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CASE REPORT

Ictal visual hallucinations and post-ictal hemianopia with anosognosia

JOSEF SPATT & BRUNO MAMOLI

Ludwig Boltzmann Institut für Epilepsie und Neuromuskuläre Erkrankungen, II. Neurolog. Abt., NKH Rosenhügel, Riedelgasse 5, 1130 Vienna, Austria

Correspondence to: Dr Josef Spatt, II. Neurolog. Abteilung, Neurologisches Krankenhaus, Rosenhügel, Riedelgasse 5, 1130 Vienna, Austria. *E-mail*: josef.spatt@chello.at

We report the case history of an adult who developed seizures with nearly pure visual symptoms due to an occipital vascular lesion. The seizures were characterized by elementary visual hallucinations in the right visual field. Interictally, a dense homonymous hemianopia was demonstrated in the clinical examination and by using perimetry, but was not recognized by the patient himself. The seizures vanished and the visual fields normalized completely after initiation of anticonvulsive treatment.
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Key words: epilepsy; occipital lobe; epilepsia partialis continua; hemianopia; anosognosia; hallucination.

INTRODUCTION

Positive visual symptoms are frequent in auras of seizures arising from the occipital lobe[1–3], but in adults they rarely occur as an isolated seizure manifestation. Ictal[4,5] and post-ictal[6–9] negative visual symptoms such as amaurosis or hemianopia have been reported[10], but they are often too short-lived to be objectively demonstrated. We are not aware of a case where visual fields were formally assessed with perimetry in epileptic hemianopia.

CASE REPORT

RS, an 83-year-old left-handed male, was admitted with recurrent attacks of positive scotoma in the right visual field. The symptoms had started the previous day and were described by the patient as multicoloured spots beginning in the lower right quadrant and later spreading across the whole right visual field. A single attack lasted for about 1 minute. The symptoms were described as very disturbing and were accompanied by substantial feelings of fear. No other symptoms were reported by the patient. In particular, he did not report any visual impairment between the attacks.

There was no history of migraine but he had suffered from an ischaemic cerebrovascular accident 1 year before. This stroke had presented with a mild right-sided hemiparesis and hemihypesthesia, which had resolved within 2 days. No visual deficits had been noted in the charts at that time.

At the current presentation, neurological examination revealed a right-sided tendon reflex accentuation as had already been described 1 year before and a new dense right-sided homonymous hemianopia. Otherwise, physical examination was unremarkable. During examination, three attacks as described before were observed. These were not accompanied by loss of consciousness, but vigilance was slightly reduced as evidenced by an impaired ability to count backwards in steps of seven. Furthermore, the patient was in a fearful state during the attacks that was felt to be more intense than expected from a psychological reaction to the visual symptoms. The fits were always accompanied by discrete clonic movements of both eyes. All observed seizures showed the same course.

Routine blood examinations were normal. Cranial CT, performed 1 day after onset of the symptoms, showed small bilateral cystic lesions in the basal ganglia and a slight frontal accentuated atrophy, alterations that had already been described 1 year before.

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During the following days the frequency of the events increased to eventually more than one seizure every 10 minutes. During administration of an EEG, two seizures were documented. They were accompanied by rhythmic slow activity (3–4 Hz) with interspersed small spikes in the left occipital and posterior temporal region. Between the seizures the EEG was normal. A perimetry was performed and confirmed the dense right-sided homonymous hemianopia.

An intravenous bolus of 10 mg diazepam was given during one such seizure. The symptoms vanished immediately and did not reappear during the next hours. Antiepileptic long-term treatment with carbamazepine was initiated and when a dose of 750 mg per day had been reached, no further seizures were observed or reported by the patient and the EEG became normal.

An MRI examination, performed 2 weeks after onset, revealed a 3 cm large gadolinium gyral enhancement at the medial surface of the left occipital lobe, which was interpreted as being of vascular origin. A single photon emission computed tomography (SPECT) using 99mTc HMPAO as tracer was performed during the period of frequent seizures and showed left occipitotemporal hyperperfusion.

In a second perimetry, administered 9 days after the first one, we found totally normalized visual fields.

DISCUSSION

We describe a case with frequent single partial seizures caused by a new vascular lesion in the left medial occipital lobe. The positive visual symptoms of the patient consisting of multicoloured dots are typical for epileptic visual hallucinations and are different from the fortification spectra described by migraine patients[7, 11].

The visual symptoms of the patient were accompanied by myoclonic jerks of the eyes and these have been reported as symptoms of occipital lobe seizures[1].

During the period of very frequent seizures, interictally a dense homonymous hemianopia was demonstrated. While it was not clear at the beginning whether this was caused by the underlying lesion or by epileptic activity, the complete and prompt resolution after termination of the seizures points to an epileptic origin of the symptom. We therefore regarded it as a Todd phenomenon. The hyperperfusion of the left occipital area demonstrated by HMPAO-SPECT also speaks in favour of a functional genesis of the visual field deficit. Similarly, bioccipital hyperperfusion was demonstrated in a patient with complete post-ictal blindness[6]. The role of ictal HMPAO-SPECT in the localization of occipital lobe seizures has been emphasized by Duncan and colleagues[12].

The patient showed no awareness of his hemianopia. Though anosognosia for visual field deficits is not a rare condition[13], in this case it was especially striking, because the patient was aware of the positive ictal symptoms. Hence, he noticed elementary visual hallucinations in the right hemifield, but he did not notice his interictal blindness in the same part of the visual field. Epileptic anosognosia—though for a hemiparesis—has been reported before[14].

The only symptom of this patient that cannot be attributed to the occipital lobe was the fearful state by which the other symptoms were accompanied. We believe that this was more intense than could be explainable as a psychological reaction to the recurring seizures, and so we interpreted it as a true epileptic symptom. A discrete spread to limbic structures including the amygdala would be the most likely explanation. The epileptogenic activity in the EEG affecting also posterior temporal leads supports this hypothesis.

One remarkable aspect of this case is that there was no overwhelming spread of epileptic activity to other brain regions. Seizures with occipital lobe origin tend to spread rapidly to contralateral occipital structures leading to complete ictal amaurosis[10]. Furthermore, there is often a spread to ipsilateral and subsequently to contralateral temporal structures leading to complex partial seizures that are very similar in their symptoms to seizures originating in the temporal lobes[1, 3]. We have no conclusive explanation for this particular behaviour, but aetiology might play a role. Three out of five patients reported by Barry and colleagues[10] with isolated occipital symptoms also had a vascular aetiology, while most documented cases of lesional occipital lobe epilepsy in adults are caused by neoplastic alterations or trauma[1, 3].

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