Case Report

Risperidone induced tardive dyskinesia

Elakkya Kolanchinathan*, Dhanya Thirookaran Harichandran, Sujatha Mangattuvalappil Balakrishnan

Department of Pharmacology, Government Medical College, Thrissur, Kerala, India

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*Correspondence:
Dr. Elakkya Kolanchinathan,
Email: elakkiyakr277@gmail.com

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ABSTRACT

Risperidone is an atypical antipsychotic drug which has been less likely to produce extrapyramidal symptoms. The aim of this case report is to illustrate that low dose risperidone may cause tardive dyskinesia. A 29 year old male patient with 9 year history of paranoid schizophrenia, developed tardive dyskinesia after receiving risperidone 2 mg for 7 years. He had received small dosages of Haloperidol before the therapy of risperidone for short periods.

Keywords: Risperidone, Atypical antipsychotic, Tardive dyskinesia

INTRODUCTION

Risperidone is an atypical antipsychotic drug, a benzisoxizole derivative.1 It is a second serotonin-dopamine antagonist which was approved by FDA (1993) next to clozapine.2 It has potent and long lasting 5HT2 antagonism.3 It has similar clinical efficacy to typical antipsychotics like haloperidol, but with substantially fewer extrapyramidal side effects (EPS).3,4 But it has a high risk of tardive dyskinesia (TD) among atypical antipsychotics.5 It was found that annual incidence of risperidone induced TD is 5.3%.6 Most of the previous studies showed that risperidone produce TD only when the dose is more than 6 mg/day. Present study reports a case of risperidone induced TD in young male patient was on risperidone of 2 mg/day.

CASE REPORT

A 29 years old male patient, presented with Paranoid schizophrenia since last 9 years. Initially, he was treated with Haloperidol 5mg BD and had marked improvement but after 2 months, he developed symptoms of parkinsonism for which he was started on Trihexiphenidyl 2mg/day. There was an exacerbation of psychiatric symptoms after one year because of irregular treatment. At that time he was treated with risperidone, which was gradually increased from 1 mg/day to 2 mg/day over a period of 6 weeks. This time also the patient showed improvement and risperidone 2 mg was continued for the next six months as maintenance therapy. After seven years of lost follow-up with continuous risperidone therapy, the patient showed abnormal involuntary movements of both hands and dystonia of trunk. Abnormal involuntary movement scale (AIMS) examination was done and scored with 13 points (>3) and diagnosed as tardive dyskinesia. The patient was switched to clozapine 12.5 mg/day, gradually increasing to 25 mg/day and vitamin E also added. During next follow up, one month later there was no change in movements and clozapine was continued.

Table 1: Assessment of causality, severity and preventability of adverse drug reactions.

<table>
<thead>
<tr>
<th>Assessment</th>
<th>Inference</th>
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<tbody>
<tr>
<td>WHO-UMC system and naranjo algorithm causality assessment</td>
<td>Probable</td>
</tr>
<tr>
<td>Modified hartwig and seigel severity scale</td>
<td>Moderate</td>
</tr>
<tr>
<td>Schumock and thornton preventability scale</td>
<td>Not preventable</td>
</tr>
</tbody>
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Tardive dyskinesia (TD) is characterized by involuntary, choreiform, athetoid or rhythmic movements of the tongue, jaw, trunk and extremities. Significant features in this case are that TD was caused by risperidone while he was on low dose of 2 mg/day. Recognised risk factors for the development of tardive dyskinesia include a minimum cumulative exposure of three months to neuroleptics, increasing age, and female gender, high dosage of antipsychotics, concomitant administration of antipsychotics and antiparkinsonian drugs, early emergence of EPS and presence of affective symptoms.

At the age of 29 our patient did not have any risk factors for developing tardive dyskinesia except early emergence of EPS and long term neuroleptic therapy. Even though this patient initially received haloperidol, he was exposed to risperidone for 8 years and there was no past history of dyskinesia. Hence the temporal relationship also suggests the diagnosis of risperidone induced TD. Majority of the studies explained that at a dose of 6 mg/day risperidone may lose the balanced 5HT₂/D₂ blocking effect, resulting in more affinity for D₂ receptors, thereby producing comparable EPS with classical antipsychotics. When the same dose continues for long time, it can lead to supersensitivity of D₂ receptors in the nigrostriatal system producing tardive dyskinesia. In our study long term treatment with risperidone 2 mg/day produced TD whether it may be due to similar mechanism.

CONCLUSION

Current study report a case of tardive dyskinesia caused by risperidone in a young male patient with a brief exposure to typical antipsychotics while he was on low dose of risperidone. The tardive dyskinesia still continuing even after one month of clozapine therapy. Clinicians should be aware of the possibility of patients developing TD though they are given the low dose risperidone.

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REFERENCES


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