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CASE REPORT
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Chronic Subdural Hematoma after Deep Brain Stimulation for Parkinson's Disease with a Lack of Post-Operative Stimulatory Effect: A Case Report

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We report a patient who had a bilateral burr-hole drainage due to the development of chronic subdural hematoma after deep brain stimulation (DBS) for the advanced Parkinson's disease. The patient showed significant improvement during intraoperative test stimulation with the absence of significant abnormal radiologic findings at the immediate postoperative CT scans. Postoperative course showed progressive deterioration of preoperative akinetic symptoms without notable benefit from therapeutic stimulation. CT scans two months after the surgery showed significant amount of chronic subdural hematoma with the upward displacement of implanted electrodes from the original targets. Bilateral burr-hole drainage was needed because of neurologic deterioration, with a special attention not to disrupt the previously implanted DBS hardware system. Follow-up CT scan revealed resolution of subdural hematoma and return of migrated electrode to its original position. Without DBS lead revision, the patient experienced benefits from the bilateral DBS therapy on follow-up.

KEY WORDS: Deep brain stimulation · Chronic subdural hematoma · Parkinson's disease · Burr-hole drainage.

INTRODUCTION

Deep brain stimulation has become effective for not only advanced Parkinson's disease, but also for dystonia, tremors and refractory epilepsy. Chan, et al.[2] classified DBS related complications into operation-related, hardware-related and stimulation-related. Operation-related complications included intracranial hemorrhages and electrode malposition. Focusing on operation-related complication, Boviatsis, et al.[1] reported that the intracerebral hemorrhage cases comprised 1.9% of all cases in the study. Also they reviewed the literature and revealed the mean rate of intracerebral hemorrhage was 3.9% regardless of the outcome. Immediate computed tomography (CT) after DBS can reveal acute intra-parenchymal, subdural or epidural hemorrhage.

Intracranial hemorrhage is one of the most significant complications following DBS surgery. Chronic subdural hematoma after DBS lead implantation is rare and no clear management of the hematoma and the electrodes exists.

We report a case of chronic subdural hematoma after DBS who did not show any abnormality on immediate postoperative imaging.

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CASE REPORT

A 64-year-old female was diagnosed with idiopathic Parkinson's disease at 2002 and had continued levodopa medications. For the last 4−5 years of medication, dyskinesia and drug on-off fluctuation was getting worse. Because the dyskinesia was extremely severe, bilateral globus pallidus internus (GPi) DBS was decided by the DBS surgery team including movement specialists and neurosurgeons. Pre-operative MRI showed no significant abnormality except mild brain atrophy (Fig. 1A). Briefly, we used 'asleep-awake-asleep' technique using intermittent infusion of Dexmedetomidine (Precedex). Kwon, et al.[5] Microelectrode recording was performed to define the target GPi region showing typical fast firing neurons, and also used for test stimulation to verify beneficial stimulatory effects without side effects. After deciding the optimal site, the therapeutic electrode (Model 3387, Medtronic) was implanted and anchored using the Stimloc (Medtronic).

Immediate postoperative CT scan showed no abnormal finding (Fig. 1B). The postoperative course was uneventful except non-painful swelling with suspicious watery content inside the pocket on her left chest wall where the pulse generator was implanted, which was resolved

spontaneously within several days. No bruise or blood oozing around the implanted site suggested it was not result from the hematoma. A small amount of bilateral subdural hygroma was noted on follow up CT scan (Fig. 1C) on 7th postoperative day. Because there was no symptom, no treatment for the subdural hygroma was needed. After discharge, she had neurological out-patient clinic follow-up. Follow-up CT after 1 month from the operation showed newly developed subacute SDH, left cerebral convexity (Fig. 2A). There was no significant mass effect of subdural hematoma, we decided to do conservative treatment and planned short-term out-patient clinic follow-up. At this time, the Levodopa medication was reduced from preoperative Levodopa equivalent dose (LEDD) of 1,350mg to 865mg by the neurologist. After 2 months from the DBS, she came to the outpatient clinic with aggravated akinetic symptom and disorientation. At this time, the LEDD was further decreased to 665mg. Gradual increasing the DBS voltage from 1.5 V to 3.5 V was not effective to control akinesia and rigidity as intraoperative result. Immediate CT scan showed significant amount of bilateral chronic SDH with a midline shift to right (Fig. 2B). There was no traumatic event according to the patient's family.

