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 and the Beck Depression Inventory (BDI) score was 35.

Table 1 Medications previously taken by the patient

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

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

Marcelo B. Sousa, 1 Telmo Reis, 2 Alexandre Reis, 2
Paulo Belmonte-de-Abreu 1,2,3

1 Psychiatry Service, Hospital de Clínicas de Porto Alegre (HCPA),
Porto Alegre, RS, Brazil. 2 Neurosurgery Service, Hospital Moinhos
de Vento (HMV), Porto Alegre, RS, Brazil. 3Department of Psychiatry, School of Medicine, Universidade
Federal do Rio Grande do Sul (UFRGS), Porto Alegre, RS, Brazil


Disclosure
The authors report no conflicts of interest.

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