Case Report Of Peri-Lead Edema Leading To Seizure After Deep Brain Stimulation Surgery And Literature Review

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Case Report

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Abstract

Background: Deep brain stimulation (DBS) has become a frequently performed surgery in patients with Parkinson's disease (PD). It is not free of surgical complications and a mysterious one is cerebral sterile peri-lead edema. Herein, we report on a case of a PD patient with symptomatic peri-lead edema presenting with seizures after surgery and recovered completely after conservative therapy.

Case presentation: A 68-year-old female, with an 8-year history of rigidity-dominant Parkinson's disease. After an all-around previous evaluation by neurologists, she was referred for bilateral subthalamic nucleus (STN) DBS surgery. There were no intraoperative complications. Two days after discharge (postoperative day six) she presented to the emergency department with a new onset of seizures. A subsequent 1.5 T MRI of the brain under stimulation off state showed obvious bilateral vasogenic edema extending from STN targets to cortex. Initiated corticosteroids (intravenous dexamethasone 10 mg per day) with a remarkable completely infectious workup. Levetiracetam (500 mg p.o. bid) was given for seizure prophylaxis. The follow-up brain 1.5T MRI imaging (postoperative day thirty-six) revealed that the edema has improved. At the patient's latest follow-up visit, 12 months after initial lead placement, she demonstrated expected improvement in bradykinesia, gait, and rigidity with a significant reduction in dyskinesia and medication requirement and no evidence of cognitive or memory impairment.

Conclusions: Symptomatic peri-lead edema is no more a relatively rare side effect of DBS surgery. A correct diagnosis can avoid futile and costly procedures such as system removal and antibiotic prescriptions. Eventually the outcome was good in most cases.

Introduction

Deep brain stimulation (DBS) has become a frequently performed surgery in patients with movement disorders including Parkinson's disease (PD). While the significant benefits of this therapeutic modality, it is not free of surgical complications. As the number of DBS procedures has increased, there is a rapid evolution in the scientific understanding of complications related to the procedure and novel complications have been recognized. Among these complications, a mysterious one is cerebral sterile peri-lead edema or occasionally named dramatic radiographic abnormalities seen after electrode placement (DRAAEP), with a widespread incidence ranging from 0.4% to 39% [1, 2]. The etiology of this vasogenic edema surrounding DBS electrodes remains unclear, and its manifestations vary widely from subtle behavioural changes to lethargy and hemiplegia, appearing from hours postoperatively to months later, or in some cases can be asymptomatic. Fortunately, good results were generally reported in most cases. The incidence of symptomatic peri-lead edema varies from 0.4% to 6.9% [3, 4]. Although this complication is uncommon and may be under-reported, the development of edema is important to both clinicians and patients particularly when symptomatic. The long-term effects of symptomatic peri-lead edema are unclear, but understanding its impact on clinical outcomes may help clinicians to counsel patients and establish management protocols. Correct diagnosis of peri-lead edema can be beneficial to patients, for example, it would prevent unnecessary removal of the device and its associated costs to the

health system. The resolution of edema may take several weeks and the treatment with steroids has been suggested [5]. Herein, we report on a case of a PD patient with symptomatic peri-lead edema presenting with seizures after surgery and recovered completely after conservative therapy.

Case Report

A 68-year-old female, with an 8-year history of rigidity-dominant Parkinson's disease, progressively refractory to medication including rasagiline, dopazide tablets and carbidopa-levodopa, was evaluated in our movement disorders clinic. At the initial evaluation, she complained of rigidity in both upper and low extremities and mild tremor in the right hand with wearing-off and peak dose dyskinesia of the right upper limb. A standardized testing using part III of the Unified Parkinson's Disease Rating Scale (UPDRS-III) prior to surgery showed a significant difference between medical on (23 points) and off state (59 points) (61% improvement). Hoehn and Yahr stage at the time of initial evaluation was 3. She had no significant comorbidities and passed all preoperative testing. A review of a brain 3.0T magnetic resonance imaging (MRI) demonstrated no abnormal findings other than ischemic changes and mild atrophy of small vessels. No contraindications to DBS surgery were found. After an all-around previous evaluation by neurologists, she was referred for bilateral subthalamic nucleus (STN) DBS surgery.

