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Cerebral Venous Infarction: A Potentially Avoidable Complication of Deep Brain Stimulation Surgery

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Abstract

Object—Despite numerous reports on the morbidity and mortality of deep brain stimulation (DBS), cerebral venous infarction has rarely been reported. We present four cases of venous infarct secondary to DBS surgery.

Methods—The diagnosis of venous infarction was based on: 1) delayed onset of new neurologic deficits on post-operative day 1 or 2, and 2) significant edema surrounding the superficial aspect of the implanted lead, with or without subcortical hemorrhage on CT scan.

Results—Four cases (0.8%/lead, 1.3%/patient) of symptomatic cerebral venous infarction were identified out of 500 DBS lead implantation procedures between July 2002 and August 2009. All four patients had Parkinson's disease (PD). Their DBS leads were implanted in the subthalamic nucleus (STN) (n=2), and the internal globus pallidus (GPi) (n=2). Retrospective review of the targeting confirmed that the planned trajectory passed within 3mm of a cortical vein in two cases

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Conflict of Interest:

1. Takashi Morishita, M.D., Ph.D.

Dr. Morishita has no disclosures to make.

2. Michael S Okun MD

No direct conflicts of interest relative to this project. Dr. Okun serves as a consultant for the National Parkinson Foundation, and has received research grants from NIH, NPF, the Michael J. Fox Foundation, the Parkinson Alliance, Medtronic peer reviewed fellowship training grants, and the UF Foundation. Dr. Okun has in the past received honoraria for DBS educational talks prior to 2010, but currently receives no support (since July 2009). Dr. Okun has received royalties for publications with Demos, Manson, and Cambridge (movement disorders books). Dr. Okun has potential royalty interest in the COMPRESS tool for DBS. Dr. Okun has participated in CME activities on movement disorders sponsored by the USF CME office.

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5. Kelly D. Foote, M.D.

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for which contrast-enhanced pre-operative MRI was available. In the other two cases, contrasted targeting images were not obtained preoperatively.

Conclusion—Cerebral venous infarction is a potentially avoidable, but serious complication. To minimize its incidence, we propose the use of high resolution, contrast-enhanced, T1 weighted MR images to delineate cerebral venous anatomy, along with careful stereotactic planning of the lead trajectory to avoid injury to venous structures.

Keywords

Deep Brain Stimulation; Venous Infarction; Complication; Adverse Event; Hemorrhage

Introduction

Deep brain stimulation (DBS) is an increasingly common procedure for movement and neuropsychiatric disorders.^{1, 2} Appropriate patient selection and preoperative planning are necessary components to optimize outcomes and minimize adverse events.^{3, 4} Typically, functional neurosurgeons use high-resolution Magnetic Resonance Images (MRI) and stereotactic planning software to select an appropriate target and trajectory for DBS lead implantation. If care is not taken to avoid superficial veins when planning the trajectory, cortical veins may be injured, which can result in venous infarction and hemorrhage.^{4, 5} Although it is generally believed to be safe to sacrifice small cortical veins in the frontal region, several reports have substantiated the risk of cerebral venous infarction following neurosurgical procedures.^{6–10} Even though adverse events associated with DBS have been receiving increased attention,^{11, 12} only a few reports have mentioned cases of venous infarction.^{4, 5, 13, 14} In addition, the diagnostic criteria and the clinical course of this complication have not been carefully reviewed. We aimed to identify the incidence and the possible mechanisms of venous infarction in this study. We also discuss diagnostic criteria and offer recommendations for management and, more importantly, prevention.

Methods

Diagnostic Criteria and Follow-up

Utilizing a prospectively-acquired database of adverse events, cases of venous infarction following DBS were identified between July 2002 and August 2009. The diagnosis of venous infarction was made when two criteria were met: 1) neurologic deficits developed in subacute fashion (post operative day one or two), and 2) computed tomography (CT) imaging revealed significant cerebral edema surrounding the superficial aspect of the implanted lead and/or characteristic subcortical hemorrhage. To corroborate the clinical diagnosis, the relationship of imaged superficial veins to the trajectory of the DBS lead was reviewed. Additionally, we reviewed our database to track the clinical course of affected patients. Unified Parkinson's Disease Rating Scale (UPDRS) Part III scores were reviewed to assess motor outcomes. Scales were assessed systematically prior to surgery, and 4 months following DBS implantation.

