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Case Report

Cerebrospinal Fluid Egress from the Quadripolar Deep Brain Stimulation Electrode for Anterior Nucleus of the Thalamus for Refractory Epilepsy

Byung-chul Son[1,2], Jin-gyu Choi[1], Sang-woo Ha[3]

[1]Department of Neurosurgery, Seoul St. Mary's Hospital, College of Medicine, The Catholic University of Korea, [2]Catholic Neuroscience Institute, College of Medicine, The Catholic University of Korea, Seoul, [3]Department of Neurosurgery, Chosun University Hospital, College of Medicine, Chosun University, Gwangju, Republic of Korea

Abstract

Deep brain stimulation (DBS) of the anterior nucleus of the thalamus (ANT) is an effective treatment for refractory epilepsy. Due to the unique location of ANT in the thalamus facing the lateral and third ventricles, transventricular DBS lead placement is an essential part of ANT DBS. However, there is no report regarding hardware problems including impedance variability in transventricular ANT DBS due to limited experience. A 45-year-old male patient with previously effective, bilateral ANT DBS presented with increasing seizure frequency and a shortened battery longevity within 2 years. Magnetic resonance imaging showed that the left-sided DBS lead was in the third ventricle leaning on the medial wall of ANT. Electrode revision was performed. Upon disconnecting the proximal lead from the extension connection, cerebrospinal fluid egress through fine gaps between the metallic electrode contacts, and electrode spacing was observed. This case raises a concern about the transventricular approach for ANT lead placement because the currently available DBS electrode lead is not waterproofed. A careful, longitudinal follow-up of DBS impedance for ANT DBS is warranted.

Keywords: *Anterior nucleus of the thalamus, cerebrospinal fluid, deep brain stimulation, electrode, epilepsy*

Introduction

Deep brain stimulation (DBS) in the anterior nucleus of the thalamus (ANT) is effective for the treatment of refractory epilepsy.[1-9] Since the ANT is located in the medial and superior corner of the thalamus facing the lateral and the third ventricle, implantation of the DBS electrode in the ANT is performed through a transventricular approach using the lateral ventricle.[1] Considering that the replacement of misplaced DBS electrodes located outside the ANT was required in 8.2% of 110 patients enrolled in the SANTE trial,[2] accurate placement of the DBS electrode within the ANT may not be an easy task.

We encountered a cerebrospinal fluid (CSF) egress through the proximal end of the DBS lead during the replacement of suboptimal ANT DBS lead into the third ventricle. Since the currently available DBS lead is designed for lead placement within the parenchymal, deep brain nuclei, and the transventricular passage of a DBS lead is rarely needed except in ANT for refractory epilepsy, this kind of hardware problem has not been previously reported.

Case Report

A 45-year-old, right-handed, male patient with bilateral ANT DBS was admitted for revision of DBS lead and replacement of implantable pulse generators (IPGs). He underwent bilateral ANT DBS in 2007 for medically refractory, bilateral occipital lobe epilepsy of 30-year duration. Chronic refractory epilepsy responded favorably to ANT DBS for the next 5 years and seizure frequency measured with percent (%) seizure reduction (i.e., [1-(seizure frequency during the past 3 months/seizure frequency of baseline 3 months before operation)] ×100) at the time of the first IPG replacement (2012) was 55%.[4] However, the frequency of seizure progressively increased in 2013 despite of increasing stimulation intensity (percent seizure reduction, 35%) and generalized tonic–clonic seizure developed again at the end of 2013. The right-sided IPG was depleted in August 2014 and the left-sided IPG was also depleted in October 2014. Despite increasing seizure frequency to

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Address for correspondence:
Prof. Byung-chul Son, Department of Neurosurgery, Seoul St. Mary's Hospital, Catholic Neuroscience Institute, College of Medicine, The Catholic University of Korea, 222, Banpo-daero, Seocho-gu, Seoul 06591, Republic of Korea. E-mail: sbc@catholic.ac.kr

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pre-ANT DBS baseline, the patient could not afford the repeated IPG replacement. In August 2015, he agreed to have the second IPG replacement and was admitted to our hospital for evaluation.

