Treatment of Catatonia in Parkinson's Disease with Electroconvulsive Therapy

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Abstract

Parkinson's disease (PD) is known to have associated nonmotor manifestations including psychiatric symptoms such as depression and psychosis. Catatonia has been reported extremely rarely in patients of PD. The case described here is a rare example of catatonia in a patient with PD with psychosis. Treatment with electroconvulsive therapy (ECT) brought improvement in symptoms of both PD and catatonia. ECT appears to be an effective treatment option in patients of PD, especially with psychiatric manifestations.

Keywords: Catatonia, electroconvulsive therapy, Parkinson's disease

Introduction

Parkinson's disease (PD) is a neurological disorder due to degeneration of dopaminergic neurons in substantia nigra, manifesting as rigidity, bradykinesia, and tremors.[1] Nonmotor manifestations of PD include a range of symptoms such as autonomic dysfunction, cardiovascular instability, urinary dysfunction, gastrointestinal dysfunction, rapid eye movement sleep behavior disorder, and psychiatric symptoms. Most common psychiatric symptoms reported are depression, anxiety, apathy, and impulsivity. Psychotic symptoms in such patients are most commonly seen due to antiparkinsonian medications.[2] Catatonia, however, is a rare phenomenon in patients with PD. It is a disorder of psychomotor disturbance, characterized by stupor, catalepsy, waxy flexibility, mutism, negativism, agitation, and stereotypy. It is defined by the presence of three or more above-mentioned features, associated with mental disorder or due to medical condition.[3] So far, only three cases of catatonia with PD

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Submission: 12.07.2018 **Revision:** 15.07.2018
Acceptance: 16.07.2018 **Published:** 25.10.2019

DOI: 10.4103/aian.AIAN_308_18

have been described [Table 1].[4-6] Here, we describe a case of a patient with PD with psychosis who developed catatonia during inpatient care and responded to electroconvulsive therapy (ECT).

Case Report

A 55-year-old male farmer from a rural background with no family history of medical or psychiatric illness had presented to a physician with complaints of insidious onset, progressive course of illness of 3 years' duration, characterized by left-sided pill rolling tremors, generalized rigidity, and slowness in movements. He was diagnosed of Idiopathic PD and was started on a combination of levodopa–carbidopa 300 mg/day which was increased to 600 mg/day over a period of 1 year. He was also on pramipexole 2 mg/day and trihexyphenidyl (THP) 8 mg/day at the time of presentation. He was noted to have psychotic symptoms for the past 1 month, characterized by persecutory delusion, hallucinatory behavior, and agitation. The patient was admitted for the management of both motor symptoms of PD and psychotic symptoms. After onset of psychotic symptoms, he was taking treatment of PD irregularly. Neurological examination revealed unilateral tremors and generalized rigidity. He was started on quetiapine 100 mg/day and increased till 300 mg/day for the management of psychotic symptoms and the rest of the medications were continued at the same doses. Two days following admission, he was noted to refuse food, water, and medicines and was actively resisting any attempts to feed him. On examination, he also had mutism, negativism, rigidity, and withdrawal with Bush–Francis Catatonia Rating Scale (BFCRS) score of 13. A diagnosis of catatonia was made in the absence of fever and elevated white blood cell count. A trial of injection lorazepam 2 mg was given following which he started to talk and respond, though the rigidity and tremors persisted. In view of catatonia, other psychotic symptoms, and prominent symptoms of PD, ECT was considered as a treatment option because ECT is often a lifesaving treatment in catatonia and helps in the treatment of PD also. After taking informed consent and necessary investigations for administering anesthesia, modified bitemporal ECT was given on alternate days. He started to show improvement in food intake and speech output after the 2nd ECT. There was also a reduction of tremors, rigidity, and psychotic symptoms following ECT. The patient continued to show further improvement till the 4th ECT. After spacing out 5th and 6th ECTs, there was no worsening of symptoms of catatonia, and ECT was stopped. At the end of the 6th ECT, BFCRS score was 2 and there was complete relief in symptoms of psychosis and PD. The dose of THP was subsequently reduced to 4 mg/day. He was continued on 600 mg of levodopa–carbidopa combination, 2 mg of pramipexole, and 400 mg of quetiapine. He is under regular follow-up and doing well for the last 6 months.

Discussion

Catatonia is fairly common in background of psychotic and mood disorders, but in patients of PD, it is a rare manifestation. In one such study, it was found that 9.45% of indoor patients of severe mental disorders (psychosis, bipolar disorder, and depression) had catatonia.[7] The mechanism behind catatonia is poorly understood. However, abnormalities in gamma-aminobutyric acid (GABA)-ergic system have been implicated in catatonia, especially the involvement of GABA-A receptors. Response to lorazepam has further added to the evidence for the same. Further studies have also noted that there may be a secondary involvement of glutamatergic system, based on the response to amantadine in those whose symptoms were refractory to lorazepam. Severe blockade in dopamine has also been proposed.[8]

