

LETTER TO THE EDITOR**TO THE EDITOR****Delayed Stroke in Globus Pallidus Internus Deep Brain Stimulation**

Keywords: Complications, DBS, Delayed stroke, Infarct, Tardive dyskinesia

Deep brain stimulation (DBS) is a common neurosurgical treatment for movement and neuropsychiatric disorders refractory to medical treatment. Common DBS targets for Parkinson's disease include subthalamic nucleus, globus pallidus pars interna (GPi), and ventral intermediate nucleus of the thalamus.

Known complications following deep brain stimulator placement include infection, intracranial and intraventricular hemorrhage (IVH), system failure, seizures, stroke and, depending on the location of stimulation, cognitive, behavioral, and motor complications.¹ There are no prior reports of delayed stroke following DBS placement.

A 59-year-old female was admitted for elective bilateral GPi DBS for tardive dyskinesia. She had an 8-year history of tardive dyskinesia refractory to medical management (tetrabenazine 37.5 mg PO HS). Her past medical history included hypertension, hyperlipidemia, diabetes mellitus insulin dependent, hypothyroidism, previous oral contraceptive pill-associated deep vein thrombosis, chronic pain, and anxiety. She was on rosuvastatin, clonazepam, amitriptyline, tetrabenazine, rabeprazole, metformin, jardiance, synthroid, sertraline, and acetylsalicylic acid (ASA).

She underwent bilateral GPi DBS. ASA was held for 5 days pre-operatively. Stage I was done awake with some remifentanyl given for positioning. The patient was not confused during any portion of the procedure. Planning was done with a pre-operative MRI. The planned trajectory did not pass through any major vascular structures or into the ventricle. We obtained O-arm spin which was merged with the pre-operative MRI and obtained coordinates for the Cosman-Roberts-Wells frame. Burr holes were done as planned, and no cortical veins were coagulated. We used three microelectrode tracts for navigation, which were adjusted by the neurophysiologist who was monitoring the recordings. We then placed the lead and confirmed positioning using intraoperative X-ray. Stage II was done under general anesthetic with prece dex and remifentanyl. Systolic blood pressure (SBP) remained within 105–140 mmHg, with no instability.

On POD#1, she was found to have a GCS of 14. She was on 4L nasal prongs with an SBP of 96. CT showed a small IVH (Figure 1A). Her Glasgow Coma Scale (GCS) declined to 12 on POD#2, and further to a GCS of 9 late in the day. She became drowsier, confused, less verbal, and developed new left-sided hemiparesis. Laboratory studies were unremarkable. Repeat CT showed bilateral caudate and right internal capsule stroke (Figure 1B). CTA showed no vascular injury or vessel occlusion. She was seen by inpatient neurology, who medically optimized her. Ultimately, she required extensive physical and occupational therapy followed by transfer to inpatient rehabilitation. She recovered well at rehab and was discharged home independently.