Surgical decompression was decided, and emergency bilateral burr-hole drainage was performed.

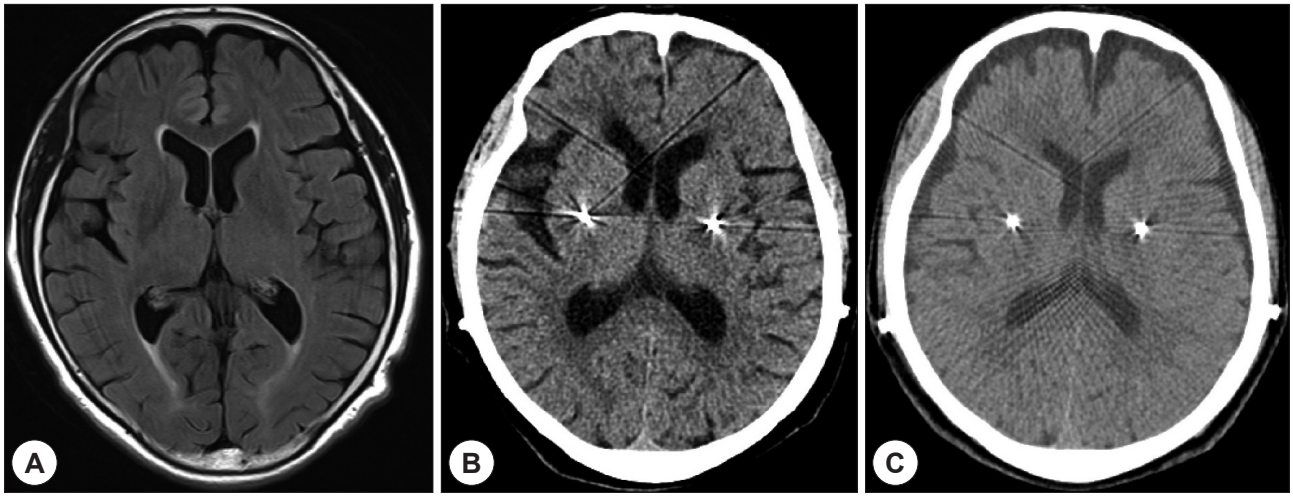

Fig. 1. A : Pre-operation magnetic resonance imaging (MRI) shows mild cortical atrophy. B : Post-operation computed tomography (CT) scan shows no abnormalities. C : CT scan on 7th postoperative day shows minimal bilateral hygroma possibly due to cortical atrophy.

