Visual hallucinations caused by Charles Bonnet Syndrome: A case study

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Introduction

Ms P presented with apsychotic visual hallucinations in the presence of decreased visual acuity. She was an 83-year-old female with no prior psychiatric complaints. Medically she suffered from Paget's disease, gastritis, osteoarthritis, dyslipidaemia and bilateral macular degeneration. This combination is known as Charles Bonnet Syndrome, after a Swiss philosopher who described it in 1796. The term hallucination is said to come from the Latin word *alucinari*, meaning "to wander in the mind", and was introduced by Esquirol in 1837. It is defined as sensory perception that has the compelling sense of reality of a true perception without a corresponding external stimulus to the corresponding sensory organ.[1] Charles Bonnet described Charles Bonnet Syndrome in his grandfather - Charles Lullin.[2] Lullin underwent a bilateral cataract extraction at the age of 89 and developed very clear visual hallucinations of buildings that were not present. He had no cognitive impairment and was fully aware of the unreality of his experiences. Visual acuity disturbance is a frequent complaint of patients in general practice.

Presentation

Ms P, an 83 year old female was referred by her GP to me for evaluation. Her 59-year-old eldest daughter accompanied her. She had consulted an ophthalmologist in March 2003 due to visual fallout. He diagnosed bilateral macular degeneration. Ms P's main complaint was visual hallucinations. She would see the faces of her deceased mother, father, and brother in front of her eyes. She also displayed metamorphosia: a visual illusion where she would see people's faces (including her own) changing shapes and sizes, for example, everyone around her would seem to develop a full beard. These symptoms were ego-dystonic and disturbing. They were unwanted and did not feel part of her imagination, but represented reality. She was, for obvious reasons, concerned about them. The symptoms had persisted for a few months but had no specific onset. Her GP also treated her for depression, with good effect, and she was totally euthymic at the time of the interview. On special testing she scored 7 on the Hamilton Depression Rating Scale (21 questions), which indicated the absence of depression. Her Folstein Mini Mental Score was 27/27, which is perfectly normal (three questions were disregarded due to her poor vision). Needham and Taylor in the USA and Teunisse in the Netherlands explored Charles Bonnet Syndrome. The latter described the following "diagnostic criteria":[3]

1. Presence of complex visual hallucinations
2. Full retention of insight into the unreal nature of the hallucinations
3. Absence of hallucinations in other sensory modalities
4. Absence of delusions

The incidence of the disorder ranges between 5.1 (1 in 20) to 14.4% (1 in 10) in patients with visual field pathology. According to Teunisse, some patients do not find the hallucinations disturbing (13 % are amused by it), but 18% do find them disturbing, while 10% find them extremely discomforting, as did Ms. P.[3] Of Teunisse's group:

- 65% of patients hallucinated weekly or monthly and 27% daily, as in Ms P's case
- 80% of hallucinations involved people, as in this case, while 38% involved animals, 25% involved plants or trees, and 15% involved buildings or scenes, as in Lullin's case
- 63% of hallucinations were in colour, 47% involved movement and 45% were seen with more clarity than "real" objects
- 53% of hallucinations lasted 1 - 60 minutes and 13% lasted less than 5 seconds
- 67% of patients experienced the hallucinations with open eyes. [3]

Prior Psychiatric History

In Ms P's case, no prior personal or family history of any mental disorders was present. A differential diagnosis for Charles Bonnet Syndrome includes: [2]

1. **Psychological events:** Mourning and stress, Sensory deprivation, Sleep deprivation, Acquired deafness, Phantom limb experiences, Flashback phenomena
2. **Due to psycho-mimetic drugs:** LSD, PCP, Peyote, Psilocybin, Cannabis
3. **Neurological aetiology:** Epilepsy, Migraine, Vertiginous, Hypnogogic and hypnopompic

Table I: Atypical Antipsychotic medications

Atypical Antipsychotic medications	Dosage range
Olanzapine	2.5 – 10 mg/day
Quetiapine fumarate	25 – 100 mg /day
Risperidone	0.25 – 1 mg/day

hallucinations

4. **Medical aetiology:** Multiple sclerosis, Serous otitis media, Intra-otic foreign bodies
5. **Pseudo-hallucinations:** Radio reception via shrapnel wound in cranial bone of left parieto-occipital region, resonating to a frequency of 560 AM, Virtual reality exposure, Malingering

Past Medical History:

Ms P suffered from bilateral macular degeneration with scotomae, Paget's disease with osteoporosis, dyslipidaemia, gastritis and osteoarthritis.

