IMAGEM EM NEUROLOGIA/IMAGE IN NEUROLOGY

Glioblastoma as an Advanced Parkinson's Disease Mimicker Glioblastoma como Mimetizador de Doença de Parkinson Avançada

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Recebido / Received: 2021-10-17 Aceite / Accepted: 2021-01-25 Publicado / Published: 2022-04-07 A 62 year-old caucasian man with advanced Parkinson's disease (PD) beginning at the age of 39, presenting a akinetic-rigid subtype and right predominance, was submitted to bilateral deep brain stimulation (DBS) of the subthalamic nuclei (**Fig. 1a**) at 51 with significant clinical improvement, although with no effect in the pre-existing dysarthria.

In the last years, he had been progressively worsening in language (dysarthrophonia), balance (postural instability, with need of a walking aid) and cervical dystonia, scoring 28 in part III of UP-DRS in the last Neurological consultation (March 2021). Moreover, the stimulation parameters for DBS were set to $3.5V/90\mu s/160Hz$ on the left and $3.2V/60\mu s/160Hz$ on the right side (voltage/ impulse duration/impulse frequency) and he was medicated with levodopa/benserazide 250/25 mg V_4 -tablet tid and pramipexole 1.05 mg id.

In April 2021, he presented in the emergency room with a 2 weeks period of severe clinical deterioration with behavioral changes, anarthria and impossible gait. His neurological examination additionally showed global aphasia, right homonymous hemianopsia, right central facial palsy and ipsilateral pyramidal syndrome, on top of the previously known, although aggravated, right akinetic-rigid parkinsonian syndrome, ipsilateral pisa syndrome and cervical dystonia.

The computed tomography (CT) scan disclosed an intra-axial lesion in the left hemisphere with solid and cystic components, displaying peripheral contrast enhancement and edema and causing a significant mass effect and



Figure 1. Figure 1a: DBS electrodes in the subthalamic nuclei, in the post-op CT scan. **Figure 1b:** Left fronto-temporo-parietal cystic lesion, with peripheral contrast enhancement and mass effect, causing a significant displacement of the DBS electrodes.

shift of the DBS electrodes (**Fig. 1b**), which remained operational although with elevated impedance in 2 non-stimulated contacts on the right electrode.

The extra-cranial extensions of the DBS system were substituted, with subsequent normalization of the impedance values, allowing the performance of a brain MR (**Fig. 2**) and



Figure 2. Large expansive cystic-necrotic lesion centered in the left temporal lobe, with intense and heterogeneous contrast enhancement and augmented perfusion, producing a significant shift in the DBS electrodes. The images are compatible with a neoplastic lesion, probably in line with a highgrade glioma (T1-weighted image with gadolinium contrast).



Figure 3. Post-op CT scan showing no midline deviation and minor displacement of the DBS electrodes. The surgical site displays a heterogeneous peripheral contrast enhancement, probably related to post-op inflammatory phenomenons or tumor remnant.

confirmation the previously found neoplasm, which was surgically removed (**Fig. 3**) and disclosed a glioblastoma, WHO IV.

Hence, we want to point out a mimicker of a long standing Parkinson's disease submitted to DBS. This phase of PD evolution typically shows a progressive deterioration in language and gait, associated with cognitive/psychiatric symptoms, all of them non-responsive to DBS adjustments, frequently requiring dopaminergic medication and non-pharmacological interventions, such as physical and speech therapy.¹

However, a rapid clinical deterioration in chronic DBS patients should make us consider several scenarios: stimulation parameters or pharmacological maladjustment, DBS system dysfunction, primary disease progression and, at last, the appearance of a new pathology.² Additionally, the development of atypical symptoms (such as aphasia and right pyramidal syndrome in our patient) points to the last option, requiring an immediate imaging study.²

The appearance of malignant glial tumors in chronically stimulated patients is rare (only 4 cases reported in the literature) and probably under-diagnosed, considering the global epidemiology of DBS patients and glial tumors. The relation between these two pathologies has been extensively investigated and no causality effect was found so far, although further studies are required.^{2,3}

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