

Tardive Meige's syndrome associated with olanzapine

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Abstract

Meige's syndrome is characterized by blepharospasm and oromandibular dystonia. It has been reported as a complication of typical antipsychotics. To the best of our knowledge, case of olanzapine-induced tardive Meige's syndrome has not been reported in the literature. We are reporting a case of Meige's syndrome developing after long term therapy with olanzapine.

Key Words

Meige's syndrome, olanzapine, tardive

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Introduction

Meige's syndrome was first described in 1910 by Meige as a syndrome of idiopathic blepharospasm and oromandibular dystonia.^[1] The syndrome is especially distressing because of bilateral involvement of orbicularis muscles resulting in difficulty in opening eyes. Meige's syndrome developing after long term first generation antipsychotics has been described.^[2,3] Literature search revealed only few cases of Meige's syndrome secondary to newer second generation antipsychotics.^[4-6] These have been generally reported to be acute in onset. We are reporting a case of Tardive Meige's syndrome secondary to olanzapine therapy, which to the best of our knowledge has not been reported so far in the literature.

Case Report

A 30-year-old married female was suffering from schizophrenia (DSM IV TR criteria) for the last 4 years. Her past, personal, and family histories were not significant. She was prescribed olanzapine 5 mg, which was increased to 15 mg per day after about 1 year of start of illness. She did not receive any treatment prior to olanzapine. Lorazepam was prescribed initially for a period of 2 weeks, which was later on tapered and withdrawn. She improved markedly with treatment. She presented to us

in follow-up on the same dose of olanzapine with complaints of intermittent inability to open her both eyes and abnormal movements involving the mandibular and neck regions. These started after about 3 years of regular treatment with olanzapine. The compliance to therapy was adequately ensured by the family members. She reported difficulty in speaking and eating when she would have these abnormal movements. These would also hamper her vision-dependent activities like watching television and cooking food. A diagnosis of blepharospasm and oromandibular dystonia was made. There were no other abnormal movements in any of the body parts. Results from a physical examination revealed that she had an irregularly repetitive blepharospasm together with spasms in which the jaw opened up and of the neck. Magnetic resonance imaging (brain), routine blood analysis, thyroid function test, EEG, and ceruloplasmin levels were all normal. Subsequently, a diagnosis of tardive Meige's syndrome was made. Naranjo Adverse drug reaction scale^[7] indicated a probable relationship with olanzapine, with a score of 6. Subsequently, olanzapine was stopped and patient was started on clozapine, which was increased to 100 mg per day. She reported improvement in her abnormal movements after about 3 month of therapy without any relapse of psychotic symptoms and has been maintaining well for the next 6 months of follow-up without any relapse of psychotic symptoms or movement disorder.

Discussion

To our knowledge, this is the first report describing tardive Meige's syndrome associated with olanzapine therapy. Previous report was of acute Meige's syndrome with olanzapine.^[4]

Besides idiopathic Meige's syndrome, there are various secondary causes of Meige's syndrome, which are further suggested by pharmacological studies indicating central

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dopaminergic preponderance as a possible biochemical basis for this syndrome.^[8,9] This hypothesis is further supported by observation of improvement with the dopamine-depleting agent tetrabenazine.^[10]

Olanzapine has Dopamine D2 receptor occupancy higher than that of clozapine or quetiapine and similar to that of risperidone,^[11] which may have accounted for the development of Meige's syndrome, which is generally regarded as a possible variant of tardive dyskinesia. The possibility that Meige's syndrome can be induced by long-term neuroleptic treatment suggests that a similar pathophysiological mechanism may play a role in both the drug-induced and idiopathic forms of this disorder. Dopaminergic supersensitivity may contribute to this pathophysiological mechanism.

The temporal relation between long term olanzapine administration, the appearance of characteristic dystonic reaction in the absence of choreoathetotic movements, the prompt response to olanzapine withdrawal and clozapine administration and young age at onset as compared to idiopathic Meige syndrome can easily rule out idiopathic Meige's syndrome.^[12] One can argue that the remission of Meige's syndrome was due to simple withdrawal of olanzapine, but clozapine was started because of risk of psychotic relapse and also because it has been shown to play significant role in the management of this syndrome.^[13,14] There have been recent reports of olanzapine-induced tardive dyskinesia and dystonia.^[15,16] Contrary to this, few reports also suggest the role of olanzapine for the management of these tardive movements induced by other antipsychotics.^[17,18]

This report suggests that Meige's syndrome can occur even with atypical antipsychotics as a tardive side effect.

Our report suggests that one can develop this disabling syndrome even with newer antipsychotics and close monitoring of patients and early warning to be given to the patients is warranted. More research on long term data on safety of newer antipsychotics is warranted.

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