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Case Report

Occipital lobe epilepsy with fear as leading ictal symptom

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ABSTRACT

Ictal fear is a semiological feature which is commonly associated with mesial temporal lobe epilepsy. Here, we describe fear as a leading symptom in cryptogenic occipital lobe epilepsy. In a patient with negative MRI findings, intracranial EEG recordings documented a strict correlation between habitual ictal anxiety attacks and both spontaneous and stimulation-induced epileptic activity in a right occipital epileptogenic area with subsequent spreading to the symptomatogenic zone in the amygdala. Circumscribed occipital topectomy led to seizure freedom. Episodes of non-epileptic fear ceased shortly afterwards. This report provides insight into pathways of propagation of epileptic activity, illustrates different etiologies of pathologic fear and underlines the importance of ictal EEG recordings.

1. Introduction

Panic attacks from anxiety disorders can be difficult to distinguish from ictal fear occurring in focal epilepsy. Even if an epileptic origin is suspected, the focal start of epileptic discharges may be difficult to identify.

Compared to panic attacks, ictal fear is a relatively rare phenomenon. Typically, it is known as an early semiological feature in mesiotemporal epilepsies [3]. The crucial role of the amygdala in the generation of ictal fear is well established [12]. Here, we report a well-documented case in which fear as the leading ictal symptom resulted from an extratemporal seizure onset zone in occipital lobe epilepsy.

2. Case report

A 47-year-old male patient was referred to our epilepsy center due to the new occurrence of panic attacks at the age of 39 years. A panic disorder was diagnosed. Psychopharmacological treatment with SSRI, psychotherapy, and antiepileptic treatment with carbamazepine, lamotrigine and levetiracetam had not been successful. The patient had shown generalized tonic–clonic seizures from the ages of 16 to 28 which had been considered alcohol-related and which had ceased when he became abstinent. Semiologically, three types of episodes with fear could be distinguished: type 1 with mild fear, sometimes occurring several times per day, type 2 with intense fear, accompanied by an epigastric sensation, and type 3 with intense fear, accompanied by an increase in heart rate, palpitations and sweating.

High-resolution MRI revealed no epileptogenic lesion. FDG-PET and ictal SPECT were suggestive of an epileptogenic region in the right anterior temporal lobe. In contrast, during 5 episodes of ictal fear, recorded during scalp video-EEG-monitoring (VEM), there was a posterior initiation of a right temporal anterior seizure pattern (Fig. 1). No interictal epileptic discharges were registered.

Invasive VEM was performed with a right temporal–parietal–occipital subdural grid, temporal–lateral and –basal subdural strips, and a temporo-mesial depth electrode (Fig. 2).

During invasive VEM with withdrawal of antiepileptic treatment, 32 habitual events were recorded: 29 episodes of mild fear (type 1) had no specific electroencephalographic correlate (in particular, no seizure pattern involving the amygdala) and therefore, were classified as non-epileptic. In contrast, three episodes were characterized by intense fear (type 2), steadily increasing over the course of the seizure and accompanied by vegetative symptoms as described above (type 3), progressing twice into an automotor seizure with secondary generalization. Electroencephalographically, repetitive bursts of low amplitude fast activity (lafa) in the right occipital region were registered (Fig. 3A) more than 1 min prior to first clinical symptoms. These lafa bursts finally initiated spike discharges in the amygdalar region (Fig. 3B). Fear was reported when these occipito-amygdalar lafa/spike discharges became repetitive (Fig. 3C). With the spread of these discharges to the hippocampus, the intensity of fear increased, and the patient reported a thoracic localization (Fig. 3D). Discharges progressed to rhythmic theta activity (Fig. 3E),

