

Atypical Charles Bonnet syndrome - A case report

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Abstract

Charles Bonnet syndrome is the cluster of complex visual hallucinations in a cognitively normal person with partial or severe blindness. It is not an uncommon disorder and can be associated with any type of vision loss. Initially documented by Charles Bonnet in his grandfather who had visual impairment from cataracts in both eyes. Charles Bonnet Syndrome (CBS) predominantly affects people with visual impairments due to old age, diabetes or other damage to the eyes or optic pathways. It may not present with all typical symptoms and intact insight. Here, a case of atypical CBS is reported where antipsychotics were effective and patient improved after one month of medications. This case highlights the importance that the CBS can occur in visual loss of any etiology.

Keywords: Charles Bonnet Syndrome, Visual hallucination, Sensory Deprivation.

Introduction

Deliberate reduction or removal of stimuli from one or more senses leads to sensory deprivation. Role of sensory stimulation in the etiology of Hallucination is often noted. Patients who became visually impaired often develop complex hallucinations with preserved cognitive status, called as Charles Bonnet syndrome. The following criterion is been recommended for its diagnosis^[1]:

1. The presence of formed complex persistent or repetitive stereotyped visual Hallucinations.
2. Full or partial retention of insight into the unreal nature of hallucinations.
3. Absence of hallucinations in other sensory modalities.
4. Absence of Primary or Secondary delusions.

Here we have an atypical case of CBS of a 70 year old male presented with visual loss followed by visual hallucinations but without insight.

Case Report

A 70 year old male was brought to the Psychiatry OPD by his son complaining of patient talking to oneself, making gestures and folding his hands in front of none since last 2 months. He was afraid that people will come

and take him by attaching wheel to his bed. He would repeatedly ask his son that some people came asking for money though was unable to recognize them. He used to serve food while dining to those people whom he could see. At times he would complain and scold his children for not protecting him from the insects, snakes and other Animals, which only he could see on walls and floor.

He had developed whitish patch over right eye and was unable to see with that eye for more than 20 years. Patient was operated for cataract of left eye, 12 yrs back. Gradually he lost his vision in this eye too and is blind in both eyes for last 2 years.

There is a history of undergoing surgery for fracture left femur 10 years back, and for benign prostatic hyperplasia and hydrocoele 1 yr back.

On physical examination he had a blood pressure of 160/90 mmHg. On ophthalmological examination, total leucomatous corneal opacity was noted in right eye and increased intra-ocular pressure with aphakia with optic atrophy in left eye. Total loss of vision without even perception of light could be noted in both eyes with complete denial of his blindness. No other neurological or executive function deficits were

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recorded.

Hemoglobin and other blood parameters were within normal limits. ECG and CT scan of the brain showed no significant abnormalities.

On mental status examination, patient was conscious and co-operative with good orientation. He had complex visual hallucination and occasional auditory hallucination with grade 1 insight. No other significant thought or cognitive abnormalities could be noted.

His symptoms were rated on following scales:

- Brief Psychiatry Rating Scale (BPRS)^[2] score of 36
- Hindi Mental Status Examination (HMSE)^[3] score of 27
- Hamilton Depression Rating Scale (HAM-D)^[4] score of 6

He was treated with olanzapine 2.5mg/day and gradually increased upto 10mg/day. He showed significant improvement in visual hallucinations and maintained so after a month of discharge.

Discussion

The exact etiology of CBS is unknown. Visual hallucination in CBS is postulated to be a secretory phenomenon. Disappearance of the cortical inhibition leads to activation of the complex visual cortex. It was found that hallucination in CBS correlates^[5] with cerebral activity in the ventral extra striate visual cortex. Our patient had developed visual hallucination following visual loss. The absence of insight over his visual loss and hallucination was not fitting into the criterion set for CBS. This could be a result of denial of neurological deficit as seen in parietal or occipital lobe lesions than as in functional hallucinations.

As presence of co-morbidities cannot rule out the pathological activation causing visual hallucinations, atypical CBS^[6] has been proposed, when certain of the criterion deviates: 1) diminished level or absence of insight towards the visual hallucinations; 2) presence of a mild cognitive decline; 3) presence of an atypical psychological reaction towards the visual hallucinations as in the case of a severe and prolonged stressful reaction; 4) presence of other hallucinatory modalities; 5) presence of a positive personal psychiatric history or a concomitant psychiatric disorder.

Limitations

We had advised to get MRI Brain to get more subtle details but we could not get as the patient was not affordable.

Conclusion

Each patient suffering from CBS should be initially evaluated psychiatrically and neurologically in order to confirm or to eliminate the presence of the most common causes of visual hallucinations. In the presence of a lowered visual acuity and a conserved cognitive functioning, the atypical CBS is diagnosed after eliminating more common disorders.

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