Case Report

Venous Infarction Following Deep Brain Stimulation of The Subthalamic Nucleus

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Abstract

Deep brain stimulation has become a popular treatment modality for medically resistant movement and neuropsychiatric disorders. As the procedure becomes more widely used, complications are being more clearly understood. Postoperative venous infarction is one of the newly introduced complications. We present a postoperative venous infarction case of deep brain stimulation of subthalamic nucleus, used in an advanced Parkinson's disease patient. Conservative approach was successful in resolving the signs and symptoms of the patient. Clinicians dealing with DBS should recognize this problem and should make intervention(s) to prevent unwanted consequences in a timely fashion.

Keywords: Deep brain stimulation, microelectrode recording, venous infarction

INTRODUCTION

Deep brain stimulation (DBS) has become a popular procedure for medically resistant movement and neuropsychiatric disorders. Venous infarction, early following DBS surgery, is a rare complication (1-6) (Table 1). In this case presentation, we describe a patient with Parkinson's disease who was treated with DBS of subthalamic nucleus and had venous infarction following the treatment.
### Table 1. Patients that developed cortical infarction following treatment with deep brain stimulation

<table>
<thead>
<tr>
<th>Author</th>
<th>Year</th>
<th>Patient age/ gender</th>
<th>Duration of primary disease (year)</th>
<th>Medical conditions</th>
<th>Nucleus</th>
<th>Signs and symptoms</th>
<th>Onset of the signs and symptoms (on postop day)</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Schwalb</td>
<td>2001</td>
<td>45-86 (one of the three patients) / N/A</td>
<td>N/A</td>
<td>Tremor</td>
<td>Left VIM</td>
<td>Right upper extremity weakness</td>
<td>0</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
<tr>
<td>Umemura</td>
<td>2003</td>
<td>68 / N/A</td>
<td>N/A</td>
<td>Parkinson’s disease</td>
<td>Left side (nucleus unknown)</td>
<td>Mild hemiparesis and seizure</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Binder</td>
<td>2005</td>
<td>75/F</td>
<td>N/A</td>
<td>Parkinson’s disease</td>
<td>Right STN</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Chang</td>
<td>2011</td>
<td>55/F</td>
<td>6</td>
<td>Parkinson’s disease</td>
<td>Right STN</td>
<td>Tonic-clonic seizure</td>
<td>0</td>
<td>N/A</td>
<td>N/A</td>
</tr>
<tr>
<td>Morishita</td>
<td>2013</td>
<td>67/F</td>
<td>16</td>
<td>Parkinson’s disease</td>
<td>Left STN</td>
<td>Aphasia, decreased consciousness</td>
<td>1</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>50/M</td>
<td>12</td>
<td>Parkinson’s disease</td>
<td>Right STN</td>
<td>Confusion, decreased consciousness</td>
<td>1</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>60/M</td>
<td>16</td>
<td>Parkinson’s disease, hypertension</td>
<td>Right GPi</td>
<td>Confusion, lethargy, left hemiparesis</td>
<td>1</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
<tr>
<td></td>
<td></td>
<td>58/M</td>
<td>7</td>
<td>Parkinson’s disease, obstructive sleep apnea</td>
<td>Left GPi</td>
<td>Confusion, abulia, expressive aphasia</td>
<td>1</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
<tr>
<td>Present case</td>
<td>2014</td>
<td>72/M</td>
<td>11</td>
<td>Parkinson’s disease</td>
<td>Left STN</td>
<td>Headache, somnolence, and dysarthria</td>
<td>2</td>
<td>Conservative</td>
<td>Recovery</td>
</tr>
</tbody>
</table>

**Abbreviations:** F: Female, M: Male, GPi: Globus pallidus internus, STN: Subthalamic nucleus, VIM: Ventral intermediate nucleus, N/A: Not available.
CASE PRESENTATION

A 72-year-old man was admitted to an outpatient clinic due to advanced Parkinson's disease (PD). He had the disease for 11 years. He had been on L-dopa therapy; however his response to the medical therapy had regressed year-by-year. Following evaluations with neuropsychiatric tools, we scheduled DBS surgery for subthalamic nucleus (STN), bilaterally. During the operation, following to local anesthesia, both microelectrode reading (Alfa Omega Micro Recording System, Nazareth, Israel) and macro-stimulation tests were performed. The coordinates of subthalamic nucleus were confirmed and DBS electrodes (St. Jude Medical 6149 40 cm lead, Minnesota, USA) were implanted. After cranial incisions were closed, general anesthesia was induced. The pulse generator (St. Jude Medical Libra XP 6644, Minnesota, USA) was inserted into the subcutaneous pocket over pectoral muscles and the system was connected with the cranial part of the system. Position of the implanted leads was confirmed to be accurate on immediate post-operative computed tomography (CT) and no pathological conditions were observed elsewhere. On post-operative 1st day, impulse generator was turned on and pre-operative drug therapy was started in the same dosage. He was discharged to home. Post-operative 2nd day, the patient was readmitted to the emergency room with headache, somnolence, and dysarthria. An emergency brain CT depicted an area of venous infarction in the left frontal subcortical region (Figure 1). We internalized and followed up the patient. Patient got better day by day with conservative treatment regimen consisting of head elevation, adequate hydration of the patient and physical rehabilitation. He was discharged on 12th day of readmission without any neurological deficit.

Figure 1: Emergency brain CT shows infarction around the lead in the left frontal region
DISCUSSION

Deep brain stimulation of STN and Globus Pallidus internus (GPi) for PD has become popular against ablative neurosurgical procedures such as thalamotomy and pallidotomy. Deep brain stimulation causes functional and reversible changes in the nucleus. Structural changes in brain parenchyma are related with gliosis (6). Despite its usefulness, DBS procedure has some per-operative, early and late onset post-surgical complications.

Entry site for microelectrode recording instruments and DBS leads is in the frontal associative cortex. Subtle frontal dysfunction might be noticed in some rare cases due to sacrifice of dural venous plexuses and cortical bridging veins. Further, this might lead to venous statis and hence infarction (4). Using high-resolution contrast enhanced MRI or CT in pre-operative planning makes entry zones and lead trajectories identified safely(1,4,6). In the presented case, pre-operative planning was made accordingly and the burr-holes were opened away from the venous structures. According to a multicenter study, risk of hemorrhage increases with increased number of microelectrode passes, which could have led to the venous infarction in our patient (7).

Clinical course of patients with venous infarction is usually benign and is successfully treated with conservative approach (1,3,5). Elevation of the head, use of anticonvulsants (if hemorrhage is identified), adequate hydration of the patient and physical rehabilitation as soon as possible following diagnosis are mandatory to facilitate clinical improvement. New neuroimaging studies should be done, whenever new neurological deficits are noticed.

In conclusion, venous infarction is one of the avoidable complications of DBS surgery. Despite subacute nature of the infarction, it can be managed with proper conservative approach. Every attempt should be made to prevent it, especially with avoidance of increased numbers of microelectrode passes.

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