

Ictal Intracranial Recording from a ‘Burned-Out Hippocampus’

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Key Words

Mesial temporal epilepsy · Presurgical evaluation · Burned-out hippocampus · Seizure propagation · Electroencephalography · Invasive recording

Abstract

Objective: To present a case involving a discrepancy in the presurgical data of a patient suffering from pharmacoresistant mesial temporal lobe epilepsy. **Clinical Presentation and Intervention:** A 47-year-old, female patient with complex partial seizures since her twenties came to be evaluated in the Epilepsy Surgery Unit. The ictal electroencephalogram suggested a left temporal epileptogenic zone and the magnetic resonance image showed an abnormality in the right mesial temporal lobe. Intracranial monitoring revealed a pacemaker zone in the right hippocampus that discharged fast spreading to the left mesial temporal lobe, a phenomenon known as ‘burned-out hippocampus’. **Conclusion:** The intracranial recording, even though it is an invasive procedure, was necessary for the presurgical evaluation of our patient. This case demonstrates the risks of using surface electroencephalography to determine localization of epileptogenic zones.

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Introduction

Patients suffering from refractory mesial temporal lobe epilepsy are good candidates for surgical treatment [1]. In some cases evidence of unilateral damage in structural studies contrasted with noninvasive ictal electroencephalography (EEG) onset, where depth electrode studies have confirmed that the ictal EEG recording on the surface was misleading [2]. Five patients of this clinical type have been described by Mintzer et al. [3], who referred to this rare phenomenon as the ‘burned-out hippocampus’. We present an intracranial recording from a patient with misleading surface EEG because of a burned-out hippocampus.

Case Report

A 47-year-old right-handed woman had been suffering from complex partial seizures (CPSs) since her twenties. Her typical seizures were heralded by an unpleasant chest sensation and a sense of anxiety. Witnesses would subsequently observe a behavioral arrest and wide-eyed stare, followed by orolimentary automatisms and verbalizations such as ‘fine, fine, fine ...’. No other clinical signs with a localizing value were noted. The seizure

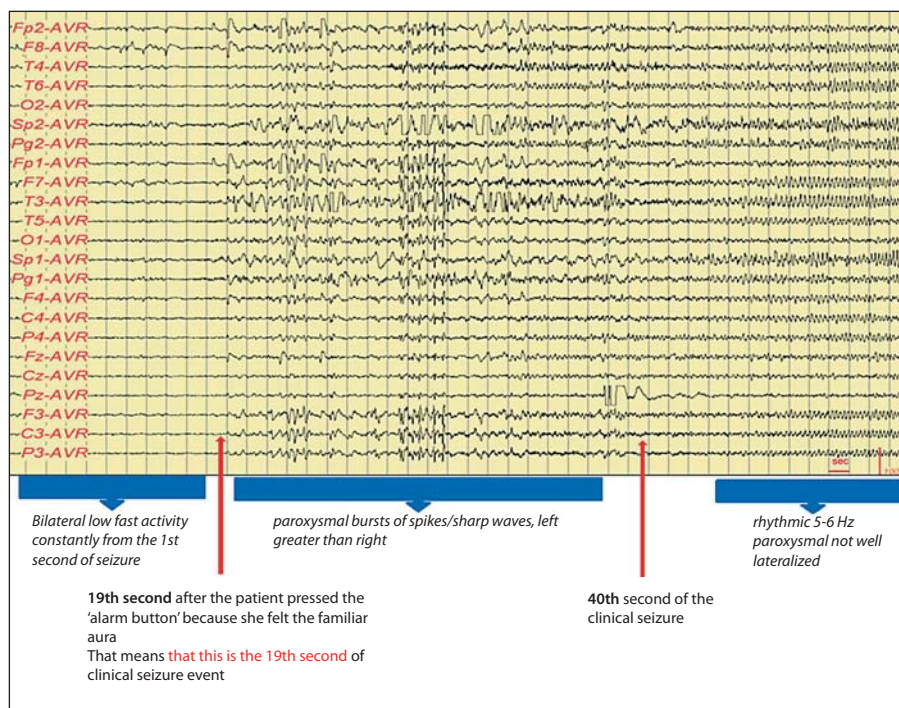


Fig. 1. Ictal scalp EEG recorded during a seizure.

lasted up to 2 min, following by 10–30 min of confusion. Despite the antiepileptic treatment she had 2–5 CPSs monthly, rarely with secondary generalization, and she underwent a presurgical work-up. Interictal routine EEGs revealed in frontotemporal areas bilateral slow waves with a right hemispheric predominance less than 70%.

