



# Symptomatic, non-infectious, non-hemorrhagic edema after subthalamic nucleus deep brain stimulation surgery for Parkinson's disease<sup>☆</sup>



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## ARTICLE INFO

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## ABSTRACT

**Object:** We review our experience with Parkinson's disease (PD) patients who underwent subthalamic nucleus (STN) deep brain stimulation (DBS) and then developed noninfectious, non-hemorrhagic, delayed, symptomatic brain edema associated with a DBS lead.

**Methods:** All PD patients who underwent STN DBS lead implantation from 2007 to 2015 were included. The same neurosurgeon performed all surgeries, typically in staged fashion, utilizing single pass microelectrode recordings (MER) within a stereotactic frame. A brain CT was obtained in recovery and subsequently if indicated.

**Results:** There were 189 patients who underwent 363 STN lead implantations among which 35 (9.6%) represent re-implantations of removed leads in 28 (14.8%) patients. Among the 363 STN leads implanted, there were 12 (3.3%) cases of delayed symptomatic edema associated with a DBS lead involving 10 (5.3%) of the patients studied. Of the 328 leads representing first-time operations, there were 9 (2.1%) cases of delayed symptomatic edema in 7 (3.7%) patients, one of whom (14.3%) presented with seizures. For lead re-implantations, there were 3 (8.6%) cases of the brain edema in 3 (10.7%) patients; all presenting with seizures. For the 35 re-implantations, the trajectory to target was the same or very similar via the same burr hole as prior surgery in 17 (48.6%); 3 (17.6%) of whom developed edema. There was no case of brain edema in the 18 re-operated cases using a different burr opening. Edema patients were treated with a course of anticonvulsant medication and dexamethasone. Lead-associated edema resolved over generally a 4 to 6-week course.

**Conclusions:** Noninfectious, non-hemorrhagic, delayed, symptomatic brain edema occurs in approximately 3% of implanted leads and is more common in re-implantations (9%) compared to new implantations (2%). In re-implantations, the edema is more common when the same trajectory is used (18%) compared to a new trajectory (0%). The edema generally occurs 3 to 8 days after implantation, although immediate post-op CT is normal and seizures are a common presenting feature.

## 1. Introduction

Many studies have documented complications associated with deep brain stimulation (DBS) surgery including infection, bleeding, neurologic deficits, and cognitive or emotional changes [1]. A complication periodically encountered, delayed symptomatic brain edema, has been recognized in the recent DBS literature [2–8]. This edema is associated with a DBS lead in the absence of hemorrhage or infection in patients who initially did very well and had unremarkable brain imaging immediately following lead implantation. In the present report, we review our experience over an 8-year period in patients with Parkinson's disease (PD) who underwent subthalamic nucleus (STN) DBS and

developed delayed symptomatic, noninfectious, non-hemorrhagic brain edema associated with a DBS lead.

## 2. Methods

All PD patients who underwent STN DBS lead (Medtronic Inc., Minneapolis, MN) implantation from January 2007 through December 2015 at the University of Kansas Medical Center (KUMC) were included in this study. Patients were evaluated prior to surgery by the same neurologist (RP). The inclusion criteria for surgery included the diagnosis of PD as defined by the United Kingdom Parkinson's Disease Society Brain Bank criteria, responsiveness to levodopa, and

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**Table 1**  
Patient characteristics.

	Age (yrs)	Disease duration (yrs)	Side	# of MER runs	Edema symptoms	Edema diagnosed (days postop)
1	63	8	Right (1st side)	1	Headache, mild confusion and balance problems	4
			Left (2nd side)	1	Mild difficulties w gait and speech, seizure	5
			Right <sup>a</sup>	1	Seizure	5
2	63	18	Left (2nd side)	3	Intermittent confusion, some memory difficulty intermittent word finding difficulty and hesitancy in speech	7
			Right <sup>a</sup>	1	Seizure	3
3	56	8	Right <sup>a</sup> (2nd side)	1	Seizure	3
4	66	3	Right (2nd side)	1	Diplopia	8
5	70	7	Left <sup>a</sup>	1	Seizures	3
6	65	4	Left (unilateral)	1	Intermittent word finding difficulty	8
7	56	7	Left (2nd side)	1	Increased slowness of speech and word finding difficulties	8
8	63	16	Left (2nd side)	3	Word finding difficulties, mild intermittent confusion, mild gait problems	5
9	65	4	Left (unilateral)	1	Progressive headache	6
10	67	2	Left (unilateral)	3	Right facial droop	7

<sup>a</sup> Re-implantations of extracted electrode.

medication-resistant motor fluctuations, dyskinesia or tremor [9,10]. Exclusion criteria for DBS included significant cognitive impairment, psychiatric or behavioral disturbances, or significant abnormalities on neuroimaging. All patients signed written informed consent approved by the KUMC Institutional Review Board to allow their data to be used for research.

