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Frontal Lesional Epilepsy: A Case Report

Frontal Lezyonal Epilepsi: Bir Olgu Sunumu

Ayşe KARATAŞ[1]
Gülnihal KUTLU[2]
Erdal YILMAZ[1]
Ayşe SERDAROĞLU[3]
Erhan BİLİR[3]
Atilla ERDEM[1]

1 Ankara University School of Medicine, Department of Neurosurgery
2 Ministry of Health Ankara Training and Research Hospital, Department of Neurology
3 Gazi University School of Medicine, Department of Pediatric Neurology

ABSTRACT

Extratemporal epilepsies are difficult to detect and to localize electrographically. The surgical success rate in extratemporal lobe epilepsy (ExTLE) has been low but might be increased by using invasive electrodes and functional mapping. We describe the preoperative evaluation and surgical approach for an ExTLE patient.

A 31-year-old man underwent intracerebral left frontal lobe abscess drainage and capsula excision four years ago. He was re-admitted with a seizure nine months after the surgery. His typical seizure was a secondary generalized tonic clonic seizure that was resistant to antiepileptic drugs. Video/EEG monitorization was performed, first with scalp electrodes and then with the subdural grid electrode. The Ojemann Cortical Stimulator was used for functional brain mapping. Language function and the epileptogenic focus area were determined. Cortical resection of the epileptogenic focus and the area causing the aura was performed with protection of motor and language function.

The postoperative course was uneventful. No postoperative deficit was observed. The patient was seizure free during two-year follow up.

Extratemporal resections must be done carefully after determining the seizure focus and the type of seizure. The safe resection boundary is defined with functional mapping. Identification of the primary motor cortex and the language cortex is necessary to avoid motor deficits and speech difficulties respectively.

KEY WORDS: Epilepsy, invasive monitoring, functional mapping

ÖZ

Ekstratemporal epilepsileri tesbit etmek ve elektrografik olarak lokalize etmek zordur. Ekstratemporal lob epilepsilerinde (EkTLE) cerrahi başarı oranı düşüktür, ancak bu oran invaziv elektrodlar ve fonksiyonel haritalama uygulamaları ile arttırılabilir. Bu yazıda bir EkTLE olgusunda cerrahi öncesi hazırlık ve cerrahi yöntem anlatılmaktadır.

31 yaşındaki erkek hastaya 4 yıl önce sol frontal intraserebral abse drenajı ve kapsül eksizyonu uygulanmıştır. Hasta operasyondan 9 ay sonra nöbet şikayeti ile başvurmuştur. Hastanın tipik nöbeti sekonder generalize tonik klonik nöbet şeklinde ve bu nöbetler antiepileptik tedaviye dirençli idi. Hastaya ilk olarak saçlı deri ve daha sonra subdural grid elektrod ile video/EEG kaydı yapıldı. Bu işlemden sonra Ojemann'ın kortikal stimülatörü ile fonksiyonel beyin haritalaması uygulandı. Lisan fonksiyonu ve epileptik foküs alanı tayin edildi. Epileptojenik foküs ve aura hissedilen alanın kortikal rezeksiyonu motor ve lisan fonksiyonları korunarak uygulandı.

Postoperative dönemde problemi ve defisiti olmayan hasta 2 yıldır nöbetsiz olarak takip edilmektedir.

Ekstratemporal rezeksiyonlar nöbet başlangıç odağı ve nöbet tipi belirlenerek dikkatli bir şekilde yapılmalıdır. Güvenli rezeksiyon sınırı fonksiyonel haritalama ile tanımlanır. Motor defisitlerin önlenmesi için primer motor korteksin ve konuşma zorluklarının önlenmesi için lisan korteksinin tanınması gereklidir.

