Charles Bonnet Syndrome

Case series

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Abstract – Since its first description in 1760, Charles Bonnet syndrome (CBS) has been reported in many studies. The main characteristics are visual hallucinations, preserved awareness of unreal visions, and absence of psychotic symptoms. CBS can occur with lesions located anywhere along the central visual pathway, from the eye to the calcarine fissure. ***Objective:*** To describe patients with CBS and carry out a review of the literature. ***Methods:*** Six patients with visual hallucinations were evaluated in an outpatient memory clinic between 2001 and 2008, and their clinical characteristics recorded. ***Results:*** Four patients were female, and the mean age was 74.5±16.9 years. Three patients had visual loss secondary to eye disease and three due to cerebral lesions. The visions consisted of animals, persons, moving objects, bizarre creatures or colored forms, and were considered disturbing by five patients. Five patients received treatment, and only three reported partial benefit from the therapy. Complete recovery was not seen in any of the subjects. ***Conclusions:*** CBS is relatively rare and its recognition is important to avoid misdiagnoses with psychiatric or dementing illnesses.

Key words: Charles Bonnet syndrome, visual hallucinations, visual loss.

Síndrome de Charles Bonnet: casuística

Resumo – Desde sua descrição em 1760, a Síndrome de Charles Bonnet (SCB) tem sido relatada em vários estudos. Suas características centrais são: alucinações visuais, consciência preservada sobre as alucinações e ausência de sintomas psicóticos. SCB pode ocorrer com lesões desde o olho até o córtex calcarino. ***Objetivo:*** Descrever pacientes com SCB e fornecer revisão da literatura. ***Métodos:*** Seis pacientes com alucinações visuais foram avaliados em um ambulatório de memória no período de 2001 a 2008 e suas características foram descritas. ***Resultados:*** Quatro pacientes eram do sexo feminino, e a idade média foi de 74,5±16,9 anos. Três dos pacientes apresentavam perda visual secundária a lesões oculares e três devido a lesões cerebrais levando a comprometimento visual. O conteúdo das visões consistia de animais, pessoas, objetos em movimento, criaturas bizarras ou formas coloridas. Cinco pacientes receberam tratamento, e apenas três relataram benefício parcial. Melhora completa não foi observada. ***Conclusões:*** SCB é relativamente rara e seu reconhecimento é importante para evitar erros diagnósticos com doenças psiquiátricas ou quadros demenciais.

Palavras-chave: síndrome de Charles Bonnet, alucinações visuais, perda visual.

Charles Bonnet Syndrome (CBS) is characterized by the presence of complex visual hallucinations, frequently associated to visual loss where patients are conscious of the fictitious nature of their hallucinations, and do not present psychotic symptoms.[1] The disorder was termed CBS in 1967 by de Morsier.[2]

The first report was of Bonnet's grandfather, who suffered from corneal degeneration and complex visual hallucinations of humanlike figures, birds, and buildings, yet manifested no cognitive or psychiatric disorders (Figure 1).[3] A wide range of hallucination types have subsequently been reported[4] and hallucinations have been described in

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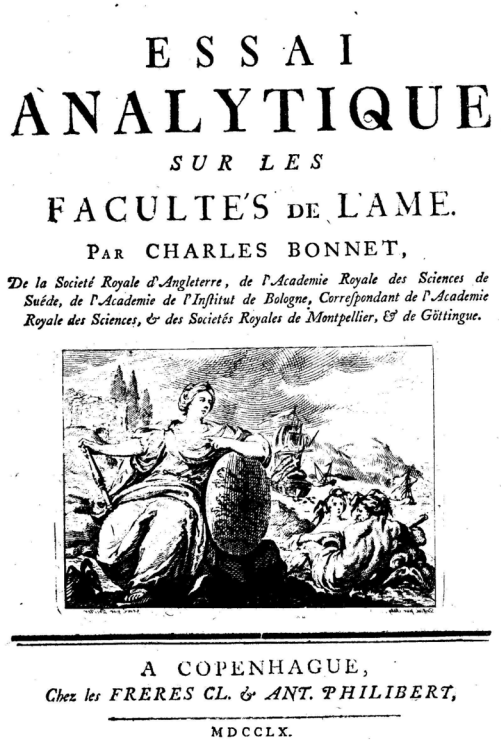
ESSAI
ANALYTIQUE
SUR LES
FACULTE'S DE L'AME.
PAR CHARLES BONNET,
De la Societé Royale d'Angleterre, de l'Academie Royale des Sciences de Suéde, de l'Academie de l'Inſtitut de Bologne, Correſpondant de l'Academie Royale des Sciences, & des Societés Royales de Montpellier, & de Göttingue.
A COPENHAGUE,
Chez les FRERES CL. & ANT. PHILIBERT,
MDCCLX.

