

# Basal ganglia hemorrhagic ablation associated with temporary suppression of obsessive-compulsive symptoms

## Ablação hemorrágica dos gânglios da base associada temporalmente à supressão dos sintomas obsessivos-compulsivos

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**Abstract** Currently, basal ganglia (BG) are considered regulators of motor and emotional activity. It's operability encompasses Obsessive Compulsive Disorder (OCD). The case of a patient suffering with severe OCD is described of note, his symptoms disappeared following a hemorrhage of the left BG. However, once the hemorrhage was reabsorbed his symptoms returned. It is possible that lesions affecting cerebral OCD association circuits may influence the evolution of obsessive-compulsive symptoms.

**Keywords** Obsessions. Compulsions. Brain Hemorrhage. Basal Ganglia.

**Resumo** Atualmente, os gânglios da base (GB) são considerados reguladores da atividade motora e emocional e seu funcionamento está envolvido com o Transtorno Compulsivo Obsessivo (TOC). Este relato descreve um caso de um paciente com diagnóstico de TOC, cujos sintomas remeteram após hemorragia nos GB à esquerda. No entanto, após a reabsorção do processo hemorrágico, os sintomas reapareceram. É possível que lesões que afetem os circuitos cerebrais associados ao TOC possam influenciar na evolução dos sintomas obsessivos compulsivos.

**Descritores** Obsessão. Compulsão. Hemorragia Cerebral. Gânglios da Base.

### Introduction

Historically, the basal ganglia (BG) was considered a regulator and modulator of motor activity<sup>1,2</sup> in Parkinson's disease, and Huntington Chorea, among others. Currently, the BG is also studied as a region participating in the regulation of emotions and behaviours of psychiatric conditions<sup>3</sup> such as Obsessive-Compulsive-Disorder (OCD).

OCD presents a vast symptomatology reflected in a large clinical spectrum.<sup>4</sup> These symptoms seem to be anatomically and functionally distributed along many cerebral sites, constituting a continuum represented by neuroanatomical loops or models.<sup>5,6</sup> For instance, based on neuroimaging, neural correlates between OCD clinical symptoms and BG striatum lesions have been proposed.<sup>7</sup> Two BG pathologies seem to emerge, one is an infarction, and the other one a hemorrhage. The infarction mostly located in the putamen, and the other a hemorrhage involving the posterior limb of the internal capsule<sup>8</sup> or BG hematomas after severe head injuries.<sup>9</sup> Specifically, BG pathology is prominent in the putamen after an infarction causing complex stereotyp-

ies,<sup>10</sup> and in the head of left caudate nucleus and the anterior portions of the body of the lateral ventricle, compatible with putamen infarct.<sup>11</sup> Moreover, in the putamen, heads of the caudate nuclei, and internal capsule<sup>12</sup> may cause<sup>17</sup> or aggravate OCD, as reported in bilateral BG lesions in adults.<sup>14</sup> Also a decreased cerebral blood flow in the right BG after a right inferior parietal infarct was reported.<sup>15</sup> In general, neuropsychiatric pathology of GB is ample, and can be observed in other clinical conditions such as in one case of Creutzfeld-Jakob disease;<sup>16</sup> hydrocephalus;<sup>17</sup> and corpus callosum agenesis and hydrocephalus<sup>18</sup> to name a few. Others have identified OCD clinical symptoms related to anatomical structure damage located in different parts of the brain.<sup>19</sup> Therefore, evidence supporting that OCD symptoms convey the results of a variety of lesions located in different parts of the brain or subcortical regions have been shown, i.e., the occupancy of cerebro-vascular accidents or tumors,<sup>19</sup> traumatic head injury,<sup>20</sup> and epilepsy.<sup>21</sup>

This is the history of a patient who has signed authorization to present his case.

### Case presentation

This is a 33-year-old married male, who has suffered from OCD since age 26. At age 29, he developed essential hypertension, but refused to be treated on account of serious difficulties to swallow tablets (phobia to swallow and choke), except for sertraline 200 mg/day/po which he took for three years. His hypertension worsened to the point that he was warned that serious complications, such as brain hemorrhage, could ensue. All along he suffered from fears of germ contamination, obsessions with harm related to his family suffering from serious illnesses or death, and aggressive behavior. Three years later, following the clinical diagnosis of arterial hypertension he sustained a brain hemorrhage. An MRI has shown a left basal ganglia hemorrhage extending into the left corona radiata and centrum semiovale, with compression of the left lateral ventricle and minimal rightward shift and with wallerian degeneration of the left cortical spinal tract. Therefore, he developed a left hemiplegia with aphasia. An electroencephalogram showed a focal slowing in the left fronto-temporal region, along with rare sharp waves discharge. These findings were indicative of structural damage to the left hemisphere.

