From the Albert Einstein Medical Center

ECT in the Treatment of a Patient With Catatonia: Consent and Complications

Marc H. Zisselman, M.D.

Richard L. Jaffe, M.D.

Acute catatonia in an adolescent or young adult can present complex clinical challenges. Prominent issues include those involving diagnosis, timely and effective treatment, and diminished capacity to provide consent. The authors describe a 19-year-old woman presenting initially with manic excitement followed by a lengthy period of mutism, immobility, and food and fluid refusal. Elevated temperature, an elevated creatine phosphokinase level, and autonomic dysfunction led to consideration of a malignant catatonic syndrome. The patient manifested rigidity accompanied by posturing and waxy flexibility. Neurologic, medical, and laboratory evaluations failed to identify an organic cause for the likely catatonia. Treatment with amantadine, bromocriptine, and lorazepam was unsuccessful. ECT was deemed appropriate but required emergency guardianship because of the patient's inability to provide consent. At the initial ECT session, the elicited seizure was followed by an episode of torsade de pointes requiring immediate cardioversion. In reviewing the ECT complication, it appeared that muscle damage due to catatonic immobility led to acute hyperkalemia with the administration of succinylcholine. Discussions were held with the patient's guardian outlining the clinical issues and the risks of additional ECT. The patient responded to eight subsequent ECT sessions administered with rocuronium, a nondepolarizing muscle relaxant. The authors provide a brief review of the diagnosis and treatment of catatonia and address issues surrounding ECT, cardiac effects, use of muscle relaxants, and the consent process.

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Case Presentation

Ms. P, a 19-year-old woman with no past psychiatric history or family psychiatric history, was voluntarily admitted to a freestanding for-profit psychiatric hospital with a chief complaint of "I need to mellow down." The history of the present illness, obtained primarily through the patient's sister, revealed 48 hours of pressured, often nonsensical speech, absent sleep, almost continuous dancing, brief spontaneous crying spells, auditory hallucinations, and the delusion that her stepfather impregnated her. The initial admission workup revealed a negative urine drug screen and pregnancy test and unremarkable CBC and chemistry panel. Ms. P was given an admission diagnosis of bipolar disorder, manic with psychosis, and was started on ziprasidone, 60 mg b.i.d., and lorazepam, 1 mg b.i.d. Two days into the hospitalization, she spiked a fever of 38.2°C and was not eating, talking, or moving. She was transferred to a local medical emergency department, where the evaluation included a lumbar puncture and head CT scan; results for both were normal, and the patient was returned to the psychiatric hospital. The fever, immobility, and food refusal persisted over the next 2 days while Ms. P was given trials of olanzapine, 10 mg orally and 5 mg i.m.; haloperidol, 5 mg i.m.; and aripiprazole, 10 mg orally. At this time, she was medically admitted to the community hospital.

In the hospital, Ms. P's heart rate ranged from 120 to 150, her respiratory rate was 30–40, and laboratory workup revealed a creatine phosphokinase level of 4,600 U/liter (normal range, 35–274 U/liter) and metabolic acidosis. An EEG displayed no epileptiform activity, and a repeat head CT scan was also read as normal. The patient remained mute and immobile, with no food or fluid intake. A nasogastric tube and an intravenous line were placed, and a dose of amantadine was administered through the tube. Ms. P. developed hives and was treated with methylprednisolone for a presumed allergic reaction. Her mental status remained unchanged, prompting her transfer to a tertiary care hospital 13 days after her original admission.

At the hospital, Ms. P was admitted to the neurology service and received admission diagnoses of acute parkinsonism with hypoxia, neuroleptic malignant syndrome, and catatonia. She was found to be nonverbal and rigid, with posturing and waxy flexibility. She had a skin rash on her left thigh. Her heart rate remained in the range of 125–150, her respiratory rate in the range of 25–35, and her temperature 38.5°C. Her pulse oximetry reading was 97% on 2 liters/minute of oxygen.

Results of a more extensive workup included negative findings from a hepatitis panel and tests for HIV, antinuclear antibody, vancomycin-resistant enterococci, thyroid-stimulating hormone, and methicillin-resistant *Staphylococcus aureus*. A repeat EEG revealed no seizure pattern and no abnormalities in background rhythm, findings of a body CT scan were normal, and echocardiography showed normal left and right ventricular size

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and function and no significant valvular regurgitation. CSF analysis was negative for fungus. A ventilation/perfusion scan was negative for pulmonary embolus. Abnormal findings included a WBC count of 21,000 (normal range, 4.8–10.8×10^3; differential not performed), an albumin level of 2.5 g/dl (normal range, 3.5–5.5 g/dl), serum protein electrophoresis with increased a2 globulin, and central line culture revealing gram-positive cocci.