***Figure 1**. Cover of Charles Bonnet's report.*

patients with lesions located anywhere from the eye to the calcarine fissure (see below).

The prevalence of CBS varies in the literature, with rates ranging from 0.4 to 15%.[5-11] More recently published studies have reported a lower prevalence of approximately 1% in Asia (0.5% in Japan).[12] In contrast, prevalence of CBS was 17.5% in 200 elderly with visual impairment in Australia,[13] and 27.5% in patients with age-related macular degeneration in the United Kingdom.[14] This syndrome has also been described in a few cases of children with vision loss.[15] Its prevalence probably underestimated, due to low disclosure by the patients, and owing to various medical conditions associated with the syndrome, as well as to the lack of knowledge about this condition among physicians.

Our aim is to describe a series of six patients with visual impairment and CBS that were evaluated by our group.

Methods

Patients with visual hallucinations referred for neurologic evaluation in an outpatient memory clinic from 2001 to 2008 (Cognitive and Behavioral Neurology Unit from the Hospital das Clínicas – University of São Paulo School of Medicine) or at one of the author's private practice (R.N.) and diagnosed with CBS (using the previously reported definition[1]) had their medical records reviewed. During the initial evaluation, besides recording of their demographical, clinical and radiological characteristics, patients were assessed to rule out cognitive impairment using scales including the Mini-Mental State Examination (MMSE).[16] The patients were subjected to other cognitive tests if deemed necessary.

Results

The data obtained on the six patients are described in Table 1. Four patients were female, and the mean age (±standard deviation) was 74.5(±16.90) years. Three patients had visual loss due to ocular disease, and three others presented cerebral lesions causing optic chiasmatic compression. All patients had significant visual loss, and developed vivid and bizarre hallucinations including animals, persons, objects or horrid and distorted images. Only one patient did not feel disturbed by the visions. Five patients received oral medications, which included acetylcholinesterase inhibitors, antidepressants and antipsychotics (alone, in combination, or subsequently tried). Although partial benefit was seen in three individuals, complete response was not seen in any of the cases.

In some cases, the hallucinations presented in an unusual manner. Patient 2 reported seeing a horse wagon moving towards her always while being in the front passenger seat of a car. Patient 5 reported visions of landscapes, with green grass and blue sky, associated with a peaceful sensation. Singularly, he was able to voluntarily change unpleasant visions (tiny creatures crawling on his food, snakes, monsters) with these pleasant hallucinations, providing him some degree of relief.

Discussion

Some risk factors for developing CBS have been described by many authors: visual impairment, cerebral damage, cognitive deficits, social isolation, sensory deprivation, and aging.[17] Most studies have shown age to be associated with CBS; where, among 500 low-vision patients the syndrome was significantly associated with an age of over 64 years, occurring in around 3% of patients aged 18 to 49 years and 15% in older elderly (75 to 84 years' old).[7] Teunisse et al. compared elderly subjects with loss of vision and CBS to those without hallucinations, and found that loneliness and low extraversion were predictors for developing CBS.[18] In this case series, the mean age was of

Table 1. Patient characteristics with Charles Bonnet syndrome.