Operative details

A Leksell Stereotactic frame (Elekta, Stockholm, Sweden) was mounted on the patient's head. A stereotactic computed tomography (CT) scan was then carried out. CT images were then merged protocol MRI of the brain using the Leksell SurgiPlan system software (Elekta, Stockholm, Sweden) . A three-dimensional reconstruction of the images was performed to identify the locations of the anterior commissure (AC) and posterior commissure (PC). The coordinates empirically chosen were 12 mm to the left and right of the AC-PC midpoint, 3 mm behind the AC-PC midpoint, and 4 mm below the AC-PC midpoint and revised according to the anatomy. After a bi-frontal incision, bilateral burr holes overlying the coronal suture were created at approximately 3.5 cm from the midline on each side. Normally, we start by implanting the microelectrode corresponding to the more symptomatic side and then the other side. Next, a microelectrode drive (Alpha&Omega Engineering Inc., Nazareth, Israel) for the left side was mounted to the Leksell frame. Microelectrode recordings (MERs) were done for physiologic mapping and started from 10.0 mm above the estimated target ,which was optimal for the left side. During insertion, a small cruciate opening was made in the dura and bipolar cautery was used only if bleeding was encountered. She exhibited improvement of her right-sided symptoms, including rigidity and tremor and there was no stimulation induced dyskinesia (SID) or other complications during intraoperative tests. Electrode (PINS L301S model, Peking, China) placement then occurred after confirmed improvement. The Leksell arc (Elekta, Stockholm, Sweden) was then reconfigured for the right side coordinates and the microelectrode drive was remounted. Procedures were same to the left side. A single microelectrode recording tract was performed on both sides. DBS leads were used for both targets connected to a subclavicular implantable pulse generator (IPG) (PINS G102RZ model, Peking, China) meanwhile under general anaesthesia. There were no intraoperative complications. A plasma-derived fibrin sealant was

used intraoperatively to prevent the loss of cerebrospinal fluid (CSF) and intracranial pneumatosis in our patient.

Postoperative course

A CT scan of her brain 6 hours postoperatively showed adequate placement of DBS electrodes in the bilateral STN, mild pneumocephalus along the frontal convexity, with no other intracranial abnormalities. She complained of a headache without nausea or vomiting. Oral medications were resumed in order to avoid happening of Parkinsonism-hyperthermia syndrome (PHS). Next day she developed lethargy and mild confusion consisting of spatial and temporal disorientation, inattention and mild aphasia and poor word retrieval in the absence of fever or meningeal irritation sings. Neurological examinations revealed mild confusion, hypokinesia and rigidity of the limbs as the preoperative medical off state. The patient exhibited normal vital signs and remained hemodynamically stable. Routine lab tests (including complete blood count and blood chemistry, C-reactive protein, erythrocyte sedimentation rate, and blood culture analysis) were within normal limits. Overall, there was a slight decrease in tremor from baseline due to microlesion effect (MLE). On the postoperative day four, the patient's neurological examination was improved and she was discharged home.

But two days after discharge (postoperative day six) she presented to the emergency department with a new onset of seizures. The symptoms were described as left facial twitching and unresponsiveness for less than five minutes, followed by 30 minutes of disorientation. There were a total of three seizures within two hours and one was monitored by physicians as a generalized tonic-clonic seizure (GTCS). Emergent CT scan revealed 3-5 cm of irregular "flame-shaped" peri-lead edema on right side of the cortex (Fig. 1). After symptomatic treatment with intravenous diazepam, intramuscular phenobarbital, intravenous drip mannitol and airway support she was hospitalized again. The immediate electroencephalography (EEG) did not show any epileptic activity except for slow background activity and increased slow wave activity due to the suppression by antiepileptic drugs (Fig. 2).

Initiated corticosteroids (intravenous dexamethasone 10 mg per day) with a remarkable completely infectious workup. Levetiracetam (500 mg p.o. bid) was given for seizure prophylaxis. A subsequent 1.5 T MRI of the brain under stimulation off state showed obvious bilateral vasogenic edema extending from STN targets to cortex (Fig. 3).

The follow-up brain 1.5T MRI imaging (postoperative day thirty-six) revealed that the edema has improved (Fig. 4). Three months later, she presented good response to DBS with complete improvement in dyskinesia and a significant decrement in tremor and hypokinesia. Levetiracetam was discontinued. The patient's cognitive function, verbal fluency and emotional state were fully restored. At the patient's latest follow-up visit, 12 months after initial lead placement, she demonstrated expected improvement in bradykinesia, gait, and rigidity with a significant reduction in dyskinesia and medication requirement and no evidence of cognitive or memory impairment.

