

Treatment of Catatonia with Electroconvulsive Therapy in a 63-Year-Old Woman

Chester Pearlman, MD
D. William Savanin, MD

Catatonia is a relatively rare phenomenon typically characterized by an acute change of mental status, a sudden onset of akinesia, and negative results on head computed tomography (CT) scan. This article presents a case of catatonia in a 63-year-old woman with a history of paranoid schizophrenia. Diagnosis, differential diagnosis, and treatment options are also discussed.

CASE PRESENTATION

A 63-year-old woman with a long history of paranoid schizophrenia is transferred from the psychiatric unit to the medical service unit because of unresponsiveness.

Past Medical and Social History

Approximately 30 years prior to her current presentation, the patient had multiple psychiatric hospitalizations for paranoid schizophrenia. She also experienced an episode of catatonia 10 years prior to her current presentation; she was treated with and responsive to electroconvulsive therapy (ECT) for the episode.

The patient has been taking haloperidol (5 mg/day) for almost 20 years and, except for the previous episode of catatonia, has had no significant psychiatric problems during that time. The patient has no history of alcohol or drug abuse. The patient shares an apartment with a roommate. She has been unemployed for many years.

Recent History

Presentation. Approximately 1 month prior to hospital admission, the patient became increasingly withdrawn and estranged from her friends and relatives. On presentation to the psychiatric unit, she demonstrated the following symptoms: loss of interest in food and a 9-lb weight loss, inability to sleep more than 1 to 2 hours per night, and difficulty making any decisions. The patient's siblings suggested that she was depressed

because of her brother's suicide that occurred a few months prior to presentation.

Admission and initial treatment. The patient was admitted to the psychiatric unit with a diagnosis of major depressive disorder, and treatment with paroxetine was initiated. Despite treatment, the patient continued to isolate herself and ate little.

Four days after admission, the patient was found in the bathroom slumped over a toilet and unresponsive. On physical examination, she was afebrile, mildly hypertensive (151/84 mm Hg), and unresponsive to verbal and painful stimuli. Muscle tone was flaccid, and no respiratory distress was observed. Transfer to the medical service unit was ordered. At the time of transfer, the patient's medications were haloperidol (5 mg/day), benzotropine (1 mg/day), paroxetine (20 mg/day), and oxazepam (15 mg at bedtime for insomnia).

Physical Examination

On examination in the medical service unit, the patient is lying on her back in bed with her eyelids half closed and eyeballs rolled backward. She shows no sign of physical injury and no reaction to verbal or tactile stimuli. She exhibits resistance when an examiner tries to open her eyes. Her pupils are normal size and reactive to light; eye movements are conjugate. Her gaze is predominantly fixed to an extreme upward right position, with occasional downward movements. Corneal reflexes are present as well as blinking responses to threat (ie, rapidly bringing a finger close to the eye of the patient). Doll's eye reflex is negative.

Dr. Pearlman is Consultant to Psychiatry Service, Boston VA Medical Center, Boston, MA, and Clinical Professor of Psychiatry, Boston University School of Medicine, Boston. At the time this article was written, Dr. Savanin was Psychiatry Chief Resident, Psychiatry Training Program, Boston University School of Medicine.

The patient's neck is supple, and she has no lymphadenopathy or thyroid enlargement. Her lungs are clear, and her breathing is shallow. Examination of the heart, abdomen, and extremities is unremarkable. Gag reflex is present; motor reflexes are normal and symmetrical throughout. Muscle tone is flaccid. Babinski's sign demonstrates bilateral, down-going toes.

Laboratory Examination

Initial laboratory tests are significant for mild anemia: hemoglobin, 11.9 mg/dL; hematocrit, 34.1%; and mean corpuscular volume, 87.2 fL. The patient also has slightly elevated globulin (4.2 mg/dL). Calcium is 9.6 mg/dL, and liver function is normal. Serum and urine toxic screens are negative.

Hospital Course and Continued Monitoring

Catatonia is suspected due to the patient's previous history and the absence of signs suggestive of herpes encephalitis or another acute neurologic event. The absence of rigidity and fever rules out neuroleptic malignant syndrome (NMS).

