Abstract

Although Charles Bonnet syndrome (CBS) appears to be more frequent than historically reported, there are very few reports about possible pharmacological treatments (1). We report the case of a CBS disappearing completely with successive monotherapies of carbamazepine and valproic acid but worsening with levetiracetam.

Key words: Hallucinations; visual loss; anticonvulsant drugs; Charles Bonnet Syndrome; carbamazepine; valproic acid, levetiracetam.

Case report

A 85-year-old woman with a history of hypertension, coronary artery disease, multi-infarct syndrome and progressive bilateral peripheral vision loss due to cataract and glaucoma was consulted for complex visual hallucinations which had appeared brutally a couple of days before and which only disappeared during sleep to reappear the next morning. She described the presence of monstrous figures in the left visual hemifield that came closer and stared at her. The patient had full insight into the hallucinatory nature of the images. Neurological examination revealed a known slight left hemiparesis with a hypoesthesia of the left side of the body and a left homonymous hemianopsia. Mini Mental State score was 30/30. EEG during the hallucinations was normal. CT imaging revealed a multi-infarct syndrome and a cortical atrophy that were slightly more important in the right hemisphere. A treatment with carbamazepine 100 mg bid made the hallucinations completely disappear. One month later, the patient complained of a worsening of her chest pain since the start of the treatment and carbamazepine was progressively discontinued and replaced by levetiracetam 250 mg bid. Hallucinations reappeared and were more vivid than ever, until a treatment with valproic acid (150 mg bid) made them disappear again.

Discussion

Often, CBS or the presence of complex visual hallucinations with preserved insight in people suffering from severe bilateral vision loss does not need pharmacological treatment, for several reasons. First, since insight is preserved, sympathetic explanation that the hallucinations are a release phenomenon in a brain that has been deprived from visual stimuli due to the vision loss seems to comfort a vast majority of patients (1). Second, although CBS patients often have hallucinations on a daily basis, they last rather seconds or minutes than hours (2, 3). Finally, they can disappear when the vision becomes more impaired or on the contrary improves (e.g. cataract surgery) (4, 5).

For our patient however, medical treatment was justified by the fact that her hallucinations were almost permanent and because she found them most distressing despite her excellent insight in the nature of the images. The reappearance of her hallucinations after discontinuing the treatment with carbamazepine is an argument in favor of the latter’s efficiency, although the new treatment with levetiracetam might have triggered new visual hallucinations (6). The absence of seizures on EEG during the hallucinations, the low doses at which carbamazepine and valproic acid where effective and the inefficacy of levetiracetam all suggest that carbamazepine and valproic acid might be efficient by other mechanisms than an antiepileptic one. Randomized controlled studies versus placebo are still needed to prove their efficacy in CBS.
REFERENCES


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