Neurological clinical examination was normal. Brain magnetic resonance imaging showed abnormal signal on the right mesial temporal lobe. Prolonged video-EEG monitoring with scalp and sphenoidal electrodes captured 3 CPSs. Electrographically, as the patient pressed the alarm button, the entire recording attenuated for a period of 19, 21 and 24 s (for the 1st, 2nd and 3rd seizure, respectively), and a bitemporal low voltage fast activity appeared with a slight predominance on the right. After this period an ictal pattern appeared characterized by high voltage sharp wave discharges, with a higher voltage in the left temporal region (fig. 1).

A Wada test suggested clear left side predominance for memory and speech. Major findings of neuropsychological evaluation suggested difficulties in data processing and especially in online data sequencing, in complex attention and visual-spatial memory defects.

The interictal EEG findings and the neuropsychological tests suggested a right side zone of functional deficit. Accordingly, the preserved speech during the seizure (even elementary) supported right side partial epilepsy [4]. However, the ictal EEG recording did not have unequivocal findings to support the scenario of right mesial temporal lobe epilepsy.

Because of the noncongruent preoperative data, an invasive study was performed in order to define the epileptogenic zone. Subdural grids were implanted and covered the right and left

frontotemporal regions. Two 4-contact depth electrodes were inserted in the mesial temporal lobes. During the recording, 2 CPSs and 1 secondary generalized seizure were captured.

The abnormal EEG activity started with restricted rhythmic spikes at the right hippocampus (fig. 2). At the 16th second, rhythmic spikes appeared in the left hippocampus as well.

The restricted rhythmic discharges in the right hippocampus were sustained for a total duration of 31–38 s and ceased, and at the same time the firing from the left hippocampus spread out to the left hemisphere and generalized (fig. 3). The patient underwent a right anterior temporal lobectomy and amygdalohippocampal removal and for 5 years has remained seizure free. The biopsy revealed a small hamartoma in the right mesial temporal lobe, as well as gliosis of the right hippocampus with normal neocortex.

Discussion

Our patient's ictal EEG was characterized by bitemporal low voltage fast activity with a slight predominance on the right. However, this is not a clear ictal pattern. A typical ictal pattern was obvious after the 20th second and characterized by high voltage sharp wave discharges, with a higher voltage in the left temporal region. This delayed appearance of epileptiform discharges raises great skepticism about their relation to the epileptogenic zone. Based on the above, no certain conclusion could be drawn

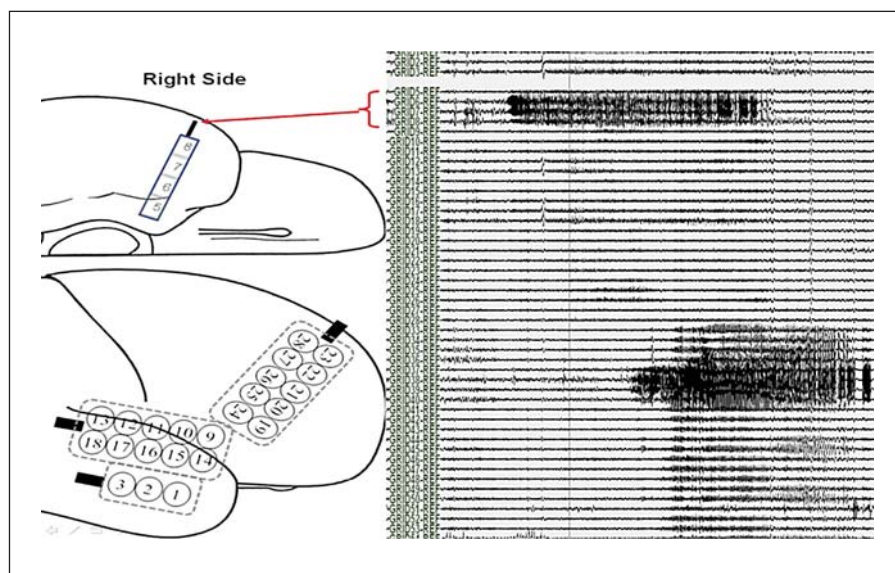


Fig. 2. Seizure which started from the right hippocampus.