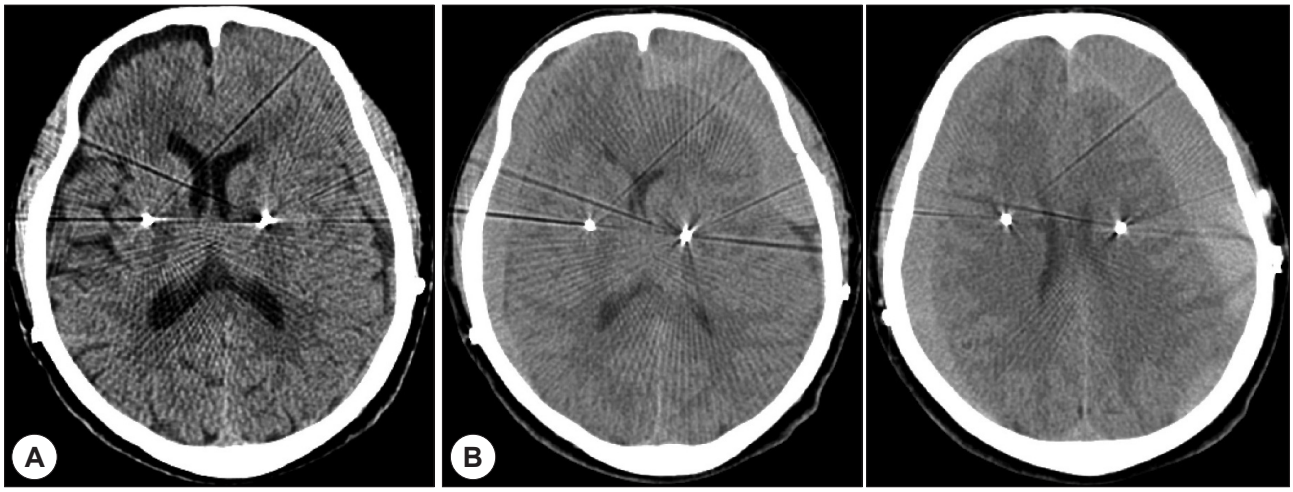

Fig. 2. A : Follow-up CT scan after 1 month from the operation shows newly developed subacute to chronic subdural hematoma, left cerebral convexity, without significant mass effect. B : Follow up CT scan after 2 month from the operation shows newly developed chronic subdural hematoma right cerebral convexity and increased amount of subdural hematoma, left cerebral convexity with heterogeneous density. Also CT scan reveals increasing mass effect of hematoma and midline shift to right. The electrode is medially shifted due to ventromedial brain displacement associated with chronic subdural hematoma.

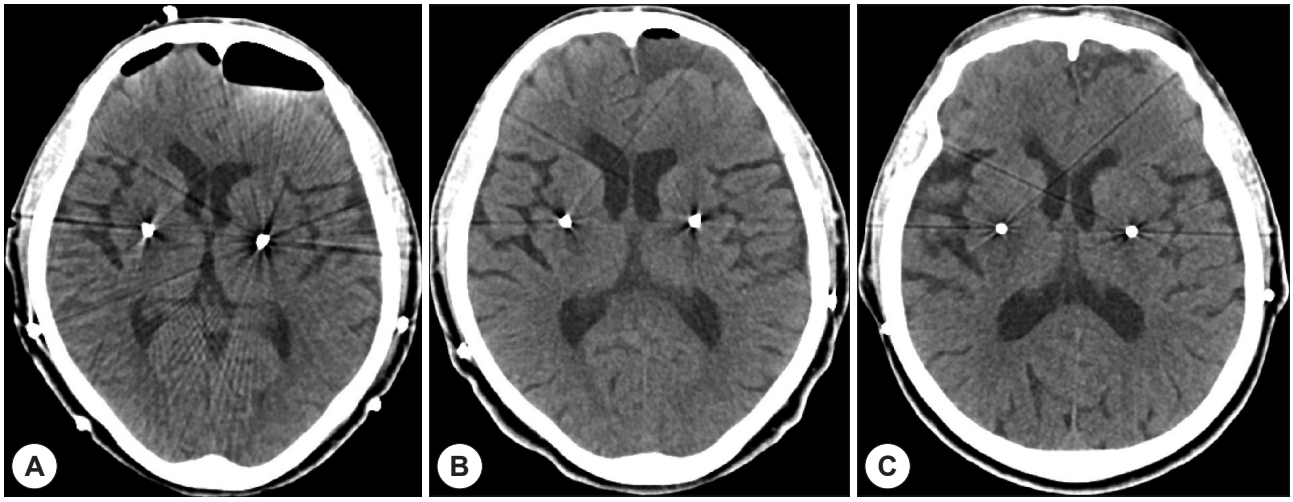

Fig. 3. A : Post-operative CT scan after bilateral burr-hole drainage. B : CT scan on 7th postoperative day. C : Follow up CT scan after 2 month from the bilateral burr-hole drainage shows resolving subdural hematoma. Also, the electrode displacement resolves with resolution of the hematoma.

In the operative field, after incision of the dura, dark chocolate colored liquefied hematoma was gushed out. Immediate postoperative CT scan showed improvement of midline shift (Fig. 3A).

Follow up CT scan on 7th postoperative day reveled that subdural hematoma was well removed with the improvement of pneumocephalus (Fig. 3B). The patient was discharged on the 7th postoperative day. After discharge, during the out-patient clinic follow-up, CT scan after 2 months from the bilateral burr-hole drainage showed no recurrence (Fig. 3C). After burr-hole drainage of the chronic subdural hematoma, the patient experienced significant benefit from the bilateral DBS therapy on follow-up.

DISCUSSION

Subdural hematoma after DBS is a rare operation-related complication, unless a patient has a falls or major head trauma. Genko, et al.[8] reviewed 9 published papers that reported a total of 11 SDHs that were observed as adverse event following DBS surgery (incidence rate range 0.2−4.2%).

Subdural hematoma commonly results from avulsion of cortical bridging veins. Markwalder.[7] studied that excessive leakage of CSF, and significant brain atrophy have the potential to increase the space between the dura mater and the pia mater, thus increasing tension on bridging veins. Therefore, excessive CSF leakage after dura incision should be prevented to reduce risk of delayed subdural hematoma. Also one should avoid the entry point where superficial or cortical vein exists or sulci traverses for preventing delayed subdural hematoma.