Case	1	2*	3	4	5	6
Gender	Male	Female	Female	Female	Male	Female
Age	90	83	68	50	63	93
Cause of visual loss	Ocular disease	Macular degeneration	Optic chiasmatic compression	Optic chiasmatic compression	Optic chiasmatic compression	Macular degeneration
Visual acuity	N/A	0.1	Light perception	Count fingers at 2 meters	Amaurosis	Left eye: null; Right eye: 20/400
Frequency	Daily	Almost daily	Daily	Daily	Daily	Daily
Length of symptoms	2 years	1.5 year	3 years	6 years	5 years	1 year
Burden	Moderate	Not disturbed	Mild	Moderate	Mild	Yes
MMSE	26	28	22	23	23	27
Brain imaging study	N/A	N/A	Right frontal encephalomalacia, suprasellar tumor with compression of optical chiasma	Bilateral frontal gliosis	Suprasellar tumor with chiasmatic compression, Right frontal cephalomalacia	Brain CT: normal
Treatment	No	Donepezil	Rivastigmine + Sertraline	Olanzapine	Donepezil + sertraline	Galantamine; sertraline; risperidone; haloperidol
Response to treatment	N/A	No benefit	Partial benefit	Partial benefit	Partial benefit	No benefit
Type of hallucination	Land division, Asian army, mountains, children	Horse wagon	Distorted faces, birds transforming into hats, pigeons with dog faces, and a persistent fluctuating pilaster	Tiny creatures, black mice, snakes, little elephants bears, and monsters	Tiny creatures, fantastic beings, some green grass scenes, branches of herbs	Women dressed in purple clothes; several identical short men with hat; yellow and Black dots drawing a net over his home walls

*Patient 2 has been previously reported 41. N/A, Not available; MMSE, Mini-mental state examination.

Table 2. Diseases associated to Charles Bonnet syndrome.

Associated disease	Reference
Macular degeneration	Vukicevic & Fitzmaurice[13]; Khan et al.[14]; Cortizo et al.[41]
Choroidal neovascularization	Brown & Murphy[6]
Treatment for macular degeneration	Meyer et al.[42]
Enucleation of the eye	Tan et al.[43]
Glaucoma	Nesher et al.[11]; Tan et al.[44]
Central retinal artery occlusion	Tan et al.[44]
Optic neuritis, multiple sclerosis	Alao & Hanrahan[34]
Lesions of optic radiation	Freiman et al.[23]
Chiasmal and pituitary lesions	Lepore et al.[45]
Stroke in medial occipital lobe	Cole[46]
Resection of occipital lobe	Choi et al.[47]
Suprasellar meningioma	McNamara et al.[48]
Temporal arteritis, giant cell arteritis	Razavi et al.[49]

74.5±16.9 years, and was in-line with the findings of other reports.[5,9,13,19]

Complex visual hallucinations have been reported following a range of different conditions, and the syndrome stems from a variety of lesions at all levels of the visual system. The most frequent cause is age-related macular degeneration and its treatment (Table 2). In our case series, fifty per cent of our patients demonstrated visual impairment secondary to ocular disease and another half to cerebral lesions of visual pathways. Although visual impairment is not mandatory for the diagnosis of CBS, most authors report a strong association between the two.[17] All cases in the present series had significant visual disturbance.

Due to the small sample of patients, we cannot draw conclusions regarding gender preponderance in this study. In a review, Menon et al.[17] found divergent results concerning gender and CBS among various studies. The frequency of the hallucinations is usually reported as occurring daily or weekly.[13,19] Patients may also report continuous hallucinations or episodic hallucinations with longer intervals.[17] In five of our patients, the hallucinations occurred daily.

