

Case Report

Tardive Dystonia with Olanzapine: A Rare Case Report

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ABSTRACT

Olanzapine is an atypical antipsychotic drug which is available in oral and injectable forms that is used for treatment of various psychiatric disorders. We report a rare case of tardive dystonia after receiving single dose of olanzapine (10 mg) in parental form. Clinicians should be very vigilant regarding this rare side effect with use of olanzapine in clinical practice.

Key words: Alcohol dependence syndrome, olanzapine, tardive dystonia


INTRODUCTION

Olanzapine is serotonin dopamine antagonist which controls positive symptoms in schizophrenia by suppressing dopamine in mesolimbic region with less deterioration of negative symptoms and extrapyramidal syndrome. It is reported to improve the symptoms of tardive dyskinesia.^[1] Even low dose of olanzapine can cause dystonic reaction. Dystonia is sustained muscle contractions frequently causing twisting and repetitive movements or abnormal postures. The disorder may be hereditary or due to secondary factors like physical trauma, infection, poisoning (e.g., lead poisoning) or reaction to neuroleptics (tardive dystonia). Cervical dystonia (spasmodic torticollis) causes the head to rotate to one side or toward chest, or back, or a combination of this posture.^[2] Tardive dystonia has an earlier mean onset age than that of many other

dystonic disorders. Men are more likely than women to develop the condition at a younger age. Young patients generally first exhibit tardive dystonia symptoms in the lower limbs, while older patients usually experience their first symptoms in the facial region.^[3] However many studies have analyzed prevalence rates and risk factors for tardive dyskinesia. The prevalence of tardive dystonia is 0.5 to 21.6% in patients who are treated with neuroleptics.^[4] We have a patient of tardive dystonia who presented in our clinic.

CASE REPORT

A male patient aged 27 years presented in our psychiatry clinic with history of alcohol intake from last 6 years. Patient started with 90 ml occasionally and gradually reached up to 500-750 ml daily to get the same desired effect. He tried many times to control his drinking pattern but failed because of severe withdrawal symptoms. History of marital disharmony and frequent fights at work was present but never had any medical or legal complications. There was no history of any other psychiatry disorders. On MSE, patient was conscious, cooperative, oriented, moderately built and nourished, eye contact and rapport established. He was having euthymic mood, normal thought content and intact higher mental functions. Insight was grade V.

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Patient was thoroughly assessed, diagnosed as F 10 (ICD-10) and admitted in psychiatry ward for management. All routine and special investigations were within normal range. He was put on intravenous line and was maintained on inj. Lorazepam 4mg three times a day, inj. Thiamine 100 mg BID. On third day evening, patient turned irritable with fluctuating consciousness. Patient was diagnosed as delirium tremens and injection olanzapine 10mg I/M were given. After 4 hours patient became restless and had spasmodic contraction of neck and head turned toward left side (acute cervical dystonia) with the repetitive sustained contractions of the orbicularis oculi (tardive blepharospasm). He was managed with promethazine 25mg and trihexyphenidyl 6mg. Delirium improved next day but dystonia continued. CT scan head was within normal range. Treatment for dystonia continued with lorazepam 2mg and trihexyphenidyl 6mg/day but no improvement was seen. Then neurological opinion was taken and patient was put on baclofen 20mg/day and dose was raised to 40mg/day over a period of 4 weeks. Spasmodic contraction of left side of neck continued. Patient was discharged after 30 days of admission on baclofen 40mg, Lorazepam 2mg, trihexyphenidyl 6mg and tetrabenazine 75mg/day. Patient regularly came to our hospital but dystonia continued. Patient showed 50 to 60% improvement in his condition subjectively and objectively after 2 years of regular treatment. Finally he was diagnosed as tardive dyskinesia.

DISCUSSION

Olanzapine is used to relief symptoms in schizophrenia, dysfunctional mood condition in bipolar to control agitation and extreme excitement, too aggression and agitation in substance use disorder. For handling emergency situations parental use of olanzapine (intramuscular) is preferred which is found to be more effective.^[5] It has lesser propensity for dystonia and extrapyramidal adverse effects than typical antipsychotics.^[6] Rapid-acting intramuscular formulation of olanzapine has a more rapid rate of absorption, as shown by higher maximal concentration (C_{max}) (two- to five folds) and an earlier time to C_{max} (30minutes vs 4 hours) than oral formulation.^[7] Tardive dystonia often develops at higher rates in patients receiving neuroleptic agents for long period than one treated for shorter period of time. There are no definitively determined factors why some patients develop this condition while others do not.^[3] Olanzapine appears to have a higher D2-receptor occupancy at therapeutic doses than other Serotonin and Dopamine Antipsychotics.^[8] Although there are reports of dystonia with oral olanzapine, the present report shows that dystonic reactions can also occur with intramuscular olanzapine in young adults. In a survey of 555 psychiatric patients, Yassa *et al* found

a prevalence rate of 34% for oral tardive dyskinesia and only 1.4% for tardive dystonia.^[9] Similarly, Friedman *et al.* found a prevalence rate of only 1.5% among 352 hospitalized psychiatric patients.^[10] One recent study by Sethi *et al* indicated a prevalence rate of 21% for tardive dystonia among veterans institutionalized long term. However, most of these cases were mild; only 20% were symptomatic.^[11]

Pathophysiology of acute dystonic reactions involves increased dopamine synthesis and release, especially as neuroleptic levels in the blood and brain decline by use of SDAs.^[12] This could have accounted for development of dystonia (torticollis type) in the patient reported as above. Tardive dystonia has an earlier mean onset age than that of many other dystonic disorders. Young patients generally first exhibit tardive dystonia symptoms in the lower limbs, while older patients usually experience their first symptoms in the facial region. Men are more likely than women to develop the condition at a younger age.^[3]

Young age with faster drug elimination, male sex and presence of delirium in substance dependence syndrome (alcohol) might be possible risk factors in our case experiencing dystonic reactions early in the course of treatment. Although the exact mechanism by which these medications cause tardive dystonia is not clearly understood, it is believed that the disorder may develop when dopamine receptor blockers cause neurons to become hypersensitive to chemicals released by the drugs. Some research suggests that the medications may act as triggers in patients already predisposed to dystonia.

Clinicians should consider the possibility of the development of tardive dystonia in susceptible individuals even at low doses insufficient to obtain a true antipsychotic action. Further reports and well-designed trials are necessary considering the potential use of intramuscular olanzapine in controlling agitation and delirious state in case of alcohol dependent subjects. This calls for a very cautious approach for its use in young age group as motor side effects are most visible and distressing side effects of psychotropic drugs. Until further data are available, careful assessments are warranted for movement disorders in patients with substance dependence disorder who are receiving olanzapine.

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