ORIGINAL ARTICLE

Peri-lead edema after deep brain stimulation surgery for Parkinson's disease: a prospective magnetic resonance imaging study

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Background and purpose: The aim of this study was to define the prevalence and characteristics of peri-electrode edema in a prospective cohort of patients undergoing deep brain stimulation (DBS) surgery and to correlate it with clinical findings.

Methods: We performed brain magnetic resonance imaging (MRI) between 7 and 20 days after surgery in 19 consecutive patients undergoing DBS surgery for Parkinson's disease. The T2-weighted hyperintensity surrounding DBS leads was characterized and quantified. Any evidence of bleeding around the leads was also evaluated. Clinical and follow-up data were recorded. In a subgroup of patients, a follow-up MRI was performed 3–6 weeks after surgery. We also retrospectively reviewed the post-operative computed tomography scans of patients who underwent DBS at our center since 2013.

Results: Magnetic resonance imaging showed a peri-lead edematous reaction in all (100%) patients, which was unilateral in three patients (15.8%). In six patients (31.6%), we detected minor peri-lead hemorrhage. Edema completely resolved in eight out of 11 patients with a follow-up MRI and was markedly reduced in the others. Most patients were asymptomatic but six (31.6%) manifested various degrees of confusional state without motor symptoms. We found no significant correlation between edema volume, distribution and any clinical feature, including new post-operative neurological symptoms. The retrospective computed tomography analysis showed that peri-electrode hypodensity consistent with edema is absent at early post-operative imaging but is common at scans performed >3 days after surgery.

Conclusions: Peri-electrode edema is a common, transient reaction to DBS lead placement and a convincing relation between edema and post-operative clinical status is lacking.

Introduction

Deep brain stimulation (DBS) is a well-established treatment for a variety of neurological diseases, particularly Parkinson's disease. The most frequently reported complications include hemorrhage, infection and hardware failure [1]. In recent years, non-infectious, delayed-onset post-operative cerebral edema around DBS electrodes has been reported as a rare complication of DBS surgery by different groups worldwide [2–13]. According to the literature, its incidence ranges from 0.4% [10] to 39% [2], but methods for its detection are heterogeneous, including studies of computed tomography (CT) and magnetic resonance imaging (MRI) performed only in selected patients and at different time intervals after surgery. Most studies report post-operative edema as a symptomatic complication [3,5–11,13], whereas others find

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it accidentally during routine imaging in asymptomatic patients [2,4,12]. The true prevalence of this entity is still unknown as no control MRI is routinely performed in the days after DBS surgery.

The purpose of our study was to define the real prevalence and MRI characteristics of peri-electrode edema in a prospective cohort of patients undergoing DBS surgery and to correlate it with clinical findings.

Methods

We performed brain MRI between 7 and 20 days after surgery in 19 consecutive patients undergoing DBS surgery for Parkinson's disease. The T2-weighted hyperintensity surrounding DBS leads was characterized and quantified. Any evidence of bleeding around the lead was also evaluated. Clinical and follow-up data were recorded. In a subgroup of patients, a follow-up MRI was performed 3–6 weeks after surgery. In addition, we retrospectively reviewed the post-operative CT scans of patients who had undergone DBS at our center since 2013. For further details see Appendix S1.

Results

Magnetic resonance imaging study

Table 1 summarizes the demographics, clinical and radiological characteristics of patients. Of the 19 patients who underwent post-operative MRI, seven were female and 12 were male. Mean age at the time of surgery was 58 ± 7.9 (range 42–70) years, with a mean disease duration of 13 ± 3.1 (range 8–17) years.

All except one patient underwent the standard staged surgical procedure. Patient 6, due to Brugada syndrome, could not safely undergo local anesthesia with lidocaine and thus the lead placement and implantable pulse generator implantation were performed during the same procedure under general anesthesia. In this case, intraoperative microelectrode recording was performed, but not stimulation.

During the surgical procedure, two microelectrodes per side were sufficient in all patients and no adjunctive traces were needed. No intraoperative complications were recorded. Immediate post-operative CT scans were unremarkable in all patients.

Five patients had a repeat CT scan before the MRI due to various degrees of confusional state. Two of them presented with small peri-electrode hemorrhage (maximum diameter 20 and 17 mm, respectively) without any mass effect, which was unilateral in one patient and bilateral in the other patient.