Prescribed medications consisted of vancomycin and ciprofloxacin for the infection in addition to lansoprazole, metoclopramide, enoxaparin, metoprolol, and bromocriptine. All medications, fluids, and feedings were administered via intravenous line or nasogastric tube.

A psychiatry consultation was requested and obtained the day after admission. The evaluation led to a diagnosis of bipolar disorder with catatonic decompensation, and treatment recommendations were for intravenous lorazepam, elimination of metoclopramide, and the pursuit of emergency guardianship for ECT.

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The lorazepam was titrated up to 6 mg t.i.d. without any notable improvement in mental status but with a slight reduction in rigidity. Despite bedside physical therapy and nursing care interventions, Ms. P developed contractures in her left hand and bilateral foot drop.

Twenty days into Ms. P's hospitalization, a guardianship hearing was held at City Hall Orphan's Court. It was determined that Ms. P's 22-year-old sister was the family member best able to serve as a temporary emergency guardian. The guardianship order was written to cover any and all medical treatments, including ECT. Informed consent for ECT was obtained from the guardian, and the first treatment was administered 2 days later.

ECT treatment was performed in the hospital recovery room following standardized procedure. ECT was administered using a MECTA spECTrum 5000Q device (MECTA Corporation, Portland, Ore.) with bitemporal electrode placement, brief pulse currents, and initial energy settings at 20% of the device's maximum. At the time of initial monitoring, Ms. P's pulse was 143. Metoprolol, 5 mg i.v., was administered before treatment, lowering her pulse to 105. Based on her body weight, methohexital, 40 mg, and succinylcholine, 30 mg, were administered before treatment. The treatment produced a 28-second modified generalized seizure. Within 10 seconds of the seizure termination, Ms. P developed torsade de pointes, a form of ventricular tachycardia in which QRS morphology varies. A code was immediately initiated; Ms. P received synchronized DC cardioversion with 120 J and regained a rapid sinus rhythm. She was subsequently intubated and transferred to the medical intensive care unit. After extubation several hours later, it was noted that Ms. P was tracking with her eyes and for the first time spoke several words.

Ms. P remained in the medical intensive care unit for 24 hours. Cardiology consultation and additional testing were unable to ascertain the etiology of her acute life-threatening rhythm disturbance. Several pharmacologic recommendations were offered if Ms. P were to undergo further ECT. At this time, multiple conversations were held with her sister, addressing the potential risks and benefits of continuing the ECT treatment course. The treatment team was unable to delineate any additional treatment options for the malignant catatonia, which, left untreated, is associated with high morbidity and mortality. There had been a glimmer of initial response to ECT, but there was now an associated risk of reintroducing the life-threatening arrhythmia with future treatment. Ultimately, the guardian chose to continue the ECT, viewing it as the best hope to "get my sister back."

Ms. P received eight additional ECT treatments with robustly positive results. The pharmacologic regimen for all subsequent treatments included pretreatment with 5 mg i.v. of metoprolol and 1 g i.v. of magnesium and then 40 mg i.v. of methohexital and 15 mg i.v. of rocuronium, a nondepolarizing, fast-onset neuromuscular blocking agent, for treatment. Neostygmine, 1.5 mg i.v., was administered after treatment to reverse the rocuronium, and glycopyrrolate, 0.2 mg i.v., was given to prevent neostygmine-induced extreme bradycardia. There was no recurrence of posttreatment rhythm disturbances. By the conclusion of these treatments, Ms. P was eating and drinking adequately to meet her dietary requirements, engaging in daily physical therapy, and communicating appropriately with the treatment team, her family, and her friends. Mental status examination revealed no mood symptoms, perceptual disturbances, or thought disorder. Lithium carbonate pharmacotherapy was initiated at the conclusion of ECT, with dose titration to a blood level of 0.8 mEq/liter. By day 52 of her hospitalization, supplemental oxygen, intravenous lines, and the nasogastric tube were removed, and Ms. P was adequately stabilized medically and psychiatrically for discharge to a rehabilitation hospital.

Ms. P remained psychiatrically stable on lithium during her 21-day rehabilitation stay. By discharge, only mild left foot drop persisted. At 3-month follow-up, Ms. P reported that she was living with her boyfriend, that her ambulation was almost back to normal, and that she had resumed her usual activities.

