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Case Report

Amisulpiride induced tardive dyskinesia: a case report

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ABSTRACT

Amisulpride is a bezamide group of antipsychotic, and like other antipsychotics, it acts by reducing signalling via the dopamine D2 receptor. It is associated with a high risk, of developing increased blood levels of the lactation hormone, prolactin and low risk, as compared with typical antipsychotics, of causing movement disorders. Tardive dyskinesia is a type of movement disorder, which is more common with typical antipsychotics but development of tardive dyskinesia is not rare with the use of atypical antipsychotics. Newer molecules are being developed to reduce the incidence of various dyskinesias, but side effects are evident even with relatively newer molecules. Amisupride is also classed with newer generation of atypical antipsychotic, used to treat schizophrenia and dysthymia. We are reporting a case of middle aged female patient suffering from schizophrenia who developed tardive dyskinesia with the use of amisulpride.

Keywords: Amisulpiride, Tardive dyskinesia

INTRODUCTION

Tardive dyskinesia (TD) is one of the commonest adverse effect associated with the long-term use of neuroleptics, characterized by involuntary movements of the tongue, jaw and / or extremities.[1] The Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-V) classifies TD as a medication-induced movement disorder i.e. involuntary athetoid or choreiform movements (lasting at least a few weeks) generally of the tongue, lower face and jaw, and extremities that can develop after short-term and long-term use of medications, as well as after discontinuation of, change in, or reduction in medications.[2] TD is a form of dyskinesia which can occur after a prolonged exposure to anti psychotic medications and other variety of drugs like anticholinergics, antidepressants, antiemetics, anticonvulsants, anti-histaminics, decongestants, antiparkinsonian drugs etc. TD prevalence is estimated to be 20-50% of all patients treated with neuroleptics, but it varies among different age groups and published studies, with prevalence increasing with advanced age.[3] Prevalence of Schizophrenia is estimated to be 1% of the total population and neuroleptics play major role in the treatment of schizophrenia. Estimated prevalence rate of TD in patients receiving neuroleptics range from 0.5-70%, with an average prevalence rate of 24%.[4]

The incidence of neuroleptic-induced TD is lower among younger individuals (3-5% per year) and higher in relatively elderly patients, particularly women, reaching incidence rates as high as 30% after 1 year of cumulative exposure to neuroleptics. Total annual incidence rate ranging from 0.8% in patients younger than 50 years to 5.3% in those older than 50 years.5 Other risk factors for development of TD are female gender, previous brain injury or dementia, early extrapyramidal symptoms and African and African American race.[6] The pathophysiology of TD remains poorly understood, and several hypotheses have been proposed that include

dopamine receptor super sensitivity, gamma-aminobutyric acid (GABA) depletion, cholinergic deficiency, oxidative stress, altered synaptic plasticity, neurotoxicity, and defective neuroadaptive signalling.[4] The first generation "typical" neuroleptics with high dopamine D2 receptor occupancy have been reported to have a higher risk of causing TD than the second- or third-generation medications, often referred to as "atypical" antipsychotics, with low D2 receptor occupancy, such as clozapine and quetiapine. It is now well recognized, however, that even atypical antipsychotics can cause TD.[7]

Prevention and early identification is the best combat for TD. Physician should prescribe the lowest possible dose of a medication least associated with TD. Treatment of TD is very difficult and there are no approved medications. However, Clozapine is the best available drug to treat TD. Other drugs Quetiapine, Olanzapine can also be used. Authors are here reporting a case of amisulpride induced TD which was successfully treated with Clozapine.

