

Visual Hallucinations and Ischemic Stroke: Review of 5 Cases

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Abstract

Background: Charles Bonnet syndrome (CBS) presents with complex, vivid, repetitive visual hallucinations that occur in people who have lost some of their vision. The most common cause is senile macular degeneration. CBS can occur, rarely, in patients whose visual pathways are affected. We present five patients with complex visual hallucinations who had occipital lobe lesions after ischemic stroke.

Case report: The patients included four men and one woman with a mean age of 71.8 ± 13.08 years. The neurological examinations showed left homonymous hemianopsia in four patients and right homonymous hemianopsia in one, plus there was hemiparesis in two patients and ataxia in one patient. An acute ischemic lesion was seen in the posterior cerebral artery territory in all patients on diffusion-weighted magnetic resonance imaging (MRI). Visual hallucinations in the hemianopic area occurred within the first 48 h of the stroke and lasted from a few minutes to half an hour. Electroencephalography (EEG) taken while symptomatic or shortly thereafter showed mild bioelectrical slowing in the right hemisphere in one patient, while the EEGs of the other patients were normal. During follow-up, the visual hallucinations disappeared in all patients within 3 months, without special treatment.

Conclusion: The correct diagnosis of CBS, and treatment if necessary, are important and require an evaluation by a multidisciplinary team that includes neurologists, ophthalmologists, and psychiatrists.

Keywords: Charles Bonnet syndrome; Occipital lobe lesion; Visual hallucination; Ischemic stroke

Introduction

Charles Bonnet syndrome (CBS) is characterized by complex, vivid, recurrent visual hallucinations that occur in people with no known psychiatric illnesses, normal consciousness and a loss of visual function [1]. This syndrome was first described by the Swedish scientist Charles Bonnet in 1760 based on the visual hallucinations his grandfather Thomas Lullin developed after cataract surgery. Bonnet also developed similar visual hallucinations associated with eye problems when he was older [2]. In 1967, the Swiss scientist George de Morsier called the syndrome Charles Bonnet syndrome [3].

The prevalence of CBS varies ranges from 0.4% to 15% in patients with impaired vision [4]. The most common cause is senile macular degeneration, and other causes are glaucoma, cataracts, post-enucleation, optic neuritis, macular photocoagulation, Leber's hereditary optic neuropathy, macular translocation, central retinal artery occlusion, and arteritic and non-arteritic anterior ischemic optic neuropathy [5-8]. It is seen more rarely in patients whose visual pathways are affected. Lesions of the occipital lobe are seen in multiple sclerosis, temporal arteritis and meningiomas [9-12].

Simple hallucinations occur with occipital lobe lesions, while complex visual hallucinations are associated with occipitotemporal and occipitoparietal visual association cortex damage [13].

This study presents five patients with complex visual hallucinations seen in our neurology clinic between 2015 and 2017 that were

diagnosed with ischemic stroke (acute unilateral/bilateral occipital lobe infarct) in the posterior cerebral artery (PCA) territory and had homonymous hemianopsia on neurological examination.

Case Report

The patients comprised four men and one woman with a mean age of 71.8 ± 13.08 years. All patients had acute-onset visual impairment: one had headache, two had unilateral weakness and one had ataxia. In the neurological examinations, left homonymous hemianopsia was found in four patients and right homonymous hemianopsia in one. In addition to these findings, left hemiparesis was present in two patients and ataxia in one. In their medical histories, four patients had hypertension, one had diabetes mellitus, one had ischemic stroke, and three had coronary artery disease. None of our patients had epilepsy, alcohol or drug abuse, Parkinson's disease, dementia, or psychiatric illnesses.

Diffusion-weighted magnetic resonance imaging (MRI) showed acute ischemic lesions on the right side in two patients, on the left side in one, and bilaterally in the PCA territory in two (Figure 1). Routine laboratory examinations detected hyperlipidaemia in two patients and the other tests were normal in all patients, including serum electrolytes, glucose and liver and kidney functions.

