Exploration of Neuroprotective Therapy

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Catatonia in two women with Parkinson's disease treated with electroconvulsive therapy

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Abstract

Catatonia is a neuropsychiatric syndrome characterized by a broad range of motor, behavioral and cognitive abnormalities. Catatonia and Parkinson's disease (PD) may show partially overlapping symptomatology. For this reason, catatonia could be misdiagnosed and overlooked in patients with severe PD, leading to a delay in proper treatment with benzodiazepines or electroconvulsive therapy (ECT). Two cases of women with PD and catatonia who have been admitted and treated with ECT at the University Hospital of Pisa are described here. Both had a history of bipolar disorder and developed withdrawn catatonia, in the context of affective episodes, approximately one year after the diagnosis of PD. In both cases, ECT was needed and successfully led to the remission of catatonic symptoms, without cognitive worsening. Since ECT appears to effectively treat catatonia in patients with PD, clinicians should consider it as a therapeutic option.

Keywords

Catatonia, Parkinson's disease, electroconvulsive therapy, mood disorder, movement disorders

Introduction

Catatonia is a psychomotor syndrome characterized by motor signs associated with mood, behavior, and thought disorders. This syndrome can occur in the context of different psychiatric conditions including affective, psychotic, and neurodevelopmental disorders, but may also be diagnosed in association with non-psychiatric conditions, including neurological, autoimmune, endocrine, and infective diseases [1]. Motor alterations that characterize catatonia typically have an acute onset and are often associated with mood changes. Stupor, mutism, negativism, rigidity, posturing, and staring are prominent motor signs [2]. Despite the differential diagnosis can be challenging, catatonia is a syndrome with a good prognosis if early identified and treated. The diagnosis can be verified by rapid relief with the intravenous administration of

benzodiazepines. The choice of treatment, whether benzodiazepines or electroconvulsive therapy (ECT), is determined by the severity of the illness, the presence of fever, and the degree of autonomic instability. The use of neuroleptics can be potentially harmful and should be avoided until catatonic signs are present [3, 4]. Catatonia may be diagnosed as a specifier that better characterize underlying mental disorders or specific medical conditions based on the presence of at least three signs from the following list: stupor, catalepsy, waxy flexibility, mutism, negativism, posturing, mannerisms, stereotypies, agitation, grimacing, echolalia, and echopraxia [5]. Since all catatonic signs pertain to the psychomotor domain, any comorbid syndrome associated with motor alterations may complicate the diagnosis of catatonia.

Parkinson's disease (PD), historically defined as a movement disorder, is now considered the prototypical neuropsychiatric condition, in which motor signs, such as tremors, rigidity, and bradykinesia, occur together with cognitive and neuropsychiatric features [6]. Depression [7], bipolar disorder [8, 9], psychosis [10], anxiety [11], impulse control disorders [12], apathy [13], and sleep disturbances [14] can frequently and significantly impact the course of PD. Despite the high psychiatric comorbidity reported in PD patients, few cases of catatonia have been reported in the literature [15–18]. Given the overlapping manifestations of catatonia and PD, especially the akinetic type, the differential diagnosis may be difficult in some cases. In severe PD patients, in fact, catatonia is frequently misdiagnosed and overlooked. However, acute akinesia, a condition that may be difficult to distinguish from catatonia, has been described in patients with PD [19]. Acute akinesia, indeed, is considered a rare complication of PD, which can occur as a result of infections, traumas, surgery, or gastrointestinal tract diseases. As in the case of malignant catatonia, the clinical picture is characterized by a sudden deterioration in motor performance persisting for 48 h or more, despite treatment, and resulting in a hypertonic-akinetic state usually associated with increases of body temperature and muscular damage, as evidenced by increments of creatine phosphokinase and myoglobin [19].

Despite symptoms of catatonia and PD may appear clinically similar, they are likely to stem from different pathological backgrounds. On one hand, catatonia has been hypothesized to reflect "top-down" dysfunction of the cortico-striatal-thalamic-cortical loop, involving both gamma-aminobutyric acid (GABA)ergic, glutamatergic, and dopaminergic systems [20]. In PD, instead, motor symptoms result from dopaminergic-mediated deficits in the striatum that lead to alterations in "bottom-up modulation" of the premotor/motor cortex [21]. However, cortico-striatal circuits in patients with PD and catatonia can be affected by multiple dysfunctions contributing to changes both in "bottom-up" and "top-down" modulation.