Diagnosis

DSM-IV-TR recognizes catatonia as a subtype of schizophrenia, a specifier for bipolar disorder and major depression, and a disorder due to a general medical condition. Criteria include the presence of marked psychomotor disturbance characterized by motoric immobility or excessive motor activity; extreme negativism or mutism; peculiarities of voluntary movement; and echophenomena. Two of five signs are required for the diagnosis except in cases of catatonia due to a general medical condition, in which only one is needed (1). While no duration criterion is specified, most investigators accept that features must

be present for at least 1 hour and must occur on two or more occasions.

While mutism and stupor are often considered the principal signs of catatonia, neither is pathognomonic. Other commonly occurring symptoms include negativism manifested by resistance of movement and instructions; posturing, including facial postures; and waxy flexibility characterized by an initial resistance to an induced movement before gradually allowing repositioning. Another common feature is echolalia, which can manifest as parrot-like repetition of the examiner's vocalizations and as echopraxia (the spontaneous repetition of the examiner's movements). Motor behavior is often repetitive, nongoal-directed, and not influenced by external stimuli. Less common features include automatic obedience, mannerisms, ambitendency, and robotic speech. While a number of catatonia rating scales exist, the Bush-Francis Catatonia Rating Scale is considered the gold standard for research and clinical use (2, 3).

Epidemiology

Catatonia occurs in approximately 5% of hospitalized patients. Almost half of patients with catatonia suffer from an underlying mood disorder, predominately bipolar disorder. An additional 10%–15% of patients with catatonia meet criteria for schizophrenia (1). Cataplexy, mannerisms, posturing, and mutism are the features most often present in this subpopulation. Catatonia has been described in children, adolescents, and pregnant women.

Malignant catatonia, Ms. P's diagnosis, was described in the medical literature long before the introduction of antipsychotic medications. It is characterized by the acute onset of excitement, delirium, fever, autonomic instability, and cataplexy. Patients with this disorder look as if they have a fulminant infectious disease. Until the advent of ECT, most such patients died. Malignant catatonia is considered a medical emergency, with reported death rates of up to 20% and frequent sustained morbidity (4). Potential significant medical complications include aspiration pneumonia, pulmonary embolism, urinary retention, decubitus ulcers, and deep-vein thrombosis (5, 6). The term "neuroleptic malignant syndrome" has been applied when the condition is associated with exposure to antipsychotic drugs. Up to 1% of patients treated with antipsychotic medications develop neuroleptic malignant syndrome, usually within the first 2 weeks of exposure (4).

Differential Diagnosis

The differential diagnosis of catatonia includes a number of neuropsychiatric illnesses that present with catatonic signs. Elective mutism, akinetic Parkinson's disease, locked-in syndrome, stiff-person syndrome, malignant hyperthermia, brainstem disease, stupor due to metabolic derangement, nonconvulsive status epilepticus, and conversion disorder are some of the conditions that can be a challenge to distinguish from catatonia (4, 7). These conditions have different associated pathology and laboratory findings and require different clinical approaches for successful treatment. Catatonia has also been reported in response to antihistamine overdose and withdrawal from levodopa, amantadine, benzodiazepines, clozapine, and anticonvulsants (7).

Malignant catatonia and neuroleptic malignant syndrome are particularly difficult to differentiate. Many researchers believe that these disorders may share a similar pathophysiology of dysregulated sympathetic nervous system activity involving alterations in neurotransmission via γ-aminobutyric acid, dopamine, and glutamate. There are no identifying clinical or laboratory characteristics that distinguish the two. Thus, the sequence of clinical symptoms, the degree of elevation in creatine phosphokinase and WBC count, metabolic acidosis, proteinuria, and myoglobinuria are not pathognomonic for either condition. Moreover, the same treatments for the conditions yield indistinguishable outcomes. For these reasons, many researchers categorize neuroleptic malignant syndrome as a specific type of malignant catatonia (4, 6, 7).

Treatment of Catatonia

When correctly identified, catatonia can be rapidly and effectively treated. Given multiple potential etiologies and subtypes, it is not surprising that published studies of catatonia treatment overwhelmingly consist of case series and open prospective trials. The etiology, severity, and pattern of catatonic features do not appear to affect treatment outcome. The long-term prognosis appears to be most closely linked to successful treatment of the underlying condition (6).

Treatment of catatonia is a team effort best accomplished in the hospital setting. All patients require attentive nursing care to ensure adequate hydration, nutrition, mobilization, and skin care. Critically ill patients with malignant catatonia or neuroleptic malignant syndrome, such as Ms. P, urgently require treatment to reverse hyperthermia, maintain stable blood pressure and cardiac rhythm, ensure adequate oxygenation, and avoid renal failure.