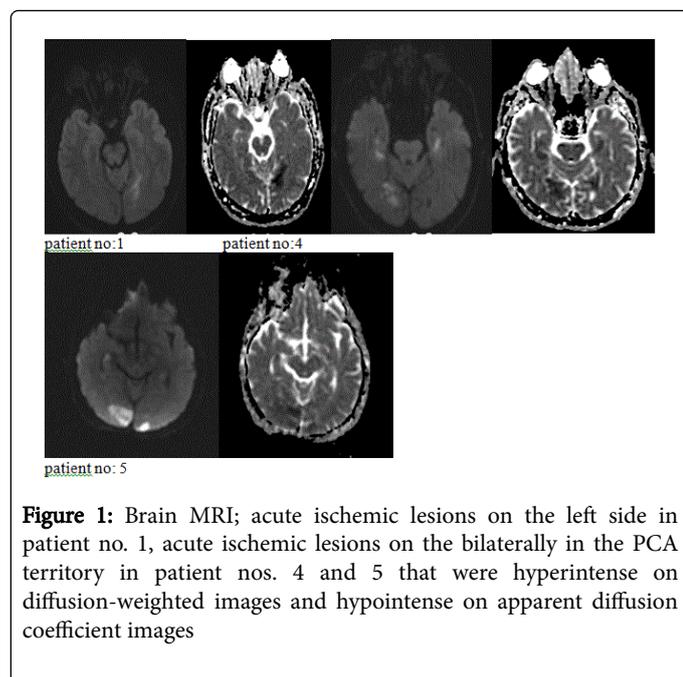


Figure 1: Brain MRI; acute ischemic lesions on the left side in patient no. 1, acute ischemic lesions on the bilaterally in the PCA territory in patient nos. 4 and 5 that were hyperintense on diffusion-weighted images and hypointense on apparent diffusion coefficient images

Within the first 48 h, the patients complained of visual hallucinations in the hemianopic area lasting from a few seconds to half an hour. The first patient saw a crowd of people and a television with indistinct faces in an office-shaped room on the right side, a man wearing light grey, and a women dressed in burgundy; the second patient saw red and green balls on the left side; the third patient saw coloured pieces of paper, newspapers, trees and people on the left side; the fourth patient saw coloured balls and nested circles in the left side; and the last patient saw his own small child moving and playing. The hallucinations occurred when the patients were completely awake and the patients reported that they knew that they were not real. The hallucinations continued all day, and were not associated with loss of consciousness, convulsions or abnormal behaviours.

Electroencephalography (EEG) performed while the patients were symptomatic or shortly thereafter showed mild bioelectrical slowing in the right hemisphere of a patient with a lesion in the right PCA territory, while the other patients' EEGs were normal (Table 1). The mean standardized Mini-Mental test score was 27 ± 1.58 (range 25-29). Neuro-ophthalmological examinations performed by an ophthalmologist were normal.

Patient number	Age/Sex	Diffusion-weighted MRI	Visual Field Defect	EEG	Characteristics of hallucinations	Duration of hallucinations
1	62/M	Left occipital lobe infarct	Right homonymous hemianopsia	Normal	Crowd of people and a television with indistinct faces in an office-shaped room on the right side, a man wearing light grey and a women dressed in burgundy	Any time
2	84/M	Right occipital lobe infarct	Left homonymous hemianopsia	Mild bioelectrical slowing	Red and green balls on the left side	Any time
3	78/F	Right occipital lobe infarct	Left homonymous hemianopsia	Normal	Coloured pieces of paper, newspapers, trees, and people on the left side	Any time
4	81/M	Bilateral occipital lobe infarct	Left homonymous hemianopsia	Normal	Coloured balls and nested circles in the left side	Any time
5	54/M	Bilateral occipital lobe infarct	Left homonymous hemianopsia	Normal	His own small child moving and playing	Any time

Table 1: Distribution of findings and characteristics of hallucinations.

None of the patients had been treated. When assessed during follow-up, the visual hallucinations remained in all patients for up to 3 months without any specific treatment, and only homonymous hemianopsia remained as a sequelae.

Discussion

The etiology of CBS is unclear. However, two important mechanisms have been proposed. The first of these is the release phenomenon. The main factor involved is sensory deprivation. Where ocular pathologies reduce sensory input, the visual association areas are affected, which leads to a reduction in the suppression of the high cortical centres, and previously subconscious perceptions are 'released' into consciousness, resulting in a visual hallucination. This mechanism

is equivalent to the phantom limb syndrome following limb amputation [14,15]. The second theory is the currently accepted theory of deafferentation, in which the absence of cortical visual sensory input due to visual impairment leads to visual hallucinations, with increased frequency of spontaneous discharge and disinhibition of neurons in the visual association cortex [5]. Ashwin observed vivid images of lions and cats, a flock of birds, a pack of hounds, chessboards and brightly coloured scarves in the hemianopic field following an occipital lobe infarct as the visual pathway was intact to the cortex and these visual hallucinations were generated from the infarcted occipital cortex ("inverse phantom vision") [16].

When studied during hallucinations, functional MRI shows increased activity in the ventral occipital lobe, while single photon emission computed tomography shows hyperperfusion in the lateral

temporal cortex, striatum, and thalamus, suggesting that these areas contribute to the formation of hallucinations [17,18].