Benzodiazepines and ECT are the mainstay therapy for catatonic symptoms [22]. ECT is highly effective in the treatment of catatonia with response rates ranging from 80% to 100% [23] and is indicated in cases refractory to benzodiazepine treatment or in malignant forms of catatonia. Notably, ECT is also a treatment option for PD patients. According to a systematic review of 116 patients with depression and PD, 93.1% of all patients experienced improvement in depressive symptoms and 83% showed improvement in motor symptoms' severity. Concurrent improvements in motor symptoms in PD patients treated with ECT for depression may be due to increased dopaminergic neurotransmission [24]. However, the beneficial effect on motor symptoms of PD is probably transient [25]. Despite no conclusions could be made because of few data, no significant deterioration of cognitive functions has been evidenced from baseline to the end of the ECT in these patients. Nevertheless, patients with PD are at higher risk of side effects than those without PD, the most common being delirium and transient confusion [26].

So far, only four cases of catatonia treated with ECT in patients with PD have been described [15–18], three of which showed a significant improvement in psychiatric, motor, and catatonic symptoms after the ECT course. Two additional cases of patients with PD and comorbid bipolar disorder who developed catatonia and were successfully treated with ECT are described in this paper. In both cases, ECT was administered bitemporally using a brief pulse stimulator Mecta 5000Q (Mecta Corporation, Lake Oswego, USA), on a twice-a-week schedule. Parameters included a pulse width of 1.0, duration ranging from 1.5 s to 4.0 s, frequency ranging from 40 Hz to 90 Hz, and a current of 0.8 A. Anesthesia was induced with intravenous thiopental (2–4 mg/kg) and muscle relaxation was assured with succinylcholine (0.5–1 mg/kg).

The initial stimulus dosage was based on the "half-age" stimulation strategy for ECT dosing [27]. Motor and electroencephalogram (EEG) seizure duration were monitored. During the ECT course, the stimulus dosage was adjusted to maintain a seizure duration of at least 25 s.

Case report

Case 1

Mrs. B.T. was a 56-year-old Caucasian woman, with a history of bipolar disorder type 2, panic, and obsessive-compulsive disorder. For these conditions, she had been successfully treated, from the age of 20, with different combinations of serotonin-reuptake inhibitors, tricyclic antidepressants, and mood stabilizers. Antipsychotic medication has never been prescribed for the patient. At age of 54, she began presenting stiffness of the limbs and bradykinesia. These motor signs first developed on the right side of the body and then spread to the other, although symptoms continued to be worse on the initially affected side. The patient was then referred to a neurologist. Nigrostriatal dopaminergic degeneration was detected on 123I-FP-CIT brain single-photon emission computed tomography 18 months after the onset of motor symptoms. An akinetic-rigid PD was diagnosed based on the Movement Disorder Society (MDS) clinical diagnostic criteria for PD [28]. Psychiatric treatments were gradually withdrawn and antiparkinsonian drugs were prescribed with a rapid and significant improvement in motor symptoms. In subsequent months, the patient stayed on the neurological follow-up. She took the antiparkinsonian therapy correctly with good control of motor symptoms. Moreover, she showed no mood or anxiety relapses.