Although previously considered a condition with "pleasant or neutral" symptoms,[1] later reports have indicated that CBS can be a cause of emotional burden to patients[6,8,10]. Santhouse et al.[19] found that in a group of 34 patients, the hallucinations generated an emotional response in 50%, and in half of these, the experiences were unpleasant. Vukicevic & Fitzmaurice[13] reported that the syndrome caused moderate or severe stress in 16 out of 35 patients. In our group, five patients reported a burden associated with the hallucinations. Although this burden was referred to as mild or moderate in most cases, it highlights the importance of correct diagnosis of CBS (reassurance of the sanity of the patient has positive effects)[7] and of the decision on whether to treat (or at least attempt to treat) the symptoms or otherwise.

Ffytche & Howard[4] devised hallucination classification into eight categories: tessellopsia (overlapping patterns, repeated geometry); hyperchromatopsia (vivid and bright colors); prosopometamorphopsia (facial distortions, misshapen and mutilated heads); dendropsia (branching forms); perseveration (persistence of details in another scene); illusory visual spread; polyopia (many equal forms); micro/macropsia. The most common patterns in their patients were tessellopsia in 37% and abnormalities of size in 42% of patients, and of these 58% reported micropsia. Among our patients, we identified descriptions matching some of these categories, such as: tessellopsia ("black dots drawing a net over his home walls"), hyperchromatopsia ("women dressed in purple clothes") polyopia (as in "several identical short men with hats"), micropsia (e.g. "little elephants") and prosopometamorphopsia (e.g. "distorted faces"). Some patients reported landscapes, animals and/or objects, which could have been seen sometime in the past by them and thus could represent perseveration or long-term pallinopsia.[4]

Particular types of hallucinations are related to some areas of the brain, where the experiences of patients with CBS are associated with activity in extra-striate cortex.[4,19] Santhouse et al. using fMRI found that, hallucinations involving faces were localized in superior temporal sulcus; objects and scenes in the ventral occipito-temporal cortex, for example.[19] The notion that these hallucinations could be a release phenomenon provoked by unusual decreased input was described by Cogan, in 1973.[20] By using single photon emission computed tomography in five patients with CBS secondary to eye disease, Adachi et al. observed that all patients had hyperperfusion in the lateral temporal cortex, striatum and thalamus, and presumed that excessive cortical compensation in these areas could precipitate the syndrome;[21] using the same methodology (fMRI), increased activity in ventral extrastriate visual cortex was observed.[19] Burke suggested that hyperactivity in any area will evoke the imagery that is coded by that area.[22] The hypothesis of deafferentation seems a reasonable cause of these hallucinations, as they are generated in visual association areas.[23] Cone photoreceptor loss, as in macular degeneration, promotes retino-thalamic-deafferentation, which leads to functional deafferentation of extrastriate cortex.[24] Burke suggested that hallucinations result from deafferentation of visual structures in the brain, or from the effective silencing of the principal afferents to these structures.[22] The hypothesis can include two streams of information: one from the periphery to the centre and another in the opposite direction. As the flow from the periphery to the centre diminishes, the contrary flow rises.[25]

Serotonergic activity is related to visual pathways and probably linked to genesis of hallucinations. Serotonin levels are lower in the sensory visual deprived cortex.[26] Visual information converging in the geniculate nucleus lateral to the visual cortex is modulated by serotoninergic projections from the brainstem.[27] Acetylcholine is another neurotransmitter involved in visual hallucinations, and concentrated in the visual thalamic nuclei and visual cortex.[28]

The syndrome has an unpredictable outcome, halluci-

natory episodes last from seconds to days, and duration of CBS may extend to years. In our cases, some therapeutic options were tried, with partial or no success. As positive outcomes are common with the passing of time, partial responses could be due to spontaneous recovery.

Treatments have been used in reports of single cases or case series in the literature. No controlled clinical trials have been published to date. Reports refer to spontaneous regression, and positive results in some cases with pharmacologic treatment. The majority of studies involve the use of antipsychotic or anticonvulsant drugs. Outcomes are variable, probably due to patient heterogeneity. In particular, the therapeutic response may diverge according to anatomical lesion, albeit ocular or in the brain.

