Case Report

Intermittent photic stimulation triggering a temporal lobe seizure in a patient with schizencephaly and pachygyria

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Abstract

Photic stimulation is a common trigger for generalized epilepsies but may rarely incite focal seizures. Aside from documented cases of photosensitive occipital lobe epilepsies, few reported instances exist of focal epilepsies being triggered by intermittent photic stimulation. The case of a 12 year-old male with known schizencephaly, pachygyria, and right temporal lobe epilepsy triggered by photic stimulation is reported. To our knowledge, this is only the eighth reported case of photosensitive temporal lobe epilepsy.

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1. Introduction

Photic stimulation has been well documented in the scientific literature as a trigger for genetic generalized epilepsies [1]. It is, however, far less common for patients with focal onset epilepsies to exhibit a photoparoxysmal response (PPR), though there are known cases of photosensitive occipital lobe epilepsies [2,3]. We are reporting the case of a 12-year-old male with known schizencephaly and pachygyria with photosensitive right temporal onset epileptic seizures.

2. Case presentation

A 12-year-old male with previously diagnosed attention deficit hyperactivity disorder and oppositional defiant disorder as well as a known history of schizencephaly and pachygyria on brain imaging (Fig. A) had a first time convulsive seizure. Prior to the initiation of any antiseizure medications, an electroencephalogram (EEG) was performed. As per the neurodiagnostic laboratory routine, intermittent photic stimulation (IPS) was performed at frequencies of 1, 3, 5, 7, 9, 10, 11, 12, 13, 14, 15, 16, 17, 18, 19, 20, 21, 23, 25, 27, and 29 Hz. This was repeated such that each frequency was tested twice. At frequencies of 13, 15, 17, and 19 Hz, the EEG demonstrated spike-and-wave discharges in the right temporal area (maximal at the T4 electrode) beginning approximately 2–3 s after initiation of the photic stimulation (Fig. B). These results were reproducible on a second set of photic stimulation. These discharges would be consistent with the Jeavons and Harding classification for a category 3, type 2 photoparoxysmal response [4]. At one point in the recording, photic stimulation precipitated a 34-second right centrotemporal nonconvulsive seizure with rhythmic spike-and-wave discharges beginning at 4 Hz, then decreasing to 1.5 Hz before a 10–20 s period of right hemisphere background attenuation. This subclinical focal seizure was self-limited. During the electrographic seizure, the patient maintained consciousness and was able to respond appropriately to questions posed by the neurodiagnostics technologist. Epileptiform discharges did not occur during a five-minute trial of hyperventilation or during N1 or N2 sleep. Also discharges were not clearly seen at other times during the wakeful state. Symptoms were not reported and clinical signs were not noted during the electrographic activity described above. Otherwise, the EEG background demonstrated a 10 Hz posterior dominant rhythm while the patient was awake with his eyes closed, normal differentiation between wake and sleep spindles denoting stage N2. Focal slowing of the background rhythm was occasionally seen over the right hemisphere with frequencies of 4–5 Hz seen during the normal wakeful state.

3. Discussion

The majority of seizures triggered by intermittent photic stimulation (IPS) have been attributed to genetic generalized epilepsies...
It is rare for patients with focal seizures to demonstrate a PPR when subjected to IPS with the exception of several reported cases of photosensitive occipital lobe epilepsies [2,3]. This has led some researchers to hypothesize that photosensitive focal seizures are exclusively occipital in origin [5,6]. However, this fails to explain reported cases of photosensitive epilepsies originating in the temporal lobe. Some authors have suggested an anatomical pathway connecting the occipital and temporal lobes to explain photosensitive temporal lobe seizures [7,8]. However, there are documented cases of photosensitive focal epilepsies without associated occipital lobe activation that would argue against this explanation. A possible explanation for temporal photosensitivity is that anatomic disruption of the optic radiations could produce a hyperexcitable neural network triggered with certain specific repetitive visual stimuli.

In 1996, Benbadis et al. reported the first known case of a photosensitive temporal lobe epilepsy without associated occipital visual cortex activity [9]. These findings were supported by EEG

**Table 1**

Comparison of clinical features and electrographic onset of previously reported focal on set photoparoxysmal response.

| Electrographic onset | Clinical features | Reference |
|----------------------|-------------------|-----------|
| Independent bitemporal | Aura of “funny feeling” followed by activity arrest, loss of awareness, and manual and orobucal automatisms lasting approximately 1 minute followed by postictal confusion and amnesia of the event | Benbadis et. al. (1996) |
| Right frontal | Behavioral arrest followed by oroalimentary automatisms occasionally followed by focal to bilateral tonic-clonic seizure | Seddigh et al. (1998) |
| Right temporal | “Indescribable terror” and a fearful expression followed by repetitive ictal speech with occasional focal to bilateral tonic-clonic seizure | Seddigh et al. (1998) |
| Right temporal | Anxious feeling, perceived environmental sounds, the patient’s face showing fright, and chewing movements followed by loss of contact, the patient repeatedly saying “help me, help me, help me”, and automatic rubbing of right hand fingers with dystonic posture and flexion of the left wrist and fingers | Isnard et al. (1998) |
| Right anterior temporal | Coughing, tachycardia, and nausea followed by vomiting with associated altered awareness, disorientation, and inability to carry out simple instructions. Imaging demonstrated foreign tissue lesion or malformation in the mesial temporal area | Thomas et al. (1999) |
| Right temporal | Ascending retrosternal sensation often followed by loss of contact, manual and oroalimentary automatisms, and dystonic posture of the left hand lasting 1–2 min followed by postictal confusion and amnesia of the event. Hippocampal and mesial temporal atrophy on imaging | Fiore et al. (2003) |
| Left temporal | Motionless staring lasting 20 s followed by forced turning of the head to the right and focal to bilateral tonic-clonic seizure lasting about 3 min | Lee et al. (2014) |

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**Fig. A.** T1 weighted magnetic resonance image demonstrating an “open lip” schizencephaly and pachygyria in the right temporo-parietal region.

**Fig. B.** Electroencephalogram in a bipolar montage demonstrating serial sharp waves in the right central temporal region (electrode positions T4/T6) with sustained photic stimulation at a frequency of 15 Hz.
evidence as well as subsequent seizure freedom following right temporal lobectomy. Since then, six additional cases of photosensitive temporal lobe epilepsy have been reported \[10–14\]. Of the seven cases reported to date, two have demonstrated associated structural abnormality on neuroimaging studies (Table 1). One case had right hippocampal and mesiotemporal atrophy and the other had either a foreign tissue lesion or malformation of the mesial temporal area. None of the previously reported studies showed evidence of associated schizencephaly or pachygyria. To our knowledge, this is the first case reported in the scientific literature of photosensitive temporal lobe epilepsy with associated schizencephaly.

4. Conclusion

Though not previously reported, this case demonstrates that congenital malformations (schizencephaly and pachygyria) can be associated with a focal PPR on EEG. Furthermore, in addition to occipital abnormalities, structural abnormalities found in the temporal lobe can also be associated with a PPR. Further description of cases involving structural anatomic lesions causing PPR could be worthwhile in understanding focal onset epilepsy syndromes.

Ethical statement

- The authors affirm that we both contributed in a substantial manner to the conception, design, analysis, drafting, and writing of this scholarly paper. Both authors have given approval to the draft in its current form.
- This paper is not currently under consideration for publication at another journal.
- No experimentation of any kind was performed on the subject of the paper; this case report is merely descriptive and all personally identifying information has been removed from the submission.

Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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