At 56 years of age, the patient experienced a depressed mood, apathy, loss of interest, and psychomotor retardation. Obsessive symptoms appeared again, in association with anxiety. The symptomatology gradually worsened with the appearance of catatonic symptoms such as motor inhibition, rigidity, and stereotypies. Two months later her state deteriorated with the appearance of mutism, negativism, and refusal to eat and drink, and she was hospitalized in our ward at the Second Psychiatry Unit of Pisa University Hospital. At that time, she was on stable doses of antiparkinsonian drugs (levodopa 600 mg/day, carbidopa 150 mg/day, and rotigotine 8 mg/day). She was also taking memantine 20 mg/day and gabapentin 600 mg/day. On admission, the patient was not able to speak. Immobility, negativism, withdrawal, staring, posturing, rigidity, waxy flexibility, and catalepsy were observed. Orientation, cognition, and content of thought could not be investigated. The patient failed to respond to external stimuli and showed a decreased reactivity to painful stimuli. Spontaneous feeding was absent, so nutrition and pharmacological treatments were administered by nasogastric tube. The Bush-Francis Catatonia Rating Scale [29] confirmed the diagnosis of catatonia with a severity score of 22 out of 69 points. Hematological, biochemical, metabolic, inflammatory, and thyroid blood tests yielded unremarkable results, except for an increase in levels of creatine phosphokinase (668 IU/L). The brain magnetic resonance imaging showed mild cortical atrophy and white matter hyperintensities of presumed vascular origin.

After the diagnosis of catatonia was made, treatment for PD was continued and intravenous lorazepam was started at 2 mg twice daily. The total daily dose of lorazepam was increased to 16 mg in the following days because of inadequate response. Despite the increase in the dose, the patient only showed a mild response to lorazepam. Rigidity slightly decreased and the patient began speaking again, although she had poor, not spontaneous, slowed down, and hypophonic speech. She was hypomimic and unable to move. Posturing, motor inhibition, and, occasionally, negativism were still present. She seemed able to recognize family members but was confused and disoriented. The psychiatric examination revealed a depressed mood and pessimistic thoughts.

Given the unsuccessful response to lorazepam, we obtained informed consent from the legal guardian of the patient and started ECT. Before beginning ECT memantine was stopped, levodopa was decreased to 200 mg/day, carbidopa to 50 mg/day, and rotigotine to 4 mg/day. After 8 ECT sessions, mood improved and catatonic symptoms partially remitted. The patient was collaborative and oriented in space, time, and person. She had a spontaneous and informative speech and resumed oral feeding with a semi-liquid diet. She showed an improvement in facial expression, reduction of rigidity, and resumption of voluntary

movements, even if she needed assistance to stand and walk. Two weeks after the end of ECT, complete remission of catatonia was obtained, and the patient was discharged and transferred to a neuro-rehabilitation unit. The therapy at discharge included gabapentin 600 mg/day, lorazepam 7.5 mg/day, levodopa 300 mg/day, carbidopa 75 mg/day, amantadine 100 mg/day, and rotigotine 4 mg/day.

Case 2

Mrs. R.A. was a 58-year-old Caucasian woman, with a history of bipolar disorder. At the age of 26, during the second post-partum, the patient experienced her first depressive episode, which was followed by recurrent seasonal mild hypomanic and depressive phases. The first psychiatric consultation was held at the age of 42 due to the worsening of depressive symptoms. The patient was then treated with several combinations of antidepressant treatments, including serotonin-reuptake inhibitors, tricyclic antidepressants, mirtazapine, and low-dose amisulpride. At the age of 45, her first psychotic depressive episode occurred, and she was first hospitalized and treated with a combination of mood stabilizers (lithium carbonate, carbamazepine), antidepressants (clomipramine), and antipsychotics (chlorpromazine, levomepromazine). Since remission was not achieved, the patient was referred to our hospital and was successfully treated with ECT. She was discharged on a combination of lithium carbonate, nortriptyline, and perphenazine. Gradually, nortriptyline and perphenazine have been reduced and stopped. The patient stayed well for several years with a therapy based on lithium carbonate. At the age of 53, she presented with a mixed manic episode with psychotic symptoms and was again hospitalized at our unit. She was then successfully treated with lithium carbonate, valproic acid, and quetiapine. Some months after discharge, she began presenting hand tremors that did not subside after the reduction of lithium carbonate. Bradykinesia and stiffness of the limbs, particularly the right one, were noticed at the age of 56, so the patient was referred to a neurologist. After 6 months, 123I-FP-CIT brain single-photon emission computed tomography showed nigrostriatal dopaminergic degeneration, PD was diagnosed based on the MDS Clinical Diagnostic Criteria for PD [28] and antiparkinsonian treatment with levodopa, benserazide, and pramipexole was prescribed.

