A Focal Cortical Dysplasia Case whose Seizure Focuses were Detected Using Interhemispheric Grid Electrode: A Technical Case Report

Epileptojenik Alanı Interhemisferik Grid Elektrodla Belirlenen Fokal Kortikal Displazi Vakası

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ABSTRACT

We report an 18-year-old boy who presented with seizures at the age of 12 years. He had 3 different kinds of seizures, occurring 4 to 5 times a week, despite adequate antiepileptic treatment. Magnetic resonance imaging (MRI) showed deep paramedian focal cortical dysplasia in the left parietal lobe. Ictal video-EEG demonstrated 5 seizures originating from the left parietal lobe, left hemisphere and right hemisphere. He was submitted to invasive recordings with two frontal strips and an interhemispherical grid that was advanced to the corpus callosum. The lesion and other seizure focuses were resected subpially down to the corpus callosum approximately 1 cm posterior to the motor area. His seizures resumed after three years of quiescence. Postoperative MRI investigation revealed remnants of dysplastic tissue deep in the interhemispheric region, which were silent during the invasive EEG monitorization. We want to re-emphasize that each patient with cortical dysplasia who is a candidate for epilepsy surgery should undergo detailed neuroradiological investigations as well as EEG monitorization, whereas complete resection of the lesion on the basis of findings that are obtained from neuroradiological examination might have a superior effect on the postoperative seizure outcome compared to the findings obtained from EEG recordings.

KEYWORDS: Cortical dysplasia, Epilepsy surgery, Parietal epilepsy

ÖZ

Biz bu yazıda 18 yaşında olan ve 12 yaşından bu yana nöbet geçiren erkek hastayı sunduk. Yeterli dozda ve sürede uygulanan antiepileptik ilaçlara rağmen haftada 4-5 kez olmak üzere 3 farklı tipte epileptik nöbetleri devam etmekteydi. Kranial MRI'da sol parietal derin paramedian yerleşimli fokal kortikal displazi alanı görüldü. İktal video EEG monitorizasyonunda sol parietal, sol ve sağ hemisfer kaynaklı 5 adet nöbet kaydedildi. Hastaya 2 frontal strip ve korpus kallosuma kadar uzanan interhemisferik grid elektrod yerleştirilerek invaziv EEG kayıtları elde edildi. Lezyon diğer epileptik odaklarla birlikte motor alanın 1 cm. posteriorundan subpial olarak rezeke edildi. Hasta postoperatif dönemde 3 yıl nöbet geçirmedi. 3 yılın sonunda nöbetleri yeniden başlayan hastanın yapılan MRI tetkikinde derin interhemisferik yerleşimli, daha önceki EEG monitorizasyonunda sessiz olan displastik doku artıkları gözlendi. Bu yazıda, epilepsi cerrahisi adayı olan fokal kortikal displazi vakalarında etkin postoperatif nöbet kontrolu için ayrıntılı nöroradyolojik incelemelerle belirlenen lezyon rezeksiyonunun, EEG monitorizasyonu ile belirlenen epileptojenik doku eksizyonundan daha effektif olduğu vurgulanmak istendi.

ANAHTAR SÖZCÜKLER: Epilepsi cerrahisi, Kortikal displazi, Parietal epilepsi

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INTRODUCTION

Focal cortical dysplasias (FCDs) are being increasingly recognized as a cause of intractable epilepsy, thanks to improved neuroimaging techniques(4,5,11). Total resection of the lesion is an effective treatment modality in these patients(1,6,14). However, even when the resection is possible, results can still be unsuccessful(20). This may be due to the inability to fully demonstrate the relation between the epileptic focus and the lesion, a wider epileptic focus than the defined lesion, or because of constraints on the resection of complicated areas of the brain with dysplastic involvement(3,9,16).

We present here a case of focal cortical dysplasia whose seizure focuses were detected by an interhemispheric grid located on the lesion. The patient was operated on, but his seizures resumed after three years of quiescence. A postoperative magnetic resonance imaging (MRI) examination revealed remnants of the lesion in the interhemispheric area.