ANAHTAR SÖZCÜKLER: Epilepsi, invaziv monitorizasyon, fonksiyonel haritalama

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Correspondence Address:
Gülnihal KUTLU
SB Ankara Eğitim ve Araştırma Hastanesi Nöroloji Kliniği
Ulucanlar-Ankara, Turkey
Phone: +90 312 363 33 30/1090
GSM : 532 4173376
e mail : drgkutlu@hotmail.com

INTRODUCTION

The development of video/EEG monitoring, MRI, PET and SPECT have made it possible to define the epileptogenic focus and have helped the selection of candidates for surgery among epileptic patients. Candidates for epilepsy surgery are those that are medically refractory, those with certain types of epilepsy syndromes and some patients with brain abnormalities such as tumors or brain malformations. This group makes up approximately 20% of all epileptic patients (4). The surgical success rate is quite high in temporal lobe epilepsy (4) but lower for extratemporal lobe epilepsy (ExTLE) (1, 2, 10). Monitoring with invasive electrodes has been carried out in ExTLE during the last few years making it possible to determine the exact epileptogenic focus and thus increase the surgical success rate (1). We present the preoperative evaluation and surgical approach of patient with ExTLE.

CASE REPORT

A 31-year-old male right-handed patient was operated on for a left frontal brain abscess at Ankara University İbn-i Sina Hospital, Department of Neurosurgery in March 2000. Intracerebral abscess drainage and capsula excision were performed. Only minimal motor dysphasia was present after the operation. He had no complaints for 8 months postoperatively. Antiepileptic treatment (diphenylhydantoin and carbamazepine) was continued regularly.

Epileptic seizures started 9 months after the operation. He defined tiredness and phonophobia as an aura and the seizure started with tremor and contraction of his hands and lost of consciousness. Foaming at the mouth was sometimes seen. This condition lasted 1-2 minutes and he was awake after half an hour. This seizures were seen once or twice a month. He had acute rheumatic fever and brain abscess operation in his medical history. A left frontoparietal insicion scar and minimal motor dysphasia were present. The left hemisphere was determined as dominant by clinical and neuropsychological tests. A craniotomy defect and encephalomalasia over the left frontal area were observed in his cranial MRI films (Figure 1, 2).

