Risperidone -Induced Tardive Dykinesia in a Young Adult Nigerian Patient: A Case Report

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SUMMARY

Background: Tardive dyskinesia is a common problem associated with long-term use of potent antipsychotic drugs. It has become common with increased use of the newer atypical drugs. The condition is manifest by abnormal orofacial, extremity, and sometimes trunk movements. Hence, the aim of this case report is to highlight that risperisone may cause tardive dyskinesia in Nigerians.

Method: An 18-year-old black Nigerian female with a 9-month history of schizophrenia developed buccolingual masticulatory tardive dyskinesia after receiving risperidone 6 mg. She had received small dosages of typical antipsychotics before receiving risperidone.

Result: Reduction in dosage of risperidone to 2 mg resulted in improvement in tardive dyskinesia and up to the last follow-up visit; she had remained free of any abnormal involuntary movements.

Conclusion: We report a case of tardive dyskinesia in a young Nigerian woman with a brief exposure to typical antipsychotic drugs while she was on the so-called "antidyskinetic dose" of risperidone. The tardive dyskinesia resolved after stopping risperidone and remained abated after reinstitution of smaller doses of risperidone. Irrespective of the safety profile of newer atypical antipsychotic drugs, a clinician must be on the lookout for rare side effects. Prevention is the best option, as treatment may be unsatisfactory.

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***Keywords*: tardive dyskinesia, risperidone, atypical antipsychotic drugs**

INTRODUCTION

The development of atypical antipsychotic drugs represents a significant advance in the treatment of patients with schizophrenia. In comparison with conventional agents, atypical antipsychotics may offer broader efficacy and appear to be better tolerated. Risperidone, a benzisoxale derivative, is a relatively new antipsychotic that has potent and long lasting serotonin $5HT_2$ antagonism[1] and is available worldwide since the early 1990s. It has been characterized as atypical, and is believed to have similar clinical efficacy to typical antipsychotics such as haloperidol, but with substantially fewer extrapyramidal side effects[1, 2]. This property is attributed to risperidone posing a smaller risk for the development of tardive dykinesia[3] especially in doses above 6mg/day.[4] Persistent abnormal movements such as tardive dyskinesia (TD) and dystonia are not uncommon side effects associated with risperidone therapy. Increasing clinical experience suggests that it may have antidyskinetic properties in the dose range of 6-16mg/day[5] particularly in buccolinguomasticatory syndrome[6]. We report a case of tardive dyskinesia caused by risperidone in a young Nigerian woman and how we managed to control it.

CASE REPORT

A young female Nigerian, aged 18, was first seen in the psychiatric department of the Federal Medical Centre Yola in July 2006 with a 9-month history of deteriorating performance at school, preference for isolation, and withdrawal associated with low mood, and a three-month history of third person type auditory hallucinations and paranoid ideation. She had no suicidal thoughts or a family history of psychiatric illness. A diagnosis of paranoid schizophrenia was made and she was started on thioridazine 25 mg three times daily and diazepam 10 mg as required. She responded partially to this regimen over the next six weeks. The dose of thioridazine could not be increased because of excessive sedation and anticholinergic side effects.

Consequently, chlorpromazine 50 mg three times daily was tried briefly but was stopped because of sedation. Finally, thioridazine and chlorpromazine were discontinued after six weeks, risperidone tablet was introduced, and its dose was slowly titrated up to 6 mg daily over the next three months. There was a significant improvement in auditory hallucination, paranoid ideation and social withdrawal. Five months later, she started developing involuntary buccolingual masticatory movements, jerky non-repetitive movements involving both arms and fingers. A neurological opinion was sought and a diagnosis of drug-induced tardive dyskinesia was made. She was on no other medication, no history of exposure to conventional antipsychotics at any time and a thorough neurological examination did not reveal any other abnormality. Her biochemical investigations were within normal limits. The involuntary movements progressively worsened and some

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residual psychotic symptoms persisted. The psychiatrist managing her added trifluoperazine 10 mg which was discontinued by the relatives after 2 months because of incapacitating involuntary movement and persistence of auditory hallucination. Finally, she was readmitted and started on deep intramuscular injection of flupentixol decanoate 20 mg every 2 weeks. After she had received two fortnightly doses of flupentixol, there was exacerbation of auditory hallucination, delusion of reference, persecutory delusions, low mood, and suicidal thoughts. The involuntary movements had worsened involving arms and legs, flickering movements of fingers, and writhing movement of trunk and neck. Risperidone was discontinued, leading to a significant improvement in the abnormal movements seen over the next two weeks, and these were barely noticeable within a few days. Psychotic symptoms also decreased remarkably over the next one week and she restarted on risperidone 2 mg daily for 4 months and was lost to follow-up. During this period, she was free of psychotic symptoms and tardive dyskinesia.