CASE REPORT

The patient was an 18-year-old boy, referred to our epilepsy center by his physician for intractable seizures. Birth and development history were normal. Simple partial seizures had developed three years ago, heralded by an aura of electricity feeling and tingling of the right arm and leg, lasting 5-15 s. He sometimes had weakness in the arm and leg, as well as loss of muscle tone lasting 20-40 s after the aura. There was no impairment of consciousness. This initial seizure semiology was suggestive of parietal lob epilepsy. His routine electroencephalograms (EEGs) indicated an epileptic abnormality originating from the parietal area of the left hemisphere, which had a tendency to generalize. The seizures occurred two-three times a week, although Valproic Acid (VPA) therapy had been started. Three months after the simple partial seizures had started, extratemporal-type complex partial seizures, lasting 30-60 seconds in the form of blank looks in the eyes, mouth smacking, uttering of meaningless words, and automatism in both hands were initiated. There was impairment of consciousness followed by postictal confusion. Over the next six months, his seizure frequency gradually increased despite adequate trials of VPA, Mysolin and Oxcarbazepine (OXC). MRI showed an area of

cortical dysplasia in the grey matter of the posterior section of the parietal lobe which was located in the deep paramedian region (Figure 1).
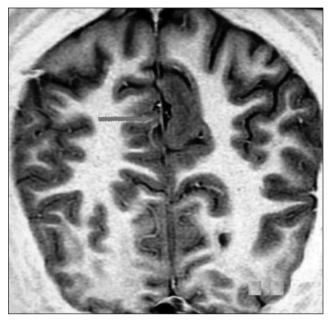

Figure 1. *T1–weighted coronal Magnetic Resonance Imaging (MRI) shows the cortical dysplastic area (arrow), which has a deep paramedian location in the grey matter of the posterior section of the left parietal lobe.*

At the time of admission to our Epilepsy Monitoring Unit, he had therapeutic serum levels of VPA and OXC. The physical and neurological examination findings were normal. Five seizures were observed during the 4-day long-term scalp video EEG. His first three seizures were in the form of extratemporal complex partial seizures lasting 45-60 seconds, which had been previously defined by the patient. However, a generalized tonic-clonic seizure lasting 40-45 seconds, not mentioned by the patient, was observed following the complex partial seizures. In the fifth seizure, the patient slowed down realizing that the seizure was coming and, after 20 seconds, an automatism developed in an extremity, as observed in temporal lobe seizures, together with dystonia in another extremity. This situation was interpreted as the spreading of epileptogenic discharges to the temporal lobe, for it was observed at the end of the seizure. The ictal EEG changes belonging to these seizures started from the P3 electrodes in the first two seizures, from the left hemisphere in the third and fifth seizures, and from the right hemisphere in the fourth seizure. The interictal EEG showed a slowdown in the left

hemisphere's centroparietal and posterior temporal areas activated by sleeping, and sharp wave discharges in the form of isolated sharp waves and sometimes bilateral synchrony. A complete correlation between the lesion defined in the MRI and the origin of the seizure could not be delineated as the patient had a hemisphere-originated seizure and the lesion was located in the parietal region, a complex area, and long-term extraoperative invasive EEG monitorization was planned. We therefore arranged placing an interhemispheric grid, which could surround the lesion tightly. A left central craniotomy was performed and a 32-contact grid electrode was lowered to the corpus callosum so that it covered the paracentral lobule and surrounded the lesion interhemispherically at a single stage (Figure 2A,B).