Charles Bonnet syndrome is characterized by the presence of complex, structured visual hallucinations in the form of people, faces, animals or trees. These may be white or coloured, static or animated [4,19]. While some of our patients saw simple shapes, balls, or circles, their other hallucinations were more complex, such as their office or a child.

The clinical course may be episodic, cyclic or chronic. While the duration is usually less than 12 months, they may persist for several years [4]. Santos Bueso et al. reported complex hallucinations that mainly involved people and faces, with movement and colour; their patient had daily episodes lasting between 3 and 5 min that evolved over 6-12 months [20]. Our patients' hallucinations lasted from a few minutes to half an hour and all disappeared within 3 months.

Cox and ffytche reported that the majority of people with CBS felt that the hallucinations had no real effect on their lives (60%), although a subset judged them to have fairly (25%) or very (8%) negative effects, and 6% reported a fairly or very pleasant effect (1%). Although initially known as "pleasant or neutral" symptoms and not an emotional burden, it has subsequently been shown that CBS creates an emotional burden in patients. CBS patients rarely have unpleasant symptoms [1,21]. Nevertheless, Santhouse et al. reported that the hallucinations were an emotional burden in 50% of 34 patients, and half of them were egodystonic [22]. Similarly, Vukicevic and Fitzmaurice reported that the syndrome caused moderate or severe stress in 16 of 35 patients [23].

Therefore, CBS may require treatment. The treatment should be individualized and include everything related to the aetiopathology of the syndrome. Comprehensive treatment includes providing adequate information to patients and their families, increasing the patient's quality of life, and treating the cause or anxiety disorder secondary to the symptoms. Several anticonvulsant, antipsychotic, and antidepressant agents have been tried, including carbamazepine, valproate, gabapentin, levetiracetam, haloperidol, olanzapine, risperidone, mirtazapine, venlafaxine, citalopram, donepezil, melperone and ondansetron [24-29].

The visual hallucinations had no effects on the daily functions of our patients. The described hallucinations were not egodystonic in any of our patients, and the patient who saw a child actually enjoyed the hallucination. In our other patients, the hallucinations were neutral and did not require treatment.

The hallucinations seen in mental disorders are mostly auditory hallucinations. Visual hallucinations are less frequent than auditory hallucinations in psychotic disorders and are related to the psychotic core, which is generally present in terms of content. The hallucinations are often accompanied by delusions or other manifestations of the mental disorder, such as schizophrenia and related mental disorders, or are accompanied by depression or mania in mood disorders.

Alcoholic hallucinosis is a rare psychiatric disorder in which isolated hallucinations dominate the picture and is distinguished from CBS by a history of chronic alcohol abuse.

Visual hallucinations can occur in patients with occipital lobe epilepsy (OLE). A diagnosis of OLE was excluded in all of our patients because they did not have epileptic activity in their EEG and the hallucinations occurred during or immediately before the EEG

examination. In addition, none of our patients were treated and the hallucinations disappeared within 3 months.

In elderly patients, hallucinations are frequently seen during the course of neurological disorders, such as Alzheimer's disease and Parkinson's disease or mental disorders such as delirium, psychosis, bipolar disorder, and drug abuse. The most common psychotic symptom in Parkinson's disease is that of visual hallucinations, with a prevalence of 22-38% [30]. None of our patients had a history of psychiatric illness or substance use. There were no signs of dementia or Parkinson's disease in any of our patients.

Conclusion

This paper is intended to increase the awareness of CBS, which we think is more common than believed. A diagnosis of CBS requires evaluation by a multidisciplinary team that includes neurologists, ophthalmologists, and psychiatrists, especially in elderly patients. It is important to ask about hallucinations in detail and perform an EEG and MRI to rule out OLE.

