

Case Report

Charles Bonnet Syndrome: Visual Hallucination in a 75 Year Blind Patient; Case Report and Review of Literature

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Article History

Submitted: 01/04/2023; Accepted: 07/05/2023; Published: 17/06/2023

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ABSTRACT

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Formed visual hallucination in a visually impaired person that is psychologically healthy is defined as Charles Bonnet Syndrome (CBS). Patients with this condition rarely present for fear of being labeled Schizophrenics. This paper aimed to highlight this rare presentation of blindness at the BarauDikko Teaching Hospital, Kaduna. The case is a 75-year-old became blind from glaucoma, cataract and complications of surgery. One year after absolute blindness, stated seeing images consisting of flashes of light and human shapes in gardens containing huts. Though neuropsychiatrist confirmed what he thought, that images were not real, but presented wanting to know whether surgery could restore his vision because of his experiences. Ophthalmologists must watch out for this rare presentation of blindness which could be distressing in an already hopeless situation.

Keywords: Visual hallucination, Charles Bonnet Syndrome, visual impairment, cataract, glaucoma, visual cortex.

INTRODUCTION

Formed visual hallucination in a visually impaired person that is psychologically healthy is defined as Charles Bonnet Syndrome (CBS).¹It was first described by Charles Bonnet a Swiss natural philosopher in 1760 on his grandfather who saw images visiting him through his cataractous eye. Low awareness by physicians and reluctance of disclosure of the condition by patients for fear of being labeled schizophrenics has made it look rare¹.

CASE DESCRIPTION

A 75-year male presented to our clinic with complaints of blindness but seeing unusual images he

knew was not real. The images consisted of human shapes in the garden with huts, occasionally interrupted by flashes of light; the images were initially prolonged and frequent but later became brief. At the time of the presentation, the flashes had stopped. The images appeared whenever he was alone and quiet in his room or seated under a tree in his compound but disappeared with movements of his head. He described feeling initially confused and agitated but later pleasant and worried. He did not give any history to suggest abnormalities of auditory, taste, smell, tremors, weakness, intake of neither medications nor smoking.

His concern of disclosure became real when his

Article Access



Website: www.wjmb.com

 10.5281/zenodo.17095157

How to cite this article

Amos B Silas. Charles Bonnet Syndrome: Visual Hallucination in a 75 Year Blind Patient; Case Report and Review of Literature. *West J Med & Biomed Sci.* 2023;4(1-2):68-70. DOI:10.5281/zenodo.17095157.

children presented him to a federal neuropsychiatric Hospital in the same city to ascertain his mind. The Neuropsychiatrists told him he did not have any Neuropsychiatric abnormality. He was reassured and given follow-up date. His desire to seek for Ophthalmologist's opinion on the possibility that he could recover his vision despite knowing the images were not real led him to our clinic.

He was diagnosed 30 years before with glaucoma and cataract and later had combine cataract and glaucoma surgery that resulted in complications with a sudden loss of vision on the right eye and a gradual loss on the left eye over several years. Over time, he experienced pain, tearing and foreign body sensation in both eyes.

The assessment showed he was oriented in person, place but vaguely in time. There was normal muscle power, bulk and tone. He could not perceive light on visual assessment and had corneal scarring of varied density in both eyes. Other findings on the right eye were flat anterior chamber (AC), 360 degrees peripheral iridocorneal touch and a pupillary membrane. On the left eye, areas of corneal epithelial bullae, a glimpse of a deep AC, iris and pupillary membrane were seen on slit-lamp examination. Goldman's applanation tonometer could not measure the intraocular pressure because of irregular mires but the globes were hard on digital palpation.

CBS was entertained because of the presence of visual loss with hallucinations and the absence of neurological and psychiatric complaints. The patient was counseled on his irreversible blindness, educated on CBS, reassured and placed on tablets Diamox 250mg bd, gutttimolol 0.5% bd, gutt hypertonic saline 5% qds and ointment chloramphenicol bd for the bullous keratopathy. He was referred back to the neuropsychiatrist that earlier attended to him. The patient did not report for his scheduled appointment.

DISCUSSION

Combine surgery is offered in coexistent cataract and glaucoma to reverse cataract visual impairment, reduce IOP and slow down optic nerve damage. However, we found the patient's IOP was not controlled evidenced by the subjective digital palpation of hard globes, likely caused by secondary complications of post operative endophthalmitis as

suggested by other findings in the AC. When IOP is High and sustained, it causes injury to the corneal endothelium and eventual corneal decompensation with epithelial bullae (Bullous keratopathy) which is reported to be caused mainly by secondary glaucoma and intraocular surgeries seen in this case². This caused the pain, tearing and FB sensation experienced by the patient. The main treatment of BK is a corneal transplant but is hardly available in Nigeria. We had to treat conservatively with medication prescribed to the patient. In addition, NSAID use is indicated but for the patient's complaint of lid swelling associated with its previous use.

Visual loss caused by glaucoma and complications of surgery in this aged patient have all been implicated in CBS³. This first case in our busy clinic supports the view that patients and care givers' role downplay the significance of the condition reported to be as high as 10-40% among visually impaired persons.⁴ Gilmour et al⁵, found that only 9% of people experiencing CBS sought medical advice. Disappointingly, only half of them received an explanation about CBS from care givers.

The explanation of CBS is that when deafferentation occurs the visual cells in the brain cortex experiences decreased signal, in some situations, the visual cells spontaneously increase their own activity releasing new fantasy pictures or information they have stored.⁶ Brain scanning has provided proof of increased activity in the ventral striae cortex of patients with CBS⁷.

CBS is self-limiting, initially, the person is in a state of fear and apprehension followed by the stage of acceptance and then it recedes in about 18 months. A study⁸ reported that 60 % of patients had resolution of their hallucinations at 18 months. No drug is known to combat CBS but is considered only for patients who are truly distressed or depressed by their visual hallucinations or have preexisting behavioral problems.

Psychoeducation and supportive psychotherapy on assurances that visual impairment is responsible for the hallucinations and that CBS is not a mental abnormality generally alleviates any fear or distress

patients pass through.¹ We opted for this form of treatment because patient's condition had improved as evidenced by the less frequent and shorter duration of complex and resolution of simple forms at presentation. However, in CBS patients that have some vision left, therapeutic interventions to improve and reverse vision loss such as surgery and optical devices have been reported to improve the hallucinations.⁹

Accidentally, the patient discovered shaking his head help in controlling the hallucinations and hence agrees with documented mechanisms of coping with CBS such as ocular and body movements.^{1,10} Others are, changing or altering the environment setting, shouting at, staring at, fixating on, approaching and/or hitting at the image^{1,10} and CBS peer support groups. It has been suggested that social and sensory isolation are contributing factors in CBS because the hallucinations are often reported when the brain is not being distracted by other stimuli.¹¹ This Suggests modifying the environment with music or noise has a possible role in treatment.

CONCLUSION

Care givers must watch out for this rare presentation of blindness which could be distressing in an already hopeless situation and management should be multidisciplinary involving psychiatrists.

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