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Hematoma-Related Nonhabitual Seizures during Subdural Electrode Monitoring

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Dear Editor,

Epilepsy surgery is an established treatment in patients with drug-resistant epilepsy that can eliminate or significantly reduce the seizure frequency. Invasive EEG monitoring is an invaluable technique for localizing and delineating the affected parts of the cortex in surgery planning for patients with intractable partial epilepsy.[1] However, invasive EEG monitoring has various complications related to the additional surgical procedures and the placement of foreign bodies in the brain or subdural space.[2]

A 31-year-old right-handed woman was admitted to the epilepsy clinic with drug-resistant seizures. She experienced frequent seizures consisting of a psychic aura, often progressing to loss of awareness and automatisms while taking oxcarbazepine and levetiracetam. Brain MRI showed a dysembryoplastic neuroepithelial tumor in the left temporal region. Interictal and ictal EEG showed that the maximum amplitude was over the left mesial temporal electrodes. Brain ^{18}F-fluorodeoxyglucose positron-emission tomography revealed hypometabolism in the left anterior-to-posterior temporal region. A multidisciplinary epilepsy committee recommended invasive monitoring.

A craniotomy was performed with left-sided subdural electrode grid placement, and 4×5, 4×8, 1×8, and 4×5 contact grid electrodes were placed in the left frontotemporal region. We used platinum contact electrodes embedded in 0.5-mm-thick flexible silicone plates (AD-Tech Medical Instrument, Racine, WI, USA). Three depth electrodes were inserted into the hippocampus.

The results of laboratory tests were normal, including the platelet count and prothrombin and partial thromboplastin times. Postoperative computed tomography showed no abnormalities other than the expected electrode artifacts.

The patient experienced three habitual automotor seizures during the first 4 days after electrode placement. During these seizures the rhythmic activity started over the left hippocampal region and spread to the basal temporal area. At 6 days after electrode placement the patient reported acute-onset tingling sensations in the left temporal area and strange feelings lasting several minutes that she not experienced previously. These feelings recurred throughout the day, as often as several times per hour. During a nonhabitual episode, the EEG showed localized rhythmic activity involving electrodes K3-K4 and K8-K9 over the inferior frontal region, which were not previously involved in her habitual seizures (Fig. 1A).

The subdural electrodes were removed 7 days after being placed, and a focal 3×2-cm well-formed hematoma was noted underlying the contacts of electrodes K3-K4 and K8-K9 over the suprasylvian frontal cortex (Fig. 1B). After evacuating the hematoma, slight indentations were evident in the underlying brain tissue, but there was no gross contusion or epileptiform discharge. An anterior temporal lobectomy with lesionectomy was performed.

The mechanism of subgrid hematoma may involve a tear or avulsion of the bridging veins, similar to that of subdural hematoma. A space present between the grid and cortex

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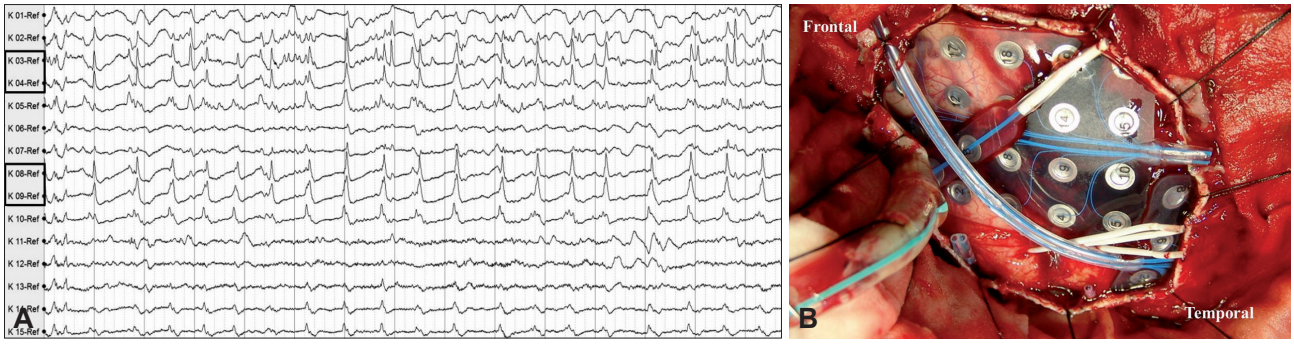

Fig. 1. A: Ictal EEG during a nonhabitual seizure. The EEG showed localized rhythmic activity involving electrodes K3-K4 and K8-K9 over the inferior frontal region, which was not previously involved in her habitual seizures. B: A left craniectomy performed 7 days after electrode placement revealed a focal 3×2-cm well-formed hematoma underlying the electrode contacts over the inferolateral frontal cortex.

during the early postoperative period after fixing a subdural grid to the dura can allow the influx of blood to the subdural/epidural space, with hematoma formation beneath the grid. Most patients exhibiting nonhabitual seizures during subdural grid implantation present with a very thin layer of blood under the implanted subdural grid upon electrode removal, with no evidence of a subdural hematoma.[3] Mechanical problems such as outstretching of the subdural grids or inappropriate cable connections can also lead to vascular damage in the subdural and subelectrode spaces.

One study found that the incidence of nonhabitual seizures was 10% during invasive EEG monitoring.[4] Although the mechanism underlying nonhabitual seizures is unknown, they could be a direct effect of the subgrid hematoma. The blood in a subgrid hematoma breaks down to form iron-containing products that could stimulate the underlying cortex and cause abnormal electrical activity so as to produce nonhabitual seizures. Activation of the coagulation cascade, which could be induced by the presence of a small postoperative subdural hematoma, has also been implicated in increased brain electrical activity and seizure production.[5] The subdural grid electrodes can also cause inflammatory changes that could increase the excitability of the underlying cortex, which could become a transient epileptogenic focus.[6]

Conflicts of Interest

The authors have no financial conflicts of interest.

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