In the current case, anatomical features of the drainage veins in the midline allowed us to place the grid electrode interhemispherically down to the corpus callosum. Two strip electrodes were also placed in parallel to the superior and middle frontal gyri (Figure 2C), and the dura and bone were closed loosely. The patient had a total of 4 seizures during the next three days. All the electroclinical seizures started either from the No. 4,12,19 grid electrodes in the deep interhemispheric areas surrounding the lesion seen in MRI or No. 5,6,13,14 grid electrodes in the parietal cortical areas (Schematic Figure 3). As a result of the cortical stimulation, the number 29,30, 31, and 32 electrodes were determined as the motor area. A very low-voltage cortical stimulation in the 15th electrode produced a seizure. The lesion that contained all of the seizure focuses, including the seizure focus approximately 1 cm posterior to the motor area, was resected subpially, and the resection was carried down to the corpus callosum in the interhemispheric fissure (Figure 4). Pathological investigation revealed TypeII cortical dysplasia. The patient had no seizures or neurological defect in the 3 years following the surgery, after which seizures resumed. Postoperative MRI examination revealed remnants of dysplastic tissue deep in the interhemispheric area (Figure 5). A second intervention was offered to the patient upon the finding that epileptic discharges originated from the lesion detected on the MRI examination, but he refused reoperation as had been in the seizure-free stage again for the last 6 months.
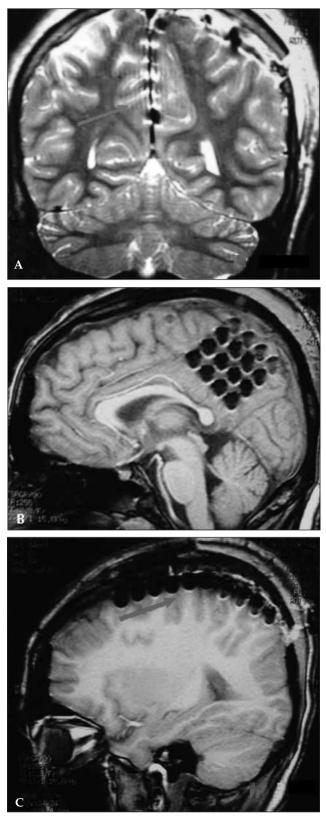

Figure 2. Appearances of the strips and grid in MRI; *A) T2–weighted coronal images (red arrow) shows the grid surrounding the lesion; B,C) T1–weighted sagittal images (red arrow) shows the position of the interhemispheric grid and location of the strip electrodes placed in the left frontal area.*
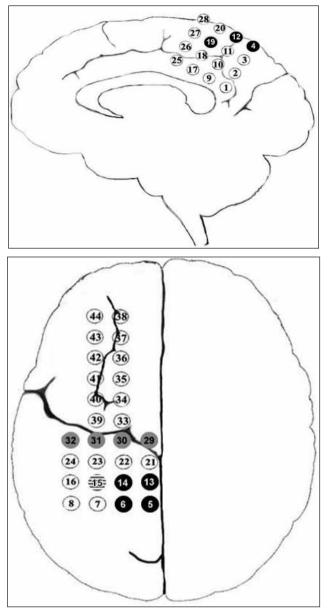
Figure 4. *Operative appearance of the lesional area and appearance after lesionectomy.*
Green electrodes (from left to right; electrodes No. 32,31,30,29) correspond to the motor area and blue electrodes (Electrodes No. 19,15,14,13,5,4) to seizure focuses.*

Figure 3. *Left interhemisferic and cortical areas. Schematic locations and numbers of the interhemispheric grid and strip electrodes. No. 4,5,6,12,13,14,19 electrodes are the areas of seizure initiation.*