Surgical Procedure

Each patient had a high-resolution, volumetric MRI scan one day prior to surgery. A Cosman-Roberts-Wells (CRW) head ring was applied under local anesthesia, then a stereotactic CT scan was obtained on the morning of the surgery. Using stereotactic targeting and mapping software developed at our institution, the CT and MRI images were fused. The anterior commissure (AC), the posterior commissure (PC), and another midline point were identified to establish an orthogonal Cartesian coordinate system. Targets were selected using a combination of indirect and direct targeting, augmented with a three-

dimensional digital brain atlas deformed to best fit the basal ganglia. Once the target was selected, the stereotactic trajectory was planned, and *it is important, in the context of this report, to acknowledge the evolution of our targeting methodology over time:*

Prior to the development of sophisticated three-dimensional targeting and microelectrode mapping software, our multipass microelectrode recording (MER) protocol required a parasagittal trajectory in order to map MER data onto parasagittal atlas plates. More importantly, early in our experience, the parasagittal entry angle was selected to correlate with the coronal suture with little regard for superficial venous anatomy. In retrospect, it was not uncommon to encounter significant venous bleeding upon dural opening when this protocol was followed, and the coagulation required to achieve hemostasis in these cases might presumably have significantly compromised local venous outflow in some cases. Furthermore, it was not uncommon practice early in our series to intentionally sacrifice a small cortical vein. This allowed more liberal parallel microelectrode recording passes using an X-Y stage, without having to alter the settings on the stereotactic frame. As our targeting and mapping software and techniques have improved to allow true three-dimensional planning and mapping, and we have better appreciated the importance of avoiding superficial venous injury, the planning of the stereotactic trajectory has evolved to our current practice of carefully adjusting the entry angles to avoid all visible blood vessels, sulci, and the ventricles.

Once the stereotactic plan was completed, the stereotactic coordinates were verified on the CRW arc and phantom. The arc was then transferred to the patient's head ring, secured, and used to determine the stereotactic trajectory and to mark the entry site on the scalp and skull. Then, a 14mm burr hole was placed at the entry site under local anesthesia. The underlying dura was coagulated and opened in stellate fashion, and hemostasis was assured with bipolar cautery. The pia mater at the gyral entry site was coagulated to avoid intra-operative superficial hemorrhage. Serial multi-pass microelectrode recording (MER) was used to develop a physiological map of the target, and to select an optimal site for implantation. The lead was then implanted and connected to an external pulse generator, and intraoperative macrostimulation was performed to confirm the thresholds for stimulation-induced side effects and clinical benefits. DBS leads were then secured in place using the Medtronic Stim-Loc cap. Fluoroscopy was used to insure that the leads were not displaced during this process. Implantable pulse generators (IPGs) were typically implanted under general anesthesia three or four weeks after the lead implantation.

Results

Out of 500 DBS lead implantations (301 patients), four cases of symptomatic cerebral venous infarction were identified (0.8%/lead, 1.3%/patient). All four patients underwent DBS for treatment of Parkinson's Disease (PD). Two of the patients underwent unilateral subthalamic nucleus (STN) DBS, and two underwent unilateral globus pallidus internus (GPi) DBS. Three of the patients developed this complication following the second lead implantation of a staged, bilateral DBS procedure after an uneventful lead implantation on the contralateral side of the brain 6 months previously. The characteristics of these patients are summarized in Table 1.

In both cases (#1 and #3) where contrasted images were available, careful review of the preoperative targeting images demonstrated the presence of a significant cortical vein within 3 mm of the planned trajectory (Figure 1). Fusion of the postoperative CT scan to the preoperative MRI scan also clearly demonstrated that the burr hole was positioned directly over the cortical vein in case 1 (Figure 2). In cases 2 and 4, the vascular anatomy was poorly visualized on preoperative targeting images. In case 2, the preoperative MRI scan was not

used due to unacceptable motion artifact, and targeting was performed using only non-contrasted CT images. In case 4, contrast medium was not given for the preoperative MRI scan, and cortical veins were consequently not well visualized. Case 2 has been reported previously.⁴

All four patients were asymptomatic immediately following surgery, however, neurological deterioration developed on postoperative day 1 in each case. Mental status decline/confusion was the initial symptom in all cases. Two patients developed dysphasia, and one developed significant hemiparesis. The neurologic condition of all patients returned to baseline after days to weeks, and the venous infarction did not result in permanent neurological deficit that was appreciable on routine neurological examination in any case. All four patients experienced improved motor function with DBS despite their subacute post-operative complications. Pre- and post-operative evaluations of motor function are summarized in Table 1 and clinical vignettes are given below.

