

Oculogyric Crisis - An Acute Dystonia with Olanzapine

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Abstract

Introduction: The advent of the atypical antipsychotics has led to marked decrease in the extrapyramidal adverse effects known to occur with typical antipsychotics. Among them, olanzapine has been found to very safe and effective with a receptor profile responsible for incidence of extrapyramidal side-effects at a rate comparable to that of placebo.

Case History: We report a case of oculogyric crisis in an 18 year old male diagnosed with first episode mania. This acute dystonic reaction occurred on Tab. Olanzapine (10 mg/day) in spite of an oral anticholinergic medication.

Conclusion: It is a potential side effect and changing the antipsychotic might be the only possible option in some cases.

Keywords: Olanzapine; Oculogyric crisis; Dopamine

Introduction

Oculogyric crisis (OGC) is characterized by spasmoidic deviations of the eyes, most commonly upward, occasionally lateral, downward, or oblique, lasting for a few minutes to several hours [1]. It occurs commonly after administration of typical antipsychotics. However, acute OGC has also been described with few atypical antipsychotics such as Risperidone [2], Ziprasidone [3], Aripiprazole [4], and Olanzapine in patients suffering from Post-encephalitic syndrome [5], Bipolar disorder [6], Generalized Anxiety Disorder [7] and Schizophrenia [8]. In all these studies, the oculogyric crisis has responded to oral anticholinergics. However, we report a case of first episode mania that developed this dystonic reaction despite receiving the rescue medications.

Case History

We describe a case of an 18 year old male admitted in our institute with 17 days history suggestive of mania with psychotic symptoms. He had an uneventful birth and developmental history and was admitted as an inpatient due to the severity of manic symptoms. Upon admission, patient was started on electroconvulsive therapy (ECT) and had received 8 such sessions to control his psychomotor agitation. He was continued on Tab. Haloperidol 15 mg/day along with these sessions. But, he developed extrapyramidal side effects of tremors and rigidity to Tab. Haloperidol following which it was cross tapered with Tab. Chlorpromazine to control the agitation. Tab. Trihexyphenidyl 4mg/day was added to control these side effects. The patient continued to have the side effects due to which Tab. Olanzapine 10 mg/day was introduced after stopping Tab. Chlorpromazine. Tab. Lithium Carbonate 900 mg/day was added after the ECT sessions and gradually hiked up to 1350 mg/day but his serum levels were observed to be 1.37 mmol/l. Patient was suspected of having lithium toxicity which was correlated with clinical signs of tremors, rigidity, slurring of speech. Lithium was subsequently stopped and as he continued to have florid affective symptoms, Tab. Sodium Valproate was started and hiked up to 1 g/day. The extrapyramidal symptoms had improved and patient was continued on this regime. Ten days on this regime, patient was seen to have attacks of rolling of eyeballs with inability to look straight lasting for few minutes occurring on 2-3 occasions. The dystonic reaction was immediately controlled with 50 mg of Inj. Promethazine and Tab. Olanzapine was stopped following which the OGC never recurred.

The patient was started on Tab. Clozapine due to poor response to the above regime and the fact that it never occurred again proved its association with Tab. Olanzapine. A computed tomography of brain and electroencephalography revealed no abnormality.

Discussion

Thus, we can see that Tab. Trihexyphenidyl 4mg/day was unable to prevent OGC in the patient receiving low dose of Tab. Olanzapine. Olanzapine has an intermediate D2 binding affinity and so is not expected to cause OGC [9]. The exact mechanism of acute OGC is not clear though improvement of OGC with anticholinergics has found dopamine deficiency and acetylcholine excess to be the likely reason [10]. It has been suggested that a compensatory dopamine release from presynaptic terminals in response to blockade of postsynaptic dopamine receptors and upregulation or increased sensitivity of postsynaptic receptors in response to diminished quantities of dopamine or both might be seen [11]. Patients with bipolar disorder have been found to have enhanced postsynaptic dopamine sensitivity [12]. This would explain the reason for OGC even on low doses of Olanzapine but fails to explain the reason for the ineffectiveness of Trihexyphenidyl. This phenomenon has also been documented previously and a change in the antipsychotic has been advised in such cases [8].

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