Once the treatment of the antiparkinsonian drugs was started the patient had a significant and sudden improvement in motor symptoms. One year later, at 58 years of age, the patient experienced a sudden increase in anxiety, restlessness, and insomnia, that rapidly evolved, within one week, into a catatonic state characterized by immobility, negativism, increased rigidity, posturing, and refusal to eat or drink. While she was referred to our hospital, the coronavirus disease 2019 (COVID-19) pandemic started, and the hospitalization had to be delayed. Meanwhile, antiparkinsonian treatment was increased (levodopa 600 mg/day, benserazide 150 mg/day), current psychiatric treatments were decreased (lithium carbonate 150 mg/day, valproic acid 500 mg/day, quetiapine 200 mg/day), and delorazepam was introduced and titrated up to 5.2 mg/day. No improvements were observed over two months and the patient was finally hospitalized at the Second Psychiatry Unit of Pisa University Hospital.

On admission, immobility, negativism, withdrawal, staring, posturing, rigidity, waxy flexibility, and catalepsy were observed. Mutism alternated with stereotyped speech and singing. Responses to external stimuli were elicited with difficulty, but reactivity to painful stimuli was preserved. While orientation to time and space was difficult to investigate, orientation toward people fluctuated. Emotional expressions alternated between perplexity and increased intensity, with rapid changes from terror to cheerfulness or sadness. Spontaneous feeding was absent. A nasogastric tube was used for nutrition and pharmacological treatment administration. The Bush-Francis Catatonia Rating Scale [29] confirmed the diagnosis of catatonia with a severity score of 23 out of 69 points. Blood tests results were within the norm, except for mild normocytic anemia, iron deficiency, slightly reduced prothrombin activity, and increased creatine phosphokinase (up to 1,051 IU/L) and C-reactive protein (8.06 mg/dL). The brain computerized tomography showed no signs of acute pathology.

After the diagnosis of catatonia was made, treatment for PD was continued in accordance with neurologists, current psychiatric treatments were discontinued, and intravenous lorazepam was started and titrated up to 16 mg, without substantial improvements over two weeks. Informed consent was obtained

from the legal guardian of the patient and ECT was initiated. During the course of ECT, amantadine and gabapentin were introduced, while intravenous lorazepam was slowly withdrawn in parallel with clinical improvements. When speech output increased and orientation improved, it became possible to explore the content of thought. Paranoid delusions and terrific experiential hallucinations emerged. Lithium carbonate was reintroduced but was not tolerated (i.e., rigidity worsened). Clozapine was finally started and slowly titrated up to 50 mg/day during the last weeks of treatment.

After 15 ECT sessions, catatonic symptoms remitted, although impairments of the movement were still evident due to severe hyposthenia and muscle hypotrophy. However, the patient was able to walk with assistance and eat autonomously, making it possible to resume oral feeding with a semi-liquid diet. She also showed improvements in facial expression and resumption of voluntary movements. She was collaborative and oriented in space and person, though had some mild disorientation in time. Spontaneous speech was present, though slowed down and hypophonic. Two weeks after the end of ECT, the patient was discharged and transferred to a neuro-rehabilitation unit. The therapy at discharge included clozapine 50 mg/day, gabapentin 900 mg/day, delorazepam 0.4 mg/day, levodopa 600 mg/day, benserazide 150 mg/day, and amantadine 100 mg/day.