Discussion

In the past view, peri-lead edema was a relatively uncommon, benign, and self-limited complication of DBS surgery. Previous recognition had mostly come in the form of case reports and small case series. Nevertheless, with a rapid increase in DBS surgeries over time, it is no more rare. The imaging findings of peri-lead edema depend on the chosen modality of radiography. It is seen as a hypo-dense area around the lead on CT scans or increased intensity on T2 weight or FLAIR MRI, sometimes with contrast enhancement.The distribution of edema is often asymmetrical. Peri-lead edema following DBS surgery includes two types: (1) limited edema in deep white matter and (2) extensive edema in surface white matter, both of which may have different mechanisms [6].

Clinical signs presented a few days to several months (4-120 days) after lead implantation. Furthermore symptomatic peri-lead edema earlier than 33 hours had also been reported [5, 7]. In one case, an interesting observation was made by Ryu et al., who detected MRI peri-lead hyperintensities suggestive of edema in 39% of 38 consecutive patients with unilaterally implanted in the STN. The authors noted that the incidence of edema was higher on earlier MRI scans. It was present in 100% of the patients who were scanned within one month after surgery, in 40% of those imaged within one to three months and in none of those who underwent imaging after three months, indicating its transient nature [8]. Similar results were found in a study by Englot et al., who performed MRI on 133 patients (totally 239 leads) after DBS leads placement. Although there was an overall incidence of edema around 6.3% of the leads, the authors found an increasing incidence of edema with the timing of imaging. It was present in 1% of leads scanned on day one, 22% of those scanned on day four and 50% of those scanned later (5–30 days) [9]. A recent report of a prospective series including a regimented series of scans examining the incidence and time course of white matter edema following DBS electrode implant, which reported the presence of edema following 80% of electrode implants from a total of fifteen patients. In this research, MRI were scheduled for 1 day, 2 weeks, 4 weeks, 6 weeks, and 10 weeks postoperatively and the first incidence of edema was present most often within the first 2 weeks [10]. Another study enrolled 221 DBS patients and identified seven patients with abnormal findings on CT images, displaying increased low attenuation circumferentially surrounding parts of the electrodes and extending into the subcortical white matter. The median duration of clinical symptoms was 10 days. After the clinical symptoms had disappeared, the abnormal findings on the CT images persisted for a certain period. The median period from the initial detection and the complete disappearance of the low attenuation lesions was 25 days [11].

In another prospective study of 19 consecutive PD patients, perilead edema was found on all the MRI images performed 7–20 days after DBS lead implantation. However, in this study only 6 patients were symptomatic associated with the perilead hemorrhage. The authors speculated that this broad variability may be explained by two main reasons: (1) the time between the surgical procedure and imaging, and (2) the different sensitivity of MRI and CT scans in detecting vasogenic edema in the presence of the lead artifacts. The signal characteristics were most consistent with vasogenic edema probably related to the inflammatory response to the DBS material [12]. Imaging researches had shown that retrospective studies examining imaging in the first postoperative week underestimated the prevalence of edema after

the DBS implantation. Furthermore, the time course of the edema was reported simply as the timing of the initial observation, while the actual onset was at some unknown point prior to this time, and resolved before the last image set. Therefore, the time course of the edema was likely longer than described.

The mean age of patients presenting with peri-lead edema was 62.3 years and the mean duration of disease was 9.9 years. The mean time to diagnosis of edema was 5.8 days postoperatively. In most patients, peri-lead edema involved the frontal convexity subcortical white matter, however, the edema along the entire course of the lead and with the involvement of basal ganglia was also noted. The mean duration for follow-up CT scans reported complete/near complete resolution or improvement of edema was 4.7 weeks [13]. Clinical manifestations were variable, ranging from asymptomatic to deterioration of the PD signs, seizure, focal neurological deficits, confusion and behavioral changes. Multiple patients have described a “fogginess”, headache and fatigue, or sometimes memory or minute behavioral changes lasting several weeks. Local and systemic symptoms of infection are absent [14]. To our knowledge, according to the most recent review of the literature, there were few case reports of symptomatic peri-lead edema in the past. Recent articles indicated that 40 cases with symptomatic peri-lead edema including two of them with cystic formation [1]. Albert J. Fenoy et al. noted 0.3% of patients with intraoperative seizure, which was similar to the reported occurrence of 0.3–2.3% of patients in large DBS series [15]. Although there was no survey on postoperative radiological procedures after DBS, literature indicated that most DBS centers, like ours, performed only a postoperative CT scan unless patients presented with new symptoms after surgery. Subsequent imaging with CT or MRI is common, but the interval varies from patient to patient and for different reasons [16, 17].

