Disclosure

The authors report no conflicts of interest.

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New-onset panic attacks after deep brain stimulation of the nucleus accumbens in a patient with refractory obsessive-compulsive and bipolar disorders: a case report

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New-onset panic attacks (PA) have been described in patients with obsessive-compulsive disorder (OCD) receiving deep brain stimulation (DBS), mostly during the intraoperative period or a few weeks after device implantation.[1,2] We report the case of a 39-year-old, right-handed man with severe treatment-refractory OCD and bipolar disorder type I (BD-I), beginning at age 17 (without any other psychiatric disorder), who developed late-onset PA after DBS implant placement.

The patient presented with obsessions of doubt, cleaning, and disgusting thoughts accompanied by checking and cleaning compulsions, with an intense need for reassurance and avoidance. Due to poor response to multiple drugs and to cognitive-behavioral therapy (Table 1), the patient underwent surgical evaluation for DBS. Implantation was performed after the patient and relatives had signed an informed consent form and following authorization from the Federal Council of Medicine. At baseline, the Yale-Brown Obsessive Compulsive Scale (Y-BOCS) score was 36[3] and the Beck Depression Inventory (BDI) score was 35.[4]

Bilateral DBS electrodes were inserted through the anterior limb of the internal capsule into the nucleus accumbens (NAcc) near the anterior commissure (Figure 1).

Table 1 Medications previously taken by the patient

Medication	Maximum dose (mg)	Duration
Clozapine	400	15 years
Fluoxetine	80	14 years
Valproate	2000	3 years
Lithium	1200	16 years
Clomipramine	250	3 years
Sertraline	200	2 years
Paroxetine	80	1 year
Fluvoxamine	300	6 years
Citalopram	60	7 months
Haloperidol	5	6 months
Risperidone	6	3 years

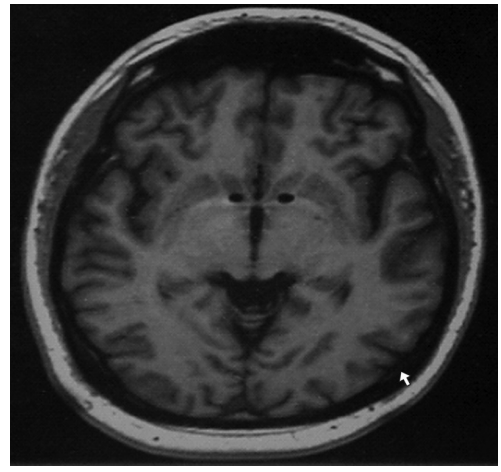


Figure 1 Magnetic resonance imaging scan showing the deep brain stimulation electrodes (Medtronic model 3387) inserted bilaterally through the anterior limb of the internal capsule into the nucleus accumbens near the anterior commissure. Cartesian coordinates of the distal end of the deepest contact relative to the mid-commissural point were: left and right: 6 mm lateral to midline, 3 mm anterior to mid-commissural point, and in the anterior commissure-posterior commissure plane.

Intraoperative evaluation of the DBS electrodes was carried out using bipolar stimulation at each contact. Pulse width and stimulation frequency ranged from 90 to 210 μs and 100 to 180 Hz, respectively. Voltage varied between 0 and 4 V, while bilateral stimulation was 3+/0-, 3+/1-, 3+/2-, and 0+/3-. The patient did not notice any change in mood or anxiety during stimulation. Testing occurred for approximately 2 to 4 minutes at each setting and the voltage was turned off before testing each contact. The patient was discharged from the hospital with the DBS regulated at 4.2 V, 150 μs, 150 Hz both sides, LL 3+, zero and 1 Neg, RR 7+, 4 and 5 Neg. Final adjustment was performed after several trials with on-off checking. Five months after surgery, the patient had experienced significant improvement of both OCD (Y-BOCS = 17) and depression (BDI = 9). Suddenly, within 12 hours of a follow-up visit involving a parameter adjustment for better control of OCD symptoms (4 V, 180 μs, 120 Hz both sides, LL C+, zero and 1 [-], RR C+, 4 and 5 [-]), the patient began to have

severe panic attacks, which were controlled after new adjustments in association with clonazepam 1 mg/day. The adjustments involved more ventral connectivity with bipolar stimulation, instead of a dorsal stimulation, and were performed because they elicited better OCD control, but possibly triggered PA. The device was turned off; however, due to patient request, it was immediately reset to the previous settings, thus limiting conclusions of causality.

Shapira et al. and Okun et al. only observed the occurrence of panic attacks by activating the most ventral contact that is located next to the NAcc.[1,2] When this region was stimulated at contact zero, it probably caused amygdala activation, thus evoking the experience of panic.[1,2] This may have occurred because of the role of the NAcc as an interface for limbic projections from the amygdala, hippocampus, and cingulate cortex, which receives input from dopaminergic-containing nuclei, while mediating the behavioral and affective changes induced by DBS.[2,5] Additionally, the patient's comorbid BD-I could have facilitated affective side effects with NAcc stimulation.

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