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Case Report Open Access

A case report of Olanzapine induced Tardive Dystonia presenting along with Catatonia

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Abstract

Tardive dystonia (TD) is a movement disorder dominated by involuntary muscle contractions associated with prolonged exposure to neuroleptics. We are reporting a unique case of Olanzapine induced TD which presented along with catatonia. Both the disorders showed significant improvement with Clozapine. We recommend use of Clozapine for the management of cases of TD with catatonia.

Introduction

Tardive dystonia (TD) is a rare syndrome associated with prolonged exposure to neuroleptics [1]. In comparison to typical antipsychotics, atypical antipsychotics have lower risk for development of TD [2]. There are only a few published case reports of Olanzapine induced TD till now [3]. TD shares some common etiological factors with catatonia including organic dysfunction and dopamine imbalance [2,4,5]. The co-occurrence of TD and catatonia is a clinical challenge both in the areas of diagnosis and management. In this context, we are reporting a case of Olanzapine induced TD which presented along with catatonia and showed improvement in both catatonia and TD with Clozapine.

Case Summary

Patient, Mr. S, 24 year old man with negative family history for any psychiatric or neurological illness presented with complaints of hearing voices which others could not perceive, fearfulness, poor socialization and aggression towards family members for 3 years. Patient was diagnosed as a case of paranoid schizophrenia and treated as inpatient and showed significant improvement in his symptoms on treatment with Haloperidol 15 mg and Trihexyphenidyl (THP) 2 mg. After one month of hospitalization, patient was discharged with complete resolution of psychotic symptoms. After discharge, the patient stopped taking medications because of development of Extrapyramidal symptoms (EPS) leading to slowness in daily activities. Within 2 months of stopping treatment, patient's symptoms reappeared. This time, he was treated with Olanzapine 15 mg with complete resolution of psychotic symptoms. After 9 months of maintaining well on Olanzapine, patient developed repeated episodes of forceful deviation of neck towards right side persisting for 10-15 minutes. This deviation was not associated with altered sensorium, pain and never happened during sleep. Patient tried to push his head back to normal position using his hands. In the beginning, the frequency of episodes was 1-2 per week, but over time the frequency increased and later deviation persisted throughout day. Along with this, patient also developed decreased speech output, poor socialization and gradual decrease in his food intake. Within one month of onset of these symptoms patient was brought to hospital. Patient was diagnosed to be suffering from dystonia. Olanzapine was discontinued and Aripiprazole was started considering the fact that it has less potential to cause EPS. The patient also received Injection Promethazine (25 mg) immediately and 50 mg daily for 3 days. But there was no improvement in dystonia. Patient's food intake, socialization and speech output worsened further and he also developed smiling and muttering to self and impulsive aggression. After 2 wks, patient was admitted. At the time of admission, physical examination revealed pallor and torticollis, right side. Patient also had signs of catatonia, with Bush Francis Catatonia Rating Scale [6] (BFCRS) score 13 and Global Dystonia Scale [7] (GDS) score 10. Investigations revealed: Hemoglobin 6.8%, white blood cell count, renal function tests, liver function tests, thyroid levels and Creatinine Phosphokinase-MB (CPK MB) were within normal range. Computerized Tomography (CT) scan, Electroencephalography (EEG) and Video EEG reports were also within normal limits. Patient was treated by injection Lorazepam up to 8 mg for two days, but there was no improvement in catatonia or dystonia. Because of the compromised food intake and impulsive aggression, Electroconvulsive therapy (ECT) was started and patient received 5 sessions of bitemporal ECT. His aggression and food intake improved. But his mutism persisted, self care deteriorated and he looked confused with BFCRS score of 9 and the dystonia persisted with GDS score 10. After this, patient was treated with Clozapine which was hiked very slowly to 150 mg. Patient showed improvement in catatonia and dystonia with BFCRS score of 0 and GDS score 5. After resolution of catatonia, THP 2 mg was started which was gradually hiked to 6 mg. Patient was discharged with Cloazapine 150 mg and THP 6 mg. After 6 months, patient is currently receiving Clozapine 100 mg and THP 8 mg with complete resolution of psychotic symptoms and with minimal disturbance with dystonia with GDS score of 1.