Patients undergoing DBS surgery for various indications by a single surgeon were stratified into elderly and younger cohorts with a cut-off at the age of 75 years. Seizures occurred in 1.2% of all patients with a similar rate between the two age groups. Although achieving the age of 75 years did not appear to increase the risk of postoperative seizures in their study [18], another study contrarily have suggested that an age of 60 years or older increased this risk. In that single-center retrospective case-control study aiming at determining the incidence of seizure following DBS electrode implantation and to evaluate the factors associated with postoperative seizures. A total of 814 DBS electrode implantations were performed on 645 patients. Overall, 22 patients undergoing placements experienced seizures with an incidence of 3.4%. Multivariate analysis suggested that age at surgery conferred a modest increased risk for postoperative seizures. Gender, primary diagnosis, electrode location, sidedness, and the number of trajectories were not significantly associated with seizures after DBS surgery [19]. One earlier study reviewed a consecutive series of 161 cases involving patients receiving implantations of 288 electrodes at a single institution for the treatment of movement disorders. Seven patients experienced postoperative seizures with an incidence of 4.3%. In 5 of the 7 cases, patients only experienced a single seizure within 24 hours of surgery. Univariate analysis identified three risk factors for postoperative seizures: abnormal findings on postoperative imaging (hemorrhage, edema or ischemia), age greater than 60 years, and transventricular electrode trajectories. And the only significant factor identified in the multivariate analysis was abnormal findings on the postoperative imaging [20].

One case report presented a nonconvulsive status epilepticus after DBS surgery. The patient with PD presenting focal seizures of the right hand and an increasing somnolence leading to a comatose state 3 days after DBS surgery. Repeated EEG indicated a status epilepticus lasting 2 months until the patient regained consciousness. This case suggested that this complication has to be considered as a differential diagnosis in somnolent patients after operation [21].

The phenomenon leading to the perilead edema was not well understood and different mechanisms have been proposed, including mechanical trauma, immune reactionand CSF tracking along the electrodes. The findings suggested that there was a high incidence of symptomatic non-hemorrhagic edema associated with the reimplantations of a previously removed lead, particularly if the path of the reimplanted electrodes were along the previous leads path. In addition, these patients presented with symptomatic edema earlier than the general group and were more likely to develop seizures [5]. Patients undergoing the leads placement using both awake and asleep procedures suggested that the perilead edema could also occur in patients without MERs and supported previous findings of no relationship between the number of brain penetrations and the development of perilead edema [4]. Saitoh et al. proposed two distinct patterns of edema that likely had different mechanisms. These authors proposed that the limited edema of the deep white matter was secondary to microvessel occlusion by the leads, while the more extensive surface white matter edema may be due to the microtrauma of the blood-brain barrier (BBB) or a hypersensitivity reaction to the electrode materials. One research enrolling 15 patients found that 2 of whom in preoperative contrast enhanced magnetic resonance angiography (CE-MRA) co-registered with post-operative CT, no vessel was detected along the electrode pathways, whereas preoperative contrast enhanced CT (CE-CT) or Susceptibility-Weighted angiography MRI (SWAN MRI) co-registered with post-operative CT showed microvessels at the site of peri-lead edema, which showed that microvessels were occluded by the electrodes in these patients [6]. Regarding the etiology, the current evidence including imaging findings, absence of fever, sterile CSF and a good response to steroids, supporting an inflammatory process and an allergic reaction to the leads as a foreign body [3]. The lack of permanent sequelae as well as peri-lead or subcortical edema without typical imaging characteristics showing cortical wedge-shaped ischemia made it unlikely to have been caused by the venous infarct. It was likely that mechanical trauma caused by the microelectrode and DBS lead insertion disrupted the BBB to some extent, possibly causing damage to a newly discovered cerebral lymphatic system which may have predisposed this surgical population to increase inflammatory or immunologic processes [22,23]. In addition, prick and/or patch test should be performed at least in patients with a history of allergic reactions or atopic patients eligible for the DBS surgery to rule out possible hypersensitivity to the electrode components [24].

Similar to the controversy surrounding the incidence and clinical meaningfulness, the appropriate treatment paradigm for a symptomatic patient was debatable. Some researchers recommended that, to avoid over-treatment and iatrogenic complications, no corticosteroid treatment should be administered to patients whose MRI shows peri-lead edema within the first 7–60 days after surgery [12].

As for seizure prevention practice is to prescribe a 1-week course of anticonvulsant therapy (phenytoin or levetiracetam) for all patients with evidence of ischemia, edema, or hemorrhage on postoperative imaging [20]. In patients with symptomatic lead edema and no history of associated seizures, the anticonvulsants were weaned off over a 1 to 2-week course following CT resolution of the edema. In patients with new onset seizure associated with post DBS lead implantation symptomatic edema, a more formal neurology assessment was suggested, likely including the EEG, to guide medication withdrawal. Occasionally hypertonic and hyperosmolar therapies were used due to the amount of mass effect [25].