References

1. Teunisse RJ, Cruysberg JR, Hoefnagels WH, Verbeek AL, Zitman FG (1996) Visual hallucinations in psychologically normal people: Charles Bonnet's syndrome. *Lancet* 347: 794e797.
2. Bonnet C (1760) Analytical test on the acuties of the soul. Copenhagen: Ferres & Philibert, pp: 426-429.
3. de Morsier G (1967) The Charles Bonnet syndrome: Visual hallucinations in the aged without mental de ciency. *Ann Med Psychol (Paris)* 2: 678-702.
4. Santos-Bueso E, Sáenz-Francés F, Serrador-García M, Porta-Etessam J, Martínez-de-la-Casa JM, et al. (2014) Prevalence and clinical characteristics of Charles Bonnet syndrome in Madrid, Spain. *Eur J Ophthalmol* 24: 960-963.
5. Burke W (2002) The neural basis of Charles Bonnet hallucinations: A hypothesis. *J Neurol Neurosurg Psychiatry* 73: 535-541.
6. Tan CS, Sabel BA, Goh KY (2006) Visual hallucinations during visual recovery after central retinal artery occlusion. *Arch Neurol* 63: 598-600.
7. Madill SA, Ffytche DH (2005) Charles Bonnet syndrome in patients with glaucoma and good acuity. *Br J Ophthalmol* 89: 785-786.
8. Toosy AT, Robertson BJ, Jayaram H, Plant GT (2006) Monocular complex visual hallucinations and their suppression by eye closure. *Eye* 20: 732-733.
9. Choi EJ, Lee JK, Kang JK, Lee SA (2005) Complex visual hallucinations after occipital cortical resection in a patient with epilepsy due to cortical dysplasia. *Arch Neurol* 62: 481-484.
10. Freiman TM, Surges R, Vougioukas VI, Hubbe U, Talazko J, et al. (2004) Complex visual hallucinations (Charles Bonnet syndrome) in visual field defects following cerebral surgery report of four cases. *J Neurosurg* 101: 846-853.
11. Rovner BW (2006) The Charles Bonnet syndrome: A review of recent research. *Curr Opin Ophthalmol* 17: 275-257.
12. Kumral E, Uluakay A, Dönmez İ (2015) Complex visual hallucinations following stroke: Epileptic origin or a deafferentation phenomenon? *Neurologist* 20: 13-15.
13. Kölmel HW (1985) Complex visual hallucinations in the hemianopic eld. *J Neurol Neurosurg Psychiatry* 48: 29-38.
14. Cogan DG (1973) Visual hallucinations as release phenomena. *Graefes Arch Klin Exp Ophthalmol* 188: 139-150.
15. Hart J (1997) Phantom visions: Real enough to touch. *Elder Care* 9: 30-32.

16. Ashwin PT, Tsaloumas MD (2007) Complex visual hallucinations (Charles Bonnet syndrome) in the hemianopic visual field following occipital infarction. *J Neurol Sci* 263: 184-186.
17. Ffytche DH, Howard RJ, Brammer MJ, David A, Woodruff P, et al. (1998) The anatomy of conscious vision: An fMRI study of visual hallucinations. *Nat Neurosci* 1: 738-742.
18. Adachi N, Watanabe T, Matsuda H, Onuma T (2000) 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* 54: 157-162.
19. Gonzalez-Delgado M, Tunon A, Salas-Puig J (2004) Síndrome de Charles Bonnet. *Neurologia* 19: 80-82.
20. Santos-Bueso E, Serrador-García M, Porta-Etessam J, Rodríguez-Gómez O, Martínez-de-la-Casa JM, et al. (2015) Charles Bonnet syndrome. A 45 case series. *Rev Neurol* 60: 337-340.
21. Cox TM, Hffytche D (2009) Negative outcome Charles Bonnet Syndrome. *Br J Ophthalmol* 98: 1236-1239.
22. Santhouse AM, Howard RJ, Ffytche DH (2000) Visual hallucinatory syndromes and the anatomy of the visual brain. *Brain* 123: 2055-2064.
23. Vukicevic M, Fitzmaurice K (2008) Butterflies and black lacy patterns: The prevalence and characteristics of Charles Bonnet hallucinations in an Australian population. *Clin Experiment Ophthalmol* 36: 659-665.
24. Batra A, Bartels M, Wormstall H (1997) Therapeutic options in Charles Bonnet syndrome. *Acta Psychiatr Scand* 96: 129-133.
25. Siddiqui Z, Ramaswamy S, Petty F (2004) Mirtazapine for Charles Bonnet syndrome. *Can J Psychiatry* 49: 787-788.
26. Moja CM, Milano E, Gasverde S, Gianelli M, Giordana MT (2005) Olanzapine therapy in hallucinatory visions related to Bonnet syndrome. *Neurol Sci* 26: 168-170.
27. Ukai S, Yamamoto M, Tanaka M, Takeda M (2004) Treatment of typical Charles Bonnet syndrome with donepezil. *Int Clin Psychopharmacol* 19: 355-357.
28. Lang UE, Stogowski D, Schulze D, Domula M, Schmidt E, et al. (2007) Charles Bonnet Syndrome: Successful treatment of visual hallucinations due to vision loss with selective serotonin reuptake inhibitors. *J Psychopharmacol* 21: 553-555.
29. Grüter T, Ayzenberg I, Gold R, Börnke C (2016) Charles Bonnet syndrome successfully treated with levetiracetam. *J Neurol* 263: 1872-1875.
30. Fénelon G, Alves G (2010) Epidemiology of psychosis in Parkinson's disease. *J Neurol Sci* 289: 12-17.