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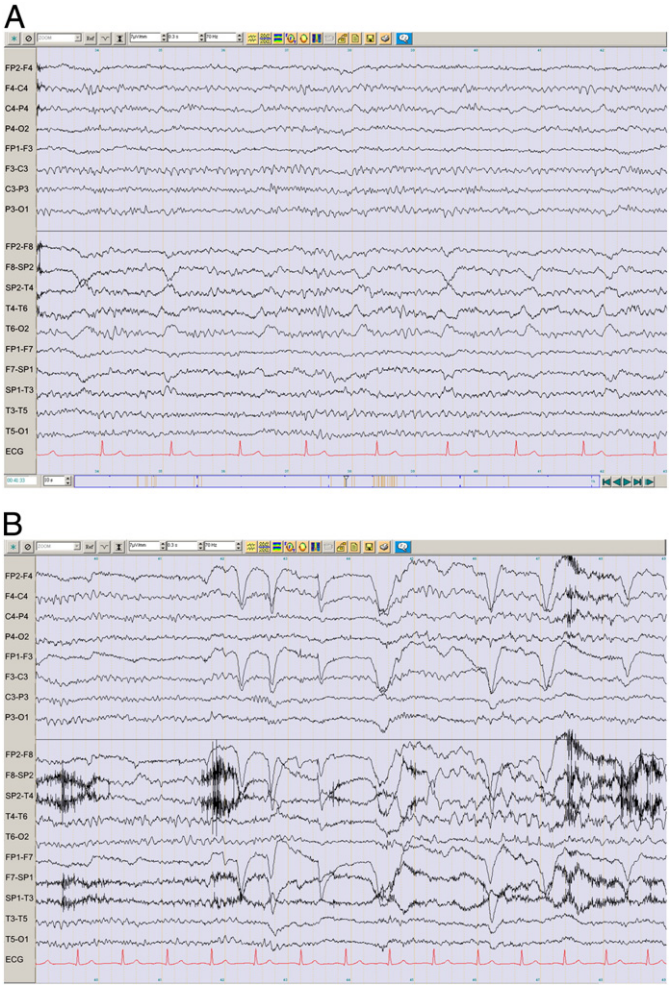

Fig. 1. Scalp-EEG: (A) Pre-ictal phase of a seizure registered during surface video-EEG-monitoring with repetitive temporal posterior occipital sharp-waves, triggering temporal anterior sharp-waves. (B) Late right temporal anterior seizure pattern.

corresponding to oral automatisms with preserved consciousness and ictal speech. Frequency and duration of lafa-bursts varied between seizures (Fig. 3F). There was no epileptic activity in the temporal lobe without occipital initiation.

Habitual fear could also be induced by electrostimulation, at both the amygdalar and occipital electrode contacts.

Surgical treatment by topectomy in the right occipital region (Fig. 4) resulted in cessation of all epileptic seizure types. Histopathology revealed a focal cortical dysplasia [7]. Episodes of non-epileptic mild fear (type 1) initially persisted. Later, treatment with an SSRI and psychotherapy led to complete remission also of these episodes. As of now, the patient has remained free of fear attacks for 38 months.

3. Discussion

Reports of panic attacks as a manifestation of extratemporal epilepsies are rare [1,5,8,11].

To our knowledge, this is the first case, which is demonstrated by intracranial EEG recordings, that ictal fear can be the leading symptom of occipital lobe epilepsy. Habitual fear was triggered by spontaneously arising epileptic activity in the occipital lobe and by electrical stimulation at the same location via the secondary propagation of discharges from the occipital epileptogenic area to the symptomatogenic zone in the amygdala. There was a striking temporal relationship between the evolution of the intracranial seizure pattern and the experienced intensity of fear. An independent epileptogenic zone in the temporal lobe was not verified.

This case illustrates that predominant ictal fear as a semiological feature commonly associated with mesial temporal lobe epilepsy may be misleading with respect to the localization of the epileptogenic zone even if PET and ictal SPECT concordantly point toward temporal abnormalities [10]. Furthermore, it underlines the necessity to take into account an epileptogenic focus distant from the symptomatogenic region.

In the present case, ictal surface EEG hinted toward seizure spread from posterior brain regions into the anterior temporal lobe. This subsequently was confirmed by the invasive EEG recordings and by seizure control resulting from a circumscribed occipital topectomy. Epileptic activity thus followed physiologically relevant pathways [4].

Interestingly, this patient additionally gives an example of the specific coexistence of epileptic fear and non-epileptic panic attacks [2,6,9]. A correct distinction may necessitate elaborate EEG investigations. In the present case, comorbidity was proven by intracranial EEG recordings as well as by successful epilepsy surgery and elimination of the residual non-epileptic panic disorder by psychotherapy and a psychotropic medication without known effect on epileptic seizures.

The mechanisms of a putative interaction between the epileptic and the psychiatric disorder remain speculative. However, the observation that only after epilepsy surgery could the panic disorder finally be treated successfully with behavioral therapy and SSRI suggests a triggering role of epilepsy for the panic disorder.

Conflict of interest statement

None of the authors has any conflict of interest to disclose.

We confirm that we have read the journal's position on issues involving ethical publication and affirm that this report is consistent with those guidelines.

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