Although intravenous amobarbital has long been considered useful in the treatment of catatonic mutism, only one double-blind randomized controlled trial, published over 15 years ago, found that amobarbital was superior to saline (8). Despite the absence of controlled studies, benzodiazepine treatment has become the standard of care. Nonmalignant forms of catatonia usually respond to parenteral lorazepam, at 6–20 mg/day. The medication is often administered 30–60 minutes before meals to elicit a transient period of increased responsiveness to maintain adequate food intake and hydration. Dosing is usually initiated at 3–4 mg/day and can be rapidly increased to

achieve symptom resolution. Some investigators believe that ECT should be initiated by the fifth day after inadequate response to lorazepam. In malignant catatonia, further delaying ECT is associated with increased mortality (4). In Ms. P's case, delays in obtaining the emergency guardianship precluded earlier ECT.

Two rapid catatonia treatment challenge tests have emerged in the literature. The lorazepam challenge consists of administering 1 mg of lorazepam intravenously and waiting up to 5 minutes for a response; if there is no response, an additional 1 mg i.v. of lorazepam is administered. The zolpidem challenge consists of administering 10 mg of zolpidem orally or through a nasogastric tube to achieve a plasma concentration between 80 and 150 ng/liter within 30 minutes of ingestion. Response usually occurs within 30 minutes and lasts 3 hours (9). A positive response generally predicts a more sustained response to a subsequent lorazepam trial. Nonresponse to these challenge tests does not preclude future lorazepam response, although higher doses are often necessary and concurrent preparation for ECT should be initiated.

In addition to lorazepam, a number of case reports suggest that atypical antipsychotics may relieve the motor as well as the nonspecific signs and symptoms of catatonia (10, 11). Discontinuation of antipsychotic medications, especially high-potency agents, is generally an initial treatment recommendation, particularly in cases of malignant catatonia or neuroleptic malignant syndrome. While all antipsychotics can induce neuroleptic malignant syndrome, a number of case reports suggest that atypical antipsychotics, including risperidone, olanzapine, and ziprasidone, can be effective in short- and long-term treatment of catatonia (10, 11).

The belief that neuroleptic malignant syndrome is an idiosyncratic response to acute dopamine blockade led to the pharmacologic treatment strategy of using pre- and postsynaptic dopamine receptor agonists in conjunction with the muscle relaxant dantrolene sodium. Generally, intravenous treatment with dantrolene is initiated for the first 48 hours, with the addition of amantadine and/or bromocriptine for absent or incomplete response. Concerns about this approach include delayed response, the risk of liver toxicity at higher doses of dantrolene, and the risk of aggravating psychosis with bromocriptine (4).

Case series and open prospective trials suggest that ECT is a highly effective treatment for all forms of catatonia, including malignant catatonia and neuroleptic malignant syndrome. The literature also supports the use of ECT for malignant catatonia in children, pregnant women, elderly persons, and medically compromised patients (12, 13). APA treatment guidelines (14) state that ECT is probably the most effective treatment for catatonic syndromes regardless of etiology. They further suggest that ECT should be considered in patients with schizophrenia and prominent catatonic features as well as in patients with bipolar disorder with catatonic features not responsive to benzodiazepines.

Bitemporal electrode placement with brief pulse currents with initial energy at half the patient's age is recommended (4). The decision to use higher-than-recommended energy settings in Ms. P's case was based on the anticonvulsive effects of the acute benzodiazepine treatment. Treatment can be administered daily in malignant catatonia or neuroleptic malignant syndrome until significant symptom resolution is achieved. Even in cases of rapid dramatic response to ECT, courses of at least six treatments are recommended to mitigate the possibility of rapid relapse (4). Treatment modifications for patients with very high seizure thresholds after high-dose benzodiazepine treatment can include use of etomidate anesthesia and the benzodiazepine antagonist flumazenil. Catatonic patients have never been included in ECT treatment trials, so recommendations regarding electrode placement, energy settings, treatment frequency, and number of treatments to prevent relapse are not evidence based.

Consent for ECT

Informed consent for ECT is a distinct process, and its application has evolved over time. The consent process includes a careful review of the procedure itself and the possible benefits, side effects, and risks of treatment; consent is also obtained for any additional interventions, such as the administration of emergency medications or procedures (such as intubation and cardioversion) should the need arise. In addition, the limitations of the treatment—an understanding that ECT is not a cure and that some form of maintenance treatment should follow a successful ECT course—must be conveyed to the patient. A discussion of the choices among various forms of electrode placement and stimulus dose takes place before the first ECT session and may require further discussion as the treatment course progresses. Recent additional recommendations by the APA Task Force on ECT include the distinction between consent for acute and for maintenance ECT, since the purpose of the treatment changes as the patient recovers (14). Given the complexity of the issues and the patient's acute cognitive and emotional distress, it is advisable to involve a family member in the process; many ECT practitioners use written materials or audiovisual presentations to supplement the clinical discussion.