Illustrative cases

Case 1

A 67 year-old female with Parkinson's disease had right STN DBS with good results, and requested contralateral stimulation be added. Six months after the right DBS implantation, a left STN DBS lead was implanted. She was discharged on post-operative day one after an uncomplicated hospital course, but that evening experienced word finding difficulties and an altered level of consciousness. When these symptoms persisted through the following morning her husband brought her to the emergency room. On examination, except for a significant but incomplete expressive dysfluency and disorientation, she was neurologically intact. A CT scan of the head demonstrated cerebral edema and subcortical hemorrhage surrounding the superficial aspect of the DBS lead. With conservative management as detailed in the discussion below (employed in each of these four cases), her dysphasia improved over the next several days, but some confusion persisted for several weeks. Her speech and cognition ultimately recovered completely.

Case 2

A 50 year-old male with PD had left STN DBS with good results, and requested contralateral DBS. After six months, a right STN DBS lead was implanted. Immediately following the procedure, the patient was neurologically intact. On postoperative day #1, however, he was noted to be somnolent and confused. A head CT scan demonstrated marked cerebral edema surrounding the superficial aspect of the DBS lead without hemorrhage (Figure 3). By postoperative day #3 he had returned to his neurologic baseline, and was discharged home with no focal neurological deficit.

Case 3

A 60 year-old male with PD had left GPi DBS with good results, and requested right DBS. After six months, a right GPi DBS lead was implanted. He was discharged on postoperative day #1 after an uncomplicated hospital course, but later that day he began to develop left-sided weakness, lethargy, and confusion. His weakness progressed and peaked on postoperative day #2, after which time his symptoms began to improve. His wife brought him to the emergency room on postoperative day #4. On examination, he was lethargic, and had 4+/5 weakness in the left biceps, grip, hand intrinsics, and dorsiflexion and plantarflexion of the foot. A head CT revealed pronounced cerebral edema surrounding the DBS lead with subcortical hemorrhage (Figure 3). Over the next several days his symptoms improved, such that by the time of discharge no weakness or focal neurological deficit could be appreciated, and his mental status had returned to baseline.

Case 4

A 58-year old male with PD underwent unilateral left GPi DBS. While he was neurologically normal immediately following the procedure, he was noted to develop word finding difficulty and confusion on postoperative day #1. A head CT initially demonstrated no significant edema or hemorrhage, but a repeat scan performed to evaluate persistent confusion on postoperative day #3 revealed a 3×5 cm irregular “flame-shaped” subcortical hemorrhage associated with pronounced cerebral edema and 2 mm of left to right subfalcine shift (Figure 3). Confusion and abulia persisted for several weeks, but he had made a full recovery with no notable neurological deficits when he was evaluated four months after the operation.

Discussion

Etiology

Venous infarction following neurosurgical procedures commonly results from intraoperative injury to cortical veins. We made a diagnosis in these four cases based on two findings as mentioned in the methods section. Cerebral edema and subcortical hemorrhage are thought to develop as a result of venous hypertension and congestion secondary to obstruction of venous outflow. Symptoms and radiographic evidence become more obvious when large veins are injured. It has been reported that sacrifice of major cortical veins during dural opening can result in acute neurological changes.⁵ On imaging, lesions due to venous infarction are likely to have irregular shapes with poorly differentiated margins, whereas arterial infarctions tend to have sharp, frequently wedge-shaped margins. In addition, reperfusion hemorrhage associated with arterial infarction generally occurs at the periphery of the lesion, whereas hemorrhage from venous infarction typically spreads from center to periphery of the edematous brain.¹⁵ These generalities are consistent with the CT findings in each of our four cases.