In this case, there was no history of head trauma and antiplatelet or anticoagulant use, but moderate brain atrophy in preoperative MRI (Fig. 1A), which is a usual finding of aged patient with Parkinson's disease. It is well known that brain atrophy is a potential prognostic factor of chronic subdural hematoma.[4] Retrospectively, the finding of bilateral subdural hygroma at the 7th postoperative day (Fig. 1C) might have been the stage of ongoing process to chronic SDH.[6]

Nowadays, progressive and chronic course of subdural hematoma is being studying and Edlmann, et al.[3] focuses on several key processes involved in chronic SDH: angiogenesis, fibrinolysis and inflammation. Understanding and research of the pathophysiology allow neurosurgeons to apply medical treatment to asymptomatic patient with chronic subdural hematoma. This might be helpful to an asymptomatic patient who underwent DBS lead implantation, because there is possible risk of electrode/lead migration during hematoma evacuation. Of course, evacuation for symptomatic and persistent chronic subdural hematoma is clinically necessary, and can be successfully performed without disruption of implanted electrodes as in this case. In our case, we performed bilateral burr-holes, one on each parietal eminence which was away from the frontal lead. We paid attention to the lead wire passage and planned the skin incision preventing dehiscence and hardware exposure through the scalp.

Follow-up CT scan after 2 months from the burr-hole drainage revealed that the electrode displacement resolved with resolution of the hematoma and no hardware revision was necessary (Fig. 3C). Yang, et al.[10] commented that gliosis formed along the track of the electrodes may function as a potential space. As a result, once the subdural hematoma is evacuated, the migrated electrode may glide back to its original location. Consequently, the DBS stimulation can remain effective without DBS revision surgery. When asymptomatic chronic subdural hematoma is managed conservatively without operation, it is bothersome to wait the electrode returns to near its initial position after spontaneous resolution of subdural hematoma.

Usually, it is known that levodopa dose can be reduced after subthalamic nucleus (STN) DBS but not after GPi DBS.[9] The anti-dyskinetic effect of STB DBS is believed through reducing the levodopa dose, but through direct effect in GPi DBS. The reduction of levodopa equivalent daily dose (LEDD) after the surgery in this case is not usual. The reason of dose reduction in this case was due to side effects of dopamine receptor agonist, mild psychosis and hallucination after the surgery. Simultaneous increasing the DBS voltage was not as effective to control patient's akinetic symptoms as have been observed intraoperatively. This might be due to gradual upward displacement of implanted electrodes from the original site by the mass effect due to chronic SDH (Fig. 2B).

We could learn several lessons from this case. Surgeons are generally careful avoiding CSF leakage during DBS surgery. Too much CSF leak may result in brain shift which may result in changes of target coordinates measured preoperatively. Brain shift may lead to acute subdural hemorrhage due to bridging-vein damage. We were also careful not to drain CSF by applying sufficient irrigation and surgical glue on the incised dura during the surgery, and immediate postoperative CT did not show any evidence of pneumocephalus or hygroma. The delayed subdural hygroma 1 week after the surgery might have been a warning sign of subsequent chronic SDH. The non-painful swelling on chest wall might have resulted from CSF leakage from the burr-hole site. The Stimloc device uses screws to secure it to the skull, but it does not always fit the curvature in our experience. We speculate the possibility of CSF leakage from the unfitted gap between the device and skull might have resulted in CSF collection on the swollen chest site, but there was no evidence.

Chronic SDH is characterized by insidious symptoms. Evolution from the subdural hygroma to chronic SDH is common which has been suggested as a natural course previously.[6] As seen in this case, even small amount of subdural hygroma may evolve to significant amount of chronic SDH. Serious results may occur in case of chronic SDH after DBS, if the physician does not identify the brain image and misinterprets the patient's akinetic symptoms as a result of reduced levodopa in the dose adjustment process.

CONCLUSION

Chronic SDH after DBS is a rare complication. A patient with over moderate brain atrophy needs more attention for chronic subdural hematoma. It may be prevented by avoiding excessive CSF leakage and a careful decision of an entry point in the operation, but not always possible as seen in the present case. The present case shows even small amount of postoperative subdural hygroma may change into a significant chronic subdural hematoma. Therefore, special attention and subsequent brain imaging are necessary, especially if there is a lack of anticipated DBS stimulatory effect.

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