- Anticonvulsants- carbamazepine;[29] valproate;[30] gabapentin[31]
- Haloperidol[32]
- Atypical neuroleptics: risperidona,[33] olanzapine[34]
- Selective serotonin reuptake inhibitors : venlafaxine, citalopram[35]
- Mirtazapine[36]
- Anticholinesterasic drugs: donepezil[37]
- 5-HT3 antagonist – cisapride[38]

Pilsin et al.[39] have raised the question over whether CBS could be a sign of the initial stages of a dementing illness, having found that patients with CBS performed worse in neuropsychological testing than controls. However, in this study, eight out of fifteen patients had no insight of the illusory nature of their hallucinations, and according to the most frequently accepted definitions,[1] patients should be diagnosed with CBS only if they are conscious of the unrealistic nature of their visions. There are reports of patients initially diagnosed with CBS – with or without mild cognitive impairment – that later developed Alzheimer's disease.[40] The association of CBS and dementia should not lead to the conclusion that CBS is a risk factor for the development of dementia, as advanced age is a risk factor for both conditions while this association (CBS and dementia) is only rarely reported. It is indeed necessary that patients manifesting visual hallucinations, even with concomitant visual impairment, should undergo a thorough evaluation to exclude underlying cognitive impairment. Long-term follow-up is also important, so that initial signs of cognitive deterioration can be detected, should they appear.

There are a number of limitations of this study. Firstly, information on imaging studies was not available in two cases. It is advisable to rule out the presence of structural abnormalities that could explain the visual hallucinations; however, in the cases reported, the absence of other focal neurological signs and accompanying symptoms on follow-up made the possibility of structural causes less likely. We also possessed no information on the influence of the symptoms of CBS on daily life activities, which could substantiate the impact of the symptoms and thus aid in management decisions (as discussed above). It should be noted however that evaluating the impact on daily life activities in visually impaired patients can be problematic.

Summing up, akin to other reported cases of CBS, our patients' visions contained vivid and colored pattern, a mixture of images, scenes, abnormal sizes, and the subjects showed preserved insight regarding these unrealistic visions. None of the patients responded well to treatment, and among those with some positive response this outcome may not have been due to treatment, but instead to spontaneous remission or fluctuating course. Despite the paucity of treatment options, awareness and recognition of CBS is of the utmost importance to avoid misdiagnoses with dementing or psychiatric illnesses and to offer patients reassurance regarding the integrity of their mental status.

References

1. Damas-Mora J, Skelton-Robinson M, Jenner FA. The Charles Bonnet syndrome in perspective. Psychol Med 1982;12: 251-261.
2. DeMorsier G. The Charles Bonnet syndrome: visual hallucinations in the aged without mental deficiency. Ann Med Psychol (Paris) 1967;2:678-702.
3. Bonnet C. Essai analytique sur les aculties de l'ame. Copenhagen: Ferres & Philibert; 1760: 426-429.
4. Ffytche DH, Howard RJ. The perceptual consequences of visual loss: "positive" pathologies of vision. Brain 1999;122:1247-1260.
5. Norton-Willson L, Munir M. Visual perceptual disorders resembling the Charles Bonnet syndrome: a study of 434 consecutive patients referred to a psychogeriatric unit. Fam Pract 1987;4: 27-31.
6. Brown GC, Murphy RP. Visual symptoms associated with choroidal neovascularization: photopsias and the Charles Bonnet syndrome. Arch Ophthalmol 1992;110:1251-1256.
7. Teunisse RJ, Cruysberg JRM, Verbeek A, Zitman FG. The Charles Bonnet syndrome: a large prospective study in Netherlands. Brit J Psychiatry 1995; 166: 254-257.
8. Teunisse RJ, Cruysberg JR, Hoefnagels WH, Verbeek AL, Zitman FG. Visual hallucinations in psychologically normal people: Charles Bonnet's syndrome. Lancet 1996;347:794-797.

