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Case Report

Case report of hyperacute edema and cavitation following deep brain stimulation lead implantation

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ABSTRACT

Background: Postoperative cerebral edema around a deep brain stimulation (DBS) electrode is an uncommonly reported complication of DBS surgery. The etiology of this remains unknown, and the presentation is highly variable; however, the patients generally report a good outcome.

Case Description: Here, we report an unusual presentation of postoperative edema in a 66-year-old female who has bilateral dentatorubrothalamic tract (specifically, the ventral intermediate nucleus) DBS for a mixed type tremor disorder. Initial postoperative computed tomography (CT) was unremarkable and the patient was admitted for observation. She declined later on postoperative day (POD) 1 and became lethargic. Stat head CT scan performed revealed marked left-sided peri-lead edema extending into the centrum semiovale with cystic cavitation, and trace right-sided edema. On POD 2, the patient was alert, but with global aphasia, right-sided neglect, and a plegic right upper extremity. Corticosteroids were started and a complete infectious workup was unremarkable. She was intubated and ultimately required a tracheostomy and percutaneous gastrostomy tube. She returned to the clinic 3 months postoperatively completely recovered and ready for battery implantation.

Conclusion: While this is an unusual presentation of cerebral edema following DBS placement, ultimately, the outcome was good similar to other reported cases. Supportive care and corticosteroids remain the treatment of choice for this phenomenon.

Keywords: Computed tomography imaging, Deep brain stimulation, Edema, Electrode, Movement disorders

INTRODUCTION

Noninfectious and self-limiting postoperative cerebral edema around a deep brain stimulation (DBS) electrode is a peculiar, uncommonly reported complication [Table 1].[1-3,8-10,13] The etiology of this clinical scenario remains unknown. Occurrence and presentation of symptoms vary, from subtle behavioral changes to lethargy and hemiplegia, presenting several hours postoperatively to several months later.[1-3,8-10,13]

In this case report, we present a highly unusual and unreported acute scenario whose precipitous decline in neurological condition was worrisome for stroke or infection. Although such a peculiar presentation was very different from other cases of edema previously reported, the management and ultimate good outcome remain similar to others reported.

Table 1: A comparison of various cases of cerebral edema after DBS surgery in the literature with the current case.

Case Study/series	Symptom onset (After surgery)	Laterality	Cavitation?	Intervention(s)	Time to recovery
Arocho-Quinones and Pahapill (2016)[1]	12 days	Unilateral	No	Operative exploration	6 weeks
Deogaonkar *et al.*, (2011)[2]*	4 days–120 days	Unilateral**	No**	Steroids, AEDs, or no intervention	7–60 days
Jagid *et al.* (2015)[8]	4 months	Bilateral	Yes***	Steroids	9 months
Lefaucheur *et al.* (2013)[10]	10 days	Unilateral	No	Steroids	3 weeks
Schoen *et al.* (2017)[13]	33 h	Unilateral	No	Steroids	1 week
Lee *et al.* (2019)[9]	6 h	Unilateral	No	Steroids	6 days
Fenoy *et al.* (current case)	1 day	Unilateral	Yes	Steroids	3 months

*This was a case series examining eight patients with cerebral edema postoperatively. **All eight patients had unilateral edema with no cavitation. ***Patient had bilateral cavitation. AED: Anti-epileptic drug, DBS: Deep brain stimulation. Note: [3] Englot DJ, Glastonbury CM, Larson PS (2011) is a retrospective cohort study and so was not included in this table. Out of 133 patients, they reported 15 instances of DBS lead edema, with the earliest recorded instance being 3 days postoperatively. The edema was typically found to be unilateral. Three patients were symptomatic, and 1 had significant gait instability that was treated with steroids

