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Catatonia After Deep Brain Stimulation Successfully Treated With Lorazepam and Right Unilateral Electroconvulsive Therapy: A Case Report

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Abstract

Objectives—The presence of a deep brain stimulator (DBS) in a patient who develops neuropsychiatric symptoms poses unique diagnostic challenges and questions for the treating psychiatrist. Catatonia has been described only once, during DBS implantation, but has not been reported in a successfully implanted DBS patient.

Materials/Methods—We present a case of a patient with bipolar disorder and renal transplant who developed catatonia after DBS for essential tremor.

Results—The patient was successfully treated for catatonia with lorazepam and electroconvulsive therapy (ECT) after careful diagnostic workup. ECT has been successfully used with DBS in a handful of cases and certain precautions may help reduce potential risk.

Conclusions—Catatonia is a rare occurrence after DBS, but when present may be safely treated with standard therapies such as lorazepam and ECT.

Keywords

electroconvulsive therapy; deep brain stimulation; catatonia

Introduction

Deep brain stimulation (DBS) is shown to be effective in cases of Parkinson's disease (PD), essential tremor (ET), tic disorders, dystonia, chronic pain, obsessive-compulsive disorder

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(OCD), and major depressive disorder (MDD). DBS also carries significant risks and side effects, including infection, intracerebral hemorrhage, cognitive impairment, psychosis, and mood disorders. Catatonia has only been described once following DBS implantation, in the immediate post-operative phase. We present a case of catatonia occurring six months after successful unilateral DBS for essential tremor that responded to lorazepam and electroconvulsive therapy (ECT).

Case Report

A 67 year-old woman received unilateral implantation of a deep brain stimulator into the left ventralis intermedius nucleus (Vim) of the thalamus in May 2012 for treatment of lifelong essential tremor resistant to medical therapy. The stimulator was turned on in June 2012 with significant improvement in tremor in her right hand and without other neurologic complication. Her DBS device is a Medtronic Activa PC-B, contacts C+,1–, amplitude 1.5 volts, pulse width 60 μ s, and frequency 185 Hz.

Her past psychiatric history was notable for bipolar disorder diagnosed at age 17, which resulted in five total hospitalizations, the last of which was in 2010 for a manic episode. Previous medication trials included lithium, valproic acid, lamotrigine, gabapentin, ziprasidone, and lorazepam. Her past medical history was significant for hypertension, obstructive sleep apnea, subtotal parathyroidectomy for primary hyperparathyroidism in 1990, lithium-induced nephrotoxicity and end-stage renal disease treated with living related donor renal transplant in 2005, and acute pancreatitis resulting in insulin-dependent diabetes mellitus in 2011. At her last outpatient nephrology visit in November 2012 she had stable renal transplant function with creatinine of 1.5 mg/dl and therapeutic tacrolimus level of 7.5 ng/mL.

Beginning in September 2012 the patient experienced worsening depressive symptoms of low mood, psychomotor retardation, decreased appetite, decreased energy, and hopelessness. At that time she was prescribed duloxetine 30 mg daily, escitalopram 20 mg daily, and quetiapine 25 mg nightly. She was switched from duloxetine to mirtazapine 15 mg daily without benefit. On December 19 the patient's depressive symptoms became so severe that she was admitted to a local psychiatry ward for inpatient treatment of severe depression with grave passive neglect.

During initial admission her depressive symptoms continued to worsen, until she was noted on her seventh hospital day to be mute, stuporous, withdrawn, and negativistic. She did not follow one-step commands, refused oral medications (including immunosuppressive agents) and nourishment by mouth. She was transferred to a general medical floor when her creatinine reached 1.9, prompting concerns of acute kidney injury and renal transplant rejection. The patient was stabilized with intravenous fluids and tube feeds via nasogastric tube (NGT), and continued to manifest intermittent catatonic symptoms as above.

Labs at that time were notable for normal chemistries and complete blood count. Her liver function tests demonstrated elevated alkaline phosphatase of 900 U/L, but were otherwise normal; FK506 level was mildly subtherapeutic at 4.0. An electroencephalogram (EEG) showed a posterior dominant awake alpha rhythm with intermittent theta waves. CT scan of

the head demonstrated Vim DBS electrode in place and mild cerebral atrophy. B12, folate, and thyroid stimulating hormone (TSH) were normal, and treponema pallidum antibodies were negative. Her DBS device was turned off with no subsequent change in her mood or catatonic symptoms. Her psychiatric medications were continued via NGT, and methylphenidate 10 mg bid and dronabinol 2.5 mg bid were added for energy and appetite stimulation without effect.

Lorazepam challenge with 2 mg IV brought about rapid improvement in stupor and mutism within 30 minutes; the patient began speaking, interacting, and feeding herself again. Lorazepam 2 mg TID was instituted, but significant sedation on this regimen led to transfer to a geriatric psychiatry ward at a tertiary care center for consideration of electroconvulsive therapy.