X-rays of the skull, neck, and chest revealing the DBS hardware were unremarkable [Figure 1a]. Due to rapid depletion of IPGs, magnetic resonance imaging (MRI) of the brain was performed to investigate the location of the lead. Axial and coronal T2-weighted MRI images (2 mm thickness) revealed that the left-sided DBS lead was placed within the third ventricle through the foramen of Monro along the medial wall of the thalamus [Figure 1b and c]. Due to shortened longevity of IPGs and modest antiepileptic efficacy, revision of ANT into the centromedian nucleus (CMN) was planned.

Under general anesthesia, the previous frontal wound was opened, and a model 3389 lead (Medtronic, Minneapolis, MN, USA) and model 37,086 extension (Medtronic, Minneapolis, MN, USA) were carefully dissected. Looking inside the connection boot, transparent CSF-like fluid was observed. After the removal of ligatures and connector boot over the lead–extension connection, the proximal end of the DBS lead was separated from the extension connection and was carefully inspected. Drops of the CSF through fine gaps between the metallic electrode contacts and the electrode spacing were observed [Figure 2]. To check the function of the electrode with external stimulation, a new extension cable was connected to the proximal end of the DBS lead and subcutaneously tunneled. Subsequent external stimulation did not elicit an electroencephalographic (EEG) evidence of driving response in scalp EEG and bilateral CMN DBS were performed.

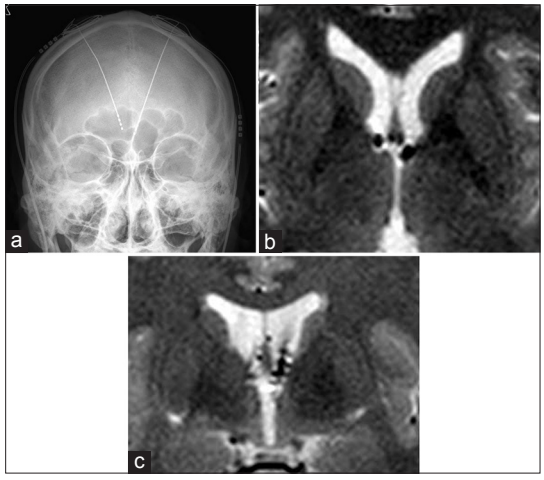

Figure 1: Prerevision radiologic assessment of the patient. (a) An anterior–posterior projection of skull X-ray showing bilateral deep brain stimulation electrode placement for the anterior nucleus of the thalamus. An asymmetrical placement was noted. A T2-weighted axial (b) and coronal (c) magnetic resonance imaging showed that left-sided anterior nucleus of the thalamus deep brain stimulation lead leaning over the anterior nucleus of the thalamus (arrow) was located within the third ventricle

Discussion

Approach to the anterior nucleus of the thalamus

The transventricular approach for the placement of DBS lead into the ANT was initially proposed by Hodaie *et al*.[1] In about a 60° sagittal plane, a quadripolar electrode was introduced with the central lead placed ~8 mm anterior to the posterior commissure and 12 mm above the intercommissural line.[1] Since the span of the model 3389 quadripolar electrode (Medtronic, Minneapolis, MN, USA) is 10.5 mm and the height of ANT is ~6 mm, the bottom one or two contacts are in the dorsomedial nucleus of the thalamus (DM) and the upper two or three in ANT.[1] The effectiveness of this transventricular DBS lead implantation was further replicated in the subsequent studies regarding ANT DBS for refractory epilepsy.[2,4-9]

Implantation of DBS electrode lead in ANT may not be an easy task. In the SANTE trial,[2] the rate of replacement for suboptimal lead placement outside the ANT was 8.2% in 110 patients enrolled. ANT is a lengthy but narrow structure, and targeting is further complicated by the thalamostriate vein representing an extensive risk zone in its immediate vicinity.[10] Furthermore, a surgical trajectory traversing the ventricle was significantly associated with an overall increased hemorrhage rate,[10-13] and avoiding the ventricle during trajectory planning is highly recommended to reduce bleeding risk and to enhance an accuracy of anatomical targeting during DBS.[13-15] However, due to a small size of ANT and characteristic location of ANT within the lateral and third ventricles, implantation of DBS lead through a transventricular trajectory to ANT seems to be an essential part of ANT DBS.

