Case Report

Amisulpride Reexposure and Tardive Dyskinesia

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ABSTRACT

To highlight the association between amisulpride and onset of tardive dyskinesia (TD) in patient suffering with psychosis not otherwise specified (NOS), who has already been treated with amisulpride for many years. A 40-year-old female suffering with psychosis NOS since 19 years presented with recurrence of positive symptoms in the form of delusion of persecution, ideas of grandiosity since last 3 months. She was treated with amisulpride up to 400 mg/day and developed involuntary oro-buccal-lingual movement within 2 months of amisulpride therapy. Amisulpride an atypical antipsychotic can lead to the development of TD in patient who already received amisulpride for many years in the past. Reexposure with amisulpride can lead to early onset of TD due to blockade of already unregulated postsynaptic supersensitive dopamine receptors.

Key words: *Amisulpride, dyskinesia, tardive*

INTRODUCTION

Tardive dyskinesia (TD), characterized by oro-buccal-lingual stereotypy, can manifest in the form of akathisia, dystonia, tics, tremor, chorea, or as a combination of different types of abnormal movements. One of the most prominent theories about TD pathogenesis is that chronic exposure to the neuroleptics results in D2 receptor upregulation with postsynaptic dopamine receptor supersensitivity. The prevalence of TD is estimated to be 20%–50% of all patients treated with neuroleptics. However, the incidence of TD is lower in patients treated with second-generation neuroleptics (risperidone, olanzapine, quetiapine, amisulpride, and ziprasidone).[1]

Access this article online	
Website: www.ijpm.info	**Quick Response Code**
DOI: 10.4103/IJPSYM.IJPSYM_140_17	

Amisulpride is an atypical antipsychotic drug with unique receptor pharmacology, which is dose-dependent. At low doses up to 300 mg/day, it enhances dopaminergic neurotransmission by preferentially blocking presynaptic dopamine D2/D3 autoreceptors. At higher doses (400–800 mg), amisulpride antagonises postsynaptic dopamine D2 and D3 receptors, preferentially in the limbic system rather than the striatum, thereby reducing dopaminergic transmission.[2]

There are reports of amisulpride-induced TD with low as well as with high dose[3,4] and improvement of TD with amisulpride.[5]

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How to cite this article: Sidana A. Amisulpride reexposure and tardive dyskinesia. Indian J Psychol Med 2018;40:91-2.

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Here, the author has reported a case of amisulpride-induced oro-buccal-lingual dyskinesia and which did not improve after dose reduction toward lower side (100 mg/day).

CASE REPORT

Ms. S 40-year-old female, diagnosed case of psychosis not otherwise specified (NOS) since last 19 years and was on tablet clozapine 200 mg/day and tablet amisulpride 200 mg/day since last 7 years. She was compliant to treatment and was maintaining well on this combination. She discontinued clozapine 1 year ago but kept on taking tablet amisulpride 200 mg/day. Around 3½ months ago, she discontinued amisulpride too and was admitted in psychiatry ward with a recurrence of a problem since last 2 weeks. Her mental status examination revealed delusion of persecution and ideas of grandiosity with absent insight. Her routine blood investigations were within normal limits. Tablet amisulpride was restarted with 50 mg/day and gradually increased up to 400 mg/day, and she was discharged after 2 weeks on tablet amisulpride 400 mg/day in stable conditions. During follow-up visits, she reported involuntary movement of lower jaw (after 2 months of restarting amisulpride). The movements of lower jaw were side-to-side, stereotypic, fast, and continuous in nature. However, these movements would disappeared during sleep and also lessen during the conversation. She was not able to eat properly due to continuous movements and also stopped attending social gathering. She was diagnosed with drug induced oro-bucco-lingual dyskinesia. The score on Abnormal Involuntary Movement Scale[6] was 16.

Tablet tetrabenazine[7] 12.5 mg BD was added and which was gradually increased to 37.5 mg/day over a period of 4 weeks without any improvement in involuntary movement. Tablet amisulpride was reduced gradually up to 100 mg/day without any significant relief in dyskinesia. Finally, amisulpride was switched over to tablet quetiapine 200 mg/day, and the dyskinesia disappeared completely within 1 week. Currently, she is maintaining well on quetiapine 400 mg/day.

DISCUSSION

This case highlights that reexposure to amisulpride can leads to the development of dyskinesia within a short period (8 weeks) in patients of psychosis NOS who had already received amisulpride for years together without any history of extrapyramidal symptoms or TD. The early onset of TD with reexposure to amisulpride explained the blocking of already upregulated and supersensitive postsynaptic dopamine receptors, and once TD has occurred, it does not improve by enhancing dopaminergic neurotransmission by preferentially blocking presynaptic dopamine D2/D3 autoreceptors as the author tried in the index case by reducing the dose of amisulpride to 100 mg/day. Hence, different doses of amisulpride neither worsen nor improve the TD as reported in the earlier studies.[3-5]

Although TD is a chronic condition, it may be reversed if the offending agent is discontinued early in the course of development of TD.[1]

In the index case, amisulpride was discontinued within 8 weeks of onset of TD, and it improved completely within 1 week of discontinuation of amisulpride.

CONCLUSION

Hence, from the current case report, one can infer that before starting amisulpride, one should be watchful for the development of TD in patients who were earlier treated with amisulpride uneventfully and TD can develop early in the course of therapy. Reducing the dose of amisulpride does not lead to improvement in TD and discontinuation of amisulpride may be helpful in complete recovery.

Financial support and sponsorship
Nil.

Conflicts of interest
There are no conflicts of interest.

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