At the geriatric psychiatry ward concern for risk of harm to the patient from ECT with DBS in place prompted further medication trials: all psychotropics were discontinued and methylphenidate 10 mg bid and dronabinol 2.5mg bid were restarted. Her Bush-Francis Catatonia Rating Scale (BFCRS) score was 9 (3 for mutism, 3 for stupor, 3 for withdrawal). On the fifth hospital day the patient experienced a witnessed generalized tonic-clonic seizure and was admitted to the neurology service. An exhaustive workup for organic causes of seizures and catatonia in an immunosuppressed patient, including serum electrolytes, complete blood count, chest x-ray, ceruloplasmin, vitamin B levels, ANA, CRP/ESR, NH3, HIV, hepatitis panel, serum lactate, lumbar puncture with viral and bacterial assays, cytology, oligoclonal bands, cell count, total protein and glucose, 14-3-3 protein, paraneoplastic antibody panel, parathyroid hormone levels, and CT scan of the brain/chest/abdomen/pelvis, were unrevealing. Magnetic resonance imaging (MRI) of the brain was not obtained due to the presence of the DBS. EEG initially showed right-sided slowing, but on repeat testing was without abnormality. Urinalysis detected a urinary tract infection and this was treated without change in condition. Alkaline phosphatase (AP) was elevated between 300 and 1000 U/L, thought due to medication effect and renal insufficiency. Quantiferon test for tuberculosis was positive but induced sputum cultures were negative. Renal transplantation consultation concluded that the patient's tacrolimus was not the cause of the current catatonic symptoms as it had been tolerated well at the current dose for years, there had been no supratherapeutic levels recently, and there were no other symptoms of tacrolimus toxicity such as tremor or headache.

The patient worsened during the first week on the neurology service, becoming completely mute, stuporous, and withdrawn, with sinus tachycardia. Percutaneous gastric tube was placed and tube feeds were initiated. Methylphenidate and dronabinol were discontinued. Rechallenge with lorazepam 2 mg IV brought about immediate improvement in catatonic symptoms, and the patient was interactive but sedated on 1.5 mg BID, with poor PO intake, psychomotor retardation, low energy, and depressed mood. When no etiology for her seizure besides medications (stimulant toxicity or benzodiazepine withdrawal) was found, the patient was transferred back to the geriatric unit for definitive treatment of catatonic depression with ECT.

Precautions taken before the first session included ensuring DBS was off with voltage set at zero; consultation with neurology, a Medtronic representative, and high-volume ECT and DBS centers for second opinions; pre- and post-ECT imaging to ensure lead placement; and palpation of the IPG, cable, and electrode cap before each session. Right unilateral electrode placement was chosen as it was felt to put the greatest distance between the ECT electrodes and the left frontal DBS hardware. Induction and modification were achieved with methohexital 50 mg and succinylcholine 50 mg before each treatment. Seizure lengths ranged from 21 to 112 seconds. ECT stimulus parameters were: current 900 mA; energy 30–50%; pulsewidth 0.25 ms; frequency 50–70 Hz; stimulus duration 6.71–7.99 seconds. The patient received twelve treatments of right unilateral ultra-brief pulse ECT with a Thymatron System IV without complication. She started to show clinical improvement after the second treatment, as evidenced by increased oral intake, spontaneous speech, and activity levels. Escitalopram was discontinued and lamotrigine 25 mg daily and aripiprazole 5 mg daily were added to her medication regimen for mood stabilization when she began to display a more labile affect and loose associations around the fourth ECT treatment. By the tenth ECT treatment she was ambulating, eating, taking medications independently, and coming out of her room to socialize in the milieu. Mood was euthymic and BFCRS score was 0. She was discharged to home and the ECT service recommended continuation ECT at tapering frequencies which she has tolerated without complication. Of note, the patient's tremor was noticeably improved after ECT with DBS device still off.

Discussion

Catatonia is a psychomotor syndrome associated with diverse medical, neurological, and psychiatric conditions, and is characterized by mutism, stupor, verbigeration, negativism, echophenomena, and motoric abnormalities such as catalepsy, stereotypies and mannerisms.¹ Catatonia has been reported only once in association with DBS, in which electrode implantation caused intracerebral hemorrhage and immediate catatonic symptoms that responded temporarily to lorazepam.² In our case the patient's catatonia occurred six months after successful deep brain stimulation implantation in the context of worsening depressive symptoms and transplant-related immunosuppression. Because neural circuits that modulate mood and cognition lie in close proximity to intracerebral DBS targets, explicating the cause of neuropsychiatric symptoms in a patient with DBS must take into account factors such as pre-implantation conditions, peri-operative and device complications, acute stimulation effects, medication adjustments, and chronic stimulation effects.³ Potential pre-implantation contributors to our patient's catatonic syndrome included pre-existing bipolar disorder, tacrolimus use, and her progressive movement disorder. She did not experience intraoperative or perioperative complications of DBS implantation. The DBS was interrogated upon admission with no evidence of malfunction of the IPG or hardware. Turning off the device and allowing for "washout" of stimulation effect for over four weeks did not yield any improvement in her catatonic depression. Discontinuation of antidepressants, mood stabilizers, and antipsychotics out of concern for exacerbation of catatonia did not result in improvement. Her one-time generalized tonic-clonic seizure was most likely a result of stimulant toxicity and benzodiazepine withdrawal and not related to the DBS or a seizure disorder.