In Ms. P's case, catatonic withdrawal rendered her incapable of providing a decision (either consent or treatment refusal), necessitating the appointment of a guardian. Guardianship requires legal adjudication, and the legal requirements vary by state. Authorization to consent to ECT is generally not included in medical guardianship and must be specifically petitioned of the court. Therefore, it is essential for the treatment team to be familiar with local laws to assist families in pursuing such guardianship.

Because ECT usually consists of a series of treatments during which time the consenter's (patient's) mental status or the risk-benefit balance may change, the consent

process must be seen as dynamic. The most common example of mental status change is transient cognitive or memory impairment caused by the treatment itself. In Ms. P's case, the development of torsade de pointes after the first treatment mandated a careful reevaluation, with Ms. P's sister, of the risk-benefit balance of further ECT. Informed consent for subsequent treatments was obtained as described in the case narrative.

Cardiac Effects of ECT

ECT results in significant cardiovascular effects, although they are generally transient and resolve without adverse sequelae. The initial electrical stimulus produces a vagally transmitted parasympathetic bradycardia or even asystole lasting several seconds (15). As the seizure develops, this state is followed by sympathetic stimulation originating in the hypothalamus and descending via the brainstem, spinal cord, paravertebral stellate ganglia, and cardiac accelerator nerves. Adrenal stimulation leads to an abrupt increase in catecholamine release, which lasts several minutes postictally. A variety of cardiac dysrhythmias may occur, usually after the seizure, with reported incidences ranging from as low as 8% to as high as 80% or more in patients with known cardiovascular disease (16). Sympathetically mediated dysrhythmias include sinus tachycardia, bigeminy, trigeminy, and ventricular tachycardia and fibrillation. In healthy young people, the tachydysrhythmias seen are generally brief, require no intervention, and do not preclude further ECT.

Ms. P had required pretreatment with metoprolol for tachycardia before the initial ECT, presumably because of sympathetic overactivity, as is often seen in catatonia; the seriousness of her dysrhythmia and the need for cardioversion necessitated a more vigorous reexamination of the procedure to further reduce risk in subsequent treatments.

Use of Muscle Relaxants During ECT

Succinylcholine, generally considered the drug of choice for muscle relaxation in ECT because of its rapid onset and offset of action, was administered at Ms. P's initial ECT session (17). However, this agent can cause potentially dangerous elevations in serum potassium levels in a variety of neuromuscular conditions, such as direct muscle injury, upper or lower motor neuron damage, some neuromyopathies, and toxic muscle injury (18, 19). In such situations, upregulation of acetylcholine receptors occurs, with spread of receptors beyond the endplate, leading to increased efflux of potassium as depolarization occurs. In situations of potential hyperkalemia, it is generally recommended that a nondepolarizing muscle relaxant, such as mivacurium, atracurium, or rocuronium, be used as an alternative (20).

While catatonic withdrawal is rarely identified as a possible setting for direct muscle damage, immobilization has been recognized as a risk factor for hyperkalemic states and may have been indirectly responsible for the episode of torsade de pointes that occurred during Ms. P's first ECT session. The development of foot drop likewise suggests neuronal injury and an elevated risk with the use of a depolarizing agent. While the dysrhythmia was unexpected and its cause could not be precisely identified, the fact that Ms. P demonstrated normal cardiovascular responses with a nondepolarizing agent at subsequent ECT sessions suggests that hyperkalemia may have occurred. In hindsight, the use of a nondepolarizing agent could have been chosen from the outset. This case illustrates a potential complication in clinical settings like a busy medical center. Anesthesiologists may lack extensive experience in ECT, and given the absence of specific ECT medication recommendations in the available literature on catatonia treatment, this issue can be overlooked.

Conclusions

Effective management of the catatonic patient is a complex undertaking and requires collaboration by a treatment team experienced with the illness, its complications, and the treatment modalities likely to be used. In addition, familiarity with local regulations for guardianship is often necessary in cases of a patient who fails to respond to initial treatment with a benzodiazepine and cannot provide informed consent to further treatment. ECT should be considered early in the course of the illness to improve prognosis and to avoid complications such as those seen in the patient described here. As the case illustrates, modifications in anesthesia technique should be considered simultaneously with the process of managing the patient's hydration and nutrition and working with the family on the guardianship and consent processes. As the treatment progresses, it is essential to maintain a collaborative relationship with family caregivers so that evolving problems can be effectively addressed.

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