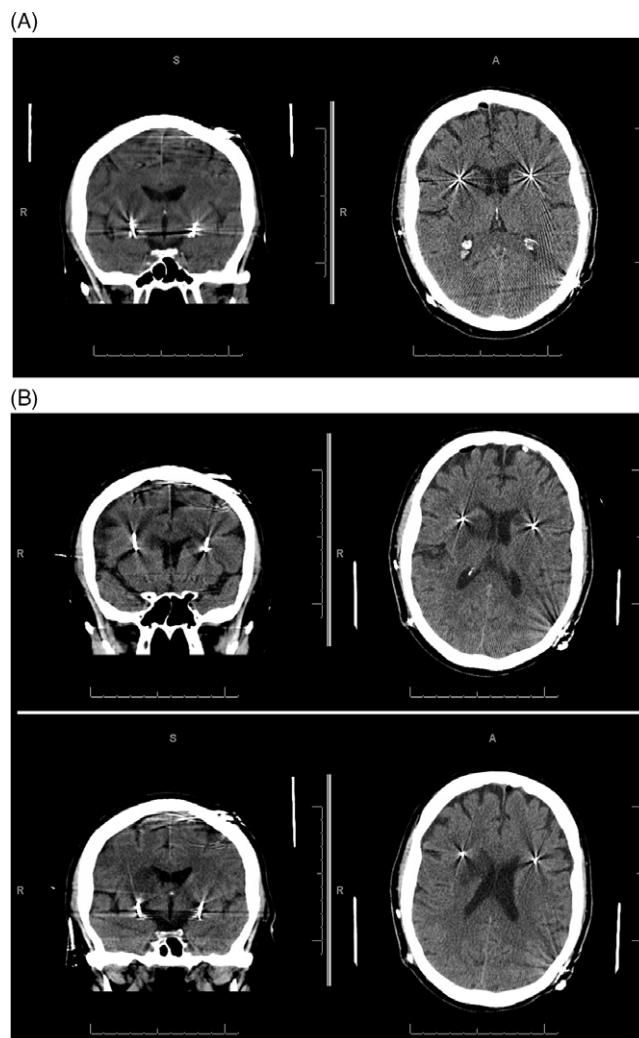


Figure 1: (A) Coronal and axial view of CT on POD1 showing a small IVH in the occipital horn of the right lateral ventricle. (B) Coronal and axial views of CT on POD2 showing new infarcts in the right internal capsule, right caudate nucleus, and left caudate nucleus.

DBS-associated infarction is rare. Six cases of hemorrhagic venous infarcts^{2,3} and 12 cases of acute ischemic infarcts^{2,4,5} have been reported. Pre-existing vascular disease, tobacco usage, and lesser vascularity of white matter tracts are known factors contributing to DBS-associated stroke.⁶ At baseline, our patient was at risk of a lacunar stroke as she had hypertension, diabetes, and hyperlipidemia, which are associated with lipohyalinosis. Intraoperative hypotension and hypoxia, which we did not encounter, have been associated with acute stroke post-DBS placement.⁶ Other reported causes include cerebral venous infarctions due to injury of cortical veins⁵ as well as post-implantation ischemia.⁴

In our presented case, stereotactic planning for burrhole entry and lead trajectory was used and planned carefully to avoid any important structures. The operative plan did not traverse any vascular structures, which is important as a trajectory from a

burrhole near the coronal suture could injure the anterior choroidal artery or lenticulostriate arteries.⁵ We also used intraoperative neurophysiological confirmation of electrode placement, which did not record any lead silencing. Despite our precautions, our patient did have a small IVH in the acute post-operative period. This has been associated with superficial venous injury,³ which we did not encounter. In a large series, 3.8% of patients experienced IVH, with 90% of these being asymptomatic.

While there are no previously reported cases of delayed stroke following DBS placement, there is a previous case series of patients experiencing delayed stroke after radiofrequency pallidotomy. A series of three patients revealed delayed internal capsule strokes following radiofrequency pallidotomy.^{7,8} The patients each had vascular risk factors and the association with the pallidotomy was based on proximity to the lesion.

It is difficult to differentiate peri-operative stroke of other causes from stroke from DBS placement. In our case, we favor the patient's stroke occurring due to DBS placement due to the lack of hemodynamic fluctuations, the proximity of the strokes to the electrodes, the presence of IVH, and the bilateral nature of the stroke. While we were performing DBS implantation as opposed to pallidotomy, our case was in many ways similar to the series by Lim et al.⁸ Our patient, like theirs, was a vasculopath who demonstrated delayed findings and had a delayed infarct near the site of the implant. It is likely that the patient's vasculopathy is the major risk factor, and that medical optimization pre-operatively may reduce the risk of delayed infarction.

In summary, we present an unusual case of delayed stroke in GPi lead placement. Risk for post-operative DBS-associated stroke is likely abrogated by the usage of stereotactic planning, pre-operative medical optimization, and avoidance of hemodynamic fluctuations.

DISCLOSURES

No conflicts of interest to disclose.

STATEMENT OF AUTHORSHIP

RMC collected data and wrote the initial draft of the report and made revisions made on comments. AP analyzed the data, revised the draft, generated the final version of the manuscript, and responded to reviewer comments. AMV was the senior surgeon and approved the submitted version of the manuscript.

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