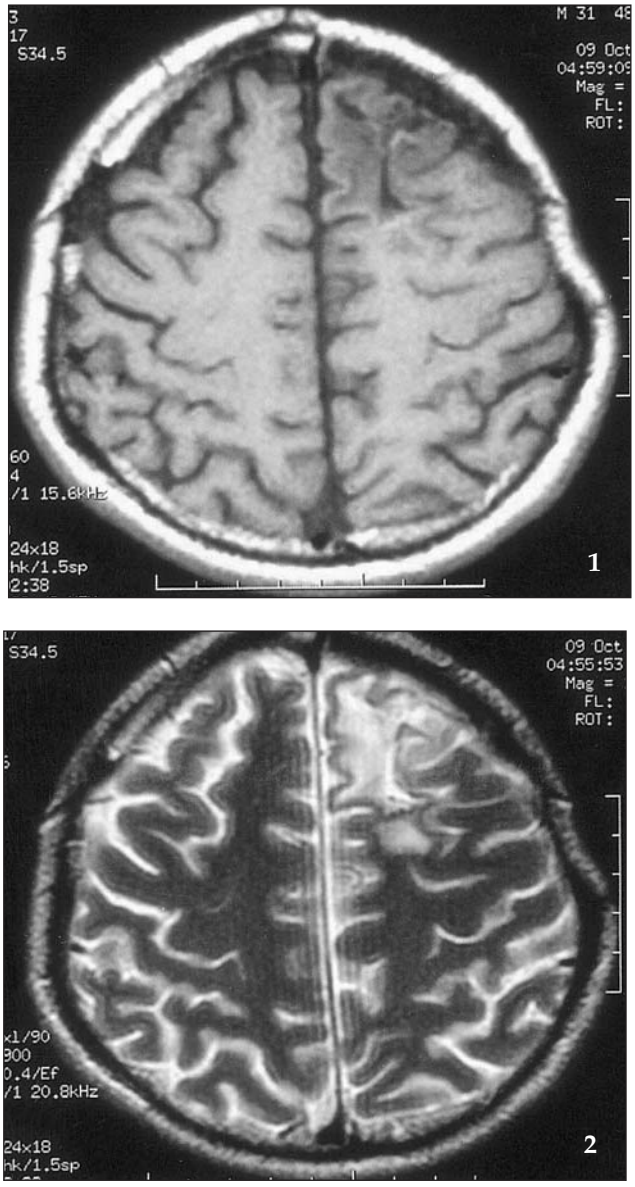


Figure 1, 2: Preoperative, T1- (1) and T2-weighted (2) magnetic resonance imaging scans, showing the craniotomy defect and encephalomalasia at the left frontoparietal area.

Sodium valporate 1500 mg/day and lamotrigine 250 mg/day were started after the first seizure. He was admitted to the Gazi University Faculty of Medicine Telemetry Center in August 2001 for the classification and localization of the seizure, and also for preparation for surgery if necessary. Video/EEG monitoring including additional anterior temporal electrodes and EEG with 32 channels were recorded during four days. 2-3 Hz continuous polymorphic delta wave and intermittant sharp waves were seen at the left central area during sleep and wake

interictal EEG (Figure 3). He had two seizures during this period which were secondary generalized tonic clonic seizures during which the patient suddenly vocalized, turned his head to the right for 5-6 seconds and had generalized tonic movement in all his extremities after tonic clonic movements of his right arm. This seizure lasted 80 seconds. Before the clinical seizure started, high amplitude sharp waves lasting 1.5 seconds at first and 4 seconds afterwards were seen at the left central region, especially at the C3 electrode position in his EEG. After the voltage supression in left hemisphere, slow and low amplitude sharp waves in the left temporal region were observed for 1 second. Slow and low amplitude sharp waves were seen in the right hemisphere after 6 seconds. Muscle artefacts in the EEG followed during generalized tonic clonic seizures. There were no postictal EEG changes after the seizures.

