# Case Report

# Severe Tardive Dystonia on Low Dose Short Duration Exposure to Atypical Antipsychotics: Factors Explored

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ABSTRACT

Tardive dystonia (TD) is a serious side effect of antipsychotic medications, more with typical antipsychotics, that is potentially irreversible in affected patients. Studies show that newer atypical antipsychotics have a lower risk of TD. As a result, many clinicians may have developed a false sense of security when prescribing these medications. We report a case of 20-year-old male with hyperthymic temperament and borderline intellectual functioning, who developed severe TD after low dose short duration exposure to atypical antipsychotic risperidone and then olanzapine. The goal of this paper is to alert the reader to be judicious and cautious before using casual low dose second generation antipsychotics in patient with no core psychotic features, hyperthymic temperament, or borderline intellectual functioning suggestive of organic brain damage, who are more prone to develop adverse effects such as TD and monitor the onset of TD in patients taking atypical antipsychotics.

**Key words:** *Antipsychotic agents, borderline intellectual functioning, tardive dyskinesia*

## INTRODUCTION

Tardive dystonia (TD), a rarer side effect after longer exposure to antipsychotics, is characterized by local or general, sustained, involuntary contraction of a muscle or muscle group, with twisting movements, generally slow, which may affect the limbs, trunk, neck, or face.[1] TD has been shown to develop in about 3% of patients who have had long-term exposure to antipsychotics.[2] If untreated, it may lead to permanent debilitation. Chronic dopamine receptor antagonism is strongly associated with TD symptoms.[3] The low risk of TD for atypical antipsychotics is thought to result from their weak affinity for dopamine receptors.[3]

Compared with typical, atypical antipsychotic agents have a greater affinity for serotonin 5-$H_{T2A}$ than dopamine $D_2$ receptors, with a low propensity to induce TD. Among this olanzapine is thought

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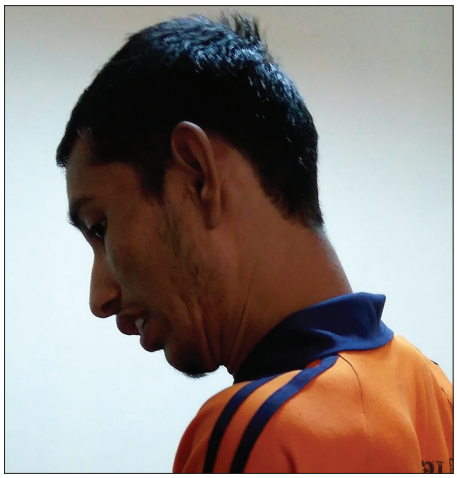

**Figure 1:** Picture of neck dystonia of patient

to have preferential action at mesolimbic over nigrostriatal dopaminergic pathways and is, therefore, associated with a very low incidence of Extrapyramidal symptom (EPS).[4] Furthermore, a retrospective analysis of controlled multicentric trials suggested that olanzapine also improves preexisting symptoms of tardive movements.[4]

## CASE REPORT

We report a case of 20-year-old male, belonging to lower socioeconomic class, educated up to 2nd standard, presented with severe unilateral dystonic left sided neck movements [Figure 1]. Careful history exploration revealed he was taking risperidone 2 mg irregularly for 2 months and then olanzapine 5 mg for another 4 months.

At 19 years, the patient presented with occasional anger outbursts, getting provoked on small matters and beating family members, running away from home, screaming episodes occasionally, fearfulness, sleep disturbance for 2 days; which was precipitated after fever. According to the mother, one friend might have threatened/made fun of him actually and after that patient stopped going out of house, and displayed above mentioned symptoms. This was interpreted as psychosis with persecutory ideas, and he was treated with risperidone 2 mg/day for 2 months and then with olanzapine 5 mg/day for 4 months.

In last two follow-ups patient did not present himself, and mother reported unusual neck movements, which were taken as a part of his overall psychopathology and not taken seriously, slight intermittent neck movements reported were missed as part of adolescent behavior problems mimicking some hero in movies.

