Seizure: European Journal of Epilepsy 67 (2019) 27–29

Contents lists available at ScienceDirect

Seizure: European Journal of Epilepsy

journal homepage: www.elsevier.com/locate/seizure

Clinical letter

Akinetopsia with achromatopsia due to focal epilepsy

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1. Introduction

Akinetopsia is a rare visual symptom of the central nervous system [1]. There are two types of akinetopsia: invisibility of moving objects, and motionless vision resembling freeze frames in a motion picture (cinematographic vision). Since there are only a few reports of akinetopsia, the corresponding region for motion perception is controversial. We herein describe a patient with ictal akinetopsia with achromatopsia whose symptoms and presentation offer insight in considering the issue.

2. Case presentation

A 68-year-old woman complained that after she experienced an uncomfortable feeling what she was looking at froze and lost its color like a monochromic photograph. She did not experience déjà vu, jamais vu, epigastric rising sensation, or olfactory sensation. During the symptom, she could hear surrounding sounds normally. After a few seconds, her vision returned to its normal condition. Her visual symptom appeared on various occasions. Her symptom had appeared one year before and recently occurred every day. She had diabetes mellitus without diabetic complications and received oral hypoglycemic agents. She did not experience any symptoms of hypoglycemia during the symptom. Neurological examination was normal. Routine blood tests including thyroid and adrenal functions were also within normal limits. Magnetic resonance imaging of her brain was normal, and no lesion was enhanced by Gadolinium. Thirty-minute electroencephalogram (EEG) was performed twice before treatment. The first EEG (Fig. 1) revealed the sharp waves four times on the right hemisphere. The phase reversal was found between F8 and T4 electrodes (Fig. 1 arrows). Two minutes after hyperventilation loading, burst of sharp waves appeared on the right frontotemporal regions. During the recording of EEG, she experienced the usual uncomfortable feeling. Although seizure spread was not observed in the second EEG, sharp waves were recorded seven times. Interictal single photon emission computed tomography (SPECT) revealed hyperperfusion in the right frontotemporal region and hypoperfusion in the bilateral occipital regions (Fig. 2, upper). Visual perception test for agnosia (edited by the Japan Society for Higher Brain Dysfunction), also performed interictally, did not show hemispatial neglect, prosopagnosia, or topographic disorientation. Wechsler Adult Intelligence Scale (3rd edition) showed VIQ 116, PIQ 108, and FIQ 114, suggesting normal intelligence. She was treated with 200 mg/day carbamazepine. EEG recorded one month after treatment showed no epileptic discharge. Her visual symptom was suppressed completely. SPECT was also improved 18 months after treatment (Fig. 2, lower).

3. Discussion

The visual symptom of our patient is considered to be akinetopsia with achromatopsia caused by epilepsy. Our patient presented cinematographic vision among the two types of akinetopsia. The focus of her epilepsy was considered to be in the right frontotemporal region based on EEG. SPECT was performed interictally. However, the facts that sharp waves were easily observed on EEG and the patient experienced visual symptom every day suggest that the focus was activated even during the interictal phase. Therefore, the hyperperfusion of the right frontotemporal region was probably due to the co-localizing epileptic activity. Interestingly, treatment with carbamazepine ameliorated not only the hyperperfusion of the epileptic focus but also the hypoperfusion of the bilateral occipital regions. Thus, both SPECT findings were most likely caused by the epileptic activity. The possible mechanism for this is remote inhibition, which is defined as the inhibition of neurons that are remote from the epileptic focus and connected with it through cortico-cortical or polysynaptic pathways.

The region responsible for akinetopsia has been thought to be in the area MT/V5. Although the original case presented by Zihl had bilateral lesions, unilateral lesion might be enough to cause akinetopsia [1]. Recently, right ventral visual cortex integrity has been reported to be important in motion perception, as well as the dorsal pathway including the area MT/V5 [2]. It is not determined how two types of akinetopsia are associated with the dorsal or ventral pathway, or the laterality of the lesion.