Interestingly, there did not appear to be any long-term sequelae from peri-lead edema, regardless of the initial symptoms, with outcome data comparable to those reported previously in all DBS patients [13]. Seizures associated with DBS electrode placement were uncommon, typically occurred early within the postoperative period, and seldom leaded to epilepsy [19]. The formation of intraparenchymal cysts seemed to be the progression of peri-lead edema, being part of the same spectrum of reactions as the DBS material with the development of intraparenchymal cysts usually accomanying the worsening of disease symptoms. Intraoperative biochemical analysis of cerebral cyst fluid revealed normal CSF characteristics [26]. If edema surrounded the stimulating tip of the lead, impedance variations might occur, which would make the delivered current unpredictable when using the voltage-controlled stimulation. Switching off the stimulation is the safest option, but is usually uncomfortable for patients; if the system allowed it, a valid alternative could be the use of constant-current stimulation which, by adapting to the impedance changes, could provide a safer and more stable stimulation control [3].

Conclusions

In **conclusion**, symptomatic peri-lead edema is no more a relatively rare side effect of DBS surgery. Its recognition is biased mainly because it is mostly asymptomatic ,but also because of the lack of early routine MRI scans in patients with DBS. Its diagnosis is based on the exclusion of other more common and serious side effects of DBS such as vascular events and infections. A correct diagnosis can avoid futile and costly procedures such as system removal and antibiotic prescriptions. Eventually the outcome was good in most cases. Statistical identification of significant risk factors for post-implantation seizures can assist in the care of patients at risk and minimize the morbidity associated with the seizure.

List Of Abbreviations

DBS, Deep brain stimulation

PD, Parkinson’s disease

DRAAEP, Dramatic radiographic abnormalities seen after electrode placement

UPDRS-III, Part III of the Unified Parkinson’s Disease Rating Scale

MRI, Magnetic resonance imaging

CT, Computed tomography

STN, Subthalamic nucleus

AC, Anterior commissure

PC, Posterior commissure

MER, Microelectrode recordings

SID, stimulation induced dyskinesia

IPG, Implantable pulse generator

CSF, Cerebrospinal fluid

PHS, Parkinsonism-hyperthermia syndrome

MLE, Microlesion effect

GTCS, Generalized tonic-clonic seizure

EEG, Electroencephalography

FLAIR, fluid-attenuation inversion recovery

BBB, Blood-brain barrier

CEMRA, Contrast enhanced MRA

SWAN MRI, Susceptibility-Weighted angiography MRI

Declarations

- Ethics approval and consent to participate

According to local ethical regulations (Ethics committee of the First Affiliated Hospital of Dalian Medical University, Dalian, China) “case reports are not prospectively planned research projects on or with people, but retrospective case descriptions of medical actions. Therefore, the ethics committee is not responsible for evaluating case reports” and consequently is waiving the necessity of an ethical approval for case reports.

- Consent for publication

We have obtained written informed consent for this publication from the patient’s family.

- Availability of data and materials

Not applicable.

- Competing interests

The authors declare they have no competing interests.

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- Authors' contributions

TL and XW: are the co-first authors who write the manuscript. CS, WL and LR: work for the treatment of the patient and draft preparation. LP and ZL: work for the review and critique of the article and contributed equally. All authors contributed to editorial changes in the manuscript. All authors read and approved the final manuscript.