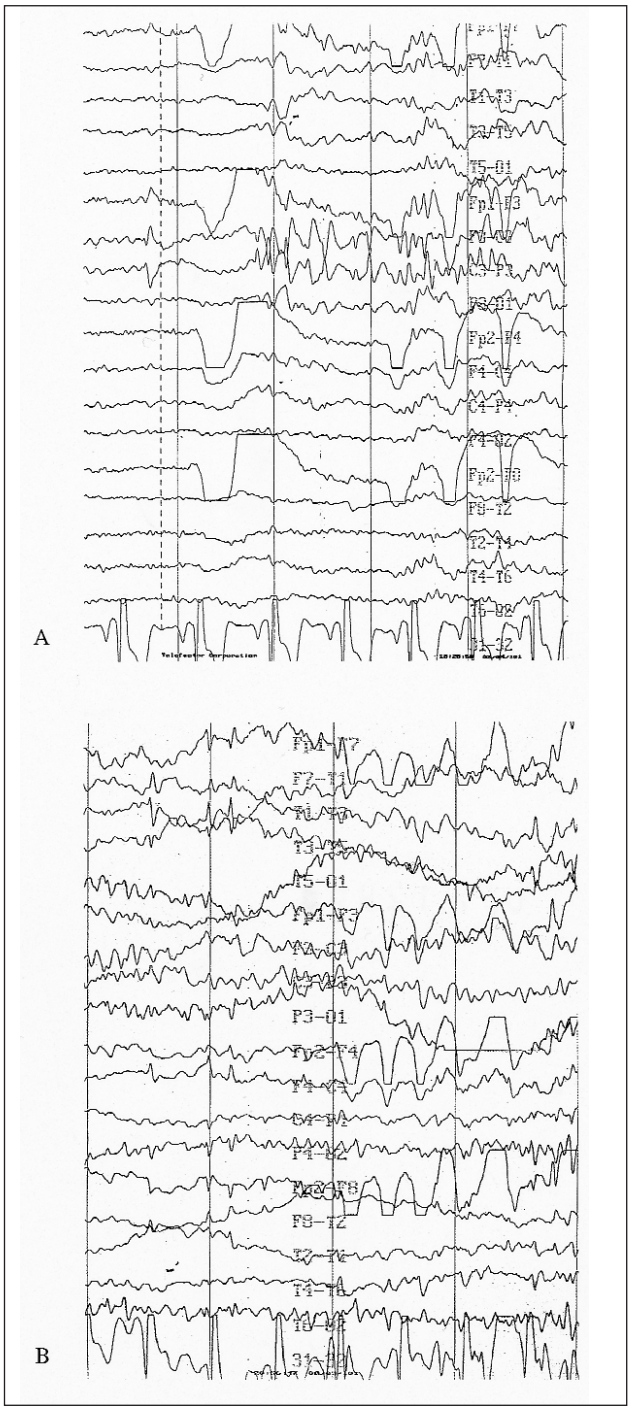


Figure 3: EEG 1: The beginning (**A**) and spreading (**B**) of the seizure in scalp video/EEG monitorization.

After the first monitoring, his typical seizure was thought to be secondary generalized tonic clonic. Interictal and ictal EEG changes started at the left central region C3 electrode position and generalized quickly. It was decided to remonitor the patient using the subdural grid electrode. A subdural grid electrode with 64 contact points was placed over the cortex via a left frontoparietal recraniotomy on October 2001 (Figure 4). The craniotomy was not big enough to place a grid electrode with 64 contact points as it had been planned for an abscess drainage procedure. This problem was solved by dissecting cortical-dural adherents to provide space for inserting the grid electrode. After this procedure, he was monitored again at the Gazi University School of Medicine Telemetry Center. The area of electrodes 33-64 which was probably the epileptogenic focus

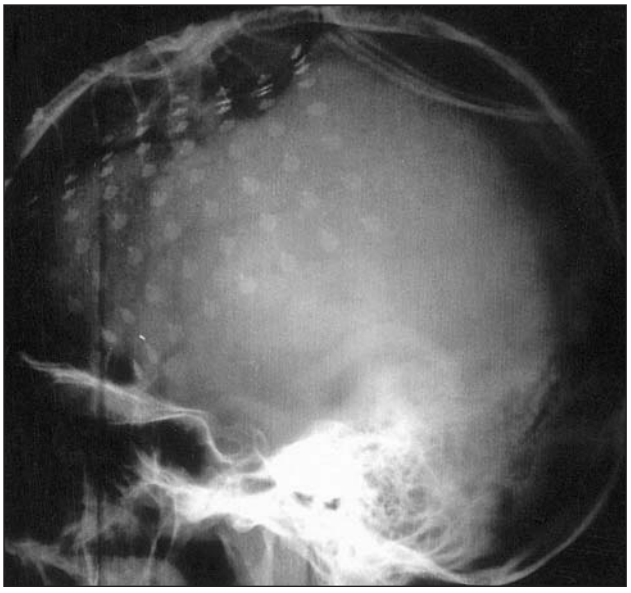


Figure 4: Lateral cranial X-ray showing the subdural grid electrode.

was evaluated first, followed by the area of 1-32 electrodes. He had no seizures during four days of monitoring. Continuous polymorphic delta activity and sharp waves were seen at the 39, 40, 46, 47, 48, 54, 55, 56, 63 and 64 electrode positions in the interictal EEG which were recording the encephalomalasic area seen on cranial MRI (Figure 5). He had no seizures during the first two days. We did not wait to record an ictal EEG because of the risk of grid electrode infection and brain edema.