Discussion

This is a case of Olanzapine induced TD. Operational Criteria for the Diagnosis of Tardive Dystonia were given by Burke et al in 1982 [1]. All five criteria are required for a definite diagnosis of TD. Our patient met all diagnostic criteria for TD including presence of chronic dystonia; absence of other involuntary movements; development during treatment with D2 receptor blockers; negative family history and ruling out of other causes of dystonia. Olanzapine induced TD is a rare presentation. But the uniqueness of this case was co-occurrence of

TD and catatonia. As per best of our knowledge, there is no reported case in existing literature of TD presenting along with catatonia. This unique clinical presentation raises many clinical and theoretical questions.

No.	Drug	Dosage	Duration	Compliance	Response	Side effects	Comments
1	T. Haloperidol	10 mg	06/06/2013-18/06/2013	Good	Improvement in psychotic symptoms	Patient developed extrapyramidal symptoms	
		15 mg	19/06/2013- 21/08/2013				
2	T. Olanzapine	10 mg	05/10/2013- 06/11/2013	Good	Improvement in psychotic symptoms	Developed dystonia in August 2014	Patient also developed catatonia at the same time
		15 mg	07/11/2013- 10/08/14				
3	T. Aripiprazole	10 mg	10/08/2014-26/08/2014	Good	No improvement in catatonia or dystonia	None	
4	T. Trihexyphenidyl	2 mg	10/08/14-22/08/14	Good	No improvement	None	
4	Inj. Promethazine	50mg	22/08/14-24/08/14	Good	No improvement	None	
5	Inj. Lorazepam	8 mg	23/08/14-25/08/14	Good	No improvement	None	
6	T. Clozapine	12.5- 150 mg	Gradual hiking of dose from 05/09/2014 to 21/10/14	Good	Improvement in both catatonia and dystonia	Sedation, hypersalivation	
		125 mg	21/10/14-20/11/14	Good	Complete resolution of psychosis and minimal impairment due to dystonia	Sedation, hypersalivation	
		100 mg	20/11/14-30/03/14			None	

Table 1: Showing treatment details of this case over a period of time

The clinical differentiation of catatonic posturing and dystonia is also important. This kind of presentation also necessitates exclusion of certain life threatening conditions including neuroleptic malignant syndrome (NMS) and epileptic seizure. NMS can be excluded by exclusion of other signs and symptoms e.g. hyperthermia, autonomic disturbances, generalized rigidity and investigations showing normal CPK MB levels and white blood cell counts. To exclude the diagnosis of epileptic seizure thorough neurological examination and investigations including EEG and video EEG are needed. Neuroimaging is mandatory in these cases to exclude presence of any organic lesion.

The co-occurrence of TD and catatonia can be explained on the basis of certain common etiological factors. Both catatonia and TD are known to be associated with organic dysfunction [4,5]. The imbalance of dopamine at D1 and D2 receptors in basal ganglia has been implicated as a cause of TD [2]. Functional dopamine imbalance in basal ganglia is also implicated in catatonia [4]. Coming to the management, Clozapine is recommended for management of TD [2]. Literature also supports efficacy of Clozapine in catatonia [4,8]. The stronger D1 binding and rapid dissociation from D2 receptors can explain its efficacy in both the disorders [2]. The existing literature identifies ECT as treatment of choice for catatonia [4]. But the data regarding use of ECT in TD is ambiguous. There are a few case reports of successful treatment of TD by ECT [9,10]. But other authors have identified ECT as a risk factor for TD [5]. In our case, ECT led to some improvement in catatonia, but the dystonia persisted. Both catatonia and dystonia improved significantly on treatment with Clozapine. We recommend use of Clozapine for the management of TD presenting along with catatonia because of its efficacy in the management of both the disorders. In our case, there was further improvement in TD on treatment with THP. Existing literature supports use of THP in higher dosage for TD [11].

Conclusion

Patients of catatonia presenting along with TD present a clinical challenge for diagnosis as well as management. We recommend thorough investigation for these patients to exclude presence of organic etiology, NMS and epileptic seizure. ECT must be used with caution in these patients. We recommend use of Clozapine for management of these cases considering its efficacy and safety in both TD and catatonia. Further research is needed in the field of etiopathogenesis and management of TD.

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