CASE REPORT

A 66-year-old female presented to our clinic with the mixed diagnosis of essential tremor and Parkinson's disease, as she had both resting and action components of tremor in bilateral upper extremities with bradykinesia and rigidity that were somewhat improved on levodopa. The tremor was largely refractory to medication and interfered with her quality of life. She underwent bilateral DBS lead electrode implantation targeting the dentatorubrothalamic tract, specifically, the ventral intermediate nucleus (Vim), in the thalamus using the standard stereotactic protocol.[4] A trajectory through the ventricle was avoided. Normally, we start by implanting the microelectrodes on the more symptomatic side and then proceed to the other side. In this case, three microelectrodes were simultaneously descended to target the left Vim (as her symptoms were worse in her right hand) first, followed by another three microelectrodes to target the right Vim. Their cannulas were used for macrostimulation to assess for improvement and to choose the best trajectory. Electrode placement (Medtronic 3387 model, Minneapolis, MN, USA) then occurred after confirmed improvement in tremor. The rostral ends of the electrodes were left in a subgaleal pocket to be accessed during a subsequent staged procedure for extension and pulse generator placement. The lead placement was verified in the operating room theater with computed tomography (CT) imaging before closure. Surgery was uncomplicated, and the patient remained interactive and conversant throughout. She was admitted to our neurosurgical ICU as per routine. Head CT performed on early postoperative day (POD) 1 was unremarkable [Figure 1a]. Physical examination revealed no deficit; the patient complained of headache with some nausea/vomiting. She desired to stay overnight. An examination later on the evening of POD 1 found her to be sleepy, and ultimately lethargic. Stat head CT performed revealed marked left-sided peri-lead edema extending into the centrum semiovale with cystic cavitation and trace right-sided edema [Figure 1b]. Physical examination on the morning of POD 2 revealed the patient to be alert but with global aphasia (not following commands and not speaking), right-sided neglect, and plegic right upper extremity. Corticosteroids (IV dexamethasone) were begun early on POD 2. She later became increasingly

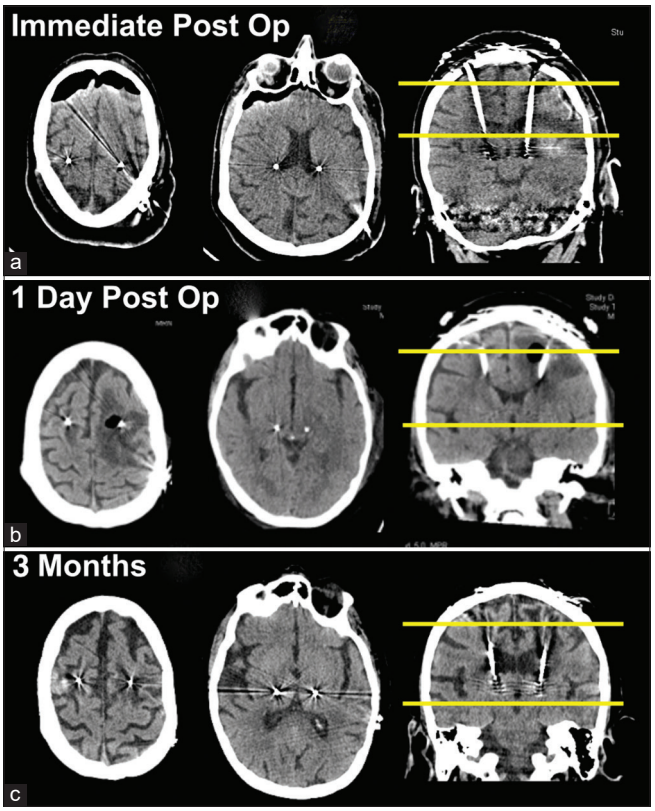

Figure 1: (a) Unremarkable head computed tomography (CT) performed early on postoperative day (POD) 1. (b) Stat head CT later on POD 1 showing marked left-sided peri-lead edema extending into the centrum semiovale with cystic cavitation and trace right-sided edema. (c) Follow-up head CT at 3-month clinic visit showing significant resolution of the peri-lead edema and cystic cavitation. The leftmost image corresponds to the top yellow line in the rightmost image, and the middle image corresponds to the bottom yellow line in the rightmost image.

lethargic, and over concerns for airway protection was intubated. Repeat head CT revealed increased edema.