As a consequence of this cerebral accident his obsessive-compulsive symptoms were absent. However, as a progression of the clot retraction began gradually to take place, his obsessive-compulsive symptoms considerably returned. We understand that the patient has three cerebral MRI's, that proved a retraction of the clot with a return of his OCD symptoms. Unfortunately, no psychological assessment to measure his OCD was taken, until he came to our facility.

It is at this point that he came for a consultation. We were able to review the past history. As previously stated, psychological assessment for OCD was not done. Therefore, the observation that his OCD symptoms disappeared or were substantially reduced, was bonafide.

Upon examination, main symptoms included hemiplegic gait, and right hemiparesis, aphasia, anxiety, fear of contamination, fear of harming others, and compulsive spitting more than 100 times per day. The patient states he was able to smell the germs, suggesting the possibility of olfactory hallucinations, as described in organic brain syndromes or monosymptomatic hypochondriasis. He strongly refused to handle bodily secretions, out of fear of contamination. He became easily angered, and was completely dependent on his wife. His neurological symptoms were very much improved, his speech was understandable, and his motor impairment considerably diminished.

The Padua Inventory-Washington State University Revision with a score of 16 indicated the presence of OCD symptoms around contamination concerns. The Yale-Brown Compulsive Scale<sup>22</sup> yielded a score of 27 (13 for obsessions and 14 for compulsions), which is relatively high. The Beck Anxiety Inventory<sup>23</sup> noted the lack of anxiety with a score of six. On the Beck Depression Inventory-II<sup>24</sup> he scored an 11 indicating mild depression. The Overvalued Idea Scale<sup>25</sup> suggested moderate overvalued ideas with a score of 5.6 (range 1 to 10).

Treatment consisted of cognitive and behavior therapy. He accepted to take sertraline 200 mg/day/per os. Patient refused to go for a brain MRI and neuropsychological testing. He was moderately adherent to treatment. Finally, after two months of therapy, he decided to discontinue treatment.

### Discussion

We would like to discuss two aspects of our patient's symptoms: his hemorrhage, and his behavior in response to the lesion.

To our knowledge, this is the first case reported in the literature of a patient with OCD whose symptoms significantly disappeared following a left BG hemorrhage of the left putamen. BG lesions associated with OCD are located in the left putamen,<sup>11-13</sup> or presenting bilateral BG lesions causing stereotyped activities with obsessive and compulsive behavior.<sup>14</sup> Our review of Medline from 1966 to 2001 for BG and hemorrhage showed 907 cases without obsessive-compulsive sequelae. The sample of 37 patients that sustained severe traumatic BG hematoma (TBGHs) failed to report OCD pathology.<sup>9</sup> Another sample reported that out 1,036 intracerebral hemorrhage, 140 patients presented left BG hemorrhage with aphasia, a similar finding to our case (Liang et al, 2001), but without a report of OCD pathology. Therefore, the likelihood that BG hemorrhage may cause OCD is minimal or nil, unless the reviewed reports emphasize cerebral damage or neurological lesions, without accounting for psychiatric symptoms.

Overall, the pathophysiology of these lesions reported considerable oxygen deprivation, as the result of severe structural damage.<sup>14</sup> Therefore, when the hemorrhage is reabsorbed, better irrigation is established and symptom decrement may be observed. Our case, unusual by experiencing an unplanned ablation reminds us of two anecdotal reports. One is a patient with severe obsessive illness who sustained a severe head injury. As a consequence of his injury, the patient developed a left prefrontal subdural haematoma. Upon his recovery the obsessional symptoms disappeared.<sup>26</sup> The other case is a man who attempted suicide by shooting his head over the left temple, causing a left frontal lobe damage. Although no frontal lobe syndrome emerged, his obsessive rituals were significantly reduced.<sup>27</sup> The connection of these three cases, associated with OCD, indicates that specific cerebral sites affected by a given lesion may ablate "therapeutically" a pre-existing disorder, that is OCD. The intriguing aspect of these three cases is the suppression of obsessive-compulsive symptoms, in clear contradiction with the expectancy of aggravated symptomatology after structural damage, as observed in the rest of the cases. Furthermore, because lesions are located in the left basal ganglia region, while the other two in the left frontal region, suggests that lesions affect association circuits linking neuronal columns of the BG and the prefrontal area, regardless of a specific anatomical location within the operational circuit, may modify obsessive-compulsive symptomatology.

The second aspect of interest was that our patient's behavior was devoid of anxiety and depression. There was a certain degree of apathy and indifference to his OCD. This atti-

tude and behaviour is consistent to similar descriptions of other patients suffering from OCD with important cerebral organicity.<sup>19</sup>

Finally, a discreet caveat may indicate one single case is not enough to hypothesize that a cerebral lesion of the putamen may explain the changes in OCD symptomatology.

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