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Figures

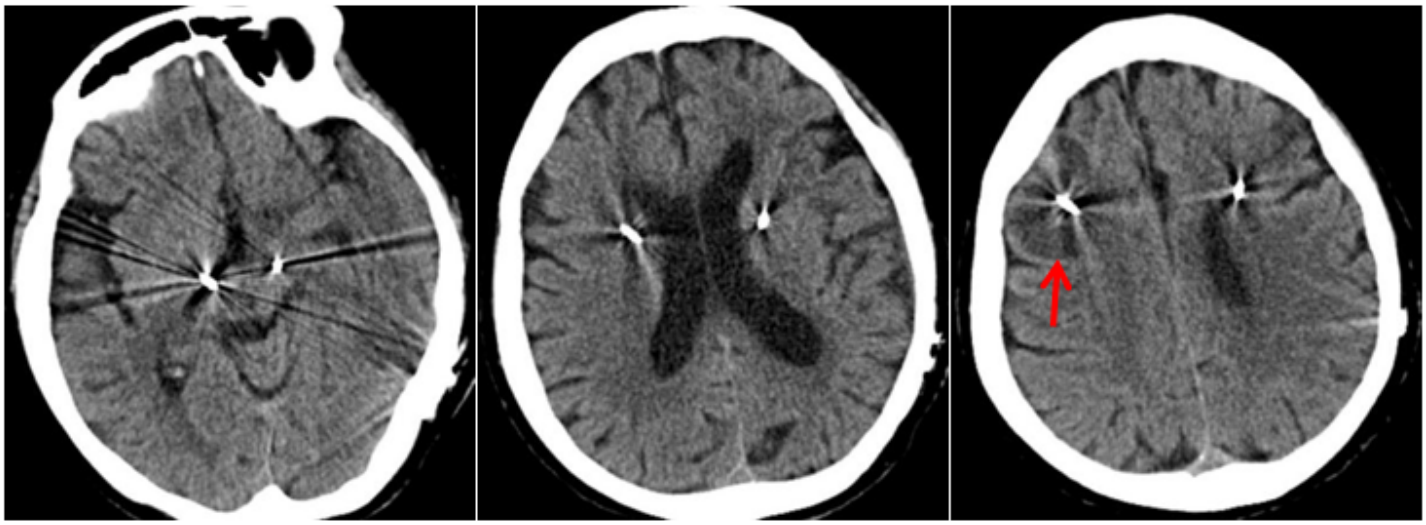

Figure 1

The immediate postoperative CT scan (postoperative day six) after the seizure attack revealed suspicious hypodensity around the electrode in the left frontal lobe, as indicated by the red arrow. Low density was not observed at the top or near the ventricles of the electrodes. Hemorrhage or significant mass effect was not observed.