One difference between our case and the previously reported cases with akinetopsia was the complicating achromatopsia. Our patient mentioned that all the visual field lost its color and became like a monochromatic photograph. As to color perception, there have been

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https://doi.org/10.1016/j.seizure.2019.03.004

Received 10 January 2019; Received in revised form 3 March 2019; Accepted 5 March 2019

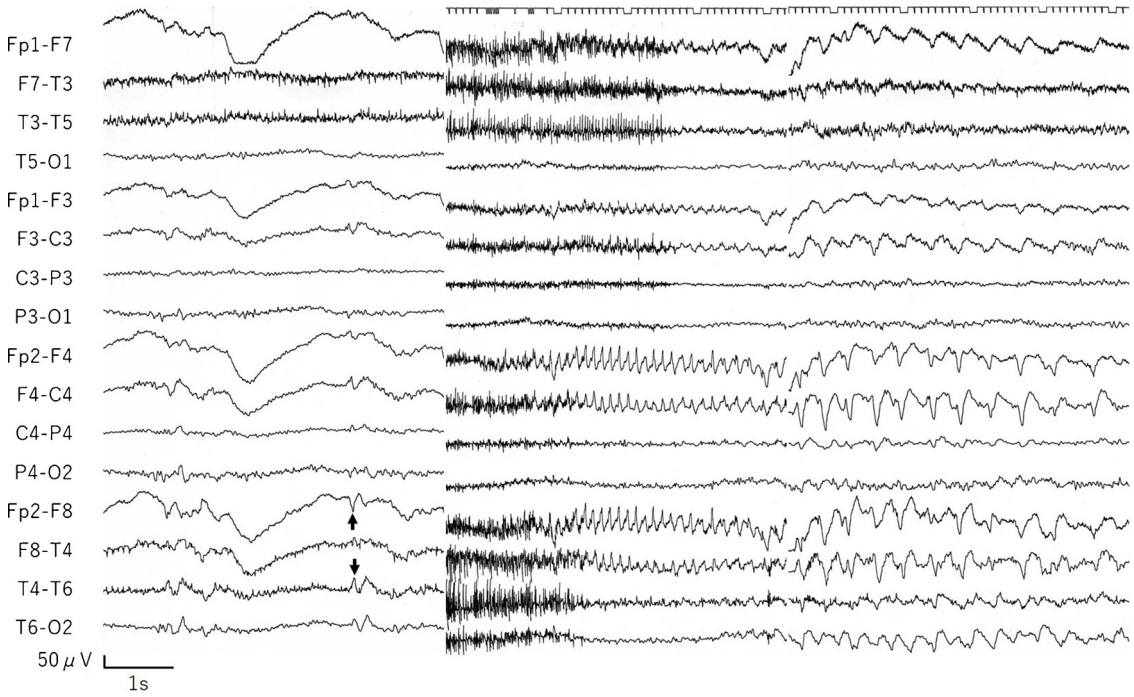

Fig. 1. Electroencephalogram. Sharp waves are observed on the right hemisphere (left). The phase reversal is found between F8 and T4 electrodes (arrows). Epileptic activity began on the right frontotemporal region (center) and disappeared (right).