The hemorrhagic complication rate is variable in many series (0.6–3.3%),^{5, 13, 16–19} however, some previously reported cases of subcortical hemorrhage might be classified as secondary hemorrhage due to venous infarction based on our criteria. Umemura et al. reported a case with subcortical hemorrhage which was not shown on a CT scan immediately following the procedure.¹³ This case was simply classified as a subcortical hemorrhage without mentioning the etiology, but we believe it was due to a venous infarction based on our diagnostic criteria. Similarly, we suspect that many cases of venous infarction have been overlooked and considered to be primary arterial hemorrhage. There may be value in considering venous injuries as a separate entity in the tracking and reporting of DBS-related adverse events.

We identified surface veins that were presumably injured in cases 1 and 3 following retrospective evaluation of the preoperative stereotactic planning. These two complications can be clearly attributed to inadequate avoidance of surface veins. In cases 2 and 4, because of suboptimal targeting images, the relationship between the DBS trajectory and cortical veins was not as clearly delineated, and we can only presume that a superficial venous injury occurred. In these two cases, our failure to adequately image the cortical venous anatomy made stereotactic targeting to avoid cortical vessels impossible, thereby increasing the risk of venous infarction. In addition, surgeons should be aware of the possibility of damaging an invisible bridging vein in the operative field. Bridging veins may escape the subarachnoid space and drain into venous channels located in the subdural or intradural space⁷, and in this situation, opening the dura through one of these ‘venous lakes’ and securing hemostasis may result in compromise of a significant venous outflow channel.

Clinical Course

When a clinician discovers subacute-onset neurologic symptoms following DBS, venous infarction should be suspected as a potential cause even if no obvious venous sacrifice occurred intraoperatively and no concerning symptoms were evident immediately following the operation. Early detection and appropriate management can minimize neurological deterioration and hasten recovery. Full recovery is common, as seen in our cases, although one patient reported in the literature had acute intraoperative onset with permanent deficits.⁵ The understanding that new postoperative neurologic deficits are attributable to a venous infarct rather than an arterial event, should suggest a likelihood for complete, or near complete recovery. This prognostic information is valuable, and can be used to encourage afflicted patients and their families. It should also be noted that even when venous infarction results in a persistent neurological deficit, activation of DBS and optimization of medical therapy will likely provide a beneficial effect.

Clinical Proposals

Neurosurgeons are trained to place emergency burr holes for external ventricular drainage and/or intracranial pressure monitoring approximately 3 cm off the midline near the coronal suture. This same “safe frontal entry site” is commonly used to implant elective ventricular shunts or ventricular access devices. These common neurosurgical procedures have been traditionally performed without image guidance even though a recent meta-analysis study revealed the incidence of the hemorrhagic complications was as high as 7%.²⁰ This anatomic region of the brain corresponds functionally to frontal associative cortex, and deficits resulting from cortical and subcortical damage in this region are typically imperceptible, hence our preference for brain penetration in this relatively noneloquent region. In many cases, the patients in question are neurologically compromised or even comatose, and subtle post-procedural frontal lobe dysfunction would not likely be perceptible. Certainly, in these emergent, life-saving procedures, this practice is warranted and reasonable. On the other hand, DBS is a purely elective procedure intended only to improve quality of life, and the acceptable risk is therefore much lower. Because DBS is an elective procedure, a careful risk versus benefit analysis should be performed for each patient, and every effort should be made to minimize the risk of the intervention.

We propose several strategies that might reasonably minimize the risk of venous infarction secondary to DBS surgery and share our protocol for management when it occurs. A high resolution, contrasted MRI scan can be used to optimize visualization of blood vessels for preoperative planning.⁵ If MRI is not available (e.g. for patients with cardiac pacemakers), we propose a high resolution stereotactic CT scan *with contrast*. We believe that taking care during preoperative stereotactic targeting to avoid visible blood vessels can minimize the likelihood of significant venous injury. Our practice when planning a DBS trajectory is also to avoid entering the cortex more than 1.5 cm posterior to the coronal suture, and we try to avoid passage through the depth of a sulcus or transgression of a ventricular wall. If a trajectory that avoids visible vessels, sulci, and ventricles is not possible, we recommend prioritizing avoidance of blood vessels, sulci, and the ventricle in that order.