DISCUSSION

Tardive dyskinesia (TD) is a serious motor side effect of chronic neuroleptic therapy. It is a complex hyperkinetic syndrome consisting of choreiform, athetoid or rhythmic abnormal involuntary movements. This young Nigerian woman developed TD while receiving risperidone. Significant features in this case are that TD was caused by so-called "antidyskinetic dose" of risperidone and TD resolved completely after a retrial with smaller doses of risperidone. This patient developed TD after receiving antipsychotic drugs for 3 months and while she was still on risperidone. Recognised risk factors for the development of TD include increasing age, female gender, a minimum cumulative exposure of three months to neuroleptics,[7] high dosages of antipsychotic drugs, concomitant administration of antipsychotic drugs and antiparkinsonian drugs,[8] and presence of affective disorders[8]. For such high-risk patients newer antipsychotic drugs such as risperidone are preferred. At the age of 18, our patient did not have any risk factors for developing TD except female gender. Even though she had received conventional antipsychotic drugs initially, she was exposed to risperidone for five months as opposed to six weeks on thioridazine and a few days on chlorpromazine before developing TD. In other words, her exposure to conventional antipsychotic drugs was not long enough to be attributed as the cause of TD. Furthermore, there was a gap of five months between discontinuation of other antipsychotic drugs and the onset of TD. We therefore implicate risperidone as the cause of TD in this young Nigerian woman. There have been a few reported cases of TD caused by risperidone[9]. It is interesting to note that almost all who developed TD with risperidone were females under the age of 50 years, as in this young Nigerian woman. It would be farfetched to draw any conclusions from these isolated case reports but the trend of risperidone targeting females for TD is worth further investigation because conventional antipsychotic drugs are likely to cause TD in elderly women[7]. The symptoms of TD subsided with discontinuation of risperidone, and our patient was symptom-free on a much smaller dose of risperidone. Higher rates of TD in blacks and lower rates in Chinese and other Asian groups have been reported,[10] but the basis for this ethnic disparity is unclear. Decades after it was first identified, there are still gaps in our understanding of the pathophysiology and aetiology of TD. Of the various mechanisms explored, dopamine receptor hypersensitivity, gamma-amino butyric acid (GABA) insufficiency, and free radical formation from catecholamine metabolism are the three most widely accepted. Because of the relatively poor understanding of the pathophysiology, no effective treatment for TD is currently available. The best approach to management remains prevention, including restricting the use of antipsychotic medications to established indications and alternative treatments when possible. When neuroleptic medications are appropriate, they should be prescribed in the smallest therapeutic doses for the shortest possible time. Atypical antipsychotic drugs should be considered as first-line therapy, and patients taking such medications should be re-evaluated every 3 to 4 months to determine their ongoing need for such medications and identify early features of TD.

REFERENCES

1. Van Kammen D. P., Marder S. R. Dopamine receptor antagonists. In: Kaplan HI, Saddock B, eds. Comprehensive textbook of psychiatry. Baltimore: Williams and Wilkins 1995; 1987–2022.
2. Song F. Risperidone in the treatment of schizophrenia: a meta-analysis of randomized controlled trials. *J Psychopharmacol* 1997; **11:** 65–71.
3. Owens D. G. Extrapyramidal side effects and tolerability of risperidone: a review. *J Clin Psychiatry* 1994; **55:** 29–35.
4. Marder S. R., Meibach R. C. Risperidone in the treatment of schizophrenia. *Am J Psychiatry* 1994; 825–835.
5. Rangwani S. R., Gupta S., Burke W. J. *et al*. Improvement of debilitating tardive dyskinesia with risperidone. *Ann Clin Psychiatry* 1996; **8:** 27–29.
6. Chouinard G. Effects of risperidone in tardive dyskinesia: an analysis of Canadian multicentre risperidone study. *J Clin Psychopharmacol* 1995; **15:** 36–44.
7. Grebb J. A. Medication induced movement disorders. In Kaplan HI, Saddock B, eds. Comprehensive textbook of psychiatry. Baltimore. Williams and Wilkins 1995; 1909–1915.
8. Van Os J., Fahy T., Jones P. *et al*. Tardive dyskinesia: who is at risk? *Acta Psychiatr Scand* 1997; **96:** 206–216.
9. Edleman R. J. Tardive dyskinesia from risperidone? *J Am Acad Child Adolesc Psychiatry* 1997; **36:** 867–868.
10. Pandurangi A. K., Aderibigbe Y. A. Tardive dyskinesia in non-western countries: a review. *Eur Arch Psychiatry Clin Neurosci* 1995; **246:** 47–52.