DISCUSSION

In this case, MRI examination, seizure semiology and EEG monitorization failed to demonstrate the association between the epileptic focus and the lesion. A 32-contact grid electrode was then located on the lesion interhemispherically and functional mapping was performed. Successively, the lesion was excised along with all epileptogenic foci detected. However, the patient's seizures resumed after three years.
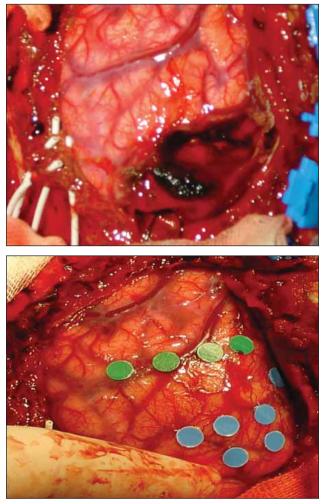
For each pathogenetic entity and underlying pharmacoresistant epilepsy, it is crucial to establish effective approaches to presurgical evaluation and operative techniques that will lead to consistently successful outcomes.(2) In our patient, FCD was located in the deep interhemispheric area of the parietal lobe. Although our patient's seizure semiology and ictal EEG specifications indicated the lesion site, he had three different seizures and a seizure originating from the right hemisphere where no lesion was present. No matter how small the parietal lobe is compared to the frontal and temporal lobes, determining the accurate localization of parietal lobe epilepsy (PLE) is difficult due to its inconsistent semiologic features.(21) The most
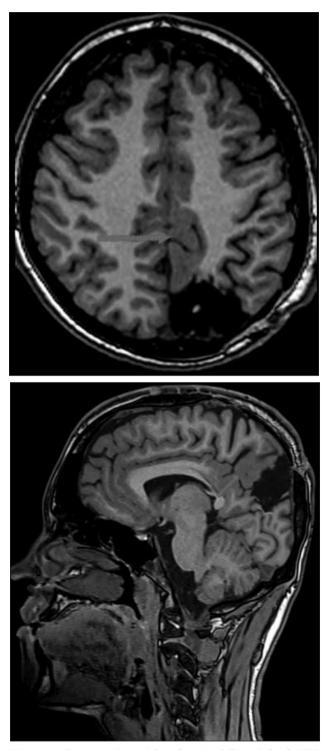
Figure 5. Postoperative axial and sagittal T1-weighted MRI scans show the residual cortical dysplastic tissue (red arrow) anterior to the resection area.

common problem encountered in evaluating PLE patients for surgery is poor EEG localization of interictal spikes and of seizure onset(15,19,21).

If the lesion has both superficial and deep interhemispheric cortical locations, as was in our case, the efficacy of surface EEG recordings in determining the seizure focus is limited because of the difficulty with electrode placement on the lesion and spread of the seizure activity relatively rapidly from the parietal lobe to the adjacent areas(8,10,12,19). These characteristics are major obstacles to successful surgical treatment. Long-term extraoperative invasive EEG monitoring with intracranial grid and strip electrodes is the ultimate step in establishing the location of seizure origin(12,17,18). However, it can be difficult to detect the seizure focus with invasive EEG monitorization or scalp EEG when the lesion has a deep interhemispheric location in the parietal area. In order to overcome this problem, we placed the grid interhemispherically down to the corpus callosum.

Patients with FCDs within complex brain structures such as the parietal area should be approached with functional mapping techniques.(1) In subjects having resistant seizures originating from the parietal area, surgical resection may lead to deficits in vision, language, praxis, attention, and higher cortical functions(18,21). Extraoperative mapping with subdural grids is therefore required before resection to detect the functional cortex and epileptogenic tissues in these patients(1). We used electrical stimulation applied to all grid electrodes excluding the seizure-initiating electrode, and seizure focuses were detected as well as the eloquent cortical areas. As a result, the lesion containing all of the epileptic foci, one just one centimeter posterior to the motor cortex, was resected subpially.

The key to the surgical success in FCD is to remove the lesion as widely as possible after determining the relation between the lesion and the seizure focus. This may not always be possible due to the location of the lesion(21). The process includes some difficulties including determination of the association between the lesion and epileptic foci and determination of the exact size of the lesion. The patient's seizures resumed despite the excision of all epileptogenic foci along with the lesion. Subsequent MRI examination proved a remnant of dysplastic tissue in the lesional area.

This case proves the importance of determining the exact size of the lesion by using neuroradiological investigations in cases of cortical dysplasia, especially those which are located in deep-interhemispheric region, to achieve an optimal

surgical outcome. It also points to the importance of complete removal of the lesion, which may not be detected by invasive EEG recordings.

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