Post-operative MRI was performed at 10 ± 3.3 (range 7–20) days after surgery. A variable degree of MRI signal alteration along electrodes was found in all patients (100%). In three patients (15.8%) this was unilateral, whereas in the others it was bilateral. The MRI signal alteration consisted of T2 and fluid-attenuated inversion recovery hyperintensity, T1 hypointensity and increased signal in both diffusion weighted imaging and apparent diffusion coefficient maps. No contrast enhancement was depicted in the first three patients, in whom contrast medium was administered. Therefore, we decided to avoid the use of gadolinium in subsequent patients, unless there was a clinical or radiological suspicion of infection. Based on radiological features, the increased T2 signal of the white matter around the track was consistent with vasogenic edema (Fig. 1). In four patients we also detected the presence of a small amount of peri-electrode bleeding, which was always asymptomatic, raising the total number of hemorrhagic patients to six (31.6%). The peri-electrode hemorrage volume ranged from 0.62 to 10.1 mL. The relative edema volumes (absolute edema volume/hemorrhage volume) ranged from 4.7 to 17.0. MRI did not show any parenchymal peri-electrode microbleeding.

The mean total volume of edema was 24.55 ± 19.5 mL. We failed to find any statistically significant correlation between edema volume and age ($r = 0.3061$, $P = 0.0932$), sex ($P = 0.7351$) or disease duration ($r = -0.2365$, $P = 0.3316$). There was also no correlation between the edema volume on each side and the first implanted subthalamic nucleus (STN) (left, $P = 0.7128$; right, $P = 0.5956$). Edema volume was larger in patients with peri-electrode bleeding (37.38 ± 22.1 mL in patients with hemorrhage versus 18.63 ± 15.62 mL in patients without hemorrhage), but this difference was not statistically significant ($P = 0.0653$). When we examined DBS sides separately, this difference was significant in patients with left-side bleeding ($P = 0.0141$) but not in those with right-side bleeding ($P = 0.0504$). Moreover, patients with bleeding often also presented with severe edema on the side contralateral to bleeding.

Edema distribution was often asymmetrical. It was peripheral (mainly subcortical) in 11 sides and peripheral plus deep in 24 (grade 1 in 15 and grade 2 in nine) sides. No patient presented with isolated deep edema (Fig. 2).

Most patients were asymptomatic but six (31.6%) manifested a transient confusional state consisting of disorientation in space and time and mild signs of frontal lobe dysfunction, such as disinhibition, inattentiveness and, in one patient, slight aphasia with poor word retrieval. No patient exhibited any new

Table 1 Demographic, clinical and imaging characteristics of patients

Patient no.	Gender	Age (years)	Disease duration (years)	PD symptom onset side	Symptoms	Edema volume (left, right, total) (mL)
1	F	62	15	Left	Confusion	35.51, 27.41, 62.92
2	M	68	8	Right	Confusion, anomias	67.62 (H), 3.56, 71.18
3	M	52	17	Right	Confusion	7.48, 5.54, 13.02
4	M	56	14	Left	Confusion	0, 7.23, 7.23
5	F	62	17	Right	None	5.98, 20.94, 26.92
6	M	59	11	Left	None	10.48, 9.83, 20.31
7	F	58	11	Right	None	1.22, 5.3, 6.52
8	F	47	8	Left	None	12.29, 18.69, 71.98
9	F	69	17	Left	None	3.29, 6.92, 10.21
10	M	42	15	Right	None	0, 2.85, 2.85
11	M	70	16	Right	Confusion	13.09, 24.41, 37.5 (H)
12	F	51	9	Left	None	4.34, 35.79 (H), 40.13
13	M	56	16	Left	None	3.31, 8.64, 11.94
14	M	49	12	Left	None	11.95, 11.82, 23.77
15	M	64	10	Left	Confusion	11.84, 4.84, 16.68
16	M	61	11	Right	None	0, 10.21 (H), 10.21
17	F	61	11	Left	None	7.83, 4.62, 12.45
18	M	61	15	Left	None	4.45, 8.75, 13.2
19	M	67	15	Right	None	30.44 (H), 17.62, 48.06

F, female; H, hemorrhage; M, male; PD, Parkinson's disease.

motor deficit. The presence of symptoms was not correlated to edema volume ($P = 0.2538$) or to its distribution ($P = 0.4561$). Symptomatic patients were older than asymptomatic patients (62 ± 6.93 vs. 56.38 ± 7.97 years old, respectively) and more commonly male (five male versus one female) but these differences did not reach statistical significance ($P = 0.1241$ and $P = 0.3331$, respectively). Based on the presence of an important edematous reaction, which the authors were not familiar with at the beginning of the study, a short corticosteroid treatment was administered to patients 1 and 2, with no significant effect on symptoms (confusion in both and mild aphasia in patient 2). All patients recovered completely within 2–4 weeks. The presence of symptoms was not statistically correlated with disease duration ($P = 0.1242$) or pre-surgery Montreal cognitive assessment score ($P = 0.3160$).