Till date, only three cases have been reported where a patient diagnosed with PD presented with catatonia and treated with ECT [Table 1].[4-6] In the first case, the patient developed catatonia after withdrawal of antiparkinsonian medications due to the development of psychotic symptoms. ECT was initiated and she showed a good response to the same.[4] In the second case, the patient developed catatonia after altering the dose of anti-PD medication due to the development of psychotic symptoms which were thought to be drug induced. Since there was a partial improvement in symptoms after increasing the dose of levodopa, ECT was started to which the patient responded well.[5] Third case report discusses about a patient with PD who developed psychosis followed by recurrent episodes of catatonia who showed short-lived, but marked response to administration of lorazepam. Hence, she was started on ECT to which she responded well, but continued to exhibit residual catatonic symptoms. However, her food

Table 1: Clinical profile of cases of Parkinson's disease with catatonia and response to electroconvulsive therapy

Author, year	Age, sex	Diagnosis	Medications	ECT sessions	Response
Suzuki *et al*., 2006[4]	62 years, female	PD with psychosis and catatonia	Levodopa–carbidopa 600 mg/day, cabergoline 4 mg/day, selegiline 10 mg/day, and zonisamide 100 mg/day	12	Improvement in symptoms of PD, psychosis, and catatonia
Kamigaichi *et al*., 2009[5]	75 years, female	PD with psychosis and catatonia	Levodopa-carbidopa 400 mg/day, pramipexole 3 mg/day, and entacapone 700 mg/day	2	Improvement in symptoms of PD, psychosis, and catatonia
Poyraz *et al*., 2016[6]	80 years, female	PD with psychosis and recurrent catatonia	Levodopa/benserazide 800/200 mg/day, memantine 20 mg/day, and pramipexol 4 mg/day	6	Residual symptoms of catatonia persisted

PD=Parkinson's disease, ECT=Electroconvulsive therapy

intake had improved, and with addition of amantadine along with PD medications, the patient showed marked improvement and was discharged.[6]

In index case and all the three cases discussed above, patients of PD developed catatonia after onset of psychotic symptoms. Hence, it is difficult to judge whether catatonia manifested due to psychosis or due to underlying PD. A trial of lorazepam was not effective and hence ECT was considered for the present case. ECT was effective and he continued to maintain the response. ECT is a well-established treatment modality for catatonia. It is often a lifesaving procedure. Response to ECT in patients with catatonia is generally good, with nearly 85% response rate. Patients show a rapid response with only a few sessions. However, it is less responsive if catatonia is of organic origin, requiring exploration of other treatment options.[9]

There are several cases where ECT has been found to be useful in patients with PD, though the exact mechanism is not clearly understood. It is hypothesized that ECT improves dopaminergic transmission and hence alleviates motor symptoms of PD.[10] There are several case reports and series where ECT has been shown to be efficacious in the management of both motor and psychiatric symptoms of PD.[10,11] However, ECT is not regularly used probably because of lack of knowledge of the same. In the index case, the patient showed improvement in both catatonic symptoms and motor symptoms of PD. Hence, ECT can be considered as a safe treatment option in patients with PD and comorbid psychiatric illness.

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.

Financial support and sponsorship

Nil.

Conflicts of interest

There are no conflicts of interest.

References

1. Ropper AH, Victor M, Samuels MA, Adams RD. Adams and Victor's Principles of Neurology. 9th ed. New York: McGraw Hill Medical; 2009.
2. Zis P, Erro R, Walton CC, Sauerbier A, Chaudhuri KR. The range and nature of non-motor symptoms in drug-naive Parkinson's disease patients: A state-of-the-art systematic review. NPJ Parkinsons Dis 2015;1:15013.
3. Taylor MA, Fink M. Catatonia in psychiatric classification: A home of its own. Am J Psychiatry 2003;160:1233-41.
4. Suzuki K, Awata S, Nakagawa K, Takano T, Matsuoka H. Catatonic stupor during the course of Parkinson's disease resolved with electroconvulsive therapy. Mov Disord 2006;21:123-4.
5. Kamigaichi R, Kubo S, Ishikawa K, Yokoyama K, Ogaki K, Usui C, *et al.* Effective control of catatonia in Parkinson's disease by electroconvulsive therapy: A case report. Eur J Neurol 2009;16:e6.
6. Poyraz BÇ, Aksoy Poyraz C, Yassa A, Arikan MK, Gündüz A, Kiziltan G. Recurrent catatonia in Parkinson disease. J Clin Psychopharmacol 2016;36:104-6.
7. Grover S, Chakrabarti S, Ghormode D, Agarwal M, Sharma A, Avasthi A, *et al.* Catatonia in inpatients with psychiatric disorders: A comparison of schizophrenia and mood disorders. Psychiatry Res 2015;229:919-25.
8. Northoff G. What catatonia can tell us about "top-down modulation": A neuropsychiatric hypothesis. Behav Brain Sci 2002;25:555-77.
9. Sienaert P, Dhossche DM, Vancampfort D, De Hert M, Gazdag G. A clinical review of the treatment of catatonia. Front Psychiatry 2014;5:181.
10. Narang P, Glowacki A, Lippmann S. Electroconvulsive therapy intervention for Parkinson's disease. Innov Clin Neurosci 2015;12:25-8.
11. Grover S, Somani A, Sahni N, Mehta S, Choudhary S, Chakravarty RK, *et al.* Effectiveness of electroconvulsive therapy (ECT) in Parkinsonian symptoms: A case series. Innov Clin Neurosci 2018;15:23-7.