterioration in socio-occupational functioning. The patient was diagnosed with Schizophrenia, paranoid subtype as per ICD-10 classification. No past or family history of any psychiatric or neurological disorders was noted and

personal history was uneventful except tobacco use in dependent pattern. Past treatment review showed that patient initially maintained well on oral olanzapine (10-20 mg/d) for around one year. Subsequently, he started complaining of recurrent episodes of involuntary up-rolling movements of the eyeballs and backward flexion of neck in full consciousness. They were preceded by sudden onset restlessness, fearfulness, irritability, autonomic symptoms and tingling sensations in limbs. The deviation of eyeballs was associated with stereotyped visual pseudo-ˇhallucinations (e.g. "shapes of humans and snakes"), commanding auditory hallucinations (e.g. "kill yourself"), unprovoked aggression and self-ˇharm attempts. These episodes usually occurred around 5-6 times a day (lasting several minutes to more than an hour), characteristically clustering towards evening and causing significant distress to the patient and family members. The episodes had persisted over several years with the same severity despite reduction of olanzapine and addition of anticholinergics, although their frequency had decreased. No other extrapyramidal adverse effects were either reported or observed during this treatment period. Due to these episodes, patient frequently became non-compliant to treatment leading to exacerbation of psychotic symptoms.

Patient was admitted and evaluated for epilepsy, dissociative disorder and other causes of movement disorders. MRI Brain revealed gliotic changes in left basal ganglia secondary to chronic lacunar infarcts while detailed neurological examination and other required investigations did not reveal any abnormality. Tardive OGC was diagnosed according to Burke's criteria and attributed to chronic exposure to olanzapine. Due to poor response of OGC to trihexyphenidyl (8 mg/d) and persisting psychopathology, olanzapine was stopped and patient was started on oral clozapine and lorazepam which were gradually optimized to 200 mg/d and 3mg/d respectively. He responded well to this regimen with significant reduction in psychotic symptoms as well as the tardive OGC episodes. The improvement has further maintained for more than a year in follow-up with infrequent mild transient episodes not interfering in functioning.

Discussion

Olanzapine has an intermediate D2 binding affinity and carries a lower risk of tardive syndromes in general, as compared to typical and some of the atypical antipsychotics. It has also been successfully used and suggested as a reasonable alternative to clozapine for treatment of tardive syndromes.[3] Olanzapine-ˇinduced tardive OGC is a very rare event with only three published case reports in world literature describing this adverse effect.[4-6] Additionally, transient psychiatric symptoms in association with the OGC episodes were observed in only one of these reports.[6] All these cases showed immediate and good response to anticholinergics not prompting any change of antipsychotic agent. However, our case responded poorly to anticholinergics and required other management approaches including use of clozapine and benzodiazepines.

Tardive syndromes are a group of motor and cognitive symptoms associated with long-term exposure to antipsychotics. Chronic use of antipsychotic is associated with up-regulation of dopaminergic receptors in the striatum and hyperactivity in the striato-pallido-thalamic pathways. The tardive movements are a result of hyperactivity in the various motor circuits of striatum. "Super-sensitivity psychosis" has been conceptualized as a tardive cognitive phenomenon due to the up-regulation of dopaminergic receptors in the cognitive circuits of striatum. OGC with associated psychotic exacerbation have been therefore considered as a motor and cognitive tardive syndrome respectively.[7] This phenomenon should be kept in mind and caution exercised while increasing doses for relapses in patients with good drug-compliance.[8] The OGC episodes in our case closely resembled the phenomenology of antipsychotic induced "paroxysmal perceptual alteration"[1-2,9]described in literature in terms of the time of day of occurrence, associated autonomic and characteristic psychiatric symptoms. These features are useful in differentiating antipsychotic-induced episodes from relapses of the underlying illness. This is clinically significant because the OGC may be brief, subtle or missed by informants and such episodes could be mistaken by clinicians for exacerbation of the psychosis. As a result, antipsychotic medication may be increased rather than reduced,

resulting in further aggravation of the condition and initiating a vicious cycle[2,9]. This case reportemphasizes the need for clinicians to be vigilant for such unusual adverse effects during olanzapine therapy and emphasizes the role of clozapine in management of both psychosis and tardive movements. The present report also highlights the presence of paroxysmal psychiatric symptoms associated with tardive OGC and discuss their probable diagnostic and treatment implications.

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Citation: **Ankit Gupta, Swarndeep Singh, Rohit Verma, Nand Kumar.** *Olanzapine-Induced Tardive Oculogyric Crisis Associated with Paroxysmal Psychiatric Symptoms: A Diagnostic Dilemma. Archives of Psychiatry and Behavioral Sciences. 2018; 1(1): 64-66.*

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