Current Medication:

Rabeprazole 10 mg daily; Alendronate 70 mg weekly; Calcium supplement I bid; Conjugated oestrogen 1,25 mg daily; Meloxicam 7,5 mg daily; Amitriptyline 25 mg nocte; Citalopram 20 mg daily; and Atorvastatin 10 mg daily

Substance use:

Neither Ms P nor her daughter noted any problems due to present or past substance misuse.

Biography:

Ms P is the youngest of five children. She had an unremarkable childhood with no specific problems. Her highest academic qualification was Standard 9 (Grade 11) and she spent her life, after the birth of her children, as a mother and homemaker. She supplemented the family income by making dresses in her spare time and this developed into a small business enterprise. She was married three times. She divorced her first husband after three months of marriage due to his alcohol abuse. Both her second and third husband passed away and she has been a widow since 1995. She currently stays in an old age home and lives off her pension and investments.

Significant risk factors for the development of Charles Bonnet Syndrome, according to Teunisse, are:[3]

1. Loss of energy
2. A large number of co-morbid somatic disorders (as are present in this case)
3. Beta blocking agents
4. Loneliness (not necessarily in Ms P's case)
5. Low extraversion
6. Shyness

It is also necessary to screen for delirium, dementia and vascular brain disease (usually in the occipito-parietal or occipito-temporal areas).

Mental Status

Ms P was a well-groomed, courteous, and friendly elderly lady. She walked unaided and accompanied by her daughter, with whom she seems to have a comfortable relationship. She spoke freely and easily and we developed rapid rapport. She seemed calm and at ease. Ms P had a full affect, which responded appropriately. Her language skills were intact, but she displayed obvious visual problems. She was fully awake and conscious and showed no signs of either a delirium or a dementia. No cognitive symptoms were elicited. Except for the complaint of visual hallucinations, she was apsychotic. Her thought processes were normal in rate, form, and content. No mood or anxiety symptoms were elicited. The patient and her daughter were counselled about her condition and reassured about the absence of major psychiatric disorders.

Investigations

CT brain scan, with special emphasis on the occipito-temporal and occipito-parietal areas, revealed generalised cerebral atrophy, in keeping with her age, but no specific pathology was noted. An electroencephalogram (EEG), with emphasis on the temporal and occipital areas and examined by two experienced neurophysiologists, was within normal limits.

Management

Treatment with Risperidone 0.5 mg nocte for two weeks was initiated as a therapeutic trial. Ms. P experienced side effects due to the Risperidone, mainly dizziness and fatigue. She also developed orthostatic hypotension, as well as signs of congestive heart failure; the Risperidone was subsequently down-titrated and stopped. Quetiapine, at a dose of 25 mg nocte, was initiated for a two-week period, to which she responded very favourably, with only minor complaints of somnolence. Ms P declined up-titration of this dose, despite its suggestion. This low dose of antipsychotic medication might have been effective via an anxiolytic mechanism rather than an antipsychotic action. The techniques developed by Teunisse and his group for dealing with the hallucinations are:[3]

1. Closing the eyes
2. Opening the eyes
3. Blinking the eyes
4. Moving the eyes rapidly
5. Looking or walking away from the hallucinations
6. Approaching the hallucinations
7. Visual fixation on the hallucinations
8. Distraction techniques
9. Hitting at the hallucinations
10. Shouting at the hallucinations.

His protocol calls for psycho-education, an improvement of physical health, medication replacement and a decrease of social isolation.According to Rovner, treatment should consist of education and reassurance.[4] Antipsychotic and anti-epileptic medications may be of value. Some of these hallucinations disappear spontaneously within 12 to 18 months. Increased lighting at home and decreased social isolation are recommended. Both the patient and clinician were satisfied with the outcome of this interesting case after prolonged treatment with Quetiapine.[5]

Conflict of interest

None declared

References

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