The patient's condition remains unchanged for 2 days. Due to no oral intake, she is given intravenous fluids. Results of electrocardiography and chest radiography are normal. A head CT scan is significant for volume loss disproportionate to the patient's age. Lumbar puncture shows no cells; protein is 56 g/dL, and glucose is 80 mg/dL. Opening pressure is 17 mm of water and closing pressure is 14 mm.

Treatment and Resolution

On the third day after the onset of catatonia, the patient awakes. On examination, she is alert, oriented only to person, and appears to be hallucinating. She is preoccupied with religious delusions, and her speech is almost incoherent. Her mental status fluctuates during the day from near catatonia to marked agitation. While agitated, she tries to get out of her bed and pulls out her intravenous line and Foley catheter.

The patient is restrained and given intramuscular lorazepam, with no response except decreased agitation. The lorazepam is administered only for 1 day. Because of the combination of the patient's prior depressive symptoms, the patient's history of good response to ECT, and the failure of lorazepam to produce an immediate response, ECT is the decided course of treatment.

The day after lorazepam administration, ECT is initiated. The patient is first anesthetized with thiopental. She is given succinylcholine to produce brief muscular

paralysis, and a stimulus of 250 mC (the measure of stimulus energy for current ECT devices) is administered.

After the first ECT treatment, the patient begins to eat and requests to take a shower for the first time since her admission. After two more ECT treatments over the next 5 days, her condition returns to normal baseline. Two days later, the patient is discharged. At discharge, she is euthymic and not psychotic. She shows good judgment and is fully capable of caring for herself.

DISCUSSION

Classification

In 1863, German psychiatrist Karl Kahlbaum first coined the term catatonia to describe a state of "negative tension." Eugen Bleuler, a Swiss psychiatrist, was first to establish a connection between schizophrenia and catatonia. Presently, catatonia refers not only to catatonic schizophrenia but also to a neurobehavioral syndrome caused by a medical or neurologic condition. Therefore, *primary catatonia*, a disorder of idiopathic or psychiatric etiology, must be differentiated from *secondary catatonia*, which develops from a non-psychiatric illness. Secondary catatonia can occur in any patient, including those with a history of catatonic schizophrenia. **Table 1** lists various types of primary and secondary catatonia.

Diagnostic Criteria

There have been several attempts to define diagnostic criteria for catatonia.¹⁻³ Each criterion contains many symptoms, including automatic obedience, grasp reflex, waxy flexibility, catatonic excitement, stereotypy, and mannerisms (ie, odd, purposeful movements such as hopping, walking tiptoe, or saluting passersby). However, the triad of mutism (or near mutism), akinesia (or severe hypokinesia), and dystonia (approximating catalepsy or rigidity) is sufficient for a diagnosis of catatonia.⁴

Incidence and Prevalence

Because of the absence of established diagnostic criteria and the variability in age, gender, and comorbid psychiatric and medical conditions in patient populations, reliable information about incidence and prevalence of catatonia is unavailable. For example, a prospective study of psychiatric inpatients using common criteria (eg, excitement, immobility, mutism, staring, posturing, grimacing, echolalia) identified a 7% incidence, whereas a 5-year retrospective study from the same institution noted a 0.5% prevalence.³ An incidence of 8% to 14% was noted in three other reports.⁵⁻⁷ In contrast, patients with catatonia represent less than 0.1%

of admissions to the authors' general hospital psychiatric service. For similar reasons, the incidence of recurrence in the same patient population is unknown.

Differential Diagnosis

Neuroleptic malignant syndrome. At least 50% of patients who present in catatonic states have a mood disorder other than schizophrenia¹⁻⁴ and are prone to the development of NMS. Because neuroleptic agents are commonly used for treatment of agitation, catatonic patients, who are often presumed to be psychotic, receive neuroleptics. The new onset of muscular rigidity caused by the neuroleptic can be misinterpreted as worsening of the catatonic state and treated with more neuroleptics. In turn, NMS often remains unrecognized and untreated and may result in death.

The differential diagnosis of catatonia and NMS is further complicated not only by similarity of symptoms but also by comorbidity.⁸ Any patient with a history of neuroleptic exposure who presents with mental status changes, rigidity, fever, and other evidence of autonomic disturbance should be treated as if he or she has NMS. For most patients with NMS, withdrawal of neuroleptics, supportive measures, and use of dantrolene and/or dopamine agonists (eg, bromocriptine) for muscular relaxation leads to recovery within 1 week. Some patients with persistent symptoms recover with the use of benzodiazepines,⁹ which supports the diagnosis of catatonia. A number of patients have been treated successfully with ECT.^{10,11} Finally, because the initial presentation of herpes encephalitis may mimic catatonia, a lumbar puncture may be warranted.