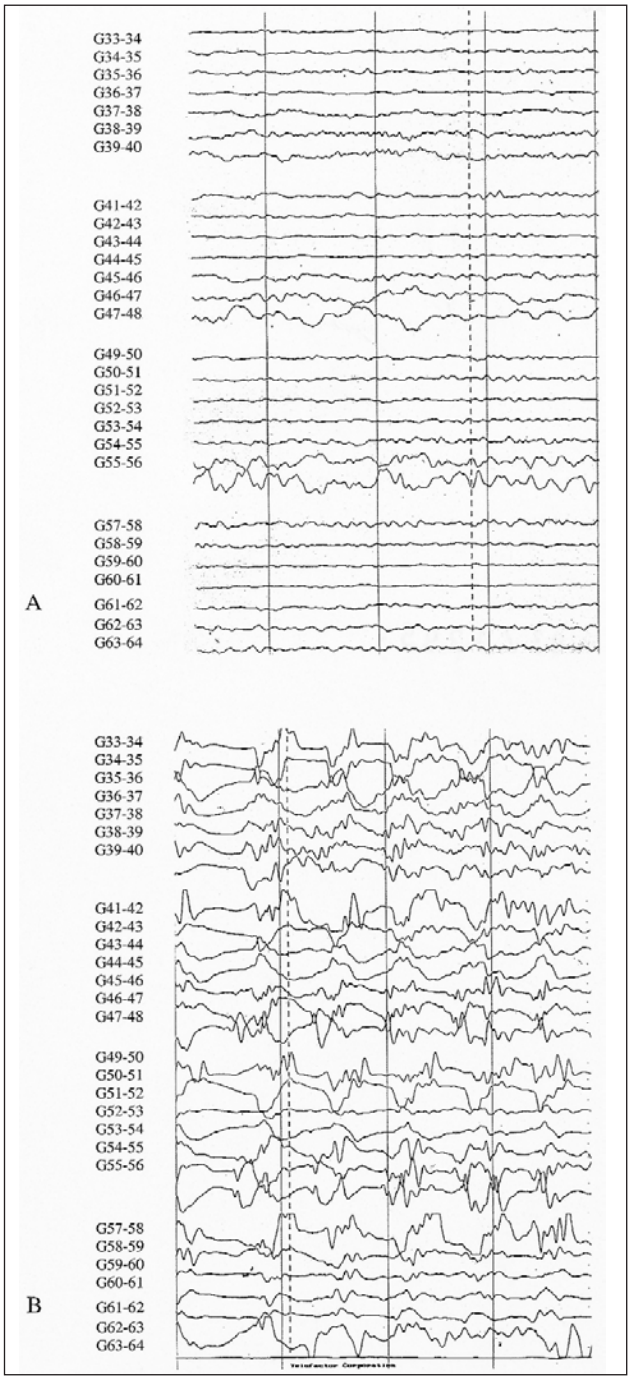


Figure 5: EEG 2: Interictal EEG findings obtained by using the subdural grid electrode (**A,B)**.

We used the Ojemann Cortical Stimulation (OCS-1, Radionics-Massachusetts-USA) for cortical mapping on the third day of hospitalization as the lesion was very close to the motor and language cortical area (Figure 6). Electrodes were removed from their place during bipolar stimulation.