Discussion

Both of our patients had a diagnosis of bipolar disorder, idiopathic PD, and catatonia. While the first patient had a rather classical course of bipolar disorder type 2, the second one transitioned through several diagnostic categories with increasing severity, experiencing, in order, major depression, hypomania, psychotic depression, mixed mania, and finally, catatonia. In both cases, the diagnosis of PD was clinically established according to the MDS clinical diagnostic criteria for PD by a neurologist with expertise in movement disorders [28]. In fact, both patients had bradykinesia in combination with rigidity and they showed a clear and dramatic beneficial response to dopaminergic therapy. Moreover, their motor symptoms were not better explained by an alternative condition, including iatrogenic parkinsonism. In fact, the patient described in case 1 has never taken antidopaminergic drugs and the patient described in case 2 has taken those medications at a dosage and for a length of time not enough to cause iatrogenic parkinsonism according to the neurologist. This last patient has mostly used quetiapine, one of the antipsychotics with the lowest risk of extrapyramidal side effects [30, 31]. Both patients had a withdrawn type of catatonia with stupor, mutism, and negativism. However, while the former patient first developed depressive symptoms gradually fading into motor inhibition, the second one had a sudden onset of catatonia, possibly emerging from a mixed affective state with both depressive and manic features. Either way, affective symptoms and a history of mood disorders supported the hypothesis that the clinical picture was secondary to a mood alteration rather than to an exacerbation of parkinsonian symptoms. Moreover, both patients did not improve after the anti-Parkinson drugs dose increase, further decreasing the probability of a worsening due to PD. Conversely, both patients improved after ECT. While the patient in case 1 recovered after 8 ECT sessions, 15 ECT sessions were needed before the catatonic signs disappeared in case 2.

Notably, ECT has been found highly effective in patients with bipolar depression and mixed syndromes [32, 33]. Importantly, the severity of psychomotor disturbances, including both motor retardation and agitation, has been positively associated with higher response rates in patients with bipolar disorder or major depression [32, 34]. Despite some authors suggesting the use of unilateral ECT in PD patients in order to reduce cognitive side effects [35, 36], bilateral ECT was adopted in our patients in accordance with most reports of ECT in catatonic patients [37]. Importantly, some authors strongly advocate bilateral ECT in critically ill patients, claiming secondary consideration for transient cognitive impairment in patients with severe catatonia [38, 39]. According to our protocol, however, a less frequent treatment schedule was used to prevent cognitive adverse effects such as delirium. Indeed, bilateral ECT was well tolerated by our patients without worsening cognition or post-ictal delirium.

To the best of our knowledge, only four cases of catatonia treated with ECT in patients with PD have been described [15–18]. Three patients were female and the age ranged between 55 years old and 80 years

old. Before the onset of catatonia, all patients had developed psychotic symptoms due to antiparkinsonian medications or attributable to PD progression. Three out of four patients had also had agitation prior to the beginning of catatonia [15–17]. In two cases, the onset of catatonia was closely related to the discontinuation or switch of antiparkinsonian drugs for psychosis [16, 18]. While three patients showed a significant improvement in psychiatric, motor, and catatonic symptoms after the ECT course, residual symptoms of catatonia persisted in an 80 years old woman with recurrent episodes of catatonia and cognitive impairment [17]. The number of sessions ranged from 6 to 12. The electrode positioning was reported only for one patient that had bilateral ECT [15].

Our cases and the four reports discussed above support the role of ECT in the treatment of catatonia in comorbidity with PD. In patients with PD, the diagnosis of catatonia requires a careful neurological assessment because of the symptomatic overlap between the two disorders. Moreover, clinical expertise in the management of catatonia and ECT procedure is needed because of the high risk of side effects in PD patients. Bitemporal positioning with a twice-a-week schedule may have guaranteed good efficacy in our patients, with a low risk of cognitive side effects. Hence, ECT is a valid option for the treatment of catatonia in PD patients. Clinicians who treat catatonic patients with neurodegenerative conditions such as PD should be aware of its safety and efficacy.

Abbreviations

ECT: electroconvulsive therapy

MDS: Movement Disorder Society

PD: Parkinson's disease

Declarations

Author contributions

CE, GEB, BT, ST, and PM: Writing—review & editing. PM, LL, and GP: Conceptualization. All authors read and approved the submitted version.

Conflicts of interest

The authors declare that they have no conflicts of interest.

Ethical approval

The study received the local Tuscany Ethical Committee's approval and was carried out in compliance with the ethical guidelines outlined in the 1964 Declaration of Helsinki and its later amendments. The study was carried out by the researchers in accordance with the protocol's approval and appropriate research standards.

Consent to participate

The authors declared that they have obtained and archived written patient consent forms for study participation.

Consent to publication

Not applicable.

Availability of data and materials

Not applicable.

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