Treatment of Catatonia

Lorazepam. The majority of catatonic patients demonstrate rapid remission with intramuscular or intravenous administration of lorazepam. Patients are usually treated with 4 to 8 mg/day of lorazepam for up to 5 days. The initial challenge is a 1-mg dose, which may be repeated in 5 minutes. Although a full trial of lorazepam takes 5 days, the first response usually occurs within 1 to 2 days.^{12,13} Successful treatment is defined as complete resolution of catatonic signs.

Electroconvulsive therapy. For patients who demonstrate no response, only a transient response, or a deterioration despite supportive therapy, ECT is the option of choice. ECT usually results in rapid improvement. There are few relative and no absolute contraindications to ECT.^{14,15}

Despite overwhelming evidence of safety and efficacy,¹⁶ the controversial image of ECT persists because of lack of awareness and understanding. Catatonic pa-

Table 1. Primary and Secondary Catatonia

Primary catatonia*

Associated with:

- Bipolar disorder
- Schizophrenia
- Major depressive disorder
- Schizoaffective disorder
- Other psychiatric disorders
- Brain atrophy

Secondary catatonia*

Secondary to:

- Herpes encephalitis
 - Neuroleptic malignant syndrome
 - Brain tumor
 - Akinetic epilepsy or mutism
 - Parkinsonism ("Parkinsonian freezing state")
 - Temporal lobe epilepsy
 - Other seizure disorders (especially, postictal state)
 - Brain trauma
 - Cerebral vascular disease
 - Toxic or metabolic encephalopathy
 - Psychotropic agents (eg, cocaine)
 - Systemic infections (eg, cholera)
 - Systemic illnesses (eg, lupus erythematosus)
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*Listed in order of most frequent to least frequent occurrence.

tients are generally uncooperative with life-support measures (ie, efforts to provide adequate fluid and food intake and maintain personal hygiene) and often die unless successfully treated. Therefore, ECT is literally lifesaving for many patients, especially those patients whose catatonia was preceded by a period of extreme agitation (sometimes called *malignant catatonia*).^{17,18}

Risks of electroconvulsive therapy. The risks of ECT result from the associated autonomic stimulation and increased intracranial pressure. If an intracranial mass is suspected, a head CT scan should be obtained before treatment. Although an intracranial mass is a relative contraindication, these patients may be treated without morbidity.^{15,16} Patients with a recent myocardial infarction, arrhythmias, labile hypertension, or intracranial aneurysms require appropriate blockade of autonomic stimulation.

Administration of electroconvulsive therapy. ECT is typically administered 2 or 3 times a week, but the rapidity of response in patients with catatonia generally makes

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treatment frequency irrelevant. Because of rapidity of response, any effect of treatment frequency cannot be reliably estimated. For twice-weekly treatment, bilateral electrode placement is common; for thrice-weekly treatment, nondominant placement is used to reduce cognitive side effects (eg, confusion, memory disturbance). For bilateral placement, a moderately suprathreshold stimulus is used, whereas nondominant placement requires at least five times suprathreshold stimulation.^{19,20} ECT is continued until improvement reaches a plateau. Unlike depressive disorders in which relapse is common and often requires maintenance ECT,²¹ patients with catatonia generally experience long periods of remission.

Failure of electroconvulsive therapy. According to Hawkins et al,¹³ ECT was ineffective in 1 of 49 cases excluding misdiagnoses (eg, encephalitis) and patients responsive to lorazepam. Treatment options when ECT fails are empirical and limited. Double stimulation (induction of two seizures in one session); use of flumazenil to block residual benzodiazepine effects, caffeine to prolong seizures, and clozapine to reduce seizure threshold; and atypical electrode placements and stimulus dosage have been attempted. Rare patients still fail to respond.

SUMMARY

Despite low incidence, catatonia is a serious diagnostic and treatment challenge. After the main causes of secondary catatonia have been ruled out, referral to a psychiatric service with ECT capability is the appropriate next step. If a trial of lorazepam fails, ECT should be used. The successful outcome with ECT in the patient in this case study confirmed the diagnosis of primary catatonia.

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