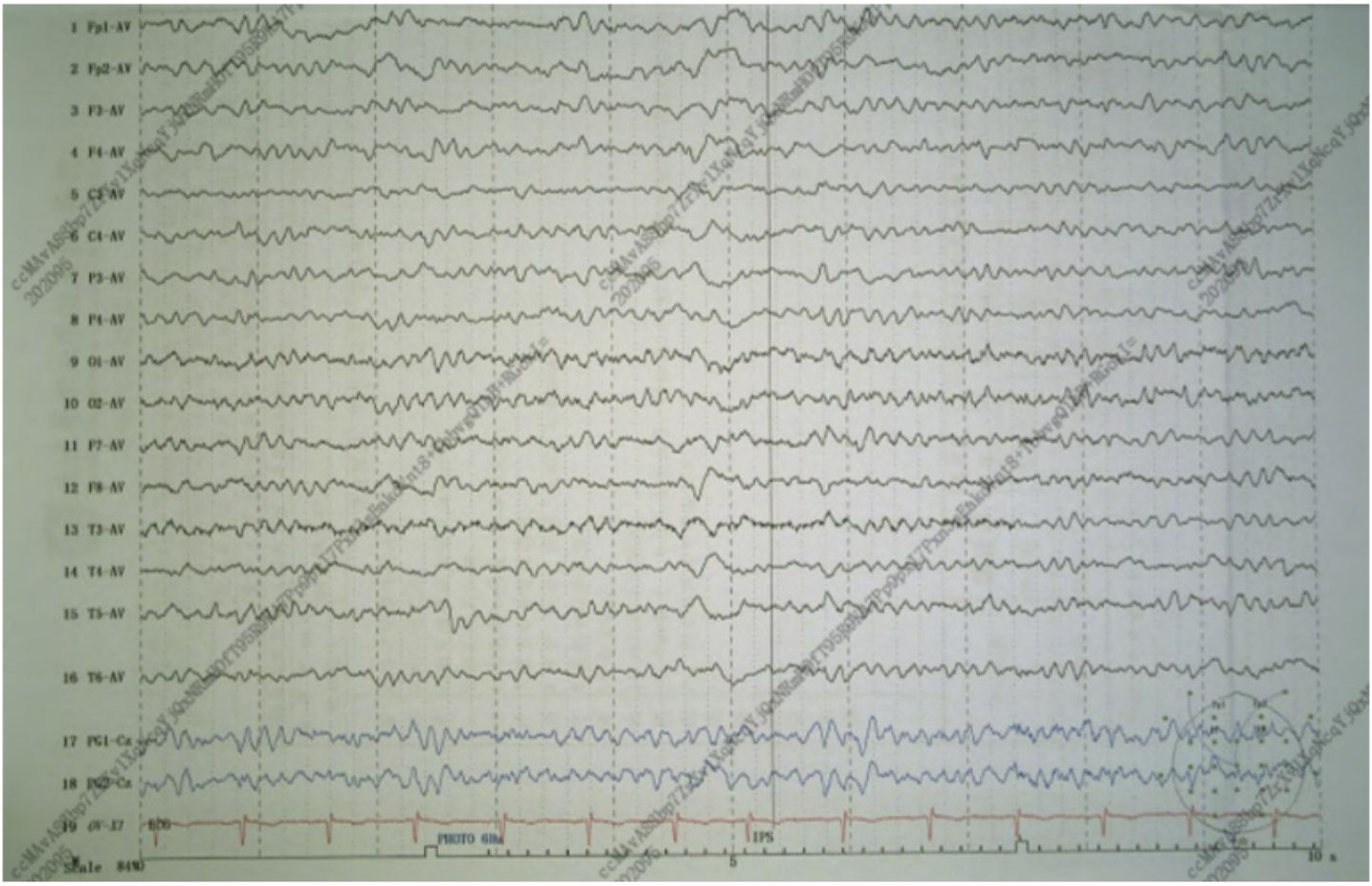

Figure 2

EEG indicated slow background activity and increased slow wave activity.