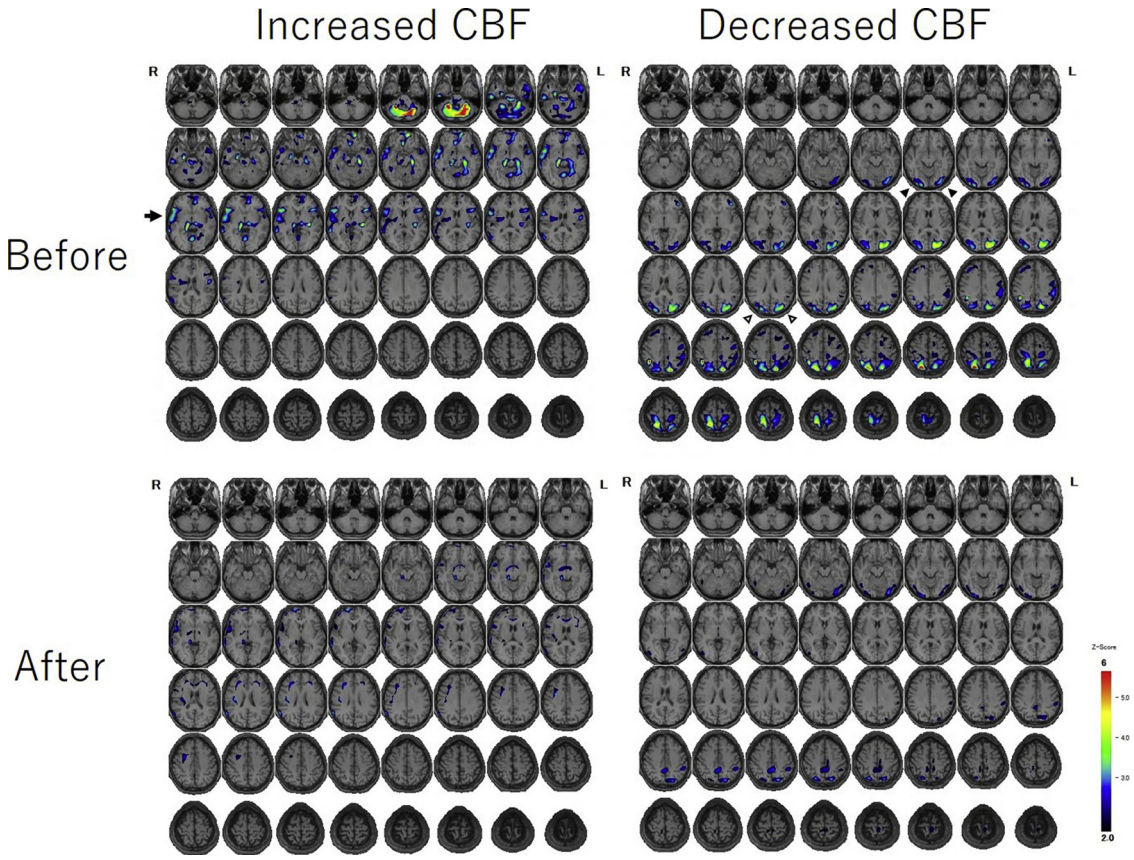

Fig. 2. Interictal single photon emission computed tomography (SPECT) using ^{99m}Tc-ethyl cysteinate dimer before treatment. The data were analyzed with the Easy Z-score Imaging System. Areas with increased (Z-score > 2) cerebral blood flow (CBF) (left) and those with decreased CBF (right) are colored. Before treatment (upper), hyperperfusion around the right superior temporal gyrus (arrow) and hypoperfusion of the bilateral occipital regions are revealed. Hypoperfused region involves the area MT/V5 (open triangles) and V4v (closed triangles). Approximately 18 months after treatment (lower), both the hyper- and hypoperfusion seen before treatment are improved.

many discussions about the existence of a color center. Bartolomeo et al. introduced interesting cases of achromatopsia. One was in a hemiachromatopsic patient and involved the inferior part of the left occipital lobe. Another patient did not show achromatopsia after a first stroke involving the left occipitotemporal hematoma, but developed full-field achromatopsia after a second stroke affecting the right occipitotemporal region [3]. Although bilateral ventral occipitotemporal lobes containing the lingual and fusiform gyrus are important in color perception, there might be right-hemisphere dominance.

The hypoperfused regions observed in our patient before treatment involved bilateral classical centers of motion (MT/V5) and color (ventral part of V4; V4v) perception. Epileptic impulses could be retrogradely conveyed from the right frontotemporal region to the ipsilateral MT/V5 and V4v via the right ventral visual pathway and suppress their functions. They could also suppress the contralateral MT/V5 and V4v via callosal connection and result in ictal full-field akinetopsia with achromatopsia.

A similar epilepsy patient showing ictal cinematographic vision was reported to have a focus in the right mesial temporal lobe apart from area MT/V5 [4]. Unlike in that patient, we could derive ictal EEG and SPECT before and after treatment. During the seizure, ictal discharge did not seem to spread to the contralateral hemisphere in our patient. Although both cases seem to show the full-field akinetopsia from the focal seizure whose foci were in the right temporal lobe, akinetopsia, at least in our case, might have been due to the inhibition of the bilateral MT/V5 area.

4. Conclusion

We reported a case of focal epilepsy showing full-field akinetopsia with achromatopsia. The focus of the epilepsy was the right frontotemporal region based on the results of EEG and SPECT. The remote inhibition of the bilateral MT/V5 and V4v via the right ventral visual pathway and callosal connection could be the cause of this rare symptom.

Conflict of interest

None.

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