Charles Bonnet syndrome versus occipital epilepsy, a diagnostic challenge

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Summary. Charles Bonnet Syndrome (CBS) is a disorder with visual deficit and complex recurrent visual hallucination in conscious patients, described for the first time by Charles Bonnet. It has been found in association with variable pathologic conditions of the eyes, central visual pathways and occipital lobe. Occipital lobe lesion is an important cause of visual field deficit associated with elementary simple hallucinations, whereas complex hallucinations are related to occipitotemporal and occipitoparietal visual association neocortex damage. (www.actabiomedica.it)

Key words: Charles Bonnet Syndrome (CBS), epilepsy, visual complex hallucinations

Introduction

Charles Bonnet Syndrome (CBS) is a disorder with visual deficit and complex recurrent visual hallucination in conscious patients, described for the first time by Charles Bonnet. It has been found in association with variable pathologic conditions of the eyes, central visual pathways and occipital lobe.Occipital lobe lesion is an important cause of visual field deficit associated with elementary simple hallucinations, whereas complex hallucinations are related to occipitotemporal and occipitoparietal visual association neocortex damage.

Case report

Woman, 79 years old, came to our observation for acute visual loss in recent bi occipital ischemic lesion. She has been transferred to our clinic for a gradual rehabilitation of the lower limbs, truck verticalization and eventually gait control.

Her past medical history was positive for ischemic cardiopathy (three aortic coronary by-pass), atrial fibrillation, diabetes, hypertension and reactive depression. She further denied auditory or other sensory hallucinations as well as headaches, fever, or trauma. There was no history of drug or alcohol abuse. She denied any psychiatric history.

In our clinic she experienced different type of visual complex hallucinations (children that plays around her, girls playing with rope, buildings with big windows). She was able to describe them and sometimes she manifested some psychomotor agitation. She describes her symptom as present for the most of time. While experiencing these hallucinations, the patient had no change in mental status and maintained full insight and awareness.

On physical examination she appeared well and in no acute distress. She was oriented to person, place and time. Her vital signs were: blood pressure 130/80 mmHg, pulse rate 80 beats/minute and regular, respirations 20 breaths/minute, temperature of 36.8°C and oxygen saturation of 96% on room air. There were no complaints of any hallucinations at the time of the exam. Neurologic examination showed no focal findings but only Babinsky bilaterally. A mental status examination did not reveal mood disturbance or cognitive deficits. Laboratory studies demonstrated a serum sodium level of 140 mEq/L and a serum chloride of 92 mEq/L. Glucose and renal function were normal. White blood cell count and urinalysis were normal. Her MRI shows a right paramedian occipital ischemia and parieto-occipital posterior left one; Eye examination manifested reduction in visual acuity (1/10) with associated cataract and hypertension retinopathy, intracranic angiographic TC excluded malformation of the intracranial circuit. The mental status examination revealed preserved orientation and consciousness. An antipsychotic therapy (Olanzapine 2,5 twice) was began without benefit. An EEG was made. It shows a slow basal activity with left (O1) occipital spikes and sharp waves. During these period the patient did not manifested loss of cousness. We decided to administrate Levetiracetam (Keppra) 500 mg twice with completed resolution of the complex hallucinations. Two weeks later she was able to deambulate with the support of physicians. She did not refer hallucinations for 60 days, time she remain to our clinic.

Discussion

Our patient experienced complex visual hallucinations with acute stroke involving the occipital lobe. She had typical CBS according to the diagnostic criteria of Gold and Rabins. The main characteristics of hallucinations were complex visual hallucinations of geometrical figures and known faces, bizarre human movements and gestures, which occurred with the eyes open and do not appeared to be just illusions triggered by low lighting levels. Following impairment or loss of vision, hallucinations developed after a period of 24/74 hours, persisted without change of characteristics. Pathophysiology of CBS is generally interpreted either as a release phenomenon (deafferentation) or it may be caused by sensory deprivation and "phantom vision," or an abnormal focus activating neuronal network.

Its frequency in patients with structural damage such as infarction or vascular malformation has suggested that isolated lesions to the occipital lobe can lead to complex visual and somatosensory hallucinations. Visual hallucinations in our patient may be developed by interruption of dorsal occipitoparietal processing system or ventral occipitotemporal processing stream that are related with perception of spatial functions and pattern discrimination and visual identification of objects. It is possible that clinicians may interpret visual hallucinations as part of the remaining visual aura in patients with occipital lobe epilepsy (OLE). However, a recent report found that complex hallucinations were never seen in patients with occipital lobe seizures

The characteristics of our patient hallucinations were identical to those of CBS and they have persisted for more than 74 h in concomitance with an EEG O1 epileptic activity. All, physicians symptoms and EEG activity, get to normalization with the antiepileptic therapy.

Conclusion

We demonstrate the persistence of complex visual hallucinations in occipiatl epileptic activity post ischaemic stroke even we know that complex hallucinations were never seen in occipital lobe epilepsy. This conditions may sometimes mimic a Charles Bonnet syndrome and it does not improve with antipsychotic therapy. Any possibility of misdiagnosis can be prevented through a detailed medical history of the nature of the visual hallucinations and through video-EEG monitoring.

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