Follow-up MRI was performed in 11 patients at a mean of 40.64 ± 11.5 (range 21–59) days after surgery. The peri-electrode signal alteration had completely resolved in eight patients (Fig. 3). The other three patients, who underwent MRI at 30, 57 and 21 days after surgery, still had a small amount of vasogenic edema along the leads (12.54, 2.08 and 10.36 mL of total volume, reduced by 18.44, 35.42 and 2.42 mL, respectively). In all cases, the distribution of residual edema was only subcortical, even if it was subcortical plus deep at the first MRI. At follow-up, no patient showed peri-electrode bleeding or microbleeding.

Computed tomography retrospective study

From January 2013 to February 2017 we performed a total of 77 bilateral DBS operations (154 sides). DBS leads were implanted in the STN in 76 patients and in the internal globus pallidus in one patient. Post-operative CT scan was performed immediately after surgery in all patients and showed a unilateral hemorrhage in two patients (2.6%) and was unremarkable in all others. Imaging was repeated in the following days (1–10 days after surgery) only in 15 (19.5%) symptomatic patients, including the two with bleeding complications detected at first CT scan. Symptoms that prompted repeated imaging varied from hemiparesis (three patients), seizures (one patient) to confusion (10 patients). In two of these, CT scan detected unilateral parenchymal hemorrhage, bringing the total number of hemorrhagic complications to four (5.2%). Peri-electrode edema was found in six patients. It was present bilaterally in two of the four patients with hemorrhage and in four of the six patients who underwent CT scan >3 days after surgery. In the earlier CT scans, which were performed in nine patients (day 1, 2 and 3 after surgery), edema was present in the two patients with hemorrhage and in one other patient. The other six early scans were unremarkable. Among the symptomatic patients, only one (1.3%) of those with hemorrhage had a persistent neurological deficit (hemiparesis) at follow-up, whereas all of the others recovered completely.