As neck dystonia increased, the patient had a severe disability as patient had to keep his hands behind his head for the support. The movement would decrease when the patient was lying down and was absent during sleep. He stopped going outside or doing household work. Sitting comfortably or walking straight became impossible in waking hours. He even stopped taking food due to severe neck movements making chewing and swallowing difficult. His Abnormal Involuntary Movement Scale scoring was in severe rating.

His birth and early developmental milestones were normal. During 2–10 years of age patient was inattentive and mildly hyperactive. Other siblings were educated with Master's degree, and patient was also sent to school, but due to inattention and restlessness, he did not pass 2nd standard after three attempts. He left the schooling. With average executive functioning and life skills, he worked as an unskilled laborer in the neighborhood shops as a helping hand. Nobody suspected him of having an intellectual disability. Between 11 and 18 years, he was found to be getting over familiar, cheerful, moody, and short tempered. Sometimes, the patient had inappropriate social judgment; for which his friends made fun of him, and teased him.

On mental status assessment, patient was found to be having borderline intellectual functioning with standard intelligence quotient test. Routine investigations, thyroid function tests, electroencephalogram, fundus examination, cervical X-ray, magnetic resonance imaging brain were normal. After consulting neurophysician, Wilson's disease and other secondary causes of dystonia were ruled out.

The patient was treated with clonazepam 1 mg total dissolved solid (TDS), tetrabenazine 25 mg TDS, trihexiphenidyl 2 mg bipolar disorder (BD). After 2 months, there was some improvement of around 30%. Baclofen 10 mg was added; increased up to 20 mg, trihexyphenidyl reduced to 2 mg. With little improvement after 4 months of treatment for dystonia, levodopa + carbidopa (100 + 25) was added by neurophysician and increased up to ½ tablet TDS and baclofen omitted.

After 12 months of treatment, patient has improved around 90% with tetrabenazine 75 mg, levodopa + carbidopa (100 + 25) - ½ tablet BD, and clonazepam 1 mg BD. He has shown no psychotic or behavioral symptoms and is not on any antipsychotics.

## DISCUSSION

Earlier case reports reported TD developing with high-dose atypical antipsychotics such as olanzapine 20 mg or aripiprazole 15 mg with longer duration

of exposure of around 12–15 months in established psychiatric illness like schizophrenia or any other psychotic illness.[5,6] EPS in general and tardive dyskinesia, in particular, have been extensively studied in schizophrenia. Even though a number of studies suggest that bipolar patients experience higher rates of EPS (parkinsonism, dystonia, akathisia) and TD compared to patients with a diagnosis of schizophrenia,[7,8] research within the BD population has been limited.

The risk is found to be 3 to 5 times higher in elderly patients compared to young patients. In addition to age, the risk is directly proportional to: Female gender, daily and total dose of the antipsychotic drug, presence of mood disorder, the use of anticholinergics with neuroleptics, previous physical therapies (electroconvulsive therapy), the presence of other physical illness such as diabetes or an organic disorder, younger age of exposure, and the presence of extrapyramidal symptoms early in treatment.[9]

This patient's severe dystonic neck movements developed within short period of 6 months of exposure to atypical antipsychotics risperidone 2 mg and then olanzapine 5 mg only, which can cause minimal extrapyramidal side effects. In this case, risk factors for developing serious disabling TD were neuroleptic exposure, borderline intellectual functioning, externalizing behavior, probable misdiagnosis, and overlooking early indicators of side effects.

This case highlights dangers of casually prescribing low dose second generation antipsychotics in patient with hyperthymic temperament and Borderline intellectual functioning with vague short lasting presenting complaints; probably misdiagnosed as psychosis; leading to such severe adverse effects because patients with organic brain damage are more prone to develop adverse effects like TD. Thus, judicious use of antipsychotics, with detailed and frequent assessments is important, and emergent stereotyped behavior or unexplained movements must be examined carefully and taken seriously.

### Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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### Conflicts of interest

There are no conflicts of interest.

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