The critical care team was concerned for fulminant gas-producing bacterial infection as suggested by neuroradiology interpretation of cavitation surrounding one lead and strongly pushed for lead removal, which was resisted. Vancomycin and meropenem were empirically begun. Systemic tests for infection, including C-reactive protein, erythrocyte sedimentation rate, and white blood cell counts, were normal, as well as blood cultures, which were ultimately negative at 24, 48, and 72 h. Such negative infectious workup and lack of change on serial repeat imaging disproved this idea. Magnetic resonance imaging could not be performed due to safety concerns at our institution with an incomplete DBS circuit. Acute venous infarction was also considered a possibility, but the radiological appearance of a cortical-subcortical typically wedge-shaped ischemic pattern was not present.[12] This patient ultimately underwent tracheostomy and percutaneous endoscopic gastrostomy placement 6 days later. She was transferred to a rehabilitation facility on a steroid taper and subsequently discharged home on POD 40. She returned to the clinic 3 months after surgery fully recovered and ready for lead extension and pulse generator placement. Follow-up CT scans at the time showed significant resolution of the peri-lead edema and cystic cavitation [Figure 1c].

DISCUSSION

The occurrence of noninfectious and postoperative edema following DBS electrode implantation is an intriguing, uncommon complication,[1-3,8-10,13] distinct from other more common complications such as hemorrhage, infection, or hardware-related issues. A study by Fenoy and Simpson,[5] reported the incidences of various complications in DBS surgery among 728 patients between 2002 and 2010. The most common postoperative complications were asymptomatic intracerebral hemorrhage (ICH) (0.5%), asymptomatic intraventricular hemorrhage (3.4%), symptomatic ICH (1.1%), and ischemic infarction (0.4%) associated with hemiparesis, and/or decreased consciousness (1.7%). As described in other reports,[1-3,8-10,13] time to presentation is variable, from within the first POD to several months later, as well as the range of findings, from subtle headache to hemiparesis, all independent of underlying diagnosis or target. Such an acute, precipitous change in neurological status on POD 1 requiring intubation concomitant with imaging findings of extensive edema with cystic changes has not been reported.

We agree with the management algorithm as proffered by others[2,3,8] that steroid therapy is the best treatment. However, in acute postoperative presentations, oftentimes in the ICU setting, the neurosurgeon is but one component of a larger multidisciplinary team that provides critical care. Such uncommon complications must be identified quickly when they arise, as they are worrisome for both the patient and health-care team. In our experience, a conservative and comprehensive approach safely ruling out stroke or infection has facilitated a safe outcome without the need for surgical lead removal. Although we and others have seen such radiographic findings of extensive edema with cystic cavitation changes,[8] we have not observed such an acute, serious presentation before.

The etiology of vasogenic edema surrounding DBS electrodes remains unknown, although the transient nature of this process with similarly presenting radiographic appearances and resolution with steroids suggests an inflammatory or immunologic process.[2,3,6,7] Lack of permanent sequelae as well as peri-lead or subcortical edema that lacks typical imaging characteristics showing cortical wedge-shaped ischemia makes it unlikely to be due to a venous infarct. The use of multiple microelectrodes and ventricular transgression has been dismissed by others as noncausative.[1-3,8,10] The reason for this is that the use of microelectrode recordings is common in subthalamic targeting, and this complication has been infrequently reported. Similarly, prior case series examining the rates of hemorrhage, hydrocephalus, infection, etc., after ventricular transgression has not reported this complication. This has led some to consider these noncontributory to this complication. Very likely, mechanical trauma due to microelectrode and DBS lead insertion disrupts the blood–brain barrier to some extent, possibly causing damage to a newly discovered cerebral lymphatic system[11,14] which may predispose this surgical population to increased inflammatory or immunologic processes.

CONCLUSION

Postoperative, transient, and symptomatic cerebral edema is an uncommon complication following DBS surgery. The acute clinical scenario described here is more serious than those previously reported, with corresponding worrisome imaging findings of cystic cavitation, resulting in a precipitous change in neurological condition. As for all cases of edema following DBS implantation, management with supportive care and corticosteroids should be the treatment strategy to effect a normal outcome.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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