It was not possible to rule out a chronic stimulation-related effect of her DBS, which led us to consider whether DBS removal would be beneficial. Influential factors in the final decision to keep the DBS in place included its established benefit for the patient's tremor; the risks of a second neurosurgery and exposure to general anesthesia; and the lack of guarantee that DBS removal would ameliorate her depression and catatonia. A literature search and communication with a high-volume DBS center did not uncover any cases or recommendations of DBS removal for psychiatric reasons. After discussion about treatment options with the patient, family, neurology, and psychiatry, the decision to proceed with electroconvulsive therapy for catatonic depression was made.

ECT in DBS

The theoretical risk of using ECT in a DBS patient is described on the Medtronic website as the "possibility of electrical impulses from the treating electrodes straying into the generator via the extender cable."⁴ Reports describing neurological injury from electromagnetic energy transmitted to DBS electrodes by cardioversion and diathermy form the basis for the manufacturer's warning.⁵⁻⁷

Since 2000 there have been a total of six published reports of successful and safe ECT in DBS patients (see Table 1).⁸⁻¹³ In each case ECT was indicated for exacerbations of major depressive disorder. Patients treated have all been female except for one male, between the ages of 56 and 78, and had PD, ET, or MDD as a reason for DBS implantation. DBS electrode location was subthalamic nucleus in four cases, ventral intermediate nucleus of the thalamus in one case, and subcallosal cingulate gyrus in one case. Side effects from ECT were typical for the stimulus techniques used, including post-treatment confusion and temporary short-term memory impairments. There were no significant complications of ECT or DBS in any of the cases.

Precautions described by the authors to avoid electromagnetic energy transmission to the DBS included the following: 1) ensuring indications for ECT were rigorously met; 2) turning off the DBS device prior to ECT; 3) setting voltage to zero in case the DBS was inadvertently turned on; 4) employing ECT electrode placement to maximize distance from DBS hardware; 5) palpation of DBS electrode, cable, and IPG to avoid direct contact with ECT electrodes; 6) and pre- and post-ECT course brain imaging to rule out DBS lead migration. Ducharme et al demonstrated that it is possible to safely turn on DBS between ECT treatment sessions, as long as it is turned off again before the next stimulus delivery.¹³ The experience on our service also supports the safety in treating a patient who has had DBS recently removed prior to ECT.

Conclusion

With the increasing likelihood of psychiatrically ill patients having a DBS device in place, the use of ECT in DBS patients will become more common. Catatonia may occur in DBS patients and requires a careful and informed differential diagnosis to determine the etiology and appropriate treatment. Our case adds to the literature by demonstrating that lorazepam

and right unilateral ultra-brief pulse ECT are safe and effective in a catatonic patient with left Vim DBS.

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Table 1

Published reports of successful ECT in DBS patients.

Authors	Year	Age/Sex	DBS	Location	Psych Dx	Delivery	Energy	mA	PW (ms)	Hz	Duration (s)	Sz Length (s)	# ECT
Moscarillo et al	2000	68F	ET	Vim (L)	MDD w Anxiety	BT	70–100%	NR	NR	NR	NR	15–40	8
Chou et al	2005	66F	PD	STN (B)	MDD w Psych Fx	BF	45–75%	0.8	1–1.4	60–90	2	21–48	9
Bailine et al	2008	78F	PD	STN (L)	MDD w Anxiety	BT	40%	0.9	1	50	2.2	45–87	7
Puigdemont et al	2009	64F	MDD	SCG (B)	MDD w Psych Fx	BF	70%	NR	NR	NR	NR	NR	9
Ducharme et al	2011	71M	PD	STN (B)	MDD	BF	NR	0.8	1	40	1.25–3.5	NR	17
Nasr et al	2011	56F	PD	STN (B)	MDD w Psych Fx	BT	100%	0.8	1	NR	4	27–76	5

F = female; M = male; Vim = Ventral intermediate nucleus; STN = subthalamic nucleus; SCG = subgenual cingulate; MDD = major depressive disorder; Fx = features; BT = bitemporal; BF = bifrontal; mA = milliamperes; PW = pulsewidth; ms = milliseconds; s = seconds; Sz = seizure