It seems that the first DBS lead in the present case was initially implanted in the anterior ANT rather than those described by Hodaie *et al*.,[1] and full span of quadripolar electrode lead (10.5 mm) does not seem to be incorporated within the central ANT. With careful inspection of MRI data, we could trace a small area of encephalomalacia

Figure 2: A photograph showing cerebrospinal fluid egress from the proximal end of quadripolar deep brain stimulation electrode lead. Cerebrospinal fluid egressing from fine gaps between the metallic electrode contacts and electrode spacing was observed

around the lead, which signified local traumatic changes. Considering the good antiepileptic efficacy during the first 2 years after the first ANT DBS and subsequent shortened longevity of the IPG, we speculated that a shallow, anterior placement of electrode lead in ANT exerted an antiepileptic efficacy, and subsequent ingress and accumulation of the CSF around the lead from the lead entry site of the thalamic surface in the lateral ventricle caused the current flow to the CSF space and shortened the longevity of the IPG.

Deep brain stimulation lead hardware

To the best of our knowledge, leakage and egress of CSF through the electrode lead was not reported in the reports dealing hardware complication. However, problem-associated short circuit in DBS treatment for movement disorders was addressed by Samura *et al*.[16] in 2012. Indeed, short circuit has already been reported to occur when tissue fluid invades connections between the lead and extension wire due to a laceration or loose connector boot ligature.[17] Unfortunately, routine investigation of the impedance of circuit hardware has not been conducted despite of regular follow-up of clinical condition with adjustment of stimulation parameters every 3–4 months' follow-up at our outpatient clinic, and a possible occurrence of short circuit was not taken into account.

We cannot conclude that the conventional, quadripolar electrode lead is mechanically unsuitable for transventricular use. However, it seems that the DBS electrode lead is not waterproof because it was designed and developed for intraparenchymal use, with intraventricular placement not considered until the introduction of ANT DBS for refractory epilepsy.[1] It is uncertain that ingress of CSF through minute gaps between the electrode contact and spacing may not happen if DBS lead had been well placed within ANT and DM, thus by avoiding exposure of electrode contacts to the ventricular CSF.

The present finding prompts a concern about long-term impedance changes in ANT DBS for refractory epilepsy. DBS electrode impedance is a major determinant of the current delivery to target tissues,[18] and changes in impedance over time may affect the long-term stability of electrical current delivery.[19] The latter study[19] showed that parenchymal DBS impedance in movement disorders decreased slowly over time, at a rate of 73 Ω/year, and active contacts had a lower impedance than inactive contacts. As an explanation for gradual decrease in impedance over time, the authors proposed an accumulation of CSF around the electrode[18] such as might be observed in cerebral atrophy of the sort seen in Parkinson's disease[20] or with normal aging,[21] and changes of the blood–brain barrier being more permeable in the vicinity of implanted electrode.[22]

Development of a fine, minute gap between the electrode and the thalamic parenchyma from tissue trauma during transventricular insertion in ANT can cause perielectrode CSF accumulation due to CSF pulsation. Furthermore, it could be expected that long-term variation in DBS electrode impedance, such as a short circuit, may develop because the currently available quadripolar electrode is not waterproof. Currently, we are checking the impedance in every patient who underwent ANT DBS at outpatient clinic and considering an extraventricular approach[23] for ANT DBS in the near future. Further, systematic follow-up with a detailed investigation of impedance variability in ANT DBS would be warranted.

Conclusion

We encountered a new hardware problem in currently available quadripolar DBS electrode during the revision of suboptimally placed ANT DBS electrode in the ventricle. CSF ingress through the gaps between the contacts in the distal lead led to dribbling from the proximal lead. This finding prompts a concern about long-term impedance variability in the transventricularly approach ANT DBS for refractory epilepsy.

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Conflicts of interest

There are no conflicts of interest.

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