The surgical method has been previously described [11,12]. All operations were performed by the same neurosurgeon (JMN). DBS lead (model 3387 or 3389, Medtronic) implantations were most commonly performed in staged fashion with monitored anesthesia care, utilizing single pass microelectrode recordings (MER) and intraoperative test stimulation. All lead implantations utilized a stereotactic head frame and for direct targeting purposes, 1.5 T or 3.0 T MRI. A paraventricular approach was planned to avoid sulci and vessels utilizing a gyrus close to the inner skull bone for brain entry. Following lead implantation, a fine-cut CT study was obtained postoperatively from the recovery room and fused to preoperative imaging to verify lead position and rule out intracranial postsurgical abnormality. When bilateral DBS was planned, the second DBS lead was implanted approximately five weeks after the first lead. A single channel neurostimulator (model 7426, 37602, or 37603, Medtronic) and tunneled extension wire were implanted one to two weeks after each respective lead. More recently, in select patients, due to the severity of their PD and coupled with advances in MRI STN visualization [13], lead implantations were performed under general anesthesia. In these patients, MER were not utilized and lead test stimulation was performed for unwanted motor side effects provided a paralytic agent was not required for the anesthesia. When bilateral leads were planned in one operation, both leads were implanted under general anesthesia and connected 1 to 2 weeks later to a dual channel neurostimulator (model 37601, Medtronic). In cases of re-implantation of a previously extracted DBS lead for reasons such as infection, lack of efficacy, side effects, or hardware breakage, at least one month was allowed following removal of the prior lead and in cases of infection, at least one month following completion of antibiotic therapy.

### 3. Results

Over the time period studied, 189 patients underwent 363 STN lead implantations. There were 137 males (72.5%) and 52 females (27.5%) with an average age of 63.5 years, ranging from 26 to 84 years of age at the time of the initial STN DBS surgery. The average disease duration at

the time of initial surgery was 10.7 years, ranging from 0.5 to 38 years. Among the 363 STN leads implanted, 35 (9.6%) represent re-implantations of removed leads in 28 (14.8%) of the 189 STN patients, resulting in bilateral STN DBS in 165 patients and unilateral DBS in 24. In 6 patients, representing 8 DBS leads, lead implantations were conducted under general anesthesia. No patient in this series had a history of seizure prior to the first DBS surgery. For patients who had re-implantation of an extracted lead, there were no cases of infection involving the central nervous system.

Among the 363 STN leads implanted, there were 12 (3.3%) cases of delayed symptomatic non-hemorrhagic brain edema associated with a DBS lead involving 10 (5.3%) of the patients studied (Table 1). Among these patients there were 7 males and 3 females, with an average age of 63.5 years (range 56–70 years) and average disease duration of 7.8 years (range 1.9–18.4 years).

Of the 328 leads which did not represent reoperations, there were 9 cases of delayed, lead-associated, symptomatic, non-hemorrhagic edema among 7 patients representing a 2.7% incidence per lead and 3.7% incidence per patient in this group (Table 1). The presentation of one (14.3%) of these symptomatic patients included seizures. Within this group, edema diagnosis was made 4 to 8 days postoperative (average 6.4 days).

Among the 35 lead re-implantations, there were 3 (8.6%) cases of delayed, lead-associated, symptomatic, non-hemorrhagic edema among 3 (10.7%) of the 28 patients involved. In all of these cases, presentations included seizures. For the 35 re-implantations, the trajectory to target was via the same burr hole as prior lead surgery in 17 (48.6%); 3 (17.6%) of these 17, developed delayed symptomatic lead-associated edema. Lead-associated edema was not encountered in the remaining 18 re-operated cases conducted via a burr opening different than that of prior surgery. Among the 3 re-operated patients who developed lead-associated edema, diagnosis was made 3 to 5 days postoperative (average 3.7 days).

Among patients who did not present with delayed symptomatic edema, representing 351 DBS leads, there was only one case of postoperative seizure (0.2%) and which occurred in the setting of delayed lead-associated hemorrhage.

All patients who developed delayed, lead-associated, symptomatic, non-hemorrhagic edema had brain CTs immediately following surgery which were without evidence of hemorrhage or edema and were at baseline neurologic function at the time of discharge the morning

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