If venous infarction is suspected due to delayed development of neurologic deficits, a repeat imaging study may provide useful diagnostic evidence even when an immediate postoperative CT scan is unrevealing. In cases of venous infarction, we propose the following as a reasonable management strategy: elevate the head of the patient's bed to optimize venous return, control blood pressure to minimize secondary subcortical hemorrhage, administer prophylactic anticonvulsants to diminish the known risk of seizure when subcortical hemorrhage is observed,²¹ avoid dehydration to optimize blood rheology, avoid steroids (the cerebral edema is not inflammatory and would not be expected to

respond to corticosteroid therapy and the procoagulant and immunosuppressive effects of corticosteroids are counterproductive in this clinical setting), insure that the patient is appropriately monitored and has adequate supervision if mental status is altered, and initiate rehabilitation therapy as early as possible. These strategies were applied in each of our four cases reported here. In addition, if a significant cortical vein is known to have been sacrificed intraoperatively, consideration may be given to extending the hospitalization for more than one post-operative day to rule out delayed sequelae before discharge.

Limitations

There are several limitations to this study: First, only two of the four cases had sufficient imaging to clearly demonstrate the presence of a prominent cortical vein at the burr hole site. While the clinical presentation in the other two cases closely parallels these two more definitive cases of venous infarction, and strongly suggests a similar etiology, the pre-operative imaging did not include high-resolution, contrast enhanced MRI. Therefore, the presumed presence of a cortical vein at the surgical entry site could not be demonstrated in these two cases. Moreover, because we did not previously assign significant importance to the preservation of frontal cortical veins, we did not document occurrences of intentional or unintentional sacrifice of cortical veins in our operative records. We are hopeful that our current practice of obtaining contrast enhanced, high resolution targeting imaging for every DBS case, and our new appreciation of the importance of preservation of cortical veins will help to prevent this complication in the future. Certainly, if and when it should occur, our ability to correlate it strongly with a venous injury will be significantly improved.

Conclusion

Cerebral venous infarction is a potentially avoidable, but serious complication of DBS surgery. The subacute development of post-operative neurological deficits after DBS lead implantation, along with characteristic CT findings of superficial edema plus or minus typical subcortical hemorrhage, may indicate venous compromise. To minimize the risk of DBS-associated venous infarction, we advocate the use of high resolution, contrast enhanced MRI to clearly delineate cerebral venous anatomy and suggest careful planning of the stereotactic trajectory to avoid injury to venous structures. We have also shared our strategy for the management of this complication when it occurs.

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Figure 1. Veins excessively close to planned trajectory in two cases of post-DBS venous infarction Crosses indicate the trajectory, and arrows indicate cortical veins. The measured distances between planned trajectory and the cortical were 1.0 mm and 2.5 mm in cases 1 and 3, respectively. A) Axial view of preoperative stereotactic MRI in case 1. B) Probe's eye view in case 1. C) Axial view of preoperative stereotactic MRI in case 3. D) Probe's eye view in case 3. In the probe's eye view, left and right are reversed.

Figure 2. Coronal suture burr hole directly over large cortical vein

Arrows indicate coronal suture on postoperative CT scan on the left and preoperative T1 weighted image with contrast on the right of the case 1. Arrowhead indicates a vein that was likely injured in this case.

Figure 3. CT findings in four cases of post-DBS venous infarction

Significant edema around the superficial aspect of the implanted lead is universally present, and frequently associated with subcortical hemorrhage. Case 4 shows a classical “flame” hemorrhage commonly seen in venous infarction.

Table 1

Patient Characteristics

Case	Age/Sex	Duration of Disease	Medical Comorbidity	Onset	Initial Symptoms of Cerebral Venous Infarction	UPDRS motor scores and % improvement		
						Preop On Meds	Preop Off Meds	4mo (Off Meds/ On DBS)
1	67/F	16 yrs	Parkinson's Disease	POD 1	Aphasia, decreased level of consciousness	14	38	27 (28.9%)
2	50/M	12 yrs	Parkinson's Disease	POD 1	Confusion, decreased level of consciousness	45	81	32 (60.5%)
3	60/M	16 yrs	Parkinson's Disease, HTN	POD 1	Confusion, lethargy, left hemiparesis	8	54	40 (25.9%)
4	58/M	7 yrs	Parkinson's Disease, Obstructive sleep apnea	POD 1	Confusion, expressive aphasia	27	52	41 (21.2%)