Lurasidone-induced Oculogyric Crisis

Sir,

Oculogyric crisis (OGC) is characterized by sustained dystonic, conjugate, and typically upward deviation of the eyes due to not only numerous conditions such as drug induced but also neurometabolic and neurodegenerative movement disorders or as a consequence of focal brain lesion. OGC is generally in acute onset but can take weeks to develop in some cases. It involves eye in isolation but can spread to surrounding areas. Neuroleptics are the main culprit in 60% drug-induced cases, with predominance of typical than atypical as in case of all dystonic reactions.[1] Lurasidone is a comparatively new drug which was approved by FDA to use in Bipolar Depression. It

antagonizes dopamine D_2 receptors and serotonin 5-HT_{2A} and 5-HT_7 receptors along with partial agonist at 5-HT_{1A} receptors. In addition, it antagonizes adrenergic $alpha_{2A}$ and $alpha_{2C}$ receptors with minimal affinity for histaminic (H_1) and acetylcholinergic muscarinic (M_1) receptors.[2] Here, we are presenting a case of OGC with the use of Lurasidone 160 mg/day in a patient with schizophrenia which is rare in literature.

A 32-year-old married male who approached to outpatient department with a history of persecutory delusion, referential delusion, 3rd person auditory hallucination, and poor self-care for the past 10 years. He was on 80 mg/day of Lurasidone for the past 2 weeks. Owing to partial response, the dose was gradually hiked to 160 mg/day. Following hike of dose in Lurasidone, patient complaint of being extremely frightened, anxious, and irritatble at a time with upward deviation of both eyes which lasted for only few minutes(<5 min). He was found to be perplexed during the episode and was unable to maintain daily activity for few hours thereafter. It happened twice in a day then particular measures to combat the situation with anticholinergic and clonazepam were taken. Hence, the symptoms did not reappear further. Investigations including blood count, electrolytes, computed tomography (CT) scan brain, liver, and renal function were in normal limit. Further drug was kept another 1 month with no improvement in psychotic symptoms. Hence, it was changed to alternate antipsychotic.

Lurasidone, being relatively newer atypical antipsychotic has lesser side effect of dystonia. Earlier a case of glossopharyngeal dystonia secondary to a Lurasidone-Fluoxetine combination was reported by Paul *et al*. Young patient with prior history of dystonia are more prone to develop dystonic reaction like OGC.[3] OGC can be due to hereditary, focal, or functional disruption of nigrostriatal pathway.[1] Here, in our case, there is no history in the family which suggest hereditary disorder. In addition, CT scan does not reveal any focal lesion. OCG could be a barrier in therapeutic success. It can be the cause of poor compliance. It can be cause of extreme agitation which can mimick worsening of psychosis.[1] Here, in our case, patient did not have any history of dystonia earlier, but developed with Lurasidone. Lurasidone, being the second generation antipsychotic can be prescrive above the first generation, but there should be watchful eyes not to miss impending life-threatening dystonic reaction.

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Conflicts of interest
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