CASE REPORT

Mrs. X middle aged female had a history of fearfulness and suspiciousness, irritability, decreased interactions, not working efficiently with disturbed sleep and appetite for last 12 years and was diagnosed as a case of schizophrenia according to ICD-10 criteria. She had been on regular treatment for the past 10 years and for the last one year she was maintained on amisulpiride 200mg per day initially, which was increased to 400 mg per day along with loraepam 2mg at bed time to take care of her disturbed sleep pattern. Now on follow-up she presented with involuntary stereotypic movements in the oro-bucco-lingual region (chewing and tongue movements inside the mouth) suggestive of tardive dyskinesia. Also, patient had developed secondary amenorrhoea since last 4 months. Her blood investigations were within normal limits including her thyroid profile. Also, NCCT Scan Head did not show any abnormality. To manage her symptoms, she was admitted and was started on trihexyphenidyl 2mg TDS and atypical antipsychotic amisulpiride was replaced by aripiprazole 10mg along with lorazepam 2mg at bed time. She started having regular menstrual cycles on above treatment and secondary amenorrhoea was resolved after stopping amisulpiride, over a few days. Gradually her chewing movements reduced but she developed restlessness and tendancy to move off and on suggestive of akathesia by aripiprazole. So aripipraole was replaced by clozapine starting with 25mg at bed time. She was also given propranolol 40mg per day till her akathesia symptoms were resolved. Clozapine dose was gradually increased to 50mg HS. Her chewing and tongue movements were also reduced to a great extent over a period of one month with well controlled psychotic symptoms. Her symptoms of TD were completely resolved after one year of treatment.

DISCUSSION

Typical antipsychotics are commonly associated with drug induced movement disorders, so there is increased usage of atypical antipsychotics, which are known to have favourable side effect profile including drug induced movement disorder. However newer generation of atypical antipsychotics do produce unwanted side effects like in present case report. The patient developed involuntary movements in the oro-bucco-lingual region for 4 weeks and amenorrhoea for last 4 months, which were reversed on withdrawal of amisulpride. Thus, occurrence of TD symptoms when the patient was on amisulpride and the prompt resolution of TD as well as secondary amenorrhoea after discontinuing amisulpride strongly suggests a case of amisulpride-induced tardive dyskinesia as well as amisulpiride induced secondary amenorrhoea.

Further there was development of akathesia, when the patient was shifted to aripiprazole suggesting aripiprazole induced akathesia. Development of aripiprazole induced akathesia also showed patient's vulnerability to develop adverse side effects of antipsychotics. So, the patient was shifted to Clozapine which is best available drug to treat TD and showed well controlled symptoms. In this case risk factors for development of TD were middle age, female gender, long duration of illness, previous exposure to typical antipsychotics, switching over to another antipsychotic and vulnerability to side effects were present. The literature is sparse, with only two case reports of tardive dyskinesia associated with amisulpride.[8,9] There are also very few case reports of amisulpiride induced tardive dyskinesia as well as secondary amenorrhoea as in this case.

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REFERENCES

1. Daniel DG, Egan MF, Wolf SS. Neuropsychiatric aspects of movement disorders. In: Comprehensive Textbook of Psychiatry. 7th Ed, Lippincott Williams and Wilkins, Philadelphia, USA. 2000.
2. American Psychiatric Association. diagnostic and statistical manual of mental disorders, 5th Ed, Arlington, VA: American Psychiatric Association. 2013.
3. Washington, DC. American Psychiatric Association. American Psychiatric Association. Diagnostic and statistical manual of mental disorders, 4th Ed, Text Revision (DSM-IV-TR). 2000:803-805.
4. Kulkarni SK, Naidu PS. Pathophysiology and drug therapy of tardive dyskinesia: current concepts and future perspectives. Drugs Today (Barc). 2003 Jan 1;39(1):19-49.
5. Correll CU, Leucht S, Kane JM. Lower risk for tardive dyskinesia associated with second-

generation antipsychotics: a systematic review of 1-year studies. Am J Psychiatry. 2004 Mar 1;161(3):414-25.
6. Tenback DE, Bakker PR, van Harten PN. Risk factors for tardive movement disorders in schizophrenia. Tijdschr Psychiatr. 2015;57(2):120-4.
7. Peña MS, Yaltho TC, Jankovic J. Tardive dyskinesia and other movement disorders secondary to aripiprazole. Mov Disord. 2011 Jan;26(1):147-52.
8. Masdrakis VG, Papadimitriou GN, Papageorgiou C, Kouzoupis A, Giailoglou D, Soldatos CR. Development of tardive dyskinesia in a patient taking amisulpride. Prog Neuropsychopharmacol Biolog Psychiatr. 2007;2(31):586-7.
9. Fountoulakis KN, Panagiotidis P, Siamouli M, Kantartzis S, Mavridis T, Iacovides A, et al. Amisulpride-induced tardive dyskinesia. Schizophrenia Res. 2006 Dec 1;88(1-3):232-4.

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