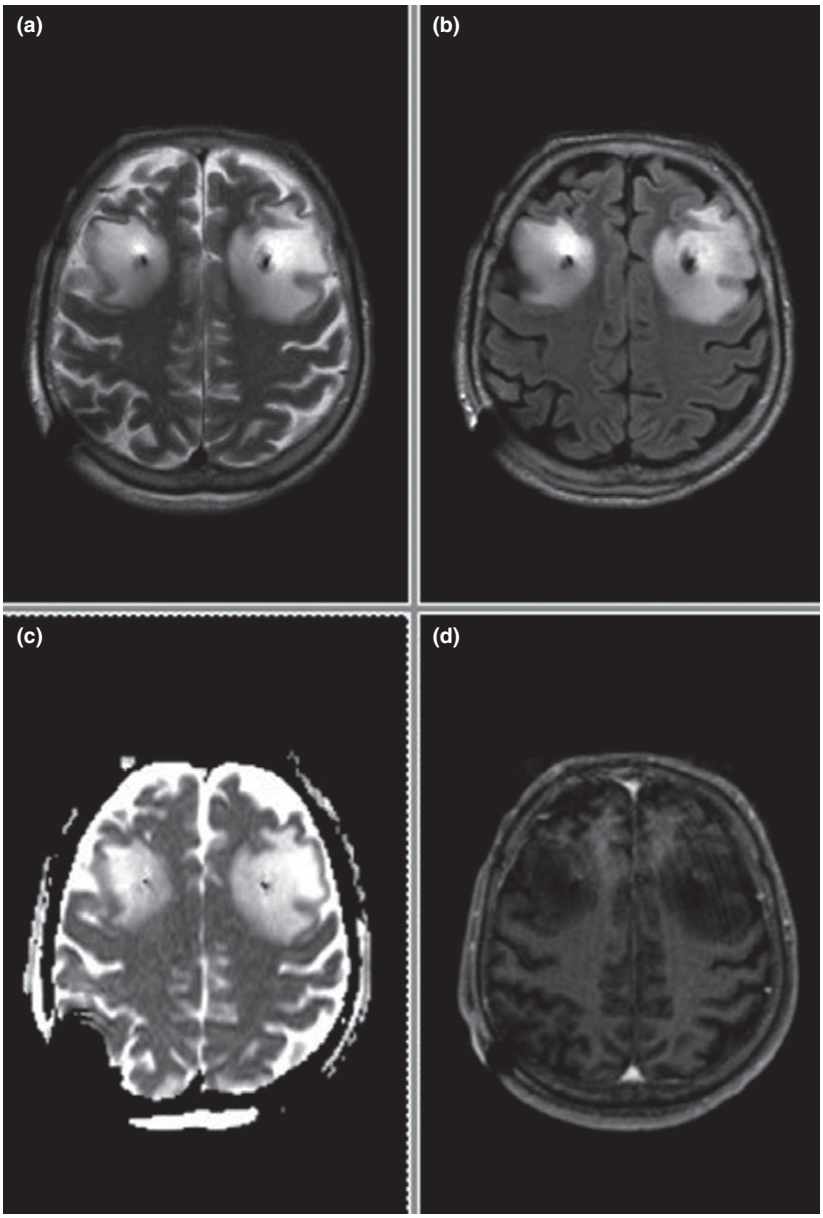

Figure 1 Axial magnetic resonance imaging features of peri-lead edema (patient 1, post-operative day 8). Peri-lead edema appears hyperintense in (a) T2 and (b) fluid-attenuated inversion recovery images with (c) high apparent diffusion coefficient and (d) no significant enhancement in T1 post-gadolinium images.

Discussion

Our prospective MRI study on peri-electrode edema following STN-DBS shows that variable degrees of T2 hyperintensity along electrodes, consistent with vasogenic edema, were present in all patients. Most patients were asymptomatic but six (31.6%) manifested various degrees of confusional state without significant motor symptoms.

We hypothesize that peri-lead edema is a common transient finding in STN-DBS implanted patients for Parkinson's disease and that a convincing relation between edema and post-operative clinical status (i.e. transient confusional state) is lacking. In fact, we found no correlation between edema volume and the presence of confusional state, which is more likely to be related to older age at the time of surgery.

A recent multicentric retrospective study by de Cuba *et al*. [10] presented a case series of 12 patients with 'idiopathic delayed onset edema' surrounding DBS leads. The authors defined this new entity as edema occurring at least 72 h after surgery in the absence of trauma, vascular events (including hemorrhage) or signs of infection. All but one of the described patients were symptomatic (with symptoms varying from confusion, seizures and focal neurological signs such as hemiparesis, dysarthria and aphasia) and edema was detected by either CT or MRI at

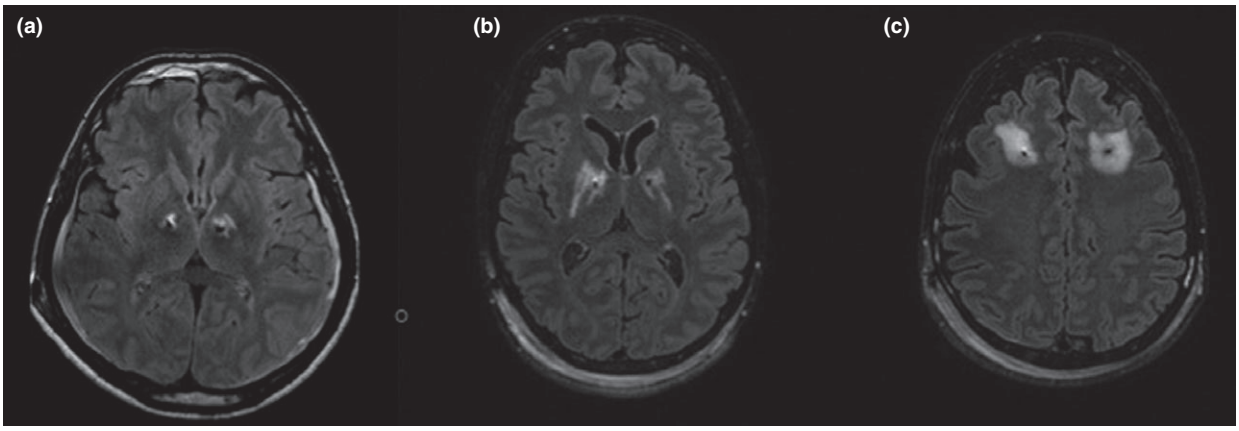

Figure 2 Fluid-attenuated inversion recovery axial images showing example of distribution of edema along the lead. (a) Deep edema grade 1: involvement of the internal capsula only (patient 3, post-operative day 14). (b) Deep edema grade 2: involvement of the internal capsula and basal ganglia. (c) Peripheral edema. Both (b) and (c) are from patient 14 on post-operative day 8.