9. Adachi N. Charles Bonnet syndrome in leprosy: prevalence and clinical characteristics. Acta Psychiatr Scand 1996;93:279-281.
10. O'Reilly R, Chamberlaine C. Charles Bonnet syndrome: incidence and demographic and clinical features. Can J Psychiatry 1996;41:259-260.
11. Nesher R, Nesher G, Epstien E, Assia E. Charles Bonnet Syndrome in glaucoma with low vision. J Glaucoma 2001;10:396-400.
12. Shiraishi Y, Terao T, Nakamura J, Tawara A. The rarity of Charles Bonnet Syndrome. J Psychiatr Res 2004;38:207-213.
13. Vukicevic M, Fitzmaurice K. Butterflies and black lacy patterns: the prevalence and characteristics of Charles Bonnet hallucinations in an Australian population. Clin Experiment Ophthalmol 2008;36:659-665.
14. Khan JC, Shadid H, Thurlby DA, Yates JR, Moore AT. Charles Bonnet syndrome in age-related macular degeneration: the nature and frequency of images in subjects with end-stage disease. Ophthalmic Epidemiol 2008;15:202-208.
15. Schwartz TL, Vahgei L. Charles Bonnet syndrome in children. J AAPOS 1998;2: 310-313.
16. Brucki SM, Nitrini R, Caramelli P, Bertolucci PH, Okamoto IH. Sugestões para o uso do mini-exame do estado mental no Brasil. Arq Neuropsiquiatr 2003; 61:777-81.
17. Menon GJ, Rahman I, Menon SJ, Dutton GN. Complex visual hallucinations in the visually impaired: the Charles Bonnet syndrome. Surv Ophthalmol 2003;48:58-72.
18. Teunisse RJ, Cruysberg JR, Hoefnagels WH, Kuin Y, Verbeek AL, Zitman FG. Social and psychological characteristics of elderly visually handicapped patients with the Charles Bonnet Syndrome. Compr Psychiatry 1999;40:315-319.
19. Santhouse AM, Howard RJ, ffytche DH. Visual hallucinatory syndromes and the anatomy of the visual brain. Brain 2000;123:2055-2064.
20. Cogan DG. Visual hallucinations as release phenomena. Arch Klin Exp Ophthalmol 1973;188:139-150.
21. Adachi N, Watanabe T, Matsuda H, Onuma T. Hyperperfusion in the lateral temporal cortex, the striatum and the thalamus during complex visual hallucinations: single photon emission computed tomography findings in patients with Charles Bonnet syndrome. Psychiatry Clin Neurosci 2000;54:157-162.
22. Burke W. The neural basis of Charles Bonnet hallucinations: a hypothesis. J Neurol Neurosurg Psychiatry 2002;73:535-541.
23. Freiman TM, Surges R, Vougioukas VI et al. Complex visual hallucinations (Charles Bonnet syndrome) in visual field defects following cerebral surgery. J Neurosurg 2004;101:846-853.
24. Plummer C, Kleinitz A, Vroomen P, Watts R. Of Roman chariots and goats in overcoats: the Syndrome of Charles Bonnet. J Clin Neurosc 2007;14:709-714.
25. Villa FJ. The Charles Bonnet Syndrome. An R Acad Nac Med (Madr) 2008;125: 387-395.
26. Qu Y, Eysel UT, Vandesande F, Arckens L. Effect of partial sensory deprivation on monoaminergic neuromodulators in striate cortex of adult cat. Neuroscience 2000;101:863-868.
27. Seeburg DP, Liu X, Chen C. Frequency-dependent modulation of retinogeniculate transmission by serotonin. J Neurosci 2004;24:10950-10962.
28. Manford M, Andermann F. Complex visual hallucinations. Clinical and neurobiological insights. Brain 1998;121:1819-1840.
29. Görgens K, Liedtke M. Charles Bonnet syndrome. Psychiatr Prax 1998; 25: 85-86.
30. Hori H, Terao T, Shiraishi Y. Treatment of Charles Bonnet syndrome with valproate. Int Clin Psychopharmacol 2000; 15:117-119.
31. Paulig M, Mentrup H. Charles Bonnet syndrome: complete remission of complex visual hallucinations treated by gabapentin. J Neurol Neurosurg Psychiatry 2001;70: 813-814.
32. Valencia C, Franco JG. Charles Bonnet syndrome: report of one case managed with haloperidol. Rev Med Chil 2008;136:347-350.
33. Maeda K, Shirayama Y, Nukina S, Yoshioka S, Kawahara R. Charles Bonnet syndrome with visual hallucinations of childhood experience: successful treatment of 1 patient with risperidone. J Clin Psychiatry 2003;64:1131-1132.
34. Alao AO; Hanrahan B. Charles Bonnet syndrome: visual hallucination and multiple sclerosis. Int J Psychiatry Med 2003;33:195-199.
35. Lang UE, Stogowski D, Schulze D et al. Charles Bonnet Syndrome: successful treatment of visual hallucinations due to vision loss with selective serotonin reuptake inhibitors. J Psychopharmacol 2007;21:553-555.
36. Siddiqui E, Ramaswamay S, Petty F. Mirtazapine for Charles Bonnet syndrome. Can J Psychiatry 2004;49:787-788.
37. Ukai S, Yamamoto M, Tanaka M, Takeda M. Treatment of typical Charles Bonnet syndrome with donepezil. Int Clin Psychopharmacol 2004;19:355-357.
38. Ranen NG, Pasternak RE, Rovner BW. Cisapride in the treatment of visual hallucinations caused by vision loss: the Charles Bonnet syndrome. Am J Geriatr Psychiatry 1999;7: 264-265.
39. Pliskin NH, Kiolbasa TA, Towle VL et al. Charles Bonnet Syndrome: an early marker for dementia ? J Am Geriatr Soc 1996;44:1055-1061.
40. Donoso SA, Silva RC, Fuentes GP, Gaete CG. Síndrome de Charles Bonnet: presentación de tres casos y revisión de la literatura. Rev Med Chile 2007; 135:1034-1039.