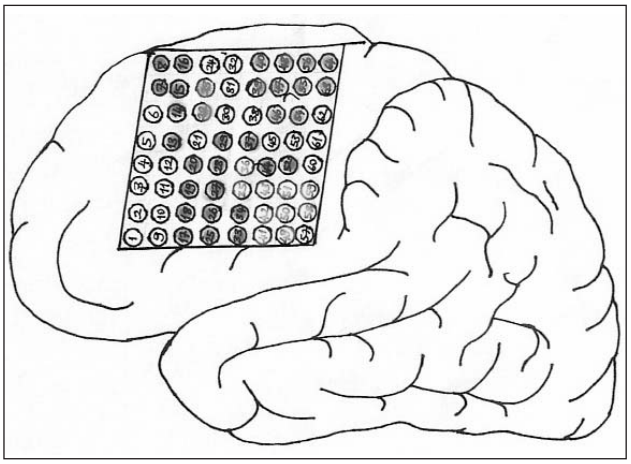


Figure 6: Grid electrode position in robotic picture.
Normal area (blue)
Electrodes 7-8 (stimulated until 5 mA)
Electrodes 13-14 (stimulated until 5 mA)
Electrodes 15-16 (stimulated until 5 mA)
Electrodes 17-25 (stimulated until 8 mA)
Electrodes 18-26 (stimulated until 8 mA)
Electrodes 19-27 (stimulated until 8 mA)
Electrodes 20-28 (stimulated until 8 mA)
Electrodes 29-37 (stimulated until 8 mA)
Electrodes 33-34 (stimulated until 7 mA)
Electrodes 44-52 (stimulated until 8.5 mA)
Lesion localization (red)
Electrodes 39-40-46-47-48-54-55-57 (not stimulated)
Electrodes 63-64 (stimulated until 5 mA) Seizure was observed
Area of aura (green)
Electrodes 22-23 (stimulated until 5 mA) Aura was observed
Language area (yellow)
Electrodes 35-36 (stimulated until 7 mA) Reading difficulty
Electrodes 41-49 (stimulated until 7.5 mA) Anomia, mild dysartria
Electrodes 42-50 (stimulated until 7.5 mA) Anomia, mild dysartria
Electrodes 58-59 (stimulated until 7.5 mA) Anomia, dysartria
Electrodes 43-51 (stimulated until 9.5 mA) Tongue dysesthesia

Afterdischarges were not seen in this electrode position because of technical reasons. However, continued EEG monitoring was used when volume conduction was present in the neighbouring electrodes during stimulation. The intensity of stimulation was increased until volume conduction in the neighbouring electrodes and clinical signs were observed. Two electrodes were stimulated for 5 seconds, starting at 0,5 mA. The stimulation was incresed by 0,5 mA each time until motor and language dysfunctions appeared. The patient developed a typical secondary generalized tonic clonic seizure after stimulation of electrodes 63-64 (5 mA) on the first day of stimulation and this indicated that the focus was near the encephalomalasic area. This seizure appeared as a result of volume conduction. We continued the mapping on the next day as the patient experienced postictal confusion after the seizure. He had tongue dysesthesia during stimulation of electrodes 43-51 (9,5 mA) and anomia and mild dysartria during stimulation of electrodes 58-59, 41-49 and 42-50 (7,5 mA). Reading difficulty was seen with stimulation of electrodes 35-36 (7 mA). He experienced aura with stimulation of electrodes 22-23 (5 mA). There was no clinical symptom with the stimulation of electrodes 7-8, 13-14, 15-16, 17-25, 18-26, 19-27, 20-28 and 33-34 (8 mA). Other electrodes were not stimulated. The EEG was carefully observed during cortical stimulation.

The language function was evaluated and the potential epileptogenic focus determined after this brain mapping. He underwent left frontoparietal recraniotomy on October 2001. The epileptogenic focus and the area that caused aura were marked and frontal cortical resection was performed (Figure 7, 8, 9). The resection was extended to the area that was not stimulated before the operation, to the adjacent midline falx, and inferiorly to the gyrus cinguli. His motor dysphasia worsened in the early postoperative period, but disappeared during follow-up. Gliosis and meningial reactive changes were confirmed pathologically. He was seizure-free on his outpatient visit two years after surgery. The patient's neurological examination was found to be normal at the last postoperative follow-up two years after the operation and he was only taking sodium valproate 1000 mg/day.