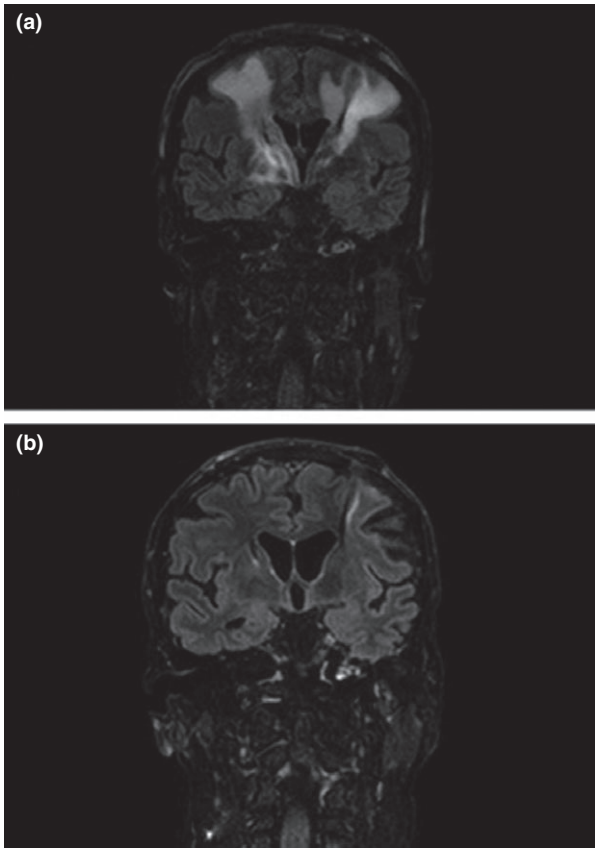

Figure 3 Fluid-attenuated inversion recovery coronal sections showing the spontaneous evolution of peri-lead edema. (a) Conspicuous peripheral and deep edema in early post-operative magnetic resonance imaging (MRI) (patient 1, post-operative day 8). (b) Complete resolution of edema in mid-term follow-up MRI (patient 1, post-operative day 47). This section reveals small artifacts along both leads.

different times after surgery, ranging from 4 to 396 days. Symptoms resolved in all cases after a mean of 28.5 days and radiological resolution was documented in all except two patients with ongoing edema at the end of follow-up (30 and 210 days). The approximate incidence of this finding was of 0.4% and thus the authors defined it as a rare complication.

In past years other authors have described radiological findings consistent with peri-electrode edema in patients following DBS lead placement [2–13]. The literature indicates an extremely wide range from 0.4% [10] to 39% [2] for this complication. We speculate that this broad variability may be explained by two main reasons: (i) the timing between the surgical procedure and the neuroimaging, and (ii) the different sensitivity of MRI and CT scans in detecting vasogenic edema in the presence of the lead artifact. It is also of note that, in previous studies, the incidence was not calculated in prospective cohorts but was based on reports of symptomatic or accidental findings in patients with DBS imaged with CT or MRI for different reasons.

Although there is no survey on post-operative radiological procedures after DBS, the literature indicates that most DBS centers, like ours, perform only a post-operative CT scan unless patients present with new symptoms on the days after surgery. Subsequent imaging with CT or MRI is common, but at different time intervals and for different reasons in each patient [14–16]. In this scenario an interesting observation was made by Ryu *et al.*, who detected MRI peri-lead hyperintensities suggestive of edema in 39% of a consecutive series of 38 patients who were unilaterally implanted in the STN. The authors noticed that the incidence of edema was higher in the earlier MRI

scans. It was present in 100% of the patients who were imaged within 1 month after surgery, in 40% of those imaged at 1–3 months and in none of those who were imaged after 3 months, suggesting its transient nature. All patients were described as asymptomatic [2]. Similar results can be found in the study performed by Englot *et al*., who performed MRI on 133 patients after DBS lead placement (239 leads). Despite an overall incidence of edema surrounding 6.3% of leads, the authors found an increasing incidence of edema depending on the timing of imaging. It was present in 1% of leads scanned on day 1, 22% of those scanned on day 4 and 50% of those scanned later (5–30 days) [4].