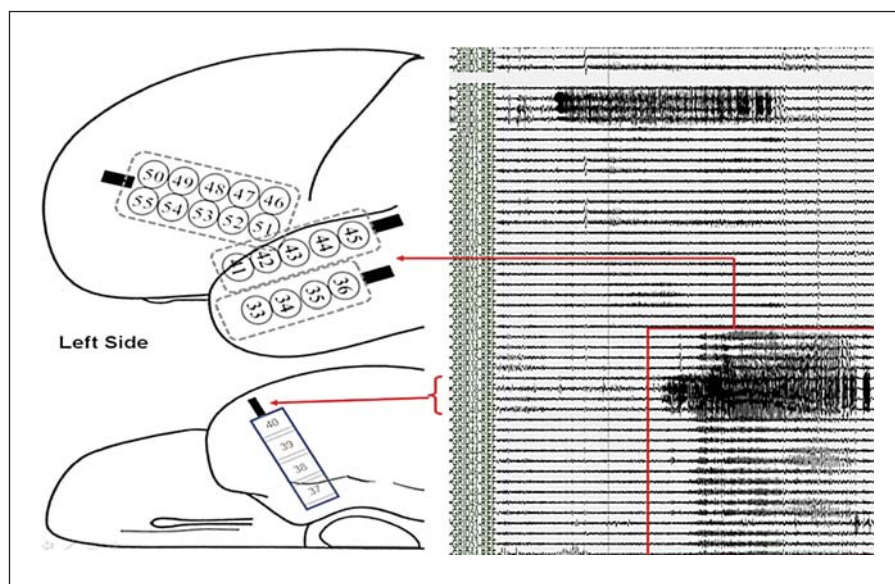


Fig. 3. Seizure spread to the left hippocampus, temporal lobe and hemisphere.

concerning the epileptic zone lateralization in our patient.

The findings from interventional long-term recordings suggested right mesial temporal epilepsy. It was revealed that the lack of obvious epileptiform activity on scalp electrodes at the beginning of the seizure was due to the very restricted abnormal activity of the right hippocampus. Only after 16 s (12, 16, 17 in 3 captured seizures), did rhythmic spikes appear in the left hippocampus, and from there expanded to the temporal neocortex.

The restricted rhythmic discharges on the right hippocampus were sustained for 31–38 s. At the same time

the firing from the left hippocampus spread out to the left hemisphere and generalized. Scalp recording became clearly epileptiform only when the electrical disturbance spread to the entire left temporal lobe.

Mintzer et al. [3] suggest that there is a disruption of the connections between the abnormal hippocampus and the cortex. In such cases, abnormal discharges lack pathways to spread ipsilaterally [3]. Instead, the discharges spread to the contralateral mesial temporal lobe, and there was a further propagation [3, 5].

Based on anatomical studies and intracranial EEG studies, Gloor et al. [5] described the possible types of

mesial temporal seizure spread. Type I was defined by seizures with onsets in the mesial temporal structures (MTS) unilaterally with initial spread to the contralateral MTS. Type II seizures originate from MTS unilaterally; their initial spread involves the ipsilateral temporal cortex and is followed by spread to the contralateral hippocampus before spreading to the contralateral temporal cortex. In type III seizures originate from unilateral MTS and have an initial spread to the contralateral temporal lobe in a diffuse manner. The type IV group of seizures includes all remaining seizures with unilateral onset in MTS and different patterns of transmission [5].

Based on the spreading process, seizures in our case could be classified as type I, because they were character-

ized by the unilateral onset in the MTS followed by spread to the contralateral MTS and only later to the contralateral cortex. This kind of seizure represents less than 10% of 220 mesial temporal lobe seizures recorded by Gloor et al. [5] from 53 patients.

Conclusion

The intracranial recording, even though it is an invasive procedure, was necessary for the presurgical evaluation of our patient. This case demonstrated the risks of using surface EEG to determine the localization of epileptogenic zones.

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