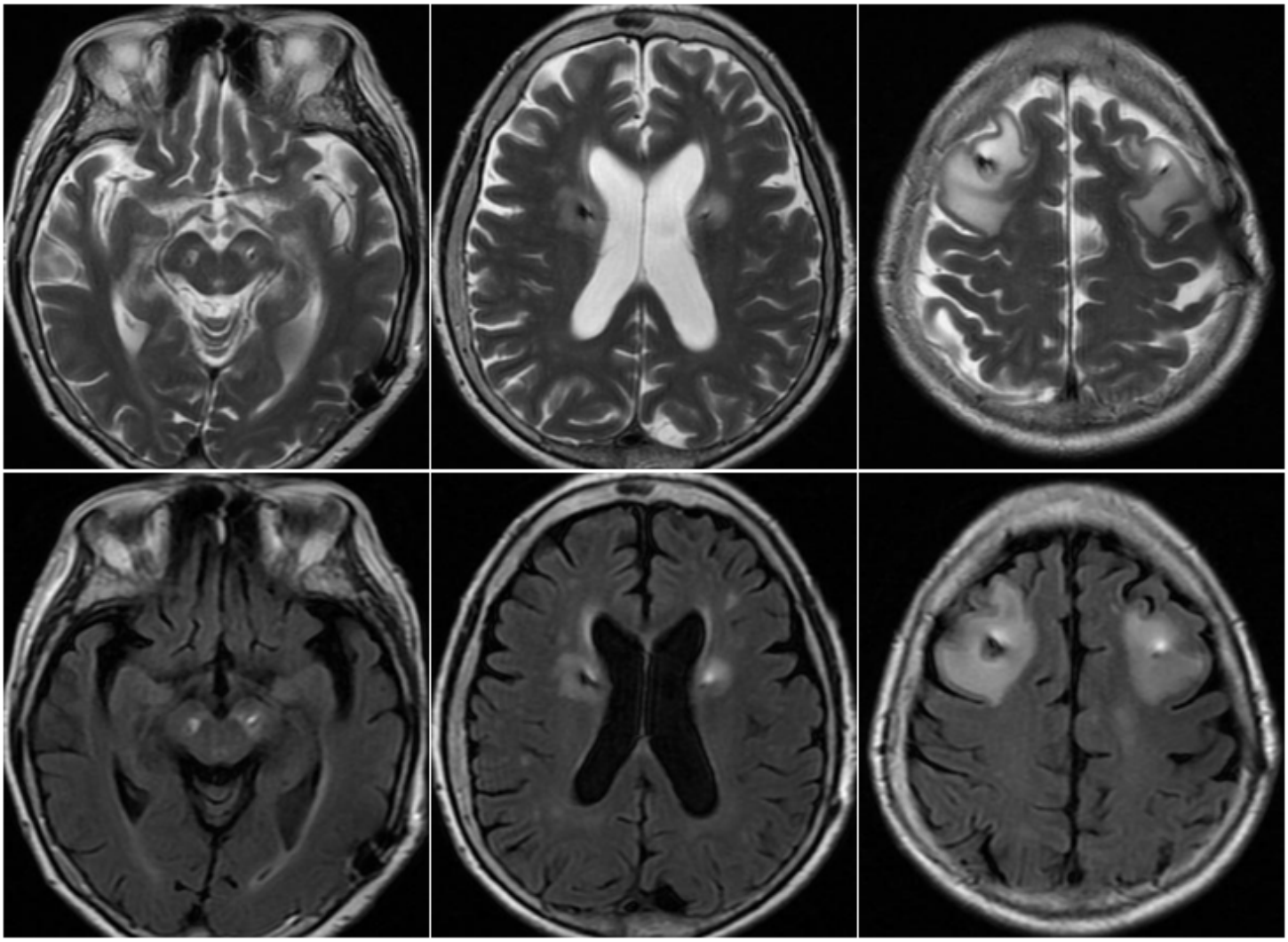

Figure 3

1.5T MRI (postoperative day eleven) showed edema surrounding the DBS leads along the entire trajectory. Increased T2 and fluid-attenuation inversion recovery (FLAIR) signal along the bilateral electrodes at the level of the bilateral STN, near the ventricles and frontal lobes with no associated mass effect.

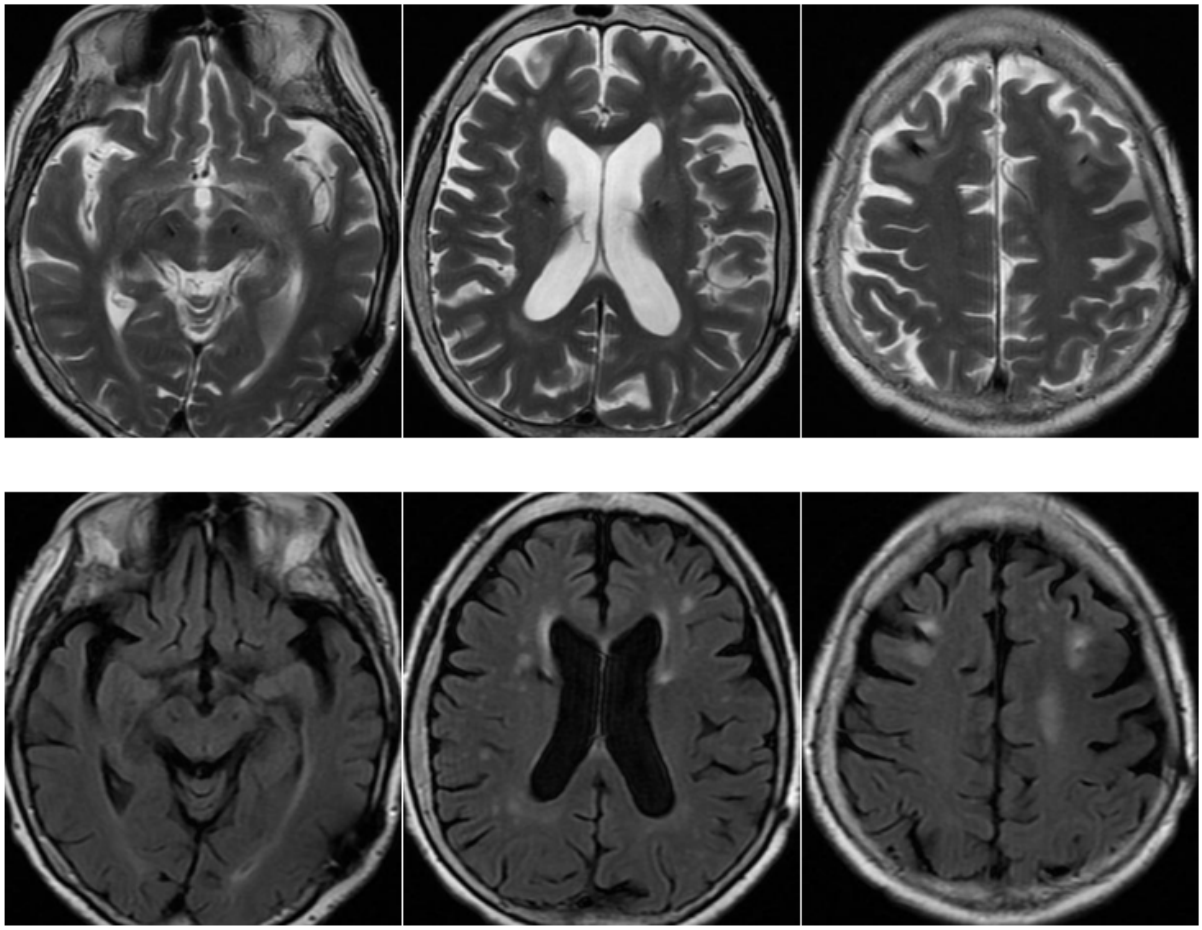

Figure 4

The follow-up MRI (postoperative day thirty-six) showed almost complete resolution of edema except for bilateral frontal lobes.