41. Cortizo V, Rosa AAM, Soriano DS, Takada LT, Nitrini R. Charles Bonnet syndrome: visual hallucinations in patients with ocular diseases- case report. Arq Bras Oftalmol 2005;68: 129-132.
42. Meyer CH, Mennel S, Hörle S, Schmidt JC. Visual hallucinations after intravitreal injection of bevacizumab in vascular age-related macular degeneration. Am J Ophthalmol 2007;143:169-170.
43. Tan CS, Sabel BA, Eong KG. Charles Bonnet syndrome (visual hallucinations) following enucleation. Eye 2006;20: 1394-1395.
44. Tan CS, Sabel BA, Goh KY. Visual hallucinations during visual recovery after central retinal artery occlusion. Arch Neurol 2006;63:598-600.
45. Lepore FE. Spontaneous visual phenomena with visual loss: 104 patients with lesions of retinal and neural afferent pathways. Neurology 1990;40:444-447.
46. Cole M. When the left brain is not right the right brain may be left: report of personal experience of occipital hemianopia. J Neurol Neurosurg Psychiatry 1999; 67:169-173.
47. Choi EJ, Lee JK, Kang JK, Lee SA. Complex visual hallucinations after occipital cortical resection in a patient with epilepsy due to cortical dysplasia. Arch Neurol 2005;62:481-484.
48. McNamara ME, Heros RC, Boller F. Visual hallucinations in blindness: the Charles Bonnet syndrome. Int J neurosci 1982;17:13-15.
49. Razavi M, Jones RD, Manzel K, Fattal D, Rizzo M. Steroid-responsive Charles Bonnet syndrome in temporal arteritis. J Neuropsychiatry Clin Neurosci 2004;16: 505-508.