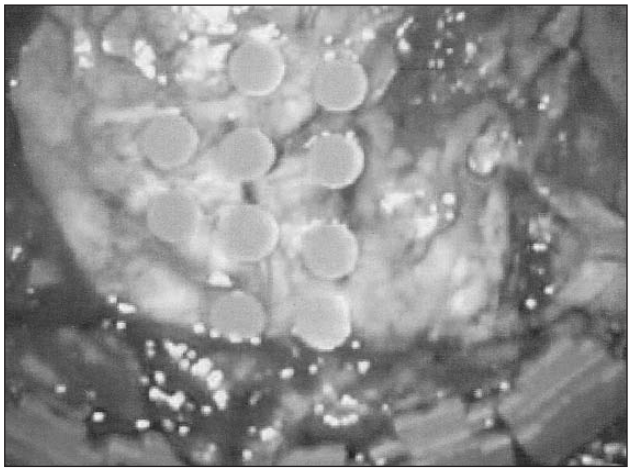
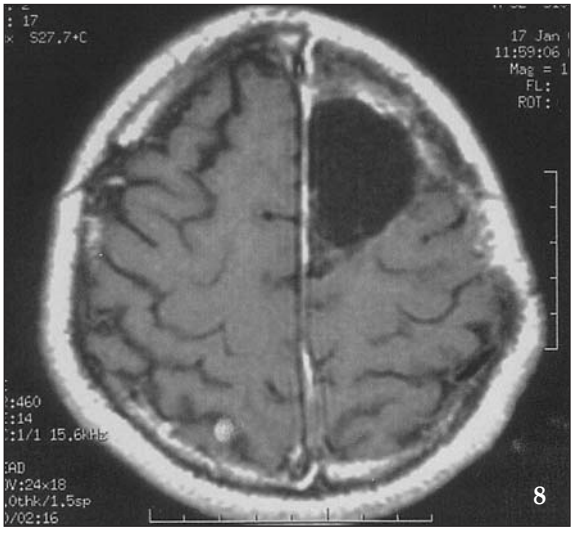


Figure 7: Intraoperative photograph, showing that the epileptogenic focus and the area causing the aura are marked.



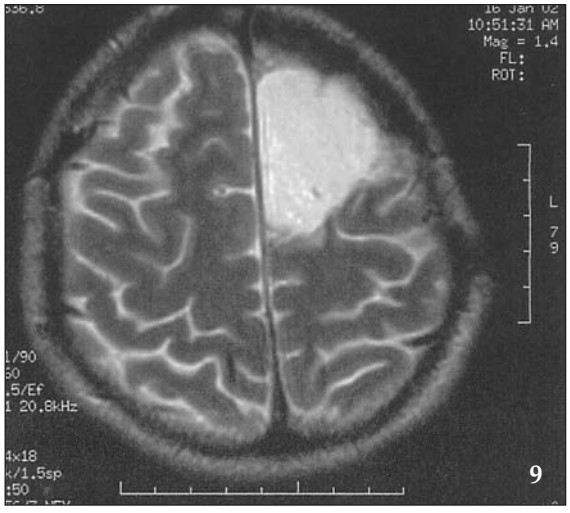


Figure 8, 9: Postoperative, T1- (8) and T2-weighted (9) magnetic resonance imaging scans, demonstrating resection of frontal cortical area.

DISCUSSION

The surgical success rate in temporal lobe epilepsy is 62-96% in previous studies (4). However, this rate is lower in ExTLE. 30-50% of the patients have been reported to be seizure-free after frontal lobectomy. Seizures decreased in 20-40% of the patients but there were no changes in 25-35% of the patients (1). The surgical success rate in seizures originating from parietal and occipital lobes is not high (11, 12) but higher if the seizures are accompanied by a structural abnormality (1). Invasive monitoring for seizure localization and determination of ictal findings is used in cases without structural abnormality (11, 12) to increase surgical success.

The incidence of epilepsy following brain abscess is reported as about 40% (6, 7, 8). The exact mechanism responsible for the development of epilepsy following brain abscess is still uncertain. The structures of the brain affected by the inflammatory process probably initiate the changes that will form an epileptic focus in the future (6).

Intracranial electrodes, especially subdural grid and strip ones, have been used for the determination of the focus in ExTLE. The placement of subdural strip electrodes is easier than the grid ones and craniotomy is not necessary to place them. The risk of infection and intracranial hemorrhage is lower than with other invasive electrodes. However, subdural strip electrodes evaluate only a small area of the brain. On the other hand, subdural grid electrodes examine larger brain areas but the complication rate is higher. The most important complications reported were intracranial hemorrhage and infection. A large brain area has been evaluated and functional brain mapping performed with subdural grid electrodes (2, 3, 5, 13). Localization of the primary motor cortex and language cortex is essential to avoid motor deficits and speech difficulties respectively in patients undergoing cortical resection of the dominant hemispheres (9).