In line with these results, our prospective MRI study confirms that peri-lead edema is a common transient finding in STN-DBS implanted patients for Parkinson's disease and that it is asymptomatic in most patients. The reason for this extremely high incidence is probably the timing of imaging (average 10 days after surgery). As in previously published studies, immediately post-operative imaging showed no edema in any of our patients. This result is also supported by our retrospective CT study, where no peri-electrode hypodensity was detected at post-operative CT scan, but was present in four of the six patients who underwent CT scan >3 days after surgery.

The type of imaging is also crucial as CT scans can underestimate the presence and entity of edema due to lead-related artifacts.

A limitation of our study could lie in the single-center nature of our investigation, meaning that the rate of complications could be biased by our center's experience with DBS surgery. However, this could represent a strength, as surgical procedures were standardized, performed in the same conditions and using the same surgical technique by a dedicated team. More importantly, we used identical microelectrodes and leads in all patients. For these reasons, we believe that our results of imaging and complications are stronger because they depend mainly on patient reaction to lead implantation and less on surgical or mechanical issues. Another limitation is the short clinical follow-up of patients. The presence and extent of edema could correlate with long-term outcome, not only of motor aspects but also of cognitive functions. Therefore, all of the present patients are enrolled in a 1-year ongoing follow-up study to analyze clinical and neuropsychological outcome in relation to edema volume and distribution.

The higher rate of symptomatic patients in our series compared with those of Ryu *et al*. [2] and Englot *et al*. [4] is probably mainly due to two factors: first, we implanted patients bilaterally in one procedure and secondly, our patients were probably hospitalized for a longer time due to the two-stage procedure (see Methods) and thus more strictly observed from a clinical and radiological point of view. Moreover, this incidence is in line with other studies in which a mild post-operative confusional state is described in approximately 10% (1–36%) of DBS patients with Parkinson's disease [16].

Our study also shows a large number of hemorrhagic complications (mostly asymptomatic or, at least, with short duration confusional state, which was also found in patients with no evidence of bleeding). Nevertheless, when considering patients together with past cases, the incidence of hemorrhage drops to 10.4%. If we consider that we would not have detected bleeding in four of these patients because they were asymptomatic, this incidence would even be lower (6.3%), which is also in line with previous studies [16]. Interestingly, we found larger edema volumes in patients with small subcortical bleeding, but in these cases the edema timing was delayed with respect to that of hemorrhage and also the extent and distribution exceeded those of usual peri-hemorrhage edema. In fact, our relative edema volumes (4.7–17.0) are lower when compared with relative edema volumes occurring around spontaneous intracranial hemorrhage, where the reported relative peri-hematomal volumes show a median of 0.46 at baseline and 0.81 at 20-h CT with a range of values from 0.0 to 5.13 [17].

The pathogenesis of peri-lead edema remains unknown. As other authors have suggested, it probably represents a para-physiological tissue reaction to external body implantation [2,4,10]. This hypothesis is strongly supported by the radiological characteristic of the edema, which mainly involves the white matter along the track independent of the presence of bleeding and in the absence of any local or systemic sign of infection. Furthermore, the transient and benign nature of edema is backed by the evidence that all cases have proven to be self-limiting, with gradual improvement or complete resolution without treatment.

In addition to patients with DBS, a similar observation was made in patients with in-dwelling external ventricular drain catheters, scanned by CT and MRI at different time intervals after catheter placement [18,19], and in patients undergoing ventricular puncture for subarachnoid hemorrhage [20]. However, these findings cannot be considered identical to ours as, in the first two articles (ventricular catheters), MRI showed the presence of a coexistence of cytotoxic and vasogenic edema and, in the third article (ventricular puncture), there was no radiological characterization of the parenchymal alterations that were generically defined as 'tissue damage'.

Conclusions

Our opinion is that peri-lead edema is a normal and constant finding in patients undergoing STN-DBS lead placement. Its recognition is biased mainly because it is mostly asymptomatic and also because of the lack of early routine MRI scans in patients with DBS. We believe that what other authors [3,7,10] describe occurring as much as 1 year after surgery is probably another entity, with a different pathogenesis and clinical presentation. Therefore, we suggest that, to avoid overtreatment and iatrogenic complications, no corticosteroid treatment should be administered to patients whose MRI shows peri-lead edema in the first 7–60 days after surgery.

Further studies on larger cohorts are needed to correlate the entity of edema with long-term outcome and complications.

Disclosure of conflicts of interest

The authors declare no financial or other conflicts of interest.

Supporting Information

Additional Supporting Information may be found in the online version of this article:

Appendix S1. Methods.

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