In our case, we considered that the seizures might be originating from the left central area after the first monitoring. This area correlated with the structural abnormality. The abnormal intensity area was located in front of the central sulcus, mainly in area 8 and partly in area 6. However, we had to identify which area of this structural abnormality the focus originated from. The location of the lesion was very close to the motor and language cortical areas. Invasive monitoring by using the subdural grid electrode was therefore necessary to identify the adjacent cortical functional areas. Resection of the epileptogenic focus and the area causing the feeling area of aura was guided by the localization data received from monitoring with the subdural grid electrode and detailed cortical functional mapping helped safe resection. The maximum safe resection boundary provided very successful control of the seizure. Our primary goal in brain mapping was the determination of the epileptogenic focus, limiting the surgery and the evaluation of cortical motor and language functions. The area causing the aura was resected as it carried a risk of becoming a refractory epileptogenic focus.

The same indications and technical methods used by many epilepsy centers were used for preparing this case for surgery. However, the fact that this case arised secondary to a brain abscess must be emphasized. There were therefore some important surgical problems such as adhesive tissue and a small craniotomy. All these risks were taken so that the patient could have a seizure-free life postoperatively.

REFERENCES

1. Cascino GD, Shorbrough FW, Trenerry MR, Marsh WR, Kelly PJ, So E: Extratemporal resections and lesionectomies for partial epilepsy: complications and surgical outcome. Epilepsia 35; 1085-1090, 1994
2. Cukiert A, Buratini JA, Machado E, Sousa A, Vieira JO, Argentoni M, Forster C, Baldauf C: Results of surgery in patients with refractory extratemporal epilepsy with normal or nonlocalizing magnetic resonance findings investigated with subdural grids. Epilepsia 42 (7); 889-894, 2001
3. Diehl B, Lüders HO: Temporal lobe epilepsy: When are invasive electrode needed. Epilepsia 41 (Suppl. 3); 61-74, 2000
4. Gilliam F, Faught E, Martin R, Bowling S, Bilir E, Thomas J, Morawetz R, Kuzniecky R: Predictive value of MRI-identified mesial temporal sclerosis outcome in temporal lobe epilepsy: An intent to treat analysis. Epilepsia 41 (8): 963-966, 2000
5. Ikeda A, Miyamoto S, Shibasaki H: Cortical motor mapping in epilepsy patients: Information from subdural electrodes in presurgical evaluation. Epilepsia 43 (Suppl. 9); 56-60, 2002
6. Koszewski W: Epilepsy following brain abscess. The evaluation of possible risk factors with emphasis on new concept of epileptic focus formation. Acta Neurochir (Wien) 113; 110-117, 1991
7. Morgan H, Wood MW, Murphey F: Experience with 88 consecutive cases of brain abscesses. J Neurosurg 38; 698-704, 1973

8. Northcroft GB, Wyke BD: Seizures following surgical treatment of intracranial abscesses. J Neurosurg 14; 249-263, 1957
9. Ojemann G, Ojemann J: Cortical language localization in left, dominant hemisphere: An electrical stimulation mapping investigation in 117 patients. J Neurosurg 71; 316-326, 1989
10. Quesney LF: Extratemporal epilepsy: Clinical presentation, pre-operative EEG localization and surgical outcome. Acta Neurol Scand Suppl 140; 81-94, 1992
11. Williamson PD, Boon PA, Thadani VM, Darcey TM, Spencer DD, Spencer SS, Novelly RA, Mattson RH: Parietal lobe epilepsy: diagnostic considerations and results of surgery. Ann Neurol 31; 193-201, 1992
12. Williamson PD, Thadani VM, Darcey TM, Spencer DD, Spencer SS, Mattson RH: Occitipal lobe epilepsy: clinical characteristics, seizures spread patterns and results of surgery. Ann Neurol 31; 3-13, 1992
13. Zumsteg D, Wieser HG: Presurgical evaluation: Current role of invasive EEG. Epilepsia 41 (Suppl. 3) ; 55-60, 2000